Your Score: 1 % (124 unanswered questions) Exit

Question # 1

Select the single best answer to the numbered question.

A 50 year old female with a ten year history of type II diabetes presents for regularly-scheduled follow up. She has no complaints, and just visited her ophthalmologist last week. Current medications include glyburide, metformin, and simvastatin. On physical exam, vital signs are virtually unchanged from previous visits, with temperature 37.1 C (99 F), HR 80, BP 140/83, RR 15, and O2 Sat 98% on room air. Neurological examination reveals diminished sensation to light touch and pinprick in a stocking distribution on the lower extremities bilaterally. Remainder of physical exam is benign. Laboratory evaluation reveals: Na+ 136, K+ 3.9 Cl- 104, HCO3- 25, BUN 15, Cr 1.0, Glucose 150; hemoglobin A1c: 7.1%; Urinalysis: negative for ketones, glucose, bilirubin, leukocyte esterase, or blood; moderate protein; Lipid profile: Total cholesterol 146, HDL 46, LDL 100. At this time, which of the following would be the most appropriate intervention?

A. Increase simvastatin
B. Increase glyburide
C. Increase metformin
D. Add hydrochlorothiazide
E. Add lisinopril

You answered: E

Explanations:

A. The patient's lipid profile is technically at her target LDL – thus, she does not need an increase in her statin (answer A). Under the current ATP III guidelines, patients with diabetes are considered to have known coronary heart disease, and therefore have a target LDL of less than 100. To review the ATP III lipid guidelines at a glance, check out http://www.nhlbi.nih.gov/guidelines/cholesterol/atglance.pdf HOWEVER, please note that although the official guideline is still LDL <100, that figure is likely to change in the future, because new evidence has come out showing that lower LDLs (<70) are better... so in the near future, this question may have two correct answers!

B. Either increasing glyburide (answer B) or increasing metformin (answer C) would be helpful for reducing the patient's blood sugar and better controlling her diabetes. However, her most recent Hgb A1c was not that bad – remember that the goal for diabetic patients is less than 7.0%. For more on Hgb A1c, including how to correlate the test result with the patient's average blood glucose, check out Diabetes Care 2002;25:275-8 or http://www.metrika.com/3medical/hemoglobin-m.html.

C. Either increasing glyburide (answer B) or increasing metformin (answer C) would be helpful for reducing the patient's blood sugar and better controlling her diabetes. However, her most recent Hgb A1c was not that bad – remember that the goal for diabetic patients is less than 7.0%. For more on Hgb A1c, including how to correlate the test result with the patient's average blood glucose, check out Diabetes Care 2002;25:275-8 or http://www.metrika.com/3medical/hemoglobin-m.html.

D. Adding hydrochlorothiazide (answer D) could improve her hypertension, and would be a reasonable first-line antihypertensive in a patient without diabetes. It is worth noting that this patient IS hypertensive – all diabetics should have a systolic blood pressure of less than 130. Still, diabetic patients have a compelling reason to begin antihypertensive therapy with ACE inhibitors or ARBs, however, due to the reno-protective effects of these drugs.

E. Key teaching point: All diabetics should be on an ACE inhibitor or ARB for cardiovascular and renal protection. This is a dense question stem, but the important things to note are that this is a patient with type II diabetes who also has proteinuria and elevated blood pressure. Since her medication list does not include any mention of an ACE inhibitor or ARB, beginning one at this time would be the appropriate next step in management (answer E). As it turns out, microalbuminuria is a risk factor for both cardiovascular disease and progression of renal disease to ESRD and dialysis – and this patient already has macroalbuminuria, since dipsticks are not sensitive enough to pick up small amounts of protein! ACE inhibitors and ARBs have been shown to reduce these outcomes – even in normotensive diabetics. All diabetics, therefore, should be on an ACE inhibitor or ARB. Since this patient is hypertensive as well, she has all the more reason to begin a medication such as lisinopril. (It is, however, also important to note that the diagnosis of hypertension should not be made on the basis of a single blood pressure measurement in a physician's office. In this case, the question stem mentions that her vital signs have been similar to those recorded in the past, so making a diagnosis of hypertension is more acceptable. Additionally, diabetics have a target blood pressure of systolic less than 130 mm Hg, a more stringent guideline than for non-diabetic patients.) As an additional teaching point, every health maintenance visit with a diabetic patient should focus on the "Diabetic Five" – these five things, in this order: 1) Smoking cessation 2) Blood pressure control 3) Lipid control 4) Aspirin/metformin 5) Glucose control Adding hydrochlorothiazide (answer D) could improve her hypertension, and would be a reasonable first-line antihypertensive in a patient without diabetes. It is worth noting that this patient IS hypertensive – all diabetics should have a systolic blood pressure of less than 130. Still, diabetic patients have a compelling reason to begin antihypertensive therapy with ACE inhibitors or ARBs, however, due to the reno-protective effects of these drugs mentioned above. Either increasing glyburide (answer B) or increasing metformin (answer C) would be helpful for reducing the patient's blood sugar and better controlling her diabetes. However, her most recent Hgb A1c was not that bad – remember that the goal for diabetic patients is less than 7.0%. For more on Hgb A1c, including how to correlate the test result with the patient's average blood glucose, check out Diabetes Care 2002;25:275-8 or http://www.metrika.com/3medical/hemoglobin-m.html. Similarly, the patient's lipid profile is at her target LDL – thus, she does not need an increase in her statin (answer A). Under the ATP III guidelines, patients with diabetes are considered to have known coronary heart disease, and therefore have a target LDL of less than 100. To review the ATP III lipid guidelines at a glance, check out http://www.nhlbi.nih.gov/guidelines/cholesterol/atglance.pdf For an overview of diabetic nephropathy, try Ritz E., Orth S. R. Primary Care: Nephropathy in Patients with Diabetes Mellitus. N Engl J Med 1999; 341:1127-1133, Oct 7, 1999. http://content.nejm.org/cgi/reprint/341/15/1127.pdf?hits=20&where=fulltext&andorexactfulltext=and&searchterm=diabetes&sortspec=Score%2Bdesc%2BPUBDATE_SORTDATE%2Bdesc&excludeflag=TWEEK_element&searchid=1&FIRSTINDEX=0&resourcetype=HWCIT

This question is not currently linked to the learning objective database.

Question problem?

Question # 2

Select the single best answer to the numbered question.

A 55 year old female comes to the emergency department complaining of a headache for the past six hours. Her headache began abruptly after she finished eating breakfast, and quickly increased to 8/10 throbbing pain located mainly over her right temple. The pain has been associated with mild nausea but no vomiting. She denies chronic or recurrent headaches, but did have one headache similar to this one two weeks ago, which resolved after taking ibuprofen and lying in a quiet, dark room. She has smoked one pack of cigarettes daily for 38 years. On physical exam, the patient has temperature of 37.0 C (98.6 F), pulse of 99, and BP 147/95. Neurological examination is nonfocal, but mild photophobia and nuchal rigidity are noted. Fundoscopic examination reveals no papilledema. Skin exam shows no lesions. CT of the head, obtained without contrast, reveals no abnormalities. What is the most appropriate next step in the management of this patient?

A. Obtain head CT with contrast
B. Lumbar puncture
C. Administer i.m. sumatriptan
D. Administer oral ibuprofen
E. Administer i.v. ceftriaxone

You did not answer this question.

Explanations:

A. CT with contrast (answer A) might help you identify a space-occupying lesion, such as a tumor - which can certainly be a cause of headache. However, such lesions are not likely to cause a sudden onset headache such as the one described in the question stem. Remember that CT without contrast is the best study to identify an acute head bleed, whether you're looking for a stroke, a hematoma, or a hemorrhage.

B. No explanation provided.

C. Sumatriptan injection (answer C) would be a fine choice if the patient were having a migraine. Migraine headache pain does lateralize in 60-70% of patients, may be associated with photophobia or nausea, and be relieved by NSAIDs or lying in a dark room. However, most patients with migraine have their first symptoms in adolescence or early adulthood. A new-onset migraine in a patient 55 years old would be very unusual. (It is important to recognize that the patient's headache two weeks ago was likely a "sentinel bleed" from an aneurysm.) Additionally, migraines are frequently associated with sensory "auras" such as visual scotomata or paresthesias.

D. Ibuprofen (answer D) is a reasonable first-line treatment for a patient with a tension headache. These headaches are typically bilateral, and would seldom become intense enough to necessitate a visit to the ER.

E. Ceftriaxone (answer E) is the most commonly used empiric antibiotic for adults with suspected bacterial meningitis since it penetrates the CSF and covers the most likely pathogens in this age group: S. pneumoniae (60%) and N. meningitides (20%). This patient is afebrile, however, and her stiff neck and photophobia are simply signs of meningeal irritation – which in this case is caused by SAH, not a bacterial infection. Meningitis may cause headache, but not as suddenly as the headache described in the question. Even if this patient was suspected to have meningitis, you would likely want to obtain an LP before giving antibiotics so that you don't sterilize the cultures! After obtaining an LP, antibiotic therapy with ceftriaxone (or cefotaxime) and vancomycin could be started. (The latter is used to cover the possibility of drug-resistant S. pneumoniae while you await results of the CSF culture.)

This question is not currently linked to the learning objective database.

Question problem?

Question # 3

Select the single best answer to the numbered question.

A four week old male infant is brought by his mother to the physician following one week of emesis. The patient's mother states that the patient has been vomiting non-bilious material immediately after each feeding, but then becomes fussy and demands to be fed again. She denies ever seeing any blood in the emesis. Over the past two or three days, the infant's vomiting has become increasingly sudden and forceful. The child is irritable, with few tears. The oropharynx is dry, the infant's fontanelles appear sunken, and moderate skin tenting is noted. Capillary refill is approximately 2 seconds. On abdominal exam, visible peristaltic waves are observed, and a 1cm firm mass is palpated in the right upper quadrant. What is the most likely laboratory finding?

A. Na+ 130, K+ 2.9, Cl- 89, HCO3- 35
B. Na+ 138, K+ 3.8, Cl- 100, HCO3- 26

B. [illegible]
C. Na+ 150, K+ 4.0, Cl- 100, HCO3- 24
D. Na+ 140, K+ 3.8, Cl- 100, HCO3- 15
E. Na+ 130, K+ 5.8, Cl- 110, HCO3- 20

You did not answer this question.

Explanations:

A. Key teaching point #1: When you see a 3-6 week old infant with projectile vomiting, and an olive-like mass on physical exam, think pyloric stenosis! Key teaching point #2: Losing HCl in emesis leads to a hypochloremic, hypokalemic metabolic alkalosis. This vignette describes a classic presentation of pyloric stenosis, a commonly-tested disorder for which the associated electrolyte abnormality is a hypochloremic, hypokalemic metabolic alkalosis (answer A). Even without recognizing that this is a case of pyloric stenosis, you should be able to think through the question and arrive at the correct answer. Think about what is going on with this patient. Repetitive vomiting of acidic gastric juices causes the loss of HCl – thus, the patient's Cl- should be low. Since you're losing H+, there will also be a metabolic alkalosis, so the HCO3- will be elevated as well. Finally, in order to maintain pH balance, the kidneys avidly reabsorb H+, but they can only do this at the expense of K+, resulting in hypokalemia. (Review Dr. Barrett's diagrams of the principal and intercalated cells of the cortical collecting duct from her second year "Diuretics" lecture to remind yourself of how this happens physiologically. http://www.med-ed.virginia.edu/courseSites/subjects.cfm?CID=1) Thinking about things this way, you're looking for the answer that shows a decrease in Cl- and K+, and an increase in HCO3- (answer A). As previously mentioned, the question stem describes a truly classic case of pyloric stenosis, which is important to recognize. Key features include: 1) palpable abdominal mass – often described as an "olive" (Sometimes, the olive is only palpable after feedings, so doing a test feeding in clinic can confirm the diagnosis.); 2) projectile vomiting; 3) vomiting immediately after feeding, then demanding to be refed - "hungry vomiters"; 4) presentation at age 3-6 weeks. The initial test of choice when the diagnosis is unclear is abdominal ultrasound, which will reveal the hypertrophied pylorus. (Barium studies may also be used, but these have the undesirable side effect of exposing an infant to radiation.) The treatment of pyloric stenosis is, of course, surgical – the patient needs a pyloromyotomy. Answer B represents essentially normal lab values, which this child would not be expected to demonstrate given his dehydration and one week history of vomiting. The primary abnormality in answer C is hypernatremia. This situation results when a patient has hypotonic fluid loss, or loses more water than they do sodium – as can happen in patients with dehydration with inadequate free water intake. It is important to realize that this patient is not losing hypotonic fluids, however – he is losing large amounts of electrolytes with each episode of vomiting. The primary abnormality in Answer D is a low bicarbonate – which, in this situation, represents an anion gap metabolic acidosis. Remember that Anion gap = sodium – chloride – bicarbonate = 12 +/- 4 The anion gap in this question stem would be 140 – 100 – 16 = 24, which is elevated. Possible etiologies of an anion gap metabolic acidosis include the things on the MUDPILES mnemonic – none of which would apply to this patient. For an eMedicine article on metabolic acidosis (including the MUDPILES mnemonic) check out: http://www.emedicine.com/ped/topic15.htm Answer E represents a patient with hyponatremia and hyperkalemia. This situation could arise in adrenal insufficiency due to insufficient aldosterone.

B. Answer B represents essentially normal lab values, which this child would not be expected to demonstrate given his dehydration and one week history of vomiting.

C. The primary abnormality in answer C is hypernatremia. This situation results when a patient has hypotonic fluid loss, or loses more water than they do sodium – as can happen in patients with dehydration with inadequate free water intake. It is important to realize that this patient is not losing hypotonic fluids, however – he is losing large amounts of electrolytes with each episode of vomiting.

D. The primary abnormality in Answer D is a low bicarbonate – which, in this situation, represents an anion gap metabolic acidosis. Remember that Anion gap = sodium – chloride – bicarbonate = 12 +/- 4 The anion gap in this question stem would be 140 – 100 – 16 = 24, which is elevated. Possible etiologies of an anion gap metabolic acidosis include the things on the MUDPILES mnemonic – none of which would apply to this patient. For an eMedicine article on metabolic acidosis (including the MUDPILES mnemonic) check out: http://www.emedicine.com/ped/topic15.htm

E. Answer E represents a patient with hyponatremia and hyperkalemia. This situation could arise in adrenal insufficiency due to insufficient aldosterone.

This question is not currently linked to the learning objective database.

Question problem?

Question # 4

Select the single best answer to the numbered question.

A 30 year old female presents to her physician with a breast mass. She first noted a small "lump" in her left breast while showering about six weeks ago. She has noted no change in the size of the mass since that time, and she denies pain or nipple discharge. Family history is significant for a paternal grandmother who had breast cancer at age 79. Physical examination reveals a soft, round, mobile 1cm mass in the lower outer quadrant of the left breast. No skin changes are noted. What is the most appropriate next step in the management of this patient?

A. Mammography
B. Refer the patient for radical mastectomy
C. Begin levonorgestrel/etinyl estradiol
D. Genetic testing for BRCA1 and BRCA2
E. Ultrasound of breast mass

You did not answer this question.

Explanations:

A. Mammograms are not the best diagnostic study in a woman under 35 years old due to the density of breast tissue.

B. Answer B might be an appropriate treatment for some breast cancers. But to make a diagnosis of cancer requires tissue, so referral for mastectomy would certainly not be appropriate at this time.

C. Many oral contraceptive pills (OCPs) contain combinations of levonorgestrel and ethinyl estradiol (answer C). There are some non-contraceptive indications for prescribing OCPs, such as dysmenorrhea or endometriosis, and some practicioners do prescribe OCPs to help reduce the pain caused by hormonal fluctuations of benign breast cysts. This patient does not complain of pain, however, and more importantly, you do not yet know what her breast mass is. While not all breast masses are pathological, they all demand an explanation. Since this patient is less than 35, she needs ultrasound.

D. Genetic testing for BRCA1 and BRCA2 (answer D) might be an appropriate step for an asymptomatic woman with a strong family history of breast cancer, or to evaluate ovarian cancer risk in a patient with proven breast cancer. There are two reasons why this response is incorrect for this question, however. First, this patient's family history of breast cancer is not terribly impressive. Breast cancer is so common that 12% of all women will have a positive family history. To identify patients at risk for BRCA1 and BRCA2 mutations, look for patients with multiple first-degree relatives (mothers, daughters, or sisters) who had breast or ovarian cancer at young ages (less than 40-50). In general, the USMLE tests the most classic presentation of any disease – for the correct answer to be BRCA1/BRCA2 testing, the question stem would have to give you more than just one relative who had breast cancer at age 79. Second, and more important, even if this patient had a family history that was suggestive for a BRCA1 or BRCA2 mutation, her problem right now is a breast mass that needs to be evaluated with imaging and potentially a biopsy. Genetic testing might help you assess the patient's lifetime breast cancer risk, but it will not help you determine if this particular breast mass is something bad or not.

E. Key teaching point: Mammogram is the preferred imaging study for women over 35, while women younger than 35 should get ultrasound to evaluate a breast mass. In women younger than 35, the breast tissue is often too dense to evaluate mammographically, and the incidence of breast cancer younger women is still very low. Studies have shown that routine mammography is not cost-effective nor clinically beneficial for younger women unless there is a high suspicion of cancer by clinical examination. The clinical characteristics of this mass are non-suspicious for malignancy, though. Features commonly associated with malignancy include hard, irregularly-shaped, immobile masses >2cm in size. Since this patient's mass is soft, small, rounded, and mobile, it is likely to be a benign fibroadenoma. Thus, the patient should receive ultrasound, and the correct answer is E. If the patient were over 35, she should get a diagnostic mammogram (answer A). Questions about the workup of a breast mass are common on the USMLE, so it is worth thinking about how the workup would proceed in this patient. If ultrasound were obtained and the mass is found to be cystic in nature, it is very unlikely to be malignant, and no further evaluation is necessary (although the cyst may be aspirated if it is causing the patient pain). If it is solid, however, tissue will be required to make a definitive diagnosis, and the patient should receive a biopsy. Since breast tissue may undergo changes related to the menstrual cycle, many clinicians will simply ask a young patient with a breast mass to return 3-10 days following her next menstrual cycle to re-evaluate the lump. Answer B might be an appropriate treatment for some breast cancers. But to make a diagnosis of cancer requires tissue, so referral for mastectomy would certainly not be appropriate at this time. Many oral contraceptive pills (OCPs) contain combinations of levonorgestrel and ethinyl estradiol (answer C). There are some non-contraceptive indications for prescribing OCPs, such as dysmenorrhea or endometriosis, and some practicioners do prescribe OCPs to help reduce the pain caused by hormonal fluctuations of benign breast cysts. This patient does not complain of pain, however, and more importantly, you do not yet know what her breast mass is. While not all breast masses are pathological, they all demand an explanation. Since this patient is less than 35, she needs ultrasound. Genetic testing for BRCA1 and BRCA2 (answer D) might be an appropriate step for an asymptomatic woman with a strong family history of breast cancer, or to evaluate ovarian cancer risk in a patient with proven breast cancer. There are two reasons why this response is incorrect for this question, however. First, this patient's family history of breast cancer is not terribly impressive. Breast cancer is so common that 12% of all women will have a positive family history. To identify patients at risk for BRCA1 and BRCA2 mutations, look for patients with multiple first-degree relatives (mothers, daughters, or sisters) who had breast or ovarian cancer at young ages (less than 40-50). In general, the USMLE tests the most classic presentation of any disease – for the correct answer to be BRCA1/BRCA2 testing, the question stem would have to give you more than just one relative who had breast cancer at age 79. Second, and more important, even if this patient had a family history that was suggestive for a BRCA1 or BRCA2 mutation, her problem right now is a breast mass that needs to be evaluated with imaging and potentially a biopsy. Genetic testing might help you assess the patient's lifetime breast cancer risk, but it will not help you determine if this particular breast mass is something bad or not.

This question is not currently linked to the learning objective database.

Question problem?

Question # 5

Select the single best answer to the numbered question.

An otherwise healthy 8 year old girl presents with two weeks of perianal pruritis. She has two younger brothers, one of whom has had similar complaints for the past few days. Physical exam reveals perianal erythema with mild excoriations. The "scotch tape test" reveals several bean-shaped white eggs. What is the most likely diagnosis in this patient?

A. Trichuriasis
B. Enterobiasis
C. Child abuse
D. Fecal soilage
E. Atopic dermatitis

You did not answer this question.

Explanations:

A. [illegible]

A. Whipworm or trichuriasis (choice A) is a common intestinal helminthic infection worldwide, with the highest prevalence in tropical regions. Hosts are usually asymptomatic, though the disease can cause loose stools that contain mucus or blood, resulting in a secondary anemia. Trichuriasis is also classically associated with rectal prolapse in a patient with a heavy parasite load. Heavy loads can affect a child's growth and cognition. Diagnosis is made by stool examination for eggs, which are barrel shaped with a hyaline plug at each end.

B. This is a classic case of enterobiasis (answer B) or "pinworm." The most common presenting symptom is intense anal itching or pruritus ani. Other symptoms (such as abdominal pain/fullness or nausea and vomiting) may occur if the worm burden is high. Girls may also present with a vulvovaginitis or urinary tract infection if the worms migrate. Eosinophilic enterocolitis and appendicitis are rarer complications. Some teaching points: 1) The first line treatment of enterobiasis is either mebendazole or albendazole - one dose is usually sufficient, but a second dose 1-2 weeks later can help prevent reinfection. Pyrantel pamoate is second line because of its side effects (nausea, vomiting, abdominal cramping, neurotoxicity, and elevated LFTs). However, pyrantel pamoate is first line if the patient is pregnant, since mebendazole and albendazole are teratogenic. 2) Enterobiasis is spready by a fecal-oral route and usually presents in kids ages 5-10 years old. It is very uncommon in children less than 2 years old. 3) The "scotch tape" test is the best way to confirm the diagnosis. It involves covering a wooden stick with scotch tape with the sticky side facing outward, and then pressing the tape against the perianal skin. Bean-shaped eggs stick to the tape which can then be visualized under a microscope. Sometimes whole adult worms can be found perianally - they are white, pin-shaped and can be up to 13 mm long. 4) Treatment should include simultaneous treatment of all household members to prevent reinfection. Bedding and clothing should be washed, fingernails should be clipped (since this is the most common place for eggs to hide out), and hygienic measures increased (handwashing and bathing). Whipworm or trichuriasis (choice A) is a common intestinal helminthic infection worldwide, with the highest prevalence in tropical regions. Hosts are usually asymptomatic, though the disease can cause loose stools that contain mucus or blood, resulting in a secondary anemia. Trichuriasis is also classically associated with rectal prolapse in a patient with a heavy parasite load. Heavy loads can affect a child's growth and cognition. Diagnosis is made by stool examination for eggs, which are barrel shaped with a hyaline plug at each end. Child abuse (choice C) is not the best answer, though it isn't unreasonable to have this on your differential when children present with genital or anal complaints. In this case, we have a clear, identifiable cause of the perianal itching. Fecal soilage (choice D) is part of the differential of anal pruritis and can be due to any cause of diarrhea or loose stools. Some patients have an abnormality with internal anal sphincter relaxation. When anal itching is otherwise unexplained, this diagnosis should be entertained. Eczema or atopic dermatitis (choice E) is certainly part of the differential diagnosis of anal pruritis, though just the history does suggest an infectious cause. This familial allergic reaction is often associated with asthma and allergic rhinitis. The rash of eczema is intensely pruritic, and usually appears as erythematous patches with scaling. In children, eczema usually occurs on the face, scalp, extremities, diaper area and trunk, and usually presents by age 7.

C. Child abuse (choice C) is not the best answer, though it isn't unreasonable to have this on your differential when children present with genital or anal complaints. In this case, we have a clear, identifiable cause of the perianal itching.

D. Fecal soilage (choice D) is part of the differential of anal pruritis and can be due to any cause of diarrhea or loose stools. Some patients have an abnormality with internal anal sphincter relaxation. When anal itching is otherwise unexplained, this diagnosis should be entertained.

E. Eczema or atopic dermatitis (choice E) is certainly part of the differential diagnosis of anal pruritis, though just the history does suggest an infectious cause. This familial allergic reaction is often associated with asthma and allergic rhinitis. The rash of eczema is intensely pruritic, and usually appears as erythematous patches with scaling. In children, eczema usually occurs on the face, scalp, extremities, diaper area and trunk, and usually presents by age 7.

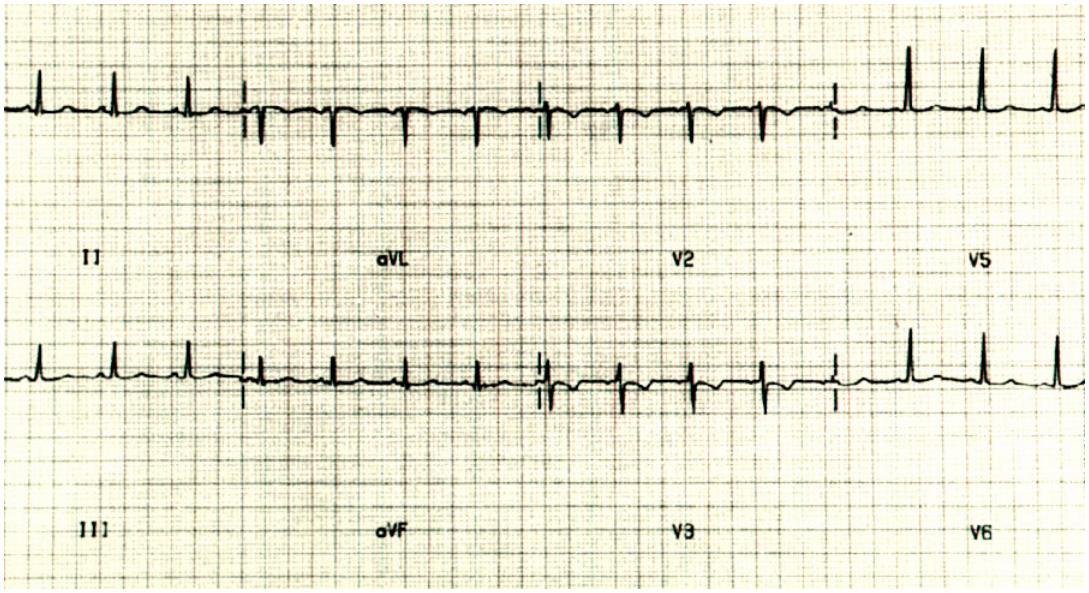
This question is not currently linked to the learning objective database.

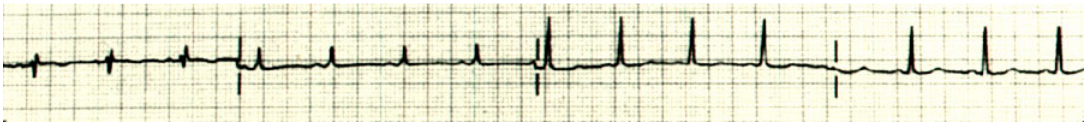
Question problem?

Question # 6

Select the single best answer to the numbered question.

A 66 year old male presents to the emergency department with chest pain. The pain began two hours ago as the patient was watching television. The pain is described as "squeezing" and is located primarily substernally with radiation to the jaw. Past medical history includes diabetes mellitus, hypertension, hyperlipidemia, and a 50 pack/year smoking habit. On physical exam, the patient appears anxious and diaphoretic. The patient is given supplemental oxygen by nasal cannula, and aspirin, morphine, and nitroglycerin are administered. EKG obtained on presentation to the ED is shown. Of the following, which is the most appropriate study to obtain next?





A. Aortogram
B. Troponin I
C. Stress echo
D. Exercise stress test
E. CT angiogram of chest

You did not answer this question.

Explanations:

A. An aortogram (answer A) used to be the gold standard for the diagnosis of an aortic aneurysm. Now, less-invasive imaging modalities such as CT, MRI, and echo have largely supplanted aortograms for the diagnosis of aortic dissection. In any case, while aortic dissection ought to be considered in patients with chest pain, this patient's history is strongly suggestive of ACS, and this should be investigated first.

B. Key teaching point: To evaluate acute coronary syndrome, you need both an EKG and serial troponins. The patient presents with symptoms highly suggestive of acute coronary syndrome (ACS). Collectively, ACS consists of three ischemic cardiac disorders: ST-elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI), and unstable angina. Distinguishing between these entities is important, as both the therapy and the prognosis depends on the diagnosis. The first thing to do when sorting out ACS is to check an EKG – the presence of ST segment elevation (>1 mm in two or more anatomically contiguous leads) confirms the diagnosis of a STEMI. STEMI carries the worst prognosis of any of any of the acute coronary syndromes, so finding "tombstones" of ST elevation on EKG is a clear indication for immediate reperfusion therapy, either with thrombolytics or percutaneous interventions in the cath lab. This patient's EKG shows little more than normal sinus rhythm. The lack of ST elevation means that the diagnosis of STEMI is ruled out. (As a sidenote: If you answered this question incorrectly because you thought that there was miniscule ST elevation on this EKG, here is the teaching point for you. When you are looking at an image on the USMLE or shelf exam, whether that image is a CT scan, a radiograph, or an EKG, expect any abnormalities to be STRIKING. The testmakers do not expect you to be a radiologist or to hold your EKG calipers up to the computer monitor. You need to use the information from the question stem to know whether you're looking for a fracture, a head bleed, atrial fibrillation, etc., but so long as you know what to look for, the finding in the image will either be obvious - or it will be absent.) So now that the patient has a relatively normal EKG, how do we distinguish between the other acute coronary syndromes - NSTEMI and unstable angina? You need a biomarker to tell you if the patient's cardiac tissue has infarcted, and although you could use CK-MB or LDH, the most sensitive and specific test is cardiac troponins (answer B). If this patient's troponin I were elevated, it would indicate ischemic necrosis of the myocardium - therefore, the patient is having an MI, even if we don't see it on EKG. The appropriate diagnosis would thus be NSTEMI. (Pathologically, to have ST segment elevation on EKG requires a full-thickness infarction of the myocardium. If an infarction does not penetrate the entire thickness of the heart wall, the heart's electrical conduction is relatively undisturbed and there will not be diagnostic findings on EKG.) The management of NSTEMI is still evolving and remains somewhat controversial. Reperfusion with primary percutaneous interventions in the cath lab is beneficial for high-risk patients, but thrombolytic therapy carries greater risks and is reserved for those patients with persisting ST elevations. Many patients with NSTEMI can also be managed medically with antiplatelet therapy and beta blockers. If the patient's troponin I were not elevated, then the patient has not yet infarcted his myocardium. The diagnosis would therefore be unstable angina, which requires intensive medical management to prevent MI. The patient should be started on some regimen of heparin, beta blockers, and antiplatelet therapy. The patient should also receive EKG monitoring and serial troponins to ensure that his unstable angina does not eventually cause infarction. An aortogram (answer A) used to be the gold standard for the diagnosis of an aortic aneurysm. Now, less-invasive imaging modalities such as CT, MRI, and echo have largely supplanted aortograms for the diagnosis of aortic dissection. In any case, while aortic dissection ought to be considered in patients with chest pain, this patient's history is strongly suggestive of ACS, and this should be investigated first. A stress echocardiogram (answer C) or exercise stress test (answer D) might be good tests to risk stratify a medically stable, low-risk patient with suspected coronary artery disease. This patient, however, requires further evaluation to rule out MI, and stressing the heart is a bad idea in a patient with probable ongoing ischemic pain. Obtaining a CT pulmonary angiogram (answer E) would be a great test to rule out pulmonary embolism, which can be a cause of chest pain. Here, though, the patient's history and cardiac risk factors strongly suggest a cardiac etiology for his pain, so until that is ruled out, searching for a PE is inappropriate.

C. A stress echocardiogram (answer C) or exercise stress test (answer D) might be good tests to risk stratify a medically stable, low-risk patient with suspected coronary artery disease. This patient, however, requires further evaluation to rule out MI, and stressing the heart is a bad idea in a patient with probable ongoing ischemic pain.

D. A stress echocardiogram (answer C) or exercise stress test (answer D) might be good tests to risk stratify a medically stable, low-risk patient with suspected coronary artery disease. This patient, however, requires further evaluation to rule out MI, and stressing the heart is a bad idea in a patient with probable ongoing ischemic pain.

E. Obtaining a CT pulmonary angiogram (answer E) would be a great test to rule out pulmonary embolism, which can be a cause of chest pain. Here, though, the patient's history and cardiac risk factors strongly suggest a cardiac etiology for his pain, so until that is ruled out, searching for a PE is inappropriate.

This question is not currently linked to the learning objective database.

Question problem?

Question # 7

Select the single best answer to the numbered question.

A 73 year old male presents to his physician complaining of cough and fatigue. His cough began 6 months ago and has steadily worsened, and is now associated with occasional expectoration of mucus streaked with bright red blood. Patient has also noted worsening dyspnea on exertion and a weight loss of 15 lbs. The patient has smoked a pack and a half of cigarettes every day for the past 60 years. Past medical history is significant for bipolar disorder treated with lithium. Physical exam shows unilateral localized wheezing on the left chest and clubbing of the distal extremities. Capillary refill is brisk. No skin tenting is observed. Chest X-ray is obtained, which shows a large mass at the left hilum. Labs show: Glucose 130, Na+ 125, K+ 4.0, Cl- 91, HCO3- 25, BUN 15, Creatinine 1.0; Plasma osmolality 270 (Normal: 282-295 mOsm/kg); Urine osmolality 650 mOsm/kg (Normal: 50 - 1400 mOsm/kg). Which of the following is the most likely mechanism for this patient's hyponatremia?

A. Increased oral intake of hypotonic fluids
B. Decreased oral intake of solutes
C. Impaired secretion of ADH in the posterior pituitary
D. Ectopic overproduction of vasopressin
E. Resistance to ADH action on the cortical and medullary collecting tubules

You did not answer this question.

Explanations:

A. Increased free water intake (answer A) describes the situation seen in psychogenic polydipsia, a disorder in which patients - most commonly schizophrenics receiving antipsychotics - drink excessive amounts of free water. The kidneys have an incredible capacity for regulating fluids and solutes. Normal patients can excrete more than 10-15 L of urine per day, so for a patient with a dietary solute intake to become hyponatremic, they would have to have a very impressive water intake!

B. Decreased solute intake (answer B) is the mechanism for "beer potomania," in which patients drink excessive amounts of beer without taking in enough food to provide adequate solutes. The minimum urine osmolality is 60mOsm/kg, so there is a certain amount of obligatory solute lost in the urine every day. If a patient is only drinking beer (and massive quantities of it) and not taking in any solutes, he can deplete his solute reserves over time just through this obligatory solute loss.

C. Answers C and E describe the abnormalities present in diabetes insipidus (DI). DI is a frequently-tested topic on the USMLE, but it is most likely to present with HYPERnatremia, not HYPOnatremia. There are two types of DI: central and nephrogenic. In central DI (answer C), there is not enough ADH produced by the pituitary, while in nephrogenic DI (answer E), there is end-organ resistance to ADH action. In either case, the kidney is no longer able to reabsorb H2O from the urine, leading to free water losses and copious polyuria. Since the body is losing more free water than solute, patients typically become hypernatremic. In children, nephrogenic DI is almost always inherited, while in adults it is commonly caused by drugs - most notably lithium. Central DI is most commonly caused by surgical trauma to the hypothalamus or pituitary, such as might occur with a trans-sphenoidal removal of a pituitary adenoma.

D. Key teaching point: When you see euvolemic hyponatremia (especially on the USMLE), think SIADH! The constellation of findings in this patient, including euvolemic hyponatremia, hypotonicity (plasma osmolality <280 mOsms), inappropriately concentrated urine, and normal renal function are diagnostic for the syndrome of inappropriate ADH secretion (SIADH). SIADH has a number of causes. The most common include: 1) Cancer - ectopic production of ADH by small cell lung cancers or pancreatic malignancies; 2) CNS disturbances - traumatic injuries, strokes, infections, and hemorrhages can all cause increased ADH production; 3) Drugs - most commonly caused by antipsychotic medications or chemotherapeutics, but the old (and seldom used) insulin secretagogue chlorpropamide is a commonly-tested causative agent. This question stem is loaded with findings consistent with primary lung carcinoma in general and small cell carcinoma in particular: a older patient with an extensive smoking history; insidious onset of cough, fatigue, and weight loss; localized wheezing and clubbing of the nails; and a chest x-ray showing a central mass. Therefore, it is overwhelmingly likely that overproduction of ADH – also known as vasopressin - by the patient's lung tumor is causing his hyponatremia (answer D). Both answers A and B are plausible explanations for hyponatremia in general. However, as mentioned above, this patient's history and physical exam strongly suggest lung cancer, which is associated with SIADH. Additionally, either of these situations would be associated with a maximally diluted urine osmolality, while the patient in the question stem has inappropriately concentrated urine. Increased free water intake (answer A) describes the situation seen in psychogenic polydipsia, a disorder in which patients - most commonly schizophrenics receiving antipsychotics - drink excessive amounts of free water. The kidneys have an incredible capacity for regulating fluids and solutes. Normal patients can excrete more than 10-15 L of urine per day, so for a patient with a dietary solute intake to become hyponatremic, they would have to have a very impressive water intake! Decreased solute intake (answer B) is the mechanism for "beer potomania," in which patients drink excessive amounts of beer without taking in enough food to provide adequate solutes. The minimum urine osmolality is 60mOsm/kg, so there is a certain amount of obligatory solute lost in the urine every day. If a patient is only drinking beer (and massive quantities of it) and not taking in any solutes, he can deplete his solute reserves over time just through this obligatory solute loss. Answers C and E describe the abnormalities present in diabetes insipidus (DI). DI is a frequently-tested topic on the USMLE, but it is most likely to present with HYPERnatremia, not HYPOnatremia. There are two types of DI: central and nephrogenic. In central DI (answer C), there is not enough ADH produced by the pituitary, while in nephrogenic DI (answer E), there is end-organ resistance to ADH action. In either case, the kidney is no longer able to reabsorb H2O from the urine, leading to free water losses and copious polyuria. Since the body is losing more free water than solute, patients typically become hypernatremic. In children, nephrogenic DI is almost always inherited, while in adults it is commonly caused by drugs - most notably lithium. Central DI is most commonly caused by surgical trauma to the hypothalamus or pituitary, such as might occur with a trans-sphenoidal removal of a pituitary adenoma.

E. Answers C and E describe the abnormalities present in diabetes insipidus (DI). DI is a frequently-tested topic on the USMLE, but it is most likely to present with HYPERnatremia, not HYPOnatremia. There are two types of DI: central and nephrogenic. In central DI (answer C), there is not enough ADH produced by the pituitary, while in nephrogenic DI (answer E), there is end-organ resistance to ADH action. In either case, the kidney is no longer able to reabsorb H2O from the urine, leading to free water losses and copious polyuria. Since the body is losing more free water than solute, patients typically become hypernatremic. In children, nephrogenic DI is almost always inherited, while in adults it is commonly caused by drugs - most notably lithium. Central DI is most commonly caused by surgical trauma to the hypothalamus or pituitary, such as might occur with a trans-sphenoidal removal of a pituitary adenoma.

This question is not currently linked to the learning objective database.

Question problem?

Question # 8

Select the single best answer to the numbered question.

A 25 year old student presents with three days of vulvar pruritis. She has had a total of six lifetime partners, and is currently sexually active with one partner and states that they "occasionally" use condoms. One year ago, she was successfully treated for a Chlamydia infection. The patient denies vaginal odor or increased vaginal discharge. Physical exam reveals slight vulvar erythema, and speculum exam shows moderate clumpy white discharge. Vaginal discharge pH is 4.0. Whiff test is negative. Wet mount results: KOH prep shows occasional budding yeast and hyphae. Saline prep shows 3 WBCs, occasional squamous cells, and no bacteria. What is the next best step in the management of this patient?

A. Clotrimazole vaginal cream
B. Azithromycin and recommend that her partner see a physician for Chlamydia screening and treatment
C. Metronidazole
D. Ceftriaxone
E. Reassurance and follow up in two weeks

You did not answer this question.

Explanations:

A. This is a classic case of vulvovaginal candidiasis, which is usually treated with a single dose of oral fluconazole or several days of miconazole or clotrimazole vaginal creams (answer A). A handful of additional teaching points… 1) A key clue to the diagnosis is the vulvar pruiritis, which is often the dominant feature of a yeast infection (and may be the only complaint from the patient). Other symptoms could include dysuria, vulvovaginal irritation, or dyspareunia. Classically, the discharge of a yeast infection is described as white with a curd-like consistency (often described as "cottage cheese" like), but many real-life patients will not complain of increased discharge. 2) The low pH (<4.5) of the vaginal discharge points to yeast; the other two main causes of vaginitis (trichomoniasis and bacterial vaginosis) create more alkaline conditions with pH > 4.5. The diagnosis of candidiasis should be confirmed by finding budding yeast and hyphae on the 10% KOH wet mount. 3) This patient's yeast infection was uncomplicated, but it is important to recognize the difference between a complicated and an uncomplicated infection. Infections are uncomplicated when the patient experiences mild to moderate symptoms, the infections are sporadic, the suspected organism is Candida albicans, and the host is otherwise healthy. Complicated yeast infections are those with any of the following features: infection occurring in a host with pre-existing conditions (uncontrolled diabetes, immunosuppression, pregnancy), severe symptoms, infection with non-albicans species, or recurrent infections (>4 per year). Questions on the USMLE seldom give you all of the characteristic features of a particular type of vaginitis, so if you're interested in reading more, here's a succinct review of the diagnosis of vaginitis from the American Family Physician: http://www.aafp.org/afp/20000901/1095.html Re-infection with Chlamydia (choice B) is not the most likely diagnosis. Chlamydia causes cervicitis, but most women are asymptomatic: possible Chlamydia symptoms include vaginal discharge and poorly described lower abdominal pain. Metronidazole (choice C) is the treatment for the other forms of vaginitis - both bacterial vaginosis (BV) or trichomoniasis. BV and trichomoniasis can present with similar symptoms, though classically BV discharge is usually described as malodorous, thin, and grey-white, while trichomoniasis is described as purulent, green-grey, or frothy. The vaginal pH for both of BV and trichomoniasis is >4.5 and the whiff test is often positive in both. The saline wet mount for BV often shows increased numbers of cocobacilli and the classic "clue cells," which are vaginal epithelial cells studded by adherent coccobacilli around the cell's edges. A saline wet mount for trichomoniasis will show motile trichomonads. Administering a ceftriaxone injection (choice D) is a treatment for acute gonorrhea, whose prevalence is much lower than chlamydia. Gonorrhea can infect any portion of the female genital tract, but most often presents as a cervicitis, urethritis or proctitis. Female patients may complain of increased discharge, vaginal pruritis or symptoms of upper tract infection such as lower abdominal pain, and dyspareunia. On speculum exam, the cervix is often friable and cervical discharge may be seen. The gold standard for diagnosis is culture on Thayer-Martin medium. While the patient's symptoms point to a yeast vaginitis, STI testing in this patient is also prudent. Reassurance with follow-up (choice E) isn't the best answer. Treatment is indicated for the relief of symptoms. Up to 20% of sexually active women of reproductive age asymptomatically harbor candida; these patients do not require treatment.

B. Re-infection with Chlamydia (choice B) is not the most likely diagnosis. Chlamydia causes cervicitis, but most women are asymptomatic: possible Chlamydia symptoms include vaginal discharge and poorly described lower abdominal pain.

C. Metronidazole (choice C) is the treatment for the other forms of vaginitis - both bacterial vaginosis (BV) or trichomoniasis. BV and trichomoniasis can present with similar symptoms, though classically BV discharge is usually described as malodorous, thin, and grey-white, while trichomoniasis is described as purulent, green-grey, or frothy. The vaginal pH for both of BV and trichomoniasis is >4.5 and the whiff test is often positive in both. The saline wet mount for BV often shows increased numbers of cocobacilli and the classic "clue cells," which are vaginal epithelial cells studded by adherent coccobacilli around the cell's edges. A saline wet mount for trichomoniasis will show motile trichomonads.

D. Administering a ceftriaxone injection (choice D) is a treatment for acute gonorrhea, whose prevalence is much lower than chlamydia. Gonorrhea can infect any portion of the female genital tract, but most often presents as a cervicitis, urethritis or proctitis. Female patients may complain of increased discharge, vaginal pruritis or symptoms of upper tract infection such as lower abdominal pain, and dyspareunia. On speculum exam, the cervix is often friable and cervical discharge may be seen. The gold standard for diagnosis is culture on Thayer-Martin medium. While the patient's symptoms point to a yeast vaginitis, STI testing in this patient is also prudent.

E. Reassurance with follow-up (choice E) isn't the best answer. Treatment is indicated for the relief of symptoms. Up to 20% of sexually active women of reproductive age asymptomatically harbor candida; these patients do not require treatment.

This question is not currently linked to the learning objective database.

Question problem?

Question # 9

Select the single best answer to the numbered question.

A seven year old female is brought to the physician by her mother because of facial swelling and dark, cola-colored urine. These symptoms began abruptly two days ago and have been associated with anorexia and malaise. There have been no known sick contacts. Her mother states that the child is up to date with her immunizations and has been in good health except for a runny nose and sore throat around two weeks ago, which resolved after a few days without treatment. Vital signs are temperature 37.2 C (98.9 F), heart rate 95, and blood pressure of 148/86. There is diffuse edema of the lower extremities, face, and eyelids. Lungs and heart are clear to auscultation. Urinalysis shows moderate hematuria and proteinuria, and dysmorphic RBCs and occasional RBC casts are noted on microscopic examination. Based on these findings, what is the most likely diagnosis?

A. IgA nephropathy
B. Alport syndrome
C. Thin basement membrane nephropathy
D. Postinfectious glomerulonephritis
E. Henoch-Schonlein purpura

You did not answer this question.

Explanations:

A. IgA nephropathy (answer A) is the most common cause of primary glomerulonephritis. The most common presentation is recurrent episodes of gross hematuria that occur around 5 days after an upper respiratory infection. To truly confirm the diagnosis requires a renal biopsy. Although the disease is generally benign, some patients will progress to end-stage renal disease, and treatment with ACE inhibitors or ARBs may minimize glomerular injury and disease progression.

B. Alport syndrome (answer B) is yet another example of a disease that is rare in real life but common on examinations. The key features are glomerular hematuria along sensorineural deafness and ocular abnormalities. Since this is an inherited disease, there should also be a family history of renal failure and deafness. The primary defect is a genetic mutation in collagen type IV.

C. Thin basement membrane nephropathy (answer C) is a benign and usually hereditary disorder that is associated with glomerular hematuria. Although minimal to moderate proteinuria may be seen on occasion, having the degree of proteinuria described in the question stem would be distinctly unusual. Most often, these patients are completely asymptomatic, and their hematuria is only picked up on routine urinalysis.

D. This is a very typical presentation of postinfectious (or poststreptococcal) glomerulonephritis (answer D). Even if you recognized this diagnosis immediately based on the history, there may be some more subtle teaching points regarding the presentation of this disease or the interpretation microscopic urinalysis. First, the key features of post-streptococcal GN were all there. These include the patient's age (<7 years old), dark brown colored urine (representing hematuria), and periorbital and peripheral edema. There is also a latent period of around 10 days following pharyngitis before symptoms of glomerulonephritis occur, although for glomerulonephritis following streptococcal impetigo, the latent period can be as long as 3-4 weeks. One diagnostic finding not mentioned was the anti-streptolysin O titer, which you would expect to be positive (indicating recent exposure to Group A strep). Finally, the finding of RBC casts is an important one. When you see casts in the urine sediment - whether WBC, RBC, or granular - you have glomerular disease. This is a useful pearl to remember – it's only when cells get squeezed through the glomerulus that they will form casts. Dysmorphic RBCs (especially acanthocytes) are also highly suggestive of glomerular disease. Patients with bleeding from sources other than the glomerulus - such as a patient with renal stones, bladder cancer, or a urinary tract infection - should have RBCs with normal morphology, since those cells are not being squeezed from the glomerulus into the collecting tubule. IgA nephropathy (answer A) is the most common cause of primary glomerulonephritis. The most common presentation is recurrent episodes of gross hematuria that occur around 5 days after an upper respiratory infection. To truly confirm the diagnosis requires a renal biopsy. Although the disease is generally benign, some patients will progress to end-stage renal disease, and treatment with ACE inhibitors or ARBs may minimize glomerular injury and disease progression. Alport syndrome (answer B) is yet another example of a disease that is rare in real life but common on examinations. The key features are glomerular hematuria along sensorineural deafness and ocular abnormalities. Since this is an inherited disease, there should also be a family history of renal failure and deafness. The primary defect is a genetic mutation in collagen type IV. Thin basement membrane nephropathy (answer C) is a benign and usually hereditary disorder that is associated with glomerular hematuria. Although minimal to moderate proteinuria may be seen on occasion, having the degree of proteinuria described in the question stem would be distinctly unusual. Most often, these patients are completely asymptomatic, and their hematuria is only picked up on routine urinalysis. Henoch-Schonlein purpura (answer E) is a systemic vasculitis that commonly affects children. The classic triad of HSP is abdominal pain; a vasculitic, raised skin rash; and renal involvement. Questions that describe "palpable purpura" usually give away the diagnosis. The renal findings are variable, but include hematuria, proteinuria, and rising creatinine.

E. Henoch-Schonlein purpura (answer E) is a systemic vasculitis that commonly affects children. The classic triad of HSP is abdominal pain; a vasculitic, raised skin rash; and renal involvement. Questions that describe "palpable purpura" usually give away the diagnosis. The renal findings are variable, but include hematuria, proteinuria, and rising creatinine.

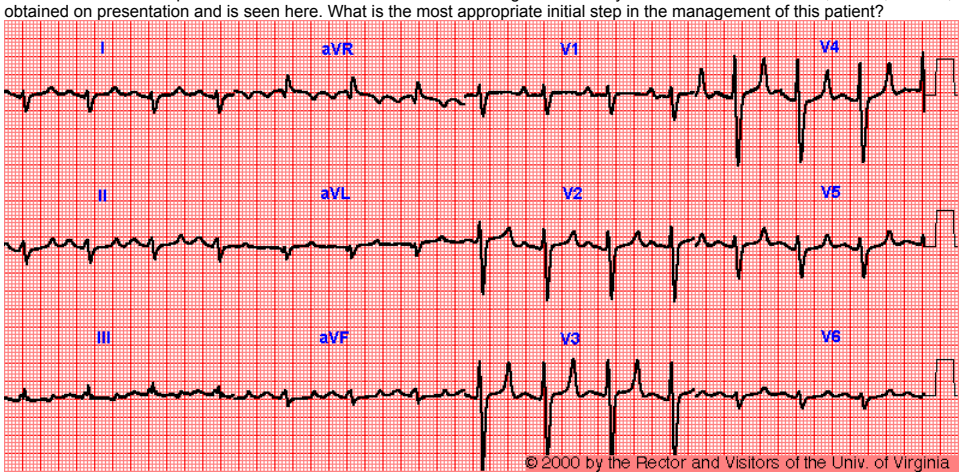
This question is not currently linked to the learning objective database.

Question problem?

Question # 10

Select the single best answer to the numbered question.

A 71 year old male comes to his physicians office complaining of muscle weakness. The weakness began insidiously several weeks ago, and has now progressed to the point where he has difficulty with normal activities such as brushing his hair or sitting unsupported. Past medical history is significant for hyperlipidemia, chronic renal insufficiency, coronary artery disease, and coronary artery bypass and grafting. Current medications include atorvastatin, fosinopril, metoprolol, aspirin, and spironolactone. Physical examination reveals diminished deep tendon reflexes and decreased motor strength. Laboratory evaluation shows Na+ 143, K+ 7.4, Cl- 101, HCO3- 28, BUN 30, and creatinine 1.8. EKG is obtained on presentation and is seen here. What is the most appropriate initial step in the management of this patient?



A. Administer i.v. insulin and glucose
B. Administer p.o. sodium polystyrene sulfate
C. Administer i.v. sodium bicarbonate
D. Administer i.v. calcium gluconate
E. Urgent hemodialysis

You did not answer this question.

Explanations:

A. Answers A, B, C, and E all are treatments for hyperkalemia – but none will provide immediate benefit for this patient, whose heart could slip into a terminal rhythm while you're working on correcting their electrolytes. The USMLE loves questions like this one that distinguish between the immediate treatment and the ultimate treatment for a condition. If you find yourself thinking that there is more than one correct response to a question, ask yourself which alternative provides the most immediate benefit. While insulin and glucose (answer A) or sodium bicarbonate (answer C) lower the serum potassium, they do so by causing a shift of potassium from the serum into cells, not by causing a net loss of total potassium. Sodium polystyrene sulfonate or Kayexelate (answer B) is a cation exchange resin that removes potassium from the gut. Its effects are not immediate. Hemodialysis (answer E) is an excellent treatment for hyperkalemia, but it is typically reserved for cases in which other methods have failed, or when there is ongoing release of potassium from injured cells (such as in a patient with severe myonecrosis).

B. Answers A, B, C, and E all are treatments for hyperkalemia – but none will provide immediate benefit for this patient, whose heart could slip into a terminal rhythm while you're working on correcting their electrolytes. The USMLE loves questions like this one that distinguish between the immediate treatment and the ultimate treatment for a condition. If you find yourself thinking that there is more than one correct response to a question, ask yourself which alternative provides the most immediate benefit. While insulin and glucose (answer A) or sodium bicarbonate (answer C) lower the serum potassium, they do so by causing a shift of potassium from the serum into cells, not by causing a net loss of total potassium. Sodium polystyrene sulfonate or Kayexelate (answer B) is a cation exchange resin that removes potassium from the gut. Its effects are not immediate. Hemodialysis (answer E) is an excellent treatment for hyperkalemia, but it is typically reserved for cases in which other methods have failed, or when there is ongoing release of potassium from injured cells (such as in a patient with severe myonecrosis).

C. Answers A, B, C, and E all are treatments for hyperkalemia – but none will provide immediate benefit for this patient, whose heart could slip into a terminal rhythm while you're working on correcting their electrolytes. The USMLE loves questions like this one that distinguish between the immediate treatment and the ultimate treatment for a condition. If you find yourself thinking that there is more than one correct response to a question, ask yourself which alternative provides the most immediate benefit. While insulin and glucose (answer A) or sodium bicarbonate (answer C) lower the serum potassium, they do so by causing a shift of potassium from the serum into cells, not by causing a net loss of total potassium. Sodium polystyrene sulfonate or Kayexelate (answer B) is a cation exchange resin that removes potassium from the gut. Its effects are not immediate. Hemodialysis (answer E) is an excellent treatment for hyperkalemia, but it is typically reserved for cases in which other methods have failed, or when there is ongoing release of potassium from injured cells (such as in a patient with severe myonecrosis).

D. Key teaching point: Hyperkalemia with EKG changes needs immediate treatment with i.v. calcium to protect the heart. Although this patient presents with a nonspecific complaint – weakness – evaluation reveals a serious underlying cause: hyperkalemia. Hyperkalemia causing EKG changes (such as the "peaked T" waves seen here) is a legitimate medical emergency, as the patient can progress to heart block or ventricular fibrillation very quickly. Thus, the most appropriate first step in management of this patient is the administration of calcium gluconate (answer D). Although this does not affect the serum K+ concentration, calcium does decrease cardiac membrane excitability and provide short-term cardioprotection from the membrane depolarizing effects of hyperkalemia. This buys you some time to work on correcting the underlying abnormality of high potassium. There are some other teaching points to be learned from this question stem as well. First, why is this patient hyperkalemic? First, he has some baseline kidney disease, which is exacerbated by the fact that his heart is likely not perfusing his kidneys as well as it might if he had no history of coronary artery disease. Remember that for a person eating a normal diet, the kidneys must excrete a certain amount of potassium each day to maintain homeostasis. Secondly, this patient's doctors likely also set him up to become hyperkalemic – he unfortunately had been taking two medications which can cause hyperkalemia: fosinopril, an ACE inhibitor, and spironolactone, a "potassium sparing" diuretic. Second, while this patient presented with weakness related to his hyperkalemia, most patients are not so "fortunate." Although muscle weakness progressing to flaccid paralysis can and does occur, most patients are asymptomatic, at least until their K+ levels get very high (above 7.0 or so). Third, it is important to recognize the classic EKG finding of hyperkalemia: the "peaked T wave." True EKG findings of hyperkalemia are not subtle. As one UVA attending says, a peaked T wave should be sharp enough to really hurt if you sat down on it. (Would you like to sit on the T wave in lead V4?) If you'd like a comprehensive online tutorial on EKG interpretation, try this one from the University of Wisconsin: http://www.fammed.wisc.edu/pcc/ecg/ecg.html Answers A, B, C, and E all are treatments for hyperkalemia – but none will provide immediate benefit for this patient, whose heart could slip into a terminal rhythm while you're working on correcting their electrolytes. The USMLE loves questions like this one that distinguish between the immediate treatment and the ultimate treatment for a condition. If you find yourself thinking that there is more than one correct response to a question, ask yourself which alternative provides the most immediate benefit. While insulin and glucose (answer A) or sodium bicarbonate (answer C) lower the serum potassium, they do so by causing a shift of potassium from the serum into cells, not by causing a net loss of total potassium. Sodium polystyrene sulfonate or Kayexelate (answer B) is a cation exchange resin that removes potassium from the gut. Its effects are not immediate. Hemodialysis (answer E) is an excellent treatment for hyperkalemia, but it is typically reserved for cases in which other methods have failed, or when there is ongoing release of potassium from injured cells (such as in a patient with severe myonecrosis).

E. Answers A, B, C, and E all are treatments for hyperkalemia – but none will provide immediate benefit for this patient, whose heart could slip into a terminal rhythm while you're working on correcting their electrolytes. The USMLE loves questions like this one that distinguish between the immediate treatment and the ultimate treatment for a condition. If you find yourself thinking that there is more than one correct response to a question, ask yourself which alternative provides the most immediate benefit. While insulin and glucose (answer A) or sodium bicarbonate (answer C) lower the serum potassium, they do so by causing a shift of potassium from the serum into cells, not by causing a net loss of total potassium. Sodium polystyrene sulfonate or Kayexelate (answer B) is a cation exchange resin that removes potassium from the gut. Its effects are not immediate. Hemodialysis (answer E) is an excellent treatment for hyperkalemia, but it is typically reserved for cases in which other methods have failed, or when there is ongoing release of potassium from injured cells (such as in a patient with severe myonecrosis).

This question is not currently linked to the learning objective database.

Question problem?

Question # 11

Select the single best answer to the numbered question.

A 30 year old female comes to her physician's office for a routine health examination. She has been in good health recently and is up to date with her gynecological examinations. Her only medication is loratadine for seasonal allergies and ibuprofen for occasional headaches. Physical examination shows temperature 37.1 C (98.8 F), pulse 80, BP 170/92, RR 14, oxygen saturation of 99% on room air. A 3/6 mid-systolic ejection murmur is present. Abdomen is nontender with a soft systolic-diastolic bruit that lateralizes to the left side. The patient is grossly intact neurologically, and fundoscopic examination shows sharp optic disc margins. Laboratory evaluation shows Na+ 141, K+ 3.9, Cl- 106, HCO3- 27, BUN 18, Cr 1.0, glucose 98. Urinalysis shows trace proteinuria and no casts. What is the most likely pathological mechanism causing this patient's hypertension?

A. Fibromuscular dysplasia
B. Oversecretion of aldosterone
C. Exogenous administration of corticosteroids
D. Atherosclerotic disease
E. Catecholamine-producing tumor

You did not answer this question.

Explanations:

A. This is a case of secondary hypertension caused by renal artery stenosis. They key clues are the markedly elevated blood pressure in an otherwise healthy young person and the presence of an abdominal bruit. In a patient without vascular risk factors, the stenosis is likely to be caused by fibromuscular dysplasia of the vessel wall (answer A) instead of atherosclerotic disease. Two quick teaching points: 1) This patient is a textbook example of fibromuscular dysplasia, which typically occurs in females under 50 years old. To confirm the diagnosis requires imaging – typically renal ultrasound with Doppler or CT or MR angiography. 2) Although the vast majority of patients with hypertension have essential hypertension, elevated blood pressure can be a sign of a more serious underlying disorder. You should always consider the diagnosis of secondary hypertension in patients who have a known onset of hypertension before age 30, patients whose blood

pressure remains elevated despite multiple medications, patients who have severe (>160/100 mm Hg) hypertension above age 55, or patients with a sudden increase in their blood pressure from a stable baseline. The remaining answer choices all are pathological mechanisms for other causes of secondary hypertension. Oversecretion of aldosterone (answer B) describes primary hyperaldosteronism or Conn's syndrome. Patients with Conn's syndrome should present with hypokalemia and hypertension. Exogenous administration of corticosteroids (answer C) is the most common cause of Cushing's syndrome, which is another cause of secondary hypertension. You should know the key features of Cushing's, which include obesity, bone loss, glucose intolerance, moon facies, a "buffalo hump," purple striae, and hypertension. Atherosclerotic disease (answer D) is certainly a cause of renal artery stenosis, and would explain her abdominal bruit. The tip off in this question was that the patient was a young, previously-healthy woman - just the kind of patient who gets fibromuscular dysplasia. Atherosclerotic plaques are more likely to cause stenosis in older patients with vascular risk factors such as smoking, diabetes, or hyperlipidemia. A pheochromocytoma is a catecholamine-producing tumor (answer E). The classic triad of symptoms includes episodic headache, sweating, and tachycardia.

B. Oversecretion of aldosterone (answer B) describes primary hyperaldosteronism or Conn's syndrome. Patients with Conn's syndrome should present with hypokalemia and hypertension.

C. Exogenous administration of corticosteroids (answer C) is the most common cause of Cushing's syndrome, which is another cause of secondary hypertension. You should know the key features of Cushing's, which include obesity, bone loss, glucose intolerance, moon facies, a "buffalo hump," purple striae, and hypertension.

D. Atherosclerotic disease (answer D) is certainly a cause of renal artery stenosis, and would explain her abdominal bruit. The tip off in this question was that the patient was a young, previously-healthy woman - just the kind of patient who gets fibromuscular dysplasia. Atherosclerotic plaques are more likely to cause stenosis in older patients with vascular risk factors such as smoking, diabetes, or hyperlipidemia.

E. A pheochromocytoma is a catecholamine-producing tumor (answer E). The classic triad of symptoms includes episodic headache, sweating, and tachycardia.

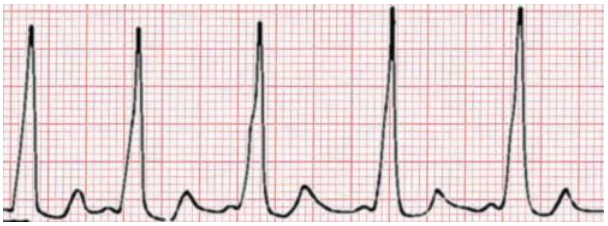
This question is not currently linked to the learning objective database.

Question problem?

Question # 12

Select the single best answer to the numbered question.

A 23 year old male presents with syncope. He reports that while walking briskly to his car, he felt his heart "racing" in his chest, and shortly thereafter passed out. The patient denies any prior syncopal episodes, but does note occasional episodes of palpitations that occur after moderate activity or during periods of increased stress. There is no family history of neurological disease, cardiac disease, or sudden cardiac death. On physical examination, pulse is 85 and regular, BP is 124/74, respiratory rate is 16, and oxygen saturation is 98% on room air. Cardiac examination reveals pulsation at the fifth intercostal space at the left parasternal area in the midclavicular line. S1 is within normal limits, and S2 is heard to split on inspiration. The remainder of the physical exam is unremarkable. EKG taken in the office shows the following tracing in lead II. Which of the following would be the most appropriate treatment for this patient's disorder?



A. Radiofrequency ablation of pre-excitation pathway
B. Urgent DC cardioversion
C. Heart transplant
D. Biventricular pacemaker placement
E. Coronary angioplasty

You did not answer this question.

Explanations:

A. This question describes a classic presentation of Wolff-Parkinson-White (WPW) syndrome, a rare disease that is frequently tested on the USMLE. This disorder is caused by the presence of an "accessory pathway," an abnormal conduction pathway that allows electrical impulses from the SA node to reach the ventricle without having to conduct through the AV node. Normal conduction through the AV node is slower than conduction through this accessory or "pre-excitation" pathway, so the ventricle gets activated at different times by the two different pathways. At rest, this produces the pathognomonic EKG finding of a delta wave, seen above. However, this pre-excitation pathway can lead to a number of cardiac arrhythmias, ranging from atrial fibrillation or flutter with a rapid ventricular response to ventricular tachycardia or fibrillation. Patients may experience a range of symptoms, from mild palpitations, to syncope, to sudden cardiac death. Although you may attempt to control the arrhythmias pharmacologically, the ultimate treatment for Wolff-Parkinson-White is radiofrequency ablation of the abnormal accessory conduction pathway (answer A). To answer this question correctly, you had to diagnose WPW from the EKG. If you'd like a more comprehensive tutorial on EKG, check out these websites from the University of Wisconsin or McGill University: http://www.fammed.wisc.edu/pcc/ecg/ecg.html http://sprojects.mmi.mcgill.ca/cardiophysio/ Briefly, to interpret the strip presented, first note the short PR interval – the P wave almost runs into the following QRS complex! In WPW, the PR interval is typically <.12s (three small boxes). The QRS is wide (>.12s) thanks to the fusion of the accessory pathway and the normal conduction pathway through the normal pathway through the AV node-His-Purjinje system. Thus, instead of a brisk, nearly vertical upstroke, you see a "slurred upstroke" – this is the delta wave that is pathognomonic for Wolff-Parkinson-White. This question also raises an important general teaching point about the causes of syncope in a young person. Far and away the most common cause of syncope in a young person is vasovagal, but you should always be aware of the more serious cardiac causes, which include Wolff-Parkinson-White, hypertrophic cardiomyopathy, congenital long QT or short QT syndromes, or valvular heart disease. Patients with Wolff-Parkinson-White typically have few or no findings on physical examination, and this patient's cardiac exam is indeed normal. The descriptions of the patient's PMI and heart sounds are those of a normal patient's. This is a common trick on the USMLE – just because a physical finding is described in detail doesn't mean that it is abnormal. (Occasionally, patients with WPW will have conduction abnormalities that result in paradoxical splitting of S2 or other abnormalities, but these are likely beyond the scope of the USMLE Step 2.)

B. DC cardioversion (answer B) is not an appropriate treatment for WPW. There are two rhythm disturbances in which urgent DC cardioversion is indicated: ventricular tachycardia and ventricular fibrillation. These are important EKG patterns to recognize, and if you are not familiar with them, make sure that you can recognize them on the USMLE. DC cardioversion may also be indicated in some other patients, such as a patient with new-onset atrial fibrillation. In these cases, depolarizing the heart with a large pulse of electrical energy may allow the heart to "reset" As described above, though, the fundamental pathology in WPW is a pre-excitation pathway, and resetting the heart does nothing to counter this abnormal conduction pathway.

C. A heart transplant (answer C) is not indicated. If you chose this answer, you might have been thinking that this patient had hypertrophic obstructive cardiomyopathy, which is a common cause of syncope in an otherwise healthy young person. However, even in that circumstance, medical management would be the most appropriate initial therapy. This patient has no symptoms of heart failure, and as with other organ transplants, the most important indication for transplantation is the patient's symptoms.

D. Biventricular pacemaker placement (answer D) would be appropriate therapy for patients with symptomatic bradyarrhythmias or sinus or AV node dysfunction. It has no role in Wolff-Parkinson-White.

E. Coronary angioplasty (answer E) is the intervention of choice for coronary artery disease, not WPW.

This question is not currently linked to the learning objective database.

Question problem?

Question # 13

Select the single best answer to the numbered question.

A 20 year old healthy female presents to her family physician for routine annual gynecological exam. She is sexually active with her monogamous male partner and currently uses the withdrawal method for contraception. Although this is her first sexual partner, she thinks her boyfriend has probably had intercourse with other women prior to their relationship. Her LMP was 2 days ago. She agrees to routine gonorrhea and Chlamydia testing. On speculum exam after swabbing for the test, some bleeding from the cervical mucosa is noted. Bimanual exam is normal. The test returns positive for N. gonorrheae. What is the best initial step in the management of this patient?

A. Reassurance and encourage condom use
B. Start and oral contraceptive pill (OCP)
C. Ceftriaxone and azithromycin
D. Doxycycline
E. Ciprofloxacin

You did not answer this question.

Explanations:

A. Reassurance (answer A) is unacceptable - gonorrhea must be treated to prevent spread and avoid complications like PID and infertility. Encouraging condom use, however, would certainly be an appropriate discussion to have with this patient to prevent future STIs.

B. Starting an oral contraceptive (answer B), though not the best next step, would probably eventually be a good idea. The withdrawal method is an inadequate form of birth control (up to 20% failure rate) so a discussion about her interest in preventing pregnancy would be a good idea. Remember, however, that OCPs don't prevent STIs.

C. This question addresses the appropriate management of a patient who has just tested positive for gonorrhea. Gonorrhea is much less prevalent than chlamydia, and patients with gonorrhea tend to be co-infected with Chlamydia. (The reverse is much less often true since chlamydia is easier to acquire statistically.) In a patient with a positive test for gonorrhea, you should therefore treat for both gonorrhea and chlamydia to avoid complications such as PID (which can lead to infertility). The combination of a one time injection of ceftriaxone and a one-time dose of azithromycin is the most appropriate choice to address the positive gonorrhea test. Some additional teaching points: 1) Gonorrhea infection is more likely to be asymptomatic in females than it is in males

the most appropriate choice to address the positive gonorrhea test. Some additional teaching points… 1) Gonorrhea infection is more likely to be asymptomatic in females than it is in males. Because gonorrhea can lead to PID and infertility, screening is important for women and recommended for all sexually active females <25 years old. 2) Remember that if a patient tests positive for chlamydia or gonorrhea, her partner MUST be treated or she will likely be reinfected. Health departments in some states employ a system of anonymous sexual partner notification, but patients should be urged to notify their partners as well to seek treatment. 3) Treating gonorrhea with a one-time ceftriaxone injection in the office ensures compliance. Several of the fluoroquinolones (like ciprofloxacin, ofloxacin, and levofloxacin) are acceptable alternatives and are orally dosed. (Remember, though - no fluoroquinolones for pregnant women (fetal toxicity) or the pediatric population (possible link with arthrotoxicity).) 4) The two most frequently used treatments for chlamydia include either a one time dose of azithromycin or one week of doxycycline. 5) A friable cervix is a sign of a possible cervicitis, which explains why this patient bled so easily when her cervix was swabbed. The other answer choices are incorrect drug regimens to address the positive gonorrhea test. Reassurance (answer A) is unacceptable - gonorrhea must be treated to prevent spread and avoid complications like PID and infertility. Encouraging condom use, however, would certainly be an appropriate discussion to have with this patient to prevent future STIs. Starting an oral contraceptive (answer B), though not the best next step, would probably eventually be a good idea. The withdrawal method is an inadequate form of birth control (up to 20% failure rate) so a discussion about her interest in preventing pregnancy would be a good idea. Remember, however, that OCPs don't prevent STIs. Doxycycline (answer C) is an acceptable treatment for chlamydia, but not for gonorrhea. Ciprofloxacin (answer D) would only address the gonorrhea infection and would go against the recommendation to also treat the likely chlamydia infection that so frequently accompanies a positive gonorrhea test result.

D. Doxycycline (answer C) is an acceptable treatment for chlamydia, but not for gonorrhea.

E. Ciprofloxacin (answer D) would only address the gonorrhea infection and would go against the recommendation to also treat the likely chlamydia infection that so frequently accompanies a positive gonorrhea test result.

This question is not currently linked to the learning objective database.

Question problem?

Question # 14

Select the single best answer to the numbered question.

A 74 year old man presents with double vision. He first noticed this several months ago, and although his symptoms wax and wane, he now experiences daily episodes of "seeing double," most frequently in the evenings. He also reports increased generalized fatigue and notes that he sometimes gets so tired at dinner that he "can hardly chew" his food. Past medical history includes osteoarthritis, hypertension, and abdominal aortic aneurysm repair. Physical examination reveals a comfortable, age-appropriate elderly gentleman with mild dysarthria. Cardiac auscultation reveals both an S4 and a 2/6 holosystolic murmur heard best at the left upper sternal border with radiation to the carotids. On neurologic exam, the patient has 5/5 strength proximally and distally. Sensation is intact and reflexes are 2+ throughout. Ocular movements are sluggish but intact in all directions. The patient has mild bilateral ptosis, which is noted to increase with sustained upward gaze. Stroking the bottom of the foot results in downward deflection of the great toe bilaterally. Which of the following is the most appropriate next step in diagnosis?

A. Administer i.v. edrophonium

B. CT of chest

C. MRI/MRA of brain and cerebral vessels

D. Temporal artery biopsy

E. CSF examination for oligoclonal bands

You did not answer this question.

Explanations:

A. This is a presentation of oculobulbar myasthenia gravis. The easiest way to confirm the diagnosis in a patient such as this one with overt ptosis is to administer i.v. edrophonium (answer A). Edrophonium is an acetylcholinesterase inhibitor with a short onset of action and half life. If you give the medication and the patient's symptoms immediately improve, you have essentially confirmed the diagnosis. Edrophonium's trade name is Tensilon, so you may hear neurologists talk about giving a patient the "Tensilon Test" to confirm MG. Alternately, EMG can diagnose MG if you see a decremental response to repetitive nerve stimulation. Here are some additional teaching points about myasthenia gravis: 1) The key feature of MG is fatigable muscle weakness. Patients will typically report that their symptoms worsen throughout the day. Oculobulbar myasthenia gravis is the most common type, resulting in the signs and symptoms presented in the question stem: double vision, ptosis, dysarthria, and difficulty chewing. 2) Myasthenia gravis has a bimodal age distribution, so there are two classic groups of patients who get MG: young women in their 20s or 30s with autoimmune disorders (RA, SLE, hyperthyroidism, etc.), and men in their 70s or 80s. 3) MG is caused by autoantibodies that bind to postsynaptic ACh receptors. A commonly tested point is distinguishing myasthenia gravis from Lambert-Eaton syndrome, which is a paraneoplastic disorder (usually associated with small cell lung cancer) in which antibodies are produced against the pre-synaptic Ca2+ channels. 4) The treatment of myasthenia gravis begins with anticholinesterase drugs like neostigmine or pyridostigmine, which increase the amount of ACh in the synapse, overcoming the antibody blockade. Prednisone or other immunosuppressive drugs are also used, and i.v. Ig or plasmaphresis are used for refractory cases to more directly target the responsible autoantibodies. 5) Myasthenia gravis almost always have some abnormality of the thymus: 75% will have thymic hyperplasia, and 15% will have an overt thymoma. Since the disease is mediated by T cells, removal of the thymus can be curative in patients who fail medical therapy. Regardless, once the diagnosis of MG has been established, it is reasonable to rule out thymoma via CT scan. If you answered B, a chest CT, you either recognized that this was a case of myasthenia gravis and were pursuing a thymoma, or you thought that this was a case of Lambert-Eaton myasthenic syndrome and were looking for a small cell lung cancer. A chest CT will provide useful – and potentially even necessary – diagnostic and prognostic information, but first you should confirm the initial diagnosis of myasthenia gravis with the edrophonium test or EMG. MRI/MRA of the brain and cerebral vessels (answer C) would be useful if you suspected a stroke or brain mass. This patient's symptoms are confined to the neuromuscular junction, however, so there is no reason to initially search out an upper motor neuron cause. A temporal artery biopsy (answer D) is useful if you are pursuing a diagnosis of temporal arteritis, which is more likely to present with jaw claudication, vision loss, and an elevated ESR. A lumbar puncture (answer E) with a CSF finding of oligoclonal bands would be consistent with a diagnosis of multiple sclerosis. To have MS, though, you must have multiple neurological complaints that are separated in time and space and cannot be explained by a single lesion.

B. If you answered B, a chest CT, you either recognized that this was a case of myasthenia gravis and were pursuing a thymoma, or you thought that this was a case of Lambert-Eaton myasthenic syndrome and were looking for a small cell lung cancer. A chest CT will provide useful – and potentially even necessary – diagnostic and prognostic information, but first you should confirm the initial diagnosis of myasthenia gravis with the edrophonium test or EMG.

C. MRI/MRA of the brain and cerebral vessels (answer C) would be useful if you suspected a stroke or brain mass. This patient's symptoms are confined to the neuromuscular junction, however, so there is no reason to initially search out an upper motor neuron cause.

D. A temporal artery biopsy (answer D) is useful if you are pursuing a diagnosis of temporal arteritis, which is more likely to present with jaw claudication, vision loss, and an elevated ESR.

E. A lumbar puncture (answer E) with a CSF finding of oligoclonal bands would be consistent with a diagnosis of multiple sclerosis. To have MS, though, you must have multiple neurological complaints that are separated in time and space and cannot be explained by a single lesion.

This question is not currently linked to the learning objective database.

Question problem?

Question # 15

Select the single best answer to the numbered question.

On routine examination, a five year old child is noted to have a loud S1 with a fixed and widely split S2 that does not vary with respiration. A soft, mid-systolic ejection murmur is heard best on the left in the second intercostal space. Remainder of physical exam is otherwise unremarkable. There is no nail clubbing, hepatomegaly, or jugular venous distension. The child is healthy and active and her mother has no health concerns. Which of the following is the most likely diagnosis in this patient?

A. Mitral valve prolapse

B. Pulmonic regurgitation

C. Tetralogy of Fallot

D. Atrial septal defect

E. Ventricular septal defect

You did not answer this question.

Explanations:

A. Although mitral valve prolapse (answer A) is often found incidentally in asymptomatic patients, the classic murmur is not present in this case. The auscultory finding of a midsystolic click (another one of those USMLE buzzwords) followed by a late systolic murmur heard best at the apex of the heart is characteristic.

B. Isolated pulmonic regurgitation (answer B) is rare, and would cause a diastolic murmu

C. The Tetralogy of Fallot (answer C) is a combination of four off-setting cardiac defects. Because the net result is a right-to-left shunting of blood, patients should have some degree of cyanosis and symptomatology. (A useful mnemonic for remembering all of the defects in Tetralogy of Fallot is "PROVE": pulmonary stenosis, right ventricular hypertrophy, overriding aorta, ventricular septal defect, and early cyanosis.)

D. This is an atrial septal defect, or ASD (answer D). There are certain "buzzwords" that you'll come across on the USMLE that almost always indicate a particular diagnosis, and this question stem has one of them. When you see fixed splitting of S2, the first thing you should think of for your shelf exam is an ASD. (As a reminder, S2 normally splits only on inspiration.) Here are some additional teaching points about ASDs… 1) ASDs, can remain asymptomatic for a long time – they are frequently picked up on routine exam. Because the pressure in the left atrium is slightly higher than the pressure in the right atrium, there is a net movement of blood from left-to-right. Over time, this overloads the pulmonary circulation and leads to pulmonary hypertension. Eventually, the pressure in the pulmonary vascular bed gets so high that the shunt reverses, causing a right-to-left shunt. This is the dreaded Eisenmenger syndrome, in which a left-to-right shunt reverses flow and turns into right-to-left shunt. 2) ASDs are silent! The murmur that is heard in this case is NOT blood flow through the ASD. Remember that to hear a murmur, you have to have turbulent blood flow. The difference in pressures between the right and left atria is so small that the blood flows easily, without turbulence. However, since there is a net left-to-right movement of blood, there is an increased volume of blood coursing across the pulmonary valve. This leads to a flow or ejection murmur heard best in the listening area for the pulmonic valve. Although mitral valve prolapse (answer A) is often found incidentally in asymptomatic patients, the classic murmur is not present in this case. The auscultory finding of a midsystolic click (another one of those USMLE buzzwords) followed by a late systolic murmur heard best at the apex of the heart is characteristic. Isolated pulmonic regurgitation (answer B) is rare, and would cause a diastolic murmur. The Tetralogy of Fallot (answer C) is a combination of four off-setting cardiac defects. Because the net result is a right-to-left shunting of blood, patients should have some degree of cyanosis and symptomatology. (A useful mnemonic for remembering all of the defects in Tetralogy of Fallot is "PROVE": pulmonary stenosis, right ventricular hypertrophy, overriding aorta, ventricular septal defect, and early cyanosis.) Ventricular septal defects (answer E) are another cause of left-to-right shunting, but the classic finding on physical exam is a low-pitched, harsh holosystolic murmur heard best at the left lower sternal border.

E. Ventricular septal defects (answer E) are another cause of left-to-right shunting, but the classic finding on physical exam is a low-pitched, harsh holosystolic murmur heard best at the left lower sternal border.

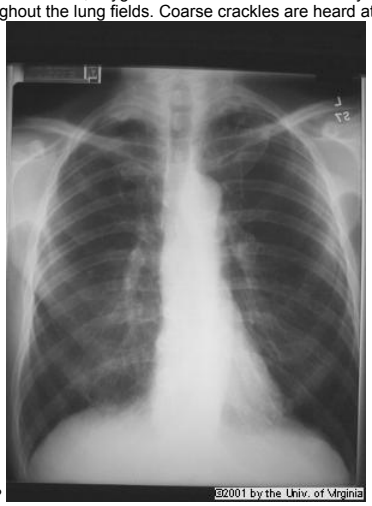
This question is not currently linked to the learning objective database.

Question problem?

Question # 16

Select the single best answer to the numbered question.

A 62 year old woman with COPD presents to her physician complaining of two days of increasing dyspnea and cough. She uses supplemental oxygen at home and has had to increase her oxygen flow to relieve her shortness of breath. Recently, her cough has been productive of copious amounts of thick, dark yellow sputum. Although she never checked her temperature at home, she thinks she might have had a fever last night. Past medical history includes hypertension and peripheral arterial disease. She has smoked 1 ½ -2 packs of cigarettes per day for the last 45 years and continues to smoke. Current medications include nebulized albuterol, tiotropium, inhaled fluticasone/salmeterol, lisinopril, and pentoxyfylline. Physical examination reveals an uncomfortable, thin female appearing older than her stated age. Vital signs are: temperature 37.2 C (99.0 F), pulse 80, blood pressure 136/70, respirations 20/min, oxygen saturation 88% on 2 L O2 by nasal cannula. The patient is using accessory muscles to breathe, and prolonged expirations and faint expiratory wheezes throughout the lung fields. Coarse crackles are heard at the lung bases. The patient's chest X-ray is shown. Which of the following is the most appropriate treatment for this patient?



A. Methylprednisolone
B. Prednisone and amoxicillin
C. Guaifenasin
D. Trimethoprim/sulfamethoxazole
E. Aminophylline

You did not answer this question.

Explanations:

A. Both methylprednisolone (answer A) and trimethoprim/sulfamethoxazole (answer D) are helpful for COPD exacerbations. However, the patient needs both antibiotics and corticosteroids, so neither one is the best answer to this question.

B. This is a patient with COPD who is experiencing an acute exacerbation of her disease. COPD is a slowly progressive disease, so sudden worsening is usually set off by a particular trigger such as an environmental irritant or an infection by bacteria or viruses. Studies have shown that corticosteroids, antibiotics, and inhaled bronchodilators all are beneficial in acute COPD exacerbations, and since this patient is already taking bronchodilators, the best answer is prednisone and amoxicillin (answer B). A few brief teaching points: 1) Although it may not make intuitive sense to treat a patient with antibiotics when it is not clear what exactly you are treating, the evidence in COPD clearly supports empiric antibiotic therapy. The antibiotics most commonly used for COPD exacerbations include amoxicillin, trimethoprim/sulfamethoxazole, and doxycycline. 2) The chest x-ray here shows the classic findings of COPD – hyperexpanded lungs, flattened diaphragms, and a narrow cardiac silhouette. This x-ray does not change your management of the patient in any way, since there are no findings to suggest an alternate diagnosis, such as consolidation suggesting a pneumonia or pneumothorax suggesting a ruptured emphysematous bulla. 3) For stable COPD, the cornerstones of treatment are inhaled beta agonists like albuterol and anticholinergic bronchodilators like tiotropium or ipratropium. Oral steroids may be need for patients with more severe disease. Supplemental oxygen has clearly been shown to prolong life in COPD patients – the only other intervention that does so is smoking cessation! Both methylprednisolone (answer A) and trimethoprim/sulfamethoxazole (answer D) are helpful for COPD exacerbations. However, the patient needs both antibiotics and corticosteroids, so neither one is the best answer to this question. Repeated studies of the mucolytic guaifenasin (answer C) have shown no benefit for either stable COPD patients or COPD exacerbations. Similarly, methylxanthines like aminophylline (answer E) or theophylline have shown no benefit for acute COPD exacerbations beyond the benefit of inhaled bronchodilators and corticosteroids.

C. Repeated studies of the mucolytic guaifenasin (answer C) have shown no benefit for either stable COPD patients or COPD exacerbations.

D. Both methylprednisolone (answer A) and trimethoprim/sulfamethoxazole (answer D) are helpful for COPD exacerbations. However, the patient needs both antibiotics and corticosteroids, so neither one is the best answer to this question.

E. Methylxanthines like aminophylline (answer E) or theophylline have shown no benefit for acute COPD exacerbations beyond the benefit of inhaled bronchodilators and corticosteroids.

This question is not currently linked to the learning objective database.

Question problem?

Question # 17

Select the single best answer to the numbered question.

A 64 year old male presents for routine health evaluation. He has been feeling well and has no complaints. Past medical history includes hypertension, osteoarthritis, and generalized anxiety disorder. Medications include hydrochlorothiazide, ibuprofen, atenolol, and paroxetine. Physical exam shows temperature 37.9 C (99.3 F), pulse 61, blood pressure 131/70, and respirations 15/min. Laboratory evaluation shows: Na+ 141; K+ 3.9; Cl- 103; HCO3- 25; BUN 18; Creatinine 1.2; WBC 9.7; Hemoglobin 10.1; Platelets 179; MCV 73 fL. Which of the following is the most appropriate next step in the management of this patient?

A. Add lisinopril
B. Measure reticulocyte count
C. Test B12 and folate levels
D. Endoscopy
E. Indirect and direct Coombs' tests

You did not answer this question.

Explanations:

A. Adding lisinopril (answer A) might be appropriate if this patient's blood pressure were elevated. In general, the goal for hypertension management is to keep the blood pressure under 140/90, although some authorities recommend lower targets (such as less than 130/80 or 120/80) for high-risk patients with diabetes or kidney disease.

B. Measuring the reticulocyte count (answer B) is not unreasonable, and if this patient were sitting in your office, you'd probably go ahead and order that test. However, the reticulocyte count is not very useful in working up a MICROCYTIC anemia – you have to use other tests to distinguish between the disorders in the "TAILS" mnemonic. The reticulocyte count allows you to separate anemias that are caused by increased red cell destruction from those that are caused by impaired red cell production, and if this patient had a NORMOCYTIC anemia, it would be the first test you should order. All microcytic anemias are caused by impaired incorporation of hemoglobin into RBCs, so unless the anemia is multifactorial (which is possible in the real world, but unlikely on the USMLE) the reticulocyte count won't help you.

C. Deficiency of either B12 or folate (answer C) can cause anemia, but the anemia tends to be macrocytic, with an MCV greater than 100.

D. Key teaching point: Iron deficiency anemia in a male is colorectal cancer until proven otherwise. This patient presents with a microcytic anemia: his hemoglobin and MCV are both low. The differential diagnosis of microcytic anemias is fairly broad, and includes a number of "zebras." (A useful mnemonic to remember the differential of microcytic anemias is "TAILS" – Thalassemias, Anemia of chronic disease (which can sometimes be normocytic), Iron deficiency anemia, Lead poisoning, and Sideroblastic anemia) Far and away the top cause of a microcytic anemia is iron deficiency anemia brought about by blood loss. Any male or postmenopausal woman who presents with iron deficiency anemia should be evaluated for an occult GI source of their bleeding, preferably with upper and lower endoscopy (answer D). This patient could have an ulcer related to his NSAID use – or he could have colorectal carcinoma. Either case deserves further evaluation. Adding lisinopril (answer A) might be appropriate if this patient's blood pressure were elevated. In general, the goal for hypertension management is to keep the blood pressure under 140/90, although some authorities recommend lower targets (such as less than 130/80 or 120/80) for high-risk patients with diabetes or kidney disease. Measuring the reticulocyte count (answer B) is not unreasonable, and if this patient were sitting in your office, you'd probably go ahead and order that test. However, the reticulocyte count is not very useful in working up a MICROCYTIC anemia – you have to use other tests to distinguish between the disorders in the "TAILS" mnemonic. The reticulocyte count allows you to separate anemias that are caused by increased red cell destruction from those that are caused by impaired red cell production, and if this patient had a NORMOCYTIC anemia, it would be the first test you should order. All microcytic anemias are caused by impaired incorporation of hemoglobin into RBCs, so unless the anemia is multifactorial (which is possible in the real world, but unlikely on the USMLE) the reticulocyte count won't help

you. Deficiency of either B12 or folate (answer C) can cause anemia, but the anemia tends to be macrocytic, with an MCV greater than 100. The indirect and direct Coombs' tests (answer E) are useful in identifying antibody mediated causes of hemolysis and anemia. Such hemolytic anemias are typically normocytic, and the patient would have an elevated reticulocyte count as well as increased LDH and bilirubin and decreased haptoglobin.

E. The indirect and direct Coombs' tests (answer E) are useful in identifying antibody mediated causes of hemolysis and anemia. Such hemolytic anemias are typically normocytic, and the patient would have an elevated reticulocyte count as well as increased LDH and bilirubin and decreased haptoglobin.

This question is not currently linked to the learning objective database.

Question problem?

Question # 18

Select the single best answer to the numbered question.

A 36 year old African-American male comes to the clinic complaining of fatigue and dark, cola-colored urine for the past five days. He denies fever, nausea or vomiting, recent travel, and i.v. drug use. Past medical history includes ulcerative colitis. Medications include sulfasalazine as well as trimethoprim/sulfamethoxazole which the patient began taking one week ago for a presumed Staphylococcal skin infection. Vital signs are temperature 36.8 C (98.2 F), pulse 78, blood pressure 118/72, respirations 14/min. Physical examination shows scleral icterus and a nontender abdomen without organomegaly. Laboratory evaluation shows: WBC 8.6; Hgb 9.1; Hct 27.3; Platelets 212; MCV 88 fL; Na+ 144; K+ 4.8; Cl- 101; HCO3- 26; BUN 14; Creatinine 1.0; Glucose 101; LDH 410 U/L; Haptoglobin 8 mg/dL. Which of the following is the most appropriate next step in management of this patient?

A. Quantitative IgM for hepatitis A virus
B. Begin darbopoietin injections
C. Begin methylprednisolone
D. Discontinue trimethoprim/sulfamethoxazole
E. Immediate transfusion of packed red blood cells

You did not answer this question.

Explanations:

A. Quantitative IgM for hepatitis A virus (answer A) is the best way to diagnose an acute hepatitis A infection, which can be a cause of jaundice. However, this patient's jaundice is not due to his liver – it's due to the fact that he is hemolyzing his red cells, as evidenced by the elevated LDH and low haptoglobin.

B. Darbopoietin injections (answer B) are the treatment for anemia due to chronic kidney disease, where the kidneys are not producing enough erythropoietin to stimulate the bone marrow to produce more RBCs. If you were to check a reticulocyte count on this patient, however, it would be elevated – his anemia is not from inadequate production of RBCs, it's from hemolysis of RBCs, as shown by the increased LDH and decreased haptoglobin.

C. Corticosteroids like methylprednisolone (answer C) are used to treat autoimmune hemolytic anemias, which can present similarly to this patient. Only the clues about the patient's race and the timing of the disease onset after starting TMP/SMZ steer you toward the diagnosis of G6PD deficiency. So why isn't this the correct answer? Before starting a new drug, unless it is urgent that you do so, you should confirm the diagnosis. Here, that would involve a peripheral smear or Coombs' testing to confirm a diagnosis of autoimmune hemolytic anemia. Your threshold for stopping a non-essential drug is usually lower, though, and if G6PD deficiency is on your differential diagnosis (and it should be) you ought to stop the potential offending drug while you try to tease out whether the patient's symptoms are due to G6PD deficiency or autoimmune hemolysis. This may seem nit-picky, but the USMLE loves these kinds of issues. Keep in mind the order that you would do things - of the choices listed, stopping TMP/SMZ is the most appropriate NEXT step.

D. This is a case of glucose-6-phosphate dehydrogenase (G6PD) deficiency, an X-linked recessive disease that tends to affect men of Mediterranean or African descent. G6PD is an enzyme in glycolysis that is responsible for the production of NADPH, which maintains glutathione in its reduced state and protects cells from oxidative stress. Since red blood cells are dependent on glycolysis and thus G6PD to produce their NADPH and glutathione, deficiency of G6PD results in RBCs that are exquisitely susceptible to oxygen radicals. Anything that increases oxidative stress results in hemolysis and an acute hemolytic anemia. The most common culprits include: viral or bacterial infections, fava beans, nitrofurantoin, quinine, dapsone, and sulfonamides. Here, the culprit was sulfamethoxazole, so that drug should be discontinued (answer D). The disease is typically self limiting, and treatment is supportive. The key diagnostic findings in this patient are the elevated LDH and decreased haptoglobin, both of which indicate that RBCs are being lysed and are releasing their contents into the serum. The patient's total and indirect (unconjugated) bilirubin are also likely elevated, given his clinical presentation of jaundice. Quantitative IgM for hepatitis A virus (answer A) is the best way to diagnose an acute hepatitis A infection, which can be a cause of jaundice. However, this patient's jaundice is not due to his liver – it's due to the fact that he is hemolyzing his red cells, as evidenced by the elevated LDH and low haptoglobin. Darbopoietin injections (answer B) are the treatment for anemia due to chronic kidney disease, where the kidneys are not producing enough erythropoietin to stimulate the bone marrow to produce more RBCs. If you were to check a reticulocyte count on this patient, however, it would be elevated – his anemia is not from inadequate production of RBCs, it's from hemolysis of RBCs, as shown by the increased LDH and decreased haptoglobin. Corticosteroids like methylprednisolone (answer C) are used to treat autoimmune hemolytic anemias, which can present similarly to this patient. Only the clues about the patient's race and the timing of the disease onset after starting TMP/SMZ steer you toward the diagnosis of G6PD deficiency. So why isn't this the correct answer? Before starting a new drug, unless it is urgent that you do so, you should confirm the diagnosis. Here, that would involve a peripheral smear or Coombs' testing to confirm a diagnosis of autoimmune hemolytic anemia. Your threshold for stopping a non-essential drug is usually lower, though, and if G6PD deficiency is on your differential diagnosis (and it should be) you ought to stop the potential offending drug while you try to tease out whether the patient's symptoms are due to G6PD deficiency or autoimmune hemolysis. This may seem nit-picky, but the USMLE loves these kinds of issues. Keep in mind the order that you would do things - of the choices listed, stopping TMP/SMZ is the most appropriate NEXT step. This patient does not need an immediate transfusion (answer E). Transfusions are reserved for patients with severe symptoms, worsening status, or fragile overall medical condition. This patient's vital signs remain stable, and he is an otherwise healthy 36 year old, so although he should be monitored, he does not need a transfusion now.

E. This patient does not need an immediate transfusion (answer E). Transfusions are reserved for patients with severe symptoms, worsening status, or fragile overall medical condition. This patient's vital signs remain stable, and he is an otherwise healthy 36 year old, so although he should be monitored, he does not need a transfusion now.

This question is not currently linked to the learning objective database.

Question problem?

Question # 19

Select the single best answer to the numbered question.

A 65 year old female with diabetes is found on routine screening to have a total serum cholesterol concentration of 198 mg/dL, with a serum HDL cholesterol of 58 mg/dL and serum LDL cholesterol of 128 mg/dL. Triglycerides are 78 mg/dL, and last hemoglobin A1c is 6.5%. The patient has no known history of coronary artery disease, does not smoke, and exercises daily. Current medications include rosuvastatin, fosinopril, glyburide, and metoprolol. Blood pressure is 129/78. Urinalysis shows no protein. Which of the following is the best management plan and treatment goal for this patient?

A. Add niacin to increase HDL to >80 mg/dL
B. Increase rosuvastatin to target LDL <100 mg/dL
C. Add gemfibrozil to decrease triglycerides to <70 mg/dL
D. Discontinue fosinopril and add hydrochlorothiazide to decrease blood pressure to <120/80
E. Begin insulin therapy to target Hgb A1c <6.0%

You did not answer this question.

Explanations:

A. Although statins and fibrates can modestly raise the HDL, niacin (answer A) is the only medication that has a profound effect on HDL concentrations. While patients ought to have as much "good" cholesterol as they can, this patient's HDL is quite good, especially for a diabetic. Theoretically, she could benefit from an even higher HDL, but evidence clearly supports getting her LDL below 70.

B. Key teaching point: Patients with diabetes are considered to have known coronary artery disease for purposes of lipid goals, and need the lowest possible LDL. This question involves lipid management. Presently, we tend to focus most of our efforts on a patient's LDL (or "bad") cholesterol, since there is the greatest amount of evidence linking elevated LDL to adverse cardiovascular events and death. In general, to figure out a patient's lipid goal, you first count up their risk factors. Cigarette smoking, a family history of premature cardiovascular disease, low "good" cholesterol (HDL <40 mg/dL), and hypertension (BP >140/90) all count as risk factors. Patients with two or more risk factors have a target LDL goal of <130 mg/dL, while patients with zero or one risk factor have an LDL goal of <160 mg/dL. However, there's a catch: patients with KNOWN coronary artery disease (previous MI, angina, etc.) need a lower LDL of <100 mg/dL. Also, a number of other diseases count as "coronary heart disease equivalents" and earn the same lower target LDL of less than 100. Patients with cerebrovascular disease, an abdominal aortic aneurysm, or peripheral arterial disease are assumed to have diseased coronary vessels as well. Patients with diabetes also fit into this category, so even though the patient in the question stem has no other risk factors, she still needs the most stringent LDL goal of less than 100. Here is a quick review of the 2001 ATP III lipid guidelines: http://www.nhlbi.nih.gov/guidelines/cholesterol/atglance.pdf One caveat about all of this: since these guidelines came out in 2001, evidence has shown a benefit of an LDL goal of <70 mg/dL for VERY high risk patients, such as those who have had a previous MI or who have numerous risk factors and comorbidities. Although statins and fibrates can modestly raise the HDL, niacin (answer A) is the only medication that has a profound effect on HDL concentrations. While patients ought to have as much "good" cholesterol as they can, this patient's HDL is quite good, especially for a diabetic. Theoretically, she could benefit from an even higher HDL, but evidence clearly supports getting her LDL below 70. Fibrates like gemfibrozil (answer C) are indeed the drugs of choice for lowering triglycerides. While elevated triglycerides are a risk factor for vascular disease and seriously elevated triglycerides can cause pancreatitis, this patient's bigger problem is her LDL, which is still above her goal. Switching fosinopril to hydrochlorothiazide (answer D) is incorrect. Diabetic patients have a compelling reason to be on an ACE inhibitor or ARB as opposed to any other class of antihypertensive, because these medications have been shown to reduce the progression of diabetic kidney disease. Beginning insulin (answer E) is not necessary at this time. The target hemoglobin A1c for diabetic patients is 7.0%, which corresponds to three month mean blood glucose level of less than 150 mg/dL.

C. Fibrates like gemfibrozil (answer C) are indeed the drugs of choice for lowering triglycerides. While elevated triglycerides are a risk factor for vascular disease and seriously elevated triglycerides can cause pancreatitis, this patient's bigger problem is her LDL, which is still above her goal.

D. Switching fosinopril to hydrochlorothiazide (answer D) is incorrect. Diabetic patients have a compelling reason to be on an ACE inhibitor or ARB as opposed to any other class of antihypertensive, because these medications have been shown to reduce the progression of diabetic kidney disease.

E. Beginning insulin (answer E) is not necessary at this time. The target hemoglobin A1c for diabetic patients is 7.0%, which corresponds to three month mean blood glucose level of less than 150 mg/dL.

This question is not currently linked to the learning objective database.

Question problem?

Question # 20

Select the single best answer to the numbered question.

A 5 year old female presents for a pre-kindergarten physical and is found to have a blood pressure of 146/85 in both arms. Past medical history is unremarkable, and the child has been healthy and asymptomatic. Physical examination reveals a short, stocky female with a broad, shield shaped chest and widely-spaced nipples. There are diminished femoral pulses bilaterally. On cardiac auscultation, a short 2/6 midsystolic murmur is heard at the left paravertebral interscapular area. Which of the following is the most likely associated finding in this patient?

A. Increased urine homovanillic acid (HVA) and vanillylmandelic acid (VMA)
B. Tonsillar hypertrophy
C. Decreased levels of thyroid stimulating hormone (TSH)
D. Chest x-ray showing notching of the ribs bilaterally
E. Sensorineural hearing loss and hematuria

You did not answer this question.

Explanations:

A. Increased urine homovanillic acid (HVA) and vanillylmandelic acid (VMA) (answer A) is seen in patients with neuroblastoma, a common childhood tumor that can cause hypertension due to catecholamine release. (As a sidenote, patients with pheochromocytomas may also have hypertension and increased urine VMA and HVA, but they should also have increased urine metanephrine and normetanephrine. Neuroblastomas have defective catecholamine production, so they are unable to produce these latter metabolites.)

B. Tonsillar hypertrophy (answer B) may cause obstructive sleep apnea, which is another cause of secondary hypertension in children. For this to be the most likely diagnosis on the USMLE, though, there should be some history of labored breathing, snoring, or daytime sleepiness. The treatment for severe disease is surgical, via adenotonsillectomy.

C. Decreased levels of thyroid-stimulating hormone (answer C) are diagnostic of hyperthyroidism, which is an important cause of secondary hypertension in both children and adults. Apart from hypertension, though, this patient has no findings that suggest hyperthyroidism.

D. This child has coarctation of the aorta, a congenital heart defect that is a common cause of secondary hypertension. If the obstruction is severe, patients will present early in the newborn period with congestive heart failure. However, if the coarctation is less severe, and if collateral vessels are able to form, patients will remain asymptomatic throughout infancy but will present later in childhood (or even in adulthood!) with hypertension. It is the development of arterial collaterals (through the intercostal arteries in an attempt to bypass the blockage in the aorta) that leads to the classic description of “rib notching” on chest x-ray (answer D). Typically, when this question is asked on the USMLE, it’s asked the other way around – you’ll be given the finding of rib notching and asked to diagnose aortic coarctation. This child also has something else: Turner’s syndrome. (A question describing a short, squat, female with a webbed neck, a shield shaped chest, or widely-spaced nipples should set off your USMLE buzzword alarms!) Patients with this disorder have a single X chromosome (the “XO” genotype), and typically present with either short stature or primary amenorrhea. However, up to 20% of patients with Turner’s syndrome may also have coarctation of the aorta, so its description here was not by coincidence. Increased urine homovanillic acid (HVA) and vanillylmandelic acid (VMA) (answer A) is seen in patients with neuroblastoma, a common childhood tumor that can cause hypertension due to catecholamine release. (As a sidenote, patients with pheochromocytomas may also have hypertension and increased urine VMA and HVA, but they should also have increased urine metanephrine and normetanephrine. Neuroblastomas have defective catecholamine production, so they are unable to produce these latter metabolites.) Tonsillar hypertrophy (answer B) may cause obstructive sleep apnea, which is another cause of secondary hypertension in children. For this to be the most likely diagnosis on the USMLE, though, there should be some history of labored breathing, snoring, or daytime sleepiness. The treatment for severe disease is surgical, via adenotonsillectomy. Decreased levels of thyroid-stimulating hormone (answer C) are diagnostic of hyperthyroidism, which is an important cause of secondary hypertension in both children and adults. Apart from hypertension, though, this patient has no findings that suggest hyperthyroidism. Sensorineural hearing loss and hematuria (answer E) are features of Alport syndrome, an inherited glomerulonephritis that is a favorite on the USMLE. Patients may present with hypertension, but here the physical exam findings suggest an obvious alternate diagnosis. The most common form is inherited as an X-linked trait, so although a female with Turner’s syndrome might be affected, it is generally rare in girls.

E. Sensorineural hearing loss and hematuria (answer E) are features of Alport syndrome, an inherited glomerulonephritis that is a favorite on the USMLE. Patients may present with hypertension, but here the physical exam findings suggest an obvious alternate diagnosis. The most common form is inherited as an X-linked trait, so although a female with Turner’s syndrome might be affected, it is generally rare in girls.

This question is not currently linked to the learning objective database.

Question problem?

Question # 21

Select the single best answer to the numbered question.

An 84 year old woman has experienced three weeks of diffuse myalgias. In addition to worsening fatigue, she has stiffness upon awakening in her shoulders, hip girdles, neck, and torso. The stiffness usually resolves after several hours. She has also had occasional low-grade fevers and a 6 pound weight loss since her symptoms began. Physical examination shows decreased active range of motion of the shoulders and neck, but no muscle tenderness. Neurological exam shows normal sensation and reflexes. No abnormal findings are noted on skin examination. Which of the following is the most likely associated finding in this patient’s disease?

A. Anti-centromere antibodies positive at 1:640
B. Absolute neutrophil count of 950/mm3 (Normal: >1800 mm3)
C. Karyotyping showing t(9,22) chromosomal rearrangement
D. Erythrocyte sedimentation rate (ESR) of 96 mm/h (Normal: <20 mm/h)
E. X-rays demonstrating expansion of the bony cortex in a mosaic pattern

You did not answer this question.

Explanations:

A. Anti-centromere antibodies (answer A) are a specific marker for scleroderma. Clinical features vary, but include thickening of the skin, Raynaud phenomenon, esophageal dysmotility, and interstitial lung disease.

B. A decreased absolute neutrophil count (answer B) is a feature of Felty’s syndrome, which consists of the triad of seropositive rheumatoid arthritis, splenomegaly, and granulocytopenia.

C. The t(9,22) chromosomal rearrangement (answer C) is the Philadelphia chromosome, which is pathognomonic for chronic myelogenous leukemia (CML). This rearrangement leads to the BCR-ABL fusion product, which is another commonly-tested aspect of the disease. While CML can cause fatigue, weight loss, and low-grade fevers, it also should include splenomegaly and abnormalities on CBC like elevated WBC or platelets and anemia. You were not given enough in the question stem to make CML the most likely diagnosis.

D. Key teaching point: A markedly elevated ESR is the hallmark of polymyalgia rheumatica. This is polymyalgia rheumatica, one of the all-time favorites of shelf exams. Patients present with the symptoms above, though they will have few findings on physical examination. The erythrocyte sedimentation rate is usually markedly elevated (answer D) and is usually above 70-80 mm/h, but sometimes over 100 mm/h. The treatment for polymyalgia rheumatica is corticosteroids – patients will usually feel MUCH better after just a few doses of prednisone! Another key teaching point about polymyalgia rheumatica is its association with giant cell arteritis or temporal arteritis. Patients with temporal arteritis will have temporal artery tenderness, headache, jaw pain, or evidence of ischemia (such as arm claudication or TIAs). The most ominous finding is transient visual loss, as temporal arteritis can occlude the arteries leading to the eye. This can lead to permanent blindness if the patient is not treated immediately. Anti-centromere antibodies (answer A) are a specific marker for scleroderma. Clinical features vary, but include thickening of the skin, Raynaud phenomenon, esophageal dysmotility, and interstitial lung disease. A decreased absolute neutrophil count (answer B) is a feature of Felty’s syndrome, which consists of the triad of seropositive rheumatoid arthritis, splenomegaly, and granulocytopenia. The t(9,22) chromosomal rearrangement (answer C) is the Philadelphia chromosome, which is pathognomonic for chronic myelogenous leukemia (CML). This rearrangement leads to the BCR-ABL fusion product, which is another commonly-tested aspect of the disease. While CML can cause fatigue, weight loss, and low-grade fevers, it also should include splenomegaly and abnormalities on CBC like elevated WBC or platelets and anemia. You were not given enough in the question stem to make CML the most likely diagnosis. Expansion of the bony cortex in a mosaic pattern (answer E) is characteristic of Paget’s disease of bone. This comes up on the USMLE from time to time, but the question stem will usually contain clues such as an older patient who complains that he is slowly going deaf or that his hat size is increasing, or who shows frontal bossing or tibial bowing on physical exam. You would also expect to see an elevated alkaline phosphatase. Deep bony pain is often a presenting complaint, but not the kind of myalgias that the patient in this question stem had.

E. Expansion of the bony cortex in a mosaic pattern (answer E) is characteristic of Paget’s disease of bone. This comes up on the USMLE from time to time, but the question stem will usually contain clues such as an older patient who complains that he is slowly going deaf or that his hat size is increasing, or who shows frontal bossing or tibial bowing on physical exam. You would also expect to see an elevated alkaline phosphatase. Deep bony pain is often a presenting complaint, but not the kind of myalgias that the patient in this question stem had.

This question is not currently linked to the learning objective database.

Question problem?

Question # 22

Select the single best answer to the numbered question.

An 18 year old female presents to her family physician to discuss options for birth control. Menarche was at age 12. Her menses occur at regular 28 day intervals, but she does have moderate adominal cramping, bloating, and occasional nausea that sometimes prevent her from going to school. She is interested in becoming sexually active but her boyfriend, who has had other sexual partners in the past, doesn’t like to use condoms. Her family history is significant for her mother having had ovarian cancer at age 40. The patient does not smoke and is otherwise healthy. What would be the most appropriate initial step in the management of this patient?

A. Recommend condom use because of the patient's family history of ovarian cancer
B. Prescribe an OCP after a work-up of her cyclical abdominal cramping
C. Start a progestin-only pill to limit the patient's intake of estrogen
D. Prescribe a combined oral contraceptive
E. Recommend an intrauterine device (IUD)

You did not answer this question.

Explanations:

A. A family history of ovarian cancer is not a contraindication to oral contraceptive use (answer A). In fact, OCPs may help to reduce this patient's risk of ovarian cancer.

A. A family history of ovarian cancer is not a contraindication to oral contraceptive use (answer A). In fact, OCPs may help to reduce this patient's risk of ovarian cancer.

B. This patient's abdominal cramping is very typical of primary dysmennorhea and does not require further work-up at this time (answer B). Again, an OCP will likely help reduce her menstrual cramping, bloating and nausea.

C. A progestin only pill (choice C) is less effective and more difficult to take, and thus would not be the best choice for this young patient who has no need to avoid estrogen exposure.

D. This case asks you to choose the best contraceptive option for the patient. While there are many factors that can lead one to pick a certain type of contraceptive, let's focus on the choices presented here. This patient has a first degree relative with ovarian cancer. Remember that any choice that prevents ovulation will REDUCE her risk of developing ovarian cancer (by suppressing "incessant ovulation"), so there is a compelling reason to choose such a method. The patient also describes classic primary dysmenorrhea that is very common in the young female population and can also be improved by starting this patient on an OCP, which is why this is the best choice for her (answer D). A few extra teaching points: 1) Primary dysmennorhea is caused by the inflammatory response (mediated by prostaglandins and leukotrienes) at menstruation that causes uterine contractions. Pain may be due to a temporary endometrial ischemic process. Combination OCPs make the endometrial lining thin, meaning that there are less inflammatory products produced at the time or menses, which in turn reduces menstrual flow and uterine contractions. 2) Questions sometimes come up regarding OCPs and cancer. Data support the assertion that OCPs help to PREVENT ovarian cancer. Currently, however, the literature does not clearly support or refute the idea that OCPs could cause breast cancer. Any patient with a family history of breast cancer should be made aware of the current uncertainty about the relationship, but advised that years of studies have shown no increased cancer risk with the more moden low-estrogen formulation pills that are prescribed today. Remember, of course, that estrogen-only supplementation should never be used in pre-menopausal women with a uterus because excessive estrogen exposure has been linked to endometrial hyperplasia and carcinoma. 3) Progestin only pills (a.k.a. "POPs" or "the mini pill") are associated with more break-through bleeding and slightly higher failure rates than the combination pill that contains both estrogen and progesterone. Progestin only pills are more difficult to take, because they must be taken at the same time every day to maintain their efficacy. They are usually reserved for women who have a compelling reason avoid estrogen. Such patients might include women with migraine headaches, smokers over age 35, patients in the postpartum period, or women with clotting disease, cardiovascular disease, uncontrolled HTN, SLE, or hypertriglyceridemia. If you're interested in a more thorough review of birth control options, try http://www.aafp.org/afp/20040215/853.html A family history of ovarian cancer is not a contraindication to oral contraceptive use (answer A). In fact, OCPs may help to reduce this patient's risk of ovarian cancer. This patient's abdominal cramping is very typical of primary dysmennorhea and does not require further work-up at this time (answer B). Again, an OCP will likely help reduce her menstrual cramping, bloating and nausea. A progestin only pill (choice C) is less effective and more difficult to take, and thus would not be the best choice for this young patient who has no need to avoid estrogen exposure. An intrauterine device (choice E) would not be the best choice at this time. IUDs are foreign bodies that can increase the risk of an upper genital tract infection if a patient acquires an STI, so they're not the best choice in a young sexually active patient who is not using condoms. In addition, dysmennorhea is a relative contraindication for IUD placement because it can worsening of this condition. The optimal patients for IUDs, therefore, are parous women in monogamous relationships.

E. An intrauterine device (choice E) would not be the best choice at this time. IUDs are foreign bodies that can increase the risk of an upper genital tract infection if a patient acquires an STI, so they're not the best choice in a young sexually active patient who is not using condoms. In addition, dysmennorhea is a relative contraindication for IUD placement because it can worsening of this condition. The optimal patients for IUDs, therefore, are parous women in monogamous relationships.

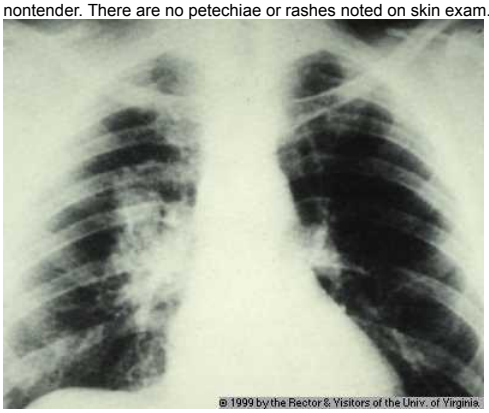
This question is not currently linked to the learning objective database.

Question problem?

Question # 23

Select the single best answer to the numbered question.

A 55 year old woman has had four days of a cough productive of dark yellow sputum. She has also experienced occasional shaking chills and has sharp chest pain that is worst on inspiration. She denies recent travel history, known sick contacts, or recent hospitalization. Past medical history is significant for osteoarthritis and hypertension. Temperature is 38.0 C (100.4 F), pulse 85, blood pressure 132/80, respirations 22/min, oxygen saturation 97% on room air. The patient is alert, oriented, and interactive. Heartbeat is regular with normal S1 and S2. There is no JVD. There are decreased breath sounds as well as egophony and positive tactile fremitus at the right lung base. Abdomen is obese, nondistended, and nontender. There are no petechiae or rashes noted on skin exam. Chest x-ray is shown. Which of the following is the most appropriate therapy for this patient?



A. Enoxaparin
B. Vancomycin and meropenem
C. Cefepime and gentamicin
D. Azithromycin
E. Immediate needle thoracostomy

You did not answer this question.

Explanations:

A. Enoxaparin (answer A) or heparin would be useful treatment if the patient had a pulmonary embolus. The constellation of signs and symptoms here is classic for pneumonia, however.

B. The combinations of vancomycin and meropenem (answer B) and cefepime and gentamicin (answer C) might sometimes be used to treat hospital acquired pneumonias. Cefepime is a fourth generation cephalosporin with good Pseudomonas activity, making it a good choice for nosocomial infections. Meropenem has excellent anti-pseudomonal activity as well, while vancomycin covers MRSA and multi-drug resistant S.pneumo. For an ICU patient or someone with a nosocomial infection, some combination of these "big gun" drugs is warranted in order to cover all of the likely suspects. They are all i.v. formulations, however, so they are too cumbersome to use as outpatient treatment. They additionally have no activity against the atypical causes of pneumonia,

which should be covered in this patient.

C. The combinations of vancomycin and meropenem (answer B) and cefepime and gentamicin (answer C) might sometimes be used to treat hospital acquired pneumonias. Cefepime is a fourth generation cephalosporin with good Pseudomonas activity, making it a good choice for nosocomial infections. Meropenem has excellent anti-pseudomonal activity as well, while vancomycin covers MRSA and multi-drug resistant S.pneumo. For an ICU patient or someone with a nosocomial infection, some combination of these "big gun" drugs is warranted in order to cover all of the likely suspects. They are all i.v. formulations, however, so they are too cumbersome to use as outpatient treatment. They additionally have no activity against the atypical causes of pneumonia, which should be covered in this patient.

D. In this case, everything points to pneumonia – from the history of a productive cough to the physical exam findings of consolidation to the CXR showing perihilar fluffy infiltrates. Because there is no history of the patient being hospitalized, this is community acquired pneumonia (CAP). The first decision to make with CAP is whether the patient should be hospitalized or not. A variety of clinical tools exist to risk stratify patient and identify those who need hospitalization. The best known and validated of these is the PORT score, which you can calculate using this tool: http://www.chestx-ray.com/Practice/PORT/PORT.html Of course, the authors of the USMLE do not expect you to calculate PORT scores, but you should get the general idea: patients who are elderly, who have unstable vital signs or altered mental status, or who have medical comorbidities have a high mortality rate from pneumonia and require hospitalization. Patients who lack these findings can potentially be managed as outpatients with close follow-up. This patient is under age 65 with stable vital signs and no serious underlying medical illnesses – she is an ideal candidate for outpatient therapy. Once you've decided that a patient can be managed as an outpatient, you should select a drug regimen that covers the likely causative agents. In this setting, the most likely bug is S. pneumoniae, with the next most likely bugs being H.influenzae and Moraxella catarrhalis. You also need to consider the "atypical" causes of pneumonia like Mycoplasma, Chlamydia, and Legionella. (In fact, the CXR shown was taken from a patient with Mycoplasma.) A significant number of S.pneumoniae strains are resistant to beta-lactams, as are many H.flu strains and almost all Moraxella strains. Atypical pneumonias are also impervious to beta-lactams and cephalosporins, so your antibiotic choices are limited to macrolides, quinolones, and tetracyclines. Although erythromycin or tetracycline could work, macrolides like clarithromycin or azithromycin (answer D) cover H.flu better and would be the best choice among those listed. Doxycycline or a "respiratory" fluoroquinolone like levofloxacin or moxifloxacin are reasonable outpatient monotherapies, as well. Enoxaparin (answer A) or heparin would be useful treatment if the patient had a pulmonary embolus. The constellation of signs and symptoms here is classic for pneumonia, however. The combinations of vancomycin and meropenem (answer B) and cefepime and gentamicin (answer C) might sometimes be used to treat hospital acquired pneumonias. Cefepime is a fourth generation cephalosporin with good Pseudomonas activity, making it a good choice for nosocomial infections. Meropenem has excellent anti-pseudomonal activity as well, while vancomycin covers MRSA and multi-drug resistant S.pneumo. For an ICU patient or someone with a nosocomial infection, some combination of these "big gun" drugs is warranted in order to cover all of the likely suspects. They are all i.v. formulations, however, so they are too cumbersome to use as outpatient treatment. They additionally have no activity against the atypical causes of pneumonia, which should be covered in this patient. Needle thoracostomy (answer E) is indicated for the urgent treatment of a tension pneumothorax, an unlikely diagnosis given this patient's history. Here, pneumothorax is explicitly ruled out by the CXR showing a lobar pneumonia. A tension pneumothorax is a real life-or-death situation, and if you suspect one clinically, there is no time for imaging studies – you should decompress the patient immediately with a needle or the placement of a chest tube.

E. Needle thoracostomy (answer E) is indicated for the urgent treatment of a tension pneumothorax, an unlikely diagnosis given this patient's history. Here, pneumothorax is explicitly ruled out by the CXR showing a lobar pneumonia. A tension pneumothorax is a real life-or-death situation, and if you suspect one clinically, there is no time for imaging studies – you should decompress the patient immediately with a needle or the placement of a chest tube.

This question is not currently linked to the learning objective database.

Question problem?

Question # 24

Select the single best answer to the numbered question.

A 15 year old girl is referred from her dentist after she bled excessively following extraction of her wisdom teeth. She also reports prolonged menses lasting 8-10 days and recurrent epistaxis. Her mother experienced lengthy postpartum bleeding after the delivery of all three of her children. Physical examination reveals scattered petechiae. Laboratory analysis shows: WBC 7.6; Hemoglobin 9.8; Hematocrit 29.1; Platelets 229; PT 12 s (normal 11-15s); aPTT 35 s (normal 20-35s); Bleeding time 13 min (normal 2-7 min). Which of the following is the most appropriate treatment for this patient's disorder?

A. Plasmaphresis
B. Desmopressin
C. Factor VIII concentrate
D. Hydroxyurea
E. Splenectomy

You did not answer this question.

Explanations:

A. Plasmaphresis (answer A) is the treatment for thrombocytopenic thrombotic purpura or TTP. The classic pentad of TTP is microangiopathic hemolytic anemia, thrombocytopenia, mental status changes or neurological abnormalities, fever, and renal dysfunction.

B. This is von Willebrand's disease, the most common hereditary bleeding disorder. This is an autosomal dominant disease that results in deficient or defective von Willebrand factor, a large glycoprotein that functions as a carrier protein for Factor VIII and also aids in platelet adhesion. To make the diagnosis, look for a positive family history and a personal history of easy bruising, mucosal bleeding, bleeding after dental procedures or tonsillectomy, or heavy menstrual bleeding. Laboratory analysis will show a normal PT, a normal or increased aPTT (depending on the degree of Factor VIII deficiency), and an increased bleeding time. The treatment for mild disease is desmopressin (answer B), which increases the production and release of von Willebrand factor from the endothelium. More severe bleeding episodes may require treatment with cryoprecipitate. A few other teaching points… 1) The symptoms here localize the bleeding disorder to the platelets. Remember that clinically, the patient with a platelet disorder is an "oozy bruisy" patient, and tends to suffer from mucosal bleeding. Patients with hemophilas or clotting factor deficiencies can still make platelet plugs, so they don't have as much minor mucosal bleeding. But since these patients can't form a stable mature clot, they are much more at risk for deep tissue bleeding like hemarthrosis. A mnemonic for remembering this is "Petechiae suggest Platelet deficiency; Cavity or joint bleeding suggests Clotting factor deficiency." 2) The PT, aPTT, and bleeding time also localize this patient's bleeding disorder to her platelets. The PT and aPTT measure the function of the coagulation cascade. (In particular, the PT measures the extrinsic pathway and is used to monitor warfarin therapy, while the aPTT measures the intrinsic pathway and is used to monitor heparin therapy.) Meanwhile, the bleeding time evaluates platelet function. This patient has a borderline elevation of her aPTT (caused by mild deficiency of Factor VIII, which is carred on von Willebrand factor) but her primary abnormality is an elevated bleeding time, indicating platelet dysfunction. 3) The family history here also distinguishes between hemophilias and von Willebrand's. Both hemophilia A (deficiency of Factor VIII) and hemophilia B (deficiency of Factor IX, also known as Christmas disease) are X linked disorders. Finding them in females would be very rare. Von Willebrand's disease, however, is autosomal dominant, and should appear in every generation of the family regardless of sex. As a sidenote, a useful mnemonic to remember which clotting factor is deficient in which hemophila is this: Hemophilia A = Hemophilia 8 (say it out loud three times fast and it'll make sense!). Plasmaphresis (answer A) is the treatment for thrombocytopenic thrombotic purpura or TTP. The classic pentad of TTP is microangiopathic hemolytic anemia, thrombocytopenia, mental status changes or neurological abnormalities, fever, and renal dysfunction. Factor VIII concentrate (answer C) is the treatment for hemophilia A. It is true that patients with von Willebrand's disease do sometimes have a deficiency of Factor VIII. However, their deficiency is not due to a lack of production of Factor VIII as it is in hemophiliacs, but rather due to a lack of Factor VIII's carrier protein, von Willebrand factor. You can correct their aPTT by administering Factor VIII, but this won't do anything to help their platelet dysfunction. Administering desmopressin fixes both. Hydroxyurea (answer D) is the treatment of choice for sickle cell disease, where it increases the production of hemoglobin F. It has no role in the treatment of von Willebrand's disease. Splenectomy (answer E) can be used to treat refractory cases of idiopathic thrombocytopenic purpura (ITP) if initial medical treatment with drugs like corticosteroids fails. Of course, to diagnose ITP requires thrombocytopenia, and this patient's platelet count is normal.

C. Factor VIII concentrate (answer C) is the treatment for hemophilia A. It is true that patients with von Willebrand's disease do sometimes have a deficiency of Factor VIII. However, their deficiency is not due to a lack of production of Factor VIII as it is in hemophiliacs, but rather due to a lack of Factor VIII's carrier protein, von Willebrand factor. You can correct their aPTT by administering Factor VIII, but this won't do anything to help their platelet dysfunction. Administering desmopressin fixes both.

D. Hydroxyurea (answer D) is the treatment of choice for sickle cell disease, where it increases the production of hemoglobin F. It has no role in the treatment of von Willebrand's disease.

E. Splenectomy (answer E) can be used to treat refractory cases of idiopathic thrombocytopenic purpura (ITP) if initial medical treatment with drugs like corticosteroids fails. Of course, to diagnose ITP requires thrombocytopenia, and this patient's platelet count is normal.

This question is not currently linked to the learning objective database.

Question problem?

Question # 25

Select the single best answer to the numbered question.

A 27 year old male has had a sore throat for two weeks. The pain has now become so great that he has great difficulty swallowing and has only been able to eat and drink small amounts over the past few days. He has had occasional fevers, but denies cough or coryza. Prior to this, the patient had been very healthy and had not been to a physician for fifteen years. Vital signs on presentation include temperature 38.0 C (100.4 F), pulse 90, blood pressure 118/75, and respirations 14/min. On physical exam, no tonsillar exudates are seen, but there are numerous soft, fluffy white plaques in the patient's tongue and oropharynx. The lesions bleed slightly when they are scraped. Which of the following is the most appropriate next step in the management of this patient?

A. Peripheral blood smear for atypical lymphocytes
B. Rapid antigen detection test for Group A beta-hemolytic Streptococcus
C. Antibody assay for measles virus IgM (MVIgM)
D. Lateral neck radiographs
E. ELISA for HIV-1 antibodies

You did not answer this question.

Explanations:

A. A peripheral blood smear showing large numbers of atypical lymphocytes (answer A) is characteristic of infectious mononucleosis from Epstein-Barr virus infection. This can be a common cause of a sore throat in a young person, and would typically be accompanied by some combination of tonsillopharyngeal exudates, tonsillar or palatal petechiae, cervical adenopathy, and hepatosplenomegaly – but EBV infection would not cause thrush

B. The rapid antigen detection test for GABHS (answer B) is an easy way to diagnose strep throat in the office. (Although this test is specific for Group A strep, a negative test should be followed up by a throat culture to ensure adequate sensitivity.) The key features of strep throat are the presence of sore throat, fever, and cervical adenopathy, and the lack of a cough or runny nose (which suggest viral etiologies). Patients with streptococcal pharyngitis usually have tonsillar exudates as well, and children with the disease sometimes get nausea and vomiting. Most importantly for this question, though, streptococcal infection is not a predisposing factor for oral candidiasis.

C. Antibody assay for measles virus (answer C) is the best way to diagnose acute measles infection. The clinical course of measles infection begins with a fever, which is soon followed by an erythematous maculopapular rash that begins on the face and spreads to the trunk and extremities. Patients with measles also have the "three c's" of cough, coryza, and conjunctivitis. If you selected this answer, you might have thought that the image showed Koplik spots, which are white or bluish-gray dots or "grains of sand" on a red base that appear in the buccal mucosa. This patient's oral lesions are diffuse plaques and are much more consistent with oral candidiasis.

D. Lateral neck radiographs (answer D) can be used in the evaluation of a stable patient with suspected epiglottitis. A protruding epiglottis is seen as a positive "thumb sign." Thanks to vaccination for H.influenza type b, acute epiglottitis is now fairly rare in children and would be very rare in an adult. In children, the triad of drooling, respiratory distress, and dysphagia should raise concern for this disease – which can be a true medical emergency since inflammation of the epiglottis can lead to airway obstruction very quickly.

E. This is a description of candidal thrush. Fungi like candida do not typically infect normal, healthy patients. Patients who are taking immunosuppressive drugs (including steroids), who are receiving hemodialysis, or have diabetes or cancer may get candidal infections, so a good history should first seek for these factors. In their absence, you need to search out causes of acquired immunodeficiency, and the most likely of those is HIV (answer E). This patient's history is concerning for esophageal candidiasis, so evaluation should probably also include EGD. A peripheral blood smear showing large numbers of atypical lymphocytes (answer A) is characteristic of infectious mononucleosis from Epstein-Barr virus infection. This can be a common cause of a sore throat in a young person, and would typically be accompanied by some combination of tonsillopharyngeal exudates, tonsillar or palatal petechiae, cervical adenopathy, and hepatosplenomegaly – but EBV infection would not cause thrush. The rapid antigen detection test for GABHS (answer B) is an easy way to diagnose strep throat in the office. (Although this test is specific for Group A strep, a negative test should be followed up by a throat culture to ensure adequate sensitivity.) The key features of strep throat are the presence of sore throat, fever, and cervical adenopathy, and the lack of a cough or runny nose (which suggest viral etiologies). Patients with streptococcal pharyngitis usually have tonsillar exudates as well, and children with the disease sometimes get nausea and vomiting. Most importantly for this question, though, streptococcal infection is not a predisposing factor for oral candidiasis. Antibody assay for measles virus (answer C) is the best way to diagnose acute measles infection. The clinical course of measles infection begins with a fever, which is soon followed by an erythematous maculopapular rash that begins on the face and spreads to the trunk and extremities. Patients with measles also have the "three c's" of cough, coryza, and conjunctivitis. If you selected this answer, you might have thought that the image showed Koplik spots, which are white or bluish-gray dots or "grains of sand" on a red base that appear in the buccal mucosa. This patient's oral lesions are diffuse plaques and are much more consistent with oral candidiasis. Lateral neck radiographs (answer D) can be used in the evaluation of a stable patient with suspected epiglottitis. A protruding epiglottis is seen as a positive "thumb sign." Thanks to vaccination for H.influenza type b, acute epiglottitis is now fairly rare in children and would be very rare in an adult. In children, the triad of drooling, respiratory distress, and dysphagia should raise concern for this disease – which can be a true medical emergency since inflammation of the epiglottis can lead to airway obstruction very quickly.

This question is not currently linked to the learning objective database.

Question problem?

Question # 26

Select the single best answer to the numbered question.

A 15 year old otherwise healthy female presents to her family physician with right lower quadrant abdominal pain. Although the pain waxes and wanes, it reaches a 7/10 on the pain scale, and ibuprofen has been ineffective. Menarche occurred at age 10 and her menses have always been regular at 26 day cycles. The patient is not sexually active. LMP was 2 weeks ago. Bimanual exam appreciates normal size ovaries, but the patient experiences pain on palpation of the right adnexae. An abdominal ultrasound reveals a 5 cm smooth

thin walled, unilocular cystic structure on the right ovary. Urine pregnancy test is negative and WBC is normal. What is the most likely diagnosis?

A. Polycystic ovarian syndrome
B. Dermoid tumor
C. Endometriosis
D. Physiological ovarian cyst
E. Tubo-ovarian abscess

You did not answer this question.

Explanations:

A. Polycystic ovarian syndrome or PCOS (answer A) is not the most likely diagnosis. PCOS is characterized by ovulatory dysfunction and hyperandrogenism. Adolescent PCOS patients can present with signs of hyperandrogenism including hirsutism, treatment resistant acne, hyperhydrosis, and alopecia or signs of anovulation such as menstrual irregularity and infertility. A polycystic ovary can be seen on ultrasound, but just finding ovary with multiple cysts on ultrasound is not enough to establish the diagnosis of PCOS. In general, the diagnosis includes a combination of menstrual irregularity, hyperandrogenism, and polycystic ovaries.

B. A dermoid tumor (a.k.a. benign cystic teratoma) (answer B) is incorrect. More common in the second and third decades of life, this benign type of ovarian tumor appears as a complex structure on ultrasound with both hypoechoic and brightly echogenic areas intermixed. There are also often areas of calcification, because the tumor can can contain hair, bone, or even teeth!

C. Endometriosis (answer C), the presence of endometrial stroma and glands outside of the uterine cavity, is not the most likely diagnosis. Though endometriosis can present as pain in the adolescent, it would likely not be seen on ultrasound. In adult women, ultrasound can sometimes identify endometriomas (a mass containing islands of the endometrial stroma and glands, also known as “chocolate cysts”), but these rarely occur in adolescence.

D. This question describes an adolescent with a functional ovarian cyst (answer D). Adolescent ovaries often contain multiple follicles in different stages of development. The majority of simple cysts result from the failure of the maturing follicle to ovulate and involute. Physiological cysts are usually asymptomatic initially, but as they get larger they can cause a feelings of pelvic fullness, constipation, and urinary frequency. Ultrasound is the appropriate test to confirm the diagnosis. A few additional teaching points… 1) Regarding management, if a fluid-filled cyst increases in size, is greater than 6 cm, or causes symptoms, a laparoscopic cystectomy is indicated. The cyst wall should be sent for pathological evaluation. Asymptomatic simple cysts less than 6 cm can be observed. 2) Benign physiological cysts will appear as thin-walled and fluid filled on ultrasound. Dermoid tumors or benign cystic teratomas often show calcification on ultrasound or abdominal x-ray. 3) The differential diagnosis of a simple cyst in an adolescent patient includes: obstructive genital lesions, ovarian tumors such as benign cystic teratoma, tubal conditions (ectopic pregnancy) and tuboovarian abcess. Polycystic ovarian syndrome or PCOS (answer A) is not the most likely diagnosis. PCOS is characterized by ovulatory dysfunction and hyperandrogenism. Adolescent PCOS patients can present with signs of hyperandrogenism including hirsutism, treatment resistant acne, hyperhydrosis, and alopecia or signs of anovulation such as menstrual irregularity and infertility. A polycystic ovary can be seen on ultrasound, but just finding ovary with multiple cysts on ultrasound is not enough to establish the diagnosis of PCOS. In general, the diagnosis includes a combination of menstrual irregularity, hyperandrogenism, and polycystic ovaries. A dermoid tumor (a.k.a. benign cystic teratoma) (answer B) is incorrect. More common in the second and third decades of life, this benign type of ovarian tumor appears as a complex structure on ultrasound with both hypoechoic and brightly echogenic areas intermixed. There are also often areas of calcification, because the tumor can can contain hair, bone, or even teeth! Endometriosis (answer C), the presence of endometrial stroma and glands outside of the uterine cavity, is not the most likely diagnosis. Though endometriosis can present as pain in the adolescent, it would likely not be seen on ultrasound. In adult women, ultrasound can sometimes identify endometriomas (a mass containing islands of the endometrial stroma and glands, also known as “chocolate cysts”), but these rarely occur in adolescence. Tuboovarian abcesss or TOA (choice E) is an abcess that involves the fallopian tube and ovary and usually is a consequence of pelvic inflammatory disease, but can follow pelvic surgery or appendicitis. This diagnosis should be considered in a patient with a history of PID who presents with lower abdominal pain. Fever and leukocytosis are often present. Ultrasound shows a thin-walled, cystic, well-demarcated homogeneous mass.

E. Tuboovarian abcesss or TOA (choice E) is an abcess that involves the fallopian tube and ovary and usually is a consequence of pelvic inflammatory disease, but can follow pelvic surgery or appendicitis. This diagnosis should be considered in a patient with a history of PID who presents with lower abdominal pain. Fever and leukocytosis are often present. Ultrasound shows a thin-walled, cystic, well-demarcated homogeneous mass.

This question is not currently linked to the learning objective database.

Question problem?

Question # 27

Select the single best answer to the numbered question.

In order to obtain a job at a prison, a 34 year old man undergoes a required chest x-ray to screen for tuberculosis. A 14mm rounded opacity is noted, surrounded by normal lung tissue. There are small calcifications within the opacity, but no adenopathy or atelectasis is seen radiographically. The patient has never smoked and has no known exposures to tuberculosis. There is no family history of lung cancer or other malignancy. On physical exam, the patient has temperature 37.0 C (98.6 F), pulse 76, blood pressure 130/83, respirations 14/min. Lungs are clear to auscultation and percussion. No nail clubbing is seen. Which of the following is the most appropriate next step in the management of this patient?

A. Repeat chest x-ray in 3 months
B. Begin rifampin, isoniazid, pyrazinamide, and ethambutol
C. Bronchoscopy
D. CT guided needle biopsy
E. Thoracotomy

You did not answer this question.

Explanations:

A. This question stem describes the management of a solitary pulmonary nodule (SPN). The differential diagnosis for such lesions is broad, and includes granulomatous diseases like old or active TB or fungal infections, benign hamartomas, scarring, and of course, cancer. Obviously, the #1 concern with SPNs is that the nodule is cancer, and that you’ll miss a malignancy that would otherwise be small enough to cure with surgery. Ultimately, the workup of a pulmonary nodule hinges on this question: just how likely is it to be malignant? You can divide patients into three categories based on whether their likelihood for malignancy is low, moderate, or high. Patients who have a low risk of malignancy can be followed by re-imaging them in several months to ensure that their lesion has not changed. Patients with a moderate chance of malignancy need further workup to evaluate their nodules. Patients with a high likelihood for malignancy will often proceed directly to thoracotomy or thoracoscopic resection. There are a number of factors that increase the likelihood that a SPN is malignant, and for a patient to be considered low risk, he or she must lack ALL of them. Having one or more of these characteristics places the individual into the moderate or high risk for malignancy and demands further workup or a tissue diagnosis. 1) Age: The risk of malignancy increases with age: only 3% of SPNs are malignant for patients 35-40, while >50% are malignant for patients over 60. Only individuals less than 35 years old are considered “low risk” for malignancy. 2) Size: Larger lesions (>3cm) are more likely malignant. Only SPNs less than 2cm can be considered low risk. 3) Radiographic appearance: Benign lesions tend to have smooth, well-circumscribed borders, while malignant nodules have irregular or spiculated borders. Calcifications are more often seen with benign lesions, although the pattern of calcification is important. 4) Rate of change: The very slowest growing lung cancers have a doubling time of around 400 days. Thus, if a lesion has been stable when compared to prior imaging for two years, it is likely benign. 5) Smoking history: Smoking is obviously far and away the #1 risk factor for lung cancer. Only non-smokers can be considered to have a low risk SPN. In short, then, only nonsmokers under 35 years old who have an SPN less than 2cm (or one that has been radiographically stable for two years) can be considered low risk for SPN. These patients can be managed with follow up imaging, either CXR or CT, every 3-6 months initially (answer A). Rifampin, isoniazid, pyrazinamide, and ethambutol (answer B) is the “R.I.P.E.” therapy for tuberculosis. This four drug cocktail is begun empirically, though ethambutol may be withdrawn depending on the bug’s sensitivity (leaving “R.I.P.” therapy). This patient’s x-ray is not diagnostic of TB, however: radiographically, active pulmonary tuberculosis looks like lobar pneumonia, with ipsilateral hilar adenopathy and atelectasis. Bronchoscopy (answer C), CT guided needle biopsy (answer D), and thoracotomy (answer E) all are techniques that may be used to evaluate lung nodules that are judged to be moderate or high risk for malignancy. The teaching point in this case is that a patient who lacks all risk factors is considered low risk for malignancy and does not require any invasive testing.

B. Rifampin, isoniazid, pyrazinamide, and ethambutol (answer B) is the “R.I.P.E.” therapy for tuberculosis. This four drug cocktail is begun empirically, though ethambutol may be withdrawn depending on the bug’s sensitivity (leaving “R.I.P.” therapy). This patient’s x-ray is not diagnostic of TB, however: radiographically, active pulmonary tuberculosis looks like lobar pneumonia, with ipsilateral hilar adenopathy and atelectasis.

C. Bronchoscopy (answer C), CT guided needle biopsy (answer D), and thoracotomy (answer E) all are techniques that may be used to evaluate lung nodules that are judged to be moderate or high risk for malignancy. The teaching point in this case is that a patient who lacks all risk factors is considered low risk for malignancy and does not require any invasive testing.

D. Bronchoscopy (answer C), CT guided needle biopsy (answer D), and thoracotomy (answer E) all are techniques that may be used to evaluate lung nodules that are judged to be moderate or high risk for malignancy. The teaching point in this case is that a patient who lacks all risk factors is considered low risk for malignancy and does not require any invasive testing.

E. Bronchoscopy (answer C), CT guided needle biopsy (answer D), and thoracotomy (answer E) all are techniques that may be used to evaluate lung nodules that are judged to be moderate or high risk for malignancy. The teaching point in this case is that a patient who lacks all risk factors is considered low risk for malignancy and does not require any invasive testing.

This question is not currently linked to the learning objective database.

Question problem?

Question # 28

Select the single best answer to the numbered question.

A 7 year old girl is brought to her physician because her parents are concerned that she is shorter than all of her classmates. The patient’s past medical history is benign, and she is otherwise healthy and thriving at home and at school. Her mother is 61 in. (155 cm) and her father is 67 in. (170 cm) tall. In reviewing her old records, it appears that her growth velocity is normal. Physical examination shows the child to be in the 3rd percentile for her age for both height and weight. The patient is Tanner stage 1 and is otherwise developmentally appropriate. The remainder of physical exam is normal. What is the most appropriate next step in the management of this patient?

A. Reassurance
B. Bone age
C. HIV testing
D. Serum IGF-1
E. Serum growth hormone level

You did not answer this question.

Explanations:

A. Reassurance (choice A) isn't the most appropriate step. The patient does meet the criteria for short stature, so a work-up is appropriate at this point.

B. This question addresses the appropriate work-up for short stature in an otherwise normal child. Short stature is defined as 2 standard deviations below normal the mean height for children of the same sex and age, so this child fits the bill. The most important initial factor to consider is whether the growth velocity is normal or not. If a child has a very low growth velocity (<5th percentile) or "falls off" the growth curve, you should investigate causes for short stature like endocrinopathies, growth hormone deficiency, malnutrition or abuse, or malignancy. However, the two most common causes of short stature are familial short stature and constitutional delay of growth, and both of these present with a normal growth velocity. The best test to differentiate familial short stature from constitutional delay is bone age (answer B), and that is the most appropriate next step for this patient. For familial short stature, bone age should match chronological age, while in constitutional delay, bone age will lag behind chronological age. A couple of additional teaching points… 1) Bone age is obtained by taking radiographs of the hand and wrist, and may be described as such on the USMLE, so don't be thrown off! 2) A child that has small stature but is overweight for their height may have growth hormone deficiency. Conversely, a child that is underweight for their height may have malnutrition. Reassurance (choice A) isn't the most appropriate step. The patient does meet the criteria for short stature, so a work-up is appropriate at this point. HIV testing (choice C) can be a cause of short stature, but is not an intial test in a patient with a normal growth velocity and the absence of signs and symptoms of illness. Serum IGF-1 (choice D) testing can be done if growth hormone deficiency is suspected, but is not an initial test in a short stature patient with normal growth velocity. Serum growth hormone level (choice E) is an appropriate test if growth hormone deficiency is suspected. Patients with GH deficiency present with severe growth failure and delayed bone age.

C. HIV testing (choice C) can be a cause of short stature, but is not an intial test in a patient with a normal growth velocity and the absence of signs and symptoms of illness.

D. Serum IGF-1 (choice D) testing can be done if growth hormone deficiency is suspected, but is not an initial test in a short stature patient with normal growth velocity.

E. Serum growth hormone level (choice E) is an appropriate test if growth hormone deficiency is suspected. Patients with GH deficiency present with severe growth failure and delayed bone age.

This question is not currently linked to the learning objective database.

Question problem?

Question # 29

Select the single best answer to the numbered question.

A 26 year old G2P1 is at 28 weeks gestation of an uncomplicated pregnancy. The patient denies decreased fetal movement, dysuria, urinary frequency or urgency, vaginal bleeding or discharge, and nausea and vomiting. Medications include a prenatal vitamin and folate and iron supplements. Physical exam shows uterine fundal height 29 cm with fetal heart rate in the 150s. Laboratory testing shows: Fasting serum glucose 115mg/dL; Maternal blood type B+. The patient's urinalysis shows the following. Color: yellow; Specific gravity: 1.020 (normal: 1.002 – 1.030); pH: 5.2; Glucose: negative; Ketones: negative; Protein: 1+; RBC: negative; Leukocyte esterase: trace; Nitrite: 2+; Urobilinogen: 0.3 EhrU/dL (normal: 0.2-1.0 EhrU/dL). Urine culture grows 25,000 cfu/mL Gram negative rods. Cervical cultures for N.gonnorheae and Chlamydia are negative. Which of the following is the most appropriate next step in the management of this patient?

A. Administer RhoGAM
B. Measure maternal serum alpha feto-protein (MSAFP)
C. Begin insulin therapy
D. Begin cephalexin
E. Reassurance and routine follow-up

You did not answer this question.

Explanations:

A. Administering RhoGAM (answer A) is an appropriate intervention if the mother were Rh antibody negative. This is typically done around 28 weeks gestation.

B. Measuring the maternal serum alpha feto-protein (answer B) is done as part of maternal screening for fetal birth defects. It is offered to women between 15-20 weeks gestation, so this patient is too far along in her pregnancy to benefit from it. Accurate gestational dates are extremely important in interpreting this test, and elevated levels can indicate multiple gestations, neural tube defects, or abdominal wall defects like gastroschisis or omphalocele.

C. Beginning insulin therapy (answer C) is not indicated at this time because the patient does not meet criteria for diabetes. If this patient had gestational diabetes mellitus, insulin would be the drug of choice if the patient could not maintain glucose control with diet and exercise alone - oral hypoglycemics are contraindicated since they can cross the placenta and cause fetal hypoglycemia. The diagnosis of gestational diabetes requires two abnormal tests: fasting serum glucose >126 mg/dL, a random glucose of >200 mg/dL, or an abnormal glucose tolerance test result (>130 or 135 for a one hour test, confirmed by abnormal results on the three hour test).

D. Key teaching point: Bacteruria of pregnancy, even if asymptomatic, should be treated with antibiotics to reduce the risk of pyelonephritis. A urinary tract infection is normally defined as significant bacteruria (usually >100,000 cfu/mL) in the presence of symptoms (dysuria, urinary urgency or frequency). Pregnant women are the exception to this, however, and even women who do not have symptoms should be treated for UTI. If left untreated, some 28% of women with asymptomatic bacteruria of pregnancy will go on to develop pyelonephritis! If this patient were not pregnant, treating her for a UTI would be of debatable benefit given her lack of symptoms and low number of bacteria on culture. Because she is pregnant, however, she should receive an antibiotic like cephalexin (answer D). In addition to cephalosporins like cephalexin, commonly used agents to treat UTIs in pregnant women include amoxicillin, amoxicillin/clavulanate, nitrofurantoin, and sulfonamides. All of these agents have adequate urinary penetration and reasonable activity against the common causative organisms (like E.coli, Klebsiella, Group B strep, etc.). However, sulfonamides (like TMP/SMZ) should be avoided in the last trimester because of the risk of causing hyperbilirubinemia of the newborn. Administering RhoGAM (answer A) is an appropriate intervention if the mother were Rh antibody negative. This is typically done around 28 weeks gestation. Measuring the maternal serum alpha feto-protein (answer B) is done as part of maternal screening for fetal birth defects. It is offered to women between 15-20 weeks gestation, so this patient is too far along in her pregnancy to benefit from it. Accurate gestational dates are extremely important in interpreting this test, and elevated levels can indicate multiple gestations, neural tube defects, or abdominal wall defects like gastroschisis or omphalocele. Beginning insulin therapy (answer C) is not indicated at this time because the patient does not meet criteria for diabetes. If this patient had gestational diabetes mellitus, insulin would be the drug of choice if the patient could not maintain glucose control with diet and exercise alone - oral hypoglycemics are contraindicated since they can cross the placenta and cause fetal hypoglycemia. The diagnosis of gestational diabetes requires two abnormal tests: fasting serum glucose >126 mg/dL, a random glucose of >200 mg/dL, or an abnormal glucose tolerance test result (>130 or 135 for a one hour test, confirmed by abnormal results on the three hour test). Reassurance and follow up (answer E) would be inappropriate because this patient's urine culture shows bacteria. She should be treated for asymptomatic bacteruria of pregnancy with antibiotics.

E. Reassurance and follow up (answer E) would be inappropriate because this patient's urine culture shows bacteria. She should be treated for asymptomatic bacteruria of pregnancy with antibiotics.

This question is not currently linked to the learning objective database.

Question problem?

Question # 30

Select the single best answer to the numbered question.

A 72 year old man comes to his physician because he has been feeling sad and "thinks he needs help." For the past several weeks, he has noted decreased appetite and an 8 lb weight loss, and he has seldom left his house, even to participate in his weekly bowling league. While he is able to fall asleep normally, he awakens after 3-4 hours and is unable to fall asleep again. He has also had increasing difficulty balancing his checkbook and writing his Christmas cards, and believes that he just can't stay focused enough to complete these tasks. Six months ago, his wife of fifty years died, and he often feels guilty that he did not treat her as kindly as he should have. When he thinks about his wife, he thinks that he would rather be dead than without her, although he denies having a plan to harm himself. Past medical history includes coronary artery disease and gout. Medications include lisinopril, allopurinol, metoprolol, furosemide, and aspirin. Which of the following is the most likely diagnosis in this patient?

A. Adjustment disorder
B. Bereavement
C. Major depressive disorder
D. Dysthymic disorder
E. Alzheimer's dementia

You did not answer this question.

Explanations:

A. Adjustment disorder (answer A) can present with a depressed mood. However, the symptoms should emerge within three months of an identifiable stressor and should resolve within six months. Additionally, this patient's symptoms are more pathological than would be explained by adjustment disorder.

B. Bereavement (answer B) is suggested by the recent loss of the patient's wife. Normal bereavement can cause depressive symptoms and even some symptoms that appear psychotic, such as hearing the loved one's voice. However, the diagnosis of bereavement is superseded by the diagnosis of major depression if there is severe impairment of functioning or suicidal ideation. Even if these factors are lacking, you should diagnose a major depressive episode if the patient meets criteria for two months.

C. This is a fairly clear-cut case of major depressive disorder (answer C), with a few twists thrown in (such as the recent death of the patient's wife) to try to throw you off. Although this patient does have features that might initially appear suggestive for the diagnosis of adjustment disorder or bereavement, it is important to remember that major depression would be the supraordinate diagnosis under the DSM-IV. That is, if a patient were to meet the criteria for, say, BOTH bereavement and major depression, the patient would be treated for major depression, since it has the more stringent criteria and carries the worse prognosis. To remember the criteria for major depression, remember the SIG: E CAPS 25 mnemonic. S – Sleep disturbance, either hypersomnia or insomnia; I – loss of Interest in usually pleasurable activities; G – feelings of Guilt or worthlessness; E – decreased Energy; C – decreased Concentration; A – Appetite disturbance, either increased or decreased; P – Psychomotor retardation; S – Suicidal ideation; 2 – symptoms must be present for at least a 2 week period; 5 – five symptoms required from the list above to make the diagnosis (and at least one of them must be either depressed mood or anhedonia). As an additional teaching point, although it did not appear in the answer choices, substance-induced mood disorder is also a possibility for this patient. It's not only drugs like cocaine and alcohol that cause substance-induced mood disorders – this patient's medication list includes metoprolol, and beta-blockers are often a cause of depression. This "trick" appears frequently enough on the USMLE that you should always watch out for it, although you should be given some clue that the symptoms began at the same time as the drug. Adjustment disorder (answer A) can present with a depressed mood. However, the symptoms should emerge within three months of an identifiable stressor and should resolve within six months. Additionally, this patient's symptoms are more pathological than would be explained by adjustment disorder. Bereavement (answer B) is suggested by the recent loss of the patient's wife. Normal bereavement can cause depressive symptoms and even some symptoms that appear psychotic, such as hearing the loved one's voice. However, the diagnosis of bereavement is superseded by the diagnosis of major depression if there is severe impairment of functioning or suicidal ideation. Even if these factors are lacking, you should diagnose a major depressive episode if the patient meets criteria for two months. The key diagnostic feature of dysthymic disorder (answer D) is its chronicity: symptoms must be present for at least 2 years, while this patient's symptoms have been present for a much shorter period of time. While dysthymia shares symptoms with major depression, the symptoms of dysthymia tend to be fewer and less pronounced. The symptoms of dysthymia alone do not meet the criteria for major depressive disorder. Mood symptoms are seen in Alzheimer's dementia (answer E)

tend to be fewer and less pronounced. The symptoms of dysthymia alone do not meet the criteria for major depressive disorder. Mood symptoms are seen in Alzheimer's dementia (answer E), so Alzheimer's can masquerade as depression and vice versa. This patient's difficulty writing letters and balancing his checkbook could be signs of dementia, but in this context they are more likely due to impaired concentration secondary to depression.

D. The key diagnostic feature of dysthymic disorder (answer D) is its chronicity: symptoms must be present for at least 2 years, while this patient's symptoms have been present for a much shorter period of time. While dysthymia shares symptoms with major depression, the symptoms of dysthymia tend to be fewer and less pronounced. The symptoms of dysthymia alone do not meet the criteria for major depressive disorder.

E. Mood symptoms are seen in Alzheimer's dementia (answer E), so Alzheimer's can masquerade as depression and vice versa. This patient's difficulty writing letters and balancing his checkbook could be signs of dementia, but in this context they are more likely due to impaired concentration secondary to depression.

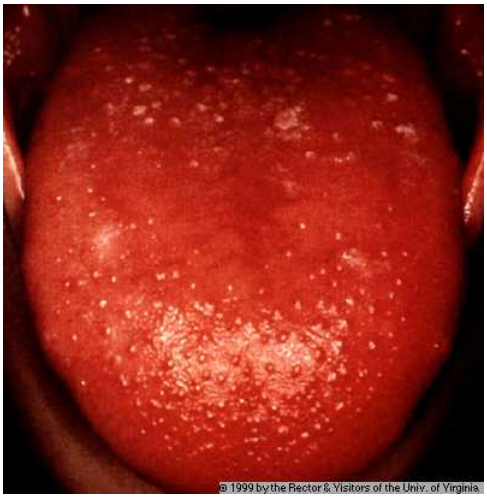
This question is not currently linked to the learning objective database.

Question problem?

Question # 31

Select the single best answer to the numbered question.

A 2 year old male child returns to the physician with continued fever and a rash. Eight days ago, he had the abrupt onset of fever, which was later accompanied by irritability and anorexia. Fevers to 104 F (40 C) have continued and have not decreased despite his mother's repeated administration of acetaminophen and ibuprofen. Physical exam reveals inflammation of the conjunctiva bilaterally without purulent drainage. There is diffuse cervical lymphadenopathy and peeling of the skin around the fingernails. Scattered target-like, erythematous, macular lesions are present on the trunk and extremities. There are no tonsillar exudates, but a photo of the child's tongue is seen here. Which of the following is the most likely diagnosis in this patient?



A. Drug hypersensitivity
B. Scarlet fever
C. Erythema infectiosum
D. Kawasaki disease
E. Rocky Mountain spotted fever

You did not answer this question.

Explanations:

A. Drug hypersensitivity reactions (answer A) can cause a variety of skin lesions, but would not cause the constellation of symptoms seen in this patient.

B. Scarlet fever (answer B) is an exotoxin-mediated complication of Group A strep infection. The key diagnostic features include a history or physical exam findings consistent with strep infection (especially streptococcal pharyngitis), a "strawberry tongue," and the famous rash. The rash of scarlet fever is – as its name implies – scarlet, and may resemble a "boiled lobster" or sunburn. Later, punctate lesions the size of pinheads give the skin a rough, sandpaper-like texture. There can be skin peeling as well, but this is a late occurrence.

C. Erythema infectiosum (answer C) is a viral exanthem of childhood caused by parvovirus B19 infection. It is alternately known as "fifth disease." The rash of erythema infectiosum has the so-called "slapped cheeks" appearance.

D. This is Kawasaki disease (answer D). Because of the likelihood of the disease causing life-threatening coronary artery aneurysms, this is a can't-miss diagnosis. A few other teaching points… 1) The diagnosis of Kawasaki disease is a clinical one, requiring fever of at least 5 days duration and at least four of the following: peripheral edema, desquamation (especially of the fingertips, palms, and soles), bilateral conjunctivitis; polymorphous, nonvesicular rash; cervical lymphoadenopathy (often unilateral); dry or fissured lips; or "strawberry tongue." For a concise review of Kawasaki disease, try this one from the BMJ: http://www.bmj.com/archive/7104/7104e2.htm 2) The "strawberry tongue" seen in the photograph is characteristic of scarlet fever or Kawasaki disease, so whenever you see it (or a description of it) think of these two things first. 3) The most serious and feared complication of Kawasaki disease is the formation of coronary artery aneurysms, and all patients need serial echocardiograms to monitor for the formation of aneurysms. Treatment of Kawasaki disease includes intravenous immunoglobulin (IVIg) and corticosteroids, as well as aspirin to prevent thrombosis. Drug hypersensitivity reactions (answer A) can cause a variety of skin lesions, but would not cause the constellation of symptoms seen in this patient. Scarlet fever (answer B) is an exotoxin-mediated complication of Group A strep infection. The key diagnostic features include a history or physical exam findings consistent with strep infection (especially streptococcal pharyngitis), a "strawberry tongue," and the famous rash. The rash of scarlet fever is – as its name implies – scarlet, and may resemble a "boiled lobster" or sunburn. Later, punctate lesions the size of pinheads give the skin a rough, sandpaper-like texture. There can be skin peeling as well, but this is a late occurrence. Erythema infectiosum (answer C) is a viral exanthem of childhood caused by parvovirus B19 infection. It is alternately known as "fifth disease." The rash of erythema infectiosum has the so-called "slapped cheeks" appearance. Rocky Mountain spotted fever (answer E) causes fever and nonspecific signs of illness, followed by the eruption of a vasculitic rash on the extremities. It is spread by ticks carrying Rickettsia rickettsii, and is treated with antibiotics (usually doxycycline).

E. Rocky Mountain spotted fever (answer E) causes fever and nonspecific signs of illness, followed by the eruption of a vasculitic rash on the extremities. It is spread by ticks carrying Rickettsia rickettsii, and is treated with antibiotics (usually doxycycline).

This question is not currently linked to the learning objective database.

Question problem?

Question # 32

Select the single best answer to the numbered question.

A 54 year old man presents with acute knee pain which began last night while he was sleeping. He is now in 9/10 pain and says that he is hardly able to walk secondary to his inability to flex his left knee. Past medical history includes hypertension, nephrolithiasis, and two similar episodes of pain that have occurred over the past 16 months, one in his toe and another in his left knee. On physical exam, the patient is visibly uncomfortable and is holding his leg perfectly still. There is marked erythema and soft tissue swelling surrounding the knee. Passive range of motion is severely diminished secondary to the patient's pain. Analysis of joint aspirate shows a WBC of 60,000 and abundant needle shaped crystals that show negative birefringence under a polarizing filter. Treatment is initiated with colchicine, prednisone, and indomethacin. What is the next most appropriate step in the management of this patient?

A. 24 hour urine collection for uric acid
B. Measurement of serum uric acid
C. Plain film x-rays of knee

D. Begin therapy with allopurinol

E. Begin therapy with probenecid

You did not answer this question.

Explanations:

A. This can be a difficult and tricky question since you are asked to choose between further diagnostic workup and beginning treatment. The illness here is gout. Given that this patient has had multiple episodes of gout, he is a prime candidate for the addition of some sort of prophylactic medication to prevent future attacks. The question is, which medication should be used? A 24 hour urine collection for uric acid will help you sort this out (answer A), and is the most appropriate option among those listed. Patients with gout can loosely be grouped into two categories: those who produce excessive amounts of uric acid ("overproducers") and those who have reduced urinary excretion of uric acid ("undersecreters"). If you collect a 24 hour urine sample and find less than 600 mg of uric acid, the person is an undersecreter, and would benefit from uricosuric therapy like probenecid (answer E) or sulfinpyrazone. These medications block the reabsorption of uric acid in the renal tubule and cause increased clearance of uric acid in the urine. However, if there is greater than 600 mg of uric acid, the patient is more likely an overproducer and would likely benefit from allopurinol (answer D) to reduce the production of uric acid. Allopurinol is a xanthine oxidase inhibitor and results in decreased production of purines and uric acid. Starting either allopurinol or probenecid in an acute flare of gout is contraindicated, however, since sudden changes in the serum uric acid concentration often cause another attack. A few brief teaching points about this question… 1) This patient presents with an acute monoarticular arthritis. The differential diagnosis for monoarticular arthritis includes septic arthritis, gout, pseudogout, reactive arthritis, and of coure early chronic arthritis. Given that this patient has had previous similar episodes, he is very unlikely to have septic arthritis, but that is always the first thing to rule out on the differential. 2) Gout and pseudogout cannot reliably be distinguished on clinical grounds – you need to look at the crystals to make a definitive diagnosis. You may be asked to use the description of the joint aspiration findings to distinguish between gout and pseudogout on the USMLE. Gout crystals are made of monosodium urate, and are needle-shaped and negatively birefringent (they turn blue when perpendicular to the axis of the polarizer). Pseudogout crystals are rhomboidal and are only weakly positively birefringent (they are yellow when perpendicular to the axis of the polarizer). 3) The treatment for an acute gout flare consists of the triad of NSAIDs, colchicine, and steroids. Acutely, the treatment of pseudogout is the same. Measuring the serum uric acid level (answer B) is not helpful. Hyperuricemia is seen in some patients with gout, but is neither sensitive nor specific enough to be of any diagnostic utility. Finding a high level (or a low level, for that matter) would not change your management of this patient in any case – the patient clinically has gout, and the joint aspiration findings confirm that diagnosis. Plain film x-rays of the patient's elbow (answer C) are also not likely to help you in this situation. In the context of a gout flare, you'll probably only see soft tissue swelling, though if a patient has an extensive history of gout you might see joint erosions or calcified tophi. In this case, however, you have confirmed the diagnosis of gout with a joint aspiration, and x-ray findings are not likely to change your management plan.

B. Measuring the serum uric acid level (answer B) is not helpful. Hyperuricemia is seen in some patients with gout, but is neither sensitive nor specific enough to be of any diagnostic utility. Finding a high level (or a low level, for that matter) would not change your management of this patient in any case – the patient clinically has gout, and the joint aspiration findings confirm that diagnosis.

C. Plain film x-rays of the patient's elbow (answer C) are also not likely to help you in this situation. In the context of a gout flare, you'll probably only see soft tissue swelling, though if a patient has an extensive history of gout you might see joint erosions or calcified tophi. In this case, however, you have confirmed the diagnosis of gout with a joint aspiration, and x-ray findings are not likely to change your management plan.

D. Patients with gout can loosely be grouped into two categories: those who produce excessive amounts of uric acid ("overproducers") and those who have reduced urinary excretion of uric acid ("undersecreters"). If you collect a 24 hour urine sample and find less than 600 mg of uric acid, the person is an undersecreter, and would benefit from uricosuric therapy like probenecid (answer E) or sulfinpyrazone. These medications block the reabsorption of uric acid in the renal tubule and cause increased clearance of uric acid in the urine. However, if there is greater than 600 mg of uric acid, the patient is more likely an overproducer and would likely benefit from allopurinol (answer D) to reduce the production of uric acid. Allopurinol is a xanthine oxidase inhibitor and results in decreased production of purines and uric acid. Starting either allopurinol or probenecid in an acute flare of gout is contraindicated, however, since sudden changes in the serum uric acid concentration often cause another attack.

E. Patients with gout can loosely be grouped into two categories: those who produce excessive amounts of uric acid ("overproducers") and those who have reduced urinary excretion of uric acid ("undersecreters"). If you collect a 24 hour urine sample and find less than 600 mg of uric acid, the person is an undersecreter, and would benefit from uricosuric therapy like probenecid (answer E) or sulfinpyrazone. These medications block the reabsorption of uric acid in the renal tubule and cause increased clearance of uric acid in the urine. However, if there is greater than 600 mg of uric acid, the patient is more likely an overproducer and would likely benefit from allopurinol (answer D) to reduce the production of uric acid. Allopurinol is a xanthine oxidase inhibitor and results in decreased production of purines and uric acid. Starting either allopurinol or probenecid in an acute flare of gout is contraindicated, however, since sudden changes in the serum uric acid concentration often cause another attack.

This question is not currently linked to the learning objective database.

Question problem?

Question # 33

Select the single best answer to the numbered question.

A 24 year old male comes to his physician after noticing multiple "bumps" on his hands and legs. There is a burning sensation in the lesions, but they do not itch. Eight days prior, the patient was found to have cold agglutinins and was started on erythromycin to treat Mycoplasma pneumoniae. The patient denies any recent travel or sick contacts, but does work with immigrants at his job as an immigration officer. Physical examination reveals numerous vesiculobullous lesions symmetrically on the palms of the hands and on the extensor surfaces of the arms and lower legs. A photograph of this patient's lesions is seen here. Which of the following is the most likely diagnosis in this patient?

A. Erythema multiforme

B. Smallpox

C. Fixed drug eruption

D. Stevens-Johnson syndrome

E. Rocky Mountain spotted fever

You did not answer this question.

Explanations:

A. This is one of the images that you should commit to memory. Note the multiple concentric lesions with a red center, a pale middle zone, and a dark outer ring – these are the "target lesions" of erythema multiforme (answer A). Although a number of cases of erythema multiforme are idiopathic, there are a variety of triggers that have been identified, from viral infections (like HSV and hepatitis viruses), bacterial infections (including Mycoplasma and Yersinia enterocolitica), and drugs (most notably antibiotics and NSAIDs). Because of the threat of bioterrorism, smallpox (answer B) has made frequent appearances on board examinations in recent years. Patients have a viral prodrome followed by the eruption of the characteristic rash that begins as macules and progresses rapidly to small papules (see these images: http://images.google.com/images?hl=en&q=smallpox&btnG=Search+Images). Because the disease is so contagious among patients who have not been immunized, immediate recognition and quarantine is absolutely imperative. Fixed drug eruptions (answer C) usually appear as an edematous plaque with a bullous center. As the lesion resolves, it leaves post-inflammatory pigmentation, but the hallmark of diagnosis is that if the patient is re-exposed to the offending drug, he or she will get the same lesion in exactly the same place! Common drug culprits include tetracycline antibiotics, barbiturates, sulfonamides, NSAIDs, and phenolphthalein. The Stevens-Johnson syndrome (answer D) used to be considered an extreme version of erythema multiforme, but now is considered a separate entity. They key features are 1) widespread blisters and 2) mucous membrane eruptions. There is also epidermal detachment – if the detachment is greater than 30% of the total body surface area, the diagnosis is toxic epidermal necrolysis (TEN), the most extreme variant of Stevens-Johnson. Rocky Mountain spotted fever (answer E) does cause a rash on the palms and soles, and thus it should be on the short list (along with secondary syphilis, erythema multiforme, and Kawasaki's disease) when you see a patient with this complaint. The illness is caused by Rickettsia rickettsii, and is spread by tick bites. Treatment is usually with doxycycline, though other antibiotics can be equally as effective.

B. Because of the threat of bioterrorism, smallpox (answer B) has made frequent appearances on board examinations in recent years. Patients have a viral prodrome followed by the eruption of the characteristic rash that begins as macules and progresses rapidly to small papules (see these images: http://images.google.com/images?hl=en&q=smallpox&btnG=Search+Images). Because the disease is so contagious among patients who have not been immunized, immediate recognition and quarantine is absolutely imperative.

C. Fixed drug eruptions (answer C) usually appear as an edematous plaque with a bullous center. As the lesion resolves, it leaves post-inflammatory pigmentation, but the hallmark of diagnosis is that if the patient is re-exposed to the offending drug, he or she will get the same lesion in exactly the same place! Common drug culprits include tetracycline antibiotics, barbiturates, sulfonamides, NSAIDs, and phenolphthalein.

D. The Stevens-Johnson syndrome (answer D) used to be considered an extreme version of erythema multiforme, but now is considered a separate entity. They key features are 1) widespread blisters and 2) mucous membrane eruptions. There is also epidermal detachment – if the detachment is greater than 30% of the total body surface area, the diagnosis is toxic epidermal necrolysis (TEN), the most extreme variant of Stevens-Johnson.

E. Rocky Mountain spotted fever (answer E) does cause a rash on the palms and soles, and thus it should be on the short list (along with secondary syphilis, erythema multiforme, and Kawasaki's disease) when you see a patient with this complaint. The illness is caused by Rickettsia rickettsii, and is spread by tick bites. Treatment is usually with doxycycline, though other antibiotics can be equally as effective.

This question is not currently linked to the learning objective database.

Question problem?

Question # 34

Select the single best answer to the numbered question.

A mother brings her 5 year old son to the doctor because he has been scratching an area on his chest for the past week. The itching is much worse at night. The patient has not had a known contact with poison ivy, has no history of a tick bite and his mother says that no one else has a rash like this. The child has been afebrile and has not been acting sick. There is a family history of eczema and so the patient's mother used some of her own steroid cream on her son's rash for the past three days, but this was not helpful. Physical exam reveals an area of small erythematous papules that are excoriated and tipped with blood crusts on his right upper chest extending under his right arm. There are several faint thin, brownish lines just under the surface of the skin. Several of the web spaces between his fingers also seem to have a similar appearing rash, as do the bottom of his feet. What is the most likely diagnosis in this patient?

A. Contact dermatitis
B. Atopic dermatitis
C. Scabies
D. Molluscum contagiosum
E. Skin atrophy and telangiectasias from the topical steroids

You did not answer this question.

Explanations:

A. Contact dermatitis (choice A), a rash resulting from direct skin exposure to an allergen or an irritant, is almost always on the differential diagnosis for a rash. The rash is usually intensely pruritic and if allergic in nature, can occur up to two weeks after exposure. The rash itself is usually papular and erythematous with indistinct margins, distributed along areas of exposure. Fluid collects in the epidermis causing vesicles and oozing. Remember that the poison ivy/oak/sumac group usually occur in linear patterns from kids brushing against the plant. When trying to determine the cause, looking at the distribution can be helpful (i.e. scalp involvement - think about hair products; face - think about cosmetics and aftershaves, etc…).

B. Atopic dermatitis or eczema (choice B) is another common type of rash considered to be familial and and allergic in nature. Most eczema first presents before age 7 and often occurs as part of the "allergic triad" with asthma and allergic rhinitis. The rash usually is intensely pruritic, appearing as erythematous patches with scaling, but can also be vesicular or papular at different stages. In children, eczema usually occurs on the face, scalp, extremities, diaper area and trunk, while in adults it tends to occur on in the flexural areas. Eczema is a clinical diagnosis and is treated with topical steroids and UV light therapy.

C. This is scabies (answer C). This problem is caused by a small mite that prduces pruritis out of proportion to the appearance of the rash. Small erythematous papules with excoriations is classic, as is the description of lesions occurring in the web spaces of the fingers. The burrow (thin gray-red lines under the skin) of the mite is pathognomonic when correctly identified, but it can be difficult to see, especially when excoriations or secondary infection are present. The rash can also appear on the flexor aspect of the wrists, anterior and posterior axillary folds, skin around the nipples, periumbilicaly, around the genital area and on the buttocks, thighs and feet, but the back and head are usually spared. A couple of other teaching points… 1) Diagnosis is based on history and physical exam focusing on the lesion distribution and appearance of the rash. It is possible to confirm the diagnosis by performing skin scrapings, but this is not necessary and has a low sensitivity (so don't use skin scrapings to rule out scabies). In difficult cases, a therapeutic trial of anti-scabies medication is often the best way to pin down the diagnosis. 2) The first-line treatment of scabies includes topical permethrin cream or oral ivermectin. Lindane cream can be used, but is second-line in the pediatric population due to its toxicity. The key to successful eradication is to treat all family members and close contacts at the same time. Fomite transfer is not as much of a concern as it is for head lice, but precautions should be taken including bagging personal items for 3 days and then washing them. Contact dermatitis (choice A), a rash resulting from direct skin exposure to an allergen or an irritant, is almost always on the differential diagnosis for a rash. The rash is usually intensely pruritic and if allergic in nature, can occur up to two weeks after exposure. The rash itself is usually papular and erythematous with indistinct margins, distributed along areas of exposure. Fluid collects in the epidermis causing vesicles and oozing. Remember that the poison ivy/oak/sumac group usually occur in linear patterns from kids brushing against the plant. When trying to determine the cause, looking at the distribution can be helpful (i.e. scalp involvement - think about hair products; face - think about cosmetics and aftershaves, etc…). Atopic dermatitis or eczema (choice B) is another common type of rash considered to be familial and and allergic in nature. Most eczema first presents before age 7 and often occurs as part of the "allergic triad" with asthma and allergic rhinitis. The rash usually is intensely pruritic, appearing as erythematous patches with scaling, but can also be vesicular or papular at different stages. In children, eczema usually occurs on the face, scalp, extremities, diaper area and trunk, while in adults it tends to occur on in the flexural areas. Eczema is a clinical diagnosis and is treated with topical steroids and UV light therapy. Molluscum contagiosum (choice D) is a common chronic localized infection caused by the pox virus. The rash appears as flesh colored, dome-shaped umbilicated papules on any part of the body. It is spread by direct skin to skin contact, autoinoculation, and from fomites. Diagnosis is usually made by the classic appearance, but histological exam can help. This infection is usually self-limited and will resolve over several months in the immunocompetent individual. Treatment outside of the genital area is for cosmetic reasons only and generally not done; it includes curettage, cryotherapy, and laser therapy. Cutaneous effects of steroid use such as skin atrophy and telangiectasias (choice E) is not the best answer. In general, it takes 2-3 weeks before this would occur even with medium to high potency steroids. Areas where the skin is thin are the most likely to be damaged by chronic high potency topical steroids. The atrophy can be reversible if therapy is terminated as soon as the changes are first noticed, otherwise the steroid induced changes can be everlasting. This patient has not been using his mom's steroid cream long enough to cause skin damage from even a high-potency topical steroid. However, the patient's mom should be told about the dangers of steroid creams and instructed to stop using the cream on her son.

D. Molluscum contagiosum (choice D) is a common chronic localized infection caused by the pox virus. The rash appears as flesh colored, dome-shaped umbilicated papules on any part of the body. It is spread by direct skin to skin contact, autoinoculation, and from fomites. Diagnosis is usually made by the classic appearance, but histological exam can help. This infection is usually self-limited and will resolve over several months in the immunocompetent individual. Treatment outside of the genital area is for cosmetic reasons only and generally not done; it includes curettage, cryotherapy, and laser therapy.

E. Cutaneous effects of steroid use such as skin atrophy and telangiectasias (choice E) is not the best answer. In general, it takes 2-3 weeks before this would occur even with medium to high potency steroids. Areas where the skin is thin are the most likely to be damaged by chronic high potency topical steroids. The atrophy can be reversible if therapy is terminated as soon as the changes are first noticed, otherwise the steroid induced changes can be everlasting. This patient has not been using his mom's steroid cream long enough to cause skin damage from even a high-potency topical steroid. However, the patient's mom should be told about the dangers of steroid creams and instructed to stop using the cream on her son.

This question is not currently linked to the learning objective database.

Question problem?

Question # 35

Select the single best answer to the numbered question.

A 36 year old G1P0A0 at 18 weeks gestation presents for routine prenatal care. She has no complaints and has felt the baby moving normally. Her only medication is a prenatal vitamin. Family history is significant for mental retardation in a nephew. Physical examination shows a fundal height of 17 cm and a fetal heart rate in the 160s by Doppler ultrasonography. Laboratory analysis shows alpha fetoprotein to be elevated to 3.5 multiples of the median (MoM). Unconjugated estriol and beta-hCG are within normal limits. This woman's fetus is most likely to have which of the following conditions?

A. Fragile X syndrome
B. Cystic fibrosis
C. Anencephaly
D. Trisomy 18
E. Trisomy 21

You did not answer this question.

Explanations:

A. Fragile X syndrome (answer A) is the most common inherited cause of mental retardation after trisomy 21. It is a trinucleotide repeat disorder, and must be diagnosed with molecular DNA testing. There is no specific pre-natal screening available, although mothers who are thought to be at risk of being carriers may elect to have themselves cytogenetically tested.

B. Testing for carriers of cystic fibrosis (answer B) is also available, and is frequently offered to parents who are at risk (such as those who have relatives with CF). The testing is based on molecular genetics, however – MSAFP plays no role in determining who carries CF mutations.

C. Alpha fetoprotein (AFP) is a plasma protein that is normally produced in the fetal liver. Since it crosses the placenta from the fetal circulation, measurement of the alpha fetoprotein in the maternal serum (the MSAFP) is a useful screening test for fetal abnormalities. Grossly, you can think of an increased AFP as being caused by any defect in which the fetal body cavity is left open. These include neural tube defects like anencephaly (answer C) or myelomeningocele and abdominal wall defects like gastroschisis or omphalocele. Obviously, of course, a larger fetus produces more AFP than a smaller one, so the interpretation of MSAFP results depends on having accurate gestational dates. Therefore, the two most common non-pathological reasons for an elevated MSAFP are multiple gestations and inaccurate dates. Finding a DECREASED MSAFP is also a sign of diseases – usually chromosomal abnormalities. Both trisomy 18 (answer D) and Down's syndrome or trisomy 21 (answer E) are associated with decreased AFP (usually <0.5 MoM). Fragile X syndrome (answer A) is the most common inherited cause of mental retardation after trisomy 21. It is a trinucleotide repeat disorder, and must be diagnosed with molecular DNA testing. There is no specific pre-natal screening available, although mothers who are thought to be at risk of being carriers may elect to have themselves cytogenetically tested. Testing for carriers of cystic fibrosis (answer B) is also available, and is frequently offered to parents who are at risk (such as those who have relatives with CF). The testing is based on molecular genetics, however – MSAFP plays no role in determining who carries CF mutations.

D. Finding a DECREASED MSAFP is also a sign of diseases – usually chromosomal abnormalities. Both trisomy 18 (answer D) and Down's syndrome or trisomy 21 (answer E) are associated with decreased AFP (usually <0.5 MoM).

E. Finding a DECREASED MSAFP is also a sign of diseases – usually chromosomal abnormalities. Both trisomy 18 (answer D) and Down's syndrome or trisomy 21 (answer E) are associated with decreased AFP (usually <0.5 MoM).

This question is not currently linked to the learning objective database.

Question problem?

Question # 36

Select the single best answer to the numbered question.

A 3 year old male is brought to the physician by his mother after she noted multiple bruises on his body. She denies any recent trauma. Two weeks ago, the patient had three days of cough and low-grade fever that resolved without antibiotic therapy, but since that time he has been well. Past medical history is otherwise unremarkable. The patient is up-to-date on all required immunizations. On physical exam, the patient is afebrile and playing happily at his mother's feet in the examination room. There are no peritonsillar exudates. Tympanic membranes are clear. There is no abdominal tenderness or organomegaly. Examination of the skin shows diffuse and numerous petechiae and purpura. Laboratory analysis reveals the following: WBC 7.5 k/mcL; hemoglobin 12 g/dL; hematocrit 36%; platelets 27 k/mcL; total bilirubin 0.7 mg/dL; AST 22 U/L; ALT 18 U/L; alkaline phosphatase 120 U/L; LDH 186 U/L; PT 11 s (normal 11-13s); PTT 22 s (normal 20-30s). Which of the following is the most appropriate initial treatment for this patient's disorder?

A. Prednisone
B. Ceftriaxone
C. Vincristine
D. Cyclophosphamide
E. Immediate platelet transfusion

You did not answer this question.

Explanations:

A. This patient has idiopathic thrombocytopenic purpura (ITP). ITP is a diagnosis of exclusion made by finding low platelets and ruling out other diseases and toxic exposures that result in decreased platelet counts. It is an autoimmune disease caused by antibody-mediated destruction of platelets, so first line therapies are high-dose corticosteroids like prednisone (answer A) as well as intravenous immunoglobulin (IVIg). Patients refractory to these treatments may require more high-powered immunosuppressant drugs or splenectomy. A few teaching points about ITP and thrombocytopenia: 1) There are two groups of patients who get ITP: children from 2-4, and adults (usually women) in their 20s-40s. Children in general do much better with the disease: over 80% of children will have a spontaneous remission of their disease, while only 20% of adults have a sustained remission. 2) It is extremely important to rule out other diseases before you make the diagnosis of ITP. Here, the patient's CBC is normal with the exception of low platelets – thus, you don't have to worry about leukemia, which would be another common cause of this presentation in a young child. DIC and TTP should also be excluded by looking at a peripheral blood smear for schistocytes or laboratory evidence of microangiopathic hemolytic anemia (increased LDH or bilirubin or decreased haptoglobin and hematocrit). Patients with ITP generally look like this patient – otherwise well except for their thrombocytopenia. 3) Up to 85% of pediatric patients will have a history of an antecedent infection. This is a common clue to the diagnosis on the USMLE. Ceftriaxone (answer B) is a third-generation cephalosporin that is used to treat serious bacterial infections like meningitis in childhood. Vincristine (answer C) is a chemotherapeutic agent sometimes used to treat refractory ITP in adults. It causes a number of serious side effects and would not be first line therapy in any case. Cyclophosphamide (answer D) has been studied for use in ITP. However, because it is a cytotoxic agent, its use in the pediatric population should be reserved for severe cases in which other treatments like steroids, IVIg, and splenectomy have failed. Immediate platelet transfusion (answer E) is not indicated at this time. The risk of spontaneous bleeding does not increase until the platelet count falls below 20,000, and patients with ITP in particular often tolerate even lower platelet counts without spontaneous bleeding. In the absence of serious active bleeding, you should hold off on giving any transfusions – remember, treat the patient, not the numbers!

B. Ceftriaxone (answer B) is a third-generation cephalosporin that is used to treat serious bacterial infections like meningitis in childhood.

C. Vincristine (answer C) is a chemotherapeutic agent sometimes used to treat refractory ITP in adults. It causes a number of serious side effects and would not be first line therapy in a child in any case.

D. Cyclophosphamide (answer D) has been studied for use in ITP. However, because it is a cytotoxic agent, its use in the pediatric population should be reserved for severe cases in which other treatments like steroids, IVIg, and splenectomy have failed.

E. Immediate platelet transfusion (answer E) is not indicated at this time. The risk of spontaneous bleeding does not increase until the platelet count falls below 20,000, and patients with ITP in particular often tolerate even lower platelet counts without spontaneous bleeding. In the absence of serious active bleeding, you should hold off on giving any transfusions – remember, treat the patient, not the numbers!

This question is not currently linked to the learning objective database.

Question problem?

Question # 37

Select the single best answer to the numbered question.

A 36 year old construction worker is brought to the emergency room after a house frame collapsed on him. His upper thighs were trapped under a heavy wooden beam for almost two hours while his co-workers worked to free him. Vital signs are temperature 37.2 C (99.0 F), pulse 90, blood pressure 136/82, respirations 16/min. The patient is alert and oriented, but obviously in pain. There are occasional superficial abrasions on the face and arms, but no large lacerations are noted. Breath sounds are present and equal bilaterally. There is a normal S1 and S2. Abdomen is nontender, nondistended, and atraumatic. The patient's legs show bilateral ecchymoses and tenderness at the upper thigh. Dorsalis pedis and posterior tibial pulses are 2+, and distal leg sensation to light touch and pinprick is intact. Laboratory analysis shows the following Na+ 145; K+ 5.3; Cl- 101; bicarbonate 22; BUN 17; creatinine 1.1; glucose 106. Dipstick urinalysis is unremarkable except for 4+ blood. Microscopic urinalysis shows 2 WBCs/hpf, 0 RBCs/hpf, 3 epithelial cells/hpf, and no casts. What is the most appropriate next step in the management of this patient?

A. Intubation and mechanical ventilation
B. Bolus with 2 L i.v. 0.9% NaCl solution
C. Cystoscopy
D. Intravenous pyelogram
E. Abdominal and pelvic CT

You did not answer this question.

Explanations:

A. Intubation and mechanical ventilation (answer A) is not appropriate, as this patient has a patent airway and is moving air well. In any patient encounter – and especially any trauma scenario – this should be the first thing that you evaluate, though.

B. Key teaching point: When you find blood on a dipstick urinalysis but there are no RBCs microscopically, think of myoglobinuria. This patient has rhabdomyolysis, a common complication of crush injuries. The two most serious complications of rhabdomyolysis are renal failure induced by myoglobinuria and electrolyte abnormalities (like hyperkalemia, hypocalcemia, and metabolic acidosis) caused by the release of massive amounts of intracellular ions. At the moment, this patient's electrolytes do not demand immediate intervention – but the finding of large blood on urine dipstick but no RBCs on microscopic analysis shows that the patient does have myoglobinuria (myoglobin cross reacts with hemoglobin on the urine dipstick). The most important intervention at this time is giving i.v. fluid (answer B) to prevent myoglobin-induced ATN. Patients with myoglobinuria may require massive amounts of fluid in order to maintain good urine output because of fluid sequestration at the site of injury. Fluid replacement is usually accomplished with normal saline at 1-2 L/hour, titrated to maintain a urine output of 200-300 mL/hour. There is also some evidence that alkalinization of the urine with mannitol or bicarbonate may help protect the kidney as well. Intubation and mechanical ventilation (answer A) is not appropriate, as this patient has a patent airway and is moving air well. In any patient encounter – and especially any trauma scenario – this should be the first thing that you evaluate, though. Cystoscopy (answer C) is the best procedure for diagnosing bladder cancer, which is the most frequent cause of gross hematuria in the absence of trauma. Intravenous pyelogram (answer D) is used to evaluate non-glomerular hematuria as well. An abdominal and pelvic CT (answer E) would certainly be a reasonable test in a trauma patient to search for additional injuries, and in real life, the patient would have likely already received this test before his lab results were back. However, given this patient's laboratory data showing myoglobinuria, he is at serious risk for developing ATN and renal failure, and obtaining a CT scan will do nothing to diminish that possibility. Among the options listed, only i.v. fluid will provide an IMMEDIATE benefit to the patient.

C. Cystoscopy (answer C) is the best procedure for diagnosing bladder cancer, which is the most frequent cause of gross hematuria in the absence of trauma.

D. Intravenous pyelogram (answer D) is used to evaluate non-glomerular hematuria as well.

E. An abdominal and pelvic CT (answer E) would certainly be a reasonable test in a trauma patient to search for additional injuries, and in real life, the patient would have likely already received this test before his lab results were back. However, given this patient's laboratory data showing myoglobinuria, he is at serious risk for developing ATN and renal failure, and obtaining a CT scan will do nothing to diminish that possibility. Among the options listed, only i.v. fluid will provide an IMMEDIATE benefit to the patient.

This question is not currently linked to the learning objective database.

Question problem?

Question # 38

Select the single best answer to the numbered question.

A seven-year old male is brought by his mother to the physician because of a bald patch on his head which has been getting larger for the past three weeks. The child has been afebrile and feeling well throughout this time. The patient's mother has seen her son scratch the patch only occasionally, and the boy says that it only itches "a little bit." Past medical history includes asthma treated with albuterol. Physical examination reveals multiple non-erythematous, scaling, well-demarcated patches on the scalp. A microscopic examination of scrapings from one of the lesions prepared with KOH shows spores, but there is no fluorescence under ultraviolet light. A photograph of the lesion is shown. Which

of the following is the most appropriate treatment for this patient's condition? 

A. Clobetasol ointment
B. Mupirocin
C. Griseofulvin
D. Permethrin cream
E. Lindane shampoo

You did not answer this question.

Explanations:

A. Clobetasol (answer A) is a superpotent steroid that is used to treat alopecia areata, an autoimmune disease that may cause recurrent patchy hair loss.

B. Mupirocin (answer C) is a topical antibiotic with good activity against S.aureus that is used to treat mild-to-moderate cases of impetigo. Impetigo can cause itching, but you should see vesicular lesions and "honey colored crusts." Occasionally, tinea capitis lesions can become superinfected with Staphylococcus, but the image assures you that is not the case here.

C. This is tinea capitis, caused by a superficial dermatophytic infection of the skin of the scalp. The fungus attacks the hair shafts and follicles, leading to the presentation seen in this question: alopecia with occasional pruritus. The treatment of choice remains griseofulvin (answer C), although terbinafine or itraconazole are efficacious as well. The diagnosis is usually confirmed, as it was in this case, by the finding of spores with a KOH prep. (Don't be bothered by the fact that there was no fluorescence under a Wood's lamp – only certain species of dermatophytes will fluoresce.) This is an important point, because the most common way of stating this question on the USMLE is to give the above presentation and then ask what test to do next! Clobetasol (answer A) is a superpotent steroid that is used to treat alopecia areata, an autoimmune disease that may cause recurrent patchy hair loss. Mupirocin (answer C) is a topical antibiotic with good activity against S.aureus that is used to treat mild-to-moderate cases of impetigo. Impetigo can cause itching, but you should see vesicular lesions and "honey colored crusts." Occasionally, tinea capitis lesions can become superinfected with Staphylococcus, but the image assures you that is not the case here. Both permethrin cream (answer D) and lindane shampoo (answer E) are used to treat scabies infection. Scabies should always be considered in a child that presents with pruritus, and physical examination may reveal excoriated mite burrows present in the web spaces of the fingers. Of the two treatments, permethrin is preferred in children because lindane can get absorbed through the skin and cause seizures.

D. Both permethrin cream (answer D) and lindane shampoo (answer E) are used to treat scabies infection. Scabies should always be considered in a child that presents with pruritus, and physical examination may reveal excoriated mite burrows present in the web spaces of the fingers. Of the two treatments, permethrin is preferred in children because lindane can get absorbed through the skin and cause seizures.

E. Both permethrin cream (answer D) and lindane shampoo (answer E) are used to treat scabies infection. Scabies should always be considered in a child that presents with pruritus, and physical examination may reveal excoriated mite burrows present in the web spaces of the fingers. Of the two treatments, permethrin is preferred in children because lindane can get absorbed through the skin and cause seizures.

This question is not currently linked to the learning objective database.

Question problem?

Question # 39

Select the single best answer to the numbered question.

A 37 year old teacher has had three months of non-productive cough. The cough seems to be worst at night and upon awakening in the morning. She denies fevers, dyspnea, chest pain, wheezing, purulent nasal discharge, or heartburn. Past medical history includes IBS and hypertension. Medications include candesartan and an oral contraceptive pill. She has never smoked or traveled outside of the United States. Physical examination reveals an afebrile, well-nourished, healthy appearing female. The oropharynx is moist and without erythema or exudates. Nasal mucosa is pink and slightly edematous. Breath sounds are clear to auscultation bilaterally. First and second heart sounds are within normal limits, and no additional heart sounds or murmurs are noted. There is no peripheral edema. Chest x-ray shows normal lung fields. Which of the following is the most appropriate intervention at this time?

A. Stop candesartan
B. Begin antihistamine and decongestant
C. Amoxicillin/clavulanate
D. Place tuberculin purified protein derivative (PPD)
E. Chest CT

You did not answer this question.

Explanations:

A. Stopping candesartan (answer A) is not likely to help. While ACE inhibitors are a common (and commonly-tested) cause of a chronic dry cough, candesartan is an angiotensin receptor blocker (ARB) and, although it can rarely cause a cough, is much less likely than an ACEI to do so. (In fact, one indication for beginning ARB therapy is having had the "captopril cough" while on ACE inhibitor therapy!)

B. This patient presents with a chronic cough, defined as any cough lasting >3 weeks. The most likely culprits in an otherwise healthy person are, in order, postnasal drip, asthma, and GERD. Since this patient has no findings that suggest GERD, asthma, or any other of the numerous other pathological causes of chronic cough, it is reasonable to treat her empirically for postnasal drip with an antihistamine and decongestant (answer B). This course of action is the most likely to both provide a diagnosis and ameliorate the patient's symptoms. In the appropriate setting, chronic cough can be a sign of CHF or intrinsic lung disease. Smokers can have chronic cough related to emphysema, chronic bronchitis, or the irritation of the tobacco smoke itself. For a great review of cough, see Irwin RS, Madison JM: The diagnosis and treatment of cough. N Engl J Med 2000 Dec 7; 343(23): 1715-21 (or online at http://content.nejm.org/cgi/reprint/343/23/1715.pdf). There are separate charts describing the common causes and treatments for patients with acute, subacute, and chronic cough. Stopping candesartan (answer A) is not likely to help. While ACE inhibitors are a common (and commonly-tested) cause of a chronic dry cough, candesartan is an angiotensin receptor blocker (ARB) and does not have this side effect. In fact, one indication for beginning ARB therapy is having had the "captopril cough" while on ACE inhibitor therapy. Amoxicillin/clavulanate (answer B) is a first-line antibiotic for patients with acute bacterial sinusitis. This patient's cough has gone on too long for it to be acute bacterial sinusitis, and she lacks other signs and symptoms such as maxillo-facial pain and purulent nasal discharge. (As a sidenote, even patients with presumed acute bacterial sinusitis should receive 7 days of therapy with decongestants and antihistamines before beginning antibiotic therapy.) Even if this patient had chronic sinusitis, antibiotics are still not likely to be the correct answer – an ENT consult is recommended, though an acute flare-up may respond to antibiotics. Placing a PPD (answer D) could help you learn if this patient had been exposed to tuberculosis. This patient has no risk factors and a normal chest x-ray, so active TB is certainly not the most likely diagnosis here. A chest CT (answer E) is unlikely to have a very high yield in this patient in the absence of clinical findings that suggest intrinsic or anatomical lung disease. An empiric trial of decongestants/antihistamines is more cost effective and more likely to yield a diagnosis.

C. Amoxicillin/clavulanate (answer B) is a first-line antibiotic for patients with acute bacterial sinusitis. This patient's cough has gone on too long for it to be acute bacterial sinusitis, and she lacks other signs and symptoms such as maxillo-facial pain and purulent nasal discharge. (As a sidenote, even patients with presumed acute bacterial sinusitis should receive 7 days of therapy with decongestants and antihistamines before beginning antibiotic therapy.) Even if this patient had chronic sinusitis, antibiotics are still not likely to be the correct answer – an ENT consult is recommended, though an acute flare-up may respond to antibiotics.

D. Placing a PPD (answer D) could help you learn if this patient had been exposed to tuberculosis. This patient has no risk factors and a normal chest x-ray, so active TB is certainly not the most likely diagnosis here.

E. A chest CT (answer E) is unlikely to have a very high yield in this patient in the absence of clinical findings that suggest intrinsic or anatomical lung disease. An empiric trial of decongestants/antihistamines is more cost effective and more likely to yield a diagnosis.

This question is not currently linked to the learning objective database.

Question problem?

Question # 40

Select the single best answer to the numbered question.

Following a 10 hour car trip, a 73 year old female develops left ankle swelling and acute shortness of breath. Ultrasound with Doppler of her left leg confirms the presence of deep venous thrombosis, and the patient is hospitalized and started on a heparin drip and oral warfarin. Two days later she is discharged from the hospital with subcutaneous heparin and warfarin. Shortly upon arrival home, she develops pleuritic chest pain and shortness of breath and presents to her physician. Physical examination reveals tachypnea and an oxygen saturation of 89% on room air. Laboratory analysis shows her activated partial thromboplastin time to be therapeutically increased, while her prothrombin time remains below the optimal therapeutic level. A ventilation/perfusion scan is obtained, which shows a large perfusion defect in the left lower lobe. Which of the following is the most appropriate intervention at this time?

A. Hospital admission for observation
B. Begin aspirin
C. Increase heparin
D. Increase warfarin
E. Placement of an inferior vena cava filter

You did not answer this question.

Explanations:

A. Hospital admission for observation (answer A) is not enough. This patient has had two PEs, one of which occurred despite adequate heparin anticoagulation. She needs to be in the hospital, but you can't just sit around waiting for the big one to happen!

B. Adding aspirin (answer B) is not likely to help. Although aspirin does have an antiplatelet effect, it is not powerful enough to provide true anticoagulation.

C. Increasing the patient's dose of heparin (answer C) should not provide any additional benefit, given that her aPTT is already in the therapeutic range. You may increase her risk of bleeding complications by increasing her dose, however.

D. Increasing the patient's dose of warfarin (answer D) will not provide any immediate benefit, and may make things worse in the near future by increasing the patient's risk of bleeding. Warfarin works by antagonizing Vitamin K and reducing the production of clotting factors II, VII, IX, and X. It takes several days of protein turnover for warfarin to become therapeutic, which is why this patient was sent home with "bridging" therapy with subcutaneous heparin. If you increase the dose of warfarin now, you'll get no immediate effect, but you could result in a supratherapeutic INR in a day or so, which would increase the patient's risk of bleeding.

E. This patient has had a pulmonary embolism while receiving anticoagulation therapy for DVT. The only intervention among those listed that is likely to help her is the placement of an inferior vena cava filter (answer E). These filters can be placed transcutaneously, and in the short term will prevent a large thrombus from making its way from her leg to her pulmonary vasculature. Placement of an IVC filter is also indicated in patients who have had a PE but who have a contraindication to anticoagulation (although the evidence of benefit is less clear in that situation). Hospital admission for observation (answer A) is not enough. This patient has had two PEs, one of which occurred despite adequate heparin anticoagulation. She needs to be in the hospital, but you can't just sit around waiting for the big one to happen. Adding aspirin (answer B) is not likely to help. Although aspirin does have an antiplatelet effect, it is not powerful enough to provide true anticoagulation. Increasing the patient's dose of heparin (answer C) should not provide any additional benefit, given that her aPTT is already in the therapeutic range. You may increase her risk of bleeding complications by increasing her dose, however. Increasing the patient's dose of warfarin (answer D) will not provide any immediate benefit, and may make things worse in the near future by increasing the patient's risk of bleeding. Warfarin works by antagonizing Vitamin K and reducing the production of clotting factors II, VII, IX, and X. It takes several days of protein turnover for warfarin to become therapeutic, which is why this patient was sent home with "bridging" therapy with subcutaneous heparin. If you increase the dose of warfarin now, you'll get no immediate effect, but you could result in a supratherapeutic INR in a day or so, which would increase the patient's risk of bleeding.

This question is not currently linked to the learning objective database.

Question problem?

Question # 41

Select the single best answer to the numbered question.

A 56 year old male with hypertension and history of drug abuse and medication noncompliance presents to the clinic for follow up. He has been feeling well since his last appointment several months ago, but does admit to cocaine use last night when he ran into some old friends. He denies any other drug use. Medications include hydrochlorothiazide, atenolol, and paroxetine. Vital signs are temperature 36.9 C (98.4 F), pulse 85, blood pressure 202/122, and respirations 14/min. Physical examination reveals a comfortable, pleasant male in no distress. There is no papilledema or retinal hemorrhage on fundoscopic exam. The patient is alert, oriented, and neurologically intact. Laboratory evaluation, including CBC, electrolytes, BUN and creatinine, and urinalysis are all unremarkable. Which of the following is the most appropriate next step in the management of this patient?

A. Obtain urine drug screen
B. Head CT without contrast
C. Administer naloxone
D. Administer oral labetalol
E. Administer intravenous nitroprusside

You did not answer this question.

Explanations:

A. Obtaining a urine drug screen (answer A) will not help in the management of this patient. He has already admitted recent cocaine use, and there is not a compelling reason to immediately search out any other drugs of abuse in his system. While in some cases it may be beneficial to have some laboratory documentation of this patient's drug use, confirming that he has used cocaine is not the top priority at this point given this patient's markedly elevated blood pressure. As a sidenote, remember that patients on the USMLE don't lie – in sharp contrast to real patients, you can take their assessment of substance use at face value unless the question stem give you obvious reason to doubt it.

B. A head CT without contrast (answer B) is the test of choice for diagnosing an acute head bleed. In a patient with malignant hypertension, you might also discover lacunar infarcts. This patient has no symptoms, though, so this test is unnecessary at this point.

C. Naloxone (answer C) is an opioid antagonist. It may be useful in a patient who has overdosed on opiates, but it has no role in hypertensive urgency or cocaine intoxication.

D. This is hypertensive urgency, defined as a systolic blood pressure >200 or a diastolic >120 in the absence of symptoms. In contrast, a hypertensive emergency is defined as increased blood pressure with signs and symptoms of end-organ damage such as papilledema, stroke, hematuria, headache, altered mental status, acute coronary syndrome, etc. The treatment of the two is different: for a hypertensive emergency, you need to give i.v. agents to get the blood pressure down within minutes. Hypertensive urgency, however, is treated with oral medications to bring down the blood pressure more slowly over a matter of hours. Thus, the best choice is oral labetalol (answer D). A couple of other teaching points… 1) Apart from its availability as a p.o. formulation, this patient has another compelling reason to be treated with labetalol instead of another agent: his recent history of cocaine use. Unlike other "pure" beta blockers, labetalol has both alpha- and beta-adrenergic blocking properties. If you treat a patient with cocaine-induced hypertension with a pure beta blocker, you're left with unopposed alpha adrenergic activation, which can cause intense vasoconstriction, ischemia, and gangrene. 2) The most common reason for elevated blood pressure is noncompliance with medical therapy. Given this patient's heart rate of 85, it is very unlikely that he has taken a recent dose of his beta blocker. Obtaining a urine drug screen (answer A) will not help in the management of this patient. He has already admitted recent cocaine use, and there is not a compelling reason to immediately search out any other drugs of abuse in his system. While in some cases it may be beneficial to have some laboratory documentation of this patient's drug use, confirming that he has used cocaine is not the top priority at this point given this patient's markedly elevated blood pressure. As a sidenote, remember that patients on the USMLE don't lie – in sharp contrast to real patients, you can take their assessment of substance use at face value unless the question stem give you obvious reason to doubt it. A head CT without contrast (answer B) is the test of choice for diagnosing an acute head bleed. In a patient with malignant hypertension, you might also discover lacunar infarcts. This patient has no symptoms, though, so this test is unnecessary at this point. Naloxone (answer C) is an opioid antagonist. It may be useful in a patient who has overdosed on opiates, but it has no role in hypertensive urgency or cocaine intoxication. Administering intravenous nitroprusside (answer E) will bring the patient's blood pressure down, but will require an inpatient (or potentially even an ICU) admission for blood pressure monitoring. If this patient were having symptoms – that is, if he were having a hypertensive emergency rather than hypertensive urgency – this would be an appropriate management plan, but in the absence of symptoms, it is a bit too much at the present moment.

E. Administering intravenous nitroprusside (answer E) will bring the patient's blood pressure down, but will require an inpatient (or potentially even an ICU) admission for blood pressure monitoring. If this patient were having symptoms – that is, if he were having a hypertensive emergency rather than hypertensive urgency – this would be an appropriate management plan, but in the absence of symptoms, it is a bit too much at the present moment.

This question is not currently linked to the learning objective database.

Question problem?

Question # 42

Select the single best answer to the numbered question.

A 71 year old male with a history of coronary artery disease and congestive heart failure presents with worsening dyspnea on exertion, orthopnea, and peripheral edema over the past week. Two years ago, he had a coronary stent placed in his circumflex artery. Catheterization at that time showed an ejection fraction of 25%, and since that time the patient had been stable with medical therapy. Past medical history also includes diabetes, hypertension, and peripheral arterial disease. Current medications include clopidogrel, lisinopril, propranolol, bumetanide, spironolactone, glipizide, and metformin. Physical examination reveals diffuse crackles bilaterally extending from the lung bases to two-thirds of the way up the lung fields. An S3 is heard, and there is 3+ pitting edema of the lower extremities bilaterally. Chest x-ray shows cardiomegaly and pulmonary edema in a "bat wing" pattern. The patient is admitted to the hospital, placed on a fluid restriction, and treated with intravenous furosemide. Following a net diuresis of 3 L, the patient feels much better. Which of the following is the next most appropriate step in the management of this patient?

A. Measurement of brain natriuretic peptide (BNP)
B. Echocardiography
C. Arterial blood gas
D. Increase propranolol
E. Endomyocardial biopsy

You did not answer this question.

Explanations:

A. Because brain natriuretic peptide or BNP (answer A) increases in CHF, measuring it is useful if you are trying to determine whether CHF is the cause of a patient's dyspnea. Given the abundance of history, physical, and radiographic findings pointing to CHF (and the absence of any cues steering you toward any other cause of dyspnea), checking a BNP is not necessary to make the diagnosis. (Actually, a head-to-head trial comparing BNP to clinical judgment for diagnosing heart failure found that while measurement of BNP was more sensitive, clinical judgment was more specific.) At this stage of the patient's treatment, it is more important to figure out if he has had a new ischemic event that triggered this episode of decompensation.

B. This patient with a known history of coronary artery disease presented with the classic features of decompensated congestive heart failure. Once he has been stabilized, the focus should turn to what precipitated the worsening heart failure. There are a number of common precipitants for CHF exacerbations, including dietary indiscretion (such as a high salt diet), medical noncompliance, new arrythmias (including atrial fibrillation), anemia, and medication changes (such as an increase in beta blockers). However, the most important cause of decompensated heart failure, and the one that should always be ruled out first, is new myocardial ischemia or infarction. An echocardiogram (answer B) will allow assessment of the patient's ejection fraction and since we know this patient's previous ejection fraction, we'll know whether his coronary artery disease has progressed. As a sidenote, another step that should be taken in the management of this patient is measurement of serum electrolytes, given his diuresis of 3 L of fluid. Because brain natriuretic peptide or BNP (answer A) increases in CHF, measuring it is useful if you are trying to determine whether CHF is the cause of a patient's dyspnea. Given the abundance of history, physical, and radiographic findings pointing to CHF (and the absence of any cues steering you toward any other cause of dyspnea), checking a BNP is not necessary to make the diagnosis. (Actually, a head-to-head trial comparing BNP to clinical judgment for diagnosing heart failure found that while measurement of BNP was more sensitive, clinical judgment was more specific.) At this stage of the patient's treatment, it is more important to figure out if he has had a new ischemic event that

triggered this episode of decompensation. Arterial blood gas measurements (answer C) are useful in quantifying the degree of hypoxia in a patient with respiratory distress or a low oxygen saturation. This patient has improved with therapy, so this test is unnecessary. Although beta-blockers like propranolol (answer D) clearly confer a survival benefit in patients with heart failure, increasing this patient's dose now is not the right strategy. Because of their negative inotropic effects, you should avoid starting or increasing a beta-blocker in decompensated heart failure. Although this patient is doing better now, there are more important things to do than increase his beta-blocker – like figure out what triggered this episode of decompensation. Endomyocardial biopsy (answer E) can confirm the diagnosis of a cardiomyopathy, which can cause heart failure. This test can be considered in patients who have been ruled out for other causes of heart failure.

C. Arterial blood gas measurements (answer C) are useful in quantifying the degree of hypoxia in a patient with respiratory distress or a low oxygen saturation. This patient has improved with therapy, so this test is unnecessary.

D. Although beta-blockers like propranolol (answer D) clearly confer a survival benefit in patients with heart failure, increasing this patient's dose now is not the right strategy. Because of their negative inotropic effects, you should avoid starting or increasing a beta-blocker in decompensated heart failure. Although this patient is doing better now, there are more important things to do than increase his beta-blocker – like figure out what triggered this episode of decompensation.

E. Endomyocardial biopsy (answer E) can confirm the diagnosis of a cardiomyopathy, which can cause heart failure. This test can be considered in patients who have been ruled out for other causes of heart failure.

This question is not currently linked to the learning objective database.

Question problem?

Question # 43

Select the single best answer to the numbered question.

A 5 year old boy presents with bedwetting. His mother states that for the past two months, he has been wetting the bed several times a week. The child is embarrassed about this new incontinence, and has had no problems in the daytime. The patient's mother insists that the child has otherwise been healthy; she denies fevers and increased appetite or fluid intake. The patient's past medical history is remarkable only for a hospitalization at age 1 for bronchiolitis. The boy lives with his mother, father, and a younger sister who was adopted from Russia several months ago. Everyone at home has been well. On physical exam, the patient is a talkative, interactive male. Speech, motor, and physical development are all normal. What is the most appropriate initial step in the management of this patient?

A. Urine culture
B. Voiding cystourethrogram
C. Recommend an enuresis alarm
D. Desmopressin
E. Urinalysis

You did not answer this question.

Explanations:

A. Urine culture (choice A) is not indicated unless the UA is positive for nitrites or white blood cells.

B. Obtaining a voiding cystourethrogram (choice B) should not be the initial step in the workup of this patient. Imaging such as VCUG or renal ultrasound is reserved for children with significant daytime symptoms, a history of unexplained UTIs, or with symptoms that suggest structural abnormalities.

C. An enuresis alarm (choice C) can be an effective treatment for nocturnal enuresis once you've determined that the bedwetting is behavioral, and not secondary to a medical condition. The alarm goes off when a sensor placed under the bed garments detects moisture. The child is taught to awaken, turn off the alarm, go to the restroom to finish urinating, change the bed garments and then return to sleep when the alarm goes off. Over time, this treatment can be very successful in reducing nocturnal events.

D. Desmopressin (choice D) is an effective medical treatment for nocturnal enuresis. Administered in the late evening, the drug helps to reduce urine production overnight, reducing nocturnal events. Starting a child on desmopressin without a work-up for the cause of the enuresis would be inappropriate.

E. New onset enuresis should prompt the physician to perform a thorough history and physical and to initially obtain a urinalysis (answer E). This single test will allow screening for urinary tract infection, a common cause of new-onset enuresis, as well as diabetic ketoacidosis, diabetes insipidus, and water intoxication. Imaging and referrals are reserved for patients with histories and physical exams that suggest a structural cause. Urine culture (choice A) is not indicated unless the UA is positive for nitrites or white blood cells. Obtaining a voiding cystourethrogram (choice B) should not be the initial step in the workup of this patient. Imaging such as VCUG or renal ultrasound is reserved for children with significant daytime symptoms, a history of unexplained UTIs, or with symptoms that suggest structural abnormalities. An enuresis alarm (choice C) can be an effective treatment for nocturnal enuresis once you've determined that the bedwetting is behavioral, and not secondary to a medical condition. The alarm goes off when a sensor placed under the bed garments detects moisture. The child is taught to awaken, turn off the alarm, go to the restroom to finish urinating, change the bed garments and then return to sleep when the alarm goes off. Over time, this treatment can be very successful in reducing nocturnal events. Desmopressin (choice D) is an effective medical treatment for nocturnal enuresis. Administered in the late evening, the drug helps to reduce urine production overnight, reducing nocturnal events. Starting a child on desmopressin without a work-up for the cause of the enuresis would be inappropriate.

This question is not currently linked to the learning objective database.

Question problem?

Question # 44

Select the single best answer to the numbered question.

A 22 year old first year law student comes to the physician with amenorrhea for the past four months. The patient admits having been under a great deal of stress adjusting to the academic burden of law school, but did finish her first semester with a 3.7 GPA. Upon questioning, she acknowledges eating large amounts of food such as an entire large pizza when she is feeling stressed. She feels guilty after she eats, however, and forces herself to vomit it all up. The rest of the time, she eats a "normal diet" of salads and granola. Physical examination reveals an extremely thin female, with height is 69 in. (175 cm), weight is 103 lbs. (47 kg). There is diffuse fine hair growth on the extremities and mild erosion

of the enamel of the anterior teeth. A callus is present on the dorsal surface of the index and middle fingers of the patient's right hand. TSH is within normal limits. A urine pregnancy screen is negative. Which of the following is the most appropriate diagnosis in this patient?

A. Anorexia nervosa
B. Obsessive-compulsive disorder
C. Borderline personality disorder
D. Bulimia nervosa
E. Hyperprolactinemia

You did not answer this question.

Explanations:

A. The criteria for anorexia nervosa include amenorrhea, body weight less than 85% of expected ideal weight, and intense fear of gaining weight or becoming fat. This patient fits those criteria, and although binging and purging behaviors are more typically associated with bulimia, her disease is more appropriately accounted for by anorexia nervosa (answer A). Put another way, anorexia nervosa is the supraordinate diagnosis in the DSM-IV – that is, a patient who seems to meet criteria for bulimia but who also meets criteria for anorexia should be diagnosed with anorexia. The reason for this is prognostic: anorexia nervosa has a staggering 20% mortality rate at 20 years. Thus, the most appropriate diagnosis in this patient is anorexia nervosa, binge-eating/purging type. As a sidenote, patients with bulimia do have a couple of characteristic signs on physical exam. Erosion of the enamel of the anterior teeth occurs due to exposure of the teeth to acidic gastric juices. There may also be enlarged parotid glands, or a positive Russell sign (described in the question stem as the callus on the patient's dominant hand due to self-induced vomiting). The presence of lanugo, or fine downy hair, can be seen in any underweight patient. Patients with anorexia may have comorbid obsessive-compulsive personality disorder or even full-blown obsessive compulsive disorder (answer B), but there is nothing in the question stem that suggests either obsessions (persistent and intrusive ideas or images) or compulsions (impulses for repetitive or stereotyped behavior). Patients with binging and purging behaviors often have comorbid borderline personality disorder (answer C). This is a commonly tested disorder on the USMLE, but none of the classic features are given to you in the question stem such as self injury or mutilation, a history of suicidal gestures, or "splitting." As described above, although purging behaviors are commonly associated with bulimia nervosa (answer D), this patient is underweight and amenorrheic, so the best diagnosis is anorexia nervosa. Hyperprolactinemia (answer E) can cause secondary amenorrhea, and after a negative pregnancy test and a normal TSH, measuring prolactin is the usual next step in diagnosing amenorrhea. Here, though, the cause of the patient's amenorrhea is clear – it's her eating disorder.

B. Patients with anorexia may have comorbid obsessive-compulsive personality disorder or even full-blown obsessive compulsive disorder (answer B), but there is nothing in the question stem that suggests either obsessions (persistent and intrusive ideas or images) or compulsions (impulses for repetitive or stereotyped behavior).

C. Patients with binging and purging behaviors often have comorbid borderline personality disorder (answer C). This is a commonly tested disorder on the USMLE, but none of the classic features are given to you in the question stem such as self injury or mutilation, a history of suicidal gestures, or "splitting."

D. Although purging behaviors are commonly associated with bulimia nervosa (answer D), this patient is underweight and amenorrheic, so bulimia is not the best diagnosis in this case.

E. Hyperprolactinemia (answer E) can cause secondary amenorrhea, and after a negative pregnancy test and a normal TSH, measuring prolactin is the usual next step in diagnosing amenorrhea. Here, though, the cause of the patient's amenorrhea is clear – it's her eating disorder.

This question is not currently linked to the learning objective database.

Question problem?

Question # 45

Select the single best answer to the numbered question.

A 18 month old female is brought to the physician by her mother, who is concerned that her child is in pain. The child has had spells of intense sceaming and crying for the past few hours; during these episodes the child doubles over and curls up into a ball. The mother also reports two episodes of non-bilious, non-bloody vomit. The mother initially thought that the patient may have caught a virus at day care, but she became more concerned one hour ago when her daughter had a bowel movement and the stool seemed to be slimy with a purplish color. On physical exam, a sausage shaped abdominal mass is palpated in the right abdomen. There is no abdominal rigidity, guarding, or rebound tenderness. An

purplish color. On physical exam, a sausage shaped abdominal mass is palpated in the right abdomen. There is no abdominal rigidity, guarding, or rebound tenderness. An abdominal ultrasound reveals a “bull’s eye” or “coiled spring” appearing lesion. The patient is made n.p.o., i.v. fluids are started, and a nasogastric suction is begun. What is the next best step in the management of this patient?

A. CT scan
B. Air contrast enema
C. Surgery
D. Colonoscopy
E. Administer a glycerin suppository

You did not answer this question.

Explanations:

A. A CT scan (choice A) is not needed at this point. Though CT can help with the diagnosis of intussusception, it is not therapeutic, can be time consuming and may require patient sedation in the pediatric population.

B. This is a typical presentation of intussusception, a condition in which part of the intestine telescopes on itself, resulting in bowel edema, ischemia, obstruction, and ultimately perforation. The patient usually presents with a history of sudden onset severe, crampy abdominal pain that is accompanied by drawing the legs up toward the abdomen and inconsolable crying. These episodes usually last 20 minutes and pain-free periods can follow. Usually the episodes become more severe and spaced closer together over time. Non-bilious vomiting can become bilious as the obstruction worsens. “Currant jelly” stool is a common description of the blood and mucus mixed stool that can occur with intussusception. Palpation of a “sausage-shaped” mass is also classic (especially on the USMLE), but is not always appreciated on physical exam. Ultrasound imaging is not mandatory for diagnosis, but if performed, it may reveal pathognomonic “bull’s eye” or “coiled spring” lesions. Prompt treatment is necessary to avoid irreversible intestinal ischemia or bowel perforation. An air contrast enema (answer B) is not only diagnostic, but can also be therapeutic by reducing the telescoped bowel. A handful of extra teaching points… 1) The child’s age is an important clue when you’re considering a diagnosis of intussusception. Intussusception is the most common cause of intestinal obstruction in ages 6 months to 36 months, but is very rare before 3 months and after 6 years of age. 2) Based on history and physical, if you have a high index of suscpicion for intussusception, it is appropriate to make the diagnosis using contrast enema, skipping other imaging modalities. 3) Water soluble contrast is used if perforation is likely, while barium contrast is appropriate only after perforation is ruled out. The new standard is an air contrast enema, which is just as efficacious as the others while also being cheaper and avoiding radiation exposure. 4) Surgery is needed if non-operative reduction is incomplete, a filling defect or mass persists, or perforation occurs (a risk of contrast enema). A CT scan (choice A) is not needed at this point. Though CT can help with the diagnosis of intussusception, it is not therapeutic, can be time consuming and may require patient sedation in the pediatric population. Surgical management (choice C) is not needed at this point. Non-surgical reduction using contrast enema is the most appropriate first step. This would be the first choice if the patient had presented with prolonged symptoms, peritoneal signs or evidence of free air. Colonoscopy (choice D) is inappropriate and not part of the diagnostic or therapeutic management of intussusception. Administering a glycerin suppository (choice E) is an acceptable treatment for fecal impaction in infants.

C. Surgical management (choice C) is not needed at this point. Non-surgical reduction using contrast enema is the most appropriate first step. This would be the first choice if the patient had presented with prolonged symptoms, peritoneal signs or evidence of free air.

D. Colonoscopy (choice D) is inappropriate and not part of the diagnostic or therapeutic management of intussusception.

E. Administering a glycerin suppository (choice E) is an acceptable treatment for fecal impaction in infants.

This question is not currently linked to the learning objective database.

Question problem?

Question # 46

Select the single best answer to the numbered question.

A 29 year old female medical student comes to her physician with gallstones after serving as a model patient for a demonstration of abdominal ultrasound techniques. The stones were located within the gallbladder, and the patient was surprised at this finding, because she never had experienced any pain. Physical examination reveals a comfortable, afebrile, obese female. Murphy’s sign is negative. Laboratory examination shows the following: WBC 7.0 K/mm3; albumin 4.2 g/dL; Alkaline phosphatase 72 U/L; ALT 16 IU/L; AST 19 IU/L; total bilirubin 0.3 mg/dL; conjugated bilirubin 0.1 mg/dL; lipase 18 U/L (normal: 10-140 U/L). Which of the following is the most appropriate next step in the management of this patient?

A. Reassurance
B. Endoscopic retrograde cholangiopancreatography (ERCP)
C. Abdominal CT
D. Ursodeoxycholic acid
E. Cholecystectomy

You did not answer this question.

Explanations:

A. This scenario describes an asymptomatic patient with cholelithiasis and normal LFTs. Some 10-20% of all Americans will develop gallstones, but of these, 60-80% will remain asymptomatic. This asymptomatic, otherwise healthy patient should be reassured (answer A) – other interventions can be considered should she become symptomatic in the future. A few brief teaching points on this case… 1) Terminology is important when dealing with gallbladder disease. Cholelithiasis (as this patient has) refers to the simple presence of gallstones in the gallbladder. Biliary colic is pain caused by a stone temporarily obstructing the cystic duct. The pain from biliary colic goes away, unlike the unrelenting right upper quadrant pain of cholecystitis, in which obstruction of the cystic duct becomes fixed and leads to inflammation. Patients with cholecystitis also have increased WBC, fever, and signs of inflammation on ultrasound like gallbladder wall thickening. Choledocholithiasis refers to the presence of a stone in the common bile duct, which can be missed on ultrasound but will lead to abnormal LFTs. Cholangitis refers to a stone obstructing the biliary or hepatic ducts, which leads to inflammation and infection and the feared symptoms of Charcot’s triad (fever/chills, RUQ pain, and jaundice) and Reynold’s pentad (Charcot’s triad plus hypotension and altered mental status). 2) A positive Murphy’s sign is the presence of RUQ pain and inspiratory arrest that occurs with palpation of the RUQ. It indicates acute cholecystitis. The USMLE in general avoids eponyms – only the best known signs and syndromes will appear on the test, but this is one that everyone should know. 3) There ARE a few indications for cholecystectomy in an asymptomatic patient. Most surgeons will recommend a cholecystectomy on children with gallstones, just as they would for patients with cirrhosis or those waiting an organ transplant. Also, patients with sickle cell disease almost always develop pigment stones, and should have an elective cholecystectomy. Finally, patients with a calcified or “porcelain” gallbladder should have it removed, because this finding is associated with a high risk of malignancy. ERCP (answer B) has both diagnostic and therapeutic value in patients with gallstones lodged in the biliary tree. It is not indicated in an asymptomatic patient. An abdominal CT (answer C) is not likely to yield any additional useful information. Ultrasound is very sensitive for detecting cholelithiasis and cholecystitis (although it is only 33% sensitive for detecting choledocholithiasis), and is thus an adequate study for this asymptomatic patient with normal LFTs. Ursodeoxycholic acid (answer D) can dissolve gallstones over time, but the gallstones will almost always recur once the medication is stopped. For this reason, it is not used in asymptomatic patients. However, it can be useful for a patient with biliary colic who wants to avoid cholecystectomy. Cholecystectomy (answer E) may be indicated for patients with cholecystitis or biliary colic, but not in an asymptomatic patient with normal labs.

B. ERCP (answer B) has both diagnostic and therapeutic value in patients with gallstones lodged in the biliary tree. It is not indicated in an asymptomatic patient.

C. An abdominal CT (answer C) is not likely to yield any additional useful information. Ultrasound is very sensitive for detecting cholelithiasis and cholecystitis (although it is only 33% sensitive for detecting choledocholithiasis), and is thus an adequate study for this asymptomatic patient with normal LFTs.

D. Ursodeoxycholic acid (answer D) can dissolve gallstones over time, but the gallstones will almost always recur once the medication is stopped. For this reason, it is not used in asymptomatic patients. However, it can be useful for a patient with biliary colic who wants to avoid cholecystectomy.

E. Cholecystectomy (answer E) may be indicated for patients with cholecystitis or biliary colic, but not in an asymptomatic patient with normal labs.

This question is not currently linked to the learning objective database.

Question problem?

Question # 47

Select the single best answer to the numbered question.

A 35 year old male with a history of bipolar disorder presents with several weeks of “depression.” Though his mood is no worse than usual, he has been feeling extremely fatigued. He has also been sleeping more lately and reports a 9 lbs. weight gain over this period. He denies racing thoughts, hallucinations, or increases in goal-directed activity. Current medications include lithium and omeprazole. On physical examination, the patient has coarse facial features and slowed speech and movements. There are areas of diffuse hair loss on the scalp, as well as periorbital puffiness and non-pitting edema of the legs bilaterally. Serum lithium level is 1.45 mEq/L (normal therapeutic range: 0.50-1.50 mEq/L). Which of the following is the most appropriate next step in the management of this patient

A. Decrease lithium dose by 50%
B. Order thyroid stimulating hormone (TSH) and free T4 levels
C. Begin escitalopram
D. Begin risperidone
E. Begin valproic acid

You did not answer this question.

Explanations:

A. Decreasing the dose of lithium (answer A) will probably only cause the patient to become subtherapeutic on his medication – it won’t necessarily correct his underlying problem here, which is hypothyroidism.

B. Although lithium is a highly effective treatment for bipolar disorder, it carries the risk of serious side effects: some 20-30% of patients will develop subclinical or overt hypothyroidism. Therefore, this patient needs to have his thyroid function investigated with TSH and free T4 levels (answer B). Should this patient have hypothyroidism, he should be treated with thyroid hormone replacement. Lithium need not be discontinued, particularly if it has been an effective treatment for the patient’s psychiatric illness. Decreasing the dose of lithium (answer A) will probably only cause the patient to become subtherapeutic on his medication – it won’t necessarily correct his hypothyroidism. Both risperidone (answer D) and valproic acid (answer E) are effective treatments for bipolar disorder. It can be used as a monotherapy, or in combination with lithium to better control symptoms. Here, though, you should rule out hypothyroidism first before you assume that the patient’s symptoms are due to his psychiatric illness.

patient's symptoms are due to his psychiatric illness.

C. Beginning an antidepressant like escitalopram (answer C) won't treat this patient's hypothyroidism, and is likely contraindicated in a patient with bipolar disorder, anyhow. Antidepressants can precipitate mania in bipolar patients, which is why the usual standard of care for such patients is mood stabilizing drugs. Atypical antipsychotic agents and anticonvulsants are also used for patients with bipolar disorder. While some psychiatrist will use standard antidepressants in their bipolar patients, they usually do with close follow up and only as part of a multi-drug therapeutic "cocktail" tailored to that particular patient.

D. Both risperidone (answer D) and valproic acid (answer E) are effective treatments for bipolar disorder. It can be used as a monotherapy, or in combination with lithium to better control symptoms. Here, though, you should rule out hypothyroidism first before you assume that the patient's symptoms are due to his psychiatric illness.

E. Both risperidone (answer D) and valproic acid (answer E) are effective treatments for bipolar disorder. It can be used as a monotherapy, or in combination with lithium to better control symptoms. Here, though, you should rule out hypothyroidism first before you assume that the patient's symptoms are due to his psychiatric illness.

This question is not currently linked to the learning objective database.

Question problem?

Question # 48

Select the single best answer to the numbered question.

A 38 year old HIV positive male presents for regularly-scheduled follow-up. He received pneumococcal and influenza vaccinations last year, and has had no opportunistic infections for several months. Current medications include efavirenz and nevirapine. Physical exam is unchanged from previous. Laboratory evaluation shows CD4+ lymphocyte count of 179/mm3. Tuberculin purified protein derivate (PPD) is placed intradermally, and is read 72 hours later as causing 4 mm of induration. Which of the following is the most appropriate next step in the management of this patient?

A. Chest x-ray
B. Clarithromycin
C. Trimethoprim-sulfamethoxazole
D. Fluconazole
E. Isoniazid, rifampin, and pyrizinamide

You did not answer this question.

Explanations:

A. A chest x-ray (answer A) is not needed. This patient's PPD should be read as negative, and in the absence of clinical suspicion for active TB, there is no reason to get this study right now.

B. Azithromycin or clarithromycin prophylaxis (answer B) should begin if the patient's CD4+ count drops below 50. It is true that many physicians do not wait until the CD4+ drops that low, and prefer to begin it around 100 or even 200, but the evidence most clearly supports the cutoff of 50, and this is the current recommendation from the IDSA.

C. Any patient with AIDS whose CD4+ count drops below 200 needs to begin prophylaxis for Pneumocystis carinii (PCP). Trimethoprim-sulfamethoxazole (answer C) is the first-line drug of choice, and should be started in this patient now. A few teaching points about this case… 1) Primary care for HIV positive patients involves a great deal of prophylaxis. All patients with HIV should receive the pneumococcal vaccine and an annual influenza vaccine. Pharmacologic prophylaxis depends on the patient's CD4+ count and symptoms. When the CD4+ count drops below 200, you should begin prophylaxis for PCP, and prophylaxis for Toxoplasma should begin when the CD4+ drops below 100. (TMP/SMZ is adequate prophylaxis for both PCP and Toxoplasma, so if the patient can tolerate it, it's usually best to try to kill two birds with one stone.) When the CD4+ count drops below 50, you should begin azithromycin or clarithromycin for Mycobacterium avium complex (MAC) prophylaxis. Prophylaxis for Candida or HSV may be required if the patient has multiple recurrences of either of these problems. If you'd like to see the complete guidelines on prophylaxis of opportunistic infections in HIV+ patients, go to http://www.cdc.gov/mmwR/preview/mmwrhtml/rr5108a1.htm 2) PPDs are interpreted differently in patients with HIV/AIDS. You count a PPD as "positive" when there is… a) >5 mm induration in a "high risk" patient – someone who has HIV/AIDS (or is otherwise immunosuppressed) or who has had close contact with a patient with active TB; b) >10 mm induration in a "moderate risk" patient – someone who is homeless, comes from a country with high TB rates, or is an i.v. drug user; c) >15 mm induration in a "low risk" patient – someone without any major risk factors for tuberculosis. A chest x-ray (answer A) is not needed. This patient's PPD should be read as negative, and in the absence of clinical suspicion for active TB, there is no reason to get this study right now. As described above, azithromycin or clarithromycin prophylaxis (answer B) should begin if the patient's CD4+ count drops below 50. It is true that many physicians do not wait until the CD4+ drops that low, and prefer to begin it around 100 or even 200, but the evidence most clearly supports the cutoff of 50, and this is the current recommendation from the IDSA. Fluconazole (answer D) is used for prophylaxis in HIV positive patients who have recurrent candidiasis. There is no CD4+ count guideline for beginning fluconazole – only the patient's symptoms. Rifampin, isoniazid, and pyrizinamide (answer E) is called R.I.P. therapy, and is used to treat sensitive strains of tuberculosis. Until you've confirmed that the strain of TB is sensitive, though, it's best to start with a four drug combo with ethambutol, also (a.k.a. RIPE therapy).

D. Fluconazole (answer D) is used for prophylaxis in HIV positive patients who have recurrent candidiasis. There is no CD4+ count guideline for beginning fluconazole – only the patient's symptoms.

E. Rifampin, isoniazid, and pyrizinamide (answer E) is called R.I.P. therapy, and is used to treat sensitive strains of tuberculosis. Until you've confirmed that the strain of TB is sensitive, though, it's best to start with a four drug combo with ethambutol, also (a.k.a. RIPE therapy).

This question is not currently linked to the learning objective database.

Question problem?

Question # 49

Select the single best answer to the numbered question.

An 18 year old G1 at 39 weeks gestation presents in labor. Her pregnancy has been complicated by excessive weight gain and gestational diabetes mellitus. Soon after presentation, the patient rapidly dilates and the infant's head descends. The infant's head is delivered without difficulty, but before delivering the body, the head appears to retract toward the pelvis. Initial attempts to deliver the body are unsuccessful. A fetal scalp electrode is attached, which shows a wavy tracing with heart rates between 140 and 150 bpm with marked beat-to-beat variability. Which of the following is the most appropriate next step in the management of this patient?

A. Caesarean section
B. Fundal pressure
C. Oxytocin
D. Suprapubic pressure
E. Fracturing the baby's clavicles

You did not answer this question.

Explanations:

A. So long as the infant is doing well, Caesarean section (answer A) is inappropriate before attempting some of the obstetrical maneuvers described above. If the situation is dire or other methods have failed, it is possible to place the infant's head back in the pelvis and go to Caesarean section (this is called the Zavanelli maneuver).

B. Applying fundal pressure (answer B) is NEVER the right thing to do for shoulder dystocia. It will not help free the infant, and may cause damage to its head and neck.

C. Oxytocin (answer C) could increase the strength of the uterine contractions, but that will not likely help the situation. Remember the "three P's" of labor? The problem here is not with the patient's "power" – it's with a mismatch between the "passage" (or pelvis) and the "passenger," so treat it appropriately.

D. This patient's delivery has been complicated by shoulder dystocia, where the infant's anterior shoulder becomes impacted behind the pubic symphysis. Often, moderate suprapubic pressure (answer D) is all that you'll need to free the fetal shoulder, so this should be attempted first and is the best option among those listed. A couple of teaching points about shoulder dystocia… 1) The risk factors for shoulder dystocia include some of the things mentioned in the question stem, as well as anything else that causes a mismatch between the "passenger" and the "passage." Fetal macrosomia, gestational diabetes, maternal obesity, postdate pregnancy, and prolonged second stage of labor are all identified risk factors. 2) Treatment for shoulder dystocia involves a number of obstetric maneuvers. Besides suprapubic pressure, delivery of the posterior arm and shoulder, flexion of the maternal hips, and rotation of the infant (the "corkscrew" maneuver) are all first-line treatments. If these fail, then more dramatic steps such as fracturing the fetal clavicles or maternal symphysis may be required. 3) Shoulder dystocia tends to show up in pediatrics questions as well because of its tendency to cause brachial plexus injuries in the infant. The most common injury is Erb's palsy, where damage to C5 and C6 result in the classic "waiter's tip" hand position. For a review of shoulder dystocia, try this one from the American Family Physician: http://www.aafp.org/afp/20040401/1707.html So long as the infant is doing well, Caesarean section (answer A) is inappropriate before attempting some of the obstetrical maneuvers described above. If the situation is dire or other methods have failed, it is possible to place the infant's head back in the pelvis and go to Caesarean section (this is called the Zavanelli maneuver). Oxytocin (answer C) could increase the strength of the uterine contractions, but that will not likely help the situation. Remember the "three P's" of labor? The problem here is not with the patient's "power" – it's with a mismatch between the "passage" (or pelvis) and the "passenger," so treat it appropriately. Fracturing the baby's clavicles (answer E) is an effective treatment for dystocia, but because it does entail some morbidity, it is reserved for cases where more simple manipulations do not suffice.

E. Fracturing the baby's clavicles (answer E) is an effective treatment for dystocia, but because it does entail some morbidity, it is reserved for cases where more simple manipulations do not suffice.

This question is not currently linked to the learning objective database.

Question problem?

Question # 50

Select the single best answer to the numbered question.

A three-week old infant is brought to the clinic after the patient's adoptive parents noted a red rash on her hands and feet. The child was born at term via vaginal delivery to a 28 year old woman who received no prenatal care and gave her child up for adoption shortly after birth. On physical examination, the infant appears lethargic and has generalized lymphadenopathy. There is profuse, blood-tinged purulent nasal discharge, and there is an erythematous maculopapular rash on the dorsal and ventral surfaces of the hands and feet. Abdominal examination reveals hypoactive bowel sounds and hepatosplenomegaly. Which of the following is the most likely explanation of these findings?

A. Congenital toxoplasmosis
B. Congenital rubella

B. Congenital rubella
C. Congenital CMV infection
D. Congenital syphilis
E. Herpes neonatorum

You did not answer this question.

Explanations:

A. The classic triad of congenital toxoplasmosis (answer A) consists of hydrocephalus, chorioretinitis, and intracranial calcifications.

B. Congenital rubella (answer B) causes deafness and cataracts as well as numerous purpuric skin lesions, leading to the presentation of the so-called “blueberry muffin” baby. Congenital rubella syndrome occurs when the mother contracts rubella early on in her pregnancy – the risk of congenital rubella syndrome is very low after 20 weeks. Since the MMR vaccine contains a live attenuated virus, there is at least a theoretical risk of causing congenital rubella syndrome, and for this reason the vaccine is avoided in pregnant women.

C. Congenital CMV infection (answer C) is difficult to diagnose, because most of the key features (jaundice, deafness, chorioretinitis, thrombocytopenia/purpura, organomegaly) overlap so much with other TORCH infections. Acute CMV infection in adults can cause an infectious mononucleosis-like syndrome, so this may be given as a clue in the question stem.

D. This is congenital syphilis (answer D). The key findings on physical exam were the rash involving the palms and soles, and the blood-tinged purulent nasal discharge (known as the “snuffles”). Lymphadenopathy and organomegaly are also characteristic. This question brings up the issue of the TORCH infections, which is an acronym for Toxoplasmosis, Other (including HIV and syphilis), Rubella, CMV, and HSV. All of these infections can cause poor feeding, fever, deafness, mental retardation, hepatosplenomegaly, and thrombocytopenia, and so you should always consider them when you see an infant with one of those conditions. (A history of no or limited prenatal care is usually a giveaway that a question is looking for a problem – such as a TORCH infection - that would have been prevented with normal prenatal care.) Each of the TORCH infections does have its own unique characteristics and “buzzwords” that help you distinguish it clinically from the others. The classic triad of congenital toxoplasmosis (answer A) consists of hydrocephalus, chorioretinitis, and intracranial calcifications. Congenital rubella (answer B) causes deafness and cataracts as well as numerous purpuric skin lesions, leading to the presentation of the so-called “blueberry muffin” baby. Congenital rubella syndrome occurs when the mother contracts rubella early on in her pregnancy – the risk of congenital rubella syndrome is very low after 20 weeks. Since the MMR vaccine contains a live attenuated virus, there is at least a theoretical risk of causing congenital rubella syndrome, and for this reason the vaccine is avoided in pregnant women. Congenital CMV infection (answer C) is difficult to diagnose, because most of the key features (jaundice, deafness, chorioretinitis, thrombocytopenia/purpura, organomegaly) overlap so much with other TORCH infections. Acute CMV infection in adults can cause an infectious mononucleosis-like syndrome, so this may be given as a clue in the question stem.

E. Herpes neonatorum (answer E) causes vesicular lesions on the skin, eyes, or mouth, but can cause life-threatening disseminated disease or encephalitis. The risk is greatest in infants born to mothers who had a primary HSV infection during pregnancy or who had a vaginal delivery at the time of a herpes outbreak.

This question is not currently linked to the learning objective database.

Question problem?

Question # 51

Select the single best answer to the numbered question.

A 56 year old man returns to the office because his calcium was found to be elevated at a previous visit. Although the remainder of his labs, including complete blood count, liver panel, and chemistries were all within normal limits, the patient’s calcium was 11.9 mg/dL (normal: 8.5-10.5 mg/dL). The patient feels well and has no complaints. Current medications include verapamil, cetirizine, and atorvastatin. EKG shows a right bundle branch block, stable from previous. Which of the following is the most appropriate initial step in the management of this patient?

A. Serum parathyroid hormone level
B. CT of chest/abdomen
C. Genetic testing for multiple endocrine neoplasia, type 1
D. Discontinue verapamil
E. Intravenous saline and furosemide

You did not answer this question.

Explanations:

A. There is a long list of things that can cause hypercalcemia, but over 80-90% of cases are caused by either malignancy or hyperparathyroidism. Of these two, the easiest to rule out is hyperparathyroidism, which is easily evaluated by drawing a serum PTH level (answer A). A few other teaching points… 1) Remember the CHIMPANZEES mnemonic for hypercalcemia? C – Calcium oversupplementation, H – Hyperparathyroidism, I – Immobility or Iatrogenic (i.e., from thiazide diuretics), M – Milk alkali syndrome, P – Paget’s disease, A – Acromegaly or Addison’s disease, N – Neoplasms, Z – Zollinger-Ellison syndrome (when it’s associated with MEN-1), E – Excess Vitamin D, E – Excess Vitamin A, S – Sarcoidosis. 2) You can remember the key signs and symptoms of hypercalcemia with the following mnemonic: “Bones, Stones, Abdominal Groans, and Psychiatric Undertones,” because the most common symptoms include bone fractures, kidney stones, vomiting and constipation, and weakness, fatigue, and altered mental status. 3) Severe hypercalcemia or hypercalcemic crisis (usually defined as the presence of severe symptoms or a calcium level over 14 mg/dL) is a medical emergency because of its tendency to cause heart conduction abnormalities. Check an EKG, and then begin therapy with i.v. fluid and furosemide. (Remember, “loops lose” calcium, while thiazide diuretics can increase calcium.) A CT of the chest and abdomen (answer B) could help you find a hidden cancer, but it would be easier just to check a parathyroid hormone related protein (PTHrp) level to see if malignancy is the cause of hypercalcemia. Checking the PTHrp might be the next step if this patient has normal PTH levels. Genetic testing for multiple endocrine neoplasia, type 1 (answer C) is available, but is not the best step right now. Patients with MEN-1 do almost always develop parathyroid tumors, but you should first confirm that hyperparathyroidism is the cause of this patient’s hypercalcemia. Verapamil (answer D) is a calcium channel blocker, but it has no clinical effect on serum calcium levels. Intravenous saline and furosemide (answer E) is appropriate therapy for a patient in hypercalcemic crisis, but this patient has no symptoms and an unchanged EKG.

B. A CT of the chest and abdomen (answer B) could help you find a hidden cancer, but it would be easier just to check a parathyroid hormone related protein (PTHrp) level to see if malignancy is the cause of hypercalcemia. Checking the PTHrp might be the next step if this patient has normal PTH levels.

C. Genetic testing for multiple endocrine neoplasia, type 1 (answer C) is available, but is not the best step right now. Patients with MEN-1 do almost always develop parathyroid tumors, but you should first confirm that hyperparathyroidism is the cause of this patient’s hypercalcemia.

D. Verapamil (answer D) is a calcium channel blocker, but it has no clinical effect on serum calcium levels.

E. Intravenous saline and furosemide (answer E) is appropriate therapy for a patient in hypercalcemic crisis, but this patient has no symptoms and an

This question is not currently linked to the learning objective database.

Question problem?

Question # 52

Select the single best answer to the numbered question.

An 83 year old female with dementia is brought to the physician by her granddaughter because of a change in her skin color, which has taken on a golden hue over the past few days. The patient’s granddaughter also reports that she has been unable to get the patient to eat or drink for the past 24 hours, and that although the patient’s urine output has decreased, her urine appeared dark brown when she changed her diaper this morning. Vital signs are temperature 39.1 C (102.4 F), pulse 105, blood pressure 77/45, respiratory rate 24, oxygen saturation 96% on room air. On physical examination, the patient is somnolent but arousable to pain. The skin is jaundiced and the sclerae are icteric. Lungs are clear to auscultation. Abdominal examination reveals decreased bowel sounds, with diffuse tenderness to palpation. There is voluntary guarding of the right upper quadrant. Which of the following is the most appropriate next step in management for this patient?

A. Abdominal CT
B. Administer 1 L bolus of 0.9% NaCl
C. Begin i.v. nafcillin
D. Urgent endoscopic retrograde pancreaticoduodenoscopy (ERCP)
E. Percutaneous transhepatic drainage tube placement

You did not answer this question.

Explanations:

A. If you answered A, C, D, or E, you missed the boat. While each of these responses potentially has some role in the diagnosis or treatment of acute cholangitis, they do nothing to help the patient’s most immediate problem. The USMLE loves this kind of question, so the take home teaching point is to ALWAYS follow the ABCs, just as you would if you were assessing a patient in real life. Abdominal CT (answer A) may help you make the diagnosis of acute cholangitis. However, since this is such a serious disease, the diagnosis is often made clinically. Radiologic diagnosis with CT or ultrasound can be helpful if the diagnosis is in doubt, but would not alter your management plan in a case as clear-cut as this one.

B. Key teaching point: Always, always remember the ABCs! This is acute cholangitis. The classic features of Charcot’s triad (RUQ pain, jaundice, and fever/chills) are all present, and the patient is rapidly moving toward the full Reynold’s pentad (Charcot’s triad plus shock and mental status changes). However, to answer this question correctly, you need not know the diagnosis – all you need to know are the ABCs (airway, breathing, and circulation). While this lady has a patent airway and is moving air appropriately, her blood pressure is too low. Her hypoperfusion will soon lead to end-organ damage if you do not expand her intravascular compartment, and the best way to do that is with an isotonic fluid like normal saline (0.9% NaCl, answer B) or lactated Ringer’s. If you answered A, C, D, or E, you missed the boat. While each of these responses potentially has some role in the diagnosis or treatment of acute cholangitis, they do nothing to help the patient’s most immediate problem. The USMLE loves this kind of question, so the take home teaching point is to ALWAYS follow the ABCs, just as you would if you were assessing a patient in real life. Abdominal CT (answer A) may help you make the diagnosis of acute cholangitis. However, since this is such a serious disease, the diagnosis is often made clinically. Radiologic diagnosis with CT or ultrasound can be helpful if the diagnosis is in doubt, but would not alter your management plan in a case as clear-cut as this one. Intravenous antibiotics (answer C) are definitely indicated in this patient – just not as immediately as i.v. fluid. However, the most common pathogens in acute cholangitis are enterics like E.coli, Klebsiella, Enterobacter, and Pseudomonas. The best antibiotic coverage, then, would target Gram negatives and anaerobes – the combination of ciprofloxacin and metronidazole is probably the most commonly used in the hospital. Nafcillin is considered an antistaphylococcal penicillin, and while it has excellent activity on MSSA and Gram positives, these are not the most likely pathogens for infections in the gut, so nafcillin would never be indicated for empiric therapy of enteric infections. Both ERCP (answer D) and percutaneous drainage (answer E) are appropriate ultimate therapies for a patient with acute suppurative cholangitis – which this patient almost certainly has given the appearance of Reynold’s pentad. Such patients need urgent decompression of their bile ducts, and ERCP, percutaneous drainage, or open surgical decompression can be used. As mentioned before, though, the most important first thing to do is to improve this patient’s blood pressure.

C. If you answered A, C, D, or E, you missed the boat. While each of these responses potentially has some role in the diagnosis or treatment of acute cholangitis, they do nothing to help the patient’s most immediate problem. The USMLE loves this kind of question, so the take home teaching point is to ALWAYS follow the ABCs, just as you would if you were assessing a patient in

real life. Intravenous antibiotics (answer C) are definitely indicated in this patient – just not as immediately as i.v. fluid. However, the most common pathogens in acute cholangitis are enterics like E.coli, Klebsiella, Enterobacter, and Pseudomonas. The best antibiotic coverage, then, would target Gram negatives and anaerobes – the combination of ciprofloxacin and metronidazole is probably the most commonly used in the hospital. Nafcillin is considered an antistaphylococcal penicillin, and while it has excellent activity on MSSA and Gram positives, these are not the most likely pathogens for infections in the gut, so nafcillin would never be indicated for empiric therapy of enteric infections.

D. If you answered A, C, D, or E, you missed the boat. While each of these responses potentially has some role in the diagnosis or treatment of acute cholangitis, they do nothing to help the patient's most immediate problem. The USMLE loves this kind of question, so the take home teaching point is to ALWAYS follow the ABCs, just as you would if you were assessing a patient in real life. Both ERCP (answer D) and percutaneous drainage (answer E) are appropriate ultimate therapies for a patient with acute suppurative cholangitis – which this patient almost certainly has given the appearance of Reynold's pentad. Such patients need urgent decompression of their bile ducts, and ERCP, percutaneous drainage, or open surgical decompression can be used. As mentioned before, though, the most important first thing to do is to improve this patient's blood pressure.

E. If you answered A, C, D, or E, you missed the boat. While each of these responses potentially has some role in the diagnosis or treatment of acute cholangitis, they do nothing to help the patient's most immediate problem. The USMLE loves this kind of question, so the take home teaching point is to ALWAYS follow the ABCs, just as you would if you were assessing a patient in real life. Both ERCP (answer D) and percutaneous drainage (answer E) are appropriate ultimate therapies for a patient with acute suppurative cholangitis – which this patient almost certainly has given the appearance of Reynold's pentad. Such patients need urgent decompression of their bile ducts, and ERCP, percutaneous drainage, or open surgical decompression can be used. As mentioned before, though, the most important first thing to do is to improve this patient's blood pressure.

This question is not currently linked to the learning objective database.

Question problem?

Question # 53

Select the single best answer to the numbered question.

A 20 year old previously healthy college female presents with two weeks of occasional dysuria and increased vaginal discharge. She has recently become sexually active, and she and her asymptomatic partner occasionally use condoms as their only method of birth control. Speculum exam reveals a friable cervix with a moderate amount of purulent cervical discharge. The patient tests positive for Chlamydia and negative for gonorrhea. UA shows pyuria but no organism is identified on gram stain. Urine culture is negative. What is the next best step in management for this patient?

A. Ciprofloxacin
B. Ceftriaxone and doxycycline
C. Azithromycin
D. Benzathine penicillin G
E. Reassurance

You did not answer this question.

Explanations:

A. A single dose of ciprofloxacin (choice A) is an acceptable treatment for gonorrhea, although bacterial resistance to fluoroquinolones is increasing.

B. The ceftriaxone and doxycycline combination (choice B) would be an appropriate choice for treating a patient with both gonorrhea and chlamydia, OR a patient with a positive gonorrhea test to address the likelihood of dual infection.

C. This question addresses the appropriate management for isolated Chlamydia infection. There are several antimicrobial options, with the two most frequently used being either a single dose of azithromycin (answer C) or a week of doxycycline. A one week course of erythromycin is also effective, but has been associated with GI upset. Fluoroquinolones like ofloxacin and levofloxacin are more expensive alternatives. For the pregnant patient, either azithromycin or erythromycin are acceptable. A few extra teaching points: 1) When you suspect Chlamydia, you must also test for gonorrhea and vice versa. This is convenient because most of the time, one swab is used for both tests which are automatically sent together. 2) Testing positive for only Chlamydia mandates treatment for only Chlamydia, BUT testing positive for only gonorrhea mandates treatment for BOTH gonorrhea and Chlamydia. Chlamydia is the most common STI, and patients with gonorrhea (which is much less prevalent than Chlamydia) tend to be co-infected with chlamydia. The reverse is much less often true since Chlamydia has a much higher prevalence in the population. 3) If a patient tests positive for Chlamydia or gonorrhea, her partner MUST be treated or she will likely be reinfected. Often male partners are asymptomatic. Health departments in some states employ a system of anonymous sexual partner notification, but patients should be urged to notify their partners as well. The other answer choices are incorrect for the management of acute Chlamydia. A single dose of ciprofloxacin (choice A) is an acceptable treatment for gonorrhea, although bacterial resistance to fluoroquinolones is increasing. The ceftriaxone and doxycycline combination (choice B) would be an appropriate choice for treating a patient with both gonorrhea and chlamydia, OR as discussed above, a patient with a positive gonorrhea test to address the likelihood of dual infection. IM benzathine penicillin G (choice D) is the one-time injection treatment option for early syphilis infection. Reassurance (choice E) is absolutely the wrong management choice for someone who has just tested positive for Chlamydia. If left untreated, about 30% of women with Chlamydia will develop pelvic inflammatory disease (PID). This inflammation of the upper genital tract is associated with infertility due to scarring of the fallopian tubes.

D. IM benzathine penicillin G (choice D) is the one-time injection treatment option for early syphilis infection.

E. Reassurance (choice E) is absolutely the wrong management choice for someone who has just tested positive for Chlamydia. If left untreated, about 30% of women with Chlamydia will develop pelvic inflammatory disease (PID). This inflammation of the upper genital tract is associated with infertility due to scarring of the fallopian tubes.

This question is not currently linked to the learning objective database.

Question problem?

Question # 54

Select the single best answer to the numbered question.

A 35 year old man presents to his physician with neck pain that began suddenly while at rest four hours ago. His past medical history is significant for a ten-year history of disorganized schizophrenia, which has been treated unsuccessfully with a number of medications including olanzapine, risperidone, and haloperidol, and he has been recently switched to thiothixene. Examination shows a disheveled male with a flat affect, holding his head twisted to the right. There is limited cervical range of motion and pronounced muscle contraction of the sternocleidomastoid and paracervical muscles. Which of the following is the most appropriate pharmacologic intervention for this patient's condition?

A. Dantrolene i.v.
B. Increase p.o. thiothixene
C. Add clozapine
D. Oral ibuprofen
E. Oral diphenhydramine

You did not answer this question.

Explanations:

A. Intravenous dantrolene (answer A) is the approprate therapy for neuroleptic malignant syndrome (NMS), a life-threatening side effect of typical antipsychotics. Features of this disorder include fever, muscle rigidity, and autonomic instability; lab values will show an increased CPK. NMS is a medical emergency, and should be treated with i.v. dantrolene or bromocriptine as well as vigorous i.v. hydration.

B. Increasing thiothixene (answer B) is not the correct answer – thiothixene has caused this patient's dystonia, so increasing it is not likely to make the situation any better. While it is true that this patient is still having some symptoms of his schizophrenia (as evidenced by his disheveled appearance) his dystonia is the more pressing issue. Even though increasing thiothixene may help improve the patient's schizophenic symptoms in the long run, it's not the best choice now.

C. Clozapine (answer C) is an atypical antipsychotic and an effective treatment for refractory schizophrenia. Given this patient's history of medication failure, its use may well be indicated for this patient at some point in the future – but right now, he needs treatment for an acute dystonic reaction. Although clozapine is the most effective medication for treating refractory schizophrenia, it carries a 0.5-1% risk of agranulocytosis, and thus its use requires weekly CBCs to monitor for this potentially life-threatening side effect. For this reason, clozapine is typically only used in patients who have failed other treatments or who have severe tardive dyskinesia as a result of typical antipsychotic use.

D. An NSAID like ibuprofen (answer D) is often a good treatment for benign neck pain - once more worrisome causes of pain have been ruled out. This patient's neck pain is not caused by minor trauma or a simple muscle strain, though – it is caused by muscle spasm brought on by the cholinergic effects of his antipsychotic medication. Ibuprofen might help treat some of this patient's pain, but diphenhydramine's anticholinergic effects will target the cause of the patient's pain.

E. This patient has experienced an acute dystonic reaction caused by his antipsychotic medication. The so-called "typical" antipsychotics (like haloperidol, thiothixene, chlorpromazine, and fluphenazine) have powerful dopamine blocking properties, which make them ideal for treating the positive symptoms of schizophrenia like hallucinations or delusions. Blocking dopamine has the indirect effect of increasing acetylcholine, so anticholinergics that penetrate the blood-brain barrier like benztropine or diphenhydramine (answer E) are the treatments of choice for these extrapyramidal reactions. The actions of typical antipsychotics outside of the nigrostriatal tract are collectively called extrapyramidal symptoms. These include: Akathisia – a subjective feeling of restlessness that develops several weeks after beginning medication; best treated by decreasing the antipsychotic, although benzodiazepines, beta-blockers, and anticholinergics may help. Pseudoparkinsonism or dyskinesia – includes classic Parkinsonian features such as a shuffling gait and cogwheel rigidity; best treated by withdrawing or decreasing the offending medication or adding a dopamine agonist like amantadine Tardive dyskinesia (TD) – nonsuppressable stereotypical facial and oral movements such as lip smacking. Decreasing or discontinuing the antipsychotic or adding an anticholinergic may initially make TD worse, since the disorder is thought to be due to dopaminergic receptor sensitization. Many – if not most - cases of TD are irreversible. Intravenous dantrolene (answer A) is the approprate therapy for neuroleptic malignant syndrome (NMS), a life-threatening side effect of typical antipsychotics. Features of this disorder include fever, muscle rigidity, and autonomic instability; lab values will show an increased CPK. NMS is a medical emergency, and should be treated with i.v. dantrolene or bromocriptine as well as vigorous i.v. hydration. Increasing thiothixene (answer B) is not the correct answer – thiothixene has caused this patient's dystonia, so increasing it is not likely to make the situation any better. While it is true that this patient is still having some symptoms of his schizophrenia (as evidenced by his disheveled appearance) his dystonia is the more pressing issue. Even though increasing thiothixene may help improve the patient's schizophenic symptoms in the long run, it's not the best choice right now. Clozapine (answer C) is an atypical antipsychotic and an effective treatment for refractory schizophrenia. Given this patient's history of medication failure, its use may well be indicated for this patient at some point in the future – but right now, he needs treatment for an acute dystonic reaction. Although clozapine is the most effective medication for treating refractory schizophrenia, it carries a 0.5-1% risk of agranulocytosis, and thus its use requires weekly CBCs to monitor for this potentially life-threatening side effect. For this reason, clozapine is typically only used in patients who have failed other treatments or who have severe tardive dyskinesia as a result of typical antipsychotic use. An NSAID like ibuprofen (answer D) is often a good treatment for benign neck pain - once more worrisome causes of pain have been ruled out. This patient's neck pain is not caused by minor trauma or a simple muscle strain, though – it is caused by muscle spasm brought on by the cholinergic effects of his antipsychotic medication. Ibuprofen might help treat some of this patient's pain, but diphenhydramine's anticholinergic effects will target the cause of the patient's pain.

This question is not currently linked to the learning objective database.

Question problem?

Question # 55

Select the single best answer to the numbered question.

A 37 year old HIV+ male presents with one week of headache and low grade fever. For the past two days, he has had nausea and vomiting. On physical exam, temperature is 38.1 C (100.6 F), pulse 80, blood pressure 108/68, respirations 15/min. The patient is somnolent but arousable. Nuchal rigidity is present. With the patient supine, passive knee extension elicits neck pain. Lungs are clear to auscultation. There are no petechiae or cutaneous hemorrhages seen. CT scan shows no focal lesion, and lumbar puncture is performed. CSF analysis shows clear, light yellow CSF with an opening pressure of 290 mm Hg (normal: 70-180 mm Hg), 25 WBC (normal 0-5) with 96% lymphocytes, 15 RBCs, a glucose of 38 mg/dL (normal: 40-70 mg/dL)and protein 50 mg/dL (normal: 15-45 mg/dL). Which of the following would be the most appropriate therapy for this patient?

A. Ceftriaxone
B. Acyclovir
C. Vancomycin and cefotaxime
D. Trimethoprim-sulfamethoxazole
E. Amphotericin B and flucytosine

You did not answer this question.

Explanations:

A. Ceftriaxone (answer A), a third generation cephalosporin, is good empiric therapy for bacterial meningitis when there is little concern for drug-resistant Streptococcus pneumoniae (DRSP). Ceftriaxone penetrates the blood-brain barrier well, and covers all of the common causes of meningitis in adults with the notable exception of DRSP.

B. Acyclovir (answer B) is used to treat herpes encephalitis, which can present with increased lymphocytes in the CSF. However, the hallmark of herpes encephalitis on CSF analysis is a markedly elevated RBC.

C. Combination therapy with vancomycin and cefotaxime (answer C) is used for empiric therapy of bacterial meningitis when there is concern for drug resistant S.pneumoniae. Cefotaxime, like ceftriaxone, is a third-generation cephalosporin that penetrates the blood-brain barrier. It has good activity against N. meningitides, H.flu, Listeria, and most strains of S.pneumo. Vancomycin covers the possibility of DRSP. Patients who have a known CSF leak (from trauma or shunting) or who have had recent neurosurgery also generally receive vancomycin to cover MRSA, though without these predisposing factors Staphylococcus is a very rare cause of meningitis.

D. Trimethoprim/sulfamethoxazole (answer D) is used in the prophylaxis or treatment of a variety of AIDS-related infections, including Pneumocystis carinii and Toxoplasma.

E. Key teaching point: An isolated elevated opening pressure is characteristic of cryptococcal meningitis. This question describes clinical symptoms and signs of meningitis in an immunocompromised patient. Although such patients certainly can get meningitis caused by the usual bugs like Streptococcus pneumoniae and Neisseria meningitides, you also have to worry about other causes like fungus in general and Cryptococcus in particular. This patient's LP shows lymphocytosis and a dramatically increased opening pressure, which are the key features of cryptococcal infection. Treatment of cryptococcal meningitis requires aggressive antifungal therapy with amphotericin B and flucytosine (answer E). Some additional teaching points for this case: 1) To confirm the diagnosis, you should order a latex agglutination test for cryptococcal antigen (CrAg) or an India ink preparation to look for encapsulated yeasts. 2) In the setting of advanced AIDS, the CSF may even appear normal because the patient is unable to mount a significant inflammatory immune response. Don't be fooled by near-normal values in an immunocompromised patient! 3) Cryptococcus can form focal "cryptococcomas" that behave as mass lesions and increase ICP. Before doing an LP, remember to look for focal neurological deficits or get a CT to prevent herniation. 4) In real life, given the patient's immunocompromised status, it would not be unreasonable to start empiric antibacterial agents in addition to the amphotericin/flucytosine. However, the CSF in bacterial meningitis should have a neutrophilic predominance, and the glucose should be lower and the protein should be higher than is seen in this case. Ceftriaxone (answer A), a third generation cephalosporin, is good empiric therapy for bacterial meningitis when there is little concern for drug-resistant Streptococcus pneumoniae (DRSP). Ceftriaxone penetrates the blood-brain barrier well, and covers all of the common causes of meningitis in adults with the notable exception of DRSP. Acyclovir (answer B) is used to treat herpes encephalitis, which can present with increased lymphocytes in the CSF. However, the hallmark of herpes encephalitis on CSF analysis is a markedly elevated RBC. Combination therapy with vancomycin and cefotaxime (answer C) is used for empiric therapy of bacterial meningitis when there is concern for drug resistant S.pneumoniae. Cefotaxime, like ceftriaxone, is a third-generation cephalosporin that penetrates the blood-brain barrier. It has good activity against N. meningitides, H.flu, Listeria, and most strains of S.pneumo. Vancomycin covers the possibility of DRSP. Patients who have a known CSF leak (from trauma or shunting) or who have had recent neurosurgery also generally receive vancomycin to cover MRSA, though without these predisposing factors Staphylococcus is a very rare cause of meningitis. Trimethoprim/sulfamethoxazole (answer D) is used in the prophylaxis or treatment of a variety of AIDS-related infections, including Pneumocystis carinii and Toxoplasma. Cryptococcal meningitis is a medical emergency that demands powerful pharmacological therapy.

This question is not currently linked to the learning objective database.

Question problem?

Question # 56

Select the single best answer to the numbered question.

A 68 year old woman has had worsening back pain for the past three months. Her pain radiates to her legs bilaterally. The pain is worst while walking or standing upright and is associated with weakness and numbness of her legs while walking. Sitting or lying down relieves the pain, but the patient also found that leaning forward over her grocery cart improved her symptoms while shopping earlier this morning. She has not had fever or bowel or bladder incontinence. Past medical history is significant for hypertension controlled with lisinopril. The patient does not smoke. Physical examination shows no tenderness along the lumbar spine and a negative straight-leg raising test. The feet are warm, and dorsalis pedis pulses are 2+ bilaterally. No saddle anesthesia is present, but the patient has weakness of the extensor hallucis longus bilaterally. Which of the following is the most likely pathological mechanism for this patient's symptoms?

A. Herniation of the nucleus pulposus against the L4 nerve roots
B. Pyogenic abscess of the epidural space in the lumbar spine
C. Atherosclerotic occlusion of the popliteal arteries
D. Malignant tumor invasion of the lumbar spine
E. Narrowing of the lumbar spinal canal

You did not answer this question.

Explanations:

A. A herniated disc compressing the nerve roots (answer A) is unlikely, since there would have to be bilateral and equal impingement to cause this patient's symptoms. Patients with herniated discs complain of shooting, "radicular" pain. The key physical exam maneuver to diagnose nerve impingement is the straight leg raise, where the patient's shooting pain is re-created by raising the contralateral leg. You should also seek out numbness in a dermatomal distribution corresponding to the level of the disc involved, as well as findings of motor weakness and reflex loss. If the L4 root were compressed, you would expect to find diminished knee jerk reflex (while S1 compression causes a diminished ankle jerk.) This patient's weakness of the extensor hallucis longus demonstrates involvement of the L5 nerve roots.

B. A pyogenic abscess of the epidural space (answer B), also known as a spinal epidural abscess, has a variable presentation depending on the location of the infection and the time of the patient's presentation in the natural history of the illness. It can be a difficult diagnosis to make, but on the USMLE you should expect to have clues that would suggest the diagnosis of infection or abscess, such as fever, localized spinal pain, a history of i.v. drug use, or a rapidly-progressive course (since the most likely causative organism is S.aureus).

C. Atherosclerotic occlusion of the popliteal arteries (answer C) causes peripheral arterial disease and symptoms of claudication. This can be difficult to distinguish from the "neurogenic claudication" of spinal stenosis, but this patient has good distal pulses and few vascular risk factors. A useful tool for assessing patients with claudication is the ankle-brachial index (ABI), whereby blood pressure measurements are taken in the arm and the ankle. If the ankle systolic pressure/arm systolic pressure is <0.6 the patient likely has claudication.

D. Cancer (answer D) can also be a cause of back pain, and like epidural abscesses, it can present in a variety of ways. Although the slowly progressive course could be consistent with malignancy, there are no other findings in the question stem to steer you in that direction. Rather, the question focuses on the history and physical exam findings – which serves as an indication that the testmakers want you to use that information to make your diagnosis. This is a valuable strategy to use on the USMLE or shelf exam – ask yourself, "What is this question supposed to be testing?" The types of clues offered in the question stem – even if you don't know exactly what they mean – may be enough to steer you toward one answer or another. In this case, cancer of the spine is such a variable disease that you would not be expected to diagnose it based on an H&P alone.

E. Key teaching point: An older patient with back pain that is worst while standing and walking but is relieved by sitting or spinal hyperflexion has lumbar spinal stenosis. This is lumbar spinal stenosis, caused most often by age-related degeneration of the lumbar spine and intervertebral discs leading to pathological canal narrowing (answer E). This leads to compression and ischemia of the spinal nerve roots, which in turn causes the typical presentation of "neurogenic claudication," where symptoms occur during ambulation because of increased metabolic demands of the nerve roots. Flexing the spine increases the canal size and relieves the stenosis, and patients may describe a variety of maneuvers involving hyperflexion of the spine that they have found to relieve the pain (such as this patient leaning over her grocery cart). Although symptoms can be managed medically, the treatment of lumbar spinal stenosis is ultimately surgical. A herniated disc compressing the nerve roots (answer A) is unlikely, since there would have to be bilateral and equal impingement to cause this patient's symptoms. Patients with herniated discs complain of shooting, "radicular" pain. The key physical exam maneuver to diagnose nerve impingement is the straight leg raise, where the patient's shooting pain is re-created by raising the contralateral leg. You should also seek out numbness in a dermatomal distribution corresponding to the level of the disc involved, as well as findings of motor weakness and reflex loss. If the L4 root were compressed, you would expect to find diminished knee jerk reflex (while S1 compression causes a diminished ankle jerk.) This patient's weakness of the extensor hallucis longus demonstrates involvement of the L5 nerve roots. A pyogenic abscess of the epidural space (answer B), also known as a spinal epidural abscess, has a variable presentation depending on the location of the infection and the time of the patient's presentation in the natural history of the illness. It can be a difficult diagnosis to make, but on the USMLE you should expect to have clues that would suggest the diagnosis of infection or abscess, such as fever, localized spinal pain, a history of i.v. drug use, or a rapidly-progressive course (since the most likely causative organism is S.aureus). Atherosclerotic occlusion of the popliteal arteries (answer C) causes peripheral arterial disease and symptoms of claudication. This can be difficult to distinguish from the "neurogenic claudication" of spinal stenosis, but this patient has good distal pulses and few vascular risk factors. A useful tool for assessing patients with claudication is the ankle-brachial index (ABI), whereby blood pressure measurements are taken in the arm and the ankle. If the ankle systolic pressure/arm systolic pressure is <0.6 the patient likely has claudication. Cancer (answer D) can also be a cause of back pain, and like epidural abscesses, it can present in a variety of ways. Although the slowly progressive course could be consistent with malignancy, there are no other findings in the question stem to steer you in that direction. Rather, the question focuses on the history and physical exam findings – which serves as an indication that the testmakers want you to use that information to make your diagnosis. This is a valuable strategy to use on the USMLE or shelf exam – ask yourself, "What is this question supposed to be testing?" The types of clues offered in the question stem – even if you don't know exactly what they mean – may be enough to steer you toward one answer or another. In this case, cancer of the spine is such a variable disease that you would not be expected to diagnose it based on an H&P alone.

This question is not currently linked to the learning objective database.

Question problem?

Question # 57

Select the single best answer to the numbered question.

A 34 year old female presents to her family physician in tears because she wants to have a baby. She has had three second trimester miscarriages over the past five years. She and her husband, who has two children from a previous marriage, desperately want to conceive, and their failure to do so has begun to cause stress in their marriage. The patient is otherwise healthy, and there is no history of sexually-transmitted infections or hematologic or rheumatologic diseases. Menarche began at age 11 and her menses are regular at intervals of 25 days. Her pelvic exam reveals a slightly retroverted uterus and normal size ovaries. What is the most likely etiology of this patient's recurrent pregnancy losses?

A. Maternal balanced translocation
B. Cervical incompetence
C. Paternal balanced translocation
D. Retroverted uterus
E. Amniotic band syndrome

You did not answer this question.

Explanations:

A. Maternal balanced translocation (choice A) is not the best answer. Balanced translocations are a cause of early pregnancy loss. It is more common for the female to carry a balanced translocation, and it is also more likely that the translocation will result in pregnancy loss if it is from maternal origin. Still, we would expect this to be a more likely cause of first trimester loss.

B. This question addresses the etiology of recurrent pregnancy loss (RPL), defined as 3 or more consecutive losses of pregnancies before the 20th week of gestation. A good way to remember causes of recurrent pregnancy loss is to think about the timing of the loss. Genetic causes tend to cause early pregnancy loss, while anatomical factors tend to cause losses later in the pregnancy. An incompetent cervix (answer B) is the only anatomical cause of RPL listed and is therefore the best answer in this case. A few teaching points… 1) The earlier the gestational age at abortion, the higher the incidence of cytogenetic defects. Abnormalities of chromosome number or structure are the most common cause of early pregnancy loss. 2) Anatomical causes of recurrent pregnancy loss tend to manifest later in pregnancy as fetal size increases. These include cervical incompetence, uterine anomalies, leiomyoma and intrauterine synechiae. 3) Immunological causes can cause pregnancy loss at any time. Antiphospholipid antibodies, for example, tend to cause both recurrent early miscarriages and repetitive late miscarriages. Maternal balanced translocation (choice A) is not the best answer. Balanced translocations are a cause of early pregnancy loss. It is more common for the female to carry a balanced translocation, and it is also more likely that the translocation will result in pregnancy loss if it is from maternal origin. Still, we would expect this to be a more likely cause of first trimester loss. Paternal balanced translocation (choice C) is not the best answer for a second semester pregnancy loss; we would expect this to cause fetal termination earlier in the pregnancy. Also, since the patient's husband has had children in previous relationships, paternal causes of infertility are less likely. A retroverted uterus (choice D) is not correct. This answer was just a distractor; a retroverted uterus is considered a normal variant of uterine position. Amniotic band syndrome (choice E) is a cause of congenital malformation, usually leading to structural limb abnormalities including complete amputation. The defects are believed to be caused by strands of amnion wrapping around a fetal body part, constricting its growth. This syndrome is not a cause of RPL.

C. Paternal balanced translocation (choice C) is not the best answer for a second semester pregnancy loss; we would expect this to cause fetal termination earlier in the pregnancy. Also, since the patient's husband has had children in previous relationships, paternal causes of infertility are less likely.

D. A retroverted uterus (choice D) is not correct. This answer was just a distractor; a retroverted uterus is considered a normal variant of uterine position.

E. Amniotic band syndrome (choice E) is a cause of congenital malformation, usually leading to structural limb abnormalities including complete amputation. The defects are believed to be caused by strands of amnion wrapping around a fetal body part, constricting its growth. This syndrome is not a cause of RPL.

This question is not currently linked to the learning objective database.

Question problem?

Question # 58

Select the single best answer to the numbered question.

A mother brings her 8 year old son to the family physician because of strange behavior for the past year that appears to be getting worse. For the past twelve months, the patient's mother has noted episodes of repetitive blinking or grimacing. These episodes appear to be beyond her son's control, and often occur at inopportune times. The patient is now being teased at school by his classmates for these behaviors. During the patient interview, the child appears to be listening intently to the conversation with his mother; there is a minute or so of rapid, repetitive blinking followed by several grunting noises from the patient. When asked, the child is aware of the blinking and grunting, but cannot explain why he was doing it. The patient denies hallucinations or bizarre thoughts and states that his mood is fine. He does well in school and has a close group of friends in his neighborhood who he enjoys playing with. His growth and development are within normal limits. What is the most likely diagnosis in this patient?

A. Autism
B. Rett syndrome
C. Tourette syndrome
D. Sydenham chorea
E. Attention deficit hyperactivity disorder

You did not answer this question.

Explanations:

A. Autism (choice A) should be suspected in kids with abnormalities in social interaction, communication, and development that begin before age 3. Patients can present with poor language development, self-injurious behavior, the use of repetitive, stereotyped behaviors, or sleep/eating disturbances. Autism is not an appropriate diagnosis for our patient who enjoys social interactions and is developmentally on target.

B. Rett syndrome (choice B) is a pervasive developmental disorder that occurs almost exclusively in females. Patients at first demonstrate normal development and then gradually lose speech and purposeful hand use. Austic symptoms, seizures, decreased head growth, stereotypic hand movements, ataxia and breathing difficulties follow. Developmental arrest can begin as early as 6 months of age, but the period of rapid deterioration usually begins between ages 2-4 years old, with the late motor deterioration occurring at around age 10. Because it makes such a distinctive diagnosis (and serves as a nice distractor), Rett syndrome makes frequent appearances on the USMLE.

C. This is a classic case of Tourette syndrome (answer C), a neurological disorder characterized by motor and phonic tics with a childhood onset. Tics are defined as sudden, brief, intermittent movements or utterances that are considered to be involuntary though they can sometimes be suppressed. This disease is believed to be inherited in an autosomal dominant fashion. A few extra teaching points… 1) Tourette Syndrome is a clinical diagnosis. Diagnostic testing may help you rule out other causes, but it will not directly help you confirm Tourette's. 2) The diagnostic criteria include the presence of witnessed motor AND phonetic tics before age 21 that are not explained by an underlying medical condition. Tics must occur many times per day, more days than not, for a period of more than one year. 3) Treatment options include dopamine agonists/antagonists, botulinum toxin injection for focal vocal and motor tics, alpha agonists, and SSRIs. The goal of therapy is to reduce the number and frequency of tics in order to improve social interactions at school, work and play. The tics themselves are not harmful. 4) PANDAS, or pediatric autoimmune neuropsychiatric disorder associated with group A streptococci, is a tic disorder that has received much recent attention. It should be excluded in a patient suspected to have Tourette syndrome. There are five diagnostic criteria for PANDAS: pediatric onset, presence of obsessive compulsive disorder and/or a tic disorder, abrupt onset with episodic symptom course, associated with group A strep infections, association with neurological abnormalities like motoric hyperactivity, choreiform movements and tics. Antibiotic therapy tends to result in improvement of the OCD symptoms. Ultimately, however, the exact relationship between PANDAS and Tourette's is not clear. One study showed that children with multiple GAS infections over a 12 month period had an increased risk of developing Tourette's. In addition, children with Tourette syndrome, OCD, or a tic disorder were more likely than controls to have had a GAS infection within 3 months of the onset of neurological symptoms. The other answers are not the most likely cause of this child's presentation. Autism (choice A) should be suspected in kids with abnormalities in social interaction, communication, and development that begin before age 3. Patients can present with poor language development, self-injurious behavior, the use of repetitive, stereotyped behaviors, or sleep/eating disturbances. Autism is not an appropriate diagnosis for our patient who enjoys social interactions and is developmentally on target. Rett syndrome (choice B) is a pervasive developmental disorder that occurs almost exclusively in females. Patients at first demonstrate normal development and then gradually lose speech and purposeful hand use. Austic symptoms, seizures, decreased head growth, stereotypic hand movements, ataxia and breathing difficulties follow. Developmental arrest can begin as early as 6 months of age, but the period of rapid deterioration usually begins between ages 2-4 years old, with the late motor deterioration occurring at around age 10. Because it makes such a distinctive diagnosis (and serves as a nice distractor), Rett syndrome makes frequent appearances on the USMLE. Sydenham chorea (choice D) is a movement disorder characterized by chorea, emotional lability and hypotonia. It is a component of acute rheumatic fever, which is a complication of group A streptococcal infection. Rheumatic fever presents 2-4 weeks after the pharyngeal infection, and symptoms include large joint migratory arthritis, carditis and valvulitis, rash, and central nervous system involvement (Sydenham chorea). Sydenham chorea usually resolves after 3 months. ADHD or attention deficit hyperactivity disorder (choice E) is a condition that manifests in early childhood with symptoms of inattention, hyperactivity and impulsivity. Symptoms usually affect all areas of life, from school to play, and usually persist into adulthood. This disorder is not associated with tics.

D. Sydenham chorea (choice D) is a movement disorder characterized by chorea, emotional lability and hypotonia. It is a component of acute rheumatic fever, which is a complication of group A streptococcal infection. Rheumatic fever presents 2-4 weeks after the pharyngeal infection, and symptoms include large joint migratory arthritis, carditis and valvulitis, rash, and central nervous system involvement (Sydenham chorea). Sydenham chorea usually resolves after 3 months.

E. ADHD or attention deficit hyperactivity disorder (choice E) is a condition that manifests in early childhood with symptoms of inattention, hyperactivity and impulsivity. Symptoms usually affect all areas of life, from school to play, and usually persist into adulthood. This disorder is not associated with tics.

This question is not currently linked to the learning objective database.

Question problem?

Question # 59

Select the single best answer to the numbered question.

A 51 year old supermarket cashier has had worsening pain and tenderness of her left leg. Her symptoms have been worsening for the past two days, ever since she had to work three consecutive days of "double shifts" due to a co-worker's absence. Medications include lisinopril for hypertension and hormone replacement therapy for hot flashes. Physical examination shows an obese female with temperature 38.1 C (100.5 F), pulse 88, BP 136/84, and respirations 14/min. There are numerous prominent varicose veins on the legs bilaterally. The left leg has a 6 inch (15 cm) linear area of erythema and edema on the medial aspect of the knee and calf, with a palpable, nodular, cordlike structure located just

underneath the skin. Doppler ultrasonography is performed on both legs and shows a single thrombus extending from the middle to distal saphenous vein on the left. Which of the following is the most appropriate next step in the management of this patient's condition?

A. Rest, elevation, non-steroidal anti-inflammatory agents, and heat
B. Enoxaparin
C. Warfarin
D. Spiral CT of the chest
E. Placement of inferior vena cava filter

You did not answer this question.

Explanations:

A. This patient has superficial thrombophlebitis, suggested by her history and physical and confirmed by ultrasound. Unlike patients with deep venous thrombosis, there is no need for anticoagulation, and treatment is symptomatic with rest, elevation, NSAIDs, and heat (answer A). A few additional teaching points from this case… 1) In addition to symptomatic treatment of her thrombophlebitis, this patient should also strongly consider discontinuing her hormone replacement therapy (HRT). While HRT is a very effective treatment for menopausal hot flashes, it also leads to a hypercoagulable state and increases the risk of both superficial venous thrombosis (SVT) and DVT. 2) Distinguishing between superficial and deep vein thrombosis is important, because while DVTs can cause pulmonary embolism, SVT does not. Both SVT and DVT can cause swelling, pain, and warmth of the affected extremity, but palpating a "cord" superficially is a giveaway that the thrombosis is truly superficial. (It is true that occasionally DVTs can be palpated, but remember that you are trying to palpate the DEEP veins of the leg – not an easy task in most patients!) Because superficial thrombophlebitis and DVT are caused by the same predisposing factors, it's always reasonable to get an ultrasound to confirm that there is not also an underlying deep vein thrombosis that could embolize. 3) The saphenous vein is a superficial vein – remember, it is harvested with a simple subcutaneous incision for CABG or vascular grafting! The femoral and popliteal veins are deep veins, so if there is a clot there, you need to start anticoagulation. A common trick is that the superficial femoral vein is still a deep vein, so a clot there requires treatment for DVT. Anticoagulation with heparin or enoxaparin (answer B) is the best immediate therapy for deep venous thrombosis. It prevents further propagation of the clot and reduces the incidence of pulmonary embolism. Because of its oral availability, warfarin (answer C) is the treatment of choice for long-term anticoagulation for DVT prophylaxis. Though you may start it acutely if a patient presents with a DVT, it will not become therapeutic for several days. Spiral CT (alternately called helical CT, CT angiography, or CT-PA, answer D) is used to diagnose pulmonary embolism, which is of course the most serious complication of deep venous thrombosis. This patient has superficial thrombophlebitis, however – not DVT. Moreover, she clinically lacks any symptoms of pulmonary embolism, so a CT would not be indicated even if she had ultrasound-proven DVT. Placement of an inferior vena cava filter (answer E) is used in patients with DVT who have an absolute contraindication to anticoagulation, such as recent surgery, head trauma, or active bleeding. They may also be used in patients who have pulmonary embolism in spite of adequate anticoagulation. Interestingly, although the concept of blocking a thrombus on its way to the lungs seems intuitive, evidence of long term benefit for IVC filters is still lacking: while patients with filters have fewer PEs in the first two weeks, there is no difference in PE or survival at two years versus patients who did not get filters.

B. Anticoagulation with heparin or enoxaparin (answer B) is the best immediate therapy for deep venous thrombosis. It prevents further propagation of the clot and reduces the incidence of pulmonary embolism.

C. Because of its oral availability, warfarin (answer C) is the treatment of choice for long-term anticoagulation for DVT prophylaxis. Though you may start it acutely if a patient presents with a DVT, it will not become therapeutic for several days.

D. Spiral CT (alternately called helical CT, CT angiography, or CT-PA, answer D) is used to diagnose pulmonary embolism, which is of course the most serious complication of deep venous thrombosis. This patient has superficial thrombophlebitis, however – not DVT. Moreover, she clinically lacks any symptoms of pulmonary embolism, so a CT would not be indicated even if she had ultrasound-proven DVT.

E. Placement of an inferior vena cava filter (answer E) is used in patients with DVT who have an absolute contraindication to anticoagulation, such as recent surgery, head trauma, or active bleeding. They may also be used in patients who have pulmonary embolism in spite of adequate anticoagulation. Interestingly, although the concept of blocking a thrombus on its way to the lungs seems intuitive, evidence of long term benefit for IVC filters is still lacking: while patients with filters have fewer PEs in the first two weeks, there is no difference in PE or survival at two years versus patients who did not get filters.

This question is not currently linked to the learning objective database.

Question problem?

Question # 60

Select the single best answer to the numbered question.

An otherwise healthy 26 year old female presents to her family physician with a rash on her neck. She just noticed it yesterday, but it is pruritic and painful. The patient has not been outdoors, and cannot think of anything in particular that could have caused the rash, other than perhaps using a new hairspray in preparation for her best friend's wedding several days ago. On physical exam, there is a papular and vesicular erythematous rash in a thin linear pattern that encircles her lower neck. The remainder of her physical exam is normal. What is the most appropriate initial step in the management of this patient?

A. Topical steroid cream
B. Oral prednisone
C. Reassurance
D. IgE testing
E. Oral antibiotics

You did not answer this question.

Explanations:

A. This is a classic case of contact dermatitis which can be induced by direct skin contact with an allergen or an irritant. The rash is usually intensely pruritic, and if allergic in nature, can occur up to two weeks after exposure to an offending allergen. The rash is usually papular and erythematous with indistinct margins, distributed along areas of exposure. The immune response causes fluid to collect in the epidermis, which can cause vesicles and oozing. The best treatment for mild or limited contact dermatitis is a medium potency topical steroid (answer A). Some additional teaching points… 1) When trying to determine the cause of a contact dermatitis, look at the distribution. (In this case, a rash encircling the neck might make you think about a necklace, and in fact nickel is found in some jewelry and is a common allergen.) Look at the clinical picture and use your common sense: scalp involvement might make you think about hair products, facial rashes could suggest cosmetics or aftershaves, while linear rashes on the extremities might be poison ivy/oak/sumac. If this patient's new hairspray were the culprit, you'd likely see some involvement on the scalp, not just the neck. Brainstorm with the patient about possible causes, because avoiding contact with the allergen or irritant will be crucial to preventing recurrence of the contact dermatitis. Patch testing is an option if you need help identifying the trigger. 2) When trying to determine the allergen of a contact dermatitis, remember that it might not be a new product or exposure that's causing the rash. A patient can become allergic to a component of a shampoo that he/she has been using for years! 3) Without evidence of secondary infection, a limited course of medium to high potency topical steroids is the best treatment for limited contact dermatitis. A course of systemic oral steroids and anti-histamines can be used to treat severe or extensive cases of contact dermatitis. Oral steroids such as prednisone (choice B) would probably be a bit too much at this point. A medium to high potency topical steroid cream should treat this limited and mild contact dermatitis without causing a great deal of systemic side effects. For patients with more diffuse involvement, you should definitely think about oral steroids. Reassurance (choice C) is not the best choice. Treatment will reduce symptoms and help the patient avoid secondary infection by scratching the rash. A discussion about the likely offender should also take place so that the patient can avoid future exposure. IgE testing (choice D) is sometimes ordered when a patient presents with atopic dermatitis or eczema, but even then is not necessary for diagnosis or treatment. Finding an elevated IgE level would not improve the patient's symptoms or help us figure out which allergen caused this patient's contact dermatitis.

B. Oral steroids such as prednisone (choice B) would probably be a bit too much at this point. A medium to high potency topical steroid cream should treat this limited and mild contact dermatitis without causing a great deal of systemic side effects. For patients with more diffuse involvement, you should definitely think about oral steroids. Although areas of contact dermatitis can become infected - especially by Staph - this patient gives us no indication that she has any need for antibiotic treatment (answer E).

C. Reassurance (choice C) is not the best choice. Treatment will reduce symptoms and help the patient avoid secondary infection by scratching the rash. A discussion about the likely offender should also take place so that the patient can avoid future exposure.

D. IgE testing (choice D) is sometimes ordered when a patient presents with atopic dermatitis or eczema, but even then is not necessary for diagnosis or treatment. Finding an elevated IgE level would not improve the patient's symptoms or help us figure out which allergen caused this patient's contact dermatitis.

E. Although areas of contact dermatitis can become infected - especially by Staph - this patient gives us no indication that she has any need for antibiotic treatment (answer E).

This question is not currently linked to the learning objective database.

Question problem?

Question # 61

Select the single best answer to the numbered question.

A 10 year old girl presents with several months of diarrhea and flatulence. Her parents report that despite the fact that she has been eating well, she has had a small weight loss during this period. She denies blood in her stool, but does report that her stools have been quite foul smelling and greasy. Physical examination reveals a pale, thin girl with blonde hair and blue eyes. Bowel sounds are slightly hyperactive, and there is no tenderness to palpation. Lungs sounds are normal. There is an erythematous, papulo-vesicular rash with signs of excoriation bilaterally at the knees and elbows. Laboratory evaluation shows serum anti-tissue transglutaminase (anti-TTG) antibodies are elevated. Which of the following is the most appropriate next step in the management of this patient?

A. Loperamide
B. Dietary avoidance of wheat, barley, and oats
C. Chloride sweat test
D. Rectal biopsy
E. Amoxicillin

You did not answer this question.

Explanations:

A. Loperamide (answer A) is an antimotility agent that can be used to symptomatically treat diarrhea. Using it here would might help the patient's symptoms, but would do nothing to affect her underlying disease process or malabsorption.

B. This patient presents with classic signs of malabsorptive diarrhea and an increased anti-TTG antibody. She has celiac disease, an autoimmune disorder triggered by exposure of the small bowel to gluten, a protein found in wheat, barley, rye, and oats. The best therapy is a gluten-free diet (answer B). Potato, tapioca, or soybean flours can be used in place of gluten-containing cereals.

to gluten, a protein found in wheat, barley, rye, and oats. The best therapy is a gluten free diet (answer B). Potato, tapioca, or soybean flours can be used in place of gluten containing cereals. Patients should be counseled to avoid dairy products initially because the damage to the small bowel may cause secondary lactose intolerance. A few additional teaching points… 1) There are a number of diagnostic tests that point toward celiac disease. Anti-gliadin antibodies, anti-tissue transglutaminase antibodies, and anti-endomysial antibodies are all used, with the anti-TTG antibodies having the greatest sensitivity and specificity. If needed, the diagnosis can be confirmed with an intestinal biopsy, which will show flattening and atrophy of the mucosal villi. 2) Many patients with celiac disease present as infants with malabsorption and failure to thrive, but an increasing number of patients do not present until later, with a significant number of diagnoses made between the ages of 10 and 40. 3) Celiac disease is an autoimmune disease, and so you should always keep in mind that autoimmune diseases tend to travel in groups. Patients have an increased risk of Type 1 diabetes, thyroid disease, and autoimmune hepatitis. In fact, this patient shows signs of one of the autoimmune diseases associated with celiac sprue; dermatitis herpetiformis, which was the rash on her knees and elbows. 4) Like any malabsorptive process, patients may develop deficiencies of the fat soluble vitamins A, D, E, and K, so USMLE question stems may lead you to the diagnosis of malabsorption by presenting the symptoms of these vitamin deficiencies. Loperamide (answer A) is an antimotility agent that can be used to symptomatically treat diarrhea. Using it here would might help the patient's symptoms, but would do nothing to affect her underlying disease process or malabsorption. The chloride sweat test (answer C) is a very sensitive test for cystic fibrosis when performed correctly. It is true that a large number of CF patients present with malabsorption (secondary to pancreatic insufficiency) rather than pulmonary complaints. However, in this case, the finding of elevated anti-TTG antibodies strongly points toward celiac disease as the cause of this patient's malabsorption. A rectal biopsy (answer D) is the test of choice for diagnosing Hirschsprung's disease, or aganglionic megacolon. The vast majority of patients with this disease present in infancy with failure to pass meconium or symptoms of distal intestinal obstruction (like bilious emesis, abdominal distension, or constipation). Antibiotic therapy with amoxicillin (answer E) is not indicated, as this patient has no infectious cause for her diarrhea. In general, antibiotics are seldom the answer for a patient with diarrhea (with the most obvious exception being a patient with C. difficile colitis). Actually, antibiotics are specifically contraindicated in patients with acute bloody diarrhea, because they may increase the risk of developing hemolytic uremic syndrome.

C. The chloride sweat test (answer C) is a very sensitive test for cystic fibrosis when performed correctly. It is true that a large number of CF patients present with malabsorption (secondary to pancreatic insufficiency) rather than pulmonary complaints. However, in this case, the finding of elevated anti-TTG antibodies strongly points toward celiac disease as the cause of this patient's malabsorption.

D. A rectal biopsy (answer D) is the test of choice for diagnosing Hirschsprung's disease, or aganglionic megacolon. The vast majority of patients with this disease present in infancy with failure to pass meconium or symptoms of distal intestinal obstruction (like bilious emesis, abdominal distension, or constipation).

E. Antibiotic therapy with amoxicillin (answer E) is not indicated, as this patient has no infectious cause for her diarrhea. In general, antibiotics are seldom the answer for a patient with diarrhea (with the most obvious exception being a patient with C. difficile colitis). Actually, antibiotics are specifically contraindicated in patients with acute bloody diarrhea, because they may increase the risk of developing hemolytic uremic syndrome.

This question is not currently linked to the learning objective database.

Question problem?

Question # 62

Select the single best answer to the numbered question.

A 53 year old female consults her physician due to progressive itching, yellowing of her skin, and fatigue. She denies any past history of jaundice and denies ever receiving a blood transfusion or abusing alcohol. Her past medical history includes hypertension and Sjogren's syndrome. Physical examination shows prominent hepatomegaly and scattered excoriations throughout the skin. Laboratory evaluation shows an elevated bilirubin, moderately elevated transaminases, and markedly elevated alkaline phosphatase with a normal albumin. Anti-mitochondrial antibodies are positive at >1:256. Which of the following is the most likely diagnosis for this patient?

A. Hepatitis C
B. Autoimmune hepatitis
C. Primary sclerosing cholangitis
D. Primary biliary cirrhosis
E. Pancreatic carcinoma

You did not answer this question.

Explanations:

A. Patients with chronic hepatitis C (answer A) have few reliable symptoms to distinguish themselves from other patients. If their disease has progressed enough, they will have the cardinal features of decompensated liver failure, including jaundice, hepatic encephalopathy, portal hypertension, etc.

B. Patients with autoimmune hepatitis (answer B) will have more striking elevations in AST and ALT than in alkaline phosphatase, reflecting the fact that the disease process is attacking hepatocytes rather than bile ducts. Remember, elevated alk. phos. localizes the pathology to the bile epithelium (or bone), while elevated transaminases indicate hepatocellular injury!

C. Primary sclerosing cholangitis (answer C) is an autoimmune disease that affects primary the larger extrahepatic bile ducts (instead of the smaller intrahepatic ducts as in PBC). There is a strong association between PSC and inflammatory bowel disease, especially ulcerative colitis – in the U.S., some 80% of patients with PSC will have coexisting IBD. (This is a VERY frequently tested fact on boards exams!) Patients may also have perinuclear anti-neutrophil cytoplasmic antibodies (p-ANCA).

D. Key teaching point: When you see anti-mitochondrial antibodies, think of primary biliary cirrhosis. This patient has primary biliary cirrhosis (answer D), and as stated above, the key finding is the high titer of antimitochondrial antibody. This is virtually pathognomonic for primary biliary cirrhosis (PBC) and distinguishes it from the other answer choices, any of which could have caused the patient's presenting complaints. A few additional teaching points about PBC… 1) This patient is a classic presentation of PBC. It is a disease that has a strong predilection for affecting females (by 6:1), most commonly in their late 40s-early 60s. It is frequently associated with other autoimmune diseases, most frequently rheumatoid arthritis and Sjogren's syndrome. Pruritus is the most frequent presenting complaint, but patients may also have jaundice, fatigue, or weight loss. 2) The ultimate treatment for primary biliary cirrhosis is a liver transplant. Treatment with immunomodulators may slow the progression of the disease, and cholestyramine can help with pruritis (since it binds bile salts). Patients with chronic hepatitis C (answer A) have few reliable symptoms to distinguish themselves from other patients. If their disease has progressed enough, they will have the cardinal features of decompensated liver failure, including jaundice, hepatic encephalopathy, portal hypertension, etc. Patients with autoimmune hepatitis (answer B) will have more striking elevations in AST and ALT than in alkaline phosphatase, reflecting the fact that the disease process is attacking hepatocytes rather than bile ducts. Remember, elevated alk. phos. localizes the pathology to the bile epithelium (or bone), while elevated transaminases indicate hepatocellular injury! Primary sclerosing cholangitis (answer C) is an autoimmune disease that affects primary the larger extrahepatic bile ducts (instead of the smaller intrahepatic ducts as in PBC). There is a strong association between PSC and inflammatory bowel disease, especially ulcerative colitis – in the U.S., some 80% of patients with PSC will have coexisting IBD. (This is a VERY frequently tested fact on boards exams!) Patients may also have perinuclear anti-neutrophil cytoplasmic antibodies (p-ANCA). If a pancreatic carcinoma (answer E) begins in the head of the pancreas, it can cause obstructive jaundice. Cancers that begin in the tail of the pancreas more often present with pain and weight loss. Making the diagnosis requires an imaging study, most often CT or ERCP, though ultrasound or MRI can be used as well.

E. If a pancreatic carcinoma (answer E) begins in the head of the pancreas, it can cause obstructive jaundice. Cancers that begin in the tail of the pancreas more often present with pain and weight loss. Making the diagnosis requires an imaging study, most often CT or ERCP, though ultrasound or MRI can be used as well.

This question is not currently linked to the learning objective database.

Question problem?

Question # 63

Select the single best answer to the numbered question.

A 23 year old student has the acute onset of dizziness and nausea and vomiting. He feels like the room is rotating around him to the left. Six days ago, he had a low-grade fever, myalgias, and a sore throat, which resolved yesterday. He denies a history of similar episodes, and his past medical history is entirely benign. Physical examination reveals mild hearing loss on the left and horizontal nystagmus that resolves when the patient looks at a fixed object. Tympanic membranes are clear. Though the patient is able to ambulate, he shows marked gait instability. There is a positive head thrust test. There is no dysarthria, motor weakness, sensory loss, facial droop, or limb dysmetria observed. Which of the following is the most likely diagnosis in this patient?

A. Labyrinthitis
B. Cerebellar infarction
C. Benign paroxysmal positional vertigo
D. Multiple sclerosis
E. Suppurative otitis media

You did not answer this question.

Explanations:

A. This patient presents with a single, sudden episode of vertigo following a viral illness. This history, accompanied by the physical examination findings of peripheral nystagmus, hearing loss, and an abnormal head thrust test, strongly point to a diagnosis of acute labyrinthitis (answer A). A few additional teaching points from this case… 1) Labyrinthitis, also known as vestibular neuritis or vestibular neuronitis, is thought to be caused by postviral inflammation of the vestibular portion of CN VIII. It is a benign, self-limited disorder that completely resolves in most patients after several days – but until it resolves, patients can be quite miserable. Treatment with corticosteroids may shorten the duration of the attack by reducing inflammation, and antiemetics, antihistamines, and benzodiazepines may ameliorate symptoms. 2) An important clue to the diagnosis here is the patient's nystagmus, which has features indicating that it is caused by a peripheral lesion rather than a central one. In general, peripheral nystagmus is inhibited by visual fixation, while central nystagmus is not (i.e., if you ask the patient to focus on a fixed point, the nysgagmus will disappear). Also, finding other signs of peripheral nerve dysfunction like tinnitus or deafness strongly suggests a peripheral lesion. 3) This patient has a positive head thrust test, which is a maneuver that few physicians other than neurologists regularly perform – but when it is mentioned on a shelf exam, it has a high positive predictive value for the correct answer being "vestibular neuritis." To do the maneuver, you rapidly turn the patient's head toward the side of the lesion. If the patient is unable to maintain visual fixation, the test is positive. A cerebellar infarction (answer B) can cause acute vertigo, but is not the most likely diagnosis here. This patient is young and has no vascular risk factors, so a stroke is unlikely. Also, patients with cerebellar infarction typically are unable to ambulate unassisted, and should have a normal head thrust test. If the diagnosis is unclear, you should get neuroimaging studies. (Remember that if you're concerned about lesions in the posterior fossa, MRI/MRA is the test of choice, not CT.) Benign paroxysmal positional vertigo (answer C) causes recurrent episodes of vertigo that last a few seconds – not a few hours, like this patient's symptoms. They are usually precipitated by specific head movements, and are thought to be caused by the movement of small calcium stones (otoliths) inside the vestibule. The tabletop Dix-Hallpike maneuver will re-create symptoms and confirm the diagnosis if it is in doubt. The diagnosis of multiple sclerosis (answer D) requires neurological findings separated in time that cannot be explained by a single lesion. Acute or chronic otitis media (answer E) may rarely cause labyrinthitis if there is accompanying inflammation of CN VIII. Here, there are no historical or physical examination findings to point you in that direction.

B. A cerebellar infarction (answer B) can cause acute vertigo, but is not the most likely diagnosis here. This patient is young and has no vascular risk factors, so a stroke is unlikely. Also, patients with cerebellar infarction typically are unable to ambulate unassisted, and should have a normal head thrust test. If the diagnosis is unclear, you should get neuroimaging studies. (Remember that if you're concerned about lesions in the posterior fossa, MRI/MRA is the test of choice, not CT.)

C. Benign paroxysmal positional vertigo (answer C) causes recurrent episodes of vertigo that last a few seconds – not a few hours, like this patient's symptoms. They are usually precipitated by specific head movements, and are thought to be caused by the movement of small calcium stones (otoliths) inside the vestibule. The tabletop Dix-Hallpike maneuver will re-create symptoms and confirm the diagnosis if it is in doubt.

D. The diagnosis of multiple sclerosis (answer D) requires neurological findings separated in time that cannot be explained by a single lesion.

E. Acute or chronic otitis media (answer E) may rarely cause labyrinthitis if there is accompanying inflammation of CN VIII. Here, there are no historical or physical examination findings to point you in that direction.

This question is not currently linked to the learning objective database.

Question problem?

Question # 64

Select the single best answer to the numbered question.

An 18 month old male presents for a well-child examination. While observing the child playing in his mother's lap, it appears that the child has a wandering right eye. The patient's mother denies any family history of visual disorders or cancer, and says that the child has appeared completely healthy lately. On physical examination, there is no tenderness to mild pressure on either eye. Sclera are non-injected. A white pupillary reflex is present on the right. With the child visually fixed on a toy, each of his eyes are separately covered and then uncovered. There is no eye movement when the patient's right eye is covered and rapidly uncovered, but the patient's right eye refixates on the toy when the left eye is covered and then uncovered. What is the most appropriate next step in the management of this patient?

A. MRI of head
B. Occlusion of left eye with patch
C. Occlusion of right eye with patch
D. Atropine eye drops
E. Reassurance

You did not answer this question.

Explanations:

A. This child presents with leukocoria, which is the hallmark physical finding of retinoblastoma. This is a very serious and life-threatening disease that requires immediate diagnosis with CT or MRI (answer A) and ophthalmologic referral. Ultimately, if the patient has retinoblastoma, he will require urgent surgery, since the tumor tends to metastasize early. A few teaching points about this case… 1) The key finding on physical examination was that of a white pupillary reflex, or leukocoria. This is always an abnormal finding that demands explanation and workup. Light normally transmits easily through the eye, and any anatomical opacity will cause a dulling of the normal red reflex to a grey or white color. Thus, disorders of the lens (like cataracts) or vitreous (like hemorrhage) can cause leukocoria – but it is also the most frequent presenting physical finding for retinal disorders like retinoblastoma, and these must be ruled out first. 2) A commonly-tested fact about retinoblastoma is its heritability: 40% of patients have mutations to both RB tumor suppressor genes. These patients may have a family history of retinoblastoma, and they are also at risk for developing bilateral disease. However, the majority of patients with retinoblastoma – 60% - have sporadic mutations that cause their illness. 3) The cover-uncover test that was described in the question stem is used to test for strabismus. In a normal patient, there should be no movement of the eyes when either one is covered and then uncovered. If the patient's eyes are misaligned, however, the non-dominant eye will jump and re-fixate once the dominant eye is uncovered. Here, the description of the test and its findings mainly serves as a distractor to try to slip the finding of leukocoria past you, but this patient does have an abnormal test, likely due to the fact that retinoblastoma has impaired his vision enough on the right that his left eye has become dominant. Occlusion therapy (answer B and C) is commonly used to treat patients with amblyopia, or decreased vision in an anatomically normal eye. The eye with better vision should be patched and occluded, forcing the patient to use the amblyopic eye. Over time, the vision in that eye will improve, and the patient will be able to use both eyes normally. If this patient had ambylopia, he should have the left eye patched (answer B), so that his wandering right eye would become stronger. Patching the right eye (answer C) would only make the problem worse. However, this patient's eye is clearly not anatomically normal – a white pupillary reflex can indicate a number of causes for this patient's decreased vision, and those should be worked up first. Atropine eye drops (answer D) can be used to accomplish "pharmacological blurring" to treat amblyopia. Administration of atropine into the eye with better vision causes pupillary dilation and loss of accommodation, forcing the eye with worse vision to work harder. It's the same principle as occlusion therapy, but doesn't force a child to wear a patch (which can be difficult to do!). Again, however, this patient does not have amblyopia, so this is an inappropriate intervention. Reassurance (answer E) is inappropriate. Strabismus is normal in early development, but not in an 18 month old. And leukocoria is never normal. This patient needs workup.

B. Occlusion therapy (answer B and C) is commonly used to treat patients with amblyopia, or decreased vision in an anatomically normal eye. The eye with better vision should be patched and occluded, forcing the patient to use the amblyopic eye. Over time, the vision in that eye will improve, and the patient will be able to use both eyes normally. If this patient had ambylopia, he should have the left eye patched (answer B), so that his wandering right eye would become stronger. Patching the right eye (answer C) would only make the problem worse. However, this patient's eye is clearly not anatomically normal – a white pupillary reflex can indicate a number of causes for this patient's decreased vision, and those should be worked up first.

C. Occlusion therapy (answer B and C) is commonly used to treat patients with amblyopia, or decreased vision in an anatomically normal eye. The eye with better vision should be patched and occluded, forcing the patient to use the amblyopic eye. Over time, the vision in that eye will improve, and the patient will be able to use both eyes normally. If this patient had ambylopia, he should have the left eye patched (answer B), so that his wandering right eye would become stronger. Patching the right eye (answer C) would only make the problem worse. However, this patient's eye is clearly not anatomically normal – a white pupillary reflex can indicate a number of causes for this patient's decreased vision, and those should be worked up first.

D. Atropine eye drops (answer D) can be used to accomplish "pharmacological blurring" to treat amblyopia. Administration of atropine into the eye with better vision causes pupillary dilation and loss of accommodation, forcing the eye with worse vision to work harder. It's the same principle as occlusion therapy, but doesn't force a child to wear a patch (which can be difficult to do!). Again, however, this patient does not have amblyopia, so this is an inappropriate intervention.

E. Reassurance (answer E) is inappropriate. Strabismus is normal in early development, but not in an 18 month old. And leukocoria is never normal. This patient needs workup.

This question is not currently linked to the learning objective database.

Question problem?

Question # 65

Select the single best answer to the numbered question.

An otherwise healthy 15 year old male comes to his physician with the complaint of facial acne. He has tried multiple over-the-counter acne washes without improvement, and he and his mother are hopeful that a medication will help. The patient's diet is acceptable but does include large amounts of fast food. He is under a moderate amount of stress juggling school and his competitive traveling soccer team, but both he and his mom feel that he is handling this well. On physical exam, the patient's face has scattered open and closed comedones and a few small papules. No scarring is noted, and his back and upper arms are not affected. What is the most appropriate initial step in the management of this patient?

A. Isotretinoin
B. Topical retinoid
C. Oral erythromycin
D. Reassurance
E. Avoidance of greasy foods

You did not answer this question.

Explanations:

A. Oral isotretinoin (answer A) is the treatment for severe acne vulgaris and is not yet indicated in this patient. Also known as 13-cis retinoic acid, it is the only drug (besides hormones) that actually reduces sebum secretion; this effect lasts up to one year after stopping therapy. Remember that this drug is teratogenic, can elevate triglycerides, can be hepatotoxic, and there also may be an association with depression and suicide. Due to the adverse effects and dangers of this medication, it is reserved for the most severe cases of acne that don't respond to first-line treatments.

B. The key to treating acne is recognizing the severity of the patient's case, ranging from the non-inflammatory comedonal acne (like this patient has) to severe inflammatory acne with large amounts of scarring. For mild comedonal acne, the best treatment is a topical retinoid and/or other topical agents such as salicylic acid, azelaic acid, glycolic acid, and benzoyl peroxide, so out of the choices listed above, a topical retinoid (answer B) is the best option at this point. After three months, if the response is inadequate, treatment can move on to a combination of the above and adding a topical antibiotic. A couple of additional teaching points… 1) Comedones take 8 weeks to mature, so any therapy must be tried for at least this long before efficacy can be determined. 2) Topical retinoids (tretinoin, adapalene) affect terminal differentiation of the follicular epithelium, thus helping to normalize follicular keratinization and prevent the formation of new comedones. 3) Benzoyl peroxide has antimicrobial and comedolytic properties and can be used for non-inflammatory comedonal acne or for more severe inflammatory acne. When combined with a topical antibiotic (like clindamycin) it can help prevent resistant strains of P. acne from emerging. 4) Topical antibiotics are used to eliminate P. acnes from follicles and help to suppress inflammation that occurs with inflammatory acne. 5) Oral antibiotics (tetracycline, doxycycline, minocycline, erythromycin, bactrim & clindamycin) are used for inflammatory acne to prevent the growth of P. acnes in the pilosebaceous unit. Oral isotretinoin (answer A) is the treatment for severe acne vulgaris and is not yet indicated in this patient. Also known as 13-cis retinoic acid, it is the only drug (besides hormones) that actually reduces sebum secretion; this effect lasts up to one year after stopping therapy. Remember that this drug is teratogenic, can elevate triglycerides, can be hepatotoxic, and there also may be an association with depression and suicide. Due to the adverse effects and dangers of this medication, it is reserved for the most severe cases of acne that don't respond to first-line treatments. Oral erythromycin (answer C) is an antibiotic that can be used to treat inflammatory acne, but there is no need to start with an oral medicine at this point since most simple comedonal acne isn't colonized with P. acne. This would only be used if the patient's intial presentation showed more of an inflammatory picture - papules and pustules with mild scarring – or if the initial treatments for comedonal acne had failed after a 3 month trial. Reassurance (answer D) is not the best choice at this point because the patient is concerned and bothered by his acne and he has already tried the over-the-counter treatment options available to him. His physical exam shows comedonal acne and there are several relatively safe treatment options available to him. Avoiding greasy foods (answer E) is not the best answer because a dietary association with acne has not been proven. This has been a controversial topic, but so far the only known association is in a small group of patients with the intake of milk (possibly because it contains natural hormonal components and other bioactive chemicals).

C. Oral erythromycin (answer C) is an antibiotic that can be used to treat inflammatory acne, but there is no need to start with an oral medicine at this point since most simple comedonal acne isn't colonized with P. acne. This would only be used if the patient's intial presentation showed more of an inflammatory picture - papules and pustules with mild scarring – or if the initial treatments for comedonal acne had failed after a 3 month trial.

D. Reassurance (answer D) is not the best choice at this point because the patient is concerned and bothered by his acne and he has already tried the over-the-counter treatment options available to him. His physical exam shows comedonal acne and there are several relatively safe treatment options available to him.

E. Avoiding greasy foods (answer E) is not the best answer because a dietary association with acne has not been proven. This has been a controversial topic, but so far the only known association is in a small group of patients with the intake of milk (possibly because it contains natural hormonal components and other bioactive chemicals).

This question is not currently linked to the learning objective database.

Question problem?

Question # 66

Select the single best answer to the numbered question.

A 19 year old female comes to her physician because of visual blurring for the past week. For the past few months, she has also has experienced daily headaches, which are worst in the morning upon awakening and improve throughout the day or after taking ibuprofen. Her past medical history is unremarkable, and her only medication is a combination oral contraceptive pill. Physical examination shows height 62 inches (157 cm), weight 206 lbs. (93 kg), temperature 37.2 C (99.0 F), pulse 72, blood pressure 118/76. Fundoscopic examination shows bilateral papilledema. Neurological examination shows no focal deficits. CT shows normal ventricles and no evidence for mass lesion, but increased volume and distension of the perioptic subarachnoid space. Lumbar puncture shows an elevated opening pressure and clear CSF with normal cell counts, protein, and glucose. Which of the following is the most likely diagnosis in this patient?

A. Aseptic meningitis
B. Craniopharyngioma
C. Dandy-Walker malformation
D. Subdural hemorrhage
E. Pseudotumor cerebri

You did not answer this question.

Explanations:

A. Aseptic meningitis (answer A) occurs when a patient has clinical signs and laboratory evidence of meningeal irritation, but negative bacterial cultures. The most common cause is viral meningitis caused by enteroviruses. This patient lacks clinical signs of meningeal irritation. Additionally, her LP is normal except for an increased pressure, while a patient with viral meningitis would be expected to have increased WBC (especially lymphocytes), slightly elevated protein, and slightly reduced glucose.

B. Craniopharyngiomas (answer B) are tumors that arise from the remnants of Rathke's pouch in the region of the pituitary. They are the third most common intracranial tumors affecting children and adolescents (behind , and generally present with pituitary insufficiency or the absolutely classic sign of a pituitary mass, bitemporal hemianopsia.

C. A Dandy-Walker malformation (answer C) is an anomaly of the fourth ventricle and cerebellum that occurs during fetal development. Although it can be diagnosed with fetal ultrasound, most patients are diagnosed via CT or MRI in infancy or childhood after presenting with slow motor development, progressive enlargement of the skull, or signs of cerebellar dysfunction or increased intracranial pressure.

D. This patient's history is not consistent with subdural hemorrhage (answer D), and in any case, this problem should show up on CT.

E. This is pseudotumor cerebri (answer E), also known as idiopathic intracranial hypertension. Its cause is unknown, but patients – classically obese, young females taking oral contraceptives – develop increased intracranial pressure and papilledema. If untreated, the papilledema can cause optic atrophy and blindness. (This is why the use of another, older name for the disease, "benign intracranial hypertension," is now discouraged.) Medical treatment with acetazolamide may help reduce the ICP in a patient without visual loss, but patients with evidence of optic nerve damage should be referred to a neurosurgeon or ophthalmologist for surgery. As an additional general teaching point, headaches that are worse in the morning or that are accompanied by nausea and vomiting should ring a bell for the headache of increased intracranial pressure. Aseptic meningitis (answer A) occurs when a patient has clinical signs and laboratory evidence of meningeal irritation, but negative bacterial cultures. The most common cause is viral meningitis caused by enteroviruses. This patient lacks clinical signs of meningeal irritation. Additionally, her LP is normal except for an increased pressure, while a patient with viral meningitis would be expected to have increased WBC (especially lymphocytes), slightly elevated protein, and slightly reduced glucose. Craniopharyngiomas (answer B) are tumors that arise from the remnants of Rathke's pouch in the region of the pituitary. They are the third most common intracranial tumors affecting children and adolescents (behind , and generally present with pituitary insufficiency or the absolutely classic sign of a pituitary mass, bitemporal hemianopsia. A Dandy-Walker malformation (answer C) is an anomaly of the fourth ventricle and cerebellum that occurs during fetal development. Although it can be diagnosed with fetal ultrasound, most patients are diagnosed via CT or MRI in infancy or childhood after presenting with slow motor development, progressive enlargement of the skull, or signs of cerebellar dysfunction or increased intracranial pressure. This patient's history is not consistent with subdural hemorrhage (answer D), and in any case, this problem should show up on CT.

This question is not currently linked to the learning objective database.

Question problem?

Question # 67

Select the single best answer to the numbered question.

A 17 year old basketball player comes to the physican after "twisting" his knee in a game one week ago. After jumping to catch a pass, he twisted on his flexed right knee and felt a sharp pain. He denies any joint dislocation or popping sounds or sensations, but was scarcely able to bear weight after the injury and had to be helped off the court. The following morning, his knee was swollen, and although his pain has improved, he has had several episodes where his knee "locks" into an extended position. On physical examination, there is a moderate effusion of the right knee and tenderness to palpation along the medial joint line. Extending the fully-flexed knee while applying a moderate varus stress causes an audible click and reproduces the patient's pain. With the knee flexed to ninety degrees, there is no movement of the tibia on the femur when the lower leg is pulled anteriorly. Which of the following is the most likely diagnosis in this patient?

A. Osgood-Schlatter disease
B. Torn anterior cruciate ligament
C. Iliotibial band syndrome
D. Slipped capital femoral epiphysis
E. Meniscal injury

You did not answer this question.

Explanations:

A. Osgood-Schlatter disease (answer A) is a commonly-tested cause of knee pain in an active adolescent who has just experienced a growth spurt. Although the exact cause remains controversial, symptoms are likely due to an avulsion of the hyaline cartilage overlying the tibial tubercle near the insertion of the patellar tendon. Patients complain of chronic and insidious anterior knee pain that may be worse with jumping, and may show prominence of the tibial tuberosity on physical exam. The condition is benign and self-limiting, and patients should improve within six to 18 months.

B. A torn anterior cruciate ligament (answer B) is on the differential diagnosis given the mechanism of injury that the patient describes. (In fact, because they are caused by similar mechanisms of injury, ACL injuries are often accompanied by damage to the medial collateral ligament (MCL) and medial meniscus, leading to the so-called "unhappy triad" injury.) Patients with torn ACLs commonly describe hearing or feeling a pop at the time of injury, and usually have impaired ability to bear weight. Swelling is common and dramatic due to hemarthrosis. However, the classic physical exam finding is a positive anterior drawer test, which is described as being absent in the question stem. A positive Lachman test (similar to the anterior drawer test, but with the knee flexed only to 20 degrees instead of 90 degrees) is also highly suggestive of ACL injury.

C. Iliotibial band syndrome (answer C) is another cause of chronic knee pain. It is seen almost exclusively in runners. Patients complain of lateral knee pain that may radiate up the thigh and toward the hip.

D. Slipped capital femoral epiphyses (answer D) is a common disorder of the hip that often presents with isolated thigh or knee pain and altered gait. The classic patient is an overweight child in early adolescence who has not yet completed puberty. The diagnosis is made with hip radiographs, and it is important to get these early, because delayed diagnosis can lead to osteonecrosis of the femoral head.

E. This patient's mechanism of injury, history, and physical exam findings are all consistent with a meniscal injury (answer E). These injuries tend to occur when twisting a flexed leg while bearing weight, especially when landing from a jump or emerging from a squatting position. If the injury is acute, patients usually have difficulty weight bearing immediately after the injury and experience swelling within 24 hours. However, since only the periphery of the meniscus contains sensory nerve fibers, patients may have little pain but prominent mechanical symptoms like knee locking, weakness, or giving way. On physical exam, the patient should have joint line tenderness and pain or clicking on the McMurray test (described in the question stem). Osgood-Schlatter disease (answer A) is a commonly-tested cause of knee pain in an active adolescent who has just experienced a growth spurt. Although the exact cause remains controversial, symptoms are likely due to an avulsion of the hyaline cartilage overlying the tibial tubercle near the insertion of the patellar tendon. Patients complain of chronic and insidious anterior knee pain that may be worse with jumping, and may show prominence of the tibial tuberosity on physical exam. The condition is benign and self-limiting, and patients should improve within six to 18 months. A torn anterior cruciate ligament (answer B) is on the differential diagnosis given the mechanism of injury that the patient describes. (In fact, because they are caused by similar mechanisms of injury, ACL injuries are often accompanied by damage to the medial collateral ligament (MCL) and medial meniscus, leading to the so-called "unhappy triad" injury.) Patients with torn ACLs commonly describe hearing or feeling a pop at the time of injury, and usually have impaired ability to bear weight. Swelling is common and dramatic due to hemarthrosis. However, the classic physical exam finding is a positive anterior drawer test, which is described as being absent in the question stem. A positive Lachman test (similar to the anterior drawer test, but with the knee flexed only to 20 degrees instead of 90 degrees) is also highly suggestive of ACL injury. Iliotibial band syndrome (answer C) is another cause of chronic knee pain. It is seen almost exclusively in runners. Patients complain of lateral knee pain that may radiate up the thigh and toward the hip. Slipped capital femoral epiphyses (answer D) is a common disorder of the hip that often presents with isolated thigh or knee pain and altered gait. The classic patient is an overweight child in early adolescence who has not yet completed puberty. The diagnosis is made with hip radiographs, and it is important to get these early, because delayed diagnosis can lead to osteonecrosis of the femoral head.

This question is not currently linked to the learning objective database.

Question problem?

Question # 68

Select the single best answer to the numbered question.

A 31 year old female presents with several weeks of progressive fatigue, weakness, and weight loss. She has also had occasional nausea and diarrhea. Past medical history includes juvenile diabetes and hypothyroidism. On physical examination, the patient has areas of bronzed hyperpigmentation on her face, neck, and the dorsum of her hands. The remainder of physical exam is within normal limits. Laboratory examination is significant for a sodium of 130 mEq/L and a glucose of 65 mg/dL. Which of the following is the most appropriate next step in the diagnosis of this patient?

A. Thyroid ultrasound
B. Measurement of insulin-like growth factor I (IGF-1)
C. Measurement of 21-hydroxylase activity
D. Cosyntropin stimulation test
E. Dexamethasone suppression test

You did not answer this question.

Explanations:

A. Thyroid radionuclide scan (answer A) is a test that is used to determine "hot" versus "cold" thyroid nodules, and might be the next step in diagnosis (along with thyroid function tests) for a patient who presents with a thyroid mass. Malignant nodules are more likely to be hypofunctioning or "cold," while a hyperfunctioning thyroid adenoma would show up as a "hot" nodule.

B. Measurement of insulin-like growth factor I or IGF-1 (answer B) is used to screen for acromegaly, which is caused by the overproduction of growth hormone. Patients have coarsened facial features, enlargement of the hands, skull, and feet, and may complain of bitemporal hemianopsia from the presence of a pituitary adenoma.

C. Finding low 21-hydroxylase activity (answer C) would confirm the most common cause for congenital adrenal hyperplasia (CAH). This condition occurs in newborns who have ambiguous genitalia and hypotension. Although the vast majority of patients present in infancy, it is possible for the diagnosis to be missed in milder cases, leading to a diagnosis in childhood following precocious puberty.

D. This patient has primary adrenal insufficiency or Addison's disease. Key features to diagnosis include fatigue, weight loss, hypotension, hyponatremia, and hypoglycemia. To confirm the diagnosis, stimulation with synthetic ACTH or cosyntropin (answer D) should be performed, along with a measurement of plasma cortisol. If the adrenals are functioning, the cortisol should rise with administration of cosyntropin. Some additional teaching points… 1) There are two types of adrenal insufficiency: primary (in which the primary problem is the adrenals themselves) and secondary (in which the adrenal hypofunction occurs secondary to another problem elsewhere). Primary adrenal insufficiency, or Addison's disease, is caused by the autoimmune destruction of the adrenal glands. It tends to occur in patients who have a history of other autoimmune disorders or symptoms (like hypothyroidism, type 1 diabetes, vitiligo, pernicious anemia, etc.). The most common cause of secondary adrenal insufficiency (and the most common cause of adrenal insufficiency overall) is iatrogenic, caused by the too-abrupt cessation of chronic steroid treatment. In this case, the pituitary's production of ACTH becomes suppressed by the chronic presence of steroids, and endogenous production of steroids is unable to keep up with demands once the exogenous steroids are stopped. 2) This patient's hyperpigmentation is a classic sign of Addison's disease. It is caused by excessive ACTH, which stimulates melanocytes. This finding actually helps distinguish between primary and secondary adrenal insufficiency, as well. In a patient who has abruptly stopped a steroid taper, for example, the pituitary has been suppressed by the presence of chronic exogenous glucocorticoids. Thus, ACTH is low, and there should be no hyperpigmentation. Patients whose adrenals are destroyed by an autoimmune process (i.e., primary adrenal insufficiency) have a hyperfunctioning pituitary that is producing lots of ACTH to try to flog the adrenals into making some glucocorticoids, and this excess ACTH stimulates melanocytes as well. 3) Besides differences in ACTH levels, there is one other important difference between primary and secondary adrenal insufficiency. In primary adrenal insufficiency, the immune system destroys the entire adrenal gland, leading to deficiencies of both glucocorticoids and mineralocorticoids (like aldosterone). As already discussed, the primary defect in secondary adrenal insufficiency is a low ACTH – the adrenals themselves are fine, so mineralocorticoid production continues unabated. Clinically, this shows up as 4) In its extreme form (Addisonian crisis) adrenal insufficiency can present with an acute abdomen, complete with peritoneal signs like rebound tenderness and guarding. This fact tends to come up on the surgery shelf as one of the very few "medical" causes of a surgical abdomen. (You'll get a description of a patient with a history of chronic steroid use who presents with an acute abdomen and then has a negative exploratory laparotomy.) 5) The other absolutely classic way that adrenal insufficiency tends to show up on the USMLE is in the context of Waterhouse-Friderichsen syndrome. This occurs in meningococcal sepsis when N.meningitides causes bilateral hemorrhagic destruction of the adrenal glands. Thyroid radionuclide scan (answer A) is a test that is used to determine "hot" versus "cold" thyroid nodules, and might be the next step in diagnosis (along with thyroid function tests) for a patient who presents with a thyroid mass. Malignant nodules are more likely to be hypofunctioning or "cold," while a hyperfunctioning thyroid adenoma would show up as a "hot" nodule. Measurement of insulin-like growth factor I or IGF-1 (answer B) is used to screen for acromegaly, which is caused by the overproduction of growth hormone. Patients have coarsened facial features, enlargement of the hands, skull, and feet, and may complain of bitemporal hemianopsia from the presence of a pituitary adenoma. Finding low 21-hydroxylase activity (answer C) would confirm the most common cause for congenital adrenal hyperplasia (CAH). This condition occurs in newborns who have ambiguous genitalia and hypotension. Although the vast majority of patients present in infancy, it is possible for the diagnosis to be missed in milder cases, leading to a diagnosis in childhood following precocious puberty. The dexamethasone suppression test (answer E) is used in the workup of Cushing's syndrome, or excess glucocorticoids. In general, high dose dexamethasone should suppress the cortisol production from a pituitary adenoma, but not the cortisol from an adrenal tumor or the ectopic production from a malignancy elsewhere.

E. The dexamethasone suppression test (answer E) is used in the workup of Cushing's syndrome, or excess glucocorticoids. In general, high dose dexamethasone should suppress the cortisol production from a pituitary adenoma, but not the cortisol from an adrenal tumor or the ectopic production from a malignancy elsewhere.

This question is not currently linked to the learning objective database.

Question problem?

Question # 69

Select the single best answer to the numbered question.

A six-month old female is brought to the clinic in December with wheezing and respiratory difficulty for the past several hours. She did have a low-grade fever two days ago accompanied by rhinorrhea, but has otherwise been well since birth. Vital signs are temperature 38.6 C (101.5 F), pulse 160, blood pressure 87/59, respirations 60/min. Nasal flaring and intercostal retractions are noted. Auscultation of the lungs reveals faint inspiratory crackles at the lung bases and diffuse expiratory wheezing. A chest x-ray shows a normal cardiac silhouette, hyperexpanded lungs, and peribronchial thickening. Which of the following tests would be most likely to confirm the diagnosis in this patient?

A. Rigid bronchoscopy
B. Echocardiogram
C. Methacholine challenge test
D. Rapid antigen test for respiratory syncytial virus
E. Sweat chloride test

You did not answer this question.

Explanations:

A. Rigid bronchoscopy (answer A) is used to remove aspirated foreign bodies in children. You should always consider this possibility in a child that presents with respiratory distress, regardless of whether there is a history of choking or putting objects in the mouth. The peak incidence of aspirated foreign bodies occurs in children between one and two years of age – the exact age when children have the physical skills (like walking and a better pincer grasp) and to start exploring their environment and putting things in their mouths! Younger children tend to aspirate food items (especially peanuts) because they lack the dentition to chew food adequately; older children tend to aspirate non-food items like coins.

B. An echocardiogram (answer B) would be helpful in identifying a cardiac cause of the patient's dyspnea. There are a variety of congenital lesions that can lead to heart failure or respiratory distress, but there is nothing in the question stem to lead you to suspect any of them immediately.

C. Methacholine challenge testing (answer C) is occasionally used to make or rule out a diagnosis of asthma. When a patient prone to bronchospasm breathes in methacholine (or histamine or cold air), there should be a significant decrease in their FEV1. Because performing the test requires a special facility and entails some risk, it is seldom done, but may be used to evaluate a patient with symptoms of asthma but normal spirometry.

D. This child has bronchiolitis, a viral lower respiratory tract infection most often caused by respiratory syncytial virus (answer D). A few teaching points about this case… 1) Epidemiologically, bronchiolitis affects younger children – especially under two years old – with the vast majority of all cases occurring in the winter. (If you've been on the pediatrics wards in the winter, this will come as no surprise to you.) Over three-fourths of all cases are caused by respiratory syncytial virus (RSV), while most of the rest of the cases are caused by parainfluenza and adenoviruses. 2) A common presenting symptom – especially in infants younger than 6 weeks old – is apnea, so when you see it, think of RSV infection! 3) Remember that wheezing on physical exam localizes the lesion to the level of the bronchioles. This is the site of bronchoconstriction in patients with asthma, and is also the area of the bronchial tree targeted by RSV – so it makes sense that both patients with asthma and patients with RSV will present with wheezing. 4) Treatment of RSV infection is largely symptomatic, with humidified oxygen, bronchodilators, and corticosteroids being the mainstays of treatment. Antiviral therapy with ribavirin may be used in children with severe disease. Immunization with palivizumab (Synagis), a monoclonal antibody against RSV, reduces the risk of hospitalization for bronchiolitis, and is given to premature infants or infants with chronic lung or heart disease. 5) Vital signs in children can be difficult to interpret, since the normal values change with age. A helpful chart can be found at http://www.emedicinehealth.com/pediatric_vital_signs/article_em.htm Rigid bronchoscopy (answer A) is used to remove aspirated foreign bodies in children. You should always consider this possibility in a child that presents with respiratory distress, regardless of whether there is a history of choking or putting objects in the mouth. The peak incidence of aspirated foreign bodies occurs in children between one and two years of age – the exact age when children have the physical skills (like walking and a better pincer grasp) and to start exploring their environment and putting things in their mouths! Younger children tend to aspirate food items (especially peanuts) because they lack the dentition to chew food adequately; older children tend to aspirate non-food items like coins. An echocardiogram (answer B) would be helpful in identifying a cardiac cause of the patient's dyspnea. There are a variety of congenital lesions that can lead to heart failure or respiratory distress, but there is nothing in the question stem to lead you to suspect any of them immediately. Methacholine challenge testing (answer C) is occasionally used to make or rule out a diagnosis of asthma. When a patient prone to bronchospasm breathes in methacholine (or histamine or cold air), there should be a significant decrease in their FEV1. Because performing the test requires a special facility and entails some risk, it is seldom done, but may be used to evaluate a patient with symptoms of asthma but normal spirometry. The sweat chloride test (answer E) is used to diagnose patients with cystic fibrosis, which should always be on the differential in any child with chronic lung problems (or diarrhea or failure to thrive). Because CF is such a serious disease, you should have a low threshold for ordering this test – but one episode of wheezing is not enough to make this the next step.

E. The sweat chloride test (answer E) is used to diagnose patients with cystic fibrosis, which should always be on the differential in any child with chronic lung problems (or diarrhea or failure to thrive). Because CF is such a serious disease, you should have a low threshold for ordering this test – but one episode of wheezing is not enough to make this the next step.

This question is not currently linked to the learning objective database.

Question problem?

Question # 70

Select the single best answer to the numbered question.

A 72 year old female with abdominal pain and unstable vital signs is found on angiography to have a ruptured abdominal aortic aneurysm. Past medical history includes hypertension, diabetes, and chronic renal insufficiency with baseline creatinine of 1.2 mg/dL. The patient is rushed to surgery and her aorta is successfully repaired in an eight hour operation where the patient receives five units of packed red blood cells and several liters of intravenous fluid. On the day following the operation, laboratory analysis reveals plasma creatinine of 2.1 mg/dL and BUN of 46 mg/dL. The fractional excretion of sodium (FeNa) is calculated to be 0.53%, and microscopic urinalysis shows occasional hyaline casts, but no RBCs, WBCs, or cellular or granular casts. Which of the following is the most likely explanation for this patient's abnormal laboratory values?

A. Cytotoxic effects of iodinated contrast material
B. Reduced blood flow to the kidneys
C. Ischemic necrosis of renal tubule cells

D. Obstruction of the distal urinary outflow system

E. Interstitial inflammation caused by drug hypersensitivity

You did not answer this question.

Explanations:

A. Cytotoxic effects of iodinated contrast material (answer A) is the mechanism of contrast nephropathy. Always consider this as a possibility in any patient who receives i.v. contrast – especially patients who are elderly or have diabetes or pre-existing chronic renal insufficiency. The clinical picture resembles ATN.

B. This patient presents with acute renal failure (ARF) and a sudden rise in her BUN and creatinine. While she has numerous possible causes for her ARF, the elevated BUN/creatinine ratio, low fractional excretion of sodium, and bland urine sediment all point to pre-renal azotemia, which can be caused by any condition that reduces blood flow to the kidneys (answer B). A few teaching points… 1) A great review article on acute renal failure is this one: http://jama.ama-assn.org/cgi/reprint/289/6/747.pdf The chart on page 3 is very helpful, as it shows a head-to-head comparison of the findings in various causes of acute renal failure. 2) What we call pre-renal "failure" is really a normal physiological mechanism that occurs in response to decreased renal perfusion pressure. Although the kidneys are failing to do their job of removing wastes from the body, the kidneys themselves haven't "failed" – if they were getting enough blood, they'd be able to do their job just fine! Therefore, here's an easy way to remember that the key feature of pre-renal disease is an elevated BUN/creatinine ratio. Remember that the body's normal BUN/Cr is 10-15:1. In ATN, that ratio doesn't change, because the body retains both BUN and creatinine equally. However, in pre-renal failure, the body retains more BUN than creatinine, leading to an increase in the BUN/Cr ratio. Therefore, if the kidneys are able to retain more BUN than creatinine, they must be intrinsically working okay – the diagnosis is pre-renal disease, not ATN. If you want a more detailed explanation, keep reading; otherwise, feel free to skip to the next teaching point! Unlike creatinine, which is freely filtered at the glomerulus and is not reabsorbed or metabolized by the kidney, about half of the filtered BUN gets passively reabsorbed, mainly at the proximal tubule. Remember that when there is decreased blood flow to the kidney, almost of the reabsorption of sodium and water occurs at the proximal tubule instead of other "downstream" segments of the nephron. Thus, urea nitrogen gets reabsorbed at the proximal tubule as well, leading to an increase in BUN that is greater than the increase in creatinine. 3) Other than pre-renal disease, there are a couple of other conditions that can increase the BUN out of proportion to the creatinine. One is a GI bleed – reabsorption of RBCs in the gastrointestinal tract will increase the BUN. Corticosteroids and tetracyclines (which inhibit protein anabolism) also can increase the BUN. 4) You can think about the FeNa in much the same way as the BUN/Cr ratio. If the FeNa is low – less than 1% - then the kidneys are avidly holding on to sodium. If they're able to do that, the kidneys themselves must be doing okay, and the problem must be that they're not getting enough blood. Patients with ATN have an elevated FeNa (>2%) because the kidney becomes like a leaky sieve and allows sodium to escape. 5) One important limitation of the FeNa is that it is affected by diuretics, which cause increased loss of sodium in the urine. In patients taking diuretics, the fractional excretion of urea (FeUrea) should be measured instead. 6) Hyaline casts are occasionally seen in normal urine, and so they are commonly mentioned as a distractor on the USMLE. Other casts are always pathological. The finding of "muddy brown" or granular casts is pathognomonic for ischemic ATN. Cytotoxic effects of iodinated contrast material (answer A) is the mechanism of contrast nephropathy. Always consider this as a possibility in any patient who receives i.v. contrast – especially patients who are elderly or have diabetes or pre-existing chronic renal insufficiency. The clinical picture resembles ATN. Ischemic necrosis of renal tubule cells (answer C) is the mechanism of ischemic ATN. This is certainly possible in a patient who has lost as much blood as this one, but the BUN/creatinine ratio should be around 15:1 and the FeNa >2%. Obstruction of the distal urinary outflow system (answer D) is the mechanism of obstructive uropathy or post-renal failure. Although a stone or iatrogenic injury can cause acute renal failure, in most cases the renal failure develops due to a more slowly progressive lesion – most commonly an enlarged prostate or malignant invasion. Obstruction can occur anywhere, from the renal pelvis to the distal urethra, but remember that only obstructions that functionally block BOTH kidneys will lead to a rise in BUN and creatinine. Interstitial inflammation caused by drug hypersensitivity (answer E) is the mechanism of acute interstitial nephritis. There is a long list of offending drugs, with methicillin being the most common culprit. The key finding that separates AIN from other causes of acute renal failure is eosinophils in the urine.

C. Ischemic necrosis of renal tubule cells (answer C) is the mechanism of ischemic ATN. This is certainly possible in a patient who has lost as much blood as this one, but the BUN/creatinine ratio should be around 15:1 and the FeNa >2%.

D. Obstruction of the distal urinary outflow system (answer D) is the mechanism of obstructive uropathy or post-renal failure. Although a stone or iatrogenic injury can cause acute renal failure, in most cases the renal failure develops due to a more slowly progressive lesion – most commonly an enlarged prostate or malignant invasion. Obstruction can occur anywhere, from the renal pelvis to the distal urethra, but remember that only obstructions that functionally block BOTH kidneys will lead to a rise in BUN and creatinine.

E. Interstitial inflammation caused by drug hypersensitivity (answer E) is the mechanism of acute interstitial nephritis. There is a long list of offending drugs, with methicillin being the most common culprit. The key finding that separates AIN from other causes of acute renal failure is eosinophils in the urine.

This question is not currently linked to the learning objective database.

Question problem?

Question # 71

Select the single best answer to the numbered question.

A 38 year old woman has had four weeks of watery, non-bloody diarrhea and cramping abdominal pain. She denies a history of similar symptoms, sick contacts, drinking unpurified water, and recent travel. There have been no changes to her diet, and she does not drink milk or eat dairy products. Current medications include esomeprazole and ibuprofen. She completed a course of amoxicillin/clavulanate for a sinus infection two months ago. On physical examination, she is afebrile. Bowel sounds are slightly hyperactive, and there is mild lower abdominal tenderness to palpation. Which of the following tests will most likely lead to the diagnosis in this patient?

A. Sudan stain for fecal fat

B. Microscopic examination of the stool for ova and parasites

C. D-xylose test

D. Barium upper gastrointestinal series

E. ELISA for C.difficile-associated toxins A and B

You did not answer this question.

Explanations:

A. The Sudan stain for fecal fat (answer A) is a qualitative assessment for steatorrhea. Fat malabsorption (such as from pancreatic insufficiency or chronic pancreatitis) can cause diarrhea, especially when it is described as malodorous, bulky, greasy stools. If you are considering this diagnosis, then a Sudan stain or 72 hour quantitative fecal fat measurement can be useful.

B. Microscopic stool analysis for ova and parasites (answer B) can diagnose diarrhea caused by organisms such as Giardia, Cryptosporidium, and Entamoeba histolytica. For patients without a travel history or risk factors such as drinking unpurified water or male homosexual activity, this test is not cost-effective.

C. The D-xylose test (answer C) is used to diagnose carbohydrate malabsorption, which can cause diarrhea.

D. A barium upper GI series (answer D) can diagnose anatomical reasons for diarrhea such as small bowel diverticula or mucosal inflammation.

E. This patient presents with chronic diarrhea (diarrhea lasting >4 weeks), and her only risk factor is recent antibiotic use. The most likely diagnosis in this case is antibiotic-associated diarrhea caused by Clostridium difficile, and the way to confirm this is by an assay for C. difficile toxins A and B (answer E). A few teaching points… 1) There is a wide spectrum of C.difficile associated illness, ranging from diarrhea without colitis all the way to full-blown pseudomembranous colitis. In almost all cases, the disease emerges following antibiotic therapy (although there does appear to be an emerging population of patients with C.difficile infection who have no exposure to antibiotics). 2) C. difficile diarrhea or colitis can occur following exposure to any antibiotic, but the most notorious culprits are clindamycin, second and third-generation cephalosporins, and amoxicillin. 3) Treatment of C.difficile infection is with metronidazole. Resistant cases may require oral vancomycin. 4) An occasionally-tested point about the treatment of C. diff is this one: If a patient who has C.diff improves with metronidazole and then relapses, you should give them one more shot at therapy with metronidazole before going to oral vancomycin. 5) A brief review of Clostridium difficile infection from the American Family Physician can be found at http://www.aafp.org/afp/20050301/921.html The Sudan stain for fecal fat (answer A) is a qualitative assessment for steatorrhea. Fat malabsorption (such as from pancreatic insufficiency or chronic pancreatitis) can cause diarrhea, especially when it is described as malodorous, bulky, greasy stools. If you are considering this diagnosis, then a Sudan stain or 72 hour quantitative fecal fat measurement can be useful. Microscopic stool analysis for ova and parasites (answer B) can diagnose diarrhea caused by organisms such as Giardia, Cryptosporidium, and Entamoeba histolytica. For patients without a travel history or risk factors such as drinking unpurified water or male homosexual activity, this test is not cost-effective. The D-xylose test (answer C) is used to diagnose carbohydrate malabsorption, which can cause diarrhea. A barium upper GI series (answer D) can diagnose anatomical reasons for diarrhea such as small bowel diverticula or mucosal inflammation.

This question is not currently linked to the learning objective database.

Question problem?

Question # 72

Select the single best answer to the numbered question.

A four year old male presents with a painful right hand. One week ago, he was bitten by his pet kitten on the hand. His mother also reports an occasional low-grade fever. On physical exam, there are several erythematous, painful cutaneous papules and pustules present near the nearly healed bite site, as well as tender unilateral axillary lymphadenopathy on the right. Which of the following is the organism most likely to be the cause of this patient's infection?

A. Methicillin-sensitive Staphylococcus aureus

B. Yersinia pestis

C. Francisella tularensis

D. Bartonella henselae

E. Pasteurella multocida

You did not answer this question.

Explanations:

A. Staphylococcus aureus (answer A) causes cellulitis, and should always be considered whenever there is a history of skin breakage. As a sidenote, remember that methicillin-sensitive and methicillin-resistant strains of S.aureus cause virtually identical illnesses and cannot be distinguished clinically (unless there is a history of treatment failure with appropriate antibiotics).

B. Yersinia pestis (answer B) causes bubonic plague. The great epidemics of the middle ages were spread by fleas that fed on rats, but today, the most common exposure leading to infection is prairie dogs (which serves as a useful clue on standardized tests).

C. Francisella tularensis (answer C) causes tularemia. Although this disease can take many forms, the most commonly tested is the ulceroglandular form, in which ulcers appear at the site of inoculation and there is tender lymphadenopathy. The bacterium is carried by ticks that feed on rabbits, ground squirrels, and deer, and is common in Arkansas, Oklahoma, Missouri, and Texas. The classic presentation on the USMLE is a patient from one of this areas with a history of hunting or skinning rabbits.

D. This is "cat scratch disease," caused by Bartonella henselae.(answer D). A few teaching points… A) The key features are 1) painful regional lymphadenopathy (so always consider cat scratch

disease in a child with enlarged lymph nodes – some questions on the USMLE aren't as straightforward as this one!) and 2) exposure to kittens, even if there is no history of a bite or scratch (full-grown cats are less of a risk). B) The disease is usually self-limited. Antibiotics like TMP/SMZ or azithromycin may shorten symptom duration by a day or two in a normal patient, and should be used in any immunocompromised patient due to the increased risk of developing disseminated disease. Painful, fluctuant lymph nodes may be aspirated to relieve symptoms. Staphylococcus aureus (answer A) causes cellulitis, and should always be considered whenever there is a history of skin breakage. As a sidenote, remember that methicillin-sensitive and methicillin-resistant strains of S.aureus cause virtually identical illnesses and cannot be distinguished clinically (unless there is a history of treatment failure with appropriate antibiotics). Yersinia pestis (answer B) causes bubonic plague. The great epidemics of the middle ages were spread by fleas that fed on rats, but today, the most common exposure leading to infection is prairie dogs (which serves as a useful clue on standardized tests). Francisella tularensis (answer C) causes tularemia. Although this disease can take many forms, the most commonly tested is the ulceroglandular form, in which ulcers appear at the site of inoculation and there is tender lymphadenopathy. The bacterium is carried by ticks that feed on rabbits, ground squirrels, and deer, and is common in Arkansas, Oklahoma, Missouri, and Texas. The classic presentation on the USMLE is a patient from one of this areas with a history of hunting or skinning rabbits. Pasteurella multocida (answer E) is found in the mouths of animals like cats and dogs and causes cellulitis after a bite. The unusual feature is that the cellulitis develops very quickly after the bite – often within just a few hours.

E. Pasteurella multocida (answer E) is found in the mouths of animals like cats and dogs and causes cellulitis after a bite. The unusual feature is that the cellulitis develops very quickly after the bite – often within just a few hours.

This question is not currently linked to the learning objective database.

Question problem?

Question # 73

Select the single best answer to the numbered question.

A three year old male comes to the physician because of delayed speech development. The patient's mother is also concerned because her son makes poor eye contact and often repetitively bites or flaps his hands. Family history is significant for a maternal uncle with mild mental retardation. On physical exam, the patient has a long face with a large jaw and large everted ears. Further testing reveals normal hearing and vision, and an IQ of 55. Which of the following is the most likely diagnosis in this patient?

A. Tay-Sachs disease
B. Fragile X syndrome
C. Down syndrome
D. Prader-Willi syndrome
E. Rett syndrome

You did not answer this question.

Explanations:

A. Tay-Sachs disease (answer A) is an autosomal recessive disorder that predominantly affects Jews of European descent. The primary defect is the absence of hexosaminidase A, which leads to the accumulation of gangliosides in the lysosomes of neurons. Patients present within the first year of life with developmental regression (i.e., losing previously acquired developmental milestones) and are usually dead by age three. A clue to the diagnosis is the finding of a "cherry red" spot in the macula.

B. This is Fragile X syndrome (answer B), the second most common chromosomal cause of mental retardation (after trisomy 21). It is an X-linked disorder that generally affects males (though female carriers may have some subtle symptoms), and because it is a trinucleotide repeat disorder, it tends to show genetic anticipation (that is, the disease becomes more severe and has an earlier onset in each generation). Key findings on physical examination include dysmorphic facial features (like a long face with a large jaw and large ears) and macro-orchidism. Patients may also display autistic-type behaviors (as the patient in this question stem did). A review of the diagnosis and management of Fragile X can be found at: http://www.aafp.org/afp/20050701/111.html Tay-Sachs disease (answer A) is an autosomal recessive disorder that predominantly affects Jews of European descent. The primary defect is the absence of hexosaminidase A, which leads to the accumulation of gangliosides in the lysosomes of neurons. Patients present within the first year of life with developmental regression (i.e., losing previously acquired developmental milestones) and are usually dead by age three. A clue to the diagnosis is the finding of a "cherry red" spot in the macula. Down syndrome (answer C) is caused by trisomy 21, and is the most common genetic cause of mental retardation. You should absolutely know the cardinal features of the disease, including up-slanting palpebral fissures, epicanthal folds, Brushfield spots, protruding tongue, and a single flexion crease in the palm (simian crease). The USMLE also frequently tests the fact that Down syndrome is associated with an increased risk of other defects and diseases, such as endocardial cushion defects and VSDs, tracheoesophageal fistula, Hirschsprung's disease, ALL, atlantoaxial instability, and (later in life) Alzheimer's disease. Prader-Willi syndrome (answer D) is caused by a paternal microdeletion or maternal disomy of a small section of chromosome 15 (while maternal microdeletion or paternal disomy causes Angelman's syndrome). The most striking feature of this disorder is obesity (caused by an insatiable appetite and obsessive food-seeking behaviors). Patients also usually have mild mental retardation and some dysmorphic features like almond shaped palpebral fissures, a narrow nasal bridge, and a down-turned mouth. Rett syndrome (answer E) is an uncommon disorder that is commonly tested (and commonly used as a distractor) on the USMLE. It affects girls, who present with deceleration of head growth (usually before age one) and rapid regression of developmental skills starting at ages 1-4. Rett syndrome is grouped on the spectrum of autistic disorders, and patients may have stereotyped movements or actions. Seizures are also not uncommon.

C. Down syndrome (answer C) is caused by trisomy 21, and is the most common genetic cause of mental retardation. You should absolutely know the cardinal features of the disease, including up-slanting palpebral fissures, epicanthal folds, Brushfield spots, protruding tongue, and a single flexion crease in the palm (simian crease). The USMLE also frequently tests the fact that Down syndrome is associated with an increased risk of other defects and diseases, such as endocardial cushion defects and VSDs, tracheoesophageal fistula, Hirschsprung's disease, ALL, atlantoaxial instability, and (later in life) Alzheimer's disease.

D. Prader-Willi syndrome (answer D) is caused by a paternal microdeletion or maternal disomy of a small section of chromosome 15 (while maternal microdeletion or paternal disomy causes Angelman's syndrome). The most striking feature of this disorder is obesity (caused by an insatiable appetite and obsessive food-seeking behaviors). Patients also usually have mild mental retardation and some dysmorphic features like almond shaped palpebral fissures, a narrow nasal bridge, and a down-turned mouth.

E. Rett syndrome (answer E) is an uncommon disorder that is commonly tested (and commonly used as a distractor) on the USMLE. It affects girls, who present with deceleration of head growth (usually before age one) and rapid regression of developmental skills starting at ages 1-4. Rett syndrome is grouped on the spectrum of autistic disorders, and patients may have stereotyped movements or actions. Seizures are also not uncommon.

This question is not currently linked to the learning objective database.

Question problem?

Question # 74

Select the single best answer to the numbered question.

A 72 year old female presents after "passing out" while walking back to her house from the mailbox. Other than an abrasion on her elbow, she denies any trauma from the fall and recovered quickly following the incident. There was no bowel or bladder incontinence or prodrome before the attack. On review of systems, the patient admits to occasional exertional chest pains that are relieved by taking her husband's nitroglycerin. Physical examination shows vital signs of heart rate 75 and regular, blood pressure 138/78, and respirations 13/min. There is a small abrasion and associated ecchymosis on the patient's left elbow, but no other signs of trauma. On cardiac auscultation, there is a harsh, III/VI systolic crescendo-decrescendo murmur heard best at the base of the heart and bilaterally at the clavicles. Lung fields are clear, and there is no peripheral edema. Neurological examination shows no focal abnormality. EKG shows normal sinus rhythm with left ventricular hypertrophy and normal PR and QT intervals. Which of the following is the most appropriate step in the management of this patient's syncope?

A. Urine drug screen
B. Head CT
C. Tilt table testing
D. Referral to a cardiologist for pacemaker placement
E. Referral to a cardiac surgeon for valve replacement

You did not answer this question.

Explanations:

A. A urine drug screen (answer A) is not useful in evaluating syncope. While some drugs – most notably anti-hypertensives (especially alpha blockers) and anti-arrythmics (which occasionally have pro-arrythmogenic effects) – can cause syncope, drugs of abuse are not likely causes, and those are what you'd pick up on a urine drug screen.

B. A head CT (answer B) is commonly ordered in patients with syncope, and while it may help rule out complications of syncope (such as a head bleed or skull fracture from a fall), it is rarely useful in revealing the cause of the syncope itself. In one study, only 1% of patients with syncope had a neurological cause, and even in those cases, a CT probably wouldn't have helped. (Most of the patients had transient ischemic attacks.) Remember that only gross anatomical detail shows up on CT, and that the pathology you'll see (like an acute bleed, an old stroke, a tumor, or dilated ventricles) are not usually conditions that present with syncope.

C. Tilt table testing (answer C) is used to help diagnose neurocardiogenic or vasovagal syncope. The test attempts to induce bradyarrhythmias or syncope by tilting the patient backwards, or if that fails, by administering nitroglycerin or isoproterenol. It is not the best choice in this patient because of her history and physical findings suggesting cardiac valvular disease.

D. Referral to a cardiologist for pacemaker placement (answer D) is inappropriate, as it ignores the diagnosis suggested by the question stem. Although arrhythmias are common causes of syncope, this patient's heart problem is primarily a structural one, not an electrical one.

E. Given the findings on physical examination, the cause of this patient's syncope is most likely to be aortic stenosis. The natural history of aortic stenosis includes a long asymptomatic period when their valvular disease does not cause any hemodynamically significant pathology . However, the ultimate course of the disease was illustrated by an old study that demonstrated that once a patient has developed one of three ominous symptoms - angina, syncope, or heart failure - their average survival time is less than two to three years, with a significant risk for sudden death. For this reason, any person with symptomatic aortic stenosis needs referral to a cardiothoracic surgeon for valve replacement (answer E). A couple of additional teaching points… 1) Syncope is defined as the abrupt and transient loss of consciousness associated with an absence of postural tone. It may sometimes be difficult to distinguish true syncope from things like seizures and cardiac arrest. Patients who describe bowel or bladder incontinence are more likely to have had a seizure, while patients who require CPR for resuscitation have more likely had a true cardiac arrest than simple syncope. 2) There are a number of causes of syncope. Approximately half of all patients with syncope have it due to neurally-mediated syncope caused by vasovagal activity, orthostatic hypotension, carotid sinus pressure, or situational events. Suspect one of these causes if a patient's syncope was preceded by emotional distress, prolonged standing, or severe pain, or if it occurred immediately after urination, defecation, coughing, or swallowing. About 25% of patients have a cardiac cause for their syncope, usually a brady- or tachyarrythmia. However, as this question illustrates, valvular disease can also be an important cause of syncope. Only a small portion of patients have a diagnosable neurologic or psychiatric illness underlying their syncope, while the rest of patients have an undefined cause of syncope. A urine drug screen (answer A) is not useful in evaluating syncope. While some drugs – most notably anti-hypertensives (especially alpha blockers) and anti-arrythmics (which occasionally have pro-arrythmogenic effects) – can cause syncope, drugs of abuse are not likely causes, and those are what you'd pick up on a urine drug screen. A head CT (answer B) is commonly ordered in patients with syncope, and while it may help rule out complications of syncope (such as a head bleed or skull fracture from a fall), it is rarely useful in revealing the cause of the syncope itself. In one study, only 1% of patients with syncope had a neurological cause, and even in those cases, a CT probably wouldn't

a fall), it is rarely useful in revealing the cause of the syncope itself. In one study, only 1% of patients with syncope had a neurological cause, and even in those cases, a CT probably wouldn't have helped. (Most of the patients had transient ischemic attacks.) Remember that only gross anatomical detail shows up on CT, and that the pathology you'll see (like an acute bleed, an old stroke, a tumor, or dilated ventricles) are not usually conditions that present with syncope. Tilt table testing (answer C) is used to help diagnose neurocardiogenic or vasovagal syncope. The test attempts to induce bradyarrhythmias or syncope by tilting the patient backwards, or if that fails, by administering nitroglycerin or isoproterenol. It is not the best choice in this patient because of her history and physical findings suggesting cardiac valvular disease. Referral to a cardiologist for pacemaker placement (answer D) is inappropriate, as it ignores the diagnosis suggested by the question stem. Although arrhythmias are common causes of syncope, this patient's heart problem is primarily a structural one, not an electrical one.

This question is not currently linked to the learning objective database.

Question problem?

Question # 75

Select the single best answer to the numbered question.

A 31 year old male with a 9 year history of ulcerative colitis presents with severe abdominal pain for the past few hours. He had been stable with sulfasalazine treatment, but one week ago was started on high-dose prednisone and loperamide for a flare. Vital signs are temperature 38.6 C (101.6 F), pulse 120, blood pressure 118/75, and respirations 20/min. The abdomen is distended, with markedly decreased bowel sounds. Though the lower abdomen is tender, there is no rebound tenderness or rigidity. Laboratory analysis is notable for a white blood count of 12,000 (normal: 4,500-11,000) and a hematocrit of 34.5% (normal: 39-49%). The remainder of the patient's labs, including LFTs, lipase, and lactic acid, are normal. Which of the following is the most likely diagnosis in this patient?

A. Clostridium difficile colitis
B. Sigmoid volvulus
C. Acute pancreatitis
D. Ischemic colitis
E. Toxic megacolon

You did not answer this question.

Explanations:

A. The big risk factor for Clostridium difficile colitis (answer A) is recent antibiotic use. Like anything else that causes inflammation of the colon, C.diff colitis can ultimately cause toxic megacolon, but it is not the best answer in this case.

B. Sigmoid volvulus (answer B) occurs when the sigmoid colon completely twists on its mesentery. It usually occurs in elderly, institutionalized patients (or very young children with malrotation). Plain film x-rays are usually diagnostic, and the rotation can frequently be reduced with a barium or water enema.

C. Acute pancreatitis (answer C) should always be considered in a patient with acute abdominal pain. However, that diagnosis is essentially ruled out here by the normal lipase level.

D. The typical patient with acute ischemic colitis (answer D) is an elderly patient with vascular risk factors for atherosclerosis or new-onset atrial fibrillation. It can be a difficult diagnosis to make, but an elevated lactic acid supports the diagnosis. The patient's presentation is usually described as "pain out of proportion to physical exam findings." Chronic mesenteric ischemia presents with "intestinal angina", where a patient has postprandial pain due to vascular insufficiency.

E. One of the most feared complications of ulcerative colitis is presented here: toxic megacolon (answer E). Some teaching points for this case… 1) The definition of toxic megacolon is total or segmental nonobstructive colonic dilation plus systemic toxicity. Although IBD is the most frequent cause, it can be caused by colon cancer, C. difficile infection, ischemic bowel disease, or volvulus. 2) To confirm the diagnosis of toxic megacolon, plain film x-rays of the abdomen should be sufficient, though a CT scan might be preferred if you want to rule out other causes of pathology. 3) Treatment of toxic megacolon is initially medical, with nasogastric suction, antibiotics, bowel rest, and i.v. fluid. If the patient worsens, though, surgical intervention may required. 4) Peritoneal signs like rebound tenderness, guarding, or rigidity indicate bowel perforation in the setting of toxic megacolon, just as they might in other scenarios. However, high-dose steroid therapy – like this patient has been receiving – may mask these signs. 5) This patient should not have been receiving loperamide. Antimotility agents, opiates, and anticholinergics all can worsen or contribute to the development of toxic megacolon. The big risk factor for Clostridium difficile colitis (answer A) is recent antibiotic use. Like anything else that causes inflammation of the colon, C.diff colitis can ultimately cause toxic megacolon, but it is not the best answer in this case. Sigmoid volvulus (answer B) occurs when the sigmoid colon completely twists on its mesentery. It usually occurs in elderly, institutionalized patients (or very young children with malrotation). Plain film x-rays are usually diagnostic, and the rotation can frequently be reduced with a barium or water enema. Acute pancreatitis (answer C) should always be considered in a patient with acute abdominal pain. However, that diagnosis is essentially ruled out here by the normal lipase level. The typical patient with acute ischemic colitis (answer D) is an elderly patient with vascular risk factors for atherosclerosis or new-onset atrial fibrillation. It can be a difficult diagnosis to make, but an elevated lactic acid supports the diagnosis. The patient's presentation is usually described as "pain out of proportion to physical exam findings." Chronic mesenteric ischemia presents with "intestinal angina", where a patient has postprandial pain due to vascular insufficiency.

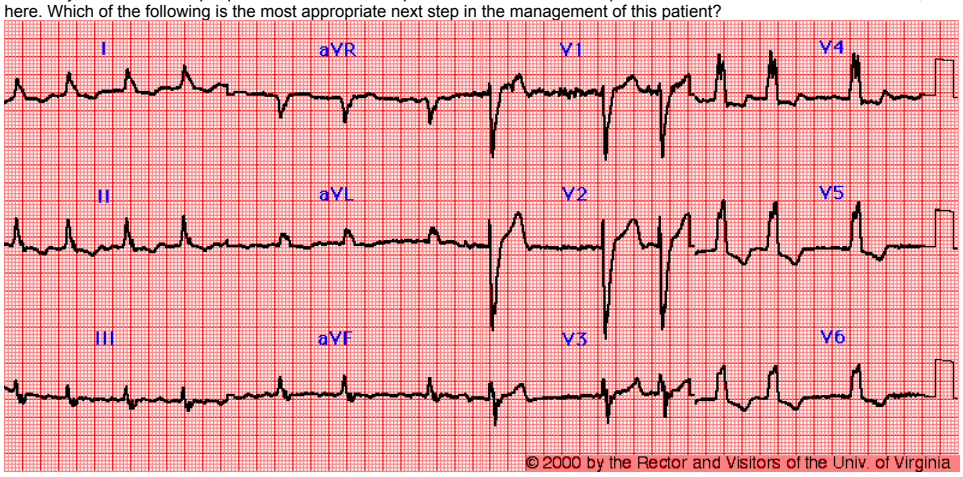
This question is not currently linked to the learning objective database.

Question problem?

Question # 76

Select the single best answer to the numbered question.

A 57 year old male presents with three weeks of fatigue. He denies depressed mood, palpitations, changes in bowel habits, syncope, or new medications. On physical examination, the patient's temperature is 36.7 C (98.6 F), pulse 100, and blood pressure 126/82. Peripheral pulses are 2+, but are noted to be irregularly irregular. Breath sounds are equal bilaterally, and there is no peripheral edema. Initial troponin I is not elevated. The patient is started on intravenous diltiazem, and an EKG is obtained. The patient's EKG is seen here. Which of the following is the most appropriate next step in the management of this patient?



A. Heparin and warfarin
B. Cardiac catheterization
C. Cardiac defibrillator implantation
D. Ibutilide
E. Electrical conversion

You did not answer this question.

Explanations:

A. This patient presents with what is likely chronic atrial fibrillation. At this point, the most appropriate intervention among those listed is to begin anticoagulation with heparin and warfarin (answer A). This question raises a number of important issues regarding the treatment of atrial fibrillation. 1) When the atrium isn't squeezing blood out appropriately, thrombus formation can result. If the atrium suddenly starts squeezing again, that thrombus can embolize, causing organ damage or stroke. For this reason, before electrical or pharmacological cardioversion is attempted, the patient should be adequately anticoagulated. (The exception to this is in a severely hemodynamically unstable patient – in that case, the risk of doing nothing is much greater than the risk of causing a stroke.) Even if you're not going to attempt cardioversion, a patient with either chronic or paroxysmal atrial fibrillation should be anticoagulated to reduce their stroke risk, which is why the best answer among those listed is answer A. 2) In atrial fibrillation, electrical impulses move randomly around the atrium instead of generating one coordinated contraction. The loss of a concerted atrial "kick" impairs left ventricular filling and can cause hemodynamic compromise, which can be mild (resulting in symptoms of fatigue) or severe, resulting in CHF, myocardial ischemia, or full-blown shock. 3) Atrial fibrillation is often accompanied by a rapid ventricular response, in which the patient's pulse may exceed 150-200 bpm. (This explains why many patients with a-fib present with palpitations or syncope.) 4) If a patient has a rapid ventricular response, the appropriate therapy to leave the patient in atrial fibrillation but control their rapid heart rate with calcium-channel blockers or beta blockers. Numerous studies have shown that this is preferable to attempting to control the patient's rhythm with antiarrhythmic drugs. Had this patient not received diltiazem already, starting rate-control therapy would have been the most appropriate next step in management. 5) Although the description of an "irregularly irregular" pulse is also a giveaway, you should know the findings of atrial fibrillation on EKG and be able to recognize them at a glance. Note the absence of p-waves – instead, all you see is a wavy baseline. QRS spikes occur at random intervals with no relation to p-waves. If you want to brush up on your EKGs, remember the University of Wisconsin website: http://www.fammed.wisc.edu/pcc/ecg/ 6) Atrial fibrillation that lasts longer than 7 days is considered chronic, and is unlikely to revert to sinus rhythm spontaneously. 7) Hyperthyroidism is a common precipitant of atrial fibrillation, and so always check a TSH. 8) For a nice management flow-chart for recent-onset atrial fibrillation, see Falk, RH. N Engl J Med 2001; 344:1067, or check out the whole review article here:

http://content.nejm.org/cgi/reprint/344/14/1067.pdf Cardiac catheterization (answer B) might be appropriate if the patient had ischemic heart disease, but here, all signs point to atrial fibrillation. Atrial defibrillators (answer C) are increasingly being used for atrial fibrillation, but are still awaiting clear and convincing evidence of benefits. Ibutilide (answer D) is an antiarrhythmic agent that can be used to pharmacologically cardiovert a patient back to normal sinus rhythm. Other agents used for this purpose include amiodarone, propafenone, and flecainide. Pharmacological cardioversion carries the same risks and precautions as electrical cardioversion, however, and is not appropriate at this time. Although there is evidence that electrical cardioversion (answer E) can be used safely without anticoagulation in patients with confirmed new-onset atrial fibrillation, this patient's symptoms have been going on much longer, and he is hemodynamically stable, so it is not the best choice.

B. Cardiac catheterization (answer B) might be appropriate if the patient had ischemic heart disease, but here, all signs point to atrial fibrillation.

C. Atrial defibrillators (answer C) are increasingly being used for atrial fibrillation, but are still awaiting clear and convincing evidence of benefits.

D. Ibutilide (answer D) is an antiarrhythmic agent that can be used to pharmacologically cardiovert a patient back to normal sinus rhythm. Other agents used for this purpose include amiodarone, propafenone, and flecainide. Pharmacological cardioversion carries the same risks and precautions as electrical cardioversion, however, and is not appropriate at this time.

E. Although there is evidence that electrical cardioversion (answer E) can be used safely without anticoagulation in patients with confirmed new-onset atrial fibrillation, this patient's symptoms have been going on much longer, and he is hemodynamically stable, so it is not the best choice.

This question is not currently linked to the learning objective database.

Question problem?

Question # 77

Select the single best answer to the numbered question.

A 25 year old male with a history of intravenous drug abuse presents with fever, chills, and shortness of breath for two days. He last used heroin five days ago. Vital signs include temperature 38.8 C (102 F), pulse 90, blood pressure 116/78, respirations 18/min. There is a grade II/VI systolic murmur heard best at the lower right sternal border. Faint crackles are heard throughout the lung fields. There are no splinter hemorrhages, peripheral petechiae, or erythematous patches on the palms or soles. No abnormalities are seen on fundoscopic exam. Neurological exam is normal. A chest x-ray shows no evidence of pneumonia. Three sets of blood cultures are drawn, and the patient is started on empiric antibiotic therapy with vancomycin and cefepime. Which of the following is the most appropriate next step in the management of this patient?

A. Naloxone
B. Buprenorphine
C. Ciprofloxacin
D. Trans-thoracic echocardiogram
E. D-dimer

You did not answer this question.

Explanations:

A. Naloxone (answer A) is an opioid antagonist, while buprenorphine (answer B) is a partial opioid agonist. Both are capable of precipitating withdrawal symptoms in patients with opiate dependence. If this patient were acutely intoxicated with heroin, naloxone might help, and if he were entering a drug rehab program, buprenorphine could be appropriate. Neither is appropriate now, and starting either would ignore the patient's more pressing medical issues.

B. Naloxone (answer A) is an opioid antagonist, while buprenorphine (answer B) is a partial opioid agonist. Both are capable of precipitating withdrawal symptoms in patients with opiate dependence. If this patient were acutely intoxicated with heroin, naloxone might help, and if he were entering a drug rehab program, buprenorphine could be appropriate. Neither is appropriate now, and starting either would ignore the patient's more pressing medical issues.

C. Ciprofloxacin (answer C) is a good drug for gram-negative flora and Pseudomonas, but those are already well-covered with cefepime. There's no reason to add ciprofloxacin now.

D. Any patient with a heart murmur and a history of i.v. drug use should be considered to have infectious endocarditis until proven otherwise. The best test to pursue that diagnosis is a trans-thoracic echocardiogram (answer D). A few teaching points… 1) Infectious endocarditis is diagnosed by the Duke criteria. Although you need not memorize them, you should have a general sense of what they are. They include major criteria such as positive blood cultures or positive echocardiograms and minor criteria such as a predisposing condition (like valvular heart disease or i.v. drug use), fever, and physical signs like Roth spots, Osler nodes, and Janeway lesions. A neat online calculator can be found at http://www.medcalc.com/endocarditis.html 2) Because they are inoculating microorganisms into their venous system, i.v. drug users are much more prone to right-sided endocarditis than the general population. This is clinically significant, and would explain why this patient had no Janeway lesions or Osler nodes: whereas a patient with a left-sided vegetation will throw septic emboli into the systemic circulation, a patient with right-sided vegetations will only embolize to the lungs. Naloxone (answer A) is an opioid antagonist, while buprenorphine (answer B) is a partial opioid agonist. Both are capable of precipitating withdrawal symptoms in patients with opiate dependence. If this patient were acutely intoxicated with heroin, naloxone might help, and if he were entering a drug rehab program, buprenorphine could be appropriate. Neither is appropriate now, and starting either would ignore the patient's more pressing medical issues. Ciprofloxacin (answer C) is a good drug for gram-negative flora and Pseudomonas, but those are already well-covered with cefepime. There's no reason to add ciprofloxacin now. A D-dimer test (answer E) measures products of fibrin degradation, and can be a useful test in excluding the diagnosis of pulmonary embolism. That's not the most likely diagnosis in this patient, though, so rule out the more likely things first.

E. A D-dimer test (answer E) measures products of fibrin degradation, and can be a useful test in excluding the diagnosis of pulmonary embolism. That's not the most likely diagnosis in this patient, though, so rule out the more likely things first.

This question is not currently linked to the learning objective database.

Question problem?

Question # 78

Select the single best answer to the numbered question.

An 18 year old college student presents with the sudden onset of fever, headache, myalgias, and nausea and vomiting twelve hours ago. He also complains of neck stiffness and photophobia. Vital signs include temperature 39.7 C (103.5 F), pulse 120, blood pressure 95/60, and respirations 20/min. On physical examination, he is somnolent but arousable. There is pronounced nuchal rigidity. With the hips flexed, attempts to straighten the knee result in spasm of the hamstrings. Small, 1-2mm petechial lesions are present on the trunk, legs, and soft palate. Therapy with intravenous fluid and supplemental oxygen is begun, and lumbar puncture is performed. Which of the following is the most likely finding on CSF analysis in this patient?

A. Increased WBC with neutrophilic predominance, increased protein, decreased glucose
B. Increased protein with oligoclonal bands of IgG
C. Xanthrochromia and increased RBCs
D. Increased WBC with lymphocytic predominance, increased protein, normal glucose
E. Increased WBC with lymphocytic predominance, numerous RBCs, elevated protein, normal glucose

You did not answer this question.

Explanations:

A. This patient has bacterial meningitis, most likely caused by N.meningitides. The most likely findings on CSF analysis would include an elevated white count with neutrophilic predominance, decreased glucose, and increased protein (answer A). Had this been a real USMLE question, you likely would have been given exact laboratory values, so you would have had to determine in which ways they differ from the norm. Don't be overwhelmed by CSF analysis – just like with other labs, a few common-sense facts will get you a long way. 1) The presence of PMNs in the CSF usually indicates bacterial infection, while finding lots of lymphocytes indicates viral infection – just as it would in the blood. 2) If the glucose in the CSF is low, then it stands to reason that something is using it up. In matter of fact, it is the PMNs that cause a low glucose, but it makes an easy-to-remember mental picture to think of bacteria as gobbling up all of the glucose (and helps explain why the glucose is normal or only slightly decreased in viral meningitis). 3) Protein is normally kept out of the CSF by the blood-brain and blood-CSF barriers, so finding an elevated protein indicates meningeal irritation. Of course, the proteins can also be antibodies, so conditions like multiple sclerosis and Guillain-Barre' may also cause elevated protein. A great article on CSF analysis (including a chart on typical findings in bacterial vs. viral meningitis) can be found in the American Family Physician: http://www.aafp.org/afp/20030915/1103.html One additional teaching point… Meningeal signs include the presence of nuchal rigidity (resistance of the neck to passive flexion), Kernig's sign (described in the question stem), and Brudzinski's sign (when there is resistance to passive flexion of the neck with both hips and knees flexed). Any and all of these signs indicate meningeal irritation. Increased protein with oligoclonal bands of IgG (answer B) is seen pathognomonic for multiple sclerosis. Xanthrochromia and increased RBCs (answer C) are seen in subarachnoid hemorrhage. Xanthrochromia results from the breakdown of red cells, so finding pink or yellow CSF is more likely to mean subarachnoid hemorrhage than a traumatic tap. An increased WBC with lymphocytic predominance, increased protein, normal glucose(answer D) would be more consistent with a viral meningitis. An increased WBC with lymphocytic predominance and numerous RBCs (answer E) would be consistent with herpes encephalitis. Apart from a traumatic tap or a subarachnoid hemorrhage, the other major cause for a large number of RBCs in the CSF is herpes encephalitis.

B. Increased protein with oligoclonal bands of IgG (answer B) is seen pathognomonic for multiple sclerosis.

C. Xanthrochromia and increased RBCs (answer C) are seen in subarachnoid hemorrhage. Xanthrochromia results from the breakdown of red cells, so finding pink or yellow CSF is more likely to mean subarachnoid hemorrhage than a traumatic tap.

D. An increased WBC with lymphocytic predominance, increased protein, normal glucose(answer D) would be more consistent with a viral meningitis.

E. An increased WBC with lymphocytic predominance and numerous RBCs (answer E) would be consistent with herpes encephalitis. Apart from a traumatic tap or a subarachnoid hemorrhage, the other major cause for a large number of RBCs in the CSF is herpes encephalitis - and this is a commonly-tested association on Step 2, so even though it's not the best answer to this question, you still ought to file it away!

This question is not currently linked to the learning objective database.

Question problem?

Question # 79

Select the single best answer to the numbered question.

A 53 year old female with type II diabetes presents is brought to the physician by her husband because of lethargy, fatigue, and decreased responsiveness. She had been feeling

well until two days ago, when she developed myalgias and upper respiratory congestion. Medications include glipizide, metformin, lisinopril, and atorvastatin. On physical examination, the patient is somnolent but arousable. Vital signs include temperature 36.7 C (98.6 F), pulse 130, blood pressure 102/64, respirations 15/min. There is no nuchal rigidity. There is poor skin turgor and the patient's mucous membranes are dry. Laboratory evaluation shows the following: Na+ 124, K+ 4.0, Cl- 92, HCO3- 24, BUN 70, creatinine 2.1, glucose 965, total protein 6.1 g/dL, albumin 4.5 g/dL, AST 12 U/L, ALT 17 U/L, Alk. Phosphatase 110 U/L, total bilirubin 0.7 mg/dL. Which of the following is the most likely diagnosis in this patient?

A. Syndrome of inappropriate antidiuretic hormone secretion (SIADH)
B. Diabetic ketoacidosis
C. Bacterial meningitis
D. Nonketotic hyperosmolar coma
E. Acute sinusitis

You did not answer this question.

Explanations:

A. SIADH (answer A) should always be considered when you encounter a patient with hyponatremia (especially one on the boards). However, as described above, this patient's hyponatremia is a direct consequence of her extremely high glucose.

B. Patients with diabetic ketoacidosis (answer B) have an elevated anion gap.

C. Bacterial meningitis (answer C) should be considered and ruled out in any patient that presents with lethargy. However, given this patient's lab findings, it is not the most likely diagnosis.

D. This diabetic patient presents with lethargy and dehydration following an upper respiratory infection. Given this patient's lack of an anion gap, the fact that she is a type II diabetic, and the degree of her blood glucose elevation, the most appropriate diagnosis is a hyperosmolar hyperglycemic nonketotic coma or HHNK (answer D). Some teaching points… 1) In patients with an elevated glucose, it is very important to distinguish between diabetic ketoacidosis (DKA) and nonketotic hyperosmolar states. Some things that help you determine the difference are… a) Type I vs. Type II diabetes – DKA almost always affects type I diabetics. Pathogenetically, in DKA, the body has metabolic fuel available but reacts as if it is starving, because there is an abolute lack of insulin. Patients with type II diabetes usually still make plenty of insulin, but their bodies are resistant to insulin's actions. If insulin resistance becomes very severe, patients with type II diabetes can develop ketoacidosis, but typically DKA is a complication of type I diabetics. b) Degree of glucose elevation – Patients with DKA very seldom have a glucose that exceeds 700 or 800 mg/dL, while patients with nonketotic hyperglycemia may have a blood glucose that exceeds 1000 mg/dL. 2) The key feature to diagnosis in a patient with DKA is – no surprise – acidosis. This is usually manifested by an increased anion gap. This patient's anion gap is (Na+) – (Cl-) – (HCO3-) = 124 – 92 – 23 = 9 Although different sources quote different values, the normal range for an anion gap is usually given as 12 +/-4, or from 8 to 16. 3) Both DKA and hyperglycemic hyperosmotic nonketotic coma are commonly precipitated by acute stress (from dehydration, infections, or drug use). Increased stress hormones and glucagon increase the patient's blood sugar and begin the pathological cascade. 4) This patient's low sodium is pseudohyponatremia and is caused by her dramatically elevated blood glucose. (The term "pseudohyponatremia" is a bit misleading, because the patient's sodium really is decreased.) Pseudohyponatremia occurs whenever there is a high concentration of glucose, triglycerides, or ketones in the blood. Though the relationship between glucose increase and sodium decrease is nonlinear, a useful rule of thumb is that the sodium concentration will drop 2.5 mEq/L for every 100 mg/dL of glucose rise above normal. SIADH (answer A) should always be considered when you encounter a patient with hyponatremia (especially one on the boards). However, as described above, this patient's hyponatremia is a direct consequence of her extremely high glucose. As described above, patients with diabetic ketoacidosis (answer B) have an elevated anion gap. Bacterial meningitis (answer C) should be considered and ruled out in any patient that presents with lethargy. However, given this patient's lab findings, it is not the most likely diagnosis. Patients with acute sinusitis (answer E) complain of nasal congestion, fever, and headache, often with pain that radiates to the upper teeth. Treatment is with decongestants and antibiotics like amoxicillin or TMP/SMZ.

E. Patients with acute sinusitis (answer E) complain of nasal congestion, fever, and headache, often with pain that radiates to the upper teeth. Treatment is with decongestants and antibiotics like amoxicillin or TMP/SMZ.

This question is not currently linked to the learning objective database.

Question problem?

Question # 80

Select the single best answer to the numbered question.

A 52 year old female comes in for a yearly physical. She has been feeling completely well, and denies chest pain, shortness of breath, weight loss, decreased exercise tolerance, or change in stool frequency or caliber. Past medical history is significant for hypertension controlled with hydrochlorothiazide. Although she has a history of several abnormal Pap smears thirty years ago, her tests have been normal since then, with her last normal Pap occurring two years ago. She had a normal mammogram earlier this year. She has smoked one half a pack of cigarettes daily for thirty-five years, and drinks 1-2 glasses of wine nightly. Her mother died at age 63 from ovarian cancer, and her father died at age 70 from lung cancer. Her siblings are alive and well. EKG shows sinus rhythm with a normal axis and intervals. Of the following, which is the most appropriate screening test to offer this patient?

A. Pap smear
B. CA-125
C. Chest x-ray
D. Exercise stress test
E. Colonoscopy

You did not answer this question.

Explanations:

A. Pap smears (answer A) are one of the most effective cancer screening tools ever devised. However, if a patient has had three or more normal tests in a row, it is not necessary to continue with yearly exams – once every three years is sufficient.

B. CA-125 (answer B) is a tumor marker for epithelial ovarian tumors. Because is it nonspecific, it is not used as a screening tool in any population, but it does have value in monitoring disease progression in patients who have already been diagnosed.

C. A chest x-ray (answer C) has never been shown to be an effective screening tool for lung cancer. By the time that lung tumors are visible radiographically, it is almost always too late for any interventions that improve patient outcomes.

D. An exercise stress test (answer D) is used to risk stratify patients who have a moderate pre-test probability of having coronary artery disease. Although this patient does smoke and has hypertension, she is still fairly young, has a normal EKG, no significant family history of CAD, and no symptoms that suggest CAD. At this point, her pre-test probability of having significant coronary artery disease is probably fairly low, and a stress test is not needed.

E. You can always count on a question or two on the USMLE about health screening. This patient is over 50 years old, so she needs some type of colon cancer screening – a colonoscopy (answer E) would therefore be appropriate. In general, the recommendations for health screening tests are… 1) Colon cancer screening should begin at age 50 in normal patients (and even younger for high-risk patients) with flexible sigmoidoscopy or colonoscopy. Fecal-occult blood tests have not been shown to change mortality, but they may be used as a screening test as well. 2) Pap smears should initially be given annually once a woman is >18 years old or becomes sexually active. However, if a woman has had no new sexual partners and three normal Pap smears in a row, you can safely screen her once every three years instead of annually. 3) Mammograms are recommended starting every year or two at age 35 or 40, and then annually after age 50. Exact recommendations still remain controversial. 4) A digital rectal exam for prostate cancer should be given to all men annually at age 50. Of course, different organizations have slightly different recommendations regarding cancer screening, and these recommendations tend to change periodically. The best place to find a quick overview of recommendations online is from the National Cancer Institute: www.nci.nih.gov/cancer_information/testing/ Pap smears (answer A) are one of the most effective cancer screening tools ever devised. However, as described above, if a patient has had three or more normal tests in a row, it is not necessary to continue with yearly exams – once every three years is sufficient. CA-125 (answer B) is a tumor marker for epithelial ovarian tumors. Because is it nonspecific, it is not used as a screening tool in any population, but it does have value in monitoring disease progression in patients who have already been diagnosed. A chest x-ray (answer C) has never been shown to be an effective screening tool for lung cancer. By the time that lung tumors are visible radiographically, it is almost always too late for any interventions that improve patient outcomes. An exercise stress test (answer D) is used to risk stratify patients who have a moderate pre-test probability of having coronary artery disease. Although this patient does smoke and has hypertension, she is still fairly young, has a normal EKG, no significant family history of CAD, and no symptoms that suggest CAD. At this point, her pre-test probability of having significant coronary artery disease is probably fairly low, and a stress test is not needed.

This question is not currently linked to the learning objective database.

Question problem?

Question # 81

Select the single best answer to the numbered question.

A 19 year old male presents to the emergency room at midnight with wheezing and shortness of breath. Although history is difficult to obtain secondary to the patient's tachypnea, he is able to state that his symptoms started one hour ago and have been progressively worsening. Past medical history is significant for seasonal allergies and asthma. Current medications include inhaled albuterol and budesonide. On physical exam, the patient appears anxious and is sitting upright. Vital signs on presentation are pulse 120, respirations 42/minute, BP 118/68, and O2 Sat 93% on room air. Nasal flaring is noted. The skin appears pale. No murmurs, rubs, or gallops are appreciated on cardiac auscultation. Bilateral, symmetrical wheezing is present. ABG obtained on presentation shows pH 7.51, pO2 70 mm Hg, and pCO2 29 mm Hg. Supplemental O2 by nasal cannula is started, and the patient is treated with nebulized albuterol and i.v. methylprednisolone. Thirty minutes after treatment, the patient is reassessed. No wheezing is noted, and breath sounds are equal bilaterally. Vitals are pulse 121, respiratory rate 35/minute, BP is 120/71, which drops to 108/65 on inspiration. Repeat ABG is obtained, showing pH 7.40, pCO2 41 mm Hg, pO2 72 mm Hg. What is the most appropriate next step in management?

A. Obtain chest x-ray
B. Begin i.v. albuterol
C. Begin i.v. theophylline
D. Administer inhaled salmeterol
E. Intubation and mechanical ventilation

You did not answer this question.

Explanations:

A. If a chest x-ray (answer A) were obtained, it is unlikely to show anything more than hyperinflation. Asthma is a clinical diagnosis, not a radiographic one, and besides, you already have more than enough information to know that this patient is having a severe asthma exacerbation. The value of a chest x-ray is in ruling out other causes of the patient's symptoms, so the accepted indications for a chest x-ray in an asthma attack include chest pain, fever >38.9 C (102 F), rales, or physical signs of an extra-alveolar air leak (pneumomediastinum, pneumothorax, subcutaneous emphysema). This patient does not have any of these. Therefore, obtaining a chest x-ray - while not unreasonable - is not the most appropriate step.

B. Head to head trials have shown that i.v. albuterol (answer B) is actually less efficacious than nebulized albuterol, and additionally causes more toxicity. Therefore, inhaled albuterol or levalbuterol remains the mainstay of asthma treatment, although ipratropium (Atrovent) may also be beneficial acutely.

C. Theophylline (answer C) has generally fallen out of favor for asthma management. While it still is used occasionally for maintenance therapy, studies have failed to show any benefit in the setting of severe, acute asthma exacerbations.

D. Salmeterol (answer D) is a long-acting beta-2 agonist used for the management of CHRONIC asthma. While there is good evidence that salmeterol may decrease symptoms and the need for rescue inhalers, some studies have suggested that long-term salmeterol use without steroids increases mortality in at least a subgroup of patients. Thus, appropriate use of salmeterol for chronic asthma remains controversial. In any event, however, there is no role for long-acting beta-2 agonists in the management of ACUTE asthma exacerbations.

E. Key teaching point: A rising pCO2 in an asthma attack signals impending respiratory failure, not improvement! It is important to recognize that this patient is getting worse, not better. Typically, in an acute asthma attack, the patient's tachypnea causes them to "blow off" CO2, resulting in a primary, uncompensated respiratory alkalosis. Rising CO2 in the face of sustained tachypnea is a very ominous sign - it shows that the patient's airways are so constricted that he is no longer able to get rid of CO2! If a normal patient began breathing at 35-40/minute (like this patient) they would very soon have a pCO2 in the teens or 20s as they blew off their CO2. This patient also appears to be fatiguing as well - his respiratory rate has dropped from 42 to 35. Therefore, he must be intubated and mechanically ventilated to prevent hypercarbia and respiratory failure (answer E). Besides the ABG findings, there are several other key clues in the question stem that show you that this patient's asthma did not improve with initial therapy. The presence of pulsus paradoxus (a drop of >10 mm Hg on inspiration) correlates directly with the severity of the attack and is a predictor of a PCO2 >35mm Hg. Additionally, the patient's absent wheezing does not represent improvement, but rather the dreaded "silent chest" of a severe asthma attack. Remember that in order to hear wheezing on auscultation, you have to be moving enough air to create a sound! This patient has poor air flow, not clear lungs. The initial treatment for this patient was completely appropriate. Management of the acute asthma exacerbation begins - like any other situation - with the ABCs (airway, breathing, circulation). Start an i.v. and begin supplemental O2. Then give B2 agonists like albuterol. In an acute situation, a nebulized dose of albuterol is usually preferable to an inhaler, since patients may be frightened and unable to adequately time and coordinate the use of an inhaler. Also, for anything other than a mild exacerbation in which symptoms resolve promptly with administration of beta-2 agonists, systemic corticosteroids (like methylprednisolone) should be given as well. Steroids take time to work, so you need to get them on board should the patient take a turn for the worse. If a chest x-ray (answer A) were obtained, it is unlikely to show anything more than hyperinflation. Asthma is a clinical diagnosis, not a radiographic one, and besides, you already have more than enough information to know that this patient is having a severe asthma exacerbation. The value of a chest x-ray is in ruling out other causes of the patient's symptoms, so the accepted indications for a chest x-ray in an asthma attack include chest pain, fever >38.9 C (102 F), rales, or physical signs of an extra-alveolar air leak (pneumomediastinum, pneumothorax, subcutaneous emphysema). This patient does not have any of these. Therefore, obtaining a chest x-ray - while not unreasonable - is not the most appropriate step. Head to head trials have shown that i.v. albuterol (answer B) is actually less efficacious than nebulized albuterol, and additionally causes more toxicity. Therefore, inhaled albuterol or levalbuterol remains the mainstay of asthma treatment, although ipratropium (Atrovent) may also be beneficial acutely. Theophylline (answer C) has generally fallen out of favor for asthma management. While it still is used occasionally for maintenance therapy, studies have failed to show any benefit in the setting of severe, acute asthma exacerbations. Salmeterol (answer D) is a long-acting beta-2 agonist used for the management of CHRONIC asthma. While there is good evidence that salmeterol may decrease symptoms and the need for rescue inhalers, some studies have suggested that long-term salmeterol use without steroids increases mortality in at least a subgroup of patients. Thus, appropriate use of salmeterol for chronic asthma remains controversial. In any event, however, there is no role for long-acting beta-2 agonists in the management of ACUTE asthma exacerbations.

This question is not currently linked to the learning objective database.

Question problem?

Question # 82

Select the single best answer to the numbered question.

A 45 year old woman presents to her family physician complaining that the big toe on her right foot is yellow and somewhat painful at times. She noticed the discoloration of her nail last week when she removed her nail polish, and has been avoiding wearing her normal summer sandals because she feels embarrassed. She is otherwise healthy and has no history of diabetes or peripheral vascular disease. Physical exam reveals a thickened, yellow first toenail on the right foot; the other nails appear healthy. Peripheral pulses are strong and equal. There are no rashes or lesions on the surrounding skin. What is the most appropriate initial step in management of this patient?

A. Griseofulvin
B. KOH examination of nail scapings
C. Reassurance
D. Nail removal
E. Nail biopsy and culture

You did not answer this question.

Explanations:

A. Prescribing griseofulvin (choice A) without establishing the diagnosis of onychomycosis can be done, but is not the best choice.

B. This patient presents with an abnormal-looking toenail. Nail dystrophies such as psoriasis, eczema, trauma and lichen planus can all be responsible for abnormal looking nails. Although a fungal infection or onchomycosis is at the top of your differential, a recent study found that only 60% of abnormal-appearing nails are caused by onchomycosis, so before starting treatment you should confirm your diagnosis by performing a KOH examination of the nail scrapings (answer B). Some additional teaching points… 1) A KOH examination of nail scrapings confirms the diagnosis of onychomycosis when dermatophytic hyphae and arthrospores are seen. Many insurance companies require this test before they'll pay for the expensive medications to treat the infection. 2) You should always treat onychomycosis in patients with a history of lower extremity cellulitis with ipsilateral toenail infection, patients with diabetes who have venous insufficiency, edema or prior cellulitis, and patients experiencing discomfort or pain. You may also treat patients like this one who want treatment for cosmetic reasons. 3) First line treatments include three months of oral terbinafine and itraconazole, which have higher cure rates than griseofulvin. Be sure to check liver aminotransferases prior to starting continuous oral therapy. Fluconazole is another less efficacious option, but does not require liver enzyme testing. Patients should be told that improvement will continue after oral medications are stopped and that it can take up to a year to fully assess cure. Topical treatments have a very low efficacy and aren't recommended. Still, cure rates even with first line agents average at about 65-70%. Prescribing griseofulvin (choice A) without establishing the diagnosis of onychomycosis can be done, but is not the best choice for the reasons described above. Reassurance (choice C) isn't the best answer. This patient is experiencing discomfort from the infected toenail and also desires treatment for cosmetic reasons. Both are acceptable reasons to treat this patient. Nail removal (choice D) would not be the best choice at this point, since oral medications are available and aren't contraindicated in this patient. The presence of a dermatophytoma (solid collection of dermatophytes under the nail) is an indication for nail removal. Nail biopsy and culture (choice E) would be the best choice only if the KOH scraping examination had been negative and you were still trying to definitively establish the diagnosis of onychomycosis.

C. Reassurance (choice C) isn't the best answer. This patient is experiencing discomfort from the infected toenail and also desires treatment for cosmetic reasons. Both are acceptable reasons to treat this patient.

D. Nail removal (choice D) would not be the best choice at this point, since oral medications are available and aren't contraindicated in this patient. The presence of a dermatophytoma (solid collection of dermatophytes under the nail) is an indication for nail removal.

E. Nail biopsy and culture (choice E) would be the best choice only if the KOH scraping examination had been negative and you were still trying to definitively establish the diagnosis of onychomycosis.

This question is not currently linked to the learning objective database.

Question problem?

Question # 83

Select the single best answer to the numbered question.

A 13 month old female presents to her doctor two weeks after being successfully treated for an uncomplicated febrile UTI. Since that time, a voiding cystourethrogram (VCUG) showed grade I reflux bilaterally, and a renal ultrasound showed normal kidneys. The patient's past medical history is otherwise unremarkable. On physical exam, the patient is afebrile. Urinalysis is negative for leukocyte esterase and nitrite and microscopic examination shows only occasional hyaline casts and squamous epithelial cells. What is the most appropriate next step in the management of this patient?

A. Culture urine monthly
B. Repeat renal ultrasound in one month
C. Begin antibiotic prophylaxis
D. Repeat VCUG in three months
E. Reassurance

You did not answer this question.

Explanations:

A. Monthly urine cultures (choice A) are not necessary. Once you start antibiotic prophylaxis, routine screening cultures are appropriate every 3-4 months at most.

B. Repeating the renal ultrasound in 1 month (choice B) won't give you any additional useful information. The renal ultrasound is a tool to demonstrate kidney size and shape, as well as anomalies such as duplication or dilation of the ureters and other gross abnormalities. The American Association of Pediatrics recommends a renal ultrasound in all young kids after the first UTI.

C. This question addresses the appropriate management of vesicoureteral reflux (VUR). VUR is graded from I-V, with V being the most severe. Current recommendations for grade I-II reflux include medical management due to the high likelihood that the reflux will resolve on its own as the child grows older. Low dose antibiotic therapy (answer C) with TMP/SMZ or nitrofurantoin is appropriate until you document that the VUR has disappeared. Until then, routine surveillance cultures should be obtained every 3-4 months and whenever the child has an unexplained fever or symptoms of a UTI. A few other teaching points… 1) 40% of children with first febrile UTI will have VUR on VCUG. Girls are twice as likely to have VUR after an intial febrile UTI. 2) Current recommendations for screening for VUR include: a) Children <5 years old with their first febrile UTI b) Any male child with a first UTI c) Girls <3 years old with a first UTI d) Kids with recurrent UTIs or kids with other prenatal renal anomalies e) Kids with 3 UTIs caused by unusual organisms 3) Screening for VUR can be done by voiding cystourethrogram or by radionuclide cystogram (RNC). The latter results in less radiation exposure and is a more common tool for annual follow-up of VUR after it has been diagnosed. 4) Grading VUR is important to guide treatment. Grade I means that the urine refluxes into the ureter during voiding. Grade II means that the urine goes into the ureter and the collecting system, but that there is NO dilation. Grade III through V have varying levels of dilation, with V being the most severe. 5) The lower the grade, the higher the probability of spontaneous resolution and therefore, the more appropriate it is to medically manage the VUR. Monthly urine cultures (choice A) are not necessary. Once you start antibiotic prophylaxis, routine screening cultures are appropriate every 3-4 months at most. Repeating the renal

ultrasound in 1 month (choice B) won't give you any additional useful information. The renal ultrasound is a tool to demonstrate kidney size and shape, as well as anomalies such as duplication or dilation of the ureters and other gross abnormalities. The American Association of Pediatrics recommends a renal ultrasound in all young kids after the first UTI. Repeating the VCUG in 3 months (choice D) is not necessary. The current recommendation for monitoring reflux includes annual screening with either VCUG or RNC. Reassurance alone (choice E) is not appropriate. It is true that there is a high likelihood that grade 1-2 reflux will resolve on its own with time, and that surgical management will likely not be needed for this patient. Still, antibiotic prophylaxis is needed at this point to prevent recurrent UTIs and renal injury.

D. Repeating the VCUG in 3 months (choice D) is not necessary. The current recommendation for monitoring reflux includes annual screening with either VCUG or RNC.

E. Reassurance alone (choice E) is not appropriate. It is true that there is a high likelihood that grade 1-2 reflux will resolve on its own with time, and that surgical management will likely not be needed for this patient. Still, antibiotic prophylaxis is needed at this point to prevent recurrent UTIs and renal injury.

This question is not currently linked to the learning objective database.

Question problem?

Question # 84

Select the single best answer to the numbered question.

A 65 year old male comes to the physician because of fatigue and increasing abdominal girth. Over the past six months, he has also experienced an unintentional weight loss of 15 lbs., which he believes is due to the fact that he feels full after only a few bites of his meals. The patient has no past medical history, as this his first visit to a physician since being discharged from the army 40 years ago. The patient denies smoking but does drink 1-3 beers every evening. On physical examination, the patient appears emaciated with a visibly distended and protuberant abdomen. No icterus is noted. Neck veins are non-distended and lungs are clear to auscultation. There is an S4 present on cardiac auscultation. Abdominal examination shows shifting dullness to percussion and a positive fluid wave. There is a firm, red, nontender nodule at the umbilicus. Examination of the skin shows no spider angiomata or palmar erythema. There is 1+ edema at the ankles bilaterally. Labaratory results include albumin of 3.6 g/dL, AST 39 U/L, and ALT 29 U/L. Paracentesis is performed, yielding clear, straw colored fluid, with an albumin concentration of 2.7 g/dL, 650 leukocytes (85% lymphocytes, 15% PMNs). No organisms are seen on Gram stain. Which of the following is the most likely cause of this patient's ascites?

A. Spontaneous bacterial peritonitis
B. Alcoholic cirrhosis
C. Budd-Chiari syndrome
D. Congestive heart failure
E. Peritoneal metastatic disease

You did not answer this question.

Default Explanation:

This is a difficult question, with a dense question stem. Answering it correctly depends on your ability to correctly interpret the results of this patient's paracentesis. In the past, much effort was put into determining whether ascites fluid was a "transudate" or an "exudate" based on the values of multiple lab tests done on the fluid and serum. (This is still the way that pleural effusions are evaluated.) However, it is now clear that you can classify ascites with 95% accuracy only by knowing the serum albumin and the ascites albumin concentration and calculating the serum-ascites albumin gradient or SAAG. If the difference between the serum albumin and the ascites albumin is greater than 1.1, then the ascites is caused by portal hypertension – with cirrhosis, right sided CHF, and Budd-Chiari syndrome being the most frequent culprits. If the SAAG is less than 1.1, then the ascites is NOT caused by portal hypertension. In this situation, the most likely causes include pancreatitis, peritonitis, and peritoneal carcinomatosis. This patient's serum albumin is 3.6, while his ascites has an albumin of 2.7. This gives him a SAAG of 0.9, and indicates that his ascites is more of the exudative variety. Among the possibilities listed, the only one that causes a low-SAAG ascites is peritoneal metastatic disease (answer E). This patient also has a finding that suggest where his primary cancer might be: the firm nodule at the umbilicus is a Sister Mary Joseph's node, which is the calling card of gastric carcinoma. An excellent photograph (and one you won't forget) of this finding is found at the bottom of this webpage: http://www.surgical-tutor.org.uk/default-home.htm?system/abdomen/gastric_ca.htm~right Spontaneous bacterial peritonitis (answer A) may cause the acute worsening of ascites in a patient with cirrhosis. The key diagnostic feature is finding >250 PMNs in the ascites fluid. A lymphocytic predominance, such as this patient has, is more consistent with peritonitis from tuberculosis or peritoneal metastases. Alcoholic cirrhosis (answer B) is the most common cause of cirrhosis in the United States, but is associated with a wide SAAG. This patient does drink alcohol, and though he may benefit from cutting back a bit, drinking only 1-3 drinks a day is unlikely to cause cirrhosis in a normal patient. Also worth noting here is the fact that on the USMLE – unlike in real life – you can "trust" the patient to report an accurate amount of alcohol intake. If the patient says that he drinks 1-3 beers a day, then he does. If the question authors want to suggest that the patient is unreliable, there will be a clue statement such as "The patient gives evasive and inconsistent accounts of his drinking." Budd-Chiari syndrome (answer C) is caused by occlusion of the hepatic venous outflow, usually by thrombus. This leads to portal hypertension and a wide SAAG. The most common predisposing factors include hypercoagulable states or myeloproliferative disorders like polycythemia vera or essential thrombocytosis. Right-sided congestive heart failure (answer D) can cause ascites with a SAAG >1.1. The signs of right-sided CHF should be present, including peripheral edema, increased JVD, and hepatomegaly.

This question is not currently linked to the learning objective database.

Question problem?

Question # 85

Select the single best answer to the numbered question.

A 19 year old college female presents to student health because she is no longer menstruating. She has missed her last 5 menses and states that the 3 prior menses were very light and unpredictable. Menarche occurred at age 10 and her menses had been occurring at regular at 24 day intervals until this recent change. She is sexually active using only condoms for birth control. She takes no medications. The patient denies breast tenderness, abdominal fullness, and nausea, though she has noticed a 10 pound weight gain over the past few months. She feels that she might have a little less energy than usual. Physical exam is within normal limits. BMI is 21. A qualitative serum beta-hCG is negative for pregnancy. What is the most appropriate next step in the management of this patient?

A. Urine pregnancy test
B. Quantitative serum beta-hCG
C. Follicle stimulating hormone (FSH)
D. Serum thyroid stimulating hormone (TSH) and prolactin
E. Serum androgen levels

You did not answer this question.

Explanations:

A. A urine pregnancy test (answer A) isn't necessary when you already have a negative serum beta-hCG test. Remember that serum testing is more sensitive than urine testing.

B. Quantitative beta-hCG testing (answer B) isn't necessary at this point. The most important use for quantitative beta-hCG testing is in ruling out or managing ectopic pregnancies.

C. FSH testing (choice C) can be part of the intial work-up for secondary amenorrhea, but wouldn't be done alone initially unless the patient had signs clearly pointing to ovarian failure (such as vasomotor symptoms from estrogen deficiency, atrophic vaginitis and dyspareunia). In an older woman, FSH could be included as part of an intial basic test panel along with TSH, PRL and beta-hCG.

D. This question describes the management of secondary amenorrhea, defined as the postmenarchal cessation of menses for either 6 months or 3 cycles in a female with previously normal cycles. While there are many lab tests one could order for her work-up, they should be done in an orderly manner to optimize cost efficiency and patient care. The first step in the secondary amenorrhea work-up is serum is to rule out pregnancy. Serum beta-hCG is more sensitive test than the home pregnancy test and should be done even if the patient has had a negative home pregnancy test. For the young female, the most common causes of secondary amenorrhea besides pregnancy are thyroid and pituitary dysfunction, so checking a serum TSH and prolactin is the next step (answer D) In addition, the patient in the question stem has had weight gain and a possible change in energy, so hypothyroidism is something in particular to be thinking about in your work-up. A few additional points… 1) The first step in the work-up of secondary amenorrhea is ALWAYS a pregnancy test, with serum beta-hCG being a bit more sensitive than urine beta-hCG. 2) Appropriate initial tests to perform after pregnancy has been ruled out include: TSH to screen for hypothyroidism, prolactin to screen for a prolactinoma, and FSH to screen for primary ovarian failure. If polycystic ovarian syndrome is suspected by the history, androgen levels can be part of the initial work-up, otherwise they are usually not part of the initial work-up. 3) For a nice review article – including simple flow-charts on how to work up both primary and secondary amenorrhea, try this article from the American Family Physician: http://www.aafp.org/afp/20060415/1374.html A urine pregnancy test (answer A) isn't necessary when you already have a negative serum beta-hCG test. Remember that serum testing is more sensitive than urine testing. Quantitative beta-hCG testing (answer B) isn't necessary at this point. The most important use for quantitative beta-hCG testing is in ruling out or managing ectopic pregnancies. FSH testing (choice C) can be part of the intial work-up for secondary amenorrhea, but wouldn't be done alone initially unless the patient had signs clearly pointing to ovarian failure (such as vasomotor symptoms from estrogen deficiency, atrophic vaginitis and dyspareunia). In an older woman, FSH could be included as part of an intial basic test panel along with TSH, PRL and beta-hCG. Obtaining serum androgen levels (answer E) isn't the best answer at this point. This is usually not included in the initial basic work-up unless signs of polycystic ovarian failure exist in the history and physical exam. PCOS can present with signs of hyperandrogenism such as treatment resistant acne, hirsutism, or with signs of ovulatory dysfunction such as irregular bleeding, infertility and amenorrhea. Androgen levels can also reveal an androgen secreting tumor. Though obtaining serum androgen levels may be appropriate later on with this patient if the initial basic tests are unrevealing, this is certainly not the best next step.

E. Obtaining serum androgen levels (answer E) isn't the best answer at this point. This is usually not included in the initial basic work-up unless signs of polycystic ovarian failure exist in the history and physical exam. PCOS can present with signs of hyperandrogenism such as treatment resistant acne, hirsutism, or with signs of ovulatory dysfunction such as irregular bleeding, infertility and amenorrhea. Androgen levels can also reveal an androgen secreting tumor. Though obtaining serum androgen levels may be appropriate later on with this patient if the initial basic tests are unrevealing, this is certainly not the best next step.

This question is not currently linked to the learning objective database.

Question problem?

Question # 86

Select the single best answer to the numbered question.

A 53 year old female presents complaining of depression, insomnia, and increasing fatigue. She also describes occasional episodes where her face and neck become red and flushed. These frequently occur at night and wake her from sleep. Her last menstrual period was five months ago. Her menses had been irregular for one year, but before then, her menstrual history was normal. Physical exam is remarkable for an atrophic vaginal mucosa with decreased secretions. Which of the following diagnostic tests is most likely to

menstrual history was normal. Physical exam is remarkable for an atrophic vaginal mucosa with decreased secretions. Which of the following diagnostic tests is most likely to confirm the cause of this patient's amenorrhea?

A. Urine beta-hCG
B. Follicle stimulating hormone (FSH)
C. Thyroid stimulating hormone (TSH)
D. Karyotyping
E. Prolactin

You did not answer this question.

Explanations:

A. Measuring a urine beta-hCG (answer A) is always the first step in working up a case of primary or secondary amenorrhea. Before chasing down other possibilities, you should always rule out the possibility of pregnancy in any reproductive-aged woman.

B. This patient presents with the classic signs, symptoms, and demographics of menopause. If the diagnosis of menopause is uncertain, the finding of an elevated FSH can confirm the diagnosis (answer B), although in a patient such as this one the clinical history and findings are likely to be sufficient. The other answer choices provide useful information, and can be part of the workup of primary or secondary amenorrhea. While most of them are reasonable tests to order, none are likely to confirm the diagnosis suggested by the question stem. Measuring a urine beta-hCG (answer A) is always the first step in working up a case of primary or secondary amenorrhea. Before chasing down other possibilities, you should always rule out the possibility of pregnancy in any reproductive-aged woman. Finding an elevated thyroid stimulating hormone (answer C) would confirm a diagnosis of hypothyroidism, which is a cause of secondary amenorrhea. It is true that some of the symptoms of menopause and hypothyroidism overlap, but the giveaway in the question stem was the patient's description of having hot flashes. If you suspect that a patient has testicular feminization syndrome, karyotyping (answer D) can reveal a 46 XY genotype. These patients will present with primary amenorrhea – that is, a complete absence of menstrual periods. The primary defect in testicular feminization syndrome is the dysfunction or absence of the testosterone receptor, which leads to a phenotypical female with male chromosomes. Hyperprolactinemia (answer E) is another cause of secondary amenorrhea, which is why after a pregnancy test, the next step in a standard amenorrhea workup is the measurement of prolactin and TSH.

C. Finding an elevated thyroid stimulating hormone (answer C) would confirm a diagnosis of hypothyroidism, which is a cause of secondary amenorrhea. It is true that some of the symptoms of menopause and hypothyroidism overlap, but the giveaway in the question stem was the patient's description of having hot flashes.

D. If you suspect that a patient has testicular feminization syndrome, karyotyping (answer D) can reveal a 46 XY genotype. These patients will present with primary amenorrhea – that is, a complete absence of menstrual periods. The primary defect in testicular feminization syndrome is the dysfunction or absence of the testosterone receptor, which leads to a phenotypical female with male chromosomes.

E. Hyperprolactinemia (answer E) is another cause of secondary amenorrhea, which is why after a pregnancy test, the next step in a standard amenorrhea workup is the measurement of prolactin and TSH.

This question is not currently linked to the learning objective database.

Question problem?

Question # 87

Select the single best answer to the numbered question.

A 10 year old male presents with a one week history of a limp that seems to be getting worse. The patient describes hip and upper leg pain that is worst when he stands with his full weight on his left foot. The pain started last week, but yesterday it suddenly worsened after he jumped off of a moving swing yesterday. On physical examination, the patient moderately obese and at Tanner stage 2 of sexual development. He walks with a pronounced limp, and refuses to bear weight on the left foot. There is marked limitation of both active and passive range of motion at the hip. Examination of other joints is within normal limits. Which of the following is the most likely diagnosis in this patient?

A. Legg-Calve-Perthes disease
B. Slipped capital femoral epiphysis
C. Transient synovitis
D. Osgood-Schlatter disease
E. Growing pains

You did not answer this question.

Explanations:

A. Legg-Calve-Perthes disease (choice A) is an idiopathic avascular necrosis of the hip that occurs in children ages 3-12 with a peak incidence is 5-7 years old. It predominantly affects males and 10% of cases are familial. Patients present with a limp and Trendelenburg gait (pelvis tilts downward on unaffected side while trunk sways toward affected side during stance). Pain is mild and usually referred to anteromedial thigh or the knee, but can lead to disuse causing atrophy of the thigh and buttock. Physical exam reveals limited internal rotation and abduction of the hip. Initial radiographs are usually normal, but bone scans can show decreased perfusion to the femoral head, and MRI will show marrow changes.

B. This is a fairly classic presentation of SCFE, or slipped capital femoral epiphysis (answer B). This is a common cause of hip pain and altered gait in obese pre-adolescents, and is caused by displacement of the capital femoral epiphysis from the femoral neck through the growth plate. A few extra teaching points… 1) The plain film x-rays for this patient would show posterior displacement of the left femoral epiphysis. This finding is often described as "ice-cream slipping off a cone." 2) It is important to recognize this diagnosis and treat it in a timely manner to prevent joint damage. Patients should be immediately referred to an orthopedic surgeon for the placement of a screw through the center of the epiphysis. Acute slips are unstable, and require a hospital admission and bed rest. 3) As mentioned above, the typical patient is an obese child in early adolescence (

C. Transient synovitis (choice C), or TS, should be in the differential of a limp, especially in the pre-school to school-age group, as it is a common disorder that presents with pain and limitation of hip motions arising without a clear cause and resolving with only conservative therapy. Most kids present with symptoms for less than 1 week, are usually afebrile and non-toxic appearing. The cause is unknown, though an infectious cause is assumed because up to half of kids with TS had a recent upper respiratory tract infection. TS is managed with NSAIDs and rest for afebrile patients with a benign WBC and ESR. Imaging is needed if the clinical picture is concerning for hip infection.

D. Osgood-Schlatter disease (choice D) is an osteochondritis of the tibial tuberosity caused by overuse in active individuals. (Sports such as basketball and gymnastics are common culprits since jumping puts a lot of stress on the tibial tuberosity.) It usually presents in a 13-14 year old boy or 11-12 year old girl who has recently undergone a growth spurt. The most common complaint is anterior knee pain that progressively worsens over time, and is improved with rest. The diagnosis is clinical and the treatment is conservative.

E. Growing pains (choice E) are recurrent self-limited extremity pain most commonly described by kids ages 2-12 years old. They are benign and resolve on their own. The cause of growing pains is unknown, and although they occur in growing children, they are not caused by growth itself. There are no radiographic findings for growing pains, so the diagnosis is one of exclusion.

This question is not currently linked to the learning objective database.

Question problem?

Question # 88

Select the single best answer to the numbered question.

A 38 year old woman has sudden onset of nausea and vomiting and severe, colicky flank pain that radiates to the perineum. On physical examination, the patient shifts position frequently and seems unable to sit still. Urinalysis shows trace protein, 3+ blood, pH = 7.2. Microscopic analysis shows many red cells and a few white cells, but no casts or bacteria. Laboratory analysis shows: Na+ 141 K+ 3.0, Cl- 119, HCO3- 12, BUN 17, Creatinine 1.0, glucose 111, calcium 9.8, magnesium 1.6, phosphate 3.0. Which of the following is the most likely etiology of this patient's electrolyte abnormalities?

A. Increased production of endogenous lactic acid
B. Decreased net secretion of H+ at the distal renal tubule
C. Deficiency of aldosterone
D. Excessive release of parathyroid hormone (PTH)
E. Physiological stress response to pain

You did not answer this question.

Explanations:

A. Increased production of lactic acid (answer A) causes lactic acidosis, which causes an increased anion gap metabolic acidosis. Common causes of lactic acidosis include circulatory failure, ischemia, and inborn errors of metabolism.

B. Key teaching point: the differential diagnosis for a NON-anion gap metabolic acidosis is short – think of RTAs and GI bicarbonate loss. This question describes a patient with nephrolithiasis who is subsequently found to have a non-anion gap metabolic acidosis. These findings are consistent with a type 1 renal tubular acidosis (RTA) in which there is impaired secretion of H+ into the distal tubule and collecting duct (answer B). This in turn leads to the decreased bicarbonate and metabolic acidosis, and the alkalinization of the urine causes nephrolithiasis. Other key findings that help you make the diagnosis of a Type 1 or distal RTA are the low K+ and urine pH>5.5. This question raises a few important teaching points, as well. 1) Questions about RTAs are very common on the shelf exams – even the surgery shelf exam has been known to have a question about RTAs on it! You need to be able to diagnose them at a glance and distinguish amongst the three types based on lab data and urine pH. This latter task unfortunately requires some first-year med student style rote memorization, but here is a helpful chart to help you with that task:

http://web.archive.org/web/20070808145429/http://www.mayoclinic.com/health/renal-tubular-acidosis/AN00642

2) Broadly speaking, there are three ways that a person can develop metabolic acidosis: 1) Increased H+ entering the body (from lactate or ketoacids, for example) 2) Inability of H+ to be excreted by the kidney (as in RTA) 3) Inappropriate loss of HCO3- in the GI tract or the kidney Situation #1 causes a metabolic acidosis with an increased anion gap, while situations #2 and #3 cause a non-anion gap metabolic acidosis. Remember that the anion gap is calculated as (Na+) – (Cl-) – (HCO3-), and should be less than or equal to 12 +/- 4. If the anion gap is elevated, you should begin working through the "MUDPILES" mnemonic to look for a cause. If the anion gap is NOT elevated but the patient has a metabolic acidosis, there are really only two common things to think about: renal tubular acidosis and GI loss of bicarbonate (caused by diarrhea or loss of GI fluid beyond the ligament of Treitz). Increased production of lactic acid (answer A) causes lactic acidosis, which causes an increased anion gap metabolic acidosis. Common causes of lactic acidosis include circulatory failure, ischemia, and inborn errors of metabolism. A deficiency of aldosterone (answer C) is the pathologic mechanism underlying Type IV RTA. This results in an inability to absorb sodium at the intercalated cells of the collecting duct, resulting in hyponatremia, hyperkalemia, and impaired H+ secretion leading to a non-anion gap acidosis. Excessive release of parathyroid hormone (answer D) causes hyperparathyroidism. The characteristic abnormality

is an elevated calcium level. The physiological stress response to pain (answer E) might result in catecholamine surges or increased cortisol, but would not cause a bicarbonate of 12 and a metabolic acidosis.

C. A deficiency of aldosterone (answer C) is the pathologic mechanism underlying Type IV RTA. This results in an inability to absorb sodium at the intercalated cells of the collecting duct, resulting in hyponatremia, hyperkalemia, and impaired H+ secretion leading to a non-anion gap acidosis.

D. Excessive release of parathyroid hormone (answer D) causes hyperparathyroidism. The characteristic abnormality is an elevated calcium level.

E. The physiological stress response to pain (answer E) might result in catecholamine surges or increased cortisol, but would not cause a bicarbonate of 12 and a metabolic acidosis.

This question is not currently linked to the learning objective database.

Question problem?

Question # 89

Select the single best answer to the numbered question.

A 46 year old homeless man is brought to the hospital after passersby witnessed him having convulsions on the street. On physical examination, the patient is disheveled, unshaven, and has a faint odor of alcohol on his breath. He is semicomatose, and arouses only to pain. Vital signs are temperature 38.3 C (101 F), pulse 115, blood pressure 166/96, respirations 15/min, and oxygen saturation 98% on room air. There is a 4cm simple laceration on his left temple. There are no other visible injuries, and the remainder of the physical examination is normal. Laboratory evaluation shows the following: Hematocrit 38%; mean corpuscular volume 109 fL; sodium 133 mEq/L; glucose 62 mg/dL; magnesium 1.0 mEq/L; albumin 3.0 g/dL; ALT 60 IU/L; AST 123 IU/L; PT 19 s. The patient is placed in mechanical restraints and i.v. fluid with glucose, thiamine, folate, and magnesium is begun. Which of the following is the most appropriate next step in the management of this patient?

A. Intravenous diazepam
B. Oral phenobarbital
C. MRI of head and neck
D. Lumbar puncture
E. Intravenous somatostatin

You did not answer this question.

Explanations:

A. This patient presents with acute alcohol withdrawal. The seizure that he suffered on the street was likely a withdrawal seizure, and given his altered vital signs, he is likely experiencing delirium tremens as well. Before completing any additional diagnostic workup, the patient needs anticonvulsant therapy, and the best choice listed is i.v. diazepam (answer A). A few additional teaching points… 1) It is important to remember that alcohol withdrawal can occur while a patient is still drinking – for example, if a person goes from drinking twenty beers a day to only ten. Don't be fooled by the fact that the patient has the odor of alcohol on his breath – he is still in withdrawal! 2) If the question stem had not described giving the patient thiamine, it would be very important to do so immediately, before giving any i.v. fluid that contains glucose. Forgetting to do this increases the risk of precipitating Wernicke's encephalopathy or Korsakoff's syndrome. 3) In real life, you probably wouldn't have laboratory values available so quickly. They're presented here for teaching purposes, to remind you of some of the key lab abnormalities seen in alcoholics. These include hypoglycemia; macrocytic anemia; elevated AST and ALT, with a 2:1 ratio of AST/ALT; low magnesium; elevated PT; and low albumin. 4) Delirium tremens (DTs) begins 48 to 96 hours after a patient's last drink or reduction in drinking, and is characterized by hallucinations, disorientation, diaphoresis, tachycardia, hypertension, and low grade fever. (This time course is important, and tends to come up on the USMLE as a patient with a questionable history of alcohol abuse who has surgery and then becomes delirious on post-op day five… remember, it's a little tougher to keep up with your drinking in the hospital!) Left untreated, delirium tremens has a mortality of 15%, so begin therapy with benzodiazepines promptly. 5) Alcoholic withdrawal seizures are a separate but related phenomenon that usually occur before the onset of DTs. Generalized tonic-clonic seizures usually occur within 48 hours, but can occur as quickly as two hours after an alcoholic's last drink. Again, the treatment of choice is benzodiazepines. Oral phenobarbital (answer B) is inappropriate because of the medication's route – p.o. medications are not the best choice for a semicomatose patient. Phenobarbital is an excellent medication for alcoholic withdrawal seizures or delirium tremens, but is usually reserved for patients who do not respond to benzodiazepines. An MRI of the head and neck (answer C) would help to rule out underlying injuries, and given this patient's history (or his inability to give one), he will need some kind of imaging to rule out a head bleed or injuries to his cervical spine. MRI is not the best choice, though, because it is so much slower than head CT or plain films of the cervical spine. Moreover, before this patient leaves for radiology, he ought to receive some type of treatment for alcohol withdrawal to prevent a seizure while he is being scanned! A lumbar puncture (answer D) may be necessary to evaluate this patient's altered mental status, but it is not the right thing to do right now. Before you do an LP, you must rule out the possibility of an intracranial mass lesion that will cause brain herniation. Radiological imaging or a good neurological examination is needed before LP, but this patient's most pressing need is anticonvulsant therapy. Intravenous somatostatin (answer E) is used in patients with acute upper GI bleeding. Although alcoholics are prone to upper GI bleeding from varices, ulcers, or esophageal rupture, there is no information in the question stem to lead you to that diagnosis.

B. Oral phenobarbital (answer B) is inappropriate because of the medication's route – p.o. medications are not the best choice for a semicomatose patient. Phenobarbital is an excellent medication for alcoholic withdrawal seizures or delirium tremens, but is usually reserved for patients who do not respond to benzodiazepines.

C. An MRI of the head and neck (answer C) would help to rule out underlying injuries, and given this patient's history (or his inability to give one), he will need some kind of imaging to rule out a head bleed or injuries to his cervical spine. MRI is not the best choice, though, because it is so much slower than head CT or plain films of the cervical spine. Moreover, before this patient leaves for radiology, he ought to receive some type of treatment for alcohol withdrawal to prevent a seizure while he is being scanned!

D. A lumbar puncture (answer D) may be necessary to evaluate this patient's altered mental status, but it is not the right thing to do right now. Before you do an LP, you must rule out the possibility of an intracranial mass lesion that will cause brain herniation. Radiological imaging or a good neurological examination is needed before LP, but this patient's most pressing need is anticonvulsant therapy.

E. Intravenous somatostatin (answer E) is used in patients with acute upper GI bleeding. Although alcoholics are prone to upper GI bleeding from varices, ulcers, or esophageal rupture, there is no information in the question stem to lead you to that diagnosis.

This question is not currently linked to the learning objective database.

Question problem?

Question # 90

Select the single best answer to the numbered question.

A 41 year old female presents with menstrual irregularities for the past several months. She also complains of increased hair growth on her face, and notes that at a recent health screening fair, she was told that she had "pre-diabetes." Her only medication is paroxetine. On physical examination, the patient's blood pressure is 160/94, and numerous purple striae are noted on the patient's abdomen. There is increased subcutaneous fat deposition in the skin of the cheeks and in the posterior cervical region. A 24-hour urine collection for cortisol is markedly elevated. Following the administration of high-dose dexamethasone, the patient's cortisol level decreases by 90%. Which of the following is the most likely cause of these findings in this patient?

A. McCune-Albright syndrome
B. Pituitary adenoma
C. Adrenal adenoma or carcinoma
D. Ectopic production of ACTH by a lung tumor
E. Surreptitious use of exogenous glucocorticoids

You did not answer this question.

Explanations:

A. McCune-Albright syndrome (answer A) is a cause of endocrine hyperfunction that usually presents precocious puberty. (This is something to file away for the pediatrics shelf exam – infants with McCune-Albright may develop breast growth or vaginal bleeding by one year of age!) It can cause an ACTH-independent Cushing syndrome, but this usually results in growth failure and hypertension in infancy. An inherited syndrome like this would be exceedingly unlikely to present in middle age.

B. This patient presents with Cushing's syndrome, which can be caused by anything that results in the increased circulation of glucocorticoids. In this case, the most likely cause of the syndrome is a pituitary adenoma that is secreting ACTH (answer B). Some teaching points… 1) You likely already know the cardinal features of Cushing's syndrome, but if not, review them. They include moon facies, a buffalo hump, purple striae, easy bruisibility, signs of androgen excess, menstrual irregularities, proximal muscle wasting, hypertension, glucose intolerance, osteoporosis, recurrent infection, and thromboembolic events. 2) You can confirm the diagnosis of Cushing's syndrome by finding an elevated cortisol on a 24 hour urine collection. This proves that the patient does in fact have increased glucocorticoids – now you've just got to figure out why. 3) The next step of diagnosis is the dexamethasone suppression test, just as was performed in the question stem. If high-dose dexamethasone suppresses the production of cortisol, then the most likely diagnosis is a pituitary adenoma. These adenomas over-produce ACTH, which causes the adrenals to produce too much glucocorticoid. However, because the adenoma still expresses receptors for cortisol, it's still susceptible to feedback inhibition by circulating steroid – it just takes a lot more "feedback" to get an adenoma to stop overproducing ACTH than it does the normal pituitary! Essentially, then, all that you're doing with a dexamethasone suppression test is suppressing the production of ACTH from the pituitary, and then seeing if that reduced ACTH in turn causes reduced cortisol. Thinking about it that way, it's easy to understand why excess cortisol does NOT suppress when it's caused by primary adrenal dysfunction or ectopic production by a tumor. Consider each of those possibilities in turn. If primary adrenal dysfunction is causing Cushing syndrome, then the adrenals are already producing steroids without stimulation by ACTH (since the production of ACTH by the pituitary would be feedback inhibited by the high circulating cortisol levels). You might be able to suppress the pituitary's production of ACTH even further by giving high dose dexamethasone, but the adrenal will still be doing what it wants to. Alternately, if the excess cortisol is caused by ectopic production of ACTH by a tumor, suppressing the pituitary's production of ACTH won't help, because that's not where the bulk of the ACTH is coming from! McCune-Albright syndrome (answer A) is a cause of endocrine hyperfunction that usually presents precocious puberty. (This is something to file away for the pediatrics shelf exam – infants with McCune-Albright may develop breast growth or vaginal bleeding by one year of age!) It can cause an ACTH-independent Cushing syndrome, but this usually results in growth failure and hypertension in infancy. An inherited syndrome like this would be exceedingly unlikely to present in middle age. Adrenal adenomas or carcinomas (answer C) may cause Cushing's syndrome, but would not be suppressed by high-dose dexamethasone for the reasons described above. Ectopic production of ACTH by a lung tumor (answer D) is also a cause of Cushing's syndrome. Although the tumor cells acquire the ability to produce ACTH, they do not have the receptors to respond to feedback from high glucocorticoid levels, leading to overproduction of cortisol. There is no reason to suspect surreptitious use of exogenous glucocorticoids (answer E) in this patient. However, if you take all comers, the most common cause of Cushing's syndrome is exogenous corticosteroid administration.

C. Adrenal adenomas or carcinomas (answer C) may cause Cushing's syndrome, but would not be suppressed by high-dose dexamethasone.

D. Ectopic production of ACTH by a lung tumor (answer D) is also a cause of Cushing's syndrome. Although the tumor cells acquire the ability to produce ACTH, they do not have the receptors to respond to feedback from high glucocorticoid levels, leading to overproduction of cortisol.

E. There is no reason to suspect surreptitious use of exogenous glucocorticoids (answer E) in this patient. However, if you take all comers, the most common cause of Cushing's syndrome is

E. There is no reason to suspect surreptitious use of exogenous glucocorticoids (answer E) in this patient. However, if you take all comers, the most common cause of Cushing's syndrome is exogenous corticosteroid administration.

This question is not currently linked to the learning objective database.

Question problem?

Question # 91

Select the single best answer to the numbered question.

A 25 year old graduate student complains of a "lump" under his clavicle for the past two weeks. He first noticed the lump after being hit in the shoulder during a rugby match. He has otherwise been well, and denies any other symptoms including fever, night sweats, weight loss, or recent infections or illnesses. On physical examination, the patient is comfortable and afebrile. There is a 4 cm, rubbery, nontender supraclavicular lymph node on the left, as well as numerous 1-2 cm enlarged cervical and axillary nodes. The clavicle is nontender, and there is full range of motion at both shoulders. There is no evidence of a draining abscess or resolving cellulitis. Laboratory evaluation shows the following. WBC 8.0 k/mcL (Normal: 4.5-11.0 k/mcL) Differential: 66% neutrophils, 30% lymphocytes, 3% monocytes, 1% eosinophils Hemoglobin 14.3 g/dL (Normal: 13.5-17.5 g/dL) Hematocrit 43% (Normal: 39-49%) Platelets 212 k/mcL (Normal: 150-450 k/mcL) Which of the following is the most likely diagnosis in this patient?

A. Acute myelogenous leukemia
B. Infectious mononucleosis
C. Hodgkin's lymphoma
D. Trauma
E. Felty's syndrome

You did not answer this question.

Default Explanation:

This description of a young, asymptomatic patient with cervical lymphadenopathy is typical for a patient with Hodgkin's lymphoma (answer C). Some teaching points… -Most patients with Hodgkin's lymphoma present with asymptomatic lymphadenopathy (70%) or an incidentally-found widened mediastinum. The presence of symptoms generally indicates a worse prognosis. Typical symptoms include the "B symptoms" of fever, night sweats, and unexplained weight loss. More rarely, patients complain of pruritus (especially after a hot shower) or severe pain following alcohol ingestion. If any of these findings are described on the USMLE, it's almost always a tipoff that the diagnosis is Hodgkin's. -There are several commonly used staging systems for Hodgkin's lymphoma, but the only one that you're likely to be tested on is the simple Ann Arbor system. Under this classification, stage I disease involves only a single lymph node region, stage II disease involves two or more lymph node regions on the same side of the diaphragm, stage III disease involves lymph node regions on both sides of the diaphragm, and stage IV describes disseminated disease. In addition to the stage, the designations "A" and "B" are used to describe the absence (A) or presence (B) of the "B symptoms" described above (so that a patient might be described as having stage II B or stage III A disease). -Demographically,

Hodgkin's disease has a bimodal age distribution, with one peak in the 20s-30s and another after age 50. It is one of a relatively small number of cancers that affects otherwise healthy young adults. Acute myelogenous leukemia (answer A) usually presents with symptoms related to pancytopenia (like easy bruising, anemia, or neutropenia). The CBC should show a high WBC, and microscopic examination should show numerous circulating myeloid blasts. If you suspect this diagnosis, the next step is a bone marrow biopsy. Infectious mononucleosis (answer B) can cause cervical lymph node enlargement, but there are usually also other symptoms like malaise, headache, tonsillitis or pharyngitis, and fever. Additionally, the peripheral blood smear should show lymphocytosis with a preponderance of atypical lymphocytes. It is highly unlikely that a single episode of minor trauma (answer D) would cause the degree of lymphadenopathy seen in this patient. Sometimes patients attribute findings to trauma when in fact the trauma only called their attention to something that was there already; this seems likely in this case. Felty's syndrome (answer E) is one of the USMLE's favorite eponyms. It consists of the triad of seropositive rheumatoid arthritis, splenomegaly, and granulocytopenia.

This question is not currently linked to the learning objective database.

Question problem?

Question # 92

Select the single best answer to the numbered question.

A 31 year old female presents to her physician in January with several days of fever, malaise, and a cough productive of yellow-green sputum. She had been feeling well until four days ago when she developed a sore throat and rhinorrhea; this was soon followed by fever, malaise, and myalgias. Two days ago she began experiencing a hacking cough that has been occasionally productive of purulent sputum. Vital signs include temperature 37.2 C (99.0 F), pulse 80, blood pressure 118/78, respirations 14/min, oxygen saturation 98% on room air. No crackles or rhonchi are appreciated on auscultation of the lungs. Which of the following is the most appropriate initial step in the management of this patient?

A. Azithromycin
B. Ceftriaxone and levofloxacin
C. Salmeterol
D. Amantadine
E. Pseudophedrine and acetaminophen

You did not answer this question.

Default Explanation:

This patient has acute bronchitis, which is inflammation of the tracheobronchial tree that is almost always caused by viruses. Thus, treatment is symptomatic, and agents like pseudophedrine and acetaminophen (answer E) may help the patient feel a little better. Some extra teaching points… -Acute bronchitis is almost always caused by viruses. This explains why multiple studies have shown that patients with acute bronchitis do not benefit from antibiotics. Chronic bronchitis, however, is a different story. Patients with COPD and chronic bronchitis who have an exacerbation SHOULD be empirically treated with antibiotics, because studies have shown a benefit to doing so. -Many patients (and unfortunately many physicians, too) mistakenly believe that yellow or green sputum is an absolute sign of bacterial infection and mandates antibiotic treatment. The yellow-green coloration comes from the myeloperoxidase of neutrophils, so discolored sputum only means that neutrophils are activated – nothing less, nothing more. While neutrophils are the cornerstone of the body's immune response to bacteria, they also mount the initial response to ANY infectious agent, including viruses. -On a similar note, a recent study indicated that calling acute bronchitis a "chest cold" improved patient satisfaction with appropriate antibiotic use. -There is no diagnostic test for acute bronchitis – it's a clinical diagnosis. The key feature is a cough productive of purulent sputum in the setting of other features suggestive of a viral upper respiratory infection. -Obviously, you don't want to miss a diagnosis of pneumonia in a patient who presents with cough and purulent sputum. If a patient has abnormal vital signs or crackles on physical exam, you should order a chest x-ray, but in the absence of these signs, it is unlikely to change your management plan. Azithromycin (answer A) is jokingly referred to by one UVA attending as "our most effective broad-spectrum antiviral," a reference to the fact that so many practicioners prescribe a Z-Pak for the common cold or other self-limited viral infections. However, azithromycin and other macrolides are very useful in the treatment of community-acquired pneumonia because they can target atypical causes of pneumonia (like Mycoplasma) that are insensitive to beta-lactams. Ceftriaxone and levofloxacin (answer B) is an antibiotic regimen that might be used in a hospitalized patient with community-acquired pneumonia. Salmeterol (answer C) is a long-acting beta-2 agonist frequently used in combination with inhaled corticosteroids for the treatment of asthma. Studies show that inhaled beta-agonists are not helpful for patients with bronchitis unless there is evidence of airway obstruction. For patients with dyspnea or wheezing, albuterol or ipratropium can be helpful. Amantadine (answer D) is an effective treatment for influenza A, but only if it is begun within 48 hours of the onset of symptoms.

This question is not currently linked to the learning objective database.

Question problem?

Question # 93

Select the single best answer to the numbered question.

A 38 year old female presents with weakness and tingling in her hands and lower extremities. She was feeling well until yesterday when she noted a "pins-and-needles" sensation in her fingers and toes. She noted weakness in her hands and legs today, and had difficulty holding a pen or walking without dragging her toes on the ground while at work today. Her past medical history includes gastroesophageal reflux and an overnight hospitalization three weeks ago for dehydration following an episode of gastroenteritis. Physical examination shows poor inspiratory effort and diminished breath sounds bilaterally. Deep tendon reflexes cannot be elicited at the ankle, knee, wrist, or elbow, and the patient has 3+ muscle strength distally and 4+ muscle strength proximally. There is a flexor plantar response. Cerebellar testing and cranial nerve testing show no gross deficit. Which of the following is the most likely diagnosis in this patient?

A. Cauda equina syndrome
B. Multiple sclerosis
C. Amyotrophic lateral sclerosis
D. Myasthenia gravis
E. Acute inflammatory demyelinating polyneuropathy

You did not answer this question.

Explanations:

A. Cauda equina syndrome (answer A) is caused by compression of the nerve roots caudal to the level of spinal cord termination (the "horse's tail" of the spinal cord – hence the name). You shouldn't miss this one on a test: patients have a characteristic presentation consisting of back pain, sciatica, "saddle anesthesia," and bowel and bladder incontinence.

B. The diagnosis of multiple sclerosis (answer B) requires multiple neurological deficits separated in time that cannot be explained with a single lesion.

C. Amyotrophic lateral sclerosis (answer C) is Lou Gehrig's disease. Patients usually present with gradual muscle weakness or bulbar palsy, but the key feature that you'll see in USMLE question stems is the simultaneous presence of upper AND lower motor neuron signs. No other neurological disorder (or at least no other disorder that will be tested on the USMLE) will cause both UMN and LMN signs simultaneously.

D. Myasthenia gravis (answer D) occurs when antibodies attack the post-synaptic acetylcholine receptors at the junction between nerve and muscle. Patients may complain of bulbar symptoms (like ptosis, diplopia, blurred vision, difficulty swallowing, or dysarthria) or distal muscle weakness, but the key feature of patients with myasthenia is that their weakness gets worse with repetitive or sustained stimulation. On the USMLE, this clue may be given to you in the physical exam, or the history by describing symptoms that get worse as the day goes on or with use of the affected

muscles.

E. This patient presents with ascending paralysis and loss of deep tendon reflexes following a self-limited gastrointestinal illness a couple of weeks ago. This is a classic presentation of acute inflammatory demyelinating polyneuropathy (answer E), also known as Guillain-Barre syndrome. A few teaching points… 1) The absolutely hallmark sign of Guillain-Barre is the lack of deep tendon reflexes on physical exam. Patients also will give a history (similar to the one here) of progressive, symmetrical, ascending weakness and paralysis. 2) The diagnosis can be made clinically, without any additional studies. However, there are two studies that may be described in question stems. First, if you do an LP, you'll find increased protein but a normal WBC. This is called albuminocytologic dissociation, and presumably occurs due to increased antibodies in the CSF (the same antibodies that were made to fight an earlier infection but are now cross-reacting with the patient's own myelin). Second, EMG studies will show evidence of demyelination. Remember, your nerves are sort of like electrical wiring, with the myelin sheath serving as the insulator. Take the insulator off, and the wire (or nerve) won't conduct as well. 3) Treatment is primarily supportive, but plasma exchange and intravenous immunoglobulin (IVIg) shorten the course of the illness. In general, the disease is self-limiting, and most patients fully recover within a few weeks. 4) The most feared complication of Guillain-Barre is respiratory compromise, which may require intubation and mechanical ventilation. The most important thing that you can do for a patient with Guillain-Barre is monitor their pulmonary function and intervene as necessary. 5) The patient's recent bout with gastroenteritis wasn't just a red herring – it was a clue to the diagnosis. AIDP or Guillain-Barre is commonly preceded by a viral or bacterial infection, and the most common culprit is Campylobacter jejuni (which of course is classically obtained after eating contaminated chicken at a picnic or a pot luck dinner). Cauda equina syndrome (answer A) is caused by compression of the nerve roots caudal to the level of spinal cord termination (the "horse's tail" of the spinal cord – hence the name). You shouldn't miss this one on a test: patients have a characteristic presentation consisting of back pain, sciatica, "saddle anesthesia," and bowel and bladder incontinence. The diagnosis of multiple sclerosis (answer B) requires multiple neurological deficits separated in time that cannot be explained with a single lesion. Amyotrophic lateral sclerosis (answer C) is Lou Gehrig's disease. Patients usually present with gradual muscle weakness or bulbar palsy, but the key feature that you'll see in USMLE question stems is the simultaneous presence of upper AND lower motor neuron signs. No other neurological disorder (or at least no other disorder that will be tested on the USMLE) will cause both UMN and LMN signs simultaneously. Myasthenia gravis (answer D) occurs when antibodies attack the post-synaptic acetylcholine receptors at the junction between nerve and muscle. Patients may complain of bulbar symptoms (like ptosis, diplopia, blurred vision, difficulty swallowing, or dysarthria) or distal muscle weakness, but the key feature of patients with myasthenia is that their weakness gets worse with repetitive or sustained stimulation. On the USMLE, this clue may be given to you in the physical exam, or the history by describing symptoms that get worse as the day goes on or with use of the affected muscles.

This question is not currently linked to the learning objective database.

Question problem?

Question # 94

Select the single best answer to the numbered question.

A 10 year old male with asthma returns to his physician for follow up. Two months ago, his teacher noted him wheezing after playing on the playground, and pulmonary function testing confirmed a diagnosis of asthma. Since that time, he has been using a rescue inhaler approximately one time a week at school to relieve wheezing and chest tightness that seem to occur following strenuous activity. He has also been using his rescue inhaler approximately one time a week at home at night when he awakens with chest tightness or difficulty breathing. Current medications include albuterol and atomoxetine. On physical exam, the patient is comfortable and afebrile. The patient's lungs are clear to auscultation, and no wheezing or prolonged expiration is noted. Which of the following is the most appropriate next step in the management of this patient?

A. Repeat spirometry
B. Cetirizine
C. Inhaled salmeterol
D. Inhaled fluticasone
E. Oral prednisone

You did not answer this question.

Explanations:

A. Performing spirometry (answer A) can establish the diagnosis of asthma by demonstrating reversible airway obstruction. Although in real practice, many patients are simply presumed to have asthma and are treated accordingly, getting pulmonary function tests to confirm the diagnosis is probably the right thing to do. However, once the diagnosis is established, you won't gain much useful information by repeating spirometry again so quickly.

B. Cetirizine (answer B) is a selective H1 antihistamine that is approved for use in children. It might be indicated if the patient had presented with seasonal allergies or allergic rhinitis, but it is not the best choice to control his asthma.

C. Salmeterol (answer C) is a long-acting beta-2 agonist that is used to treat asthma. A combination of fluticasone and salmeterol (Advair) is available, and might be the next step for this patient if his asthma gets worse. However, there is still controversy regarding the appropriate of long acting beta agonists in chronic asthma. Currently all medications containing salmeterol or formoterol are labeled with a "black box" warning noting that they may increase the risk of asthma related death.

D. You should know the appropriate management for both acute and chronic asthma. This question stem describes a patient with mild, persistent asthma – he needs an inhaled steroid like fluticasone (answer D) to control his symptoms. The key to assessing chronic asthma is the "Rule of 2's." If a patient's asthma is controlled, then he or she should require their rescue inhaler less than 2 times per week in the day and less than 2 times per month at night. If a patient's asthma is uncontrolled, you have to keep adding medications in a stepwise fashion until it is controlled. Uncontrolled asthma will lead to a progressive decline in lung function, and potentially unnecessary hospitalizations or even death. If you'd like to review a chart of the stepwise treatment for asthma, here are the recommendations from the National Asthma Education and Presentation Program: http://www.ncbi.nlm.nih.gov/books/bv.fcgi?rid=asthma.figgrp.1090 Performing spirometry (answer A) can establish the diagnosis of asthma by demonstrating reversible airway obstruction. Although in real practice, many patients are simply presumed to have asthma and are treated accordingly, getting pulmonary function tests to confirm the diagnosis is probably the right thing to do. However, once the diagnosis is established, you won't gain much useful information by repeating spirometry again so quickly. Cetirizine (answer B) is a selective H1 antihistamine that is approved for use in children. It might be indicated if the patient had presented with seasonal allergies or allergic rhinitis, but it is not the best choice to control his asthma. Salmeterol (answer C) is a long-acting beta-2 agonist that is used to treat asthma. A combination of fluticasone and salmeterol (Advair) is available, and might be the next step for this patient if his asthma gets worse. However, there is still controversy regarding the appropriate of long acting beta agonists in chronic asthma. Currently all medications containing salmeterol or formoterol are labeled with a "black box" warning noting that they may increase the risk of asthma related death. Oral prednisone (answer E) is used for acute asthma exacerbations, but for chronic asthma, inhaled steroids are bioequivalent and cause fewer systemic side effects.

E. Oral prednisone (answer E) is used for acute asthma exacerbations, but for chronic asthma, inhaled steroids are bioequivalent and cause fewer systemic side effects.

This question is not currently linked to the learning objective database.

Question problem?

Question # 95

Select the single best answer to the numbered question.

A 24 year old student seeks treatment for nasal congestion, sneezing, and a runny nose. His symptoms occur daily for four to six weeks every year in the spring and fall, and are worse when he is outdoors or doing yardwork. Recently, he has been taking over-the-counter pseudophedrine and chlorpheniramine, but these have not significantly improved his symptoms. The patient's family history includes a mother with asthma and a brother with eczema. On physical exam, the conjunctivae are injected, and the patient's palpebrae are swollen. The nasal turbinates are boggy and edematous, and are bluish-gray in color. There is no lymphadenopathy or thyromegaly. Which of the following is the most appropriate step in the management of this patient?

A. Diphenhydramine
B. Fexofenadine
C. Nasal beclomethasone
D. Guaifenasin
E. Measure serum IgE levels

You did not answer this question.

Explanations:

A. Diphenhydramine (answer A) is a first-generation antihistamine. Although efficacious, it may cause sedation or anticholinergic side effects. It is not the best choice for this patient, who has not responded to antihistamines.

B. Like loratadine, desloratadine, and cetirizine, fexofenadine (answer B) is a second-generation or "nonsedating" antihistamine. It is not the best choice for this patient, who has not responded to oral antihistamine therapy.

C. This patient presents with seasonal allergies and allergic rhinosinusitis, and his symptoms have remained uncontrolled despite therapy with decongestants and antihistamines. At this point, treatment with nasal corticosteroids like beclomethasone (answer C) is indicated to help relieve the patient's symptoms. Some teaching points about this case… 1) The single most effective treatment for allergic rhinitis is inhaled nasal corticosteroids – studies have shown them to be more efficacious than oral antihistamines or nasal cromolyn. They can be used as monotherapy, but are more effective when used in combination with other classes of medication. 2) The first-line treatment for mild to moderate allergic rhinitis is decongestants (like pseudophedrine) and antihistamines. If patients have more severe or more persistent symptoms, nasal steroids may be needed. 3) Despite what advertisements for Claritin or Zyrtec might suggest, there is no evidence to suggest that second-generation antihistamines (like loratadine, cetirizine, and fexofenadine) are any more effective at treating allergic rhinitis than first generation antihistamines (like diphenhydramine or chlorpheniramine). Second-generation antihistamines DO cause less sedation and fewer anticholinergic side effects, however. 4) The "allergic triad" consists of eczema, asthma, and allergic rhinitis. A positive family or personal history of any of the three is important when you're considering a diagnosis of any of them. 5) Ultimately, the only true "cure" for patients with allergies is allergen identification and avoidance (if possible), or immunotherapy and desensitization. Think about getting allergen testing if a patient does not respond to medications. Diphenhydramine (answer A) is a first-generation antihistamine. Although efficacious, it may cause sedation or anticholinergic side effects. Like loratadine, desloratadine, and cetirizine, fexofenadine (answer B) is a second-generation or "nonsedating" antihistamine. Guaifenasin (answer D) is a mucolytic agent that is used to loosen secretions and make coughs more productive. It has no role in the treatment of allergic rhinosinusitis. Although many patients with allergic rhinitis have elevated serum IgE levels (answer E), many do not, and many "normal" patients may have an elevated IgE. Thus, this test is neither sensitive nor specific for allergic rhinitis, and is seldom used today. The same is true for ordering a serum eosinophil level, although both tests could potentially be useful in select cases where the diagnosis is in doubt.

D. Guaifenasin (answer D) is a mucolytic agent that is used to loosen secretions and make coughs more productive. It has no role in the treatment of allergic rhinosinusitis.

E. Although many patients with allergic rhinitis have elevated serum IgE levels (answer E), many do not, and many "normal" patients may have an elevated IgE. Thus, this test is neither sensitive nor specific for allergic rhinitis, and is seldom used today. The same is true for ordering a serum eosinophil level, although both tests could potentially be useful in select cases where the diagnosis is in doubt.

This question is not currently linked to the learning objective database.

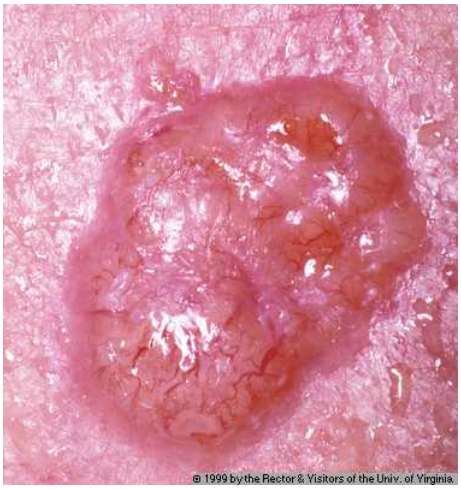
This question is not currently linked to the learning objective database.

Question problem?

Question # 96

Select the single best answer to the numbered question.

At the insistence of his wife, a 71 year old farmer presents to have a “bump” on his shoulder evaluated. The bump has been there for several years, and though it is not painful, it does itch from time to time. The patient does report many years of sun exposure while working on his farm. Physical examination reveals a comfortable, fair-skinned gentleman. On the patient’s left shoulder, there is a smooth, 3cm by 4cm papular lesion with numerous dilated blood vessels throughout. A photo of this lesion is seen here. Which of the following



is the most appropriate therapy for this patient?

A. Podophillin resin
B. Shave biopsy
C. Local excision
D. Topical tacrolimus
E. Excision, lymph node biopsy, and radiation

You did not answer this question.

Explanations:

A. Podophyllin resin (answer A) is an anti-mitotic agent that is used to treat warts, especially genital warts.

B. A shave biopsy (answer B) might be indicated if the diagnosis was in doubt, but at a glance, you can tell that this is a clear-cut basal cell carcinoma. Shaving off a portion of it to send to pathology will only put the patient through two procedures instead of just one, since the tumor will ultimately require removal.

C. This is a basal cell carcinoma, the single most common cancer in humans. Note the smooth, pearly appearance and multiple telangiectasias. Fortunately, basal cell carcinomas rarely metastasize. However, they can grow and become locally destructive (especially in a cosmetically-sensitive location such as the face.) For that reason, the gold standard of therapy is removal of the tumor through some type of local therapy such as cryosurgery, local radiation, topical 5-fluorouracil, or surgical excision (answer C). Skin cancers are commonly tested on the USMLE – usually in a photographic format. You should recognize the key features of squamous cell carcinoma, basal cell carcinoma, melanoma, and actinic keratoses. For a quick photographic overview, try this skin cancer atlas from Loylola University: http://www.meddean.luc.edu/lumen/MedEd/medicine/dermatology/content.htm Podophyllin resin (answer A) is an anti-mitotic agent that is used to treat warts, especially genital warts. A shave biopsy (answer B) might be indicated if the diagnosis was in doubt, but at a glance, you can tell that this is a clear-cut basal cell carcinoma. Shaving off a portion of it to send to pathology will only put the patient through two procedures instead of just one, since the tumor will ultimately require removal. Topical tacrolimus (answer D) is used to treat some cases of psoriasis. Patients with psoriasis will have non-itchy plaques with a “silvery scale” on the elbows, knees, lower back, or scalp. Excision, lymph node biopsy, and radiation (answer E) is a strategy that might be used for a patient with a malignant melanoma. Melanomas are aggressive cancers that metastasize early and often. Patients with anything other than the most minor melanomas will require some steps beyond local excision, including lymph node biopsy, chemotherapy, and radiation.

D. Topical tacrolimus (answer D) is used to treat some cases of psoriasis. Patients with psoriasis will have non-itchy plaques with a “silvery scale” on the elbows, knees, lower back, or scalp.

E. Excision, lymph node biopsy, and radiation (answer E) is a strategy that might be used for a patient with a malignant melanoma. Melanomas are aggressive cancers that metastasize early and often. Patients with anything other than the most minor melanomas will require some steps beyond local excision, including lymph node biopsy, chemotherapy, and radiation.

This question is not currently linked to the learning objective database.

Question problem?

Question # 97

Select the single best answer to the numbered question.

A 10 year old boy returns to the physician for a non-healing rash on his arms, legs, and face. Two weeks ago, he initially developed a small, intensely pruritic area of erythema on his arm that progressed into small fluid-filled blisters. He was initially treated with prednisone for contact dermatitis, but after the rash continued to spread, he was given a prescription for cephalexin. Despite finishing the course of cephalexin, the rash has continued to spread to his legs, neck, and face. The patient’s immunizations are up-to-date, and he has never been hospitalized and takes no chronic medications. On physical examination, the child is comfortable and pleasant, with vital signs including temperature 36.8 C (98.2 F), pulse of 90/min, blood pressure 100/68, and oxygen saturation of 99% on room air. There are numerous linear areas of erythema and excoriation are noted on the patient’s arms, legs, neck, and face. The lesions appear to be in various stages of evolution: some are vesicular, while some are covered with a golden-yellow crust. Gram stain of one of the lesions shows numerous Gram-positive cocci in clumps. Which of the following is the most appropriate treatment for this patient at this time?

A. Trimethoprim-sulfamethoxazole
B. Acyclovir
C. Dicloxacillin
D. Vancomycin
E. Varicella zoster immune globulin (ZVIG)

You did not answer this question.

Explanations:

A. This patient’s history and physical exam findings are classic for impetigo, and the finding of S.aureus on Gram stain merely confirms your clinical suspicion. However, this patient’s infection did not respond to what would seem to be appropriate therapy with cephalosporins. This very strongly suggests that the infection is caused by a resistant strain of S.aureus – most likely community-acquired methicillin-resistant Staphylococcus aureus, or CA-MRSA. CA-MRSA is an increasing problem that has attracted a great deal of media attention lately. Always suspect this diagnosis when there is a history of treatment failure with appropriate beta-lactam or cephalosporin antibiotics. Unlike hospital-associated MRSA, which is generally susceptible only to high-powered antibiotics like vancomycin and linezolid, CA-MRSA strains (fortunately) usually retain susceptibility to clindamycin and trimethoprim-sulfamethoxazole (answer A). Mupirocin ointment is also effective if the patient has only a small patch of impetigo, but topical treatment is impractical for patients with larger areas of infection. For a great review of community-acquired MRSA, try Mayo Clinic Proceedings, September 2005; 80(9): 1201-1208, or check it out online here: http://www.mayoclinicproceedings.com/pdf%2F8009%2F8009crc%2Epdf Acyclovir (answer B) is an antiviral agent for herpesviruses. If you picked this answer, you might have thought that this patient had chickenpox. Indeed, varicella is often described on the USMLE as an itchy rash with multiple lesions in various stages of evolution. You should always treat varicella in an immunocompromised host, because the disease can be life-threatening – however, in a healthy child, the disease is self-limiting and treatment is supportive. Dicloxacillin (answer C) is an anti-staphylococcal penicillin. If this child had normal, garden-variety impetigo, it would be an excellent choice for treatment. The fact that this patient’s infection did not respond to cephalosporin, though, strongly suggests that this infection is CA-MRSA, so beta-lactams won’t help. Vancomycin (answer D) is an effective treatment for both hospital acquired and community acquired strains of MRSA. However, vancomycin is only available in an i.v. form, so giving it to this child will require an inpatient admission. For a patient with stable vitals, this is not the best initial option. Varicella zoster immune globulin or VZIG (answer E) is used for prophylaxis of varicella in susceptible or immune compromised patients. To be effective, it must be given within 96 hours after exposure (i.e., during the viral incubation period). Even if this child had chickenpox, giving VZIG now won’t help him.

B. Acyclovir (answer B) is an antiviral agent for herpesviruses. If you picked this answer, you might have thought that this patient had chickenpox. Indeed, varicella is often described on the USMLE as an itchy rash with multiple lesions in various stages of evolution. You should always treat varicella in an immunocompromised host, because the disease can be life-threatening – however, in a healthy child, the disease is self-limiting and treatment is supportive.

C. Dicloxacillin (answer C) is an anti-staphylococcal penicillin. If this child had normal, garden-variety impetigo, it would be an excellent choice for treatment. The fact that this patient’s infection did not respond to cephalosporin, though, strongly suggests that this infection is CA-MRSA, so beta-lactams won’t help.

D. Vancomycin (answer D) is an effective treatment for both hospital acquired and community acquired strains of MRSA. However, vancomycin is only available in an i.v. form, so giving it to this child will require an inpatient admission. For a patient with stable vitals, this is not the best initial option.

E. Varicella zoster immune globulin or VZIG (answer E) is used for prophylaxis of varicella in susceptible or immune compromised patients. To be effective, it must be given within 96 hours after exposure (i.e., during the viral incubation period). Even if this child had chickenpox, giving VZIG now won't help him.

This question is not currently linked to the learning objective database.

Question problem?

Question # 98

Select the single best answer to the numbered question.

A 43 year old teacher returns to her physician after routine lab evaluation revealed elevated liver transaminases with an ALT of 158 U/L and an AST of 108 U/L. The patient has been in her normal state of health, and has no complaints. Past medical history is significant for diabetes mellitus and hypertension, and current medications include metformin and enalapril. The patient denies foreign travel, blood transfusions, or use of over-the-counter or herbal medications. She is a Jehovah's Witness and does not smoke or use alcohol. Physical examination shows a comfortable, obese female. There is no organomegaly, right upper quadrant or abdominal tenderness, jaundice, scleral icterus, asterixis, or spider angiomata. Laboratory evaluation shows the following: Hepatitis A IgM: negative; Hepatitis A IgG: positive; Hepatitis B surface antigen (HBsAg): negative; Hepatitis B surface antibody (HBsAb): positive; Hepatitis B core antibody (HBcAb): negative; Hepatitis C PCR: negative. Which of the following is the most likely explanation for this patient's elevated liver enzymes?

A. Hepatitis A infection
B. Hepatitis B infection
C. Adverse effect of metformin
D. Fatty infiltration of the liver
E. Alcohol abuse

You did not answer this question.

Explanations:

A. This patient's serologies eliminate the possibility of current hepatitis A (answer A) or hepatitis B infection (answer B). The interpretation of hepatitis serologies is a favorite on Step 2, so don't be intimidated - it's easy to do if you think about it! Hepatitis A is the easiest to interpret: since IgM is the first antibody formed in response to any infection, a positive IgM for hepatitis A represents an acute infection. IgG is formed later, so a positive IgG indicates past infection with hepatitis A. However, unlike hepatitis B and C, hepatitis A only causes acute hepatitis, not chronic disease. Therefore, IgM indicates active, acute hepatitis A, and IgG represents past exposure and immunity, since by the time the body forms IgG to the virus, the infection is long gone! Hepatitis C is also pretty easy to interpret. Hepatitis C only VERY rarely causes an acute hepatitis, so iff the PCR shows the presence of hepatitis C virus, then the patient has chronic hepatitis C infection. Hepatitis B is a little tougher. The most important thing to remember is that the immune system can only form antibodies to something that it has seen before. Think about what each of the antibodies are, and it's easy to figure out a patient's hepatitis B status. The first thing to look at is the hepatitis B surface antigen (HBsAg), which is the actual virus itself. If the HBsAg is positive, then the patient has active hepatitis B infection – now you've just got to figure out if it's acute or chronic. Similarly, if the patient has a negative HBsAg, then they don't currently have hepatitis B, and you need to look at the rest of their serologies to figure out if they're immune or not. The next thing to look at is the surface antibody (HBsAb). If a patient has antibodies to the surface antigen – the outside of the virus – then that patient is immune to hepatitis B infection, because the patient's antibodies bind to the outside of the virus before it has a chance to unleash its genetic material and replicate. When we vaccinate for hepatitis B, we try to stimulate the production of HBsAb, since this is the only antibody that protects the patient from infection. Third, look at the core antibody (HBcAb). If it's positive, then the patient has had an previous infection with hepatitis B virus – because only in the context of an infection will the virus unravel and expose its core antigens. All this may sound complicated, but there are really only four possible permutations of these serologies that you'll see on the USMLE Step 2. (In real life, things are more complicated because of the presence of a "window period" in which results are more difficult to interpret.) Those four possibilities are: 1) A patient who has never had hepatitis B, and has never been vaccinated. Their serologies will ALL be negative, since they've never been exposed to any part of the hepatitis B virus. 2) A patient who has been vaccinated for hepatitis B. This patient will have a positive hepatitis B surface antibody (HBsAb), since this is the antibody that provides protection against HBV, but all of their other serologies will be negative. 3) A patient with past hepatitis B infection who has since cleared it. This patient will have a positive surface antibody AND a positive core antibody, since the only way that the immune system learns what the inside of the hepatitis B virus looks like is by having seen it in the context of an infection. However, a patient with a past infection should NOT have detectable hepatitis B surface antigen, since they've cleared the virus. 4) A patient with chronic hepatitis B infection. This patient will have a positive viral surface antigen and a positive core antibody, but a negative surface antibody. Remember, the surface antibody is the protective one – patients who have chronic HBV never develop that protection (or else they wouldn't have acquired a chronic infection!).

B. This patient's serologies eliminate the possibility of current hepatitis A (answer A) or hepatitis B infection (answer B). The interpretation of hepatitis serologies is a favorite on Step 2, so don't be intimidated - it's easy to do if you think about it! Hepatitis A is the easiest to interpret: since IgM is the first antibody formed in response to any infection, a positive IgM for hepatitis A represents an acute infection. IgG is formed later, so a positive IgG indicates past infection with hepatitis A. However, unlike hepatitis B and C, hepatitis A only causes acute hepatitis, not chronic disease. Therefore, IgM indicates active, acute hepatitis A, and IgG represents past exposure and immunity, since by the time the body forms IgG to the virus, the infection is long gone! Hepatitis C is also pretty easy to interpret. Hepatitis C only VERY rarely causes an acute hepatitis, so iff the PCR shows the presence of hepatitis C virus, then the patient has chronic hepatitis C infection. Hepatitis B is a little tougher. The most important thing to remember is that the immune system can only form antibodies to something that it has seen before. Think about what each of the antibodies are, and it's easy to figure out a patient's hepatitis B status. The first thing to look at is the hepatitis B surface antigen (HBsAg), which is the actual virus itself. If the HBsAg is positive, then the patient has active hepatitis B infection – now you've just got to figure out if it's acute or chronic. Similarly, if the patient has a negative HBsAg, then they don't currently have hepatitis B, and you need to look at the rest of their serologies to figure out if they're immune or not. The next thing to look at is the surface antibody (HBsAb). If a patient has antibodies to the surface antigen – the outside of the virus – then that patient is immune to hepatitis B infection, because the patient's antibodies bind to the outside of the virus before it has a chance to unleash its genetic material and replicate. When we vaccinate for hepatitis B, we try to stimulate the production of HBsAb, since this is the only antibody that protects the patient from infection. Third, look at the core antibody (HBcAb). If it's positive, then the patient has had an previous infection with hepatitis B virus – because only in the context of an infection will the virus unravel and expose its core antigens. All this may sound complicated, but there are really only four possible permutations of these serologies that you'll see on the USMLE Step 2. (In real life, things are more complicated because of the presence of a "window period" in which results are more difficult to interpret.) Those four possibilities are: 1) A patient who has never had hepatitis B, and has never been vaccinated. Their serologies will ALL be negative, since they've never been exposed to any part of the hepatitis B virus. 2) A patient who has been vaccinated for hepatitis B. This patient will have a positive hepatitis B surface antibody (HBsAb), since this is the antibody that provides protection against HBV, but all of their other serologies will be negative. 3) A patient with past hepatitis B infection who has since cleared it. This patient will have a positive surface antibody AND a positive core antibody, since the only way that the immune system learns what the inside of the hepatitis B virus looks like is by having seen it in the context of an infection. However, a patient with a past infection should NOT have detectable hepatitis B surface antigen, since they've cleared the virus. 4) A patient with chronic hepatitis B infection. This patient will have a positive viral surface antigen and a positive core antibody, but a negative surface antibody. Remember, the surface antibody is the protective one – patients who have chronic HBV never develop that protection (or else they wouldn't have acquired a chronic infection!).

C. Because metformin (answer C) is cleared renally, it should not cause increases in LFTs. However, you should never give metformin to a patient with severe pre-existing liver disease because of the increased risk of causing lactic acidosis, the most feared side effect of metformin use.

D. This is a common problem in outpatient medicine: elevated liver enzymes in an asymptomatic patient. Adverse medication effects, infections, autoimmune diseases, and alcohol abuse can all cause elevated LFTs, but those causes are not likely in this patient. In an overweight patient with diabetes and elevated LFTs, the most likely cause – once other things have been ruled out – is very likely to be fatty infiltration of the liver (answer D) or non-alcoholic steatohepatitis (NASH). If you're interested, there is a very helpful article on working up elevated liver enzymes in an asymptomatic patient in the American Family Physician from March 15, 2005. You can see it online at: http://www.aafp.org/afp/20050315/1105.html This patient's serologies eliminate the possibility of current hepatitis A (answer A) or hepatitis B infection (answer B). The interpretation of hepatitis serologies is a favorite on Step 2, so don't be intimidated - it's easy to do if you think about it! Hepatitis A is the easiest to interpret: since IgM is the first antibody formed in response to any infection, a positive IgM for hepatitis A represents an acute infection. IgG is formed later, so a positive IgG indicates past infection with hepatitis A. However, unlike hepatitis B and C, hepatitis A only causes acute hepatitis, not chronic disease. Therefore, IgM indicates active, acute hepatitis A, and IgG represents past exposure and immunity, since by the time the body forms IgG to the virus, the infection is long gone! Hepatitis C is also pretty easy to interpret. Hepatitis C only VERY rarely causes an acute hepatitis, so iff the PCR shows the presence of hepatitis C virus, then the patient has chronic hepatitis C infection. Hepatitis B is a little tougher. The most important thing to remember is that the immune system can only form antibodies to something that it has seen before. Think about what each of the antibodies are, and it's easy to figure out a patient's hepatitis B status. The first thing to look at is the hepatitis B surface antigen (HBsAg), which is the actual virus itself. If the HBsAg is positive, then the patient has active hepatitis B infection – now you've just got to figure out if it's acute or chronic. Similarly, if the patient has a negative HBsAg, then they don't currently have hepatitis B, and you need to look at the rest of their serologies to figure out if they're immune or not. The next thing to look at is the surface antibody (HBsAb). If a patient has antibodies to the surface antigen – the outside of the virus – then that patient is immune to hepatitis B infection, because the patient's antibodies bind to the outside of the virus before it has a chance to unleash its genetic material and replicate. When we vaccinate for hepatitis B, we try to stimulate the production of HBsAb, since this is the only antibody that protects the patient from infection. Third, look at the core antibody (HBcAb). If it's positive, then the patient has had an previous infection with hepatitis B virus – because only in the context of an infection will the virus unravel and expose its core antigens. All this may sound complicated, but there are really only four possible permutations of these serologies that you'll see on the USMLE Step 2. (In real life, things are more complicated because of the presence of a "window period" in which results are more difficult to interpret.) Those four possibilities are: 1) A patient who has never had hepatitis B, and has never been vaccinated. Their serologies will ALL be negative, since they've never been exposed to any part of the hepatitis B virus. 2) A patient who has been vaccinated for hepatitis B. This patient will have a positive hepatitis B surface antibody (HBsAb), since this is the antibody that provides protection against HBV, but all of their other serologies will be negative. 3) A patient with past hepatitis B infection who has since cleared it. This patient will have a positive surface antibody AND a positive core antibody, since the only way that the immune system learns what the inside of the hepatitis B virus looks like is by having seen it in the context of an infection. However, a patient with a past infection should NOT have detectable hepatitis B surface antigen, since they've cleared the virus. 4) A patient with chronic hepatitis B infection. This patient will have a positive viral surface antigen and a positive core antibody, but a negative surface antibody. Remember, the surface antibody is the protective one – patients who have chronic HBV never develop that protection (or else they wouldn't have acquired a chronic infection!). Because metformin (answer C) is cleared renally, it should not cause increases in LFTs. However, you should never give metformin to a patient with severe pre-existing liver disease because of the increased risk of causing lactic acidosis, the most feared side effect of metformin use. Alcohol abuse (answer E) can be hard to rule out in real-life patients, given the natural reluctance to admit excessive alcohol intake. However, on the USMLE, you can trust a patient's account of their substance use. Also, as mentioned previously, alcoholic hepatitis characteristically causes a 2:1 ratio of AST/ALT.

E. Alcohol abuse (answer E) can be hard to rule out in real-life patients, given the natural reluctance to admit excessive alcohol intake. However, on the USMLE, you can trust a patient's account of their substance use. Also, alcoholic hepatitis characteristically causes a 2:1 ratio of AST/ALT.

This question is not currently linked to the learning objective database.

Question problem?

Question # 99

Select the single best answer to the numbered question.

A 25 year old graduate student returns to her physician with complaints of abdominal pain and diarrhea, which have been persistent nearly a year. She continues to experience nearly-daily episodes of postprandial abdominal pain and visible abdominal distension, which are relieved by the passage of stool. During these episodes, her stools are loose and gassy, and while she denies ever passing blood in her stool, she has periodically noticed large quantities of mucus. Workup to this point has been unrevealing, and has included thyroid function tests, colonoscopy, celiac sprue antibodies, and fecal occult blood testing. She denies fevers, weight loss, nausea and vomiting, and reflux symptoms. The patient's

past medical history is significant only for depression, which is well controlled with sertraline. On physical examination, the patient appears healthy. She has normoactive bowel sounds and a nontender abdomen. Hemoccult is negative. Which of the following is the most appropriate next step in the management of this patient?

A. Reassurance, education, and dietary modification
B. Barium enema
C. Culture of the stool for ova and parasites
D. Mesalamine
E. Exploratory laparotomy

You did not answer this question.

Explanations:

A. This patient has irritable bowel syndrome or IBS, which is defined as abdominal pain and altered bowel habits (which can be diarrhea, constipation, increased urgency, etc.) in the absence of any other medical explanation for these symptoms. It is therefore a diagnosis of exclusion – but this patient has undergone a fairly extensive workup already! According to a position statement from the American College of Gastroenterology, unless a patient has certain "alarm symptoms" like hematochezia, a weight loss of >10 lbs, family history of colon cancer, fevers, anemia, or severe diarrhea, testing with flexible sigmoidoscopy, barium enema, colonoscopy, fecal occult blood tests, stool for ova and parasites, and thyroid function tests are NOT recommended! There is no "cure" for IBS. Certain symptomatic treatments can be helpful (such as antispasmodic agents for pain or antidiarrheals for patients with diarrhea-predominant symptoms) but the cornerstones of management remain building a strong therapeutic relationship, providing patient education and setting realistic expectations, managing comorbid illnesses (including depression/anxiety), identifying and avoiding dietary triggers, and increasing dietary fiber (answer A). There are two new, specific pharmacologic treatments for IBS that deserve mention. Alosetron is a 5-HT3 antagonist (similar to ondansetron) that is beneficial in females with diarrhea-predominant IBS. However, it has been associated with ischemic colitis, and its use is currently under strict FDA control. Tegaserod (Zelnorm) is a new (and much-advertised) treatment for women with constipation predominant IBS, and it does appear to produce some modest improvements in symptoms. If you'd like to read more, this article from the American Family Physician reviews the treatment of IBS and also contains some useful charts with diagnostic criteria: http://www.aafp.org/afp/20051215/2501.html A barium enema (answer B) is unlikely to reveal anything pathological in this patient, who recently had a negative colonoscopy. Culturing the stool for ova and parasites (answer C) is also unlikely to be very revealing. Some parasites (like Giardia) can produce a chronic, watery diarrhea, but there should be some clue in the question stem suggesting parasite exposure (recent travel, drinking unpurified water, etc.). Furthermore, this patient's history and symptom complex is very consistent with IBS – no further workup is necessary unless her symptoms change. While mesalamine (answer D) is used in the treatment of inflammatory bowel disease or IBD, it has no role in IBS, and this patient's symptoms are not particularly concerning for either Crohn's or ulcerative colitis. An exploratory laparotomy (answer E) is entirely unnecessary at this point. This patient has IBS, not an acute abdomen.

B. A barium enema (answer B) is unlikely to reveal anything pathological in this patient, who recently had a negative colonoscopy.

C. Culturing the stool for ova and parasites (answer C) is unlikely to be very revealing. Some parasites (like Giardia) can produce a chronic, watery diarrhea, but there should be some clue in the question stem suggesting parasite exposure (recent travel, drinking unpurified water, etc.). Furthermore, this patient's history and symptom complex is very consistent with IBS – no further workup is necessary unless her symptoms change.

D. While mesalamine (answer D) is used in the treatment of inflammatory bowel disease or IBD, this patient's symptoms are not particularly concerning for either Crohn's or ulcerative colitis.

E. An exploratory laparotomy (answer E) is entirely unnecessary at this point. This patient has IBS, not an acute abdomen.

This question is not currently linked to the learning objective database.

Question problem?

Question # 100

Select the single best answer to the numbered question.

A 24 year old female returns to her physician after a routine Pap smear showed atypical squamous cells of undetermined significance. She has no past history of abnormal Pap smears or sexually-transmitted infections, and other testing from her last visit (including beta-hCG and testing for gonorrhea and chlamydia) was negative. She has a lifetime total of four sexual partners, and is currently sexually active with one partner, using only an oral contraceptive pill for birth control. Menarche was at age 11, and menses have been unremarkable. PCR reveals the presence of human papillomavirus DNA, type 18. Which of the following is the most appropriate next step in the management of this patient?

A. Rapid plasma reagin (RPR) testing
B. Begin imiquimod
C. Endometrial biopsy
D. Colposcopy
E. Reassurance and routine follow-up

You did not answer this question.

Explanations:

A. Rapid plasma reagin, or RPR (answer A) is a screening test for syphilis, which is thankfully a rare disease today. While this patient should be offered testing for sexually-transmitted infections, screening for syphilis is only recommended in patients who are pregnant or who are considered high risk (men who have sex with men, commercial sex workers, and incarcerated prisoners). (As a sidenote, antibody tests like the RPR or VDRL are just screening tests for syphilis - a positive result should be followed up with a confirmatory test like darkfield microscopy or FT-ABS. This trick has been known to show up on the USMLE from time to time.)

B. Imiquimod (answer B) is an immunomodulator used to treat genital warts. Because it is a cream that must be applied locally and then washed off, it cannot be used for internal HPV infections. Remember also that visible genital warts (or condyloma acuminate) are most often caused by different types of HPV than those that cause cancer. (Types 6 and 11 are most frequently implicated in genital warts, while 16, 18, and 31 are the usual cancer culprits.)

C. An endometrial biopsy (answer C) is a useful test to evaluate endometrial carcinoma. It might have been indicated if the patient had presented with atypical glandular cells on Pap smear (not squamous cells), or if she were over 35 or had irregular menstrual bleeding.

D. This question involves the management of an abnormal Pap smear. Based on just what makes the Pap smear abnormal – atypical squamous cells, atypical glandular cells, etc. – the patient's management is different. Trying to memorize all of the possibilities will quickly become overwhelming, so the best thing to do is try to remember a few simple principles. 1) Squamous cells come from the ectocervix, while glandular cells come from the uterus or endocervix. 2) The Pap smear is a screening test, not a diagnostic test – it's designed to identify all of the patients who are at risk for cervical cancer. Unless the results come back showing obvious malignancy, a Pap smear won't tell you what exactly is going on inside a patient – you'll need further testing to do that. 3) High grade lesions demand more urgent management than low grade ones. If a patient has a high-grade lesion, they need to go to colposcopy right away. 4) Patients with low grade lesions or atypical cells of unknown significance have an intermediate risk of developing cervical cancer, and they need further testing to risk-stratify them. 5) Cervical cancer is caused by HPV, so HPV testing is usually the best way to help risk stratify patients initially. There are a number of "high risk" strains of HPV, but the most notorious offenders are types 16, 18, and 31. This patient has atypical cells on her Pap and is positive for a worrisome strain of HPV. The atypical cells alone put her at an intermediate risk for cervical cancer, but having an oncogenic strain of HPV bumps her into a higher risk category. She should receive colposcopy (answer D). No one will expect you to memorize the algorithms for working up abnormal Pap smears – any question that you encounter should be answerable just using common sense and clinical judgment. Still, to familiarize yourself with the official guidelines for the initial management of an abnormal Pap smear, go to: http://www.guideline.gov/algorithm/4659/NGC-4659_1.html Rapid plasma reagin, or RPR (answer A) is a screening test for syphilis, which is thankfully a rare disease today. While this patient should be offered testing for sexually-transmitted infections, screening for syphilis is only recommended in patients who are pregnant or who are considered high risk (men who have sex with men, commercial sex workers, and incarcerated prisoners). (As a sidenote, antibody tests like the RPR or VDRL are just screening tests for syphilis - a positive result should be followed up with a confirmatory test like darkfield microscopy or FT-ABS. This trick has been known to show up on the USMLE from time to time.) Imiquimod (answer B) is an immunomodulator used to treat genital warts. Because it is a cream that must be applied locally and then washed off, it cannot be used for internal HPV infections. Remember also that visible genital warts (or condyloma acuminate) are most often caused by different types of HPV than those that cause cancer. (Types 6 and 11 are most frequently implicated in genital warts, while 16, 18, and 31 are the usual cancer culprits.) An endometrial biopsy (answer C) is a useful test to evaluate endometrial carcinoma. It might have been indicated if the patient had presented with atypical glandular cells on Pap smear (not squamous cells), or if she were over 35 or had irregular menstrual bleeding. Reassurance and routine follow up (answer E) is inappropriate. Only a normal Pap smear gets routine follow up – anything else demands a workup of some sort.

E. Reassurance and routine follow up (answer E) is inappropriate. Only a normal Pap smear gets routine follow up – anything else demands a workup of some sort.

This question is not currently linked to the learning objective database.

Question problem?

Question # 101

Select the single best answer to the numbered question.

An otherwise healthy 2 year old male returns for follow-up of bilateral ear effusions. Several months ago, the patient completed a course of amoxicillin for bilateral acute otitis media, but later follow up revealed persistent effusions. At his last visit, audiometry revealed only slight hearing loss bilaterally (<20 db). Today, his mother states that she does feel that he doesn't seem to hear as well as his siblings, though he has been afebrile and has no other symptoms. Physical exam reveals nonerythematous, immobile tympanic membranes with persistent effusions bilaterally. Repeat audiometry reveals a bilateral hearing deficit of 42 decibels. What is the most appropriate next step in the management of this patient?

A. Chlorpheniramine and pseudophedrine
B. Prednisolone
C. Referral for tympanostomy tube placement
D. Cefdinir
E. Follow up in three months

You did not answer this question.

Explanations:

A. Antihistamines and decongestants like chlorpheniramine and pseudophedrine (choice A) have no proven benefit in the treatment of otitis media with effusion and are not recommended.

B. Steroids like prednisolone (choice B) are not recommended for OME treatment. There is little evidence for their efficacy.

C. This question addresses the appropriate management of otitis media with effusion, or OME. This is a common complication of acute otitis media, and is defined as the presence of a middle ear effusion in the absence of signs of inflammation. In most cases, OME will resolve without medical intervention - but since it can cause a conductive hearing loss until then, intervention may be

required to prevent interference with the child's development. Studies have noted that children who spent prolonged time with middle ear effusions have lower scores on tests of speech, language, and cognitive abilities. In general worsening hearing loss, or hearing loss greater than 30-40 dB, should be managed with an ENT consult and tympanostomy tube placement (answer C), and that is the best choice in this case. Less severe cases may be watched expectantly, since the natural history of the disease is to resolve in time. Antihistamines and decongestants like chlorpheniramine and pseudophedrine (choice A) have no proven benefit in the treatment of otitis media with effusion and are not recommended. Steroids like prednisolone (choice B) are not recommended for OME treatment. There is little evidence for their efficacy. Prescribing a course of antibiotics like cefdinir (choice D) is not the best option at this time. Though antibiotics can lead to a small improvement in OME, there is no proven benefit beyond the first month. This patient's problem is not an infection – it's a persistent effusion that is now causing worsening hearing loss. Cefdinir or amoxicillin/clavulanate would be good choices if the patient's acute otitis media had not resolved with amoxicillin, however. Watchful waiting (choice E) is inappropriate. With a child this young, you can't sit around and wait while their hearing worsens – otherwise, they'll miss out on critical speech and language development!

D. Prescribing a course of antibiotics like cefdinir (choice D) is not the best option at this time. Though antibiotics can lead to a small improvement in OME, there is no proven benefit beyond the first month. This patient's problem is not an infection – it's a persistent effusion that is now causing worsening hearing loss. Cefdinir or amoxicillin/clavulanate would be good choices if the patient's acute otitis media had not resolved with amoxicillin, however.

E. Watchful waiting (choice E) is inappropriate. With a child this young, you can't sit around and wait while their hearing worsens – otherwise, they'll miss out on critical speech and language development!

This question is not currently linked to the learning objective database.

Question problem?

Question # 102

Select the single best answer to the numbered question.

A 24 year old woman with a history of recurrent headaches returns for follow up. For the past year and a half, she has experienced severe headaches accompanied by nausea and vomiting several times per week. Her pain is pulsating and throbbing, and typically affects the left temporal region of her head. The headaches last at least several hours, but have lasted as long as three days and have required her to miss several days of work. Past medical history is significant for major depression and dysmenorrhea. Her mother has a history of similar headaches. Several weeks ago, she was seen and given a prescription for oral sumatriptan. Today, she states that she continues to have several headaches each week, just as she did previously, but taking sumatriptan and lying in a dark room helps relieve them within a few hours. Her neurological examination is normal. There is no papilledema. Which of the following is the most appropriate step in the management of this patient?

A. Inhalation of 100% oxygen
B. Head CT with and without contrast
C. Lumbar puncture
D. Amitriptyline
E. Intranasal dihydroergotamine

You did not answer this question.

Explanations:

A. Inhalation of 100% oxygen (answer A) is the most effective abortive therapy for cluster headaches. Although this is not the best treatment for this patient (who does not have cluster headaches and does not need additional abortive therapy even if she did) you should commit this fact to memory, because it has a way of surfacing on the USMLE.

B. Obtaining head CT with and without contrast (answer B) is not indicated in this patient with chronic, stable headaches and no abnormalities on neurological examination. If this patient's headaches were not so typical of migraines, or if her symptoms had changed in intensity or frequency, an imaging study might be indicated, but in the absence of all these things, the American Academy of Neurology does not recommend neuroimaging. You should have a low threshold for obtaining an imaging study in a patient with a severe, acute headache or in a patient with neurological abnormalities, however.

C. A lumbar puncture (answer C) will not likely provide useful information in a patient who has had headaches for a year and a half. Acutely, if a patient describes "the worst headache of their life," you may need an LP to rule out the possibility of a subarachnoid hemorrhage.

D. This patient presents with recurrent migraine headaches. Although sumatriptan seems to be able to abort her headaches once they've started, she is still having several headaches per week, so she is a prime candidate for some type of prophylactic medication. There are a number of choices for migraine prophylaxis – the most frequently used are beta-blockers like propranolol or timolol, anticonvulsants like topiramate or valproic acid, and tricyclic antidepressants like amitriptyline. Given this patient's history of depression, an antidepressant like amitriptyline (answer D) might be the best choice for her. If you'd like to read more about the choice of medication for migraine prophylaxis, try this review from the American Family Physician: http://www.aafp.org/afp/20060101/72.html Inhalation of 100% oxygen (answer A) is the most effective abortive therapy for cluster headaches. Although this is not the best treatment for this patient (who does not have cluster headaches and does not need additional abortive therapy even if she did) you should commit this fact to memory, because it has a way of surfacing on the USMLE. Obtaining head CT with and without contrast (answer B) is not indicated in this patient with chronic, stable headaches and no abnormalities on neurological examination. If this patient's headaches were not so typical of migraines, or if her symptoms had changed in intensity or frequency, an imaging study might be indicated, but in the absence of all these things, the American Academy of Neurology does not recommend neuroimaging. You should have a low threshold for obtaining an imaging study in a patient with a severe, acute headache or in a patient with neurological abnormalities, however. A lumbar puncture (answer C) will not likely provide useful information in a patient who has had headaches for a year and a half. Acutely, if a patient describes "the worst headache of their life," you may need an LP to rule out the possibility of a subarachnoid hemorrhage. Dihydroergotamine (answer E) is an ergot alkaloid that is used as an abortive treatment for migraines. Because it may cause severe side effects such as coronary or cerebral vasoconstriction, it is reserved for patients who do not respond to triptans or NSAIDs as abortive therapy. However, while this patient could potentially benefit from a medication with an intranasal route, her primary issue is her need for prophylactic medication, not better abortive medication.

E. Dihydroergotamine (answer E) is an ergot alkaloid that is used as an abortive treatment for migraines. Because it may cause severe side effects such as coronary or cerebral vasoconstriction, it is reserved for patients who do not respond to triptans or NSAIDs as abortive therapy. However, while this patient could potentially benefit from a medication with an intranasal route, her primary issue is her need for prophylactic medication, not better abortive medication.

This question is not currently linked to the learning objective database.

Question problem?

Question # 103

Select the single best answer to the numbered question.

A 16 year old female presents with abdominal pain and fever. The pain began 12 hours ago, and was initially focused in the umbilical area, but has since shifted to the right lower quadrant. She has also experienced nausea and had one episode of vomiting. She is sexually active with one partner, and her last menstrual period was three weeks ago. She has not eaten anything since yesterday and does not feel hungry. Vital signs include temperature 38.1 C (100.5 F), pulse 96/min, blood pressure 112/70. The patient is lying very still on the examination table with her hips flexed. Her abdominal examination shows voluntary guarding and tenderness to palpation greatest in the right lower quadrant, approximately midway between the umbilicus and the anterior iliac spine. Laboratory analysis shows a white blood count of 14,000. Which of the following is the most appropriate next step in the management of this patient?

A. Diagnostic peritoneal lavage
B. Doxycycline
C. Bolus with 3 L i.v. normal saline (0.9% NaCl)
D. CT of abdomen
E. Urine beta-hCG

You did not answer this question.

Explanations:

A. Diagnostic peritoneal lavage (answer A) is used to evaluate for intra-abdominal bleeding in a patient who is too unstable to go to CT. Its main use is in trauma patients, and while it is effective, it is gradually being replaced by ultrasound (which is better at localizing injuries or detecting retroperitoneal injury). This patient is fairly stable, though, and the most likely diagnosis is appendicitis, not a ruptured spleen or lacerated liver.

B. Starting doxycycline (answer B) is a bad idea before you do a pregnancy test, because tetracycline antibiotics are known to be teratogenic. Furthermore, this patient has appendicitis – she needs surgery, not medical management. (If you suspected that the patient had perforated her bowel, antibiotics might be appropriate, but even then you'd want to select agents with good coverage of bowel flora – maybe something like ciprofloxacin and metronidazole, but not doxycycline.)

C. Giving a bolus of 3 L normal saline (answer C) would definitely be the best step in managing a patient with hypotension or suspected blood loss. Giving this patient i.v. fluid is not a bad idea – it's just not the most important thing right now. (And a 3 L bolus is an awful lot of fluid to give all at once to a patient like this!)

D. A CT of the abdomen (answer D) would be a bad idea at this point – pregnancy is not a diagnosis that you want to make radiographically! Moreover, although a CT can be helpful in evaluating patients with appendicitis (especially by ruling out other causes of abdominal pain), imaging is not absolutely necessary – you can make the diagnosis clinically.

E. This patient presents with a classic case of appendicitis. Because the patient is young and sexually active, though, you should rule out pregnancy with a urine beta-hCG before you do any further studies or go to the O.R. In this case in particular, you want a beta-hCG to rule out an ectopic pregnancy – but the teaching point for this question is more general. Regardless of patient complaint, it's always a good idea to determine if the patient is pregnant before you start a potentially teratogenic medication or send her to surgery or radiology. If you missed this question, learn your lesson now! The USMLE loves questions like these, because the order in which you proceed with a workup IS important. On the boards, and in real life, you don't order tests before you evaluate the ABCs, you don't do an LP before you do a head CT or check for papilledema, and you don't do anything non-emergent to a sexually-active reproductive aged female until you rule out pregnancy. Expect to see these situations on Step 2. Diagnostic peritoneal lavage (answer A) is used to evaluate for intra-abdominal bleeding in a patient who is too unstable to go to CT. Its main use is in trauma patients, and while it is effective, it is gradually being replaced by ultrasound (which is better at localizing injuries or detecting retroperitoneal injury). This patient is fairly stable, though, and the most likely diagnosis is appendicitis, not a ruptured spleen or lacerated liver. Starting doxycycline (answer B) is a bad idea before you do a pregnancy test, because tetracycline antibiotics are known to be teratogenic. Furthermore, this patient has appendicitis – she needs surgery, not medical management. (If you suspected that the patient had perforated her bowel, antibiotics might be appropriate, but even then you'd want to select agents with good coverage of bowel flora – not doxycycline.) Giving a bolus of 3 L normal saline (answer C) would definitely be the best step in managing a patient with hypotension or suspected blood loss. Giving this patient i.v. fluid is not a bad idea – it's just not the most important thing right now. (And a 3 L bolus is an awful lot of fluid to give all at once to a patient like this!) A CT of the abdomen (answer D) would be a bad idea at this point – pregnancy is not a diagnosis that you want to make radiographically! Moreover, although a CT can be helpful in evaluating patients with appendicitis (especially by ruling out other causes of abdominal pain), imaging is not absolutely necessary – you can make the diagnosis clinically.

you can make the diagnosis clinically.

This question is not currently linked to the learning objective database.

Question problem?

Question # 104

Select the single best answer to the numbered question.

A 44 year old female presents with epigastric abdominal pain, nausea, and vomiting. Her symptoms began after eating fried chicken and ice cream at a carnival two nights ago, and have progressively worsened. Physical examination shows an obese, afebrile female, with pulse 110 and blood pressure 96/60. There is mild, diffuse tenderness to palpation throughout the abdomen and worst at the epigastrium. Bowel sounds are absent, and there is no rebound tenderness or guarding. Laboratory evaluation shows the following: WBC 12,000/mm3; Hct 49%; AST 18; ALT 25; Lipase 320; Total bilirubin 2.0. Right upper quadrant ultrasound reveals numerous gallstones, but no gallbladder wall thickening or pericholecystic fluid collection. Which of the following is the most likely diagnosis in this patient?

A. Acute pancreatitis
B. Biliary colic
C. Cholecystitis
D. Choledocholithiasis
E. Ovarian torsion

You did not answer this question.

Explanations:

A. This patient's symptoms and physical exam suggest a diagnosis of acute pancreatitis (answer A), and the elevated lipase level confirms your suspicions. The RUQ ultrasound showing gallstones identifies the etiology of her pancreatitis. A few teaching points from this case… 1) The two most common etiologies of acute pancreatitis in the U.S. are alcohol abuse (which causes thickening of pancreatic secretions and contraction of the sphincter of Oddi) and gallstones (which cause obstruction of the pancreatic duct). There are a number of less frequent but still important causes like drugs (especially thiazides and steroids), hyperlipidemia, and instrumentation (like ERCP). 2) The treatment for pancreatitis is supportive care, designed to correct fluid and electrolyte disturbances. This is a very serious disease, with an overall mortality of around 10%! Maybe the best way to think about pancreatitis is to imagine that it's a huge retroperitoneal burn. Thanks to third-spacing of fluids, patients can become dehydrated very quickly. 3) The diagnosis of pancreatitis is usually made very easily, by the finding of an elevated amylase or lipase level. This single test is sensitive and specific enough to rule out or rule in a diagnosis of pancreatitis. Biliary colic (answer B) is pain caused by the temporary obstruction of the cystic duct by a gallstone. The pain is typically post-prandial, but resolves within a few hours. If the stone becomes fixed, the patient will develop cholecystitis. Cholecystitis (answer C) is the inflammation of the gallbladder caused by a fixed obstruction of the cystic duct by a gallstone. The key word here is "inflammation", and you need to see signs of it on ultrasound (like wall thickening, distension, or pericholecystic fluid collection) in order to make the diagnosis. Lots of people have gallstones, but only 20% of them ever develop symptoms. Choledocholithiasis (answer D) occurs when a gallstone becomes lodged in the common bile duct. This causes more than just inflammation of the gallbladder – because the liver is obstructed, too, there will be elevation of liver transaminases. Ovarian torsion (answer E) is always on the differential for a female with acute abdominal pain. However, it usually causes unilateral lower abdominal pain, and it typically occurs in a patient who has pathologically enlarged ovaries (such as an ovarian tumor).

B. Biliary colic (answer B) is pain caused by the temporary obstruction of the cystic duct by a gallstone. The pain is typically post-prandial, but resolves within a few hours. If the stone becomes fixed, the patient will develop cholecystitis.

C. Cholecystitis (answer C) is the inflammation of the gallbladder caused by a fixed obstruction of the cystic duct by a gallstone. The key word here is "inflammation", and you need to see signs of it on ultrasound (like wall thickening, distension, or pericholecystic fluid collection) in order to make the diagnosis. Lots of people have gallstones, but only 20% of them ever develop symptoms.

D. Choledocholithiasis (answer D) occurs when a gallstone becomes lodged in the common bile duct. This causes more than just inflammation of the gallbladder – because the liver is obstructed, too, there will be elevation of liver transaminases.

E. Ovarian torsion (answer E) is always on the differential for a female with acute abdominal pain. However, it usually causes unilateral lower abdominal pain, and it typically occurs in a patient who has pathologically enlarged ovaries (such as an ovarian tumor).

This question is not currently linked to the learning objective database.

Question problem?

Question # 105

Select the single best answer to the numbered question.

A 58 year old male with a history of diabetes mellitus and coronary artery disease presents with failure to achieve or sustain an erection. This problem has been persistent for the past year, and has been causing a great deal of stress in his marriage. When questioned regarding nocturnal erections, he states that he cannot recall the last time that he awakened with an erection. Previously, this had been a typical occurrence. Current medications include atorvastatin, glyburide, metformin, isosorbide dinitrate, and metoprolol. Physical examination reveals normal external genitalia. There is diminished sensation to pinprick in the distal lower extremities bilaterally. Which of the following is the most appropriate initial step in the management of this patient?

A. Sertraline
B. Yohimbine
C. Alprostadil
D. Tadalafil
E. Reassurance

You did not answer this question.

Explanations:

A. Sertraline (answer A) is an SSRI used most frequently for patients with depression or anxiety. However, because its side effects include mild erectile dysfunction and anorgasmia, studies have shown sertraline and other SSRIs to be useful in some men with premature ejaculation.

B. Yohimbine (answer B) is an alpha-2 blocker that was widely used for erectile dysfunction before more modern treatments became available, but there remains little evidence supporting its efficacy. So far, the only studies to show a benefit for yohimbine over placebo have been in men with psychogenic ED.

C. This patient presents with a common problem in outpatient medicine: erectile dysfunction (ED). First-line therapy for such patients is usually a phosphodiesterase inhibitor like sildenafil, vardenafil, or tadalafil (answer D) because these medications are both efficacious and easy to use. They do, however, have some side effects associated with their use, the most serious of which is the risk of hypotension and syncope when given in combination with nitrates. (In fact, if a patient has taken sildenafil and then has a myocardial infarction, current recommendations state that nitrates should not be given for 24 hours!) For this patient, then, the best choice is an injectable prostaglandin analogue like alprostadil (answer C). Although very effective, many men cannot tolerate a penile injection; for such patients, an intra-urethral version of the drug is available, or other treatments such as vacuum devices might be suggested. A few other teaching points for this case… 1) The cause of this patient's ED is clear: it's due to diabetic neuropathy or vascular damage or some combination of the two. However, in other patients, it may be difficult to tease apart organic vs. psychological causes of ED. The best way to do this is by asking about nocturnal penile tumescence. If a patient awakens with an erection, then the "machinery" (nerves, vasculature, etc.) needed to obtain an erection is intact, and the cause of the patient's ED is overwhelmingly likely to be psychological. 2) As many as 50% of diabetic males will develop impotence within six years of the onset of diabetes. Often, impotence can be the first sign of diabetic neuropathy. Sertraline (answer A) is an SSRI used most frequently for patients with depression or anxiety. However, because its side effects include mild erectile dysfunction and anorgasmia, studies have shown sertraline and other SSRIs to be useful in some men with premature ejaculation. Yohimbine (answer B) is an alpha-2 blocker that was widely used for erectile dysfunction before more modern treatments became available, but there remains little evidence supporting its efficacy. So far, the only studies to show a benefit for yohimbine over placebo have been in men with psychogenic ED. Reassurance (answer E) is inappropriate. There are treatments available for ED, and given the stress that this patient's disorder is causing in his marriage, these treatments should at least be offered to him.

D. This patient presents with a common problem in outpatient medicine: erectile dysfunction (ED). First-line therapy for such patients is usually a phosphodiesterase inhibitor like sildenafil, vardenafil, or tadalafil (answer D) because these medications are both efficacious and easy to use. They do, however, have some side effects associated with their use, the most serious of which is the risk of hypotension and syncope when given in combination with nitrates. (In fact, if a patient has taken sildenafil and then has a myocardial infarction, current recommendations state that nitrates should not be given for 24 hours!) For this patient, then, the best choice is an injectable prostaglandin analogue like alprostadil (answer C). Although very effective, many men cannot tolerate a penile injection; for such patients, an intra-urethral version of the drug is available, or other treatments such as vacuum devices might be suggested.

E. Reassurance (answer E) is inappropriate. There are treatments available for ED, and given the stress that this patient's disorder is causing in his marriage, these treatments should at least be offered to him.

This question is not currently linked to the learning objective database.

Question problem?

Question # 106

Select the single best answer to the numbered question.

A 23 year old woman presents with a two week history of bloody discharge from her right breast. Family history is significant for a 58 year old aunt who was recently diagnosed with breast cancer. On physical examination, the right breast is slightly tender to palpation in the area of the nipple. There are no masses, and the skin shows no dimpling, retractions, or inflammatory changes. There is no axillary adenopathy. Urine beta-hCG is negative. Which of the following is the most likely diagnosis in this patient?

A. Intraductal papilloma
B. Infiltrating ductal carcinoma
C. Acute mastitis
D. Paget's disease of the breast
E. Fibroadenoma

You did not answer this question.

Explanations:

A. Most questions on the USMLE test your ability to reason through a clinical case and arrive at the correct answer by using just a touch of knowledge and a lot of clinical judgment and deductive reasoning. Other questions test more isolated facts, and this question is one of these. Commit this fact to memory: the most common cause of isolated bloody nipple discharge in a healthy young female is an intraductal papilloma (answer A). Versions of this question are common, and in recent years one or another has shown up on the surgery, OB/GYN, and family medicine shelf exams! Infiltrating ductal carcinomas (answer B) can also cause bloody nipple discharge. (After all, bleeding from the nipple simply means that there is a friable lesion of some sort in the mammary duct.) In this patient, though, the risk for cancer is low. Cancer is extremely rare in women this young, and remains fairly rare until age 40. For women under 39, the incidence of breast cancer is less than 1 in 239; from age 40-59, that jumps to 1 in 25. It is also worth pointing out that this patient's family history for breast cancer is not particularly worrisome – breast cancer is, after all, such a common cancer that many women will have a relative who is affected. However, patients who have a first degree relative (mother, sister) who develops cancer before the age of 40 need to be screened aggressively (and potentially tested for BRCA1 and BRCA2 mutations). Acute mastitis (answer C) is commonly tested on the USMLE. Patients are typically breastfeeding mothers who develop fever and a hard, red, tender area of the breast. The most common causative organism is S. aureus, so treatment is usually with dicloxacillin, cephalexin, or amoxicillin/clavulanate. Paget's disease of the breast (answer D) is another favorite of the USMLE, and presents with a scaly, eczematous lesion on the nipple or breast. There may be inflammatory skin changes caused by the underlying carcinoma that lead to the often-described "peau d'orange" appearance. Nipple discharge is not a common feature, but women may give a history of bra-staining from ulcerated, raw, or vesicular lesions. A fibroadenoma (answer E) is the most common cause of a breast mass in a young healthy patient. The masses tend to be firm, rubbery, and well-circumscribed.

B. Infiltrating ductal carcinomas (answer B) can also cause bloody nipple discharge. (After all, bleeding from the nipple simply means that there is a friable lesion of some sort in the mammary duct.) In this patient, though, the risk for cancer is low. Cancer is extremely rare in women this young, and remains fairly rare until age 40. For women under 39, the incidence of breast cancer is less than 1 in 239; from age 40-59, that jumps to 1 in 25. It is also worth pointing out that this patient's family history for breast cancer is not particularly worrisome – breast cancer is, after all, such a common cancer that many women will have a relative who is affected. However, patients who have a first degree relative (mother, sister) who develops cancer before the age of 40 need to be screened aggressively (and potentially tested for BRCA1 and BRCA2 mutations).

C. Acute mastitis (answer C) is commonly tested on the USMLE. Patients are typically breastfeeding mothers who develop fever and a hard, red, tender area of the breast. The most common causative organism is S. aureus, so treatment is usually with dicloxacillin, cephalexin, or amoxicillin/clavulanate.

D. Paget's disease of the breast (answer D) is another favorite of the USMLE, and presents with a scaly, eczematous lesion on the nipple or breast. There may be inflammatory skin changes caused by the underlying carcinoma that lead to the often-described "peau d'orange" appearance. Nipple discharge is not a common feature, but women may give a history of bra-staining from ulcerated, raw, or vesicular lesions.

E. A fibroadenoma (answer E) is the most common cause of a breast mass in a young healthy patient. The masses tend to be firm, rubbery, and well-circumscribed.

This question is not currently linked to the learning objective database.

Question problem?

Question # 107

Select the single best answer to the numbered question.

A 73 year old male is brought to the physician by his wife, who is concerned about a rapid functional decline over the past several months. Her husband used to manage the family's finances, but he has become increasingly forgetful, and now is unable to perform this task or even to remember the last few words of a conversation. Over this time, he has also developed difficulty walking, and recently has become incontinent of urine. Mini-mental state examination shows impaired short term memory. Thyroid function tests and serum B12 levels are normal. Which of the following is the most likely finding on CT imaging of the head?

A. Gross atrophy of the caudate nucleus
B. Biconvex, extra-axial area of hemorrhage that does not cross suture lines
C. Multiple ring-enhancing lesions throughout the brain
D. Cortical atrophy and ventricular enlargement
E. Ventricular enlargement without cerebral atrophy

You did not answer this question.

Explanations:

A. Gross atrophy of the caudate nucleus (answer A) is seen in patients with Huntington's disease, a favorite topic on the USMLE. It's a trinucleotide repeat disorder that's inherited in an autosomal dominant fashion, so there should be a positive family history (or perhaps on the USMLE, a patient who was orphaned and does not know their family history). Patients present in their 30s or 40s with chorea, depression, and cognitive decline.

B. A biconvex, extra-axial area of hemorrhage that does not cross suture lines (answer B) is a description of an epidural hematoma. Caused by the tearing of the middle meningeal artery, epidural hematomas present rapidly – not subacutely, like this patient did. Although fewer than 20% of patients actually have it, a classic clue to the diagnosis of an epidural hematoma on the USMLE is a history of a "lucid interval" following the initial recovery from a blow to the head and preceding the later neurological decline. A subdural hematoma (caused by bleeding from a bridging vein) CAN present more subacutely, especially in the elderly, and patients may not recall any initial trauma.

C. Multiple ring enhancing lesions throughout the brain (answer C) is the hallmark of cerebral toxoplasmosis, which affects HIV-positive or immunocompromised patients. You may encounter questions on the internal medicine shelf regarding the workup or diagnosis or ring-enhancing lesions in patients with HIV. Radiographically, you can't tell the difference between the ring-enhancing lesion of toxoplasmosis and the other major cause of a ring-enhancing lesion in an HIV positive patient: primary CNS lymphoma. However, when multiple lesions are present, the diagnosis is more likely to be toxoplasmosis.

D. Cortical atrophy and ventricular enlargement (answer D) is seen in Alzheimer's dementia. The primary process is the loss of cortical neurons, which leads to the loss of brain mass and the subsequent development of enlarged ventricles (leading to "hydrocephalus ex vacuo"). Again, the key feature that distinguishes NPH from other causes of dementia is the rapid development of urinary incontinence and gait instability.

E. This patient presents with the classic triad of normal pressure hydrocephalus: dementia, gait disturbance, and urinary incontinence. A CT in this patient would likely show ventricular enlargement (hydrocephalus) WITHOUT cerebral atrophy (answer E). A few teaching points for this case… 1) Patients with normal pressure hydrocephalus (NPH) can present subacutely, just like a patient with Alzheimer's disease. However, although patients with Alzheimer's disease can develop gait disturbances and incontinence, these occur fairly late in the course of the disease, once there has been a longer period of decreased cognitive functioning. In patients with NPH, although memory loss may be noticed first, it is quickly accompanied by the other symptoms of the triad. 2) The most common cause of dementia in the elderly is Alzheimer's disease. However, don't assume this diagnosis! Both on the USMLE and in the clinic, when a patient presents with dementia, the most important initial step is to test for a correctable "medical" cause of dementia like B12/folate deficiency or hypothyroidism. 3) One of the best ways to confirm a diagnosis of NPH is to remove a large quantity of CSF via lumbar puncture and then see if the patient clinically improves. (In particular, you're looking for a near-immediate improvement in gait.) Because this simulates a surgical shunting procedure, this test may also help predict whether the patient will respond to surgical therapy. Gross atrophy of the caudate nucleus (answer A) is seen in patients with Huntington's disease, a favorite topic on the USMLE. It's a trinucleotide repeat disorder that's inherited in an autosomal dominant fashion, so there should be a positive family history (or perhaps on the USMLE, a patient who was orphaned and does not know their family history). Patients present in their 30s or 40s with chorea, depression, and cognitive decline. A biconvex, extra-axial area of hemorrhage that does not cross suture lines (answer B) is a description of an epidural hematoma. Caused by the tearing of the middle meningeal artery, epidural hematomas present rapidly – not subacutely, like this patient did. Although fewer than 20% of patients actually have it, a classic clue to the diagnosis of an epidural hematoma on the USMLE is a history of a "lucid interval" following the initial recovery from a blow to the head and preceding the later neurological decline. A subdural hematoma (caused by bleeding from a bridging vein) CAN present more subacutely, especially in the elderly, and patients may not recall any initial trauma. Multiple ring enhancing lesions throughout the brain (answer C) is the hallmark of cerebral toxoplasmosis, which affects HIV-positive or immunocompromised patients. You may encounter questions on the internal medicine shelf regarding the workup or diagnosis or ring-enhancing lesions in patients with HIV. Radiographically, you can't tell the difference between the ring-enhancing lesion of toxoplasmosis and the other major cause of a ring-enhancing lesion in an HIV positive patient: primary CNS lymphoma. However, when multiple lesions are present, the diagnosis is more likely to be toxoplasmosis. Cortical atrophy and ventricular enlargement (answer D) is seen in Alzheimer's dementia. The primary process is the loss of cortical neurons, which leads to the loss of brain mass and the subsequent development of enlarged ventricles (leading to "hydrocephalus ex vacuo"). Again, the key feature that distinguishes NPH from other causes of dementia is the rapid development of urinary incontinence and gait instability.

This question is not currently linked to the learning objective database.

Question problem?

Question # 108

Select the single best answer to the numbered question.

A 14 year old boy with a history of insulin-dependent diabetes mellitus is brought to the clinic by his mother, who is concerned about worsening lethargy over the past few days. Current medications include glargine and lispro insulin, and the patient does his own injections and blood glucose monitoring. Review of symptoms is positive for polyuria, polydipsia, and generalized weakness. Vital signs include temperature 36.9 C (98.4 F), pulse 120, blood pressure 94/52, and respiratory rate 24/min. On physical examination, a fruity odor is noted on the patient's breath. The patient's skin and mucous membranes are dry, and there is decreased skin turgor. Which of the following sets of values would be most likely found on this patient's arterial blood gas (ABG)?

A. pH = 7.11, pCO2 = 73, PaO2 = 54, HCO3- = 28
B. pH = 7.20, pCO2 = 20, PaO2 = 98, HCO3- = 13
C. pH = 7.34, pCO2 = 60, PaO2 = 60, HCO3- = 31
D. pH = 7.48, pCO2 = 45, PaO2 = 98, HCO3- = 30
E. pH = 7.56, pCO2 = 20, PaO2 = 95, HCO3- = 24

You did not answer this question.

Explanations:

A. Answer A represents a severe acute respiratory acidosis. The primary problem is the retention of CO2. Since the kidneys have not yet had time to compensate, the bicarbonate is in the normal range. Patients with acute respiratory acidosis usually have some problem that impairs their ability to get rid of CO2 – like airway obstruction, intoxication with sedatives or narcotics, or head trauma leading to an impaired respiratory drive.

B. This patient has diabetic ketoacidosis (DKA), which you should suspect in any patient who presents with symptoms such as these – especially when they are known to be diabetic. DKA is a classic example of metabolic acidosis, so you're looking for the answer choice with a low pH, decreased bicarbonate, and decreased pCO2, which is answer B. Quickly assessing a patient's ABG is an important skill on the wards and on the USMLE. Fortunately, on Step 2, you will not be asked to perform advanced calculations like the Winter's formula or tested on complicated mixed

primary disorders. You WILL have to be able to look at an ABG and quickly determine if the patient is acidemic or alkalemic, whether their primary problem is respiratory or metabolic, and whether there has been compensation or not – so you need to be absolutely comfortable doing those things. A quick tutorial from the University of Connecticut on the interpretation of ABGs can be found online at: http://fitsweb.uchc.edu/student/selectives/TimurGraham/Stepwise_approach.html Answer A represents a severe acute respiratory acidosis. The primary problem is the retention of CO2. Since the kidneys have not yet had time to compensate, the bicarbonate is in the normal range. Patients with acute respiratory acidosis usually have some problem that impairs their ability to get rid of CO2 – like airway obstruction, intoxication with sedatives or narcotics, or head trauma leading to an impaired respiratory drive. Answer C represents a chronic respiratory acidosis. The kidneys have had a chance to compensate, so the bicarbonate is elevated and the pH is near normal. This is the type of situation you might see in a patient with COPD who retains CO2. Answer D is a compensated metabolic alkalosis. The primary problem is either the loss of H+ (such as from vomiting) or the gain or retention of excess HCO3- (such as from a contraction alkalosis). The lungs retain CO2 in an effort to decrease the pH. Answer E shows an acute respiratory alkalosis. The patient is breathing too rapidly, leading to the loss of CO2 and an alkalemia. Since the bicarbonate is normal, you know that this is acute – the kidneys have not had a chance to compensate yet. You might see an ABG like this one in a patient who is having a panic attack or a mild-to-moderate asthma attack.

C. Answer C represents a chronic respiratory acidosis. The kidneys have had a chance to compensate, so the bicarbonate is elevated and the pH is near normal. This is the type of situation you might see in a patient with COPD who retains CO2.

D. Answer D is a compensated metabolic alkalosis. The primary problem is either the loss of H+ (such as from vomiting) or the gain or retention of excess HCO3- (such as from a contraction alkalosis). The lungs retain CO2 in an effort to decrease the pH.

E. Answer E shows an acute respiratory alkalosis. The patient is breathing too rapidly, leading to the loss of CO2 and an alkalemia. Since the bicarbonate is normal, you know that this is acute – the kidneys have not had a chance to compensate yet. You might see an ABG like this one in a patient who is having a panic attack or a mild-to-moderate asthma attack.

This question is not currently linked to the learning objective database.

Question problem?

Question # 109

Select the single best answer to the numbered question.

A 23 year old G2P2 presents on postpartum day 10 with fever and a tender left breast. Her symptoms began two days ago, and have been accompanied by myalgias, chills, and malaise. She has been breastfeeding her new daughter, and reports that this has been going well, and that her child has appeared healthy since birth. Vital signs include temperature 38.4 C (101.1 F), pulse 90, and blood pressure 112/68. On physical examination, the patient's left breast is erythematous and engorged, and very tender to palpation. No masses or areas of fluctuance are appreciated. The right breast is unremarkable. Which of the following is the most appropriate initial step in the management of this patient?

A. Incision and drainage
B. Mammography
C. Tetracycline
D. Dicloxacillin
E. Cephalexin and temporary cessation of breastfeeding

You did not answer this question.

Explanations:

A. Incision and drainage (answer A) is the appropriate treatment for a breast abscess, which is a common complication of mastitis. It is not needed at this time, though, for two reasons. First, there was no sign of abscess on physical exam (no mass or fluctuance). Second, and more important, it takes time for bacteria to wall themselves off and form an abscess. Two days is not long enough. If this patient's symptoms persist, though, she should be re-evaluated and perhaps receive an ultrasound to search for an abscess.

B. Mammography (answer B) is useful in older women as a screening or diagnostic test for breast cancer. This patient has mastitis, though, and radiographs will not show you anything useful. If you're concerned about a possible abscess, the test of choice would be ultrasound.

C. Tetracycline antibiotics (answer C) are not the best choice. Not only do they have limited efficacy for S. aureus, but they also are contraindicated for use in children younger than eight years of age because they can cause discoloration of hypoplasia of the tooth enamel. As a general rule, don't give any drug to a pregnant woman or a breast feeding mother unless it's okay if the baby gets dosed, too.

D. This patient has acute mastitis, a common complication of breastfeeding. The causative organism is usually S. aureus, but strep and E. coli may occasionally be the culprits. Whatever the infectious organism is, it comes from the breastfeeding infant's mouth or throat, so there is no reason to tell the mother to stop breastfeeding. (In fact, continuing to breast feed lessens the risk of abscess formation!) Appropriate antibiotics include the anti-staphylococcal penicillins like dicloxacillin (answer D) and cloxacillin, beta-lactams with beta-lactamase inhibitors like amoxicillin/clavulanate, and cephalosporins like cephalexin. Local measures like heat or ice packs, analgesics, and breast support will also help relieve symptoms. Incision and drainage (answer A) is the appropriate treatment for a breast abscess, which is a common complication of mastitis. It is not needed at this time, though, for two reasons. First, there was no sign of abscess on physical exam (no mass or fluctuance). Second, and more important, it takes time for bacteria to wall themselves off and form an abscess. Two days is not long enough. If this patient's symptoms persist, though, she should be re-evaluated and perhaps receive an ultrasound to search for an abscess. Mammography (answer B) is useful in older women as a screening or diagnostic test for breast cancer. This patient has mastitis, though, and radiographs will not show you anything useful. If you're concerned about a possible abscess, the test of choice would be ultrasound. Tetracycline antibiotics (answer C) are not the best choice. Not only do they have limited efficacy for S. aureus, but they also are contraindicated for use in children younger than eight years of age because they can cause discoloration of hypoplasia of the tooth enamel. As a general rule, don't give any drug to a pregnant woman or a breast feeding mother unless it's okay if the baby gets dosed, too. Cephalexin (answer E) would be an appropriate antibiotic choice, but since the infectious organism came from the baby initially, there is no need to tell the mother to stop breast feeding.

E. Cephalexin (answer E) would be an appropriate antibiotic choice, but since the infectious organism came from the baby initially, there is no need to tell the mother to stop breast feeding.

This question is not currently linked to the learning objective database.

Question problem?

Question # 110

Select the single best answer to the numbered question.

A 6 year old boy from Ghana presents with chest and abdominal pain for the past day. His pain began suddenly while at rest last night, is a 9/10 in intensity and unrelieved by ibuprofen, and is localized mainly to his epigastrium and left upper quadrant, back, and chest. Past medical history is unremarkable, and immunizations are up-to-date. On physical examination, the patient is afebrile and normotensive but tachycardic. Lungs are clear to auscultation, and there is moderate tenderness to palpation in the left upper quadrant. Laboratory evaluation shows a white blood count of 11,000 and a hematocrit of 31%. Chest x-ray shows no atelectasis or infiltrate, and abdominal x-ray shows no free air and moderate enlargement of the spenic silhouette. The patient is given i.v. fluids, ceftriaxone, and morphine. Which of the following is the most appropriate next step in the management of this patient?

A. ELISA for HIV-1 antibodies
B. Hemoglobin electrophoresis
C. CT of chest
D. Vancomycin
E. Hydroxyurea

You did not answer this question.

Explanations:

A. ELISA for HIV-1 antibodies (answer A) is not the best choice – this patient is not presenting with an opportunistic infection or risk factors that would suggest HIV as the cause of his symptoms.

B. Like cystic fibrosis, sickle cell disease is an illness that can present at a number of different times and in a number of different ways in children. Patients may present with asymptomatic anemia, stroke, priapism, failure to thrive, aplastic anemia (especially following a parvovirus B19 infection), or recurrent bacterial infections, just to name a few. Overall, perhaps the most common presentation is the one seen by this patient: pain and anemia, or an acute painful crisis. To confirm the diagnosis of sickle cell disease, the best test is hemoglobin electrophoresis (answer B). A few other teaching points… 1) Today, newborn screening for sickle cell disease is almost universal - so most children with the disease get picked up early. The fact that this child was born outside of the United States is an important clue, since it explains why he was able to "escape" an earlier diagnosis. 2) Most children with sickle cell disease present earlier than this one. Levels of fetal hemoglobin begin to decline in most children after about six months, so the most common time for an initial presentation is between 6 months and 1 year of age. However, many children will have a persistence of fetal hemoglobin until they are much older, and it is not too uncommon to have children present at even 10 or 12 years old! Children with less severe phenotypes of sickle cell disease may also present later. 3) Patients with sickle cell disease and a parvovirus B19 infection may present with an aplastic crisis. This is a serious condition that is a favorite of USMLE question-writers. 4) Another classic USMLE presentation is a patient with sickle cell disease and osteomyelitis. The most common causative organism is Salmonella (while normal patients with osteomyelitis tend to be infected with organisms like S. aureus). 5) One of the most serious complications of sickle cell disease is the acute chest syndrome, which consists of chest pain, tachypnea, leukocytosis, and pulmonary infiltrates on CXR. ELISA for HIV-1 antibodies (answer A) is not the best choice – this patient is not presenting with an opportunistic infection or risk factors that would suggest HIV as the cause of his symptoms. A chest CT (answer C) is not likely to help you with diagnosis or management, given that this patient's paucity of pulmonary symptoms and negative chest x-ray. Vancomycin (answer D) is not needed at this time. Children with sickle cell disease are often treated empically with ceftriaxone or a similar drug because their splenic dysfunction leads to susceptibility to encapsulated bacteria like S. pneumoniae, N. meningitides, and H. influenza. Unless you have a specific reason to suspect another pathogen (like MRSA) that would not be covered by a third generation cephalosporin, vancomycin is not needed. Hydroxyurea (answer E) is a commonly-used treatment for patients with SS sickle cell disease. It stimulates the production of fetal hemoglobin (Hgb F), which is resistant to sickling. Before you start therapy, though, it would be prudent to confirm the diagnosis!

C. A chest CT (answer C) is not likely to help you with diagnosis or management, given that this patient's paucity of pulmonary symptoms and negative chest x-ray.

D. Vancomycin (answer D) is not needed at this time. Children with sickle cell disease are often treated empically with ceftriaxone or a similar drug because their splenic dysfunction leads to susceptibility to encapsulated bacteria like S. pneumoniae, N. meningitides, and H. influenza. Unless you have a specific reason to suspect another pathogen (like MRSA) that would not be covered by a third generation cephalosporin, vancomycin is not needed.

E. Hydroxyurea (answer E) is a commonly-used treatment for patients with SS sickle cell disease. It stimulates the production of fetal hemoglobin (Hgb F), which is resistant to sickling. Before you start therapy, though, it would be prudent to confirm the diagnosis!

This question is not currently linked to the learning objective database.

Question problem?

Question problem?

Question # 111

Select the single best answer to the numbered question.

A 9 year old African-American female is brought to the physician by her mother, who is concerned about a “lump” in the patient’s left breast. She noted this lump two weeks ago, and is concerned because the patient’s grandmother was recently diagnosed with breast cancer. The child is otherwise well, and has not yet had onset of menses. On physical examination, the left breast and areola are enlarged and are Tanner stage 3. There is a small tender mass beneath the areola on the left. The right breast is flat and prepubertal. There is no axillary adenopathy. The external genitalia are grossly normal, and are covered with fine velus hair. Which of the following is the most appropriate next step in the management of this patient?

A. Reassurance and anticipatory guidance
B. Transdermal ethinyl estradiol
C. Measurement of serum luteinizing hormone (LH) level
D. Mammography
E. Open biopsy of breast

You did not answer this question.

Explanations:

A. Pathologic breast diseases are extremely rare in childhood and adolescence, while the unilateral onset of breast development is both common and commonly-tested (some version of this question has appeared on both the pediatrics and the family medicine shelf exams in recent memory!). Breast development often (if not usually!) begins asymmetrically, and breasts can differ by as much as two Tanner stages before such development is considered abnormal. This patient’s mother should be reassured that her daughter is normal, and counseled regarding upcoming pubertal changes (answer A). Three additional teaching points… 1) The normal sequence of pubertal development in females is thelarche (breast budding), pubarche/adrenarche (development of pubic and axillary hair), growth spurt, and menarche. 2) Precocious puberty should be evaluated if you see sexual changes in girls younger than 7 or 8 years old or boys younger than 8 or 9. 3) Another commonly encountered issue on the USMLE involves the development of gynecomastia in pubertal boys. Again, this is a normal variant – it occurs in almost half of all teenage boys at least temporarily, and usually resolves on its own within 6 to 18 months. Unless there is reason to suspect a more serious underlying disorder (such as a feminizing testicular tumor or hypogonadism), the best thing to do is patient reassurance. Transdermal ethinyl estradiol (answer B) is a treatment for delayed puberty in girls, while testosterone may be used for boys. Measurement of serum luteinizing hormone (LH) level (answer C) is useful in distinguishing “central” causes of precocious puberty from GnRH independent causes of precocious puberty: in the former, levels of LH should be high, while in the latter, they are low or normal. Neither this patient’s age nor her presentation are concerning for anything other than normal development, though. Mammography (answer D) is actually contraindicated in the workup of a breast mass in a child because of the extremely low incidence of malignancy, the poor quality images due to breast density, and the risk of causing cancers due to radiation exposure. If you need imaging, ultrasound is the best study to obtain. An open biopsy of the breast (answer E) is definitely not indicated now – the incidence of cancer is extremely low in children and adolescents, but even if it weren’t, it would be prudent to pursue some imaging studies first before proceeding with such an invasive test.

B. Transdermal ethinyl estradiol (answer B) is a treatment for delayed puberty in girls, while testosterone may be used for boys.

C. Measurement of serum luteinizing hormone (LH) level (answer C) is useful in distinguishing “central” causes of precocious puberty from GnRH independent causes of precocious puberty: in the former, levels of LH should be high, while in the latter, they are low or normal. Neither this patient’s age nor her presentation are concerning for anything other than normal development, though.

D. Mammography (answer D) is actually contraindicated in the workup of a breast mass in a child because of the extremely low incidence of malignancy, the poor quality images due to breast density, and the risk of causing cancers due to radiation exposure. If you need imaging, ultrasound is the best study to obtain.

E. An open biopsy of the breast (answer E) is definitely not indicated now – the incidence of cancer is extremely low in children and adolescents, but even if it weren’t, it would be prudent to pursue some imaging studies first before proceeding with such an invasive test.

This question is not currently linked to the learning objective database.

Question problem?

Question # 112

Select the single best answer to the numbered question.

A 4 year old Hispanic child is brought to his pediatrician after he developed swelling of his eyes and ankles over the past week. Temperature is 37.4 C (99.3 F), pulse is 120/min, blood pressure 104/61, and respirations 28/min. In addition to periorbital swelling and 2+ edema of the ankles, he has a protuberant abdomen with a positive fluid wave. Laboratory evaluation shows BUN 7 mg/dL, creatinine 0.4 mg/dL, albumin 1.4 g/dL, and cholesterol 498 mg/dL. Urinalysis shows no blood and 4+ protein. Which of the following is the most likely associated finding in this patient’s disorder?

A. "Apple green" birefringence under polarized light with Congo red stain
B. Fusion of epithelial foot processes on electron microscopy
C. Low complement C3 and increased anti-streptolysin O titer
D. Presence of cytoplasmic anti-neutrophil antibody (ANCA) staining
E. Granular deposits of IgG and C3 in the basement membrane

You did not answer this question.

Default Explanation:

This is a typical case of minimal change disease, the most common cause of the nephrotic syndrome in a young child. Note that all four key features of the nephrotic syndrome are present (proteinuria, edema, hypoalbuminemia, and hyperlipidemia) and that there is no hematuria, no elevation in blood pressure, and no impairment of renal function. Minimal change disease is so often the cause of nephrotic syndrome in children that empiric treatment with steroids is typically begun without need to confirm the diagnosis with biopsy. Although minimal change disease produces no changes on plain light microscopy, electron microscopy will show effacement or fusion of epithelial foot processes (answer B). This is a common trick of the USMLE: you’ll be asked to make a diagnosis and then extend your knowledge one step further to answer a question about treatment or associated findings. The remaining answers are characteristic findings of other renal diseases. “Apple green” birefringence under polarized light with Congo red stain (answer A) is the key diagnostic feature of amyloidosis. This disease is caused by the deposition of proteins into the extracellular space, and is commonly associated with multiple myeloma or chronic inflammatory diseases like tuberculosis or rheumatoid arthritis. Patients usually present with progressive renal failure. Increased anti-streptolysin O titer and decreased C3 (answer C) is seen in acute post-streptococcal glomerulonephritis. While this disease does most often affect children, there should be hematuria and a history of a Group A strep skin or throat infection. The presence of cytoplasmic anti-neutrophil antibody staining, or c-ANCA (answer D) indicates Wegener’s granulomatosis, which is a multisystem disease that presents with upper and lower respiratory symptoms and nephritic syndrome. The presentation is not as acute as in this stem, and would include hematuria and red cell casts. Granular deposits of C3 and IgG in the basement membrane (answer E) is the “spike and dome” pattern seen in membranous nephropathy, which is the most common cause of nephrotic syndrome in adults. In some cases, it is associated with hepatitis B infection or treatment of RA with gold.

This question is not currently linked to the learning objective database.

Question problem?

Question # 113

Select the single best answer to the numbered question.

A 57 year old female with hypertension presents for scheduled follow-up. On physical examination, the patient’s blood pressure is 128/80, but a new mass is palpated on the patient’s left thyroid lobe. The mass is firm, smooth, and nontender, and measures 2cm by 1cm. There is no cervical lymphadenopathy or lid lag, and the patient has no history of head or neck radiation or thyroid disease. Laboratory evaluation shows TSH of 1.9 mU/L and calcium of 9.9 mg/dL. Which of the following is the most appropriate next step in the management of this patient?

A. Reassurance
B. Radionuclide thyroid scan
C. Fine needle aspiration
D. Begin propylthiouracil
E. Total thyroidectomy

You did not answer this question.

Explanations:

A. Reassurance (answer A) is inappropriate. Although the great majority of patients with thyroid nodules do not have thyroid cancers, some do. Before you reassure the patient that her nodule is benign, you need to confirm that it is.

B. A radionuclide thyroid scan (answer B) involves giving radioactive iodine to a patient, which quickly gets concentrated in active thyroid tissue. This allows you to see so-called “hot” nodules that are hyperfunctioning and taking up excess iodine, as well as “warm” nodules (that take up iodine just like the surrounding normal thyroid tissue) and hypofunctioning “cold” nodules that do not take up iodine. If this patient were clinically hyperthyroid, this would be the correct answer, since you’d need to determine if the hyperthyroidism was caused by a hyperfunctioning adenoma, or if the patient had background hyperthyroidism and just happened to have a thyroid nodule, too. In a euthyroid patient, however, this test is unlikely to give you any additional information – the patient’s nodule is very likely a “cold” nodule, or else she would have clinical and lab evidence of hyperthyroidism.

C. This patient presents with a common incidental finding: a thyroid nodule. Perhaps as many as 5-10% of the population has a thyroid nodule, and of these, 5-10% will have a thyroid malignancy, while the rest will have benign cysts, adenomas, or multinodular goiters Your major objective, then, in working up a patient with a thyroid nodule is to rule out thyroid cancer. The first step is to check a TSH and determine if the patient is hypo-, hyper-, or euthyroid. Patients who are hyperthyroid likely have a hyperfunctioning adenoma, which is very unlikely to be malignant, but requires treatment to prevent thyrotoxicosis. If the patient is euthyroid – like the patient in this stem – you need to get some tissue so that you can distinguish between benign nodules and malignant ones (neither of which are usually hormonally active). The best way to get tissue is with a fine needle aspiration (answer C). Three additional teaching points… 1) There’s a review of the management of thyroid nodules in the American Family Physician that has a nice algorithm for the workup of thyroid nodules. Once you get beyond the initial step or two, there are a lot of different possibilities, and it may help to look at the graphic to see them all at [illegible]

and it may help to look at the graphic to sort them all out. You can read the article online at: http://www.aafp.org/afp/20050201/559.html. 2) This may seem like an obvious point, but the single best test to determine if a patient has normal thyroid function or not is the TSH. If the TSH is within the normal range, then that patient is euthyroid – period. (If the TSH is low or high, you may have to do some interpretation. Most likely, the problem is with the thyroid, but abnormal levels can also result from disease in the pituitary or hypothalamus.) You may encounter questions on the USMLE that ask you to interpret lots of different thyroid function tests: free T4, T3, reverse T3, thyroid binding globulin, etc. Don't get confused! Regardless of the other values, if the TSH is normal, the patient is euthyroid. 3) There's a reason that this question mentions the patient's calcium level. About 5% of all patients with thyroid carcinoma have medullary carcinoma, or cancer of the C-cells that produce calcitonin. Among this group, many patients will have Multiple Endocrine Neoplasia type II, which also includes pheochromocytomas (a cause of hypertension). MEN is rare, but it's something to keep in the back of your mind when you see real patients, and especially when you encounter presentations like this one on the USMLE. Reassurance (answer A) is inappropriate. Although the great majority of patients with thyroid nodules do not have thyroid cancers, some do. Before you reassure the patient that her nodule is benign, you need to confirm that it is. A radionuclide thyroid scan (answer B) involves giving radioactive iodine to a patient, which quickly gets concentrated in active thyroid tissue. This allows you to see so-called "hot" nodules that are hyperfunctioning and taking up excess iodine, as well as "warm" nodules (that take up iodine just like the surrounding normal thyroid tissue) and hypofunctioning "cold" nodules that do not take up iodine. If this patient were clinically hyperthyroid, this would be the correct answer, since you'd need to determine if the hyperthyroidism was caused by a hyperfunctioning adenoma, or if the patient had background hyperthyroidism and just happened to have a thyroid nodule, too. In a euthyroid patient, however, this test is unlikely to give you any additional information – the patient's nodule is very likely a "cold" nodule, or else she would have clinical and lab evidence of hyperthyroidism. Propylthiouracil (answer D) is a drug used to treat hyperthyroidism. It blocks the production of thyroid hormones and prevents the conversion of T4 to T3. This patient is euthyroid, however, so treatment with a medication like this would be inappropriate. A total thyroidectomy (answer E) is the ultimate treatment for a patient with a proven thyroid malignancy. However, since this is a risky surgery (damage to the recurrent laryngeal nerves or superior laryngeal nerves is not uncommon) that leaves the patient with a lifelong requirement for thyroid hormone replacement, you need to be sure that the patient has a malignancy first.

D. Propylthiouracil (answer D) is a drug used to treat hyperthyroidism. It blocks the production of thyroid hormones and prevents the conversion of T4 to T3. This patient is euthyroid, however, so treatment with a medication like this would be inappropriate.

E. A total thyroidectomy (answer E) is the ultimate treatment for a patient with a proven thyroid malignancy. However, since this is a risky surgery (damage to the recurrent laryngeal nerves or superior laryngeal nerves is not uncommon) that leaves the patient with a lifelong requirement for thyroid hormone replacement, you need to be sure that the patient has a malignancy first.

This question is not currently linked to the learning objective database.

Question problem?

Question # 114

Select the single best answer to the numbered question.

A 13 year old, 121 lb (55 kg) male presents with nausea and periumbilical abdominal pain and is diagnosed with appendicitis. Following an uncomplicated laparoscopic appendectomy, the patient is comfortable and stable, with vital signs in the normal range. The patient is not yet eating. Laboratory analysis shows electrolytes within normal limits and creatinine at pre-operative baseline. Which of the following is the most appropriate maintenance fluid and infusion rate for this patient?

A. Normal saline (0.9% NaCl) + 20 mEq KCl at 70 mL/h
B. Ringer's lactate at 70 mL/h
C. Normal saline (0.9% NaCl) at 95 mL/h
D. 5% dextrose and ¼ normal saline (0.225% NaCl) + 20 mEq KCl at 95 mL/h
E. 5% dextrose and ¼ normal saline (0.225% NaCl) + 20 mEq KCl at 120 ml/h

You did not answer this question.

Explanations:

A. Because normal saline (answers A and C) is isotonic, it tends to stay in the intravascular compartment a little better instead of immediately redistributing to tissues. For this reason, it's commonly used for fluid resuscitation – after all, if you're trying to bump someone's blood pressure, the fluid needs to stay in the blood vessels. Using normal saline for long periods of time can cause problems, though, which is why it's not used as a maintenance fluid. It contains sodium and chloride at concentrations of 154 mEq/L, but as you'll recall, the body's preferred concentration of ions in body fluids is a sodium of around 140 mEq/L and chloride of around 100 mEq/L. If you give normal saline to a patient as a maintenance fluid, over time, their serum levels of Na+ and Cl- will begin to approximate the 154 mEq/L of the fluid that you're pumping into them! The biggest risk is that you'll induce a hyperchloremic metabolic acidosis by infusing large volumes of normal saline.

B. Compared to normal saline, lactated Ringer's solution (answer B) is also isotonic, but more closely resembles the ionic content of body fluids: it contains potassium, calcium, and (as the name indicates) lactate. Because lactate is metabolized to bicarbonate by the liver, giving LR can help buffer an acidosis if present. (This is in stark contrast to normal saline, which can actually create a hyperchloremic metabolic acidosis if it's given in large volumes - this is one reason why LR is the surgeon's favorite i.v. fluid.) Remember that LR is the only standard i.v. fluid that contains potassium, so it's the only fluid order that you wouldn't need to tack on the "+20 mEq KCl" to the end of.

C. Because normal saline (answers A and C) is isotonic, it tends to stay in the intravascular compartment a little better instead of immediately redistributing to tissues. For this reason, it's commonly used for fluid resuscitation – after all, if you're trying to bump someone's blood pressure, the fluid needs to stay in the blood vessels. Using normal saline for long periods of time can cause problems, though, which is why it's not used as a maintenance fluid. It contains sodium and chloride at concentrations of 154 mEq/L, but as you'll recall, the body's preferred concentration of ions in body fluids is a sodium of around 140 mEq/L and chloride of around 100 mEq/L. If you give normal saline to a patient as a maintenance fluid, over time, their serum levels of Na+ and Cl- will begin to approximate the 154 mEq/L of the fluid that you're pumping into them! The biggest risk is that you'll induce a hyperchloremic metabolic acidosis by infusing large volumes of normal saline.

D. This question asks you to perform two tasks – first, you must determine the appropriate intravenous solution, and second, you must determine how quickly you want to give it. Let's start with the first task: deciding which fluid to give. For maintenance fluids, you want to give the patient something that will replenish their daily losses of both water and electrolytes – most importantly sodium and potassium. Patients who are otherwise normal need only a small amount of sodium and potassium to replace the amount lost in the urine, sweat, and tears. In general, you'll need 3 mEq of Na+ and 2 mEq of Cl- for every 100 mL of fluid given. Remembering that normal saline has sodium and chloride at a concentration of 154 mEq/L, you can do some calculations and figure out the patient's exact electrolyte requirement and pick your fluid accordingly. Alternately, you can just take our word for it that one quarter normal saline with 20mEq KCl is the most commonly available solution that comes closest to the exact calculation, and thus is the most commonly used maintenance fluid for normal patients. Most of the time, you'll also choose to use a dextrose containing fluid as well – either 5% dextrose (D5) or 10% dextrose (D10) – to give the patient a little extra nutrition, especially in the immediate postoperative period. (If you'd like more detailed information about the other intravenous fluids listed, keep reading to the bottom of the page or click on the incorrect answers. Otherwise, keep going.) So now that you've decided which fluid to use, you have to figure out the rate - obviously, a larger patient needs a greater quantity of fluid in 24 h than a smaller one! The easiest way to calculate a maintenance fluid rate is to use the so-called 4-2-1 rule. For the first 10kg of a patient's weight, they need 4 mL/kg/h; for the second 10 kg, they need 2 mL/kg/h; and for each kg above 20 kg, they need 1 mL/kg/h. This patient weighs 55 kg, so for the first 10 kg of his body weight, he needs 40 mL/h. For his second 10 kg, he needs an additional 20mL/h. Finally, he needs 35 mL/h for his additional 35 kg over 20 kg (35+20 = 55). Add them all up, and you get 40 mL/h + 20 mL/h + 35 mL/h = 95 mL/h. Therefore, the correct answer to the question is answer D, 5% dextrose and ¼ normal saline at 95 mL/h. One other teaching point may be helpful, since questions asking you to calculate fluid requirements are so common on the USMLE. If you need a maintenance fluid rate, you use the 4-2-1 rule, as described above. However, if you need a total daily fluid requirement, you can either multiply your rate by 24 h, or use the 100-50-20 rule. Similar to the 4-2-1 rule, a patient needs 100 mL/kg for their first 10 kg, 50 mL/kg for their second 10 kg, and 20 mL/kg for each kg thereafter. Because normal saline (answers A and C) is isotonic, it tends to stay in the intravascular compartment a little better instead of immediately redistributing to tissues. For this reason, it's commonly used for fluid resuscitation – after all, if you're trying to bump someone's blood pressure, the fluid needs to stay in the blood vessels. Using normal saline for long periods of time can cause problems, though, which is why it's not used as a maintenance fluid. It contains sodium and chloride at concentrations of 154 mEq/L, but as you'll recall, the body's preferred concentration of ions in body fluids is a sodium of around 140 mEq/L and chloride of around 100 mEq/L. If you give normal saline to a patient as a maintenance fluid, over time, their serum levels of Na+ and Cl- will begin to approximate the 154 mEq/L of the fluid that you're pumping into them! The biggest risk is that you'll induce a hyperchloremic metabolic acidosis by infusing large volumes of normal saline. Compared to normal saline, lactated Ringer's solution (answer B) is also isotonic, but more closely resembles the ionic content of body fluids: it contains potassium, calcium, and (as the name indicates) lactate. Because lactate is metabolized to bicarbonate by the liver, giving LR can help buffer an acidosis if present. (This is in stark contrast to normal saline, which can actually create a hyperchloremic metabolic acidosis if it's given in large volumes - this is one reason why LR is the surgeon's favorite i.v. fluid.) Remember that LR is the only standard i.v. fluid that contains potassium, so it's the only fluid order that you wouldn't need to tack on the "+20 mEq KCl" to the end of. Quarter-normal saline with dextrose and 20 mEq of KCl (answer E) is indeed the appropriate i.v. fluid for this patient – but giving it at a rate of 120 mL/h is a bit much.

E. Quarter-normal saline with dextrose and 20 mEq of KCl (answer E) is indeed the appropriate i.v. fluid for this patient – but giving it at a rate of 120 mL/h is a bit much.

This question is not currently linked to the learning objective database.

Question problem?

Question # 115

Select the single best answer to the numbered question.

A 27 year old G2P2A0 at six weeks postpartum presents with depressed mood, dry skin, and problems breast feeding. Though she was initially able to breastfeed without difficulty, she has been unable to produce adequate lactation to feed her daughter, and lately her milk has completely "dried up." Her most recent pregnancy was uneventful, but delivery of her healthy female infant was complicated by severe uterine atony requiring multiple blood transfusions and treatment with prostaglandins. On physical examination, the patient has a blood pressure of 90/64. Skin tenting and fine wrinkles are noted around the eyes and mouth. There are diminished deep tendon reflexes with a prolonged relaxation phase. Laboratory evaluation is notable for a hemoglobin of 10.5 g/dL, thyroid stimulating hormone of 0.1 U/mL, and prolactin of 2 ng/mL (normal 0-20 ng/mL). Which of the following is the most likely diagnosis in this patient?

A. Sheehan syndrome
B. Grave's disease
C. Uterine rupture
D. Hashimoto thyroiditis
E. Pituitary prolactinoma

You did not answer this question.

Explanations:

A. This patient has Sheehan syndrome, hypopituitarism caused by postpartum infarction of the pituitary. You can almost guarantee that some presentation of Sheehan syndrome will come up on the OB/GYN shelf exam, so it's one of the eponyms that you should commit to memory when you're studying for the USMLE. Just four quick teaching points about this case… 1) Pregnancy leads to hypertrophy of the pituitary to up to twice its normal size – so following acute hypovolemia or blood loss, ischemic necrosis can develop, especially in the anterior pituitary, which is more removed from the arterial blood supply. Patients will present with signs and symptoms resulting from the loss of all of the anterior pituitary hormones: hypothyroidism from loss of TSH, adrenal insufficiency from loss of ACTH, lactation failure from loss of prolactin, and amenorrhea and sexual dysfunction from the lack of FSH and LH. 2) One potentially tricky thing about this case was the low TSH

level, which we normally assume means that a patient is HYPERthyroid. That's because most of the time, thyroid disorders are caused by a problem with the thyroid itself – either it's making too little hormone or too much, and the pituitary tries to speed it up or slow it down by making more or less TSH. If the pituitary itself fails, then the situation becomes a little more complicated. The bottom line is, you can't assume that a low TSH always means hyperthyroidism or that a high TSH always means hypothyroidism – look at the whole clinical picture. 3) Although Sheehan syndrome and pan-hypopituitarism classically occur in postpartum females for the reasons described above, it can occur in other patients. Most notably, patients with clotting disorders or sickle cell disease may occasionally infarct their pituitary, so don't feel like the disease only occurs in pregnant females – if the clinical picture fits, go with it. 4) Whenever you read a description of a patient with decreased DTRs with a prolonged relaxation phase, you are reading a description of hypothyroidism. This is a classic finding that you should commit to memory. Grave's disease (answer B) is the most common cause of hyperthyroidism in adults in the US. It is caused by circulating autoantibodies that activate the thyroid gland (unlike the destructive autoantibodies that cause hypothyroidism in Hashimoto thyroiditis). Clinically, the patient will present with signs of hyperthyroidism and a low TSH. This patient's low TSH is due to pituitary dysfunction, though – not a primary problem with her thyroid. Uterine rupture (answer C) causes catastrophic bleeding at the time of delivery. It most commonly occurs in women who have had previous Caesarean sections. Caused by autoimmune destruction of the thyroid cells, Hashimoto thyroiditis (answer D) is the most common cause of hypothyroidism in the United States. Clinically, the patient will have signs of hypothyroidism, but since the thyroid gland itself is malfunctioning, the level of TSH should be HIGH. You should consider a pituitary prolactinoma (answer E) if you have a patient who presents with galactorrhea – not difficulty breastfeeding. The medical treatment for prolactinomas is a commonly tested fact on the USMLE – the mainstay of therapy is dopamine agonists like bromocriptine.

B. Grave's disease (answer B) is the most common cause of hyperthyroidism in adults in the US. It is caused by circulating autoantibodies that activate the thyroid gland (unlike the destructive autoantibodies that cause hypothyroidism in Hashimoto thyroiditis). Clinically, the patient will present with signs of hyperthyroidism and a low TSH. This patient's low TSH is due to pituitary dysfunction, though – not a primary problem with her thyroid.

C. Uterine rupture (answer C) causes catastrophic bleeding at the time of delivery. It most commonly occurs in women who have had previous Caesarean sections.

D. Caused by autoimmune destruction of the thyroid cells, Hashimoto thyroiditis (answer D) is the most common cause of hypothyroidism in the United States. Clinically, the patient will have signs of hypothyroidism, but since the thyroid gland itself is malfunctioning, the level of TSH should be HIGH.

E. You should consider a pituitary prolactinoma (answer E) if you have a patient who presents with galactorrhea – not difficulty breastfeeding. The medical treatment for prolactinomas is a commonly tested fact on the USMLE – the mainstay of therapy is dopamine agonists like bromocriptine.

This question is not currently linked to the learning objective database.

Question problem?

Question # 116

Select the single best answer to the numbered question.

A 12 month old male is brought to the physician for a well-child checkup. He has been healthy, and his mother's only particular concern is that he has grown increasingly uneasy around any adults other than her. The child eats three meals a day, and is able to feed himself small finger foods. He can pull to stand and can temporarily stand unsupported, but he has to hold onto furniture or adults in order to walk. His mother reports that he responds by turning his head when his name is called, and that he is able to shake his head "no." He babbles frequently, but has not yet said "Mama" or other simple words. Which of the following most accurately represents this child's development?

A. Motor – normal; social – normal; language – normal
B. Motor – delayed; social – normal; language – normal
C. Motor – normal; social – delayed; language – normal
D. Motor – normal; social – normal; language – delayed
E. Motor – delayed; social – delayed; language – delayed

You did not answer this question.

Explanations:

A. This is not a normal 12 month old, so answer A is not the best response.

B. This child does not have isolated motor delay (answer B). There are two key motor milestones that occur at 12 months of age, and both are present here. First, this is the time when kids usually start walking, though they typically still require support. Second, kids usually perfect their pincer grasp around this time – a skill that they happily use to feed themselves, just as the child in this question stem!

C. Answer C is incorrect because socially, this child is on-target. Most children develop pronounced stranger anxiety around nine months of age, and will continue to prefer their mother (or primary caregiver) over all others for months to years.

D. This child is developmentally on target except for one glaring delay in the area of language (answer D). Most children start using "mama" and "dada" nonspecifically around 9 months of age, and use those words specifically by 12 months. You're guaranteed to encounter this style of question regarding developmental milestones on your pediatrics shelf exam - but they've been known to pop up on the family medicine shelf, too. It's nearly impossible to memorize all of the major developmental milestones of early childhood, but you can get a lot of mileage out of just knowing a few of the major ones and then using your common sense. You can find a helpful chart (including a few mnemonics) online at: http://www.clinicalexam.com/pda/peds_ref_developmental_milestones.htm#mnemonics, and if you'd like some more detailed information, you can go straight to the American Academy of Pediatrics website: http://www.aap.org/healthtopics/stages.cfm. This is not a normal 12 month old, so answer A is not the best response. This child does not have isolated motor delay (answer B). There are two key motor milestones that occur at 12 months of age, and both are present here. First, this is the time when kids usually start walking, though they typically still require support. Second, kids usually perfect their pincer grasp around this time – a skill that they happily use to feed themselves, just as the child in this question stem! Answer C is incorrect because socially, this child is on-target. Most children develop pronounced stranger anxiety around nine months of age, and will continue to prefer their mother (or primary caregiver) over all others for months to years. Because this child is developmentally appropriate in two out of three areas, answer E is incorrect.

E. Because this child is developmentally appropriate in two out of three areas, answer E is incorrect.

This question is not currently linked to the learning objective database.

Question problem?

Question # 117

Select the single best answer to the numbered question.

A 45 year old male presents with intermittent chest pain for the past month. He describes burning, retrosternal pain that usually occurs after eating or while lying down or bending over. He also notes that he has been hoarse lately, especially upon awakening in the morning. Past medical history includes a father and grandfather who died of coronary artery disease in their 60s. In the office, the patient is comfortable, and physical examination is entirely unremarkable. Which of the following is the most appropriate initial step in the management of this patient?

A. Metoclopramide
B. Ranitidine
C. Lansoprazole
D. Lifestyle modification and smoking cessation
E. Upper endoscopy

You did not answer this question.

Explanations:

A. Metoclopramide (answer A) is a prokinetic agent. It is a commonly used therapy for conditions like diabetic gastroparesis, and does have some use in patients with refractory GERD. However, it's not a first-line treatment.

B. By blocking H2 receptors, ranitidine (answer B) reduces the production of gastric acid. Since ranitidine (Zantac) and its sister medications like cimetidine (Tagamet) and famotidine (Pepcid) are available over-the-counter, they are often less expensive for patients than proton-pump inhibitors. This patient does not require H2 blockers in particular or even medication in general at this point, though.

C. Proton-pump inhibitors like lansoprazole (answer C) are generally easier to take and provide better symptom relief than H2 blockers. However, PPIs are best used in patients in whom other more conservative therapies have failed, so they're not the best initial step for this patient.

D. This patient is experiencing classic symptoms of a common disorder that affects up to 30% of the U.S. population: gastroesophageal reflux disease, or GERD. Despite what advertisements for proton-pump inhibitors suggest, the best first line therapy for GERD is non-medical. Ideally, treatment for GERD should occur in four steps or stages. Lifestyle modifications (like elevating the head of the bed six inches, decreasing fat intake, losing weight, eating smaller meals, not eating for three hours before bedtime, and stopping smoking) will improve or eliminate the symptoms of GERD in a large group of patients, so answer D is stage 1 therapy for any patient with GERD, and is the best first step for this patient. If patients still have symptoms after making lifestyle modifications, stage 2 of GERD therapy involves the use of as-needed medications like antacids and H2 blockers. If symptoms are uncontrolled, patients need stage 3 therapy of continuous pharmacological therapy with proton-pump inhibitors (PPIs) or H2 blockers. Patients who don't respond to these regimens may need prokinetic agents or surgical intervention like fundoplication. If you'd like more information, a short, simple review of the diagnosis and management of GERD from the American Family Physician can be found online at: http://www.aafp.org/afp/990301ap/1161.html. Metoclopramide (answer A) is a prokinetic agent. It is a commonly used therapy for conditions like diabetic gastroparesis, and does have some use in patients with refractory GERD. However, it's not a first-line treatment. By blocking H2 receptors, ranitidine (answer B) reduces the production of gastric acid. Since ranitidine (Zantac) and its sister medications like cimetidine (Tagamet) and famotidine (Pepcid) are available over-the-counter, they are often less expensive for patients than proton-pump inhibitors. This patient does not require H2 blockers in particular or even medication in general at this point, though. Proton-pump inhibitors like lansoprazole (answer C) are generally easier to take and provide better symptom relief than H2 blockers. However, PPIs are best used in patients in whom other more conservative therapies have failed, so they're not the best initial step for this patient. Upper endoscopy (answer E) is essential for the diagnosis of the complications of GERD (like Barrett's esophagus) but is not a sensitive diagnostic test for GERD itself - only 50% of patients with GERD have macroscopic evidence of it on endoscopy! Most of the time, the diagnosis of GERD can be made clinically, and unless you're trying to rule out another cause of the patient's symptoms (like a peptic ulcer) EGD is not necessary. In rare cases where you need a definitive diagnostic test, the gold standard is ambulatory pH monitoring.

E. Upper endoscopy (answer E) is essential for the diagnosis of the complications of GERD (like Barrett's esophagus) but is not a sensitive diagnostic test for GERD itself - only 50% of patients with GERD have macroscopic evidence of it on endoscopy! Most of the time, the diagnosis of GERD can be made clinically, and unless you're trying to rule out another cause of the patient's symptoms (like a peptic ulcer) EGD is not necessary. In rare cases where you need a definitive diagnostic test, the gold standard is ambulatory pH monitoring.

This question is not currently linked to the learning objective database.

Question problem?

Question problem?

Question # 118

Select the single best answer to the numbered question.

A 27 year old G0 presents with heavy menstrual periods and pain during menses. Her symptoms began two and a half years ago when she discontinued her oral contraceptive medication so that she could attempt to become pregnant. Her symptoms seem to be getting worse, and her pain is now only minimally controlled with over-the-counter medications. Current medications include escitalopram and as-needed ibuprofen and naproxen. Pelvic examination reveals a nontender, fixed, retroverted uterus. Speculum examination shows small bluish spots in the posterior fornix, but no cervical drainage or discharge. There is moderate uterosacral nodularity on rectovaginal examination. The patient's hematocrit is 30%, and her urine beta-hCG is negative. Which of the following is the most likely diagnosis in this patient?

A. Endometrial carcinoma
B. Pelvic inflammatory disease
C. Endometritis
D. Premenstrual dysphoric disorder
E. Endometriosis

You did not answer this question.

Explanations:

A. Endometrial carcinoma (answer A) is a high yield topic for the USMLE. It's the most common gynecological cancer, and it almost always presents as irregular vaginal bleeding, especially in a post-menopausal patient. (This is why you always have to work up vaginal bleeding in a post-menopausal female!) To make the diagnosis, get an endometrial biopsy. Other commonly tested facts about endometrial carcinoma include its major risk factor (unopposed estrogen exposure) and its most important prognostic indicator (histologic grade, instead of stage like most other cancers). Endometrial carcinoma is primarily a cancer of older women, and would be very rare in a patient this young without some risk factor.

B. Pelvic inflammatory disease (answer B) – a.k.a. PID or acute salpingitis - is a complication of sexually transmitted disease. It is primarily a disease that affects young women, who present with pelvic or adnexal pain, an elevated WBC, and cervical motion tenderness. Patients will test positive for N. gonorrhoeae or C. trachomatis. However, the infection in PID is almost always polymicrobial, so treatment is usually with a broad-spectrum cephalosporin plus doxycycline or azithromycin.

C. Endometritis (answer C) refers to an infection of the endometrium, usually following childbirth or instrumentation of the uterine cavity. It is the most common cause of postpartum fever; patients present with fever, lower abdominal pain, foul-smelling lochia, and uterine tenderness on pelvic exam. The infection is usually polymicrobial, and the standard antibiotic therapy is clindamycin (for Gram positives and anaerobes) plus gentamicin (for Gram negatives).

D. Premenstrual dysphoric disorder (answer D) is a psychiatric diagnosis made by the finding of pathological mood symptoms (such as irritability, mood lability, anger, anxiety, and depression) in the two weeks before menses. The time frame is the key to diagnosis if you encounter this disorder on the USMLE: in contrast to other mood disorders, symptoms are only present before and during the time of menses! There are also often associated physical symptoms like bloating, breast tenderness, or headache.

E. This patient has endometriosis (answer E). The question stem includes several key clues and classic descriptions that you ought to know – often the diagnosis isn't so clear cut! For example, a "fixed, retroverted uterus" or blue spots in the posterior fornix are both classic USMLE buzzwords for endometriosis, as is the history of infertility. Other clues not found in this question stem include dyspareunia (especially with deep thrusting), rectal pain during menstruation, pain with defection, or tender bilateral adnexal masses palpable during menstruation. Though you may suspect endometriosis clinically, to confirm the diagnosis, you need to directly visualize the endometrial implants surgically, by laparoscopy or laparotomy. (The lesions will appear as dark red, blue, or purple lesions, frequently called "powder burns," "mulberry lesions," or "chocolate cysts.") Endometriosis can be treated either medically (with OCPs, Depo-Provera injections, danazol, or GnRH agonists like leuprolide) or surgically (by hysterectomy, lysis of adhesions, or removal of endometrial implants). If you'd like more information, a review of endometriosis – written by none other than Dr. Mounsey herself! – can be found online at: http://www.aafp.org/afp/20060815/594.html. Endometrial carcinoma (answer A) is a high yield topic for the USMLE. It's the most common gynecological cancer, and it almost always presents as irregular vaginal bleeding, especially in a post-menopausal female!) To make the diagnosis, get an endometrial biopsy. Other commonly tested facts about endometrial carcinoma include its major risk factor (unopposed estrogen exposure) and its most important prognostic indicator (histologic grade, instead of stage like most other cancers). Endometrial carcinoma is primarily a cancer of older women, and would be very rare in a patient this young without some risk factor. Pelvic inflammatory disease (answer B) – a.k.a. PID or acute salpingitis - is a complication of sexually transmitted disease. It is primarily a disease that affects young women, who present with pelvic or adnexal pain, an elevated WBC, and cervical motion tenderness. Patients will test positive for N. gonorrhoeae or C. trachomatis. However, the infection in PID is almost always polymicrobial, so treatment is usually with a broad-spectrum cephalosporin plus doxycycline or azithromycin. Endometritis (answer C) refers to an infection of the endometrium, usually following childbirth or instrumentation of the uterine cavity. It is the most common cause of postpartum fever; patients present with fever, lower abdominal pain, foul-smelling lochia, and uterine tenderness on pelvic exam. The infection is usually polymicrobial, and the standard antibiotic therapy is clindamycin (for Gram positives and anaerobes) plus gentamicin (for Gram negatives). Premenstrual dysphoric disorder (answer D) is a psychiatric diagnosis made by the finding of pathological mood symptoms (such as irritability, mood lability, anger, anxiety, and depression) in the two weeks before menses. The time frame is the key to diagnosis if you encounter this disorder on the USMLE: in contrast to other mood disorders, symptoms are only present before and during the time of menses! There are also often associated physical symptoms like bloating, breast tenderness, or headache.

This question is not currently linked to the learning objective database.

Question problem?

Question # 119

Select the single best answer to the numbered question.

A 33 year old female with a three year history of recurrent gastric and duodenal ulcers despite medical management is found on subsequent evaluation to have a fasting gastrin level of 989 pg/mL (normal: <100 pg/mL). Abdominal CT scan reveals a 2cm mass in the head of the pancreas, and surgical exploration is planned. Following successful resection of this mass, the patient's fasting gastrin level is found to be 1020 pg/mL. Pathological analysis of the resected specimen shows it to be a glucagonoma. Which of the following is the most likely additional associated finding in this patient?

A. Squamous cell carcinoma of the lung
B. Hyperplasia of the parathyroid glands
C. Pheochromocytoma
D. Pancreatic adenocarcinoma
E. Breast carcinoma

You did not answer this question.

Explanations:

A. Far and away the greatest risk factor for squamous cell carcinoma of the lung (answer A) is smoking. This type of cancer does not have any particular association with MEN syndromes.

B. This patient presented with recurrent ulcers and a high gastrin level – a history that is diagnostic for Zollinger-Ellison syndrome (ZES). In this syndrome, one or many gastrin-secreting tumors cause the overproduction of gastric acid, leading to symptoms of mucosal ulceration, diarrhea, malabsorption, and GERD. In 75% of cases, ZES is sporadic – but in the other 25%, it's a part of Multiple Endocrine Neoplasia syndrome, type 1 (MEN-1). This patient gives us reason to believe that she is part of the latter group: when the surgeons resected the tumor that ought to have been causing her symptoms, they found that it wasn't producing gastrin, it was producing glucagon! Although it's possible that a young patient could simultaneously develop a glucagonoma and a gastrinoma, it's much more likely that these findings are related as part of MEN-1. And of the answer choices listed, the one that is commonly also found in patients with MEN-1 is parathyroid hyperplasia (answer B). You may encounter questions on this topic on your surgery shelf, but even if you don't, you're guaranteed to get at least one question on the MEN syndromes when you take Step 2. Here are some mnemonics to help you remember them! 1) MEN-1 is the "three P's": patients tend to have Parathyroid hyperplasia, Pancreatic islet tumors, and Pituitary tumors in addition to gastrinomas and ZES. 2) Patients with MEN-2A get medullary thyroid cancer, phenochromocytomas, and hyperparathyroidism, so the mnemonic here is "2 MPH" (think "two miles per hour"). If you wonder why pheochromocytoma doesn't count as one of the previous mnemonic's three P's, you've got to use your imagination. Some people remember that each of the three P's of MEN-1 are followed by a vowel, while the 'p' in pheochromocytoma is followed by a consonant. Others remember that each of the three P's of MEN-1 make the "P" sound, while the 'p' in pheochromocytoma makes an "F" sound. 3) Patients with MEN-2B get medullary thyroid carcinoma and pheochromocytoma, but NOT hyperparathyroidism. Here, the mnemonic is "2 PM." Finally, you should know a couple of other things about gastrinomas and Zollinger-Ellison syndrome. First, obtaining a fasting gastrin level is a good first step in making the diagnosis of gastrinoma/ZES. You should also check the basal acid secretion, which should be elevated as well. A secretin stimulation test can be used if further diagnostic confirmation is needed. In a normal patient, administration of secretin will DECREASE the gastrin level, but in patients with ZES, there is a paradoxical INCREASE. Once you've determined that a patient has ZES, the challenge becomes trying to locate the tumor so that you can remove it. The best way to do this is NOT with regular CT (as was done in the question stem!) but instead with a more specialized scan like an octreotide or somatostatin scan, which gets the tumors to "light up" on your images. Far and away the greatest risk factor for squamous cell carcinoma of the lung (answer A) is smoking. This type of cancer does not have any particular association with MEN syndromes. Pheochromocytomas (answer C) are a feature of MEN-2A and MEN-2B, but are not associated with MEN-1. Pancreatic adenocarcinoma (answer D) is incorrect. The tumors that patients with MEN get are tumors of neuroendocrine origin. Although the pancreas is a frequent site for tumors to be found, those tumors arise out of the islet cells, not the exocrine pancreas. Over 90% of "run-of-the-mill" pancreatic cancer is adenocarcinoma. It's unusual in patients younger than 45, and most patients are older than 60. The disease is almost invariably fatal. Many patients present with painless jaundice or Courvoisier's sign" (a palpable, nontender gallbladder), so these are big clues to the diagnosis should you encounter the disease on the USMLE. This patient is no more at risk for breast carcinoma (answer E) than any other patient with her age, family history, and genetics. Remember, the 'E' in MEN stands for endocrine – the tumors that these patients get are in the endocrine system.

C. Pheochromocytomas (answer C) are a feature of MEN-2A and MEN-2B, but are not associated with MEN-1.

D. Pancreatic adenocarcinoma (answer D) is incorrect. The tumors that patients with MEN get are tumors of neuroendocrine origin. Although the pancreas is a frequent site for tumors to be found, those tumors arise out of the islet cells, not the exocrine pancreas. Over 90% of "run-of-the-mill" pancreatic cancer is adenocarcinoma. It's unusual in patients younger than 45, and most patients are older than 60. The disease is almost invariably fatal. Many patients present with painless jaundice or Courvoisier's sign" (a palpable, nontender gallbladder), so these are big clues to the diagnosis should you encounter the disease on the USMLE.

E. This patient is no more at risk for breast carcinoma (answer E) than any other patient with her age, family history, and genetics. Remember, the 'E' in MEN stands for endocrine – the tumors that these patients get are in the endocrine system.

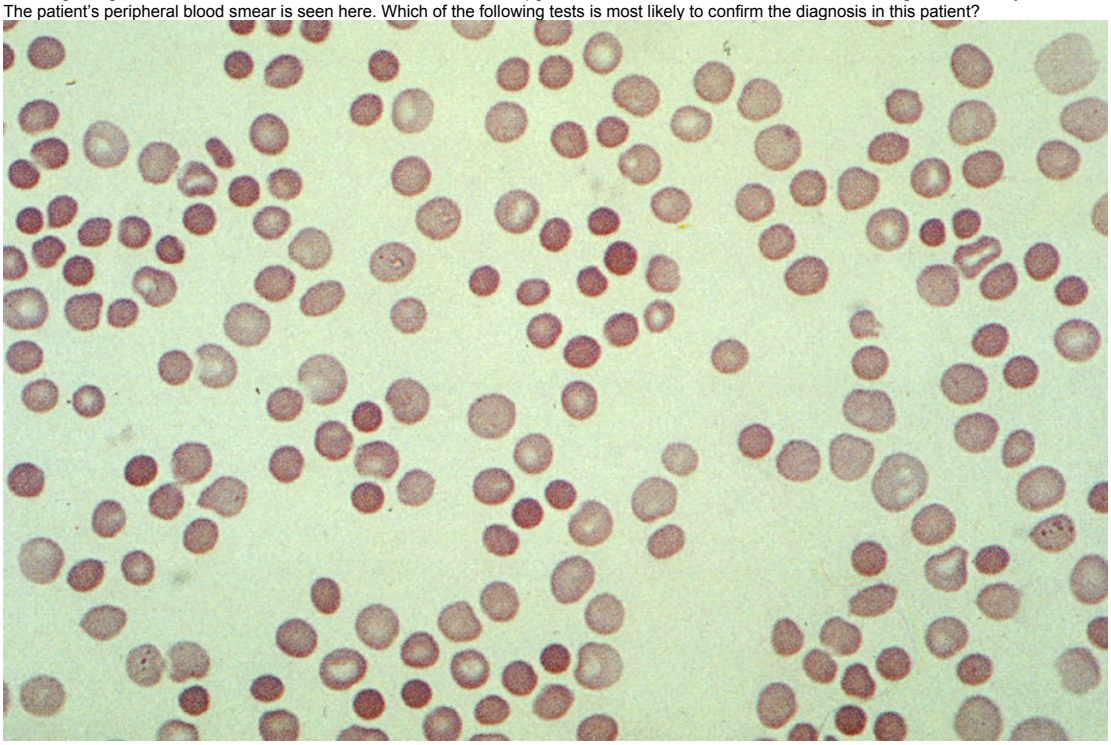
This question is not currently linked to the learning objective database.

Question problem?

Question # 120

Select the single best answer to the numbered question.

A 17 year old female presents for follow-up of her anemia. Three months ago, routine lab work showed a hematocrit of 31%, and the patient was started on oral iron supplements. In the office today, the patient feels well and denies recent illnesses or medications, menorrhagia, hematemesis, hematochezia or melena, and trauma. Physical examination shows a comfortable female in no acute distress. There is moderate pallor of the conjunctivae bilaterally, and the spleen is palpable 1 cm below the left costal margin. There is no spooning of the nails, and position and vibratory sensation are intact on neurological exam. The stool is negative for occult blood. Further laboratory analysis shows the following: WBC 7.1 k/mL, Hgb 10 g/dL, Hct 30%, Platelets 220 k/mL; MCV 85 fL, MHC 31 pg, MCHC 39%; LDH 150 U/L, Total bilirubin 1.2 mg/dL, Reticulocytes 4%. A direct Coomb's test is negative. The patient's peripheral blood smear is seen here. Which of the following tests is most likely to confirm the diagnosis in this patient?



A. Bone marrow biopsy
B. Osmotic fragility test
C. Hemoglobin electrophoresis
D. Quantitative glucose-6-phosphage dehydrogenase enzyme activity
E. Thick and thin blood smears

You did not answer this question.

Explanations:

A. A bone marrow biopsy (answer A) allows you to assess the site of red blood cell production, and may confirm a diagnosis of leukemia or hypoproliferative anemia. However, this patient's reticulocyte count is increased – her bone marrow is doing okay (just not quite good enough to keep up with the destruction of erythrocytes outside of the marrow!).

B. Just like Dr. Innes, the USMLE testmakers love anemia, so you should know the cardinal features of the important anemias by heart. This question describes a patient with an antibody-negative hemolytic anemia (negative Coomb's, but elevated LDH/bilirubin/reticulocytes), an elevated MCHC, and a peripheral smear showing spherocytes. These signs all point to a diagnosis of hereditary spherocytosis – a diagnosis that would best be confirmed with the osmotic fragility test (answer B). A few additional teaching points about hereditary spherocytosis and anemia in general… 1) Hereditary spherocytosis is an autosomal dominant trait, so family members in every generation should be affected. The fundamental defect is in the production of spectrin or other erythrocyte membrane proteins. Without a strong cytoskeleton, the RBC isn't strong enough to withstand everyday stresses, resulting in deformation of the cell from a biconcave disc to a sphere (the geometric form with the least surface area). These cells are also susceptible to destruction in the spleen (leading to splenomegaly) or in a hypotonic bath (which is why the osmotic fragility test is used to confirm diagnosis). 2) An isolated increased MCHC is virtually pathognomonic for hereditary spherocytosis – in fact, some hospitals screen children for spherocytosis with this test! 3) Just like patients with sickle cell disease, patients with hereditary spherocytosis can develop aplastic anemia with parvovirus B19 infection. This tidbit is also one that tends to show up on standardized tests. A bone marrow biopsy (answer A) allows you to assess the site of red blood cell production, and may confirm a diagnosis of leukemia or hypoproliferative anemia. However, this patient's reticulocyte count is increased – her bone marrow is doing okay (just not quite good enough to keep up with the destruction of erythrocytes outside of the marrow!). Hemoglobin electrophoresis (answer C) is the test of choice when you're trying to confirm or rule out hemoglobinopathies like sickle cell disease or thalassemias. The test does just what it sounds like: separates various types of hemoglobin on a gel, helping you determine abnormal hemoglobins that are there (like hemoglobin S in sickle cell disease) or normal hemoglobins that are missing (like alpha or beta chains in thalassemias). However, neither thalassemia or sickle cell disease is the most likely diagnosis in this patient. If the USMLE gives you a photo of a peripheral smear for a patient with sickle cell disease, you'll see obviously sickled RBCs – you won't have to use your imagination. And thalassemias tend to present with a microcytic anemia, not a normocytic one like this patient has. Quantitative glucose-6-phosphate dehydrogenase (G6PD) enzyme activity (answer D) is used to diagnose G6PD deficiency. Usually, this is an X-linked disorder, so finding it in a female would be unlikely. Additionally, the clinical history here doesn't fit. Patients with G6PD have episodes of hemolysis following exposure to increased oxidative stress from drugs (like sulfa drugs), infections, or classically, fava beans. Thick and thin blood smears (answer E) remain the diagnostic standard for malaria. The disease is mosquito borne, and the causative organism is one of various species of Plasmodium. Because the parasite lyses red blood cells as part of its life cycle, patients develop an antibody-negative hemolytic anemia – but this would be very rare as a patient's sole finding. The predominant feature is usually episodic spiking fevers, and there should also be a positive travel history.

C. Hemoglobin electrophoresis (answer C) is the test of choice when you're trying to confirm or rule out hemoglobinopathies like sickle cell disease or thalassemias. The test does just what it sounds like: separates various types of hemoglobin on a gel, helping you determine abnormal hemoglobins that are there (like hemoglobin S in sickle cell disease) or normal hemoglobins that are missing (like alpha or beta chains in thalassemias). However, neither thalassemia or sickle cell disease is the most likely diagnosis in this patient. If the USMLE gives you a photo of a peripheral smear for a patient with sickle cell disease, you'll see obviously sickled RBCs – you won't have to use your imagination. And thalassemias tend to present with a microcytic anemia, not a normocytic one like this patient has.

D. Quantitative glucose-6-phosphate dehydrogenase (G6PD) enzyme activity (answer D) is used to diagnose G6PD deficiency. Usually, this is an X-linked disorder, so finding it in a female would be unlikely. Additionally, the clinical history here doesn't fit. Patients with G6PD have episodes of hemolysis following exposure to increased oxidative stress from drugs (like sulfa drugs), infections, or classically, fava beans.

E. Thick and thin blood smears (answer E) remain the diagnostic standard for malaria. The disease is mosquito borne, and the causative organism is one of various species of Plasmodium. Because the parasite lyses red blood cells as part of its life cycle, patients develop an antibody-negative hemolytic anemia – but this would be very rare as a patient's sole finding. The predominant feature is usually episodic spiking fevers, and there should also be a positive travel history.

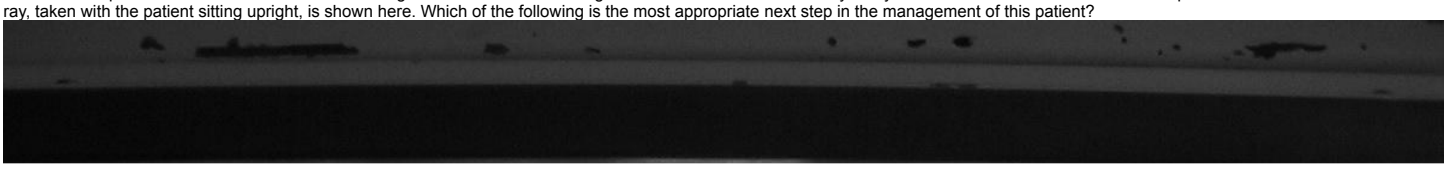
This question is not currently linked to the learning objective database.

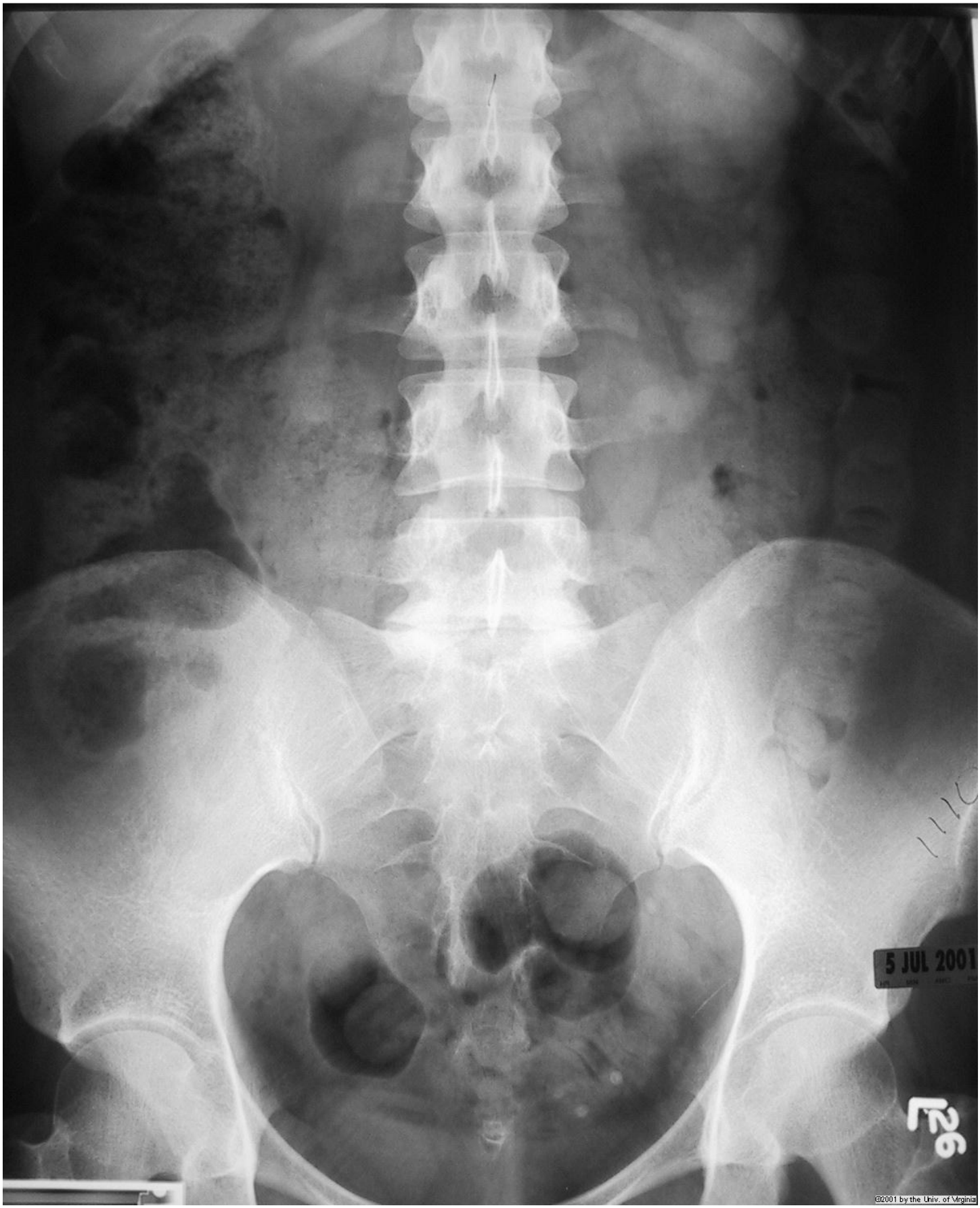
Question problem?

Question # 121

Select the single best answer to the numbered question.

A 66 year old male presents with the abrupt onset of pain in the left lower abdomen, which has been progressively worsening over the past two days. The pain is now constant, and he has also experienced fevers and mild diarrhea. He denies dysuria, nausea, and hematochezia. On physical exam, the patient is alert and in mild distress. Vital signs include temperature 38.1 C (100.6 F), pulse 90, and blood pressure 131/84. The patient's bowel sounds are decreased, and the abdomen is tender to palpation with voluntary guarding in the left lower quadrant. There is no costovertebral angle tenderness. Stool is negative for occult blood. Laboratory analysis shows a WBC of 14.5 k/mm3. The patient's abdominal x-ray, taken with the patient sitting upright, is shown here. Which of the following is the most appropriate next step in the management of this patient?





A. Bowel rest, metronidazole, and ciprofloxacin
B. Microscopic examination of the stool for ova and parasites
C. Barium enema
D. Urgent colonoscopy
E. Emergent laparotomy

You did not answer this question.

Explanations:

A. This patient presents with fever, leukocytosis, and left lower quadrant abdominal pain – the cardinal features of diverticulitis. Since he is hemodynamically stable and has a benign abdominal x-

ray, he can safely be treated medically with a liquid diet and a course of antibiotics (answer A). This question stem brings up a few good teaching points. 1) In patients with diverticulitis (or most any other acute abdominal pain) one of your foremost concerns is ruling out perforation. The best way to do this quickly is with a simple upright abdominal film, just as was done here. What you're looking for is abdominal free air, which would appear as dark, radiolucent pockets just below the lungs (since air settles underneath the diaphragm when the patient is upright). Here, though, you are shown an essentially normal abdominal film – all of the air pockets appear to be within loops of bowel. In the context of the clinical case, this finding means that the patient can safely be managed medically – there is no gross abdominal perforation. (If you'd like to review what an abdominal perforation looks like, check out the UVA Radiology teaching website: http://www.med-ed.virginia.edu/courses/rad/gi/index.html) However, there's a bigger teaching point here that transcends this particular question: When you encounter images on the USMLE, expect any abnormalities to be striking. Either there will be an obvious finding (assuming you know what to look for!) or there won't be anything at all. Here, it's the latter. 2) To treat diverticulitis, you need to cover the predominant colonic flora – that is, gram negatives and anaerobes. Most of the time, this means two drugs: a second- or third-generation cephalosporin or a fluoroquinolone for the gram negatives, and metronidazole for the anaerobes. Alternately, you can treat with amoxicillin/clavulanate alone (remember that any beta-lactam with a beta-lactamase inhibitor has good activity against anaerobes, too). 3) There's a big difference between diverticulITIS and diverticulOSIS. Don't be tricked. Diverticulosis simply refers to the condition of having outpouchings (diverticuli) of the colon; diverticulitis refers to the obstruction, infection, and inflammation of those diverticuli. Diverticulitis presents like the patient in this question stem – with fever and abdominal pain. In contrast, most of the time, diverticulosis is asymptomatic. If the diverticuli erode into an artery, though, the patient will present with painless lower GI bleeding (diverticular bleeding is actually the #1 cause of lower GI bleeding in older adults – this is a commonly pimped fact!). If you'd like more information, you can read a review of diverticular disease from the American Family Physician online at: http://www.aafp.org/afp/20051001/1229.html Microscopic examination of the stool for ova and parasites (answer B) is not indicated. You should order this test in a patient with a recent travel history or risk factors for parasitic infections (like drinking unpurified water), or in a patient who has chronic, unexplained diarrhea. A barium enema (answer C) is contraindicated acutely in patients with suspected diverticulitis. Although this is an excellent study for identifying colonic diverticuli in a healthy patient, giving barium to a patient with diverticulitis could have disastrous consequences if the patient had perforated their bowel and the barium leaked into the peritoneum! Here, although the abdominal plain film shows no abdominal free air, you still don't need to get a barium enema – you can make the diagnosis of diverticulitis clinically, without any imaging study. (If the diagnosis is unclear, or if you need to rule out other possibilities, the imaging method of choice is CT.) Colonoscopy (answer D) is contraindicated in the acute phase of diverticulitis. Proceeding with endoscopy at this point could cause a colonic perforation or exacerbate a pre-existing one. You don't need a colonoscopy to make the diagnosis of diverticulitis – you can do that on clinical grounds alone (although CT is the imaging study to get if you need to solidify your diagnosis). However, all patients who are successfully treated for diverticulitis should get a colonoscopy within a month or two to rule out the possibility of a malignancy. Emergent laparotomy (answer E) is required in cases of diverticulitis that result in macroperforation of the colon. These patients develop physical exam features of a "surgical abdomen," and you'd expect to see free air on the abdominal x-ray.

B. Microscopic examination of the stool for ova and parasites (answer B) is not indicated. You should order this test in a patient with a recent travel history or risk factors for parasitic infections (like drinking unpurified water), or in a patient who has chronic, unexplained diarrhea.

C. A barium enema (answer C) is contraindicated acutely in patients with suspected diverticulitis. Although this is an excellent study for identifying colonic diverticuli in a healthy patient, giving barium to a patient with diverticulitis could have disastrous consequences if the patient had perforated their bowel and the barium leaked into the peritoneum! Here, although the abdominal plain film shows no abdominal free air, you still don't need to get a barium enema – you can make the diagnosis of diverticulitis clinically, without any imaging study. (If the diagnosis is unclear, or if you need to rule out other possibilities, the imaging method of choice is CT.)

D. Colonoscopy (answer D) is contraindicated in the acute phase of diverticulitis. Proceeding with endoscopy at this point could cause a colonic perforation or exacerbate a pre-existing one. You don't need a colonoscopy to make the diagnosis of diverticulitis – you can do that on clinical grounds alone (although CT is the imaging study to get if you need to solidify your diagnosis). However, all patients who are successfully treated for diverticulitis should get a colonoscopy within a month or two to rule out the possibility of a malignancy.

E. Emergent laparotomy (answer E) is required in cases of diverticulitis that result in macroperforation of the colon. These patients develop physical exam features of a "surgical abdomen," and you'd expect to see free air on the abdominal x-ray.

This question is not currently linked to the learning objective database.

Question problem?

Question # 122

Select the single best answer to the numbered question.

A 63 year old woman with a history of breast cancer presents for follow-up. Five years ago, the patient was diagnosed with lymph node-positive invasive ductal carcinoma of the right breast, for which she underwent lumpectomy and breast radiation followed by chemotherapy. The patient has been feeling very well recently, and though she does note some recent irregular menstrual spotting and bleeding, she denies chest pain, shortness of breath, and bone or joint pains. The patient is a G0, and menopause occurred at age 53. Current medications include hydrochlorothiazide and tamoxifen. Physical examination reveals a pleasant, obese Caucasian female in no distress. Heart rate is regular, with normal S1 and S2. Lung fields are clear to auscultation bilaterally. Pelvic exam shows an anteverted uterine fundus. Adnexae are non-enlarged and nontender. There is a small amount of brownish blood in the vaginal vault. No masses or lymphadenopathy are found on breast examination. Bilateral mammography shows no change from previous. Which of the following is the most appropriate next step in the management of this patient?

A. Measurement of serum CEA and CA-15-3
B. Endometrial biopsy
C. CT of chest and abdomen
D. Technetium-99 bone scan
E. Reassurance and routine follow up

You did not answer this question.

Explanations:

A. Both CEA and CA-15-3 (answer A) are serum markers capable of detecting an early breast cancer recurrence. However, they are neither sensitive nor specific, and there is no evidence yet that routine measurement improves either mortality or quality of life. Thus, they are not recommended as routine tests for breast cancer follow-up.

B. Key teaching point: Post-menopausal bleeding is never normal – get an endometrial biopsy! This question describes a woman with post-menopausal bleeding, and tests a simple fact – that post-menopausal bleeding is not normal, and demands a workup. Don't be thrown off by all of the other "window dressing" in the question stem! There are a number of things that can cause post-menopausal bleeding, ranging from uterine and cervical polyps to atrophic vaginitis. However, the most common cause, and the most important one to rule out, is endometrial hyperplasia or carcinoma. The test of choice for doing this is an endometrial biopsy (answer B). Here are two quick teaching points for this case… 1) The single biggest factor for endometrial hyperplasia and carcinoma is ESTROGEN EXPOSURE. Most all of the specific risk factors for the disease are things that increase the patient's lifetime exposure to estrogen: nulliparity, early menarche, late menopause, obesity (remember, adipocytes have aromatase and convert androgens to estrogen), and unopposed estrogen therapy (including SERMs like tamoxifen!). Other risk factors include diabetes mellitus and hypertension. 2) Unlike most cancers, where clinical stage is the most important prognostic indicator, histological grade is the most important prognosticator for endometrial carcinoma. Both CEA and CA-15-3 (answer A) are serum markers capable of detecting an early breast cancer recurrence. However, they are neither sensitive nor specific, and there is no evidence yet that routine measurement improves either mortality or quality of life. Thus, they are not recommended as routine tests for breast cancer follow-up. Neither a CT of the patient's chest and abdomen (answer C) or a technetium bone scan (answer D) is needed. According to the latest recommendations from the American Society of Clinical Oncology, imaging studies should not be routinely ordered for breast cancer follow-up. Routine imaging has not been shown to improve mortality (or alter the therapeutic approach) for any group of breast cancer survivors, and so in the absence of symptoms suggesting metastasis (like bone pain, neurological deficits, or pulmonary symptoms), these studies are not indicated. If you're interested in seeing the complete 2006 recommendations for breast cancer follow-up, you can find them online at: http://www.guideline.gov/summary/summary.aspx?doc_id=9908&nbr=5304&ss=6&xl=999. Reassurance and routine follow-up (answer E) would be appropriate from the point of view of the patient's breast cancer, but is inappropriate given the fact that she now has post-menopausal bleeding. Even one drop of blood in a postmenopausal woman not on hormone replacement therapy demands an explanation!

C. Neither a CT of the patient's chest and abdomen (answer C) or a technetium bone scan (answer D) is needed. According to the latest recommendations from the American Society of Clinical Oncology, imaging studies should not be routinely ordered for breast cancer follow-up. Routine imaging has not been shown to improve mortality (or alter the therapeutic approach) for any group of breast cancer survivors, and so in the absence of symptoms suggesting metastasis (like bone pain, neurological deficits, or pulmonary symptoms), these studies are not indicated. If you're interested in seeing the complete 2006 recommendations for breast cancer follow-up, you can find them online at: http://www.guideline.gov/summary/summary.aspx?doc_id=9908&nbr=5304&ss=6&xl=999.

D. Neither a CT of the patient's chest and abdomen (answer C) or a technetium bone scan (answer D) is needed. According to the latest recommendations from the American Society of Clinical Oncology, imaging studies should not be routinely ordered for breast cancer follow-up. Routine imaging has not been shown to improve mortality (or alter the therapeutic approach) for any group of breast cancer survivors, and so in the absence of symptoms suggesting metastasis (like bone pain, neurological deficits, or pulmonary symptoms), these studies are not indicated. If you're interested in seeing the complete 2006 recommendations for breast cancer follow-up, you can find them online at: http://www.guideline.gov/summary/summary.aspx?doc_id=9908&nbr=5304&ss=6&xl=999.

E. Reassurance and routine follow-up (answer E) would be appropriate from the point of view of the patient's breast cancer, but is inappropriate given the fact that she now has post-menopausal bleeding. Even one drop of blood in a postmenopausal woman not on hormone replacement therapy demands an explanation!

This question is not currently linked to the learning objective database.

Question problem?

Question # 123

Select the single best answer to the numbered question.

A 73 year old man with a past medical history of hypertension, coronary artery disease, and osteoarthritis presents for medical evaluation and pre-operative clearance before undergoing a left hip arthroplasty. He is eager to proceed with the operation, and states that he has been feeling well and denies chest pain, dypnea, or syncope. Two years ago, the patient had a non-ST elevation myocardial infarction and underwent two vessel angioplasty and stent placement, and the patient tolerated general anesthesia without complication ten years ago when he had successful bilateral inguinal hernia repairs. Current medications include atorvastatin, clopidogrel, metoprolol, aspirin, and enalapril. On physical exam, vital signs include pulse 64, blood pressure 126/72, respirations 12/min, and oxygen saturation 98% on room air. Lung fields are clear to auscultation. There is a harsh, II/VI mid-systolic ejection murmur heard best over the second right intercostal space with radiation to the right neck. There is no peripheral edema. EKG shows normal sinus rhythm and left ventricular hypertrophy. Which of the following is the most appropriate decision regarding this patient's scheduled surgery?

A. Advise the patient to proceed with his surgery as planned
B. Discontinue metoprolol
C. Obtain pre-operative echocardiogram
D. Obtain pre-operative coronary angiography
E. Advise the patient to cancel the procedure

You did not answer this question.

Explanations:

A. This question asks you to make a pre-operative assessment of a patient who is about to undergo major surgery. Your major concern in this situation is the patient's heart – perioperative MIs remain a significant source of morbidity and mortality, especially in older patients with cardiac risk factors. The American Heart Association has created guidelines to cost-effectively risk stratify cardiac patients for noncardiac surgery – you can see the complete algorithm online, along with a helpful review from the American Family Physician, at: http://www.aafp.org/afp/20070301/656.html. The AHA algorithm can get a little complicated, and you certainly won't be expected to memorize it for the USMLE. What you will have to do, though, is have a general understanding of the algorithm and be able to use a little bit of clinical judgment! The gold standard for cardiac clearance is recent coronary angiography or recent revascularization. So long as a patient has not had a change in their clinical status, a patient is ready for the O.R. if they've had a successful revascularization within the past 5 years or a "clean" coronary angiogram within the past six months. That means our patient, who had stents placed two years ago, can be medically cleared for surgery as scheduled (answer A). The situation becomes more complicated if your patient has NOT had a recent revascularization or angiogram, or if they've got more serious cardiovascular risk factors. Still, if you encounter these situations on the USMLE, just use your clinical judgment! In general, if a patient has risk factors like decompensated CHF, significant arrhythmias, or an MI within the past thirty days, they're high risk for cardiac perioperative morbidity and mortality, they need coronary angiography before they go to the operating room. If the patient has other, milder risk factors - like stable CHF, mild angina, diabetes, or uncontrolled hypertension - they'll be intermediate or low risk, and you should start with a stress test. Keep in mind that the purpose of your evaluation is to find patients with significant heart disease that is severe enough to cause problems in the perioperative period – you're not trying to rule out the possibility of heart disease altogether. No cardiologist would guarantee you that the patient in this question stem has pristine coronary arteries, or that he has no heart disease whatsoever – but they might say that you can have reasonable confidence that his heart disease will not cause problems for him in the immediate perioperative period. Stopping metoprolol (answer B) is the wrong thing to do. There is a definite benefit in providing prophylactic beta-blockers to high risk cardiac patients perioperatively. For low or intermediate risk patients, the evidence of benefit is less clear, so deciding who to give beta-blockers to remains a bit of a controversial topic. However, there is definitely no need to discontinue a beta-blocker in a patient that had previously been taking one. Obtaining an echocardiogram (answer C) is not necessary pre-operatively. Although an echocardiogram would give you information about this patient's ejection fraction and any wall motion abnormalities, it's not as useful as a stress test or a coronary angiogram in evaluating his pre-operative cardiac risk – and even those tests are unnecessary in this situation given this patient's recent revascularization. If you selected this option, you might have been concerned about this patient's heart murmur. Certainly, any patient with a new murmur (or a change in an old one) deserves evaluation with echo. Given the quality and location of this patient's murmur, he likely has aortic stenosis. This is a commonly tested disorder on the USMLE, and you may remember that the timing of valve replacement surgery is guided by the patient's symptoms – especially if they've developed syncope, angina, or congestive heart failure, which are all ominous symptoms that portend poor survival. However, considering that mild aortic stenosis due to valvular calcification is so common in older patients, and the fact that this patient's symptoms do not appear to be dictate valve replacement imminently, you probably don't need to postpone his surgery to evaluate his valves. Coronary angiography (answer D) is the single best test to determine a patient's cardiac readiness for surgery. However, it's also an invasive test, so non-invasive imaging like stress testing should generally be done first – unless the patient has so many risk factors for coronary artery disease that their pre-test likelihood of severe CAD is so high that it obviates the need for other tests. This patient does not fall into such a high-risk category. More importantly, the patient has been clinically stable since his last coronary angiography and intervention, and this occurred recently enough that you can feel reasonably confident that he does not need further testing. Advising the patient to cancel the procedure (answer E) is unnecessary. If the patient had major cardiac risk factors like decompensated CHF, severe valvular disease, or unstable coronary syndromes, it would be appropriate to postpone or cancel an intermediate-risk, elective procedure like hip replacement. This patient does not fall into that high-risk category, though.

B. Stopping metoprolol (answer B) is the wrong thing to do. There is a definite benefit in providing prophylactic beta-blockers to high risk cardiac patients perioperatively. For low or intermediate risk patients, the evidence of benefit is less clear, so deciding who to give beta-blockers to remains a bit of a controversial topic. However, there is definitely no need to discontinue a beta-blocker in a patient that had previously been taking one.

C. Obtaining an echocardiogram (answer C) is not necessary pre-operatively. Although an echocardiogram would give you information about this patient's ejection fraction and any wall motion abnormalities, it's not as useful as a stress test or a coronary angiogram in evaluating his pre-operative cardiac risk – and even those tests are unnecessary in this situation given this patient's recent revascularization. If you selected this option, you might have been concerned about this patient's heart murmur. Certainly, any patient with a new murmur (or a change in an old one) deserves evaluation with echo. Given the quality and location of this patient's murmur, he likely has aortic stenosis. This is a commonly tested disorder on the USMLE, and you may remember that the timing of valve replacement surgery is guided by the patient's symptoms – especially if they've developed syncope, angina, or congestive heart failure, which are all ominous symptoms that portend poor survival. However, considering that mild aortic stenosis due to valvular calcification is so common in older patients, and the fact that this patient's symptoms do not appear to be dictate valve replacement imminently, you probably don't need to postpone his surgery to evaluate his valves.

D. Coronary angiography (answer D) is the single best test to determine a patient's cardiac readiness for surgery. However, it's also an invasive test, so non-invasive imaging like stress testing should generally be done first – unless the patient has so many risk factors for coronary artery disease that their pre-test likelihood of severe CAD is so high that it obviates the need for other tests. This patient does not fall into such a high-risk category. More importantly, the patient has been clinically stable since his last coronary angiography and intervention, and this occurred recently enough that you can feel reasonably confident that he does not need further testing.

E. Advising the patient to cancel the procedure (answer E) is unnecessary. If the patient had major cardiac risk factors like decompensated CHF, severe valvular disease, or unstable coronary syndromes, it would be appropriate to postpone or cancel an intermediate-risk, elective procedure like hip replacement. This patient does not fall into that high-risk category, though.

This question is not currently linked to the learning objective database.

Question problem?

Question # 124

Select the single best answer to the numbered question.

Over the past several weeks, a 37 year old nurse with a history of depression and generalized anxiety disorder has experienced near-daily episodes of sweating, anxiety,

palpations, and tremor. The patient denies any association with identifiable stressors, and states that the symptoms can occur at any time of day, but most frequently affect her at work. During her most recent episode, she lost consciousness and had to be revived by her co-workers. In the office, the patient is initially pleasant and alert, but quickly grows irritable, tremulous, and after a few minutes, somnolent. Following administration of supplemental oxygen by nasal cannula and a 1 L bolus of 0.45% NaCl with 5% dextrose, the patient feels much better. Labs taken at the time of her symptoms include the following: Na+ 137 mEq/L, K+ 3.9 mEq/L, Cl- 99 mEq/L, HCO3- 24 mEq/L, BUN 11 mg/dL, Creatinine 0.7 mg/dL, Glucose 36 mg/dL, TSH 1.1 mcU/mL, Free T4 8 mcg/dL, T3 160 ng/dL, Insulin 96 mcU/mL (normal: <100 mcU/mL), C-peptide 0.03 ng/mL (normal: 0.70-1.89 ng/mL), Parathyroid hormone, N-terminal 318 pg/mL. Toxicology is negative for cocaine, amphetamines, opiates, benzodiazepines, and cannabis. Serum assay for sulfonylureas is negative. Which of the following is the most likely diagnosis in this patient?

A. Thyrotoxicosis
B. Insulinoma
C. Multiple endocrine neoplasia, type 1
D. Panic disorder
E. Factitious disorder

You did not answer this question.

Explanations:

A. Severe thyrotoxicosis (answer A) may present with agitation, delirium, psychosis, and alterations in vital signs (tachycardia, hyperthermia, etc.). However, this patient has a normal TSH, so she does not have a physiological cause for hyperthyroidism. Exogenous administration of thyroid medications causing factitious hyperthyroidism does occur, but this patient has normal T3 and T4 levels, ruling out that possibility.

B. Insulinomas (answer B) are among the most serious causes of hypoglycemia. Making a correct diagnosis is critical – if a patient has one, they need surgery to prevent tumor spread, but if they don't have one, you don't want to subject them to unnecessary surgery! The way to make the diagnosis is by inducing hypoglycemia with a 72-hour fast, and then measuring serum insulin levels. In a normal patient, insulin levels should be suppressed as blood glucose levels drop, but in a patient with an insulinoma, there will be inappropriately high levels of insulin, even in the face of hypoglycemia. Once you've confirmed the diagnosis with the 72-hour fast, the challenge then becomes localizing the tumor and removing it surgically. Although this patient does indeed have inappropriately high levels of insulin despite hypoglycemia, there's something else about her lab results that should tip you off regarding an alternate diagnosis.

C. Insulinomas and other pancreatic islet cell tumors are associated with multiple endocrine neoplasia, type 1 (answer C). However, even if this patient had an insulinoma, making a diagnosis of MEN-1 would be premature without evidence of the other components of the syndrome. (Remember, MEN-1 is the "three P's" of parathyroid hyperplasia, pancreatic islet cell tumors, and pituitary tumors.) Almost all patients with MEN-1 have primary hyperparathyroidism, so checking a calcium or PTH level is critical.

D. Patients with panic disorder (answer D) experience recurrent panic attacks AND develop persistent concern about having additional attacks or worry about the implications of having attacks. (It's this latter part that distinguishes panic attacks from panic disorder – a commonly tested fact on the psych shelf and Step 2.) Because of their panic attacks, patients with panic disorder frequently develop agoraphobia. Panic attacks may cause physical symptoms, especially feelings of choking, chest pain, nausea, sweating, and tremor, and while this patient does have some of those symptoms, she has a good medical reason for having them: hypoglycemia.

E. Key teaching point: if you find a high insulin level in a patient with a low C-peptide level, think about surreptitious insulin use and factitious hypoglycemia. Here, the answer is all in the labs. You can expect to encounter numerous questions on the USMLE that test your ability to analyze lab results. When you come across these questions, be methodical: the first step is to determine if and how the values presented depart from normal values. Work quickly, but be thorough – the answer to the question frequently will turn on just one or two values! Next, figure out what the clinical implications of those deviations are. This is the tougher part, but as you do your reading and test preparation, try to build an idea of why each test is ordered, and what an abnormal result means. Using this strategy here, you'll see that this patient has two abnormal lab values. First, her glucose is markedly low – so now you know the likely cause of her symptoms, and why she improved when she received i.v. fluid containing dextrose. Second, she has a low C-peptide, a result that requires a bit more explanation. Recall that, in order to produce insulin, your pancreas first synthesizes proinsulin – a protein that is subsequently cleaved into a molecule of active insulin and a molecule of inactive C-peptide. Therefore, in any patient with normal physiology, you should find equal amounts of insulin and C-peptide. The patient here has an elevated insulin level given her profound hypoglycemia (low blood sugar should suppress insulin secretion!) and has a nearly-undetectable C-peptide. What this tells you is that the insulin in this patient did not come from her pancreas – it came from a pharmaceutical company, who kindly removed the C-peptides from the insulin molecules for her. This patient has been injecting herself with insulin to create her symptoms, so the correct diagnosis is factitious disorder (answer E). Just three brief teaching points… 1) Remember that factitious disorder is a psychiatric diagnosis on the spectrum of the somatoform disorders. The way to distinguish among these disorders is by considering both the MOTIVATION for the patient's behavior, and their AWARENESS of it. Patients with somatization disorder are unaware that they are "creating" their symptoms, and they do so unconsciously, for primary or secondary gain. Patients with factitious disorder consciously create their symptoms, but have unconscious reasons for wanting to do so, while malingering patients have both an awareness of what they're doing and their motivation for doing it (to obtain secondary gain). 2) This question tells you that the patient is a nurse and has a history of depression and anxiety disorders – both of these are typical standardized test clues for a diagnosis of factitious disorder. Comorbid psychiatric illnesses – especially depression and anxiety – are risk factors for somatoform disorders. Healthcare providers probably don't have any increased risk for factitious disorder, but their knowledge and background help them more effectively "game" the system. 3) Factitious hypoglycemia can be caused by the ingestion of oral hypoglycemics, too – not just exogenous insulin. If this is the case, C-peptide levels will NOT be decreased, because the drug is simulating normal physiology by stimulating the pancreas to secrete more insulin. Severe thyrotoxicosis (answer A) may present with agitation, delirium, psychosis, and alterations in vital signs (tachycardia, hyperthermia, etc.). However, this patient has a normal TSH, so she does not have a physiological cause for hyperthyroidism. Exogenous administration of thyroid medications causing factitious hyperthyroidism does occur, but this patient has normal T3 and T4 levels, ruling out that possibility. Insulinomas (answer B) are among the most serious causes of hypoglycemia. Making a correct diagnosis is critical – if a patient has one, they need surgery to prevent tumor spread, but if they don't have one, you don't want to subject them to unnecessary surgery! The way to make the diagnosis is by inducing hypoglycemia with a 72-hour fast, and then measuring serum insulin levels. In a normal patient, insulin levels should be suppressed as blood glucose levels drop, but in a patient with an insulinoma, there will be inappropriately high levels of insulin, even in the face of hypoglycemia. Once you've confirmed the diagnosis with the 72-hour fast, the challenge then becomes localizing the tumor and removing it surgically. Insulinomas and other pancreatic islet cell tumors are associated with multiple endocrine neoplasia, type

1 (answer C). However, even if this patient had an insulinoma, making a diagnosis of MEN-1 would be premature without evidence of the other components of the syndrome. (Remember, MEN-1 is the "three P's" of parathyroid hyperplasia, pancreatic islet cell tumors, and pituitary tumors.) Almost all patients with MEN-1 have primary hyperparathyroidism, so checking a calcium or PTH level is critical. Patients with panic disorder (answer D) experience recurrent panic attacks AND develop persistent concern about having additional attacks or worry about the implications of having attacks. (It's this latter part that distinguishes panic attacks from panic disorder – a commonly tested fact on the psych shelf and Step 2.) Because of their panic attacks, patients with panic disorder frequently develop agoraphobia. Panic attacks may cause physical symptoms, especially feelings of choking, chest pain, nausea, sweating, and tremor, and while this patient does have some of those symptoms, she has a good medical reason for having them: hypoglycemia.

This question is not currently linked to the learning objective database.

Question problem?

Question # 125

Select the single best answer to the numbered question.

A 63 year old male presents with fever and urinary frequency for the past two days. He also notes urinary urgency and feelings of incomplete voiding. On physical exam, the patient has a temperature of 37.9 C (100.3 F), pulse 90, and blood pressure 121/76. Bowel sounds are normoactive, and there is no abdominal or costovertebral angle tenderness. On digital rectal exam, the prostate is slightly enlarged, nodular, boggy, and tender to palpation. Stool is negative for occult blood. The patient's urinalysis shows cloudy urine that is negative for ketones, glucose, protein, and blood, and positive for leukocyte esterase on dipstick analysis. Which of the following is the most appropriate treatment for this patient?

A. Moxifloxacin
B. Tamsulosin
C. Trimethoprim/sulfamethoxazole
D. Anidulafungin
E. Finasteride

You did not answer this question.

Explanations:

A. Most fluoroquinolones can be used to treat urinary tract infections and prostatitis, but moxifloxacin (answer A) is the exception to this rule. Moxifloxacin is a fourth-generation or "respiratory" fluoroquinolone, so named because of its activity against drug-resistant S.pneumoniae (DRSP) and anaerobes. Like other earlier generation fluoroquinolones, it has excellent activity against Gram negatives – BUT moxifloxacin does not concentrate in the urine like other fluoroquinolones, so it should not be used for UTIs or prostatitis (which is often accompanied by simultaneous bacterial colonization or infection of the lower urinary tract).

B. Tamsulosin (answer B) is a specific alpha-1 adrenergic blocker that is useful in the treatment of bladder outlet obstruction secondary to BPH. Since the portion of the urethra that courses through the prostate is innervated by alpha-1 receptors, treatment with alpha blockers like tamsulosin, terazosin, and doxazosin can reduce the smooth muscle tension in the urethra and improve urinary symptoms.

C. This patient's fever, dysuria, and boggy, tender prostate gland all point to a diagnosis of acute bacterial prostatitis. The recommended antibiotic treatment for prostatitis is either a fluoroquinolone like ciprofloxacin or levofloxacin or trimethoprim/sulfamethoxazole (answer C). These are the same first-line drugs used to treat urinary tract infections, because the causative organisms are the same – usually Gram negative rods or coliforms. Two brief teaching points about this case… 1) Although this patient has acute bacterial prostatitis, chronic prostatitis may be more common overall. Patients experience urinary symptoms (which may mimic BPH), or may have recurrent urinary tract infections interspersed with asymptomatic periods. These patients may have a normal physical exam, so diagnosis depends on symptoms and lab findings from a patient's urine and expressed prostatic secretions. Treatment is still with fluoroquinolones or TMP/SMZ, though you must treat for a longer period of time – usually 6-12 weeks – to eradicate the infection. 2) Prostatitis in a younger patient is likely caused by N.gonorrhoeae or C.trachomatis, not gut flora. Treatment is the same as it is for gonococcal or chlamydial urethritis: fluoroquinolones or cephalosporins for gonorrhea, and doxycycline or azithromycin for chlamydia. Most fluoroquinolones can be used to treat urinary tract infections and prostatitis, but moxifloxacin (answer A) is the exception to this rule. Moxifloxacin is a fourth-generation or "respiratory" fluoroquinolone, so named because of its activity against drug-resistant S.pneumoniae (DRSP) and anaerobes. Like other earlier generation fluoroquinolones, it has excellent activity against Gram negatives – BUT moxifloxacin does not concentrate in the urine like other fluoroquinolones, so it should not be used for UTIs or prostatitis (which is often accompanied by simultaneous bacterial colonization or infection of the lower urinary tract). Tamsulosin (answer B) is a specific alpha-1 adrenergic blocker that is useful in the treatment of bladder outlet obstruction secondary to BPH. Since the portion of the urethra that courses through the prostate is innervated by alpha-1 receptors, treatment with alpha blockers like tamsulosin, terazosin, and doxazosin can reduce the smooth muscle tension in the urethra and improve urinary symptoms. Anidulafungin (answer D) is an antifungal agent in the echinocandin class. It is particularly useful for treating Candida infections – especially those caused by non-albicans species like C.parapsilosis and C.glabrata (which may be resistant to treatment with drugs like fluconazole). Although case reports exist of Candida spp. causing prostatitis, they are very rare. Finasteride (answer E) is a 5-alpha reductase inhibitor used to treat bladder outlet obstruction caused by BPH. Finasteride (and its cousin, dutasteride) work by blocking the conversion of testosterone to dihydrotestosterone, a more potent androgen. Over time, treatment with these drugs can reduce the prostate size and symptoms of BPH. (As a sidenote, finasteride is also available for the treatment of male pattern baldness, sold under the trade name Propecia.)

D. Anidulafungin (answer D) is an antifungal agent in the echinocandin class. It is particularly useful for treating Candida infections – especially those caused by non-albicans species like C.parapsilosis and C.glabrata (which may be resistant to treatment with drugs like fluconazole). Although case reports exist of Candida spp. causing prostatitis, they are very rare, and the first-line treatment should be an antibacterial agent.

E. Finasteride (answer E) is a 5-alpha reductase inhibitor used to treat bladder outlet obstruction caused by BPH. Finasteride (and its cousin, dutasteride) work by blocking the conversion of testosterone to dihydrotestosterone, a more potent androgen. Over time, treatment with these drugs can reduce the prostate size and symptoms of BPH. (As a sidenote, finasteride is also available for the treatment of male pattern baldness, sold under the trade name Propecia.)

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