USMLE-STEP-1 Q&As

United States Medical Licensing Step 1

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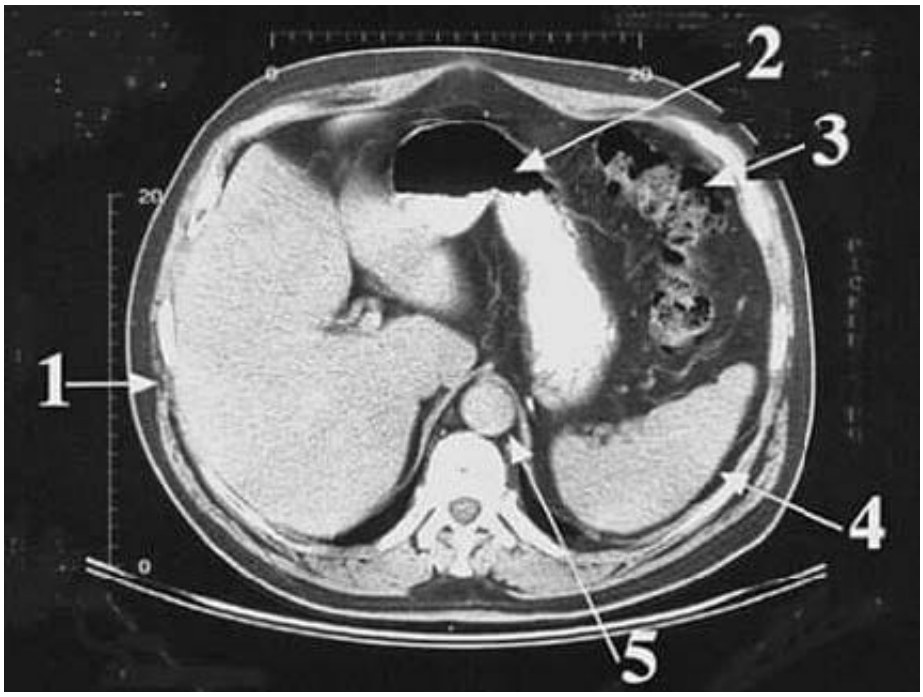
QUESTION 1

Arrow 4 in following figure, is pointing to which of the following structures?



A. abdominal aorta

B. colon

C. liver

D. spleen

E. stomach

Correct Answer: D

Section: Anatomy The spleen (arrow 4) lies to the left of the abdominal cavity. It is in contact with the left side of the stomach (arrow 2) and lodges against the left paravertebral gutter. The abdominal aorta (choice A, arrow 5) is seen as the circular structure immediately anterior to the vertebra. The colon (choice B, arrow 3) is the convoluted structure to the left anterior aspect of the abdominal cavity. The large liver (choice C, arrow 1) occupies most of the right side of the abdominal cavity. The stomach (choice E, arrow 2) is located between the colon and the liver, and in this case, contains liquid contrast material.

QUESTION 2

Which of the following drugs is useful in treatment of gout with recurrent renal urate stones because it decreases the excretion of uric acid?

A. allopurinol

B. aspirin

C. colchicines

D. indomethacin

E. sulfinpyrazone

Correct Answer: A

Section: Pharmacology Allopurinol and its metabolite alloxanthine inhibit xanthine oxidase, thus preventing conversion of xanthine and hypoxanthine to uric acid. Although xanthine and hypoxanthine then accumulate, these compounds are more soluble than uric acid and less likely to deposit in joints or precipitate in the urine. Most doses of aspirin (choice B) increase retention of uric acid, especially low doses. Colchicine (choice C) is an inhibitor of microtubule function that brings relief in an acute gout attack by inhibiting the motility of granulocytes and preventing the formation of mediators of inflammation by leukocytes. Because of its toxicity at higher doses, it is now used chiefly at low doses to prevent acute attacks. Indomethacin (choice D) is an NSAID that inhibits COX and reduces formation of prostaglandins and eicosanoids involved in gouty arthritis. It has no effect on the formation of uric acid and very little on its excretion. Sulfinpyrazone (choice E) and probenecid are uricosuric agents--they increase the excretion of uric acid by the kidney. Renal uric acid excretion is determined by the balance between the amount filtered plus that actively secreted and the amount undergoing passive and active reabsorption. At very low doses, these agents inhibit active secretion and thus promote retention of uric acid. At higher (clinical) doses, both active secretion and active reabsorption are inhibited, with the result that excretion is enhanced.

QUESTION 3

The location of the lesion in Horner syndrome (either preganglionic or postganglionic) can be determined by the use of certain sympathomimetics. Which of the following agents would distinguish between a preganglionic versus a postganglionic lesion?

A. amphetamine

B. atenolol

C. epinephrine

D. isoproterenol

E. phenylephrine

Correct Answer: A

Section: Pharmacology Indirectly acting sympathomimetics (e.g., amphetamines, cocaine) are useful in this situation because their action requires the presence of intact postganglionic noradrenergic neurons. Thus administration of hydroxyamphetamine into the eye will cause mydriasis in a Horner patient if the lesion is preganglionic (postganglionic neuron intact), but not if the lesion is postganglionic. In contrast, directacting sympathomimetics (choices C, D, E) will have the same effect regardless of the location of the lesion. Beta antagonists (choice B) will not produce any effect on the pupil.

QUESTION 4

Which of the following drugs is selectively antifungal because of the difference between cholesterol (in mammalian cell membranes) and ergosterol (in fungal membranes)?

A. amphotericin B

B. caspofungin

C. fluconazole

D. griseofulvin

E. terbinafine

Correct Answer: A

Section: Pharmacology Amphotericin B binds selectively to ergosterol and forms artificial pores in fungal membranes. These pores result in loss of homeostasis and death of the fungal cell. Caspofungin (choice B) is a member of the echinocandins, the newest antifungal antibiotics. These agents disrupt the fungal cell wall by inhibiting the synthesis of beta(1-3) glucan. Fluconazole (choice C) and other azoles (ketoconazole, itraconazole, voriconazole, posaconazole) inhibit ergosterol synthesis. Griseofulvin (choice D) interferes with microtubule function and cell division in fungi. Terbinafine (choice E) inhibits squalene epoxidase in fungi and reduces ergosterol synthesis.

QUESTION 5

A 7-year-old boy is examined by his pediatrician because of complaints of severe cramping pain in his legs whenever he rides his bike. He also expriences nausea and vomiting during these attacks. The child has noted that the severity of the cramps is most intense after dinners that include baked potatoes or pasta, and sometimes bread. Clinical studies undertaken following a treadmill test demonstrate myoglobinuria, hyperuricemia, and increased serum bilirubin. Which of the following enzyme deficiencies is associated with these clinical findings?

A. glucose-6-phosphatase

B. glycogen synthase

C. liver glycogen debranching enzyme

D. muscle phosphofructokinase

E. muscle phosphorylase

Correct Answer: D

Section: Biochemistry A deficiency in muscle phosphofructokinase results in glycogen storage disease type VII (Tarui disease). Clinically, the symptoms seen in Tarui disease are very similar to those seen in muscle phosphorylase deficiency (choice E), glycogen storage disease type V (McArdle disease) such as exercise-induced cramping and early fatigue. There are five clinical characteristics allowing distinction between Tarui and McArdle diseases: exercise intolerance is evident in childhood, is more severe, and is associated with nausea and vomiting; the intolerance is particularly acute following meals rich in carbohydrates; hyperuricemia is more severe; compensated hemolytic anemia is evidenced by increased serum bilirubin and reticulocyte count, and lastly; an abnormal polysaccharide is present in muscle fibers. Deficiency in glucose-6-phosphatase (choice A) is one cause of glycogen storage disease type I (specifically type Ia, von Gierke disease). Classic symptoms of this deficiency include neonatal hypoglycemia and lactic acidosis. If symptoms do not appear until the third or fourth month they include hepatomegaly and hypoglycemic seizures. Liver glycogen synthase deficiency (choice B) presents with morning fatigue and ketotic hypoglycemia on fasting--both of which rapidly disappear on feeding. Symptoms can be rapidly relieved and chemical signs corrected by introducing frequent protein-rich meals and nighttime feedings of suspensions of uncooked corn starch. Deficiency in glycogen debranching enzyme (choice C) results in glycogen storage disease type III (Cori or Forbes disease). Symptoms of type III disease are short stature, variable skeletal myopathy, cardiomyopathy, hepatomegaly, and hypoglycemia.

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