USMLE-STEP-1 Q&As

United States Medical Licensing Step 1

Pass USMLE USMLE-STEP-1 Exam with 100% Guarantee

Free Download Real Questions & Answers **PDF** and **VCE** file from:

https://www.geekcert.com/usmle-step-1.html

100% Passing Guarantee
100% Money Back Assurance

Following Questions and Answers are all new published by USMLE Official Exam Center

- **Instant Download** After Purchase
- **100% Money Back** Guarantee
- **365 Days** Free Update
- **800,000+** Satisfied Customers

QUESTION 1

A 23-year-old man sees his physician to ask about the recent appearance of several large closely spaced bumps on his elbows. Suspecting that these are fatty eruptions, the physician tests the man\\'s blood for lipid, cholesterol, and lipoprotein levels. Results show elevated cholesterol and triglycerides and the presence of a variant form of very low-density lipoprotein (VLDL) identified as beta-migrating VLDL (VLDL). Amore careful analysis of the biochemical properties of the apoproteins associated with the beta-VLDL particles identifies a form of apo E that has a more negative charge than apo E from normal individuals. These results indicate the individual is afflicted with which of the following hyperlipoproteinemias?

A. type I (familial LPL deficiency)

B. type II (FH)

C. type III (dysbetalipoproteinemia)

D. type IV (familial hypertriglycerolemia)

E. Wolman disease

Correct Answer: C

Section: Biochemistry Familial dysbetalipoproteinemia (type III hyperlipoproteinemia) results from a genetic variant in the apo E gene that causes poor interaction of chylomicron remnants and VLDLs with the apo E receptor. This results in the presence, in the serum, of beta-migrating VLDL (- VLDLs), which are cholesterol- rich remnants of both intestinal chylomicrons and hepatic VLDL. Diagnosis of type III hyperlipoproteinemia is indicated by elevated plasma cholesterol and triglyceride, xanthomas (fatty eruptions under the skin), and of course the presence of -VLDL. Type I hyperlipoproteinemia (choice A) results from defects in the activity or activation of LPL and results in the massive accumulation of chylomicrons in the plasma. The disease is usually detected in childhood following recurrent attacks of abdominal pain, hepatosplenomegaly, and pancreatitis. Familial hypercholesterolemia (choice B) is the result of defects in the LDL receptor. The defects lead to characteristic elevation in LDL, deposition of LDL-derived cholesterol in the tendons and skin and in the arteries. Individuals homozygous for defective LDL receptors have severe hypercholesterolemia (6501000 mg/dL) and coronary heart disease begins early in childhood with death caused by myocardial infarct before the age of 20. Type IV hyperlipoproteinemia (choice D) is associated with overproduction VLDLs. An associated glucose intolerance and hyperinsulinemia are also seen in this disorder. Wolman disease (choice E) is caused by a deficiency in lysosomal acid lipase and results in massive accumulation of cholesteryl esters and triglycerides in most tissues. The disease is almost always fatal before the age of 1 year.

QUESTION 2

Which of the following familial cancers results from a defect in the tumor suppressor gene, p53?

A. familial adenomatous polyposis coli (APC)

B. Li Fraumeni syndrome

C. neurofibromatosis type 1

D. retinoblastoma

E. Wilms tumor

Correct Answer: B

Section: Biochemistry Numerous cancers are caused by the loss of function of tumor suppressor genes. LFS is a rare form of inherited cancer that involves breast and colon carcinomas, soft-tissue sarcomas, osteosarcomas, brain tumors, leukemia, and adrenocortical carcinomas. These tumors develop at an early age in LFS patients. The tumor suppressor gene found responsible for LFS is p53. Mutant forms of p53 are found in approximately 50% of all tumors. The normal p53 protein functions as a transcription factor that can induce either cell-cycle arrest or apoptosis (programmed cell death) in response to DNA damage. FAP (choice A) is a rare inherited form of colon cancer. Germline mutations in the APC gene are responsible for FAP. All cases of neurofibromatosis (choice C) arise by inheritance of a mutant allele. Roughly 50% of all affected individuals carry new mutations, which appear to arise paternally, possibly reflecting genomic imprinting. The gene responsible for type 1 neurofibromatosis is termed NF1. Germline mutations at the NF1 locus result in multiple abnormal melanocytes 168 3: Biochemistry (caféau lait spots) and benign neurofibromas. Some patients also develop benign pheochromocytomas and CNS tumors. Asmall percentage of patients develop neurofibrosarcomas, which are likely to be Schwann cell derived. Retinoblastoma (choice D) is a tumor of retinal cells, which develops in children between birth and 4 years of age. The gene responsible is termed the retinoblastoma susceptibility gene (RB) and the protein product pRB. Wilms\\' tumor (choice E) is a form of childhood kidney cancer. The gene responsible for this disease has been identified and is called WT1 (Wilms\\' tumor 1).

QUESTION 3

Exhibit: Please refer to the exhibit. A 14-month-old baby boy is brought to your office by his mother because he seems to be in pain whenever he tries to move. During your physical examination you note bowing of his legs, depression of the sternum with outward projection of the ends of the ribs, reluctance to move his limbs, and numerous bruises on his legs as well as gingival hemorrhages. These findings lead you to suspect that this child suffers from a dietary deficiency of which of the following vitamins?

A. A
B. B_1 (thiamine)
C. B_{12} (cyanocobalamin)
D. C (ascorbate)
E. D (calciferol)
F. K

A. A

B. B

C. C

D. D

E. E

F. F

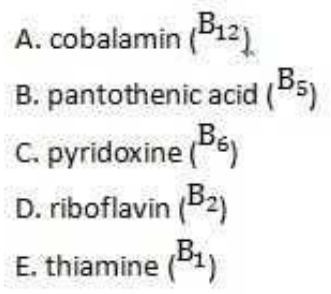
Correct Answer: D

Section: Pathology and Path physiology Deficiencies of vitamin C and vitamin D can produce similar skeletal abnormalities in young children such as those listed. However, a major difference is that vitamin C deficiency is accompanied by hemorrhages, as seen in this child. This also leads to hemarthrosis (bleeding into joints) that makes movement very painful. Vitamin A deficiency (choice A) is associated with night blindness, with or without keratomalacia and papular dermatitis. Vitamin B1 deficiency (choice B) produces beriberi marked by polyneuropathy, heart failure, and edema (or Wernicke syndrome in chronic alcoholics). Vitamin B12 deficiency (choice C) produces megaloblastic anemia

and subacute combined degeneration of the spinal cord. Vitamin D deficiency (choice E) produces osteomalacia in adults and rickets in children due to defective mineralization of bone. Vitamin K deficiency (choice F) can result in a bleeding diathesis because it is required for the activity of clotting factors II, VII, IX, and X.

QUESTION 4

Exhibit:



Please refer to the exhibit. When fatty acids with odd numbers of carbon atoms are oxidized in the beta-oxidation pathway the final product is 1 mole of acetyl- CoA and 1 mole of the 3-carbon molecule, propionyl-CoA. In order to use the propionyl carbons, the molecule is carboxylated and converted ultimately to succinyl-CoA and fed into the TCA cycle. Which of the following represents the vitamin cofactor required in one of the steps of this conversion?

A. A

B. B

C. C

D. D

E. E

Correct Answer: A

Section: Biochemistry Propionyl-CoA is converted to succinyl- CoAin a series of reactions using three different enzymes. It is first carboxylated in an ATPdependent reaction catalyzed by propionyl- CoA carboxylase, an enzyme that requires biotin as a cofactor. The product of the first reaction, d-methylmalonyl-CoA is then converted to lmethylmalonyl-CoA by methylmalonyl-CoA racemase. Finally, methylmalonyl-CoA is converted to succinyl-CoA by the cobalaminrequiring enzyme, methylmalonyl-CoA mutase. None of the other vitamins (choice B, C, D, and E) are required in this pathway.

QUESTION 5

Which of the following inhaled general anesthetic agents has an MAC that exceeds normal atmospheric pressure?

A. enflurane

B. halothane

C. isoflurane

D. nitrous oxide

E. sevoflurane

Correct Answer: D

Section: Pharmacology The MAC value for nitrous oxide is 105110 % (i.e., 510% greater than atmospheric pressure), meaning that for nitrous oxide to be used as the sole anesthetic agent, hyperbaric conditions would have to be used to deliver both the nitrous oxide for anesthesia and oxygen needed for life. The other commonly used inhaled agents, including the halogenated hydrocarbons enflurane, isoflurane, halothane, and sevoflurane (choices A, B, C, and E) have MAC values of 6% or less. Although these data might be interpreted to imply that nitrous oxide is not useful in anesthesia, nitrous oxide is very valuable because of its very low toxicity and additive effects with more potent (but more toxic) agents. It is therefore commonly combined with other inhaled agents, especially the halogenated hydrocarbons. For example, 0.5% MAC of nitrous oxide plus 0.5% MAC of isoflurane delivers a full anesthetic concentration of gases to the lungs but requires