RAD II and DT II

Final term

BIOCHEMISTRY

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B.S Radiology

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Section: B

Write note on following questions each carries equal marks

1) Write down the 4 steps involve in beta oxidation?
   Beta-oxidation comprises of four steps:
   1) Dehydrogenation catalyzed by acyl-CoA dehydrogenase, which eliminates two hydrogens between carbons 2 and 3.
   2) Hydration catalyzed by enoyl-CoA hydratase, which adds water across the double bond.
   3) Dehydrogenation catalyzed by 3-hydroxyacyl-CoA dehydrogenase, which produces NADH.
   4) Thiolytic cleavage catalyzed beta-ketothiolase, which cuts the terminal acetyl-CoA group and forms a new acyl-CoA which is two carbons shorter than the previous one.
   The shortened acyl-CoA then reenters the beta-oxidation pathway

2) Write down clinical significance of the following enzymes

a) Alkaline phosphatase

The popular of sustained elevated ALP levels are related with disorders of the liver or bone, or both. Therefore, these organ systems are of prime consideration in the differential diagnosis.

A variety of primary and secondary hepatic complaints may be linked with elevated serum ALP levels. Since production is increased in reply to cholestasis, serum ALP activity provides a sensitive indicator of obstructive and space-occupying cuts of the liver. The latter includes neoplastic and infiltrative diseases . Bilirubin excretion is compromised only with extensive biliary obstruction or diffuse hepatic cell disruption; therefore, differential elevation of ALP relative to serum bilirubin provides an early indicator for obstructive or space-occupying

conditions. Hepatic cell scratches are manifested by hyperbilirubinemia and dominant serum elevation of parenchymal enzymes, such as aminotransferases; ALP elevations may be only negligible.

b) Creatine kinase

Two of the three main isoenzymes of creatine kinase (CK), MM and MB, experience postsynthetic adaptation resulting in the creation of five isoforms. On the basis of their relative electrophoretic migration, the isoforms are designated MM3, MM2, MM1 MB2, and MB1 . CK isoform formation is a naturally occurring phenomenon mediated by carboxypeptidase (CPase) N. CPase N cleaves the carboxy-terminal amino acid, lysine, from the M subunits of MM3 to form MM2 and MM1 and from the M subunit of MB2 to form MBV MM3 and MB2 are tissue isoforms, and MM2, MM1, and MB1 are formed after the tissue isoforms are released into the circulation. The CK isoforms can be used as markers for confirmation of an acute myocardial infarction (AMI) and for successful reperfusion of AMI patients treated with thrombolytic therapy. Both MM3 and MB2 are significantly increased in the blood shortly after an AMI. Total MM3 and MB2 and the MM3/MM1 and MB2/MBl ratios are all elevated subsequent to AMI and prior to total CK and CK-MB levels. The CK-MB isoforms are superior to the MM isoforms for diagnosing AMI and reperfusion because they are more myocardial tissue specific. The CK isoforms can be assayed using electrophoresis, isoelectric focusing, chromatofocusing, highperformance liquid chromatography, and immunoinhibition. Prompt, complete, and delicate electrophoresis procedures allow CK isoform analysis to be used for diagnosis and treatment of patients as early as 2 to 6 hours after AMI.

c) gamma-glutamyl transferase

Gamma-glutamyltransferase (GGT) is chiefly present in liver, kidney , and pancreatic cells. Little amounts are present in other tissues. Even though renal tissue has the highest level of GGT, the enzyme present in the serum seems to initiate primarily from the hepatobiliary system, and GGT activity is raised in any and all forms of liver disease. It is maximum in cases of intra- or posthepatic biliary obstruction, reaching levels some 5 to 30 times normal. GGT is more sensitive than alkaline phosphatase (ALP), leucine aminopeptidase, aspartate transaminase, and alanine aminotransferase in detecting obstructive jaundice, cholangitis, and cholecystitis; its rise occurs earlier than with these other enzymes and persists longer. Only ordinary increases occur in infectious hepatitis, and in this condition, GGT determinations are less useful diagnostically than are measurements of the transaminases. High increases of GGT are also observed in patients

with either primary or secondary (metastatic) neoplasms. High levels of GGT are noted not only in the sera of patients with alcoholic cirrhosis but also in the majority of sera from persons who are heavy drinkers. Studies have stressed the value of serum GGT levels in detecting alcohol-induced liver disease. Elevated serum values are also perceived in patients receiving drugs such as phenytoin and phenobarbital, and this is thought to reflect induction of new enzyme activity.

Normal values are seen in different muscle diseases and in renal failure. Normal values are also seen in cases of skeletal disease, children older than 1 year, and in healthy pregnant women-conditions in which ALP is higher.

3) How many proteins are involve in electron transport chain and how do electrons move in the electron transport chain?

The electron transport chain (ETC) is a sequence of complexes that transmist electrons from electron donors to electron acceptors through redox (both reduction and oxidation occurring simultaneously) reactions, and links this electron transfer with the transfer of protons (H+ ions) across a membrane. The electron transport chain is built up of peptides, enzymes, and other molecules.

There are four complexes composed of proteins, and the aggregation of these four complexes, together with associated mobile, accessory electron carriers, is called the electron transport chain. The electron transport chain is present in numerous copies in the internal mitochondrial membrane of eukaryotes and the plasma membrane of prokaryotes. Note, though, that the electron transport chain of prokaryotes may not require oxygen as some live in anaerobic conditions. The usual feature of all electron transport chains is the existence of a proton pump to build a proton gradient across a membrane.

In the electron transfer chain, electrons pass along a succession of proteins to generate an ejection type force to move hydrogen ions, or protons, across the mitochondrial membrane. The electrons start their reactions in Complex I, ongoing onto Complex II, crossed to Complex III and cytochrome c via coenzyme Q, and then lastly to Complex IV. The complexes themselves are complex-structured proteins fixed in the phospholipid membrane. They are united with a metal ion, such as iron, to help with proton removal into the intermembrane space as well as other functions. The complexes also experience configurationally changes to let openings for the transmembrane movement of protons. These four complexes keenly transmit electrons from an organic metabolite i.e glucose. When the metabolite breaks down, two electrons and a hydrogen ion are released and then picked up by the coenzyme NAD+ to become NADH, releasing a hydrogen ion into the cytosol.

4) Write down the steps involved in uric acid formation

Beta-oxidation comprises of four steps:

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2) Hydration catalyzed by enoyl-CoA hydratase, which adds water across the double bond.

3) Dehydrogenation catalyzed by 3-hydroxyacyl-CoA dehydrogenase, which produces NADH.

4) Thiolytic cleavage catalyzed beta-ketothiolase, which cuts the terminal acetyl-CoA group and forms a new acyl-CoA which is two carbons shorter than the previous one.

The shortened acyl-CoA then reenters the beta-oxidation pathway.

5) How uric acid formation takes place in body?

Uric acid is a waste product created during the normal collapse of purines, naturally occurring substances found in foods for example mushrooms, liver, anchovies, mackerel and dried beans according to the NIAMS. Uric acid is normally washed out of the blood by the kidneys, and passes out of the body through urine.

Hyperuricemia is referred as excess of uric acid in the blood. Uric acid passes through the liver, and enters your bloodstream. Most of it is expelled (removed from your body) in your urine, or passes through your intestines to regulate "normal" levels.

Normal Uric acid levels are 2.4-6.0 mg/dL (female) and 3.4-7.0 mg/dL (male). Normal values will differ from laboratory to laboratory.