

Liver pathology Programmed control

1. Hepatic coma is characteristic of:
 1. disorientation in time and space
 2. cramps
 3. increased urea blood level
 4. increased blood ammonia
 5. high prothrombin index
 6. hypokalemia with extracellular alkalosis
2. Hepatic coma is characterized by:
 1. increased blood urea
 2. increased prothrombin index
 3. high ratio albumins/globulins
 4. indol and scatol accumulation in blood
 5. appearance of false neuromediators in blood
 6. hemorrhagic syndrome
3. Point to characteristic changes in metabolism of patient with hepatic insufficiency:
 1. hypoalbuminemia
 2. dysproteinemia
 3. low level of blood aminoacids
 4. increased blood urea
 5. aminoaciduria
 6. high blood oncotic pressure
4. Disturbances in protein metabolism in liver insufficiency seem to be the following:
 1. decreased blood level of aminoacids
 2. aminoaciduria
 3. increased level of ammonia in blood
 4. increased blood fibrinogen
 5. transferrin deficiency
 6. increased blood oncotic pressure
5. Cholestatic syndrome is characteristic of following types of jaundice:
 1. prehepatic (hemolytic)
 2. intrahepatic (parenchymatous)
 3. posthepatic (mechanical)
6. Acholia appears in the following types of jaundice:
 1. prehepatic
 2. intrahepatic
 3. posthepatic
7. Cholemia is possible when the following types of jaundice:
 1. prehepatic
 2. intrahepatic
 3. posthepatic
8. Posthepatic jaundice reveals itself in form of:
 1. lemon-yellow tint of skin
 2. increased non-conjugated blood bilirubin
 3. appearance of conjugated bilirubin in the blood
9. Posthepatic jaundice reveals itself in form of:
 1. lemon-yellow tint o skin
 2. increased non-conjugated blood bilirubin
 3. bradycardia
 4. steatorrhea
 5. creatorrhea
 6. increased blood urea
10. Hepatic encephalopathy is associated with the following factors accumulation in the blood:
 1. toxins including ammonia from the gut
 2. urea
 3. conjugated bilirubin
 4. false neuromediators
 5. short-chained fatty acids
 6. GABA
11. High protein diet in patient with portosystemic shunting could provoke unfavorable consequences in course of liver disease?
 1. yes
 2. now
12. In posthepatic jaundice the following substances give urine dark color:
 1. conjugated bilirubin
 2. non-conjugated bilirubin
 3. urobilin
 4. sterkobilin
 5. cholesterol
13. Blood contents of following enzymes are growing up in hepato-cellular form of jaundice:
 1. alanin-aminotransferase
 2. 5 -nucleosidase
 3. alkaline phoshatase
 4. aspartat-aminotransferase
14. When cholestasis the following blood enzymes are growing up:
 1. alkaline phoshatase
 2. 5 -nucleosidase
 3. alanin-aminotransferase
 4. aspartat-aminotransferase
15. Dark color of urine in patient with prehepatic jaundice associated with presence in urine:
 1. urobilin
 2. non-conjugated bilirubin
 3. sterkobilin
 4. conjugated bilirubin
16. Mixed form of jaundice reveals itself by the following enzymes high activity:
 1. alkaline phoshatase
 2. 5 -nucleosidase
 3. alanin-aminotransferase
 4. aspartat-aminotransferase
17. The following shifts in blood biochemistry may be evidences of mixed form of hepatic jaundice:
 1. increased conjugated bilirubin
 2. increased urea
 3. hypoalbuminemia
 4. high activity of aminotransferases
 5. increased non-conjugated bilirubin
 6. high activity of alkaline phosphatase

18. The symptoms of mixed form of hepatic jaundice seem to be the following:
1. hyperglycemia
 2. increased blood alkaline phosphatase
 3. cholemia
 4. decreased prothrombin index
 5. hypercholic stool
19. Point to the causes of primary cholestasis:
1. obstruction of common bile duct
 2. increased bile density in dehydration
 3. tumor of pancreatic head
 4. cholangitis
 5. infectious hepatitis
20. Possible causes of secondary cholestasis are:
1. dehydration of an organism
 2. obstructio of common bile duct
 3. cholangitis
 4. infectious edema of Vater's papilla
 5. infectios hepatitis

Tests of second level

21. Give an explanation of such symptoms as:gynecomastia, testes atrophia, and decreased libido in men with liver cirrhosis
22. The main symptoms of portal hypertention are:
1...2...3...4...5...
23. Mechanisms of A,D,E,K avitaminosis in patient with acholia are the following:
1...2...3...
24. Direct bilirubin could be revealed in urine when its level in blood is more than? ...
25. What tint of skin covers is characteristic if hepatic jaundice? ...
26. List below the symptoms of cholemia:
1...2...3...4...5...
27. Why in a shaking of urine of patient with light form of hepatic jaundice it is darker and more foaming than urine of patient with heavy form? ...
28. Which symptom may be revealed on skin surface of anterior abdomenwall of patient with liver cirrhosis?
29. Which minimal level of blood bilirubin is accompanied by appearance of skin yellow tint? ...
30. Main signs of acholia are:
1...2...3...4...
31. Name the cerebrotoxins which can provoke an acute liver insufficiency:
1...2...3...4...
32. Give an explanation of hepato-lienal syndrome in patient with liver chirrrosis
1. ...2...3...
33. list the types of portal hypertension:
1...2...3...
34. The main pathogenetic features of hemorrhagic syndrome developing in patient with portal hypertension are:
1...2...3...

35. List the mechanisms involving in ascytes formation in a patient with portal hypertension:
1...2...3...4...5...
36. What tint of skin is characteristic of posthepatic jaundice?
37. What tint of skin is characteristic of prehepatic jaundice?
38. What are the mechanisms of false mediator action to CNS in patients with acute liver insufficiency?
1...2...3...
39. Explain pathogenesis of such symptoms as:
1.jaundice 2. specific odor from the mouth 3. hemorrhagic syndrome observing in a patient with hepatic coma
40. Case. Patient B. is delivered to emergency department with the triad of symptoms: jaundice, multiple skin hemorrhages and mercaptan odor out the mouth in breathing.
1. Which pathology has got the patient?
2. Explain pathogenesis of described symptoms.