General Pathophysiology

PROTEIN METABOLISM

1. The degradation of proteins is selectively disturbed when there is deficit of (pick the most complete answer):

   1. Saliva, amylase, lipase, carboxypeptidases.


   4. Pepsinogen, amylase, lipase, hydrochloric acid.

   5. Hydrochloric acid in the stomach and bicarbonates in the duodenum.

2. Impaired degradation and absorption of proteins is not present in:

   1. Diseases of the stomach.

   2. Diseases of the pancreas.

   3. Disturbance in the endocrine function of the pancreas.

   4. Disturbed motion of the gastro-intestinal tract.

   5. Malnutrition with essential amino acids.

3. In patients with coeliac disease is present:


   2. Inability to degrade the gliadin component of gluten in cereals.

   3. Pepsin and trypsinogen deficiency.


   5. 1, 2, 4.
4. What are the consequences of disturbed degradation and absorption of proteins in the GIT:

1. Hypoproteinemia, hypoalbuminemia, edemas.
2. Hypoproteinemia, hyperferritinemia, anemia.
3. Dysproteinemia with hypervolemia.
4. Elevated level of glycosylated Hb.
5. Micromolecular paraproteinemia.

5. Under the influence of the intestinal bacteria the undegraded and unabsorbed proteins are subjected to:

1. Fermentation.
2. Decay.
4. Halytosis.
5. Steatosis.

6. What is not a consequence of plasma protein levels disturbance:

1. The colloid osmotic pressure of blood.
2. The transport of lipids, hormones, iron, calcium, bilirubin etc.
3. Proton buffering.
4. The defensive function of blood.
5. Coagulation process.

7. Hypoproteinemia is usually due to:

1. Decreased albumins.
2. Decreased fibrinogen.
3. Decreased α-globulins.
4. Decreased α- and β- globulins.
5. Decreased glycoproteins.

8. Which of the following is not accompanied by hypoproteinemia:
   1. Protein malnutrition, disturbed degradation and absorption.
   2. Liver diseases.
   3. Increased degradation of proteins in the organism.
   4. Increased protein loss through the kidneys and the GIT.
   5. Dehydration.

9. Hyperproteinemia is most often due to:
   1. α1 - globulins.
   2. Fibrinogen.
   3. Albumins.
   4. γ-globulins or paraproteins.
   5. α1 –antitrypsin

10. Dysproteinemia as a clinical finding means:
    1. Low proteins.
    2. High albumins.
    3. Altered ratio between the different fractions of plasma proteins.
    4. The presence of pathologic proteins.
    5. “Debut” of inflammatory proteins.
11. In hyperammonemia most active in the detoxication of ammonia are:
   1. The lungs.
   2. The skin.
   3. The bones.
   4. The striated muscles.
   5. The cartilages.

12. The striated muscles detoxify ammonia through:
   1. Aromatic amino acids.
   2. Ketogenesis.
   3. Lactic acid in the muscle cells.
   5. Ornithin cycle.

13. After the full detoxificating capacity of ammonia in the muscles is reached pathogenetically important is the detoxification in:
   1. The brain.
   2. Белите дробове.
   3. The skin.
   4. The bones.
   5. The cartilages.

14. The theory about the “energy depletion” in the brain in hyperammonemia is related to:
   1. Transformation of valine, isoleucine and leucine into α-keto acids.
   2. Transformation of α-ketoglutarate into glutamate and glutamine.
   3. Transformation of phenylalanine into phenylpyruvic acid.
4. Compulsory oversynthesis of acetylcholine.

5. Glutamate and aspartate deficiency.

15. The toxic effect of hyperammonemia to CNS is related mostly to:

1. Stable blockage of the N-type voltage dependent Ca 2+ channels.

2. Inactivation of the mechanism to excrete Cl from the neurons.

3. Suppression of the K+/Na+ pump.

4. Suppressed presynaptic captation of neurotransmitters.

5. Increased K+ excretion from the neurons.

16. When there are elevated levels of nitrogen in the blood, what is actually elevated:

1. Nitrogen contained in proteins.

2. Nucleoproteins.


5. Biogenic amines.

17. The different types of elevated nitrogen levels in blood are:

1. Productive.

2. Due to inactivation.

3. Retentive.


5. 1, 3, 4.

6. 1, 2, 3, 4.
18. Productive hypernitrogenemia is not characteristic for:

1. Liver failure.
2. Neoplasms.
3. Haemolytic anemias.
4. Chronic renal failure.
5. Tissue degradation.

19. A key pathogenetic factor in gout is:

1. Hyperammonemia.
2. Hyperuricemia.
3. Hyperglycemia.
4. Hypercalcemia.
5. Hypercapnia.

20. Gout is a disease related to:

1. Disturbance in the metabolism of hemoglobin.
2. Disturbance in the metabolism of lipoproteins.
3. Disturbance in the metabolism of tyrosine.
4. Disturbance in the metabolism of purines.
5. Disturbance in the metabolism of long chain fatty acids.

21. Increased production of uric acid is due to:

1. Defect in the enzyme hypoxanthine-guanine phosphoribosyltransferase.
2. Decreased inhibition of glutamine phosphoribosylpyrophosphate amidotransferase.
3. Defect in glucose-6-phosphatedehydrogenase.
22. Secondary gout develops when there is:

1. Increased production of nucleic acids.
2. Decreased excretion of nucleic acids through the kidneys.
3. Increased production of aromatic amino acids.
4. Increased production of branch chain amino acids.
5. 1, 2.
6. 3, 4.

23. In the basis of the gout exacerbation lies:

1. A distrophic process.
2. An inflammatory process.
3. A necrotic process.
4. An atrophic process.
5. A hypertrophic process.

24. Disturbance in the middle stages of protein metabolism is present when there is a problem in:

1. Oxidative desamination.
2. Decarboxylation and transamination.
3. β-hydroxy - β-methylglutaryl CoA-cycle in the liver.
4. 1, 2.
5. 1, 2, 3.
25. Phenylketonuria is a result of disturbed function of:
   1. Glucose-6-phosphatedehydrogenase.
   2. Phosphoribosyltransferase.
   4. Pyruvatkinase.
   5. Phenylalaninehydroxylase.

26. Phenylketonuria is a reason for:
   1. Severe damage of CNS.
   2. Albinism.
   3. Myxedema.
   4. Pituitary nanism.
   5. Lung emphysema.

27. Disturbed metabolism of tyrosine leads to:
   1. Alkaptonuria.
   2. Tyrosinuria.
   4. Myoglobinuria.
   5. 1, 2.
   6. 1, 3, 4.

28. Hyperaminoaciduria is a consequence of:
   1. A transport defect in the renal tubules
   2. Tubular oversecretion of amino acids.
3. Hyperaminoacidemia over the threshold.

4. Increased postnephronal diffusion of amino acids.

5. 1, 3.

6. 2, 3, 4