General pathophysiology

LIPID METABOLISM

1. Alimentary chylomicronemia is sustained in:
   1. Lipoprotein lipase deficiency.
   2. Inhibited lipoprotein lipase.
   3. Hypoalbuminemia.
   4. Hyperalbuminemia.
   5. 1, 2, 3.
   6. 1, 2, 4.

2. Direct source of LDL are:
   1. VLDL.
   2. IDL.
   3. HDL.
   4. VLDL and HDL.
   5. Chylomicrones.

3. The main pathogenic factor for atherosclerosis is:
   1. Blood hyper-LDL.
   2. Blood hyper-VLDL.
   3. Blood hypo-HDL.
   4. Hyperproteinemia.
   5. Hypercoagulation.

4. The main reason for the atherogenic effects of LDL is:
   1. Their cholesterol ester content
   2. Their TriAcylGlycerols (TAGs) content.
   3. Their oxidized form (oxy-LDL)
   4. Their protein component.
   5. Their phospholipids content.

5. The main reason for the atherogenic effects of oxy-LDL is their ability to:
   1. Form interplatelet bounds.
   2. Damage the endothelial membrane.
   4. Induce dysproteinemia.
   5. 2, 3.
   6. 1, 2, 4.

6. Macrophages are capable of phagocytizing oxy-LDL due to:
   1. Chemotactic factors
   2. Lipid peroxides produced subendothelially by the arterial wall intimal layer.
   3. Specific receptors for oxy-LDL detection
   4. Cytokines produced by the arterial wall intima.
7. "Foam cells" are:
   1. Specialized cleaning LDL-macrophages.
   2. Transformed endothelial cells.
   3. Fixed multinucleate cells.
   4. Activated multipotent cells.
   5. Modified platelets.

8. Where does atherosclerotic plaque develop?
   1. In the subendothelial space of the arterial wall.
   2. In the medial layer of the arterial wall.
   3. Bellow the medial layer of the arterial wall.
   4. Bellow the adventitious layer of the arterial wall.
   5. In the adventitious layer of the arterial wall.

9. Reversibility of the atherosclerotic plaques is determined mainly by:
   1. The presence of "Foam cells"
   2. The presence of smooth muscle cells.
   3. The presence of extracellular collagen
   4. Platelet adhesion.
   5. Plaque capillarization.

10. Conditions for ketonemia are:
    1. Increased mobilization of the free fatty acids (FFA) from fat depots.
    2. Depressed beta-oxidation in the muscles
    3. Krebs cycle activation in the liver.
    4. β-hydroxy-β-methylglutaryl CoA cycle activation in the liver.
    5. 1, 4.
    6. 1, 2, 3.

11. Which plasma lipid constellation represents the highest atherogenic risk?
    1. Hyperchylomicronemia and hypoHDL-lipoproteinemia.
    2. HyperLDL and hyperHDL lipoproteinemia.
    3. Hyper VLDL, LDL and HDL lipoproteinemia.
    4. HyperLDL and hypo HDL lipoproteinemia
    5. HypoHDL and hypoLDL lipoproteinemia.

12. In pathophysiologic aspect obesity is divided into:
    1. Hypertrophic and atrophic obesity
    2. Aplastic, hypoplastic and hyperplastic obesity
    3. Obesity with increased volume of fat, decreased volume of fat and with disturbed fat distribution.
    4. Alimentary, regulatory and metabolic obesity.
5. Alimentary obesity, obesity due to decreased physical activity, hereditary obesity.

13. Hyperinsulinemia leads to obesity by:
   1. Stimulating the production of glycerol-3-phosphate.
   2. Pentose cycle (NADPH) activation.
   3. Increased acetyl CoA synthesis.
   4. Inhibiting the activity of hormone-sensitive lipase.
   5. 1, 2, 4.
   6. 1, 2, 3, 4.

14. Blocked VLDL formation in the liver leads to:
   1. Cirrhosis
   2. Lipid dystrophy
   3. Hemochromatosis
   4. Hepatocytic regeneration.
   5. Hepatocytic apoptosis.

15. VLDL synthesis in the hepatocytes is impaired in:
   1. Suppressed apoprotein synthesis.
   2. Lipid/apoprotein decomposition.
   3. Impaired VLDL secretion.
   4. Lipid (TG, PhL, Cholesterol) synthesis dissociation in hepatocytes.
   5. 1, 2, 3, 4.

16. Which hormone ratio determines liver ketogenic potential:
   1. Glucocorticosteroids/thyroxine.
   2. Glucagon/insulin.
   3. Tropichormones/somatomedin.
   5. Renin/plasmin.

17. Ketonemia is a manifestation of:
   1. Increased ketogenesis in the liver.
   2. Keto-production from adipocytes.
   3. Suppressed extrahepatic ketolysis.
   4. Blocked hepatic ketolysis.
   5. 1, 3.
   6. 1, 2, 4.

18. Receptor-independent pathway of elimination of plasma LDL is mainly presented in:
   1. Adipocytes.
   2. Fibrocytes.
   3. Mononuclear phagocyte system.
   4. Myofibers.
   5. Epithelium.

19. Which are the mechanisms that protect cells from accumulating cholesterol?
   1. Own cholesterol synthesis (HMG-CoA reductase) inhibition.
2. Increased esterification of free cholesterol (AHA).
3. Hiding (decomposition) of LDL-receptors and decreased synthesis
4. Increased cholesterol export – contact with HDL3
5. 1, 2, 3.
6. 1, 3, 4

20. The antiatherogenic effect of HDL is associated with:
   1. Adsorption, esterification and transport of cell cholesterol to the liver.
   2. Inhibition of LDL oxidation.
   3. Prolongation and enhancing the effects of prostacyclin.
   4. Binding and inhibition of bacterial lipopolysaccharides
   5. 1, 2, 3, 4.

21. Mandatory trigger of atherogenic vascular damage is:
   1. Endothelial dysfunction.
   2. Hyperlipoproteinemia.
   3. Hyperuricemia.
   5. Pericytes remodeling.

22. Atherogenic endothelial dysfunction is associated with:
   1. Increased endothelial permeability.
   2. Reduced platelet resistance.
   3. Increased adhesion of blood cells.
   4. 1, 2.
   5. 1, 2, 3.

23. Endothelial dysfunction (caused by hyperlipoproteinemia) is a result of:
   1. Increased endothelial membrane cholesterol.
   2. Increased rigidity of the endothelial cells.
   3. Endothelial separation and restriction.
   4. Increased endothelial permeability.
   5. 1, 2, 3, 4.

24. Exogenous hyperlipidemias:
   1. Hyper HDL – lipoproteinemia.
   2. Hyperchylomicronemia.
   3. Hyper LDL – lipoproteinemia.
   5. Hyper IDL – lipoproteinemia

25. Regarding the lipoprotein-lipase NaCl acts as a:
   1. Cofactor.
   2. Inhibitor.
   3. Activator.
   4. Signal modulator.
   5. NaCl does not affect the activity of LPL.
26. How does hypoalbuminemia lead to hyperlipidemia?
   1. Impaired LPL secretion.
   2. Enhanced LPL.
   3. **Incomplete acceptance of the released FFA (free fat acids).**
   4. Impaired LPL binding to lipoproteins
   5. Stabilizing the structure of chylomicrones.

27. Which is the inhibitor of LPL during cholestasis?
   1. Bilirubin.
   2. **Bile salts.**
   3. ALP (alkaline phosphatase).
   4. ASAT and ALAT.
   5. Cholesterol.

28. Inadequate and/or delayed leptin secretion leads to:
   1. Redistribution of triglycerides between adipocytes.
   2. Loss of triglycerides from adipocytes.
   3. Appetite suppression.
   4. **Accumulation of triglycerides in adipocytes.**
   5. Activation of the satiety center.

29. Secretion of leptin leads to:
   1. Enhanced lipogenesis.
   2. Direct stimulation of lipolysis.
   3. **Regulation of the relationship between lipolysis and lipogenesis.**
   4. Activation of hormone-sensitive lipase.
   5. Stimulation of catecholamine beta-receptors.

30. The amount of leptin in the circulation correlates with:
   1. Physical capacity.
   2. **Adipose tissue volume.**
   3. Visceral organs size.
   4. Pituitary tropichormones.
   5. Lipoproteins concentration.

31. What is the most characteristic behavior for Homo sapiens regarding obesity?
   1. To control his food biorhythms.
   2. To regulate satiety.
   3. **To eat without being hungry.**
   4. To eat without chewing.
   5. To stimulate his sense of hunger.