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.
    3. Stimulate macrophagephagocytosis in subendothelium.
    4. Induce dysproteinemia.
    5. 2, 3.
    6. 1, 2, 4.

6. Macrophages are capable of phagocyting oxy-LDL due to:
    1. Chemotactic factors
    2. Lipid peroxides produced subendothelially by the arterial wall intimal layer.
    3. Specific receptors for oxy-LDLdetection
    4. Cytokines produced by the arterial wall intima.

5. 1, 2, 4.

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 theatherosclerotic plaque 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:
   1Increased mobilizationof 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 CoAcycle activationin the liver.
   5. 1, 4.
   6. 1, 2, 3.

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

12. In pathophysiologic aspect obesity is divided into:
   1. Hypertrophicandatrophic obesity
   2. Aplastic, hypoplasticandhyperplastic obesity
   3. Obesity with increased volume of fat, decreased volume of fat and with disturbed fat distribution.
   4. Alimentary, regulatory andmetabolic obesity.

5. Alimentary obesity, obesity due to decreased physical activity, hereditary obesity.

13. Hyperinsulinemia leads to obesity by:
    1. Stimulatingthe production ofglycerol-3-phosphate.
    2. Pentosecycle ($NADPH_2$) activation.
    3. IncreasedacetylCoA synthesis.
    4. Inhibiting the activityofhormone-sensitive lipase.
    5. 1, 2, 4.
    6. 1, 2, 3, 4.

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

15. VLDLsynthesis 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:
    1Glucocorticosteroids/thyroxine.
    2. Glucagon/insulin.
    3. Tropichormones/somatomedin.
    4. Catecholamines/glucocorticosteroids.
    5. Renin/plasmin.

17. Ketonemiais a manifestation of:
    1. Increasedketogenesisin the liver.
    2. Keto-production from adipocytes.
    3. Suppressedextrahepaticketolysis.
    4. Blockedhepaticketolysis.
    5. 1, 3.
    6. 1, 2, 4.

18. Receptor-independent pathway of eliminationof plasmaLDLismainly presentedin:
    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 cholesterolsynthesis(HMG-CoA reductase) inhibition.

2. Increasedesterificationof freecholesterol (AHA).
3. Hiding(decomposition) of LDL-receptors anddecreased synthesis
4. Increasedcholesterolexport – 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. Mandatorytriggerofatherogenicvascular damageis:
1. Endothelialdysfunction.
2. Hyperlipoproteinemia.
3. Hyperuricemia.
4. Structuralvascular"injury" - lesion.
5. Pericytes remodeling.

22. Atherogenicendothelial dysfunctionis associated with:
1. Increasedendothelialpermeability.
2. Reducedplateletresistance.
3. Increasedadhesionof 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. Increasedrigidityof theendothelial cells.
3. Endothelialseparation andrestriction.
4. Increasedendothelialpermeability.
5. 1, 2, 3, 4.

24. Exogenoushyperlipidemiais:
1. Hyper HDL – lipoproteinemia.
2. Hyperchylomicronemia.
3. Hyper LDL – lipoproteinemia.
4. Hyper VLDL – 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. Incompleteacceptanceof the releasedFFA (free fat acids).
    4. Impaired LPL binding to lipoproteins
    5. Stabilizingthe structure ofchylomicrones.

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. Inadequateand /or delayedleptin secretionleads to:
    1. Redistribution oftriglyceridesbetweenadipocytes.
    2. Lossof triglyceridesfromadipocytes.
    3. Appetite suppression.
    4. Accumulation of triglyceridesin adipocytes.
    5. Activationof thesatietycenter.

29. Secretion of leptin leads to:
    1. Enhancedlipogenesis.
    2. Directstimulation of lipolysis.
    3. Regulation ofthe relationship betweenlipolysisandlipogenesis.
    4. Activation ofhormone-sensitive lipase.
    5. Stimulattion ofcatecholaminebeta-receptors.

30. The amountof leptinin the circulationcorrelateswith:
    1. Physical capacity.
    2. Adipose tissue volume.
    3. Visceralorgans size.
    4. Pituitarytropichormones.
    5. Lipoproteins concentration.

31. What is the most characteristic behavior for Homo sapiens regarding obesity?
    1. To controlhis foodbiorhythms.
    2. To regulatesatiety.
    3. To eatwithout beinghungry.
    4. To eatwithoutchewing.
    5. To stimulatehis sense ofhunger.