General pathophysiology

CARBOHYDRATE METABOLISM

1. Blood glucose cannot be sensed by the β-cells of the Langerhans islets when there is impairment of:

    1. GLUT-4.
    2. GLUT -2.
    3. GLUT -1.
    4. GLUT -3.
    5. GLUT -9

2. Important pathogenetic stages of DM type I are:

    1. Genetic predisposition to damaging the β-cells.
    2. Viral infections.
    3. Autoimmune mechanism.
    4. Insulin resistance.
    5. 1, 2, 3.
    6. 1, 2, 4.

3. In DM type I there could be damages in the following chromosomes:

    1. 9, 22.
    2. 6, 11.
    3. 7, 12.

4. 2, 8.

5. X, У.

4. Important risk factors for development of DM type II are:

1. Obesity, family predisposition.

2. Hyperkinetic lifestyle, normosomnia.

3. Hypouricemia, cachexia, family predisposition.

4. Smoking.

5. Often viral infections.

5. In DM the plasma levels of FFA are elevated because of:

1. Activation of the hormone-sensitive triacylglycerol-lipase.

2. Inhibition of the resynthesis of the triacylglycerols.

3. Activation of cholesterol synthesis.

4. 1, 2.

5. 1, 2, 3.

6. The hypertriacylglycerolemia in diabetic patients is mostly due to:

1. Increased synthesis of VLDL in the liver.

2. Inhibition of the lipoprotein lipase.

3. Hyperchylomicronemia.

4. 1, 2, 3.

5. 1, 2.

7. The activated gluconeogenesis in DM is mainly regarding the amino acid:

1. Alanine.
2. Valine.
3. Leucine.
4. Isoleucin.
5. Glutamine.

8. The high level of cholesterol in DM is due to:
    1. Activated synthesis.
    2. Inhibited degradation.
    3. Disturbed utilization.
    4. Unknown reason.
    5. 1, 2, 3.

9. Major pathogenetic stages in the vicious circle of diabetic ketoacidotic coma:
    1. Hyperketonemia hypovolemia.
    2. Adynamia, hypothermia.
    3. Glucosuria, polydipsia.
    4. Tachycardia, somnolence.
    5. All of the above.

10. Peripheral neuropathy in DM is due to:
    1. Accumulation of sorbitol and fructose.
    2. Myoinositol deficiency.
    3. Elevated plasma concentration of FFAs.

4. 1, 2.

5. 1, 2, 3.

11. Which regulatory constellation leads to hypoglycemia:

1. Hyperinsulinism and hypercontrainsulinism.

2. Hypoinsulinism and hypercontrainsulinism.

3. Hypoinsulinism and hypocontrainsulinism.

4. Hyperinsulinism and hypocontrainsulinism.

5. 1, 4.

6. 2, 3.

12. Hypoglycemia could be a result of:

1. Suppressed intestinal glucose absorption.

2. Suppressed glycogenolysis in the liver.

3. Decreased gluconeogenesis.

4. Increased glucose uptake in the insulin-dependent tissues.

5. All of the above.

13. Which HLA haplotypes present a high risk for developing DM type I:

1. DR1 and DR2.

2. DR3 and DR4.

3. DR5 and DR6.

4. DR7 and DR8.

5. DR9 and DR10.

14. Which mechanism lies at the basis of developing DM type I:

1. Increased destruction of insulin in the liver.
2. Autoimmune-provoked destruction of the **β**-cells.
3. Production of abnormal insulin.
4. Paralysis of the β-cells with insulin-dependent destruction.
5. Ischemic destruction of the β-cells.

15. Select the mechanisms that lead to insulin resistance:

1. Abnormal (count, structure) insulin receptors.
2. Inefficient glucose-stimulated insulin secretion.
3. Post-receptor suppression of the insulin signal.
4. Increased extraction and secretion of insulin from the tissues.
5. 1, 3.
6. 1, 2, 4.

16. The “vulnerability” of the β-cells in genetic predisposition to DM is presented as:

1. Unmotivated β-cell apoptosis.
2. Increased sensitivity to external provocateurs (viruses, toxins).
3. Autochthonic autoimmune β-cellular rejection.
4. Hypoxic β-cellular hypersensitivity.
5. Normoglycemic β-cellular vulnerability.

17. The insulin resistance in DM type II is:

1. Genetically determined lack of insulin action.
2. Lowered insulin action.
3. Distorted cellular effect of insulin.
4. New, unexpected insulin effect.
5. Toxic-dependent universal loss of insulin sensitivity.

18. Major pathogenetic stage of the disturbed carbohydrate metabolism in DM is:

1. β-cellular vulnerability.
2. Variations in plasma glucose levels.
3. Stable hyperglucosemia.
4. The peak of intercurrent hyperglucosemia.
5. The levels of non-glucose carbohydrates.
6. Insulinemia.

19. What are the metabolic defects typical for DM type II:

1. Formation of insulin resistance.
2. Increased engagement of glucose as a source of energy.
3. Disturbed insulin secretion after glucose loading.
4. More easily facilitated intestinal absorption and tubular reabsorption of glucose.
5. 1, 3.
6. 1, 2, 4.

20. DM with normo- or hyperinsulinemia is an indication of:

1. Compensated DM.

2. Neurovegetative hyperglycemia.

3. Elongated half-life of insulin.

4. Insulin resistance that is present.

5. Development of non-insulin-dependent glucose metabolism.

6. 1, 3.

21. It is pathognomonic for a patient with DM to have:

1. Absolute or relative insulin deficiency.

2. Obligatory hypercontrainsulinemia.

3. Stable hyperglucosemia.

4. Obesity.

5. 1, 3.

6. 1, 2, 4.

22. Which is the major pathogenetic element that is common for the different types of DM:

1. Hypoinsulinemia.

2. Hyperglucagonemia.

3. Hyposomatostatinism.

4. Hypercontrainsulinism.

5. Hypercontrainsulinemia.

6. Hypoinsulinism.

23. Stable diabetic hyperglycemia is a result of:

1. Overproduction of glucose in the liver.

2. Increased muscular glycogenolysis.

3. Difficulties in glucose utilization in the tissues.

4. Increased glucose consumption and absorption.

5. 1, 3.

6. 2, 3, 4.

24. What mechanisms are involved in hyperglycemic toxicity:

1. Non-enzymatic glycosylation of proteins.

2. Activation of the polyol pathway of glucose degradation.

3. Hyperglycemic stimulation of glycolysis.

4. Domination of the hexose monophosphate shunt.

5. 1, 2.

6. 1, 2, 3, 4.

25. The major (most important) mechanism for hyperketonemia in DM is:

1. Increased ketogenesis in the liver.

2. Decreased utilization of ketons in the muscles.

3. Redistribution of ketons from the liver to the tissues.

4. Development of abnormal ketogenesis outside the liver.

5. Impossible degradation of ketons in the liver.

26. Ketogenesis in the liver in absolute insulin deficiency is activated because of:

1. Elevated levels of FFAs in the plasma and in the hepatocytes.

2. Activated gluconeogenesis.

3. Increase of the levels of the free carnitine in the hepatocytes.

4. Stimulated acylcarnitinetransferase.

5. 1, 3, 4.

6. 1,2,4.

27. Hyperosmolar non-ketogenic coma is a complication of:

1. Insulin-dependent DM type I.

2. Renal diabetes.

3. Symptomatic (secondary) diabetes.

4. Non-insulin-dependent DM type II.

5. Drug-induced DM.

28. Which is the most dangerous complication of DM type I:

1. Ketoacidotic coma.

2. Ischemic cerebral infarction (stroke).

3. Acute myocardial infarction.

4. Acute peripheral vascular occlusion.

5. Hyperosmolar coma.

6. Acute pulmonary edema.

29. Which complication in DM is a representation of carbohydrate “starving”:

1. Polyneuropathy.

2. Adynamia.

3. Cataract.

4. Retinopathy.

5. Itching.

30. Which complication is a direct consequence of hyperglycemic toxicity:

1. Microangiopathy.
2. Impotence
3. Cachexia.
4. Diabetic foot.
5. Hypercholesterolemia.