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THE BASIS OF BIOCHEMICAL CHANGES IN THE COURSE OF DIABETES MELLITUS

Abstract: *The article deals with enzymatic changes in diabetes mellitus (DM), which is characterised by absolute or relative insulin deficiency. DM is divided into insulin-dependent (IDDM) and insulin-independent (IND) types. All forms of diabetes are characterised by an increase in blood glucose concentration, which is caused by a decrease in the rate of glucose utilisation by tissues due to insulin deficiency or a decrease in its biological action. Insulin plays a key role in the regulation of carbohydrate and lipid metabolism, and its deficiency leads to metabolic disturbances that can cause coma, such as diabetic coma.*

Keywords: Diabetes mellitus, insulin, glucose, enzymatic changes, metabolism, diagnosis, prevention, coma.

Diabetes mellitus (DM) is a disease characterised by absolute or relative insulin deficiency. According to medical statistics, patients with DM account for about 5% of the population in many countries. The risk of diabetes doubles for every 20 per cent of overweight, and among the elderly (over 65) almost one in five people has diabetes. According to the World Health Organisation, diabetes is classified according to its pathogenesis and clinical course into two main forms: type I insulin-dependent diabetes (IDDM) and type II insulin-independent diabetes (IIID) [1,6]. All forms of DM are characterised by an increase in blood glucose concentration. Decreased glucose tolerance is also observed in cases of latent (latent) forms of DM. Hyperglycaemia is caused by a decrease in the rate of glucose utilisation by tissues due to insulin deficiency or a decrease in the biological action of insulin in target tissues. With insulin

deficiency, the amount of glucose transfer proteins (GLUT-4) on the membranes of insulin-dependent cells (adipose tissue and muscle) decreases. In hepatocytes, myocytes and lipocytes, low levels of insulin result in inhibition of the hexokinase (glucokinase) response, leading to decreased glucose-6-phosphate formation. Low concentrations of insulin, which controls the activity of glycolysis enzymes and almost all enzymes of the pentose phosphate pathway, and low amounts of glucose-6-phosphate are responsible for the inhibition of glycolysis and the pentose phosphate pathway [3,8].

The organism of DM patients satisfies its energy needs, first of all, at the expense of lipid oxidation. This causes a profound rearrangement of lipid metabolism. In a healthy organism, insulin promotes fat deposition in adipose tissue and decreases its utilisation [2,5]. The mechanisms of this are multiple and are not limited only to the preservation of lipids by increasing the catabolism of exogenous glucose: 1.Insulin increases the synthesis of fatty acids from glucose in hepatocytes and adipocytes. Under its influence it activates the reaction of acetyl-CoA carboxylation with subsequent formation of malonyl-CoA, the enzyme acetyl-CoA carboxylase (acetyl-CoA: CO2 ligase) is the target of the hormone; 2. Insulin counteracts the effects of all lipolytic hormones (adrenaline, glucagon, TSH, glucocorticoids) and promotes the creation of excess isocitrate and α-ketoglutarate - activators of acetyl-CoA carboxylase; 3.It is known that fatty acids are transported from the liver to adipose tissue as part of very low density lipoproteins (VLDL) secreted by the liver. Insulin increases the activity of lipoprotein lipase, which carries out the clearance of LDL with the transfer of fatty acids into adipocytes; 4.Insulin accelerates the transport of glucose into adipocytes and inhibits the main lipolytic enzyme of adipose tissue cells - hormone-dependent lipase; 5. Under the action of insulin, activation of glycolysis provides lipogenesis by the plastic pathway (via α-glycerophosphate), and activation of the pentose phosphate pathway supplies NADPH+ [4,9].

Disorders of carbohydrate, lipid and protein metabolism in DM can lead to the development of coma (acute complications). Diabetic coma is manifested by a sharp disorder of all body functions with loss of consciousness. The main causes of diabetic coma are acidosis and tissue dehydration. Coma states in DM can be characterised by three main forms: ketoacidotic, hyperosmolar and lactacidotic. Lack of treatment or inadequate treatment can cause: 1) diabetic ketoacidosis; 2) hyperosmolar coma; 3) hypoglycaemia [7,11].

The most important biochemical criteria of IDDM are:1. Glucose tolerance test: plasma blood glucose level above 10 mmol/l 2 hours after a sugar load indicates DM. 2. Determination of glycosylated haemoglobin: in diabetes its level increases 2-3 times (normal - 5.8-7.2% of total haemoglobin). 3.Absence or low levels of insulin and C-peptide in blood and urine: in normality, insulin and C-peptide are secreted in equimolar concentrations. Since approximately 2/3 of insulin is retained by the liver, the ratio of insulin/C-peptide in the portal vein and peripheral vessels is normally 1/3. The value of C-peptide levels in serum or urine allows a fairly accurate assessment of the functional status of pancreatic β-cells. 4. Albuminuria: in DM the daily excretion of albumin is about 30-300 mg - microalbuminuria (in norm - about 8 mg) [12].

Conclusions: Diabetes mellitus is the most common endocrinological disease in the world and its development is associated with a violation of carbohydrate metabolism in the body. The initial stages of this disease, at which it is still possible to suspend the pathological process, are almost asymptomatic - a person does not experience pain and discomfort, the only way to suspect it is to determine the level of blood glucose. Diabetes mellitus is dangerous with its complications. Patients with diabetes have an increased risk of heart attacks and strokes. By preventing diabetes mellitus type 2, you act for the benefit of the whole organism. Proper nutrition, moderate physical activity, weight control are basic concepts for the prevention of not only diabetes, but also hypertension,

stroke, heart attack. For early detection of diabetes mellitus, it is necessary to determine the sugar level at least once a year.

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