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Diabetes Mellitus: Etiology, Pathogenesis, Treatment

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ANNOTATION: Diabetes mellitus is a chronic disease characterized by relative or absolute insulin deficiency. At the same time, a form of diabetes mellitus is possible both genetically conditioned and without hereditary predisposition. Type 1 and type 2 diabetes are most common, which differ not only in initiating factors but also in the mechanism of development. The incidence of this pathology continues to increase. In recent years, the incidence of type 2 diabetes among children has increased. Regardless of the type of disease, carbohydrate, protein, lipid metabolism is impaired. Severe complications of diabetes mellitus are the development of ketoacidotic, hypoglycemic com, the pathogenesis of which is also different. The quality of life of patients with diabetes depends largely on the presence of late complications, which include microangiopathy and macroangiopathy. The principles of treatment include carrying out replacement hormone therapy (insulin therapy), administration of oral sugar-reducing drugs. Compliance with the principles of dietary therapy, optimal mode of physical activity allow to significantly improve the quality of life of patients. Doctors of all specialties should be wary of this disease.

KEYWORDS: diabetes mellitus, insulin, hyperglycemia, insulin resistance, hypoglycemic coma.

Diabetes mellitus is a chronic disease that develops when the pancreas does not produce enough insulin (in which case it is diabetes mellitus type 1) or the body uses the produced insulin inefficiently (accordingly, this option characterizes type 2 diabetes mellitus).

Insulin is known to be produced by the beta cells of the pancreas. Its most important function is participation in the metabolism of carbohydrates. With the help of this hormone, the pancreas regulates the level of glucose in the blood, since insulin delivers glucose to the cells, thereby helping to reduce blood sugar.

Distinguish between genetically determined and without hereditary predisposition forms of diabetes mellitus. In the classification of this disease, diabetes mellitus types 1 and 2, gestational diabetes, as well as other specific types of diabetes mellitus are distinguished [1]. Other specific types include, for example, juvenile MODY diabetes (this type is accompanied by genetic defects in β -cell function); genetic abnormalities, caused by dysfunction of insulin receptors; various endocrinopathies; diabetes on the background of chronic pancreatitis and others.

According to the modern hypothesis, the predisposition to diabetes is determined by two or more genes and is carried out with the participation of endogenous and exogenous factors. Installed also that genetically determined diabetes mellitus is heterogeneous. The pathogenesis of type 1 and type 2 diabetes is different. First of all, it should be noted that in type 1 diabetes there is an absolute insufficiency of insulin, and in type 2 diabetes - relative.

First, let's take a closer look at the pathogenetic mechanisms of type 1 diabetes. This disease is accompanied by destruction of pancreatic β -cells, which, in fact, and determines the absolute insufficiency of insulin in this type of diabetes. As a rule, clinical symptoms appear when the majority of β -cells are affected. Thus, thirst, polyuria begin to disturb the patient with the destruction of approximately 90% of β -cells. The onset of type 1 diabetes is more common in childhood.

Genetic predisposition is determined by certain types and combinations of genes of the HLA system [2]. To the genetic markers of type 1 diabetes include the genes of the HLA-DQA1 and DQB1 loci. In addition, in addition to the genes of the HLA system, the insulin gene is involved in the inheritance of predisposition to type 1 autoimmune diabetes mellitus; a gene encoding an IgG heavy chain; T-cell receptor β -chain gene; genes of tumor necrosis factors and other cytokines [3]. The initiating factors of type 1 diabetes include viruses (Coxsackie, cytomegaloviruses, measles, rubella, mumps), food components - bovine albumin, gliadin (barley protein), chemicals, for example,

streptozacin [4]. It is likely that the combination of initiating factors matters, with pancreatotropic viruses being the most significant.

Now consider the features of the pathogenesis of type 2 diabetes mellitus, the frequency of which is much higher than the first. Thus, genetic factors are among the predisposing factors. The type of inheritance is polygenic, the disease is inherited in an autosomal dominant manner. Among the many contributing factors type 2 diabetes mellitus, age and obesity are of the greatest importance (in 90% of patients). Most often, this disease affects the elderly. Of great importance is the abdominal form of obesity. It is important that, unlike subcutaneous fat adipocytes, visceral adipocytes are characterized by resistance to antilipolytic action of insulin and hypersensitivity to the lipolytic action of catecholamines [4]. Largely contributing to obesity a factor is hypodynamia, that is, a sedentary lifestyle. Thus, insulin resistance develops in type 2 diabetes. That is, the level of insulin in the blood of patients will be normal or even elevated (in contrast to the level of insulin in type 1 diabetes). At the same time, hyperinsulinemia undoubtedly has a compensatory character [5]. Insulin-dependent tissues for various reasons become insensitive to insulin. That is why insulin deficiency is relative, and not absolute, as in type 1 diabetes. However, in the late stage of type 2 diabetes, depletion of pancreatic β -cells occurs, which already leads to hypoinsulinism.

Microscopic examination of the pancreas in diabetes mellitus can reveal a decrease in the number and size of the islets of Langerhans. You can also detect a slight decrease in the mass of islet cells of Langerhans, the cells are dystrophically changed.

Insulin deficiency (relative - in type 2 diabetes or absolute - in type 1 diabetes) leads to a decrease in glucose supply to tissues, that is, to their energy starvation. In response, the secretion of contra-insular hormones increases - primarily glucagon, the breakdown of glycogen in the liver and muscles is activated. The result is hyperglycemia. When the glucose level exceeds the "renal threshold", the patient will experience glucosuria. Due to hyperglycemia, blood osmolality increases (glucose is an osmotically active substance). Clinically it is manifested by the fact that the patient, along with glucosuria, has polyuria, and, consequently, the accompanying symptoms - polydipsia, dry mouth. In this way, The leading value among metabolic disorders in diabetes mellitus is a violation of carbohydrate metabolism.

Protein metabolism also suffers in diabetes. Protein breakdown increases, a negative nitrogen balance is formed. Excessive protein catabolism interferes with normal the course of regenerative processes, which is associated with the fact of poor healing of tissues after their traumatization in patients with diabetes mellitus [3].

On the part of lipid metabolism, activation of lipolysis is noted. Subsequently, lipid peroxidation is activated, ketone bodies are formed. Activation lipolysis and a decrease in lipogenesis, lead to a decrease in the patient's body weight. Excess formation of ketone bodies is accompanied by the development of ketoacidosis. Further tissue hypoxia develops. All these stages of pathogenesis determine the clinic of ketoacidotic coma.

Ketoacidotic coma is formed against the background of severe insulin deficiency. This is possible in the case of late diagnosis of diabetes mellitus, with insufficient administration of insulin (for example, skipping the introduction of insulin), an increase in the need for insulin (against the background of intercurrent diseases, stress), as well as gross violations of the diet. Thus, in ketoacidotic coma, severe decompensation of carbohydrate, fat, protein, water-electrolyte metabolism and acid-base balance develops with the development of severe acidosis [6].

An equally serious complication of diabetes is hypoglycemic coma, which develops with a pronounced decrease in blood glucose levels. Leading provoking factors are overdose of insulin; violation of the diet (skipping meals); unplanned physical activity; various intercurrent diseases accompanied by vomiting, loose stools, anorexia; dysfunction of the liver and kidneys. With a decrease in glucose in the blood plasma, contrainsular hormones are released in an increased amount. In connection with a pronounced deficiency of glucose, energy supply in the neurons of the central nervous system suffers, which is accompanied by the development of hypoxia. Cell damage is noted against the background of hypoxia. Causes of cellular damage are primarily associated with intracellular accumulation of Na^+ and Ca^{2+} , which provoke intracellular edema, as well as with increased peroxidation processes. lipid oxidation damaging cell membranes [3].

Hyperlactacidemic coma is less common. The causes of its appearance are circulatory failure, massive bleeding, severe liver damage and kidneys, etc. The accumulation of lactate leads to lactic acidosis. Hyperlactacidemic

coma develops against the background of severe tissue hypoxia. Against the background of hypoxia, anaerobic glycolysis is activated, which, in fact, leads to the accumulation of lactic acid.

Due to insulin deficiency, hyperosmolar coma may also develop. This complication of diabetes is more common in older people with diabetes mellitus 2 type. Hyperosmolar coma is characterized by severe dehydration. The cause may be infectious diseases, injuries, burns, concomitant endocrine pathology. Against the background of the above reasons, the need for insulin increases. As a rule, the patient retains residual secretion of insulin, but it is not enough to eliminate the increasing hyperglycemia. Plasma hyperosmolarity leads to tissue dehydration. In the clinic, patients with hyperosmolar coma have neurological disorders associated primarily with dehydration of brain structures. Considering hyperglycemia, there is an increase in thirst, polyuria, polydipsia within a few days.

The quality of life of patients with diabetes mellitus depends largely on the presence of late complications, which include microangiopathy (more often in type 1 diabetes) and macroangiopathy (more often in type 2 diabetes).

Microangiopathy is characterized by changes in the vessels of the microvasculature, especially the retina and kidneys. The main etiological factor of these complications is long-term hyperglycemia. Glycation of plasma proteins and vascular walls plays an important role in pathogenesis. The sorbitol pathway for glucose utilization is activated. It is accompanied by the accumulation of sorbitol. Inside the cell, osmotic pressure rises, which leads to osmotic edema. Thus, metabolic disorders develop. In addition, against the background of microangiopathies, deposition of immune complexes in the basement membrane of the vessels is possible. Diabetic retinopathy is a common cause of blindness, diabetic nephropathy is accompanied by the development of chronic renal failure.

Diabetic neuropathy is accompanied by damage to the nervous system: motor, sensory, autonomic nerves. To metabolic disorders against the background of neuropathy in diabetes include: accumulation of sorbitol, glycosylation of structural proteins of the nerve fiber (the cause of demyelination), accumulation of free radicals (oppression of the antioxidant system). Diabetic neuropathy plays an important role in diabetic foot syndrome. At the same time, demyelination of nerve fibers is noted, which in turn leads to a violation of sensitivity by the type of "socks". Among the fundamental mechanisms for the development of diabetic foot syndrome, the most important are distal sensorimotor and autonomic neuropathy [4]. Perhaps the formation of trophic ulcers in the area of constant pressure on certain areas of the foot. Inflammatory autolysis of the underlying soft tissues develops. Clinical forms of diabetic foot are neuropathic, ischemic, neuroischemic (mixed). In the neuropathic form, there is sufficient safety arterial circulation of the lower extremities. The ischemic form is characterized by the presence of atherosclerosis of the arteries of the lower extremities.

The basis of macroangiopathies is atherosclerosis, especially damage to the vessels of the brain, heart, and extremities [7]. As already noted, macroangiopathies are more often detected in patients with type 2 diabetes mellitus. In the pathogenesis of the development of atherosclerosis and thrombosis, lipid metabolism disorders, dysfunction of the endothelium, proliferation of arterial smooth muscle cells and oxidative stress. Lipid metabolism disorders are manifested by hyperlipidemia. This increases LDL and VLDL and reduces HDL levels. Thrombus formation is facilitated by increased adhesion to the endothelium of platelets and activation of platelet aggregation. From activated platelets and macrophages secrete growth factors, which affects the proliferation of arterial smooth muscle cells. Atherosclerosis of the arteries causes ischemia and necrosis. One of the most formidable complications is myocardial infarction.

Microscopic examination of atherosclerosis reveals the processes of dystrophy, proliferation, thickening of the basement membranes of the endothelial lining in the capillaries of the skin, skeletal muscles, glomeruli of the kidneys, renal tubules, meninges of the kidneys, Bowman's capsule. Microscopic examination in myocardial infarction can detect the deposition of lipoglycan in the wall of arterioles and capillaries, followed by obliteration of the lumen of the vessels.

The main principle of treatment of type 1 diabetes mellitus is hormone replacement therapy - insulin therapy [8]. Currently used genetically engineered human insulins. Insulins are distinguished: basal - of medium duration and long-acting; prandial - fast acting. At type 2 diabetes mellitus insulin therapy is not always carried out. Indications may be insulin deficiency (a tendency to ketoacidosis), ineffectiveness of oral hypoglycemic drugs. It should be remembered about such complications of insulin therapy as hyperglycemia, lipodystrophy.

In type 2 diabetes, oral hypoglycemic drugs are used [9]. The choice of drug is determined by the need to influence the links of pathogenesis [10]. In some cases, drugs are shown that stimulate β -cells, and hence the secretion endogenous insulin. In other cases, drugs are used that increase the sensitivity of insulin receptors in muscle and adipose tissue. That is, this therapy will reduce insulin resistance.

It is mandatory to follow the principles of diet therapy. The calorie content of the daily diet and the composition of food ingredients in type 1 diabetes are physiological. However, refined carbohydrates are completely excluded. An assessment of digestible carbohydrates according to the system of bread units (XE) is necessary to adjust the dose of insulin before meals. In type 2 diabetes in overweight individuals, the calorie content of the diet is limited (approximately 1800 kcal per day), fractional meals are recommended (5-6 times a day in small portions). In order to prevent postprandial hyperglycemia, easily digestible pure carbohydrates, such as sugar, honey, jam, confectionery. It is recommended to eat foods rich in fiber, which slows down the absorption of carbohydrates. It is also a preventative postprandial hyperglycemia.

The next principle of the treatment of diabetes is the optimal mode of physical activity. In type 1 diabetes mellitus, the main requirement for a regimen of physical activity is dosage, that is, a clear distribution of physical activity during the time of day (hours of postprandial hyperglycemia). It should be remembered that the regime of physical activity is used only in the phase of diabetes compensation. In type 2 diabetes, physical activity increases energy expenditure, in general help reduce insulin resistance. However, physical activity can cause hypoglycemia, so blood glucose control is necessary. Also, One of the most important methods for the treatment of diabetes is diet therapy[11].

Currently, there is an upward trend in the incidence of diabetes mellitus. In recent years, the incidence of type 2 diabetes mellitus among children has increased. Type 2 diabetes is often diagnosed late, which increases the proportion of complications of the disease. Complications of diabetes mellitus include disorders of many organs and systems. Most often, these disorders are observed from the side of the nervous system, from the organs of vision and kidneys. Alertness in relation to this disease should be shown by doctors of all specialties. Early diagnosis, the choice of the right therapy, education in the "school of diabetes" are of paramount importance in ensuring the quality of life of patients.

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