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# MAIN RISK FACTORS FOR DIABETIC RETINOPATHY

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Retinal damage is one of the specific complications of diabetes; blindness in patients with diabetes occurs 25 times more often than in the general population [7, 9]. Among the factors that cause the progression of diabetic retinopathy are the degree of compensation of carbohydrate metabolism, duration of diabetes, age, arterial hypertension, nephropathy, pregnancy, smoking [6, 10,]. It is an undeniable fact that chronic hyperglycemia plays a major role in the development of Diabetic Retinopathy [1, 5, 6]. According to an international study - Diabetes Control and Complication Trial, maintaining satisfactory glycemic control in a group of people with no vascular complications helped reduce the risk of developing: diabetic retinopathy - by 76%, diabetic neuropathy - by 60%, microalbuminuria - by 30%, albuminuria - by 54%. Normoglycemia reduces the risk of diabetes complications by 12%, myocardial infarction by 16%, and microvascular complications by 25%. Blood pressure control reduces all complications by 24%, strokes by 44%, heart failure by 56%, microvascular complications by 37%, and mortality by 32% [2, 4, 5].

Age of patients Diabetes mellitus can be considered a risk factor. It is well known that Diabetic Retipopathy is extremely rare in childhood. However, with the onset of puberty, microvascular complications, including diabetic retinopathy, rapidly progress. This is due to the fact that during this period a powerful hormonal change occurs, accompanied by the production of a large number of counter-insular factors - pituitary tropic hormones, sex steroids, growth factors. The developing decompensation of diabetes mellitus can be explained by a rapid increase in body weight and, as a result, an increase in the need for insulin. The period of puberty is the most dangerous period from the point of view of progression of DR [3, 4, 5, 9, 10]. The involvement of hypertension as a major risk factor in the development and progression of DR has been proven [6, 8, 9].

The relationship between smoking and progression of retinopathy is unclear. Smoking probably causes the development of hypoxia and cerebral vasospasm, which affects the progression of DR. The WESDR study (2008) found a relationship between different HLA antigens and DR, and established a relationship with the level of glycohemoglobin, hypertension, duration of diabetes, and increasing proteinuria. A 5.4 times more frequent development of proliferative retinopathy was noted compared to the group where HLA haplotypes DR3 and DR4 were not detected. At the same time, observation of a cohort of patients with HLA DR4 did not reveal progression of DR compared to the group without these antigens. This process is likely to be more controlled by other risk factors. The biochemical processes leading to morphological changes in the retina have been fairly well studied [1, 2, 5]. The trigger point is chronic hyperglycemia, which leads to activation of aldose reductase activity, increased non-enzymatic glycation of proteins, changes in the myo-inositol phosphatidimenositol mechanism, increased activity of protein kinase C, decreased heparin sulfate proteoglycan, increased autoxidation of glucose, changes in the activity and levels of vasoactive substances such as endothelin, prostanoids, nitric oxide, histamine, etc. [6, 5, 9]. Due to acidosis and metabolic disorders at the cellular level, the electrical properties of red blood cell membranes change, which is the main reason for the increase in blood viscosity in diabetic retinopathy [6]. One of the mechanisms of the pathological process may be thickening of the basement membrane of the capillaries, which in turn, hypothetically leads to the closure of the retinal capillaries. However, this hypothesis was not confirmed, since the use of aldose

reductase inhibitors in patients with type 1 diabetes did not prevent the progression of DR. The relationship between non-enzymatic and enzymatic protein glycation and the progression of diabetic retinopathy has been proven [1, 2, 7]. According to the authors [1, 4, 5, 8], diabetic retinopathy develops as a consequence of retinal ischemia. Vascular damage, consisting of thickening of the basement membrane, loss of pericytes, focal proliferation of endothelial cells, obliteration of capillaries, the appearance of microvascular shunts - this entire complex of lesions is diagnosed as diabetic microangiopathy. In addition to damage to the vessel wall, there is a change in blood viscosity and the properties of blood cells. Hyperglycemia, which is a consequence of a lack of insulin, causes an increase in the release of growth hormone [1,2]. Increased levels of growth hormone under conditions of hypoinsulinemia alter protein synthesis by hepatocytes, which leads to dysproteinemia; increased levels of fibrinogen and α2-globulin enhance erythrocyte aggregation, hyperglycemia impairs the production of prostocyclin by endothelial cells; increased aggregation of erythrocytes and platelets causes hemodynamic disturbances in the microcirculation system; deterioration of blood flow in the microcirculatory system leads to hypoxia and ischemia of the retina; Hypoxia and ischemia of the retina cause excessive production of vasoproliferative growth factor, which stimulates the growth of new vessels around the optic disc and in other areas of the retina. Vasoproliferative factors are peptides with pronounced mitogenic properties. Similar properties are attributed to fibroblast growth factors – FGF, vascular endothelial factor – VEGF, insulin-like growth factor – IGF and others [3, 5].

Another factor that can cause the progression of DR is too rapid normalization of carbohydrate metabolism parameters after the start of intensive insulin therapy for poorly compensated diabetes in young patients. The same negative effect was noted in patients with type 2 diabetes when switching from tableted hypoglycemic drugs to insulin therapy [5,6,7,9]. According to a number of studies, sharp fluctuations in glycemia during the day, equally with long-term hyperglycemia, are the main causes of the development of retinopathy[5,6,8].

The type of diabetes therapy, according to some authors, also plays a role in the appearance and progression of DR. Based on long-term observations of diabetic patients, it has been established that the risk of developing retinopathy is highest in patients receiving insulin, which is associated with a more severe course of diabetes in this category of patients. The likelihood of developing a proliferative form of DR is 2-5 times higher in them compared to patients treated with diet or oral hypoglycemic agents[9, 10,11].

High blood pressure is a strong independent risk factor for the development of DR, especially in patients with type 2 diabetes. The most significant studies that examined the effect of arterial hypertension on the development of diabetic retinopathy were the EUCLID (EURODIAB Controlled Trial of Lisinopril in Insulin-Dependent Diabetes Mellitus) and UKPDS (United Kingdom Prospective Diabetes Study) programs. The results demonstrated a significant reduction in the risk of blindness and a reduction in the rate of progression of DR in patients in the intensive BP control group[35,75].

Patients with diabetic nephropathy have a high probability of having microvascular changes in the fundus (96%) [10,11]. In type 2 diabetes, microalbuminuria is an independent predictor of the development of retinopathy, as well as an indirect marker of the condition of the fundus vessels [43]. The results of the EURODIAB program showed that the correlation between an increase in blood pressure and the level of albumin excretion is determined only in patients with

retinopathy, regardless of metabolic control and duration diabetes This fact suggested that DR in arterial hypertension is an important independent risk factor for the progression of nephropathy.

The assumption of an inherited predisposition to various microvascular complications of diabetes is based on the fact that some patients do not develop retinopathy despite poor glycemic control. The survey results showed that the severity of retinopathy in parents correlates with the severity of the complication in children; this connection was especially strong between mother and child.

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