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CAUSES OF DIABETIC RETINOPATHY: INFLAMMATORY MARKERS AND GENES

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Abstract: Diabetic retinopathy is one of the most dangerous and common complications of diabetes in the eye, and it is recognized as the leading cause of blindness worldwide. Most patients with diabetes develop this complication over time, and while previously high glucose levels, vascular changes, hypoxia, or oxidative stress were considered the main factors, today scientific studies have confirmed that genetic factors, along with inflammatory markers, play a leading role in the pathogenesis of this disease.

Keywords: diabetic retinopathy, inflammatory markers, genes, genetic polymorphism, interleukin, angiogenesis, TNF-alpha, VEGF, ICAM-1, disease pathogenesis.

The mechanism of development of diabetic retinopathy is the result of pathological changes in the micro- and macrovascular structures of the retina due to impaired sugar metabolism. Violation of the hemato-retinal barrier, weakening of microtomirs, expansion of capillaries, accumulation of cholesterol and other lipids, oxidative stress and hypoxia-all these processes serve the development of diabetic retinopathy. However, in recent years, the focus of the scientific community has increasingly shifted to inflammatory reactions and genetic polymorphisms. Inflammatory markers play an important role in the development of diabetic retinopathy. Many studies have shown that inflammatory cytokines, specifically interleukins and tumor necrosis factors (TNF-alpha), are elevated in diabetic retinopathy. Molecules such as interleukin-1beta, interleukin-6, interleukin-8, and MCP-1 are activated in the retinal networks as a result of diabetes and cause hyperpermeability of blood vessels. These processes result in increased retinal vascular permeability, accumulation of exudates, microaneurysms, microaneurysms, neovascularization, and ultimately, the risk of severe central vision loss.

The duration of moderate inflammation, which is one of the main pathological factors in the development of diabetic retinopathy, leads to the activation of enzymes in the walls of blood vessels. Specifically, the enzyme MMP (matrix metalloproteinases) is activated and initiates extratcellular matrix degradation. This triggers morphological and functional changes in the tissues of the cornea. Continuous exposure to inflammatory markers activates angiogenesis, resulting in the formation of new, weak, and porous blood vessels on the surface of the retina. Genetic factors, particularly certain gene polymorphisms and their influence on the development of diabetic retinopathy, have become a very relevant topic today. Some studies have shown that the activity and polymorphism of the VEGF (vascular endothelial growth factor) gene are one of the most important elements in the development of retinopathy. Increased VEGF levels stimulate the formation of new blood vessels, which can lead to damage, swelling, and bleeding in the retina. Another factor is the aldolase reductase gene (AKR1B1) and its genetic variants, which may also contribute to the pathogenesis of diabetic retinopathy. This gene is responsible for the process of converting glucose to sorbitol, and an increase in sorbitol levels damages endothelial cells, resulting in increased latent inflammation in the microcirculation of the retina [1].

Scientific experiments have confirmed that the expression of molecules such as IQGAP1, ICAM-1, E-selectin, and TGF-beta is significantly increased in diabetic conditions. ICAM-1 (intracellular adhesion molecule-1) causes inflammation in the inner lining of blood vessels,

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leukocyte adhesion to the wall, local hypoxia, and microinfarctions. This creates the conditions for further deterioration of the retina. High levels of tumor necrosis factor cause the exacerbation of proinflammatory processes, increased damage to the mucous membrane, apoptosis, and oxidative stress. The higher the level of these markers, the more severe the retinopathy in diabetic patients. The danger of inflammation is that it can negatively affect the general condition of large and small blood vessels not only in the retina, but throughout the body. The genetic background plays a crucial role in the development of micro- and macrovascular changes in the body, depending on age, gender, disease duration, and other external factors. Therefore, people with diabetic retinopathy may have the same level of diabetes, but the severity of retinopathy may vary. For example, even in the early stages of diabetes, susceptibility to diabetic retinopathy is reflected through genetic polymorphisms. In such situations, the risk of the disease can be predicted in advance using genetic markers and molecular diagnostic methods. Genetic polymorphisms related to the immune system, particularly variants associated with the HLA system, are being extensively studied in current research for their potential impact on the development of diabetic retinopathy. A correlation was found between severe forms of retinopathy and the HLA-Diabetic retinopathy3, HLA-Diabetic retinopathy4 gene variants. Polymorphism of cytokine and adhesion molecule genes is considered an important genetic basis that determines the individual course, severity, and outcome of the disease [2].

Polymorphism of metalloproteinases, chemokines, and growth factor genes has an important role in the pathogenesis and course of diabetic retinopathy, while previously identified using experiments on more animals, is still finding evidence in human populations today. Genetic variants of endothelin-1, angiopoietin, matrix metalloproteinases, and other growth factors also increase the risk of diabetic retinopathy. Elevated levels of inflammatory markers in serum and, in rare cases, in ocular fluid have been reported in numerous laboratory and clinical studies. Changes in these markers at each stage of retinopathy are of great importance in predicting the prognosis of the disease, as well as in monitoring the results of modern anti-VEGF and immunomodulatory therapy. Inflammatory markers are being widely used not only as a diagnostic and prognostic tool, but also to monitor the course of the disease and to assess the response to new drugs. Changes in the retinal vascular wall, increased cell adhesion, apoptosis, and fibrosis processes are involved in the development of retinopathy with the participation of various molecules. Many growth factors and cytokines, such as TGF-beta and PDGF, accelerate the process of angiogenesis and fibrosis. This leads to the growth of new, fragile, easily damaged blood vessels in the eye tissue [3].

Continuous monitoring of inflammation and genetic markers plays an important role in developing treatment algorithms for early stages of diabetic retinopathy. Based on initial clinical and biochemical signs, it is possible to prevent or slow the progression of retinopathy by taking into account age, gender, disease duration, and the patient's individual genotype. Treatment of patients with new generation drugs is important in cases where laboratory and molecular studies have shown the presence of inflammation and gene polymorphisms in diabetic retinopathy. Today, research is underway in many countries to suppress the progression of diabetic retinopathy using new biological drugs, anti-VEGF drugs, and ursodeoxycholic acid-based toxic modulating methods. Elevated levels of inflammatory markers such as interleukins, TNF-alpha, ICAM-1, and similar molecules directly accelerate the progression of diabetic retinopathy. Clinical observations show that high levels of these markers lead to an increase in clinical symptoms and rapid loss of vision in patients. Genetic polymorphisms determine individual risk of disease and severity of Diabetic Retinopathy. For example, patients with certain variants of the VEGF or ICAM-1 genes are more likely to develop proliferative forms of retinopathy, the

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most dangerous and difficult to treat. This requires the widespread introduction of individualized approaches and genetic screening methods in modern ophthalmology [4].

Controlling inflammation and preventing the severe consequences of the disease by identifying genetic predispositions remain key areas of advancement in the prevention and treatment of diabetic retinopathy. By implementing effective measures against inflammation and individual genetic factors in the early stages of disease development, it is possible to reduce the number of cases of blindness caused by diabetic retinopathy [5].

Conclusion:

Inflammatory markers and genetic factors play a key role in the development of diabetic retinopathy. The pathogenesis of the disease is polyetiological, in which chronic hyperglycemia, oxidative stress, and angiogenesis are important, along with the activation of inflammatory reactions and certain gene polymorphisms. Persistent increases in inflammatory markers such as interleukins, TNF-alpha, and ICAM-1 lead to vascular damage and the formation of new blood vessels. Genetic polymorphisms allow us to determine the course and severity of the disease at the individual level. In the prevention and treatment of diabetic retinopathy, it is possible to study the inflammatory and genetic bases, constantly monitoring them, prevent the consequences of the disease by developing new drugs, and dramatically reduce the incidence of blindness statistics.

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