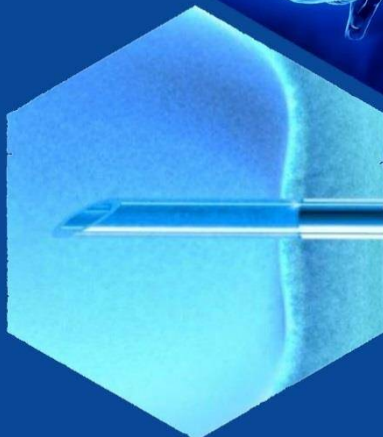
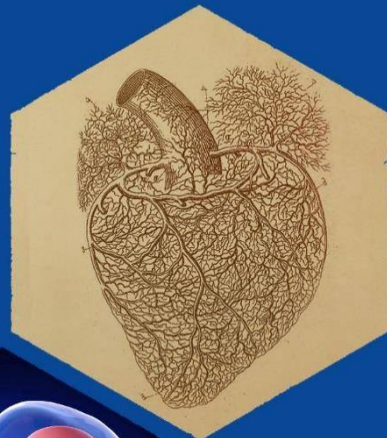


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METHYLENE TETRAREDUCTASE POLYMORPHISM AS A RISK FACTOR FOR RETINAL VESSEL OCCLUSION (A Literature Review)



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Abstract. Relevance. Disorders of one-carbon metabolism, mediated by MTHFR activity and leading to hyperhomocysteinemia and epigenetic dysregulation, are an understudied but potentially key link in the pathogenesis of occlusive retinal vascular lesions, particularly in patients without traditional cardiovascular risk factors. **Aim of the study.** To investigate risk factors for retinal vascular occlusion. **Results.** In cases of confirmed hyperhomocysteinemia, the primary objective is to identify and address possible causes. These include deficiencies in folate, vitamins B12 and B6, chronic kidney disease, certain medications and dietary factors. **Conclusions.** Disturbances in one-carbon metabolism, mediated by MTHFR activity, lead to reduced production of S-adenosylmethionine and the development of global hypomethylation, which is accompanied by dysregulation of vascular endothelial gene expression (eNOS, VEGF, ICAM-1) and the development of endothelial dysfunction, which plays a central role in the pathogenesis of occlusive retinal vascular lesions.

Keywords: gene polymorphism, retinal vascular occlusions, diagnosis, homocysteine.

METILEN TETRADUKTAZA POLIMORFIZMI RETINA TOMIRLARI OBKLYUZIYASI UCHUN XAVF OMILI SIFATIDA (Adabiyotlar sharhi)

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Annotatsiya. Dolzarbligi. MTHFR faolligi bilan bogʻliq boʻlgan va gipergomotsisteinemiya hamda epigenetik disregulyatsiyaga olib keladigan bir uglerodli almashinuvning buzilishi, ayniqsa anʼanaviy yurak-qon tomir xavf omillari boʻlmagan bemorlarda, toʻr parda qon tomirlarining okklyuziv shikastlanishlari patogenezida kam oʻrganilgan, ammo potensial asosiy boʻgʻin hisoblanadi. Tadqiqotning maqsadi. Toʻr parda qon tomirlari okklyuziyasining xavf omillarini oʻrganish. Natijalar. Tasdiqlangan gipergomotsisteinemiya asosiy vazifa ehtimoliy sabablarni aniqlash va bartaraf etishdan iborat. Bularga folat kislotasi, B12 va B6 vitaminlari yetishmovchiligi, surunkali buyrak kasalliklari, ayrim dori vositalari va ovqatlanish omillari kiradi.

Xulosalar. MTHFR faolligi bilan bogʻliq boʻlgan bir uglerodli metabolizmning buzilishi S-adenozilmetionin ishlab chiqarilishining pasayishiga va global gipometillanishning rivojlanishiga olib keladi, bu qon tomir endoteliysi genlari (eNOS, VEGF, ICAM-1) ekspressiyasining buzilishi va endoteliy disfunksiyasining rivojlanishi bilan birga keladi, bu toʻr parda qon tomirlarining okklyuziv shikastlanishlari patogenezida markaziy rol oʻynaydi.

Kalit soʻzlar: genlar polimorfizmi, toʻr parda qon tomirlari okklyuziyasi, tashxis, gomotsistein.

ПОЛИМОРФИЗМ МЕТИЛЕНОВОЙ ТЕТРАРЕДУКТАЗЫ КАК ФАКТОР РИСКА ОККЛЮЗИИ СЕТИЧНЫХ СОСУДОВ (Обзор литературы)

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Аннотация. Актуальность. Нарушения одноуглеродного обмена, опосредованные активностью МТГФР и приводящие к гипергомоцистеинемии и эпигенетической дисрегуляции, являются малоизученным, но потенциально ключевым звеном в патогенезе окклюзионных поражений сосудов сетчатки, особенно у пациентов без традиционных сердечно-сосудистых факторов риска. Цель исследования. Исследовать факторы риска окклюзии сосудов сетчатки. Результаты. При подтвержденной гипергомоцистеинемии основной задачей является выявление и устранение возможных причин. К ним относятся дефицит фолиевой кислоты, витаминов В12 и В6, хронические заболевания почек, некоторые лекарственные препараты и факторы рациона питания. Нарушения одноуглеродного обмена, опосредованные активностью МТНFR, приводят к снижению выработки S-аденозилметионина и развитию глобальной гипометилизации, которая сопровождается дисрегуляцией экспрессии генов эндотелия сосудов (eNOS, VEGF, ICAM-1) и развитием дисфункции эндотелия, что играет центральную роль в патогенезе окклюзионных поражений сосудов сетчатки.

Ключевые слова: полиморфизм генов, окклюзии сосудов сетчатки, диагностика, гомоцистеин.

Background. Over the past two decades, our understanding of the pathogenesis of vascular diseases has evolved significantly: whilst previously the focus was on classic risk factors such as hypertension, diabetes mellitus and hyperlipidaemia, it is now becoming clear that these mechanisms alone are insufficient to explain vascular events in a significant proportion of patients, particularly those of young and middle age. In ophthalmology, this is evident in cases of retinal vascular occlusions and chronic central serous chorioretinopathy, which frequently develop in the absence of marked cardiovascular pathology, suggesting the existence of additional, less well-understood links in the pathogenesis. In this context, disorders of one-carbon metabolism—which underpins key cellular processes, ranging from DNA methylation and nucleotide synthesis to the regulation of gene expression and the maintenance of vascular homeostasis—are of particular interest [1, 4].

The enzyme methylenetetrahydrofolate reductase (MTHFR) plays a central role in this metabolic pathway; its activity determines the efficiency of homocysteine remethylation and the formation of the universal donor of methyl groups—S-adenosylmethionine. Polymorphisms in the MTHFR gene, as well as deficiencies in vitamins B2, B12 and folates, can disrupt these processes, leading to hyperhomocysteinemia, endothelial dysfunction, oxidative stress and chronic inflammation — conditions directly involved in the development of ischaemic and thrombotic lesions in the eye's microcirculatory bed. Current evidence suggests that MTHFR is not merely a component of the folate cycle, but a key regulator of epigenetic stability, influencing, via methylation, the expression of genes that control vascular tone, angiogenesis and the permeability of the blood-retinal barrier [2, 5].

Despite the accumulated evidence, the role of one-carbon metabolism disorders in the aetiology of occlusive retinal lesions remains insufficiently systematised, and clinical approaches to the diagnosis and management of these disorders in ophthalmological practice are often limited [3, 6]. This study aims to conduct an in-depth investigation of the risk factors associated with dysfunction of MTHFR-dependent pathways, which is of direct relevance to the development of personalised strategies for the prevention and early detection of patients at high risk of vascular complications affecting the eye.

Aim of the study. To investigate the risk factors for retinal vascular occlusion.

Results. Patients with retinal vein occlusion, central retinal artery occlusion and even chronic central serous chorioretinopathy (CSCR) often do not have any significant cardiovascular disease. This fact points to the existence of additional pathogenetic mechanisms, among which disorders of one-carbon metabolism—which underpins methylation processes, DNA synthesis, gene expression regulation and the maintenance of vascular homeostasis—are of particular interest.

The key enzyme in this metabolic pathway is methylenetetrahydrofolate reductase (MTHFR), the activity of which determines the efficiency of homocysteine remethylation and the formation of the universal donor of methyl groups — S-adenosylmethionine (SAM). Polymorphisms in the MTHFR gene can disrupt these

processes, creating the conditions for endothelial dysfunction, oxidative stress, vascular inflammation and thrombus formation.

One-carbon metabolism is an interconnected system of biochemical reactions comprising the folate cycle, the methionine cycle and the transsulphuration pathway. Despite the relative simplicity of the individual reactions, it is this system that ensures the functioning of virtually all the body's cells.

The main functions of one-carbon metabolism include:

the synthesis of purines and pyrimidines; DNA replication and repair; the regulation of gene expression; the formation of cell membrane phospholipids; the synthesis of neurotransmitters; the formation of creatine; the synthesis of melatonin; regulation of antioxidant defence; and maintenance of vascular endothelial function.

Thus, a disruption at any stage of this cycle has a systemic effect on virtually all organs; however, tissues with a high metabolic rate, including the brain, the retina and the vascular endothelium, are the most sensitive.

The folate cycle. Folic acid obtained from food, following a series of conversions, forms tetrahydrofolate (THF), which acts as a carrier of one-carbon units. Subsequently, under the action of serine hydroxymethyltransferase, 5,10-methylenetetrahydrofolate is formed — the direct substrate for the MTHFR enzyme. It is at this stage that the key reaction of the entire cycle takes place.

The MTHFR enzyme catalyses the reduction of 5,10-methylenetetrahydrofolate to 5-methyltetrahydrofolate — the only form of folate capable of donating a methyl group for the remethylation of homocysteine to methionine. This reaction requires the presence of flavin adenine dinucleotide (FAD), which is formed from vitamin B2 (riboflavin). Therefore, even in the absence of genetic disorders, a riboflavin deficiency can reduce MTHFR activity.

The methionine cycle. The resulting 5-methyltetrahydrofolate donates a methyl group to homocysteine.

This reaction is catalysed by the enzyme methionine synthase (MTR), for which methylcobalamin (vitamin B12) acts as a cofactor.

This results in the formation of methionine.

Methionine then reacts with ATP to form S-adenosylmethionine (SAM) — the body's primary donor of methyl groups.

SAM is involved in more than 200 methylation reactions, including:

DNA methylation; RNA methylation; protein methylation; myelin synthesis; phosphatidylcholine synthesis.

MTHFR as a regulator of epigenetic processes. In recent years, our understanding of the biological role of the MTHFR enzyme has expanded significantly. Whilst it was previously regarded primarily as a component of folate metabolism, MTHFR is now recognised as one of the key regulators of cellular epigenetic stability, including in the tissues of the eye.

This is because MTHFR activity determines the formation of S-adenosylmethionine (SAM) — a universal donor of methyl groups. It is SAM that drives the majority of methylation reactions underpinning the regulation of gene

expression, including genes involved in the functioning of the retina, the choroid, the optic nerve and the endothelium of the ocular vessels.

Methylation involves the attachment of a methyl group ($-\text{CH}_3$) to DNA, RNA, protein and lipid molecules. This process does not alter the nucleotide sequence, but it can significantly influence gene activity, determining whether genes are expressed or repressed.

Thus, MTHFR influences not only homocysteine metabolism but also the condition of ocular structures via epigenetic regulation, which is particularly important for tissues with high metabolic activity, such as the retina and the pigment epithelium.

The role of S-adenosylmethionine. S-adenosylmethionine is the primary metabolic donor of methyl groups. It is involved in more than two hundred biochemical reactions, including: DNA and histone methylation; microRNA regulation; the synthesis of cell membrane phospholipids; melatonin production; creatine synthesis; polyamine synthesis; myelin formation; and the regulation of the activity of numerous enzymes.

Of particular importance in ophthalmology are the processes associated with maintaining the structure of photoreceptor cell membranes, the functioning of the retinal pigment epithelium, the stability of the blood-retinal barrier, and the regulation of angiogenesis in the choroid and retina. A reduction in MTHFR activity leads to decreased SAM production and, simultaneously, an increase in the concentration of S-adenosylhomocysteine (SAH), which is a potent inhibitor of most methyltransferases.

This results in a state of global hypomethylation. It is precisely this process that is currently regarded as one of the key mechanisms of vascular and neurosensory ageing, including in the tissues of the eye.

Dysregulation of vascular gene expression. Epigenetic changes are accompanied by disrupted expression of a large number of genes regulating vascular wall function and microcirculation in the structures of the eye. These include genes encoding: endothelial NO synthase (eNOS); vascular endothelial growth factor (VEGF); endothelin-1; superoxide dismutase; catalase; glutathione peroxidase; interleukin-6; tumour necrosis factor α ; and the cell adhesion molecules ICAM-1 and VCAM-1.

In the context of ocular pathology, this leads to impaired regulation of choroidal blood flow, increased vascular permeability, activation of inflammatory responses and enhanced neovascularisation. Consequently, the balance shifts towards vasoconstriction, inflammation and a prothrombotic state, which is significant in diabetic retinopathy, retinal vein occlusions, ischaemic optic nerve lesions and a number of chorioretinal diseases.

Endothelial dysfunction is a central component of vascular pathology. The modern concept of vascular disease regards endothelial dysfunction as an early stage in most vascular disorders. The endothelium is not only the inner lining of blood vessels but also a highly active endocrine organ that regulates: vascular tone;

microcirculation; blood clotting processes; inflammatory reactions; leukocyte adhesion; vascular wall permeability; and angiogenesis.

Endothelial dysfunction underlies disturbances in blood flow within the retinal and choroidal vessels, and also contributes to the development of macular oedema, retinal ischaemia and abnormal vascular permeability.

Hyperhomocysteinemia causes direct damage to endothelial cells. **Homocysteine** induces the formation of reactive oxygen species, impairs mitochondrial function and reduces the bioavailability of nitric oxide. This results in a reduction in the vessels' capacity for physiological vasodilation, including within the eye's microcirculatory bed.

In conditions of hyperhomocysteinemia, the following occur simultaneously: endothelial damage; increased blood viscosity; platelet activation; increased expression of tissue factor; reduced fibrinolysis; and enhanced inflammatory infiltration of the vascular wall. It is precisely the combination of these processes that creates the conditions for the development of retinal venous occlusion, diabetic microangiopathy, ischaemic lesions of the arterial bed and impaired perfusion of the optic nerve. Furthermore, impaired endothelial function in the choroidal vessels may alter the permeability of the choroidal capillaries and the hydrostatic pressure within the choroid. This mechanism is of particular interest when discussing the possible role of folate cycle disorders in the pathogenesis of central serous chorioretinopathy, age-related macular degeneration and other chorioretinal diseases.

Neuroinflammation. Recent studies show that retinal ischaemia is accompanied by marked activation of microglia. Activated microglial cells begin to synthesise pro-inflammatory cytokines: interleukin-1 β ; interleukin-6; tumour necrosis factor- α ; MCP-1. In the presence of hyperhomocysteinemia, these processes are further exacerbated. Consequently, the inflammatory response becomes not only a consequence of ischaemia, but also an independent factor in the progression of damage to neurosensory tissue.

Discussion. Current evidence suggests that disturbances in one-carbon metabolism may be considered one of the potential components of the complex pathogenesis of central retinal artery occlusion. It is not the MTHFR polymorphism itself that is of greatest clinical significance, but rather its impact on the functional state of the vascular endothelium, homocysteine levels, oxidative stress and epigenetic regulation. Further research in the field of personalised ophthalmology may determine the role of these biomarkers in risk stratification and the development of preventive strategies for patients with ischaemic retinal lesions.

Where hyperhomocysteinemia is confirmed, the primary objective is to identify and address potential causes. These include deficiencies in folate, vitamins B12 and B6, chronic kidney disease, certain medications and dietary factors. Any identified deficiencies should be corrected in accordance with current clinical guidelines. When prescribing folate and B vitamins, it is important to take the patient's baseline status into account and avoid the unjustified use of high doses without laboratory confirmation.

Conclusions.

1. Disruptions in one-carbon metabolism, mediated by MTHFR activity, lead to reduced production of S-adenosylmethionine and the development of global hypomethylation, which is accompanied by dysregulation of vascular endothelial gene expression (eNOS, VEGF, ICAM-1) and the development of endothelial dysfunction, which plays a central role in the pathogenesis of occlusive lesions of the retinal vessels.

2. Hyperhomocysteinemia associated with MTHFR gene polymorphisms induces oxidative stress, endothelial damage, platelet activation and pro-inflammatory cytokines; when combined with impaired microcirculation, this creates the conditions for thrombus formation and ischaemia in the ocular vasculature, particularly in patients without traditional risk factors.

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8 ЖИЛД, 1 СОН

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