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Eye microcirculation in glaucoma. Part 2. Disorders of regional hemodynamics

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ABSTRACT

Glaucoma is one of the leading causes of blindness worldwide. The etiology of primary glaucoma is usually divided into mechanical and vascular mechanisms. Research of the vascular component of glaucoma was going on since the beginning of the last century with continuous improvement of diagnostic methods from invasive to high-tech non-contact ones. Modern and promising methods are: ultrasound examination in color Doppler mapping and pulsed Doppler modes, optical coherence tomography angiography, and laser speckle flowgraphy. The review describes specific for glaucoma blood flow changes in ocular vessels, correlating with functional and structural changes: decrease of vascular density in macular, parafoveolar, and peripapillary areas, decrease of the integral indicator of microcirculation, decrease of the indicators of volume and linear blood flow velocities in retinal and choroidal vessels, impaired retrobulbar blood circulation. The analysis of literature data is presented concerning the investigation of hemodynamic disturbances in ocular vessels in normotensive glaucoma and glaucoma in myopic eyes, in systemic blood flow disturbances (arterial hypertension and hypotension) in patients with glaucomatous optic neuropathy.

Keywords: glaucoma; hemodynamics; blood flow; macula; choroid; myopia; normotensive glaucoma; arterial hypertension; hypotension.

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Микроциркуляция глаза при глаукоме. Часть 2. Нарушения регионарной гемодинамики

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АННОТАЦИЯ

Глаукома — одна из основных причин слепоты в мире. В этиологии первичной глаукомы выделяют механический и сосудистый механизмы. Исследования сосудистого компонента при глаукоме ведутся с начала прошлого века с совершенствованием способов диагностики от инвазивных до высокотехнологичных бесконтактных. Современными и перспективными методами являются: цветовое допплеровское картирование, оптическая когерентная томография с функцией ангиографии и лазерная спекл-флоуграфия. В обзоре описаны характерные для глаукомы нарушения кровообращения в сосудах глаза, коррелирующие с функциональными и структурными изменениями: снижение сосудистой плотности макулярной, парафовеолярной и перипапиллярной зон, снижение интегрального показателя микроциркуляции, снижение показателей объёмной и линейной скорости кровотока в сосудах сетчатки и хориоидеи, изменения ретробульбарной гемоциркуляции. Представлен анализ данных литературы по изучению нарушений гемодинамики в сосудах глаза при нормотензивной глаукоме и глаукоме в миопических глазах, при системных нарушениях кровообращения (артериальной гипертензии и гипотензии) у пациентов с глаукомной оптической нейропатией.

Ключевые слова: глаукома; гемодинамика; кровоток; макула; хориоидея; миопия; нормотензивная глаукома; артериальная гипертензия; гипотензия.

Как цитировать

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INTRODUCTION

Glaucomatous optic neuropathy is a challenge to modern ophthalmology, as the incidence and visual disability caused by this socially significant disease continue to grow worldwide, despite significant advances in its treatment [1, 2]. The number of patients is predicted to rise to 111.8 million by 2040 [3]. In the Russian Federation, 1,250,558 glaucoma cases were reported in 2022. Glaucoma is defined as a group of multifactorial diseases with similar clinical, morphological, and functional manifestations. Mechanical and vascular factors are considered the main causes of glaucoma. The mechanical theory of glaucoma considers axonal compression caused by increased intraocular pressure (IOP), whereas the vascular theory highlights the role of ocular blood flow deficiency and decreased perfusion pressure [4]. The previous review discussed the structure and examination methods of the ocular and retrobulbar blood flow, including color flow Doppler (CFD) and pulsed wave Doppler (PWD), optical coherence tomography–angiography (OCTA), and laser speckle flowgraphy (LSFG). This review is focused on the signs of ocular blood flow disorders observed in primary open-angle glaucoma (POAG), including in patients with myopia, and in normal-tension glaucoma (NTG); the systemic circulation effect on the course of glaucomatous optic neuropathy was also described.

MACULAR AREA

Using OCTA, Chen et al. [5] revealed decreased macular vessel density (VD) in the superficial layer in POAG compared with the normal age range. In 2021, Cano et al. confirmed a decrease in macular VD in POAG and associated it with reduced total retinal blood flow [6]. Yarmohammadi et al. [7] reported parafoveal VD in POAG of 51.1% compared with 54.5% in healthy eyes, which correlated with retinal nerve fiber layer (RNFL) thickness. Xu et al. [8] also described a positive correlation between a decrease in density of deep macular vessels and structural retinal damage in POAG. They reported that macular VD and blood flow in moderate and advanced POAG were significantly lower than at the early stage. A group of scientists led by A. Tao revealed a relationship between a decrease in macular VD and glaucoma perimetry data [9]. Li et al. interpreted the initial expansion of the avascular foveal area as a predictor of a high risk of glaucomatous structural defects [10]. Zhang et al. [11] reported that a sharp increase in IOP caused by an acute angle-closure glaucoma was accompanied by an expansion of the avascular foveal area and reduction in macular VD.

PERIPAPILLARY AREA

Yarmohammadi et al. [7] reported that normal peripapillary VD was 62.7% compared with 61.4% in preperimetric glaucoma and 58% in POAG, which correlated with the visual field indices and RNFL thickness. Triolo et al. [12] also reported a positive correlation between the peripapillary VD and RNFL thickness. Son et al. noted that location of the peripapillary hypovascular area correlated with the RNFL defect [13]. Shin et al. [14] demonstrated that the rate of peripapillary VD decrease correlated with changes in the visual field indices [14]. Wang et al. [15] found that a decrease in peripapillary VD precedes structural defects and interpreted vascular disorders as an etiological component of glaucomatous optic neuropathy. Using LSFG, Petrov et al. [16] identified a significant decrease in the integrated parameters of blood flow (blood flow in large vessels, tissue blood flow, and total area blood flow) in the optic disc and peripapillary retina, as well as changes in pulse wave parameters.

CHOROID

Published data report a decrease in choroidal blood flow in glaucoma based on OCTA results and a correlation of the results obtained with structural and functional changes [17, 18]. Kim and Lin revealed a relationship between a decrease in the choriocapillaris density in POAG and a decrease in RNFL thickness [19, 20]. Kim et al. [21] found a significant decrease in density of the peripapillary choroidal vessels in glaucoma compared with healthy matched eyes. Rao et al. [18] and Lee et al. [22] described the relationship between choroidal blood flow deficiency and progression of visual field defects. Jo et al. [23] revealed negative changes of the visual field indices over 3 years in 70% of patients with reduced choroidal blood flow and only in 22% of individuals with normal values. Park et al. [24] followed-up 108 patients with POAG for 2.6 years and analyzed the perimetry parameters. They determined that a decrease in peripapillary choroidal VD can be considered a predictor of glaucoma progression; however, no correlation of this parameter with structural OCT changes was noted. Bhalla et al. [25] revealed a relationship between peripapillary choroidal VD and the optic neuropathy stage and suggested using this criterion in the diagnosis of glaucoma and predicting its course.

GLAUCOMA AND MYOPIA

The incidence of myopic refraction among children and adults is 11.7% and 26.5%, respectively. The prevalence of myopia has increased almost 3-fold over 20 years [26]. Currently, glaucoma incidence in patients with myopia is also increasing [27]. Biomechanical properties of fibrous membranes — corneal hysteresis, lamina cribrosa, and optic disc sensitivity to ocular hypertension — have been demonstrated to change when axial length increases in

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myopia [28, 29]. In addition, there is evidence suggesting higher IOP in patients with increased axial length [29, 30].

Degeneration and dystrophy of the posterior segment in myopia and glaucoma generally have common etiological factors and, one way or another, are associated with trophic changes, which is confirmed by several studies of blood flow in the retina, choroid, and retrobulbar vessels. OCTA shows that the severity of choroidal blood flow deficiency and a decrease in vascular density in patients with glaucoma and myopia is significantly higher than in patients with myopia alone. Laser Doppler flowmetry in patients with glaucoma demonstrated that a decrease in subfoveal choroidal blood flow velocity depended on refraction and was higher in myopia than in emmetropia [31]. A similar trend was noted when comparing macular [32] and peripapillary [33] vascular density in the same patient groups. Na et al. [34] reported a decrease in choroidal blood flow in the areas of visual field defects in patients with glaucoma and myopia. Shin et al. [35] found a relationship between the degree of decrease in choroidal blood flow and the glaucoma stage in patients with myopia.

Despite the individual and circadian variability in choroidal thickness [36], a series of publications was focused on studying its structural characteristics and hemodynamics in patients with myopia and glaucoma. Fujiwara et al. [37] noted choroidal thinning with each diopter of myopia by 8.7 μm, and Ho et al. [38] reported choroidal loss of 6.2 μm. In the study performed by Li et al. [39], subfoveal choroidal thickness decreased by 58.2 μm with each millimeter increase in the anterior-posterior axis. Banitt et al. [40] revealed a decrease in peripapillary choroidal thickness in patients with glaucoma, with no changes in the macula. Other authors demonstrated a decrease in average choroidal thickness in the peripapillary and parafoveal area in patients with NTG [41, 42]. Kurysheva et al. [43] found a significant decrease in average choroidal thickness in patients with the perimetric glaucoma compared with preperimetric glaucoma. One study [44] showed that average thickness of the foveolar choroid in myopia and NTG was half of that in the control group. Eskina et al. revealed a 1.5-fold decrease in choroidal thickness in patients with glaucoma and high myopia compared with patients with myopia and no glaucoma [45].

Mamikonian et al. [46] performed flowmetry in patients with myopia and glaucoma and noted blood supply deficiency in patients without IOP compensation, which depended not only on the degree of IOP increase above the individual normal, but on possible features of ocular vascular system in patients with both diseases.

Konoplyanik et al. [47] performed ultrasound with an assessment of blood flow in the brachiocephalic arteries and intracranial vessels. The authors noted that occlusive and stenotic lesions and pathologically tortuous main head and neck arteries were more common in patients with POAG and myopia than in those with myopia alone.

LSFG data showed a decrease in mean blur rate (MBR), representing microcirculation in the optic disc, in patients with glaucoma and myopia, which allowed determining the glaucoma stage in myopic eyes [48]. Yokoyama et al. [49] revealed a significant correlation between a decrease in the optic disc microcirculation parameters and visual field defects.

NORMAL-TENSION GLAUCOMA

Normal-tension glaucoma (NTG) is a multifactorial disease characterized by progressive apoptosis of retinal ganglion cells with conditionally normal IOP. Compromised ocular blood flow is one of the central factors in the NTG pathogenesis. Vascular disorders, including vasospasm and autoregulatory dysfunction, decrease perfusion of the optic disc, choroid, and retina and lead to glaucomatous optic neuropathy [50].

In 1970s, V.V. Volkov performed studies of the relationship between intraocular, blood, and intracranial pressure. The analysis of signs of an unfavorable glaucoma course with concomitant arterial hypotension showed that blood pressure (BP) should be monitored when assessing the individual IOP normal range [51]. Tielsch et al. [52] determined that BP decrease leads to reduction not only in ocular perfusion pressure, but also in IOP. However, Kosior-Jarecka et al. [53] showed that systemic arterial hypertension was observed in NTG twice as often. The vascular theory of the glaucoma pathogenesis was also confirmed by several Russian studies [54, 55]. Most studies of NTG revealed various blood flow disorders, from blood flow dysregulation to vascular structural changes. Primary vascular dysregulation as a factor of NTG development was called Flammer syndrome, which is characterized by low BP, cold extremities, insomnia, and increased resistance to blood flow in retrobulbar vessels [56, 57]. Optic disc hemorrhages, pathognomonic for NTG, are associated with episodes of nocturnal systemic hypotension [58]. Examination of patients with NTG and Raynaud disease (vasospastic disorder, angiopathy mainly affecting the small terminal arteries and arterioles) suggested that NTG progression with low IOP may be more dependent on peripheral vasospasm [59].

Fundus fluorescein angiography revealed lengthening of the arteriovenous phase in NTG, indicating compromised retinal blood flow [60, 61]. Other studies in patients with NTG revealed lengthening of the choroidal phase and a significant correlation between the arteriovenous phase lengthening, ocular perfusion and blood pressure [61, 62].

Using ultrasound with an assessment of blood flow in Color Flow Doppler and Pulsed Wave Doppler modes, most authors reported a decrease in blood flow velocity and an increase in peripheral resistance index in the ocular artery and short posterior ciliary arteries in patients with NTG [63–67].

Several authors revealed signs of ischemic damage to small vessels and atrophy of the corpus callosum in combination with cerebral infarctions in patients with NTG using magnetic resonance angiography (MRA) [68–70]. Moreover, NTG showed a deeper defect of the paracentral visual field in patients with an MR pattern of ischemic cerebral lesions compared with patients without ischemic brain changes [71].

LSFG demonstrated a decrease in the pulse wave parameters in moderate NTG compared with the normal range and advanced disease, which confirms the role of increased vascular resistance in the glaucoma pathogenesis [72]. Mursch-Edlmayr et al. [73] studied microcirculation of the optic disc and revealed a decrease in MBR and pulse wave (BOT and FAI) in patients with glaucoma. Takeyama et al. [74] carried out a comparative assessment of the diagnostic value of OCTA (VD of the optic discs and peripapillary retina) and LSFG parameters (MBR of the entire studied optic discs, MBR of large vessels, and MBR of the microcirculatory bed) and found a significant decrease in all these parameters of microcirculation in NTG.

GLAUCOMA AND CHANGES IN BLOOD PRESSURE

Studies of the relationship between IOP and blood pressure are still ongoing. Various authors suggest that both arterial hypertension and hypotension can be both compensatory protective mechanisms and risk factors for progression of glaucomatous optic neuropathy [75, 76]. Nocturnal hypotension, and abnormal circadian BP fluctuations are considered potential systemic vascular risk factors for glaucoma [75, 77].

Arterial hypertension

Currently, high BP induces an IOP increase in two possible ways. Firstly, an increase in volumetric blood flow and capillary perfusion pressure in the ciliary body stimulates high aqueous production, and, secondly, episcleral venous pressure decreases aqueous outflow [75, 78]. An increase in BP by 10 mmHg raises IOP on average by 0.28 mmHg.

A series of studies showed that, on the one hand, arterial hypertension can increase volumetric blood flow and ocular perfusion pressure; on the other hand, a chronic persistent increase in BP contributes to progressive endothelial dysfunction caused by hypertensive microvascular damage, which suppresses vasoreactivity and leads to hypoperfusion of blood vessels [79, 76]. A meta-analysis performed in 2020 found that arterial hypertension is the most significant risk factor for POAG of systemic vascular diseases [80]. Gangwani et al. [81] reported that elevated BP significantly correlates with thinning of the retinal nerve fiber layer and increased IOP.

Arterial hypotension

Currently, BP is known to be a variable parameter controlled by the normal circadian rhythm. Healthy individuals experience a physiological nighttime BP dipping of about 10%–20% compared with daytime. Some conditions may lead to an abnormal nighttime BP dip and increase the risk of pathological changes in several organs and systems, including the optic nerve [75]. Although published data are contradictory, over 10 clinical studies aimed at BP monitoring have confirmed that nocturnal systemic hypotension is a risk factor for development and progression of open-angle glaucoma [82]. A BP decrease even by 10%–20% is considered to contribute to a significant drop in ocular perfusion pressure, with a significant risk of progression of glaucomatous optic neuropathy [83]. As these pressure fluctuations are within the physiological range, a mechanism of insufficient vascular autoregulation is possible. Melgarejo et al. [84] showed that an increased risk of glaucoma is associated with a sharp, approximately 20% drop in nocturnal BP, irrespective of the overall BP. Raman et al. [85] noted that in patients with glaucoma and diastolic ocular perfusion pressure below 35 mmHg, the disease progresses 2.3 times more often than in patients with pressure above 43.7 mmHg. Pillunat et al. [86] proposed the so-called Dresden safety range for nocturnal BP in POAG ranging from 65 to 90 mmHg. Kwon et al. [58] determined optimal minimum nighttime diastolic BP of 60–70 mmHg. Structural damage to the optic nerve progresses slower in patients with controlled IOP and nighttime BP within this range.

A retrospective cohort study performed in 2020 demonstrated that minimal daytime systolic and diastolic BP, as well as nocturnal hypotension, may be a potential risk factor for glaucoma progression [87]. Patients with systolic BP below 107 mmHg showed a more pronounced thinning of the peripapillary retinal nerve fiber layer, and if diastolic BP was below 63 mmHg, thickness of the inner plexiform layer and ganglion cell layer in the macular area was significantly decreased, which confirms the need to maintain target daytime BP [87]. Jammal et al. [83] performed the Duke Glaucoma Registry analysis and revealed an accelerated progression of RNFL thinning in patients with glaucoma and lower mean, systolic and/or diastolic BP while on systemic hypotensive therapy [83].

CONCLUSION

Modern clinical ophthalmology offers a wide range of methods for examining ocular blood flow. The noninvasive techniques described in this review are becoming increasingly relevant for the diagnosis of vascular disorders in various eye diseases, including glaucoma. An integrated approach combining various methods of studying ocular blood flow seems to be the most promising for providing complete understanding of microcirculation and blood perfusion. It opens up new aspects of the pathogenesis of glaucomatous optic neuropathy and allows for a more accurate diagnosis of the disease and creation of a personalized treatment approach.

ADDITIONAL INFO

Authors' contribution. All authors made a substantial contribution to the conception of the study, acquisition, analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study. Personal contribution of each author: Yu.S. Petrov, E.N. Orlova, T.N. Kiseleva, T.D. Okhotsimskaya, O.I. Markelova — design, collecting and preparation of samples, data analysis, writing the main part of the text, literature review, making final edits.

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