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Myopia and Some of its Complications

Sardorbek Khaydarov
Fergana Medical Institute of Public Health

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ABSTRACT

This review article examines modern views on etiological factors and the pathogenesis of complications associated with myopic refraction. Domestic and foreign studies aimed at identifying the mechanisms of development of this pathology using modern diagnostic methods are analyzed. Also summarized is the literature data that myopia is characterized not only by changes in refraction and the relationship of the anatomical structures of the eye, but also by a number of pathological processes associated with impaired hemodynamics and hydrodynamics of the eye.

INTRODUCTION

About 1.6 billion people in the world have refractive errors. According to J.H. Kempen et al. by 2025, 2.5 billion people will suffer from myopia [1]. Myopia continues to be one of the most common eye diseases in the world and the most common cause of vision loss. According to the World Health Organization, the number of people suffering from myopia in developed countries varies from 10 to 90%.

MATERIALS AND METHODS

The World Health Organization has chosen low vision with uncorrected refractive errors as one of the leading areas for eliminating preventable blindness by 2025 [4]. Uncorrected myopia creates difficulties in performing visual work, reduces professional adaptation and worsens the quality of life [2]. Myopia can be accompanied by retinal detachment and myopic maculopathy, which are the cause of disability in young working age [3]. When taking into account all age groups of the population in the general structure of visual disability, myopia is 18.0% and occupies the third ranking place.

Myopia is a polyetiological disease. There are many theories about the origin of myopia.

Thus, J. Otsuca in 1956 associated the occurrence of myopic refraction with the pathological tone of the ciliary muscle, leading to a weakening of accommodation. As a result, the ciliary muscle, and then the choroid, atrophy, and the sclera stretches.

According to the theory of A.I. Dashevsky, formulated by him in 1973, the formation of myopic refraction is also based on the weakening of the accommodative ability of the ciliary muscle, but due to autonomic dystonia and unfavorable conditions for near visual work. This leads to the formation of prespasm, and when it intensifies the spasm of accommodation, inhibition of the convergent, accommodative and pupillary reflexes occurs, which leads to persistent tension of the extraocular muscles. An increase in intraocular pressure during temporary convergent

elongation of the eye and the accumulation of residual microdeformations in the sclera lead to the development of axial myopia.

RESULTS AND DISCUSSION

And genetic linkage studies have identified 18 possible loci on 15 different chromosomes that are associated with myopia, but none of these loci are part of the genes that cause myopia. Instead of a simple single-gene locus controlling the onset of myopia, the cause may be a complex interaction of many mutated proteins acting in concert. The hereditary factor can manifest its effect in the form of morphological inferiority of the ciliary muscle, anatomical features of its attachment, innervation, blood supply, as well as in the form of genetic inferiority of collagenogenesis of the sclera. Experimental studies have shown that visual information based on the feedback principle by changing the production of neurotransmitters by retinal cells can change the biology of the scleral matrix, in particular, change the level of synthesis of scleral proteoglycans [3]. Violation of the biomechanical properties of the scleral capsule, caused by changes in its metabolism and microstructure, constitutes the pathogenetic basis for the progression of myopia.

Significant changes, on which the prognosis for vision with myopia primarily depends, occur in the central part of the fundus. These changes are degenerative in nature. For example, E.S. Avetisov and L.P. Flick developed a classification of changes, according to which 5 stages are distinguished. The first stage includes initial changes in the optic nerve head in the form of a scleral ring, the formation of cones up to ¼ of the disc diameter (DD). During the second stage, initial disturbances in fundus pigmentation, changes in the shape and color of the optic nerve head, and cones of up to 1/5 DD appear. The third stage is characterized by pronounced disturbances in the pigmentation of the fundus, an increase in the spaces between the choroidal vessels, large cones - up to 1 DD. The fourth stage includes depigmentation, the cone is more than 1 DD. In the fifth stage, an extensive cone of more than 1 DD is observed, a true staphyloma of the posterior pole [1].

Myopic transudative dystrophy is characterized by the early development of newly formed vessels.

That is, the basis of transudative maculopathy is choroidal neovascularization. Choroidal neovascularization is a slowly progressive process manifested by the growth of pathological vessels of the choroid and the development of fibrovascular tissue under the retinal pigment epithelium and neuroepithelium. Choroidal neovascularization has a similar structure to choriocapillaris and is a continuation of the choroidal vessels. The appearance of a neovascular membrane is a response to ischemia of retinal tissue. But the question of the true cause of the disorders and whether the disorders preceding the formation of choroidal neovascularization are a consequence of elongation of the eyeball or a genetically determined abiotrophic process remains unclear [4]. When the axial length of the eye is over 26.5 mm, the incidence of choroidal neovascularization is 5–10% of all myopes with this type of refraction; subfoveal localization of the neovascular membrane is most often detected. Newly formed vessels tend to evolve. Quite rapid growth of the neovascular membrane in area and the addition of fibrous scarring are noted. In myopia, subretinal neovascular membrane (SNM) is usually divided into active and inactive forms [3]. Active SUI is characterized by significant extravasation and is easily detected during fluorescein angiography. The main feature is hyperfluorescence in the early phase, which increases as the study progresses and is accompanied by dye extravasation in the late phase [4]. The inactive subretinal neovascular membrane stains faintly and does not bleed through.

The diagnosis of “active SUI” can be made if 3 signs are present:

1. subretinal hemorrhages;

2. the presence of a lesion in the area of the complex – retinal pigment epithelium (RPE) – Bruch's membrane – choriocapillaris;
3. an increase in the average thickness of the retina in the foveal zone;
4. increase in total macular volume;
5. an increase in the volume of the retina in the foveal zone.

Professor G. Coascas et al. in 2015, they formulated criteria for the activity and inactivity of newly formed vessels using optical coherence tomography in the angio mode (angio-OCT) [5].

They included the following activity criteria:

1. Tortuous, well-defined vessels.
2. Numerous small capillaries.
3. Presence of anastomoses and loops.
4. Presence of peripheral arcades.
5. Presence of a perifocal hypo-intense halo.

Accordingly, the criteria for inactivity are:

1. Linear newly formed vessels (long, thread-like).
2. Rare and voluminous vessels.
3. No loops.
4. Absence of a perifocal hypo-intense halo.

The growth of vessels from the choroid under the neuroepithelium and retinal pigment epithelium is based on complex biochemical and immunological mechanisms, the nature of which is still insufficiently deciphered.

In addition to the changes in the retina and choroid described above in patients with myopic refraction, disorders often occur that combine changes in both hemodynamics and hydrodynamics with the development of glaucoma, the course of which also has its own characteristics in myopia.

Previously, the role of increased intraocular pressure (IOP) in the pathogenesis of glaucoma was considered dominant, but later it became known that the level of intraocular pressure is only one of the risk factors for the development of glaucoma. In addition to IOP, there are a number of other factors that cause damage to optic nerve fibers and ganglion cells. One of the risk factors for the development of open-angle glaucoma is myopia.

The Cochrane Eyes and Vision Group describes glaucoma as a disease characterized by morphological changes of the optic nerve head in the form of enlargement and progression of cupping, followed by a typical narrowing of the visual fields due to loss of retinal ganglion cells and, usually, an accompanying increase in intraocular pressure. A block - weakened sclera, impaired hydrodynamics, intraocular pressure - is one of the basic links in the development mechanism of both myopia [3] and glaucoma, which often causes difficulties in recognizing glaucoma against the background of myopia.

Ophthalmotonus in myopia is, as a rule, underestimated, the optic nerve head may have a larger size, oblique incision, and physiological extended excavation. Currently, there are several classifications of glaucoma, but only the classification of V.V. Volkova, proposed in 2001, distinguishes three clinical forms, among which there is a form with weakening of the strength of

the cribriform plate of the sclera in myopic disease. According to the Blue Mountains Eye Study and the Beijing Eye Study, K. Singh, S.C. Lin, conducted in China and the USA, the risk of glaucoma in patients with 6D myopia or more increases significantly, while the main methods used to diagnose glaucoma (computer perimetry, discoscopy, optical coherence tomography of the optic nerve head) often do not allow reliably differentiate the pathological process in the early stages of development from the physiological state characteristic of myopia.

CONCLUSION

Thus, the presented review of the literature shows that myopia is characterized not only by changes in refraction and the relationship of the anatomical structures of the eye, but also by a number of pathological processes associated with impaired hemodynamics and hydrodynamics of the eye, which is accompanied by a whole cascade of biochemical reactions with the formation of chronic neurodegenerative processes. The lack of a holistic understanding of the etiology and pathogenesis of these diseases dictates the need for their further study.

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