Short Communication
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New concept of etiopathogenesis and treatment of primary open-angle glaucoma

Abstract

Primary open-angle glaucoma (POAG) is one of the main causes ofirreversible blindness. There is no single theory of POAG development. The trigger ofthe disease is unknown. Treatment is ineffective.

The author's concept: the trigger of the glaucomatous process is a genetically programmed small distance between the ciliary body and the lens equator (less than the age-related norm) in the presbyopic period of life. This causes weakness of accommodation and triggers further pathological changes in the eye. For proof of concept, large-scale studies of the anterior segment of the eye (ultrasound biomicroscopy) should be performed in presbyopes with ophthalmic hypertension, with and without POAG, with measurement of the distance "ciliary body - lens equator". Objective acomodometry is also necessary to determine the volume of accommodation in the above patients. In case of confirmation of the author's concept, a 3-component phased treatment of POAG is proposed: 1. Surgical expansion of the ciliary body ring (scleral thinning) - non-penetrating supraciliary sclerectomy or application of antimetabolites to the supraciliary sclera; 2. Phaco emulsification of the lens with implantation of an IOL model that maximizes the volume of pseudo - accommodation; 3. antiglaucomatous microinvasive surgery.

Keywords: glaucoma, ciliary body, lens equator distance, accommodation weakness,non-penetrating supraciliary sclerectomies, antimetabolite applications

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Introduction

The purpose of the work: to develop a concept of the etiopathogenesis of POAG. Glaucoma is one of the main causes of irreversible blindness.[1] In 2020, 76 million people will experience vision loss due to glaucoma, and in 2040 - 111.8 millionpeople. Primary open-angle glaucoma (POAG) accounts for up to 80% of all glaucoma, the pathogenesis of which is still unclear.[2–4] Intraocular pressure (IOP) is the only risk factor that can currently be modified to partially preventglaucomatous vision loss. Modern glaucoma treatment includes: Rho kinase inhibitors; Latanoprostene Bunod; prostaglandin implants; nanotechnology for drug delivery; cannabinoids; stem cell therapy; neuroprotection; nutritional supplements - but all these methods do not solve the problem of glaucoma.[5,6] In 50% of patients with glaucoma and compensated IOP, glaucomatous damage to the optic nerve continues. This means that we are affecting the symptom of the disease, not its cause.

Due to the lack of a single theory of POAG, the trigger of the glaucomatous process is unknown, and POAG treatment is ineffective. To develop a concept of the etiopathogenesis of POAG and to propose a new strategy for its treatment. We drew attention to the following fact: with ophthalmic hypertension, only 10% of eyes develop glaucoma. Thus, in 90% of cases, it is a benign condition when the increased IOP due to hypersecretion does not lead to loss of visual function. Let's ask ourselves the question: is it possible that ophthalmic hypertension isa protective compensatory reaction aimed at eliminating malfunctions in the structure of the eye? We believe that an increase in IOP (due to increased intraocular fluid secretion)in ophthalmic hypertension is aimed at stretching the fibrous capsule of the eye - primarily its anterior segment - to improve the functioning of the accommodative apparatus. It has been established that the earliest symptom of glaucoma is weakness of accommodation.[7,8] Presbyopes need reading glasses that are 0.5 to 0.75 dioptersstronger than those appropriate for the patient's age and refraction.

How can this be explained?

It is known that the diameter of the lens is constantly increasing (by 20 microns per year). In our opinion, at a certain period of life (more often presbyopic), in anatomically predisposed eyes (which is genetically programmed) with a "tight"anterior segment, the distance between the ciliary body and the lens equator becomes less than the age-related norm. The ciliary ligaments begin to sag slightly, which weakens the effect of the ciliary muscle on the lens, and the muscle partially works "idle". Weakness of accommodation develops. In order to improve the efficiency of the adaptive device, it is necessary to increase the distance between the ciliary body and the equator of the lens. At the stage of ophthalmic hypertension, in most cases (90%), this is achieved by stretchingthe fibrous capsule of the eye under the influence of elevated IOP. In 10% of cases, localized ischemia of the anterior segment of the eye developsin the course of glaucoma. The blood supply to the ciliary body decreases by 2 times.[9] Violation of ciliary trophism triggers pathological biochemical processes in the anterior segment of the eye. Cytotoxic factors lead to obliteration of the drainage apparatus, further increase in ophthalmotonus, weakening of the supporting properties of the sclera and stretching of the eye capsule. This compensatory mechanism is aimed primarily at stretching the anterior segment of the eye, which could lead to an increase in the diameter of the ciliary ring, an increase in the tension of the Zinn' s ligaments and normalization of the accommodation apparatus.

Why are these mechanisms activated?

Because accommodation (scanning for danger at different distances) has played an important role in the survival of humans as a species in the course of evolution. And the human body has developed a number of compensatory reactions - the initial stages of most eye diseases (glaucoma, myopia, cataracts, uveitis, etc.) - aimed at restoring accommodation.

Let's get back to glaucoma

If the decrease in ciliary muscle performance (due to ischemia) is compensated by anincrease in the tension of the ciliary ligaments (due to stretching of the anterior segment of the eye), then accommodation is restored (stabilized glaucoma). Otherwise, the eye falls into a vicious circle with loss of visual function (uncompensated glaucoma).

It should be noted that these mechanisms are also triggered in secondary glaucoma - the anterior segment of the scleral capsule of the eye becomes thinner.[10]

In light of the above, it is interesting to look at the structure of the sclera in thenormal eye: the thickness of the sclera near the limbus is 0.5 mm; at the site of attachment of the extraocular muscles - 0.3 mm; at the equator - 0.6 mm; in the posterior region - 1 mm.[11] In other words, the sclera is much thinner in the anteriorpart of the eye than in the posterior part. Let's ask a question: Wasn't this done by the Creator so that when IOP increases, the anterior segment of the eye (where the accommodation device is located) would be stretched first and foremost and the volume of accommodation would be restored? There are many studies on posterior scleral deformation in glaucoma and myopia (these diseases have common pathogenesis)[11,12] but in our opinion, thebody is primarily interested in stretching the anterior scleral segment.[13]

What do we offer?

To help the eye with ophthalmic hypertension to stretch the anterior scleralmembrane (i.e. to interrupt the glaucomatous process). This should increase the distance "ciliary body - lens equator", restore accommodation and normalize IOP, which will prevent glaucomatous neuro- opticopathy and vision loss in the future. There are studies on changes in corneal thickness (which is part of the fibrous capsule of the eye) during prostaglandin treatment.[14,15] This fact suggests a close relationship between the thickness of the supporting membrane of the eye andthe level of IOP. In our opinion, thinning of the anterior segment of the sclera can be achieved surgically (rounded supraciliary non-penetrating sclerectomies - imitation of anteriorscleral staphyloma in advanced myopia and glaucoma.[16,17] or by applying antimetabolites - 5-fluorouracil, mitomycin C, etc. - to the sclera in the projection of the ciliary body. The parameters of these operations should be established experimentally(since each method has its own potential risks.[18– 20] The concept of POAG etiopathogenesis and treatment proposed by us isconfirmed by R.A. Schachar.[21]The author restored the accommodative capacity of presbyopic eyes by surgically increasing the diameter of the eyeball in the ciliary bodyprojection using radial sclerotomies. The volume of accommodation increased by 4-6diopters. Supraciliary scleral incisions also reduce IOP.[22,23]

Based on the above, we propose to surgically thin the anterior segment of sclera glaucoma. However, due to the fact that the diameter of the lens progressively increases with age and after a certain time after surgery the distance "ciliary body - lens equator" may decrease again (which, in our opinion, explains the temporary effect of surgical treatment of presbyopia by supraciliary radial sclerotomies - the "unoperated eye effect"), the 2nd stage should be phacoemulsification of the lens with implantation of the IOL. The optimal choice of the IOL model is the one with the maximum pseudo - accommodation volume (EDOF, etc.). The 3rd stage is an anti-glaucomatous microinvasive surgery (in case of severe changes in the drainage apparatus of the eye). Among the methods of glaucoma prevention, it is advisable to use walks in thefresh air, moderate sports (when looking into the distance, the sympathetic nervous system is activated - the pupil dilates - the distance "ciliary body - lens equator" increases (synergistically) - the functioning of the accommodation apparatus improves).

A few words about optical correction for glaucoma

Myopic refraction is often present or developing in glaucomatous eyes. We believe that it should be corrected as little as possible, and if possible, not on a permanent basis. Why? In uncorrected myopia, the pupil is wide (stimulation of the sympathetic nervous system), which means that the distance "ciliary body - lens equator" widens and the functioning of the accommodation apparatus improves.[24,25] This creates conditions for the stabilization of glaucoma. If we completely correct myopia, there is no need to activate the sympathetic nervous system, the pupil becomes narrower, the above distance decreases, the functioning of the accommodation apparatus deteriorates, and conditions for glaucoma progression appear. To confirm the proposed concept of POAG etiopathogenesis and its treatment strategy, large-scale studies of the anterior segment of the eye (ultrasound biomicroscopy) should be performed in presbyopes with ophthalmic hypertension, with and without POAG, with measurement of the distance "ciliary body - lensequator". Objective acomodometry is also necessary to determine the volume of accommodation in the above patients. Experimentally, the parameters of scleral thinning operations in the anterior segment of the eye should be determined. In conclusion, I would like to note the following: the Holy Scriptures say that only in Eternity will there be no disease. Our proposed concept of glaucoma development and treatment is unlikely to completely solve the problem of POAG blindness. But we hope to slow down the process of vision loss from this disease as much as possible and give patients a decentquality of life for many years.

Conclusion

A. There is no single theory of POAG development, the trigger of the process is unknown, and treatment is ineffective.

B. The author's concept of POAG etiopathogenesis: the trigger of the glaucomatous process is a genetically programmed small distance between theciliary body and the lens equator (less than the age-related norm) in the presbyopic period of life, which causes poor accommodation and triggersfurther pathological changes in the eye.

C. A 3-component phased treatment of POAG was proposed: - surgical dilation ofthe ciliary body ring (non- penetrating supraciliary sclerectomy or application of antimetabolites to the supraciliary sclera); - phacoemulsification of the lens with implantation of an IOL model that maximizes the volume of pseudo- accommodation; - anti-glaucomatous microinvasive surgery.

D. In order to confirm the author's concept of POAG development and treatment,large-scale studies of the anterior segment of the eye (ultrasound biomicroscopy) should be performed in presbyopes with ophthalmic hypertension, with and without POAG, with measurement of the distance "ciliary body - lens equator". Objective acomodometry is also necessary to determine the volume of accommodation in the above patients. Theparameters of surgical procedures for scleral thinning in the ciliary body projection should be established experimentally.

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Conflicts of interest

The author declares that there are no conflicts of interest.

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