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EC OPHTHALMOLOGY
Commentary

# Glaucoma - A New Look at Etiopathogenesis and Treatment

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**Received:** September 07, 2022; **Published:** September 28, 2022

## Abstract

Primary open-angle glaucoma (OPG) is a multifactorial disease that ranks second among the causes of blindness in the world. There is no single theory of the development of PVCG. The trigger of the disease is unknown.

The author's concept: The trigger of the glaucoma process is a genetically programmed small distance between the equator of the lens and the ciliary body (smaller than the age norm) in the presbyopic period of life. This causes the weakness of accommodation and triggers further pathological changes in the eye.

To confirm the concept, large-scale studies of the anterior segment of the eye (AS-OCT) should be carried out in presbyopes with ophthalmic hypertension, with and without PVCG, with the measurement of the "lens equator - ciliary body" distance. Objective accommodation is also necessary to determine the amount of accommodation in the above-mentioned patients.

If the author's concept is confirmed, a 3-component staged treatment of PVCG is proposed: 1. non-penetrating supraciliary sclerectomy; 2. phacoemulsification of the lens with IOL implantation; 3. anti-glaucomatous microinvasive surgery.

***Keywords:*** *Glaucoma; "Lens Equator-Ciliary Body" Distance; Accommodation Weakness; Non-Penetrating Supraciliary Sclerectomies*

Primary open-angle glaucoma (OPG) is one of the most severe forms of ophthalmopathology, which accounts for about 80% of all glaucomas and ranks second among the causes of blindness in the world.

Epidemiological research data show that PVCG is a complex multifactorial disease and occurs as a result of the combined interaction of risk factors and genetic polymorphisms. Risk factors: systemic - advanced age, racial and hereditary predisposition, vascular diseases, hypercholesterolemia, hypothyroidism, diabetes, smoking; local - individual features of the optic nerve head, drainage system and vascular structures of the eye, the presence of concomitant eye diseases. It has been proven that the influence of genetic factors reaches 60%.

Family history is one of the main risk factors for the occurrence of glaucoma, which is realized in the anatomical features of the anterior part of the eye - reduced depth of the anterior chamber, increased thickness of the lens. The proposed treatment: cataract phacoemulsification with IOL implantation and anti-glaucomatous surgery only slightly slows down the glaucomatous process and postpones vision loss.

Until now, there is no single theory of the development of glaucoma, the trigger of the disease has not been identified. Therefore, the number of people blinded by glaucoma, despite modern advances in the diagnosis and treatment of this disease, does not decrease.

We offer the following view on the etiopathogenesis of PVCG.

An early symptom of PVCG is weakness of accommodation. Presbyopes with PVCG need stronger reading glasses than those corresponding to their age and refraction.

### How can this be explained?

It is known that the diameter of the lens is constantly increasing (by 20 microns every year). We believe that at a certain period of life (more often - presbyopic) in anatomically inclined eyes (which is genetically programmed) with a "tight" anterior segment, the distance between the equator of the lens and the ciliary body becomes smaller than the age norm. Zinn's ligaments begin to sag somewhat, which weakens the effect of the ciliary muscle on the lens, the muscle partially works "idle". Weakness of accommodation develops.

In order to improve the efficiency of the accommodation apparatus, it is necessary to increase the distance between the ciliary body and the equator of the lens. Expansion of the ciliary ring is achieved by local ischemia of the anterior segment of the eye.

It has been proven that in the case of glaucoma, the blood supply of the ciliary body decreases by 2 times. Violation of ciliary body trophism triggers pathological biochemical processes in the anterior part of the eye. Cytotoxic factors lead to obliteration of the drainage apparatus, increased ohthalmotonic, weakening of the supporting properties of the sclera and stretching of the eye capsule. This compensatory mechanism is aimed, first of all, 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 cinnabar ligaments, and normalization of the work of the accommodation apparatus.

If the decrease in the performance of the ciliary muscle (due to ischemia) is compensated by an increase in the tension of the cinnabar ligaments, accommodation is restored (stabilized glaucoma). In the opposite case, a defective circle (uncompensated glaucoma) develops. Note that even with secondary glaucoma, the anterior segment of the scleral capsule of the eye becomes thinner.

The concept of the etiopathogenesis of PVCG proposed by us is confirmed in the work of RA Schachar. The author restored the accommodation capacity of presbyopic eyes by operatively increasing the diameter of the eyeball in the projection of the ciliary body with the help of radial sclerotomies. The amount of accommodation increased by 4 - 6 diopters.

Based on the above, we propose to surgically expand the ring of the ciliary body by forming supraciliary rounded non-penetrating sclerectomies (imitation of anterior staphyloma of the sclera in neglected glaucoma). This should increase the distance "lens equator - ciliary body" and accordingly, restore the performance of the accommodation muscle, stabilize glaucoma. But due to the fact that the diameter of the lens progressively increases with age and after a certain time the distance "lens equator - ciliary body" may decrease (which, in our opinion, explains the temporary effect of surgical treatment of presbyopia with supraciliary radial sclerotomies - "the effect of the unoperated eye"), then the 2nd stage should be phacoemulsification of the lens with IOL implantation.

**Volume 13 Issue 10 October 2022**