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Uveopathies: a new look at etiopathogenesis and treatment

Abstract

Currently, the cause of uveopathies is unknown, there is no radical treatment. The final of these diseases is blindness from glaucoma. The author developed a hypothesis of the etiopathogenesis of uveopathies, according to which the trigger of these diseases is the weakness of accommodation caused by the genetically determined excessively small distance between the ciliary body and the equator of the lens. The following approaches to solving the problem of uveopathies are proposed: a). formation of laser mydriasis (at the initial stages of the process); b). surgical expansion of the ring of the ciliary body - the formation of supraciliary non-penetrating sclerectomies (imitation of anterior staphyloma of the sclera in neglected myopia and glaucoma); in). phacoemulsification of the lens with IOL implantation (optimal final refraction of the artificial eye - simple myopic direct type astigmatism up to 1.5 diopters). It is assumed that these operations will affect the trigger of uveopathy - a genetically determined weakness of accommodation, Uveopathies (Fuchs' syndrome, essential mesodermal progressive dystrophy of the iris, glaucoma-cyclitic crisis) are not fully understood diseases.

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Introduction

The cause of these pathologies is unknown.[1–3] All three nosologies are accompanied by mydriasis, iris atrophy, and increased intraocular pressure. Preventive treatment (corticosteroids, hypotensive drugs) does not stop repeated attacks and progression of these diseases. Operations aimed at improving filtering in the corner of the front camera are not successful. The final uveopathy is glaucoma with loss of visual functions.[4–7]

Purpose

To develop a hypothesis of the etiopathogenesis of uveopathies and to propose new approaches to their treatment.

One of the symptoms of uveopathy is rapid eye fatigue. We assumed that this symptom is related to the weakness of accommodation in the eye affected by uveopathy.

We present a clinical case of Fuchs syndrome.

Patient I.G., 48 years old, was first diagnosed with Fuchs' uveopathy in the left eye during the selection of reading glasses. Complaints of eye fatigue, more in the left eye, when working at a close distance. She had never used glasses before.

Objectively: vis OD: 0.8-0.9 / 1.0 with cyl -1.0D ax 25;

Vis OS: 0.9 does not correct.Intraocular pressure (IOP): 19/27 mm Hg. OD is calm, the diameter of the pupil is 3 mm, the optical medium is transparent, the fundus is normal. OS - calm, multiple stellate precipitates on the corneal endothelium, pupil - partial destruction of the pigment border, tendency to mydriasis (diameter - 3.1 mm); lens - subcapsular vacuoles, in the vitreous body - fine-grained opacities in the posterior parts, fundus - normal.

In May 2023, the patient was treated in the eye department of the Chernivtsi Regional Clinical Hospital (anti-inflammatory, hypotensive therapy).

At discharge: vis OD: 0.8-0.9 /1.0 with cyl -1.0D ax 25;

vis OS: 0.9/1.0 with sph + 0.25D.

IOP = 19/19 mm Hg. Art. OD - unchanged; OS - calm, isolated stellate precipitates on the corneal endothelium, pupil - tendency to mydriasis (diameter - 3.1 mm), lens - subcapsular vacuoles, in the vitreous body - fine-grained opacities in the posterior parts, fundus - normal. Instrumental methods: gonioscopy - the angle of the anterior chamber is open, without features; ultrasound examination of both eyes (B - method) - the retina is adjacent, in the vitreous body of the left eye - heterogeneous preretinal opacities; computer perimetry according to Humphrey - field of vision of both eyes - normal.

In the «cold» period of Fuchs' syndrome, we performed accomodometry in the above-mentioned patient. Proximetry was performed with a standard test object in the form of a Landolt ring (the dimensions correspond to a visual acuity of 0.7-0.8 from a distance of 33 cm). The volume of accommodation (OA) was calculated according to the Donders formula:

OA = P-R, where P is the closest point of clear vision expressed in diopters, R is the clinical refraction of the eye.Refractometer data: OD: sph 0.00, cyl - 1.25D ax 25;

OS: sph +0.25D, cyl 0.00.

Obtained data: OA OD: without correction - 4.25D, with correction - 3.65 D.

OA OS: without correction - 2.35D, with correction - 2.55D.

From the above, it can be seen that without correction the right eye has a greater OA than the left -4.25D versus 2.35D, (this is explained by the fact that simple myopic astigmatism of the direct type up to 1.5 D, increasing the depth of the focal area of the eye, provides the maximum amount of accommodation (in including - pseudoaccommodation) in comparison with other types of refraction.[8] But when the refractive factor does not work - the refractive errors are corrected - the OA in the right eye is still greater than in the left (3.65D vs. 2.55D).

How can this be explained?

The accommodation apparatus consists of the ciliary body, ciliary ligaments and lens. If the distance between the ciliary body and the

equator of the lens at a certain period of a person's life becomes less than the age norm, then the cinnabar ligaments partially sag, their influence on the lens decreases, and accommodation weakens. If the drop in OA exceeds age indicators (including presbyopic ones), then eye pathology develops (myopia, cataracts, glaucoma, uveitis, etc.), which in the initial stages of the process is aimed at increasing OA. This is due to the fact that accommodation (scanning for danger at different distances) played an important role in the survival of humans as a species in the process of evolution. Therefore, the body developed compensatory reactions (various types of eye pathology) designed to restore accommodation. The brain in each case calculates the expediency of forming one or another nosology (or their combination) in such a way that it is as ergonomic as possible.[9] In our opinion, the trigger of uveopathies is accommodation weakness, as we assume that in such eyes, an excessively small distance between the ciliary body and the equator of the lens is genetically determined. This prediction is supported by the data of M.V. Panchenko. with co-authors who, during ultrasound biomicroscopy of the eyes of patients with glaucomatous crises and Fuchs syndrome, found that in the «affected» eyes there was a significant increase in the thickness of the ciliary body in comparison with healthy paired eyes (both in the active phase and in the remission stage).[10,11] It follows that in eyes with uveopathy, the distance «ciliary body - lens equator» should be reduced. Uveopathies are characterized by mydriasis, iris atrophy, and increased IOP.

What are these symptoms aimed at?

Innervation of iris muscles and ciliary muscles is synergistic. When the pupil dilates, the ring of the ciliary body also dilates. Thus, the distance «ciliary body - lens equator» increases and the work of the accommodation apparatus improves. It is not for nothing that mydriasis (due to atrophy of the pupil sphincter), weakness of pupillary reactions is observed in uveopathies - the body is interested in the fact that the distance between the ciliary body and the lens is constantly increased. Changes in the angle of the anterior chamber due to inflammatory-dystrophic processes lead to deterioration of the outflow of intraocular fluid and an increase in IOP. This symptom is aimed at stretching the capsule of the eyeball and, first of all, the front part of the eye, in order to increase the excessively small distance between the ciliary body and the equator of the lens, and thereby facilitate the work of the accommodation apparatus. Another interesting reaction of the eye to the weakness of accommodation is atrophy of the iris up to the formation of through holes in it in essential mesodermal progressive dystrophy of the iris. In our opinion, this is a natural imitation of Laser-Vision glasses, designed to increase the depth of the focal area of the eye and restore accommodation. Cataracts often develop with Fuchs syndrome. This process starts, as a rule, with the formation of myopic astigmatism. This refraction also increases the amount of accommodation.

Why do all these natural compensatory reactions in most cases not stop the progression of uveopathies?

Because they are «delayed», they appear already with a developed clinical picture of diseases, and only soften their course. In addition, the lens continuously grows during life (both thickness and diameter), which also with age leads to a significant decrease in the distance «ciliary body - equator of the lens», especially in eyes where this distance is genetically determined to be excessively small.

How do we see ways to solve the problem of uveopathies?

First, large-scale studies should be conducted: a). objective accommodometry of eyes with uveopathy and paired healthy eyes; b). ultrasound diagnosis of the anterior segment with measurement of the distance «ciliary body - lens equator» of eyes with uveopathy and paired healthy eyes. If there is a statistically significant difference in the indicated indicators in healthy and diseased eyes (with uveopathy - smaller OA and a smaller distance «ciliary body - lens equator»), we offer the following:

a) In the initial stages of uveopathy (before the presence of mydriasis) - form laser mydriasis;

b) To surgically expand the ring of the ciliary body - to form supraciliary non-penetrating sclerectomies (imitation of anterior staphyloma of the sclera in neglected myopia and glaucoma);

c) Perform phacoemulsification of the lens with IOL implantation (calculation of the IOL is such that the final refraction of the artificial eye is simple myopic astigmatism of the direct type up to 1.5 diopters; it is advisable that the optical part of the IOL be smaller than the standard diameter). In our opinion, the listed operations will affect the trigger of uveopathy - a genetically determined weakness of accommodation caused by a «tight» front segment of the eye - and will prevent blindness from glaucoma or significantly postpone it. The indicated ways of solving the problem of uveopathies can be useful for other eye pathologies, the etiology of which has not been established.

Conclusion

A. Currently, the cause of uveopathy is unknown, there is no radical treatment. The final of these diseases is blindness from glaucoma.

B. The author developed a hypothesis of the etiopathogenesis of uveopathies, according to which the trigger of these diseases is the weakness of accommodation caused by the genetically determined excessively small distance between the ciliary body and the equator of the lens.

C. The following approaches to solving the problem of uveopathies are proposed: a). formation of laser mydriasis (at the initial stages of the process); b). surgical expansion of the ring of the ciliary body - the formation of supraciliary non-penetrating sclerectomies (imitation of anterior staphyloma of the sclera in neglected myopia and glaucoma); c). phacoemulsification of the lens with IOL implantation (optimal final refraction of an artificial eye - simple myopic direct type astigmatism up to 1.5 diopters).

D. It is assumed that the listed operations will affect the trigger of uveopathy - the weakness of accommodation caused by the «tight» front segment of the eye - and will prevent or significantly postpone blindness from glaucoma.

E. These approaches can be useful for other eye pathologies, the etiology of which has not been established.

Note: Measurement of the distance «ciliary body - lens equator» in our patient I.G. with Fuchs syndrome was not performed for technical reasons.

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None.

Conflicts of interest

The author declares that there are no conflicts of interest.

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