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Age maculodystrophia: the hypothesis of etiopathogenesis

Abstract

Age-related macular dystrophy (AMD) is one of the main causes of vision loss in old age. The etiopathogenesis of AMD has not been fully elucidated, therefore there is no radical treatment of AMD.

The proposed hypothesis is that the trigger of AMD may be a genetically determined weakness of accommodation (related to an excessively small distance «ciliary body - lens equator»). It is necessary to carry out objective accomodometry, OCT diagnostics of the distance «ciliary body - lens equator» in older people with healthy eyes and with ophthalmopathology (including AMD). If the hypothesis is confirmed, it is proposed to: a) surgically expand the ring of the ciliary body; b) to form a simple direct myopic astigmatism of up to 1.5 diopters on an artificial eye with AMD (by calculating the optical power of the IOL or modeling the corneal surface with a femtosecond laser).

Keywords: age related macular dystrophy, accommodation weakness, simple direct myopic astigmatism up to 1.5 diopters

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Introduction

Age-related macular dystrophy (AMD) is one of the main causes of vision loss in old age.[1–5] Currently, there is no radical treatment for this disease.[1,4,6,7] The «dry» form of AMD is not treated, with the «wet» form, anti-VEGF therapy is used, which is very traumatic, does not give results in all patients, and has long-term side effects. Stem cell strategy, gene therapy are innovative methods, but there are still many unworked points. What explains the difficulties in the treatment of AMD? This is due to the fact that AMD is a multifactorial disease, the etiopathogenesis of which has not been fully elucidated.[3,8,9]

Scientists have developed 4 theories of AMD:

1) Primary aging of the retinal pigment epithelium and Bruch's membrane;
2) Damage to the retina by products of lipid peroxidation;
3) Primary genetic factors;
4) Pathological changes in the blood supply of the eyeball.[1]

None of the specified theories helped solve the issue of AMD treatment. In our opinion, modern therapy affects individual mechanisms of the pathological process in the retina, and not its cause. We offer our view on the etiopathogenesis of AMD. It is known that after the age of 40 the accommodative ability of the eye drops and presbyopia develops. This process is explained by the loss of elasticity of the lens, weakening of the ciliary muscle. But RASchachar restored the accommodative capacity of the presbyopic eye by operatively increasing the diameter of the eyeball in the projection of the ciliary body (radial sclerotomies). The volume of accommodation increased by 4-6 dioptersSo, the main cause of presbyopia is not involutive processes in the lens and ciliary muscle. Against the background of presbyopia, age-related ophthalmopathology (cataract, glaucoma, macular dystrophy) develops - the etiopathogenesis of which is also not fully understood. It is logical to assume that the cause of these processes can be one and the same.

Our concept: since accommodation (the ability to scan for danger at different distances) has played an important role in the survival of humans as a species in the process of evolution, the human body has developed compensatory reactions aimed at maintaining the accommodative capacity of the eye. If the loss of accommodation exceeds presbyopic indicators, age-related ophthalmopathy develops. Cataracts, glaucoma, and macular dystrophy in the initial stages provide an increase in the amount of accommodation.[10]

How does this happen?

If at a certain age the distance between the ciliary body and the equator of the lens becomes less than the physiological (presbyopic) norm (which is genetically programmed), the ciliary ligaments sag a little more (than in presbyopia), their influence on the lens is more pronouncedly weakened, and the volume of accommodation is significantly reduced. The brain controls this situation and triggers the development of compensatory reactions to restore accommodation (in each specific case, the most ergonomic way to solve the problem is calculated: cataract, glaucoma, AMD or their combinations).

If it is AMD, then the formation of drusen in the central zone of the retina moderately reduces vision, which facilitates the work of the accommodation apparatus. If this is enough to restore the volume of accommodation - the process stabilizes («dry form»). If not, the eye falls into a vicious circle and develops a «wet form» of AMD with a practical loss of central vision. The main factors of the occurrence of AMD - age, smoking, heredity.[11,12]

- can lead to an excessive narrowing of the pupil and, accordingly, a decrease in the distance «ciliary body

- equator of the lens» (synergistic innervation of the muscles of the ciliary body and iris).

From this point of view, it is interesting to consider the occurrence of AMD after cataract surgery. Cataract itself is a compensatory reaction of the body to a drop in the accommodative ability of the eye (note that the starting refraction of a cataractous eye is usually myopic astigmatism). B.L. Radzihovsky determined that in the initial stages of cataract, the volume of accommodation increases by 2 times.[13–15] In other words, the brain, starting cataractogenesis, solves the problem of restoring accommodation. When we operate on a cataract, we break

this mechanism, and then the brain triggers another compensatory reaction: a decrease in central vision (drusen or neovascularization in the macula). The work of Sandberg MA.[15] is very indicative in this regard: hypermetropia of 0.75 diopters or more is a serious risk factor for the transition of the «dry form» of AMD to neovascular AMD. This study shows the role of tension of weakened accommodation (on the background of an excessively small distance «ciliary body - lens equator») in the emergence of a dramatic situation - the formation of a «wet form» of AMD.

To confirm the proposed hypothesis of the etiopathogenesis of AMD, large-scale studies of people over 50 years of age with healthy eyes and various ophthalmopathology, including AMD, should be conducted. Objective accommodometry, OCT - examination of the anterior segment of the eye with measurement of the distance «ciliary body - equator of the lens» is required. If our hypothesis is confirmed, we see the following ways to solve the AMD problem: Prevention:

a. healthy lifestyle, rejection of bad habits;

b. full correction of hypermetropia;

With a genetically confirmed predisposition to AMD:

I. Expansion of the ring of the ciliary body by forming supraciliary non-penetrating sclerectomies (imitation of anterior staphyloma of the sclera:[16]

II. If cataract surgery is necessary: calculate the optical power of the IOL in such a way that the final refraction of the eye is simple myopic direct-type astigmatism up to 1.5 diopters (which provides the largest volume of pseudoaccommodation);[17] in some cases (when the eye is already artificial), the specified refraction can be achieved by modeling the corneal surface with a femtosecond laser.

III. Perhaps such an approach will not solve the problem of AMD permanently, but, in our opinion, it will be able to delay the decrease in vision and the degree of its loss.

Conclusion

A. The trigger of AMD can be a genetically determined weakness of accommodation associated with an excessively small distance «ciliary body - equator of the lens».

B. To confirm this hypothesis, large-scale studies of people over 50 years of age with healthy eyes and ophthalmopathology (objective accomodometry, OCT diagnosis of the distance «ciliary body - lens equator») should be conducted.

C. If there is a statistically significant difference according to the indicated indicators between healthy eyes and eyes with ophthalmopathology, the following ways of solving the problem of AMD are proposed:

a. Expansion of the ring of the ciliary body by forming supraciliary non-penetrating sclerectomies;

b. During cataract surgery on eyes with AMD, calculate the optical power of the IOL in such a way that the final refraction of the eye is simple myopic direct-type astigmatism up to 1.5 diopters (provides the maximum amount of pseudoaccommodation); in. the specified refraction can be achieved by modeling the corneal surface with a femtosecond laser.

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None.

Conflicts of interests

The authors declared that there are no conflicts of interest.

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