Short Communication
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New concept of etiopathogenesis and treatment of keratoconus (analytical study)

Abstract

There is no single theory of the etiopathogenesis of keratoconus, which creates a serious problem for the prevention and treatment of this disease.

Keywords: etiopathogenesis, keratoconus

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The author's concept

The trigger of keratoconus is genetically programmed weakness of accommodation in emmetropic eyes without physiological astigmatism. Eye fatigue with excessive visual load and general weakening of the body triggers structural changes in the cornea with the formation of myopic astigmatism, which provides the eye with the maximum depth of the focal area and, accordingly, the largest volume of Pseudoaccommodation. If the volume of accommodation is restored and the eye stops getting tired during visual work, the keratoconus stabilizes, if not, the keratoconus progresses, the eye falls into a vicious circle and goes blind. Genetically programmed weakness of accommodation is realized anatomically in eyes in which at a certain period of life the distance between the lens equator and the ciliary body becomes less than the age-related norm.

Surgical ways to increase this distance are as follows: a). laser mydriasis; b). scleral thinning in the ciliary body projection by forming rounded non-penetrating sclerectomies or by applying antimetabolites.

Prevention of keratoconus: healthy lifestyle, limiting gadgets, wearing sunglasses.

To confirm this concept of etiopathogenesis and treatment of keratoconus, large-scale studies of the anterior segment of the eye in patients with keratoconus and in patients with healthy eyes with measurement of the distance "lens equator - ciliary body" should be performed. It is also necessary to determine the volume of accommodation in the above patients.

Keratoconus (KS) is a bilateral and asymmetric ectatic corneal disease that leads to irregular astigmatism and severe visual loss.[1,2] It develops in the 2-3rd decade of life and progresses, as a rule, by the 4th decade. The global prevalence of KS is 1.38 cases per 1000 population.[3]

Modern methods for diagnosing KS (often using artificial intelligence): topo- and tomography of the cornea, pachymetry, measurement of corneal biomechanical parameters, higher-order aberrations.[4–6]

Despite the in depth knowledge of ultrastructural changes and biochemical disorders in KS, the mechanism underlying the disease remains a mystery. The lack of absolute clarity regarding the factors that trigger and stimulate the development of KS poses a serious problem for the prevention and treatment of this pathology.[2,3]

Existing treatment methods, according to the severity of the process in the cornea, include: glasses; rigid gas-permeable contact lenses; cross-linking; intra-corneal segments; deep anterior layer keratoplasty; through keratoplasty.[2,7–10]

In our opinion, these methods only "extinguish" the symptoms of the disease, but do not affect its cause, so they do not prevent the progression of corneal ectasia. Currently, KS is considered a multifactorial disease.

Risk factors include: family history, eye friction, atopy, allergies.[2,7,9,11] But what is the trigger of the disease is unknown.

What provokes the development of KS?

We noticed a number of interesting facts

- Socioeconomic factors (poverty) were more significant predictors of the severity of KS and the need for corneal transplantation compared to clinical factors (which are usually given more attention). Medicare and Medicaid recipients were more likely to undergo corneal transplantation compared to patients with commercial insurance.[12]
- Significantly higher prevalence of KS in the Middle East and South Asia compared to Europe and America.[2,3,9,13]
- Let's look at the problem of CS not locally, but more broadly. Poverty is often associated with malnutrition, bad habits, and neglected chronic diseases. All this weakens the human body. If poverty is one of the determinants of the severity of the course of KS, then this factor can be realized through poor accommodation.

Let's look at this issue in more detail

This is the era of digitalization. A high visual load against the background of a sedentary lifestyle and negative socio-economic factors causes weakness of the ciliary muscle.

In the works of academician N.O. Puchkovskaya, it was shown that KS occurs in eyes that had an ideal optical structure with an almost complete absence of physiological astigmatism before the disease.[14] With a high visual load, such eyes quickly get tired because the focal area of the eye is minimal, and, accordingly, the zone of clear vision is minimal when accommodation is not working. If at the same time the human body is weakened, which means that the ciliary muscle is also weak, which increases visual fatigue.

Since accommodation - the ability of the eye to scan for danger at different distances - has played an important role in the survival of humans as a species during evolution, the human body has developed various compensatory reactions (the initial stages of most eye diseases) to help weakened accommodation recover.

In the case of KS, this is a change in the structure of the cornea to form astigmatism. Let us explain our opinion.

In the early stages of KS, patients have myopic astigmatism.[15] And this is not accidental. Of all the types of refraction, myopic astigmatism provides the eye with the greatest depth of the focal area and, therefore, the greatest amount of pseudo-adjustment.[16] Pseudo-adaptation allows a person to see clearly at different distances without involving the accommodative muscle (i.e., the eye "rests" looking at most objects in space, except for the near vision zone, where the accommodative muscle is activated).

If the total of pseudo-adaptation and true adaptation restores the volume of accommodation and visual fatigue disappears, the KS stabilizes (which we often see in the clinic). If not, the KS progresses (against the background of a severely weakened ciliary muscle, it is almost impossible to avoid the progression of KS): correct astigmatism turns into incorrect astigmatism, while the cornea becomes significantly thinner, cloudy, corneal dropsy occurs - the eye falls into a vicious circle and goes blind.

Interestingly, myopic astigmatism also occurs in the transplanted corneal graft during keratoplasty. This suggests that the cause of KS is not eliminated by the operation and the compensatory reaction to restore weakened accommodation is triggered again!

How to prevent KS?

Increasing physical activity, general health improvement, limiting the use of gadgets.

Particular attention should be paid to children with "perfect" emmetropic refraction (without physiological astigmatism). But there is a nuance here. Not all eyes with "perfect" refraction develop KS under excessive visual load. There must still be genetically programmed anatomical prerequisites for weakness of accommodation - a small distance (less than the age norm) between the equator of the lens and the ciliary body. What happens in this case?

The ciliary ligaments sag slightly, their effect on the lens capsule is weakened, and the volume of accommodation decreases.[17]

An interesting fact is that the incidence of KS in Saudi Arabia is 4790 cases per 100,000 adolescents, while in Russia it is 0.2-0.4.[2] We assume that the reason for such a striking difference may be excessive insolation in the East: the pupil is consistently very narrow and, accordingly, the distance "lens equator - ciliary body" also decreases significantly, which in genetically predisposed individuals and those in a borderline state triggers the process of corneal conization.

To restore the accommodative capacity of such eyes, it is necessary to increase the distance "lens equator - ciliary body".

How does nature work?

In eyes with KS, there is: a). a moderate dilation of the pupil (since the innervation of the iris and ciliary body muscles is synergistic, the distance "lens equator - ciliary body" increases in parallel with mydriasis); b). an increase in intraocular pressure (in order to slightly stretch the fibrous capsule of the eye and also increase the specified distance). But these compensatory reactions are often "late" and only mitigate the course of the disease.

If accommodation in eyes with KS is not fully restored and the process does not stabilize, it is advisable to: a) perform laser mydriasis; b) thin the sclera in the ciliary body projection by forming rounded non-penetrating sclerectomies (imitation of anterior scleral staphyloma in advanced glaucoma and myopia) or by applying antimetabolites.[18]

The parameters of these operations should be established experimentally.

If the proposed surgery does require keratoplasty (although this is unlikely), the refractive errors of the graft will be much smaller and, accordingly, the patient's visual acuity will be much higher than with the traditional approach.

In the light of the above, we can explain cases of paralytic mydriasis after standard keratoplasty: when we replace an astigmatic cornea with a nearly spherical one, sometimes the conditions for the work of the weakened accommodative muscle become "unbearable" (when the focal area almost disappears) - and then the body shuts down accommodation (paralysis of the iris sphincter is accompanied by paralysis of the ciliary muscle sphincter- cycloplegia occurs).

Thus, summarizing the above, we can say that the treatment of KS requires new approaches aimed at restoring accommodation in the affected eyes.

To confirm the proposed concept of etiopathogenesis and treatment of KS, large-scale studies of the anterior segment of the eye in patients with KS and in patients with normal eyes should be performed with measurement of the distance "lens equator - ciliary body" and determination of the volume of accommodation.

Conclusion

1. There is no single theory of the etiopathogenesis of KS, which creates a serious problem for the prevention and treatment of this disease.
2. The author's concept: the trigger of KS is genetically programmed weakness of accommodation in emmetropic eyes without physiological astigmatism. Eye fatigue with excessive visual load and general weakening of the body triggers structural changes in the cornea with the formation of myopic astigmatism, which provides the eye with the maximum depth of the focal area and, accordingly, the largest volume of pseudo-accommodation.
3. If the volume of accommodation is restored and the eye stops getting tired during visual work, the KS stabilizes, if not, the KS progresses, the eye falls into a vicious circle and goes blind.
4. Genetically programmed weakness of accommodation is realized anatomically in eyes in which at a certain period of life the distance between the equator of the lens and the ciliary body becomes less than the age-related norm. Surgical ways to increase this distance are as follows: a). laser mydriasis; b). scleral thinning in the projection of the ciliary body by forming rounded non-penetrating sclerectomies or by applying antimetabolites.
5. Prevention of KS: healthy lifestyle, limiting gadgets, wearing sunglasses.
6. To confirm this concept of etiopathogenesis and treatment of keratoconus, large-scale studies of the anterior segment of the eye in patients with KS and in patients with healthy eyes with measurement of the distance "lens equator - ciliary body" should be performed. It is also necessary to determine the volume of accommodation in the above patients.

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Conflicts of interest

The author declares that there are no conflicts of interest.

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