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Autoimmune scleritis: A new look at etiopathogenesis and treatment

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The eye is a lamp for the body. So. If your eye is healthy, so is everything, your body will be light. And if only Your eye was evil, so was your whole body.Yours will be dark. (The Gospel of St. Matthew 6:22,23).

Summary

At present, the etiopathogenesis of autoimmune scleritis is not fully understood, the trigger of the disease is unknown, and treatment is ineffective.

The author's hypothesis: the trigger of scleritis may be a weakness of accommodation caused by genetic («tight» anterior segment of the eye) and epigenetic (chronic stress) factors. In order to restore accommodation, it is proposed to: a). form rounded non-penetrating supraciliary sclerectomies in the upper half of the eyeball (imitation of anterior scleral staphyloma); b).to form simple myopic astigmatism in the cornea up to 1.5 diopters, which gives the maximum amount of pseudo-accommodation (imitation of the effect of peripheral ulcerative keratitis on the refraction of the eye). c). if necessary, to form laser mydriasis. The parameters of operations should be established experimentally.

This surgical approach can be applied to the treatment of systemic autoimmune diseases, which are often associated with scleritis and may have a common trigger - poor accommodation.

Keywords: autoimmune scleritis, poor accommodation, lens equator-ciliary body distance, anterior scleral staphyloma, simple myopic astigmatism up to 1.5 diopters, laser mydriasis

Introduction

Autoimmune scleritis is a rare inflammatory eye disease caused by occlusive vasculitis of the deep episcleral plexus with a risk of scleral ischemia and necrosis.[1–3]

Complications of anterior scleritis: uveitis, peripheral ulcerative keratitis, cataracts, glaucoma, perforation of the eyeball; complications of posterior scleritis: macular edema, optic nerve, serous retinal detachment, secondary angle-closure glaucoma. These complications lead to serious vision loss.[4–8]

To date, the etiopathogenesis of scleritis remains unclear. A certain role in the etiopathogenesis of this disease is attributed to T- and B-cells, cytokines, metalloproteinases.[5,9] but the corresponding drugs that inhibit their production in the body have not revolutionized the treatment of scleritis.

In most cases (with the exception of infectious etiology), this disease is considered idiopathic,[5,7,3] often associated with systemic autoimmune diseases, which, like scleritis, are considered one of the most mysterious and difficult pathologies to treat.[2,4,10–12]

The lack of accurate knowledge about the cause of scleritis leads to the fact that the therapy of this disease is very aggressive (non-steroidal anti-inflammatory drugs, corticosteroids, immunosuppressants, biological drugs) with a lot of severe side effects, lasts for years and does not prevent recurrence of the disease with vision loss.[13,4,5,11,3,14–16] Obviously, this therapy is symptomatic, not pathogenic.

What can be a trigger for the process in sclera?

We offer our point of view. Let us first consider anterior scleritis.

Necrotic changes in the sclera occur in the projection of the ciliary body; the consequence of scleritis is scleral thinning in this area.[13,7] This mechanism of supraciliary scleral thinning is universal - it is observed in other autoimmune eye diseases with unclear etiology (for example, it has recently been found that thinning of the anterior sclera is a biomarker of myopia progression.[17]

Thus, scleritis changes the anatomy of the anterior segment of the eye.

Why does nature do this?

We believe that the trigger of the process in the sclera is a weakness of accommodation caused by a small (less than the age-related norm) distance «lens equator - ciliary body» (genetic factor) and weakness of the ciliary muscle (epigenetic factor).

When does this happen?

As a rule, in case of chronic stress. Against the background of prolonged stress, the pupil is stably constricted, and the ciliary body ring is narrowed in parallel (synergistic parasympathetic innervation of the iris sphincters and ciliary muscle). At the same time, the zinc ligaments sag slightly, their effect on the lens capsule is weakened, and the volume of accommodation decreases. In order to restore the volume of accommodation, it is necessary to increase the distance «lens equator - ciliary body». There are many mechanisms for this, in this case, scleral thinning due to ischemia or necrosis. Under the influence of intraocular pressure, the diameter of the anterior segment of the eye increases, the distance «lens equator - ciliary body» increases, and if the ciliary muscle is sufficient to restore accommodation, the process stabilizes. If not, scleritis recurs, the eye falls into a vicious circle and goes blind.[18]

What is accommodation in historical terms?

Accommodation (the ability to scan for danger at different distances) has played an important role in the survival of humans as a

species during evolution. Therefore, the human brain has developed many mechanisms aimed at restoring weakened accommodation or minimizing the load on it. This is because the dysfunction of the accommodative muscle (the most active muscle in the human body and requiring large energy expenditures of the brain to ensure its functioning) causes many problems in the body, as the brain, with weak accommodation, is forced to pay more attention to the visual system (which in normal conditions consumes 50% of brain resources), neglecting the needs of other vital organs and systems.

In case of anterior scleritis, the brain tries to «make life easier» for the weakened accommodation by forming a number of complications: -anterior uveitis (designed to increase intraocular pressure to stretch the eye capsule); -cataracts (to reduce visual acuity, myopize the eye); peripheral ulcerative keratitis (to form corneal astigmatism, which will expand the focal area of the eye and increase the volume of pseudo-adjustment).

Posterior scleritis occurs in younger patients compared to patients with anterior scleritis.[19–22]

This, in our opinion, suggests that the weakness of accommodation in posterior scleritis occurs earlier and is more pronounced. Therefore, the brain tries to solve the problem of accommodation (to facilitate its work) by more aggressive methods than in anterior scleritis: edema of the macula, optic nerve, serous retinal detachment - to reduce visual acuity; annular ciliochoroidal detachment, edema of the ciliary body, forward displacement of the iris-crystalline lens diaphragm - in order to: a). myopize the eye (myopia minimizes the load on accommodation); b). form secondary angle-closure glaucoma (increase intraocular pressure, stretch the eye capsule and increase the distance «lens equator - ciliary body», which will improve the functioning of the accommodation apparatus).

A very interesting fact that works in favor of this hypothesis is that in posterior scleritis, internal ophthalmoplegia (mydriasis, paralysis of accommodation) is observed.[5] We explain this phenomenon as follows: the conditions of the accommodation apparatus in these eyes are so unbearable that the brain simply shuts down accommodation (isolated paralysis of the nuclei of the III pair of cranial nerves).

The modern protocol for the treatment of scleritis involves blocking the inflammatory process (non-steroidal anti-inflammatory drugs, corticosteroids, immunosuppressants, biological drugs).[2,7,13,10,11,15,16] Since the purpose of inflammation is to create comfortable conditions for the work of the adaptive apparatus, and we interfere with this process (i.e., go against nature), such therapy lasts for years and is accompanied by recurrent inflammation with vision loss. [4,10,13]

What do we offer?

1. At the first signs of scleritis (optical coherence tomography, ultrasound,[5,9,13,17,22–30] help in the diagnosis), surgically expand the anterior segment of the eye - form rounded non-penetrating supraciliary sclerectomies in the upper half of the eyeball (imitation of anterior scleral staphyloma[31,32] - the area and number are determined experimentally).
2. If the inflammation in the sclera after sclerectomies is not completely resolved, simple myopic astigmatism up to 1.5 diopters can be formed with corneal refractive surgery (gives the maximum amount of pseudo-accommodation due to the depth of the focal area[33] imitation of the effect of peripheral ulcerative keratitis on the refraction of the eye).

If we anatomically create comfortable conditions for the sclerotic eye's adaptive apparatus to work in a timely manner and neutralize chronic stress, then, in our opinion, the scleral process will stabilize and there will be no need for long-term aggressive anti-inflammatory therapy, and patients will have a better chance of not going blind.

As for systemic autoimmune diseases, which are associated with scleritis in 50% of cases,[2–5,10,12,13] there is a wise saying: «We choose stress, and stress chooses disease».

Chronic stress, as we noted above, causes pupil constriction, which activates the parasympathetic nervous system. Prolonged overexcitation of the parasympathetic system leads to disease.[34]

The spectrum of diseases is different for each individual: eye diseases, somatic diseases, mental diseases («where there is a thin line, there is a tear» - that is, what is programmed genetically). It can be a single pathology or several diseases. Such comorbidity is described in the Bible: «Have mercy on me, O Lord... for my eyes are worn out with grief, my soul and my bowels are exhausted.» (Psalm 31:10).

The human optic-vegetative system works in such a way that not only negative emotions affect the pupil, but also the size of the pupil (according to the principle of «direct feedback») can affect the state of the body as a whole.[35,36]

At present, autoimmune diseases (rheumatoid arthritis, systemic lupus erythematosus, granulomatosis with polyangiitis, etc.) are considered incurable.

It is worth noting that scleritis in severe rheumatoid arthritis is often a harbinger of mortality.[5,16] That is, in a critical situation for the body, the brain triggers processes in the eye with excessively weakened accommodation (due to a severe systemic disease) aimed at restoring it, hoping that this will improve the condition of the body as a whole. But this reaction is too late. It is like an «act of despair»!

Necrotizing scleritis develops (to increase the distance «lens equator - ciliary body»), but the ciliary muscle is so weakened that accommodation is not restored. In addition, this scleritis is usually treated aggressively, and the patient quickly dies from damage to internal organs (side effects of drugs,[4]), without waiting for the positive effect of anterior segment expansion planned by nature.

In light of the above (given the close association of autoimmune diseases with scleritis and assuming that they may have a common trigger), we propose to apply to systemic autoimmune diseases the surgical approach described above for the treatment of scleritis. Perhaps this complex will include laser mydriasis.

Indications for eye surgery in autoimmune pathology should be developed on the basis of experimental studies.

Conclusion

- At present, the etiopathogenesis of autoimmune scleritis is not fully understood, the trigger of the disease is unknown, and treatment is ineffective.
- The author's hypothesis: the trigger of scleritis may be a weakness of accommodation caused by genetic («tight anterior segment of the eye») and epigenetic (chronic stress) factors.
- To restore accommodation, it is proposed at the first manifestations of scleritis: a). to form rounded non-penetrating supraciliary sclerectomies in the upper half of the eyeball (imitation of anterior scleral staphyloma); b).to form simple myopic astigmatism in the cornea up to 1.5 diopters, which gives the maximum amount of pseudo-accommodation (imitation of the effect of peripheral ulcerative keratitis on the refraction of the eye); c). The parameters of operations should be established experimentally.

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- This surgical approach can be applied to the treatment of systemic autoimmune diseases, which are often associated with scleritis and may have a common trigger - poor accommodation.

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None.

Conflicts of interest

The author declares that there are no conflicts of interest.

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