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INFLUENCE OF DEXAMETHASONE ON SCLEROMALACIA AND RHEUMATOID SCLERA NODULES IN PROGRESSIVE HIGH MYOPIA (PERFORATIVE SCLEROMALACIA)

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Annotation: This review article is devoted to the analysis of the risk of developing physiological scleromalacia during the development of myopia in adolescents and its prevention. Information about the risk of developing physiological scleromalacia during the development of myopia in adolescents is one of the first markers of the development of myopia, and the primary signs of scleromalacia are observed in high myopia as a result of untreated diseases of the gastrointestinal system, collogenous diseases and chronic diseases. Colitis. To diagnose the risk of developing physiological scleromalacia during the development of myopia in adolescents, it is necessary to identify general diseases of the body, conduct a clinical analysis, and study the morphology and biomechanical structure of the sclera.

Key words: scleromalacia, fibroblast, tropocollogen, perforation.

Van der Hoeve (1931), based on two cases, and drew attention to a condition characterized by the spontaneous appearance of scleral perforations, which he called perforant scleromalacia. A somewhat similar case was previously described by Holthouse (1893), and Kuhnt (1912) reported a case of scleral perforation associated with smallpox vacciniform fluid. Under the term "necrotizing scleritis" Rocha (1933) described two more cases, but later agreed to accept the earlier designation. Van der Hoeve (1934) compared his original material with that of Roche and mentioned two further cases. He noted that diegeasis was bilateral in most patients and began in the anterior sclera with yellow or grayish subconjunctival nodules associated with progressive scleral necrosis progressing to perforation exposing the uvea. In three of these cases there was a history of pre-existing chronic joint disease of the ankylosing type, and he associated this feature with the condition of the eyes, forming a separate syndrome. After van der Hoeve recognized the disease, further cases were reported by Eber (1934), Voino (1935), Kiele (1937), Kast (1937), Soriano and Riva (1937), Urrets Zavalia et al (1937). Verhoff and King (1938) examined fourteen cases, which they found. They were able to confirm the originally described features of ocular manifestations and emphasize the connection with rheumatoid arthritis. They were the first to point out that the histological changes in scleral nodules are essentially similar to those in subcutaneous nodules in rheumatoid arthritis, and suggested that the reaction may be due to the deposition of a chemical produced during certain metabolic disorders; urate deposition in gout was a similar process. Indeed, it is interesting to note that very recently van der Hoove (1948) put forward the theory that perforant scleromalacia results from a disorder of lipid and cholesterol metabolism and that he associates it with a group of diseases that includes Hand-Schiller-Christian disease.

Niemann-Pick disease, Tay-Sachs disease and xanthomatosis. NORMAN ASHTON AND HOBBS Since the publication of Verhoeff and King (1938), numerous cases discussed by Francois (1951) have been added to the literature, and it has become apparent, as Franceschetti and Bischler (1950) pointed out and Malbran and Manzitti (1951) note that there is there is a danger of confusing perforating scleromalacia with a number of other conditions that are significantly different, although in some respects clinically similar. However, in typical cases of perforating scleromalacia, there is clearly an etiological connection between the scleral lesions and concomitant rheumatoid arthritis, and we agree with those who believe that until Until the exact cause of the disease is known, it is more appropriate to consider scleromalacia as a manifestation of rheumatoid arthritis, thus belonging to the group of

collagenoses. This term was originally proposed by Klemperer, Pollack and Baer (1942) to refer to a number of acute and chronic diseases of unknown etiology in which extensive changes in connective tissue are observed, especially in the extracellular substance. This category includes rheumatic fever, rheumatoid arthritis, polyarteritis, acute lupus erythematosus, generalized scleroderma and dermatomyositis.

The change in fibrinoid collagen that is such a striking feature of scleromalacia is what would be expected in a collagen disease affecting tissue as rich in collagen as the sclera. This position, together with further arguments in favor of considering the pathological changes in scleromalacia identical to those that occur in other tissues in collagenoses, was especially well presented by Christensen (1951), Stillerman (1951) and Swan (1951). Although the inclusion of apparently unrelated disorders in the group of collagen diseases does not necessarily imply an etiological connection between them, their development is believed to be associated with immune processes and the deleterious effects of hypersensitivity. The beneficial effects of ACTH and cortisone in such conditions have been demonstrated in detail and appear to be able to control the disease process by suppressing the inflammatory response resulting from antigen-antibody coupling in tissues (Dougherty, 1951). On the other hand, Klemperer (1950) suggested that there may be a primary anomaly in the chemical composition of the ground substance, and since we now know that the formation of collagen tissue and ground substance is influenced to some extent by the adrenal cortex and other hormones (Russell, 1950), It is reasonable to assume that disease of this system may arise from a hormonal imbalance, which ACTH or dexamethasone therapy can correct. Whatever the mechanism of action of such treatment, it would obviously be of interest to determine its effect on the course of perforated scleromalacia, and the purpose of this article is report treatment of such a case with dexamethasone and noticeable changes in the ocular system. the damage is described in detail. The subsequent death of the patient as a result of submitting the eye for pathological examination allowed comparison of the histological picture with the eyes of other patients in whom such treatment was not possible Klemperer (1950) suggested that there may be a primary abnormality in the chemistry of the ground substance, and since we now know that the formation of collagen tissue and ground substance is influenced to some extent by the adrenal cortex and other hormones (Russell, 1950), it is reasonable to assume that disease of this system may arise from a hormonal imbalance, which ACTH or dexamethasone therapy can correct. Whatever the mechanism of action of such treatment, it would obviously be of interest to determine its effect on the course of perforated scleromalacia, and the purpose of this article is to report on the treatment of such case of dexamethasone, noticeable changes in the ocular system.

RHEUMATOID NODES OF THE SCLERA. Disease history. A 34-year-old female patient suffering from progressive rheumatoid arthritis of approximately 5 years' duration was first seen by one of us on March 14, 2021. By this time, perforated scleromalacia was fully established in both eyes. A history of bilateral scleritis that occurred 9 months ago and the very recent appearance of scleral perforations was later obtained from the patient, an assistant ophthalmic surgeon at the Andijan Ophthalmological Hospital. Inspection. Many yellowish episcleral nodules measuring about 3-4 mm. in diameter, and in front of both eyes there were perforations of the sclera, covered with intact conjunctiva, through which a slightly bulging bluish-black choroid could be seen. In some of the perforations, yellowish-white accumulations of scleral tissue were visible, but there was no evidence of an inflammatory cause, and the medium was completely clear. However, mild conjunctival injections and photophobia were observed.

RHEUMATOID SCLERAL NODULES with perforation in one eye. Later, systemic treatment at a dose of 100 mg became possible. daily, and the healing process continued so that by the end of the week several perforations showed a fairly strong covering of new scleral tissue.


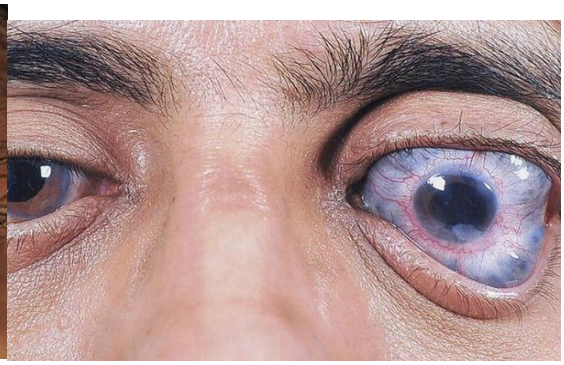

Fig. 1. Scleromalacia of a rheumatoid patient Fig. 2. large temporal perforation of the left

June 14, 2021, and in fig. 2 at the same time - large temporal perforation of the left eye. Coverage of new scleral tissue is almost complete, but in one area an intermediate stage with fine fibrillar formations is still clearly visible.

Upon magnification, the early fibrillary appearance of new scleral tissue is visible. After dexamethasone was discontinued, the conjunctival injection and photophobia returned within about a day, but they resolved again when administration was resumed (September 15, 2021) with hourly dexamethasone 100 mg drops. dexamethasone intramuscularly for 8 days. Significant ocular lesions showed steady improvement with increased scleral regeneration, but none healed completely and some showed little further change until the condition began to deteriorate in mid-December; fresh perforations and episcleral nodules appeared in both eyes (December 28, 2021), dexamethasone drops were administered every 4 hours one week before death.

В хронически инфильтрированной эписклерите позади зоны изъязвления присутствуют участки некроза, а в медиальной прямой мышце наблюдается выраженная инфильтрация хроническими воспалительными клетками. Спереди от язвы инфильтрированная склера и рубцовая периферическая роговица заметно истончены. Передняя часть склеры на височной стороне глазного яблока также хронически инфильтрирована и истощена, наблюдается диффузная гиалинизация стромальных пластинок. На срезах узелков в верхней височной области видно, что они расположены в наружных слоях склеры и в сухожильном расширении места прикрепления наружной прямой мышцы. Они состоят из масс гиалинизированных и полностью дегенерированных склеральных и сухожильных волокон, окруженных бледной, четко очерченной зоной палицадирующих фибробластов, которая, в свою очередь, окружена зоной хронических воспалительных клеток, в которой преобладают плазматические клетки и лимфоциты, а эозинофилы и полиморфно-ядерные клетки особенно малочисленны. В задней половине склеры на носовой стороне имеется несколько узелков (наиболее необычная особенность при склеромаляции).

In chronically infiltrated episcleritis, areas of necrosis are present behind the ulcerated area, and in the medial rectus muscle there is marked infiltration of chronic inflammatory cells. Anterior to the ulcer, the infiltrated sclera and scarred peripheral cornea are noticeably thinned. The anterior part of the sclera on the temporal side of the eyeball is also chronically infiltrated and depleted, and diffuse hyalinization of the stromal plates is observed. Sections of the nodules in the superior temporal region show that they are located in the outer layers of the sclera and in the tendon extension of the attachment of the external rectus muscle. They consist of masses of hyalinized and completely degenerated scleral and tendon fibers, surrounded by a pale, well-defined zone of palatidal fibroblasts, which in turn is surrounded by a zone of chronic inflammatory cells, in which plasma cells and lymphocytes predominate, with eosinophils and polymorphonuclear cells especially few in number. There are several nodules in the posterior half of the sclera on the nasal side (the most unusual feature in scleromalacia).

The anterior choroid is atrophic, while the posterior part has advanced choroidal sclerosis associated with chronic inflammatory infiltration, in which one or two focal accumulations of purulent cells are

present. There is some swelling of the nerve fiber layer in the retina, but there is no significant abnormality in the optic nerve. The histological picture is perforating scleromalacia. The sections described above were compared with histological preparations from six other cases of perforated scleromalacia not treated with dexamethasone, and sections showing approximately the same stage of disease were selected for comparison.

Interestingly, Mundy et al (1951) reached almost identical conclusions from a similar study in a closely related condition. They performed three scleral biopsies in cases of rheumatoid arthritis with bilateral episcleral nodules: one biopsy before and two during systemic cortisone treatment. The histology was similar to that of scleromalacia. The nodules gradually decreased in size and became barely noticeable by the fourteenth day of treatment. Although these investigators were unable to correlate this dramatic clinical improvement with a comparable degree of histologic change, they noted that the cellular infiltration became predominantly mononuclear due to a decrease in the number of neutrophilic and eosinophilic polymorphonuclear cells. The third biopsy showed proliferation of fibrocytes which, as in our case, tended to form a palisade around the fibrinoid necrosis.

Discussion In summary, our study shows that dexamethasone is able to have a beneficial effect on perforated scleromalacia lesions, as evidenced by reduction of nodules and scleral regeneration in necrotic areas.

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