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Corneal siderosis

Case Description

A 77yomalepatient presentedfor the first time in May 2023. His medical history was noticeable fortype II diabetes treated with metformin, and prostate hyperplasia. His ocular history included an ocular trauma in September 2022 when cutting the lawn with a motor scythe. At the time of injury, the ophthalmologist only noticed a subconjunctival hemorrhage in the left eye. In December 2022, vision OS gradually decreased, and a cataract was diagnosed. Cataract surgery with implantation of an intraocular lens was performed in April 2023, however, vision did not improve. The ophthalmic surgeon prompted a CT of the left orbit, and a foreign body could be detected in the eye. With this information, the patient was referred to the University Eye Hospital, Munich.
On examination in May 2023, visual acuity was 20/25 OD and 1/25 OS. Intraocular pressures were normal OU. The right eye showed pseudophakia with an otherwise normal exam. The left eye presented mild upper lid ptosis,pancornealrust-colored stromal opacities and hemosiderin lines as well as mild folds in Descemet's membrane. The endothelium was covered with partly pigmented endothelial precipitates. An intraocular lens was in place following cataract surgery. Heterochromia with a greenish-brown iris discoloration was evident as well as anisocoria. Fundus exam OS was limited by the corneal changes, but did not reveal an intraocular foreign body. Ocular ultrasound was performed and could exclude intraocular foreign material, but ultrasound biomicroscopic examination disclosed an echo-rich structure at 6 o'clock inferiorly in the sclera and the adjacent ciliary body. Pattern-Visual Evoked Potentials (VEP) did not show any reproducible signals OS, but Flash VEP disclosed reproducible answers.The photopic and scotopic answers in the ERG were significantly diminished, and OCT showed an atrophic appearing retina.

In June 2023, the ciliary body foreign body was removed via sclerotomy and the use of an external magnet. Deferoxamine 10% eye drops were instituted 4x/d. As the cornea did not clear, a penetrating keratoplasty was performed on March 6, 2025. Half of the corneal explant was sent for histopathological examination; the remaining was sent for further work ups.

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Macroscopy

4x7,5 measuring half corneal explant with yellowish, hazyappearance

Light microscopy (H&E, PAS, Prussian Blue staining)

Corneal explant covered with multilayered corneal epithelium. The epithelium is absent in the periphery and partly shows single cell edema. The epithelial basement membrane is WNL. Bowman's layer exhibits single breaks. The corneal stroma demonstrates a regular structure. Within keratocytes and throughout the corneal tissue brownish granules are visible. Descemet's membrane is intact with single excrescences peripherally. The endothelium is flattended and reduced in number. The before mentioned brownish granules are also visible in some endothelial cells. Prussian Blue staining discloses iron deposition throughout the cornea.

Diagnoses

Corneal siderosis in a case of ocular siderosis after long-standing iron foreign body of the ciliary body; mild cornea guttata; mild endothelial cell loss

Differential diagnoses

- Iron deposition after corneal refractive surgery
- Iron deposition after orthokeratology contact lenses
- Ocular hemosiderosis after intraocular hemorrhage

Discussion

Siderosis bulbi may develop in an eye with a retained iron-containing foreign body. Von Graefe in 1860 was the first to call attention to the discoloration of the tissue of the eye from retained metallic particles (1), and Bunge in 1890 was the first to apply the name siderosis bulbi (2). The pathological changes depend on the size, chemical composition, and locations of the particle, whereas the posterior segment and the ciliary body are the worst locations (3).

Classical clinical findings in siderosis bulbi include iris heterochromia, pupillary mydriasis, cataract formation, retinal pigmentary degeneration, and an ERG-response that is initially hypernormal, but gradually decreases (3,4). All these features were present in our patient.

Siderotic cataract formation occurred in 11/14 patients with siderosis bulbiand was managed with cataract extraction by Sneed et al. as in our patient. Foreign bodies can be removed by sclerotomy and external magnet – as in our case – and pars plana vitrectomy which is especially helpful in the extraction of posteriorly located foreign bodies and the removal of metallic foreign bodies that have lost their magnetic properties (5). Interestingly, removal of the foreign body can lead to clinical reversal of siderotic findings(6).

It seems that cell damage occurs by deposition of iron as ferritin in the cytoplasm of cells, especially in the form of siderosomes(7).

Ultrastructural studies of cornea, iris, and lens from a case of ocular siderosis due to a retained intraocular foreign body showed widespread degeneration of the lens epithelium, iris stromal cells, and iris pigment epithelium associated with intra- and extracellular siderosomes. The cornea showed siderosomes only within keratocytes in contrast to our patient where iron deposition was noticed within keratocytes but also extracellularly (8). Goldsmith also reports the heaviest siderotic deposits on the anterior surface, sphincter, and dilator muscles of the iris, the trabeculae of the anterior chamber angle, the subcapsular lens epithelium, epithelium of the ciliary body and throughout the peripheral retina (3).

Deferoxamine is a chelating agent with a high affinity for free iron ions. Subconjunctival use of deferoxamine has been shown to prevent the development of ocular siderosis (9-11), however in advanced siderosis, deferoxamine cannot reverse the toxic effects of iron as it cannot remove the bound iron ions from the tissue (10,11). Moreover, deferoxamine-related toxicity limits its use in the management of ocular siderosis (12). Deferoxamine was used in our case as topical eye drops without any noticeable effect.

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