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Siderosis Bulbi Resulting From an Unknown Intraocular Foreign Body

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Siderosis bulbi is a sequel of retained, iron-made, intraocular foreign body (IOFB).[1–13] Iron, released from the IOFB, is toxic to the ocular tissues including the retina and trabecular meshwork[1] and it may cause iris discoloration, mydriasis, anterior subcapsular cataract, reduction in electroretinography amplitude, and glaucoma.[1–13] Prompt surgical removal of the IOFB should be performed to prevent these complications.[8,9]

In this report, we want to present a case of siderosis bulbi with an unknown, retained intraocular metallic foreign body.

CASE REPORT

A 35-year-old man presented with periocular pain, redness, and progressive decrease of vision in his left eye for 1 year. He denied any history of systemic disease. He had no family history of glaucoma. His only history of trauma was a train crash at the age of 2. A complete ophthalmologic examination including dilated fundoscopy was performed by an ophthalmologist at an other hospital in his town, and he was diagnosed with glaucoma secondary to Fuchs' heterochromic uveitis. He has been on brimonidine and timolol maleat-dorzolamide fixed combination twice daily for 1 year to control his intraocular pressure (IOP). Topical corticosteroids were also used to control his pain and redness.

Best-corrected visual acuity was 20/20 in the right eye and counting fingers at 3 m in the left eye. The ocular examination of the right eye was within normal limits. Slit-lamp examination of the left eye revealed heterochromia and clear lens. Iris color in the involved left eye was lighter than the right eye (Fig. 1). Fundus examination of the left eye revealed glaucomatous optic atrophy. Gonioscopic examinations showed open-angle with increased pigmentation. The IOP was found to be 40 mm Hg. Repeated fundus examination after pupillary dilatation revealed an unknown retained IOFB in the nasal part of the retina, which had been missed at the first examination in his town (Fig. 2). The orbital computerized tomography (CT) also showed the IOFB (Fig. 3). The amplitude of the a and b waves were decreased in electroretinography.

He underwent trabeculectomy with mitomycin C to control IOP. One day after surgery the IOP was 8 mm Hg. After 1 week, the encapsulated IOFB (1.5 mm diameter) was removed via pars plana approach by using intraocular forceps. Any evidence of an entrance site of the foreign body could not be found during the surgeries. The IOFB was analyzed and low concentration of iron-containing metallic alloy was found. He was followed-up for about 3 months. At the final examination, the visual acuity was 20/200, the IOP was 8 mm Hg without medications and there was no retinal abnormality. Also, he had no pain, redness, or irritation.

DISCUSSION

Retained metallic IOFB can produce open-angle glaucoma months to years after the injury.[1–13] It is postulated that iron released from the foreign body is toxic to several ocular tissues, including the trabecular meshwork and the retina. Decreased outflow facility is the main cause of glaucoma because of siderosis.[1–7] Iron retinotoxicity leads to a dysfunction of all layers of retina and causes a reduction in electroretinography amplitude which may be reversible in the early period of the disease.[10,11]

Most of the cases with siderosis have a definite history of trauma. But our patient was an accountant and he did not have a definite history of trauma except a train crash 33 years ago. He has been followed-up for glaucoma and heterochromia for the last 1 year. The involved eye was lighter than the other eye because of the damage of iris pigment epithelium. Although both of these symptoms were evidences of siderosis, the diagnosis of the ferrous IOFB was missed at the hospital in his town. Barr et al.[12] reported a case with unexplained heterochromia in whom CT was used to confirm the presence of IOFB which could not found by clinical and X-ray examinations. McGimpsey et al.[13] reported a case with delayed presentation of an IOFB 25 years after the trauma. But different from our case, retinal IOFB was detected at his routine ophthalmologic examination without any signs of

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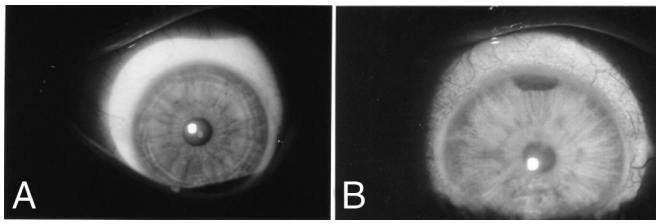


Fig. 1. *Slit-lamp appearance of the right (*A*) and left eye (*B*) at the first postoperative day after trabeculectomy.*

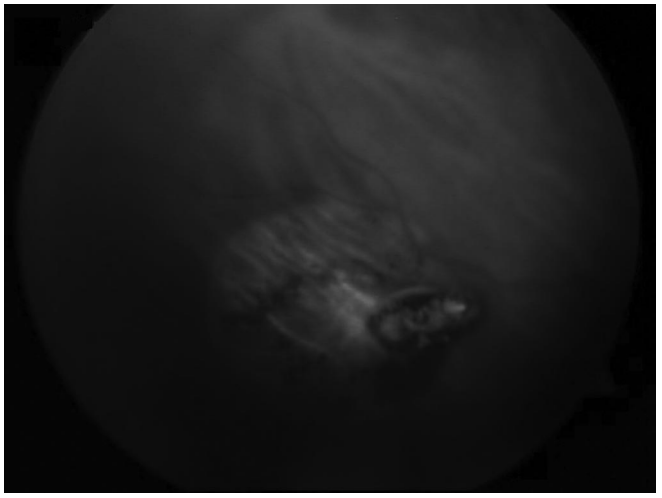


Fig. 2. *Foreign body located in the nasal part of retina.*

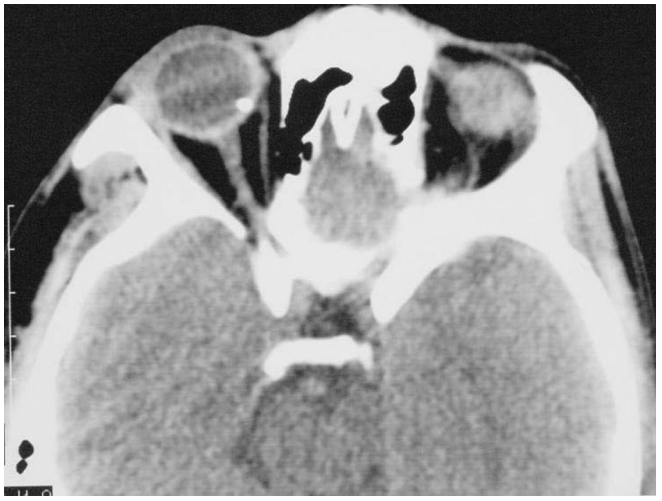


Fig. 3. *Metallic intraocular foreign body identified on computerized tomography scan.*

siderosis such as heterochromia, glaucoma, or a decrease in the visual acuity. The IOFB was considered to be a nonferrous object such as stainless steel or aluminum because of the absence of the evidence of siderosis. Also, he was an adult (33 years old) at the time of trauma. But in our case, the IOFB should have cause phthisis in the eye of a 2-year-old child within 33 years so, the train crash was not considered to be the reason of siderosis. For this reason, the patient was seemed to be unaware of an other innocuous trauma. However, the IOFB in our case was a small, low concentration of iron-containing metallic alloy and it was encapsulated. If the train crash was the reason of siderosis, the IOFB should have been reabsorbed completely and be asymptomatic for 32 years. But this theory seemed almost impossible to us.

Our report emphasizes the importance of detailed examination of such patients with unilateral glaucoma even in the absence of definite trauma to prevent delayed or missed diagnosis of siderosis. These eyes must be examined carefully with dilated indirect ophthalmoscopy, gonioscopy, ultrasound, and CT. Also, the most important factor for the treatment of siderosis is prevention.

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