



Long-standing iron-containing intraocular foreign body without siderosis

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Abstract

An intraocular iron foreign body may cause many ocular complications, sometimes even blindness. Therefore, if possible, it should be removed to avoid ocular siderosis. We report a case in which a foreign body was trapped in the retina for over 33 years, which did not lead to ocular siderosis.

Keywords: Iron foreign body, siderosis.

Introduction

An iron-containing intraocular foreign body usually leads to ocular siderosis if not removed promptly. We report a case involving an iron-containing intraocular foreign body (IOFB) situated on the retina for over 33 years, which did not lead to the expected ocular siderosis.

Case History

A 53 year old man presented to our hospital for routine eye check up. He had no ocular complaints. However, he gave past history of trauma to right eye 33 years earlier. This occurred while he was hammering a piece of iron plate. On consulting an ophthalmologist he was treated with topical antibiotics.

At this presentation, Best corrected visual acuity in both eyes were 6/6. His intraocular pressure were normal in both eyes. On examination, anterior segment showed nuclear sclerosis grade 2 in both eyes, the vitreous was clear. There was a scar along the inferior arcade with fibrous band extending to the periphery and vitreous incarceration in the right eye. On close examination this revealed a foreign body (fig 1). He also had epiretinal membrane over macula (fig 3). There was no evidence of the original injury, as the site of entry was through the sclera, which formed a clinically

undetectable. Self-sealing wound, without lens involvement. USG B scan confirmed the findings detected on fundus examination. In view of his past ocular history, an electroretinogram was performed, revealing generalised decrease in the rod and cone response, which is not consistent with siderosis.



Figure 1. The iron-containing intraocular foreign body is situated within the retina, with vitreous incarceration in the infero-temporal quadrant in the right eye.

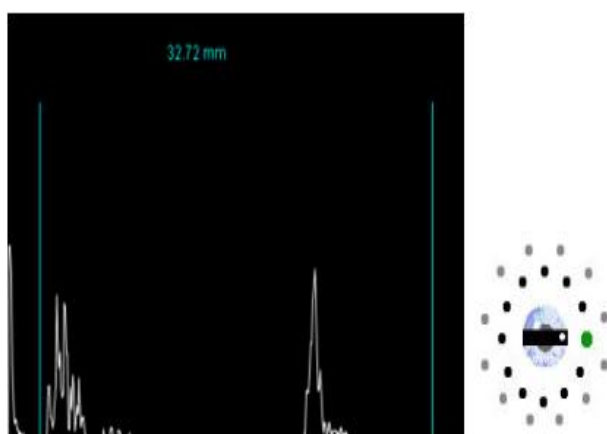
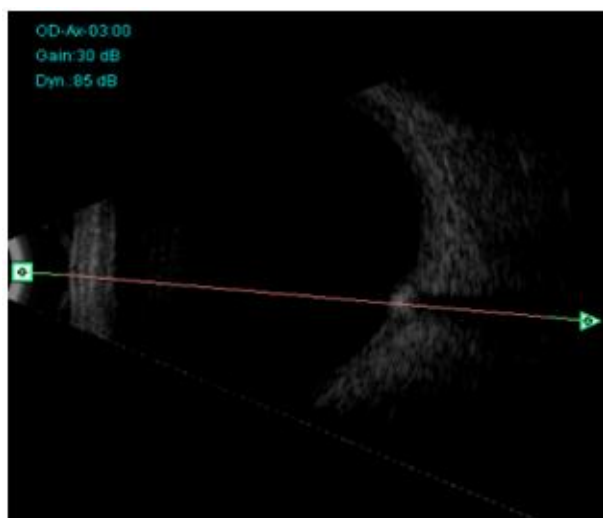


Figure 2. Ultrasound B scan showing an inferior hyperechoic lesion with after shadow and high amplitude in A scan which is highly suggestive of intraocular foreign body(fig 2).



3.CT orbit showing retained intraocular foreign body in the posterior segment of right eye

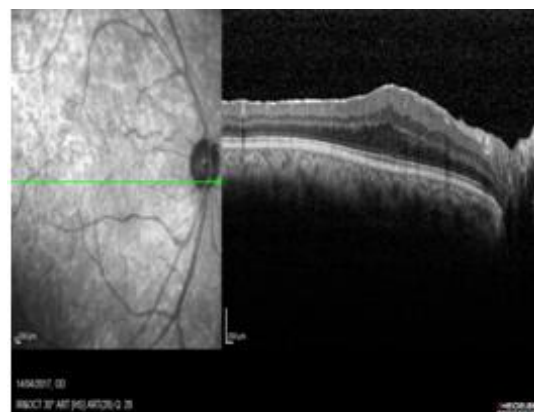


Figure 4.OCT right eye showing epiretinal membrane over macula with normal outer retinal layers. No intraretinal fluid collection.

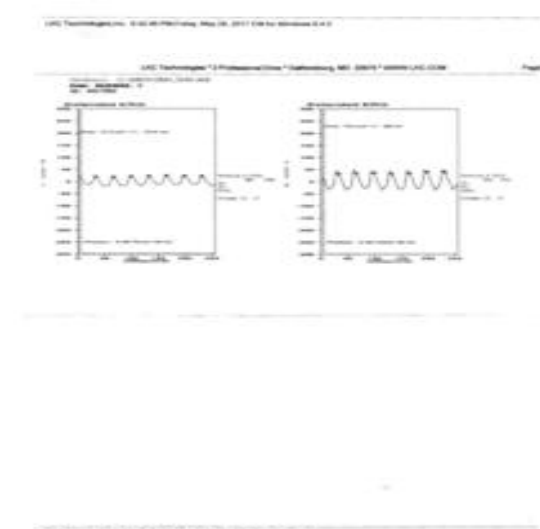
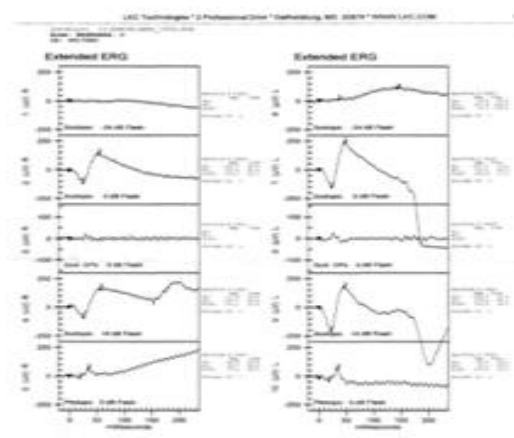


Figure 5. ERG revealing generalised decrease in the rod and cone response, which is not consistent with siderosis

Discussion

Ocular siderosis is a degenerative process caused by a retained iron-containing intraocular foreign body (IOFB).¹ It usually occurs between 2 months to 2 years. The iron ferritin present in the iron foreign body enters the cytoplasm of ocular cells resulting in the formation of siderosomes which result in the release of free radicals producing oxidative damage.² Retinal photoreceptors and RPE cells are more susceptible. This can be detected clinically and by electroretinography.

Clinical signs of ocular siderosis are iris heterochromia, pupil dilatation and poor reaction to light, cataract, brown deposits on anterior lens capsule, vitreous opacity, peripheral retinal pigmentation which eventually progress to diffuse retinal pigmentation, narrowed retinal vessel, optic atrophy and secondary glaucoma.

ERG initially shows an increased a-wave and normal b-wave with time, amplitude of b wave decreases eventually ERG becomes extinguished. After removal of an intraocular foreign body, amplitudes of rod and cone responses will improve; however, the oscillatory potential amplitudes remain unchanged with lower values. These findings suggest that iron retinotoxicity leads to a dysfunction of all layers but the changes may be reversible if timely surgical removal is performed¹.

Our case showed that there was no evidence of ocular siderosis even after 33 years of retained iron-containing intraocular foreign body. This is made possible by the encapsulated gliosis of the foreign body, which protected against ocular siderosis. If late onset ocular siderosis occurs, the offending material should be removed surgically and the patient followed to monitor progression.

In conclusion, a long-standing retained iron-containing intraocular foreign body on the retina outside the macula can have a good outcome, without siderosis developing, but this is rare. A small intraocular foreign body can be missed easily, so thorough clinical and radiological evaluation is most important for all cases of penetrating ocular trauma.

References

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