Unilateral Ocular Siderosis Bulbi Due to Missed Metallic Intraocular Foreign Body Masquerading as Anisocoria of Neurological Origin: A Case Report

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Patient: Male, 38-year-old
Final Diagnosis: Intraocular foreign body
Symptoms: Visual field defect
Medication: —
Clinical Procedure: Intraocular foreign body removal
Specialty: Ophthalmology

Objective: Unusual clinical course
Background: Ocular siderosis is an uncommon cause of vision loss due to a retained ferrous intraocular foreign bodies (IOFB) that cause iron deposition in ocular tissues. The most common manifestations are cataract formation, diffuse pigmentary changes of the retinal pigment epithelium, iris heterochromia, dilated pupils, secondary glaucoma, iritis, and cystoid macular edema.

Case Report: We report a case of 38-year-old man who presented with a left dilated pupil and visual field defect. Neurological examination results were normal. Brain magnetic resonance imaging revealed a gross artifact at the site of the left globe. The visual field test showed a peripheral arcuate nasal visual field defect in the left eye. Ophthalmic examination revealed peripheral pigmentary changes and a black elongated and elevated lesion located very anterior and inferior-temporal and attached to the retina with fibrous tissue. A computed tomography scan revealed a 1×1-mm-round hyperdense IOFB in the left vitreous cavity. The diagnosis of siderosis bulbi secondary to a missed IOFB was established. The patient underwent a pars plana vitrectomy for removal of the IOFB. Two weeks later, rhegmatogenous retinal detachment developed, and repair with silicon oil injection was done. One year after the last operation, the best corrected visual acuity in the left eye was 6/120, with normal intraocular pressure and an attached posterior pole.

Conclusions: This case highlights the importance of investigating for a retained IOFB in cases of unilateral retinitis pigmentosa changes.

Keywords: Eye Foreign Bodies • Retinitis Pigmentosa • Siderosis • Vitrectomy

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Background

Open-globe injury (ogi) is a common cause for significant vision loss, which has a negative effect on patient quality of life. The most common cause of OGI is work-related intraocular foreign bodies (IOFBs), which account for 18% to 41% of all OGIs. More than half of IOFBs (58-88%) are located within the posterior segment. IOFB size and nature, site of injury, and the consequent complications determine the visual prognosis after OGIs [1].

The natural types of IOFBs can be divided into metallic or non-metallic types. More than 80% are metallic in nature [2]. Each type has its own implications on modality of diagnosis, infectious risk, and long-term visual prognosis. The metallic IOFBs are associated with less risk for endophthalmitis. However, they may cause intraocular toxicity that can lead to acute and long-term visual loss, particularly in cases of ocular siderosis [2].

Ocular siderosis is an uncommon cause of vision loss that was first described with the term “siderosis bulbi” [3,4] and results from iron deposition within the ocular tissue from a retained ferrous IOFB. The condition may appear from a few days to many years following injury, and any ocular tissue from the cornea to the optic nerve can be affected. The most common manifestations are cataract formation (especially anterior subcapsular), diffuse pigmentary changes of the retinal pigment epithelium, iris heterochromia, often in conjunction with a dilated pupil, secondary glaucoma, iritis, and cystoid macular edema [4].

We report the case of a 38-year-old man who presented with unilateral ocular siderosis bulbi due to a missed IOFB.

Case Report

A 38-year-old man, who was not known to have any medical illnesses, presented to the neurology department with a mild dilated pupil and reduced peripheral field of vision in the left eye for a duration of 2 months. The patient denied a history of headache, weakness, sensory changes, speech abnormalities, and ataxia. He reported a history of trauma to the left eye, which consisted of a broken piece of a grinding iron machine going into the eye, 6 months prior to his presentation. At that time, he was treated conservatively for corneal abrasion with lubricating and antibiotic eyedrops. The neurological examination was normal at that time.

At his second presentation, a neurological cause was suspected by the neurology team. Accordingly, visual field testing and brain magnetic resonance imaging (MRI) were performed. The brain MRI revealed a gross artifact at the site of left globe

**Figure 1.** Brain and orbit magnetic resonance imaging showed a typical susceptibility artifact that obscured the anatomical structures of the left orbit and surrounding regions. Such artifacts are typically associated with ferromagnetic substances.

According to the aforementioned findings, the patient was referred to our ophthalmic department. On examination, the best corrected visual acuity was 6/6 in both eyes. The right eye was found to be normal. However, a left mild anisocoria was detected. Also, an oblique spontaneously sealed old corneal wound measuring 3 mm and located at 3 o’clock near the limbus was found in the left eye, along with a corresponding iris defect posterior and slightly inferior to the corneal wound (Figure 3). The lens was clear, the anterior chamber was deep and quiet, and the intraocular pressure was normal. During fundus examination of the left eye, peripheral pigmentary changes resembling bone spicules seen in retinitis pigmentosa were revealed. Also, a black elongated and elevated lesion was located very anterior and inferior-temporal and attached to the retina with fibrous tissue (Figure 4). The disc and macula appeared healthy. The diagnosis of a missed IOFB was suspected, and the corneal wound with the corresponding iris defect was the supposed entry pathway.

Subsequently, a B-scan was done and showed a hyperechoic, highly reflective inferior-temporal lesion with minimal shadowing (Figure 5A). An orbit computed tomography (CT) scan was performed and revealed a 1×1-mm-round hyperdense IOFB
Figure 2. Visual field testing showed double arcuate scotoma (A) denser nasally in the left eye and (B) normal in the right eye.
in the left vitreous cavity within the inferior-temporal region, with no associated orbital fractures (Figure 5B, 5C). The right eye globe, pre-septal soft tissue, both optic nerves, extraocular muscles, and lacrimal apparatus were normal.

A full-field electroretinogram was performed, according to the standards of the International Society for Clinical Electrophysiology of Vision, showing that the 5 responses of the standard electroretinogram were normal in the right eye and abnormal in the left eye. The amplitudes of the scotopic rods and combined rod-cone responses were normal in the right eye but reduced in the left eye. Also, wavelets of oscillatory potentials were normal in the right eye but hardly detectable and reduced in the left eye. Furthermore, photopic cone responses and flicker responses were normal in the right eye but reduced in the left eye (Figure 6).

The diagnosis of siderosis bulbi secondary to a missed IOFB was established. The patient underwent a pars plana vitrectomy for removal of the IOFB. The IOFB was adherently encapsulated within the chorioretinal tissue. It was removed after multiple trials by endoforecops, and the dimensions were 3×2×1 mm (Figure 7). Bleeding from the site of where the IOFB was removed developed twice during the course of surgery, and hemostasis was achieved by endocautery and elevating the intraocular pressure. At the end of the surgery, an endolaser was applied around the IOFB site, and sulfur hexafluoride gas was injected. Two weeks later, rhegmatogenous retinal detachment developed, and surgical revision and repair with silicon oil injection were performed. Six months later, the patient underwent silicon oil removal, internal limiting membrane peeling, and phacoemulsification with an intraocular lens implant. One year after the last operation, the best corrected visual acuity in the left eye was 6/120, with normal intraocular pressure and attached posterior pole.

**Discussion**

Ocular siderosis is a result of iron deposition from a ferrous IOFB. It can present as a cataract in the lenses, glaucoma, iris heterochromia, mydriasis, and retinitis pigmentosa changes. On electroretinogram, the changes of ocular siderosis are an increase in “a” waves followed by a continual reduction of “b” waves [1-4].

Zhu et al reviewed and retrospectively analyzed 24 eyes of 24 patients diagnosed with siderosis bulbi. The average age of the patients in their series was 39.9±12.2 years, and the majority of patients (83.33%) reported a history of trauma. The period between the ocular injury and the diagnosis of ocular siderosis ranged from 1 month to 240 months (43.55±68.74 months). The long period for diagnosis was related mainly to the delay of presentation in patients (54.17%). Other factors related to the delay was a missing diagnosis or delayed referral by physicians (25%), absence of history of trauma (16.67%), and undetected IOFB by CT (4.17%) [5].

A careful detailed history and slit lamp examination with high suspicion index for IOFB is essential for any patient presenting with even trivial ocular trauma. In many cases, patients may not experience pain or loss of vision following the trauma [1-5].

B-scan, X-ray, CT scan, and MRI have been used to detect and diagnose IOFBs. The appropriate imaging modality for visualization and localization depends on the suspected nature and location of the IOFB. CT scan is the modality of choice because it is highly sensitive, not operator dependent, does not affect the globe, and can detect single or multiple IOFBs. Using the CT scan, we can check the orbital and facial skeleton, bilateral globes, and retrobulbar area [5,6].

MRI is used only when the presence of a metallic IOFB is ruled out. When the patient has a history of known metallic IOFB or has engaged in high-risk activity, it has been recommended by Brunberg et al to perform orbital plain films before MRI [6]. Metallic IOFB can cause a magnetic susceptibility artifact or metallic artifact on MRI even with small quantities.
Figure 4. (A, B) Multicolor fundus photograph using Heidelberg OCT spectralis 55 degree lens. Both eyes looks normal. (C, D) Multicolor fundus photograph using wide-angle Heidelberg OCT spectralis (102 degree). The right eye is normal, while the left eye showed peripheral pigmetary changes. (E, F) Multicolor fundus photograph using wide-angle Heidelberg OCT spectralis (102 degree). The right eye is normal, while the left eye showed peripheral pigmentary changes and small intraocular foreign body (IOFB) in the far anterior periphery inferotemporal.
Figure 5. (A) B-scan of the left eye showed the hyperechoic shadow of the intraocular foreign bodies (IOFB). (B, C) Computed tomography scan revealed the IOFB.

Within the MRI field, these substances become highly magnetized and can cause massive geometric distortion of anatomical structures [6].

There are few reports demonstrating MRI of a metallic IOFB [7-10]. Most recently, Zhang et al described 2 cases of very small (0.5 mm) intraocular ferromagnetic particles that were visualized using MRI. They suggested that these small particles can be safely visualized on MRI because the particles did not appear to be large enough to cause ocular damage. They concluded that a size of 3×1×1 mm was required to cause movement when exposed to the magnetic field [10].
However, in cases of chronic IOFBs, the removal is governed by ocular siderosis development and the site of the IOFB. If there is siderosis, the IOFB should be removed. Surgically, IOFBs can be removed either by an external approach (sclerotomy with large electromagnet) or by an internal approach (vitrectomy followed by forceps or internal magnet use) [11].

Dhoble and Khodifad introduced a newer surgical technique that is suitable for all sizes of IOFBs, known as the “handshake technique”, in which an IOFB is removed through a sclerocorneal tunnel using 2 intraocular magnets introduced through pars plana vitrectomy sclerotomies [14].

Conclusions

Our study revealed that young men who have had work-related injuries are at the greatest risk for ocular siderosis. Therefore, we recommend that protective glasses are used by workers.

Despite clinical improvement, IOFBs continue to be underestimated, and ocular siderosis develops mainly owing to delayed presentation or missed diagnosis. Therefore, we also recommend further education on ocular injuries for ophthalmologists and neurologists in primary hospitals. A comprehensive ophthalmologic examination is mandatory. A CT scan is the imaging tool of choice, and MRI should be avoided in suspected cases of metallic IOFBs.

Conflicts of Interests

None.

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