Case Report

An Unusual Case of Low Vision and Anisocoria Considered a Neurological Finding in the Emergency Department: Ocular Siderosis

Osman Özen, Menekşe İnal Özen

Kastamonu Training and Research Hospital, Clinic of Ophthalmology, Kastamonu, Türkiye

Abstract

We present the case of a patient who came to the emergency department with a significant decrease in vision and dilated pupil in the left eye. Since neurological pathologies were primarily considered, diffusion brain magnetic resonance imaging (MRI) and brain computed tomography (CT) were requested. After the results were reported as normal, we were consulted. On examination, the anterior segment was normal but we detected shiny pearl-like formations in the anterior vitreous, condensation at the inferior of the posterior vitreous, and a scar in the macula. When we evaluated the orbital section of the current brain CT, we detected an intraocular foreign body (IOFB). On the brain MRI, we saw a large artifact that obscured the left orbit and surrounding anatomical structures. When we questioned again, we learned that he had been admitted to another emergency department two months prior due to an object hitting his left eye, where the eye was only washed with saline. Our case emphasizes that ocular siderosis caused by IOFBs should be kept in mind in the differential diagnosis of anisocoria, especially before MRI. Because metallic objects may move during MRI, undiagnosed IOFBs can cause serious ocular side effects.

Keywords: Anisocoria, iron mydriasis, intraocular foreign body, magnetic resonance imaging, ocular siderosis

Cite this article as: Özen O, Özen İ. An Unusual Case of Low Vision and Anisocoria Considered a Neurological Finding in the Emergency Department: Ocular Siderosis. Turk J Ophthalmol. 2024;54:354-357

Address for Correspondence: Osman Özen, Kastamonu Training and Research Hospital, Clinic of Ophthalmology, Kastamonu, Türkiye
E-mail: dr.oozen16@gmail.com **ORCID-ID:** orcid.org/0000-0003-1685-6885
Received: 12.08.2024 **Accepted:** 03.10.2024

DOI: 10.4274/tjo.galenos.2024.77783

Introduction

Eye injuries are among the most common occupational accidents and reasons for emergency department admission. Studies report that patients presenting with eye trauma are 22-25 years of age on average and predominantly male.[1,2] Intraocular foreign bodies (IOFBs) account for 18-41% of all open-globe injuries.[3] IOFBs diagnosed after penetrating eye injuries cause severe intraocular toxic reactions of varying severity depending on their material composition, duration, shape, and size.[4]

In 1890, Bunge described a series of ocular changes that occurred as a result of iron-containing IOFBs, which he called ocular siderosis (OS).[3] OS may occur months or years after trauma and can affect all ocular tissues, from the cornea to the optic nerve. The most common findings are cataract formation, mydriasis, iris heterochromia, secondary glaucoma, iritis, vitreous opacities, diffuse pigment changes in the retinal pigment epithelium, macular edema, and attenuated responses on electroretinography.[5] Radiographs, orbital computed tomography (CT), and ultrasound are considered the gold standard for the early detection of IOFBs in patients presenting with eye injuries.[6] Magnetic resonance imaging (MRI) can also be used to detect non-metallic IOFBs. However, MRI is contraindicated for metallic IOFBs because of their potential to produce motion artifacts and even serious adverse ocular effects.[7]

In this case report, we present a patient with an overlooked IOFB whom we clinically diagnosed as having OS. This case highlights the importance of careful history-taking and a high suspicion of trauma when examining patients employed in industrial workplaces and presenting with visual impairment while working.

Case Report

A 22-year-old man presented to the emergency department with complaints of mild left pupil dilation and a marked

decrease in vision in the left eye that he noticed upon waking in the morning. He had no other complaints, and the results of neurological examinations were normal. In the emergency department, diffusion brain MRI and brain CT examinations were first performed to rule out a neurological cause. However, when imaging was reported as normal, the ophthalmology department was consulted due to the absence of central nervous system pathology.

On ophthalmological examination, there was anisocoria with a larger pupil in the left eye (Figure 1). Direct and indirect light reflexes were normal on the right and weak but present on the left, with no relative afferent pupil defect detected in either eye. Color vision was normal on the right but could not be evaluated on the left because of low vision. Best corrected visual acuity (BCVA) in Snellen decimal units was 1.0 on the right and 0.1 on the left. Intraocular pressure was normal, corneas were clear, and the anterior chamber, iris, and lens appeared normal bilaterally. In the gonioscopic angle examination of the left eye, increased pigment was noticed at the angle. Shiny, pearl-like formations were observed in the anterior vitreous, while the posterior vitreous appeared condensed in the inferior periphery. Color fundus and fundus autofluorescence images (TRC-50DX, Topcon Corporation, Tokyo, Japan) revealed a macular scar in the inferior parafoveal area (Figure 2). Optical coherence tomography (OCT) (Cirrus HD-OCT 5000, Carl Zeiss Meditec AG, Jena, Germany) showed choroidal rupture in the area of the macular scar and irregularity of the retinal outer layers consistent with scarring (Figure 3). In the light of these findings, the images were examined with the suspicion of IOFB. On the orbital part of the brain CT scan, a metallic IOFB was noticed in the left eye (Figure 4). Moreover, brain MRI showed a large artifact obscuring the anatomical structures of the left orbit and surrounding regions (Figure 5). When we questioned the patient again in detail, he reported that two months earlier, he had presented to the emergency department of a different hospital because of a foreign body in his left eye. After examination, the eye had been washed with saline (Polyflex 0.9% isotonic sodium chloride solution, Polifarma Pharmaceuticals, Tekirdağ, Türkiye) and he was told there was no other pathology. We informed the patient of his condition and referred him to a more advanced center, where he underwent lens-sparing vitrectomy and IOFB removal surgery. At postoperative 1 month, the mydriasis had resolved and his BCVA remained unchanged at 0.1.

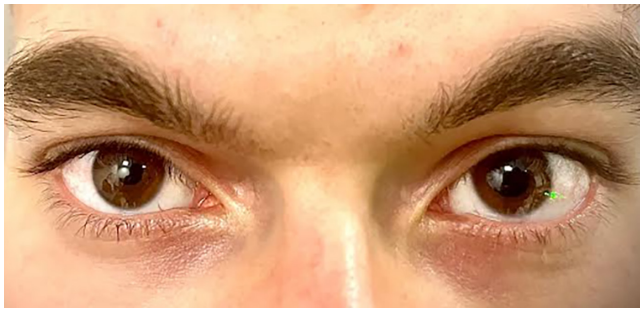

Figure 1. Anisocoria with a larger pupil in the left eye

Discussion

This case of anisocoria accompanied by low vision demonstrates the potential clinical consequences that can arise because of an overlooked IOFB, even months later. There are a limited number of cases in the literature presenting the clinical findings of posttraumatic OS. The clinical manifestations in these cases include heterochromia, mydriasis, cataract, secondary glaucoma, anterior and posterior uveitis, retinal vessel sheathing, and retinal pigmentary changes.[8] In our case, there was no additional finding other than unilateral mydriasis and visual impairment associated with a parafoveal scar.

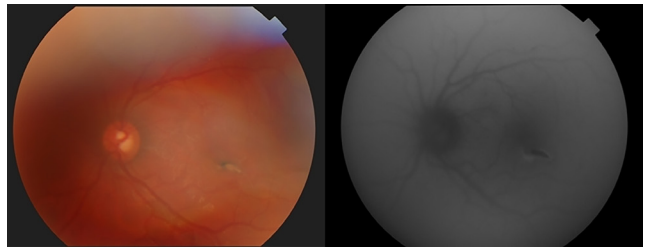

Figure 2. A scar in the lower parafoveal region

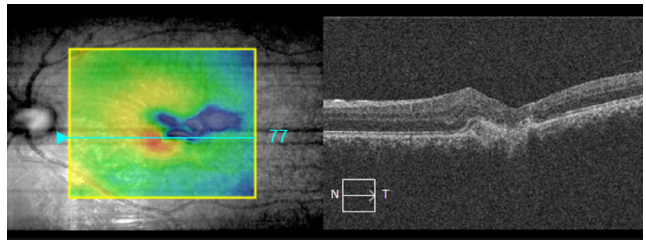

Figure 3. Optical coherence tomography image of the parafoveal scar

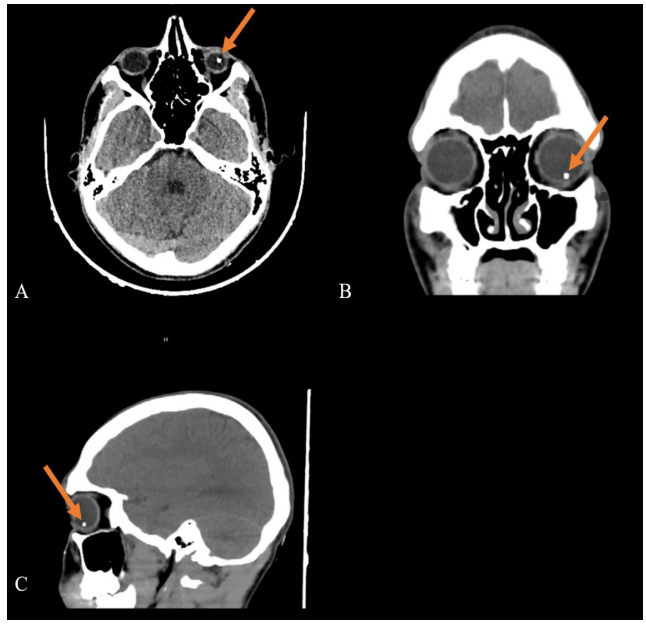

Figure 4. Computed tomography images of the metallic intraocular foreign body in the left eye (orange arrows): A) axial section, B) coronal section, C) sagittal section

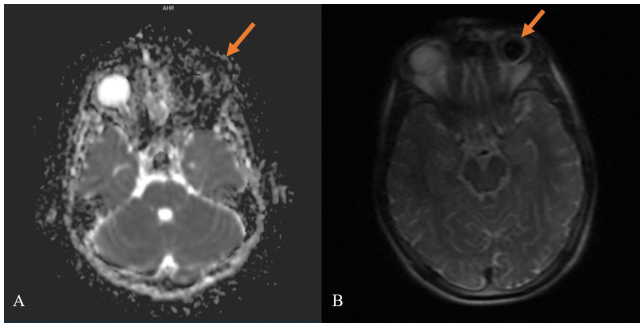

Figure 5. Diffusion magnetic resonance images in the axial plane showing typical magnetic susceptibility artifact (orange arrows) obscuring the anatomical structures of the left orbit and surrounding regions

An enlarged pupil may be the initial sign of OS, as in our case. New-onset anisocoria is an important condition that should always be investigated. Differential diagnoses for anisocoria include Horner syndrome, acute neurological anisocoria, physiological anisocoria, Argyll Robertson pupil, pharmacologically shrunken pupil, and Adie (tonic) pupil.[9,10] The term "iron mydriasis", on the other hand, has been used to characterize cases of mydriasis caused by OS after overlooking an IOFB, which is often not suspected, and should be included in the differential diagnosis.[11,12] Mydriasis is thought to occur as a result of OS-induced parasympathetic neuropathy, and the pupil shows a hypersensitive reaction to diluted pilocarpine, like Adie pupils.[11,13,14]

Various animal studies have shown that ferromagnetic bodies can move considerably during MRI, potentially leading to intraocular complications.[15,16] Gunenc et al.[17] showed that foreign bodies such as iron, chromium, and solder can move 7-10 mm within bovine eyes during MRI. As we did not have a comparative orbital CT scan obtained after MRI, we do not know whether the IOFB moved in our case. However, the fact that we were unable to detect an IOFB visible on CT in the mid-inferior vitreous during our fundus examination suggests that it may have shifted to a far-peripheral location. The large dark area observed on MRI in our case was a magnetic susceptibility artifact. Susceptibility artifacts appear on clinical MRI due to the presence of even a small amount of a metal-containing substance, which can cause signal loss and large geometric distortions of anatomical structures, as in our case.[7]

There are very few case reports in the literature showing MRI of a metallic IOFB in vivo.[16,17,18,19,20] A handful of case reports have documented ocular complications after MRI that were caused by overlooked IOFBs. Kelly et al.[21] first reported sudden unilateral vision loss due to vitreous hemorrhage after a brain MRI in 1986. In a similar case, a patient presented with sudden eye pain and loss of vision immediately after MRI. Ophthalmological examination revealed a small paracentral corneal scar and 50% hyphema.[18] In both cases, subsequent CT revealed the presence of an IOFB. Vote and Simpson[22] reported rapid progression of traumatic cataracts due to IOFB after MRI in 2001, and a more recent case report described a case of microhyphema after MRI resulting from the dislocation of an IOFB embedded in the iris.[23] In contrast, Platt et al.[20] reported that an orbital IOFB was detected in a 12-year-old child undergoing routine brain MRI examination without causing any ocular complications. Similarly, our patient seems to have another rare case of orbital IOFB that was noticed on brain MRI but caused no ocular adverse effects associated with MRI. The eye is considered one of the most vulnerable parts of the body in terms of metallic fragments. Therefore, to avoid surprises such as those seen in the present case or worse, it has been recommended that patients and other personnel be carefully questioned and screened for IOFBs before entering the controlled area of the MRI room.[24]

Despite clinical improvements, IOFBs can still be overlooked, and OS occurs mainly as a result of delayed admission or missed diagnosis. Our report highlights the importance of a detailed ophthalmologic examination prior to MRI in patients with unilateral fixed or poorly reactive dilated pupil, especially young male patients and even in the absence of a definitive trauma history, to prevent delaying or overlooking the diagnosis of OS. Despite having appropriate screening protocols prior to imaging, metallic IOFBs unnoticed before MRI can cause a number of adverse events from monocular blindness to other intraocular complications. Therefore, we believe this case demonstrating that "iron mydriasis" should be kept in mind during the differential diagnosis of anisocoria will be a good reminder for clinicians.

Ethics

Informed Consent: Obtained.

Declarations

Authorship Contributions

Concept: O.Ö., M.İ.Ö., Design: O.Ö., M.İ.Ö., Data Collection or Processing: M.İ.Ö., Analysis or Interpretation: O.Ö., M.İ.Ö., Literature Search: O.Ö., M.İ.Ö., Writing: O.Ö.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

References

1. Zhu L, Shen P, Lu H, Du C, Shen J, Gu Y. Ocular trauma score in siderosis bulbi with retained ıntraocular foreign body. Medicine (Baltimore). 2015;94:e1533.
2. Greven CM, Engelbrecht NE, Slusher MM, Nagy SS. Intraocular foreign bodies: management, prognostic factors, and visual outcomes. Ophthalmology. 2000;107:608-612.
3. Ballantyne JF. Siderosis bulbi. Br J Ophthalmol. 1954;38:727-733.
4. Shu W, Dunaief JL. Potential treatment of retinal diseases with ıron chelators. Pharmaceuticals (Basel). 2018;11:112.
5. Asproudis I, Zafeiropoulos P, Katsanos A, Kolettis C. Severe self-ınflicted acute ocular siderosis caused by an ıron tablet in the conjunctival fornix. Acta Medica (Hradec Kralove). 2018;60:160-162.
6. Steel DH, Rosseinsky DR, James CR. Acute retinal toxicity caused by the bimetallic electrochemical action of a galvanized steel intraocular foreign body. Retina. 1998;18:77-78.
7. Herrick RC, Hayman LA, Taber KH, Diaz-Marchan PJ, Kuo MD. Artifacts and pitfalls in MR imaging of the orbit: a clinical review. Radiographics. 1997;17:707-724.

8. Casini G, Sartini F, Loiudice P, Benini G, Menchini M. Ocular siderosis: a misdiagnosed cause of visual loss due to ferrous intraocular foreign bodies-epidemiology, pathogenesis, clinical signs, imaging and available treatment options. Doc Ophthalmol. 2021;142:133-152.
9. Dowlut MS, Curragh DS, Napier M, Herron B, McIlwaine G, Best R, Chan W. The varied presentations of siderosis from retained intraocular foreign body. Clin Exp Optom. 2019;102:86-88.
10. Monteiro ML, Ulrich RF, Imes RK, Fung WE, Hoyt WF. Iron mydriasis. Am J Ophthalmol. 1984;97:794-796.
11. Hope-Ross M, Mahon GJ, Johnston PB. Ocular siderosis. Eye (Lond). 1993;7:419-425.
12. Monteiro ML, Coppeto JR, Milani JA. Iron mydriasis. Pupillary paresis from occult intraocular foreign body. J Clin Neuroophthalmol. 1993;13:254-257.
13. O'Duffy D, Salmon JF. Siderosis bulbi resulting from an intralenticular foreign body. Am J Ophthalmol. 1999;127:218-219.
14. Keeney AH. Intralenticular foreign bodies. Arch Ophthalmol. 1971;86:499-501.
15. Williams S, Char DH, Dillon WP, Lincoff N, Moseley M. Ferrous intraocular foreign bodies and magnetic resonance imaging. Am J Ophthalmol. 1988;105:398-401.
16. Lagouros PA, Langer BG, Peyman GA, Mafee MF, Spigos DG, Grisolano J. Magnetic resonance imaging and intraocular foreign bodies. Arch Ophthalmol. 1987;105:551-553.
17. Gunenc U, Maden A, Kaynak S, Pirnar T. Magnetic resonance imaging and computed tomography in the detection and localization of intraocular foreign bodies. Doc Ophthalmol. 1992;81:369-378.
18. Ta CN, Bowman RW. Hyphema caused by a metallic intraocular foreign body during magnetic resonance imaging. Am J Ophthalmol. 2000;129:533-534.
19. Zhang Y, Cheng J, Bai J, Ren C, Zhang Y, Gao X, Cui X, Yang YJ. Tiny ferromagnetic intraocular foreign bodies detected by magnetic resonance imaging: a report of two cases. J Magn Reson Imaging. 2009;29:704-707.
20. Platt AS, Wajda BG, Ingram AD, Wei XC, Ells AL. Metallic intraocular foreign body as detected by magnetic resonance imaging without complications- a case report. Am J Ophthalmol Case Rep. 2017;7:76-79.
21. Kelly WM, Paglen PG, Pearson JA, San Diego AG, Soloman MA. Ferromagnetism of intraocular foreign body causes unilateral blindness after MR study. AJNR Am J Neuroradiol. 1986;7:243-245.
22. Vote BJ, Simpson AJ. X-ray turns a blind eye to ferrous metal. Clin Exp Ophthalmol. 2001;29:262-264.
23. Lawrence DA, Lipman AT, Gupta SK, Nacey NC. Undetected intraocular metallic foreign body causing hyphema in a patient undergoing MRI: a rare occurrence demonstrating the limitations of pre-MRI safety screening. Magn Reson Imaging. 2015;33:358-361.
24. Bailey W, Robinson L. Screening for intra-orbital metallic foreign bodies prior to MRI: review of the evidence. Radiography (Lond). 2007;13:72-80.