International Journal Dental and Medical Sciences Research
Volume 6, Issue 1, Jan-Feb- 2024 pp 294-296 www.ijdmsrjournal.com ISSN: 2582-6018

Imaging of post traumatic optic nerve avulsion in a pediatric patient: A rare case with review of literature

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Date of Submission: 20-01-2024 Date of Acceptance: 30-01-2024

ABSTRACT: A case of partial optic nerve avulsion in a 7 year old female who had a blunt trauma to left eye due to injury by the handle bar of a bicycle . Patient presented with sudden loss of vision in left eye.On clinical examination, posterior segment hemorrhage was present with obscuration of optic nerve on fundoscopy.But imaging was necessary for the diagnosis . After performing B-scan and magnetic resonance imaging , diagnosis was made.

I. INTRODUCTION

Optic nerve avulsion falls under the spectrum of traumatic optic neuropathy which results in sudden and complete loss of vision in majority of its cases after the trauma(1). It is usually caused by a blunt trauma of significant force causing a decelerating injury to the globe causing sudden rotation of the globe within the orbit(2). It is rare cause of permanent vision loss(3). Typically in cases of traumatic eye injury the incidence of neuropathy is 0.5-5% with optic nerve avulsion even less.(1).

The diagnosis is often made clinically by presence of excavated optic nerve apperance on fundoscopy(3). It is seldom difficult to confirm the presence of optic nerve head avulsion clinically, as vitreous hemorrhage in the posterior segment precludes the nerve head(4). Hence imaging is crucial in such cases to confirm the diagnosis(5).We report a case of 7year female child of blunt trauma to left eye resulting intoavulsion of optic nerve with permanent loss of vision in the affected eye.

II. CASE REPORT

A 7 year femalechild wasbroughtto ophthalmology out patient department with sudden vision loss in left eye after traumato the left eyeone day back by the handle bar of the bicycle while riding the bicycle. The vision in the left eye was only perception of light and in the right eye was 6/6. A relative afferent pupillary defect was present in left eye.Slit lamp test showed chemosis, anhydrosis, linear 4 mm conjuctival tear in the inferior bulbar conjunctiva witheyelid edema. However, cornea , lens and anterior chamber were normal.

On a 20D fundus examination , vitreous hemorrhage was noted which was arising from the disc with optic nerve head relatively obscured by the hemorrhage, also called the “pit sign”. The retina was pale with attenuated vessels around the disc . hemorrhage around the disc was also noted(Figure 1). However no retinal tear was demonstrated. Clinical and fundoscopic findings were in favour of optic nerve avulsion . Patient was immediately referred for B- scan . B-scan with a high frequency linear array transducer revealed a organised vitreous hemorrhage with origin from the head of optic nerve causing altered ehogenicity(Figure 2). The retinal step sign was also present (Figure 3).

Magnetic resonance imaging (MRI) was also done to know the extent of injury and adjacent vital tissue damage. The diagnosis was cemented by the disruption of the area of lamina cribrosa , thickening of the nerve diameter with obliteration of the dural space with hypointensity in the nerve (Figure 4)and posterior segment hypointensities originating from the area of optic nerve head consistent with optic nerve head avulsion and vitreous hemorrhage (Figure 5) .Since, only clinical examination was suboptimal due to non visualisation of optic nerve head , imaging was crucial in reachngthe diagnosis . After confirmation on MRI, patient was avoided from the highly aggresive megadose corticosteroid treatment and put on conservative managemnet,as assigned in a case of irreversible optic nerve damage .

III. DISCUSSION:

Optic nerve, the second cranial nerve is created from retinal ganglion cell axons. The meningeal liningssurround the optic nerve as it travels from sclera of the eyeball to orbit. The nerve further travels via the optic chiasma, and ultimately ends in the occipital lobes. The nerve has intracranial as well as extra cranial components, with, the extracranial part further dividing into intraorbital and intraocular component(6). The nerve is most susceptible to injury by blunt trauma where it enters the optic

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canal(7).Ocular trauma seldom results in optic nerve avulsion. The force of the trauma disinserts optic nerve from the retina, choroid and the lamina cribrosa. This may happen due to head injury with or without fracture(8). The optic nerve and lamina cribrosa move backward inside the covering dura if the avulsion is complete(7). In optic nerve avulsion, there is retraction of lamina cribrosa from scleral rim. This has been described in both complete and incomplete avulsion (4). When an incomplete or partial avulsion occurs, only a portion of the nerve's dural covering is removed, leaving the remaining portion of the nerve in situ(9).In complete evulsion the nerve and the dura, both are completely disinserted and pulled away from the globe whereas in partial avulsion only the nerve fibers are sheared and retracted and dural sheath remains in place(8).Instantaneous forward acceleration of the head could detach the globe from the optic nerve due to excessive stretching (7).Displacement of the optic nerve could also occur due to sudden increase in IOP which pushes back the nerve away from the globe or sudden rotation of the globe could shear off the fibers of the optic nerve (1). A projectile could also cause injury to the optic nerve(7).In an early setting , within 24 hours , the clinical presentation is more diagnostic as the posterior segment is not completely occluded due to the hemorrhage and better visualization of the optic nerve head is possible. However , after 24 hours vitreous hemorrhage masks the nerve head and it becomes difficult to determine the status of optic nerve(1).In such circumstances , as the case in our patient ,imaging becomes mandatory to confirm diagnosis.On B-scan ultrasound examination, the important findings which suggests optic nerve avulsion are vitreous hemorrhage originating from the area of optic canal , retinal step sign , obscured optic nerve , retinal detachment or a clear posterior defect (1). Our case demonstrated three of the above criteria, i.e the vitreous hemorrhage originating from the area of the disc , retinal step sign and nerve obscuration.

On MRI imaging disruption in the area of the lamina cribrosa , posterior chamber hypointensity arising from the area of head of optic nerve and obliteration of the dural space by blood around the nerve fibers was present, also fulfilling the pathognomic criterion (1).Other imaging modalities, like computed tomographycan help in delineating orbital fractures.Fluorescein angiography which shows the perfusion of retinal vessels (7).

In emergency, B- scan examination is most valuable, as it is readily available, and less time consuming and offers real time examination of the posterior segment(1). No treatment, whether medical or surgical has been fully effectivein saving vision in case of optic nerve avulsion, in eithe partial or complete nerve avulsion.(1) The use of megadose methyprednisolone (>5,400 mg/d), is still controversial in post-traumatic optic neuropathy . According to the International Study on Optic Nerve Trauma (1999), it is postulated that treating traumatic optic neuropathy with high-dose corticosteroids may even be deleterius(10). Imaging will guideto unjust use of corticosteroids or unnecessary surgical intervention if definitive diagnosis of avulsion of optic nerve is confirmed , especially in pediatric cases (1).

IV. CONCLUSION

Avulsion of optic nerve is rare but, serious cause of permanent vision loss in blunt eye trauma. The clinician must be vigilant in such instances where fundoscopic examination is equivocal . Such cases needproper clinical as well as multimodality imaging approach to avoid unwarranted surgical and steroid therapy.

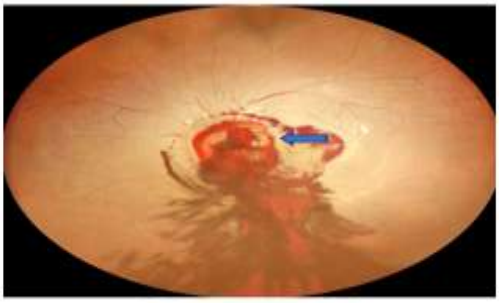

Figure 1. Fundoscopy showing hemorrhage in the vitreous hemorrhage,retinal pallor and hemorrgae around optic disc with obscuration of optic nerve head(blue arrow).

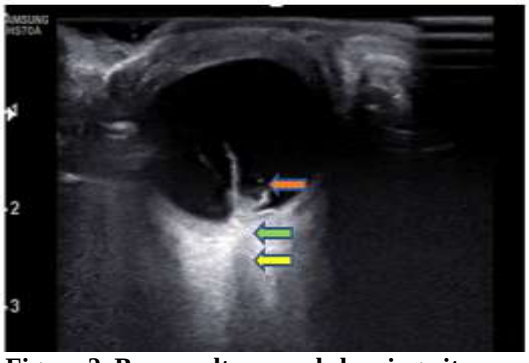

Figure 2. B-scan ultrasound showing vitreous hemorrhage (red arrow), obscuration of optic nerve head (green arrow)and altered echogenicity posterior to optic nerve- globe junction (yellow arrow).

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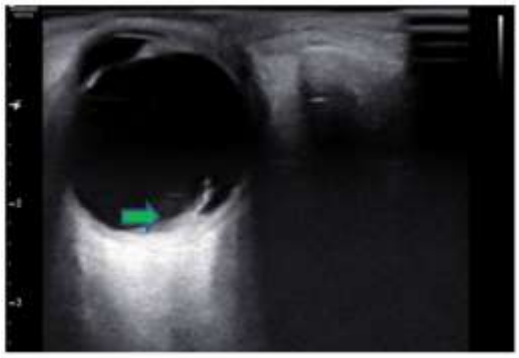

Figure 3.B-scan ultrasound showing retinal step sign (green arrow).

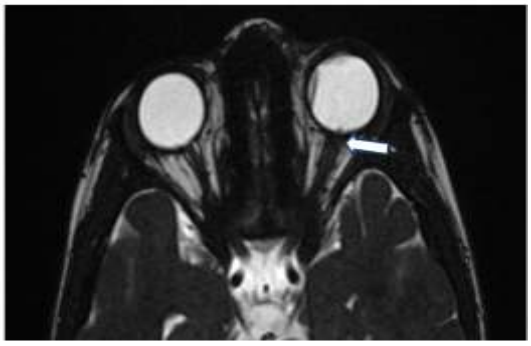

Figure 4. Axial T2-weighted MRIorbit showingwidened, irregular optic nerve-globe junction with disruption in the region of lamina cribrosa and obliteration of perineural space in the left eye(white arrow).

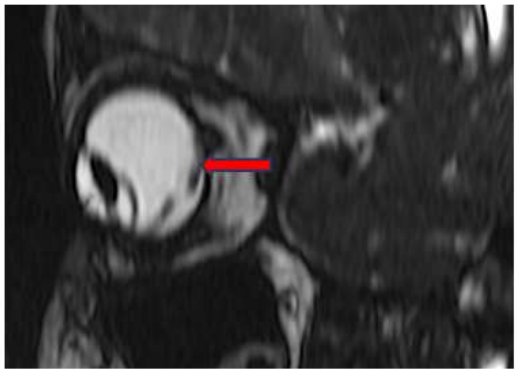

Figure 5.Sagittal T2-weighted MRI of left orbit showing posterior segmenthypointensity origination from the region of lamina cribrosa representing vitreous hemorrhage (red arrow).

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