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Case Report

Traumatic optic neuropathy with concealed CRAO following blunt trauma in a young male patient: A rare case report

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ABSTRACT

Traumatic Optic Neuropathy (TON) is a condition which results from vulnerable nature of the optic nerve to direct and indirect trauma leading to functional impairment of vision.[1] Several varieties of direct optic nerve injury may be identified: optic nerve avulsion, transection, optic nerve sheath haemorrhage, orbital haemorrhage, and orbital emphysema. Central retinal artery occlusion (CRAO) is the sudden blockage of the central retinal artery, resulting in retinal hypoperfusion, leading to rapidly progressive cellular damage, and vision loss. An embolism is the most common cause of CRAO. The degree of collateralization and the duration of retinal ischemia determines the retinal survival.[2] There are only few reported cases of central retinal artery occlusion caused by blunt trauma·We report a case of traumatic optic neuropathy presenting along with central retinal artery occlusion following a high speed blunt trauma to eye in an otherwise healthy young male.

1. Introduction

Trauma to head and face which is often a result of road traffic accidents or falls is the most common cause of visual impairment and blindness.[3,4] Direct injury to the optic nerve should be distinguished from indirect injury. Penetrating trauma and orbital fractures associated with mid-facial fractures are responsible for direct injury to optic nerve. Indirect optic nerve injury caused by transmitted force of impact in head injury is more common.[5] Irreversible loss of vision could occur following blunt trauma due to optic neuropathy or rarely due to vascular occlusion.[6] Inner layers of the retina have been found to be thinned out in patients with central retinal artery occlusion (CRAO). Proteolysis mediated by calpain, a calcium activated protease has been postulated to be one of the factor for this thinning. This theory is supported by a model experiment demonstrating decreased proteolysis using Calpain inhibitor SNJ-1945.[7]

2. Case Report

A 22-year-old male presented with severe diminution of vision in his right eye following a high-speed blunt trauma with a metallic object 4 weeks back. He was working on a lock making machine when his right eye got hit by small steel particle coming at high speed from the assembly line. As told by the patient himself, it impacted the white of the right eye which was followed by pain, redness and sudden diminution of vision. The pain and redness subsided over days. The patient gave history of improvement in vision over the next 2 weeks, but then he noticed sudden, severe and painless diminution of vision in his right eye.

The visual acuity of right eye at presentation was no perception of light. Intraocular pressure was normal, ocular motility was full in all gazes. On slit lamp examination, there was no abnormality seen in lids, conjunctiva and

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cornea. The pupil of right eye was fully dilated and non-reactive but there was no sign of sphincter pupillae tear. There was early posterior subcapsular cataract seen in right eye. Vitreous degeneration with floating clump of vitreous exudate was seen. (Figure 1 a)

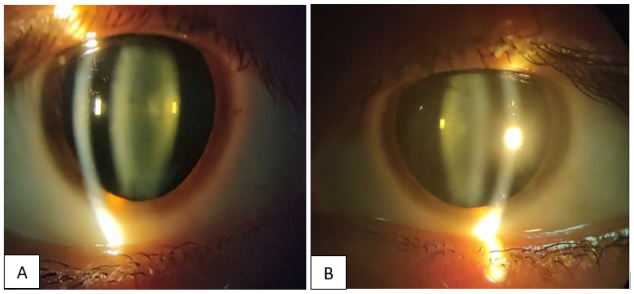


Fig. 1:

Dilated fundus examination revealed pale optic disc with obscured margins and gliosis around it. Peripapillary retinal folds were seen which extend up to macula. Diffuse severe arteriolar attenuation and cattle tracking of the vessels was present (Figure 2 a). Ophthalmological examination of the left eye was unremarkable. (Figure 1 b, Figure 2 b)

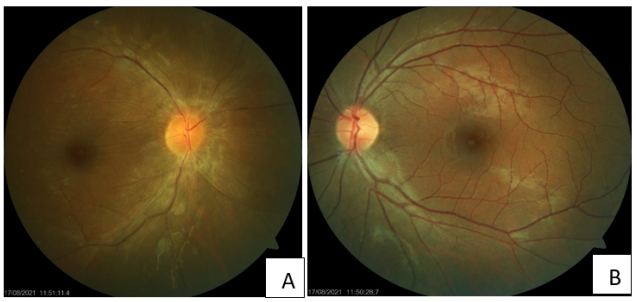


Fig. 2:

Another interesting finding in this case was a presence of chorioretinal atrophic patch temporal to macular with retinal pigment epithelial hypertrophy and a band of vitreous lying over it. (Figure 3 a, b)

This can be due to traumatic choroidal rupture with RPE hyperplasia over it and a band of resolving vitreous haemorrhage attached to it.

On fluorescein angiography, though the arteriolar perfusion was present in right eye, marked generalised arteriolar attenuation could be seen as compared to left eye which strongly points towards acute transient non-arteritic central retinal artery occlusion caused due to trauma. The foveal avascular zone was also increased in size (Figure 4 a). The fluorescein angiography of left eye was within normal limits. (Figure 4 b)

On ocular coherence tomography (OCT) examination, there was severe loss of ganglion cells and macular atrophy

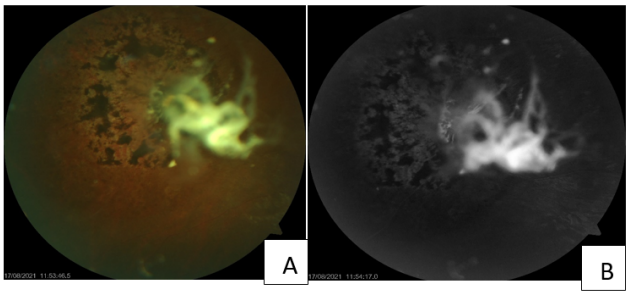


Fig. 3:

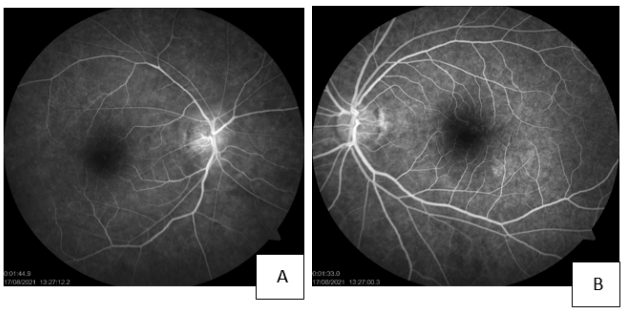


Fig. 4:

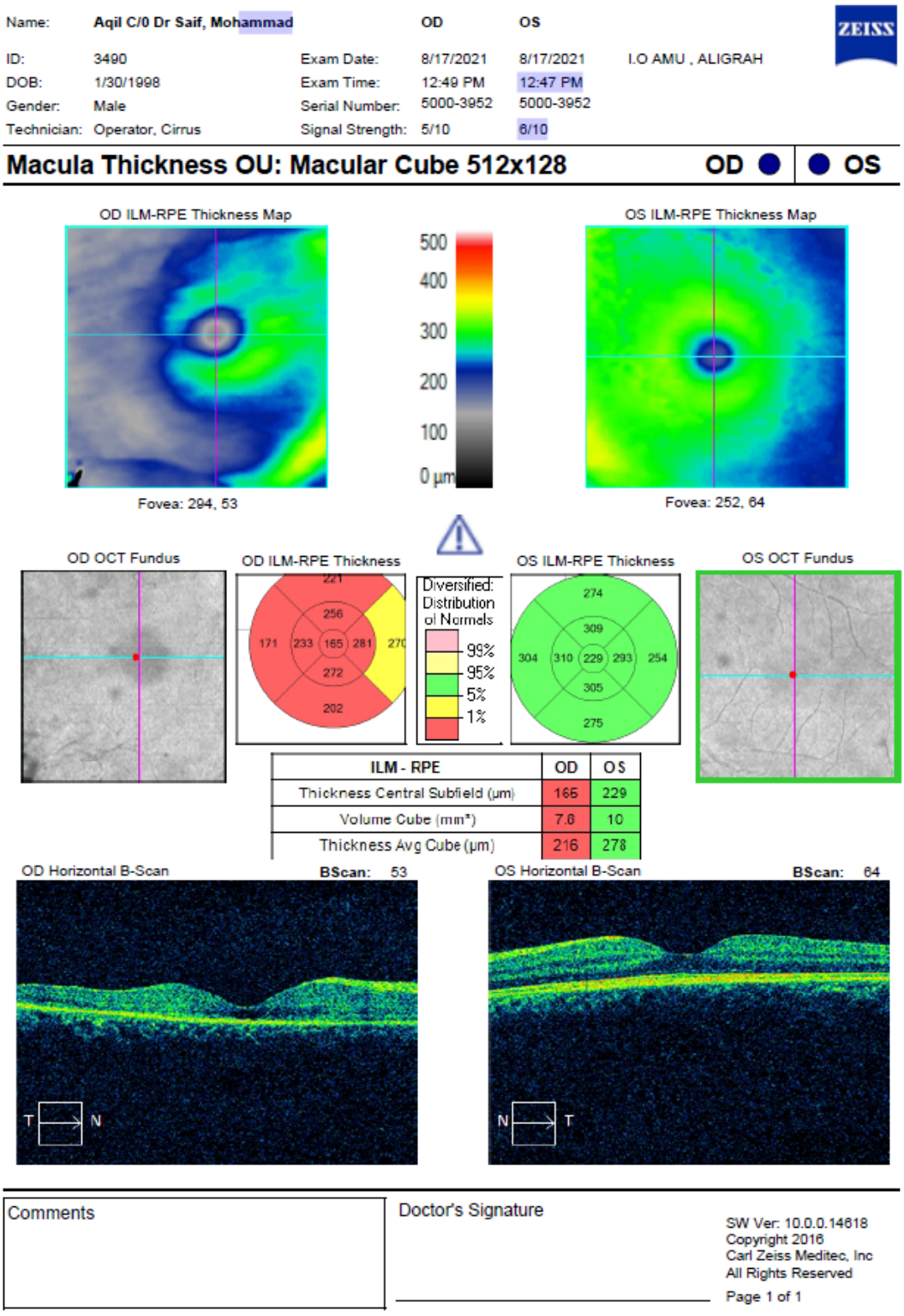
in right eye pointing towards damage due to acute ischemia during the episode of CRAO (Figures 5 and 6).

3. Discussion

In the previous literature, ocular trauma has rarely been reported as a solitary causative factor for CRAO. Inner layers of retina are supplied by central retinal artery and are effected by ischemia in an event of CRAO. Axonal retinal nerve fibre layer (RNFL), the somatic ganglion cell layer (GCL), inner plexiform layer (INPL), and the inner nuclear layer (INL) constitute the inner retina from inside out. The long RNFL fibres converge as the optic nerve.[8] CRAO most occur in elderly and is associated with hypertension and dyslipidemia. With exception of coagulopathies, the presentation in young is rare.[9]

In our case, the exact cause of CRAO could not be discovered. The mechanism for CRAO could be postulated as multifactorial, being initiated by mechanical compression from high-speed blunt trauma causing focal vasospasm and endothelial damage due to sudden compression of the globe,[10] compartment syndrome causing optic nerve head ischaemia.[11] Swollen optic nerve compressing on the central retinal artery could have further accentuated it.[12]

The marked decrease in GCL layer thickness detected on OCT is a marker of vascular compromise to macula in setting of trauma and it can be used to retrospectively detect vascular event even in cases where angiography is normal.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DOB: 1/30/1998
Exam Time: 12:49 PM 12:47 PM
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Technician: Operator, Cirrus
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500
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OS ILM-RPE Thickness Map
Fovea: 294, 53
Fovea: 252, 64
OD OCT Fundus
OD ILM-RPE Thickness
221
256
171
233
165
281
272
202
Diversified Distribution of Normals
99%
95%
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274
309
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293
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Thickness Central Subfield (µm)
165
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Volume Cube (mm³)
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Thickness Avg Cube (µm)
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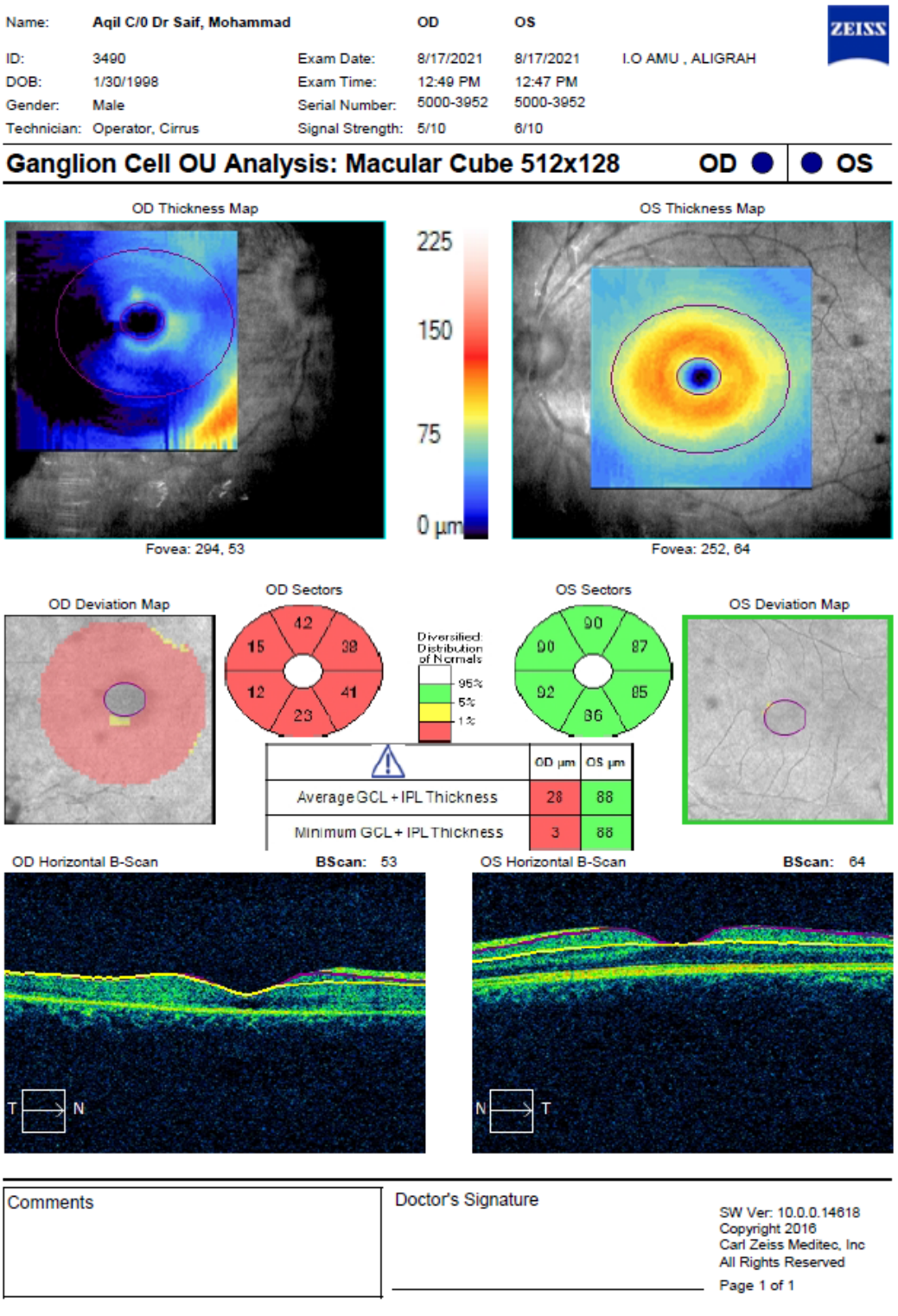
Fig. 5: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Gender: Male
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Signal Strength: 5/10 6/10
Ganglion Cell OU Analysis: Macular Cube 512x128 OD OS
OD Thickness Map
225
150
75
0 µm
OS Thickness Map
Fovea: 294, 53
Fovea: 252, 64
OD Deviation Map
OD Sectors
42
15
38
12
41
23
Diversified Distribution of Normals
95%
5%
1%
OS Sectors
90
90
87
92
85
86
OS Deviation Map
OD µm
OS µm
Average GCL + IPL Thickness
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Minimum GCL + IPL Thickness
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86
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Fig. 6:

4. Conclusion

Blunt trauma causing retinal vascular occlusion is quite rare. Along with commotio retinae and optic nerve injury, the possibility of vascular occlusion following blunt trauma should be kept in mind. Careful observation and patient education is important to prevent irreversible visual loss following trauma. OCT with optic nerve head and nerve fibre layer analysis can aid in the diagnosis, grading and monitoring of traumatic optic neuropathy. Ganglion cell layer thickness analysis appears to be a valuable tool to diagnose and monitor the effect of vascular compromise to retina due to trauma. It can be also used to retrospectively document damage to macula caused by vascular compromise even when recirculation has been established and angiography shows patent arterioles.

5. Declaration of Patient Consent

The author certify that they have obtained all appropriate patient consent. The patient has given consent for his images and other clinical information to be reported in the journal. They understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

6. Source of Funding

Nil.

7. Conflicts of Interest

There are no conflicts of interest.

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