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Case Report

Traumatic Central Retinal Artery Occlusion: Case Report and Review of the Literature

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Abstract

Central retinal artery occlusion (CRAO) is an uncommon entity in ophthalmic practice. Reported etiologies are numerous, and involve systemic and ocular preexisting pathologies. A traumatic etiology for CRAO is rare with anecdotal reports and poor visual outcomes. No specific treatment guidelines exist to date. We report a case of traumatic CRAO in a young female without any systemic vascular risk factors; it is clinical course and management. Physician awareness to this rare condition is important for prompt diagnosis and treatment while the retinal tissue is viable and visual restoration is possible.

Keywords: Central Retinal Artery Occlusion; Trauma; Paracentesis

Abbreviations

CRAO	: Central Retinal Artery Occlusion
IOP	: Intraocular Pressure
RAPD	: Relative Afferent Pupillary Defect

Introduction

Central retinal artery occlusion (CRAO) was first described by Von Graefe in 1859 [1]. Although various etiologies for CRAO have been reported in the past, ocular trauma as a causative factor has rarely been described [1–7]. CRAO remains a therapeutic challenge in modern medicine, and the catastrophic visual consequences warrant prompt diagnosis and treatment in an attempt to salvage viable retinal tissue. We describe a case of CRAO that occurred after a relatively minor blunt ocular trauma, it's clinical presentation and visual implications.

Case Report

A 34-year-old female presented to the emergency department two days after a sudden decrease in vision in her right eye following a hand strike to it. Her past medical and ocular history was unremarkable.

Ophthalmologic examination of the right eye was positive for relative afferent pupillary defect (RAPD). Visual acuity was finger counting temporally and light perception nasally; intraocular pressure (IOP) measured 10 mmHg. The anterior chamber

and vitreous were clear. There was marked retinal pallor between the vascular arcades and a foveal cherry-red spot [Figure 1a]. Findings of left eye examination were within normal limits. A diagnosis of right CRAO was made.

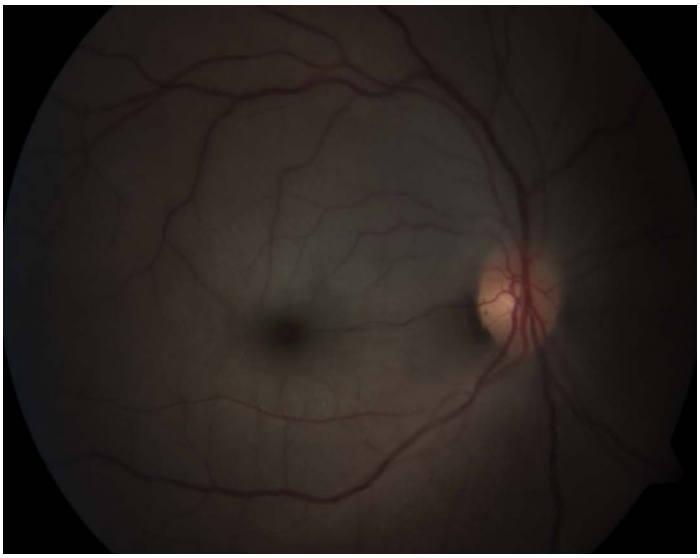

Figure 1a. Right eye. Color funduscopy demonstrating marked retinal pallor between the vascular arcades and a foveal cherry red spot.

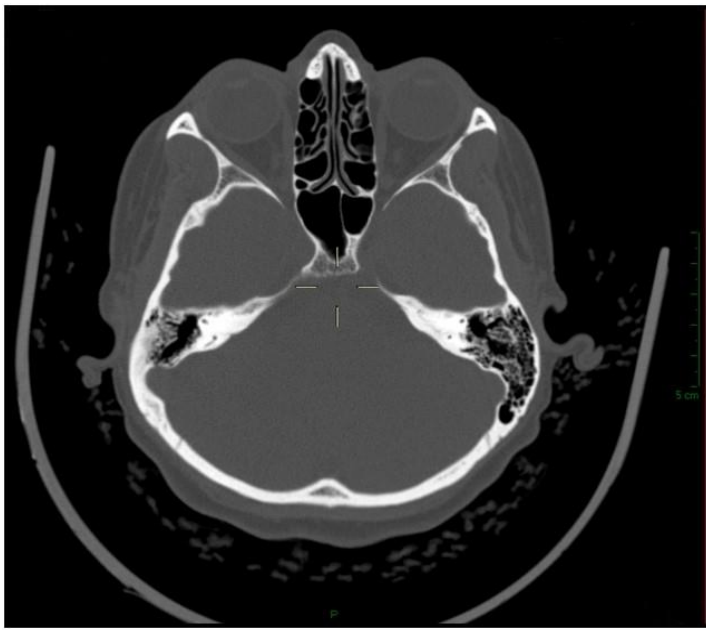

Figure 1b. Computerized tomography scan demonstrating intact orbital bony structure and no orbital hematoma.

The immediate loss of vision following the hand strike strongly suggested trauma as the causative factor. Coagulation disorders were assessed and ruled out as possible etiologies. Due to the ocular trauma reported on anamnesis, the patient was referred for orbital directed computerized tomography scan. No orbital fractures or hematomas were found [Figure 1b]. Optical coherence tomography demonstrated ischemic retinal edema in the inner retina secondary to the occlusion [Figure 1c] while intravenous fluorescein angiography demonstrated no delay in arterial filling with normal retinal perfusion [Figure 2a-c].

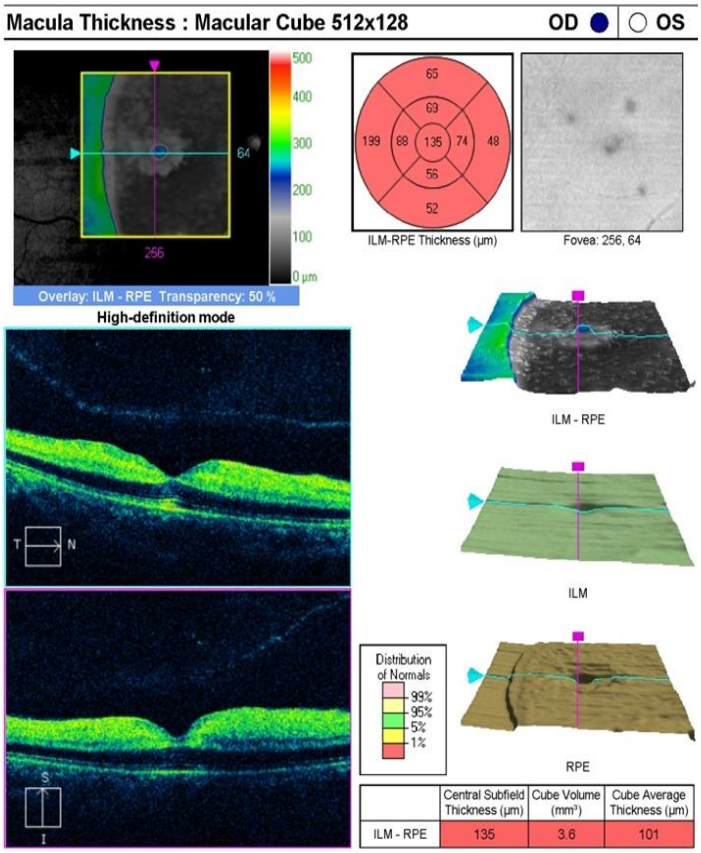

Figure 1c. Right eye optical coherence tomography scans demonstrating ischemic retinal edema in the inner retina.

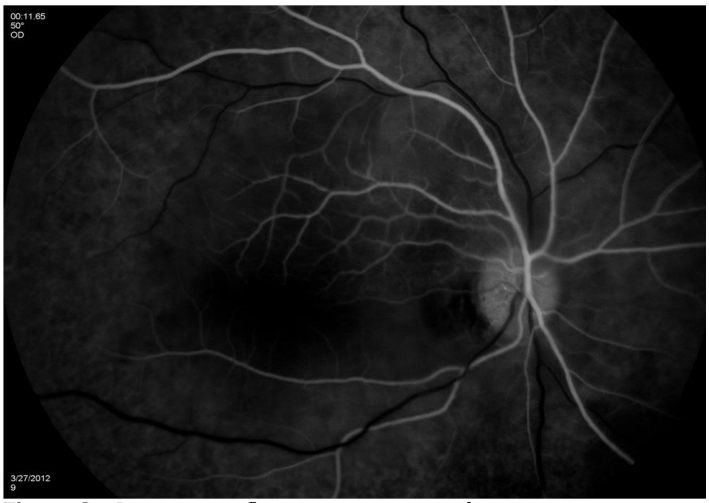

Figure 2a. Intravenous fluorescein angiography.
Right eye. Arterial phase, and beginning of venous laminar flow, demonstrating no arterial delay.

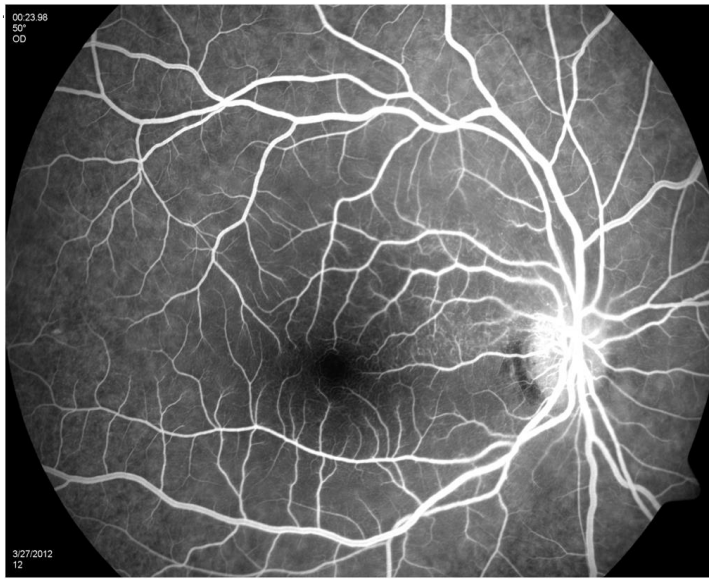

Figure 2b. Right eye. Arteriovenous phase demonstrating intact retinal perfusion.

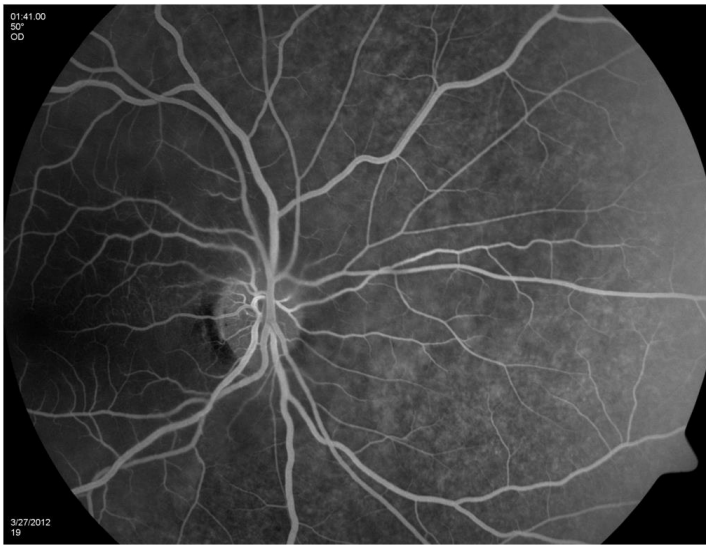

Figure 2c. Right eye. Late phase demonstraing intact retinal perfusion and normal periphery.

Although no treatment modality has been proven effective in such circumstances in the literature, given the patient's young age and extremely poor visual acuity, paracentesis was performed to lower the intraocular pressure, and systemic steroids were administered to reduce the marked retinal edema. Despite these measures no improvement in visual acuity was evident on follow up.

Discussion

The first report of CRAO by Von Graefe in 1859, described a vascular obstruction which was attributed to an embolic event associated with endocarditis [1]. A rare condition estimated at 1 in 10,000 outpatient visits [1], CRAO has been associated with a thrombus formation at or proximal to the lamina cribrosa, impending retinal perfusion. Although CRAO is also rare in children and young adults, several associations have been reported including migraine with presumed vasospastic mechanisms, coagulation abnormalities, hypertension, significantly elevated IOP, cardiac disorders, sickle cell disease, connective tissue diseases, leukemia and temporal arteritis [2,7]. Our patient had none of the predisposing risk factors for vascular occlusion. Ocular vascular obstruction as a result of trauma, especially CRAO as seen in our case, has rarely been reported [1–7].

The proposed pathophysiology of traumatic CRAO is controversial. Several authors suggested that a sudden, trauma-induced mechanical deformation of the eye leads to acute stretching of the retinal vessels and damage to the endothelium and intima of the blood vessel wall. This may expose subintimal tissue to the bloodstream, induce platelet aggregation and initiate a coagulation cascade and thrombus formation [6,7]. Local vasospasm, which is known to occur in trauma, may exacerbate the occlusion [5,7]. We assume both mechanisms contributed to the artery occlusion seen in our case.

CRAO typically presents with painless sudden persistent visual loss which may occur over hours and deteriorate over weeks [1,6]. A history of amaurosis fugax is sometimes noted. Vision usually diminishes to counting fingers to light perception, accompanied by a positive RAPD. The anterior segment appears normal, excluding traumatic injuries, and the fundi may appear normal on initial presentation. A whitening of the retina due to tissue hypoxia follows, with a characteristic cherry-red spot noted in 90% of cases [1]. Box carting of retinal vessels may also appear and splinter hemorrhages are common. Extensive hemorrhages are rare unless a central retinal vein occlusion is also present. Fluorescein angiography typically shows a slow in arteriovenous transit time, with delayed retinal arterial filling and arterial narrowing or obstruction, while choroidal filling is normal [1]. Within 4-6 weeks the retinal pallor resolves, and optic disc atrophy and pallor develop with macular retinal pigment epithelium changes.

Blunt ocular trauma may also result in commotio retina, choroidal ruptures, macular holes, retinal detachments, retinal and vitreous hemorrhages, and retinitis sclopetaria [7]. It is noteworthy that instant vision loss following trauma may be related to optic nerve damage rather than vascular occlusion.
Numerous management modalities have been proposed for the treatment of CRAO, yet effective treatment has not been

proven and there are no established guidelines. If a patient presents within 90-120 minutes of vascular occlusion, attempts are usually made to lower IOP and dislodge the suspected occluding emboli or thrombus to a more peripheral arterial branch. This may be achieved by ocular massage, anterior chamber paracentesis or pharmacologic interventions with acetazolamide and topical beta blockers [1,8]. Attempts have also been made to administer urokinase or total plasminogen activator at the site of occlusion via catheterization of the ophthalmic artery [9–13]. Systemic anticoagulation, systemic venous thrombolysis, and hyperbaric oxygen treatments have also been described [8,14]. Despite attempts at treatment, prognosis of CRAO is usually poor, and most patients do not regain useful vision [1,15-16].

We presented a case of traumatic CRAO, a rare entity in clinical practice. Our patient presented late after the traumatic insult, and treatment attempt did not improve the visual outcome. Clinicians should be alert to this uncommon condition in patients who present with visual deterioration after blunt ocular trauma. Although debates exits regarding the effectiveness of treatment, initial attempts to improve visual prognosis are often made.

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