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Case Report

Macular infarction and traumatic optic neuropathy following blunt ocular trauma

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Abstract

Macular infarction is a visually disabling condition caused by a variety of reasons. It has rarely been described in association with blunt ocular trauma. We describe the case of a young healthy male who sustained injury with a bull's leg and presented with severe visual loss owing to macular infarction and traumatic optic neuropathy. This report of an angiographically documented macular infarct secondary to ocular contusion highlights an additional feature in the spectrum of ocular findings following blunt trauma that might lead to a severe and permanent affliction of vision.

Keywords: Macular infarction, Blunt trauma, Traumatic optic neuropathy

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Introduction

Blunt ocular trauma can result in a myriad of findings including commotion retinae, choroidal ruptures, macular hole, retinal detachment, retinal and vitreous hemorrhage.[1] Isolated cases of retinal vascular occlusions in otherwise healthy patients have been reported in association with ocular contusion.[2–6] We describe an unusual case of macular infarction and traumatic optic neuropathy (TON) following blunt ocular trauma that resulted in severe, irreversible visual loss in a young male.

Case report

A 15-year-old boy presented with loss of vision in his left eye following trauma with a bull's leg 2 days back. The right eye had an unaided visual acuity of 20/20. He denied perception of light in the left eye. There was mild periorbital bruising in the left eyelids. Ocular movements were full and free. The right pupil reacted directly, but not consensually to light. Anterior segment examination revealed anterior chamber flare and traumatic mydriasis in the left eye. Goldmann applanation tonometry measured the intraocular pressure as 16 mmHg in both eyes. Fundus examination of the left eye showed clear media, mild pallor of the optic disk and subretinal hemorrhage inferior to the disk. There was retinal whitening at the posterior pole with few intraretinal hemorrhages and a cherry red spot (Fig. 1a). The peripheral fundus was unremarkable. The right fundus showed no abnormality.

Fluorescein angiography of the right eye demonstrated an area of hypofluorescence at the macula with occlusion of the surrounding small retinal arterioles and venules suggesting nonperfusion. Staining of the optic nerve head was noted in the late phase. In addition, blocked fluorescence was observed in the region corresponding to the subretinal hemorrhage (Fig. 1b and c). Spectral domain optical coherence tomography (SD-OCT) of the macula revealed inner retinal hyperreflectivity indicating ischemia (Fig. 1d).

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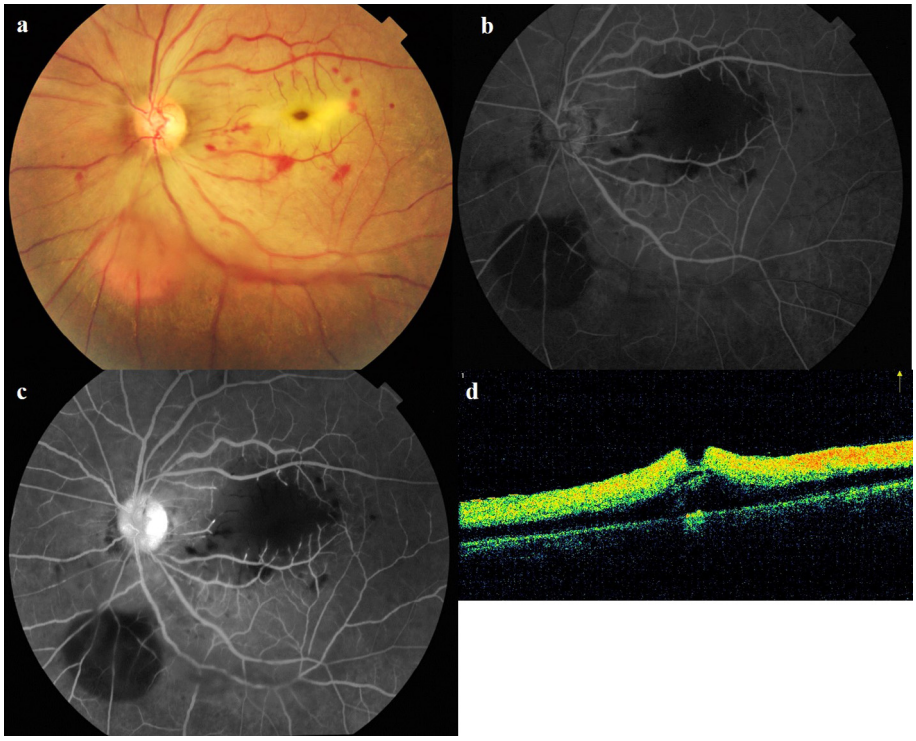

Figure 1. (a) Fundus photograph of the left eye at presentation showing retinal whitening at the posterior pole with retinal hemorrhages and a cherry red spot. There was an area of subretinal hemorrhage inferior to the disk. (b) Fluorescein angiography demonstrated widening of the foveal avascular zone and abrupt termination of the pre-capillary arterioles, indicating macular infarction. The area below the optic disk showed blocked fluorescence due to the hemorrhage. (c) Late phase of the angiogram showed staining at the optic nerve head, in addition to nonperfusion of the macula. (d) Spectral-domain optical coherence tomography scan through the left macula revealed hyperreflectivity of the inner retinal layers indicating acute ischemia.

There was no clinical or laboratory evidence suggestive of systemic lupus erythematosus (SLE) or sickle cell hemoglobinopathy in the patient or his family. Computed tomography did not reveal any fracture or abnormality of the optic nerve. The patient received intravenous bolus therapy with methylprednisolone for 72 h followed by oral prednisone. At one week follow-up, there was a decrease in the retinal whitening and hemorrhages at the macula (Fig. 2a). However the patient failed to gain any vision in this eye. Pallor of the left optic disk and foveal atrophy was observed at one month (Fig. 2b) with macular thinning on SD-OCT (Fig. 2c).

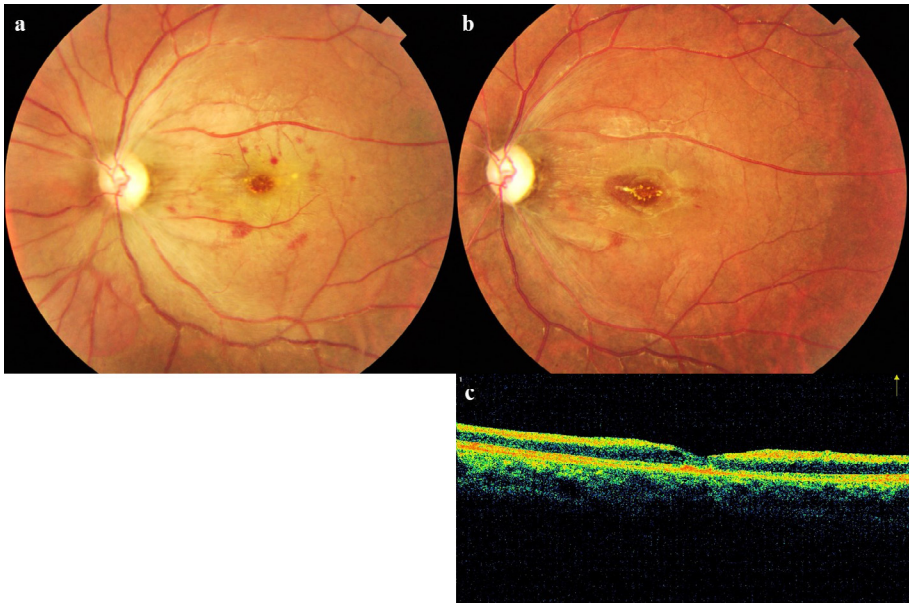

Figure 2. (a) Fundus photograph of the left eye at one week follow-up showing decreased retinal whitening and hemorrhages, with foveal thinning. (b) At one month, optic disk pallor was noted with foveal atrophy. (c) Spectral-domain optical coherence tomography scan demonstrated macular thinning.

Discussion

The macula is predominantly supplied by long, thin lumened end arterioles and capillaries with absent collateral circulation. Thus it is highly predisposed to vaso-occlusions in thrombotic disorders and ischemia in cases of macular hypoperfusion. Gass first described various disorders resulting in macular ischemia including retinal artery and vein occlusions, carotid artery occlusion, malignant hypertension, diabetes, radiation retinopathy and retinal telangiectasis.[7] Additional causes of macular infarction are Behcet disease, sarcoidosis,[8] sickle cell disease,[9] SLE[10] and aminoglycoside toxicity.[11] Macular infarction in the setting of blunt ocular trauma has been described rarely. To the best of our knowledge, only 2 cases have been reported previously.[2]

Central retinal artery occlusion (CRAO) has been reported following trauma, with or without concomitant systemic disorders such as hemoglobinopathies and coagulation abnormalities. The possible mechanisms include compression of the central retinal artery by a hematoma or raised intraorbital pressure due to swelling of orbital soft tissue.[4] Damage to the endothelial cells of the artery with exposure of the underlying collagen in the subintimal tissue stimulates platelet aggregation and thrombus formation. In the absence of evidence suggesting compression of the artery, reflex vasospasm of the injured vessel facilitating arterial thrombotic occlusion[12] or disruption of the endothelium from acute stretching of the retinal vessels due to sudden deformation of the globe[2] may be likely mechanisms. Subsequent dislodging of the arterial thrombus into smaller perifoveal arterioles could have resulted in macular infarction in our case. Small branch vessel occlusions, mainly arteriolar, that occur following ocular contusion and affect the macula can result in a cherry-red spot as well, and its presence does not necessarily point to a CRAO.[2]

TON typically occurs following a history of head or midfacial trauma. The commoner form of TON is an indirect injury to the optic nerve where compression forces are transmitted to the orbital apex and optic canal resulting in a compartment syndrome where swelling exacerbates ischemia. The injury to the axons is thought to be induced by shearing forces that are transmitted to the fibers or to the vascular supply of the nerve.[13] The immediate and profound visual loss in our case was likely the result of the associated optic nerve injury.

In summary, blunt ocular trauma is another entity that may result in the rare event of macular infarction and needs to be considered in the differential diagnosis. Clinicians need to be aware of this association as fluorescein angiography is not performed routinely in patients following trauma, but may demonstrate the perifoveal arterial occlusion in suspected cases. Blunt ocular trauma following injury with a bull's leg is rare, and previously not described.

Conflict of interest

The authors declared that there is no conflict of interest.

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