• Letter to the Editor •

Central retinal artery occlusion and traumatic optic neuropathy following blunt ocular trauma

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Dear Editor,

We are writing to present a case report of central retinal artery occlusion (CRAO) and traumatic optic neuropathy (TON) with immediate visual loss caused by blunt facial trauma with a soccer ball. CRAO is an ophthalmic emergency with an estimated incidence of 10 in 1 million[1]. There are few reported cases of retinal artery occlusion (RAO) caused by ocular contusion[2-22]. Similarly, TON is a rare cause of severe permanent visual impairment caused by an injury, with an estimated incidence of 1 in 1 million[23]. Combined CRAO and TON occur very rarely and have been reported in isolated cases of ocular trauma[12,16-18,20]. The aim of this article is to assess the clinical presentation and possible mechanism for development of trauma-associated CRAO. This article also summarises all 17 cases of RAO associated with trauma that were published in the literature in English.

SUBJECTS AND METHODS

A detailed analysis of the literature in English has been performed. The following key words: central retinal artery, retinal artery occlusion, traumatic optic neuropathy, central retinal vein occlusion, trauma, blow out fracture, head injury and the following Medical Subject Headings: retinal diseases, optic nerve diseases, fluorescein angiography, macula lutea, and blindness were used for literature review.

The analysis revealed a total of 22 cases (from 21 authors) describing RAO associated with ocular trauma in healthy persons. Early reports describing traumatic CRAO provided insufficient information concerning the mechanism, clinical presentation and final visual acuity (VA) and are hence excluded in this analysis[2-4]. Reports not written in English have not been included in this analysis. Only 17 reports published in English literature from November 1987 to January 2017 are included and described in this assessment.

The CRAO sickle cell trait patients reports are not included in this assessment[24-27]. Sickle cell trait in the presence of precipitating factors, such as blunt ocular trauma may lead to localized hypoxia, promotion of erythrocytes sickling leading to vaso-occlusion and resultant blood stagnation[24-28]. In addition, cases of CRAO as consequences of traumatic cavernous sinus fistula have not been presented in this analysis[29-30]. In these cases, the elevation of pressure in the cavernous sinus increases the pressure in the central retinal artery causing the arterial pressure in the retina to obstruct the retinal circulation, which results in a progression from stasis retinopathy to CRAO[29-30]. Cases of CRAO as a result of surgical intervention such as retrobulbar anaesthesia for phacoemulsification[31] and vitrectomy[32], or other orbital[33] and periorbital interventions[34] are also not included in this assessment. In these cases, the anaesthetic agent vasoconstrictive effects on the central retinal artery, the mechanical effect or even abnormal arterial anastomosis could have potentially resulted in the development of CRAO[31-34]. This article includes two cases of macular vessel occlusion after ocular trauma, presented by Dalma-Weiszhausz *et al*[11] and one macular infarction case and TON presented by Goel *et al*[20] which the authors described as non-correspondent to Purtscher's retinopathy. The current study was approved by the University Clinical Centre Tuzla Ethics Committee. Informed consent was obtained from the patient and his parents after receiving an explanation of the investigative nature and intent of the study and tenets of the Helsinki Declaration were followed.

CASE REPORT

A 15-year-old boy had been presented with a sudden loss of vision in his left eye, immediately after blunt facial trauma. The boy was hit by a soccer ball. Clinical examination performed 1h after the injury revealed VA of 20/20 in the right eye and

no light perception (NLP) in the left eye. The patient stated that he had sustained a minor hit by the ball in his left cheek. On clinical examination, erythema was noted on the maxillary and infraorbital region with no other signs of trauma. Anterior segment examination of both eyes was unremarkable and the measured intraocular pressure was 15 mm Hg bilaterally. Right eye fundus examination was normal. However, the left eye fundus examination revealed a diffusely pale retina with retinal vessel attenuation, and there were no signs of retinal haemorrhage. The right pupil reacted normally to direct light, while the left pupil was unresponsive to light and showed an afferent pupillary defect. Extraocular movements were full on both eyes.

Slit-lamp examination of the left eye on the subsequent morning, twelve hours after the injury, revealed a cherry red spot in the fovea with a significant diffuse retinal oedema (Figure 1). The fundus examination, colour vision, visual fields and visually evoked potentials (VEP) of the right eye were normal. The eyeball and the optic nerve ultrasonography (US) examinations were normal in both eyes. Optical coherence tomography (OCT) showed normal retina in the right eye and severe retinal oedema in the left eye (Figure 2A). Computed tomography (CT) and magnetic resonance imagining (MRI) showed no signs of fractures and no signs of optic nerve (ON) or brain abnormalities. Colour Doppler ultrasonography of the heart, head and carotids were normal. Neurological and paediatric examinations were normal. Laboratory tests, including the inflammatory markers ones, were within reference range. There was no suggestive evidence of familial conditions and the patient's twin brother was of good health.

The patient was treated conservatively for acute CRAO with topical mydriatics and steroids, oral acetazolamide, intravenous mannitol, and methylprednisolone 1 g for three days. This was followed by oral prednisone tapering for 4wk. Surgical options were discussed with the patient and his family but, however, they were declined. Ten days after the trauma, the signs of retinal flow restoration and retinal oedema diminution were noted but VA remained unchanged. Fluorescein angiography (FA) one month after the trauma was normal in the right eye, while in the left eye it showed restoration of central retinal flow, leakage around the ON head in all phases and a small foveal leakage. The arterial time was normal (Figure 3). The final diagnosis was that of CRAO complicated with TON.

Three months following the trauma, the VA in the left eye remained at NLP. Slit-lamp examination revealed a normal anterior eye segment, optic disc pallor and no signs of neovascularization development. The OCT showed small residues of retinal oedema and significant reduction in retinal nerve fiber layer thickness in the left eye. During the follow up 3y after the traumatic incident, a discrete left eye exotropia, retinal vessel attenuation with ON atrophy and no signs of neovascularization were noted.

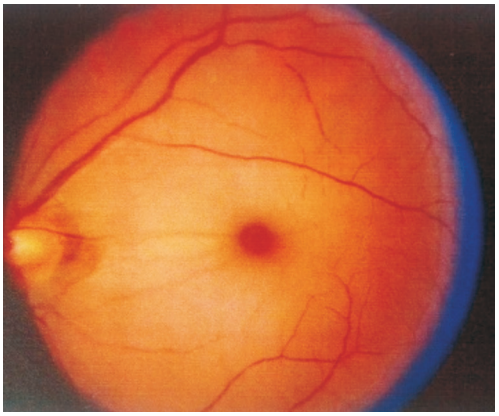

Figure 1 Fundus photography 12h after the accident, presenting diffuse retinal oedema and cherry red spot.

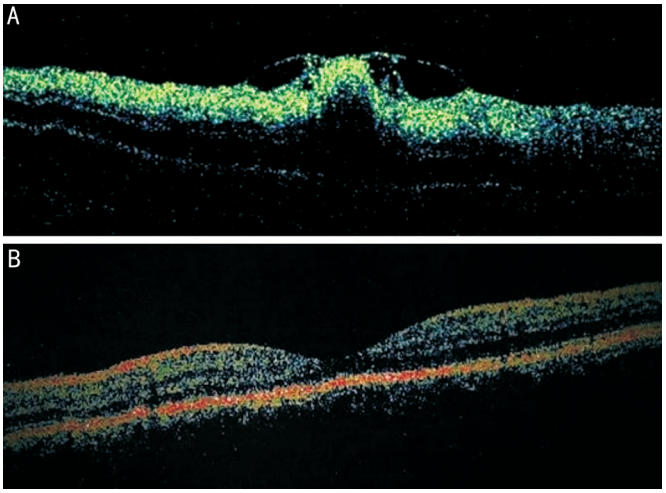

Figure 2 Initial and 3mo OCT following the trauma A: Initial OCT presenting an irregular foveal contour with a hyperreflective, thickened inner retina. The outer retina is relatively hyporeflective because of blocking form the thickened inner retina. The inner-outer segment junction and the external limiting membrane are intact; B: OCT image 3mo following the trauma presents the normal foveal contour with discrete residues of the intraretinal oedema.

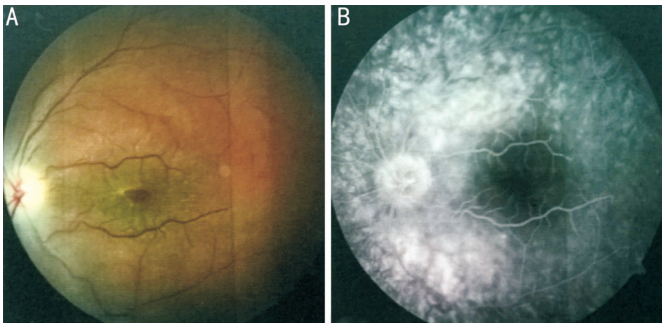

Figure 3 Fundus photography and fluorescein angiography 3mo following the trauma A: Fundus photography presenting optic nerve head pallor and residues of retinal oedema; B: FA presenting restored central retinal flow, leakage around optic nerve head.

DISCUSSION

Retinal vascular occlusion is potentially harmful for the visual functioning. Ocular vascular occlusive disorder is associated with arterial hypertension, diabetes mellitus, renal disease, ischemic heart disease and carotid artery disease[35]. Rare

reports present cases of RAO following ocular contusion with different clinical features when compared with RAO secondary to vasculopathy[7-22]. Thus, these cases can be presented as isolated CRAO[8-10,13-15,19,21], small branch vessel occlusions in both central and peripheral retina[11], simultaneous CRAO and central retinal vein occlusion (CRVO)[7,22], simultaneous CRAO and TON[12,16-17,20] and even as simultaneous CRAO, CRVO and TON[18].

In our case, clinical examination, US, OCT, FA and MRI found no signs of eye perforation or ON avulsion (ONA). Severe retinal oedema was noted on clinical examination and OCT, with changes in retinal vasculature and a "cherry red spot" which was congruent with a diagnosis of CRAO. Unchanged VA after retinal flow restoration and oedema resolution with noted leakage around the ON in FA one month after the injury indicate that this is also a case of TON.

A summary of all 17 cases of trauma associated CRAO in healthy patients found in literature is presented in Table 1. RAO associated with trauma occurred in both genders and in patients aged between 6 and 47y, in both eyes, after mild sports related trauma or after more serious trauma such as during a road traffic accident. Most of these cases presented with sudden painless visual loss which occurred either immediately or a few hours after the trauma. However, late onset CRAO was noted in one case which occurred 6mo after the trauma with a ball[8]. In most cases, anterior segment appeared to be normal with positive relative afferent pupillary defect (RAPD). This may be accompanied with conjunctival hyperaemia or haemorrhage, hyphema, or severely swollen eyelids in cases of orbital blow out fractures. Associated findings include periocular bruising, mandibular and orbital fractures and loss of consciousness. Posterior segment findings included an oedematous and pale retina, arteriolar attenuation and cherry red spot (noted in 70% of cases: 12 out of 17 cases). This assessment also presents 3 cases of simultaneous CRAO and CRVO where flame shaped and pre-retinal hemorrhage were noted as well[7,18,22]. FA showed either delayed retinal filling and arterial narrowing or obstruction with normal choroidal filling. In cases of macular infarction, FA also demonstrated areas of hypofluorescence at the macula, while additional staining of the ON head is noted in cases of associated TON[11,20].

The exact cause of the CRAO could not be determined in these cases. The anatomy of the ON may provide an explanation of how a mild blow to the cheek could result in such a devastating eye injury. With no evidence of direct ocular trauma, the facial contusion could have induced a compression force which was transmitted to the orbit, resulting in stretching of the blood vessels. This in turn could induce focal vasospasm or retinal vessel stretching resulting in endothelium damage which leads to thrombus formation and vessel occlusion with consequent retinal and ON ischemia[11,14,18-19,21-22]. Our patient suffered complete loss of vision immediately after the trauma indicating that ON damage was present immediately and that ON damage was not a result of prolonged ischemia due to vascular occlusion. This is similar to previous CRAO and TON reports[12,16-17]. There were no signs of bone fracture on CT scans and no signs of ON damage or oedema on MRI and US, which suggests that this is a case of indirect TON, and that the ON was damaged at the level of lamina cribrosa[1].

Each patient in this article, except three of them were treated conservatively. Narang *et al*[13] and Zahavi and Rosenblat[19] performed paracentesis, while Vaitheeswaran *et al*[17] performed optic canal decompression. Visual outcome in patients with trauma associated CRAO is poor, with 50% of cases being left with an immediate and final VA of NLP. Only one patient had a functionally useful VA[17]. In these cases, retinal oedema resolves weeks after the initial incident, and optic disc pallor (as a result of ON atrophy) and changes in macular pigment epithelium occur consequently[19]. Out of four known cases of CRAO and TON, the three were managed conservatively while one underwent invasive treatment. In the first case, CRAO was associated with optic nerve avulsion after direct ocular trauma and the final VA was light perception[12]. In the second case, there was direct ocular trauma and initial VA was NLP which did not change after conservative therapy[16]. The third case occurring after facial trauma was initially treated with optic canal decompression and direct ophthalmic fibrinolysis which resulted in VA improvement to a final VA of 6/9[17]. In a case of traumatic macular infarction with TON after a hand strike, conservative treatment also resulted in a final VA of NLP[18]. In our case, a mild facial trauma one, the patient was treated conservatively and initial VA was NLP. Invasive methods of treatment were considered but not adopted due to financial constraints. The results of research on trauma associated CRAO, especially CRAO with TON, suggest that each case has to be assessed individually. In addition, it is necessary to recognize CRAO with possible TON early and commence all possible treatment as VA prognosis would be very poor otherwise.

In conclusion, we have presented a rare case of CRAO combined with TON as a result of mild head trauma, resulting in complete and permanent visual loss in a young adult. This case highlights the possibility of severe visual loss in a case of mild facial trauma and clinicians should be aware of its potential devastating consequences.

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Table 1 Clinical characteristics of patients with RAO associated with trauma

Author	Age (y)	Gender	Side	Object and mechanism	Presenting VA (symptom onset)	Periocular and anterior segment finding	Posterior segment finding	Final diagnosis	Treatment	Final VA	Associated findings and remarks
Noble and Alvarez, 1987[7]	28	F	Left	Finger jabbing in the eye	NLP (immediate)	Linear conjunctival laceration	Pale optic disc, narrow retinal arterioles, cherry red spot, engorged retinal veins with few small haemorrhages	CRAO and CRVO	Conservative	NLP	None
Garzozi *et al*, 1990[8]	6	M	Left	Hit with a ball in the cheek	NLP (6mo)	6mo before hematoma of the lower eyelid	Oedema of the posterior pole and a cherry red spot	CRAO	Conservative	NLP	Late onset of the CRAO: 6mo after the trauma in left cheek
Cohen *et al*, 1993[9]	14	M	Right	Punch in the eye	NLP (4d)	Normal finding	Intraretinal haemorrhages, attenuation of the retinal arteries and veins and retinal ischemia	CRAO	N/A	N/A	Diagnosed with orbital ultrasonography
Umeed and Shafquat, 2004[10]	15	M	Right	Hit by a football in face	Hand movements (2d)	Minimal conjunctival congestion and flare in anterior chamber	Retinal pallor and swelling with subsequent arterial attenuation	CRAO	Conservative	3/60	Insignificant hematoma in the orbital apex
Dalma-Weiszhausz *et al*, 2005[11] (Patient 2 and patient 4)	34	M	Left	Bullet wound	NLP (3d)	Normal finding	Pale macular area, with cherry red spot and retinitis sclopetaria	Macular infarction and possible TON	Conservative	NLP	No consciousness for 3d examined on 9th day
	42	F	Both eyes	Trauma in motor vehicle accident	Subjective complete visual loss (7d)	Normal finding	Pale optic disc and retina, with narrowed arterioles and vein engorgement	Macular infarction and possible TON	Conservative	Hand motion in right and 20/400 in left eye	Coma for 7d
Chong and Chang, 2006[12]	14	M	Left	Finger gouging	Light perception (immediate)	Periorbital bruising and microscopic hyphaema	Pre and subretinal haemorrhage surrounding optic disc and retinal oedema with cilioretinal sparing	CRAO and ONA	Conservative	Light perception	Later complicated with neovascular glaucoma
Narang *et al*, 2007[13]	29	M	Both eyes	Traffic accident	NLP in both eyes (12h)	Laceration on left side of forehead, left eye, enophthalmos.	Both eyes: pale disc oedema, retinal whitening, attenuated arteries with cherry red spot, together with yellow shiny emboli in the left eye	CRAO	Paracentesis and conservative	Bilateral NLP	Unconscious patient CT orbit: fracture of roof and lateral wall with later thrombus in right internal carotid artery
Himori *et al*, 2009[14]	20	F	Right	Traffic accident	NLP (immediate)	Severely swollen eyelids, corneal oedema, mydriasis and hyphaema	Milky white retinal oedema and cherry red spot	CRAO	Conservative	Light perception	Severe blow out fracture which was later operated
Ghose and Subhabrata, 2011[15]	30	M	Both eyes	Head injury when falling at a construction site	Finger counting in right and hand movement in left eye (2h)	Subconjunctival haemorrhage in left eye	White out retina with cherry red spot and few intraretinal haemorrhages	CRAO	Conservative	Finger counting in right and hand movement in left eye	Mandible fracture
Cumurcu *et al*, 2011[16]	10	M	Left	Hit by a football in face	NLP (12h)	Normal	Pale macular area and disc, with cherry red spot and areas of retinal whitening	CRAO and TON	Conservative	NLP	Immediate visual loss with signs of CRAO visible on the second day
Vaitheeswaran *et al*, 2014[17]	10	M	Left	Fall from height	NLP (6h)	Normal	Disc oedema with retinal pallor, arteriolar attenuation and venous dilatation	CRAO and TON	Operative optic canal decompression	6/9	Postoperative visual field showing restriction with preservation of central fields
Kumar Singh *et al*, 2014[18]	22	M	Left	Traffic accident	NLP	Lower eyelid laceration, subconjunctival haemorrhage	White-out retina cherry red spot, pale optic disc with narrow arterioles. Next day flame shaped and pre-retinal haemorrhages	CRAO, CRVO and TON	Conservative	NLP	None
Zahavi and Rosenblat, 2015[19]	34	F	Right	Hand strike	Finger counting (2d)	Normal	Retinal pallor and cherry red spot	CRAO	Paracentesis and conservative	Finger counting	None
Goel *et al*, 2016[20]	15	M	Left	Trauma with a bull's leg	NLP (2d)	Periorbital bruising of the eyelids, anterior chamber flare and traumatic mydriasis	Retinal whitening at the posterior pole with few intraretinal haemorrhages, a cherry red spot with a mild pallor of the optic disk	Macular infarction and TON	Conservative	NLP	None
Filev *et al*, 2016[21]	47	M	Right	Hit by a broomstick	Hand movements (2h)	Small conjunctival laceration	A pale oedematous papilla, a macular oedema with cherry red spot as well as narrowed retinal vessels	CRAO	Conservative (Rheological therapy)	0.05	None
Bouraoui *et al*, 2017[22]	33	F	Left	Projection of a stone	NLP (10d)	Corneal oedema with total hyphaema	Swollen optic disc with, enlarged and tortuous veins, peripheral haemorrhages and a retinal whitening of the posterior pole with a cherry-red spot	CRAO and CRVO	Washout of the anterior chamber and conservative	NLP	None
Current study	15	M	Left	Hit by a football in face	NLP (1h)	Discrete skin redness in left cheek	Diffusely pale retina with retinal vessel attenuation, next day cherry red spot	CRAO and TON	Conservative	NLP	Later FA presented ON hiperfluorecence as sign of TON

RAO: Retinal Artery Occlusion; VA: Visual acuity; NLP: No light perception; CRAO: Central retinal artery occlusion; CRVO: Central retinal vein occlusion; N/A: Not available; TON: Traumatic optic neuropathy; ONA: Optic nerve avulsion; CT: Computed tomography; FA: Fluorescein angiography; ON: Optic nerve.

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