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CORRESPONDENCE

Sir,

Valsalva and Purtscher's retinopathy with optic neuropathy in compressive thoracic injury

Compressive thorax injury is commonly observed in road traffic accidents which leads to a variety of posterior segment ocular abnormalities, of which rarely seen are Purtscher's retinopathy, Valsalva retinopathy, and traumatic optic neuropathy.[1] We report a rare case with compressive chest injury which led to Purtscher's retinopathy with traumatic optic neuropathy in one eye and Valsalva retinopathy in the other.

Case report

We report the case of a 30-year-old male who presented with compressive chest injury when his tractor turned over the road edge. After an episode of unconsciousness for 2 h, he complained of no vision in the right eye and reduced vision in the left eye. He reported to our centre after 2 weeks of systemic treatment for local abrasions and pain. Detailed examination revealed healing ecchymosis and abrasions on the chest. His vital parameters were stable. No underlying fractures or internal organ injury was detected upon thorough examination by an orthopaedic surgeon and physician. The visual acuity was no perception of light in the right eye and best corrected 3/60 in the left eye. There was no evidence suggestive of direct trauma in the form of ocular perforation, conjunctival/lid lacerations, angle recession, lens subluxation, or retinal tears. Ocular movements were normal. The applanation pressure in both eyes was 18 mmHg. The pupil was fixed and dilated in the right eye and demonstrated an afferent pupillary defect, while the left pupil was brisk and reactive. A detailed fundus examination revealed total disc pallor with multiple soft exudates in the right eye and a resolving superior subhyaloid haemorrhage encroaching on the macular area in the left eye (Figure 1). The flash VER revealed an extinguished response in the right eye and a delayed latency in the left eye. Fundus fluorescein angiography revealed delayed filling, capillary nonperfusion, venous staining in the right eye, and blocked hypofluoresence corresponding to the subhyaloid haemorrhage in the left eye. X-ray orbits and CT scan head failed to reveal any other pathology or fractures. No obvious injury to optic canals was noted.

He was diagnosed as a case of resolving Purtscher's retinopathy with traumatic optic neuropathy in the right eye and Valsalva retinopathy in the left eye with subhyaloid haemorrhage, based on the clinical picture and evidence of trauma. The poor visual prognosis was explained to the patient in the right eye in view of advanced optic atrophy. He was advised Nd: YAG hyaloidotomy for the subhyaloid haemorrhage in the left eye with explained prognosis, which is a known beneficial treatment for this condition.[2]

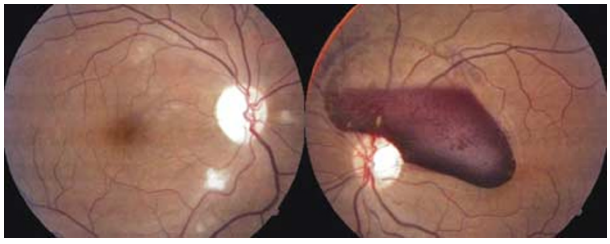

Figure 1 Fundus photo of right eye showing resolving Purtscher's retinopathy with traumatic optic neuropathy. Fundus photo of left eye showing Valsalva retinopathy with resolving preretinal haemorrhage.

Comment

Putscher's retinopathy is a rare observation, which presents to the clinician as loss of vision in a patient with a history of a possible precipitating event such as recent major trauma, pancreatitis, childbirth, or renal failure. [3,4] Ocular findings include retinal haemorrhages, disc pallor, soft exudates, retinal oedema and ischaemia of the posterior pole possibly due to embolisation of the peripapillary terminal arterioles supplying the superficial peripapillary capillary net. Since the fundus picture in the right eye was not very typical of Putscher's retinopathy, we assume it was probably resolving as it was 20 days old at presentation.

Although optic atrophy has been associated with Purtscher's retinopathy,[5] the poor vision associated with rapid development of disc pallor in right eye suggests a diagnosis of traumatic optic neuropathy. The nonrecordable VER and associated history of loss of consciousness also indicate that a concussive force could have led to indirect optic nerve injury. Although the CT scan showed normal optic nerve canals, the fixed part of the optic nerve is susceptible to indirect injury by shear forces at the entrance of the optic canals by concussive forces. Management of such cases of indirect optic nerve trauma is controversial, with many advocating observation, corticosteroids, or surgical intervention.[6,7] However, no clear benefit for corticosteroid therapy or optic canal decompression surgery has been observed and the visual prognosis is poor in many cases.[6–8]

The left-eye fundus was typical of Valsalva retinopathy which is usually observed following strenuous exertion, weight lifting, emesis, violent coughing, tenesmus, end stage labour, blowing musical instruments, and crush or

compression injuries.[9] It is caused by forcible exhalation against a closed glottis, thereby creating a sudden increase in intrathoracic/intra-abdominal pressure, which leads to rapid rise of intraocular venous pressure with rupture of perifoveal capillaries and a preretinal haemorrhage.

Purtscher's retinopathy, traumatic optic neuropathy and Valsalva retinopathy are rare entities associated with trauma that have different clinical presentations caused by dissimilar mechanisms, and it is very uncommon to observe them together in either eye as seen in our case. In spite of the severe nature of the ocular injury, it is surprising that our patient was systemically stable with no significant bony or organ injury. Compressive thoracic injury has varied ocular presentations caused by ill-understood pathophysiological mechanisms and new ocular findings associated with it continue to baffle us.

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Sir,

Treatment of polypoidal choroidal vasculopathy with transpupillary thermotherapy: an interventional case report

A 71-year-old Indian male patient presented to the retina–vitreous service of our tertiary care hospital with the complaints of progressive decrease in vision in the right eye over the past 6 months. Ocular examination revealed a best-corrected visual acuity of 6/12 and 6/6 (distance vision) and N18 and N6 (near vision) in the right and the left eye, respectively. Fundus examination of the left eye was unremarkable. Indirect ophthalmoscopy of the right eye revealed a serous detachment of the macula with extensive lipid exudation and an area of thickening superior to the disc suspicious of a choroidal neovascular membrane (CNVM) (Figure 1a). Fundus fluorescein angiography (FFA) demonstrated a progressively increasing irregular hyperfluorescence in that region and pooling of dye in the serous detachment in the macula but was otherwise noncontributory to the diagnosis. Indocyanine green angiography (ICGFA) was carried out that showed a branching vascular network in the inner choroid ending in two terminal hyperfluorescent polypoidal dilatations in the superior juxtapapillary region suggestive of leaking polypoidal choroidal neovascularisation (CNV) (Figure 2a). There were also two areas of ill-defined hyperfluorescence superior and superonasal to the foveola. Based on the above findings, a diagnosis of polypoidal choroidal vasculopathy (PCV) of the right eye was made.

In view of the long duration of persisting poor visual acuity, the macular involvement, and the abundant lipid suggesting an active exudative process, it was decided to treat the leaking polypoidal lesions. After informed consent, the patient underwent transpupillary thermotherapy (TTT) to the juxtapapillary vascular network inclusive of the polypoidal lesions (2 spots, 2 mm in size, 250 mW power, 1 min duration, end point: no visible reaction). Follow-up at 1 month revealed a resolved macular detachment and a reddish-orange subretinal lesion superior to the fovea that was typical of PCV (Figure 1b). The ICGFA revealed the disappearance of the treated polypoidal lesions (Figure 2b). It also showed a row of aneurysmal dilatations of the inner choroidal vascular network superior to the fovea,