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Traumatic optic neuropathy and central retinal artery occlusion following blunt trauma to the eyebrow

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A 15-year-old boy suffered from blunt trauma to the left eyebrow (Figure 1A arrow). Three hours after the trauma, he had a visual acuity (VA) of no light perception (NLP) in the left eye. Relative afferent pupillary defect was positive in the left eye. The intraocular pressure of each eye was 15 mm Hg. Anterior segment examination results were unremarkable. Funduscopic examination results were normal on the first day. Computed tomography and magnetic resonance imaging showed edema in the left optic nerve without optic canal fractures. Thus, we clinically diagnosed traumatic optic neuropathy. Therefore, intravenous bolus therapy with methylprednisolone (1000 mg) was initiated. On the third day, a pale macular area with a cherry-red spot was noted in the left eye (Figure 1B). Fluorescein angiography (FA) showed early-stage central retinal artery occlusion (CRAO) with capillary nonperfusion (Figure 1C). Late-stage of FA showed markedly retarded influx of fluorescein into the retinal arteries. We performed paracenthesis. However, VA was still NLP.

It is well known that blunt trauma to the lateral eyebrow causes optic nerve damage. Previously, isolated cases of CRAO and optic nerve damage following ocular trauma have been reported, although extremely rarely.[1] To our knowledge, this is the second documentation of CRAO that developed on the few days after traumatic optic neuropathy following blunt trauma to the eyebrow.

The mechanism of traumatic optic nerve injury could be direct mechanical compression of the optic nerve; combined forces of compression and/or traction to central retinal artery; or the compression effect on the small nutrient vessels feeding the optic nerve.[2] Namely, compression forces transmitted to the orbital apex cause a compartment syndrome, whereby compression leads to a vicious cycle of

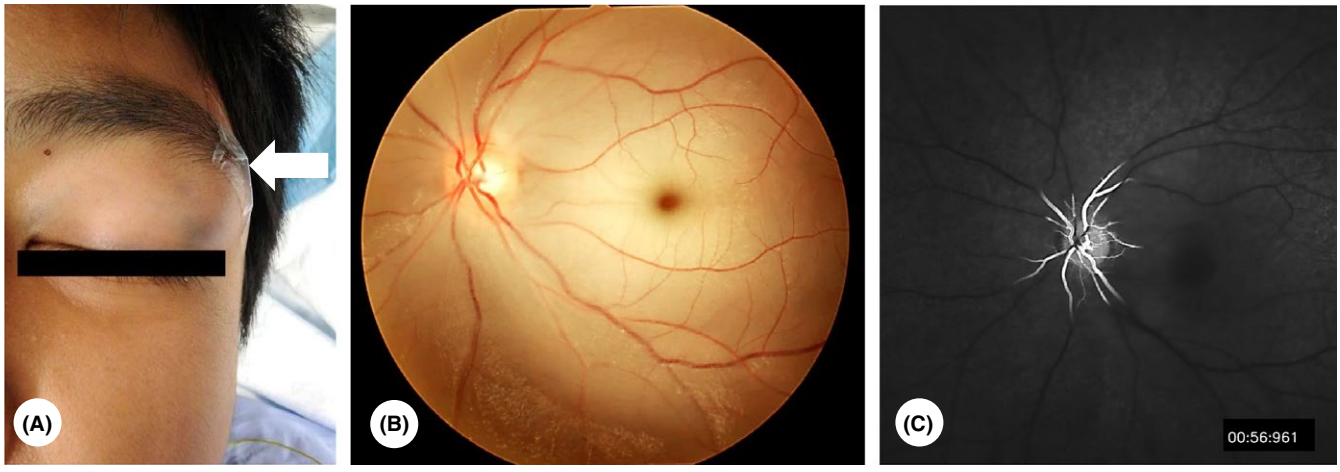

FIGURE 1 Blunt trauma to the eyebrow (A), fundus photograph (B) and fluorescein angiography (C)

swelling and ischemia, release of free oxygen radicals, and damage to the axons.[2] We, thus, speculate that the development of CRAO may well be initiated by mechanical compression of the central retinal artery during the earlier phase, and further facilitated by local vasospasm and/or compression of the central retinal artery due to the swelling of the optic nerve at the optic canal during the later phase. Finally, clinicians should be aware of the potential for blunt ocular trauma resulting in not only optic nerve damage but also retinal artery occlusion.

CONFLICT OF INTEREST

The authors have stated explicitly that there are no conflicts of interest in connection with this article.

INFORMED CONSENT

Informed consent was obtained from all individual participants included in the study.

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