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Review article

Traumatic optic neuropathy—Clinical features and management issues

Patrick Yu-Wai-Man [a, b, c, d, *]

[a] Wellcome Trust Centre for Mitochondrial Research, Institute of Genetic Medicine, Newcastle University, Newcastle upon Tyne, UK
[b] Newcastle Eye Centre, Royal Victoria Infirmary, Newcastle upon Tyne, UK
[c] UCL Institute of Ophthalmology, University College London, London, UK
[d] Moorfields Eye Hospital, London, UK

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ABSTRACT

Traumatic optic neuropathy (TON) is an uncommon cause of visual loss following blunt or penetrating head trauma, but the consequences can be devastating, especially in cases with bilateral optic nerve involvement. Although the majority of patients are young adult males, about 20% of cases occur during childhood. A diagnosis of TON is usually straightforward based on the clinical history and examination findings indicative of an optic neuropathy. However, the assessment can be difficult when the patient's mental status is impaired owing to severe trauma. TON frequently results in profound loss of central vision, and the final visual outcome is largely dictated by the patient's baseline visual acuities. Other poor prognostic factors include loss of consciousness, no improvement in vision after 48 hours, the absence of visual evoked responses, and evidence of optic canal fractures on neuroimaging. The management of TON remains controversial. Some clinicians favor observation alone, whereas others opt to intervene with systemic steroids, surgical decompression of the optic canal, or both. The evidence base for these various treatment options is weak, and the routine use of high-dose steroids or surgery in TON is not without any attendant risks. There is a relatively high rate of spontaneous visual recovery among patients managed conservatively, and the possible adverse effects of intervention therefore need to be even more carefully considered in the balance.

1. Classification

Traumatic optic neuropathy (TON) refers to any insult to the optic nerve secondary to trauma. It can be classified depending on the site of injury (optic nerve head, intraorbital, intracanalicular, or intracranial) or according to the mode of injury (direct or indirect).[1,2] In direct TON, there is significant anatomical disruption to the optic nerve, for example, from a projectile penetrating the orbit at high velocity (Fig. 1), or as a result of optic nerve avulsion (Fig. 2). Indirect TON is caused by the transmission of forces to the optic nerve from a distant site, without any overt damage to the surrounding tissue structures. The deformative stress transmitted to the skull from blunt trauma is concentrated in the region of the optic canal. The intracanalicular segment of the optic nerve is particularly susceptible to this form of injury, because the dural sheath is tightly adherent to the periosteum at this specific location.[3,4] The intracranial portion of the optic nerve in close proximity to the falciform dural fold is the next most common site at risk of injury.[5] In one report using computerized tomography (CT) imaging, about half of all TON cases were found to have an associated sphenoidal bone fracture, an indirect measure of the significant compressive forces involved at impact.[6] However, both direct and indirect mechanisms can contribute to optic nerve damage, and a clear distinction is not always possible.

2. Pathophysiology

The pathophysiology of indirect TON is likely to be multifactorial, and the concept of primary and secondary injury has been proposed.[7,8] Following trauma, there is an immediate shearing of a

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* Wellcome Trust Centre for Mitochondrial Research, Institute of Genetic Medicine, Newcastle University, Central Parkway, Newcastle upon Tyne, NE1 3BZ, UK.
E-mail address: Patrick.Yu-Wai-Man@ncl.ac.uk.

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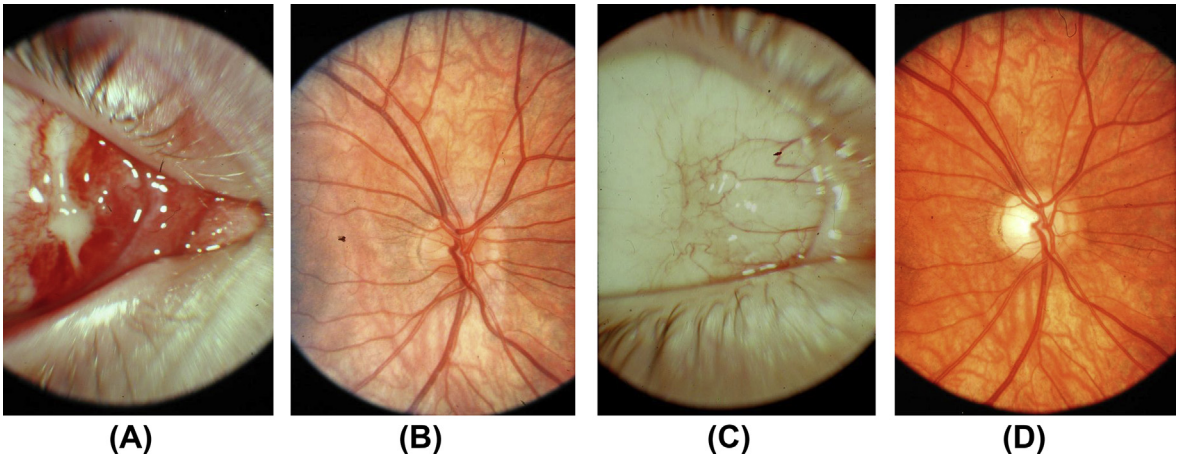

Fig. 1. Direct traumatic optic neuropathy. (A) Entry site of a projectile in the medial canthal region of the right eye. (B) The patient's posterior pole was normal when he was assessed shortly after the accident. His visual acuity at that time was no perception of light. (C) Conjunctival scar over the entry site. (D) Optic disc pallor, more marked temporally, was apparent 6 weeks later. The patient's visual acuity had not improved and he was subsequently lost to follow-up. (Courtesy of Professor David Taylor, Institute of Child Health, London, UK.)

proportion of retinal ganglion cell axons, an irreversible process that results in neuronal loss. There is then a degree of optic nerve swelling within the tight confines of the optic canal secondary to direct mechanical trauma and vascular ischemia. The ensuing compartment syndrome further impairs the already compromised blood supply to surviving retinal ganglion cells, setting up a downward spiral toward apoptotic cell death. This two-stage model of TON forms the basis for optic nerve decompression by medical or surgical means, in order to break this vicious cycle and to preserve the remaining retinal ganglion cells that survived the initial insult.

3. Epidemiology

TON is an uncommon cause of visual loss following blunt or penetrating head trauma with a reported incidence of 0.7–2.5% in published case series.[9–12] A recent national epidemiological survey of TON in the United Kingdom found a minimum prevalence in the general population of one in 1,000,000.[13] The vast majority of affected patients are young adult males (79–85%) in their early 30s. The most common causes of TON in this patient group are motor vehicle and bicycle accidents (49%), falls (27%), and assaults (13%).[13,14] In the pediatric population, the majority of TON cases are secondary to falls (50%) and road traffic accidents (40%).[15]

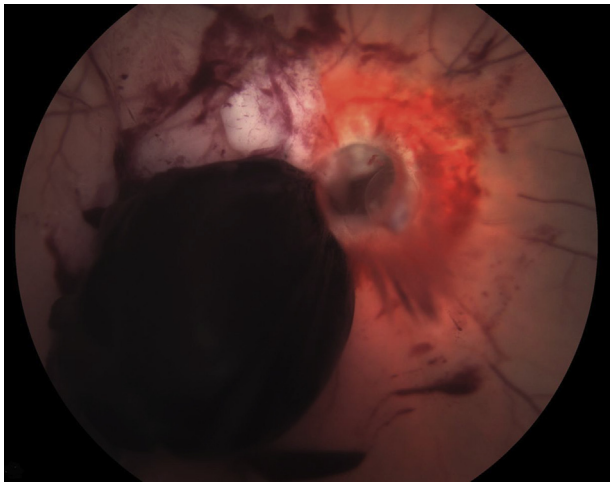

Fig. 2. Traumatic optic nerve avulsion following a road traffic accident. (Courtesy of Dr Scott Schoenberger, Vanderbilt Eye Institute, Nashville, TN, USA.)

4. Clinical assessment

TON is a clinical diagnosis supported by a history of direct or indirect trauma to the head or face. The injury can sometimes be trivial, and a careful history of the incident must be elicited from the patient and any other witnesses that might have been present especially when dealing with children or unconscious patients. A detailed record should also be kept as cases of TON are not infrequently the subject of future medicolegal proceedings. Although usually straightforward, the clinical assessment can sometimes prove difficult in the setting of severe trauma when the patient's level of consciousness is impaired. In this scenario, it is essential to exclude possible reversible causes of visual loss that require immediate attention, for example, a retrobulbar hemorrhage. The patient's baseline visual acuity should be clearly documented in the notes and even if only a bedside examination is possible, this can still be achieved with a portable vision chart and the use of a pinhole occluder. A thorough examination of the eye and the adnexal structures is mandatory, with particular care taken to exclude associated orbital or facial fractures requiring more specialized maxillofacial input. Except when neurosurgical monitoring of the pupil is required, a detailed dilated examination of the posterior pole must be carried out to document the state of the optic disc, any associated retinal or vitreous hemorrhages, and the possibility of an intraocular foreign body in cases of penetrating trauma. A high degree of clinical vigilance must also be maintained because TON can infrequently be associated with delayed visual loss secondary to the development of an optic nerve sheath hematoma (Fig. 3). The following features are consistent with a diagnosis of TON. (1) Unilateral or bilateral ocular involvement. (2) A relative afferent pupillary defect except in bilateral symmetric cases. A relative afferent pupillary defect is an important clinical sign, and in patients with mild TON, it can be the only objective evidence of optic nerve dysfunction prior to the development of overt optic atrophy. (3) Variable loss of visual acuity ranging from normal to no light perception. Between 40% and 60% of patients present with severe visual loss of light perception or worse at baseline.[13–17] (4) Impairment of color vision. (5) Variable visual field defects.