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Traumatic optic neuropathy

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The optic nerve is vulnerable to indirect and direct trauma causing functional impairment of vision. Optic nerve injuries occur in the setting of head injury which is often a consequence of road traffic accidents or falls. The diagnosis of optic nerve injury may be delayed by the presence of other life-threatening injuries. The signs of trauma to the optic nerve are clinical and the examination of acutely injured patients poses difficulties for the clinician. In this article, I shall consider the diagnosis of optic nerve injuries and discuss the dilemmas of management that may arise.

Classification of optic nerve injuries

Direct injury to the optic nerve should be distinguished from indirect injury. Direct injury arises from penetrating trauma, especially orbital fractures associated with mid-facial fractures. Several varieties of direct optic nerve injury may be recognised ophthalmoscopically or with imaging techniques: optic nerve avulsion, transection, optic nerve sheath haemorrhage, orbital haemorrhage, and orbital emphysema. Indirect optic nerve injury is more common. The force of impact in a head injury may be transmitted to the optic nerve. This complication of head injury was known to Hippocrates who noted that blows to the eyebrow may cause blindness. The frequency of optic nerve injury occurring in closed head injury varies from 0.5 to 5%.[1] The site of injury causing blindness is usually the forehead or supraorbital ridge, less commonly the temporal region. Generally, the blow is severe causing loss of consciousness, but occasionally the trauma may be slight and the patient only slightly dazed. The clinical sequence of events is typical; the patient recovers consciousness after head injury and discovers that the sight of one eye is damaged or lost. Ocular examination is initially normal apart from a relative or absolute afferent pupillary defect. A wide variety of visual field defects may occur. Optic atrophy develops after 4–6 weeks.

Direct optic nerve injury

There are distinctive clinical syndromes of direct optic nerve injury that may be recognised by clinical examination and neuro-imaging using CT or MRI scanning[2] (Figures 1 and 2).

Optic nerve avulsion usually follows severe orbital trauma, but cases have been reported following relatively trivial injury. Vision loss is usually severe and immediate. Ophthalmoscopy shows absence of the optic disc and a ring of haemorrhage. Imaging may confirm the diagnosis but may show an intact sheath. Ultrasound may be helpful.[3] The nerve tears at the lamina cribrosa, perhaps as a result of rotation of the globe, raised intraocular pressure or sudden retropulsion followed by anterior displacement. No treatment is possible.

Optic nerve transection occurs as a complication of midfacial trauma and orbital fracture. There is no light perception, corroborated by visual evoked potentials. CT scanning may show the bone fragment transecting the optic nerve. No treatment is effective.

Optic nerve sheath haemorrhage may be difficult to recognise clinically and yet cause potentially reversible visual failure. An expanded nerve sheath associated with proptosis should raise the possibility of a haematoma in the optic nerve sheath, which can be drained via a sheath fenestration.

Orbital haemorrhage may be diffuse or localised to the orbit. There is an associated proptosis and ophthalmoplegia. The injury to optic nerve function results from raised pressure in the orbit and may be relieved by elevating the head and administering Diamox to lower the intraocular pressure. If conservative measures fail, then a lateral canthotomy and

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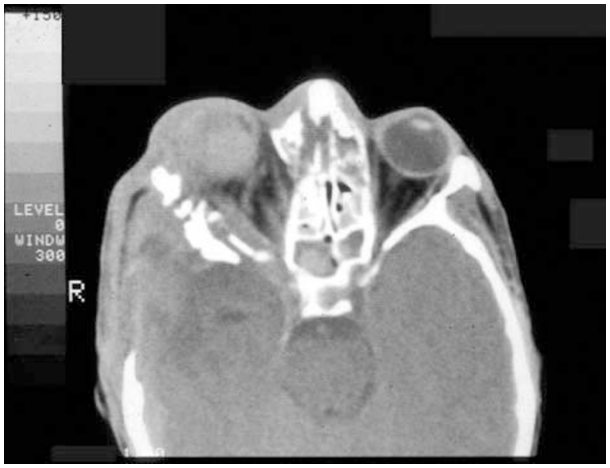

Figure 1 CT scan of orbit showing disrupted optic nerve following penetrating trauma (Sarkies[2]). Reprinted with permission from Elsevier.

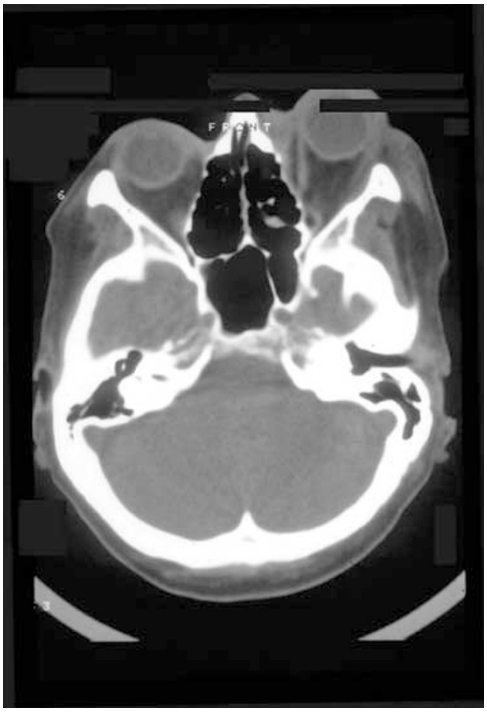

Figure 2 CT scan of orbit showing proptosis and distension of optic nerve sheath with probable haematoma (Sarkies[2]). Reprinted with permission from Elsevier.

drainage of orbital haemorrhage may restore function.

Orbital emphysema is a well-recognised clinical entity in the setting of injury to the paranasal sinuses. Hair-line fractures of the thin bone lining the orbital walls may produce a ball-valve effect so that air accumulates in the orbit and causes proptosis and compression of the eye and nerve. Drainage of the air by a retrobulbar needle has been effective.

Indirect optic nerve injury

The mechanism of damage to the optic nerve by closed head injury has been extensively studied. After blunt trauma to the superior orbital rim or fronto-temporal region of the cranium, compression forces are transmitted to the orbital apex and optic canal.[4] Within the canal, the optic nerve dura fuses with the periosteum of the bone. Since the vasculature of the optic nerve in the canal is pial, compression and contusion of the nerve produce a compartment syndrome whereby swelling exacerbates the ischaemia.[5] The optic nerve is also vulnerable to a shearing injury under the fixed edge of the falciform dural fold just before it enters the optic canal. Histopathological studies by Crompton[6] of 174 patients who died after closed head injury found evidence for haemorrhage in the optic nerve sheath (83%), in the nerve interstitium (36%), and shearing lesions and ischaemic necrosis of the intracanalicular and intracranial segments of the nerve.

Treatment of indirect optic nerve injury

The management of indirect optic nerve injury is controversial. There may be delay in diagnosis, and occasionally the loss of visual function appears as a delayed complication of head injury. The arguments for treatment of indirect optic nerve injuries are based upon the hypothesis that secondary injury to the axons occurs as a result of vasospasm and swelling within the optic canal. Experimental studies of optic nerve injury have employed crush, stretch, or severing injuries. The cellular mechanisms involved in CNS injury are incompletely understood. To summarize, several cellular messengers are activated by the trauma response. The release of oxygen-free radicals results in peroxidation of lipid cell membranes. Bradykinin and kallidin are activated following injury: these agents influence free radical production, intracellular calcium production, and arachidonic acid release from neurons. Subsequently cell-mediated inflammation certainly plays a prominent part in experimental models of optic nerve injury.

Very high-dose corticosteroids limit free-radical amplification of the injury response. The use of high-dose corticosteroids after optic nerve injury increased in the 1980 s following the report of Anderson *et al*.[4] The rationale was strengthened by studies on spinal cord injury. The second National Acute Spinal Cord Injury Study (NASCIS 2) was a multicentre, randomised, double-blind, placebo-controlled study of patients with acute spinal cord injury.[7] Patients were treated with

placebo, naloxone, or methylprednisolone, administered with an initial dose of 30 mg/kg followed by an infusion at 5.4 mg/kg/h for 24 h. Patients were evaluated by pinprick, light touch, and motor function initially, then at 6 weeks and 6 months. The study showed that the treatment with methylprednisolone within 8 h of injury resulted in a significant improvement in motor and sensory function compared to placebo-treated patients. Further analysis of the data suggested that methylprednisolone treatment initiated more than 8 h after spinal cord injury may be detrimental.[8] NASCIS 3 established that treatment should be continued for 24 h if initiated within 3 h after injury, but for 48 h if initiated within 3–8 h.[9]

Surgery to decompress the optic canal, via intracranial, transethmoidal, endonasal, even sublabial approaches has been advocated in many retrospective reports.[10–13] The reports of outcome after surgery are subject to several limitations: there is a tendency to operate on patients with no light perception, some case series include patients with direct optic nerve injury, many patients are also treated with steroids in high dosage, and there is an inherent difficulty defining improvement when the first assessment is made at the bedside and the final assessment in the clinic. Retrospective reports of the use of high-dose steroids are also subject to similar criticisms.[14–16]

The natural history of traumatic optic neuropathy has not been studied prospectively, but several authors have reported that spontaneous recovery may occur in at least a third of patients.[17–19] There has been no randomised, placebo-controlled study of patients with indirect optic nerve injury. A study of current practice in 16 countries was carried out between 1994 and 1997.[20] A total of 133 patients (127 unilateral, six bilateral) who were initially assessed within 3 days of the optic nerve injury were treated with corticosteroids, optic canal decompression surgery, or observed without treatment. At least 1 month of follow-up was required for inclusion. Nine patients were untreated, 85 treated with corticosteroids, and 33 with decompression surgery. Visual acuity increased by 3 or more lines in 32% of the surgery group, 57% of the untreated group, and 52% of the steroid group. More patients whose initial vision was no light perception were treated with surgery. After adjustment for the initial vision, there were no significant differences between any of the treatment groups. There was no indication that the dose or timing of corticosteroid treatment or the timing of surgery was associated with an increased probability of visual improvement. The number of patients studied was sufficient to detect major differences between the treatment groups. Therefore, this study has not defined a standard protocol of treatment for indirect optic nerve injury.

High-dose steroids may have serious side effects; optic nerve decompression surgery may entail further complications as a result of damage to neighbouring structures in the orbit and region of the sella. In view of the uncertainties about the value of intervention, particularly after 8 h, specific treatment may not be indicated for many patients with indirect optic nerve injury. It is doubtful that a randomised controlled clinical trial of the use of high-dose steroids or decompression surgery will be undertaken in this condition. Measures to avoid injury and research to establish better neuroprotective strategies offer the best hope of progress in the future.

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