A 35 year old man presented to the emergency department after a fall of approximately six feet from a ladder. A witnessed confirmed loss of consciousness for a period of one to two minutes. He had no past medical history of note and had no visual problems before the fall.

On arrival in the emergency department the patient was complaining of severe headache, nausea, and poor vision in his right eye.

On examination the primary survey was normal and his GCS was 15. Secondary survey revealed a small occipital hae-matoma, but no evidence of injury to the face. Eye movements and fundi were normal. Visual acuity in the left eye was 6/6 with a normal pupillary response to light, but visual acuity in the right eye was 6/60 with a right afferent pupillary defect. The remainder of the secondary survey was normal.

Computed tomography of brain and orbits showed an undisplaced fracture through the floor of the right optic canal with no narrowing of the canal itself. It also showed an undisplaced fracture involving the greater wing of the right sphenoid extending into the lateral wall of the sphenoid sinus. There was no haematoma of the soft tissues of the right orbit.

The skull radiograph (fig 1) and relevant CT scan (fig 2) are shown.

He was referred to ophthalmology and a diagnosis of traumatic optic neuropathy was made. He was treated with high dose methylprednisolone using the National Spinal Cord Injury Study protocol. On discharge visual acuity in the right eye remained 6/60 and follow up at one month showed no improvement.

**REFERENCES**


9 Taylor SL. Sudden visual loss after closed head injury: a rare cause of sudden visual loss after trauma. The aetiology and management are discussed.


**DISCUSSION**

This phenomenon was first described by Hippocrates over 2000 years ago. It was defined by Walsh and Hoyt as “traumatic loss of vision without external or initial ophthalmoscopic evidence of injury to the eye or its nerve”. In closed head injury the incidence is approx. 0.5% to 5% and is more common with frontal impact. Spontaneous recovery of vision occurs in less than 50%.

The optic nerve can be divided into the intraocular portion and the intraorbital/intracranial portion. Direct impact to the eye affects the intraocular portion and immediate loss of vision suggests laceration, avulsion, or severe contusion of the...
nerve, which normally results in ischaemic necrosis and permanent blindness. Indirect impact, as in this case, affects the nerve proximal to the intraocular portion. The force is transmitted to the optic nerve and vascular supply and there are three potential mechanisms of visual loss:

- Nerve injury: direct or indirect
- Vascular supply can be occluded, severed, or compromised by spasm
- Compression of the vascular supply or nerve

Immediate visual loss tends to occur with the first two mechanisms and is normally irreversible. However, focal concussive injury of the optic nerve may result in immediate visual loss and this may improve. Compression of the nerve or vascular supply more commonly presents as delayed and progressive visual loss and this is potentially reversible. These mechanisms may overlap and it is therefore important to search for potentially reversible causes.

There are three management options:

- No intervention
- Intravenous methylprednisolone
- Surgical decompression

The use of methylprednisolone is based on the results of the second National Acute Spinal Cord Injury Study. Methylprednisolone is thought to have antioxidant properties, thus limiting free radical damage. It is also believed to reduce traumatic oedema and limit vasospasm. However, there is no conclusive evidence of significant benefit in cases of traumatic optic neuropathy.

Surgical decompression is believed to reduce oedematous compression of the optic nerve and vascular supply within the optic canal. Again, there is no conclusive evidence of benefit in the empirical treatment of traumatic optic neuropathy by surgical decompression.

The main problem in management seems to be the diversity of aetiologies, which precludes one definitive treatment for all cases. Unfortunately the International Optic Nerve Trauma Study failed to recruit enough patients to continue as a randomised controlled trial and thus the debate on treatment continues.

As prognosis is poor, each case should be judged individually and the management discussed with a specialist.

Emergency department management:

- Recognition of the problem
- Prompt discussion with an ophthalmology specialist
- Early computed tomography to define the exact nature of the injury and identification of those patients who may benefit from surgical intervention, for example, optic canal fractures with bony impingement and/or severe swelling.
- Commencement of intravenous methylprednisolone in the emergency department if recommended by a specialist.

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**Figure 2**  
Computed tomography of the brain showing fluid in sphenoid and ethmoid sinuses and the fracture of the sphenoid wing.

**REFERENCES**