

Management of traumatic optic neuropathy – a study of 23 patients

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Abstract

Twenty three patients with traumatic optic neuropathy were managed by medical and surgical treatment as follows. High dose intravenous steroids were initiated in all patients. If visions did not improve significantly after 24 to 48 hours decompression of an optic nerve sheath haematoma by medial orbitotomy and neurosurgical decompression of the optic canal were considered based on computed tomographic scan findings. Nine of 16 patients who received steroids only showed significant improvement. One of three showed improvement on optic nerve decompression after steroid failure; three or four showed improvement with combined optic nerve sheath decompression by the medial orbitotomy and decompression of the optic canal by frontal craniotomy. A lucid interval of vision after injury and an enlarged optic nerve sheath were associated with an improved prognosis. Five of the 23 patients had a lucid interval and all five had a final improved vision, while only five of 18 patients without a lucid interval improved. Similarly seven of the nine with an enlarged optic nerve sheath showed improvement while only three of 10 patients (three bilateral cases) who presented with no light perception improved with medical and surgical treatment. While a prospective controlled study of the management of traumatic optic neuropathy is necessary this preliminary study suggests that treatment of traumatic optic nerve sheath haematoma by optic nerve sheath decompression should be considered in selected patients.

Although the natural history of traumatic optic

neuropathy is unknown recent studies suggest that high dose steroids, and even surgical decompression of the optic canal, may restore vision in selected patients.¹⁻⁷ While visual recovery after decompression of an enlarged optic nerve sheath due to traumatic haemorrhage has been reported this treatment has received little attention in the literature.⁸⁻¹¹ The results of 23 patients who received high dose steroids and underwent decompression of the optic nerve sheath and the optic canal are presented.

Patients and methods

All patients in this series had loss of vision after trauma with an afferent pupillary defect in the involved eye and were treated within the first 48 hours of injury. According to the protocol at UMD-NJ Medical School, a regional trauma centre, all patients received a complete ophthalmic examination by one of us. Patients with penetrating ocular injuries or optic nerve avulsion were excluded from the study. All patients received an initial 1 g loading dose of Solu Medrol (methylprednisolone) and then 250 mg intravenously every 6 hours for 72 hours after medical clearance by the trauma team. Patients with increased intraorbital pressure due to haemorrhage were treated by emergency lateral canthotomy with drainage of subperiosteal or intraorbital haemorrhage. High resolution brain and orbital computed tomography (CT) scans were obtained while treatment was instituted. Thin 1.5 cm sections through the intraorbital and intracanalicular optic nerve were obtained to define 'surgically treatable pathology'; enlarged optic nerve sheath with presumed intrasheath haemorrhage; or narrowing of the optic canal or orbital apex by bone spicules. If vision improved

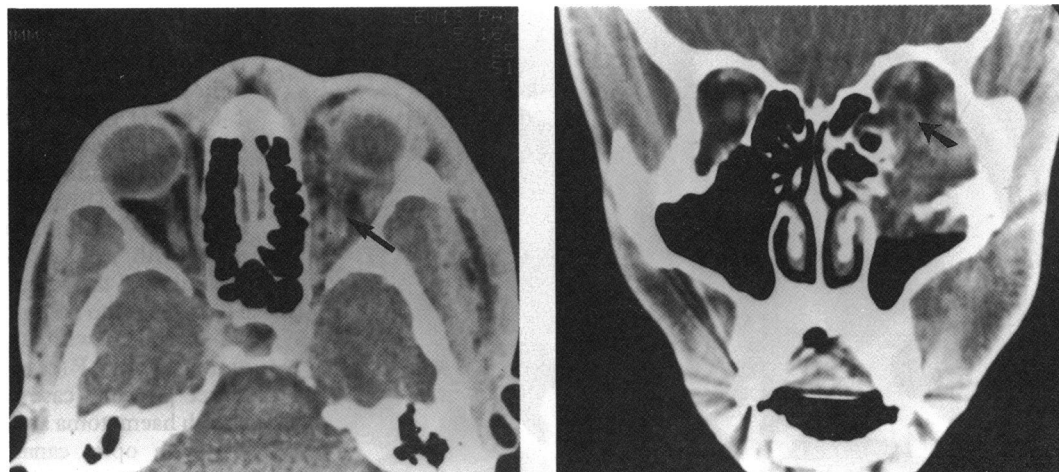


Figure 1 (Case 1) Enlarged optic nerve sheath (arrow) shown of axial (left) and coronal (right) CT scan. Note 'ring' of increased soft tissue density corresponding to blood in the subarachnoid space that surrounds the relatively lucent optic nerve. Patient had return of vision to 20/25 with intravenous steroids and no surgery.

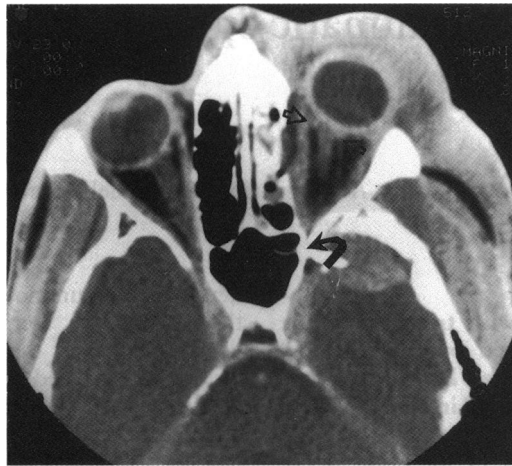
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Figure 2 (Case 23) Lateral wall impinges on orbital apex (curved arrow). The retrobulbar portion of the nerve appears thickened (straight arrows). Patient underwent elevation of the lateral wall of the orbit and craniotomy with decompression of the root of the optic canal. The optic nerve was markedly shrunken at the level of the canal; the sheath contained a paste-like substance. The patient had no return of vision.



over the first 24 to 72 hours steroids were continued for a total of 5 to 7 days and were then tapered. If vision did not improve after 24 to 72 hours of steroids decompression of an optic nerve sheath by medial orbitotomy and decompression of the optic canal with removal of apical orbital bone fragments by frontal craniotomy were considered if on CT scan: (1) the optic nerve sheath appeared enlarged by presumed intrasheath haemorrhage; or (2) the optic canal or optic nerve at the orbital apex appeared compressed by bone spicules (Figs 1-3). If vision did not improve and no 'surgically treatable pathology' was present on CT scan steroids were gradually tapered after 3-4 days. Patients with medical problems and, early in the study, those with unilateral involvement were not always considered surgical candidates.

SURGICAL TECHNIQUE

In all patients the decompression of the optic nerve sheath was performed by a conjunctival approach under general anaesthesia similar to that described for pseudotumour cerebri.^{12,13} A 270 degree conjunctival peritomy was performed. The superior and inferior rectus muscles were isolated on 4-0 silk traction sutures. The medial rectus muscle at its insertion was sutured with a double armed 5-0 vicryl suture that was locked

Figure 3 (Case 19) Axial CT scan shows enlarged nerve (arrows) due to sheath haematoma behind globe.



at each edge of the muscle; the muscle was then cut at its insertion and detached from the globe as if a standard muscle recession were to be performed. The two free arms of the vicryl suture were taped without tension with a 1/4 inch steri-strip to the patient's brow and cheek on the opposite side. Care was taken to avoid traction and damage to the medial rectus muscle. Multiple running bites were taken through the stump of the muscle with a 4-0 silk traction suture. A Schepens retractor or thin ribbon retractor was placed in the medial orbit between the globe and the medial rectus muscle. Cottonoids, cotton tip applicators, and suction facilitated exposure of the optic nerve sheath through the operating microscope. A no 11 blade was used to make three 2-3 mm longitudinal incisions into the nerve sheath away from superficial blood vessels. The medial rectus muscle was resutured into position after the traction sutures were removed and the conjunctival peritomy closed.

The neurosurgical procedure was then performed through a coronal flap by the neurosurgeons (AK, MS). The optic canal was unroofed and any bone spicules that impinged on the nerve were removed. All patients continued to receive steroids for 72 hours after surgery. For those with improvement in vision after surgery the steroids were continued for a total of 5 to 7 days and then tapered.

Results

SURGICAL FINDINGS

At surgery a swollen haemorrhagic nerve sheath was identified in three of the seven patients and a clear yellowish fluid was recovered after the incision into the nerve sheath. In patient no 17, no blood or fluid was recovered at the time of the optic nerve sheath decompression yet the vision returned to 20/25.

Case no 23 presented with NLP vision; the entire lateral wall of the orbit was displaced posteromedially against the optic nerve (Fig 2). At the time of neurosurgical decompression of the optic nerve at the optic canal the nerve sheath was filled with a whitish pink paste.

VISUAL RECOVERY

Nine of 16 patients showed significant improvement on steroids alone; seven (case nos 1-6, 10) of the nine who showed improvement had an enlarged optic nerve sheath (Table 1). Case nos 12 and 16 had no significant pathology on CT scan. Three of the nine patients required immediate bedside canthotomy prior to treatment with steroids for increased intraorbital pressure. Of the seven patients (case nos 7, 8, 9, 11, 13-15) who had no improvement on steroids six presented with no light perception (NLP) and one with LP (case no 9) in the involved eye. Three patients (case nos 7-9) with optic nerve sheath haematoma and two patients (case nos 13, 15) with optic canal fractures did not have surgery for a variety of reasons including medical contraindications.

One of three patients who did not respond to

Table 1 Patients with traumatic optic neuropathy treated with steroids alone

Case no/age/sex	Type of injury	Initial/final vision	CT finding
1. 26, M	Blunt trauma, bat	CF 2 ft/20/25	Optic nerve sheath haematoma
2. 44, M	Blunt trauma	20/70–2/20/20–2	Optic nerve sheath haematoma
3. 15, M	Fall from bicycle	CF 1 ft/20/400	Optic nerve sheath haematoma
4. 27, M	Blunt trauma	20/200/20/40	Optic nerve sheath haematoma
5. 18, M	Motor vehicle accident	CF 2ft/20/30	Optic nerve sheath haematoma
6. 58, M	Sinus endoscopy, surgery	CF 3 ft/20/25	Optic nerve sheath haematoma
7. 42, M	Blunt trauma, fist	NLP/NLP	Possible optic nerve sheath haematoma
8. 13, M	Knife to orbit	NLP/NLP	Optic nerve sheath haematoma
9. 90, M	Motor vehicle accident	LP/LP	Optic nerve sheath haematoma
10. 54, M	Blunt trauma	CF 3 ft/20/20	Possible optic nerve avulsion Optic nerve sheath haematoma, left tripod fracture
11. 27, M	Gunshot right orbit	NLP/NLP	No significant pathology*
12. 53, M	Motor vehicle accident	20/400/20/25	No significant pathology
13. 36, M	Fist	NLP/NLP	Apical medial optic canal fracture
14. 27, M	Gunshot left orbit	NLP/NLP	No significant pathology
15. ?, M	Motor vehicle accident	NLP/NLP	Optic canal fracture
16. ?, M	Blunt trauma	20/800/20/40	No significant pathology

*Central retinal artery occlusion.

CF=counting fingers, NLP=no light perception, LP=light perception.

Table 2 Characteristics of patients with traumatic optic neuropathy treated with steroids and optic nerve sheath decompression

Case no/age/sex	Type of injury	Initial/final vision	CT finding
17. 28, M	Knife to orbit	CF 1 ft/20/25	Optic nerve sheath haematoma
18. 18, M	Gunshot to orbits	Bilateral NLP/NLP	Right optic nerve sheath haematoma*
19. 27, M	Motor vehicle accident	NLP/NLP	Right optic nerve sheath haematoma

*Left optic nerve avulsed and not treated; right optic nerve sheath haematoma decompressed.

Table 3 Characteristics of patients with traumatic optic neuropathy treated with steroids, optic nerve sheath decompression, and neurosurgical decompression of the optic canal*

Case no/age/sex	Type of injury	Initial/final vision	CT finding
20. 12, M	Gunshot to orbits	NLP both eyes/NLP OD 20/200 OS	Right optic nerve avulsion, left optic nerve sheath haematoma with bone spicules at orbital apex
21. 32, M	Gunshot to orbits	NLP both eyes/NLP OD HM OS	Right optic nerve avulsion, left optic nerve sheath haematoma with bone spicules at orbital apex
22. 34, F	Motor vehicle accident	NLP both eyes/ improved pupillary reaction both eyes†	Left optic nerve sheath haematoma, bilateral optic canal fracture
23. 28, M	Blunt trauma	NLP/NLP	Optic nerve sheath haematoma, apical bone directly impinging on optic nerve

*In each case finding on CT was treated surgically. Patients with presumed avulsion were not treated.

†Unable to assess exact vision due to mental status after injury.

HM=hand movements.

steroid treatment showed improvement on optic nerve sheath decompression by medial orbitotomy of an enlarged sheath (Table 2).

Three patients who did not respond to steroids had optic nerve sheath decompression; one of the patients improved to 20/25 (case no 17) (Table 2). Four patients who did not respond to steroids had combined optic nerve sheath decompression and neurosurgical decompression of the optic canal; three showed improved vision (Table 3).

All five patients with a lucid interval of vision after the injury had a final improved vision while only five of 16 patients without a lucid interval improved. Seven of 10 patients (three bilateral cases) who presented with NLP had no improvement with any treatment. Three (four eyes) of the 10 with improvement had combined optic nerve sheath decompression and neurosurgical decompression of the optic canal.

No medical or surgical complications occurred in this series of 23 patients.

Discussion

The present data suggest the efficacy of steroids and possibly surgery in selected patients with traumatic optic neuropathy. All 23 patients initially received high dose steroids. Nine of 16 patients showed significant improvement on steroids without surgery. In patients without visual improvement after 24 to 48 hours of intravenous steroids surgery was considered if: (1) an enlarged optic nerve sheath with presumed intrasheath haemorrhage decompression of the retrobulbar portion of the optic nerve sheath, or (2) a narrowed optic canal or orbital apex by bone spicules were present on CT scan. To date the role of optic nerve fenestration in the treatment of traumatic optic neuropathy has not been clearly elucidated.^{8–10}

One of three patients showed visual improvement after optic nerve sheath decompression. Three of four showed improvement with combined optic nerve sheath decompression and neurosurgical decompression of the optic canal by neurosurgical frontal craniotomy. One patient despite such combined surgery showed no improvement in vision. Decompression of the optic canal may alternatively be performed by medial orbitotomy with a skin incision or by a transantral approach.^{2,14–16} The latter approach may have less morbidity than a craniotomy; however there were no complications in this series and frontal craniotomy allows complete decompression of the optic canal and removal of apical orbital bone fragments.

Optic nerve sheath haematomas are relatively rare and are difficult to demonstrate radiographically. For these reasons there are only a few reported cases of decompression of an enlarged optic nerve sheath.^{8–11} In this series fluid was not always recovered at the time of optic nerve sheath decompression and in one such patient (case no 17) the final vision was 20/25. The findings may be due to (1) oedema that involves the entire nerve and not just the subarachnoid space, or (2) the collection of blood may be focal away from the surgical site. Magnetic resonance imaging may be helpful in localising blood.

The natural history of traumatic optic neuropathy is unknown, and even the exact mechanism by which corticosteroids reduce optic nerve injury is unclear.^{1–7} The rationale for treatment is as follows. Steroids probably decrease the intraneural or extraneural oedema and relieve compression of the nerve fibres.⁴ By reducing vasospasm steroids may also limit contusion necrosis of the nerve.⁵

The effect of steroids on the resorption of an optic nerve sheath haematoma is also unclear. Surgical evacuation of non-resorbing nerve sheath haematoma should be considered when initial treatment with steroids fails to improve vision.^{8–11} Timely surgical optic nerve sheath decompression is initiated in order to avoid irreversible damage to the axons of the optic nerve compressed by the subarachnoid blood within the swollen nerve sheath.

The data show that a favourable prognosis is associated with (1) a lucid interval, and (2) an enlarged optic nerve sheath. All five patients with a lucid interval of vision after the injury had

a final improved vision. Seven of the nine who showed improvement with steroids had an enlarged optic nerve sheath. Poor prognosis is associated with (1) initial NLP vision, (2) no lucid interval, and (3) displacement of large bone fragments against the optic nerve (case no 23). Seven of 10 patients (three bilateral cases) who presented with NLP had no improvement with any treatment. Only five of 15 patients who did not have a lucid interval ultimately improved.

Patients who present without a lucid interval and with NLP vision should be evaluated and treated. In the three patients (case nos 20–22) who presented with NLP and subsequently improved, the final vision was 20/200, hand motions, and indeterminable in the third patient due to intracranial injuries. The latter patient had bilaterally improved pupillary responses.

In summary optic nerve sheath decompression alone or combined with decompression of the optic canal may be indicated in selected patients who fail to respond to high dose intravenous steroids. The definitive role of surgery in the management of traumatic optic neuropathy remains unclear until a protocol is adopted on a multicentre level. Initiation of steroids for 24 to 48 hours affords sufficient time for consultations, radiological studies, and conference with the patient and family prior to any surgical intervention.

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