Objective: To assess the visual outcome in cases of traumatic optic neuropathy treated with a combined therapy protocol of methylprednisolone injections and endoscopic optic nerve decompression.

Design: Prospective, nonrandomized study.

Setting: Academic tertiary care referral center.

Patients: The study included 44 patients with posttraumatic indirect optic nerve injury.

Main Outcome Measure: Visual acuity.

Results: Visual improvement was achieved in 31 patients (70%) when treatment was initiated within 7 days of injury, whereas only 10 patients (24%) showed improvement when the treatment was started after more than 7 days. The time lapse after injury and treatment, degree of visual loss, and computed tomographic evidence of canalicular and pericanalicular fractures were found to be significant prognostic factors.

Conclusions: Endoscopic optic nerve decompression is a minimally invasive procedure that does not cause any adverse cosmetic effects. The risk-benefit ratio suggests that the combined therapy protocol of methylprednisolone injections and endoscopic optic nerve decompression results in a better visual outcome, without any major risks.


OPTIC NERVE damage, which can occur in 5% of cases after head injury, is classified into direct and indirect injuries.1 Direct injuries, which are caused by a penetrating injury to the area of the optic nerve, result in poor visual recovery. Indirect injuries are caused by forces that are transmitted at different levels after blunt head trauma.2 The most common site of indirect optic nerve injury is the optic canal.3 In cases of traumatic optic nerve injury, the increased intracanalicular pressure causes vascular compromise, with ischemia and interruption of the neurofeedback mechanism, leading to blindness. Decompression of the optic nerve by partial removal of the optic canal wall can relieve intracanalicular pressure and angular strangulation and reestablish nerve function.4 Therefore, various surgical approaches, such as the transfrontal craniotomy5 and extranasal transethmoidal,6 transantral ethmoidal,7 lateral facial,8 and endoscopic procedures, were developed to access the optic nerve.9,10 The endoscopic approach is used because the optic canal is related to the lateral wall of the sphenoidal sinus.4

METHODS

The present prospective study of traumatic optic neuropathy was conducted on 44 consecutive patients at the Postgraduate Institute of Medical Education and Research, Chandigarh, India, from October 2000 to March 2002. All patients with indirect optic nerve injuries were treated with injections of methylprednisolone (30 mg/kg per day). During steroid therapy, visual acuity was monitored periodically to assess improvement or worsening of the patients' vision. Computed tomography of the paranasal sinuses and orbit was performed to evaluate the extent of the injuries. Endoscopic optic nerve decompression was performed in cases in which there was (1) failure of improvement of vision after 72 hours of methylprednisolone therapy; (2) progressive visual loss during steroid therapy; or (3) total blindness with computed tomographic evidence of optic nerve compression.

The procedure was performed using 0° and 30° nasal telescopes with the patients under general anesthesia. After ethmoidectomy and sphenoidotomy, the bulge that was caused by the internal carotid artery and optic nerve was identified in the lateral wall of
the sphenoidal sinus. The medial wall of the optic canal was thinned out with a microdrill and removed with a microcurette. The annulus of Zinn and the optic nerve sheath were not incised in any of the cases. After surgery, the patients were observed for visual acuity and received regular follow-up after intervals of 1 week, 1 month, and 3 months. Follow-up included testing of visual acuity, funduscopic, and field charting.

For assessment of visual outcome, all patients were categorized into the following 3 groups: group 1 included 11 patients who received treatment within 3 days of injury; group 2 included 12 patients who received treatment within 4 to 7 days; and group 3 included 21 patients who received treatment after 7 days.

The patients’ vision was considered to have improved if there was an increase of 3 lines or more on the Snellen visual chart or if their vision had increased from nonperception of light to perception of light, from perception of light to hand motion, or from hand motion to finger counting. Treatment was considered to be successful when vision improved to at least 3/60, based on the World Health Organization’s definition of blindness.

**RESULTS**

This prospective, nonrandomized, interventional study included 44 patients with a unilateral optic nerve injury who had completed a minimum follow-up of 3 months (range, 3-14 months; mean, 3.8 months). The mean±SD age of the patients, who were predominantly male (93%), was 28.6±11.0 years. Car crashes were the most common cause of trauma (63%), followed by falls and assaults (Figure 1). Thirty-six patients presented with complete loss of vision, and 8 with diminished vision. Of the 36 patients with complete loss of vision, the mode of onset was sudden in 18 (50%), gradual in 8 (22%), and unknown in 10 (28%); of the 8 patients with diminished vision, the mode of onset was sudden in 3 (38%) and gradual in 5 (62%). Thirty-three patients (75%) presented with loss of consciousness, 22 (50%) with epistaxis, 3 (7%) with watery nasal discharge, 3 (7%) with seizures, and 10 (23%) with associated injuries, eg, head injury, maxillofacial injury, and long-bone fracture (Figure 2). A relative afferent pupillary defect was detected in 40 patients (91%); it could not be elicited in the other patients due to orbital apex syndrome (n=3 [7%]). Initial fundus evaluation revealed a normal optic disc in 40 patients (91%), and computed tomography revealed a canalicular fracture in the optic canal in 15 patients (34%).

All 44 patients were treated with injections of methylprednisolone. The vision of 10 patients improved with steroid therapy alone, and they were discharged on an oral regimen of prednisolone (1 mg/kg per day for 11 days, followed by a tapering of the dosage). Thirty of 34 patients whose vision failed to improve with methylprednisolone therapy underwent endoscopic optic nerve decompression after the advantages and risks of the surgery were explained to them. Surgery was not performed in 4 patients because of optic atrophy. During surgery, optic canal fracture was seen in 15 patients (50%) and compression of the optic nerve was seen in 12 patients (40%); the optic nerve was edematous and lacerated in 9 and 2 of the 12 patients, respectively, and an intrasheath hematoma was found in 1 patient. Carotid bleeding occurred in 1 patient (3%) during surgery, but was successfully controlled, and synechia occurred after surgery in 6 patients (20%). There was no worsening of vision after surgery in any patient.

After combined therapy, vision was improved in 8 (73%) of 11 patients in group 1; in 8 (67%) of 12 patients in group 2; and in only 5 (24%) of 21 patients in group 3. After surgery, 6 (54%), 6 (50%), and 3 (14%) of the patients in groups 1, 2, and 3, respectively, regained their vision (>3/60) (Figure 3).

The significant prognostic factors were time lapse between injury and treatment, degree of visual loss, and computed tomographic evidence of canalicular and pericanalicular fracture. Visual improvement was achieved in 16 (70%) of 23 patients in whom treatment was started before 7 days after injury, whereas there was improvement in only 5 (24%) of 21 patients in whom treatment was started after 7 days. The difference between the 2 groups was significant (χ² = 8.30; P<.01). Based on the degree of visual loss, 13 (36%) of 36 patients with complete loss of vision demonstrated improvement, whereas all 8 patients with residual vision showed improvement (χ² = 8.30; P<.01). Optic canal fracture was documented on computed tomographic scans in 15 patients,

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**Figure 1.** Mechanism of trauma. CC indicates car crashes.

**Figure 2.** Clinical presentation of traumatic optic neuropathy. LOC indicates loss of consciousness; CSF, cerebrospinal fluid.

**Figure 3.**
3 (20%) of whom showed visual improvement ($\chi^2 = 5.43; P<.05$) (Figure 4).

**COMMENT**

Decreased visual acuity was the only morbidity associated with traumatic optic neuropathy. The only ocular abnormality that was diagnostic of optic nerve injury was the relative afferent pupillary defect, which is elicited by swinging a flashlight. In the present study, the ocular manifestation that was most commonly associated with optic nerve injury was subconjunctival hemorrhage, with no immediate changes in the optic disc. The ocular manifestations that were observed in our study are similar to those reported in other studies. The incidence of optic canal fracture in traumatic blindness has been variously reported from 6% to 92%. In our series, computed tomography revealed optic canal fracture in 15 patients (34%), all of whom had a poor visual outcome even after treatment with methylprednisolone and endoscopic optic nerve decompression.

The treatment of traumatic optic neuropathy includes keeping the patients under observation, administering corticosteroid therapy, or performing optic nerve decompression with or without steroid therapy. There is no available optimal management protocol, as most of the published data are either retrospective or presented in case reports. The International Optic Nerve Trauma Study, which was undertaken to formulate an optimal management protocol, failed because of the limited number of eligible patients. The majority of published data do not clearly define the criteria of visual improvement; some studies define improvement as an increase in 1 to 3 lines in visual acuity. Methylprednisolone therapy is advocated as the initial treatment of choice because of its neuroprotective mechanism. An improvement of 44% to 82% after steroid therapy has been reported. The largest series on steroid treatment was published by the International Optic Nerve Trauma Study, which reported an improvement in 54% of patients after 3 months of follow-up. The intracanalicular portion of the optic nerve is the site most frequently involved in indirect optic nerve injury. The rationale of optic nerve decompression involves partially removing the optic canal to decompress the nerve within the canal in order to limit the damaging effect of compression and to reestablish nerve function. The documented improvement after optic nerve decompression with or without steroid therapy varies from 27% to 82%. Fujitani et al reported a 48% improvement in a large series of patients with optic nerve decompression. Kountakis et al reported an improvement of 82% after surgery in their series of 17 patients, whereas the overall improvement was 74% after high-dose steroid therapy and surgery.

Of the 44 patients in our study, 10 showed improvement after steroid therapy and 11 showed improvement after endoscopic optic nerve decompression, with an overall improvement of 48%. Statistical analysis revealed total blindness, canalicular fracture, and late presentation as poor prognostic factors. In cases of total blindness with computed tomographic evidence of optic nerve compression, early surgery should be undertaken. In cases with residual vision, with or without evidence of canalicular fracture, it is prudent to wait for the results of steroid therapy before optic nerve decompression is undertaken. In cases in which the patients present late, with poor prognostic factors, the risk-benefit ratio must be analyzed before any treatment is offered. The combined therapy protocol results in a better visual outcome when treatment is started within 7 days of injury.

In the past, external transethmoidal optic canal decompression was the procedure of choice for the treatment of extracranial optic nerve decompression, but now endoscopic optic nerve decompression is preferred because it is less invasive and does not cause cosmetic problems. The endoscopic approach offers many advantages over the traditional approaches, eg, decreased morbidity, preservation of olfaction, rapid recovery time, lack of adverse cosmetic effects, and less operative stress. As with any surgical procedure, this approach also has some disadvantages and limitations. An iatrogenic injury to the nerve fascicle can occur during any surgical procedure that involves the optic nerve, whereas canalicular endoscopic optic nerve decompression carries additional risks and requires the skills of an experienced endoscopic surgeon. Conchal pneumatization is one of the absolute contraindications to this procedure. Endoscopic optic nerve decompression is a minimally invasive procedure and has
proved to be safe and cosmetically acceptable when performed by an experienced surgeon.

**CONCLUSIONS**

The combined therapy protocol of methylprednisolone injections and endoscopic optic nerve decompression should be offered to patients who present within 7 days of injury. The injections of methylprednisolone should begin as soon as traumatic optic neuropathy is suspected or diagnosed. Endoscopic optic nerve decompression is indicated whenever there is (1) failure of improvement of vision after 72 hours of methylprednisolone therapy; (2) progressive visual loss during steroid therapy; or (3) total blindness with computed tomographic evidence of optic nerve compression.

 Patients who present soon after injury must be treated with a combined therapy protocol for a better visual outcome. Endoscopic optic nerve decompression was found to be safe and free from any mortality or morbidity in expert hands.

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**REFERENCES**