

Lateral orbitotomy for traumatic optic neuropathy and traumatic ophthalmoplegia: Is it beneficial?

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Abstract

This was a retrospective study of 12 eye injuries concomitant with closed head injury, that were treated by the authors in Sanglah General Hospital, Bali during 2008. The purpose of this study was to evaluate the efficacy of lateral orbitotomy for nerve compression in eye injuries. Optic nerve compression at the optic canal can cause traumatic optic neuropathy; and nerve compression of 3rd, 4th and 6th nerves at the superior orbital fissure can cause traumatic ophthalmoplegia. Optic nerve decompression with steroids or surgical interventions or both have therefore been advocated to improve visual prognosis in traumatic optic neuropathy. To date it is not known whether lateral orbitotomy is beneficial in these traumatic injuries. There were 12 patients in this study, consisting of 11 males and 1 female with diagnosis of traumatic optic neuropathy in 10 patients and traumatic ophthalmoplegia in 2. Lateral orbitotomy procedures were done and all patients were also given high doses of steroids preoperatively. The mean visual improvement ratio was 43.3 ± 22.3 . Ten patients improved (83.3 %) and 2 patients did not. There were no clinical deterioration and side effects from the operative procedure. This study suggests that lateral orbitotomy may possibly be beneficial as treatment for traumatic optic neuropathy and ophthalmoplegia.

INTRODUCTION

Recently, head trauma in developing countries is increased due to many traffic accidents that involve head and adjacent structures, i.e. eye, nose, jaw, ear and the neck. Traumatic optic neuropathy is a devastating potential complication of closed head injury. The hallmark of an optic neuropathy, traumatic or otherwise, is a loss of visual function, which can manifest by subnormal visual acuity, visual field loss, or color vision dysfunction. The presence of an afferent pupillary defect strongly suggests a prechiasmal location of the injury and is necessary to substantiate the diagnosis of traumatic optic neuropathy. Vision loss associated with traumatic optic neuropathy can be partial or complete and temporary or permanent.¹

Traumatic ophthalmoplegia is also a potential complication that should be suspected if injury affects the edge of the orbital rim. Trauma can break the thin bone that borders the orbital structures and fracture of the superior orbital fissure can cause neural entrapment. The third, fourth and sixth nerves can be compressed by bone fragments and cause partial or total paralysis of the eye movement.

In the early 1900s, transcranial unroofing of the optic canal was the surgical procedure of

choice for traumatic optic neuropathy treatment. In the 1920s, Sewell performed a transthemoidal optic canal decompression by removing the lamina papyracea and medial wall of the optic canal. The lateral approach to the orbit was first proposed in 1889 by Krönlein and modified by Berke in 1953, and by Maroon and Kennerdell in 1976.^{1,2} During this period, systemic corticosteroid treatment was also extended to treatment of traumatic optic neuropathy. Currently, endoscopic optic nerve decompression via an intranasal and transthemoidal or transsphenoidal approach has gained popular support.^{1,3-5}

There are limited published studies on the effect of lateral orbitotomy for traumatic ophthalmoplegia and optic neuropathy. However, use of methylprednisolone and optic decompression alone have been described.¹⁻³ In this study, we evaluate the experience of lateral orbitotomy for traumatic optic neuropathy and traumatic ophthalmoplegia in the Sanglah General Hospital, Bali.

METHODS

This was a retrospective study, based on 12 eye injuries who also had closed head injury, treated by the authors in Sanglah General Hospital, Bali

during the year 2008. The demographics of the patients, types of injury, visual acuity scores before and after surgery, visual improvement ratio, eye movements and side effects of surgery were recorded. The side effect looked for were visual acuity deterioration, worsening of eye movement, dural tears, collapse of the eye globe or wound infection.

The ICD-9-CM Visual Acuity Score was used for evaluation of the visual ability of each patient (Table 1). The visual improvement ratio was evaluated by a formula as below.

$$\frac{\text{Postoperative visual acuity score} - \text{Preoperative visual acuity score}}{\text{Preoperative visual acuity score}} \times 100\%$$

Operative Technique

In this study, before surgery was conducted, all patients received a high dose steroid bolus intravenously (methylprednisolone 30mg/kg) which was continued by maintenance dose

(methylprednisolone 2mg/kg TID i.v.) until 3 days after the surgery. Under general anesthesia, Lazy S-shaped skin incision was used and curved up to the eye brow and then posteriorly along the upper margin of the zygomatic arch for approximately 35 to 40 mm from the lateral canthus to avoid to damage the frontal branches of the facial nerve. After skin incision, the fascia of the temporal muscle was incised along the skin incision, the temporal muscle was dissected subperiostally and retracted posteriorly to expose the lateral orbital bone. Pneumatic Cranial perforator (Aesculaps

was used to open a bone window at the pterion. The hole connected the intraorbital space to the intracranial space. The periosteum of the edge of the orbital rim was dissected, and carefully the hole was enlarged using 2 mm Kerrison rongeur. The

Table 1. Visual acuity score and impairment classes

Impairment classes (Based on ICD 9-CM)		Visual acuity (1 meter notation)	Visual acuity score
Near normal vision	Range of normal vision	1/1	100
		1/1.25	95
Low vision	Near normal vision	1/1.6	90
		1/2	85
		1/2.5	80
		1/3.2	75
		Moderate	1/4
	1/5		65
	1/6.3		60
	1/8		55
	Severe		1/10
		1/12.5	45
1/16		40	
1/20		35	
Profound		1/25	30
	1/30	25	
	1/40	20	
	1/50	15	
	Near blindness	Near blindness	1/63
1/80			5
1/100 or less			0
Total blindness		No light perception	0

periorbita was dissected from the inner surface of the lateral wall of the orbit. After the lateral orbital wall was removed, the optic canal then unroofed.² In 2 cases, this was followed by superior orbital fissure decompression. After the decompression was completed, homeostasis was established and the periosteum was reapproximated with the anterior temporal duramater. The subcutaneous tissue and skin were closed with Vicryl 3.0 (Ethicon Ltd.,UK).

Postoperatively, all patients were monitored on in the ward and their vital signs checked every 2 hours on the first day and every 6 hours throughout the hospital stay. The visual acuity scores were checked preoperatively and 2 weeks post operatively. The patient was discharged when patient could ambulate, had adequate pain control, and all patients were scheduled for follow-up examination in 3 months.

RESULTS

During the study period 12 patients were admitted, consisting of 11 males and 1 female. The patients were mostly in their 3rd decade or younger (9 patients, 75%), and 4th decade or older

(3 patients, 25%). The diagnoses were traumatic optic neuropathy in 10 patients, and traumatic ophthalmoplegia in 2 patients. In all patients, decompressive lateral orbitotomy surgery was conducted. Decompression for optic nerves only was done in 10 patients and with decompression for other orbital nerves (cranial nerve III, IV and VI) in 2 patients. Ten patients showed improved visual acuity, and 2 patients did not. Both the patients with traumatic ophthalmoplegia had improvement in orbital movement after the surgery. (Figure 1)

The preoperative visual acuity ranged from 0 to 1/6, and postoperative visual acuity ranged from 0 to >3/6. The visual improvement ratio ranged from 0 to 60%, 7 patients (58.3%) had ratio of 60%, 3 patients were 20% and 2 patient had ratio of 0 and 40% respectively. The mean improvement ratio was 43.3 ± 22.3 . None of the patients had clinical deterioration and side effect from lateral orbitotomy, with no patient having deterioration of visual acuity, worsening of eye movement, dural tears, collapse of the eye globe or wound infection.

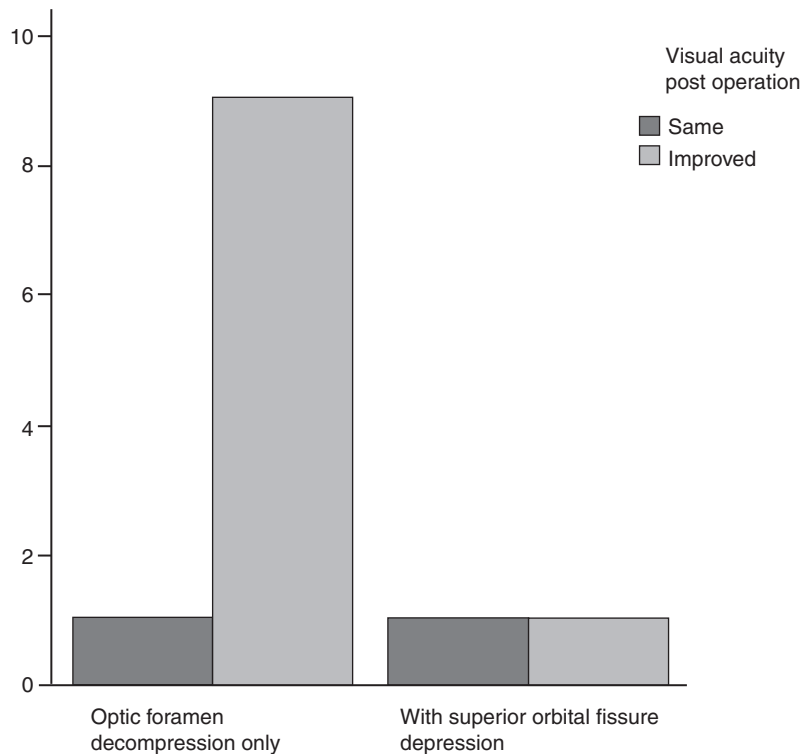


Figure 1. Visual acuity following lateral orbitotomy

DISCUSSION

Trauma can precipitate various pathophysiological conditions that ultimately manifest as visual dysfunction and ophthalmoplegia. Trauma-induced injury to the optic nerve and other orbital nerves can occur anywhere along the nerve's intraorbital-to-intracranial length. Direct traumatic optic neuropathy is the term used when the optic nerve is impinged, crushed, or transected. Indirect traumatic optic neuropathy occurs in the absence of direct optic nerve injury and is more common than direct traumatic optic neuropathy. Beside injury to the optic nerve, there is also possibility of the superior orbital foramen compression by the bone fragment of the fractured lateral orbital rim, which cause partial or total traumatic ophthalmoplegia due to 3rd, 4th and 6th nerves compression.¹⁻³

In the United States, incidence of indirect traumatic optic neuropathy is approximately 2.5% in patients with midface trauma and 2-5% in patients with closed head injury. Traumatic optic neuropathy is most commonly caused by motor vehicle and bicycle accidents (15-75% of cases, depending on the series). Falls (15-50% of cases) are the next most common cause, followed by physical violence and recreational sports. The diagnosis of traumatic optic neuropathy is clinical. Patients with midfacial and cranial trauma should elicit a high index of suspicion for traumatic optic neuropathy. In addition, although 50% of patients with traumatic optic neuropathy present with a visual acuity of light perception or no light perception, nearly 20% of patients have a visual acuity of 20/200 or better, meanwhile there is no report on traumatic ophthalmoplegia.¹

Hippocrates noted the association of trauma just above the eyebrow and gradual vision loss. By the 18th century, the relationship between frontal trauma and vision loss with an absence of ocular injury was well appreciated. In 1879, Berlin described the first pathologic examination of the optic nerve after head trauma. In 1890, Battle first distinguished penetrating direct from non penetrating indirect optic nerve injuries. Historically, observation, medical corticosteroid therapy, or optic canal decompression has been advocated for the treatment of traumatic optic neuropathy.¹

The exact pathophysiology of traumatic optic neuropathy and traumatic ophthalmoplegia are poorly understood. Traumatic optic neuropathy, is an indirect event that occurs during or shortly after blunt trauma to the superior orbital rim, lateral orbital rim, frontal area, or cranium. Elastic

deformation of the sphenoid then allows transfer of the force to the intracanalicular segment of the optic nerve. Contusion of the intracanalicular optic nerve axons and pial microvasculature produces localized optic nerve ischemia and edema. The edematous ischemic axons result in further neural compression within the fixed-diameter bony optic canal, precipitate a positive feedback loop, and trigger the development of an intracanalicular compartment syndrome and the similar pathophysiology probably could explain about traumatic ophthalmoplegia.^{1,3}

Medical or surgical intervention or a combination of both may be indicated for patients with indirect traumatic optic neuropathy and traumatic ophthalmoplegia. Indications for treatment are based on clinical judgment. Absolute indications for intervention, including optic canal decompression and superior orbital fissure, have not been validated by controlled outcome studies; currently, physicians must decide on therapy for traumatic optic neuropathy and traumatic ophthalmoplegia without a consensus on standard of care. In the Cochrane Database of Systematic Reviews, no randomized controlled trials were identified for either the use of steroids alone or with surgical treatment for traumatic optic neuropathy and traumatic ophthalmoplegia. Citing reports of visual recovery rates of 40-60% with conservative management, the decision to proceed with surgery or high-dose steroids alone depends on the clinical judgment and surgical skills of the surgeon as well as informed consent of the patient to appreciate the benefits and risks of both treatments. A recently published study from Iran reported a randomized placebo-controlled trial of the use of intravenous high-dose steroids versus saline in 31 patients with traumatic optic neuropathy; the authors found no statistically significant improvement in visual acuity in the 2 groups.^{1,3,4,6,7}

The use of steroids in the treatment of traumatic optic neuropathy and traumatic ophthalmoplegia should be carried out with special care in patients who are at risk of complication from the drug, eg, those with diabetes mellitus, gastric ulcers, and osteoporosis. In addition, a randomized controlled trial on the use of steroids in patients with acute traumatic brain injury found a higher risk of death in the steroid group, leading investigators to prematurely terminate the trial.⁸ Although the mechanism of higher mortality in the patients who received steroids remains to be elucidated, this should be taken into account in the decision to treat patients with traumatic optic neuropathy

and head injury with steroids.³ The most widely accepted contemporary treatments for traumatic optic neuropathy have included observation, steroids, and surgical decompression, but concerns about the use of steroids in patients with acute brain trauma has led to recent recommendations not to treat traumatic optic neuropathy with steroids.^{9,10,11}

Within the limitations of the study design, the authors conclude that there were useful clinical improvements of visual acuity by the lateral orbitotomy. The role of lateral orbitotomy for traumatic optic neuropathy and ophthalmoplegia, defined as surgical decompression undertaken following steroids treatment remains unclear and controversial. However, this limited studies point to some benefit when this treatment is used as salvage therapy on patients who are not completely blind after steroids therapy failed.

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