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Original article

Endoscopic decompression of the optic canal for traumatic optic neuropathy

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ABSTRACT

Purpose: Traumatic optic neuropathy (TON) is a serious complication of head trauma with the incidence rate of 0.5%–5%. The aim of this study was to investigate the therapeutic efficacy of endoscopic decompression of the optic canal for optic nerve injuries.
Methods: In this study, 11 patients treated in our hospital from January 2009 to January 2015 with the visual loss resulting from TON were retrospectively reviewed for preoperative vision, visual evoked potential (VEP) scan, surgical approach, postoperative visual acuity, complications, and follow-up results.
Results: All these patients received endoscopic decompression of the optic canal. At the 3-month follow-up, the visual acuity improvement rate of the 11 patients was 45.5%. The vision acuity of 2 cases improved from hand movement to 0.08 and 0.3 after operation. Another patient's vision acuity returned to 0.05 compared to light sensation preoperatively. Two cases had finger counting before surgery but they had a vision acuity of 0.4 and light sensation respectively after surgery. However, the other 6 cases' vision did not improve after surgery.
Conclusion: Endoscopic decompression of the optic canal is an effective way to cure TON. VEP could be used as an important reference for preoperative and prognosis evaluation. Operative time after trauma is only a relative condition that may affect the therapeutic effect of optic canal decompression. Poor results of this procedure may be related to the severity of the optic nerve injury.
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Introduction

Currently surgical decompression of the optic canal is the main approach for traumatic optic neuropathy (NOC) in neurosurgery. Theoretically, optic nerve decompression reduces intracanalicular pressure and allows for the removal of any impinging bony fragment, assisting in the re-establishment of nerve function. Surgical decompression has been performed by our neurosurgeons with different surgical techniques (endoscopic, transorbital and transcranial approaches). To explore the safety and effectiveness of endoscopic optic nerve decompression in the treatment of TON, we reviewed 11 cases who presented with TON and underwent surgery in the last 5 years at Department of Neurosurgery, Lanzhou University Second Hospital.

Materials and methods

Patients

This study consisted of 11 patients including 8 males and 3 females with the age ranging from 18 to 67 years and an average of 36 years, who had undergone endoscopic optic canal decompression in our department after head injury over 5-year period (Jan.2009–Jan.2015). Preoperative eyesight examinations found no light sensation in 5 cases, light sensation in one, hand movement in two, finger counting in two, and vision acuity of 0.1 in one. Among the 11 patients, eyesight was found declined immediately after injury in 4 patients, while the eyesight in the other 7 patients became gradually worse.

The interval between injury and operation was 6 h–30 days. High resolution CT (HRCT) scan of the head and visual evoked

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potential (VEP) were performed on all patients at initial presentation. Patients were excluded if they did not receive VEP examination before surgery. The endoscopic optic canal decompression was not performed for patients with serious head trauma (Glasgow Coma Scale <8). The results were analyzed using unpaired *t*-test.

Therapies

General anesthesia and controlled hypotension were introduced in all patients throughout optic nerve decompression. The nasal cavity was decongested using xylocaine with adrenaline in a concentration of 1:100,000. The middle turbinate was opened, the ground lamella, posterior ethmoids and then the sphenoid sinus were entered and the sinus ostium was widened in the inferomedial direction and lateral wall bone was removed in the region of the whole optic canal. The nerve was decompressed in the whole segment of the canal and then medicated pack was placed in the nasal cavity, which was removed on the first postoperative day (Fig. 1).

A high dose of antibiotics and methylprednisolone (20 mg/kg per day for 2–3 days) were administered, and 20% glyceryl alcohol was given via intravenous drip, 5 ml/kg each time, 2–3 times a day for dehydration and anti-edema. Adenosine disodium was given via intravenous drip; vitamin B1 and mecobalamin were injected intramuscularly to nourish the cranial nerves.

Assessment of therapeutical effect

Visual acuity is divided into 5 levels: no light sensation, light sensation, hand movement, finger counting, and standard logarithmic visual acuity chart "visible". The visual acuity was compared before and after operation. Eyesight improved greater than 2 levels was noted as excellent, 1 level as good, and no obvious improvement as invalid.

Results

In this series, traffic accidents were the main cause of head injury involved in 8 cases, fall from height in 2 cases and blunt trauma to the head in 1 case. Right side was involved in 7 cases and left in 4 cases. The vision acuity of 2 cases improved from hand movement to 0.08 and 0.3 after operation. Another case's vision acuity returned to 0.05 compared to light sensation preoperatively. Two cases had finger counting before surgery while they had a vision acuity of 0.4 and light sensation respectively after surgery. However, the other 6 cases did not improve after surgery.

Two patients had complications during surgery. Cerebrospinal fluid rhinorrhea (CSFR) occurred in one case when the sheath of the optic nerve was being incised. CSFR developed in another case when the broken bone fragments were being excised. All the two cases of CSFR were cured by mucosal flap transplantation (the mucosa from excised middle turbinate) and fibrin glue.

Patients were examined for visual acuity after 3 months postoperatively. Among the 11 patients, the eyesight showed improvement in 5 patients. Among them, the vision acuity improved from hand movement preoperatively to 0.08 and 0.3 postoperatively in 2 cases; the vision acuity returned to light sensation postoperatively from no light sensation preoperatively in 1 case; the vision acuity recovered to 0.4 in 1 case and light sensation in 1 case postoperatively from finger counting preoperatively. However, the other 6 cases did not show their eye sight improvement after operation. Among 6 patients who underwent the surgery within 2 weeks after injury, the eyesight improved in 3 cases; however among 5 patients who underwent the surgery over 2 weeks after injury, only 1 patient's eyesight improved ($p < 0.01$).

Discussion

TON is a serious complication of closed head trauma. The incidence rate is 0.5%–5%.[1] It is divided into primary and secondary traumatic optic neuropathy.[2] The primary TON is involved in overactive, rupture, contusion, and distort of the optic nerve. This type of injury always leads to immediate blindness. Surgical effect is disappointing.[3] The secondary TON may be compromised by nerve edema both within the confines of the bony optic canal and the optic sheath.[4] If a patient becomes blind progressively, it is most likely that he has secondary TON and surgery should be performed. Our data showed 4 patients lost eyesight immediately after trauma. Even though the surgery was carried out posttraumatically, none of their eyesight improved eventually. But 7 cases suffered secondary TON, 5 of them got improvement.

Indications for surgery are mainly dependent upon the results of preoperative VEP scans. A prolonged absolute latency on VEP often suggests visual block physiologically.[5] It is generally believed that optic nerve demyelination is induced by edema, ischemia, or hypoxia after trauma. Amplitude reduction often suggests neuroaxonal impairment and diminishment in number. All our cases accepted VEP scan before surgery. VEP could be used as an important reference for preoperative and prognosis evaluation.

It is generally considered that the result of optic nerve decompression will not be satisfactory if surgery is performed 2 weeks or more after trauma. Nevertheless, in our study 3 cases underwent surgery 2 weeks posttraumatically, the eyesight got improved in one case. The result was similar to that reported by Thakar et al,[6] suggesting that operative time after trauma is only partly affected factor for the therapeutic effect of optic canal decompression.

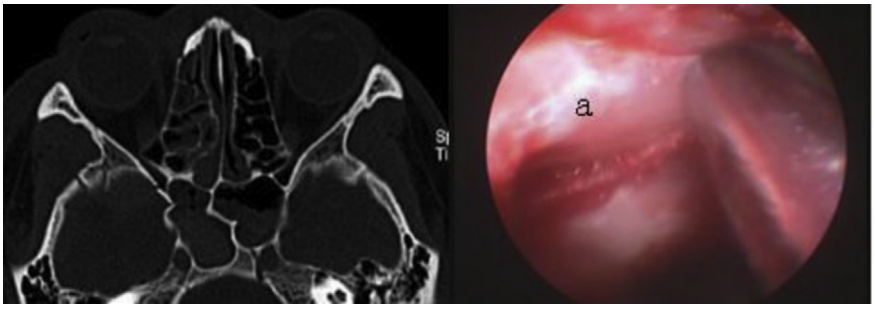


Fig. 1. A male patient, 19 years old, with left TON. CT scan showing fractures of the left lateral wall of sphenoid sinus and the optic canal. Under endoscope, the left optic nerve (a) is seen after endoscopically opening the optic canal.

If CSFR is detected intraoperatively, immediate measures should be taken to reduce intracranial pressure. The mucosa should be transplanted into the fistula, and collagen sponge and fibrin glue should be used to fill it in.[7] Patients should be confined to bed for 10–14 days after surgery. CSFR was seen in 2 cases in our series and resolved by relevant treatment.

It is generally believed that endoscopic optic nerve decompression offers several advantages over other surgical approaches.[8] It requires no external incision. There is no orbital retraction during the procedure. The endoscopes could provide an optimal visual field.[9] Based on our experience, we believe that endoscopic decompression of the optic canal is a safe and effective therapy for TON. The therapeutic effect relies on adequate decompression of the optic canal and skillful surgical technique.

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