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Outcome of endoscopic trans-ethmosphenoid optic canal decompression combined with steroid and nerve growth factor therapy for short - time traumatic optic neuropathy

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内窥镜下经蝶筛径路视神经减压术联合激素及神经生长因子治疗短期外伤性视神经病变

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摘要

目的:介绍内窥镜下经筛蝶径路视神经减压术治疗短期外伤性视神经病变的临床效果。

方法:回顾性研究 151 例首诊小于 5d 的外伤性视神经病变患者,所有患者入院后用甲基强的松针治疗 3d,其中 117 名患者激素治疗后视力无显著提高。术后对激素治疗后视力无显著提高患者局部和全身使用激素和神经生长因子。根据术前视力,患者分为 A 组(无光感组)和 B 组(有残余视力组)。

结果:117 例激素治疗后视力无显著提高患者中,70 例视力有提高,有效率为 59.8%。A 组和 B 组有效率分别为 44.8% 和 80.0%。B 组有效率显著高于 A 组($\chi^2=14.781$, $P<0.05$)。

结论:内窥镜下经筛蝶径路视神经减压术联合激素和神经生长因子治疗短期外伤性神经病变可能是一种有效的治疗方法,术前有残余视力患者疗效好于无光感患者。然而,对于无光感患者也可能通过此治疗提高视力。

关键词:内窥镜下经蝶筛径路视神经减压;激素;神经生长因子

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Abstract

• AIM: To present the efficacy of Endoscopic trans-ethmosphenoid optic canal decompression (ETOCD) combined with steroid and nerve growth factor (NGF) therapy in patients with short-time traumatic optic neuropathy(TON).

• METHODS: A retrospective analysis of 151 TON patients (151 eyes) were performed. Their first treatment were all initiated within 5 days of injury. All patients were treated with methylprednisolone for 3 days. There were 117 patients did not improve with methylprednisolone treatment were offered ETOCD. Local and systemic administration of steroid and NGF were given to them after surgery. According to the visual acuity (VA) preoperatively, all patients who underwent surgery were divided into Group A [no light perception (NLP)] and Group B (with residual vision).

• RESULTS: VA was improved in 70 of 117 patients after surgery, with a total efficacy of 59.8%. The effective rate of Group A and B were 44.8% and 80.0% respectively. Group B had significantly higher effectiveness rate than Group A ($\chi^2=14.781$, $P<0.05$).

• CONCLUSION: ETOCD combined with steroid and NGF may be an useful way to improve VA in short-time TON patients. Patients with preoperative residual vision had better prognosis in VA postoperatively. However, NLP patients may benefit from surgery as well.

• KEYWORDS: endoscopic trans-ethmosphenoid optic canal decompression; steroid; nerve growth factor
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INTRODUCTION

Traumatic optic neuropathy (TON) is a relatively rare but potentially serious complication of head injury. Affecting about 5% of patients with closed head injuries, TON often

causes devastating permanent visual loss[1]. It can be caused by direct or indirect injury. Direct optic injury usually results from optic nerve avulsion or laceration, or from direct fracture of the optic canal. Indirect optic injury is caused by increased intracanalicular pressure after an injury, which usually initiates a cascade of molecular and chemical mediators leading to secondary disorders[2]. The prognosis of direct optic injury is usually quite poor. But indirect injuries, such as edema, hematoma or moderate bony optic nerve compression, may derive benefit from treatment[3].

TON is not a new problem. More than 2000 years ago, Hippocrates wrote that 'there is a dimming of vision in those wounds which are in the brow and slightly above in as much as the wound is more recent they see better but as the scar matures there is further darkening'. Although this disease has been recognized for a long period of time, the best treatment for TON remains unknown. Treatment recommendations in the previous literature include observation, corticosteroid therapy, surgical decompression of the optical nerve canal and corticosteroid therapy combined with surgical decompression[4-7]. Theoretically, optic nerve decompression relieves intracanalicular pressure and allows for the removal of any impinging bony fragment, assisting in the re-establishment of nerve function. Systemically administered steroids have a similar effect, resulting in a 'medical decompression'[8]. Meta-analysis showed that both administration of steroid and/or surgical decompression contributed to the prognosis of traumatic optic neuropathy[9]. A recent study in China showed that nerve growth factor (NGF) can assist in improving the effective rate of TON[10]. The initial visual acuity (VA) is a strong predictor of prognosis. Visual recovery was thought to be particularly poor when the initial vision was no light perception (NLP)[11]. Another strong predictor is the time between the trauma and first treatment. Rajiniganth *et al*[12] found 70% of efficacy when treatment was initiated within 7 days of injury, whereas the effective rate dropped to 24% when treatment started after more than 7 days. Dhaliwal *et al*[13] also mentioned in his latest review that 57% of patients who received surgery within 3 days after the antecedent event had visual improvement after surgery, whereas only 51% of patients who received surgery more than 7 days after the antecedent event improved in VA after surgery.

Our study aimed to investigate the efficacy of ETOCD combined with steroid and NGF therapy in patients with short-time TON . And compared the outcome of the treatment between patients with NLP preoperative and those with residual vision.

SUBJECTS AND METHODS

The study was authorized by The Eye Hospital of Wenzhou Medical University and followed the principles outlined in the declaration of Helsinki (2008). It was approved by the Institutional Ethics Committee (208-519) (Medical Ethics Committee, Wenzhou Medical University, Wenzhou, Zhejiang Province, China). As this was a retrospective study with de-identified data, informed consent was not required. Medical records of patients with TON, who were treated in department of Orbital & Oculoplastic Surgery, Eye hospital of Wenzhou Medical University between Sep. 2012 and Sep. 2015, aging from 7 to 64, were reviewed. First treatment was started within 5 days of injury in all subjects. The diagnosis of TON was made by the presence of vision decrease after direct or indirect head trauma, with a relative afferent pupillary defect. Patients whose post-traumatic visual loss were not related to optic nerve dysfunction, such as traumatic cataract, traumatic retinal detachment, vitreous hemorrhage were excluded in the study.

All patients were first treated with high-dose methylprednisolone (30 mg/kg/day) for 3 days. VA was reevaluated then. If steroid treatment did not benefit in VA patients were offered ETOCD. According to the VA preoperatively, the surgery patients were divided into Group A (NLP) and Group B (with residual vision).

All of the procedures were performed under general anesthesia by a single orbital surgeon (Wu WC) as previously described[14-17]. During endoscopic optic nerve decompression, a routine endoscopic sphenoethmoidectomy was performed using the Messerklinger technique with preservation of the middle turbinate. The sphenoid face is opened widely, and the bulge of the optic nerve canal is identified along the lateral wall of the sphenoid sinus, superior to the carotid artery. In some patients, the optic canal may be identified initially in a posterior ethmoid or Onodi cell, which can be identified on preoperative computed tomography (CT) scan[18]. Identification and opening of the Onodi cell is important to provide adequate surgical exposure and allow full access to the optic canal. After complete sphenoethmoidectomy, The lesser wing of the sphenoidal bone and the medial wall of the optic canal, which spans from the orbital aperture to the cranial cavity, were thinned with a microdrill and removed with a microcurrette (Figure 1A,1B). Then, the periorbita of the orbital apex, annulus of Zinn and the optic nerve sheath were incised with a sharp 9# MVR knife (Figure 1C, 1D). Finally, at the end of the surgery the operating field of the optic canal was covered by a piece of sterile gelatin sponge that was immersed in dexamethasone (5 mg/2 ml) and mouse-derived NGF (30 μg/ml) [Staidson (Beijing) Biopharmaceuticals Co., Ltd] (Figure 1E).

Postoperative care included intravenous methylprednisolone (30 mg/kg/day) for 3 days and intravenous ceftriaxone for 7 days. At the third postoperative day, the dexamethasone and mouse-derived NGF were locally administered as performed at the end of surgery after removal of the gelatin, and this procedure was done every two days for a total of five times. Intramuscular mouse-derived NGF was prescribed for 1 month post-operation.

To be included in our study, at least 3-month follow up were required. Clinical data at the initial, 1 month and 3 months were extracted from patients' medical records. Available data included history of head trauma (initial visit), VA, and any

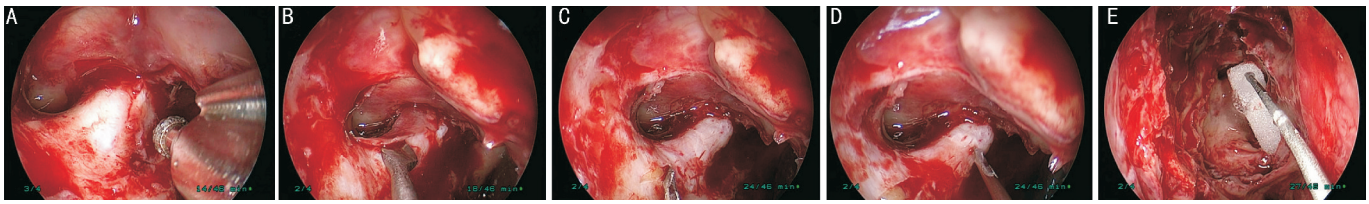

Figure 1 The procedures of ETOCD A: First, using a microdrill to thin the medial wall of the optic canal; B: Then remove thinned medial wall of optic canal with a microcurrette; C: split the periorbita of the orbital apex and annulus of Zinn with a sharp 9# MVR knife. D: Finally, the optic nerve sheath was split in dot (1-2 mm in length, avoiding visible vessels, 10-15 points in total). E: operating field of the optic canal were covered by a piece of sterile gelatin sponge that was immersed in dexamethasone (5 mg/2 ml) and mouse-derived NGF.

complications if applicable. 3-month VA was considered as final VA in our study.

A patient's VA was considered to have improved if a) there was an increase of more than one line on the Snellen visual chart; b) an improvement from no light perception to light perception or better; c) an improvement from light perception to hand motion or better; d) an improvement from hand motion to finger counting or better.

Statistical analyses were performed with SPSS version 17.0 (SPSS inc., Chicago, IL, USA). χ^2 test was used to compare effective rate between Group A and Group B.

RESULTS

All 151 patients, aging from 7-64 years old (29.85±13.71), were enrolled in this retrospective non-randomized study. Eleven of them were female while another 140 were male. Seventy-three of them had TON in left eye, 78 in right eye.

Thirty-four patients had improvement in visual acuity after steroid treatment and declined further surgery. Another 117 patients whose vision failed to improve with steroid treatment underwent ETOCD. Sixty-seven patients were enrolled in Group A and 50 patients were enrolled in Group B. No severe complications (cranial infection, cerebrospinal fluid leak, severe epistaxis, etc.) were observed in these 117 patients. Clinical characteristic of these 117 patients were listed in Table 1. VA was improved in 70 out of these 117 patients, with a total effective rate of 59.8%.

In 67 eyes with NLP vision preoperatively, 30 eyes (45.6%) had improvement in visual acuity. These patients had postoperative light perception (LP) in 7 eyes, hand motion (HM) in 4 eyes, finger counting (FC) in 9 eyes and better than FC in 10 eyes. Eight of 9 eyes with LP preoperatively had improved postoperative vision, including HM in 3 eyes, FC in 2 eye and better than FC 3 eyes. Seven of 9 eyes with HM preoperatively had improved postoperative vision, including FC in 3 eyes and better than FC in 4 eye. Fourteen of 17 eyes with better than FC vision preoperatively had improved postoperative vision. No patients had decreased visual acuity after ETOCD.

The effective rate in Group A and Group B were 44.8% and 80% respectively. Group B had significantly higher effective rate than Group A after treatment ($\chi^2=14.781$, $P<0.05$).

DISCUSSION

TON is a rare but serious complication after head trauma. A national epidemiological study of TON in the UK found a minimum prevalence in the general population of 1 in 1000000. The vast majority of patients are in their early 30's[29]. Motor vehicle is the most common cause of TON, followed by bicycle accident, falls and assaults[11,19]. We enrolled 151 patients in our study, aging from 7-64 years old, with a mean of 29.85±13.71y, among which 96 patients (63.58%) were 20-49 years old. The most common cause of TON in our study was car accident (84/151), followed by falls (35/151), assaults (27/151) and others (5/151).

TON can be caused by direct or indirect injury. Theoretically direct optic injury usually results from optic nerve avulsion or laceration or from direct fracture of optic canal. Indirect optic nerve injury is caused by increased intracanalicular pressure after injury[2]. But the precise pathogenesis of TON is still unclear. However, various studies trying to explain the mechanism of optic nerve damaging in TON were conducted. Some studies suggested that forces applied to the frontal bone were transferred and concentrated in the optic canal region, which would lead to optic nerve damaging[20]. Others demonstrated that patients with indirect injury to optic nerve had been consistent with localization of the lesion to this area which may cause damaging in optic nerve[21]. Increased intracanalicular pressure after injury may initiate a cascade of molecular and chemical mediators, leading to secondary disorders such as intraneural oedema, haematoma, altered microvasculature or cerebrospinal fluid circulation, and interruption of direct axoplasmic transport, which can be the reasons of optic nerve damaging in TON[2].

The treatment of TON is still a matter of controversy[22-24]. No optimal option is available now. Although observation alone showed improvement in VA in some cases[25], most ophthalmologists do not think it is enough for TON, especially for those with NLP. Steroid, operation or steroid combined with operation are the top 3 treatment for TON patients.

High-dose steroid is usually the first choice for TON nowadays. An improvement of 4.3-82.0% after high-dose steroid therapy has been reported[8,12,26]. High-dose corticosteroids can provide an antioxidant effect, thereby protecting neural tissue from the free radicals that form after injury. It will also improve perfusion in damaged tissues by limiting the production of prostaglandins[27]. It may also reduce post-traumatic edema, contusion necrosis, and vasospasm,

Table 1 Clinical characteristic of 117 patients with short-time TON who underwent ETOCD

Characteristic	Case(n)(%)
Injury type	
Fall	29 (24.8)
Car accident	63 (53.8)
Assault	21 (17.9)
Other	4 (3.4)
Hemorrhage within the post-ethmoid and/or sphenoid sinus (Figure 2A)	
Y	52 (44.4)
N	65 (55.6)
Orbital bone fracture (Figure 2B)	
Y	52 (44.4)
Lateral wall	16 (30.8)
Medial wall	12 (23.1)
Floor	6 (11.5)
Roof	2 (3.8)
Multiple fracture	16 (30.8)
N	65 (55.6)
Optic canal fracture (Figure 2C)	
On image scan	39 (33.3)
Found during surgery	53 (45.3)
None	64 (54.7)
Fracture of skull base (Figure 2D)	
Y	9 (7.7)
N	108 (92.3)
Optic nerve edema (Figure 2E)	
Y	7 (5.9)
N	110 (94.1)
Subdural hemorrhage	
Y	5 (4.3)
N	112 (95.7)
Baseline visual acuity	
NLP	67 (57.3)
LP	9 (7.7)
HM	9 (7.7)
FC	15 (12.8)
>0.1	17 (14.5)
Visual acuity postoperative	
NLP	37 (31.6)
LP	8(6.8)
HM	9(7.7)
FC	18 (15.4)
>0.1	45 (38.5)

TON: Traumatic optic neuropathy; ETOCD: Endoscopic trans-ethmosphenoid optic canal decompression; NLP: No light perception; LP: Light perception; HM: Hand motion; FC: Finger counting.

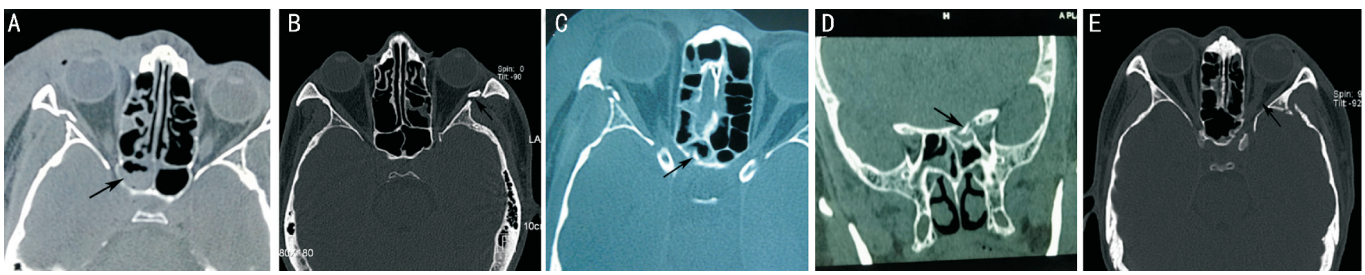

Figure 2 The image of hemorrhage within the post-ethmoid and/or sphenoid sinus (A), orbital bone fracture (B) and Optic canal fracture (C), fracture of skull base (D), and optic nerve edema (E).

and thus aid functional recovery[28]. But optic atrophy will become apparent at 3-4 weeks after TON[12]. Steroid using cannot benefit optic nerve functional recovery at that time. Therefore, time between the first treatment and injury is an important factor for prognosis. Patients enrolled in our study were all short-time TON. First treatment were all initiated within 5 days of injury. We believed that high-dose steroid should benefit some of these patients. So we treated them with methylprednisolone (30 mg/kg/day) for 3 days and reevaluated VA after the steroid treatment. Thirty-four of them improved in VA then. Another 117 patients who didn't improve in VA were offered ETOCD.

Optic nerve decompression physically decompresses the nerve within the canal, thereby creating space for the nerve to swell and limiting the damaging effect of the compartment syndrome[29]. Different approaches have been advocated for optic nerve decompression, including transcranial, transantral, intranasal microscopic and endoscopic approaches[30-31]. ETOCD was recently introduced by some ENT specialists for treatment of TON. The endoscopic approach offers many advantages, including decreased morbidity, preservation of olfaction, ideal cosmetic results without external scarring, no risk of injury to the developing teeth in children and a shorter recovery time. Most importantly, the endoscopic approach provides an excellent view of the orbital apex[8].

Yang *et al*[32] recommended that high dose steroid therapy should be the primary treatment for TON, and ETOND can be performed as an adjuvant to steroid therapy in cases where steroids failed. However, Yexun Song's study revealed that no differences were detected in the outcomes between patients who received surgery plus preoperative steroids and those under-went surgery alone. It was believed that it was not necessary to wait several days to assess the effect of steroid before surgery[33]. Since all cases in our study were short-time TON, steroids' medical decompression effect may relieve intracanalicular pressure and may help re-establish optic nerve function. Steroids were given to all patients in our study. Steroids were prescribed for 3 days after surgery for surgery patients as well.

Whether to split the nerve sheath during ETOCD is another controversial issue. Xu *et al*'s[34] study demonstrated that nerve sheath incision is not obligatory for the improvement of visual acuity during endoscopic optic nerve decompression for the treatment of TON. The annulus of Zinn at the anterior end of the fibrous sheath may contribute to edema[35]. However, slitting of the sheath will increase the risk of cerebrospinal fluid leakage, incidence of ophthalmic artery injury and secondary injury to the optic nerve. We did optic nerve sheath splitting during the surgery in this study. We performed punctate optic nerve sheath splitting instead of traditional way, thereby decreasing the incidence of complication (like cerebrospinal fluid leak and local vascular damage) and creating space for local administration of dexamethasone and mouse-derived NGF postoperation. We also decompressed the orbit apex by opening the periorbital of the orbital apex and annulus of Zinn to further decrease intracanal pressure.

NGF is another treatment for TON, which was raised up in recent years. Many ENT specialists used it as an adjunctive therapy for TON[36]. A recent meta-analysis in China with 7 random-controlled trails (399 eyes in total) showed that mouse-derived NGF can improve the effective rate of TON[10].

An improvement of 40-63.5% after ETOCD has been reported[18,34,37]. For patients with NLP, the visual acuity improvement after ETOCD differed from 10% to 30%, and few of them could have practical VA after surgery[38-40]. In our study, VA improvement was achieved in 70 patients out of 117 patients (59.8%) after ETOCD. An effective rate of 44.8% (30/67) for patients with NLP preoperative were observed in our study. About 14.92% (10/67) of them had practical VA (>0.01) postoperative. The possible reasons for these high effective rates were as follows: 1) short time between the injury and the first treatment. All cases in our study started their first treatment within 5 days from injury. This early treatment may be an important reason; 2) punctate optic nerve sheath splitting and opening the periorbital of the orbital apex and annulus of Zinn during the surgery. These 2 motions during the surgery can absolutely decrease intracanal pressure and decrease the incidence of complication; 3) local administration of dexamethasone and mouse-derived NGF, intravenous methylprednisolone and intramuscular mouse-derived NGF postoperation. In our study, the medicine (steroids and NGF) were locally administered as performed at the end of surgery after removal of the gelatin in all patients at the third postoperative day, and this procedure was done every two days for a total of five times. Since the sphenoid sinus is a perfect place to store the medications, this kind of local administration used after surgery can provide nutrition for the optic nerve continually. In addition, opening the optic nerve canal will assist in medicine absorbing. Intramuscular mouse-derived NGF was prescribed for 1 month post-operation.

Therefore, data suggested that ETOCD combined with steroid and NGF therapy is a useful management for TON with satisfactory prognosis.

The initial VA was thought to be a strong predictor of prognosis. The prognosis of TON is very poor especially for those with NLP. Visual recovery was particularly poor when the initial vision was NLP[11]. Previous studies showed the effective rate of NLP patients were only 10-30%[38-40], much lower than the overall effective rate of TON patients after ETOCD[18,34,37]. And few of NLP patients could have practical VA after surgery[38-40]. Dhaliwal *et al*[13] noted in his review that 41% of patients with NLP preoperatively saw

improvement after surgery, whereas those with LP (89%), HM (93%), or FC (85%) fared better. An effective rate of 44.8% (30/67) and 80% (40/50) were observed for patients with NLP and patients with residual vision preoperative respectively in our study. Patients with residual vision preoperative had significantly higher effective rate than NLP patients. This result was consistent with previous studies[41-44]. Song *et al*'s[33] data showed that hemorrhage within the ethmoid and/or sphenoid sinus was associated with NLP and was a prognostic factor for unrecovered visual acuity in TON with complete blindness, but the precise mechanism is unclear. Seiff *et al*[45] found that the presence of an optic canal fracture was associated more frequently with NLP than when it was absent. This presence of optic canal fracture may serve as an indicator of the severity of the force transferred to the TON[46].

Patients suffered from short-time TON with residual vision before ETOCD have better surgical outcome than those with NLP. However, patients with NLP preoperative may benefit from surgery as well. ETOCD combined with steroid and NGF therapy may be a useful management for patients with short-time TON with satisfactory prognosis. Large-sampled controlled prospective researches are needed to further prove the rationality of ETOCD for patients with TON and to work out the exact surgical indication.

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