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Endoscopic optic nerve decompression for the treatment of traumatic optic neuropathy*

Rong-San Jiang[1, 2], Chen-Yi Hsu[1], Bing-Herng Shen[3]

[1] Department of Otolaryngology, Taichung Veterans General Hospital, Taichung, Taiwan, Republic of China
[2] Department of Medicine, Chung-Shan Medical and Dental College, Taichung, Taiwan, Republic of China
[3] Department of Ophthalmology, Taichung Veterans General Hospital, Taichung, Taiwan, Republic of China

SUMMARY

Optic nerve decompression has been accomplished by a variety of procedures. Since 1995, endoscopic optic nerve decompression (EOND) has been used to treat traumatic optic neuropathy (TON) patients in our hospital after medical treatment failed. To date, 17 TON patients have received EOND in our hospital and have been followed up for more than half a year. After decompression, vision improved in 9 patients, remained the same in 6, and became worse in 2. The dura was incidentally exposed during the operation in 1 patient. We conclude that it is easier and more precise to perform optic nerve decompression by EOND than by other optic nerve decompression procedures. However, its efficacy still needs further investigation.

Key words: endoscopic optic nerve decompression, traumatic optic neuropathy

INTRODUCTION

Optic nerve decompression has been practised since 1916 as a treatment for several disorders which cause loss of vision (Osguthorpe and Sofferman, 1988; Sofferman, 1995). One of the common indications for optic nerve decompression is traumatic optic neuropathy (TON) (Joseph et al., 1990). Frontotemporal craniotomy was the common early method for decompressing the optic nerve (Steinsapir and Goldberg, 1994). However, this approach entailed a high rate of morbidity (Amrith et al., 1993). In 1926, Sewall used external ethmoidectomy to approach the medial optic canal and began extracranial optic nerve decompression (Sofferman, 1995). This technique was popularized by the Japanese in the 1960s and 1970s and was considered more effective with less morbidity than the craniotomy approach (Osguthorpe and Sofferman, 1988; Joseph et al., 1990; Knox et al., 1990). Since then, other forms of extracranial decompression have been suggested, including the transantral-ethmoidal approach, a combined lateral and medial orbitotomy, sublabial transnasal approach, intranasal microscopic technique, and lateral facial approach (Knox et al., 1990; Steinsapir and Goldberg, 1994). Each has its own advantages and disadvantages, so the method should be chosen on a case-by-case basis (Joseph et al., 1990; Sofferman, 1995). Recently, the endoscopic endonasal technique has been widely applied in the treatment of many disorders. In 1991, Aurbach reported the use of this technique for decompression of the optic nerve (endoscopic optic nerve decompression, EOND) in the German literature (Silberman and Chow, 1995). Lately, Luxenberger et al. (1998) also reported their experience with EOND. They concluded EOND offers many advantages over the traditional approaches, including decreased morbidity, rapid recovery time, and no external scars.

In 1982, Anderson et al. introduced a new treatment option to treat TON, a high dose of steroids (Sofferman, 1995). Since then, much experience has been accumulated to demonstrate the efficacy of steroids for restoring vision. Moreover, steroid therapy has been shown to give better results than surgical decompression (Matsuzaki et al., 1982). Therefore, in our hospital, high dose steroid therapy became the primary treatment for TON. However, if the steroid therapy failed, optic nerve decompression was advised to rescue the vision. In this study, we report our experience using EOND to decompress the optic nerve for the treatment of TON in patients for whom high dose steroid therapy failed.

MATERIAL AND METHODS

When TON patients were sent to or came to our hospital, they were seen by ophthalmologists after a neurosurgical condition was excluded or under control. If visual impairment was caused

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by optic neuropathy, they were treated with a course of steroids which consisted most commonly of high dose (250 mg) intravenous methylprednisolone every six hours for 3 days when no contraindication existed. Vision was re-evaluated after the high dose steroid treatment.

When vision did not improve satisfactorily, otolaryngologic consultation was requested for EOND. Before performing EOND, axial and coronal computed tomography (CT) of the paranasal sinuses was done to evaluate the optic nerve, the medial wall of the orbit, the thickness of the optic tubercle, the bony wall and location of the internal carotid artery, and the degree of pneumanization of the sphenoid sinus. Magnetic resonance imaging was not done to evaluate the situation of the optic nerve since the optic nerve was shown to be normal on CT in all cases. The physician met with the patient and his family to obtain consent for EOND, during which the procedures, results, and possible complications of EOND were explained. If the patient was willing to receive EOND, a statement of informed consent was signed, and EOND was performed on the next operation day (less than 1 week).

During EOND, the patient was put under general anesthesia, prepped and draped in the routine manner for endoscopic sinus surgery. Cotton pledgets which were soaked in cocaine solution were placed in the nasal cavity. The sphenopalatine vessels were vasoconstricted through the greater palatine foramen with a solution of lidocaine 2% and epinephrine 1:100,000. This solution was also injected into the nasal mucosa, as for endoscopic sinus surgery, to enforce vasoconstriction.

A routine endoscopic ethmoidectomy was performed first. When the sphenoid sinus was identified, the ostium was opened wide. An attempt was made to locate the optic nerve and carotid artery on the lateral wall of the sphenoid sinus in order to speed up the later decompression procedures and reduce the risk of the operation. Next, the posterior ethmoid sinus was deliberately and clearly dissected to expose the orbital apex and optic ring.

Table 1. History and prognosis.

Patient	Lucid Interval*	Period from Trauma to EOND** (Days)	Period from Steroid to EOND (Days)	Prognosis (Vision)
1	-	26	17	no change
2	+	25	16	improved
3	+	13	5	worse
4	-	11	8	improved
5	-	67	60	worse
6	?	87	84	improved
7	-	28	25	improved
8	-	13	11	improved
9	+	21	2	improved
10	+	13	5	no change
11	-	33	18	no change
12	?	18	15	improved
13	-	7	3	no change
14	+	11	7	improved
15	-	74	70	improved
16	+	42	33	no change
17	-	3	0	no change

*: - meaning no interval; + meaning interval existed; ? meaning no record of an interval

**: endoscopic optic nerve decompression

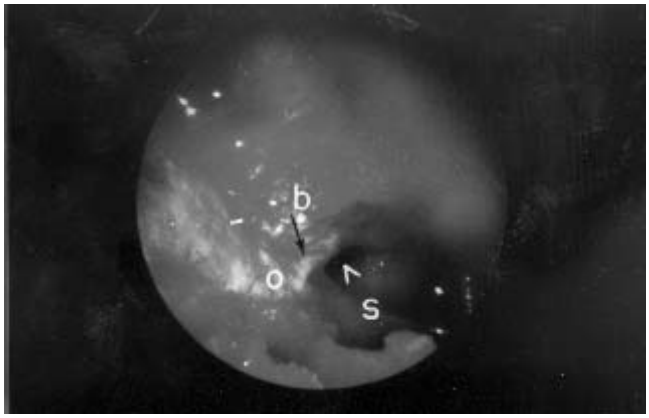

Figure 1. The decompressed optic nerve (arrow). b: skull base; s: right sphenoid sinus; o: partially exposed periorbita of medial wall of orbital apex. The infraoptic recess is indicated by a white arrowhead.

Under endoscopic observation, a long-hand microdrill with a 2.3-mm cutting burr was used to remove a piece of the lamina papyracea on the medial wall of the orbital apex. However, a diamond burr might be less traumatic. After that, we continued working backward to remove the lamina papyracea with a pair of small forceps until the optic ring was reached at the posterior end of the orbital apex.

Because the bone of the anterior end of the optic canal, the optic ring, is usually very thick, it was first thinned by drilling. In order to avoid thermal injury to the optic nerve and surrounding tissue, frequent irrigation was needed. Then, the thinned optic ring was removed with forceps. Following this, decompression of the medial wall of the optic canal was continued backward either with small forceps alone or in combination with a microdrill, depending on the thickness of the bony wall. With this technique, the optic canal was decompressed 180 degrees medially, from the optic tubercle to near the optic chiasm (Figure 1). However, the sheath of the optic nerve was not incised in our cases.

During the procedure, bleeding was minimal. Therefore, nasal packs were not necessary after the operation. A short course of low-dose steroids was given to decrease the postoperative edema of the optic nerve. Postoperatively, ophthalmologic consultation was requested to monitor the change in visual acuity within 24 hours or when the patient noticed visual change. After a hospital stay of several days, the patient was discharged to undergo regular follow-up in the ophthalmologic clinic to monitor his visual condition.

RESULTS

Since 1995, there have been 17 TON patients who have received EOND in our hospital and have been followed up for more than half a year. All of them were male. Their ages ranged from 14 to 45 years with a mean of 20.7 years. The right eye was involved in 13 patients, and the left eye was involved in the other 4. The causes of TON were a traffic accident in 13 patients, falling down from a height in 3 patients, and head injury during a big earthquake in 1 patient. Bony fracture of the orbital wall was shown in only 3 cases on the CT scans, but no other orbital or optic nerve disorder was found. The period from trauma to EOND varied from 3 to 87 days.

Table 2. Visual acuity.

Patient	before Treatment	after Steroid Treatment	after EOND*
1	NLP[1]	NLP	NLP
2	NLP	NLP	CF/30cm[2]
3	HM[3]	20/800	NLP
4	HM/20cm	HM/30cm	20/200
5	LP[4]	20/600	NLP
6	No Record[5]	HM/15cm	HM/45cm
7	No Record	HM/30cm	20/1200
8	No Record	20/200	20/100
9	20/200	20/200	20/67
10	NLP	NLP	NLP
11	20/200	20/200	20/200
12	HM/150cm	CF/50cm	20/1200
13	NLP	NLP	NLP
14	20/600	20/400	20/500
15	CF/10cm	CF/20cm	20/1200
16	NLP	NLP	NLP
17	NLP	NLP	NLP

* endoscopic optic nerve decompression; 1: no light perception at all; 2: finger counting at the distance of 30cm; 3: hand movement; 4: light perception; 5: visual acuity not recorded

Before treatment, 6 patients had no light perception at all, 5 patients had vision worse than 20/1200, 3 were better than 20/1200, and visual activity was not recorded for the remaining 3. The existence of a lucid interval of preserved vision after trauma was recorded in 6 patients. Nine patients did not have a lucid interval. The other 2 patients did not have any records regarding lucid interval (Table 2).

After high dose steroid therapy, vision was improved in 6 patients. Eight patients maintained the same level of vision after treatment. No treatment results were recorded for the remaining 3. Vision after treatment ranged from no light perception at all in 6 patients, worse than 20/1200 in 5, to better than 20/1200 in 6 (Table 2).

After EOND, vision was improved in 9 patients, remained the same in 6, and became worse in the other 2 (Table 2). Among them, 7 patients had no light perception at all, 2 patients had vision worse than 20/1200, and 8 were better than 20/1200.

The dura was incidentally exposed in 1 patient during the operation, but no cerebrospinal fluid leakage was seen. No treatment was given, and he has continued to do well.

DISCUSSION

The proper strategy for the management of TON is still under debate, partly because the natural history of TON remains unclear (Mauriello et al., 1992; Steinsapir and Goldberg, 1994). Spontaneous recovery of vision has been observed in some patients, but not in most patients (Miller, 1990; Wolin and Lavin, 1990). In 1982, Anderson et al. introduced a new treatment option, a high dose of steroids (Sofferman, 1995). Since then, much experience has been accumulated to demonstrate the efficacy of steroids for restoring vision. Moreover, steroid therapy has even been shown to give better results than surgical decompression (Matsuzaki et al., 1982). Since the intracanalicular part of the optic nerve is most frequently injured in TON, it is not surprising that surgical decompression of the optic canal has been attempted in the treatment of TON (Steinsapir and Goldberg, 1994; Sofferman, 1995). Although there has been no randomized, prospective, and well-controlled trial to clarify the role of surgical decompression, its efficacy has been declared in many reports (Miller, 1990; Mauriello et al., 1992; Amrith et al., 1993; Steinsapir and Goldberg, 1994).

Initially, surgical decompression of the optic canal was most frequently performed transcranially (Steinsapir and Goldberg, 1994). Because of this approaches morbidity, it has mostly been replaced by the extracranial method (Sofferman, 1995). There are several options for an extracranial approach (Fujitani et al., 1986). Recently, endoscopic endonasal surgery has been applied to many different types of cases, including optic nerve decompression (Papay et al., 1989; Silberman and Chow, 1995).

The endoscopic endonasal technique not only has the advantage of avoiding facial incision, as opposed to the external ethmoidectomy method (which is most commonly used nowadays), but also provides a more direct approach to the optic nerve (Silberman and Chow, 1995; Sofferman, 1995). Therefore, it shows the least morbidity (Stankiewicz, 1989; Luxenberger et al., 1998). Although it is an endonasal approach, it provides the best view among all extracranial methods of the operative field by way of the endoscope, so that a precise procedure can be performed (Zeitouni et al., 1994).

However, EOND has the same disadvantages as other endoscopic endonasal surgeries. It should be performed only by an experienced endoscopic surgeon (Silberman and Chow, 1995; Luxenberger et al., 1998). Moreover, intraoperative bleeding is a bigger problem in the endoscopic method than in other procedures (Stankiewicz, 1989). Furthermore, when EOND is performed on the lateral wall of the sphenoid sinus, it is more difficult, and carries a higher risk of injury to the internal carotid artery than other endoscopic endonasal surgeries (Cheung et al., 1993). By the same token, as EOND removes the medial wall of the optic canal inside the sphenoid sinus, evaluating the pneumanization of the sphenoid sinus preoperatively is a prerequisite with non-pneumanization being a contraindication of EOND (Silberman and Chow, 1995).

While EOND has become the main surgical approach for optic nerve decompression in some hospitals, strict indications for and timing of EOND in TON patients has not been established (Kountakis et al., 1997; Luxenberger et al., 1998). In the Kountakis et al. (1997) and Luxenberger et al. (1998) series, conservative treatment with megadose steroids was tried before EOND. The same strategy was also applied in our cases.

Although Joseph et al. (1990) suggested that optic nerve decompression be performed not later than 7 days after injury, improvement in vision has been reported in TON patients who received EOND 7 days, 13 days, and 6 weeks after the trauma (Kountakis et al., 1997; Luxenberger et al., 1998). Since our hospital receives many referrals and is a busy medical centre, it is difficult for our TON patients to receive EOND within 1 week after the trauma. One of our patients received EOND 87 days after injury, yet showed an improvement of visual acuity. Furthermore, the timing of EOND did not seem an important fac-

tor in determining the efficacy of EOND in our cases (Table 1), but EOND was performed only twice within 7 days after the injury, so we can not draw any conclusions about what might happen with a shorter interval.

Optic nerve sheath fenestration was done in most cases of the Kountakis et al. (1997) and Luxenberger et al. (1998) series. However, its effectiveness is still controversial (Silberman and Chow, 1995). Therefore, we did not incise the optic nerve sheath in any of our cases.

The efficacy of EOND was not evaluated in a random, well-controlled way in this study. The 52.9% improvement rate of vision in our patients was comparable to the improvement rate (50%, 7/14 patients) of EOND reported by Luxenberger et al. (1998). A better result (75%, 6/8 patients) of EOND was reported by Kountakis et al. (1997), but optic canal fracture existed in all of their cases. In contrast, optic canal fracture was not found in any of our cases. However, the effect of steroid therapy has not mentioned in the reports of Kountakis et al.(1997) and Luxenberger et al. (1998). Therefore, it must be assumed that the efficacy of EOND might not be comparable between different patients groups. On the other hand, the improvement of vision after EOND in our patients seemed unrelated to the previous steroid therapy although it is impossible to exclude completely the possible attribution of steroid therapy to a successful outcome. One of our patients received EOND 84 days after completion of high dose steroid therapy. Some vision was restored after EOND. Six of 9 patients with improved vision after EOND received EOND more than 10 days after steroid therapy (Table 1).

Among those 6 patients whose vision remained the same after EOND, 5 had no have light perception before EOND. On the other hand, only 1 patient with no light perception before EOND showed improvement after EOND. This implied that lack of light perception before EOND indicated a poor prognosis despite EOND. In Luxenberger et al. (1998) series, 10 TON patients had no light perception before EOND. Among them, 6 patients remained without light perception after EOND.

Although no complications were noted in any of the Luxenberger et al. (1998) patients with EOND, an iatrogenic injury to the nerve fascicle itself or the ophthalmic artery might occur. Despite the fact that some of our cases showed significant improvement in vision after EOND, vision became worse in 2 cases. These were among our early cases (3rd and 5th cases), when we had little experience with EOND. The decompressed optic nerve was over-manipulated by instruments in the 3rd case with some fragmented nerve fascicle noted during the operation. On the other hand, the operative course was rather smooth in the 5th case. In both cases, a cutting burr was used to decompress the medial wall of the optic canal. Another possible cause of loss of vision after EOND might be thermal injury. The irrigation during drilling might not have been sufficient in the 5th case. Continuous irrigation during drilling might be preferred to prevent thermal injury of the decompressed optic nerve (Kuppersmith et al., 1997). Over-manipulation and thermal injury of the decompressed optic nerve was carefully avoided in later cases.

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Rong-San Jiang, M.D.
Department of Otolaryngology,
Taichung Veterans General Hospital,
Taichung, Taiwan 40705,
Republic of China

Tel.: +886-4-359-2525 extension 5401
Fax: +886-4-359-6868
E-mail: rsjiang@vghtc.vghtc.tw.gov