Traumatic Optic Neuropathy in Two Patients With Different Manifestations and Outcomes

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Traumatic optic neuropathy is a rare condition due to direct or indirect injury associated with head trauma. The optimal management modality is still controversial, though spontaneous visual recovery has been reported. We hereby present two patients, 18 and 52 years old, respectively, with traumatic optic neuropathy. They had different clinical presentations and had different outcomes after treatment with high-dose steroids. According to the results of these two patients, the initial clinical presentation of traumatic optic neuropathy may be important in predicting its outcome. (Mid Taiwan J Med 1999;4:269-73)

Key words

optic neuropathy, trauma

INTRODUCTION

Optic nerve injury may result from direct trauma to the optic nerve or compression of the optic nerve by edema, hematoma or fractured bony fragments. The optimal management of vision loss after facial trauma is still controversial because of the low incidence and difficulty of evaluation using a prospective study [1,2]. While high-dose steroid has become the treatment of choice [3,4], optic canal decompression is advocated by some authors [5-7]. We have encountered two patients with traumatic optic nerve injury. The initial clinical presentations, imaging findings, treatment options, and outcomes are discussed.

CASE REPORTS

Case 1

An 18-year-old man was sent to our emergency department after a motorcycle accident with the chief complaints of left facial swelling, vision loss of the left eye, and bloody rhinorrhea. There was no initial loss of consciousness. Physical examination revealed an alert man with marked swelling over the left side of the face and lower eyelid. Visual acuity was normal in the right eye but no light perception or reflex in the left eye. Brain and orbital computed tomography (CT) scan revealed traumatic subdural hemorrhage (SDH) and subarachnoidal hemorrhage (SAH) over the left frontal lobe (Fig. 1) and fractures of the left maxilla, zygoma, and lateral orbital wall (Fig. 2).

Although emergency surgical decompression was suggested, the patient and his family refused to accept the suggestion. Then he received 250 mg of intravenous methylprednisolone every 6 hours for 3 days. On the second day of hospitalization, fundoscopic examination showed mild disc hemorrhage and commotio retinae over the left eye. The hospitalization course was complicated by meningitis and he was discharged on the 14th

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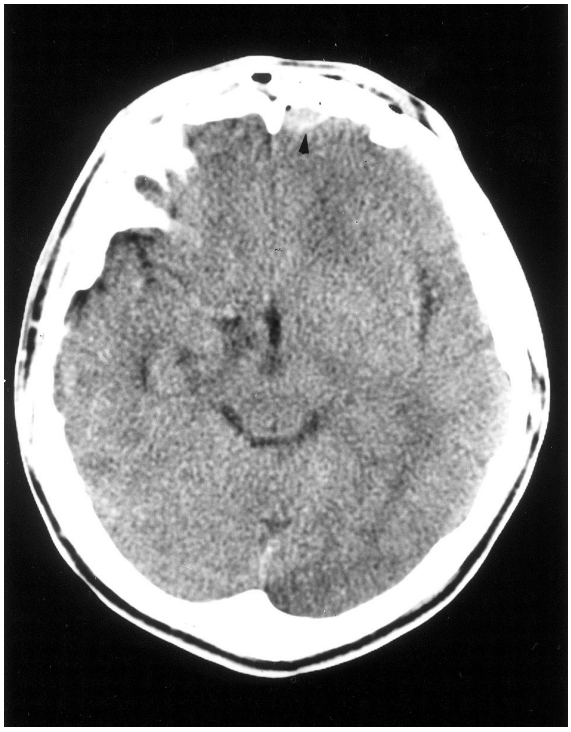

Fig.1 Brain CT scan of case 1. Minimal subdural hematoma (arrowhead) and subarachnoid hemorrhage over left frontal lobe without mass effect were noted.

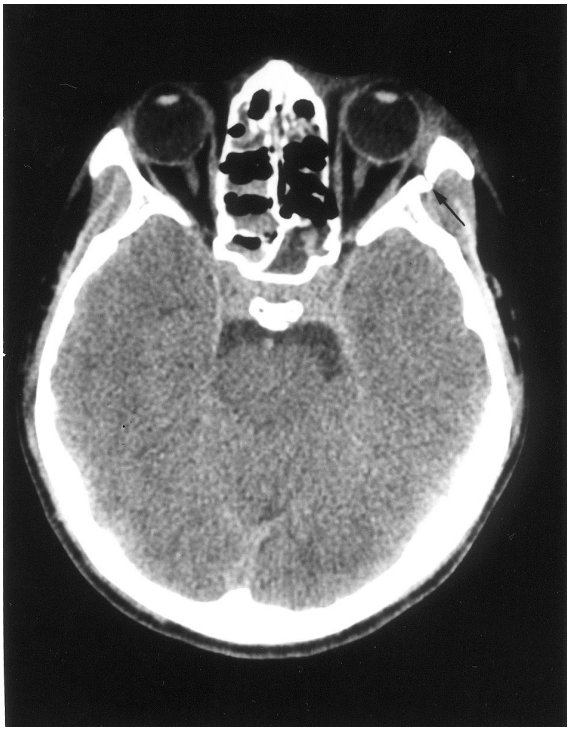

Fig.2 Orbital CT scan of case 1. Fracture at left lateral orbital wall (arrow) without intraocular or intracanalicular lesion is noted.

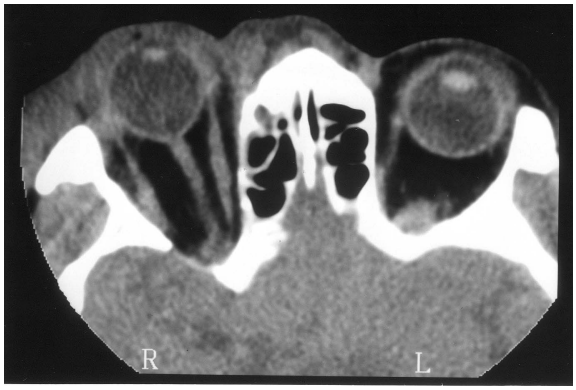

Fig.3 Orbital CT scan of case 2. There is no intraocular or intracanalicular lesion.

day. During a 5-month follow-up period at the neurosurgery clinic, the vision of the left eye did not improve.

Case 2

A 52-year-old man was brought to our emergency department with the chief complaint of facial swelling and blurred vision over the right eye after a violent assault. Ophthalmologic examination of the right eye showed a semi-dilated pupil without light reflex, mildly increased intraocular pressure (27 mmHg), and visual acuity of light perception only. The results of fundoscopic examination were normal. Orbital CT scan was performed which showed a blow-out lesion of the right orbit with depression of the right orbital content (Fig. 3). He was admitted to our ophthalmology ward where methyl-prednisolone (250 mg intravenously every 6 hours for 3 days), mannitol injection, and diamox were prescribed. The vision of the right eye improved gradually and recovered completely 2 weeks after the injury.

DISCUSSION

Traumatic optic neuropathy is a rare condition and is reported in less than 5% of all head injury patients [8]. Indirect compression of the nerve by retrobulbar hemorrhage or perineural edema, or secondary to vascular spasm or thrombosis are more common than

direct injury due to penetration or fracture at the intracanalicular region [6,7]. Anderson et al indicated that with increased loads above the blow region, stresses were concentrated near the optic foramen, leading to intracanalicular nerve compression [3]. The vision loss may vary in severity and time of onset, while up to one third of these injuries improve spontaneously [9]. Any patients with suggested optic nerve contusion should receive a complete eye examination and orbital CT scan with attention to the optic canal.

If no fracture or hematoma of the canal is noted, most authors recommend medical therapy with steroids to reduce edema, stabilize cell membranes, and decrease circulatory spasms. Most therapeutic regimes are based on the "megadose" steroid protocol by Anderson et al [3]. Spoor et al [4] used a protocol with methylprednisolone based on doses which have been used in spinal cord injury studies. Despite the absence of a well-designed prospective study, steroid treatment appears to improve the outcome [3,4,9,10] even though treatment is delayed [10].

On the other hand, some authors advocate optic canal decompression as an alternative or additional therapy to steroids. Various techniques of optic canal decompression, either intracranial or extracranial, have been performed in recent years. Results of optic nerve decompression in a large series of patients reported by Fukado [5] showed visual recovery in more than 40% of patients. This unsatisfactory result raised serious doubt as to the benefits of surgery; however, better results have been reported in recent years though a well-controlled prospective study is still absent [6,7]. The interval and indications for surgical intervention in patients with TON are still controversial. Most authors proposed optic nerve decompression for those patients with delayed visual recovery [6,8] or optic canal fracture without recovery after steroid treatment [10].

Though the different therapies are difficult to be evaluated prospectively, some predictors of prognosis should be considered. The immediate visual loss is associated with a poorer outcome. Pediatric patients had relatively poorer recovery when compared with adults. Poor recovery was also reported in patients with no light perception [8,10] or with optic canal fracture [10,11]. Visual evoked potentials is a reliable test in assessing the integrity of the visual pathway and its predictive value has been well established [12,13]. However, this test is not widely used and was not even mentioned in recent reports in the management of patients with optic nerve injury [8].

In our case 1, immediate vision loss with no light perception or intracanalicular lesions noted using CT scan indicated severe optic nerve injury. Poor outcome could be expected and immediate optic nerve decompression might not be helpful. Only 20% of the patients with no light perception will achieve visual improvement while receiving steroid treatment [10]. As for case 2, the initial visual acuity was only of light perception yet he improved completely after medical treatment. He was rather fortunate because most patients only achieved partial improvement [10].

In conclusion, although various types of treatment for patients with traumatic optic neuropathy have been proposed, none showed absolute superiority over the others. The initial visual acuity with light perception is a key factor in predicting the outcome of visual recovery.

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創傷性視神經病變：兩個不同臨床表現及結果之病例報告

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傷性視神經病變爲頭部外傷少見之併發症，其自然恢復之情況有之，但仍有許多研究者希望藉由各種治療法改進其預後，其中最常使用者爲短期大劑量之類固醇合併或不合併視神經管減壓手術。本文介紹兩個病例報告，分別爲18及52歲之男性,住院時前者無光感應，後者則有，在接受類固醇治療後之結果大不相同，由此顯示受傷後時之視力是否有光感應以上對其視力恢復與否影響很大，同時回顧相關文獻一併討論。（中台灣醫誌 **1999;4:269-73**）

關鍵詞

視神經病變，創傷

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