

Traumatic Optic Neuropathy Caused by Blunt Forehead Trauma: Case Report

Künt Kafa Travmasini Takiben Travmatik Optik Nöropati: Olgu Sunumu

HASAN MİRZAI, ESİN FATMA ERKİN, CUMHUR TOSUN

Celal Bayar University, Departments of Neurosurgery (HM, CT) and Ophthalmology (EFE), Manisa, Turkey

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Abstract: A 28-year-old man sustained blunt forehead trauma in a traffic accident. He had a right orbital roof fracture and there was loss of vision in the right eye. At presentation, the visual acuity in the injured eye was light perception without projection, and there was a right afferent pupillary defect. There were no abnormalities on ophthalmoscopic examination. Computed tomography and magnetic resonance imaging demonstrated a fracture of the right orbital roof and showed that the optic nerve was intact. The patient was diagnosed with traumatic optic neuropathy, and was started on 1000 mg methylprednisolone therapy 18 hours after the trauma occurred. The vision in the traumatized eye improved to the level of 2/10 at 3 weeks after therapy, and remained at this level thereafter. Traumatic optic neuropathy associated with blunt forehead trauma and the various treatment strategies for this condition are discussed.

Key words: blunt trauma, corticosteroid therapy, orbital fracture, traumatic optic neuropathy

Özet: 28 yaşında erkek olgu trafik kazasında künt kafa travmasına maruz kaldı. Sağ gözde görme kaybıyla beraber sağ orbita tavan fraktürü vardı. İlk görme keskinliği projeksiyon olmaksızın ışık hissi düzeyindeydi ve sağ afferent pupil defekti mevcuttu. Oftalmoskopik muayene normaldi. Komputere tomografi ve manyetik rezonans görüntüleme sağ orbita tavan fraktürü olduğunu optik sinirin intakt olduğunu gösterdi. Olguya travmatik optik nöropati teşhisi konuldu ve travmadan 18 saat sonra 1000 mg metilprednizolon tedavisi başlandı. Görme keskinliği tedaviden sonraki 3. haftada 2/10 düzeyine ulaştı ve bundan sonra stabil seyretti. Künt alın bölgesi travması ile ilişkili travmatik optik nöropati ve tedavi stratejileri tartışıldı.

Anahtar kelimeler: Künt travma, kortikosteroid tedavisi, orbita fraktürü, travmatik optik nöropati

INTRODUCTION

Traumatic optic neuropathy is an uncommon but often devastating cause of permanent visual loss after blunt or penetrating injury. The most frequent form is indirect damage caused by a concussive blow

to the head, especially to the forehead (11). Indirect traumatic optic neuropathy is defined as visual loss without clinical evidence of injury to the eye or optic nerve (15). The majority of victims are young males, and bicycle and motor vehicle accidents are the most common causes of damage (9,11,15). This case report

discusses unilateral traumatic optic neuropathy associated with forehead trauma.

CASE REPORT

A 28-year-old male sustained blunt forehead trauma in a traffic accident. He was admitted to our hospital several hours after the injury. Physical and neurological examinations revealed that the patient was alert and had a Glasgow coma scale score of 15. His cranial nerve examination was unremarkable, apart from absence of the direct pupillary light reflex in the right eye, and of the indirect pupillary light reflex in the left eye. Cranial and orbital computed tomography showed no significant cerebral pathology, but demonstrated a fracture of the roof of the right orbit and pneumoorbita. Also, the optic canal was confirmed intact and no retrobulbar hematoma was detected (Figure 1). Magnetic resonance imaging revealed that the right optic nerve was intact (Figure 2 a,b).

On initial ophthalmologic examination at bedside, the level of vision in the right eye was light perception without projection, and the left eye had reasonably good vision. The patient's pupils were isocoric, but there was an afferent pupillary defect on the right. The external ophthalmologic examination was normal except for periocular ecchymosis and a hematoma of the right upper lid. The intraocular pressure in both eyes was within normal limits. The ophthalmoscopic exam revealed

no abnormal findings, and there were no stigmata of lesions typically associated with blunt ocular trauma. Eye movements were normal in all directions, and were unrestricted. The patient was diagnosed with traumatic optic neuropathy associated with blunt forehead trauma. Approximately 18 hours post-trauma, he was started on a course of 250 mg intravenous (IV) methylprednisolone 4 times daily for 3 consecutive days.

At the completion of the IV corticosteroid therapy, the level of vision in the right eye was finger counting at 50 cm, but there was still no direct

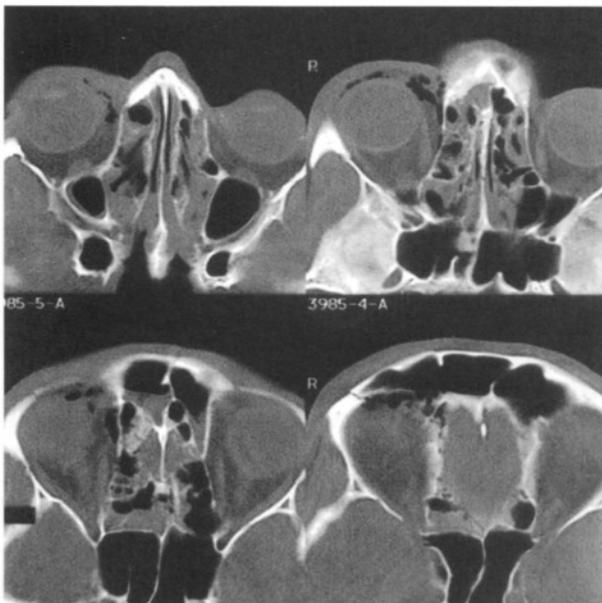


Figure 1: Orbital computed tomography demonstrates a right orbital roof fracture and pneumoorbita.

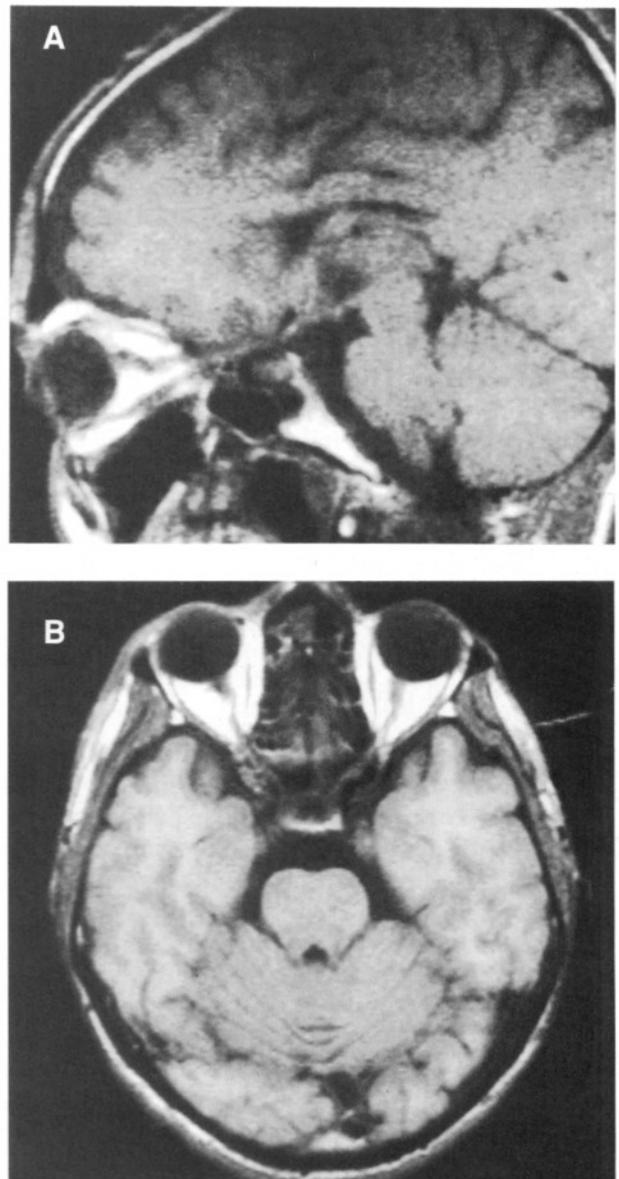


Figure 2 a,b: Sagittal (a) and axial (b) T1-weighted magnetic resonance images. The right optic nerve is intact.

pupillary light reflex in the right eye. The patient was discharged from hospital on 60 mg oral prednisolone daily. The oral steroids were tapered slowly, and were discontinued at 2 weeks after the accident. Three weeks after the injury, the visual acuity in the right eye was 2/10 on the Snellen chart. The eye had no color vision, and there was still no change in the right afferent pupillary defect. Ophthalmoscopic examination revealed pallor of the temporal aspect of the right optic disc. The patient was examined again 10 weeks post-trauma. At this stage, the visual acuity in the injured eye was still 2/10, and the right afferent pupillary defect and lack of color vision persisted. The ophthalmoscopic examination revealed right optic atrophy.

DISCUSSION

Trauma can have extremely serious effects on the optic nerve. While penetrating injuries can injure the nerve directly, the most common form of traumatic optic neuropathy is indirect, resulting from non-penetrating facial and/or forehead trauma (1,8,11,12,13).

Traumatic optic neuropathy is frequently associated with loss of consciousness, but sometimes occurs with only minor injury to the periorbital area. The severity of visual loss in these cases is only weakly correlated with level of consciousness (9), and there is often no fracture of the optic canal or any other orbital fracture (9,15). Typically, the retina and optic disc appear normal at first, and the only objective finding at presentation is an afferent pupillary defect. Optic atrophy does not become apparent for 3-4 weeks (11).

Our current knowledge of the pathophysiologic mechanisms behind indirect traumatic optic neuropathy is limited. Retrobulbar hematoma can result in optic neuropathy due to pressure from the lesion and the direct effect of blood waste products on the optic nerve (5). Our patient did not have a retrobulbar hematoma, so other pathophysiologic factors must have caused the optic neuropathy in this case.

Panje et al. (13) suggested that frontal head trauma might cause stretching of the optic nerve. In addition, Lessell (9) proposed that ischemia is the pathogenetic mechanism in some cases. Our patient had a fracture of the orbital roof on the injured side. Gross et al. (6) demonstrated that frontal loading focuses stress on the roof of the orbit just anterior to

the optic foramen. Considering this, it is important that all patients with orbital roof fracture be checked for evidence of visual loss due to traumatic optic neuropathy.

While the diagnosis of indirect traumatic optic neuropathy can usually be made with the aid of complete history and examination, optimal management is controversial. The current strategies are corticosteroid therapy, surgical decompression of the optic canal, and observation (4,7,9,10,11,14). Administering megadoses of corticosteroids within 8 hours of injury has been shown to reduce paralysis and other disabilities, but does not alleviate neurologic deficits in patients with spinal cord injuries (2). Large doses of corticosteroids slow the process of cellular degeneration, reduce swelling in damaged tissue, and increase the blood supply to the injured area which, in turn, decreases cell damage secondary to ischemia and hypoxemia (3). Accordingly, when megadose corticosteroids are used to treat traumatic optic neuropathy, it is suggested that they may decrease intraneural and extraneural edema, and reduce compression of the optic nerve fibers, thus increasing blood supply and salvaging some of the nerve fibers (15).

In a series of 36 patients who sustained indirect injury to the optic nerve, Seiff (14) reported that visual acuity improved in 62% and 33% of those given high-dose intravenous dexamethasone and those given no treatment, respectively, with the difference being insignificant; however, the steroid-treated patients' vision improved at a significantly faster rate.

Spoor and colleagues (15) compared results in patients treated with megadose IV methylprednisolone to outcomes in patients treated with high-dose dexamethasone. There was no significant difference between the two groups in terms of improved visual acuity. In addition, they found no statistical associations between the visual improvement that was achieved and total blindness at presentation, mechanism of injury, or time elapsed from injury to treatment. However, Steinsapir and Goldberg (16) suggested that megadose corticosteroid therapy must be started within 8 hours, which makes traumatic optic neuropathy a true ophthalmic emergency.

Wollin and Lavin (17) described four patients with traumatic optic neuropathy who recovered vision after having presented with total blindness. Three of these individuals were treated with

corticosteroids, and one improved spontaneously. The authors emphasized that even total blindness does not rule out recovery of vision, and that treatment should always be attempted (15).

Since the intracanalicular segment of the optic nerve is a common site of injury, surgical decompression of the optic canal may be a reasonable treatment option (9). This can be accomplished by either the transcranial or extracranial (transthemoidal and transsphenoidal) route. Joseph et al. (7) reported that optic canal decompression through external ethmoidectomy combined with perioperative dexamethasone treatment yielded favorable results in 11 of 14 patients, including 3 of 5 who had no light perception initially. Similarly, Levin and coauthors (10) reported favorable results in patients with indirect traumatic optic neuropathy who were treated with transthemoidal decompression of the optic canal in addition to perioperative steroids. Since the time elapsed from injury to surgery, initial visual acuity, and presence of an optic canal fracture did not influence outcome, they recommended that treatment be considered in patients with no light perception, and in cases where there is a delay between injury and treatment. Mine et al. (12) compared the outcomes for 34 patients with indirect optic neuropathy who were managed with and without surgical decompression. In the cases where initial visual acuity was hand movement or better, they found that vision improvement was significantly greater when surgery was performed.

Cook and co-workers (4) performed meta-analysis on a large group of published cases of traumatic optic neuropathy. They noted that recovery of vision was significantly better in treated patients than in non-treated individuals. There was no significant difference in improvement among patients treated with corticosteroids alone, those treated with combined surgical decompression and corticosteroids, and those treated with surgical decompression alone. The results also showed that degree of recovery was highly dependent on the severity of initial injury. Further, they noted better recovery in cases where there was no orbital fracture than in cases with these fractures, and better recovery in cases where the fracture was anterior as opposed to posterior.

The International Optic Nerve Trauma Study is a recently published comprehensive prospective study on traumatic optic neuropathy. This investigation compared visual outcomes in 133

traumatic optic neuropathy cases that were treated with either corticosteroids or optic canal decompression surgery, or observed without treatment (11). The findings revealed no significant differences in outcome among the three groups. The authors also reported that dosage of corticosteroid therapy, the timing of this treatment, and the timing of surgery were not associated with increased probability of visual improvement. On these bases, they concluded that neither steroids nor decompression surgery should be considered as a part of standard care for patients with traumatic optic neuropathy.

In the case described in this report, we used megadose methylprednisolone to treat optic neuropathy. There was no significant change in the patient's condition initially, but marked improvement in visual acuity occurred within 3 weeks. Similarly, Seiff (14) noted that corticosteroid-treated patients may not begin to improve until 5 days after treatment is started.

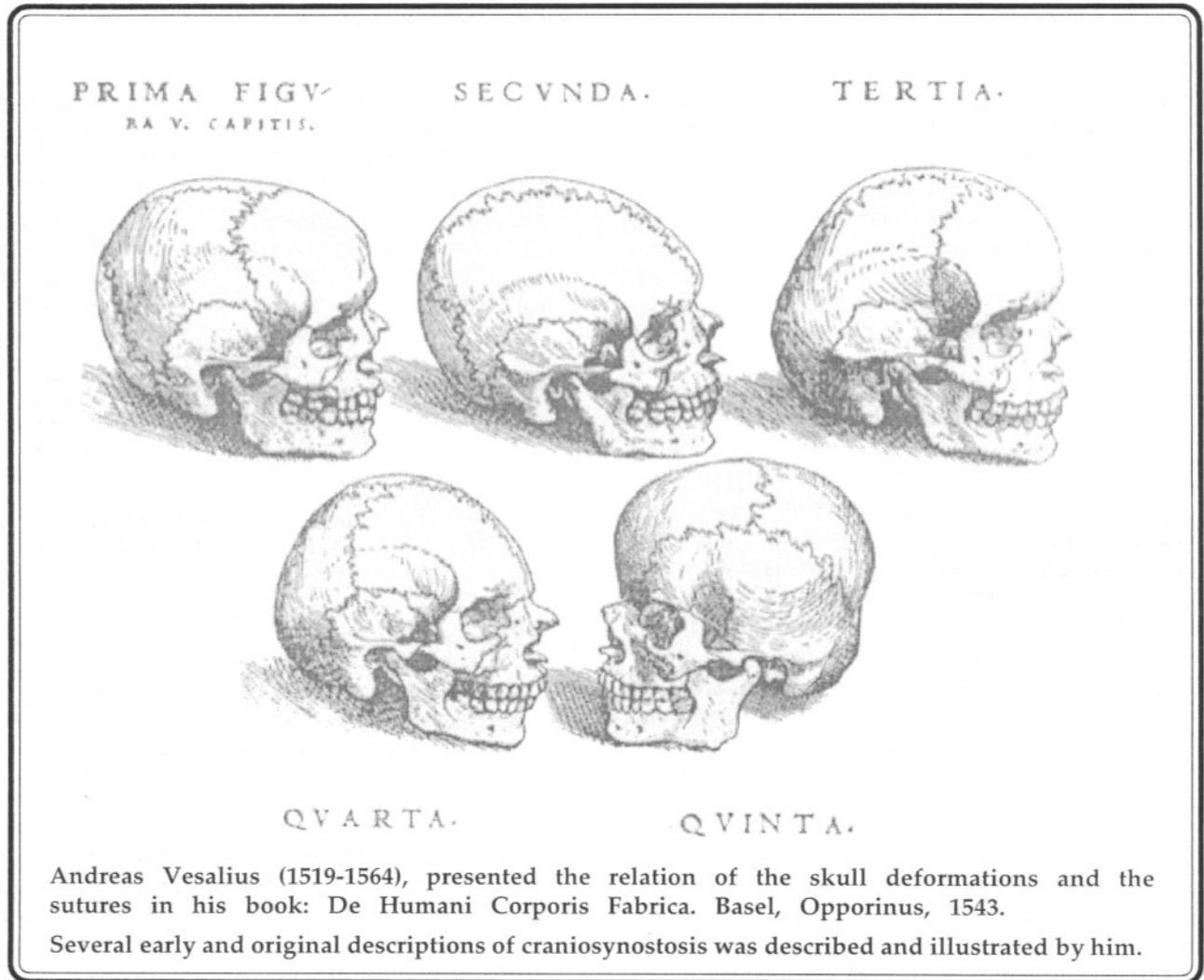
In conclusion, traumatic optic neuropathy associated with blunt forehead trauma is a potentially vision-threatening process that requires comprehensive clinical assessment and appropriate neuroimaging. The treatment options are corticosteroid therapy, surgical decompression of the optic canal, or observation alone. It seems clinically reasonable to decide whether to treat or to strictly observe on a case-by-case basis.

Correspondence: Doç. Dr. Esin Erkin
Mithatpaşa Cad. No: 144/9
35260 Karataş İZMİR
Tel: 0-236-2349070
Fax: 0-236-2370213
E-mail: esinerkin@hotmail.com

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Andreas Vesalius (1519-1564), presented the relation of the skull deformations and the sutures in his book: De Humani Corporis Fabrica. Basel, Opporinus, 1543.

Several early and original descriptions of craniosynostosis was described and illustrated by him.