

Successful delayed treatment of the traumatic orbital apex syndrome by nasal endoscopic decompression surgery

Yuhua Tong, Guohai Chen, Fangzheng Jiang, Wencan Wu¹

To report a patient with traumatic orbital apex syndrome, who fully recovered visual and extraocular function following surgery. A 34-year-old male presented with visual and extraocular function disorders in his right eye following traffic accident, who was referred to our hospital 5 weeks after accident. The patient underwent endoscopic optic nerve and orbital apex decompression with topical and systemic application of nerve growth factor and steroids after a failed trial of mega-dose intravenous corticosteroids. Visual acuity improved to 20/20 at 3 weeks after surgery, and the right eye globe moved in most directions at 1 year, which remained stable at 3 years. Surgical decompression should be considered even when symptoms have been present for over a month.

Key words: Endoscopic, orbital apex syndrome, surgery, trauma

Access this article online

Website:

www.ijo.in

DOI:

10.4103/0301-4738.171005

Quick Response Code:



Traumatic orbital apex syndrome (OAS) is a complex disease that combines features of the superior orbital fissure syndrome with traumatic optic neuropathy. We report a case of traumatic OAS, which was successfully treated by delayed decompression of impinged nerves, had recovery of visual and ocular function.

Case Report

A 34-year-old male was referred to emergency center of a district hospital with heavy type craniocerebral trauma following traffic accident, presenting with a Glasgow Coma Scale score of 5. He was treated in the Intensive Care Unit (ICU) and Department of Neurosurgery for 5 weeks. After recovering from the serious craniocerebral trauma, he was referred to our hospital for visual and extraocular function disorders in his right eye. The main impact was sustained on the right side of his face. The right best-corrected visual acuity (BCVA) was 20/200 with positive relative afferent pupil defect (RAPD). The pupils were anisocoric 4.5 mm on the right versus 3 mm on the left. He had complete ophthalmoplegia and ptosis on the right side [Fig. 1a]. He also had lost the right corneal reflex and had paresthesias over the right frontal region. The optic media such as the iris, lens, and vitreum were found intact, and the left optic disc was also found intact.

A Humphrey visual field documented the marked tunnel vision in the patient's right eye [Fig. 2a]. Computed tomography (CT) revealed right sided orbital and zygomatic fractures, without obviously optic canal fracture or narrowing

of the superior orbital fissure [Fig. 3a-c]. The diagnosis was made of right-sided traumatic OAS associated with right sided orbital and zygomatic fractures.

There was no clinical improvement after 3 days of pulsed 1-g intravenous methylprednisolone therapy. And then, an endoscopic optic nerve and orbital apex decompression was performed by Dr. Wu.

The patient was placed in a supine position, and the procedure was performed under general anesthesia. A standard endoscopic sphenoethmoidectomy was performed using a 45° endoscope (Karl Storz, Tuttlingen, Germany). Fracture fragment clearly identified in the optic canal to be impinging on the optic nerve [Fig. 3d] was carefully removed. Moreover, bone of the medial wall of orbital apex was carefully removed. The bony optic canal was thinned out with a microdrill from the orbital aperture to the cranial cavity and removed with a microcurette. Afterward, the orbital fasciae in the medial side of orbital apex, the common tendinous ring and the sheaths of optic nerve were incised under the nasal endoscope. Sufficient decompression of the medial part of the apex and the optic canal was accomplished successively. Following surgery, the patient received 5 days of pulsed 1-g corticosteroid therapy.

Postoperatively, vision improved to 20/100 at the day 2, 20/50 at the day 4, 20/40 at the day 5, and 20/20 at 3 weeks

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

For reprints contact: reprints@medknow.com

Department of Ophthalmology, Quzhou People's Hospital, Quzhou, Zhejiang 324000, ¹Department of Orbital and Oculoplastic Surgery, Eye Hospital of Wenzhou Medical University, Wenzhou, Zhejiang 325027, China

Correspondence to: Dr. Guohai Chen, Department of Ophthalmology, Quzhou People's Hospital, Quzhou 324000, Zhejiang, China. E-mail: cgh8467@163.com

Manuscript received: 29.03.15; **Revision accepted:** 03.11.15

Cite this article as: Tong Y, Chen G, Jiang F, Wu W. Successful delayed treatment of the traumatic orbital apex syndrome by nasal endoscopic decompression surgery. Indian J Ophthalmol 2015;63:728-30.



Figure 1: Photographs of eye positions in four directions before and after surgery: Primary gaze, supraduction, infraduction, adduction, and abduction (top to bottom). (a) Complete ophthalmoplegia and ptosis can be seen on the right side before surgery. (b) The ptosis was partially recovered, and infraduction and adduction were observed 3 weeks after surgery. (c) The ptosis was recovered, and most ocular movements are markedly improved, except for abduction at 2 months after surgery. (d) Full range of eye movement in all direction of gaze was seen 1 year after surgery

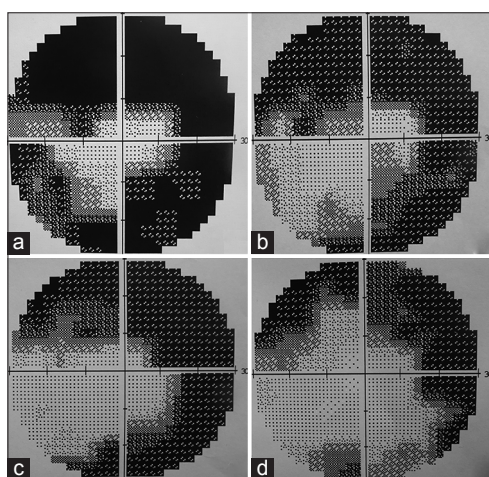


Figure 2: Humphrey visual fields of the right eye. (a) The right eye with significant constriction of the visual field before surgery. (b-d) Humphrey visual fields of the right eye after surgery at 3 weeks, 1 year, and 3 years, respectively. The visual field was improved, which remained stable at 3 years, but with slightly restricted

after the operation. Three days after surgery, a right corneal reflex was elicitable, and a prompt pupillary light reflex was detected with negative RAPD. His left eyelid ptosis was partially recovered at 3 weeks postoperative and completely recovered at 2 months [Fig. 1b and c]. The loss of sensation over his right forehead also completely recovered at 2 months. His right eye globe started to demonstrate infraduction and adduction at 3 weeks after surgery [Fig. 1b]. Most ocular

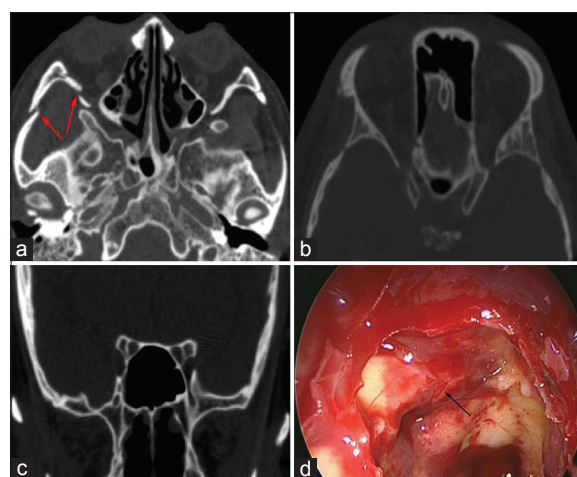


Figure 3: Preoperative computed tomography and a clinical photograph of the optic nerve prior to decompression. (a) An axial orbital computed tomography scan showing a right-sided orbital and zygomatic fractures (red arrow). (b and c) An axial orbital computed tomography and a coronal section computed tomography scan illustrating that there was no obviously optic canal fracture or narrowing of the superior orbital fissure. (d) Fracture fragments clearly identified in the optic canal to be impinging on the optic nerve (black arrow)

movements are markedly improved, except for abduction at 2 months after surgery, and full range of eye movement in all direction of gaze was seen at 1 year [Fig. 1c and d]. Humphrey visual fields documented that the visual field was improved, which remained stable at 3 years, but with slightly

restricted [Fig. 2b-d]. Although the right optic disc found to be pale and the visual field was slightly restricted, he ultimately regained BCVA of 20/20 with full eye movement and negative RAPD for the right eye and remained stable at 3 years.

Discussion

OAS is a complex disease caused by a variety of pathological factors, such as trauma, foreign bodies, fungi, cephalic and facial infections, and tumor invasion of the orbital apex.^[1] This case illustrates all of the hallmarks of an OAS, which include visual loss from optic neuropathy, ophthalmoplegia resulting from multiple cranial nerves (oculomotor, trochlear, and abducens), and numbness in the territory of the ophthalmic branch of the trigeminal nerve.^[1,2]

The conditions of patients with traumatic OAS are relatively complicated, and most of the patients may have a history of coma. As a result, symptoms of OAS are usually neglected in the early stage, leading to delayed diagnosis and treatment. This case was presented with a Glasgow Coma Scale score of 5, and treated in the ICU and Department of Neurosurgery for 5 weeks. In order to diagnose traumatic OAS in the early stage, the visual acuity and eye movement should be checked immediately after the patient recovered consciousness.

The rarity of traumatic OAS has made it difficult to define treatment guidelines for this condition. Gossman *et al.*^[3] proposed classification of traumatic optic neuropathy into direct and indirect categories. This same distinction may be helpful when applied to the management traumatic OAS as the treatment strategy is quite different between direct and indirect injuries, in terms of the indications for, and urgency of surgery. Surgical intervention is recommended for the direct type of traumatic OAS.^[4]

In this case, CT revealed right-sided orbital and zygomatic fractures, without obviously optic canal fracture or narrowing of the superior orbital fissure. The diagnosis was first made of indirect type of the right traumatic OAS. In a series of cases of traumatic superior orbital fissure syndrome, Chen and Chen^[5] reported that the patients treated with steroids have a better

chance of neurologic recovery than those with observation alone (70% vs. 42.1%). We opted to use mega-dose steroid protocol to reduce swelling of the impinged nerves, in this case, but there was no clinical improvement after 3 days of pulsed 1-g intravenous methylprednisolone therapy. And then, an endoscopic optic nerve and orbital apex decompression was performed. Fracture fragment was clearly identified in the optic canal to be impinging on the optic nerve during the operation, which indicated that some optic canal fractures might be missed in CT. Following surgery, the patient fully recovered visual and extraocular function.

The treatment for traumatic OAS is controversial, but endoscopic optic nerve and orbital apex decompression could be considered in patients who fail to respond to corticosteroids. This case suggests that managing the traumatic OAS with a decompression of affected nerves is an effective strategy. Surgical decompression should be considered even when symptoms have been present for over a month.

Financial support and sponsorship

Medical Science and Technology Program of Zhejiang Province (2012KYB231) (YH Tong).

Conflicts of interest

There are no conflicts of interest.

References

1. Yeh S, Foroozan R. Orbital apex syndrome. *Curr Opin Ophthalmol* 2004;15:490-8.
2. Imaizumi A, Ishida K, Ishikawa Y, Nakayoshi I. Successful treatment of the traumatic orbital apex syndrome due to direct bone compression. *Craniomaxillofac Trauma Reconstr* 2014;7:318-22.
3. Gossman MD, Roberts DM, Barr CC. Ophthalmic aspects of orbital injury. A comprehensive diagnostic and management approach. *Clin Plast Surg* 1992;19:71-85.
4. Li Y, Wu W, Xiao Z, Peng A. Study on the treatment of traumatic orbital apex syndrome by nasal endoscopic surgery. *Eur Arch Otorhinolaryngol* 2011;268:341-9.
5. Chen CT, Chen YR. Traumatic superior orbital fissure syndrome: Current management. *Craniomaxillofac Trauma Reconstr* 2010;3:9-16.