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Traumatic orbital third nerve palsy

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Abstract

We present a case series of three patients who developed acute traumatic orbital third nerve palsies. To our knowledge, reported cases have mainly been localised to the intracranial course of the nerve and often associated with visual impairment. Those in which the orbit is the site of injury are rare. Our case series highlights the importance of careful preoperative assessment of patients with orbital trauma (particularly when there is a coexisting fracture) and the need to assess ocular movements and pupillary reactions to distinguish between a neurogenic and soft tissue injury. Early diagnosis is helpful in deciding on the timing of the operation and enables patients to be given appropriate counselling to make sure that their expectations are realistic.

Keywords: Orbital trauma; third nerve palsy

Introduction

Traumatic third nerve palsies are usually sustained in severe, high-speed, closed-head injuries, and are often associated with other cranial nerve injuries and neurological deficits.[1] They are rare, with a recorded incidence of 1.1% - 1.2%,[2] and those purely of an orbital origin have been reported in only a handful of cases.[2–5] To our knowledge, this is the first case series of isolated traumatic third nerve palsies associated with localised orbital trauma.

Method

This is a retrospective case series of patients who were managed by the multidisciplinary orbital service of a regional trauma centre. Data collected from electronic medical records and case notes included ophthalmic, orthoptic, and maxillofacial assessments, and radiological reports. The series adheres to the tenets of the Declaration of Helsinki as amended in 2008.

Results

Case 1: superior division third nerve palsy (Fig. 1)

This 32-year-old man was assaulted and lost consciousness at the time of the injury. A computed tomogram (CT) of the orbit on the day of injury showed fractures of the right orbital floor and medial wall, with no sign of fracture in the base of the skull, or brain injury. He presented to our unit one week after injury with ptosis. On examination he had right complete ptosis and restricted ocular motility consistent with a diagnosis of a right partial (superior branch) third nerve palsy. His visual acuity was 6/7.5 OD 6/5 OS at presentation. He had severe enophthalmos and the anterior segment showed tears in the right iris sphincter that made it difficult to assess involvement of the pupil. Examination of the fundus was normal.

To avoid exacerbating the neurogenic injury, the repair of the fracture with a preformed titanium plate was delayed for

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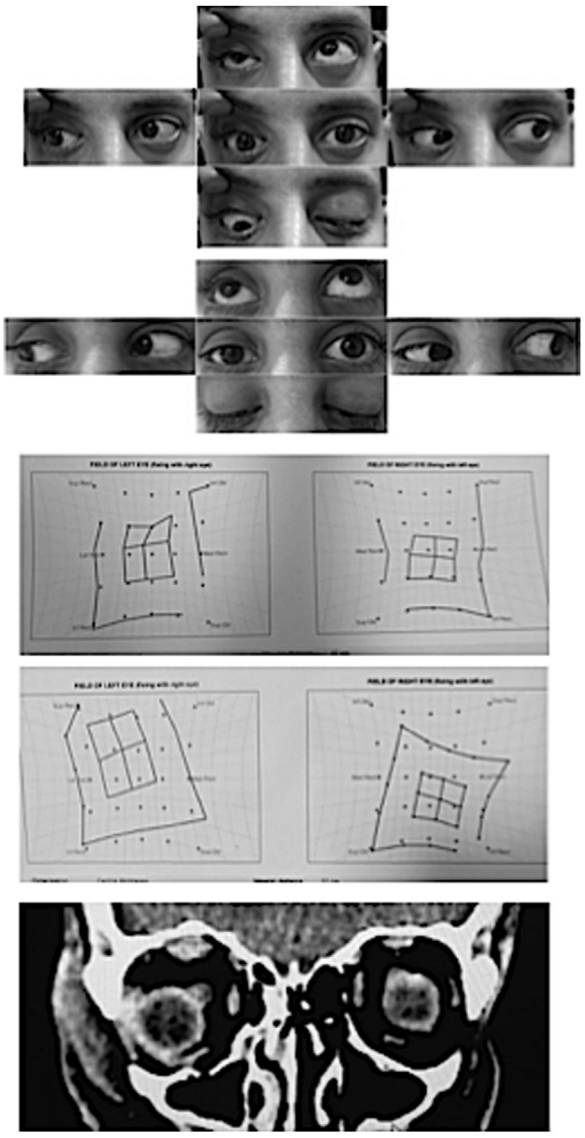

Fig. 1. Case 1 (from top): eye movements at presentation and 4 months after injury, Hess chart at presentation, and 4 months after injury, computed tomogram of the orbits at presentation.

two months. Ten months after injury, however, he still has residual ptosis and symptomatic diplopia, and remains under review.

Case 2: inferior division third nerve palsy (Fig. 2)

This 43-year-old man was assaulted, but did not lose consciousness at the time of injury, and presented to our unit three days later with diplopia. He did not have left ptosis but had restricted left ocular motility and a dilated pupil, consistent with left partial (inferior branch) third nerve palsy. Visual acuity was 6/6 OU and examination of the anterior segment and fundus was otherwise normal. CT showed fractures of the left orbital floor and zygoma but no fracture of the base of the skull, or brain injury.

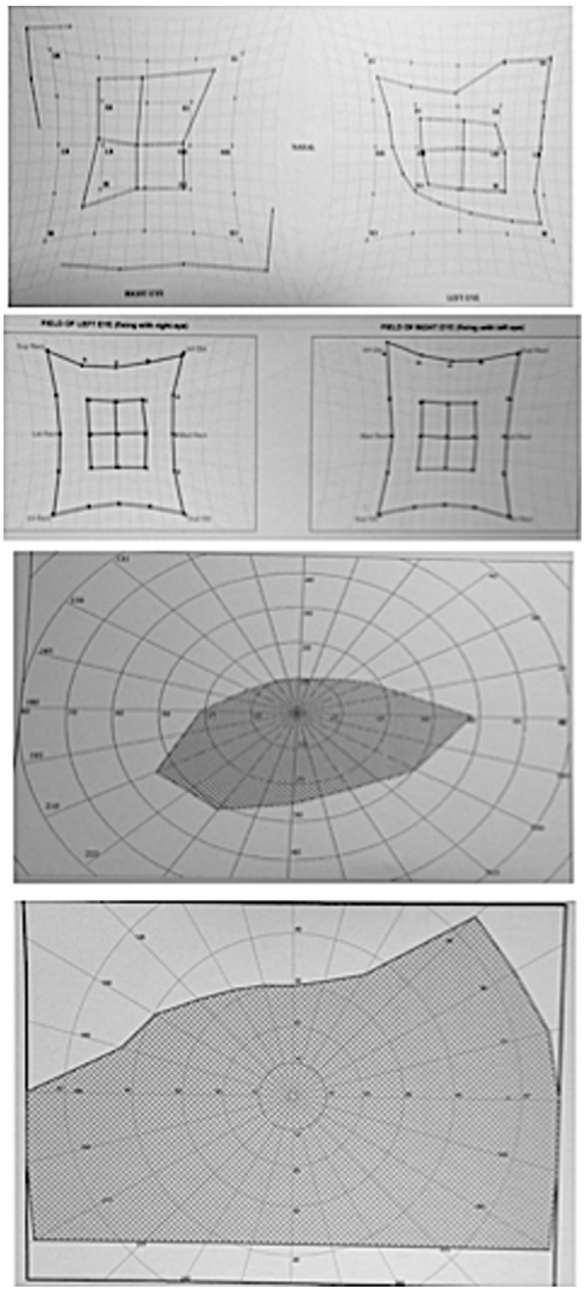

Fig. 2. Case 2 (from top): Hess chart at presentation and 5 months after injury, field of binocular single vision at presentation and 5 months after injury.

In view of the zygomatic fracture he had the left orbital and zygomatic fractures repaired two weeks after injury. His diplopia gradually improved and the pupil recovered. He was discharged from clinic review five months after injury as his diplopia no longer interfered with his daily life.

Case 3: inferior division third nerve palsy (Fig. 3)

This 18-year-old man was assaulted but did not lose consciousness at the time of injury, and presented to our unit on the day of his injury with diplopia and loss of vision. His

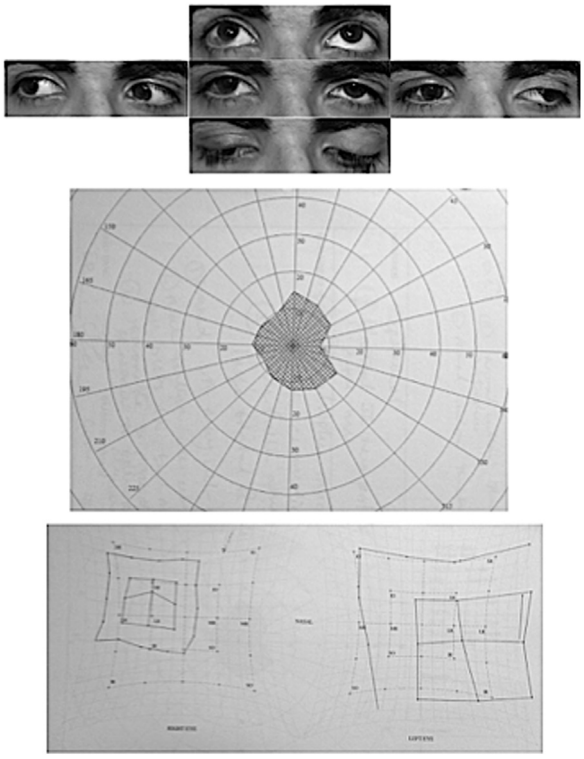

Fig. 3. Case 3 (from top): eye movements, Hess chart, and field of binocular single vision at presentation.

visual acuity was 6/18 OD 6/4 OS. He did not have ptosis but had restricted right ocular motility with a dilated pupil, consistent with right partial (inferior branch) third nerve palsy. Fundoscopy showed commotio retinae in the right posterior pole, which accounted for the reduction in vision. CT showed fractures of the right orbital floor and medial wall with no evidence of a fractured skull base or brain injury.

The fractures were repaired with a preformed titanium plate two weeks after injury. His diplopia and dilated pupil resolved completely, but unfortunately the commotio retinae resulted in permanent damage to the foveal photoreceptors. His right vision did not recover, and remained at 6/18 OD. He was discharged from clinic review six months after injury.

Discussion

Trauma accounts for roughly 12% of all third nerve palsies, and damage may be caused directly or indirectly.[6] The oculomotor nerve originates from the nucleus of the third nerve in the midbrain. Fibres from the third nerve nuclei pass either side of the cerebral aqueduct through the red nucleus, and exit by way of the substantia nigra through the interpeduncular fossa. On emerging from the brainstem, the nerve passes between the superior cerebellar and posterior cerebral arteries. It pierces the dura mater anterior and lateral to the posterior clinoid process, and passes between the free and attached borders of the tentorium cerebelli. It traverses the cavernous sinus, and divides into superior and inferior branches before entering the orbit through the superior orbital fissure. The superior branch supplies the levator palpebrae superioris and superior rectus. The inferior branch supplies the medial rectus, inferior rectus, and inferior oblique, and carries the parasympathetic fibres that supply the sphincter pupillae and ciliary muscle.[7]

Indirect injury by compression, haemorrhage, and ischaemia, may affect the nerve at multiple points in its course. Direct trauma is most common at certain vulnerable points, including the exit from the brainstem and the point at which it crosses the petroclinoid ligament.[8] Direct injury or indirect compression at the orbital apex may result in a partial or complete third nerve palsy.[3] This is likely to be the pathogenesis in all the cases in this series. Although one patient reported losing consciousness for a short period immediately after the injury, none had radiological signs of brain injury or fractured skull base.

Complete third nerve palsies typically present with complete ptosis (caused by paralysis of the levator palpebrae superioris), mydriasis (caused by compression of the parasympathetic fibres that supply the sphincter pupillae), and reduced ocular motility. The eye adopts a "down and out" position because of the preserved action of the lateral rectus and superior oblique, which are supplied by the abducens and trochlear nerves, respectively. Partial third nerve palsies have some or all of these features to a lesser degree.

At first presentation it is often difficult to diagnose a third nerve palsy in the context of concurrent orbital fractures. Up to 86% of patients with orbital fractures have diplopia at first presentation, as pain, soft tissue swelling, and entrapment of fat or muscle in the fracture often result in ptosis and restricted ocular motility.[9,10] Most cases settle spontaneously or within four weeks of repair, and only 5.56% report residual diplopia.[11] In general, patients with traumatic third nerve palsies have a poorer prognosis. The severity of the symptoms and the prognosis vary with the degree of damage. Ho et al reported that patients with paralytic strabismus secondary to traumatic cranial nerve palsies had a median recovery time of 10 months, compared with three months for those of a vascular origin, and four months for idiopathic cranial nerve palsies.[12] Kuo et al reported a recovery rate for ptosis and external ophthalmoplegia after a traumatic third nerve palsy of 100% and 60%, respectively.[13] Many patients, however, adopt head positions that minimise diplopia and enable them to continue their daily activities. Patching, prisms, and chemodenervation of the lateral rectus with botulinum toxin are all temporary measures that may help to increase the field of binocular single vision during recovery. Strabismus surgery is generally not done within the first six months to allow for maximum spontaneous recovery.[14]

Patients with a suspected third nerve palsy require thorough ophthalmic assessment. Mydriasis, which presents with an enlarged pupil that reacts poorly to light, may be shown by careful assessment of the pupillary reactions and comparison of pupil size in light and darkness. Ptosis secondary to third

nerve palsy presents with a reduced height of the upper lid and appreciably reduced or absent levator function. Assessment of ptosis should include measurements of the palpebral fissure (distance between the margins of the upper and lower lids), margin-reflex distance (distance between the margin of the upper lid and pupillary light reflex), and levator function (distance between the position of the lid at upgaze and downgaze).

Careful assessment of ocular motility, with forced duction testing if required, will differentiate between a neurogenic and mechanical pattern of ocular restriction. An orthoptic review with formal documentation of eye movements and diplopia using a Hess chart and field of binocular single vision will aid diagnosis and provide an objective baseline with which to monitor recovery. Imaging should be done in all patients to exclude other causes of traumatic third nerve palsy such as brain injuries, fractures of the skull base, carotid cavernous fistulas, and disease of the cavernous sinus.[15]

In conclusion, although isolated third nerve palsies are rare complications of orbital trauma, their possibility should be considered in patients with orbital fractures, even if the injury is mild. Careful preoperative assessment of ocular movements and pupillary reactions will help to distinguish between a third nerve palsy and soft tissue injury, and will enable patients to be counselled appropriately about their prognosis and the time to recovery. Surgeons should take neurogenic injury into consideration when planning the repair of orbital and facial fractures.

Conflict of interest

We have no conflicts of interest.

Ethics statement/confirmation of patients' permission

This case series was approved by the hospital's research ethics department. We were unable to contact the patients for written consent to use their clinical photographs. We have therefore anonymised details and cropped all clinical photographs to prevent identification.

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