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Case Report

Isolated unilateral oculomotor nerve palsy following a mild head injury

Lt Col Avinash Mishra ^{a,*}, Somesh Aggarwal ^b, Lt Col Nitin Vichare ^c,
Lt Col Anirudh Singh ^d

^a Classified Specialist (Ophthalmology), Military Hospital, Ahmedabad, India

^b Associate Professor, M & J Western Regional Institute of Ophthalmology, Ahmedabad, India

^c Classified Specialist (Ophthalmology), Command Hospital (Southern Command), Pune 411040, India

^d Classified Specialist (Ophthalmology), Army Hospital (R&R), Delhi Cantt, India

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Introduction

Cranial nerve paralysis following severe head injuries are very common indeed, however their being involved following mild injuries are rare.^{1,2} Here we report one such case of isolated oculomotor nerve palsy (ONP) following a mild head injury. Isolated ONP due to closed head trauma is in itself uncommon, with an incidence ranging from 0 to 15%³ However its involvement following only a mild trauma is extremely rare, and which to the best of our knowledge has been reported of only a very few times earlier in literature.^{2–5}

Case report

A 24 year old male patient presented with a history of sustaining a lacerated wound on his left eyebrow due to a fall from his motorcycle one day back. There was no history of any loss of consciousness or vomiting post injury. Since the patient was on leave at the time of his accident, he received the initial first aid treatment at a nearby local hospital where his eyebrow wound was sutured. The very next day he reported to this centre. General examination on presentation revealed a normal blood pressure (120/78 mm of Hg) and pulse rate 74/min. His Glasgow Coma Scale (GCS) was 15 with no other sign of any associated cranial/cervical injury. There was also no raccoon eye, battle's sign, rhinorrhea or otorrhea noted. Ocular examination showed a well sutured left eyebrow wound with no other sign of any local ocular injury; however he had complete ptosis in the left eye. The best corrected visual acuity was a normal 6/6, measured after lifting his eyelid. The left eyeball was displaced outwards and downwards (infraducted and abducted). The ocular movements were restricted, with absence of adduction and elevation; however abduction was full. Depression was accompanied by intorsion, maximally when the eye was abducted. The pupil was dilated (6 mm) and non-reactive to light (Fig. 1a–b). Fundus was essentially normal. The right eye was uninvolved with a normal vision, range of movement and a normal sized, reactive pupil. The patient was subjected to the necessary laboratory investigations,

* Corresponding author. Tel.: +91 (0) 9408330655.

E-mail address: avim27@yahoo.com (A. Mishra).

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Fig. 1 – (a) Complete ptosis. (b) Ocular movements (Left eye) on presentation – All ocular movements grossly restricted except abduction. Left pupil too fully dilated.

however they were all normal (Table 1). Computed tomography (CT) of the head and orbits was normal except for bulky belly of left lateral rectus muscle, due a suspected haematoma. It did not reveal any causative cerebral and vascular lesion or orbital and cranial fractures. The patient was further evaluated with a Magnetic Resonance Imaging

Table 1 – Laboratory investigations carried out on presentation and their results.

Serial no	Investigation	Result
1.	Haemoglobin	13.5 g/dl
2.	Total Leucocyte count	$9 \times 10^9/L$
3.	Differential Leucocyte Count	Neutrophils: $5.5 \times 10^9/L$ Lymphocytes: $2.5 \times 10^9/L$ Monocytes: $0.6 \times 10^9/L$ Eosinophils: $0.3 \times 10^9/L$ Basophils: $0.06 \times 10^9/L$
4.	Blood Sugar (Random)	80 mg/dL
5.	Erythrocyte sedimentation rate	<10 mm
6.	Serum cholesterol	221 mg/dL

(MRI) of the brain and orbits, which too confirmed the earlier CT scan findings (Fig. 2a). A diagnosis of isolated ONP following mild head injury was made and he was kept under observation. By the end of three months, the patient was symptomatically much better with his pupil reducing to a normal 3 mm size and showing a normal reaction to light. The ocular movements too became full and free except for a mild restriction in extreme adduction. However mild ptosis still persisted (Fig. 3). A repeat MRI at this stage too revealed a total resolution of his left lateral rectus muscle swelling (Fig. 2b). He is presently being followed up regularly as an outpatient (Fig. 4).

Fig. 2 – (a) MRI Brain and Orbits – Revealing a bulky belly of left lateral rectus muscle. (b) MRI Brain and Orbits (3 Months later) – Revealed a total resolution of his left lateral rectus muscle swelling.

Fig. 3 – Mild residual ptosis, ocular movements (Left eye) 3 months post injury. All ocular movements full and free except mild restriction in adduction in extreme gaze. Left pupil too normal in size and reaction.

Discussion

The oculomotor nerve is a somatic and visceral motor nerve which innervates the levator palpebrae superioris and most of the ocular muscles (except the lateral rectus and the superior oblique muscles). With its parasympathetic fibres, it innervates the iris constrictor and the annular portion of the ciliary muscle. When injured, it results in ptosis, pupils that are non-reactive to light and restricted ocular movements.⁴ ONP may be congenital or acquired, complete or partial, pupil-sparing or pupil-involving, isolated or accompanied by signs of more extensive neurological involvement. Examination of the pupils helps in differentiating between the surgical from the medical causes of ONP, with them being involved in surgical causes like trauma, compressive lesions or posterior communicating artery aneurysm, with or without subarachnoid haemorrhage. While pupil-sparing third cranial nerve palsies are usually medical in nature eg. ischaemic lesions.⁶

Fig. 4 – Mild residual ptosis.

Traumatic ONP is mostly seen in severe head injuries along with associated skull or cervical spine fractures and intracranial injuries.⁷ When faced with such a case initially all other additional injuries should be ruled out by a thorough clinical/neurological examination, followed by urgent imaging (CT scan, MRI).¹ However as no lesion other than a suspected haematoma in the lateral rectus muscle was detected, further aggressive intervention was not contemplated and the patient was managed with just a regular follow-up.

The actual mechanism of damage to the oculomotor nerve from minor head trauma associated with negative brain imaging studies is still not clear.² One study suggested that the downward displacement of the brainstem at the time of impact directly injured the pupillomotor fibres on the ventromedial surface of the third nerve at the posterior petroclinoid ligament.⁵ While another study postulated that in addition to mechanical injuries such as rootlet avulsion and distal fascicular damage, disturbances in blood supply or adverse biochemical effects arising from head injury may injure the cranial nerves.³

Conclusion

The prognosis of traumatic ONP is very unpredictable and recovery is slow and prolonged, with full recovery being very uncommon.^{1–3} Previous studies have revealed that the recovery rate of ptosis was rapid and high (95%–100%), while it was lesser for external ophthalmoplegia, and least for internal ophthalmoplegia.^{7,8} However isolated ONP after mild head injury being rare, its clinical and functional outcome has not been evaluated in detail. It has also been suggested that these patients in whom no lesion is demonstrated on the initial CT scan have a good prognosis, with a greater chance of recovery⁹ as was observed in this case, who showed a nearly complete recovery within such a short period.

Normally a period of 6–12 months should be allowed for spontaneous recovery to occur before any surgical intervention is considered. During this observational period if the patient suffers from severe diplopia then the temporary options available includes occlusion of an eye with pads, wearing prism lenses or even botulinum toxin injection into the lateral rectus muscle. Strabismus surgery should only be contemplated when there is no further improvement.¹⁰ However due to involvement of multiple extraocular muscles; this is extremely difficult.³

Conflicts of interest

All authors have none to declare.

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