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Case Report

Delayed and isolated oculomotor nerve palsy following minor head trauma

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Abstract

Background: The purpose of this study was to consider the mechanism of isolated oculomotor nerve palsy after minor head trauma.

Case Description: We report a rare case of delayed and isolated oculomotor nerve palsy following minor head trauma. A 19-year-old boy complained of double vision 1 day after a minor head trauma. Neuro-ophthalmic examination showed isolated left oculomotor nerve palsy. Computed tomography and magnetic resonance imaging examination revealed no abnormal findings and steroid therapy was administered for a week. Three months after the injury, the ptosis and extraocular movements had fully resolved, although the pupillary light reflex was still abnormal.

Conclusions: Delayed and isolated oculomotor nerve palsy may be caused by an injury at the point where the oculomotor nerve runs over the posterior petroclinoid ligament. Because edema of the damaged oculomotor nerve might result in constriction at the point where the nerve pierces the dura of the cavernous sinus, symptoms of oculomotor nerve palsy appeared late after trauma. Steroid treatment reducing edema could be effective for delayed and isolated oculomotor nerve palsy following minor head trauma.

Key Words: Minor head trauma, oculomotor nerve injury, steroid

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INTRODUCTION

The severity of head trauma is significantly higher in patients with traumatic brain injury (TBI) and cranial nerve injury than in those with intact cranial nerve function, with a greater number of radiological abnormalities on computed tomography (CT) scans and poor neurological outcome.[4] The trauma required to damage the oculomotor nerve is extensive and is usually associated with loss of consciousness, basilar skull fracture, or subarachnoid hemorrhage. Minor head trauma, without loss of consciousness or abnormal findings on radiological imaging, is unlikely to cause cranial nerve palsy.[4,13] Therefore, isolated oculomotor nerve palsy in minor head trauma without an initial loss of consciousness or radiological abnormalities on CT scans is extremely rare.[3,12,20] Therefore, an isolated oculomotor nerve palsy that occurs after minor head trauma should raise concerns about an underlying lesion that could be compressing, stretching, or infiltrating the oculomotor nerve, predisposing it to dysfunction following an otherwise inconsequential

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injury. Previous studies have reported that oculomotor nerve palsy after minor head trauma is the presenting sign of cerebral aneurysmal compression or uncal herniation due to intracranial tumor in some patients.[5,9,18,21] In addition to aneurysms and tumors, other causes of isolated oculomotor nerve palsy include microvascular ischemia, infection, and inflammation. Here, we report a rare case of delayed and isolated oculomotor nerve palsy after minor head trauma, and describe its mechanism and treatment.

CASE REPORT

A 19-year-old boy with an unremarkable medical history suffered a bruise to his occiput during a game of rugby. He did not lose consciousness and continued playing. Next day, he visited the Department of Neurosurgery complaining of headache. The neurological exam and CT was normal [Figure 1]. However, he had noticed drooping of his left eyelid and double vision with onset on the second day; subsequent examination revealed an isolated left oculomotor nerve palsy with a nonreactive pupil [Figures 2-4]. No raccoon eye, Battle's sign, rhinorrhea, otorrhea, or other abnormalities were noted in the neurological exam. CT, magnetic resonance imaging (MRI), and MR angiography showed no abnormal findings [Figure 1]. Oral betamethasone was prescribed at a dose of 8 mg per day for 1 week. Repeat MRI and MRA 1 week later also showed no abnormal findings. Over the next 3 months, the ptosis and ocular motility resolved fully, however, the left pupil was still larger and less reactive to light than the right [Figures 2 and 3].

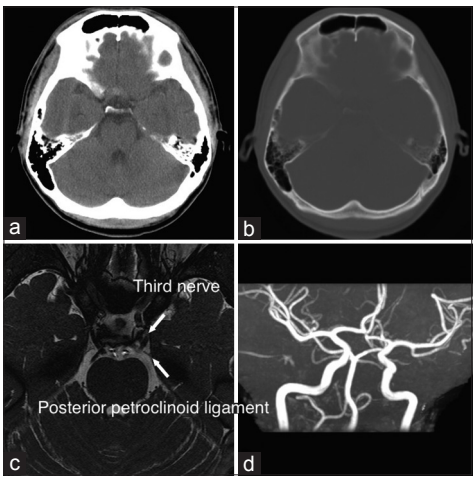

Figure 1: Computed tomography and magnetic resonance imaging. CT and MRI examinations demonstrate no abnormal findings. No intracranial hemorrhage (a) and no basilar fracture (b) are apparent in axial CT images on day 1. An axial heavy T2-weighted MRI image (c) shows the left oculomotor nerve running over the posterior petroclinoid ligament and no apparent damage to the nerve on day 7. No aneurysm is visible in MR angiography (d)

DISCUSSION

Oculomotor nerve palsy may be caused by damage to the nerve at any point along its path between the oculomotor nucleus in the midbrain and the extraocular muscles within the orbit. There are reports of injury to the oculomotor nerve at its exit from the brainstem, from the superior orbital fissure,[8] and at the tentorial shelf after herniation.[11] These reported third nerve injuries were all caused by severe head trauma. In the present case, repeated CT and MRI did not reveal any subtle findings, including hemorrhage at the midbrain exit site of the oculomotor nerve or bending of the ipsilateral oculomotor nerve at the posterior petroclinoid ligament, which have been reported to cause isolated third nerve palsy.[1,10] In addition, absence of other associated signs and symptoms, such as hemiparesis and other cranial nerve palsies, indicated that the damage might not have occurred in the midbrain, cavernous sinus, or superior orbital fissure.

It has been suggested that traumatic oculomotor nerve injury with minor head trauma occurs because of differential movements between the brainstem and supratentorial structures, which can stretch the nerve and cause distal fascicular damage.[15] This mechanism for direct oculomotor nerve injury is strongly related to the anatomy of this nerve. The oculomotor nerve pierces the dura of the cavernous sinus through the oculomotor triangle, consisting of the anterior and posterior clinoid processes and the petrous apex. The medial margin of this triangle is formed by the interclinoid ligament, which extends from the anterior to the posterior clinoid process. The lateral margin is formed by the anterior petroclinoid ligament, which extends from the anterior clinoid process to the petrous apex, whereas the posterior margin is formed by the posterior petroclinoid ligament, which extends from the posterior clinoid process to the petrous apex. The oculomotor nerve runs over the tough posterior

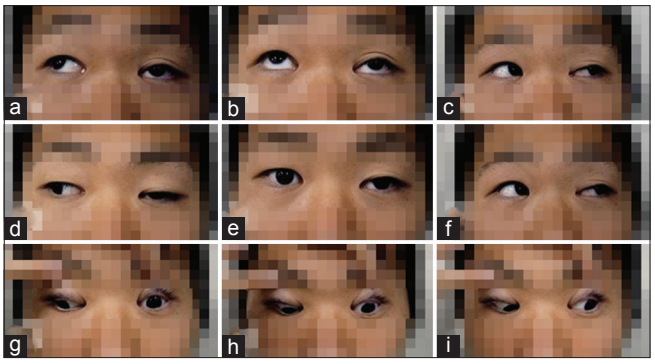

Figure 2: Photographs of the patient's extraocular movements. (a) right supraduction; (b) supraduction; (c) left supraduction; (d) right lateral gaze; (e) mid-position; (f) left lateral gaze; (g) right infraduction; (h) infraduction; (i) left infraduction

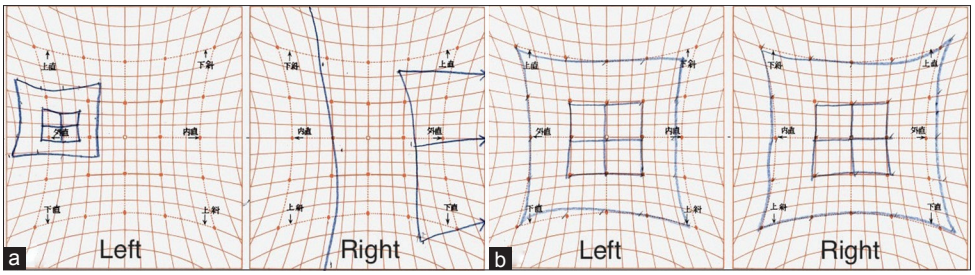

Figure 3: Hess chart on day 1 (a), and 3 months after injury (b)

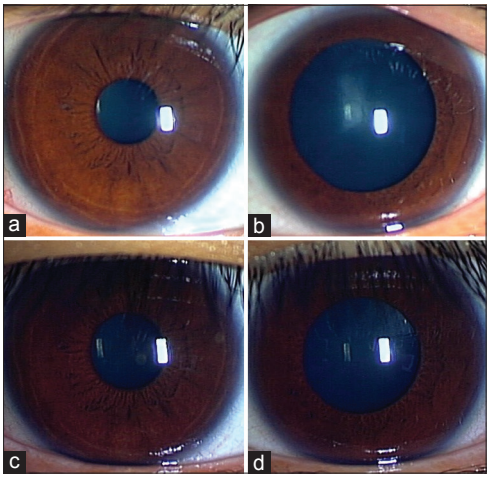

Figure 4: Photograph of the right (a) and left pupil (b) on day 1, and right (c) and left pupil (d) 3 months after injury

petroclinoid ligament. Direct oculomotor nerve injury with minor head trauma may occur at this point, as the brainstem moves downward at the time of injury, the nerve then becomes swollen, and ischemia could be caused by dural constriction at the point where the nerve pierces the dura of the cavernous sinus.[10,16] This could explain why the symptoms appeared a day after head trauma in this case.

Isolated third nerve palsy without any other symptoms such as loss of consciousness or radiological abnormalities is exceptionally rare, this being the 6th case reported in the literature [Table 1].[2,10,15,17,19] Regarding the treatment of third nerve palsy with head injury, only two of the 6 reported cases were treated with steroids. Although the site of injury to the third nerve was not clearly demonstrated, the suggested mechanism described above is supported by the effectiveness of steroid treatment for third nerve injury with minor head trauma, similar to the treatment rationale in patients with delayed facial nerve palsy. The facial canal is at its narrowest in the labyrinthine segment; therefore, any swelling of the facial nerve would cause compression, and the meager blood supply would add to the damage. Hence, steroid therapy has been proposed to help alleviate delayed facial palsy.[6,7,14]

Prism therapy and strabismus surgery may be helpful in patients with oculomotor nerve palsy. However, indications for these treatments and their effectiveness are limited. Until now, there is no satisfactory surgical treatment for patients with complete oculomotor nerve palsy.

Table 1: Literature review of reported cases of isolated oculomotor nerve palsy following minor head trauma

Case no.	Authors and year	Sex; age (years)	Abnormal findings on CT/MRI	Steroid treatment	Diplopia
1	Muthu, *et al*., 2001	F; 52	No	No	Remained
2	Chen, *et al*., 2005	F; 42	No	No	Remained
3	Kaido, *et al*., 2006	M; 11	No	No	Recovery
4	Takeuchi, *et al*., 2008	F; 65	No	Yes	Recovery
5	Najafi, *et al*., 2012	M; 40	No	No	Remained
6	Present case	M; 19	No	Yes	Recovery

CONCLUSION

We report that minor head trauma can cause isolated oculomotor nerve palsy in the absence of loss of consciousness and abnormalities on brain CT, MRI, and MRA. The mechanism underlying oculomotor nerve injury following minor head trauma is still unclear, although injury at the posterior petroclinoid ligament is suggested. Steroid therapy may possibly be effective for functional recovery.

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Conflicts of interest
There are no conflicts of interest.

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