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• Case Report •

Bilateral Oculomotor Nerve Palsy after Head Trauma: A Case Report

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We introduce a patient who was suffered from isolated traumatic bilateral oculomotor nerve palsy after head trauma. The patient presented with bilateral ptosis and abnormal pupilary responses with slightly drowsy mentality at first. Performed images demonstrated some hematomas along subarachnoid, intraventricular, subdural spaces and multiple small supratentorial contusions. There was no bony abnormality or ligament injury. We assumed that small amount of interpeduncular hematoma might be the proper lesion associated with oculomotor nerve palsies, since the clinical symptom and signs presented bilaterally and the oculomotor neural fascicles run through the interpeduncular fossa. [J Trauma Inj 2017; 30: 66-69]

Key Words: Bilateral, Oculomotor nerve palsy, Head trauma

I. Introduction

Cranial nerve palsies occurred after severe head trauma. Injury of the cranial nerve is the result of imported kinetic forces to brain and cranium.(1) Especially, traumatic oculomotor palsy is usually associated with severe traumatic brain injury, basilar skull fracture, orbital injury or subarachnoid hemorrhage.(2) However, isolated traumatic oculomotor nerve palsy without other cranial nerve palsies is uncommon on the literature review. Furthermore, traumatic isolated bilateral oculomotor nerve palsy is extremely rare whereas most previous studies have reported unilateral cases.(3)

II. Case Report

A 59-year-old female visited our institute after falling down with unconsciousness state at home. While she was transferred to our emergent medical center, her level of consciousness had recovered. She had no previous history of diabetes, hypertension or hyperlipidemia and denied any history of smoking or alcoholism. On the initial neurological assessment, she seemed slightly drowsy but had intact cognitive, motor and sensory function. Bilateral ptosis and dilated, non-reactive pupil measuring 5 mm with limitation of medial and vertical movements of both eyeballs were presented (Fig. 1, 2).

Brain computed tomography (CT) scan showed diffuse subarachnoid hemorrhage (SAH) on basal cistern and bilateral sylvian fissure, subdural hemorrhage (SDH), multifocal hemorrhagic contusions and intraventricular hemorrhage (IVH) in both occipital horns. Particularly, subarachnoid hemorrhage also noted in the interpeducular, left ambient and quadrigeminal cisterns. There was relatively thick hematoma around the left quadrigeminal plate (Fig. 3).

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2 mm-thickness-sectioned basal skull CT for better evaluation of bony structure indicated no bony abnormality. Further imaging with magnetic resonance imaging (MRI) was performed few days later and demonstrated the same SAH, SDH, IVH and multifocal contusions as the CT scan. The hematoma in the in the cisterns around left quadrigeminal plate was noted and the midline and left tectum showed partial hyperintensity in FLAIR image. The interpeduncular cisternal hemorrhage was also observed in MRI (Fig. 4). However, we were not able to figure out any evidence of direct oculomotor nerve damage. No vascular lesion was demonstrated on CT angiography.

The patient was treated conservatively and underwent ventriculoperitoneal shunt for hydrocephalus at 1 month. While she had her pupil back to normal in early stage, her extraocular muscle movements were improving slowly during the hospital stay. After 2 months of trauma, her right oculomotor function got fully recovered whereas her left eye still had partial oculomotor dysfunction.

III. Discussion

In our case, bilateral oculomotor nerve palsy was identified without other cranial nerve dysfunction. Performed images showed subarachnoid hemorrhage in basal and sylvian cisterns and perimesencephalic hematomas in ambient and quadrigeminal cisterns. We could presume that these hematomas could compromise oculomotor nerves bilaterally. Ashfaq et all. reported a case which was associated with the ocu-

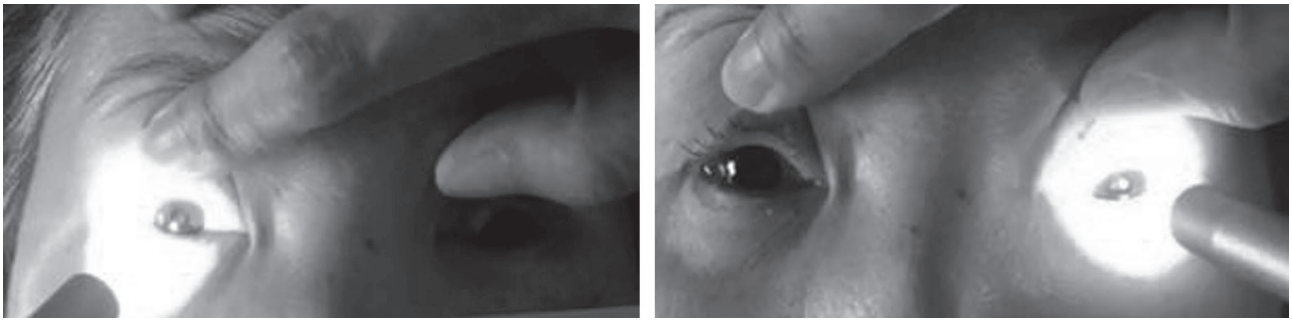

Fig. 1. Neurologic assessment of light reflex; Initially, non-reactive pupils measuring 5 mm size were identified.

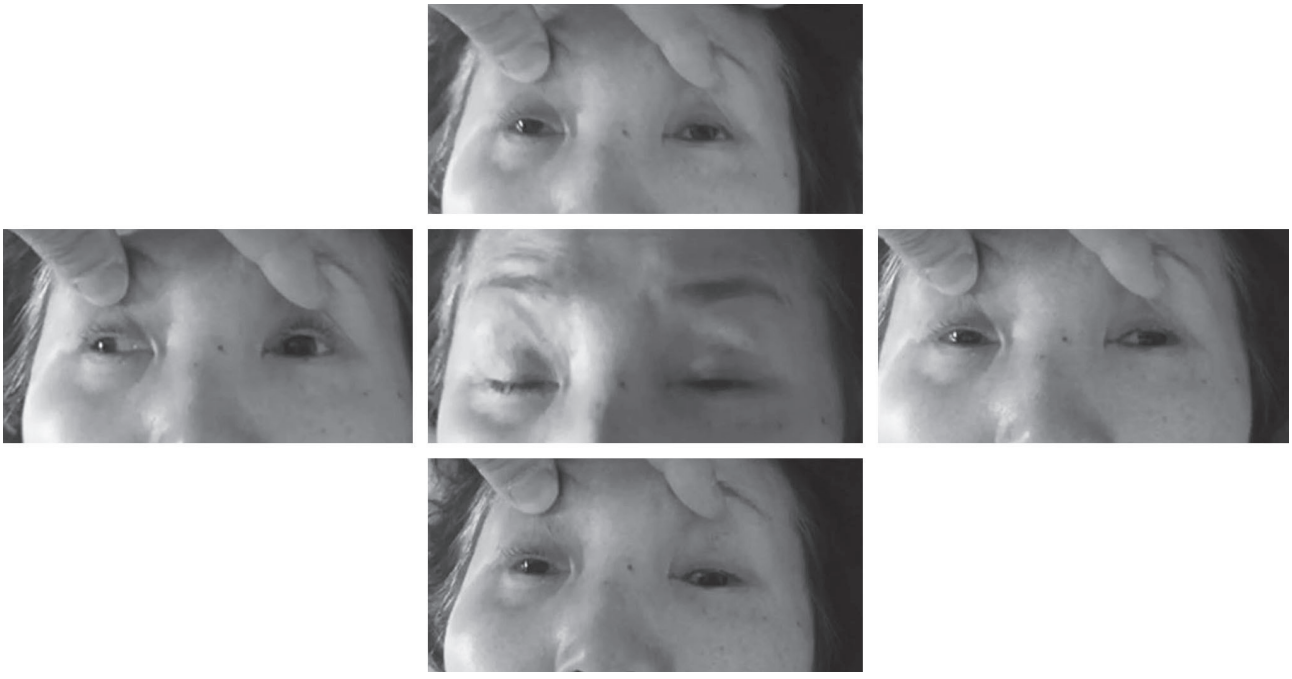

Fig. 2. Neurologic assessment of extraocular muscles; Limitations of medial and vertical movements of both eyeballs were presented.

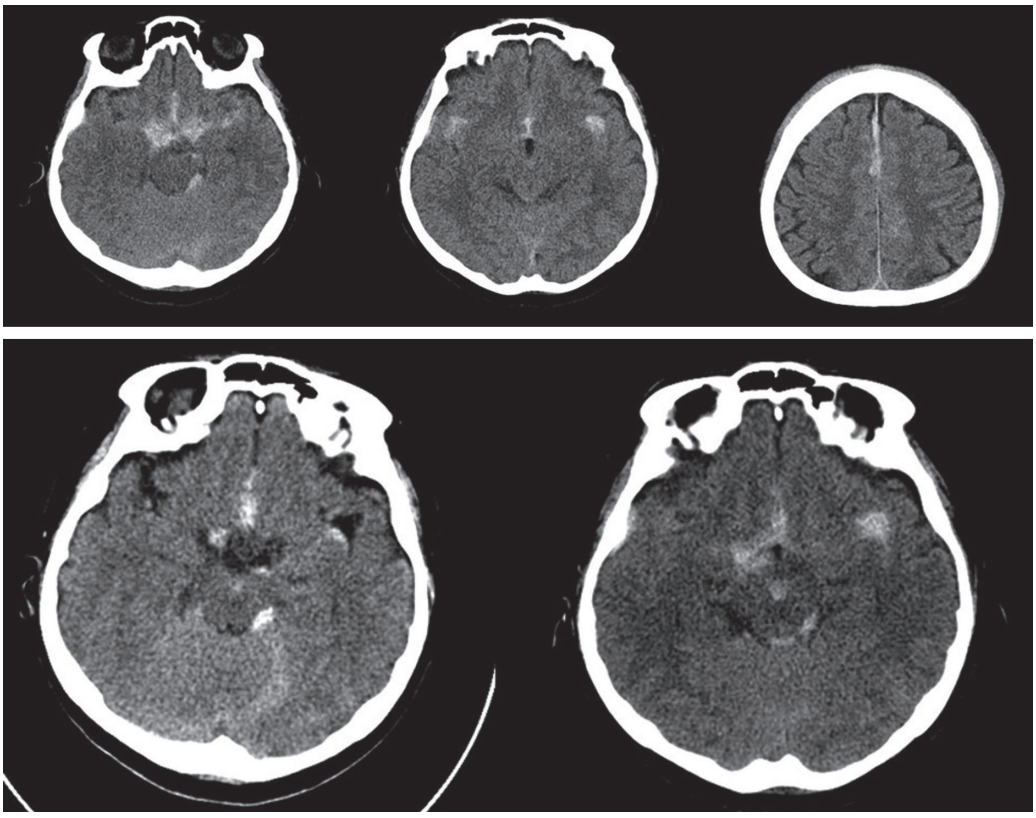

Fig. 3. Brain computed tomography (CT); Diffuse subarachnoid hemorrhage (SAH) on basal cistern and bilateral sylvian fissure, subdural hemorrhage (SDH) around falx were identified. Subarachnoid hemorrhage also noted in the interpeducular, left ambient and quadrigeminal cisterns.

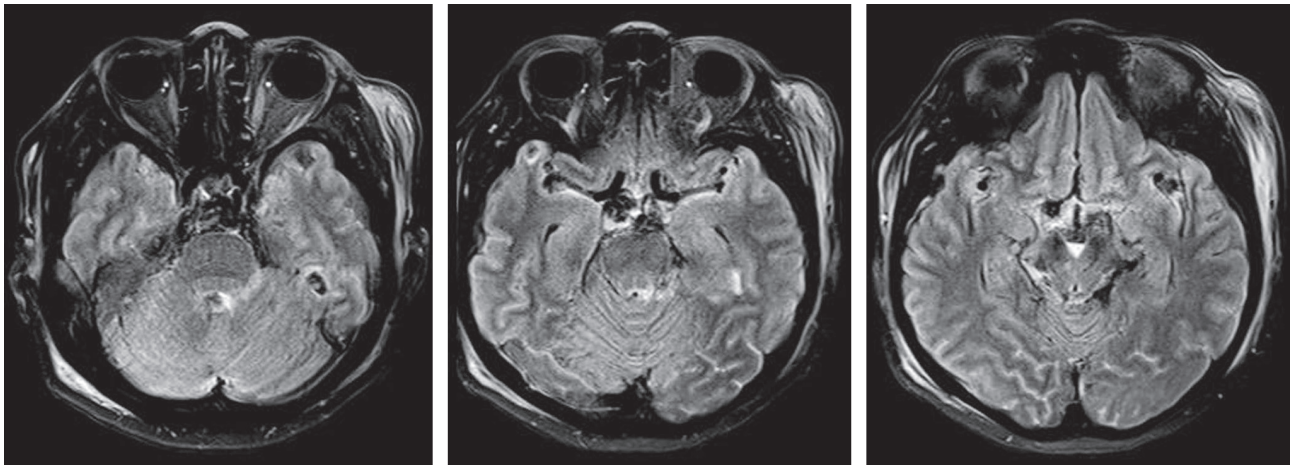

Fig. 4. Brain magnetic resonance image (MRI); The interpeduncular cisternal hemorrhage was observed in MRI.

lomotor nerve palsy compromised by mesencephalic hematomas involving the oculomotor fascicle.(4) But comparing with our case, unilateral oculomotor palsy was noted and had good correlation with focal dense hematoma in their case. According to his case report, significant clinical improvement was noted within 3 months.

In the process of localization of the oculomotor palsy, thin-thickness-sectioned CT scan of basal skull and specialized MRI protocol, which allowed identifying the ligament injury of adjacent structure to the oculomotor nerve, should be included. Christopher et al. demonstrated that complex fracture of the sella turcica and impingement of the bony nerve canals could

bring out the oculomotor palsy.(5) Takanobu et al. also demonstrated that unilateral traumatic oculomotor palsy in 11-year-old boy after traffic accident was given rise to the posterior petroclinoid ligament damage which made the bending of ipsilateral oculomotor nerve.(6) In these cases, unilateral ptosis had improved and an ocular motility examination became normal within few months.

Few cases about bilateral oculomotor palsy have already been reported. Fujino et al. reported bilateral complete oculomotor palsy after traffic accident and suspected the association with bilateral damage of the oculomotor nerves near the petroclinoid ligament.(7) Rush reviewed two cases which lack any clinical and radiologic details.(8) Kruger et al. tried to demonstrate the lesion of oculomotor palsy with CT and MRI respectively in his two cases and failed. The peripheral lesion could therefore be suspected in his review.(9) Nakashima et al. reported complete bilateral oculomotor palsy after motor vehicle accident. Initial Glasgow coma scale was poor in this case, and ptosis and oculomotor motility was partially recovered after few months.(10) Patel et al. represented the distribution of post-traumatic cranial nerve injury after head trauma. In this article, total 23 patient were suffered from cranial nerve palsy among 794 head injured patient. 11 patients were associated with basal skull fracture. 12 patients were associated with third nerve palsy and 2 of them suffered from bilateral palsies. They supposed direct injury mechanism and rostrodoral midbrain interruption lead to bilateral oculomotor palsies.(3)

In our case, we couldn't figure out the definite reason why the oculomotor palsy occurred. During localization of the lesion site, demonstrated images of oculomotor nerves and adjacent structures, including bone, tentrium and ligaments, were taken and revealed nothing specific except minimal perimesencephalic hematomas. We assumed that the nuclear palsy caused by midbrain injury is most probable and peripheral lesion near the petroclinoidal ligament should be ruled out.

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