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Case Report

# Delayed Bilateral Abducens Nerve Palsy after Head Trauma

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Although the incidence of unilateral abducens nerve palsy has been reported to be as high as 1% to 2.7% of head trauma cases, bilateral abducens nerve palsy following trauma is extremely rare. In this report, we present the case of a patient who developed a bilateral abducens nerve palsy and hypoglossal nerve palsy 3 days after suffering head trauma. He had a Glasgow Coma Score (GCS) of 15 points. Computed tomography (CT) images demonstrated clivus epidural hematoma and subarachnoid hemorrhage on the basal cistern. Herein, we discuss the possible mechanisms of these nerve palsies and its management.

**KEY WORDS :** Abducens nerve palsy · Hypoglossal nerve palsy · Epidural hematoma · Clivus.

## INTRODUCTION

Berlit et al.[5] evaluated 165 patients suffering from abducens nerve palsy as the main presenting symptom. A vascular origin (29.7%), inflammatory diseases (19.4%) and tumors (10.9%) were the most common causes, while traumatic abducens paresis (3.1%) was rare.

Although the incidence of unilateral abducens palsy from head trauma has been reported to be as high as 1% to 2.7%, bilateral palsy is rare. It comprises approximately 10% of abducens nerve palsy patients and is typically associated with additional intracranial, skull, and cervical spine injuries[5,7,14,17,22,23]. Closed head injury (CHI) with palsy of an ocular motor nerve was more severe than CHI without ocular motor nerve palsy, as measured by the GCS[8]. We present a case in which bilateral abducens nerve and unilateral hypoglossal nerve palsy developed with a high Glasgow Coma Score (GCS) 3 days after head trauma.

## CASE REPORT

A 6-year-old boy was admitted to our hospital with multiple injuries suffered after being struck by a car while walking. At the time of the accident, he had lost consciousness for about 5 minutes. His initial neurological examination revealed non-specific findings. No gaze palsy was noted on initial assessment and diplopia was not present. He had a GCS of 15 points and showed several abrasions with scratches over the right forehead. Other systemic examinations were normal. Computerized tomography (CT) images showed a hyperdense retroclival epidural hematoma with subarachnoid hemorrhage and intraventricular hemorrhage, from the mid-clival region and extending to the basal cistern (Fig. 1). Neither a fracture line nor a dislocation in the skull base and occipito-cervical region could be identified in the CT scans. Follow-up CT scans taken 4 hours later showed no newly developed

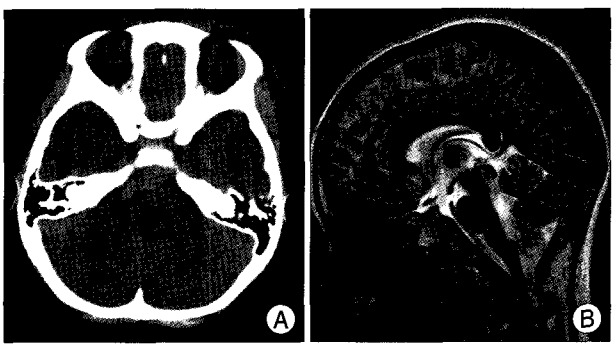


**Fig. 1.** Brain computed tomography scan (A) showing high density on retroclival area and brain T2-weighted magnetic resonance sagittal image (B) of 2 months after trauma showing nearly total resolving of previous hematoma.

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**Fig. 2.** Photographs showing bilateral gaze palsy. A : Primary position. B : Right gaze. C : Left gaze.

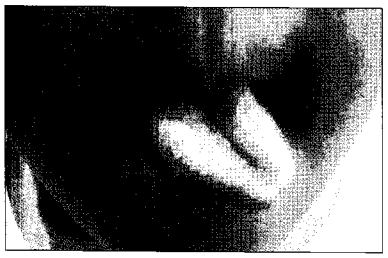
**Fig. 3.** Photograph showing tongue deviation to left.

hemorrhage. On the third day after the accident, a diplopia was noted on the lateral gaze. Neurologic examination revealed abduction deficits of both eyes and unilateral hypoglossal nerve palsy (Fig. 2, 3). Follow-up CT imaging showed no newly developed abnormal lesions. Methyl-prednisolone was started immediately for the cranial nerve palsies and he was discharged from the hospital on the twentieth day of admission. Magnetic resonance imaging (MRI) of the brain was performed 2 months after his admission and it revealed a normal clivus-brainstem relationship without any sign of residual hematoma (Fig. 1). By the sixth month after the initial injury, his bilateral abducens, hypoglossal nerve palsies were completely resolved (Fig. 4).

## DISCUSSION

The differential diagnosis for traumatic lateral gaze palsy includes brain stem lesion such as diffuse axonal injury, peripheral nerve injury with or without basilar skull or cervical fracture, and lateral rectus muscle injury or entrapment[4]. In the literature, bilateral sixth nerve palsy also can occur by increased intracranial pressure, dural puncture, whiplash injury, hangman's fracture and halo traction[2-4,10,16,19,24]. The mechanism for this bilateral palsy is controversial. Physical examination and appropriate imaging tests such as high resolution CT or MRI can identify the cause of a traumatic gaze palsy[1,4].

The abducens nerve is the most susceptible cranial nerve to trauma, because of its long intra-cranial course. Anatomically, abducens nerve consists of intracisternal, intra-cavernous and intra-orbital parts. After exiting the pontomedullary sulcus, it courses through the prepontine cistern, then joins the cavernous sinus in the petroclival region and innervates the lateral rectus muscle in the orbita. The abducens nerve has three angulations in the petroclival region : at the dural entry point, petrous apex and at the point where it joins the lateral wall of the internal carotid artery[6,18]. These angulation points are vulnerable.

The mechanism of traumatic abducens nerve palsy might fall into one of two categories : direct mechanical injury and indirect injury. The former results from nerve compression by a hematoma or a surrounding structure such as ligament or dura matter. The latter results from nerve ischemic change due to vessel compression or vasospasm.

Most head injuries are due to two basic mechanisms, contact or acceleration. However, static loading force is another, rarer type of head injury, also known as crushing head injuries. This force causes avulsion of the petrous bone from the foramen lacerum to the outer side of the bone, which stretches and directly injures the abducens nerve[12]. Takagi et al.[21] pointed out that it is impossible for the abducens nerve to be damaged at the petroclinoid ligament (Gruber's lig.) by the upward movement of the brain stem, because the abducens nerve is fixed downward below this ligament by the dura and apex of the petrous pyramid. In human cadaveric studies, most of the abducens nerve injuries have been shown to take place in the petroclival region[20]. Histopathological examinations have revealed edema and perineural hemorrhages predominantly at the locations of dural entry point and petrous apex. Associated petrous bone fractures and cervical trauma have been revealed in radiological examinations of patients with abducens nerve palsies[3,12,13]. Dukes and Bannerjee[9] described a case of a unilateral hypoglossal nerve palsy after a hyperextension "whiplash" neck injury as being caused by traction and/or hematoma. Currently, the most accepted


**Fig. 4.** Photograph showing complete recovery by the sixth month after the initial injury.

theory proposes that trauma to the head can cause downward displacement of the abducens nerve in Dorello's canal, with contusion against the petrous ridge[21].

Delayed lower cranial nerve palsy can occur following occipital condyle fracture[7,17,22,23]. In our case, cranial and cervical CTs and MRIs confirmed that there was no fracture or dislocation. Delayed traumatic abducens nerve palsies have been reported, with its possible etiology being injury to the branches of the meningodorsal artery[6]. In our case, acute retroclival epidural hematoma and traumatic subarachnoid hematoma were thought as the causative factors for the delayed bilateral abducens nerve palsies, as well as the left hypoglossal nerve palsies, because it could directly compress and indirectly injure the cranial nerves by setting up vasospasm on their vessels. Other possible causes include a brain stem lesion, increased intracranial pressure and orbital fracture with lateral rectus entrapment. A brain stem lesion was unlikely because there was no seventh nerve palsy and no pyramidal tract findings. In any event, CT and MRI confirmed no brain stem lesion. In addition, there were no clinical signs or imaging results consistent with increased intracranial pressure, medial rectus entrapment or orbital trauma, and no orbital fracture was seen on the CT images.

Although the overall spontaneous recovery rate of traumatic abducens nerve palsy is high[11], a complete or bilateral case has a poor recovery rate. Unilateral cases resolve spontaneously in 72% of cases against only 12% of bilateral cases (6-month follow-up period)[11,15]. In our case, complete recovery was seen after 6 months.

## CONCLUSION

The presence of a bilateral abducens nerve palsy is tho-ught to indicate severe trauma to the head and neck, and patients with bilateral palsy show a tendency to have a low GCS. However, the patient in the present case exhibited a high GCS with no definite head and neck injury except a clivus epidural hematoma. Traumatic clivus epidural hematoma is not exclusive to pediatric cases and should be considered whenever acute or delayed bilateral cranial nerve palsy is present in the context of head trauma. Although the complete recovery rate is relatively low in bilateral palsy, this case resulted in a complete recovery with conservative treatment.

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