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A case of traumatic bilateral abducens and unilateral hypoglossal nerve palsy

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Patient: Female, 47
Final Diagnosis: Traumatic bilateral abducens • unilateral hypoglossal nerve palsy
Symptoms: Diplopia
Medication: —
Clinical Procedure: —
Specialty: Neurology

Objective: Rare disease
Background: Incidence of unilateral abducens palsy from head trauma has been reported to be as high as 1% to 2.7%, but bilateral abducens nerve palsy is extremely rare.
Case Report: We present a case in which bilateral abducens nerve and unilateral hypoglossal nerve palsy developed with a high Glasgow Coma Score (GCS) 3 hours after head trauma due to a motor vehicle crash.
Conclusions: This case highlights the occurrence and management of posttraumatic bilateral sixth nerve palsy.

Key words: abducens nerve injury • hypoglossal nerve injury • trauma

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Background

The incidence of unilateral abducens palsy from head trauma has been reported to be as high as 1% to 2.7%, but bilateral abducens nerve palsy is extremely rare [1]. It accounts for approximately 10% of abducens nerve palsy patients and is typically associated with additional intracranial, skull, and cervical spine injuries [2].

We present a case in which bilateral abducens nerve and unilateral hypoglossal nerve palsy developed with a high Glasgow Coma Score (GCS) 3 hours after head trauma due to a motor vehicle crash.

Case Report

A 47-year-old woman was admitted to the emergency service because of a motor vehicle crash in which she experienced head trauma. She was sitting in the front seat of the car and was not using a seatbelt. At the time of the accident, she lost consciousness for about 5 minutes. Her first neurological examination revealed non-specific findings. No gaze palsy was noted on initial assessment and diplopia was not present. She had a GCS of 15 points and showed several abrasions with scratches over the right forehead and tongue. Other systemic examinations were normal. Computerized tomography (CT) images of the skull and parenchyma were normal (Figure 1).

Approximately 3 hours after the concussion, she had headache and diplopia. Neurologic examination revealed lateral gaze palsy of both eyes (Figures 2 and 3). The hypoglossal nerve was not initially evaluated because of stitches in her tongue. A follow-up neurologic examination revealed tongue deviation to the left side and CT imaging showed no newly developed abnormal lesions (Figure 4). In her cranial magnetic resonance imaging (MRI) she had contusion areas in the parietal and occipital lobes (Figure 5).The MRI angiography result was normal (Figure 6). Digital substraction angiography was performed after 1 month to exclude fistula formation and the result showed no abnormality. She was treated with methylprednisolone and was discharged with antibiotics and oral steroids prescribed for a period of 3 weeks. At 1-year follow-up, her sixth nerve palsy and hypoglossal nerve palsy was fully recovered (Figures 7 and 8).

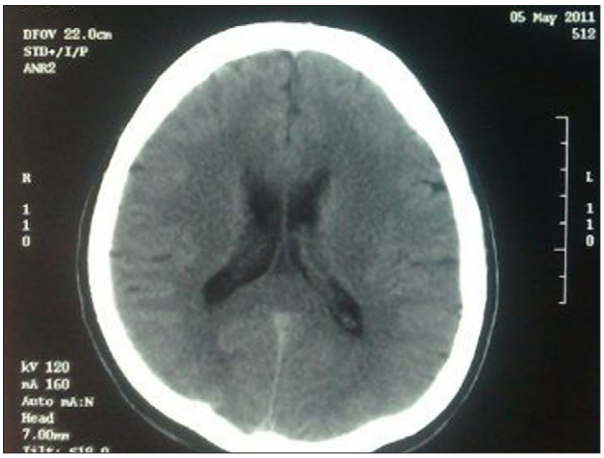

Figure 1. Brain Computed Tomography showing no abnormality.

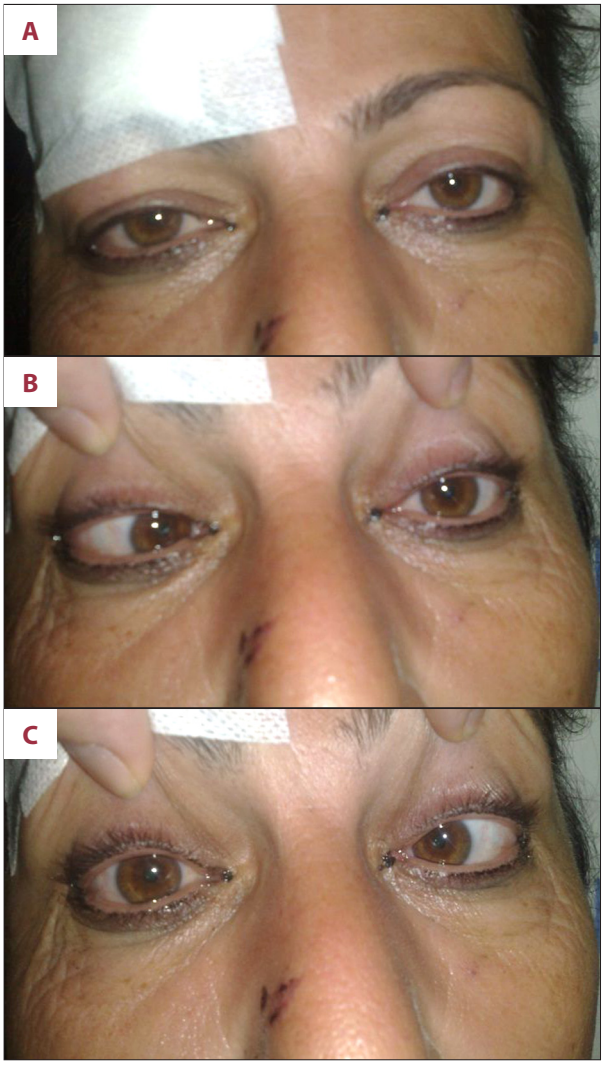

Figure 2. After the accident photographs showing bilateral gaze palsy (**A**) primary position; (**B**) left gaze; (**C**) right gaze.

Discussion

Because of its long intracranial course, the abducens nerve is the cranial nerve most susceptible to trauma. Lesions causing abducens nerve palsy may be located in the brain stem, subarachnoid space, petroclival region, cavernous sinus, or in the orbit along the course of the nerve. The abducens nucleus is

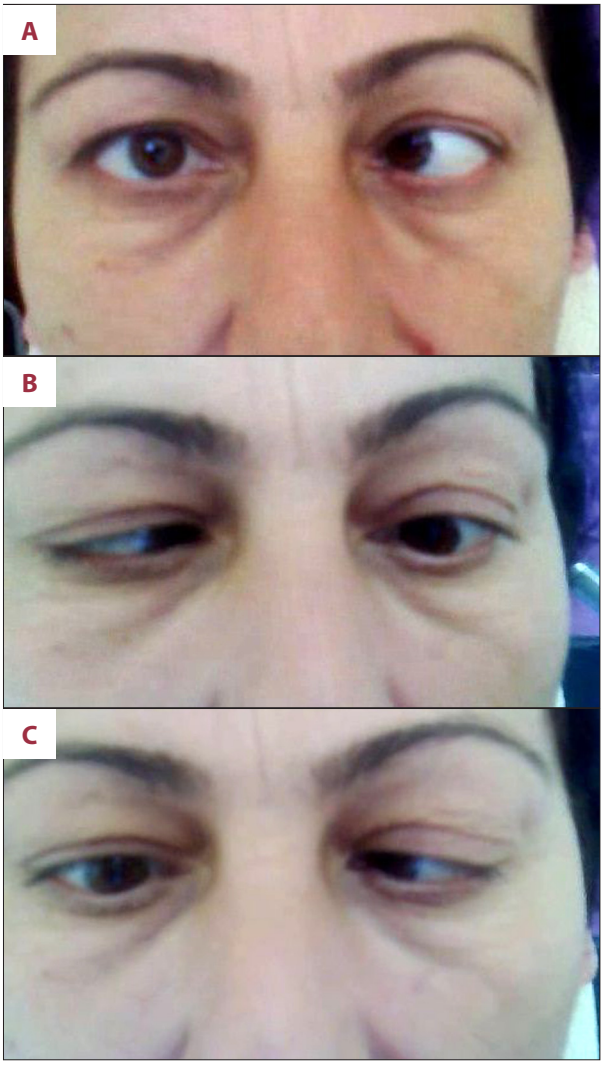

Figure 3. After 2 months of accident no recovery at bilateral sixth nerve palsy (**A**) primary position; (**B**) left gaze; (**C**) right gaze.

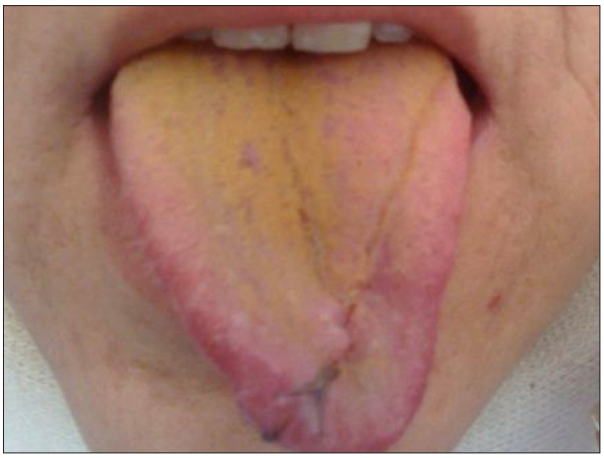

Figure 4. Tongue deviation to left showing unilateral hypoglossal nerve palsy.

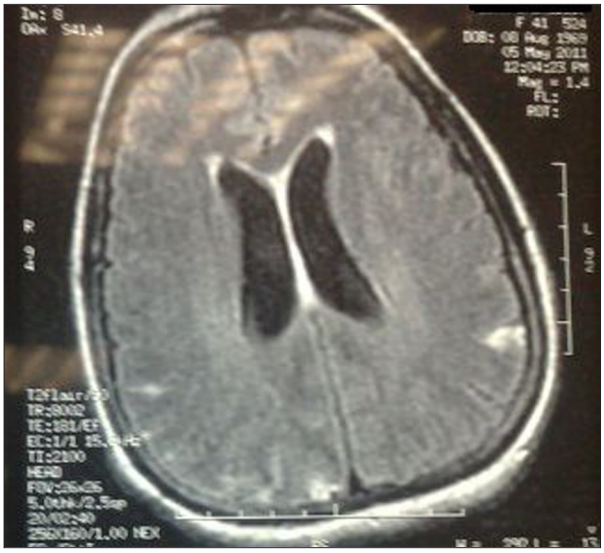

Figure 5. MRI showing contusion areas at parietal and occipital lobes.

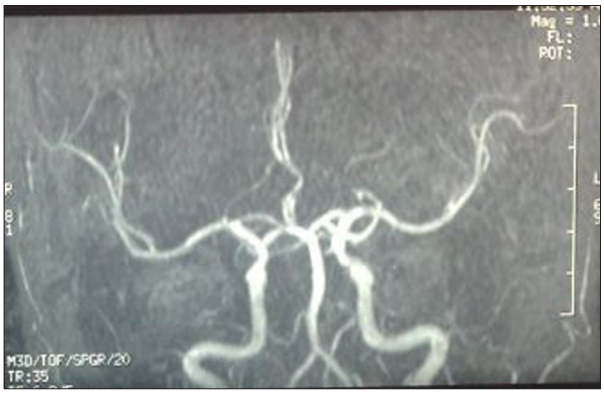

Figure 6. Normal MR angiography.

located in the pontine tegmentum, just ventral to the fourth ventricle, and axons of the seventh cranial nerve loop around the abducens nucleus. Fascicles emerging from the nucleus course forward through the pontine tegmentum to emerge from the ventral surface of the brain stem. The subarachnoid portion of the sixth nerve lies in the prepontine cistern, and also inside Dorello's canal. Dorello's canal was described in earlier studies as a short, osteofibrous channel between the petrous apex and Gruber's ligament. The abducens nerve flattens on the lateral side of the internal carotid artery (ICA) where the anastomosis point of the nerve meets with the periarterial sympathetic plexus of the ICA, and then course on the inferolateral side of the ICA in the cavernous sinus to enter the orbita through the annulus of Zinn [3–5].

The abducens nerve has 3 angulations in the petroclival region: at the dural entry point, the petrous apex, and at the point where it joins the lateral wall of the internal carotis artery. These angulation points are vulnerable [6].

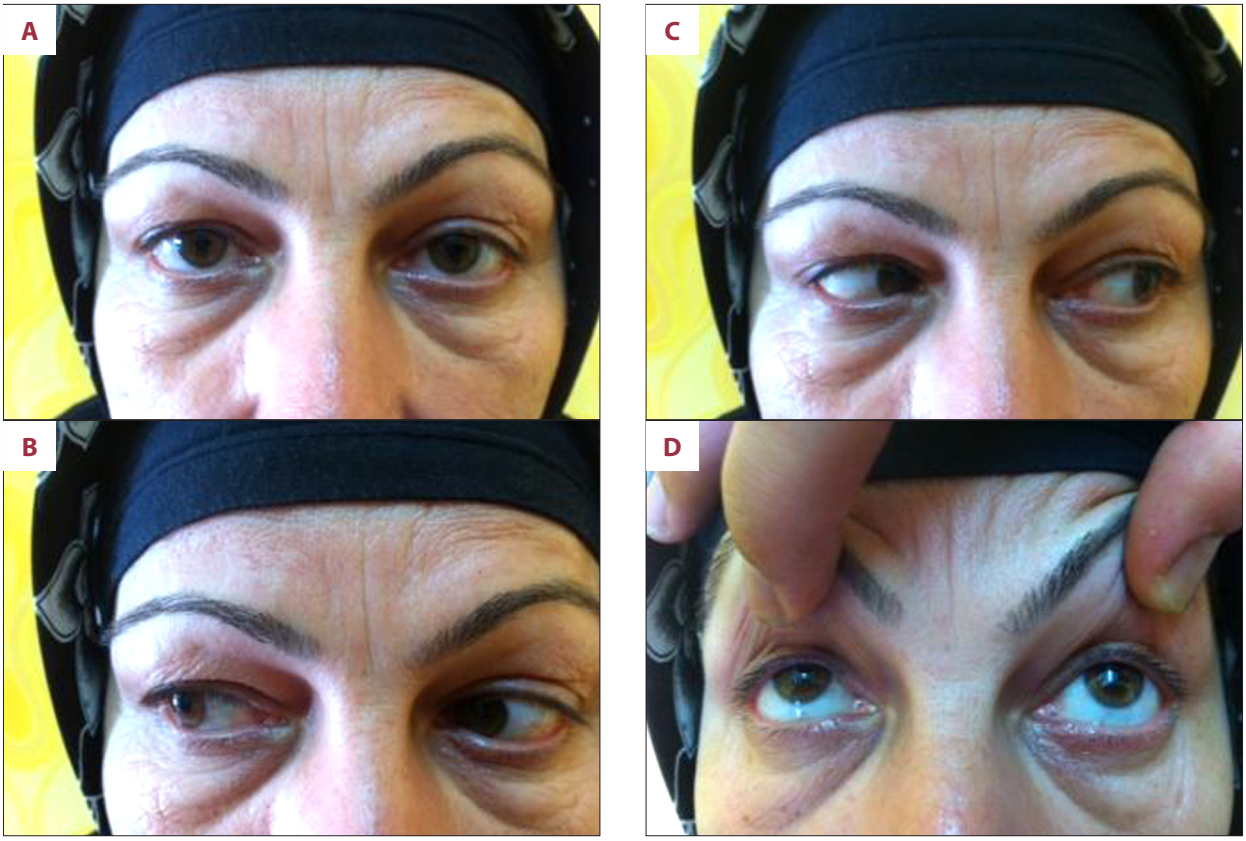

Figure 7. Full recovery of sixth nerve palsy.

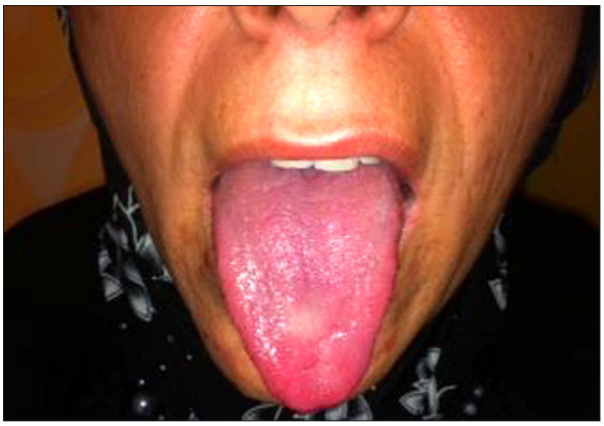

Figure 8. Full recovery of hypoglossal nerve palsy.

Berlit et al. evaluated 165 patients with 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, and traumatic abducens paresis (3.1%) was rare [7]. Associated petrous bone fractures and cervical trauma have been revealed in radiological examinations of patients with abducens nerve palsies [8,9]. Currently the most accepted 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. Posttraumatic bilateral abducens palsy has been reported mainly in various case reports, thus emphasizing its relative rarity. The mechanism of this bilateral palsy is controversial [10]. In the literature, bilateral sixth nerve palsy also can occur by increased intracranial pressure, dural puncture, whiplash injury, hangman's fracture, and halo traction [11–13].

In our case, hyperextension 'whiplash' neck injury was thought to be the causative factor for the bilateral sixth nerve and hypoglossal nerve palsy, similar to the case reports by Dukes and Bannerjee and by Kim et al. [14,15].

Mutyala et al. reported a spontaneous improvement rate of 72% in unilateral traumatic sixth-nerve palsy and 12% in bilateral traumatic sixth nerve palsy (6 month follow-up period) [16]. Holmes et al. reported an overall spontaneous recovery rate of 73%. Spontaneous recovery was more frequent in unilateral cases (84%) than in bilateral cases (38%) [17]. Treatment of post-traumatic bilateral abducens nerve palsy is usually conservative, although Kao et al. showed that such patients treated with subtenon injection of botulinum toxin showed higher recovery rates than patients treated conservatively [18]. Holmes et al. concluded that non-recovery from acute traumatic sixth nerve palsy was associated with complete palsy and/or a bilateral palsy.

Conclusions

Bilateral abducens and hypoglossal nerve palsy due to whiplash without fracture or dislocation, as in our case, seems to be extremely rare. Our patient was managed conservatively with oral steroids and, despite the low recovery rate, the patient showed total improvement of abducens and hypoglossal nerve palsy at 1-year follow-up. This case highlights the occurrence and management of posttraumatic bilateral sixth nerve palsy and hypoglossal nerve palsy.

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