

Bilateral Lower Cranial Nerve Palsy after Closed Head Injury: A Case Report and Review of Literature

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Abstract

Paralysis of the lower cranial nerves is uncommon after closed head injuries. Most cases reported are unilateral and associated with base of skull fractures, usually involving the occipital condyles. Bilateral lower cranial nerve palsy is even less common, with only a handful of cases reported in literature. A 17-year-old girl presented to us after she was involved in a side-on collision with a car while driving a scooter. She sustained traumatic brain injury requiring mechanical ventilation. Detailed neurological evaluation revealed bilateral paralysis of the IXth, Xth, and XIIth cranial nerves with no evidence of a fracture of the base of skull or brain stem injury. A traction type of injury to the nerves arising from a whiplash mechanism may have led to paralysis of the lower cranial nerves in our patient. An exhaustive review of literature revealed 11 reports of bilateral lower cranial nerve palsy associated with closed head injuries; there were only four cases without underlying fracture of the occipital condyles. Our patient made a complete recovery over a period of 4 months. A traction type of injury to the lower cranial nerves may occur due to a whiplash mechanism. This type of injury may be associated with a favorable outcome.

Keywords: Bilateral, closed head injury, lower cranial nerves, whiplash injury

INTRODUCTION

Cranial nerve palsy associated with traumatic brain injury has been widely studied;^[1] however, there are relatively few reports involving the lower cranial nerves. Bilateral involvement of the lower cranial nerves is even less common, and most case reports in literature are related to fractures involving the base of the skull, especially the occipital condyles.^[2] We report a case of bilateral IXth, Xth, and XIIth nerve palsies following closed head injury in a young girl, without any evidence of brain stem injury or associated fractures of the base of the skull. We also summarize the findings of an extensive review of previous reports of bilateral lower cranial paralysis following closed head injury.

CASE REPORT

A 17-year-old girl presented to our emergency department after she was involved in a side-on collision with a car while driving a scooter. She lost consciousness briefly, and passers-by who were at the scene brought her to the hospital. On initial evaluation, she was maintaining airway and breathing comfortably at 20/min with an oxygen saturation of 96% on

air. Her pulse rate was 90/min, and she had a blood pressure of 112/70 mmHg. She was found to be restless and irritable with a score of 13 (E3, V4, M6) on the Glasgow Coma Scale. Her pupils were 3 mm bilaterally and reacting to light; she was not cooperative for a more detailed neurological evaluation. She had a lacerated wound on her lower lip and a few abrasions on the shoulder and the neck.

Following the initial evaluation, she underwent a whole-body computed tomography (CT) scan. She had minor contusions on the left temporal lobe with minimal cerebral edema, and her cervical spine showed a chip fracture of the tip of the odontoid process. A retropharyngeal hematoma was noted [Figure 1], extending from the skull base to the C4 vertebral level, and her CT chest showed contusions on the right lung. Over the next few hours, she became more tachycardic with heart rates up to 120/min. As she became increasingly restless, she was

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intubated and mechanically ventilated. She was commenced on mannitol in view of the cerebral edema and was administered levetiracetam as antiseizure prophylaxis.

Over the next 72 h, after ceasing sedative medication, her level of sensorium improved, and she was able to obey verbal commands. However, she remained tachycardic during this period. She was extubated on day 4 but had to be reintubated within a few hours as she was pooling secretions and unable to maintain her airway. An magnetic resonance imaging (MRI) scan on day 6 showed contusions and edema, similar to the findings on CT. The retropharyngeal hematoma persisted. As she continued to have a weak cough, a percutaneous tracheostomy was performed on day 9. Over the next 3–4 days, she had a recurrent collapse of her left lung which required bronchoscopy and she developed a pneumonic infiltrate requiring antibiotic therapy. However, she could be gradually weaned off mechanical ventilation and was able to tolerate spontaneous breathing on a “T” piece. She remained awake, appropriate, and cooperative during this period.

On detailed neurological evaluation, she was found to be unable to move her tongue from side to side, although limited protrusion was possible. She had no sensation on the posterior wall of the pharynx, and palatal movements were absent bilaterally. Video-laryngoscopic examination revealed immobile vocal cords on both sides, fixed in an intermediate position, with pooling of secretions in the vallecula and pyriform fossa [Figure 2]. These clinical findings were consistent with bilateral paralysis of the IXth, Xth, and XIIth cranial nerves. Considering the requirement for long-term tube feeding, a percutaneous endoscopic gastrostomy (PEG) was performed. She was discharged for continued care at home with the PEG and tracheostomy tubes *in situ*.

On continued neurological follow-up over the next few weeks, she showed gradual improvement of neurological function. Her cough reflex became stronger and she regained mobility of the tongue. The vocal cords also revealed a gradual, although

slower, improvement. By 14 weeks after the trauma, she had made a complete recovery of all the cranial nerves involved. Her tracheostomy was decannulated, and the PEG tube was removed at this stage.

DISCUSSION

Unilateral paralysis of the lower cranial nerves was first described by Collet following a gunshot injury to the mastoid.^[3] There are several reports of lower cranial nerve palsies following closed head injuries; most are unilateral and associated with skull base fractures, usually involving the occipital condyles.^[4] Several hypotheses have been proposed to explain the mechanism of causation of such injuries, including nerve compression by bony fragments and edema of the nerve secondary to ischemia.^[5] In the absence of direct trauma, a “stretch” type of nerve injury has been postulated due to hyperextension of the neck.^[6] There are few reports of bilateral lower cranial nerve palsy following closed head injury; most are associated with skull base fractures.^[2,7] The hypoglossal nerve may be especially prone to a closed type, hyperextension injury due to its anatomical proximity to the transverse process of the atlas, making it vulnerable to traction injury.^[8]

We performed a comprehensive search of PubMed, Embase, and Google Scholar using the search terms “bilateral,” “cranial nerve,” and “trauma.” The reference lists of the retrieved articles were reviewed to identify additional reports. We came across 11 cases of bilateral lower cranial nerve palsy associated with closed head injuries. Data are presented on the mechanism of injury, cranial nerves involved, associated fractures of the skull or the cervical spine, other injuries, and outcome from the neurological deficits [Table 1].^[2,4,6-14] Fracture of the occipital condyles was observed in all except four cases;^[6,8,9,11] a fracture of the base of the odontoid process was seen in one case.^[9]

Our patient had dysphagia, pooling of secretions, tongue immobility, and persistent tachycardia, due to bilateral IXth, Xth, and XIIth nerve paralysis following a closed head



Figure 1: Retropharyngeal hematoma on the computed tomography scan (arrow)

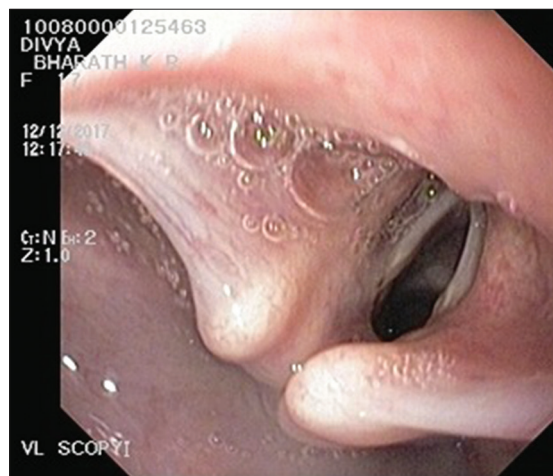


Figure 2: Video-laryngoscopy showing vocal cords in an intermediate position bilaterally with pooling of secretions

Table 1: Reports of bilateral lower cranial nerve palsy associated with closed head injury

Authors	Mechanism	Nerves involved	Neurological deficits	Associated fracture	Other injuries	Outcome
Helliwell <i>et al.</i> ^[6]	Motor vehicle accident	Bilateral VI th and X th	Bilateral vocal cord paralysis, dysphonia, stridor	None	None	No improvement at 18 months
Spencer <i>et al.</i> ^[7]	Motor vehicle accident	Bilateral X th	Bilateral vocal cord paralysis	Fracture occipital condyle. Burst fracture lateral mass of C1	Multiple contusions, subdural hematoma, face, chest, abdomen, and orthopedic injuries	No improvement at 11 weeks
Leventhal <i>et al.</i> ^[2]	Motor vehicle accident	Bilateral VI th , VII th , and X th	Dysphagia	Fracture left occipital condyle, temporal bones bilaterally	Extradural hematoma, blunt abdominal injury, long bone fractures, facial laceration	Complete recovery at 3 months
Brennan <i>et al.</i> ^[8]	Motor vehicle accident	Bilateral XII th	Dysarthria	None	Superficial injuries to the head and face	Complete recovery in 3 days
McCleary ^[9]	Trip and fall, face first	Bilateral X th and XII th	Stridor, palatal paralysis, dysphonia, flaccid tongue	Fracture of the odontoid process	Retropharyngeal hematoma	Near-complete recovery at 4 weeks
Paley and Wood ^[10]	Motor vehicle accident	Bilateral XII th	Dysphagia, dysarthria, immobile tongue	Fracture left occipital condyle	Hemothorax, diaphragmatic rupture	Partial recovery at 6 months
Freixinet <i>et al.</i> ^[11]	Motor vehicle accident	Bilateral XII th	Dysphagia, aspiration, atrophy of the tongue	None	Flail chest, neck laceration	Complete recovery at 6 months
Lam and Stratford ^[12]	Motor vehicle accident	Right VII th , bilateral XII th	Paucity of tongue movements, atrophy of the tongue. Right facial weakness, right hemiplegia	Fracture right occipital condyle	Clavicle and rib fractures, lung contusion	Partial recovery of tongue atrophy at 2 years
Legros <i>et al.</i> ^[4]	Motor vehicle accident	Bilateral VI th , left VII th , bilateral X th	Aspiration, dysphonia, dyspnea on exertion	Fracture occipital condyle left	Right lateral extradural hematoma	No improvement at 18 months
Shekhar <i>et al.</i> ^[13]	Crash during stock car racing	Bilateral XII th and VI th	Dysarthria, diplopia	Fracture left occipital condyle	Subdural hematoma	Complete recovery at 1 year
Yoo <i>et al.</i> ^[14]	Fall in an inebriated state	Bilateral IX th and X th	Dysphagia, bilateral vocal cord paralysis	Bilateral occipital condyle fracture	Subarachnoid hemorrhage	Mild improvement of vocal cord function, persisting dysphagia at 1 year

injury. There were several features strongly suggestive of a flexion–extension or “whiplash” type of injury in the present case. She had a type I fracture of the odontoid process, typically seen in this type of injury.^[15] A type I fracture of the odontoid is considered relatively stable^[16] and unlikely to have caused the neurological deficits in our patient. Our patient sustained a retropharyngeal hematoma, extending from the base of the skull to the C4 level. Retropharyngeal hematomas occur with hyperextension and whiplash type of injuries.^[17,18] There are previous reports of retropharyngeal abscesses causing lower cranial nerve palsy;^[19] however, in our literature search, we did not find a similar nerve injury due to direct compression by a retropharyngeal hematoma. A primary brain stem injury, leading to lower cranial nerve palsy, may occur due to shearing close to the edge of the tentorium or due to hyperextension of the cervical vertebrae.^[20] However, there was no evidence of brain stem injury on the MRI scan in our patient.

It is more likely that bilateral paralysis of IXth, Xth, and XIIth nerves in our case was caused by a traction type of injury arising from a whiplash mechanism. Complete recovery from neurological deficits also lends support to

this mechanism.^[11] We came across four previous reports of bilateral lower cranial nerve palsy without fractures involving the base of the skull,^[6,8,9,11] however, none of them involved combined injury to the IXth, Xth, and XIIth nerves as in our patient. Our patient gradually improved, with complete recovery of all the nerves involved over a period of 4 months.

CONCLUSION

Bilateral lower cranial nerve paralysis may occur due to a whiplash mechanism, without underlying fractures of the skull base or a primary brain stem injury; this type of injury may be associated with a favorable outcome.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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