CASE REPORT

Delayed unilateral vocal cord paralysis caused by minor head trauma

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Abstract Vocal cord paralysis caused by lower cranial nerve paralysis originating from skull base fracture is rare. Delayed unilateral glossopharyngeal and vagus nerve paralysis after minor head injury is extremely rare. In this paper, we present the case of a 49-year-old man who sustained delayed-onset right vocal cord paralysis and dysphagia in a fighting accident. High-resolution computed tomography of the skull base revealed a bony disruption in the wall of the jugular foramen. Cranial nerve paralysis may be a distinguishable sign of skull base fracture in head injuries. Considering the severe consequences of the injury, comprehensive neurological and radiological examinations are required to evaluate the condition of the skull base. Such patients can be treated conservatively with nasogastric or gastrostomy feeding to avoid choking and aspiration. Thyroplasty may be considered for patients in whom choking persists for more than 6 months.

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1. Introduction

Vocal cord paralysis can result from several conditions, including thyroid or cervical surgery, tracheal intubation, cervical spine injury, medullary infarction, internal carotid artery dissection, and neurodegenerative or neuromuscular disease. However, it has rarely been reported after minor head trauma. Unilateral delayed vocal cord paralysis after minor head trauma is extremely rare. According to our review of the relevant literature, only one case with delayed isolated IX and X nerve palsies following an occipital condyle fracture has been reported.1–3 In this paper, we describe a case of delayed unilateral vocal cord paralysis after minor head trauma.

2. Case Report

A 49-year-old man was beaten with a stick on the posterior side of his head when he was drunk. He experienced...
headache, dizziness, and tinnitus on recovering from his drunkenness; however, no hoarseness or dysphagia was noted at that time. Two days later, he visited our emergency department because of coughing, hoarseness, fever, and progressive difficulties in swallowing. The patient had been healthy before this incident; he was alert and exhibited no motor abnormalities.

Ecchymosis was observed in the right mastoid area; tinnitus and right hearing impairment were noted. Otolaryngology revealed that both tympanic membranes were intact, and a Rinne test was normal. A Weber test revealed shifting of his auditory function to the right side. Brainstem auditory-evoked potentials showed a right peripheral hearing conduction defect.

Pyramidal tract signs were absent, and all sensation modalities were preserved in his trunk and extremities. All cranial nerves were normal, except for the glossopharyngeal and vagus nerves. Furthermore, Horner’s syndrome and abnormal cerebellar functions were absent. His touch sensation was reduced on the right side of the pharynx, and a gag reflex could not be induced on either side. Physical examination showed right hemiparesis of the soft palate, and laryngoscopy revealed right vocal cord paralysis. Barium esophagography showed paralysis of the hypopharynx and upper esophagus.

No fracture was seen on the initial plain X-ray films of the skull or cervical spine, and C-spine dynamic X-ray showed no subluxation. An emergency computed tomography (CT) scan of the brain was normal; however, the craniovertebral junction was not examined. A high-resolution thin-section CT of the skull base revealed a right jugular tubercle of the occipital bone fracture (Figure 1). Magnetic resonance imaging showed only a mild cerebellar edematous change close to the right jugular tubercle, and no medullary infarction, intra-axial lesion, or blockage of the inferior cerebellopontine cistern. Angiography revealed no vascular abnormalities.

On admission, the patient was treated with antibiotics for aspiration pneumonia. Owing to his dysphagia, he was fed a liquid diet through the nasogastric route. An otolaryngology reported unilateral vocal cord paralysis during examination (Figure 2). Tinnitus and right hearing impairment had improved approximately 3 months after onset. The patient could eat orally 5 months after onset; however, he still frequently experienced choking. Medialization thyroplasty was performed by an otolaryngologist 7 months after symptom onset. The patient recovered; he could eat a full diet orally and speak in an intelligible voice 4 years after surgical correction.

3. Discussion

Glossopharyngeal, vagus, and accessory nerves exit the cranial vault through the jugular foramen. Jugular foramen syndrome refers to a group of symptoms arising from lesions at the jugular foramen that controls the functions of these nerves. These symptoms include the loss of taste in the posterior two-thirds of the tongue; anesthesia of the ipsilateral pharynx and larynx; paralysis of the vocal cord, palate, and pharynx (caused by vagus and glossopharyngeal nerve injury); and paralysis of the trapezius and sternocleidomastoid muscles (caused by accessory nerve damage). Severe head trauma with a basilar skull fracture across the jugular foramen or brain stem injury can produce this syndrome; however, delayed, isolated glossopharyngeal and vagus nerve paralysis caused by minor head trauma is extremely rare.

Tumor formation close to the jugular foramen and jugular bulb occlusion are the major causes of jugular foramen syndrome; however, in our case, the clinical history and imaging studies excluded this etiology. This syndrome can also be caused by damage to the blood vessels...
supplying the cranial nerve, but imaging revealed no infarction or vascular abnormalities in our patient.

One possible explanation for lower cranial nerve deficits following a fracture at the jugular foramen was that some cranial nerves were directly compressed by displaced bone fragments.6,7 By contrast, delayed lower cranial nerve palsy can be caused by ischemic changes1 or the fracture repair process associated with dense ossification.7 In our case, the most likely mechanism that contributed to delayed nerve palsy was associated with nerve edema after direct occipital impaction, which resulted in a linear fracture through the inferior occipital bone and jugular tubercle. Temporary tinnitus and hearing impairments were associated with acceleration–deceleration injury to the petrous bone that affected the cochlea or its hair cells.

In this study, the patient presented with normal functions of the trapezius and sternocleidomastoid muscles. Why did the accessory nerve remain intact when cranial nerves IX and X were affected? The spinal root of the accessory nerve may be less vulnerable than the glossopharyngeal and vagus nerves (cranial nerve)2; however, the actual cause remains unknown.

In cases of head injury associated with a motor vehicle accident or fall from a height, a skull plain radiograph or regular brain CT may not be adequate to rule out a basilar fracture. Therefore, we emphasize that high-resolution thin-section CT scanning of the cranial base and craniocervical junction is the gold standard for diagnosis.

In conclusion, skull base fracture can occur without massive head trauma. In patients presenting with hoarseness and dysphagia caused by head trauma, high-resolution thin-section CT scan of the cranial base and craniocervical junction is recommended, even when no abnormal signs are observed in regular radiographic examination. Such patients can be treated conservatively with nasogastric or gastrostomy feeding to avoid choking and aspiration. Thyroplasty may be considered for patients in whom choking persists for more than 6 months.

References