

Occipital Condyle Fracture with Associated Hypoglossal Nerve Injury

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Occipital condyle fracture (OCF) is a rare injury that was first described by Bell in 1817. In fact, there have been only 96 more reported cases of occipital condyle fractures from 1817 to 1994 of which only 58 survived.¹ Occipital condyle fractures can sometimes go unnoticed or under-diagnosed as they are not always evident on plain radiographs of the cervical spine. Also, in rare cases OCFs can cause damage to the hypoglossal nerve which passes through the hypoglossal canal which is near the occipital condyle. The presence of specific symptoms and clinical signs should lead to the correct diagnosis. This paper describes a patient who was diagnosed with OCFs, but not hypoglossal nerve damage until 20 days following admission to hospital. We point out many factors that contributed to this delayed diagnosis, which ultimately caused severe discomfort to the patient.

A 25-year-old male was involved in a motor vehicle accident in which he was the passenger. When admitted he had a Glasgow Coma Score (GCS) of E4V4M6 and had a hemotympanum on the left. He was also complaining of cervical pain and tenderness as well as difficulty breathing and speaking. Radiographic investigation revealed that the patient had suffered fractures of the 9th and 10th ribs. Furthermore, this individual had undisplaced fractures of the anterior arch of C1 and of both occipital condyles. A computerized tomographic (CT) scan of the cranial vertebral junction showed that the occipital condyles were medially and inferiorly fractured. The CT scans revealed no other spinal fracture.

Although the patient was conscious on admission he had respiratory obstruction which was due to a retropharyngeal hematoma and also to pharyngeal edema. The patient was intubated the same day and attempts to extubate the patient failed twice. Because of these problems (swallowing difficulty in both oral phase and pharyngeal phase) which were confirmed by bedside testing and a video swallow study done 20 days after admission, bilateral hypoglossal nerve palsy was diagnosed.

The patient was managed expectantly and mobilized in an Aspen collar. Because the patient could not move his tongue, a percutaneous endogastric (PEG) tube was inserted until hypoglossal nerve function recovered. Three weeks following insertion, his tongue movements appeared to be improving to the point that he was no longer having difficulty swallowing secretions.

Fracture of the occipital condyle is a rare entity. As a result, there is very limited knowledge concerning this subject. We can speculate that this injury is more common than reported. The



Figure 1: The coronal view of the craniovertebral junction shows a medial and inferior fracture of both the left and right occipital condyles. The right fracture is indicated by the arrow.

literature supports this claim. In a study done by Bloom et al², thin section CT scans were used to identify OCF in 55 patients who had suffered from high energy blunt trauma to the head or neck. Of these 55 cases, nine had suffered an OCF. Another report done by Bulcholz et al³ examined 100 motor vehicle crash fatalities, among which two cases of OCF were found postmortem.

In accordance with the Anderson and Montesano⁴ classification system of OCF, our patient suffered from a Type I OCF, which is classified as an impacted fracture caused by axial loading of the skull on the atlas. Comminution of the occipital condyles is evident in this type of fracture and there is no displacement into the foramen magnum. This injury is normally

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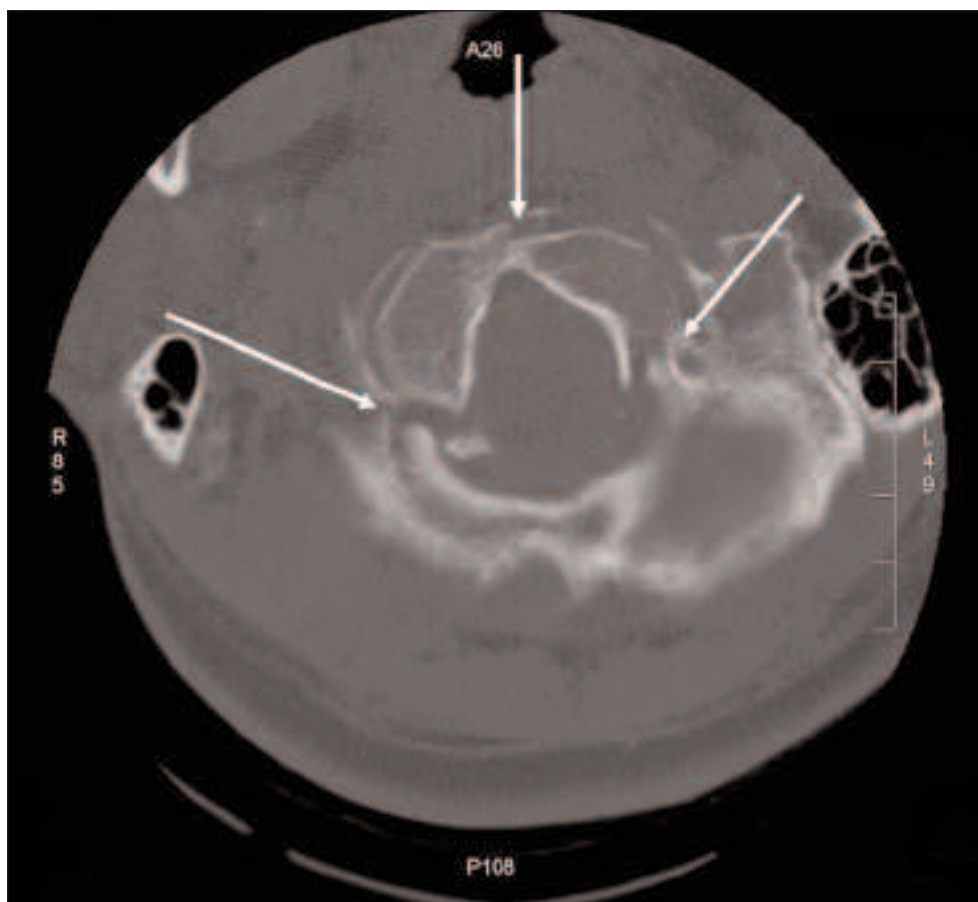


Figure 2: The axial view shows a medial fracture of both the left and right occipital condyles.

considered stable as the tectorial membrane and the alar ligaments remain intact. However, because of the hypoglossal nerve injury in this particular patient, an extra precaution, the placement of the Aspen collar to immobilize the basilar skull fracture, was taken to ensure an optimal recovery. This mode of treatment has been demonstrated in previous studies to effectively immobilize the basilar skull and cervical spine region.⁵⁻⁷

Hypoglossal nerve damage is a secondary effect of an OCF. The hypoglossal nerve emerges from the inferior part of the medulla oblongata and then passes through the hypoglossal canal of the occipital condyle to innervate the intrinsic and extrinsic muscles of the tongue.⁶ Hypoglossal nerve palsy occurs when the nerve has been compressed or avulsed as a result of an OCF. Damage to this nerve causes restricted movement of the tongue.

Injury to the hypoglossal nerve can present clinical symptoms immediately following injury or even months after. In one report⁷ the onset of symptoms occurred three months following injury with symptoms including slurred speech and hemiatrophy of the tongue. Our patient presented symptoms immediately following injury including difficulty in breathing and speaking. However, these symptoms were not attributed to hypoglossal

nerve injury because the patient, in addition to hypoglossal nerve injury, had pharyngeal edema caused by a difficult intubation. Furthermore, although it is common for a patient to lose consciousness following an OCF, there have been many reports in which a patient who has suffered an OCF has maintained a normal level of consciousness.⁸⁻¹⁴ Our patient did not appear to have any other neurological deficit.

In our patient, it may have been possible to identify the damage to the hypoglossal nerve sooner. During his stay, he was intubated for a long period of time because each time he was extubated he would begin coughing. Furthermore, following extubation it was noted that he had difficulty protruding his tongue, which should have been a clear indication of hypoglossal nerve damage. A similar study showed three main reasons why this type of injury is often overlooked.¹⁵ First, clinicians are less sensitive in their evaluation of cases such as ours since there is limited knowledge related to this rare injury. Second, plain radiographs do not provide an accurate assessment of the region of interest. Third, because of the nature of the injury, most patients are unconscious which makes a thorough examination difficult to complete. Our experience with this case, illustrates this.

It has been shown in studies that high-speed deceleration is a common source of injury to the craniovertebral junction.¹⁶⁻¹⁸ We feel, therefore, that it is vital for clinical tests to be done that will effectively identify injury to the hypoglossal nerve whenever there is a history of rapid deceleration and fracture of occipital condyle. Furthermore, we agree with the opinion that OCFs should be included in the list of differential diagnoses in patients with traumatic lower cranial nerve palsies.¹⁹

We prescribe three simple clinical tests that should be employed by clinicians when evaluating an individual who has suffered from an OCF. First, the patient can be asked to protrude the tongue. It will deviate to the paralysed side because of the action of the normal contralateral genioglossus which produces a stronger movement than the other tongue muscles. Second, the patient can be asked to forcefully push on one cheek with the tongue. If after assessing the strength of this push it is found to be weak, then one can conclude that the hypoglossal nerve opposite the cheek (weak contralateral genioglossus) is damaged. Third, one can analyze the tongue itself; if the tongue muscles are wasted then this is a strong indication of a lower motor neuron palsy, that is, a hypoglossal nerve injury.²⁰

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