RADITIONALLY, the diagnosis of skull-base fractures has been made on the basis of clinical findings such as cranial nerve palsies and cerebrospinal fluid (CSF) leaks. It is difficult to define clearly basilar skull fractures on standard x-ray films and such fractures have reportedly been identified in only 20% of cases. Of these cases, occipital condyle fractures (OCFs) make up a separate anatomoclinical entity because of their special features, their rarity, and the fact that they are difficult to diagnose. Due to the strategic anatomical location of the occipital condyles, their traumatic lesions may be accompanied by immediate or delayed paralysis of an individual nerve, or several lower cranial nerve palsies. Reports of OCF have been rare and have consisted mostly of single case reports. These fractures are difficult to diagnose by conventional means and a routine x-ray film may not show enough of the normal anatomical relationships to allow an accurate diagnosis. Therefore, the use of computerized tomography (CT) is vital to obtain an accurate evaluation.

We describe the case of a man who developed isolated glossopharyngeal and vagus nerve paralysis several days after suffering a closed head injury. A depressed fracture of the occipital condyle was diagnosed using CT scans. We discuss the anatomoclinical features, mechanisms, diagnosis, and treatment of this rare lesion.

Case Report

This 62-year-old man suffered a closed head injury in a motor vehicle accident. He was treated initially at another institution, where he was found to have mild confusion and disorientation with no neurological abnormalities.

FIG. 1. Axial computerized tomography scan showing a displaced fracture with bone fragment of the right occipital condyle (arrow).
Occipital condyle fracture and delayed paralysis

The rest of the physical examination was normal. No fractures were seen on initial plain x-ray films of the skull or cervical spine. An emergency CT scan of the brain was considered to be normal, although it did not include the craniocervical junction. After 2 days of observation the patient was neurologically intact, and he was discharged without treatment. A few days later he developed hoarseness, difficulty in swallowing, coughing, and fever. He was admitted to a different hospital where he was treated for a respiratory infection. Because of persistent dysphagia and difficulty in controlling secretions, a nasogastric tube was inserted. Aspiration pneumonia and posttraumatic paralysis of the ninth and 10th cranial nerves were diagnosed. He was discharged from the hospital.

Four months after the accident his symptoms persisted and the patient was sent to our center by his insurance company for a neurological evaluation. On clinical examination the patient was alert and well oriented. Findings included a nasogastric tube in place, head and neck movements that were not limited, and no torticollis. Neurological evaluation revealed that the first through eighth cranial nerves were intact. He suffered hoarseness and dysphagia, the gag reflex was depressed on the right side, and there was a deviation of the uvula to the left side. The trapezius, sternocleidomastoid, and tongue muscles were normal. There was no cerebellar dysfunction and no long tract signs. An otolaryngologist confirmed a paralysis of the right vocal cord with direct laryngoscopy.

Plain x-ray films of the skull and cervical spine were normal. The patient subsequently underwent a high-resolution CT scan that revealed a fracture of the right occipital condyle with a bone fragment in the posterior fossa (Fig. 1). Direct coronal CT with the window level set for bone visualization showed more clearly the avulsion of the right occipital condyle and medial and upper bone fragment displacement (Fig. 2). To obtain more anatomical details of the relationship of the occipital condyle, jugular foramen, and craniocervical junction, we made three-dimensional reconstructions of the scans, which offered a clear anatomical perspective (Fig. 3). Further workup included magnetic resonance (MR) imaging, which showed no intraaxial lesions and occupation of the inferior cerebellopontine cistern recess by the displaced bone fragment (Fig. 4). The nasogastric tube was removed, a gastrostomy was performed, and swallowing rehabilitation was begun. Six months after his cranial trauma, the patient has still not recovered completely.

![Fig. 2. Direct coronal computerized tomography scan with the window level set for bone visualization demonstrating medial and upward displacement of the occipital condyle fragment (arrow). The atlantoaxial joint does not appear to have suffered any dislocation.](image)

![Fig. 3. Three-dimensional reconstruction of computerized tomography views into the craniocervical junction from behind. Left: View clearly showing the upward displacement of bone fragment into the posterior fossa (arrow). Right: Oblique view showing that the morphological appearance of the jugular foramen osseous ring (arrow), located next to the fracture, is normal.](image)

![Fig. 4. Magnetic resonance images showing no intraaxial lesions and inferior cerebellopontine cistern occupation by osseous displacement (arrows). Left: Flash sequence (40°), coronal view. Right: Axial T2-weighted image.](image)
Discussion

Although they are among the more rare traumatic lesions of the skull base and have received little attention, OCFs constitute an anatomoclinical entity because of their special features. The vital neuroanatomical relationship of the occipital condyle with the surrounding structures means that, in theory, traumatic lesions in this area may be accompanied by damage to the brainstem and the cerebellar, vascular, or lower cranial nerves. However, in clinical practice, the most frequently noted neurological deficit in OCF is lower cranial nerve paralysis. Because brainstem and vascular lesions are often life-threatening, some authors attribute their clinical rarity to this fact. Nevertheless, OCFs are very uncommon skull-base fractures. After performing postmortem studies, Bucholz and Burkhead\(^6\) found only two cases of unilateral OCF, and Alker, \textit{et al.},\(^1\) did not find any. Considering the relationship of the occipital condyle to the jugular foramen with the ninth to 11th cranial nerves and to the hypoglossal canal with the 12th cranial nerve, it is not difficult to imagine how an OCF can produce lower cranial nerve dysfunction.\(^9,18,22\) There have been reports of cases with symptoms ranging from isolated paralysis of the hypoglossal nerve\(^21\) to full lower cranial nerve palsies (Collet–Sicard syndrome).\(^15\) There are a variety of possible nerve dysfunction combinations as far as OCFs are concerned, as in our case in which paralysis of the ninth and 10th cranial nerves was found. There have also been case reports of OCF without neurological deficit.\(^2\)

The exact mechanism of injury in this case is uncertain, but previous case reports have indicated hyperextension of the neck associated with a vertical force to the craniocervical junction.\(^15\) Bridgman and McNab\(^3\) have suggested that the occipital condyle may be fractured by a shear force applied to it through the superior facet of the atlas, with the fractured bone displaced into the posterior fossa but remaining attached anteriorly to the odontoid through the strong alar ligaments. The mechanism of injury in our case probably resulted from compression and secondary edema of the ninth and 10th cranial nerves in their cisternal portion caused by the condyle bone fragment and upward displacement. The timing of onset of paralysis is important. Immediate onset paralysis usually indicates a more severe degree of nerve injury, and delayed paralysis is usually due to pressure and secondary nerve edema. Generally, the prognosis for delayed onset paralysis is much more favorable than for the immediate onset variety.\(^6\)

Clinically, OCF is typically suspected in patients who show immediate symptoms of lower cranial nerve palsies; it is less likely to be suspected when the neurological deficit is delayed, and even less so when the patient is neurologically intact. The OCF may not be detected with an initial x-ray examination or even with a routine CT, as happened in our case, in which the CT, which did not include the craniovertebral junction, was considered to be normal. It is well known that OCFs are difficult to diagnose with plain skull x-ray films.\(^24\) In the two cases that Bolender, \textit{et al.},\(^3\) reported, both the plain skull and the cervical spine x-ray films were normal, even in retrospect, as in our case. As Spencer, \textit{et al.},\(^23\) stated, high-resolution CT scanning with sagittal and coronal reconstructions is the diagnostic procedure of choice because it provides adequate visualization of the base of the skull and does not require excessive movement of the patient. Nevertheless, if there is no craniocervical instability, the direct coronal CT with the window level set for bone visualization offers an optimum evaluation in demonstrating the anatomy and morphology of OCF. The three-dimensional CT reconstruction offers us a general perspective of the lesion.

Because of the absence of bone artifacts, MR imaging complements the study demonstrating the relationship between bone fragments, the brainstem, and CSF spaces.\(^17\) In our case it shows the occupation of the inferior cerebellar pontine cistern due to osseous displacement as well as the integrity of the cerebrospinal axis. Because it provides an assessment of ligamentous injury, MR imaging is also helpful in evaluating craniofacial junction stability. In an interesting study, Dickman, \textit{et al.},\(^20\) delineate the normal and pathological anatomy of the transverse alar ligament and conclude that MR imaging accurately depicts the anatomical integrity of the transverse ligament.

The management of this type of lesion has not been well established. Surgery for OCF might be indicated for two reasons: decompression and stabilization.\(^23\) Although surgical neural decompression has been suggested,\(^4\) most authors agree that treatment should be conservative because patients make a good functional recovery from unilateral cranial nerve lesions.\(^13,15,16,20\) If there is dysfunction involving the ninth and 10th cranial nerves, maintenance of adequate nutrition by nasogastric tube or gastrostomy and swallowing rehabilitation, avoiding aspirations whenever possible until the patient has recuperated, is mandatory. Operative stabilization of these fractures has not been reported because OCFs are considered to be stable injuries.\(^16\) Based on fracture morphology, anatomy, and biomechanics, Anderson and Montesano\(^2\) have described three types of OCF. Type I is described as an impacted fracture, type II as a basilar skull fracture with extension into the occipital condyle, and type III as an avulsion fracture. Types I and II are stable and the authors recommend treatment with a semiconstrained cervical orthosis; only type III is potentially unstable and requires rigid immobilization.

References

8. Desai SS, Coumas JM, Danylevich A, et al: Fracture of the...
Occipital condyle fracture and delayed paralysis


Manuscript received April 18, 1995.
Accepted in final form September 5, 1995.
Address reprint requests to: Enrique Urculo, M.D., Service of Neurosurgery, Policlinica Guipuzcoa, Parque Miramon S/N, 20011 San Sebastian, Spain.