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Occipital Condylar Fractures: A Review[1]

The purpose of this review article is to summarize the epidemiology, pertinent anatomy, mechanisms of injury, and classification systems of occipital condylar fractures (OCFs), as well as their clinical presentation and screening, the importance of computed tomography (CT) for detection, and current treatment options. The authors emphasize the rate of occurrence of OCFs, which may be detected in as many as 16% of patients with craniocervical injury. Clinical presentation is not specific, and OCF is not readily diagnosed at physical examination. Failure to diagnose may result in substantial morbidity, and thus accurate diagnosis is mandatory for both therapeutic and medicolegal implications. The diagnosis is most likely to be made with CT. Thin-section CT technique is the method of choice to evaluate the traumatized craniocervical junction. OCFs should be suspected in all patients sustaining high-energy blunt trauma to the head and/or upper cervical spine, resulting from axial loading, lateral bending and/or rotation, and/or direct blow. Besides a CT study assessing potential intracranial injuries, these patients require CT of the craniocervical junction. Radiologists should be aware of the types of OCFs and associated injuries.

Index terms:
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Abbreviations:
CCJ = craniocervical junction
OCF = occipital condylar fracture

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Traditionally thought of as rare, occipital condylar fractures (OCFs) have recently been considered as an underdiagnosed condition that likely occurs with greater frequency than is generally accepted (1). OCFs can easily be missed because the clinical manifestation is highly variable and the results of physical examination are usually nonspecific (2–4); however, long-term morbidity due to pain and limited motion, serious neurologic deficits, or even death may result from undiagnosed or untreated OCFs (5). Thus, an accurate diagnosis is mandatory for both therapeutic and medicolegal implications. OCFs are traumatic lesions of the skull base that are rather frequently, but not necessarily, associated with severe head, brain, and cervical spine trauma resulting from high-speed deceleration insults (6–8). They are unusual clinical injuries because of the strategic anatomic location of the occipital condyles within the craniocervical junction (CCJ) (3,9–11). Familiarity with the types of OCFs, as well as their mechanisms of injury and clinical manifestation, is essential for radiologists evaluating patients with craniocervical trauma. As treatment options change, precise identification of the type and extent of OCF, as well as of concomitant lesions of the CCJ, is becoming increasingly important in determining appropriate management (5).

EPIDEMIOLOGY

In the past, OCFs were considered to be extremely rare because of their difficult detection with conventional radiography. To our knowledge, the first case was identified in 1817 by Sir Charles Bell at the autopsy study of a victim of a fall (12); the second case was described in 1900 (13). The first radiographic evidence of an OCF in vivo was reported in 1962 (14). The first computed tomographic (CT) scans of OCFs were published in 1983 (15,16). Since then, the widespread use of CT and the improvements in its technology, as well as better trauma care, have resulted in an increase in OCF reports in the literature (17–23). However, the true frequency of OCFs is still unknown. In a postmortem radiologic examination of 312 victims of traffic accidents (24), an OCF was reported in two of the 186 patients with head and/or neck injury. In a postmortem analysis of 112 consecutive victims of fatal motor vehicle accidents (25), an OCF was noted in two of the 26 patients with cervical spine injuries. From another postmortem study of 155 persons killed in traffic accidents,

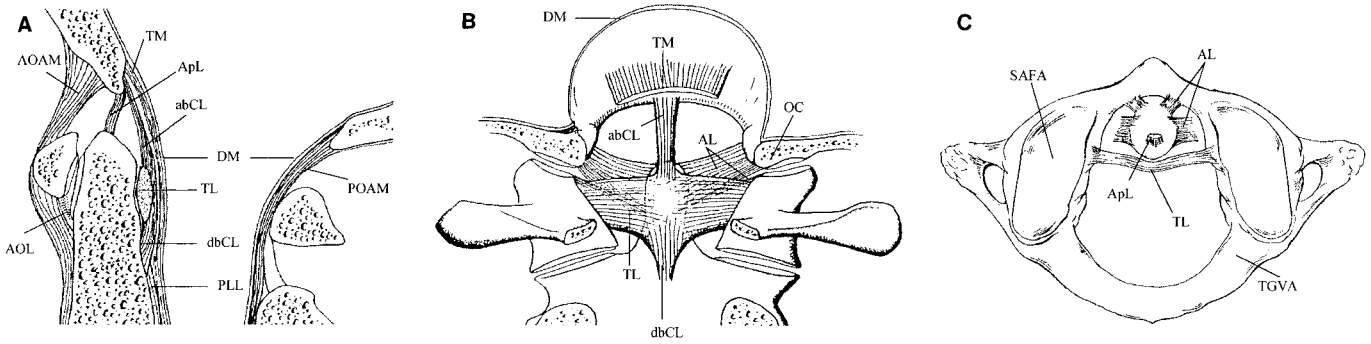


Figure 1. Schematics show the anatomy of the CCJ. *A,* Midsagittal view. *B,* Posterior view of coronal section passing through the occipital condyles and posterior arches of the atlas and axis after dissection of the dura mater, posterior longitudinal ligament, and tectorial membrane. *C,* Transverse view from above of the median atlanto-odontoid joints after dissection of the occiput, dura mater, posterior longitudinal ligament, tectorial membrane, and anterior and posterior occipitoatloid membrane. *abCL* = ascending band of the cruciform ligament, *AL* = alar ligaments (atlantal and occipital portions), *AOAM* = anterior occipitoatloid membrane, *AOL* = atlanto-odontoid ligament, *ApL* = apical ligament, *dbCL* = descending band of the cruciform ligament, *DM* = dura mater, *OC* = occipital condyle, *PLL* = posterior longitudinal ligament, *POAM* = posterior occipitoatloid membrane, *SAFA* = superior articular facet of the atlas, *TGVA* = transverse groove of the atlas for vertebral artery, *TL* = transverse ligament of the atlas, *TM* = tectorial membrane.

OCFs were seen in three of the 66 patients with trauma to the cervical spine or CCJ (26–28). Other authors (29) described the occurrence of OCFs in 25 of 600 patients who died in traffic accidents but did not report the number of patients with craniocervical injuries. As regards the CT evaluation of patients surviving their craniocervical trauma, one study (30) of patients with severe head injury (defined as major trauma resulting in a substantially altered mental status and a Glasgow Coma Scale score of 3–6 on admission) reported a 4% incidence of OCFs. Another study (31) that included patients sustaining severe nonpenetrating cervical trauma with a mean Glasgow Coma Scale score of 10.8 (range, 3–15) reported a 3% incidence of OCFs. In a more recent study (1) that expanded the inclusion criteria to incorporate all patients with appropriate mechanisms of injury (ie, high-energy blunt trauma to the head and neck involving components of either axial compression, lateral bending or rotation, or direct blow), and irrespective of Glasgow Coma Scale score, the resultant incidence of OCFs was 16% (nine of 55).

ANATOMY

The occipital condyles are the prominences of the paired lateral exoccipital segments of the occipital bone, which form the foramen magnum together with the basioccipital segment anteriorly and the supraoccipital or squamosal segment posteriorly (32,33). The bone around the foramen magnum constitutes the uppermost border of an extremely complex three-unit joint with intricate functional relationships between the occiput, atlas, and axis (ie, the CCJ or occipitoatlantoaxial complex) (33–38). The CCJ includes six synovial-lined articulations: the paired occipitoatloid joints, the anterior and posterior median atlanto-odontoid joints, and the paired atlantoaxial joints. The most common shape of the occipital condyle is oval or beanlike; it slopes inferiorly from lateral to medial in the coronal plane and makes an angle with the midsagittal plane of 25°–28° in adults (35) (Fig 1). The occipitoatloid articulations are "cup-shaped" paired joints between the convex surfaces of the occipital condyles and the concave superior surfaces of the articular facets of the atlas. In the coronal plane, both the occipital condyles and the superior articular facets of the atlas slope downward medially. The anterior atlanto-odontoid articulation lies between the anterior arch of the atlas and the anterior aspect of the odontoid process of the axis. The posterior atlanto-odontoid articulation is between the posterior aspect of the odontoid process of the axis and the anterior cartilaginous aspect of the transverse portion of the cruciform ligament (ie, the transverse ligament of the atlas). The atlantoaxial articulations are paired joints between the inferior articular facets of the atlas and the superior articular facets of the axis (Fig 1).

Stability of the CCJ is provided by a number of ligamentous structures that can be divided into two groups according to their attachments. The anterior longitudinal ligament, cruciform ligament, tectorial membrane, and nuchal ligament attach to all three bones. The anterior occipitoatloid membrane, atlantoodontoid ligament, apical ligament of the dens, alar ligaments, posterior occipitoatloid membrane, and the atlantoaxial membrane attach to two bones each (Fig 1). The anterior longitudinal ligament attaches to the anterior body of the axis, anterior arch of the atlas, and anterior inferior edge of the occipital bone after running the entire length of the spine. In the upper cervical spine, the anterior longitudinal ligament appears as a thin, translucent structure. The cruciform ligament has transverse and vertical portions. The transverse portion is the major one and is most commonly known as the transverse ligament of the atlas. It extends between osseous tubercles on the medial aspects of the lateral masses of the atlas and consists almost exclusively of collagen fibers (38). Vertical portions include an ascending band, attached to the anterior edge of the foramen magnum, and a descending band, attached to the posterior aspect of the body of the axis. The tectorial membrane is a broad and fairly strong band that is regarded as the cephalic extension of the posterior longitudinal ligament running from the posterior surface of the body and odontoid process of the axis to the anterolateral edge of the foramen magnum. It is located between the cruciform ligament and the atlas anteriorly and the anterior dura mater posteriorly. The nuchal ligament, which extends from the posterior border of the occiput to the spinous process of C7, is attached to the spinous processes of the cervical vertebrae and the interspinous ligaments. The anterior occipitoatloid membrane runs from the cephalad portion of the anterior arch of

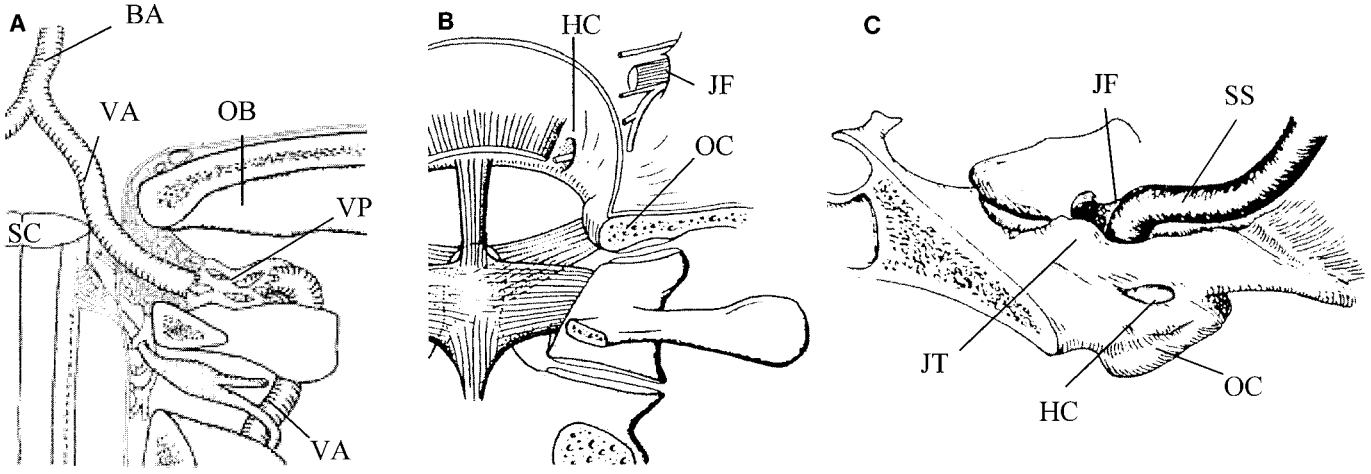


Figure 2. Schematics show the anatomic relationships of the occipital condyles with the surrounding neurovascular structures. *A*, Posterior view of a coronal section passing through the occipital bone and the posterior arches of the atlas and axis. *B*, Posterior view of a coronal section passing through the occipital condyles and the posterior arches of the atlas and axis anterior to the section in *A* after dissection of the spinal cord, vertebral artery, dura mater, posterior longitudinal ligament, and tectorial membrane. *C*, Three-quarter view of sphenoid, temporal, and occipital (anterior and lateral portions) bones. *BA* = basilar artery, *HC* = hypoglossal canal, *JF* = jugular foramen, *JT* = jugular tubercle, *OB* = occipital bone, *OC* = occipital condyle, *SC* = spinal cord, *SS* = sigmoid sinus, *VA* = vertebral artery, *VP* = venous plexus surrounding the vertebral artery. (Modified and reprinted, with permission, from reference 53.)

the atlas to the anterior edge of the occiput and is considered to be part of the anterior longitudinal ligament. The atlanto-odontoid ligament runs between the anterior surface of the odontoid process of the axis to the caudal portion of the anterior arch of the atlas.

The alar ligaments are paired structures that arise from the dorsolateral aspect of the odontoid process of the axis and run obliquely to connect with the inferomedial aspect of the occipital condyles and the lateral masses of the atlas. They consist mainly of collagen fibers, although a few elastic fibers may be identified in the marginal regions (38). The apical ligament of the dens connects the apex of the odontoid process of the axis with the anterior edge of the foramen magnum. It lies between the ascending band of the cruciform ligament and the anterior occipitoatloid membrane. The posterior occipitoatloid membrane attaches to the posterior margin of the foramen magnum and to the posterior arch of the atlas, while the posterior atlantoaxial membrane runs between the posterior arches of the atlas and axis. The basiocciput and the occipital condyles together form the attachments for the paired alar ligaments, the apical ligament of the dens, and the ascending band of the cruciform ligament.

The clinical importance of OCFs is due to the proximity of the occipital condyles to the medulla oblongata, vertebral arteries, and lower cranial nerves (33,39–43). The medulla oblongata, meninges, vertebral arteries, anterior and posterior spinal arteries, and veins that communicate with the internal vertebral venous plexus pass through the foramen magnum, which is bound by the previously described segments of the occipital bone. In particular, the configuration of the atlantoaxial segment of the vertebral artery normally allows about 35° of head and atlas rotation. At the upper atlantal surface, the vertebral artery curves posteriorly to its transverse groove on the atlas, behind the superior atlantal articular facet. The vertebral artery usually does not contact the groove directly but is separated by the vertebral venous plexus. The vertebral artery enters the subarachnoid space by piercing the posterior occipitoatloid membrane and dura mater just medial to the occipital condyle. Within the subarachnoid space, the vertebral artery takes either a straight or (especially in the elderly) a curved path around the lateral and anterior aspects of the spinal cord and medulla oblongata to merge with its counterpart at the lower end of the pons to form the basilar artery (Fig 2a) (39).

Within the base of each occipital condyle lie the hypoglossal (anterior condyloid) canals through which pass the hypoglossal nerve (cranial nerve XII), a meningeal branch of the ascending pharyngeal artery, and an emissary vein (Fig 2b, 2c) (33,43). Lateral to the occipital condyle and hypoglossal canal and posterior to the carotid canal is the jugular foramen (posterior foramen lacerum), which is a true canal containing the cranial nerves IX–XI, inferior petrosal sinus, internal jugular vein, and posterior meningeal artery. It lies inferolaterally within the temporal bone, between its petrous segment, anterolaterally, and the occipital bone, posteromedially (Fig 2b, 2c). The variability in bone formation around the primitive posterior foramen lacerum, the unequal development of the lateral sinuses, and the complex anatomic relationships of the neurovascular structures with each other result in asymmetry and variability of the jugular foramen anatomy (44). The jugular foramen is usually divided into a small pars nervosa anteromedially and a larger pars vascularis posterolaterally by a dural band or, much less commonly, a bony septum, which is attached to the jugular spine of the petrous bone and jugular process of the occipital bone. Through the pars nervosa usually pass the glossopharyngeal nerve (cranial nerve IX), the Jacobson nerve (a branch of cranial nerve IX), and the inferior petrosal sinus. Through the pars vascularis usually pass the vagus nerve (cranial nerve X), the Arnold nerve (a branch of cranial nerve X), the spinal accessory nerve (cranial nerve XI), the internal jugular vein, the posterior meningeal artery, and small meningeal branches of the ascending pharyngeal artery (38–44). Posterior to the occipital condyle is the condyloid fossa, an indentation on the exocranial surface of the skull base. At its anterior margin, the posterior condyloid canal (condylar fora-

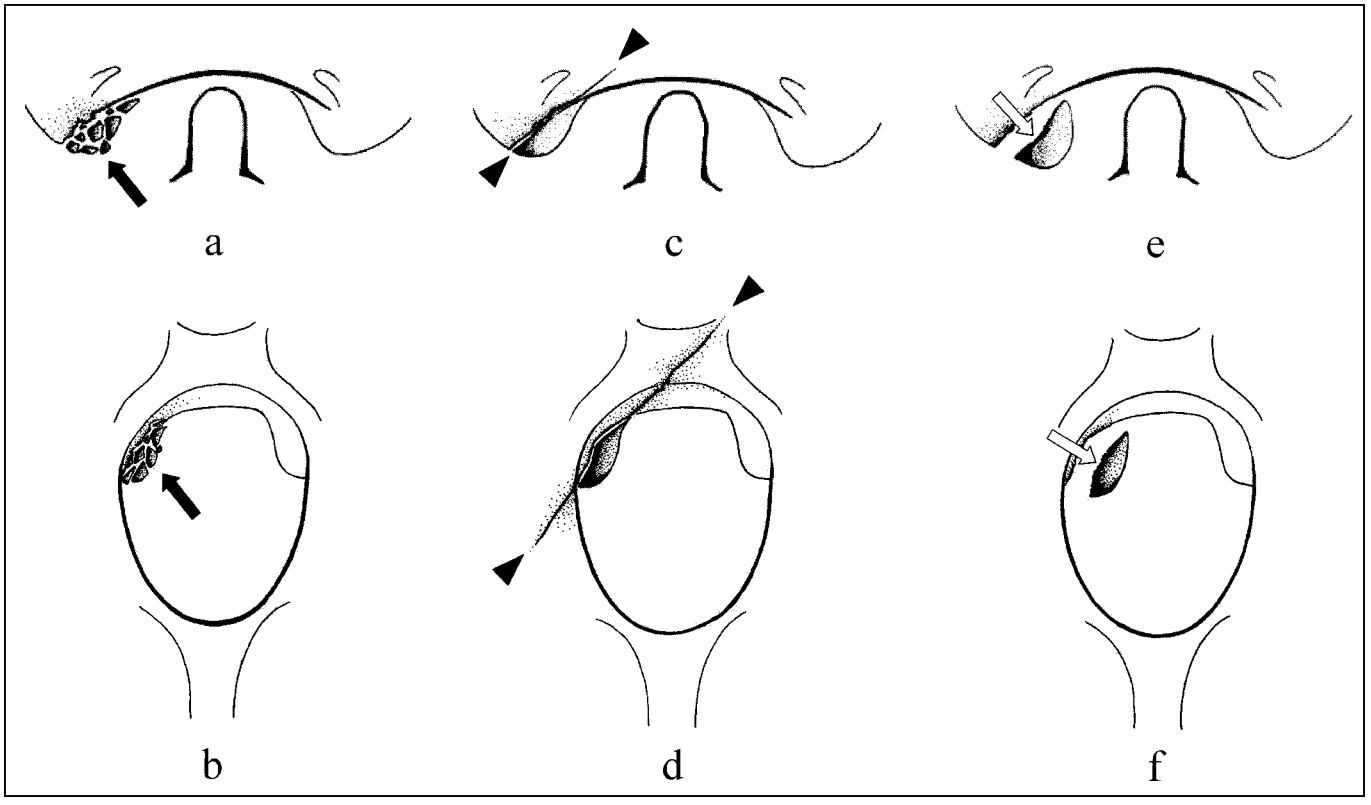


Figure 3. Schematics show the types of OCF according to the classification system of Anderson and Montesano (8). *a*, Coronal and *b*, transverse views from below show a type I OCF, which is a comminuted fracture of the occipital condyle (black arrows) with minimal or no fragment displacement into the foramen magnum. *c*, Coronal and *d*, transverse views from below show a type II OCF, which is a basilar skull fracture (arrowheads) extending into the occipital condyle. *e*, Coronal and *f*, transverse views from below show a type III OCF, which is a fracture with a fragment displaced medially from the inferomedial aspect of the occipital condyle (white arrows) into the foramen magnum.

men) is commonly identified, through which pass anastomotic venous channels from the sigmoid sinus to the suboccipital venous plexus (45).

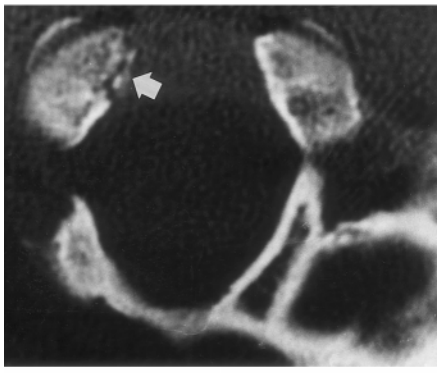
It is clear that a displaced and migrated fragment resulting from an OCF, as well as a through-and-through fracture involving the hypoglossal canal and/or the jugular foramen, can produce impingement on the medulla oblongata and/or vascular structures and/or lower cranial nerves.

MECHANISMS OF INJURY

The CCJ functions as a unit during flexion, extension, lateral bending, and axial rotation; however, it has a limited and specific range of motion (35,46,47). The configuration of the occipitoatloid joint allows good flexion-extension and some lateral bending but negligible axial rotation. At this level, flexion is limited by osseous contact of the anterior portion of the foramen magnum with the odontoid process; however, the major mechanical stability is largely dependent on the integrity of the tectorial membrane, which limits extension, and alar ligaments, which limit axial rotation and lateral bending. The configuration of the atlantoaxial and atlanto-odontoid joints provides good and extensive axial rotation; however, less flexion-extension, and essentially no lateral bending, is allowed. At this level, the major mechanical stability is provided through the odontoid process and the ring of anatomic structures surrounding it (anterior arch of the atlas anteriorly and laterally, transverse ligament of the atlas posteriorly). Flexion is further limited by the tectorial membrane, extension is limited by the tectorial membrane and other posterior structures, and axial rotation is limited by the alar ligaments (35). The configuration of the atlas favors its role as a bearing between the occipital condyles and the superior articular facets of the axis, with cervical movements basically determined by the occipital condyles and the axis (48). The only direct connections between the occiput and the axis are the tectorial membrane, the paired alar ligaments, the apical ligament of the dens, and the ascending band of the cruciform ligament (Fig 1).

From these considerations, as well as the anatomic description, it is readily discernible that the stability of the CCJ is much more dependent on the integrity of the ligamentous structures than on the remaining structures (35,46,47). In case of disruption of the ligaments running from the occiput to the atlas, some attachments remain through the odontoid ligaments. Similarly, in case of failure of the ligaments running between the atlas and axis, some attachments of the axis to occiput still remain. Most CCJ unstable injuries result from the destruction of a number of ligaments in both occipitoatloid and atlantoaxial joints. At the occipitoatloid joint, the most important structures for mechanical stability are the tectorial membrane and the paired alar ligaments. Division of these structures in cadavers resulted in destabilization of the occipitoatloid joints so that dislocation of the skull on the atlas could occur (46).

Another major consideration is that



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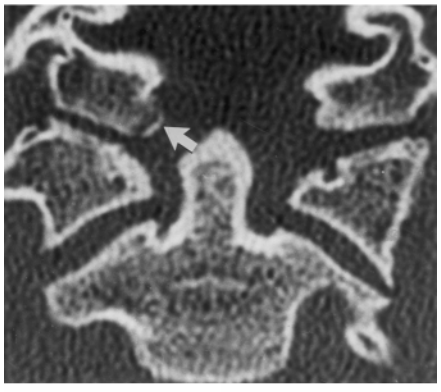
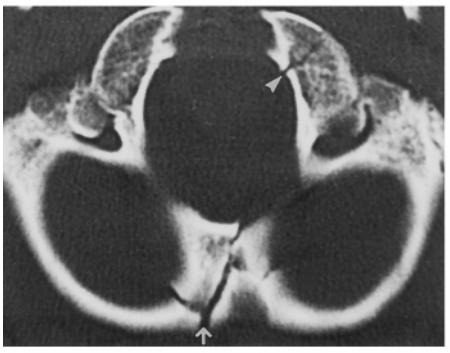


b.

Figure 4. Type I OCF in a 39-year-old woman involved in a motor vehicle accident as a driver. Glasgow Coma Scale score at admission was 13, without lower cranial nerve palsy. **(a)** Transverse and **(b)** direct coronal CT scans demonstrate an impaction fracture of the inferomedial aspect of the right occipital condyle (arrow) with minimal fragment displacement. Associated injuries included facial fractures (not shown). The patient was treated with a soft cervical collar.



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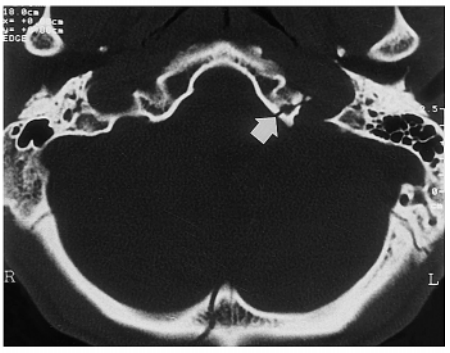

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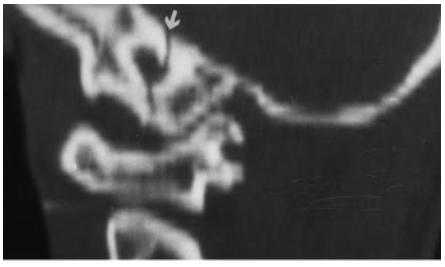


c.

Figure 5. Type II OCF in an 18-year-old man involved in a motor vehicle accident as a pedestrian. Glasgow Coma Scale score at admission was 8, without lower cranial nerve palsy. **(a)** Transverse CT scan shows a linear fracture of the left occipital condyle (arrowhead), which is an extension of a comminuted skull base fracture (arrow). **(b)** Transverse CT scan demonstrates a bone fragment (arrow) medial to the left jugular foramen. **(c)** Two-dimensional oblique sagittal reformation CT image clearly demonstrates the craniocaudal extent of the fracture line (arrow), which involves the osseous ring of the hypoglossal canal. Associated injuries included cortical contusions and a wedge fracture of C6 (not shown). The patient was treated with a halo vest.

trauma involving the CCJ is also influenced by the mass and position of the skull in relation to the long axis of the cervical spine at the time of injury (47). Rarely is a direct axial load supplied to the spine itself; rather, it is transferred from the skull base down through the cervical spine. The location of the force applied to the skull determines the forces transferred to the cervical spine (these include axial loading or asymmetric axial loading with lateral bending, symmetric or asymmetric forces applied to the posterior occiput, and hyperflexion or hyperextension forces, in association with distraction and lateral rotation forces). However, combined injuries are extremely frequent. For example, in motor vehicle accidents, especially rear-end collisions, the head, initially slightly rotated, will go into maximal rotation followed by a "whiplash" movement caused by the impact. In this particular mechanism of injury, the alar ligaments, which limit axial rotation, are most vulnerable (38).

Finally, the configuration of the CCJ resulting from the normal sagittal and transverse diameters of the foramen magnum and cervical spinal canal (the upper portion of which is wider than the lower portion with a relatively greater space for the upper spinal cord) explains how traumatic injuries with fragment displacement can occur with fewer neurologic defects than occur in traumatic lower cervical spine injuries (35). Severe craniocervical injuries, with or without substantial occipitoatloid dislocations, even though unstable, may occur with no neurologic damage (48–50). However, this is complicated by the fact that massive head injury and intracranial trauma often accompany upper cervical injuries.

CLASSIFICATION SYSTEMS

In 1987, Saternus (7) attempted to classify OCFs on the basis of the forms of strain applied. However, the most widely used classification system is the one proposed in 1988 by Anderson and Montesano (8) who divided OCFs into three types, depending on their morphology and mechanism of injury (Fig 3). Type I is an impaction-type fracture resulting in a comminution of the occipital condyle, with or without minimal fragment displacement (Figs 3a, 3b, 4). The mechanism of injury is believed to be axial loading of the skull onto the atlas, similar to a Jefferson fracture of the atlas, with or without lateral bending. It is considered a stable entity because the tectorial membrane and contralateral alar ligament are intact; however, bilateral lesions may be unstable (1). A type II OCF is part of a more extensive basioccipital fracture, involving one or both occipital condyles (Figs 3c, 3d, 5–7). The mechanism of injury is a direct blow to the skull. An intact tectorial membrane and alar ligaments preserve stability. A type III OCF is an avulsion type of fracture near the alar ligament resulting in medial fragment displacement from the inferomedial aspect of the occipital condyle into the foramen magnum (Figs 3e, 3f, 8–10). The mechanism of injury is forced rotation, usually combined with lateral bending. After occipital condylar avulsion, the contralateral alar ligament and tectorial membrane may be stressed and "loaded," resulting in a partial tear or complete disruption. Thus, the type III OCF is considered a potentially unstable injury. The inferior portion of the clivus may be disrupted too (50).

Recently, Tuli et al (5) proposed a new classification system for the management and treatment of OCFs, based on the absence or presence of fragment displace-

ment and stability of the CCJ as assessed with radiographic, CT, or magnetic resonance (MR) imaging evidence of ligamentous injury (Fig 11) (5,35,51). Tuli et al divided OCFs into type 1, or undisplaced, and type 2, or displaced. Type 2 OCFs are subdivided into type 2a if no ligamentous injury is detected and type 2b if ligamentous injury is detected. Types 1 and 2a are considered to be stable lesions, whereas type 2b is considered to be unstable. This functional classification system considers OCFs as part of the wide spectrum of craniocervical injuries, without considering any distinction in OCF anatomy and morphology. Moreover, it suggests that avulsion fractures of the occipital condyle and alar ligament injury represent a necessary, but not sufficient, cause of craniocervical instability (52).

CLINICAL PRESENTATION

The clinical presentation of patients with an OCF is highly variable. Most severe neurologic deficits reported in patients with OCF seem to be related to the severity of head injury rather than to the OCF itself (intraaxial contusion or hematoma, subarachnoid hemorrhage, increased intracranial pressure, etc).

Brainstem and vascular lesions are clinically rare because they are generally fatal. However, cranial nerve deficits (53,54), hemi- or quadraparesis (1,3,10,23), and signs and symptoms of vertebrobasilar ischemia (Fig 10) should alert both the attending physician and the radiologist to the possibility of an associated OCF.

Lower cranial nerve palsy is the most frequently noted neurologic deficit, with varying combinations ranging from isolated paralysis (55–57) to full ninth through 12th cranial nerves palsies (Collet-Sicard syndrome) (57–60). However, according to the review by Tuli et al (5), which was mainly based on case reports, only 31% of 51 patients with an OCF have a lower cranial nerve palsy. Furthermore, in the prospective study by Bloom et al (1), only one of the nine patients with an OCF had a lower cranial nerve palsy. The latter rate of occurrence reflects our experience. In the review by Tuli et al, the manifestations of lower cranial nerve palsy were immediate after trauma in 63% of the cases (5,10,58), with a delay of a few days to months in 37% (5,55,61). It has been hypothesized that a delayed presentation is secondary to the bone healing process and nerve pressure due to callous formation or mobilization of a bone fragment that was not adequately stabilized initially (55). In association with lower cranial nerve palsies, other symptoms have occasionally been reported, such as dysphagia from retropharyngeal hematoma (62) or torticollis from concomitant atlantoaxial rotatory fixation (Fig 6) (11). High cervical pain and impaired skull mobility without loss of consciousness or neurologic deficits have been described in association with OCFs (31,49,50,63,64). Patients are generally unconscious; however, some may remain conscious and responsive (8,49,54).

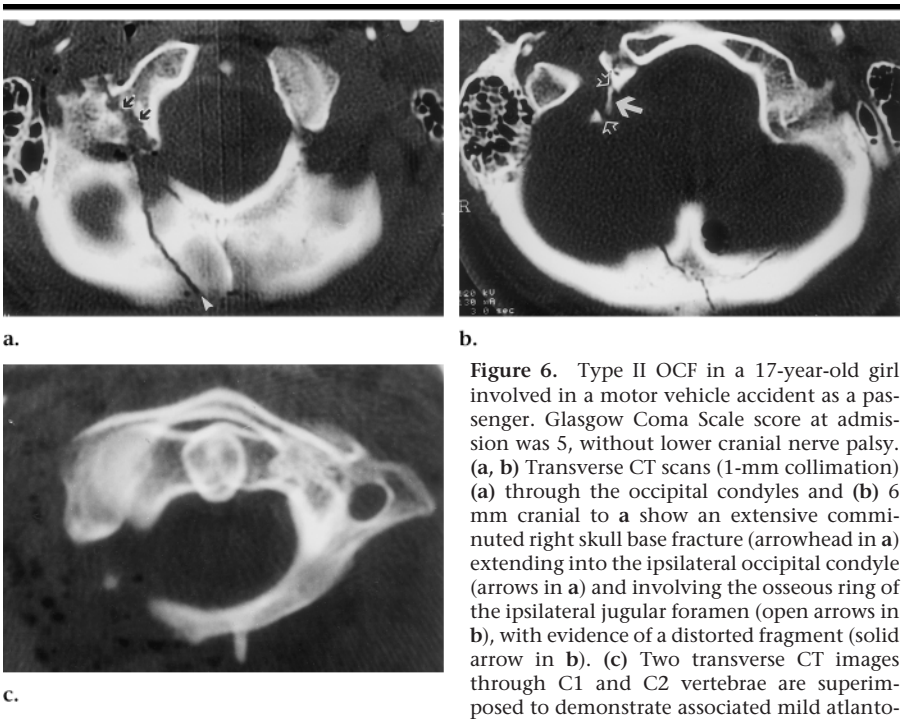
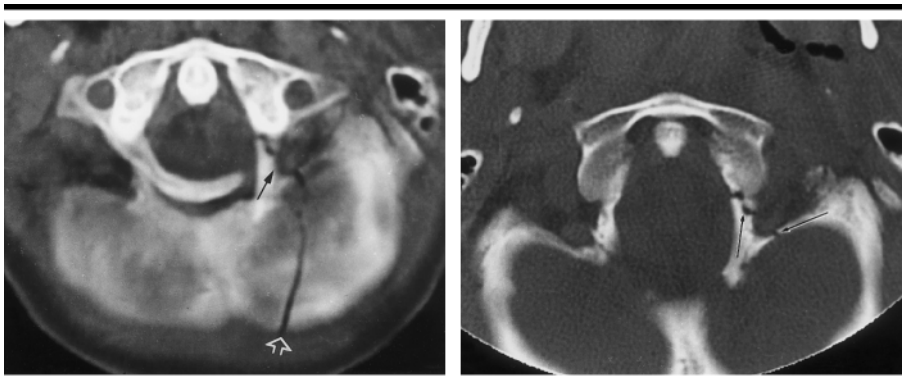


Figure 6. Type II OCF in a 17-year-old girl involved in a motor vehicle accident as a passenger. Glasgow Coma Scale score at admission was 5, without lower cranial nerve palsy. **(a, b)** Transverse CT scans (1-mm collimation) **(a)** through the occipital condyles and **(b)** 6 mm cranial to **a** show an extensive comminuted right skull base fracture (arrowhead in **a**) extending into the ipsilateral occipital condyle (arrows in **a**) and involving the osseous ring of the ipsilateral jugular foramen (open arrows in **b**), with evidence of a distorted fragment (solid arrow in **b**). **(c)** Two transverse CT images through C1 and C2 vertebrae are superimposed to demonstrate associated mild atlantoaxial rotatory subluxation. Associated injuries included right petrous temporal bone fractures and pneumocephalus (not shown). The patient was treated with a Philadelphia cervical collar.

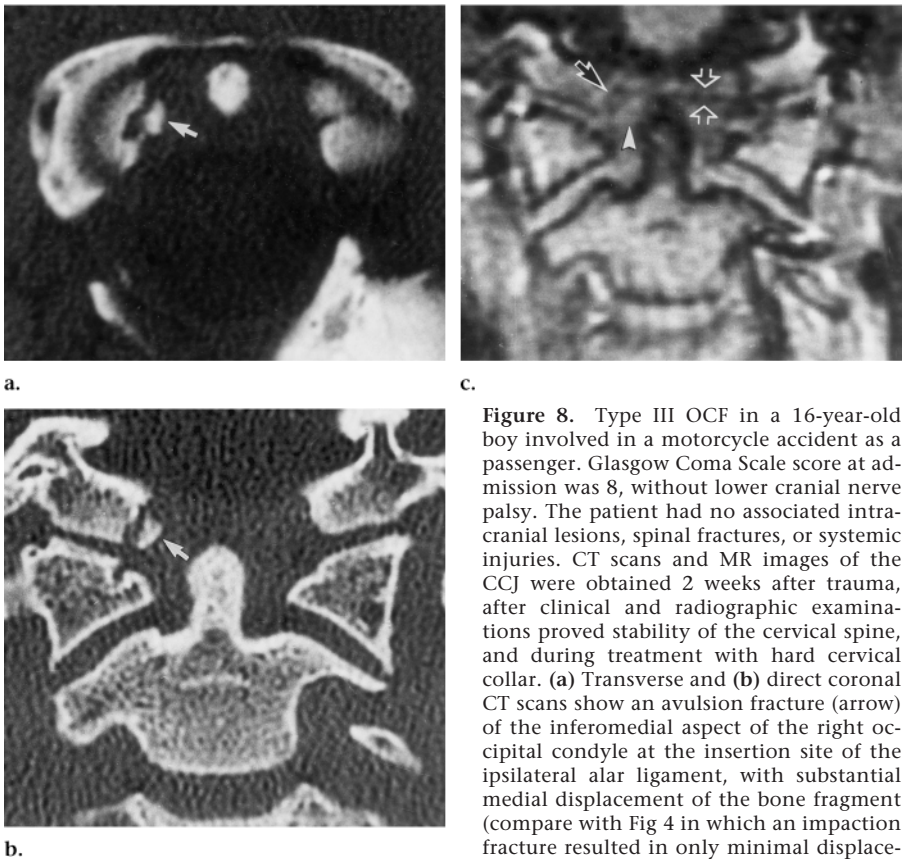
SCREENING

Because of the high variability in clinical presentation, as well as the lack of specificity of signs and symptoms, diagnostic imaging is essential for the diagnosis of OCFs. Skull and cervical spine radiographs obtained routinely in patients with multiple trauma generally do not show any abnormality because of facial skeleton superimposition. CT is the method of choice for the diagnosis of OCFs. But when should a CT study of the CCJ to search for an OCF be performed? Some authors (2,3) have suggested the following as parameters that should arouse suspicion for an OCF: presence of posttraumatic palsies of cranial nerves IX, X, XI, or XII; retropharyngeal or prevertebral soft-tissue swelling; occipital skull base fracture; fracture or dislocation of the axis or atlas; posttraumatic spasmodic torticollis; or unexplained persistent posttraumatic upper-neck pain with normal conventional radiographs. However, the patient's consciousness may be so impaired that detailed testing of cranial nerves is not possible (33). Furthermore, as already stated, cranial nerve palsy should be considered uncommon and may be related to different mechanisms altogether (brainstem contusion, reversible ischemia, etc). Additional causes of neck pain and prevertebral soft-tissue swelling are not uncommon in patients who have experienced severe trauma, such as those associated with midfacial fractures (65). We think, as other authors do (1,2,4), that there are no specific predictors of OCF. Clinical findings are generally inconsistent, and, in our experience, most patients had mild to moderate Glasgow Coma Scale scores. Thus, we agree that, despite the clinical presentation and Glasgow Coma Scale score, OCF must be suspected in all patients sustaining high-energy blunt trauma to the head and/or the upper cervical area resulting from axial loading or rotation, lateral flexion or bending, and/or direct blow (1,8).



a. b.

Figure 7. Type II OCF in a 23-year-old woman who fell from a horse. Glasgow Coma Scale score at admission was 10, without lower cranial nerve palsy. These **(a)** 5-mm and **(b)** 1-mm collimation transverse CT scans clearly demonstrate a left basilar skull fracture (open arrow in **a**) extending ipsilaterally through the jugular foramen and into the occipital condyle (solid arrows). Associated injuries included right subdural hematoma and a wedge fracture of T12 (not shown). The patient was treated with a hard cervical collar.



a. c.

b.

Figure 8. Type III OCF in a 16-year-old boy involved in a motorcycle accident as a passenger. Glasgow Coma Scale score at admission was 8, without lower cranial nerve palsy. The patient had no associated intracranial lesions, spinal fractures, or systemic injuries. CT scans and MR images of the CCJ were obtained 2 weeks after trauma, after clinical and radiographic examinations proved stability of the cervical spine, and during treatment with hard cervical collar. **(a)** Transverse and **(b)** direct coronal CT scans show an avulsion fracture (arrow) of the inferomedial aspect of the right occipital condyle at the insertion site of the ipsilateral alar ligament, with substantial medial displacement of the bone fragment (compare with Fig 4 in which an impaction fracture resulted in only minimal displacement of the bone fragment). **(c)** Coronal T1-weighted MR image shows a subtle area of high signal intensity (arrowhead) in the soft tissues medial to the bone fragment (solid arrow), with retraction of the atlantal portion of the ipsilateral alar ligament. This area showed high signal intensity also on the T2*-weighted images (not shown) and was most likely due to edema, inflammation, and hemorrhage at the site of ligamentous injury. The open arrows indicate the normal atlantal portion of the left alar ligament.

RADIOLOGIC DIAGNOSIS

The radiographic evaluation of the CCJ has traditionally been difficult, and a number of lesions may be undetected or poorly understood (65,66). Occipital condyles may be visualized on some skull views; however, it is difficult to clearly define basilar skull fractures on standard radiographs obtained in traumatized patients, such fractures having been reported in only 20% of cases (67). Both anteroposterior and lateral (cross-table technique) views of the cervical spine fail to depict the occipital condyles because of superimposition of the maxilla and occiput on the anteroposterior view and superimposition of the occipital condyles themselves and, frequently, of mastoid processes on the lateral view. Furthermore, the evaluation of a prevertebral soft-tissue shadow on the lateral view may be limited by many factors such as patient positioning, endotracheal intubation, or adenoidal tissue. The open-mouth (odontoid) view is designed to demonstrate the atlantoaxial relationship in the anteroposterior projection and may include the occipital condyles and the occipitoatloid joints; however, this view is impossible to obtain in patients who are unconscious or intubated or have severe mandibular or facial injuries (65,68,69). In the past, the diagnosis of OCF was obtained with conventional or complex motion tomography; however, discrete impaction fractures could be missed.

Since its introduction, CT has been considered the method of choice for the diagnosis of OCF; however, a routine brain CT examination in traumatized patients may not enable detection of these fractures (61). Rather than brain CT to assess a potential intracranial injury (70), cranial CT including the CCJ should be performed. The CT study should routinely include 5-mm-thick transverse images beginning at the lower border of C2. Images must be reviewed with window width and level optimized for the evaluation of both brain parenchyma and osseous structures. If required, the scan data can then be reviewed again using a high-spatial-resolution, bone- or edge-enhancement reconstruction algorithm. In this way, important information can be obtained in a very short period of time. In cases with a high index of suspicion, the radiologist must complement the examination with a study of the CCJ. A thin-section technique is the method of choice to evaluate the traumatized CCJ (71). Generally, 1–2-mm-collimation transverse sections, 1-mm table indexing, 1-second scanning, 200–240 mAs, 120 kV, 12- to 14-cm display field of view, and a high-spatial-resolution algorithm are best for assessment of skull base and CCJ anatomy. Both direct transverse scanning and two-dimensional multiplanar reformations are strongly recom-

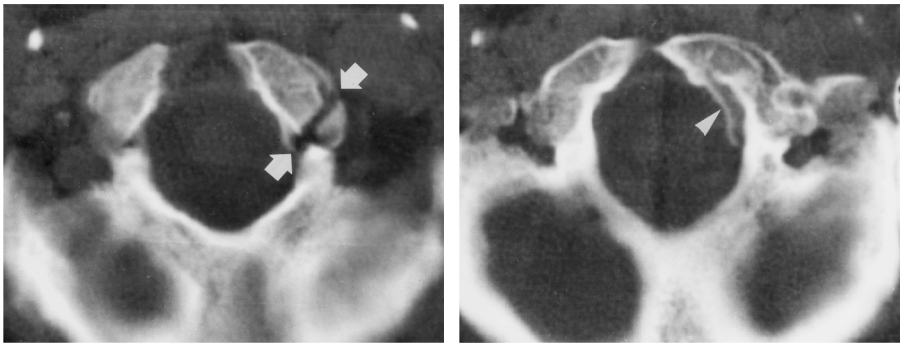


Figure 9. Type III OCF in a 24-year-old woman involved in a motor vehicle accident as a passenger. Glasgow Coma Scale score at admission was 7, with cranial nerves IX and X palsy. Transverse CT scans (1-mm collimation) **(a)** through the occipital condyles and **(b)** 3 mm cranial to **a** show left OCF (arrows in **a**) with fragment displacement (arrowhead in **b**) into the foramen magnum. Associated injuries included lateral craniocervical subluxation (not shown). The patient was treated with occipitocervical fusion.

mended for the most accurate assessment of the type of fracture and degree of CCJ displacement (72). Direct coronal scanning is not advisable in unstable patients or in patients with potential or confirmed spinal fractures and/or systemic injuries. Three-dimensional shaded-surface reconstruction CT images are dramatically impressive in displaying the fracture and detailing its extent; however, their diagnostic value is limited (73,74). Helical (spiral) CT scanning enables high-quality coronal and sagittal reformations and three-dimensional reconstructions of overlapping transverse images, with a minimum of motion artifact and without additional radiation exposure. The study may be obtained with a collimation of 1 mm, a pitch of 1 mm, and a section reconstruction interval of 1 mm. It is advisable to obtain the CT scan as soon as possible after craniocervical trauma for early detection of an OCF and anticipate possible complications that may be clinically silent. Follow-up CT is then recommended, 10 or 12 weeks after injury, to document fracture healing (3).

MR imaging does not yield relevant additional diagnostic information concerning the OCFs but is the best ancillary diagnostic tool complementing CT for evaluation of associated soft-tissue craniocervical trauma. The application of MR imaging in the assessment of ligamentous structures, particularly the tectorial membrane and the transverse ligament of the atlas, is well established and continually increasing (36,50,51,75). MR imaging is extremely valuable for the evaluation of the fractured segment in relation to the surrounding structures (ie, the cerebrospinal fluid spaces, brainstem, and neurovascular structures). In those patients with suspected vascular injury, the use of MR angiography may prevent the necessity of conventional angiography (Fig 10) (3,10,18,58). Additionally, MR imaging is better than CT for the assessment of associated brain and brainstem injuries, as well as for intracranial

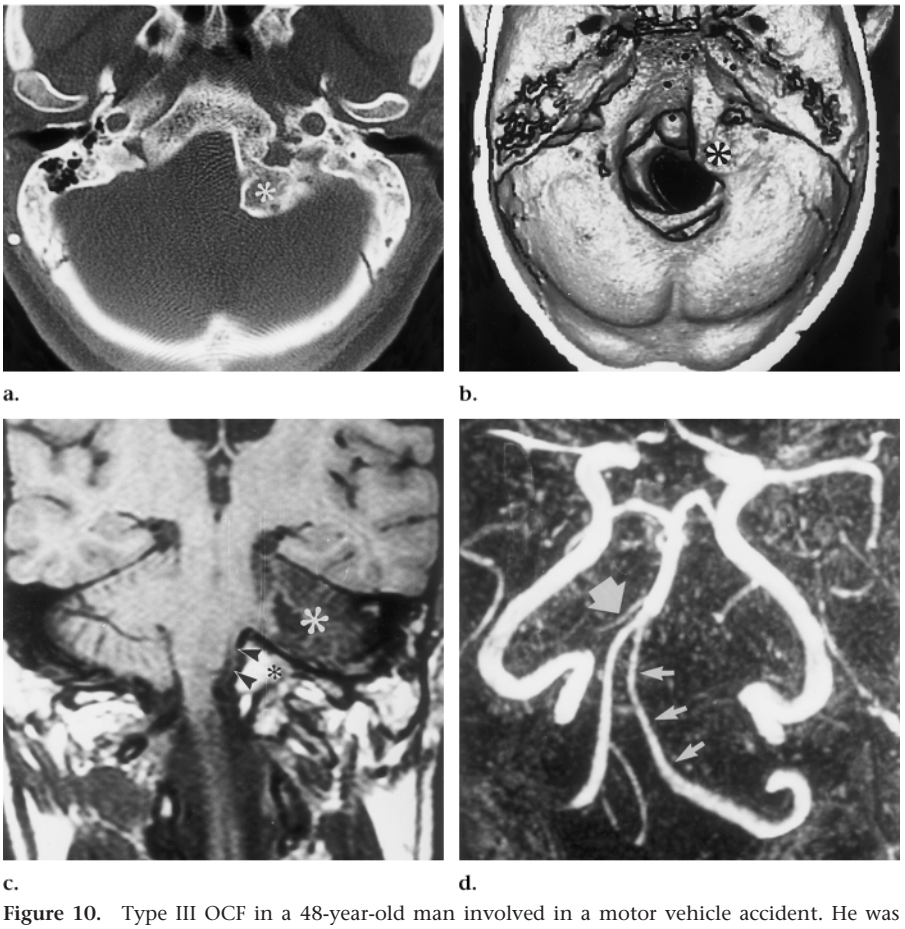


Figure 10. Type III OCF in a 48-year-old man involved in a motor vehicle accident. He was admitted to another institution where CT scans suggested a fracture of the left occipital condyle with superomedial displacement of a bone fragment. The patient was treated with a hard cervical collar and, 3 months later, was transferred to our institution for further treatment. On admission, the patient was conscious and had a left Collet-Sicard syndrome and signs of cerebellar dysfunction. **(a)** Transverse CT scan and **(b)** surface rendered three-dimensional CT reformation from above demonstrate a healed left OCF with medial upward displacement of a bone fragment (asterisk). **(c)** Coronal T1-weighted MR image better depicts the displacement of the left occipital condylar fragment (black asterisk) in the posterior fossa, impinging on the medulla (arrowheads); a left cerebellar infarction is clearly seen (white asterisk). **(d)** Frontal reconstruction of a three-dimensional time-of-flight MR angiogram shows that the distal left vertebral artery is distinctly narrowed and displaced (small arrows) by the medial upward displacement of the left occipital condylar fragment. The source images (not shown) demonstrated the related cranial displacement and entrapment of the caudal loop of the left posteroinferior cerebellar artery and lack of evidence of the left anteroinferior cerebellar artery. Large arrow = right anteroinferior cerebellar artery.

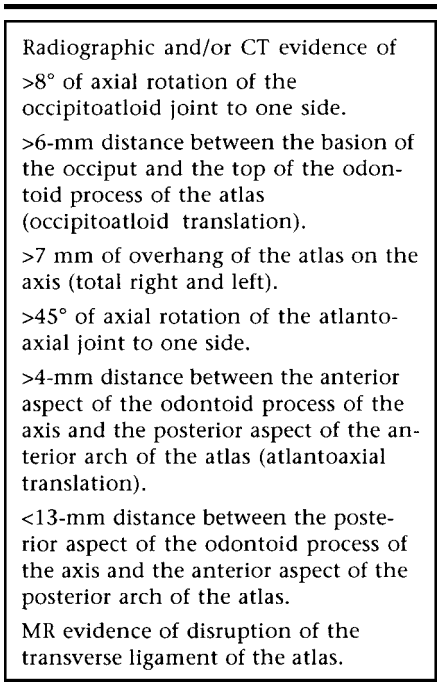


Figure 11. Imaging criteria for CCJ instability (5,35). On the basis of the results of radiography, CT, and/or MR imaging, CCJ is considered to be stable if none of the criteria are detected and unstable if a single criterion or a combination of them are detected (5).

hemorrhage, although CT is still considered the current standard for the evaluation of acute subarachnoid hemorrhage (76,77).

MANAGEMENT

To date, management of OCFs has not been well established because of the small number of cases described in the literature, as well as the lack of prospective studies investigating follow-up of the different treatment modalities. Furthermore, the long-term implications of OCFs are not well known. In the review by Tuli et al (5), four of the six patients who did not receive treatment developed deficits, such as delayed cranial nerves IX through XII or IX and X palsy (53) and delayed (55) or fluctuating (19) isolated cranial nerve XII palsy (5). The therapeutic strategy is generally conservative. The need for surgery is controversial and has been advocated for craniocervical stabilization and/or neurovascular decompression. Treatment is initially directed toward reduction and stabilization with external fixation. Most authors treat Anderson and Montesano types I and II OCFs with a semirigid or rigid cervical collar and type III with a rigid cervical collar, halo traction vest, or surgical fixation (1,3,61). Tuli et al (5) have proposed that undisplaced OCFs do not require immobilization, displaced OCFs with a stable CCJ may be treated with a hard cervical collar, and displaced OCFs with an unstable CCJ require rigid external fixation or surgical fixation. However, craniovertebral subluxation is usually treated with cervical traction and early immobilization in a halo vest (71). The halo traction vest allows adjustments in reduction and helps maintain the correct position during and after surgery. Surgical fixation of the CCJ is performed by means of posterior fusion (occipitoatlantoaxial arthrodesis). The two common approaches involve the use of a bone graft with or without wire (78–81), but several technical innovations have been applied to these two conventional methods (82,83).

Removal of a fracture fragment compressing the vertebral artery and/or the brainstem has been performed in patients with a stable CCJ (10,84); however, most authors suggest that conservative therapy may suffice, even when brainstem compression is present (3).

CONCLUSION

Accurate determination of the true incidence of OCF is difficult because the traumatized patient may be asymptomatic or the condition may be masked by death or concomitant injuries or may be delayed in manifestation. Nevertheless, OCFs should not be considered uncommon, occurring possibly in as many as 16% of patients with craniocervical injury. OCF should be suspected in all patients sustaining high-energy craniocervical trauma from an appropriate mechanism of injury (ie, high-energy blunt trauma to the head and/or neck involving components of either axial compression, lateral bending, axial rotation, or direct blow), regardless of the clinical condition and physical examination results. The great potential of these fractures for long-term morbidity due to pain and limited motion, serious neurologic deficits, or even death explains the rising therapeutic and medicolegal implications of an accurate diagnosis. Clinically, an OCF is generally suspected in patients immediately showing symptoms of lower cranial nerve palsy, because of the lack of specificity of brainstem-related symptoms. OCF is less likely to be suspected when the neurologic deficit is delayed, or even lesser when the patient is unconscious or neurologically intact. Besides a CT study assessing potential intracranial injuries, these patients also require a CT study of the CCJ.

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