Skull Base Fractures and Their Complications

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KEYWORDS

• Skull base trauma • CSF leak • Basilar skull fracture • CSF rhinorrhea

KEY POINTS

- Skull base fractures are managed based on associated intracranial injury and complications, including vascular and cranial nerve injury and cerebrospinal fluid (CSF) leak.
- Anterior cranial fossa fractures, particularly comminuted and oblique frontobasal fractures, are commonly associated with CSF leak, either acute or delayed in presentation.
- Transverse middle cranial fossa fractures extending through the carotid canal are at increased risk for vascular injury, and should prompt screening with vascular studies, such as CT angiography.
- Thin-section multiplanar CT reformations, as well as 3-dimensional reconstructions, are helpful in the detection of subtle skull base fractures.

INTRODUCTION

Head trauma is one of the most common reasons for visits to the emergency department in the United States. According to the 2013 National Trauma Data Bank maintained by the American College of Surgeons, of 833,311 adult trauma admissions reported from 805 facilities across the United States, approximately 36% sustained an injury to the head.[1] Skull base fractures, those fractures that extend through the floor of the anterior, middle, or posterior cranial fossa, occur in an estimated 7% to 16% of nonpenetrating head injuries, and are due to a relatively high-velocity trauma, most often high-speed motor vehicle accidents, although motorcycle collisions, pedestrian injuries, falls, and assault are additional associated causes.[2] Penetrating trauma, particularly gunshot wounds, are seen much less frequently, accounting for less than 10% of cases.[3]

Skull base injury is often seen in the setting of complex facial or orbital fractures, and detection of basilar skull fractures is important, as even linear nondisplaced fractures can be associated with numerous critical complications, including intracranial and orbital injuries, cerebrospinal fluid (CSF) leak, cranial nerve palsies, and vascular injuries. Although facial fractures often require repair to improve function and cosmesis, the management of patients with skull base injury is dependent on the extent of associated intracranial injury and other complications. The associated risk and extent of complications often depends on the location and pattern of the fracture, which is in turn determined by the mechanism of injury and type of impact.

NORMAL ANATOMY

The skull base is made up of 7 bones, the paired frontal and temporal bones, and the unpaired ethmoid, sphenoid, and occipital bones. It is divided into anterior, central, and posterior regions, which form the floor of the anterior, middle, and posterior cranial fossae.

The anterior skull base, formed by the frontal and ethmoid bones, separates the anterior and inferior frontal lobes and olfactory structures within

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the anterior cranial fossa from the orbits and the sinonasal cavity. The lateral and anterior borders of the anterior cranial fossa are formed by the orbital plate of the frontal bone and the posterior table of the frontal sinus. Inferiorly, the floor of the anterior cranial fossa is formed by the cribriform plates and roof of the ethmoid sinuses. The posterior border between the anterior and central skull base is formed by the lesser wing of the sphenoid bone, including the clinoid process, and the planum sphenoidale (Fig. 1).

Deep clefts lateral to the midline crista galli form the olfactory grooves, which house the olfactory bulbs. The floor of the olfactory groove is formed by the cribriform plates, which are inherently thin, with multiple small foramina through which the small branches of the olfactory nerve pass. The lateral lamella is a thin bone connecting the cribriform plate with the fovea ethmoidalis, or the roof of the ethmoid sinuses, all part of the ethmoid bone. In addition to the cribriform plate foramina, the anterior skull base contains the anterior and posterior ethmoid artery foramina, which should not be confused with fractures; these may represent significant sources of epistaxis, if injured (Fig. 2).

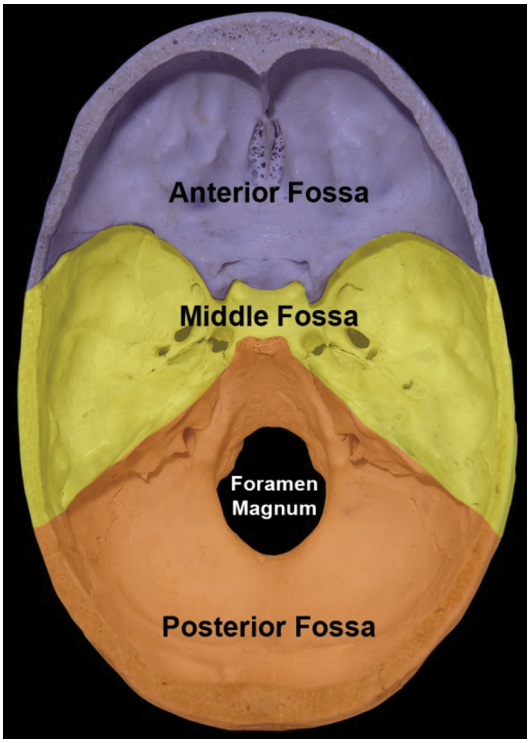

Fig. 1. Axial photograph of the skull base with overlays demonstrating the anterior, middle, and posterior cranial fossae. (*Courtesy of* Kevin Makowski and Eric Jablonowski, Emory University, Atlanta, GA.)

The central skull base, formed by the sphenoid and anterior temporal bones, separates the pituitary gland (within the sella), the cavernous sinuses (including the carotid artery and cranial nerves), the Meckel cave, and the temporal lobes superiorly from the sphenoid sinus anteriorly and inferiorly, and the extracranial soft tissues deep to the skull base inferiorly, including the masticator, parotid, parapharyngeal, and pharyngeal mucosal spaces. The anterior border of the central skull base is formed by the posterior margin of the lesser wing of the sphenoid bone, clinoid process and tuberculum sella. The floor is formed by the greater wing and central body of the sphenoid bone, the sphenoid sinus, and the sella. The posterior border between the central and posterior skull base is formed by the superior margin of the petrous ridge of the temporal bone, the basi sphenoid portion of the clivus, and the dorsum sella (see Fig. 1). In addition to housing the pituitary gland, the central skull base contains numerous foramina and canals through which many important structures pass, including cranial nerves (CNs) II to VI and the internal carotid artery (Fig. 3, Table 1).

The posterior skull base is formed by the posterior temporal bone and the occipital bone, and separates the posterior fossa structures, including the cerebellum and brainstem, from the extracranial soft tissues: the posterior nasopharynx, retropharyngeal space, carotid space, and perivertebral space. The anterior border is formed by the petrous ridge of the temporal bone superiorly, and the clivus (basi occiput portion) inferiorly. The inferior border includes the occipital condyles and the mastoid portion of the temporal bone, and the posterior skull base extends posteriorly to the squamous portion of the occipital bone (see Fig. 1). Some consider the temporal bone proper to be the lateral, or posterolateral skull base.

The largest foramen of the skull base, foramen magnum, is located within the posterior skull base, and transmits the medulla oblongata (cervicomedullary junction), vertebral arteries, and spinal portion of CN XI. Other major foramina within the posterior skull base include the internal auditory canal (CN VII, VIII, and labyrinthine artery), jugular foramen (pars nervosa anteriorly: CN IX, inferior petrosal sinus, and Jacobsen nerve; pars vascularis posteriorly: CN X, XI, Arnolds nerve and jugular bulb), and hypoglossal canal (CN XII) (Figs. 4 and 5).

PATHOLOGY

Fractures through the skull base are often the sequelae of high-velocity impact and may be linear

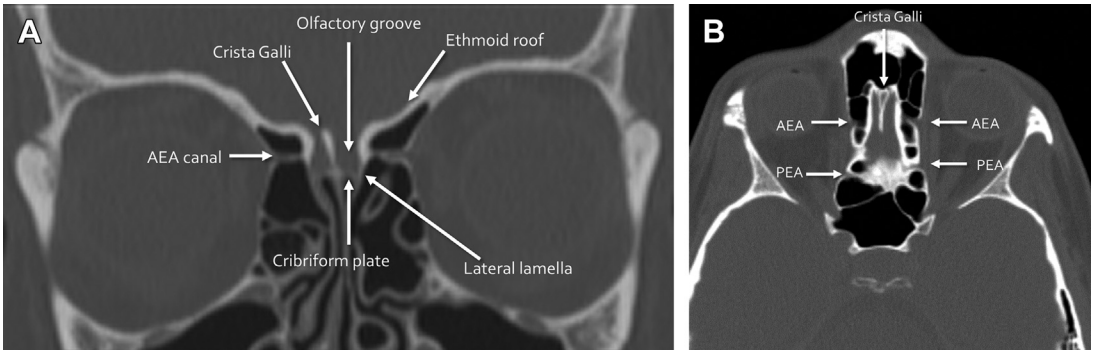

Fig. 2. Anterior skull base. (*A*) Coronal CT demonstrating the olfactory groove, bordered by the crista galli, cribriform plate, and lateral lamella. The lateral lamella extends cranially to form the ethmoid roof. Note the horizontal trajectory of the anterior ethmoid artery canal, with its corticated margins and medial tapering. (*B*) Axial CT showing bilateral anterior and posterior ethmoid artery canals (AEA and PEA respectively), corticated and in a characteristic location that should not be mistaken for fracture.

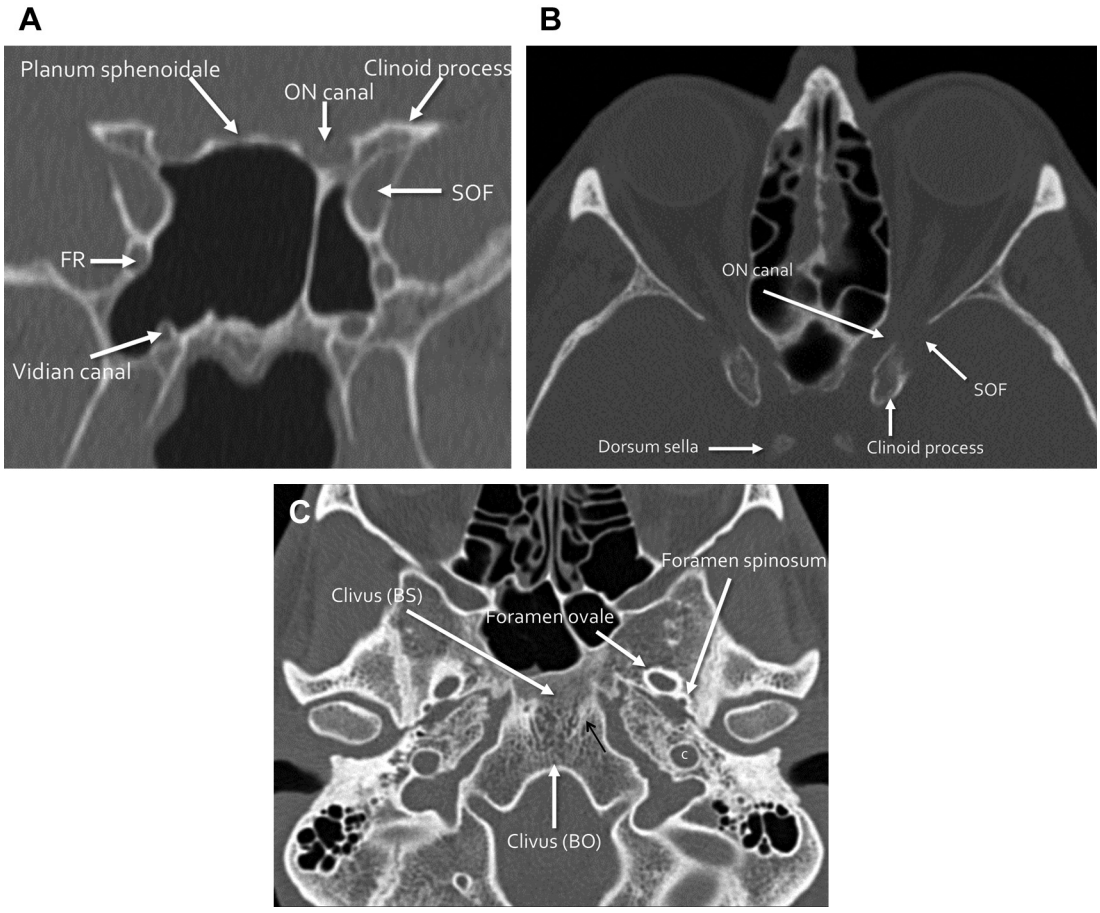

Fig. 3. Central skull base. (*A*, *B*) Coronal and axial CT image demonstrating the relationship of the optic nerve and superior orbital fissure to the clinoid process, with the optic nerve medial to the clinoid process, and the superior orbital fissure inferior and lateral to the clinoid process. Note the planum sphenoidale, foramen rotundum (FR) transmitting V2 along the lateral sphenoid sinus, and vidian canal transmitting the vidian nerve inferomedially. (*C*) Axial CT through the middle cranial fossa floor showing the "high-heel shoe" appearance of foramen ovale (the "toe of the shoeprint"), and foramen spinosum (the "heel") within the sphenoid bone. The carotid canal ("c") enters the petrous portion of the temporal bone, extending cranially with vertical and horizontal segments. The basisphenoid (BS) portion of the clivus superiorly and anteriorly is located within the middle cranial fossa, whereas the basi occiput (BO) portion of the clivus is a portion of the occipital bone, within the posterior cranial fossa. Note the sclerotic fused sphenooccipital synchondrosis/fissure (*thin black arrow*). ON, optic nerve; SOF, superior orbital fissure.

Table 1
Central skull base foramina and their contents

Foramen	Contents
Optic nerve canal	CN II (optic nerve) Ophthalmic artery
Superior orbital fissure	CN III (oculomotor nerve) CN IV (trochlear nerve) CN V1 (ophthalmic branch of the trigeminal nerve) CN VI (abducens nerve) Superior ophthalmic vein
Foramen rotundum	CN V2 (maxillary branch of trigeminal nerve)
Foramen ovale	CN V3 (mandibular branch of trigeminal nerve)
Carotid canal	Internal carotid artery Sympathetic plexus
Foramen spinosum	Middle meningeal artery
Vidian canal	Vidian nerve and artery
Foramen lacerum	Cartilage

Abbreviation: CN, cranial nerve.

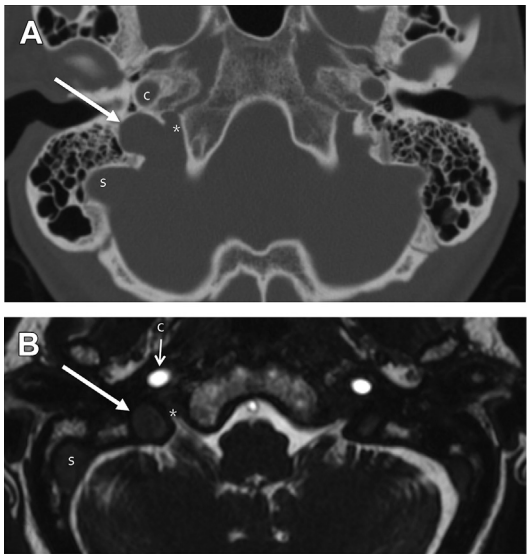

Fig. 4. Posterior cranial fossa and skull base. (*A*, *B*) CT and MRI images showing the sigmoid sinus (s) as it extends caudally toward the jugular foramen (*arrow*), with the dominant pars vascularis posteriorly and the pars nervosa (*asterisk*) anteromedially. The pars nervosa contains the inferior petrosal sinus and glossopharyngeal nerve (seen on T2-weighted MRI). Note the proximity of the jugular foramen to the carotid canal (c).

or comminuted, with a potentially complex imaging appearance. Fracture patterns and associated complications depend on the location of the injury, which is often determined by the mechanism of injury and type of impact. **Table 2** outlines the most commonly encountered complications associated with site of injury.

Anterior Skull Base Fractures

Classification/Etiology

Direct frontal trauma often results in anterior skull base fractures, so-called frontobasal injuries, with the "frontal" component of fractures involving the upper facial third (frontal bone/sinus and superior orbital rim), and "basal" component of fracture involving the anterior skull base (cribriform plate, ethmoid roofs, and planum sphenoidale). Numerous classification systems for frontobasal injuries have been described in an attempt to stratify complications and guide management. One of the more recent anatomic classification systems evolved based on cadaveric studies demonstrating unique reproducible fracture patterns, similar to Lefort facial fractures, and classifies frontobasal fractures into types I through III.[4,5]

Type I frontobasal fractures are generally associated with a relatively lower impact frontal injury, and are defined as linear fractures that initially parallel the cribriform plate, then may extend posteriorly along the sella and petrous ridge to separate the anterior and middle cranial fossa from the posterior cranial fossa. These generally are more medially located, involving the medial third of the supraorbital rim (**Fig. 6**), and are less frequently associated with complications. Type II fractures are composed of more lateral vertical linear fractures of the frontal calvarium and anterior skull base and often extend to involve the lateral two-thirds of the supraorbital rim, the squamous portion of the temporal bone, the orbital roof, the lateral orbital wall, or the orbital apex. These are more frequently associated with CSF leak and intracranial injury (**Fig. 7**). Finally, type III fractures combine central and lateral frontobasilar fractures, often with comminution of the entire frontal bone, orbital roof, and lateral cranial vault (**Fig. 8**). Type II and III fractures are more frequently associated with concomitant midface injuries and are thought to be related to a higher-velocity impact from a lateral or inferior frontal or supraorbital vector. As one might suspect, because type III fractures are associated with the greatest force, they are most often associated with complications such as intracranial injury and CSF leak, reportedly in up to 25% of cases.[5]

One additional classification system, devised by Piccirilli and colleagues,[6] divides frontobasal

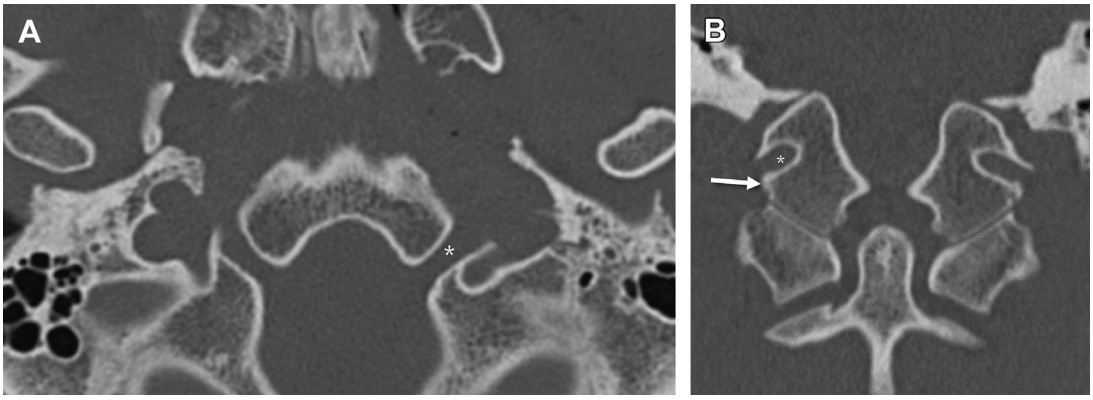

Fig. 5. Posterior cranial fossa. (*A, B*) Axial and coronal CT images showing the hypoglossal canal (*asterisk*) in relation to the occipital condyle (*arrow*).

fractures into types A to C, each denoting a different surgical approach for management. Type A fractures involve only the anterior table of the frontal sinus, whereas type B fractures involve the posterior table (Fig. 9). Type C fractures include any frontobasal injury that does not involve the frontal sinus. Fractures extending through the posterior table of the frontal sinus (type B), particularly when comminuted, in addition to potentially causing CSF leak, can ultimately result in the development of a mucocele, due to entrapped mucosa along the fracture line. Thus, these fractures may require a different surgical approach, including frontal sinus obliteration (removing the mucosa and filling the sinus with fat or other materials, such as hydroxyapatite) or cranialization (removal of the posterior table mucosa and bone, and performing any duraplasty needed at the time). Both of these surgeries should involve surgical obstruction of the outflow tracts.[7] Some investigators currently advocate cranialization if the fracture involves more than 25% of the posterior table of the frontal sinus.[8] Less displaced fractures can be managed conservatively, or via endoscopic approaches.[8,9]

Complications

The management of skull base fractures is based on anticipated potential complications. Although many facial fractures may require repair to maintain function and cosmesis, skull base fractures often require repair only if there is associated intracranial injury requiring decompression, persistent CSF leak, or significant cranial nerve or vascular injury. Also, extensive fractures through the posterior table of the frontal sinus may require surgical repair to prevent mucocele formation. Anterior skull base injuries are frequently seen in conjunction with frontal lobe contusions and intraorbital injuries, which are discussed in the article by

Table 2
Location of skull base fracture and frequently associated complications

Location	Complication
Anterior skull base	Intraorbital injury Sinonasal CSF leak/meningoencephalocele Anosmia (CN I injury)
Central skull base	Vascular injury (ICA occlusion, dissection, pseudoaneurysm, aneurysm, CCF) Cranial nerve injury (optic nerve, CN III, IV, V, and VI) Horner syndrome
Posterolateral skull base (temporal bone)	Vascular injury (ICA) CN VII or VIII injury Mastoid CSF leak/meningoencephalocele
Posterior skull base	Venous vascular injury or vertebrobasilar injury Lower cranial nerve injuries (CN IX, X, XII, or XII) Craniocervical junction and cervical spine injuries

Abbreviations: CCF, carotid cavernous fistula; CN, cranial nerve; CSF, cerebrospinal fluid; ICA, internal carotid artery.

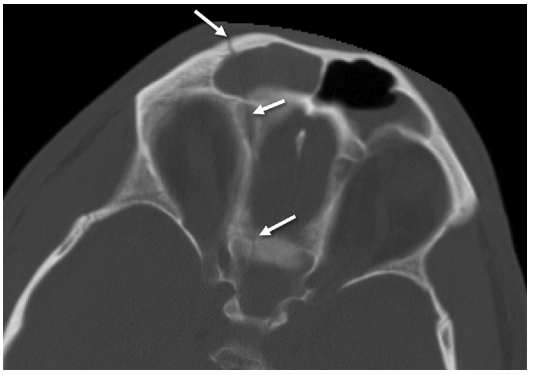

Fig. 6. Type I frontobasal fracture. Axial CT demonstrating a linear fracture through the anterior cranial fossa medially (*arrows*), traversing the anterior and posterior table of the frontal sinus, paralleling the cribriform plate, then extending through the planum sphenoidale.

Gupta and colleagues, elsewhere in this issue. The most commonly associated complications are CSF leak (with or without meningitis), and injury to the olfactory nerve, resulting in anosmia; these are unique to the anterior skull base.

CSF leak after trauma occurs when there is both an osseous defect and a tear of the closely adherent dura, leading to egress of CSF from the subarachnoid space into the sinonasal cavity (in the setting of frontobasal fractures) or into the middle ear cavity and mastoid air cells (in the setting of temporal bone trauma). CSF rhinorrhea or otorrhea is the usual clinical presentation. There are rare reports, in the setting of complex orbital roof injuries, of oculorrhea and intermittent periorbital swelling and tearing due to accumulation of CSF.[10] The more comminuted and displaced fractures, such as type III frontobasal fractures, carry

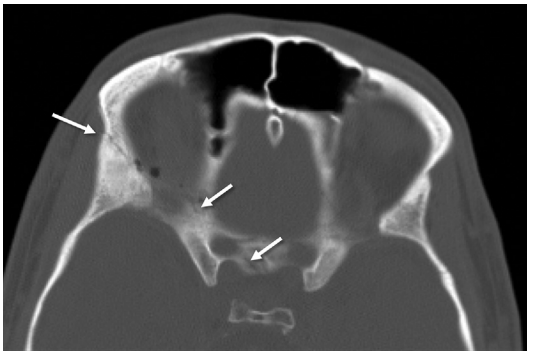

Fig. 7. Type II frontobasal fracture. Axial CT demonstrating a linear fracture through the lateral aspect of the anterior cranial fossa (*arrows*), traversing the orbital roof, and extending along the optic nerve canal to the tuberculum sella posteriorly.

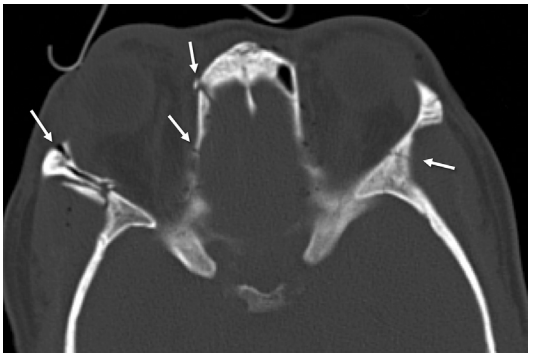

Fig. 8. Type III frontobasal fracture. Axial CT image demonstrating a comminuted and displaced transversely oriented anterior skull base fracture (*arrows*) extending across the orbital roofs and ethmoid roofs, with involvement of the lateral orbital walls. Given the comminution and medial and lateral anterior skull base involvement, this is a type III frontobasal fracture. Note pneumocephalus and orbital and soft tissue emphysema.

the greatest risk of CSF leak. Communication with the flora of the nasal or middle ear cavities results in meningitis, reportedly in up to 50% of cases, if the leak is not repaired.[11] The risk is approximately 1% in the first 24 hours, increasing to 18% at the end of 2 weeks (**Fig. 10**).[12,13] In fact, any patient presenting clinically with recurrent episodes of meningitis (particularly in the setting of prior trauma) should be evaluated for occult CSF fistula.[12]

Traumatic CSF leak, which is the most common form of CSF leak, occurs in 10% to 30% of skull base fractures, and most often presents as CSF rhinorrhea (in 80% of cases).[2,14,15] Eighty percent of cases will present in the first 48 hours after injury, and most (up to 95%) will present with CSF rhinorrhea in the first 3 months after trauma. The delay in symptom onset is primarily due to resolution of the initial posttraumatic edema and hemorrhage, combined with increased mobility as the patient progresses through rehabilitation. However, a small minority of patients (5%) will present in a more delayed fashion, months to even decades after their trauma, possibly due to fracture fragments slowly eroding and thinning the dura over time (**Fig. 11**).[16]

Most posttraumatic CSF leaks, up to 85%, are acute in presentation, and heal spontaneously with conservative management such as bed rest, head elevation, and stool softeners.[17–19] Occasionally patients will require CSF diversion with lumbar drain or external ventricular drain placement if the leaks do not resolve in approximately 2 to 7 days.[12,18] One recent study demonstrated good results in high-risk patients with the

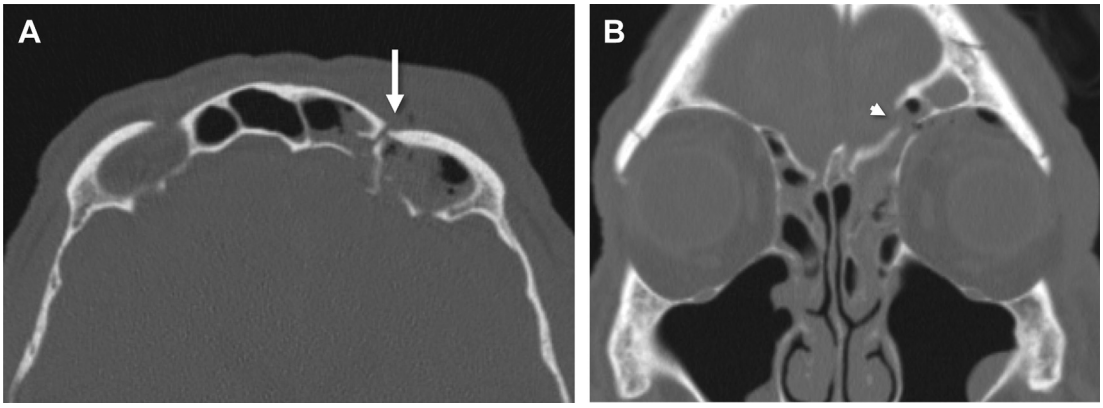

Fig. 9. Axial (*A*) and coronal (*B*) CT images of a comminuted and displaced frontobasal fracture with extension through both anterior (*arrow*) and posterior (*arrowhead*) tables of the frontal sinus and orbital roof, with intraorbital hematoma and emphysema.

administration of acetazolamide in the early posttraumatic period.[20] Of course patients presenting with acute CSF rhinorrhea with intracranial pathology requiring immediate surgical treatment, such as frontal lobe hematomas or depressed skull fractures, will undergo open intracranial repair of fractures and duraplasty at the time of the surgery. Those with persistent leaks for longer than 7 to 10 days, in spite of CSF diversion, often require repair, either intracranial or endoscopic. Larger skull base defects (>1.5 cm) or severely comminuted fractures, particularly if a meningoencephalocele is present, are associated with a worse prognosis for spontaneous resolution, and will require surgical repair.[12,21,22] The use of prophylactic antibiotics to avoid meningitis is a controversial topic. A recent large meta-analysis reviewing the outcomes of 1241 patients showed overall no statistically significant decrease in meningitis in patients treated with prophylactic antibiotics.[23] Similarly, a Cochrane review of 2168 patients in 2011 showed no evidence to support the use of prophylactic antibiotics, but recommended the need for large randomized controlled trials in the future.[14]

Loss of smell, or anosmia, as a result of olfactory nerve injury, is a relatively common complication

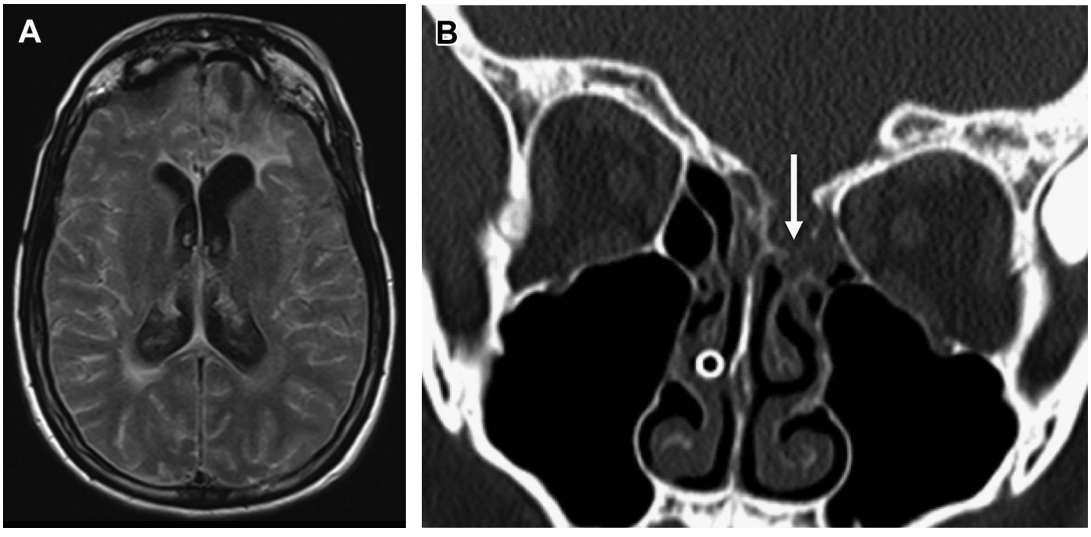

Fig. 10. A 32-year-old woman with history of motor vehicle accident 9 years previously, presented with CSF rhinorrhea, altered mental status and seizure. (*A*) Axial FLAIR image shows encephalomalacia in the anterior and inferior left frontal lobe, adjacent to the left frontal bone deformity from prior depressed skull fracture. Note nonsuppression of CSF signal in the sulci and ventricles compatible with meningitis and ventriculitis; there was growth of gram-positive cocci on lumbar puncture. (*B*) Coronal reformat CT shows a large left lateral lamella and ethmoid roof defect (*arrow*), with polypoid nondependent soft tissue adjacent to the dehiscence, compatible with a posttraumatic meningoencephalocele, the source of the CSF leak.

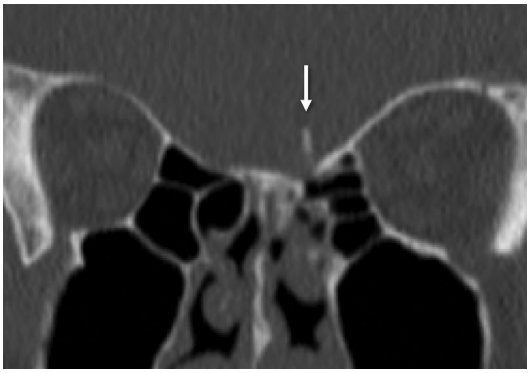

Fig. 11. A 73-year-old man with persistent rhinorrhea since trauma 6 months previously, positive for beta 2 transferrin. Coronal CT image demonstrates a focal defect within the left ethmoid roof with an adjacent vertically oriented fracture fragment (*arrow*), likely the source of dural tear.

after trauma, with an overall incidence of up to 7%, and an increased risk in anterior skull base fractures, particularly medial fractures along the cribriform plates. Traumatic CSF leak, particularly when repair is required, has been associated with an increased risk of anosmia as well. Only approximately 10% of all patients with traumatic anosmia are estimated to recover sense of smell, often in a delayed fashion, months to years after the injury.[24]

Central Skull Base Fractures

Classification/Etiology

Extension of frontobasal fractures from frontal impact can occur in a sagittal or oblique fashion through the central skull base with involvement of the sella and sphenoid sinus, and, in some cases, the temporal bone (**Fig. 12**). Transverse fractures in a coronal plane through the central skull base are common and are usually the result of high velocity lateral impact to the lateral frontal bone, zygoma, temporal, or parietal bone. Several transverse fracture patterns have been described, including transverse or oblique/diagonal patterns; these are related to the mechanism of injury. Transverse fractures extend through the sphenoid sinus, either anteriorly or posteriorly, depending on type of impact, and can propagate laterally through the greater sphenoid wing and squamosal temporal bones, or be associated with temporal bone fractures (**Fig. 13**). The more anterior

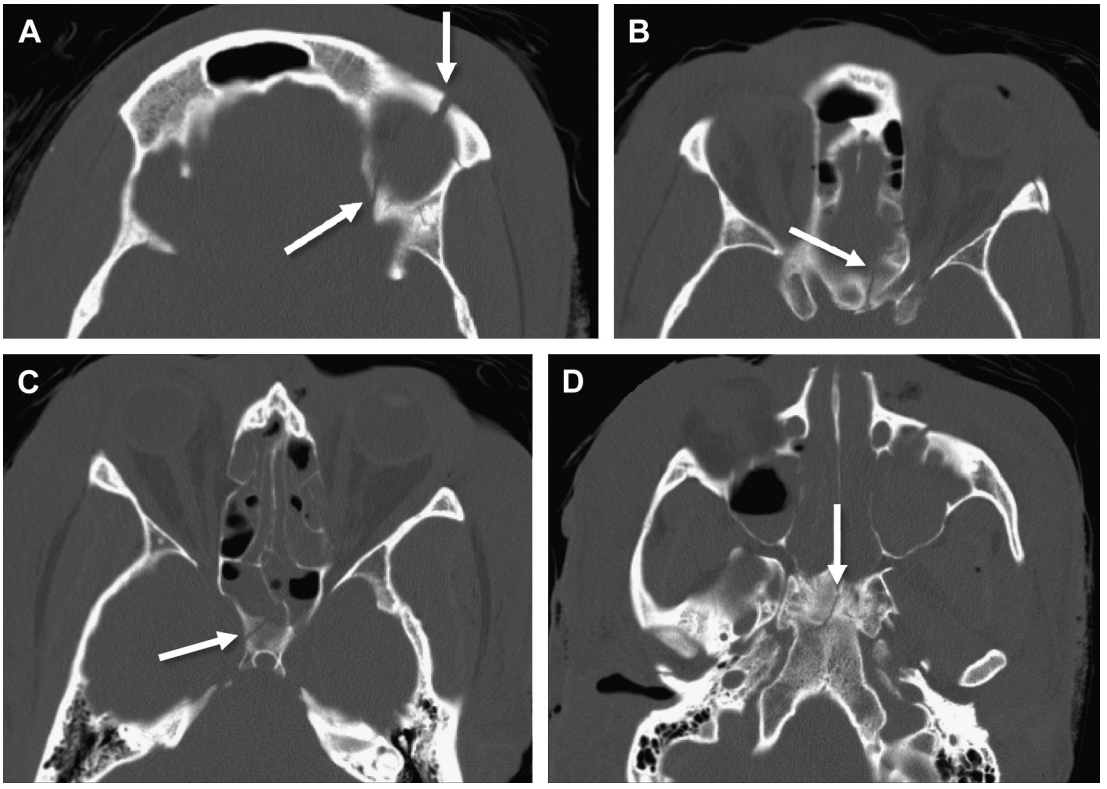

Fig. 12. Complex comminuted anterior and central skull base fractures. (*A–D*) Sagittal obliquely oriented complex comminuted left frontobasal fracture (*arrows*) extending along the supraorbital rim and orbital roof (*A*), along the left ethmoid roof and planum sphenoidale (*B*), through the sphenoid sinus walls (*C*), and into the basi sphenoid portion of the clivus (*C*, *D*).

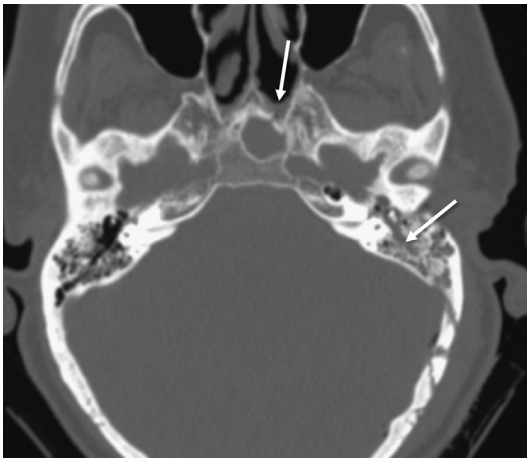

Fig. 13. Longitudinal left temporal bone fracture extends medially to involve the sphenoid sinus walls (*arrows*).

transverse fractures often involve the posterior aspect of the anterior cranial fossa (posterior ethmoid roof) and may extend through the clinoid processes, involving both the orbital apex and the superior orbital fissures and potentially resulting in cranial nerve or vascular injuries. The more posterior transverse patterns may involve the temporal bones bilaterally, extending along the sphenopetrosal synchondrosis, with involvement of the posterior sphenoid sinus and/or clivus (**Fig. 14**). Oblique central skull base fractures, often associated with facial fractures and type II or III frontobasal fractures, are more often associated with CSF leak. Transverse fractures are more commonly associated with vascular and cranial nerve injuries, although these can be seen in both types of central skull base fractures, depending on their course.[25,26]

Involvement of the clivus with central and posterior skull base fractures deserves special mention. These fractures are rare, accounting for about 2% of all cranial fractures, but are associated with high mortality (24%–80%) because of location and proximity to the brainstem, as well as high incidence of neurologic and vascular injury (up to 46%).[27] There are 2 characteristic patterns of clival injury: transverse or oblique, and longitudinal.[27,28] Transverse or oblique fractures, similar to other transverse central skull base fractures, are caused by a lateral or crushing injury, and are commonly associated with cranial nerve and internal carotid artery (ICA) injury (**Fig. 15**). Conversely, longitudinal clival fractures are more complex, are due to axial loading mechanism from the vertex, and are often associated with vertebrobasilar injury and brainstem infarction.[29] Longitudinal clival fractures are seen in conjunction with craniocervical junction injuries, and retroclival hematomas. A hallmark of clival fracture clinically is the presence of sixth nerve palsy, due to the location of the Dorello canal within the clivus.[30] Other cranial nerve injuries are also frequently seen.[27,28]

Complications

Central skull base fractures are primarily managed based on associated complications. Many are associated with intracranial injuries, including multicompartmental hemorrhage, temporal lobe parenchymal contusions, and diffuse axonal injury (DAI), as these injuries are associated with high-velocity impact (**Fig. 16**). Anterior middle cranial fossa epidural hematomas, also called benign

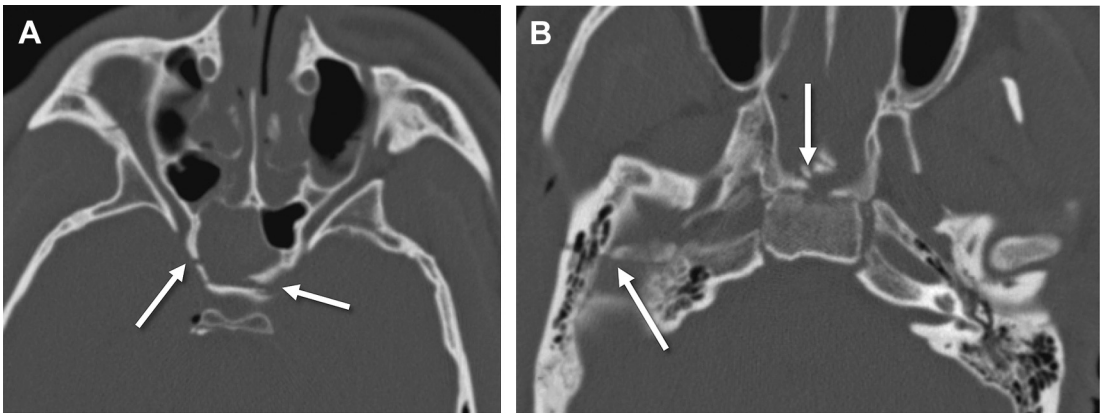

Fig. 14. Temporal bone fractures are often associated with central skull base fractures. When a temporal bone fracture is detected, careful assessment of the sphenoid sinus walls and clivus is important. Note transversely oriented fracture through the central skull base (*arrows*) extending through the sphenoid sinus walls (*A*), and extending inferiorly in the coronal plane to involve the basi sphenoid portion of the clivus and right temporal bone (*B*).

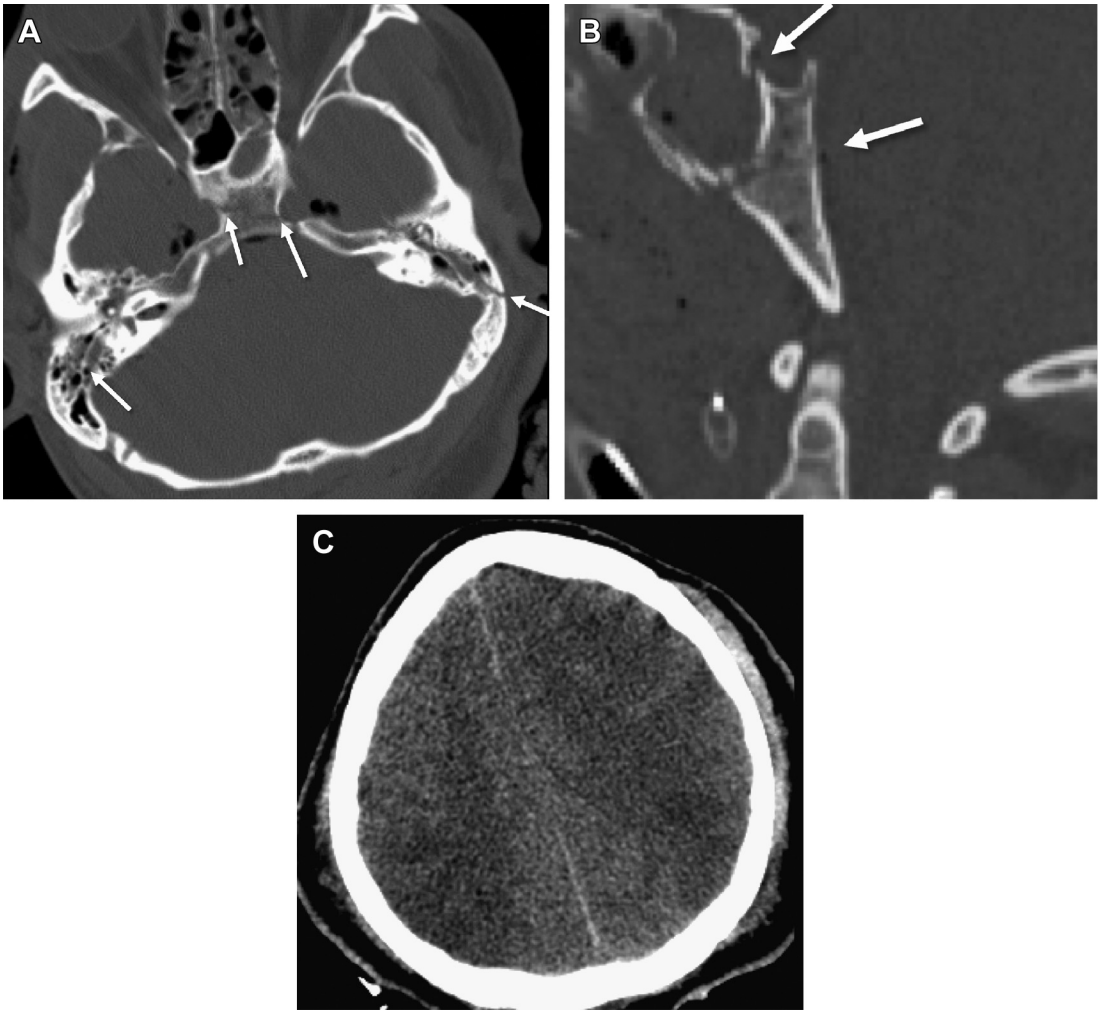

Fig. 15. Complex transversely oriented central skull base fracture (*arrows*) (*A*) extending through bilateral temporal bones, and transversely oriented through the clivus (*A*, *B*) and sella (*B*). The fracture extended through the carotid canals with pneumocephalus (*A*); bilateral ICA territory infarcts resulted (*C*). The patient died soon after this CT was performed.

venous epidural hematomas, occur in conjunction with injury to the greater wing of the sphenoid bone. These are postulated to be a result of venous injury to the sphenoparietal sinus, and have a benign self-limited course, not requiring surgical drainage (**Fig. 17**).[31] CSF leaks caused by fracture extension through the sphenoid sinus occur less frequently than in the frontobasal region, but are common with comminuted fractures.[32]

The most frequently encountered complications associated with central skull base fractures are vascular complications. Blunt cerebrovascular injury is reported in approximately 1% of all head and facial traumas, with risk increased to 8.5% in the setting of basilar skull fractures.[33–35] Fractures extending through the central skull base are particularly at risk for ICA injury, due to this vessel's course through the central skull base and cavernous sinus. However the basilar artery is also vulnerable because of its proximity to the clivus. Overall, the fractures that are most frequently associated with vascular injury are clival fractures, fractures through the sella/sphenoid sinuses, orbital apex fractures, petrous ridge fractures, and occiput and occipital condyle fractures.[35] Fractures involving the carotid canal have an increased probability of neurovascular injury, with a positive predictive value of approximately 35% in one series (**Fig. 18**).[36]

The spectrum of vascular complications includes frank transection/laceration, dissection,

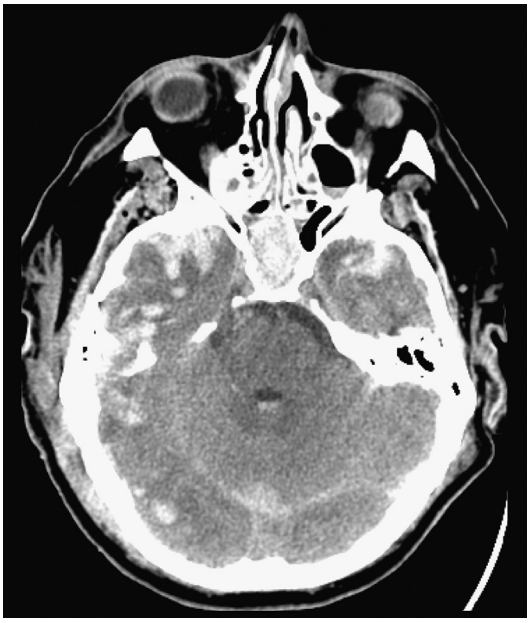

Fig. 16. Common intracranial findings associated with central skull base fractures are temporal lobe contusions, extra-axial hemorrhage, intraventricular hemorrhage, and blood within the sphenoid sinus.

aneurysm/pseudoaneurysm, incarceration (entrapment in a fracture line) or dural arteriovenous (AV) fistula (most commonly carotid cavernous fistula). Stroke from thromboembolic disease due to arterial dissection is reported in 31% in the setting of blunt carotid artery injury and 14% with blunt vertebral artery injury. This same study showed that stroke rates decreased to 6.8% and 2.8%, respectively, for patients who received anticoagulation.[37] Therefore, early detection and treatment with anticoagulation has been the mainstay of traumatic dissection management, and screening high-risk patients with vascular imaging (most often using computed tomography angiography [CTA] or magnetic resonance angiography [MRA]) is routine.

Although cerebrovascular injury in the setting of trauma is discussed in the article by Gentry and colleagues elsewhere in this issue, the carotid cavernous fistula (CCF) deserves special mention, as it is a rare but unique complication associated with transverse or oblique central skull base fractures, occurring in 3.8% of all basilar skull fractures in one series.[38] CCF presents as a traumatic direct connection between the ICA and the cavernous sinus, forming a high-flow fistula. Clinical presentation is with exophthalmos, bruit, chemosis, vision loss, and ophthalmoplegia, with potential resulting blindness, stroke, or death. Symptoms of CCF may present acutely, within hours of the injury, or in a delayed fashion, weeks to months after the injury. Clinical detection may be difficult in comatose or intubated patients, and a high index of suspicion should be present in patients with middle cranial fossa fractures. CCFs are treated most frequently with endovascular therapies using detachable balloon coil or other embolic materials, or with covered stent placement in an attempt to preserve ICA flow.[39]

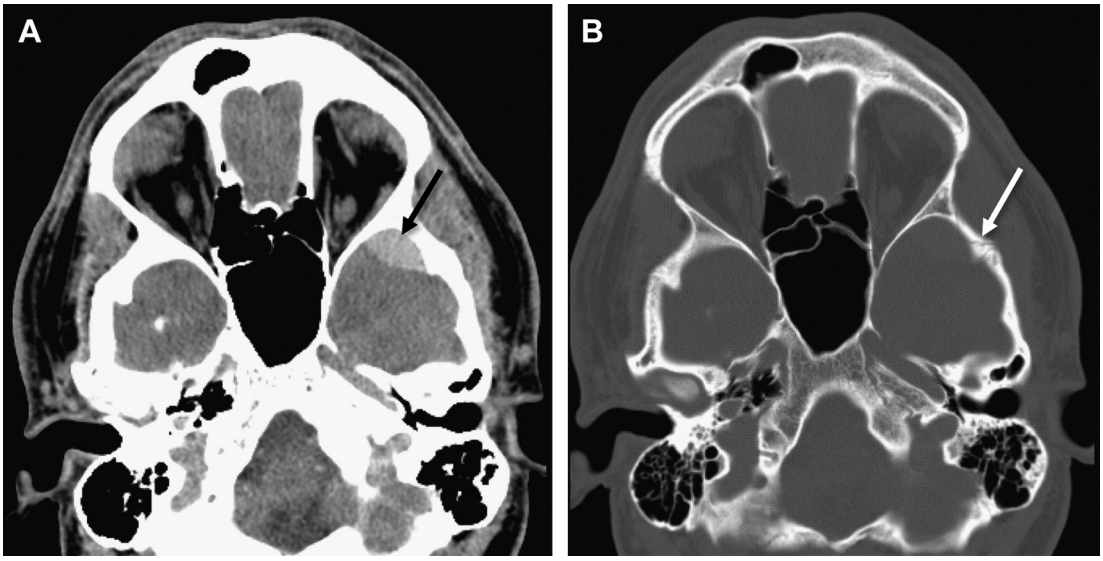

Fig. 17. (*A*) Epidural hematoma at anterior left middle cranial fossa (*black arrow*), adjacent to fracture line on axial CT (*arrow* on *B*). This most likely represents a venous epidural hematoma related to sphenoparietal sinus injury. These often have a benign, self-limited course, not requiring surgical drainage.

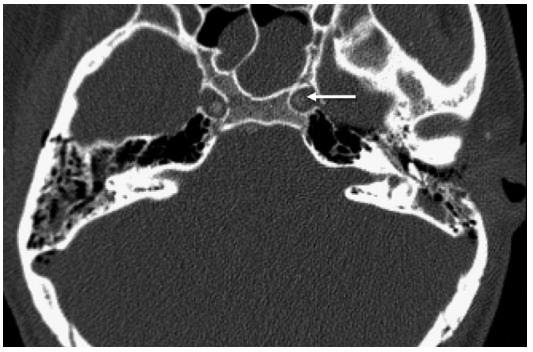

Fig. 18. Axial CTA demonstrating left temporal bone fracture extending through the left carotid canal. Note left ICA irregularity and narrowing (*arrow*), consistent with dissection.

Cranial nerve deficits may present in an acute fashion, often due to cranial nerve laceration, or in a delayed fashion, possibly because of neuronal stretching or edema. More delayed presentation injuries are associated with a better prognosis for recovery of symptoms. Fractures extending through the clinoid process and the superior orbital fissure may present with a syndrome of third, fourth, and sixth nerve palsies, as well as anesthesia in V1 distribution. Decreasing visual acuity owing to compression of the optic nerve is the hallmark of the orbital apex syndrome. Both of these clinical syndromes may necessitate surgical decompression to preserve cranial nerve function, particularly if the fractures are comminuted and impinging on the optic nerve, or if there is a hematoma contributing to a compressive optic neuropathy (Fig. 19).[40] Fractures through the sella may present with optic nerve compression and bitemporal hemianopsia. Involvement of the cavernous sinus or clivus can cause CN III, IV, V1, V2, or VI neuropathies. Partial Horner syndrome (ptosis, miosis, without anhidrosis) may develop in the posttraumatic setting because of interruption of the postganglionic sympathetic fibers traveling along the ICA plexus. Although this most often reflects dissection in the posttraumatic setting, it also may result from fracture extending through the petrous portion of the temporal bone, along the carotid canal, or in the region of the Meckel cave.[41] Vascular imaging should be performed in the evaluation of posttraumatic Horner syndrome.

Posterior Skull Base Fractures

Classification/Etiology

Most posterior cranial fossa fractures occur as a result of a lateral and/or posterior blow to the occiput, and involve the occipital bone with frequent extension to the petrous temporal bone. No simple classification scheme exists for the description of all posterior skull base fractures.

Temporal bone fractures are often treated independently, and described as either transverse

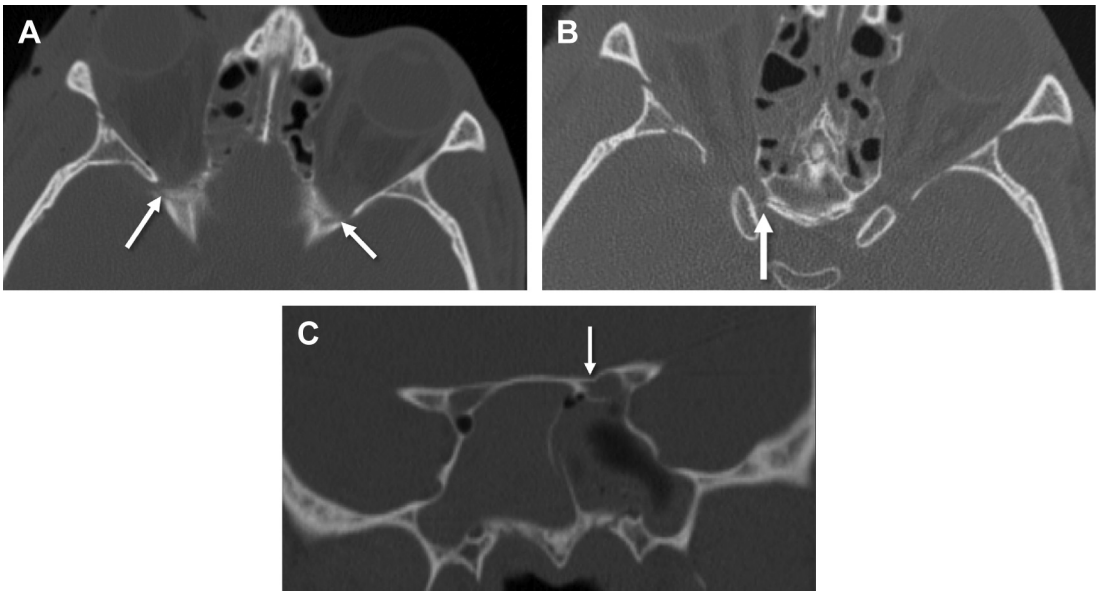

Fig. 19. Visual loss is an important complication of skull base fracture. (*A*) Axial CT showing a comminuted fracture extending through the base of the clinoid processes bilaterally, involving the superior orbital fissures (*arrows*). (*B*) Axial CT of another patient demonstrating fracture line traversing the right optic nerve canal (*arrow*). (*C*) Coronal CT of another patient demonstrating fracture extending through the left optic nerve canal (*arrow*).

or longitudinal, or otic capsule sparing or violating.[42,43] These are described in the article by Kennedy and colleagues elsewhere in this issue. Fractures involving the clivus (basi occiput) inferiorly have been discussed previously. Occipital condyle fractures are rare, and are classified by the imaging appearance and presumed mechanism as either type 1 (impacted condyle with comminution, due to axial loading), type 2 (basilar skull fracture with linear extension into the occipital condyle), or type 3 (avulsion fracture at the attachment site for the alar ligaments) (**Fig. 20**).[44]

Complications

Posterior skull base fractures are frequently associated with posterior fossa intracranial injuries, including extradural, subdural, and cerebellar parenchymal hematomas. Posterior fossa epidural hematomas are the most frequently encountered complication associated with posterior skull base fractures, and are often venous due to injury to the transverse or sigmoid dural venous sinuses or the jugular bulb. These are more frequently seen in children and can expand rapidly, leading to sudden clinical deterioration from fourth ventricular or brainstem compression. Early diagnosis and management is imperative (**Fig. 21**). Small hematomas without mass effect may be managed conservatively with clinical and radiologic follow-up, whereas larger hematomas with mass effect often require decompression.[45–47]

Posterior skull base fractures through the venous sinuses or jugular foramen causing venous sinus injury can also predispose to venous sinus thrombosis or the long-term complication of dural AV fistula. Venous sinus thrombosis can present with worsening intracranial hypertension, venous infarctions, or hemorrhage.[48] Follow-up studies demonstrating increased density in the expected location of the venous sinuses should prompt evaluation with CT venography (**Fig. 22**).

Lower cranial nerve injuries may occur in posterior skull base fractures, again depending on the location and pattern of injury. Temporal bone fractures may be associated with both CN VII and VIII injuries. Facial paralysis, along with CSF leak, is one of the most common indications for surgical intervention in temporal bone fractures. Transverse fractures through the temporal bone are more commonly associated with facial paresis than longitudinal fractures, with injury typically at the level of the internal auditory canal or labyrinthine segments. When facial nerve injury occurs in the setting of longitudinal fractures, it is most commonly at the level of the geniculate ganglion (**Fig. 23**).[40,42,43] Fractures extending through the jugular foramen, in addition to causing vascular injury or thrombosis, also predispose to glossopharyngeal, vagal, or spinal accessory nerve injuries, resulting in dysphagia with loss of the gag reflex, ipsilateral vocal cord paralysis with hoarseness, and paralysis of the ipsilateral sternocleidomastoid and trapezius musculature (**Fig. 24**). Fracture through the hypoglossal canal, as can be seen with adjacent occipital condyle injuries, can predispose to ipsilateral tongue deviation and hemiatrophy (**Fig. 25**).[40,49,50]

As with anterior cranial fossa fractures, comminuted fractures through the temporal bones may be complicated by CSF leaks with or without associated meningoencephaloceles, particularly if the fractures are displaced through the tegmen tympani or mastoideum, or along the posterior petrous ridge. These fractures may result in CSF

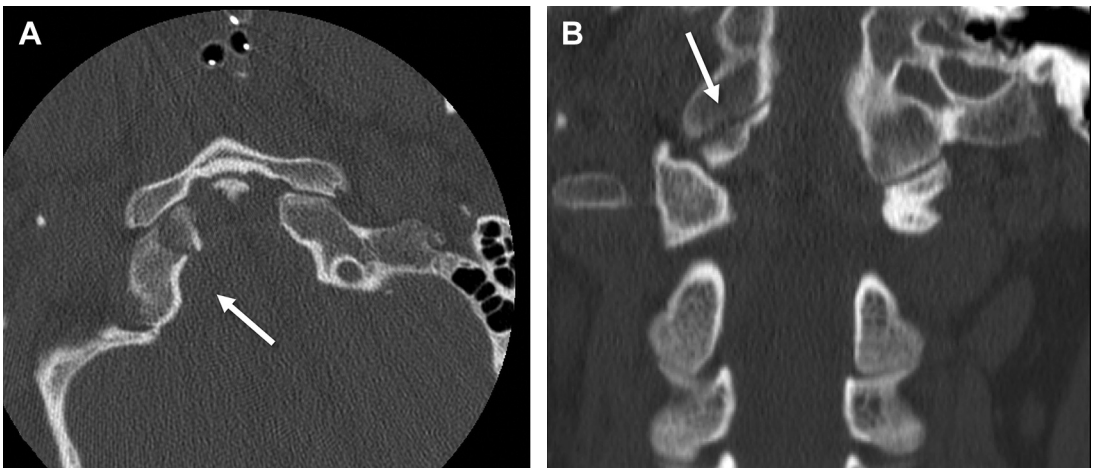

Fig. 20. Axial (*A*) and Coronal (*B*) CT images demonstrating a mildly diastatic type III avulsion fracture of the right occipital condyle (*arrows*).

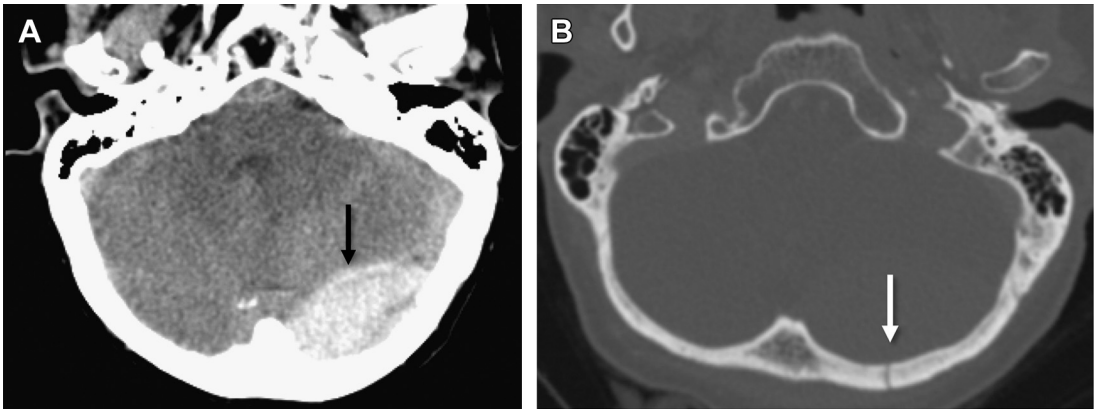

Fig. 21. (*A*) Axial soft tissue window through the posterior fossa demonstrating a large epidural hematoma (*black arrow*), causing mass effect on the fourth ventricle. (*B*) Bone window CT image shows the underlying occipital bone fracture (*arrow*).

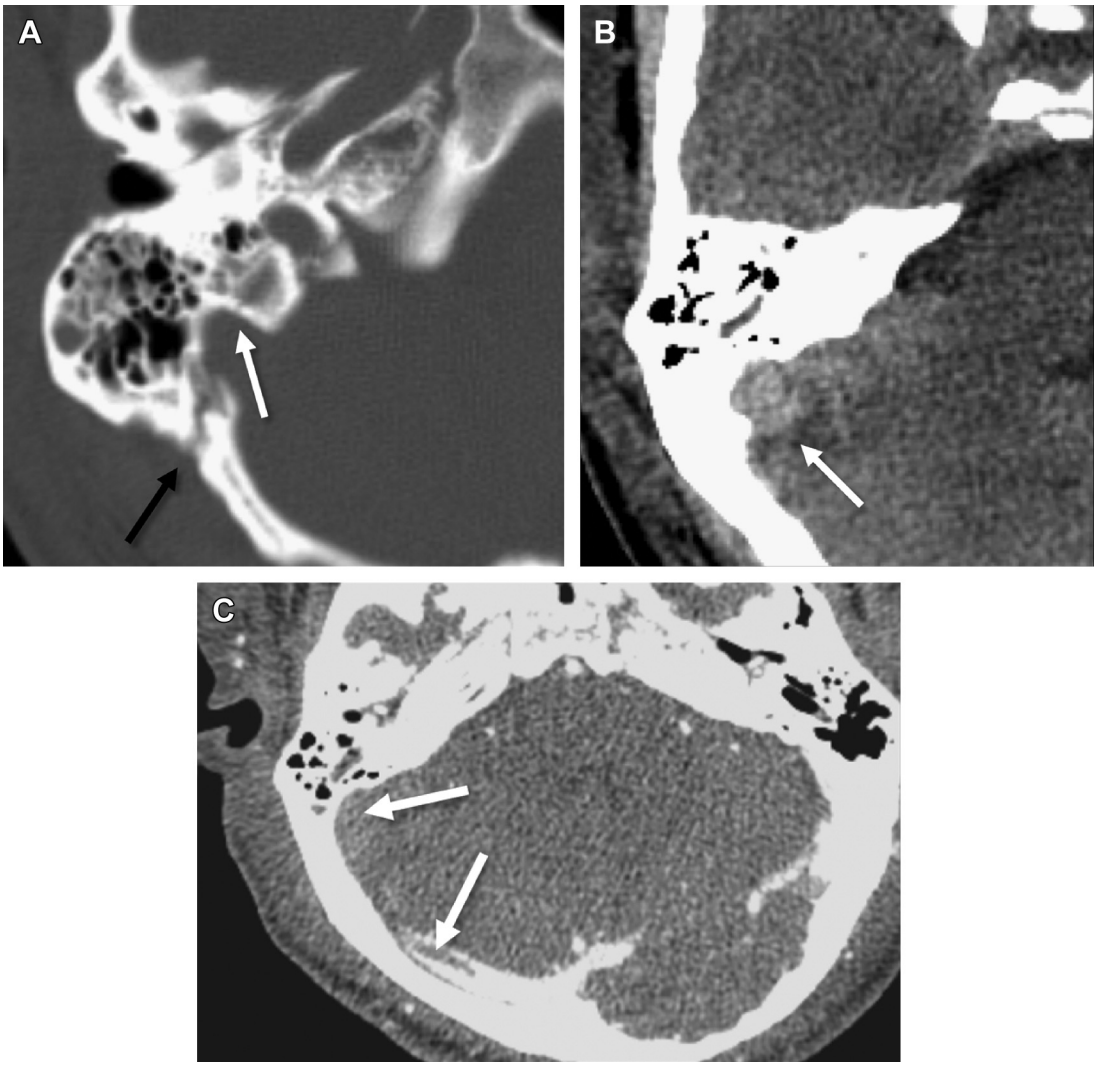

Fig. 22. (*A*) Axial CT image through the right temporal bone and posterior fossa showing diastasis of the right occipitomastoid suture (*black arrow*), with subtle fracture extending medially through the sigmoid sinus (*arrow*). (*B*) Noncontrast head CT shows increased density in the expected location of the sigmoid sinus (*arrow*). (*C*) CTA/CTV image demonstrates a clot in the right transverse and sigmoid sinus (*arrows*).

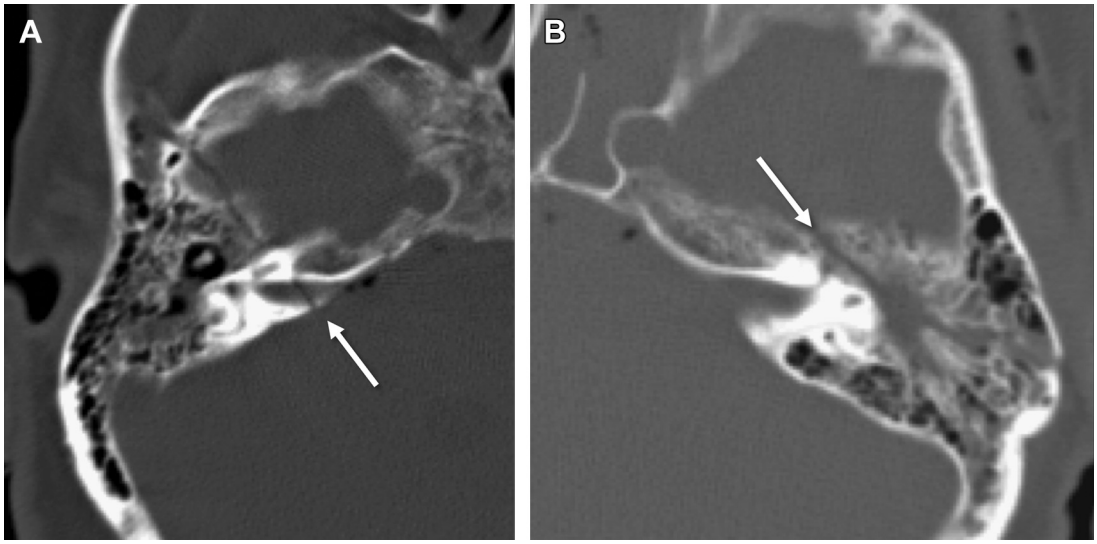

Fig. 23. Facial nerve palsy and hearing loss are complications of skull base fractures. (*A*) Medial transverse fracture (with respect to the petrous ridge) through the right temporal bone, extending through the internal auditory canal (*arrow*). (*B*) Longitudinal fracture through the left temporal bone extending through the expected location of the geniculate ganglion (*arrow*).

otorrhea or rhinorrhea (via extension through the Eustachian tube), as well as traumatic meningocele, and predispose the patient to meningitis (**Fig. 26**). As in anterior skull base trauma, presentation may be acute or delayed.

Finally, basi occiput clival and occipital condylar fractures extending through the craniocervical junction often present with vertebrobasilar injury as well as cervical spine injuries, with varying degrees of ligamentous injury and possibility for craniocervical dissociation (**Fig. 27**). Therefore, fractures in this region should prompt vascular imaging with CTA or MRA, as well as magnetic resonance imaging (MRI) of the cervical spine to assess for ligamentous injuries.[3,29,35]

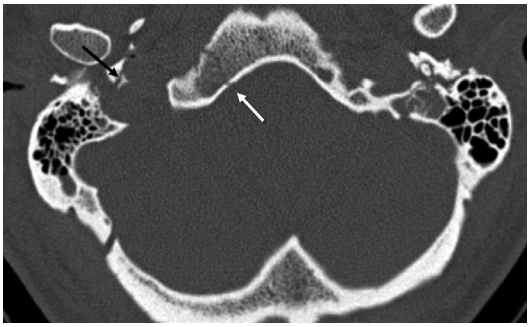

Fig. 24. Complex right posterior skull base fracture extending through the right occipital bone, along the right jugular foramen (pars vascularis) and carotid canal (*black arrow*). There is also a longitudinal fracture of the basi occiput of the clivus (*arrow*).

DIAGNOSTIC CRITERIA

In addition to more general indicators of craniofacial injury, such as low Glasgow coma scale scores, clinical findings associated with skull base fracture include periorbital ecchymosis, Battle sign (postauricular ecchymosis), hemotympanum, and CSF otorrhea or rhinnorhea.[51,52] Acute cranial nerve deficits in the setting of head trauma should raise the suspicion for skull base fracture.[53] The imaging diagnosis is made by detecting the fracture line on CT as a noncorticated, noninterdigitating lucency in skull base bone, with or without adjacent pneumocephalus, sinus or middle ear/mastoid opacification, or intraorbital emphysema.

CSF leak is most often diagnosed clinically by CSF rhinorrhea or otorrhea, clear drainage that increases with Valsalva or positional maneuvers. The gold standard confirmatory test for the presence of CSF in the rhinorrhea or otorrhea is the beta 2 transferrin assay, or the newer beta trace protein assay. These proteins are specific to CSF, and the assays confer a very high sensitivity and specificity for CSF leak, with rare false positives in the setting of liver failure. Only a few drops are required to make the diagnosis.[54,55]

DIFFERENTIAL DIAGNOSIS

Skull base fractures can often be difficult to detect on CT, particularly if linear and noncomminuted, and a thorough knowledge of skull base anatomy is necessary to avoid diagnosing

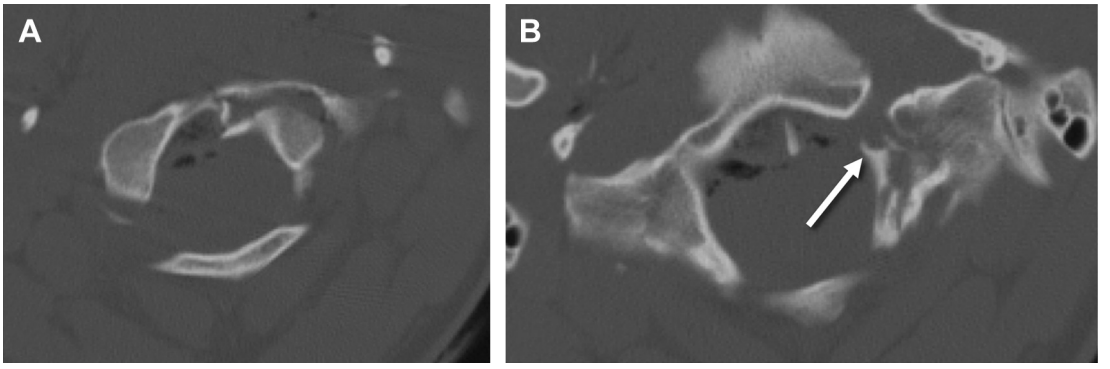

Fig. 25. This patient is at risk for both hypoglossal nerve injury and vertebral artery dissection. (*A*) Comminuted fracture of the left occipital condyle. (*B*) Note fracture fragment (*arrow*) extending into the left hypoglossal canal.

"pseudofractures." Many small neural and vascular channels and foramina that can be seen on multidetector CT (MDCT) can mimic fracture lines, as can sutures and skull base fissures. Additionally, suture lines also can become diastatic and widened after skull base injury.

In the anterior skull base, the anterior and posterior ethmoid artery canals may be confused with skull base fractures, and one should note the corticated margins and tapering nature (see **Fig. 2**B). The supraorbital canal is a characteristic notch along the medial third of the supraorbital rim, which transmits the supraorbital artery and nerve. The sphenofrontal suture as it courses along the superolateral orbit can mimic a fracture line, and one should note the interdigitating and corticated lucency and characteristic location (**Fig. 28**). Additionally, the metopic suture located along the frontal bone in the midline can persist in up to 10% of adults as a lucency between the frontal sinuses along the interfrontal sinus septum.[56]

Within the central skull base, the vidian canal, as well as minor neurovascular channels associated with the foramen rotundum and foramen ovale, can simulate fractures. Specifically, the palatovaginal canal, a canal inferomedial to the vidian canal extending from the pterygopalatine fossa to the roof of the pharynx that carries the pterygovaginal artery (branch of the maxillary artery) and the pharyngeal nerve, can simulate a fracture on axial images (**Fig. 29**). Several canals and channels can persist within the clivus and mimic fractures.

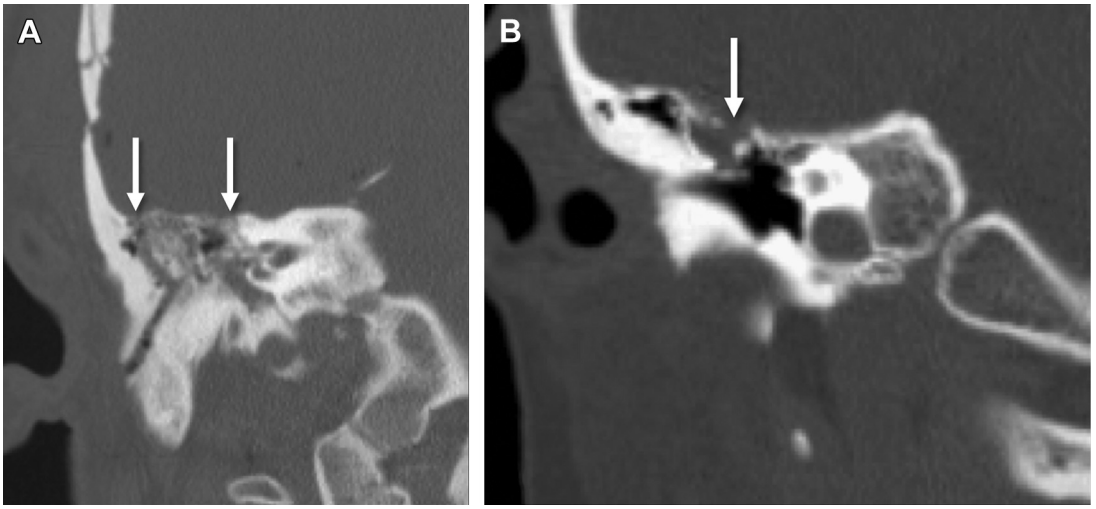

Fig. 26. Acute and chronic tegmen defects, with risk of CSF leak. (*A*) Coronal CT shows displaced and comminuted right temporal bone fractures (*arrows*) extending through the tegmen with scattered pneumocephalus. (*B*) Coronal CT of a patient with a history of remote trauma demonstrates a defect along the tegmen (*arrow*), and adjacent meningoencephalocele.

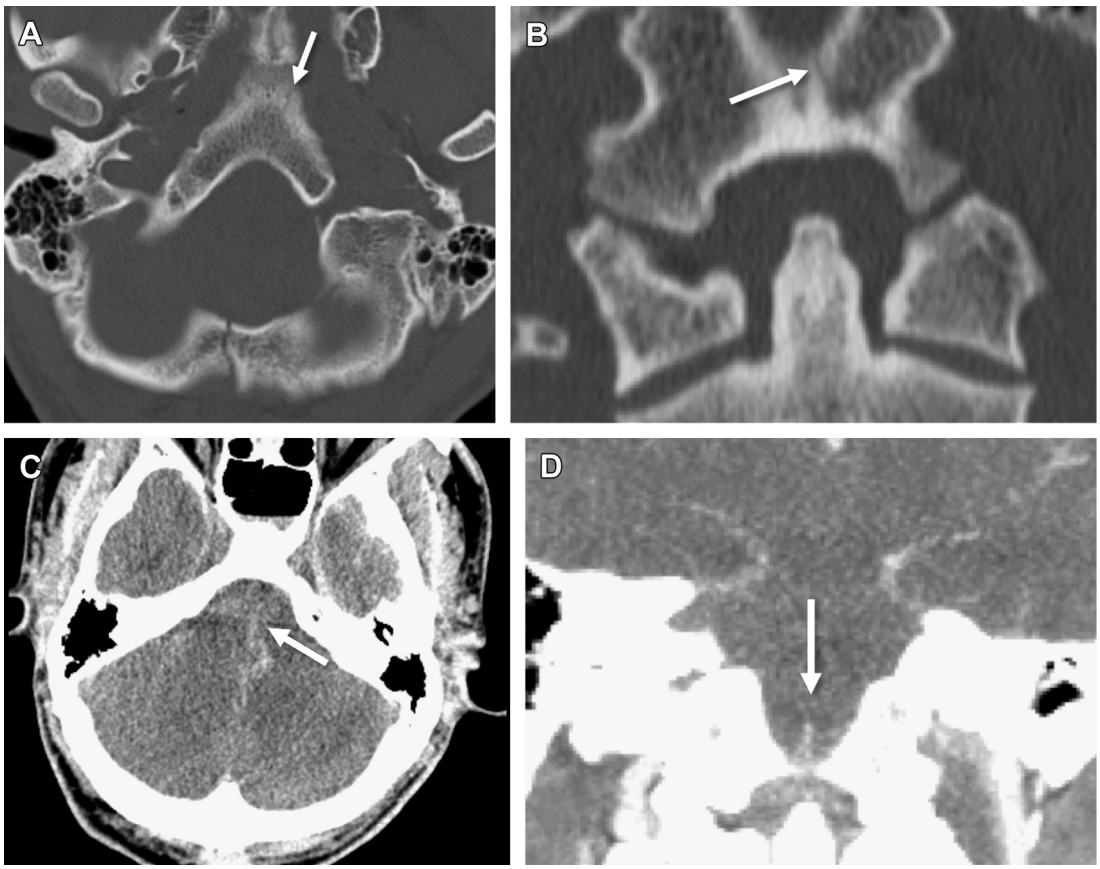

Fig. 27. CTA should be considered to assess for vascular injury in the setting of clivus fractures. Axial CT through the posterior fossa (*A*) showing a right paramedian occipital bone fracture extending through the foramen magnum, with a linear nondisplaced fracture extending along the left paramedian basi occiput portion of the clivus (*arrow*), in a longitudinal orientation (*A*, *B*). In spite of the relatively nondisplaced appearance of the longitudinal clivus fracture, there were devastating complications with brainstem hemorrhage (*arrow*) (*C*), as well as basilar artery occlusion (*arrow*) (*D*). The patient did not survive the injury.

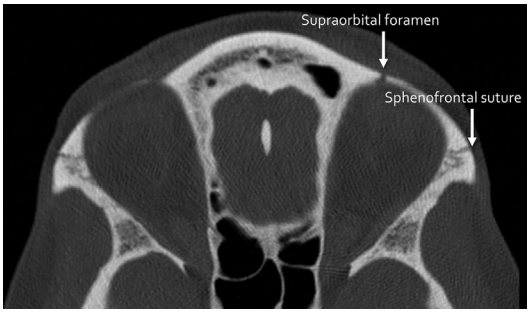

Fig. 28. Anterior cranial fossa pseudofractures, including the supraorbital foramen and the interdigitating sphenofrontal suture line characteristically located along the superolateral orbital rim.

Within the superior basi sphenoid clivus, the craniopharyngeal or persistent hypophyseal canal can be rarely seen in the adult as a linear longitudinally oriented canal coursing anteriorly and inferiorly from the sella toward the nasopharynx. More inferiorly within the midline of the basi occiput of the clivus, an anatomic variant, the medial basal canal (or canalis basilaris medianus) may exist, likely a notochordal remnant. The sphenooccipital synchondrosis, or transverse clival cleft, separates the basi sphenoid from the basi occiput and usually closes in teenage years, but can persist into the 20s, mimicking a transverse clival fracture. Finally, other sutures and fissures that can mimic fracture lines include the petro occipital fissure, which transmits the inferior petrosal sinus, as well as the sphenosquamosal suture (lateral to the foramen spinosum) and the sphenopetrosal synchondrosis (between the foramen ovale and the carotid canal) (**Fig. 30**).[56]

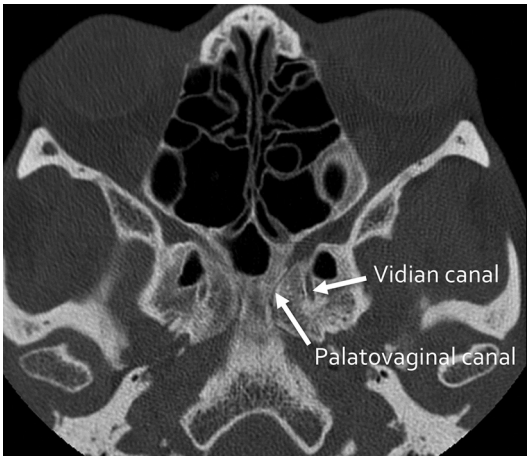

Fig. 29. One of the many central skull base pseudofractures, the palatovaginal canal, an inferior sphenoid bone canal extending to the pterygopalatine fossa and located inferomedial to the well-corticated vidian canal.

Within the posterior skull base, there are a number of emissary venous canals in the mastoid and occipital bones extending to the posterior condylar region that may be asymmetric and mimic fracture lines. Additionally, the occipitomastoid suture, as it extends to the jugular foramen, is often asymmetric and may have few interdigitations, mimicking a fracture. This suture also may become diastatic and widened in the setting of posterior skull base fracture (Fig. 31). There are many temporal bone pseudofractures, discussed in detail in the article by Kennedy and colleagues on temporal bone trauma elsewhere in this issue. These include the tympanomastoid suture posteriorly, as well as the tympanosquamous suture,

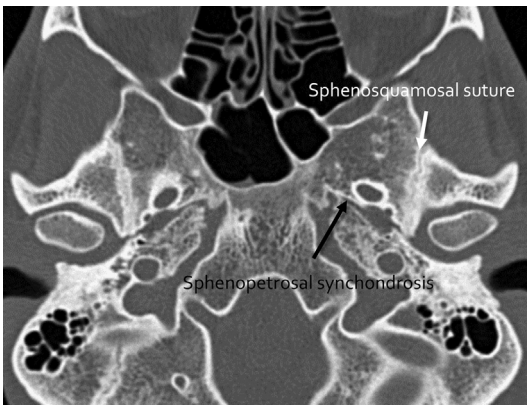

Fig. 30. Axial CT showing the corticated and interdigitating sphenosquamosal suture (*arrow*) lateral to the foramen spinosum, and the sphenopetrosal synchondrosis between foramen ovale and the carotid canal.

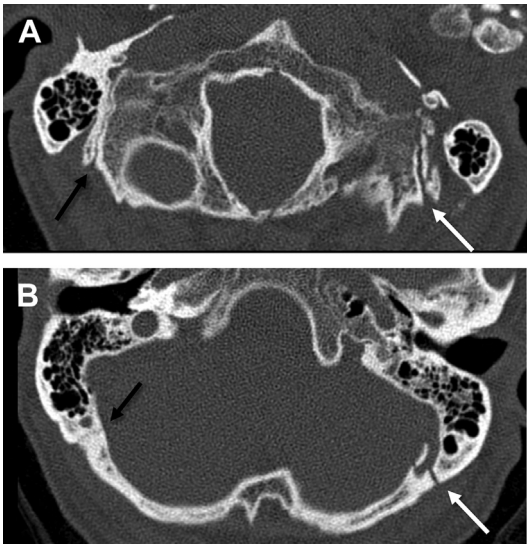

Fig. 31. (*A, B*) Axial CT showing posttraumatic diastasis of the left occipitomastoid suture line (*arrow*), compared with the right, which is normal (*black arrow*).

which lies anterior to the external auditory canal and continues medially as the petrotympanic suture (Fig. 32). Additionally, the petrosquamous suture, as well as a multitude of neurovascular channels, including the singular and subarcuate canals, also may mimic fractures.[56,57]

IMAGING FINDINGS

The first imaging modality necessary in the patient with suspected skull base trauma is a noncontrast head CT, as patients will be initially managed and triaged based on intracranial injuries. Pneumocephalus should raise suspicion for a fracture through the anterior skull base or the temporal bone. Additionally, significant or serially increasing pneumocephalus suggests a dural tear and possible CSF leak, although pneumocephalus has been reported in some cases of cranial extension of extensive subcutaneous emphysema in the absence of skull base fracture.[58]

Face CT, with multidetector thin-slice CT images through the face and skull base, also should be obtained in any patient with a suspected skull base fracture. Soft tissue windows are assessed for intraorbital emphysema, extraocular muscle injury, retrobulbar or subperiosteal hemorrhage, globe rupture, intraocular hemorrhage or lens dislocation, as well as for facial soft tissue injuries and paranasal sinus hemorrhage.

Thin-section bone algorithm images should be scrutinized and reconstructed in the coronal and sagittal planes, as multiplanar reconstruction has

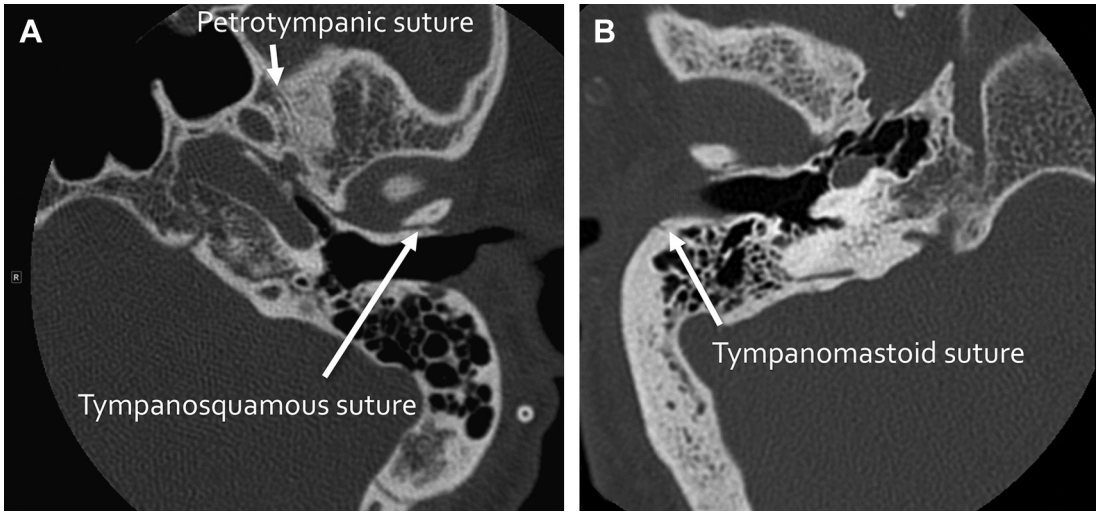

Fig. 32. Temporal bone CT images showing pseudofractures, including the tympanosquamous suture that continues medially as the petrotympanic suture (*A*), and the tympanomastoid suture (*B*).

been shown to improve fracture detection.[59] Manipulating the data on a 3-dimensional (3D) workstation and optimizing the window and level settings is often helpful. Additionally, 3D volumetric reconstruction can be useful to detect the fractures, assess any facial deformity, and to aid in surgical planning for reconstruction.[60] A skull base fracture is a noncorticated, noninterdigitating lucency through the skull base, often seen with pneumocephalus or intraorbital emphysema, and opacification and air-fluid levels in adjacent sinuses or mastoids. Displacement of bone fragments should be noted, as these can be associated with dural tear. Complex facial fractures often are associated with highly comminuted anterior cranial fossa fractures (type III frontobasal fractures). Fractures along the anterior cranial fossa, particularly along the cribriform plates and ethmoid roofs, as well as along the carotid canals, may be subtle.

Fractures through the carotid canal, particularly when there is intracanalicular air, or those extending through the clivus, should prompt evaluation with CT or MRA. Studies with 16-slice multidetector CTA have shown up to 92% negative predictive value, suggesting that CTA can replace the gold standard digital subtraction angiography (DSA) for screening of suspected blunt carotid and vertebral artery injuries.[61] Additionally, significantly enlarged superior ophthalmic veins, or asymmetric fullness or air within the cavernous sinus, should prompt vascular evaluation with CTA or DSA to assess for carotid-cavernous fistula. Any suspicion of thromboembolic disease, such as clinical or imaging findings of stroke, should prompt vascular imaging. Finally, increased density in the expected location of venous sinuses may represent extra-axial hemorrhage or even venous sinus thrombosis, and should prompt evaluation with CT venography (CTV).

One of the most obvious imaging findings of blunt carotid and vertebral artery injury on CTA or CTV is vascular occlusion (due to occluding dissection or thromboembolic disease) with lack of vessel opacification. However, dissection or pseudoaneurysm also can be more subtle, manifesting as vessel wall irregularity, luminal narrowing, or focal tapering of the affected vessel (**Fig. 33**).[62] CCF findings include prominent early filling and enlargement of the affected cavernous sinus, with ipsilateral prominent superior ophthalmic veins, as well as proptosis and intraorbital fat stranding. Engorged inferior petrosal sinus and other draining veins also may be seen. DSA shows fistulous communication between the

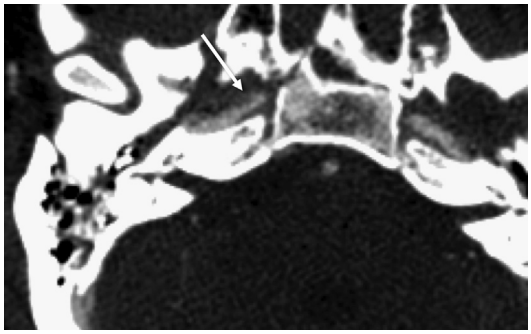

Fig. 33. CTA demonstrating right temporal bone fracture causing luminal irregularity and narrowing of the horizontal petrous segment of the right internal carotid artery (*arrow*), compatible with dissection.

carotid artery and the cavernous sinus (**Fig. 34**).[38] Traumatic true aneurysms, involving all of the layers of the arterial wall, also may be seen as focal outpouchings of the affected vessel, most often involving the extradural cavernous segments of the internal carotid artery in the setting of skull base fractures. Rupture of such a cavernous aneurysm may similarly result in carotid cavernous fistula formation. Other potential sites of true aneurysms include the distal cortical vessels, including the distal anterior cerebral artery pericallosal branches due to shear along the falx (**Fig. 35**).[63,64] Although CTA remains an adequate screening tool for vascular injury, DSA remains the gold standard for further evaluation of any suspected injuries on CTA.

MRI findings in the setting of trauma include hemorrhage and parenchymal contusion on T1-weighted, T2-weighted, gradient echo (GRE) and fluid-attenuated inversion recovery (FLAIR) images. Diffusion-weighted and GRE images can be helpful in the detection of infarction and punctate shear injury (DAI). Loss of flow voids in the vascular structures, or crescentic increased T1 and T2 signal around the periphery of the carotid or vertebral arteries, may herald vascular occlusion or dissection, respectively. Time of flight and contrast-enhanced MRA imaging findings of carotid occlusion or dissection, as well as CCF, are similar to those with CTA. The technique of dynamic contrast-enhanced MRA may be helpful in the noninvasive workup of possible CCF,

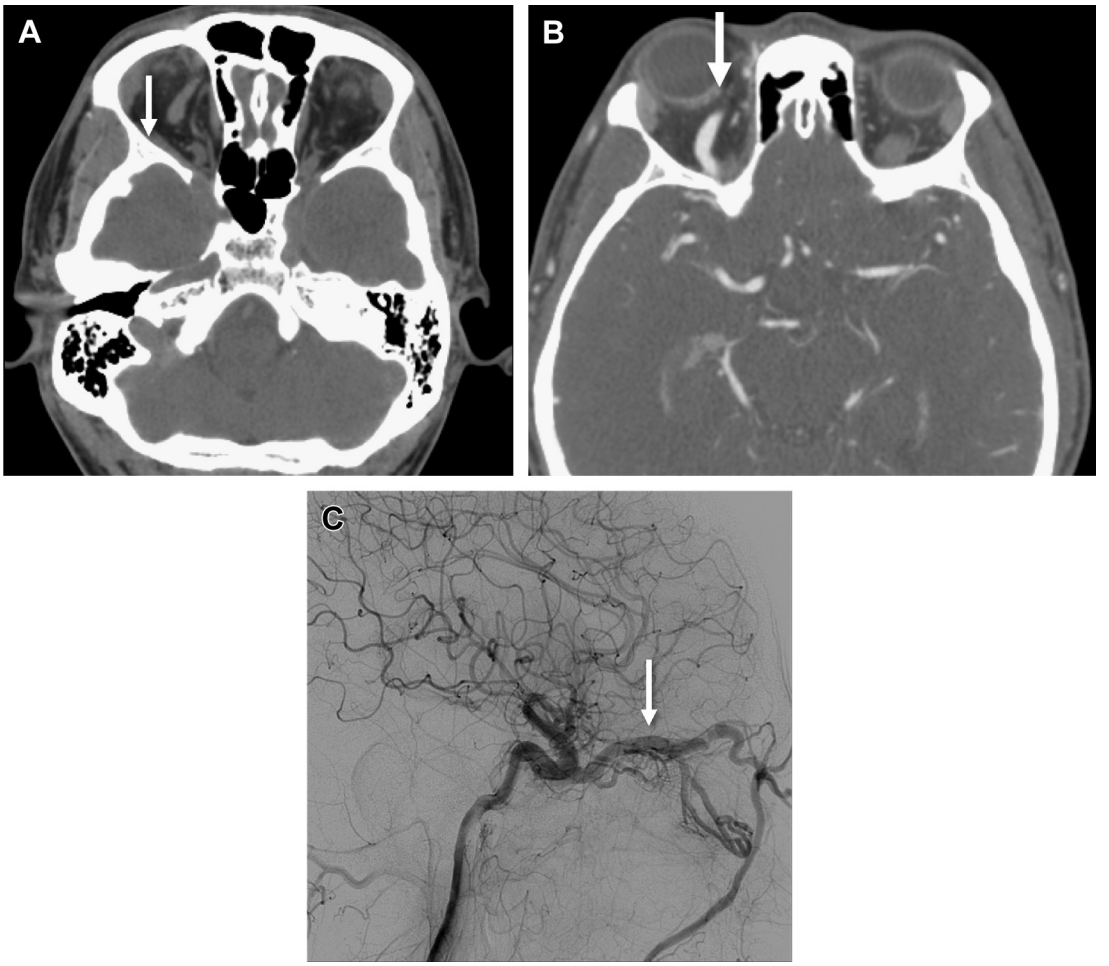

Fig. 34. Soft tissue windows of the head demonstrating asymmetric prominence of the right superior ophthalmic vein (*arrow*) (*A*), with CTA images (*B*) demonstrating early arterial enhancement of the dilated right superior ophthalmic vein (*arrow*) and mild proptosis. The findings are suggestive of carotid cavernous fistula. (*C*) Digital subtraction angiography from a right ICA injection demonstrating early filling of the cavernous sinus and enlarged superior (*arrow*) and inferior ophthalmic veins, confirming a carotid cavernous fistula.

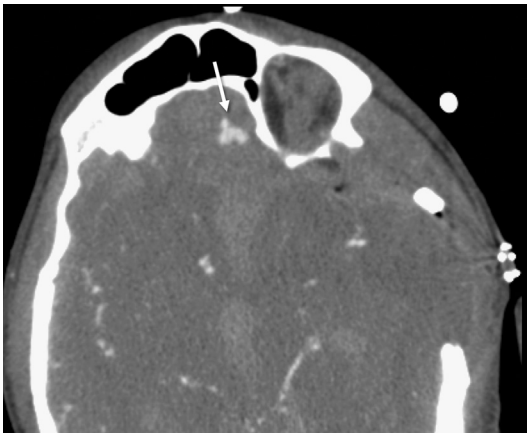

Fig. 35. Images from axial CTA demonstrating a lobulated posttraumatic aneurysm arising from the left anterior cerebral artery (*arrow*), discovered when the patient developed increased parenchymal and subarachnoid hemorrhage after a decompressive craniectomy. This complication was thought to be due to release of the initial tamponade on the ruptured aneurysm.

demonstrating early filling of the cavernous sinus and venous structures.

The imaging workup of patients presenting with symptoms of delayed CSF leak in the setting of prior trauma, including CSF rhinorrhea, otorrhea, or meningitis, should begin with thin-section high-resolution MDCT through the face and skull base, including the mastoids. Localizing the leak and characterizing the size of the skull base defect is imperative, as many repairs are performed endoscopically. MDCT has a reported 92% sensitivity for the detection of the site of the osseous defect, and is often used for intraoperative guidance for the repair.[65,66] Imaging findings suspicious for the site of CSF leak include an osseous defect or irregularity, with fluid level or opacification in the adjacent sinus. Polypoid nondependent soft tissue adjacent to the defect is characteristic of a meningoencephalocele, best assessed on MRI.[65,67] If the patient has a verified (ie, positive beta 2 transferrin) and persistent leak, and only one osseous defect and potential site of CSF leak, the imaging workup is complete, and the patient should proceed to surgery for repair.[65]

However, patients with multiple osseous defects or potential sites of CSF leak, including prior fractures through both the tegmen and the anterior skull base, may require further imaging with cisternography to determine the source of the active leak. CT cisternography with intrathecal iodinated contrast can be helpful in detecting the source of the leak; however, it requires the patient to be actively leaking, or able to elicit the leak at the time of the examination. The technique initially requires performing precontrast thin-section MDCT imaging of the sinuses and mastoids, to assess for baseline increased density in the sinus due to blood, inspissated secretions, or neoosteogenesis. The patient then undergoes lumbar puncture with intrathecal myelographic iodinated contrast followed by repeat CT of the sinuses and mastoids after head down and provocative maneuvers in an attempt to opacify the basilar cisterns and elicit the leak. Postcontrast imaging is often performed in both prone direct coronal imaging (in an effort to elicit the leak) and supine axial acquisitions with thin-section reformations, to mimic the precontrast series. Imaging findings suggesting the site of the leak include increased density and pooling of contrast in the sinuses or mastoids adjacent to the site of an osseous defect, which also can be detected by increase in Hounsfield units on region of interest measurements. Limitations of this technique include radiation to the patient, the time-intensive nature of the interpretation comparing the precontrast and postcontrast images, the invasive nature of the procedure, with small but inherent risks related to intrathecal contrast and lumbar puncture, and that the patient must be actively leaking at the time of the study.[65]

MRI cisternography is a noninvasive, nonionizing technique using heavily T2-weighted sequences, fat-saturated spin-echo sequences, and/or high-resolution 3D T2-weighted GRE sequences through the skull base, in addition to thin-section T1 precontrast and postcontrast images in multiple planes. This technique is useful in characterizing the contents of suspected meningoencephaloceles, and can detect a CSF leak as a continuous column of T2 hyperintense material extending from the subarachnoid space into the extracranial soft tissues. Although reportedly accurate in up to 89% of cases, it is best when combined with high-resolution CT, which will still be necessary for bony detail and preoperative planning (**Fig. 36**).[68–70]

PITFALLS IN DETECTION OF SKULL BASE TRAUMA AND COMPLICATIONS

In addition to the overcalling of pseudofractures due to normal sutures, one of the greatest pitfalls in the imaging of skull base trauma is the failure of fracture detection. It is imperative to use multiplanar reformations, often with additional 3D reformations, in the evaluation of suspected skull base fractures, particularly in the presence of opacified sinuses or mastoids and pneumocephalus. The use of multiplanar reformations has been proven to increase fracture detection rates.[59] However, interestingly and anecdotally, there are occasions in which fracture lines and trajectories may be

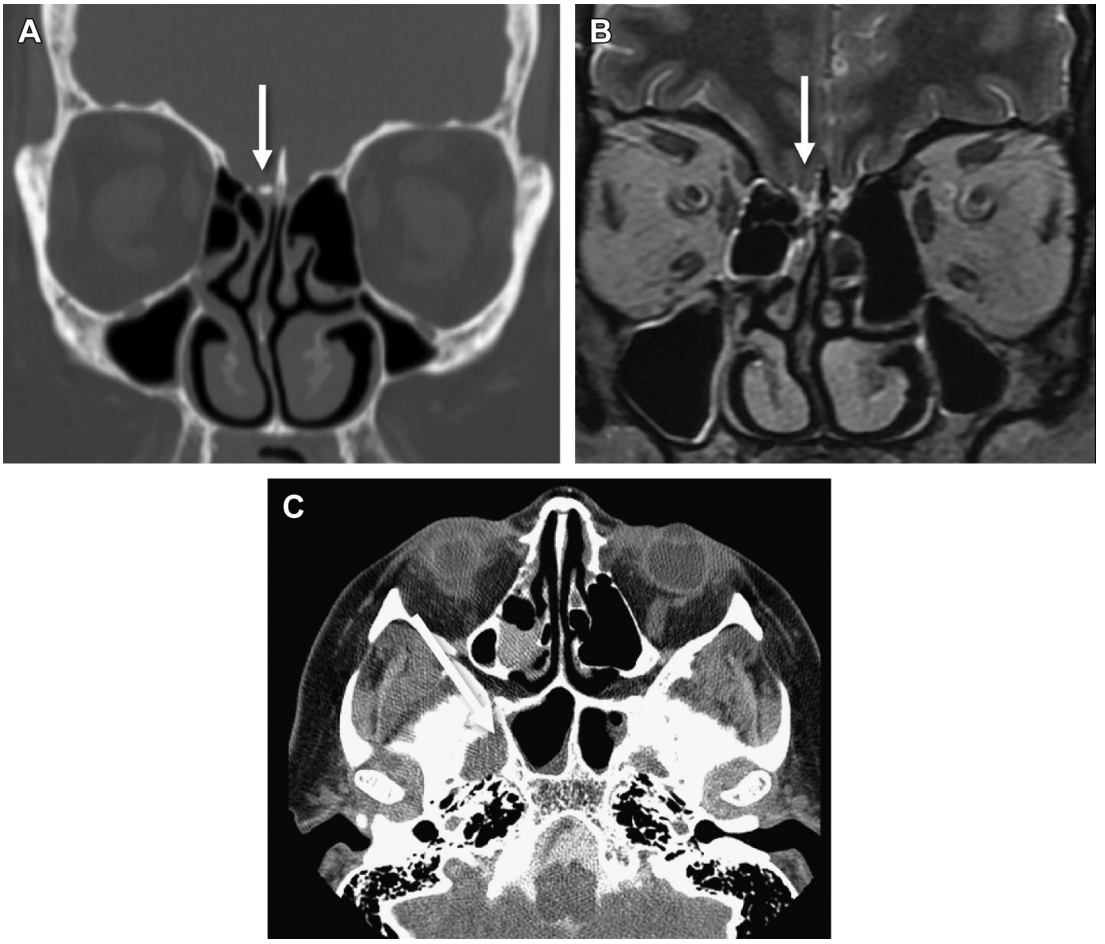

Fig. 36. (*A*) Coronal CT in a patient with a remote history of trauma and CSF rhinorrhea, with positive beta 2 transferrin, showing a defect with sclerosis along the right cribriform plate and lateral lamella (*arrow*). Another defect in the left sphenoid sinus with adjacent soft tissue was also noted, therefore the patient underwent MRI, and ultimately CT cisternography. (*B*) Coronal T2-weighted MRI image demonstrates slight sagging of the right gyrus rectus and olfactory bulb, suspicious for the site of the leak (*arrow*). Postcontrast CT cisternogram image (*C*) demonstrating pooling contrast (*arrow*) in the right posterior ethmoid, compatible with the site of the leak.

better seen on thicker-section bone algorithm images; therefore, using a combination of all of the data from thick and thin bone images is important (**Fig. 37**). One recent study recommended using curved maximum intensity projection reformations through the skull base to enhance detection rates of subtle skull base fractures.[71] Frequently missed fractures include occipital condylar fractures, subtle anterior cranial fossa fractures, and occult temporal bone fractures. Streak artifact, either from metal artifact (particularly in gunshot wound injuries) or dental amalgam can also limit evaluation of the skull base. Additionally, streak artifact through the petrous and cavernous segments of the internal carotid arteries can make evaluation on CTA challenging. Optimizing window and level settings and the use of dual-energy CT may be helpful in this setting.[72]

One of the greatest pitfalls in the imaging workup of posttraumatic CSF leaks, particularly in the delayed presentation, are those patients presenting with intermittent or suspected leaks who are unable to collect fluid for beta 2 transferrin analysis. In these patients, it is important for imaging to help confirm the diagnosis of CSF leak, as well as identify the site of the leak, particularly if high-resolution CT shows multiple osseous defects. Prior imaging modalities have been limited in the workup of these patients; however, the new technique of MRI cisternography with the administration of intrathecal gadolinium has evolved, with reported sensitivity of up to 100% for high-flow CSF leaks and 70% for intermittent leaks. In addition to the absence of ionizing radiation and relatively easy interpretation, this technique shows particular promise in the complicated

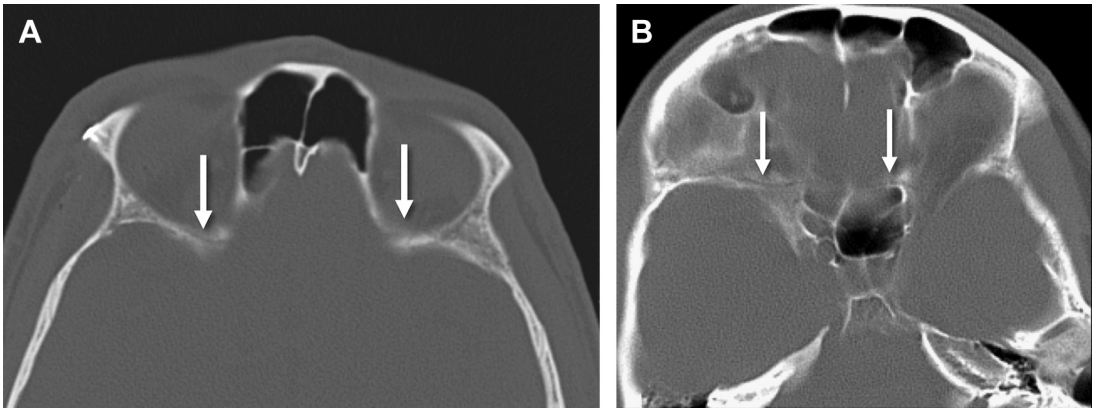

Fig. 37. Pitfall of skull base trauma imaging. Fractures may be more obvious on thicker images, but both thin and thick should be assessed. (*A*) A lucency is noted on the 0.625 slice thickness image (*arrows*), possibly a fracture. (*B*) Fracture is much better seen on the thicker 5-mm bone window image (*arrows*).

patients with intermittent leaks, due to the possibility of delayed imaging up to 24 hours after injection (**Fig. 38**).[73] However, large prospective trials and long-term safety studies are still lacking for this technique, and although most studies performed in the European literature have shown no adverse or unexpected effects with doses used, the intrathecal administration of gadolinium contrast is not currently approved by the Food and Drug Administration in the United States. Literature-based recommended algorithm for the imaging workup of chronic posttraumatic CSF leaks is described in **Fig. 39**.

WHAT THE REFERRING PHYSICIAN NEEDS TO KNOW

Regardless of classification systems used, delineating fracture extent; degree of comminution; fracture fragments; associated intracranial and intraorbital injury; and involvement of certain anatomic regions, foramina, and vascular channels is important. **Box 1** has a checklist of structures to assess in the evaluation of each skull base fracture.

In our practice, CTA/V is routinely recommended for any complex skull base fracture that involves the carotid canal, cavernous sinus, or major venous channel, including the transverse sinus, superior sagittal sinus, and jugular foramen. This imaging is done urgently, as soon as the fracture is detected, often before the patient leaves the emergency room and is admitted to the surgical intensive care unit. We are careful to coordinate the imaging and intravenous contrast doses, as patients with multiple trauma often undergo contrast CT of the abdomen and chest as well as the brain. If an arterial injury is present, the vascular neurologist is consulted, as anticoagulation in the trauma

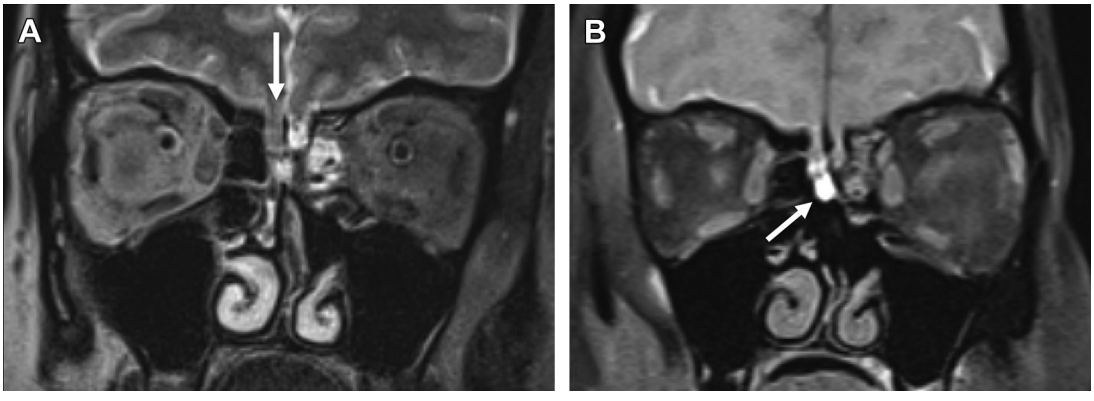

Fig. 38. Patient with intermittent CSF leak, and history of prior trauma. (*A*) Coronal T2-weighted images from noncontrast MRI cisternography demonstrating sagging of the right gyrus rectus (*arrow*), with adjacent T2 hyperintensity in the superior nasal cavity, suspicious for a meningocele. A second defect (not shown) was also present and MRI cisternography was requested to determine the source of the leak. (*B*) Fat-saturated T1-weighted image from MRI cisternogram after intrathecal gadolinium demonstrates contrast opacification of the small right meningocele (*arrow*). The left ethmoid sinuses do not fill with contrast, so the secretions are not CSF.

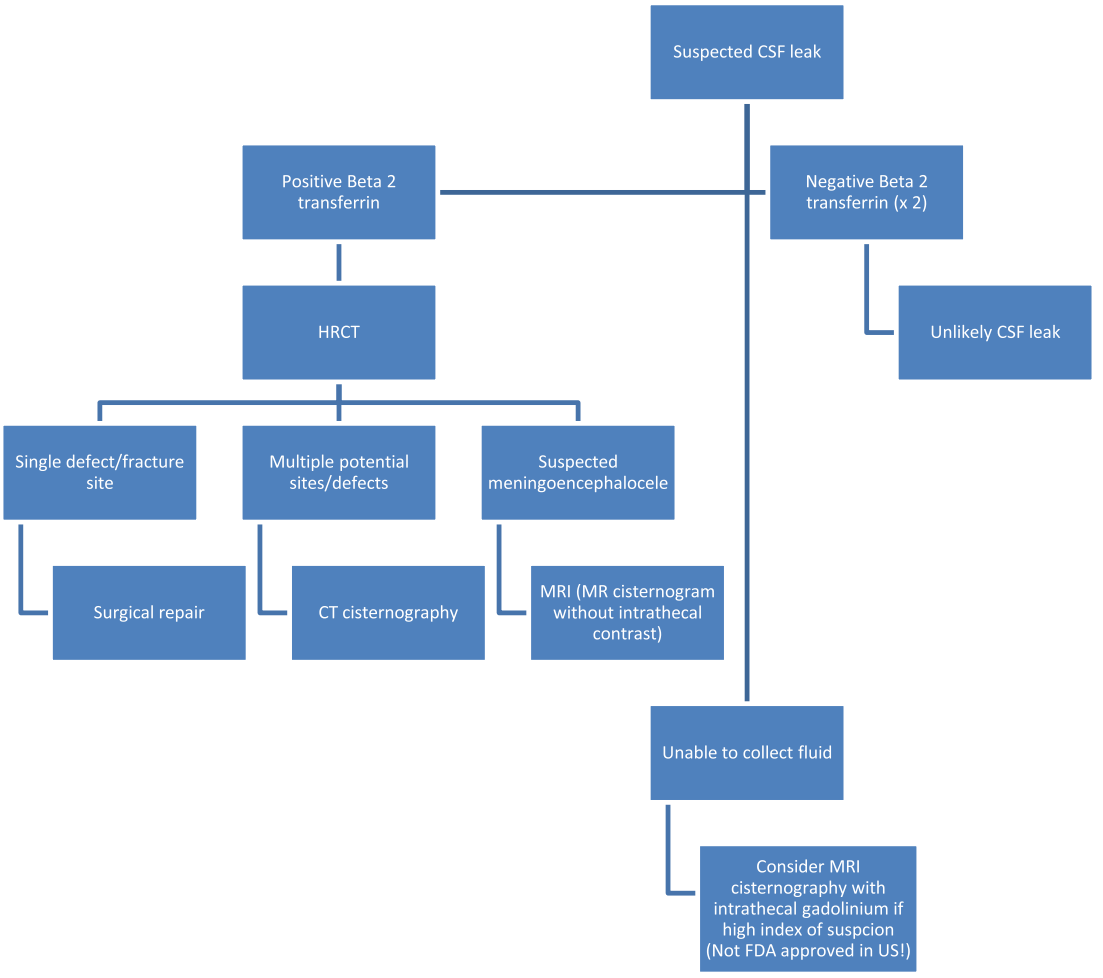

Fig. 39. Algorithm for imaging workup of chronic posttraumatic CSF leak.

Box 1
What the referring physicians need to know

Checklist of sites to assess for skull base fracture involvement.

- Posterior table of frontal sinus
- Anterior skull base
- Optic canal
- Superior orbital fissure
- Skull base foramina involvement (ie, foramen rotundum/ovale)
- Carotid canal
- Sigmoid sinus/jugular foramen
- Tegmen
- Clivus
- Occipital condyle
- Hypoglossal canal

patient is possible, despite intracranial and abdominal hematomas, but the risk versus benefit must be considered.

CSF leak, on the other hand, is a complication that is considered later, in the rehabilitation period. Most small dural defects close spontaneously and require no advanced imaging. Cranial nerve injury, especially the facial nerve, should be diagnosed in the acute to subacute period, using a combination of the clinical examination and imaging, so that neural repair can be considered.

FUTURE CONSIDERATIONS/SUMMARY

Skull base fractures are a frequent complication of high-impact trauma, and due to the inherent anatomic relationships of the skull base, even relatively linear nondisplaced fractures may be associated with significant intracranial complications, including CSF leak; therefore, their detection is important. Additionally, continued improvement

in endoscopic techniques have led to fewer open surgeries for repair, necessitating better and more accurate assessment of skull base fractures and resultant defects. MDCT scanner and reconstruction software improvements will likely continue to facilitate fracture detection, with the faster, newer-generation scanners allowing for less motion, and contributing to better CTA techniques. Dual-energy CT may be helpful in the future as well. Finally, MRI with intrathecal gadolinium is a promising new technique for the evaluation of CSF leak, particularly in the setting of intermittent or suspected leaks.

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