Cerebrovascular Trauma

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KEYWORDS

• Cerebrovascular • Trauma • Dissection • Diagnosis • Therapy

KEY POINTS

- Significant recent progress has been made in the recognition, screening, diagnosis, and treatment of blunt cerebrovascular injury (BCVI).
- Although controversy still exists as to optimal screening algorithms and best diagnostic modality, the vital and growing role of noninvasive imaging in identifying patients at high risk for BCVI and in characterizing the injury itself has been clearly established.
- There has been promising early work in stratifying BCVI patients into risk categories by initially evaluating them with high-resolution head, maxillofacial, and cervical computed tomographic (CT) examinations with the ultimate goal of maximizing diagnostic yield and enabling prompt initiation of therapy.
- Further work is needed to delineate the mechanistic relationship between craniofacial fractures and BCVI.
- Recent studies indicate the incidence of BCVI may be much higher (1%–3%) than initially reported (0.1%), due to the wider utilization of aggressive screening algorithms and noninvasive imaging.
- A high index of suspicion is necessary to identify BCVI, since many patients exhibit a latent, asymptomatic period.
- Untreated BCVI is associated with high morbidity and mortality. Identification and treatment of patients while they are asymptomatic has been shown to improve outcomes.
- CT angiography is the study of choice for initial imaging of traumatic CVI, although magnetic resonance imaging/magnetic resonance angiography demonstrates considerable value in characterizing vessel injury as well as associated ischemic complications.
- Current screening algorithms reinforce the importance of high-resolution head, maxillofacial, and cervical spine CT in identifying patients at high risk for BCVI.

INTRODUCTION

Historical Perspective and Significance of Traumatic Blunt Cerebrovascular Injury

The recognition of blunt cerebrovascular injury (BCVI) as an important diagnostic entity has occurred only in the past 2 decades, with continued current debate as to best practices in regards to screening, diagnosis, treatment, and follow-up.

The true incidence of BCVI in the setting of trauma is still not known but has been greatly underestimated in the past, largely because of a lack of routine imaging of asymptomatic patients. Before 1990, less than 200 total blunt carotid artery injury (BCAI) cases had been described in the literature.[1] Regionalization of trauma care caused these "uncommon" injuries to be funneled into fewer referral centers, generating greater interest in improving diagnosis. Many studies before the mid 1990s reported a 0.1% overall incidence of blunt injury to the carotid artery in trauma victims.[2–5] With subsequent utilization of aggressive screening criteria,

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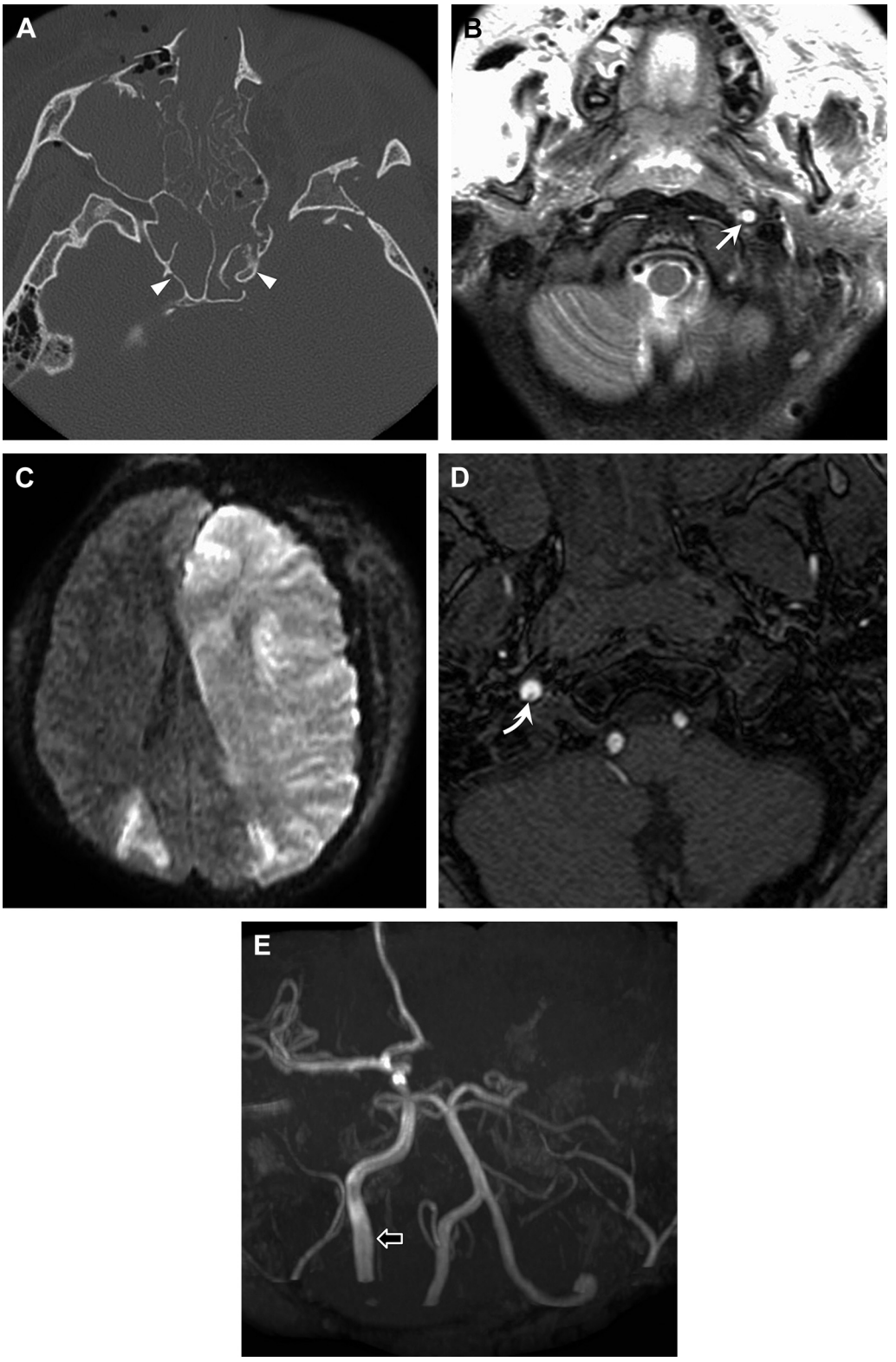


Fig. 1. Traumatic right internal carotid dissection and left internal carotid occlusion. (*A*) Axial CT shows bilateral fractures of the carotid canals (*arrowheads*) with more severe displacement on the left. (*B*) Axial T2-weighted and (*C*) diffusion-weighted MR images reveal a left carotid occlusion (*arrow*) and bilateral hemispheric infarcts, greater on the left. (*D*) 3D-TOF source and (*E*) maximum intensity projection (MIP) MRA images reveal a lack of flow in the left ICA and a dissection flap of the right ICA (*curved arrow*). The MIP image reveals slight overall enlargement of the distal right ICA (*open arrow*) but does not clearly reveal the dissection itself.

however, the incidence of documented cerebrovascular injury in blunt trauma patients increased 10-fold to 1%, and even higher (2.7%) when applied to patients with Injury Severity Scores of greater than or equal to 16.[1,6–11] Although initial emphasis was placed on carotid arterial injury (CAI), the incidence of vertebral artery injuries (VAI) from blunt trauma was found to range from 0.53% to 0.73%.[10,12]

Despite the relative infrequency of BCVI, devastating complications are very common in patients with documented injuries (**Fig. 1**). A 1998 review of the literature reported BCAI mortalities of 23% to 28%, with even higher rates of permanent neurologic deficit (48%–58%).[7] Similarly, a mortality of 8% and permanent morbidity of 14% to 24% have been reported in untreated patients with blunt VAI.[10,12] Over the past decade, a growing body of evidence has revealed that a significant percentage of BCVI patients present in a delayed fashion, with ischemic events following a latent asymptomatic period. Antithrombotic medical therapy has been recently shown to decrease the incidence of posttraumatic stroke significantly and improve final neurologic outcome, emphasizing the importance of early diagnosis.[1,7,12,13]

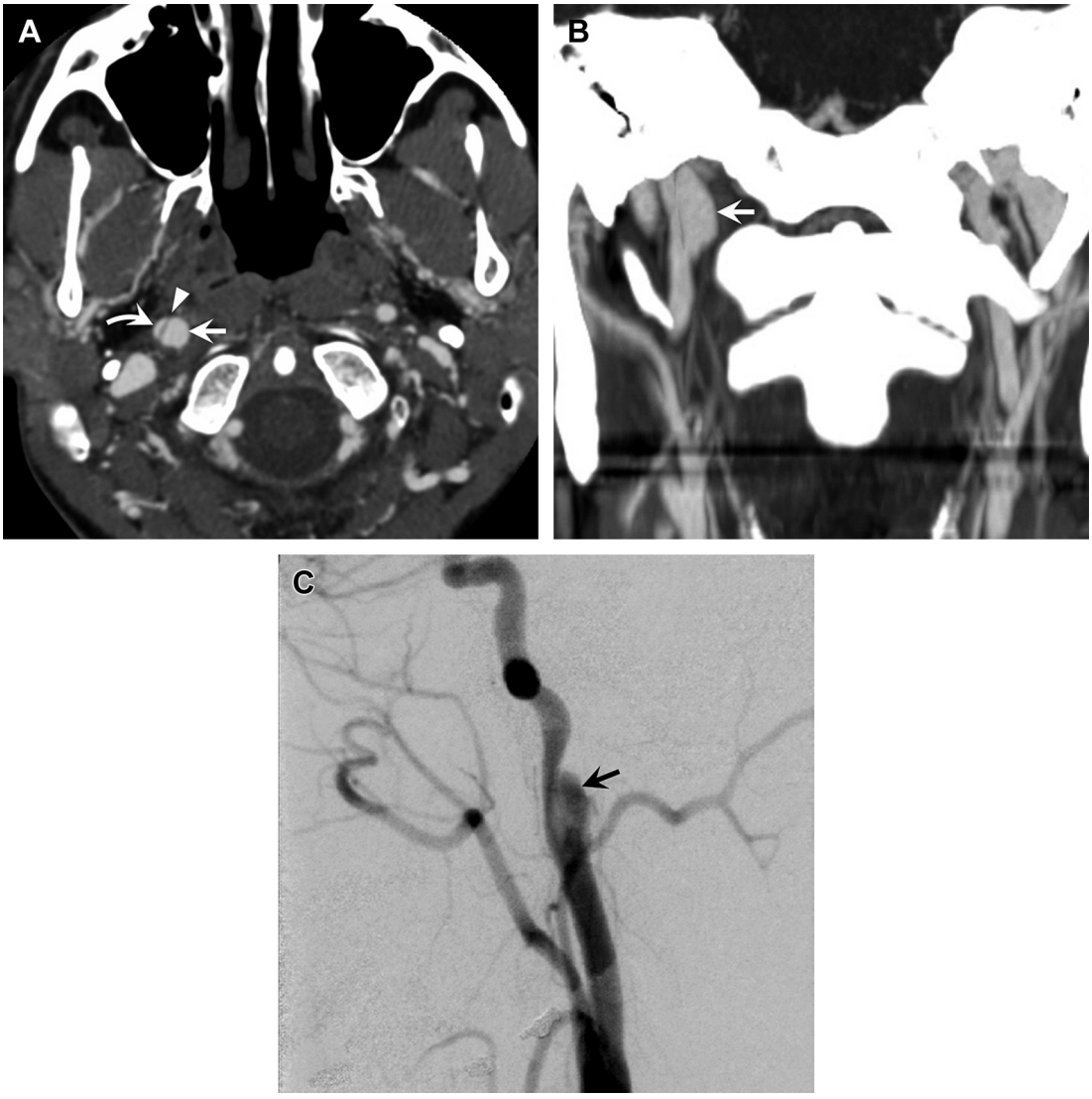


Fig. 2. Right internal carotid dissection and pseudoaneurysm. (*A*) Axial CTA image reveals a dissection flap (*arrowhead*) with a large pseudoaneurysm (*arrow*) that severely compresses the true ICA lumen (*curved arrow*). (*B*) Coronal 2D reconstructed CTA image and (*C*) lateral angiogram confirm the dissection, pseudoaneurysm (*arrow*), and compressed true ICA lumen.

As a result, aggressive screening protocols have been instituted, with an emphasis on utilization of noninvasive imaging modalities, such as computed tomographic angiography (CTA) and magnetic resonance angiography (MRA).

ANATOMY AND PATHOLOGY

Mechanisms and Patterns of Cerebrovascular Injury

Commonly accepted physiologic mechanisms of traumatic cerebrovascular injury include extreme cervical hyperextension/rotation, direct blunt vascular trauma, intraoral trauma, and direct laceration from bony fracture fragments.[14] Traumatic cerebrovascular dissections typically result from rapid deceleration of the body and resultant stretching of the involved vessel. This mechanism can be seen in patients following motor vehicle accidents, assault, pedestrian accidents, falls, and with hanging accidents.[7] Although consistently implicated as a risk factor for BCVI, the mechanisms associated with craniofacial fractures are not as well delineated.

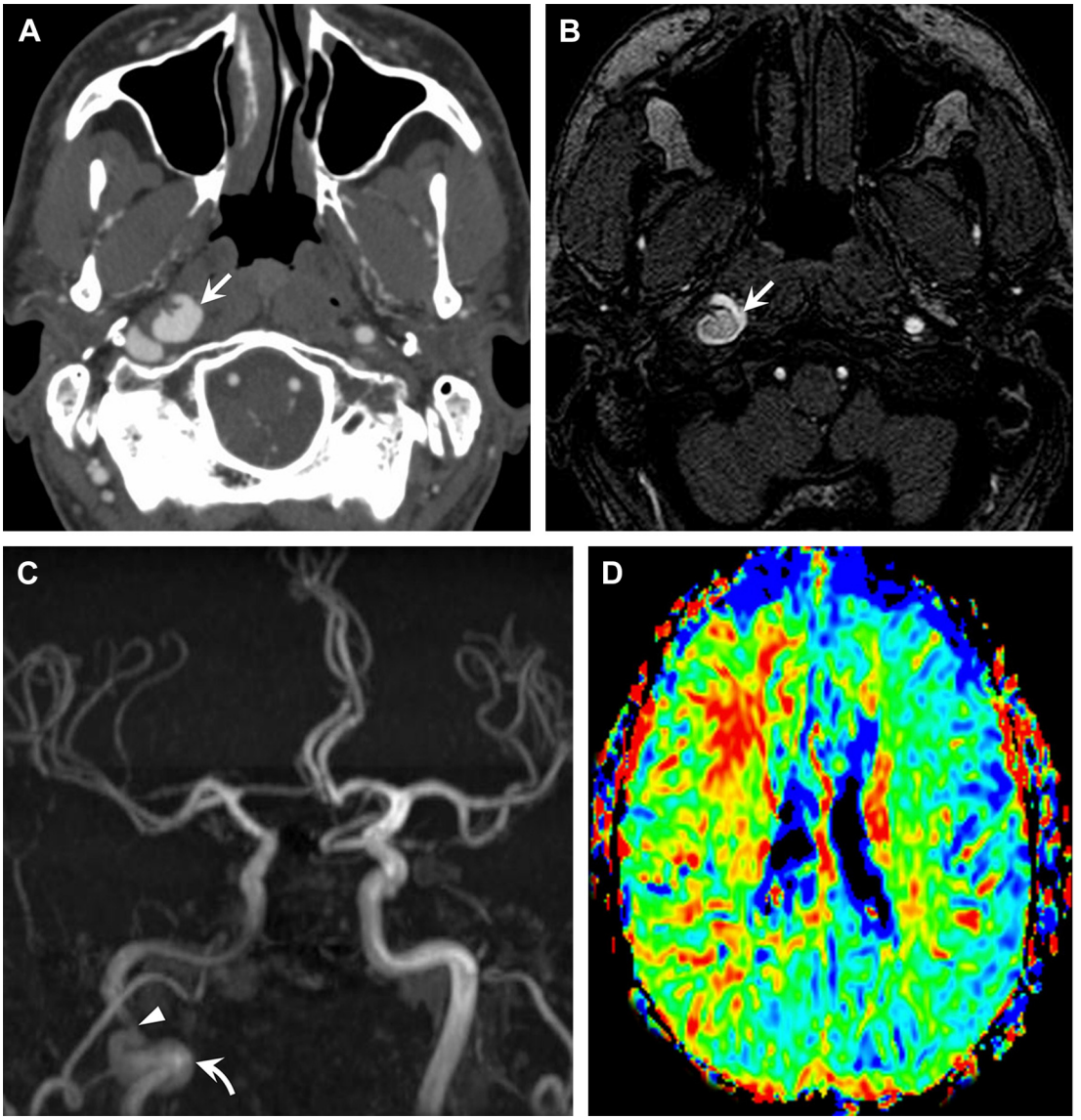


Fig. 3. Right internal carotid dissection, pseudoaneurysm, and hemispheric perfusion defect. (*A*) Axial CTA and (*B*) 3D-TOF MRA images reveal an enlarged, irregular ICA lumen compatible with traumatic pseudoaneurysm (*arrow*). (*C*) MIP MRA image confirms the pseudoaneurysm (*curved arrow*) and narrowed true ICA lumen (*arrowhead*). (*D*) Perfusion images demonstrate a prolonged transit time in the right hemisphere, indicating a risk of subsequent infarct.

The extracranial segments of the carotid and vertebral arteries are more vulnerable to traumatic injury than the intracranial segments because of their close relationship to surrounding osseous structures and relative greater mobility.[15,16] Extracranial carotid artery injuries most commonly occur in the distal cervical internal carotid artery (ICA) (**Figs. 2** and **3**). Injury is thought to result from stretching over the lateral masses of the cervical vertebrae (particularly C1-3) in the setting of head hyperextension and contralateral hyperrotation, and from impingement on the styloid process during head rotation. The ICA may also be compressed between the mandible or hyoid bone and the cervical spine during neck hyperflexion.[14–20] Prior studies have reported that superior displacement of the pterygoid plates (as in the case of Le Fort type fractures) poses a risk to the ICA inferior to the foramen lacerum.[21] Displaced bony fragments from skull base fractures may also lead to direct injury of the ICA.[14]

Extracranial VAI most commonly involves the V2 and V3 segments, because the vessel travels through the bony transverse foramina and around C1, respectively (**Figs. 4** and **5**).[22] Displaced fracture fragments of the cervical spine may directly lacerate these segments of the vertebral arteries.[14] Injuries to the V3 and V4 segments occur more commonly without associated cervical spine fracture/dislocation than injuries to the V2 segment.[23–25]

Although there is a relative dearth of data on incidence, it is generally accepted that intracranial cerebrovascular injury is less common than extracranial BCVI. Basilar skull fracture, certain patterns of facial fracture, and fractures extending through the carotid canal have been reported as risk factors for intracranial arterial injury (**Figs. 6** and **7**).[26] Manifestation of injury includes vascular compression, dissection, dissecting aneurysm, occlusion, arterial rupture, and arteriovenous fistula (carotid-cavernous).

Pathophysiology of BCVI

Different mechanisms of carotid and VAI contribute to a varied appearance on imaging. Blunt cerebrovascular dissection usually begins with a trauma-induced intimal tear or primary intramural hematoma.[27,28] With intimal injury, exposed subendothelial collagen initiates platelet aggregation to form thrombus (**Figs. 8** and **9**), which may produce vessel stenosis or occlusion or result in distal embolization (see **Fig. 8**). A dissecting hematoma within the media may propagate cranially to narrow or occlude the vessel (**Figs. 10** and **11**), or focally expand the adventitia to form a traumatic dissecting aneurysm (also referred to as "pseudoaneurysm").

IMAGING

Imaging Findings of BCVI

It is important to be familiar with the spectrum of findings and imaging pitfalls associated with the diagnosis of vascular injury on CTA, magnetic resonance (MR) imaging, MRA, and conventional angiography. Ultrasound imaging plays a limited role in diagnosis of cerebrovascular injury (see

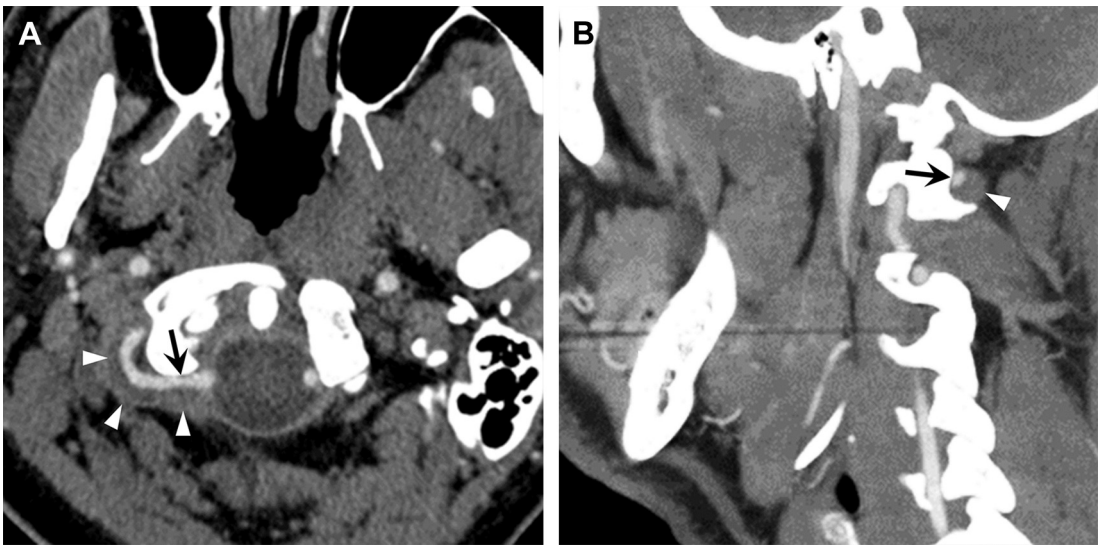


Fig. 4. Traumatic right vertebral dissection and intramural hematoma. (*A*) Axial and (*B*) sagittal CTA images show mild compression of the true lumen of the V3 segment of the right vertebral artery (*arrows*) by an extensive intramural hematoma (*arrowheads*). (*Courtesy of* Rihan Khan, MD.)

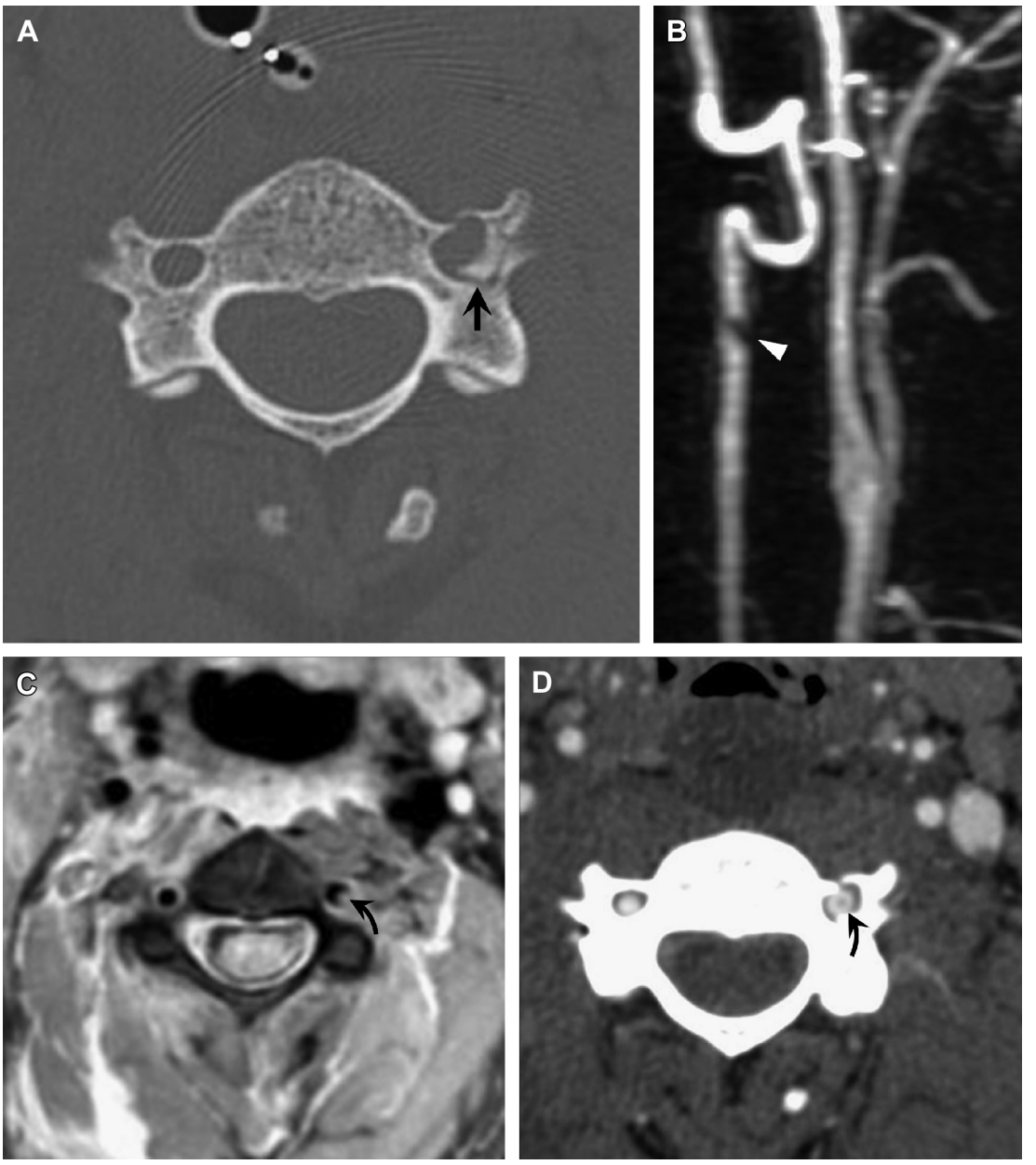


Fig. 5. Displaced foramen transversarium fracture, vertebral artery dissection, and intraluminal thrombus. (*A*) Axial CT shows a comminuted foramen transversarium fracture with displaced bone fragments (*arrow*). (*B*) Contrast-enhanced MRA reveals a dissection flap (*arrowhead*) within the left vertebral artery lumen. (*C*) Contrast-enhanced MR and (*D*) CTA images confirm the presence of intraluminal thrombus (*curved arrow*) at the dissection site.

Fig. 8), because of its poor detection of specific signs of BCVI lesions in the depth of the neck, where many ICA and VAI occur.[29]

In those patients at risk for BCVI, imaging of the entire cerebrovascular system should be performed from the aortic arch through the circle of Willis, as vessel injury may be remote from other signs of trauma. Another important imaging principle in the assessment for BCVI is to be aware of the high rate of multiple lesions. Several series have reported that up to 22% to 43% of injuries are bilateral (Figs. 12 and 13).[12,30–32]

Digital subtraction angiography (DSA) has been considered the gold standard for diagnostic evaluation of BCVI for many years (see Figs. 2 and 13). It is limited, however, by an inability to characterize the thickness and configuration of the arterial wall, the requirement to transport the patient

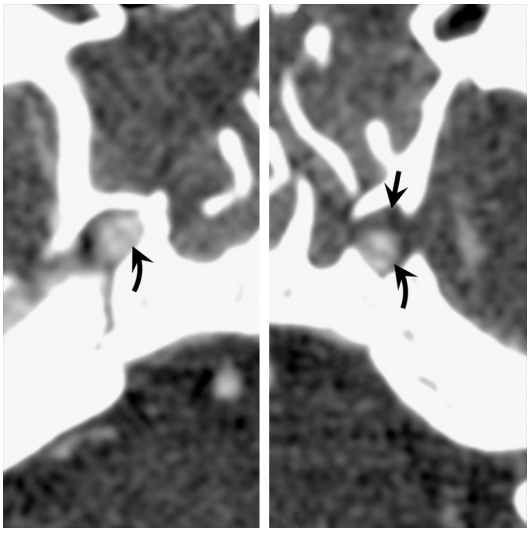


Fig. 6. Subtle bilateral ICA dissections with intimal flaps. The CTA image reveals bilateral ICA dissections with subtle intraluminal intimal flaps (*curved arrows*) consistent with a Denver grade 2 injury. Additionally seen is extraluminal hematoma (*arrow*) that does not compress the arterial lumen.

outside the emergency department, its invasive nature, and risk of procedural complications. The classic angiographic finding of dissection is an eccentric, long segment, tapered stenosis ("string sign") often associated with intimal irregularity (see **Fig. 13**). Focal narrowing with a more distal site of dilatation ("string-and-pearl sign") (see **Fig. 13**) can also be present.[33] Tapered stenosis with a concomitant dissecting aneurysm, occlusion, and isolated dissecting aneurysm are the most common imaging findings, in that order.[33] Pathognomonic imaging signs on DSA, such as intimal flap or a double lumen, are seen less commonly.

CTA provides the advantage of both high-spatial and high-contrast resolution of the arterial wall and lumen (see **Figs. 2–9**). In contradistinction to the 2 projections typically obtained with conventional angiography, CTA allows profiling of the entire 360° circumference of the arterial lumen, increasing sensitivity for detection of minor vessel injury. Inclusion of an unenhanced head CT is an essential component of a CTA protocol, to evaluate for associated intracranial hemorrhage and/or ischemia. These nonenhanced images may occasionally demonstrate injuries of the distal segments of injured carotid and vertebral arteries. Dissecting intramural hematomas can manifest on unenhanced CT as a hyperdense crescent-shaped mural lesion, often visualized near the skull base. On CTA, the same pathologic abnormality will be seen as luminal narrowing caused by crescentic intramural hematoma, which is usually isodense to muscle.[34] Because this may be difficult to distinguish from atherosclerotic disease, recognizing that dissection will typically spare the carotid bulb is essential to making the correct diagnosis. Often the intramural hematoma causes overall enlargement of the external vessel diameter, despite narrowing of the lumen (see **Figs. 2** and **3**).[35] Other reliable signs of dissection on CTA include the identification of an intimal flap or dissecting aneurysm (see **Figs. 2**, **6**, and **9**). Multiplanar 2-dimensional (2D), curved planar 2D, and 3-dimensional (3D) reformations can be obtained to create images that are comparable to those seen with conventional angiography. Although these reformations are complementary, it is absolutely essential to evaluate the thin-section axial CT source images systematically for signs of vascular injury, as it may be obscured on 2D reconstructed images.

The MR imaging appearance of dissection is highly dependent on the age of the intramural hematoma, the surrounding tissues, and MR imaging sequences used for evaluation.[36] The MR imaging appearance of the hematoma will follow the known age-dependent signal intensity of paramagnetic iron (see **Figs. 1**, **3**, **10**, and **11**). The intramural hematoma is usually most apparent in the subacute stage.[36,37] Subacute hematomas (containing methemoglobin) demonstrate characteristic findings on fat-suppressed T1-weighted images (see **Fig. 10**). The intramural hematoma will be seen as a high-intensity crescentic lesion adjacent to an eccentric flow void, which represents the residual lumen. The subacute intramural hematoma, with its short T1 values, will also be evident on noncontrast time-of-flight (TOF) MRA and can be mistaken for flow on these images (see **Fig. 10**). Phase-contrast and contrast-enhanced MRA will more clearly differentiate flow from the adjacent intramural hematoma. The intramural hematoma often causes overall enlargement of the external vessel diameter (see **Figs. 3** and **10**). Important pitfalls of MR imaging include the relative isointense appearance of acute (<7 days) and chronic (>2 months) hematoma on T1-weighted imaging, which blends in with surrounding tissues with fat suppression.[38] Dephasing and signal dropout on TOF images caused by turbulent flow in the horizontal petrous segment of the ICA can mimic intraluminal thrombus or dissection. Signal loss from in-plane flow or slab artifact on TOF imaging can also result in poor signal in horizontal segments of carotid or vertebral artery branches. Systematic evaluation of source images on TOF or contrast-enhanced MRA images is essential, to avoid missing subtle injury that may not be as apparent on reformations.

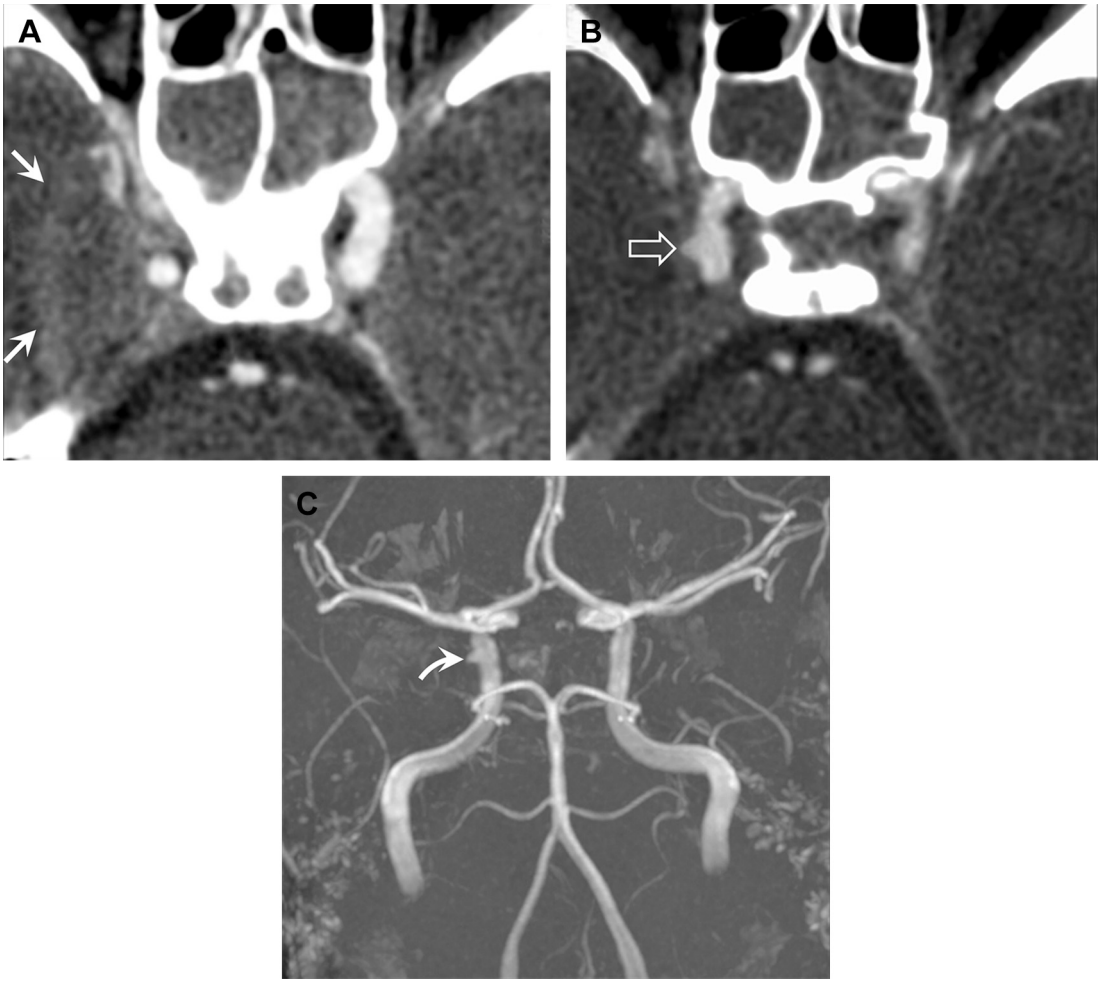


Fig. 7. Small traumatic internal carotid pseudoaneurysm. (*A*, *B*) CTA images reveal a large parasellar epidural hematoma (*arrows*) with a probable small traumatic ICA pseudoaneurysm (*open arrow*). (*C*) The aneurysm (*curved arrow*) is confirmed on a 3D-TOF MIP image. Extensive central skull base fractures likely partially avulsed an intracavernous ICA branch from the parent artery.

Color Duplex ultrasound has a limited role in the evaluation of BCVI patients, as 90% of traumatic lesions occur in acoustically nonassessable segments of the carotid and vertebral arteries.[29] The cephalad parts of the extracranial ICA and VA are difficult to image, requiring use of low-frequency sector transducers and reliance mainly on hemodynamic abnormalities for diagnosis of dissection. Most dissecting aneurysms are missed.[39] That said, specific signs of dissection may be detected in the more accessible proximal ICA, with mural hematoma manifesting as a thickened hypoechoic vessel wall on B-mode or high-frequency sector transducers.[40] Intimal flaps and double lumens are occasionally depicted (see **Fig. 8**).

BCVI Classification

Traumatic cerebrovascular injuries can be classified by the location of injury (intracranial vs extracranial) and/or the extent of vessel wall involvement. The mildest form of injury is merely extrinsic compression of the lumen by extramural hematoma, (**Fig. 14**) without a true tear of the vessel wall. True vascular tears may affect just the intima, both the intima and the media, or may extend through the entire thickness of the vessel wall. The greater the extent of wall disruption, the more abnormal the vessel configuration will appear on imaging studies (**Figs. 15–20**).

The emerging literature on blunt BCVI in the early to mid-1990s prompted the call for a formal injury grading scale that could stratify injuries

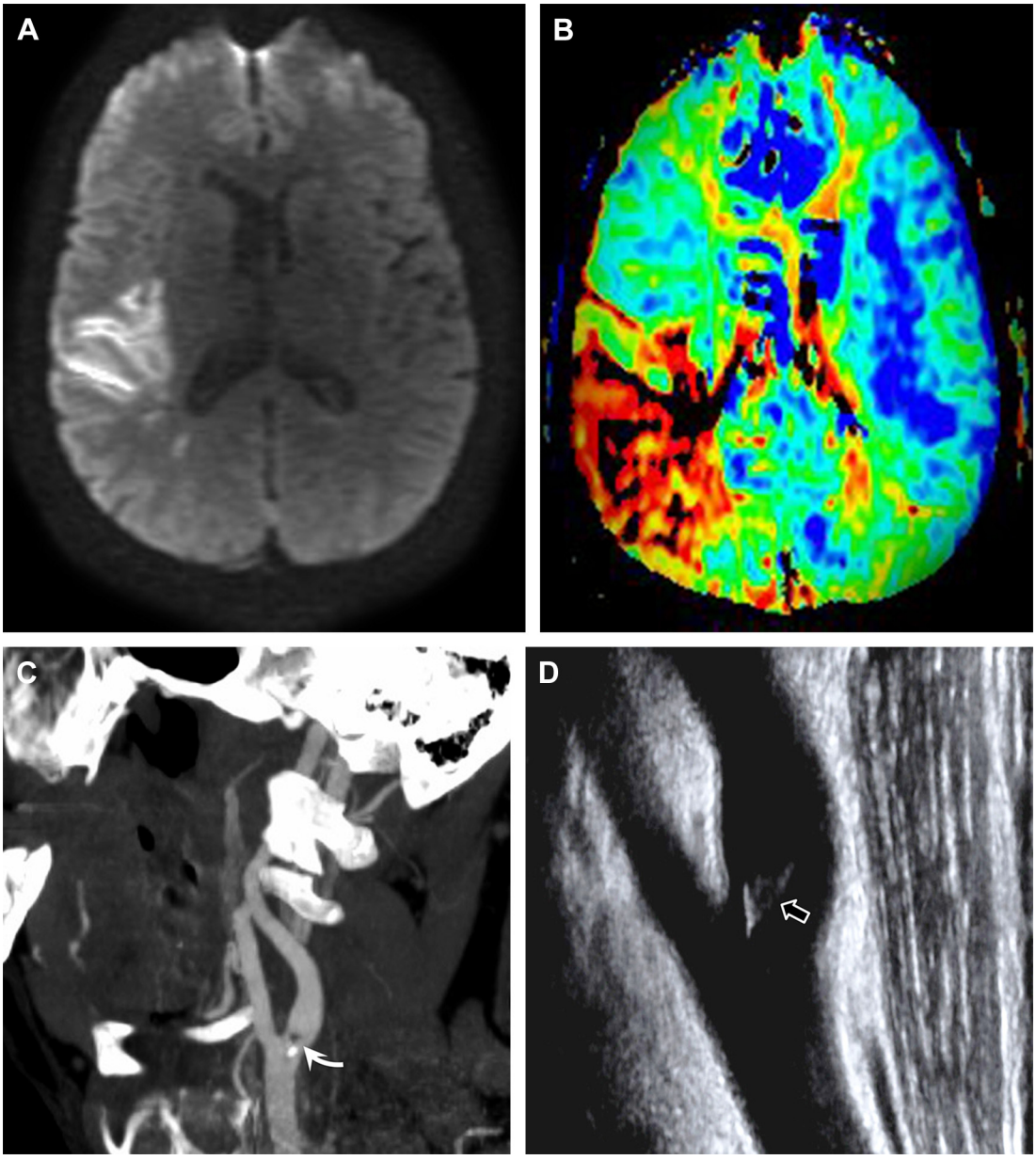


Fig. 8. Subtle traumatic dissection with intraluminal thrombus and embolic stroke. (*A*) Diffusion-weighted and (*B*) perfusion MR images. This patient presented with a right hemispheric infarct 24 hours following a motor vehicle accident and blunt anterior neck trauma. Note the size of the perfusion deficit is greater than the size of the diffusion-weighted image abnormality, indicating an ischemic penumbra. (*C*) Sagittal 2D reformatted CTA image reveals a filling defect due to thrombus (*curved arrow*) within the lumen of the ICA without other abnormality. (*D*) Duplex sonogram confirms the presence of intraluminal clot (*open arrow*) likely due to a subtle traumatic dissection.

by type, location, and neurologic presentation, as well as provide prognostic and therapeutic value.[2] As a result, a cerebrovascular injury grading scale was developed at Denver Health Medical Center in 1999, based on the conventional arteriographic imaging appearance of lesions. This Denver grading scale has been widely used in subsequent large prospective and retrospective series throughout the surgical and trauma literature. This literature has demonstrated that different injury grades may have differing risks of morbidity and mortality, distinct responses to therapy, and differing final neurologic outcomes.[9,11,12,29,32,41–44]

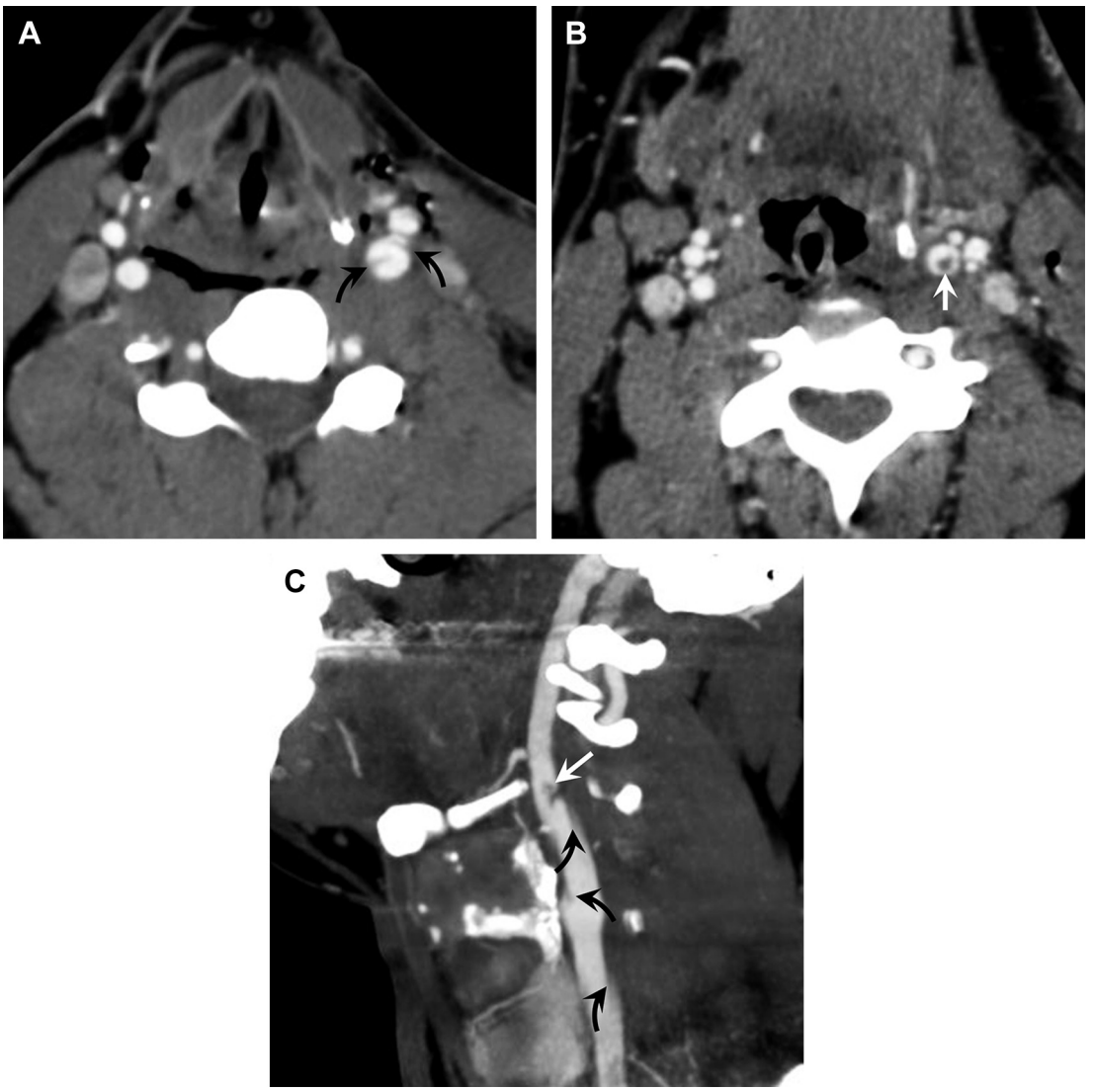


Fig. 9. Traumatic dissection with intraluminal thrombus. Axial (*A*, *B*) and sagittal 2D reformatted (*C*) CTA images reveal a complex dissection of the proximal ICA (*black curved arrows*) with evidence of intraluminal thrombus (*arrow*), indicating a Denver grade 2 injury. This patient is at great risk for subsequent distal embolization unless treated with antithrombotic agents.

Broadly, the Denver grading scale (**Table 1**) categorizes lesions as nonhemodynamically significant injuries (grade I) (see **Fig. 12**), potentially hemodynamically significant dissections and hematomas (grade II) (see **Figs. 5**, **6**, **8–13**), pseudoaneurysms (grade III) (see **Figs. 2**, **3**, **7**, and **18**), occlusions (grade IV) (see **Figs. 1** and **20**), and vessel transections with free extravasation (grade V) (see **Figs. 15–17** and **19**). Specifically, grade I injury is defined as irregularity of the vessel wall or a dissection with less than 25% luminal stenosis. Grade 2 injuries consist of a dissection of the vessel wall with greater than 25% luminal stenosis or a dissection with a visible intimal flap.[30] Some have proposed grade V injuries be separated into noncontained rupture with free extravasation and intravascular rupture (arteriovenous fistula).[45]

Role of Imaging in BCVI Screening

A significant percentage of BCVI patients may present with an initially asymptomatic period.[1,7,12,13] Thus, a window of opportunity exists to treat traumatic lesions before irreversible, catastrophic complications develop (see **Fig. 20**). Identifying

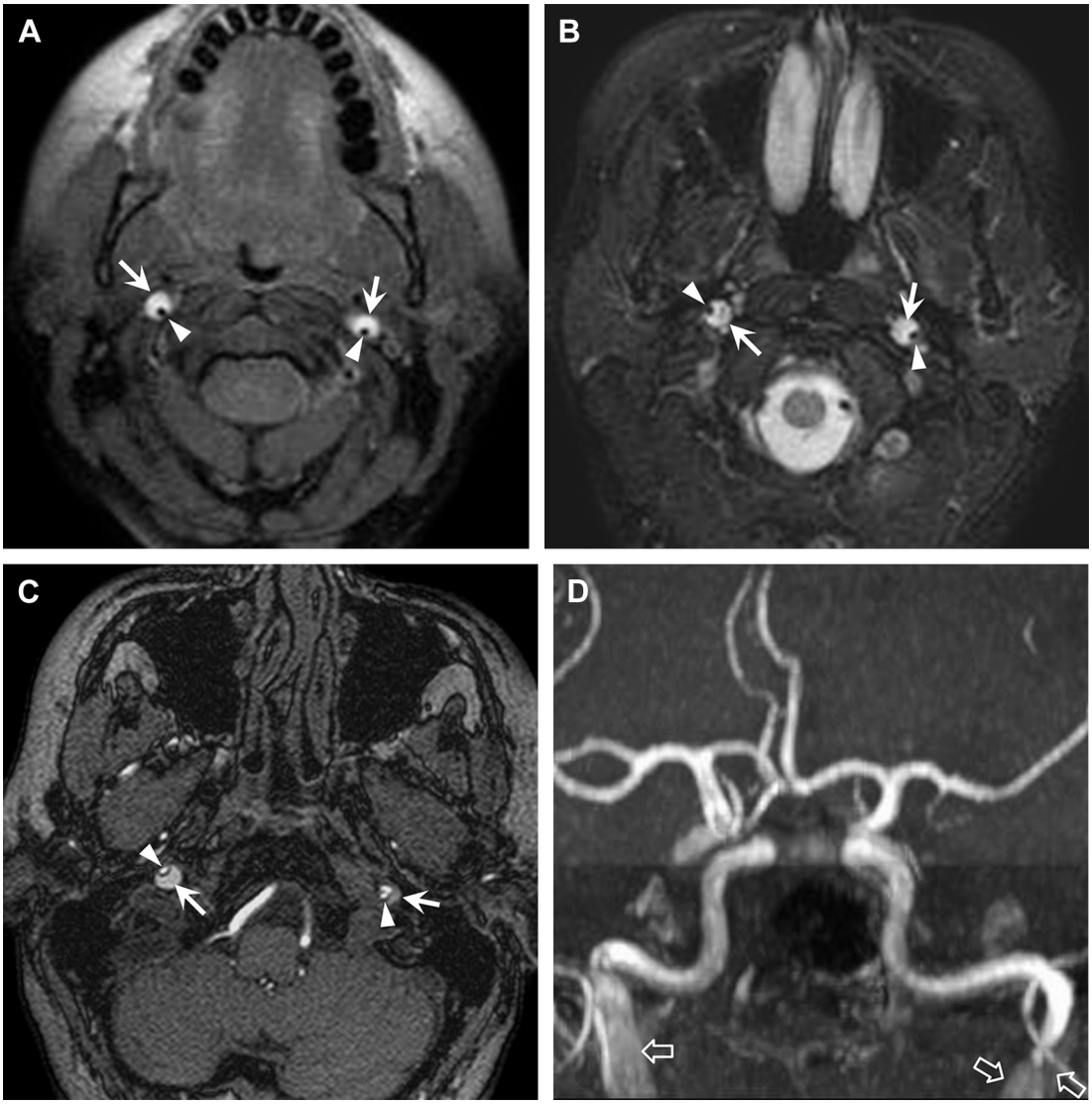


Fig. 10. Bilateral ICA dissections causing severe luminal compromise. Axial proton density-weighted (PD-W) (*A*) and T2-weighted (*B*) MR images as well as source (*C*) and MIP (*D*) images from a 3D-TOF MRA demonstrate bilateral ICA dissections 1 week following neck trauma. Note that the true lumens (*arrowheads*) are severely narrowed by intramural hematoma (*arrows*). The intramural hematoma is hyperintense and very visible on the PD-W and T2-weighted images. The hematoma is also hyperintense on the T1-weighted source image, which can be mistaken for flow on the MIP image (*open arrows*).

BCVI before the onset of symptoms is the primary goal of imaging, to facilitate prompt initiation of adequate antithrombotic medical therapy. Early effective treatment has been demonstrated to improve neurologic outcomes and prevent stroke.[1,7,12,13] Diagnosis and implementation of treatment during this silent period has been the "holy grail" of BCVI, driving the use of aggressive and liberal screening protocols to capture this population. Although challenges and controversy exist in defining the population at risk, the integral role of noninvasive high-resolution CT screening in both asymptomatic and symptomatic patients has been clearly established.

Several prior studies have looked at various clinical and radiographic criteria to try and predict which patients with craniocervical trauma are at a high risk of cerebrovascular injury. The 2 most widely implemented screening protocols are based on the Memphis criteria, developed by Miller and colleagues,[10] and the Denver criteria, developed by Biffl and colleagues.[7,30] Most

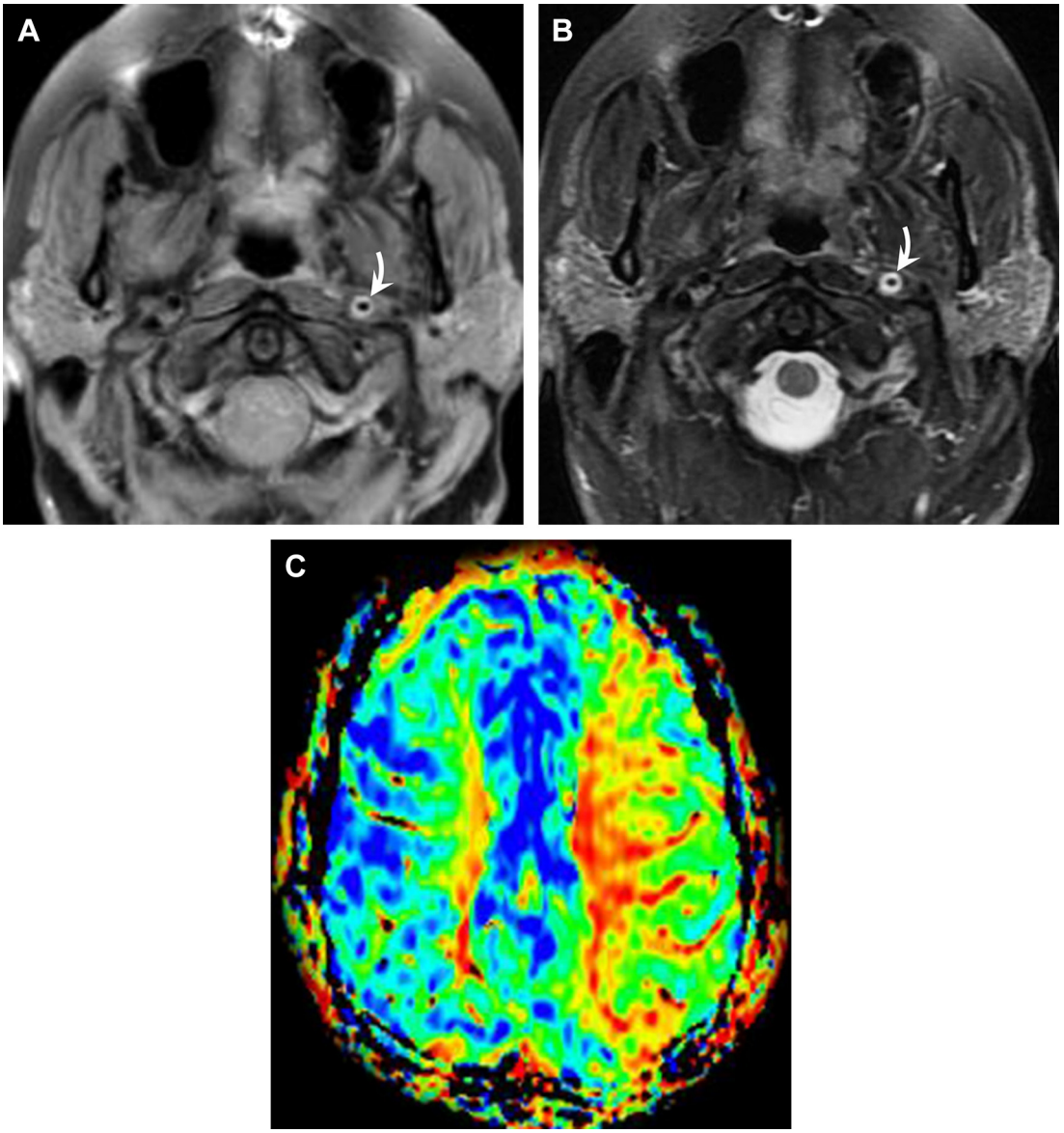


Fig. 11. Left ICA dissection with severe luminal compromise and perfusion deficit. Axial PD-W (*A*) and T2-weighted (*B*) MR images as well as a perfusion MR image (*C*) demonstrate a left ICA dissection with circumferential intramural thrombus (*curved arrows*). The carotid lumen is severely narrowed and there is prolonged transit time on the perfusion image, indicating a risk for subsequent infarct.

recently, the Eastern and Western Trauma Associations have published recommended screening algorithms based on systematic analysis, observational studies, and expert opinion.[29,41] Despite evidence for improved BCVI detection with aggressive screening algorithms, retrospective studies have demonstrated that up to 20% to 34% of BCVI patients still fail to meet established screening criteria.[7,23,30,46] This high percentage of potentially "missed" diagnoses indicates significant room for improvement of existing algorithms.

Craniofacial fractures have been repeatedly implicated as a cause of BCVI in numerous retrospective studies, particularly CAI, although the association has been poorly characterized.[1,2,12,46,47] The Western and Eastern Trauma Associations currently recommend screening all patients with cervical spine, basilar skull, and Le Fort II or III facial fractures for the presence of BCVI. This recommendation, however, was based on retrospective series, which established facial fracture diagnoses from review of International Classification of Diseases, Ninth Revision (ICD-9) codes,

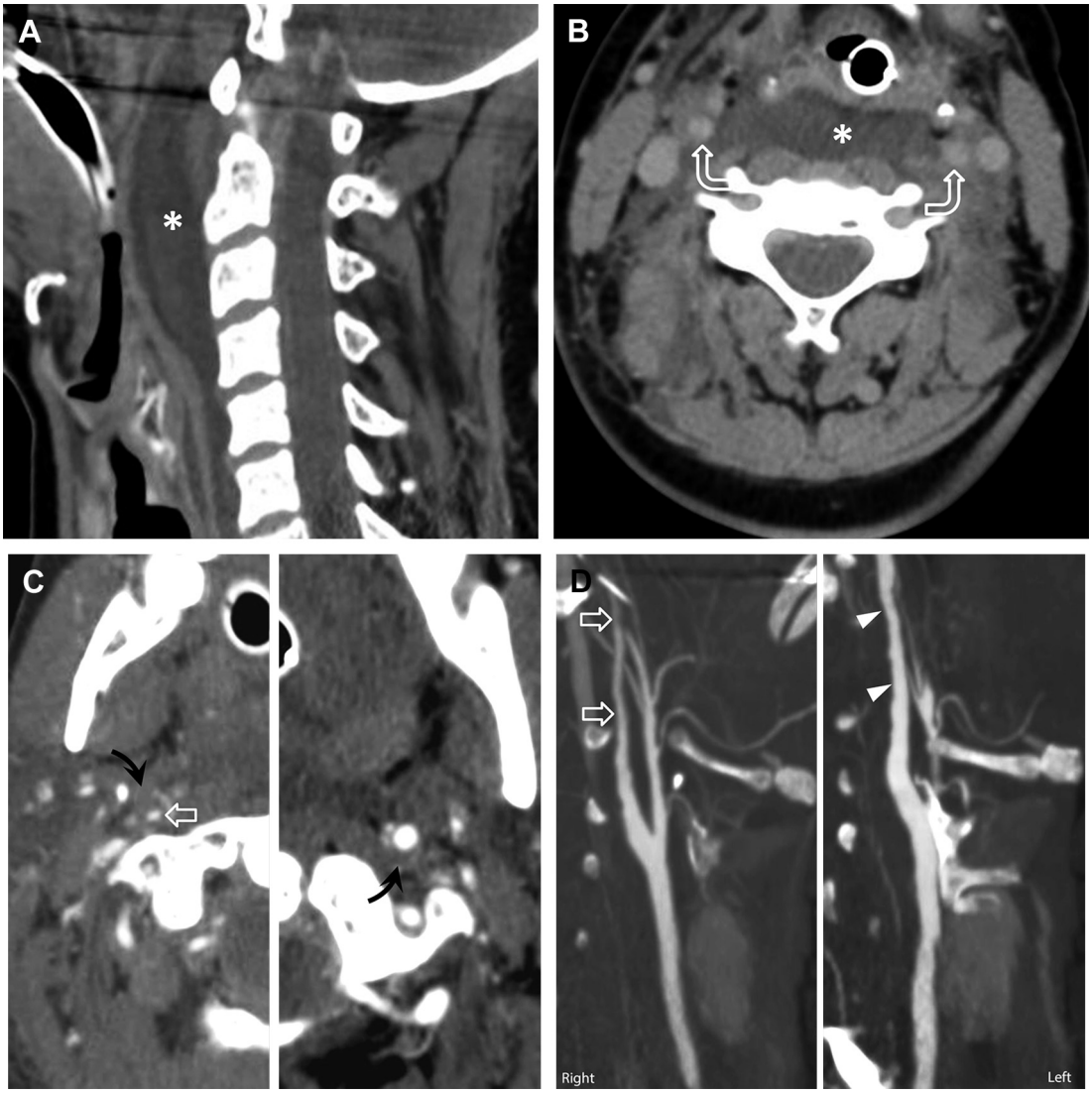


Fig. 12. Bilateral ICA dissections with "string" signs. Sagittal (*A*) and axial (*B*) CT images demonstrate a large retropharyngeal hematoma (*asterisk*), which extends laterally to the carotid spaces (*curved open arrows*), suggesting the possibility of traumatic BCVI and the need for a CTA. (*C*) Axial and (*D*) bilateral sagittal CTA images reveal intramural hematoma (*curved arrows*) causing severe right (Denver grade 2) (*open arrows*) and mild left (Denver grade 1) (*arrowheads*) luminal compromise (string sign).

rather than evaluation and classification of facial fractures by retrospective review of available clinical imaging.

Several small studies have attempted to address the association between facial fractures and BCAI but have lacked the power to perform statistical analysis.[48–50] A recent large retrospective series of 4398 patients with blunt mechanism facial fractures attempted to determine whether specific patterns of facial fracture are associated with an increased risk of carotid artery injury, and if so, whether they could be a valuable contribution to the screening criteria in place.[44] Results indicated that bilateral fractures of any "facial third," complex midface fractures including all Le Fort type injuries, and subcondylar mandibular fractures (especially in the setting of associated skull base fracture) conferred an increased risk of BCAI (see **Figs. 4**, **6**, and **12**).[44] Analysis of adding Le Fort type I injury to existing screening criteria demonstrated resultant increased sensitivity, although positive and negative predictive values remained unchanged.

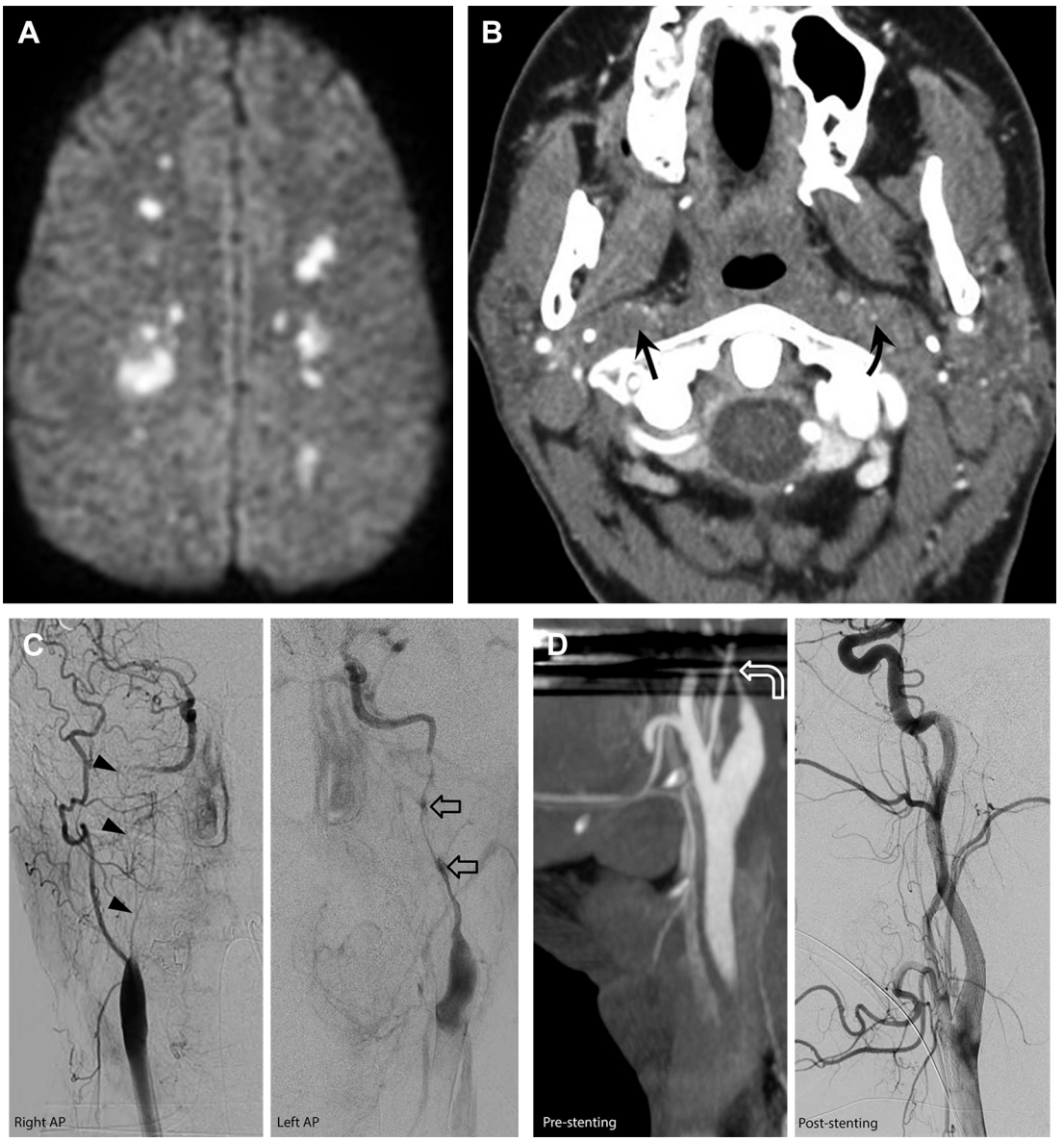


Fig. 13. Bilateral terminal zone infarcts due to ICA dissections with "string" signs. (*A*) Axial diffusion-weighted image demonstrates bilateral acute terminal zone infarcts compatible with hypoperfusion, raising the strong possibility of bilateral ICA injury. (*B*) Axial CTA image confirms a left carotid string sign (*curved arrow*) and possible right carotid occlusion (*arrow*). (*C*) Bilateral carotid angiograms demonstrate severe bilateral tapered ICA stenoses (Denver grade 2) due to ICA dissections. There is severe right ICA luminal compromise (string sign) (*arrowheads*) and an irregular left ICA stenosis with several focal dilatations (string-and-pearl sign) (*open arrows*). (*D*) Prestenting and poststenting images of the left ICA reveal marked improvement of the severe left ICA stenosis (*curved open arrow*).

A retrospective series of 1882 patients with craniocervical trauma similarly evaluated patterns of craniofacial fracture and association with BCVI, using relative risk (RR) calculations.[45] Mandible and midface fractures, when considered as a whole, only mildly increased the risk for BCVI (RR 1.4% and 1.3%, respectively). However, when subsets of midface fractures were considered, a markedly elevated risk was demonstrated for fractures of the sphenotemporal buttress (RR 3.9%) and orbital roof/rim fractures that extended into the central skull base (RR 2.8%). Interestingly,

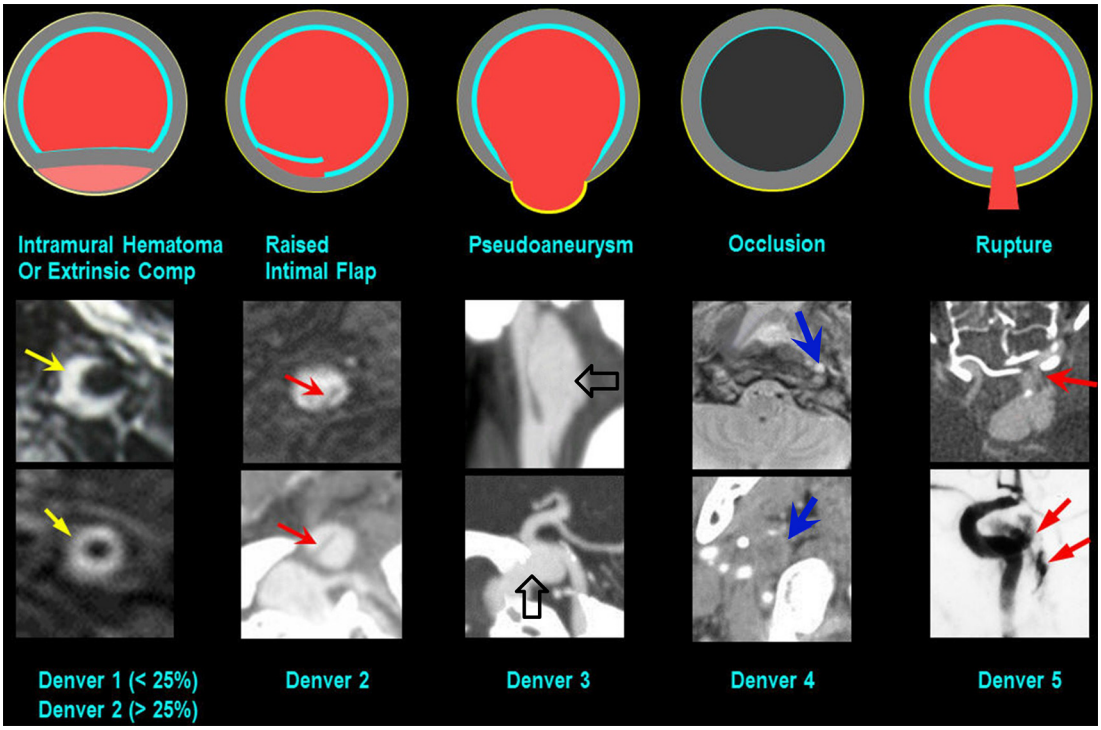


Fig. 14. Pathologic abnormality and classification of traumatic cerebrovascular injury. This diagram demonstrates the most common types of traumatic vascular injuries, their Denver grade, and their appearances on CTA, MRA, and angiogram images. Denver grade 1 is characterized by less than 25% luminal compromise by intramural or extramural hematoma. Grade 2 indicates either greater than 25% luminal compromise (*yellow arrows*) or a raised intimal flap (*red arrows*). Denver grade 3 is due to a pseudoaneurysm (*open arrows*), whereas grade 4 indicates a vessel occlusion (*blue arrows*). Free rupture and extravasation is a grade 5 injury (*red arrows*).

Le Fort fractures by themselves did not correlate with significantly higher risk unless they were associated with fractures of the carotid canal. Facial or skull base fractures of any type extending into the carotid canal, especially when associated with greater than 2-mm displacement, were associated with a much higher risk of BCVI (RR 5.2%). Of note, atlanto-occipital subluxation/dislocation conferred the highest risk, wherein 1 in 5 patients had vertebral artery dissection, pseudoaneurysm, vessel transection, or arteriovenous fistula.

Thus, emerging data suggest it is possible to stratify patients into higher BCVI risk categories by evaluating them with initial high-resolution head, maxillofacial, and cervical spine CT examinations, although further work in uncovering mechanistic relations between craniofacial injury patterns and BCAI is needed to maximize diagnostic yield and better guide prompt management.

Role of Imaging in BCVI Diagnosis

Imaging not only plays a vital role in identification of patients at high risk for BCVI that require further vascular workup but also in characterizing the injury itself (**Box 1**, **Tables 2** and **3**). Among other remaining key and somewhat controversial issues, debate continues in regards to the optimal diagnostic modality.

As discussed, conventional angiography is the gold standard for detecting vascular injury. Liberalized use of 4-vessel DSA has raised concerns, however, due to its invasive, time-consuming, and resource-intensive nature.[7,13,31,32,46] The risks of invasive DSA are not inconsequential, with the accepted complication rate (including stroke) ranging between 0.1% and 1.0% of angiographic studies performed to detect BCVI.[13,31]

In contrast to DSA, CTA can be performed in many hospitals without the necessity of transporting the patient outside the emergency department. With the dissemination of more advanced multidetector CT (MDCT) scanners (≥16 channels), CTA has emerged as the preferred diagnostic modality in most institutions.[51] Studies comparing 16-slice MDCT and conventional DSA have suffered from diagnostic inconsistencies, which in part appear related to the experience of the radiologists

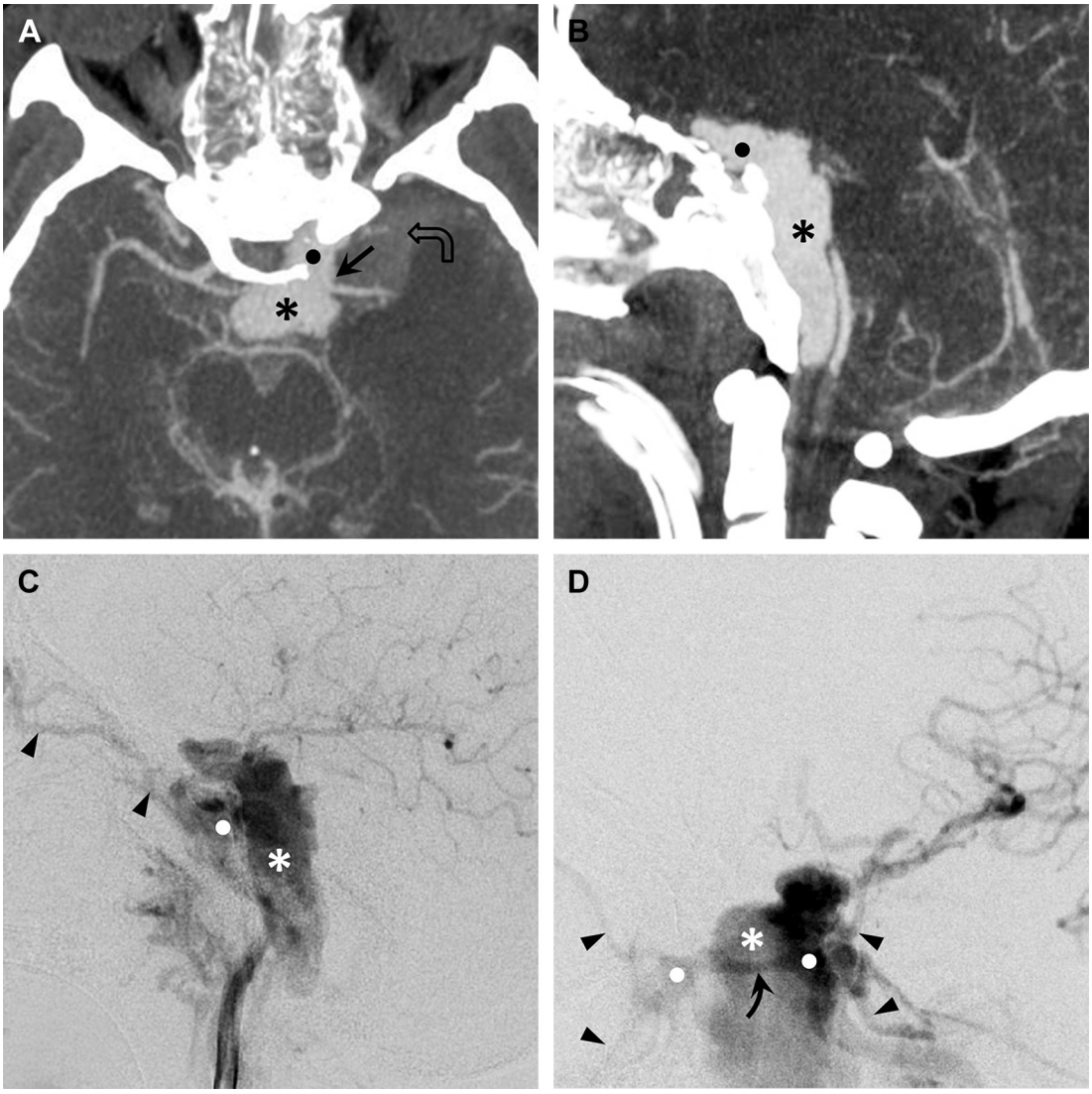


Fig. 15. ICA transection, free contrast extravasation, and carotid-cavernous fistula. (*A*) Axial and (*B*) sagittal MIP images from a CTA demonstrate extensive dilatation of the left cavernous sinus (•) as well as extravasation of contrast into a semi-contained prepontine hematoma (*asterisk*) and subarachnoid space (*curved open arrow*). The left ICA (*arrow*) is not well seen due to the ICA transection. (*C*) Lateral and (*D*) anteroposterior angiogram images confirm free extravasation of contrast into the prepontine cistern (•), early entry of contrast into the cavernous sinuses (*asterisk*), and filling of the superior and inferior ophthalmic veins (*arrowheads*). Note that contrast fills both of the cavernous sinuses via an anterior intercavernous vein (*curved arrow*).

interpreting the traumatic BCVI studies. Some investigators reported CTA sensitivities and specificity in detecting BCVI of up to 100%, although one study reported sensitivity of 64-slice MDCT at just 54%.[52–54] In contradistinction, Fakhry and colleagues[55] reported oversensitivity (high false positive rate) of CTA. Although CTA may be slightly less accurate than conventional angiography in detecting subtle intimal injuries, it provides rapid and accurate assessment of vascular injuries and is considered the study of choice for asymptomatic patients deemed at high risk of vascular injury. In patients with normal or equivocal findings on CTA, angiography may be warranted to definitively exclude an injury when clinical suspicion is high. More recently, whole-body MDCT protocols have been proposed to evaluate both vascular injury and cervical spine integrity with one spiral

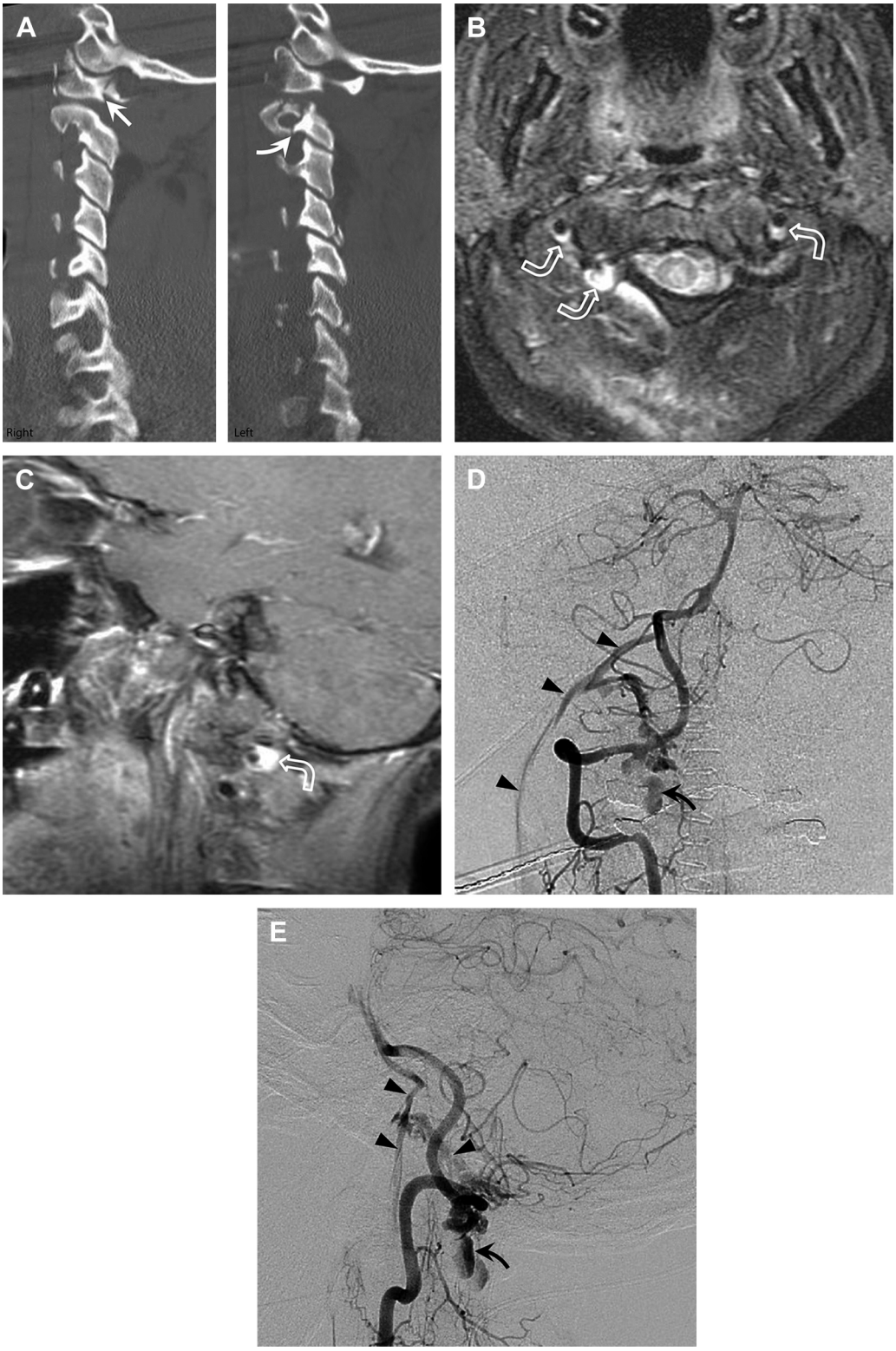


Fig. 16. Partial right vertebral artery transection and vertebral artery–venous fistula. (*A*) Bilateral sagittal CT images demonstrate fractures of the right C1 posterior arch (*white arrow*) and left C2 foramen transversarium (*curved white arrow*), both of which are known high-risk radiographic findings for BCVI. Axial (*B*) and right sagittal (*C*) T1-weighted fat-suppressed contrast-enhanced MR images reveal areas of intramural contrast extravasation (*curved white arrows*) at sites of vascular injury. Lateral (*D*) and anteroposterior (*E*) angiogram images reveal early entry of contrast into the perimedullary venous plexus (*arrowheads*), confirming an arteriovenous fistula. Additionally present are free contrast extravasation and loculated collections of contrast (*curved arrow*) within the paravertebral soft tissues, representing a Denver grade 5 injury.

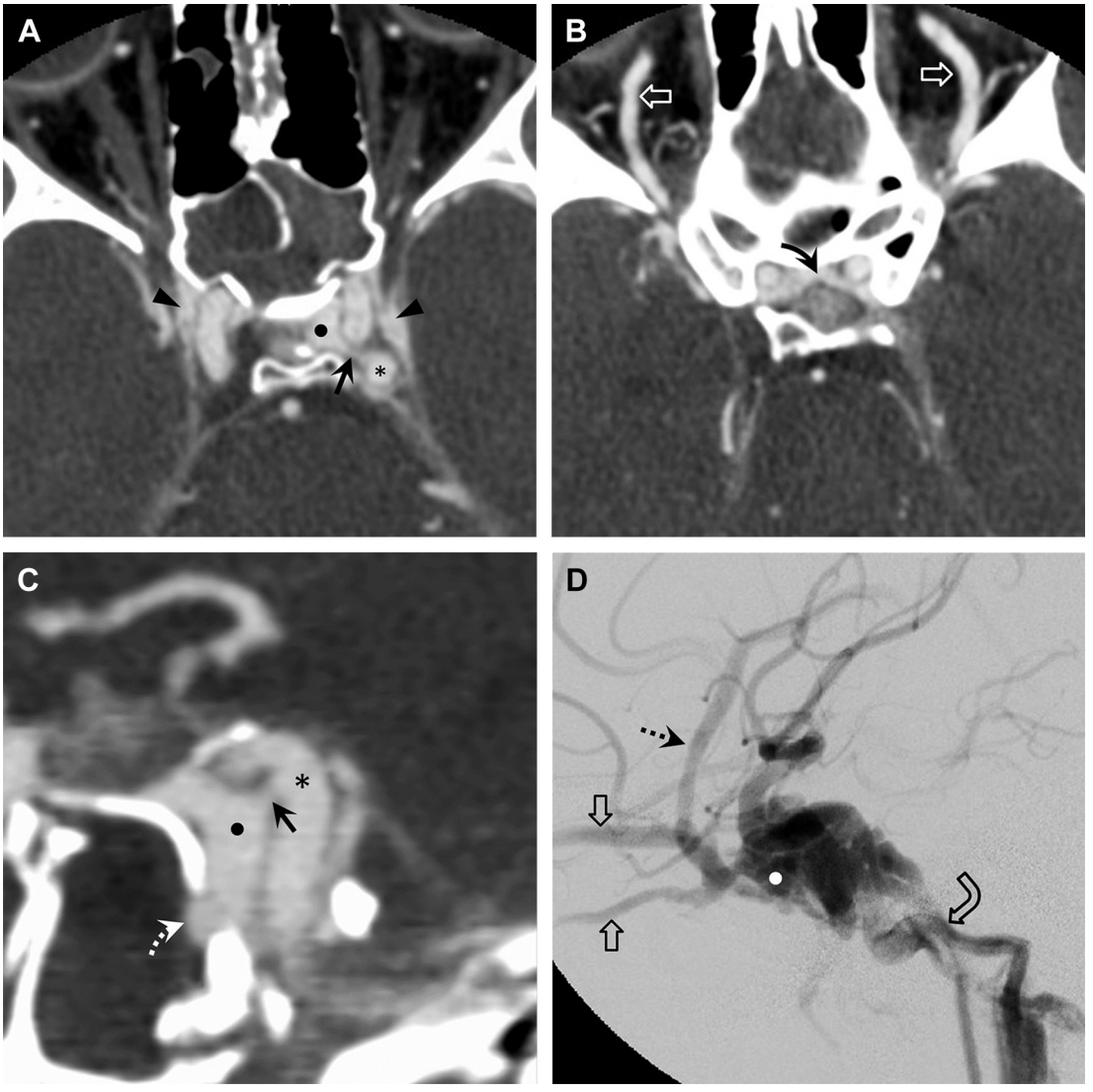
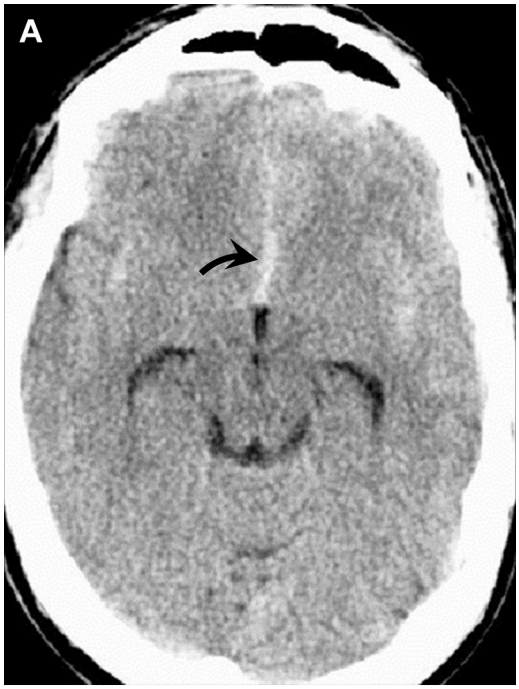


Fig. 17. Traumatic carotid-cavernous fistula. (*A*, *B*) Axial CTA images demonstrate early filling and dilatation (arterialization) of the cavernous sinuses (*arrowhead*) due to fistulous connection (*arrow*) between the left ICA (*asterisk*) and a large cavernous sinus venous varix (•). There is dilatation of the superior ophthalmic veins (*open arrows*). Bilateral cavernous sinus involvement is due to a large patent anterior intercavernous vein (*curved arrow*). (*C*) Sagittal 2D reconstructed CTA image confirms the fistulous connection (*arrow*) between the ICA (*asterisk*) and venous varix (•). The patient is at very high risk of future epistaxis due to extension of the venous varix through a fracture into the sphenoid sinus (*curved dashed arrow*). (*D*) Lateral angiogram confirms early entry of contrast into the cavernous sinus (•), ophthalmic veins (*open arrows*), sphenoparietal sinus (*dashed arrow*), and inferior petrosal sinus (*curved open arrow*).

acquisition and contrast dose.[56] The diagnostic quality of this technique has not yet been proven comparable to individual CTA protocols, however.

MRA with MR imaging is a capable alternative to CTA, with both relative advantages and disadvantages. Disadvantages of MRA include the requirement of patient transportation outside the emergency department, a somewhat time-consuming screening process of the patient for contraindications to the magnet, and greater difficulty of monitoring injured patients inside the bore of the magnet. In addition, MRA may not be available in some centers; high-quality images are more difficult to obtain, and the interpretation of the study requires more experience. At least one prospective study demonstrated MR imaging in combination with MRA to have excellent sensitivity and specificity in detecting carotid artery



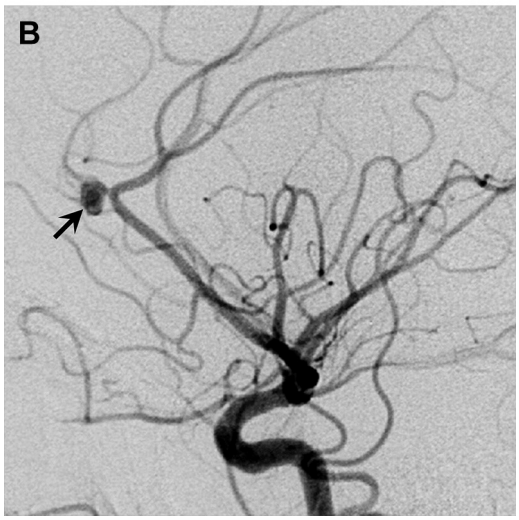


Fig. 18. Traumatic pericallosal aneurysm. (*A*) Axial CT images demonstrate extensive posttraumatic subarachnoid hemorrhage within the anterior interhemispheric fissure (*curved arrow*), greater than expected considering the degree of the patients' other intracranial injuries. A high index of suspicion for traumatic CVI is indicated and additional workup is needed. (*B*) Lateral angiogram depicts a traumatic pericallosal aneurysm (*arrow*). Posttraumatic aneurysms in this location likely result from partial avulsion of cortical arterial branches from the pericallosal artery due to excessive shift of the frontal lobes in relation to the more fixed corpus callosum.

dissection (95% and 99%, respectively) (see **Figs. 3**, **5**, **10**, and **12**) compared with conventional DSA, although it fared poorly with VAI detection (sensitivity and specificity of 60% and 58%, respectively).[57] Numerous other studies report less favorable MR imaging/MRA results.[23,31,57] MRA has not gained general acceptance as the preferred screening modality, but may be the study of choice for the evaluation of BCVI patients with symptoms suggestive of trauma-induced stroke (see **Fig. 1**). MR imaging and MRA are especially useful for vascular injuries causing ischemic complications, identifying coexisting infarcts, and evaluating for perfusion defects (see **Figs. 1**, **3**, **8**, and **11**).

Duplex ultrasonography has a limited role in the assessment of cerebrovascular injuries because of its limitations in visualizing the entire cerebrovascular system, its inability to characterize direct signs of injury in most cases, and poor overall sensitivity and specificity. Consequently, ultrasound is not recommended as a screening tool for possible BCVI.

BCVI Treatment and Follow-Up

The development of the Denver grading scale enabled investigation into prognostic and treatment implications associated with the varying degrees of traumatic cerebrovascular injury.[30] The primary management strategies for BCVI have included observation, surgical repair, antithrombotic drugs, and endovascular therapy. Although consensus on optimal patient treatment and follow-up is lacking, decisions and recommendations typically take into consideration patient symptoms and injury location/grade (anatomic description).

BCVI has been historically associated with high morbidities and mortalities when untreated. Management through observation alone is therefore not recommended, unless there are significant comorbidities contraindicating more aggressive treatment strategies.

Given the pathophysiology of intimal injury and subsequent platelet aggregation in BCVI, it is not surprising that antithrombotic agents have been used in an attempt to improve patient outcomes. Although the literature reveals some contradictory results, the overall body of evidence has indicated that the use of antithrombotic agents in BCVI can significantly improve mortality and prevent permanent neurologic deficits in patients.[1,13,32] No direct, controlled comparison studies of heparin versus antiplatelet agents have been performed to demonstrate superiority in outcome, although one of several subgroup analyses showed slight

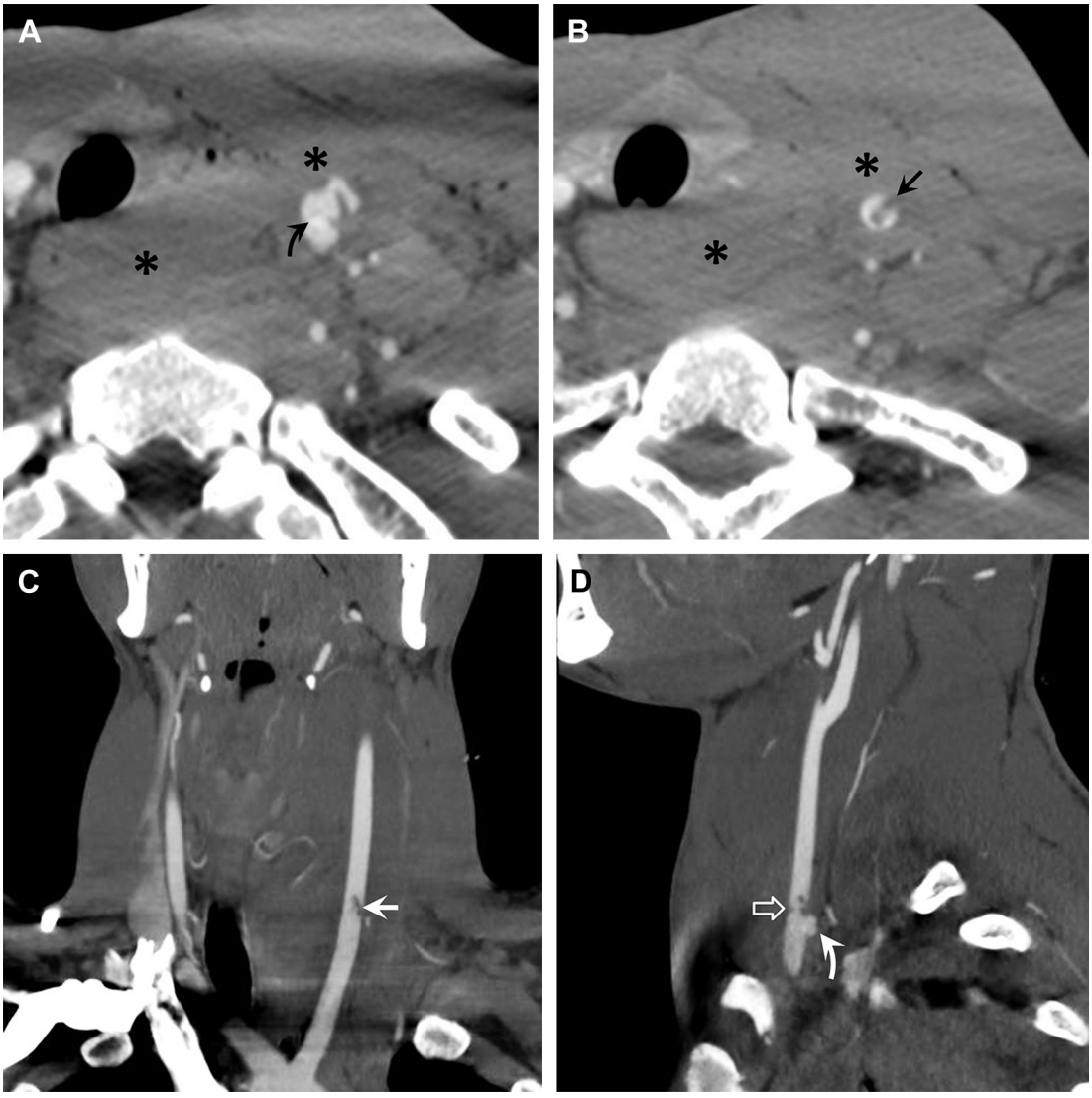


Fig. 19. Incomplete left common carotid artery transection. (*A*, *B*) Axial CTA images demonstrate extensive retrotracheal and carotid space hematoma (*asterisks*) with traumatic disruption of the left common carotid artery and a large pseudoaneurysm (*curved black arrow*). A large intraluminal dissection flap (*black arrow*) is also present. (*C*) Coronal and (*D*) sagittal 2D reconstructed CTA images confirm near complete vessel transection (*open arrow*), pseudoaneurysm formation (*curved white arrow*), and a large intimal flap (*white arrow*).

improvement in BCVI neurologic outcome with heparin treatment.[7] Recent Eastern Association for the Surgery of Trauma management guidelines advocate treatment of grade 1 and grade 2 injuries with either heparin or antiplatelet therapy, citing equivalent efficacy.[29] Anticoagulation is not without risk. Serious bleeding complications have been reported, particularly in patients with documented intracranial hemorrhage before initiation of therapy.[7] Conservative anticoagulation protocols have been recommended, although no optimal regimen or duration of therapy has been established.[23,29]

Interestingly, evidence suggests it is the lower grade lesions (Denver grading scale I and II) that are the most dynamic, with 8% of grade I and 43% of grade II BCVI lesions progressing on follow-up DSA imaging in the 7 to 10 days following the injury.[31] Approximately 60% of patients with grade I and II injuries required change in management. These findings give credence to routine follow-up of such lesions, either by

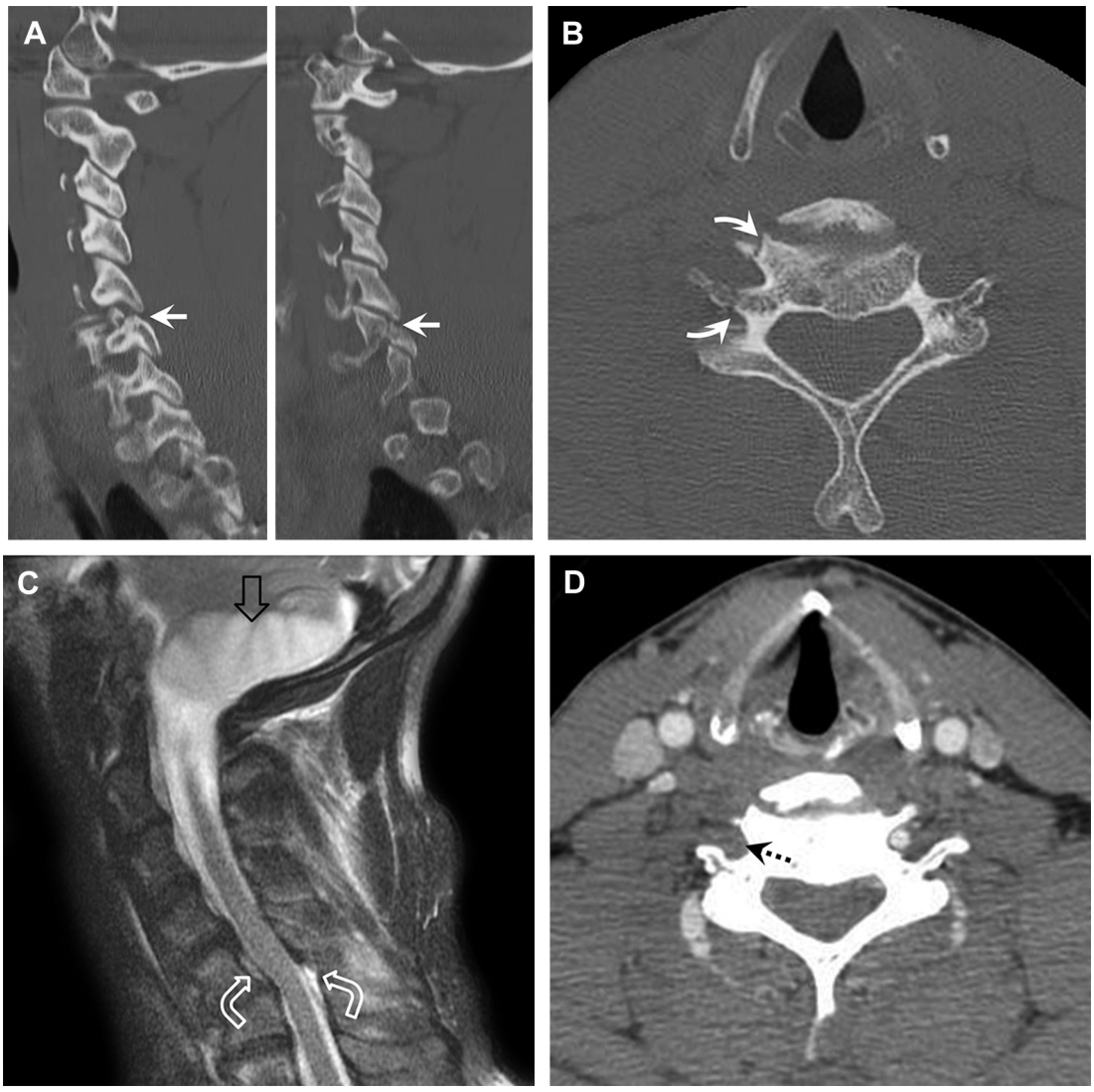


Fig. 20. Right vertebral artery occlusion and "asymptomatic" posterior inferior cerebellar artery infarct. Sagittal (*A*) and axial (*B*) CT images of a chiropractor who sustained a motor vehicle accident demonstrate fractures (*arrows*) of the right superior articular facet and lateral mass of C5. There is involvement of the C5 foramen transversarium (*curved arrows*). There is no subluxation on the sagittal images. (*C*) A sagittal T2-weighted MR imaging scan was obtained to rule out ligamentous injury. There is new C5–C6 vertebral subluxation and evidence of ligamentous injury (*curved open arrows*). An "asymptomatic" posterior inferior cerebellar artery infarct (*open arrow*) is present. (*D*) Axial CTA image confirms occlusion of the right vertebral artery (*dashed arrow*).

conventional DSA or by noninvasive (CTA, MR/MRA) imaging.

Complete vessel occlusions (grade IV lesions) may recanalize as part of their natural history, although they do not typically do so in the early postinjury period. Despite a high risk of stroke in complete vessel occlusion, anticoagulation has been demonstrated to improve outcomes in this population.[30] The optimal agent, duration, and end point in therapy have not been established for this group.

Grade III lesions (dissecting aneurysm) place the patient at risk for a thromboembolic event and progression to vessel occlusion or rupture. Carrying the highest rate of mortality, grade V lesions are devastating and intervention is often preempted by patient demise (see **Fig. 15**).[30] These higher grade lesions have been found to rarely heal with

Table 1
BCVI Denver grading scale

Injury Grade	Description
I	Luminal irregularity or dissection with <25% luminal narrowing
II	Dissection or intramural hematoma with ≥25% luminal narrowing, intraluminal thrombus, or raised intimal flap
III	Pseudoaneurysm
IV	Occlusion
V	Transection with free extravasation

From Biffl WL, Moore EE, Offner PJ, et al. Blunt carotid arterial injuries: implications of a new grading scale. J Trauma 1999;47(5):845–53; with permission.

antithrombotic therapy alone; therefore, surgical management has been traditionally advocated. In the last decade, however, endovascular therapy has been used much more frequently, because of the number of BCVIs that are not amenable to open surgical treatment.[11] DiCocco and colleagues[11] advocate aggressive endovascular therapy for grade II, III, and V lesions, citing after-diagnosis stroke rates similar to that of medical therapy (4%), despite treating lesions associated with much higher stroke risks. Clear risk-benefit ratios for endovascular treatments have not been well established, and concerns have been raised regarding stent-related complications and the need for expensive dual antiplatelet therapy following placement of drug-eluting stents.[58]

Box 1
BCVI imaging pearls and pitfalls

Pearls

- There is a high rate of BCVI multifocality (up to 43%).
- CTA has emerged as the preferred diagnostic modality for BCVI in most institutions.
- Vessel injury may be remote from other signs of trauma; thus, imaging from the aortic arch through the circle of Willis is essential.
- Mural hematoma may mimic the appearance of atherosclerotic plaque on CTA; however, in contradistinction to atherosclerotic disease, BCVI will typically spare the carotid bulb.

Pitfalls

- Multiplanar 2D and 3D reformations may obscure vessel injury; systematic evaluation of thin-section axial CT and MR imaging source images for signs of vascular injury is crucial.
- On noncontrast TOF MRA, BCVI manifested as subacute intramural hematoma (with its short T1 values) can be mistaken for flow. Phase contrast and contrast-enhanced MRA will more clearly differentiate flow from the adjacent intramural hematoma.
- On fat-suppressed T1-weighted MR imaging, the relative isointense appearance of acute (<7 days) and chronic (>2 months) hematoma blends in with surrounding tissues, making it difficult to detect.
- Dephasing and signal dropout on TOF MRA images caused by turbulent flow in the horizontal petrous segment of the ICA can mimic intraluminal thrombus or dissection.

Table 2
Imaging screening criteria for BCVI (asymptomatic patients)

Screening Criteria Adapted from Biffl et al[41]	Denver Modification of Screening Criteria[41]	Memphis Screening Criteria[10]

• Displaced midface or complex mandibular fracture (in setting of cervical rotation/hyperflexion or extension)
• Cervical vertebral body fracture
• Basilar skull fracture involving the carotid canal
• Fracture in proximity to ICA or VA
• Diffuse axonal injury
• Anoxic brain Injury
	• Displaced Le Fort II or III fracture
	• Cervical spine fracture with:
	  ○ Subluxation
	  ○ Extension into transverse foramen
	  ○ C1–C3 involvement
	• Basilar skull fracture with carotid canal extension
	• Diffuse axonal injury
	• Anoxic brain injury
		• Le Fort II or III fracture
		• Cervical spine fracture
		• Skull base fracture involving the foramen lacerum

Table 3
Craniofacial trauma radiologic risk factors for BCVI

Mundinger et al[44]	Alsheik et al[45]
• Bilateral fractures of any "facial third" • Complex midface fracture (Le Fort I, II, III) • Subcondylar mandibular fracture (in the setting of associated skull base fracture)	• Sphenotemporal buttress fracture • Orbital roof/rim fracture with central skull base extension • Facial or skull base fractures with carotid canal extension (especially with >2 mm displacement) • Atlanto-occipital subluxation/dislocation (1 in 5 patients had VA injury)

SUMMARY

Significant recent progress has been made in the recognition, screening, diagnosis, and treatment of BCVI. Although controversy still exists as to optimal screening algorithms and best diagnostic modality, the vital and growing role of noninvasive imaging in identifying patients at high risk for BCVI and in characterizing the injury itself has been clearly established. There has been promising early work in stratifying BCVI patients into risk categories by initially evaluating them with high-resolution head, maxillofacial, and cervical CT examinations with the ultimate goal of maximizing diagnostic yield and enabling prompt initiation of therapy. Further work is needed, however, to delineate the mechanistic relationship between craniofacial fractures and BCVI.

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