Imaging for Blunt Carotid and Vertebral Artery Injuries

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KEYWORDS

• Cerebrovascular • Carotid artery • Vertebral artery • Injury
• Trauma

Over the past decade, multiple studies have provided the scientific rationale to promote the early identification and treatment of blunt carotid artery injuries (CAIs) and blunt vertebral artery injuries (VAIs), collectively known as blunt cerebrovascular injuries (BCVIs).[1–5] Initially BCVIs were thought to have unavoidable, devastating neurologic outcomes, but several reports suggested that anticoagulation improves neurologic outcome in patients suffering ischemic neurologic events.[6–9] Further study elucidated a latent period of blunt carotid and vertebral injuries; this asymptomatic period, before the onset of stroke, permits early identification of a patient's BCVIs and institution of treatment. Screening protocols, based on patient injury patterns and mechanism of injury, have been developed to identify high-risk patients so that appropriate imaging may be performed early in the postinjury period.[10] Current studies suggest that early antithrombotic therapy in asymptomatic patients with BCVIs reduces stroke rates and prevents neurologic morbidity[1–3,5,9–12]; hence, identification of injuries with appropriate imaging is paramount.

HISTORICAL PERSPECTIVE

BCVIs were first recognized over 30 years ago, but the majority presented with symptoms of neurologic ischemia.[13–18] Crissey and Bernstein[13] postulated 4 fundamental mechanisms of injury: direct blow to the neck, hyperextension with contralateral rotation of the head, laceration of the artery by adjacent fractures involving the sphenoid or petrous bones, and intraoral trauma. The most common mechanism causing CAIs is hyperextension resulting from the stretching of the carotid artery over the lateral articular processes of C1-C3.[19] VAIs are likely a combination of direct injury, which is caused by

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associated fractures of the vertebrae involving the transverse foramen through which the artery courses, and hyperextension-stretch injury, which is caused by the tethering of the vertebral artery within the lateral masses of the cervical spine. Regardless of mechanism, there is intimal disruption of the carotid or vertebral artery. This intimal tear becomes a nidus for platelet aggregation that may lead to emboli or vessel occlusion.

Although the initial focus of BCVIs management was recognizing the injury and treating the devastating neurologic sequelae, subsequent efforts have been directed at diagnosing and treating these injuries during the "silent period," before the onset of stroke. Some patients with BCVIs may present with symptoms of cerebral ischemia within an hour of injury; early identification and treatment in these patients is difficult if not impossible. However, most patients with BCVIs exhibit a latent period between their original injury and the onset of stroke. This time frame range from hours to up to 14 years, but the majority seems to develop symptoms within 10 to 72 hours.[1,2,5,6,18–21] Diagnosing BCVIs during this "silent period" affords the opportunity for treatment before and to prevent neurologic sequelae.

Aggressive screening for BCVIs was initially suggested in the mid-1990s[9,10] after recognizing that specific patterns of injuries were associative.[6,7,22] A recently published report questioned the utility of such an aggressive screening approach,[23] whereas other studies have a screening yield of more than 30% in high-risk populations.[3–5,11] Indications for imaging have been proposed that identify a high-risk population of patients based on injury patterns.[1,3,5,11,19,24,25]

INDICATIONS FOR IMAGING

The initial screening protocol initiated in Denver in 1996 was relatively liberal, in an attempt to include all potential injury mechanisms and patterns.[10] The screening criteria included (1) an injury mechanism compatible with severe cervical hyperextension or rotation or hyperflexion, particularly if associated with displaced or complex midface or mandibular fracture; (2) closed head injury consistent with diffuse axonal injury of the brain; (3) near-hanging injury resulting in cerebral anoxia; (4) seat belt abrasion or other soft tissue injury of the anterior neck resulting in significant cervical swelling or altered mental status; (5) basilar skull fracture involving the carotid canal; and (6) cervical vertebral body fracture or distraction injury, excluding isolated spinous process fracture. A multivariate analysis of injury mechanisms and patterns was performed to identify high-risk factors, and 4 injury patterns were identified that were independent predictors of CAIs: Glasgow Coma Score (GCS) less than 6, petrous bone fracture, diffuse axonal brain injury, and LeFort II or III fracture.[26] Patients with any of these risk factors had a risk of 41% for CAIs. In those with all 4 injuries, the risk of CAI increases to 93%. In this same study by Biffl and colleagues, the only significant risk factor for VAI was cervical spine injury. Subsequent analysis of VAIs by Cothren and colleagues[27] found that nearly all cervical spine injury–related VAIs were associated with subluxations, foramen transversarium fractures, and fractures involving C1-C3. Based on these studies, a high-risk patient population has been identified that should undergo imaging to exclude BCVIs (**Box 1**).[24–26] However, in early series, up to 20% of patients with BCVI had none of these injuries,[26] with screening performed based on clinical suspicion of injury. With the improved accuracy of noninvasive screening modalities, there is a tendency to liberalize screening to capture all injuries, rather than try to restrict screening to the highest-risk groups.[28] These groups may include patients with mandible fractures, those with upper thoracic trauma combined with cranial injuries, and the pediatric population. To date, there have not been any large-scale analyses to determine the yield of such protocols.

Box 1
Denver screening criteria for BCVIs

Signs/Symptoms of BCVIs

Arterial hemorrhage from neck or nose or mouth

Cervical bruit in patients younger than 50 years

Expanding cervical hematoma

Focal neurologic deficit (transient ischemic attack, hemiparesis, vertebrobasilar symptoms, Horner syndrome)

Neurologic examination incongruous with head computed tomographic (CT) scan findings

Stroke on CT scan or magnetic resonance imaging

Risk Factors for BCVIs

High-energy transfer mechanism with

LeFort II or III fracture

Cervical spine fracture patterns: subluxation, fractures extending into the transverse foramen, fractures of C1-C3

Basilar skull fracture with carotid canal involvement/petrous bone fracture

Diffuse axonal injury with GCS less than 6

Near hanging with anoxic brain injury

Clothesline type injury or seat belt abrasion with significant swelling, pain, or altered mental status

DIAGNOSTIC IMAGING

A major focus of the recent literature on BCVIs has been the optimal screening diagnostic test. Four-vessel arteriography has long been considered the gold standard to diagnose BCVIs. Undoubtedly, many clinicians question the need for subjecting patients to angiography. Angiography is invasive, labor intensive, and costly; risks include complications related to catheter insertion (1%–2% hematoma, retroperitoneal bleeding, arterial pseudoaneurysm), contrast administration (1%–2% renal dysfunction, allergic reaction), infection, exposure to radiation, and stroke (<1%).[2,5] In addition, if angiography is not available at smaller hospitals, the patient requires emergent transfer for definitive evaluation.

Duplex ultrasonography (US) is widely used for imaging the extracranial carotid arteries for atherosclerotic disease; however, experience in diagnosing BCVIs is limited. In a multicenter review, US had 86% sensitivity for identifying internal carotid artery (ICA) injuries.[6] In that population of patients, the lesions missed by US were located at the base of the skull. Because most CAIs involve the distal ICA at or near the base of the skull, this is conceptually a major weakness of this imaging modality. Likewise, artifact from the bony canal encasing the vertebral artery may obscure a low-grade injury. Furthermore, although US can provide indirect evidence of injuries by detecting turbulence or other blood flow disturbances, these findings are not routinely seen in patients with stenoses less than 60%. In a recent series of over 1400 blunt trauma patients, the overall sensitivity of US was just 39%, with US missing 8 injuries that resulted in stroke.[29] Consequently, US is not recommended for BCVI screening.

Magnetic resonance angiography (MRA) seemed to be an attractive alternative to angiography (**Fig. 1**). MRA is noninvasive, does not require contrast administration, and detects cerebral ischemia earlier than CT scanning. Several reports advocate

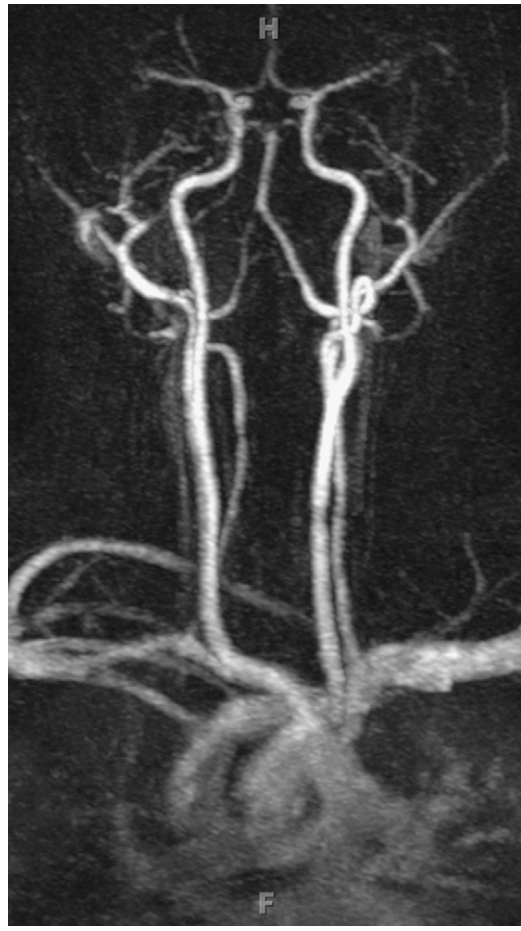

Fig. 1. MRI of the carotid and vertebral arteries.

use of MRA to diagnose BCVIs.[30–32] However, several trials, including those from Denver and Memphis, have documented poor sensitivity and specificity of MRA.[2,33,34] In addition, with issues of timely availability and incompatibility of equipment, MRA is not considered a reliable or optimal screening test for BCVIs.

CT angiography (CTA) has emerged as the preferred screening test for BCVIs. In addition to being a noninvasive imaging modality, most patients undergoing screening for BCVIs have indications for CT scanning of other regions. Hence, imaging can often be accomplished with only one "road trip." With high-speed scanners, the duration of imaging has been markedly reduced, as has the amount of contrast required, with dye loads being less than that used for conventional angiography. In addition, the use of coronal and sagittal reconstructions permits identification of injuries in 3 dimensions, with correlation to associated spine or skull trauma. CTA interpretation may be limited by streak artifacts from foreign bodies, motion artifacts, and beam hardening by dense venous contrast (**Fig. 2**). Optimal identification of injuries may be associated with the experience of the radiologist, with subtle findings otherwise missed (**Fig. 3**). The accuracy of early generation 1- to 4-slice CTA was poor,[2,33] with sensitivities between

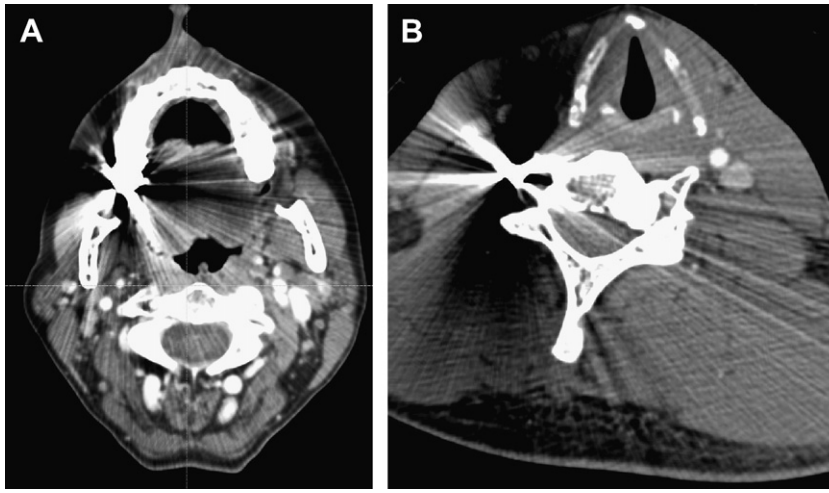

Fig. 2. Streak artifacts from foreign bodies such as dental work (*A*) and bullet fragments from prior penetrating trauma (*B*) may limit CTA interpretation.

47% and 68% and specificity of 67%. BCVI identification improved with the introduction of multidetector-row CTA.[28,35–37]

Four published studies have evaluated the accuracy of 16-slice CTA compared with arteriography. Eastman and colleagues[38] evaluated 162 patients with CTA, of whom 146 agreed to angiography. Reported screening yield was 28%, with an overall incidence of BCVIs of 1.25%. This study reported 100% sensitivity of 16-slice CTA for CAIs, and 96% sensitivity for VAIs, with 1 false-negative CTA of a grade 1 injury. The Harborview group performed arteriography on 82 patients who had had a normal screening CTA and initially found that CTA missed 7 BCVIs, for a negative predictive

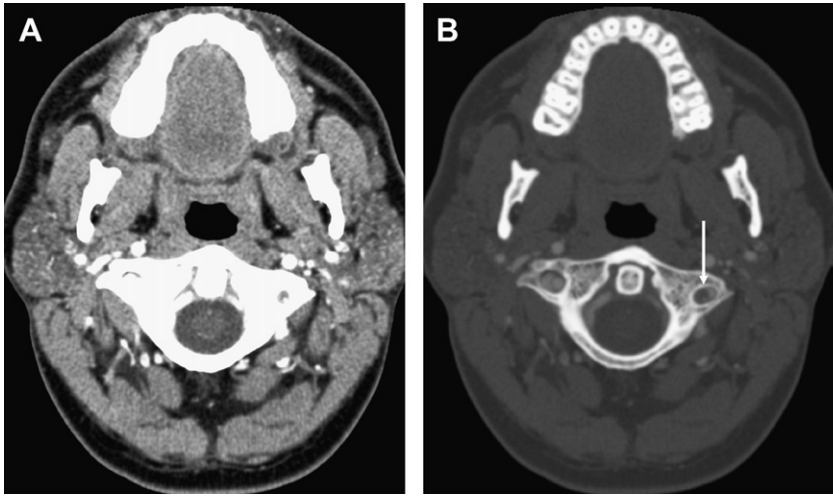

Fig. 3. (*A, B*) Bone windows on CTA often are more optimal in diagnosing a vertebral artery injury (*arrow in B*).

Box 2
Denver grading scale for BCVIs

Grade 1: irregularity of the vessel wall or a dissection/intramural hematoma with less than 25% luminal stenosis

Grade 2: intraluminal thrombus or raised intimal flap is visualized, or dissection/intramural hematoma with 25% or more luminal narrowing

Grade 3: pseudoaneurysm

Grade 4: vessel occlusion

Grade 5: vessel transection

value of 92%.[39] However, retrospective review of the CTA images found that the injuries were evident in 6 of the 7 patients and that the seventh patient's abnormality was most likely not traumatic in origin. Although selection bias exists in this study's design, it does illustrate the importance of experience in identifying BCVIs on a CTA; all missed injuries occurred in the first half of the study period. Two studies offer a note of caution in adopting CTA as the preferred imaging modality. Malhotra and colleagues[40] screened 119 patients with 92 undergoing confirmatory angiography; they reported a 43% false-positive and 9% false-negative rate for CTA. However, as in the series of Utter and colleagues,[39] the inaccuracy of CTA seemed to be related in large part to the radiologists' inexperience, as all of the missed BCVIs occurred in the first half of the study period. In the second half of the study, the sensitivity and negative predictive value of CTA was 100%. Each of these studies[39–40] recognizes that injuries in the region of the skull base seem to be the most difficult to identify, underlining the importance of carefully examining this high-risk region. The final study to evaluate CTA and arteriography by Goodwin and colleagues[41] reported the worst results for high-resolution CTA. They report the sensitivity for 16-slice CTA to be 29% and 64-slice CTA to be 54%. The authors acknowledge that the impact of the interpreting radiologist as a contributing factor has not been evaluated in any studies to date. Without quality control it is difficult to understand how best to interpret this study's impact on screening options for BCVIs. Conversely, a preliminary report by Fakhry and colleagues[42] indicates that CTA may be oversensitive in diagnosing BCVIs.

Overall, it seems that 16-slice (or more) CTA is reliable for screening for clinically significant BCVIs but that the accuracy diminishes with fewer detector rows. If CTA is not available, conventional angiography is the gold standard. In patients with

Table 1
Stroke rate by blunt cerebrovascular injury grade

	Grade of Injury	Stroke Rate by Grade
CAI	1	3%
	2	14%
	3	26%
	4	50%
	5	100%
VAI	1	6%
	2	38%
	3	27%
	4	28%
	5	100%

a normal CTA but high clinical suspicion or an equivocal CTA, angiography may be warranted to definitively exclude an injury.

INJURY GRADING SCALE

With the recognition of varied luminal irregularities comprising BCVIs (dissection, pseudoaneurysms, occlusion, and transection), was the identification of disparate outcomes.[6,9] An injury grading scale was developed[19] not only to provide an accurate

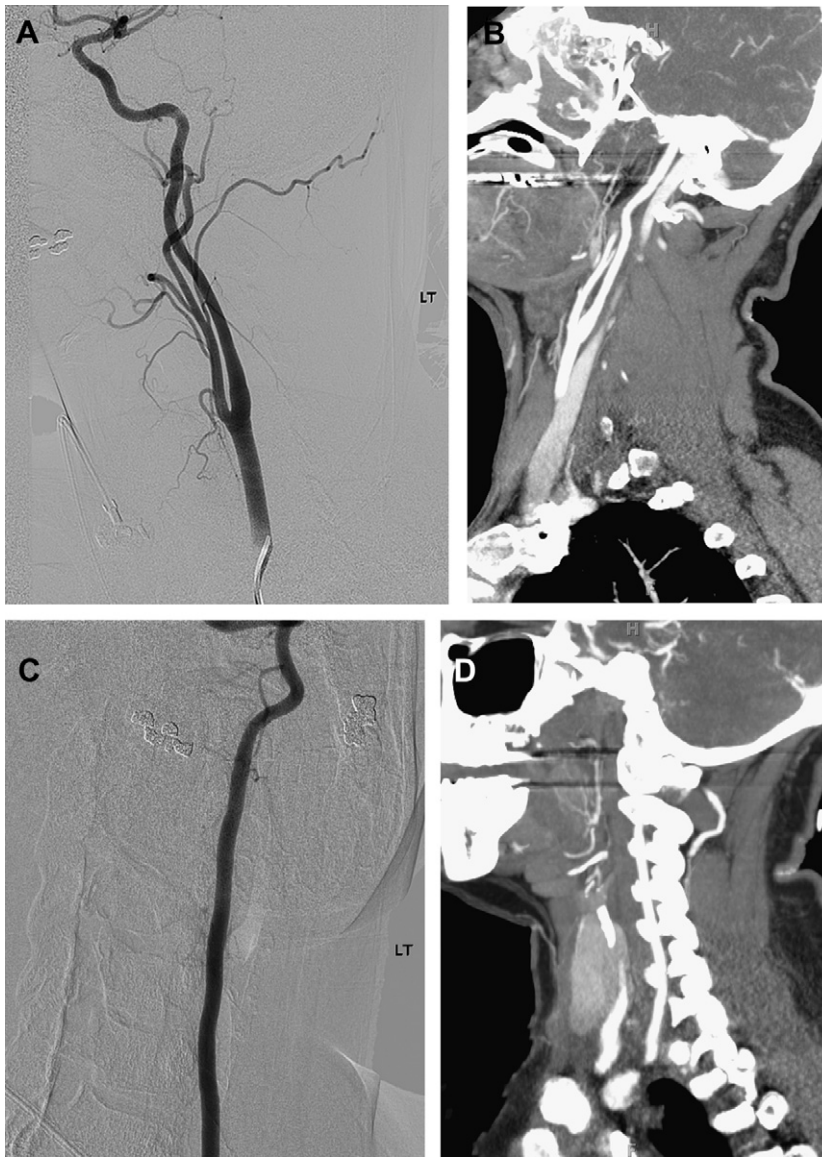

Fig. 4. Normal vasculature of the carotid artery (*A, B*) and vertebral artery *(C, D)* on angiography and CTA imaging.

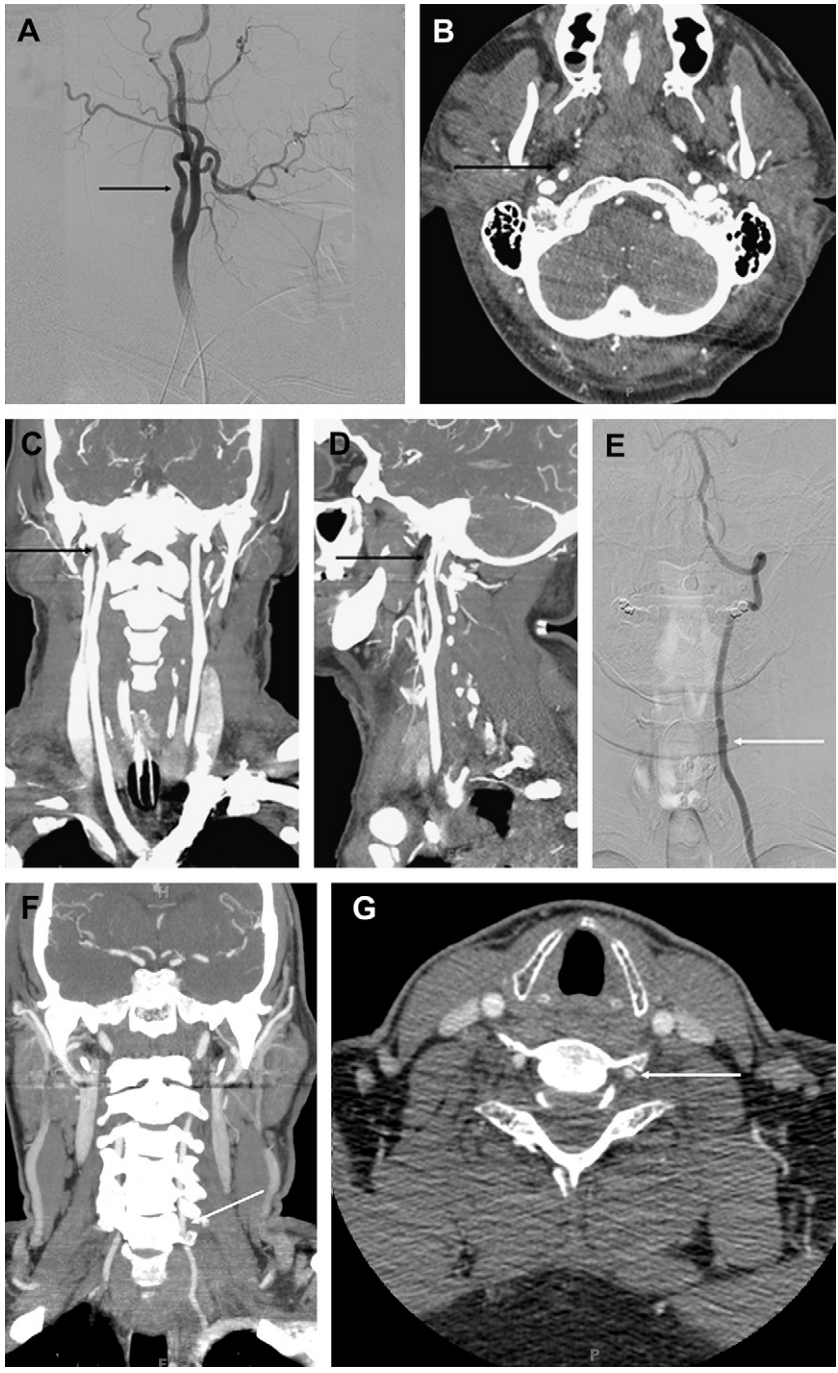

Fig. 5. Grade 1 injury to the carotid artery (*A–D*) and vertebral artery (*E–G*).

description of the injury but also to define stroke risk by injury grade (**Box 2**). Untreated injuries have an overall stroke rate of 21% to 64%[1,10,11]; CAIs have increasing stroke rate by increasing grade, whereas VAIs tend to have a more consistent stroke rate of approximately 20% for all grades of injury (**Table 1**).[2] When reviewing a patient's CTA or angiogram, recognition of normal vasculature is important (**Fig. 4**). A grade I injury is an intimal irregularity or dissection with less than 25% luminal narrowing (**Fig. 5**). Grade 2 injuries are dissections or intramural hematomas with greater than or equal to 25% luminal narrowing, intraluminal clot, or a visible intimal flap (**Fig. 6**). Pseudoaneurysms are defined as a grade 3 injury (**Fig. 7**). A complete occlusion is grade 4 injury (**Fig. 8**), and transection with active extravasation is grade 5 injury (**Fig. 9**).

TIMING OF IMAGING

All patients with indications for screening, and no contraindications to antithrombotic therapy, undergo imaging as soon as possible. For patients who do not undergo CTA of the neck on initial trauma imaging, repeat imaging should be performed as soon as possible. In labile patients, or those at risk for contrast-induced nephropathy, one may delay imaging if the patient has a contraindication to antithrombotics (intracranial hemorrhage, ongoing bleeding, high-grade solid organ injury); identification of an injury when treatment cannot be instituted is not paramount.

Patients with identified BCVIs undergo repeat imaging 7 to 10 days after their initial diagnostic study. The importance of follow-up imaging is particularly salient in patients

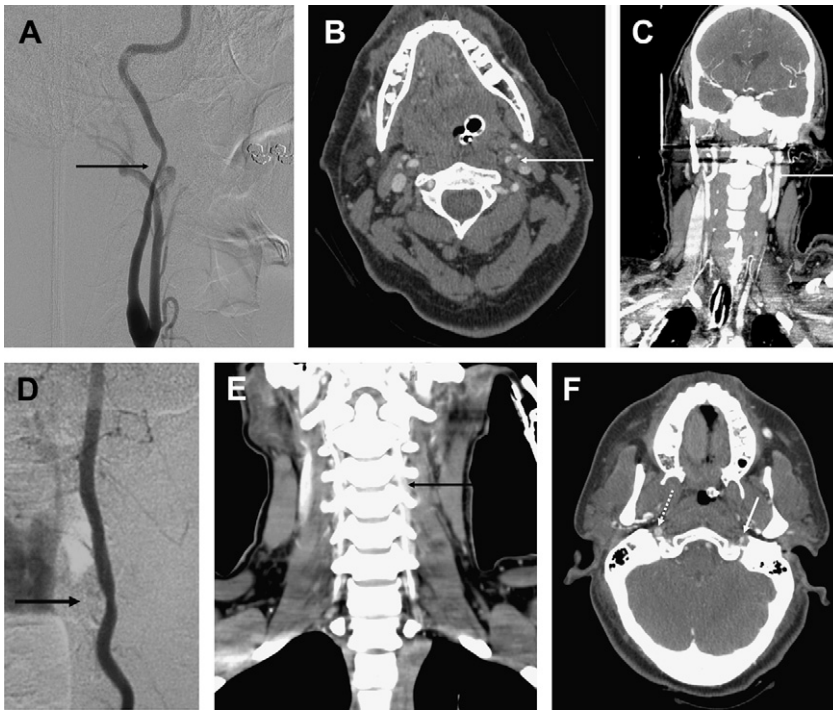

Fig. 6. Grade 2 injury to the carotid artery (*A–C*) and vertebral artery (*D–F*), with luminal narrowing greater than 25%. (*E, F*) Two different patients: normal caliber right vert (*dashed arrow in F*) and narrowed lumen of left vertebral artery (*solid arrow in F*).

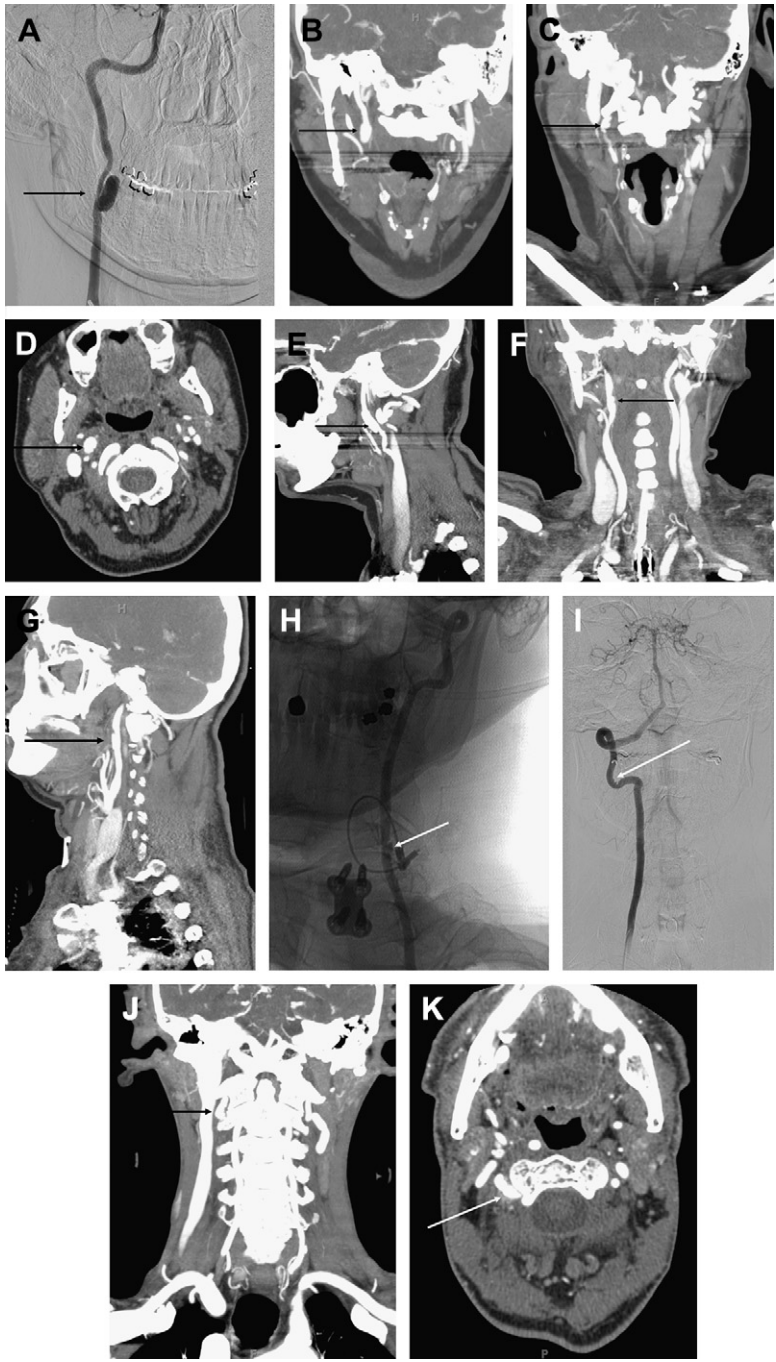

Fig. 7. Pseudoaneurysms of the carotid (*A–G*) and vertebral (*H–K*) artery are classified as grade 3 injuries.

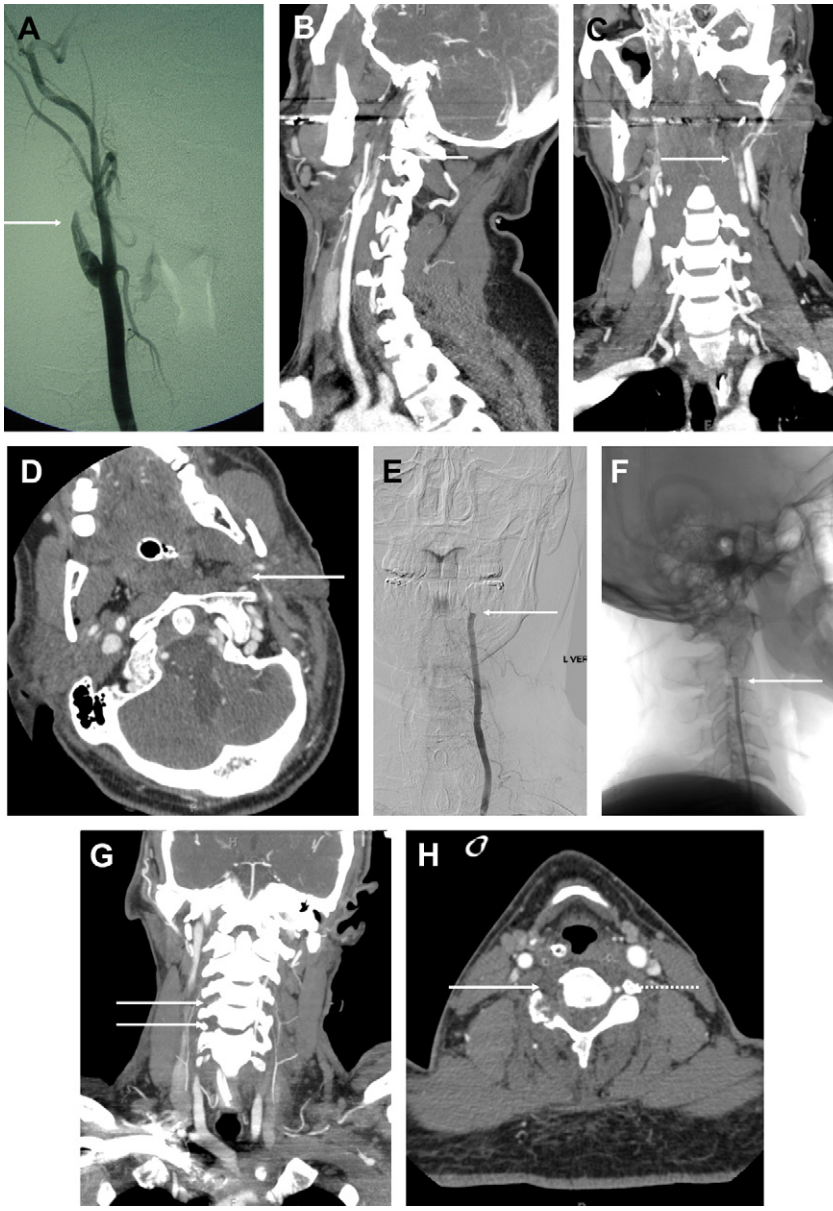

Fig. 8. Grade 4 injury, complete vessel occlusion, to the carotid artery (*A–D*) and vertebral artery (*E–H*). (*C*) Occluded left carotid artery with contrast fading out at the tip of the arrow; contrast within the internal jugular vein is evident just lateral to this. (*D*) Occluded left carotid artery with no contrast seen at the tip of the arrow. (*G*) Occluded right vertebral artery with no contrast seen within the foramen transversarium. (*H*) Occluded right vertebral artery with no contrast seen in the foramen transversarium (*solid arrow*) with a normal appearing left vertebral artery (*dashed arrow*).

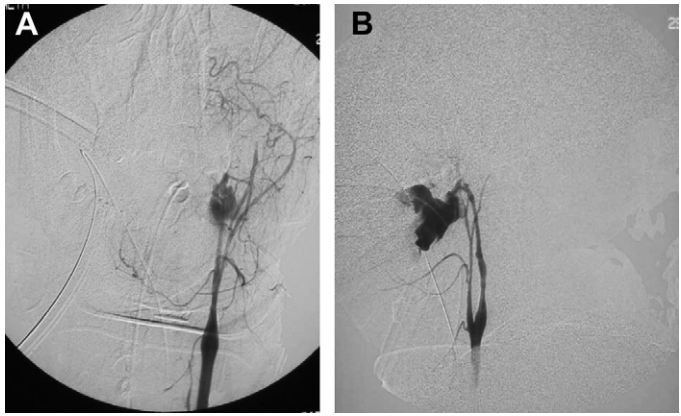

Fig. 9. (*A, B*) Grade 5 injury of the carotid artery with free contrast extravasation from the transected vessel.

with grade 1 injuries; more than half of grade 1 injuries completely heal, allowing cessation of antithrombotic therapy.[1,2] Conversely, less than 10% of all grade 2, 3, and 4 injuries heal, with injury progression rates of approximately 12% for all treated BCVIs.[1] Some investigators have advocated an endovascular approach to pseudoaneurysms,[43] hence supporting the use of repeat angiography to diagnose such lesions. The authors' most recent evaluation of endovascular stents in patients with postinjury BCVIs, however, suggests that antithrombotic therapy remains the gold standard treatment.[9] However, other investigators have supported the use of endovascular techniques with appropriate postprocedure antiplatelet agents.[12,44] Patients with carotid or vertebral artery occlusions may not require reimaging, as approximately 80% show no change on follow-up imaging.[1,2]

TREATMENT OF BCVIs

After the recognition that BCVIs were responsible for patients' adverse neurologic events, treatment modalities were debated. The vast majority of these lesions occur in surgically inaccessible areas of the blood vessels, either high within the carotid canal at the base of the skull or within the foramen transversarium. Such a location makes the standard vascular repair approaches, including reconstruction or thrombectomy, challenging if not impossible. Initial therapy for BCVIs was based on anecdotal reports of neurologic improvement with heparinization in patients suffering stroke related to BCVIs.[6,7,9] Subsequently, intravenous heparin was thought to be the treatment of choice for those asymptomatic patients with blunt injuries,[2,4] with a modified protocol to reduce the incidence of bleeding in multisystem trauma patients.[10,19] As a result of the ease of administration, the initiation of antiplatelet agents gained favor.[2,21,45] Although the optimal regimen remains unanswered, there seems to be equivalence between the 2 therapies.[1,2,4,5] Which therapeutic agent is used, must continue to be evaluated in prospective studies. With an attendant permanent neurologic morbidity rate up to 80% and mortality rate up to 40%,[20,46,47] prompt treatment of diagnosed injuries is critical. Patients who are diagnosed early and treated with antithrombotics almost universally avoid stroke.[1,4,5] After initiation of antithrombotics, treatment is empirically continued for 6 months. Comprehensive long-term follow-up beyond the acute hospitalization has not been reported in the

literature, as is true in most trauma population studies. The Memphis group has the longest follow-up of patients with CAIs,[44] but this seems to be a selected group. Therefore, whether these injuries heal or persist over the lifetime of the patient is unknown.

SUMMARY

Screening, diagnostic imaging, and treatment of BCVIs have evolved over the past 3 decades. Currently, protocols exist for screening based on injury mechanism and associated injuries. Prompt initiation of antithrombotic therapy after identification of injuries in asymptomatic patients reduces the incidence of stroke. Surgeons caring for the multiply injured should screen for carotid and vertebral artery injuries in high-risk patients.

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