Imaging of blunt cerebrovascular injuries

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Abstract

Blunt cerebrovascular injuries are uncommon but potentially devastating injuries that can lead to stroke and death. While uncommon, appreciation of the seriousness of these injuries, a high index of suspicion in high risk patients, and aggressive screening of multitrauma patients leads to early diagnosis of asymptomatic lesions that may be amenable to treatment prior to the onset of ischemia. The radiologist can play a vital role in the early diagnosis, follow-up, and, in some cases, treatment of these challenging injuries.

Keywords: Blunt trauma; Carotid artery injury; Vertebral artery injury; Diagnosis

1. Introduction

Historically, blunt cerebrovascular injuries (BCVI) were considered uncommon in the general blunt trauma population. Although many of these lesions lead to stroke and death, diagnosis prior to onset of symptoms can be challenging. With increased appreciation of these injuries, increasing therapeutic options, and increasing access to ever advancing diagnostic imaging modalities, this group of injuries has received greater attention in the trauma literature and from those caring for acutely traumatized patients.

The purpose of this article is to provide the radiologist with a review of blunt cerebrovascular injuries with special attention to diagnosis and those clinical factors that impact diagnosis and the radiologist’s role in patient management.

2. Incidence

Blunt cerebrovascular injuries (BCVI) are generally considered uncommon injuries. Among all hospital admissions for blunt trauma, blunt carotid artery injuries (BCI) are encountered in 0.14–1.1% of patients [1–3] and blunt vertebral artery injuries (BVI) are encountered in 0.4–0.53% [4,5]. Although the overall incidence of BCVI ranges from 1.1 to 1.4% [6,7] of all blunt trauma patients, it is more frequently diagnosed in more severely injured patients. For example, Mutze et al. [8] reported an incidence as high as 2.7% in blunt patients with an injury severity score equal to or greater than 16.

3. Outcomes

BCVIs result in high mortality and morbidity. For BCI, the overall mortality and morbidity rates are 17–38 and 32–67% [1–3,5,7,9], respectively. Overall mortality and morbidity rates for BVI are generally lower, although morbidity still ranges from 14 to 24% [5,10] and mortality ranges from 8 to 18% [4,9]. The high morbidity and mortality are, in part, attributable to the severity of concurrent injuries frequently associated with BCVI, although BCVI-specific mortality itself is high. In one retrospective review of 22 patients with BCVI, McKevitt et al. [11] reported a stroke rate of 60% and BCVI-specific mortality of 25%. In a prospective study conducted by Cothren et al. [9], BCI-specific mortality was 21%, while BVI-related mortality was 18%. Moreover, reflecting a stroke’s globally deleterious impact on a patient, Cothren reported higher overall mortality in those with BCVI-related stroke (32% for BCI and 18% for BVI) compared to those without stroke (7% for both BCI and BVI).

4. Mechanism of injury

Hyperextension and contralateral rotation of the head and neck, with the cervical internal carotid stretched against the transverse processes of the upper three cervical vertebrae, is
Fig. 1. A 82-year old woman with atlanto-occipital dissociation and bilateral vertebral artery injuries following motor vehicle collision. 3D volume rendered (a) and axial images (b) obtained with whole-body multidetector computed tomography (WB-MDCT) protocol. Magnetic resonance angiogram (MRA) (c) and T1 weighted spin echo magnetic resonance image (MRI) (d). WB-MDCT demonstrates right vertebral artery segmental occlusion from C1 to foramen magnum (curved arrow), focal left vertebral artery central narrowing from intramural hematoma at level of foramen magnum, and bare occipital condyles (asterisks). MRA demonstrates absent flow in distal right vertebral artery with focal narrowing of injured left vertebral artery (arrow). Left vertebral artery high T1 signal (arrowhead) indicates intramural hematoma from dissection.
considered the most common cause of BCI [12]. Also, extension of basilar skull fractures through the carotid canal may cause petrous carotid injury [12]. Less common causes of BCI include a direct blow to the neck (e.g. assault, near hanging, etc.) [12] and intraoral trauma from either a hard foreign object (e.g. falling against a toothbrush) [12] or posteriorly displaced mandible fracture fragments [13].

Since most of the vertebral arteries are fixated within the transverse foramina, they are vulnerable to injuries induced by displaced bone fragments or stretching related to rotation and subluxation [14,15]. In cases of craniocervical junction distraction or dislocation, the vertebral arteries may be crushed against C1 [14] or stretched over the dura [16] (Fig. 1).

5. Clinical presentation and associated injuries

The diagnosis of BCVI is typically suspected in patients presenting with overt signs of fixed neurologic deficits that are unexplained by cerebral computed tomography (CT). Other signs, such as transient ischemic attacks, neck hematomas, Horner’s syndrome, or cervical carotid bruit, also suggest BCVI, although they can be difficult to identify in the acute multitrauma patient [17], especially those with concomitant traumatic brain injury.

Several studies suggest prompt treatment reduces BCVI-related stroke and mortality rates [4,5,7,18]. To this end, several retrospective studies have described numerous clinical and imaging findings that may serve as triggers for screening of blunt trauma patients for BCVI [5,11,12,17,19–22]. Table 1 lists the authors’ institutional guidelines for predicting BCVI. Despite a lack of consensus regarding the most significant independent predictors for BCVI, it has been shown that the greater the number of risk factors, the greater the likelihood of an associated vascular injury [12].

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<th>Table 1</th>
<th>Authors’ institutional guidelines for identifying trauma patients at high risk for blunt cerebrovascular injury</th>
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<td>1. Cervical spine fracture with</td>
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<td>A. Foramen transversarium involvement, or</td>
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<td>B. 30% or more subluxation, or</td>
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<td>C. Significant rotation or distraction mechanism (e.g. unilateral interfacetal dislocation, atlanto-occipital distraction, etc.)</td>
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<td>2. Basilar skull fracture crossing carotid canal, foramen lacerum, or cavernous sinuses</td>
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<td>3. Severe facial fractures (LeFort II or III; naso-orbital ethmoid complex, facial smash)</td>
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<td>4. Carotid or vertebral artery perivascular hematoma by CT or MD-CTA</td>
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<td>5. Horner’s syndrome</td>
<td></td>
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<td>6. Glasgow Coma Score ≤ 6 at 24 h after initial assessment</td>
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<tr>
<td>7. Neurologic examination incongruent with brain imaging</td>
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<td>8. Stroke or transient ischemic attack</td>
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<td>9. Hanging attempt with cervical hematomas or cervical spine fractures</td>
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CT, Computed tomography; MD-CTA, multidetector computed tomographic angiography. \References: Lee et al. [16], Miller et al. [19], Kerwin et al. [20], Cothren et al. [21], Mirvis et al. [23] and Parbhoo et al. [24].

Many proposed predictors of BCVI can be readily identified during routine imaging of acutely injured patients [11,17,22]. In a retrospective review of 75 patients with BCVI, Biffl et al. [22] identified diffuse axonal injury (DAI) (Fig. 2), LeFort II or III fractures (Fig. 3), and petrous temporal bone fractures with carotid canal extension (Fig. 4) as independent risk factors for BCI. Retrospective studies conducted by Berne et al. [25] and Utter et al. [17] support the association of skull base fractures with BCVI. BVI and cervical spine injury have been strongly associated in several studies [4,7,10,21,17,22,24,26]. In one study [4], 38 patients with BVI were 11 times more likely than not to have a cervical spine injury. In another study [21], 77% of patients with BVI demonstrated cervical spine injuries, with most exhibiting either fracture involving a transverse foramen.
Fig. 3. A 44-year old male imaged with WB-MDCT following motor cycle crash. 3D volume rendered image (a) of face demonstrates extensive facial fractures that include LeFort II and III patterns (arrowheads). Sagittal multiplanar reconstruction (MPR) (b) demonstrates characteristic short-tapered post-traumatic occlusion of ICA just distal to carotid bulb (arrow).

(Fig. 5), subluxation or dislocation, or injury level at C1 to C3. Parbhoo et al. [24] report a high frequency of BVIs among those with unilateral facet dislocations, which is an association supported by the authors’ experience.

Some significant clinical risk factors can be inferred by findings on routine imaging. For example, a very low (≤8) Glasgow Coma Score (GCS) [11,22] has been associated with BCVI. Severe traumatic brain injury revealed by cerebral imaging may suggest a low GCS, and then allow the radiologist to propose evaluation for BCVI. Similarly, a high (≥3) thoracic abbreviated injury score (AIS) [11] has been linked to BCVI (Fig. 6). Since imaging plays the primary role in determining

Fig. 4. A 23-year old with skull base fractures following motor vehicle collision. Computed tomography (CT) (a) demonstrates displacement of fragments into carotid canal (arrowheads). (b) Angiogram demonstrates petrous ICA (curved arrow) and cavernous ICA (arrow) pseudoaneurysms.
Fig. 5. A 22-year old male with vertebral artery injury induced brainstem stroke following motor vehicle collision. Admission cervical spine CT (a) demonstrates C6 left transverse process fractures involving foramen transversarium (black arrow) (b) and angiogram demonstrates associated abrupt occlusion of the proximal left vertebral artery (white arrow). Diffusion weighted (c) brain MRI performed 10 days later demonstrates rostral brain stem infarct (arrowhead).

6. Location and types of vascular injury

Blunt cerebrovascular injuries may occur anywhere along the carotid and vertebral artery distributions, and evaluation of both circulations from their origins through their intracranial watershed is vital. However, coinciding with the frequent association with cervical spine injuries, most BVIs occur at the foraminial (i.e. V2) vertebral artery segments [26]. As predicted by the
dominant mechanism of injury, BCIs typically affect the internal carotid artery just below the skull base [C.W. Sliker, unpublished data], although a significant number occur at or above the skull base [12].

Notably, a large number of those with BCVI manifest more than one injured vessel (Figs. 1 and 6). Multiarterial injury, in various combinations, has been reported in 18–38% of cases [18,20,27,28]. Given the violent and complicated force vectors
Fig. 7. A 17-year old with female with femur fracture (not shown) and ICA injury following motor vehicle collision. Admission WB-MDCT (not shown) demonstrated narrowed distal left ICA cervical segment. Coronal maximum intensity projection (MIP) computed tomography angiogram (CTA) image (a) acquired day #2 demonstrates left ICA pseudoaneurysm (white arrow) and normal adjacent lumen. Coronal MIP CTA image (b) acquired day #7 and angiogram acquired day #9 (c) demonstrate larger pseudoaneurysm (black arrow) and near occlusion of adjacent ICA lumen (white arrowheads). Angiogram acquired after endovascular stent placement (d) demonstrates normal ICA lumen (black arrowheads) and minimal pseudoaneurysm filling (curved arrow).
Table 2
Biffl blunt cerebrovascular injury grading system

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<th>Injury grade</th>
<th>Description</th>
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<tr>
<td>I</td>
<td>Luminal irregularity (i.e. intimal injury) or dissection with &lt;25% luminal narrowing</td>
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<tr>
<td>II</td>
<td>Dissection or intraluminal hematoma with ≥25% luminal narrowing, intraluminal thrombus, raised intimal flap, or hemodynamically insignificant AVF</td>
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<tr>
<td>III</td>
<td>Pseudoaneurysm</td>
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<tr>
<td>IV</td>
<td>Occlusion</td>
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<tr>
<td>V</td>
<td>Transection or hemodynamically significant AVF</td>
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AVF, Arteriovenous fistula. Reference: Biffl et al. [27].

The forms of injuries identified in the carotid and vertebral arteries are similar to those encountered in arteries elsewhere in the body. Typical types of injury include intimal injury, spasm, dissection with intramural hematoma, raised intimal flap, intramural hematoma, pseudoaneurysm, occlusion, transection with active hemorrhage, and arteriovenous fistulae (AVF) [16,29,30]. Dissections may vary in degree of luminal compromise from minimal to near occlusion. Combinations of injury can be seen (e.g. pseudoaneurysm with dissection, raised intimal flap with intraluminal thrombus, etc.) in the same artery. With occlusion or high grade luminal narrowing, inadequate collateral flow and/or hypotension can lead to arterial insufficiency and ischemia, while intimal injury may promote platelet aggregation and embolization [12]. Pseudoaneurysms may promote platelet aggregation with distal embolization or compress the native vessel lumen [31] with resultant downstream underperfusion. Rarely, pseudoaneurysm rupture or transection may lead to ischemia or hemodynamic compromise [12].

In an effort to predict risk of stroke, Biffl et al. [29] devised a blunt carotid artery injury grading scale based on the anatomic features of injury. In a subsequent report [27], the scale (Table 2) was revised to include AVFs, and it was used to grade BVIs as well as BCIs. When BCVIs are reported with the Biffl scale, the highest grade injury identified determines the score for that particular vessel. At the authors’ institution, all injuries in a particular vessel are described, although only one may be used to determine overall grade of injury. In addition to providing some prognostic information, the grading scale is a useful reporting shorthand that is easily understood by the traumatologist.

7. Diagnosis of BCVI

7.1. Angiography

Four-vessel cervicocerebral angiography is considered the reference standard for diagnosing BCVI [10,20]. As an invasive test, angiography carries an inherent risk of serious complications, including stroke and hemorrhage. Yet, with experienced operators, routine angiographic assessment of trauma patients, who are generally young and without pre-existing vascular disease, is a safe procedure. For example, in one report by Cothren et al. [9], 727 blunt trauma patients were angiographically screened for BCVI, and there was one instance (0.1%) of angiography-related stroke and two cases (0.3%) of conservatively-managed puncture-site hematomas. In those in whom BCVI is strongly suspected clinically, the risks would seem to be tolerable. Still, in a screening population without hard clinical signs of BCVI, many may consider potential complications of angiography unacceptable given the relative rarity of these injuries. Moreover, angiography is also expensive and resource intensive. Consequently, other diagnostic modalities, including Doppler ultrasound, magnetic resonance angiography (MRA), and computed tomographic angiography (CTA), are being increasingly used to diagnose BCVI.

7.2. Multidetector computed tomographic angiography—scanning options

Computed tomography (CT) is an important tool commonly used to evaluate multitrauma patients for head, spine, chest, abdomen, and pelvic injuries. With the advances in multidetector computed tomography (MDCT) technology, multidetector computed tomographic angiography (MD-CTA) is increasingly used to evaluate the arterial systems throughout the body, and, recent reports describe its use to diagnose BCVI [6,7,17,32,33]. With the use of MDCT, routine diagnosis of BCVI does not need to be limited only to scans targeted to the craniovascular region. Current MDCT scanners, used in concert with contrast bolus timing, allow continuous scanning through all regions of interest in the typical multitrauma patient. Utilization of whole-body MDCT scanning techniques permits simultaneous detection of craniovascular, skeletal, thoracic, and organ injuries while forgoing the scanning time and excess radiation [34] inherent to traditional segmental scanning techniques.

In the authors’ trauma center, targeted neck MD-CTA and whole-body MDCT have been performed for over 3 years. Currently, both 16- and 40-detector row scanners are in use, though the 40-detector row scanner is preferred for most whole-body and neck MD-CTA scans (Table 3). In most multitrauma patients, the whole-body scanning protocol is routinely used. The targeted neck MD-CTA protocol is utilized in those cases where clinical interest is directed entirely to the craniovascular arterial system, in addition to cases where the patient is considered at risk for harboring a BCVI and the whole-body MDCT provided suboptimal or equivocal evaluation of the neck vessels. With both protocols, advanced post-processing techniques (e.g. curved multiplanar reconstructions, volume rendering, etc.) are used liberally on an as needed basis.

7.3. Multidetector computed tomographic angiography—diagnostic accuracy

The relatively few studies that have investigated the accuracy of CTA when used to diagnose BCVI are limited by small cohorts.
of injured patient and varied study designs. Compared to the reference standard, angiography, reports of single-slice spiral CTA yielded disappointing results; sensitivities for BCI and BVI were 47–68% [10,19] and 53% [10], respectively. Recently, studies investigating the accuracy of MD-CTA have emerged. In 2006, Biffl et al. [32] and Berne et al. [6] reported their experience screening 331 and 435 patients, respectively, for BCVI with 16-slice MD-CTA. In both studies, injuries (combined total of 42 patients) were confirmed with angiography while those patients with normal (i.e. no BCVI) MD-CTA results were followed clinically for signs of neurovascular complications. Neither group reported delayed neurological complications of BCVI in those with normal MD-CTA examinations, though neither followed normal MD-CTA examinations with angiography. Therefore, in both reports, true accuracy of 16-slice MD-CTA for detecting BCVI was not determined. In a prospective study that also appeared in 2006, Eastman et al. [33] evaluated 146 at-risk patients with both 16-slice MD-CTA and angiography, and they determined 16-slice MD-CTA to have a sensitivity of 97.7% and specificity of 100% when used to diagnose BCVI. Despite these impressive results, the BCVI cohort consisted of only 43 patients [33], which is a small number when studying an uncommon entity; this is a limitation shared by the other two studies. Moreover, Eastman et al. [33] report only 46 injuries in the 43 patients diagnosed with BCVI, which is far from the previously reported multivessel injury rate of 18–32% in patients with BCVI [18,20,28]. Although the results of this study are promising, the small cohort of injured patients and the atypically small number of patients with multiple injured vessels suggest the data may not reflect the true accuracy of 16-slice MD-CTA when used to evaluate the blunt trauma population at large for BCVI. Controversies notwithstanding, the authors feel that MD-CTA (16-slice or greater) plays a valuable role in evaluating patients with suspected BCVI, but, until proven by a large (preferably multicenter and prospective) trial, the authors do not think that MD-CTA should be considered diagnostically equivalent to angiography.

7.4. Magnetic resonance angiography

Few studies have assessed the diagnostic accuracy of magnetic resonance angiography (MRA) relative to angiography when used to detect BCVI. In one study by Biffl et al. [10], 16 patients at risk for BCVI were examined with both MRA and angiography. Angiography confirmed BCVI in three patients, and the collective sensitivity and specificity of MRA for BCVI were 75 and 67%, respectively. In a slightly larger study conducted by Miller et al. [19] 21 at-risk patients were examined with both MRA and angiography. Among the four patients with BCI, the sensitivity of MRA was 50% and its specificity was 100%. Vertebral artery injuries were found in 17 patients, and the sensitivity and specificity of MRA were 47 and 97%, respectively.

Though its sensitivity is limited, the use MRA to diagnose BCVI has several potential advantages relative to other diagnostic options. Among them, the examination does not utilize ionizing radiation or require intravenous contrast. However, in most centers, magnetic resonance (MR) imaging is not as rapidly accessible as CT. In addition to the time and personnel required to transport the patient to the MR suite, which is remote from patient care areas in many centers, the MR environment itself is not conducive to the management of the acutely injured patient. Consequently, even if future well-designed studies demonstrate that contemporary MRA techniques have diagnostic accuracies
comparable or exceeding those of CTA, the authors feel that the physical limitations of MRA should limited it use in favor of MD-CTA (16 detector row or greater) or angiography, with the use of MRA relegated to instances when the other two diagnostic options are unavailable, their results are equivocal, or when the use of iodinated contrast material and/or ionizing radiation is contraindicated.

7.5. Sonography

Although operator dependent, ultrasound is a portable, inexpensive tool in common use by traumatologists, and Duplex Doppler techniques are established means for diagnosing and monitoring non-traumatic cerebrovascular disease. Therefore, ultrasound is an attractive option for diagnosing BCVI. However, since the intracranial internal carotid arteries and most of the vertebral arteries are encompassed by bony canals, the ability of ultrasound to detect potentially significant injuries (e.g. intimal flaps, pseudoaneurysms) without associated flow disturbances can be limited. The few studies that have discussed the sensitivity of Doppler ultrasound for detecting BCVI report disappointing results. In one early study [35], Doppler ultrasound diagnosed 14 patients with BCI within the neck for a sensitivity of 86%, although it missed two injuries near the skull base. In a more recent study, Mutze et al. [8] detected BCVI in 0.9% of 1471 patients screened with Doppler ultrasound with sensitivity of only 38.5%. The eight patients in whom Doppler ultrasound missed injury came to clinical attention as result of cerebrovascular ischemia, supporting the notion that injuries missed by sonography are potentially significant. Given its limitations, Doppler ultrasound should only be utilized when the use of other imaging techniques is impossible.

8. Injury evolution

Both blunt carotid and vertebral arteries tend to be dynamic lesions. That is, over the course of days or weeks, the severity of both treated and untreated lesion may either progress or regress. In their review of 114 carotid artery and 65 vertebral artery injuries followed with angiography 7–10 days after sustaining injury, Biffl et al. [27] noted that grade I and II injuries frequently healed by initial follow-up, and therapy could then be discontinued in 57% of grade I and 8% of grade II lesions. Conversely, 8% of grade I lesions and 43% of grade II lesions progressed, leading to more aggressive therapeutic interventions. Ninety-three percent of grade III injuries and 82% of grade IV injuries were unchanged, although in the authors’ experience, these lesions, especially carotid injuries, change with greater frequency. Notably, Biffl et al. noted no significant difference in injury progression or regression among patient populations receiving systemic anticoagulation, antiplatelet therapy, or no medical therapy [27].

At the authors’ trauma center, blunt cerebrovascular injuries are followed with the imaging modality that most satisfactorily characterized it during the initial work-up; this is usually an MDCT angiogram. The first follow-up imaging is typically performed between 24 and 72 h after initial diagnosis. During this brief interval, BCVI frequently change enough to alter management, which is particularly important given the relative frequency of coexisting injuries (e.g. unstable cervical spine injury requiring surgery, major visceral injury, etc.) that may complicate treatment with anti-thrombotic agents. A second follow-up examination is usually obtained within 1 week to 1 month (Fig. 7a and b). Beyond this point, the timing of repeat imaging is generally based on injury morphology and response to management. Uncommonly, rapid morphologic changes render differentiation between injury progression and vascular remodeling associated with healing difficult, and, in these cases, more frequent follow-up imaging can be especially useful.

9. Treatment

Though treatment of some BCVI may be impossible or fruitless due to early extensive stroke or other contraindications (e.g. intracranial hemorrhage, spinal cord injury) [36], multiple authors have suggested that treatment with either medical therapy or endovascular techniques can either prevent or limit BCVI-related neurologic deficits [2–5,18,19,37,38]. To this date, the optimum treatment regimen has yet to be determined, but the current trend is towards medical therapy as option of choice. Many consider anticoagulation first line therapy [2–5,9,18,19,29], with antiplatelet therapy reserved for those with contraindications to anticoagulation. In one study conducted by Miller et al. [5], timely treatment with either anticoagulation or antiplatelet therapy lowered stroke incidence to 6% in patients diagnosed with BCI and to 0% in those with BVI (compared to 50% for both untreated BCI and BVI). Antiplatelet has been proposed by others [37] to exhibit efficacy comparable to anticoagulation while reducing the risk of hemorrhagic complications. At this time, the superiority of one approach over the other is unproven.

Of special interest to radiologists, endovascular repair with stent placement has been used to treat BCIs when lesions are refractory to medical therapy [31,38] (Fig. 7c and d), medical therapy is contraindicated [38], or either angiography or perfusion imaging demonstrates evidence of cerebral hypoperfusion [38]. In a study reported by Cohen et al. [38], 10 patients with post-traumatic cervical internal carotid artery (ICA) dissections were treated with endovascular stent placement. There were no procedure-related complications or stent occlusions, and at a mean clinical follow-up time of 16 months four patients demonstrated no deterioration in neurologic status while six patients improved. In contrast, Cothren et al. [39] reported a 21% complication rate (three strokes and one subclavian artery dissection) and an occlusion rate of 45% among 23 patients with post-traumatic cervical ICA pseudoaneurysms treated with stents. In a cohort of 18 patients who received only medical therapy, none suffered stroke or progressive deficit after initiation of treatment, and one suffered carotid artery occlusion despite anticoagulation. Interestingly, this report contradicts an earlier one from the same institution, in which Coldwell et al. [31] described uncomplicated endovascular treatment of cervical ICA pseudoaneurysms in 14 patients in whom a mean clinical follow-up of 16 months revealed no evidence of stroke or delayed stent
complication. It must be noted that in Cohen’s study, all patients received antiplaetelet therapy with aspirin and clopidogrel [38], while those suffering stent occlusion in Cothren’s study only received anticoagulation [39], thereby suggesting that the antiplaetelet therapy either alone or in combination with the stents contributed to the more promising results reported by Cohen. Clearly, the role and methods of endovascular repair of BCVI remain areas in need of continued investigation.

Endovascular therapy of vertebral artery injuries is also a diagnostic option. Although emergent coil embolization of vertebral artery transactions is an appropriate approach [40], the merits of a comparable approach to less severe injuries remain incompletely explored. Since Biff et al. [27] suggest the highest rate of BVI-related stroke (38%) occurs with grade II injuries, and up to 75% of traumatic vertebral artery occlusions can reanализe [24], coil embolization of occluded vessels has been proposed [41] to prevent reconstitution and the risk of distal thromboembolization. While this approach may pose no additional harm to the patient, its long term merits measured against those of medical therapy and the immediate risks of an endovascular procedure are unknown. Although dissections and pseudoaneurysms can be addressed with endovascular occlusion, this should be done with caution with documentation of adequate collateral flow through the contralateral vertebral artery and circle of Willis established.

10. Conclusion

Blunt cerebrovascular injuries are infrequently encountered, and a high index of suspicion coupled with understanding of injury mechanism and recognition of associated injuries facilitates early diagnosis and treatment of asymptomatic lesions. Based on available resources, each institution must determine in what setting and with what means will the evaluation for BCVI be initiated. Although its accuracy remains unproven, liberal screening with 16-slice (or higher) MD-CTA facilitates early diagnosis and treatment. In cases where MD-CTA results are suspicious, equivocal or normal in high-risk patients, or clearly positive in those whom endovascular therapy is an option, prompt follow-up with four-vessel cerebral angiography is recommended. Regardless of the diagnostic and therapeutic approaches utilized to address blunt cerebrovascular injuries, the authors advocate that institutions caring for blunt trauma patients adopt a coordinated and systematic approach in order efficiently utilize resources while optimizing the outcomes of those diagnosed with these uncommon but potentially devastating injuries.

References


