Current Neurology and Neuroscience Reports (2018) 18: 98
https://doi.org/10.1007/s11910-018-0906-7

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Management of Blunt Cerebrovascular Injury

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Published online: 23 October 2018

Abstract

Purpose of Review This review provides an updated summary of blunt cerebrovascular injury (BCVI) to guide clinicians in its early diagnosis and prevention and treatment of stroke associated with such injury.

Recent Findings Untreated BCVI causes stroke in 10–40% of patients, but more than half will not present with stroke symptoms initially. Risk of stroke is highest in the first 7 days, with a peak in the first 24 h. Computed tomography (CT) angiography is currently the screening modality of choice, although digital subtraction angiography may still be required in some cases. Antithrombotic therapy is the mainstay of treatment and has proven safety in trauma patients. In carefully selected patients, endovascular intervention may also be beneficial.

Summary BCVI is a potentially preventable cause of stroke. A high index of suspicion is needed as emergent screening during initial evaluation can provide a window for stroke prevention. Screening all patients with injuries that would otherwise prompt CT scans of the neck or chest is recommended. Treatment is guided by grade of injury. Early treatment with antithrombotics has been shown to be both effective and safe.

Keywords Stroke · Blunt cerebrovascular trauma · Dissection · Cerebrovascular injury · Carotid artery · Vertebral artery

Introduction

Blunt cerebrovascular injury (BCVI) is a rare but serious complication following trauma. BCVI that results in carotid dissection, vertebral dissection, and/or pseudoaneurysm formation carries a high risk of ischemic stroke when not recognized and treated early. Intimal tearing initiates a thrombogenic cascade that may result in arterial stenosis, occlusion, or distal thromboembolism. The risk of stroke increases with severity of trauma and vessel injury [1–3] and is associated with significant morbidity and mortality [2–5].

The most common mechanism of BCVI is by motor vehicle accident, either as a passenger or pedestrian. Other causative mechanisms of injury include falls, assault, sports-related injuries, environmental or workplace accidents, direct strike, or any other high acceleration-deceleration event that results in neck hyperextension, hyperflexion, or rotational trauma [1, 6, 7, 8••, 9]. Additionally, chiropractic cervical manipulation is a common practice that also may cause or worsen cervical arterial dissection and is likely underreported, with significant reporting bias and case misclassification plaguing studies [10–12].

Since the widespread adoption of CTA for screening in trauma admissions, the incidence of BCVI is estimated to be as high as 3% [13, 8••]. Untreated BCVI is associated with stroke in 30–40% of patients with carotid artery injuries and in 10–15% with vertebral artery injuries [3, 8••, 14, 15•]. With treatment, the incidence of ischemic stroke is reduced to 0.5–5%, with significant mortality benefit [15•, 16]. However, only about 37% of patients present with stroke symptoms at the time of initial evaluation [9]. Therefore, the key to appropriate management of BCVI is early identification and initiation of therapy [7]. Here, we review the current literature regarding screening, characterization, and treatment of BCVI.

This article is part of the Topical Collection on *Stroke*

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Evaluation of Blunt Cerebrovascular Injury

Screening

Many patients with BCVI may not have neurologic symptoms at presentation [8••]. The time to stroke due to BCVI varies

widely and can occur over a week after the initial trauma [17–21]. Approximately, 60% of strokes occur within 3 days of injury and over 85% by 7 days, with fewer than half exhibiting neurologic symptoms prior to arrival to the hospital [9]. Aggressive screening results in a much lower incidence of stroke in those who present without symptoms, decreasing the incidence in some studies to < 1% [16–18, 22, 23]. Therefore, recognition and treatment is necessary and cost effective in most cases [16].

The current standard of practice is to screen both symptomatic and asymptomatic high-risk individuals. Although various screening criteria exist in the literature, high-risk factors are relatively uniform [5, 7, 18, 24] (Table 1). The Denver protocol, first proposed in 1999 by Biffl et al. has become the standard from which other protocols have been developed [25]. The criteria identified patients at risk for BCVI using clinical and radiographic evidence of stroke or vascular injury and independent factors, including skull and cervical spine fractures and high-energy injury mechanisms. Of the various risk factors, cervical spine injury has the strongest association with BCVI [7, 25–28]. Traumatic brain injury and basal skull fractures are more likely to result in carotid injuries, whereas cervical spine injury is more likely to result in vertebral artery injury [29].

Due to limitations in accurately determining mechanism and force of injury from the clinical history, surrogate markers for force may be utilized as these have been found to be strong predictors of BCVI. The Denver criteria were expanded in 2011 to include mandible fractures, complex skull fractures, traumatic brain injury, scalp degloving, thoracic vascular injuries, and upper rib fractures (summarized in Table 1 [8••, 18]). The expanded protocol was externally validated to have high sensitivity (97%) and moderate specificity (42%) [30]. However, despite endorsement of these criteria by consensus groups such as the Eastern Association for the Surgery of Trauma Guidelines and Western Trauma Associations [5, 31], their use is likely underutilized in current practice [32].

Table 1 Screening criteria for BCVI according to expanded Denver criteria [8]

Patients to be screened	
Signs or symptoms of BCVI	Risk factors for BCVI
- Potential arterial hemorrhage from neck, nose, or mouth - Cervical bruit in patients < 50 years old - Expanding cervical hematoma - Focal neurologic deficit: TIA, hemipareisis, vertebrobasilar symptoms, Horner's syndrome - Stroke on CT or MRI - Neurologic deficit inconsistent with head CT	- High-energy transfer mechanism - Displaced mid-face fracture (Lefort II or III) - Mandible fracture - Complex skull fracture/basilar skull fracture/occipital condyle fracture - Cervical spine fracture, subluxation, or ligamentous injury at any level - Severe traumatic brain injury (TBI) with and GCS < 6 - Near-hanging with anoxia - Clothesline-type injury or seat belt abrasion with significant swelling, pain, or altered mental status - Scalp degloving - Blunt cardiac rupture - Upper rib fractures

Implementation of the expanded Denver criteria for screening was shown to assist with identification of most of the 20% of patients previously missed with older screening criteria (15% of originally missed plus an additional 21% more) [8••]. Some centers have reported a fivefold increase in identification of BCVI with implementation of formal screening protocols over clinical or radiographic risk factors alone [33, 34••]. It is important to recognize that there may be a delay to identify BCVI due to early focus on management of life-threatening injuries, intubation, or sedation. Therefore a high index of suspicion and early screening during initial evaluation may provide a window for prevention of stroke [16, 35]. In light of this, screening in all patients with injuries that would otherwise prompt CT scans of the neck or chest should be strongly considered [33]. The risk of contrast nephropathy is low, and given the potential for severe disability or death from stroke, the benefit of screening likely outweighs the risk of contrast-related complications for most [36••]. Improvements in technology, therapy, and screening have resulted in drastic reductions in stroke mortality and mortality over the past 3 decades despite increased incidence, but prospective multicenter studies are still needed to help further refine screening protocols for BCVI [15•].

Methods of Screening for BCVI

Computed Tomographic Angiography

At this time, no level I evidence exists to guide the choice of screening modality for BCVI. Computed tomographic angiography (CTA) is considered by most to be the screening test of choice given the cost, safety, speed at which it can be obtained, and ability to simultaneously evaluate other structures (such as bones and soft tissues) routinely screened in trauma patients [37, 38]. The amount of contrast used for these studies is less than that required for four-vessel cerebral digital subtraction angiography (DSA) or arch aortography. The sensitivity of CTA in recent studies approached that of DSA for identification of BCVI (with sensitivity of 98% and specificity of 100%), an improvement over the lower sensitivity of initial studies [5, 34••, 39, 40]. Accuracy may vary depending on experience of the radiologist or other provider reviewing the images. Using a CT with ≥ 16 slices (64 slices are preferred, if available) and having studies read by a trained neuroradiologist appears to improve sensitivity [38, 41]. Technological advances in CT imaging continue to enable faster image

acquisition with improved spatial resolution. DSA should be considered if a CT scanner with at least 16 slices it not available, or if BCVI is suspected despite negative CTA [41]. It is important to recognize that there are mimics to BCVI, including atherosclerotic plaque, fibromuscular dysplasia, vertebral artery hypoplasia, or aggressive vessel tortuosity, and therefore some have advocated for follow-up digital subtraction angiography (DSA) to exclude false positives as well [41].

Digital Subtraction Angiography

DSA remains the gold standard for diagnosis of BCVI. However, because it is more invasive, may be delayed due to availability or ability to perform in a critically injured patient, and is associated with complications such as dissection and thromboembolism in 1–3% of cases [42, 43], it has been largely replaced by CTA as a screening tool in this population. DSA also does not provide much information about the vessel wall beyond identification and characterization of vessel wall hematomas, although it does easily demonstrate collateral circulation. Since an endovascular intervention can be performed concomitantly, DSA may be useful to screen for BCVI in patients felt to be at particularly high risk. DSA may also be used to confirm questionable CTA findings before exposing patients to antithrombotic therapy.

A recent natural history study of BCVI categorized as "indeterminate" on initial CTA showed a quarter of such patients progressed to a radiographically confirmed BCVI, with 5% developing a stroke or transient ischemic attack [44•]. Options for managing this population may include further evaluation with DSA, or empiric treatment and serial monitoring with repeat CT angiography if the risk of hemorrhage is felt to be reasonably low.

Some have advocated for confirmatory testing by DSA in all patients who are found to have BCVI on CTA to avoid unnecessary antithrombotic use [15•, 41]. The necessity of confirmatory DSA testing and clinical significance of overtreating false-positive BCVI is unclear and highlights the need for further studies and updated consensus guidelines. In our practice, we do not routinely recommend DSA for patients with positive CT angiography, instead reserving DSA for those cases in which there is a clinical indication for more detailed vessel imaging, such as clinically indeterminate cases, unexplained neurological findings, or those planned for potential intervention.

Magnetic Resonance Angiography

Magnetic resonance angiography (MRA) is not recommended as a single-imaging modality for the diagnosis of BCVI, but may have a complementary role. It can help differentiate between dissection, intramural hematoma, thrombus, or atherosclerotic plaque, especially with the use of high-resolution double inversion recovery black-blood imaging techniques [45, 46]. It also does not require potentially nephrotoxic contrast and provides added information about ligamentous/spinal injuries and cerebral infarction. However, it takes longer to acquire, which may incur delay in critically injured patients, may not be readily available at all centers, and may not be preferred in patients with lines or medical devices that are not MRI-compatible. Furthermore, in the acute (< 7 days) and chronic (> 2 months) stages, an intramural hematoma appears isointense on MRI, thereby lowering sensitivity of detection [42, 47].

Duplex Ultrasound

Ultrasound is not recommended for screening for BCVI [5]. Approximately 90% of lesions are not sonographically accessible. The scan is also operator-dependent, has lower sensitivity, and is more likely to miss dissecting aneurysms [5, 42].

Transcranial Doppler

Transcranial Doppler (TCD) is not a first-line screening tool for BCVI, but may be used as an adjunct to evaluate carotid artery stenosis and monitor for hemodynamic failure or microembolic signals (MES) to help identify patients at risk for ischemic events [48–51]. Use of TCD for monitoring of vertebral artery MES may not provide benefit, but mean flow velocity asymmetry or elevation in pulsatility index may potentially be predictive of injury in vertebral arteries [50–52].

Treatment

A grading scale for BCVI involving the carotid or vertebral arteries was developed to standardize injury severity and guide therapy (Table 2) [53]. Overall, stroke incidence increases with higher grades of injury, although stroke and neurologic outcome were not found to depend on grade for vertebral artery injuries [6]. Current guidelines from the EAST and Western Trauma Associations recommend antithrombotic therapy (initially with either unfractionated heparin or antiplatelet therapy), endovascular therapy, or surgical repair based on the location and grade (Table 2) [5, 31].

Antithrombotic Therapy

To date, there have been no prospective randomized-controlled trials to definitively guide treatment in BCVI. However, there are multiple retrospective studies demonstrating improved outcomes with antithrombotic therapy, with reduction of stroke incidence to < 10%. The rate of stroke can be as low as < 1% when asymptomatic individuals are identified and treated early [3, 16, 18, 22, 54, 55]. Given that

Table 2 Denver grade-based CTA findings, treatment, and follow-up for BCVI [3, 6, 18]

Injury grade	I	II	III	IV	V
Denver grading system	Irregularity of vessel wall dissection or IMH with < 25% narrowing	Intraluminal thrombus; dissection, small AVF, or IMH with > 25% narrowing	Pseudoaneurysm	Occlusion	Transection
CTA findings	Nonstenotic luminal irregularity, intimal flap, or wall thickening with < 25% stenosis	Luminal hypodensity, intimal flap, or wall thickening with > 25% stenosis	Eccentric contrast-filled outpouching limited by periarterial tissue	Lack of any intraluminal enhancement, carotid occlusions (abrupt or tapered), vertebral occlusion (usually abrupt)	Irregular extravascular collection of contrast, not limited by periarterial tissues, increases in density on delayed images if obtained
Stroke incidence (%)	8% carotid	14% carotid	26% carotid	50% carotid	100% carotid
	6% vertebral	38% vertebral	27% vertebral	28% vertebral	100% vertebral
Initial therapy	Antithrombotic[a]	Antithrombotic[a]	Antithrombotic[a]	Antithrombotic[a]	Direct pressure on actively bleeding area until surgical intervention
Surgical/endovascular therapy	Not needed	Rarely needed, but consider if neurologic symptoms, progression of dissection, or if refractory to therapy[$]	Consider if symptomatic or if > 1 cm[$]	Stenting typically not beneficial, but thrombectomy ± stenting may be considered if stroke recognized within 6 h[b]	Emergent intervention
Timing of follow-up imaging	7–10 days, then every 3–6 months until healed	7–10 days, then every 3–6 months until healed or definitive management	7–10 days, then every 3–6 months or based on symptoms	Based on symptoms	Based on symptoms
Long-term therapy	Antiplatelet therapy until healed	Antiplatelet therapy until healed or definitive surgical treatment	Antiplatelet therapy until healed or definitive surgical treatment	Lifelong antiplatelet	No data available, consider if symptomatic

[a] Treatment recommended unless contraindicated, see discussion in text regarding choice of UFH vs antiplatelet therapy for initial therapy

[b] See discussion regarding endovascular treatment in body of text, emerging field

AVF arteriovenous fistula, *IMH* intramural hematoma, *UFH* unfractionated heparin

thromboembolism is felt to be the primary mechanism for ischemic stroke in BCVI [56], treatment with antithrombotic therapy is the mainstay of therapy for grades I–IV injuries [2, 3]. While some guidelines have suggested observation as a possible option for grade I injuries, this is not often chosen given the significant morbidity and mortality of stroke and the relatively low risk associated with antithrombotic therapy.

Antithrombotic therapy should be initiated immediately for all BCVI patients without contraindications as it has clearly shown to significantly reduce the chance of stroke [3, 8••, 17, 31]. Patients who present initially with solid organ injury, traumatic brain or spinal cord injury, active bleeding, or hemorrhagic neurologic injury may also benefit from early antithrombotic therapy once stabilized and bleeding is controlled, without increased incidence of bleeding complications [57–59]. TCD with MES detection in the setting of carotid injury may potentially be useful for clinical decision-making when weighing the risk of stroke versus the risk of hemorrhage with early initiation of antithrombotic therapy in such complex cases [56].

The choice of antiplatelet therapy versus anticoagulation as initial therapy depends upon grade of BCVI, concomitant injuries, neurological symptoms, and the volume of infarcted territory at risk for hemorrhagic transformation. Current antiplatelet regimens include aspirin 81–325 mg or clopidogrel 75 mg daily, whereas anticoagulation is generally achieved initially with unfractionated heparin, with subsequent transition to oral anticoagulation after hospitalization. In patients at risk for hemorrhagic complications, unfractionated heparin should be given at a dose of 10 U/kg/h without bolus to achieve a more conservative goal-activated partial thromboplastin time (aPTT) of 40–50 s. Low molecular-weight heparin may be considered as an alternative to unfractionated heparin, but has not been studied well in this population. Antiplatelet agents have the advantage of being easier to administer, less expensive, and relatively well tolerated in trauma patients, and several studies have demonstrated lower rates of bleeding when compared to anticoagulation [2, 4, 17, 60]. Some trials and several retrospective studies have suggested that antiplatelet therapy is at least as effective as heparin for stroke prevention [3, 4, 17, 60–62] and is a reasonable choice, especially in patients for whom no surgical intervention planned [4, 17]. The CADISS trial group and others found no difference in efficacy of antiplatelet and anticoagulant drugs for the prevention of recurrent stroke after symptomatic carotid and vertebral artery dissections [63–65]. A recent study in which 70% of BCVI patients had concomitant brain injury noted antiplatelet use in the majority of patients, with no report of progressive intracranial hemorrhage, further supporting the safety of antiplatelet therapy in this population [66]. Heparin is chosen as initial therapy by some groups over antiplatelet therapy due to ease of reversibility and short half-life in trauma patients who may require surgical intervention.

Endovascular Stenting

For the vast majority of BCVI patients, initial treatment with antithrombotic therapy alone is adequate (Table 2). Some patients may benefit from endovascular stenting [67–69]; however, there is controversy regarding this procedure in the literature. Stenting is typically reserved for higher-grade lesions, such as dissections with significant narrowing (grade II) and early neurologic deficits, enlarging (> 1.0 cm) carotid pseudoaneurysms (grade III), or grade V lesions that are not surgically accessible [5, 31, 70]. Although endovascular therapy is generally safe in this population [2, 4], one group reported no increase in stroke burden or mortality following a reduction in the number of stenting procedures at their institution [70]. The overwhelming majority of BCVIs will heal with antithrombotic therapy alone [71•]. Current American Heart Association/American Stroke Association guidelines for treatment of ischemic stroke recommend stenting only for those patients with definite recurrent ischemic stroke due to extracranial carotid or vertebral artery dissection despite medical therapy given the lack of established benefit [72]. Stenting also requires several months of dual antiplatelet therapy, which carries a higher risk of bleeding, further making the case for judicious use of stents.

While most traumatic aneurysms resolve spontaneously or remain stable and can be managed with antiplatelet therapy alone, large or enlarging aneurysms may also benefit from endovascular therapy [73]. Unfortunately, data regarding the stroke risk, natural history of traumatic extracranial aneurysms, and outcomes are lacking.

Surgical Therapy

Grade V injures (transection with extravasation) should have immediate intervention with application of pressure to the site (if possible) and surgical intervention in attempt to control the hemorrhage and restore blood flow if possible. Mortality and incidence of stroke are very high with grade V injuries. Patients without profound neurologic deficit or coma do better with repair than with vessel ligation/occlusion [5, 74, 75].

Duration of Treatment

Duration of antithrombotic therapy is based on grade of injury and any potential endovascular/surgical intervention (Table 2). For long-term therapy, patients on anticoagulation should be transitioned to antiplatelet therapy, as antiplatelets have superior safety data compared to long-term anticoagulation, and there is no evidence of added benefit of anticoagulation [17, 31, 64]. In our practice, we rarely continue anticoagulation for longer than 6 months. Patients with arterial dissections or pseudoaneurysms who are started on heparin should have a follow-up imaging with repeat CTA in 7–10 days. If the vessel

is occluded at the time of follow-up imaging, they are changed to lifelong antiplatelet therapy alone.

Grade I injuries are often treated with antiplatelet therapy until the vessel appears healed on follow-up imaging, usually for 3–6 months [72]. Some have also completely discontinued antithrombotics if the vessel appears healed on radiographic follow-up at 7–10 days. Grades II and III injuries will require lifelong antiplatelet therapy, provided the lesion remains stable. However, if the vessels appear to be healed after 3–6 months of therapy, discontinuation of antiplatelet therapy may be considered as well. Grade IV lesions require lifelong antiplatelet therapy to reduce the risk of stroke. However, anticoagulation or short-term dual antiplatelet therapy may be considered if additional symptomatic embolic events occur despite adherence to antiplatelet therapy. If the patient has undergone an endovascular stent placement, a minimum of 6 months of dual antiplatelet therapy is required.

Acute Ischemic Stroke Treatment

Early thrombolysis (up to 4.5 h after symptom onset) with intravenous tissue plasminogen therapy (tPA) remains the gold standard for acute ischemic stroke. Administration of tPA to BCVI patients requires careful consideration of concomitant injuries. Those with intracranial hemorrhage, clinical or radiographic evidence of head trauma, or aortic dissection should not be treated with IV tPA. Thrombolytics can be used safely in patients with extracranial cervical arterial dissection, but less is known regarding safety with intracranial dissection [72, 76, 77], which carries an increased risk of hemorrhage. In cases of confirmed or suspected aortic arch dissection, tPA should not be used. Current guidelines suggest careful consideration of IV tPA administration in patients with major trauma (excluding head trauma) or surgery within the preceding 14 days, weighing the risk of bleeding from injuries against the severity and potential disability from stroke [72]. Tenecteplase may be considered for intravenous thrombolysis as an alternative to alteplase [78], although this has not been selectively tested in BCVI patients.

In addition to thrombolysis, mechanical thrombectomy may be considered in BCVI patients within 6 h of stroke symptom onset if accompanied by an acute occlusion of the proximal cerebral vasculature on imaging. Good outcomes have been reported with mechanical thrombectomy in patients with carotid artery dissection and intracranial occlusion, with or without stenting [79, 80]. The recently published DEFUSE-3 and DAWN trials demonstrated benefit from mechanical thrombectomy in treatment of acute stroke due to large vessel occlusion up to 16 or 24 h respectively from time of last known normal in very carefully selected patients [81, 82]. While these studies did not specifically include trauma patients, and dissection with flow-limiting stenosis requiring stenting was an exclusion criterion for the DAWN trial, endovascular intervention within the extended time window could be considered for BCVI patients. Taken together, these data suggest potential benefits for mechanical thrombectomy for acute stroke secondary to large-vessel occlusion in the anterior circulation. More data are needed regarding safety and benefits in the BCVI population.

It is very important to keep in mind that there is a diminished benefit of acute stroke therapy with any delay between symptom onset and time of intervention, so rapid identification and treatment of stroke is paramount for good neurologic outcomes. Trauma patients may be intubated, sedated, or paralyzed, and have significant injuries obscuring portions of the neurologic examination. Furthermore, as trauma providers lack routine expertise in neurologic examination and management of ischemic stroke, patients may miss the short-treatment window due to lack of early stroke recognition. A close relationship between trauma surgeons, vascular neurology, neurosurgery, and other neurointerventionalists can help improve timely and appropriate treatment of ischemic stroke due to BCVI.

Conclusions

BCVI is a potentially preventable cause of stroke, a cerebrovascular disorder that can result in significant morbidity and mortality. Most patients are neurologically asymptomatic at time of presentation, or may have a neurological evaluation obscured due to critical illness, and thus a high index of suspicion is needed to identify BCVI during the window for stroke prevention or acute treatment. In light of this, screening with CTA in all patients with injuries that would otherwise prompt CT scans of the neck or chest is recommended. Treatment of BCVI is guided by grade of injury. Early treatment with antithrombotics has been shown to be both effective and safe. Rarely will patients require surgical intervention for their BCVI. Acute stroke therapy with thrombolytics or mechanical thrombectomy is possible, but special consideration must be given to frequent comorbidities in trauma patients, and a multidisciplinary team effort for rapid identification and treatment of acute ischemic stroke is critical.

Acknowledgements The authors would like to thank Dr. Howard S. Kirshner for the topic title and initial topic review.

Compliance with Ethical Standards

Conflict of Interest Christina A. Wilson reports personal fees from UpToDate, personal fees from Medlink Neurology, outside the submitted work. David K. Stone and Vyas T. Viswanathan each declare no potential conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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- • Of importance
- •• Of major importance

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