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BLUNT CEREBROVASCULAR INJURY

Evidence Based Medicine Guideline

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SUMMARY

Blunt cerebrovascular injuries (BCVI) are identified in up to 3% of all blunt trauma. These injuries are associated with significant morbidity and mortality if not appropriately diagnosed and managed in a timely manner. CT angiography remains the screening modality of choice and antithrombotic therapy the mainstay of treatment.

RECOMMENDATIONS

- **Level 1**
  - **None**
- **Level 2**
  - **Patients should be screened for BCVI according to the Expanded Denver Criteria:**
    - **Patients with the following signs/symptoms:**
      - **Potential arterial hemorrhage from neck/nose/mouth**
      - **Cervical bruit in patients < 50 years of age**
      - **Expanding cervical hematoma**
      - **Focal neurological defect**
      - **Neurological abnormality inconsistent with CT head or stroke on CT/MRI**
    - **Patients with the following risk factors associated with high-energy transfer mechanism:**
      - **High-energy transfer mechanism**
      - **Le Fort II or III fractures / mandible fracture**
      - **Complex skull/basilar/occipital condyle fracture**
      - **Severe TBI with GCS < 6 or associated thoracic injury**
      - **Cervical spine fracture or ligamentous injury at any level**
      - **Near hanging with anoxic brain injury**
      - **Seatbelt abrasion with significant swelling, pain, or acute mental status change**
      - **Thoracic vascular injuries**
      - **Scalp degloving**
      - **Blunt cardiac rupture**
      - **Upper rib fractures**
  - **CT angiography is the screening modality of choice**
- **Level 3**
  - **Patients who present with potential BCVI should be considered for CT angiography during initial CT imaging**
  - **Early initiation of antithrombotic therapy (Aspirin 81mg PO daily) is recommended for the prevention of stroke-related morbidity and overall mortality for BCVI Grades I-IV**
  - **Endovascular stenting should not be routinely used to treat Grade II/III BCVI**
  - **Follow-up CT angiography should be performed in 7-10 days to monitor response to therapy**

LEVEL OF RECOMMENDATION DEFINITIONS

- **Level 1:** Convincingly justifiable based on available scientific information alone. Usually based on Class I data or strong Class II evidence if randomized testing is inappropriate. Conversely, low quality or contradictory Class I data may be insufficient to support a Level I recommendation.
- **Level 2:** Reasonably justifiable based on available scientific evidence and strongly supported by expert opinion. Usually supported by Class II data or a preponderance of Class III evidence.
- **Level 3:** Supported by available data, but scientific evidence is lacking. Generally supported by Class III data. Useful for educational purposes and in guiding future clinical research.

INTRODUCTION

Blunt cerebrovascular injuries (BCVI) describe non-penetrating traumatic injuries involving the carotid and/or vertebral vessels. The most common mechanisms of injury are either hyperextension/flexion and rotation of the neck or a direct blow to the neck. Various types of vascular injury may occur including the development of intimal flap/dissection, occlusion/thrombosis, pseudoaneurysm, complete transection or a combination of these lesions (1). Mortality rates associated with BCVI range from 20-30%. Stroke remains the most feared complication occurring in up to 20% of patients (2). Despite this, patient selection for screening has remained controversial given limited studies. Because outcome is compromised by diagnostic delay or lack thereof, a high index of suspicion should be maintained for those patients at risk. The goal should be to diagnose and treat these injuries early prior to the patient becoming symptomatic through diagnostic screening programs as delays in treatment can be catastrophic. With the advent of multi-detector helical scanners, computed tomography angiography (CTA) has become the screening modality of choice. A grading scale was developed to standardize injury severity (Table 1) and guide therapy. Current guidelines recommend antithrombotic therapy, endovascular therapy, or surgical repair based upon the grade (2,3).

TABLE 1: Denver Grading Scale for BCVI

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

LITERATURE REVIEW

Incidence and Screening

In a landmark paper in 1996, Fabian's group from Memphis retrospectively reviewed the records of 67 patients with 87 blunt injuries treated over nearly 11 years and found that blunt carotid injury (BCI) was present in 0.67% of patients admitted after motor vehicle crashes (one in every 150 patients), and in 0.33% of blunt injuries in general (one in every 304 patients) (4). The circumstances that prompted clinical suspicion and angiographic diagnosis for BCI were: 1) physical findings demonstrating soft-tissue injury to the anterior neck (41%); 2) a neurological examination that was not compatible with the brain CT (34%); 3) development of a neurological deficit after hospital admission (43%) and 4) Horner's syndrome (9%). Overall mortality rate was 31%, with 76% of the deaths directly related to BCI-induced strokes. Of the 46 survivors, 63% had good neurological outcome, 17% had a moderate outcome, and 20% had a bad outcome. Most of the patients were treated with heparin, which was the only significant factor associated with improvement in neurological outcome ($p< 0.01$).

In 1997, Parikh et al. performed a retrospective review on patients sustaining BCI over a 6-year period (5). Those patients with combined head and chest trauma were found to have a 14-fold increase in the likelihood of carotid injury. In this series, there was an incidence of 0.24% among blunt trauma patients. 53% of patients presented with abnormal neurological finding not explained by cranial CT. Focal neurological findings were present in 42% of patients. Of the survivors, 45% of patients had significant impairment. The overall mortality was 5%.

Biffl et al. found the overall incidence of BCI to be 0.38% (6). However, with more aggressive screening of asymptomatic patients, the incidence was found to be 1.07%. Head injury was present in 61% and basilar skull fractures with involvement of the carotid canal in 25%. Facial fractures were present in 34% and cervical spinal column fractures in 25%. Overall mortality was 17% and, of the survivors, 32% suffered permanent severe neurological disability. Stroke rate increased with grade. 7% of Grade 1 injuries progressed as compared to 70% of Grade 2 injuries despite anticoagulation. Heparin therapy was protective against stroke in patients with Grade 4 injury. Grade 5 injuries were typically found to be devastating.

In another study, Biffl et al. identified the following four risk factors for carotid artery injury based on multiple regression analysis: 1) GCS score ≤ 6, 2) petrous bone fracture, 3) diffuse axonal brain injury, and 4) Le Forte II or III fractures (7). If a patient had all 4 risk factors, they had a 93% probability of carotid artery. If the patient had a cervical spine fracture, they had a 33% probability of having a vertebral artery injury (VAI).

Berne et al. reported a BCVI incidence of 0.49% (8). Overall mortality was 59% with 80% of the deaths directly attributable to the BCVI. The median time until diagnosis was 12.5 hours in all patients with BCVI and 19.5 hours in non-survivors. Five patients had a delay in diagnosis of greater than 48 hours, all of which developed complications. Six patients presented with a GCS of 15 and four of them died.

Miller et al. prospectively evaluated an aggressive screening protocol utilizing angiography (9). The screened population included all patients with cervical spine fractures, LeFort II or III facial fractures, Horner's syndrome, skull base fractures involving the foramen lacerum, neck soft tissue injury or neurological abnormalities unexplained by intracranial injuries. They identified an incidence of BCVI of 1.03% among blunt admissions and had a screening yield of 29%. 79% of patients with carotid artery injury (CAI) were diagnosed before the onset of ischemia.

Cothren et al. identified VAI in 33% of those with cervical spine fractures (10). Overall mortality in the group screened was 5%. Stroke rate in those patients with BCVI was 33% (all carotid injuries). This study clearly shows that screening patients at risk identifies injuries. It also demonstrates the improvement of morbidity and mortality with earlier diagnosis.

Miller et al. followed up their previous studies with a 5-year review after a heightened institutional awareness for these types of injuries (11). Angiography was used liberally for the following indications: neurological deficit not consistent with brain imaging, neck hematoma, Horner's syndrome, basilar skull fracture through the foramen lacerum, cervical spine fracture through the transverse foramen, and severe complex facial fracture. CAI was diagnosed in one of every 191 blunt trauma patients (0.5%) and VAI was diagnosed in one of every 233 blunt trauma patients (0.4%). Stroke attributable mortality was less than the 24% in the previous study ($p=0.03$). Signs of ischemia were less (34% vs. 77%; $p<0.001$) leading one to believe that these lesions were being diagnosed and treated earlier with resultant improved outcomes. Those diagnosed and treated prior to the onset of ischemia had better outcomes. As in the prior study, those treated with heparin had better neurological outcomes.

Kerwin et al. evaluated their aggressive BCVI screening program in 48 patients among whom 21 injuries were identified (44% of those screened) (12). The overall incidence of BCI/BVI for blunt trauma was 1.1%. The frequency of abnormal angiograms for the indications listed was fracture through the foramen transversarium (60%), unexplained paresis (44%), basilar skull fracture (42%), unexplained neurological examination (38%), and anisocoria (33%).

Burlew et al. prospectively evaluated all patients presenting to Denver Health Medical Center between 1997 and 2010 who were diagnosed with BCVI (13). They found 418 patients with BCVI during that period. Of these patients, 313 underwent evaluation based on previously defined risk factors for screening, 83 had no risk factors for screening, and 22 presented with neurological symptoms. Of the 83 patients who did not meet current guidelines for screening, 22 had mandible fractures. They also found that 15 patients with BCVI had basilar skull fractures or occipital condyle fractures that did not extend to carotid canal and would therefore not have been screened based on previous guidelines. The authors concluded that the importance of injury pattern alone as a screening tool is not sufficient for identification of patients at risk for BCVIs. They recommended considering mechanism of injury combined with injury pattern to determine patients at greatest risk of BCVIs who should undergo further screening. They recommended the use of CTA alone based on screening criteria for evaluation and diagnosis.

Many screening criteria have been proposed over the years to identify patients at risk. Studies performed at the Denver Health Medical Center and the University of Tennessee Health Science Center proposed the Denver Criteria and the Memphis Criteria (Tables 2 and 3 respectively) (2). The Denver Criteria initially suggested screening in severe cervical hyperextension / rotation / hyperflexion injuries or in the presence of cervical body fractures, while the Memphis Criteria suggested screening in all cervical spine fractures. In 2007, the Denver group suggested a modification to include only specific cervical spine fracture patterns (complex cervical spine fractures such as subluxation, extension into the foramen transversarium, or upper C1–C3 fractures) for prompt screening.

TABLE 2. Memphis Screening Criteria
Unexplained neurological deficit
Horner's syndrome
LeFort II or III (unilateral or bilateral)
Cervical spine injury
Skull base fractures involving the foramen lacerum
Neck soft tissue injury (e.g., seatbelt injury or hanging)

TABLE 3. Denver Screening Criteria
Signs/symptoms of BCVI
Potential arterial hemorrhage from neck/nose/mouth
Cervical bruit in patient <50 years of age
Expanding cervical hematoma
Focal neurological deficit
Neurological deficit inconsistent with head CT
Stroke on CT or MRI
Risk factors for BCVI
High-energy transfer mechanism
Displaced midface fracture (LeFort II or III)
Mandible fracture
Complex skull/basilar/occipital condyle fracture
Severe TBI with GCS <6
Cervical spine fracture, subluxation, or ligamentous injury at any level
Near hanging with anoxic brain injury
Clothesline type injury/seat belt abrasion with swelling, pain, or altered mental status
TBI with chronic thoracic injuries
Scalp degloving
Thoracic vascular injuries
Blunt cardiac rupture
Upper rib fractures

Later studies showed that nearly 20% of cases of BCVI were missed using the Modified Denver Criteria. In 2016, Geddes et al. found that there was a significantly increase in the identification of BCVI following the introduction of the expanded screening criteria (14). They identified the overall incidence of BCVI increased to 2.99% from 2.36% and the BCVI-related neurologic event incidence decreased from 12% to 9.3%. Therefore, the Denver group modified the screening for a second time creating the Expanded Denver Criteria in 2012 to include mandible fractures, complex skull fractures, traumatic brain injury with thoracic injuries, scalp degloving, and thoracic vascular injuries.

Due to improved imaging technology and introduction of standard screening protocols, the reported incidence of BCVI has increased over the years from 0.1% to 1-2% (15). In a 10-year retrospective single center study, Hundersmarck et al. found an overall BCVI incidence of 0.59%. Forty-two percent of all BCVI patients had associated traumatic brain injury. The overall incidence of BCVI among the blunt trauma patients, polytrauma, basilar skull fracture, and cervical trauma was 0.59%, 1.5%, 1.6%, and 7.3% respectively. The cervical spine injury subgroup showed the strongest association followed by polytrauma, and basilar skull fractures.

Leichtl et al. hypothesized that only universal screening would identify clinically relevant BCVIs. In their study, 4659 adult blunt trauma activations were screened with full body CT including CT angiography of the neck. They found that 23% of patients with BCVI Grades 3 or higher would not have been screened and subsequently found to have a BCVI if following the Denver Criteria and the ACS Trauma Quality Improvement Program Best Practices

Guidelines. Given their results, they concluded that programs across the country should consider the implementation of universal screening (16).

Method of Evaluation

In a prospective evaluation completed in 2004, Berne and colleagues evaluated the use of Helical CT as a method for BCVI screening (17). Their indications for screening were similar to those described above. Angiogram was performed only if the studies were equivocal or unclear for any reason. The incidence of BCVI was 0.60% for all blunt trauma patients and 3.7% among those screened. Results of CTA for BCVI were as follows: sensitivity, 100%; specificity, 94%; positive predictive value (PPV), 37.5%; and negative predictive value (NPV), 100%. It should be noted that these CTAs were not all compared to the gold standard angiogram, thus a true sensitivity and specificity was not obtained. Frequent clinical examinations were performed to determine if a patient did not have an injury. Perhaps low-grade injuries were missed that did not have clinical consequence. Another part of this study deserves mention. The study was initially done with a 4-slice scanner and subsequently with a 16-slice scanner. In comparing these two subgroups, the incidence of BCVI increased (0.38% to 1.05%) and the prevalence increased (2.5% to 6.9%) with the use of the newer technology. Specificity increased from 90.8% to 98.7% and PPV improved from 22.2% to 83.3%.

In 2005, Bub et al. performed a retrospective review of patients that had both a CTA and angiography that were independently reviewed by radiologists in a blinded fashion (18). There was not a protocol in place with regard to which patients received these studies. 32 patient studies were evaluated. Most of the CTA studies were performed on a 4-slice scanner. Among the three radiologists reviewing the studies, sensitivity of CTA for CAI ranged from 83% to 92% and specificity from 88% to 98%. For VAI, sensitivity was 40% to 60% and specificity was 90% to 97%. Interestingly, inter-observer variability was greater for angiography than CTA reinforcing the fact that even the gold standard is not perfect.

In 2006, Schneidereit et al. evaluated the use of an eight-slice multi-detector CT scanner (19). The overall incidence of BCVI increased from 0.17% to 1.1% with the use of aggressive screening protocols. Delayed stroke rate and injury-specific mortality fell from 67% to 0% ($p<0.001$) and 38% to 0% ($p=0.002$) respectively. Mortality fell from 38% to 10.5% ($p=0.049$). When evaluating patients that had both CTA and angiography performed, the specificity for CTA was found to be 58%. No patient with a negative screening CTA developed delayed neurological sequelae attributable to BCVI. Using univariate logistic regression analysis, only the presence of a cervical spine injury was predictive of BCVI.

Four months later, three consecutive issues of the Journal of Trauma contained reports evaluating CTA in the evaluation of BCVI. Biffl and colleagues evaluated 16-slice CTA (331 patients) and concluded that it detected all clinically significant injuries (20). 5.4% of those screened and 0.66% of blunt trauma admissions were found to have suffered a BCVI. Four patients had false-positive CTA studies. No patient with a normal CTA developed neurological signs or symptoms consistent with BCVI.

In the following issue, Eastman and colleagues further evaluated CTA with the use of a 16-channel scanner and did a prospective, head-to-head comparison with angiography (21). The overall incidence of BCVI was 1.25% and the incidence within the screened population was 28.4%. 146 patients received both CTA and angiography and 43 had a BCVI. In 98%, the results of the CTA and angiography were concordant. There was a single false negative CTA in a patient with a Grade I vertebral injury. The overall sensitivity, specificity, PPV, NPV, and accuracy for the diagnosis of BCVI were 97.7%, 100%, 100%, 99.3%, and 99.3% respectively. The authors concluded that a 16-channel, multi-slice CTA is a safe, effective and sensitive diagnostic test modality for the detection of BCVI.

Berne and colleagues then reported their 2-year experience with 16-slice multi-detector CTA in the evaluation of BCVI (22). 435 patients were screened, and 24 patients had injuries for an overall incidence of 1.2% and 5.5%. These results were statistically higher than their numbers during the “4-slice era” (0.38% and 2.4% respectively, $p<0.01$). Overall mortality was 25%. Angiograms were performed to further evaluate positive studies. No patient with a negative CTA was subsequently identified as having or developed neurological symptoms attributable to a missed BCVI.

In 2011, Emmet et al investigated screening modalities for patients with high risk of BCVI (23). During a 29-month period, 748 out of 20,049 patients were identified who had undergone a subtraction angiogram after blunt force trauma. Of these patients, 117 were found to have a BCVI with an incidence of 16% in the screened population.

They found that the number of screening criteria present increased the incidence of BCVI. Patients with only one screening criteria had between an 6-17% rate of BCVI, while patients with two or more screening criteria had an incidence of 22%. Of these patients, 44 underwent angiography based on CTA abnormalities alone with 19 patients being diagnosed with BCVI based on angiography. Only five patients underwent angiography after developing symptoms after initial CTA evaluation was negative, of these only one patient had BCVI. Based on these results, the authors suggested that CTA be used as an additional screening trigger and not a screening test due to the poor sensitivity of the test.

In 2018, Brommeland et al. performed a systemic literature review to create best practice guidelines (24). In their study, they found that although digital subtraction angiography (DSA) remains the gold standard in detecting BCVI, it is time-consuming, carries higher procedure-related risks, and is it not readily available at all institutions. They found that modern CT scanners have increased sensitivity and specificity, are more cost-effective, and have been attributed to reduced time from injury to diagnosis and subsequent earlier implementation of medical therapy.

Management and Follow-up

Chothern and colleagues conducted a prospective observational study evaluating the efficacy of continuous infusion heparin in reducing stroke rate in patients with BCVI (25). The patient population included those with blunt trauma who were admitted to a Level I trauma center. Of the 13,280 patients admitted, 643 patients underwent diagnostic four-vessel cerebrovascular angiography and 114 patients were identified as having carotid artery injuries. Of those with blunt carotid artery injury, 73 asymptomatic patients received one of four different anticoagulation regimens: 1) continuous infusion heparin, 2) subcutaneous low-molecular weight heparin, 3) anti-platelet agents, or 4) no therapy. Fifty-four patients (74%) received continuous infusion heparin at 15 units/kg/hour without a loading dose, titrated to achieve aPTT between 40 to 50 seconds. Two patients (3%) received dalteparin sodium 5000 units once daily. Seventeen patients (23%) received antiplatelet agents (aspirin or clopidogrel). Forty-one patients did not receive anticoagulation. Ischemic neurological events did not occur in any patient who received anticoagulation while they developed in 46% of those who did not receive any type of anticoagulation therapy confirming the value of anticoagulation in BCVI. The authors did not comment on the comparative efficacy of the different pharmacological therapies.

Fabian and colleagues conducted a retrospective chart review of patients with BCVI admitted to a Level I trauma center over almost ten years (26). Sixty-seven patients with 87 BCIs were treated. There were 54 intimal dissections, 11 pseudoaneurysms, 17 thromboses, 4 carotid cavernous fistulas, and 1 transected internal carotid artery. Anticoagulation with heparin was initiated at the time of diagnosis in 76% of patients while others received no treatment. The goal of heparin therapy was a partial thromboplastin time (aPTT) of 40-50 seconds. Heparin therapy was maintained for 1-3 weeks. Following heparin therapy, warfarin was initiated and continued for 3-6 months with a target prothrombin time of 15-18 seconds and an international normalized ratio (INR) of 1.8. Heparin therapy was independently associated with improvement in neurologic outcomes and survival. Heparin therapy resulted in decreased mortality when compared with those not on heparin treatment (20% vs. 100%, $p<0.01$).

Anti-platelet therapy was compared with anticoagulation therapy in a study by Wahl and colleagues (27). A retrospective chart review was performed on all adult patients admitted to a trauma center during a seven-year period. Twenty-two patients were identified to have BCI injuries with eight patients being observed without heparin or antiplatelet agent therapy. Of these, two patients died of head injuries and the remaining six survived with fair to good neurological exams at discharge. Seven of 22 patients were treated with heparin with aPTT goal of 40-60 seconds. Heparin therapy was started within the first 24 hours after admission in three of seven patients. There were four major bleeding complications which resulted in discontinuation of heparin. None of these patients were over anticoagulated. All survivors had fair to good neurological exams at discharge and two patients were discharged on warfarin. Of the remaining seven patients who received antiplatelet therapy, all had fair to good neurological exams at discharge. There were no differences in neurologic outcomes between those patients receiving anticoagulation and those receiving antiplatelet therapy. Heparin therapy resulted in a significantly higher rate of bleeding complications ($p=0.05$) (Table 4).

TABLE 4. Contraindications/Precautions to Heparin Therapy
Hypersensitivity to heparin
Active bleeding
Severe thrombocytopenia
Increased risk of hemorrhage, such as:
Traumatic injuries e.g., liver laceration, intracranial hemorrhage, spinal cord injury
Dissecting aneurysm
Hemophilia or other blood disorders
Epidural catheter
Subacute bacterial endocarditis
Uncontrolled hypertension

Perry and colleagues published a retrospective case series describing 17 patients with BCI (28). They found a decrease in morbidity and mortality in those patients that were treated with surgical intervention vs. observation alone. Martin et al. performed a retrospective review of eight patients over 10 years who had suffered blunt trauma to the carotid vessels (29). Three of the patients were treated surgically, one patient was anti-coagulated with heparin, and the other four patients were observed without any specific treatment. Of the three patients undergoing surgical intervention, all had complete neurological recovery or remained asymptomatic.

The challenge with surgical management of BCVI is that most lesions are surgically inaccessible. With advances in endovascular stenting, BCVI that are not amenable to open surgical repair may now be effectively treated. Duke et al. reported on six patients undergoing stenting for pseudoaneurysm of the carotid artery after blunt trauma (30). All patients were anticoagulated with heparin followed by coumadin for eight weeks, and then aspirin for another four weeks. On repeat angiography, five of the patients had complete healing of their injuries at five months and one at seven months. No stenosis or thrombosis was reported in this series during the 2-7-month follow-up. A more recent study published in 2007 by Edwards and his colleagues evaluated antithrombotic therapy and endovascular stents as treatment for blunt carotid injuries (31). One hundred ten patients over a 10-year period were diagnosed with 133 BCVI (23 bilateral). Twenty-two endovascular stents were place (18 for pseudoaneurysms and four for dissection). These patients had a mean follow-up of 30 months, none of which experienced any peri-procedural complications.

In a recent study by Cothren et al., the risks and benefits of endovascular stenting for BCVI were evaluated (32). They performed an analysis of their database over an eight-year period. Forty-six patients were identified as having a pseudoaneurysm after BCVI. Patients with persistent pseudoaneurysm on repeat imaging (7-10 days) were candidates for stenting. All patients without contraindications received anticoagulation therapy. Of those 46 patients, 23 had stents placed. Four patients suffered complications during stent placement: three strokes and one subclavian artery dissection. Follow-up angiography was obtained on 38 of the patients: 18 who had undergone stenting plus antithrombotic therapy and 20 who received antithrombotic therapy alone. Carotid occlusion rates were found to be 45% in the stent group vs. 5% in the antithrombotic therapy alone group.

Biffl et al. performed a database review over an 11-year period (33). During that time, 171 patients were diagnosed with BCVI. The injury grade breakdown was 137 Grade I, 52 Grade II, 32 Grade III, 25 Grade IV, and 8 Grade V. One hundred fourteen (73%) carotid and 65 (67%) vertebral arteries were restudied with arteriography 7 to 10 days after the injury. 82% of Grade IV and 93% of Grade III injuries were unchanged. However, 57% of Grade I and 8% of Grade II injuries healed, allowing cessation of therapy, whereas 8% of Grade I and 43% of Grade II lesions progressed to pseudoaneurysm formation, prompting surgical intervention. There was no significant difference in healing or in progression of injuries whether treated with heparin or antiplatelet therapy or untreated. However, they believe heparin may improve the neurologic outcome in patients with ischemic deficits and may prevent stroke in asymptomatic patients.

The 2020 Eastern Association for the Surgery of Trauma Guidelines analyzed the literature comparing ten studies (2). A total of 713 patients received antithrombotic therapy (ATT) and 253 did not. Stroke rates were 9.8% and 33.6% respectively. Mortality was 16.6% in the ATT group vs. 40.4% in the non-ATT group. They recommended initiation of ATT once the diagnosis of BCVI is established with careful consideration for bleeding complications.

The routine use of endovascular stenting has remained controversial. For the majority of BCVI, ATT alone is adequate. Stenting is typically reserved for higher-grade lesions, such as dissections with significant narrowing (Grade II), enlarging pseudoaneurysms, or Grade V lesions not surgically accessible (3). Earlier studies had promoted stenting, but more recent studies have shown complications including in-stent thrombosis and neurological deficits. The 2020 Eastern Association for the Surgery of Trauma Guidelines analyzed three studies. A total of 171 patients underwent stenting for Grades II and III and 323 patients did not and received ATT alone. The stroke rate was found to be higher in the stenting group, 4.1% vs. 2.2%. The guidelines committee concluded that the potential benefits of endovascular stenting do not outweigh the potential harms.

TABLE 5. Recommended Management of BCVI based on grade (2)

Injury grade	I	II	III	IV	V
Initial therapy	ATT	ATT	ATT	ATT	Direct pressure
Surgical/endovascular therapy	Not needed	Rarely needed	Consider if symptomatic	Stenting not beneficial; thrombectomy with/without stenting if recognized within 6h	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	Based on symptoms	
Long-term therapy	Until healed			Lifelong therapy	Unknown

ATT: Antithrombotic therapy

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