BLUNT CEREBROVASCULAR INJURIES

SUMMARY

Blunt cerebrovascular injury (BCVI) may be present in greater than 1% of those with blunt trauma. Aggressive screening strategies uncover injuries in up to 44% of those screened. These injuries are responsible for significant morbidity and mortality if not appropriately diagnosed and treated in a timely.

RECOMMENDATIONS:

- **Level 1**
  - **None**
- **Level 2**
  - **Patients with the following injury patterns / presentation should be screened:**
    - **Coma unexplained by CT findings**
    - **Lateralizing neurological deficits**
    - **Cervical spine injuries**
    - **Lefort II or III facial fractures, mandible fractures, or skull base fractures involving the foramen lacerum**
    - **Severe epistaxis**
    - **Horner's syndrome**
    - **Significant neck soft tissue injury / neck seat belt sign**
    - **A history of strangulation or near hanging**
  - **CT angiography is the preferred method to screen for BCVI**
  - **Follow-up CT angiography should performed, for Grade I-III lesions, 7-10 days following injury to monitor response to therapy**
- **Level 3**
  - **Heparin therapy is safe and should be considered if:**
    - **No contraindications are present AND the anticipated benefit outweighs the risk of bleeding in patients at high risk**
    - **Warfarin therapy should be considered following treatment with heparin**
  - **Anti-platelet therapy may be considered in patients without contraindications**
  - **Therapy should be continued for 3 to 6 months**
  - **In patients with minimal or no symptoms, open repair of surgically-accessible Grade II-V lesions is recommended over endovascular intervention or no repair**
  - **Injury grade-specific recommendations**
    - **Grade I - antithrombotic therapy alone**
    - **Grade II & III - antithrombotic therapy + surgical intervention**
    - **Grade IV - antithrombotic therapy alone**
    - **Grade V - open repair vs. endovascular repair / angioembolization +/- antithrombotic therapy**

EVIDENCE DEFINITIONS

- **Class I:** Prospective randomized controlled trial.
- **Class II:** Prospective clinical study or retrospective analysis of reliable data. Includes observational, cohort, prevalence, or case control studies.
- **Class III:** Retrospective study. Includes database or registry reviews, large series of case reports, expert opinion.
- **Technology assessment:** A technology study which does not lend itself to classification in the above-mentioned format. Devices are evaluated in terms of their accuracy, reliability, therapeutic potential, or cost effectiveness.

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.

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INTRODUCTION

BCVI involving the carotid and/or vertebral vessels is a potentially devastating and commonly underdiagnosed injury. Most such injuries occur with rapid deceleration, hyperextension, and rotation of the neck, all of which stretch the internal carotid artery (ICA) and can produce an intimal tear with resultant thrombosis and embolization. Pseudoaneurysms may form that can bleed, enlarge, and compress adjacent structures or be a source of emboli. Many other mechanisms and patterns of injury may be present and are beyond the scope of this review. Because outcome is compromised by diagnostic delay, maintaining a high index of suspicion for these injuries among patients at risk is very important. Focal neurological deficits that do not correlate with cranial CT results are a common, yet often late finding. The ultimate goal should be to diagnose and treat this injury early *prior to* the patient becoming symptomatic through the use of broad scale diagnostic screening programs directed at populations at risk as delays in treatment can be catastrophic.

After one decides that an individual is at increased risk for injury to the extracranial cerebrovasculature, the next question centers around choosing the appropriate diagnostic test. Angiography has long been considered the gold standard. This test is invasive, costly, time consuming, and requires specialized staff that may not be immediately available. Early reports evaluating the diagnostic accuracy of CT angiography (CTA) and magnetic resonance angiography (MRA) showed that these modalities may miss injuries that are clinically significant. With the advent of multi-detector helical scanners, however, it appears that CTA is the diagnostic test of choice to detect these injuries.

Blunt carotid injury (BCI) is a rare but potentially devastating injury with a reported incidence of 0.08 to 0.27% (1). BCI results in severe disability or death if undiagnosed. Rates of mortality and neurologic morbidity ranging from 5-40% and 40-80%, respectively, have been reported among patients presenting with BCI (1). The mechanism of injury is usually due to direct trauma mainly from motor vehicle crashes followed by assaults and falls (2). Various types of vascular injury may occur, including the development of intimal flap/dissection, occlusion/thrombosis, pseudoaneurysm, carotid cavernous fistula, complete transection or combination of these lesions (1). Complications of BCI may result from either damage to the arterial wall or intima. Dissection due to arterial wall damage results in hemodynamic instability and damage to the arterial intima exposes subendothelial collagen which is a thrombogenic surface and potent platelet aggregator (1).

The optimal management of BCI is not well-defined. Surgical intervention, anticoagulation and antiplatelet therapy have been described. The goal of anticoagulation therapy is to prevent cerebral embolization and avoid permanent occlusion (1). Several studies have evaluated the use of systemic heparin as the anticoagulant of choice for BCI.

LITERATURE REVIEW

Incidence / Identifying Groups At Risk

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 (1 in every 150 patients), and in 0.33% of blunt injuries in general (1 in every 304 patients). 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 subsequent to 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. The majority of the patients were treated with heparin, which was the only independently significant factor associated with improvement in neurological outcome ($p< 0.01$) (3).

In 1997, a retrospective review was performed on patients sustaining BCI over a 6 year period (n=20) (4). 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% (4).

In an attempt to further classify these types of injuries, Biffl et al. described a BCVI grading scale in 1999:

Grade 1 = irregularity of the vessel wall or luminal dissection with less than 25% luminal stenosis
Grade 2 = intraluminal thrombus OR
a raised intimal flap OR
dissections with associated luminal narrowing more than 25%
Grade 3 = pseudoaneurysms
Grade 4 = vessel occlusions
Grade 5 = complete vessel transection with free contrast extravasation.

Biffl found the overall incidence of BCI to be 0.38%. However, with more aggressive screening of asymptomatic patients, the incidence was found to be an astonishing 1.07%. Head injury was present in 61%, and 25% had basilar skull fractures with involvement of the carotid canal. Facial fractures were present in 34%, and 25% had cervical spinal column fractures. The 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 (5).

The following four risk factors for carotid artery injury were identified 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. 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) (6).

Berne et al. reported a BCVI incidence of 0.49%. Overall mortality was 59%, and 80% of the deaths were 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 4 of them died (7).

In 2002, Fabian et al. prospectively evaluated an aggressive screening protocol utilizing angiography. 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. VAI were found in 33% of those with cervical spine fractures. Overall mortality in the group screened was 5%. Stroke rate in those patients with BCVI in this series was 33% (all carotid injuries) (8,9). This study clearly shows that screening patients at risk identifies injuries. It also demonstrates the improvement of morbidity and mortality with earlier diagnosis.

In 2001, Fabian et al. followed up their previous studies with a 5 year review after a heightened institutional awareness for these types of injuries (10). 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 1 of every 191 blunt trauma patients (0.5%) and VAI was diagnosed in 1 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.

In 2001, Kerwin et al. evaluated their aggressive BCVI screening program in 48 patients among whom 21 injuries were identified (44% of those screened). 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%) (11).

In 2002, Miller et al evaluated Fabian's screening protocol by obtaining a four vessel angiography on 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 those with neurological abnormalities unexplained by intracranial injuries (12). Overall screening yield was 29%. The incidence of CAI was similar to the previous study, however, the incidence of VAI increased (10,13). The stroke rate in VAI was markedly lower at 0% as compared to 14% in the previous study.

In 2003, Biffl et al. sought to determine which cervical spine fracture patterns were predictive of VAI's through the use of a prospective, aggressive screening protocol using angiography. 605 patients underwent diagnostic angiography and 92 patients were found to have a VAI (15% of those screened). 77% had associated cervical spine fractures and the majority of the fracture patterns were subluxations (55%) or involved extension of the fracture through the transverse foramen (26%). Excluding subluxations, 13 of 15 patients with VAI's and fractures were located in the upper spine from C1 to C3. 9 patients had an associated CAI. One third of patients with these high-risk fracture patterns had a VAI (9).

Although many centers have decided to implement screening protocols, there is still a general lack of consensus. The American Association for the Surgery of Trauma (AAST) has recently opened a multicenter study to prospectively collect data on screened patients.

In 2012, Burlew et al. out of Denver Health Medical Center, looked at all patients prospectively between 1997 and 2010 who were diagnosed with BCVI. They found 418 patients 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 BCVIs had basilar skull fractures or occipital condoyle 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 (12).

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. Their indications for screening were similar to those described above. Angiograms were only performed 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 CTA's 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 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% (13).

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. 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 (14).

In 2006, Schneidereit evaluated the use of an eight-slice multi-detector CT scanner. 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% (there were 8 falsely positive CTA studies for a variety of reasons). 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 (15).

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. 5.4% of those screened and 0.66% of blunt trauma admissions were found to have suffered a BCVI. 17 injuries were imaged with both CTA and angiography. Four patients had false-positive CTA studies. No patient with a normal CTA developed neurological signs or symptoms consistent with BCVI (16).

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, which was the largest study to date. 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, multislice CTA is a safe, effective and sensitive diagnostic test modality for the detection of BCVI (17).

Most recently, Berne and colleagues reported their 2 year experience with 16-slice multi-detector CTA in the evaluation of BCVI. 435 patients were screened and 24 patients had injuries for an overall incidence of 1.2% and 5.5% of those screened. 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. Interestingly, a detailed description of the angiography findings was not disclosed in the study. No patient with a negative CTA was subsequently identified as having, or developed neurological symptoms attributable to a missed BCVI (18).

In 2011, Emmet et al looked into screening modalities for patients with high risk of BCVI. 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 that a patient had 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 5 patients underwent angiography after developing symptoms after initial CTA evaluation was negative, of these only 1 patient had BCVIs. 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. (19)

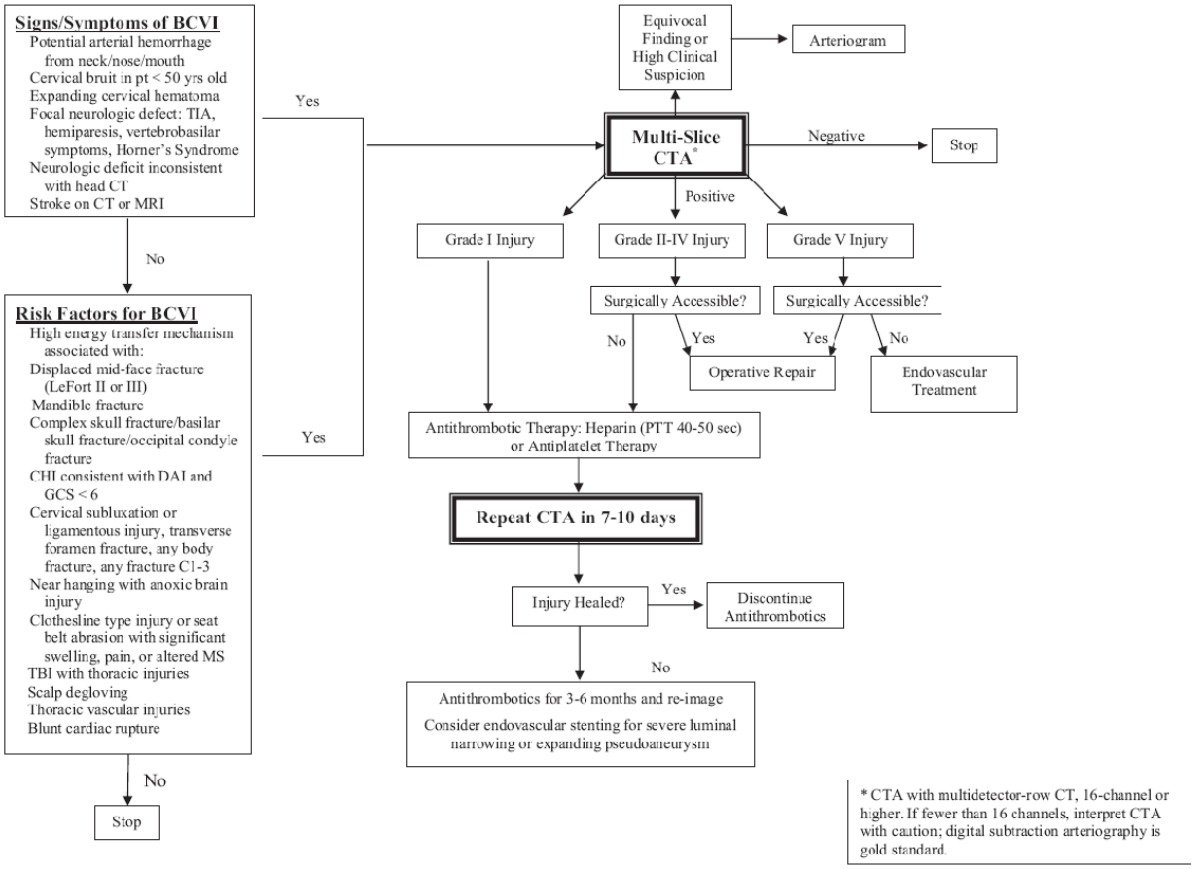

Figure 1: The New Denver Health Medical Center BCVI Screening Guideline (12)

Treatment Recommendations

Cothern and colleagues conducted a prospective observational study evaluating the efficacy of continuous infusion heparin in reducing stroke rate in patients with BCI (20). The patient population included those with blunt trauma who were admitted to the Level I trauma center. Of the 13,280 patients admitted to the center, 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% (19/42) of those who did not receive any type of anticoagulation therapy. The authors did not comment on the comparative efficacy of the different pharmacological therapies in BCI (Class II).

Fabian and colleagues conducted a retrospective chart review of patients with BCI admitted to a Level I trauma center from December 1984 to September 1995 (21). 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 thromboplasin 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%, $p<0.01$) (Class III).

Anti-platelet therapy was compared with anticoagulation therapy in a study by Wahl and colleagues (22). A retrospective chart review were performed for all adult patients admitted to a trauma center from January 1992 to December 1999. Twenty-two patients were identified to have BCI injuries. Of the 22 patients, eight were observed, but not treated with heparin or antiplatelet agents. 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 a 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 ($p = 0.05$) (Class II).

Table 1: Contraindications/Precautions to Heparin Therapy

- Hypersensitivity to heparin
- Active bleeding
- Severe thrombocytopenia
- Increased risk of hemorrhage, such as:
  - Selected traumatic injuries (i.e. severe 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 in 1990 describing 17 patients with blunt carotid injury. They found a decrease in morbidity and mortality in those patients that were treated with surgical intervention vs observation alone (23). Martin et al. performed a retrospective review of eight patients over 10 years who had suffered blunt trauma to the carotid vessels. 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 (24). Based on this limited data, the Western Trauma Association and Eastern Association for the Surgery of Trauma have recommended surgical management for surgically accessible Grade II - V blunt cerebrovascular injuries (25, 26) (Class III). Indirect evidence of benefit for this approach comes from observational studies of patients with penetrating cerebrovascular injury, which have found lower rates of neurological morbidity and mortality for patients undergoing neck exploration and vascular repair compared with non-operative management.

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. 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 (27). A more recent study published in 2007 by Edwards and his colleagues evaluated antithrombotic therapy and endovascular stents as treatment for blunt carotid injuries. One hundred ten patients over a 10 year period were diagnosed with 133 BCI (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 (28). (Class III)

In a recent study by Cothren et al, the risks and benefits of endovascular stenting for BCVI was evaluated. 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 (29).

Monitoring Response to Therapy

Biffl et al. performed a database review over an 11 year period. During that time, 171 patients were diagnosed with BCVI. Mechanism of injury was MVC (50%), fall (11%), auto vs pedestrian (11%), or other (29%). One hundred fourteen patients had 157 carotid artery injuries (43 bilateral), and 79 patients had 97 vertebral artery injuries (18 bilateral). 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. Eight-two percent 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, heparin may improve the neurologic outcome in patients with ischemic deficits and may prevent stroke in asymptomatic patients (30). (Class II)

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