Blunt Cerebrovascular Injury Practice Management Guidelines: The Eastern Association for the Surgery of Trauma

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Background: Blunt injury to the carotid or vertebral vessels (blunt cerebrovascular injury [BCVI]) is diagnosed in approximately 1 of 1,000 (0.1%) patients hospitalized for trauma in the United States with the majority of these injuries diagnosed after the development of symptoms secondary to central nervous system ischemia, with a resultant neurologic morbidity of up to 80% and associated mortality of up to 40%. With screening, the incidence rises to 1% of all blunt trauma patients and as high as 2.7% in patients with an Injury Severity Score of ≥16. The Eastern Association for the Surgery of Trauma organization Practice Management Guidelines committee set out to develop an EBM guideline for the screening, diagnosis, and treatment of BCVI.

Methods: A computerized search of the National Library of Medicine/National Institute of Health, Medline database was performed using citations from 1965 to 2005 inclusive. Titles and abstracts were reviewed to determine relevance, and isolated case reports, small case series, editorials, letters to the editor, and review articles were eliminated. The bibliographies of the resulting full-text articles were searched for other relevant citations, and these were obtained as needed. These papers were reviewed based on the following questions: 1. What patients are of high enough risk, so that diagnostic evaluation should be pursued for the screening and diagnosis of BCVI? 2. What is the appropriate modality for the screening and diagnosis of BCVI? 3. How should BCVI be treated? 4. If indicated, for how long should antithrombotic therapy be administered? 5. How should one monitor the response to therapy?

Results: One hundred seventy-nine articles were selected for review, and of these, 68 met inclusion criteria and are excerpted in the attached evidentiary table and used to make recommendations.

Conclusions: The East Practice Management Guidelines Committee suggests guidelines that should be safe and efficacious for the screening, diagnosis, and treatment of BCVI. Risk factors for screening are identified (see table 1), screening modalities are reviewed indicating that although angiography remains the gold standard, multi-planar (≥=8 slice) CT angiography may be equivalent, and treatment algorithms are evaluated. It is noted that change in the diagnosis and management of this injury constellation is rapid due to technological advancement and the difficulties inherent in performing randomized prospective trials in this patient population.

Keywords: trauma, vascular, blunt, carotid, cerebrovascular

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SCOPE OF THE PROBLEM

Blunt injury to the carotid or vertebral vessels (blunt cerebrovascular injury [BCVI]) is diagnosed in approximately 1 of 1,000 (0.1%) patients hospitalized for trauma in the United States unless a screening program has been initiated.[1] However, the majority of these injuries are diagnosed after the development of symptoms secondary to central nervous system ischemia, with a resultant neurologic morbidity of up to 80% and associated mortality of up to 40%.[2] When asymptomatic patients are screened for BCVI, the incidence rises to 1% of all blunt trauma patients and as high as 2.7% in patients with an Injury Severity Score ≥16.[3,4] Key issues that need to be addressed in the diagnosis and management of BCVI include what population (if any) merits screening for asymptomatic injury, what screening modality is best, what is the appropriate treatment for BCVI (both symptomatic and asymptomatic), and what constitutes appropriate follow-up for these injuries.

PROCESS

Identification of References

A computerized search of the National Library of Medicine/National Institute of Health, Medline database was performed using citations from 1965 to 2005 inclusive. The search terms, "cerebrovascular trauma," or "carotid artery," or "vertebral artery" AND wounds and injuries (mesh heading) AND "blunt," limited to the English language returned approximately 1,500 citations. Titles and abstracts were reviewed to determine relevance, and isolated case reports, small case series, editorials, letters to the editor, and review articles were eliminated. The bibliographies of the resulting full-text articles were searched for other relevant citations, and these were obtained as needed. One hundred seventy-nine articles were selected for review, and of these, 68 met inclusion criteria and are excerpted in the attached eviden-

tiary table (see Table, Supplemental Digital Content 1, http://links.lww.com/TA/A33).

Quality of the References

The Eastern Association for the Surgery of Trauma's, "Utilizing Evidence Based Outcome Measures to Develop Practice Management Guidelines: A Primer" was used as the quality assessment instrument applied in the development of this protocol.[5] Articles were classified as classes I, II, or III according to the following definitions:

Class I: Prospective, randomized, controlled trial (there were no class I articles reviewed).
Class II: Clinical studies in which the data were collected prospectively, and retrospective analyses that were based on clearly reliable data. Types of studies so classified include observational studies, cohort studies, prevalence studies, and case-control studies. There were 27 class II studies identified.
Class III: Studies based on retrospectively collected data. Evidence used in this class includes clinical series, database or registry reviews, large series of case reviews, and expert opinion. There were 41 class III studies identified.

Establishment of Recommendations

A committee consisting of 10 trauma surgeons was convened to review the data and establish these recommendations using definitions as established by the Eastern Association for the Surgery of Trauma Practice Management Guidelines Committee:[5]

Level I: The recommendation is convincingly justifiable based on the available scientific information alone. This recommendation is usually based on class I data; however, strong class II evidence may form the basis for a level I recommendation, especially if the issue does not lend itself to testing in a randomized format. Conversely, low quality or contradictory class I data may not be able to support a level I recommendation. No level I guidelines were supported by the literature.
Level II: The recommendation is reasonably justifiable by available scientific evidence and strongly supported by expert opinion. This recommendation is usually supported by class II data or a preponderance of class III evidence. Seven level II guidelines were established by the literature.
Level III: The recommendation is supported by available data, but adequate scientific evidence is lacking. This recommendation is generally supported by class III data. This type of recommendation is useful for educational purposes and in guiding future clinical research. Nine level III guidelines are proposed for this topic.

RECOMMENDATIONS

Question addressed: What patients are of high enough risk, so that diagnostic evaluation should be pursued for the screening and diagnosis of BCVI?

Level I: No level I recommendations can be made.

Level II:

1. Patients presenting with any neurologic abnormality that is unexplained by a diagnosed injury should be evaluated for BCVI.
2. Blunt trauma patients presenting with epistaxis from a suspected arterial source after trauma should be evaluated for BCVI.

Level III:

1. Asymptomatic patients with significant blunt head trauma as defined below are at significantly increased risk for BCVI and screening should be considered. Risk factors are as follows:
   - Glasgow Coma Scale score ≤ 8;
   - Petrous bone fracture;
   - Diffuse axonal injury;
   - Cervical spine fracture particularly those with (i) fracture of C1 to C3 and (ii) fracture through the foramen transversarium;
   - Cervical spine fracture with subluxation or rotational component; and
   - Lefort II or III facial fractures
2. Pediatric trauma patients should be evaluated using the same criteria as the adult population.

Question addressed: What is the appropriate modality for the screening and diagnosis of BCVI?

Level I: No level I recommendations can be made.
Level II:

1. Diagnostic four-vessel cerebral angiography (FVCA) remains the gold standard for the diagnosis of BCVI.
2. Duplex ultrasound is not adequate for screening for BCVI.
3. Computed tomographic angiography (CTA) with a four (or less)-slice multidetector array is neither sensitive nor specific enough for screening for BCVI.

Level III:

1. Multislice (eight or greater) multidetector CTA has a similar rate of detection for BCVI when compared with historic control rates of diagnosis with FVCA and may be considered as a screening modality in place of FVCA. Conflicting studies have been published however (see the Scientific Rationale section).

Question: How should BCVI be treated? This refers a grading scheme proposed by Biffl et al.[6] Grading scale:

- Grade I—intimal irregularity with $<25\%$ narrowing;
- Grade II—dissection or intramural hematoma with $>25\%$ narrowing;
- Grade III—pseudoaneurysm;
- Grade IV—occlusion; and
- Grade V—transection with extravasation.

Level I: No level I recommendations can be made.
Level II:

1. Barring contraindications, grades I and II injuries should be treated with antithrombotic agents such as aspirin or heparin.

Level III:

1. Either heparin or antiplatelet therapy can be used with seemingly equivalent results.
2. If heparin is selected for treatment, the infusion should be started without a bolus, a guideline for activated partial thromboplastin time goal cannot be determined.
3. In patients in whom anticoagulant therapy is chosen conversion to warfarin titrated to a prothrombin time-international normalized ratio of 2 to 3 for 3 months to 6 months is recommended.
4. Grade III injuries (pseudoaneurysm) rarely resolve with observation or heparinization, and invasive therapy (surgery or angiointerventional) should be considered. N.B. carotid stents placed without subsequent antiplatelet therapy have been noted to have a high rate of thrombosis in this population.[7]
5. In patients with an early neurologic deficit and an accessible carotid lesion operative or interventional repair should be considered to restore flow.
6. In children who have suffered an ischemic neurologic event (INE), aggressive management of resulting intracranial hypertension up to and including resection of ischemic brain tissue has improved outcome as compared with adults and should be considered for supportive management.

Question: For how long should antithrombotic therapy be administered?
No recommendations can be made.

Question: How should one monitor the response to therapy?

Level I: No level I recommendation can be made.
Level II:

1. Follow-up angiography is recommended in grades I to III injuries. To reduce the incidence of angiography-related complications, this should be performed 7 days postinjury.

Level III: There are no level III guidelines for this question.

SCIENTIFIC FOUNDATION

Screening and Diagnosis

In symptomatic patients who undergo FVCA for the indications of unexplained neurologic symptoms or arterial epistaxis, the diagnosis of BCVI is made in a significant percentage of cases (38–100%) and is clearly recommended as a reason to pursue the diagnosis.[8–10]

Screening asymptomatic patients at risk for BCVI is more controversial. Multiple studies have indicated that patients with BCVI often present hours to days before the onset of symptoms.[11–13] Failure to identify and treat these injuries can result in significant mortality and morbidity.[14] It is clear that screening for BCVI by essentially any modality can diagnose BCVI before the onset of symptoms at rates up to 10 times higher than previously identified.[3] On the basis of this data, a number of individuals recommend screening blunt trauma patients at risk for BCVI using FVCA as the diagnostic modality.[3,15–17] There is some countervailing opinion.

In a database review of 35,000 patients, Mayberry et al.determined that only 17 were diagnosed with blunt cardiac injury, of which 11 became symptomatic. Of these, only two were asymptomatic for >2 hours postadmission, and of these two, only one met criteria for screening. Based on this data, Mayberry et al.[18] concluded that screening was futile in light of the inability to diagnose the injury before the development of symptoms. The majority of the available data does not support this finding. The preponderance of the evidence supports the recommendation that patients at risk for BCVI can be identified and diagnosed before the onset of symptoms with the application of an appropriate screening modality.

Criteria for Screening/Risk factors

The mechanism of BCVI seems to be associated with cervical hyperextension and rotation, hyperflexion, or direct blow.[10] The factors that are most closely associated with the finding of BCVI are direct evidence of neurologic deficits as noted above. In asymptomatic patients, a number of factors have been associated with increased risk of BCVI. Biffl et al. performed linear regression analysis of a liberally screened patient population (N = 249) and found that there were 4 independent risk factors for blunt carotid arterial injury (BCAI). These were (1) Glasgow Coma Scale score <6; (2) petrous fracture; (3) diffuse axonal injury; and (4) LeFort II or III fracture. Patients who had any of the above risk factors had a risk of 41% for BCAI. This risk increased to 93% in the presence of all 4 factors. The only risk factor for blunt vertebral artery injury (BVAI) was presence of cervical spine fracture. However, 20% of patients diagnosed with BCVI selected for screening by the criteria in Table 1 did not have the independent risk factors identified by regression analysis, indicating that broad selection criteria are necessary to prevent missed injuries.[10] Cothren et al.[19]retrospectively reviewed patients with BVAI and found that complex cervical

TABLE 1. Screening Criteria for BCVI Adapted From Biffl et al[10] (With Permission)

Screening Criteria for BCVI
Injury mechanism
Severe cervical hyperextension/rotation or hyperflexion, particularly if associated with
Displaced midface or complex mandibular fracture
Closed head injury consistent with diffuse axonal injury
Near hanging resulting in anoxic brain injury
Physical signs
Seat belt abrasion or other soft tissue injury of the anterior neck resulting in significant swelling or altered mental status
Fracture in proximity to internal carotid or vertebral artery
Basilar skull fracture involving the carotid canal
Cervical vertebral body fracture

spine fractures involving subluxation, fracture into the foramen transversarium, or C1 to C3 fractures were closely associated with this injury. In a prospective review of screening with digital four-vessel cerebral angiography (DFVCA), Cothren et al. used criteria similar to that proposed by Biffl et al. and modified to incorporate those specific cervical spine fracture patterns shown to increase risk of BVAI to select patients for evaluation. Seven hundred twenty-seven patients (4.6%) of all blunt trauma patients were studied, and 244 were diagnosed with BCVI for a screening yield of 34%.[17] An isolated cervical seat belt sign without other risk factors and normal physical examination has failed to be identified as an independent risk factor in two retrospective studies and should not be used as the sole criteria to stratify patients for screening.[20,21]

Screening Modality

Duplex Sonography

Multiple studies have shown that duplex sonography is not sensitive enough for screening for BCVI, with an overall sensitivity from as low as 38.5%[4] to as high as 86% (the latter for carotid injuries alone).[1,22] Duplex ultrasonography cannot be recommended as a screening modality for BCVI.

Angiography

Arguments have been made that DFVCA, in an appropriate group, is safe, sensitive, and cost effective. Biffl et al.[10] report a 27% rate of positive screening angiogram when asymptomatic patients were screened according to the criteria in Table 1. Cothren et al.[17] used DFVCA in 727 asymptomatic patients who met screening criteria (Table 2), in which, he found 244 patients with injury (34% screening yield). In patients who were initially asymptomatic and could not have antithrombotic therapy, there was a 21% (10 of 48) rate of INE, whereas in those treated with heparin, low-molecular-weight heparin, or antiplatelet agents, only 1 of 187 had an INE. By using this internal data, Cothren et al. estimated that the identification and treatment of asymptomatic BCVI in these 187 patients prevented 32 strokes. This comes at an expense (charge data) of $6,500 per angiogram for a total of ~$154,000 per stroke avoided. Cothren et al. concludes that this is cost effective, and screening with DFVCA should be pursued. The argument against the utilization of DFVCA (aside from that against screening per se) is that it is expensive (~$1,500; unpublished data, Memorial Health University Medical Center, Savannah, GA), carries an inherent risk of stroke (1–2%),[23] and is impractical to apply at many institutions.[24]

TABLE 2. Denver Modification of Screening Criteria for BCVI Adapted From Cothren et al[51] (With Permission)

Denver Modification of Screening Criteria
Signs/symptoms of BCVI
Arterial hemorrhage
Cervical bruit
Expanding cervical hematoma
Focal neurological deficit
Neurologic examination incongruous with CAT scan findings
Ischemic stroke on secondary CAT scan
Risk factors for BCVI
High-energy transfer mechanism with
Lefort II or III fracture
Cervical spine fracture patterns: subluxation, fractures extending into the transverse foramen, fractures of C1–C3
Basilar skull fracture with carotid canal involvement
Diffuse axonal injury with Glasgow Coma Scale score ≤6
Near hanging with anoxic brain injury

Magnetic Resonance Angiography

To date, because magnetic resonance angiography (MRA) is noninvasive and requires no contrast administration, MRA/magnetic resonance imaging has been gaining popularity as an alternative to DFVCA for the diagnosis of BCVI. Although a number of studies describe the use of MRA to identify BCVI[25–28] at this time, the few direct studies that do exist indicate that sensitivity and specificity is significantly lower than that of DFVCA. In a (albeit small) direct comparison of MRA versus angiography, Miller et al.[29] found a sensitivity of 50% for CAI and 47% for VAI. Levy et al.[30] also reported a significantly lower sensitivity for magnetic resonance imaging and MRA than angiography for the diagnosis of BCVI. It seems that, based on this data, MRA cannot be recommended as the sole modality for the screening of BCVI.

Computed Tomographic Angiography

Early CTA with one- to four-slice scanners is not sensitive enough to qualify as an adequate screening modality for BCVI. In a prospective study of CTA on a single-slice scanner versus DFVA, Biffl et al.[31] reported a sensitivity and specificity of 68% and 67%, respectively. Similarly Miller et al.[29] compared four-slice CTA versus DFVCA and showed that CTA performed poorly with a sensitivity of 47% for CAI and 53% for VAI. However, sensitivity and specificity seems to improve in direct relationship to improvements in technology. In a prospective study that included images obtained from single-, four-, and eight-slice scanners, Bub et al. report improvement in image quality and concomitant improvement in sensitivity and specificity as the number of detectors increases. The overall results for the mixed population (reported as ranges from different observers) were 83% to 92% sensitivity and 88% to 92% specificity for the carotid artery and 50% to 60% sensitivity and 90% to 97% specificity for the vertebral artery.[32] Berne et al. screened patients with 4-slice and, later, 16-slice scanner CTA in a study in which only positive CTA studies underwent confirmatory angiography, showing an overall sensitivity (for symptomatic BCVI) and specificity of 100% and 94%, respectively. Interestingly, the incidence of BCVI detected went up from 0.6% with the earlier machine to 1.05% with the newer device, approaching historic incidence of BCVI as diagnosed by DFVCA, and the comparative specificity improved from 90.8% to 98.7%.[24] In a follow-up study, Berne et al. screened patients for BCVI solely with a 16-slice scanner. In this prospective study, Berne et al.[33] showed that the detected incidence of BCVI goes up threefold when changing from a 4-slice scanner to a

16-slice scanner, with a resulting incidence of 1.2%, which is similar to that found by screening with DFVCA. In a similar study in which only positive 16-slice CTA studies were followed by DFVCA, Biffl et al. reversed an earlier recommendation[34] that CTA was not adequate for screening for BCVI reporting a sensitivity of 100% for symptomatic BCVI.[35] Schneidereit et al.[36] report similar findings and give a diagnosed incidence for BCVI of 1.4% using a 16-slice scanner. Although these studies are interesting, obviously a true sensitivity can only be obtained via direct comparison between CTA and DFVCA. At this time, 2 studies have directly compared 16-slice CTA versus angiography for screening for BCVI with somewhat conflicting results, and 1 study evaluated a portion of their negative CTA patients with digital cerebral angiography.

Eastman et al. performed 162 CTAs, followed by 146 confirmatory DFVCA studies (12 patients refused consent, 4 were discharged, and 6 died of nonneurologic causes before the study being obtained). Twenty carotid injuries and 26 vertebral artery injuries were identified with 1 false negative CTA (a grade I vertebral artery injury) for a screened population incidence of 28.4% and an overall incidence of 1.25%. The overall sensitivity, specificity, positive predictive value, negative predictive value (NPV), and accuracy were 97.7%, 100% 100%, 99.3%, and 99.3%, respectively.[37] Conversely, Malhotra et al. performed a study with similar design in which 119 patients underwent screening with CTA using a 16-slice multidetector row CT. Of these, 92 patients underwent angiography (3 refused consent, 24 were not offered FVCA secondary to perceived high risk of contrast nephropathy). In this study, the sensitivity and specificity of CTA was 74% and 84%, respectively. However, as noted within the article, this finding must be interpreted with some caution, because all of the false negative CTAs were obtained in the first half of the study period and may be because of the learning curve of the radiologists reading the studies. In the latter half of the study, the specificity and NPV climbed to 100%.[38] One further study has been performed by Utter et al. in which certain patients with a normal CTA underwent digital subtraction angiography (DSA) for confirmation; however, the performance of the confirmatory DSA was at the discretion of the clinical team, thereby instituting selection bias in the design.[39] Of the 271 patients screened with CTA who had normal studies, 82 underwent DSA. In these patients, the NPV of CTA was found to be 92%.

Blunt Cerebrovascular Injuries in Children

There is a relative paucity of information on the screening, diagnosis, and management of BCVI in children, and what is available primarily consists of isolated case reports and small case series. In one review of the National Pediatric Trauma Registry, Lew et al. found an overall incidence of 0.03%, which is lower than that of the adult trauma population and speculated that it may be because of the increased elasticity of the younger children's blood vessels. They did note that another possibility was that the difference was secondary to decreased detection in children and the retrospective nature of the study. Children aged younger than 6 years seemed to be at higher risk, making up 73% of patients with BCVI, whereas they made up only 36% of the registry patients. Chest trauma (in particular clavicle fracture) and severe head injury (basilar skull fracture, intracranial hemorrhage) were associated with a higher risk of BCVI in the pediatric population.[40] In a case review of five patients with blunt cardiac injury, Duke and Partington[41] recommend initial treatment of the arterial injury to be the same as in adults. Where recommendations differ is that they go on to recommend aggressive management of intracranial hypertension in children up to and including resection of infracted tissue because of improved outcome in pediatric patients in contradistinction to the dismal outcome of postischemic intracranial hypertension in adults.

Treatment of BCVI

Surgery

A number of studies from the 1980s and 1990s have concluded that if individuals have minimal or no symptoms and an accessible carotid lesion, they do well with operative intervention and, therefore, recommend repair of any more than minor intimal irregularities.[42–44] However, most of these studies also note that if the patient presents with profound neurologic deficit, revascularization does not improve outcome. In all studies that have compared ligation versus repair, those patients who do not have a profound deficit do much better with repair.[45,46] Karlin and Marks,[47] for example, found 7.8% mortality in patients undergoing repair versus 50% in those undergoing ligation and that, furthermore, those patients who did not have a deficit before surgery did not develop one if revascularized. Finally a vast majority of these studies including Richardson et al.[48] indicate that if the patient presents with a dense neurologic deficit, neither operation nor anticoagulation improves outcome. All of these studies, however, were of class III quality.

Anticoagulation

There have been a number of studies attempting to evaluate the impact of antithrombotic agents on the progression or development of sequelae of BCVI. As is not unexpected, the results have been somewhat contradictory, but the weight of the evidence seems to support the administration of antithrombotic agents to those patients with BCVI who do not have contraindications for such. A series of retrospective studies[15,49–51] found that administration of antithrombotic agents reduces the rate of neurologic sequelae after BCVI. Fabian et al. also indicated that mortality also improves with heparinization in this population. Although there has not been a direct, controlled comparison of heparinization versus antiplatelet agents (aspirin or clopidigrel) in the prevention of cerebral vascular crash (CVC) after BCVI, a number of studies performed subgroup analysis in an attempt to address this question. In one of these studies Biffl et al.[3] compared those patients treated with aspirin versus heparin and found a trend toward reduction in CVC for those treated with heparin (1% vs. 9%; $p = 0.07$). However, studies by Wahl et al.,[52] Cothren et al.,[51] and a second study by Biffl et al.[53] failed to demonstrate a difference in outcome between the two modalities. In these previously mentioned studies, both Cothren et

al. and Biffl et al. still recommend heparinization as first-line therapy for those patients without contraindications, reserving antiplatelet agents for those not deemed to be candidates for anticoagulation.

Serious bleeding complications can accompany aggressive anticoagulation regimens. In a mixed population of patients with both blunt and penetrating carotid injury, Nanda et al.[54] found that in patients with a preexisting intracerebral hemorrhage, anticoagulation resulted in worsening in two of three patients. Extracranial hemorrhage is another frequent complication of systemic heparinization in patients with multiple injuries. For example, in a previously mentioned study, Biffl et al.[3] noted that bleeding, which required either transfusion or cessation of heparin, was encountered in 54% of patients, prompting him to recommend a conservative protocol for the initiation and maintenance of the heparin infusion and tight control of activated partial thromboplastin time to within 40 seconds to 50 seconds in a later study.[53]

Angiointerventional Therapy

There have been several preliminary, class III studies that have indicated the safety and feasibility of catheter-directed therapy to include embolization of pseudoaneurysms and stenting of intimal injuries.[55–58] A more recent class II study by Cothren et al.[7] indicated that the carotid artery occlusion rate in patients who underwent stenting is much higher than that of patients with BCAI who were treated with antithrombotic agents alone. This resulted in a rate of complications (three CVC and one subclavian artery dissection) of 21% in stented patients versus 5% in nonstented patients (none who received anticoagulation suffered a CVC). The author adds that the reason for this may be that patients who had undergone stenting were then treated with heparin and not antiplatelet agents and recommends a study to evaluate this.

Monitoring Response to Therapy

In a class II study, Biffl et al.[53] found that follow-up angiography changes management in 61% of BCVI, particularly in that grades 1 and 2 injuries often go on to complete healing or to form a pseudoaneurysm within 7days to 10 days. The author notes that the complication rate of angiography was significantly higher if the follow-up procedure was performed within 7 days and recommends that at least that amount of time to be allowed to lapse before follow-up angiography.

FUTURE DIRECTIONS

Screening

BCVI is a rare entity (though not as rare as formerly thought), which requires a high index of suspicion to identify before the onset of symptoms. The clinical and cost effectiveness of a screening program depends on disease-specific, test-specific, and organizational issues, as well as the utility (or futility) of the treatment modalities available. Further prospective investigation is necessary to further refine the screening criteria so as to maximize the disease incidence in the screened population, which will increase accuracy and decrease costs.

Treatment

The optimum modality for the treatment of BCVI is as yet undetermined. Prospective studies will be necessary to compare invasive intervention versus anticoagulation. Furthermore, the optimal anticoagulation regimen is as yet unknown in terms of agent (antiplatelet vs. heparinoid vs. warfarin), as well as the duration and end point of therapy. Cleary, there is room for further study in this regard. In light of the relative rarity of the disease entity, systematic, multi-institutional studies will be required to answer this question.

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