Western Trauma Association Critical Decisions in Trauma: Screening for and Treatment of Blunt Cerebrovascular Injuries

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The Western Trauma Association (WTA) develops algorithms to provide guidance and recommendations for particular practice areas, but does not establish the standard of care. The WTA develops algorithms based on the evidence available in the literature and the expert opinion of Western Trauma Association members. The algorithm (Fig. 1) and accompanying comments represent a safe and sensible approach that could be followed at most trauma centers. We recognize that there will be patient, personnel, institutional, and situational factors that may warrant or require deviation from the recommended algorithm. We encourage institutions to use this guideline to formulate their own local protocols.

The algorithm contains letters at decision points; the corresponding paragraphs in the text elaborate on the thought process and cite the pertinent literature. The annotated algorithm is intended to (a) serve as a quick bedside reference for clinicians; (b) foster more detailed patient care protocols that will allow for prospective data collection and analysis to identify best practices; and (c) generate research projects to answer specific questions concerning decision making in the management of adults with BCVI.

HISTORICAL PERSPECTIVE

Blunt carotid injury (BCI) and blunt vertebral injury, collectively known as BCVI, have historically been considered rare, but potentially devastating, events. Early multicenter reviews collectively reported BCI-related mortality rates of 23%, with 48% of survivors suffering permanent severe neurologic sequelae.1–4 In these reviews, the collective incidence of BCI was noted to be 0.1% among blunt trauma victims admitted to trauma centers. In the latter part of the 1990s, with awareness heightened by the landmark series from Memphis,5 the reported incidence of BCI increased to 0.24%–0.4%.5–7 The Denver group confirmed that many injuries were clinically occult6 and instituted liberal screening of asymptomatic patients in the mid-1990s.8 Screening for BCVI has now become widespread, and several centers have reported an incidence of BCVI exceeding 1% of blunt trauma admissions.9–15

ANNOTATED TEXT FOR ALGORITHM

A. Trauma patients with any of the following signs or symptoms should be considered to have BCVI until proven otherwise: arterial hemorrhage from neck, mouth, nose, ears; large or expanding cervical hematoma; cervical bruit in a patient younger than 50 years; focal or lateralizing neurologic deficit, including hemiparesis, transient ischemic attack, Horner’s syndrome, oculosympathetic paresis, or vertebrobasilar insufficiency; evidence of cerebral infarction on computed tomography (CT) or magnetic resonance imaging (MRI) scan; or neurologic deficit that is incongruous with CT or MRI findings. Any of these findings should prompt emergent diagnostic evaluation and interventions directed at hemorrhage control or stroke management.

B. Although it has been suggested that screening of asymptomatic patients is futile,16–18 there is evidence that stroke rates are significantly lower in treated patients when
Diagnosis and Management of Blunt Cerebrovascular Injuries

A

B

C

D

E

F

G

H

I

Stop

Emergent

Urgent

Repeat 16-Slice CTA in 7-10 days

Injury Healed?

Yes

Discontinue Antithrombotics

No

Consider stenting for severe luminal narrowing or expanding pseudoaneurysm

Antithrombotic Therapy: Heparin (PTT 40-50 sec)

Endovascular Treatment

Grade I-IV Injury

Grade V Injury

Positive

Negative

No

Endovascular Treatment

CT angiography with multidetector-row CT, 16-channel or higher. If fewer than 16 channels, interpret CTA with caution.

If Signs/Symptoms or high clinical suspicion and (-)CTA, consider arteriogram as the gold standard.

If Grade II-V injury is surgically accessible and patient has not suffered completed stroke, pursue operative repair.

Heparin is preferred in the acute setting, as it is reversible and may be more efficacious than antiplatelet drugs.

Stenting should be performed with caution, and appropriate antithrombotic therapy administered concurrently.

Aspirin alone (75-150 mg daily) is adequate and should be considered lifelong as its risk profile is superior to coumadin.

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Fig. 1. Algorithm for the diagnosis and management of blunt cerebrovascular injuries in adults.

compared with untreated patients. Moreover, the Denver group has demonstrated that screening and treatment of BCVI is cost effective. Identification of a high-risk group for screening has been debated in the literature. The fundamental mechanisms of internal carotid artery injury include (a) cervical hyperextension or hyperflexion with rotation, stretching the internal carotid artery over the lateral articular processes of cervical vertebral bodies C1–C3; (b) direct cervical trauma; (c) intraoral trauma; and (d) basilar skull fracture involving the carotid canal. The vertebral artery is most commonly injured from C-spine injuries, especially subluxations and fractures of the foramen transversarium. Analyses of screening have identified the following list of high-risk factors for BCVI: (a) an injury mechanism compatible with severe cervical hyperextension with rotation or hyperflexion; (b) Lefort II or III midface fractures; (c) basilar skull fracture involving the carotid canal; (d) closed head injury consistent with diffuse axonal injury with Glasgow Coma Scale score <6; (e) cervical vertebral body or transverse foramen fracture, subluxation, or ligamentous injury at any level, or any fracture at the level of C1–C3; (f) near-hanging resulting in cerebral anoxia; or (g) seat belt or other clothesline-type injury with significant cervical pain, swelling, or altered mental status. With the improved accuracy of noninvasive screening modalities, there is a tendency to liberalize screening in an attempt to capture all injuries, rather than restricting screening to groups with the highest risk. Broadened screening guidelines may include combined thoracic and cervical or cranial injuries, but there have not been any large-scale analyses to determine the yield of such protocols.

C. Four-vessel biplanar cerebral arteriography (ART) has been considered the gold standard for diagnosis of BCVI. Unfortunately, it is invasive and resource intensive, and its risks include complications related to catheter insertion (1–2% hematoma; arterial pseudoaneurysm), contrast administration (1–2% renal dysfunction; allergic reaction), and stroke (<1%). Duplex ultrasonography is widely used for imaging the extracranial carotid arteries. However, because of its technical limitations and poor sensitivity in clinical trials, there is virtually no role for ultrasonography for BCVI screening. Similarly, with
its documented poor sensitivity and specificity for BCVI, magnetic resonance angiography is not considered a standard screening test for BCVI. In contrast, CT angiography (CTA) has emerged as the preferred screening test for BCVI. Although the accuracy of early-generation CTA was poor, it was improved with multidetector-row (4- and 8-slice) CTA. Sixteen-slice CTA has been adopted by a number of centers and seems to reliably identify clinically significant BCVI. Three published studies have evaluated the accuracy of 16-slice CTA compared with ART. Eastman et al. reported 100% sensitivity of 16-slice CTA for carotid and 96% sensitivity for vertebral artery injuries. Utter et al. performed ART on a subset (30%) of their patients with normal CTA and initially found that CTA missed seven BCVI among 82 patients, for a negative predictive value of 92%. However, retrospective review of the CTA images found that the injuries were evident in six of the seven patients, and that the seventh patient’s abnormality was most likely not traumatic in origin. Malhotra et al. have offered a note of caution, reporting 43% false-positive and 9% false-negative rates for CTA. However, as in the series of Utter et al., the inaccuracy of CTA seemed to be related in large part to the radiologists’ inexperience, as all of the missed BCVI occurred in the first half of the study period. Thus, it seems that 16-slice (or more) CTA is reliable for screening for clinically significant BCVI, but that the accuracy diminishes with fewer detector rows. If CTA is not available, ART is the gold standard. If ART is not available, it is recommended that an institutional clinical practice guideline be outlined that considers transfer to a trauma center for patients at high risk. In the setting of a symptomatic patient and normal noninvasive screening study, ART is recommended to definitively exclude injury.

D. A relatively high false-positive rate suggests that CTA may be oversensitive. However, ART may be warranted in the setting of high clinical suspicion and a normal CTA to definitively exclude an injury.

E. The primary management strategies for BCVI include observation, surgical repair, antithrombotic drugs, and endovascular therapy. In determining the treatment for an individual, the location and grade of the injury (Table 1) as well as symptomatology must all be considered. Given the high morbidity and mortality rates historically associated with untreated BCVI, observation should not be chosen unless there are contraindications to alternative strategies. Currently, surgical therapy for BCVI is limited. Grade I injuries are associated with a low enough stroke risk that surgical repair is not justified. Repair is warranted in higher-grade injuries, but surgical access is often precluded by involvement of the carotid artery at the base of the skull. Consequently, nonsurgical management is the first-line treatment of BCVI. There are no published prospective randomized studies comparing treatment strategies; management recommendations are made based on retrospective analyses of patients managed per institutional protocols. Early reports recommended systemic anticoagulation with heparin (no bolus; 10 U/kg/h to target partial thromboplastin time 40–50 s), demonstrating improved neurologic outcomes among symptomatic patients and stroke prevention among asymptomatic patients. A few retrospective, uncontrolled case series, as well as more recent large reports from Memphis and Denver suggest that systemic heparinization and antplatelet therapy (clopidogrel 75 mg daily or aspirin 325 mg daily) are equally efficacious in stroke prevention. In the absence of controlled data, systemic heparin may be preferred among patients with neurologic symptoms and in those who have no contraindications. The Memphis data demonstrate systemic heparinization’s clear efficacy in improving neurologic outcomes among symptomatic patients. Furthermore, although statistical significance was not achieved, the large series in Denver suggests that heparin may be superior to antplatelet therapy in stroke prevention ($p = 0.07$) and in neurologic improvement after ischemic insult ($p = 0.15$).

F. Grade V injuries are associated with high mortality and mandate immediate attempts at control. Urgent surgical repair is indicated for accessible lesions, but the majority is inaccessible and require endovascular techniques.

G. A follow-up imaging study is recommended 7 days to 10 days after injury or for any change in neurologic status. Follow-up imaging led to a change in therapy in 65% of grade I and 51% of grade II injuries and helped plan therapy for grade III injuries.

H. In the setting of progressive severe vessel narrowing or pseudoaneurysm enlargement, stents have been deployed in an effort to maintain vascular patency. A review of the Denver experience found a 17% incidence of stent-related complications and 45% occlusion rate, suggesting that risks exceed benefits. On the other hand, the Memphis group reported a good safety and patency record. It is clear that controlled trials are needed, caution should be used during stent deployment to avoid dislodging unstable thrombus, and poststent antithrombotic therapy is critical. It is not advisable to deploy stents within the first several days after injury.

I. It has been recommended that patients receive long-term antithrombotic therapy, but the optimal drug and duration have not been studied. In the absence of documented healing of the vessel, it is reasonable to provide some treatment, as stroke has been reported as long as 14 years after injury. Coumadin was recommended in early series, but with the apparent efficacy of antiplatelet therapy in the early period, it seems that long-term antiplatelet therapy is preferable to warfarin for its safety.

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**TABLE 1. Blunt Carotid and Vertebral Arterial Injury Grading Scale**

<table>
<thead>
<tr>
<th>Injury Grade</th>
<th>Description</th>
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<tbody>
<tr>
<td>I</td>
<td>Luminal irregularity or dissection with &lt;25% luminal narrowing</td>
</tr>
<tr>
<td>II</td>
<td>Dissection or intramural hematoma with ≥25% luminal narrowing, intraluminal thrombus, or raised intimal flap</td>
</tr>
<tr>
<td>III</td>
<td>Pseudoaneurysm</td>
</tr>
<tr>
<td>IV</td>
<td>Occlusion</td>
</tr>
<tr>
<td>V</td>
<td>Transection with free extravasation</td>
</tr>
</tbody>
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cost profile. Aspirin and clopidogrel have different mechanisms of action; in addition, some individuals are resistant to the effects of one or both drugs. Several studies have evaluated the safety and efficacy of dual antiplatelet therapy (aspirin combined with clopidogrel) in a number of clinical situations. Dual therapy is indicated in the setting of acute coronary syndromes and percutaneous coronary interventions, with or without stent placement. On the other hand, it is not recommended in patients who have had a stroke or transient ischemic attack, based on increased bleeding risk and the lack of benefit or increase in mortality. More studies are necessary to determine the risk:benefit of dual therapy in BCVI. Lifelong antiplatelet therapy is recommended if the lesion persists. Aspirin is currently the agent of choice, but newer agents with reversible effects may be preferable in the future. Platelet mapping may one day assist in choice of drug and dose.

REFERENCES