B
lunt cerebrovascular injury (BCVI) is rare but poten-
tially devastating, with a reported stroke rate of 3% to
59%, and disproportionately affects young adults.1,2 The
location and severity of injury are dependent on the mecha-
nism and can include intimal tear, intramural hematoma, pseudo-
aneurysm, dissection, occlusion, or transection as defined
by the Denver criteria.1,2 Clinical diagnosis can be obfuscated
by a lack of symptoms and confounding injuries in patients
with trauma. The Denver criteria are based on angio-
graphic findings; however, current screening protocols now use
computed tomography, which has broadened screening proto-
cols, resulting in increased identification of BCVIs. However,
despite a robust classification scheme, there remains a large
cohort of patients presenting with indeterminate BCVI
(IBCVI) findings on initial imaging that do not strictly meet the
Denver criteria. It is unknown what percentage of these le-
sions resolve, progress to a true BCVI, remain indeterminate
when followed up serially, or result in cerebrovascular acci-
dent (CVA) or transient ischemic attack (TIA). Optimal treat-
ment of these indeterminate lesions is also unknown. In this
series, we review our institutional experience with IBCVI to
determine the natural history of IBCVI, the risk of CVA or TIA,
rate of resolution or recategorization, and demographic and/or
clinical variables that affect outcomes.

IMPORTANCE The Denver criteria grade blunt cerebrovascular injuries (BCVIs) but fail to
capture many patients with indeterminate findings on initial imaging.

OBJECTIVE To evaluate outcomes and clinical significance of indeterminate BCVIs (IBCVI).

DESIGN, SETTING, AND PARTICIPANTS A retrospective review of all patients treated for BCVIs
at our institution from January 1, 2007, through July 31, 2014, was completed. Patients were
divided into 2 groups: those with true BCVIs as defined by the Denver criteria and those with
IBCVI, which was any initial imaging suggestive of a cerebrovascular arterial injury not
classifiable by the Denver criteria.

MAIN OUTCOMES AND MEASURES Primary outcomes were rate of resolution of IBCVI,
freedom from cerebrovascular accident (CVA) or transient ischemic attack (TIA), and 30-day
dortality.

RESULTS We identified 100 patients with 138 BCVIs: 79 with true BCVIs and 59 with IBCVI.
With serial imaging, 23 IBCVI (39.0%) resolved and 21 (35.6%) remained indeterminate,
whereas 15 (25.4%) progressed to true BCVI. The rate of CVA or TIA in the IBCVI group was
5.1% compared with 15.2% in the true BCVI group (P = .06). Of the 15 total CVA or TIA, 11
(73.3%) resulted from carotid injury and 4 (26.7%) from vertebral artery occlusion (P = .03).
By Kaplan-Meier analysis, there was no difference in freedom from CVA or TIA for the 2
groups (P = .07). Median clinical follow-up was 91 days. Overall and 30-day mortality for the
entire series were 17.4% and 15.2%, respectively. There was no difference in long-term or
30-day mortality between true BCVI and IBCVI groups.

CONCLUSIONS AND RELEVANCE Detection of IBCVI has become a common clinical conundrum
with improved and routine imaging. Indeterminate BCVI is not completely benign, with
25.4% demonstrating anatomical progression to true BCVI and 5.1% developing
cerebrovascular symptoms. We therefore recommend serial imaging and antiplatelet therapy
for IBCVI.
All CTA studies were performed with a 64-section Philips giography (MRA), and digital subtraction angiography (DSA). Magnetic resonance angiography (CTA), magnetic resonance angiography (MRA), and digital subtraction angiography (DSA). All CTA studies were performed with a 64-section Philips Ingenuity Core (Philips Healthcare) at 120 kVp, 280 mAs, and 1-mm section thickness. All MRA was performed with 3.0-T Philips Ingenia MR (Philips Healthcare) with 3-mm overcon-}

Table 1. Modified Denver Criteria for Blunt Cerebrovascular Injury

<table>
<thead>
<tr>
<th>Grade</th>
<th>Definition</th>
<th>Stroke Rate, % (CAI/VAI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Irregularity of vessel wall or dissection with &lt;25% luminal narrowing</td>
<td>3.6</td>
</tr>
<tr>
<td>II</td>
<td>Intraluminal thrombus, raised intimal flap, dissection or intramural hematoma with &gt;25% luminal narrowing</td>
<td>14:38</td>
</tr>
<tr>
<td>III</td>
<td>Pseudoaneurysm</td>
<td>26:27</td>
</tr>
<tr>
<td>IV</td>
<td>Occlusion</td>
<td>50:28</td>
</tr>
<tr>
<td>V</td>
<td>Transection</td>
<td>100:100</td>
</tr>
</tbody>
</table>

Abbreviations: CAI, carotid artery injury; VAI, vertebral artery injury.

Methods

Patients evaluated for BCVI or iBCVI from January 1, 2007, through July 31, 2014, at Oregon Health and Science University were identified retrospectively from a prospective trauma registry and International Classification of Diseases, Ninth Revision, codes. Data were collected from the trauma registry and the electronic medical record (EMR; Epic Systems Corp). Tobacco use was defined as any prior history of smoking or current use. Obesity was defined as a body mass index, calculated as the weight in kilograms divided by height in meters squared, greater than 30. Coronary artery disease was considered present if documented in the EMR. Hyperlipidemia, diabetes mellitus, and hypertension were determined by the EMR or use of medications to treat these conditions. Mechanism of injury, injury severity scores (ISSs), Glasgow Coma Scale (GCS) scores, and the presence of concomitant injuries, such as facial, skull, or cervical fractures, were obtained from the trauma registry database. The study was approved by the institutional review board of Oregon Health and Science University. Informed consent was waived.

True BCVI was defined as any injury on initial imaging distinctly classified by the Denver criteria (Table 1).1,2 Suspected BCVIs on initial imaging not distinctly defined by the Denver criteria were classified as iBCVI. Commonly, iBCVIs were documented as stretch injury, vasospasm, possible dissection or intimal flap, or possible linear opacity or luminal irregularity or narrowing by the reading staff radiologist. The same grading criteria were used when serial imaging was performed. Radiographic resolution was defined as any iBCVI that was no longer present on serial imaging. Reclassification of iBCVI was any iBCVI later reclassified by the Denver criteria by subsequent imaging. An iBCVI that was neither resolved nor reclassified was counted as remaining indeterminate.

Initial imaging was the first modality to directly evaluate the cerebrovascular arteries on patient admission. Serial imaging was defined as any additional imaging of any modality to study a BCVI or iBCVI. Types of serial imaging included cervical B-mode and duplex ultrasonography (DUS), computed tomography angiography (CTA), magnetic resonance angiography (MRA), and digital subtraction angiography (DSA). All CTA studies were performed with a 64-section Philips Ingenuity Core (Philips Healthcare) at 120 kVp, 280 mAs, and 1-mm section thickness. All MRA was performed with 3.0-T Philips Ingenia MR (Philips Healthcare) with 3-mm overcon-

Results

We identified 100 patients with 138 BCVIs: 79 with true BCVIs and 59 with iBCVIs (Table 2 and Figure 1). Initial diagnostic imaging modalities included CTA (121 [87.7%]), MRA (5 [3.6%]), DUS (5 [3.6%]), and DSA (4 [2.9%]). Serial imaging was performed for 52 BCVIs (65.8%) and 43 iBCVIs (72.9%) using CTA (50 [53.2%]), DSA (23 [24.5%]), DUS (15 [16.0%]), and MRA (6 [6.4%]). Median time from initial imaging to follow-up imaging was 1.5 days. When iBCVIs were followed up serially, 15 iBCVIs (25.4%) were reclassified by the Denver criteria, 23 (39.0%) resolved, and 21 (35.6%) remained indeterminate. The iBCVIs reclassified as true BCVIs were grade I (3 [20.0%]), grade II (10 [66.7%]), and grade III/IV (2 [13.3%]). Indeterminate carotid artery injuries (CAIs) were more likely to be reclassified as true BCVIs than vertebral ar-

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tery injuries (VAIs) (9 [36.0%] vs 5 [14.7%]; \( P = .04 \)). Indeterminate VAIs compared with indeterminate CAIs (17 [50.0%] vs 5 [20.0%]; \( P = .02 \)) and iBCVIs with a concomitant cervical spine fracture were more likely to remain indeterminate (2 [8.0%] vs 1 [32.4%]; \( P = .03 \)). The type of imaging modality (CTA, MRA, DUS, or DSA), comorbidities, ISS, GCS score, concomitant facial or skull fractures, or type of treatment modality received did not affect the rate of resolution or reclassification of iBCVIs.

Initial and serial imaging modalities used were similar between the true BCVI and iBCVI groups. The number of injuries followed up with serial imaging (52 true BCVIs [65.8%] vs 43 iBCVIs [72.9%]; \( P = .38 \)) were also similar between the 2 groups. Mechanism of injury was comparable between the true BCVI and iBCVI groups, with most injuries being a result of motor vehicle collisions (41 true BCVIs [51.9%] vs 26 iBCVIs [44.1%]; \( P = .82 \)). Patients with true BCVIs and iBCVIs were treated with antiplatelet agents with similar frequency (32 [40.5%] vs 31 [52.5%]; \( P = .27 \)); however, more patients with true BCVIs were treated with an anticoagulant compared with those with iBCVIs (23 [29.1%] vs 8 [13.6%]; \( P = .03 \); Figure 2).

The numbers of patients not receiving any medical therapy were similar between the 2 groups (BCVI, 17 [21.5%] vs iBCVI, 18 [30.5%]; \( P = .23 \)). Three patients with true BCVIs were treated invasively with carotid artery stenting for carotid pseudoaneurysm as well as an open carotid artery interposition graft (n = 2) and internal carotid endarterectomy (n = 1). The patient treated with carotid endarterectomy had a long-segment, grade II BCVI with peak systolic velocities of 294 cm/s and acute thrombus in the distal common carotid on DUS. No patient with an iBCVI was treated surgically (Figure 2).

For the series, a total of 15 known CVAs or TIs occurred: 12 (15.2%) in patients with true BCVIs and 3 (5.1%) in patients with iBCVIs on initial imaging (\( P = .06 \)). Freedom from CVA or TIA was similar between patients with true BCVIs and iBCVIs (Figure 3). Median time from injury to CVA or TIA for all patients was 48 hours, with 11 CVAs or TIs (73.3%) occurring within 72 hours of injury. Overall, 3 CVAs or TIs occurred within 1 week of injury and 1 at 6.1 months from a grade II CAI in a 48-year-old man taking therapeutic warfarin. The patient

Table 2. Patient Characteristicsa

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>BCVI (n = 79)</th>
<th>iBCVI (n = 59)</th>
<th>( P ) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD), y</td>
<td>41.7 (21.2)</td>
<td>46.9 (19.5)</td>
<td>.16</td>
</tr>
<tr>
<td>Male</td>
<td>53 (67.1)</td>
<td>33 (55.9)</td>
<td>.30</td>
</tr>
<tr>
<td>CAD</td>
<td>2 (2.5)</td>
<td>4 (6.8)</td>
<td>.30</td>
</tr>
<tr>
<td>Hypertension</td>
<td>14 (17.7)</td>
<td>23 (39.0)</td>
<td>.02</td>
</tr>
<tr>
<td>Smoking</td>
<td>32 (40.5)</td>
<td>31 (52.5)</td>
<td>.02</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>9 (11.4)</td>
<td>1 (1.7)</td>
<td>.02</td>
</tr>
<tr>
<td>BMI</td>
<td>26.3 (6.5)</td>
<td>25.6 (4.8)</td>
<td>.56</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>2 (2.5)</td>
<td>7 (11.9)</td>
<td>.05</td>
</tr>
<tr>
<td>ISS, mean (SD)</td>
<td>28.4 (15.4)</td>
<td>26.4 (15.6)</td>
<td>.49</td>
</tr>
<tr>
<td>GCS score, mean (SD)</td>
<td>10.6 (5.1)</td>
<td>10.3 (5.4)</td>
<td>.75</td>
</tr>
<tr>
<td>Intracerebral hemorrhage</td>
<td>32 (40.5)</td>
<td>29 (49.2)</td>
<td>.35</td>
</tr>
<tr>
<td>Fracture</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cervical spine</td>
<td>34 (43.0)</td>
<td>25 (42.4)</td>
<td>.80</td>
</tr>
<tr>
<td>Skull</td>
<td>12 (15.2)</td>
<td>12 (20.3)</td>
<td>.44</td>
</tr>
<tr>
<td>Facial</td>
<td>18 (22.8)</td>
<td>16 (27.1)</td>
<td>.61</td>
</tr>
<tr>
<td>Carotid</td>
<td>39 (49.4)</td>
<td>25 (42.4)</td>
<td>.53</td>
</tr>
<tr>
<td>Vertebral</td>
<td>40 (50.6)</td>
<td>34 (57.6)</td>
<td>.53</td>
</tr>
<tr>
<td>Left side</td>
<td>40 (50.6)</td>
<td>28 (47.5)</td>
<td>.85</td>
</tr>
<tr>
<td>Initial imaging</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CTA</td>
<td>66 (90.0)</td>
<td>55 (93.2)</td>
<td>.09</td>
</tr>
<tr>
<td>MRA</td>
<td>3 (3.8)</td>
<td>2 (3.4)</td>
<td>.47</td>
</tr>
<tr>
<td>DUS</td>
<td>3 (3.8)</td>
<td>2 (3.4)</td>
<td>.47</td>
</tr>
<tr>
<td>DSA</td>
<td>2 (2.5)</td>
<td>2 (3.4)</td>
<td>.31</td>
</tr>
<tr>
<td>Serial imaging</td>
<td>52 (65.8)</td>
<td>43 (72.9)</td>
<td>.65</td>
</tr>
</tbody>
</table>

Abbreviations: BCVI, blunt cerebrovascular injury; BMI, body mass index; CAD, coronary artery disease; CTA, computed tomography angiography; DSA, digital subtraction angiography; DUS, duplex ultrasonography; GCS, Glasgow Coma Scale; iBCVI, indeterminate blunt cerebrovascular injury; ISS, injury severity score; MRA, magnetic resonance angiography.

a Data are presented as number (percentage) of patients unless otherwise indicated.
with a CVA or TIA at 6 months returned after a grade II left carotid artery dissection with intermittent dysarthria consistent with thromboembolic TIAs and consistent with his left carotid BCVI. This patient was initially treated with intravenous heparin and transitioned to warfarin with a goal international normalized ratio (INR) of 2.0 to 3.0. However, when he returned with TIA symptoms, his INR was subtherapeutic (INR, 1.2). Subsequent DUS revealed mild left carotid stenosis and a stable dissection. The patient was managed medically with optimization of therapeutic anticoagulation. He has now been followed up for 6 years with annual DUS, maintaining a therapeutic INR, and no recurrence of TIA symptoms.

The only factor associated with a statistically significant increased risk for CVA or TIA was the presence of CAI over VAI (11 [73.3%] vs 4 [26.7%]; P = .03). Distribution of CVA or TIA by injury grade after reclassification is shown in Figure 4. There was no difference in the rate of CVA or TIA with use of antiplatelets, anticoagulants, dual therapy, surgical treatment, or no therapy. Two of 3 patients in the iBCVI group had a CVA or TIA while not receiving medical therapy. There were no CVAs or TIs among patients treated with endovascular or open surgery. Of the 3 patients with a CVA or TIA in the iBCVI group, 2 were reclassified as having true BCVIs. One was reclassified as having a grade 2 CAI by MRA and had a CVA or TIA several hours after admission before receiving medical therapy. The second iBCVI patient with CVA or TIA was reclassified as having a grade IV VAI by DSA after mild cranial nerve deficit was noted 48 hours from the time of injury. Before diagnosis, the patient was not receiving medical therapy. Only one CVA or TIA occurred in a patient with an iBCVI that remained indeterminate on follow-up imaging. This event occurred in a patient with an indeterminate CAI while undergoing antiplatelet therapy within 48 hours from injury. Thus, with follow-up imaging, the actual rate of CVA or TIA for the iBCVI group was only 1.7% rather than 5.1%.

Overall and 30-day mortality for the series was 17.4% and 15.2%, respectively, with a median follow-up of 91 days. The presence of a CVA or TIA did not affect long-term or 30-day mortality (log-rank test, P = .26) but predicted discharge to a skilled nursing facility (P < .001). There was no difference in long-term or 30-day mortality between true BCVI and iBCVI groups. By univariate analysis, categorical factors predicting 30-day mortality for the entire series included intracerebral hemorrhage (P = .004), facial fractures (P = .05), and no therapy (P = .004). Patients not surviving 30 days had significantly higher mean (SD) ISSs (41.7 [13.2] vs 24.6 [14.2]; P < .001) and lower mean (SD) GCS scores (6.4 [5.1] vs 11.3 [4.8]; P < .001) than surviving patients. Protective factors included history of smoking (P = .003) and use of antiplatelet therapy, anticoagulant therapy, or both (P = .004). Binary logistic regression was performed to assess patient factors predicting 30-day mortality. Thirty-day mortality was increased significantly with in-
creased age (odds ratio [OR] per year increase, 1.07; 95% CI, 1.02-1.14), increased ISS (OR per unit increase, 1.09; 95% CI, 1.01-1.19), presence of intracerebral hemorrhage (OR, 10.3; 95% CI, 1.50-193.6), and decreased GCS score (OR, 0.84; 95% CI, 0.70-0.98).

Discussion

The Denver criteria for BCVI are well validated; however, many patients fall outside this scheme due to indeterminate initial imaging characteristics. We have noted a significant number of iBCVIs at our institution. This is the first study, to our knowledge, to evaluate the natural history and outcomes of iBCVI. We found that nearly all iBCVIs (93.2%) are identified by CTA on admission and that, with follow-up imaging, 39.0% of iBCVIs will resolve and 25.4% will be reclassified, usually as grade 1 or II true BCVIs. The iBCVIs that remain indeterminate are typically VAIs, which carry a low risk of CVA or TIA. The risk of CVA or TIA from iBCVI is 5.1%, but excluding injuries reclassified as a true BCVI, that risk is only 1.7%. Last, we found that mortality for these lesions is dependent on age, ISS, intracerebral hemorrhage, and GCS scores.

Our general algorithm for screening for BCVIs includes CTA at the time of presentation for patients with cervical spine fractures, complex facial fractures, cervical seatbelt sign, or neurologic deficits on physical examination. Although DSA has long been considered the criterion standard diagnostic test for BCVI given its high sensitivity and specificity, it is a poor screening test because of the associated risks of access site complications, contrast allergy or nephrotoxicity, radiation exposure, stroke risk of up to 1%, and overall cost and need for specialized personnel and resource availability. Therefore, noninvasive imaging modalities have emerged, and CTA is currently the preferred screening modality at most centers. This modality is quick, safe, less expensive, and more accessible to smaller hospitals than DSA and also offers the opportunity to evaluate for other nonvascular injuries. The sensitivity of CTA varies from 51% to 98% based on trauma institution, interpreting radiologists, and the section thickness and quality of the CT scan, but specificity is high, ranging from 85% to 97%. In addition, CTA affords broader screening, reduced time to diagnosis, and decreased incidence of CVA.

At our institution, vascular surgical consultation is routinely obtained for BCVI or iBCVI. Depending on associated injuries and initial grade of the BCVI at presentation, serial imaging is typically performed within 7 days of injury using noninvasive imaging with DUS or CTA. For iBCVI, we prefer to perform additional imaging within 48 hours to definitively rule in a true BCVI, establish a Denver grade, or rule out an injury. We reserve DSA for very select patients with skull base lesions not well visualized by noninvasive imaging or for BCVIs with an indication for intervention (carotid-cavernous fistula, worsening carotid pseudoaneurysm, or dissection). In our study, comorbidities and injury severity among patients with true BCVI and iBCVI were well matched, suggesting that patients with iBCVIs were appropriately screened.

Serial imaging was traditionally routine for all patients with BCVIs given the dynamic nature of BCVIs. However, Wagenar et al14 followed up patients with serial angiography and found that 50% of grade I and 18% of grade II injuries based on the Denver criteria resolved with medical treatment, whereas only 8% and 2% of grade III and IV injuries resolved after an imaging follow-up period of 11 days. Similarly, upgrading of injuries occurred in only 10% to 27% of grade I and II BCVIs but only 2% of grade III injuries. On the basis of these findings, the authors recommend against early additional imaging for high-grade injuries because these lesions rarely resolve early in their treatment course. In our study, it is possible that iBCVIs that resolved (39.0%) were actually mild grade I injuries at first imaging but resolved with medical treatment before additional imaging, similar to findings in other studies.14,15

The time from BCVI to CVA is well described and commonly occurs within 10 to 72 hours from the time of injury. Therefore, early identification with aggressive screening protocols based on patterns of injury to identify at-risk patients combined with early treatment reduces the risk of CVA from BCVI. In our study, 72.8% of iBCVIs and 75.9% of true BCVIs were treated with medical therapy. Patients receiving no therapy (25.4% overall) had a contraindication to antithrombotic therapy, such as intracerebral hemorrhage or unstable solid organ injury, and these patients had worse outcomes with higher 30-day mortality but no difference in CVA or TIA risk. These patients require a multispecialty approach to determine the risks and benefits of medical therapy in the face of numerous other injuries. Furthermore, open surgical interventions or carotid artery stenting require intraoperative anticoagulation and routine postoperative antplatelet and even dual antplatelet therapy for carotid artery stents. In our series, only 3 patients underwent an intervention. We generally reserve carotid artery stenting or open surgery for expanding pseudoaneurysms, dissection, or high-grade stenosis.

Treatment of BCVIs has not been standardized, and there are no prospective, randomized studies. Thromboembolism is the predominant underlying cause of CVA or TIA after BCVI. Therefore, antithrombotic therapy has been the primary medical therapy proved to reduce CVA risk in prospective, nonrandomized studies.15,21 Early attention was given to intravenous unfractionated heparin; however, hemorrhagic complications were noted in 8% to 16% of cases, and up to 30% of patients with BCVIs are not candidates for therapeutic anticoagulation because of solid organ or head injury.15,19,23-25 The use of antplatelets has recently become more common, and retrospective studies24-27 have found equivalence or superiority of antplatelets over anticoagulants in CVA reduction. We tend to treat complex dissections or pseudoaneurysms with therapeutic heparin and use aspirin primarily for grade I and mild grade II CAI and nearly all VAIs. In our study, patients receiving antplatelets or anticoagulants had improved 30-day mortality rates compared with those receiving no therapy; however, the type of therapy did not affect the rate of CVA or TIA. This finding highlights that the overall injury severity and the presence of intracerebral hemorrhage predict survival rather than treatment of the BCVI and that the mortality benefit of
medical therapy is confounded by the presence of severe nonvascular injuries. Two of 3 CVAs or TIs in the iBCVI cohort occurred in patients who were not receiving medical therapy. We therefore recommend antiplatelet therapy for iBCVI based partly on this finding that patients receiving no medical therapy do worse than others but also based on the finding that 25.4% of iBCVIs will be reclassified as true BCVIs, which carries a known risk of CVA or TIA as well. Although most iBCVIs are reclassified as low-grade lesions, 21.4% are reclassified as grade III/IV BCVIs. Therefore, iBCVIs are not benign and warrant antiplatelet therapy and serial imaging.

We do not yet have a standardized protocol for management of BCVI at our institution. In general, we primarily use aspirin as our antiplatelet. If the lesion resolves on follow-up imaging, use of aspirin is discontinued. If the lesion persists unchanged, we continue therapy indefinitely. If the lesion has anatomical progression on follow-up imaging, we recommend systemic anticoagulation with warfarin with a goal INR of 2.0 to 3.0 or surgical or endovascular intervention. Most patients have early additional imaging within 48 hours of diagnosis or if symptoms develop. Surveillance is then performed every 6 months with DUS or annually if the BCVI remains stable.

Our study is limited by its retrospective design and possible selection bias. The studies were typically read by a single radiologist, so it is possible that interobserver variability may exist. A prospective study masking multiple interpreters of the imaging is needed to determine whether iBCVIs are actually indeterminate, subtle findings or a failure to adequately describe the imaging findings. However, CTA is thought to overestimate detection of BCVI, especially for low-grade lesions, which may partially explain the high rate of resolution of iBCVIs, suggesting a minor intimal injury or a false-positive result on initial imaging.15,28,29 The experience of the interpreting radiologist has been linked with the sensitivity and specificity of CTA for BCVI in several studies.30,31 Our study does not assess the experience of the reading radiologists, but we believe our radiologists are generally experienced with BCVI given the high volume of blunt trauma and the broad screening protocol used at our institution.

Conclusions

This is the first study, to our knowledge, to describe the natural history and report outcomes of iBCVIs, which is now a common entity with the advent of broader, noninvasive screening protocols and improved imaging. We found that 39.0% of iBCVIs will resolve and 25.4% will be reclassified on follow-up imaging within 48 hours as true BCVIs; most are commonly grade I or II injuries. Nonetheless, risk of a CVA or TIA associated with iBCVI is 5.1%, and the stroke risk is highest for CAIs. Lastly, mortality with iBCVI is similar to true BCVI and is dependent on intracranial and global injury severity. For iBCVI, we therefore recommend early initiation of antiplatelet therapy and serial follow-up imaging with noninvasive CTA or DUS to rule out a true BCVI or to reclassify the injury by the Denver criteria.

REFERENCES

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Invited Commentary

Important Clarifications About Treating Indeterminate Vascular Injuries to the Neck

Jason T. Lee, MD

The increasing reliance on and relative availability of high-quality cross-sectional imaging of vascular structures has altered the workup of patients with blunt trauma. Traditional 2-dimensional angiography and the extensive resources necessary to perform these procedures in critically injured patients have largely been replaced by rapid computed tomography angiography and 3-dimensional reconstructions; therefore, the early diagnosis and treatment of vascular injuries in patients with blunt trauma have undergone a paradigm shift. Neck injury provides a particularly challenging subcohort of patients with trauma because it is often difficult to predict the progression of carotid and vertebral injuries and its effect on neurologic status. Crawford et al1 presented a timely and contemporary study of 100 patients with 138 blunt cerebrovascular injuries (BCVIs), focusing on the more challenging diagnostic dilemma of the indeterminate injury.

This study demonstrated that from 2005 to 2014, computed tomography angiography was used as the initial diagnostic modality to diagnose BCVI in more than 90% of patients, highlighting the paradigm shift toward this technology. It was not long ago that surgical residents and students learned the indications for catheter-directed angiography based on zones of the neck. Of the 138 BCVI injuries identified in the Crawford et al study,79 were clearly identified by the well-accepted Denver criteria grade and subsequently triaged and treated appropriately, most often with antiplatelet and/or anticoagulation therapy. These 79 injuries led to 12 cerebral vascular accidents/transient ischemic attacks, with an overall 30-day mortality of 15.2%, highlighting the importance of a prompt diagnosis and treatment.

The main importance of this study is related to the natural history of the indeterminate because it has not been rigorously reported on in the prior literature. In this series, there were 59 indeterminate BCVIs, 73% that underwent serial imaging, which then translated to 24% reclassified as actual BCVIs, 39% that resolved, and 37% that were indeterminate. Unfortunately, there was no demographic or radiographic factor that correlated with either rate of resolution or reclassification of indeterminate BCVIs. Fortunately, the cerebral vascular accident/transient ischemic attack rate was only 5.1% in the indeterminate BCVI group. The Crawford et al study3 shows the importance of trying to correctly classify BCVI, particularly when the initial study modality is indeterminate. Of the BCVIs that were still indeterminate after the serial study, most were vertebral artery injuries that carried a much lower cerebral vascular accident/transient ischemic attack risk than carotid artery injury.