Blunt traumatic occlusion of the internal carotid and vertebral arteries

Clinical article

RYAN P. MORTON, M.D.,1 BRIAN W. HANAK, M.D.,1 MICHAEL R. LEVITT, M.D.,1 KATHLEEN R. FINK, M.D.,2 ERIC C. PETERSON, M.D.,2 MARCELO D. VILELA, M.D.,4 LOUIS J. KIM, M.D.,1 AND RANDALL M. CHESNUT, M.D.1

Departments of 'Neurological Surgery and 2Radiology, University of Washington at Harborview Medical Center, Seattle, Washington; 3Department of Neurological Surgery, University of Miami Miller School of Medicine, Miami, Florida; and 4Department of Neurological Surgery, Hospital Mater Dei, Belo Horizonte, Brazil

Object. The stroke rate, management, and outcome after blunt cerebrovascular occlusion (Biffl Grade IV injury) is not well defined, given the rarity of the disease. Both hemodynamic failure and embolic mechanisms have been implicated in the pathophysiology of subsequent stroke after blunt cerebrovascular occlusion. In this study, the authors evaluated their center’s experience with Biffl Grade IV injuries, focusing on elucidating the mechanisms of stroke and their optimal management.

Methods. A retrospective review identified all internal carotid artery (ICA) or vertebral artery (VA) Biffl Grade IV injuries over a 7-year period at a single institution.

Results. Fifty-nine Biffl Grade IV injuries were diagnosed affecting 11 ICAs, 44 unilateral VAs, and 2 bilateral VAs. The stroke rates were 64%, 9%, and 50%, respectively. Of the 11 Biffl Grade IV ICA injuries, 5 presented with stroke while 2 developed delayed stroke. An ipsilateral posterior communicating artery greater than 1 mm on CT angiography was protective against stroke due to hemodynamic failure (p = 0.015). All patients with Biffl Grade IV injuries affecting the ICA who had at least 8 emboli per hour on transcranial Doppler (TCD) ultrasonography developed an embolic pattern of stroke (p = 0.006). Treatment with aspirin versus dual antiplatelet therapy had a similar effect on stroke rate in the ICA group (p = 0.5) and all patients who suffered stroke either died (n = 3) or required a decompressive hemicraniectomy with subsequent poor outcome (n = 4). All 10 strokes associated with Biffl Grade IV VA injuries were embolic and clinically asymptomatic. In VA Biffl Grade IV injury, neither the presence of emboli nor treatment with antiplatelet agents affected stroke rates.

Conclusions. At the authors’ institution, traumatic ICA occlusion is rare but associated with a high stroke rate. Robust collateral circulation may mitigate its severity. Embolic monitoring with TCD ultrasonography and prophylactic antiplatelet therapy should be used in all ICA Biffl Grade IV injuries. Unilateral VA Biffl Grade IV injury is the most common type of traumatic occlusion and is associated with significantly less morbidity. Embolic monitoring using TCD and prophylactic antiplatelet therapy do not appear to be beneficial in patients with traumatic VA occlusion. (http://thejns.org/doi/abs/10.3171/2014.2.JNS131658)

Key Words • blunt cerebrovascular injury • Biffl grade • stroke • trauma • TCD • emboli • internal carotid artery • vertebral artery • vascular disorders • traumatic brain injury

Blunt cerebrovascular injury is defined as a vertebral or carotid arterial structural wall injury resulting from nonpenetrating trauma. Biffl and colleagues created a now commonly used grading scale based on the radiographic appearance of the injured artery. Complete traumatic parent vessel occlusion (Biffl Grade IV) is believed to be associated with the greatest risk of ischemic stroke, reported in 13%–24% of vertebral artery (VA) and 50%–90% of internal carotid artery (ICA) occlusions in a few small series. Both embolic and hemodynamic failure mechanisms (watershed infarcts) have been implicated in the development of stroke after traumatic vascular occlusion but the optimal treatment of such injuries has not been well established. We studied our relatively large population of Biffl Grade IV injuries to determine the rate of stroke and efficacy of our protocols.

Methods

Study Population

This study was approved by the Institutional Review Board of Harborview Medical Center in Seattle, Washington. We retrospectively examined all head and neck CT angiography reports using a key-word computerized search of all reports containing the key words “Biffl,” and/or “blunt,” and/or “traumatic,” and/or “dissection,” and/or “occlusion,” and/or “intimal flap,” and/or “narrowing” between 2005

Abbreviations used in this paper: ACoA = anterior communicating artery; ICA = internal carotid artery; mRS = modified Rankin Scale; PCoA = posterior communicating artery; TCD = transcranial Doppler; VA = vertebral artery.
Blunt traumatic occlusion of the ICA and VA

and 2011. These reports were then manually sorted and the imaging reviewed to find all blunt cerebrovascular injuries that were diagnosed by a board certified neuroradiologist. Radiological and clinical data were recorded. In patients who suffered stroke, the pattern of diffusion restriction on MRI or hypodensity on CT was used to determine if the stroke pattern was primarily due to hemodynamic failure (Fig. 1A) or emboli (Fig. 1C). Some cases presented with or developed a combination of both mechanisms (Fig. 1D). Functional outcomes were assessed at 6 months or longest follow-up evaluation after injury using the modified Rankin Scale (mRS).

**Screening Protocol**

Our institution uses adapted Denver2 and Memphis7 criteria (Table 1) as indications for obtaining vascular imaging after trauma. After blunt cerebrovascular injury was diagnosed, daily transcranial Doppler (TCD) ultrasonography embolic monitoring was performed for at least 3 days, starting within 24 hours of admission. Embolic monitoring was performed on the basilar or posterior cerebral arteries in the case of VA injuries, and on the ipsilateral middle cerebral artery in the case of ICA injuries. Monitoring was performed for 15 minutes, and any high-intensity transient signals counted were multiplied by 4 to estimate the rate of emboli per hour.11 Neurological examinations were performed hourly for at least 24 hours in all patients, and any detected deficit prompted immediate cerebral imaging. Brain MRI was also obtained in some comatose patients or in those with severe spinal injury precluding neurological examination.

**Treatment Protocol**

All patients diagnosed with Biffl Grade IV injury received daily aspirin (325 mg orally or 300 mg rectally) within 24 hours of admission unless contraindicated due to intracranial hemorrhage, need for other surgical procedures, or other systemic bleeding concerns. In patients with such contraindications, aspirin was started as soon as it was considered safe by the critical care team. In patients with ICA occlusion, 75 mg of clopidogrel daily was initiated prophylactically in addition to aspirin in 4 of 10 patients who survived longer than 24 hours. Patients with persistent or progressive embolic signals on TCD despite antiplatelet therapy were considered for systemic heparin therapy as an alternative by discretion of the treatment team. Daily TCD monitoring continued until emboli were eliminated or devastating stroke occurred. Patients were maintained on oral antiplatelet/anticoagulation agents for at least 6 weeks after injury.

**Collateral Circulation Analysis**

Computed tomography angiography of the head and neck was performed with the patient receiving 100 ml of iodinated contrast at a rate of 4 ml/sec. During peak arterial phase, 1-mm axial images were obtained from the aortic arch through the circle of Willis. Studies were performed on either a GE LightSpeed 16-slice CT scanner or a Siemens Somatom Definition AS 64-slice CT scanner. Computed tomography angiography images were reviewed on a GE PACS workstation. Circle of Willis components, including the anterior communicating artery (ACoA) and right and left posterior communicating arteries (PCoAs), were identified on axial source images.

---

**Table 1: A combination of Denver and Memphis screening criteria for blunt cerebrovascular injury in the setting of trauma**

<table>
<thead>
<tr>
<th>Any of the following:</th>
</tr>
</thead>
<tbody>
<tr>
<td>a) hemorrhage of potential arterial origin;</td>
</tr>
<tr>
<td>b) expanding cervical hematoma;</td>
</tr>
<tr>
<td>c) carotid bruit, patient &lt;50 yrs old;</td>
</tr>
<tr>
<td>d) cerebral infarction on CT or MRI;</td>
</tr>
<tr>
<td>e) unexplained central or lateralizing neurological deficit, TIA, or Horner's syndrome</td>
</tr>
</tbody>
</table>
| or high-energy mechanism w/:
  | a) LeFort II or III fracture; |
  | b) complex mandible fracture; |
  | c) punctate hemorrhage in the brainstem, corpus callosum, and/or basal ganglia (w/ GCS score <6); |
  | d) cervical spine subluxation; |
  | e) C-spine fractures extending to the transverse foramen; |
  | f) any fracture of C-1, C-2, or C-3 |

* C-spine = cervical spine; GCS = Glasgow Coma Scale; TIA = transient ischemic attack.

---

**Fig. 1.** Axial diffusion-weighted MR images (A, C, and D) and non-contrast CT scan (B) showing patterns of stroke. Case 6. Sequence revealing a small right-sided watershed infarction (A) that subsequently progressed (B). The origin of this patient’s stroke was characterized as “hemodynamic failure.” Case 11. Sequence revealing infarction in two different arterial distributions (C), which was characterized as “embolic” in nature. This patient’s stroke subsequently progressed, and he developed stroke from hemodynamic failure on subsequent MRI (D), creating a mixed picture.
Transverse vessel measurements of the maximal luminal diameter were made at the point the vessel was optimally visualized by a board-certified neuroradiologist (K.R.F.).

Results

From approximately 40,000 trauma admissions during the 7-year study period, 400 vessels with blunt cerebrovascular injury were detected (1%). Fifteen percent of all blunt cerebrovascular injuries (n = 59) were Biffl Grade IV injuries (11 ICAs, 44 unilateral and 2 bilateral VAs). At our institution, the overall stroke rate was 20%. Rates of stroke for ICA, VA, and bilateral VA occlusion were 64%, 9%, and 50%, respectively.

Internal Carotid Artery Biffl Grade IV Injury

There were 11 Biffl Grade IV ICA injuries (Table 2). Five patients (46%) presented with neurological deficit/stroke (1 embolic, 3 hemodynamic failure/watershed, and 1 combination), while 2 (18%) developed strokes in a delayed fashion (both a combination of embolic stroke and hemodynamic failure). All patients who suffered stroke saw marked progression of their stroke (Fig. 1) and either died (n = 3) or required decompressive hemicraniectomy with poor outcome (mRS Score > 2) at the longest follow-up (n = 4). The 4 patients without stroke had good outcome (mRS Score 1) at longest follow-up.

Collateral Circulation. All 6 patients who suffered a hemodynamic failure pattern of stroke had an ipsilateral PCoA of less than 1 mm diameter on CT angiography. Three of the 4 patients without stroke had an ipsilateral PCoA diameter greater than 1 mm, while the fourth (Table 2, Case 5) developed rapid recanalization of his ICA as described later. Thus, an ipsilateral PCoA greater than 1 mm, while the fourth (Table 2, Case 5) developed rapid recanalization of his ICA as described later. Anterior communicating artery and contralateral PCoA diameter varied but did not correlate with stroke due to hemodynamic failure.

Embolic Monitoring. Nine of the 11 patients with Biffl Grade IV ICA injuries underwent TCD emboli monitoring. Of the 2 patients who did not undergo monitoring, one (Case 7, Table 2) progressed rapidly to brain death in the emergency room, and the other (Case 9, Table 2) had an unstable airway on admission and suffered a hemodynamic failure stroke while in the operating room, potentially from low blood pressure. For the 9 monitored patients, 3 (33%) had 8 or more emboli per hour and all had radiographic evidence of embolic stroke (p = 0.006). Two of these patients had progressive emboli (up to 72/hr) despite aspirin and clopidogrel therapy. One of these patients was treated with systemic heparin that did reduce the number of emboli to 0, but nonetheless still required a decompressive hemicraniectomy for malignant edema that developed due to the ischemic burden. There was no effect of the addition of clopidogrel on the number of emboli or the rate of embolic stroke (p = 0.5) in this small cohort.

There was one case (Case 5, Table 2) of rapid ICA recanalization. This 18-year-old man presented with a left Biffl Grade IV ICA injury (Fig. 2A) without stroke and with poor collateral circulation (ipsilateral PCoA diameter < 1.0 mm) on CT angiography. He underwent cerebral angiography that revealed partial recanalization of his previously occluded ICA, with subsequent residual Biffl Grade II and III (pseudoaneurysm) injuries (Fig. 2B). His MRI confirmed no embolic stroke (not shown). He was treated with dual antiplatelet therapy and received 3 days of TCD ultrasonography with emboli monitoring. He never developed emboli. A follow-up angiogram at 6 weeks showed progressive stenosis of the ICA with no progression of the pseudoaneurysm. He underwent elective stent placement at this time (Fig. 2C arrow). His 6-month mRS score was 1.

Unilateral VA Biffl Grade IV Injury

Forty-four unilateral VA injuries were detected and none presented with clinical evidence of stroke. Overall, 4 patients (9%) developed stroke as detected by follow-up imaging (CT or MRI). All had an embolic pattern in the cerebellar hemisphere or occipital lobes. No patient developed malignant edema and no surgical or endovascular interventions were performed.

Antiplatelet Therapy. Antiplatelet therapy was initiated within 24 hours of admission in 25 patients, after 24 hours in 13 patients, and never in 6 patients due to other

<table>
<thead>
<tr>
<th>Case No.</th>
<th>PCoA Diameter (mm)</th>
<th>ACoA Diameter (mm)</th>
<th>Hemodynamic Failure/Embolic Stroke</th>
<th>≥8 Emboli/Hr</th>
<th>Treatment</th>
<th>mRS Score at Longest Follow-Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.1</td>
<td>1.0</td>
<td>none</td>
<td>no</td>
<td>aspirin</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>1.4</td>
<td>1.2</td>
<td>none</td>
<td>no</td>
<td>aspirin</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>1.5</td>
<td>1.5</td>
<td>none</td>
<td>no</td>
<td>aspirin</td>
<td>1</td>
</tr>
<tr>
<td>4</td>
<td>1.2</td>
<td>1.0</td>
<td>stroke</td>
<td>yes</td>
<td>aspirin + clopidogrel 3</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>0.5</td>
<td>0.7</td>
<td>failure</td>
<td>no</td>
<td>aspirin + clopidogrel 1</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>0.8</td>
<td>1.3</td>
<td>failure</td>
<td>no</td>
<td>aspirin + clopidogrel 4</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>not visualized</td>
<td>0.4</td>
<td>failure</td>
<td>not applicable none; brain dead</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>0.6</td>
<td>1.5</td>
<td>failure</td>
<td>no</td>
<td>aspirin</td>
<td>6</td>
</tr>
<tr>
<td>9</td>
<td>0.5</td>
<td>1.1</td>
<td>failure</td>
<td>not applicable none (airway issue)</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>not visualized</td>
<td>0.9</td>
<td>failure + stroke</td>
<td>yes</td>
<td>aspirin + clopidogrel 5</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>0.6</td>
<td>0.8</td>
<td>failure + stroke</td>
<td>yes</td>
<td>aspirin</td>
<td>6</td>
</tr>
</tbody>
</table>
severe traumatic injuries. Starting aspirin therapy within 24 hours showed a trend toward reducing the incidence of emboli (27% vs 6.9%) but this was not significant (p = 0.19). Additionally, there was no difference in stroke rate between the groups.

**Embolic Monitoring.** Six patients with unilateral Biffl Grade IV VA injuries developed emboli on routine TCD monitoring. All 6 received follow-up MRI and only 1 had evidence of stroke. Of the 38 patients without emboli, 3 (79%) developed stroke on MRI. Thus, the presence of emboli was not predictive of stroke (p = 0.45) in this group; overall sensitivity and specificity for positive emboli monitoring and stroke was 25% and 87.5%, respectively. The positive and negative predictive values of emboli and stroke were 16.7% and 92%, respectively.

**Bilateral VA Biffl Grade IV Injury**

Two patients presented with bilateral Biffl Grade IV VA injuries. One of these patients sustained a C-5 flexion/distraction fracture and the other a unilateral C3–4 locked facet injury. Both patients underwent MRI before surgical correction of the spinal fracture. Neither patient developed emboli on routine TCD. Both were treated with aspirin, one within 24 hours and the other within 48 hours of presentation. The patient with C-5 quadriplegia underwent follow-up MRI 3 days later that demonstrated multiple strokes in the cerebellum and thalamus. The other patient, with C-4 quadriplegia, had no emboli, large bilateral PCoAs (> 1.5 mm), and negative follow-up MRI.

**Discussion**

At our institution, complete occlusion of the ICA carries a substantial risk of stroke and poor outcome, with 64% of patients in the current series presenting with, or developing, stroke. This rate is consistent with previous smaller studies in the literature. We found that an ipsilateral PCoA of more than 1 mm on CT angiography was protective against watershed stroke. This result is similar to those in studies of chronic ICA occlusion and in acute basilar artery or VA occlusion studies in which PCoA diameters of less than 1 mm on MR angiography were predictive of ischemia. At our institution, outcome for patients suffering stroke from Biffl Grade IV ICA occlusion was uniformly poor, regardless of stroke mechanism.

Based on our institutional experience, in patients with complete ICA occlusion and stroke, rapid assessment of collateral circulation on CT angiography may help determine future management. In patients with poor collateral circulation, treatment should focus on keeping mean arterial pressure elevated in efforts to prevent watershed ischemia, as was present in our series. Given the poor natural history of patients with an ipsilateral PCoA diameter less than 1 mm, a cerebral angiogram could be considered to evaluate for a “string sign” or a recanalization amenable to further endovascular or anticoagulation treatment, depending on the confounding systemic injuries. In patients with an ipsilateral PCoA diameter greater than 1 mm on CT angiography, single antiplatelet therapy using aspirin appears to be just as effective as dual antiplatelet therapy in our small study. Should a patient develop 8 or more emboli per hour on daily TCD monitoring, adding a second antiplatelet agent or switching to heparin infusion could be considered prophylactically. Regardless of treatment, a lack of collateral circulation or recanalization as well as a high embolic burden are associated with poor rates of survival and poor functional outcome. Additionally, prospective evaluation of any of these recommendations is clearly warranted.

Heparin is the most widely reported anticoagulant for complete occlusion from blunt cerebrovascular injury. However, the observed intracranial hemorrhage rate of 8%–16% in these patients coupled with the fact that 36% of patients with blunt cerebrovascular injury are not candidates for intravenous anticoagulation because of systemic traumatic injuries, has lead many centers to initiate antiplatelet therapy instead. Several retrospective studies have reported that neurological outcome with antiplatelet therapy is equivalent or superior to heparin anticoagulation.

The natural history of traumatic VA occlusion at our institution was more benign with a 9% stroke rate, all embolic in nature. None of these strokes were symptomatic and none required surgical intervention. Our stroke rate is lower than the 13%–24% reported in previous series.
We did not find a strong association with embolic monitoring and stroke, and an MR image was obtained only if neurological status was indeterminate or unknown. It is likely that smaller, asymptomatic strokes associated with VA occlusion remained undiagnosed due to a lack of indication for MRI screening, but these may not be clinically relevant.

In patients with traumatic occlusion of the VA, daily TCD embolic monitoring was not helpful in diagnosing subsequent stroke, although our stroke rate is likely under-reported due to a lack of routine MRI in asymptomatic patients. Additionally, antiplatelet administration was given at various times—within 24 hours, after 24 hours, and never—without a significant reduction in emboli or stroke rate regardless of which cohort patients were in. This suggests that neither TCD screening nor prophylactic antiplatelet administration should be regularly used in the management of traumatic VA occlusion. Our data do not suggest routine MRI for asymptomatic patients with VA occlusion, although we are unable to determine from this retrospective study whether TCD monitoring or antiplatelet therapy is necessary after the diagnosis of stroke is made.

This study suffers from several limitations. First, it is a retrospective study, and although patients were identified consecutively and treated according to a defined protocol, there was no control arm in the study. Second, even though our series of ICA occlusions is the largest reported, the number of patients is still small, reflecting the rarity of this condition but also reducing our statistical power. Third, despite the fact that our screening criteria were quite broad, there is a real chance we may have missed patients who did not meet our modified Denver/Memphis criteria (Table 1) who ultimately had an asymptomatic blunt injury to the ICA or VA. Fourth, this study is a single institutional experience and the results and conclusions should be tempered with this limitation. Lastly, the true stroke rate among patients with VA occlusion remains undefined, as MRI was not routinely used in all patients.

Conclusions

Traumatic ICA occlusion is rare but associated with a high stroke rate. In this study, all patients who suffered stroke either died or required decompressive craniotomy due to malignant progression. Robust collateral circulation may mitigate its severity. Embolic monitoring with TCD and prophylactic antiplatelet therapy should be used in all ICA occlusions. Unilateral VA occlusion is more common and its detectable stroke rate is low. Embolic monitoring with TCD and prophylactic antiplatelet therapy does not appear to be beneficial in patients with traumatic VA occlusion.

Disclosure

Dr. Kim has ownership in, and is a patent holder for, SPI Surgical, Inc.; serves as a consultant to Aesculap and MicroVention; and has received clinical or research support for this study from Volcano, Inc.

Author contributions to the study and manuscript preparation include the following. Conception and design: Morton, Hanak, Peterson, Vilela, Kim, Chesnutt. Acquisition of data: Morton, Hanak, Levitt, Fink, Vilela, Chesnutt. Analysis and interpretation of data: Morton, Hanak, Levitt, Fink, Chesnutt. Drafting the article: Morton, Hanak, Levitt, Fink, Peterson, Kim. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Morton. Statistical analysis: Morton. Administrative/technical/material support: Fink, Kim, Chesnutt. Study supervision: Peterson, Vilela, Kim, Chesnutt.

References