Timing and mechanism of ischemic stroke due to extracranial blunt traumatic cerebrovascular injury

Clinical article

CHRISTOPH J. GRIESENNAUER, M.D.,1 J. BRETT FLEMINING, M.D.,1 BOYD F. RICHARDS, M.D.,1 LUIS P. CAVA, M.D.,2 JOEL K. CURÉ, M.D.,2 DURAI D. YOUNAN, M.D.,1 LIMIN ZHAO, M.D.,3 ANDREI V. ALEXANDROV, M.D.,3 KRISTIAN BARNLINN, M.D.,1 TRACY TAYLOR, R.N.,1 AND MARK R. HARRIGAN, M.D.1

Departments of 1Surgery, 2Radiology, and 3Neurology, University of Alabama at Birmingham, Alabama

Object. Extracranial cerebrovascular injury is believed to be an important cause of neurological injury in patients who have suffered blunt trauma. The authors sought to determine the timing and mechanisms of ischemic stroke in patients who suffered traumatic cerebrovascular injury (TCVI).

Methods. This is a prospective study of all patients with TCVI who were admitted to a Level I trauma center during a 28-month period. All patients who suffered blunt trauma and had risk factors for TCVI underwent screening CT angiography (CTA) of the head and neck on admission. All patients with either an ischemic stroke or CTA suggesting TCVI underwent confirmatory digital subtraction angiography (DSA). Patients with DSA-confirmed TCVI were treated with 325 mg aspirin daily; all patients were observed during their hospitalization for the occurrence of new ischemic stroke. In addition, a subset of patients with TCVI underwent transcranial Doppler ultrasonography monitoring for microembolic signals.

Results. A total of 112 patients had CTA findings suggestive of TCVI; 68 cases were confirmed by DSA. Overall, 7 patients had an ischemic stroke in the territory of the affected artery prior to or during admission. Four of the patients had their event prior to diagnosis with CTA and 2 occurred prior to DSA. In 1 patient the ischemic stroke was found to be due to an extracranial atherosclerotic carotid plaque, and this patient was excluded from further analysis. All patients with ischemic stroke had brain CT findings consistent with an embolic mechanism. Two (8.7%) of 23 monitored patients with TCVI had microembolic signals on transcranial Doppler ultrasonography.

Conclusions. Most ischemic strokes due to TCVI are embolic in nature and occur prior to screening CTA and initiation of treatment with aspirin.

(key words • carotid artery • vertebral artery • blunt trauma • ischemic stroke • vascular disorders)

Traumatic cerebrovascular injury due to high-energy nonpenetrating injury has increasingly been recognized recently to be due to aggressive screening protocols using cerebral angiography or CTA. Despite the ability to reliably detect TCVI in patients with injury patterns known to be associated with extracranial CA or VA injuries, management of these patients is controversial, and no standard of care has yet been established.9,24 Numerous authors have recommended either anticoagulation therapy4,7,10,13,17,28 or antiplatelet therapy12,19,23,41 for patients with TCVI, based on the idea that TCVI is a significant risk factor for ischemic stroke. However, systemic anticoagulation carries a significant risk of hemorrhage in trauma patients,4,17,21,28,29 and the efficacy of antiplatelet therapy remains unproven. Although a number of studies have reported an association between TCVI and ischemic stroke, only a handful of studies have examined the timing of stroke in this setting26,40 and none have attempted to clarify the mechanism of stroke in patients with TCVI. Here, we report on patients sustaining a new ischemic stroke from TCVI collected from a prospective cohort including all patients with TCVI admitted to a Level I trauma center over a 28-month period.

Abbreviations used in this paper: BA = basilar artery; CA = carotid artery; CTA = CT angiography; DSA = digital subtraction angiography; GCS = Glasgow Coma Scale; ICA = internal carotid artery; MCA = middle cerebral artery; MES = microembolic signal; TCD = transcranial Doppler; TCVI = traumatic cerebrovascular injury; VA = vertebral artery.
Methods

All patients admitted to a Level I trauma center during a 28-month period were prospectively studied for TCVI. Multislice CTA with contrast load for the neck was performed on admission in all patients who sustained blunt trauma and had risk factors for TCVI (skull fracture, cervical spine fracture, severe head injury, Le Fort II or III fracture, Horner syndrome, focal neurological deficit, or cervical soft-tissue injury) upon presentation to the trauma center. Trauma patients presenting with signs and symptoms of an ischemic stroke as well as asymptomatic patients with CTA findings suggestive of TCVI underwent confirmatory DSA. All patients with DSA-confirmed TCVI were treated with daily aspirin (325 mg), which was started at the time of diagnosis. All patients were observed for the occurrence of a new ischemic stroke during their hospitalization.

A subset of patients with DSA-confirmed TCVI underwent TCD ultrasonography monitoring for MESs when monitoring of an intracranial vessel distal to TCVI was technically feasible. Each TCD ultrasonography examination was done prior to initiation of aspirin treatment. The TCD ultrasonography studies were done with 2-MHz, power motion (M-mode) Doppler (PMD-150, Spencer Technologies, Inc.). Monitoring of the MCA or BA corresponding to the extracranial CA or VA injury was done for 30 minutes. Microembolic signal detection was done using a low-gain Doppler spectra display, 64-point fast Fourier transform analysis, and a sample volume of 3 mm. The MESs were identified according to previously published criteria. The detection threshold of the relative intensity increase for MESs was set to 4 dB or greater. This study was approved by the institutional review board at the University of Alabama at Birmingham.

Results

During a 28-month period (April 1, 2008, to March 31, 2011), 6631 patients with blunt trauma were admitted. A total of 1219 of these patients (18%) with risk factors for TCVI underwent screening CTA. A total of 112 patients (9.2%) had CTA findings indicating TCVI; of these, 68 patients had a TCVI confirmed by DSA. The average time from admission to CTA was 9.82 hours (median 2.08 hours, standard deviation 6.16 hours) (Fig. 1 gray-shaded graph). Four of these patients (Cases 1–4) (Figs. 2 and 3) had their event prior to diagnosis with CTA, and 1 patient (Case 5) (Fig. 4A and B) suffered stroke after CTA but prior to DSA diagnosis of TCVI. One patient (Case 6) underwent screening CTA on admission that was negative for TCVI; this patient suffered a delayed stroke later during the hospital stay (Fig. 4C and D). Four patients (66%) were involved in a motor vehicle collision, 1 patient (16%) was a motorcycle accident victim, and 1 patient (16%) was assaulted. Five patients with anterior circulation infarctions were found to have a total of 6 CA injuries, and 1 patient sustained a posterior circulation infarction (Case 4) related to a VA injury. Of the 5 patients with anterior circulation infarctions, a left ICA injury was present in 3 patients (Cases 2, 3, and 5), a right ICA injury in 1 patient (Case 6), and bilateral ICA injuries in 1 patient (Case 1).

Patients With an Ischemic Event Prior to CTA and DSA.

Four patients (Cases 1–4) developed a TCVI-related cerebral ischemic event prior to CTA and DSA. Two patients had left ICA injuries (Cases 2 and 3) and sustained infarctions of the MCA territory; one of these patients (Case 3) also had the posterior cerebral artery territory affected. Both patients experienced symptoms of left cerebral infarction including dysphasia and contralateral hemi- or monoparesis before CTA and DSA. Other than a focal neurological deficit (Cases 2 and 3) and a severe head injury (GCS Score 6) (Case 2), no other known risk factors for TCVI were present. Both patients were treated with antiplatelet therapy; the patient in Case 2 also underwent endovascular thrombectomy. At the time of discharge from the hospital, this patient had persistent dysphasia and hemiparesis. The patient in Case 3 left the hospital neurologically intact. The patient found to have bilateral ICA dissections (Case 1) presented with left hemiparesis on admission and received antiplatelet therapy. The focal neurological deficit was the only risk factor for TCVI that was present. The neurological deficit persisted upon discharge. One patient (Case 4) presenting after a motor vehicle collision sustained a dissection of the left VA that resulted in a left cerebellar infarction.

Patients With TCVI and Ischemic Stroke

Overall, 7 patients who suffered blunt trauma had an ischemic stroke upon arrival or during admission. All of these patients underwent CTA and subsequent confirmatory DSA. Radiographic findings were indicative of an embolic stroke mechanism in all patients. The ischemic stroke in one of the patients was found to be due to an extracranial atherosclerotic carotid plaque, and this patient was excluded from further analysis. The 6 remaining patients (male/female ratio 2:4, mean age 43 ± 13 years) had an ischemic stroke attributable to a TCVI (Table 1), representing incidences of 8.8% among patients with TCVI and 0.09% among all blunt trauma admissions. The average time from admission to CTA for the stroke patients was 4.4 hours (median 2.08 hours, standard deviation 6.16 hours) (Fig. 1 gray-shaded graph). Four of these patients (Cases 1–4) (Figs. 2 and 3) had their event prior to diagnosis with CTA, and 1 patient (Case 5) (Fig. 4A and B) suffered stroke after CTA but prior to DSA diagnosis of TCVI. One patient (Case 6) underwent screening CTA on admission that was negative for TCVI; this patient suffered a delayed stroke later during the hospital stay (Fig. 4C and D). Four patients (66%) were involved in a motor vehicle collision, 1 patient (16%) was a motorcycle accident victim, and 1 patient (16%) was assaulted. Five patients with anterior circulation infarctions were found to have a total of 6 CA injuries, and 1 patient sustained a posterior circulation infarction (Case 4) related to a VA injury. Of the 5 patients with anterior circulation infarctions, a left ICA injury was present in 3 patients (Cases 2, 3, and 5), a right ICA injury in 1 patient (Case 6), and bilateral ICA injuries in 1 patient (Case 1).

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Stroke in blunt traumatic cerebrovascular injury

The TCVI was detected on an initial screening CTA study that was obtained because of cervical spine fractures. This infarction remained clinically silent throughout the hospital stay.

Patient With an Ischemic Event After CTA and Prior to DSA. One patient (Case 5) became symptomatic after CTA but prior to the DSA. Computed tomography angiography showed a left CA occlusion. The patient was started on an aspirin regimen at that time. Digital subtraction angiography was performed within a few hours of diagnosis and was confirmed by a left CA dissection with thrombus. The patient initially had a GCS score of 8 and withdrew to stimulation in all extremities symmetrically. Prior to cerebral angiography on hospital Day 2, the patient developed right hemiparesis. The patient underwent endovascular thrombus aspiration and stent placement. At last clinical follow-up, the patient was still dysphasic and had right hemiparesis.

Patient With an Ischemic Event After Negative Screening CTA. One patient (Case 6) developed a delayed right MCA territory infarction due to a right ICA dissection. Since the patient had a cervical spine fracture, a screening CT angiogram was obtained on admission and was negative for TCVI, even on retrospective review. Thus, no antiplatelet agent was started. On hospital Day 10, the patient developed sudden onset of left upper-extremity weakness. She was started on an aspirin regimen at that time and had residual left-sided weakness upon discharge from the hospital.

Transcranial Doppler Ultrasonography Examination

A total of 23 patients underwent TCD ultrasonography examinations to assess for microemboli prior to starting aspirin therapy (Table 2). None of these patients had evidence of stroke. Two patients (8.7%, male/female ratio 1:1, mean age 42 ± 17 years) had MESs on TCD ultrasonography. One patient (Case 7) had bilateral ICA dissecting aneurysms and a left VA dissection; MESs were present in the right MCA at a rate of 2 MESs/hr. The other patient (Case 8) had a left VA dissecting aneurysm; MESs were identified in the BA at a rate of 10 MESs/hr. A repeat TCD ultrasonography examination performed on hospital Day 3 after the patient had been started on aspirin therapy showed resolution of the MESs.

Discussion

Our study showed a low rate (1.0%) of TCVI incidence among blunt trauma admissions within the range of previously reported incidences of TCVI among patients with blunt trauma, which range from 0.075% to 1.7%.12,22,27,38 The rate of ischemic stroke in this series was 8.8%. This stroke rate is comparable to one report40 and is lower than that in others.4,27,28

Timing of Ischemic Stroke in Patients With TCVI

Timing of diagnosis of ischemic stroke related to the time of diagnosis of TCVI is critical. In order for aggressive screening for TCVI to be justified, convincing clinical evidence is mandated to demonstrate that the ability to diagnose and treat TCVI actually results in prevention of stroke, the most serious complication of TCVI.26 Although some previous studies reported ischemic stroke rates of 25%–58% and mortality rates of 31%–59% in patients with a known traumatic dissection,1,14,33,37 a number of more recent studies have indicated a mortality rate of 10% or lower.19,28,29,36,41 A recent series of 147 patients with TCVI found an overall stroke rate of 12.2% with a stroke mortality rate of 50%. Carotid artery injuries were more likely to result in stroke (13.3%) than VA injuries (8.2%).40 Overall, compared with CA injuries, VA injuries might be more benign, given that unilateral VA injuries will only cause symptoms of vertebrobasilar ischemia in a minority of patients (12%–20%),1,28,35 and even bilateral occlusion may remain silent.4

The natural history of asymptomatic TCVI is largely...
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Mechanism</th>
<th>TCVI Lesion</th>
<th>Associated Injuries</th>
<th>Timing of Diagnosis</th>
<th>Timing of Stroke Onset</th>
<th>CTA Results</th>
<th>Cross-Sectional Imaging Findings</th>
<th>TCVI Grade</th>
<th>Neurological Findings on Admission</th>
<th>Neurological Findings on Discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>38, F</td>
<td>MCC</td>
<td>bilat ICA dissection</td>
<td>CHI, SDH, pelvic Fx, splenic laceration, mandible Fx, foot Fx</td>
<td>after stroke onset</td>
<td>before CTA/angiogram</td>
<td>CTA: rt ICA dissection aneurysm</td>
<td>CTH: rt MCA infarction</td>
<td>1</td>
<td>GCS Score 10T, dysconjugate gaze, lt hemiparesis</td>
<td>lt hemiparesis (GOS Score 2)</td>
</tr>
<tr>
<td>2</td>
<td>23, F</td>
<td>MVC</td>
<td>lt ICA dissection/thrombus</td>
<td>pelvic Fxs, lumbar spine transverse process Fx, femur Fx, retroperitoneal hematoma</td>
<td>after stroke onset</td>
<td>before CTA/angiogram</td>
<td>CTA: lt CCA, carotid bifurcation thrombus</td>
<td>CTH: lt MCA infarction</td>
<td>2</td>
<td>GCS Score 6T, rt hemiparesis</td>
<td>dysphasia, rt hemiparesis (GOS Score 3)</td>
</tr>
<tr>
<td>3</td>
<td>39, M</td>
<td>MVC</td>
<td>lt ICA dissection/occlusion</td>
<td>none</td>
<td>after stroke onset</td>
<td>before CTA/angiogram</td>
<td>CTA: lt ICA occlusion</td>
<td>MRI brain: lt MCA, PCA territory infarcts</td>
<td>4</td>
<td>GCS Score 15, rt facial droop, rt upper-extremity paresis</td>
<td>neurologically intact (GOS Score 5)</td>
</tr>
<tr>
<td>4</td>
<td>64, M</td>
<td>MVC</td>
<td>lt VA occlusion</td>
<td>C-2 VB, Type III odontoid Fxs, lt C-2 pars interarticularis Fx, rt C-1 lat mass Fx, lt C-6, T-1 facet Fxs, rib Fxs, clavicle Fx, T-8, thoracic transverse process Fxs, pulmonary effusion, scalp laceration</td>
<td>clinically silent ischemic event</td>
<td>before CTA/angiogram</td>
<td>CTA: lt VA occlusion</td>
<td>CTH: negative; MRI brain: lt cerebellar infarction</td>
<td>4</td>
<td>GCS Score 15, neurologically intact</td>
<td>neurologically intact (GOS Score 5)</td>
</tr>
<tr>
<td>5</td>
<td>47, F</td>
<td>assault, blunt trauma to head</td>
<td>lt ICA dissection/thrombus</td>
<td>rt hyoid Fx</td>
<td>before stroke onset</td>
<td>after CTA/before angiogram</td>
<td>CTA: lt ICA occlusion</td>
<td>CTH: lt MCA infarction</td>
<td>2</td>
<td>GCS Score 8T, withdraws to pain in all extremities</td>
<td>dysphasia, rt hemiparesis (GOS Score 3)</td>
</tr>
<tr>
<td>6</td>
<td>41, F</td>
<td>MVC</td>
<td>rt ICA dissection</td>
<td>C-2 VB Fx, lt acetabulum Fx, hip dislocation, lt knee Fx, bilat ankle injury, peritoneal free fluid, thyroid contusion, pulmonary contusion, rib Fx, neck soft-tissue swelling</td>
<td>after stroke onset</td>
<td>after CTA (screening CTA negative for TCVI) before angiogram</td>
<td>CTA: negative</td>
<td>MRI brain: lt ACA/MCA border zone infarction</td>
<td>1</td>
<td>GCS Score 11T, follows commands on rt, lt limited motor exam due to associated injuries</td>
<td>lt upper-extremity paresis, lt lower motor exam limited due to cast/immobilizer (GOS Score 3)</td>
</tr>
</tbody>
</table>

* ACA = anterior cerebral artery; CCA = common carotid artery; CHI = closed head injury; CTH = CT of head; Fx(s) = fracture(s); GOS = Glasgow Outcome Scale; MCC = motorcycle crash; MVC = motor vehicle crash; SDH = subdural hematoma; PCA = posterior cerebral artery; T = tracheostomy tube; VB = vertebral body.
unknown, and current treatment strategies are controversial. Several papers that emphasized the threat of TCVI of causing strokes did not provide any information about when the stroke occurred.\textsuperscript{3,8,13,18,21,28,29} Up to this point, only 2 papers actually commented on the temporal relationship between diagnosis of TCVI and occurrence of stroke.\textsuperscript{26,40} In our series, 4 (67\%) of the 6 patients who developed a TCVI-related stroke had symptoms prior to any kind of diagnostic workup including CTA or cerebral angiography and before any kind of treatment could be initiated. Even though 1 patient developed stroke-related symptoms shortly after TCVI was diagnosed by CTA, there was insufficient time for any effective treatment to be initiated. One patient developed a delayed new neurological deficit on hospital Day 10. This patient underwent initial screening CTA because of the presence of cervical fractures; the findings were false negative for TCVI. Thus, none of the strokes in this series were preventable. The time from admission to CTA was also determined. Half of the 68 patients with TCVI confirmed by DSA underwent CTA within 2.73 hours of admission. Five of the 6 patients who suffered stroke underwent CTA within 3 hours of admission.

While several published series have described a latency period between injury and infarction,\textsuperscript{4,5,7,11,13,20,21,25,29,30,32} our observation is more consistent with more recent reports in which a significant number of TCVI patients (44\%\textsuperscript{26} and 82\%\textsuperscript{26}) had evidence of strokes on admission. Mayberry et al.\textsuperscript{26} reported on 17 patients with blunt CA injury. During the time period of this study, workup for TCVI was only initiated for focal neurological signs or neurological symptoms unexplainable by results of CT scanning of the brain. No routine screening protocol based on previously recommended risk factors was in place.\textsuperscript{6} Eleven patients (65\%) developed a blunt CA injury–related stroke, and 9 (82\%) of those suffered stroke within 2 hours. The remaining 2 patients had a delayed diagnosis (9 and 12 hours after injury) and were treated with anticoagulation therapy. Only 1 of the 2 patients had

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{image1.png}
\caption{\textbf{A and B}: Case 1. Angiogram (anterior view) of the right extracranial carotid system showing a dissection of the cervical ICA (arrow) (A). Magnetic resonance FLAIR image showing an MCA territory infarction (arrow) (B). \textbf{C and D}: Case 2. Angiogram (anterior view) of the extracranial left carotid system showing a dissection and thrombus extending from the common carotid artery into the ICA (arrows) (C). Computed tomography scan showing a left MCA territory infarction (arrow) (D).}
\end{figure}

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{image2.png}
\caption{\textbf{A and B}: Case 3. Angiogram (lateral view) showing occlusion of the proximal left ICA (arrow) (A). Diffusion-weighted MR image showing focal areas of ischemic change in the left hemisphere (arrows) (B). \textbf{C and D}: Case 4. Angiogram (lateral view) of the cervical left VA showing occlusion of the artery (arrow) with partial reconstitution at C-1 (arrowhead) (C). Diffusion-weighted MR image showing the focal area of ischemic change in the left cerebellar hemisphere (D).}
\end{figure}
risk factors for TCVI, an observation also made in this series where 50% of patients who had a TCVI-related stroke did not have risk factors for TCVI. In another study, 44% of patients had evidence of stroke on admission, 33% were diagnosed within 72 hours, and in only 22% of patients stroke occurred after 1 week. Conclusively, there is convincing evidence that a significant number of TCVI-related strokes occur before any treatment can be initiated. However, this leads to the conclusion that patients with TCVI diagnosed by screening in the absence of TCVI-related stroke symptoms will likely have a benign course and are unlikely to develop a delayed ischemic event.

**Mechanism of Ischemic Stroke in Patients With TCVI**

No previous article has provided any information about the pattern or presumed mechanism of stroke in patients who have suffered TCVI. Thromboembolism and hemodynamic insufficiency are the 2 potential mechanisms of TCVI-related stroke in most cases. Patterns of stroke on imaging in this series were all consistent with a thromboembolic mechanism. A thromboembolic mechanism is also supported by the finding of MESs in 8.7% of patients with clinically silent TCVIs. An analysis of the types of vascular lesions may also shed light on the mechanisms of stroke in TCVI. In this study, 2 patients had Grade I and 2 patients had Grade II lesions. Both Grade I and II lesions cause luminal narrowing without blood flow arrest. Under these circumstances, hemodynamic insufficiency is an unlikely mechanism of stroke. Complete arterial occlusion (TCVI Grade IV) carries the highest risk of stroke and neurological morbidity since vessel occlusion may be associated with both thromboembolic injury and hemodynamic insufficiency. In this series both patients with Grade IV lesions had imaging findings consistent with a thromboembolic mechanism. Carotid artery occlusion appears to carry a higher risk of stroke than unilateral occlusion of the VA as evidenced by one study in which 58% of patients with Grade IV lesions of the CA developed a stroke compared with 13.2% with Grade IV VA lesions. This finding is intuitively explained by prominent collateral circulation in the posterior circulation. Conclusively, thromboembolism appears to be the most likely mechanism in the majority of patients with TCVI lesions.

**Microembolism in Patients With TCVI**

The presence of microemboli in patients with TCVI is another indicator of a thromboembolic mechanism in TCVI-related strokes. In spontaneous CA dissection, TCD ultrasonography has been used to monitor for the presence of cerebral emboli, and detection of embolic signals has been associated with an increased risk of ischemic events in patients with spontaneous dissections. Utilization of TCD ultrasonography in the setting of TCVI is less established even though embolic signals on TCD ultrasonography have been reported in up to 60% of patients with traumatic CA dissections at some point during their hospital course. Transcranial Doppler ultrasonography monitoring for embolic signals also allows assessing the success of the initiated therapy. In sponta-

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**TABLE 2: Patients with MESs on TCD ultrasonography**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Mechanism of Injury</th>
<th>TCVI Lesion</th>
<th>Associated Injuries</th>
<th>TCD Ultrasonography Results</th>
<th>CTA Results</th>
<th>TCVI Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>30, F</td>
<td>MVC</td>
<td>bilat ICA dissecting aneurysm, lt VA dissection</td>
<td>facial Fx, pelvic Fxs, extremity Fxs</td>
<td>MESs in rt MCA; 2 MESs/hr</td>
<td>bilat ICA dissections</td>
<td>3 (ICA), 1 (VA)</td>
</tr>
<tr>
<td>8</td>
<td>55, M</td>
<td>MVC</td>
<td>lt VA dissecting aneurysm/occlusion</td>
<td>C-2 odontoid, VB, lt lat mass Fxs</td>
<td>MESs in BA (10/hr); repeat TCD on hospital Day 3 after treatment w/ aspirin; no MESs</td>
<td>lt VA dissection/occlusion</td>
<td>3</td>
</tr>
</tbody>
</table>

![Fig. 4. A and B: Case 5. Angiogram (lateral view) of the extracranial left carotid system. A dissection of the ICA with an intraluminal thrombus (arrow) is present (A). Computed tomography showing a left MCA territory infarction (arrow) (B). C and D: Case 6. Angiogram (anterior view) of the extracranial ICA showing a relatively modest dissection of the medial wall of the vessel (arrow) (C). Diffusion-weighted MR image showing focal areas of ischemic change in the right hemisphere (arrow) (D).](image-url)
Stroke in blunt traumatic cerebrovascular injury

neous CA dissection, anticoagulation with heparin has been found to stop ultrasound evidence of microemboli in 70% of patients with ICA dissection within 24 hours of administration.1,2 Of the 23 patients who underwent TCD ultrasonography for MES monitoring, only 2 patients had MESs. Neither of these patients had radiographic or clinical evidence of stroke. In both patients, the MESs were associated with dissecting aeurysms (Grade III TCVI lesions). Repeat TCD ultrasonography after initiation of aspirin therapy showed resolution of MESs.

Yield of Screening CTA in Patients With Blunt Trauma

The most common indication for a screening CTA in the patients with TCVI in this study was a cervical spine fracture; of the 40 patients undergoing a CTA because of a cervical spine fracture, 1 (2.5%) was found to have an ischemic stroke attributable to the TCVI. Of the 4 patients who underwent CTA for cervical soft-tissue injury, 1 (25%) had a stroke. All of the patients who underwent CTA because of a focal neurological deficit were found to have an ischemic stroke. Ischemic stroke did not occur in any of the 4 patients who underwent CTA because of a seat belt sign. Therefore, the presence of a focal neurological deficit not explainable by other injuries was the most predictive of having a TCVI, and a seat belt sign was the least predictive. Interestingly, a seat belt sign, in the absence of other indicators of TCVI, has been previously reported to be a poor indicator of TCVI.15

Antithrombotic Therapy in Patients With TCVI

In this series, all patients with DSA-confirmed TCVI were treated with aspirin, and no hemorrhagic complications were encountered. Most authors agree that some form of antithrombotic therapy is appropriate for most patients who have suffered TCVI. The type of antithrombotic therapy is more controversial. Of all antithrombotic therapy options, intravenous heparin has traditionally been most popular. However, the rate of hemorrhagic complication is high,7,17,21,28,29 and about one-third of patients have comorbidities precluding them from receiving this type of therapy.40 On the other hand, antiplatelet agents such as aspirin or clopidogrel have been shown to be at least equivalent17,21,41 or even superior28 to anticoagulation therapy in combination with a more favorable side-effect profile.40 To this date, no prospective, randomized trial has been completed that shows superiority of one therapy over others. Several trials have demonstrated a lower rate of ischemic events in patients with TCVI receiving antithrombotic therapy than in those not receiving these agents.8,13,21,28,29,40 However, caution is warranted when conclusions are drawn from these trials. Stroke rates in patients with asymptomatic TCVI who were started on antithrombotic agents were compared with patients with TCVI in which a cerebral infarction was present at the time of diagnosis. Herein, the conclusion that antithrombotic therapy lowers the risk of cerebral ischemic events is flawed by the assumption that initially asymptomatic patients would have developed a stroke without antithrombotic therapy. This series provides evidence that the natural history of asymptomatic TCVI might be different and more benign and not warrant aggressive management with anticoagulation.

Limitations of the Study

Only symptomatic ischemic strokes were identified. Because routine follow-up imaging was not done in all patients who suffered TCVI, clinically silent strokes may have not been identified. Stein et al.40 reported on 3 patients with asymptomatic ischemic strokes in patients who have suffered TCVI discovered on imaging performed for other reasons. In addition, patients who have suffered blunt trauma frequently have multiple injuries, including traumatic neurological injuries. The presence of multiple injuries in these patients may have obscured the identification of TCVI-related neurological injuries as well as possible complications of aspirin therapy.

Conclusions

The overall incidence of ischemic stroke among patients who have suffered TCVI is relatively low. Our data suggest that the majority of TCVI-associated strokes occurring before the diagnosis of TCVI could have been diagnosed using a CTA screening protocol, and aspirin treatment could have been given to all patients with DSA-confirmed TCVIs.

Thus, the natural history of asymptomatic TCVI, identified by CTA screening, may be more benign than previously thought and might not require more aggressive intervention other than antiplatelet agents. Also, these data indicate a thromboembolic mechanism in patients with stroke caused by TCVI; this finding is a justification for antithrombotic medical therapy in patients with TCVI. A comprehensive prospective study of TCVI with neurological imaging in all patients would further clarify the timing and outcomes associated with TCVI.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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