Extracranial traumatic aneurysms due to blunt cerebrovascular injury

Clinical article

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Objective. Traumatic aneurysms occur in 10% of extracranial blunt traumatic cerebrovascular injuries (TCVI). The clinical consequences and optimal management of traumatic aneurysms are poorly understood.

Methods. A prospective study of TCVI at a Level I trauma center identified 7 patients with 19 extracranial traumatic carotid artery or vertebral artery aneurysms. An additional 6 patients with 7 traumatic aneurysms were followed outside of the prospective study, giving a total of 13 patients with 26 traumatic aneurysms. All patients were treated with 325 mg aspirin daily and underwent clinical and imaging follow-up beyond the initial hospitalization. Endovascular treatment was reserved for aneurysms demonstrating significant enlargement on follow-up imaging. Clinical and radiographic features were assessed.

Results. The 7 patients with traumatic aneurysms identified in the prospective cohort comprised 10.3% of all patients with TCVI. Two (15.4%) of the 13 total patients suffered an ischemic stroke in the setting of TCVI with traumatic aneurysm formation. No patient experienced an ischemic stroke or new symptoms after the initiation of antiplatelet therapy. Clinical and radiographic follow-up averaged 15.8 months (range 0.4–41.7 months) and 22.0 months (range 6.6–55.7 months), respectively. Ten (38.5%) of 26 aneurysms were not visualized on last follow-up, 10 (38.5%) were smaller, 1 (3.8%) was unchanged, and 5 (19.2%) were larger. Saccular aneurysms were more likely to enlarge than fusiform aneurysms (33.3% vs 11.8%). Results of a Fisher exact test tend to support the assertion that the 2 different aneurysm morphologies behave differently (p = 0.07). Two saccular aneurysms were treated with stenting.

Conclusions. The majority of traumatic aneurysms can be managed with an antiplatelet regimen of 325 mg aspirin daily and serial imaging. Saccular aneurysms have a greater tendency to enlarge when compared with fusiform aneurysms.

Key Words • carotid artery • vertebral artery • blunt trauma • dissecting aneurysm • traumatic aneurysm • pseudoaneurysm • traumatic cerebrovascular injury • vascular disorders

Blunt extracranial traumatic cerebrovascular injury (TCVI) results from high-energy trauma and is present in approximately 1% of patients admitted after blunt trauma.14,18,22 Traumatic nonpenetrating cerebrovascular injury is increasingly recognized among patients with blunt trauma due to aggressive screening protocols utilizing CT angiography (CTA). Approximately 3%–9% of patients with TCVI suffer ischemic stroke attributable to the cerebrovascular injury.14,18,22 Making carotid artery (CA) and vertebral artery (VA) injuries a significant cause of stroke in young people.19 Traumatic aneurysms of the extracranial CA and VA have been reported in 15%–23% and 4%–8% of TCVI cases respectively.2,22 The clinical consequences of extracranial CA and VA aneurysms in this setting are poorly understood. Although spontaneous extracranial CA and VA aneurysms have been associated with embolic ischemic stroke in 0%–21% of cases,10,23 the risk of thromboembolic stroke associated with traumatic extracranial aneurysms has not yet been defined. In addition, the history of traumatic aneurysms managed expectantly has not been assessed. Because of this lack of clarity, the management of traumatic aneurysms associated with TCVI is controversial. While some authors recommend conservative management with antithrombotic therapy, others advocate endovascular treatment with stent placement or coil embolization.14,19,23

A series of 13 patients (7 identified prospectively and 6 identified outside the prospective study) with traumatic aneurysms and follow-up extending beyond the initial hospitalization are presented.

Abbreviations used in this paper: CA = carotid artery; CTA = CT angiography; DSA = digital subtraction angiography; ICA = internal carotid artery; TCVI = traumatic cerebrovascular injury; VA = vertebral artery.
Methods

Traumatic cerebrovascular injury is defined as a blunt traumatic injury to the CA or VA resulting in dissection, traumatic aneurysm formation, occlusion, or transection. Traumatic aneurysms were defined as a focal dilation of the extracranial CA or VA wall following blunt trauma.

Prospective Study

All patients admitted to a Level I trauma center during a 28-month (April 1, 2008, to March 31, 2011) period were prospectively studied for TCVI. A 64-section multidetector CTA with contrast load for the neck was performed on presentation to the trauma center in all patients with blunt trauma and risk factors for TCVI (skull fracture, facial fractures, Horner’s syndrome, focal neurological deficit, cervical spine fracture, or cervical soft-tissue injury). Trauma patients presenting with signs and symptoms of an ischemic stroke, as well as asymptomatic patients with CTA findings suggestive of TCVI, underwent confirmatory digital subtraction angiography (DSA).

Additional Cases

An additional 6 patients, not enrolled in the prospective cohort, were included in this study. Two of these 6 patients presented prior to the prospective study, 1 was transferred from another hospital following partial evaluation and treatment, and the remaining 3 patients presented after completion of the prospective study.

Traumatic Aneurysm Measurement Technique

Both CTA and DSA were interpreted by the senior author (M.R.H.) in conjunction with 2 neurosurgical resident coauthors (P.M.F. and C.J.G.). Traumatic aneurysms were classified as either saccular or fusiform. Depth (in mm) was reported for saccular aneurysms. Both depth and length (in mm) were reported for fusiform aneurysms in the following manner: “x by y” mm with “x” as the depth and “y” as the length. On follow-up imaging, aneurysms were classified as being smaller, stable, or larger based on depth measurement.

Management of Patients With TCVI and Traumatic Aneurysms

All patients with TCVI were treated with 325 mg aspirin per day after diagnosis. Two patients underwent CA stenting for treatment of a traumatic aneurysm; these patients were found to have significant enlargement of their saccular internal carotid artery (ICA) traumatic aneurysms on follow-up imaging. All patients were observed for the occurrence of a new ischemic stroke during their hospitalization and were monitored after hospital discharge by clinic visits and serial imaging.

Statistical Analysis

The sociodemographic characteristics of the patients and aneurysms were summarized using means and medians for continuous variables and percentages for categorical variables. Univariate analysis was performed using the Fisher exact test, and the depth at initial diagnosis was adjusted for using logistic regression, ignoring the intraclass correlation due to multiple outcomes per patient. The analysis was performed using SAS software (version 9.3, SAS Institute Inc.). This study was approved by the institutional review board.

Results

Prospective Study

During a 28-month period (April 1, 2008, to March 31, 2011), 6631 patients with blunt trauma were admitted to the University of Alabama at Birmingham; 1219 patients with risk factors for TCVI underwent screening CTA. A total of 112 patients had CTA findings indicating TCVI; of these, 68 patients had a TCVI confirmed by DSA and 44 patients had no evidence of TCVI on DSA. Of these 68 patients with DSA-confirmed TCVI, 7 (10.3%) were found to have traumatic aneurysms of the extracranial CA and/or VA. The overall incidence of traumatic aneurysm formation following blunt trauma with risk factors for TCVI was 0.5%. Patient characteristics are presented in Table 1.

Additional Cases

Six additional patients with traumatic aneurysms were included in this study. Two of these 6 patients presented prior to the prospective study, 1 was transferred from an outside hospital following partial evaluation and treatment, and the remaining 3 presented after completion of the prospective study. Patient characteristics are presented in Table 1.

Patients With Traumatic Aneurysms

Seven of the 68 patients enrolled in the prospective study with DSA-confirmed TCVI were found to have a total of 19 traumatic aneurysms. An additional 6 patients, included in this study but discovered outside the prospective study, harbored 7 traumatic aneurysms for a total of 13 patients with 26 traumatic aneurysms. No patient with a known traumatic aneurysm was excluded from the study. Three (23%) of these 13 patients were male. The average age at presentation was 32.4 years (range 16–56 years). A motor vehicle collision was the mechanism of injury in 12 (92.3%) of these 13 patients, and pedestrian versus motor vehicle was the mechanism of injury in the remaining patient (7.7%). Indications for screening CTA in the 13 patients found to have traumatic aneurysms included closed head injury in 8, facial fractures in 7, cervical spine fracture in 5, skull fracture in 4, and a seat belt sign in 1. The patient in Case 9 did not undergo screening CTA; she was found to have sustained a TCVI during evaluation after she suffered an ischemic stroke. Nine (69.2%) of the 13 patients presented with 2 or more known risk factors for TCVI. One patient had a Horner’s syndrome associated with the injured artery; no patient with a traumatic aneurysm had symptoms caused by mass effect by the aneurysm or bleeding from the aneurysm. Patient characteristics are presented in Table 1.

Imaging Findings at Initial Diagnosis and at Follow-Up

The right cervical ICA was involved in 13 (50%) of
Traumatic aneurysms and cerebrovascular injury

TABLE 1: Patient and aneurysm characteristics*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>no. of patients w/ traumatic aneurysms</td>
<td>13</td>
</tr>
<tr>
<td>age in yrs</td>
<td>mean 32.4, range 16–56</td>
</tr>
<tr>
<td>no. of males†</td>
<td>3 (23)</td>
</tr>
<tr>
<td>clinical follow-up in mos</td>
<td>mean 15.8, range 0.4–41.7</td>
</tr>
<tr>
<td>mechanism of injury†</td>
<td>MVC 12 (92.3), pedestrian vs motor vehicle 1 (7.7)</td>
</tr>
<tr>
<td>closed head injury</td>
<td>8 (61.5)</td>
</tr>
<tr>
<td>facial fracture</td>
<td>7 (53.8)</td>
</tr>
<tr>
<td>cervical spine fracture</td>
<td>5 (38.5)</td>
</tr>
<tr>
<td>skull fracture</td>
<td>4 (30.8)</td>
</tr>
<tr>
<td>seat belt sign</td>
<td>1 (7.7)</td>
</tr>
<tr>
<td>multiple risk factors</td>
<td>9 (69.2)</td>
</tr>
<tr>
<td>no. of traumatic aneurysms</td>
<td>26</td>
</tr>
<tr>
<td>rt cervical ICA (C1)</td>
<td>13 (50)</td>
</tr>
<tr>
<td>lt cervical ICA (C1)</td>
<td>12 (46.2)</td>
</tr>
<tr>
<td>lt VA (V3)</td>
<td>1 (3.8)</td>
</tr>
<tr>
<td>no. of saccular aneurysms</td>
<td>9</td>
</tr>
<tr>
<td>mean size at presentation in mm</td>
<td>mean 6.8, range 2–14</td>
</tr>
<tr>
<td>no. of fusiform aneurysms</td>
<td>17</td>
</tr>
<tr>
<td>mean size at presentation in mm</td>
<td>mean 2.1/7.3, range 1–3/2–16</td>
</tr>
<tr>
<td>radiographic follow-up in mos</td>
<td>mean 22.0, range 6.6–55.7</td>
</tr>
<tr>
<td>ischemic complications†</td>
<td>total no. w/ ischemic strokes 3 (23.1), no. w/ ischemic strokes associated w/ a traumatic aneurysm 2 (15.4)</td>
</tr>
<tr>
<td>treatment†</td>
<td>antiplatelet medication 13 (100), endovascular stenting 2 (15.4)</td>
</tr>
</tbody>
</table>

* MVC = motor vehicle collision.
† Values are presented as the number of patients (%).
‡ Values are presented as the number of aneurysms (%).

26 aneurysms, the left cervical CA in 12 (46.2%), and the left VA (V3 segment) in 1 aneurysm (3.8%). Six (46.2%) of the 13 patients harbored more than 1 traumatic aneurysm, with 6 being the maximum number identified in any single patient. The average size at initial diagnosis by either CTA or DSA was 6.8 mm (range 2–14 mm) for the 9 saccular aneurysms and 2.1 mm by 7.3 mm (range 1–3 mm by 2–16 mm) for the 17 fusiform aneurysms. Fourteen (53.8%) of the 26 traumatic aneurysms were visualized on the initial CTA study, with an average size of 6.8 mm (range 2–14 mm) for saccular aneurysms and 2.4 mm by 10.6 mm (range 1–3 mm by 5–16 mm) for fusiform aneurysms. The remaining cases had evidence of TCVI on screening CTA or MRI without visualization of a traumatic aneurysm. Twenty-five (96.2%) of the 26 traumatic aneurysms were visualized on confirmatory DSA, with an average size of 8.9 mm (range 3–25 mm) for saccular aneurysms and 2.4 mm by 9.6 mm (range 1–6 mm by 2–28 mm) for fusiform aneurysms. A single traumatic aneurysm, measuring 11 mm, was identified 3.3 months after the initial injury and was discovered on follow-up CTA that was performed to evaluate a known TCVI (Case 9). Initial MRI and confirmatory DSA performed in this patient showed bilateral ICA dissections but did not show a traumatic aneurysm. The average length of radiographic follow-up was 22.0 months (range 6.6–55.7 months). Computed tomography angiography was performed for last radiographic follow-up in all but 1 patient who underwent DSA at the last radiographic follow-up (Case 10). Final follow-up imaging of the 9 saccular aneurysms showed that 1 (11.1%) was not visible, 4 (44.4%) were smaller, 1 (11.1%) was stable, and 3 (33%) aneurysms were larger (Table 2, Fig. 1). Final follow-up imaging of the 17 fusiform aneurysms revealed that 9 (52.9%) were not visible, 6 (35.3%) were smaller, and 2 (11.8%) were larger. Two (7.7%) of 26 traumatic aneurysms were treated with placement of a covered stent due to evidence of significant aneurysm growth (Cases 1 and 10). The average size at presentation for the 1 saccular aneurysm that was no longer visible, the 4 that were smaller, the 1 that was stable, and the 3 that were larger was 2 mm, 6.2 mm (range 4–11 mm), 6 mm, and 9.3 mm (range 3–14 mm), respectively (Table 2). The average size at presentation for the 9 fusiform aneurysms that were no longer visible, the 6 that were smaller, and the 2 that were larger was 1.7 mm by 5.3 mm (range 1–2 mm by 2–12 mm), 2.8 mm by 10 mm (2–3 mm by 5–16 mm), and 2 mm by 8 mm (range 2 mm by 7–9 mm), respectively (Table 2). Two of the 5 aneurysms that were larger on radiographic follow-up were treated with endovascular stenting; these aneurysms were saccular and measured 11 mm and 14 mm at presentation. The average size of the persisting 8 saccular aneurysms and 8 fusiform aneurysms was 7.6 mm (range 1–25 mm) and 2.25 mm by 9.9 mm (range 1–5 mm by 4–18 mm), respectively.

Outcomes (smaller, stable, larger, or not visualized on last follow-up imaging) of saccular traumatic aneurysms versus fusiform traumatic aneurysms were compared. Results tend to support the assertion that the 2 different aneurysm morphologies behave differently, but these results did not reach statistical significance (p = 0.07). When outcomes of larger versus smaller + stable + not visualized were evaluated with respect to aneurysm morphology, saccular aneurysms were 3.75 times more likely...
to enlarge on final follow-up imaging; this result was not statistically significant (OR 3.75 [95% CI 0.49–28.4], p = 0.3). When adjusting for aneurysm depth at initial diagnosis, saccular aneurysms were 1.08 times more likely to enlarge on last follow-up imaging; this result was not statistically significant (OR 1.08 [95% CI 0.05–21.5], p = 0.96) (Table 3).

Eight (80%) of the 10 aneurysms that were not visible on follow-up CTA were not visible on initial CTA and were only seen on subsequent DSA. All of these aneurysms were fusiform, and the average size was 1.8 mm by 4.6 mm (range 1–2 mm by 2–12 mm).

Eleven (84.6%) of the 13 patients underwent confirmatory DSA following their screening CTA. The screening CTA revealed 7 saccular aneurysms measuring 5.6 mm (range 2–14 mm) and 5 fusiform aneurysms measuring 2.4 mm by 10.6 mm (range 1–3 mm by 5–16 mm), and subsequent DSA revealed 7 saccular aneurysms measuring 7.9 mm (range 3–25 mm) and 17 fusiform aneurysms measuring 2.4 mm by 9.6 mm (range 1–6 mm by 2–28 mm). Timing of CTA and DSA was separated by an average of 3.4 days (range 0–12 days). The average size of the 12 fusiform aneurysms that were only visualized on DSA was 2 mm by 5.9 mm (range 1–3 mm by 2–12 mm).

Ischemic Events in Patients With TCVI and Traumatic Aneurysms

Three (23.1%) of 13 patients had ischemic strokes attributable to the TCVI. Two of these 3 patients (Cases 10 and 12) were found to have traumatic aneurysms on initial workup; the remaining patient (Case 9) developed a traumatic aneurysm that was discovered on follow-up CTA performed to evaluate known CA dissections. Thus, 2 (15.4%) of 13 patients experienced an ischemic stroke in the setting of TCVI with traumatic aneurysm formation. Two of the 3 patients (Cases 9 and 10) suffered their ischemic event prior to diagnosis of a TCVI and prior to the administration of aspirin. The time course of the remaining patient’s (Case 12) ischemic event in relation to the diagnosis of TCVI and the administration of aspirin is unclear. There were no deaths related to ischemic complications. The patients in Cases 10 and 12 underwent screening CTA and at least 1 DSA session. The patient in Case 9 underwent screening MRI followed by 2 DSA sessions. Radiographic findings were indicative of an embolic stroke mechanism in all patients.

Case 9. This 33-year-old woman presented after being struck by a motor vehicle. She was admitted without undergoing screening CTA because of a lack of identifiable risk factors for a TCVI. She experienced a neurological decline after admission manifested by altered mental status. Brain MRI revealed bilateral anterior circulation ischemic infarctions. A subsequent DSA study showed bilateral cervical ICA dissections. Initial DSA and CTA did not demonstrate a traumatic aneurysm; however, follow-up CTA performed 3.3 months after the insult showed a saccular 11-mm right cervical ICA traumatic aneurysm. On a follow-up CTA, 55.7 months after the initial trauma, the aneurysm had decreased to 5 mm in size. Following the diagnosis of TCVI and bilateral ischemic strokes, the patient’s initial event was treated with an abciximab infusion for 24 hours, followed by 325 mg aspirin daily and 75 mg clopidogrel daily. The clopidogrel was stopped 4 months after the event, and 325 mg aspirin daily was continued indefinitely. It is important to note that the patient’s ischemic event occurred prior to the diagnosis of TCVI.

**TABLE 2: Traumatic aneurysm appearance on last follow-up imaging***

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Healed or Not</th>
<th>Smaller</th>
<th>Stable</th>
<th>Larger</th>
</tr>
</thead>
<tbody>
<tr>
<td>total no. of traumatic aneurysms</td>
<td>10/26 (38.5)</td>
<td>10/26 (38.5)</td>
<td>1/26 (3.8)</td>
<td>5/26 (19.2)</td>
</tr>
<tr>
<td>saccular aneurysms</td>
<td>1/9 (11.1)</td>
<td>4/9 (44.4)</td>
<td>1/9 (11.1)</td>
<td>3/9 (33.3)</td>
</tr>
<tr>
<td>fusiform aneurysms</td>
<td>9/17 (52.9)</td>
<td>6/17 (35.3)</td>
<td>0</td>
<td>2/17 (11.8)</td>
</tr>
<tr>
<td>mean size of traumatic aneurysms at presentation in mm</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>saccular</td>
<td>2</td>
<td>6.2</td>
<td>6</td>
<td>9.3</td>
</tr>
<tr>
<td>fusiform (diameter, length)</td>
<td>1.7, 5.3</td>
<td>2.8, 10</td>
<td>2, 8</td>
<td></td>
</tr>
</tbody>
</table>

* Values are presented as the number of aneurysms (%) unless otherwise stated.
† Eight of the 9 fusiform aneurysms were not visualized on initial CTA and were only seen on subsequent DSA.

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**Fig. 1.** Traumatic aneurysm behavior. Each black line represents 1 aneurysm. The red lines represent multiple aneurysms with the number of aneurysms noted to the left of each line. The one marked x7 includes 6 fusiform aneurysms and 1 saccular aneurysm.
and prior to the administration of antiplatelet medication. She made a full recovery and was noted to be neurologically intact at her 4-month follow-up visit.

Case 10. This 28-year-old man was involved in a motor vehicle collision and was admitted with a Glasgow Coma Scale score of 14; he was found to be neurologically nonfocal on examination (Fig. 2). Screening CTA, performed because of facial fractures, cervical spine fractures, and subdural hematoma, revealed a right cervical ICA dissection, a saccular left cervical ICA traumatic aneurysm measuring 11 mm, and left-sided anterior circulation ischemic infarctions. Thus, this patient had suffered the ischemic event prior to the diagnosis of TCVI and the administration of aspirin. Follow-up CTA performed 33.4 months after diagnosis showed that the aneurysm had increased in size to 18 mm. The patient was started on 325 mg aspirin daily at initial diagnosis, and the traumatic aneurysm was treated via endovascular techniques with a covered stent 37.8 months after diagnosis due to significant enlargement of the aneurysm on follow-up imaging.

Case 12. This 56-year-old woman, who was involved in a motor vehicle collision, was neurologically intact on admission (Fig. 3). Screening CTA, performed because of facial and skull fractures, revealed a saccular right ICA traumatic aneurysm measuring 2 mm on screening CTA and 3 mm on confirmatory DSA. Magnetic resonance imaging was performed 12 days later for workup of right-sided ptosis and revealed bilateral anterior circulation ischemic infarctions. Given that the patient’s ischemic infarctions were clinically silent and likely not the cause of her right-sided ptosis, the time course of the ischemic infarctions in relation to diagnosis of TCVI and the administration of aspirin is uncertain. It is also important to note that the ischemic infarctions were discovered on MRI following DSA and thus could be a complication of DSA rather than sequelae of TCVI. Follow-up CTA performed 7.3 months after the trauma showed that the traumatic aneurysm was no longer visible. The patient was treated with 325 mg aspirin at diagnosis, and this was continued indefinitely. At last follow-up, 7.3 months after the initial event, the patient was noted to have a mild right-sided Horner’s syndrome but was otherwise neurologically intact.

Management of Patients With TCVI and Traumatic Aneurysms

All patients diagnosed with TCVI by either CTA or DSA were treated with antiplatelet medications. One patient (7.7%; Case 9) was treated with an abciximab infusion for 24 hours after the diagnosis of bilateral anterior circulation ischemic infarctions. She was then transitioned to 325 mg aspirin daily and 75 mg clopidogrel daily. The clopidogrel was discontinued 4 months after the event, and 325 mg aspirin was continued indefinitely. Two patients (15.4%; Cases 1 and 10) underwent endovascular treatment with a covered stent for treatment of their cervical carotid traumatic aneurysms. One of these patients (Case 10) experienced embolic phenomena related to his TCVI and was treated with 325 mg aspirin daily at diagnosis. Follow-up CTA performed 33.4 months after the event showed growth of the traumatic aneurysm from 11 mm, at diagnosis, to 18 mm. Because of concern for continued growth of the traumatic aneurysm in an otherwise healthy young patient, endovascular treatment was recommended. Covered stent deployment was done 37.8 months after diagnosis without complication; the traumatic aneurysm was 16 mm at the time of stenting. Endovascular treatment with a covered stent was also recommended for the other patient (Case 1); however, this patient was treated with stent deployment at another center, and details of his procedure are not known. The remaining 10 (76.9%) of 13 patients were treated only with 325 mg aspirin daily.

Clinical Outcomes of Patients With Traumatic Aneurysms

Patients underwent clinical follow-up for an average of 15.8 months (range 0.4–41.7 months) and imaging follow-up for an average of 22.0 months (range 6.6–55.7 months). There were no traumatic aneurysm ruptures, and no patient experienced a neurological event related to the TCVI after the initiation of antiplatelet therapy. One patient (Case 12) had a Horner’s syndrome that was present on initial diagnosis and was unchanged at last follow-up.

### TABLE 3: Statistical evaluation of fusiform and saccular aneurysms

<table>
<thead>
<tr>
<th>Parameter</th>
<th>No. of Aneurysms (%)</th>
<th>p Value*</th>
<th>OR (95% CI), p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fusiform (n = 17)</td>
<td>Saccular (n = 9)</td>
<td></td>
</tr>
<tr>
<td>change in morphology</td>
<td></td>
<td>0.07</td>
<td></td>
</tr>
<tr>
<td>larger</td>
<td>2 (11.8)</td>
<td>3 (33.3)</td>
<td></td>
</tr>
<tr>
<td>smaller</td>
<td>6 (35.3)</td>
<td>4 (44.4)</td>
<td></td>
</tr>
<tr>
<td>stable</td>
<td>0</td>
<td>1 (11.1)</td>
<td></td>
</tr>
<tr>
<td>not visualized</td>
<td>9 (52.9)</td>
<td>1 (11.1)</td>
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</tr>
<tr>
<td>grouped comparison</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>larger</td>
<td>2 (11.8)</td>
<td>3 (33.3)</td>
<td></td>
</tr>
<tr>
<td>smaller + stable + not visualized</td>
<td>15 (88.2)</td>
<td>6 (66.7)</td>
<td></td>
</tr>
</tbody>
</table>

* Fisher’s exact test.
† Adjusting for depth of aneurysm at initial diagnosis.
The remaining 2 patients who suffered ischemic events related to their TCVI (Cases 9 and 10) made full neurological recovery and remained asymptomatic.

Discussion

Findings of the Prospective Cohort

The prospective arm of our study revealed a 10.3% incidence of traumatic CA and VA aneurysms among patients with DSA-confirmed TCVI following blunt trauma. Previously reported rates of CA and VA traumatic aneurysm formation ranged from 13%—49%2,9,13,22 and 4%—8%,13,22 respectively. In this arm, a motor vehicle collision was the mechanism of injury in 100% of patients. The most common risk factor for a traumatic aneurysm formation was head injury (57.1%) followed by cervical spine fracture (42.9%). Multiple traumatic aneurysms were encountered in 71.4% of the cases; this study also suggests that the CA is more susceptible to traumatic aneurysm formation than the VA.

Saccular Versus Fusiform Traumatic Aneurysms

This series of 26 traumatic aneurysms comprised 9 saccular (34.6%) and 17 fusiform (65.4%) aneurysms. Two of the saccular aneurysms were found in patients who had suffered an ischemic stroke; a third patient with an ischemic stroke due to TCVI later developed a saccular aneurysm after the stroke. No ischemic strokes occurred in patients with fusiform traumatic aneurysms. Saccular aneurysms may carry a greater risk of thromboembolic stroke than fusiform aneurysms because of the potential for blood stasis and thrombosis within the aneurysm. Alternatively, saccular traumatic aneurysms may result from tearing of the arterial wall and exposure of subendothelial elements to circulating platelets and clotting factors. Fusiform traumatic aneurysms, in contrast, may result from stretching of the artery wall, rather than tearing.

Saccular aneurysms were also more likely to enlarge and were less likely to have resolved (or not to have been visualized) on last follow-up imaging when compared with fusiform aneurysms. In addition, both traumatic aneurysms that were treated with stenting due to significant enlargement were saccular aneurysms. For a given internal fluid pressure, wall tension is proportional to the radius of the vessel. Saccular aneurysms tend to have a greater depth, resulting in a larger arterial radius and thus greater wall tension than fusiform aneurysms. Therefore, saccular aneurysms may be more prone to enlargement than fusiform aneurysms. A greater tendency of saccular aneurysms to enlarge may also reflect greater damage to the arterial wall in comparison with fusiform aneurysms, which may occur due to stretching rather than tearing of the arterial wall. Moreover, 2 of 3 saccular aneurysms with an initial size larger than 6 mm were enlarged on final follow-up imaging, whereas only 1 of 6 saccular aneurysms that were 6 mm or smaller were enlarged, suggesting that there may be an initial aneurysm size threshold above which saccular aneurysms are more likely to enlarge over time. Despite not reaching statistical significance, there was a trend toward behavioral differences between saccular and fusiform aneurysms as evidenced by the Fisher exact test (p = 0.07), with saccular aneurysms tending to enlarge (OR 3.75 [95% CI 0.49–28.4], p = 0.3). This difference decreased when adjusting for aneurysm depth at diagnosis (OR 1.08 [95% CI 0.05–21.5], p = 0.3); thus, while saccular aneurysms are more likely to enlarge on follow-up imaging, they are also more likely to be larger on presentation. The increased growth rate is presumed to be a function of both the inherent tendency for saccular aneurysms to enlarge, as well as the tendency for saccular aneurysms to present with a greater depth. The small number of cases in this report precludes statistically significant conclusions; because of the relative rarity of blunt traumatic aneurysms, a more comprehensive study of traumatic aneurysms will likely require a multicenter effort.
Value of CTA for the Diagnosis and Surveillance Imaging of Traumatic Aneurysms

Digital subtraction angiography has obvious limitations in terms of time, cost, availability, and risk associated with the procedure. High-resolution multidetector CTA is now available at most trauma centers and is increasingly used for screening for TCVI. Studies assessing the accuracy of 16-section multidetector CTA compared with DSA in trauma patients at risk for TCVI found sensitivity, specificity, positive predictive, and negative predictive values of 74%–97.7%, 84%–100%, 63%–100%, and 90%–99.3%, respectively. The sensitivity of CTA is increased with greater severity of TCVI. The diagnostic accuracy of CTA, specifically for traumatic aneurysms, particularly small ones, is limited. In one study, CTA missed 13% of traumatic aneurysms. While studies utilizing 16-section multidetector CTA have found only rare false-positive or false-negative findings for traumatic aneurysms, a 2010 systematic analysis of the 64-section multidetector CTA found flow artifacts mimicking arterial dissection or occlusion in 14% of patients. In the present study, 11 patients harboring 24 aneurysms underwent screening CTA followed by confirmatory DSA. Only 12 of those 24 aneurysms were seen on CTA resulting in sensitivity for CTA of 50%; there were no false-positive findings for traumatic aneurysms. The average size of the fusiform aneurysms not seen on CTA but subsequently detected on DSA was 2 mm by 5.9 mm (range 1–3 mm by 2–12 mm). A possible explanation for the low sensitivity is the inability of CTA to reliably detect small, longitudinally oriented aneurysms. It should also be noted that TCVI is a dynamic disease process and imaging modalities separated in time are subject to bias due to injury progression and/or healing.

We use CTA (and not DSA) for routine follow-up surveillance imaging of traumatic extracranial aneurysms. The benefits of CTA in this setting, in terms of ease, cost, and patient safety, seem to offset the lack of accuracy in the detection of TCVI lesions compared with DSA. It is reasonable to assume that many TCVI lesions, after several months of treatment with an antithrombotic medication, will heal and that any persisting or new lesion that might develop by the time of the follow-up surveillance CTA, but be below the threshold for detection by CTA, would not be clinically significant.

Ischemic Stroke in Patients With Traumatic Aneurysms

In previous studies, extracranial traumatic CA and VA aneurysms have been found to be a risk factor for ischemic stroke due to distal thrombotic embolization and/or flow-limiting stenosis. The overall rate of ischemic stroke in the present series was 23.1%, with 15.4% of patients having an ischemic stroke. None of the patients had an ischemic stroke after initiation of antiplatelet therapy. The findings of the present study are similar to a previous study that reported a 26% and 27% rate of ischemic stroke for traumatic aneurysms of the CA and VA, respectively. It is noteworthy that no patient in the present study suffered a clinically evident ischemic stroke after the initiation of antiplatelet medication.

Other Clinical Manifestations of Traumatic Aneurysms

Aside from cerebral ischemia due to thromboembolism, extracranial CA and VA aneurysms can cause symptoms due to hemorrhage, Horner’s syndrome, or mass effect on adjacent structures. Rupture of extracranial traumatic aneurysms appears to be a rare event, and no aneurysm ruptures were seen in the present study. Akiyama and colleagues reported a case of a patient presenting with a ruptured 4-cm extracranial ICA traumatic aneurysm. The patient was treated successfully with endovascular stenting. Screening of patients with risk factors for TCVI and follow-up imaging in patients with TCVI may identify patients at risk for hemorrhage due to significantly enlarging traumatic aneurysms.

One patient in the present study (Case 12) had a persistent mild Horner’s syndrome related to her TCVI involving the ICA. Horner’s syndrome is reported in 7% of cases with blunt carotid injury and can also present following traumatic injury of the VA as a result of neura-
praxia of the cervical sympathetic chain rather than arterial injury itself.\textsuperscript{2,13,21} Aside from this case, we did not observe any other local complication related to traumatic aneurysm formation (such as pain, neurovascular compression, or cosmetic deformity).

Management of Patients With Traumatic Aneurysms

Optimal management of extracranial CA and VA traumatic aneurysms is not yet clear, and Class I medical evidence is lacking.\textsuperscript{15} A variety of management strategies for TCVI are in use and include anticoagulation, antiplatelet agents, and endovascular stenting.\textsuperscript{6} We favor antiplatelet management with 325 mg aspirin daily and serial imaging for most TCVI patients.

Endovascular treatment of TCVI lesions has become more common in recent years.\textsuperscript{7,13} However, because the natural history of traumatic aneurysms is so poorly understood, it is difficult to determine the net benefit to patients of endovascular treatment. We reserve endovascular treatment (usually deployment of a covered stent) of extracranial traumatic aneurysms to those lesions that are found to have significant enlargement on follow-up imaging (depth > 15 mm). The rationale for treating only aneurysms larger than 15 mm is that any significant further enlargement may make the aneurysm relatively difficult to treat without sacrifice of the parent artery. Another justification for restricting stenting to patients found to have enlargement on follow-up imaging is that these trauma patients are beyond the acute phase after the original injury, and they would be expected to better tolerate dual antiplatelet therapy. Furthermore, some traumatic aneurysms decrease in size or resolve over time, obviating the need for endovascular treatment.

Limitations

The principal limitation of this study is the relatively small number of cases. Blunt TCVI injuries only constitute approximately 1% of blunt trauma admissions, and traumatic aneurysms only occur in about 10% of TCVI cases, and TCVI is present in only about 1% of blunt trauma admissions. A more comprehensive study will require a multicenter effort. Another limitation of this study is that the traumatic aneurysms were primarily identified with screening CTA performed in patients with established risk factors for TCVI. Since approximately 20% of TCVIs occur in patients without risk factors for TCVI,\textsuperscript{5,11,22} it is likely that a number of clinically silent traumatic aneurysms were not identified in patients who did not undergo a screening CTA.

Conclusions

Traumatic aneurysms occur in approximately 10% of patients who have suffered nonpenetrating blunt cerebrovascular injury. While 23% of patients in this series suffered an ischemic infarction with 15.4% experiencing an ischemic event associated with a traumatic aneurysm, there were no definitive cases of ischemic stroke after the initiation of antiplatelet therapy. Additionally, only 2 patients underwent endovascular stenting for the treatment of enlarging traumatic aneurysms. Traumatic aneurysms, while a risk factor for ischemic complications, can be safely and effectively managed in the majority of cases with an antiplatelet regimen of 325 mg aspirin daily and serial imaging to assess for aneurysm growth.

Disclosure

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

Author contributions to the study and manuscript preparation include the following. Conception and design: Harrigan, Foreman, Griessenauer. Acquisition of data: Harrigan, Foreman, Griessenauer. Analysis and interpretation of data: Harrigan, Foreman, Griessenauer. Drafting the article: Foreman, Griessenauer. 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: Harrigan. Statistical analysis: Foreman, Falola. Administrative/technical/material support: Foreman, Griessenauer. Study supervision: Harrigan, Foreman.

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