Traumatic cerebrovascular injury: clinical characteristics and illustrative cases

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OBJECTIVE Traumatic cerebrovascular injury (TCVI) is a rare and serious complication of traumatic brain injury (TBI). Various forms of TCVIs have been reported, including occlusions, arteriovenous fistulas, pseudoaneurysms, and transections. They can present at a variety of intervals after TBI and may manifest as sudden episodes, progressive symptoms, and even delayed fatal events. The purpose of this study was to analyze cases of TCVI identified at a single institution and further explore types and characteristics of these complications of TBI in order to improve recognition and treatment of these injuries.

METHODS The authors performed a retrospective review of cases of TCVI identified at their institution between 2013 and 2016. A total of 5178 patients presented with TBI during this time period, and 42 of these patients qualified for a diagnosis of TCVI and had adequate medical and imaging records for analysis. Data from their cases were analyzed, and 3 illustrative cases are presented in detail.

RESULTS The most common type of TCVI was arteriovenous fistula (86.4%), followed by pseudoaneurysm (11.9%), occlusion (2.4%), and transection (2.4%). The mortality rate of patients with TCVI was 7.1%.

CONCLUSIONS The authors describe the clinical characteristics of patients with TCVI and provide data from a series of 42 cases. It is important to recognize TCVI in order to facilitate early diagnosis and treatment.

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KEYWORDS traumatic cerebrovascular injury; vascular injury; traumatic brain injury; penetrating brain injury

TRAUMATIC cerebrovascular injury (TCVI) is a cerebrovascular structural injury caused by a traumatic brain injury (TBI).¹⁴ TCVI is an uncommon complication of TBI and has been reported to occur in 0.8%–1.7% of cases.^{4,22,27,28} TCVIs have been reported in various forms, including occlusion from vasospasm, dissection, or thromboembolism; arteriovenous fistula (AVF); pseudoaneurysm; and transection of a vessel.^{3,4} The timing of TCVI with respect to the causal TBI may be early or late.²² Additionally, the clinical presentation of TCVI varies according to the pathology. Sudden onset of neurological deficits from cerebrovascular occlusions tends to occur in acute periods,¹¹ whereas signs and symptoms of AVFs are ongoing and progressive for several weeks after the initial injury.¹⁶ Pseudoaneurysms have been reported in various natural histories. Larson et al. reviewed cases of traumatic intracranial aneurysms and described the presentation as ranging from asymptomatic incidental findings to fatal massive epistaxis.¹⁵

The objective of the present study was to describe the

clinical characteristics of patients with TCVI. Toward that end, we present both descriptive analyses of a series of cases identified at our institution and details of 3 illustrative cases from that series.

Methods

Population Study

This study was a retrospective review of cases involving patients treated at Songklanagarind Hospital, a level I trauma center, between 2013 and 2016 with a diagnosis of TBI. The patients' medical records and neuroimaging studies were obtained from the hospital information system.

According to the clinical practice at our institute, TBI patients who underwent CT angiography (CTA) or cerebral angiography or both during the acute period had one or more of the following: neurological deficits that could not be clarified from findings on CT of the brain; a Glasgow Coma Scale (GCS) score of 3–8; thick, diffuse subarachnoid hemorrhage; severe, localized subarachnoid

ABBREVIATIONS AVF = arteriovenous fistula; CTA = CT angiography; GCS = Glasgow Coma Scale; GOS = Glasgow Outcome Scale; ICA = internal carotid artery; MCA = middle cerebral artery; PCA = posterior cerebral artery; PCoA = posterior communicating artery; TBI = traumatic brain injury; TCVI = traumatic cerebrovascular injury.

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hemorrhage; excessive bleeding from the mouth, nose, or ear(s); cervical spine fractures at the C1–3 levels; cervical fracture subluxations; or a penetrating mechanism of injury. After hospital discharge, patients who developed new neurological signs or symptoms, such as orbital bruit, delayed epistaxis, or pulsatile proptosis, required further vascular investigations during follow-up. In order to identify patients with TCVI for inclusion in the present study, we reviewed the electronic medical records of the hospital database for TBI patients from the date of injury to the last follow-up date or December 31, 2017.

In order to qualify for inclusion in the study, patients had to have developed a TCVI after a TBI. Fifty-eight patients were excluded because their medical records were inaccessible or the initial neuroimaging studies were unavailable. A total of 42 of 5178 patients with TBI developed a TCVI and had medical records and initial imaging studies available for review and were ultimately included in our analysis.

Following Bodanapally et al.⁴ and Biffi et al.,³ TCVI was defined as intradural arterial injury from a primary force including blunt or penetrating injury and categorized into 4 groups: stenosis/occlusion; transection; pseudoaneurysm; and AVF. Cases of injuries to extracranial and extradural vessels were excluded, as were cases of cerebral infarction from secondary insults, such as a brain hypoxia.

Data extracted from the medical records included demographic characteristics, mechanism of injury, type of intracranial injury, other associated injuries, treatment, and outcome. Additionally, severity of TBI was categorized as mild, moderate, or severe according to the patient's GCS score.¹²

Neuroimaging studies described the types of intracranial injury, such as skull fracture, epidural hematoma, subdural hematoma, and cerebral contusion. The surgical treatments, outcome at hospital discharge, and the last follow-up were reviewed. The study was performed with permission from the Ethics Committee of the Faculty of Medicine, Songklanagarind Hospital, Prince of Songkla University.

Statistical Analysis

Patient characteristics, mechanism of injury, intracranial injury, associated injury, and TCVI characteristics were analyzed. The results are presented as proportions and mean values (with standard deviations). The test results of associations between several factors and the TCVI characteristics were analyzed by the chi-square test and Fisher's exact test. A *p* value < 0.05 was considered to be statistically significant. The statistical analyses were performed with the R version 3.4.0 software (R Foundation).

Results

We found that TCVI occurred in 0.8% of the 5178 TBI patients treated at our institution during the study period, based on a median follow-up duration of 50 days (IQR 31–120 days). Table 1 shows the clinical characteristics of the 42 patients with TCVI. More than two-thirds were male, and the mean age was 33.0 ± 14.0 years (range 16–72 years). The most common mechanism of injury was

TABLE 1. Clinical characteristics of 42 patients with TCVI

Characteristic	Value
Age in yrs	
0–19	6 (14.3)
20–60	34 (81.0)
>60	2 (4.8)
Sex	
Male	32 (76.2)
Female	10 (23.8)
Mechanism of injury	
Motorcycle accident	29 (69.0)
Car accident	5 (11.9)
Fall from a height	1 (2.4)
Assault	1 (2.4)
Secondary blast injury	1 (2.4)
Shotgun injury	3 (7.1)
Object penetrating into head	1 (2.4)
Pedestrian	1 (2.4)
Severity	
GCS 13–15	26 (61.9)
GCS 9–12	2 (4.8)
GCS 3–8	14 (33.3)
Signs & symptoms	
Proptosis	28 (66.7)
Bruit	9 (21.4)
Epistaxis	4 (9.5)
Alteration of consciousness	4 (9.5)
Cranial nerve involvement	2 (4.8)
Incidental finding	1 (2.4)
Mean no. of accident symptoms × days (SD)	73.6 (119.6)
Pupillary reaction	
Reactive, both pupils	21 (50.0)
Unequal	17 (43.6)
Non-reactive, both pupils	4 (10.4)
Intracranial injury on admission date	
Basilar skull fracture	25 (59.5)
Cerebral contusion	12 (31.0)
Subdural hemorrhage	10 (23.8)
Subarachnoid hemorrhage	5 (11.9)
Epidural hematoma	4 (9.5)
Intraventricular hemorrhage	1 (2.4)
Midline shift on admission date	
<5 mm	35 (83.3)
≥5 mm	7 (16.7)
Basal cistern obliteration on admission date	5 (11.9)
Maxillofacial injury	14 (33.3)
Orbital injury	13 (31.0)

Data are n (%) unless otherwise indicated.

TABLE 2. Injury characteristics and treatment in 42 cases of TCVI

Characteristic	Value
Type of TCVI	
Arteriovenous fistula	35 (86.4)
Pseudoaneurysm	5 (11.9)
Occlusion	1 (2.4)
Transection	1 (2.4)
Vessel affected	
Internal carotid artery	35 (85.4)
Posterior cerebral artery	2 (4.8)
Posterior communicating artery	2 (4.8)
Pericallosal artery	1 (2.4)
Ophthalmic artery	1 (2.4)
Middle cerebral artery	1 (2.4)
Treatment	
Endovascular	38 (90.5)
Surgical	2 (4.8)
No definite treatment (unstable vital signs)	2 (4.8)
Mean duration of follow-up in mos (SD)	33.8 (33.2)
Outcome per GOS	
Death	3 (7.1)
Vegetative state	1 (2.4)
Severe disability	3 (7.1)
Moderate disability	20 (47.6)
Good recovery	15 (35.7)

Data are n (%) unless otherwise indicated.

road traffic injury (83.3%), especially motorcycle collision (69.0%), whereas penetrating injury occurred in only 11.9% of the cases. Most of the patients in the study population (61.9%) had mild head injuries, and the remaining patients had moderate to severe head injuries. The common signs and symptoms of TCVI were proptosis, audible bruit, and epistaxis. Proptosis and bruit were associated with fistulas, whereas epistaxis was associated with ruptured pseudoaneurysm ($p = 0.02$). The timing of clinical symptom development related to TCVI ranged from immediate to 505 days after impact. The most common intracranial injury before TCVI was basilar skull fracture; maxillofacial and orbital injuries were found, respectively, in 14% and 13% of the 42 patients with TCVI.

According to Bodanapally et al.⁴ and Biffl et al.,³ TCVIs are divided into 4 groups: stenosis/occlusion, pseudoaneurysm, AVF, and transection.²⁸ Characteristics of TCVI injuries are demonstrated in Table 2. In the present study, the two most common types of TCVI were AVF (86.4%) and pseudoaneurysm (11.9%). The injuries were located at the internal carotid artery (ICA; 85.4%), the posterior cerebral artery (PCA; 4.8%), the posterior communicating artery (PCoA; 2.4%), the pericallosal artery (2.4%), the ophthalmic artery (2.4%), and the middle cerebral artery (MCA; 2.4%).

Since the most common pathology of TCVI in the present study was AVF, endovascular intervention was the

FIG. 1. Case 1: Secondary blast injury followed by right ICA occlusion. **A:** Photograph showing penetration of wooden fragments into the right orbit at both medial and lateral sites. Blurring has been applied for privacy. **B:** Axial CT bone-window image showing wooden fragments in the right orbit. **C:** Axial CT angiogram showing the tip of a fragment through the orbital apex and compressing the right ICA. **D:** 3D reconstruction image showing reperfusion into the right ICA via the circle of Willis.

major management. However, surgical management could play a role in pseudoaneurysms, depending on the location and architecture of the aneurysm. Outcomes of TCVI varied in severity and included good recovery (35.7%), moderate disability (47.6%), severe disability (7.1%), vegetative state (2.4%), and death (7.1%), according to the Glasgow Outcome Scale (GOS) score.

Illustrative Cases

Case 1: ICA Occlusion From Secondary Blast Injury

A 26-year-old soldier was injured by an explosion while on duty. Wooden fragments penetrated the right orbit (Fig. 1A). CT scans of the patient's head demonstrated that the tip of one of these fragments had penetrated to the anterior clinoid process, causing a right ICA occlusion (Fig. 1B and C). The treatment plan was to restore blood flow to the supraclinoid part of the ICA, MCA, and PCA via the patent circle of Willis (Fig. 1D). Unfortunately, the patient developed unstable vital signs due to hemorrhage (with bleeding from the nose and ear) after the initial investigations. Although resuscitation and massive blood transfusion were performed, the patient had persistent hypotension and died 1 day later.

Case 2: MCA Transection With Contrast Extravasation

A 34-year-old male patient sustained a shotgun injury to the head. At the scene, he developed cardiac arrest and received cardiopulmonary resuscitation. After resuscita-

FIG. 2. Case 2: Shotgun injury followed by right MCA transection with contrast extravasation. **A:** 3D reconstruction of the skull showing a mosaic fracture of the bifrontal bone. **B:** Axial CT head bone-window image showing the bifrontal bone fracture and multiple pellets. **C:** Axial CTA head showing metallic pellets through the right MCA. **D:** Coronal CTA head showing contrast extravasation via transected MCA on the right side.

tion, his vital signs returned after arrival at the hospital but he had a GCS score of 3 with fixed dilated pupils. The head CT showed bilateral frontal bone fractures with retained multiple shotgun pellets in both frontal lobes (Fig. 2A and B). CTA showed that pellets had penetrated the right MCA, and contrast extravasation was observed (Fig. 2C and D). The patient developed hypotension and a second cardiac arrest. Unfortunately, this time he was unresponsive to cardiopulmonary resuscitation and died.

Case 3: Right PCA Pseudoaneurysm

A 17-year-old male patient was injured in a motorcycle collision. Upon initial examination, he had a GCS score of 15 and was bleeding from the right ear. A CT scan demonstrated subarachnoid hemorrhage at the suprasellar, interpeduncular, and ambient cistern with basilar skull fracture (Fig. 3A and B). He was admitted for close observation, but developed deterioration of consciousness within 2 hours after admission. A repeat CT brain revealed new right temporal contusion and subarachnoid hemorrhage with 6.1-mm thickness behind the dorsum sellae (Fig. 3C). Decompressive craniectomy was performed. The GOS score at hospital discharge was 2 (vegetative state). One month after discharge, the patient's surgical wound was tense, and a follow-up CT of the brain showed hydrocephalus and an enhanced oval mass placed in the third ventricle (Fig. 3D). The 3D reconstruction image of the intracranial vessels showed a bilobulated aneurysm rising from the right PCA that was believed to have developed as a result of the vessel

FIG. 3. Case 3: TBI from motorcycle collision followed by right PCA aneurysm. **A:** Axial CT showing diffuse subarachnoid hemorrhage at the basal cistern. **B:** Axial CT bone-window image showing right petrous bone fracture. **C:** Repeat CT showing new right temporal contusion with subarachnoid hemorrhage at the prepontine cistern. **D:** Follow-up CT showing hydrocephalus and an enhanced oval mass in the third ventricle. **E and F:** 3D reconstruction of CT angiogram (E, coronal view) of intracranial vessels and digital subtraction angiogram (F, sagittal view) showing a bilobulated right PCA aneurysm.

impacting the dorsum sellae (Fig. 3E and F). Endovascular coiling was successfully performed. As of the most recent follow-up, the patient remained in a vegetative state.

Discussion

Mechanical force loading on a vessel generates TCVI via the front of the head. Therefore, orbital, maxillofacial, and basilar skull fractures are common contact injuries. Characteristics of TCVI have been described in various types. In 1999, Biffi et al.³ reported a grading scale for blunt carotid arterial injury, distinguishing 5 injury grades: grade I, “luminal irregularity or dissection with < 25% luminal narrowing”; grade II, dissection or intramural hematoma with ≥ 25% luminal narrowing, intraluminal thrombus, or raised intimal flap”; grade III, pseudoaneurysm; grade IV, occlusion; and grade V, “transection with free extravasation.” Bodanapally et al.,⁴ in their 2015 article on arterial injuries after penetrating brain injury, described 4 types of intracranial arterial injury: traumatic intracranial aneurysm, dissection, occlusion, and carotid-cavernous fistulas. In this current study, we stratified the types of TCVI into 4 groups: stenosis/occlusion, pseudoaneurysm, AVF, and transection.²⁸

Cerebral angiography is the standard tool for detecting TCVI; however, this method is invasive and sometimes inappropriate in an acute setting. CTA was reported to be the appropriate initial diagnostic tool in an emergency setting because it is less time-consuming and less invasive.^{10,19} The sensitivity of CTA to detect intracerebral vascular injuries is 72.7% (95% CI 49.8%–89.3%),⁵ while the sensitivity of a CTA to detect carotid artery injury of the neck ranges from 98% to 100%. CTA has lower sensitivity to detect a TCVI because the intracranial arteries are noticeably smaller in caliber, and beam-hardening metallic artifacts impair resolution of intracranial vessels.^{2,10,19,20,24} However, screening for TCVI with CTA is accepted practice in traumatic patients before confirming a diagnosis from cerebral angiography.

TCVIs may be caused by both direct and indirect forces on a vascular surface and have various manifestations, which depend on the morphology of the lesion. Synchronized injuries of the artery and the adjacent vein may lead to a pathophysiological formation of an AVF.⁸ The pathogenesis of an occlusion is classified into 4 causes: embolism from the ICA, vasospasm, thrombus formation, and dissection.¹⁸ Additionally, a force that sharply cuts vessels results in complete transection with contrast extravasation. Pseudoaneurysms occur from a shearing force that deforms the wall of a vessel.¹⁷ A PCA aneurysm can occur when the vessel accelerates to the sharp edge of the tentorium cerebelli, dorsum sellae, or clivus.^{17,32} Moreover, if the pericallosal artery is located near the falx cerebri in the coronal plane, such an incident may lead to the formation of a pericallosal artery aneurysm.^{21,23} Additionally, a direct force from a penetrating injury such as a gunshot wound or secondary blast injury can strain the arterial wall and allow blood to leak through the wall and then be contained by the adventitia or surrounding perivascular soft tissue.¹⁷

The mortality in the present study was 7.1% of overall TCVI cases. However, Onda et al. reported a TCVI-related

death rate of 14.3%.²² Early detection and treatment are essential for optimal outcome. The two common treatment options for TCVI are surgery and endovascular coil embolization. Endovascular intervention is feasible in cases of AVF,^{9,25,30} and treatments of pseudoaneurysm have been discussed elsewhere.^{14,25,29} Based on expert opinion, the type of TCVI and anatomical considerations, such as size, location, and morphological features, determine which treatment is most appropriate for the individual patient. The treatment approach must be considered on a case-by-case basis because atypical locations and variable shapes are often encountered. Surgical clipping, repair, trapping, or excision and bypass of an aneurysm are considered to be surgical treatment options, whereas endovascular interventions such as coiling and endovascular occlusion are alternative treatments for distal branch or fusiform aneurysms.^{13,15,29}

In traumatic vascular occlusion or stenosis management, intravascular volume expansion/hemodilution therapies are mentioned in the literature, but the outcomes are poor due to secondary cerebral infarction.^{26,31} In our first illustrative case, a surgical approach to remove the wooden foreign bodies with proximal control preparation was the plan. Unfortunately, the patient's clinical status was unstable and he died. Early diagnosis and treatment of transection are crucial. CTA is a rapid and effective diagnostic test. The next step is either an endovascular approach or surgical repair.¹ Endovascular interventions may be preferable in cases of transection because surgical treatment is time-consuming.^{6,7,22}

We recognize that this study is limited by its retrospective design and that the number of TCVI cases might be underdetected because our institution did not routinely perform either cerebral angiography or CTA of the brain in all TBI patients. However, vascular imaging of all patients with TBI would not represent a typical real-world situation. In patients considered to be at high risk for vascular injuries, such as those with penetrating injury, severe head injury, or basilar skull fracture,^{4,15,17,28} cerebral angiography or CTA should be performed.

Conclusions

We propose types and characteristics of TCVIs. A TCVI is an uncommon complication of TBI, but it can be life-threatening. Early detection allows for prompt treatment and should help improve outcomes.

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Disclosures

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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Conception and design: Tunthanathip, Kaewborisutsakul. Acquisition of data: Tunthanathip. Analysis and interpretation of data: Tunthanathip. Drafting the article: Tunthanathip, Oearsakul, Sakarunchai, Kaewborisutsakul. Critically revising the article: Tunthanathip. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Tunthanathip. Statistical analysis: Tunthanathip. Administrative/technical/material support: Tunthanathip. Study supervision: Phuenpathom, Sae-Heng, Oearsakul.

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