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Traumatic Injury of Major Cerebral Venous Sinuses Associated with Traumatic Brain Injury or Head and Neck Trauma: Analysis of National Trauma Data Bank

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Abstract

Background—The natural history and epidemiological aspects of traumatic injury of major cerebral venous sinuses are not fully understood. We determined the prevalence of traumatic injury of major cerebral venous sinuses and impact on the outcome of patients with traumatic brain injury, and/or head and neck trauma.

Methods—All the patients who were admitted with traumatic brain injury or head and neck trauma were identified by ICD-9-CM codes from the National Trauma Data Bank (NTDB), using data files from 2009 to 2010. NTDB represents one of the largest trauma databases and contains data from over 900 trauma centers across the United States. Presence of thrombosis, intimal tear, or dissection (traumatic injury) of major cerebral venous sinuses was identified in these patients by using Abbreviated Injury Scale predot codes. Admission Glasgow Coma Scale (GCS) score, Injury Severity Score (ISS), In-hospital complications, and treatment outcome were compared between patients with and without traumatic injury of major cerebral venous sinuses.

Results—A total of 76 patients were identified with traumatic injury of major cerebral venous sinuses among 453,775 patients who had been admitted with head and neck trauma. The rate of penetrating injury was higher among patients with traumatic injury of major cerebral venous sinuses (11.8% versus 2.5%, $p = 0.0001$). The patients with traumatic injury of major cerebral venous sinuses had a significantly higher rate of intracranial hemorrhage in comparison to patients without traumatic injury of major cerebral venous sinuses. The odds of in-hospital mortality remained significantly higher for patients with traumatic injury of major cerebral venous sinuses after adjusting for age, gender, admission GCS score, ISS injury type, and presence of intracranial hemorrhage [odds ratio (OR): 6.929; 95% confidence interval (CI) 1.337–35.96; $p < 0.020$]. The odds of discharge to nursing home remained higher for patients with traumatic injury of major cerebral venous sinuses after adjusting for potential confounders (OR: 1.8401; 95% CI 1.18–2.85, $p < 0.0065$).

Conclusion—Although infrequent, traumatic injury of major cerebral venous sinuses in head and neck trauma is associated with higher rates of in-hospital mortality and discharge to a nursing home.

Keywords

Traumatic brain injury; head and neck trauma; cerebral venous sinuses; cerebral venous thrombosis; cerebral venous dissection

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Introduction

Traumatic brain injury (TBI) can cause cerebral venous thrombosis presumably due to direct injury to cerebral venous sinuses with consequent occlusion and thrombosis [1–6]. Blunt traumatic brain injuries cause traumatic injury of major cerebral venous sinuses by skull fractures extending to a dural sinus or jugular bulb [3,5,6], and half of these patients develop occlusive thrombosis. In penetrating TBI, the projectiles can directly traverse the dural sinuses including cavernous venous sinuses [7]. There are numerous reports of developing arteriovenous fistulas when cerebral venous sinus thrombosis is secondary to penetrating injuries [8–11]. Most of the data available are derived from multiple small single-center studies. In the 2010 review of literature, the authors discussed a total of 206 cases which were reported in almost 40 publications [3]. Additional single-center studies have also been published later which discussed patient clinical and neuroimaging data [2,5]. Small number of patients from single-center studies also lead to under-representation of various etiological and clinical factors in the TBI cohort limiting generalization of results [12,13]. There is also a paucity of data regarding outcomes of patients with TBI associated with traumatic injury of major cerebral venous sinuses [3].

In this study, we determined the prevalence of traumatic injury of major cerebral venous sinuses and associated hospital events, treatments, and outcomes in patients with head and neck trauma or TBI, using a nationally representative database.

Methods

Study population

We analyzed patients registered in the National Trauma Data Bank (NTDB) between January 1, 2009 and December 31, 2010. The details of the methodology and data elements ascertained in NTDB have been published previously [14,15]. Briefly, NTDB is the largest trauma database compiled by the American College of Surgeons in the United States. NTDB collects clinical, demographic, external causes, and outcome data using predefined instructions for data ascertainment and entry for approximately 3 million cases from over 900 registered U.S. trauma centers.

We identified all the patients who were admitted to hospitals with TBI or head and neck trauma using the International Classification of Diseases, ninth revision, clinical modification (ICD-9-CM) diagnosis codes for TBI (800.0–801.99, 803.0–804.99, or 850.0–854.19) and head and neck injury (802, 802.0–802.9, and 802.20–802.39), similar to the previously published studies [14–16]. We identified patients with thrombosis, intimal tear, or dissection of cerebral venous thrombosis using Abbreviated Injury Scale (AIS) predot codes as follows: 120,806, cavernous sinus thrombosis, intimal tear, dissection; 122,006, sigmoid sinus thrombosis, intimal tear, dissection; 122,204, sinus or major vein thrombosis, intimal tear, dissection; 122,406, superior longitudinal (sagittal) sinus thrombosis, intimal tear, dissection; and 122,606, transverse sinus thrombosis, intimal tear, dissection. Prehospital neurologic deterioration was defined as a decrease in GCS score of 2 or more points between GCS score ascertained by Emergency Medical Services (EMS) and GCS score obtained in the Emergency Department (ED).

Study variables

Variables including patient's demographics, and clinical variables including admission Injury Severity Score (ISS), Glasgow Coma Scale (GCS) score, type of head injury (blunt or penetrating), initial systolic blood pressure, and intracranial hemorrhage (subtypes) were analyzed. We also identified prehospital neurological deterioration which was defined as a decrease in GCS score of ≥2 points between GCS score ascertained by EMS and GCS score obtained in ED. We queried the data for in-hospital complications such as stroke (defined as an embolic, thrombotic, or hemorrhagic vascular accident or stroke with motor, sensory, or cognitive dysfunction that persists for 24 or more hours), myocardial infarction, pneumonia, and deep vein thrombosis. The presence of thrombolysis and endovascular procedures were identified by using ICD-9 procedure codes of (38.01, 38.02, 38.12, 38.31, 38.32, 38.41, 38.42, 38.62,38. 82, and 39.56–39.59) and (00.55, 00.61, 00.62, 39.50, 39.72, 39.74, and 39.90) for surgical and endovascular procedures, respectively. Main outcome measures included total hospital stay, in-hospital mortality, and intensive care unit (ICU) days for each case. Hospital outcome was quantified by identifying discharge to home, discharge to nursing facility, and in-hospital mortality.

Statistical analysis

We compared the demographic and clinical characteristics, the rate of in-hospital complications, ICU days, hospital length of stay, ventilator days, in-hospital mortality, and discharge destination between patients with traumatic injury of major cerebral venous sinuses and to those without such injuries. Chi-square test and *t* test were used for categorical data and for continuous data,

respectively, with a p value < 0.05 considered statistically significant.

Multivariate linear regression analyses were performed to determine the effect of traumatic injury of major cerebral venous sinuses on ICU days, hospital length of stay, and ventilator days after adjusting for age, gender, admission GCS score, and ISS. Similarly, multivariate logistic regression analyses were performed to identify the impact of traumatic injury of major cerebral venous sinuses on in-hospital mortality and discharge destination after adjusting for the confounding factors. We used the SPSS 24 software for statistical analysis.

Results

A total of 76 patients were identified with traumatic injury of major cerebral venous sinuses among 453,775 patients who were admitted with head and neck trauma or TBI; 453,699 patients without traumatic injury of major cerebral venous sinuses. The location of traumatic injury of cerebral venous sinuses was in cavernous (n = 3), sigmoid (n = 36), superior longitudinal (n = 9), transverse (n = 28), and other sinus/major vein (n = 7). The mean age for patients with traumatic injury of cerebral venous sinuses was lower than the mean age in years for patients without traumatic injury of cerebral venous sinuses [27.64 (95 % CI 22.28–32.81 vs. 37.59 (95 % CI 37.50–37.69); p = 0.007]. The patients with traumatic injury of cerebral venous sinuses had an overall significantly lower admission GCS score (9.23 vs. 12.94 p value < 0.0001).

The rate of penetrating injury was higher among patients with traumatic injury of major cerebral venous sinuses (11.8% vs. 2.5%, p = 0.0001). The patients with traumatic injury of major cerebral venous sinuses had significantly higher rates of intracranial hemorrhages in comparison to patients without cerebral venous thrombosis. The rates of all subtypes of intracranial hemorrhages were higher in patients with traumatic injury of major cerebral venous sinuses as follows: subdural 60.5% versus 10.9%; subarachnoid 55.3% versus 11.3%; epidural 13.2% versus 1.7%; and intracerebral 17.1% versus 3.68% (p-value < 0.0001). The rates of pneumonia and deep venous thrombosis were higher in patients with traumatic injury of major cerebral venous sinuses (15.8% vs. 3.5% p-value < 0.0001 and 9.2% vs. 1.1% p-value < 0.0001, respectively) and there was a trend towards higher rates of in-hospital strokes in patients with traumatic injury of major cerebral venous sinuses (1.3% vs. 0.3% p-value 0.067).

The mean length of ICU stay (9.83 vs. 5.17 days; p < 0.0001) and hospital stay (11.23 vs. 6.15 days; p < 0.001) was nearly two times higher in patients with traumatic injury of major cerebral venous sinuses (Table 1). The rate of in-hospital mortality was two-fold higher in patients with traumatic injury of major cerebral venous sinuses (16% vs. 6.5%, p = 0.0001). The rate of discharge to home was significantly lower in patients with traumatic injury of major cerebral venous sinuses(48% vs. 60.8%, p = 0.0001) and the rate of discharge to nursing facility was higher in these patients in comparison to patients without traumatic injury of major cerebral venous sinuses(36% vs. 22.5%, p < 0.0001). The mortality according to the location of the traumatic injury of major cerebral venous sinuses was as follows: cavernous 25%, sigmoid 25%, superior sagittal 16.7%, transverse 41.7%, and other sinus/major vein 0%.

The ICU stay and hospital length of stay were not significantly higher for patients with traumatic injury of major cerebral venous sinuses after adjusting for age, gender, admission GCS score, ISS injury type, and presence of intracranial hemorrhage. The odds of in-hospital mortality remained significantly higher for patients with traumatic injury of major cerebral venous sinuses after adjusting for age, gender, admission, GCS score, ISS injury type, and presence of intracranial hemorrhage [odds ratio (OR): 6.929; 95% confidence interval (CI) 1.337–35.96); p < 0.020]. The odds of discharge to nursing home remained higher for patients with traumatic injury of major cerebral venous sinuses after adjusting for age, gender, admission GCS score, ISS injury type, and presence of intracranial hemorrhage (OR: 1.8401; 95 % CI 1.18–2.85; p < 0.0065).

Discussion

We observed that 76 of 453,775 patients had traumatic injury of major cerebral venous sinuses in the NTDB. A higher prevalence of penetrating injuries was observed in patients with traumatic injury of major cerebral venous sinuses. Vascular injury, particularly in arteries, is a well-recognized but infrequent event with penetrating [17–20]. The injury probably occurs due to penetration of major cerebral venous sinuses with foreign body and bone fragments and distortion and displacement due to mechanical forces initiated within the cranium. A higher prevalence of intracranial hemorrhages, particularly subarachnoid and subdural hemorrhages, were observed in patients with traumatic injury of major cerebral venous sinuses. Previous studies have also recognized the relationship between intracranial hemorrhages, lower GCS scores, and traumatic injury of major cerebral venous sinuses [6]. There is a possibility that subarachnoid and subdural hemorrhages are concurrent

Table 1. Baseline characteristics and clinical characteristics and outcomes of patients with and without traumatic injury of major cerebral venous sinuses among patients with traumatic brain injury, head and neck trauma: Analysis of National Trauma Data Bank 2009–2010

Variables	Patients with traumatic injury of major cerebral venous sinuses (*N* = 76)	Patients without traumatic injury of major cerebral venous sinuses (*N* = 453,699)	*P* value
Age (mean, 95% CI)	27.6 (22.8–32.8)	37.5 (37.5–37.6)	0.007
Gender			0.954
Men	50 (65.8%)	300,050 (66.1%)	
Women	26 (34.2%)	153,431 (33.8%)	
EMS arrival time (min)	41.59 (26.59–56.61)	40.15 (39.56–40.75)	0.951
ED arrival time (min)	163.92 (135.41–192.43)	288.20 (283.03–293.38)	0.543
Admission systolic blood pressure	122.68 (113.64–131.73)	131.44 (131.33–131.56)	
Admission ISSAIS (mean, 95% CI)	26.4 (24.0–28.9)	13.2 (13.1–13.2)	0.514
Type of Injury			
Blunt	59 (77.6%)	422,178 (93%)	<0.0001
Penetrating	9 (11.8%)	11,394 (2.5%)	<0.0001
Other	8 (10.5%)	20,127 (4.4%)	<0.0001
Admission GCS Score	9.23 (7.94–10.52)	12.94 (12.93–12.96)	<0.0001
Mild (GCS > 12)	34 (44%)	347,070 (76%)	
Moderate (9–12)	3 (3.9%)	19,441 (4.2%)	
Severe (GCS < 9)	36 (47%)	65,322 (14%)	
Prehospital neurological deterioration	9 (11.8%)	22,242 (4.9%)	0.005
Comorbidities			
History of cerebrovascular disease	3 (3.9%)	9444 (2%)	0.255
History of myocardial infarction	0 (0%)	5713 (1.25%)	0.325
Diabetes mellitus	2 (2.6%)	354,780 (7.8%)	0.092
History of hypertension	6 (7.9%)	89,759 (19.78%)	0.009
History of alcohol abuse	4 (5.3%)	40,015 (8.8%)	0.274
Intracranial hemorrhage			
Subdural	46 (60.5%)	49,380 (10.88%)	<0.0001
Subarachnoid	42 (55.3%)	51,197 (11.3%)	<0.0001
Epidural	10 (13.2%)	7934 (1.7%)	<0.0001
Intracerebral	13 (17.1%)	16,722 (3.68%)	<0.0001
Type of Facility			
Community/nonteaching	15 (19.7%)	208,187 (45.9%)	<0.0001
University	59 (77.6%)	227,483 (50.1%)	<0.0001
In-hospital complications			
Pneumonia	12 (15.8%)	16,055 (3.5%)	<0.0001
Stroke	1 (1.3%)	1158 (0.3%)	0.067
ARDS	3 (3.9%)	8469 (1.9%)	0.180
Acute kidney injury	0 (0%)	3116 (0.7%)	0.468
Deep vein thrombosis	7 (9.2%)	5083 (1.1%)	<0.0001
Pulmonary embolism	0 (0%)	1494 (0.3%)	0.616
Cerebral edema	0 (0%)	1108 (0.24%)	0.451
Cerebral compression (herniation)	0 (0%)	488 (0.10%)	NA
Raised intracranial pressure	0 (0%)	1 (0.0002%)	0.986
Procedures			
Surgical procedures			
Decompression of skull fracture craniotomy	4 (5.2%)	5090 (1.1%)	0.105
Lobectomy	1 (1.3%)	369 (0.08%)	0.304
Hemispherectomy	0 (0%)	7 (0.001%)	0.932
Lobotomy	0 (0%)	27 (0.005%)	0.867
Thrombolysis	0 (0%)	116 (0.025%)	7.26

Abbreviations used: *CI* = confidence interval; *GCS* = Glasgow Coma Scale; *ISSAIS* = Injury Severity Score, Abbreviated Injury Scale; *EMS* = Emergency Medical Services; *ED* = Emergency Department; min = minutes; ARDS = acute respiratory distress syndrome.

sequelae of initial trauma that resulted in the injury of major cerebral venous sinuses. An alternate explanation is that tearing of one or more bridging veins (cerebral cortex to dura or venous sinuses) occurs along with injury of major cerebral venous sinuses that initiates subdural hemorrhage. The cerebral venous pressure increases due to concurrent thrombosis or mechanical occlusion of sinuses leads to increased outflow resistance in the intact bridging veins and continued tearing and bleeding from bridging veins [21,22]. An increased vulnerability of bridging veins in subdural space is thought to be due to vein thickness ranging from 10 to 600 μm [23]. Although, the bridging veins continue into the subarachnoid space within arachnoid trabeculae, the vein thickness ranging from 50 to 200 μm with denser collagen fibers arranged in a circumferential manner within the subarachnoid space providing more support. However, bridging veins in subarachnoid space may also be vulnerable to increased outflow resistance in addition to cortical veins [22].

In-hospital mortality remained significantly higher among those with traumatic injury of major cerebral venous sinuses even after adjusting for injury type, GCS score strata, and presence of intracranial hemorrhages. Similarly, the rate of discharge to nursing home was

Table 2. The results of multivariate analyses determining the effect traumatic injury of major cerebral venous sinuses in patients with traumatic brain injury and head and neck trauma: Analysis of National Trauma Data Bank 2009–2010

Outcome	Patients with traumatic injury of major cerebral venous sinuses (*N* = 76)	Patients without traumatic injury of major cerebral venous sinuses (*N* = 453,699)	*P* value (univariate analysis)	OR (95% CI)*	*P* value
ICU days (mean, 95% CI)	9.8 (7.0–12.6)	5.1 (5.1–5.2)	<0.0001	0.9 (0.9–1.0)	0.121
Length of hospital stay (mean, 95% CI)	14.3 (10.9–17.6)	5.7 (5.7–5.8)	<0.0001	1.0 (0.97–1.0)	0.715
Ventilator days (mean, 95% CI)	11.2 (7.4–15.0)	6.1 (6.0–6.2)	0.10	1.0 (0.9–1.0)	0.446
Discharge to home	36 (48.0%)	279,593 (60.8%)	<0.0001	1.9 (0.5–7.3)	0.312
Discharge to nursing facility	27 (36.0%)	88,751 (22.5%)	<0.0001	1.8 (1.1–2.8)	<0.0065
In-hospital mortality	12 (16.0%)	25,816 (6.5%)	<0.0001	6.9 (1.3–35.9)	<0.020

*Adjusted for age, gender, ISSAIS, GCS score, injury type, and presence of intracranial hemorrhages

Abbreviations used: *CI* = confidence interval; *OR* = odds ratio; *ICU*= intensive care unit; *GCS* = Glasgow Coma Scale; *ISSAIS* = Injury Severity Score, Abbreviated Injury Scale.

high among patients with traumatic injury of major cerebral venous sinuses. Patients with traumatic injury of major cerebral venous sinuses had higher rates of pre hospital neurological deterioration and lower GCS scores at admission suggesting early neurological injury. The mechanism by which traumatic injury of major cerebral venous sinuses increases in-hospital mortality is presumably related to occlusive thrombosis with subsequent increase in intracranial pressure, cerebral edema, venous infarction, and/or intraparenchymal hemorrhages. Patients with traumatic injury of major cerebral venous sinuses may also have a higher risk subdural and subarachnoid hemorrhages as mentioned above. There appeared to be a trend towards higher rates of in-hospital strokes in patients with traumatic injury of major cerebral venous sinuses. Whether some of these strokes represented venous infarction could not be identified within our analysis [1]. Parenteral anticoagulation remains the first line of treatment in patients with nontraumatic cerebral venous thrombosis [24,25]. The rates of death or dependency in patients with nontraumatic cerebral venous thrombosis appear to be low [25]. The higher rates of or dependency in patients with cerebral venous thrombosis associated with TBI may be attributable to concurrent intracranial hemorrhages and parenchymal injury (contusions and axonal injury). An additional component may be attributable to high rates of intracranial adverse effects with parenteral anticoagulation in the setting of TBI, which may be required in the setting of cerebral venous thrombosis [26,27]. We also found that patients with injury to transverse venous sinuses appear to have higher mortality although the numbers available for comparative analysis were small.

In the current analysis, we identified TBI and head and neck trauma patients using primary ICD-9-CM diagnostic codes in hospitals records. ICD-9-CM diagnostic codes are frequently used in epidemiological studies of TBI [28,29]. A previous study reported a sensitivity of 89% for the presence of severe TBI, and sensitivity/specificity were > 80% for any skull fracture and intracranial hemorrhage [30]. In general, specificity appeared to be higher for most codes used to identify TBI and head and neck trauma patients. However, using ICD-9-CM diagnostic codes may underestimate mild TBI and those seen in the outpatient setting [30]. We identified traumatic injury of major cerebral venous sinuses using AIS predot codes that reflect the patient's injuries using six digits. The AIS was developed by the Association for the Advancement of Automotive Medicine [31,32]. The predot codes were designed to code various types of injury based on a standard dictionary. Each entry in a trauma registry dataset is assigned a six-digit AIS code number. One section of six-digit AIS code numbers is allocated to head trauma, which is subdivided into the whole area (massive destruction of cranium and brain, penetrating injury and scalp injury), intracranial vessels, cranial nerves (cranial nerves I to XII), internal organs, and skeletal. The six-digit AIS code numbers have been used in previous TBI studies [33].

We acknowledge that the current estimates of traumatic injury of major cerebral venous sinuses may be an underestimate of actual prevalence due to variations in imaging protocols. The AIS code numbers are more likely be assigned if the clinically significant event occurred. We analyzed outcome based on the discharge destination. A previous study had demonstrated that discharge destination correlated to death or disability defined by the modified Rankin scale at 3 and 12 months in post stroke survivors [34]. Based on the nature and limitation of the current study, we are unable to differentiate whether the poor outcome of these patients results from the actual injury or thrombosis of the cerebral venous sinus or due to the severity of initial injury to the brain. We believe that this may be a combi-

nation of both. The high rate of in-hospital mortality highlights the need for more in-depth studies in TBI and head and neck trauma patients focusing on the traumatic injury of major cerebral venous sinuses. Of particular interest is the question of whether in-hospital mortality can be reduced by early diagnosis and more focused therapeutic approaches such as endovascular interventional procedures.

Currently, there are limited data regarding endovascular approaches in such scenarios [1,8–11]. Studies that identify high-risk patients who could benefit from such approaches and optimal timings of such interventions could also be useful as an initial step to reduce the current gap in our knowledge.

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