Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Traumatic cerebral venous sinus thrombosis is uniformly common across skull fracture patterns in blunt head trauma

James E Wiseman

james.wiseman@som.umaryland.edu

University of Maryland Baltimore https://orcid.org/0000-0003-0732-3374

Tyler L Holliday

University of Maryland Baltimore

Bethany L Strong

University of Maryland Baltimore

Jeremy S Rubin

University of Maryland

Gary T Schwartzbauer

University of Maryland Baltimore

Thomas M Scalea

University of Maryland Baltimore

Deborah M Stein

University of Maryland Baltimore

Research Article

Keywords: CVST, TBI, Skull fractures, traumatic brain injury, venous sinus injury

Posted Date: March 24th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-9097240/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Abstract

Background

Traumatic cerebral venous sinus thrombosis (tCVST) is a recognized complication of traumatic brain injury (TBI) with important clinical implications. Although temporal bone fractures are associated with tCVST, the contribution of other skull fracture patterns remains unclear. We hypothesized that all skull fractures confer risk of tCVST and sought to quantify this risk across different fracture patterns.

Methods

This was a retrospective review of adult blunt-mechanism TBI with ≥ 1 skull fracture admitted to a Level 1 trauma center over an 11-year period. The primary outcome was tCVST. Patients who did not undergo venous-phase imaging were considered indeterminate and excluded. Candidate predictors were identified via univariable analysis; multivariable regression was used to estimate independent predictors of tCVST.

Results

Among 673 eligible patients, 428 had definitive imaging; 147 had tCVST and 281 were confirmed tCVST-negative. tCVST patients were younger (40.3 vs 45.7 years, $p = 0.006$) and had higher Injury Severity Scores (22.7 vs 19.8, $p = 0.004$). tCVST prevalence increased with head AIS (0% in AIS 2, 19.6% in AIS 3, 42.1% in AIS 4, and 44.4% in AIS 5; $p < 0.001$). Temporal fractures were associated with tCVST (42.0% vs. 24.2%, *p < 0.001)*, as was fracture burden (1 region = 25.9%, 5 regions = 66.7%, *p = 0.002).* Isolated temporal (33.3%), occipital (27.3%), frontal (23.5%), and parietal (16.7%) fractures also increased risk. On multivariable analysis, independent predictors of tCVST included head AIS (OR 4.48, 95% CI 1.41–14.26), temporal fracture (OR 8.0, 95% CI 2.14–29.99), and male sex (OR 6.45, 95% CI 1.1–37.3).

Conclusions

tCVST occurred at high rates across all fracture patterns, with no reliably low-risk subgroup. These findings suggest that routine venous-phase imaging should be considered in adults with moderate or severe TBI and any skull fracture, rather than selective imaging based on fracture location alone.

Level of Evidence/Study Type: Level IV, Diagnostic test/criteria

Introduction

Traumatic cerebral venous sinus thrombosis (tCVST) is an increasingly recognized complication of traumatic brain injury (TBI).[1,2] This condition involves the formation of a blood clot within the dural venous sinuses, the confluences of the brain's cortical veins that provide the primary cerebral blood outflow. The resultant obstruction can lead to worsening cerebral edema, exacerbate intracranial hypertension, and contribute to secondary injury or herniation. In non-trauma populations, CVST

accounts for approximately 0.5% – 3.0% of all strokes,[3,4] and emerging data suggest substantial long-term functional sequelae in survivors.

An association between tCVST and skull fractures is well-described.[5–8] The precise nature of this relationship, however, is unclear. It is unknown whether tCVST and skull fractures simply occur concurrently due to shared mechanisms, or if specific fracture patterns exert causal effects that predispose to sinus thrombosis. Prior work has emphasized temporal and occipital bone fractures, particularly with basilar extension, whereas the contributions of frontal and parietal bone fractures, or more complex multiregional fracture patterns are less well understood.

Conventional non-contrasted head computed tomography (CT) is insufficient to exclude tCVST.[9,10] CT angiography without venous-phase imaging has an estimated sensitivity below 80%. No existing guidelines currently define which TBI patients with skull fractures warrant dedicated venous-phase imaging. In practice, clinicians must decide whether fracture characteristics or other markers of injury severity can identify patients at sufficiently low risk to safely defer venous imaging. We therefore sought to determine whether specific skull fracture patterns or injury characteristics identify a subgroup with low probability of tCVST that might justify selective screening strategies. We hypothesized that tCVST prevalence is elevated across all blunt TBI patients with skull fractures, and that markers of brain injury severity can guide screening practices.

Methods

All adult (age ≥ 18 years) presenting to a single Level I trauma center between January 1, 2012, and December 31, 2022 with blunt head trauma and at least one skull fracture were identified. Demographic, clinical, and radiographic data were abstracted through structured chart review. The STrengthening the Reporting of Observational studies in Epidemiology (STROBE) guideline was used to ensure proper reporting of methods, results, and discussion. The protocol was reviewed by the local Institutional Review Board and was determined to be exempt under 45 CFR 46.104(d).

Identification of tCVST

The primary outcome was presence of tCVST. Thrombosis was classified as present if radiographic evidence of thrombosis was reported on computed tomography angiography (CTA) or venography (CTV), or magnetic resonance angiography (MRA) or venography (MRV). Because CTA is not a definitive test for ruling out tCVST, tCVST-negative status required a negative CTV or MRV, whereas CVST-positive status required explicit radiologist-documented thrombosis on any vascular imaging modality. tCVST-negative patients were excluded from the final analysis if they did not have confirmatory venous-phase imaging. All studies were interpreted contemporaneously by board-certified radiologists with trauma imaging expertise.

To minimize misclassification bias, patients were considered definitively negative for tCVST only if they underwent venous-phase imaging (CTV or MRV) that demonstrated no venous thrombosis. Patients who did not undergo CTV or MRV were classified as indeterminate and excluded from analyses evaluating associations with tCVST. To assess whether this exclusion introduced bias, key injury characteristics were compared between verified and indeterminate patients.

Skull Fracture Characterization

To examine whether skull-fracture combinations influenced tCVST risk, fractures were grouped into five anatomic regions: temporal, parietal, occipital, frontal, and skull base. Each region was coded as present or absent. Fracture complexity (overall fracture burden) was defined as the total number of distinct fractured regions per patient. Specific combinations with biological plausibility were coded as binary variables and carried forward into statistical analysis and regression model development.

Statistical Analysis

Continuous variables were non-normally distributed by Shapiro-Wilk and skewness-kurtosis testing and are presented as mean ± standard deviation or median (interquartile range) as appropriate. Group comparisons used the Mann-Whitney U-tests for continuous variables and chi-square or Fisher's exact tests for categorical variables.

Head AIS was treated as an ordinal variable for trend testing and as a continuous predictor in regression models after assessing for non-linearity. The relationship between increasing head AIS scores and the presence of tCVST was evaluated using Cuzick's nonparametric test for trend. Fracture complexity (fractured region count) was evaluated first in a univariable model, and then in a multivariable model adjusting for age, sex, Glasgow Coma Scale (GCS) category, intracranial hematoma type and thickness, Injury Severity Score (ISS), and head Abbreviated Injury Severity (AIS) score.

Univariable logistic regression was applied to screen for associations between demographic factors, injury severity markers, fracture patterns, presence and size of intracranial hematomas, blunt cerebrovascular injury (BCVI) and tCVST. Continuous predictors were modeled per-unit increase. Binary and categorical variables used absence of the characteristic as the reference category. Hematoma size variables were modeled only among patients with that hematoma type. Odds ratio (OR) with 95% confidence intervals (CIs) were reported.

Causal Framework and Covariate Selection

A directed acyclic graph (DAG) based on established TBI pathophysiology guided variable selection (Fig. 1). The DAG indicated that age and mechanism of injury are primary determinants of skull fracture patterns and downstream injury severity. Sex was retained due to known sex-related differences in TBI outcomes. Variables such as epidural hematoma (EDH) size, degree of midline shift, GCS, and head AIS

were positioned as mediators on the causal pathway from skull fracture to tCVST and were therefore intentionally excluded from the adjustment set in association models to avoid overadjustment bias. BCVI was evaluated as a clinically-available marker that may help identify patients at risk for tCVST in a screening context, rather than as a presumed mechanistic cause of venous thrombosis. On the basis of the DAG, only age and mechanism constituted the sufficient adjustment set, with sex included a priori on clinical grounds.

In parallel with association analyses, we developed a predictive model to identify patients at elevated risk for tCVST. Predictors with a p-value < 0.20 from univariate analyses, those with biological plausibility, or those previously linked to venous sinus injury were included in a least absolute shrinkage and selection operator (LASSO) penalized logistic regression model for final multivariable regression model development. Penalization parameters for the LASSO model were selected using cross-validation to minimize prediction error. Continuous predictors were evaluated on their native scales and assessed for collinearity prior to model fitting. Variables retained by the LASSO procedure were subsequently entered into a standard multivariable logistic regression model to obtain unbiased coefficient estimates and corresponding confidence intervals. The number of candidate predictors was restricted relative to the number of observed tCVST events to reduce the risk of model overfitting. Model discrimination was assessed using the area under the receiver operating characteristic curve (AUC), and calibration was evaluated using the Hosmer-Lemeshow test. The DAG informed causal adjustment for association analyses, whereas the LASSO procedure was used separately to develop a predictive model for identifying patients at increased risk of tCVST. All analyses were performed using Stata/SE 19.5 (StataCorp LLC, College Station, TX). Statistical significance was defined as $p < 0.05$.

Artificial Intelligence Declaration

ChatGPT5.0 (OpenAI, San Francisco, California) was consulted during final manuscript review to assist with proofreading and to improve manuscript readability. Any such use was fully aligned with the Committee on Publication Ethics[11] and International Committee of Medical Journal Editors[12] recommendations regarding acceptable use in scholarly publication.

Results

Overall Cohort

A total of 673 blunt trauma patients with skull fractures were identified. tCVST was diagnosed in 147 (21.8%). Of these, 115 (78.2%) had tCVST identified by dedicated venous-phase imaging (CTV), and 32 (21.8%) were diagnosed by CTA alone. All tCVST diagnoses were made using CT-based imaging; no cases were diagnosed on the basis of MRV. Among the 526 patients without tCVST, a total of 281 (53.4%) underwent venous-phase imaging (276 CTV and 5 MRV), while 245 patients (46.6%) had CTA only without confirmatory study. These 245 patients were considered indeterminate and excluded from

subsequent risk analyses. The final cohort therefore consisted of 428 patients with confirmed tCVST status, including 147 tCVST-positive (34.2%) and 281 tCVST-negative (65.8%) individuals. (Fig. 2)

Baseline Characteristics

Patients with tCVST were younger than tCVST-negative patients (Table 1). Sex distribution was similar between groups, while hospital and ICU length of stay were longer in the tCVST-positive group. Injury severity was higher in tCVST-positive patients. Head AIS distribution also differed significantly: Cuzick's nonparametric test of trend demonstrated a strong stepwise increase in tCVST prevalence with rising head AIS ($z = 4.60$, $p < 0.0001$), indicating a clear ordinal relationship between head injury severity and thrombosis.

Table 1
Demographic and clinical characteristics of patients with and without tCVST.

Variable	tCVST - (n = 281)	tCVST + (n = 147)	*p - value*
Age, years (mean ± SD)	45.7 ± 19.4	40.3 ± 17.8	0.006
Sex, n (%)			
Male	215 (76.2)	110 (74.8)	0.44
Female	66 (23.8)	37 (25.2)	
Hospital LOS, days (mean ± SD)	11.0 ± 11.9	13.2 ± 13.4	0.02
ICU LOS, days (mean ± SD)	6.3 ± 9.1	8.0 ± 11.1	0.05
Ventilator days (mean ± SD)	4.8 ± 7.5	5.2 ± 7.5	0.09
GCS total (mean ± SD)	11.0 ± 4.5	10.6 ± 4.4	0.21
Head AIS, n (%)			
AIS 2	2 (100.0)	0 (0.0)	< 0.001
AIS 3	127 (80.4)	31 (19.6)	
AIS 4	85 (56.5)	64 (43.5)	
AIS 5	65 (55.6)	52 (44.4)	
AIS 6	2 (100.0)	0 (0.0)	
ISS (mean ± SD)	19.8 ± 9.5	22.7 ± 9.8	0.002
Trauma ISS (mean ± SD)	0.86 ± 0.20	0.86 ± 0.22	0.6
Mortality, n (%)	28 (9.9)	19 (12.9)	0.4

Skull Fractures and Fracture Complexity

Among the 428 patients, 243 had temporal bone fractures (Table 2). tCVST occurred in 42.0%, significantly higher than in patients without. Skull base fractures also demonstrated a significantly higher rate. Isolated parietal, occipital, and frontal fractures were not individually associated with differences in tCVST prevalence.

Table 2
Rate and odds of tCVST by fracture pattern.*

Fracture Type	Present	Absent	p-value	Unadjusted OR (95% CI)
Frontal Bone (%)	21/78 (26.9)	126/350 (35.9)	*0.1*	0.7 (0.8–1.8)
Basilar (%)	12/38 (31.6)	9/40 (22.5)	*0.4*	1.6 (0.5–5.0)
Parietal Bone (%)	46/122 (37.7)	101/306 (33.0)	*0.4*	1.2 (0.8–1.9)
Right	22/51 (43.1)	122/374 (32.6)	*0.8*	1.1 (0.6–1.8)
Left	21/54 (38.9)	123/371 (33.1)	*0.9*	1.0 (0.6–1.6)
Occipital Bone	77/212 (36.3)	70/216 (32.3)	*0.4*	1.2 (0.8–1.8)
Basilar (%)	13/41 (31.7)	64/171 (37.4)	*0.5*	0.8 (0.3–1.7)
Temporal Bone (%)	102/243 (42.0)	45/185 (24.2)	*< 0.001*	2.3 (1.5–3.5)
Basilar (%)	80/176 (45.5)	22/67 (32.8)	*0.08*	1.7 (0.9–3.2)
Right	58/143 (40.6)	89/21 (31.7)	*0.07*	1.5 (1.0–2.2)
Left	61/141 (43.3)	86/283 (30.4)	*< 0.01*	1.8 (1.2–2.7)
Skull Base (%)	101/268 (37.7)	46/160 (28.6)	*0.05*	1.5 (1.0–2.3)
Temporal Bone + Occipital Bone (%)	46/98 (46.9)	101/330 (30.6)	*0.003*	2.0 (1.3–3.2)
Temporal Bone + Parietal Bone (%)	36/77 (46.8)	111/351 (31.5)	*0.01*	1.9 (1.1–3.2)
Temporal Bone + Frontal Bone (%)	10/23 (43.5)	137/405 (33.7)	*0.3*	1.5 (0.6–3.8)
Parietal Bone + Occipital Bone (%)	19/40 (47.5)	128/388 (32.9)	*0.06*	1.8 (0.0–3.7)
Frontal Bone + Occipital Bone (%)	3/11 (27.3)	144/417 (34.4)	*0.6*	0.7 (0.1–3.0)

Temporal fractures involving the skull base showed a tCVST prevalence of 45.5% versus 32.8% in non-basilar temporal fractures. Parietal, occipital, and frontal fractures with or without skull base extension were not individually associated with differences in prevalence. Basilar and non-basilar fractures of the occipital bone had similar tCVST prevalence. Several fracture combinations conferred particularly high risk. Temporal-occipital fractures were associated with a 46.9% tCVST rate versus 30.5% for all other combinations. Similarly, temporal-parietal fractures carried a prevalence of 46.8% versus 31.5% for other

patterns. Occipital-parietal fractures had a 47.5% prevalence of tCVST, although this difference was not significant.

tCVST occurred in 25.9% of patients with a single fractured region, increasing to 66.7% in those with five fractured regions (p = 0.02). tCVST was present in 29/87 (33.3%) isolated temporal, 27/99 (27.3%) occipital, 8/34 (23.5%) frontal, and 3/18 (16.7%) parietal bone fractures. In a simple, univariate logistic regression, each additional fractured region increased tCVST odds by 45% (OR 1.45, 95% CI 1.16–1.81; p = 0.001). In a multivariable model adjusting for age, sex, GCS, and intracranial hemorrhage, fracture count did not retain significance (adjusted OR 1.24, 95% CI 0.95–1.64; p = 0.12). SDH remained independently associated with tCVST (adjusted OR 1.67, 95% CI 1.02–2.74; p = 0.04), and higher ISS correlated with increased tCVST odds (adjusted OR 1.03 per point, 95% CI 1.01–1.06; p = 0.01). Specific fracture locations were not independently associated with tCVST. Model discrimination was acceptable (AUC 0.71) and calibration was good (Hosmer–Lemeshow p = 0.53).

Intracranial Hemorrhage, Mass Effect, and BCVI

As reflected in Table 3, on univariate analysis, younger age was associated with higher tCVST odds. Higher head AIS was strongly predictive, and increasing ISS also demonstrated a significant association. Subdural hematoma (SDH) presence was significantly associated with tCVST on univariable analysis, whereas SDH thickness was not. EDH presence alone was not associated with tCVST, but EDH thickness demonstrated a modest association. Increasing midline shift was independently associated with tCVST. BCVI was associated with increased odds of tCVST. Sex, initial GCS, and isolated occipital fractures were not significantly associated on univariable analysis. Extraluminal sinus compression occurred in 127/428 patients (29.7%) and at similar rates in patients with and without tCVST (36.2% vs. 33.6%, p = 0.60).

Table 3
Univariate, non-fracture-related predictors of tCVST.

Variable	OR	95% CI	*p-value*
Age	0.98	0.97–0.99	*< 0.01*
Male sex	0.93	0.58–1.47	*0.7*
GCS	0.98	0.94–1.02	*0.38*
Head AIS	1.8	1.4–2.3	*< 0.001*
ISS	1.03	1.01–1.05	*< 0.01*
Subdural hematoma (present)	1.9	1.2–3.0	*< 0.01*
Epidural hematoma (present)	0.8	0.5–1.3	*0.4*
SDH size (mm)	1.02	0.98–1.07	*0.3*
EDH size (mm)	1.06	1.00–1.11	*0.04*
Midline shift (mm)	1.08	1.02–1.14	*0.01*
BCVI (present)	2.42	1.24–4.73	*< 0.01*

LASSO logistic regression was performed using the variables represented in Table 3, and retained four variables for inclusion in the final multivariable model: temporal fracture, EDH thickness, midline shift, and BCVI. Age and sex were added based on significance from univariate analysis (Table 4). Upon fitting the model, head AIS (treated continuously) was independently associated with tCVST, as were temporal fracture, and male sex. EDH size, the midline shift, and BCVI lost significance after adjustment. The model explained approximately 26% of the variability in tCVST risk (pseudo $R^2 = 0.257$) that we observed. When applied to the 245 patients classified as indeterminate due to lack of venous-phase imaging, the model estimated that approximately 83 patients may have harbored undiagnosed tCVST.

Table 4
Predictors of tCVST assessed in the final regression model. Overall model p-value = 0.005.

Predictor	Adjusted OR	95% CI	*p-value*
Age (per year)	0.99	0.96–1.02	0.61
Male Sex	6.45	1.12–37.26	0.04
Admission GCS (per point)	0.97	0.86–1.11	0.68
Head AIS (per unit)	4.48	1.41–14.26	0.01
Temporal Bone Fracture (present)	8.00	2.14–29.99	0.002
Epidural Hematoma Thickness (per mm)	1.00	0.93–1.09	0.91
Midline Shift (per mm)	1.05	0.88–1.25	0.62
BCVI (present)	3.06	0.59–15.92	0.18

Fracture Laterality and Risk of tCVST

To evaluate whether the side of skull fracture correlated with the laterality of the venous thrombosis we performed a subset analysis of those patients with tCVST. Patients with left temporal fractures had a four-fold increased risk of left-sided tCVST (20.7% vs. 5.1%, RR 4.04, 95% CI 2.67–6.12; $p < 0.001$). Patients with right temporal fractures demonstrated a similar elevation in risk of right-sided tCVST (16.0% vs. 3.5%; RR 4.56, 95% CI 2.77–7.52; $p < 0.001$). Right parietal fractures were also associated with an increased risk of right-sided tCVST (13.5% vs. 6.5%; RR 2.07, 95% CI 1.22–3.52; $p = 0.007$). Left parietal fractures did not significantly increase the risk of left-sided thrombosis (9.3% vs. 1.4%; RR 1.56, 95% CI 0.95–2.55; $p = 0.082$). The presence of either a frontal or parietal fracture increased the risk of superior sagittal sinus thrombosis nearly five-fold (3.6% vs. 0.7%; RR 4.78, 95% CI 1.55–14.70; $p = 0.003$).

A substantial number of thromboses occurred without corresponding overlying fractures: 31/87 (35.6%) left transverse or sigmoid tCVSTs lacked a corresponding left-sided fracture. 21/65 (32.3%) right transverse or sigmoid tCVSTs lacked a right-sided fracture; 4/16 (25%) superior sagittal sinus thromboses occurred without an overlying fracture.

Discussion

The principal finding of this study is that no skull fracture pattern identified a reliably low-risk subgroup for tCVST. For every variant prevalence consistently exceeded 15%, and temporal, temporal-occipital, and temporal-parietal fracture patterns approached 50%. The 42% prevalence associated with temporal fractures mirrors the work by Slasky et. al.[5], who reported a rate of 38%. Similarly, Hoffman[13] reported a 60.5% rate of “cerebral venous injury” in a sample of TBI patients taken from a four-year period from the National Trauma Databank, although it is unclear how many of these represented tCVST. While certain fracture combinations nearly doubled the odds of tCVST, baseline risk associated with any skull fracture remained strikingly high. These findings argue against selective imaging based solely on fracture location; any skull fracture in moderate or severe TBI may represent a high-risk marker warranting venous-phase imaging.

Because many patients with negative CTA did not undergo CTV or MRV, we examined whether their exclusion biased our estimates. We applied the final LASSO-derived model derived from patients with verified imaging to the 245 classified as “indeterminate.” This analysis estimated that 83 patients (nearly one-third) likely harbored undiagnosed tCVST. Indeterminate patients closely resembled tCVST-positive patients with respect to GCS, head AIS, SDH prevalence, and overall fracture burden, differing primarily in specific fracture locations, which were themselves associated with high tCVST prevalence in both groups. Classifying these patients as tCVST-negative would have resulted in substantial disease misclassification and biased effect estimates toward the null. Their exclusion was thus statistically justified and clinically conservative, although it may have increased the observed prevalence of tCVST by introducing selection bias.

Head AIS demonstrated a strong stepwise increase in tCVST risk on trend testing but behaved inconsistently when modeled categorically due to sparse strata and collinearity. Together with our fracture analyses, this suggests that global head injury severity contributes to risk, but specific fracture patterns and associated intracranial pathology (e.g., SDH, EDH thickness, midline shift) may be more useful for screening decisions. In practice, the presence of any skull fracture should lower the threshold for venous-phase imaging regardless of AIS category.

To determine whether higher-risk fracture combinations simply reflected more severe injuries, we compared head AIS distributions among patients with temporal–occipital or temporal–parietal fractures and those without these combinations. Temporal–occipital fractures were associated with slightly higher AIS, but temporal–parietal fractures occurred across a similar AIS distribution while still demonstrating markedly increased tCVST prevalence. This suggests that their elevated risk cannot be fully explained by global head injury severity, and may instead reflect biomechanical forces intrinsic to multiregional skull disruption such as shear, sinus tethering, or endothelial stretch.

Extraluminal sinus compression, commonly considered a risk factor for tCVST[10] and present in roughly one-third of our cohort, did not emerge as an independent predictor and was omitted by our LASSO

regression. This likely reflects mediation by upstream variables such as EDH thickness. While EDH is classically attributed to arterial bleeding (85–90%), 10–15% are from venous sources, often dural sinus injury. The absence of a clear compression-thrombosis association probably reflects the limitations of CT scan in characterizing the magnitude of compression, or in discriminating this compression from true thrombosis. Additionally, some EDHs likely originated directly from sinus wall injury and resultant endothelial injury–a well-described risk factor for thrombosis–but the contribution of this type of injury to tCVST prevalence cannot be determined from our data.

Previous literature emphasizes fractures overlying or adjacent to the affected sinus as central to the pathogenesis of tCVST.[5,7,8] In our cohort, however, only about 65–75% of tCVSTs occurred with an ipsilateral fracture. Up to one-third occurred without any anatomically-corresponding fracture, implicating additional mechanisms such as rotational or shear forces, sinus tethering, remote endothelial injury, mechanical stretching, or altered intracranial pressure dynamics. Our findings therefore challenge a purely local fracture-contact model of tCVST formation and support a broader understanding in which skull fractures portend exposure to significant biomechanical forces that can trigger sinus damage even at distant anatomic sites. Instead, the presence of skull fracture may function primarily as a marker of the magnitude and distribution of biomechanical forces transmitted to the venous sinuses during injury.

Our analytic approach was strengthened by explicit use of a DAG to conceptually distinguish true confounders from mediators and colliders. This framework clarified which variables temporally precede skull fracture patterns and tCVST and therefore warranted adjustment. The association between BCVI and tCVST identified in this study, for example, should be interpreted as a screening signal reflecting injury severity and fracture-related risk, rather than evidence of a direct biological relationship. Accordingly, restricting adjustment to age, sex, and injury mechanism helped avoid overadjustment that might have attenuated true associations. This allowed more accurate estimation of the relationship between fracture patterns and tCVST, and reduced the likelihood that our findings reflected modeling error rather than underlying causal structure.[14]

A key limitation of our study is that our trauma databases do not systematically capture tCVST as a diagnosis, necessitating the use of skull fracture as a proxy. This approach is well-supported by recent literature. Kapitansky et. al.[15] reported simultaneous skull fractures in 169/170 (99.4%) tCVST patients. In a 2024 series by Schwartz et. al., 73/76 patients (96.0%) also had fractures,[16] and a 2021 Norwegian study found fractures in all 73 tCVST patients they studied.[17] Accordingly, fracture-negative tCVST appears to be rare and unlikely to meaningfully change our prevalence estimates. Nevertheless, our findings cannot be generalized to TBI patients without fractures. More comprehensive tCVST capture irrespective of fracture status is needed to fully characterize risk in the larger TBI patient population. Our findings demonstrate, however, that a direct anatomic relationship between a fracture sinus is not necessary for tCVST formation; up to one-third of thromboses arose contralateral to visible fractures. This suggests that skull fractures function as powerful markers for the biomechanical environment that predisposes to venous sinus injury rather than as necessary structural precursors. Additionally, although

LASSO regression was used to provide a data-driven approach to variable selection, standard errors and confidence intervals from the final multivariable model do not fully account for the uncertainty introduced during the selection process. As such, measures of statistical significance should be interpreted cautiously. Similarly, we were limited in our ability to account for potential inter-radiologist variability in the level of detail used to describe skull fractures and other intracranial pathology.

Given that even the lowest-risk fracture pattern in our cohort carried a tCVST prevalence exceeding 15%, and several multiregional patterns approached or exceeded 45%, no fracture pattern reliably identified a low-risk subgroup. Coupled with model-based estimates suggesting that one in three patients without venous-phase imaging likely harbored an undiagnosed thrombosis, these findings argue against selective screening strategies. Rather, they support routine venous-phase imaging (CTV or MRV) in all adults with moderate or severe blunt TBI who present with any skull fracture. Until prospective studies identify a reliably low-risk subgroup, clinicians should maintain a low threshold for venous-phase imaging in adults with moderate or severe blunt TBI and skull fractures.

This recommendation must be balanced against the resource implications of routine venous-phase imaging, including additional time, contrast exposure, and financial cost. In many trauma systems, venous-phase studies are not routinely obtained at initial presentation, and implementation of a universal screening strategy would require additional imaging in a subset of patients. These considerations underscore the need for future work to define the most efficient and cost-effective screening strategies. Although no diagnoses of tCVST were made on the basis of MRV in this cohort, MRV was occasionally used to confirm the absence of tCVST. This pattern appears to reflect care team discretion rather than the relative diagnostic performance of MRV versus CT-based venous imaging.

Our work in tCVST must now clarify the clinical implications of the diagnosis. Our ongoing efforts suggest that the consequences of tCVST likely include difficulty managing intracranial hypertension, worsening of existing intracranial hemorrhage, and increased risk of venous strokes.[18] Major questions remain regarding indications for medical therapy in treatment of this condition, how to balance anticoagulation against the dangers of coexistent bleeding risks, and optimal treatment duration and the impact of treatment on functional outcomes. Moreover, there may be an emerging role for the endovascular management of tCVST. Future work must focus not only on refining diagnostic strategies, but also on defining the therapeutic and outcome implications of tCVST, so that treatment can move from empiric to evidence-based practice.

Conclusion

Traumatic cerebral venous sinus thrombosis appears to be substantially more common among patients with skull fractures than previously appreciated. In this single-institutional cohort, no fracture pattern reliably identified a low-risk subgroup, and model-based estimates suggested that a substantial proportion of thromboses remain undiagnosed when venous-phase imaging is not obtained. Our findings challenge the model of tCVST formation driven by direct contact of the affected sinus with an overlying

fracture, and instead favor a broader biomechanical model in which skull fractures serve as markers of force that can potentiate thrombosis even at remote locations. Until prospective studies define a clearly low-risk population, routine or low-threshold venous-phase imaging should be strongly considered in adults with moderate or severe blunt TBI and any skull fracture. Clarifying the clinical implications of tCVST will be essential to guide future therapeutic strategies.

Declarations

This manuscript complies with the instructions for authors as published on the Neurocritical Care manuscript submission website.

Author Contribution Declaration:

JEW – Study conception and design, acquisition of data, statistical analysis and interpretation, manuscript drafting and revision, final manuscript approval.

TLH – Study conception and design, data analysis, manuscript revision and final approval.

BLS – Study conception and design, data analysis, manuscript revision, and final approval.

JSR – Study conception and design, statistical review and revision, manuscript review and revision, final manuscript approval.

GTS – Study conception and design, data analysis and interpretation, manuscript review and revision, final manuscript approval.

TMS – Study conception and design, data analysis and interpretation, manuscript review and revision, final manuscript approval.

DMS - Study conception and design, data analysis and interpretation, manuscript review and revision, final manuscript approval.

All authors have made substantial contributions to the manuscript as outlined above, and take collective responsibility for the results published herein. Permission for submission of the final manuscript has been provided by all authors.

This manuscript has not been published elsewhere and is not under consideration by any other journal.

The study reported by this manuscript adheres to all relevant ethical guidelines and best practices, has undergone appropriate Institutional Review Board review, and a statement regarding IRB approval appears within the text of the manuscript. The study was deemed to be exempt based on IRB review.

Conflict of interest disclosure statement: The authors have no competing interests to disclose.

The STROBE reporting checklist was utilized in the composition of this manuscript, and is included with the submission in accordance with author instructions.

Sources of Funding Statement: No funding was received to support this work.

References

1. Netteland DF, Mejlænder-Evjensvold M, Skaga NO, et al. Cerebral venous thrombosis in traumatic brain injury: a cause of secondary insults and added mortality. J Neurosurg JNS. 2021;134(6):1912–20. 10.3171/2020.4.JNS20511.
2. Giladi O, Steinberg DM, Peleg K, et al. Head trauma is the major risk factor for cerebral sinus-vein thrombosis. Thromb Res. 2016;137:26–9. Epub 2015 Nov 26. PMID: 26653366.
3. Koopman K, Uyttenboogaart M, Vroomen PC et al. Long-term sequelae after cerebral venous thrombosis in functionally independent patients. *J Stroke Cerebrovasc Dis.* 2009 May-Jun;18(3):198–202. 10.1016/j.jstrokecerebrovasdis.2008.10.004. PMID: 19426890.
4. Saposnik G, Bushnell C, Coutinho JM, On behalf of the American Heart Association Stroke Council, et al. Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation; Council on Cardiovascular and Stroke Nursing; and Council on Hypertension. Diagnosis and Management of Cerebral Venous Thrombosis: A Scientific Statement From the American Heart Association. Stroke. 2024 March;55:e77–90.
5. Slasky SE, Rivaud Y, Suberlak M et al. Venous Sinus Thrombosis in Blunt Trauma: Incidence and Risk Factors. *Journ Comp Assist Tom.* 2017.41(6):891–897, 2017. | 10.1097/RCT.0000000000000620
6. Zhao X, Rizzo A, Malek B et al. Basilar skull fracture: a risk factor for transverse/sigmoid venous sinus obstruction. *J Neurotrauma*. 2008;25(2):104 – 11. 10.1089/neu.2007.0351. PMID: 18260793.
7. Gabay S, Dunetz G, Bergman L, et al. Are isolated linear fractures over major dural venous sinuses a risk factor for sinus thrombosis in mild TBI? Neuroradiology. 2025;67(7):1773–81. 10.1007/s00234-025-03670-6. Epub 2025 Jun 23. PMID: 40549183.
8. Hendrickson S, Murahari S, Scotter J et al. Venous sinus injuries are common with occipital skull fractures. *Scand J Trauma Resusc Emerg Med.* 2014;22(Suppl 1):O8. 10.1186/1757-7241-22-S1-O8. PMCID: PMC4123231.
9. Saitta L, Resaz M, Robba C, et al. Neuroimaging for neurovascular complications of traumatic brain injury. Crit Care. 2025;29(1):362. 10.1186/s13054-025-05586-2. PMID: 40826095; PMCID: PMC12362949.
10. Xu W, Gao L, Li T, et al. The Performance of CT versus MRI in the Differential Diagnosis of Cerebral Venous Thrombosis. Thromb Haemost. 2018;118(6):1067–77. 10.1055/s-0038-1642636. Epub 2018 Apr 25. PMID: 29695023.
11. Council COPE. COPE position – Authorship and AI – English. https://doi.org/10.24318

/cCVRZBMS. 2024 Committee on Publication Ethics (CC BY-NY-ND 4.0). https://publicationethics.org. Accessed November 28, 2025.

12. International Committee of Medical Journal Editors, Defining the Role of Authors and Contributors, Artificial Intelligence (AI)-Assisted Technology. 2025. https://www.icmje.org/recommendations/browse/roles-and-responsibilities/defining-the-role-of-authors-and-contributors.html. Accessed November 28, 2025.
13. Hoffman H, Otite FO, Chin LS. Venous Injury in Patients with Blunt Traumatic Brain Injury: Retrospective Analysis of a National Cohort. Neurocrit Care. 2022;36:116–22. 10.1007/s12028-021-01265-6. https://doi-org.proxy-hs.researchport.umd.edu/.
14. Textor J, van der Zander B, Gilthorpe MK, et al. Robust causal inference using directed acyclic graphs: the R package 'dagitty'. Int J Epidemiol. 2016;45(6):1887–94.
15. Kapitanski L, Thiyagarajah K, Makedonov I, et al. Clinical characteristics, management and outcomes of traumatic cerebral venous sinus thrombosis. J Trauma Acute Care Surg. 2025;00(00):1–7.
16. Schwartz DA, Talbott J, Callen A, et al. Classification of traumatic injury to the dural venous sinus using CT venography. J Neuroimaging. 2024;34:205–10.
17. Netteland DF, Sandset EC, Mejlænder-Evjensvold M, et al. Cerebral venous sinus thrombosis in traumatic brain injury: A systematic review of its complications, effect on mortality, diagnostic and therapeutic management, and follow-up. Front Neurol. 2023;13:1079579. PMID: 36698879; PMCID: PMC9869151.
18. Ragurajaprakash K, Senthilkumar R, Sathish Prabu SS et al. Post-traumatic cerebral venous sinus thrombosis – Institutional study and literature review. *Interdisciplinary Neurosurgery.* 2022 27, 2022, ISSN 2214–7519. https://doi.org/10.1016/j.inat.2021.101398

Figures

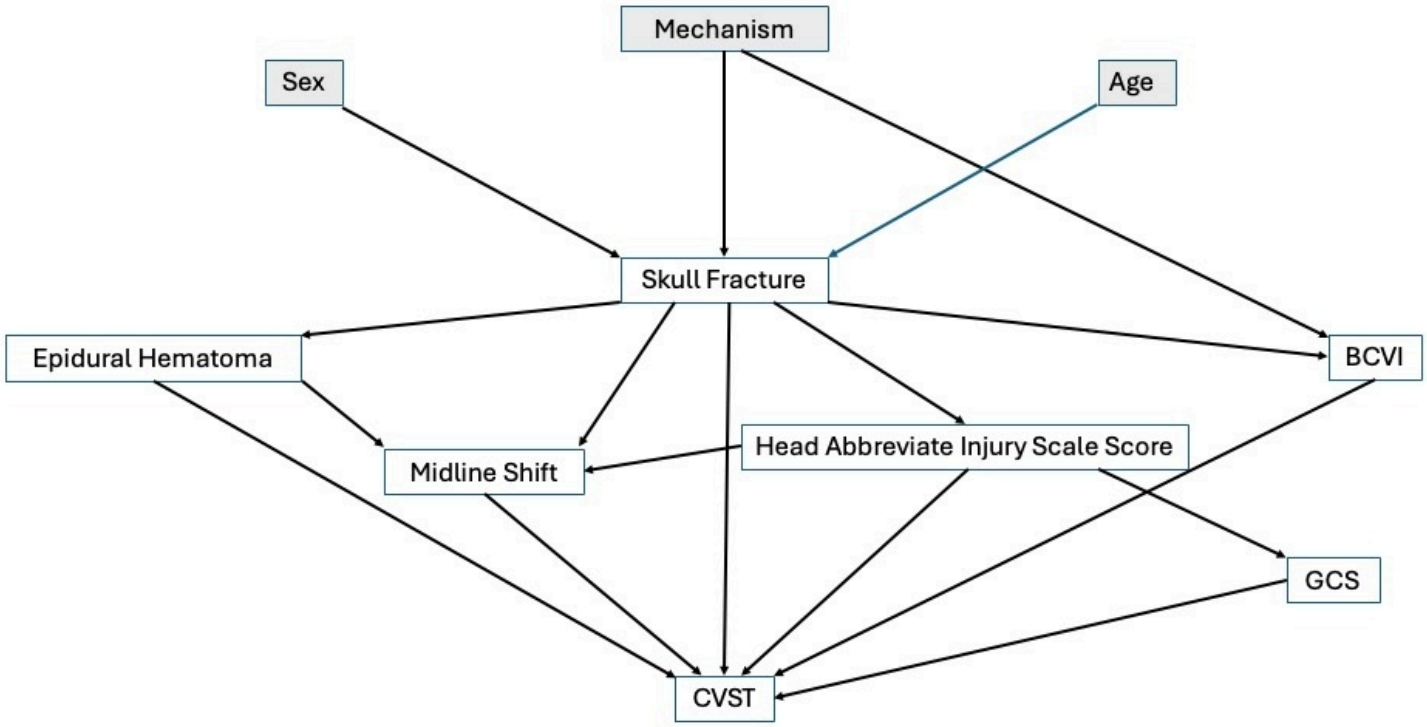


Figure 1

Directed Acyclic Graph (DAG) depicting the assumed causal relationship between skull fracture patterns and the development of tCVST. Age, sex and mechanism were identified as confounders. Head AIS, epidural hematoma, degree of midline shift, and GCS were modeled as downstream consequences of fracture and excluded from the adjustment set to avoid overadjustment bias.

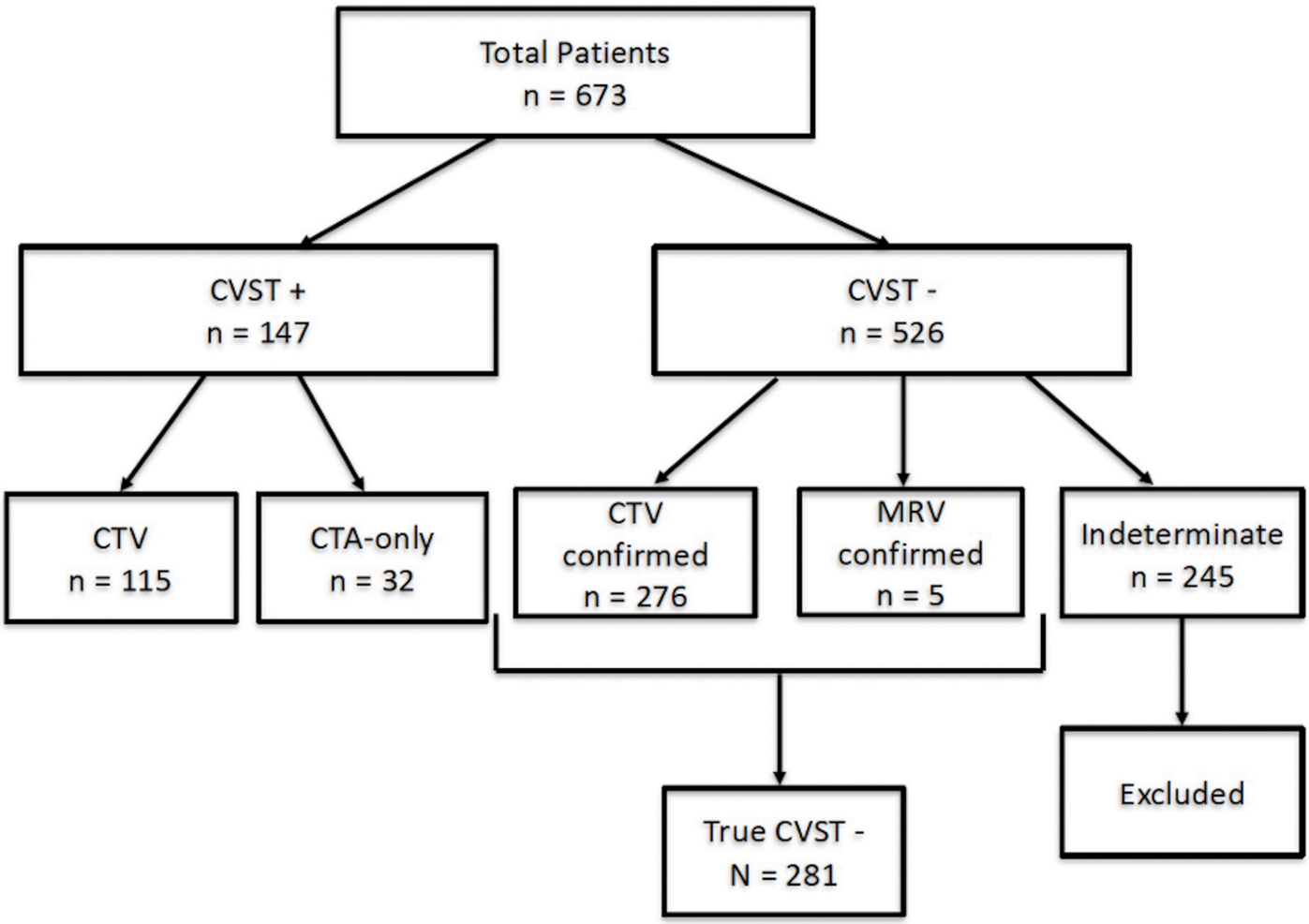


Figure 2

Flowchart outlining patient identification, imaging verification of tCVST status, and assembly of the final study cohort.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- STROBEchecklistcompleted.pdf