Development of cerebral vasospasm following traumatic intracranial hemorrhage: incidence, risk factors, and clinical outcomes

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OBJECTIVE Limited evidence exists characterizing the incidence, risk factors, and clinical associations of cerebral vasospasm following traumatic intracranial hemorrhage (tICH) on a large scale. Therefore, the authors sought to use data from a national inpatient registry to investigate these aspects of posttraumatic vasospasm (PTV) to further elucidate potential causes of neurological morbidity and mortality subsequent to the initial insult.

METHODS Weighted discharge data from the National (Nationwide) Inpatient Sample from 2015 to 2018 were queried to identify patients with tICH who underwent diagnostic angiography in the same admission and, subsequently, those who developed angiographically confirmed cerebral vasospasm. Multivariable logistic regression analysis was performed to identify significant associations between clinical covariates and the development of vasospasm, and a tICH vasospasm predictive model (tICH-VPM) was generated based on the effect sizes of these parameters.

RESULTS Among 5880 identified patients with tICH, 375 developed PTV corresponding to an incidence of 6.4%. Multivariable adjusted modeling determined that the following clinical covariates were independently associated with the development of PTV, among others: age (adjusted odds ratio [aOR] 0.98, 95% CI 0.97–0.99; $p < 0.001$), admission Glasgow Coma Scale score < 9 (aOR 1.80, 95% CI 1.12–2.90; $p = 0.015$), intraventricular hemorrhage (aOR 6.27, 95% CI 3.49–11.26; $p < 0.001$), tobacco smoking (aOR 1.36, 95% CI 1.02–1.80; $p = 0.035$), cocaine use (aOR 3.62, 95% CI 1.97–6.63; $p < 0.001$), fever (aOR 2.09, 95% CI 1.34–3.27; $p = 0.001$), and hypokalemia (aOR 1.62, 95% CI 1.26–2.08; $p < 0.001$). The tICH-VPM achieved moderately high discrimination, with an area under the curve of 0.75 (sensitivity = 0.61 and specificity = 0.81). Development of vasospasm was independently associated with a lower likelihood of routine discharge (aOR 0.60, 95% CI 0.45–0.78; $p < 0.001$) and an extended hospital length of stay (aOR 3.53, 95% CI 2.78–4.48; $p < 0.001$), but not with mortality.

CONCLUSIONS This population-based analysis of vasospasm in tICH has identified common clinical risk factors for its development, and has established an independent association between the development of vasospasm and poorer neurological outcomes.

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KEYWORDS vasospasm; intracranial hemorrhage; trauma; subarachnoid hemorrhage; database; predictive model; decision-tree analysis

ALTHOUGH cerebral vasospasm represents an area of intensive investigation in the setting of aneurysmal subarachnoid hemorrhage (SAH), comparatively less is known of this entity following traumatic intracranial hemorrhage (tICH). Documented rates of

incidence range widely (5%–63%) as a result of minimal surveillance for its development and the application of varied imaging and diagnostic modalities for its detection.¹ The etiology of posthemorrhagic vasoconstriction of the cerebral vasculature remains unclear, while the prognostic

ABBREVIATIONS aOR = adjusted odds ratio; DCI = delayed cerebral ischemia; GCS = Glasgow Coma Scale; HCUP = Healthcare Cost and Utilization Project; IVH = intraventricular hemorrhage; LOS = length of stay; NIS = National (Nationwide) Inpatient Sample; PTV = posttraumatic vasospasm; ROC = receiver operating characteristic; SAH = subarachnoid hemorrhage; TBI = traumatic brain injury; tICH = traumatic intracranial hemorrhage; VPM = vasospasm predictive model.

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significance of vasospasm in traumatic brain injury (TBI) has largely been unexamined. As the incidence of TBI continues to increase in the US,² it is paramount to further elucidate potential causes of neurological morbidity and mortality subsequent to the initial insult. This study uses data from a national inpatient registry to characterize the incidence, risk factors, and clinical outcomes of posttraumatic vasospasm (PTV) on a large scale.

Methods

Data Source

The National (Nationwide) Inpatient Sample (NIS), developed and maintained by the Healthcare Cost and Utilization Project (HCUP), is among the largest publicly accessible inpatient care databases in the US. Yearly unweighted data approximate 7,000,000 patients, reflecting a 20% stratified sample of all HCUP-participating community hospitals nationally. The large sample size afforded by the NIS allows for substantive inquiry into healthcare utilization, access, charges, quality, and outcomes, as well as reliable reproduction of national estimates annually. Data elements include demographic characteristics, hospital and regional information, diagnoses, procedures, and discharge disposition for all documented patients. More information regarding the NIS and data access can be found at <https://www.hcup-us.ahrq.gov>. Given the public accessibility and de-identified nature of the information in this database, this study did not meet the requirements for institutional review board approval. For the same reason, patient consent was not required.

Patient Selection and Cohort Development

International Classification of Diseases, Tenth Revision, Clinical Modification (ICD-10-CM) codes were used to identify patients with admission diagnoses for intracranial injury (ICD-10-CM S06.X) from 2015 (fourth quarter, October through December) to 2018 and included focal traumatic hemorrhage of the cerebrum, brainstem, and cerebellum (ICD-10-CM S06.3X), traumatic SAH (S06.6X), and intraventricular hemorrhage (IVH) due to trauma (ICD-10-CM I61.5 with S06.X). Patients were included if they underwent diagnostic cerebral angiography (ICD-10-PCS B31XXXX) during the index hospitalization for their trauma admission; this was to ensure that radiographic evidence was used to confirm the presence or absence of cerebral vasospasm (ICD-10-CM I67.84), which was identified in accordance with previous literature.^{3,4} Intracranial arterial angioplasty for the treatment of vasospasm was identified using the relevant procedural identifier (ICD-10-PCS 037GXXX). The finalized cohort was queried to identify demographic characteristics (age and female sex) as well as comorbid conditions and clinical variables associated with the development of vasospasm.

Clinical Endpoints

The primary clinical endpoint of this analysis was the development of PTV. Clinical outcomes (routine discharge to home or self-care, mortality, and hospital length of stay [LOS]) were evaluated as a secondary endpoint.

Statistical Analysis

All analyses were performed within a complex samples function with appropriate stratum and cluster variables and discharge weights per HCUP guidelines to account for NIS sampling design and, ultimately, to yield accurate national estimates. Descriptive statistics were performed to measure variation in baseline demographic and clinical characteristics as well as in outcomes between patients with traumatic hemorrhage who developed PTV and those who did not develop PTV. Dichotomous variables were assessed using Pearson's chi-square test and are presented as count (%), while estimated marginal means for continuous baseline parameters were computed using the t-test, within a complex samples general linear model, and reported with SEM. Statistical significance was evaluated at $p < 0.05$ for univariable comparisons.

A multivariable logistic regression model composed of all baseline clinical parameters identified significant independent associations with the development of PTV. Beta (β) coefficients representing effect sizes of significant parameters (x) were generated to construct a tICH vasospasm predictive model (tICH-VPM) described by the following expression:

$$\sum_{i=0}^n \beta_0 x_0 + \beta_1 x_1 \dots \beta_n x_n.$$

Receiver operating characteristic (ROC) curve analysis was subsequently performed to evaluate the discriminatory capacity of this model, while the Youden index was used to identify sensitivity and specificity based on the coordinate points of the highest combination of these two parameters on the ROC curve. Finally, decision-tree analysis was performed using a chi-square automatic interaction detection (CHAID) algorithm using all baseline parameters as candidate variables for node construction with adjusted Bonferroni testing for decision-making.

Results

Among 5880 identified patients with tICH, 375 developed PTV for an overall incidence of 6.4%; 75 (20%) of these patients were subsequently treated with cerebral angioplasty. Univariable comparison of baseline characteristics and clinical variables between patients who developed PTV and those who did not develop PTV is shown in Table 1. Following multivariable logistic regression analysis, the following clinical covariates were independently associated with the development of PTV: age (adjusted odds ratio [aOR] 0.98, 95% CI 0.97–0.99; $p < 0.001$), female sex (aOR 1.80, 95% CI 1.43–2.27; $p < 0.001$), admission Glasgow Coma Scale (GCS) score < 9 (aOR 1.80, 95% CI 1.12–2.90; $p = 0.015$), SAH (aOR 2.46, 95% CI 1.45–4.12; $p = 0.001$), IVH (aOR 6.27, 95% CI 3.49–11.26; $p < 0.001$), obesity (aOR 2.34, 95% CI 1.65–3.37; $p < 0.001$), diabetes mellitus (aOR 0.45, 95% CI 0.31–0.67; $p < 0.001$), hypertension (aOR 1.77, 95% CI 1.36–2.30; $p < 0.001$),

TABLE 1. Comparison of baseline demographic and clinical characteristics, hospital course, and outcomes between patients with tICH with and without development of cerebral vasospasm

	Total Cohort (n = 5880)	Vasospasm (n = 375, 6.4%)	No Vasospasm (n = 5505, 93.6%)	p Value
Baseline demographic & clinical characteristics				
Mean age, yrs (SE)	55.9 (0.3)	51.6 (0.9)	56.2 (0.3)	<0.001
Female sex	2285 (38.9)	190 (50.7)	2095 (38.1)	<0.001
Admission GCS score <9	200 (3.4)	25 (6.7)	175 (3.2)	<0.001
Intraparenchymal hemorrhage	1100 (18.7)	50 (13.3)	1050 (19.1)	0.006
SAH	4995 (84.9)	320 (85.3)	4675 (84.9)	0.830
IVH	230 (3.9)	45 (12.0)	185 (3.4)	<0.001
Comorbidities				
Obesity	390 (6.6)	50 (13.3)	340 (6.2)	<0.001
Diabetes mellitus	1060 (18.0)	40 (10.7)	1020 (18.5)	<0.001
Hypertension	3230 (54.9)	225 (60.0)	3005 (54.6)	0.041
Congestive heart failure	575 (9.8)	40 (10.7)	535 (9.7)	0.550
Chronic renal disease	485 (8.2)	25 (6.7)	460 (8.4)	0.250
Hyperlipidemia	1580 (26.9)	85 (22.7)	1495 (27.2)	0.058
Substance use				
Tobacco smoking	900 (15.3)	80 (21.3)	820 (14.9)	0.001
Cocaine	95 (1.6)	15 (4.0)	80 (1.5)	<0.001
Alcohol	1135 (19.3)	70 (18.7)	1065 (19.3)	0.747
Laboratory & vital parameters				
Leukocytosis	475 (8.1)	45 (12.0)	430 (7.8)	0.004
Acute anemia	NR	NR	NR	<0.001
Hyperglycemia	275 (4.7)	NR	265 (4.8)	0.057
Hyponatremia	1040 (17.7)	85 (22.7)	955 (17.3)	0.009
Hypernatremia	710 (12.1)	60 (16.0)	650 (11.8)	0.016
Hypokalemia	1115 (19.0)	120 (32.0)	995 (18.1)	<0.001
Fever	215 (3.7)	30 (8.0)	185 (3.4)	<0.001
Acute elevated blood pressure	220 (3.7)	40 (10.7)	180 (3.3)	<0.001
Clinical outcomes				
Routine discharge	2075 (35.3)	85 (22.7)	1990 (36.1)	<0.001
In-hospital mortality	630 (10.7)	50 (13.3)	580 (10.5)	0.091
Mean LOS, days (SE)	13.2 (0.2)	18.8 (0.5)	12.8 (0.2)	<0.001

NR = not reported due to low case number (n < 11).

Values represent the number of patients (%) and were compared using chi-square analysis. Continuous parameters are presented as mean (SE) and were compared by t-test. Boldface type indicates statistical significance evaluated at p < 0.05 for univariable comparisons.

congestive heart failure (aOR 1.67, 95% CI 1.13–2.45; p = 0.009), tobacco smoking (aOR 1.36, 95% CI 1.02–1.80; p = 0.035), cocaine use (aOR 3.62, 95% CI 1.97–6.63; p < 0.001), fever (aOR 2.09, 95% CI 1.34–3.27; p = 0.001), hyperglycemia (aOR 0.31, 95% CI 0.16–0.60; p = 0.001), hypernatremia (aOR 1.42, 95% CI 1.03–1.94; p = 0.032), hypokalemia (aOR 1.62, 95% CI 1.26–2.08; p < 0.001), acute anemia (aOR 11.47, 95% CI 3.04–43.28; p < 0.001), and acute elevated blood pressure (aOR 3.23, 95% CI 2.15–4.87; p < 0.001) (Table 2).

The tICH-VPM achieved an area under the ROC curve of 0.748 (sensitivity = 0.612, specificity = 0.810, and Youden index = 0.422) (Fig. 1). In decision-tree analysis,

IVH served as the parent node while age subgroups and hypokalemia were secondary nodes in the clinical algorithm of risk factors for development of PTV (Fig. 2).

Patients who developed PTV were routinely discharged at a rate of 22.7%, had a mean LOS of 18.8 days, and died during hospitalization at a rate of 13.3% (Table 1). Following multivariable logistic regression analysis, PTV was independently associated with lower likelihood of a routine discharge (aOR 0.60, 95% CI 0.45–0.78; p < 0.001) and an extended hospital LOS (evaluated at the 75th percentile for durations of hospitalization in the aggregate cohort, corresponding to 18 days) (aOR 3.53, 95% CI 2.78–4.48; p < 0.001), but not with mortality (Table 3).

TABLE 2. Multivariable analysis to determine associations of clinical characteristics with development of cerebral vasospasm

Clinical Characteristic	β Coefficient	aOR (95% CI)	p Value
Age	−0.02	0.98 (0.97–0.99)	<0.001
Female sex	0.59	1.80 (1.43–2.27)	<0.001
Admission GCS score <9	0.59	1.80 (1.12–2.90)	0.015
Intraparenchymal hemorrhage	—	1.11 (0.76–1.62)	0.604
SAH	0.90	2.46 (1.45–4.12)	0.001
IVH	1.84	6.27 (3.49–11.26)	<0.001
Obesity	0.86	2.34 (1.65–3.37)	<0.001
Diabetes mellitus	−0.79	0.45 (0.31–0.67)	<0.001
Hypertension	0.57	1.77 (1.36–2.30)	<0.001
Congestive heart failure	0.51	1.67 (1.13–2.45)	0.009
Chronic renal disease	—	1.04 (0.65–1.67)	0.863
Hyperlipidemia	—	0.83 (0.62–1.12)	0.227
Tobacco smoking	0.31	1.36 (1.02–1.80)	0.035
Cocaine use	1.29	3.62 (1.97–6.63)	<0.001
Alcohol use	—	0.75 (0.55–1.00)	0.053
Leukocytosis	—	1.19 (0.82–1.71)	0.361
Fever	0.74	2.09 (1.34–3.27)	0.001
Hyperglycemia	−1.17	0.31 (0.16–0.60)	0.001
Hyponatremia	—	1.30 (0.99–1.71)	0.056
Hypernatremia	0.35	1.42 (1.03–1.94)	0.032
Hypokalemia	0.48	1.62 (1.26–2.08)	<0.001
Acute anemia	2.44	11.47 (3.04–43.28)	<0.001
Acute elevated blood pressure	1.17	3.23 (2.15–4.87)	<0.001

Effect sizes of baseline clinical covariates from unadjusted analysis for associations with development of vasospasm presented as aORs with 95% CIs following multivariable logistic regression analysis with all baseline clinical characteristics as model covariates. Boldface type indicates statistical significance evaluated at $p < 0.05$. β coefficients from significant parameters were subsequently used to construct the tICH-VPM.

Discussion

The incidence of PTV has historically been difficult to ascertain due to lack of routine assessment. Since it is often not assessed unless there are clinical indications, including delayed cerebral ischemia (DCI), asymptomatic vasospasm is rarely detected. Furthermore, SAH, a major predictor for the development of cerebral vasospasm, is present in as many as 60% of patients with TBI.^{1,5} Previously, it has been shown that vasospasm occurs in as little as 5% and as much as 18.6% of TBI-associated ICH. Now, with the improvement of widespread imaging modalities, the incidence has been estimated to be as high as 63%, although with a larger risk of false positives. In this study, we found the incidence of PTV to be 6.4%, consistent with rates reported from traditional diagnostic imaging modalities.⁶

PTV has been thought to develop similarly to the better-understood pathophysiology of post-SAH vasospasm, whereby the extravasation of blood into the subarachnoid

FIG. 1. ROC curve analysis for the tICH-VPM.

space and resultant release of neuroinflammatory substances through its breakdown cause direct irritation to the cerebral vasculature.⁷ However, this does not explain the incidence of vasospasm in patients lacking SAH after trauma.⁸ In this case, in vitro studies have found that direct trauma through pulling and shearing forces can induce vasospasm as well.^{9,10} Symptomatic vasospasm can be readily caught by consistent neurological examinations, although at that point the effects are often irreversible.¹¹

Traditional comorbid risk factors found in small cohort and population-based studies include high-grade SAH, intracerebral hematoma, and epidural and subdural hematoma formation.¹ Clinical presentations associated with the development of PTV include fever, elevated Injury Severity Scores, decreasing GCS, and the extent of involved cerebrum following TBI.^{12–14} Based on multivariable analysis, we found female sex to be a demographic predictor of PTV, while decreasing age was associated with increased odds of developing PTV. Clinically, the presence of IVH, SAH, and an admission GCS score < 9 were found to be predictive of PTV, in descending order. In terms of comorbidities, anemia, cocaine use, acute elevated blood pressure, obesity, fever, hypokalemia, hypertension, congestive heart failure, hypernatremia, and tobacco use were associated with increased risk of developing PTV, in descending order as well.

In a decision-tree algorithm, IVH was found to be the parent node in establishing the risk of developing PTV. Case reports on the incidence of vasospasm in patients with ruptured arteriovenous malformations have found IVH to be linked with delayed vasospasm as well as DCI 14 days after the initial hemorrhage, perhaps due to stasis of lysed blood products in the CSF space.^{15–17} In a previous analysis of one institution's database on ICH, Amuluru et al. found the incidence of vasospasm in instances of IVH to be 13.9%, with a subsequent DCI incidence of 11.1%, constituting an independent pathophysiological risk fac-

FIG. 2. Decision-tree algorithm for the development of cerebral vasospasm following tICH.

tor.¹⁸ Interestingly, IVH was a far more robust independent predictor of vasospasm in comparison with SAH, the hemorrhage type traditionally associated with its development.

Potential biomarkers for the development of PTV identified in this study include fever, hypokalemia, and hypernatremia, all of which were independent predictors in multivariable modeling. In a previous analysis of biomarkers for the development of vasospasm after SAH, a study that used the Nationwide Readmissions Database found hypernatremia, hypotension, and anemia to be correlated with vasospasm development. The authors acknowledged that poor hemodynamics leading to cerebral hypoxia can result in DCI and linked fever and leukocytosis with the degree of clot burden and vasospasm in these patients.³ Hypokalemia does not have a clear clinical relationship with the development of vasospasm, although previous analyses have postulated electrolyte-induced imbalances in sympathetic activation and other autonomic processes as a potential explanation. Conversely, perhaps sympathetic activation secondary to brain injury, and a known pathogenesis of vasospasm in patients with SAH, also contributes to the development of hypokalemia in these patients.^{3,19}

While we identified many similar comorbidities of TBI that are associated with vasospasm as have similarly been shown, including obesity, hypertension, and tobacco smoking, we identified hyperglycemia and diabetes mellitus as protective factors. Although it has been shown that

glycemic indices can predict outcomes after SAH, with a higher incidence of complications and mortality, the link with the establishment of vasospasm is less clear.²⁰ Perhaps the long-term microvascular effects of poor glycemic control and development of cholesterol plaques and vessel narrowing provide a scaffolding which counters the vasospastic properties of blood breakdown products; however, this must be further investigated.

DCI, induced by the development of vasospasm, is one of the most significant causes of morbidity and mortality following an initial SAH insult. In one cohort study out of Columbia University, DCI was second only to rebleeding as the leading cause of death secondary to the initial bleed in SAH.²¹ The link between vasospasm and clinical

TABLE 3. Multivariable analysis to determine the association of cerebral vasospasm with clinical outcomes

Clinical Outcome	aOR (95% CI)	p Value
Routine discharge	0.60 (0.45–0.78)	<0.001
In-hospital mortality	1.31 (0.92–1.86)	0.130
Extended LOS (75th percentile)	3.53 (2.78–4.48)	<0.001

Effect sizes of cerebral vasospasm for clinical outcomes presented as aORs with 95% CIs following multivariable logistic regression analysis with all baseline clinical characteristics as model covariates. Boldface type indicates statistical significance following Bonferroni correction for multiple comparisons.

outcomes remains poorly understood in the setting of TBI. It has been documented that, while early PTV is linked with poorer outcomes, late PTV and overall PTV have no association.²² This can be due, in part, to the association of early PTV with the development of DCI and late PTV being an asymptomatic finding.²³ Our results show that while the presence of vasospasm leads to less routine discharge and an extended hospital LOS in the TBI cohort, it has no overall increase in the rate of in-hospital mortality. Nonetheless, vasospasm emerged as an independent predictor of poorer neurological outcomes irrespective of the severity of TBI and comorbidity burden.

Limitations

The primary limitations of this investigation are those traditionally associated with retrospective studies that use administrative registry data, by which all clinical variables are derived from billing codes assigned following patient discharge. Cerebral vasospasm was identified using a singular billing code, and, thus, severity and specific localization could not be evaluated in this analysis. Moreover, diagnosis codes do not carry a temporal qualifier, which prevented documentation of when vasospasm occurred and, therefore, introduces potential inaccuracy with clinical covariates included in this analysis (such as laboratory markers) due to the inability to ascertain whether they occurred before or after the development of vasospasm. These limitations notwithstanding, the large cohort size and utilization of national data underscore the generalizability of the findings of our study, which, to our knowledge, is the first of its kind to evaluate vasospasm in tICH on a large scale.

Conclusions

This large-scale analysis using national registry data identified risk factors for the development of PTV in tICH and employed predictive modeling and decision-tree algorithms to aid in the identification of patients at risk. Moreover, it has provided evidence for worse neurological outcomes in patients developing PTV. Further investigation is warranted for management strategies and optimal modalities for detection of PTV in patients with tICH.

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