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Clinical and radiographic characteristics of traumatic brain injury patients undergoing endovascular rescue for posttraumatic vasospasm

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Abstract:

BACKGROUND: Cerebral vasospasm is a serious sequela of traumatic brain injury (TBI) which leads to further neurologic injury subsequent to the initial trauma. The natural history and associated risk factors are not well understood. The objective of this study is to evaluate the clinical and radiographic characteristics of patients with TBI.

METHODS: This is a descriptive case series of all patients with TBI who underwent cerebral angiogram for evaluation and rescue therapy for posttraumatic arterial vasospasm (PTV) between October 2017 and November 2019. The association of clinical and radiographic characteristics with cumulative severity of angiographic vasospasm was evaluated. The clinical characteristics comprised of age, sex, Glascow Coma Scale (GCS) and need for surgery. The radiographic characteristics were presence of subarachnoid hemorrhage (SAH), location of SAH, presence of contusion, presence of subdural hemorrhage (SDH), and presence of pseudoaneurysm.

RESULTS: Twenty-two patients with PTV were identified requiring 69 cerebral angiograms (mean: 3.2; range: 1–9 angiograms per patient) during this period. The average age upon presentation was 40 years old, 81% of the patients were male sex, and the average GCS was 6.8. 67% of the patients underwent craniotomy or craniectomy. All patients had SAH, although only 60% had cisternal SAH. Parenchymal contusion was noted on 90% as well as SDH in 90%. The PTV was noted between 3 and 19 days after trauma. There was more vasospasm involving proximal arteries and higher severity of vasospasm in patients with cisternal SAH. Otherwise, there was no strong association between the clinical or radiographic characteristics and cumulative severity noted on the angiograms for each patient.

CONCLUSION: Posttraumatic vasospasm can be detected as early as posttrauma day 3–19 in patients with TBI and SAH. The absence of cisternal SAH does not rule out the occurrence of the vasospasm during the course of treatment.

Keywords:

Subarachnoid hemorrhage, traumatic brain injury, vasospasm

Introduction

Traumatic brain injury (TBI) is a major source of death and disability worldwide.[1] Although primary injury from the trauma can lead to marked brain damage, the optimal diagnostic and treatment strategy can down modulate further disability from the secondary injury. Posttraumatic arterial vasospasm (PTV)

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is a well-established secondary phenomenon that could lead to further ischemia and infarction of the downstream brain parenchyma, similar to such process after aneurysmal subarachnoid hemorrhage (aSAH).[2]

Transcranial Doppler (TCD) screening and angiographic evaluation of patients with TBI admitted to the neurocritical care unit are more widely adopted in the past decade. Medical and rescue endovascular therapy for PTV is also more readily being used and studied.[3,4]

However, unlike aSAH which has been extensively studied, there are limited studies on the natural history of PTV.[5] Its associated risk factors and the optimal treatment strategy of PTV are not well understood. We aim to evaluate the clinical and radiographic characteristics of patients with TBI who underwent cerebral angiogram for evaluation and rescue treatment of PTV.

Methods

This is a retrospective observational study evaluating the clinical and radiographic characteristics associated with the occurrence of angiographic vasospasm after TBI. The study was approved by the institutional review board.

The inclusion criteria for this study were (1) age >18 years, (2) admission to hospital with blunt TBI, (3) elevated velocities on daily TCDs concerning for cerebral vasospasm with or without new onset of neurological deficits concerning for ischemia, and (4) cerebral angiogram confirming the presence of vasospasm.

The clinical characteristics including age, sex, Glasgow Coma Scale (GCS), and need for craniotomy or craniectomy of the patients fitting the inclusion criteria between the years of 2017 and 2019 at our institution were retrospectively chart abstracted. The presence and location of intraparenchymal contusion, extraaxial hemorrhage, and pseudoaneurysm were retrospectively abstracted after a review of the computed tomography (CT) and CT angiogram of the head upon presentation. The timing, location, and severity of vasospasm were also abstracted after a review of the cerebral angiograms.

The severity of vasospasm was categorized as mild, moderate, and severe for intracranial internal carotid artery, M1, M2, M3-4, A1, A2, A3-4, Basilar, P1, and P2-4 segments.[6] For quantitative evaluation of the severity of vasospasm for each anatomical section, Grades 1, 2 and 3 were assigned for mild, moderate and severe respectively. Total burden of vasospasm was calculated as the sum of the severities for affected segment. Descriptive statistics was used to evaluate the association of the abovementioned baseline clinical and radiographic characteristics with severity of the vasospasm.

To evaluate the location of SAH on the severity of vasospasm, the severity was compared in the patients with cisternal blood to purely superficial cortical blood. The anatomic pattern of vasospasm was also evaluated by comparing the severity of proximal vasospasm involving ICA, M1, A1, Basilar, or P1 compared to distal vasospasm.

To evaluate the temporal evolution of PTV, the total burden of vasospasm for each cerebral angiogram for each patient was normalized over the sum of the burden of all angiograms during that admission, using which a histogram of the severity by the day of admission was plotted.

Clinical trial registry

This work is a retrospective analytical study. No clinical trials were involved.

Results

Out of 612 patients admitted to the neurocritical care unit with TBI, we identified 22 patients who were noted to have posttraumatic vasospasm on cerebral angiogram. There were 69 cerebral angiograms performed in this cohort. Patients had between 1 and 9 angiograms per admission with an average number of 3.2 angiograms. The baseline clinical and radiographic characteristics of this cohort are summarized in Table 1. PTV occurred between Days 3 and 19 after admission. The normalized PTV severity burden showed the peak severity between Days 7 and 14 after admission [Figure 1].

Table 1: Summary of baseline clinical and radiographic characteristics of patients included and their association with vasospasm severity

Clinical and radiographic characteristics	Percentage	*P*
Age (years), mean±SD	40±14	0.8
GCS, mean±SD	7±4	0.1
Number of angiograms, mean±SD	3±2	
Female	18	0.4
Required craniotomy/ectomy	67	0.3
SAH	100	0.0*
Cisternal	48	
Cortical	100	
Thick	55	
Parenchymal contusion	90	0.9
Intraventricular hemorrhage	29	0.6
Subdural hemorrhage	90	0.6
Epidural hemorrhage	10	0.5
Pseudoaneurysms	10	0.06

*Statistical significance defined as $P < 0.05$. GCS: Glasgow Coma Scale, SD: Standard deviation, SAH: Subarachnoid hemorrhage

There was significantly more severe vasospasm involving the proximal arteries (P = 0.005) and patients with cisternal SAH had significantly more severe vasospasm compared to the ones with only cortical SAH (P = 0.04). There were no significant interactions between the location of SAH and location of vasospasm [Figure 2].

There are no other strong correlations between any of the clinical or radiographic variables presented in Table 1 and the cumulative severity of the PTV noted on the angiograms based on univariate and multivariate analyses. Pearson correlation coefficient and P value from two-tailed t-test are depicted in Table 1 to depict the univariate association of the noted variable and the PTV severity.

Discussion

Arterial vasospasm is a delayed consequence of TBI which can markedly affect the neurologic recovery and adversely impact the functional outcome. The epidemiology and risk factors for this phenomenon are not well understood. The current knowledge of posttraumatic vasospasm is predominantly based on the limited number of observational case series and review articles based on these series which have restricted the definition of vasospasm to elevated TCD velocities. In this study, we focused on the description of clinical and radiographic characteristics of patients with TBI who had angiographic evidence of PTV.

Many of the mentioned case series report an earlier onset and shorter duration of PTV compared to aSAH.[7] However, in this study, the time course is similar to the time course described in the aSAH cohort starting by Day 3 and lasting around 2 weeks with the highest severity within the 2nd week after the ictus.[8] This observed time course is similar to the PTV time course within the wartime case series which was based on angiographic findings.[9] The difference in these observations could be function of the sensitivity of the modality used and the frequency of screening for detection of vasospasm. Most series were only limited to the use of TCD for the detection of PTV. The sensitivity of TCD for the detection of vasospasm has been reported to be as low as 67%.[10] The observed time course is also function of the frequency and duration of screening as well. Martin *et al*. who proposed a shorter duration of this process in their pilot study and later larger series noted that this process does persist up to 2 weeks after the ictus when they continued to follow them with daily TCD monitoring.[2,11]

In this series, craniotomy and craniectomy performed in all patients were early on after presentation to alleviate the mass effect from the contusion/hematoma and associated edema not for mass effect from cytotoxic edema from infarction secondary to delayed cerebral ischemia. Angiographic vasospasm in all patients with surgery were encountered after surgery.

The only factor significantly associated with the severity of vasospasm in this cohort was the presence of cisternal SAH. This is the pattern similar to the aneurysmal SAH. In this study, the highest burden of vasospasm observed involved the proximal arteries. However, interestingly, the distribution of the vasospasm was not associated with the distribution of SAH, which is unlike the pattern observed in aneurysmal SAH.[12] The severity of the vasospasm observed was also not associated with the thickness of SAH unlike aneurysmal SAH.[13] These observations support the fact there are other factors besides the presence of blood and the product of blood breakdown involved underlying mechanism of PTV. The other factors postulated involved in such process are mechanical stretching, inflammation, calcium dysregulation, contractile proteins, products of metabolism, and cortical spreading depression, many of which are also thought to be involved in aSAH.[14]

This is one of the few studies of the natural history and clinical and radiographic characteristics associated

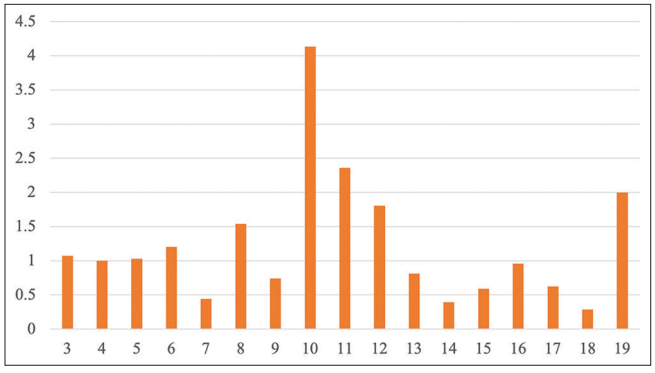

Figure 1: Histogram of the cumulative severity of vasospasm normalized to the treatment days

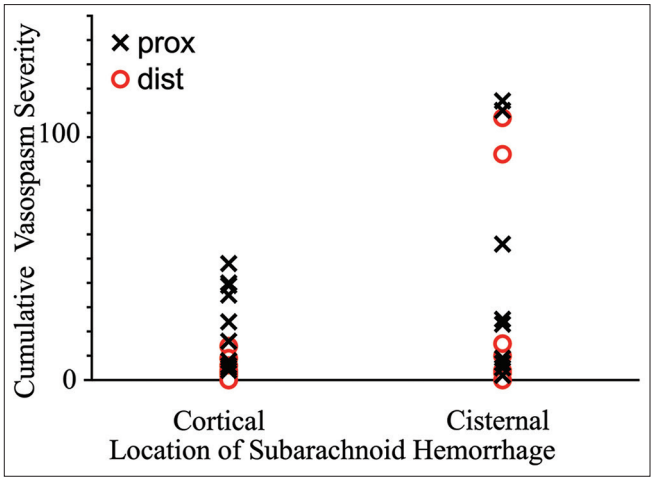

Figure 2: cumulative severity and location of vasospasm during admission based on location of subarachnoid hemorrhage

with the severity of vasospasm in the patients with PTV proven by angiogram. The patients included had a wide age range and wide range of clinical presentations including mild, moderate, and severe TBI. This is a retrospective study with a limited number of patients, general treatment recommendations based on these data are made with reservations. The study population consisted of patients with TBI who have developed angiographic vasospasm and association of each characteristic with the severity of vasospasm and not with the presence of vasospasm.

Distribution of the SAH was abstracted from the presenting CT. The distribution of the hemorrhage and or thickness of hematoma could have changed after craniotomy/craniectomy in the patients that required such intervention which could have further affected the severity of vasospasm, although this matter was not accounted for in the analysis. The patients who had significant vasospasm received intra-arterial verapamil infusion such treatment could have further affected the severity and location of vasospasm on the following days.

Conclusion

Posttraumatic vasospasm can be angiographically detected at posttrauma Day 3–19 in patients with TBI and SAH. Cisternal SAH is associated with more severe vasospasm, although the absence of cisternal SAH does not rule out the occurrence of the vasospasm during the course of treatment.

Author contributions

KK conceived and designed the analysis, collected data, performed the analysis, and wrote the manuscript. LLPM, HS, and NK collected data contributed to the analysis and reviewed the manuscript. RJ, ST, MN, GC, MBB, PV, and GC contributed to the analysis and reviewed the manuscript. VS conceived and designed the analysis and wrote the manuscript.

Ethical approval

This study has been approved by the UCLA IRB (IRB# 18-001238, dated on 11/27/2016).

Declaration of helsinki

We hereby confirm that this study was performed in accordance with the ethical standards details in the declaration of Helsinki.

Declaration of patient consent

The participants were informed about the data from their visit being used for research before inclusion of their data in the study.

Data availability statement

The data that support the findings of this study are available on request from the corresponding author, VS. The data are not publicly available due to potentially sensitive patient information.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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