

A study of vasospasm in traumatic brain injury with subarachnoid hemorrhage.

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ABSTRACT:

Introduction: Cerebral vasospasm (CV) is common following subarachnoid hemorrhage (SAH). However, its impact on neurological outcome, especially in head trauma, has not been yet elucidated. Controversy exists about the true relationship between TBI, SAH and Traumatic Vasospasm. Hence, this study aims to determine the association of vasospasm in TBI with SAH.

Methods: This is Observational cross-sectional study with 124 head injury patients. 31 patients were excluded. Transcranial Doppler ultrasonography (TCD) was conducted on daily bases in all patients with traumatic brain injury (TBI). Vasospasm in the MCA and ACA was defined by a mean Flow velocity (FV) exceeding 120 cm/s and three times the mean FV of the ipsilateral ICA.

Results: Among 93 included patients, 72 (77%) were male and 21 (23%) were female. Mean age was (35+10) years. Mean GCS score was (11+4.1). 61 (66%) patients suffered with severe head injury. Vasospasm was detected in 45 % (42) of the total patients. Vasospasm was severe among 4.3% (4 patients), and moderate among 65.6% (61 patients). Association was found between severity of trauma and the severity of vasospasm in MCA ($r=0.41$ and 0.38 , p value < 0.005) and in ACA ($r=0.25$, p value < 0.005). The presence of SAH is highly correlated with an amplified incidence of vasospasm. The patients who developed vasospasm, 55% (23) had SAH whereas 45% (19) didn't have SAH, the corresponding p value is 0.04 which is significant.

Conclusion: The high incidence of vasospasm is associated with SAH in severe TBI patients. Further studies are recommended to determine predictors of vasospasm in TBI patients with SAH.

Keywords: Transcranial Doppler ultrasonography, Traumatic brain injury, Intracranial vasospasm, Subarachnoid hemorrhage.

Introduction:

Cerebral Vasospasm (CV) following subarachnoid hemorrhage (SAH) is most comprehensively studied topic.¹ With the development in neuro-diagnostic procedures like angiography and ultrasonography, vasospasm can be detected in traumatic brain injury (TBI).^{2,3} Transcranial Doppler ultrasonography (TCD) has appeared as a new reliable and sensitive technique

for diagnosis and estimation of cerebral arterial vasospasm following SAH. Numerous published studies have emphasized the magnitude of this phenomenon in aneurysmal rupture as well as in head injury TBI with an incidence rate of around 1.5 million per year bear a substantial societal load.⁴ It is the leading cause of death in USA.⁵ The primary injury in TBI is irrevocable but avoidable, whereas the secondary injury due to

hypotension, hypoxia, seizure and infection is remediable.^{6,7} Thus by diminishing the secondary injury, mortality and morbidity can be reduced including brain ischemia from vasospasm.^{6,8} Vasospasm is associated with deferred neurological drop in aneurysmal SAH patients. However, the role is less vibrant in TBI. It appears initially for little duration and frequently without significant neurological penalties in TBI patients.⁹ In literature, Substantial controversy exists about the true relationship between TBI, SAH and Traumatic Vasospasm.⁶ Hence this study was planned and conducted. The study aims not only to determine the association of vasospasm in TBI with SAH. But also, to evaluate the other potential risk factors.

Methods:

An Observational cross-sectional study was conducted in department of Neurosurgery, Upendra Devkota Memorial National Institute of Neurological and allied sciences, Bansbari, Kathmandu. A total of 124 patients with head injury were enrolled in hospital during the time period of one year. The exclusion criteria for this study were less than 72 hours' hospital stay, any sedative drug received before, any neurosurgery before TCD, patients who received heparin or warfarin (anticoagulation drugs) and those with penetrating head injury. Ninety-three patients met inclusion criteria. Severity of head injury was categorized into mild, moderate and severe. The GCS score less than 9 were termed as severe, scores 9 to 12 as moderate & 13-15 as mild. For diagnosing cerebral vasospasm among patients, TCD ultrasonography measurements was done daily as per technique described by Aaslid et al.¹⁰ These measurements were performed by the principal investigator using TC2-64 B Doppler ultrasound and 500M Doppler. Vasospasm was defined by mean of flow velocity (FV > 120cm/sec) with a Lindegaard index above three. Lindegaard Ratio is defined as a ration of

mean velocity in the MCA to mean velocity in ipsilateral extracranial internal carotid artery, classified as <3 = hyperemia if <3, Vasospasm if >3 (3-6=Mild, >6=Severe vasospasm) as per Lindegaard et. El.¹¹

The vasospasm was further distinguished into levels as severe, moderate and mild upon FV > 200, 150-200 and <150 respectively. CT scans were performed and reviewed by neuroradiologist for all patients. By using modified Fisher scale, severity of subarachnoid hemorrhage (SAH) was determined. This scale comprises of a) No subarachnoid hemorrhage b) Diffuse deposition of blood or a thin sheet of blood of less than 1 mm thickness & c) A thick layer (0.1 mm) of subarachnoid blood or clots in the subarachnoid cisterns.¹² Ethical clearance was taken from the ethical committee of the hospital and all the information related to the patients were recorded as per clinical practices.

Statistical difference between patient with vasospasm and without was calculated by using "t" test and association was assessed by using chi square test. A p-value of less than 0.05 was considered as statistically significant. All the analysis was carried out by using SPSS (statistical package for social science) software version 23.

Results:

Out of 93 patients, 72 (77%) were male and 21 (23%) female. The age ranged from 12-70 years with a mean (35+10). The main cause of head injury was motor vehicle collision (80.65%) followed by Physical assaults (8.60%) as listed in table 1.

On an average a patient was monitored for 9 days (range= 4-20 days). Mean GCS score was 11/15. Sixty-one (66%) patients suffered with severe head injury, 19 (20.4%) with moderate and 13(14%) with mild respectively. The vasospasm was detected in 45% (42) of the total 93 patients, whereas the severe vasospasm

was detected in 4.3% (4), moderate in 65.6% (61) and mild in 30.1% (28).

Table 1: Causes of head injury

Causes of head injury	Frequency	Percentage
Motor Vehicle collision	75	80.65%
Assaults	8	8.60%
Gunshot Wounds	4	4.30%
Falls	6	6.45%
Total	93	100%

In our study, we found a link between severity of trauma and the manifestation of vasospasm in MCA, ACA and BA during the first-and second-week. There was significant association between severity of trauma and the severity of vasospasm in MCA ($r = 0.41$ and 0.38 , p value < 0.005) and in ACA ($r = 0.25$, p value < 0.005).

The finding for CT establishes SAH in 75% (70) patients with Traumatic Brain Injury (TBI). The presence of SAH is highly correlated with an amplified incidence of vasospasm. The patients who developed vasospasm, 55% (23) had SAH whereas 45% (19) didn't have SAH. The corresponding p value is 0.04 which is significant. The patient with thick layers of subarachnoid blood on CT had a tendency to develop traumatic vasospasm (54.1% vs. 41.4%) with statistically significant p value of 0.035. There was also significant increase in incidence of traumatic vasospasm with epidural hematomas and subdural hematomas. (Table 2).

Discussion:

The most common cause of SAH is trauma.¹³ Incidence of vasospasm in patients with TBI increased with the existence of SAH.¹⁴ Vasospasm is more common between day 4-14 following SAH and it seldom appear prior to day three.^{15,16} In few other studies almost half of the SAH patients with vasospasm develop late cerebral ischemia allied with 20% incidence of morbidity and

mortality.¹⁷ Vasospasm contributes to secondary brain damage by producing ischemia or by adding intensity to existing ischemia, in head injury, apart from this there may be other factors may induce ischemia for example edema, systemic hypoxia, and hypotension. Therefore, vasospasm plays an important role in aggravating previously existing or impending ischemia following TBI with SAH.¹⁸ Newly developed techniques like TCD in recent years, traumatic and post traumatic CV has been recognized frequently. It is reported in 5-59% of the patients following SAH.¹⁸ In our study we include patients with mild, moderate & severe trauma while in previously existing studies only focus was on severe traumatic category ($GCS < 9$).¹⁹ Moreover we report a significant relation between severity of trauma and the appearance of Vasospasm for MCA and ACA (p value < 0.05). These findings are similar to previous published studies but contrast to others.^{20,21} The contrast may appear due to different selection procedure and definition of vasospasm.

Another finding of the study is presence of SAH which is significant and highly correlated with an amplified incidence of vasospasm. Several other studies reported this correlation on CT scan thus providing enough bases for our findings.¹⁶ However, existence of SAH does not lead to vasospasm all the time.^{16,22} Moreover in our study we established a significant relation to the increase in vasospasm with epidural hematomas and subdural hematoma. Patients with these hematomas are more frequently tends to develop vasospasm.

Conclusions: In conclusion, the high incidence of vasospasm is associated with SAH in more severe TBI patients. Epidural hematomas and subdural hematomas were also associated with increased incidence of vasospasm. Further studies are required for the prediction and treatment of vasospasm in TBI patients with SAH.

Table 2: Initial CT findings with or without Traumatic Vasospasm

	Epidural Hematoma	Subdural Hematoma	Intracerebral Hematoma	Intraventricular Hematoma
Vasospasm	10	15	12	5
No Vasospasm	6	11	14	17
P value	0.03	0.03	0.31	0.42

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