A proposed scoring system to screen for vasospasm following aneurysmal subarachnoid hemorrhage

Joseph Erroll V NAVARRO , *Jose C NAVARRO

Abstract

Vasospasm has been known to cause permanent morbidity in 40-70% of survivors who suffered from subarachnoid hemorrhage (SAH). Early recognition of vasospasm is the key to better outcome of SAH. Cerebral angiography is expensive and impractical as a monitoring tool. Transcranial Doppler is operator dependent, and not readily available. The objective of this study is to devise a non invasive tool to screen for cerebral vasospasm following SAH.
The proposed vasospasm score was based on clinical and cranial CT scan features. The features are: hypertension, admission World Federation of Neurosurgeons Score (WFNS), amount of blood in the cisterns and subarachnoid space, intraventricular hemorrhage and hydrocephalus. Thirty six patients with aneurismal SAH were assessed and correlated with the angiogram for vasospasm.

The patients' vasospasm score and their corresponding sensitivity and specificity were: 1 (100%, 0%), 2 (100%, 8%), 3 (100%, 8%), 4 (100%, 8%), 5 (91%, 46%), 6 (74%, 85%), 7 (48%, 85%), 8 (26%, 23%), 9 (3%, 100%), 10 (4%, 100%). A receiver operator characteristic curve was constructed that yielded a cut-off score of 6. The score of 6 was a good trade-off between sensitivity (74%) and specificity (85%).

A clinical vasospasm score was proposed to screen for vasospasm after SAH. A score of 4 to 6 was found to correlate with angiographic vasospasm. Prospective study is required to validate the scoring system.

INTRODUCTION

Subarachnoid hemorrhage (SAH) constitutes 10% of all hemorrhagic strokes.[1] Rebleeding and vasospasm are common occurrences after SAH. Vasospasm remains the leading cause of death and permanent disability after SAH and 40-70% of survivors have permanent neurological deficits.[2] Vasospasm is a potentially treatable condition and the early detection and institution of treatment would yield a better outcome. It usually occurs between 3-7 days after SAH.[3,4] Its maximum severity occurs between 5-14 days and may last up to 3-4 weeks.

Presently, the demonstration of vasospasm by cerebral angiography is accepted as the "gold standard." Angiographic vasospasm can occur in the absence of clinical manifestations in 21-70% of patients with SAH.[5] However, for clinical practice, it is invasive, expensive and impractical as daily monitoring tool.[6] Recently, transcranial Doppler (TCD) is being used to diagnose and monitor cerebral VSP after aneurysmal SAH. TCD has been found to be highly specific[7], is non-invasive, can be performed repeatedly and is economical, hence ideal for screening and monitoring. However, TCD is very operator dependent, not readily available in some institutions and the absence of acoustic window in insonation for some patients imposes further limitations.

For routine clinical practice, there is need for a fast, accurate, reliable, non-invasive and inexpensive screening tool for the diagnosis of vasospasm. Review of current literature did not show any clinical scoring systems that can be utilized as a screening and monitoring tool for vasospasm that has a good correlation to 4-vessel angiography. The purpose of this study was to create a vasospasm score for screening, using established risk factors, amount of subarachnoid blood and compare it with 4-vessel angiography.

METHODS

Review of published literature revealed the different statistical significant risk factors that could be predictive of vasospasm after aneurysmal SAH.[8] These risk factors were used to form the components of the vasospasm score. The risk factors were: hypertension, admission WFNS, CT clot thickness, intraventricular hemorrhage, and hydrocephalus. Cranial CT scan were used to determine the presence or absence of subarachnoid clot, intraventricular hemorrhage and hydrocephalus. Dichotomous factors (hypertension, IVH, and hydrocephalus) were then given numerical values (0 if absent, 1 if present). Continuous variables (admission WFNS, clot thickness) were given values from 0-4 depending on their severity. The values were added to form a total score. The maximum score was 11 (Table 1).

In order to correlate the proposed vasospasm score to angiographic vasospasm, the medical records of all patients admitted and diagnosed to have aneurysmal SAH in University of Santo Tomas Hospital from 2001 to 2004 were reviewed. A total of 75 patients were identified. Inclusion criteria were: (1) SAH confirmed by cranial CT scan or lumbar puncture; (2) Complete clinical charts available; (3) Cranial CT scan available for review and (4) availability of 4-vessel angiograms. All criteria are to be met for inclusion into the study.

The data collected from chart review and cranial CT scan were: Presence of hypertension, admission Glasgow coma scale (GCS), date of ictus, admission WFNS, the amount of clot in the subarachnoid space (diffuse thin – layer of subarachnoid blood < 1mm; diffuse thick – layer of subarachnoid blood > 3mm; local thin – confined to one cistern < 1mm; local thick – confined to one cistern > 3mm), presence of intraventricular hemorrhage, and hydrocephalus. Each patient was graded and given a total score. (Table 1)

Cerebral angiogram was taken as gold standard to determine vasospasm. Cerebral angiographies were performed for all patients from day 2 to day 8 from ictus. Angiographic schedules were largely dictated by the financial capacity of the patients. Still, the majority would fall on the time course of vasospasm. Using the method of Unterberg [9], vessel diameters were measured at the narrowest portions of the A1, A2, M1, M2 and C1-C2 segments using a caliper measured in millimeters and making a comparison with extradural internal carotid artery segments (C4-C5) not exposed to subarachnoid blood. Angiographically confirmed vasospasm was defined as moderate to severe (>30%) narrowing of the diameter of the cerebral vessel lumen.[9]

Table 1: Proposed vasospasm score for screening of vasospasm following aneurysmal subarachnoid hemorrhage

Risk Factors	Score
Hypertension	
No	0
Yes	1
Admission WFNS*	
II	1
III	2
IV	3
V	4
Clot thickness	
Local thin	1
Local thick	2
Diffuse thin	3
Diffuse thick	4
Intraventricular hemorrhage	
Yes	0
No	1
Hydrocephalus	
No	0
Yes	1

*WFNS (World Federation of Neurosurgeons Scoring System)
Grade 0: Intact aneurysm;
Grade I: Glasgow Coma Scale (GCS) 15, major focal deficit (Aphasia, hemiparesis or hemiplegia) absent;
Grade II: GCS 13-14, major focal deficit absent;
Grade III: GCS 13-14, major focal deficit present;
Grade IV: GCS 7-12, major focal deficit present or absent;
Grade V: GCS 3-6, major focal deficit present or absent

The specificity, sensitivity, positive and negative predictive values were computed using a 2x2 table for each score. The Receiver Operator Characteristic (ROC) curve was plotted and the cut-off score was determined. All statistical computations were entered using the Excel software.

RESULTS

A total of 36 patients satisfied the inclusion criteria. There were 14 (39 %) males and 22 (61%) females. The age ranged from 28 to 77 years old with a median of 52±5 years. Most patients (69%) in this study had hypertension. The majority of the cranial CT scan findings exhibited diffuse thick clot (80%). Hydrocephalus and intraventricular hemorrhage were seen in about a quarter of patients. Angiographic vasospasm was seen in the majority of patients (63%). (Table 3)

The number of patients and their respective scores were: 1 (1 patient), 4 (7 patients), 5 (9 patients), 6 (6 patients), 7 (6 patients), 8 (4 patients), 9 (2 patients), 10 (1 patient). Correlation between sensitivity and specificity were inversely proportional (Table 2). Positive predictive value reached 100% for scores 6, 9, and 10. There was an increasing trend in positive predictive values from scores 7 to 10. This signifies an increasing predictive capability as the score reached 6 and above.

The Receiver Operator Characteristic (ROC) curve was plotted using the sensitivity and false positive rate of each score. (Fig 1) A score of 6 with sensitivity of 74% and specificity of 85% gave a good trade-off between the sensitivity and specificity following this ROC curve. The area under the curve was computed at 0.8 or 80%.

Table 2: Proposed vasospasm score with their sensitivity and specificity

SCORE	SENSITIVITY	SPECIFICITY
1	100%	0%
2	100%	8%
3	100%	8%
4	100%	8%
5	91%	46%
6	74%	85%
7	48%	85%
8	26%	23%
9	13%	100%
10	4%	100%

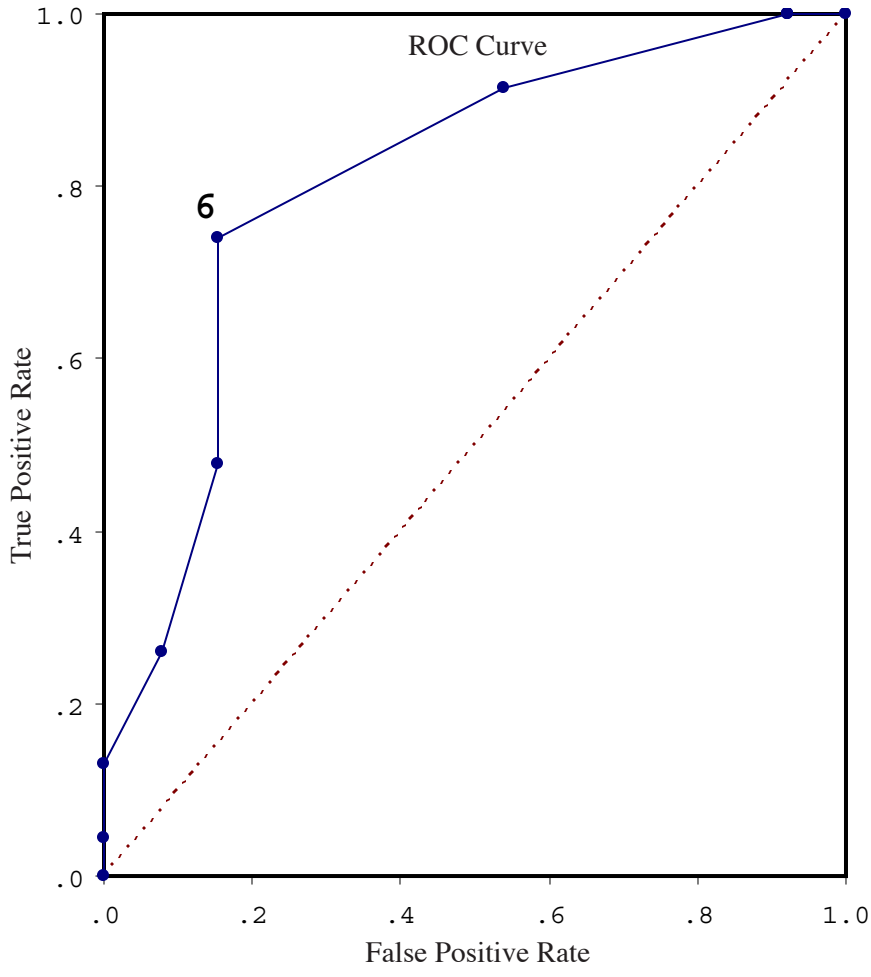
Table 3: Patients' demographic data

Total number of patients	36
Sex distribution Female Male	 22 (61%) 14 (39%)
Hypertension	25 (69%)
Clot thickness	
Diffuse thick	29 (80%)
Diffuse thin	6 (16%)
Local thick	1 (4%)
Local thin	0
Intraventricular hemorrhage	9 (25%)
Hydrocephalus	8 (23%)
Vasospasm	23 (64%)

DISCUSSION

Angiographically detectable vasospasm occurs in about 21 to 70%[10] of patients with SAH. A clinical scoring system that is reasonably sensitive and specific would be cost effective, convenient and thus beneficial to both patients and physicians. Review of literature has not yielded any study that would parallel this work. The convenience of a clinical scoring system would also decrease the use of other non-invasive bedside modalities such as the TCD which has been compared with 4-vessel angiography.[11]

Our study showed that a score of 4 to 6 was found to correlate with angiographic vasospasm. A score of 6 has a good trade off between sensitivity and specificity. At this score, the sensitivity is 76% and specificity is 85%. It also had a 100% positive predictive value and a 43% negative predictive value. This gives confidence that at this score, there is likely to be vasospasm, as well as avoiding a significant number of patients being falsely labeled and thus subjected to other confirmatory tests such as cerebral angiography. Furthermore, other clinical parameters should also be monitored on these patients such as hypertension, deteriorating consciousness, occurrence of focal neurologic deficits and hydrocephalus that may suggest the onset of vasospasm.



Area under the curve (AUC) 0.80 = 80%

Fig. 1: Receiver Operator Curve (ROC)

A limitation of this study is the small number of subjects enrolled. We recommend further prospective studies to validate the utility of this scoring system.

REFERENCES

1. da Costa jr LB, de Morais JV, de Andrade A, . Surgical treatment of intracranial aneurysms: Six year experience in Belo, MG, Brazil. 2004; 62(2-A): 245-9.
2. Goddard AJP, Raju PPJ, Gholkar A. Does the method of treatment of acutely ruptured intracranial aneurysms influence the incidence and duration of cerebral vasospasm and clinical outcome? 2004; 75: 868-72.
3. Bendok BR, Getch CC, Malish TW, Batjer HH. Treatment of aneurysmal subarachnoid hemorrhage. 1998; 18: 521-31.
4. Pasaqualin A. Epidemiology and pathophysiology of cerebral vasospasm following subarachnoid hemorrhage. 1998; 42: 215-6.
5. Ikeda K, Toshiaki I, Hidemichi S, Hitoshi T, Masatoshi N, Kazutaka O. Colloid osmotic pressure (COP) can be good indicator of occurrence of vasopspasm following subarachnoid hemorrhage. 2003; 9(2): CR43-7.
6. Vajkoczy P, Horn P, Thome C, Munch E, Schmiedek P. Regional cerebral blood flow monitoring in the diagnosis of delayed ischemia following aneurysmal subarachnoid hemorrhage. 2003; 98: 1227-34.
7. Lysakowski C, Walder B, Costanza MC, Tramer MR. Transcranial Doppler versus angiography in patients with vasospasm due to a ruptured cerebral aneurysm: A systematic review. 2001; 32: 2292-8.
8. Macdonald RL, Rosengart A, Huo D, Karrison

T. Factors associated with the development of vasospasm after planned surgical treatment of aneurysmal subarachnoid hemorrhage. 2003; 99: 644-52.

9. Unterberg AW, Sakowitz OW, Sarrafzadeh AS, Benndorf G, Lanksch WR. Role of bedside microdialysis in the diagnosis of cerebral vasospasm following aneurysmal subarachnoid hemorrhage. 2001; 94: 740-9.
10. Condette-Auliac S, Bracard S, Anxionnat R, . Vasospasm after subarachnoid hemorrhage: interest in diffusion-weighted MR imaging. 2001; 32: 1818-24.
11. Crissard P, Proust F, Lanlais O. Vasospasm diagnosis; theoretical and real transcranial Doppler sensitivity. 1995; 136: 181-5.