

# Predictors of Cerebral Infarction in Aneurysmal Subarachnoid Hemorrhage

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**Background**—Clinical and radiologic predictors of cerebral infarction occurrence and location after aneurysmal subarachnoid hemorrhage have been seldom studied.

**Methods**—We evaluated all patients admitted to our hospital with aneurysmal subarachnoid hemorrhage between 1998 and 2000. Cerebral infarction was defined as a new hypodensity located in a vascular distribution on computed tomography (CT) scan.

**Results**—Fifty-seven of 143 patients (40%) developed a cerebral infarction. On univariate analysis, occurrence of cerebral infarction was associated with a worse World Federation of Neurological Surgeons grade ( $P=0.01$ ), use of ventriculostomy catheter ( $P=0.01$ ), preoperative vasospasm ( $P=0.03$ ), surgical clipping ( $P=0.02$ ), symptomatic vasospasm ( $P<0.01$ ), and vasospasm on transcranial Doppler ultrasonography (TCD) or repeat angiogram ( $P<0.01$ ). On multivariable analysis, only presence of symptoms ascribed to vasospasm ( $P<0.01$ ) and evidence of vasospasm on TCD or angiogram predicted cerebral infarction ( $P<0.01$ ). TCD and angiogram agreed on the diagnosis of vasospasm in 73% of cases (95% CI, 63% to 81%), but the diagnostic accuracy of this combination of tests was suboptimal for the prediction of cerebral infarction occurrence (sensitivity, 0.72; specificity, 0.68; positive predictive value, 0.67; negative predictive value, 0.72). Location of the cerebral infarction on delayed CT was predicted by neurological symptoms in 74%, by aneurysm location in 77%, and by angiographic vasospasm in 67%.

**Conclusions**—Evidence of vasospasm on TCD and angiogram is predictive of cerebral infarction on CT scan but sensitivity and specificity are suboptimal. Cerebral infarction location cannot be predicted in one quarter to one third of patients by any of the studied clinical or radiological variables. (*Stroke*. 2004;35:1862-1866.)

**Key Words:** cerebral angiography ■ forecasting ■ stroke ■ subarachnoid hemorrhage  
■ ultrasonography, transcranial, Doppler

Poorly understood and insufficiently treated, cerebral vasospasm remains one of the major threats to patients with aneurysmal subarachnoid hemorrhage (SAH).<sup>1,2</sup> The reported incidence of brain infarction resulting from vasospasm varies according to the imaging technique used for diagnosis. Rates of cerebral infarction ranged between 24% and 35% in recent studies using computed tomography (CT) scan,<sup>3,4</sup> but was much higher when relying on magnetic resonance imaging (MRI) to identify ischemic lesions, reaching up to 81% in one of the largest series.<sup>5</sup>

Brain ischemia may occur in patients without apparent vasospasm on transcranial Doppler ultrasonography (TCD) or angiogram. Conversely, documented vasospasm does not always portend neurological deterioration.<sup>6</sup> Most studies assessing the diagnostic accuracy of TCD or angiogram used delayed neurological ischemic deficits (or symptomatic vasospasm) as the primary outcome measure. Much less is

known about the value of these diagnostic tests to predict cerebral infarction on brain imaging.

## Materials and Methods

We reviewed the clinical and radiological information of all patients admitted to the Mayo Clinic with acute aneurysmal SAH between January 1998 and December 2000, and the study was approved by our Institutional Review Board. Patients were included in the study if they had been admitted to our institution within 7 days of SAH onset and had a ruptured aneurysm documented by angiogram. Patients with fusiform, traumatic, and mycotic aneurysms were excluded. We reviewed 153 consecutive patients admitted during the study period. No patients who had denied access to their medical records for research purposes were included in the study. Ten patients were excluded from further analysis because they had declined to provide authorization to use their medical data for research purposes (3 patients), they had died before a follow-up CT scan could be obtained (4 patients), or had incomplete medical or radiological records (3 patients). Thus, the final study population consisted of 143 patients.

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TCD recordings of the mean blood flow velocity (cm/sec) of the major anterior circulation vessels were measured through the trans-temporal window using a 2-MHz hand-held transducer probe. Studies were performed daily or every other day by 2 experienced technicians. Mean arterial velocities >120 cm/sec on the anterior, middle, or posterior cerebral arteries were deemed indicative of vasospasm. Patients typically had 1 angiographic study 3 to 5 days after aneurysm treatment. Repeated angiograms (up to 5 studies in this series) were performed when the diagnosis of vasospasm remained in question or endovascular treatment was being considered. Angiographic vasospasm was considered present when there was unequivocal narrowing of the arterial vessel lumen by visual inspection. It was regarded as severe when the estimated narrowing of the arterial vessel lumen exceeded 50% of the normal caliber. Angiographic vasospasm was defined as focal if it was limited to either the anterior circulation on one side or the posterior circulation on the other side. Conversely, the vasospasm was considered diffuse if it was bilateral or involved vessels in both the anterior and the posterior circulations. When vasospasm was focal, the affected arteries on TCD and angiogram were specifically tabulated.

Upon admission, all patients were treated with intravenous fluids to maintain euvoolemia. Before aneurysm was secured, systolic blood pressure was kept <160 mm Hg in most cases. Selection of treatment with craniotomy and clipping or endovascular coil occlusion resulted from a consensus reached between the treating neurosurgeon and the interventional neuroradiologist after analyzing risks and chances of success of both therapeutic modalities on each particular case. Factors considered during the decision-making process included aneurysmal dome to neck ratio, presence of major or perforating branches off the aneurysm, and surgical or endovascular accessibility. A single interventional neuroradiologist performed all endovascular procedures. Oral nimodipine was administered to all patients for prophylaxis of cerebral vasospasm from the time of admission following a standardized protocol (60 mg every 4 hours for 21 days). Hemodynamic augmentation therapy was rapidly instituted in patients with symptomatic vasospasm. Typically, crystalloids were initially administered to achieve a central venous pressure >8 mm Hg. If symptoms persisted, phenylephrine or a combination of phenylephrine and dopamine were infused to raise the mean arterial pressure by 20% to 25% (or to levels >120 mm Hg if baseline mean arterial pressure was <100 mm Hg). In refractory cases, a pulmonary artery catheter was placed and boluses of crystalloid were administered until increase in pulmonary artery wedge pressure did not result in further increase in systolic volume index (preload optimization). Also, intravenous dobutamine was infused and titrated to keep the cardiac index >3.5 (cardiac index optimization). Endovascular procedures (angioplasty or intra-arterial papaverine) were performed when medical treatment failed to reverse the symptoms. Timing of endovascular intervention was decided by the treating consultants.

Cerebral infarction was defined radiologically as a new hypodensity located in a vascular distribution on the CT scan. Cerebral infarctions possibly related to complications of surgery or angiography (such as large vessel occlusion, perforator vessel occlusion, or arterial rupture or dissection) were noted but excluded from further analysis. Resolving postsurgical hypodensities were considered consistent with brain edema from retraction. All arterial territories involved were recorded to assess the correlation with the vessels showing vasospasm on TCD or angiogram. A single vascular neurologist (A.A.R.), who was blinded at the time to clinical information and functional outcome, reviewed all CT scans and ascribed each cerebral infarction to the corresponding vascular territory using validated arterial territory maps.<sup>7</sup>

Symptomatic vasospasm was defined as documented arterial vasospasm consistent with new neurological deficits presenting within 21 days after onset of SAH and not explained by other causes of neurological deterioration (rebleeding, acute or worsening hydrocephalus, electrolyte disturbances, or hypoxia or seizures). Clinical deficits were classified as focal if the patient had new signs of neurological impairment but remained alert or only drowsy. All focal deficits were ascribed to the vascular territory that could best explain

the symptoms. Global deficit was defined by the presence of stupor or coma (Glasgow coma scale sum score <10).

The primary outcome measure in our study was the occurrence of radiographic cerebral infarction. Secondary outcome measure was the functional status at the time of last follow-up using the modified Rankin scale (mRS) for assessment.

### Statistical Analysis

We used the SAS software (SAS Institute Inc, SAS OnlineDoc Version 8). To evaluate whether there was a univariate association between cerebral infarction and quantitative risk factors, we used Wilcoxon rank sum tests. For categorical risk factors and cerebral infarction, we used  $\chi^2$  tests.

We further evaluated predictors of cerebral infarction in a multivariable setting using 2 distinct logistic regression models. In the first model, we specified a set of 7 independent variables in advance of any analyses. These variables were selected based on their presumed biological and clinical importance. The variables were sex, age, radiographic vasospasm by TCD or angiogram, Fisher grade, World Federation of Neurosurgery (WFNS) grade, SAH treatment, and aneurysm location. The number of variables chosen was limited by the number of cerebral infarction events. To expand on this analysis, we fit a second logistic regression model that included the 6 variables that were univariately associated with cerebral infarction ( $P<0.05$ ).

We tested the sensitivity and specificity of TCD versus angiogram using the McNemar test among patients with cerebral infarction who received both radiographic measures and among the patients without cerebral infarction who were studied by both techniques. All statistical tests were 2-sided.

### Results

The final analysis included 143 patients with acute aneurysmal SAH. Median age was 56 years (range 22 to 88 years) and 99 patients (69%) were women. Fifty percent of patients had history of smoking. After initial resuscitation, 112 patients (78%) were WFNS grade I to III. On admission CT scan, Fisher grade 3 was assigned to 76 patients (53%). The ruptured aneurysm was located in the anterior circulation in 107 patients (75%). Ninety-seven patients (68%) underwent surgical clipping, and 46 patients (32%) were treated with endovascular coil embolization. Median time to aneurysm treatment was 2 days (range 1 to 18 days). Every patient had at least 1 CT scan after surgery or endovascular treatment and at least one more CT scan after the diagnosis of vasospasm was made. Considering all the patients, mean time from SAH onset to last CT scan during acute hospitalization was 12 days (range 5 to 32 days). Twenty-three patients (16%) underwent endovascular treatment for vasospasm (angioplasty with or without intra-arterial papaverine in 11 patients and 12 intra-arterial papaverine only), with 8 patients requiring more than one treatment session. Only 1 case of cerebral infarction was attributed to a complication from endovascular treatment of vasospasm; this infarction was not included in the analysis.

Radiological cerebral infarctions occurred in 57 patients (40% of the study population). The distribution of demographic, clinical, and radiological characteristics according to cerebral infarction occurrence is shown in Table 1. On univariate analysis, clinical grade by WFNS ( $P=0.01$ ), use of ventriculostomy catheter ( $P=0.01$ ), preoperative vasospasm ( $P=0.03$ ), surgical clipping ( $P=0.02$ ), symptomatic vasospasm ( $P<0.01$ ), and evidence of vasospasm by TCD or angiogram ( $P<0.01$ ) were significantly associated with the occurrence of cerebral infarction. Using 2 different multiple

**TABLE 1. Distribution of Demographic, Clinical, and Radiological Characteristics (Continuous Variables) According to Cerebral Infarction Occurrence in 143 Patients With Acute Aneurysmal SAH**

Variable	N	Cerebral Infarction Absent	Cerebral Infarction Present	P
Total population, n (%)	143	86 (60)	57 (40)	
Age, median (range)	143	56 y (22–87)	56 y (33–88)	0.96
Aneurysm size, median (range)	135	7 mm (2–25)	8 mm (3–26)	0.14
Time to treatment, median (range)	142	2 d (1–18)	2 d (1–14)	0.27

logistic regression models, only symptomatic vasospasm and evidence of vasospasm by TCD or angiogram remained predictive of cerebral infarction. Details of the multivariate model are displayed in Tables 2a and 2b.

TCD indicated presence of vasospasm in 85 of 120 of patients (71%) on whom this study was serially performed. Meanwhile, vasospasm was noted in 60 of 113 patients (53%) who had follow-up angiograms. The rate of agreement between TCD and angiogram in the detection of vasospasm in our population was 73% (95% CI, 63% to 81%). The angiogram was marginally more specific (63% versus 44%,  $P=0.083$ ) but less sensitive than TCD (75% versus 90%,  $P<0.01$ ) in predicting the occurrence of cerebral infarction. In fact, the odds ratio (OR) estimate for the occurrence of cerebral infarction was higher in patients with vasospasm detected by TCD (OR, 7.4; 95% CI, 2.6 to 21) than in those with vasospasm detected by an angiogram (OR, 5.1; 95% CI, 2.2 to 11.7), although with very wide CIs. We then studied the effects of combining the results of both tests on their diagnostic accuracy for the prediction of cerebral infarction. When both studies showed vasospasm, the positive predictive value of this diagnostic combination for the occurrence of cerebral infarction was 67%. Conversely, when both studies indicated absence of vasospasm, the negative predictive value of this combination was 72% (Table 3).

The location of vasospasm on TCD and on angiogram accurately predicted cerebral infarction location on CT scan in 82% and 67% of cases, respectively. Thus, there was no evidence of angiographic vasospasm in the arterial tree corresponding to the area of radiological cerebral infarction in 33% of patients with cerebral infarction. Aneurysm location corresponded to the area of cerebral infarction in 77% of

patients. Focal symptoms predicted cerebral infarction location in 74% of patients.

Cerebral infarction was predictive of worse functional outcome. The median mRS score was 1 among patients who did not have a cerebral infarction and 3 among those patients who did ( $P<0.01$ ). Seventy percent of patients with cerebral infarction had poor functional outcome (mRS  $>2$ ) compared with 17% in the group without cerebral infarction. The mortality rate at last follow-up was 23% in the group of patients with cerebral infarction versus only 5% among patients without cerebral infarction ( $P<0.01$ ). Median follow-up among survivors was 12 months (range 1 to 36 months). Median length of stay was significantly longer for the group of patients with cerebral infarction ( $17.5\pm 8.6$  days versus  $14\pm 10.8$  days for the group without cerebral infarction;  $P<0.01$ ).

## Discussion

In our referred hospital population, almost 80% of patients with aneurysmal SAH presented with a good clinical grade, but more than half had a CT pattern associated with a high probability of developing vasospasm (Fisher grade 3). TCD indicated vasospasm in 71% of patients and angiography in 53%. The incidence of brain infarction on CT scan was 40%. Symptomatic vasospasm and evidence of vasospasm by TCD or angiogram predicted cerebral infarction occurrence on multivariable logistic regression analysis. TCD and angiography were concordant in the detection of vasospasm in nearly three quarters of patients. Whereas angiography was more specific, TCD was more sensitive in predicting the occurrence of cerebral infarction. When the results were concordant, the combination of both tests only predicted the

**TABLE 2A. Predictors of Cerebral Infarction Using a Prespecified Multivariate Logistic Regression Model in 143 Patients With Acute Aneurysmal SAH**

Variable	Odds Ratio*	95% CI*	P*
Male vs female	0.51	0.21, 1.24	0.14
Age (5-year increase)	1.03	0.89, 1.20	0.70
TCD or angiogram vasospasm vs neither	8.28	2.57, 26.74	$<0.001$
Fisher grade (1-unit increase)	1.08	0.65, 1.81	0.76
WFNS (1-unit increase)	1.26	0.91, 1.74	0.17
Clip vs coil treatment	2.02	0.79, 5.10	0.14
Aneurysm location (posterior vs anterior)	1.06	0.40, 2.84	0.91

\*Based on having all other variables from this table in the model.

**TABLE 2B. Predictors of Cerebral Infarction Using Multivariate Logistic Regression Model With Statistically Significant Variables on Univariate Analysis**

Variable	Odds Ratio*	95% CI*	P*
TCD or angiogram vasospasm vs neither	4.62	1.13, 18.88	0.033
WFNS (1-unit increase)	0.62	0.38, 1.01	0.054
Clip vs coil treatment	1.25	0.39, 4.03	0.71
Preoperative vasospasm	1.53	0.40, 5.89	0.54
EVD†	1.44	0.42, 4.99	0.56
Symptomatic	47.50	12.33, 183.06	$<0.0001$

\*Based on having all other variables from this table in the model.

†EVD indicates extraventricular drain.

**TABLE 3. Sensitivity, Specificity, Positive Predictive Value, and Negative Predictive Value of Vasospasm as a Diagnostic Predictor of Cerebral Infarction in 143 Patients With Acute Aneurysmal SAH**

	N	Spasm	Sensitivity	Specificity	PPV*	NPV†
TCD	120	85	0.90	0.44	0.55	0.86
Angiogram	113	60	0.75	0.63	0.60	0.77
Either TCD or angiogram	137	96	0.93	0.45	0.52	0.90
Both TCD and angiogram	96	49	0.72	0.68	0.67	0.72

\*PPV indicates proportion of patients with vasospasm detected who had a cerebral infarction; NPV, proportion of patients with no vasospasm detected who did not have a cerebral infarction.

occurrence of cerebral infarction in approximately two thirds of cases. Similarly, cerebral infarction location was predicted more sensitively by the location of vasospasm on TCD. In fact, one third of the patients with brain infarction had no vasospasm documented by angiography in the vessel perfusing the area of ischemia. The presence of brain infarction on a CT scan correlated with increased mortality and worse functional outcome.

In this study, treatment modality (surgical clipping versus endovascular coiling) was not independently associated with the occurrence of cerebral infarction in multivariable analysis, despite a trend suggesting increased risk with surgical clipping. The confidence intervals were very wide, suggesting that significance may have been limited by a low patient number. In a larger study, we found surgical clipping to be associated with increased risk of stroke compared with endovascular coil embolization in patients with a good clinical grade.<sup>8</sup> In the current series, the Fisher grade did not correlate with the occurrence of cerebral infarction in either univariate or multivariable analysis. A previous study has questioned the predictive value of the Fisher grade.<sup>9</sup> Software-based techniques to quantify SAH on admission CT scan may be of greater value in predicting vasospasm.<sup>10</sup>

Although focal symptomatic vasospasm may often be recognized by the development of localizing signs, the more common diffuse vasospasm may manifest more insidiously with a declining level of consciousness. As a consequence, defining symptomatic vasospasm is frequently challenging, both in research studies and at the bedside. For that reason, we chose brain infarction documented by CT scan as the main outcome measure of our study, as opposed to the more commonly used symptomatic vasospasm.

Studies using MRI have revealed that delayed ischemic lesions after SAH are usually bilateral and multifocal, often involve the frontal lobes, and are not uncommonly asymptomatic.<sup>5,11</sup> It has been postulated that the lack of correlation of some of these lesions with angiographically-documented arterial narrowing may be because angiography does not have the sensitivity to visualize small-vessel spasm.<sup>11</sup> Small-vessel spasm could also explain the dissociation between evidence of vasospasm by TCD or angiography and the presence of cognitive impairment after SAH.<sup>12</sup> In fact, ischemic lesions, but not symptomatic vasospasm, have been found to be predictive of cognitive dysfunction in these patients.

Microembolism could be an alternative explanation for the occurrence of brain ischemic damage in the absence of vasospasm. Microembolic signals are frequently detected by

TCD monitoring in patients with SAH with and without vasospasm;<sup>13</sup> however, the clinical relevance of this finding remains to be established.

In general, TCD fares rather modestly in studies that used angiography as the “gold standard” for the definition of vasospasm.<sup>7,14</sup> Meanwhile, when clinical deterioration was used to define vasospasm, TCD was found to be as sensitive as cerebral angiography.<sup>15</sup> Against this background, our results offer new information that may enhance our understanding of the value of these 2 diagnostic techniques. We found that, in our experience, TCD was actually more sensitive but less specific than angiography in predicting the occurrence of cerebral infarction on CT scan. TCD and angiography disagreed in slightly more than 1 in 4 cases, and the combination of both tests still failed to provide high diagnostic accuracy.

In this study, the topography of the cerebral infarction on a CT scan was not predicted reliably by the location of documented vasospasm (by TCD or angiography), the location of the ruptured aneurysm, or the clinical manifestations of the patient. Therefore, cerebral ischemia was not infrequently subclinical or clinically subtle, and it may have been caused by vasospasm that could not be detected by our standard diagnostic techniques.

We acknowledge that our study design has limitations. Although we tried to restrict our definition of cerebral infarction on a CT scan to focus on lesions likely caused by vasospasm, we cannot exclude that some of the ischemic lesions may have been caused by mechanisms other than vasospasm (eg, perforator vessel occlusion unnoticed at the time of surgical clipping or coil embolization, or delayed consequences of a dissection provoked during catheterization but undetected on an angiogram). Our definition of ultrasonographic vasospasm did not include measurement of the Lindegaard ratio; this measurement could have enhanced the specificity of TCD results by helping differentiate true vasospasm from hyperemia.<sup>16</sup> Conversely, the diagnostic yield of angiography may have been limited by inappropriate timing or insufficient repetition of angiograms in patients who developed delayed ischemic damage. Also, it is conceivable that less conservative criteria for the definition of angiographic vasospasm could have enhanced the sensitivity of the test. Metallic artifacts from clips and coils used to secure the ruptured aneurysm may have interfered with the precise interpretation of findings in the CT scan, especially in the posterior fossa. Nevertheless, we consider that none of these potential limitations negates the validity of our results.



We conclude that evidence of vasospasm on TCD and angiogram is predictive of cerebral infarction on a CT scan. However, the sensitivity and specificity of these tests in predicting the occurrence of cerebral infarction are suboptimal.

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