Patterns of Cerebral Infarction in Aneurysmal Subarachnoid Hemorrhage

Alejandro A. Rabinstein, MD; Stephen Weigand, MS; John L.D. Atkinson, MD; Eelco F.M. Wijdicks, MD

Background and Purpose—The aim of this study was to analyze the distribution patterns of delayed cerebral ischemia after subarachnoid hemorrhage (SAH) and the factors that determine their occurrence.

Methods—We analyzed clinical and radiological data of 143 consecutive patients with aneurysmal SAH. Computed tomography scan revision was blinded to clinical information. Superficial infarctions were defined as territorial lesions with cortical involvement. Perioperative infarctions were excluded.

Results—Fifty-six patients (39%) had cerebral infarctions. They were unilateral in 34 patients (61%) and involved a single territory in 29 (52%). Location was cortical in 34 patients (61%), deep in 10 (18%), and combined cortical and deep in 12 (21%). Single infarctions were cortical in 23 of 28 cases (79%). Deep territory ischemia was more common with multiple lesions (16/28, 57% versus 6/29 with single lesions, 21%; P<0.01). Single infarctions occurred frequently in the territory of the ruptured aneurysm (22/28 patients; 79%), whereas multiple infarctions were often distant to the site of rupture (21/28 cases, 75%). History of diabetes (P=0.05), early hydrocephalus (P=0.05), and requirement of external ventricular drainage (P=0.02) were associated with the occurrence of multiple infarctions on univariate analysis. On multivariable analysis, this association only remained significant for the requirement of external ventricular drainage.

Conclusion—The 2 most common patterns of delayed cerebral ischemia after aneurysmal SAH are single cortical infarction, typically near the ruptured aneurysm, and multiple widespread lesions including subcortical locations and often unrelated to the site of aneurysm rupture. These 2 patterns may represent different pathophysiological mechanisms or different degrees of severity of the same vascular process. (Stroke. 2005;36:992-997.)

Key Words: computed tomography ■ stroke ■ subarachnoid hemorrhage

Cerebral vasospasm remains a leading cause of disability after aneurysmal subarachnoid hemorrhage (SAH). Rates of cerebral infarction caused by vasospasm range between 24% and 35% when defined using computed tomography (CT) scan and may be as high as 81% when magnetic resonance imaging (MRI) is used for diagnosis. Yet, the pattern of ischemic damage caused by vasospasm has not been thoroughly studied.

Early descriptions of the distribution of ischemic lesions caused by vasospasm based on CT scans available at the time highlighted the multiplicity and cortical involvement of the infarctions. More recent studies, particularly those using MRI, report the frequent occurrence of subcortical, often apparently asymptomatic, ischemic areas. The factors that predict the development of multiple widespread lesions in these patients have not been defined.

The goal of this study was to assess the radiological patterns of the cerebral infarctions caused by vasospasm and to define what clinical variables play a role in determining the distribution and extent of the ischemic damage in a large cohort of consecutive patients with aneurysmal SAH.

Materials and Methods

We reviewed clinical and radiological information of all patients admitted to Mayo Clinic with acute aneurysmal SAH between January 1998 and December 2000 within 7 days of SAH onset. Patients with fusiform, traumatic, and mycotic aneurysms were excluded. We reviewed 153 consecutive patients admitted during the study period. Ten patients were excluded from further analysis because they had declined to provide authorization to use their medical data for research purposes (3 patients), had died before a follow-up CT scan could be obtained (4 patients), or had incomplete records (3 patients). Consequently, 143 patients comprised the final study population. Our institutional review board approved the research protocol and monitored the investigation.

Collected data included general demographic information, history of hypertension, previous stroke, smoking, or diabetes mellitus, onset of SAH, World Federation of Neurological Surgeons clinical grade at presentation, radiological Fisher grade, location and size of the aneurysm, presence of early hydrocephalus, use of ventriculostomy catheter, treatment modality chosen to secure the aneurysm (surgical clipping or endovascular coil occlusion), and timing of treatment in relation to SAH onset.

We recorded the results of serial transcranial Doppler (TCD) studies and angiograms. TCD recordings of the mean blood flow velocity (cm/sec) of the major anterior circulation vessels were measured through the transtemporal window using a 2-MHz hand-
held transducer probe. Studies were performed by 2 experienced technicians daily or every other day. Mean arterial velocities >120 cm/sec on the anterior, middle, or posterior cerebral arteries were deemed indicative of vasospasm. Patients typically had 1 angiogram 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 entertained. 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. Conversely, vasospasm was considered diffuse if it was bilateral or involved vessels in both anterior and posterior circulations. When vasospasm was focal, the affected arteries on TCD and angiogram were tabulated.

Cerebral infarction was defined radiologically as a new hypodensity on CT scan located in a vascular distribution. Cerebral infarctions possibly related to complications of surgery or angiography (such as large-vessel occlusion, perforator vessel occlusion, arterial rupture, or dissection) were excluded from the analysis. Resolving postsurgical hypodensities were considered consistent with brain edema from retraction. Infarcts were grouped into the following categories: unilateral or bilateral, single or multiple arterial territories, and cortical or deep location. 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.8,9

Symptomatic vasospasm was defined as documented arterial vasospasm consistent with new neurological deficits presenting within 21 days after the onset of SAH and not explained by other causes of neurological deterioration (rebleeding, acute or worsening hydrocephalus, electrolyte disturbances, 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 for assessment.

Statistical Analysis
Data were recorded directly into a Microsoft Excel spreadsheet. We used the SAS version 8 software (SAS OnlineDoc Version 8; SAS Institute Inc) to check the data for internal consistency, to identify invalid or illogical values, and to perform all analyses.

We used nonparametric tests for continuous variables. When the outcome of interest had 2 categories, we used Wilcoxon rank sum tests, and when it had 3 categories we used Kruskal–Wallis tests instead. For categorical variables, we used χ² tests when the sample size was sufficiently large and Fisher exact tests when analyzing smaller samples. All tests were 2-sided. Level of significance was established at P<0.05.

We used univariate logistic regression models to assess the effect of independent variables on the endpoints of interest. These endpoints included having bilateral versus unilateral cerebral infarctions, multiple versus single infarctions, involvement of multiple vascular territories versus a single territory, and purely cortical ischemia versus involvement of any deep location. All variables that were significant at the 0.10 level in the univariate analysis were included in a logistic model and backward stepwise selection was used to remove terms 1 by 1 until those remaining were significant at the 0.05 level. Analyses were performed using R (R Development Core Team, 2004). R is a language and environment for statistical computing (R Foundation for Statistical Computing) and the Design package (http://biostat.mc.vanderbilt.edu/wiki/bin/view/Main/Design).

Results
One hundred forty-two patients with acute aneurysmal SAH were included in the final analysis. Median age was 56 years (range, 22 to 88 years), and 98 (69%) were women. Fifty percent of patients had history of smoking. After initial resuscitation, 111 patients (78%) had a good clinical grade (World Federation of Neurological Surgeons grade I to III). Radiological Fisher grade 3 was assigned to 75 patients (53%) on the basis of the admission CT scan. The ruptured aneurysm was located in the anterior circulation in 106 patients (75%), with the most frequent location being the anterior communicating artery (34%). Ninety-six 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 1 more CT scan after the diagnosis of vasospasm was made. Mean time from SAH onset to last CT scan during acute hospitalization was 12 days (range, 5 to 32 days) across the whole population. Symptoms attributed to vasospasm occurred in 70 patients (49%). 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 >1 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 56 patients (39% of the study population). Cerebral infarctions were unilateral in 34 patients (61%) and involved a single arterial territory in 29 cases (52%). Multiple infarctions occurred in 28 patients (50%). Location was solely cortical in 34 patients (61%), solely deep in 10 (18%), and combined cortical and deep in 12 (21%). When a single infarction was present, it was cortical in 22 of 28 cases (79%). Deep territory ischemia was significantly more frequent in patients with multiple lesions (16/28, 57% versus 6/28 with single lesions, 21%; P<0.01). Whereas patients with single infarctions frequently had ischemia in the territory of the ruptured aneurysm (22/28 patients; 79%), ischemic lesions distant to the ruptured aneurysm were commonly found among patients with multiple infarctions (21/28 cases, 75%) (P<0.01 for the difference in ischemia limited to the arterial territory corresponding to the ruptured aneurysm).

The clinical manifestations of the various patterns of infarction are summarized in Table 1. Symptomatic vasospasm was documented in 50 of 56 patients (89%) with cerebral infarction. Asymptomatic presentations were significantly more common in patients with deep infarctions only (40% versus 9% of those with cortical involvement; P=0.03). In patients with a single cortical infarct, TCD showed focal vasospasm in 55% of cases and angiogram in 56%. Diffuse vasospasm was diagnosed by TCD in 50% and by angiogram in 65% of patients with multiple infarctions. Endovascular measures to treat vasospasm were applied in 18 of 56 patients (32%) in whom cerebral infarction developed. The pattern of brain ischemia in this subgroup of patients was single cortical lesion in 9 cases, multiple cortical infarctions in 3, multiple

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TABLE 1. Clinical Presentations of Brain Infarctions According to Topographic Pattern of Delayed Ischemia in Patients With Aneurysmal Subarachnoid Hemorrhage

<table>
<thead>
<tr>
<th>Pattern of Brain Infarction</th>
<th>Clinical Presentation</th>
<th>Asymptomatic</th>
<th>Focal Deficits*</th>
<th>Global or Nonlocalizing†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single cortical</td>
<td></td>
<td>3</td>
<td>15</td>
<td>4</td>
</tr>
<tr>
<td>Single deep</td>
<td></td>
<td>3</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Multiple cortical</td>
<td></td>
<td>1</td>
<td>6</td>
<td>5</td>
</tr>
<tr>
<td>Multiple deep</td>
<td></td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Multiple combined</td>
<td></td>
<td>0</td>
<td>7</td>
<td>5</td>
</tr>
</tbody>
</table>

*Focal deficits were considered present when the patient had documented hemiparesis, aphasia, or neglect.
†Global or nonlocalizing presentations included confusion or decreased level of consciousness without associated focal deficits.

depth in 1, and a combination of deep and cortical infarctions in 6. Table 2 provides further details on the results of TCDs and angiograms according to pattern of brain infarction. The Figure displays the distribution of ischemic lesions in all the patients included in this series.

The analysis of the predictors of cerebral infarction in general for this cohort was presented in a previous article. Patients with multiple cerebral infarctions were slightly older than those with single ischemic lesions (mean age, 61 versus 55 years) but the difference did not reach statistical significance (P=0.16). History of diabetes (P=0.05), early hydrocephalus (P=0.05), and requirement of external ventricular drainage (P=0.02) were statistically associated with the occurrence of multiple infarctions on univariate analysis. On multivariable analysis, the only requirement of external ventricular drainage was independently associated with the outcome of multiple infarctions. Although multiple infarctions were also more common in patients with poor clinical grade (67% versus 45% among patients with good clinical grade) and those with Fisher grade 3 (60% versus 36% among those with other radiological grades), neither World Federation of Neurological Surgeons grades nor Fisher grades were statistically predictive of the development of multiple infarctions. Table 3 shows the odds ratios of the various clinical and radiological variables included in the analysis.

Poor outcome was more common among patients who had cerebral infarction (modified Rankin score >2 in 70% versus 17% among patients without infarction; P<0.01). 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 length of stay was significantly longer for the group of patients with cerebral infarction (17.5±8.6 days versus 14±10.8 days for the group without cerebral infarction; P<0.01). Compared with patients with single cerebral infarction, those who had multiple infarctions tended to have worse functional outcome (median modified Rankin score 4.0 versus 3.0) and longer hospital stays (median of 9 days versus 6 days), although these differences failed to achieve statistical significance.

Discussion

In this large series of consecutive patients with aneurysmal SAH, we identified 2 common patterns of cerebral infarction from delayed cerebral ischemia: single cortical infarction typically in the proximity of the ruptured aneurysm and multiple widespread lesions often involving subcortical regions and frequently distant from the site of aneurysm rupture. Deep infarctions were more often clinically silent in the acute phase. The focal or diffuse distribution of vasospasm by TCD or angiogram failed to predict reliably the subsequent pattern of brain infarction.

The frequent occurrence of multifocal ischemia in patients with vasospasm after SAH was highlighted in a seminal article by Hijdra et al. This study assessed 57 patients with delayed ischemic damage who were studied with CT scan or necropsy. A single arterial territory was involved in 19 of 47 patients (40%) with hypodensities on CT scan; the rest were found to have multivascular or diffuse ischemia. Furthermore, only 1 of the 18 necropsy cases exhibited changes restricted to a single vascular region. The majority of lesions were cortical, either in territorial or watershed distribution, and only 2 patients with lesions of deep brain structures were noted. The authors concluded that vasospasm is most often a multivascular or diffuse process.

TABLE 2. Results of Transcranial Doppler and Angiographic Studies in Patients With Delayed Ischemic Damage After Aneurysmal Subarachnoid Hemorrhage

<table>
<thead>
<tr>
<th>Pattern of Infarction</th>
<th>Diagnostic Study</th>
<th>Focal Vasospasm</th>
<th>Findings Diffuse Vasospasm</th>
<th>Negative</th>
<th>Insufficient Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single cortical</td>
<td>TCD</td>
<td>11</td>
<td>9</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Single deep</td>
<td>Angiogram</td>
<td>9</td>
<td>4</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Multiple cortical</td>
<td>TCD</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Multiple deep</td>
<td>Angiogram</td>
<td>2</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Multiple combined (cortical+deep)</td>
<td>TCD</td>
<td>3</td>
<td>5</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Angiogram</td>
<td>1</td>
<td>3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0</td>
<td>2</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>4</td>
<td>5</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>6</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
Our results confirm but also expand the findings described by Hijdra et al. We found the same proportion of patients with single cortical lesions (23/57 patients; 40%), but the number of cases with deep infarctions was much larger. The differences may be related to improvement in the quality of CT imaging, as suggested by the frequent finding of subcortical ischemic lesions when diffusion weighted imaging is used. The high prevalence of very extensive ischemic areas in the series of Hijdra et al may also be explained by the common use of antifibrinolytics and fluid restriction at the time of the study.

Studies using MRI have revealed that delayed ischemic lesions after SAH are usually bilateral and multifocal. These lesions often involve the frontal lobes, and they are not uncommonly considered asymptomatic, at least during the acute phase. The prevalence of lesions considered ischemic was much greater on MRI performed after a mean interval of 3 years after SAH when compared with the finding of hypodense areas on CT scan at 3 months (81% on MRI versus 57% on CT scan). In a series of patients studied with serial MRI during the first month after SAH, 34% of patients were found to have symptomatic infarction and 23% had asymptomatic infarction. Asymptomatic lesions tended to involve the territory of deep perforating arteries. Therefore, MRI studies offered findings that are in close agreement with our main results.

The occurrence of deep subcortical lesions and the lack of a consistent correlation with TCD and angiographic results seem to point to a mechanism other than the well-defined vasospasm at the level of the circle of Willis. The possibility of small-vessel spasm is supported by experimental data demonstrating endothelial dysfunction, and histopathological evidence of luminal narrowing in intraparenchymal small arteries in an animal model of SAH. Moreover, preliminary data from human studies indicate that autoregulatory responses are impaired after SAH and microcirculatory changes manifested by prolonged cerebral circulation time may lead to decreased regional cerebral blood flow. Less researched but equally intriguing is the alternative that microembolism could contribute to the occurrence of small infarcts in patients with SAH.

In the present study, diabetic patients and those with acute hydrocephalus requiring external ventricular drainage were at higher risk for multiple brain infarctions on univariate analysis. Older age has been previously associated with the occurrence of vasospasm-induced cerebral infarction, despite the fact that younger patients tend to have more severe vasospasm. However, we did not find a strong association between multiplicity of ischemic lesions and age. Hydrocephalus has been reported to predict occurrence of brain ischemia. Requirement of external ventricular drainage, the only variable independently associated with multiple cerebral in-
Vasospasm After Subarachnoid Hemorrhage

**TABLE 3. Odds Ratios of Clinical and Radiological Variables in the Prediction of Different Patterns of Cerebral Infarction Ascribed to Vasospasm After Subarachnoid Hemorrhage**

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>Bilateral vs Unilateral</th>
<th>Multiple CI vs Single</th>
<th>Multiple Vascular Territories vs Single</th>
<th>Any Deep vs Surface Only</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex, male vs female</td>
<td>1.5 (0.46, 5.0)</td>
<td>1.2 (0.37, 3.9)</td>
<td>0.92 (0.28, 3.0)</td>
<td>1.0 (0.31, 3.5)</td>
</tr>
<tr>
<td>Age, per 10-y increase</td>
<td>1.2 (0.8, 1.9)</td>
<td>1.4 (0.88, 2.1)</td>
<td>1.3 (0.84, 2.0)</td>
<td>1.2 (0.78, 1.8)</td>
</tr>
<tr>
<td>Previous stroke</td>
<td>0.27*</td>
<td>2.1 (0.18, 24.0)</td>
<td>2.2 (0.19, 26)</td>
<td>0.76 (0.065, 9)</td>
</tr>
<tr>
<td>Smoking</td>
<td>1.1 (0.36, 3.1)</td>
<td>0.56 (0.19, 1.6)</td>
<td>0.49 (0.17, 1.4)</td>
<td>1.1 (0.36, 3.1)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.3 (0.45, 4.0)</td>
<td>1.6 (0.63, 4.6)</td>
<td>1.8 (0.6, 5.2)</td>
<td>1.3 (0.45, 4.0)</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>5.2 (0.51, 54)</td>
<td>0.11*</td>
<td>0.048*</td>
<td>1.6 (0.21, 12)</td>
</tr>
<tr>
<td>Preoperative hydrocephalus</td>
<td>2.7 (0.88, 8.4)</td>
<td>3.3 (1.1, 9.6)</td>
<td>2.8 (0.95, 8.4)</td>
<td>2.0 (0.66, 5.9)</td>
</tr>
<tr>
<td>Preoperative vasospasm</td>
<td>1.1 (0.31, 4.2)</td>
<td>1.0 (0.28, 3.6)</td>
<td>1.1 (0.31, 3.9)</td>
<td>0.72 (0.19, 2.8)</td>
</tr>
<tr>
<td>WFNS, poor grade (V to V) vs good grade (I to III)</td>
<td>1.8 (0.53, 6.1)</td>
<td>2.2 (0.62, 7.6)</td>
<td>1.6 (0.48, 5.5)</td>
<td>0.82 (0.23, 2.9)</td>
</tr>
<tr>
<td>Fisher grade, per 1-grade increase</td>
<td>1.1 (0.53, 2.3)</td>
<td>0.82 (0.4, 1.7)</td>
<td>0.8 (0.39, 1.7)</td>
<td>0.84 (0.41, 1.7)</td>
</tr>
<tr>
<td>Aneurysm location, posterior vs anterior</td>
<td>1.0 (0.26, 4.2)</td>
<td>2.8 (0.64, 12)</td>
<td>1.8 (0.44, 7.2)</td>
<td>1.7 (0.43, 6.8)</td>
</tr>
<tr>
<td>Time to treatment, per day from onset of hemorrhage</td>
<td>0.83 (0.41, 1.7)</td>
<td>0.72 (0.36, 1.4)</td>
<td>0.73 (0.37, 1.5)</td>
<td>0.63 (0.3, 1.3)</td>
</tr>
<tr>
<td>Coiling vs clipping</td>
<td>1.4 (0.36, 5.2)</td>
<td>3.3 (0.78, 14)</td>
<td>0.88 (2.2, 0.56)</td>
<td>0.51 (0.12, 2.2)</td>
</tr>
<tr>
<td>Requirement of external ventricular drainage</td>
<td>3.2 (1.0, 9.8)</td>
<td>4.5 (1.5, 14)</td>
<td>3.8 (1.2, 11)</td>
<td>1.3 (0.43, 3.7)</td>
</tr>
<tr>
<td>Vasospasm by TCD or angiogram</td>
<td>0.7 (0.09, 5.1)</td>
<td>0.11*</td>
<td>0.01*</td>
<td>0.67 (0.087, 5.1)</td>
</tr>
<tr>
<td>Symptomatic vasospasm</td>
<td>2.0 (0.20, 21)</td>
<td>3.2 (0.32, 33)</td>
<td>3 (0.29, 31)</td>
<td>0.19 (0.019, 2.0)</td>
</tr>
</tbody>
</table>

All values shown as odds ratio (95% confidence interval), P value. Unless otherwise indicated, the odds ratio presented is for those with vs those without the variable under analysis. TCD indicates transcranial Doppler; WFNS, World Federation of Neurological Surgeons.

*P value for Fisher exact test. Univariate odds ratio estimates not available because of a zero count in the table of outcome vs predictor.

Fractional on multivariable analysis, may reflect more severe SAH. A reduced ischemic threshold caused by these risk factors may help explain the higher prevalence of multiple infarctions in these patients.

Although we tried to restrict our definition of cerebral infarction on 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, delayed consequences of a dissection provoked during catheterization but undetected on angiogram, or edema from retraction injury that may have resolved after performance of the last CT scan).17,18 Metallic artifacts from clips and coils used to secure the ruptured aneurysm may have also interfered with the precise interpretation of findings on CT scan, especially in the posterior fossa. Yet, the incidence of cerebral infarction we found is comparable to that reported in previous prospective series using CT scan.19

In conclusion, single cortical infarction in the area of the ruptured aneurysm and multiple infarctions, often including bilateral and subcortical lesions, are the 2 most frequent patterns of delayed cerebral ischemia after aneurysmal SAH. Future research should attempt to answer if these 2 patterns represent different pathophysiological mechanisms or different degrees of severity of the same vascular process.

**Acknowledgments**

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**References**


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