Impact of Basilar Artery Vasospasm on Outcome in Patients With Severe Cerebral Vasospasm After Aneurysmal Subarachnoid Hemorrhage

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Background and Purpose—The purpose of the present study was to evaluate the impact of basilar artery (BA) vasospasm on outcome in patients with severe vasospasm after aneurysmal subarachnoid hemorrhage (aSAH).

Methods—Sixty-five patients with clinically suspect severe cerebral vasospasm after aSAH underwent cerebral angiography before endovascular treatment. Vasospasm severity was assessed for each patient by transcranial Doppler measurements, angiography, and 99mTc-ethylcysteinate dimer single-photon emission computed tomography (ECD-SPECT) imaging. Percentage of BA narrowing was calculated in reference to the baseline angiogram.

Results—BA narrowing ≥25% was found in 23 of 65 patients, and delayed brain stem (BS) hypoperfusion, as estimated by ECD-SPECT, was found in 16. Fourteen of 23 patients with BA narrowing ≥25% experienced BS hypoperfusion, whereas only 2 of 42 patients with ≥25% BA narrowing experienced BS ischemia (P<0.001). Stepwise logistic regression after adjusting for age with Hunt and Hess grade, Fisher grade, hydrocephalus, and aneurysmal location as covariables revealed BA narrowing ≥25% and delayed BS hypoperfusion to be significantly and independently associated with unfavorable 3-month outcome (P=0.0001; odds ratio, 10.1; 95% CI, 2.5 to 40.8; and P=0.007; odds ratio, 13.8, 95% CI, 2.18 to 91.9, respectively).

Conclusions—These findings suggest for the first time that BA vasospasm after aSAH is an independent and significant prognostic factor associated with poor outcome in patients with severe cerebral vasospasm requiring endovascular therapy. Further study should be done to evaluate the role of interventional therapy on outcome in patients with posterior circulation vasospasm. (Stroke. 2006;37:2738-2743.)

Key Words: angiography • basilar artery • cerebral blood flow • single-photon emission computed tomography • subarachnoid hemorrhage • vasospasm

Cerebral vasospasm (VS) remains a major cause of morbidity and mortality after aneurysmal subarachnoid hemorrhage (aSAH). Although many studies have demonstrated that significant arterial narrowing in the anterior circulation is associated with reduced regional cerebral perfusion and worse outcome, little is known about VS in the vertebrobasilar system and its effect on brainstem (BS) perfusion and outcome after aSAH.1–5 Most studies on basilar artery VS (BAVS) have been based mainly on transcranial Doppler (TCD) measurements in a mixed group of patients with traumatic and spontaneous SAH, without complementary perfusion measurements in the affected posterior circulation territories. Indirect information regarding the outcome of traumatic brain injury (TBI) with BAVS has suggested a poor outcome for patients with BAVS as measured by TCD.2,3 Recently, we showed that BAVS is associated with delayed ischemia in the posterior circulation territories, particularly in the BS, and these data suggested that BAVS is associated with poor 1-month neurological outcome.6 However, because those patients with BAVS had a higher incidence of anterior circulation VS than did patients without BAVS, a conclusion regarding its impact on patient outcome could not be made. The purpose of the present study was to evaluate the effect of BAVS on the outcome of patients with suspected clinically severe VS after aSAH.

Subjects and Methods

Patients

The study was approved by the local ethics committee. Between January 2001 and March 2004, 853 patients with aSAH were treated...

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2738
at Harborview Medical Center. Of these, 423 underwent serial 99mTc-ethylcysteinate dimer single-photon emission computed to-
mography (ECD-SPECT) imaging for the diagnosis of VS-related perfusion impairments, and 83 underwent endovascular intervention
for VS. Of these, 83 patients met the study criteria, and 23 of them have been reported in a previous study.6

There were 38 female and 27 male patients, with an average age of 49.6 years (range, 25 to 76). Medical records and imaging studies were reviewed retrospectively. Indications for endovascular inter-
vention for VS were delayed ischemic deterioration that did not respond to hypervolemia-hypertension-hemodilution therapy (n=39) or a severe, delayed perfusion impairment in the middle and anterior cerebral artery (MCA and ACA, respectively) territories and/or BS, as estimated by ECD-SPECT imaging in those patients for whom a neurological evaluation was not reliable (n=26).

All patients underwent 4-vessel cerebral angiography before endovascular therapy. Patients included in the study met the following
criteria: (1) Aneurysms had been secured by clipping or coiling
within 48 hours after the initial bleeding. (2) Baseline 4-vessel
cerebral diagnostic angiography, performed within 48 hours of the
initial bleed, did not show narrowing, stenosis, or occlusion of the
vertebral or basilar arteries. (3) Unimpaired brain and BS perfusion
on baseline ECD-SPECT imaging was performed after surgery or
coiling and within 72 hours of the initial bleeding. (4) ECD-SPECT
imaging was done before endovascular therapy. (5) Daily TCD
measurements were taken for 2 weeks or until VS resolution.

The severity of neurological impairment on admission was as-

Assessment of VS Severity

VS severity was assessed by 3 methods: (1) degree of narrowing in
the MCA, ACA, and BA by comparing diameters in the anteropos-
terior or Towne’s view and lateral projection arteriograms against
the baseline admission angiogram; (2) duration of VS in days, defined
by daily TCD measurements; (3) appearance of perfusion impair-
ment as estimated by ECD-SPECT; and (4) appearance of late brain
infarction.

Angiography and Endovascular Intervention

All angiography was performed on a biplane system (Integris 5000,
Philips Medical Systems), with selective catheterization of the right
and left internal carotid artery and either the left or right vertebral
artery, depending on arterial dominance. Measurements of the MCA,
ACA, and BA diameters were performed with image magnification
and digital calipers on an electronic image viewing system (Centric-
ity, General Electric Measurements systems). Two observers per-
formed all measurements, and the interobserver disagreement was
≈10%. VS was graded as mild (0% to 24% narrowing), moderate
(25% to 49% narrowing), or severe (≥50% narrowing). Our indica-
tions and protocol for balloon angioplasty and intra-arterial injection
of papaverine have been previously reported.9,10 Balloon angiop-
asty was performed only in those vessels that were found to have
moderate or severe vasospasm. Affected vessels included the internal
carotid arteries, M1, A1 segments, BA, and the vertebral arteries.

SPECT Imaging

99mTc ECD-SPECT imaging techniques used, data acquisition meth-
ods, and interpretations have been previously reported.6,12–14 The
appearance of new areas with decreased uptake compared with
global and cerebellar uptake represented delayed hypoperfusion.
Crossed cerebellar diaschisis was taken into account when there were
contralateral cerebral hemispheric defects. For analysis and descrip-
tive purposes, significant BS hypoperfusion was defined as an
average uptake <70% of uptake compared with the global cerebral
hemispheric uptake and/or baseline BS uptake (representing moder-
ate and severe BS hypoperfusion). Uptake of <50% in the anterior
circulation territories or BS was regarded as severe hypoperfusion.
Two nuclear medicine physicians analyzed all SPECT image read-
ings, and these were congruent.

TCD Measurements

The intracranial MCA and mean flow velocities (FVs) were mea-
sured through temporal windows, and VS was diagnosed according
to criteria suggested by Aaslid et al13 and Lindegaard et al: severe
MCA VS as a mean FV of >200 cm/s and a hemispheric index >6. The
BA FVs were measured through the foramen magnum,1 and
BAVS was defined whenever the FV was >85 cm/s, according to
criteria suggested by Sloan et al.4

Management Protocol

All patients were admitted to the intensive care unit after initial imaging
studies, including a noncontrast head CT and CT angiogram, had been
obtained and were resuscitated according to established standard-of-care
guidelines. Unconscious patients were intubated, ventilated, and admin-
istered intravenous propofol and fentanyl for sedation and analgesia.
Intracranial pressure was monitored in all unconscious patients with a
Camino fiberoptic catheter, and in patients with hydrocephalus, an
external ventricular catheter was placed. Oral nimodipine and phenytoin
were administered routinely. All patients received hypervolemia-
hypertension-hemodilution therapy as initial treatment for VS, guided
by use of a central venous catheter and arterial catheter (all patients had
a mean arterial pressure >100 mm Hg and a central venous pressure
>10 mm Hg during the tests).

Statistical Analysis

For all data presented as mean±SD, the various subgroups were
compared by parametric ANOVA, and for categorical data, Fisher’s
exact test was used. The significance of associations between categorical
variables and outcome scores at discharge and at the 3-month follow-up
examination was assessed with the Mantel-Haenszel χ² test. For con-
tinuous variables, Spearman correlation was used to assess significance.
Stepwise logistic regression was used for multivariate analysis to evalu-
ate the impact of BAVS on outcome. Differences were considered
significant when they reached a probability value of <0.05.

Results

Clinical and Demographic Data

Clinical and demographic data of the patients with and
without BAVS and BS hypoperfusion are presented in Tables
1 and 2.

Basilar Artery Vasospasm

BA narrowing of >25% was found in 23 of 65 patients (37%)
included in the study. Of these, 14 patients had narrowing of >50%.
Of 23 patients with BA narrowing of >25%, 14 (60%) experienced
significant BS hypoperfusion, whereas only 2 of 45 (5%) patients
with BA narrowing of <25% experienced BS hypoperfusion.
Patients with BA narrowing of >25% had demographic character-
istics, clinical conditions, and anterior circulation VS severity
parameters similar to those of patients with BA narrowing of <25%
(Table 1), except for a higher incidence of significant bleeding (87%
versus 67%, P<0.0345), a higher incidence of posteriorly located aneurysms (35% versus 26%, insignificant [NS]), thalamic nuclei hypoperfusion (56% versus 25%, NS), and BS hypoperfusion (87% versus 5%, P<0.001). In only 3 of 65 patients was balloon angioplasty limited to the posterior circulation arteries.

### TABLE 1. Demographic and Clinical Parameters, Treatment Modality, VS Severity, and Outcome in 65 Patients Undergoing Endovascular Therapy for Cerebral VS in Relation to BA Narrowing as Demonstrated by Angiography

<table>
<thead>
<tr>
<th>BA</th>
<th>Unimpaired BS</th>
<th>Hypoperfusion BS</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of patients</td>
<td>65</td>
<td>42</td>
<td>23</td>
</tr>
<tr>
<td>Age, mean ± SD, y</td>
<td>50 ± 10</td>
<td>52 ± 11</td>
<td>NS</td>
</tr>
<tr>
<td>Male/female</td>
<td>27/38</td>
<td>19/23</td>
<td>8/15</td>
</tr>
<tr>
<td>H&amp;H classification</td>
<td>Grades I–III</td>
<td>28</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>Grades IV and V</td>
<td>14 (33%)</td>
<td>8 (35%)</td>
</tr>
<tr>
<td>Fisher score</td>
<td>1 and 2</td>
<td>14</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>3 and 4</td>
<td>28 (67%)</td>
<td>20 (87%)</td>
</tr>
<tr>
<td>Aneurysmal location</td>
<td>Posterior circulation</td>
<td>11 (26%)</td>
<td>8 (35%)</td>
</tr>
<tr>
<td></td>
<td>Hydrocephalus</td>
<td>14 (33%)</td>
<td>8 (35%)</td>
</tr>
<tr>
<td></td>
<td>Coiling</td>
<td>8 (19%)</td>
<td>6 (26%)</td>
</tr>
<tr>
<td>TCD measurements, mean ± SD, d</td>
<td>Severe MCA VS</td>
<td>4 ± 1</td>
<td>5 ± 1.3</td>
</tr>
<tr>
<td></td>
<td>MCA VS</td>
<td>10 ± 2</td>
<td>10 ± 2.31</td>
</tr>
<tr>
<td></td>
<td>BS</td>
<td>2.3 ± 1.6</td>
<td>9.7 ± 2.2</td>
</tr>
</tbody>
</table>

### Distribution of hypoperfusion on SPECT

<table>
<thead>
<tr>
<th>BA</th>
<th>Unimpaired BS</th>
<th>Hypoperfusion BS</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>BA Narrowing =&lt;25%</td>
<td>23 (55%)</td>
<td>14 (46%)</td>
<td>NS</td>
</tr>
<tr>
<td>BA Narrowing =≥25%</td>
<td>28 (75.5%)</td>
<td>17 (65%)</td>
<td>NS</td>
</tr>
<tr>
<td>Thalamic nuclei</td>
<td>14 (24.5%)</td>
<td>13 (56%)</td>
<td>NS</td>
</tr>
<tr>
<td>PCA territory</td>
<td>5 (10.2%)</td>
<td>3 (18%)</td>
<td>NS</td>
</tr>
<tr>
<td>BS</td>
<td>2 (5%)</td>
<td>14 (87.5%)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

### Angiographic findings (2 arteries per patient)

<table>
<thead>
<tr>
<th>BA</th>
<th>Unimpaired BS</th>
<th>Hypoperfusion BS</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCA narrowing =&lt;50%</td>
<td>52 (43%)</td>
<td>24 (47%)</td>
<td>NS</td>
</tr>
<tr>
<td>ACA narrowing =&lt;50%</td>
<td>57 (66%)</td>
<td>28 (56%)</td>
<td>NS</td>
</tr>
</tbody>
</table>

### Brain infarction

<table>
<thead>
<tr>
<th>BA</th>
<th>Unimpaired BS</th>
<th>Hypoperfusion BS</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCA territory</td>
<td>13 (31%)</td>
<td>7 (30%)</td>
<td>NS</td>
</tr>
<tr>
<td>ACA territory</td>
<td>19 (45%)</td>
<td>10 (43.5%)</td>
<td>NS</td>
</tr>
</tbody>
</table>

### GOS at discharge

<table>
<thead>
<tr>
<th>BA</th>
<th>Unimpaired BS</th>
<th>Hypoperfusion BS</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Favorable (4 and 5)</td>
<td>13</td>
<td>4</td>
<td>NS</td>
</tr>
<tr>
<td>Unfavorable (1–3)</td>
<td>29 (69%)</td>
<td>19 (83%)</td>
<td>NS</td>
</tr>
</tbody>
</table>

### 3-month GOS

<table>
<thead>
<tr>
<th>BA</th>
<th>Unimpaired BS</th>
<th>Hypoperfusion BS</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Favorable (4 and 5)</td>
<td>28</td>
<td>5</td>
<td>NS</td>
</tr>
<tr>
<td>Unfavorable (1–3)</td>
<td>15 (36%)</td>
<td>18 (78%)</td>
<td>0.0016</td>
</tr>
</tbody>
</table>

GOS indicates Glasgow Outcome Scale; PCA, posterior circulation arteries. Other abbreviations are as defined in text.

### BS Hypoperfusion

Significant BS hypoperfusion was found in 16 of 65 patients (25%). Eleven (75%) had BA narrowing of >50%, and 14 (87%) had BA narrowing of >25%, whereas only 1 patient of 49 (2%) who did not experience significant BS hypoperfusion
had BA narrowing of >50% and 9 (18%) had BA narrowing of >25%. Patients with significant BS ischemia were similar in regard to their clinical condition, demographic characteristics, and anterior circulation VS severity parameters to patients without BS hypoperfusion (Table 2), except for a higher proportion of poor-grade patients (50% versus 31%, NS), a higher incidence of significant bleeding (88% versus 73%, NS), a higher incidence of posteriorly located aneurysms (37% versus 26%, NS), and a higher incidence of thalamic nuclei hypoperfusion (56% versus 24%, NS).

Impact of BAVS on Patient Outcome
The impact of age, clinical condition, and VS severity parameters on patient outcome is presented in Table 3. Univariate analysis identified age (divided by decades; \( P=0.012 \), odds ratio [OR], 2.18; 95% CI, 1.18 to 3.84), high H&H grade (\( P=0.0206 \); OR, 3.78; 95% CI: 1.24 to 11.5), significant bleeding (Fisher grade III and IV; \( P=0.0248 \), OR, 4; 95% CI, 1.21 to 13.23), hydrocephalus (\( P=0.01 \); OR, 4.72; 95% CI, 1.47 to 15.17), BA narrowing of >25% (\( P=0.0016 \); OR, 6.27; 95% CI, 2.08 to 21.72), BA narrowing of >50% (\( P=0.036 \); OR, 5.6; 95% CI, 1.12 to 28.0), and significant BS hypoperfusion (\( P=0.0227 \); OR, 4.3; 95% CI, 1.24 to 15.45) to be associated with an unfavorable 3-month outcome (Table 3). Multivariable analysis after adjusting for age with aneurysmal location, hydrocephalus, H&H grade, and Fisher grade as covariables showed that BA narrowing of >25%, BA narrowing of >50%, and significant BS hypoperfusion, as estimated by ECD-SPECT, were independent variables significantly associated with an unfavorable 3-month outcome (Table 4).

### TABLE 3. ORs and 95% CIs for Unfavorable Outcome at Discharge and at 3 Months (GOS 1–3) of Various Demographic, Clinical, and Hemodynamic Parameters, Including BA Narrowing and BS Hypoperfusion

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Unfavorable Outcome at Discharge</th>
<th>Unfavorable 3-Month Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>( P=0.38 ); ( OR=2.1 ); 1.04–4.24</td>
<td>( P=0.0206^* ); ( OR=3.78 ); 1.24–11.5</td>
</tr>
<tr>
<td>H&amp;H score</td>
<td>( P=0.087 ); ( OR=3.33 ); 0.85–13.15</td>
<td>( P=0.0248^* ); 4; 1.21–13.23</td>
</tr>
<tr>
<td>Fisher score</td>
<td>( P=0.145 ); ( OR=1.58 ); 0.452–5.55</td>
<td>( P=0.0016^* ); 6.72; 2.08–21.72</td>
</tr>
<tr>
<td>Aneurysmal location (posterior vs anterior circulation)</td>
<td>( P=0.357 ); 1.92; 0.48–7.75</td>
<td>( P=0.0227^* ); 4.3; 1.24–15.45</td>
</tr>
<tr>
<td>Surgery VS coiling</td>
<td>( P=0.168 ); ( OR=1.6 ); 0.62–15.32</td>
<td>( P=0.01^* ); 4.72; 1.47–15.17</td>
</tr>
<tr>
<td>Hydrocephalus</td>
<td>( P=0.006^* ); 12.44; 1.53–101.55</td>
<td>2.08–21.72</td>
</tr>
<tr>
<td>TCD measurements, d</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MCA VS</td>
<td>( P=0.461 ); ( OR=1.095 ); 0.75–1.14</td>
<td>( P=0.01^* ); 4.72; 1.47–15.17</td>
</tr>
<tr>
<td>Severe MCA VS</td>
<td>( P=0.624 ); ( OR=0.9 ); 0.58–1.39</td>
<td>( P=0.0248^* ); 4; 1.21–13.23</td>
</tr>
<tr>
<td>BAVS</td>
<td>( P=0.143 ); ( OR=2.7 ); 0.57–6.05</td>
<td>( P=0.01^* ); 4.72; 1.47–15.17</td>
</tr>
<tr>
<td>Angiography findings</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MCA narrowing ( \leq 50% )</td>
<td>( P=0.847 ); ( OR=0.91 ); 0.34–2.41</td>
<td>( P=0.813 ); 0.88; 0.33–2.41</td>
</tr>
<tr>
<td>ACA narrowing ( \leq 50% )</td>
<td>( P=0.063 ); ( OR=3.09 ); 0.94–10.13</td>
<td>( P=0.847 ); 0.91; 0.34–2.42</td>
</tr>
<tr>
<td>ECD-SPECT</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ACA territory ischemia</td>
<td>( P=0.985 ); ( OR=1.01 ); 0.3–3.41</td>
<td>( P=0.107 ); 0.37–3.13</td>
</tr>
<tr>
<td>MCA territory ischemia</td>
<td>( P=0.132 ); ( OR=0.65 ); 0.21–2.03</td>
<td>( P=0.623 ); 1.31; 0.49–3.52</td>
</tr>
<tr>
<td>Thalamic nuclei</td>
<td>( P=0.375 ); ( OR=0.42 ); 0.22–1.59</td>
<td>( P=0.298 ); 0.59; 0.14–1.3</td>
</tr>
<tr>
<td>Brain infarction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MCA territories</td>
<td>( P=0.132 ); ( OR=0.47 ); 0.14–1.3</td>
<td>( P=0.214 ); 0.59; 0.22–1.59</td>
</tr>
<tr>
<td>ACA territories</td>
<td>( P=0.321 ); ( OR=0.702 ); 0.34–1.98</td>
<td>( P=0.493 ); 0.203; 0.34–1.7</td>
</tr>
<tr>
<td>BA narrowing ( \leq 50% )</td>
<td>( P=0.151 ); ( OR=4.76 ); 0.57–40.01</td>
<td>( P=0.036^* ); 5.6; 1.12–28.0</td>
</tr>
<tr>
<td>BA narrowing ( \leq 25% )</td>
<td>( P=0.376 ); ( OR=3.09 ); 0.45–5.52</td>
<td>( P=0.0016^* ); 6.72; 2.08–21.72</td>
</tr>
<tr>
<td>BS hypoperfusion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Estimated by ECD-SPECT</td>
<td>( P=0.5284 ); ( OR=1.73 ); 0.43–7.03</td>
<td>( P=0.0227^* ); 4.3; 1.24–15.45</td>
</tr>
</tbody>
</table>

*Significant \( P \) value.

### TABLE 4. ORs and 95% CIs for Unfavorable Outcome (GOS 1–3) at 3 Months by Multivariate Analysis After Adjusting for Age (in Decades) With the Covariables*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>( P )</th>
<th>OR</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>BA narrowing ( \leq 25% )</td>
<td>( P=0.001 )</td>
<td>10.1</td>
<td>2.5–40.8</td>
</tr>
<tr>
<td>BA narrowing ( \leq 50% )</td>
<td>( P=0.03 )</td>
<td>8.1</td>
<td>1.2–53.3</td>
</tr>
<tr>
<td>BS hypoperfusion</td>
<td>( P=0.007 )</td>
<td>13.8</td>
<td>2.1–91.9</td>
</tr>
</tbody>
</table>

*HH grade, Fisher score, aneurysmal location (posterior circulation vs anterior circulation); tested separately for BA narrowing of \( \leq 25\% \), BA narrowing of \( \leq 50\% \), and BS hypoperfusion. BS hypoperfusion represent <70% of baseline or global uptake as estimated by ECD-SPECT.
Discussion
Although VS after aSAH was described >50 years ago and has been uniformly recognized by the neurosurgical community for >20 years, many questions remain unanswered regarding the clinical significance of posterior circulation VS. Should the posterior circulation be monitored for VS? Does BAVS lead to reduced collateral perfusion to affected anterior circulation territories, or does it reduce perforating arterial flow to the BS? What is its impact on patient and tissue outcome?

Most studies on VS in the posterior circulation have been based mainly on TCD measurements, sometimes in a mixed group of TBI and spontaneous SAH patients. Soustiel et al. and Lee et al. have suggested that BAVS as measured by elevated TCD FVs is associated with poor outcome after TBI. In a comparative analysis that included ECD-SPECT imaging and TCD measurements, we recently reported that BAVS is associated with delayed posterior circulation ischemia and poor 1-month outcome. However, the true impact on patient outcome could not be delineated, because the intensity of the anterior circulation VS could not “quantified” and because most of the patients with BAVS had anterior circulation VS as well. Nevertheless, we thought that we could better investigate the impact of BAVS on the outcome of patients with clinically suspected severe VS requiring endovascular therapy. We tried to relate it to severity in this select group, for whom we had better hemodynamic data, taking into consideration that these are the patients in whom VS has the worst impact on outcome. In this study, we have tried to “quantify” the severity of VS in the anterior circulation and whether BAVS increases the severity of anterior circulation VS. Our findings relied on the use of TCD, angiography, SPECT, and late brain CT scan to make these assessments. All patients with BAVS had concomitant anterior circulation VS, and comprehensive data on their hemodynamic status were available. Therefore, from the data presented in Tables 1 and 2, we initially attempted to evaluate whether there was a difference in the anterior circulation vasospasm parameters between patients with and without posterior circulation vasospasm. These quantified data regarding the intensity of anterior circulation VS showed that for patients with clinically suspected severe VS, the intensity of anterior circulation VS (as related to the degree of arterial narrowing, the duration of VS, anterior circulation territory perfusion impairments, and tissue outcome) was the same as for patients with anterior circulation VS alone that was severe enough to refer them to interventional therapy. Nevertheless, the findings do not suggest that for all patients with aSAH, the existence of BAVS would increase the intensity of anterior circulation VS by reducing collateral flow, therefore predisposing a higher proportion of these patients to experience clinically significant VS. The findings show that although patients with BAVS had similar demographics, clinical characteristics, and similar intensity of their VS in the anterior circulation and were subjected to the same therapy, their outcome was significantly worse than in patients without BAVS.

Our findings also suggest that BAVS is highly associated with delayed BS hypoperfusion. Because BS perfusion occurs mainly through the perforating arteries, VS in the BA might result in reduced perfusion to the perforating arteries that feed the BS through Venturi effects, as was suggested by Soustiel et al. Nevertheless, a Venturi-like effect may not necessarily explain reduced perfusion in cases where BA narrowing is <50%. We should consider that VS of the perforating artery contributes to hemodynamic impairments. This possibility is suggested by the higher incidence of thalamic hypoperfusion found in patients with BAVS, which can indicate the involvement of perforating vessels.

The incidence of BAVS (35%) and BS ischemia (25%) in the present study population is similar to that documented in previously reports. However, one must remember that these figures do not represent the true prevalence and intensity of BAVS in the general population of aSAH patients. BAVS and the resulting BS hypoperfusion in the study population were associated with a higher incidence of posteriorly located aneurysms and in general, with a more intense bleeding, suggesting that involvement of the posterior circulation arteries in the VS process was probably because of clots from direct bleeding or from significant bleeding from an anterior circulation aneurysm, with spilling of blood into the posterior cistern.

Conclusions
This study suggests for the first time that BAVS is an independent prognostic factor highly associated with an unfavorable outcome in patients with clinically suspected severe VS after aSAH requiring endovascular treatment. Currently, because the study is retrospective and without a control group, we cannot make a statement regarding the role of intervention for posterior circulation VS, and its impact on clinical course and perfusion in the affected brain territories is unclear. However, although patients with BAVS were treated by balloon angioplasty, their outcome was unfavorable. Furthermore, patients with BAVS failed to improve during the follow-up period, although their outcome was not significantly worse at discharge. These results suggest a devastating role for BAVS and the resulting BS hypoperfusion on the long-term outcome of patients with severe cerebral VS. Further studies should be done to evaluate the role of posterior circulation VS monitoring and of benefit from therapeutic interventions.

Disclosures
None.

References


Impact of Basilar Artery Vasospasm on Outcome in Patients With Severe Cerebral Vasospasm After Aneurysmal Subarachnoid Hemorrhage
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