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

Gill E. Sviri, David W. Newell, David H. Lewis, Colleen Douville, Basavaraj Ghodke, Minku Chowdhary, Arthur M. Lam, David Haynor, Menashe Zaaroor and Gavin W. Britz

Stroke. 2006;37:2738-2743; originally published online September 28, 2006;
doi: 10.1161/01.STR.0000244765.29502.85
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0039-2499. Online ISSN: 1524-4628

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

Gill E. Sviri, MD, MSc; David W. Newell, MD; David H. Lewis, MD; Colleen Douville, BA, RVT; Basavaraj Ghodke, MD; Minku Chowdhary, MD; Arthur M. Lam, MD; David Haynor, MD; Menashe Zaaroor, MD, DSc; Gavin W. Britz, MD, MPH

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 ^{99m}Tc-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 know 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

Received May 8, 2006; final revision received June 14, 2006; accepted June 28, 2006.

From the Department of Neurosurgery (G.E.S., D.H.L., M.Z.), Rambam (Maimonides) Medical Center, Haifa, Israel; Swedish Medical Center/Providence Campus (D.W.N., C.D.), Seattle Neuroscience Institute, Seattle Wash; and the Departments of Neurological Surgery (M.C., G.W.B.), Radiology (B.G., D.H.), and Anesthesiology (A.M.L.), Harborview Medical Center, University of Washington, Seattle.

Correspondence to Gill E. Sviri, MD, MSc, Department of Neurosurgery, Rambam (Maimonides) Medical Center, Haifa, Israel. E-mail sviri@u.washington.edu

Stroke **is available at http://www.strokeaha.org** **DOI: 10.1161/01.STR.0000244765.29502.85**

at Harborview Medical Center. Of these, 423 underwent serial ^{99m}Tc-ethylcysteinate dimer single-photon emission computed tomography (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 intervention 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 assessed by Hunt and Hess (H&H) grading,[7] and for statistical purposes, grades I through III were defined as good and grades IV and V were defined as poor. The bleeding intensity on CT was scored according to Fisher grade,[8] and for statistical purposes; grades I and II were defined as nonsignificant bleeding and grades III and IV were defined as significant bleeding. Delayed ischemic deterioration was defined as a worsening of the neurological condition that could not be attributed to rebleeding, hydrocephalus, or postoperative complications. Neurological outcome was assessed by the Glasgow Outcome Scale[9] for all patients at discharge and at 3 months; for statistical purposes, scores of 1 through 3 were defined as an unfavorable outcome and scores of 4 and 5 were defined as a favorable outcome. Follow-up data were acquired during office visits and by contacting the patients or their primary care physicians by telephone or letter.

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 anteroposterior 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 impairment 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 (Centricity, General Electric Measurements systems). Two observers performed 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 indications and protocol for balloon angioplasty and intra-arterial injection of papaverine have been previously reported.[10,11] Balloon angioplasty 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

^{99m}Tc ECD-SPECT imaging techniques used, data acquisition methods, 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 descriptive 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 moderate 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 readings, and these were congruent.

TCD Measurements

The intracranial MCA and mean flow velocities (FVs) were measured through temporal windows, and VS was diagnosed according to criteria suggested by Aaslid et al[15] and Lindegaard et al[16]: 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,[17] and BAVS was defined whenever the FV was >85 cm/s, according to criteria suggested by Sloan et al.[1]

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 administered 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 χ^2 test. For continuous variables, Spearman correlation was used to assess significance. Stepwise logistic regression was used for multivariate analysis to evaluate 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 42 (5%) patients with BA narrowing of <25% experienced BS hypoperfusion. Patients with BA narrowing of >25% had demographic characteristics, 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%

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

	BA Narrowing <25%	BA Narrowing ≥25%	*P*
No. of patients			
65	42	23	
Age, mean±SD, y	50±10	52±11	NS
Male/female			
27/38	19/23	8/15	NS
H&H classification			
Grades I–III	28	15	
Grades IV and V	14 (33%)	8 (35%)	NS
Fisher score			
1 and 2	14	3	
3 and 4	28 (67%)	20 (87%)	0.0345
Aneurysmal location			
Posterior circulation	11 (26%)	8 (35%)	NS
Hydrocephalus	14 (33%)	8 (35%)	NS
Coiling	8 (19%)	6 (26%)	NS
TCD measurements, mean±SD, d			
Severe MCA VS	4±1	5±1.3	NS
VS	10±2	10±2.31	NS
BAVS	2.3±1.6	9.7±2.2	<0.001
Distribution of hypoperfusion on SPECT			
MCA territory	23 (55%)	14 (61%)	NS
ACA territory	28 (75.5%)	17(65%)	NS
Thalamic nuclei	14 (24.5%)	13 (56%)	NS
PCA territory	5 (10.2%)	3 (18%)	NS
BS	2 (5%)	14 (87.5%)	<0.001
Angiographic findings (2 arteries per patient)			
MCA narrowing ≤50%	52 (43%)	24 (47%)	NS
ACA narrowing ≤50%	57 (66%)	28 (56%)	NS
Brain infarction			
MCA territory	13 (31%)	7 (30%)	NS
ACA territory	19 (45%)	10 (43.5%)	NS
GOS at discharge			
Favorable (4 and 5)	13	4	
Unfavorable (1–3)	29 (69%)	19 (83%)	NS
3-month GOS			
Favorable (4 and 5)	28	5	
Unfavorable (1–3)	15 (36%)	18 (78%)	0.0016

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

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%, $P<0.0001$). In only 3 of 65 patients was balloon angioplasty limited to the posterior circulation arteries.

TABLE 2. Demographic and Clinical Parameters, Treatment Modality, VS Severity, and Outcome in 65 Patients Undergoing Endovascular Therapy for Cerebral VS in Relation to Estimated BS Perfusion by ECD-SPECT

	Unimpaired BS Perfusion	BS Hypoperfusion	*P*
No. of patients			
65	49	16	
Age, mean±SD, y	49±10.4	53±10.64	NS
Male/female			
27/38	21/28	6/10	NS
H&H classification			
Grade I–III	34	8	
Grade IV and V	15 (31%)	8 (50%)	NS
Fisher score			
1 and 2	13	2	
3 and 4	36 (73%)	14 (88%)	NS
Aneurysmal location			
Posterior circulation	13 (26.5%)	6 (37.5%)	NS
Hydrocephalus	16 (33%)	6 (37.5%)	NS
Coiling	11 (22.5%)	3 (19%)	NS
TCD measurements, d			
Severe MCA VS	4±1	4±1.16	NS
MCA VS	10±2	10±2.29	NS
BAVS	3.6±1.5	10.1±2	<0.001
Distribution of hypoperfusion on SPECT			
MCA territory	27 (55%)	10 (56%)	NS
ACA territory	33 (75.5%)	12(65%)	NS
Thalamic nuclei	13 (24.5%)	14 (56%)	NS
Angiographic findings			
2 arteries per patient			
MCA narrowing ≤50%	46 (43%)	18 (47%)	NS
2 arteries per patient			
ACA narrowing ≤50%	64 (66%)	21 (56%)	NS
BA narrowing ≤25%	9 (18%)	14 (87.5%)	<0.001
BA narrowing ≤50%	1 (2%)	11 (69%)	<0.001
Brain infarction			
MCA territory	15 (31%)	5 (31%)	NS
ACA territory	25 (51%)	7 (44%)	NS
GOS at discharge			
Favorable (4 and 5)	14	3	
Unfavorable (1–3)	35 (71%)	13 (81%)	NS
3-month GOS			
Favorable (4 and 5)	29	4	
Unfavorable (1–3)	20 (41%)	12 (75%)	0.0227

See text and the footnote to Table 1 for an explanation of abbreviations.

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

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

	Unfavorable Outcome at Discharge			Unfavorable 3-Month Outcome		
	P	OR	95% CI	*P*	OR	95% CI
Age	0.38	2.1	1.04–4.24		2.18	1.18–3.84
H&H score	0.087	3.33	0.85–13.15	0.0206*	3.78	1.24–11.5
Fisher score	0.145	1.58	0.452–5.55	0.0248*	4	1.21–13.23
Aneurysmal location (posterior vs anterior circulation)	0.357	1.92	0.48–7.75	0.779	1.31	0.43–4.03
Surgery VS coiling	0.168	1.6	0.62–15.32	0.503	3.09	0.50–5.09
Hydrocephalus	0.006*	12.44	1.53–101.55	0.01*	4.72	1.47–15.17
TCD measurements, d						
MCA VS	0.461	1.095	0.75–1.14	0.453	0.92	0.86–1.93
Severe MCA VS	0.624	0.9	0.58–1.39	0.719	1.38	0.65–1.42
BAVS	0.143	2.7	0.57–6.05	0.019	4.6	1.92–13.46
Angiography findings						
MCA narrowing ≤50%	0.847	0.91	0.34–2.41	0.813	0.88	0.33–2.41
ACA narrowing ≤50%	0.063	3.09	0.94–10.13	0.847	0.91	0.34–2.42
ECD-SPECT						
ACA territory ischemia	0.985	1.01	0.3–3.41	0.9	1.07	0.37–3.13
MCA territory ischemia	0.132	0.65	0.21–2.03	0.623	1.31	0.49–3.52
Thalamic nuclei	0.375	0.42	0.22–1.59	0.298	0.59	0.14–1.3
Brain infarction						
MCA territories	0.132	0.47	0.14–1.3	0.214	0.59	0.22–1.59
ACA territories	0.321	0.702	0.34–1.98	0.493	0.203	0.34–1.7
BA narrowing						
≤50%	0.151	4.76	0.57–40.01	0.036*	5.6	1.12–28.0
≤25%	0.376	3.09	0.45–5.52	0.0016*	6.72	2.08–21.72
BS hypoperfusion						
Estimated by ECD-SPECT	0.5284	1.73	0.43–7.03	0.0227*	4.3	1.24–15.45

*Significant *P* value.

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), hydrocephalous ($P=0.01$; 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 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*

	P	OR	95% CI
BA narrowing ≤25%	0.001	10.1	2.5–40.8
BA narrowing ≤50%	0.03	8.1	1.2–53.3
BS hypoperfusion	0.007	13.8	2.1–91.9

*HH grade, Fisher score, aneurysmal location (posterior circulation vs anterior circulation); tested separately for BA narrowing of ≥25%, BA narrowing of ≥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.[18,19] 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.[1–5] Soustiel et al[5] and Lee et al[2] 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[6] that BAVS after aSAH 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.[19] 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.[6]

The incidence of BAVS (35%) and BS ischemia (25%) in the present study population is similar to that documented in previously reports.[5,6] 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

1. Sloan MA, Burch CM, Wozniak MA, Rothman MI, Rigamonti D, Permutt T, Numaguchi Y. Transcranial Doppler detection of vertebrobasilar vasospasm following subarachnoid hemorrhage. *Stroke*. 1994;25:2187–2197.
2. Lee JH, Martin NA, Alsina G, McArthur DL, Zaucha K, Hovda DA, Becker DP. Hemodynamically significant cerebral vasospasm and outcome after head injury: a prospective study. *J Neurosurg*. 1997;87:221–233.
3. Soustiel JF, Bruk B, Shik B, Hadani M, Feinsod M. Transcranial Doppler in vertebrobasilar vasospasm after subarachnoid hemorrhage. *Neurosurg*. 1998;43:282–291.
4. Soustiel JF, Shik V, Shreiber R, Tavor Y, Goldsher D. Basilar vasospasm diagnosis: investigation of a modified 'Lindegaard Index' based on imaging studies and blood velocity measurements of the basilar artery. *Stroke*. 2002;33:72–77.
5. Soustiel JF, Shik V, Feinsod M. Basilar vasospasm following spontaneous and traumatic subarachnoid haemorrhage: clinical implications. *Acta Neurochir (Wien)*. 2002;44:137–144.

6. Sviri GE, Lewis DH, Correa R, Britz GW, Douville CM, Newell DW. Basilar artery vasospasm and delayed posterior circulation ischemia after aneurysmal subarachnoid hemorrhage. *Stroke*. 2004;35:1867–1872.
7. Hunt WE, Hess RM. Surgical risk as related to time of intervention in the repair of intracranial aneurysms. *J Neurosurg*. 1968;28:14–20.
8. Fisher CM, Kistler JP, Davis JM. Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computerized tomographic scanning. *Neurosurgery*. 1980;6:1–9.
9. Jennett B, Bond M. Assessment of outcome after severe brain damage: a practical scale. *Lancet*. 1975;1:480–484.
10. Srinivasan J, Eskridge J, Grady MS, Newell DW, Winn HR. Endovascular therapy for vasospasm. *Clin Neurosurg*. 2002;49:261–273.
11. Newell DW, Eskridge J, Aaslid R. Current indications and results of cerebral angioplasty. *Acta Neurochir Suppl*. 2001;77:181–183.
12. Elliott JP, Newell DW, Lam DJ, Eskridge JM, Douville CM, Le Roux PD, Leweis DH, Mayberg MR, Grady MS, Winn HR. Comparison of balloon angioplasty and papaverine infusion for the treatment of vasospasm following aneurysmal subarachnoid hemorrhage. *J Neurosurg*. 1998;88:277–284.
13. Rajendran JG, Lewis DH, Newell DW, Winn HR. Brain SPECT used to evaluate vasospasm after subarachnoid hemorrhage: correlation with angiography and transcranial Doppler. *Clin Nucl Med*. 2001;26:125–130.
14. Lewis DH, Eskridge JM, Newell DW, Grady MS, Cohen WA, Dalley RW, Loyd D, Grothaus-King A, Young P, Winn HR. Brain SPECT and the effect of cerebral angioplasty in delayed ischemia due to vasospasm. *J Nucl Med*. 1992;33:1789–1796.
15. Aaslid R, Huber P, Nornes H. Evaluation of cerebrovascular spasm with transcranial Doppler ultrasound. *J Neurosurg*. 1984;60:37–41.
16. Lindegaard KF, Nornes H, Bakke SJ, Sorteberg W, Nakstad P. Cerebral vasospasm diagnosis by means of angiography and blood velocity measurements. *Acta Neurochir (Wien).* 1989;100:12–24.
17. Fujioka KA, Douville CM. Anatomy and freehand examination. In: Newell DW, Aaslid R, eds. *Transcranial Doppler*. New York: Raven Press; 1992:9–32.
18. Muizelaar J. The need for a quantifiable normalized transcranial Doppler ratio for the diagnosis of posterior circulation vasospasm. *Stroke*. 2002;33:78.
19. Soustiel JF, Levy E, Bibi R, Lukaschuk S, Manor D. Hemodynamic consequences of cerebral vasospasm on perforating arteries: a phantom model study. *Stroke*. 2001;32:629–635.