

Clinical Article

Brainstem hypoperfusion in severe symptomatic vasospasm following aneurysmal subarachnoid hemorrhage: role of basilar artery vasospasm

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Summary

Background. The hemodynamic effects of vertebrobasilar vasospasm are ill defined. The purpose of this study was to determine the effects of basilar artery (BA) vasospasm on brainstem (BS) perfusion.

Methods. Forty-five patients with delayed ischemic neurological deficits (DIND) following aneurysmal subarachnoid hemorrhage (SAH) underwent cerebral angiography prior to decision-making concerning endovascular treatment. BA diameter was compared with baseline angiogram. Regional brainstem (BS) cerebral blood flow (CBF) was qualitatively estimated by ^{99m}Tc ethyl cysteinate dimer single photon emission computed tomography (ECD-SPECT).

Findings. Delayed BS hypoperfusion was found in 22 (48.9%) of 45 patients and BA narrowing of more than 20% was found in 23 (51.1%). Seventeen of 23 (73.9%) patients with BA narrowing of more than 20% experienced BS hypoperfusion compared to 6 of 22 (27.3%) patients with minimal or no narrowing ($p=0.0072$). Patients with severe and moderate BS hypoperfusion had higher degree of BA narrowing compared to patients with normal BS perfusion and mild BS hypoperfusion ($p<0.001$). The three-month outcome of patients (n=22) with BS hypoperfusion was significantly worse compared to patients (n=23) with unimpaired ($p=0.0377$, odd ratio for poor outcome 4, 1.15–13.9 95% confidence interval).

Interpretation. These findings suggest that the incidence of BA vasospasm in patients with severe symptomatic vasospasm is high and patients with significant BA vasospasm are at higher risk to experience BS ischemia. Further studies should be done to evaluate the effects of endovascular therapy on BS perfusion and the impact of BS ischemia on morbidity and mortality of patients with severe symptomatic vasospasm.

Keywords: Subarachnoid hemorrhage; vasospasm; basilar artery; angiography; SPECT.

Introduction

Cerebral vasospasm remains a major cause for morbidity and mortality after aneurysmal subarachnoid

hemorrhage (aSAH) and many studies have demonstrated that significant arterial narrowing in the anterior circulation is associated with reduced regional cerebral perfusion [3, 10, 12, 18]. Nevertheless, not much is known about vasospasm in the vertebrobasilar system [8, 15, 19, 21, 23, 25] and most studies done on basilar artery (BA) vasospasm were based mainly on transcranial Doppler (TCD) measurements in a mixed group of patients with traumatic and spontaneous SAH without complementary perfusion measurements of the affected posterior circulation territories. The clinical hemodynamic significance of BA vasospasm has not been defined, maybe since the hemodynamic effects of vasospasm were studied and judged by perfusion measurements in the affected post stenosis territories. Furthermore, hypothalamus-perforating vessels are traditionally blamed for delayed ischemic neurological deterioration (DIND) presenting as altered consciousness. Nevertheless, significant BA vasospasm affecting a long BA segment theoretically would reduce the BA perforating flow through Venturi like effect [22] subsequently leading to brainstem (BS) ischemia. Recently we have reported that patients with highly elevated blood flow velocities in the BA following aSAH as measured by transcranial Doppler (TCD) are at higher risk to experience posterior circulation ischemia [25].

The aim of the present study was to evaluate the effects of BA vasospasm on BS perfusion. We looked for the incidence and severity of BS ischemia in

patients with severe symptomatic vasospasm requiring endovascular therapy in correlation with severity BA vasospasm. Single photon emission computed topography (SPECT) imaging has been used for perfusion assessment in which advancement in imaging technology enables estimation of BS relative blood flow [4, 14, 19, 25, 26, 28].

Patients and methods

Patients

The study was approved by the Human Subjects Committee of the University of Washington.

We retrospectively reviewed the medical records for 373 patients with aSAH who were treated at Harborview Medical Center between November 2002 and May 2004. Of them, 45 patients (29 were female and 16 male) had angiography and SPECT imaging as part diagnostic work-out for symptomatic cerebral vasospasm and were found to meet with the following criteria (1) DIND, which was defined as a worsening of the neurological condition that could not be attributed to rebleeding, hydrocephalus, post-operative or systemic complications. (2) Initial 4 vessel diagnostic angiography, which was done within 48 hours of initial bleeding and did not showed narrowing, stenosis or occlusion of the vertebral or basilar arteries. (3) Unimpaired BS perfusion was observed on baseline SPECT imaging done after clipping or coiling and within 72 hours of initial bleeding. (4) Additional SPECT imaging done before the second angiography. (5) Aneurysm was secured by clipping or endovascular therapy within 48 hour from the initial bleeding.

Outcome was defined by Glasgow outcome scale (GOS) 3 months after the injury [13].

Management protocol

All patients were admitted to the ICU after initial imaging studies, including a noncontrast head CT and CT angiogram had been obtained, and were resuscitated using established standard of care guidelines. A diagnostic cerebral angiogram was performed within 12 hours of admission. Aneurysms were treated either through a craniotomy or endovascular coiling within 48 hours from the initial hemorrhage. Hydrocephalus was treated initially by external ventricular drainage and then after by lumbar drainage or shunt. The intracranial pressure was monitor by "Camino" fibro optic intraparenchymal catheter in all the comatose patients. Oral Nimodipine was administered routinely. All patients received as initial treatment for CVS hypervolemia, hemodilution and hypertension (triple H) therapy guided by the use of a central venous catheter and arterial catheter (all patients had mean arterial pressure (MAP) > 110 mmHg and central venous pressure > 10 mmHg during the test).

SPECT imaging

Each patient was injected with approximately 1,110 MBq (30 mCi) ^{99m}Tc ethyl cysteinate dimer (ECD) (Bristol-Myers-Squibb Medical Imaging, North Billerica, MA), and images were obtained approximately 35 minutes later. All images were acquired using a Prism 3000 triple-headed tomographic scanner (Philips Medical Systems, Cleveland, OH) with low-energy, high-resolution collimators. A 20% window was centered on the 140 kilo-electron volt photopeak of ^{99m}Tc ECD-SPECT images were acquired in a step-and-shoot manner with 64 steps, each lasting 25 seconds, acquired over 360° using clockwise

rotation. Images were processed with a Wiener prefilter and RAMP filter for resolution recovery. Software attenuation correction with a coefficient of 0.11 cm^{-1} was used in all patients with intact cranial bones. Hypoperfusion was defined mild, moderate, or severely decreased uptake compared with global average cerebral hemispheric and baseline study uptake and determined by two nuclear medicine physicians via consensus reading. Normal regional cerebral perfusion data are available in adults and show that on average, uptake in BS is 88% of whole brain cortical average blood flow [28]. Operationally, values ranging from 80% to 70% in BS, described as mild hypoperfusion have been reported in Chronic Fatigue Syndrome and are visually apparent as a mild reduction in uptake [1]. Values that between 60–70% reflect moderate hypoperfusion and those below 60% reflect severe hypoperfusion.

Angiographies

Four-vessel cerebral angiography was obtained during the phase of DIND. Measurements of the BA diameter were made in the anterior/posterior or Towne's view and lateral projection and were compared baseline angiogram according to method used by Sloan *et al.* [18]. Quantification of vasospasm using angiographic images from multiple studies is not straightforward because of differences in patient positioning and image magnification. We used the distance between the medial margins of the two posterior cerebral arteries (PCAs) at their widest point as an internal standard to correct for these effects. We measured vasospasm on the anterior/posterior views of the posterior circulation, which were generally obtained in a Caldwell projection, by computing the ratio of the BA diameter to the inter-PCA distance on each study. Since the inter-PCA distance does not change significantly between studies, this makes it possible to compare the diameter of the BA on later studies to the initial diameter, which was assumed to represent the no-vasospasm diameter. On the lateral projection, the BA diameter was measured and compared with the sella turcica. The smallest BA diameter was taken for description the narrowing percentages. For analysis purposes the BA was divided into three equal segments: proximal, middle and distal. Patterns of VS were defined as focal if it involved only one segment and diffuse if it involved two or three segments.

All angiography was performed on a biplane system (Integris 5000, Philips Medical Systems) using selective catheterization of either the left or right vertebral artery (if both arteries were injected, care was taken to use the same side of injection for both sets of images). Measurements were performed with image magnification and digital calipers on an electronic image viewing system (Centricity, General Electric Medical Systems).

All measurements were performed by two observers whose inter-observer agreement was approximately 10% (differences in measurement of BA diameter up to 5%).

TCD measurements

Initial TCD evaluation was performed in all patients within the first 48 hours after the onset of SAH. The intracranial vertebral and basilar artery mean FVs (MFVs) were measured through the foramen magnum according to the technique described by Fujioka and Douville [8].

Statistical analysis

For all data presented as mean \pm standard deviation, the various subgroups were compared by ANOVA and student t-test. For categorical data Fisher's exact tests were used. Differences were considered significant when they reached a p value of less than 0.05.

Results

Patient population

210 of 373 (56%) patients reviewed had baseline and at least one additional follow-up ^{99m}Tc ECD-SPECT imagines. Of them, 45 patients meet with study criteria. Thirty-three of 210 (15.7%) with baseline and additional ^{99m}Tc ECD-SPECT imagines experienced delayed BS ischemia, 22 of them were among 45 patients included in the study. Of 11 patients who did not meet with the study criteria, four presented with DIND and 7 were none symptomatic.

The average age of 45 included patients was 50.1 ± 11.5 and the range was 29–72 years. The H&H grade [11] on admission was 2.83 ± 0.97 , and the Fisher's bleeding score [7] was 3.05 ± 0.94 .

Brainstem ischemia and clinical correlation

Patients were studied between days 4 and 15 after the bleeding (median day: 6). Delayed BS ischemia was

found in 22 (48.9%) of 45 patients. In 11 (50%) of them the ischemia was mild; in 7 (31.8%) was moderate and in 4 (18.2%) was severe. The outcome of patients with BS ischemia was not significantly worse compared to patients without BS hypoperfusion. Patients with BS hypoperfusion had significantly elevated risk for unfavorable outcome (GOS 1–3) as compared to patients without BS hypoperfusion ($p = 0.0377$, odd ratio for poor outcome: 4, 95% confidence interval: 1.15–13.9, Table 1).

Patients with BS ischemia had higher Fisher's score (3.39 ± 0.96 vs. 2.71 ± 1.1 ; $p = 0.0328$; Table 1) and presented more with DIND manifested as alerted consciousness compared to patients without BS ischemia ($p = 0.007$; Table 1).

Brainstem ischemia and basilar artery vasospasm

Of the 45 patients examined, 23 (51.1%) had angiographically determined BA vasospasm (narrowing > 20%). Of these 13 patients had BA narrowing between 20 and 50% and 10 patients had more than 50%

Table 1. Clinical presentation, bleeding intensity, aneurysmal location and outcome of patients with normal BS perfusion and impaired BS perfusion

	Normal BS perfusion on ^{99m}Tc ECD-SPECT	Impaired BS perfusion on ^{99m}Tc ECD-SPECT	<i>p</i> value
No. of patients	23	22	
Age	51 ± 10.9	49.2 ± 12.1	NS
Sex (m/f)	7/16	9/13	NS
H&H's grade			
– I–II	13	10	
– III–V	10	12	NS
Fisher's score	2.71 ± 1.1	3.39 ± 0.96	0.0328
Re-bleeding	3	4	
Aneurysmal location			
– Anterior circulation	19 (82%)	15 (68.2%)	
ACA	8	8	
MCA	4	2	
ICA	7	5	
– Posterior circulation	4 (18%)	7 (31.2%)	NS
Hydrocephalus	7	8	NS
Increased ICP	3	2	NS
DIND			NS
– Focal	17 (74%)	16 (72.3%)	NS
– Altered consciousness	7 (30.4%)	18 (81.8%)	0.007
Anterior circulation territories ischemia	22 (95.7%)	20 (91%)	NS
– ACA territories	17	16	
– MCA territories	8	11	NS
Anterior circulation vasospasm	21 (91.3%)	19 (90%)	
TCD BA mean flow velocities	92 ± 22 cm/sec	114 ± 29 cm/sec	<0.01
3 month GOS	16	8	
– Favorable (4–5)	7	14	
– Unfavorable (1–3)			0.0377

Anterior circulation ischemia was defined as delayed hypoperfusion in the MCA and ACA territories and thalamic nuclei; anterior circulation vasospasm was defined by angiography findings; outcome was assessed by Glasgow Outcome Scale (GOS) one month after the initial hemorrhage.

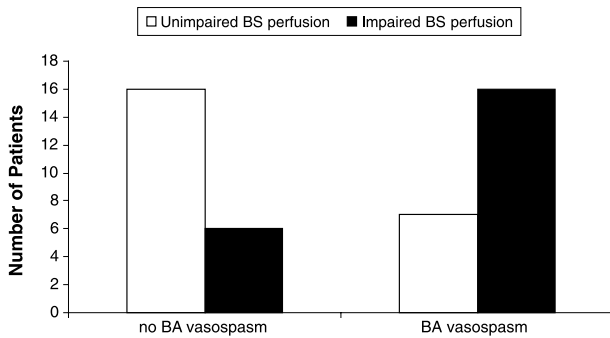


Fig. 1. Correlation between BA vasospasm and degree of BS hypoperfusion as estimated by ^{99m}Tc ECD-SPECT imaging in 45 patients (normal normal BS perfusion; mild mild reduction in perfusion; moderate/severe moderate and severe reduction in perfusion ($p < 0.001$, for variability between the values))

narrowing. Sixteen of 23 (73.9%) patients with BA vasospasm experienced BS hypoperfusion compared to 6 of 22 (28.6%) patients without BA vasospasm ($p = 0.0072$; relative risk (RR) for BS hypoperfusion 2.39; Fig. 1). Eight of 10 (80%) patients with more than 50% BA narrowing experienced BS hypoperfusion compared to 14 of 35 (40%) patients with less than 50% BA narrowing ($p = 0.0351$; RR = 4.181). All patients (n=4) with severe BS hypoperfusion experienced BA narrowing of more than 50%. All patients (n=7) with moderate BS hypoperfusion experienced BA vasospasm. Patients with moderate and severe BS hypoperfusion had significantly higher degree of BA narrowing compared to patients with mild hypoperfusion and normal perfusion ($p < 0.001$; Fig. 2). In SPECT imaging did not show BS hypoperfusion in 7 patients with BA narrowing of more than 20%, in 5 of them the narrowing was focal. However, the narrowing was diffuse in 13 out of 16 patients who experienced BS hypoperfusion ($p = 0.0257$).

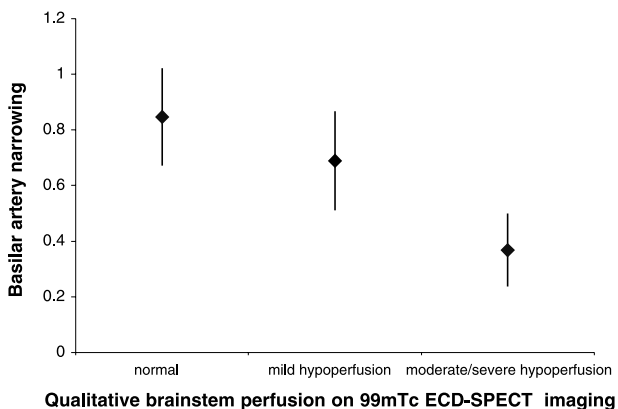


Fig. 2. BA narrowing and the incidence of BS perfusion impairments estimated by ^{99m}Tc ECD-SPECT ($p = 0.0072$) (Basilar artery narrowing is presented as $1 - \text{BA diameter} / \text{baseline BA-diameter}$)

Discussion

In the present study we evaluated the correlation between qualitative BS reCBF estimated by ^{99m}Tc ECD-SPECT imaging and reCBF and angiography findings in patients with severe symptomatic vasospasm after aSAH. The findings suggest that the incidence of delayed BS ischemia in patients with severe symptomatic vasospasm following aneurysmal SAH is high and delayed BS hypoperfusion can be found mainly in patients with BA vasospasm. Furthermore, the severity of BS hypoperfusion correlates with the severity of BA vasospasm. Unlike the cortex the blood flow to the BS arrives mainly through perforating arteries and vasospasm of the BA might reduce the blood flow through them due to a Venturi like effect [22]. Using a phantom model Soustiel *et al.* [22] found that as the narrowing in the parent vessel worsens or extends in length, the flow in the perforating vessels is not only reduced but flow separation appears, which in turn produces a Venturi-like effect responsible for pressure collapse at the aperture of the perforating vessels. These phenomena appeared when the narrowing was higher than 50% and consistent with our findings showing that significant BS ischemia (70% or less than the normal BS perfusion) is associated with significant BA vasospasm (>50% narrowing). This is consistent with our finding that the severity of BS hypoperfusion was in correlation with the severity of the stenosis. Furthermore, patients with moderate and severe BA-VS who maintained normal BS perfusion had only focal VS, which probably did not impair the perfusion through the perforating vessels. As was shown by Soustiel *et al.* [22], not only the severity of the narrowing contributes to the Venturi like effects and the creation of flow separation but also the length of the narrowing.

Six of our patients experienced BS ischemia without BA vasospasm. However, in all of them mild hypoperfusion was found, which is associated with 70–80% of normal perfusion. These changes could have resulted either from involvement of small vessels in the vasospasm process [16, 29] or from a false positive error of the SPECT imaging.

In regard to the use of SPECT imaging for regional cerebral blood flow estimation there are some limitations of the method. Firstly, it is known that there is some non-linearity of uptake of ^{99m}Tc ECD in brain at higher flow values (states of hyperemia) [3]. This drawback is less of a limitation when lower levels of cerebral blood flow are being evaluated. Secondly, SPECT has somewhat of a poorer spatial resolution as compared to PET,

for example. This problem is considerably worse in older single-headed gamma camera systems. Current state-of-the-art SPECT devices with multi-detector configurations, including the triple head SPECT camera used in this study, have a spatial resolution on the order of 7 to 8 mm. Finally, SPECT is not absolutely quantitative if arterial blood sampling and analysis are not performed. This feature would be of greatest concern if all brain vessels are in vasospasm and equally affected. Although SPECT provides only a relative and qualitative estimation of the CBF it has been found by various authors to be a reliable method for measurements of CBF in the setting of vasospasm [3, 10, 17, 18, 20, 28]. In our institution, SPECT imaging has been used for more than 14 years for diagnosis of vasospasm. Performing more than 600 ^{99m}Tc ECD-SPECT studies per year for this indication, we have found [5, 6, 16, 18, 25] as other authors [2, 3, 10, 20, 28], SPECT to be a very reliable imaging modality for assessment of vasospasm-related perfusion impairments and it has been established in our institution as a standard of care for this purpose. Nevertheless, the findings in the present study suggest that the risk for BS hypoperfusion in the present of diffuse and significant BA vasospasm is high.

All the patients included in the present study experienced DIND and angiography studies were done as part of the diagnostic work-up before endovascular therapy. This group included patients with severe vasospasm, which were not responsive to medical therapy. The high incidence of BA found in this group of patients suggests that patients with vasospasm related ischemic deterioration have higher incidence of diffuse vasospasm involving the posterior circulation arteries. Furthermore, patients with BS ischemia had higher Fisher's bleeding score compared to patients without BS ischemia and although most of the aneurysm were located in the anterior circulation, the more intense bleeding could have caused disruption of the posterior arachnoid membranes and deposition of the clots around the posterior circulation arteries resulting in BA vasospasm and subsequently BS ischemia [7, 28]. Furthermore, since most of the patients with BS ischemia presented with consciousness impairments, it might suggest that altered consciousness maybe in part related to BA vasospasm.

Soustiel *et al.* [24] have reported that elevated TCD flow velocities in the BA can be found in up to 53.4% of the patients with SAH of different etiologies. However, the incidence of BA vasospasm among the patients with aSAH is probably lower since elevated BA TCD flow velocities may represent in some of the cases hyperemia

[15, 19, 23]. Nevertheless, in patients with severe symptomatic vasospasm the incidence of BA vasospasm is high (50%) and given its hemodynamic effects, BA vasospasm probably play more significant role in vasospasm after aSAH [25]. Furthermore, patients with significant BS ischemia had worse outcomes compared to patients with only mild reduced perfusion or normal perfusion. Lee *et al.* [15] reported that elevated TCD flow velocities in the vertebrobasilar system are associated with poorer neurological outcome in traumatic SAH patients. Soustiel *et al.* [24] suggested that BA vasospasm is an independent factor associated with poor prognosis after SAH. In a previous study we have found that elevated TCD BA-FVS are associated with poor one month outcome after aSAH, however most of these patients experienced anterior circulation vasospasm, which unable us to drawn a clear conclusion regarding the outcome [25]. The present findings, raise questions regarding possibility of ischemic damage to the BS as a result of vasospasm? Whether BA vasospasm is a predictor of worse outcome in patients with cerebral vasospasm and whether it should be monitor and treat? Due to the limitation of the present study: retrospective data collection and none consecutive series we cannot make a statement regarding the impact of posterior circulation vasospasm on outcome and whether it an independent factor influencing the outcome. However, based on the present findings we can suggest that once interventional therapy is considered for treatment of vasospasm, the posterior circulation arteries should be evaluated and endovascular therapy intervention should be considered in case the BA is significantly and diffusely narrowed. The current findings showing correlation between BA_TCD FVs and BS ischemia are consisting with our previously reports [25] and by Soustiel *et al.* [24] reports that TCD measurements as well as CT angiography can help identify patients with significant BA vasospasm can provide information about the existing and intensity of the BA vasospasm. However, the reversibility of BS ischemia after intervention has not been studied [25] and the effects of this treatment on the outcome of patients with BS ischemia should be further investigated.

Conclusion

The findings suggest that the incidence of BA narrowing in patients with symptomatic vasospasm is high and patients with significant BA vasospasm are at high risk to develop BS ischemia. Further studies should be done

to evaluate the effects BS ischemia on patient outcome and the benefit of therapeutic interventions for BA vasospasm.

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Comment

In the present manuscript, Sviri and co-workers, report on a retrospective angiographic and functional assessment of posterior circulation vasospasm following SAH. Their 45 patient study population is fairly representative with a slight bias towards patients with higher Fisher grades. Repeat conventional angiography was used in order to assess basilar artery (BA) constriction. Functional CBF studies using SPECT were performed to determine brainstem perfusion. The authors demonstrate that the incidence of BA vasospasm and of relevant brainstem hypoperfusion is around 50%. The degree of BA constriction correlated

with the degree of brainstem perfusion impairment. Also, the degree of brainstem perfusion impairment correlated with outcome, despite the relatively small number of patients. The authors conclude that the incidence of hemodynamically relevant BA vasospasm is high and should be addressed more carefully in future clinical research in the field of SAH.

This study addresses an important issue in the field of SAH and vasospasm research. It aims at the analysis of vasospasm-associated brainstem hypoperfusion and stresses the importance of the concept of hemodynamically relevant vasospasm. The work is stimulating in that

it provokes a discussion about the usefulness of monitoring brainstem perfusion and its therapeutic implications.

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