Case Reports

Role of Transcranial Doppler in Neuroradiological Treatment of Intracranial Vasospasm

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Background and Purpose: The expanded role of interventional neuroradiological treatment for post-subarachnoid hemorrhage vasospasm has highlighted the diagnostic utility of transcranial Doppler studies in this condition. The role of transcranial Doppler in follow-up and determining the need for repeat intervention has not been previously emphasized.

Summary of Report: Intracranial angioplasty for clinically evident vasospasm after subarachnoid hemorrhage was performed in four patients. In two patients, transcranial Doppler flow velocities remained elevated despite initial anatomic correction of the vasospasm. Reangiography revealed new areas of involvement by vasospasm. Reangioplasty or papaverine infusion treatment of the new lesions resulted in decreased flow velocities and clinical improvement in all patients.

Conclusions: Transcranial Doppler has a more significant role than has been previously emphasized in the management of patients undergoing interventional neuroradiological treatment for intracranial vasospasm. Specifically, the persistence of elevated transcranial Doppler flow velocities after intracranial angioplasty suggests the need for repeat angiographic evaluation and possibly further therapy. (Stroke 1993;24:299–303)

Key Words • cerebral vasospasm • subarachnoid hemorrhage • ultrasonics

The development of interventional neuroradiological techniques permitting selective catheterization of intracranial vessels has opened numerous opportunities for therapy where such options have previously been limited. Intracranial balloon angioplasty has become an available and accepted treatment for medically intractable vasospasm after subarachnoid hemorrhage since its first report by Zubkov et al in 1984.1 Decreased flow velocities on transcranial Doppler ultrasonography (TCD) after successful angioplasty have been noted, suggesting the utility of TCD in postangioplasty follow-up.2 We describe four patients with vasospasm after subarachnoid hemorrhage who underwent intracranial intervention including angioplasty and intra-arterial papaverine infusion. All patients were monitored after surgery with TCDs, which correctly predicted the presence of vasospasm before clinical manifestations and provided follow-up data on patients after therapy.

In two patients, persistent elevated flow velocities on TCD after angioplasty indicated the appearance of new areas of spasm. This observation led to retreatment of the new lesions with significant clinical recovery. These cases provide further evidence to suggest the beneficial effects of angioplasty in vasospasm and also illustrate the role of TCD in the management of post–subarachnoid hemorrhage vasospasm. This role includes but is not limited to diagnosis and follow-up, which may indicate the need for repeat angiographic evaluation and therapy, particularly during the posthemorrhage period when recurrent vasospasm is most likely.

Subjects and Methods

TCD examinations were performed using a 2-MHz pulsed probe via the transtemporal and transorbital approaches. Depth of insonation was 65–75 mm for siphon, 60–70 mm for anterior cerebral artery (ACA), and 45–60 mm for middle cerebral artery (MCA) signals.

Patient 1

A 45-year-old left-handed woman was admitted to an outside institution after the acute onset of headache, seizure, and loss of consciousness. Computed tomographic (CT) scan on admission revealed subarachnoid hemorrhage filling the perimesencephalic and suprasellar cisterns and dilatation of the lateral and third ventricles. A ventriculostomy was placed. Angiography revealed a posteriorly pointing aneurysm rising from the tip of the basilar artery. On day 4 the patient experienced an acute loss of consciousness and cardiac arrest, from which she was successfully resuscitated. Repeat CT scan revealed new hemorrhage within the lateral, third, and fourth ventricles as well as increased blood in

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TABLE 1. Flow Velocities in Patient 1

<table>
<thead>
<tr>
<th>Day 8—Before angioplasty</th>
<th>Day 9—After angioplasty 1 (MCA)</th>
<th>Day 10—After angioplasty 2 (ICA)</th>
</tr>
</thead>
<tbody>
<tr>
<td>L ICA</td>
<td>142</td>
<td>124</td>
</tr>
<tr>
<td>L MCA</td>
<td>290</td>
<td>300</td>
</tr>
<tr>
<td>L ACA</td>
<td>120</td>
<td>112–124</td>
</tr>
</tbody>
</table>

Values are centimeters per second. MCA, middle cerebral artery; ICA, internal carotid artery; L, left; ACA, anterior cerebral artery.

the basal subarachnoid cisterns. The patient was transferred to the Hospital of the University of Pennsylvania, where aneurysm clipping was performed on day 6 via a left-sided pterional approach. It was noted at the time of surgery that the aneurysm was surrounded by a large amount of subarachnoid clot, which initially obscured the basilar bifurcation and the aneurysm.

Postoperatively, the patient was awake but remained confused and had mild right hemiparesis. She was maintained on hypertensive, hypervolemic, and calcium channel blocker therapy for the prevention of vasospasm. During the following 2 days TCDs indicated elevated flow velocities in the left MCA, ACA, and supraclinoid internal carotid artery (ICA) (Table 1). On day 8, over a period of approximately 4 hours, she developed right hemiparesis and global aphasia.

Immediate angiography showed obliteration of the aneurysm and mild spasm of the basilar artery. Severe spasm of the left MCA and ACA was present with a normal caliber of the supraclinoid ICA (Figure 1). Angioplasty of the M1 segment of the MCA was performed with consequent normalization of the caliber of both the M1 and distal segments of the MCA. Immediate mild improvement in the patient’s aphasia was noted. She was able to state her name and location and followed simple commands with her left extremities but demonstrated only antigravity strength in her right lower extremity.

The next day the patient exhibited sustained mild improvement in aphasia but continued right hemiparesis. TCD identified continued elevated velocities in the MCA, ACA, and supraclinoid ICA. Because of the continued focal deficits and associated abnormal TCD values, repeat angiography was performed.

The previously treated area of the MCA remained normal in caliber. A new area of spasm had developed in the supraclinoid ICA just proximal to the origin of the posterior communicating artery. Angioplasty of the supraclinoid ICA was performed with normalization of caliber in the area of spasm. TCD flow velocities measured immediately after the second angioplasty were decreased in both the MCA and ACA. Improvement in aphasia and right hemiparesis was noted within hours of the second procedure. Continued improvement has occurred with presently no clinical evidence of aphasia or weakness.

Patient 2

A 50-year-old woman was admitted after subarachnoid hemorrhage. Her examination was remarkable for a decreased level of alertness without focal motor deficits. CT scan showed subarachnoid blood, most prominently in the left Sylvian fissure. Angiographic examination showed a large left MCA aneurysm, the presumed source of the hemorrhage. Right ophthalmic, right ICA bifurcation, and basilar tip aneurysms were also present. The next day (day 2 after the ictus), clipping of the left MCA bifurcation aneurysm was performed, and the patient awoke without deficit. Slight elevation of flow values was present until the fifth postoperative day, when velocities in the left siphon increased to 128 cm/sec and those in the left MCA increased to 156 cm/sec (normal, 54±13 cm/sec and 62±12 cm/sec, respectively). At that time, the patient developed right hemiparesis and aphasia. Angiographic examination on day 7 after the ictus showed vasospasm diffusely involving the left MCA including the area of the aneurysm clip (Figure 2). Intracranial angioplasty was performed with restoration of normal caliber of the proximal M1 to within 5 mm of the aneurysm clip. Despite spasm in the region of the clip and in more distal MCA segments, more distal angioplasty was considered unwise because of possible rupture of the abnormal vessel at the previous aneurysm location.

The patient exhibited a mild increase in strength after angioplasty with only minimal improvement in speech. Persistently elevated flow velocities after the procedure resulted in reangiography the next day. Diffuse spasm was identified at and distal to the aneurysm clip. Infusion of papaverine (300 mg over 1 hour) into the left ICA resulted in decrease in diffuse distal spasm. Decreased flow velocities were present on postinfusion TCD, and recovery of right-side motor function and speech occurred over several days.

Patient 3

A 59-year-old man underwent CT scan after being found unconscious at home. Considerable clot was present in the perimesencephalic and suprasellar cisterns with hydrocephalus. After ventriculostomy, the patient was able to localize painful stimuli and intermittently follow commands. Angiographic examination showed a basilar tip aneurysm, which was clipped the next day. After surgery, his level of alertness improved but with only intermittent ability to follow commands. On day 8 after the ictus, a further decrease in his continued poor mental status and elevated flow values as high as 132 cm/sec in the basilar artery (normal, 42±10 cm/sec) prompted angiographic examination. Spasm of the intradural dominant left vertebral artery and proximal basilar artery was present. Angioplasty was performed with good anatomic results. A decrease in flow values in the vertebral and basilar artery was noted after the procedure. Over the ensuing days, slow improvement in his level of alertness occurred with an increased ability to follow commands. His course deteriorated over the next 6 weeks with persistent sepsis and gastrointestinal bleeding, which led to the diagnosis of carcinoma of the esophagus and the patient’s death.

Patient 4

A 65-year-old woman presented with severe headache without neurological deficit. CT evidence of blood in the basal cisterns was present, and angiography showed a large anterior communicating aneurysm. Aneurysm clipping was performed, and the patient awoke without deficit. Two days later increased TCD values were noted, with levels as high as 192 cm/sec in the left
FIGURE 1. Angiogram, patient 1. Top left panel: Left internal carotid artery (ICA) injection anteroposterior view shows severe spasm of the proximal middle cerebral artery and anterior cerebral artery. Bottom left panel: Anteroposterior view of left ICA after angioplasty demonstrates normal caliber of M1 with improved filling of distal middle cerebral artery branches. Supraclinoid ICA is normal in caliber. Top right panel: Twenty-four hours later, new area of spasm has appeared in supraclinoid ICA. Bottom right panel: Lateral view of left ICA after second angioplasty shows dilation of supraclinoid ICA to normal caliber.

carotid siphon and 152 cm/sec in the left MCA (normal, 54±13 cm/sec and 62±12 cm/sec, respectively). The patient became intermittently confused and progressively less responsive and mute. Angiography on day 4 after the ictus revealed only minimal spasm of the right MCA but severe spasm of the left MCA. Left MCA angioplasty was performed with normalization of the vessel caliber. During the next 3 days her level of alertness improved and speech returned in conjunction with a decrease of TCD values.

Discussion

Endovascular balloon dilatation was first applied to the treatment of intracranial vasospasm by Zubkov et al in 1984.1 Since that time, several series have been published emphasizing patient selection and technical aspects of the procedure. Sustained improvement of new onset neurological deficits was observed in 80% and 69% of patients in the two largest series.45 These studies suggest that sustained improvement in neurological deficits secondary to post–subarachnoid hemorrhage vasospasm may be accomplished by prompt angioplasty treatment.

Intracranial angioplasty is performed in conjunction with more standardized antivasospasm regimens including hypertensive hypervolemic therapy and the use of calcium channel blocking agents. Only after development of new severe neurological deficits in the face of maximal medical therapy is angioplasty considered. Before intracranial angioplasty, CT scan is obtained to exclude other causes of acute neurological deterioration including rebleeding, large areas of infarction, and
hydrocephalus. Diagnostic angiography is then performed to identify vasospasm in an accessible segment of artery responsible for the deficit.

Several anatomic situations limit the application of angioplasty in individual patients. The presence of an unclipped aneurysm supplied by the vasospastic segment represents a relative contraindication to angioplasty. Angioplasty of spasm involving distal branches poses an unacceptably high risk of vessel rupture with presently available angioplasty techniques. Angioplasty is not performed in arterial segments immediately adjacent to clipped aneurysms because damaged arterial wall may rupture. After the identification of an appropriate lesion, the balloon angioplasty catheter is placed in the arterial segment, and gentle dilation of the vessel is accomplished under fluoroscopic guidance.

Published cases of balloon angioplasty as an adjunct to the management of post-subarachnoid hemorrhage vasospasm suggest its usefulness. Our cases confirm this benefit and highlight the role of TCD data in clinical decision making for patients undergoing this relatively new form of treatment.

Recent reports indicate that intra-arterial papaverine may be effective in the relief of vasospasm after subarachnoid hemorrhage. This medication was used for treatment of distal MCA spasm and provided significant clinical benefit to patient 2.

TCD is a clearly established method for diagnosing and following the time course of cerebral vasospasm, with sensitivity estimated at 80–100%. Elevated TCD values were evident before the development of clinical deficits despite maximal medical therapy and resulted in the decision to initiate angiographic examination of these patients.

In the first patient treated, persistently elevated TCD flow velocities in the absence of significant clinical improvement were identified several hours after the first angioplasty. TCD correctly suggested an area of new spasm involving the supraclinoid ICA, which was responsible for high velocity in the previously dilated artery.
MCA. After correction of the new focal ICA narrowing by a second angioplasty procedure, TCD values immediately decreased toward normal levels. Concomitantly, sustained clinical improvement in the patient’s condition occurred. High velocity values distal to a focal stenotic segment as seen in this patient reflect maximal dilation of the distal arteriolar-capillary system to compensate for the proximal flow-limiting lesion.

In our second patient, the presence of an aneurysm clip and distal spasm precluded optimal angioplasty therapy. Persistent TCD elevation and incomplete resolution of clinical deficits again indicated repeat intervention using intra-arterial papaverine, which was followed by a decrease in flow velocities. Although the significance of elevated flow velocities must always be interpreted in light of the clinical neurological picture, continued elevation of flow velocity in the distribution of a previously angioplastied vessel may suggest new areas of involvement by spasm and the need for repeat intervention.

All patients treated improved neurologically after neuroradiological intervention. No complications attributable to the procedures occurred. The data provide further evidence of the value of angioplasty in properly selected patients suffering neurological deficits secondary to post–subarachnoid hemorrhage vasospasm and suggest a possible value for intra-arterial papaverine therapy.

This series further validates the substantial value of TCD data in management decisions regarding endovascular therapy for cerebral vasospasm. The ability of TCD to diagnose subclinical vasospasm and identify a lack of response to conventional treatment regimens allows identification of patients who may benefit from more invasive therapy. After angioplasty, continued elevation of flow velocity despite apparently adequate intervention should raise suspicion of new or worsening areas of spasm. In the absence of clinical neurological improvement, repeat intervention should be considered.

References
Role of transcranial Doppler in neuroradiological treatment of intracranial vasospasm.
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