Transluminal angioplasty for treatment of intracranial arterial vasospasm

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Percutaneous transluminal angioplasty for treatment of intracerebral arterial vasospasm is now being performed in selected cases. Thirty-six vascular territories in 13 patients, ranging in age from 15 to 73 years, have been treated with a new silicone microballoon device. This balloon has allowed mechanical dilatation of segmental and diffuse areas of spastic intracerebral blood vessels less than 1 mm in diameter with return to normal luminal diameter. Follow-up angiography has documented improved cerebral perfusion without return of spasm.

In 10 patients (77%), vasospasm was due to subarachnoid hemorrhage following rupture of an intracranial aneurysm. In three patients (23%), spasm with resultant neurological decline occurred during detachable balloon embolization therapy for treatment of an aneurysm. In each case, the vessel caliber returned to normal size following balloon dilatation. In nine (69%) of the 13 cases, balloon dilatation resulted in improvement of neurological function within minutes to hours following the procedure.

Transluminal angioplasty techniques may offer an alternative form of therapy in the management of symptomatic arterial vasospasm.

KEY WORDS • vasospasm • angioplasty • subarachnoid hemorrhage • aneurysm rupture • interventional neuroradiology

Intracranial arterial vasospasm due to subarachnoid hemorrhage (SAH) remains a leading cause of serious morbidity and mortality from aneurysm rupture. Despite a myriad of pharmacological agents that have been evaluated to prevent or reverse the neurological sequelae of cerebral ischemia, no drug has thus far been consistently effective.

Thirteen patients are reported in whom intravascular dilatation by angioplasty of the intracranial vessels achieved a prompt reversal of vasospasm involving 36 vascular territories. There was improvement of the patient’s neurological status in nine of these 13 cases. Antecedent pharmacological therapy had no apparent clinical effect.

Clinical Material and Methods

The indications for treatment by balloon angioplasty at our institution are: 1) failure of a patient to respond to conventional medical and pharmacological therapy, with clinical evidence of continued neurological decline and angiographic evidence of vasospasm; 2) radiographic evidence by computerized tomography and/or magnetic resonance imaging that infarction has not developed in the vascular distribution of spasm; or 3) vasospasm during intravascular balloon embolization therapy for an intracranial aneurysm with resultant neurological decline.

Thirty-six vascular territories in 13 patients, ranging in age from 15 to 73 years, were treated by intravascular balloon dilatation. Table 1 presents a clinical summary of the patients treated, including age and sex, location of the aneurysm, neurological grade on admission and at the time of angioplasty, early results and late outcome following treatment, and vessels treated. Ten patients (77%) were treated for vasospasm due to SAH following aneurysm rupture. Three patients (23%) were treated for clinically symptomatic vasospasm associated with aneurysm treatment by detachable balloon embolization therapy. Table 2 lists the vascular territories associated with aneurysm treatment by detachable balloon embolization therapy. Table 2 lists the vascular territories associated with aneurysm treatment by detachable balloon embolization therapy.
Transluminal angioplasty for arterial vasospasm

TABLE 1
Clinical summary of 13 patients undergoing transluminal angioplasty*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Location of Aneurysm</th>
<th>Neurological Grade†</th>
<th>Early Results at Admission</th>
<th>Late Outcome</th>
<th>Vessels Treated</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>52, M</td>
<td>distal BA</td>
<td>III</td>
<td>improved clinically (III)</td>
<td>excellent</td>
<td>BA, rt &amp; lt PCA, lt VA, rt &amp; lt ICA, rt &amp; lt MCA</td>
</tr>
<tr>
<td>2</td>
<td>61, M</td>
<td>superior cerebellar artery</td>
<td>II</td>
<td>improved clinically (II)</td>
<td>good</td>
<td>rt VA, rt BA, rt &amp; MCA</td>
</tr>
<tr>
<td>3</td>
<td>38, M</td>
<td>MCA</td>
<td>IV-V</td>
<td>improved clinically (III)</td>
<td>excellent</td>
<td>rt ICA, rt MCA</td>
</tr>
<tr>
<td>4</td>
<td>51, F</td>
<td>BA</td>
<td>II</td>
<td>improved clinically (III)</td>
<td>died</td>
<td>lt VA, BA, rt &amp; lt PCA, lt ICA, lt MCA</td>
</tr>
<tr>
<td>5</td>
<td>42, M</td>
<td>distal BA</td>
<td>II-III</td>
<td>excellent (II)</td>
<td>excellent</td>
<td>BA</td>
</tr>
<tr>
<td>6</td>
<td>42, M</td>
<td>MCA</td>
<td>III</td>
<td>improved clinically (III)</td>
<td>good</td>
<td>rt ICA, rt MCA, rt ACA</td>
</tr>
<tr>
<td>7</td>
<td>60, M</td>
<td>post. inferior cerebellar artery</td>
<td>IV</td>
<td>improved clinically (III)</td>
<td>died</td>
<td>lt VA, BA, rt &amp; lt PCA</td>
</tr>
<tr>
<td>8</td>
<td>56, M</td>
<td>MCA</td>
<td>IV</td>
<td>no change (V)</td>
<td>died</td>
<td>rt ICA, rt MCA</td>
</tr>
<tr>
<td>9</td>
<td>46, F</td>
<td>carotid ophthalmic artery</td>
<td>V</td>
<td>no change (V)</td>
<td>died</td>
<td>rt ICA</td>
</tr>
<tr>
<td>10</td>
<td>44, F</td>
<td>distal VA</td>
<td>II</td>
<td>improved clinically (I-II)</td>
<td>excellent</td>
<td>rt VA</td>
</tr>
<tr>
<td>11</td>
<td>73, F</td>
<td>cavernous carotid artery</td>
<td>II</td>
<td>improved clinically (I-II)</td>
<td>good</td>
<td>rt ICA</td>
</tr>
<tr>
<td>12</td>
<td>75, F</td>
<td>MCA</td>
<td>I</td>
<td>improved clinically (II)</td>
<td>good</td>
<td>lt MCA</td>
</tr>
<tr>
<td>13</td>
<td>35, F</td>
<td>MCA</td>
<td>I</td>
<td>improved clinically (II)</td>
<td>excellent</td>
<td>lt MCA</td>
</tr>
</tbody>
</table>

* BA = basilar artery; PCA = posterior cerebral artery; VA = vertebral artery; ICA = internal carotid artery; MCA = middle cerebral artery; ACA = anterior cerebral artery.
† Neurological grade based on the Hunt and Hess classification.24

Angioplasty of the intracerebral vessels was performed with a custom-made device consisting of a nonvalved silicone microballoon attached to a No. 2 French catheter.* The balloon delivery system consists of a coaxial No. 2/4 French polyethylene set passed through a No. 7.3 French untapered catheter. Balloons utilized measured either 0.85 or 1.5 mm in diameter before inflation. For dilatation of the anterior or posterior circulation, the No. 7.3 French catheter is positioned into the internal carotid or vertebral artery using a No. 5 French inner catheter and a 0.035-in. guide wire. The No. 5 French catheter and wire are then removed, leaving the untapered No. 7.3 French catheter in place through which the angioplasty catheter system is introduced.

Angiographic assessment of the progress of the angioplasty can be accomplished by injecting contrast material into the No. 7.3 French catheter around the No. 4 French catheter. The No. 2 French catheter can be shaped into a variety of curves to facilitate entry into various intracranial arterial branches. Dilatations are performed by briefly and repeatedly inflating and deflating the balloon with 0.05 to 0.30 cc of iodinated contrast material, depending on the size of the balloon selected. Each dilatation maneuver is followed by advancement of the balloon catheter into the next constricted arterial segment. Any areas of the artery that remain constricted as “skip areas” may be dilated during the balloon-withdrawal phase of the procedure. The procedure is performed with the patient under local anesthesia in the neuroangiography suite under x-ray fluoroscopic guidance. Continuous neurological assessment is performed throughout the procedure.

Illustrative Cases

Case 2: Diffuse Vasospasm of Posterior Circulation

This 61-year-old man presented with a large SAH. His neurological condition was Hunt and Hess24 Grade II, and he complained of headache and a stiff neck. Cerebral angiography demonstrated an aneurysm arising at the junction of the right superior cerebellar artery and basilar artery without evidence of vasospasm (Fig. 1a). Forty-eight hours later the patient acutely deteriorated, becoming comatose, unresponsive to commands or painful stimuli, and ventilator-dependent (Grade IV-V).

Repeat cerebral angiography demonstrated diffuse vasospasm of the intracerebral portion of the vertebral artery, the entire basilar artery, and both posterior cerebral arteries (Fig. 1b). Angioplasty of the vertebral artery and proximal and mid segments of the basilar
artery was performed without significant change in the patient's neurological condition (Fig. 1c). Following dilatation of the distal basilar and right and left posterior cerebral arteries (Fig. 1d), the patient became responsive and was able to follow commands. Four hours later his neurological condition improved to Grade II and he was extubated. Two days later his aneurysm was surgically clipped, and at examination 30 months after surgery he continues to do well.

**Case 3: Vasospasm of Anterior Circulation**

This 38-year-old man presented with a large SAH and intraparenchymal hemorrhage due to rupture of a giant 25-mm middle cerebral artery aneurysm (Fig. 2a). The patient was comatose, required intubation, and had only semipurposeful movement (Grade IV–V). The aneurysm was clipped immediately.

Four days following surgery the patient developed decerebrate posturing. An angiogram demonstrated moderate to severe vasospasm of the supraclinoid internal carotid and right middle cerebral arteries (Fig. 2b). Both vessels were dilated by balloon angioplasty, with restoration of normal luminal diameter (Fig. 2c and d). The patient's course of pentobarbital and dopamine was tapered and over the next 5 to 7 days he made a gradual clinical improvement. After 3 weeks he was awake, alert, and oriented, and had 80% to 90% of normal function. At follow-up examination 12 months after angioplasty, the patient remains neurologically intact.

**Case 5: Focal Vasospasm of Basilar Artery**

This 42-year-old man presented with SAH due to rupture of a large distal basilar artery aneurysm. Twenty days after the hemorrhage an angiogram demonstrated focal vasospasm of the mid-basilar artery (Fig. 3a). In order to treat the aneurysm by intravascular detachable balloon embolization therapy, angioplasty of the spastic segment of the mid-basilar artery was performed, without complication. A balloon was then easily directed into the aneurysm, inflated to occlude the neck and dome, and detached (Fig. 3b).

Follow-up angiography immediately after the procedure and at 3 months and 1 year demonstrated normal luminal diameter of the basilar artery without evidence of intimal damage, and complete obliteration of the aneurysm. At his 3-year follow-up examination the patient continues to do well.

**Results**

In this series of 13 patients treated for vasospasm, 10 presented with rupture of an intracranial aneurysm resulting in SAH. Vasospasm with resultant neurological decline occurred between 2 and 7 days following the hemorrhage. Despite vigorous medical therapy including volume expansion, induced hypertension, and pharmacological intervention no improvement occurred; therefore, balloon dilatation therapy was performed. Three patients, who were in Grade V neurological condition (Cases 7, 8, and 9), comatose, and moribund, exhibited no improvement following angioplasty and eventually died from brain-stem and/or cerebral ischemia. One patient (Case 4) showed mild improvement following angioplasty but 24 hours later suffered a hemorrhagic infarct and died.

Four patients (Cases 1, 2, 3, and 6) had dramatic improvement within several hours after dilatation of the anterior and posterior circulation. These patients improved from Grade IV–V to Grade II–III condition, and were awake and responsive to commands. Two patients with focal vasospasm (Cases 5 and 10) had
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Fig. 2. Case 3. a: Internal carotid artery angiogram demonstrating a giant 25-mm aneurysm arising from the right middle cerebral artery. b: Angiogram following surgical clipping showing vasospasm (arrow) of the supraclinoid and proximal middle cerebral artery. c: Intraoperative film demonstrating balloon angioplasty (arrow) of the internal carotid and proximal middle cerebral artery. d: Postangioplasty arteriogram demonstrating return of normal luminal diameter after dilatation of the supraclinoid internal carotid artery and proximal middle cerebral artery (arrows).

Dilatation performed prior to balloon embolization therapy to allow passage of a detachable balloon into the ruptured aneurysm. In three patients (Cases 11, 12, and 13), as a result of manipulation of detachable balloons into the aneurysm for therapy, vasospasm developed with resultant neurological decline. Angioplasty was performed to reperfuse the affected vascular territory. Following dilatation there was immediate and prompt reversal of the patients' neurological decline.

In all cases, the angiogram following balloon dilatation demonstrated a return to normal luminal diameter of the treated blood vessel(s). There was also angiographic evidence of improved circulation time. In the patients who survived and in whom follow-up angiography was performed, the vessels dilated by angioplasty exhibited continued patency without recurrence of stenosis, intimal injury, or any other evidence of vessel damage.

Discussion

Transluminal treatment of arteriosclerotic obstruction of vessels by means of percutaneously introduced catheters dates from Dotter and Judkins' report in 1964. A variety of coaxial delivery systems and catheters have been devised over the past two decades. Balloons of latex, woven Dacron, polyolefin, and silicone have been affixed to catheters constructed of Teflon, polyethylene, and polyurethane. Currently, transluminal angioplasty is in widespread use in the treatment of atherosclerotic disease of coronary and peripheral arteries, including brachiocephalic vessels. Angioplasty in intimal hyperplasia, arteritis, and other arterial diseases is less well defined.

In 1984, Zubkov, et al., reported transluminal angioplasty for arterial vasospasm following SAH. Reporting on 105 dilatations in 33 patients, they noted a generally favorable result with regression of focal neurological signs. This report stimulated our own interest in developing a mechanical method to dilate constricted intracranial arteries. In designing our system, we favored the use of a soft silicone balloon rather than a latex balloon because of our concern that a latex balloon might rupture an intracranial vessel. In addition, silicone balloons easily conform to the shape of the vessel lumen and have an inherent ability to advance within the vessel during dilatation. This permits continuous antegrade dilatation and facilitates the continued progress of the catheter.

Cerebral vasospasm usually affects the vessels of the
circle of Willis and immediate stems of major cerebral arteries, regardless of the exact location of the aneurysm. The spasm usually begins in the proximal vessels, even when the bleeding site is more distal. The spasm may be diffuse (involvement over long distances), segmental (band-like areas of constriction), or local (narrowing limited to the vicinity of the aneurysm). Diffuse vasospasm seems to be the most ominous clinically; Sano and Saito, for example, reported a fatal outcome in 10 of 22 patients with diffuse spasm. This correlates with our own series in which four of eight patients with severe diffuse vasospasm died.

The exact mechanism and pathophysiology of cerebral vasospasm and resultant ischemic dysfunction are poorly understood. Suggested theories include an increase in vascular tone due to release of vasoconstrictive endogenous substances within the vessel wall and/or the subarachnoid blood and clot,1,11,13 intimal hyper trophy and myonecrosis,1,13 and muscular contraction of the vessel wall with subsequent anoxic injury and intimal proliferation.7,14,17,35,37 Despite the use of a great variety of pharmacological agents and attempts at early surgical intervention to minimize or reverse vasospasm, no agent has been found entirely effective once neurological decline occurs.

The appropriate timing and patient selection for angioplasty need to be addressed. In patients who developed vasospasm during attempted endovascular balloon placement for treatment of their aneurysm, neurological deterioration was acute. Once dilatation was performed, neurological decline was immediately reversed. We therefore believe that acute vasospasm due to endovascular balloon manipulation can be effectively treated. For patients with severe diffuse vasospasm that has been unresponsive to aggressive medical therapy, and with magnetic resonance or computerized tomography evidence of infarction, angioplasty is probably not warranted. In one patient (Case 7) angioplasty of the vertebral artery, the entire basilar artery, and both posterior cerebral arteries was performed after the patient had been in Grade V neurological condition for 14 days. This patient had neurological evidence of brain-stem infarction and, even though angiographically there was improved posterior circulation perfusion after angioplasty, there was no improvement in neurological status. In Case 4, following angioplasty of a spastic middle cerebral artery, the patient developed a hemorrhagic infarct in the basal ganglia within 24 hours of the procedure and died. This was most likely due to reperfusion of infarcted tissue following the angioplasty procedure. It thus seems that the optimal time to perform angioplasty for spastic arteries is during the acute period. If patients demonstrate clinical evidence of rapid neurological decline and fail a regimen of medical therapy to reverse the effects of hyperperfusion, angioplasty should be considered. From our early observations, those patients whom we have treated as early as possible (within 24 to 36 hours after onset) have more favorable results than those treated after 72 hours.

There are still many unresolved issues regarding vasospasm. These include the role of the microcirculation (for example, the striate and brain-stem perforating vessels) and their communication with larger vessel reperfusion. Does restoration of blood flow to the middle cerebral and basilar artery territories necessarily improve perfusion to the microcirculation? The differences in acute versus chronic spasm of vessels remain unclear, and the changes that occur with angioplasty need to be clarified. The proper timing for angioplasty versus pharmacological or other surgical intervention shall remain a controversial subject until larger groups of patients can be accumulated.

Thus far, it appears that mechanical dilatation of spastic intracerebral arteries caused by either SAH or intravascular balloon embolization therapy is possible. Particularly in the acute stages of symptomatic vasospasm, angioplasty may be effective for improving cerebral perfusion to critical territories of the brain.

References
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Manuscript received October 7, 1988. Accepted in final form April 27, 1989. Address for Dr. Cahan: Department of Neurosurgery, University of California, Irvine, California 92717. Address reprint requests to: Randall T. Higashida, M.D., Department of Radiology, Interventional Neuroradiology Section, L-352, University of California, 505 Parnassus Avenue, San Francisco, California 94143-0628.