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Severe symptomatic vasospasm: the role of immediate postoperative angioplasty

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The clinical success of angioplasty for symptomatic vasospasm following subarachnoid hemorrhage (SAH) depends on early intervention and can best be achieved after the aneurysm is occluded. However, patients presenting with unsecured ruptured aneurysms and established clinical vasospasm offer a dilemma for the surgeon. The authors describe the cases of five such patients who underwent acute clipping of aneurysms followed by immediate postoperative angioplasty between 1988 and 1992. All were referred at least 5 days after SAH. Severe vasospasm compatible with the clinical presentation was confirmed by angiography. The patients met the department's criteria for angioplasty but, because of unclipped aneurysms, were first taken to the operating room for a craniotomy and aneurysm obliteration. Angiography was repeated immediately after surgery. Arterial narrowing had progressed during surgery in two patients. In all patients, postoperative mechanical dilatation was achieved with the use of a silicone microballoon. Following angioplasty, transcranial Doppler ultrasound flow velocities and single-photon emission computerized tomography evaluation indicated improved cerebral perfusion compared to preoperative determinations. Four patients improved clinically and made a good recovery. In this subgroup of patients presenting with proven symptomatic vasospasm and an unclipped but ruptured aneurysm, urgent surgical obliteration of the aneurysm followed by immediate postoperative angioplasty may be a safe and reasonable means to improve outcome.

KEY WORDS • **aneurysm** • **angioplasty** • **interventional radiology** • **vasospasm** • **subarachnoid hemorrhage**

CEREBRAL vasospasm resulting in ischemic stroke remains the leading treatable cause of death and disability after aneurysm rupture.[7,8,15,16] Once an ischemic neurological deficit develops, few currently available medical treatments are consistently effective. By contrast, balloon dilatation is a highly effective mechanical means of reversing subarachnoid hemorrhage (SAH)-induced vasospasm that is refractory to medical therapy.[3,9,10,21,24,25,37]

Two factors appear critical to the success of angioplasty: 1) the aneurysm should be secured prior to angioplasty to prevent rehemorrhage,[3,20,21,24,25] and 2) there is an increased likelihood of preventing permanent neurological injury when the procedure is performed within 18 hours after the onset of symptoms.[3,10,21,24,25,37] Although the use of early surgery for aneurysmal SAH is increasing, some patients still undergo delayed neurosurgical evaluation, presenting with an ischemic deficit due to severe vasospasm and an unsecured, recently ruptured aneurysm. Such patients pose a management dilemma, since the presence of vasospasm and associated ischemic deficit is regarded as a contraindication to immediate surgery and evidence for delayed surgery.[2,5,11,26,27,31,32] These patients, however, may often progress to stroke despite medical management and remain at risk for rehemorrhage. In addition, medical therapies such as hypervolemic hypertensive therapy can precipitate rehemorrhage from the unsecured aneurysm.

The rationale for delaying surgery in the presence of vasospasm has not been supported by recent experimental and clinical studies.[1,4,17,18,22,28,35] Since angioplasty can benefit many patients who continue to deteriorate despite hypervolemic hypertensive therapy and the use of calcium channel blockers,[3,9,10,21,24,25,37] urgent aneurysm obliteration and immediate postoperative angioplasty might be a reasonable approach in patients who present with severe symptomatic vasospasm and an unsecured ruptured aneurysm. The present report describes the use of this technique in five patients with delayed ischemic deficits following SAH.

TABLE 1

*Clinical characteristics and course of five patients undergoing angioplasty immediately after aneurysm clipping**

Case No.	Age (yrs), Sex	Admission Grade†	Aneurysm Location	SAH to Surgery (days)‡	Clinical Deficit	Vessels Dilated	Angioplasty Response (7 days)	Clinical Response	Outcome§
1	35, M	V	rt PICA	5	decreased LOC	lt VA, P_1, P_2, basilar	normal	improved	moderate
2	58, F	III	rt PCoA	7	lt hemiparesis	rt ICA, M_1, M_2; A_1 failed	slight narrowing of rt ICA; MCA normal	improved	moderate
3	30, F	III	rt MCA	5	lt hemiparesis	rt ICA, M_1, M_2	normal	improved	good
4	55, F	V	ACoA	5	decreased LOC	lt M_1, M_2; both A_1's failed	MCA normal	no change	dead
5	39, M	III	lt MCA	5	rt hemiparesis; dysphasia	lt ICA, M_1, M_2	normal	hemiparesis improved	moderate

* PICA = posterior inferior cerebral artery; PCoA = posterior communicating artery; MCA = middle cerebral artery; ACoA = anterior communicating artery; SAH = subarachnoid hemorrhage; LOC = level of consciousness; VA = vertebral artery; ICA = internal carotid artery.

† Hunt and Hess[12] grade at admission to Harborview Medical Center.

‡ Interval between SAH and admission to Harborview Medical Center for surgery and angioplasty.

§ Glasgow Outcome Scale score as described by Jennett and Bond.[14]

Clinical Material and Methods

Patient Population

During a 4-year period (June, 1988, to June, 1992), five patients admitted to Harborview Medical Center and the University of Washington Affiliated Hospitals underwent immediate postoperative angioplasty for vasospasm. The clinical characteristics of these patients are listed in Table 1. Four patients were initially cared for at other hospitals and referred to the University of Washington 5 days after SAH, when clinical deterioration occurred. The remaining patient presented 7 days after aneurysm rupture. Subarachnoid hemorrhage was confirmed by computerized tomography (CT) and each patient underwent preoperative transcranial Doppler ultrasound (TCD) studies and single-photon emission CT (SPECT) evaluation. The distribution of ruptured aneurysms diagnosed by angiography was as follows: two arose from the middle cerebral artery (MCA) and one each from the posterior inferior cerebellar artery, the posterior communicating artery, and the anterior communicating artery.

Surgical Management

Prior to surgery, patients were maintained in a normovolemic state and received calcium channel blockers (nimodipine, 60 mg four times daily). At surgery, each patient was given mannitol (1.5 gm/kg) and lumbar cerebrospinal fluid drainage was initiated for brain relaxation. Intraoperative hypotension and dehydration were avoided. All aneurysms were clipped using standard microsurgical techniques; temporary clipping was not used. Following aneurysm obliteration, the adjacent vessels were bathed in papaverine (300 mg/100 ml saline).

A Swan-Ganz catheter was placed intraoperatively and, once the aneurysm was occluded, aggressive volume expansion and hemodilution were begun using colloid, crystalloid, and occasionally blood products to maintain the pulmonary capillary wedge pressure between 16 and 18 mm Hg, cardiac output greater than 6 liters/min and hematocrit between 30% and 33%. Nimodipine (60 mg four times daily) was administered routinely. Immediately after surgery, repeat CT, TCD, and SPECT examinations were obtained.

According to our standard protocol, the decision to perform immediate postoperative angioplasty was based on the following criteria: 1) a preoperative neurological deficit, such as focal motor weakness or a decreased level of consciousness; 2) exclusion of other causes of clinical deterioration, such as hydrocephalus, cerebral swelling, or hypoxia; 3) no evidence of cerebral infarction on head CT scans; 4) an area of hypoperfusion compatible with the neurological deficit identified by SPECT; 5) TCD evidence of severe spasm, such as an MCA:internal carotid artery (ICA) ratio greater than 6:1; and 6) angiographic evidence of severe vasospasm in a location that could be responsible for the ischemic deficit. All five patients in this series met these criteria.

Angioplasty was performed in the interventional neurovascular suite, using a silicone microballoon attached to a variable-stiffness microcatheter.* The patients were placed under general anesthesia and full heparinization, and the transfemoral percutaneous approach was used. First a repeat angiogram was performed and compared with the preoperative studies to confirm both the severity of vasospasm and obliteration of the aneurysm. High-resolution subtraction angiography and road mapping were used to guide the microballoon to the vasospastic vessel; once the balloon was correctly positioned, the vessel was mechanically dilated by gentle inflation (0.5 atm) and deflation of the balloon under continuous fluoroscopic control. Following angioplasty, a repeat angiogram was obtained to document vascular dilatation.

After completion of the procedure, heparinization was stopped and the patient was returned to the neurosurgical intensive care unit. Hypervolemic therapy, calcium channel blockers, and intensive monitoring

* Silicone microballoon manufactured by Interventional Therapeutics Corp., San Francisco, California; variable-stiffness microcatheter manufactured by Target Therapeutics, San Jose, California.

were continued. Transcranial Doppler ultrasonography was repeated daily for 7 to 10 days and postangioplasty SPECT was obtained. Angiography was repeated 1 week and again 1 month after the procedure. Patients were considered to have responded to angioplasty if they improved at least two Glasgow Coma Scale[38] points or demonstrated two grades of motor improvement within 24 hours of the procedure. Long-term follow-up data were obtained at 6 months by telephone interview and/or clinic visit. The best level of function was graded according to the Glasgow Outcome Score.[14]

Results

The results of angioplasty and patient outcome are illustrated in Table 1. All patients underwent balloon dilatation within 24 hours of the onset of symptoms and in three patients the procedure was performed within 18 hours. Each patient had demonstrated angiographic evidence of severe vasospasm before aneurysm surgery. Compared to angiographic studies obtained before aneurysm surgery, postoperative arterial narrowing was judged to be worse in two patients and unchanged in two patients. In the remaining patient (Case 3) postoperative angiography demonstrated improvement and angioplasty was therefore deferred. However, the patient continued to deteriorate clinically and repeat TCD studies demonstrated a further increase in blood flow velocities. Angiography repeated 4 hours later again showed arterial narrowing; angioplasty was performed at that time.

Angiographic Results

Angiographic evidence of vasospasm resolution was found in 15 of the 18 vessels in which balloon dilatation was attempted. By contrast, untreated vessels remained in spasm. Follow-up angiograms indicated that no vessel was injured by the procedure. Furthermore, the vessel caliber obtained by mechanical dilatation persisted at both short- and long-term follow-up studies. No complications occurred; however, on three occasions the balloon could not be navigated into the constricted A_1 segment of the anterior cerebral artery. Very severe arterial narrowing occasionally hampered balloon entry; this was overcome by advancing the guidewire ahead of the balloon.

Cerebral Perfusion

In addition to the restoration of normal vessel caliber, angiographic studies confirmed improved perfusion following angioplasty. Both TCD and SPECT studies correlated with improved cerebral perfusion. In the four patients who demonstrated clinical improvement, TCD blood flow velocities decreased and remained below preangioplasty levels. By contrast, velocities remained elevated or subsequently increased in untreated vessels. Improvement in cerebral perfusion, which correlated with clinical improvement, was demonstrated in three patients by SPECT evaluation. In one patient (Case 5), CT demonstrated loss of the gray-white junction in the left posterior temporal and parietal regions 1 day after angioplasty. Whereas hypoperfusion in the entire left MCA distribution was observed preoperatively on SPECT evaluation in this patient, following angioplasty a perfusion deficit was observed only in the left parietal region. One patient (Case 4) did not undergo SPECT evaluation.

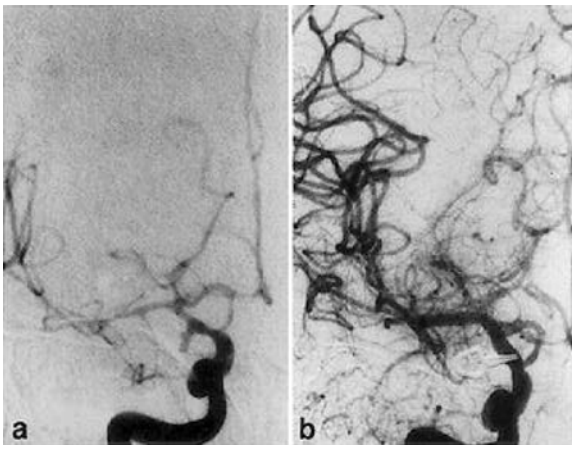

FIG. 1. Right carotid angiograms, anteroposterior view, in Case 2. a: Preoperative angiogram, demonstrating moderate spasm of the right supraclinoid internal carotid artery (ICA), severe spasm in the right M_1 and M_2 segments of the middle cerebral artery, and a posterior communicating artery aneurysm. b: Postoperative and postangioplasty angiogram demonstrating obliteration of the aneurysm and improved vessel caliber of the ICA and the M_1 and M_2 segments.

Clinical Outcome

Four of the five patients undergoing immediate postoperative balloon dilatation for vasospasm demonstrated immediate and sustained clinical improvement and had a favorable outcome at long-term follow-up review (Table 1). Amelioration of the neurological deficit was generally observed within hours after the procedure. The remaining patient (Case 4) developed bilateral anterior cerebral artery infarcts after an unsuccessful attempt to dilate both A_1 segments and subsequently died.

Illustrative Cases

Case 2

This 58-year-old woman suffered an SAH from a ruptured right posterior communicating artery aneurysm. Seven days later she developed progressive left-sided weakness and was admitted to Harborview Medical Center. Examination confirmed a left hemiparesis with 1/5 distal motor function in both the upper and lower extremities. A head CT scan demonstrated mild ventricular enlargement and subarachnoid blood, particularly in the right sylvian fissure. Cerebral angiography demonstrated a right posterior communicating artery aneurysm, moderate spasm of the right supraclinoid carotid artery, and severe spasm of the right M_1 and M_2 segments of the MCA (Fig. 1a). The right A_1 segment of the anterior cerebral artery filled from the left side. A perfusion defect in the right frontotemporal

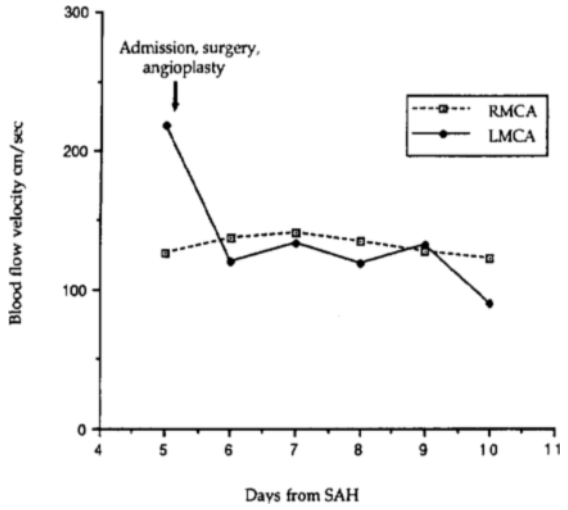

FIG. 2. Graph illustrating blood flow velocities recorded by transcranial Doppler ultrasound in Case 5. Left middle cerebral artery (LMCA) velocities demonstrate a sustained decrease in velocities following angioplasty. RMCA = right middle cerebral artery.

region was identified by SPECT evaluation. At surgery, a ventriculostomy was placed and the aneurysm obliterated with a clip. Severe attenuation of the ICA and M_1 vessel caliber was observed. These vessels were bathed in papaverine, and no response was seen. Following surgery there was no clinical improvement. The severity of spasm remained unchanged on the postoperative angiogram.

Angioplasty was successfully performed in the right supraclinoid carotid artery and M_1 and M_2 segments (Fig. 1b). The right A_1 segment could not be dilated. Within hours of the procedure, full motor strength returned and improved perfusion was documented by SPECT. When angiography was repeated 7 days later, there was mild narrowing of the right supraclinoid carotid artery but normal M_1 and M_2 segments. The patient later required a ventriculoperitoneal shunt. At 6 months she had returned to work but was moderately disabled because of mild left-hand clumsiness.

Case 5

This 39-year-old man suffered a severe headache and seizure. Five days later he developed a right hemiparesis and dysphasia and was transferred to Harborview Medical Center. A head CT scan demonstrated SAH in the left sylvian fissure. Transcranial Doppler blood flow velocities were markedly elevated (MCA:ICA ratio 8.76:1, Fig. 2). Angiography revealed a left MCA aneurysm and severe spasm in the left ICA and MCA (Fig. 3a). Hypoperfusion in the left frontal, temporal, and parietal regions was observed on SPECT examination. Following a craniotomy and aneurysm occlusion, the left ICA and MCA were successfully dilated by angioplasty (Fig. 3b and c). The patient demonstrated immediate clinical improvement following the procedure and regained the ability to move his right side; however, he continued to have mild speech difficulties. Two days after angioplasty, a follow-up CT scan demonstrated loss of the gray-white junction in the posterior temporoparietal region. A SPECT evaluation revealed a perfusion deficit compatible with the abnormality demonstrated on CT but improved perfusion in the frontal region. Sequential TCD blood flow velocity recordings demonstrated sustained improvement (Fig. 2); the left MCA:ICA ratio was 4.44:1 on the day after angioplasty. The patient was moderately disabled at his 6-month follow-up evaluation.

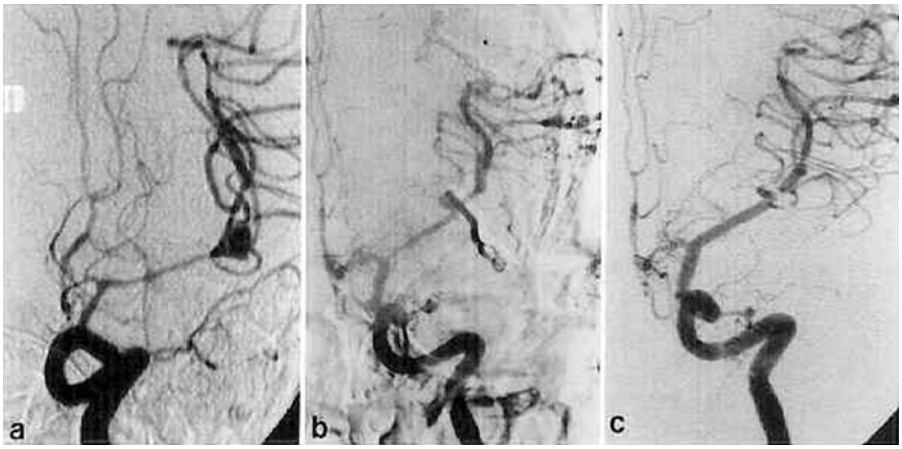

FIG. 3. Left carotid angiograms, anteroposterior view, in Case 5. a: Preoperative angiogram demonstrating moderate internal carotid artery (ICA) and severe middle cerebral artery (MCA) spasm and an MCA aneurysm. b: Angiogram obtained following surgery. The aneurysm has been occluded, but severe spasm persists in the left MCA. c: Postangioplasty angiogram demonstrating return of the normal luminal diameter after dilatation of the ICA and MCA.

Discussion

In this study, we report our experience in the management of five patients who presented with severe symptomatic vasospasm after SAH and an unsecured ruptured aneurysm. Rather than delay surgery, we performed aneurysm clipping and immediate postoperative angioplasty. Four patients demonstrated lessening of their vasospasm and sustained clinical improvement.

Vasospasm and Timing of Surgery

It is often contended that surgical manipulation of cerebral blood vessels intensifies the arterial narrowing visible after SAH and thus may precipitate ischemic stroke.[30,36] Recent experimental and clinical observations, however, do not support this hypothesis.[1,4,17,18,28,35] For example, Findlay, *et al.*,[4] were unable to demonstrate angiographically any exacerbation of arterial narrowing after mechanically manipulating vasospastic vessels in a primate model of vasospasm. These results are consistent with those of other experimental studies, both *in vitro* and *in vivo*, demonstrating the attenuated reactivity of cerebral arteries to vasoactive substances in vessels chronically exposed to periadventitial blood.[1,17,18] In clinical studies, Solomon, *et al.*,[35] did not find a correlation between infarct due to delayed cerebral ischemia and the timing of surgery, provided that postoperative prophylactic hypervolemic therapy was instituted early. Furthermore, in xenon studies evaluating cerebral perfusion after SAH, it was found that surgery did not have a detrimental effect on cerebral blood flow.[21,28] Thus, the poor results and ischemic events that surgeons have observed after operating in the presence of vasospasm may result from other factors, such as hypotension, dehydration, or hypoxia encountered during surgery. We therefore postulate that aneurysm clipping followed by immediate postoperative angioplasty for patients presenting with severe symptomatic vasospasm and an unsecured aneurysm is feasible and may be a reasonable management approach to these patients.

Angioplasty and Vasospasm

Many pharmacological and medical therapies have been used to avert or attenuate the severity of arterial narrowing following SAH.[39] Although clinical benefits have been observed with some of these treatments, particularly calcium channel blockers and hypervolemic therapy,[29,34] none is consistently effective once a neurological deficit develops. By contrast, recent experience with percutaneous transfemoral angioplasty indicates that this procedure can benefit a subgroup of patients who continue to deteriorate clinically despite maximum medical therapy, and in many instances can reverse an ischemic neurological deficit after it has developed.[3,9,10,21,24,25,37]

Based on our experience,[3,21,24,25] we believe that angioplasty should be attempted only if an ischemic neurological deficit fails to respond to maximum medical and pharmacological therapy. There are numerous technical factors that influence safety, but two factors are critical to the clinical success of the procedure. 1) The ruptured aneurysm should be occluded before angioplasty is attempted.[3,20,21,24,25] 2) Most studies indicate that the likelihood of reversing an ischemic neurological deficit is enhanced by early intervention.[3,10,21,24,25,37] As our experience with balloon dilatation has increased, we have expanded our indications to include symptomatic patients in whom volume expansion and hypertension may not be safe, such as patients with an unsecured ruptured aneurysm. These patients undergo prompt operative occlusion of the ruptured aneurysm and then proceed directly to angioplasty, rather than incurring further delays. The clinical success of the procedure, however, depends on both balloon dilatation of the narrowed arterial segment and careful hemodynamic management in the perioperative period.

Ultrasonography and SPECT

We have found that TCD and SPECT are useful in deciding which patients may benefit from immediate postoperative balloon dilatation. The use of TCD studies allows frequent analysis of blood flow velocities, which correlate with the severity of vasospasm. A blood flow velocity greater than 200 cm/sec or an MCA:ICA ratio greater than 6:1 is invariably associated with severe symptomatic vasospasm.[6,13,23,24,33] The use of SPECT provides both an assessment of regional cerebral blood flow and a measure of the degree of compensation in the microcirculation in the presence of proximal arterial narrowing.[19,24] While these additional tests are useful, the final decision to perform immediate postoperative angioplasty is based on repeated postsurgical neurological evaluation and CT to exclude cerebral infarction. This requires close cooperation with the neuroanesthesiologist to allow a reliable neurological examination at the conclusion of surgery.

In summary, this study demonstrates that immediate postoperative angioplasty is possible in patients who present with severe symptomatic vasospasm and an unsecured ruptured aneurysm. Despite severe vasospasm, worsening focal deficits, and poor neurological grade, four of the five patients survived surgery and made moderate to good recoveries. This report does not imply that surgery in the presence of severe vasospasm is without risk, but it does find that this procedure offers a reasonable chance of neurological recovery to this subgroup of patients who might otherwise progress to disabling cerebral infarction.

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