Angioplasty for Symptomatic Vasospasm

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A protocol has been developed for angioplasty in patients with symptomatic vasospasm refractory to conventional therapies. This protocol involves early surgery with hypervolemic therapy, calcium-channel blockers, and ICP and transcranial Doppler (TCD) monitoring. In symptomatic patients who fail this therapy, transluminal angioplasty has been performed using high-resolution digital fluoroscopy and low-pressure silicon balloons. For the first 21 patients treated with angioplasty, marked improvement in neurologic status was observed within 48 hours for 15 patients. Among those patients not improving, 2 were treated more than 72 hours after onset of symptoms or had lesions which were inaccessible to the angioplasty catheter. Two patients with unsecured aneurysm at the time of the procedure subsequently had subarachnoid hemorrhage at 3 and 7 days after angioplasty. Among 15 patients who improved after angioplasty, clinical improvement corresponded to immediate decrease in TCD velocity which persisted over time. Follow-up angiography at periods from 1 week to 18 months has shown no recurrence of the vasospasm or evidence of arterial injury. Angioplasty represents a potentially important therapy for the treatment of symptomatic vasospasm refractory to conventional therapies.

I. INTRODUCTION

Despite advances in the medical therapy of cerebral vasospasm after subarachnoid hemorrhage (SAH), a certain proportion of patients remain refractory to optimal therapy. In this subgroup of patients, clinical outcome remains poor, with death and disability primarily related to cerebral ischemia secondary to delayed arterial narrrowing. Transluminal balloon angioplasty involves the mechanical dilation of a vessel at a segment of narrowing through the use of an inflatable intravascular balloon. This technique has previously been applied to the dilation of coronary and peripheral vascular arteries in the setting of athrosclerosis. In 1984, Zubkov et al (1), first reported the treatment of cerebral vasospasm after SAH using transluminal balloon angioplasty. Subsequent reports (2, 3) have similarly described the application of this technique, in which generally favorable results were obtained in patients with symptomatic vasospasm refractory to conventional therapy. This report describes our experience in 21 patients treated using this technique at the Universtiy of Washington.

II. MATERIALS AND METHODS

During the period July, 1988 through April, 1990, 160 patients with angiographically verified aneurysmal subarachnoid hemorrhage were treated at the University of Washington hospitals. Patients were treated whenever possible according to a standardized protocol. Immediately upon admission, radiologic evaluation consisted of

CT Scan, contrast infusion CT Scan and four vessel angiography. Patients in all grades underwent surgery and clipping of the aneurysm responsible for SAH as soon as possible (usually within 72 hours of hemorrhage). In the immediate post-operative period and throughout the first 7 to 14 days after SAH, patients were treated in an Intensive Care Unit with continuous monitoring of hemodynamic parameters (via Swan-Ganz catheter), intracranial pressure, transcranial Doppler, neurologic status and frequent CT Scans. Hypervolemic therapy was initiated prophylactically in the immediate post-operative period using colloid, crystalloid and blood replacement to maintain the left ventricular and diastolic pressure (LVEDP) in the range of 10-15mm Hg. Ventricular drainage was instituted as necessary and elevated intracranial pressure was treated when present using hyperosmolar agents while maintaining LVEDP and cardiac output. When TCD or clinical evidence of impending vasospasm was present, patients were treated with additional volume expansion and augmentation of cardiac output using inotropic agents. In addition, all patients with altered neurologic status underwent CT Scan to rule out other etiology for the change.

The following guidelines were established for patient selection for transluminal angioplasty. These criteria included (a) new onset of a neurologic deficit not attributable to other causes (hydrocephalus, hematoma, mass effect, etc.); (b) no evidence of infarction on recent CT Scan; (c) neurologic deficit not reversed by hypervolimic/hypertensive therapy; and (d) angiographically apparent vasospasm in a vascular distribution corresponding to the clinical deficit. In nearly all cases, vasospasm according to the above criteria was confirmed by the presence of increased velocities by TCD in the affected vessels. In the early segment of this study, symptomatic vasospasm was treated by angioplasty at various intervals after onset. The initial results suggested, however, that delay greater than 48 hours after onset of symptoms was associated with the poor result. Subsequently, in the latter 15 patients reported, angioplasty was instituted within less than 48 hours. Similarly, among the first group of patients, angioplasty was performed regardless of whether the aneurysm had been surgically obliterated. However, based upon re-bleeding after angioplasty in two cases (below), we have subsequently limited the procedure to only those patients in whom the aneurysm has been successfully clipped.

Two varieties of custom-made angioplasty microballoon have been utilized in these patients. A polyethylene microballoon (Target Therapeutics Corp, San Jose, CA) has an inflated diameter of 2.5mm and a length of 15mm. This balloon has a higher inflation pressure (5 ATM) and uses a steerable microguide wire for navigation. The advantage of the polyethelene balloon is that it can be guided into difficult branches, especially those such as the A-1 segment of the anterior cerebral artery which frequently arises at a right angle to the parent ICA. The second type of microballoon is made of silicone (Interventional Therapeutics Corp, South San Fransicso, CA) and measures 3mm by 12mm when fully inflated. This balloon has a low inflation pressure (0.5 ATM), thus reducing the risk of vessel rupture or endothelial damage. The low-pressure inflation of this balloon is offset by the lack of a steerable guidewire, and thus its use is limited in certain vessels. Both the polyethelene and silicone balloons are attached to a 150 cm variable stiffness microcatheter for transfemoral catheterization. Although larger ballons are available, these are potentially hazardous due to the risk of overdistension and vessel rupture, and are probably not necessary for intracranial arteries.

The angioplasty procedure is performed using high resolution digital fluoroscopic equipment with road-mapping capability to prevent inadvertent insertion of the balloon into side branches. Patients are fully heparinized during the procedure, with protamine reversal at its conclusion. In cooperative or comatose patients, neuroleptic analgesia is used; otherwise, general anesthesia is employed. We now dilate all vessels demonstrating angiographic narrowing at the time of angioplasty, regardless of whether the arterial narrowing is asymptomatic at that time. Following the procedure, all patients are returned to the Neurosurgical Intensive Care Unit for continuous monitoring as above. Repeat angiography is performed immediately at the conclusion of the angioplasty procedure, and at one week post-angioplasty. In two patients, follow-up angiograms have been obtained at 6 and 18 months following angioplasty.

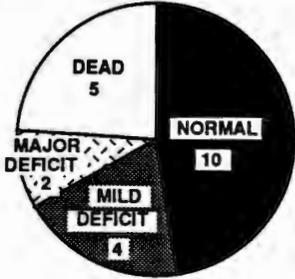## III. RESULTS

OUTCOME AFTER ANGIOPLASTY

Figure 1

Fifteen of 21 patients who underwent balloon angioplasty according to the above criteria showed sustained improvement following the procedure (Figure 1). Improvement was defined as either (a) a two-point increase on the Glascow Coma Scale (GCS), or (b) an improvement of two grades of motor strength within 48 hours of the procedure. Both criteria for improvement were used because either scale alone may not necessarily reflect the improvement in clinical status observed, e.g. a resolution of focal motor deficit without change in level of consciousness. In a majority of those patients showing clinical improvement, neurologic deficits were reversed or ameliorated immediately or within a few hours after angioplasty. The remaining deficits resolved over 12 to 48 hours. In no cases did deficit recur in the distribution of a dilated artery, although in one patient clinical vasospasm developed in the distribution of a vessel not dilated at the time of angioplasty.

Among the 7 patients with poor outcomes, 5 deaths occured. Two patients in the early part of the series died from recurrent hemorrhage from unclipped aneurysms. One patient re-bled one week after angioplasty while awaiting balloon embolization of an unclippable aneurysm. The second re-bleed occured in a patient with a mycotic posterior cerebral artery aneurysm who had initially improved but succumbed to his second hemorrhage at 72 hours after angioplasty. Three other deaths occured in patients who were moribund at the time of angioplasty, one of whom was not treated until 72 hours after onset of symptoms. In one patient without improvement, a prior cervical carotid artery occlusion precluded effective angioplasty. A single late complication of the procedure was due to delayed occlusion of a left middle cerebral M2 branch at six weeks following angioplasty. This particular vessel had been dilated with a high-pressure silicone balloon which ruptured during dilation, presumably damaging the endothelium and predisposing to vessel occlusion. A mild hemiparesis referable to the MCA branch occlusion cleared completely within 7 days and there was no permanent deficit.
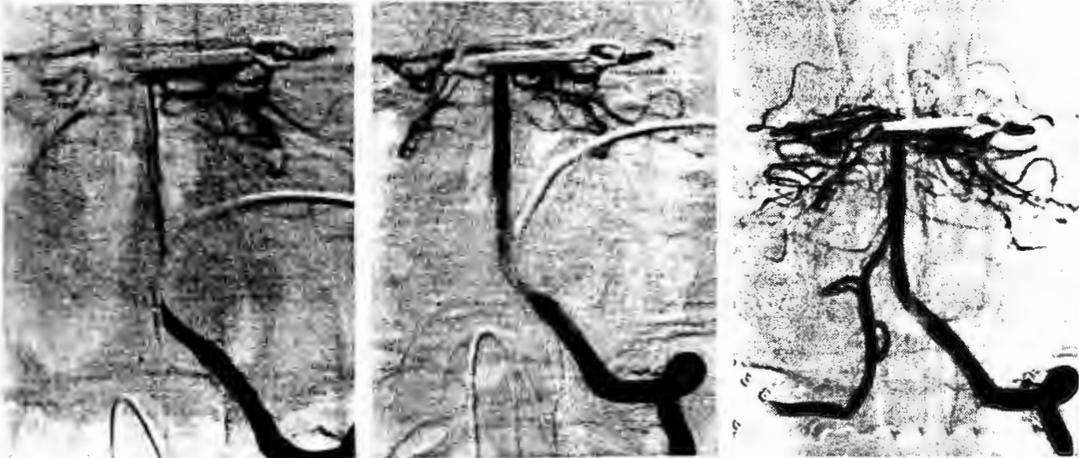

Figure 2: Subtraction anteroposterior vertebrobasilar angiograms in patient with symptomatic vasospasm on post-bleed day 6 (left), immediately following angioplasty (center), and 18 months after angioplasty. (right) Restoration of basilar and posterior artery caliber was accompanied by rapid improvement in clinical status.

Long term follow-up for periods from 2-18 months was available in all 17 surviving patients. Clinical improvement referable to angioplasty has persisted in all patients (except the MCA occlusion described above) during this period of follow-up. Repeat angiogram in 2 patients at 6 and 18 months following angioplasty showed normal vessel caliber without irregularity in the dilated segment (Figure 2).

TCD monitoring similarly demonstrated the immediate and persistent reduction in flow velocities in the dilated vessels folowing angioplasty. In most cases, elevated flow velocities were immediately diminshed to within the normal range and remained so for the duration of the perioperative period, corresponding to improvement in clinical status.

IV.DISCUSSION

In patients with symptomatic vasospasm refractory to conventional therapy, transluminal balloon angioplasty produced a profound rapid improvement in a majority of cases, which persisted over time (2,3). Neurologic improvement correlated with long-term increases in arterial diameter by angiography and increased flow velocities by transcranial Doppler (3). The effectiveness of angioplasty in the treatment of vasospasm substantiates the hypothesis that narrowing of large basal conducting arteries is the primary cause of cerebral ischemia in this disorder, rather than effects of subarachnoid blood upon smaller collateral arteries or brain parenchyma. In addition, the immediate and persistent resolution of arterial narrowing after angioplasty implies that prolonged vasoconstriction from perivascular vasoactive agents may not play a significant role in chronic vasospasm. Bevan, et al (4) have shown that primate cerebral arteries chronically exposed to subarachnoid blood become less distensible and less sensitive to vasoactive agents in vitro. In addition, ultrastructural examination of human (5) and animal (6) cerebral arteries after SAH has demonstrated the existence of increased collagen deposition in the arterial wall. It has been proposed that mechanical dilatation from angioplasty disrupts the collagenous matrix of the inelastic fibrotic artery in vasospasm (3).

V. REFERENCES

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