

Treatment of Cerebral Vasospasm with Transluminal Angioplasty

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1. Abstract

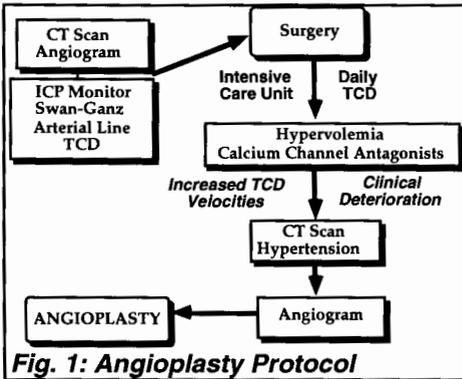
In the last five years, we have performed transluminal angioplasty on 139 vessels in 50 patients who developed a delayed ischemic deficit after subarachnoid hemorrhage that was not responsive to hypervolemic hypertensive therapy and calcium channel antagonists. Angiographic resolution of vasospasm was achieved in 98% of vessels in which balloon dilatation was attempted. Improvement in cerebral perfusion, which correlated with clinical improvement, was demonstrated by SPECT and TCD evaluation in the majority of patients. Thirty two patients (64%) demonstrated sustained clinical improvement and had favorable outcomes at six month follow up. Early intervention and the prior obliteration of the aneurysm were critical to the successful outcome of this procedure. The overall results of this series indicate that in selected cases, balloon dilatation of arteries with delayed narrowing after SAH can offer marked improvement for patients with an ischemic deficit refractory to conventional modalities of therapy.

2. Introduction

The inconsistent results of medical therapies for vasospasm are in part related to their lack of efficacy once a neurologic deficit occurs. By contrast, recent experience with percutaneous transluminal angioplasty indicates that this technique can benefit a subgroup of patients who demonstrate progressive clinical deterioration, despite maximal medical therapy, and reverse a delayed ischemic deficit after it has developed (1,2,3,5,7,8). The present report describes the use of this technique in fifty patients with delayed ischemic deficits following subarachnoid hemorrhage.

3. Clinical Materials And Methods

During a five year period (June 1988-December 1992), 50 patients admitted to Harborview Medical Center and the University of Washington Affiliated Hospitals after aneurysm rupture underwent percutaneous angioplasty for symptomatic vasospasm. Subarachnoid hemorrhage was confirmed by computerized tomography (CT) and the presence of an aneurysm diagnosed by four vessel angiography. Surgery and obliteration of the aneurysm responsible for the SAH were performed as soon after rupture as possible (within 72 hours). For the first 10 days after SAH, patients were managed in the intensive care unit with continuous monitoring of hemodynamic parameters (arterial and Swan Ganz catheters), intracranial pressure, daily transcranial Doppler (TCD) and sequential single photon emission computed tomography (SPECT) evaluation. Aggressive volume expansion and hemodilution were initiated prophylactically and Nimodipine (60mg orally every 4 hours) was routinely administered.



The decision to perform angioplasty was based on the following criteria: 1) a new neurologic deficit, such as motor weakness or 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 and 4) severe angiographic vasospasm in a location that could be responsible for the ischemic deficit (Fig. 1). Angioplasty was performed by the transfemoral percutaneous approach using a silicone microballoon

(Interventional Therapeutics Corp., San Francisco, CA) attached to a variable stiffness microcatheter (Target Therapeutics, San Jose, CA) under general or neuroleptic analgesia. After completion of the procedure, an angiogram was obtained and hypovolemic therapy, calcium channel blockers and intensive monitoring were continued. Angiography was repeated one week after the procedure. Patients were considered to have responded to angioplasty if they improved at least two Glasgow Coma Scale points or demonstrated two grades of motor improvement within 24 hours of the procedure. Long term follow up data was obtained at six months by telephone interview and/or clinic visit. The patients best level of function was graded according to the Glasgow Outcome Score.

4. Results

TABLE 1: RESULTS OF ANGIOPLASTY FOR REFRACTORY SYMPTOMATIC VASOSPASM

PATIENTS	VESSELS	ANGIOGRAPHIC IMPROVEMENT	CLINICAL IMPROVEMENT	COMPLICATIONS
50	139	136/139 (98%)	32/50 (62%)	Occlusion-1 Rebleeds-3 Vessel rupture-1

4.1 Angiographic results: The angiographic resolution of vasospasm was achieved in 98% of all vessels in which dilatation was attempted. In many cases after the proximal segment was dilated, distal narrowing resolved. By contrast, untreated vessels remained in spasm. Followup angiograms, including three obtained 18 months after the procedure, demonstrated that increases in vessel caliber obtained by mechanical dilatation were persistent in both short- and the long-term follow-up. Technical difficulties were encountered in the following circumstances. First, very severe narrowing occasionally prevented balloon entry. This was overcome by advancing the guidewire preceding the balloon. Second, access to vessels forming sharp corners, e.g. the A1 takeoff, was often limited. Again, a guidewire or curved balloon sometimes improved entry into these vessels. Third, after one vessel of a bifurcation or trifurcation was dilated it hampered entry into the other branches, as preferential flow and vessel shape directed the balloon into the first branch.

4.2 Cerebral perfusion: In addition to the restoration of normal vessel caliber, improved angiographic perfusion was observed following angioplasty; both TCD and SPECT studies correlated with improved cerebral perfusion (6). In patients demonstrating clinical improvement, TCD blood flow velocities decreased and remained below pre-angioplasty levels. By contrast, velocities continued to increase in untreated vessels, some of which became symptomatic at later times. Improvement in cerebral perfusion was demonstrated by resolution of perfusion defects on SPECT scans in the majority of patients.

4.3 Clinical outcome: Thirty two patients (64%) undergoing balloon dilatation for vasospasm demonstrated immediate and sustained clinical improvement and had favorable outcomes at long term follow up (Table 1). Amelioration of the neurological deficit was generally observed within minutes to hours after the procedure and was best achieved with early intervention. Four patients died (8%) from complications related to angioplasty. The remaining 14 patients did not demonstrate clinical improvement, despite angiographic resolution of spasm in most instances. Most of the patients who failed angioplasty were in poor-grade groups (Hunt and Hess grades IV and V) at admission.

4.4 Complications: Five complications, four of which resulted in death, were encountered early in this series. Three unsecured, previously ruptured aneurysms rebled following angioplasty. A single vessel rupture occurred from overdistension of the balloon. One patient developed a delayed branch occlusion of a dilated vessel and suffered a mild stroke. This vessel had been dilated with a more rigid balloon which is no longer in use.

5. Discussion

In this study we report our experience with 50 patients who underwent angioplasty for symptomatic vasospasm after aneurysm rupture. Thirty two patients demonstrated sustained clinical improvement and had a favorable outcome at long term follow up. The excellent results in this group of patients are particularly significant in comparison to the usually poor outcome observed in patients with vasospasm refractory to other treatments. Apart from technical considerations, three factors are critical to the success of this procedure. First, the aneurysm should be secured, before attempting angioplasty, to prevent rehemorrhage (1,2,3,5,7,8). Second, the most dramatic clinical improvements are observed when the procedure is performed within 18 hours of the onset of symptoms (1,2,3,5,7,8). Finally, angioplasty is not intended to be used alone, but rather should supplement other treatment modalities to optimally treat vasospasm. Therefore, in our protocol all patients receive prophylactic hypervolemia and calcium channel blockers, even if asymptomatic. In addition, sequential TCD evaluation and regular SPECT examination provides a means to diagnose and follow the development of vasospasm, and in particular, predict the likelihood of symptomatic vasospasm.

The overall results of angioplasty indicate that in selected cases, balloon dilatation of arteries with delayed narrowing after SAH can offer marked improvement for patients with an ischemic deficit refractory to conventional modalities of therapy. Despite the success of this new technique, many issues remain unresolved. Little is known regarding the mechanism by which angioplasty affects cerebral arteries in spasm or the optimal time to offer treatment. The role for prophylactic angioplasty in patients with significant asymptomatic spasm remains

uncertain. The patient with severe symptomatic spasm and an unclipped, ruptured aneurysm presents a challenging clinical dilemma. Whether angioplasty affects only large capacitance arteries at the Circle of Willis or indirectly augments flow through smaller perforating arteries has not been determined. Similarly, it is not clear whether successful restoration of proximal flow by angioplasty always results in improved distal perfusion. Finally, new adjuncts to the endovascular treatment of vasospasm such as local intra-arterial papaverine infusion (4) may provide additional means to treat these patients.

As further experience is accumulated, the role of angioplasty in vasospasm will be further defined and better understood. Current experience indicates that angioplasty is a viable therapeutic tool in the comprehensive management of SAH and can lead to dramatic neurologic improvement in select patients who otherwise would progress to a severe and disabling stroke or death.

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