Intracranial stenting: Which patients and when?

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A significant cause of ischemic stroke is intracranial atherosclerotic disease caused by either hypoperfusion or distal embolization. The incidence has been reported to be 6% to 10% in whites, 6% to 22% in blacks, 11% in Hispanics, and 11% to 22% in Asians. Prognosis and morbidity for patients with intracranial stenosis vary widely, with the morbidity rate ranging from 10% to 46% per year, independent of medical therapy.[1] The surgical option of an extracranial-to-intracranial bypass procedure has not been shown to be of significant benefit over optimal medical therapy for these patients.[2]

Over the past decade, a number of centers have been reporting their experience with intracranial angioplasty and stenting as a treatment option for patients in whom maximal medical therapy with antiplatelet and anticoagulant medications has failed. Although prospective randomized studies have not yet been performed, results from these centers have indicated that this procedure is technically feasible and that there are good preliminary data demonstrating efficacy.

PROCEDURE AND TECHNIQUE FOR INTRACRANIAL ANGIOPLASTY/STENTING

A baseline brain CT or MRI scan is initially performed to assess for evidence of cerebral ischemia, infarction, or both. Hemodynamic quantitative blood flow studies using CT xenon perfusion imaging, MR perfusion/diffusion imaging, nuclear medicine perfusion imaging, or positron emission tomography are also performed to assess the degree of perfusion to brain tissue. All patients undergo a four-vessel diagnostic cerebral arteriogram to determine the site and degree of stenosis, collateral circulation, and associated vascular pathology.

Patients then receive systemic anticoagulation with intravenous heparin (100 units/kg), and the lesion is carefully crossed under fluoroscopic guidance with a microguidewire (0.014 inches) and a balloon angioplasty catheter (2.0 to 4.0 mm in diameter) that matches the normal luminal diameter. The balloon is inflated for 5 to 10 seconds across the lesion until the plaque is sufficiently dilated. In most cases, a metallic stent is then placed across the lesion to further improve the luminal diameter and to reduce the incidence of vessel dissection with secondary restenosis.

Patients are then carefully monitored in the neurologic intensive care unit for 24 to 48 hours, with close attention paid to anticoagulation levels and blood pressure levels. They are then discharged on antiplatelet medications: clopidogrel 75 mg/day or ticlopidine 250 mg twice a day for 4 to 6 weeks, plus aspirin 325 mg/day indefinitely.

RESULTS TO DATE

In 1996, Higashida et al[3] published their early experience in 33 patients treated with intracranial balloon angioplasty after failure of best medical therapy; they reported a 69.7% technical success rate with improved neurologic outcome, although there was a 30.3% rate of associated stroke and death. Clark et al[4] reported a series of 17 patients in whom 22 vessels were treated with balloon angioplasty: the success rate was 72%, and the 30-day morbidity rate was 11.7%. Connors and Wojak[5] reported a retrospective analysis of balloon angioplasty in 70 patients with intracranial atherosclerosis: the overall stroke rate was 4.2%, the mortality rate was

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2.9%, and there were no technical failures.

More recently, several centers have reported on the use of intracranial stents for these lesions. Among 10 patients with 12 intracranial atherosclerotic lesions, Mori et al[6] reported an 80% technical success rate in accessing the lesion with a stent; in those patients who received stents, there were no periprocedural complications and there was significant improvement of neurologic symptoms during the 8 to 14 months of follow-up. Gomez et al[7] reported a series of 12 patients who underwent elective stenting of the basilar artery after episodes of vertebrobasilar ischemia; medical therapy had failed in all of these patients. Stent placement was successful in all cases, with improvement in luminal diameter from a mean of 71.4% to a mean of 10.3%, without any procedural complications. Clinical follow-up at 0.5 to 16 months (mean, 5.9 months) demonstrated no new complications, clinical improvement in all patients, and residual symptoms in only 2 patients. The researchers concluded that intracranial stenting was feasible and posed minimal risk to the patient, but its long-term impact was still not known.

■ DISCUSSION

In patients suffering from medically refractory transient cerebral ischemia, stroke, repetitive strokes, or other focal neurologic deficits stemming from intracranial symptomatic atherosclerotic lesions, intracranial balloon angioplasty, stenting, or both may be a useful therapeutic procedure. The development of better balloon catheters and stent delivery systems has dramatically reduced the technical difficulties and failures previously associated with these intracranial techniques. Although best medical therapy has not yet been determined for these patients, extrapolation from the extracranial circulation for carotid atherosclerotic disease indicates that if a direct surgical or endovascular revascularization procedure can be performed with acceptable technical success rates (ie, low rates of periprocedural complications), it may be better than medical therapy in certain types of patients. Clearly, once medical therapy with antiplatelet and/or anticoagulant medications has failed in a patient, then either an endovascular procedure or a surgical bypass procedure may be indicated as a possible alternative.

Although long-term follow-up (> 2 to 5 years) is not yet complete for patients who have undergone intracranial balloon angioplasty and/or stenting, the short-term results appear to be encouraging in terms of improving symptoms and decreasing the risk of major stroke.

As medical therapies and endovascular treatment techniques both continue to improve, hope remains that ever better treatment options will be available for patients with intracranial atherosclerotic lesions.

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