Intracranial Percutaneous Transluminal Angioplasty for Arteriosclerotic Stenosis

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Background: Patients with intracranial arteriosclerotic disease have significant morbidity and mortality rates, and some are unresponsive to medical treatment and have unacceptable surgical risks. Percutaneous transluminal angioplasty of the intracranial vessels is a possible alternative to surgery.

Objectives: To present our experience with percutaneous transluminal angioplasty and to summarize our data.

Patients and Methods: Sixteen patients underwent intracranial percutaneous transluminal angioplasty for high-grade arteriosclerotic stenosis based on strict inclusion and exclusion criteria. All patients had symptoms referable to the stenosis except one. Angioplasty was performed in 6 intracranial vertebral arteries, 3 basilar arteries, 5 middle cerebral arteries, and 3 distal internal carotid arteries. One patient had concomitant stent placement.

Results: There was 1 treatment failure secondary to tortuous vascular anatomy. Vessel caliber was increased to more than 80% of normal in 6 patients and to 50% to 70% of normal in 6 patients, with a reduction of symptoms. Three intimal dissections occurred during angioplasty; one of these, in a pre-cavernous segment of the internal carotid artery, was stented. One patient restenosed within 1 month of treatment. The remaining treated arteries remained patent during follow-up of 3 months to 2 years. Stroke as a complication occurred in 2 patients, 1 mild and 1 severe. There was no mortality.

Conclusions: Occlusive arteriosclerotic disease involving the intracranial cerebral vessels can be managed medically with antiplatelet and anticoagulant drug therapy or surgically. However, in patients who are unresponsive to medical therapy or who have unacceptable surgical risks, percutaneous transluminal angioplasty is an attractive alternative that can be performed in selected patients with relatively low risk and good clinical outcome.

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PERCUTANEOUS transluminal angioplasty (PTA) was first performed by Dotter and Judkins1 for femoral artery stenosis using a coaxial catheter system, and PTA is now an established therapy for renal, coronary, and iliac artery stenosis. In 1980 Sundt et al2 first reported successful intracranial angioplasty of the basilar artery in 2 patients.

Recent improvements in microwire and microballoon catheter technology have resulted in PTA being used as treatment for symptomatic hemodynamically significant focal stenosis lesions in the extracranial and intracranial circulation. Patients in whom intracranial PTA has been used are often unresponsive to medical therapy and are considered to be at high surgical risk. Despite its experimental status, multiple medical centers around the world have reported that the technique is relatively safe in selected patients.

We present our 5-year experience with 16 patients with intracranial artery stenosis who met specific criteria and were treated with PTA after failing medical therapy.

RESULTS

The clinical findings and neuroangiographic features of patients before and after angioplasty are given in the Table, and angiograms of patients 5, 7, 11, and 14 are presented in Figures 1, 2, 3, and 4, respectively.

VERTEBRAL ARTERY ANGIOPLASTY

Five patients were treated for intracranial vertebral artery (V4 segment) stenosis. There were no complications related...
PATIENTS AND METHODS

PATIENTS

All intracranial PTAs performed at the University of Illinois at Chicago between April 1994 and April 1999 were reviewed. There were 16 patients (13 men and 3 women), aged 38 to 77 years, in whom angioplasty was attempted. Percutaneous transluminal angioplasty was technically successful in all but 1 patient, in whom vessel tortuosity prevented us from navigating the balloon catheter into the cavernous internal carotid artery (ICA). The initial clinical presentation was transient ischemic attacks in 12 patients and stroke in 3; 1 patient was treated prophylactically as a measure of stroke prevention before cardiac bypass surgery.

DIAGNOSTIC ANGIOGRAPHY

All patients underwent diagnostic neuroangiography including aortic arch and 4-vessel cerebral subtraction angiography of the cervical and intracranial portion of the carotid and vertebral arteries. Four-vessel angiography was performed to evaluate the pial collateral supply to the stenotic vessels, potential tandem stenotic lesions, and the length and diameter of the stenotic segment. The degree of stenosis was measured by the ratio of the luminal diameter at the level of the stenosis (by taking the smaller diameter on either the anteroposterior or lateral projection) to the luminal diameter immediately proximal to the stenosis. After documentation of symptomatic intracranial stenosis, all patients except 1 were treated with medical therapy consisting of oral anticoagulant drugs and, in selected patients, antiplatelet agents. Patient 1 did not receive medical therapy because the angioplasty was performed for stroke prevention before cardiac bypass surgery.

SELECTION CRITERIA

Selection criteria for intracranial PTA include (1) stenosis of more than 60% secondary to arteriosclerotic disease and (2) transient ischemic attacks or stroke referable to the stenotic vessels despite medical therapy.

The stenotic intracranial vessels consisted of 3 intracranial ICAs, 5 (M1 or proximal M2 segment) middle cerebral arteries, 3 basilar arteries, and 6 intracranial vertebral arteries (V4 segment). One patient had a second angioplasty of the V4 segment. The only exclusion criteria in our protocol was in 1 patient with a large area of infarct seen on brain computed tomographic scans or magnetic resonance images. Informed consent was obtained from patients after fully explaining the 5% to 10% periprocedural risks of stroke or death. All patients were treated under general anesthesia to allow the use of effective simultaneous biplane digital roadmapping, which significantly decreased procedural time and the risk of technical complications.

PROCEDURE

A 7F sheath was placed in the femoral artery. Systemic anticoagulation was achieved with an initial bolus of 3000 to 7000 U of intravenous heparin sodium followed by an additional 1000 U/h throughout the procedure. Adequate anticoagulation was confirmed by an activated coagulation time of 250 to 300 seconds.

A 7F guiding catheter, either Envoy (Cordis, Miami, Fla) or 7F Platform (Target Therapeutics Corp, Fremont, Calif), was positioned with the tip in the distal cervical ICA or vertebral artery, depending on which vascular territory was to be angioplastied. A Stealth balloon microcatheter over 0.014-in microwires (Target Therapeutics Corp) was used for this procedure initially. In later cases, we used a Stratus balloon microcatheter (MIS, Sunnyvale, Calif), and in the most recent case, a VALOR coronary angioplasty balloon microcatheter (Cordis) was used. The tip of the 7F guiding catheter was placed at the level of the skull base in either the carotid or vertebral artery. Digital roadmapping in anteroposterior and lateral projections was performed. The balloon and guidewire combination was then passed through a rotating hemostat valve and attached to the guide catheter.

Balloon catheter diameter was always selected to be slightly smaller than the normal diameter of the angioplastied vessel. The choice of inflated diameter and length of balloon was determined by the width of the normal blood vessel and the length of the stenotic lesion.

Through the guiding catheter and with the guidance of the roadmap, the balloon catheter and microwire systems were navigated across the stenotic lesion. The balloon was inflated to 6 to 7 atm for 10 seconds; the maneuver might be repeated 2 or 3 times depending on the response of the stenotic lesion. The balloon was then deflated but left in position; contrast (100% Omnipaque 300) was injected through the guiding catheter to determine the need for additional dilation. After the balloon was removed, an angiogram was again performed to assess vessel stenosis and evidence of distal embolization.

POSTTREATMENT MANAGEMENT

Heparin administration was continued after the procedure, with the dose adjusted to maintain the partial thromboplastin time at 60 to 80 seconds. All patients were extubated in the neuroangiography suite. The femoral artery sheath was removed while the patient was still receiving heparin. All patients were monitored in the neurosurgical intensive care unit for at least 48 hours. A detailed neurological examination was performed immediately after angioplasty in the neuroangiography suite and then daily during hospitalization. All patients were clinically evaluated at 1 month.
## Clinical Findings and Neuroangiographic Features of Patients Undergoing Intracranial Percutaneous Transluminal Angioplasty

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age, y/Sex</th>
<th>Clinical Findings</th>
<th>Vessels Involved</th>
<th>Stenosis, %</th>
<th>Before Angioplasty</th>
<th>After Angioplasty</th>
<th>Complications</th>
<th>Clinical Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>77/M</td>
<td>For prevention of cerebellar stroke before emergent cardiac bypass</td>
<td>RVA:V4 segment</td>
<td>70</td>
<td>10</td>
<td>None</td>
<td>No stroke after cardiac bypass</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>68/M</td>
<td>Episode of diplopia and worsening headache; needed another session of angioplasty because of recurrence of symptom after 1 mo</td>
<td>RVA:V4 segment</td>
<td>60</td>
<td>30</td>
<td>None</td>
<td>Disappearance of symptoms</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>61/M</td>
<td>Dizziness, diplopia, dysarthria, and leg weakness</td>
<td>RVA:V4 segment</td>
<td>90</td>
<td>20</td>
<td>None</td>
<td>Minor dizziness at 2-y follow-up</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>57/M</td>
<td>Eyes and leg weakness and dizziness</td>
<td>RVA:V4 segment</td>
<td>90</td>
<td>30</td>
<td>None</td>
<td>Disappearance of TIAs at 4-y follow-up</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>76/M</td>
<td>Dizziness</td>
<td>RVA:V4 segment</td>
<td>80</td>
<td>30</td>
<td>None</td>
<td>Disappearance of dizziness improved except for progressive hearing loss at 4-mo follow-up</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>68/M</td>
<td>Quadriplegia, facial numbness, and bilateral visual and hearing loss</td>
<td>Proximal basilar</td>
<td>80</td>
<td>50</td>
<td>None</td>
<td>Disappearance of symptom at 3-mo follow-up</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>59/M</td>
<td>Left facial paresthesia, slurred speech, and increased difficulty walking</td>
<td>Proximal basilar</td>
<td>80</td>
<td>30</td>
<td>None</td>
<td>Disappearance of symptom at 3-mo follow-up</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>61/M</td>
<td>Dizzy spells and tingling of leg</td>
<td>LBJ</td>
<td>80</td>
<td>40</td>
<td>Small intimal dissection</td>
<td>Neurologically intact</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>63/M</td>
<td>Fluctuating level of consciousness and psychosis</td>
<td>RMCA—diffuse stenosis of other cerebral vessel, PCA supraclinoid, ICA</td>
<td>80</td>
<td>20</td>
<td>None</td>
<td>No clinical improvement</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>76/M</td>
<td>Multiple episodes of TIAs and dysphasia</td>
<td>LMCA</td>
<td>90</td>
<td>30</td>
<td>None</td>
<td>No more episodes of TIA; clinical improvement at 4-mo follow-up angioplasty</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>52/M</td>
<td>Multiple episodes of left-sided weakness and slurred speech and memory loss</td>
<td>RMCA</td>
<td>80</td>
<td>20</td>
<td>None</td>
<td>Slowly improving at 3-mo follow-up</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>68/M</td>
<td>Left arm weakness (TIA); another angiogram at 2-y follow-up showed restenosis of angioplasty segment</td>
<td>RMCA</td>
<td>90</td>
<td>20</td>
<td>None</td>
<td>Disappearance of symptoms at 2-y follow-up despite recurrence of stenosis</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>45/M</td>
<td>Left-sided hemiplegia from previous left cerebral hemispheric stroke</td>
<td>RMCA</td>
<td>&gt;90</td>
<td>Occlusion</td>
<td>Occlusion</td>
<td>Mild deterioration of left-sided hemiplegia and good pial collateral</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>61/M</td>
<td>Episodes of TIA, left-sided weakness, and slurred speech</td>
<td>RICA precavernous segment</td>
<td>90</td>
<td>10</td>
<td>Dissection; necessitated stenting the next day</td>
<td>Recurrence of same symptoms at 2-y follow-up</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>42/F</td>
<td>Left hemispheric stroke</td>
<td>LICA cavernous</td>
<td>&gt;95</td>
<td>Failed</td>
<td>Failed because of tortuosity of artery</td>
<td>. . .</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>38/F</td>
<td>Right-sided upper extremity weakness</td>
<td>LICA supraclinoid</td>
<td>&gt;95</td>
<td>Occlusion</td>
<td>Dissection with a shower of emboli to LMCA branches</td>
<td>Aphasia and right hemiparesis</td>
<td></td>
</tr>
</tbody>
</table>

*RVA indicates right vertebral artery; LVBJ, left vertebrobasilar junction; RMCA and LMCA, right and left middle cerebral artery, respectively; PCA, posterior communicating artery; RICA and LICA, right and left internal carotid artery, respectively; and TIA, transient ischemic attack.*
BASILAR ARTERY ANGIOPLASTY

Three patients were treated for symptomatic basilar artery stenosis: 2 had proximal basilar artery stenosis and 1 had left vertebrobasilar junction stenosis. The degree of stenosis was more than 80% in all patients. Vessel caliber was increased to more than 50% of normal, with disappearance of symptoms in 2 patients (patients 6 and 7) and no change in symptoms in the third patient (patient 8); average follow-up in these patients was 4 months. Patient 8 had a small intimal flap at the distal end of the angioplastied segment without adverse effects or change in symptoms.

MIDDLE CEREBRAL ARTERY ANGIOPLASTY

Five patients with symptomatic middle cerebral artery stenosis (M1 segment) were treated with PTA. The degree of stenosis was more than 80% in all patients. Vessel caliber was increased to more than 70% of normal in 4 patients. Disappearance or reduction of symptoms was achieved in 3 patients (patients 10-12), with follow-up of 4 months to 2 years. A fourth patient (patient 13) had vessel occlusion after PTA, resulting in mild deterioration of preexisting left-sided hemiparesis. Symptoms were unchanged in the fifth patient (patient 9) despite good angioplasty results. One patient (patient 12) developed restenosis of the angioplastied segment after 2 years but remained neurologically intact with no symptoms.

ICA ANGIOPLASTY

Three patients had symptomatic intracranial ICA stenosis. In 1 patient (patient 15), PTA was unsuccessful because of the inability to navigate the balloon catheter into the tortuous cavernous portion of the ICA. A second patient (patient 16) had more than 95% stenosis of the C2 portion of the left internal carotid siphon distal to the origin of the ophthalmic artery and proximal to the origin of the posterior communicating artery. This was complicated by dissection at the angioplasty site with a shower of emboli, resulting in occlusion of the left M1 segment of the middle cerebral artery. Fibrinolysis with 250,000 U of urokinase within 30 minutes was unsuccessful in dissolving the clot in the M1 segment. The patient developed aphasia and right-sided hemiplegia. The third patient (patient 14) had 90% stenosis of the precavernous right ICA complicated by dissection at the site of angioplasty, prompting the placement of a Palmaz stent. The patient remained symptom free for 2 years but then developed recurrent symptoms.
Hemodynamically significant stenosis of intracranial arteries can cause distal embolization with subsequent cerebral ischemia or infarction. Turbulent blood flow combined with loss of arterial intimal smoothness at the site of stenosis might initiate and propagate thrombosis with microembolism, resulting in cerebral ischemia or infarction.

Despite the significant morbidity and mortality resulting from symptomatic intracranial arterial stenosis, there are no established surgical or interventional therapies at this time. It is still controversial whether selected patients might benefit from an external to internal carotid arteries bypass procedure, and the indications are not as clear-cut as those for carotid endarterectomy (an established surgical treatment), which significantly reduces the risk of cerebral emboli and ischemia in patients with symptomatic atherosclerotic stenotic disease of the extracranial carotid artery with high-grade stenosis. Currently, anticoagulant and antiplatelet therapy is used for hemodynamically significant and symptomatic intracranial arterial stenosis.

Percutaneous transluminal angioplasty of stenotic blood vessels has proven to be an effective therapy in other body locations. Iliac artery angioplasty is well established in the treatment of lower extremity ischemia and multilevel occlusive disease. Successful treatment of intracranial arterial stenosis with PTA has been reported in a small number of patients by Higashida,7-9 McKenzie,7 Takis,8 Touho,9 and Clark10 and their coworkers.

Several factors contribute to the limited use of intracranial PTA. First, the reported series and patient encounters have not yielded consistent results, complication rates have varied, and clinical outcomes have been uncertain. Well-established indications for intracranial angioplasty are lacking. The risk of distal embolization has been reported to be at least 1% in patients undergoing iliac PTA; however, the true incidence is probably much higher. Distal embolization is of low clinical significance in the lower extremities; however, the tolerance of neuronal tissue to microembolization is significantly lower, and this factor may be a significant deterrent to the use of intracranial PTA.

Dissection and occlusion of the angioplastied segment of the intracranial artery, although rare, can be catastrophic, as is the lack of a method to treat dissection. Rupture of an intracranial artery during PTA is a devastating risk to the patient and will likely result in death.

The rate of stroke as a major complication in our series is 13% (2/15); one patient developed aphasia and right-sided hemiplegia and the second patient had deterioration of a preexisting left-sided weakness. Higashida et al4-6 found a 33% stroke rate in 18 patients,
Touho\textsuperscript{9} reported a 15\% rate in 13 patients, Clark et al\textsuperscript{10} had a 9\% rate in 22 patients, McKenzie et al\textsuperscript{7} reported an 8\% rate in 12 patients, and Takis et al\textsuperscript{8} reported a 50\% rate in 8 patients. Strokes associated with intracranial PTA can be caused by distal embolization, arterial dissection, occlusion, vasospasm, or rupture of the angioplastied artery. The effect of distal embolization can theoretically be reduced by aggressive anticoagulation with heparin during and for at least 48 hours after angioplasty, keeping the partial thromboplastin time at 60 to 80 seconds.

Coronary microstents are under investigation for the intracranial cerebral arteries, although recently Phatouros et al\textsuperscript{11} reported successful percutaneous endovascular use of a Gianturco-Roubin-2 coronary stent (Cordis) for the treatment of an acute atheroembolic occlusion of the basilar artery. We believe further investigative trials are warranted to more firmly establish the efficacy and safety of intracranial microstents, which offer promise to a group previously considered untreatable. Stents have been proven to reduce complications in coronary artery angioplasty\textsuperscript{12} and provide a means of treating dissection resulting from angioplasty.\textsuperscript{13}

Arterial vasospasm at the site of angioplasty can be treated aggressively with papaverine hydrochloride or nitroglycerin. Inflating the balloon to a diameter (slightly) less than that of the adjacent normal arterial luminal diameter lessens the risk of arterial rupture.

The complication rate in this series is in the lower range of the reported experiences in the literature. There are several possible reasons for this. First, we selected the discrete rather than the segmental stenotic lesions of the cerebral arteries to treat in our series. Second, the balloon diameter was always slightly smaller than the normal diameter of the artery to be angioplastied, reducing the risk of cerebral artery rupture. Third, the use of general anesthesia and simultaneous biplane roadmapping reduces procedural time and complications. In addition, the technology has become much safer and easier to use.

**CONCLUSIONS**

Intracranial PTA can now be achieved with less morbidity and mortality than in the past in selected patients. We think that our relatively low morbidity is essentially because we do not try to restore the stenotic artery to its normal diameter. We accept the criticism that our study is only retrospective. The only accepted scientific way to demonstrate that PTA with or without a stent is superior to medical therapy is a randomized trial. It seems that there is enough evidence that such a trial is worth undertaking.

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