Percutaneous transluminal angioplasty in the treatment of atherosclerotic disease of the anterior cerebral circulation and hemodynamic evaluation

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Since its introduction by Grüntzig, et al., for use in the treatment of coronary artery stenosis, the interventional radiological technique of percutaneous transluminal angioplasty (PTA) with a balloon catheter has come into clinical use for dilation of atherosclerotic cerebral arteries. Recently, Higashida, et al., reported the use of PTA for treatment of hemodynamically significant (> 70%) stenosis involving the posterior circulation in 41 patients with vertebrobasilar insufficiency. Many reports have also been made concerning the usefulness of angioplasty in the treatment of atherosclerotic stenosis of the cervical portion of the internal carotid artery (ICA) and the distal portion of the common carotid artery. However, no systematic findings have yet been reported of hemodynamic evaluation prior to and following PTA.

In this study, PTA was performed in 19 patients with clinically symptomatic atherosclerotic stenosis in the carotid arterial system. Cerebral perfusion was evaluated prior to and after PTA in the regions affected by stenosis.

Clinical Material and Methods

Patient Selection

Between December 1992 and June 1993, PTA was performed in 19 patients between 56 and 76 years of age (65.9 ± 6.2 years, mean ± standard deviation) for clinically symptomatic atherosclerotic stenotic lesions at or distal to the C-5 segment of the carotid arterial system. The degree of stenosis was at least 70% of luminal diameter in all 19 patients. Locations included two lesions in the C-5 segment, three in the C-4 segment, and three in the C-2 segment of the carotid artery, six in the M1 segment, and three in the M2 segment of the middle cerebral artery, and two in the A1 segment of the anterior cerebral artery.

Both prior to and more than 6 months after PTA, angiograms were performed and cerebral perfusion was measured using 99mTc-hexamethyl-propyleneamine-oxime single-photon emission computerized tomography, before and after the administration of 10 mg/kg acetazolamide. Percutaneous transluminal angioplasty could be performed in 13 (68.4%) of the 19 patients. The mean degree of stenosis (± standard deviation) was 83.1% ± 8.6% before PTA, but only 35.8% ± 17.3% on the follow-up angiograms. Restenosis was detected in follow-up angiograms in five (38.5%) of the 13 patients. Seven of the 13 patients exhibited improvement in their neurological condition after PTA and had shown subnormal cerebral perfusion and subnormal vasodilatory response to administration of acetazolamide prior to undergoing PTA. On the other hand, the remaining six patients exhibited no improvement in neurological condition after PTA, and four of these patients (66.7%) had shown normal perfusion and five (83.3%) had shown normal vasodilatory response to administration of acetazolamide prior to undergoing PTA. These findings suggest that PTA may be indicated for patients with atherosclerotic stenotic lesions in the anterior cerebral circulation who have subnormal cerebral perfusion and low vasodilatory response to administration of acetazolamide.

Key Words • anterior cerebral circulation • atherosclerosis • cerebral perfusion • vasodilatory response • acetazolamide • percutaneous transluminal angioplasty
complete cerebral angiography, and cerebral perfusion studies. The relationship between neurological improvement and hemodynamic changes after PTA was retrospectively studied.

Cerebral Perfusion Studies

Cerebral perfusion was measured with $^{99m}$Tc-hexamethyl-propyleneamine-oxide (HMPAO) single-photon emission CT (SPECT). The $^{99m}$Tc-HMPAO SPECT studies were performed using a triple-head system with low-energy parallel hole collimators. Patients were placed in the supine position with the head fixed in a hemicylindrical plastic head holder. The orbitomeatal line served as a reference for patient position. During examination, changes in the relative position of the patient’s head were carefully monitored.

The $^{99m}$Tc-HMPAO was prepared using a nonradioactive kit† according to the recommendations of the manufacturer. Following ligand preparation and after completion of quality control, a solution of 370 MBq was withdrawn from the vial and immediately injected into the patient. The injection was made with the patient’s eyes closed. Data acquisition was begun 5 minutes after the injection, using a three-head rotating gamma camera. After the rotation of each gamma camera with a 3° step angle, with one step each 18 seconds, and using a $128 \times 128$ acquisition matrix, projection data from 128 steps were acquired over a 15-minute period. Sorenson’s technique ($\mu = 0.11 \text{ cm}^{-1}$) was used for collection of attenuation data and a cut-off frequency of 0.55 cycle/pixel for the Butterworth filter was used to generate reconstructed images. The thickness of the transaxial slices was 10 mm. After completion of data acquisition during the baseline examination prior to the administration of acetazolamide, 10 mg/kg acetazolamide was injected intravenously. Fifteen minutes later, 555 MBq of $^{99m}$Tc-HMPAO was injected, and data acquisition was restarted 5 minutes after this second injection of the tracer. The acquired data yielded combined pre- and postacetazolamide acquisition values, and decay-corrected subtraction of the preacquisition values from the combined pre- and postacetazolamide acquisition values yielded postacetazolamide values. Regions of interest (ROIs) were set on the cerebral perfusion map obtained by $^{99m}$Tc-HMPAO SPECT prior to and after administration of acetazolamide, in the territory of the cerebellum (ROI-1) and bilaterally in the MCA territories (ROI-2 and ROI-3) (Fig. 1).

The value obtained by dividing radioisotope uptake per pixel in either ROI-2 or ROI-3 (the affected side) by the average radioisotope uptake per pixel in ROI-1 was defined as cerebral perfusion (CP) in the affected territory of the MCA. The change in cerebral perfusion following administration of acetazolamide was defined as vasodilatory response (VDR): % VDR = [(postacetazolamide CP) - (preacetazolamide CP)] / (preacetazolamide CP) × 100. Cerebral perfusion studies were conducted before, immediately after, and more than 6 months following PTA. Cerebral perfusion and vasodilatory response values obtained before PTA and more than 6 months after PTA were compared.

Age-matched control values of cerebral perfusion and vasodilatory response were obtained in 10 patients with

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† Ceretec nonradioactive HMPAO kit manufactured by Amersham International, Buckinghamshire, England.
the territory of the cerebellum (ROI-1; left) and bilaterally in the territory of the middle cerebral arteries (ROI-2 and ROI- 3; right).

Ménière’s disease who were between 42 and 78 years of age (mean 61.9 ± 9.2 years). Control values of cerebral perfusion and vasodilatory response were defined as follows:

control CP = (right hemisphere CP + left hemisphere CP)/2

control VDR = (right hemisphere VDR + left hemisphereVDR)/2.

Intravascular Technique

Stenosis of the ACA and MCA. A No. 7 French coaxial guiding catheter‡ was positioned in the cervical segment of the ICA via the transfemoral route. Then PTA was performed using a dilation balloon catheter, steerable guidewire, and valve-wire.§ The balloon diameter to be used for the PTA procedure was determined by measuring the normal caliber of the vessels both above and below the site of stenosis; the diameter of the balloon chosen approximated but did not exceed the normal luminal diameter, because overdistention may cause vascular dissection. The microballoon catheters used were 2.0 mm to 3.5 mm in diameter (in 0.5-mm increments). The dilation balloon catheter was introduced into the lesion through the guide catheter following intravenous administration of 3000 IU heparin. The guidewire was used first for selective catheterization at the site of stenosis, and was then exchanged for the valve-wire when the dilation catheter reached the site of stenosis. Following occlusion with the valve-wire, the balloon was inflated using a gauged inflator.§

The balloon was then inflated up to the maximum recommended pressure of 3 to 7 atm for less than 20 seconds. Fluoroscopy was performed to determine whether dilation of the vessel had clearly been induced; if dilation was not observed, a second or third attempt at dilation was made. It was thought best that the balloon be inflated for less than 20 seconds so as not to induce further ischemia in regions of the brain suffering vascular compromise.

Following PTA, selective angiography was performed to determine the degree of dilation obtained, whether dissection had occurred, and to evaluate the intracranial circulation for signs of distal embolization. The patient’s neurological state was carefully observed before, during, and after the PTA procedure. Doses of 500 ml/day of low-molecular-weight dextran containing 8000 IU heparin were administered intravenously for 1 or 2 days following PTA, after which patients received 200 mg/day of aspirin for 2 months.

Stenosis of the ICA. Given the possibility of dislodging an embolus from ulcerative plaque in the carotid artery, a No. 7 French coaxial balloon catheter system* was used for guiding and blocking and only one attempt was made at angioplasty of the ICA. The balloon catheter was positioned in the cervical segment of the ICA via the transfemoral route. The dilation balloon catheter was introduced into the lesions through the occlusion balloon catheter. The dilation balloon catheters used were between 2.5 and 3.5 mm in diameter; the balloon was inflated up to the maximum recommended balloon pressure of 3 to 7 atm for less than 20 seconds. Just before deflation, the balloon of the occlusion catheter was inflated to occlude the ICA. Potential atherosclerotic particles or clots that might have been dislodged during inflation of the dilation balloon catheter were aspirated forcefully with a 5-ml syringe through the occlusion balloon catheter, following deflation of the dilation catheter and after withdrawal of the dilation catheter (Fig. 2). Approximately 1.8 ml of blood could be aspirated in 10 seconds following deflation of the dilation catheter (Fig. 2B).

Patients were followed clinically after PTA, and follow-up angiography and cerebral perfusion studies were performed more than 6 months after the procedure to determine the results of therapy. Cerebral perfusion and vasodilatory response in the patients who underwent PTA were compared with those in the control group. The follow-up period ranged from 6 to 12 months (mean 8.7 ± 1.9 months).

Results

Percutaneous transluminal angioplasty was performed in 13 (68.4%) of the 19 patients. The other six patients had severe atherosclerotic change proximal to the stenotic lesions that precluded positioning of the dilation catheter within the lesions.

Clinical Outcome

Patients Treated by PTA. Seven of the 13 patients treated showed a marked improvement in their neurological state. In Cases 3, 8, and 15, repetitive TIAs that existed before PTA resolved completely. In Cases 9, 12, 13, and 14, persistent and fluctuating neurological deficits im-

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* Berenstein occlusion balloon catheter obtained from Medi-tech, Watertown, Massachusetts.

‡ Guide catheter, Model GCS 7/5, obtained from Nycomed Ingenor, Paris, France.

§ Stealth dilation balloon catheter, Seeker guidewire, and valve wire obtained from Target Therapeutics, Fremont, California.

|| Indeflator Plus 20 manufactured by Advanced Cardiovascular Systems, Inc., Temecula, California.
proved and became stable, although the deficits did not disappear entirely.

Six patients showed no improvement in their neurological state. In Cases 2, 5, 10, 11, 17, persistent neurological deficits continued, and in Case 1 repetitive TIAs occurred even after PTA.

Patients Not Treated by PTA. In Cases 4, 6, 7, 16, 18, and 19, PTA was attempted to dilate a stenotic segment of the ICA, MCA, or ACA, but the dilation catheter could not be introduced into the stenotic segment (Table 1). In these cases, persistent and unstable neurological deficits did not show any improvement. In two patients (Cases 7 and 18) neurological deterioration was found 2 months after admission, due to the appearance of further obstruction in the distal portion of the stenotic lesion.

Morphological Findings Before and After PTA

The mean degree of stenosis was 83.1% ± 8.6% before PTA, was significantly less, 25.0% ± 9.6%, immediately after PTA, and was slightly higher, at 35.8% ± 17.3%, on the follow-up angiograms. Restenosis was detected on the follow-up angiograms in five (38.5%) of the 13 lesions treated. Clinical improvement following PTA was noted in seven (53.8%) of the 13 treated patients. Three patients with 70% stenosis prior to PTA (Cases 2, 11, and 17) had angiographic evidence of decreased stenosis but exhibited no clinical improvement after PTA (Table 1).

Hemodynamic Changes Before and After PTA

Cerebral perfusion and vasodilatory response in the unaffected side, except in Case 3, were within control limits (± 2 standard deviations). In Case 3, these parameters were much less than the control values (> 2 standard deviations less than the mean) due to severe infarction in the left cerebral hemisphere.

Cerebral perfusion in the affected side was less than the control value (> 2 standard deviations less than the mean) in nine (69.2%) of the 13 treated patients (Fig. 3 left), and vasodilatory response in the affected side was less than the control value in eight (61.5%) of these patients (Fig. 3 right). All seven patients (100%) who exhibited clinical improvement after PTA had demonstrated subnormal cerebral perfusion prior to PTA, whereas only two (33.3%) of the six patients who did not exhibit clinical improvement after PTA had shown prior subnormal cerebral perfusion (Fig. 3 left). The former percentage was significantly higher than the latter (chi-square test, p < 0.01). All seven patients (100%) who exhibited clinical improvement after PTA had demonstrated subnormal vasodilatory response prior to the procedure, but only one (16.7%) of the six patients who did not exhibit clinical improvement after PTA had exhibited a prior subnormal vasodilatory response (Fig. 3 right). The former percent-
age was again significantly higher than the latter (chi-square test, \( p < 0.005 \)). Low cerebral perfusion (0.547) and vasodilatory response (−8.79) values were found in Case 10, in which clinical improvement was not obtained because, it is believed, the patient’s neurological deficits were serious and irreversible cerebral damage had already occurred before PTA. Thus, increases in cerebral perfusion (0.646) and vasodilatory response (−6.97) values occurring after PTA could not overcome previously existing deficits. Case 10 was excluded from the following statistical analysis for this reason.

Cerebral perfusion and vasodilatory response in the seven patients who showed clinical improvement after PTA, five patients who did not show clinical improvement after PTA, and the 10 control patients with Ménière’s disease were compared (Table 2). In the seven patients who improved, mean cerebral perfusion and vasodilatory response values prior to PTA were 0.695 ± 0.060 and −8.87 ± 0.52, respectively; both these values were significantly lower than control values (unpaired t-test, \( p < 0.001 \)). Cerebral perfusion and vasodilatory response values in these seven patients after PTA were 0.805 ± 0.045 and −5.87 ± 0.82, respectively; both these values were again significantly lower than control values (unpaired t-test, \( p < 0.05 \) and \( p < 0.02 \), respectively). Both cerebral perfusion and vasodilatory response values were significantly higher after PTA than before the procedure in the seven patients who showed clinical improvement (paired t-test, \( p < 0.002 \) and \( p < 0.001 \), respectively).

In the five patients who did not show clinical improvement, mean cerebral perfusion and vasodilatory response values prior to PTA were 0.815 ± 0.023 and −4.32 ± 1.03, respectively. The former value was slightly but significantly lower than the control value (unpaired t-test, \( p < 0.05 \)). In this group of patients there was no significant difference in cerebral perfusion values before and after PTA. In addition, there was no significant difference in vasodilatory response before and after PTA in these five patients (Table 2). Age-matched control values of cerebral perfusion and vasodilatory response were 0.850 ± 0.029 and −3.59 ± 2.10, respectively.

In the five patients with stenosis of the ICA, atherosclerotic debris and clots were aspirated through the occlusion balloon catheter following deflation of the dilation catheter or after withdrawal of the dilation catheter (Fig. 2B and C).

Complications

One patient (Case 2) suffered asymptomatic arterial dissection just after PTA, which was, however, transient and not demonstrated on the follow-up angiograms. Two patients (Cases 9 and 14) with stenosis in the M1 segment suffered transient aggravation of hemiparesis after PTA. However, by 1 month after PTA both patients had an improved neurological condition better than that prior to the procedure.

Illustrative Cases

Case 3

This 57-year-old man was admitted to our institution on April 3, 1993, with repetitive TIAs characterized by left-sided hemiparesis. He had suffered cerebral infarction in the territory of the left MCA due to thrombotic occlusion 1 year before admission. The TIAs began occurring in February 1993, and although he had been medicated daily with 200 mg of aspirin orally, the TIAs continued to occur frequently. A magnetic resonance (MR) imaging study obtained on admission disclosed a large infarction in the territory of the left MCA and a small subcortical infarction in the territory of the right MCA.

On April 6, 1993, cerebral angiography was performed and disclosed 90% stenosis of the C4 segment of the right ICA, with no significant collateral circulation via the anterior or posterior communicating arteries (Fig. 4 and Fig. 5A). A \(^{99m}\)Tc-HMPAO SPECT cerebral perfusion study disclosed a region of low perfusion in the territory of the right MCA (CP = 0.732), and demonstrated that vasodilatory response was reduced in the relevant region (VDR = −9.09).
On April 13, PTA was performed on the C-4 segment of the right ICA with a 3.5 × 10-mm dilation catheter (Fig. 5B), with dilation of the stenotic lesion to 30% stenosis (Fig. 5C). Postoperatively, complete remission of the TIAs was noted; the patient was treated with 22 mg of aspirin per day. Follow-up angiography performed on December 15, 1993, demonstrated mild restenosis of approximately 40% (Fig. 5D). A 99m Tc-HMPAO SPECT cerebral perfusion study performed on the same day disclosed a slight increase in cerebral perfusion in the territory of the right MCA (CP = 0.785) and a markedly increased vasodilatory response compared with that prior to PTA (VDR = −6.06).

Case 15

This 75-year-old woman was admitted to our institution on January 9, 1993, with repetitive TIAs characterized by right-sided hemiparesis and motor aphasia. The TIAs began in November 1992. An MR imaging study performed on admission disclosed no abnormalities. Cerebral angiography was performed on January 11, 1993 and demonstrated stenosis of the M2 segment of the left MCA (Fig. 6A). The 99m Tc-HMPAO SPECT images demonstrated subnormal cerebral perfusion and subnormal vasodilatory response in the territory of the left MCA (CP = 0.762, VDR = −8.59). The patient underwent PTA of the stenotic M2 segment (Fig. 6B) on January 16, 1993, with dilation of the stenotic lesion to 20% stenosis (Fig. 6C). Follow-up angiography performed on December 21, 1993, demonstrated no restenosis (Fig. 6D). Cerebral perfusion studies obtained after the follow-up angiography showed marked improvement in cerebral perfusion and vasodilatory response compared to before PTA (CP = 0.833, VDR = −5.35). The patient experienced no TIAs following the PTA procedure.

Discussion

Hemodynamic insufficiency and intraarterial embolization due to arterial stenotic-occlusive disease are possible causes of cerebral ischemia in the anterior circulation. The frequency of embolic phenomena in the anterior circulatory system is significantly higher than in the posterior circulation. However, the subgroup of patients with ischemic attacks in the anterior circulation, including complete stroke and/or repetitive TIAs as well as hemodynamic compromise, will benefit from bypass surgery or PTA. Several investigators have suggested that extracranial-intracranial bypass is effective in preventing cerebral ischemic attacks in patients with occlusive carotid artery disease only when patients display marked reduction of cerebral perfusion reserve due to inadequacy of collateral pathways. Use of the PTA technique would be much more reasonable in treating stenosis of the distal portion of the ICA, MCA, or ACA. In patients with such stenosis, ischemic attacks may continue to occur despite medication. Hemodynamic factors are considered to play a role in the pathogenesis of ischemic attacks.

Cerebral perfusion reserve has been evaluated principally in studies of brain metabolism involving determination of the regional cerebral metabolic rate of oxygen consumption and the regional oxygen extraction fraction, and in studies of cerebral vasodilatory response to carbon dioxide or acetazolamide. The acetazolamide test has frequently been used in assessments of cerebral perfusion reserve.

Percutaneous transluminal angioplasty has come to be widely used for the treatment of coronary artery disease and accepted as a therapeutic modality in that discipline. Although it has been used for the treatment of cerebrovascular disease, it is not yet widely accepted as a therapeutic modality in this area, because it has the potential to induce distal embolization of atherosclerotic debris and to cause inadvertent occlusion of perforating branches originating from the first segment of the MCA or basilar artery when plaque is compressed. As opposed to occlusion in coronary and systemic arteries, occlusion of small, single perforating vessels in the brain can prove devastating.

Théron, et al., introduced the use of a triple coaxial catheter system designed to protect the anterior cerebral circulation from distal embolization of thrombus and atherosclerotic debris during PTA. This device can be introduced in the cervical segment of the ICA. In the present study, we used a No. 7 French occlusion balloon catheter as a blocking balloon to safely perform PTA in the ICA.

Purdy, et al., reported the first case of dilation of an atherosclerotic MCA that resulted in improved cerebral
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perfusion. In their case cerebral perfusion in the left hemisphere, as measured by $^{99m}$Tc-HMPAO SPECT, was increased after the PTA procedure. Pistoia, et al.,28 reported a patient in whom stable xenon CT studies performed immediately before and after PTA for treatment of vasospasm documented the effect of the procedure. These authors used stable xenon CT to quantitatively measure perfusion changes before and after PTA. However, a systematic study of regional cerebral perfusion and perfusion reserve prior to and after PTA in the treatment of atherosclerotic stenotic cerebrovascular disease has not been reported. Knop, et al.,21 reported the use of $^{99m}$Tc-HMPAO SPECT with acetazolamide challenge to detect hemodynamic compromise in occlusive cerebrovascular disease. They concluded that the method is useful for assessment of the adequacy of hemispheric collateral pathways in patients with severe occlusive cerebrovascular disease.

In the present study, $^{99m}$Tc-HMPAO SPECT with acetazolamide challenge, a qualitative rather than quantitative test, was used to evaluate local cerebral perfusion and hemodynamic compromise. The author attempted to evaluate these parameters objectively by computing ratios of activity for cerebellar regions of interest. By definition, if the percentage of the acetazolamide-induced increase in perfusion in the cerebrum equals that in the cerebellum, the vasodilatory response is zero. The mean value of the vasodilatory response in the control group was slightly less than zero ($-3.59\%$) in this study. Thus, in the control group the percentage of an acetazolamide-induced increase in perfusion in the cerebellum was slightly higher than in the cerebrum when cerebral perfusion was measured by $^{99m}$Tc-HMPAO SPECT.

In this study, PTA could not be performed in six (31.6\%) of the 19 patients due to technical difficulties. Restenosis was detected on follow-up angiograms in five (38.5\%) of the 13 patients treated by PTA. However, restenosis was mild and its degree was less than 70\%, except in Case 9. Clinical improvement was noted following PTA in seven (53.8\%) of the 13 treated patients. Thus the overall clinical success rate was 36.8\% (seven of 19).

All seven patients who showed clinical improvement after PTA had subnormal cerebral perfusion and a subnormal vasodilatory response to acetazolamide. On the other hand, of the six patients who showed no neurological improvement after PTA, four (66.6\%) had normal cerebral perfusion and five (83.3\%) had normal vasodilatory response to acetazolamide. These findings suggested that PTA may be indicated for patients who have at least 80\% stenosis in the anterior circulation and both subnormal cerebral perfusion in the resting state and subnormal vasodilatory response to administration of acetazolamide, because PTA can be used to dilate an atherosclerotic stenotic lesion to increase perfusion pressure in the distal portion of the carotid arterial system.

In summary, our findings suggest that performance of $^{99m}$Tc-HMPAO SPECT cerebral perfusion studies before and after administration of acetazolamide is useful for the selection of candidates for PTA, although it is the author’s belief that a randomized study with a larger number of patients is needed to confirm the indications for PTA.

References

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<th>% Restenosis (Follow-Up, mo)</th>
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* Abbreviations: PTA = percutaneous transluminal angioplasty; TIAs = transient ischemia attacks; NA = not applicable.
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<td>-3.76 ± 1.29</td>
</tr>
<tr>
<td>no (five patients)</td>
<td>0.850 ± 0.029</td>
<td>NA</td>
<td>-3.59 ± 2.10</td>
<td>NA</td>
</tr>
<tr>
<td>control group (10 patients)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Data are expressed as the mean ± standard deviation (SD). PTA = percutaneous transluminal angioplasty; CP = cerebral perfusion; VDR = vasodilatory response to administration of acetazolamide; NA = not applicable.
† p < 0.001 compared with the control value.
‡ p < 0.001 compared with the mean for the five patients who did not show clinical improvement.
§ p < 0.05 compared with the control value.
∥ p < 0.002 compared with the value prior to PTA.
** p < 0.02 compared with the control value.
††p < 0.01 compared with the mean for the five patients who did not show clinical improvement.
‡‡p < 0.001 compared with the value prior to PTA.