Hemodynamic Evaluation of the Effect of Percutaneous Transluminal Angioplasty for Atherosclerotic Disease of the Vertebrobasilar Arterial System

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Abstract

The relationship between clinical improvement after percutaneous transluminal angioplasty (PTA) and hemodynamic condition in vertebrobasilar insufficiency was evaluated in 43 patients between 45 and 86 years of age with clinically symptomatic atherosclerotic stenotic lesions in the posterior circulation. The 43 patients had a total of 51 stenotic lesions, including 17 in the first segment of the vertebral artery, 32 in the fourth segment of the vertebral artery, and two in the basilar artery. Angiography was performed and cerebral perfusion was measured with technetium-99m-hexamethylpropyleneamine oxime single photon emission computed tomography before and after administration of 10 mg/kg acetazolamide prior to and more than 7 months after PTA. Mean stenosis was 81.3 ± 7.4% before PTA, but only 41.5 ± 17.4% at follow-up. Eighteen of the 24 patients with improved neurological condition after PTA had subnormal (<mean - 2 SDs) cerebral perfusion before PTA. Twenty of these 24 patients had subnormal vasodilatory response to administration of acetazolamide before PTA. Clinical improvement following PTA was noted in only one of the 12 patients with a single stenotic lesion of the first segment, but in 23 of the 31 patients with intracranial stenotic or multiple stenotic lesions. PTA in the posterior circulation is indicated for patients with atherosclerotic stenotic intracranial lesion or multiple stenotic lesions who have subnormal cerebral perfusion and low vasodilatory response to administration of acetazolamide.

Key words: vertebrobasilar insufficiency, cerebral perfusion, vasodilatory response, acetazolamide, percutaneous transluminal angioplasty

Introduction

Percutaneous transluminal angioplasty (PTA) is a widely used therapeutic modality for the treatment of coronary artery disease. PTA has been used for the treatment of cerebrovascular disease, but is not widely accepted because of the potential for distal embolization by atherosclerotic debris and inadvertent occlusion of perforating branches originating from the first segment of the middle cerebral artery or basilar artery when plaque is crushed. In contrast to the coronary and systemic arteries, occlusion of small and single perforators in the brain can prove devastating. Small perforating branches do not have a collateral blood supply, and infarction will result in serious neurological deficits.

PTA to dilate an atherosclerotic middle cerebral artery has achieved improved cerebral perfusion in the left cerebral hemisphere as measured by technetium-99m-hexamethyl-propyleneamine oxime (99mTc-HMPAO) single photon emission computed tomography (SPECT). Recently, PTA was used to treat atherosclerotic disease of the vertebral and basilar arteries in a total of 42 lesions. However, a systematic study of regional cerebral perfusion prior to and after PTA in the posterior circulation has not been reported.

This retrospective study evaluated cerebral perfusion in the affected regions prior to and after PTA in 43 patients with clinically symptomatic and atherosclerotic stenosis involving the posterior circulation to determine the relationship between clinical improvement after PTA and hemodynamic condition in the posterior circulation before PTA.
Clinical Materials and Methods

I. Patient selection

PTA was performed in 43 patients aged between 45 and 86 years (mean ± SD 65.8 ± 8.0 years) for clinically symptomatic atherosclerotic stenotic lesions involving the extra- and/or intracranial vertebrobasilar arterial system between December 1992 and October 1994. The degree of stenosis was more than 70% in all patients. Patients with vertebrobasilar insufficiency and less than 70% stenosis were excluded. The 43 patients had no other stenotic lesions in the cerebral arteries. A total of 51 stenotic lesions were treated, including 17 in the first segment of the vertebral artery, 32 in the fourth segment of the vertebral artery, and two in the basilar artery. All patients with ischemic symptoms in the posterior circulation had definitive ischemia according to the rigid criteria of Cartlidge et al. No patient had ischemic symptoms or stenosis/occlusion in the territories of the internal carotid arteries. The vertebrobasilar arterial systems were not completely isolated from the carotid territory circulation. Computed tomography (CT) and/or magnetic resonance (MR) imaging showed small brain stem infarction and cerebellar infarction in 36 and 17 of the 43 patients, respectively.

All 43 patients had completed stroke and/or transient ischemic attacks (TIAs) and their neurological symptoms transiently and intermittently fluctuated and worsened despite various treatments, including hypervolemia, anticoagulation, and/or antiplatelet aggregation therapy.

All patients underwent radiological diagnostic evaluation including CT and/or MR imaging, complete cerebral angiography, and cerebral perfusion study.

II. Cerebral perfusion study

Cerebral perfusion was measured with 99mTc-HMPAO SPECT, performed using a triple-head SPECT system (Neurocam; General Electric Co., Milwaukee, Wis., U.S.A.) with low-energy parallel hole collimators.

Data acquisition in the resting state was begun 5 minutes after injection of 370 MBq of 99mTc-HMPAO with a three-head rotating gamma camera. The thickness of the transaxial slices was 10 mm. After completion of data acquisition (128 × 128 acquisition matrix) in the baseline examination (prior to the administration of acetazolamide), 10 mg/kg of acetazolamide was injected intravenously.

Fifteen minutes later, 555 MBq of 99mTc-HMPAO was injected, and data acquisition was restarted 5 minutes after this second injection. The acquired data yielded combined pre-/post-acetazolamide acquisition values. Decay-corrected subtraction of the pre-acetazolamide acquisition value of radioisotope count from the combined pre-/post-acetazolamide acquisition value yielded post-acetazolamide value for each pixel. Pre- and post-acetazolamide tomographic images were obtained following reconstruction using the pre- and post-acetazolamide acquisition values. Regions of interest (ROIs) were selected on the cerebral perfusion map obtained by 99mTc-HMPAO SPECT prior to and after administration of acetazolamide in the territory of the cerebellum (ROI-1) and bilaterally in the territory of the middle cerebral arteries (ROIs-2 and 3) (Fig. 1). Selection of these ROIs was standardized from case to case.

Percentage regional perfusion in the posterior circulation was defined as mean uptake per pixel in ROI-1/(mean uptake per pixel in ROI-2 + ROI-3) × 100. The change in regional perfusion following administration of acetazolamide was defined as vasodilatory response calculated as (regional perfusion after acetazolamide - regional perfusion prior to acetazolamide)/regional perfusion prior to acetazolamide × 100.

Cerebral perfusion studies were conducted before and more than 7 months after PTA. The regional perfusion and vasodilatory response before PTA and more than 7 months after PTA were compared.

III. Intravascular technique

A 7-Fr coaxial guiding catheter (GCA 7/5; Nycomed Ingenor, Paris, France) was positioned in the vertebral artery or the subclavian artery via the transfemoral route. PTA was performed using a Stealth dilation balloon catheter (Target Therapeutics, Fremont, Calif., U.S.A.). The balloon diameter...
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, since over dilation sometimes induces vascular dissection. The microballoon catheters used were between 2 and 3.5 mm in diameter (in 0.5-mm increments). The Stealth dilation balloon catheter was introduced into the lesions through the guiding catheter following intravenous administration of 3000 IU of heparin. The balloon was inflated up to the maximum recommended balloon pressure of 3 to 7 atm. for less than 60 seconds using a gauged inflator (Indeflator Plus 20™, Advanced Cardiovascular Systems, Inc., Temecula, Calif., U.S.A.). If dilation was not observed, a second or third dilation was performed.

Following PTA, selective angiography was performed to determine the degree of dilation obtained, to determine whether dissection had occurred or not, and to evaluate the intracranial circulation for signs of distal embolization. The neurological state of the patient was carefully observed before, during, and after the PTA procedure. Doses of 500 ml/day of low-molecular-weight dextran containing 8000 IU of heparin were administered intravenously for 1 or 2 days following PTA, following which patients received 330 mg/day of aspirin.

Patients were followed up by clinical examination, and angiography and cerebral perfusion studies to assess the results of therapy more than 7 months after PTA, because restenosis usually occurs within 3 to 5 months after PTA and no further stenosis occurred on lesions free from 3- to 5-month restenosis. The relationship between clinical improvement after PTA and change in hemodynamic condition measured by 99mTc-HMPAO SPECT was evaluated. The follow-up period ranged from 7 to 19 months (mean 13.4 ± 3.0 months). Control values of regional perfusion and vasodilatory response were obtained in 14 patients with Meniere’s disease between 42 and 78 years of age (62.1 ± 9.1 years). Values of hemodynamic parameters less than the control value were defined as subnormal. Values of hemodynamic parameters less than the control value or less than -0.66%.

Regional perfusion before PTA in 22 of the 43 patients was abnormally lower than the control value (less than the age-matched mean ± 2 SDs or lower than 105.9%). Vasodilatory response before PTA in 23 of the 43 patients was abnormally less than the control value or less than -0.66%.

Twenty-four patients showed clinical improvement after PTA, of whom 18 had subnormal regional perfusion and 20 had subnormal vasodilatory response prior to PTA. In contrast, 19 patients did not show clinical improvement after PTA, of whom four had subnormal regional perfusion and three had subnormal vasodilatory response prior to PTA. The former two rates were both significantly larger than the latter two rates. Thirty-one patients had intracranial stenotic or multiple stenotic lesions, of whom 22 had subnormal regional perfusion and 23 had subnormal vasodilatory response. All 12 patients with a single stenosis of the first segment of the vertebral artery had normal regional perfusion. However, there was no significant difference (p = 0.069). Restenosis was found in one of two lesions of the basilar artery. The degree of the restenosis was mild except in Cases 9, 12, 27, and 43. These four patients had severe restenosis of 90%, 70%, 70%, and 90%, respectively. Cases 9 and 43 were retreated by PTA and the stenosis improved to 50% and 25%, respectively. These four patients were also medically treated by antiplatelet therapy, but showed no improvement.

II. Clinical improvement after PTA

Clinical improvement was noted following PTA in 24 of the 43 patients: Only one of the 12 patients with a single stenotic lesion of the first segment of the vertebral artery, and 23 of 31 patients with intracranial stenotic or multiple stenotic lesions. The rate of improvement was significantly different (chi-square test, p < 0.0001). The 18 patients with completed stroke and neurological symptoms transiently fluctuating and worsening prior to PTA showed no further fluctuation/worsening of the neurological deficits or infarction after PTA. Eight of the 43 patients had repetitive TIAs before PTA and five of these eight patients showed no further TIAs after PTA. One patient still had TIAs after PTA, but the frequency of the TIAs was markedly decreased. The other two patients still had TIAs after PTA at the same frequency.

III. Hemodynamic changes prior to and after PTA

Age-matched control values of regional perfusion and vasodilatory response were 117.3 ± 5.7% and 3.88 ± 2.27%, respectively.

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Results

I. Morphological findings before and after PTA

The degree of stenosis was 81.3 ± 7.4% before PTA, significantly less at 31.6 ± 12.1% just after PTA, and then slightly increased at 41.5 ± 17.4% at follow-up. Restenosis occurred in 24 of the 51 lesions at follow-up. Restenosis was detected in 11 of 17 lesions of the first segment of the vertebral artery, and 12 of 32 lesions of the fourth segment of the vertebral artery. However, there was no significant difference (p = 0.069). Restenosis was found in one of two lesions of the basilar artery. The degree of the restenosis was mild except in Cases 9, 12, 27, and 43. These four patients had severe restenosis of 90%, 70%, 70%, and 90%, respectively. Cases 9 and 43 were retreated by PTA and the stenosis improved to 50% and 25%, respectively. These four patients were also medically treated by antiplatelet therapy, but showed no improvement.

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and vasodilatory response prior to PTA.

Regional perfusion and vasodilatory response prior to PTA in the 24 patients who showed clinical improvement after PTA were 96.2 ± 11.7% and 3.93 ± 2.82%, respectively, both significantly lower than the control values (unpaired t-test, p < 0.0001). Regional perfusion and vasodilatory response in these 24 patients after PTA were 114.3 ± 7.7% and 0.94 ± 1.59%, respectively, with the latter significantly lower than the control value (unpaired t-test, p < 0.0001). Both regional perfusion and vasodilatory response were significantly improved after PTA (paired t-test, p < 0.01 and p < 0.01, respectively). Regional perfusion and vasodilatory response prior to PTA in the 19 patients who did not show clinical improvement were 114.2 ± 7.6% and 1.74 ± 2.77%, respectively, with no significant difference compared with the control values. There was no significant difference in regional perfusion or vasodilatory response before and after PTA (paired t-test). Regional perfusion and vasodilatory response before PTA in the 24 patients were both significantly lower than in the 19 patients (unpaired t-test, p < 0.0001 and p < 0.01, respectively).

IV. Complications

TIAs or strokes occurred as complications of the procedure in only Case 43. Three patients (Cases 13, 23, and 42) suffered transient asymptomatic arterial dissection just after PTA, which was not present at the follow-up. Case 43 with diffuse basilar artery stenosis suffered pontine infarction after PTA and subsequent neurological deterioration, but her neurological condition improved to that before PTA after 1 month.

Illustrative Case

A 58-year-old male (Case 25) was admitted on June 3, 1993 with repetitive TIAs manifesting as quadriparesis, dysarthria, and/or loss of consciousness, beginning in April 1993. He had received oral administration of 330 mg of aspirin per day, but the TIAs continued to occur frequently. MR imaging on admission disclosed cerebellar and pontine infarction (Fig. 2).

Cerebral angiography performed on June 4, 1993 disclosed 80% stenosis of the first segment of the right vertebral artery and 95% stenosis of the fourth segment of the left vertebral artery (Fig. 3 left). The

Fig. 2 Case 25. T₂-weighted magnetic resonance image showing cerebellar and pontine infarction (arrows).

Fig. 3 Case 25. Left vertebral angiograms, showing severe stenosis of the fourth segment of the left vertebral artery before percutaneous transluminal angioplasty (PTA) (left) and marked dilation just after PTA (center), with no restenosis at 15-month follow-up (right).
right vertebral artery was hypoplastic. $^{99m}$Tc-HMPAO SPECT cerebral perfusion study disclosed a region of low perfusion (80.3%) in the posterior circulation (Fig. 4 upper left). Vasodilatory response after administration of acetazolamide in the posterior circulation was markedly reduced (−7.19%) (Fig. 4 lower left). On June 6, PTA with a 2.5 x 10 mm Stealth catheter was performed on the first segment of the right vertebral artery, with dilation of the stenotic lesion to only 30% stenosis. Second PTA was performed on the stenotic lesion of the fourth segment of the left vertebral artery on June 13, 1993. The Stealth catheter used for the second PTA was 3.0 x 10 mm. Marked dilation of the stenotic lesion was achieved (Fig. 3 center). Postoperatively, complete remission of the TIAs was obtained with 330 mg of aspirin per day. The patient was discharged on June 20, 1993.

Follow-up angiography performed on September 12, 1994 demonstrated maintenance of dilation and no evidence of restenosis (Fig. 3 right). $^{99m}$Tc-HMPAO SPECT cerebral perfusion study performed on the same day disclosed an increase in cerebral perfusion (110.2%) in the posterior circulation (Fig. 4 upper right) and increased vasodilatory response (2.41%) compared with that prior to PTA (Fig. 4 lower right).

Discussion

Atherosclerosis, arteriolar sclerosis, and intraarterial embolization are all possible causes of vertebrobasilar ischemia in patients with intracranial vertebrobasilar arterial occlusive disease. The frequency of embolic stroke in the posterior circulation system is significantly lower than in the carotid arterial system in such patients.20,26,29) Thrombosis developing in a region with preexisting atherosclerotic stenosis was the cause of 94% of basilar artery occlusions and 68% of vertebral artery occlusions.5)

Extracranial vertebral artery disease is seldom associated with signs or symptoms of ischemia unless blood flow through both vertebral arteries has been compromised.20) Decreased regional cerebral blood flow in the posterior circulation system induced by postural hypotension is significantly greater in patients with vertebrobasilar insufficiency than in normal subjects, and dysautoregulation is present in the former. Hemodynamic factors and dysautoregulation appear to be involved in the pathogenesis of vertebrobasilar insufficiency.20) In our previous study, hemodynamic compromise of the posterior circulation was the primary cause of vertebrobasilar ischemia.36)

Extracranial-intracranial bypass may be effective in preventing cerebral ischemic attacks in patients with occlusive carotid artery disease only if marked reduction of cerebral perfusion reserve is present due to inadequacy of collateral pathways.2,8,9,28,39) Cerebral perfusion reserve has been evaluated principally by 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,2,6,11,15-17,21,22,24,25,27,37) The acetazolamide test is frequently used in assessments of cerebral perfusion reserve.16,17,27,37)

In our study, cerebral perfusion was measured by $^{99m}$Tc-HMPAO SPECT in 43 patients with atherosclerotic disease in the vertebrobasilar arterial sys-
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tem. Twelve patients with a unilateral stenotic lesion in the extracranial vertebral artery (the first segment of the vertebral artery) had neither normal perfusion nor subnormal vasodilatory response, and only one of these patients showed clinical improvement after PTA. This patient (Case 5) suffered from reversible ischemic neurological deficits and TIAs associated with left Wallenberg’s syndrome. Cerebral angiography showed that the left vertebral artery was severely stenotic in its first segment, ended in the posterior inferior cerebellar artery, and received no collaterals from the contralateral vertebral artery. Tc-HMPAO SPECT showed that cerebral perfusion and hemodynamic reserve in the left cerebellar hemisphere were below normal prior to PTA, but were significantly increased after PTA. These findings suggest that patients with a single stenosis of the first segment of the vertebral artery and normal regional perfusion and vasodilatory response have marked development of collaterals to a distal portion of the stenosis via the contralateral vertebral artery (and various extracranial vessels). Repetitive and/or unstable ischemia in those patients may be due to intraarterial embolization and/or small vessel disease. In contrast, many of the 31 patients with intracranial lesions or multiple stenotic lesions had subnormal perfusion (22 patients) and subnormal vasodilatory response (23 patients), respectively.

Twenty-four patients showed clinical improvement after PTA, of whom 18 had subnormal perfusion and 20 had subnormal vasodilatory response. In contrast, 19 patients showed no neurological improvement after PTA, of whom 15 had normal perfusion and 16 had normal vasodilatory response. Intraarterial embolization and/or small vessel disease is a possible cause of vertebrobasilar insufficiency in patients who showed no neurological improvement after PTA. Eighteen patients with completed stroke and transiently fluctuating and worsening neurological symptoms prior to PTA showed no further fluctuation/worsening of the symptoms after PTA. Improvement of hemodynamic reserve in the posterior circulation was thought to be the main cause of disappearance of fluctuation/worsening of the neurological symptoms after PTA, and blood flow could be maintained even in the hypotensive state. These findings suggest that PTA in the posterior cerebral circulation may be indicated for patients with intracranial stenotic and/or multiple lesions with subnormal cerebral perfusion in the resting state and subnormal vasodilatory response to administration of acetazolamide in the posterior cerebral circulation, since PTA of an atherosclerotic stenotic lesion in the posterior cerebral circulation can increase perfusion pressure in the distal portion of the vertebrobasilar arterial system. Tc-HMPAO SPECT perfusion studies prior to and after administration of acetazolamide are useful for selection of candidates for PTA. However, remodeling of plaque by PTA may also result in distal embolization and ischemic attacks in the posterior circulation in patients with vertebrobasilar insufficiency. In our study, Case 43 with diffuse stenosis in the upper third of the basilar artery suffered aggravation of neurological symptoms just after PTA in association with brain stem infarction. The infarction was probably due to dissection of fractured plaque through the wall of the parent vessel and occlusion of the ostium of perforating branches which supply the brain stem.

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Commentary

Much of the experience with percutaneous transluminal angioplasty (PTA) in treating atherosclerosis of the vertebrobasilar system is anecdotal. Despite some remarkable success stories, the procedure carries the risk of distal embolization, arterial dissection, occlusion of the internal carotid artery, demonstrating rare complications and mild restenosis.

In terms of cerebral perfusion using $^{99m}$Tc-HMPAO SPECT, perfusion and Diamox response were reduced in patients with clinical improvement. In contrast, both were normal in patients without clinical improvement. Patients with clinical improvement revealed normal perfusion and improved Diamox response after PTA, which indicates the improvement of cerebral perfusion pressure and the resolution of the autoregulatory vasodilatation. These findings are very compatible and support the hemodynamic significance in the treatment of vertebrobasilar insufficiency. In addition, patients with single stenotic lesion in the proximal vertebral artery (first segment) had normal perfusion and Diamox response, achieving no clinical improvement except one. This was explained as being caused by small vessel disease and/or intraarterial embolization. It seems that there is a hemodynamic difference according to the stenotic site of the vertebral artery. However, Higashida et al. (ref. 13 of this article) reported that all patients with proximal vertebral artery stenosis improved clinically following PTA, though cerebral perfusion study was not done.

This paper provides the indications of PTA for atherosclerotic disease of vertebrobasilar artery, based on cerebral perfusion study. Perhaps other institutions will be inspired to use PTA, and a lot of data
over a longer period may be collected about its usefulness.

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Differential diagnosis between hemodynamic stroke and thromboembolic stroke is often difficult from the clinical presentation. In such cases, CBF study is necessary for indicating vascular reconstruction treatment. SPECT study may provide essential information for deciding the indication. Although PTA is less invasive than conventional bypass surgery, it has its own complications, such as arterial dissection, acute arterial occlusion, distal embolism, etc. Endovascular surgeons are expected to disclose the possible complications and their morbidities, as well as long-term outcome.

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Drs. Touho and Karasawa have carried out an interesting study on the hemodynamic evaluation of the effect of percutaneous transluminal angioplasty (PTA) for atherosclerotic disease of vertebrobasilar arterial system. At first sight it seems that they have established the indication for PTA in atherosclerotic disease of the vertebrobasilar system based on the perfusion study and on the vasodilatory response. It would be interesting if the authors could provide the number of the patients in which the posterior cerebral artery is filled via the posterior communicating artery, and if there is any relationship between the presence of the fetal pattern posterior communicating artery and the results of the perfusion study and the vasodilatory response. The additional blood flow to the ischemic vertebrobasilar system provided by a "fetal" posterior communicating artery might lead to results close to the control values of the perfusion study and the vasodilatory response.

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