Acute Thrombolytic Therapy and Subsequent Angioplasty for Atherosclerotic Stenosis of the Basilar Artery

—Case Report—

Shinichi NAKANO, Kiyotaka YOKOGAMI, Ryuji YAMADA, Tomokazu GOYA*, and Shinichiro WAKISAKA*

Department of Neurosurgery, Junwakai Memorial Hospital, Miyazaki; *Department of Neurosurgery, Miyazaki Medical College, Miyazaki

Abstract

A 50-year-old male presented with severe atherosclerotic stenosis of the basilar artery at its origin with very poor blood flow distally, manifesting as sudden onset of deterioration of consciousness to semicomatose with decerebrate posture. He regained consciousness dramatically after acute thrombolysis, although right hemiparesis persisted due to left pontine infarction. Follow-up angiography after 3 months of antiplatelet and anticoagulation therapy demonstrated severe residual stenosis of the basilar artery. Percutaneous transluminal angioplasty (PTA) resulted in wide patency of the basilar artery stenosis with excellent blood flow distally. Combination of acute thrombolytic therapy and subsequent PTA is an effective treatment for severe basilar artery occlusive disease.

Key words: thrombolysis, angioplasty, atherosclerosis, basilar artery

Introduction

Acute severe brainstem stroke with angiographically proven basilar artery occlusion has a poor prognosis, with a mortality of at least 75%.

However, acute thrombolysis for vertebrobasilar artery occlusion by local intra-arterial infusion of thrombolytic agents has recently been demonstrated to achieve a better clinical outcome.

Improved balloon catheter systems have also allowed percutaneous transluminal angioplasty (PTA) for atherosclerotic basilar artery stenosis to be performed successfully and safely.

We describe a patient with symptomatic atherosclerotic stenosis of the basilar artery at its origin with very poor blood flow distally, who was admitted semicomatose with decerebrate posture, but was treated successfully by acute thrombolysis and subsequent PTA.

Case Report

A 50-year-old male had suffered from frequent transient ischemic attacks consisting of right-sided numbness and vertigo for 1 month. He suffered loss of consciousness and was admitted semicomatose with decerebrate posture. Computed tomography (CT) revealed no hemorrhagic lesion. Cerebral angiography disclosed severe stenosis of the basilar artery at its origin with very poor blood flow distally (Fig. 1).

A 5-French catheter was advanced to the terminal portion of the second segment of the left vertebral artery (C-2 level) and 960,000 units of urokinase was infused into the artery. After completion of thrombolytic therapy, about 6 hours after onset, angiography revealed restoration of the blood flow in the basilar artery (Fig. 2). His consciousness level improved dramatically, although right hemiparesis and angiographical evidence of severe stenosis of the basilar artery still remained. Magnetic resonance (MR) imaging and CT demonstrated left pontine infarction in the distribution of the perforating...
branches of the basilar artery.

A course of anticoagulation therapy with intravenous administration of heparin was begun, subsequently changed to combination therapy of antiplatelet and anticoagulation medication (ticlopidine 200 mg/day and warfarin 5 mg/day). Follow-up angiography after 3 months of combination therapy demonstrated that the severe stenosis of the basilar artery at its origin still persisted. After much discussion with the patient regarding alternatives for treatment, he elected to undergo PTA.

A 7.5-French coaxial guide catheter was inserted through the right femoral artery into the left vertebral artery and advanced to the terminal portion of the second segment. A Stealth angioplasty balloon catheter with a maximum diameter of 2.5 mm (Target Therapeutics, Fremont, Cal., U.S.A.) was selected. After intravenous administration of 5000 units of heparin, the balloon catheter was advanced into the stenosis and inflated to 1 atm initially and subsequently up to 3 atm. A total of six inflations for 30 seconds each were performed at intervals of 1 or 2 minutes. Low molecular weight dextran (500 ml/3 hrs) and an additional 1000 units of intravenous heparin at 1-hour intervals were given during the dilatation procedure. Angiography immediately after the angioplasty revealed reduction in the stenosis (Fig. 3). There was no change in his neurological status during the procedure.

Postoperatively, he continued to receive antiplatelet agent (ticlopidine 200 mg/day). Low molecular weight dextran (500 ml/day) and anticoagulation therapy with an antithrombin agent (argatroban 30 mg/day) were also given for 7 days after the angioplasty. For a few days after the
angioplasty, he complained of transient double vision due to mild left abducens nerve paresis. Follow-up MR imaging revealed no new infarction. Follow-up angiography 1, 3, and 6 months after the angioplasty revealed adequate, continuous patency of the basilar artery without restenosis (Fig. 4). After rehabilitation, he returned to his previous employment, although the right hemiparesis still remained.

Discussion

Occlusion of the basilar artery is generally accompanied by life-threatening brainstem syndromes incompatible with normal survival. Kubik and Adams described 18 patients with brainstem infarction due to basilar artery occlusion and emphasized the sudden onset and frequently fatal outcome. Patients with basilar artery stenosis often develop sudden serious brainstem syndromes when the occlusion becomes complete due to newly developed fresh thrombus. Therefore, local intra-arterial infusion of thrombolytic agents in patients with acute severe brainstem syndrome indicates that local intra-arterial infusion of thrombolytic agents should be tried as soon as possible, within 5 or 6 hours if possible.

Previously, patients with severe stenosis of the basilar artery were usually treated with antiplatelet or anticoagulation therapy (or both). The many small perforating branches arising directly from the basilar artery to the brainstem make angioplasty of the basilar artery very difficult to perform. Occlusion of these small perforating arteries during angioplasty may have serious consequences, such as major stroke or death. Therefore, only patients who have failed to improve with maximum medical therapy were considered for angioplasty of the basilar artery. However, improved microballoon and catheter technology now allows PTA of the basilar artery. Most angioplasty balloons have a maximum inflation diameter greater than the average inside diameter of the normal basilar artery, which result in excessive dilatation and intimal injury in the stenotic and normal portions. The Stealth microballoon catheter system provides graduated inflation diameters down to the average inside diameter of the normal basilar artery and uniform pressure-modulated inflation, which allows mild dilatation of only the stenotic portion without dilatation of the normal portion. Therefore, PTA of a quite limited stenosis may be performed safely without occlusion of the perforating arteries.

The Stealth microballoon catheter also has an open-ended system, which allows injection of thrombolytic agents if distal embolization occurs during the angioplasty procedure. This microballoon system has been proved to achieve PTA of the basilar artery safely and successfully. Therefore, patients with a history of life-threatening acute brainstem syndromes due to the severe stenosis of the basilar artery by local administration of intra-arterial streptokinase or urokinase within hours of the symptom onset. Significant improvements in survival and clinical outcome were apparent in patients with recanalization compared with those without recanalization.

Intracerebral hemorrhage may occur following thrombolytic therapy, but the rate of hemorrhagic transformation is not so high. In our patient, the duration of ischemia was relatively long (about 6 hrs) and right hemiparesis due to pontine infarction persisted. However, the thrombolytic therapy did not cause intracerebral hemorrhage, and his level of consciousness and survival quality improved dramatically. Therefore, angiographical confirmation of basilar artery occlusion in patients with acute severe brainstem syndrome indicates that local intra-arterial infusion of thrombolytic agents should be tried as soon as possible, within 5 or 6 hours if possible.

Fig. 4 Follow-up left vertebral angiogram 6 months after the angioplasty, showing further remodeling of the basilar artery stenosis which was less severe than in the early postangioplasty study (arrow).
can be treated by PTA of the basilar artery when the stenosis is quite limited.

The combination of acute thrombolytic therapy and subsequent PTA of the basilar artery can improve the prognosis for patients with acute severe brainstem stroke. Long-term follow-up is required to assess the efficacy of this combination therapy.

References


Address reprint requests to: S. Nakano, M.D., Department of Neurosurgery, Junwakai Memorial Hospital, 1119 Komatsu, Miyazaki 880-21, Japan.