Thrombolysis and angioplasty for acute occlusion of intracranial vertebrobasilar arteries

Report of three cases

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Three cases of intracranial vertebrobasilar occlusion were successfully treated in the acute stage by thrombolysis and angioplasty. All three patients were admitted to the hospital because of consciousness disturbance and other brain-stem signs. Initial angiography revealed intracranial vertebrobasilar occlusions. At first, a microcatheter was introduced into the distal site of the occlusion and thrombolysis was attempted by using urokinase. Recanalization was achieved in all cases but severe stenosis of the intracranial vertebral and basilar arteries was found. The recanalization was followed by transluminal balloon angioplasty and the stenosis was successfully resolved. Marked neurological improvement was achieved in each case. Follow-up cerebral angiography demonstrated sufficient patency at the angio-plasty site after 3 to 6 months.

Residual severe stenosis of vertebrobasilar arteries after thrombolytic therapy carries the possibility of reocclusion. Combining angioplasty with thrombolysis to avoid rethrombosis and obtain sufficient distal blood flow is of significant benefit in treating vertebrobasilar occlusion.

KEY WORDS • arterial occlusion • intracranial vertebrobasilar artery • thrombolysis • angioplasty

Treatment Techniques

A computerized tomography (CT) scan was obtained in patients at hospital admission and the possibilities of intracranial hemorrhage and fresh infarction were excluded. At first, 3000 U heparin was intravenously injected into the patient, after which 1000 U heparin was injected every hour. Thrombolysis was performed through a Tracker-18 catheter (Target Therapeutics, Fremont, CA) with the tip placed just distal to the occluded site. Urokinase (120,000 U in 10 ml saline) was infused for 10 minutes, after which angiography was repeated. The maximum dose of urokinase was 960,000 U. After thrombolysis, angioplasty was performed using a Stealth balloon dilation catheter (Target Therapeutics) with a balloon sized 2 or 2.5 mm × 1 cm. Initially, the inflation pressure was 3 atm and the duration of inflation was 20 seconds. Multiple inflations of low pressure with 3 to 5 atm were repeated until satisfactory dilation was obtained. Systemic heparinization was continued for 48 hours after the percutaneous transluminal angioplasty (PTA). Thereafter antiplatelet drugs were ad-
ministered. Postangioplasty angiography was performed at 2 weeks and again at 3 to 6 months to evaluate restenosis.

Case Reports

Case 1

This 64-year-old man presented with sudden onset of consciousness disturbance, deteriorating into coma. He was admitted to our hospital 5 hours after onset of the symptoms.

Examination. A CT scan revealed small bilateral cerebellar infarction, but showed no lesion in the brainstem. Emergency angiography showed occlusion of the left VA in the intracranial portion (Fig. 1). The patient’s right VA was hypoplastic and terminated in the posterior inferior cerebellar artery. Both posterior cerebral arteries were the so-called fetal type.

Treatment. A microcatheter was navigated through the occluded left VA. After 480,000 U urokinase was infused, recanalization of the artery was attained; however, severe stenosis of the VA (V4 portion) and the BA was found. A 2.5-mm PTA balloon catheter was introduced via a transfemoral approach. At first, the stenotic portion of the left VA was dilated by expanding the balloon four times for 20 seconds each inflation. The stenotic portion of the BA was then dilated in the same manner. Thereafter, excellent distal blood flow was obtained, although there was 70% residual stenosis.

Fig. 1. Case 1. A: Left VA angiogram demonstrating total occlusion of the left VA (V4 portion). B: After thrombolytic therapy, the VA is open but severe stenosis is found in the VA and BA. Both posterior cerebral arteries were the so-called fetal type. C: After angioplasty, excellent distal blood flow is obtained, although there is 70% residual stenosis. A small cut is found in the BA at the angioplasty site. D: Angiogram obtained 6 months later showing the same patency. In the anteroposterior view there appears to be a small aneurysmal dilation at the angioplasty site; however, the oblique view reveals no aneurysm.

Fig. 2. Case 2. A: Left VA angiogram showing total occlusion of the lower BA. B: After urokinase therapy, the BA is open, but there is severe stenosis in the lower BA. C: After angioplasty, full dilation is achieved. D: Angiogram obtained 3 months posttreatment showing full patency.
residual stenosis. A small tear was found in the BA at the site of angioplasty. However, no clinical deterioration was observed and the patient’s consciousness became clear after these procedures.

Posttreatment Course. Magnetic resonance (MR) images obtained 1 week after treatment showed a small cerebellar infarction. Despite this finding, the patient was discharged from our hospital with minimal cerebellar ataxia. Follow-up angiography performed 6 months posttreatment showed the same amount of residual stenosis. The distal blood flow was sufficient and the small tear had disappeared. In the anteroposterior view there seemed to be a small aneurysmal dilation at the site at which the balloon was expanded; however, in the oblique view no aneurysm could be found.

Case 2

This 57-year-old man presented with sudden onset of consciousness disturbance and left hemiplegia.

Examination. A series of CT scans revealed no lesion. Emergency angiography showed occlusion of the BA and poor collateral flow via the posterior communicating arteries (Fig. 2).

Treatment. A microcatheter was introduced through the occlusion via a transfemoral approach and 480,000 U urokinase was infused intraarterially. Recanalization was achieved; however, there was a remnant of severe stenosis. A 2.5-mm PTA balloon catheter was introduced through the stenotic area and was expanded 10 times at 3 to 5 atm for 20 seconds each time. There were no complications and almost full dilation of the BA was achieved.

Posttreatment Course. Follow-up MR imaging performed 1 week after the procedures demonstrated a small infarction of the cerebellum but no lesion in the brainstem. The patient returned to his previous career 2 weeks after onset of the symptoms. Follow-up angiography performed 3 months later showed excellent dilation of the BA.

Case 3

This 49-year-old man presented with acute hearing loss and vertigo.

Examination. Neurological examination demonstrated a mild consciousness disturbance, left facial paresis, jerky nystagmus, and deafness in the left ear. Emergency angiography revealed 80% stenosis of the right VA orifice, occlusion of the left subclavian artery, and occlusion of the lower BA trunk (Fig. 3).

Treatment. A microcatheter was introduced via a transfemoral approach and 960,000 U urokinase was infused. Recanalization of the BA was achieved; however, 90% residual stenosis was found. A 5-mm PTA balloon catheter was introduced and expanded two times at 6 atm at the right VA orifice. A 2.5-mm PTA balloon catheter was then navigated into the BA and was inflated 11 times at 5 atm for 20 seconds each time. Sufficient dilations of the VA and BA were obtained.

Posttreatment Course. Magnetic resonance imaging demonstrated infarcts in the left anterior cerebellar territory. The patient was discharged from our hospital 5 weeks after the procedures with minimum truncal ataxia.

Discussion

Acute vertebrobasilar occlusion usually causes death or severe neurological deficits. A direct surgical procedure provides little benefit for acute vertebrobasilar occlusion; however, thrombolytic therapy has shown a significant effect on recanalization and has provided the patient with a better clinical outcome than medical treatment. All of our patients demonstrated rapid improvement in their consciousness level after recanalization of vertebrobasilar occlusion by thrombolysis; however, severe stenotic lesions were left.

The most common vascular lesion is atherosclerosis found in posterior circulation ischemia.
Transluminal angioplasty of supraaortic extracranial cerebral arteries is widely performed.\(^5,6,11,12,14,21\) However, angioplasty of intracranial vertebrobasilar arteries is reported infrequently because of the potential life-threatening risk of vessel rupture, thromboembolic occlusion, and brainstem infarction due to damage to the small perforating arteries.\(^8,9,18\) Transluminal angioplasty for intracranial vertebrobasilar arteries was first reported by Sundt and associates in 1980.\(^17\) Later Sundt\(^16\) reported that four of six patients died as a result of distal embolization or overdilated vessel damage. Recent improvements in balloon instruments and techniques have made angioplasty of the intracranial stenotic lesion safer and more stable.\(^1,8,9,18\) However, Higashida, et al.,\(^9\) reported encountering technical difficulty when performing angioplasty in distal VAs and BAs and a 37.5% (three of eight patients) major complication rate.\(^9\) Terada and associates\(^18\) reported a 33.3% immediate complication rate. The major reason for the complications in both reports was damage to perforating arteries that arose from the distal VA and BA. We used a relatively low pressure of 3 to 5 atm and repetitive dilations to avoid overdilation and damage to the perforating arteries. In Case 1, a small cut in the BA occurred, but fortunately, no neurological deterioration was observed. As Terada and associates noted in their report, the natural course of intracranial vertebrobasilar stenosis is not yet completely known. We also think that vertebrobasilar stenosis carries a relatively high possibility of infarction, as several reports of atherosclerotic carotid stenosis had shown.\(^18\) Our good results show that thrombolysis was effective in combination with angioplasty in treating intracranial vertebrobasilar stenosis.

References


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