Case Report

The Stent Placement for Acute Basilar Artery Occlusion in Japan

Susumu Yamaguchi1*, Kentaro Hayashi1, Nobutaka Horie1, Yohei Tateishi1, Shuji Fukuda1, Tsuyoshi Izumo1, Akira Tsujino2 and Izumi Nagata1

1Department of Neurosurgery, Nagasaki University School of Medicine, Japan
2Stroke Center, Nagasaki University School of Medicine, Japan

Abstract

Acute basilar artery (BA) occlusion is associated with high morbidity and mortality rates. Progress has been made in endovascular treatments for acute ischemic stroke. Here, we present a case of acute basilar artery occlusion treated with stent placement. A 72-year-old man was referred to the emergency department of our hospital. Basilar occlusion was detected by magnetic resonance imaging. The patient was treated with intra-arterial urokinase; however, persistent severe BA stenosis was diagnosed. Percutaneous transluminal angioplasty was performed unsuccessfully because of vascular recoil or residual atherosclerosis. Because of the risk of reocclusion, a balloon expandable stent was placed at the BA stenosis. The postoperative course was good, and the patient was discharged 1 month later. In patients with acute BA occlusion, although intracranial stent placement for acute ischemic stroke has certain risks such as dissection, subacute thrombosis, and perforator infarction, it has great potential to recanalize the steno-occlusion associated with atherosclerosis.

ABBREVIATIONS

BA: Basilar artery; GCS: Glasgow Coma Scale; NIHSS: National Institute of Health Stroke Scale; MRI: Magnetic Resonance Imaging; VA: Vertebral Artery; UK: Urokinase; PTA: Percutaneous Transluminal Angioplasty; mRS: Modified Rankin Scale

INTRODUCTION

Basilar artery (BA) occlusion is associated with a high rate of unfavorable clinical outcomes, including death, locked-in state, and coma [1]. The factors associated with clinical outcomes are the clinical state at presentation, the location of the occlusion, the time to treatment, and the degree of recanalization [2-6]. We herein present a case of BA occlusion treated with endovascular stenting and discuss its clinical implications with a literature review.

CASE PRESENTATION

A 72-year-old man who had a history of hypertension and hyperuricemia suffered from dysarthria and left hemiparesis when he woke up. He was transported to our hospital by ambulance 2 hours later. Neurological examination showed dysarthria and left hemiparesis. The patient scored 13 on the Glasgow Coma Scale (GCS, E3V4M6) and 8 points in the National Institute of Health Stroke Scale (NIHSS). On the way to the magnetic resonance imaging (MRI) room, he became comatose (GCS4, E1V1M2) and tetraplegic. His respiration became ataxic, and he was intubated. Diffusion-weighted imaging showed scattered small hyperintensity lesions in the left cerebellum and left occipital lobe (Figure 1A). Fluid-attenuated inversion recovery imaging showed no apparent signal changes, and BA occlusion was identified by MR angiography (Figure 1B). Intravenously administered tissue plasminogen activator treatment was not indicated because the onset was not identified accurately. Therefore, endovascular treatment was selected. Digital subtraction angiography showed BA occlusion (Figure 2A). A 6-F guiding catheter was advanced into the left vertebral artery (VA). A 2.5-F microcatheter was navigated through the occluded BA. Injection of contrast medium at the guiding catheter and microcatheter allowed visualization of an atherothrombotic occlusion of the tip and lower portion of the BA (Figure 2B). After administration of a dose of 120000 units of urokinase (UK) from the microcatheter, the tip of the BA was recanalized (Figure 2C). A second dose of UK was administered, but the BA stenosis remained (Figure 2D). The physical examination showed the persistence of consciousness disturbance and bilateral pupillary dilatation. Percutaneous transluminal angioplasty (PTA) was performed with a Gateway 2.5 × 9 mm (Boston Scientific, San Leandro, CA) below the
nominal pressure. The improvement of the stenosis resulted in an improvement of the consciousness disturbance to E3VTM6, and the bilateral pupillary dilatation disappeared. However, a few minutes later, the blood flow to the BA was impaired because of vascular recoil, and the patient became comatose, with pupils dilated. Although PTA was performed several times, the effect was temporary. We decided to place a stent for the BA stenosis. After administration of aspirin 200 mg and dipyridamole 300 mg via nasogastric tube and intravenous infusion of argatroban, an Integrity 2.5 × 18 mm (Medtronic, Tokyo, Japan) was placed from the VA (V4 portion) to the BA successfully. After the treatment, the consciousness disturbance was improved to GCS E4VTM6, and the bilateral pupil dilatation disappeared. The next day, the results of MRI showed a small new infarction at the dorsal pons and cerebellum, and the patency of the BA was confirmed with MR angiography (Figure 3). The postoperative course was uneventful, and the patient was discharged after 32 days. Despite the presence of lightheadedness and left medial longitudinal fasciculus at discharge, his NIHSS score was 2 and his mRS score was 2.

**DISCUSSION**

There are two major causes of acute BA occlusion, namely atherothrombotic occlusion and embolism. Embolism tends to occur at the bifurcation of the BA and atherothrombotic occlusion tends to occur in the middle or lower portion of the BA. In the present patient, after the recanalization of the BA with thrombolysis, the stenosis of the middle portion of the BA remained. Therefore, we considered this as an atherothrombotic lesion and treated it with repeated PTA instead of thrombus retrieval; however, the effect was temporary because of vascular recoil. Interestingly, consciousness disturbance and dilation of pupils resolved after PTA, whereas the neurological deterioration was synchronized with the vascular recoil. These neurological
changes occurring during the interventional treatment were valuable for our decision to administer additional treatments.

There are two strategies for the treatment of residual stenosis after recanalization of the intracranial artery. One is direct stent placement, and the other one is stent placement following a failed PTA. A total of 37 patients with residual BA stenosis were treated with stent placement during the acute phase [7-10]. A good outcome (mRS score: 0 to 2) was obtained in 19 patients (51%), and a poor outcome (mRS score: 3 to 6) was obtained in 18 patients (49%). The mortality rate was 16% (6 patients). Stent placement in intracranial arteries is associated with several complications, including cerebral infarction caused by perforator occlusion [11]. The plaque is frequently fragile, and stent placement can cause plaque rupture leading to perforator occlusion. Perforator occlusion by this mechanism may have occurred in some of the 18 patients with poor outcome. To avoid this complication, the pressure used for PTA should be lower than the nominal pressure.

In Japan, stent placement for intracranial artery stenosis is not approved by health insurance plans. Therefore, stents designed for intracranial arteries are not available in Japan. In the present case, we used a coronary artery stent. The stent placement was effective, and the prognosis was excellent. Stent placement for the treatment of intracranial atherothrombotic occlusion has great potential as an alternative treatment.

CONCLUSION

Acute BA occlusion is frequently associated with poor outcomes. A favorable outcome can be achieved by early recanalization. In the present patient, the neurological changes occurring during the interventional treatment were valuable and stent placement finally led to a favorable outcome.

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REFERENCES