Severe stenosis and occlusion of the posterior circulation result in serious symptoms and a high risk of death. Thrombolysis and endovascular treatment within a therapeutic window can be used to treat an acute occlusion of the vertebrobasilar artery. Recent reports have described the successful recanalization of non-acute occluded coronary, subclavian, and internal carotid arteries, but there have been few reports of intracranial endovascular recanalization of symptomatic intracranial occlusions during the subacute and chronic periods. Without recanalization, patients are at high risk of potentially fatal sequelae, including arterial dissection, thromboembolism, cerebral hemorrhage, deep perforating artery occlusion, and vascular rupture. We describe here a patient with a vertebrobasilar artery occlusion who was successfully treated with a Solitaire stent 2 months after symptom onset. Endovascular revascularization may be a promising treatment for patients with non-acute intracranial artery occlusion, poor collateral circulation, and deterioration after medical treatment.

Case Report. A 74-year old man was admitted to the Neurology Department of the First Hospital of Jilin University, Changchun, China with vertigo, double vision, and walking instability for 2 months. His condition had deteriorated during the previous 2 weeks, with weakness of his left limbs and choking while drinking and eating. Two months earlier, he had reported transient vertigo and double vision, occurring twice per day for 10 minutes each. By the third day, these symptoms became continuous; he also reported nausea, vomiting, and walking instability. A CT and computed tomography angiography (CTA) scanned at a local hospital resulted in a diagnosis of ischemic stroke of the posterior circulation. Both vertebral arteries and
the proximal basilar artery were occluded on CTA (Figure 1A). He recovered completely after 6 weeks of antiplatelet and fluid expansion therapy. Two weeks prior to presentation at our hospital, he suffered from diarrhea, followed by vertigo, double vision, and walking instability, as well as several new symptoms, including left hemiplegia, dysarthria, choking while drinking and eating, bilateral deafness, and sialorrhea from the right angle of the mouth. Since conservative treatment at a local hospital did not result in significant symptom improvement, he was transferred to our hospital for endovascular surgery. Physical and neurological examinations at admission showed that his blood pressure was 170/90 mm Hg and that he had dysarthria, right peripheral facial palsy, grade 4 muscle strength in his left limbs, dysmetria on finger-nose and heel-kneel-shin tests, and positive bilateral Babinski signs. A brain MRI showed bilateral, multiply infarcted lesions in the cerebellum and pons (Figures 1B & 1C). Transcranial Doppler sonography (TCD) showed that the blood in his left posterior cerebral artery was supported by his homolateral internal carotid artery, with the blood signal showing low velocity and high resistance. The TCD; however, did not explore the blood flow signal from his right vertebral artery. Vascular ultrasound of the neck showed that the blood signals of both vertebral arteries had low velocity and high resistance.

On the second day after admission, angiography under local anesthesia showed that both distal vertebral arteries and the proximal basilar artery were occluded (Figures 1D & 1E). We observed retrograde filling of the basilar tip and bilateral posterior cerebral arteries via the stenotic left posterior communicating artery. Angioplasty and stent implantation were performed to improve the posterior cerebral circulation on the third day under general anesthesia. A microcatheter was advanced into the distal left vertebral artery over a PT2 guidewire (Boston Scientific, Miami, FL, USA) and passed through the occluded segment. Angiography showed that the distal basilar artery and

![Patient imaging before and after revascularization of vertebrobasilar artery occlusion 2 months after symptom onset showing: A) Computed tomography angiography 2 days after symptom onset, showing occlusion of both vertebral arteries and the proximal 2/3 segment of the basilar artery (arrowhead), whereas the left posterior communicating artery was open (arrow). B) and C) MRI 2 months after symptom onset. B) T2-weighted MRI showing bilateral, multiple infarcted lesions in the cerebellum (arrows). C) Apparent Diffusion Coefficient MRI, showing multi-hypointense lesions, suggestive of an acute infarction (arrows). D) Angiography showing occlusion of both vertebral arteries (arrows). E) Left common carotid angiogram showing that the left posterior communicating artery was open, but there was local severe stenosis (arrowhead). F) Angiograph showing near normal patency of the basilar artery after stenting.](image-url)
both posterior cerebral arteries were patent, with the microcatheter located in the true lumen of the basilar artery. With the help of the microcatheter, an exchange micro guidewire was navigated past the basilar artery into the right posterior cerebral artery. A Submarine balloon 2.5×20 mm (Submarine Rapido, Invatec, Italy) was delivered to the point of occlusion and angioplasty was gently performed under a pressure of 6 atm along the occluded segment from the left vertebral to the basilar artery. Subsequent angiography of the left vertebral artery demonstrated restored antegrade flow to the basilar artery territory via the occluded segment, although the basilar artery had slight residual stenosis. The stenotic segment was 24 mm in length and 2.8 mm in diameter. The balloon was withdrawn, and a Solitaire stent measuring 4×20 mm (ev3, Inc., Irvine, California) was advanced to the point of the stenotic segment and deployed. Repeat angiography showed near normal patency of the basilar artery (Figure 1F), and the stent was then detached electrolytically. Five days after the operation, his symptoms had improved significantly; he could ambulate with the support of a family member, and required moderate assistance for activities. He was started on 300 mg/day aspirin (Bayer HealthCareAG, Beijing, China) and 75 mg/day clopidogrel (Plavix, Sanofi-Aventis, HangZhou, China) upon admission to our hospital. At the time of 3-month clinical follow-up evaluation, he could ambulate with a walker and needed minimal assistance for activities. The TCD revealed no restenosis of the stent.

**Discussion.** The ability of our patient to survive for 2 months with occlusion of both vertebral arteries and the basilar artery, and with non-severe symptoms of plegia, was likely due to the left posterior communicating cerebral artery, which supported blood flow to the posterior circulation. This compensation by the collateral circulation, however, was limited, leading to an ischemic stroke. His diarrhea induced more serious hypoperfusion of the posterior circulation. Without revascularization of the vertebrobasilar artery, his prognosis would have been worse and he could have died at any time.

Acute occlusion of the basilar or vertebral artery has been associated with high risks of stroke, death, and poor outcome in survivors. Timely recanulation, by thrombolysis, angioplasty, stenting or mechanical thrombectomy, is critical to improve clinical outcomes. Although intracranial endovascular recanalization of acute intracranial occlusions has been described, there have been few reports regarding the subacute and chronic periods. On literature review using PubMed, we could not find any previous reports describing the combination of angioplasty and Solitaire stent placement to treat a chronic intracranial artery. Some patients may survive occlusion of the intracranial vertebrobasilar artery with mild or no disability, whereas others may develop recurrent ischemic events despite optimal medical treatment. The strategy for managing these patients is still unclear.

Our patient underwent successful intracranial revascularization more than 2 months after symptom onset. Since artery dissection is a frequent complication of endovascular revascularization of the intracranial artery, a successful operation depends on ensuring stent placement in the true lumen of the basilar artery. Rupture of the intima flap may occlude the origin of the perforating branches of the basilar artery, which may be fatal even if recanalization is accomplished by stenting in a false lumen.

We chose to use a Solitaire self-dilating stent, not a Wingspan stent because the occluded segment of the basilar artery was 24 mm in length, longer than the longest Wingspan stent (20 mm). Overlap of the 2 stents in the occluded segment can make the overlapped region vulnerable to in-stent restenosis, as well as being more costly. We therefore utilized a Solitaire device, which is usually used to treat a wide-necked aneurysm or thrombectomy in a patient with an acute ischemic stroke. Due to its low radial strength, use of a Solitaire stent in angioplasty is relatively rare. We utilized a Solitaire stent because the balloon was completely dilated at a pressure of 4 atm, suggesting that the thrombus in the vessel was fresh, vascular calcification was not serious, and low radial force could prevent elastic recoil. Additionally, implantation of a Solitaire stent 4 mm in diameter into a small vessel of diameter 2.8 mm can result in a large degree of overlap with the walls of the stent, increasing its radial force.

Furthermore, the effective length of a Solitaire stent is 20 mm and its actual length is 31 mm, allowing it to cover a lesion 24 mm in length. Finally, a Solitaire stent can be retrieved by the delivery catheter unless more than two-thirds of the stent length has been deployed.

At the time of reporting, our patient’s symptoms have improved and his basilar artery is patent, with no restenosis, indicating that the operation was successful.

In conclusion, although endovascular revascularization was successful in our patient, there was a potential risk of death. Angioplasty and stenting may be an alternative treatment for patients with non-acute intracranial artery occlusions, whose collateral circulation is poor and whose symptoms have deteriorated despite medical treatment.
References


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