Recanalization of Subacute Intracranial Vertebral Artery Occlusion with Stent Implantation: A Case Report

JingFu, Luchao Jing, Ruifang Chen, Shize Li

Department of Neurology, Zhengzhou Central Hospital, Zhengzhou University
Department of Neurology, Zhengzhou Central Hospital, Zhengzhou University, No.195, Tongbai Road, Zhongyuan District, Zhengzhou, Henan Province, 45003, China

Abstract

Background: Reperfusion therapy is the most effective treatment for acute ischemic stroke. At present, multiple clinical studies have shown that endovascular treatment is efficacy and safety for acute ischemic stroke of anterior circulation artery occlusion disease in 24 hours. However, indications, time windows and treatment methods of reperfusion therapy for posterior circulation ischemic stroke are still highly controversial.

Methods and Results: We report a case of ischemic stroke of posterior circulation after 61 hours of onset. Imaging examinations show that there existed occlusion of corresponding vessels and obvious ischemic penumbra. Symptoms of the patient were progressively worsening and medical treatment was poor, therefore, corresponding vessel was opened by stenting. The post-circulatory brain tissue low perfusion status and clinical defect symptoms of the patients have been improved a lot.

Conclusions: Reperfusion therapy for posterior circulation ischemic stroke may be safe and effective. Moreover, there may be a wider reperfusion time window for patients with posterior circulation ischemic stroke.

Keywords: Ischemic Stroke; Stent Implantation; Posterior Circulation; Reperfusion Therapy; Time Window

Case report

A 65-year right-handed woman was referred to the neurologic clinic of our hospital with complaints of 58-hour persistent dizziness and slurred speech. When she got up in the morning 58 hours before admission, she felt dizzy and walked unsteadily, then her speech became slurred few minutes later. CT scans of the skull revealed no abnormalities in the local hospital. She received antiplatelet drugs, but the dizziness and slurred speech continued. Left limb weakness, weak holding capacity and walking dragging appeared 24 hours ago. She came to our hospital for further evaluation. She had a two-year history of uncontrolled hypertension. She denied the history of diabetes mellitus and coronary heart disease. On examination, the blood pressure of her left arm was as high as 170/90mmHg. She was conscious and had good mental status. Extraocular movements were intact. Bilateral pupil diameter and light reflex was normal. She was dysarthric severely and has difficulty to extend the tongue. Bilateral pharyngeal reflex was weakened. Her strength in the proximal portion of left arm was IV, distal portion was III, the strength in left leg was III, proximal portion of right arm was normal, distal portion was IV, and the strength of right lower extremity was V-. Her left Babinski’s sign was positive. In addition, the National Institutes of Health Stroke Scale score NIHSS was 11. The Modified Rankin Scale (MRS) was 4. Water Swallow test was grade III.

Brain computed tomography (CT) revealed no cerebral hemorrhage except some lacunar infarctions and encephalomalacia focus 8 hours after onset. Magnetic resonance imaging (MRI) showed that right thalamus, pons, right cerebellar hemisphere existed acute or subacute cerebral infarction 58 hours after onset (Figure 1). And magnetic resonance angiography (MRA) showed that the bilateral intracranial vertebral artery, the basilar artery, the bilateral posterior cerebral artery and its branch lumen are severe stenosis (Figure 2). 58 hours after onset, magnetic resonance perfusion imaging (PWI) revealed that cerebral blood flow perfusion were decreased in the pons and bilateral cerebellar hemisphere lesions, time to peak (TTP) and mean
transit time (MTT) were prolonged, and the ischemic penumbra was larger than the core infarct area obviously (Figure 3). Color Doppler ultrasonography of the cervical vessels showed thickening of intima-media of bilateral carotid arteries with plaque formation and hypoechoic wall of right vertebral artery. The right vertebral artery showed a high impedance pattern.

Figure 1: Brain MRI showed that right thalamus, pons, and right cerebella hemisphere exist acute or subacute cerebral infarction. (black arrow indicates position of cerebra linfarction)

Figure 2: MRA showed that the bilateral intracranial vertebral artery, the basilar artery, the bilateral posterior cerebral artery and its branch lumen are severe stenosis
Figure 3: PWI showed cerebral blood flow perfusion were decreased in the pons and bilateral cerebellar hemisphere lesions, peak time and mean transit time were prolonged, and the ischemic penumbra was larger than the core infarct area.

Treatment process

61 hours after onset, cerebral angiography and intracranial vertebral artery mechanical thrombectomy and stent implantation were performed (Figure 4). Postoperative PWI showed that the range of left cerebellar hemisphere and pons lesions were significantly reduced compared with the previous one. Aspirin, clopidogrel and atorvastatin calcium tablets antithrombotic treatment were given for 14 days after operation. At discharge, the NIHSS was 5, the Water Swallow test was grade II, and MRS was 1. After discharge for one month, the NIHSS was 0, the Water Swallow test was grade 0, and MRS was 0.

Figure 4: Cerebral angiography showed the left vertebral artery with obvious stenosis before vascular opening (A and B). Cerebral angiography showed the left vertebral artery was opened after intracranial vertebral artery mechanical thrombectomy and stent implantation, and the basilar artery, the bilateral middle cerebral artery, the superior cerebellar artery and the distal small artery are developed (C and D).

Discussion

Reperfusion therapy is the most effective treatment for acute ischemic stroke. At present, it mainly includes thrombolytic drugs and endovascular treatment. Most of the evidence for these treatments come from clinical studies of anterior circulation cerebral ischemic stroke\(^1\). Indications, time windows, and treatment methods of reperfusion therapy for posterior circulation ischemic stroke are still highly controversial. In this case, the responsible vessel was opened by stenting after 61 hours of onset. The post-circulatory brain tissue low perfusion status and clinical defect symptoms of the patients had been improved a lot. This suggests that there may be a wider reperfusion window for patients with posterior circulation ischemic stroke. The posterior circulation is more resistant to ischemia than the anterior circulation, and the possible reasons are as follows. Firstly, the posterior circulation has a rich collateral branch, which can be quickly compensated by the posterior circulation artery stenosis or occlusion, and relieve the ischemic symptoms. In addition, the current imaging studies show that there is a certain similarity between the blood supply pattern of the brainstem and the spinal blood supply model\(^2\). There is a blood supply pattern similar to the spinal artery coronary artery between the long and short circumflex branches of the basilar artery. They have abundant anastomotic branches, which are more

Citation: Shize Li et al. (2019), Recanalization of Subacute Intracranial Vertebral Artery Occlusion with stent Implantation: A Case Report. Int J clinical & case, 3:3, 49-52.
tolerant to ischemia. However, the paramedian midbrain of the basilar artery is the terminal artery without collateral compensation. If the paramedian midbrain is involved, ischemia tissue is irreversible with poor prognosis.

Secondly, when there is a thrombus in the basilar artery or vertebral artery, the posterior circulation is compensated by the posterior communicating artery. The peak pressure of the anterior circulation is several milliseconds earlier than the posterior circulation in the cardiac cycle. Therefore, the pressure gradient due to the peak difference will cause the thrombus to move within the posterior vertebral artery of the basilar artery. In this case, there is a certain gap and blood flow between the thrombus and the arterial wall. The blood supply from the brain stem will not disappear completely. This phenomenon has been confirmed in angiography of patients with basilar artery disease. This can explain the phenomenon that some patients with severe basilar artery lesions without obvious ischemic lesions.

There is a lot of controversies about the lack of large-scale clinical studies of endovascular treatment of posterior circulation macrovascular disease. In the published small sample of literature, screening patients with potential benefit is the primary issue. There are three ways to screen suitable patients at present. Firstly, cerebral perfusion imaging is used to assess whether there is an ischemic penumbra. The effectiveness of this approach has been confirmed in small sample studies. This method is also confirmed in this case. Secondly, specific imaging scores such as posterior circulation CT Alberta score and pons-midbrain index can be used to predict the prognosis of patients and determine whether endovascular treatment is feasible. Finally, acute cerebral infarction with low infarction rate or chronic progression may left longer reperfusion tissue window, therefore, a longer reperfusion time window may still exists for ischemic stroke patients with recurrent and progressive exacerbations of symptoms, and active endovascular treatment is the only option for patients with progressively worsening symptoms and poor medical treatment.

We reported that this patient was far beyond the conventional time window of reperfusion therapy. However, the patient’s symptoms gradually progressed and the lesions caused by brain stem perforating lesions were more limited and imaging showed the possibility of ischemic penumbra. Those suggest that the patient has indications for intravascular reperfusion therapy. In conclusion, the indications and treatment methods of endovascular treatment for cerebral infarction caused by posterior circulation macroangiopathy need a large sample of multi-center clinical research to further clarify.

References