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Case Report

Ipsilateral limb hemiplegia after left carotid artery stent implantation: one case report

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Abstract: Purpose: This research reported a case of initiation part of bilateral carotid arteries and left vertebral artery stenosis, to investigate the treatment of hypoperfusion cerebral infarction after stent implantation. Methods: A 65-year-old male patient received cerebral angiography and stent implantation in left vertical artery and left carotid artery. Hypotension and left limb hemiplegia occurred during the operation. The patient was treated by Voluven and dopamine to control the blood pressure, and clopidogrel and aspirin for antiplatelet. Results: The myodynamia of left upper and lower limbs recovered to degree five and three, respectively. Reexamination of brain MR+DWI showed new infarction in right frontal and parietal lobe, and cingulate gyrus. The patient discharged on the fourth day after operation. Conclusion: Hypoperfusion cerebral infarction may appear during the stent implantation. It mostly involves the blood vessel with worst hemodynamics. Immediate intervention can improve the prognosis.

Keywords: Carotid artery, stenosis, stent, hypoperfusion

Introduction

Stent implantation for carotid artery stenosis is an alternative to carotid endarterectomy [1]. Carotid artery lesions mainly occur in the starting department, which is the important factor of intracranial circulation ischemia and infarction. In addition to the drug treatment, endovascular treatment has become one of the most important therapeutic approaches for artery stenosis. At present, carotid artery stenting has attracted more and more attention in the treatment of carotid artery stenosis. Carotid artery stent has exhibited good effectiveness in primary and secondary prevention of ischemic stroke [2]. At the same time, some of the potential complications also cannot be ignored. Here we present a case of watershed cerebral infarction after left carotid artery stent implantation while the patient was treated symptomatically in time.

Case report

A 65-year-old man was admitted to our hospital with a complaint of blurred vision and dizzy. The symptom relieved after a few minutes. Head and neck CTA examination revealed suspicious severe stenosis in bilateral internal carotid and the opening of left vertebral artery, together with right vertebral artery occlusion. Brain CT demonstrated right occipital lobe infarction. The patient suffered from hypertension for five months with the highest systolic pressure at 180 mmHg and smoking history at 900/year. Carotid artery ultrasound showed bilateral carotid artery intima thickening combined with multiple plaques, including the right ulceration plaque. Left common carotid artery stenosis (middle branch <50%), bilateral bulbus caroticus stenosis (70-99%), right vertebral artery occlusion, and left vertebral artery stenosis (70-99%). TCCD exhibited lesion in the bilateral extracranial internal carotid artery and left extracranial vertebra artery, right vertebral artery occlusion, anterior communicating artery opening (left to right), and right collateral of extracranial internal carotid artery opening (**Figures 1**, **2**).

We initiated therapy with clopidogrel 75 mg, aspirin 0.1 g, and Lipitor 20 mg once a day

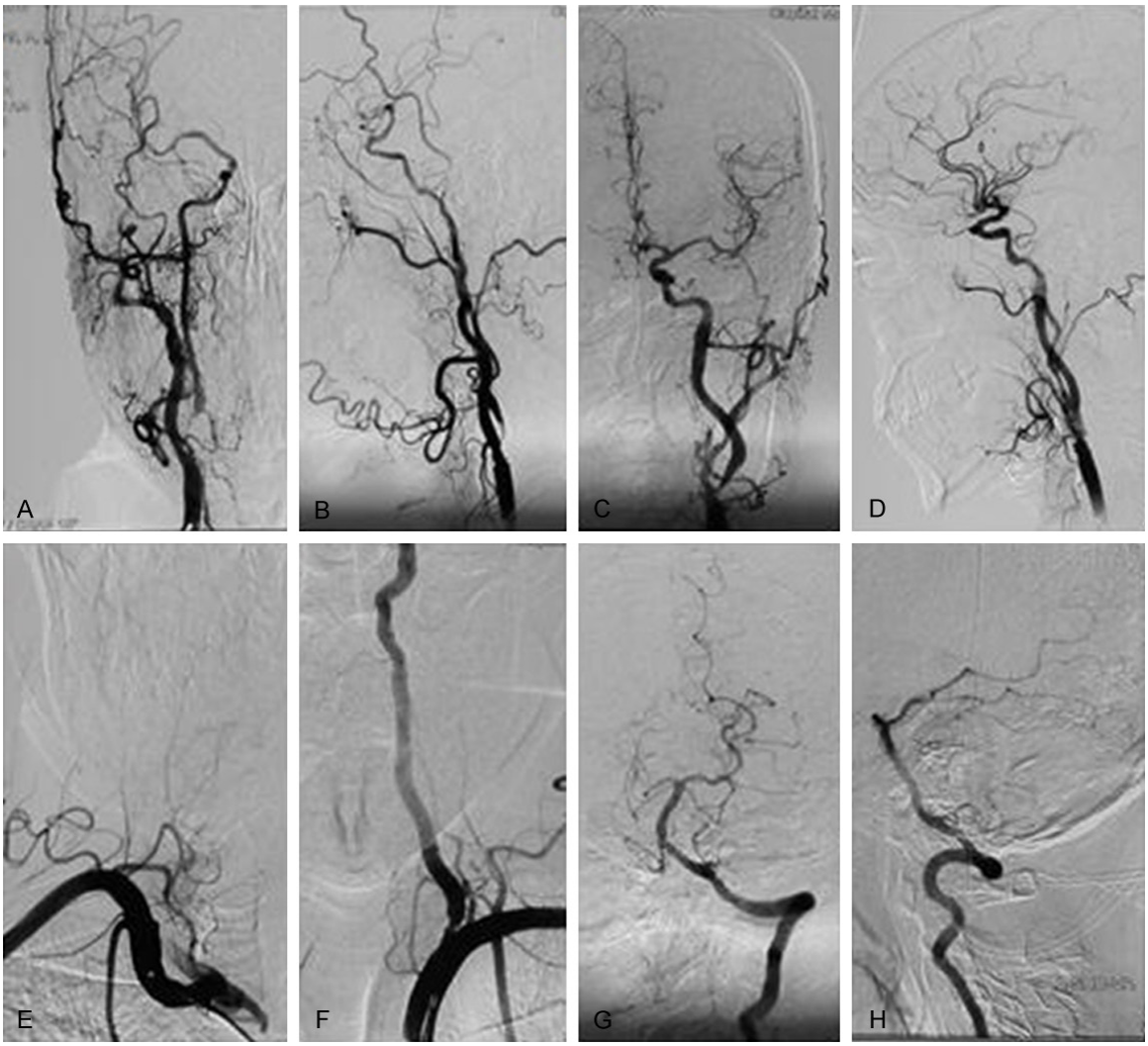

Figure 1. Cerebrovascular DSA. A. Right carotid artery normotopia. B. Right carotid artery side position. C. Left carotid artery normotopia. D. Left carotid artery side position. E. Right subclavian artery. F. Left subclavian artery. G. Left vertebral artery intracranial part normotopia. H. Left vertebral artery intracranial part side position.

three days before the procedure. The patient was treated by general anesthesia and the 8F arterial catheter was put into the right femoral artery by Seldinger technique to make the whole body heparinization. The 8F guide catheter was put to the left subclavian artery and the PILOT 150 microguide wire was placed in the distal end of left vertebral artery V2. With the help of PILOT 150 microguide wire, the BLUE (Cordis) 5.0 mm × 15 mm saccule stent was sent to the stenosis part expanded at 12 atm. Radiography reexamination showed stenosis disappear and intracranial blood supply improvement. Next, the left carotid artery stenting was performed. Since the systolic pressure reached 200 mmHg, the patient was treated by urapidil. After sending the 5.0 mm protective umbrella (Cordis), a 5.5 mm × 30 mm sacculus was put in. The patient appeared twilight state and was treated by 8 atm quickly expansion. The haziness improved but the response was still bluntness. The response improved after the treatment of 9 mm × 40 mm stent (PRECISE Cordis). The operation was finished when the myodynamia of left upper limb reached degree 4, while the left lower limb attained degree 3^-, together with hypermyotonia. The symptom aggravated after 13 hours as the lower limb myodynamia declined to degree 0. Vascular ultrasound suggested severe right internal

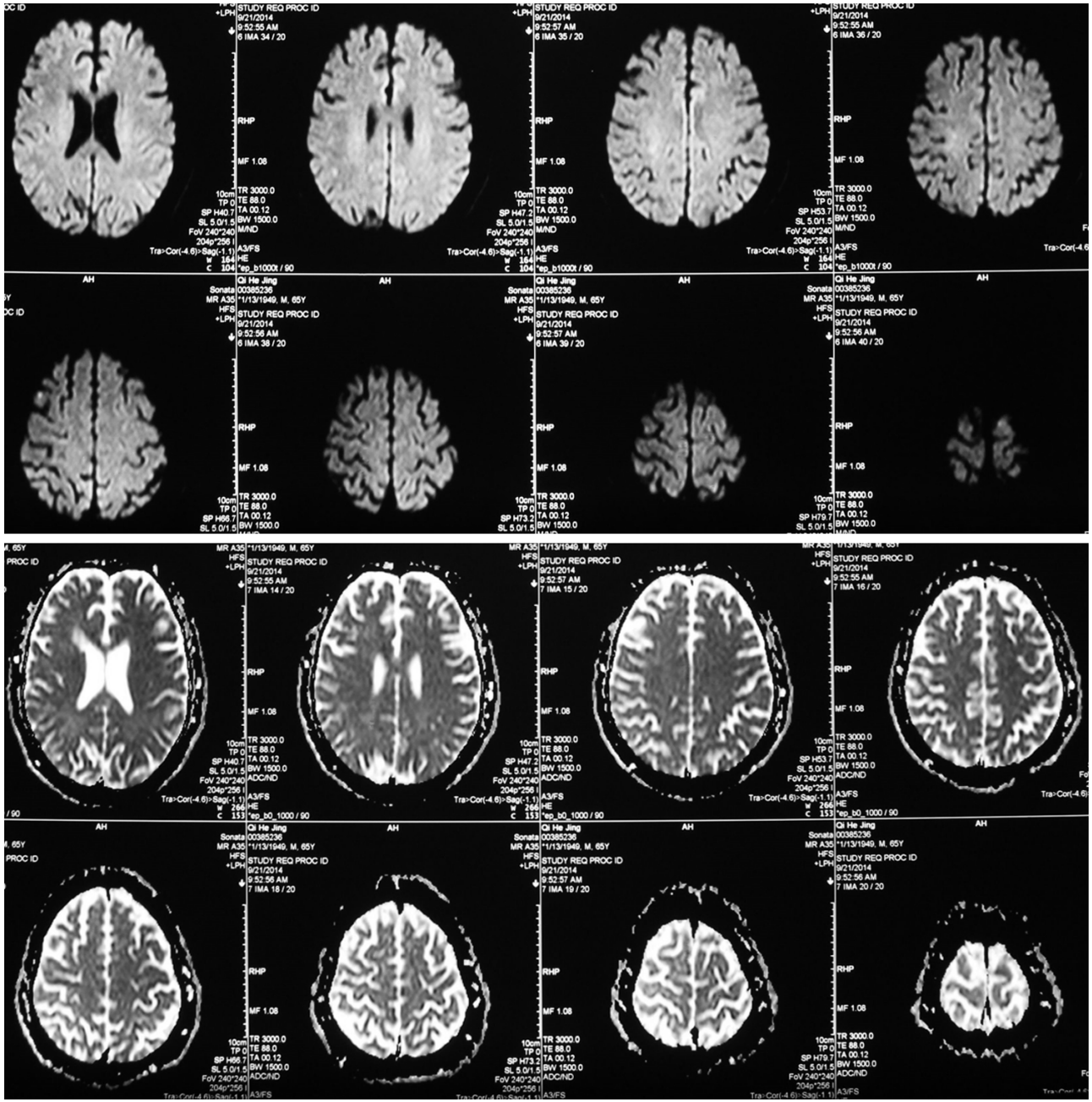

Figure 2. Brain MRI (upper, DWI; low, ADC).

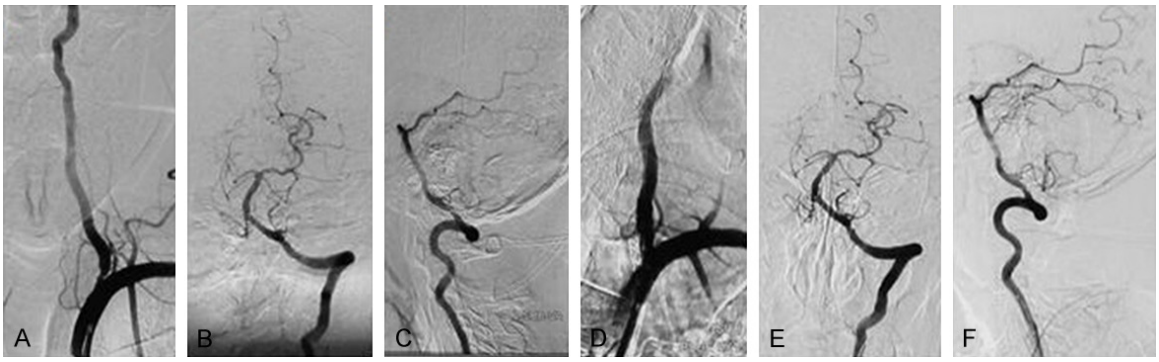

Figure 3. Left vertebral arterial stenting. A-C. Pre-operation. D-F. Post-operation.

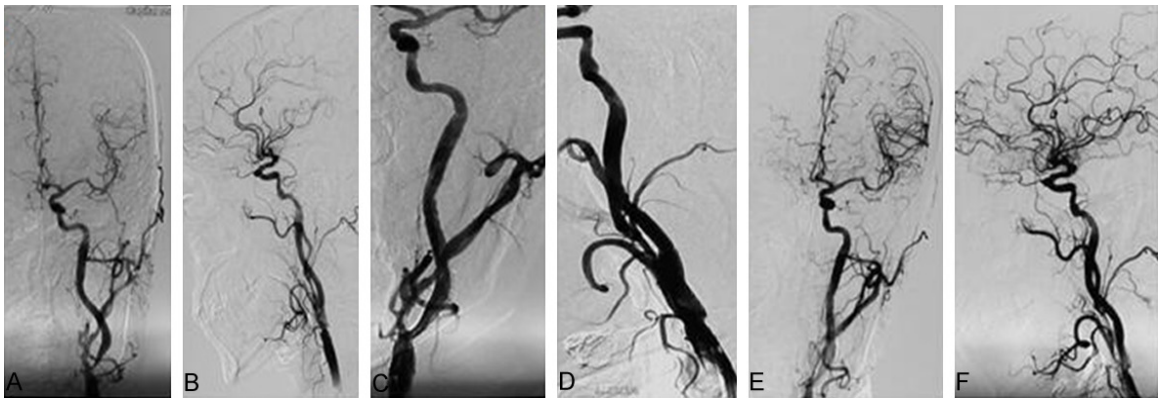

Figure 4. Left carotid artery stenting. A. Pre-operative left carotid artery normotopia. B. Pre-operative left carotid artery side position. C-F. Post-operative left carotid artery.

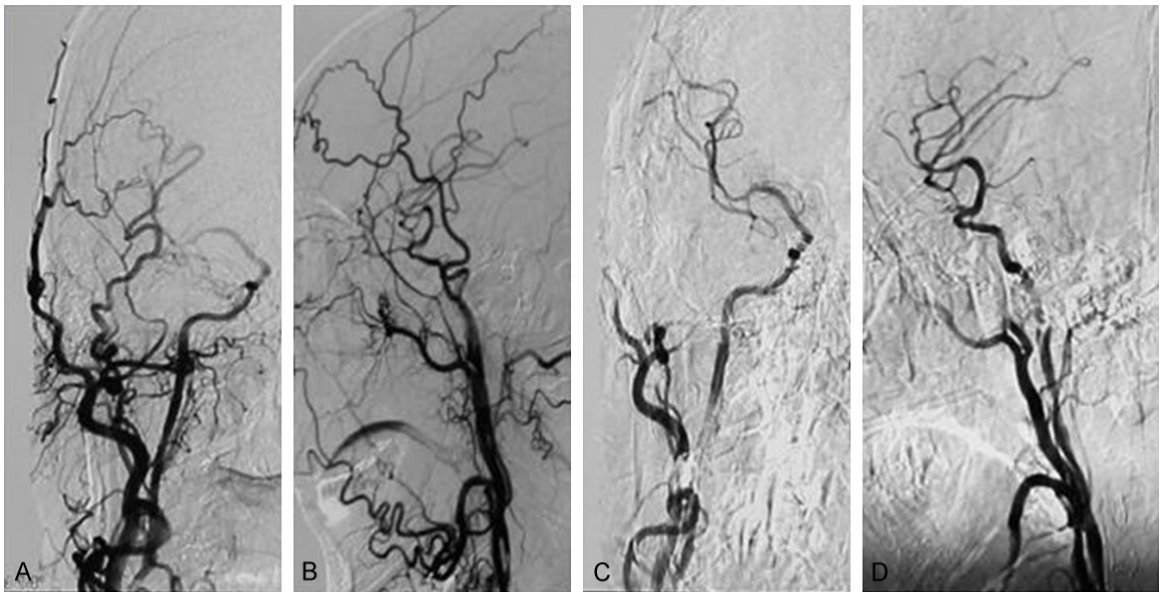

Figure 5. Right carotid artery stenting. A. Pre-operative right carotid artery normotopia. B. Pre-operative right carotid artery side position. C. Post-operative right carotid artery normotopia. D. Post-operative right carotid artery side position. Reexamination of TCCD revealed intracranial blood supply obviously improved, while the lateral branch of right carotid artery was closed. Brain MR+DWI indicated new infarction in right frontal and parietal lobe, and cingulate gyrus. The myodynamia of left upper limb achieved degree 4, while it was degree 4- in left lower limb.

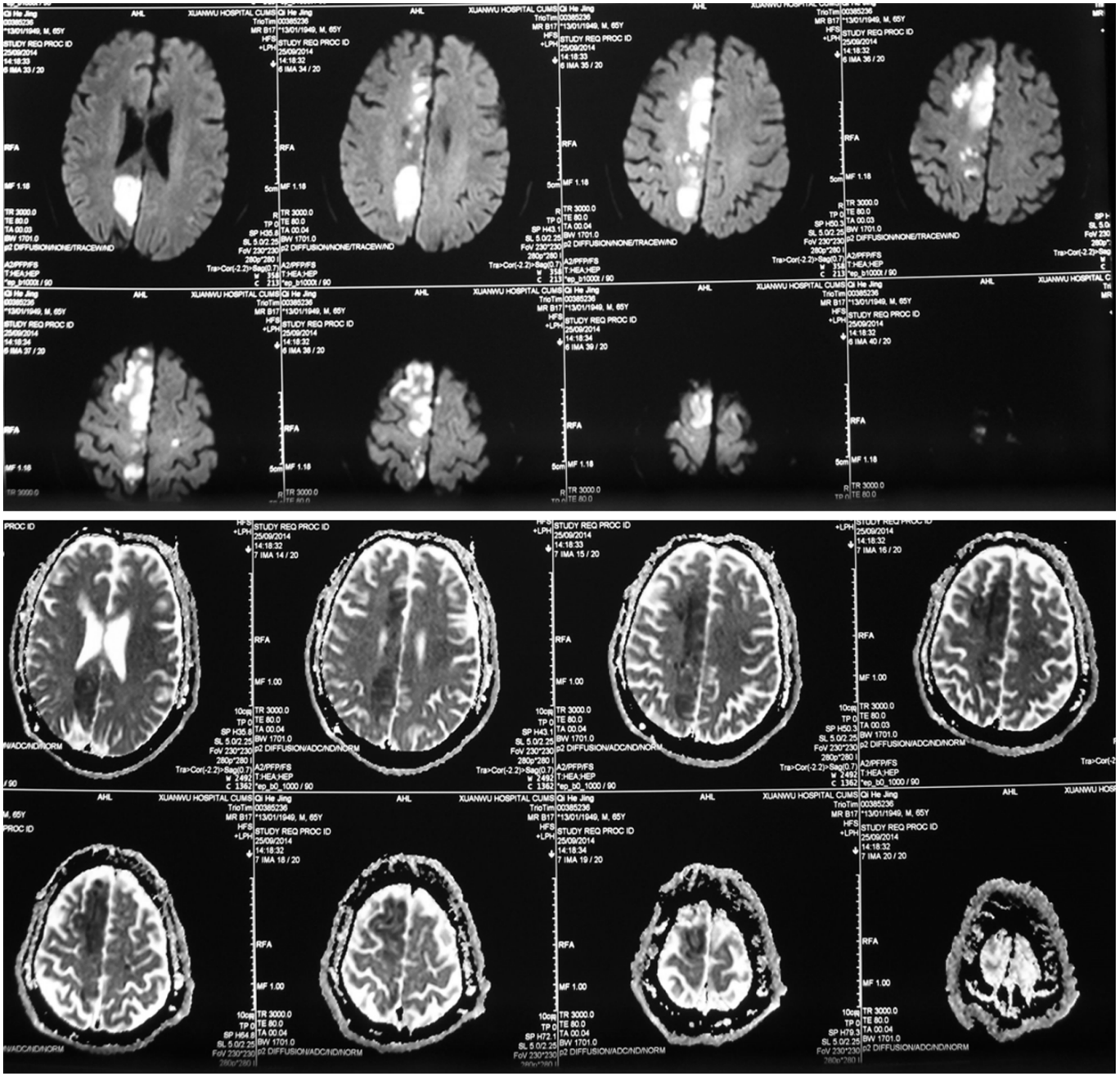

Figure 6. Brain MRI (upper, DWI; lower, ADC).

carotid artery stenosis. At last, the patient received right internal carotid artery stenting after 16 hours (**Figures 3**-**6**).

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Discussion

The incidence of stroke gradually rises in the elderly population following the society development. Meanwhile, it is also featured as rejuvenation. At present, the lowest average total incidence of cerebral apoplexy was 115.61/100,000 [3]. Stroke is a kind of disease with high morbidity and mortality. About 75% of cerebral apoplexy patients suffer from different degree of disability. Stroke has exceeded heart disease to become the leading cause of death during the last investigation. Carotid stenosis is the independent risk factor of cerebral arterial thrombosis [4], which accounts about 20-30%. Carotid endarterectomy (CEA) is the gold standard for the surgical treatment of carotid artery stenosis. Since CEA shows high risk to patient with high cervical segment stenosis, bilateral carotid lesions, and advanced age, carotid artery stenting is widely applied because of its wider indication, relative fewer contraindications, minimal invasion, quick recovery, and relatively simple technology. Several studies have confirmed the effectiveness and safety of stent implantation in carotid stenosis [5]. It was further verified that stent implantation has similar curative effect and safety compared with CEA in the treatment of carotid stenosis [6]. In recent years, the operation volume of stent implantation had exceeded CEA following the improvement of surgical technique and the development of surgical instruments. Carotid artery stenting becomes one of the first choices for the treatment of carotid stenosis together with CEA.

The complications of carotid artery stenting mainly include postoperative hypotension, hyperperfusion syndrome, local hematoma, cerebral hemorrhage, embolic detachment, and restenosis, etc. It was reported that 11.6% of asymptomatic internal carotid artery stenosis patient occurred postoperative hypotension after stent implantation [7]. In this study, the patient appeared intraoperative fall of blood pressure and left limb paralysis. Right anterior cerebral artery imaging disappeared after stenting. Hemiplegia may be caused by three reasons, such as hypoperfusion, postoperative hyperperfusion, and embolic detachment. However, they require different types of principle of management. Hypoperfusion should be corrected by dilatation and rising pressure, hyperperfusion needs controlling hypertension, while embolic detachment has to be treated by thrombolysis. Hyperperfusion syndrome (HPS) proposed by Sundt is caused by the loss of normal self-regulation mechanism and long-term hypoperfusion state [8]. Intracranial perfusion pressure significantly elevated, while blood flow volume obviously increased after the stenosis was relieved, resulting in the loss of regulation in peripheral vascular bed, blood brain barrier destruction, angiogenic cerebrocellular edema, and intracranial hemorrhage. It usually appears in the few hours to days after relieving the vascular stenosis. Imaging exhibits brain parenchyma or subarachnoid hemorrhage, and brain tissue swelling [9, 10]. As no contrast medium exosmosis occurred after hemiplegia in this study, HPS could be excluded. From the view of hemodynamics, detached emboli typically flow to the large blood vessels with fastest velocity. In this patient, embolic detachment may block the ipsilateral middle cerebral artery, resulting in right limb paralysis. Right anterior cerebral artery A1 branch showed no image before operation, indicating incomplete Willis ring. Right anterior cerebral artery can be compensated by the left anterior cerebral artery through anterior communicating artery. The cortex arterioles undergo the longest distance to reach the blood supply region, thus it is easy to be affected by hypotension because of poor collateral circulation compensation. To sum up, we considered the patient was suffered from hypoperfusion ischemic stroke, therefore performed dilation and boosting. The patient quickly recovered and the brain MRI also supported our judgement.

Postoperative hypotension is the most common complication of carotid artery stenting, while the hypoperfusion cerebral infarction is rare. The distance between stenosis and furcation (≤10 mm), eccentric stenosis, and calcifi-

cation are the risk factors of hypotension after stent implantation [11]. Patients with contralateral internal carotid artery stenosis or occlusion more easily experience postoperative hypotension [12]. Short-term hypotension is directly stimulated by the sacculus or stent, leading to the vagus nerve excitability elevation, heart rate slow down, and blood pressure fall. It was suggested that improvement of cerebral blood flow perfusion was significantly positively related to the degree of carotid artery stenosis, but not with the improvement of carotid stenosis degree [13]. It indicated that the excessive expansion of stenosed artery does not increase the cerebral blood flow perfusion speed. It was reported that the normal electrophysiological activities of the nerve cells could be maintained when the cerebral blood flow reached more than 50% of the normal [14]. Complete Willis ring is an important approach to establish intracranial vascular collateral circulation. In conclusion, the hypoperfusion cerebral infarction during stent implantation may be caused by following factors. Firstly, the stenosis segment near the sphere of internal carotid artery and is characterized as eccentricity and calcification. Secondly, the lesions combined with contralateral internal carotid artery stenosis or occlusion. Thirdly, incomplete Willis ring without other collateral compensation. Lastly, the excessive expansion of stenosis segment. Thus, reduce the times and strength of balloon dilation can reduce the risk of hypotension. On the other hand, positively dilation and continuous boosting can improve the prognosis. Larger scale clinical trial is needed to further investigate the best clinical intervention on hypoperfusion cerebral infarction in the future.

Disclosure of conflict of interest

None.

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