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Case Report

Crossed Cerebellar Diaschisis and Cerebral Infarction After Cerebral Hyperperfusion Syndrome Following Carotid Artery Stenting: A Case Report

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Abstract

Crossed cerebellar diaschisis (CCD) often occurs after ischemic or hemorrhagic stroke that does great damage to the cortico-ponto-cerebellar pathway (CPCP). Nevertheless, as far as we know, CCD due to cerebral hyperperfusion syndrome (CHS) following carotid artery stenting (CAS) is rare. We report a case where CCD and new cerebral infarction after CHS following CAS was effectively treated by intravenous use of edaravone and mannitol. The patient was a 74-year-old female. She developed dizziness and vomiting accompanied with weakness of the right limb for 8 days and was admitted to our hospital. Computed tomography angiography scan revealed severe stenosis at the beginning of the left internal carotid artery. Further digital subtraction angiography (DSA) revealed severe stenosis at the beginning of the left internal carotid artery, with a stenosis rate of approximately 90%. Therefore, she underwent left CAS implantation. After the operation, the patient developed disturbance of consciousness and decreased muscle strength in the right limb. Intravenous infusion of edaravone and mannitol were then started. After 10 days of medical treatment, the condition of patient improved to mild hemiparesis. The findings in this present case strongly suggest that CHS after CAS for carotid artery stenosis may cause transient CCD.

Keywords

Carotid Artery Stenting, CCD, Hyperperfusion Syndrome, Carotid Artery Stenosis, Digital Subtraction Angiography

1. Introduction

In the 1980s, crossed cerebellar diaschisis (CCD) was first described [1]. CCD is defined as a decrease in matching metabolism and blood flow in the contralateral cerebellar hemisphere of supratentorial lesions. It is often occurs after ischemic or hemorrhagic stroke that damages the cortico-ponto-cerebellar pathway (CPCP) [2-4]. This phenomenon is usually secondary to decreased cerebral perfusion. Nevertheless, it may also be related to supratentorial cerebral hypoperfusion syndrome after vascular reconstruction surgery [5]. Previous study found that CCD due to cerebral hyper-

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perfusion syndrome (CHS) following cerebrovas-cular reconstruction is rare [5].

CHS is mainly occurs after revascularization surgery, including carotid endarterectomy and superficial temporal artery tomiddle cerebral artery anastomosis [6-8]. As far as we know, there have been few reports of transient CCD and new cerebral infarction due to CHS after carotid artery stenting (CAS).

We report a case of transient CCD and new cerebral infarction secondary to CHS following CAS.

2. Case Report

A 74-year-old female developed dizziness and vomiting accompanied with weakness of the right limb, which lasted for 8 days and was admitted to our hospital. The patient has a history of hypertension and diabetes, and takes drugs irregularly. Magnetic resonance imaging (MRI) revealed that there is a fresh lacunar infarction (LI) of the right medulla oblongata (Figure 1 A). Computed tomography angiography (CTA) scan revealed severe stenosis at the beginning of the left internal carotid artery (Figure 1 B). Therefore, the diagnosis were LI and the left internal carotid artery stenosis. Considering the presence of the left internal carotid artery in patient, further digital subtraction angiography (DSA) assessment of cerebrovascular condition is necessary. Therefore, we performed DSA on patient which revealed severe stenosis at the beginning of the left internal carotid artery, with a stenosis rate of approximately 90% (Figure 1 C).

Based on the comprehensive imaging results, the indications for endovascular treatment of severe stenosis at the beginning of the left internal carotid artery in patient is clear. Therefore, she underwent left CAS implantation (Figure 1 D).

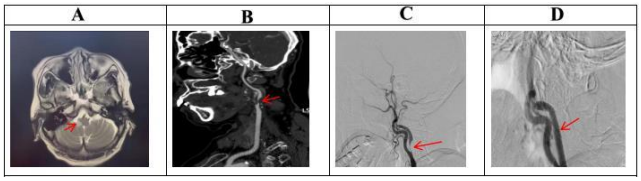

Figure 1. *A: Magnetic resonance imaging revealing a fresh lacunar infarction of the right medulla oblongata (red arrow). B, C: Computed tomography angiography and digital subtraction angiography revealing severe stenosis at the beginning of the left internal carotid artery (red arrow). D: After carotid artery stent implantation (red arrow).*

After the procedure, patient developed disturbance of consciousness and decreased muscle strength in the right limb. We immediately re-examined the brain computed tomography (CT). Brain CT found that diffuse high density shadows can be seen in the left frontal lobe, temporal lobe, parietal lobe, occipital lobe, insula lobe and basal ganglia area, the left brain sulcus and gyrus become shallow (Figure 2 A). Based on brain the CT imaging results, we believed that patient had developed CHS after CAS. We immediately administered intravenous injections of mannitol and edaravone, and used urapidil to control blood pressure between 120-130 mmHg. As the patient's right limb strength improves in three days later, we arranged for her to undergo diffusion weighted imaging (DWI) and arterial spin labeling perfusion (ASL) examinations. Brain DWI found new cerebral infarction in the left basal ganglia area, thalamus and lateral ventricle (Figure 2 B). ASL perfusion confirmed reduced perfusion in the left cerebral hemisphere, basal ganglia area and right cerebellar hemisphere, which suggests CCD (Figure 2 C, D).

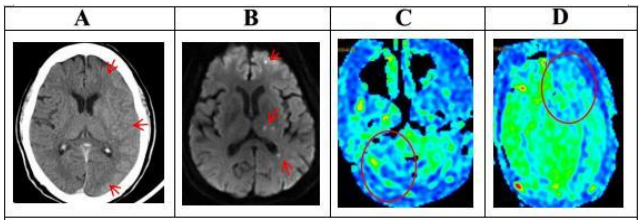

Figure 2. *A: Brain computed tomography revealing postoperative hyperperfusion (red arrow). B: Brain diffusion weighted imaging revealing new cerebral infarction in the left basal ganglia area, thalamus and lateral ventricle (red arrow). C, D: Arterial spin labeling perfusion revealing reduced perfusion in the left cerebral hemisphere, basal ganglia area and the right cerebellar hemisphere (red circle).*

Considering the patient's symptoms have improved compared to before, we continued to maintain the above treatment plan. After 10 days of treatment with the aforementioned drugs, patient's condition improved to mild hemiplegia. Examination of brain computed tomography perfusion imaging (CTP) displayed CCD had disappeared (Figure 3 A, B, C, D).

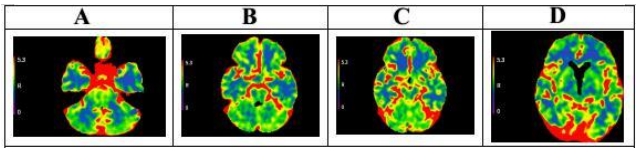

Figure 3. *A, B, C, D: Examination of brain computed tomography perfusion imaging revealing crossed cerebellar diaschisis disappeared.*

The patient was discharged 3 weeks after surgery with mild hemiplegia. No cerebrovascular events occurred during the follow up period.

3. Discussion

This is a very rare case of transient CCD and new cerebral

infarction following CHD after CAS for adult carotid artery stenosis.

Currently, prophylactic surgeries such as carotid endarterectomy (CEA) or CAS have been considered to prevent ischemic strokes and treat arterial stenosis [9]. Previous study confirmed no significant difference in stroke prevention between CAS and CEA [10, 11]. Compared with CEA, CAS has less invasiveness and has become an alternative method for CEA treatment of symptomatic or asymptomatic carotid artery stenosis [12, 13]. The most common complication after CAS is new infarction detected by DWI [14]. The incidence rate after CAS is 37% (9% -70%), while that after CEA is 10% (0% -27%) [14]. Schillinger and his team had confirmed that expansion of stent will expand the narrow lumen, but the radial force of this expansion on the carotid artery wall can cause plaque collapse and enter the lumen [15]. Therefore, during the deployment of stent, the contents of plaque (lipids, thrombi, and calcification) slipped over the stent support, leading to cerebral infarction (new DWI lesion) [15]. Our patient developed new cerebral infarction after CAS, we believed was related to plaque detachment after stent implantation. The subsequent CHS leading to plaque blockage to the distal small vessels. CHS mainly includes cephalalgia, seizure, focal neurological deficits, and intracerebral hemorrhage, and is sometimes fatal [16]. Therefore, we suggest that patients after CAS should pay close attention to whether the above symptoms appear for early intervention. Immediate cerebral blood flow monitoring after surgery can also help monitor the occurrence of CHS and prevent neurological complications related to high perfusion caused by sedation and blood pressure control [17].

There had been report of transient CCD after CEA surgery in the past [18], however, as far as we know, there are currently no reports of CCD caused by CHS after CAS. Generally, interruption of the CPCP causes CCD [19, 20]. Prevous study confirmed that human frontal cortex, especially the premotor and prefrontal regions, has a strong regulatory effect on metabolism of contralateral cerebellum [21]. Therefore, postoperative hyperperfusion may functionally inhibit the relatively wide cerebral cortex and reduce metabolic activity in contralateral cerebellar hemisphere, which is a distant effect [5]. The patient's brain CT significantly revealed excess blood flow in the left cerebral hemisphere. Although brain CT revealed no midline structure shifted, patient developed confusion. We supposed that CHS caused contralateral CCD and CCD may affect the brainstem reticular activation system. We speculated that the presence of ischemic infarction or incomplete Willis ring in posterior circulation supply area before CAS may increase the risk of CHS causing disturbance of consciousness and infarct side CCD after CAS. Therefore, CCD may be a marker of early deterioration of CHS. This speculation requires further research on a large sample size. Early onset of disturbance of consciousness in CHS requires attention to CCD, especially in patients with incomplete Willis ring or ischemic infarction in posterior circulation supply area before CAS. Although this phenomenon is rarely reported, further research is needed to elucidate the relationship between CCD and CHS.

4. Conclusion

The findings in this present case strongly suggest that CHS after CAS for carotid artery stenosis may cause transient CCD. We speculated that the presence of ischemic infarction or incomplete Willis ring in posterior circulation supply area before CAS may increase the risk of CHS causing disturbance of consciousness and infarct side CCD after CAS. Therefore, CCD may be a marker of early deterioration of CHS. Although the clinical significance is still obscure, this phenomenon indicates the CPCP is interrupted due to CHS, suggesting postoperative critical care and careful observations of cerebral hemodynamics are recommended to prevent CHS.

Declarations

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Author Contributions

WQ-N: data collection, analysis and manuscript writing. SQ-C, SP-D: data collection and manuscript writing. SS-Z: manuscript revision and interpretation of data. All authors read and approved the final manuscript.

Data Availability

The authors declare that data supporting the findings of this study are available within the article.

Conflicts of Interest

The authors declare no conflicts of interest.

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