Case Report

Bilateral Atherosclerotic Internal Carotid Artery Occlusion with Intact Cerebral Glucose Metabolism: A Case Report

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Background: Mild neurologic deficits concomitant with bilateral internal carotid artery occlusion (BICAO) is very rare and its treatment is still unclear. *Case Report:* Herein, we report a case of a 67-year-old man with BICAO. The collateral circulation was rich, the symptoms were mild, and only standard pharmacotherapy was prescribed. Follow-up Mini-Mental State Examination, fluorine-18-fluorodeox yglucose positron emission tomography, and magnetic resonance perfusion-weighted imaging were performed for 6 months. *Results:* The results showed uniform reduction in perfusion throughout the brain, normal glucose uptake by the brain, and no ischemic events and cognitive impairment during the follow-up period. *Conclusions:* For BICAO patients who are with mild neurologic deficits and good cerebral collateral and metabolism, the timely administration of pharmacotherapy might be safe and effective. Thus, in our patient, a favorable prognosis was achieved, but further follow-up is still required. **Key Words:** Internal carotid artery—occlusion—glucose uptake.

Bilateral internal carotid artery occlusion (BICAO) is very rare, and its treatment is still unclear.[1] Theoretically, BICAO may cause severe ischemia in the anterior circulation, resulting in severe neurologic deficits. Thus, blood vessel reconstruction is needed to attenuate the ischemia. We report a case of a patient with BICAO who showed mild neurologic deficits associated with the rich collateral circulation and normal glucose uptake by the brain. This was the first case of BICAO—dynamically monitored with fluorine-18-fluorodeoxyglucose ([^{18}F]FDG) positron emission tomography (PET)—in which glucose uptake by the brain was not significantly impaired.

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Case Presentation

A male patient aged 67 years was admitted for slurred speech and weakness of the left upper limb. This patient had a history of hyperlipidemia, hypertension, and smoking. On admission, his blood pressure was 156/82 mm Hg, pulse was 82 beats/minute, and heart rhythm was normal. The patient was conscious and cooperative, but slurred speech was noted. He answered the questions correctly, and the strength of his left upper limb was grade 4. Blood biochemistry showed a total cholesterol of 6.14 mmol/L, triglyceride of 1.82 mmol/L, and low-density lipoprotein cholesterol of 4.2 mmol/L. Erythrocyte sedimentation rate, rheumatism-related antibodies, thyroid function, D-dimer level, and virus serology were normal. Transcranial Doppler showed (1) possible severe stenosis of the bilateral internal carotid arteries (ICAs) at their origin; (2) increased blood flow through the bilateral external carotid artery and reversed blood flow through the bilateral ophthalmic arteries; (3) significantly reduced vascular resistance and increased flow through the bilateral vertebral–basilar arteries. Brain

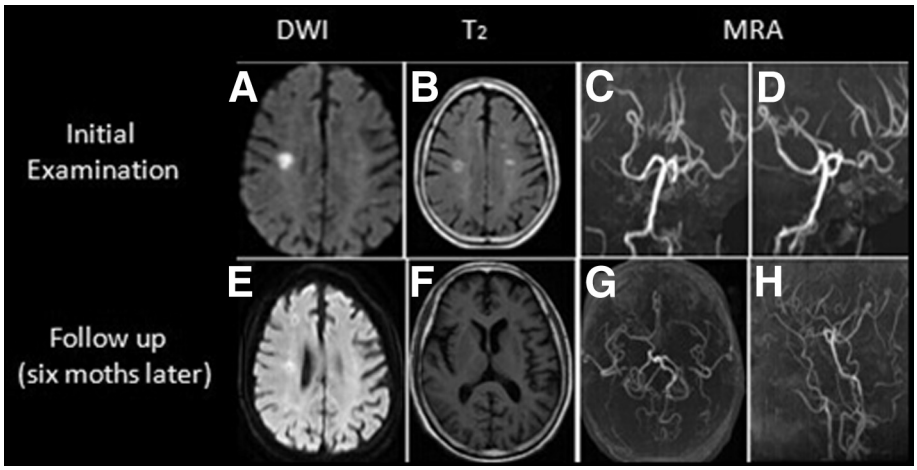

Figure 1. *The brain MRI and MRA findings. Diffusion-weighted (A) and T2-weighted (B) images reveal a subacute infarct at the base of the right corona radiata. MRA (C, D) shows the absence of both internal carotid arteries and supply of the left MCA and ACA by an abnormally dilated posterior communicating artery. MRI and MRA (E-H) at 6 months after therapy show similar findings and the absence of new ischemic foci. Abbreviations: ACA, anterior cerebral artery; MCA, middle cerebral artery; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging.*

magnetic resonance imaging (MRI) revealed subacute infarction of the corona radiata, and MR angiography (MRA) revealed the complete occlusion of the bilateral ICAs and the bilateral middle and anterior cerebral arteries (Fig 1). Digital subtraction angiography (Fig 2) confirmed the complete occlusion of the bilateral ICAs at their origin and revealed a rich collateral flow to the anterior circulation. Initial and follow-up brain MR perfusion-weighted imaging and [^{18}F]FDG PET (Fig 3 and Table 1) revealed a decreased cerebral blood flow (CBF) and a largely normal mean standardized uptake value (SUVavg) throughout the brain.

After admission, dual antiplatelet therapy with aspirin and clopidogrel and drugs to lower lipids and control blood pressure were administered. After treatment for 15 days, the symptoms were resolved, a Mini-Mental State Examination score was 28/30 and the patient was discharged. During the 6-month follow-up period, no recurrence was observed. After 6 months, the findings from Mini-Mental State Examination, transcranial Doppler, and MR angiography were similar to those before, and cranial MRI found no new ischemic foci (Figs 1 and 3). Comparisons of CBF and SUVavg before and after therapy (Table 1) showed the development of collateral flow from the posterior circulation to the anterior circulation and unchanged brain metabolism.

Discussion

BICAO can be caused by moyamoya disease, atherosclerosis, radiation injury, trauma, and fibromuscular dysplasia.[4-6] In this patient, complete BICAO was

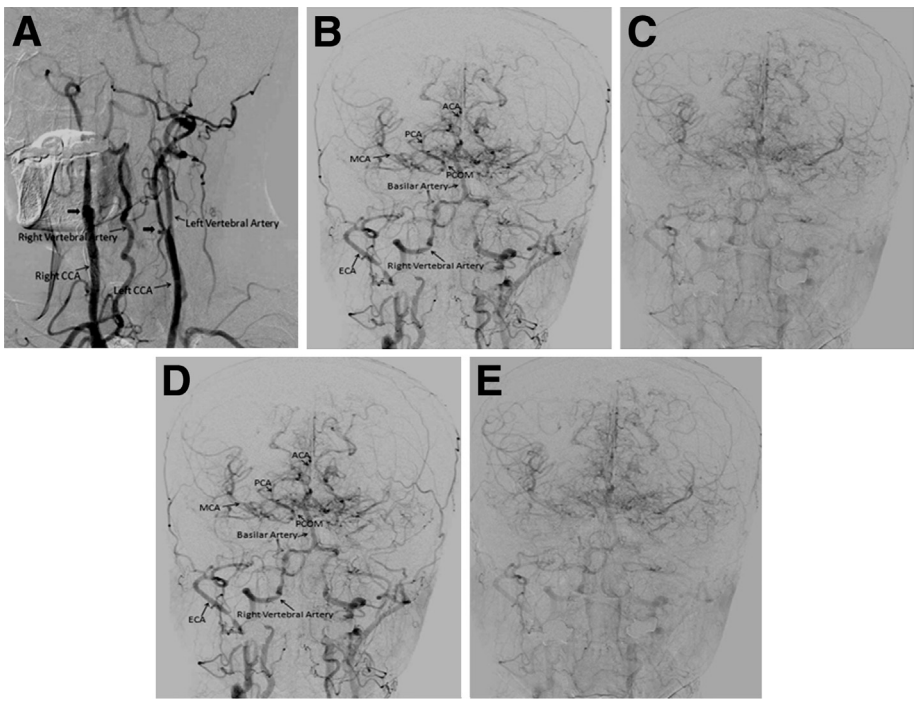

Figure 2. *DSA. (A) The occlusion of the bilateral ICAs at the level of bifurcation (arrow). (C, E) Excessively developed network of collateral vessels extending from the leptomeningeal vessels to the anterior and middle cerebral artery territory (panel B and D, early arterial phase; panel C and E, late arterial phase). (B) Rich collateral supply to the anterior circulation through the posterior communicating artery. (D) Reversed flow through the ophthalmic artery to the bilateral internal carotid arteries. Abbreviations: ACA, anterior cerebral artery; CCA, common carotid artery; DSA, digital subtraction angiography; ECA, external carotid artery; ICA, internal carotid artery; MCA, middle cerebral artery; PCA, posterior cerebral artery; PCOM, posterior communicating artery.*