Frontal Lobe Infarction Due to Hemodynamic Change after Surgical Revascularization in Moyamoya Disease
—Two Case Reports—

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Abstract

A 60-year-old female and a 40-year-old male underwent surgical revascularization for moyamoya disease and suffered small infarction in the ipsilateral frontal lobe 3 or 4 days postoperatively. Neuroimaging suggested that the bypass flow had caused rapid progression of occlusive changes in the carotid forks, a diminishing of moyamoya vessels, and flow reduction in the anterior cerebral artery ipsilateral to surgery, leading to critical ischemia in the frontal lobe. Surgical revascularization improves the outcome of patients with moyamoya disease, but postoperative management such as hydration is important to avoid ischemic complications due to frontal lobe infarction.

Key words: moyamoya disease, bypass surgery, cerebral ischemia, anterior cerebral artery, frontal lobe, complication

Introduction

Moyamoya disease is a peculiar cerebrovascular disorder characterized by spontaneous stenosis or occlusion of the bilateral carotid forks associated with abnormal vascular networks at the base of the brain (“moyamoya” vessels). The “moyamoya” vessels are dilated medullary arteries providing important collateral circulation pathways to maintain normal cerebral perfusion, which otherwise would be disturbed by the occlusive changes of the carotid forks. Cerebral angiography often shows the distal branches of the anterior (ACA) and/or middle cerebral arteries (MCA) as opacified via the “moyamoya” vessels.³⁰

Surgical revascularization procedures such as superficial temporal artery (STA)-MCA anastomosis and/or indirect synangiosis can improve cerebral hemodynamics, and resolve ischemic attacks in both children and adults.⁷,⁹,¹¹,¹²,¹⁷,¹⁸,²³ However, surgical revascularization may be complicated by ischemic stroke.¹⁰,¹⁶,¹⁰–²² In such cases, the cerebral infarction develops in the cortical areas that were hemodynamically compromised before surgery.¹⁰

We treated two patients who developed cerebral infarction in the ipsilateral frontal lobe several days after surgical revascularization. Neuroimaging revealed that the revascularization surgery for the territory of the MCA had resulted in rapid progress of carotid fork stenosis, reducing blood flow in the ACA. Such complications are very specific to moyamoya disease.

Case Reports

Case 1: A 60-year-old female had an episode of transient left hemiparesis 2 years before. She suffered sudden onset of headache, and was admitted to our hospital. Neurological examination on admission revealed no deficit. Computed tomography (CT) demonstrated intraventricular hemorrhage, and cerebral angiography revealed severe occlusive changes of the bilateral carotid forks associated with moyamoya vessels. The origin of the right ACA was occluded, and the distal branches were opacified via the moyamoya vessels (Fig. 1 upper). Her symptoms resolved within 2 weeks of onset. Single photon emission CT (SPECT) showed that regional cerebral blood flow (rCBF) was normal, but reactivity to acetazolamide was reduced in the right frontal lobe, suggesting reduced perfusion reserve.¹³

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Two months later, she underwent STA-MCA anastomosis and encephalo-duro-arterio-myo-synangiosis (EDAMS) on the right to prevent the recurrence of ischemic or hemorrhagic stroke. The postoperative course was uneventful until she developed transient weakness of the left leg, lasting for 5 minutes, 4 days after operation. Magnetic resonance (MR) imaging revealed a small infarction in the deep white matter of the right frontal lobe (Fig. 2). Three-dimensional time-of-flight MR angiography taken 7 days after surgery showed disappearance of the signal of the pericallosal arteries, which was observed on preoperative three-dimensional MR angiography (Fig. 3). SPECT on the same day demonstrated decreased blood flow in the right frontal lobe. She was treated with intravenous infusion of low-molecular dextran solution. She did not suffer any further transient ischemic attacks (TIAs). Follow-up angiography performed 3 months after surgery revealed marked attenuation of the
moyamoya vessels and development of collateral flow via the external carotid artery system in the territories of the MCA and ACA. The right ACA was not opacified because of diminished moyamoya vessels (Fig. 1 middle, lower). Postoperative vertebral angiography showed no change in the collateral circulation. SPECT taken 3 months after surgery showed complete normalization of rCBF and acetazolamide reactivity in the right cerebral hemisphere.

Case 2: A 40-year-old male experienced transient weakness of the right extremities, and was admitted to our hospital. Neurological examination on admission revealed no abnormalities. MR imaging showed no infarction, but cerebral angiography demonstrated stenosis of the bilateral carotid forks associated with moyamoya vessels. The bilateral ACAs were slowly opacified through the moyamoya vessels of the left side (Fig. 4 upper). SPECT demonstrated reduced rCBF and decreased reactivity to acetazolamide in the left hemisphere (Fig. 5 left).

He underwent STA-MCA anastomosis and EDAMS on the left. The postoperative course was uneventful until he suddenly developed mild weakness of the right arm and dysphasia 3 days after surgery. Laboratory examination revealed postoperative anemia based on a hemoglobin concentration of 8.5 g/dl. He was treated with blood transfusion and intravenous infusion of low-molecular dextran solution to increase oxygen transport to the brain. Diffusion-weighted MR imaging immediately after the onset revealed a small cortical infarction in the frontal lobe (Fig. 6). SPECT on the same day showed reduced rCBF in the left frontal lobe including the ACA territory (Fig. 5 center). His neurological symptoms completely resolved within 2 weeks of the onset.
Fig. 6 Case 2. Diffusion-weighted magnetic resonance (MR) image (left) taken just after neurological deterioration showing an acute ischemic lesion in the frontal cortex. Follow-up T2-weighted MR image (right) confirming the small infarction in the same region.

Follow-up angiography 2 months later showed good development of collateral flow via the external carotid artery system into the MCA and ACA territories, marked progression of the stenosis of the carotid fork, and disappearance of moyamoya vessels on the left. Antegrade filling of the bilateral ACAs was not observed (Fig. 4 middle, lower). Postoperative vertebral angiography showed no change in the collateral circulation through the posterior pericallosal arteries. Follow-up SPECT taken one month after surgery showed increased rCBF in the left cerebral hemisphere, including the frontal lobe (Fig. 5 right). Subsequently, he underwent STA-MCA anastomosis combined with EDAMS on the right 6 months after the first operation. Anemia was not detected after the second surgery, and the postoperative course was uneventful.

Discussion

Our patients suffered TIAs or reversible ischemic neurological deficits due to frontal lobe infarction 3 to 4 days after surgical revascularization for moyamoya disease. The development of such infarction has not been reported previously, although cerebral infarction is a known complication of surgery for moyamoya disease in areas, which are hemodynamically compromised on preoperative studies. The neuroimaging and neurological findings are very similar in both cases. Preoperative angiography showed the origins of the ACA(s) were occluded and the peripheral ACA(s) were opacified via the developed moyamoya vessels. MR imaging revealed development of infarction in the frontal lobe ipsilateral to surgery after the patients suffered stroke or TIAs 3 to 4 days after surgery. Neurological symptoms were mild and did not persist. Postoperative angiography showed that the MCA areas were widely fed by the direct and indirect bypass flow, and that occlusive changes in the carotid fork had progressed. In addition, antegrade filling of the ACA(s) had disappeared due to diminished moyamoya vessels. Postoperative SPECT taken soon after the onset of postoperative events demonstrated transient decrease of rCBF in the ipsilateral frontal lobe, including the ACA territory. These observations strongly suggest that postoperative development of bypass flow had stimulated occlusive changes in the carotid fork and diminished moyamoya vessels, resulting in blood flow reduction in the ACA which was opacified through the moyamoya vessels before surgery. Postoperative flow reduction in the ACA is considered to cause cerebral infarction in the ipsilateral frontal lobe, and this correlates very well with postoperative MR angiography findings in Case 1, which showed that the flow signals of the pericallosal arteries had completely vanished only 7 days after surgery. Such a postoperative complication has never been recognized in patients with moyamoya disease, although surgical revascularization sometimes causes TIAs of the bilateral legs due to ischemia of the paracentral lobules supplied by the ACA.

Intracranial arterial stenosis may progress to occlusion after STA-MCA anastomosis, and the patients sometimes develop ischemic stroke because of postoperative occlusion of the intracranial internal carotid artery (ICA) or MCA stenosis. Postoperative occlusion was observed in six of 19 patients with intracranial ICA or MCA stenosis, although these patients remained asymptomatic. New collateral circulation through the anastomosed STA may produce a critical flow reduction through the stenotic arteries, leading to thrombotic occlusion. Most reported patients suffered symptomatic occlusion within 2 weeks of the operation, suggesting surgery is very likely to promote occlusive changes compared to the natural history of the stenotic arteries.

The angiographical findings of moyamoya disease are classified into six stages. Follow-up studies have shown that the occlusive changes in the carotid forks of moyamoya disease progress very slowly until adolescence and then stabilize. In contrast to this natural course, angiographical changes after surgical revascularization are very rapid. Once collaterals develop after surgery, the occlusive changes of the carotid forks rapidly progress to the advanced stages and the moyamoya vessels markedly diminish.
within a few months. Angiographic staging of moyamoya disease may advance further in patients with extensive rather than minimal development of collaterals. Such rapid occlusive changes of the carotid forks are very similar to those occurring in patients who underwent STA-MCA anastomosis for intracranial ICA or MCA stenosis. However, the postoperative frontal lobe infarction in our patients must be specific to moyamoya disease, because adequate bypass flow caused progression in the occlusive changes over the origins of the MCA and ACA, and diminished the moyamoya vessels which provided the main collaterals for the ACA (Fig. 5).

Various factors have been proposed as the risk indicators of perioperative ischemic complications: Frequent TIAs, indirect bypass procedures, hemodynamic compromise, dehydration, crying, hypotension, and hypercapnia as well as hypocapnia. However, neither of our patients had any risk indicator except for hemodynamic compromise before surgery. Direct revascularization such as STA-MCA anastomosis does improve outcome and reduce the risk of perioperative ischemic stroke compared with indirect surgery, because the bypass flow through the anastomosed STA can improve cerebral hemodynamics immediately after surgery. However, direct bypass surgery also carries the risk of rapid changes in the carotid forks and/or rapid diminishing of moyamoya vessels. Therefore, perioperative anemia, dehydration, and other risk factors must be avoided to prevent frontal lobe infarction due to disturbed flow in the ACAs after direct bypass surgery for moyamoya disease.

References


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