Symptomatic Intracranial Artery Stenosis Due to an Unknown Embolus Following Cardiac Surgery: A Case Report

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Abstract

Intracranial artery occlusion due to a foreign body is a complication associated with cardiac surgery that is treated by various techniques. However, little is known about appropriate strategies for symptomatic intracranial artery stenosis due to an unknown embolic source. We reported a case of middle cerebral artery (MCA) stenosis after mitral valve repair (MVR) for infective endocarditis (IE). An 80-year-old man presented with right hemiplegia. MR angiography findings were normal, and diffusion-weighted imaging revealed subtle ischemic change in the left MCA territory. The patient was diagnosed with cardioembolic stroke owing to IE and performed MVR. Four days later, he suddenly presented with consciousness disorder and left hemiplegia. Computed tomography (CT) confirmed a very low-density area within the right MCA. MR angiography revealed right MCA stenosis, which corresponded to the low-density area on CT images. Diffusion-weighted imaging revealed new ischemic change in the right MCA territory. Angiography confirmed an irregular stenosis at the right M2 with antegrade blood flow, and the hemiplegia resolved during angiography. Conservative therapy was performed; however, the resting 123 I-IMP-single photon emission CT revealed moderate perfusion defect in the right MCA territory, and transient left hemiplegia appeared every few days. Therefore, 19 days after the initial transient ischemic attack, the patient was performed superficial temporal artery–MCA anastomosis, and the patient responded with a good clinical course without recurrence of the ischemic symptoms. This strategy may be a safe and effective treatment for symptomatic intracranial artery stenosis due to an unknown embolic source.

Keywords: infective endocarditis, intracranial artery stenosis, mitral valve repair, superficial temporal artery to middle cerebral artery anastomosis, unknown embolic source

Introduction

Intracranial foreign body embolus is a possible complication following cardiac surgery. The previous literature describes major intracranial artery occlusion owing to intravascular foreign bodies that include valve tissue,1 chordae tendineae,2 calcified and collagenous tissue,3 epicardial pacing wires, and catheter tips.4,5 These foreign bodies were removed by various surgical embolectomy or endovascular retrieval techniques; however, little is known about the appropriate strategies for symptomatic intracranial artery stenosis due to an unknown embolic source.

In this study, we report a case of symptomatic middle cerebral artery (MCA) stenosis due to an unknown embolic source after mitral valve repair (MVR) for infective endocarditis (IE). We performed superficial temporal artery–middle cerebral artery (STA–MCA) anastomosis. The transient mild hemiplegia completely disappeared after surgery, and
the patient did not suffer neurological deterioration during the follow-up period.

**Case Description**

An 80-year-old man with a history of paroxysmal atrial fibrillation and dental decay presented with sudden mild right hemiplegia and fever, and was admitted to a local hospital. Computed tomography (CT) scans confirmed no abnormal findings (Fig. 1A). MR angiography findings were almost normal (Fig. 1B), and diffusion-weighted imaging revealed subtle acute ischemic change in the left MCA territory (Fig. 1C). Transthoracic echocardiography revealed mitral valve...

**Fig. 1** (A–C) Image findings on admission at the previous hospital. (D–I) Image findings 4 days after the mitral valve repair. (A) CT image showing no abnormal findings. (B) MR angiography image showing almost normal findings. (C) Axial magnetic resonance (MR) diffusion-weighted image showing subtle acute ischemic change involving the left middle cerebral artery (MCA) territory. (D) CT image showing a low-density lesion (arrow) within the right MCA. The density value was approximately −40 to −20 Hounsfield units. (E) MR angiography image showing stenosis of the M2 of the right MCA (arrow). (F) Axial MR diffusion-weighted image showing new ischemic change involving the right MCA territory. (G) T1-weighted MR image showing arterial lumen narrowing at the stenotic lesion (arrow). (H, I) T2-weighted (H) and T2-star (I) MR images showing that the stenotic lesion was markedly hypo-intense (arrow).
vegetation. The patient was diagnosed with cardioembolic cerebral stroke owing to IE; therefore, he was referred to our hospital to undergo MVR for IE.

Four days after the procedure, the patient suddenly presented with consciousness disorder and left hemiplegia. His Glasgow coma scale score was 12 (E3V4M5), and his National Institutes of Health Stroke Scale (NIHSS) score was 22. CT confirmed a very low-density area within the MCA, with a density value of approximately −40 to −20 Hounsfield units (Fig. 1D). MR angiography revealed stenosis of the M2 of the right MCA (Fig. 1E), which corresponded to the low-density area on CT findings. Diffusion-weighted imaging also revealed new ischemic change in the right MCA territory (Fig. 1F). T1-weighted MR images (Fig. 1G) demonstrated arterial lumen narrowing at the stenotic lesion. T2-weighted (Fig. 1H) and T2-star (Fig. 1I) MR images showed that the stenotic lesion was markedly hypo-intense. Angiography demonstrated 62% irregular stenosis at the superior M2 of the right MCA with antegrade peripheral blood flow (Fig. 2A and 2B). The patient’s hemiplegia disappeared during angiography; therefore, we initiated aspirin and high-intensity statin therapy. However, despite conservative therapy, transient mild-to-moderate left hemiplegia appeared every few days during physical therapy for walking, and the resting 123 I-IMP-single photon emission CT revealed moderate perfusion defect in the right MCA territory, which was a 12% decrease compared with contralateral side. Nineteen days after the initial transient ischemic attack, we anastomosed right STA frontal and parietal branches to the M4 of the right MCA, which
supplied the distal segment of the M2 stenotic lesion (Fig. 3A). Indocyanine green video-angiography confirmed the patency of the bypass graft (Fig. 3B).

The patient’s postoperative course was uneventful. CT showed a very low-density area in the MCA, which did not change compared with preoperative findings (Fig. 4A). MR angiography confirmed the patency of the STA–MCA anastomosis (Fig. 4B). T1-weighted (Fig. 4C) and T2-weighted (Fig. 4D) MR images also confirmed that the stenotic lesion was unchanged. The transient left hemiplegia completely disappeared after surgery. The patient was discharged with a modified Rankin scale score of 1, and he did not suffer neurological deterioration during the follow-up period. The latest follow-up CT (Fig. 4E) and MR angiography (Fig. 4F) 18 months after surgery showed that the stenotic lesion was unchanged.

**Discussion**

We reported a case of symptomatic MCA stenosis due to an unknown embolic source after MVR for IE. We performed STA–MCA anastomosis, and the patient was discharged without additional deficits.

The incidence of reinfection of newly implanted valves in patients with active IE is approximately 2%–3%. Multiple cortical branch infarction was the most common lesion, which usually involved the distal MCA territory. Although no studies stated intracranial artery stenosis due to IE, several studies described that septic cerebral embolism caused intracranial artery occlusion. Also, patients’ CT scans showed high density area in the occluding lesions in these studies. In our case, MR images revealed multiple and cortical branch infarction at the distal MCA. However, MR angiography and conventional angiography demonstrated intracranial artery stenosis. CT scans revealed a very low-density area in the stenotic lesion, and the density value was approximately −40 to −20 Hounsfield units. Therefore, the cause of the stenotic lesion was not septic cerebral embolism related to IE. Air bubbles entering the cerebral circulation during cardiac surgery may obstruct blood flow.
reported that air bubbles entering the cerebral arteries during cardiac surgery were alarmingly numerous; however, most bubbles were too small to be harmful.\(^{13}\) Takizawa et al. observed the transfer of air bubbles within the cerebral vessels.\(^{14}\) Generally, CT findings were calibrated using the density values of air (−1000 Hounsfield unit) and pure water (0 Hounsfield unit) as references. In our case, the stenotic lesion was dot-like in appearance and did not change during the 18-month follow-up. The lesion’s density value was approximately −40 to −20 Hounsfield units; therefore, this lesion was not caused by air bubbles. In our case, MR angiography revealed almost normal findings before cardiac surgery; so arteriosclerotic stenosis was also considered absent. Previous studies of cardiac surgery reported intracranial foreign bodies consisting of valve tissue,\(^1\) chordae tendineae,\(^2\) calcified and collagenous tissue,\(^3\) epicardial pacing wires, and catheter tips.\(^4,5\) However, to the best of our knowledge, no studies have reported intracranial artery stenosis because of foreign bodies with unique image findings in the cerebral circulation after cardiac surgery. The unknown embolic source in our case may have been foreign body material, such as Gore-Tex surgical suture, Teflon pledges for suture reinforcement, or fibrin glue, which was used as a hemostatic agent. In our case, the cause was unclear; however, we could investigate the image findings in detail and it may help to consider the etiology and appropriate treatment strategy of similar cases in the future.

Several treatment strategies for intravascular foreign bodies in the cerebral circulation have been reported. A small number of studies reported successful microsurgical embolectomy of the intravascular foreign bodies consisting of detachable balloons and coil fragments in the cerebral circulation.\(^5,16\) Endovascular retrieval techniques were also viable and successful options.\(^7,18\) Fitzgerald et al. represented that the patient was noted to have hemiplegia on the second post-cardiac operative day, and angiography showed occlusion of the ICA distal cavernous and supraclinoid segments.\(^18\) The patient underwent endovascular mechanical thrombectomy with aspiration catheter and obtained the complete revascularization of occluded ICA. The histopathological assessment of the clot demonstrated that there were cells intertwined with the “foreign body” material, which they suspected could potentially be part of a sewing ring of the mechanical valve prosthesis. However, in the present case, we confirmed that the lesion was the proximal M2 of the MCA, which was caused by an unknown embolic source. Endovascular treatment is usually recommended for patients with symptomatic intracranial arteriosclerotic stenosis who are refractory to medical therapies,\(^9\) and angioplasty or stenting may be considered as a treatment option.\(^20,21\) However, in this case, the risk of endovascular treatment with aspiration catheter or stent retriever could not be estimated, because the cause of stenosis of our case was unknown. Although MCA stenosis was not severe, the resting 123 I-IMP single photon emission CT showed moderate perfusion defect and the patient suffered frequent transient ischemic attack. Therefore, we considered that the patient needed revascularization surgery and performed STA–MCA anastomosis, because STA–MCA anastomosis was able to revascularize without touching the stenotic lesion caused by unknown embolic source.

**Conclusions**

We described a case of symptomatic intracranial artery stenosis due to an unknown embolic source, with unique imaging findings following MVR for IE. STA–MCA anastomosis was performed, and the patient had a good clinical course. STA–MCA anastomosis may be a safe and effective treatment strategy for patients with symptomatic intracranial artery stenosis owing to an unknown embolic source, compared with surgical embolectomy or endovascular retrieval techniques.

**Informed Consent**

The patient gave consent to publish the details of this case.

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**Conflicts of Interest Disclosure**

None.

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