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Case Report

Superficial Temporal Artery-middle Cerebral Artery Anastomosis for Ischemic Stroke due to Dissection of the Intracranial Internal Carotid Artery with Middle Cerebral Artery Extension

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A 31-year-old man presented with a sudden-onset headache, right hemiparesis, and dysarthria on day 0 and was diagnosed with acute ischemic stroke due to dissection of the left intracranial internal carotid artery with middle cerebral artery extension. His symptoms progressed despite the institution of treatment, suggesting progression of the dissection. On day 5 after symptom onset, the patient underwent superficial temporal artery-middle cerebral artery anastomosis. No new ischemic stroke event occurred after surgery. Cerebral angiography performed 6 months after surgery showed spontaneous resolution of the dissection. The patient recovered to a modified Rankin Scale score of 2 and was able to return to work. The results of the present case suggest that superficial temporal artery-middle cerebral artery anastomosis is an effective treatment for ischemic stroke due to dissection of the intracranial internal carotid artery with middle cerebral artery extension.

Keywords: STA MCA bypass, internal carotid artery dissection, middle cerebral artery dissection, ischemic stroke

Introduction

Dissection of the intracranial internal carotid artery and/or middle cerebral artery presenting with ischemic stroke is a rare condition, and it can cause ischemic stroke in young individuals. Acute treatment for this condition includes superficial temporal artery-middle cerebral artery anastomosis, conservative therapy, and stent placement; however, there is no established treatment strategy.

Here, we performed superficial temporal artery-middle cerebral artery anastomosis for the peripheral middle cerebral artery in a patient with ischemic stroke due to dissection of the intracranial internal carotid artery with middle cerebral artery extension who exhibited progressive dissection in the acute stage and obtained successful results. We report herein this case.

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Case Presentation

A 31-year-old man without relevant medical history or history of injury developed sudden-onset left temporal pain, followed by right hemiparesis, dysarthria, and disturbance of consciousness on day 0. Because of these symptoms, he was unable to call for help. He was found by his colleague on day 2, and transferred to our hospital on an emergency basis. A neurological examination upon arrival showed his consciousness level was E4V2M6 based on the Glasgow Coma Scale. He demonstrated right hemiparesis, with manual muscle testing (MMT) scores of 4 and 3, as well as motor aphasia. Initial brain computed tomography (CT) 2 days after the onset of symptoms showed infarcts in the left caudate nucleus, insular cortex, lenticular nucleus, and middle cerebral artery territory, and his Alberta Stroke Program Early CT Score was 5/10 (Fig. 1A). Three-dimensional CT angiography (3D-CTA) on day 3 showed stenosis in the left internal carotid artery at its origin from the supraclinoid portion (Fig. 2A). Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) were performed 3 h after the 3D-CTA was performed on day 3. Diffusion-weighted imaging (DWI) showed infarcts in the same regions as those seen on CT (Fig. 1B), and T2*-weighted imaging revealed hemorrhagic infarction (Fig. 1C). Magnetic resonance angiography showed poor visualization of the left internal carotid artery to the middle cerebral artery; moreover, there was a string sign extending between the end of the internal carotid artery and the middle cerebral artery, and a defect area was present in a part of the superior trunk of the left middle cerebral artery (Fig. 2B). T1-weighted images showed a crescent-shaped area of high signal intensity, suggestive of mural thrombosis (Fig. 3A), at the supraclinoid portion of the internal carotid artery on T2-weighted images (Fig. 3B). (CISS) MRI showed a vascular cavity (Fig. 3D), which was confirmed as the false lumen of the dissection using time-of-flight (TOF) MRI (Fig. 3E). Based on clinical and radiological findings, the patient was diagnosed with ischemic stroke due to a dissection extending from the internal carotid artery to the middle cerebral artery.

After admission, the patient was treated by neurologists without administration of antiplatelet medicine because of the hemorrhagic infarction. Although the patient's symptoms improved transiently, 4 days after the onset, his right hemiparesis

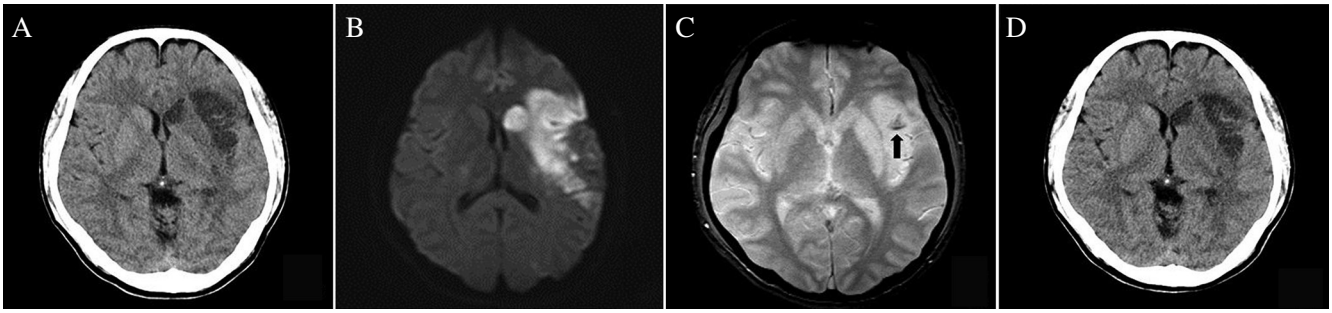

Fig. 1 (A) Initial CT 2 days after onset of symptoms showing infarctions in the region of the left caudate, and lenticular nucleus, insular cortex, anterior and superior middle cerebral artery (MCA) territory. (B) Diffusion-weighted MRI 3 days after onset of symptoms showing hyperintense lesions in the left MCA area and left basal ganglia. (C) T2*-weighted MRI on day 3 revealing bleeding demonstrated by an area of low signal intensity (arrow) in the region of infarction in the frontal lobe. (D) Repeat CT on day 4 not showing expansion of area of infarction.

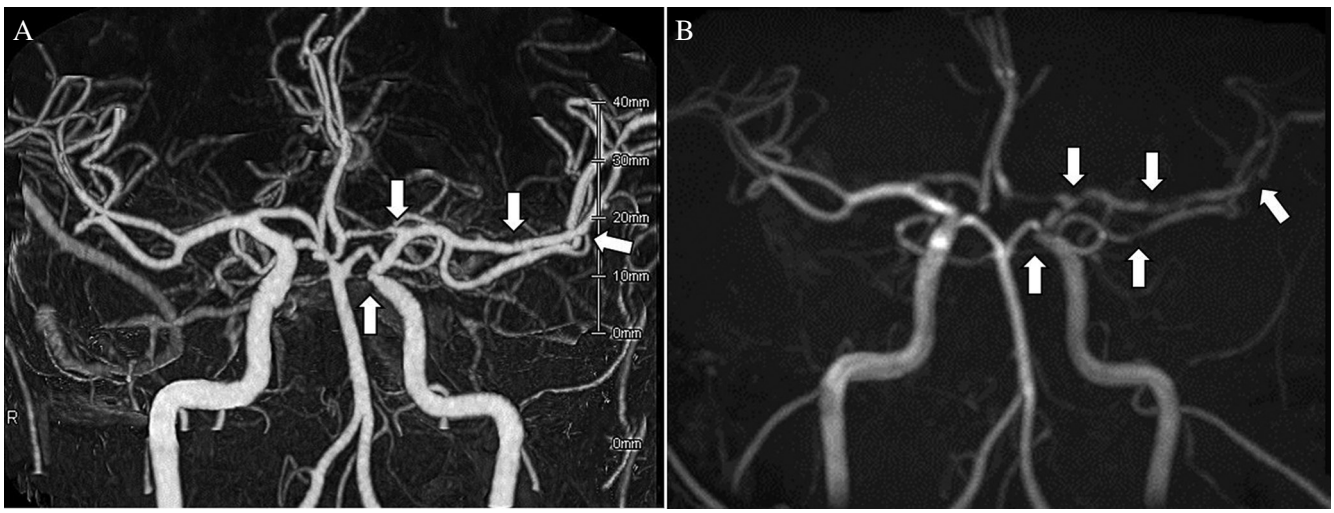

Fig. 2 (A) Three-dimensional computed tomography-angiography (3D-CTA) on day 3 showing the terminal portion of the left internal carotid artery and a superior branch of the left MCA demonstrate slight-to-moderate stenosis compared to its right-sided counterpart (arrow). (B) Magnetic resonance angiogram (MRA) obtained 3 h after the 3D-CTA on day 3, revealing string sign with extension from the distal supraclinoid segment of the left internal carotid artery to the horizontal segment of the MCA (arrow) without obvious aneurysmal dilatation.

deteriorated (MMT score of 2), and his aphasia worsened, suggesting progression of the dissection. A repeat CT on day 4 showed no new evidence of expansion of the areas of infarction and bleeding (Fig. 1D). Following worsening of his symptoms, we were consulted by his neurologists regarding the adaptation in surgery that would be required in this case. We considered that the surrounding cortical ischemia could be salvaged by superficial temporal artery-middle cerebral artery (STA-MCA) anastomosis. On day 5, the patient underwent emergency STA-MCA anastomosis. End-to-side anastomosis was performed between the parietal branch of the left STA and the M4 segment of the middle cerebral artery on the surface of the left side of his brain, and patency of the bypass graft was confirmed using indocyanine green angiography.

Postoperative 3D-CTA on day 10 confirmed progression of the dissection extending from the left internal carotid artery to the middle cerebral artery, as well as patency of the graft to the M4 segment of the dissected peripheral middle cerebral artery (Fig. 4A). Postoperative MRI 18 days after the onset of symptoms showed no evidence of a new ischemic stroke, and MRA confirmed patency of the bypass graft (Fig. 4B).

The patient's postoperative course was favorable, and he was discharged to a rehabilitation hospital approximately a month after symptom onset, following which, he was discharged home and returned to work. Cerebral angiography performed 6 months after onset showed spontaneous resolution of the dissection extending from the left internal carotid artery to the middle cerebral artery, and patency of the graft used for the left STA-MCA anastomosis was confirmed (Figs. 5A and 5B). Follow-up MRA performed 16 months later showed that the MCA dissection had healed, and the flow through the STA-MCA anastomosis was reduced (Fig. 4C). The only residual neurological symptom he demonstrated was decreased grip strength in his right hand. Paralysis of his right lower extremity and dysarthria had resolved and he showed a modified Rankin Scale score of 2.

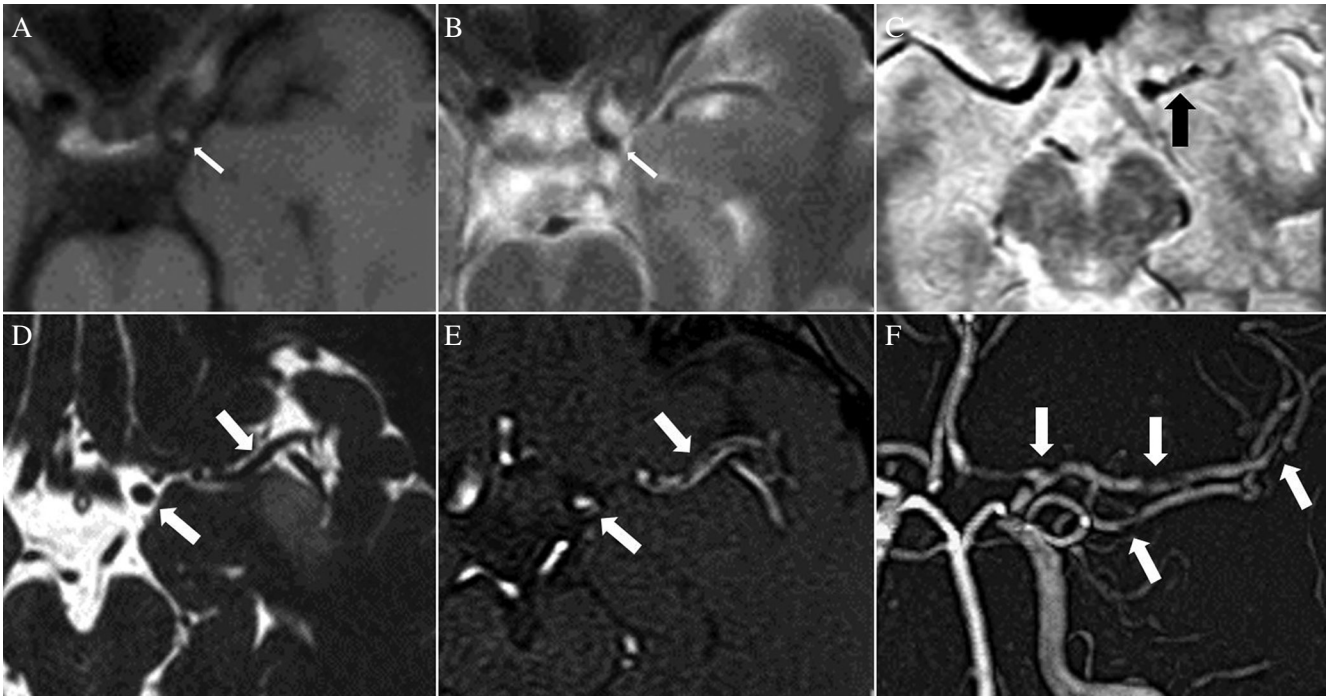

Fig. 3 MRI images obtained 3 h after the 3D-CTA on day 3. (A) T1WI MRI showing high signal crescent sign (arrow) within the wall of the vessel. (B) T2WI MRI showing a vascular cavity (arrow). (C) The outer diameter of the left MCA is seen to have decreased on T2*-weighted imaging due to the presence of a suspected thrombus within the left MCA (arrow). (D) Constructive Interference in Steady State (CISS) MRI showing a vascular cavity (arrow). (E) Time of flight (TOF) MRI showing dilatation of the pseudolumen in the supraclinoid portion of the left internal carotid artery and horizontal portion of the left MCA (arrow). (F) MRA revealing a string sign in the left MCA (arrow).

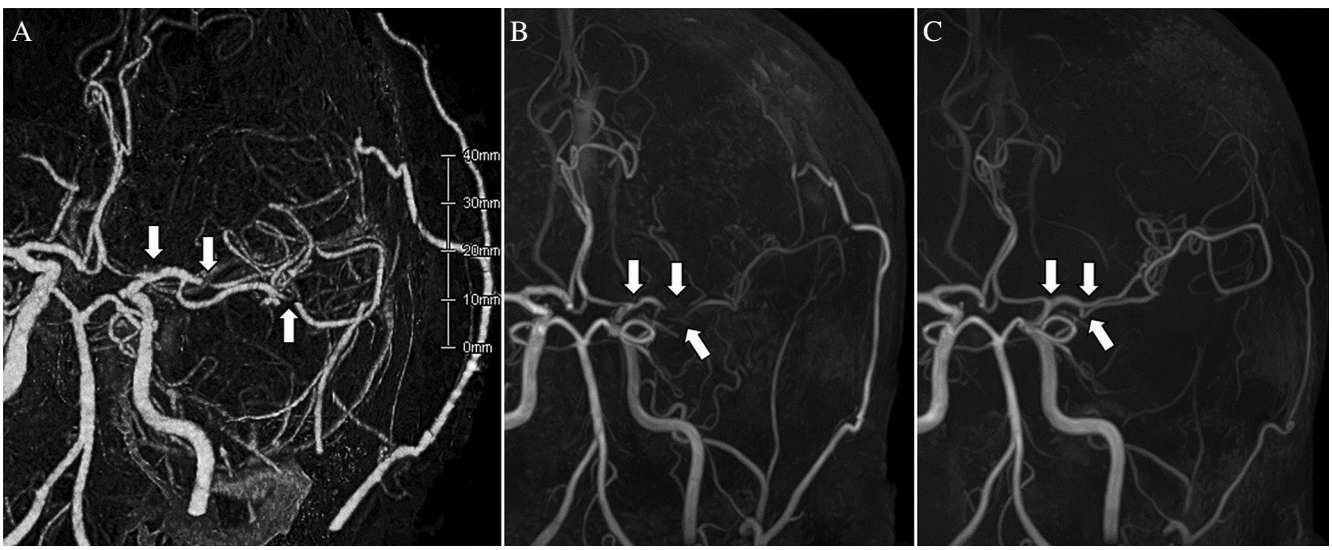

Fig. 4 (A, B) Postoperative 3D-CTA on day 10 (A) and MRA on day 18 (B) revealing clear string and pearl sign in the left M1 portion (arrow). The superficial temporal artery-middle cerebral artery (STA-MCA) anastomosis is seen to be patent. (C) Follow up MRA 16 months later showing recanalization of the MCA (arrow) and flow reduction through the STA-MCA anastomosis.

Discussion

Intracranial internal carotid artery dissection presenting with ischemic stroke is rare and is one of the causes of an ischemic stroke in young individuals. Based on a nationwide survey conducted by Yamaura et al. which included 322 patients with non-traumatic intracranial internal carotid artery dissection, only six patients demonstrated intracranial internal carotid artery dissection presenting with ischemic stroke.[1)] Lin et al. reported that the mean age of 35 patients with dissection of the internal carotid artery with middle cerebral artery extension was 22.2 ± 12.9 years (range 4–60 years), and 33 events involved ischemic stroke.[2)] Studies

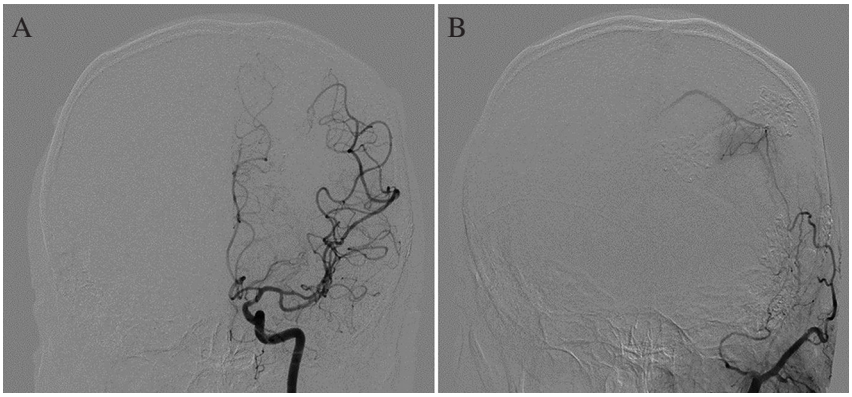

Fig. 5 Follow up angiogram 6 months later. (A) Left internal carotid angiogram showing recanalization of the MCA. (B) Left external carotid angiogram showing good flow through the STA-MCA anastomosis.

Table 1 Summary of STA-MCA bypass cases

Author	Age/Sex	Image	Dissection	Duration	Anticoagulant	Outcome
Kitani et al. (1987)[10]	18/M	Small infarction	Rt. C2, M1, M2	2 days	+	MD
Park-Matsumoto and Tazawa (2000)[11]	32/M	Lacuna infarction	Rt. C2	2 weeks	–	GR
Ogiwara et al. (2005)[12]	22/M	Small infarction	Rt. C1, MCA	1 h	+	GR
Oka et al. (2008)[13]	26/M	Small infarction	Rt. C1	15 days	+	GR
Present case	31/M	Small infarction	Lt. C1, C2, MCA	6 days	–	GR

GR: good recovery, MD: moderately disabled, SD: severely disabled.

pertaining to the treatment and outcomes associated with ischemic stroke due to dissection of the internal carotid artery and/or middle cerebral artery suggest that acute stent placement,[3–5] conservative therapy using antiplatelet drugs,[6–9] and acute STA-MCA anastomosis,[10–13] are management strategies associated with good outcomes.

While some authors recommend acute STA-MCA anastomosis,[10–16] there are only four reported cases of internal carotid artery dissection treated with acute STA-MCA anastomosis.[10–13] Thus, it is difficult to determine the surgical indication. Oka et al. analyzed patients presenting with ischemic stroke following dissection of the intracranial internal carotid artery and classified them into 3 types: (A) Severe initial symptoms with poor outcomes. (B) Mild initial symptoms without exacerbation and with good outcomes. (C) Relatively mild initial symptoms with acute exacerbation and variable outcomes. They reported that bypass surgery is indicated for type C patients.[13] In five cases of internal carotid artery dissection treated with acute STA-MCA anastomosis, including our present case, the mean age was 25.8 years (18–32 years), and all patients were men who would be classified as type C patients based on the classification by Oka et al. (their initial symptoms are relatively mild but are exacerbated in the acute stage) (Table 1). We reasoned that STA-MCA anastomosis for the peripheral branch (M4) of the superior trunk of the middle cerebral artery could improve blood flow into the cortical region that had been spared from infarction, as in our case, with progressive dissection.

Indications of antithrombotic treatment for dissection of the internal carotid artery along its course in the cervical area continue to remain controversial. Based on a review article presented by Schievink, imaging studies suggest that more than 90% of infarcts due to dissection are thromboembolic rather than hemodynamic in etiology. Anticoagulation using intravenous heparin followed by administration of oral warfarin has been recommended for all patients with acute dissections of the carotid or vertebral artery to prevent thromboembolic complications.[17] The Cervical Artery Dissection in Stroke Study (CADISS), a randomized trial, demonstrated that there was no difference between the efficacy of antiplatelet and anticoagulant drugs with respect to preventing stroke and death in patients with symptomatic carotid and vertebral artery dissection. However, stroke was rare in both groups and much rarer than reported in some observational studies.[18] Daou et al. report that the rate of new or recurrent events is similar with use of antiplatelet agents and anticoagulants in treating intracranial and extracranial carotid and vertebral artery dissection. In patients with intracranial dissection, antiplatelet therapy was initiated in 63.1%, 19.7% received anticoagulants, and 14.5% were administered combined treatment.[19] According to Lin's review, anticoagulant therapy was used in 11 of 54 cases (death in four and good recovery in seven cases), antiplatelet therapy was used in 2 cases (death in 1). In addition to medical treatment, surgical therapy was performed in 11 cases (STA-MCA anastomosis in two cases).[2] In the present case, we diagnosed the patient with middle cerebral artery dissection based on evaluation of all presenting symptoms including a sudden-onset headache and his subsequent clinical course and imaging results. Conservative therapy was initially performed, but the patient's symptoms deteriorated; moreover, imaging confirmed progression of the dissection of his left middle

cerebral artery. However, we avoided administration of antithrombotic therapy to avoid the risk of re-bleeding during the perioperative period. In the 206 patients with intracranial arterial dissections followed-up by Mizutani, subarachnoid hemorrhage occurred within 3 days in 81 patients who reported preceding headache. The longest interval within which the subarachnoid hemorrhage occurred was 11 days. Intracranial artery dissections identified within a few days of occurrence may carry the risk of bleeding.[20] In the present case, the patient was discharged to a rehabilitation hospital on day 26, and antiplatelet therapy (aspirin) was initiated following which he showed an uneventful posttreatment course without episode of re-bleeding.

Ischemic stroke caused by cerebrovascular disease is a major cause of mortality and morbidity, and noninvasive *in vivo* assessment of arterial wall integrity is paramount.[21,22] Initial 3D-CTA obtained on day 3 showed the terminal portion of the left internal carotid artery demonstrated slight-to-moderate stenosis compared to its counterpart on the right (Fig. 2A). Postoperative 3D-CTA on day 10 revealed a clear string and pearl sign in the left M1 portion of the left MCA (Fig. 4A). Cerebral angiography is considered the standard method to diagnose cerebral artery dissection. However, in the present case, comparison between CISS and TOF images was useful for evaluation of the dissection. The outer diameter of the left MCA was noted to have decreased on T2*-weighted imaging, owing to a suspected thrombus within the left MCA. Jang et al. report that a, bulging intramural hematoma was seen on T2* corresponded to an eccentric high signal rim on T1-weighted imaging.[23] High-resolution (HR) cross-sectional MRI is also reportedly useful for the diagnosis of cerebral artery dissection.[21,22,24] Choi's report states that HR-MRI is useful in definitively diagnosing an intimal flap and double lumen in intracranial artery dissections, and the presence of an internal hematoma and aneurysmal dilatation can be additional helpful criteria.[21] Such less-invasive diagnostic methods should be explored further.

Recent studies have reported that stent placement for ischemic stroke occurring due to dissection of the intracranial internal carotid artery is associated with good outcomes.[3–5] Endovascular treatment allows immediate recanalization of the occluded artery and resolution of the dissection.[5] However, in the present case, this procedure could not be performed due to nonavailability of an endovascular surgeon. Identifying the true lumen in cases of a large dissection extending from the internal carotid artery to the middle cerebral artery in cases similar to the present case might be difficult.

It should be noted that even if an STA-MCA anastomosis had not been accomplished, this patient would have had a good prognosis. However, the ischemic symptoms in this patient were progressive from the time of their onset, and imaging demonstrated progression of the MCA dissection. Therefore, we performed peripheral bypass grafting for arterial dissection, which salvaged the ischemic cortex that had received reduced cerebral blood flow. Follow-up cerebral angiography performed 6 months later showed spontaneous resolution of the dissection. Ogiwara et al. have reported a case wherein the patient showed a similar spontaneous resolution.[12] A follow-up MRA 16 months later showed recanalization of the middle cerebral artery and flow reduction through the STA-MCA anastomosis, indicating that cerebral blood flow was maintained during the acute phase via the STA-MCA bypass, which however ceased to be important in the chronic stage (Fig. 4C). Thus, we can deduce that a dissection can resolve in the long term and massive brain ischemia can be prevented if bypass grafting can be performed during the time course when ischemic symptoms due to cerebral artery dissection tend to worsen.

Conclusion

The present case indicates that superficial temporal artery-middle cerebral artery anastomosis can contribute to a favorable outcome in patients with progressive ischemic stroke due to dissection of the internal carotid artery with middle cerebral artery extension.

Conflicts of Interest Disclosure

The authors declare that they have no conflicts of interest. All authors who are members of The Japan Neurosurgical Society (JNS) have registered online Self-reported COI Disclosure Statement Forms through the website for JNS members.

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