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# Revascularization with superficial temporal artery–middle cerebral artery anastomosis in spontaneous intracranial internal carotid artery dissection: illustrative case

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**BACKGROUND** Because of ischemic symptoms, intracranial internal carotid artery (IICA) dissection has no established treatment guidelines. The authors report a case of IICA dissection in which an emergency superficial temporal artery–middle cerebral artery (STA-MCA) bypass was performed.

**OBSERVATIONS** A 46-year-old woman presented with a headache and left hemiplegia. Her cortical symptoms appeared on day 10, and an STA-MCA bypass was performed because of cerebral hypoperfusion. Her postoperative patency was good, and cortical symptoms improved. Contrast-enhanced magnetic resonance imaging (MRI) was performed in the acute phase with wall contrast. From day 18, the internal carotid artery delineation improved, and the patient was transferred for rehabilitation without worsening symptoms. A literature review of spontaneous IICA dissection with revascularization procedures was conducted to discuss the indications, timing, treatment modalities, and surgical outcomes.

**LESSONS** The STA-MCA bypass provides supplemental cerebral blood flow and can prevent critical complications. Contrast-enhanced MRI in the acute phase of dissection can show a wall contrast effect and assist in predicting disease progression.

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**KEYWORDS** digital subtraction angiography; internal carotid artery dissection; intracranial carotid artery dissection; superficial temporal artery–middle cerebral artery bypass; cerebral blood flow; single-photon emission computed tomography

Internal carotid artery (ICA) dissection is rare, and intracranial ICA (IICA) dissection is less common than extracranial ICA dissection.[1] IICA dissection has a high frequency of ischemia, but there have been cases of subarachnoid hemorrhage immediately after ischemia resulting from IICA dissection; thus, both ischemia and hemorrhage should be considered.[2] Conservative treatment and revascularization have been reported in previous studies, especially stenting, balloon percutaneous transluminal angioplasty (PTA), and emergency superficial temporal artery–middle cerebral artery (STA-MCA) bypass.[3–5] However, the European Stroke Organisation (ESO) has not established clear treatment guidelines for extracranial and intracranial artery dissection.[6] A nationwide Japanese survey of treatment outcomes for IICA dissection reported a possible benefit of treatment intervention before or immediately after a worsening of stenosis associated with dissection, but no clear conclusion was reached.[7] We report a case of emergency STA-MCA bypass for IICA dissection based on cerebral perfusion assessment and clinical symptoms, as well as the usefulness of cerebral perfusion assessment together with intraoperative findings. We also report a literature review of patients who underwent emergency revascularization for spontaneous IICA dissection.

## Illustrative Case

### History and Examination

A 46-year-old woman presented with hypertension but no history of smoking. Four days before admission, the patient developed transient left hemiplegia, dysarthria, and headache. Magnetic resonance imaging (MRI) was performed at another hospital. Right IICA stenosis (C2 portion) was observed; however, infarction was not noted. On admission to our institution, left hemiplegia, dysarthria, and headache suddenly recurred. Upon arrival at the hospital, her Glasgow Coma Scale score was E4V5M6, and left facial paralysis, dysarthria, and

**ABBREVIATIONS** ASL = arterial spin labeling; CT = computed tomography; DSA = digital subtraction angiography; ESO = European Stroke Organisation; ICA = internal carotid artery; ICG = indocyanine green; IICA = intracranial internal caotid artery; MCA = middle cerebral artery; MMT = manual muscle strength test; MRA = magnetic resonance angiography; MRI = magnetic resonance imaging; NIHSS = National Institutes of Health Stroke Scale; PTA = percutaneous transluminal angioplasty; RA = radial artery; SPECT = single-photon emission computed tomography; STA-MCA = superficial temporal artery–middle cerebral artery.

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left hemiplegia with manual muscle strength test (MMT) grade 4 in the upper and lower limbs were observed. Her National Institutes of Health Stroke Scale (NIHSS) score was 4. Additionally, MRI revealed infarcts in the right basal ganglia and frontal lobe as well as stenosis in the right C2 portion (Fig. 1). She was started on tissue plasminogen activator 2 hours 9 minutes after the onset; however, during administration, paralysis worsened to MMT grade 1 in the upper extremity and MMT grade 2 in the lower extremity, and the NIHSS score worsened to 7. She underwent emergency digital subtraction angiography (DSA), which revealed stenosis and a double-lumen sign at the C1–C2 portion. A diagnosis of IICA dissection was established (Fig. 1). The anterior choroidal artery was visualized in the true lumen of the dissection. As the right ICA was perfused to the periphery of the right middle cerebral artery (MCA) region on right internal carotid arteriography, the patient was conservatively treated without revascularization surgery. Aspirin and clopidogrel were initiated on day 2, with clear consciousness and no symptom worsening. On day 3, MRI and magnetic resonance angiography (MRA) revealed decreased visualization of the right ICA (Fig. 2). Cerebral blood flow was evaluated using arterial spin labeling (ASL), with no difference between the left and right sides. With stable cerebral blood flow and no symptom worsening, we added argatroban to conservative treatment and monitored with imaging. MRI was performed on days 4 and 5, but no changes were observed on the images. On day 5, vessel wall imaging, consisting of a single-slab three-dimensional T1-weighted fast spin echo sequence via enhanced MRI, was performed, and wall contrast was consistent with the location of the stenosis (Fig. 2). On day 9, new neurological symptoms of left hemispatial neglect appeared, and the NIHSS score worsened to 11. Furthermore, MRI revealed a new infarct in the cortical area, an intra-arterial sign on fluid-attenuated inversion recovery, further aggravation of the ICA stenosis on MRA, and decreased cerebral blood flow on the right side detected by ASL (Fig. 2). Therefore, she underwent emergency DSA. Right ICA angiography revealed wall irregularities from C2 to C1 and delayed peripheral contrast of the MCA. Cross-flow through the anterior communicating artery was also worse than that on day 1 on left ICA angiography, with slight development of collateral blood flow from the posterior circulation. Cerebral angiography revealed hypoperfusion in the right cortical region (Fig. 2), necessitating urgent revascularization surgery. Considering the risk of hemorrhagic complications from catheter entry into the false lumen and the risk of infarction from stenting or balloon PTA because the anterior choroidal artery was drawn from the true lumen, emergency STA-MCA bypass was performed rather than endovascular treatment.

## Surgical Procedure

Surgery was performed while the patient was under general anesthesia. An incision was made just above the STA parietal branch, from the front of the right auricle to the midline. The frontal and parietal branches of the STA were harvested, frontotemporal craniotomy was performed, and the dura mater was incised in a U-shape. Opening the sylvian fissure revealed a thrombus in the anterior trunk (M2) and surface vessels of the brain. Indocyanine green (ICG) video angiography revealed hypoperfusion of the cortical region. The parietal branch was anastomosed to the frontal lobe. Pressure measurement was performed using a branch of the STA after anastomosis. The other branch of the STA was connected to the arterial line, and a clip was temporarily placed on the main trunk of the STA to measure MCA pressure before anastomosis. Simultaneously, the radial artery (RA) pressure was 131/61 mm Hg (mean pressure 84 mm Hg), the STA pressure was 106/75 mm Hg (85 mm Hg), and the pre-anastomosis MCA pressure was 35/32 mm Hg (33 mm Hg). We removed the temporal clip and measured the post-anastomosis MCA pressure, which had increased to 90/66 mm Hg (74 mm Hg). The frontal branch was anastomosed to the temporal lobe. ICG video angiography was performed again, which showed blood flow inflow from the STA to the cortex, indicating improved cerebral perfusion (Fig. 3, Video 1).

**VIDEO 1.** Clip showing the surgical procedure. The parietal and frontal branches of the STA were harvested, cut, and heparinized. The temporalis muscle was incised, and a frontotemporal craniotomy was performed. ICG video angiography revealed a peripheral arterial delay. The anastomotic sites of the frontal and temporal lobes were determined, and a rubber sheet was placed

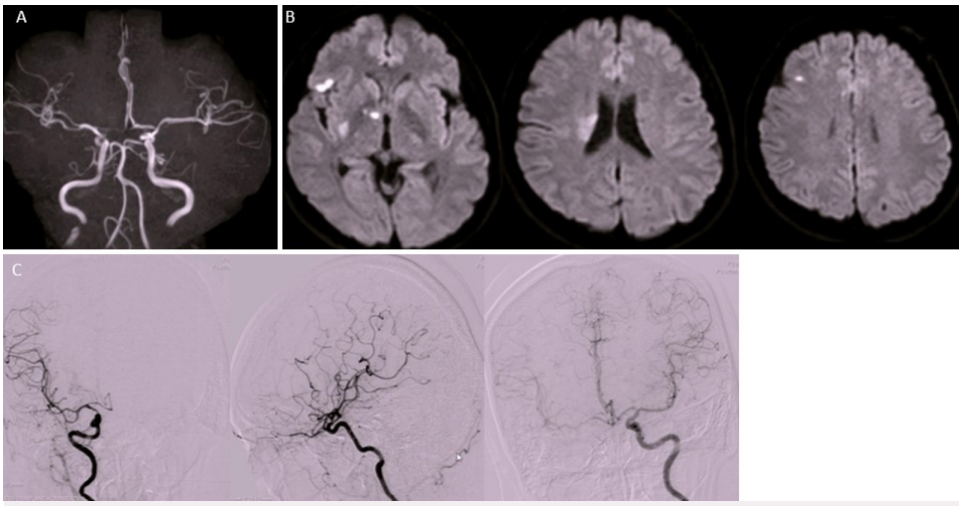


**FIG. 1.** MRA reveals ICA stenosis on the C2 portion on day 1 (**A**). Diffusion-weighted images show infarction in the basal ganglia and frontal lobe on day 1 (**B**). Right ICA angiography reveals stenosis and a double-lumen sign at the C1–C2 portion, but the right ICA is perfused to the periphery of the right MCA region on day 1 (**C**).

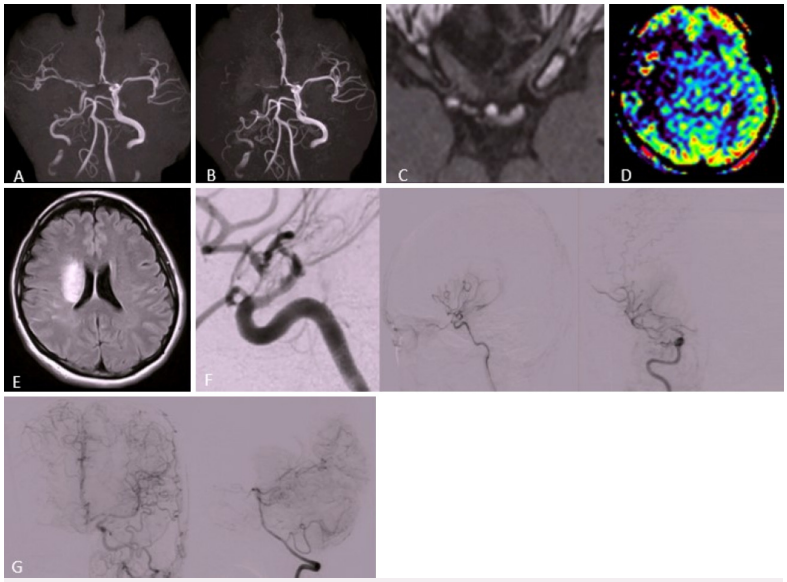


**FIG. 2.** MRA reveals exacerbation of right ICA stenosis from day 3 to day 9 (**A**). Magnetic resonance vessel wall imaging reveals wall contrast consistent with the site of stenosis on day 5 (**B**). MRI reveals decreased blood flow on ASL (**C**). Intra-arterial sign in the MCA region on fluid-attenuated inversion recovery (**D**) and new infarction in the frontal lobe on diffusion-weighted imaging (**E**) on day 9. ICA dissection showed progression on DSA (**F**), with delayed blood flow to the peripheral right MCA (**G**), decreased cross-flow from the contralateral side, and slightly increased blood flow from the posterior circulation.

under the vessels. The STA was trimmed and anastomosed with a 10-0 nylon thread. ICG video angiography was performed again to confirm improved blood flow from the STA to the brain surface. The STA is protected by the temporalis muscle and sutured layer-by-layer to close the head wound. rt = right. Click here to view.

The day after surgery (day 10), there were no new neurological symptoms, and the hemispatial neglect disappeared. MRA revealed good patency of the bypass, improved MCA delineation compared with preoperatively, and only a small cortical watershed infarction in the frontal lobe. Single-photon emission computed tomography (SPECT) revealed increased perfusion in the right MCA. From the day after surgery (day 10), the patient was managed with lacosamide (200 mg) as an anticonvulsant, hypotension, and bed rest with attention to hyperperfusion. From day 12, only clopidogrel was resumed. Repeat MRI studies demonstrated gradual improvement in vascularization from the IICA to the MCA from day 18. DSA was performed on day 33; the C2 to C1 dissection had improved, and right ICA angiography revealed good peripheral delineation of the MCA. Contrast-enhanced MRI was performed on day 39; although there had been a wall contrast effect during dissection, the lumen became visible. On day 65, the patient was alert, had left hemiplegia (upper-extremity MMT grade 1, lower-extremity MMT grade 2), had no apparent cortical symptoms, had an NIHSS score of 8, and was transferred to the hospital for rehabilitation. She was hospitalized for 6 months for rehabilitation; the left hemiplegia remained (upper-extremity MMT grade 3, lower-extremity MMT grade 4), but she was able to walk on her own. MRI was performed again 8 months after onset, and there were no obvious new infarctions or occlusion of bypass vessels.

### Informed Consent

The necessary informed consent was obtained in this study.

## Discussion

### Observations

Given that IICA dissection is a rare condition,[1] a literature review was conducted to investigate the indications for and timing of treatment, patient backgrounds, therapeutic procedures, and outcomes to inform future treatment decisions. This review included cases of non-traumatic IICA dissection angiography, contrast-enhanced computed tomography (CT), or MRA, with evidence of a flap, intramural thrombus, or changes in these findings over time. We reviewed IICA dissections using endovascular or surgical interventions. Cases in which the dissection involved the middle or anterior cerebral artery were excluded. Moreover, reports obtained from the literature search were reviewed, along with this case reported from our hospital. The review was conducted using all English- and Japanese-language studies and case reports published before April 2024, and we searched the PubMed and medical journal databases. Keywords included “intracranial internal carotid artery dissection” and “ischemic stroke,” and reports on clinical studies of this disease were selected.

There were 16 cases of endovascular or surgical intervention for IICA dissection, including our case.[8–19] The average age was 32.9 (12–63) years. The location of ischemia was the basal ganglia in 5 patients, cortical lesions in 3 patients, and both in 6 patients. Surgical treatment included emergency STA-MCA bypass in 2 patients and STA-MCA bypass with ICA trapping in 1 patient. Endovascular treatment included mechanical thrombectomy in 1 patient, balloon PTA in 1 patient, and stenting in 11 patients. In each stenting case, intra-arterial

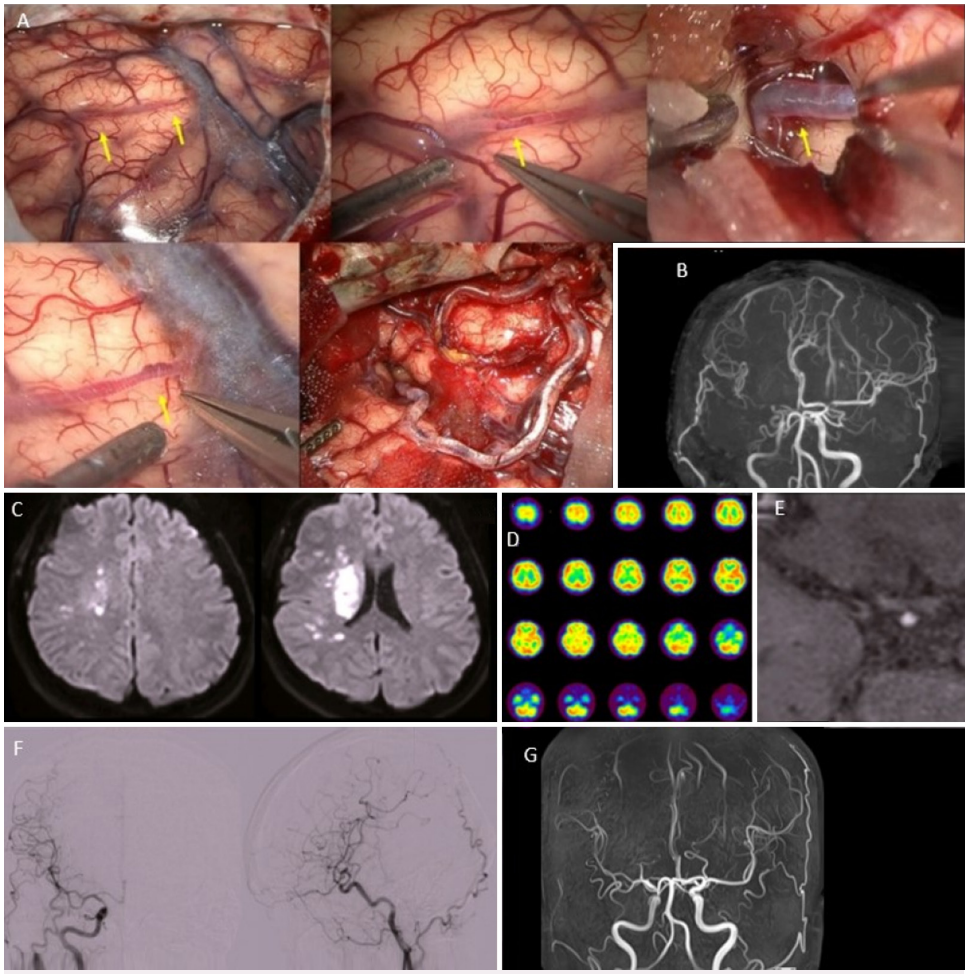


**FIG. 3. A:** There were thrombi (*arrows*) in the cerebral surface and anterior trunk (M2), and the STA was anastomosed to the frontal and temporal lobes. MRA reveals bypass patency (**B**) and no new infarction (**C**), and SPECT reveals increased blood flow (**D**) on the day after surgery. Contrast-enhanced MRI (**E**) shows contrast in the vessel wall. However, the lumen became visible on day 39. ICA angiography (**F**) reveals improved vessel delineation on day 33. MRA (**G**) reveals improved vessel delineation of the ICA 8 months after the onset.

thrombolysis and balloon PTA were performed before stenting. Eight patients were treated on the day of dissection onset, and all were treated with endovascular treatment. Of the patients treated the day following the onset of dissection, 4 received treatment within 1 week, and 4 received treatment after 1 week. Of the 8 patients surgically treated the day following onset, 1 used antiplatelet agents, 6 used both antiplatelet and anticoagulant agents, 1 did not use antithrombotic agents, and none used anticoagulant agents only as preoperative medical therapy. Eleven patients had a re-exacerbation of symptoms before surgery, 4 within 1 day, 4 within 1 week, and 3 after 1 week. Fifteen patients were treated for worsening clinical symptoms or acute re-exacerbation. One patient was treated because stenosis of the ICA worsened on imaging.[16]

There were only 2 surgical complications: left temporal lobe hemorrhage after stent placement on the 11th day of dissection,[9] and cerebral hemorrhage in the left basal ganglia 4 hours after balloon PTA on the day of dissection.[11] The latter patient had a worse prognosis, but there were no other patients with a worse prognosis compared with the preoperative period. No cases of recurrent postoperative cerebral infarction occurred during the mean follow-up of 9.1 (0.5–44) months (Table 1).

In the previous Nationwide Study of Nontraumatic Intracranial Arterial Dissection, IICA dissection was a rare condition, occurring in 10 of 322 patients, with only 6 patients presenting with ischemia.[20] The ESO guidelines state that endovascular revascularization for main artery occlusion due to IICA dissection should be evaluated in terms of risks and benefits, but there are no set treatment guidelines.[6] A nationwide Japanese survey reported that among 28 patients with IICA dissection, 23 (82.1%) were treated with antithrombotic drugs as initial therapy, and half of them showed improvement in symptoms or the prevention of worsening. In contrast, endovascular therapy was performed in 8 patients (28.6%) and STA-MCA bypass in 1 patient (3.6%). Two patients were treated before the aggravation of symptoms or imaging findings. In addition, 7 patients were treated after some

**TABLE 1. Surgical treatment for spontaneous intracranial ICA dissection**

| Authors & Year | Sex | Age (yrs) | Site | Lesion of Ischemia | Tx | Time Btwn Op & Onset (days) | Preop Antithrombotic Therapy | Re-Aggravation of Sx From Onset | Complication | Last mRS Score | FU Period After Op (mos) |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Park-Matsumoto & Tazawa, 2000 [16] | M | 32 | Rt | Basal ganglia | STA-MCA bypass | 21 | — | — | — | 0 | 1 |
| Oka et al., 2008 [15] | M | 26 | Rt | Cortical | STA-MCA bypass+ICA trapping | 15 | Antiplatelet, anticoagulation | Day 15 | — | 0 | ND |
| Surdell et al., 2009 [18] | F | 47 | Rt | Cortical | Stent | 2 | Aspirin, enoxaparin sodium | Day 2 | — | 0 | 8 |
| Lai et al., 2010 [14] | F | 12 | Rt | Basal ganglia & cortical | Intra-arterial thrombolysis, stent | 1 | — | 8 hrs | — | 1 | 6 |
| Kondoh et al., 2012 [13] | M | 17 | Lt | Cortical | Stent | 1 | — | — | — | 1 | 24 |
| Kondoh et al., 2012 [13] | F | 29 | Rt | Basal ganglia & cortical | Stent | 4 | Argatroban, aspirin, clopidogrel, cilostazol, heparin | Day 4 | — | 1 | 44 |
| Kim et al., 2015 [12] | M | 20 | Rt | Basal ganglia & cortical | Stent | 1 | — | 20 hrs | — | 0 | 3 |
| Kim et al., 2015 [12] | M | 40 | Lt | ND | Stent | 1 | — | 20 hrs | — | 1 | 3 |
| Kim et al., 2015 [12] | M | 55 | Lt | ND | Stent | 1 | — | 23 hrs | — | 1 | 3 |
| Shindo et al., 2017 [17] | M | 16 | Rt | Basal ganglia | Stent after PTA | 7 | Clopidogrel, heparin | Day 7 | — | 0 | 3 |
| Kikuchi et al., 2018 [8] | M | 12 | Rt | Basal ganglia | Mechanical thrombectomy | 1 | — | — | — | 1 | 24 |
| Gramegna et al., 2020 [10] | M | 63 | Rt | Basal ganglia | Flow diverter stent (balloon PTA after 3 mos) | 1 | — | — | — | 2 | 3 |
| Ishida et al., 2022 [11] | F | 38 | Lt | Basal ganglia | Balloon PTA | 1 | — | — | Intracranial hemorrhage | 4 | ND |
| Yamada et al., 2022 [19] | M | 20s | Lt | Basal ganglia & cortical | Stent | 3 | Argatroban, aspirin, clopidogrel | Day 3 | — | 2 | 1 |
| Funatsu et al., 2023 [9] | M | 40 | Lt | Basal ganglia & cortical | Stent | 11 | Aspirin | Day 11 | Intracranial hemorrhage (cerebral hyperperfusion syndrome) | 0 | 0.5 |
| Present case | F | 46 | Rt | Basal ganglia & cortical | STA-MCA bypass | 9 | Argatroban, aspirin, clopidogrel | Day 9 | — | 4 | 4 |

FU = follow-up; ND = no data; Sx = symptom; Tx = treatment; — = none.

aggravation. None of these patients experienced postoperative aggravation. The risk of aggravation was reported to be higher for stenotic dissection than for occlusive dissection.[7]

According to one study, one-third of patients undergoing IICA dissection had severe initial symptoms and a poor prognosis (type A), one-third had mild initial symptoms and a good prognosis without acute exacerbation (type B), and one-third had relatively mild disease with subsequent acute exacerbation (type C).[15] In our review, cases in whom treatment intervention was performed on the day of onset were classified as type A, and cases in whom treatment intervention was performed the day following onset were classified as type C. Some patients were treated on the 15th day after onset, and close observation of clinical symptoms and imaging evaluations were important.[15]

After admission, our patient underwent an urgent STA-MCA bypass to supplement blood flow after repeat MRI, including ASL, which revealed cortical symptoms and decreased cerebral blood flow; cerebral angiography revealed hypoperfusion in the right MCA territory due to advanced dissection. In our review, 22 patients were evaluated using perfusion MRI[9] and xenon CT,[16] and all patients, including the patient in the present report, showed reduced perfusion compared with the contralateral side. When our patient was evaluated using ASL, there was no hypoperfusion on admission but there was significant hypoperfusion preoperatively. Therefore, when IICA dissection is diagnosed with ischemic onset, it is useful to evaluate cerebral blood flow, assess for the progression of dissection lesions in the short term if there is no decrease, and evaluate cerebral blood flow again if there is dissection progression in order to determine whether revascularization is necessary. A case of cerebral hemorrhage due to hyperperfusion immediately after intracranial stenting has been previously reported.[9,11] Regarding revascularization procedures for intracranial atherosclerosis, it is important to maintain strict antihypertensive control and evaluate blood flow using SPECT and perfusion CT/MRI.[21]

We suggest surgical intervention based on cerebral perfusion evaluation after a diagnosis of ischemia with IICA dissection. After the diagnosis of IICA dissection, we will evaluate cerebral blood flow, and if there is a decrease in cerebral blood flow, we suggest surgical intervention; however, if there is no decrease in cerebral blood flow, we suggest conservative treatment. If there is worsening of clinical symptoms or imaging findings, cerebral blood flow should be evaluated again to decide on an intervention (Fig. 4).

The revascularization procedures for IICA dissection include STA-MCA bypass and endovascular treatment. Endovascular treatment is relatively quick. Its disadvantages include the risk of dissection of the perforating branch, resulting in cerebral hemorrhage due to recanalization, risk of perforator occlusion due to stent placement,[22] risk of arterial rupture because of microcatheter entry into the false lumen of the dissection,[23] risk of bleeding due to the load of balloon stent dilation,[13] and absence of insurance coverage for stent placement for IICA dissection.[17]

The STA-MCA bypass can take longer than endovascular treatment,[24] and bleeding complications should be considered, as 2 of the 3 cases in this review were performed under antithrombotic medication.[15,16] However, early bypass for acute IICA dissection may preserve cerebral blood flow during the acute phase of dissection, and retrograde blood flow from the bypass graft may stabilize the dissected flap.[5] Because the surgery is not performed near the perforating branch, the risk of perforation of the dissection and obstruction of the perforating branch can be avoided. In this case, endovascular

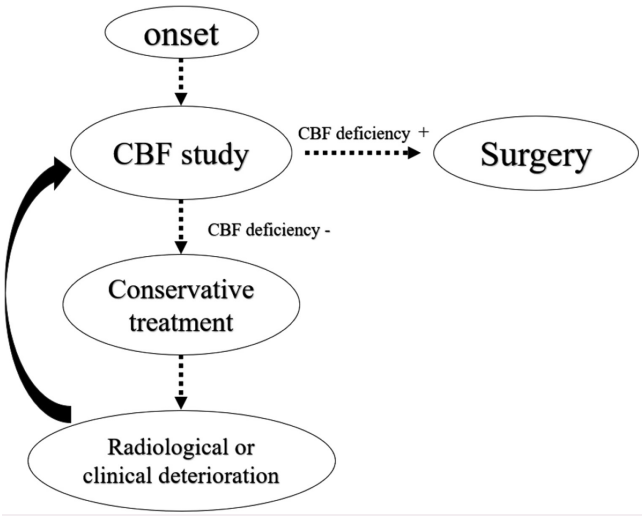


**FIG. 4.** Algorithm for the treatment of IICA dissection. CBF = cerebral blood flow; + = yes; – = no.

treatment with DSA was not performed because the true lumen was not well delineated and there was a high risk of perforation into the false lumen. Occlusion with stenting was considered risky because the anterior choroidal artery was delineated from the true lumen of the dissection, and stenting for stenosis due to intracranial dissection in Japan is an off-label use. Intraoperative findings in this case showed a thrombus in the M2 and peripheral MCA. Possible causes of thrombus formation include embolization of the thrombus formed in the false lumen of the dissection[18] and thrombus formation associated with hypoperfusion before surgery.

Matano et al. reported that the intraoperative MCA pressure measurement was a predictor of increased postoperative perfusion in STA-MCA bypass for atherosclerotic lesions, and in this case, MCA pressure and RA pressure were intraoperatively measured.[25] The MCA/RA ratio before bypass surgery was 39.3%, similar to that reported during bypass surgery for atherosclerotic disease.[25] Anastomosis increased MCA/RA to 88.1%, and one intraoperative STA anastomosis increased MCA pressure to 48.8%. Evaluation of pre-anastomosis MCA pressure, the rate of increase in pressure, and the pre-anastomosis MCA/RA pressure ratio were possible predictors of postoperative hyperperfusion. Cerebral hemorrhage associated with hyperperfusion syndrome is a complication after revascularization surgery.[9] Further studies are required to accumulate more cases of acute revascularization associated with dissection and to further investigate the predictors of hyperperfusion.

All cases in this review that had bleeding due to postoperative hyperperfusion were endovascular, with stent placement or balloon PTA from the IICA to the MCA.[9,11] A rapid increase in cerebral blood flow after revascularization is associated with hyperperfusion, especially if the blood flow acceleration in the infarcted artery is more than 30%–40% compared to the nonaffected side, as measured by transcranial Doppler ultrasonography, or if there is a 70%–80% increase in cerebral blood flow compared to the unaffected side, as measured by perfusion imaging.[26] The two cases of hemorrhage associated with hyperperfusion may have been caused by a sudden increase in cerebral blood flow due to stenting or balloon PTA, and placement of a low-flow STA-MCA bypass may help avoid the risk of hyperperfusion.[27]

All patients received calcium channel blockers as postoperative antihypertensive drugs. However, calcium channel blockers should be avoided in antihypertensive management to prevent hyperperfusion due to their vasodilatory and cerebral blood flow–increasing effects.[28] In our case, we performed vessel wall imaging using contrast-enhanced MRI on days 5 and 39 after the onset. A wall contrast effect was observed during the acute phase of dissection. Saito et al. reported that in vertebral artery dissection, contrast effects on the vessel wall were observed in the order of bleeding, headache, and incidental groups, and these contrast effects accurately reflected the instability of the dissection in the acute phase.[29] Moreover, they reported that dissection contrast might be due to an inflammatory response, physical destruction of the vessel wall by dissection, or the healing process of inflammation. Unlike vertebral arteries with a well-developed vasa vasorum, dissection of the ICA may have a fundamentally different significance of contrast findings in the vessel wall, and further case accumulation will be necessary to clarify the significance of contrast findings in the detection of dissection. Moreover, with respect to the relationship between moyamoya disease and the contrast effect on the vessel wall, Larson et al. reported a correlation between the degree of stenosis and the reduction of the outer wall diameter,[30] and Muraoka et al. reported that the contrast effect on the vessel wall might be related to the appearance of neurological symptoms during follow-up and might help indicate progressive disease.[31] Contrast-enhanced MRI for IICA dissection has not been previously reported, and contrast-enhanced MRI may play an adjunctive role in predicting the course and neurological symptoms of dissection, as MRI on day 5 after the acute onset of dissection showed a strong contrast effect.

### Lessons

Cerebral perfusion studies may be useful in determining the indications for revascularization in IICA dissection with ischemic onset. Endovascular treatments, including STA-MCA bypass and stenting, are the revascularization procedures of choice, and the risks and benefits of each should be considered for treatment. IICA dissection is still a rare disease and should continue to be studied with more cases.

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## Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

## Author Contributions

Conception and design: Nounaka, Matano, Isayama, Murai. Acquisition of data: Nounaka, Matano, Isayama, Murai. Analysis and interpretation of data: Nounaka, Isayama, Ideguchi, Murai. Drafting the article: Nounaka, Isayama, Murai. Critically revising the article: Nounaka, Matano, Isayama, Ideguchi, Murai. Reviewed submitted version of manuscript: Nounaka, Fujita, Isayama, Murai. Approved the final version of the manuscript on behalf of all authors: Nounaka. Statistical analysis: Nounaka, Isayama, Murai. Administrative/technical/material support: Nounaka, Isayama, Murai. Study supervision: Nounaka, Isayama, Murai.

## Supplemental Information

Videos

*Video 1*. https://vimeo.com/1002766351.

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