

Case Report

Middle cerebral artery dissection causing subarachnoid hemorrhage and cerebral infarction: Trapping with high-flow bypass preserving the lenticulostriate artery

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Abstract

Background: Spontaneous intracranial arterial dissection (IAD) is an increasingly important cause of stroke, such as subarachnoid hemorrhage (SAH) and hemodynamic or thromboembolic cerebral ischemia. IAD usually occurs in the posterior circulation, and is relatively rare in the anterior circulation including the middle cerebral artery (MCA). Various surgical and endovascular methods to reduce blood flow in the dissected lesion have been proposed, but no optimum treatment has been established.

Case Description: An 80-year-old woman with dissection in the M1 portion of the MCA manifesting as SAH presented with repeated hemorrhage and cerebral infarction in the area of the inferior trunk of the MCA. High-flow bypass to the MCA was performed and the dissecting lesion was trapped. Prevention of repeated hemorrhage was achieved, and blood flow was preserved to the lenticulostriate artery as well as the MCA area distal to the lesion.

Conclusions: Treatment strategy for IAD of the MCA should be planned for each patient and condition, and surgery should be performed promptly to prevent critical rebleeding given the high recurrence rate. In addition, preventing re-rupture of the IAD, and preserving important perforators around the lesion and blood flow distal to the dissection should be targeted by the treatment strategy.

Key Words: Bypass, intracranial arterial dissection, lenticulostriate artery, middle cerebral artery, trap

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INTRODUCTION

Spontaneous intracranial arterial dissection (IAD) has become increasingly important as a cause of stroke, such as subarachnoid hemorrhage (SAH) and hemodynamic or thromboembolic cerebral ischemia, with the development of diagnostic imaging methods.^[14] Spontaneous IAD mainly occurs in the posterior circulation, and is relatively rare in the anterior circulation including the middle

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cerebral artery (MCA).^[1,4,9,12,13,15,16,18,19] Early repair of the affected vessels is usually recommended in patients with SAH due to the high risk of rebleeding.^[10,14,19] Various surgical and endovascular treatment methods have been proposed, but no consensus has been reached on achieving secure hemostasis and preservation of adequate blood flow to the perforating artery from the dissecting lesion as well as the distal area.

We describe a case of IAD in the MCA treated with trapping of the dissecting lesion and high-flow bypass using a radial artery graft (RAG) to the MCA, which preserved an important perforating artery, the lenticulostriate artery (LSA).

CASE REPORT

History

An 80-year-old woman with a past medical history of hypertension, diabetes mellitus, dyslipidemia, renal failure, angina pectoris, and dementia presented with headache and decline in cognitive function persisting for 3 days. Head computed tomography (CT) at a local hospital disclosed SAH, and she was referred to our hospital at night. On admission, her Glasgow Coma Scale score was 14, and CT demonstrated SAH, with a thick hematoma in the right sylvian cistern [Figure 1a and b]. Digital subtraction angiography (DSA) of right carotid artery revealed severe stenosis and irregular aneurysmal dilatation in the M1 portion of the MCA, and poor perfusion of the MCA territory compared with the anterior and posterior cerebral artery areas [Figure 2a and b]. In addition, the stenotic anterior temporal artery (ATA) originated from the proximal portion of the lesion, and the inferior trunk and LSA branched off just distal to the lesion. We considered that the right MCA dissection had

caused the SAH, and planned surgery to prevent repeated hemorrhage on the next morning.

On the day after admission, CT before surgery identified increased hematoma, indicating repeated hemorrhage, and diffusion-weighted imaging revealed infarctions mainly in the right MCA area [Figure 1c and d]. We assumed the cause of infarction was embolism originating from the MCA dissection. We planned trapping of the dissection and high-flow bypass using the RAG to the distal MCA, intended to prevent repeated hemorrhage from the dissection, stop thrombus moving off from the lesion, and preclude infarction by maintaining adequate blood flow in the MCA area including perforators from the M1 portion.

Operation

Neuroanesthesia was induced under monitoring of somatosensory evoked potentials (SSEPs) of the left extremities and motor evoked potentials (MEPs) of the left upper limb. A curvilinear frontotemporal skin incision was made to expose the right cervical carotid bifurcation, and the superficial temporal artery (STA) was meticulously prepared under the operating microscope. The RAG was harvested concurrently by another neurosurgeon. Frontotemporal craniotomy was performed, and a subzygomatic tunnel was made for the RAG. The sylvian fissure was split under the operating microscope, the hematoma was irrigated and removed carefully, and the M2 and M3 portions of the MCA were exposed. First, a back-up STA-M3 bypass was made distal to the M2 portion for RAG anastomosis. Then, the harvested RAG was gently pulled through the subzygomatic tunnel, that is, between the lateral pterygoid muscle and the temporal muscle from the cranium to the neck through the lateral corridor of the stylohyoid muscle and the posterior belly of the digastric muscle toward the external carotid artery (ECA).^[7] The distal end of the RAG was anastomosed to the M2 (superior trunk) of the MCA, and the proximal end was anastomosed to the ECA. The patency of the anastomosis was confirmed with microvascular Doppler flowmetry. After no change in the SSEPs and MEPs was confirmed, the sylvian fissure was split proximally. The internal carotid artery (ICA) was exposed before the M1 portion of the MCA. The M1 portion was exposed carefully from both sides, proximally and distally. The large dissecting aneurysm with a purplish-red wall was identified arising from the M1 trunk, part of the vessel wall was broken, and thrombus had protruded [Figure 2c]. The dissecting aneurysm of M1 portion was trapped and the arteries arising from both sides of the lesion were preserved, that is the ATA, inferior trunk of the MCA, and LSA. After trapping, microvascular Doppler flowmetry confirmed anterograde flow in the ATA, inferior trunk, and LSA, as well as bypass flow from the STA and RAG. No significant SSEP and MEP changes were observed throughout the procedures [Video 1].

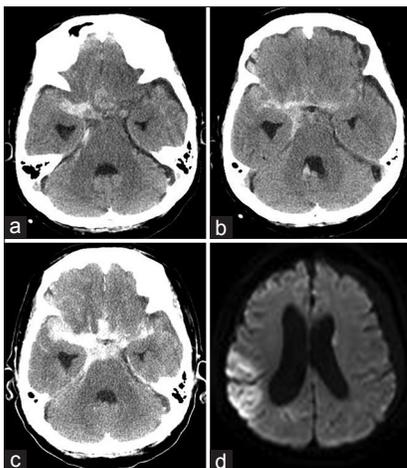


Figure 1: CT scans on admission demonstrating subarachnoid hemorrhage, with a thick hematoma in the right sylvian cistern (a and b). CT scan showing increased hematoma on the day before surgery (c), and diffusion-weighted image revealing infarctions mainly in the right middle cerebral artery area (d)

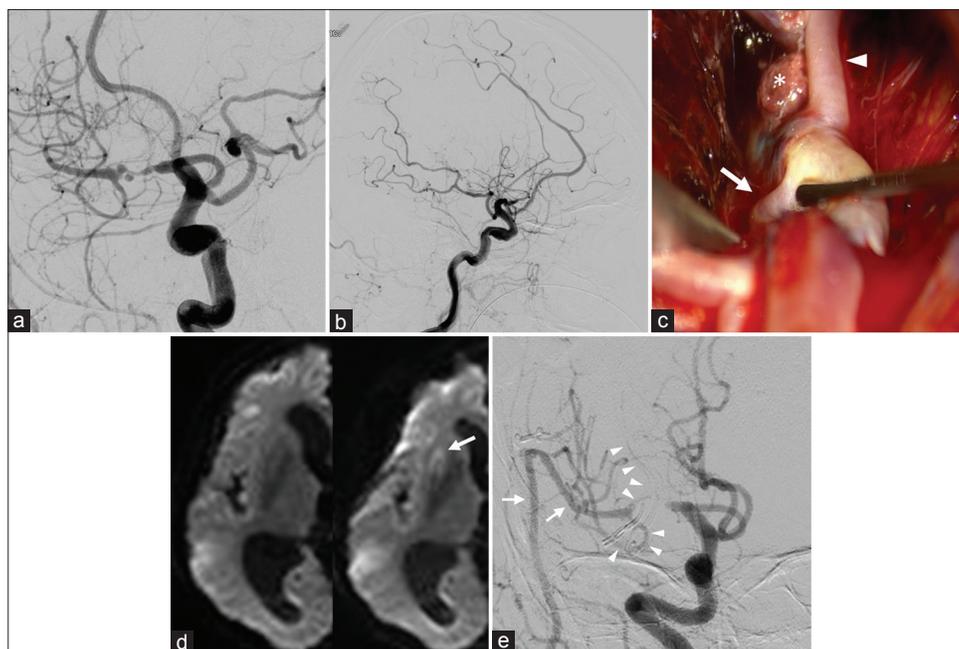


Figure 2: Preoperative DSA, left anterior oblique (a) and lateral (b) projections, showing severe stenosis and irregular aneurysmal dilatation in the M1 portion of the middle cerebral artery (MCA), and poor perfusion of the MCA territory compared with the anterior and posterior cerebral artery areas. Dissecting aneurysm with a purplish-red wall arising from the M1 trunk was found at surgery, just proximal to the lenticulostriate artery (LSA, arrow) and inferior trunk (arrowhead) of the MCA, part of the vessel wall was broken, and thrombus (asterisk) had protruded (c). Postoperative diffusion-weighted image revealing a new small infarction in the anterolateral part of the putamen (d, arrow), and DSA showing good patency of the radial artery graft (arrows), LSA, and inferior trunk of the MCA (arrowheads), and no flow to the dissecting lesion (e)

Postoperative course

Postoperative CT detected no increase in hemorrhage. Postoperative magnetic resonance angiography showed good patency of the bypass, and diffusion-weighted imaging revealed a new small infarction in the anterolateral part of the putamen [Figure 2d]. DSA showed good patency of RAG and no flow to the dissecting lesion 23 days after the operation. The MCA distal to the dissection, including the M2 inferior trunk and LSA, was filled favorably by the high-flow bypass [Figure 2e]. We could not confirm flow to the ATA originating just proximal to the dissection. The patient underwent ventriculoperitoneal shunting for hydrocephalus, and she is gradually recovering with rehabilitation.

DISCUSSION

Patients with IAD causing SAH are usually treated with surgical or endovascular procedures because the mortality for patients with IAD and SAH ranges between 19% and 50%, and the recurrence rate of SAH is reported as high as up to 40%.^[1,10,14] No optimum treatment for IAD has been established, although various treatment methods have been proposed.^[11] Reconstructive methods, such as selective aneurysm sac occlusion with clips or coils and stenting, are intended to preserve the parent artery, whereas deconstructive methods, such as parent artery

occlusion by trapping or proximal occlusion, sacrifice the parent artery. Reconstructive methods are often difficult to perform for IAD given the non-saccular shape of the dissecting aneurysm, whereas deconstructive methods carry the risk of cerebral infarction resulting from insufficient collateral supply, including any perforating artery from the lesion.

Three previous cases have illustrated in detail the surgical treatment of SAH caused by dissection of the M1 portion of the MCA.^[4,9,12] These cases were treated with circumferential wrapping with clip-reinforced cotton gauze, M1 ligation with STA-MCA bypass, and aneurysm body clipping with wrapping. Prevention of repeated SAH was effective in all 3 cases, but cerebral infarction in the MCA area occurred in 2 cases, and temporal lobectomy was needed in one case. These outcomes indicate that prevention of both infarction of the MCA area distal to the dissecting lesion and repeated hemorrhage must be achieved. Preserving the flow of the LSA is also important in the treatment of M1 dissection,^[8] but these previous cases did not discuss the presence of perforating arteries from the M1 portion.

In our present case, the ruptured dissection was located in the M1 portion of the MCA, near the ATA, LSA, and inferior trunk of the MCA, and repeated hemorrhage occurred. In addition, cerebral infarction of the MCA inferior trunk area developed before surgery. We

assumed the cause was thromboembolism originating from the dissection, because a hemodynamic cause would have limited the infarction area to the inferior trunk of the MCA. We had to plan measures for preventing rebleeding and thromboembolic infarction in the early phase given the high risk of repeated hemorrhage and infarction. The optimal treatment method in this case needed to consider three points as follows: how to prevent repeated hemorrhage, how to prevent thromboembolic infarction, and how to avoid cerebral infarction in the perforating arteries from the M1 portion as well as perfusion areas of the distal MCA. First, no flow into the dissection should be allowed to prevent hemorrhage, because this patient suffered repeated hemorrhage after admission. Second, the thrombus in the dissecting lumen should be fixed in place to prevent migration to distal portions of the MCA. Reconstructive methods aiming to maintain the parent artery are not suitable for these needs, whereas deconstructive trapping of the dissecting lesion might be the best option. Third, the appropriate flow out design would be critical to maintain blood flow and to avoid late thrombosis in the nearby important perforator, the LSA,^[3,11,17] as well as ensure adequate perfusion of the vast distal vascular beds of the MCA.

Fortunately in this case, the LSA originated just distal to the dissecting lesion. If the LSA had been involved in the lesion, deconstructive methods such as trapping would have sacrificed the LSA, and reconstructive treatment such as aneurysm sac clipping or stenting would have been necessary. We considered that low-flow bypass was ideal if that would perfuse areas of middle cerebral artery adequately through the term of vasospasm after subarachnoid hemorrhage, because the patient was old and had variable medical complications.

To examine whether the low-flow bypass would replace M1 substantially or not, meticulous collateral evaluation by balloon occlusion test, which would be a predictive and reliable test, should have been performed, but we could not perform due to the unavailability of an endovascular team in our institution. Even with endovascular expertise, we would have avoided balloon occlusion test because of the embolic risk related to the dissecting lesion already causing infarction. In addition, the long-term cerebral ischemic risk would not be predictable. For these reasons, we could not get evidence that low-flow bypass would replace M1 substantially in the future, and we considered that ECA-RAG-M2 bypass with appropriate outflow design might provide sufficient flow to the MCA area and perforating artery of M1 to counteract the abrupt therapeutic M1 occlusion and continue through the term of vasospasm after SAH. Therefore, we planned to form the ECA-RAG-M2 bypass followed by trapping of the dissecting lesion between the inferior trunk of the M2 and ATA, so that the bypass

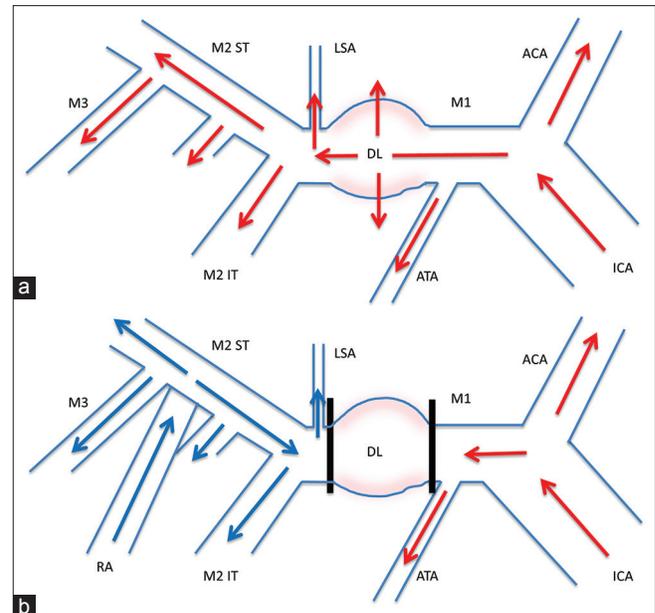


Figure 3: Schematic drawing of the surgery. Preoperative (a) and postoperative (b) hemodynamics are shown. Preoperatively, the internal carotid artery (ICA) supplied all of the anterior cerebral artery (ACA) and middle cerebral artery (MCA) territories, including the dissecting lesion (DL) with risk of rebleeding. With construction of the external carotid artery (ECA)-radial artery (RA)-M2 bypass and trapping of the DL in M1, the ICA would perfuse the ACA and anterior temporal artery (ATA) territories, and the ECA-RA-M2 bypass would supply the MCA territory distal to the lesion including the lenticulostriate artery (LSA), with no blood flow into the dissection. Red arrows: blood flow from ICA, blue arrows: blood flow from ECA-RA-M2 bypass, IT inferior trunk, ST superior trunk

flow would perfuse the MCA territory and LSA, and flow out to the inferior trunk of the M2, with the anterograde flow from the ICA passing to the ATA, and no flow in the dissection [Figure 3].

We made STA-M3 bypass for insurance during temporary clamping of following anastomosis between RAG and M2. Although this supportive anastomosis increases the total time of the operation, it is useful for avoiding major ischemic injury during large-caliber anastomosis because it is impossible to know how long the recipient M2 can endure temporary occlusion during anastomosis.^[5-7] Also, we would like to make the anastomosis between RAG and M2 wide and accurate carefully, because this graft would replace M1 in the future. Actually, duration of temporary occlusion during STA-M3 and RAG-M2 were 15.0 and 31.3 minutes, respectively, so the preceding STA-M3 anastomosis was effective as means of support during RAG-M2 anastomosis.

Surgery was performed successfully as planned, and prevented both repeated hemorrhage and cerebral infarction in the MCA territory distal to the dissection. Flow to the LSA and M2 inferior trunk was supplied from the bypass as shown by postoperative DSA. New infarction

occurred in the anterolateral part of the putamen, which may be the territory of the lateral LSA,^[2] as most of the LSA territories including the posterior limb of the internal capsule were observed with good patency of the LSA on angiography. Perioperative stenosis or temporary occlusion due to brain shift might be responsible for this new infarction, but the true mechanism is unknown given the good patency of the LSA. In contrast, no flow from the ICA to the ATA was seen on angiography. We confirmed weak anterograde flow in the ATA after trapping, but the ATA might have occluded after the operation, given that severe stenosis at the origin of the ATA was observed on preoperative angiography, and the ATA might not be sufficiently large to act as an effective outlet of flow from the ICA. However, no new infarction was seen in the ATA territory, possibly because collateral flow to this area had developed well beforehand due to the severe stenosis of the ATA.

Treatment strategy for IAD of the MCA should be planned for each patient and condition, and surgery should be performed promptly to prevent critical rebleeding after SAH considering the high recurrence rate. In addition, preventing rerupture of the IAD and preserving important perforators around the lesion and blood flow distal to the dissection should be targets of the treatment strategy.

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Conflicts of interest/disclosures

The authors declare that they have no financial or other conflicts of interest in relation to this research and its publication.

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