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Endovascular Coiling for a Ruptured Middle Cerebral Artery-lenticulostriate Artery Bifurcation Aneurysm Suspected to Be a Pseudoaneurysm: A Case Report

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Abstract

Intracranial pseudoaneurysm is a rare entity. Pseudoaneurysm can change its shape dynamically, and the optimal treatment must be selected on the basis of the individual situation. Due to the fragility of the pseudoaneurysm wall, surgical intervention can be difficult. Moreover, a middle cerebral artery-lenticulostriate artery bifurcation aneurysm is uncommon. Surgical intervention carries a risk of ischemic complications in perforating branches. We treated a 43-year-old woman with cardiopulmonary arrest due to a subarachnoid hemorrhage. A right middle cerebral artery-lenticulostriate artery bifurcation aneurysm was detected, which was suspected to be a pseudoaneurysm. The aneurysmal shape changed dynamically, probably because of thrombus formation and resolution. Delayed cerebral vasospasm was also observed. A simple coil embolization was performed initially on day 13 after onset, but early recanalization was observed on day 26, and a second coil embolization was carried out with good obliteration on day 34. The patient subsequently had mild left hemiparesis and mild cognitive dysfunction. After 4 months of rehabilitation, the hemiparesis resolved. Angiography 6 months after onset showed that good obliteration was maintained. For this rare complex condition of a pseudoaneurysm and middle cerebral artery-lenticulostriate artery bifurcation aneurysm, tailored endovascular treatment may be a feasible option.

Keywords: pseudoaneurysm, middle cerebral artery-lenticulostriate artery bifurcation aneurysm, proximal lenticulostriate artery aneurysm, coil embolization, subarachnoid hemorrhage

Introduction

Intracranial pseudoaneurysm is a rare entity, representing approximately 1% of all intracranial aneurysms.[1] Intracranial pseudoaneurysms can cause hemorrhagic stroke and are associated with a high mortality rate of ≥20%.[1] Management of intracranial pseudoaneurysm is controversial because it depends on the cause, location, and shape of the aneurysm. Additionally, a pseudoaneurysm can change its shape dynamically and the optimal treatment must be selected on the basis of the specific situation. An aneurysm located at the bifurcation of the middle cerebral artery (MCA) and the lenticulostriate artery (LSA) is also a rare condition, estimated to account for 4% of MCA aneurysms.[2] In cases in which whether a ruptured aneurysm is true or pseudo is unclear, direct surgery may be the more feasible treatment because the aneurysm can be directly visualized. However, because of the fragility of the pseudoaneurysm wall, direct surgeries are often difficult,[1] and in MCA-LSA aneurysms, a risk for ischemic complications in the perforating branches of the proximal MCA exists.[3] There are some reports on endovascular treatment for proximal MCA aneurysms,[4-6] but the pseudoaneurysm case is rarely cited. In this report, we successfully used coil embolization to treat a ruptured aneurysm that was considered to be a pseudoaneurysm located at the MCA-LSA bifurcation. The patient and the patient's family provided informed consent.

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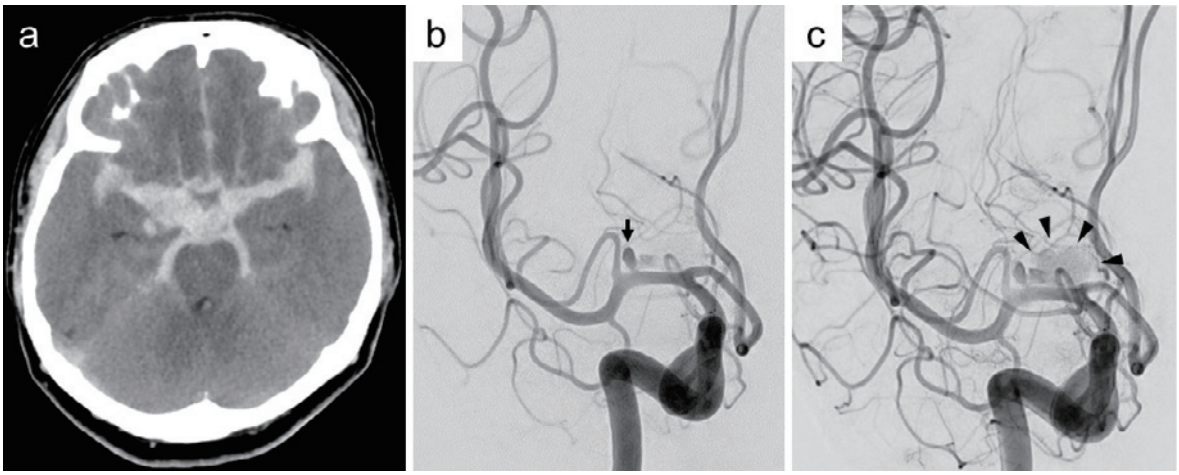

Fig. 1 (a) Noncontrast head computed tomography showing diffuse subarachnoid hemorrhage at onset. (b) Right internal carotid angiogram, anteroposterior, demonstrating a right middle cerebral artery saccular aneurysm (arrow). (c) Right internal carotid angiogram, delayed phase, presenting a pseudoaneurysmal cavity (arrowheads) connected to the right middle cerebral artery aneurysm.

Case Report

In this report, a 43-year-old, right-handed Japanese woman, without any past history including head trauma, presented with loss of consciousness. The patient was brought to the emergency department of our hospital and presented with respiratory arrest and ventricular fibrillation during the initial evaluation. Cardiopulmonary resuscitation was given to the patient, and she was revived approximately 3 minutes later. Brain computer tomography (CT) images displayed a diffuse subarachnoid hemorrhage (Fig. 1a). Due to neurogenic pulmonary edema and takotsubo cardiomyopathy, this case became complicated. Digital subtraction angiography (DSA) showed a 3.6 mm × 1.8 mm saccular aneurysm found at the superior surface of the proximal segment of the right MCA (Fig. 1b). The aneurysm was connected to an irregularly shaped aneurysm-like cavity, which displayed delayed opacification and delayed clearance of contrast material (Fig. 1c). We suspected the aneurysm was a pseudoaneurysm. When treating ruptured intracranial aneurysms, endovascular treatments have an advantage over surgical clipping in outcomes at 1 year after treatment.[7,8] The incidence of symptomatic vasospasm after subarachnoid hemorrhage was less in endovascular treatment than in surgical clipping.[9] Conversely, other reports revealed that the outcome of endovascular treatments and surgical clipping are equivalent, and the clinical outcomes were not influenced by the treatment modality.[10] Nevertheless, these reports were not limited to pseudoaneurysms, and the optimal treatment of intracranial pseudoaneurysms is still controversial.[1] Because the wall of a pseudoaneurysm is thin and fragile, we considered that neck clipping would be difficult. Additionally, some perforating branches arise at the superior surface of the proximal MCA; thus, neck clipping could result in ischemic complications in the right hemisphere. Ischemia of the perforating branches could not be avoided even if bypass surgery was combined. To prevent rerupture of the aneurysm, we selected an endovascular intervention. Deep sedation under general anesthesia was maintained, and on the day after onset, endovascular treatment was attempted. However, the right internal carotid angiogram just before the endovascular treatment revealed no filling of the aneurysm dome, where a tiny protrusion remained at the origin of the aneurysm (Fig. 2a, b). Lateral LSA was detected from the neck of the aneurysm, which could not be detected during the previous DSA. Spontaneous thrombosis in the aneurysmal lumen was indicated; thus, no endovascular intervention was given at that time. CT revealed the ischemic lesion at the right basal ganglia (Fig. 2c), but there was no evidence of rebleeding. To promote the clearance of subarachnoid hematoma, lumbar spinal drainage was carried out. To prevent delayed cerebral vasospasm, selective endothelin A receptor antagonist (clazosentan) was initiated beginning 5 days after onset. At 6 days from onset, general anesthesia and endotracheal intubation were terminated. The patient showed moderate left hemiparesis due to the infarction at the right basal ganglia. A CT angiography at 7 days from onset demonstrated cerebral vasospasm at the bilateral internal carotid artery, bilateral MCA, and right anterior cerebral artery, but there was no evidence of recanalization of the MCA aneurysm (Fig. 3a).

CT angiography 12 days after onset showed recanalization of the MCA aneurysm but did not indicate subsequent rebleeding from the aneurysm. DSA presented a 3 mm × 3 mm saccular, regular, round aneurysm without a cavity, which appeared to be a pseudoaneurysm (Fig. 3b,

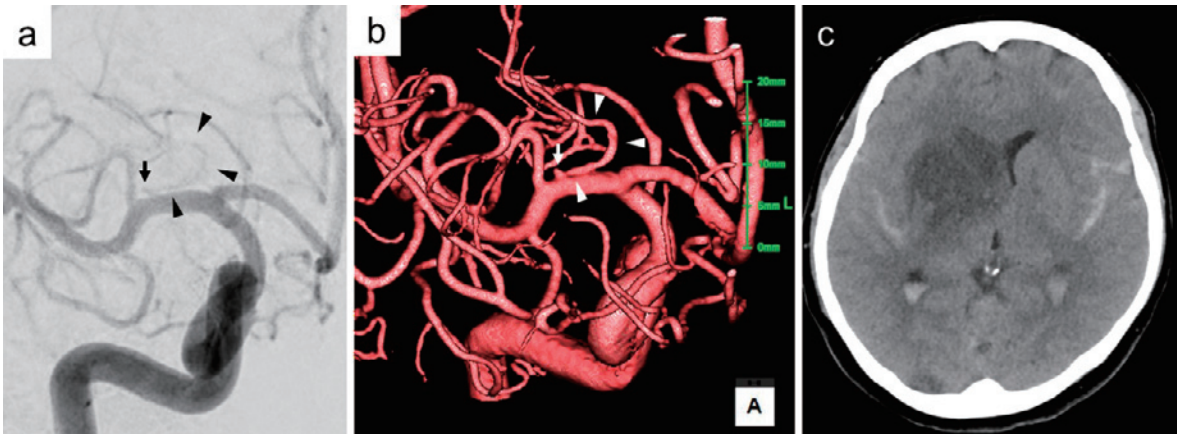

Fig. 2 (a) Right internal carotid angiogram, anteroposterior, and (b) three-dimensional angiogram on the day after onset, which shows the disappearance of the sac of the middle cerebral aneurysm and a tiny protrusion (arrow) and lateral lenticulostriate artery (arrowheads) at the right middle cerebral artery. (c) Noncontrast head computed tomography on the day after onset, which indicates an ischemic or edematous lesion at the right basal ganglia.

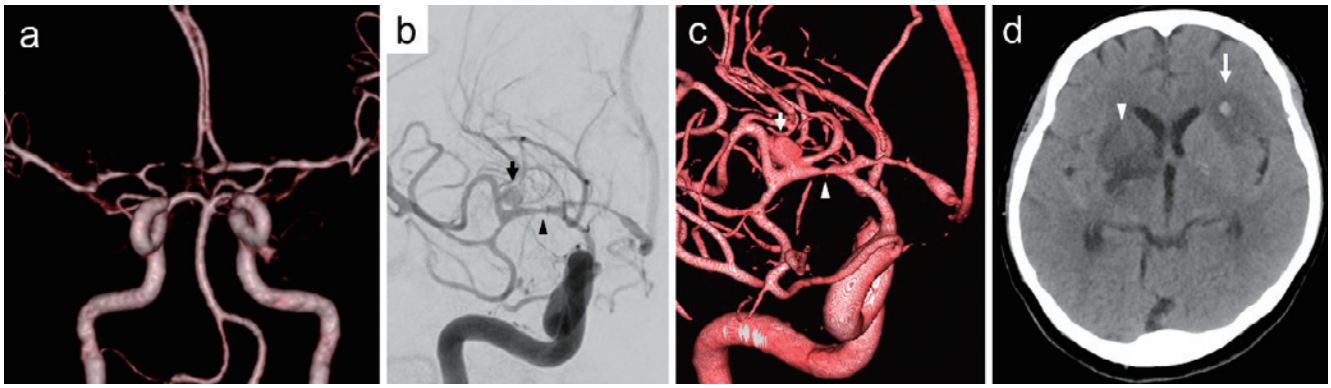

Fig. 3 (a) Three-dimensional computed tomographic angiography at 7 days after onset, revealing cerebral vasospasms at the bilateral internal carotid artery, bilateral middle cerebral artery, and right anterior cerebral artery. (b) Right internal carotid angiogram, anteroposterior, and (c) three-dimensional angiogram showing recurrence of the right middle cerebral artery aneurysm (arrow) and a severe vasospasm at the proximal middle cerebral artery (arrowhead). (d) Noncontrast head computed tomography at 20 days from onset showing a hemorrhagic infarction in the left frontal lobe (arrow). An ischemic lesion in the right basal ganglia remained (arrowhead).

c). The lateral LSA was shown to arise from the aneurysmal neck. Cerebral vasospasm remained at the ipsilateral internal carotid artery, MCA, and anterior cerebral artery. At 13 days after onset, endovascular treatment was conducted under general anesthesia. We considered that an adjunctive technique, including double-catheter, balloon remodeling, or stent-assisted technique, would be difficult because of the vasospasm in the proximal MCA. In the actual endovascular procedure, a balloon-tipped guiding catheter (9 Fr, Optimo; Tokai Medical Products, Aichi, Japan) was placed at the proximal right internal carotid artery to enable the blood flow control if needed, and distal access catheter (3.4 Fr, TACTICS; Technocrat Corporation, Aichi, Japan) was placed at the cavernous portion of the right internal carotid artery. The coaxial system of a microcatheter (Headway Duo; TERUMO, Tokyo, Japan) and a microguidewire (Synchro SELECT; Stryker, Fremont, CA, USA) could pass the vasospasm region, and the microcatheter could be placed into the aneurysm. Three detachable coils were placed into the aneurysm with a simple technique: (1) 3.5 mm × 7.5 cm Galaxy G3 XSFT (Cerenovus, Irvine, CA, USA), (2) 2 mm × 3 cm Target Nano (Stryker), and (3) 1.5 mm × 2 cm Target Nano. As per our assessment, the rupture point of the aneurysm was located at the medial side where the irregularly shaped aneurysm-like cavity was observed at the initial DSA. Thus, the endovascular treatment would be considered successful at least when the medial portion of the aneurysm was embolized. The actual procedure was complete when there was no filling in the whole of the aneurysm and the lateral LSA was preserved (Fig. 4a). During the procedure, intravenous administration of a 3000 U bolus of heparin was employed

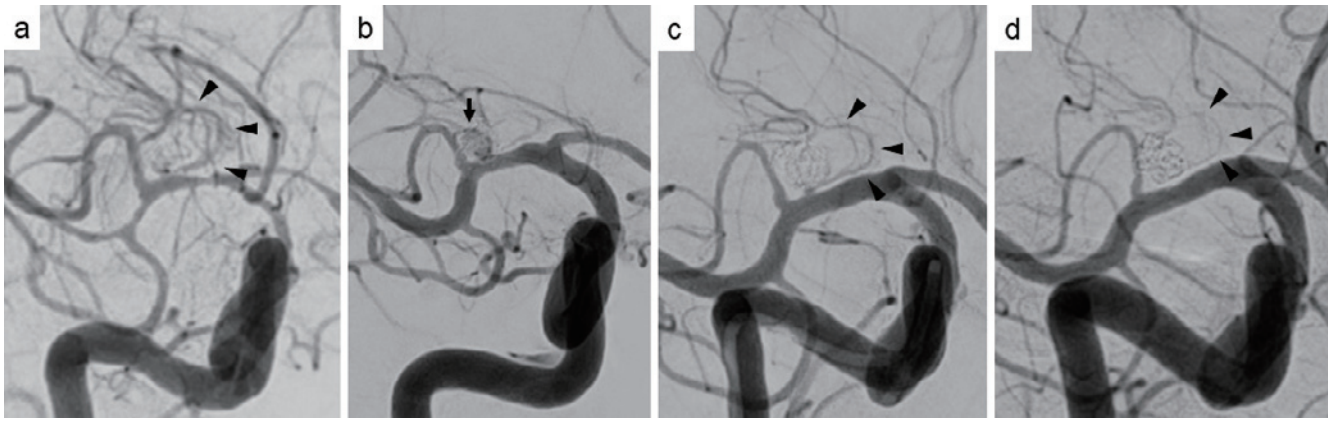

Fig. 4 (a) Postoperative right internal carotid angiogram after the initial endovascular treatment with complete obliteration of the aneurysm and preservation of lateral lenticulostriate artery (arrowheads). (b) Right internal carotid angiogram 13 days after the initial endovascular treatment, which demonstrates recurrence of the right middle cerebral artery aneurysm (arrow) and recovery from cerebral vasospasm in the middle cerebral artery. (c) Postoperative right internal carotid angiogram after the second endovascular treatment, with complete obliteration of the aneurysm and preservation of lateral lenticulostriate artery (arrowheads). (d) Right internal carotid angiogram 6 months after onset showing complete obliteration of the aneurysm and preservation of lateral lenticulostriate artery (arrowheads).

after placement of the first coil. There were no periprocedural complications detected. Head CT at 20 days from onset revealed a hemorrhagic infarction in the left frontal lobe due to vasospasm, which resulted in executive dysfunction (Fig. 3d).

DSA at 26 days from onset showed recurrence of the MCA-LSA aneurysm and coil compaction (Fig. 4b), but CT presented no evidence of rebleeding, and cerebral vasospasm was absent. Endovascular intervention was carried out again at 34 days from onset under general anesthesia. The same catheters and guidewires of the initial endovascular treatment were utilized (9 Fr Optimo, 3.4 Fr TACTICS, Headway Duo, Synchro SELECT). Preoperatively, we considered that stent-assisted coil embolization was feasible to reduce the risk of recurrence. However, good obliteration of the aneurysm was achieved via simple coil embolization, as follows: (1) 2.0 mm × 2.5 cm Target Tetra (Stryker) and (2) and (3) 1.0 mm × 1.0 cm Avenir (Wallaby Medical, Shanghai, China) (Fig. 4c). Accordingly, stent placement was not carried out. During the procedure, a 3000 U bolus of heparin was administered intravenously. There was no periprocedural complication detected. Head CT angiography at 47 days from onset showed no recurrence of the aneurysm. The patient had mild left hemiparesis and mild cognitive dysfunction, scored 3 on the modified Rankin scale, and was transferred to the rehabilitation hospital 48 days after onset. Her hemiparesis resolved within a month, but the cognitive dysfunction remained. She was discharged to home 138 days after onset with a score of 2 on the modified Rankin scale. DSA at 6 months from onset revealed that the good obliteration of MCA-LSA aneurysm was maintained and the LSA branches were preserved (Fig. 4d).

Discussion

In this case, angiography of the aneurysm showed some unusual findings, which include delayed filling, changing shape, and retention of contrast material after the venous phase, suggesting the presence of pseudoaneurysm.[11] However, this was not demonstrated by direct viewing. Usually, pseudoaneurysm is accompanied by head trauma, infection, or connective tissue disease.[1] In our case, the patient had no relevant past history. We also considered the possibility of arterial dissection or blood blister-like aneurysm, but there were no angiographical findings suggestive of arterial dissection (e.g., pearl and string sign, double lumen sign, stenosis, or dilatation). More than 90% of blood blister-like aneurysms are located at nonbranching sites of the internal carotid artery[12]; thus, the blood blister-like aneurysm in our case, occurring at the bifurcation of MCA-LSA, was atypical. A few reports have described pseudoaneurysm formation due to a ruptured cerebral true aneurysm.[11,13-16] According to these reports, a pseudoaneurysm is formed in a blood clot adhering to the rupture point.[15] A massive blood clot around a ruptured true aneurysm contains an extension of the true aneurysm lumen.[13] The shape of the lumen of a pseudoaneurysm can be altered by thrombosis. In our case, most of the aneurysmal lumen disappeared at the second DSA, so we concluded that spontaneous thrombosis had occurred inside the aneurysm. However, we could not distinguish the extent of the true aneurysm from that of the pseudoaneurysm. The infarction in the right basal ganglia might have occurred because of occlusion of the LSA orifice by blood clots in the aneurysmal lumen.

When it is unclear whether a ruptured aneurysm is a

true aneurysm or a pseudoaneurysm, direct surgery may be more a feasible treatment because the aneurysm can be directly visualized. However, direct surgeries are difficult because the wall of a pseudoaneurysm is thin and fragile.[1,13] Additionally, clipping of a superior-wall type aneurysm at the proximal MCA carries a risk of ischemic complications.[3] Neck clipping must avoid perforator occlusion, because some perforating branches, including the LSA, are present on the superior surface of the proximal MCA. A temporary clip may cause infarction of the perforating branches.[3] Even if the neck clipping is successfully carried out, rotation of the clip may cause delayed infarction.[3,17] Bypass and trapping for an MCA-LSA aneurysm cannot secure the blood flow of the LSA.

In several articles, the efficacy of endovascular intervention for proximal MCA aneurysms has been reported.[4,5] Simple coil embolization is the most common option (42%-68%),[4,5] and in some cases, stent-assisted coiling or a balloon remodeling technique is required. In approximately 6% of cases, procedure-related postoperative infarction is reported.[4,5] In our case, we selected simple coil embolization for several reasons, as follows: First, the aneurysmal neck was narrow. Second, the placement of multiple catheters would occlude the blood flow of the MCA in the initial treatment, because the proximal MCA had severe vasospasm. Third, stent placement in the acute phase of subarachnoid hemorrhage presents a risk of thrombotic complications. A case report of a ruptured MCA-LSA aneurysm treated via simple coil embolization mentioned that endovascular embolization was technically feasible for a ruptured proximal LSA aneurysm and that sparing of the LSA was crucial to avoid postoperative infarctions.[18]

In our case, early recurrence of the aneurysm was observed. We presume that the coil migrated into the thrombus of the pseudoaneurysm lumen; regretfully, we could not prove the existence of the thrombus in the pseudoaneurysm lumen on magnetic resonance imaging. Reportedly, 61.9% of partially thrombosed intracranial aneurysms treated with intraluminal embolization presented major recurrence, the main cause of which was coil migration or compaction.[19] To prevent aneurysmal recurrence and rebleeding due to coil migration, close follow up is important. If the aneurysm recurs, adjunctive techniques such as stent-assisted coiling and flow-diverting stent can be considered.

In this report, we present a case of a ruptured MCA-LSA aneurysm that we suspected to be a pseudoaneurysm. Tailored endovascular treatment based on the individual situation may be a feasible option for treating pseudoaneurysm. Employing an endovascular technique in consideration of the preservation of perforating branches may yield favorable outcomes for MCA-LSA aneurysm. Considering that recurrence of the pseudoaneurysm can occur even in the early postoperative period after coil embolization, close follow up is crucial.

Conflicts of Interest Disclosure

The authors have no conflicts of interest to declare.

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