Case Report

Partially thrombosed middle cerebral artery-lenticulostriate artery aneurysm with native radiological examinations suggesting proximal lenticulostriate artery aneurysm: A case report

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ABSTRACT

Background: Preservation of the lenticulostriate artery (LSA) is crucial. LSAs usually cannot be spared with LSA aneurysms, when surgical clipping/excision or endovascular embolization of the LSA itself is performed. On the other hand, the LSA should be separated and preserved for proximal middle cerebral artery (M1)-LSA aneurysms.

Case Description: We report a case of M1-LSA aneurysm with native radiological examinations suggesting LSA aneurysm. The highlight of this unusual case was that during surgery, the aneurysm orifice was almost covered with thrombus and blood flow in an aneurysm that appeared separate from M1. Partial thrombectomy-clip reconstruction was performed, and M1 and LSAs were well preserved.

Conclusion: Even with currently developed radiological modalities, thrombosed intracranial aneurysms may be misdiagnosed, depending on intraluminal flow conditions. Intraoperative findings from craniotomy sometimes contribute to a better understanding of the pathophysiology and decisions on appropriate treatment strategy.

Keywords: Intracranial aneurysm, Lenticulostriate artery aneurysm, Middle cerebral artery aneurysm, Thrombosed intracranial aneurysm

INTRODUCTION

Preservation of the lenticulostriate artery (LSA) is crucial to the treatment of intracranial lesions such as intracranial aneurysms and tumors. LSAs usually cannot be spared with LSA aneurysms, when surgical clipping/excision or endovascular embolization of the LSA itself is performed to cure LSA aneurysms.

On the other hand, the LSA should be separated and preserved for proximal middle cerebral artery (M1)-LSA aneurysms. We present herein an exceedingly rare case of M1-LSA aneurysm with native radiological examinations suggesting proximal LSA aneurysm.
CASE REPORT

History and examination

A 23-year-old woman presented with a 1-day history of the right hemiparesis before admission to our clinic. She was otherwise neurologically intact with normal mental status, sensation, reflexes, and coordination. No hematological or electrolyte abnormalities, underlying pathologies such as vasculitis, or autoimmune diseases such as systemic lupus erythematosus were identified.

Initial magnetic resonance imaging (MRI) and computed tomography (CT) of the head demonstrated acute infarction of the left basal ganglia, but no subarachnoid hemorrhage [Figure 1a and b]. A small, high-density spot on CT and high-intensity spot on T1-weighted MRI was shown beside the infarction [Figure 1b and c]. Subsequent MR angiography and CT angiography (CTA) demonstrated what appeared to be a 2.3-mm fusiform aneurysm with a left LSA origin [Figure 1d and e].

The cerebral infarction of the left basal ganglia was not aggravated on repeat CT or MRI, and neurological symptoms resolved within 2 weeks. However, DSA revealed filling of 7.8 mm of the fusiform aneurysm near the left M1 segment, with the body of the aneurysm separate from M1 [Figure 1f]. The aneurysm was diagnosed as proximal LSA aneurysm. Because the size of the aneurysm was increasing and the risk of rupture was increasing, we decided to perform surgical clipping of the proximal LSA itself to collapse inflow into the aneurysm.

Operation

Left pterional craniotomy was performed. Intraoperatively, the orifice of the aneurysm was identified not on the proximal LSA but on M1. The aneurysm was brown in color and was diagnosed as a thrombotic aneurysm [Figure 2a and b]. The orifice of the aneurysm was almost entirely covered with thrombus, and the M1-LSA aneurysm was found to have been mistakenly interpreted as LSA aneurysm on preoperative DSA. Some small LSAs and arteries supplying the surrounding brain were seen to be adherent to the surface of the thrombosed aneurysm. Simple neck clipping was unsuitable, because M1 was collapsed with thrombus after neck clipping, so partial thrombectomy was performed with temporary clips on the parent artery without heparin administration [Figure 2c and d]. After thrombectomy, neck clipping with

Figure 1: Preoperative radiological examinations. (a) Initial axial-view diffusion-weighted magnetic resonance imaging (MRI) shows acute stroke within the territory of the lenticulostriate artery. (b and c) Initial axial-view computed tomography (CT) shows a high-density spot (b), and axial-view T1-weighted MRI shows a high-intensity spot at the base of the anterior perforated substance (c). (d and e) Initial multiplanar reconstruction MR angiography (slab: 15.0 mm thickness) (d) and CT angiography (e) on coronal view show an aneurysm separate from the proximal middle cerebral artery. (f) Digital subtraction angiography at the 2-week follow-up shows the aneurysm has grown.
a fenestrated clip was performed, preserving the parent artery and LSAs, but not preserving one of the small perforator's adherents to the aneurysm [Figure 2e and f].

**Postoperative course**

Postoperatively, right hemiparesis re-aggravated, and a small new infarction was detected on MRI in the basal ganglia beside the previous infarction [Figure 3a]. The aneurysm was not filled, and the left M1 and LSAs were well preserved on DSA [Figure 3b and c]. She was discharged to a rehabilitation hospital with a modified Rankin Scale (mRS) Grade 2, and then discharged home with mRS Grade 0 at 3 months postoperatively.

**DISCUSSION**

LSA aneurysms causing cerebral ischemia are exceedingly rare. Lama et al. presented 42 cases of LSA aneurysm, including one case of their own, identified in a review of the literature since 1960,[4] and Chaohui et al. identified 45 cases of LSA aneurysm, including three cases of their own, from the literature since 1987.[1] Almost all cases from these reviews were ruptured aneurysms (intracerebral hemorrhage, intraventricular hemorrhage, and subarachnoid hemorrhage), with only two aneurysms associated with ischemic stroke. On the other hand, M1-LSA aneurysms are well known, and Elsharkawy et al. reported the incidence of M1-LSA aneurysms as 13% among their 1309 consecutive modern cases of MCA aneurysms.[3]

The key strategy for LSA aneurysms and for M1-LSA aneurysms is quite different. In general, preserving the LSA is impossible because the LSA is the parent artery of the LSA aneurysm. Surgical clipping/excision of the LSA itself has been allowed and performed, and endovascular embolization of the LSA has recently been increasing.[1,4] On the other hand, complications of the LSA should be avoided with M1-LSA aneurysms. To preserve the LSA, both surgical clipping and endovascular embolization with/without stenting are performed for M1-LSA aneurysms. Diagnosing LSA aneurysm or M1-LSA aneurysm is therefore crucially important.

Lawton et al. classified their 68 thrombotic aneurysms into six types by organized intraluminal thrombus and solid mass.[5] Our aneurysm case, with thrombus extending to the aneurysm neck, may be classified as “concentric type (17.25% of Lawton’s series),” but exceptional situations like our aneurysm are not mentioned. Even though the high-intensity spot on T1-weighted MRI was interpreted as thrombus in the aneurysm, the precise location of the thrombus could not be determined before the operation because its small size. In addition, although preoperative CTA and DSA demonstrated the aneurysm separate from the M1, we might have thought

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**Figure 2:** Intraoperative microscope views. (a-f) A left frontotemporal approach shows the thrombosed aneurysm (asterisk) and perforators (triangle) of the left proximal middle cerebral artery (M1) (a), some small lenticulostriate artery (LSAs) and arteries supplying the surrounding brain (arrows) adherent to the surface of the aneurysm (b), collapsed M1 after neck clipping with fenestrated clip without thrombectomy (c), thrombectomy of the aneurysm with trapping of M1 (d), collapsed small LSA adherent to the aneurysm (arrow) (e), and preserved M1 and LSA (triangle) flow after thrombectomy-clip reconstruction with indocyanine green videoangiography (f). Fr, frontal lobe; Tm, temporal lobe.
of the aneurysm causing the ischemic stroke as M1-LSA aneurysm rather than LSA aneurysm in terms of frequency.

At present, although recently developed radiological modalities are often quite useful and worthwhile to diagnose conditions of thrombosed intracerebral aneurysms, craniotomy can help reveal the truth. Darkwah Oppong et al. reported a ruptured, masked aneurysm subsequent to thrombosis of the associated vessel, and intraoperative findings were useful for deciding the treatment strategy.[2]

In our case, endovascular treatment may have been difficult because the LSA was originally a thin feature, but craniotomy was a more appropriate option from the perspective of protecting LSA. In general, management of thrombotic aneurysm remains challenging in the field of neurosurgery, especially for aneurysms located in the LSA. Intraoperative infarction sometimes occurs with temporary ischemia of the parent artery (trapping or vasospasm) or translocation of aneurysmal thrombus, even in the absence of flow reduction. In our case, adhesion between small perforators and the aneurysm was too tough to separate and allow preservation of perforator flow [Figure 2e].

**CONCLUSION**

Partially thrombosed aneurysm may be misdiagnosed depending on intraluminal flow conditions. This report reminds us that intraoperative exploration by craniotomy may contribute to a better understanding of the pathophysiology and selection of an appropriate treatment strategy.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent.

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**Conflicts of interest**

There are no conflicts of interest.

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