



Case Report

A rare intracranial fusiform thrombosed aneurysm of the distal middle cerebral artery: A case report

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ABSTRACT

Background: Intracranial aneurysms of the distal middle cerebral artery are rare, and most etiologies are infection or dissection. We present an extremely rare intracranial fusiform thrombosed aneurysm of the distal middle cerebral artery with histopathological confirmation of a pseudoaneurysm.

Case Description: Our patient, a 68-year-old female, was previously healthy and had no history of infection or trauma. A fusiform thrombosed aneurysm of the distal middle cerebral artery was detected incidentally. The patient was treated successfully with trapping and resection of the aneurysm followed by superficial temporal artery to middle cerebral artery anastomosis. Xanthochromic and hypertrophic arachnoid membranes around the aneurysm were noticed, and a thrombus was detected inside the lesion. The aneurysmal wall had hyalinized connective tissue incompletely surrounded with intima, with no media or adventitia. Pathologically, it was a pseudoaneurysm.

Conclusion: We report an extremely rare case of a pseudoaneurysm of the distal middle cerebral artery. We discuss the etiology of the lesion, with a literature review, and propose that the appearance and increase of the pseudoaneurysm was followed by microbleed of an aneurysm unrelated to the branching zone.

Keywords: Distal middle cerebral artery, Intracranial aneurysm, Pseudoaneurysm

INTRODUCTION

Intracranial aneurysms of the distal middle cerebral artery are rare, and most etiologies are infection or dissection.^[2-4,6-8,10,13-15,17,18] Distal middle cerebral artery aneurysms with histopathological analysis are very rare; only 17 cases, including our case, have been reported.^[1-4,6-8,10,12-18] Here, we present an extremely rare intracranial fusiform thrombosed aneurysm of the distal middle cerebral artery with histopathological confirmation of a pseudoaneurysm.

CASE REPORT

Our patient, a 68-year-old female, was previously healthy and had no history of infection or trauma. She had no symptom and no neurological deficit. A brain checkup using magnetic resonance imaging (MRI) showed an abnormal lesion (15 mm in diameter) of the left parietal lobe surface [Figure 1a and b]. Eight years earlier, a brain checkup using MRI had demonstrated slight arterial dilatation of the same portion retrospectively [Figure 1c]. Cerebral angiography revealed a fusiform aneurysm

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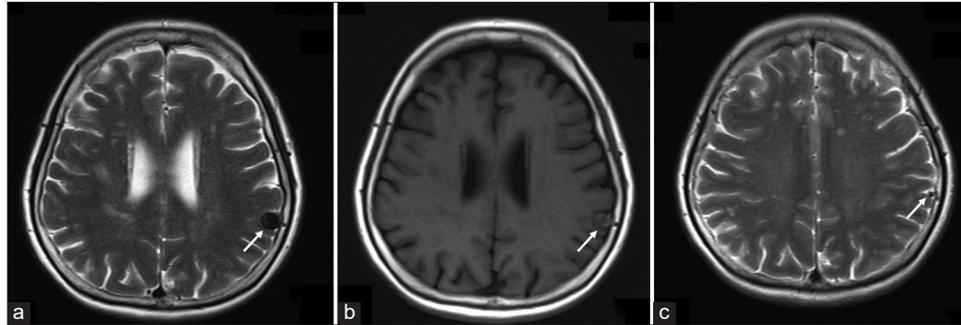


Figure 1: T1-weighted magnetic resonance imaging (MRI) (a) and T2-weighted MRI (b) shows an abnormal lesion of the left parietal lobe surface (arrows). T2-weighted MRI performed eight years earlier (c) demonstrates slight arterial dilatation of the M4 portion of the left middle cerebral artery (arrow).

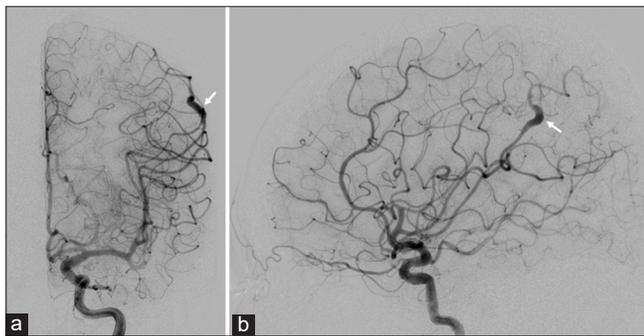


Figure 2: Left carotid angiograms (a: anteroposterior view, b: lateral view) shows a fusiform aneurysm of the M4 portion of the left middle cerebral artery (arrows).

of the M4 portion of the left middle cerebral artery (angular artery) [Figure 2a and b]. We diagnosed a fusiform thrombosed aneurysm of the distal middle cerebral artery. The patient had no history of headaches or high fever, and the abnormal finding was not detected in the laboratory data or the echocardiogram. The patient and her family wished her to undergo surgery, and she gave her informed consent.

Left front-parietal craniotomy and superficial temporal artery to middle cerebral artery anastomosis were performed, and the aneurysm was trapped and resected. The aneurysm was confirmed from the brain surface, and xanthochromic and hypertrophic arachnoid membranes and significant arachnoid adhesion around the aneurysm were noticed [Figure 3a]. Mural thrombus was identified on the wall of the aneurysm [Figure 3b]. Histologically the wall of the aneurysm was composed of hyalinized connective tissue incompletely surrounded with intima, of which internal elastic lamina was disrupted. There was no media or adventitia in the aneurysmal wall [Figure 3c and d]. Pathological diagnosis was a pseudoaneurysm. The postoperative course was uneventful, without transient amnesic aphasia. The patient was discharged two weeks after the surgery, with no neurological deficit.

DISCUSSION

Intracranial aneurysms of the distal middle cerebral artery are rare, and most etiologies are infection or dissection.^[2-4,6-8,10,13-15,17,18] Rinne *et al.* reported anatomic and clinical features of 561 patients with 690 middle cerebral artery aneurysms, including only 25 cases (3.6%) of distal middle cerebral artery aneurysms.^[11] Only 17 cases of distal middle cerebral artery aneurysms with histopathological analysis have been reported including our case [Table 1].^[1-4,6-8,10,12-18] Of those 17 cases, only three, including our cases, were incidental. The histopathological diagnoses were: a dissecting aneurysm in nine cases, a pseudoaneurysm in five cases, and a saccular aneurysm in three cases. Among the five pseudoaneurysms, four (the exception being our case) were caused by infection. The etiology of the pseudoaneurysm is unclear in our case.

Intracranial pseudoaneurysms are rare, and represent about 1% of all intracranial aneurysms, with an associated mortality of 20% or higher.^[19] A pseudoaneurysm is the product of a damaged vessel wall, resulting in an encapsulated hematoma in communication with the ruptured artery. The most common cause of pseudoaneurysm is trauma. Other causes are infection, iatrogenic events, radiation, and connective tissue disease, sometimes, they occur spontaneously.^[19] Furthermore, an aneurysmal rupture may cause a pseudoaneurysm.^[5]

Mizutani *et al.* proposed classification of nonatherosclerotic aneurysms unrelated to the branching zones.^[9] They were classified into four types, based on the lesional patterns of the internal elastic lamina and the state of intima: classic dissecting aneurysm (Type 1), segmental ectasia (Type 2), dolichoectatic dissecting aneurysm (Type 3), and saccular aneurysm unrelated to the branching zone (Type 4). Type 4 aneurysms arose in areas with minimally disrupted internal elastic lamina without intimal thickening, and there was a risk of rupture.

In our case, the distal middle cerebral artery aneurysm seemed to be related to the slight arterial dilatation of

Table 1: Summary of distal middle cerebral artery aneurysms with histopathological analysis.

Author (year)	Age/Sex	Onset	Portion	Histopathological diagnosis (etiology)
Bamford, et al. (1986) ^[4]	56/M	Rupture	M4	Pseudoaneurysm (infection)
Bandoh, et al. (1987) ^[3]	26/F	Rupture	M4	Pseudoaneurysm (infection)
Sasaki, et al. (1991) ^[14]	41/F	Rupture	M2	Dissecting aneurysm
Piepgras, et al. (1994) ^[10]	56/F	Incidental	M3	Dissecting aneurysm
Hashimoto, et al. (1995) ^[6]	44/M	Rupture	M3	Dissecting aneurysm
Anzai, et al. (1999) ^[2]	26/F	Rupture	M2	Dissecting aneurysm
	52/F	Rupture	M3	Dissecting aneurysm
Tsuboi and Adachi (2002) ^[17]	55/F	Rupture	M4	Pseudoaneurysm (infection)
Sakamoto, et al. (2003) ^[13]	65/F	Rupture	M3	Dissecting aneurysm
Yamakawa, et al. (2003) ^[18]	21/F	Rupture	M4	Pseudoaneurysm (infection)
Saito, et al. (2007) ^[12]	54/M	Rupture	M4	Saccular aneurysm
Inukai, et al. (2010) ^[7]	57/M	Rupture	M4	Dissecting aneurysm
Takemura, et al. (2010) ^[16]	56/F	Rupture	M2	Saccular aneurysm
Sato, et al. (2012) ^[15]	57/M	Rupture	M3	Dissecting aneurysm
Kinoshita, et al. (2014) ^[8]	41/F	Rupture	M2	Dissecting aneurysm
Anno, et al. (2018) ^[1]	76/F	Incidental	M4	Saccular aneurysm
Present case (2020)	67/F	Incidental	M4	Pseudoaneurysm (unclear)

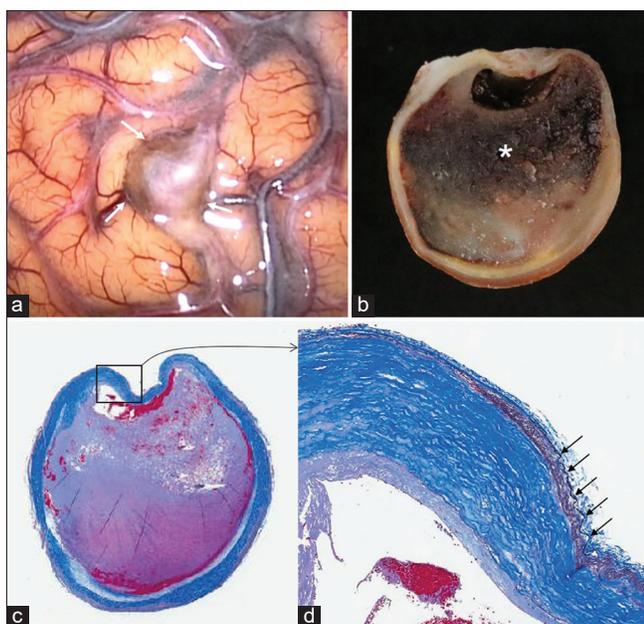


Figure 3: Intraoperative view (a) demonstrates an aneurysm confirmed from the brain surface, and xanthochromic and hypertrophic arachnoid membranes around the aneurysm (arrows). Macroscopically there is a mural thrombus (asterisk) on the thick wall of the aneurysm (b). An elastic Masson stain (c) shows no distinctive structure of the artery but incomplete intima in the aneurysmal wall. There is little intima with disrupted internal elastic lamina (arrows) in the outermost layer of the aneurysm (d).

the same portion that was demonstrated 8 years earlier. Intraoperative findings of xanthochromic and hypertrophic arachnoid membrane and the significant arachnoid adhesion around the aneurysm suggested previous microbleed. We propose that the pseudoaneurysm appeared and increased

gradually followed by microbleed of an aneurysm unrelated to the branching zone of the left angular artery. We speculate that the original aneurysm would be classified as a saccular aneurysm unrelated to the branching zone (Mizutani classification Type 4).

CONCLUSION

We report an extremely rare case of an incidental pseudoaneurysm of the distal middle cerebral artery. We propose that the pseudoaneurysm appeared and increased followed by microbleed of an aneurysm unrelated to the branching zone. Although the natural course of such a lesion is unclear, we believe retrospectively that without appropriate treatment, the aneurysm in our case was most likely to cause future major bleeding.

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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