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## Case Report

# Distal middle cerebral artery dissection with concurrent completely thrombosed aneurysm manifesting as cerebral ischemia. A case report and review of the literature <sup>☆</sup>

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## ABSTRACT

We report the case of a 70-year-old woman who presented with an acute ischemic stroke involving the left frontal operculum secondary to an M2 dissection and a concomitant completely thrombosed aneurysm of the left distal middle cerebral artery. Initial imaging work-up was inconclusive due to the lack of typical radiographic features and only repeated imaging studies pointed towards the presence of an arterial dissection combined with a completely thrombosed aneurysm. The aneurysm was partially clipped and wrapped with excellent clinical result at 1-year follow-up. The clinical, imaging and therapeutic challenges of this rare entity are discussed.

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## Introduction

Acute ischemic infarct secondary to complete thrombosis of an unruptured non-giant intracranial aneurysm is

uncommon and it is estimated to occur in 0.3% of all patients presenting with acute ischemic stroke or transient ischemic attack [1]. Aneurysms originating from branches of the middle cerebral artery distal to the main bifurcation represent 1.1%–7% of all MCA aneurysms and the most

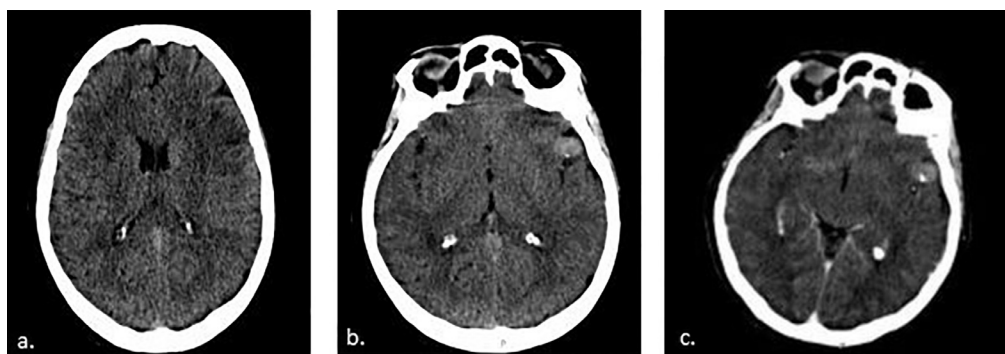
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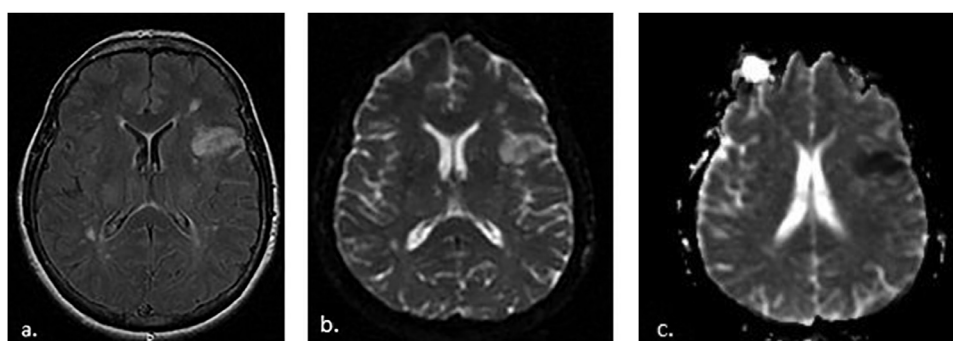
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**Fig. 1 – Brain CT on admission. (a) Acute ischemic changes affecting the left frontal operculum are noticed. (b + c) There is a hyperdense lesion in the anterior left Sylvian fissure. The lesion shows no enhancement after intravenous contrast administration and demonstrates focal peripheral calcifications.**



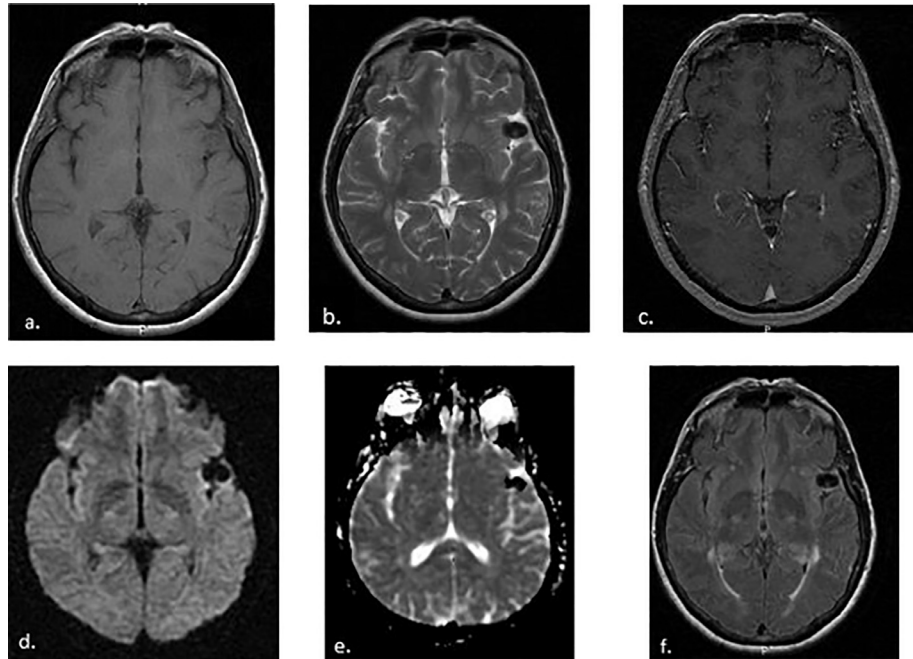
**Fig. 2 – The FLAIR (a), Diffusion weighted image (b) and the ADC map (c) on initial presentation show an acute ischemic stroke in the left frontal operculum.**

common etiology for their formation is infection with mycotic emboli, trauma, vasculitis, tumor emboli or dissection [2,3]. Thrombosis of these aneurysms resulting in brain ischemia is extremely rare [4]. The coexistence of an ischemic infarct and thrombotic lesion occasionally poses a diagnostic and therapeutic dilemma. We report a patient who suffered an acute ischemic stroke due to complete thrombosis of a distal middle cerebral artery aneurysm, probably of dissecting origin, which was misdiagnosed as a neoplastic lesion at primary imaging workup. The patient eventually underwent surgery with partial clipping and wrapping of the aneurysm.

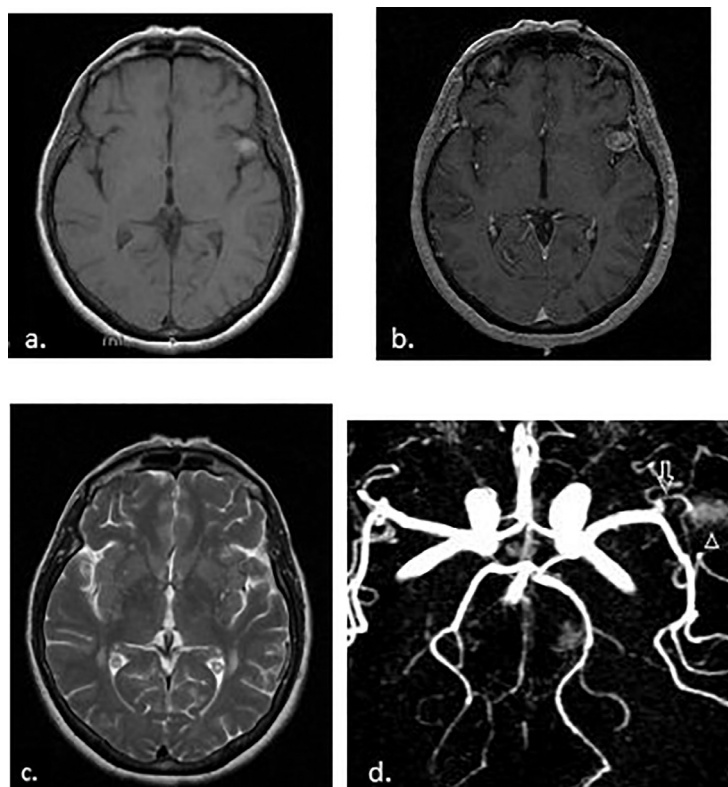
## Case

A 70-year-old woman presented to the emergency department 10 hours after abrupt onset of right-sided weakness and difficulty speaking. Her past medical history was positive for hypertension and diabetes mellitus. Brain computed tomography showed loss of grey-white differentiation in the left frontal operculum (Fig. 1). Additionally, a well demarcated hyperdense lesion with focal peripheral calcification adjacent to the left sylvian fissure was recognized. The patient was started on 75 mg oral acetylsalicylic acid per day.

Brain magnetic resonance images obtained the next day showed an acute ischemic lesion affecting the left frontal operculum (Fig. 2.) and a 17 mm ovoid mass-like lesion in the left anterior sylvian fissure. The lesion was isointense on T1-weighted images, hypointense on T2-weighted images and showed slight peripheral rim enhancement. No diffusion restriction was evident. Susceptibility artefacts secondary to blood products inside the lesion were seen on T2\* images but there was no evidence of subarachnoid hemorrhage or perilesional edema (Fig. 3). Standard blood tests, electrocardiography and echocardiographic studies were normal. A magnetic resonance imaging (MRI) performed 3 weeks later showed stability of the size of the lesion. However, the lesion was now hyperintense on T1-weighted and isointense on T2-weighted images with marked rim enhancement. MR angiography showed occlusion of the prefrontal M2 segment at the level of the lesion (Fig. 4). These findings suggested the possibility of a thrombosed aneurysm, probably of dissecting origin, as the most likely diagnosis. Digital subtraction angiography showed no lesion opacification and occlusion of the left prefrontal artery (Fig. 5). The patient was operated, and the intraoperative findings confirmed the diagnosis of a dissecting and completely thrombosed aneurysm of the left M2 segment completely incorporated into the wall of the parent artery (Fig. 6). The aneurysm was partially clipped and wrapped with



**Fig. 3** – The lesion is isointense on T1-weighted images (a), hypointense on T2-weighted images (b). The contrast enhanced T1 sequence demonstrates slight peripheral rim enhancement (c). There is no diffusion restriction on the diffusion images (d + e). There is no perilesional edema on FLAIR images (f).



**Fig. 4** – Brain MRI after 3 weeks. The lesion appears now hyperintense on T1-weighted images (a) and isointense on T2-weighted images (c). Marked concentric enhancement is observed on the T1 post-contrast image. (d) TOF MIP angiographic image. The lesion (white arrowhead) appears on the course of left M2 segment. Focal stenosis (white arrow) and dilatation of the vessel is noticed which is occluded distally.

**Table 1 – Reported cases of totally thrombosed aneurysms.**

Author	Symptoms	Size	Location	CT/MRI	Initial diagnosis	Treatment
Chihi [14]	Weakness of the right leg Headache	54 mm	ACA	Heterogeneous Calcifications Perifocal edema	Cerebral cystic echinococcosis	Aneurysm clipping and resection
Fifi [19]	Migraine	10 mm	A2	Heterogeneous Calcifications Hypointense rim on T2 Subtle enhancement Restricted diffusion	Dermoid cyst	Trapping and aneurysm resection
Spallone [24]	Hemiparesis	NA	VA	Hyperdense on CT	Vestibular schwannoma	Aneurysm clipping
Lee [16]	Dizziness Paraesthesias	19 mm	VA	Enhancement	Vascular wall tumor	Observation
Lan [18]	Headache	45 mm	PICA	Hypointense rim on T2	Epidermoid cyst	Aneurysm clipping and resection
Woo [15]	Dizziness Left sided weakness	20 mm	PICA	Hyperdense on CT Blooming artifacts on SWI Edema “Target sign”	Ependymoma	Aneurysm clipping and resection
Päsler [23]	Headache Hearing loss	NA	AICA	Enhancement	Vestibular schwannoma	Aneurysm resection AICA reconstruction
Lim [17]	Headache	25 mm	PICA	Hyperdense on CT Calcifications	Cavernous angioma	Aneurysm clipping and resection
Nguyen [20]	Seizures	10 mm	Distal MCA	Peripheral enhancement Blood products Edema	Metastasis	Aneurysm trapping and resection
Kim [21]	Headache	19 mm	M1	Hyperdense on CT Edema Rim enhancement	Glioma Metastasis Ganglioglioma	Aneurysm clipping and resection
Trungu [22]	Tinnitus Headache	12 mm	Distal M2	Hyperdense on CT Edema Enhancement	Cavernous angioma	Aneurysm clipping and resection

Abbreviations: ACA, anterior cerebral artery; ACOM, anterior communicating artery; AICA, anterior inferior cerebral artery; CT, computer tomography scan; CTA, computer tomography angiography; DSA, digital subtraction angiography; ICA, interior carotid artery; MCA, middle cerebral artery; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; NA, not available; VA: vertebral artery; PICA, posterior inferior cerebral artery, ICA: interior carotid artery, MCA: middle cerebral artery, AICA: anterior inferior cerebral artery, CT: computer tomography scan, CTA: computer tomography angiography, MRI: magnetic resonance imaging, MRA: magnetic resonance angiography, SWI, susceptibility weighted imaging; VA, vertebral artery, DSA: digital subtraction angiography, NA: not available.

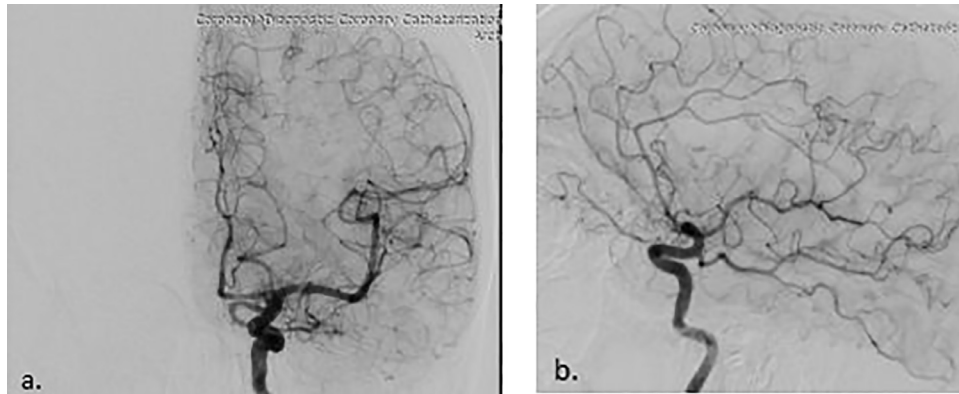
an uneventful postoperative course. The patient remains in excellent clinical condition at 1-year follow-up.

## Discussion

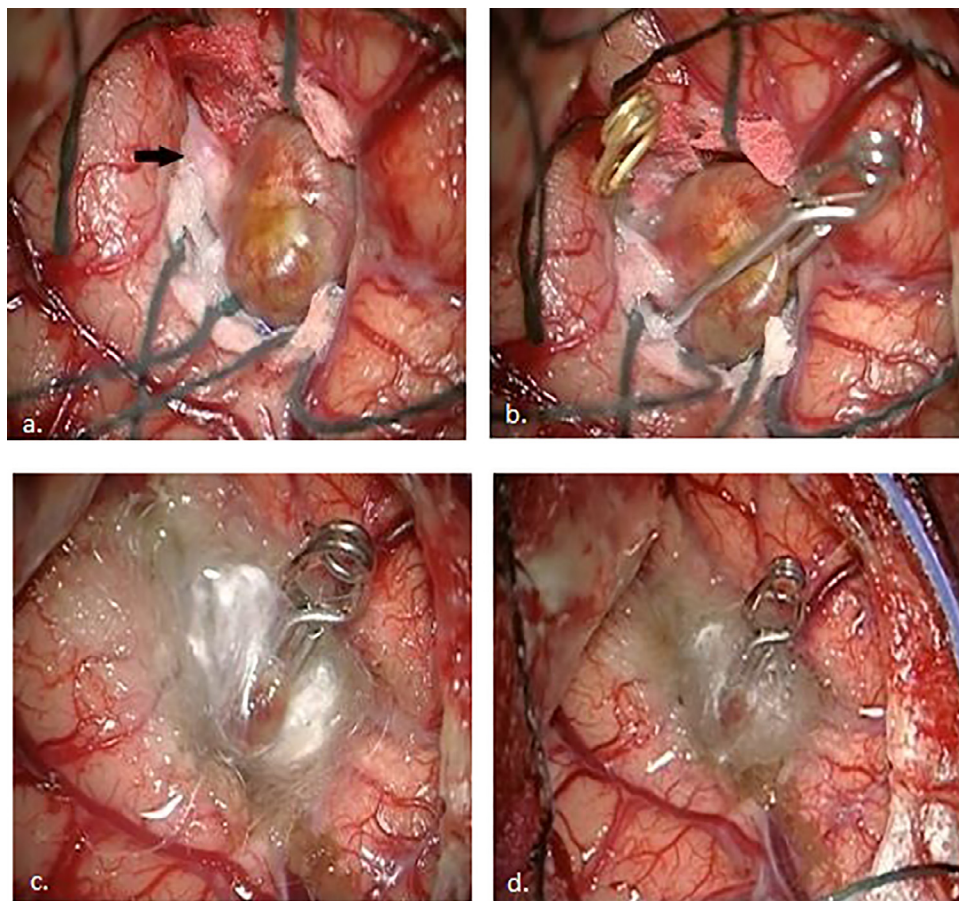
Dissecting intracerebral aneurysms are usually encountered in younger people and frequently manifest with subarachnoid hemorrhage but may also provoke ischemia in 6% to 34.6% of cases. The M1 segment is affected in 57%–69% of cases, while dissection of the M2–M3 segment is commonly associated with the formation of an aneurysm secondary to the disruption of the internal elastic vessel lamina [5–9]. This creates a pseudolumen which communicates with the true lu-

men of the vessel. Thrombosis of the pseudolumen of the dissected vessel may cause stenosis or occlusion of the parent artery or distal embolization resulting in cortical and subcortical cerebral ischemia or hemodynamic compromise resulting in watershed infarcts [10,11]. In the present case the most likely mechanism of infarction was the stenosis or occlusion of the dissected left prefrontal M2 segment.

Imaging characteristics of partially thrombosed giant cerebral aneurysms have been well documented, but the diagnosis is challenging when thrombosis is complete. Characteristic CT features include nonhomogeneous high-density attenuation of the aneurysm lumen, “target sign”, peripheral rim enhancement and curvilinear calcifications. Magnetic Resonance may demonstrate a lumen with heterogeneous signal on T1- and T2-weighted images, “onion skin” appearance on noncontrast



**Fig. 5 – The digital subtraction angiography demonstrates no lesion and occlusion of the left prefrontal M2 segment.**



**Fig. 6 – Intraoperative photographs showing a large thrombosed aneurysm. (a) A yellowish round mass was observed arising from the left M2 segment (black arrow). (b + c + d). A vascular clip was applied and the aneurysm was subsequently wrapped. (Color version is available online.)**

T1 weighted images, a flow void sign, thrombus non-enhancement or rim enhancement along the thrombus margin [12,13]. Eleven similar cases have been reported in the literature (Table 1) and were commonly mistaken for neoplasms on initial imaging work up with the final diagnosis commonly established during surgery [14–24]. Only 3 aneurysms affected the middle cerebral artery. Perilesional edema and heteroge-

neous enhancement were documented in 5 cases respectively, peripheral rim calcifications in 3 cases, rim enhancement on MRI in 2 cases, hypointense rim on T2-weighted images in 2 cases and restricted diffusion in 1 case. Aneurysms appeared with varying signal intensity on T1- and T2-weighted images, representing thrombus of different age; acute thrombus appeared isointense on T1-weighted images and hypointense

on T2-weighted images, while subacute thrombus could appear hyperintense on T1- and hypointense on T2-weighted or hyperintense on both T1- and T2-weighted images. Angiographic studies, when available, didn't demonstrate aneurysm opacification but only distal occlusion of the parent artery. In our case the aneurysm showed features of thrombosis like focal calcification and subtle rim enhancement; however, these findings were not specific as they may be encountered in other lesions such as cavernous angiomas or hemorrhagic tumors. The relatively rapid change of signal characteristics and the concentric enhancement on follow up MRI raised the suspicion of a thrombosed aneurysm. Furthermore, the focal lumen narrowing followed by dilatation and distal occlusion of the parent artery raised the possibility of a dissection. Other imaging findings related to dissection like "pearl and string" sign, intimal flap, double lumen were absent. Digital subtraction angiography was non- diagnostic due to complete thrombosis of the aneurysm.

To date there are no definitive guidelines for the optimal treatment of ischemic infarcts resulting from thrombosed cerebral aneurysm, dissecting or not. Calviere et al. [1] reported 15 patients who presented with ischemic stroke or transient ischemic attack with the coexistence of an unruptured aneurysm on the symptomatic cerebral artery. Aneurysm thrombosis was present in 10 patients. Thirteen patients were treated with an antiplatelet agent (acetylsalicylic acid, clopidogrel or a combination of both). After a mean follow up of 393 days, no ischemic recurrence occurred; however, there was a high rate of partial or complete recanalization of aneurysm thrombosis (70%) and 2 cases of aneurysm rupture. A similar high rate of recanalization secondarily to antithrombotic treatment was reported by Guillon et al. [25] who stressed the importance of cautious use of antithrombotics, fibrinolytics or other intra-arterial therapies. Several other case reports have documented aneurysm rupture or recanalization after antithrombotic treatment [26–28]. Nevertheless, Arauz et al. [29] described 3 patients with ischemic stroke and unruptured thrombosed aneurysms treated with low dose of acetylsalicylic acid with no hemorrhagic complication, recanalization or recurrent ischemia at follow-up. Our present patient received acetylsalicylic acid. Though there was no evidence of subarachnoid hemorrhage or new ischemic changes, the aneurysm showed signal changes on follow-up MRI at 3 weeks.

Thrombosed aneurysms should be closely followed by imaging to detect recanalization, growth, mass effect or dissection progression. In such cases, early endovascular embolization or surgical obliteration of the aneurysm is mandatory to reduce the risk of rupture, recurrent distal embolism or progression of infarction [1,30–32]. Concerning dissecting aneurysms presenting with ischemia surgical treatment has shown better results compared to conservative treatment [6,31]. Surgery may require techniques other than direct clipping, including reconstructive neck clipping combined with aneurysm wrapping or indirect aneurysm occlusion with or without bypass [33]. In our case the aneurysm could not be clipped because of the dissecting morphology and was eventually treated by partial clipping and wrapping to decrease the bleeding risk. As the current knowledge for the treatment of dissecting thrombosed intracranial

aneurysm relies on a limited number of case reports, the optimal treatment strategy should be considered on an individual basis.

## Conclusion

Completely thrombosed large distal dissecting middle cerebral artery aneurysms represent a diagnostic and therapeutic challenge. The combination of parenchymal ischemia and hemorrhagic lesion on the course of the corresponding feeding artery should raise the suspicion of a thrombosed aneurysm. The optimal treatment strategy should be considered on an individual basis.

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