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Case Report

# Delayed rebleeding of a spontaneously thrombosed aneurysm after subarachnoid hemorrhage

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## Abstract

**Background:** This report provides a rare documentation of spontaneous thrombosis of a ruptured aneurysm followed by delayed recanalization and subsequent rerupture.

**Case Description:** A 47-year-old female presented with spontaneous subarachnoid hemorrhage (SAH). Four aneurysms were identified on CT angiogram including a basilar apex aneurysm, considered source of bleeding. Cerebral angiogram on postbleed day (PBD) #1 showed spontaneous thrombosis of basilar apex aneurysm. The patient was discharged to a nursing home on PBD #18 after two subsequent studies showed no recanalization of the basilar aneurysm. The patient returned on PBD #26 with a second episode of spontaneous SAH. The previously thrombosed basilar aneurysm had recanalized and reruptured, which was now treated with coil embolization.

**Conclusion:** We are not aware of a previous report of saccular cerebral aneurysm documenting spontaneous thrombosis after SAH and recanalization with second hemorrhage. This occurrence presents a dilemma regarding the timing and frequency of subsequent cerebrovascular imaging and treatment.

**Key Words:** Aneurysm, embolization, recanalization, subarachnoid hemorrhage, thrombosis

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## INTRODUCTION

Spontaneous thrombosis of cerebral aneurysms is known to occur.[1,3,10] Spontaneous thrombosis after subarachnoid hemorrhage (SAH) may result in early false-negative vascular studies prompting delayed vascular studies when the initial workup is negative.[5] This report provides rare documentation of spontaneous thrombosis of a ruptured aneurysm followed by delayed recanalization and subsequent rerupture. The challenges of managing a known, thrombosed intracranial aneurysm are discussed.

## CASE REPORT

A 47-year-old female presented with spontaneous SAH. Computed tomography angiography (CTA) demonstrated diffuse SAH and intraventricular haemorrhage [Figure 1a]. Four aneurysms were identified including an 8 mm basilar apex aneurysm, a 7 mm anterior communicating artery (AComm) aneurysm, and smaller bilateral middle cerebral artery aneurysms [Figure 1b]. Based on the pattern of bleeding and the size of the aneurysms, the basilar apex aneurysm was identified as the source of bleeding.

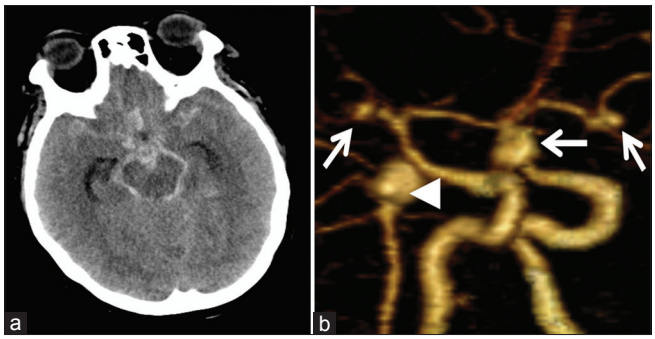

**Figure 1: Initial presentation with diffuse subarachnoid hemorrhage around basal cisterns (a) and CTA demonstrating a basilar apex aneurysm (arrow head) as probable source and three additional anterior circulating aneurysms (arrows)**

## First intervention

Catheter angiography on postbleed day (PBD) #1 showed irregularity at the basilar apex with no filling of the dome of the basilar aneurysm, consistent with spontaneous thrombosis [Figure 2a-d]. The AComm aneurysm was treated with coil embolization. No anticoagulation was given.

## Subsequent course

CTA on PBD #2 showed no filling of the basilar aneurysm. Catheter angiography on PBD #8 showed moderate vasospasm of the basilar artery, fullness of the basilar apex with less irregularity of the apex, but no filling of the dome or neck of the aneurysm [Figure 2e-f]. Routine CT imaging showed progressive resolution of SAH and the neurological exam improved. The patient was transferred to a long-term acute care facility after an 18-day hospital stay. At the time of discharge she was arousable to voice and following simple commands with a mild paresis of the left upper extremity and paralysis in the right upper extremity.

## Second presentation and intervention

A rapid decline in her neurologic functioning occurred on PBD #26. Head CT showed new acute SAH in the fourth ventricle and in the prepontine cistern, extending inferiorly along the medulla [Figure 3a]. Catheter angiography on PBD #27 showed recanalization of the basilar apex aneurysm [Figure 3b-d]. There was no filling of the anterior communicating artery aneurysm. The basilar artery aneurysm was treated with coil embolization [Figure 3e and f].

# DISCUSSION

We are not aware of a previous report of saccular cerebral aneurysm documenting spontaneous thrombosis after SAH and recanalization with second hemorrhage. We performed a PubMed database search with the following key words: "intracranial aneurysm", "rupture", "spontaneous thrombosis", "rerupture", and "recanalization". Spontaneous thrombosis is uncommon

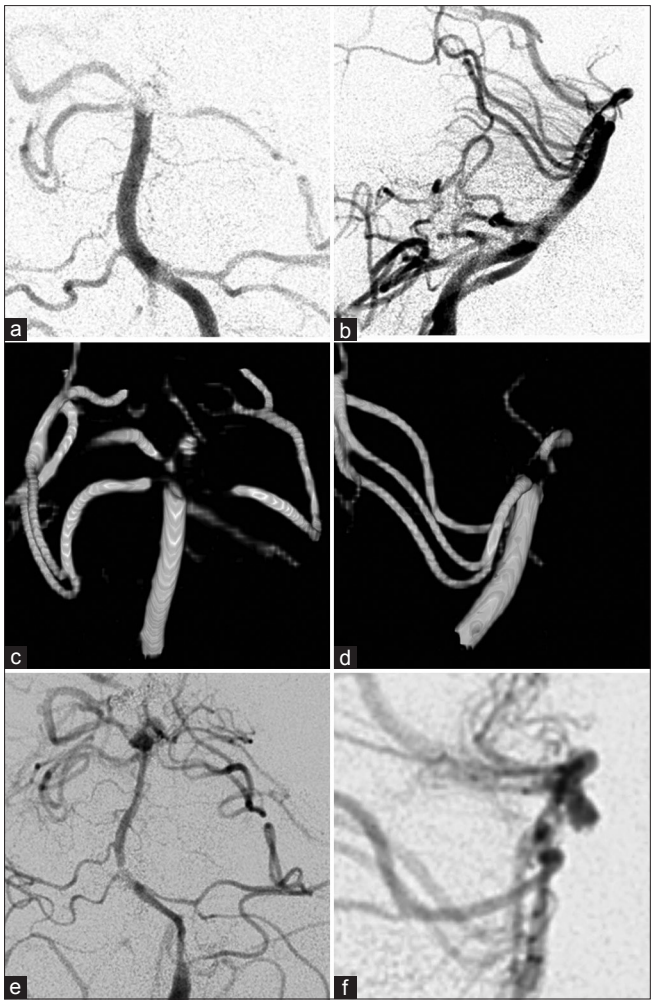

**Figure 2: Postbleed day 1 (PBD#1) catheter angiogram demonstrating irregular basilar apex without evidence of aneurysm filling indicating spontaneous thrombosis (a: AP and b: lateral views; c and d 3D reconstructions in AP and lateral views). PBD#8 catheter angiogram again did not show aneurysm filling (e and f). Note significant vasospasm of basilar trunk in (e). Lateral view is magnified to show persistent thrombosis of basilar apex aneurysm (f)**

but well documented.[1-3,10,12] Khurana *et al.* describe a case of ruptured giant middle cerebral artery aneurysm with thrombosis and fatal rerupture, but do not angiographically document complete thrombosis after initial rupture.[6] Atkinson *et al.* reported recanalization of a posterior cerebral artery fusiform aneurysm and subsequent surgical treatment.[1] They identified six previous reported cases of angiographic reappearance of a known, thrombosed aneurysm, but did not report rebleeding. Explanations for spontaneous thrombosis have considered anatomic and hemodynamic factors, inflammation, and hypercoagulability.

More recently, hemorrhage after apparent "complete" thrombosis has been reported with endovascular flow-diversion treatment.[4,7] Raymond *et al.* studied thrombosis and hemorrhage in a swine model.[11] They found that thrombosis was accompanied by degeneration

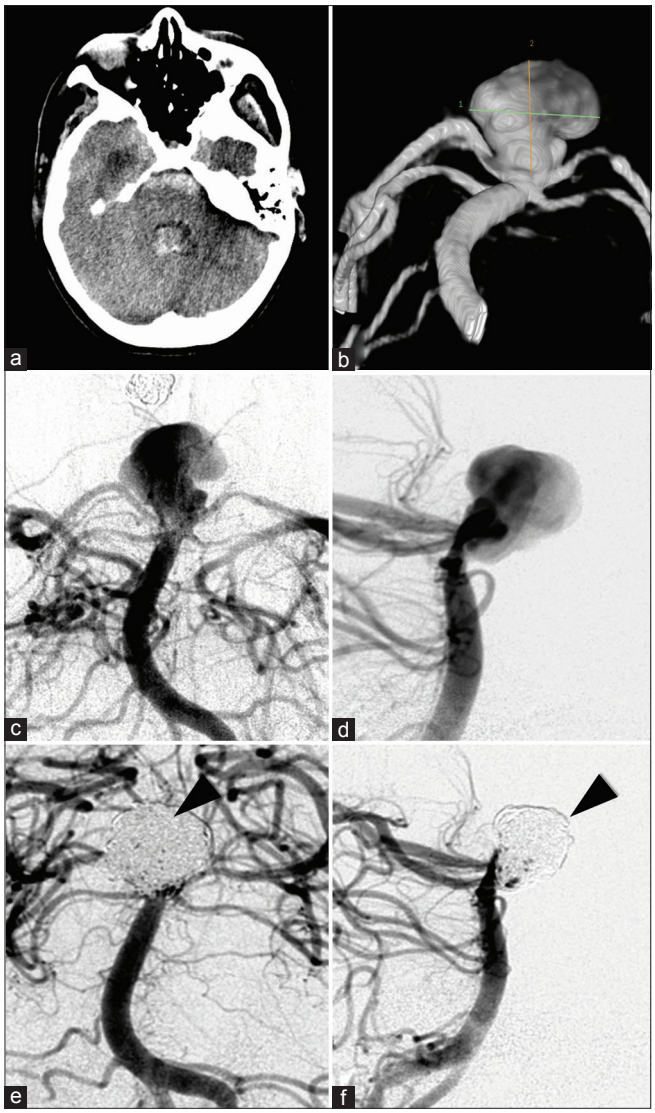


**Figure 3: Rapid decline of patient on PBD#26 demonstrated new SAH on CT scan (a) and redemonstration of previously thrombosed basilar apex aneurysm on catheter angiography (b: 3D reconstruction; c: AP and d: lateral views), which was subsequently coil embolized (e: AP, f: lateral views). Arrowheads demonstrate coil mass in e and f.**

of elastic fibers and smooth muscle cells and that recanalization and recirculation into the degenerated wall precipitated rupture. It may be that the rate of thrombosis after flow-diversion-sometimes occurring over weeks or months-is more likely to promote or allow neovascularization of the aneurysm wall than the acute thrombosis that is caused by coil embolization. Moreover, relative hypoxia in the thrombosing aneurysm also alters specific biological pathways influencing vascular remodeling.[8] In fact, hypoxia inducible factor-1a (HIF-1a) is upregulated in the aneurysm wall and has been shown to promote pathological smooth muscle remodeling and neo-vascularization, likely contributing to aneurysm progression and rupture.[9]

Our practice in patients with SAH (exceeding the perimesencephalic cistern) and negative CTA has been to perform catheter angiography immediately, and at one week and four weeks as needed. Patients with negative immediate angiography are screened for spinal pathology with magnetic resonance imaging (MRI). We intended to follow the same protocol with this patient, however, the basilar aneurysm recanalized and reruptured prior to the delayed study. The likelihood of this sequence of events is too low to justify a more aggressive imaging protocol. However, if faced again with the same situation, we will likely screen weekly with CTA or MRA for at least the first month and less frequently thereafter.

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