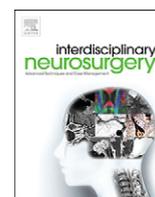




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Symptomatic regrowth of a small intracranial aneurysm that had ruptured and completely thrombosed: a case report

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ABSTRACT

We report a case of small internal carotid–posterior communication artery (IC–PC) aneurysm that was completely thrombosed after initial bleeding, but subsequently became symptomatic, causing a mass effect. A 54-year-old woman initially presented with grade-five subarachnoid hemorrhage from a small right IC–PC aneurysm. The aneurysm was treated conservatively and completely thrombosed within 35 days. The patient slowly recovered and remained well until 4 years later, when she developed right oculomotor nerve palsy. Imaging revealed relapse of the aneurysm, and repair led to symptom resolution. This case offers a reminder that totally thrombosed aneurysms carry a risk of regrowth if left untreated.

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Introduction

Spontaneous aneurysmal thrombosis is not an uncommon event in the natural history of intracranial aneurysms after subarachnoid hemorrhage (SAH). Such thrombosis, however, is known to be partial in most cases; total thrombosis of a small aneurysm is rare. On the other hands, recanalization of a thrombosed aneurysm is usually observed in the acute-to-subacute phase, and few reports have noted chronic-phase recanalization and growth of a totally thrombosed small aneurysm. Here, we report a case of a small ruptured aneurysm that was totally thrombosed during the acute phase of SAH, but relapsed and caused a mass effect four years after the initial bleeding event.

Case report

A 54-year-old woman suddenly collapsed and was transferred to our institution in October 2008. On arrival, she was deeply comatose with moderately dilated pupils. Computed tomography (CT) revealed diffuse thick SAH (Fig. A1). Three-dimensional computed tomographic angiography (3D-CTA) showed a right-sided internal carotid–posterior communicating (IC–PC) aneurysm, which had a diameter of 2 mm (Fig. A2). The patient had been receiving anticoagulant therapy because of a clinical history of deep venous thrombosis, and her prothrombin time–international normalized ratio was 2.54. Considering her poor clinical condition, conservative therapy was initially administered. Follow-up digital-subtraction angiography (DSA) was performed twice (3 and 14 days after onset), showing no cerebral vasospasm. Thirty-five days after initial bleeding, 3D-CTA revealed

that the aneurysm was almost thrombosed (Fig. A3). We regarded the aneurysm as having been cured naturally and the patient was transferred to another hospital without surgical intervention. She recovered slowly and returned home thereafter.

In September 2012, four years after the SAH, she revisited our institution complaining of double vision. Neurological examination revealed right partial oculomotor palsy. Magnetic resonance angiography using a 3-T system demonstrated an irregular shaped, large right IC–PC aneurysm (Fig. B.1, 2). The small thrombosed aneurysm was presumed to have subsequently recanalized and increased in size, compressing the oculomotor nerve. Because the patient showed impaired renal function, 3D-CTA and DSA were not performed. She underwent expedited surgery, and the aneurysm was successfully repaired. Postoperatively, her oculomotor palsy was improved, and she was transferred to a short-term rehabilitation facility.

Discussion

Thrombosis of ruptured intracranial aneurysms after SAH is a well-known phenomenon. Complete thrombosis is seen in 1%–2% or less of all ruptured aneurysms [1]. Such complete thrombosis has been considered to represent spontaneous cure of the aneurysm. On the other hand, some reports have described a risk of recanalization or even further hemorrhage after complete thrombosis [2,3]. No consensus has been reached regarding surgical intervention for the management of spontaneously thrombosed aneurysms. The feasibility of such intervention may depend on the condition of the individual patient and/or institutional policies. In the present case, a follow-up examination was not performed after the patient was transferred to another hospital, in light of her poor clinical status at the initial bleeding event. However, the small aneurysm was growing, and reached a diameter of 10 mm or larger, over the course of four years during which follow-up examinations were not performed. We

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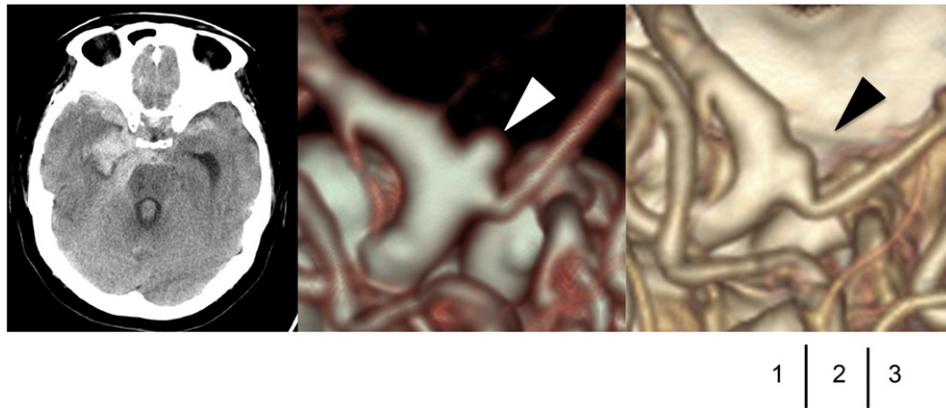


Fig. A. Radiological images at the first admission. (1) Axial CT shows a diffuse, thick SAH with parenchymal hematoma in the right temporal lobe. (2) 3D-CTA reveals a posterior-projecting small IC-PC aneurysm (white arrowhead). (3) 3D-CTA 35 days after bleeding shows complete occlusion of the aneurysm (black arrowhead).

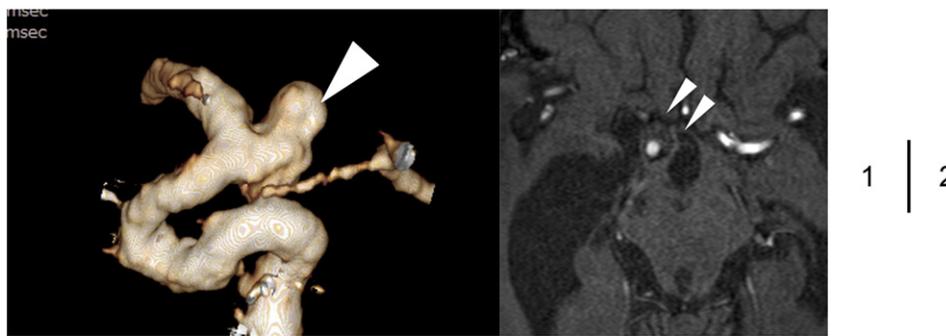


Fig. B. Magnetic resonance angiography (MRA) four years after initial bleeding. (1) 3D-MRA delineates a large IC-PC aneurysm (white arrowhead). (2) Original axial 3D-MRA reveals partial thrombosis of the aneurysm (double white arrowheads).

should have performed radiological examinations continuously after the patient's transfer. At the very least, annual radiological follow-up is recommended for such cases of completely thrombosed aneurysm.

It has been suggested that various factors may be mechanistically involved in the spontaneous thrombosis of intracranial aneurysms, including aneurysm shape (particularly in terms of a high dome/neck ratio), vasospasm, systemic hypotension, antifibrinolytic therapy, compression of the parent artery by the aneurysm, thrombosis of the parent artery, and compression by surrounding brain edema and clotting in the brain [3]. Most of the underlying pathogeneses could involve Virchow's triad for vessel thrombosis: stasis, hypercoagulopathy, or endothelial injury. Our case appeared morphologically unique. First, the aneurysm was small and showed a low dome/neck ratio. Second, parent arteries (including the internal carotid artery and the fetal-type posterior communicating artery) were neither compressed nor stenotic. No vasospasm or systemic hypotension was observed. Furthermore, the patient had been receiving anticoagulant treatment. Other factors, such as endothelial injury or hypercoagulability after cessation of warfarinization, were presumably involved in thrombosis in this case.

The proposed mechanisms for recanalization include liquefaction of the thrombus and subsequent intrathrombotic dissection, surgical intervention, and abatement of vasospasm. Indeed, spontaneous disappearances and reappearances of small ruptured saccular aneurysms have been documented [3–5]. In all cases, recanalization occurred by the end of vasospasm. In the present case, however, the aneurysm was not recanalized for at least 35 days after bleeding. Reviewing the literature, we found no similar cases with recanalization a month or more after

initial bleeding. This case supports the notion that an entire thrombosed aneurysm can recanalize and regrow, long after the initial bleeding event.

Conclusion

This present report offers a reminder that long-term follow-up is important for patients showing total thrombosis of the aneurysm, and that complete thrombosis of the aneurysm is not equivalent to aneurysmal cure.

Ethical standards

The present report was reviewed and approved by an independent ethics committee at our institution.

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