

## Case Report

# Recanalization of Completely Thrombosed Non-Giant Saccular Aneurysm Mimicking as *De Novo* Aneurysm

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Partial thrombosis of giant aneurysms is not uncommon however, complete angiographic occlusion occurs less frequently. In the case of non-giant aneurysms, complete thrombosis and recanalization has been rarely reported. A 31-year-old man presented to the emergency department with sudden bursting headache. Brain computed tomography (CT) revealed diffuse subarachnoid hemorrhage on the left side. Both CT angiography (CTA) and digital subtraction angiography showed suspicion of small left anterior choroidal artery aneurysm. We performed surgical exploration. In the operation field, anterior choroidal artery aneurysm of 2 × 2 mm with broad neck and friable appearance was observed. Because we could not clip without sacrificing the anterior choroidal artery, we performed wrapping only. Follow up CTA after 7 months demonstrated 4 mm right internal carotid artery bifurcation aneurysm. The patient underwent aneurysmal neck clipping. During the operation, 9 × 13 mm sized thrombosed aneurysm was detected and completely clipped. We initially thought this aneurysm to be a *de novo* aneurysm however, it was an aneurysm that had recanalized from a completely thrombosed aneurysm. This case report provides an insight into the potential for complete thrombosis and recanalization of non-giant aneurysms.

**KEY WORDS :** Cerebral aneurysm · Thrombosis · Recanalization

## INTRODUCTION

The incidence of spontaneous thrombosis of intracranial aneurysms whether completely or partially varies depending on their location, size and origin. There are many reports of spontaneous thrombosis of aneurysms, but they are usually giant aneurysms. Even in giant aneurysms, partial thrombosis is not uncommon in the natural history, however complete angiographic occlusion occurs less frequently<sup>2)</sup>.

In non-giant aneurysms, complete thrombosis and recanalization has been only rarely reported<sup>1,2,3,6,9,12,13)</sup>. We describe here a very rare case of recanalization of completely thrombosed non-giant saccular aneurysm confused as a *de novo* aneurysm.

## CASE REPORT

A 31-year-old man with sudden bursting headache was admitted to the emergency department. Brain computed tomography (CT) revealed diffuse subarachnoid hemorrhage (SAH) predominantly on the left side (Fig. 1). On the basis of the results of both CT angiography (CTA) and digital subtraction angiography, the patient was suspected of having small left anterior choroidal artery aneurysm (Fig. 2). We performed surgical exploration. In the operation field, anterior

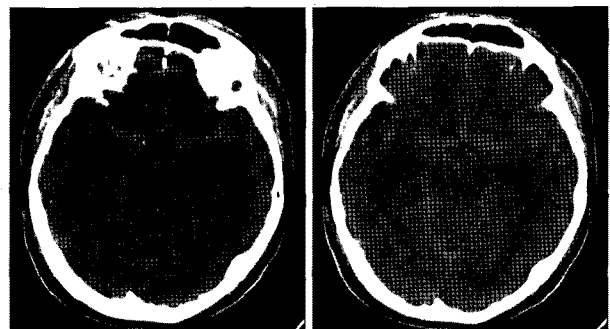
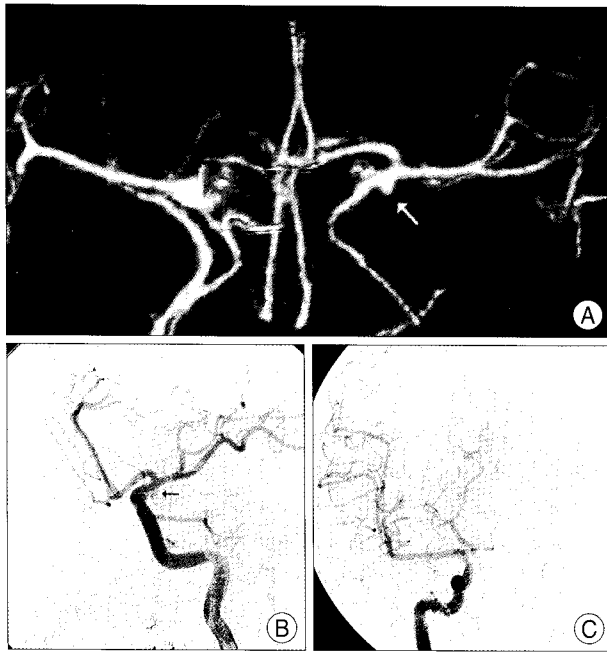
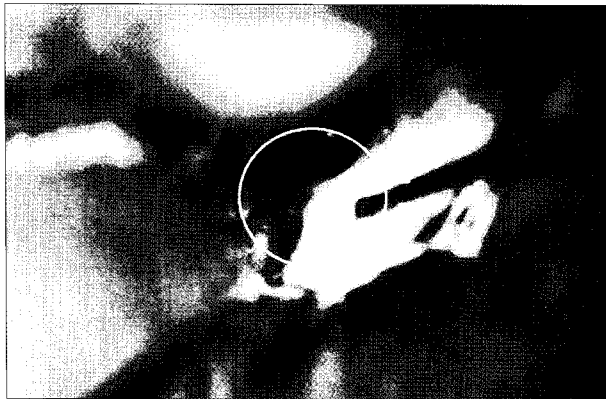


Fig. 1. Brain computed tomography scans at admission showing diffuse subarachnoid hemorrhage which slightly left side dominant.

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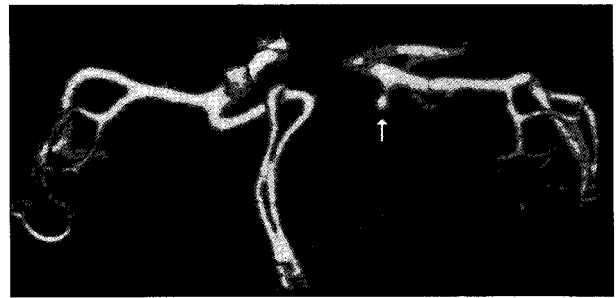


**Fig. 2.** Brain computed tomography angiography (A) and left oblique projection of cerebral angiography (B) shows a small left anterior choroidal artery aneurysm (arrow). Right cerebral angiogram (C) showing no abnormal findings.



**Fig. 3.** Intraoperative photograph (1st operation) demonstrating a 2 × 2 mm sized, friable nature anterior choroidal artery aneurysm (yellow circle).

choroidal artery aneurysm of approximately 2 × 2 mm with a broad neck and friable appearance was observed (Fig. 3). Extensive SAH and an adherence of tiny blood clot to aneurysmal dome, which was thought to be the rupture point, were observed. Because clipping was impossible without sacrificing the anterior choroidal artery, only wrapping was performed using pericranium and surgical glue. Follow up CTA after 7 months revealed a 4 mm right internal carotid artery bifurcation aneurysm (Fig. 4), which was initially thought to be a *de novo* aneurysm. The patient underwent aneurysmal neck clipping. During the operation, thrombosed aneurysm of 9 × 13 mm was detected and completely clipped using 10 mm straight and 9 mm curved standard clips (Fig. 5). The aneurysm was calcified and dark blue color



**Fig. 4.** Follow up brain computed tomography angiography after 7 months showing about 4 mm sized right internal carotid artery bifurcation aneurysm (white arrow).



**Fig. 5.** Intraoperative photograph (2nd operation) demonstrating about 9 × 13 mm sized thrombosed aneurysm (red circle).

owing to thrombosis. Because the neck portion of aneurysm was not tender owing to calcification, 2 clips were used for reinforcement. The aneurysm was not a *de novo* aneurysm but an aneurysm that recanalized from completely thrombosed aneurysm. The postoperative follow-up was uneventful.

## DISCUSSION

Spontaneous thrombosis of aneurysms is a well documented phenomenon that has been noted in approximately 50% of giant aneurysms, however, the incidence of complete thrombosis in giant aneurysms ranges between 13% and 20%<sup>8,14</sup>. In non-giant aneurysms, the occurrence of these phenomena is rare. Edner et al.<sup>3</sup> had suggested that only 1-2% of ruptured intracranial aneurysms will undergo spontaneous and complete thrombosis.

Various factors influence the spontaneous thrombosis of an aneurysm. The major contributing factor is volume to neck ratio of the aneurysm to the balance between thrombogenesis and thrombolysis<sup>1</sup>. In aneurysms with a relatively small neck, intraluminal thrombosis may occur. Other biophysiological parameters such as the aneurysm age, hemodynamics in the parent artery (e.g., vasospasm), direct distortion of the parent artery by aneurysmal sac, endothelial damage

due to intrasaccular turbulent flow, and the angiographic procedure itself have been also proposed<sup>5,7,11</sup>. Cohen et al.<sup>2</sup> suggested that the tamponade effect exerted by the parenchymal bleeding around aneurysm can be another factor inducing thrombosis of aneurysm. Such extraluminal pressure may have modified not only volume to orifice ratio of aneurysm but also other mentioned factors, especially the hemodynamics in the middle cerebral artery secondary to external compression, arterial distortion or transient acute vasospasm. Antifibrinolytic agents have been related to spontaneous thrombosis of ruptured aneurysms. Local inhibition of plasminogen activators in and around the aneurysm wall may cause spontaneous aneurysm thrombosis during treatment with antifibrinolytic drugs<sup>4</sup>. In our patient, antifibrinolytic agent was used once. However, we thought that there was no relationship between thrombosis of aneurysm and antifibrinolytic agent because imaging study was obtained before administration of antifibrinolytic agent.

It should be noted that many of thrombosed non-giant aneurysms were ruptured aneurysms and the patients were relatively younger (mean age 29.4 years, including present case) in the review of the literatures<sup>2,3,6,12,13</sup>. These findings suggest that hemodynamic change (e.g., flow arrest) and arterial change (e.g., contraction of artery or aneurysm) due to aneurysmal rupture play a key role inducing thrombosis of non-giant aneurysms. These changes may have modified aforementioned factors, especially volume to orifice ratio. The relationship between patient's age and complete thrombosis of ruptured non-giant aneurysms has not been described so far. However, younger age is one of risk factors for vasospasm after SAH. The reasons for age-related difference in the incidence of vasospasm are not clearly understood. There are three proposed mechanisms : age-related decrease in cerebrovascular reactivity due to reduced reactivity to spasmogens, age-related differences of structural and mechanical changes and age-related differences in immunological reactivity<sup>10</sup>. From these reasons, we suggest that younger patient group may be more prone to thrombosis of aneurysm because younger patients may have higher vascular reactivity which induces hemodynamic and arterial change.

On the other hand, there's no evidence that the formation of thrombus is regarded as a good sign. Even if the lumen of aneurysm was not detected on the four-vessel angiography, still there's a risk of SAH. As demonstrated in our patient, recanalization may be another event in the dynamic disease process. However, the mechanism of recanalization is poorly understood. Liquefaction of the thrombus and subsequent intrathrombotic dissection by blood flow could be one possible explanation<sup>2</sup>.

## CONCLUSION

This case provides an insight into the potential for complete thrombosis and recanalization of non-giant aneurysms and supports the importance of periodic follow-up for the patients harboring a thrombosed aneurysm. We raise the possibility that complete thrombosis of non-giant aneurysms may be higher in younger patients who experience SAH due to rupture of aneurysm, although such an assumption can't be based on small series of case reports.

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