

## A Pseudoaneurysm Appeared after Rebleeding

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A woman who had a spontaneous subarachnoid hemorrhage (SAH) and temporal intracerebral hemorrhage (ICH) without any causative lesions on computed tomography (CT) and digital angiography at the day of the stroke. She was considered to have an angiographically negative SAH and scheduled for a repeated angiography. While she was waiting for the next study, she developed a second hemorrhage. CT angiography showed an aneurysmal shadow in the course of the posterior cerebral artery. After the operation, the aneurysm proved to be a pseudoaneurysm.

**KEY WORDS :** Subarachnoid hemorrhage · Angiography · Pseudoaneurysm · Posterior cerebral artery.

### Introduction

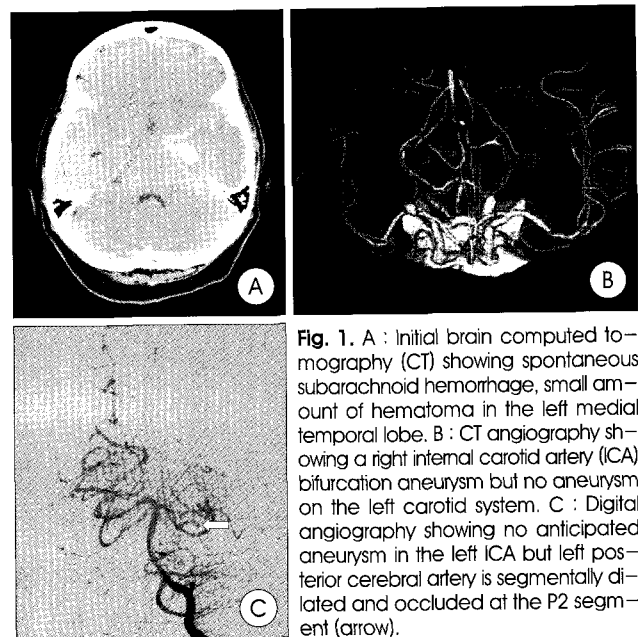
The causes of spontaneous subarachnoid hemorrhage (SAH) are known to be the rupture of cerebral aneurysms in 80% and initially negative angiography ranges between 15-20%. Among these, 5-20% reveal aneurysms on repeated angiography<sup>1,4</sup>. Initially negative angiography is due to a very small aneurysm, thrombosis of an aneurysm, vasospasm, and rupture of a superficial artery or vein.

Pseudoaneurysm develops at the site of the arterial rupture and it is thought to be the result rather than the cause of hemorrhage. The causes of arterial rupture are trauma, infection, neoplasm, spontaneous dissection, and vasculopathy. We are reporting a woman who presented with SAH and temporal ICH, whose angiography didn't disclose a definite aneurysmal shadow at initial study, so that the angiography was interpreted as negative. After rebleeding, pseudoaneurysm appeared in the posterior cerebral artery (PCA) course and initial angiography was re-examined, which disclosed some abnormalities.

### Case Report

A 65-year-old woman presented with suddenly developed headache. At admission, she was slightly drowsy but had no other neurological abnormalities. She had a history of having an untreated, unruptured right ICA bifurcation aneurysm which was diagnosed 7 years prior to admission.

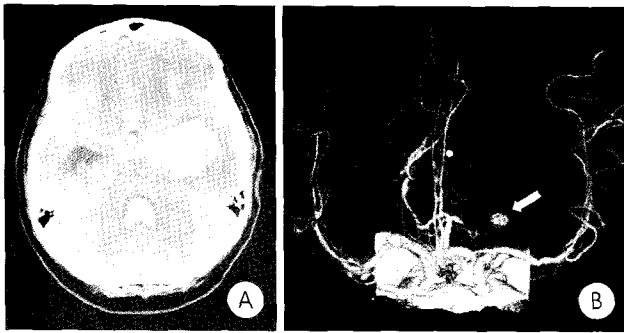
Brain CT showed SAH, small amount of ICH in the left medial temporal lobe and intraventricular hemorrhage (IVH) in the lateral ventricles (Fig. 1A). It was highly suggestive of ruptured left posterior communicating artery (P-com) aneurysm that CT angiography was done. CT angiography showed very sclerotic carotid arteries and previously diagnosed right ICA bifurcation aneurysm but there was no aneurysm on the left carotid system (Fig. 1B). Left PCA was not fully visualized



**Fig. 1.** A : Initial brain computed tomography (CT) showing spontaneous subarachnoid hemorrhage, small amount of hematoma in the left medial temporal lobe. B : CT angiography showing a right internal carotid artery (ICA) bifurcation aneurysm but no aneurysm on the left carotid system. C : Digital angiography showing no anticipated aneurysm in the left ICA but left posterior cerebral artery is segmentally dilated and occluded at the P2 segment (arrow).

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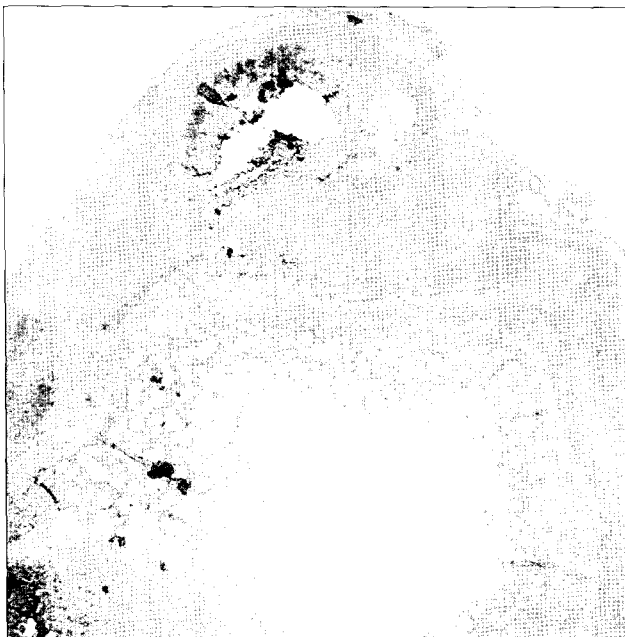
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**Fig. 2.** Seven days after the ictus. A : Brain computed tomography (CT) showing large amount of left temporal hematoma, subarachnoid and intraventricular hemorrhage in all ventricles. B : CT angiography showing occlusion of left P2 segment and 8x7mm round aneurysmal dilatation at P2 course (arrow).

but ignored at that time. To further investigate vascular pathology, transfemoral digital angiography was performed. There was no anticipated aneurysm in the left ICA but left PCA was segmentally dilated and occluded at the P2 segment (Fig. 1C).

After the angiography, she was treated conservatively and scheduled for a repeated angiography 2 weeks later. Seven days after the hemorrhage, she suddenly became comatous with pupillary dilatation. Brain CT showed large amount of left temporal ICH, SAH and IVH in all ventricles (Fig. 2A). CT angiography showed occlusion at left P2 segment and  $8 \times 7$ mm round aneurysmal dilatation at the nonvisualized P2 course (Fig. 2B). Emergency craniotomy was performed. Left sylvian fissure was widely dissected and PCA was traced distally. Round aneurysmal dilatation was found at P2 segment. The aneurysm was dissected from the surrounding tissue and the neck was clipped but the aneurysm was cut off from the neck at the



**Fig. 3.** Aneurysmal wall is composed of hematoma capsule (H&E, X40).



**Fig. 4.** Follow up computed tomographic angiography showing fully visualized left posterior cerebral artery and disappearance of the aneurysmal shadow (arrow).

moment of clipping. The blood pumping neck was clipped and temporal hematoma was evacuated. The aneurysm was sent for pathologic examination but the report revealed it a hematoma capsule (Fig. 3). Follow up CT angiography showed fully visualized left PCA and disappearance of the aneurysmal shadow (Fig. 4) but the patient became severely disabled.

## Discussion

When a patient present with spontaneous SAH and temporal ICH, he or she is considered of having a ruptured posterior communicating aneurysm. But contrary to the author's expectation, the presented patient showed aneurysmal shadow neither on CT nor on digital angiography in the lesion side except a previously diagnosed unruptured aneurysm on the other side. We neglected minor abnormalities of the left PCA at the initial study. After rebleeding, CT angiogram showed occlusion of left PCA at the P2 segment and an aneurysmal dilatation at the PCA course.

The aneurysm proved to be a pseudoaneurysm eventually. Initially negative angiography for spontaneous SAH has been explained as thrombosis, vasospasm, total disruption of an aneurysm, microaneurysm and cryptic vascular malformation<sup>4,7,8</sup>. Microaneurysm could be destroyed at the moment of bleeding that resulted in negative angiography<sup>1</sup>. Spinal vascular malformation, rupture of superficial artery or vein have also been suggested as the possible cause of bleeding<sup>3</sup>. Increased intracranial pressure and focal blood clot might interfere dye filling into the aneurysm and supposedly contribute to early negative angiography. If angiography is performed within 3 days of spontaneous SAH, it is too early to develop vasospasm. Rupture of a small artery or vein might be one of the causes of repeated

negative angiography.

In spontaneous SAH, there are some different CT features that suggest aneurysmal rupture. Intraventricular hemorrhage, thick SAH, bilateral sylvian SAH, multiple cisternal SAH and hydrocephalus are highly suggestive of aneurysmal origin<sup>1,3,6,7</sup>. It is still in question what caused the bleeding in this case. After the rebleeding and CT angiography, the author thought the aneurysmal dilatation which was not visualized on initial CT angiography caused the bleeding. But after confirming the aneurysm as a pseudoaneurysm, the aneurysmal dilatation was considered to be not the cause but the result of the bleeding.

We re-examined the initial CT and digital angiography, which showed minute dilatation and occlusion of the PCA, suggesting an arterial dissection. PCA dissecting aneurysms have been rarely reported, often triggered by sudden head motion<sup>2</sup>. Most of dissecting aneurysms have no recognizable cause. Syphilis, migraine, cystic medial necrosis, fibromuscular dysplasia, connective tissue disease and trauma are known to be the underlying causes. The most common site of dissection was reported to be at P1-P2 segment, bordering a free edge of tentorium suggesting that trauma play a role<sup>5</sup>. But in this case, she had no recognizable underlying cause and the operative finding was not consistent with a dissecting aneurysm. It was speculated that any one of an arterial dissection, a small malformed vessel or a microaneurysm caused the bleeding to completely destroy the lesion initially and rebleeding produced a pseudoaneurysm which appeared after the rebleeding.

## Conclusion

If spontaneous subarachnoid hemorrhage is highly suggestive of aneurysmal origin, the angiogram should be scrutinized by magnified and/or 3D view before the planning of repeated angiography to prevent rebleeding.

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