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Case Report

# *De novo* Aneurysm Formation on Internal Carotid Artery at Origin of Thick Posterior Communicating Artery: 7 Years after Transient Occlusion of Contralateral Internal Carotid Artery


## Abstract

The incidence of *de novo* intracranial aneurysm formation has been reported to be 0.84% per year. It is rare for *de novo* aneurysm formation to be observed on serial radiological examinations. A 64-year-old male with a history of right internal carotid artery (ICA) occlusion 7 years ago had subarachnoid hemorrhage (SAH) due to a ruptured left ICA aneurysm at the bifurcation of the posterior communicating artery (PComA). At the time of ICA occlusion, the left PComA was thick, about 3.0 mm in diameter, and no aneurysm was detected on radiological examinations. Thirty-eight months later, a small aneurysm was detected on the left ICA on magnetic resonance angiography (MRA). At the onset of SAH, the aneurysm was larger than that observed on the previous MRA. Left frontotemporal craniotomy was performed, and the aneurysm was clipped. A thick PComA might contribute to the development of an aneurysm at its origin due to hemodynamic stress. Persistent hemodynamic stress may cause enlargement of an aneurysm in 4 years and its subsequent rupture. In patient with a thick PComA, close observation is necessary to screen for *de novo* formation of a cerebral aneurysm.


**Keywords:** *Cerebral aneurysm, de novo, internal carotid artery, posterior communicating artery, thick*


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## Introduction

Although the *de novo* formation of a cerebral aneurysm is well known, it is not common to observe it on radiological examinations. Risk factors of *de novo* aneurysm formation are age, female sex, smoking, and hypertension.[1,2] Recently, we treated a patient with a ruptured aneurysm of the internal carotid artery (ICA) at the bifurcation of the posterior communicating artery (PComA). The PComA was thick on angiography. In this patient, the formation of a new cerebral aneurysm was detected on magnetic resonance imaging (MRI) a few years after the initial radiological examination. Subsequently, growth and rupture of an aneurysm were observed. In this report, we describe our experience of treatment for a patient with a *de novo* IC-PComA aneurysm accompanied by a thick PComA. We discuss the radiological findings and etiology of a *de novo* aneurysm.

## Case Report

### Past history

A 57-year-old male with a history of hypertension and smoking habit suffered from cerebral infarction of the right cerebrum due to ICA occlusion. He showed consciousness disturbance, left hemiparesis, and dysarthria. Angiography and magnetic resonance angiography (MRA) demonstrated the occlusion of the right ICA. The blood flow in the right ICA territory was supplied through the anterior communicating artery and PComA. The left PComA was thick, with a diameter of about 3.0 mm [Figure 1a]. However, no aneurysmal protrusion was detected in the left ICA on either angiography or MRA [Figure 1b]. Single-photon emission computed tomography (CT) revealed decreased cerebral blood flow in the right cerebrum. His symptoms were gradually improved by medication. He was discharged on the 16th day. Recanalization of the right ICA was confirmed on MRI



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obtained 5 months later, and no aneurysm was detected on the left ICA [Figure 1c]. MRA after 8 months showed mild stenosis of the right ICA in the neck [Figure 1d]. MRA after 9 months also showed no cerebral aneurysm on the left ICA [Figure 1e]. MRA obtained 38 months after cerebral infarction showed a small protrusion on the left ICA at the origin of the PComA. The size of the protrusion was about 2 mm. The left PComA was thick, as observed on the initial radiological examinations [Figure 1f]. For 46 months after the detection of a small aneurysm, radiological examinations to observe an aneurysm were not performed.

### Present illness

Seven years after cerebral infarction, at the age of 64 years old, he suddenly developed consciousness disturbance. CT revealed subarachnoid hemorrhage [Figure 2a]. Angiography demonstrated a left IC-PComA aneurysm [Figure 2b]. The aneurysm became larger than that observed on MRA 4 years ago, at 4.3 mm × 3 mm × 3 mm. Left frontotemporal craniotomy was performed, and the aneurysm was clipped. During the operation, the thick PComA was observed running toward the medial side [Figure 2c]. The aneurysm tip was incised and pathologically examined, revealing that there were red blood cells, platelets, and fibrin; however, there were no vascular components, suggesting thrombus at the tip of the aneurysm. Postoperative angiography demonstrated complete clipping of the aneurysm [Figure 2d]. Symptoms due to vasospasm were not noted. On the 23rd day, the right ventriculoperitoneal shunt was performed. He was discharged on the 56th day without neurological deficits.

## Discussion

The annual incidence of *de novo* intracranial aneurysm formation has been reported to be 0.84%.[1] As an acquired risk factor of *de novo* aneurysms, therapeutic parent artery occlusion has been described.[3] In such a case, hemodynamic stress loaded on the parent artery may induce aneurysm formation. The period between occlusion and the detection of a *de novo* aneurysm is 2–20 (average: 9.1) years.[3]

On the other hand, spontaneous *de novo* aneurysms have also been reported, and their frequency is low compared with that in cases with parent artery occlusion.[4] Factors related to spontaneous *de novo* aneurysm formation include a genetic factor,[5,6] hemodynamic stress,[2] a tiny bleb,[7] an infundibular dilatation,[8] and atherosclerosis.[9] In addition, hypertension and smoking are independent risk factors.[6] Karekezi *et al*.[8] reported a case of infundibular dilatation of the PComA that led to a saccular aneurysm at the origin of the anterior choroidal artery on the same side. Although the location of the aneurysm is different from PComA infundibular dilatation, there is a possibility that a *de novo* aneurysm may develop at a site close to infundibular dilatation. Sámano *et al*.[9] reported a case of *de novo* IC-PComA aneurysm with atherosclerotic stenosis in the proximal ICA. They suggested that atherosclerotic ICA led to decreased antegrade blood flow in the ICA and subsequent reversal flow through the PComA. These conditions loaded hemodynamic stress on the region of the PComA origin and resulted in the new formation of an aneurysm at the IC-PComA junction. They reported that even atherosclerotic stenosis could be a risk factor of

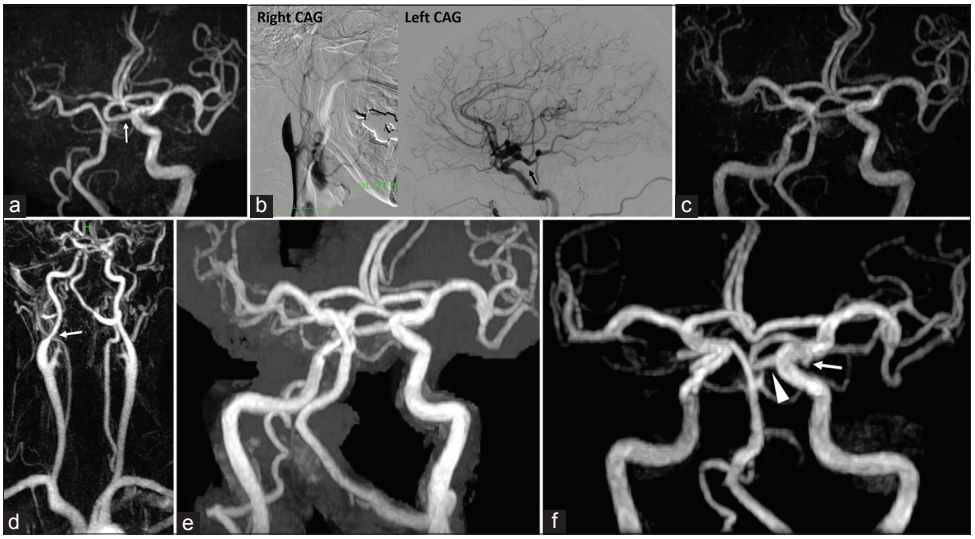


**Figure 1: (a) Magnetic resonance angiography performed 7 years ago showing occlusion of the right internal carotid artery and a thick posterior communicating artery on the left (arrow). (b) Angiography showing occlusion of the right internal carotid artery and thick left posterior communicating artery (arrow). No aneurysmal protrusion can be seen in the left internal carotid artery. (c) Magnetic resonance imaging in the 5th month showing recanalization of the right internal carotid artery, and no aneurysm on the left internal carotid artery. (d) Magnetic resonance angiography after 8 months showing mild stenosis at the right internal carotid artery in the neck (arrow). (e) Magnetic resonance angiography after 9 months showing no cerebral aneurysm on the left internal carotid artery. (f) Magnetic resonance angiography obtained 38 months after cerebral infarction showing a small protrusion (arrow) on the left internal carotid artery at the bifurcation of the thick posterior communicating artery (arrowhead)**

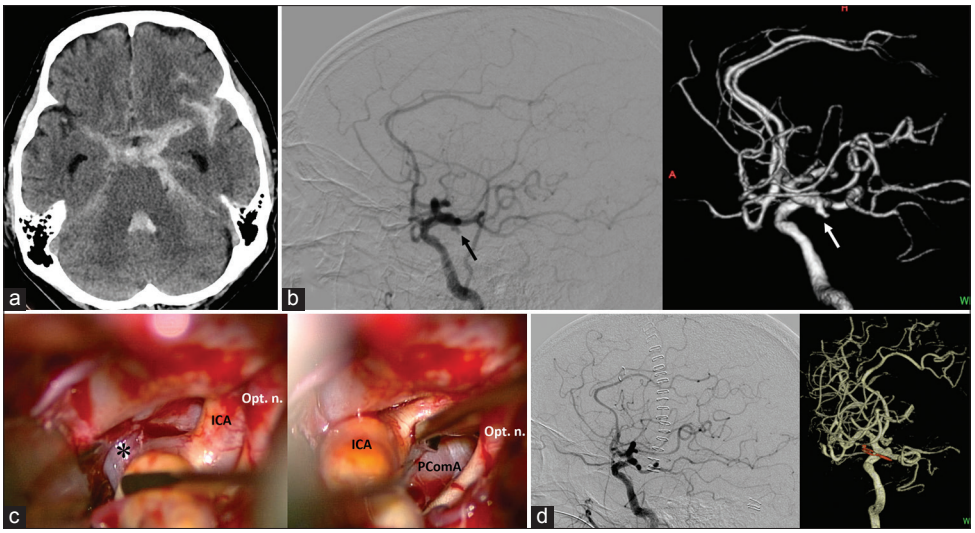


**Figure 2: (a) Computed tomography obtained 7 years after transient right internal carotid artery occlusion revealing subarachnoid hemorrhage. (b) Angiography demonstrating a left internal carotid-posterior communicating artery aneurysm (arrow). (c) Intraoperative photograph showing the aneurysm (asterisk) and the thick posterior communicating artery running toward the medial side. (d) Postoperative angiography demonstrating complete clipping of the internal carotid-posterior communicating artery aneurysm**

*de novo* aneurysm formation. Yang *et al*.[7] reported three cases of *de novo* aneurysm development from a tiny bleb on junctional dilatation at the PComA in 5 years. They concluded that such a tiny bleb could develop into an aneurysm, especially on the dominant side of the PComA.

Hemodynamic stress at the location of the PComA origin might be the main cause of aneurysm formation at this site. Even the asymmetric condition of the circle of Willis is a risk factor of aneurysm development.[10,11] The PComA was thick, about 3.0 mm in diameter, in our case. Furthermore, the left ICA showed atherosclerotic changes. According to Avci *et al*.,[12] the average diameter of the PComA was 1.8 mm on MRI and 1.7 mm on cadaveric examinations. The diameter varied from 0.5 to 3.03 mm on cadaveric examinations. Can *et al*.[13] examined the parameters associated with PComA aneurysms. They reported that a larger PComA diameter was significantly correlated with the presence of a PComA aneurysm. They concluded that a large PComA may have a significant effect on parent artery hemodynamics and subsequent aneurysm development. In our case, the PComA was thick, which might contribute to the development of an aneurysm at its origin due to hemodynamic stress. Persistent hemodynamic stress induced the enlargement of the aneurysm and subsequent rupture. However, we encountered cases with a thick PComA without an aneurysm at its origin. Therefore, in addition to a thick PComA, other factors such as hypertension or smoking might be prerequisites to form a new aneurysm.

van der Schaaf *et al*.[14] reported that *de novo* aneurysms were mainly <5 mm (95%) and located most frequently at the middle cerebral artery (63%). On the other hand, aneurysms visible retrospectively were most frequent in the PComA (21%). They also reported that there was no relationship between the development of *de novo* aneurysms or enlargement and the duration of follow-up or between enlargement and the initial aneurysmal size.[14] In our case, the patient suffered from cerebral infarction due to transient occlusion of the right ICA 7 years ago. Aneurysm or vascular irregularity was not detected in the contralateral left ICA on the initial angiography. Thirty-eight months later, a small bleb was detected at the origin of the thick left PComA. In 4 years, the bleb enlarged and finally ruptured. Although ICA occlusion did not persist on the contralateral right side, some blood flow alteration might be induced after transient ICA occlusion. Hemodynamic stress might be loaded on the left ICA combined with thick PComA. There is a possibility that transient occlusion of the ICA and residual mild ICA stenosis may have effects on blood flow changes in the contralateral ICA.

## Conclusion

In our patient with a *de novo* IC-PComA aneurysm, the thick PComA might have contributed to *de novo* aneurysm formation at its origin. In patients with a thick PComA, serial radiological observation is necessary to screen for *de novo* aneurysm formation.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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## Conflicts of interest

There are no conflicts of interest.

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