

## Thrombosed Giant “True” Posterior Communicating Artery Aneurysm Treated by Trapping and Thrombectomy

### Abstract

Giant “true” posterior communicating artery (PCOM) aneurysms are rare and the best surgical treatment for them is unclear. We present a case of 85-year-old woman with this type of lesion, 35 mm in diameter, successfully treated by trapping and thrombectomy via pterional approach without complications. There were no perforating arteries originating from the aneurysmal wall. The patient had an uneventful postoperative course. The key for successful treatment for such lesions is preservation of perforators, as trapping may result in ischemic complications. However, our case indicates that trapping and thrombectomy might have relatively low risks for development of such complications, supposing that the thrombosis within the giant “true” PCOM aneurysm induced spontaneous obliteration of perforators, arising from the aneurysmal dome, and that collateral flow from the posterior cerebral artery already compensated the corresponding territories.

**Keywords:** Giant aneurysm, surgical complications, thrombectomy, trapping, true posterior communicating artery aneurysm

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### Introduction

A “true” posterior communicating artery (PCOM) aneurysm is considered to completely originate from the PCOM. Its prevalence has been reported to be 1.3% of all intracranial aneurysms.<sup>[1]</sup> “True” PCOM aneurysms are relatively rare, and only three of them have been reported as giant;<sup>[2,3]</sup> therefore, their features and the best surgical treatment are still unclear. A surgical case of progressively enlarging thrombosed giant PCOM aneurysm treated by trapping and thrombectomy without any sequelae is described in the present report.

### Case Report

An 85-year-old woman was diagnosed with a left asymptomatic PCOM aneurysm during a prophylactic brain evaluation by magnetic resonance imaging (MRI) 2 years ago. She had a medical history of hypertension and mild emphysema. Angiography revealed a 12-mm diameter aneurysm originated from the PCOM [Figure 1a]. Because of her age, follow-up was recommended to her. The aneurysm, however, enlarged to be 30 mm in diameter in the subsequent 2 years and compressed the brainstem and thalamus with the contained thrombotic material [Figure 1b]. Clinical deficits

progressed to right hemiparesis and left oculomotor nerve palsy, approximately 5 months before admission. Moreover, deterioration of the level of consciousness occurred 1 month before admission. She was referred to our institution for surgical treatment of the aneurysm when the lesion reached 35 mm in diameter as seen on MRI [Figure 1c].

On admission, she had an impaired level of consciousness (Glasgow coma scale [GCS] E4V2M5 = 11), left total oculomotor nerve palsy, and right hemiparesis (3/5 manual muscle testing [MMT]). Oral intake was not possible and she was completely bedridden (score of 5 on the modified rankin scale [mRS]). Three-dimensional computed tomography (3D-CT) angiography revealed that the giant aneurysm originated from the PCOM, almost obscuring this structure; it had blebs and was directed ventrally and superiorly [Figure 1d].

A left frontotemporal craniotomy was created. The Sylvian fissure was opened wide. Sylvian veins were dissected from the temporal lobe, and the temporal tip was retracted posteriorly. The aneurysm was approached and its origin only from the PCOM was confirmed. The oculomotor nerve was severely displaced medially. The midbrain was also severely

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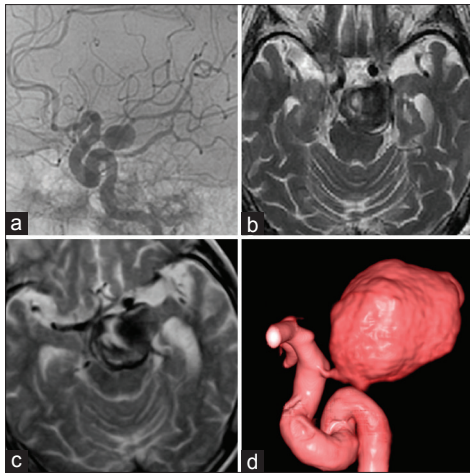
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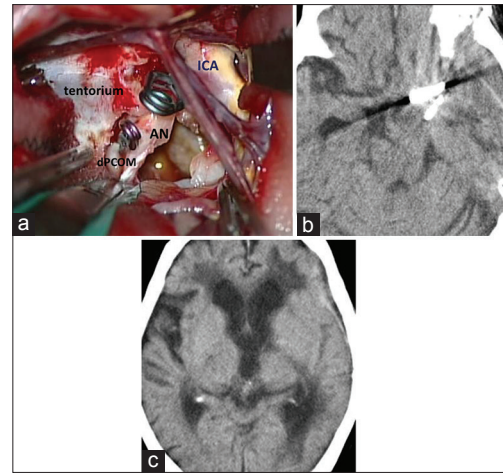
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**Figure 1:** (a) Angiography 4 months after diagnosis shows the aneurysm to be 12 mm in diameter, originating from the posterior communicating artery. (b) Magnetic resonance imaging 2 years and 3 months after diagnosis shows the aneurysm with thrombus enlarging to be 30 mm in diameter and compressing the brainstem. (c and d) The lesion expanded in addition to 35 mm in diameter on magnetic resonance imaging (c) and Three-dimensional computed tomography angiography. Six months later (d) the aneurysm had expanded superiorly from the posterior communicating artery. The right posterior cerebral artery was not visible

compressed. Vasa vasorum were not present on the aneurysmal surface, and there were no perforating arteries originating from the aneurysmal wall. The origin of the PCOM was detected by retraction of the anterior part of temporal lobe, facilitating placement of the proximal clip. The aneurysm was located medially to the tent and the distal part of the PCOM (dPCOM) running behind the tent was not initially observed because it was severely compressed inferiorly by the aneurysm. After placement of the proximal clip at the origin of the PCOM, the tent was cut and the dPCOM from the aneurysm could be seen behind the tent. The other trapping clip was placed just distal to the aneurysm on the PCOM. The aneurysmal dome was opened and thrombectomy was performed [Figure 2a]. The thrombus was relatively soft and easy to remove completely. The anterior choroidal artery could be dissected away from the aneurysmal wall. The aneurysmal wall was incompletely resected because of severe adhesion to the brainstem. During surgery, sensory- and motor-evoked potentials were stable. Postoperative CT confirmed brainstem decompression without ischemic lesions [Figure 2b and c].

The patient had an uneventful postoperative course. Complete aneurysm exclusion was confirmed on 3D-CT angiography 2 weeks after operation. The patient's neurological condition improved, with consciousness recovering to GCS 15 and right hemiparesis decreasing to 4/5 MMT. She could walk and eat with assistance 1 month after operation. The preoperative complete oculomotor nerve palsy improved partially. The Glasgow outcome scale was 3 and mRS improved from 5 to 4 at discharge 1 month after operation.



**Figure 2:** (a) Surgical view via left trans-Sylvian approach. The aneurysm was trapped and the dome was opened with complete thrombectomy. Vasa vasorum around the aneurysm were not detected. ICA – Internal carotid artery; AN – Aneurysm; dPCOM – Distal part of posterior communicating artery; (b and c) postoperative computed tomography shows decompression of the brainstem and no ischemic lesions

## Discussion

The risk of rupture in cases of giant internal carotid (IC)-PCOM aneurysms is very high.<sup>[4]</sup> The risks in giant “true” PCOM aneurysms have not been precisely described; however, if these are similar to that in IC-PCOM aneurysms, a giant “true” PCOM aneurysm should be considered very dangerous. In the present case, although the patient was of advanced age (85-year-old), the decision to operate was based on the rupture risk in addition to her progressively deteriorating severe neurological deficits secondary to mass effects of the aneurysm.

In cases of thrombosed giant aneurysms, it is expected that complete occlusion with thrombectomy might be useful to relieve the existence of severe mass effects.<sup>[5]</sup> In the present case, thrombectomy was performed because of the severe preoperative neurological deficit due to the mass effect by the aneurysm on the brainstem. Complications due to perforators were not observed and neurological symptoms subsided after operation.

In cases of “true” PCOM aneurysms, preserving the perforators is key to successful treatment because many important branches originate from the PCOM.<sup>[3]</sup> Only two cases of giant true PCOM aneurysm have been reported previously,<sup>[2,3]</sup> and this is the first case in which trapping was performed. Although trapping may not make it possible to preserve perforating branches,<sup>[1]</sup> in the present case, this technique was performed because no patent perforators were detected from the aneurysmal wall. Moreover, it was likely that the thrombosis within giant PCOM aneurysm might have induced spontaneous obliteration of perforators, arising from the aneurysmal dome, and that collateral flow from the posterior cerebral artery might have already compensated the corresponding territories.<sup>[3]</sup>

The present report describes the surgical treatment of a thrombosed giant “true” PCOM aneurysm with severe mass effect to the brainstem. The key for successful treatment is preservation of perforators originating from the PCOM, as trapping may result in ischemic complications. However, with proper micro-anatomical evaluation, trapping and thrombectomy for a thrombosed giant “true” PCOM aneurysm might have relatively low risk for the development of ischemic complications.

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#### **Conflicts of interest**

There are no conflicts of interest.

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