Endovascular treatment of a true posterior communicating artery aneurysm

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Abstract
Background: Posterior communicating artery (PCoA) aneurysms are most commonly located at the junction of the internal carotid artery and the PCoA. “True” PCoA aneurysms, which originate from the PCoA itself, are rarely encountered. Most previously reported cases were treated surgically mainly before the endovascular option became available.

Case Description: A 53-year-old male presented with sudden onset of right hemiparesis and aphasia. Left middle cerebral artery (MCA) stroke was diagnosed. Further studies revealed a 3 mm left PCoA aneurysm arising from the PCoA itself, attached to neither the internal carotid artery nor the posterior cerebral artery. Endovascular treatment was performed and the aneurysm was coiled completely.

Conclusion: Technical advances in endovascular interventional technology have permitted an additional approach to these lesions. The possible endovascular significance of the treatment of true PCoA aneurysms is discussed.

Key Words: Cerebral aneurysm, endovascular coiling, posterior communicating artery aneurysm, true posterior communicating artery aneurysm

INTRODUCTION

Posterior communicating artery (PCoA) aneurysm is one of the most frequent types of intracranial aneurysm, accounting for approximately 25% of all intracranial aneurysms."¹²¹³" Traditionally, PCoA aneurysms arise at the junction of the internal carotid artery (ICA) and the PCoA, at the point where the PCoA originates from the posterolateral aspect of the ICA. These aneurysms are sometimes called junctional aneurysms. A recent meta-analysis of reported cases of true PCoA aneurysms, in which the aneurysm actually arises from the PCoA, estimated that these aneurysms account for 6.8% of all PCoA aneurysms and 1.3% of all intracranial aneurysms.¹⁷ Surgical treatment has traditionally been preferred and reports of endovascular treatment of true PCoA aneurysms are rare. In the present report we describe the case of a true PCoA aneurysm successful treated using endovascular coiling.

CASE REPORT

A 53-year-old, right-handed male patient presented with sudden onset of right hemiparesis and aphasia. Left middle cerebral artery (MCA) stroke was diagnosed. The
patient’s medical history included systemic hypertension and dyslipidemia. Previously undiagnosed atrial fibrillation was then identified. Systemic fibrinolytic therapy was administered first without result; local intraarterial fibrinolytic treatment and mechanical thrombectomy were then performed, achieving recanalization of the distal MCA.

Computed tomography angiography (CTA) that was performed during the diagnosis of the stroke also revealed an aneurysm of the PCoA [Figure 1]. No signs of subarachnoid hemorrhage were observed in the basal cisterns, and the patient had no focal cranial nerve deficits prior to the stroke. The aneurysm measured 3 mm and had a posterior orientation. A day after the stroke, magnetic resonance imaging (MRI) showed a mass in the interpeduncular fossa that was larger than expected [Figure 2]. The mass measured 14 mm, projected medially, and was compatible with a partially thrombosed aneurysm.

Bilateral carotid and left vertebral angiography [Figures 3 and 4] revealed an aneurysm arising from the left PCoA itself. The saccular-type aneurysm originated 3 mm distal to the ICA bifurcation, with a 3.5 mm dome height and a 1.5 mm neck width. A fetal-type posterior cerebral artery (PCA) was present on the left. We opted for endovascular treatment of the aneurysm because the configuration of the aneurysm was favorable in our opinion, rather than surgical clipping. Endovascular occlusion was performed under general anesthesia in a separate procedure. A guide catheter was introduced to the right ICA. Then a microcatheter was placed into the aneurysm sac in the PCoA. Once the microcatheter was placed three coils were packed (3 × 80, 2 × 60, and 2 × 40 mm) until no additional coil could fit into the
lumen. Postcoiling angiogram showed complete occlusion of the aneurysm [Figure 5]. The postprocedure course was uneventful.

At the 6-month follow-up, magnetic resonance angiography (MRA) did not show recanalization. The patient exhibited marked neurological improvement with therapy, presenting with mild right hemiparesis and motor dysphasia with mild nonfluent speech and normal comprehension.

DISCUSSION

Aneurysms originating directly from the PCoA are very unusual. Postmortem observations made by Dandy indicated that aneurysms that were interpreted as PCoA aneurysms based on their angiographical appearance were actually aneurysms of the ICA. True PCoA aneurysms typically arise within 2-3 mm of the origin of the PCoA. The incidence of true PCoA aneurysms is difficult to assess using preoperative angiography because it is challenging to discriminate between this type of aneurysm and junctional aneurysms. The higher incidence rates reported in recent studies may be a result of the increased spatial resolution of modern CTA and digital subtraction angiography (DSA) equipment.

Although the mechanisms by which aneurysms originate, grow and rupture remain controversial, evidence indicates that hemodynamic factors play a significant role. Hemodynamic stress might be important in true PCoA aneurysms because as in this case, these aneurysms are often associated with a dominant PCoA (fetal-type) in which the PCoA is the major blood supply to the PCA, which is consistent with higher flow through this artery. Kaspera et al. reported a true PCoA aneurysm coexisting with an ICA occlusion and documented increased velocity and turbulent blood flow in both communicating arteries, which suggested that the consequent increase in wall shear stress (WSS) might lead to aneurysm formation. In addition, in a CTA-based morphometric analysis of 77 aneurysms of the PCoA complex, including 10 true PCoA aneurysms, He et al. determined the calibers of the ipsilateral PCoA, the P1 segment of the PCA and the P2 segment of the PCA. The analysis revealed that true PCoA aneurysms presented larger PCoA and P2 segments and smaller P1 segments than junctional aneurysms. The observed differences were statistically significant. Further, ruptured junctional aneurysms were statistically larger in size than ruptured true PCoA aneurysms. The authors postulated that true PCoA aneurysms might be more prone to rupture than junctional aneurysms of similar size.

Microsurgical clipping for treating these aneurysms has traditionally been performed. After splitting the Sylvian fissure, the ICA is followed posteriorly to identify the origin of the PCoA. In contrast to traditional junctional aneurysms, which arise on the posterolateral wall of the ICA, true PCoA aneurysms arise in what is typically an intraoperative blind spot 2-3 mm posterior to the origin of the PCoA. In treating these aneurysms, it is critical to preserve the patency of both the PCoA and the 4–14 thalamoperforating arteries that arise from the PCoA. Branches from the PCoA supply the optic chiasm, oculomotor nerve, mammillary body, tuber cinereum, cerebral crura, ventral thalamus, and the rostral portion of the caudate nucleus. Preservation of the artery is important even if it is hypoplastic. As the oculomotor nerve typically runs lateral to the PCoA, preservation of this nerve is another important surgical consideration.

In a recent meta-analysis, He et al. performed a comprehensive review of true PCoA aneurysm cases reported to date. Seventy cases were retrieved. Among these cases, 42.9% occurred in men and 57.1% occurred in women. Information regarding rupture status was only provided in 49 cases; among these cases, 8 were reported as ruptured (89.8%) and 5 were reported as unruptured (10.2%). No significant differences in rupture status were observed with respect to age or to the side or shape of the aneurysm.

The complex anatomy of true PCoA aneurysms with successive sharp branch angles within a short distance may preclude safe endovascular access. Since most of these aneurysms arise within 2-3 mm of the ICA and PCoA junction, successful cannulation of the aneurysm with a microcatheter may prove challenging. These aneurysms have traditionally been treated surgically because it was the only treatment available. Technical advances in endovascular interventional technology have permitted an additional approach to these lesions. Reports of endovascular treatment of true PCoA aneurysms are rare because it is a rare condition per
In a recent review, Almeida-Pérez et al.[2] included only seven cases of true PCoA aneurysms treated using endovascular coiling. This case is the eighth reported case. The location of the neck along the inferior surface of the PCoA is felt to be an unfavorable configuration for endovascular coiling,[6] because the microcatheter must be navigated into the PCoA and directed inferiorly into the aneurysm. At times, the acuity of the approach angle may be too sharp to establish a stable microcatheter position. Balloon-assisted techniques may reduce the risk of possible coil extrusion into the parent vessel.

CONCLUSION

Aneurysms originating directly from the PCoA are rarely encountered. Most previously reported cases were treated surgically, and reported cases of endovascular treatment are scant.

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