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Original Article

"True" posterior communicating aneurysms: Three cases, three strategies

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Abstract

Background: The authors provide a review of true aneurysms of the posterior communicating artery (PCoA). Three cases admitted in our hospital are presented and discussed as follows.

Case Descriptions: First patient is a 51-year-old female presenting with a Fisher II, Hunt-Hess III (headache and confusion) subarachnoid hemorrhage (SAH) from a ruptured true aneurysm of the right PCoA. She underwent a successful ipsilateral pterional craniotomy for aneurysm clipping and was discharged on postoperative day 4 without neurological deficit. Second patient is a 53-year-old female with a Fisher I, Hunt-Hess III (headache, mild hemiparesis) SAH and multiple aneurisms, one from left ophthalmic carotid artery and one (true) from right PCoA. These lesions were approached and successfully treated by a single pterional craniotomy on the left side. The patient was discharged 4 days after surgery, with complete recovery of muscle strength during follow-up. Third patient is a 69-year-old male with a Fisher III, Hunt-Hess III (headache and confusion) SAH, from a true PCoA on the right. He had a left subclavian artery occlusion with flow theft from the right vertebral artery to the left vertebral artery. The patient underwent endovascular treatment with angioplasty and stent placement on the left subclavian artery that resulted in aneurysm occlusion.

Conclusion: In conclusion, despite their seldom occurrence, true PCoA aneurysms can be successfully treated with different strategies.

Key Words: Etiology, physiopathology, treatment, true posterior communicating artery aneurysms

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INTRODUCTION

Twenty-five percent of intracranial aneurysms arise from the internal carotid artery (ICA) at the posterior communicating artery (PCoA) origin, making this site the second most common location after anterior communicating artery (ACoA) aneurysms.[4] True PCoA aneurysms represent about 1.3% of all intracranial

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aneurysms and 6.8% of all PCoA aneurysms.[4] The so-called "true" PCoA aneurysm was first described by Yoshida *et al*.[12] in 1979. He attempted to formalize the nomenclature of aneurysms originating directly from the PCoA, 2–3 mm distal to the junction of ICA with PCoA. The PCoA vascular regional anatomy is treacherous due to the numerous perforators arising from the posterior half of the arterial wall extending from the communicating segment of the carotid artery, that terminate on the optic chiasma and tract, the floor of the third ventricle, the infundibulum, the posterior perforated substance, and the medial temporal lobe.[3,12] Variations of the circle of Willis are well described and could have a primordial role in the development of berry aneurysms along with other risk factors such as vascular genetics, changes in wall shear stress, smoking, hypertension, and gender.[7]

CASE REPORTS

Case report 1

L.M.S is a 51-year-old who experienced a paroxysmal, severe headache along with confusion and presented to a secondary hospital emergency department within a few hours of onset. She had a medical history of poorly controlled hypertension, and a significant smoking history (40 cigarettes/day for 20 years). A computed tomography (CT) scan demonstrated a subarachnoid hemorrhage (SAH) with the preponderance of hemorrhage centered in the right Sylvian fissure. A clinical diagnosis of aneurysmal SAH (Hunt and Hess Grade III, Fisher Grade II) was made.

Four-vessel digital subtraction angiography (DSA) revealed a small, true saccular aneurysm with a fetal-type right PCoA. The aneurysm was 3 mm × 4 mm in size, with a postero-superiorly directed 2 mm neck [Figure 1].

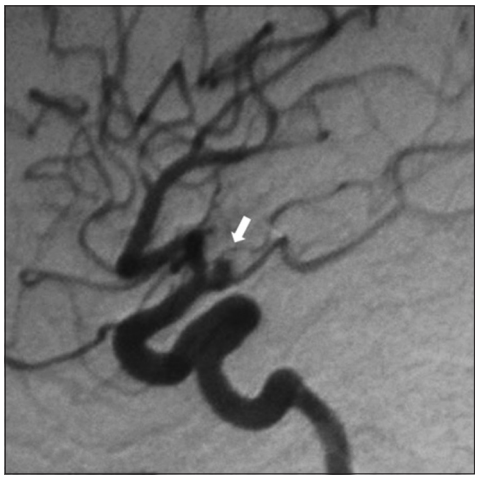

Figure 1: Lateral view of right internal carotid artery angiography performed on June 18, 2012. Small true saccular aneurysm (white arrow) of the right posterior communicating artery, 3 mm × 4 mm size, neck diameter of 2 mm, postero-superiorly directed, and fetal pattern of ipsilateral posterior communicating artery

The angiogram also revealed a hypoplasic A1 segment of the right anterior cerebral artery. She was transferred to our hospital 30 days after her initial presentation, and her neurological exam was nonfocal with mild meningismus (Hunt and Hess Grade I). The patient underwent an uneventful and successful microsurgical clipping of the saccular aneurysm through a pterional approach [Figure 2]. The patient developed a postoperative mild paresis of the ipsilateral oculomotor nerve, which resolved by postoperative day 3. A postoperative head CT revealed no obvious complications. The patient was discharged from the hospital 4 days after surgery without any neurological deficits.

Case report 2

M.C.S., a 53-year-old female, with a previous history of hypertension and smoke (20 cigarettes/day for 30 years), presented a headache of moderate intensity for 5 days, in the occiptocervical region, partially responsive to nonsteroidal anti-inflammatory drugs, followed by a sudden increase in pain intensity, along with right-sided hemiparesis, when she was admitted in a secondary hospital. The head CT was normal, but a lumbar puncture demonstrated SAH (Hunt and Hess Grade III, Fisher Grade I). The four-vessel DSA then revealed a true saccular aneurysm of the right PCoA, 6 mm × 3 mm in size, inferiorly oriented, with a 2 mm neck, and a saccular aneurysm of the left ophthalmic segment of ICA, 12 mm × 10 mm in size, superiorly oriented, with a 5 mm neck [Figure 3]. We performed the surgical clipping of both lesions with a single pterional approach, on the left side, including an intradural clinoidectomy [Figure 4]. Postoperative physical examination and CT scan revealed no further complications and she was discharged 4 days after the surgery with complete recovery of muscle strength during follow-up.

Case report 3

A 69-year-old male patient presented to a secondary hospital emergency department with a sudden headache followed by momentary loss of conscience and cranial nerve paresis. After 18 days of the initial symptoms, he was transferred to our hospital. By physical examination after admission, the patient presented with slight headache and neck stiffness, blood pressure on left arm of 140 mmHg × 110 mmHg and right arm of 170 mmHg × 100 mmHg. Neurological exam showed a conscious and oriented patient with right oculomotor (III) and trochlear (IV) paresis. The CT scan revealed a Fisher Grade III SAH [Figure 5a]. The subsequent four-vessel DSA revealed a right true PCoA aneurysm [Figure 5b and c], occlusion of the left subclavian artery [Figure 6a], right vertebral artery steel phenomena to the left vertebral artery [Figure 6b] and >80% right ICA stenosis. The therapeutic discussion was based on the fact that the steel was so important that no enhancement of the superior segment of the basilar artery and its

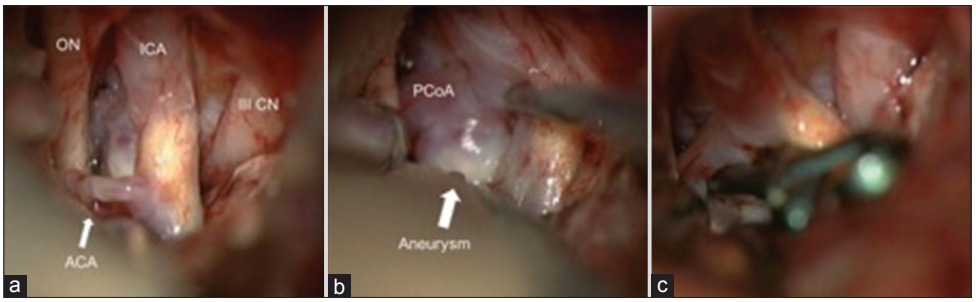

Figure 2: (a) From the left to the right, optic nerve, anterior cerebral artery, internal carotid artery and third cranial nerve. (b) Optic nerve and internal carotid artery slightly retracted to expose the optic carotid triangle with the aneurysm inside it. (c) Clip in the neck of the aneurysm

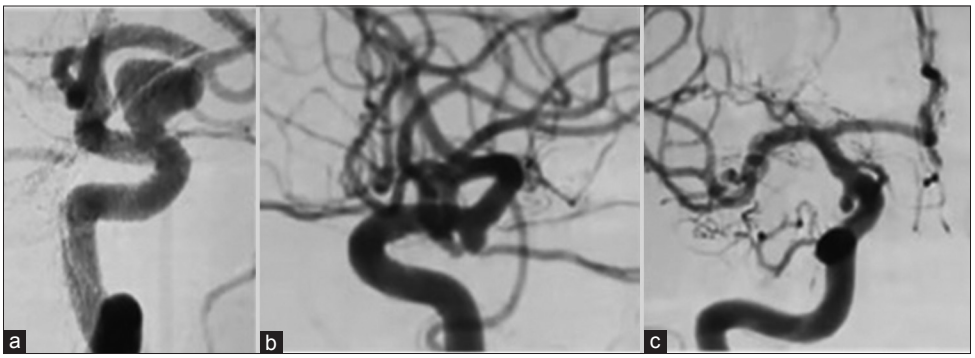

Figure 3: (a) Contralateral oblique view of left internal carotid artery angiography, with a saccular aneurysm of the ophthalmic segment, 12 mm × 10 mm, 5 mm neck. (b) Lateral and (c) anteroposterior views of right internal carotid artery angiography, with a true saccular aneurysm of the posterior communicating artery, 6 mm × 3 mm, 2 mm neck

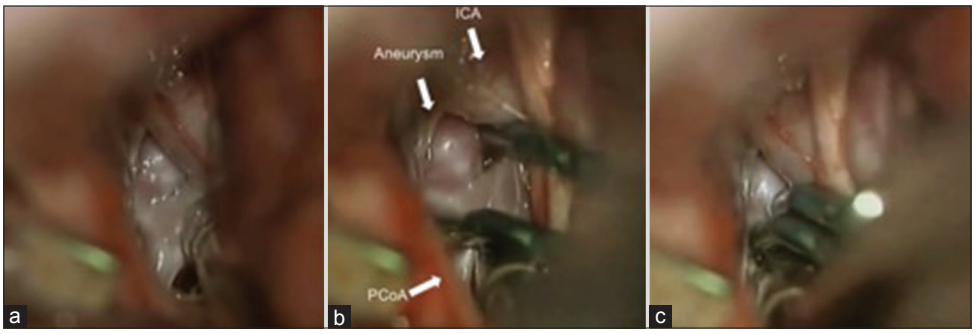

Figure 4: (a) Intraoperative view (pterional contralateral approach) of the posterior communicating artery with its true aneurysm, before clipping. (b) During and (c) after clipping

rami were seen on posterior circulation angiogram, and were mainly supplied by the right PCoA. In this manner we believed that the aneurysm might be related to the high flow through the PCoA, despite the important stenosis in right ICA. Our hypothesis was that normal flow restoration due to correction of the left subclavian artery obstruction would correct the steel phenomenon, diminishing the high flow through the PCoA, with possible aneurysmal exclusion. The patient underwent recanalization and stent placement on the left subclavian artery [Figure 7a], resulting in restoration of flow through the left vertebral artery [Figure 7a]. Full replenishment of the vertebra-basilar circulation and posterior cerebral arteries (PCAs) [Figure 7b] was observed. The flow through the PCoA was lowered and contrast stagnation inside the aneurysm was observed [Figure 7c]. Late control angiogram of the aortic arch and right ICA showing adequate flow through the left subclavian artery and aneurysm's circulation exclusion [Figure 8].

DISCUSSION

True PCoA aneurysms are rare with pooled data revealing that they represent 1.3% (95% confidence interval [CI] 0.8–1.7%) of all intracranial aneurysms, and 6.8% (95% CI 4.3–9.2%) of all PCoA aneurysms.[4] In a recent systematic review and meta-analysis of individual patient data, the mean patient age of symptom onset was 53.5 years, (53.5

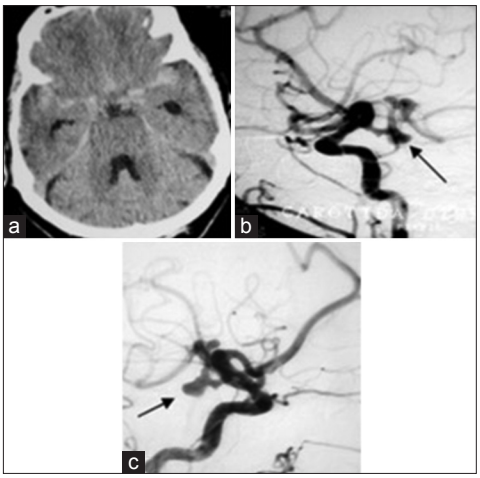

Figure 5: (a) Axial computed tomography scan showing Fisher III subarachnoid hemorrhage. (b and c) Right internal carotid artery angiogram showing the saccular true posterior communicating artery aneurysm (black arrows)

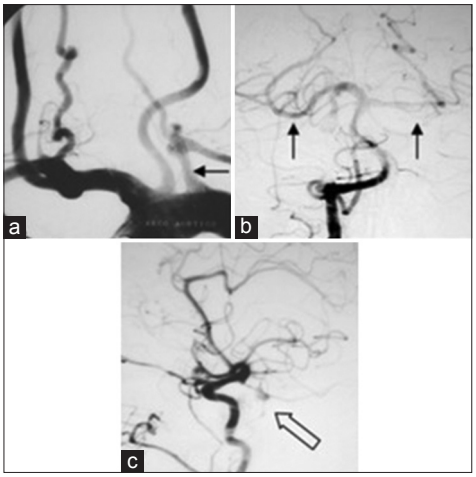

Figure 7: (a) Aortic arch angiogram showing the arterioplasty procedure and stent placing on the left subclavian artery (black arrow). (b) Left vertebral angiogram after arterioplasty with adequate filling of the vertebrobasilar circulation and posterior cerebral arteries. (c) Right internal carotid artery after arterioplasty and diminished flow on the posterior communicating artery with contrast stagnation inside the aneurysm (empty black arrow)

± 15.4 years), and ranged from 23 to 79 years.[4] At the time of this publication, 49 patients with true PCoA have been reported in the literature, with the majority presenting with rupture (89.8%). There have been no significant reported differences in ruptured status between age of occurrence ($P = 0.321$), left versus right aneurysm ($P = 0.537$) and shape of aneurysm ($P = 0.408$).

There is a paucity of literature regarding the pathophysiology of true PCoA aneurysm. Aneurysms of the PCoA can occur at the junction with the ICA, PCA or the proximal PCoA itself.[5] It is well known that the supraclinoid carotid artery, after the emergence of the superior hypophyseal artery, turns upward toward the anterior perforated substance to form a curve that is

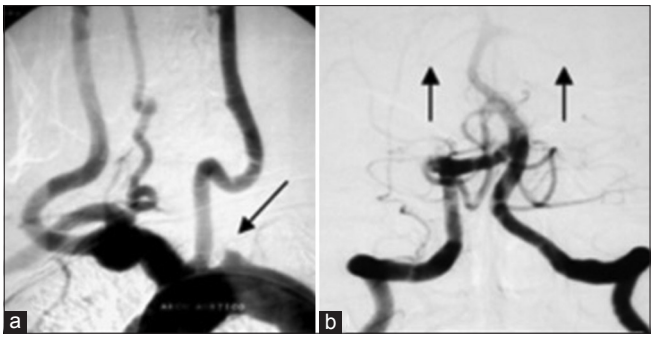

Figure 6: (a) Aortic artery angiogram showing left subclavian artery occlusion (black arrow). (b) Right vertebral artery angiogram showing steel phenomenon from the right vertebral artery to the left vertebral and basilar and posterior cerebral arteries low flow (black arrows)

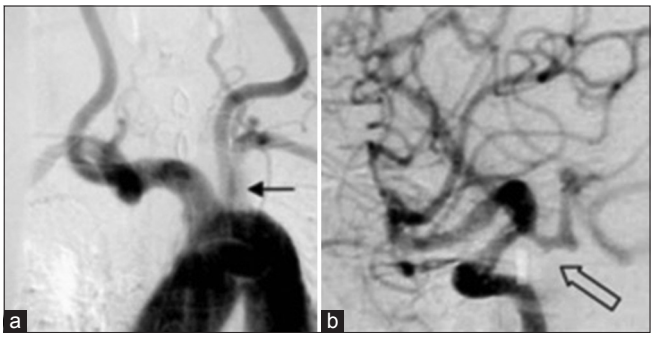

Figure 8: (a) Late control angiogram of the aortic arch and right internal carotid artery showing adequate flow through the left subclavian artery (black arrow). (b) Aneurysm's circulation exclusion

convex posteriorly.[3] The most common variety of carotid artery aneurysm arises at the communicating segment of the ICA.[3] This epidemiology supports the four principles of the operative treatment of intracranial aneurysms: (1) They arise at a branching points on the parent artery; (2) they occur at a curve or turn in the artery; (3) sacullar aneurysms point in the direction of the proximal segment hemodynamic vector; (4) there is a consistent group of perforating arteries situated at each aneurysm site that need to be preserved to achieve an optimal patient outcomes.[3] These aneurysms arise from the posterior wall of the carotid artery near the apex of this turn, immediately above the distal edge of the origin of the PCoA.[12] Hemodynamic stressors contribute to aneurysm formation and may be associated with parent vessel size and aneurysm location.[5] Morphometric analysis has been performed using CT angiography to determine the exact origin of true PCoA aneurysms.[5] In a total of 77 PCoA aneurysms analyzed, 10 (13%) were found to be true PCoA aneurysms. The ipsilateral PCoA/P1 ratio (1.77 ± 0.44 vs. 0.82 ± 0.46, $P = 0.0001$) and ipsilateral P2/P1 ratio (1.73 ± 0.40 vs. 1.22 ± 0.41, $P = 0.0003$) were significantly higher in true PCoA aneurysms, suggesting that true PCoA aneurysms have a larger PCoA relative to the ipsilateral P1 segment. Junctional aneurysms had a larger size than true PCoA aneurysms ($P = 0.03$), but the

rupture rate was not statistically different between both groups (~80%, *P* = not significant). This suggests that true PCoA aneurysms might be more prone to rupture than junctional aneurysms, although needs further analysis to confirm those conclusions.

It is well known that hemodynamic alterations within the ICA from either anatomic/pathologic hypoplastic segments or iatrogenic occlusion can directly influence the development of intracranial aneurysms.[1,6,11] There is also evidence that "de novo" aneurysms can arise in the ACoA or contralateral ICA after coiling or clipping of intracranial aneurysms.[2,10] Kaspera *et al*.[8] demonstrated, through Doppler ultrasonography, that there is both increased velocity and turbulence of blood flow in PCoA when patients are submitted to ICA occlusion. These can subsequently lead to the development of true PCoA aneurysms.

Microsurgical understanding of this unique anatomy is also essential for minimizing morbidity associated with surgical clipping. It is critical to note that for true PCoA aneurysms, the neck arises distal to the origin of the PCoA, and therefore resides in what is traditionally considered an intra-operative "blind spot." The PCoA must be followed posteriorly to visualize the aneurysm neck for microsurgical clipping.[9]

CONCLUSION

In conclusion, true PCoA aneurysms are rare. Much is still unclear regarding its pathophysiology. Preoperative anatomical understanding and microsurgical facility are paramount for treatment, and special consideration must be paid to the perforating arteries. The seldom occurrence of such lesion does not imply that treatment will have a poor outcome, and different strategies may be used.

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Conflicts of interest
There are no conflicts of interest.

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