Unilateral infundibular origin of a fetal posterior communicating artery, associated with an unruptured fusiform aneurysm of the terminal segment of internal carotid artery. A case report

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SUMMARY

Fetal-type posterior communicating artery (PComA) is considered when it serves as a dominant source of blood supply to the distal segment of the posterior cerebral artery (PCA) instead of being supplied as usual from the basilar arterial system. The infundibular origin of the PComA from the internal carotid artery (ICA) is a commonly observed anatomical variant, which has important implications for cerebrovascular dynamics. Infundibular dilatation may be complicated by a saccular aneurysm. Nevertheless, we present for the first time a case of infundibular origin of a right PComA of a fetal type, associated with a fusiform aneurysm of the terminal segment of the ICA. We hypothesize that the infundibular origin of PComA occurred due to the persistence of the fetal pattern of PComA and that it was a predisposing factor for the development of a fusiform aneurysm at the terminal segment of ICA. We recommend follow-up MRI for infundibula because of the high risk of their evolution into true aneurysms.

Key words: Fetal posterior communicating artery – Infundibular origin – Internal carotid artery – Fusiform aneurysms – Saccular aneurysm – Anatomical variations

INTRODUCTION

The posterior communicating artery (PComA) arises from the terminal segment of the internal carotid artery (ICA), and connects ICA to the posterior cerebral artery (PCA) thus linking the anterior and posterior parts of the circle of Willis (CoW). The diameter of PComA normally ranges between 0.3-1.6 mm (Foreman et al., 2016).

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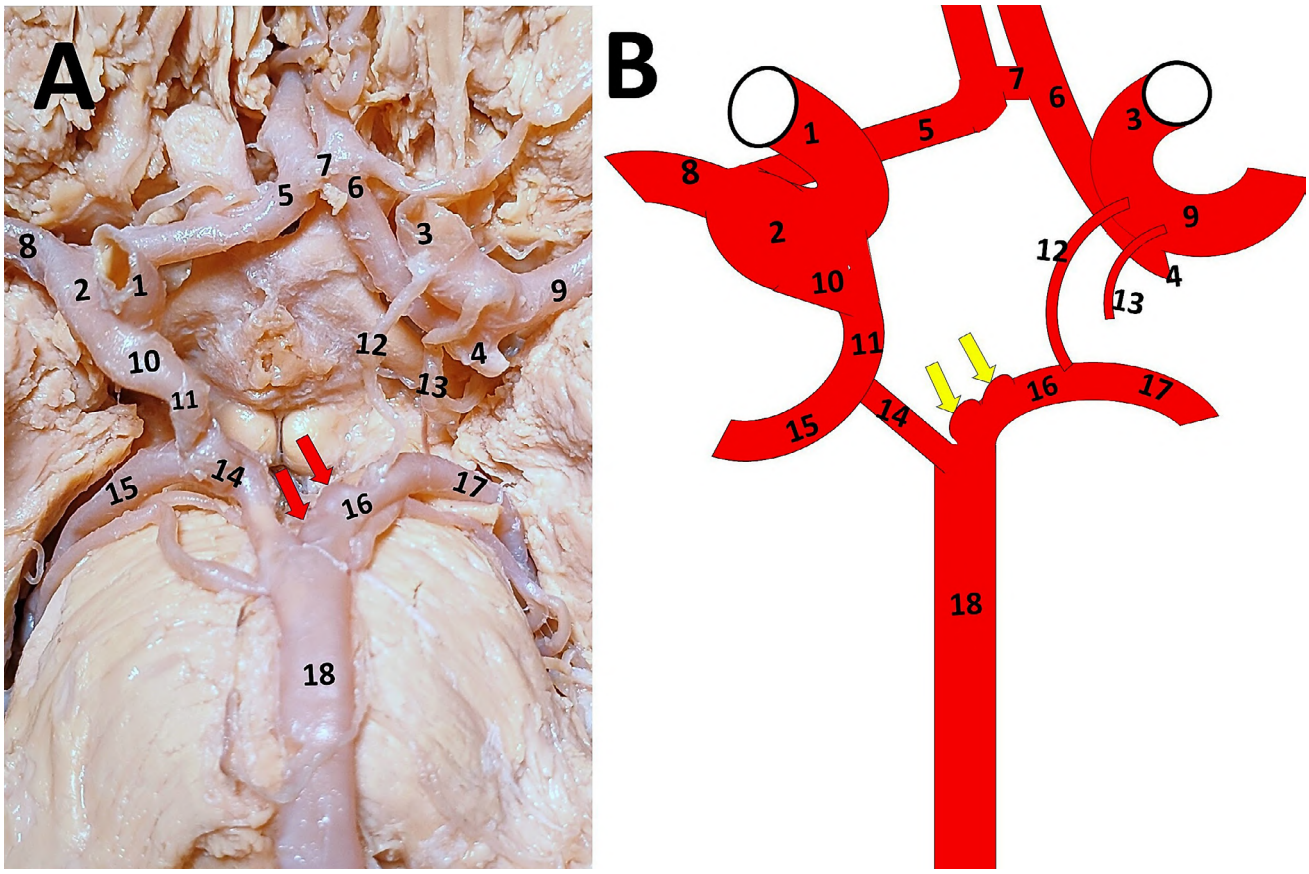

Fig. 1.- A) Dissected circle of Willis of the presented case. **B)** Diagrammatic illustration of the case. 1. Right internal carotid artery, 2. Fusiform dilatation of right internal carotid artery, 3. Left internal carotid artery, 4. Small berry aneurysm at the left internal carotid artery, 5. Right anterior cerebral artery, 6. Left anterior cerebral artery, 7. Anterior communicating artery, 8. Right middle cerebral artery, 9. Left middle cerebral artery, 10. Infundibular origin of the right posterior communicating artery, 11. Right posterior communicating artery (Fetal variant), 12. Hypoplastic left posterior communicating artery (Classic type), 13. Anterior choroidal artery, 14. P1 segment of right Posterior cerebral artery, 15. P2 segment of right posterior cerebral artery, 16. P1 segment of left posterior cerebral artery, 17. P2 segment of left posterior cerebral artery, 18. Basilar artery. Two berry aneurysms in P1 segment of left PCA are marked by the arrows.

During embryonic development, if the proximal (P1) segment of PCA is absent or hypoplastic, the PComA persists and serves as the dominant source of blood supply to the PCA territory. This variant is termed a fetal PComA (FPComA) (Elzawawy et al., 2024). In a recent study, the incidence of FPCA was 5.6% (Bhanu et al., 2020).

Anatomical Variations in CoW can affect hemodynamics causing abnormal flow patterns or turbulence leading to aneurysm formation. They can impact the location and type of aneurysmal transformation, may alter the approach to aneurysm treatment, and can cause ischemic stroke. Identifying anatomical variations requires detailed imaging, such as MRA angiography, and CT angiography. Interpreting these images correctly can be challenging, particularly in distinguishing between normal variations and pathological changes.

The PComA frequently shows an ampullary dilation at its origin from the ICA, known as an infundibulum with a 6% - 25% incidence rate (Satoh et al., 2006). It is sometimes misdiagnosed as a distal ICA saccular aneurysm (PComA aneurysm) (San Millán Ruiz et al., 2020). Some authors consider the infundibulum as a benign anatomical variant (Tarkiainen et al., 2024) while others report the transformation of the infundibulum into a saccular aneurysm (Fischer et al., 2011). However, to the best of our knowledge, no previous study reported the evolution of an infundibular origin of PComA into a fusiform dilatation at the terminal part of ICA.

CASE REPORT

A rare cerebrovascular variation in the form of an infundibular origin of a right FPComA, associated with an unruptured fusiform aneurysm of the terminal communicating (C7) segment of the ICA

was demonstrated during a cadaveric dissection (Fig. 1). The ophthalmic (C6) segment of the right ICA showed a diameter of 3.65 mm, whereas the communicating (C7) segment of the ICA showed a fusiform dilatation with a diameter of 5.25 mm and a length of 8.84 mm. The right PComA showed an infundibulum at its origin from the right ICA with a large diameter of 3.55 mm at its base. The right PComA was large in diameter (2.71 mm) and had a distal attachment to the junction between small size P1 (1.74 mm) and large size P2 (2.65 mm) segments of the PCA. The right MCA, right ACA P1 segment, and P2 segment showed average diameters of 3.30 mm, 2.21 mm & 2.95 mm, respectively. On the left side, the diameter of ICA was average (3.72 mm) with a small berry aneurysm near its termination. Left MCA, ACA P1 segment, and P2 segment showed average diameters of 3.11 mm, 2.81 mm & 2.85 mm, respectively. The left PComA was hypoplastic (0.65 mm in diameter). The P1 segment of the left PCA had a diameter of 3.00 mm and showed two berry aneurysms on its anterior aspect; the proximal one had a diameter of 3.54 mm while the distal one had a diameter of 3.63 mm. The P2 segment of the left PCA was of average diameter (2.61 mm) and didn't show any aneurysmal dilations. The basilar artery (BA) had an average diameter (4.13 mm) with no associated variation in BA, vertebral, or cerebellar arteries. Table 1 shows a summary of the diameters of the brain arteries of the present case.

Table 1. The diameters of the brain arteries in millimeters.

	Right	**Left**
ICA ophthalmic (C6) segment	3.65	3.72
ICA Aneurysm:	5.25	
PComA Infundibulum	3.55	
PComA	2.71	0.65
PCA P1 segment	1.74	3.00
PCA P2 segment	2.65	2.61
PCA P1 segment aneurysms: Proximal one Distal one		 3.54 3.63
MCA	3.30	3.11
ACA P1 segment	2.21	2.81
ACA P2 segment	2.95	2.85

DISCUSSION

We present a case of infundibular origin of a right FPComA, associated with a fusiform aneurysm of the terminal (C7) segment of right ICA. To the best of our knowledge, this is the first report of such a case.

In the developing embryo, the PCA arises from ICA. Later on, the blood supply from ICA becomes insufficient to support the enlarging hindbrain, and PCA makes anastomosis with the termination of BA. At later stages, the proximal portion of embryonic PCA thins out and forms the adult PComA, thus BA becomes the source of supply to PCA. If this hemodynamic shift does not take place, PComA does not regress but persists as a fetal type in adult (Coulier, 2018). The diameter of PComA in adults may be small (< 1mm), moderate (1-2 mm), or large (>2 mm) (Jones et al., 2021). In the present case, the left PComA was hypoplastic (0.65 mm in diameter) confirming a classical type, whereas the right PComA was large in diameter (2.71 mm) as compared to the P1 segment (1.74 mm) of right PCA confirming the partial fetal-pattern (Coulier, 2018).

The right PComA had an infundibular dilatation at its origin. It is sometimes misdiagnosed as a distal ICA saccular aneurysm (PComA aneurysm). Distinguishing features include a diameter of 3 mm or less, and a smooth ampullary shape with a PComA arising from its apex and without an aneurysmal neck (San Millán Ruiz et al., 2020). In the present case, although having a large diameter (3.55 mm) at its base, the infundibulum was smooth without a neck and the PComA originated from its apex (Fig. 2). Yuan et al. (2020) defined certain hemodynamic and morphological differences between aneurysms and infundibular dilations; they emphasize that the shape of the dilation is more reliable than the size. Some authors believe that overstretching due to hemodynamic factors such as hypertension can cause defects in the elastic fibers of the wall of the infundibula (Karekezi et al., 2014). Other authors reported that an infundibulum may represent an incompletely regressed remnant of previously dominant fetal vessels (Fischer et al., 2011). We hypothesize that the infundibulum occurred due to the persistence of the fetal pattern of PComA.

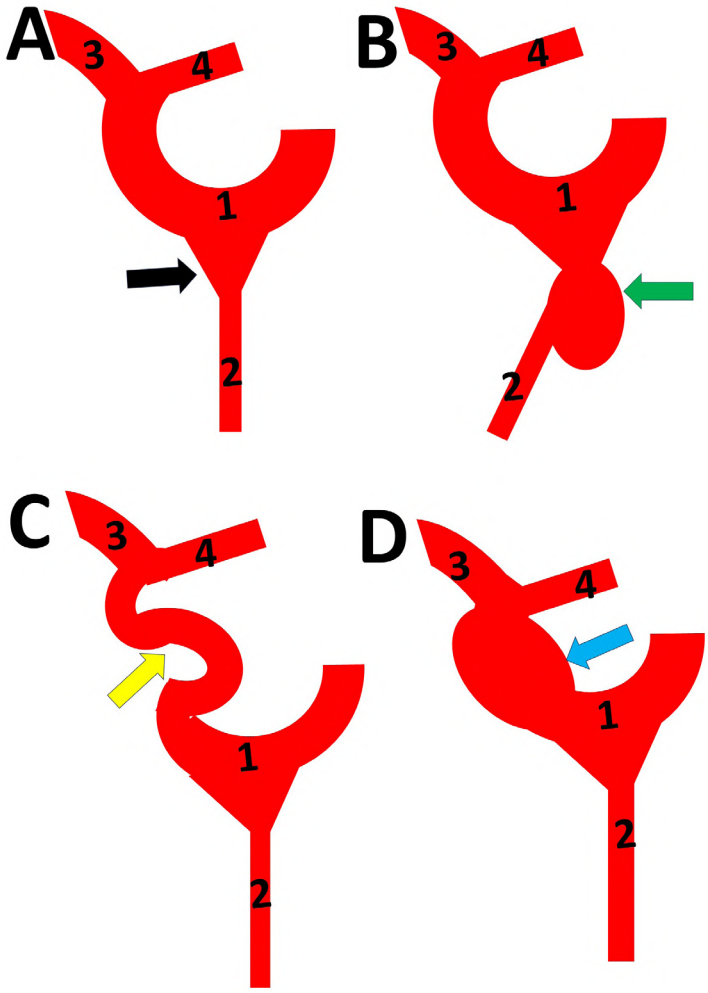

Fig. 2.- Illustrative diagram discriminating infundibulum from a saccular aneurysm, dolichoectasia, and fusiform aneurysm. **A)** Infundibular dilatation of the posterior communicating artery at its origin from the internal carotid artery, highlighting the smooth ampullary shape without an aneurysmal neck, which differentiates it from a saccular aneurysm. **B)** Saccular aneurysm at the origin of posterior communicating artery. **C)** Dolichoectasia of the terminal segment of internal carotid artery. **D)** Fusiform aneurysm of the terminal segment of internal carotid artery, complicating the infundibular origin of posterior communicating artery. 1. Internal carotid artery, 2. Posterior communicating artery, 3. Middle cerebral artery, 4. Anterior cerebral artery. Black arrow: the infundibular origin of the posterior communicating artery, green arrow: saccular aneurysm of the internal carotid artery, yellow arrow: Dolichoectasia of the terminal segment of the internal carotid artery, blue arrow: fusiform aneurysm complicating the infundibular dilatation of posterior communicating artery.

In this study, we report for the first time the evolution of the PComA infundibulum into a fusiform dilatation with a diameter of 5.25 mm and a length of 8.84 mm at the terminal segment of ICA instead of the frequently reported saccular aneurysm. For intracranial aneurysms, fusiform aneurysms are less common (10-20%) when compared to saccular aneurysms (80-90%). Fusiform aneurysms balloon out on all sides of the blood vessel, while saccular aneurysms balloon out only on one side. Fusiform aneurysms are treated by stenting, flow modulation, bypass surgery, decompression, or observation if the risks of intervention outweigh the potential benefits while saccular aneurysms are treated by coiling, flow diversion, clipping, or observation for small asymptomatic aneurysms (Awad et al., 2017).

We hypothesize that the PComA infundibulum caused this unusual development of the fusiform aneurysm. Another finding that supports our explanation is the presence of other aneurysms in the form of berry aneurysms; a small one in the left ICA and two in the left PCA-P1 segment having diameters of 3.54 mm and 3.63 mm for the proximal and distal aneurysms, respectively. Arrambide-Garza et al. performed a systematic review and metanalysis to evaluate the FPComA as a risk factor for the formation, rupture, and recurrence of the PComA aneurysm and they found a significant association between the presence of

FPComA and aneurysm formation and rupture of aneurysms. This may be triggered by the hemodynamic alterations caused by the variation, leading to changes in the vessel wall (Arrambide-Garza et al., 2023). Another differential diagnosis for the fusiform aneurysm is dolichoectasia. According to Del Brutto et al. (2017), fusiform aneurysms refer to extreme circumferential ballooning of the entire vessel wall for a short segment compared to dolichoectasia which affects a long segment with less widening. Furthermore, true PComA aneurysms are reported to be associated with fetal PComA (Karekezi et al., 2014). However, Min et al. reported a higher incidence of infundibulum with the adult-type PComA (Min et al., 2014).

Fischer et al. (2011) reported the evolution of infundibula into aneurysms. They recommend close MRA follow-up of all PComA infundibula especially if FPComA is detected. Yearly mid-term and long-term follow-up MRI are recommended for infundibula with a diameter of 3 mm or more. In patients with other aneurysms, with a de novo aneurysm, or with a familial incidence of aneurysms, the risk of transformation of an infundibulum to an aneurysm may be increased, and thus follow-up should be even more strict. Karekezi et al. (2014) reported a case of infundibular transformation over 10 years and its progression into a saccular aneurysm and subsequent rupture. Infundibular widening of cerebral arteries can develop into a true aneurysm, especially in FPComA. ICA fusiform aneurysms can cause turbulent flow, thrombus formation, and emboli occluding the branches of ICA resulting in a territory infarction (Zhang et al., 2021). Zada et al. (2008) reported that the ICA-PComA aneurysms have a more considerable risk for infarction and subsequent neurological complications with surgical or endovascular obliteration. The decision of surgical versus endovascular treatment of fetal PCA aneurysms must be carefully considered, given the greater potential for ischemic injury with parent vessel occlusion (Zada et al., 2008).

Meanwhile, Tarkiainen et al. (2024) considered the infundibular dilatations as stable benign anatomical variants and, thus, do not need prophylactic treatment or imaging follow-up, and that the subarachnoid hemorrhage (SAH) reported from the infundibula was caused by misdiagnosed pre-aneurysmal lesions. Thus, there are no conclusive guidelines for follow-up of PComA infundibula.

CONCLUSION

We recommend that there is no need for immediate surgical intervention for the management of the PComA infundibula, although, yearly MRA follow-up is highly recommended. In addition, we recommend MRA follow-up every 6 months for infundibula with a diameter of more than 3 mm, associated with other aneurysms, or associated with a familial history of similar aneurysms. We believe that the presence of FPComA during embryonic development can alter the hemodynamics in the CoW, and create a pressure gradient and turbulent flow that can affect the wall of the terminal segment of ICA and predispose to the development of fusiform aneurysms later on.

Ethics approval

This work was performed on cadaveric specimens obtained from the dissection room of the Anatomy Department, College of Medicine and Health Sciences, National University for Science and Technology. The ethics committee of the College of Medicine and Health Sciences, National University for Science and Technology, approved the research protocol (IRB No: NU/COMH/EBC0031/2022).

Authors` Contribution

Conceptualization: HRHE, EME. Data acquisition: All authors. Data analysis or interpretation: HRHE, EME. Drafting of the manuscript: All authors. Critical revision of the manuscript: HRHE, EME. Approval of the final version of the manuscript: all authors.

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