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Fetal-Type Posterior Communicating Artery in a Concurrent Anterior and Posterior Circulation Infarct: A Case Report and Literature Revue

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Abstract **Case Report**

The fetal-type posterior cerebral artery (FTPCA) denotes an anomaly within the posterior cerebral circulation, characterized by the distal segment of the posterior cerebral artery (PCA) being supplied by a branch originating from the internal carotid artery (ICA) through the fetal posterior communicating artery (fetal PCOM). The presence of a fetal posterior communicating artery represents a recognized variation within the cerebral vascular anatomy, observed in 4-29% of individuals. This variant may pose distinct challenges in both the detection and management of cerebrovascular conditions or strokes. We report a 60-year-old man who presented with sudden onset left sided weakness and aphasia. A brain CT scan was performed, revealing a right posterior and middle cerebral arteries territories infarct with bilateral fetal-type posterior communicating arteries. CTA brain is commonly done in ischemic stroke cases to assess presence of large vessel occlusions and intracranial or extracranial atherosclerotic disease. However, this case depicts its additional role in detecting anatomical variants of cerebral circulation. In terms of clinical importance, presence of multiple territories infarction portends a poorer neurological outcome.

Keywords: Fetal-type posterior communicating artery, circle of willis, posterior cerebral artery, ischemic stroke.

INTRODUCTION

The posterior cerebral artery (PCA) is a significant branch originating from the basilar artery within the posterior circulation of the brain. Its primary role is to supply blood to the occipital lobe, the inferomedial temporal lobe, and parts of the posterior inferior parietal lobe [1]. Fetal origin of the PCA is a frequently encountered variation in the posterior cerebral circulation, with an estimated prevalence ranging from 15% to 32% of individuals [2]. This common variation is typically identified post-ischemic stroke occurrence or during noninvasive or invasive cerebral angiography conducted for various medical reasons.

Fetal-type posterior cerebral artery (FTPCA) refers to a condition where the primary blood supply to the posterior cerebral artery (PCA) territory arises from the internal carotid artery (ICA) through the posterior communicating artery (PCOM), coupled with either the absence or underdevelopment (hypoplasia) of the P1 segment of the PCA. This variation of the PCOM is termed fetal PCOM. Van Raamt et al. proposed the designation "full fetal-type PCA (full FTPCA)" when the P1 segment is entirely absent, and "partial FTPCA" when the P1 segment is hypoplastic [3]. In both scenarios, imaging studies such as computed tomography angiography (CTA) of the brain reveal an enlarged posterior communicating artery compared to the norm. While FTPCA is considered a normal variant, it may lead to inadequate collateral circulation, and an enlarged posterior communicating artery may pose an increased risk for ischemic injury to both the anterior and posterior cerebral regions [4]. A simultaneous infarction involving multiple arterial territories indicates a poorer neurological prognosis. We present a case of concurrent infarction involving the right middle cerebral artery (MCA) and PCA territories, while sparing the anterior cerebral artery (ACA).

CASE REPORT

A 60-year-old male patient presented with an acute onset of left-sided hemiparesis and dysarthria. He denied experiencing headache, blurred vision, vomiting, or numbness. There were no reported seizures, chest pain, or palpitations. He has a history of active smoking but no known diagnosis of diabetes, hypertension or dyslipidemia.

A brain scan CT was performed and revealed:

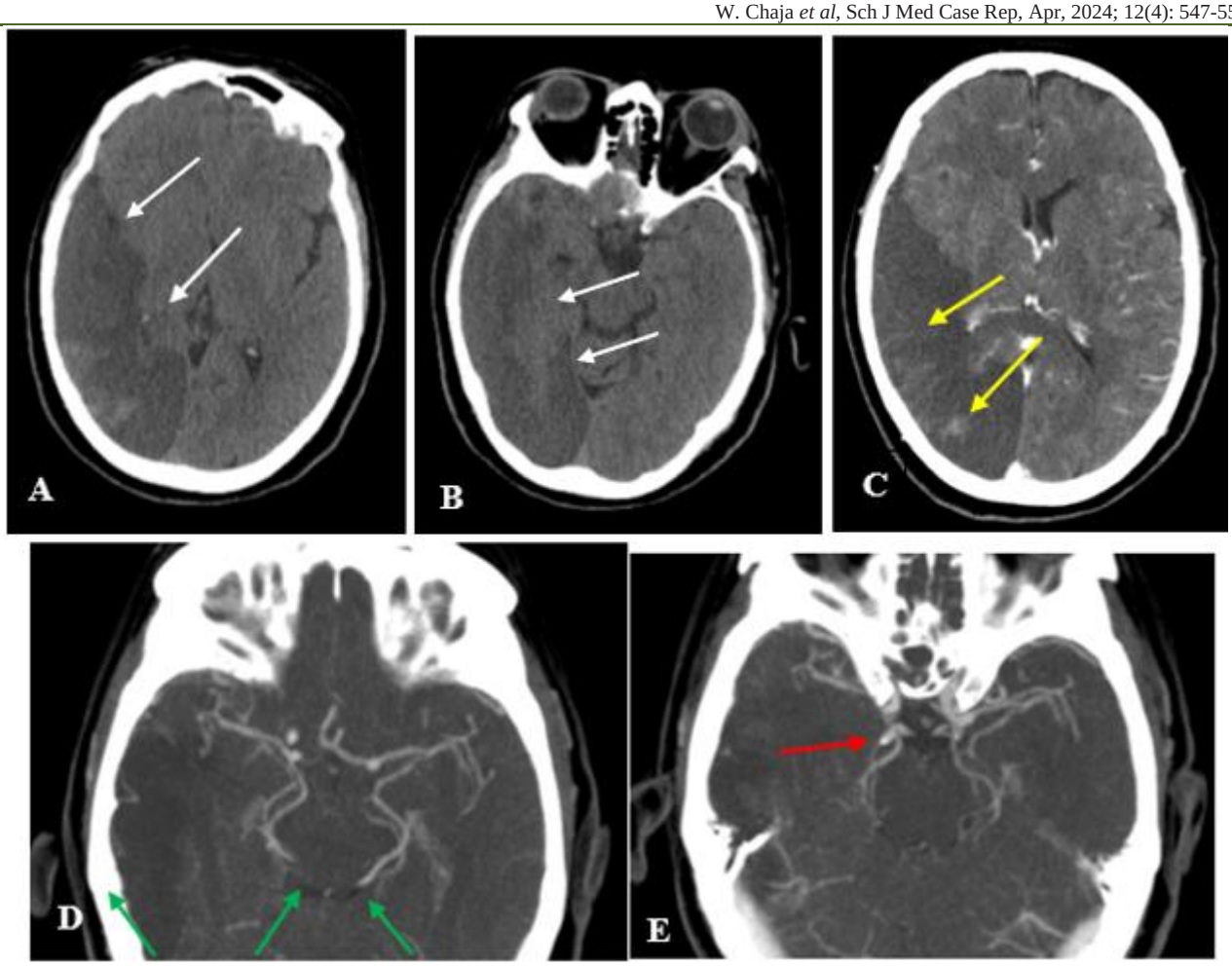


Figure 1: Axial images of brain CT scan without contrast (A,B) and with contrast (C,D) showing cortico-subcortical hypodensity areas (white arrows) in the right temporo-parieto-occipital region with gyriform enhancement (yellow arrows), accompanied by a slender appearance of the basilar trunk and absent P1 segment of both PCA (green arrows). It is associated with a slender morphology of the internal carotid artery in both its intracavernous and clinoid segments (red arrows)

The diagnostic of a well established concurrent right MCA and PCA territory infarctions with bilateral fetal-type posterior communicating arteries was confirmed.

DISCUSSION

The growth of the occipital and temporal lobes, particularly their basal regions, is influenced by that of the frontal lobe [5]. This sequential development affects the maturation of the posterior cerebral arteries (PCAs), which are the last to develop. Interestingly, variations in the posterior circulation do not appear to be influenced by the prior condition of the basilar artery (BA) [5].

In embryos between 28 and 30 days old, the cranial and caudal divisions of the primitive internal carotid artery (ICA) are established. These divisions anastomose with the longitudinal neural arteries, which subsequently contribute to the formation of the BA [5, 6]. The primitive posterior communicating artery (PComA) and the stem of the PCA originate from the caudal branch of the primitive ICA. Normally, the caliber of the primitive PComA decreases as the vertebrobasilar system develops. However, if the primitive PComA persists, the primary blood supply to the PCA will be from the ICA via the PComA, leading to a fetal-type PCA (FPCA) [7].

In cases of FPCA, endovascular interventions may benefit from an approach via the carotid rather than the vertebrobasilar route. Neurosurgeons must take care to avoid occluding an FPCA when treating ICA-PComA aneurysms to prevent ischemic events in the PCA territory. Additionally, an FPCA could offer collateral circulation to the posterior brain in the event of an embolus occluding the proximal portion of the BA. However, the occurrence of ischemic events also depends on the collateral connections of the leptomeningeal vessels, which determine whether a vertebrobasilar or ICA territory supply is available [8].

A fetal origin of the posterior cerebral artery (PCA) is a frequently encountered variation in the posterior circulation, estimated to affect 15-32% of the population. In this variation, the posterior

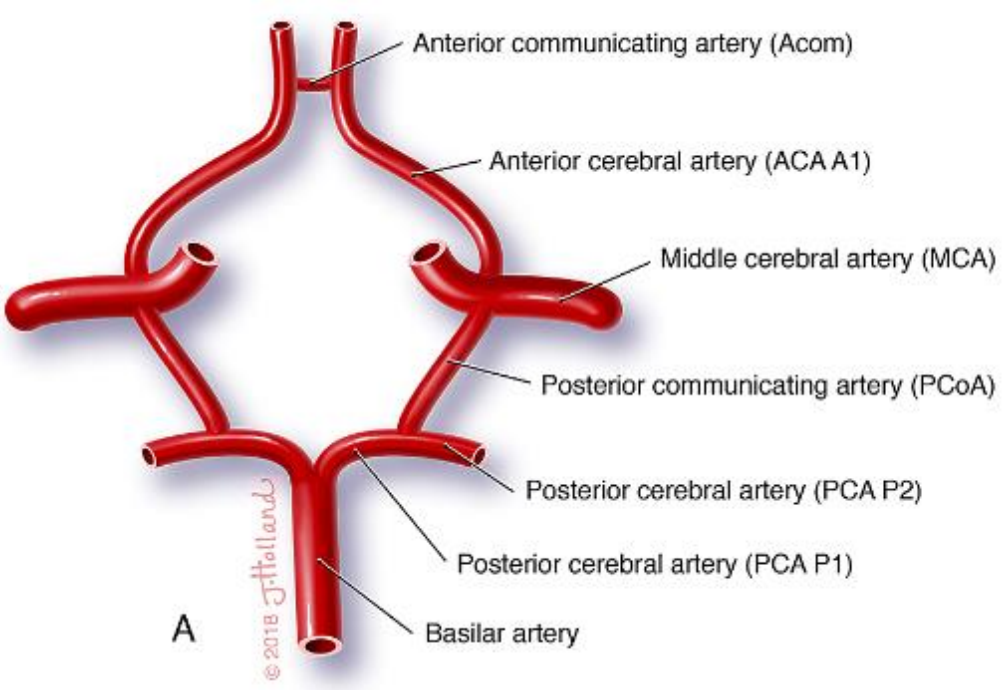
communicating artery (PCOM) tends to be larger than the P1 segment of the PCA, which may be hypoplastic or absent. Consequently, the majority of blood supply to the PCA territory is provided through the PCOM from the ipsilateral internal carotid artery (ICA). This variant of the PCOM is referred to as fetal PCOM.

Our patient suffered a concurrent left MCA and PCA territory infarcts due to the presence of right fetal PCOM (fetal origin of PCA). Nevertheless, CT angiography did not show acute thrombus in either vessel, which could be due to lysed clot or the clots could have migrated further distally. Distal occlusions are not readily visible on CT angiography.

Alternatively, a concurrent infarction could potentially originate from cardiac sources, where a thrombus originating from the intracardiac region might migrate to an intracranial artery via the internal carotid artery, leading to occlusion of the left middle cerebral artery (MCA) and posterior cerebral artery (PCA), with the left posterior communicating artery (PCOM) facilitating cross embolization from the internal carotid artery to the PCA P2 segment or its distal branches. Unfortunately, 24-hour cardiac monitoring (Holter examination) and echocardiography were not conducted to confirm the possibility of cardioembolism.

According to van Raamt *et al*., a complete fetal-type PCA (full FTPCA) carries a higher risk of vascular insufficiency compared to a partial FTPCA due to the absence of leptomeningeal anastomoses between the anterior and posterior circulation [3]. A series of cases reported by Nico *et al*., demonstrated that patients with fetal origin of the PCA are more likely to experience a posterior circulation infarct rather than concurrent infarcts affecting both the anterior and posterior circulation. This phenomenon is supported by a theory suggesting an increased pressure gradient between the internal carotid artery and PCA due to hemodynamic changes resulting from stenotic lesions in the vertebrobasilar system or significant atherosclerosis of the proximal internal carotid artery (Figure 2) [9, 11].

The present case of fetal-type PCA (FTPCA) and concurrent infarction involving the PCA and MCA territories illustrates the anatomical variability and clinical significance of PCA variants. FTPCA appears to be the underlying mechanism of paradoxical infarction in this patient. To the best of our knowledge, reports of concurrent multiple territory infarctions are rare and significantly contribute to the poor outcomes associated with major acute ischemic strokes (TACI and PACI). This case illustrates a typical presentation of TACI and the subsequent use of hyperacute stroke reperfusion therapy as treatment for TACI. Additionally, it underscores the importance of CT angiography of the brain in guiding the management of acute ischemic stroke [10].



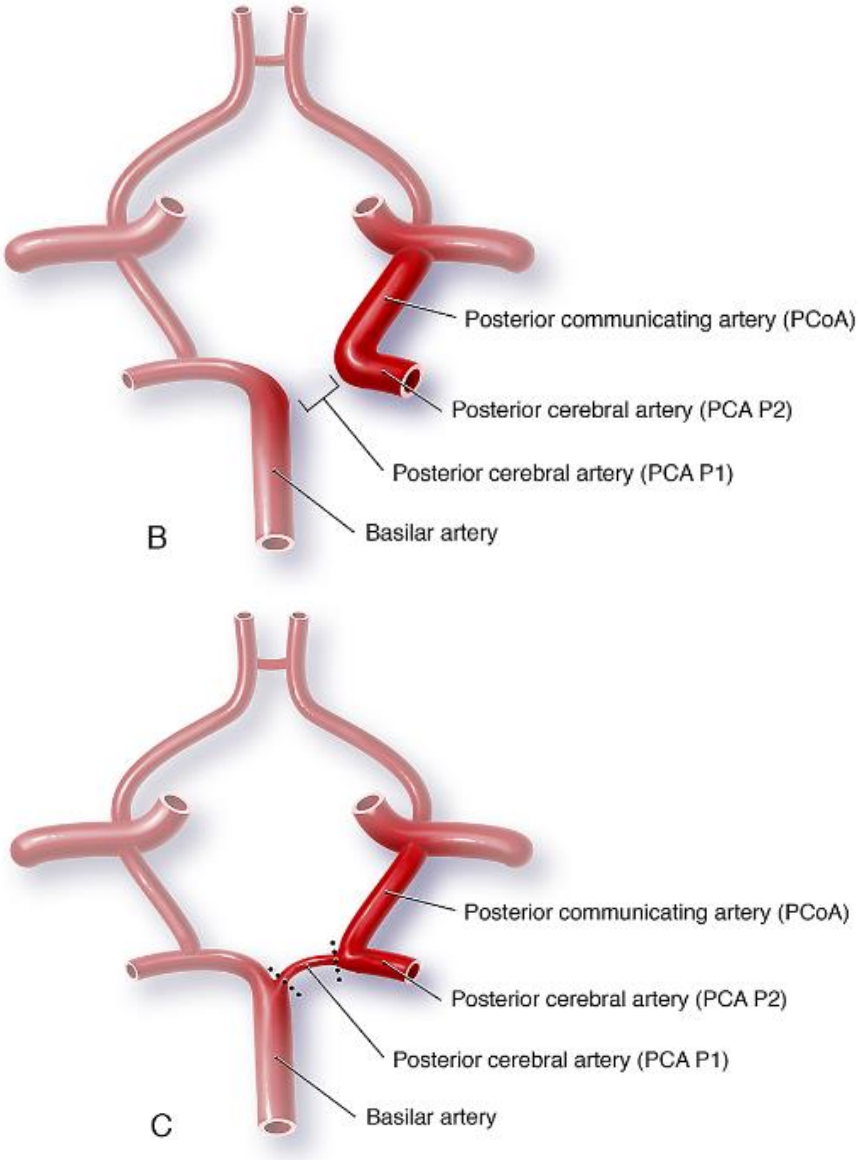


Figure 2: Circle of Willis. Normal configuration of the A) complete Circle of Willis, noting segments of posterior cerebral artery. Abnormal configurations include B) the true fetal posterior communicating artery with an absent P1 segment and C) a fetal-type posterior communicating artery with a hypoplastic P1 segment [11]

Moreover, this case helps elucidate why the patient developed concurrent multiple territory infarcts. FTPCA may exacerbate the extent and severity of anterior circulation strokes by permitting additional infarction in the PCA territory. However, whether FTPCA independently increases the overall risk of stroke in the absence of other risk factors remains unclear. The optimal stroke prevention regimen for individuals with FTPCA and one or more stroke risk factors also requires further investigation. Risk factor management remains crucial in stroke prevention.

CONCLUSION

This case is an example of a bilateral fetal-type posterior communicating artery, with the anterior circulation supplying the PCA via the internal carotid artery. The fetal-type PComA is also associated with increased complications in both endovascular and surgical therapy, with a unique pattern of infarcts due to the abnormal blood supply to the brain. Despite current efforts, research has been mixed and it is unclear whether the presence of a fetal or fetal-type PComA is associated with increased risk of infarction or stroke.

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