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Bilateral Fetal-Type Posterior Cerebral Arteries with Vertebrobasilar Hypoplasia: A Rare Vascular Variant Presenting as Recurrent Posterior Circulation Stroke

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Abstract

Background: Bilateral fetal-type posterior cerebral artery (fPCA) is a rare anatomic variant of the circle of Willis. Its clinical significance, particularly when associated with vertebrobasilar hypoplasia, is poorly understood.

Case presentation: We report a 58-year-old man with recurrent posterior circulation ischemic events. His first episode was characterized by left-sided ataxia, followed six months later by right-sided sensory loss. MRI revealed multiple chronic lacunar infarcts, small vessel ischemic changes, and cortical atrophy. MR angiography demonstrated bilateral fPCA with vertebrobasilar hypoplasia, and MR venography revealed hypoplastic left transverse and sigmoid sinuses.

Conclusion: This case illustrates the rare coexistence of bilateral fPCA and vertebrobasilar hypoplasia as a potential mechanism for recurrent posterior circulation ischemia. Recognition of such variants may help refine prognosis and guide preventive strategies.

Keywords: Basal Ganglia Diabetes Mellitus, Hypoplastic Left Transverse

Introduction

The circle of Willis provides critical collateral circulation for cerebral perfusion. Variants in its configuration are common, but some have important hemodynamic consequences. Fetal-type posterior cerebral artery (fPCA) is one such variant in which the PCA originates predominantly from the internal carotid artery (ICA), with a diminutive or absent P1 segment of the basilar artery.

Unilateral fPCA is relatively frequent, reported in 20–30% of angiographic studies, whereas bilateral fPCA is rare, with a prevalence of only 2–4% [1]. While unilateral fPCA has been associated with concurrent anterior and posterior circulation infarcts [5], bilateral fPCA is rarely

highlighted in clinical literature. Its coexistence with vertebrobasilar hypoplasia may further compromise posterior circulation and predispose to recurrent ischemia.

We report a patient with recurrent posterior circulation stroke in the setting of bilateral fPCA and vertebrobasilar hypoplasia, highlighting the novelty and clinical relevance of this rare vascular constellation.

Case Presentation

A 58-year-old male, with risk factors of long-standing hypertension, type 2 diabetes mellitus, and chronic smoking, presented on two separate occasions within a year.

First episode: sudden-onset left-sided ataxia, imbalance, and mild dysarthria.

Second episode (six months later): right-sided numbness and tingling with gait unsteadiness.

No family history of stroke or thrombophilia was reported. On both occasions, neurological examination confirmed posterior circulation involvement: left limb and truncal ataxia during the first admission, and contralateral hemibody sensory loss with subtle cerebellar signs in the second.

Imaging Findings

MRI Brain:

Multiple chronic lacunar infarcts: bilateral centrum semiovale, corona radiata, basal ganglia, right frontal lobe, right insular cortex, and left lateral medulla.

Grade 2 small vessel ischemic changes.

Age-related cortical atrophy.

Susceptibility-weighted imaging (SWI): multiple blooming foci in bilateral cerebral hemispheres and pons, suggestive of microhemorrhages.

MRA: Hypoplastic vertebrobasilar system with bilateral fetal-type PCAs.

MRV: Hypoplastic left transverse and sigmoid venous sinuses.

Review of Literature

Variants of the circle of Willis are well recognized. Kapoor et al. [1] demonstrated a 2–4% prevalence of bilateral fPCA, significantly lower than unilateral forms. Hartkamp et al. [2] and van Raamt et al. [3] emphasized that these variants compromise collateral capacity.

Schomer et al. [4] and van der Hoeven et al. [5] showed that even unilateral fPCA can predispose to large infarcts due to dependence on carotid inflow. A recent case described by Dutta et al. [6] reported bilateral PCA arising directly from the ICAs causing two-territory stroke, underlining the pathogenic potential of bilateral variants.

However, reports linking bilateral fPCA with recurrent posterior circulation ischemia remain scarce, making the present case a novel contribution. The additional presence of vertebrobasilar hypoplasia further limited perfusion reserve, creating a unique substrate for stroke recurrence.

Discussion

The bilateral fPCA configuration shifts posterior circulation perfusion almost entirely to the ICA, diminishing the role of the basilar artery [1,3]. This arrangement reduces collateral options, particularly if ICA flow is compromised, and increases hemodynamic stress on small vessels. Our patient's recurrent lacunar infarcts and microhemorrhages likely reflect this chronic hemodynamic imbalance superimposed on small vessel disease.

The clinical presentations—left-sided ataxia and right hemibody sensory disturbance—were consistent with multifocal posterior circulation ischemia, correlating with the medullary and thalamic pathways supplied by the compromised vertebrobasilar system.

Importantly, bilateral fPCA is often overlooked in stroke evaluation. Its recognition is crucial because it alters prognosis and may justify more aggressive vascular risk factor control.

Conclusion

We describe a rare case of recurrent posterior circulation ischemia in a patient with bilateral fetal-type PCA and vertebrobasilar hypoplasia. The novelty of this case lies in the coexistence of bilateral fPCA and posterior circulation compromise, a combination seldom reported in literature. Identifying such anatomical variants on routine MRA may provide insights into stroke mechanisms and aid in prevention strategies.

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