RESEARCH PAPER

# A Case Of Acute-On-Chronic Ischemic Stroke In The Posterior Cerebral Artery Territory Associated With Complex Posterior Circulation Variants

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**Abstract**

Ischemic stroke in the posterior circulation accounts for a significant portion of all cerebrovascular events and often presents diagnostic challenges due to variable clinical manifestations and complex vascular anatomy. We report a case of a 61-year-old female patient with acute-onset neurological symptoms, who underwent magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) of the brain. The imaging revealed an acute/subacute infarct in the left posterior cerebral artery (PCA) territory, characterized by gyriform T2/FLAIR hyperintensities with diffusion restriction. This acute event was superimposed on chronic infarcts in the left gangliocapsular region and thalamus, with associated chronic small vessel ischemic changes in the bilateral frontoparietal white matter. Time-of-flight (TOF) MRA demonstrated a complex arrangement of posterior circulation variants, including a non-fusion of the vertebral arteries (VA) where the left VA continued as the basilar artery and the right VA terminated as the anterior inferior cerebellar artery (AICA), a fetal origin of the right PCA, and a hypoplastic left PCA. This case highlights the crucial role of advanced neuroimaging in characterizing both the parenchymal consequences and the underlying vascular anomalies in stroke. It underscores how anatomical variants can create hemodynamic vulnerabilities, predisposing individuals to ischemic events, particularly in the context of chronic microvascular disease. The findings emphasize the need for a comprehensive vascular evaluation to understand stroke etiology and guide secondary prevention strategies.

**Keywords:** Posterior Cerebral Artery, Ischemic Stroke, Magnetic Resonance Angiography, Vertebral Artery Anomaly, Fetal Posterior Cerebral Artery

**How to cite this article:** Manickam M, Murugan G, Manikandan S. A Case of Acute-on-Chronic Ischemic Stroke in the Posterior Cerebral Artery Territory Associated with Complex Posterior Circulation Variants. Int J Drug Deliv Technol. 2026;16(12s): 821-824. DOI: 10.25258/ijddt.16.12s.97

## Introduction

Ischemic stroke remains a leading cause of morbidity and mortality worldwide. Strokes affecting the posterior circulation, which supplies the brainstem, cerebellum, thalamus, and occipital lobes, comprise approximately 20-25% of all ischemic strokes [1]. The clinical presentation can be diverse, ranging from visual field deficits and dizziness to altered consciousness, often leading to delays in diagnosis. The underlying etiology is most commonly embolism (cardioembolic or artery-to-artery) or in-situ small vessel disease, but large artery atherosclerosis also plays a significant role [2].

The posterior circulation exhibits a high degree of anatomical variability. Common variants include hypoplasia of one or both vertebral arteries (VA), a fetal-origin posterior cerebral artery (PCA) where the PCA arises from the internal carotid artery (ICA) rather than the basilar artery, and variations in the configuration of the circle of Willis [3, 4]. While often asymptomatic, these variants can alter cerebral hemodynamics. For instance, a hypoplastic VA or a fetal-type PCA can create boundary zones vulnerable to hypoperfusion, especially in the setting of systemic hypotension or occlusive disease in the feeding arteries [5].

Advanced neuroimaging, particularly MRI with diffusion-weighted imaging (DWI) and MR angiography, is paramount in the acute stroke setting. DWI is the most sensitive sequence for detecting acute

cytotoxic edema, while MRA provides a non-invasive roadmap of the intracranial and extracranial vasculature [6]. Here, we present a case where MRI identified an acute-on-chronic infarct pattern, and MRA revealed a constellation of posterior circulation variants that likely contributed to the patient's stroke pathophysiology.

**Case Presentation**

A 61-year-old female patient presented to the emergency department with complaints of sudden-onset, painless visual disturbance, described as difficulty seeing objects to her left side, which began approximately 24 hours prior to admission. Her family also reported that she had been experiencing some confusion and word-finding difficulties over the past day. Her medical history was significant for long-standing hypertension and type 2 diabetes mellitus, for which she was on irregular medication. There was no documented history of prior stroke or transient ischemic attack (TIA).

On admission, her vital signs were stable, though her blood pressure was elevated at 160/90 mmHg. A neurological examination revealed a left homonymous hemianopia on visual field testing by confrontation. She had mild expressive aphasia but no motor or sensory deficits in the limbs. Her gait was stable, and cerebellar signs were absent. A baseline non-contrast CT brain (not provided) was reportedly unremarkable for acute hemorrhage but showed subtle chronic small vessel changes.

To further evaluate her symptoms, an MRI of the brain was performed on a 3T scanner. The protocol included axial T1-weighted, T2-weighted, diffusion-weighted (DWI), susceptibility-weighted (GRE), and coronal T2-FLAIR sequences, as well as a 3D TOF MRA of the circle of Willis and a 2D TOF MR venography.

The MRI demonstrated well-defined, predominantly gyriform areas of T2 and FLAIR hyperintensity in the left occipital cortex, forceps major, left hemi-splenium of the corpus callosum, and the adjoining inferior temporal gyrus. These areas showed marked hyperintensity on DWI and corresponding low signal on the ADC map, confirming restricted diffusion and indicating an acute/subacute infarct. A subtle area of similar signal abnormality was noted in the left thalamus. Associated findings included subtle volume loss and mild dilatation of the ipsilateral occipital horn of the lateral ventricle, suggesting chronicity in some of the affected territories. Figure 1 demonstrates chronic small vessel ischemic changes as confluent periventricular and deep white matter hyperintensities

Additionally, there were multiple punctate and confluent T2/FLAIR hyperintensities in the bilateral frontoparietal white matter consistent with chronic small vessel ischemic disease. Chronic lacunar infarcts were also noted in the left gangliocapsular region and left thalamus. The brainstem and cerebellum appeared normal. There was generalized prominence of the sulcal and cisternal spaces, indicating age-appropriate atrophy.

The 3D TOF MRA revealed several anatomical variants is shown in Figure 1. There was a non-fusion of the vertebral arteries. The left vertebral artery was dominant and continued as the basilar artery. The right vertebral artery was hypoplastic and terminated as the right anterior inferior cerebellar artery (AICA) without joining to form the basilar artery. Furthermore, the right posterior communicating artery (PCom) was prominent and continued as the right PCA, a configuration known as a fetal origin of the right PCA. The left PCA was noted to have a narrowed caliber compared to the right and arose from the basilar artery. The anterior communicating artery was seen continuing as the anterior cerebral arteries. The intracranial internal carotid arteries and their bifurcations, as well as the proximal anterior and middle cerebral arteries, showed no significant luminal narrowing. The MR venography (Figure 3) showed normal flow in the dural venous sinuses, with no evidence of thrombosis.

Based on these findings, the patient was diagnosed with an acute/subacute infarct in the left PCA territory, superimposed on chronic infarcts and small vessel disease. She was started on dual antiplatelet therapy, a high-dose statin, and strict blood pressure and glycemic control were initiated. She was referred for speech and vision rehabilitation therapy.

**Discussion**

This case illustrates a complex cerebrovascular event in a patient with a high burden of traditional vascular risk factors and underlying anatomical variants. The imaging findings provide a clear picture of the acute pathology an infarct in the PCA territory and offer plausible mechanisms for its occurrence.

The MRI findings are classic for an acute ischemic stroke. The gyriform pattern of restricted diffusion in the left occipital cortex and forceps major is characteristic of a PCA territory infarct, which commonly presents with contralateral homonymous hemianopia, as seen in our patient [7]. The involvement of the splenium of the corpus callosum can contribute to disconnection syndromes, potentially explaining the patient's word-finding difficulties. The presence of

chronic lacunar infarcts in the left gangliocapsular region and thalamus, along with extensive white matter hyperintensities, indicates a significant burden of chronic cerebral small vessel disease, likely secondary to her poorly controlled hypertension and diabetes [8].

The most intriguing aspect of this case, however, is the complex vascular anatomy revealed by the MRA. The described "non-fusion" of the vertebral arteries, where the right VA is hypoplastic and ends as the AICA, represents an extreme form of VA asymmetry or hypoplasia. Vertebral artery hypoplasia (VAH) is a common finding, present in up to 10-15% of the population, and is increasingly recognized as a risk factor for posterior circulation stroke, particularly in the young [9]. A hypoplastic VA can result in reduced blood flow and lower shear stress in the basilar artery, promoting atherogenesis and creating a hemodynamic environment susceptible to embolism [10].

Furthermore, the finding of a fetal-type PCA on the right and a hypoplastic left PCA is significant. In a fetal PCA configuration, the dominant blood supply to the occipital lobe comes from the anterior circulation (ICA) via a large PCom, rather than the posterior circulation (basilar artery) [3]. While this is a common variant, it can have clinical implications. On the left side, where the PCA is noted to be of narrowed caliber and arises from the basilar artery, the blood supply may be tenuous. This combination—a left PCA that is intrinsically small and reliant on a posterior circulation that is itself supplied by a single, albeit dominant, left VA—creates a "double-hit" hemodynamic vulnerability.

We propose two non-mutually exclusive mechanisms for this patient's acute left PCA stroke:

1. **Artery-to-Artery Embolism:** The dominant left VA and the basilar artery, with altered flow dynamics due to the non-fused right VA, could be a nidus for atherosclerotic plaque formation. A thromboembolus originating from this site could have traveled distally to lodge in the left PCA.
2. **Hemodynamic Insufficiency:** Given the hypoplastic left PCA and the fact that the entire posterior circulation is dependent on a single left VA, a transient drop in systemic blood pressure or increased metabolic demand could have led to hypoperfusion in the vulnerable left PCA borderzone territory, resulting in a watershed-type infarct. The acute-on-chronic appearance, with subtle volume loss in the affected area, suggests repeated episodes of ischemia.

The normal MRV effectively rules out cerebral venous thrombosis, which can occasionally mimic arterial stroke in presentation [6]. The absence of significant large-artery stenosis in the carotid or middle cerebral arteries directs the etiological focus towards the posterior circulation.

This case underscores the importance of a comprehensive vascular imaging workup in stroke patients, not just to identify occlusions or stenoses, but also to understand the individual's unique vascular architecture. Recognizing these variants helps the clinician appreciate the underlying hemodynamic state and can guide therapeutic decisions, such as the intensity of blood pressure management to avoid hypoperfusion [5].

**Conclusion**

We report a case of an acute-on-chronic ischemic stroke in the left posterior cerebral artery territory in a 61-year-old female with hypertension and diabetes. The acute infarct was identified on DWI MRI, while the underlying chronic microvascular disease was evident on T2/FLAIR sequences. Time-of-flight MR angiography was crucial in unmasking a complex arrangement of posterior circulation variants, including a non-fused hypoplastic right vertebral artery terminating as AICA, a dominant left vertebral artery, and a hypoplastic left PCA. These anatomical findings created a state of hemodynamic vulnerability, which, in conjunction with traditional risk factors, likely contributed to the stroke pathogenesis. This case highlights the critical role of multimodal MRI in not only diagnosing acute stroke but also in elucidating its underlying etiology, thereby informing more personalized and effective secondary prevention strategies.

**Figures and Legends**

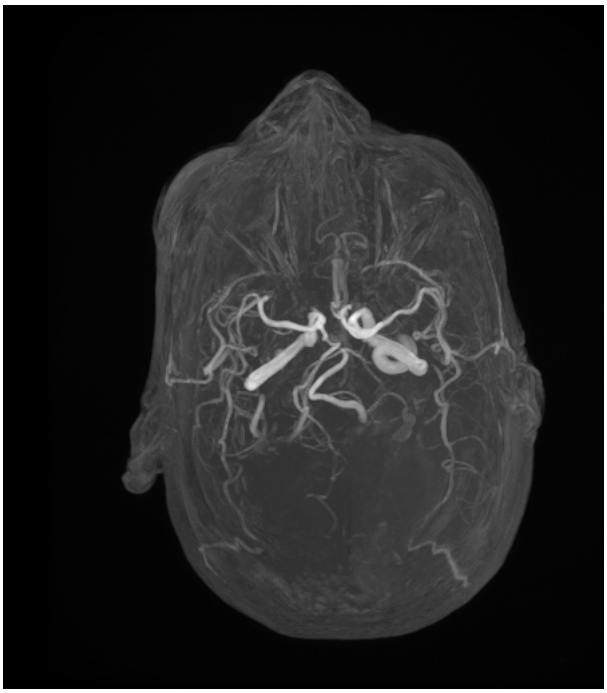

**Figure 1: MRI angio section**

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