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CLINICAL RELEVANCE OF CONGENITAL POSTERIOR CIRCULATION VARIANTS IN EXERCISE-INDUCED HEADACHE: A CASE REPORT

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ABSTRACT

Case report of a young adult with headache triggered by intense physical exertion who was found to have congenital posterior circulation variants, including fetal-type posterior cerebral artery and vertebral artery hypoplasia. The patient presented with unilateral pressure-like headache, photophobia, visual scotomas, and paresthesias after vigorous exercise, without symptoms during mild to moderate activity. Laboratory tests and brain CT were unremarkable. Angio–MRI revealed right fetal-type PCA and left vertebral artery hypoplasia. Current literature provides limited evidence regarding the relationship between these variants and non-ischemic manifestations such as exertional headache. This case highlights the potential hemodynamic implications of posterior circulation variants and reinforces the need for further research on their clinical significance.

Keywords: Headache, Congenital vascular anomaly, Fetal pattern, Vertebral artery hypoplasia, Physical exertion.

INTRODUCTION

Exercise-induced headache is recognized by the International Headache Society as a primary headache disorder that may occur after vigorous physical activity and is generally benign in nature. However, the presence of atypical features, refractoriness to standard management, or association with structural abnormalities warrants thorough investigation to exclude secondary causes.

Congenital variants of the posterior circulation, such as a fetal-type posterior cerebral artery (PCA) and vertebral artery hypoplasia (VAH), are relatively common in the general population and for decades have been regarded as incidental findings. Nevertheless, recent evidence

suggests that these variants may influence cerebral hemodynamics, vasodilatory reserve, and flow autoregulation, particularly under conditions of increased metabolic stress.

Despite growing interest in the role of these variants in ischemic events, few studies have addressed their potential association with non-ischemic manifestations, such as exercise-induced headache. The aim of this case report is to describe a patient with exercise-triggered headache who simultaneously presents a fetal-type PCA and vertebral artery hypoplasia, highlighting the potential clinical relevance of these anatomical variations.

CASE REPORT

A 30-year-old male patient, previously healthy, with a history of episodic migraine since the age of 17, began at the age of 30 to experience a new and recurrent pattern of headache triggered exclusively by intense physical exertion. The episodes typically occurred after high-intensity activities, such as high-intensity interval training (HIIT), and were not triggered by light or moderate exercise.

The headache was pulsatile, unilateral, and lasted between 2 and 48 hours. Episodes were accompanied by photophobia, scintillating scotomas described as “floaters,” dizziness, and transient paresthesia of the fingers. There were no headache episodes during exertion; all symptoms began after completion of physical activity. The patient denied a family history of primary headache disorders, stroke, fibromuscular dysplasia, or vascular malformations.

At admission, previously undiagnosed systemic arterial hypertension was identified (170 × 120 mmHg). Routine laboratory tests were within normal limits. Transthoracic echocardiography demonstrated concentric left ventricular remodeling. Exercise stress testing revealed an exaggerated hypertensive response without evidence of myocardial ischemia. Olmesartan 40 mg/day was initiated, resulting in normalization of blood pressure levels, but without reduction in headache frequency or intensity. Contrast-enhanced cranial computed tomography showed no abnormalities.

Given the refractoriness of symptoms and the exclusively post-exertional pattern, magnetic resonance angiography of the brain was performed, which revealed:

• Internal carotid arteries and their intracranial segments were patent, with no significant stenosis.
• Anterior and middle cerebral arteries showed no abnormalities.
• Right posterior cerebral artery demonstrated predominant supply from the posterior communicating artery, consistent with a fetal-type posterior cerebral artery.
• Left vertebral artery hypoplasia was identified, with preserved caliber of the right vertebral artery and the basilar artery.
•No aneurysms, arteriovenous malformations, or venous thrombosis were detected.

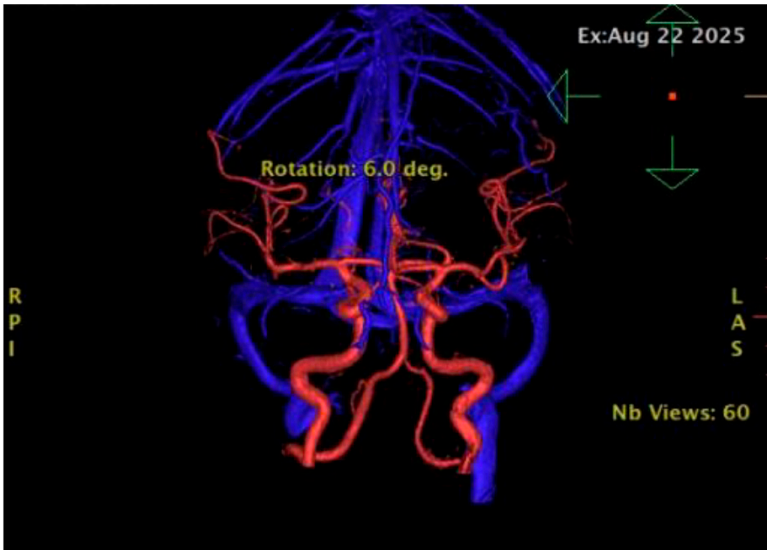

Figure 1. Cranial magnetic resonance angiography demonstrating hypoplasia of the left vertebral artery. Personal archive.

In light of these findings, further investigation was pursued with carotid and vertebral Doppler ultrasonography, performed later during clinical follow-up. The study demonstrated preserved antegrade flow, with no stenosis, occlusions, or findings suggestive of vertebral steal syndrome.

The patient was followed for approximately two years, with sustained blood pressure control and maintenance of a healthy body weight. Despite overall improvement in the cardiovascular profile, exertion-triggered headache persisted with stable characteristics, reinforcing the hypothesis that the identified congenital variants of the posterior circulation may play a modulatory role in the pathophysiology of the condition.

DISCUSSION

Congenital variants of the posterior circulation, such as the fetal-type posterior cerebral artery (PCA) and vertebral artery hypoplasia (VAH), result from alterations in the embryological development of the vertebrobasilar system. During embryogenesis, posterior cerebral circulation initially depends on the carotid system through the posterior communicating arteries. Persistence of this secondary supply, due to incomplete regression of the primordial connections, gives rise to the fetal-type PCA, in which the posterior cerebral artery receives predominant blood flow from the internal carotid artery rather than from the vertebrobasilar system.[1-3]

Vertebral artery hypoplasia, in turn, results from incomplete development of the cervical branches that form the vertebral artery, leading to caliber asymmetry and, in some cases, reduced hemodynamic contribution to the posterior circulation.[4–6] Although both conditions are considered common anatomical variants, they may carry potential clinical implications depending on the physiological context.[7]

Association between headache and posterior circulation variants

Although these variants have traditionally been classified as benign findings, a growing body of literature suggests a possible relationship between anatomical patterns of the Circle

Figure 2. (A) Patient positioning for pre-oxygenation. (B) Video laryngoscopy.

of Willis and clinical manifestations, including primary or exertion-induced headaches. Recent studies have shown that structural asymmetries may influence vascular reactivity, regional hemodynamic reserve, and flow distribution during states of increased metabolic demand, such as vigorous exercise.[2,5,6,8,9,10]

Importantly, although the literature remains limited and heterogeneous, there are reports associating recurrent headaches, including exertional headache, with alterations such as vertebral artery hypoplasia and fetal-type posterior cerebral artery, particularly when these variants coexist.[2,3,5]

Proposed pathophysiological mechanisms include:

1. Reduction in vasodilatory reserve of the posterior circulation, leading to transient perfusion imbalance;
2. Asymmetry in the response to carbon dioxide and in neurovascular autoregulatory mechanisms;[9]
3. Predisposition to focal post-exercise hyperperfusion due to reduced capacity for flow accommodation;
4. Increased trigeminovascular sensitivity in migraine patients, modulated by anatomical variations.

Our patient presented with two potentially contributory factors:

- a right fetal-type posterior cerebral artery;
- left vertebral artery hypoplasia.

This combination may alter posterior circulation perfusion dynamics during conditions of abrupt increases in systemic blood flow, such as those occurring during high-intensity exercise.

At present, however, the evidence remains limited, and there is no consensus regarding a causal relationship between these variants and post-exertional headache, although recent publications suggest a plausible association that warrants further investigation.

RESULTS

Congenital variants of the posterior circulation, such as a fetal-type posterior cerebral artery and vertebral artery hypoplasia, are often considered incidental findings. However, this report reinforces that in symptomatic individuals, particularly those with exercise-triggered headache, these variants may act as anatomical modulators of regional hemodynamics and trigeminovascular sensitivity.

Although the available literature describes similar cases, systematic studies exploring the role of these variants in the pathophysiology of exertion-induced headache remain scarce. Thus, this case adds to the growing body of evidence suggesting that congenital vascular anomalies, whether isolated or combined, may have clinical relevance beyond incidental findings, supporting the need for further investigation through case series and population-based studies.

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EDITORIAL AND REVIEW

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