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Bilateral Vertebral Artery Dissection and Patent Foramen Ovale Leading to Cerebellar Ischemia in a Young Adult: Case Report and Literature Review

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Abstract

Cervical artery dissection and patent foramen ovale (PFO) are relevant causes of ischemic stroke (IS) in young adults. We report the case of a 35-year-old previously healthy man who presented with severe vertigo, gait ataxia, and bradycardia, later diagnosed with cerebellar infarction secondary to bilateral vertebral artery dissection. Further investigation revealed the presence of a PFO. This case underscores the importance of thorough etiological investigation of IS in young adults, focusing on the identification of cervical artery dissections and structural cardiac abnormalities as risk factors.

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Keywords

Arterial dissection; Patent foramen ovale; Ischemic stroke; Young adult; Cerebellar infarction.

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Introduction

Ischemic stroke (IS) in young adults presents a diagnostic challenge, with an etiology distinct from that observed in older populations. Among the less common causes, cervical artery dissection and patent foramen ovale (PFO) stand out as underdiagnosed conditions with significant prognostic impact [1,2].

This report describes a rare case of cerebellar infarction associated with bilateral vertebral artery dissection and PFO in a young patient, along with a review of the underlying pathophysiological mechanisms and discussion on diagnostic and therapeutic approaches.

Case Report

A 35-year-old man with no prior medical history presented to the emergency department with severe vertigo, intractable vomiting, gait ataxia, and bradycardia. He denied headache.

Initial cranial CT scan was unremarkable. He received symptomatic treatment and was discharged for outpatient follow-up.

Seeking further evaluation, the patient reported that three days before symptom onset, he had attended a music event where he remained with his neck extended for prolonged periods while taking photos for professional reasons.

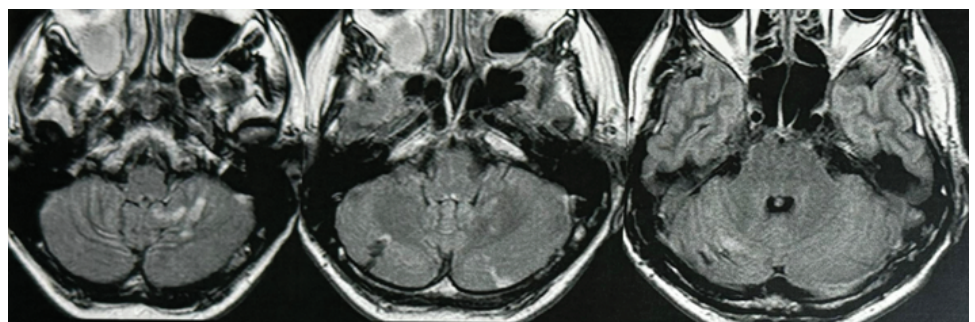


Figure 1. Axial brain MRI (T2 FLAIR sequences) showing hypointense areas in the bilateral cerebellar hemispheres with loss of cortico-subcortical differentiation, consistent with subacute ischemic lesions. There is symmetric involvement of the posterior cerebellar lobes, with no significant mass effect or midline shift. Findings are compatible with bilateral cerebellar infarctions, possibly related to vertebral artery dissection or paradoxical embolism via PFO.

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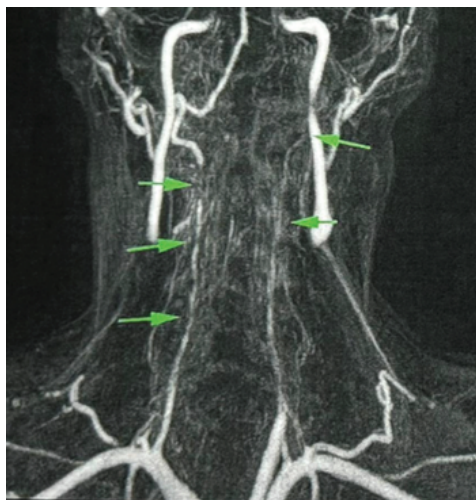


Figure 2. Cervical MR angiography revealing extensive dissection throughout the right V2 segment, with multifocal narrowing and a critical stenosis in the distal right V2 segment. Findings suggestive of dissection in the proximal left V3 segment, along with a probable intraluminal thrombus in the distal V3 and along the V4 segment, extending to the origin of the left PICA, leading to occlusion/subocclusion of these arterial segments. Consequently, there is slow flow and narrowing of the left V1 and V2 segments.

He was admitted for further investigation. Brain MRI (Figure 1) and MR angiography (Figure 2) of the head and neck vessels revealed bilateral cerebellar ischemic infarctions and bilateral vertebral artery dissection.

The patient was started on oral anticoagulation and antiplatelet therapy (apixaban 5 mg twice daily and aspirin 100 mg daily), resulting in clinical improvement and discharge after four days.

One week later, during a bike ride, he experienced another episode of malaise. Repeat MR angiography showed progression of the vertebral dissection.

During outpatient follow-up, he remained asymptomatic, with no new neurological deficits. He reported a prior history suggestive of an undiagnosed ischemic event, including transient right-sided paresthesia four years earlier and severe vertigo during childhood.

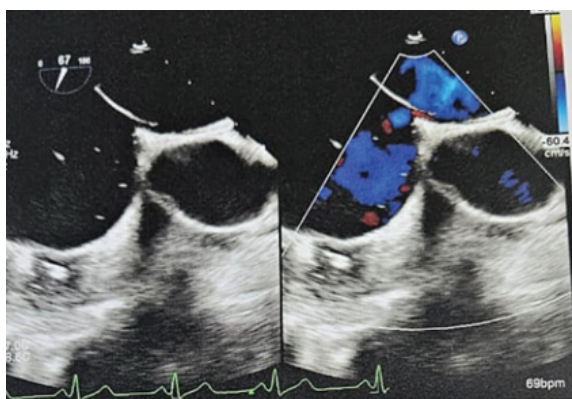


Figure 3. TEE in bicaval view, without (left) and with (right) color Doppler, showing separation between the septum primum and septum secundum, forming an interatrial tunnel consistent with PFO. Doppler imaging reveals left-to-right transseptal flow, confirming spontaneous interatrial shunt, suggestive of hemodynamically significant communication.

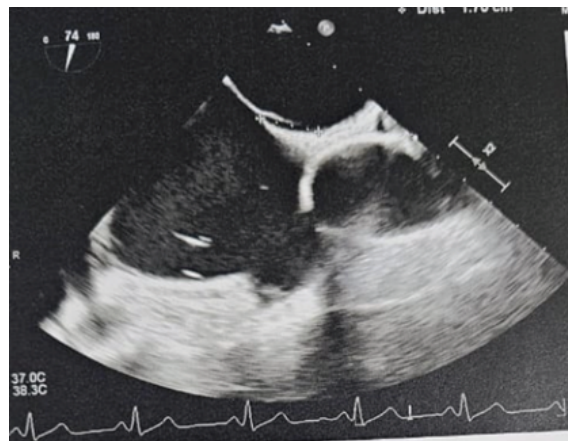


Figure 4. TEE in bicaval view showing septal separation at the foramen ovale site, forming an interatrial tunnel measuring approximately 17 mm in length and 3 mm in height. Left-to-right transseptal flow is evident, consistent with spontaneous shunt, suggestive of PFO.

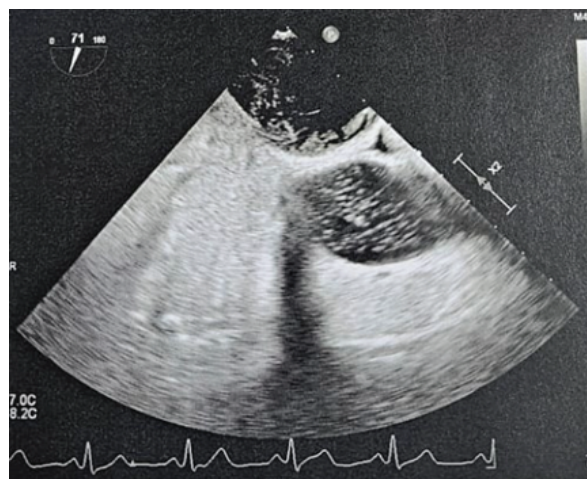


Figure 5. Contrast TEE (agitated saline) demonstrating adequate opacification of the right heart chambers. Early and abundant passage of microbubbles from the right atrium to the left atrium indicates a significant spontaneous interatrial shunt through the PFO, even in the absence of Valsalva maneuver, confirming a high-grade right-to-left communication.

Given his clinical and neurological history, a transesophageal echocardiogram (TEE) was performed, revealing a PFO (Figures 3, 4, and 5).

The patient subsequently underwent successful endovascular PFO closure without complications and remains under multidisciplinary outpatient follow-up.

Discussion

Vertebral artery dissection (VAD) is a significant cause of IS in young adults. Although relatively rare in the general population (accounting for about 2% of all strokes), its incidence can rise to 25% among patients under 45 years of age [1,2]. VAD accounts for a substantial proportion of disabling neurological events in previously healthy individuals, making early diagnosis critical.

The vertebral artery's anatomy, particularly the V3 segment, predisposes it to mechanical shear and traction forces during neck hyperextension or rotation. This segment, located between C2 and the foramen magnum, is anchored at anatomical

points that make it vulnerable to intimal injury during abrupt movements [3,4]. Such injuries may occur after minor trauma, cervical manipulation, or even routine activities such as yoga, weightlifting, or prolonged hyperextension positions—as in this case [5,6].

The case closely resembles the "hairdresser syndrome," described by Go et al. [6], in which prolonged neck hyperextension leads to ischemic symptoms in the posterior circulation. Recent literature highlights the importance of recognizing this arterial injury mechanism even in patients without traditional cardiovascular risk factors [7,8].

In parallel, PFO is present in approximately 15–25% of the general population [9]. While often asymptomatic, it has been strongly associated with cryptogenic stroke—particularly in individuals under 60—being identified in up to 35% of such cases [10]. The most accepted mechanism is paradoxical embolism, in which venous thrombi cross the PFO and enter the arterial cerebral circulation [10,11].

Recent trials, such as CLOSE, REDUCE, and DEFENSE-P, have demonstrated the benefit of percutaneous PFO closure for secondary prevention in selected cases, significantly reducing the risk of recurrent IS compared to medical therapy alone [12–14]. Current guidelines from the American Academy of Neurology and the European Stroke Organisation recommend thorough PFO evaluation in cryptogenic stroke patients under 60, especially when associated with atrial septal aneurysm or moderate-to-severe shunting [14,15].

In this case, the coexistence of VAD and PFO suggests the involvement of multiple mechanisms contributing to the ischemic events. Although VAD alone can explain the cerebellar infarction, the presence of PFO increases the risk of recurrence and should be factored into the long-term therapeutic plan. Initial management with anticoagulation and antiplatelet therapy was appropriate, and evaluation for potential PFO closure should always involve a multidisciplinary team [14].

In summary, this case highlights the need to investigate nontraditional causes of IS in young adults, such as arterial dissection and PFO. MR angiography, CT angiography, and contrast-enhanced TEE are essential diagnostic tools. Treatment should be individualized, involving neurologists, cardiologists, and radiologists, to prevent recurrent events and long-term neurological sequelae.

Conclusion

The association of vertebral artery dissection and PFO represents a potentially underdiagnosed cause of IS in young adults. This case reinforces the importance of detailed history-taking, comprehensive etiological investigation, and multidisciplinary management to prevent recurrence and guide individualized treatment strategies.

Conflict of Interests

The authors have no conflict of interests to declare

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