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Vertebrobasilar Insufficiency Exacerbation in the Context of an Active COVID-19 Infection

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Abstract

Background: Vertebrobasilar insufficiency (VBI) is caused by impaired blood flow in the posterior cerebral circulation. Coronavirus disease 2019 (COVID-19) has been associated with endothelial dysfunction and vascular inflammation that may exacerbate underlying cerebrovascular disease.

Case Presentation: A 57-year-old male with hypertension and type 2 diabetes mellitus presented with acute worsening of chronic vertigo, nausea, and vomiting. Computed tomography angiography demonstrated stenosis of the basilar, left vertebral, and left internal carotid arteries, while magnetic resonance imaging excluded an acute cerebrovascular accident. The patient tested positive for SARS-CoV-2 and was treated symptomatically with dual antiplatelet therapy. His symptoms improved, but he left the hospital against medical advice before completing treatment.

Conclusion: This case suggests a potential association between acute COVID-19 infection and exacerbation of vertebrobasilar insufficiency in patients with underlying cerebrovascular atherosclerosis. Further studies are needed to clarify this relationship.

Keywords

Vertebrobasilar Insufficiency, COVID-19, Vertigo, Cerebrovascular Disease, Posterior Circulation Ischemia

Introduction

Vertebrobasilar insufficiency (VBI) describes the constellation of symptoms that arise from transient and recurrent ischemia of the brain due to partial or complete occlusion of the vertebral and basilar arteries [1,2]. Also known as the posterior circulation, the vertebrobasilar arteries supply blood to the brainstem, cerebellum, subcortical structures, and the occipital and temporal lobes [1]. Accordingly, VBI presents with the acute onset of vertigo as the most common symptom, often accompanied by ataxia, syncope, and dysarthria [1] (**see Appendix A**).

Approximately 25% of ischemic events affecting

brain function occur in the posterior circulation [1]. Because of the redundant circulation provided by the contralateral vertebral artery and the internal carotid artery via the posterior communicating artery, symptomatic VBI usually results from partial or complete occlusion of the bilateral vertebral arteries or the basilar artery [1]. The most common cause of VBI is hemodynamic instability secondary to vertebrobasilar artery stenosis [1]. VBI is positively associated with advanced age, atherosclerotic disease, and conditions associated with impaired sympathetic control, such as diabetes mellitus [1]. Systemic disease is usually a prerequisite for producing the hemodynamic instability and subsequent failure of the collateral circulation necessary for the onset of VBI symptoms [1].

The differential diagnosis of VBI includes other causes of acute-onset dizziness, such as benign paroxysmal positional vertigo, acute labyrinthitis, stroke, and transient ischemic attack (TIA) involving other vascular territories [1]. To identify the location of partial or complete occlusion, computed tomography (CT) and magnetic resonance imaging (MRI) have been shown to be comparable, although CT may produce fewer false-positive findings [2]. For the diagnosis of acute infarction in the posterior circulation, MRI is superior to CT [1]. Caussé et al. [3] identified vertebrobasilar deprivation nystagmus as a sensitive physical examination finding that may assist in the diagnosis of VBI.

Coronavirus disease 2019 (COVID-19) is a viral illness caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), an enveloped, single-stranded, positive-sense RNA virus [4]. Although SARS-CoV-2 is primarily known for causing upper respiratory symptoms, the virus is now recognized to affect a variety of organs, including the heart, brain, eyes, and vasculature [5]. Previous case-control studies have demonstrated associations between SARS-CoV-2 infection and vascular dysfunction, including the progression of atherosclerosis and an increased incidence of thromboembolic events [5].

Although a causal relationship has not yet been established, one proposed mechanism relates to the manner in which the virus enters and establishes infection in the host [5]. SARS-CoV-2 is thought to bind to the angiotensin-converting enzyme 2 (ACE2) receptor to enter host cells [5,6]. Because ACE2 receptors are also expressed on endothelial cells, viral binding may damage blood vessels and induce inflammation, thereby activating the coagulation cascade [5,6]. Additionally, a case-control study found that individuals with a recent SARS-CoV-2 infection had greater arterial stiffness, as evidenced by increased brachial-ankle pulse wave velocity and carotid-femoral pulse wave velocity, compared with healthy controls [7].

This case study highlights a potential association between acute SARS-CoV-2 infection and worsening vertebrobasilar insufficiency. We present this report to propose that the vascular inflammation and endothelial dysfunction associated with COVID-19 may destabilize underlying atherosclerotic disease, leading to the exacerbation of vertebrobasilar insufficiency and the acute worsening of dizziness.

Case Report

A 57-year-old male with a past medical history of hypertension and type 2 diabetes mellitus presented to the Emergency Department (ED) with acute worsening of dizziness. The patient reported a four-month history of recurrent episodes of vertigo, which had significantly intensified over the past day and were now associated with new-onset nausea and vomiting. He denied any head trauma, loss of consciousness, headaches, or sensory deficits.

A code stroke was activated according to the local protocol. The patient's physical examination was unremarkable, and his National Institutes of Health Stroke Scale (NIHSS) score was 0. Vital signs were within the normal range, except for a blood pressure of 147/82 mmHg and a temperature of 99.1°F. Laboratory workup was notable for a positive SARS-CoV-2 test but was otherwise unremarkable. An electrocardiogram (EKG) showed normal sinus rhythm.

Computed tomography (CT) of the brain without contrast was unremarkable. However, a subsequent CT angiogram of the brain and neck revealed mild stenosis of the basilar artery, moderate stenosis of the left

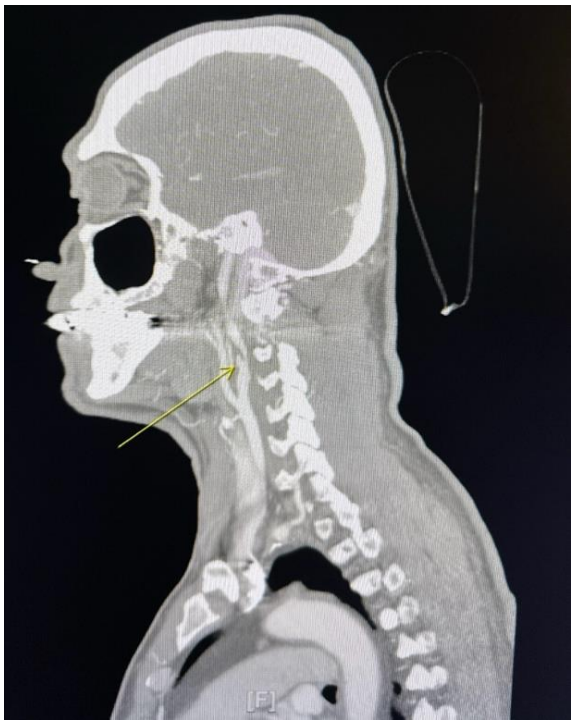

Figure A:

CT Brain/Neck Angiogram demonstrating moderate stenosis of the left internal carotid artery.

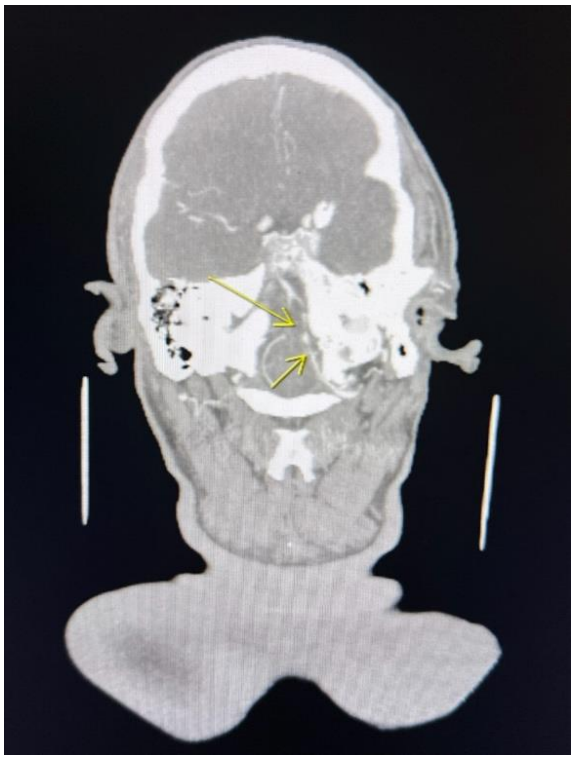

Figure B:

CT Brain/Neck Angiogram revealing two focal areas of severe stenosis of the distal left vertebral artery.

internal carotid artery (**Fig-1A**), and severe stenosis of the distal left vertebral artery (**Fig-1B**), raising concern for a cerebrovascular accident (CVA). Interventional neurology was consulted, and magnetic resonance imaging (MRI) of the brain was obtained, which ruled out an acute CVA but demonstrated global atrophy and chronic microvascular ischemic changes. A chest X-ray and CT of the abdomen and pelvis with contrast were non-contributory.

The patient was treated in the ED for nausea and dizziness, receiving intravenous (IV) fluids, IV ondansetron (Zofran), IV famotidine (Pepcid), meclizine, IV diazepam (Valium), and aspirin. IV metoclopramide (Reglan) was administered for persistent nausea, and the patient's condition improved with observation. He was diagnosed with an acute COVID-19 infection and stenosis of the basilar artery, left vertebral artery, and left internal carotid artery. He was started on dual antiplatelet therapy (clopidogrel and aspirin) and admitted to the medicine team for further observation.

The subsequent clinical course is unknown, as the patient left the hospital against medical advice before completing treatment.

Discussion

This case report describes a patient whose VBI symptoms worsened in the setting of an acute COVID-19 infection. Notably, apart from dizziness and nausea, the patient did not exhibit typical upper respiratory symptoms such as cough, rhinorrhea, sore throat, or fever.

The significance of this case lies in its contribution to the growing body of evidence suggesting that COVID-19 infection may exacerbate atherosclerotic disease and its clinical manifestations. Current literature indicates that SARS-CoV-2 infection can provoke a systemic inflammatory response, endothelial dysfunction, and a prothrombotic state, all of which may worsen underlying vascular pathology [8,9]. In patients with pre-existing vertebrobasilar stenosis, these pathophysiological changes may compromise cerebral perfusion and precipitate or intensify ischemic symptoms.

Future research utilizing animal models is needed to elucidate the mechanisms by which SARS-CoV-2

aggravates atherosclerosis and vascular dysfunction. Establishing clear causal pathways may facilitate the development of targeted therapeutic interventions aimed at mitigating COVID-19-associated vascular complications and improving clinical management.

Additionally, this case underscores the importance of considering COVID-19 infection in the differential diagnosis of patients presenting with stroke-like symptoms, particularly in those with multiple risk factors for atherosclerotic disease, including hypertension, type 2 diabetes mellitus, and hyperlipidemia. Early testing for SARS-CoV-2 may allow clinicians to provide appropriate counseling to reduce disease transmission and initiate timely management for patients at risk of severe COVID-19-related complications.

Limitations

Although COVID-19 has been associated with vascular dysfunction and acute cardiovascular complications, the precise mechanisms remain incompletely understood. Therefore, a direct causal relationship between SARS-CoV-2 infection and exacerbation of this patient's pre-existing vertebral artery stenosis cannot be established. Additionally, dizziness is a recognized manifestation of acute COVID-19 and may independently explain the patient's symptoms, particularly in the absence of focal neurological deficits [1]. However, the patient's history of chronic dizziness with acute worsening during infection raises the possibility of superimposed vertebrobasilar insufficiency exacerbated by virus-associated vascular effects. Further investigation is needed to clarify the impact of SARS-CoV-2 on pre-existing cerebrovascular disease and its role in symptom progression.

Conclusion

This case illustrates a unique presentation of VBI exacerbation in the setting of active COVID-19 infection, highlighting how the virus may exacerbate underlying vascular abnormalities. The vascular dysfunction and hemodynamic instability associated with COVID-19 should be considered when evaluating patients with atherosclerotic disease who present with transient ischemic symptoms.

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None.

Conflict of Interest

The authors have read and approved the final version of the manuscript, and the authors declares no conflicts of interest.

References

[1] Lima Neto AC, Bittar R, Gattas GS, Bor-Seng-Shu E, Oliveira ML, Monsanto RDC, Bittar LF. Pathophysiology and Diagnosis of Vertebrobasilar Insufficiency: A Review of the Literature. Int Arch Otorhinolaryngol. 2017 Jul;21(3):302-307. [**PMID**: 28680502]

[2] Benjamin R, Lui F. Vertebrobasilar Insufficiency. [Updated 2025 Dec 1]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK482259/

[3] Causse JB, Conraux C, Causse J. Le nystagmus de privation vertébro-basilaire [Vertebral-basilar artery insufficiency nystagmus (author's transl)]. Ann Otolaryngol Chir Cervicofac. 1978 Mar;95(3):225-34. French. [**PMID**: 666225]

[4] Naqvi AAT, Fatima K, Mohammad T, Fatima U, Singh IK, Singh A, Atif SM, Hariprasad G, Hasan GM, Hassan MI. Insights into SARS-CoV-2 genome, structure, evolution, pathogenesis and therapies: Structural genomics approach. Biochim Biophys Acta Mol Basis Dis. 2020 Oct 1;1866(10):165878. [**PMID**: 32544429]

[5] Karakasis P, Nasoufidou A, Sagris M, Fragakis N, Tsioufis K. Vascular Alterations Following COVID-19 Infection: A Comprehensive Literature Review. Life (Basel). 2024 Apr 24;14(5):545. [**PMID**: 38792566]

[6] Beyerstedt S, Casaro EB, Rangel ÉB. COVID-19: angiotensin-converting enzyme 2 (ACE2) expression and tissue susceptibility to SARS-CoV-2 infection. Eur J Clin Microbiol Infect Dis. 2021 May;40(5):905-19. [**PMID**: 33389262]

[7] Schnaubelt S, Oppenauer J, Tihanyi D, Mueller M, Maldonado-Gonzalez E, Zejnilovic S, Haslacher H, Perkmann T, Strassl R, Anders S, Stefenelli T, Zehetmayer S, Koppensteiner R, Domanovits H, Schlager O. Arterial stiffness in acute COVID-19 and potential associations with clinical outcome. J Intern Med. 2021 Aug;290(2):437-43. [**PMID**: 33651387]

[8] Abou-Ismail MY, Diamond A, Kapoor S, Arafah Y, Nayak L. The hypercoagulable state in COVID-19: Incidence, pathophysiology, and management. Thromb Res. 2020 Oct;194:101-15. Epub 2020 Jun 20. Erratum in: Thromb Res. 2021 Aug;204:146. [**PMID**: 32788101]

[9] Markus HS, van der Worp HB, Rothwell PM. Posterior circulation ischaemic stroke and transient ischaemic attack: diagnosis, investigation, and secondary prevention. Lancet Neurol. 2013 Oct;12(10):989-98. [**PMID**: 24050733]

[10] Téllez-Villa JA, Figueroa-Casanova R, Mosos-Patiño MB, Pérez-Rivera CJ, Guarnizo-Bustamante NP, Molina-Sánchez FJ, Zapata-Diaz OE, Gil-Aldana V. Foville syndrome: a neurological emergency of clinical relevance. case report [Síndrome de Foville: Una Emergencia Neurológica de Gran Relevancia Clínica. Reporte de Caso]. Case reports. 2022 Dec;8(2):1. French.