ISSN: 2641-6271
DOI: 10.33552/OAJAP.2026.08.000690
Open Access Journal Of Addiction and Psychology
Iris Publishers

Letter to the Editor

Could Vertebrobasilar Insufficiency Represent an Overlooked Vascular Contributor to Psychiatric Disorders?

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Received Date: June 29, 2026

Published Date: July 07, 2026

Abstract

We read with great interest the growing body of literature investigating the neurobiological basis of psychiatric disorders. While neurotransmitter dysfunction, genetic susceptibility, and psychosocial factors remain central to current pathogenetic models, the potential contribution of chronic cerebrovascular insufficiency has received comparatively little attention.

Keywords: Vertebrobasilar insufficiency; Depression; Anxiety; Panic attack; Phobia; Obsessive compulsive disorder; Bipolar disorder; Schizophrenia; Schizoaffective and Personality disorders.

Introduction

In a recently published study, we identified medial temporal lobe hypoperfusion in a subgroup of patients diagnosed with vertebrobasilar insufficiency (VBI) (1). Interestingly, several of these patients also presented with clinically significant psychiatric symptoms despite the absence of structural abnormalities on conventional brain magnetic resonance imaging (MRI).

This observation may not be coincidental. The medial temporal lobe contains key components of the limbic system, including the hippocampus, amygdala, entorhinal cortex, and Para hippocampal structures. These regions are critically involved in emotional regulation, motivation, memory processing, stress responses, and social behavior. Dysfunction within limbic networks has long been implicated in major psychiatric disorders, including depression, anxiety disorders, bipolar disorder, schizophrenia, and personality disorders.

Importantly, these structures are largely supplied by branches of the posterior cerebral artery, which originates from the vertebrobasilar circulation. Consequently, chronic reductions in vertebrobasilar blood flow may lead to functional impairment of limbic circuits and contribute to the development or worsening of psychiatric symptoms, even in the absence of detectable infarction or structural brain injury.

This mechanism may help explain why some patients with VBI exhibit psychiatric manifestations that cannot be adequately accounted for by conventional neuroimaging findings. It may also explain why certain patients remain resistant to standard psychiatric interventions despite receiving appropriate pharmacological and psychological treatment.

A growing body of evidence suggests that cerebral hypoperfusion can alter neuronal metabolism, synaptic plasticity, neurotransmitter regulation, and functional connectivity within large-scale neural networks. Chronic hypoperfusion of limbic structures may therefore represent a biologically plausible mechanism linking vertebrobasilar circulatory disturbances to psychiatric symptomatology.

An important clinical challenge is that many of these patients may escape diagnosis. Because VBI frequently represents a chronic hypoperfusion state rather than an acute ischemic process, routine brain MRI often appears normal. Consequently, psychiatrists evaluating patients with treatment-resistant psychiatric disorders may remain unaware of a potentially relevant vascular abnormality unless dedicated vascular investigations are performed.

In our clinical practice, VBI is diagnosed using cervical and cerebral magnetic resonance angiography (MRA) or computed tomographic angiography (CTA), supplemented by cerebral perfusion studies. These investigations frequently reveal vascular pathology and regional perfusion deficits that are not apparent on conventional structural imaging. We generally prefer MRA because of its lower susceptibility to imaging artifacts and avoidance of ionizing radiation. However, CT perfusion offers the additional advantage of quantitatively measuring regional cerebral blood flow (mL/min/100 g of brain tissue), thereby providing an objective assessment of hypoperfusion severity.

These observations underscore the need for closer collaboration between psychiatrists, neuroradiologists, and neurosurgeons experienced in the diagnosis and management of VBI. Patients presenting with psychiatric symptoms, particularly those who are resistant to standard pharmacological and psychotherapeutic interventions, may benefit from further vascular evaluation when clinical findings suggest possible posterior circulation compromise.

The clinical significance of recognizing VBI in psychiatric patients is considerable. First, failure to identify an underlying vascular component may contribute to persistent symptoms and suboptimal treatment outcomes.

Second based on our experience, more than 90% of vascular abnormalities associated with VBI are located within the cervical vasculature. Such lesions are often more accessible to treatment than intracranial targets and may be managed with lower morbidity and lower cost than highly invasive interventions such as deep brain stimulation, stereotactic procedures, or complex neurosurgical approaches (2).

Although our observations do not establish a causal relationship between VBI and psychiatric disorders, they raise an important question: could a subset of treatment-resistant psychiatric patients harbor an unrecognized vascular disorder contributing to their symptoms? We believe this possibility deserves serious consideration and systematic investigation.

Accordingly, we propose that:

1. Awareness of vertebrobasilar insufficiency should be increased among psychiatrists and mental health professionals, regarding the potential coexistence of VBI and psychiatric disorders.

2. Vascular imaging and perfusion studies should be considered in selected psychiatric patients, particularly those with treatment-resistant symptoms or accompanying atypical clinical presentations, dizziness, vertigo, occipital headaches, visual disturbances, gait imbalance, or other features suggestive of vertebrobasilar dysfunction.

3. Multidisciplinary collaboration between psychiatrists, neuroradiologists, neurologists and neurosurgeons should be encouraged when evaluating complex cases.

4. Developing multidisciplinary treatment protocols that address both psychiatric manifestations and the underlying vascular pathology in patients with coexisting VBI and psychiatric disorders.

5. Finally, prospective studies involving larger patient cohorts are needed to determine the prevalence of medial temporal hypoperfusion among psychiatric patients, clarify the causal relationship between VBI and psychiatric symptoms, and evaluate whether correction of vertebrobasilar circulation abnormalities can improve psychiatric outcomes. Such investigations may open a new avenue in the understanding and treatment of selected psychiatric disorders with an underlying vascular component. If confirmed, this hypothesis could identify a potentially treatable vascular component in a subset of psychiatric disorders and open new avenues for both diagnosis and therapy.

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