Cerebral hemodynamics and vascular dementia: Identifying opportunities for early intervention

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SUMMARY

Vascular dementia (VaD), unlike Alzheimer's disease, is often preventable and can be slowed down or halted with early intervention. VaD results from impaired cerebral blood flow due to conditions like atherosclerosis or Moyamoya disease, leading to repeated small strokes and, consequently, cognitive decline. VaD can exist separately or co-exist with Alzheimer's disease, with the latter commonly being of a more insidious onset and involving beta amyloid protein depositions in the brain. VaD may be missed due to the focus on treating the stroke symptoms, and sometimes be interpreted as normal age-related cognitive decline. Thus, patients with vascular risk factors, who present with acute or acute-on-chronic neurological deficits that co-exist with features of subtle memory or executive functional changes, should have prompt vascular evaluation using neuroimaging.

INTRODUCTION

World Dementia Day 2025 was observed on 21st September this year with the theme "Ask About Dementia. Ask About Alzheimer's". Although Alzheimer's disease is a well-recognised subtype of dementia worldwide, the prevalence of vascular dementia (VaD) is fairly higher in Asian countries due to a larger population having cardiovascular risk factors.[1] Dementia encompasses a spectrum of progressive neurocognitive disorders characterised by impairments in memory, language, and higher executive functions. Among its various forms, Alzheimer's disease remains the most common, yet its aetiology is largely unknown and effective disease-modifying treatments remain limited. In contrast, VaD, which is also known as multi-infarct dementia, arises from cerebrovascular pathology linked to cardiovascular risk factors, and may present with either abrupt or insidious onset.[2] Importantly, VaD offers a window for intervention, particularly in younger individuals. Timely detection and management of VaD may alter disease progression in this cohort of patients.

VaD results from cumulative neuronal injury secondary to multiple cerebral infarctions, which may occur even without overt motor deficits. Clinicians should therefore maintain vigilance when encountering younger patients reporting progressive memory decline or executive dysfunction. Such presentations should prompt consideration of vascular aetiologies and appropriate diagnostic workup.

While transient ischemic attacks (TIAs) often trigger evaluation for cerebrovascular risk, there is growing evidences which support the findings of more subtle cognitive changes, particularly those involving executive function as an early indicator of cerebral hemodynamic compromise. Progressive alterations in cerebral blood flow (CBF) and cerebral perfusion have been well documented in several cerebrovascular disorders, including advanced large-artery atherosclerotic disease and Moyamoya disease.[3] These conditions are marked by chronic narrowing or occlusion of major intracranial arteries, leading to compensatory dilation of smaller vessels and the development of fragile collateral networks. Ultimately, reduced delivery of oxygen and nutrients to the brain leads to the symptoms of VaD.

To address this issue of chronic cerebral hypoperfusion, an adaptive mechanism of neovascularisation occurs, and although initially adaptive, it is prone to failure. These small-calibre vessels lack the capacity to adequately regulate CBF, rendering the brain vulnerable to episodes of hypoperfusion and subsequent neuronal loss. Impairment of cerebral autoregulation in these settings significantly elevates the risk of recurrent strokes, which can be detected on magnetic resonance imaging (MRI) brain scans. The sequelae of this compromise in CBF are progressive cognitive decline leading to VaD.

Hence, early identification of vascular contributions to cognitive impairment is crucial. Current modern neuroimaging, including both invasive and non-invasive modalities, commonly utilising MRI or nuclear medicine imaging, which can offer a detailed assessment of cerebral morphology, blood perfusion, and haemodynamics, respectively.[2,3,4] When vascular pathology is identified early, clinicians may pursue targeted interventions, ranging from aggressive risk factor modification to antiplatelet therapy or, in selected cases, surgical revascularisation such as extracranial-to-intracranial (EC–IC) bypass procedures.

As the population ages and the burden of dementia rises, it is essential for clinicians to recognise individuals at heightened risk, particularly those who smoke, consume alcohol excessively, or have a strong family history of cardiovascular disease.[5] Subtle cognitive symptoms in such patients warrant careful evaluation to identify potentially reversible or modifiable vascular causes.

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CONCLUSION

Recognition of the clinical signs combined with timely vascular risk assessment and neuroimaging remain the cornerstones of managing vascular dementia. Many modifiable risk factors can be addressed and represent critical opportunities to prevent or delay cognitive decline, reinforcing the importance of a comprehensive diagnostic approach to detect early dementia.

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