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The Evolving Role of Cardiac and Carotid Ultrasound in the Assessment of Cerebral Small Vessel Disease: A Contemporary Review

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Abstract: Cerebral small vessel disease (CSVD) is a significant contributor to vascular cognitive impairment, stroke, and functional decline in the aging population. Although brain MRI remains the diagnostic gold standard, its limitations in accessibility and early detection have necessitated the search for more widely available, non-invasive screening tools. This review synthesizes current evidence regarding the evolving role of cardiac and carotid artery ultrasonography in the assessment of CSVD. Echocardiographic parameters—particularly left ventricular concentric hypertrophy, left atrial dysfunction, and impaired diastolic filling—are consistently associated with a higher neuroimaging burden of CSVD and cognitive decline, independent of traditional risk factors. Carotid ultrasound metrics, including increased intima-media thickness, plaque burden, elevated pulsatility and stiffness indices, reduced end-diastolic velocity, and low wall shear stress, similarly correlate with lacunes, white matter hyperintensities, and enlarged perivascular spaces. Emerging evidence supports an integrated "cardiac-carotid-cerebral" hemodynamic axis, wherein dysfunction at the cardiac pump or large artery conduit level propagates microvascular injury. Combined multiparametric ultrasound profiles, enhanced by machine learning algorithms, show promise for early risk stratification and for distinguishing between CSVD etiologies. Future longitudinal studies and the standardization of advanced techniques (e.g., left atrial strain, carotid wall shear stress) are necessary to translate these ultrasound biomarkers into routine clinical practice and to test whether interventions aimed at improving cardiac or carotid function can slow the progression of CSVD.

Keywords: Cerebral Small Vessel Disease; Echocardiography; Carotid Ultrasound; Vascular Cognitive Impairment.

1. Introduction: The Intersection of Cardiac, Carotid Ultrasound, and Cerebral Small Vessel Disease

Cerebral small vessel disease (CSVD) represents a spectrum of pathological processes affecting the brain's small arteries, arterioles, capillaries, and venules. It is a leading cause of vascular cognitive impairment, stroke, and functional decline in the aging population. The diagnosis and characterization of CSVD have traditionally relied on brain magnetic resonance imaging (MRI), which visualizes parenchymal consequences such as white matter hyperintensities (WMH), lacunes, cerebral microbleeds (CMBs), and enlarged perivascular spaces (EPVS). However, MRI is not universally accessible, can be contraindicated, and often identifies disease at a relatively advanced stage. Consequently, there is a pressing need for accessible, non-invasive tools that can aid in risk stratification, early detection, and mechanistic understanding of CSVD.

Cardiac and carotid artery ultrasonography emerge as promising candidates in this context. Echocardiography provides a direct window into cardiac structure and function, which are intimately linked to cerebral perfusion and hemodynamic stress. Cardiac pathologies such as left ventricular hypertrophy, diastolic dysfunction, and atrial myopathy may contribute to cerebral hypoperfusion, increased pulsatility, and endothelial dysfunction—key drivers of CSVD. Concurrently, carotid ultrasound assesses large artery atherosclerosis, arterial stiffness, and local hemodynamic forces, which reflect systemic vascular health and directly influence cerebral circulation. The interplay between cardiac output, large artery conduit function, and cerebral microvascular integrity forms a critical pathophysiological axis in CSVD.

This review synthesizes the latest evidence on the application of transthoracic echocardiography and carotid ultrasound in CSVD. We examine specific echocardiographic and carotid parameters associated with CSVD neuroimaging burden and cognitive outcomes, explore their potential for integrated risk stratification, and discuss future directions for translating these imaging biomarkers into clinical practice.

2. Echocardiographic Parameters and Their Association with CSVD Burden and Cognitive Outcomes

Echocardiography offers a comprehensive assessment of cardiac chambers, myocardial mechanics, and systolic and diastolic function. Recent studies have elucidated specific echocardiographic signatures associated with CSVD, moving beyond traditional risk factors like hypertension to identify direct cardiac contributors.

2.1. Cardiac Structural Remodeling and CSVD Phenotype.

Left ventricular (LV) geometry, particularly concentric hypertrophy, shows a strong association with CSVD markers and cognitive decline. In a memory-clinic cohort, concentric LV hypertrophy was independently associated with a higher count of cortical cerebral microinfarcts (CMIs), a novel MRI

marker of CSVD, and with increased odds of dementia[1]. The relationship between concentric hypertrophy and cognitive function appeared to be partly mediated by cortical CMIs, suggesting a pathway where cardiac remodeling promotes cerebral micro-embolization or hypoperfusion leading to micro-infarction. Furthermore, increased left ventricular mass (LVM), a biomarker of chronic systemic hypertension, is linked to specific hemorrhagic CSVD phenotypes. Patients with intracerebral hemorrhage (ICH) due to arteriolosclerosis or mixed CSVD patterns exhibit significantly higher LVM compared to those with cerebral amyloid angiopathy (CAA), indicating that cardiac structural changes may help differentiate underlying CSVD etiologies[2]. This association extends to other arteriopathies; intracranial arterial dolichoectasia (IADE), linked to arteriolosclerotic CSVD markers, is also associated with greater LVM [3].

Left atrial (LA) pathology, assessed via size or strain imaging, provides critical insights into LA reservoir, conduit, and contractile functions. Reduced LA conduit strain (LAScd) has been specifically and independently associated with vascular cognitive impairment (VCI), but not with neurodegenerative markers, in patients without atrial fibrillation (AF) [4]. This parameter was also correlated with a higher burden of cerebral small vessel disease on MRI and circulating biomarkers of inflammation and neuronal injury. In contrast, simple LA enlargement (LAA) measured by area showed a more complex relationship in one study, where normal values were paradoxically associated with more WMH, possibly indicating earlier CSVD detection prior to cardiac structural adaptation [5]. In patients with AF-related stroke, a larger right atrial diameter was associated with a greater burden of EPVS in the centrum semiovale [6].

2.2. Cardiac Functional Parameters and Cerebral Perfusion.

LV systolic and diastolic function are fundamental determinants of cerebral perfusion. Impaired LV diastolic function, indicated by a low or high mitral inflow early-diastolic velocity/late-diastolic velocity (E/A) ratio, is consistently associated with a higher total CSVD burden, lacunes, and periventricular WMH in community-based populations [7]. This association persists even when LV systolic function, measured by ejection fraction (LVEF) or fractional shortening (LVFS), shows no significant link, highlighting the primacy of diastolic filling in maintaining steady cerebral blood flow. However, in clinical cohorts with established cognitive concerns, LV systolic dysfunction is also significantly associated with lower global cognitive scores and a diagnosis of VCI, with CSVD markers mediating about 22.5% of this association [8].

The link between cardiac function and cerebral hemodynamics is further supported by studies integrating echocardiography with arterial spin labeling (ASL) MRI. In patients with arteriosclerotic CSVD, a lower LVEF and a larger right atrial diameter were independently associated with reduced cerebral blood flow (CBF) in deep brain regions[9]. Reduced deep regional CBF itself was an independent predictor of a higher CSVD burden. This reinforces the concept of a "cardiac-cerebral hemodynamic axis" where impaired cardiac pump function leads to chronic cerebral hypoperfusion, contributing to CSVD progression.

2.3. Special Populations and Integrated Biomarkers

The relationship between echocardiographic parameters and CSVD is modified in specific clinical settings. In patients with atrial fibrillation, left ventricular posterior wall thickness (LVPW) is associated with a higher risk of lacunes and overall CSVD burden[6]. While the CHA2DS2-VASc score and left atrial diameter showed limited predictive value for cerebral microbleeds in elderly AF stroke patients overall, the score was indicative in a subgroup with diabetes mellitus[10]. Furthermore, systemic microangiopathies like Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL) exhibit cardiac involvement, with studies demonstrating blunted coronary flow reserve due to microvascular dysfunction independent of brain lesion load[11]. This underscores CSVD as a systemic disorder.

Beyond imaging parameters, circulating cardiac biomarkers like N-terminal pro-brain natriuretic peptide (NT-proBNP) are elevated in patients with recent lacunar infarcts and higher CSVD burden. Notably, this association persists independently of standard echocardiographic measures of cardiac structure and function, suggesting that NT-proBNP may capture subtler cardiac stress or neurohormonal activation relevant to CSVD pathogenesis[12].

3. Carotid Artery Ultrasound Features: Linking Atherosclerosis, Hemodynamics, and CSVD

Carotid ultrasound serves as a non-invasive tool to evaluate the structural and functional state of a major conduit to the brain. Parameters of atherosclerosis, arterial stiffness, and local hemodynamics provide complementary information about vascular health that correlates with CSVD burden and cognitive function.

3.1. Atherosclerotic Burden and Plaque Characteristics.

Carotid intima-media thickness (IMT) is a well-established marker of subclinical atherosclerosis. Increased IMT is independently associated with the presence of cognitive impairment in CSVD patients and with worse performance in attention, processing speed, and executive function[13]. Beyond IMT, the presence and characteristics of carotid plaque offer more granular risk stratification. The presence of bilateral plaque in the common carotid artery (CCA) is an independent risk factor for lacunar infarction[14]. Plaque morphology also matters; features such as maximum plaque length and thickness are significantly associated with the severity of white matter hyperintensities[15]. This suggests that the overall atherosclerotic burden in large arteries, potentially reflecting a systemic inflammatory and endothelial dysfunctional state, is intimately linked to cerebral microvascular injury.

3.2. Hemodynamic and Stiffness Parameters.

Carotid ultrasound provides dynamic measures of blood flow and vessel compliance. Peak systolic velocity (PSV) and end-diastolic velocity (EDV) are fundamental hemodynamic indices. In community studies, a lower CCA-PSV is associated with a higher total CSVD score[16]. More consistently, a reduced CCA-EDV shows robust positive

correlations with total and regional cerebral perfusion (particularly in gray matter) and is associated with a lower risk of lacunes[16]. This indicates that impaired diastolic flow in the carotid tree, possibly reflecting increased downstream microvascular resistance or reduced cardiac output during diastole, is a marker of poor cerebral perfusion and CSVD risk.

Derived indices of vascular resistance and stiffness, such as the resistance index (RI), pulsatility index (PI), and pulse wave velocity (PWV), are elevated in CSVD. Increased RI and PI of the CCA and internal carotid artery (ICA) are positively associated with lacunar infarction[14]. Stiffness parameters like the carotid-femoral PWV (cf-PWV) and the ultrasound-based PWV-β are independent predictors of lacunar infarction and are associated with cognitive impairment, attention deficits, and executive dysfunction in CSVD patients[13, 14]. Estimated PWV (ePWV), calculated from age and mean arterial pressure, is also strongly associated with WMH volume and other CSVD markers, with particularly pronounced effects in Hispanic and non-Hispanic Black individuals[17]. Elevated pulsatility and stiffness transmit excessive pulsatile energy into the fragile cerebral microvasculature, promoting endothelial damage and blood-brain barrier disruption.

3.3. Wall Shear Stress and Advanced Hemodynamic Assessment.

Emerging techniques like vector flow mapping (VFM) applied to carotid ultrasound allow estimation of wall shear stress (WSS), the frictional force exerted by flowing blood on the vessel wall. Low carotid WSS, indicative of disturbed flow and endothelial dysfunction, is negatively correlated with total CSVD scores, especially with a high burden of EPVS in the basal ganglia, and is associated with executive dysfunction[18]. Intriguingly, in this study, IMT was not associated with CSVD burden, suggesting that WSS may be a more sensitive hemodynamic marker of early microvascular injury than structural thickening.

The correlation between carotid and cerebral hemodynamics can be altered in specific diseases. In vertebrobasilar dolichoectasia (VBD), a condition of elongated and dilated basilar artery, the normal correlation between vertebral artery flow velocity and posterior circulation CBF is lost, and global brain hypoperfusion is observed[19]. This suggests impaired cerebrovascular autoregulation, linking large artery tortuosity to CSVD-like hemodynamic failure.

4. Emerging Applications in Risk Stratification and Integrated Diagnostic Approaches

The convergence of cardiac and carotid ultrasound findings is paving the way for more sophisticated, multi-parametric approaches to CSVD risk assessment, moving beyond single markers to integrated profiling.

4.1. Combined Cardiac and Carotid Profiling.

Studies increasingly demonstrate that a combination of echocardiographic and carotid ultrasound parameters offers superior predictive value. For instance, a multivariate model integrating clinical risk factors, deep regional CBF from MRI, and echocardiographic parameters (LVEF and right atrial diameter) showed high predictive accuracy for identifying patients with a high CSVD burden[9]. Similarly, carotid ultrasound parameters such as IMT, β-stiffness index, and PWV-β are independently related to a moderate-to-severe CSVD burden score[13]. These findings suggest that a "vascular health check" combining cardiac and carotid ultrasound could identify individuals at high risk for significant CSVD before the onset of overt neurological symptoms or advanced MRI changes.

4.2. Machine Learning for Risk Stratification.

To handle the complexity of multi-parameter data, machine learning (ML) approaches are being explored. One study developed ML models using routine echocardiographic and hematological parameters (e.g., fibrin formation rate) to stratify patients into low and high CSVD burden groups, achieving high discriminative performance (AUC up to 0.885) with Random Forest algorithms[20]. This approach highlights the potential of leveraging routinely available clinical and imaging data to create accessible screening tools for CSVD risk in settings where MRI is not feasible.

4.3. Unveiling Systemic Microvascular Disease.

Ultrasound findings are also being contextualized within the framework of systemic microvascular rarefaction—a reduction in functional capillary density. Observational study protocols like the CRUCIAL study aim to link non-invasive assessments of sublingual microcirculation and retinal vascular density (as potential surrogates) with advanced cardiac and brain MRI markers in patients with vascular cognitive impairment and heart failure with preserved ejection fraction (HFpEF)[21]. This paradigm positions CSVD not as an isolated brain disorder, but as one manifestation of a widespread microvascular failure syndrome, with cardiac and carotid ultrasound serving as accessible windows into this systemic process.

4.4. Clinical Workflow and Differential Diagnosis.

Carotid and cardiac ultrasound play practical roles in the diagnostic workflow for CSVD mimics. For example, a patent foramen ovale (PFO)-associated stroke can be misdiagnosed as CSVD if only routine transthoracic echocardiography is used, underscoring the need for more sensitive techniques like contrast-enhanced transcranial Doppler or transesophageal echocardiography in cryptogenic cases[22]. Furthermore, as noted, echocardiographic measures like LVM may help in phenotyping the likely underlying arteriopathy (arteriolosclerosis vs. CAA) in patients with hemorrhagic CSVD[2].

5. Conclusion and Future Directions

Cardiac and carotid ultrasound have evolved from tools assessing isolated organ function to integral components in the evaluation of cerebral small vessel disease. Echocardiography reveals that structural changes like LV concentric hypertrophy and LA dysfunction, as well as functional impairment particularly in diastole, are robustly associated with CSVD neuroimaging burden and vascular cognitive impairment. Carotid ultrasound extends this assessment to the conduit arteries, linking atherosclerotic burden, increased arterial stiffness, and altered hemodynamics (especially reduced diastolic flow and low wall shear stress) to microvascular brain injury.

The emerging evidence supports an integrated model where cardiac pump performance, large artery buffering capacity, and cerebral microvascular integrity are inter-dependent. Dysfunction at any level can propagate injury downstream, contributing to the pathogenesis of CSVD. The clinical promise lies in using these accessible, non-invasive ultrasound parameters for early risk stratification, monitoring disease progression, and potentially as surrogate endpoints in therapeutic trials targeting vascular contributors to cognitive decline.

Future research should focus on several key areas: First, large-scale longitudinal studies are needed to establish the temporal relationship and predictive value of specific ultrasound parameters for incident CSVD and cognitive decline. Second, standardization of advanced techniques like LA strain and carotid WSS measurement is crucial for widespread clinical adoption. Third, further integration of multi-modal data—combining ultrasound, circulating biomarkers, and brief cognitive tests—through validated algorithms or machine learning models could create powerful point-of-care assessment tools. Finally, elucidating whether interventions that improve cardiac function (e.g., specific heart failure therapies) or carotid hemodynamics can slow CSVD progression will be the ultimate test of this pathophysiological linkage, opening new avenues for preventing vascular cognitive impairment.

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