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Transcranial Color Doppler Ultrasound and Chronic Kidney Disease

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ABSTRACT

Chronic kidney disease (CKD) and hemodialysis (HD) are associated with significant cerebrovascular complications, including impaired cerebral autoregulation, reduced cerebral blood flow (CBF), and increased risk of stroke and cognitive decline. Both kidney and brain share similar vascular vulnerabilities, with hemodynamic instability playing a central role in the development of cerebrovascular damage. This minireview discusses the pathophysiological mechanisms linking CKD, HD, and cerebrovascular disease, highlighting the impact of impaired CBF and autoregulation, as well as the role of advanced imaging techniques like transcranial Doppler (TCD) in assessing cerebral hemodynamics. We also examine the effects of HD on CBF, cognitive function, and the development of white matter lesions.

Keywords: Chronic Kidney Disease; Hemodialysis; Cerebrovascular Disease; Cerebral Blood Flow; Transcranial Doppler; Cerebrovascular Reactivity

Abbreviations: CKD: Chronic Kidney Disease; HD: Hemodialysis; CBF: Cerebral Blood Flow; TCD: Transcranial Doppler; GFR: Glomerular Filtration Rate; CAD: Coronary Artery Disease; IMT: Intima-Media Thickness; ASL: Arterial Spin Labeling; eGFR: Glomerular Filtration Rate; ESRD: End-Stage Renal Disease; MFV: Mean Flow Velocity; UF: Ultrafiltration Volumes; TCCD: Transcranial Color Doppler; PCA: Posterior Cerebral Artery; ACA: Anterior Cerebral Artery; ICA: Internal Carotid Artery; PSV: Peak Systolic Velocity; EDV: End-Diastolic Velocity; CVR: Cerebrovascular Reactivity; PACNS: Primary Central Nervous System Vasculitis

Introduction

A reduction in glomerular filtration rate (GFR) is associated with a 40% increase in the risk of cerebrovascular events and impairs cerebral autoregulation, facilitating vascular remodeling and a decrease in cerebral blood flow (CBF) [1-6]. From a cardiovascular perspective, reduced renal function is strongly correlated with the development of coronary artery disease (CAD): GFR is a significant predictor of carotid intima-media thickness (IMT), atherosclerosis progression, and fatal vascular events. Patients with chronic kidney disease (CKD) have a high prevalence of carotid artery calcifications and unstable plaques, increasing the risk of cerebrovascular embolisms [2,7,8]. The brain and kidneys share a considerable metabolic demand at rest and tightly autoregulated blood flow. In CKD, vascular remodeling may impair local cerebral blood flow autoregulation, rendering white matter particularly vulnerable to ischemic injury. Advanced imaging techniques, such as arterial spin labeling (ASL), allow non-invasive quantification of CBF and offer new insights into the mechanisms linking CKD and cerebrovascular disease. CKD is defined by an estimated glomerular filtration rate (eGFR) of less than 60 mL/min/1.73 m^2 or the presence of albuminuria.

Both markers are associated with increased cardiovascular mortality but have distinct implications for cerebrovascular risk: albuminuria is correlated with both ischemic and hemorrhagic stroke, whereas reduced eGFR is primarily associated with ischemic stroke. The risk of cerebrovascular and cardiovascular events further increases when CKD patients reach end-stage renal disease (ESRD) and begin hemodialysis (HD). Hemodynamic variations induced by the extracorporeal fluid removal process make HD particularly stressful for

organs with high blood flow demands, such as the kidneys and brain, both characterized by similar microvascular regulation. Changes in blood flow and blood composition during HD increase vascular permeability and endothelial dysfunction, contributing to the development of cerebrovascular pathologies, including stroke, closely linked to hemodynamic instability [6-8]. Patients on HD have an incidence of stroke that is 8–10 times higher than the general population, with a greater prevalence of hemorrhagic events (around 20%) [1-5]. The initiation of chronic therapy appears to represent a particularly critical moment: the incidence of stroke increases in the three months preceding the start of HD and peaks within the first 30 days of treatment [9]. It is hypothesized that repeated reductions in CBF during dialysis sessions may contribute to ischemic brain lesions, which are associated with cognitive decline [10].

The risk of coronary artery disease also increases after the onset of HD, progressively worsening over time [11]; carotid artery disease has been identified as a predictor of cognitive decline [5]. Overall, available evidence suggests that hemodynamic instability induced by HD exacerbates vascular damage and promotes cognitive decline. Monitoring cerebral perfusion in HD patients can be performed using Transcranial Doppler (TCD), a non-invasive tool easily applicable at the bedside. Studies on HD patients have shown, through TCD, a reduction in the mean flow velocity (MFV) correlated with ultrafiltration volumes (UF) [12]. Most research has focused on hemodynamic variations during dialysis sessions, comparing pre- and post-HD CBF. However, there is a lack of studies exploring cerebral perfusion and autoregulation on non-dialysis days, when the patient is not affected by waste accumulation or acute hemodynamic stress induced by HD.

Technical Principles of TCCD

Transcranial color Doppler (TCCD) is a non-invasive ultrasound technique that provides real-time evaluation of cerebral circulation by analyzing blood flow. Its high reproducibility and the ability to be performed at the patient's bedside have made it widely used in intensive care settings, where it serves as a critical tool in many critical conditions, such as intracranial hypertension, post-subarachnoid hemorrhage vasospasm, hydrocephalus, acute cerebral ischemia, and brain death diagnosis [8]. TCCD combines two-dimensional imaging, color Doppler, and pulsed Doppler, allowing precise identification of the major cerebral arteries and the analysis of the velocity spectrum. The low-frequency phased array probe (2–2.5 MHz) enables ultrasound penetration through bony structures. The main acoustic windows are: the transtemporal window, which insonates the mesencephalon, diencephalon, third ventricle, and Willis circle (middle cerebral artery - MCA, anterior cerebral artery - ACA, posterior cerebral artery - PCA); the transorbital window, for the ophthalmic artery and carotid siphon; the suboccipital window, for the vertebral and basilar arteries; and the submandibular window, useful for evaluating the extracranial portion of the internal carotid artery (ICA) and calculating the Lindegaard index (Mean MCA flow velocity / Mean ICA flow velocity). Doppler analysis focuses on peak systolic velocity (PSV), end-diastolic velocity (EDV), and mean flow velocity (MFV), from which resistance and pulsatility indices (RI and PI) are derived. The latter, correlated with cerebrovascular resistance and intracranial pressure, normally ranges between 0.5 and 1.2 [8,13-16].

Cerebral and Renal Hemodynamics in CKD

Recent studies have shown that CKD is associated with profound alterations in both cerebral and renal hemodynamics, with a pathophysiological parallelism related to the transmission of aortic pulsatility. In an analysis conducted on 110 adults without chronic kidney disease and 66 nephropathy patients, Kosaki, et al. [17] evaluated cerebral and renal blood flow via TCD and duplex ultrasound, along with aortic pressure differential (via tonometry). It was found that in CKD patients, there is greater renal pulsatility (increased PI and RI); however, no significant differences were observed in cerebral blood flow, although PI and RI indices were correlated between the two groups after adjustments for age, sex, and nephropathy ($rs \approx 0.64–0.70$, $p < 0.001$) [17]. The aortic pressure differential was an independent predictor of pulsatility indices in both districts. In a study by Belluardo et al. on hemodialysis patients (HD), additional alterations were found: reduced MFV, increased cerebrovascular reactivity, diffuse carotid atherosclerosis, and a high prevalence of cognitive deficits [18]. The correlation between Doppler indices (MFV, EDV, BHI) and cognitive performance suggests a role for chronic hypoperfusion and vascular stiffness in cognitive decline during HD.

The reading of these two studies defines a pathophysiological continuum: in the early stages of nephropathy, the transmission of aortic pulsatility alters renal and cerebral hemodynamics; in advanced stages of the disease, cerebral hypoperfusion, autoregulatory dysfunction, carotid atherosclerosis, and cognitive decline emerge. This view highlights the shared vascular vulnerability of the brain and kidneys and the utility of combined monitoring of both districts in risk stratification [9-12,17-20]. Several systemic factors (anemia, hypertension, diabetes) can impair cerebrovascular autoregulation. However, some studies with continuous TCD monitoring show that mean cerebral blood flow remains surprisingly stable during dialysis sessions, despite significant hemorheological variations. Even in cases of intradialytic hypotension, cerebral perfusion does not undergo significant changes, suggesting preserved autoregulatory capacity in the short term. These data, however, contrast with results from other studies that documented a reduction in CBF during HD sessions, a phenomenon correlated with the development of diffuse subcortical white matter lesions and cognitive dysfunction [10]. Interventions aimed at improving hemodynamic stability, such as better management of dialysate temperature, appear to mitigate such damage, suggesting a potential neuroprotective effect. Although the circulatory stress induced by HD constitutes a plausible ischemic insult, it often occurs in the context of already impaired cerebrovascular function, making the brain particularly vulnerable to damage. In this scenario, cerebro-

vascular reactivity (CVR) has emerged as a non-invasive indicator of cerebral vascular functionality. CVR reflects the capacity of cerebral vessels to dilate or constrict in response to vasomotor stimuli, typically changes in CO_2 levels. Preserved CVR would counteract reductions in CBF induced by HD, thereby reducing the risk of ischemic damage [10,11]. Clinical assessment of CVR requires a method to measure CBF—such as transcranial Doppler or magnetic resonance imaging—and a stimulus capable of altering CO_2 levels. CVR is expressed as the ratio between the change in CBF and the change in CO_2, and abnormal values have been associated with cerebrovascular diseases.

Reduced CVR has been linked to an increased risk of stroke, cognitive decline, and cortical thinning. In conditions like concussion, CVR discriminates between symptomatic individuals and controls, even when CBF at rest is similar; in leukoaraiosis, it represents an early functional marker of at-risk brain tissue, predicting progression toward white matter hyperintensities [20]. A recent pilot study compared CVR in HD patients, CKD patients, and healthy controls via TCD and controlled hypercapnia: HD patients exhibited significantly reduced CVR compared to the other groups; the deficit was independent of blood pressure, cardiac output, or dialysis duration, with no significant differences between healthy subjects and non-dialytic CKD patients; in CKD, CVR was not correlated with renal function. These findings suggest that hemodialysis exposes the brain to greater ischemic vulnerability during the hemodynamic fluctuations typical of the procedure [20]. Certainly, a full understanding of the immediate and long-term effects of hemodialysis (HD) on cerebral circulation, brain structure, and cognitive function remains limited. Recent studies have attempted to clarify these interactions using multimodal methodologies integrating hemodynamic, neuropsychological, and neuroimaging assessments. A large prospective observational study conducted on 97 adults undergoing chronic hemodialysis (median age 59 years) monitored real-time cerebral blood flow dynamics using transcranial Doppler, assessing mean flow velocity (MFV) during the entire dialysis session. Cognitive function was tested both during and outside dialysis sessions, with a 12-month follow-up.

Brain MRI was used to measure atrophy, white matter hyperintensity burden (WMH), and diffusion parameters, correlating these outcomes with changes in MFV and cognitive performance. During dialysis, a significant reduction in MFV was observed, proportional to ultrafiltration volumes. The extent of this decline was associated with intradialytic cognitive decline, involving global, executive, and verbal fluency functions. After one year, 73 patients were re-evaluated, and 34 underwent a new MRI. In patients who continued dialysis, greater reduction in MFV correlated with lower cognitive scores and progression of WMH load, indicating worsening small vessel disease. A subgroup of 15 patients who underwent kidney transplantation provided a valuable comparison. In the 12 patients who were assessable before and after transplantation, memory performance (delayed recall test) improved significantly. Additionally, increased fractional anisotropy of white matter—an indicator of microstructural integrity—was correlated with improved executive functions. Overall, these data confirm that HD induces transient reductions in cerebral blood flow associated with acute cognitive dysfunction, while prolonged exposure to dialysis is associated with progressive cerebrovascular deterioration. In contrast, kidney transplantation seems to partially reverse these alterations, with cognitive recovery and improvement in brain diffusion parameters [9-12,17,18,20]

Other Nephrological Applications of TCCD

Another interesting field for transcranial Doppler could be vasculitis. Currently, the literature does not provide strong evidence, but studies converge in establishing that in primary central nervous system vasculitis (PACNS) and systemic vasculitis with cerebral involvement (e.g., Takayasu arteritis, polyarteritis nodosa, giant cell arteritis), TCD can identify accelerated blood flow due to inflammatory stenosis and that it can be useful for monitoring therapeutic response, showing normalization of flow velocity with effective immunosuppressive therapy. Potential applications are also recognized in evaluating micro- and macrovascular changes in type 2 diabetes, but extensive data on diabetic nephropathy are lacking. PI and mean flow velocities (Vm) might be two useful markers for the detection of diabetic cerebrovascular changes [21-23]. The increase of PI of ICA and MCA might reflect the microangiopathic changes of cerebral vessels in diabetic patients [24].

Conclusion

Transcranial Doppler represents a valuable diagnostic tool in the integrated study of cerebral hemodynamics, with crucial applications in evaluating vascular changes associated with CKD and hemodialysis. Current evidence indicates that the kidney and brain share similar vascular vulnerabilities, with vascular pulsatility and aortic stiffness playing a central role in the hemodynamic parallelism between the two organs. Therefore, transcranial monitoring represents a privileged window into the vascular pathophysiology of CKD, with potential implications for risk stratification and the prevention of cognitive decline. Many fields of application remain underexplored, and TCD can now be integrated with other non-invasive imaging techniques, such as MR angiography or CT perfusion, for a multimodal approach to diagnosing and following up on cerebrovascular vasculitis.

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