

Cognitive Dysfunction Across the Chronic Kidney Disease Continuum: Current Evidence, Clinical Implications, and Future Directions

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ABSTRACT

Background and Objective:

Cognitive impairment is a prevalent and under-recognized phenomenon associated with chronic kidney disease (CKD). It becomes more common as kidney function declines, peaking among patients on dialysis. However, few studies have systematically compared the cognitive profile and clinical implications of cognitive dysfunction between non-dialysis CKD and dialysis-dependent kidney failure. This narrative review aimed to collate and compare recent findings regarding the clinical characteristics of cognitive impairment associated with non-dialysis CKD and dialysis.

Methods: A structured narrative review was performed. Databases searched included PubMed/MEDLINE, Scopus, Web of Science and Google Scholar (1 January 2020 to 15 May 2026). Eligible reports included cognitive performance data in adults with non-dialysis chronic kidney disease (CKD), haemodialysis (HD) or peritoneal dialysis (PD).

Results: Pooled prevalence is approximately 32% in non-dialysis chronic kidney disease (CKD), 39% in peritoneal dialysis (PD) and 53% in haemodialysis (HD). Executive function, attention and processing speed are most commonly impaired, with relative preservation of memory and language until later stages. Accumulation of uremic toxins, small-vessel cerebrovascular disease, inflammation, oxidative stress and Blood–Brain-Barrier disruption contributes to cognitive decline in kidney disease. In HD, recurrent intradialytic hypotension and osmotic stress are additional accelerators of white-matter injury and cognitive decline with increased temporal variability. Cognitive impairment is an independent predictor of non-adherence to treatment, low health-related quality of life, hospitalisation and mortality.

Conclusion: Cognitive impairment represents a clinical challenge in chronic kidney disease. Current evidence suggests that routine screening with sensitive cognitive tests and multidisciplinary management can help mitigate cognitive decline in these patients. There is a need for future research comparing longitudinal cognitive profiles, cognitive batteries specific for CKD, and interventional trials targeting potentially modifiable dialysis-related factors.

Keywords: Chronic kidney disease; Cognitive impairment; Haemodialysis; Peritoneal dialysis; Kidney–brain axis; Uremic toxins; Neuroinflammation

1. INTRODUCTION

Chronic kidney disease (CKD) is a global health problem affecting over 850 million people worldwide and is among the leading causes of death from preventable disease. In addition to cardiovascular, metabolic, and hematologic abnormalities, CKD is increasingly recognized as a cause of central nervous system damage. Cognitive deficits ranging from mild executive dysfunction to severe dementia are common manifestations of CKD that accelerate mortality (1,2). The burden of cognitive dysfunction is not distributed evenly. The prevalence and severity increase with decreasing eGFR and after the start of dialysis. Deficits in patients with non-dialysis CKD are typically mild to moderate, gradually developing, and less severe compared to those observed in patients on dialysis, who experience additional physiological load due to dialysis itself, namely intradialytic hypotension, fluid balance shifts, and inadequate removal of uremic toxins during dialysis, all of which contribute to more severe and variable cognitive deficits. Patients on peritoneal dialysis seem to be in an intermediate position. Therefore, the cognitive status of CKD patients should be evaluated considering their treatment stage, since the relative contribution of disease-associated and dialysis-associated factors to cognitive decline can differ significantly (3,4).

Although several systematic reviews and meta-analyses have been conducted to explore the overall prevalence or mechanisms of cognitive dysfunction, only a limited number of studies have compared the cognitive phenotype, pathophysiological pathways, neuroimaging findings, or clinical consequences across the non-dialysis and dialysis continuum, or between haemodialysis (HD) and peritoneal dialysis (PD). The present narrative review aimed to provide a focused comparison of cognitive dysfunction in non-dialysis chronic kidney disease (CKD) against dialysis patients (5,6).

1.1 Objectives

The specific aims of this review are to:

1. Summarise quantitative prevalence estimates of cognitive impairment stratified by CKD stage and dialysis modality.
2. Contrast the cognitive domains most affected in non-dialysis versus dialysis populations.
3. Integrate shared and modality-specific pathophysiological mechanisms.

4. Evaluate current assessment strategies and their practical limitations in each setting.
5. Identify clinical implications and priority research gaps arising from the comparative perspective.

2. LITERATURE SEARCH AND REVIEW METHODOLOGY

This article is a structured narrative review aiming to provide a synthesis of the cognitive dysfunction literature across the CKD continuum. The narrative review was chosen as the design to allow for critical appraisal of the heterogeneity of study designs, cognitive batteries, and populations included in the studies.

2.1 Search Strategy

Electronic searches were performed in PubMed/MEDLINE, Scopus, Web of Science and Google Scholar. The Boolean search strategy used was as follows:

("chronic kidney disease" OR CKD OR "end-stage renal disease" OR ESRD OR "kidney failure") AND ("cognitive impairment" OR "cognitive dysfunction" OR cognition OR dementia OR "mild cognitive impairment") AND (dialysis OR haemodialysis OR haemodialysis OR "peritoneal dialysis" OR "non-dialysis" OR "pre-dialysis")

Medical Subject Heading (MeSH) terms such as Chronic Kidney Disease, Cognitive Dysfunction and Renal Dialysis were combined with free-text synonyms. The search was limited to English language and human adult studies. The time window for the search was set between 1 January 2020 and 15 May 2026. This window was selected in order to identify recent high-quality evidence on the association between chronic kidney disease and cognitive decline/dysfunction. Nevertheless, it allowed to include most of the relevant systematic reviews and large-scale studies published up to the present date and which served as the backbone of the current research. Earlier landmark studies were also considered if they appeared in the reference lists of the selected articles or recent

2.2 Eligibility Criteria and Synthesis Approach

Eligible studies comprised observational cohorts, cross-sectional surveys, randomised trials, systematic reviews and meta-analyses reporting on cognitive outcomes in adults with chronic kidney disease (CKD) stages 1-5, haemodialysis (HD) or peritoneal dialysis (PD). Paediatric cohorts, acute kidney injury populations and mechanistic studies lacking human relevance were

excluded. Evidence was thematically organised according to the comparison questions specified in the objective. Where possible, quantitative prevalence estimates were primarily extracted from the largest meta-analyses available. Domain-specific cognitive patterns and mechanistic relationships were qualitatively synthesised across study designs. Study design limitations, heterogeneity in cognitive domains assessed and potential residual confounding were explicitly considered in the interpretation of findings.

3. EPIDEMIOLOGY: PREVALENCE AND DOMAIN PATTERNS ACROSS THE CONTINUUM

3.1 Prevalence Estimates by Disease Stage and Treatment Modality

A 2024 systematic review and meta-analysis of 50 studies with 25,289 patients reported an overall pooled prevalence of cognitive impairment of 40% (95%CI 33-46%). Prevalence estimates varied considerably depending on the treatment group: 32% (95%CI 25-38%) in non-dialysis chronic kidney disease (CKD), 39% (95%CI 26-53%) in patients on peritoneal dialysis (PD), as high as 53% (95%CI 38-69%) in those on haemodialysis (HD), and 26% (95%CI 15-38%) after kidney transplantation. The estimates from restricted analyses to maintenance HD patients suggest that approximately 50% (95%CI 49-51%) of them have some degree of cognitive impairment (4,5).

Also, prevalence increases sharply with a decrease in the level of kidney function before the initiation of dialysis. According to one study, the prevalence rates were 10% for estimated glomerular filtration rate (eGFR) ≥ 60 ml/min/1.73 m², 47% for eGFR 30-60 ml/min/1.73 m², and 61% for eGFR < 30 ml/min/1.73 m². Therefore, cognitive decline risk increases significantly even at an advanced stage of chronic kidney disease (CKD) before dialysis and becomes especially prominent after dialysis initiation.

The prevalence of geographic differences is substantial, with notably higher pooled prevalence rates in Africa (approximately 58%) and Asia (approximately 44%) compared to Europe (34%) and North America (37%). Distinctions in the studied case mix, levels of education, accessibility to healthcare, and screening instruments utilized appear to drive these variations. Significantly, the choice of screening tool plays a pivotal role; the higher pooled prevalence rate using the Montreal Cognitive Assessment (MoCA) exceeds that of the Mini-Mental State Examination (MMSE) by

approximately 20% (57% vs. 27%). Such a gap may be attributed to the inadequate ability of the MMSE to capture the executive dysfunction and attention deficits characteristic of CKD (5).

3.2 Domain-Specific Cognitive Profiles in Non-Dialysis and Dialysis Populations

Across the entire continuum of chronic kidney disease or CKD, the cognitive functions, which are most frequently impaired, are executive functioning, attention, and processing speed. These three areas are especially susceptible to subcortical vascular injury and metabolic derangements, with uraemia being a part of this. In contrast to these, both episodic memory and language are relatively preserved till advanced stages of the disease where dialysis takes over. Working memory is, however, significantly impaired from the start of dialysis.

Table 1 below represents the comparative pattern of the domains of cognitive impairment. In non-dialysis chronic kidney disease (CKD), there is a mild to moderate deficit of cognitive performance which is usually manifested first within the executive domain and progresses slowly. In haemodialysis (HD), similar domains are severely impaired which are mostly unstable during the dialysis cycle with a deterioration of function immediately after dialysis. Peritoneal dialysis (PD) has an intermediate pattern of cognitive impairment with less temporal instability and overall, better preserved cognitive function compared to HD possibly due to more stable haemodynamic (7).

Table 1. Domain-specific cognitive impairment in non-dialysis CKD versus dialysis populations

Cognitive domain	Non-dialysis CKD	Haemodialysis	Peritoneal dialysis
Executive function	Mild to moderate; early involvement	Marked and progressive	Intermediate; more stable than HD
Attention	Reduced, especially in advanced stages	Significantly impaired; often worse post-HD	Impaired but less fluctuating
Processing speed	Slowed in stages 4–5	Severely reduced and fluctuating	Moderately reduced

Working memory	Mild deficits	Moderate impairment	Mild to moderate
Episodic memory	Largely preserved early	Mild to moderate decline	Relatively preserved
Language	Generally preserved	Mild impairment in late stages	Preserved
Visuospatial ability	Minimal	Moderate in some cohorts	Variable
Temporal variability	Low; gradual progression	High; linked to dialysis cycle	Low to moderate

3.3 Comparison of Haemodialysis and Peritoneal Dialysis

The head-to-head comparisons of the outcomes of patients who had received PD or HD in terms of cognitive function have been consistently reporting better results for PD than for HD. First, a meta-analysis including 11 studies comprising nearly 200,000 patients reported that the relative risk (RR) for cognitive impairment was 0.63 (95% CI, 0.51-0.79) for PD versus HD. Second, a more recent meta-analysis aggregating 26 studies encompassing more than 326,000 patients found that the standardized mean difference (SMD) was -0.46 (95% CI, -0.62 to -0.29) for cognitive function in favour of PD and an odds ratio (OR) was 1.68 (95% CI, 1.25-2.25) for incident dementia. The risk of dementia was reported to be higher in the HD group. However, the possible confounding by indication should be noted because the HD-treated patients were apparently older and had more prevalent comorbidities. On the other hand, the same beneficial effect of PD was found in the subgroup analysis of patients under 65 years old (8).

4. PATHOPHYSIOLOGY: SHARED AND DIALYSIS-SPECIFIC MECHANISMS

Cognitive decline among patients suffering from chronic kidney disease is multifactorial and complex. Several key pathways are involved that cut across the disease continuum for the majority of the patients. Moreover, there is a list of additional factors responsible for cognitive decline that occur once the dialysis treatment has started. The differentiation between core mechanisms and

dialysis-related ones is critical in order to understand how the severity differs in each case and what interventions are required (9,10).

4.1 Shared Mechanisms Across Non-Dialysis and Dialysis CKD

As glomerular filtration rate decreases, protein-bound uremic toxins, for example, indoxyl sulfate and p-cresyl sulfate, as well as other low and middle molecular weight toxins, accumulate in the circulation. They cause dysfunctions in neuronal signalling, increase oxidative stress and mitochondrial damage, and reduce cerebral blood flow via injuring endothelial cells. These toxins are partially cleared by conventional haemodialysis, leading to their accumulation and subsequent worsening of CNS manifestations following dialysis (11,12).

The microvasculature of the kidney and the brain have several similarities, because of which both these systems tend to experience such complications as endothelial dysfunction, arterial stiffness, and impairment of autoregulation, all of which contribute to chronic cerebral hypoperfusion. The latter manifests itself through a series of signs and symptoms, including white-matter hyperintensity, lacunar infarction, and cortical thinning, which can be detected using an MRI. The development of such foci in the CNS is common in both non-dialysis patients with ESKD and patients on dialysis. Inflammation and oxidative stress that characterize CKD play a major role in the pathogenesis. It also leads to the activation of microglia and the release of pro-inflammatory cytokines, resulting in additional damage to neurons, as well as the disruption of the integrity of the blood-brain barrier, allowing uremic toxins and inflammatory mediators to penetrate. This interaction takes place already at stages 4 and 5 of non-dialysis CKD, as well as on dialysis (9,13). Diabetes, hypertension, anaemia, and depression are some of the most common comorbidities that accompany CKD and can independently lead to declines in cognitive function through multiple mechanisms. The interactions of various factors lead to significant additional cognitive impairments during both non-dialysis and dialysis treatment.

4.2 Dialysis-Specific Mechanisms

Once renal replacement therapy is initiated, several other important contributors to stress that are rarely encountered among non-dialysis CKD patients emerge. Intradialytic hypotension, frequently encountered during haemodialysis, leads to repetitive episodes of cerebral hypoperfusion, which, when occurring on top of impaired autoregulation, lead to progressive white-matter injury. Moreover, increased ultrafiltration rate may challenge cerebral autoregulation

capacity resulting in subclinical brain damage, even in the absence of hypotension. Acute alterations in osmolality and electrolytes during dialysis lead to temporary cognitive dysfunction, often referred to as dialysis encephalopathy syndrome; only partial recovery takes place during the interdialytic period, and these cognitive deficits accumulate with each dialysis session. Finally, incomplete removal of protein-bound and middle molecules during dialysis results in continuous exposure to neurotoxins, despite the dialysis procedure being otherwise regular (13).

Peritoneal dialysis does not lead to rapid haemodynamic and osmotic adjustments associated with intermittent haemodialysis, thereby reducing acute cerebrovascular burden. Nonetheless, continuous glucose exposure from dialysate and the risk of peritonitis may add additional metabolic and inflammatory burden. Overall, the risk is lower for peritoneal dialysis when compared to haemodialysis (14).

4.3 Integrated Conceptual Model

Shared uremic, vascular and inflammatory mechanisms create a substrate of subcortical cognitive vulnerability that intensifies with decreasing eGFR. Exposure to haemodialysis adds further recurrent ischaemic and osmotic challenges to the mix, resulting in more severe and temporally variable white-matter injury typical of incident haemodialysis patients (15). The less aggressive peritoneal dialysis procedure falls in between. The effect of age, education, frailty and co-morbidity combines across these stages in a manner similar to that observed in the Figure 1.

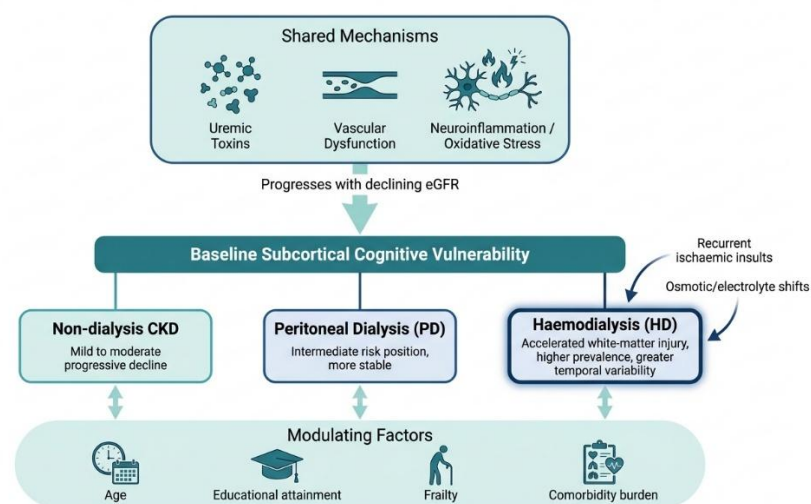


Figure 1. Integrated pathophysiological model of cognitive dysfunction across the CKD continuum.

5. NEUROIMAGING CORRELATES OF COGNITIVE IMPAIRMENT IN CKD

Structural magnetic resonance imaging consistently reveals alterations of white-matter hyperintensities, decreased grey-matter volume, and cortical thinning in patients with advanced non-dialysis chronic kidney disease (CKD) and those on dialysis. These findings are most closely linked to executive dysfunction and processing speed deficits, with a greater burden of white-matter hyperintensity and lower grey-matter volume typically observed in haemodialysis patients, presumably reflecting cerebral hypoperfusion during dialysis. In addition, reduced hippocampal volume is associated with memory impairment in these patients (16,17).

Unlike standard MRI, diffusion tensor imaging (DTI) offers unique insights into microstructural injury by providing specific biomarkers, including fractional anisotropy and mean and radial diffusivity. Reduced fractional anisotropy was consistently found across the continuum of chronic kidney disease (CKD) with a pooled effect size of approximately -0.70 (95% CI, -0.84 to -0.56), indicating widespread disruption of white-matter integrity, whereas mean and radial diffusivity were increased. DTI abnormalities were more severe in dialysis-dependent than in nondialysis CKD and partially reversed after kidney transplantation in patients receiving dialysis (18). Arterial spin labeling MRI revealed increases in cerebral blood flow, predominantly in the hippocampus and thalamus, in non-dialysis CKD, unlike the pattern of hypoperfusion observed in Alzheimer's disease; this pattern can be modified by dialysis or transplantation (19).

Resting-state functional MRI showed decreased amplitude of low-frequency fluctuation and disrupted functional connectivity in the default-mode network, salience network, and executive-control network. Coupling between cerebral haemodynamic and neuronal activity was also impaired in the hippocampus and basal ganglia, and an altered susceptibility in the basal ganglia discovered by quantitative susceptibility mapping may emerge as a potential biomarker of cognitive risk (20,21). Overall, neuroimaging studies suggest that CKD is associated with a predominantly vascular and subcortical pattern of injury that becomes more severe from non-dialysis to haemodialysis patients, with peritoneal dialysis exhibiting an intermediate effect (22,23). There is a need for future research employing standardised protocols and longitudinal designs examining the transition from non-dialysis to dialysis maintenance haemodialysis, as

illustrated in Fig. 2 the transition from non-dialysis to dialysis remain important priorities, as seen in Figure 2.

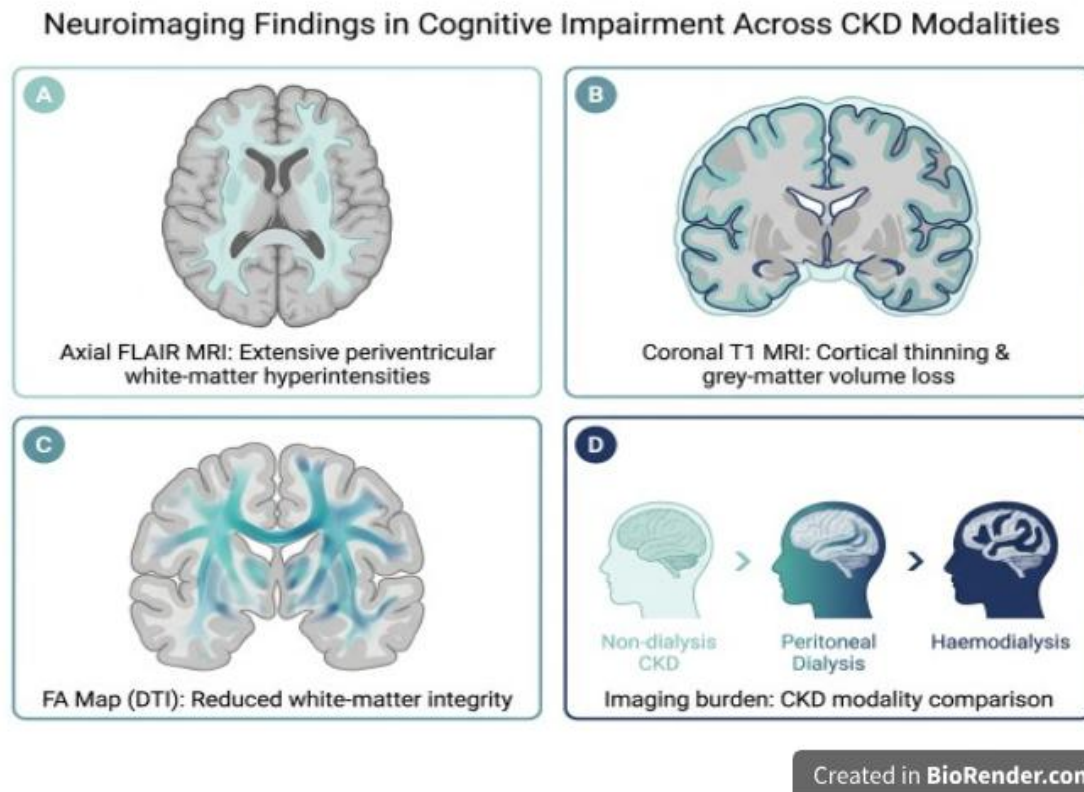


Figure 2. Neuroimaging findings associated with cognitive impairment across the CKD continuum

6. ASSESSMENT AND SCREENING OF COGNITIVE FUNCTION IN CKD

Because the majority of cognitive deficits in chronic kidney disease are related to dysfunction of higher-order cognitive abilities such as executive function, attention, and processing speed rather than memory or orientation, cognitive screening instruments that emphasize these domains are more appropriate than the standard Mini-Mental State Examination. The Montreal Cognitive Assessment (MoCA), Trail Making Test Part B (TMT-B), and Digit Symbol Substitution Test (DSST) have been found to have better sensitivity in this population. Table 2 lists common cognitive screening batteries and their utility in patients with chronic kidney disease. (24,25)

Table 2. Cognitive screening instruments relevant to CKD clinical practice

Instrument	Time	Key domains	Sensitivity in CKD	Practical notes
MoCA	10 min	Executive, memory, attention, visuospatial	High	Preferred first-line tool; education adjustment needed
MMSE	5–10 min	Orientation, memory, language	Low for executive deficits	Not recommended as sole screening tool in CKD
3MS	15 min	Global cognition	Moderate	Useful in research; longer administration time
TMT-B	5 min	Executive function, processing speed	High	Sensitive to dialysis-related change; requires training
DSST	5 min	Processing speed, attention	High	Useful supplementary test; less education-biased

MoCA = Montreal Cognitive Assessment; MMSE = Mini-Mental State Examination; 3MS = Modified Mini-Mental State; TMT-B = Trail Making Test B; DSST = Digit Symbol Substitution Test.

The timing of tests is of great importance in haemodialysis. The cognitive performance may be significantly variable during the interdialytic interval, being frequently lower after dialysis sessions, thus tests should be scheduled preferably in the middle of the week and before dialysis. In contrast, in non-dialysis chronic kidney disease and peritoneal dialysis patients, the timing is less relevant; however, stability of performances is more evident when the same time interval is used for the assessments. The influence of the patient's education, level of language proficiency, impaired hearing, and depression should also be considered. No one test is optimal, and probably a pragmatic approach that uses a brief screen followed by more extensive assessment of specific cognitive domains if the initial test is abnormal is most feasible in a busy clinical practice (26,27).

7. CLINICAL IMPLICATIONS OF THE COMPARATIVE PERSPECTIVE

7.1 Treatment Adherence, Self-Management and Safety

Executive dysfunction and attention deficits are strongly associated with the multiple self-management tasks required by patients with CKD, including medication adherence, diet and fluid restriction, and, in patients undergoing peritoneal dialysis, adherence to aseptic procedures. Poor adherence to these measures is linked to increased progression of disease and to hospitalization. Furthermore, because executive dysfunction is both more common and more severe in patients receiving haemodialysis, the relative risk of these negative patient outcomes is even greater for these patients than for those on peritoneal dialysis (28,29).

7.2 Quality of Life, Functional Independence and Mortality

Cognitive impairment is independently associated with lower health-related quality of life, loss of functional independence, increased caregiver burden, higher rate of hospitalization, and increased risk of all-cause and cardiovascular mortality in patients on non-dialysis and dialysis. The question as to whether cognitive dysfunction is an epiphenomenon of overall burden of disease or it is causally implicated in the process is yet to be completely elucidated; however, its impact on outcome is evident (30).

7.3 Implications for Dialysis Modality Selection and Prescription

When the maintenance of cognitive function is a priority, for instance, in young people, people with cognitive deficits, or those with a high level of self-care, peritoneal dialysis may be preferable to hemodialysis (31,32). For patients undergoing hemodialysis, measures to minimize the risk of intradialytic hypotension such as dialysate cooling, reduced ultrafiltration rates, longer or more frequent dialysis, and careful management of dry weight may help reduce cerebral damage. Thus, despite the lack of randomized trials, cooled dialysate, which has been tested in feasibility trials, can be considered an option for preserving cognitive function (33,34).

7.4 Multidisciplinary Care and Caregiver Involvement

Optimal management requires multidisciplinary approach including nephrologists, neurologists or geriatricians, psychologists, dieticians and nursing staff. Regular cognitive screening should be performed which allows to evaluate drug therapy (excluding anticholinergics and other cognition-lowering medications), anaemia and blood pressure control, depression treatment course and caregiver education (35).

7.5 Kidney Transplantation

Kidney transplantation often improves attention, memory, and executive function by normalizing metabolic homeostasis, but some deficits related to preexisting cerebrovascular disease and neurotoxicity of immunosuppressive drugs are often observed. Moreover, cognitive performance after kidney transplantation requires monitoring (36,37).

8. CRITICAL APPRAISAL OF THE EVIDENCE BASE

Several limitations in the present literature base must be recognized. Firstly, the majority of the included studies are cross-sectional, and little evidence comes from longitudinal studies following patients from an advanced non-dialysis CKD through the transition to dialysis initiation. Secondly, the cognitive domains assessed using different cognitive batteries differ between studies, limiting the possibility for quantitative pooling and comparisons between cognitive domains (38,39). In observational studies comparing the cognitive outcomes of haemodialysis and peritoneal dialysis initiation, there is often an insufficient control for potential confounders such as age, educational level, comorbidity, and indication for dialysis (40). Publication bias and reporting bias cannot be excluded. Finally, few randomised trials have investigated interventions specifically targeting the preservation of cognitive function in chronic kidney disease (41,42).

These limitations do not negate the overall finding of the evidence; cognitive decline was common, was present across the range of kidney function, and was increased in prevalent haemodialysis patients. They do, however, necessitate caution in interpreting the specific prevalence estimates and the comparison of different dialysis modalities (43,44).

9. FUTURE DIRECTIONS AND RESEARCH PRIORITIES

Several areas of research seem to be of particular interest in this review, based on the comparison of the provided articles. First, there is a need for prospective cohort studies enrolling patients who present with advanced non-dialysis chronic kidney disease (CKD) and follow them through the transition to dialysis, with repeated cognitive assessments and neuroimaging at multiple time points (45,46). Second, developing and validating a brief cognitive battery specifically targeting domains relevant to executive and attentional functions, which could be feasibly implemented in the dialysis unit setting, would help standardize cognitive evaluations and advance both clinical practice and research (47).

Randomised trials of haemodynamically stabilising dialysis strategies, such as dialysate cooling, increased frequency or duration of dialysis and ultrafiltration profiling, with cognition as a pre-specified endpoint, are needed to identify potentially modifiable treatment factors (48). Concurrently, discovery studies on candidate biomarkers, such as uremic solutes, inflammatory mediators and neurodegenerative markers, may facilitate early identification of patients at risk. Lastly, implementation science studies are needed to develop and evaluate the effectiveness of screening algorithms and multidisciplinary management pathways in clinical practice (49,50,51). The research priorities and corresponding study designs have been summarised in Table 3.

Table 3. Priority research gaps and recommended study designs

Research gap	Current limitation	Recommended direction
Longitudinal comparative data	Dominance of cross-sectional designs	Multi-centre cohorts spanning the non-dialysis to dialysis transition
Standardised assessment	Heterogeneous instruments and timing	CKD-specific battery with protocolised timing relative to dialysis
Modality effect causality	Confounding by indication	Emulated trials or carefully matched prospective designs
Intervention evidence	Few randomised trials with cognitive endpoints	Haemodynamic optimisation and multi-domain prevention trials
Biomarkers and imaging	Limited clinical integration	Prospective studies correlating imaging, cognition and uremic toxins

10. CONCLUSION

Cognitive impairment is a prevalent, progressive and prognostically important feature of the CKD continuum. Quantitative data demonstrate a clear gradient in burden: approximately one-third of patients with non-dialysis CKD, nearly two-fifths of those treated with peritoneal dialysis, and more than half of patients receiving haemodialysis meet criteria for cognitive impairment. Executive dysfunction, attentional deficits and slowed processing speed dominate the clinical picture. Shared uremic, vascular and inflammatory mechanisms operate throughout the continuum. Haemodialysis adds recurrent haemodynamic and osmotic insults that further accelerate cognitive

decline, while peritoneal dialysis occupies an intermediate position. Recognition of these comparative differences should inform routine screening practice, modality counselling, dialysis prescription and the design of future neuroprotective trials. Integrating standardised cognitive assessment into nephrology care represents a feasible and necessary step toward more patient-centred management of CKD.

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