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Preserving Mobility and Cognition in Elderly CKD Patients: Community-Based Strategies from Japan

Kazuhiko Tsuruya

Nara Medical University, Japan

Japan is the world's most rapidly aging society, with more than 28% of its population aged ≥ 65 years. Consequently, the increasing burden of chronic kidney disease (CKD) among older adults has made geriatric nephrology a cornerstone of clinical practice. In this "super-aged" society, the goals of CKD management have shifted from merely delaying disease progression or prolonging survival to preserving functional status—particularly mobility and cognitive function—which are essential for maintaining independence, quality of life, and appropriate healthcare utilization. Emerging evidence indicates that CKD is closely linked to frailty, sarcopenia, and cognitive impairment through multiple interrelated mechanisms, including vascular dysfunction, chronic inflammation, oxidative stress, uremic toxins, and mineral-bone disorders. These processes contribute significantly to both physical and cognitive decline in older patients. In dialysis patients, additional factors such as intradialytic hypotension and cerebral hypoperfusion further exacerbate neurological vulnerability, placing elderly individuals at heightened risk of falls, disability, institutionalization, and mortality. Cognitive impairment is increasingly recognized as a critical domain that requires systematic assessment and targeted intervention. Key strategies to support cognitive health include routine screening, medication review to minimize polypharmacy, and caregiver education. Although brain atrophy is known to progress in patients with CKD, data comparing different dialysis modalities remain limited. We previously reported that brain atrophy progresses more rapidly in patients undergoing hemodialysis (HD), partly attributable to intradialytic hypotension. To address this gap, we conducted brain magnetic resonance imaging (MRI) in patients with non-dialysis-dependent CKD (ND-CKD) and those with end-stage kidney

disease on peritoneal dialysis (PD) and HD, and compared longitudinal changes in the gray matter volume ratio (GMR) using statistical parametric mapping (SPM). The annual decline in GMR was significantly greater in PD patients than in both ND-CKD and HD patients. Furthermore, we found a significant association between GMR and Trail Making Test (TMT) scores, highlighting the impact of brain atrophy on executive function. We also investigated the underlying mechanisms using a CKD mouse model, in which impaired spatial working memory and increased hippocampal oxidative stress were observed. Notably, these abnormalities were ameliorated by treatment with the antioxidant tempol or the renin-angiotensin system inhibitor telmisartan. Additionally, vascular calcification plays a critical role in clinical outcomes among elderly CKD patients. It increases arterial stiffness and contributes to hemodynamic instability, which may indirectly accelerate cognitive decline. We evaluated the progression of the coronary artery calcium score (CACS) in patients with ND-CKD, PD, and HD. Our results showed that CACS progressed more rapidly in dialysis patients than in those with ND-CKD, and was significantly faster in PD patients compared with HD patients. These findings suggest that the unique metabolic environment and fluid volume status associated with PD may promote accelerated vascular calcification. They underscore the importance of stringent mineral and fluid management to protect both cardiovascular and cerebrovascular health. In this lecture, we will review our most recent findings on cognitive impairment, brain atrophy, and vascular calcification in CKD—particularly in elderly patients—and provide a comprehensive overview of the relevant clinical and epidemiological evidence.

Keywords: Geriatric nephrology, Cognitive impairment, Brain atrophy, Vascular calcification, Oxidative stress