Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
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Clinical Insights on Nutrition and Sarcopenia in CKD

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Sarcopenia in chronic kidney disease (CKD) is considered a clinical phenotype that significantly contributes to adverse outcomes. Its prevalence increases with declining kidney function and is strongly associated with frailty, hospitalization, and mortality. Importantly, sarcopenia in CKD reflects a distinct pathophysiological process characterized by metabolic, inflammatory, and nutritional derangements. The underlying mechanisms are multifactorial. Chronic low-grade inflammation plays a pivotal role by activating proteolytic pathways such as the ubiquitin–proteasome system while suppressing muscle protein synthesis. Insulin resistance further exacerbates anabolic resistance, limiting the effectiveness of dietary protein intake. Metabolic acidosis, commonly observed in advanced CKD, promotes muscle protein breakdown through glucocorticoid-mediated pathways. In addition, the accumulation of uremic toxins disrupts mitochondrial function and accelerates oxidative stress, further contributing to muscle loss. Hormonal alterations, including reduced levels of anabolic hormones (e.g., growth hormone), also impair muscle maintenance. From a nutritional standpoint, protein-energy wasting (PEW) is a key driver of sarcopenia in CKD. Reduced appetite, dietary restrictions, and systemic inflammation collectively lead to inadequate intake of energy and protein. However, nutritional management in CKD presents a clinical paradox: while sufficient protein intake is necessary to preserve muscle mass, excessive protein consumption may accelerate kidney function decline in non-dialysis patients. Therefore, individualized protein prescriptions based on CKD stage and metabolic status are essential. Recent clinical evidence supports a more tailored approach to dietary management. Adequate energy intake must be prioritized to prevent catabolism, as insufficient caloric intake leads to the utilization of endogenous protein stores. The quality of protein is also critical; essential amino acids, particularly leucine, play a key role in

stimulating muscle protein synthesis. Plant-dominant dietary patterns are gaining attention due to their anti-inflammatory properties and potential to reduce metabolic acidosis, while maintaining nutritional adequacy when appropriately planned. Adjunctive strategies include the use of ketoacid analogues in combination with low-protein diets, which may help maintain nitrogen balance while minimizing renal burden. Supplementation with vitamin D and omega-3 fatty acids has also been explored for their potential benefits on muscle metabolism and inflammation, although the evidence is not yet conclusive. The integration of resistance exercise is crucial to overcoming anabolic resistance and improving muscle strength and physical function. Clinically, early identification of sarcopenia using validated tools such as grip strength, gait speed, and muscle mass assessment should be incorporated into routine CKD care. This allows for timely intervention before significant muscle loss occurs. A multidisciplinary approach involving nephrologists, dietitians, and exercise professionals is essential for effective management. In conclusion, sarcopenia in CKD is a clinically significant and multifactorial condition that may be attenuated through targeted interventions. Moving beyond traditional nutrient restriction, a precision nutrition framework that integrates nutritional adequacy, dietary quality, and physical activity offers a promising strategy to improve patient outcomes.

Keywords: chronic kidney disease , metabolic acidosis, muscle wasting , nutritional intervention , sarcopenia