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Sarcopenia in Patients with Chronic Kidney Diseases

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ABSTRACT

Background: Sarcopenia, characterized by a progressive decrease in skeletal muscle mass and decline in muscle function, is a common factor amongst patients with chronic kidney disease (CKD). Purpose of review: This narrative review summarizes the most recent evidence regarding the epidemiology, pathophysiology, clinical implications, diagnostic criteria, and therapeutic strategies for sarcopenia in CKD patients. Methods: The study used a recent literature synthesis method. Results: 25-50% of patients with CKD have the presence of sarcopenia. Major contributors are chronic inflammation, oxidative stress, and alterations in hormone signaling. It has been advocated that muscle strength is a more reliable predictor of poor outcomes than muscle mass. Conclusion: Early recognition and an organized approach play an important role in enhancing their clinical course.

Keywords: Sarcopenia, chronic kidney disease, muscle strength, renal rehabilitation, inflammation

1. INTRODUCTION

Sarcopenia is characterized by an age-related, generalized, yet slow loss of muscle mass and muscle strength. As of 2019, it is now recognized officially as a specific musculoskeletal disease, with a focus on muscle function rather than just muscle mass (Cruz-Jentoft & Sayer, 2019). The prevalence of sarcopenia among CKD patients is disproportionately higher than that seen in the general population. It can involve 25% of all CKD patients and as many as 50% of patients on dialysis, in a meta-analysis of studies involving over 17,000 patients through 2023. Weakened muscle force was noted in up to 50% of the cases (Duarte et al., 2024).

Skeletal muscle atrophy in CKD results from the interplay of myriad metabolic and inflammatory processes that favor catabolism, while inhibiting anabolic repair processes (Wang & Mitch, 2014). Consequent protein depletion is a significant factor contributing to worse outcomes for this patient group.

2. REVIEW METHODS

The literature review period was from January to June 2024. The PubMed, Scopus, and Web of Science databases were used to conduct an extensive search using the keywords "sarcopenia," "chronic kidney disease," "muscle strength," "renal rehabilitation," and "inflammation." We limited our search to articles published in English from 2014 to 2024, with priority given to recent meta-analyses, clinical trials, and consensus statements. Duplicates, titles, and abstracts were screened for relevance to sarcopenia in CKD patients. Studies that focused on pediatric populations, animal models, unrelated musculoskeletal disorders, or did not address CKD-specific aspects were excluded. Full-text review was then applied to the remaining articles, and 16 publications meeting the inclusion criteria were selected for synthesis. These references correspond to the complete list cited in the present review. The selection process is described in a Prisma Consort Chart (Figure 1).

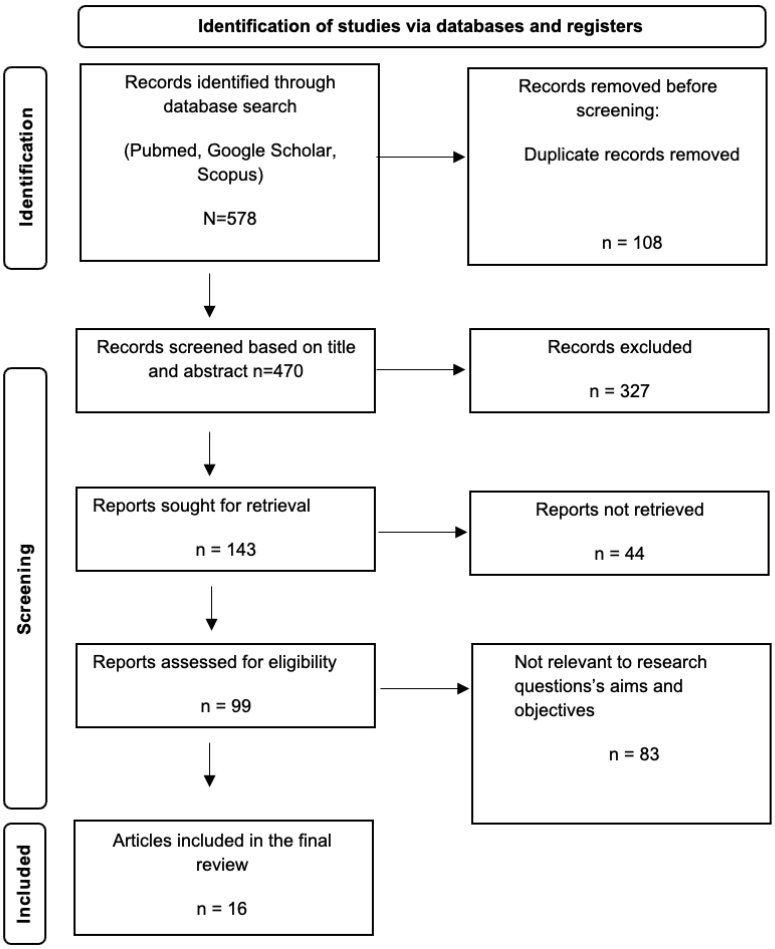

Figure 1. Prisma chart consort

3. RESULTS & DISCUSSION

Pathophysiology

Understanding the pathophysiology of sarcopenia in chronic kidney disease (CKD) is not straightforward. Researchers identified several molecular pathways that appear to be involved. For example, the Akt/mTOR cascade and insulin-like growth factor-1 (IGF-1) signaling are believed to regulate the synthesis and breakdown of muscle proteins. On the other hand, proteolytic systems, which include the ubiquitin–proteasome pathway and enzymes, for example, caspase-3, appear to drive muscle loss by promoting protein degradation (Ju & Yi, 2023). Inflammatory cytokines like tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), along with oxidative stress, increase muscle protein breakdown (Wang & Mitch, 2014; Cheng et al., 2022).

Myostatin levels and IGF-1 expression mediated impaired functioning of satellite cells, contributing to the compromise of muscle regeneration (Cheng et al., 2022). Other pathophysiologic factors, such as metabolic acidosis, insulin resistance, and hyperactivity of the renin–angiotensin–aldosterone system, play a role in a vicious circle whereby loss of muscle homeostasis occurs. A list of key biological mechanisms of CKD-associated sarcopenia is provided in Table 1.

Table 1: Biological Mechanisms in CKD-Associated Sarcopenia

Mechanism	Description
Insulin/IGF-1 Resistance	Impaired anabolic signaling and synthesis
Akt/mTOR Suppression	Disruption of protein synthesis pathways
Ubiquitin–Proteasome Pathway	Protein degradation escalated
Cytokines (TNF-α, IL-6)	Promote muscle catabolism
Oxidative Stress	Damage to muscle tissue

Clinical Consequences

Decreased muscle strength in CKD is a better predictor of mortality and poor functional outcomes than muscle mass (Isoyama et al., 2014). The drop in physical performance is linked to a faster rate of disease progression to ESRD and higher use of healthcare services (Ibrahim et al., 2016). Bleeding and iron deficiency are common, and afflicted sufferers often have serious fatigue, are immobile, and increasingly reliant on aid in activities of daily living, and suffer heavy indirect quality of life losses. The estimated prevalence of sarcopenia by CKD stage is shown in Table 2.

Table 2: Estimated Prevalence of Sarcopenia by CKD Stage

CKD Stage	Estimated Prevalence (%)
Non-dialysis (3–4)	20–30%
Dialysis (stage 5D)	40–50%
All CKD (mean)	24.5%

Assessment in Clinical Practice

The diagnostic process of sarcopenia is usually algorithmically based and multi-staged. Potential at-risk individuals are identified through initial screening tools, including the SARC-F questionnaire and the gait speed test. Muscle function is subsequently measured by handgrip dynamometry and muscle mass by imaging techniques, such as dual energy-X-ray absorptiometry (DXA) or bioelectrical impedance analysis (BIA) (Cruz-Jentoft et al., 2019; Guida et al., 2020). From the assessment of severity, physical performance tests: 4-meter walking speed, Short Physical Performance Battery (SPPB), and Timed Up and Go (TUG) test are used.

Therapeutic Interventions

Currently, resistance training is the only intervention with solid evidence that it can slow or even reverse sarcopenia in people living with CKD. Research has shown that this type of exercise may raise muscle mass and strength by about 13–31%, depending on how frequently and intensely it is performed (Geneen et al., 2022). Aerobic activities, which are primarily designed to benefit the heart and lungs, have also shown some modest but functional improvements in muscle function (Ma et al., 2022). When resistance and endurance exercises are combined, the effects tend to complement each other, as they work on separate aspects of muscle health and together may produce stronger overall results (Heiwe & Jacobson, 2014); (Figure 2).

In addition to exercise, nutritional strategies deserve attention. Ensuring sufficient protein intake — and, in selected cases, using keto-analog supplements to help reduce nitrogen load — has been highlighted as an essential part of dietary planning (Narasaki et al., 2024). A number of new medications are being tested, including agents that block myostatin and others with anabolic effects, and these could potentially expand the ways we treat the condition in the coming years (Cha, 2024; O'Sullivan et al., 2018).

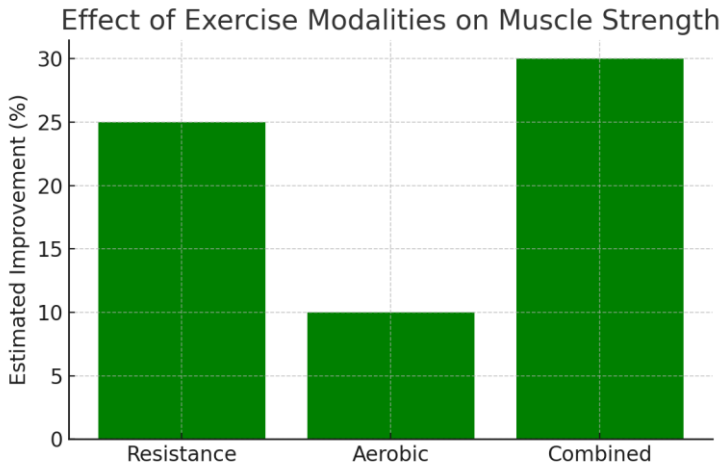

Figure 2: Estimated Muscle Strength Improvement by Exercise Type

Guideline Recommendations

Regular exercise is recommended by kidney disease: Improving Global Outcomes (KDIGO) 2024 guidelines for comprehensive CKD care. They advise treating patients without any contraindications with >150 min of moderate-intensity physical activity/week and two or more times/week muscle-strengthening activities (KDIGO, 2024). These modifiable factors target maintenance of physical function, quality of life, and possibly sarcopenia-related decline.

4. CONCLUSION

Sarcopenia is a common but potentially reversible condition in CKD patients. Its early identification and selective management, specifically with resistance-based and multimodal rehabilitative interventions, have the potential to greatly impact clinical recovery and the overall health status of the patients. Going forward, the field is in great need of the development of personalized intervention protocols, molecular target investigation, and improved incorporation of rehabilitative care in routine nephrology care.

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Author's Contribution

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Conflict of interest
The authors declare that there is no conflict of interest.

Data and materials availability
All data associated with this work are present in the paper.

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