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Pathophysiology and Potential Treatment of Uremic Sarcopeni

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Uremic sarcopenia, a significant complication associated with chronic kidney disease (CKD), involves the degradation of skeletal muscle mass, strength, and function, severely impacting quality of life and increasing all-cause mortality. Our current findings highlight the molecular mechanisms and potential interventions for managing this muscle-wasting condition. Research has identified the pivotal role of endoplasmic reticulum stress and the unfolded protein response (UPR) in the development of sarcopenia, exacerbated by uremic toxins such as indoxyl sulfate (IS) in CKD and advanced glycation end-products (AGEs) in diabetes mellitus (DM). IS impairs myoblast differentiation and proliferation, promoting muscle atrophy through mechanisms that include the upregulation of atrogin-1 and modification of myogenic markers. It is noteworthy that IS exposure leads to divergent effects on UPR signaling of phospho-eIF2α and XBP1, influencing myoblast differentiation. Similarly, AGEs contribute to muscle atrophy in DM, a condition that shares similarities with uremic sarcopenia. Administering alagebrium chloride to inhibit AGE formation represents a promising strategy for mitigating muscle dysfunction associated with diabetic myopathy. Further insights from the studies indicate that alendronate, typically used for osteoporosis, also exhibits potential in combating muscle loss by modulating sirtuin-3 (SIRT3) pathways, thus providing dual benefits for bone and muscle health. Additionally, low-intensity pulsed ultrasound (LIPUS) has emerged as a novel, non-invasive strategy to ameliorate muscle wasting in CKD, enhancing muscle mass and strength by restoring Akt activation and reducing atrogenic markers. Collectively, these studies demonstrate the complexity of uremic sarcopenia's pathophysiology and foster a multidisciplinary approach to treatment, ranging from pharmacological interventions to physical therapy techniques. Each strategy contributes uniquely to understanding the mechanisms driving muscle loss in CKD, paving the way for the development of comprehensive treatments to improve patient outcomes in uremic sarcopenia.

Keywords: sarcopenia, uremia, intervention