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Low muscle mass in patients receiving hemodialysis: correlations with noncoronary vascular calcification and the incidence of vascular access failure

Seok-hyung Kim, Gwangho Choi, Jong-woo Yoon, Hyunsuk Kim
Department of Internal Medicine-Nephrology, Chuncheon Sacred Heart Hospital, Korea, Republic of

Objectives: Sarcopenia is characterized by an age-related decline of skeletal muscle mass with low muscle strength or functional disability. Vascular calcification (VC) occurs commonly and is associated with cardiovascular disease in chronic kidney disease (CKD) patients. We aimed to investigate the correlations of low muscle mass with the quantified vascular calcification score (VCS) of the arm of vascular access, as well as whether low muscle mass is associated with incidence of vascular access failure.

Methods: The VCS was measured on non-contrast arm CT using the Agatston method. The lower muscle mass (LMM) group was defined as subjects whose skeletal muscle mass of the lower extremities measured by bioelectrical impedance was lower than the median. Higher VC was defined as a score of 500 or higher, corresponding to the highest 40% of VCS. Univariate and multivariate logistic regression analyses were used to explore the association between LMM and VC.

Results: Seventy-five patients were included, of whom 42 (56.0%) were men. The median age was 64 years (interquartile range 58-72 years). The median vintage of hemodialysis was 49.4 months (range 32.1-99.2 months). There were no significant differences between the non-LMM and LMM groups in sex, ESRD etiology, and type of vascular access. However, age and hemodialysis vintage were significantly older in the LMM group. LMM was associated with VC (hazard ratio [HR] 3.562; 95% CI 1.341-9.463, $P = 0.011$). After adjusting for hemodialysis vintage, systolic blood pressure, and diabetes, LMM was independently associated with VC (HR 10.415, 95% CI 2.357-46.024, $P = 0.002$). The risk of vascular access failure was higher in LMM group (HR 3.652, 95% CI 1.135-11.749, $P = 0.03$). VC was a full mediator in the relationship between LMM and recurrent vascular access failure.

Conclusions: LMM increased the risk of VC and may be suggested as a potential predictor of vascular access failure.