Significant Loss of Skeletal Muscle Mass Occurs after Femoral Fragility Fracture

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Femoral fragility fractures lead to significant loss of physical function and independence because of skeletal muscle atrophy after injury. Factors that drive muscle loss are physical inactivity and nutrition deficiencies. Baseline inadequate nutrition, restricted oral intake after injury, and increased metabolic demand required for fracture healing result in critical nutrient deficiencies. The body compensates by entering a catabolic state, breaking down healthy, functional muscle mass. A better understanding of how muscle mass responds to injury is needed to critically evaluate nutrition and rehabilitation interventions designed to prevent muscle loss and optimize functional recovery for these older adults. The purpose of this study was to document sarcopenia, nutrition status, and changes in muscle mass following femoral fragility fractures.

METHODS: A two-center prospective observational study at two hospitals (Level 1 trauma center and Level 2 regional medical center) enrolled individuals ≥65 years old admitted for operative fixation of a low-energy femoral fracture. Baseline body composition was assessed within 72 hours of admission using multifrequency bioelectrical impedance and repeated 6 and 12 weeks after injury. Sarcopenia was defined by gender-specific cutoffs for the appendicular skeletal muscle mass index (ASMI), <6.3 ASMI for females and <8.5 ASMI for males. Malnutrition was defined by Mini Nutritional Assessment®. Wilcoxon Signed Rank test was used to assess 6 and 12-week change from baseline in lean body mass (LBM) and skeletal muscle mass (SMM). Wilcoxon Rank Sum test was used to compare the change in body composition in those with versus without malnutrition and sarcopenia. LBM and SMM results are presented as median (interquartile range).

RESULTS:

Thirty participants (27% male) age 76.9±8.9 years were enrolled. At baseline, 11 (37%) were sarcopenic, and 17 (59%) had baseline malnutrition.

Six weeks after injury, LBM and SMM decreased 2.81kg (-6.03 to -0.3), p<0.001) and 1.75kg (-3.30 to -0.40, p<0.001), respectively. By 12 weeks, participants lost 5.19kg of LBM (-7.94 to -2.15, p<0.001) and 3.04 kg of SMM (-4.49 to -1.45), p<0.001).

Participants with adequate baseline nutrition status lost more LBM at 6 weeks compared to those with malnutrition (-6.32 kg (-8.26 to -1.85) versus -2.31 kg (-3.50 to -0.20), p=0.042). While no difference was observed in LBM loss between those with versus without sarcopenia, the prevalence of sarcopenia increased 22% by 12 weeks after injury.

DISCUSSION AND CONCLUSION: Femoral fragility fractures resulted in devastating losses of lean body mass and skeletal muscle mass 12 weeks after injury. Inadequate baseline nutrition was common, but participants with adequate baseline nutrition lost more muscle mass, indicating that future investigations of interventions to prevent muscle loss should focus on all fragility fracture patients regardless of nutrition status. These results highlight the need for further investigation into interventions to mitigate muscle loss after injury.