Iron metabolism abnormality in skeletal muscle atrophy associated with chronic renal failure

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Introduction and aims: Skeletal muscle atrophy is often observed in chronic renal failure (CRF) patients. However, the molecular mechanism of skeletal muscle atrophy in CRF has remained unclear. Iron is an essential trace metal for all living organisms. On the other hand, excessive iron catalyzes the formation of highly toxic hydroxyl radicals via the Fenton reaction. The purpose of this study was to determine whether iron is involved in CRF-related skeletal muscle atrophy.

Methods: In this study, we divided 8-weeks-old C57BL/6J mice into two groups: vehicle-treated group (control mice) and adenine-injected group (CRF mice).

Results: Iron content was elevated in the skeletal muscle in CRF mice. Although the expression of divalent metal transporter 1 did not change, the expression of transferrin receptor and ferroportin were downregulated in the skeletal muscle in CRF mice. The expression of ferritin heavy chain and ferritin light chain were upregulated in the skeletal muscle in CRF mice. CRF mice showed increased oxidative stress in the skeletal muscles.

Conclusions: These results suggest that iron accumulation mediated oxidative stress has the potential to accelerate skeletal muscle atrophy in CRF.