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LETTER

Cortisol and the muscle-bone axis: response to comments by Molfino et al.

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Dear Editor,

The points raised by Molfino et al. [1] in their comment to our article [2] open a new scenario for the management of skeletal fragility in patients with endogenous hypercortisolism. Sarcopenia is a well-known complication of glucocorticoid excess [3] potentially involved in the pathogenesis of fragility fractures [4]. However, the clinical challenge is to understand whether and how sarcopenia reverts after resolution of hypercortisolism. Most of the considerations highlighted in our recent paper [2] concerning the outcome of skeletal fragility in patients with cured hypercortisolism may be also applied to sarcopenia. As a matter of fact, there is evidence to suggest that muscle structure and function may persist abnormal for several months after remission of hypercortisolism [5], prompting the search for specific therapeutic approach to sarcopenia.

As correctly indicated by Molfino et al., adequate nutrition, physical activity [6], and vitamin D supplementation [7] have anabolic effects on bone and muscles, improve muscle function, reduce risk of falls and fractures, and should be employed to reduce the catabolic effects of hypercortisolism on bone and muscle. Furthermore, dietary supplementation with omega 3 fatty acids has been suggested to be useful for prevention and treatment of sarcopenia [8] and osteoporosis [9]. Moreover, in frail elderly, exercise and amino acid supplementation induce an increase in bone and muscle mass and muscle strength in sarcopenic elderly [6]. The cost-effectiveness of these therapeutic approaches in the specific clinical context of endogenous hypercortisolism is still largely unknown and need to be clarified. Moreover, we believe that further research is needed to identify substances acting on bone and muscle that could counteract the catabolic effects of hypercortisolism on these tissues.

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