Obesity, sarcopenia and postmenopausal osteoporosis: An interlinked triad!

Obesity has been conventionally, considered as protective toward bone health. Currently, this view is being challenged and obesity is considered as detrimental to bone health. In the present issue of Journal of Midlife Health, the author, whereas reviewing the epidemiological studies and pathophysiological mechanisms of obesity-related osteoporosis, emphasizes the emerging evidence about the adverse effect of obesity on bone health.[1] A positive relation has been shown of ectopic fat and serum lipids with bone marrow fat in young men and women.[2] A negative correlation has recently been shown between truncal fat (TF) and bone mineral density (BMD).[3] The authors also showed that a TF was directly related to insulin insensitivity and inflammatory cytokines.[3] Prevalence of metabolic syndrome (MS), known to be strongly associated with insulin resistance (IR) and inflammatory cytokines, is high amongst peri- and post-menopausal (PM) Indian women.[4-6] Obesity and MS, having common determinants such as IR and inflammatory cytokines,[7] would thus (probably) be closely associated with osteoporosis in a large number of PM Indian women. The first question then one would want to ask is what would be the bone health in metabolically healthy obese (MHO) PM women? One wonders if the earlier observation of obesity being protective toward bone health had emerged because the individuals observed were MHO.

The second question to be asked is whether skeletal muscles play any role in this obesity-related osteoporosis. In a comparative cross-sectional study of PM women with and without osteoporosis, a reduction in muscle strength (back flexor and extensor strength) was observed in women with osteoporosis as compared to those without osteoporosis.[8] A significant correlation existed between appendicular skeletal muscle (ASM) mass relative to height and trabecular as well as cortical bone geometry and microstructure. The authors find low levels of insulin-like growth factor-binding protein 2 in relation with low relative ASM in men and women similar to the low levels earlier they reported with low BMD and high bone resorption markers. How does ASM exert its influence on bone or vice a versa? It is only recently that muscle is being recognized as an endocrine organ that secretes myokines.[9] Mark Hemrick has hypothesized and provided some evidence for muscle-secreted factors (myokines) that influence bone health via endocrine and paracrine pathways besides ASM exerting its mechanical influence on the bone mass.[10,11] Their group have identified several osteogenic myokines such as insulin-like growth factor-1 and fibroblast growth factor-2 at the muscle-bone interface that participates in bone formation. Myostatin, an anti-ostogenic myokine, exerts inhibitory effect on the muscle and causes bone loss. Myostatin-inhibitors induce increase in muscle mass and bone density and loss of fat.[12] However, human data are sparse and more studies are required to assess the clinical application of myostatin-inhibitors in management of sarcopenic obesity and associated cardiometabolic dysfunction. Similarly, growth hormone-releasing hormone analogues have also been proposed as possible therapy for Sarcopenic Obesity (SO) group.[13]

Meanwhile, it is important that we identify women with sarco-obese osteoporotic postmenopausal women and offer them a well-tailored energy-restricted, high protein diet with resistance exercise program.[14-16]

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