OBESITY IN POSTMENOPAUSAL OSTEOPOROSIS

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Introduction. Obesity is a chronic disease that is closely correlated with bone mineral density (BMD), resulting in contradictory effects. The aim of this study is to assess the potential risks in overweight individuals postmenopause and to identify new therapeutic targets for improving quality of life.
Materials and Methods. To achieve the proposed objective, a review of the scientific literature of 10 bibliographic sources from the last 5 years was carried out, using the search engines PubMed, Cochrane Library, MedScape, Biomed Central.
Results. Obesity, characterized by an excessive accumulation of adipose tissue, influences the morbidity of patients with osteoporosis. Adipokines, osteoblasts and chondroblasts originate from pluripotent mesenchymal stem cells (MSCs), suggesting the correlation between adipose tissue and bone. Lower concentration of adiponectin in obese individuals stimulates osteoblast synthesis through production of receptor activator of factor kappa-B ligand (RANKL), decreases osteoprogerin secretion. Leptin, a hormone derived from subcutaneous adipose tissue, directly influences bone remodeling through specific receptors on the surface of osteoblasts and chondroblasts, as well as through the activation of fibroblast growth factors (FGF 23). Indirectly, leptin blocks serotonin receptors and reduces the synthesis of serotonin that favors bone growth. In the postmenopausal period, the endocrine function of estrogen secretion is taken over by the adrenal glands, through the increased secretion of adrostenedione, and its aromatization leads to an increase in the level of estrogens in the blood. Bone protection is also provided by the conversion of dihydroepiandrosterone to estrone by activation of aromatase P450 in osteoblasts. Adipose tissue secretes proinflammatory cytokines that alter hormone secretion with effects on bone mineral density. IL-6 and TNF-α accelerate bone resorption by activating osteoclasts and upregulating RANKL/RANK/osteoprogerin. TNF-α induces bone resorption by activating nuclear factor kappaB (NF-κB), which regulates RANKL-induced effects, favoring osteoclast synthesis.
Conclusions. Mechanisms of adipokines, circulating steroid hormones (estrogens, androstendion, estrone), and proinflammatory cytokines IL-6 and TNF-α influence bone metabolism. At the same time, monitoring the serum level of adiponectin and leptin provides useful information in the early detection and treatment of osteoporosis.
Keywords. Postmenopausal osteoporosis, estrogens, obesity, bone metabolism, adipose tissue.