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Short Communication

A Brief Note on Osteoporosis in Males

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DESCRIPTION

Osteoporosis is currently recognized as a very important public health problem in old men as fragility fractures are complicated by increased morbidity, mortality, and social prices. Here we summary few recent findings in pathophysiology, diagnosis, and treatment of male osteoporosis [1]. Osteoporosis is a general skeletal disease characterized by reduced bone density and micro architectural deterioration of bone; this ends up in fracture risk. Bone fractures are a significant pathological state within the more and more older population. The old suffering from femoral fractures will die in less than a year (15%-25%) or become dependent (50%). A single vertebral fracture doubles the risk of limb fracture within a year and multiple vertebral fractures impair patients' quality of life and increase mortality. Major osteoporotic fractures at spine and hip are a social and economic burden; in developed countries, the risk for osteoporotic fractures at the wrist, hip, or spine is half-hour to 35% to 45%.

Although osteoporosis is perceived by the population as a women disease, one in eight men aged older than fifty years will undergo a fragility fracture throughout his lifetime; the most common sites for fragility fractures in men are forearm, vertebrae, and hip, however additionally fractures of different sites as ribs, pelvis, and collar bone are related to male osteoporosis. Almost 35% of hip fractures occur in men and mortality, within the first year a hip fracture, is higher in men compared to women. Men don't experience speedy bone loss as women do once after menopause; instead; they undergo a slow bone loss with age; this bone loss begins by the sixth decade at an average rate of 0.5% to 1.0% per annum and is followed by growing incidence of fractures [2].

Considering these data, osteoporosis in old men should be considered as a serious public health concern and as a life threatening disease; despite this thought, male osteoporosis remains an underdiagnosed and undertreated condition. Bone is a living tissue that undergoes continuous remodeling due to the combined action of bone cells: the Osteoblasts (OBs) that build up new bone matrix and also the Osteoclasts (OCs) that reabsorb bone. Among the bone matrix, Osteocytes (OSs), the mature form of OBs, regulate bone turnover by leading OBs and OCs activities. In osteoporosis, OBs and OCs activities are unbalanced with increased bone reabsorption and decreased bone deposition; this imbalance turns in bone loss and leads to fracture risk [3]. Several diseases alter the balance between bone formation and bone reabsorption and induce bone loss; in women, 25% to 40% of osteoporosis is secondary to extraskeletal diseases, and this proportion rises till 60% in men.

Other than secondary causes, aging may be a primary reason for bone loss in men and also in women; it induces bone loss through hormonal changes and age-related formative cell disfunction. Hormonal changes during aging are responsible for bone loss; specially, decreased levels of sexual hormones and relative increase in adrenal cortical steroid negatively influence bone remodeling. It is wide accepted that the decrease in sex steroid concentrations with age is related to decreased bone density and increase fracture risk in men; yet, the decline of testosterone hormone in men is gradual and not common to all the aged population. A recent paper on the wide cohort of men taking part within the MrOs study demonstrates that men with all-time low bioavailable estradiol had greater risk of fractures, whereas men with the lowest free testosterone hormone had no increased fracture risk after adjustment for estradiol [4]. Thus, the authors recommend that the bioavailability of estradiol, more than testosterone, is responsible for increased fracture risk in old men.

Excess of glucocorticoids both endogenous and exogenous is thought to be detrimental for bone; glucocorticoids have an effect on bone mainly by decreasing OB function. Glucocorticoid action relies upon the expression of 11 beta-hydroxysteroid dehydrogenase isozymes that interconvert active adrenal cortical steroid and inactive cortisol. Bone tissue is ready to convert cortisol in active cortisol due to this catalyst, whose expression will increase with aging. Thus, old persons are more sensible to endogenous and exogenous glucocorticoid; this leads to a relative hypercortisolism and probably in bone damage.

Although the incidence of fractures in men is increasing due to the ageing population, male osteoporosis remains an underdiagnosed and undertreated disease. Yet, there's growing awareness of the problem as a major public health concern [5]. Osteoporotic fractures increase morbidity and mortality of old

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men along with a substantial increase in public health cost. The pathophysiology of primary male osteoporosis includes hormonal changes and cellular ageing. 60% of fractures in men are due to secondary osteoporosis. Clinical instruction to address osteoporosis treatment in men comprehends the diagnosis of fragility fractures, the mensuration of BMD, and fracture risk estimation mainly through FRAX. The approved pharmacological treatments have shown to be effective in male osteoporosis; yet, a lot of analysis is required to handle the effectively in preventing nonvertebral fractures and to look at the comparative effectiveness of the various osteoporosis treatments in male.

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