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Commentary

Causes and Prevention of Osteoporosis in Postmenopausal Women

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DESCRIPTION

World Health Organization (WHO) characterizes regular menopause as at least 12 continuous months of amenorrhea not because of physiologic and pathologic causes. Measurements show that the mean age of regular menopause is 51 years in industrialized countries, contrasted with 48 years in poor and non-industrialized nations. With the typical life span reaching 70 years, most women will spend more than one-third of their life beyond the menopausal transition. Menopause is a natural physiological phenomenon developed from primary ovarian failure secondary to apoptosis. Ovarian capability declines with age. The beginning of menopause includes the decreasing production of estradiol, as well as increasing levels of Follicle-Stimulating Hormone (FSH). During the menopausal transition period, ladies will undergo various side effects, for example, hot flashes, night sweats, vaginal atrophy and dryness, dyspareunia, sleep disturbance and mood swings. Other than these, osteoporosis is the most prevalent disease in menopausal women and is connected with bad quality of life.

Osteoporosis, a multifactorial systemic skeletal disease, is distinguished by low Bone Mineral Density (BMD) and deterioration of bone tissue bringing about bone fragility. BMD estimated by dual X-beam absorptiometry is the highest quality level to diagnose osteoporosis. BMD decreases with age, thus primary osteoporosis basically happens in women 10-15 years after menopause and old men around 75-80 years of age. The accomplishment of peak bone mass is crucial to bone health and has an imperative impact on preventing osteoporosis and ensuing fractures in later years. It is accounted for that hip fracture could be diminished by 30% with an increase in peak bone mass of 10%. Bone mass growth begins from youth and goes on into adulthood, and peak bone mass can be accomplished during the twenties for the spine and hip while different bones, like the radius, reach a peak at age of 40 years. From that point onward, bone mass typically declines. By the age of 70 years, the bone mass has diminished by 30-40%.The determinant of the peak bone mass is hereditary variables. Studies found several genetic variants which are connected with bone mass, including low-density Lipoprotein Receptor-Related Protein 5 (LRP5), Osteoprotegerin (OPG), Sclerostin (SOST), estrogen receptor 1, and the Receptor Activator of NF-κB (RANK) pathway genes. Different factors including nutrition, smoking and exercise may likewise play a role during the peak bone mass accumulation.

Osteoclasts (bone resorbing cells) and osteoblasts (bone forming cells) are two kinds of cells that basically form the bone multi-cell unit, planning to regulate the balance between bone resorption and bone formation. The normal bone remodeling process comprises of five stages: the resting stage, the activation stage, the resorption stage, the reversal stage, and the formation stage. There are two stages of bone loss in women: The first happens predominantly in trabecular bone and beginning at menopause. It results from estrogen deficiency, and prompts a disproportionate increase in bone resorption as compared with development. This stage could be characterized as menopause related bone loss.

Being a systemic skeletal disease, osteoporosis turns into a significant public health and financial issue that is related with increased mortality and morbidity. Postmenopausal women are susceptible to primary osteoporosis since osteoporosis is firmly connected with estrogen inadequacy. During the menopausal transition period, the drop of estrogen prompts more bone resorption than arrangement, bringing about osteoporosis. The regularity of osteoporosis and related fracture are higher in postmenopausal women than in men. Since low estrogen levels are the main cause of postmenopausal osteoporosis, menopause hormone treatment is considered as the main line decision for prevention of osteoporosis.

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