

A Brief Note on Diagnosing Postmenopausal Osteoporosis

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DESCRIPTION

Postmenopausal Osteoporosis (PMOP) may be a common type of Osteoporosis (OP), which may be a condition affecting 35% of women over 60 years in China. Estrogen is an important component within the bone metabolism and its deficiency after menopause might result in rapid bone loss, which is greatest at intervals in the first 2-3 years after the menopause transition. What is more, the number of bones being reabsorbed will increase sharply in response to menopause beginning, whereas the number of bones being formed will increase moderately [1]. At present, Dual-Energy X-ray Absorptiometry (DEXA) scans are a basic approach to measuring Bone Mineral Density (BMD) to determine bone strength and risk of fracture.

Osteoporosis has currently been outlined by the World Health Organization (WHO) as an abnormal bone mineral density (Tscore) that is 2.5 SD below the mean peak worth in young adults. It raises a risk issue for the fracture to the standing of a diagnostic criterion; but, it has ignored the vital role of different determinants of bone strength and also the higher risk of fracture related to a precise level of bone mineral density in older women. Besides, it doesn't indicate that technique should be used or wherever bone mineral density should be measured. Therefore, the exploration of recent biomarkers might facilitate clinicians to monitor and treat the patients with PMOP as early as possible.

Insulin-like Growth Factor 1 (IGF-1) is taken into account because the most abundant growth factor within the bone matrix, maintaining bone mass in adulthood. Likewise, in agerelated osteoporosis in humans, it's been reported that people with osteoporosis were 45% lower in bone marrow IGF-1 concentrations compared to individuals without osteoporosis. Specifically, IGF-1 and a number of other of its binding proteins are positively related to bone mass. Therefore, it's a significant predictive value as a standard for the risk of osteoporosis and incident fractures. Recently, another star molecule that has been found to be closely related to the state of bone turnover is leptin. Adipocytes are vital parts concerned with modulating energy expenditure and bone cell activities.

Studies have indicated that adipocytes and osteoblasts derived from similar mesenchymal somatic cell precursors, obesityinduced adipocyte differentiation and lipid accumulation *in vivo* lead to reduced osteoblast differentiation and bone formation. It's been demonstrated that the increased expression of leptin and decreased adiponectin expression are related to obesity [2-4]. Leptin is taken as a potent bone cell modulator. Classically, regulation of bone metabolism was believed to be achieved by activating β -2 adrenergic receptors on osteoblasts, with a consequent decreasing in osteoblasts differentiation and increasing in osteoclasts activity. Hence, increased secretion of leptin has been projected that is detrimental to bone formation while supportive for bone resorption.

There are many candidate markers that change over time, like body fluid IGF-122 and leptin. Thus, the aim of this study was to work out that one demonstrated the greatest utility within the early detection of women with low bone mass or osteoporosis. We tend to hope our data might shed light on the utility of many new potential biomarkers for the diagnosis and treatment response monitoring of PMOP. The relationship between obesity and osteoporosis has been widely studied, and the link between obesity and increased bone mass had been recently confirmed by epidemiological evidence. Leptin, a product of adipocytes, plays a big role in regulating appetite [5]. It directly promotes the differentiation and proliferation of osteoblasts. The serum levels of leptin are absolutely related to BMD which leptin levels are reduced in osteoporotic women. However, the most effective leptin on BMD continues to be underneath in both in vivo and in vitro studies, as these results are inconsistent among completely different populations.

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