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Editorial

Secondary Osteoporosis: Endocrine and Metabolic Causes of Bone Mass Deterioration

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Secondary osteoporosis results from medical conditions or treatments that interfere with the attainment of peak bone mass and/or may predispose to accelerated bone loss. Although secondary osteoporosis is less common, it is becoming more frequently diagnosed. Apart from the well-defined risk of secondary osteoporosis in patients requiring long-term corticosteroids therapy, an increasing list of dietary, lifestyle, endocrine, metabolic, and other causes of bone mass deterioration has been identified (Table 1). Recently it has been demonstrated that, in contrast to primary osteoporosis which is associated with age, gender, and family history, secondary osteoporosis shows a prevalence in men similar to that in women (men 21% versus women 17.5%) [1]. However, at presentation with a recent clinical vertebral or nonvertebral fracture, at least 27% of patients have previously unknown contributors to secondary osteoporosis, which are treatable or need follow-up [2]. Therefore, a careful medical examination always should be carried out in each patient with a recent fracture to exclude potentially reversible causes of bone loss. In comparison with primary osteoporosis, treatment of secondary osteoporosis is usually more complex and requires treating the underlying disease.

The main focus of this special issue is on current research that advances our understanding of the mechanisms underlying the endocrine and metabolic causes of bone mass deterioration. In the paper "*Bone mineral density accrual determines energy expenditure with refeeding in anorexia nervosa and supersedes return of menses*," the authors in the clinical study examined the disproportionate increase in resting energy expenditure that occurs with refeeding of women with anorexia nervosa to determine if it was related to bone mass increase. They found that prolonged nutritional rehabilitation may lead to recovery from osteopenia (a common finding in anorexia nervosa) and resumption of menses in the women who remain amenorrheic with low bone mineral density (BMD). The paper entitled "*Protective role of black tea extract (BTE) against non-alcoholic steatohepatitis (NASH)-induced skeletal dysfunction*" was aimed to examine the chemoprotective actions of aqueous BTE on decreased BMD induced by nonalcoholic steatohepatitis in the Wistar rats. Some previous studies have suggested that habitual tea consumption might have beneficial effect on BMD in adults. In this study, the authors confirmed this suggestion in rats and also found that BTE may influence levels of RANKL, osteoprotegerin, and bone turnover markers. The paper "*Bone health in patients with multiple sclerosis*" describes the up-to-date diagnostic criteria and includes detailed review of the common risk factors, pathophysiology, and treatment options of secondary osteoporosis in patients with multiple sclerosis. The paper "*A roadmap to the brittle bones of cystic fibrosis*" summarizes the current knowledge on the risk for osteoporosis in this autosomal recessive disorder. Unlike primary osteoporosis, bone disease in cystic fibrosis begins at a young age and is associated with significant morbidity due to fractures and decreased lung function. This paper reviews the pathophysiology, current clinical practice

Table 1: Causes of secondary osteoporosis.

Dietary
Anorexia nervosa
Excessive protein intake
Excess vitamin A
Inadequate vitamin D intake
Smoking
Excess alcohol intake
Parenteral nutrition
Lifestyle
Low physical activity
Prolonged immobilization
Endocrine
Adrenal insufficiency
Cushing's syndrome
Diabetes mellitus
Hyperthyroidism
Hypogonadism
Hypopituitarism
Pregnancy
Metabolic
Malabsorption syndrome
Chronic metabolic acidosis
Systematic diseases
End-stage renal disease
Primary biliary cirrhosis
Inflammatory bowel disease
Cystic fibrosis
Rheumatoid arthritis
Chronic obstructive pulmonary disease
Mastocytosis
Chronic inflammation
Surgery/transplantation
Bariatric surgery
Organ transplantation
Medications
Corticosteroids
Antiepileptics
Selective serotonin-reuptake inhibitors
Heparin

guidelines, and future therapies for treating bone disease associated with cystic fibrosis. Another paper in this issue, *"Are selective serotonin reuptake inhibitors a secondary cause of decreased bone density?"*, deals with severe complication of therapy with commonly prescribed selective serotonin-reuptake inhibitors (SSRIs). Although multiple consistent findings reveal a trend suggesting that SSRI use may negatively impact bone and result in lower BMD, a definitive causal relationship cannot be drawn. However, a growing body of evidence suggests an association between SSRI use and bone loss which seems sufficient to consider adding SSRIs to the list of medications that contribute to secondary osteoporosis. Another review paper in this issue, *"Secondary osteoporosis in patients with juvenile idiopathic arthritis,"* focuses on focal and systemic bone loss seen in juvenile idiopathic osteoporosis. Several clinical and epidemiological studies are reviewed in order to highlight putative factors that may contribute to bone loss in juvenile idiopathic osteoporosis, including low lean mass, growth retardation, impact of proinflammatory cytokines on bone remodeling, RANK/RANKL/osteoprotegerin imbalance, and abnormal Wnt signaling.

The papers presented in this special issue depict some novel and hitherto not intensively studied aspects of secondary osteoporosis, underscoring the complexity of mechanisms that predispose to bone mass deterioration induced by metabolic and endocrine risk factors. The editors thank the authors of all submissions and hope that the content of this special issue will be useful for clinical practice and future research.

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