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Editorial

Osteoporosis in Thyroid Pathology

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Dear colleagues, all you are aware of osteoporosis and thyroid pathology in your routine clinical setup, I am reaching you all out to shed light on role of hyperthyroidism, hypothyroidism, subclinical hypothyroidism or subclinical hyperthyroidism.

Dual energy Xray absorptiometry (DEXA) is employed to measure the bone mineral density, a standard deviation low bone mass of 2.5 from young adults (T- score) is defined as Osteoporosis [1]. An imbalance between the process of bone formation and resorption will lead to Osteoporosis, a high incidence in general population of upto 50% of postmenopausal women [2]. Primary causes of osteoporosis are age and menopause [3]. Secondary causes of osteoporosis are systematic lupus erthematosus, human immunodeficiency virus infection, central nervous system disorders, nutrition disorders, hematologic disorders, gastrointestinal disorders, endocrine disorders, chronic obstructive pulmonary disease, rheumatoid arthritis, renal failure, liver disease and diverse medications [3-5]. It can lead to loss of height, back pain, change in posture, or intense pain following a fracture, as it increases the fracture risk by two-fold for every standard deviation below the mean of a young adult [2,6].

Metabolism and Cell differentiation in human body is regulated by thyroid hormones. Alteration of bone metabolism leading to osteoporosis and fragility fractures is due to complication of thyroid pathology. The negative impact of hyperthyroidism on bone metabolism is well established but hypothyroidism, subclinical hypothyroidism or subclinical hyperthyroidism role is not available much in the literature [7].

The receptors of thyroid hormone are located in bones, testis, placenta, muscle, liver, heart, lungs, pituitary gland, nervous system and other tissues. TRα1, TRβ1 and other subtypes of thyroid hormone receptors are members of the nuclear receptor superfamily which regulate important physiological and developmental processes by mediating the action of thyroid hormone. In the presence or absence of thyroid hormone ligand- dependent transcription factors; TRs bind thyroid hormone to facilitate the expression of target genes. TRα1, TRβ1 located in nucleus shuttle rapidly between nucleus and cytoplasm. TRα1 and TRβ1 will interact with importins and exportins respectively for translocation across the nuclear envelope. TR subtypes are due to enigmatic cytoplasmic functions leading to diversity of cellular response to thyroid hormone [8].

The important complications of thyroid disorders are Osteoporosis and fragility fractures associated with increased mortality. Bone metabolism and fracture risk is influenced by thyroid disorders. Decreased BMD and increased risk of fractures are associated with hyperthyroidism and subclinical hyperthyroidism. Levothroxine overtreatment for hypothyroidism has a similar impact on bone health as hyperthyroidism. Radioiodine treatment for subclinical hyperthyroidism can improve bone health. Osteoporosis or fragility fractures are not associated with subclinical hypothyroidism

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