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Journal of Osteoporosis and Physical Activity

Perspective

Study of Osteoporosis in Hyperthyroid Patients

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DESCRIPTION

Thyroid diseases are among the commonest endocrine disorders. Few investigations had detailed the prevalence of hyperthyroidism. Thyroid hormones are vital for skeletal development and growth. The bone effect of hyperthyroidism is characterized by accelerated bone turnover caused by direct stimulation of bone cells from high thyroid hormone concentrations thusly; this might result in deficiency of bone mass. Hyperthyroidism increases bone yield, with increases in both osteoclast and osteoblast activities. Thus, the bone remodeling circle is shortened, even though all phases of the cycle are not impacted equally. Early diagnosis of osteoporosis and management of thyrotoxic state is thus imperative to lessen the risk of fracture and improve the quality of life of patients with this disorder.

Bone health in hyperthyroidism is a poorly reported part of thyrotoxicosis particularly in sub-Saharan Africa, likely because osteoporosis is considered a disease in developed countries and is considered to comprise problems compared contrasted with infectious diseases in developing countries. The bone changes in hyperthyroidism are described by an improved bone turnover in both trabecular and cortical bone prompting expanded porosity and assembly of bone minerals. Cortical bone is impacted to a greater extent than trabecular bone.

Even though osteoporosis is known to be normal among the aging population, particularly in females; however, hyperthyroidism develops in osteoporosis in any case of age or gender. Moreover, with the varied clinical presentations of hyperthyroidism, a relationship between proximal myopathy and osteoporosis was laid out. Hence, the occurrence of proximal myopathy must raise suspicion of metabolic bone disease. Moreover, increased bone osteoblastic and osteoclastic activities were analyzed which brought about an accelerated bone turnover for bone resorption prompting low bone density.

The mechanisms of bone loss can be attributed to the adverse impact of the excess spread of thyroid hormones and reduction of TSH on bone cells acting either separately or in cooperative through the hypothalamic-pituitary-thyroid axis. This study affirmed that free triiodothyronine has either an immediate action on osteoblastic cells *in vivo*, which in turn mediates osteoclastic bone resorption or through direct action on osteoclastic cells by means of the thyroid receptors present on the bone cells. Bassett study confirmed that the abundance of thyroid hormone rather than thyrotropin lack induces osteoporosis in hyperthyroidism.

The particular role of TSH as a mediator of bone loss has not yet been laid out together. While one study proposed a direct role for low TSH alone as an intermediary of bone loss. Abe study exhibited a direct role of TSH as a negative controller of bone turnover by repressing the development and survival of osteoclasts, as well as the inhibition of osteoblast separation and the expression of type 1 collagen expression through TSH receptors in osteoblasts and osteoclast cells.

An indirect impact by means of TSH activity was reported in a study by Morimura et al. where it is noticed that an increased expression of type 2 iodothyronine deiodinase in human osteoblasts which was stimulated by Thyrotropin (TSH). Although, a decrease in the levels of TSH in a hyperthyroid state prompts a decrease in expression of type 2 iodothyronine deiodinase chemical culminating in high levels of triiodothyronine and stimulating bone resorption.

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Received: 01-Sep-2022, Manuscript No. JOPA-22-19136; **Editor assigned**: 05-Sep-2022, PreQC No. JOPA-22-19136 (PQ); **Reviewed:** 19-Sep-2022, QC No. JOPA-22-19136; **Revised:** 26-Sep-2022, Manuscript No. JOPA-22-19136 (R); **Published:** 06-Oct-2022, DOI: 10.35841/2329-9509.22.10.319

Citation: Shavlokhova M (2022) Study of Osteoporosis in Hyperthyroid Patients. J Osteopor Phys Act. 10: 319.