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Review Article

A REVIEW: MECHANISTIC INSIGHTS INTO THE EFFECT OF THYROID DISORDERS ON ESTROGEN LEVEL AND BONE MINERAL DENSITY

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ABSTRACT

Thyroid hormone serves as an indispensable component for the optimum functioning of various biological systems. They curb body's metabolism, regulates the estrogen level, regulates bone turnover, essential for skeletal development and mineralization. Within the scope of knowledge, it is intimately familiar that thyroid disorders have widespread systemic manifestations, among which in hypothyroidism, even though elevated TSH (thyroid-stimulating hormone) may reduce estrogen level which in turn stimulates osteoclasts and thus cause osteoporosis, while hyperthyroidism accelerates bone turnover. Hypothyroidism does not directly interfere with the skeletal integrity, but treatment with levothyroxine for the suppression of TSH to bring the hypothyroid patient to euthyroid state for a long haul; lead to simultaneous reduction in bone mass and in (bone mineral density) BMD.

After the initial relevation of the correlation between thyroid disorders and osteoporosis in numerous studies have emphasized that both hypo and hyperthyroidism either directly or indirectly affects the bone mineral density or leads to the progression of osteoporosis.

Therefore the present study is aimed and so designed to review all the possible associations between them and the impact of thyroid disorders on estrogen level and bone mineral density.

The main findings of this review indicate that both excesses as well as deficiency of thyroid hormone can be potentially deleterious for bone tissue.

Keywords: Bone cells, Bone mass, Calcium, Chondrocytes, Estrogen, Interleukins, Levothyroxine

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INTRODUCTION

Osteoporosis is one of the most plebeian progressive skeletal diseases described by the quirky disorientation of bone tissue and bone mass. It can affect a person of any age, gender and groups, but in older people, there is a steep increase in the incidence of fracture [1]. The struts that make a mesh-like structure within bones become thin, causing the bone to become fragile, which lead to break them easily and increases the incidence of fracture [2].

Breaking of bones and fragile bone has the same meaning and sometimes we can use both the terms interchangeably. In osteoporosis, the susceptibility to fracture gets increased which is characterized by abnormal architectural arrangement of bone tissue [3]. In a person with osteoporosis a minor bump or fall is sufficed to break the bone [4]. The most common parts that get affected by osteoporosis are the spine, hip, wrists (forearm) and shoulder [5].

There are two main hypotheses which we can relate to the development of osteoporosis. The first one is–ineluctable breakdown of bones due to changes in bone matrix which leads to wasting of bones and other tissue. It is generally associated with age. The second one is the nutritional hypothesis which generally accomplishes two main nutrients (calcium and protein) [5]. A report was also released by the United State government in 2004 with a recommendation for physician that, it is mandatory to adopt a pyramidal approach for osteoporosis. The third level includes treatment either with anabolic medications or antiresorptive medications. The second level includes the management and secondary cause of osteoporosis and the baseline include nutrients such as calcium and Vitamin D as well as physical therapy such as fall prevention [6].

If osteoporosis remain untreated for a long haul it gives rise to unnecessary extreme pain, altered body posture, disability in moving, climbing stairs and increased mortality [7]. Spinal fracture sometimes called vertebral fracture, which may lead to quirky curvature of the spine and loss of height [8].

With the wide range of condition Receptor activator of nuclear factor κ β ligand (RANK-L) is responsible for mediating the osteoclast-mediated bone resorption [9]. RANK-L (317 amino acid peptide) is the bone-specific gene and have a major role in stimulating, differentiation and inhibition of osteoclasts apoptosis [10]. For physiological reclamation of osteoclast some kind of cells and cytokines are responsible. In the bony surface the monocyte osteoclast precursors, activate, proliferate and fuse to form multinucleated osteoclasts and expresses a "receptor on their surface, for the activation of nuclear factor k-beta (RANK). In the presence of trophic factor M-CSF (Macrophage colony-stimulating factor) RANK-Ligand binds to this receptor. Binding of RANK-L to RANK, transformed osteoclast precursor to mature osteoclast and the process of bone resorption will take place and a bone resorption pit is formed. Osteoprotegerin (another protein) prevents the binding of RANK-L to RANK thus prevents bone resorption [11].

German pathologist Pomner originated the term osteoporosis by using three bone disorders. Osteomalacia (softening of bones) is a condition where a large number of unqualified bone tissue. It is due to the deficiency of Vitamin D and calcium. Osteoporosis (weak and brittle bones) is a condition where a large number of bone tissue are calcified and osteitis fibrosa, is a complication of hyperparathyroidism, where bone is eroded by osteoclasts and replaced with fibrous tissue [12]. The first case of osteoporosis was reported by Von Recklinghausen in 1891 in a young lady, who died due to hyperthyroidism with a feature of "worm-eaten appearance of long-bones", while in 1920 another researcher Plummer reported the same case with similar descriptions. Their finding indicates that thyroid disorder is a risk factor for osteoporosis and routine monitoring of thyroid hormones is mandatory [13]. Osteoporosis characterized by excessive bone loss, too little bone formation, or a combination of both [14].