Primer On The Metabolic Bone Diseases And Disorders Of Mineral Metabolism Read Online

In early part of the century, emphasis of studies in thyrotoxicosis was on calcium—phosphorus metabolism. With the introduction of antithyroid drugs and radioiodine therapy in s, clinically apparent hyperthyroid bone disease became less common. In s, with the availability of serum 25 OH D assay, there was resurgence of interest in vitamin D metabolism in hyperthyroidism.

Recently introduced methods of bone density measurement like dual-energy X-ray absorptiometry DEXA, as well as biochemical markers of bone resorption and formation, have led to further interest in hyperthyroidism related bone disease. Histomorphometric studies demonstrate that thyroid hormones increase the activation of new remodeling cycles and stimulate osteoclastic and osteoblastic activity in trabecular and cortical bone.

In an in vitro organ culture of fetal rat bone, Mundy et al. Histologically, an increase in number and activity of osteoclasts was detected. These cells appeared similar to those seen in cultured bone treated with PTH. The mechanisms of thyroid hormone induced bone resorption include cAMP-mediated, increased sensitivity of beta adrenergic receptors to catecholamines, increased sensitivity of bone cells to PTH, osteoclast activator factor and interleukin-1 IL-1 mediated increased bone resorption.

To investigate the molecular mechanism of osteoporosis in thyroid disease, Basstt et al. The majority of patients with hyperthyroidism in the West have normal or increased serum total calcium levels and the mean plasma calcium concentration is higher than in the control subjects.

Till, only 31 cases of thyrotoxic hypercalcemia were reported in literature. Hypercalcemia usually resolves after attainment of euthyroid state. Reversibility of hypercalcemia has been observed with all therapeutic modalities, i. Magnitude of disturbances in serum calcium in hyperthyroidism correlates with serum T3 levels.

Symptomatic hypercalcemia responds to rehydration, use of corticosteroids, calcitonin and phosphate therapy. Renal calcium excretion is usually increased in hyperthyroidism and correlates positively with excess thyroid hormone levels and cortical osteoclastic activity. It is caused by enhanced mobilization of bone mineral in hyperthyroid state and remains elevated even on calcium deficient diet.

In kidney, the filtered calcium load is enhanced due to increase in serum ultrafiltrable calcium and glomerular filtration rate GFR as well as reduced tubular reabsorption because of suppressed PTH levels. There are variable reports on serum phosphorous levels in patients with hyperthyroidism.

Most of the studies indicate hyperphosphatemic state. However, a few studies show normal or low levels of serum phosphorous. These effects lead to increased filtered load of phosphorous in patients with hyperthyroidism. Antithyroid treatment normalizes serum phosphorous concentration. The raised levels of serum alkaline phosphatase levels could be either of hepatic or of bone origin. Following treatment, serum alkaline phosphatase levels remain elevated for several months suggesting increased bone turnover continues even after restoration of a normal metabolic rate.
Bouillon and DeMoor first reported a decrease in serum PTH concentration in patients with hyperthyroidism. There is inverse relationship between serum calcium and serum PTH levels, indicating that increased serum calcium levels inhibit PTH secretion from parathyroid gland.

Suppressed PTH levels also explain the raised serum phosphorous and increased maximal tubular absorption rate for phosphorous. However, no correlation was observed between serum 25 OH D and bone histomorphicity. Recently, Yamashita et al. Serum 24,25 OH2 D levels are increased in patients with hyperthyroidism and they correlate with serum thyroid hormone levels.

Hyperthyroidism is an important cause of secondary osteoporosis. Early studies have used conventional radiography to assess bone mineral content. Tasi et al. DEXA allows rapid, accurate and highly reproducible assessment of mineral content with a minimal exposure to radiation. Bayley et al. Reversibility of both bone mineral mass and body muscle mass was recorded after 1 year of treatment with radiiodine therapy. Krokher et al. In this study, lumbar bone mineral content increased by 3. Most of the subsequent studies have shown significant increase in BMD following treatment. There are few published studies from India on hyperthyroidism and bone density. They also demonstrated reversal of bone loss after 1 year treatment with antithyroid medications.

In a recently concluded study, we have found that in contrast to Western data, hypercalcemia is not a feature of Indian patients with hyperthyroidism. BMD was compared in vitamin D-deficient and -sufficient patients, and it was observed that vitamin D deficient patients have more severe bone loss.

The author has also reported reversibility of bone loss after 1 year of medical therapy at hip and spine but deterioration of bone loss at fore arm. Thus, Indian patients with thyrotoxicosis are different from the Western patients from bone mineral homeostasis point of view. These patients have hypocalcemia rather than hypercalcemia as seen in the West and this is due to associated vitamin D deficiency. Future scientific work is needed to study the effect of vitamin D in therapeutic doses in patients with hyperthyroidism with concomitant vitamin D deficiency.

In summary, patients with hyperthyroidism have significant impact on bone mineral homeostasis. Western data suggest that these patients have hypercalcemia, hyperphosphatemia, raised alkaline phosphatase and reduced BMD. However, the available data from India suggest that due to concomitant vitamin D deficiency, these patients have normal calcium levels and increased bone loss. The signs and symptoms of hypothyroidism in general are opposite of thyrotoxicosis but this may not be true as far as bone metabolism is concerned.

For example, the fracture risk in patients with hypothyroidism is increased as reported by Vestergaard et al. In patients with hyperthyroidism, fracture risk was only significantly increased around the time of diagnosis [incidence rate ratio IRR between 1. In hypothyroidism, fracture risk was significantly increased both before and after diagnosis with a peak around the time of diagnosis IRR between 2.

This study concluded that fracture risk is increased in hyperthyroidism and hypothyroidism. Thyroid surgery seems associated with a decreased fracture risk in hyperthyroid patients. It seems that there is increase in bone density in adult subjects with hypothyroidism, but the bone quality is poor which is responsible for the possible increase in fracture in these patients. In Trosimo study, Grimnes et al. Calcaneo Osteo Sono assessment indices OSI of right feet were measured by ultrasound bone densitometer.

This suggests that hypothyroidism affects bone structure as assessed by QUS. Thyroid hormones play an important role in bone mineral homeostasis and bone density.

Both hyperthyroidism and, to some extent, hypothyroidism are associated with reduced BMD leading to increased fracture risk. With changing worldwide geographic occurrence of hip fractures, it is important to keep in mind the impact of thyroid disorders as a secondary cause of osteoporosis. Indian data suggest that majority of Indian patients with hyperthyroidism have concomitant vitamin D deficiency which aggravates bone loss. Further research is needed to study the impact of vitamin D supplementation in these subjects with hyperthyroidism on bone density and fracture risk reduction.

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This is an open-access article distributed under the terms of the Creative Commons Attribution-Noncommercial-Share Alike 3. This article has been cited by other articles in PMC. Abstract Thyroid diseases have widespread systemic manifestations including their effect on bone metabolism. Keywords: Bone metabolism, bone mineral density, hyperthyroidism, hypothyroidism, India, thyroid disorders.


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In patients with hyperthyroidism and they correlate with serum thyroid hormone levels. Hyperthyroidism is an important cause of osteoporosis. Serum 25 OH D levels are inversely correlated with bone histomorphometry. Recently, Yamashita et al. showed that serum 24,25 OH 2 D levels are suppressed in patients with hyperthyroidism. There is an inverse relationship between serum calcium and serum PTH levels, indicating that increased serum calcium levels inhibit PTH secretion from parathyroid gland.

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Thus, Indian patients with thyrotoxicosis are different from the Western patients from bone mineral homeostasis point of view. These patients have hypocalcemia rather than hypercalcemia as seen in the West and this is due to associated vitamin D deficiency. Future scientific work is needed to study the effect of vitamin D in therapeutic doses in patients with hyperthyroidism with concomitant vitamin D deficiency. In summary, patients with hyperthyroidism have significant impact on bone mineral homeostasis.

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