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The effect of thyroid hormone dysfunction on bone's metabolism

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Abstract

Decreased bone mineral density due to osteoporosis is the main cause of fragility fractures and leads to reduced quality of life. This can cause increased morbidity and mortality. Disturbance of balance between bone formation and bone resorption is dangerous. It can cause loss of bone mass and disruption of its architecture. Correct development, achievement of peak bone mass and normal functioning of human skeleton depend on different factors. Thyroid hormones play an important role in bone metabolism. Both excess as well as deficiency of fT_4 and fT_3 can be

Keywords: Thyroid hormones, thyroid diseases, osteoporosis, fractures

Introduction

Aim

The aim of this study is to review the current literature concerning the role of thyroid hormones on bone metabolism ^[1].

In this systemic mini review, we studied the association between reduced bone mineral density because of thyroid hormone dysfunction and. The research for literature was carried out using online databases like Pub Med, Research Gate, MEDLINE and Google Scholar. The studies written in English and published in peer reviewed journals were considered. The original research paper data were included.

The keywords used were, thyroid hormones, hypothyroidism, hyperthyroidism, osteoporosis, bone mineral density, femoral neck. The titles and abstracts for each study were thoroughly read. The references from each article were further searched upon to look for various other studies.

The studies were cross-sectional in nature. The studies had prospective design and included reduced bone mineral density as the outcome. Few studies also analyzed other risk factors associated with spontaneous fractures. Also, one study which evaluated the bone mineral density in pre-menopausal women was included. No cadaver studies were included and no scoring system was done to evaluate any of these studies.

Results and discussion

In the literature search many studies about thyroid dysfunction were found but only few had data regarding association between thyroid dysfunction and bone metabolism. 6 studies were reviewed for this study.

In a Cross-sectional cohort study done by L E van Rijn *et al.* ^[2], among 2584 randomly selected peri-menopausal Dutch women who participated in osteoporosis screening program in Eindhoven, The Netherlands, reported that higher fT_4 concentrations are independently related to low BMD at lumbar spine. The study had its own limitations. Firstly, a cross-sectional design was used and it was not possible to determine whether there was relationship between fT_4 or TSH and loss of BMD or incident fracture. Secondly, only fT_4 and not fT_3 levels were assessed and bone markers were not evaluated.

A cross sectional study was done by Margaret C Garin *et al.* ^[3] on thyroid status and BMD in a subset of 1317 participants in USA. No association was found between subclinical

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hypothyroidism and incident hip fracture compared with euthyroidism, when assessed at a single time point or persisting at two time points, in either women [hazard ratio (HR) 0.91, 95% confidence interval (CI) 0.69-1.20 for a single and HR 0.79, 95% CI 0.52-1.21 for two time points] or men (HR 1.27, 95% CI 0.82-1.95 for a single and HR 1.09, 95% CI 0.57-2.10 for two time points). Likewise, no association was found between subclinical hyperthyroidism and incident hip fracture in either sex (HR 1.11, 95% CI 0.55-2.25 in women and HR 1.78, 95% CI 0.56-5.66 in men). No association was found between subclinical thyroid dysfunction and BMD at the lumbar spine, total hip, or femoral neck sites.

BoDing *et al.* [4] performed retrospective cross-sectional population cohort study on women aged ≥ 65 years. All 1097 subjects had no overt thyroid dysfunction, 47 had subclinical hyperthyroidism and 100 had subclinical hypothyroidism. Results showed the femoral neck (FN) BMD was lower in women with lower TSH, with a high prevalence of osteoporosis and osteopenia ($p = 0.036$). The differences were not significant among subclinical hyperthyroid, subclinical hypothyroid and euthyroid women. Low TSH was related to low BMDs at FN by multiple logistic regression analysis corrected for age and BMI.

Tuchendler, D *et al.* [5] underwent a study having 38 women with hyperthyroidism, 40 with hypothyroidism and 41 healthy women and performed selected hormonal, immunological and biochemical tests, measurement of concentrations of bone turnover markers and densitometry. On initial evaluation, lower cortical bone density was found in patients with hyperthyroidism (femoral neck). After 12 months, an increase in BMD was seen, but it was still lower than in the control group. Statistically significantly higher concentrations of bone turnover markers, decreasing from the sixth month of treatment, were noted only in the group with hyperthyroidism. Statistically significant differences were not noted in the femoral neck nor in the lumbar spine BMD in patients with hypothyroidism. Hyperthyroidism poses a negative effect on bone metabolism. Hypothyroidism in premenopausal females does not have any influence on bone density.

GuriGrimnes *et al.* [6] underwent a study to explore the relationship between serum TSH and bone mineral density (BMD) in a healthy population. This study included 993 postmenopausal females and 968 males with valid measurements of BMD at the hip and forearm in the fifth Tromsø study conducted in 2001. Participants with major diseases or medication affecting BMD or thyroid function were excluded. After multivariate adjustment, the 28 men and 18 women with serum TSH below the 2.5 percentile had significantly lower BMD at the ultradistal (women) and distal (both sexes) forearm than the 921 men and 950 women with serum TSH in the normal range. Also, the 25 postmenopausal women with serum TSH above the 97.5 percentile had significantly higher BMD at the femoral neck than women with serum TSH in the normal range. Within the normal range of serum TSH, serum TSH was not associated with BMD. The small groups of men and women with serum TSH consistent with hyperthyroidism had lower BMD at the forearm than those with serum TSH in the normal range.

Anders Svare *et al.* [7] performed a cross-sectional, population-based study, in which 5778 women without and 944 with self-reported thyroid disease aged ≥ 40 years involved. The lower BMD was most prominent for women with TSH < 0.10 mU/L. For women with TSH levels ≥ 0.5 mU/L, no differences in BMD were seen. Compared to self-reported euthyroid

women, self-reported hyperthyroid women had increased odds for osteoporosis both distally (OR 1.35, 95% CI 1.00–1.82) and ultra-distally (OR 1.48, 95% CI 1.10–1.99). The prevalence of osteoporosis was higher in women who reported hyperthyroidism than in women without self-reported thyroid disease.

Conclusion

Overt hyperthyroidism leads to decreased BMD and increased fracture risk. Subclinical hyperthyroidism also causes a small reduction in BMD and increased risk of fracture but only in men and postmenopausal women. Although it is still unclear if bone changes observed in state of thyrotoxicosis are related to lack of TSH or to excess of thyroid hormones or both of them. Hypothyroidism has controversial influence on bone metabolism but probably leads to increased fracture risk. The authors recommend that long term studies with well-defined risk assessment and outcome measures are needed to formulate appropriate guidelines for preventive measures.

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