The main autoimmune and nonautoimmune etiologies of endogenous hyperthyroidism do not seem to influence the increased prevalence of morphometric vertebral fractures and osteoporosis in Portuguese men

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1. Introduction

Thyroid dysfunction is one of the most common causes for consultation in Endocrinology Departments. A meta-analysis to evaluate its incidence and prevalence in Europe found 6.7% of undiagnosed thyroid dysfunction, being 1.72% for undiagnosed hypothyroidism and 0.75% for previously diagnosed hyperthyroidism [1]. The prevalence of hyperthyroidism is about 10 times more common in women than in men in nondeficient iodine populations and is about 0.5%–2%. The more important etiologies are Graves’ disease and toxic multinodular goiter, while autonomously functioning adenoma and thyroiditis are not so common [2].

In animal models, namely in thyroid receptor (TR)β mutant mice, the studies show an accelerated skeletal development and
adult osteoporosis due to supraphysiological stimulation of skeletal 
TR2a due to disruption of the hypothalamus–pituitary–thyroid axis. The 
other studies also show that T3 exerts catabolic actions in adult bone 
and that those effects of disrupted or increased T3 action seem to 
 predominate over the skeletal responses to thyroid-stimulating 
hormone (TSH) [3].

In hyperthyroidism, the excess of circulating thyroid hormones 
can lead to an increase of bone resorption, either by acting directly 
on osteoclasts or indirectly on osteoblasts [4]. Also TSH seems to be 
a negative regulator of bone remodeling, inhibiting the formation, 
the survival of osteoclasts and the differentiation of osteoblasts 
[5,6]; however, this effect has not been totally clarified because 
experiments in mice with a loss-of-function TSH receptor, the bone 
loss seems to be independent of TSH levels [7].

The hyperthyroidism is a known important cause of secondary 
osteoporosis and of increase in fracture risk. One study in a male 
population investigated for the etiology of osteoporosis, found that 
a previous history of hyperthyroidism was present in about 5% [8].

However, most studies are done in postmenopausal women and 
in patients with exogenous hyperthyroidism due to thyroxine use 
after thyroidectomy for thyroid carcinoma.

The clinical studies in men with endogenous hyperthyroidism are 
very scarce, namely those addressing about osteoporotic fractures 
risk and the possible impact of the several etiologies of endogenous 
hyperthyroidism (autoimmune, toxic goiter) on that risk. This 
subject is important especially for the older populations because 
they are already prone to osteoporosis and to fragility fractures, 
which can be associated with precocious mortality [9]. Recent 
studies in male populations, show that accelerated loss of bone 
mineral density (BMD) at the hip is a risk factor for mortality that is 
not explained by comorbidity burden, change in weight or physical 
activity [10].

Vertebral fractures are among the most common fractures in 
osteoporosis. About 69% of patients with vertebral fractures are 
unaware of them, not only because they are very frequently 
asymptomatic, but also because patients are not routinely or 
accurately imaged. It is important to diagnose it, because their 
presence predicts the occurrence of future osteoporotic fractures all 
over the skeleton [11,12]. They occur more frequently in patients 
with a dual-energy X-ray absorptiometry (DXA) diagnosis of low 
bone mass rather than osteoporosis, showing that besides BMD, 
other factors contribute to the risk of osteoporotic fractures [12].

Vertebral fracture assessment (VFA) by DXA is a spine imaging 
with DXA scanners which may represent a better alternative to 
conventional radiography in the diagnosis of vertebral fractures, 
due to lower radiation dose and also to greater convenience for the 
patient as it can be done at the same time of DXA [13]. Despite 
not being used routinely in our country and in other bone metabolic 
units around the world, we find it very useful in clinical practice to 
detect prevalent fractures [14–16]. A previous study by our group in 
young hyperthyroid men, showed an increased prevalence of 
vertebral fractures detected by VFA [17].

In the last years, trabecular bone score (TBS) was defined as an 
indirect index of bone microarchitecture and of bone quality. It is 
determined from the grey-level texture metric variation analysis of 
the 2-dimensional lumbar spine DXA images, quantifying local 
variations in pixels intensities. The experimental variogram 
method is used to estimate the bone microarchitecture. An 
increased TBS assessment associates with better bone micro-
architecture, while a reduced TBS estimation correlates with fragile 
skeletal microarchitecture. It was shown that TBS is associated with 
the structure of the bone tissue and it may detect differences be-
tween DXA scans that show identical BMD amounts. It is an easy 
tool and clinical studies suggest that it improves (in addition to 
clinical risk factors and BMD) the prediction of fracture risk not only
in osteoporosis but also in some metabolic bone diseases. TBS 
seems also to be more sensitive than BMD in identifying secondary 
osteoporosis, namely hyperparathyroidism, adrenal adenomas and 
iatrogenic Cushing [18–20]. As far as we know, there are no TBS 
data in men with endogenous hyperthyroidism.

So, the aims of this cross-sectional case-control study were to 
evaluate the effects of endogenous clinical hyperthyroidism and 
their main etiologies in the body composition (BMD and total body 
fat and lean masses), in the TBS and in the prevalence of silent 
vertebral fractures using VFA technology (confirmed by X-ray) of 
a population of Portuguese men aged more than 50 years.

2. Methods

From an initial population of 119 men (78 with 
hyperthyroidism + 41 controls) which were admitted to the Endo-
ocrinology Department for diagnosis and treatment, we selected 41 
men aged over 50 years with clinical hyperthyroidism to participate 
in this study. For each patient, an age (limits 6–11 months) and 
stature (limits 1–3 cm) matched control person without diseases or 
medications affecting bone metabolism, was drawn from a random 
sample of patients of the Endocrinology Department. Exclusion 
criteria for both patients and controls were: subclinical hyperthy-
roidism, hypo/hyperparathyroidism, hypogonadism, diabetes 
mellitus, hyper/hypocortisolism, vitamin D deficiency, inflamma-
tory bowel disease, malabsorption diseases, liver/renal diseases, 
medications affecting the skeleton, and alcohol habits.

Regarding the etiology of the patients with hyperthyroidism, 20 
cases were Graves’ disease, 14 cases were toxic multinodular goiter 
and 7 cases were toxic nodule.

No patient had previously been treated empirically for osteo-
porosis or reduced bone mass or hyperthyroidism. We cannot be 
sure of the duration of the hyperthyroidism before the beginning 
of antithyroid medication but possibly it ranged at least from 3 to 12 
months.

Also, past history of fragility fractures and symptoms of verte-
bral fracture were excluded in both patients and controls. All pa-
tients and controls had a full clinical examination and body mass 
index (BMI) (kg/m²) was calculated.

In both groups, BMD (g/cm²) at the lumbar spine (L1–L4), at 
the hip (femoral neck and total), at the distal radius (1/3 or 33%) and 
at the whole body and total body tissue composition including soft 
body lean and fat masses (kg) were studied by DXA using the QDR 
Discovery W radiodensitometer (Hologic Inc., Marlborough, 
MA, USA) of the Lisbon Clinic of Endocrinology Diabetes and 
Metabolism, Lda. The fractured vertebrae were excluded from the 
analysis.

According to the International Society of Clinical Densitometry 
of official positions, in both groups the BMD was qualified by the 
lowest T-score obtained at the lumbar spine, at the hip and at the 
distal radius (33%) in osteoporosis, low BMD and normal BMD [21].

TBS was obtained from the pixel grey-level texture metric 
variation analysis of the 2-dimensional lumbar spine DXA images 
(iNsight software, Medimaps, Mérignac, France) [21]. The normal 
range for TBS in European men is considered: higher or equal to 
1.310 - high TBS, low fracture risk; between 1.230 and 1.310 - me-
tium TBS, medium fracture risk; less or equal to 1.230 - low TBS, 
high fracture risk [22,23].

The lateral images of thoracolumbar spine in DXA scan (VFA) 
were used to detect fractures and those were classified according to 
type (wedge, biconcave, crush) and severity (% of deformity) by 
Genant semiquantitative method, by one qualified endocrinologist. 
This method combines the qualitative visualization of the spine 
with the morphometric measurements of the vertebral body height 
in 6 points [14].
All patients had thoracolumbar spine X-ray (on frontal and lateral projections) on the same day or within a few days which was reviewed by one qualified radiologist. In a few instances where there was disagreement, a second radiologist was consulted. Conventional radiographs were electronic images produced by digital X-ray equipment and were viewed using a high-resolution viewing workstation designed for medical image reading. Their final reports were established as the gold standard for proven vertebral fractures, and only the positive cases in both VFA and X-ray were considered and included in the results.

Fasting blood samples were collected for measurement of serum chemistries, blood counts and hormones. Serum free T3, free T4, and thyroid-stimulating hormone (TSH) were assayed by an electrochemiluminescence immunoassay (Roche Diagnostics, Basel, Switzerland) and total calcium and phosphorus were assayed by enzymatic colorimetry (Roche Diagnostics).

All patients and controls gave their informed consent, according to the approved protocol by the ethic committee of the institution and based on Helsinki declaration.

The data were statistically analyzed using the Statgraphics Centurion XVI version 16.107.01 (Statpoint Technologies, Inc., The Plains, VA, USA). All the results are expressed as mean ± standard deviation. After testing for normal distribution, the Student t-test was used to compare the differences in parametric data between the groups. The Fisher exact test was used to compare the number of fractures in both groups. P-value of <0.05 was considered statistically significant.

### 3. Results

The mean age, height, weight, BMI, and total body lean and fat masses of the groups are shown in Table 1. In the hyperthyroidism group, there was a significant decrease in the mean total lean body mass (Table 1). Regarding BMD at the several skeletal sites, we found significant decreases in the mean BMD at the distal radius (Table 2 and Fig. 1).

The means of TBS in both groups were in the considered normal range and there were no significant differences between the groups (hyperthyroidism [range, 1.063–1.515; mean, 1.356 ± 0.10] vs. control [range, 1.079–1.493; mean, 1.328 ± 0.11]), as shown in Table 2. Moreover, in the hyperthyroidism group, TBS values were similar between the 10 men with fractures and those without it (1.354 ± 0.05 vs. 1.359 ± 0.05 respectively, P = 0.9). In the control group, TBS correlated with weight, total femur BMD and total fat and lean masses, while in the hyperthyroidism group this tool correlated just with whole body BMD and T-score.

The prevalence of decreased BMD and osteoporosis, as well as, the prevalence of vertebral fractures (detected by both VFA and X-ray) were significantly increased in the hyperthyroidism group (Table 3). Also in this group, fractures were detected in 10 cases by both VFA and X-ray.

In the control group, the only fracture detected was in T11 mild wedge, while in the hyperthyroidism group 7 fractures were

### Table 1

| Variable          | Control (n = 41) | Hyperthyroidism (n = 41) | P-value |
|-------------------|-----------------|--------------------------|---------|
| Age, yr           | 62.8 ± 7.8      | 62.9 ± 8.0               | NS      |
| Weight, kg        | 82.6 ± 13.1     | 76.3 ± 12.8              | 0.029   |
| Height, m         | 1.69 ± 0.05     | 1.69 ± 0.06              | NS      |
| Body mass index, kg/m² | 28.8 ± 4.1 | 26.6 ± 3.7               | 0.012   |
| Lean mass, kg     | 58.16 ± 7.7     | 52.3 ± 5.7               | 0.039   |
| Fat mass, kg      | 23.16 ± 6.7     | 19.6 ± 5.8               | NS      |

Values are presented as mean ± standard deviation. NS, not significant.

### Table 2

The BMD at several skeletal sites and TBS in the hyperthyroidism and control groups.

| Variable          | Control (n = 41) | Hyperthyroidism (n = 41) | P-value |
|-------------------|-----------------|--------------------------|---------|
| BMD, g/cm²        |                 |                          |         |
| L1–L4             | 1.035 ± 0.12    | 1.038 ± 0.22             | NS      |
| Femoral neck      | 0.854 ± 0.15    | 0.800 ± 0.13             | NS      |
| Total hip         | 1.024 ± 0.13    | 0.967 ± 0.13             | NS      |
| Distal radius (33%) | 0.769 ± 0.05 | 0.722 ± 0.08             | 0.005   |
| Whole body        | 1.189 ± 0.08    | 1.143 ± 0.12             | NS      |
| TBS L1–L4         | 1.328 ± 0.11    | 1.356 ± 0.10             | NS      |

Values are presented as mean ± standard deviation. BMD, bone mineral density; TBS, trabecular bone score; NS, not significant.

All patients had thoracolumbar spine X-ray (on frontal and lateral projections) on the same day or within a few days which was reviewed by one qualified radiologist. In a few instances where there was disagreement, a second radiologist was consulted. Conventional radiographs were electronic images produced by digital X-ray equipment and were viewed using a high-resolution viewing workstation designed for medical image reading. Their final reports were established as the gold standard for proven vertebral fractures, and only the positive cases in both VFA and X-ray were considered and included in the results.

Fasting blood samples were collected for measurement of serum chemistries, blood counts and hormones. Serum free T3, free T4, and thyroid-stimulating hormone (TSH) were assayed by an electrochemiluminescence immunoassay (Roche Diagnostics, Basel, Switzerland) and total calcium and phosphorus were assayed by enzymatic colorimetry (Roche Diagnostics).
Values are presented as number (%).

Control (n = 41)  Hyperthyroidism (n = 41)  P-value

BMD qualification
Normal  20 (48.8)  9 (21.9)  
Reduced  17 (41.5)  20 (48.8)  0.015
Vertebral fractures  4 (9.7)  12 (29.3)  0.007

Values are presented as number (%).

BMD, bone mineral density.

localized in the thoracic spine and 3 at the lumbar spine, 2 biconcave moderate degree and 8 wedge mild degree. BMD qualification of the 11 men with fractures was: control group - reduced BMD; hyperthyroidism group - reduced BMD in 3, osteoporosis in 1, and normal in 6.

In the hyperthyroidism group, there were significant increases in bone formation markers (osteocalcin and total alkaline phosphatase) and in β-crosslaps (CTX) after age-adjustment (Table 4).

When we compared patients with Graves’ disease with patients with toxic goiter, BMD at all skeletal sites, BMD qualification, prevalence of fractures and TBS were similar in both groups, but the total lean mass was significantly lower in patients with Graves’ disease (Table 5A). Also, there were significant increases regarding free T4 and total alkaline phosphatase in the Graves’ disease group (Table 5B).

4. Discussion

In hyperthyroidism, the duration of the bone remodeling cycle, which usually lasts about 7 months, can occur almost half that time (3–4 months) because the rate of bone turnover is accelerated, both bone resorption and formation are increased, so, the natural existing balance disappears leading to a bone resorption phase exceeding the bone formation phase; consequently there is an incomplete substitution with new bone cells and loss of mineralized bone. This progressively leads to a reduced BMD and osteoporosis development, reduced bone strength and consequently to a higher osteoporotic fracture risk. It usually affects both axial and appendicular skeleton, however it is usually more pronounced in areas with predominant cortical bone like the femoral neck and the distal radius [24,25].

This study was done in a population of men aged over 50, with non-iatrogenic clinical hyperthyroidism naïve of treatment, and we found that there was a slight tendency for a lower mean BMD in all skeletal regions except the lumbar spine, and a significant decrease of mean BMD at the distal radius. These results could be explained by the small number of patients, but we can also speculate that some small vertebral deformities (but not fractures, which were excluded), could contribute to small increases in areal BMD at the lumbar spine.

El Hadidy et al. [26] studied a hyperthyroid male population aged 23–65 years and found a significant decrease in the “lower half radius” BMD by DXA, which was related to both severity and duration of the hyperthyroidism; however, no other skeletal regions were studied and fractures prevalence was not assessed. In our present study men were aged more than 50 years and the scans were performed at the radius 1/3 or 33%. A previous study by our group in younger hyperthyroid men, showed significant BMD decreases in all skeletal regions and an increase in the prevalence of vertebral fractures detected by VFA [17].

Another study evaluating BMD at both lumbar spine and femoral neck, but in normal euthyroid men around 50 years-old, suggested that serum TSH concentration at the lower end of the reference range may be associated with a lower BMD [27].

The studies in men about the impact of the several etiologies of endogenous clinical hyperthyroidism on BMD and vertebral fractures prevalence are very scarce; the same study by El Hadidy et al. [26] found that z-scores of BMD at the lower half of the left radius in patients with Graves’ disease was not significantly different from those with toxic multinodular goiter. In females, all these etiologies of hyperthyroidism cause an increase in the prevalence of osteoporosis, but they do not seem to influence the impact on BMD [28]. Also in our study, there were no differences in the BMD between patients with Graves’ disease and patients with toxic goiter, but there was a significant reduction in the total lean mass in patients with Graves’ disease, probably because of

Table 3
The BMD qualification and the number of vertebral fractures in the hyperthyroidism and control groups.

| Variable                  | Control (n = 41) | Hyperthyroidism (n = 41) | P-value |
|---------------------------|-----------------|--------------------------|---------|
| BMD qualification         |                 |                          |         |
| Normal                    | 20 (48.8)       | 9 (21.9)                 |         |
| Reduced                   | 17 (41.5)       | 20 (48.8)                | 0.015   |
| Vertebral fractures       | 4 (9.7)         | 12 (29.3)                | 0.007   |

Values are presented as mean ± standard deviation.

Table 4
The biochemical, hormonal and bone markers data in the hyperthyroidism and in the control groups.

| Variable                  | Control (n = 41) | Hyperthyroidism (n = 41) | P-value |
|---------------------------|-----------------|--------------------------|---------|
| TSH, μIU/mL               | 1.63 ± 0.7      | 0.12 ± 0.1               | 0.000   |
| Free T4, ng/dL            | 1.16 ± 0.1      | 2.2 ± 1.6                | 0.000   |
| Free T3, pg/mL            | 3.43 ± 0.5      | 6.8 ± 5.7                | 0.029   |
| Calcium, mg/dL            | 9.37 ± 0.2      | 9.59 ± 0.4               | NS      |
| Phosphorus, mg/dL         | 3.12 ± 0.4      | 3.05 ± 0.4               | NS      |
| iPTH, pg/mL               | 52.26 ± 18.7    | 51.08 ± 32.4             | NS      |
| Total alkaline phosphatase, U/L | 62.87 ± 12.7 | 106.06 ± 48.5 | 0.000   |
| Bone alkaline phosphatase, μg/L | 15.8 ± 4.2 | 22.3 ± 13.5 | NS      |
| Osteocalcin, ng/mL        | 9.48 ± 5.8      | 15.71 ± 12.8             | 0.045   |
| CTX, ng/mL                | 0.2 ± 0.1       | 0.7 ± 0.3                | 0.045   |

Values are presented as mean ± standard deviation.

TSH, thyroid-stimulating hormone; iPTH, intact parathyroid hormone; CTX, β-slaps; NS, not significant.

4 After age-adjusted; but the difference between the means is nonsignificant without correction.

Table 5A
The BMD qualification, the number of vertebral fractures and the lean mass in patients with Graves’ disease and toxic goiter.

| Variable                  | Graves (n = 20) | Toxic goiter (n = 21) | P-value |
|---------------------------|----------------|----------------------|---------|
| BMD qualification         |                 |                      |         |
| Normal                    | 5 (25.0)        | 4 (19.1)             |         |
| Reduced                   | 9 (45.0)        | 11 (52.4)            | NS      |
| Osteoporosis              | 6 (30.0)        | 6 (28.6)             | NS      |
| Vertebral fractures       | 3 (15.0)        | 7 (33.3)             | NS      |
| Lean mass, kg             | 52.16 ± 5.4     | 56.92 ± 9.1          | 0.000   |
| Trabecular bone score     | 1.353 ± 0.11    | 1.360 ± 0.09         | NS      |

Values are presented as number (%). BMD, bone mineral density.

Table 5B
The biochemical, hormonal and bone markers data in the Graves’ disease and in the toxic goiter groups.

| Variable                  | Graves (n = 20) | Toxic goiter (n = 21) | P-value |
|---------------------------|----------------|----------------------|---------|
| TSH, μIU/mL               | 0.05 ± 0.1     | 0.2 ± 0.1            | NS      |
| Free T4, ng/dL            | 2.8 ± 1.9      | 1.6 ± 1.0            | 0.019   |
| Free T3, pg/mL            | 8.2 ± 6.9      | 5.4 ± 4.0            | NS      |
| Calcium, mg/dL            | 9.5 ± 0.4      | 9.7 ± 0.4            | NS      |
| Phosphorus, mg/dL         | 30.0 ± 9.5     | 30.1 ± 9.5           | NS      |
| iPTH, pg/mL               | 46.5 ± 30.2    | 55.4 ± 26.3          | NS      |
| Total alkaline phosphatase, U/L | 128.7 ± 54.8 | 84.8 ± 29.8 | 0.007   |
| Osteocalcin, ng/mL        | 18.6 ± 14.7    | 13.1 ± 10.6          | NS      |
| CTX, ng/mL                | 0.90 ± 0.4     | 0.51 ± 0.2           | NS      |

Values are presented as mean ± standard deviation.

TSH, thyroid-stimulating hormone; iPTH, intact parathyroid hormone; CTX, β-slaps; NS, not significant.
the more severe and/or longer duration of the hyperthyroidism which is very hard to define.

Weight loss and gastrointestinal changes like increased gut motility and consequent malabsorption of proteins, minerals and vitamins, are frequently seen in hyperthyroidism; this explains the significant decreases observed in total body lean mass and BMI in the hyperthyroidism group. Moreover, the weight reduction trend which is associated with a low bone mass, could also contribute to the BMD decrease in the hyperthyroidism group, because we found significant differences in both weight and total body fat mass between groups.

The Rotterdam study, done in a large sample of elderly Caucasian men and women, suggested that besides the effect of weight on bone density, there is also a direct effect of thyroid function on bone tissue [29].

In the hyperthyroidism group, we found 10 vertebral fractures confirmed by both VFA and X-ray, from mild to moderate degrees, and with a BMD qualification from normal to osteoporosis. So, the decreases in BMD may contribute, but cannot totally explain the increase in fracture risk observed in this population. Total lean mass could be another important factor, as well as parameters related to bone quality.

A population-based study of around 11,000 patients with diffuse and nodular toxic goiter with a mean age of 60 years old of both sexes, showed that fracture risk was only significantly increased at the time of diagnosis decreasing to normal after it [30].

Several studies done in patients with thyrotoxic-induced hyperthyroidism showed that fracture risk was higher in older men and, mainly, in women with a very suppressed TSH [31,32]. However, until now, it has not been clarified that iatrogenic hyperthyroidism does affect bone in a totally similar way as hyperthyroidism due to toxic goiter or autoimmune diseases.

Regarding TBS, the clinical studies in men are scarce and in men with hyperthyroidism are just a few. In our study, the means of TBS in both hyperthyroidism and control groups were in the considered normal range and there were no significant differences between the groups. A possible explanation is that in this endocrine disease the cortical bone is usually more affected than the trabecular one, which is the bone evaluated in TBS. By the other hand, small vertebral deformations (another complication of osteoporosis but not as severe as fragility bone fractures) may lead to artifacts in the analysis of the 2-dimensional lumbar spine DXA images, and/or TBS values obtained from those images. Another explanation could be that the time with nontreated hyperthyroidism was not long enough to develop bone microarchitecture changes measured by TBS.

In a retrospective case-control study, Leib et al. [20] showed that men with fragility fractures had lower TBS values than men without it, but it was not clear a causative direct association between the decrease in TBS and fracture risk.

Ock et al. found in Graves’ disease male and female patients a significant increase of TBS values from 1.377 to 1.390 after antithyroid therapy, however it was a noncontrolled study and those scores are considered within normal range [22,23,33].

In our study, we found significant increases in bone formation markers (osteocalcin and total alkaline phosphatase) and in CTX only after age-adjustment. Moreover, the patients with Graves’ disease had significantly higher levels of total alkaline phosphate than patients with toxic goiter. Studies in both men and women with hyperthyroidism, show that bone formation and bone resorption markers are increased and correlate with the disease severity [26,34,35].

The strength of this study resides in the fact of being done in a male population, not previously treated for hyperthyroidism, osteoporosis or low bone mass and showing that they already had some bone and lean masses decreases as well as vertebral fractures and increase in bone turnover markers.

A limitation for this study could be the relatively low number of patients. Future studies with bigger male populations and evaluating the effects of antithyroid treatment will be important to better understand the bone disease of endogenous hyperthyroidism.

5. Conclusions

In this controlled study of men aged over 50, with endogenous hyperthyroidism, we found a significant decrease in cortical BMD, namely at distal radius as well as a significantly increased prevalence of reduced BMD and osteoporosis and of asymptomatic vertebral fractures. The results of this study using VFA technology (confirmed by X-ray), suggest that BMD and lean mass changes in men aged over 50 with nontreated endogenous hyperthyroidism may contribute to the development of osteoporosis and fragility vertebral fractures, but there are very probably other implicated factors related to bone quality. The lean mass and the total alkaline phosphatase are more affected in Graves’ disease than in toxic goiter.

These data support the urgency of detecting silent fractures in this endocrine disease especially in elderly people, in order to start a treatment for both thyroid and bone. Moreover, we suggest that hyperthyroid men should perform DXA, routinely, not only at lumbar spine and femur, but also at the distal radius.

Conflicts of interest

No potential conflict of interest relevant to this article was reported.

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