Journal of Clinical and Nursing Research, 2025, Volume 9, Issue 6
http://ojs.bbwpublisher.com/index.php/JCNR
Online ISSN: 2208-3693
Print ISSN: 2208-3685

Analysis of the Clinical Characteristics of Bone Loss in Hospitalized Patients with Graves' Disease

Lifang Yang

Health Industry College, Guangdong Open University (Guangdong Polytechnic Institute), Guangzhou 510091, Guangdong, China

Abstract: *Objective*: To analyze the clinical characteristics of bone loss in hospitalized patients with Graves' disease. *Methods*: The clinical data of hospitalized patients with Graves' disease were collected. According to the results of bone density examinations, they were divided into a normal bone density group, a low bone mass group, and an osteoporosis group. The normal bone density group was used as the control group to analyze the clinical characteristics of bone loss. *Results*: The incidence of bone loss in patients with Graves' disease was 80.72%, with osteoporosis accounting for 39.16% and low bone mass accounting for 41.57%. The incidences of hyperthyroid heart disease, Graves' ophthalmopathy, and leukopenia in the osteoporosis group and the low bone mass group were significantly higher than those in the normal bone density group, reaching 84.62%, 60.87%, and 34.38%, respectively ($P < 0.05$). The age of the osteoporosis group with Graves' disease was 50.88 ± 12.03 years old, which was higher than that of the normal bone density group (40.03 ± 12.58 years old). The disease course was 55.66 ± 14.21 days, longer than that of the normal bone density group (43.38 ± 8.55 days). FT4 was 61.69 ± 8.42 pmol/L, higher than that of the normal bone density group (51.01 ± 6.77 pmol/L), while TSH was 0.08 ± 0.51 μIU/ml, lower than that of the normal bone density group (0.22 ± 0.55 μIU/ml). The blood phosphorus was 1.25 ± 0.29 mmol/L, lower than that of the normal bone density group (1.34 ± 0.27 mmol/L), with statistical significance ($P < 0.05$). In the low bone mass group, FT3 (13.08 ± 9.05 pmol/L) and FT4 (46.14 ± 3.46 pmol/L) were lower than those in the normal bone density group, with statistical significance ($P < 0.05$). Logistic regression analysis revealed that age, disease course, and TSH were contributing factors to bone loss. *Conclusion*: Patients with Graves' disease are prone to bone loss, and age, disease course, and TSH are contributing factors to bone loss.

Keywords: Graves' disease; Hospitalized patients; Bone density

Online publication: July 4, 2025

1. Introduction

Thyroid hormones are essential for normal bone development and play a crucial role in the differentiation and maturation of bone tissue cells. Excessive thyroid hormones can affect normal bone metabolism and lead to bone

loss. This article analyzed 166 patients with Graves' disease admitted to the Endocrinology Ward from April 2018 to April 2025 who had undergone bone density examinations. Taking normal bone density as the control, the clinical characteristics of bone loss in Graves' disease were obtained by analyzing the differences in clinical conditions among osteoporosis, low bone mass, and normal bone density. The results are reported as follows:

2. Subjects and methods

2.1. Subjects

The clinical data are sourced from 166 patients with Graves' disease admitted to the Endocrinology Ward from April 2018 to April 2025 who had undergone bone density examinations (70 male patients and 96 female patients). Bone density examinations are all performed using the Hologic QDR4500A fan-beam bone densitometer produced by Hologic, Inc., USA.

2.2. Research methods

A retrospective analysis of hospitalized cases is conducted. The patients' gender, age, height, weight, disease course, admission and discharge diagnoses, treatment processes, thyroid function and antibody tests, various biochemical tests, electrocardiograms, bone density examination results, etc. are recorded in detail. The case data of patients diagnosed with Graves' disease and who had undergone bone density examinations are retrieved. According to the results of bone density examinations, they are divided into a normal bone density group (32 patients, 19.28%), a low bone mass group (69 patients, 41.57%), and an osteoporosis group (65 patients, 39.15%). Statistical analysis is performed using the SPSS17.0 software package. Statistical descriptions of the samples are carried out. The differences in the means of the measurement data of the three groups are compared by analysis of variance (expressed as $\overline{x}$ ±SD). The chi-square test is used for count data and rank data. A P-value < 0.05 was considered statistically significant.

3. Results

3.1. Different bone densities and other complications of Graves' disease

The incidences of complications such as hyperthyroid heart disease, leukopenia, and Graves' ophthalmopathy in the osteoporosis group and the low bone mass group were significantly higher than those in the normal bone density group, with statistical significance ($P < 0.05$). The numbers of occurrences and percentages were 55 cases (84.62%), 42 cases (60.87%), and 11 cases (34.38%), respectively.

3.2. Clinical conditions of different bone densities (Table 1)

As shown in **Table 1**, the age, disease course, and FT4 of the osteoporosis group with Graves' disease were higher than those of the normal bone density group, while TSH and blood phosphorus were lower. The percentage of normal-sized thyroids was higher than that of the normal bone density group, and the percentage of grade II thyroids was lower than that of the normal bone density group, with statistical significance ($P < 0.05$). In the low bone mass group, FT3 and FT4 were lower than those in the normal bone density group, with statistical significance ($P < 0.05$). It can be seen that the degree of bone loss is not proportional to the thyroid hormone level and the degree of goiter. There is also a trend that the lower the TSH level, the more severe the bone loss.

Table 1. Clinical conditions of different bone densities in patients with Graves' disease ($\overline{x}$ ±SD)

Items	Normal bone density group	Low bone mass group	Osteoporosis group
Age (years)	40.03 ± 12.58	44.68 ± 11.14	50.88 ± 12.03△
Disease course (days)	43.38 ± 8.55	45.02 ± 9.50	55.66 ± 14.21△
T3 (mmol/L)	5.05 ± 3.27	4.41 ± 2.91	7.49 ± 4.15
T4 (mmol/L)	231.04 ± 87.16	192.20 ± 80.44	237.38 ± 145.47
FT3 (pmol/L)	15.24 ± 10.79	13.08 ± 9.05△	17.47 ± 11.80
FT4 (pmol/L)	51.01 ± 6.77	46.14 ± 3.46△	61.69 ± 8.42△
TSH (μIU/ml)	0.22 ± 0.55	0.15 ± 0.57	0.08 ± 0.51△
Blood Calcium (mmol/L)	2.21 ± 0.17	2.24 ± 0.21	2.17 ± 0.17
Blood Phosphorus (mmol/L)	1.34 ± 0.27	1.32 ± 0.27	1.25 ± 0.29△
Normal-sized Thyroid (%)	15.28	32.34	49.38△
Grade I Thyroid (%)	19.05	21.20	16.28
Grade II Thyroid (%)	61.90	42.46	29.56△

*Note: △indicates a significant difference compared with the normal bone density group ($P < 0.05$); the disease course is expressed as $\overline{x}$ ±SE.

3.3. Logistic regression analysis

A logistic regression analysis was performed with bone density as the dependent variable and clinical conditions as independent variables. The results showed that age, disease course, and TSH were contributing factors to bone loss, as shown in **Table 2**.

Table 2. Logistic regression analysis of bone density and clinical conditions

Parameters	P-value	OR	95%CI
Age	0.01	1.08	0.73–2.38
Disease Course	0.04	2.39	1.87–2.71
TSH	0.02	1.57	1.19–2.07

4. Discussion

In 1891, German scholars such as Gorka first confirmed that hyperthyroidism could promote the development of osteoporosis [1]. Thyroid hormones can stimulate osteoclast differentiation and inhibit osteoblast differentiation. Huang *et al.* believed that high thyroid hormone levels were important factors affecting the bone turnover rate and osteoporosis in hyperthyroid patients [2]. Many scholars believed that hyperthyroidism was one of the common causes of osteoporosis [3, 4]. The reported incidence of osteoporosis complicated with hyperthyroidism at home and abroad is approximately 20–50% [5]. In this study, the incidence of bone loss was 80.72%, and the incidence of osteoporosis was 39.16%, which was consistent with the clinical reports. The age and disease course of the osteoporosis group in this study were greater than those of the normal bone density group, which was consistent with the clinical reports that the incidence and severity of bone loss in hyperthyroid patients increase with age and

that bone density decreases with the prolongation of the duration of high-concentration thyroid hormones [6, 7].

Wang believed that both high thyroid hormones and low TSH could affect bone remodeling [8]. Many scholars believed that the thyroid hormone that controls bone loss and bone density in adults was mainly T3, which mainly acts through the thyroid hormone receptor-α [9–11]. Simsek *et al*. clarified that it mainly acts through the thyroid hormone receptor-α1. Khamisi studied the role of vitamin D in the bone metabolism of patients with Graves' disease and emphasized the risk of osteoporosis that may be caused by vitamin D deficiency [12]. Yang *et al.* showed that T4 could reduce bone density and increase the risk of fractures [13]. Qi studied the causal relationship between thyroid dysfunction and bone loss and pointed out that an imbalance in thyroid hormone levels may have a negative impact on bone health [14]. Branstetter explored the mechanism and treatment options for osteoporosis caused by hyperthyroidism and emphasized the importance of early intervention and comprehensive management in the prevention and treatment of such osteoporosis [15]. Wen *et al.* expounded on the impact of hyperthyroidism on bone metabolism and its related mechanisms and emphasized the negative effects of thyroid hormones on bone health under pathological conditions [16]. Zhou *et al.* analyzed the association between hyperthyroidism and osteoporosis through epidemiological studies and pointed out that the incidence of osteoporosis in hyperthyroid patients was significantly increased [17]. Delitala explored the clinical characteristics of hyperthyroid patients and the risk factors for osteoporosis and emphasized the impact of hormone levels, age, and other health conditions on the risk of osteoporosis [18]. Hong reviewed the methods for evaluating and managing bone health in patients with thyroid dysfunction [19]. Ma *et al.* explored the association between hyperthyroidism and osteoporosis and its research progress [20].

Chen *et al.* analyzed the related factors of decreased bone density in hyperthyroid patients [5]. Fan *et al.* described the clinical characteristics and treatment progress of bone loss in patients with Graves' disease [21]. Tsevis *et al.* explored the impact of hyperthyroidism on bone metabolism and its mechanism of action [22]. In this study, the FT3 and FT4 levels in the low bone mass group were lower than those in the normal bone density group, but those in the osteoporosis group were higher, indicating that there was no direct relationship between FT3, FT4, and bone loss. Chen found that in young and middle-aged hyperthyroid patients, bone density decreased with the increase in serum FT3 and FT4 concentrations and gradually decreased with the decrease in serum TSH concentration, and osteoporosis was most obvious in patients with the lowest TSH [5]. Deng *et al.* also believed that TSH had a protective effect on bones, and a low TSH level could lead to a decrease in bone density [23]. Yao *et al.* clarified that low TSH in hyperthyroidism could cause an increase in TNF-α, and the increase in TNF-α promoted bone loss caused by high thyroid hormones [24]. However, Wen *et al.* believed that bone loss in hyperthyroidism did not depend on the TSH level in the circulation [16]. In this study, the TSH in the osteoporosis group was 0.08 ± 0.51 μIU/ml, lower than that in the normal bone density group, supporting the view that a low TSH level promotes a decrease in bone density. Chae *et al.* reduced the risk of osteoporosis by adjusting the TSH concentration of patients after thyroid cancer surgery to 1 mU/L [25].

Owen *et al*. attributed osteoporosis to abnormal bone mineralization from the perspective of the biomineralization theory [26]. Fu found that the serum calcium and phosphorus levels in hyperthyroid patients were increased, and the serum calcium and phosphorus levels were positively correlated with the thyroid hormone levels [7]. However, in this study, there was no difference in blood calcium. The blood phosphorus in the osteoporosis group was 1.25 ± 0.29 mmol/L, lower than that in the normal bone density group. This may be related to the increase in the parathyroid hormone/calcitonin ratio in hyperthyroid patients, which promotes the excretion of phosphorus in the urine [28].

There are many factors that affect bone density, and hyperthyroidism is just one of them. It can be seen from this study that the impact of thyroid function on bone density is certain. Gorka *et al.* believed that osteoporosis caused by hyperthyroidism could be reversed after the thyroid function returned to normal for a certain period of time [1]. Supplementation with vitamin D and bisphosphonates could increase bone density [27]. Reddy *et al.* believed that treating hyperthyroidism could reverse lost bone mass, improve bone metabolism, and reduce the risk of fractures [28]. However, Tuchendler *et al.* found that the low bone density of the femoral neck in hyperthyroid patients increased significantly after 12 months of antithyroid treatment but was still lower than that of the normal control group [29]. In clinical work, it is necessary to monitor the bone density of hyperthyroid patients, especially those with advanced age, long-term illness, extremely high thyroid hormone levels, and extremely low TSH levels. Timely treatment of hyperthyroidism, supplementation with vitamin, and the use of bisphosphonates can prevent the occurrence of osteoporosis.

5. Conclusion

This study analyzed bone loss in hospitalized Graves' disease patients and found a high prevalence of bone density abnormalities. Over 80% of patients experienced bone loss, with nearly 40% having osteoporosis. Bone loss was linked to older age, longer disease duration, and lower TSH levels. Patients with bone loss also had higher rates of complications like hyperthyroid heart disease and Graves' ophthalmopathy. Notably, bone loss severity did not directly correlate with thyroid hormone levels (FT3/FT4). Logistic regression identified age, disease duration, and TSH as key risk factors. Graves' disease is strongly associated with bone loss, requiring clinical attention to monitor bone density, especially in older patients or those with long-term illness and low TSH. Early intervention for thyroid dysfunction may help prevent osteoporosis.

Disclosure statement

The author declares no conflict of interest.

References

[1] Gorka J, Taylor-Gjevre RM, Arnason T, 2013, Metabolic and Clinical Consequences of Hyperthyroidism on Bone Density. International Journal of Endocrinology, 22: 638–727. DOI: 10.1155/2013/638727

[2] Huang L, Li X, Mu L, et al., 2011, Prevalence of Osteoporosis and Characteristics of Bone Turnover Markers in Patients with Graves' Disease. Chinese Journal of Endocrinology and Metabolism, 27: 906–910. DOI: 10.3760/cma.j.issn.1000-6699.2011.11.009

[3] Hudec S, Camacho P, 2012, Secondary Causes of Osteoporosis. Endocrine Practice, 19(1): 1–31.

[4] Ercolano MA, Drnovsek ML, Silva Croome MC, et al., 2013, Negative Correlation Between Bone Mineral Density and TSH Receptor Antibodies in Long-Term Euthyroid Postmenopausal Women With Treated Graves' Disease. Thyroid Research, 6(1): 11.

[5] Chen L, Zhao S, Dai X, 2021, Correlation Analysis of Bone Density and Serum Bone Metabolism Indexes in Patients With Hyperthyroidism. Chinese Journal of Primary Medicine and Pharmacy, 28(4): 495–499.DOI: 10.3760/cma.j.issn.1008-6706.2021.04.004

[6] Cao X, Zhang Z, Wang N, 2024, Correlation Study on the Level of Thyrotropin and Bone Density in Elderly Patients.

China Science and Technology Journal Database Medicine, 2024(2): 67–70.

[7] Fu Y, Wu D, Zhao E, et al., 2024, Evidence-Based Basis and Possible Mechanisms of Systemic Bone Loss After Osteoporotic Fractures. Chinese Journal of Osteoporosis, 30(5): 749–754, 774.

[8] Wang S, Wang Y, Zhu L, et al., 2023, Effects of TSH Suppressive Therapy on Bone Mineral Density (BMD) and Bone Turnover Markers (BTMs) in Patients With Differentiated Thyroid Cancer in Northeast China: A Prospective Controlled Cohort Study. Endocrine, 79(1): 113–124.

[9] Qiao H, Ding H, Sun J, 2023, Correlation Among Bone Density, Bone Metabolism and Adipocytokines in Patients With Hyperthyroidism. Henan Medical Research, 32(22): 4091–4095.

[10] Liao Q, He S, Xiao Z, et al., 2021, Correlation Analysis of the Positive Rate of TRAb in Serum Thyroid Function and the Levels of TSH, FT_3, FT_4, T_3, T_4 in Pregnant Women With Hyperthyroidism During Pregnancy. Chinese and Foreign Medical Research, 19(11): 81–84.

[11] Simsek Y, Tofil H, Rosenthal M, et al., 2024, Nuclear Receptor Corepressor 1 Levels Differentially Impact the Intracellular Dynamics of Mutant Thyroid Hormone Receptors Associated With Resistance to Thyroid Hormone Syndrome. Molecular and Cellular Endocrinology, 594: 112373.

[12] Khamisi S, Lundqvist M, Rasmusson A, et al., 2023, Vitamin D and Bone Metabolism in Graves' Disease: A Prospective Study. Journal of Endocrinological Investigation, 46: 425–433.

[13] Yang X, Yang L, 2023, Relationship Between Subclinical Thyroid Dysfunction and Osteoporosis and Research Progress. Advances in Clinical Medicine, 13(6): 10124–10129.

[14] Qi W, Wang D, Hong Y, et al., 2024, Investigating the Causal Relationship Between Thyroid Dysfunction Diseases and Osteoporosis: A Two-Sample Mendelian Randomization Analysis. Scientific Reports, 14: 12784.

[15] Branstetter R, Islam R, Toups C, et al., 2023, Mechanisms and Treatment Options for Hyperthyroid-Induced Osteoporosis: A Narrative Review. Cureus, 15(11): e48798.

[16] Wen B, Xiang Q, 2022, Research Progress on the Pathogenesis and Clinical Characteristics of Hyperthyroid Osteoporosis. China Modern Medicine, 29(32): 28–32.

[17] Zhou Q, Li J, 2024, Study on the Relationship Between Thyroid Hormone Sensitivity and Osteoporosis in Euthyroid Population. Chinese Journal of Endocrinology and Metabolism, 40(4): 320–325.

[18] Delitala A, Scuteri A, Doria C, 2020, Thyroid Hormone Diseases and Osteoporosis. Journal of Clinical Medicine, 9(4): 1034.

[19] Hong AR, Kang HC, 2023, Evaluation and Management of Bone Health in Patients With Thyroid Diseases: A Position Statement of the Korean Thyroid Association. Endocrinology and Metabolism (Seoul), 38(2): 175–189.

[20] Ma T, Chen D, Wang W, 2022, Relationship Between Osteoporosis and Hyperthyroidism. Chinese Journal of Osteoporosis, 28(3): 380–385, 439.

[21] Fan Y, Shao M, Wang J, et al., 2022, Correlation Analysis of Thyrotropin Receptor Antibodies and Bone Turnover Markers in Newly Diagnosed Patients With Graves' Disease. Chinese Journal of Endocrinology and Metabolism, 38(5): 7.

[22] Tsevis K, Trakakis E, Pergialiotis V, et al., 2018, The Impact of Thyroid Diseases on Bone Density and Biochemical Markers of Bone Metabolism. Hormone Molecular Biology and Clinical Investigation, 35(1): 20180039.

[23] Deng T, Zhang W, Zhang Y, et al., 2021, Thyroid-Stimulating Hormone Decreases the Risk of Osteoporosis by Regulating Osteoblast Proliferation and Differentiation. BMC Endocrine Disorders, 21: 49.

[24] Yao N, Li P, Geng C, et al., 2021, Research Progress on the Pathogenesis of Postmenopausal Osteoporosis Mediated by TNF-α. Chinese Journal of Osteoporosis, 27(3): 454–458.

[25] Chung CW, Choi HS, Kong SH, et al., 2021, Postmenopausal Women's Bone Health Measurement After Thyrotropin Suppression Therapy for Differentiated Thyroid Cancer: Bone Mineral Density and Trabecular Bone Score. Journal of Clinical Medicine, 10(9): 1964.

[26] Owen X, Qi Z, 2006, Relationship Between the Levels of Calcium, Phosphorus, and Magnesium in the Serum of 53 Hyperthyroid Patients and Osteoporosis. Guangdong Medical Journal, 27(7): 1068–1069.

[27] Zhu J, Chen K, Wang H, et al., 2005, Abnormal Thyroid Function and Bone Loss in Elderly Women. Chinese Journal of Osteoporosis, 11(4): 464–466.

[28] Reddy PA, Harinarayan CV, Sachan A, et al., 2012, Bone Disease in Thyrotoxicosis. Indian Journal of Medical Research, 135: 277–286.

[29] Tuchendler D, Bolanowski M, 2013, Assessment of Bone Metabolism in Premenopausal Females With Hyperthyroidism and Hypothyroidism. Endokrynologia Polska, 64(1): 40–44.