ISSN: 2581 – 3633
PubMed - National Library of Medicine - ID: 101745081

International Journal of Medical Science and Advanced Clinical Research (IJMACR)

Available Online at:www.ijmacr.com

Volume – 6, Issue – 3, June - 2023, Page No. : 229 - 235

Cross-Sectional Study of Osteoporosis Among Women With Thyroid Dysfunction

[1]Dr. R. B. Uppin, Professor, KAHER, Department of Orthopaedics, J. N. Medical College, Belagavi 590010, Karnataka, India.

[2]Dr. D. Uday Kumar, Assistant Professor, Department of Orthopaedics, Arundhathi Institute of Medical Sciences and Hospital, Hyderabad 500043, Telangana, India.

[3]Dr. S. K. Saidapur, Associate Professor, KAHER, Department of Orthopaedics, J. N. Medical College, Belagavi 590010, Karnataka, India.

[4]Dr. Raunak Pareek, Post-Graduate, KAHER, Department of Orthopaedics, J. N. Medical College, Belagavi 590010, Karnataka, India.

Corresponding Author: Dr. Raunak Pareek, Post-Graduate, KAHER, Department of Orthopaedics, J. N. Medical College, Belagavi 590010, Karnataka, India.

How to citation this article: Dr. R. B. Uppin, Dr. D. Uday Kumar, Dr. S. K. Saidapur, Dr. Raunak Pareek, "Cross-Sectional Study of Osteoporosis Among Women With Thyroid Dysfunction", IJMACR- June - 2023, Volume – 6, Issue - 3, P. No. 229 – 235.

Type of Publication: Original Research Article

Conflicts of Interest: Nil

Abstract

Thyroid hormone affects the rate of bone replacement. Too much thyroid hormone (i.e., thyroxine) in your body speeds the rate at which bone is lost. If this happens too fast, the osteoblasts may not be able to replace the bone loss quickly enough. If the thyroxine level in your body stays too high for a long period or the thyroid-stimulating hormone (TSH) level in your body stays too low for a long period, then there is a higher risk of developing osteoporosis. The objective was to assess the risk factors associated with osteoporosis. Data was collected from type 2 diabetes mellitus patients attending outpatient at Orthopedics and Diabetology department who were willing to undergo DEXA Scan in 106 patients. Various risk factors were evaluated through a questionnaire. T Scores and Z Scores were evaluated for the presence of osteoporosis based on WHO Criteria. In this cross-sectional study 106 patients with thyroid dysfunction were studied using DEXA scan. The overall prevalence of osteoporosis as per our findings was 20.75% (Hyperthyroid osteoporotic were 41.18% and Hypothyroid osteoporotic were 11.11%), osteopenia 20.75% and thyroid patients with normal BMD were 58.49 %. High prevalence of osteoporosis in type 2 diabetes mellitus patients is a cause for concern. As the diagnosis and long-term treatment of osteoporosis and

consequent fractures are expensive for the individual as well as the health system, there is a need for careful consideration in determining the risk factors as well as the future course of action on scientific evidence.

Keywords: Prevalence, Osteoporosis, Type 2 Diabetes Mellitus, DEXA.

Introduction

Thyroid hormone plays a pivotal role in normal endochondral ossification and skeletal development, linear growth, maintenance of bone mass, and efficient fracture healing. Osteoporosis being the metabolic bone disease with probably the highest prevalence can be classified as primary or secondary.[1] Primary osteoporosis is more commonly found in postmenopausal women, while secondary osteoporosis can occur at any age and can be caused, for example, by endocrinopathies, including hormonal dysfunction of the thyroid gland. Thyroid hormones show pleiotropic effects in not just the osseous tissue but the body as a whole. An excess or deficiency of thyroxine (fT4) and triiodothyronine (fT3) can therefore represent a risk for the bones. Osteoporosis has traditionally been considered a disorder of postmenopausal women, but low bone mass and accelerated bone loss can also occur early in life causing premenopausal osteoporosis.[2] There are a few risk factors that increase a woman's risk of premenopausal osteoporosis, including drugs, hormonal and nutritional factors, and physical in-activity, which need to be identified and managed accordingly.

The literature has evidence that the normal cycle of bone turnover decreases to half from approximately 200 days in hyperthyroidism and increases in hypothyroidism to approximately 700 days.[3] There is some evidence to suggest that hyperthyroidism reduces BMD by one-tenth in each cycle of bone turnover, and its counterpart increases it by approximately 17% for each cycle of bone turnover. Lower BMD in hyperthyroidism leads to higher susceptibility to fractures. Although some studies have suggested that bone mass increases in hypothyroidism, the risk persists due to increased bone stiffness. Hyperthyroidism is widely accepted to reduce BMD and consequently increase this risk. Literature shows different opinions about the effects of hypothyroidism, hyperthyroidism and their treatments in bone pathology.

Hyperthyroidism and osteoporosis

Thyroid hormones are extremely important in achieving the expected bone mass. Therefore, if there is any aberration in the balance between resorption and formation; The bone formation as a process is affected (duration might decrease to one third of the baseline) and ultimately causes poor mineralization (approximate loss of 10% of bone mineralization per cycle). All of this eventually leads the aforementioned consequence of compromised BMD and subsequently, fractures. Another factor that is associated with decreasing the BMD in hyperthyroidism is the increase in the blood concentration of IL-6. IL-6 is known as an activator for the production of osteoclasts and facilitates the action of the parathormone in the bones. Increase in the secretion of thyroid hormones causes a negative calcium balance owing to hypercalcemia and hypercalciuria.[3] This has a direct involvement in causing osteoporosis in such patients who are already at risk.

Hypothyroidism and Osteoporosis

Hypothyroidism is one of the most rampant endocrinological disorder in the world. The prevalence of hypothyroidism in India is 10-11%. The state of inadequate formation of T3 and T4 is termed

hypothyroidism. Thyroid stimulating hormones (TSH) directly influence bone remodeling by the TSH receptor, which is found in OB and osteoclast precursor cells. TH are essential for bone growth and remodeling. This increase in BMD in females having hypothyroidism may be secondary to a reduced metabolic rate during hypothyroidism, leading to a reduction in the rate of the bone resorption process and a greater net gain in bone.

There is a decrease in osteocalcin during hypothyroidism that leads to osteosclerosis and an increase in BMD. In addition, a fall in serum calcium and vitamin D levels leads to poor bone quality, as both of them are essential for bone remodeling and for maintaining a normal BMD level in bones. TSH has a direct impact on TSH receptor mediated bone remodeling found in OB and osteoclast progenitor cells. Therefore, an increase in thyroid hormones leads to an increase in cortical thickness and reduced activity of OBs, which leads to slow and prolonged maturation of the bones.[5] Furthermore, slow and decreased bone remodeling due to hypothyroidism leads to decreased bone matrix protein, such as osteocalcin, while increased mineralization causes bone sclerosis, further making these patients prone to bone fractures with hypothyroidism. However, reduced serum calcium and vitamin D level lead to impaired bone quality. Furthermore, an increase in BMD with osteosclerosis leads to increased bone stiffness, which increases the susceptibility to fractures in patients with hypothyroidism.[4]

Materials and methods

Selection Criteria: The study was approved by the Ethical and Research Committee of Jawaharlal Nehru Medical College, Belagavi. After finding the suitability as per inclusion and exclusion criteria, patients were selected for the study and briefed about the nature of the study, the interventions used and written, informed consent was obtained. The consented patients were enrolled in the present study. Further, descriptive data of the participants and risk factors were evaluated through a Questionnaire. The study was hospital based and data were collected from thyroid disorder patients either hyper or hypothyroid patients undergoing DEXA scan, attending outpatient department of Orthopaedics and Endocrinology, KLES Dr Prabhakar Kore Hospital and Medical Research Centre, Belagavi, during the period from November 2018-December 2020. A total of 106 patients suffering from thyroid disorder and who were willing to undergo DEXA scan were selected for the study. Only females aged between 18 to 45 years were included in this study.

Inclusion criteria

Women suffering from thyroid disorder both hypo and hyperthyroidism (female, old and new case) and who have not undergone partial or total hysterectomy or oophorectomy.

Exclusion criteria

Patients on following medication which is known to affect calcium metabolism, long-term steroids, phenytoin, eltroxin, heparin, thiazide diuretics, oestrogen, thiazolidinediones (TZDs) were excluded. Patients with following long-term diseases like chronic liver/kidney diseases, chronic skin disease, malignancy and rheumatoid arthritis were also not considered.

Procedure

In all cases the following variables were analyzed: Approximately 10ml blood sample collected level for TSH, T3 and T4 evaluated in all patients. Serum calcium, phosphorous, alkaline phosphatase are normal measured. The bone density based on lumbar vertebra

and neck of femur, for densitometry DEXA scan were used. The DEXA scan was the gold standard in the assessment of BMD. The variables analyzed were age, diet, socio-economic status, sunlight exposure, smoking, alcoholism, occupation, family history, body mass index and duration of thyroid.

Assessment of data: The bone mineral density (BMD in g cm^{-2}) 'T' and 'Z' scores were determined. The 'T' score compares the BMD result with that of a young adult of the same gender with a peak bone mass while 'Z' score compares the BMD result with people of the same age group size and gender. Data were analyzed as follows by following the WHO criteria.

Table 1: BMD Distribution

BMD	Number	Percentage
Normal	64	60.37
Osteopenia	18	16.98
Osteoporosis	24	22.64

Table 2: BMD with status of Hyperthyroidism and Hypothyroidism

		Thyroid state	
		Hyperthyroidism	Hypothyroidism
BMD	Normal	14	50
	Osteopenia	10	8
	Osteoporosis	16	8

Table 3: Age groups with BMD

AGE		Hyperthyroidism+ Hypothyroidism
18-25	Normal	14
	Osteopenia	0
	Osteoporosis	0
26-35	Normal	48
	Osteopenia	10
	Osteoporosis	20
35-45	Normal	2
	Osteopenia	8
	Osteoporosis	4

Figure 1: Dexa Evaluation Apparatus

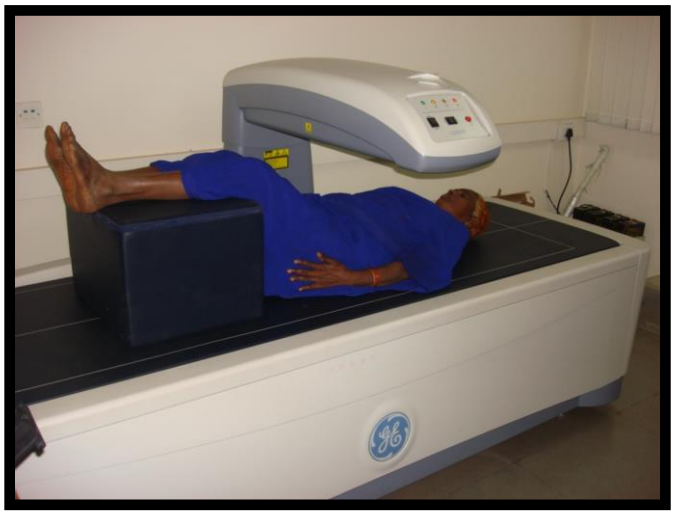

Figure 2: Dexa Scan of Lumbar Spine

Figure 3: Dexa Scan of Dual Hip

Discussion

Thyroid disorders are a chronic metabolic disorder. It shows pleiotropic effects in not just the osseous tissue but the body as a whole. An excess or deficiency of thyroxine (fT4) and triiodothyronine (fT3) can therefore represent a risk for the bones. In addition, osteoporosis is a silent predator which has impending as well as irreversible negative impact on eventual morbidity and mortality. In this cross-sectional study 106 patients with thyroid disorder were studied using DEXA scan by ruling out other secondary causes. The existence of osteoporosis in every patient was checked and matched against any thyroid pathology. This was significant as no other studies have been carried out in this region joining the dots between this condition and its associated risk factors. The overall prevalence of osteoporosis in our sample size was 22.64% (Hyperthyroid osteoporotic were 47.05% and Hypothyroid osteoporotic were 11.11%), osteopenia 16.98% and thyroid patients with normal BMD were 60.37%. Thyroid hormones are extremely important in achieving the expected bone mass. Therefore, if there is any aberration in the balance between resorption and formation; The bone formation as a process is affected (duration might decrease to one third of the baseline) and ultimately causes poor mineralization (approximate loss of 10% of bone mineralization per cycle). All of this eventually leads the consequence of compromised BMD and subsequently, fractures.

Tuchendler D, Bolanowski et.al carried out an evaluation of bone metabolism compared it with the control group.[7] The initial evaluation presented a statistically notable decrease in BMD in the lower femoral neck (as expressed by the Z-Score), in women having hyperthyroidism. J. Foldes et al. conducted a cross-sectional a study, wherein the BMD was calculated at various sites using DEXA in premenopausal women with hyperthyroidism study concluded that no significant effect was observed in premenopausal women with hyperthyroidism in premenopausal women.[6]

Tuchendler D. et al examined the relationship of bone metabolism in premenopausal with hypothyroidism and compared it with the control group.[7] At the initial examination, there was no decrease in BMD in such participants affected by hypothyroidism. No notable difference was found between the femoral neck and the lumbar spine BMD in the hypothyroid group, expressed by the Z-Score. Greenspan S.L, Greenspan F.S. et al. studied premenopausal women with hypothyroidism that measured skeletal integrity treated with long-term L-thyroxine therapy. This study provides positive data and supporting evidence suggesting that long-term treatment with L-thyroxine could lead to changes in skeletal integrity and the associated reduced BMD of the hip and spine.

Age of the sample size in our study ranged from 18 to 45 years. Average age of was 33 years. A majority of osteoporotic thyroid patients numbering 24 (22.64% in that Hyperthyroid osteoporotic were 47.05% and Hypothyroid osteoporotic were 11.11%) were greater

than 30 years of age.

The burden of morbidity from osteoporosis has significant medical, social and financial implications. Life style is the bedrock of facilitating prevention of this condition and the knowledge of the associated risk factors need to be harnessed to assure the same. Some of them include regular exercise, adequate dietary calcium (1000 mg/day), adequate vitamin D (600 IU/day) and cessation of smoking and alcohol intake.[8-10]

In our study majority of the osteoporotic patients among thyroid disorders gave a history of sedentary work (housewives, office work, and retired men), inadequate exposure to sunlight, lower BMI, Vegetarians, smokers, alcoholic and on long term treatment for hypothyroidism. Weight bearing exercises have shown to have a positive impact on the net and peak bone mass, and ameliorates the loss of the same along with mechanical stress in accordance to multiple cross-sectional studies.

Distinguishing between diagnostic and prognostic use of BMD measurement is of prime importance. As a diagnostic tool, it gives data regarding the presence of this condition in accordance to the predetermined cut off values. It can be used as a prognostic tool in determining the risk and probability of osteoporosis in the immediate or prolonged future.

Central DEXA, has long been established a reliable technology for the diagnosis and an aid in the management of declining BMD. It is now extensively used all over the world. As the diagnosis and long-term treatment of osteoporosis and consequent fractures are expensive for the individual as well as the health system, there is a need for careful consideration in determining the risk factors as well as the future course of action on scientific evidence. The detection of a key risk factor should alert the attending physician to a need for further assessment and intervention, pharmacologic as well as non-pharmacologic, to prevent fracture.

Conclusion

The overall prevalence of Osteoporosis in premenopausal women in this study in 22.64% out of which hyperthyroid osteoporotic were more as compared to hypothyroidism. Like most other public health problems of widespread magnitude, treatment alone cannot help a society or world as a whole to cope with the scourge of osteoporosis. Also, since there is no therapy available that has an inherent ability to completely replenish the lost bone mass, the importance of prevention before cure gathers further essence. Thyroid hormonal levels should be assessed in known cases and suspected cases. Hormonal levels should be maintained with in normal levels to reduce effect on bone mineral density. In addition, risk factors that contribute to higher number of falls such as impaired balance, cardiovascular disease, neuropathies should be identified and minimized by implementing a multicentric program that combines regular exercise, vitamin D supplementation, withdrawal of psychotropic medications, visual assessment, environmental hazard assessment and modification, and the use of hip protectors.

Areas of future research should include assessment of skeletal effects of novel drugs, subset evaluation of patients with thyroid in osteoporosis treatment trials, and intervention studies to reduce falls in patients with thyroid disorders.

Acknowledgment: We would like to thank our institute for always backing us up with trust and responsibility. We would also like to extend a warm regard to our

department of orthopedics for always being there for us and helping us out in all the research activities.

References

1. Glaser DL, Kaplan FS. Osteoporosis: definition and clinical presentation. Spine.1997 Dec 15;22(24):12S-6S.
2. Chavassieux P, Seeman E, Delmas PD. Insights into material and structural basis of bone fragility from diseases associated with fractures: how determinants of the biomechanical properties of bone are compromised by disease. Endocrine reviews. 2007 Apr; 28(2):151-64.
3. Consensus statement of the expert group meeting. New Delhi: Osteoporosis Society of India; 2003.
4. Biondi B, Wartofsky L. Treatment with thyroid hormone. Endocr Rev. 2014;35:433–512.
5. Heemstra KA, Hamdy NA, Romijn JA, Smit JW. The effects of thyrotropin-suppressive therapy on bone metabolism in patients with well-differentiated thyroid carcinoma. Thyroid. 2006;16:583–591.
6. Sheppard MC, Holder R, Franklyn JA. Levothyroxine treatment and occurrence of fracture of the hip. Arch Intern Med. 2002;162:338–343.
7. Tuck SP, Francis RM. Testosterone, bone and osteoporosis. Front Horm Res. 2009;37:123–132.
8. Krupski TL, Smith MR, Lee WC, Pashos CL, Brandman J, Wang Q, Botteman M, Litwin MS. Natural history of bone complications in men with prostate carcinoma initiating androgen deprivation therapy. Cancer. 2004;101:541–549
9. Malcolm JB, Derweesh IH, Kincade MC, DiBlasio CJ, Lamar KD, Wake RW, Patterson AL. Osteoporosis and fractures after androgen deprivation initiation for prostate cancer. Can J Urol. 2007;14:3551–3559.
10. Leder BZ, LeBlanc KM, Schoenfeld DA, Eastell R, Finkelstein JS. Differential effects of androgens and estrogens on bone turnover in normal men. J Clin Endocrinol Metab. 2003;88:204–210.