"CROSSSECTIONAL STUDY OF OSTEOPOROSIS AMONG WOMEN WITH THYROID DYSFUNCTION"

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LIST OF ABBREVIATIONS USED

BMD	-	Bone Mineral Density
BMI	-	Body Mass Index
BMC	-	Bone Mineral Content
OB	-	Osteoblasts
OC	-	Osteoclasts
TH	-	Thyroid Hormone
TSH	-	Thyroid Stimulating Hormone
TSHR	-	Thyroid Stimulating Hormone Receptor
OPG	-	Osteoprotegerin
PBM	-	Peak Bone Mass
DEXA	-	Dual Energy X-ray Absorptiometry
SPA	-	Single Photon Absorptiometry
H/O	-	History of
Ht.	-	Height
IP No.	-	Inpatient Number
Lt	-	Left
OP.No.	-	Out Patient Number
qUS	-	Quantitative Ultrasound
pDEXA	-	Peripheral Dual Energy X-ray Absorptiometry
PTH	-	Parathyroid Hormone
QCT	-	Quantitative Computerized Tomography
RA	-	Radiographic Absorptiometry
ROM	-	Range of movement
BUA	-	Broadband ultrasound attenuation
WHO	-	World Health Organization

ROI	-	Region of interest
IVA	-	Instant Vertebral Assessment
LVA	-	Lateral vertebral Assessment
CAD	-	Coronary Heart Disease
NOF	-	Neck of femur
Rt	-	Right
Sl No.	-	Serial Number
USG	-	Ultra Sonography
Wt.	-	Weight
SERMs	-	Selective estrogen receptor modulators
RA	-	Radiographic Absorptiometry
TNF-α	-	Tumor necrosis factor α
IL-6	-	Interleukin 6
RANKL	-	Receptor activator of nuclear factor-kappa B ligand
PA	-	Postero-Anterior
HRT	-	Hormone Replacement Therapy

ABSTRACT

TITLE: "CROSSSECTIONAL STUDY OF OSTEOPOROSIS AMONG WOMEN WITH THYROID DYSFUNCTION"

INTRODUCTION

Osteoporosis is a growing health problem recognized in both developed and developing countries associated with substantial morbidity and socio-economic burden worldwide. Demographic trends with longer life expectancy and a lifestyle characterized by low physical activity contribute to an increasing incidence ofosteoporosis. Thyroid disorders are risk factor for osteoporotic fractures.

AIMS AND OBJECTIVES

1. To know the prevalence of osteoporosis in thyroid disorder patients using dual energy X ray absorptiometry (DEXA) scan
2. To asses the risk factors associated with osteoporosis

MATERIALS AND METHODS

Data was collected from Thyroid disorder patients attending outpatient at Orthopaedics and Endocrinology department who were willing to undergo DEXA Scan. DEXA was done at hip and spine to assess BMD. Various other risk factors were evaluated through a questionnaire. T Scores and Z Scores were evaluated for the presence of osteoporosis based on WHO Criteria.

RESULTS

53 thyroid disorder patients were studied. Prevalence of osteoporosis at the spine was 20.75 % while at the hip was 15.09%. The overall prevalence of osteoporosis was 20.75% out of which hyperthyroid osteoporotic were 41.18% and hypothyroid osteoporotic were 11.11%. Predominantly osteoporotic thyroid patients had lower body weight and lower body mass index with reduced duration of exposure of sunlight. Time since on medication for thyroid disorder was also associated with increased risk of osteoporosis in hypothyroid patients. The prevalence of osteoporosis was higher among the group with positive history of alcohol.

CONCLUSION

High prevalence of osteoporosis in Hyperthyroid patients and hypothyroid patients who are on long term treatment is a cause for concern. Measures such as adequate Vitamin D and calcium intake, physical activity, and exposure to sunlight will be useful in preventing occurrence of osteoporosis. 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, Thyroid disorder, DEXA

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INTRODUCTION

Osteoporosis is a skeletal condition in which characteristic feature is ameliorated density (mass/volume) of an otherwise normal mineralized bone.[1] The biomechanical integrity of trabecular or spongy bone may be compromised causing it to lose elasticity and cortical bone may become more porous and thin. While thinner bone alone is not necessarily more prone to fracture, bone that is both thinner and has compromised biomechanical properties is more prone to fracture.[2]

Osteoporosis is defined as "a systemic skeletal disease characterized by low bone mass and micro-architectural deterioration of bone tissue, with a consequent increase in bone fragility and susceptibility to fracture".[3,4] This suggests that the assessment of bone mineral density (BMD) is a pivotal component in diagnosis of the disease.

The **WHO** defines it as follows: **(Table 1)**

Level	Definition
Normal	Bone density is within 1 SD (+1 or -1) of the young adult man.
Osteopenia	Bone density is between 1 and 2.5 SD below the young adult mean (-1 to -2.5 SD).
Osteoporosis	Bone density is 2.5 SD or more below the young adult mean (-2.5 SD or lower).
Severe osteoporosis (established osteoporosis)	Bone density is more than 2.5 SD below the young adult mean, and there have been one or more osteoporotic fractures

Dual energy X-ray absorptiometry (DEXA) scanning is the best method to measure BMD.[3]

Of prime concern are vertebral (spinal) and hip joint fractures. The former can result in debilitating sequalae, including loss of height, intense back pain and permanent deformity. The latter is a common indication for surgery and hence can subsequently cause a loss of working days and inadvertently of independent living.[3]

Thyroid hormone (TH) plays an important role in normal endochondral ossification and is essential for skeletal development, linear growth, maintenance of bone mass, and efficient fracture healing .[7] Therefore, if there is any aberration in the balance between resorption and formation, the osseous synthesis 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 to the aforementioned consequence of compromised BMD and subsequently, fractures.

The hormone T3 acts on the TRa receptors in both OBs (OB) and osteoclasts (OC). It is believed to accelerate synthesis of OBs (bone formation) as well as osteoclasts (bone resorption), thereby increasing the turnover rate of the bone.

It still remains to be asserted whether T3 acts directly or indirectly on the osteoclast using the osteoprotegerin activator / receptor of the kappa B ligand pathway (OPG / RANKL) of the central factor .The pituitary gland, like T3, might also act directly on bone cells through the action of the thyroid stimulating hormone (TSH) on the TSH receptor (TSHR) present in both, the OBs and osteoclasts.[8,9]

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 as previously stated, are important 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 bone resorption process and a greater net gain in bone. In western countries the peak incidence of osteoporosis occurs at about 70-80 years of age, in India it may afflict those 10-20 years younger, at age 50-60.[10]

The world's Osteoporosis - **Time Bomb** " is ticking, with projected global burden of Osteoporosis hip fractures expected to exceed six million by 2050".[11]Thirty percent to fifty percent of women and 15-30% of men have lifetime risk for osteoporosis and osteoporosis related fractures all over the world .[12]

Estimates indicate that the number of osteoporotic hip fractures occurring in the world will rise from 1.66 million to 6.26 million by the year 2050, thereby implying an urgent need for preventive strategies. Projections indicate that by the year 2050 45% of osteoporotic fractures will occur in Asia.[13]

Lifestyle factors including smoking, low levels of weight-bearing physical activity and compromised nutritional status can further contribute to fracture risk.[14]

A misconception exists that it is always a result of bone loss, when in fact bone loss is a common occurrence as both women and men age. Women however, typically have a lower peak bone mass (PBM) than men and experience rapid bone loss during the first 5-10 years of the postmenopausal period.[5]

The combination of bone loss relative to PBM and rate of postmenopausal bone loss results in a higher fracture risk in women earlier than men.[5]

The bone remodelling cycle is regulated not only by hormones, but also cytokines, tumour necrosis factor (or TNF-a) and growth factors. Regulatory proteins can be influenced by a number of factors, including nutritional status (e.g. compromised vitamin D status), physical activity, age and illness, which subsequently alter bone turnover.[15]

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. 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, subclinical hypothyroidism, subclinical hyperthyroidism and their treatments in bone pathology.

Osteoporosis not only causes fractures, it causes people to be bedridden with problems of back pain, loss of height, kyphosis, pneumonias and pulmonary thromboembolism.

AIMS AND OBJECTIVES

- Primary objective –

To find out the prevalence of osteoporosis in thyroid disorder patients using dual energy X ray absorptiometry (DEXA) scan

- Secondary objective –

To find out the risk factors affecting osteoporosis:

1. Age
2. Diet
3. Socio economic status
4. Sunlight exposure
5. Smoking
6. Alcoholism
7. Occupation
8. Body Mass Index

REVIEW OF LITERATURE

Bone is a living, dynamic and specialized connective tissue with a mineralized collagen scaffold to support the skeleton of the body, which is perpetually renewed and leads to a complete turnover of the adult skeleton every ten years. [16]Bone tissue is generally divided into two types, cortical and trabecular bone, which are identical in their chemical composition. Cortical bone is a dense, compact structure that is very resistant to bending and torsion and, with the exception of the periosteum, has a slow turnover rate. It forms the diaphysis of long bones and the outer part of all skeletal structures. It is primarily used to give mechanical strength and protection to vital organs. Trabecular bone is less dense, more elastic and has a higher turnover rate than cortical bone. Anatomical sites that are common in the trabecular bone include the epiphyses and metaphysis of the long bones and are also the main component of the ribs, shoulder blades and flat skull bones. Bone not only provides strength and support for the body, but also serves as a place for the development and storage of blood cells. Bones accumulate micro-damage from loading, but are unique in their ability to repair themselves. [17-19]The complexity and processes involved in the anabolic and catabolic aspects of bone metabolism (i.e. formation by OBs and absorption by osteoclasts) and the influence of nutritional and environmental factors all contribute to susceptibility to diseases and disorders that may affect bone health.

The Skeletal Anatomy:[20]

The proximal femur consists of spongy bone, invested by a thin layer of compact bone. The trochanteric region includes more of spongy bone.

Trabecular System: Fig 1

In 1838, **Ward** described the internal trabecular system of the femoral head. The trabeculae are oriented along the lines of stress. There are five normal groups of trabeculae, as defined by Ward.

Primary Compressive Trabeculae: These are the strongest trabeculae, extending from the medial cortex at the base of the femoral neck to the subchondral bone of the superomedial part of the head.

Primary Tensile Trabeculae: These extend from the inferior region of the foveal area across the head and superior portion of the femoral neck into the greater trochanter, hence to the lateral cortex.

Secondary Compressive Trabeculae: These extend from the medial femoral cortex in the region of the lesser trochanter towards the greater trochanter.

Secondary Tensile Trabeculae: These extend from the lateral femoral cortex, inferior to the primary tensile trabeculae towards the middle of the femoral neck.

Greater Trochanteric Trabeculae: These extend from the superior border of the greater trochanter to its base. The space bounded by the primary compressive and tensile trabeculae and the secondary compressive trabeculae is known as the **Ward's Triangle**.

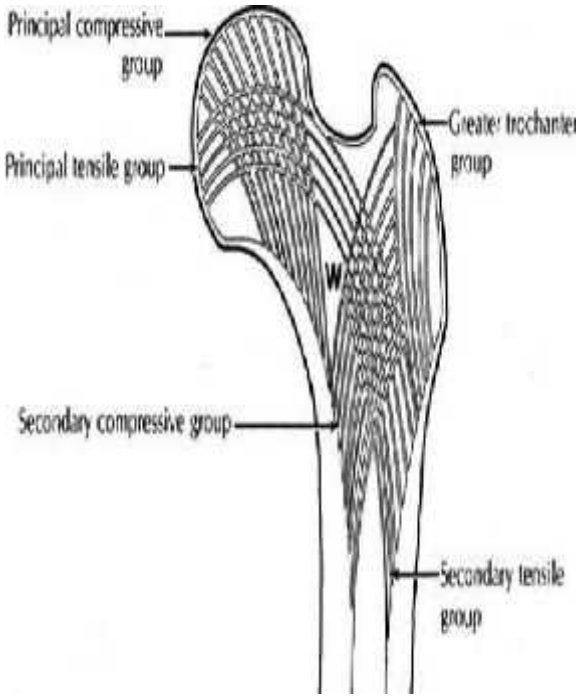

Fig 1: Trabecular pattern of the femur

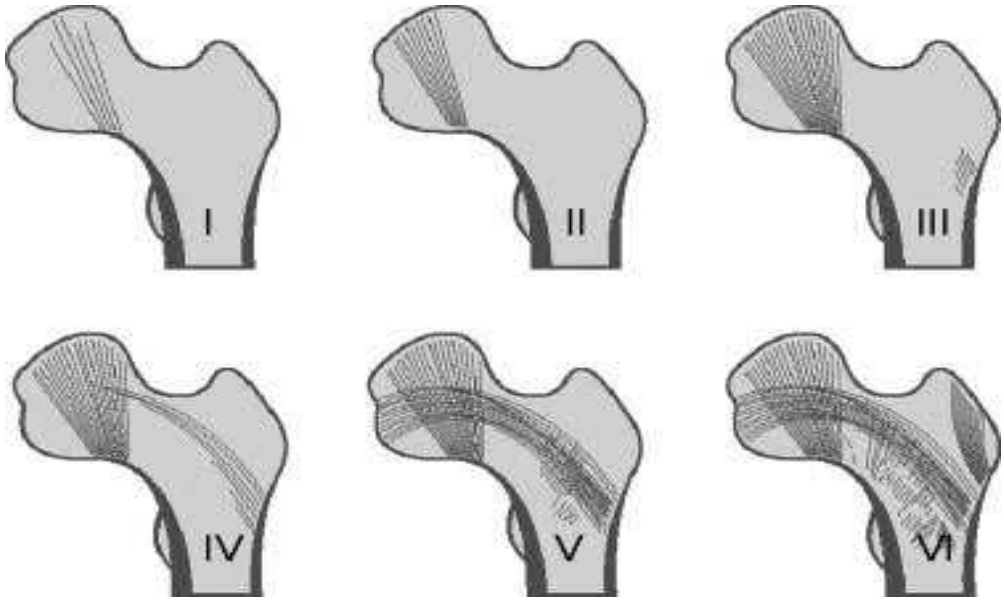

Fig 2: Singh's Index

Singh M.

Introduced a method to determine the degree of osteoporosis by evaluating the trabecular pattern of the proximal femur as observed on radiographs. The degree of osteoporosis is classified from 1 to 6.

Singh's Index: [21]

Grade VI: All normal trabecular groups are visible, and the proximal end of the femur appears to be completely occupied by cancellous bone.

Grade V: The structure of the principle tensile and compression trabeculae is accentuated. The Ward triangle appears prominently.

Grade IV: Principle tensile trabeculae are significantly reduced, but can be traced from the lateral cortex to the top of the femoral neck.

Grade III: There is a break in the continuity of the principal tensile trabeculae opposite the greater trochanter. This grade indicates definite osteoporosis.

Grade II: only the principal compressive trabeculae are highlighted; The others were more or less completely absorbed.

Grade I: Even the principal compressive trabeculae are significantly reduced and no longer noticeable.

Osteoporosis

Osteoporosis is a frequent age related disease characterized by generalized loss of bone mass which can result in fracture with trivial fall.[22,23] Fracture risk prediction is a critical element in fracture prevention. This led to the definition of osteoporosis evolving over the years to improve predictability. It was not until 1994 that the WHO officially defined osteoporosis in terms of BMD and fracture history. [21] This was the result of the efforts of an international panel of scientific experts to

evaluate the risk of fractures and its possible use in the detection of postmenopausal osteoporosis. The committee examined various approaches to define osteoporosis based on BMD. However, not everyone was able to overcome the problem of overlapping BMD between those with fragility fractures and those without fragility fractures. Assessment of bone minerals has been found to provide a risk index, similar to how hypercholesterolemia poses a risk of coronary heart disease. However, this index did not reflect all the risk elements. The panel then established four general diagnostic categories based on bone mineral content (BMC) and proximity to baseline for young adults. These categories include normal, osteopenia, osteoporosis, and severe osteoporosis.[24] The panel also emphasized the importance of distinguishing between the diagnostic use of BMD measurements by providing information on the presence of osteoporosis with the chosen threshold values and the prognostic use, in which bone density values are considered a factor of risk.[24]

The diagnosis of osteoporosis is based on **measurements of BMD** and the WHO defines it as follows: **(Table 1)**

Level	Definition
Normal	Bone density is within 1 SD (+1 or -1) of the young adult man.
Osteopenia	Bone density is between 1 and 2.5 SD below the young adult mean (-1 to -2.5 SD).
Osteoporosis	Bone density is 2.5 SD or more below the young adult mean (-2.5 SD or lower).
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Dual energy X-ray absorptiometry (DEXA) scanning is the best method to measure BMD.[3]

Table 2: Etiology of osteoporosis

Secondary causes	
Endocrine	**Cushing's syndrome** [25-34] **Hyperthyroidism** [35-45] **Hyperparathyroidism** [51-60] **Diabetes mellitus** [61-70] **Hypogonadism** [46-50]
Nutritional deficiencies	**Calcium** **Vitamin D** **Malabsorption** **Malnutrition**
Immunological and inflammatory	**Inflammatory bowel disease** [71-74] **Rheumatoid arthritis** [75-81]
Drugs	**Corticosteroids** [25-34] **Anticonvulsants** [82-86] **Anticoagulants** [87-95]
Neoplastic	**Leukemia** **Neuroblastoma**
Immobilisation	**Cerebral palsy** **Paraplegia**

Clinical Manifestation

Patient with osteoporosis however, have been reported to present with certain generalized complaints such as a dull pain, tenderness, stiffness, swelling, kyphosis, loss of height and vasomotor disturbances. Bending and lifting increases the risk of vertebral fractures. Pelvis, hip, femur, vertebral, humerus, forearm are commonly occurring fractures in osteoporosis. Vertebral muscle strength is documented to be lower in female population than their male counterparts.

OSTEOPOROSIS RISK FACTORS:

Risk factors are characteristics that increase the probability of developing a particular disease or illness. Risk factors for osteoporotic factors include age, sex, race, geographic region, diet, lifestyle, hormonal status, bone density, bone quality, body mass index, and medical comorbidities.

Clinical risk factors in the World Health Organization.

Fracture Risk Assessment Tool (FRAX) [96]

- Country of residence
- American ethnicity. models only: Caucasian, black, Spanish and Asian)
- Age (allows ages between 40 and 90 years)
- sex
- Weight and height to calculate the body mass index.
- Previous fragility fracture, including radiological vertebral fractures.

- Family history of osteoporosis (father with hip fracture)
- Current smoking
- Glucocorticoid use (prednisolone 5 mg or more daily for three months and more) current or past)
- Rheumatoid arthritis (diagnosis confirmed by the doctor)
- Alcohol consumption (3 units a day or more)
- Secondary osteoporosis
- Bone mineral density works without bone mineral density. (Nelson, Watts, 2010)

1.1. Age

Older people are more predisposed to osteoporotic fractures than the young people. In particular, the possibility of hip fractures increases markedly after 70 years of age, irrespective of the gender and geographical location. This increased fracture risk is believed to be due to both the senile reduction in BMD of the proximal femur and the age-related increase in falls. It is also related to the increase in comorbidities in older people (Dontas, Yiannakopoulos, 2007). [97]

BMD decreases and, in turn, the risk of osteoporosis increases with age. A considerable rise in prevalence has been shown every decade after age 60. Survey III of the US National Survey of Health and Nutrition. USA (NHANES) found that the prevalence of osteoporosis in non-Hispanic American white women was 27 percent (50-59 years), 32 percent (60-69 years)), and 27 percent was 41 percent for those 70 and older.[98]

1.2. Demographic factors

Race is another risk factor for osteoporosis. Racial differences in bone health have been associated with several key traits. Differences in body size [99], bone size [100], skeletal loss rate [101-103], and hip geometry [104,105] have been concluded to partially explain the observed differences in racial fracture risk. These differences have multifactorial explanations, including differences in bone metabolism [106] and the onset of puberty [107]. Longitudinal data with baseline BMD values and fracture results for nonwhites are limited, and it is still an enigma whether the T scores obtained from BMD measurements in nonwhite females have the same value in terms of fracture prediction.[108] Rate of fractures are higher in white Scandinavian women than in North American women of matched age. The risk of osteoporotic fractures throughout life at age 50 was estimated at 46 percent in women and 22 percent in men in Sweden, 40 percent in USA, and 13 percent in the United States. In India Men to women than Westerners (Dontas, Yiannakopoulos, 2007) [14]. Afro-Caribbean women have a higher BMD than white women at any age because the maximum bone mass is higher and the rate of loss is slower. [109]

1.3. Hormonal factors: gender differences

The maximum bone mass in women is lower than in men.[110] The increased bone loss in menopausal women and its higher decline tendency compared to men suggest that hip fractures occur in women of all ages. Most researchers report a 2: 1 ratio between hip fractures in males and females over the age of 65.[110] Other hormonal factors that are implicated include premature menopause, 1^o or 2^o amenorrhea, hyperthyroidism, hyperadrenocorticism, and primary and secondary hypogonadism in men (Dontas), Yiannakopoulos, 2007). In women, the timing of menopause and

subsequent decrease in estrogen has a say in the rate of bone loss. Similarly, hypogonadal men may have higher bone loss rates and higher fracture rates later in life (Anne Sutcliffe, 2005). [111] Osteoporosis is less common in men, but it is still a major problem. In the Framingham osteoporosis study, the annual percentage of bone loss was 0.86 to 1.21 percent in women in various settings and 0.04 to 0.90 percent in men. However, secondary causes of osteoporosis are more common in men and affect approximately 40 percent of cases. [112]

1.4. Family and medical history

Fracture risk factors include a history of fragility fracture, a family history or genetic factor fracture, low BMD, low BMI, weight loss, a resting heart rate of more than 80 beats per minute, arthritis. rheumatoid, the chronic use of corticosteroids, anticonvulsants, and loop diuretics and risk of falling (eg, due to neuromuscular, cardiovascular, and vestibular disorders, visual disorders, dementia, consumption of certain medications, and polypharmacy) (Dontas, Yiannakopoulos, 2007). [14] The risk of an osteoporotic fracture is approximately doubled in the presence of a previous fracture.[113]Individual BMD decreases with increasing numbers of family members with osteoporosis. In general, family history is a more sensitive predictor of osteoporosis risk than the history of single mothers or fathers.

1.5. Genetics

Genetic factors play a role in BMD between the ages of 20 and 30 and in the loss of BMD post-menopause.[114] Fifty percent of the maximum bone mass, geometry, strength, and bone architecture depend on genetic makeup.[115]Other studies report a maximum bone mass of 75 percent depends on genetic factors such as the vitamin D

receptor gene, the estrogen receptor gene, and the collagen receptor gene.[116] The strong association between BMI and maximum bone mass may be due in part to common genetic influences. (Chaudhri Tauseef, 2006).[117] Osteoporosis has a genetic component, and if a parent has a disease or a history of hip fracture, a person has an increased risk of fracture. (Elliott Mary, 2011). [118]

1.6. Nutrition

Trace elements such as copper, manganese and zinc are necessary for the development of growth and the maintenance of healthy bones (ChaudhriTauseef, 2006).[117] Nutritional components such as magnesium, fluoride, ascorbic acid and vitamin K act biologically at the bone level. Vitamins A, B6 and D are also essential for physiological bone formation. [119]

Calcium

Calcium is important for different physiological body functions, such as muscle contraction, blood clotting, and transmission of the nerve impulse.

Cross-sectional studies showing increased calcium intake in younger age group may increase BMD in children, adolescents, and young women. [120] With low levels of calcium in the blood, parathyroid hormone is excreted, leading to the formation of calcitriol, leading to bone resorption and calcium release. A high serum calcium inhibits this and the bone turnover normalizes. Chronic low calcium intake from food reduces bone mass and increases the risk of osteoporosis and broken bones.[122]

A study from India shows in the lowest socioeconomic groups; Calcium intake is only 300 mg / dl, which is 700mg less than the required amount. Envelope in India 40% of people from socioeconomically weak groups suffer from chronic energy deficiency and have enough energy, protein, calcium and other micronutrients. [123]

Vitamin D

Vitamin D, which is known for improving absorption of calcium from the intestine, is also vital for maintaining bone mass through its action on other cellular processes such as bone mineralization. Vitamin D facilitates bone mineralization at the OB level by improving differentiation and stimulating proximal tubular phosphate absorption in the kidney.

The latter function improves bone mineralization by contributing phosphate, one of the two main crystalline salts deposited in the mineralized bone matrix. A meta-analysis by Papadimitropoulos et al. ,[124] examined all randomized, placebo-controlled studies with vitamin D and its analogues in the past two decades. They reported a significant decrease in spine fractures (i.e., a 37% risk reduction) in women taking vitamin D compared to placebo. Other research that supports the positive effects of vitamin D includes Chapuy et al.[125]Who distributed 800 international units of IUU) cholecalciferol or vitamin D3 to nursing home residents for 18 months. A 35% decrease in the incidences of hip fracture has been reported with this supplemental regimen. [126]

1.7. Alcohol

Chronically heavy alcohol consumption is generally seen as a risk factor for osteoporotic fractures and low bone density.[127] Although effects of controlled alcohol

intake on the skeleton is uncertain, it can affect calcium metabolism and lead to reduced bone density.

1.8. Smoke

Smoking is an established main risk factor for bone loss.[128,129] Current and previous smokers in the Hawaii Osteoporosis Study (1303 men ages 51 to 82 years old when recruiting) had a lower BMD, especially on the calcaneus and radius.[130] A dose-dependent relationship to both the duration and the number of cigarettes smoked was found. Currently smoking has a direct association with significantly increased risk of all types of fractures in men and women, with the effect slowly waning after a person stops smoking.

1.9. Exercise

Physical activity is also a risk factor that affects bone remodeling.[131-133] Unloading the skeleton reportedly reduces activity and number of OBs,[134-136] which Garetto et al. Probably due to decreased osteoprogenitor cell proliferation.[137] Howe et al. showed in a comprehensive review that weight training in postmenopausal women had a significantly higher effect on BMD than controls who did not exercise. [138] Physical activity is essential for the skeleton because the consequent stress and muscle activity stimulate bone formation and increases bone mass, while immobilization leads to rapid bone loss. The positive reactions of the skeleton are site-specific for the stress pattern. The initial age of the activity is critical, with the benefit to the bone doubling if the activity is started before or during puberty.

1.10. menopause

Post-menopausal osteoporosis in women, leads to an increased morbidity and mortality.[139] The same report assumes that this is an easily avoidable health burden for women and is easily avoidable. In the world, the average age for women reaching menopause is around 52, but it is comparatively less in India and more hysterectomy has been reported in urban India.[140] According to WHO, India by 2030 the postmenopausal women population will be the second highest in the world after China. [141]

1.11. Lower body weight and BMI:

Literature shows low BMI, possibly associated with more osteoporotic fractures and osteoporosis. A low BMI, which causes lower maximum bone mass and has resulted in more bone loss and leads to osteoporosis and osteoporotic fractures.[142] Epidemiological studies show that low body weight is a major determinant and risk factor for hip fractures. [143]

1.12. Low sun exposure

Certain multicentric studies show a strong relation between lower sun exposure and hip fractures in those over 50 years of age have been published.[142,144] Vitamin D is essential for bone health and has had an impact on the growth and development of children. Its deficiency leads to increased bone turnover, increased bone loss and in turn, fractures. [145]In developing countries, especially in India, Vitamin D levels are comparatively lower than western colleagues, and studies show that there is one Correlation with BMD. [146]

1.13. Secondary causes

Gastrointestinal diseases are also among the unchangeable risk factors for osteoporosis. Malabsorption of vitamins and minerals, especially vitamin D and calcium, underlies many conditions. There is consistent evidence negative correlation between BMD, Cohn's disease and Ulcerative colitis due to a decrease in bone formation and accelerated bone resorption. Hematological disorders (e.g. thalassemia and pernicious anemia) and hypogonadal conditions, thyrotoxicosis and anorexia nervosa are the main causes of secondary osteoporosis in men and women. [147]

BMD assessment

The densitometric measurement of bone mass is of central importance for the diagnosis of osteoporosis and the decision about the treatment to prevent fractures. BMD measurements are used to make a diagnosis of post-menopausal osteoporosis, determine the risk of fracture, identify intervention candidates, and evaluate changes in bone mass over time in both treated and untreated patients.

Techniques for assessing bone mass

Various techniques are available for assessing bone mass. The method should be as safe and precise as possible for diagnostic use in patients. In addition, advantages such as low costs, quick and easy checking, good reference data and easy to interpret results are important factors. However, two main methods are used in the clinic, DEXA and quantitative ultrasound (QUS), which are described in more detail below.

Quantitative computed tomography (QCT) is mainly used in research, and the literature also contains descriptions of magnetic resonance imaging (MRI) and digital radiogrammetry for assessing bone mass.

2.1 Quantitative computed tomography

QCT using standard CT equipment is a technique that allows a three-dimensional assessment of the BMD. Cortical and trabecular bones can be assessed separately, the bone geometry can be measured and the trabecular bone structure can be mapped. There are machines for both axial and peripheral techniques, in which the latter are primarily used in research.

The limitations of the QCT are a 10-20 times higher radiation dose compared to DEXA, limited reference data, higher costs and the need for more qualified personnel both in the laboratory and when interpreting the results. [148]

2.2 Quantitative ultrasound

QUS is a technique for estimating bone strength or quality by analyzing the alternation of an ultrasound wave after passage through trabecular and cortical bones. The most common skeletal site for this method is the heel and calcaneus. The equipment is comparatively inexpensive, small and portable. QUS does not predict fracture or standard axial DXA, but is comparable to peripheral to moderate to strong correlation DEXA. QUS has also proved to be predictive of fractures regardless of bone mass. It is therefore assumed that another aspect of bone strength is measured along with BMD. The ultrasound signal is affected by various material properties, including bone mass, microarchitecture and tissue elasticity. A clear advantage of QUS is that the patient is not exposed to ionizing radiation. However, since the

precision is less than that of DXA, the method is not recommended for follow-up or clinical studies and is therefore not preferred for clinical evaluation. [149]

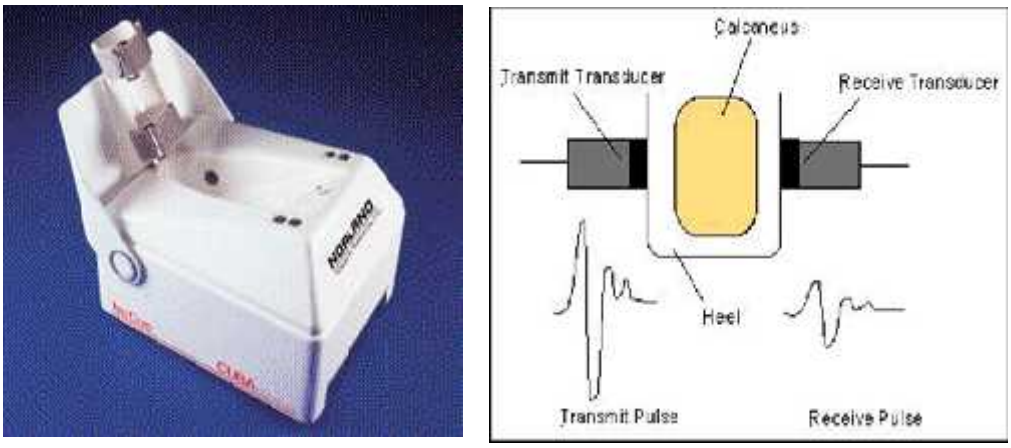


Fig 3: Calcaneal ultrasound

2.3 Dual energy X-ray absorptiometry

The DEXA technology currently used is a further development of earlier methods. Single x-ray absorptiometry followed single and double photon absorptiometry with higher radiation doses and longer exposure times. The modern DEXA technique was introduced in the 1980s and has been the standard method for assessing the BMD for the diagnosis of osteoporosis since 1994. The DEXA measurement uses two X-rays with different energy levels, with the low-energy beam weakening soft tissue and the high-energy beam weakening soft tissue and bone. Subtracting the low energy scan from the high energy gives a BMC and an estimate of density with area BMD in g / cm^2, which is the most commonly used unit. BMC, BMD, lean mass and fat mass can be obtained from a whole-body DEXA scan, but data from regions such as the hip and lumbar spine are mainly used in clinical settings. These locations are most accurate for predicting hip or vertebral fractures. DEXA enables an accurate, precise and reproducible assessment of bone mineral

content and BMD and assists in the detection of osteoporosis before the actual occurrence of clinical fractures. Of all the technologies available for measuring the BMD, the central DEXA is the technology of choice.[150]

DEXA scans of the hip [1]

The BMD of the hip can be measured in different regions, including the NoF, trochanter, intertrochanter, ward triangle and total hip. With most of the interest in the entire hip, measurement errors can be minimized. However, caution is required when measuring the Ward triangle. Due to its small area, there is a higher probability of measurement errors, and the anatomical location itself can be variably defined by different manufacturers.

In contrast to the spine, in which the cancellous bone is evenly distributed and thus has a uniform pattern of bone loss, the hip has a variable BMD, since different areas of the femur consist of different percentages of cancellous bone and have different fracture risks

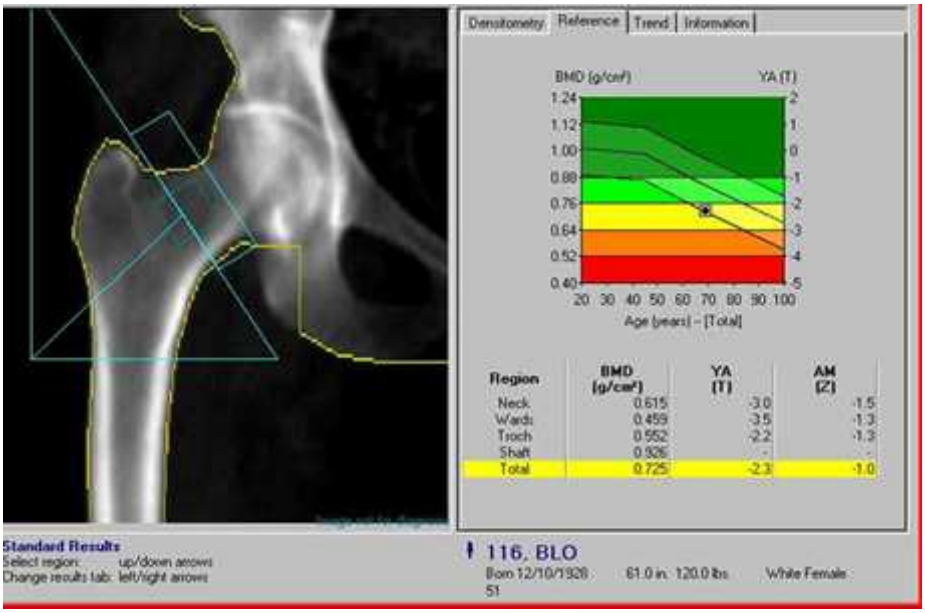


Fig 4: DEXA scan of the hip

DEXA scans of the spine [1]

DEXA scans of the spine can focus either on the postero-anterior PA projection or on a side view of the lumbar vertebrae. Typically, a region is scanned with L1-L4 or L2-L4. General limitations in measuring vertebral bone density in the PA projection include the confusing presence of osteophytes, calcification of the paraspinal ligaments, calcification of the aorta, and vertebral scoliosis.

In addition, existing vertebral body compression fractures can be interpreted as increased bone density. Side views eliminate the potential artifacts from osteophytes or aortic calcifications. However, side view limitations include the increased amount of soft tissue in this projection and the overlap of the ribs and pelvis. This, in turn, reduces the number of vertebrae that can be analyzed, which affects the accuracy of the measurements and thus reduces the usefulness of lateral measurements to track response to therapy.

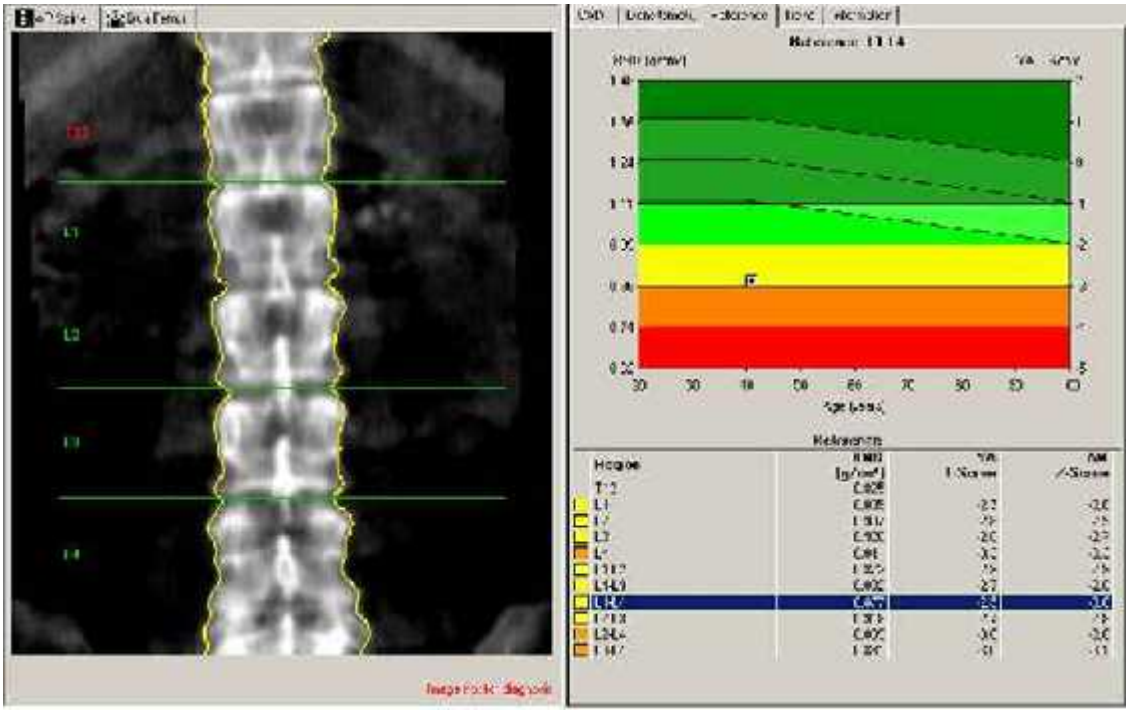


Fig 5: DEXA scan of the lumbar spine

DEXA for measuring the BMD and for detecting vertebral fractures [151]

Another recent consideration when choosing the anatomical location is DEXA's ability to detect vertebral fractures as a supplement to a BMD measurement. Only 20-30% of vertebral body fractures are clinically recognized, the rest are detected on the side on x-rays of the lateral spine. X-rays of the lateral spine are not recommended as part of the risk assessment for osteoporosis due to the cost, radiation exposure and that a separate procedure would be required for the X-ray along with BMD study.

However, with DEXA, images of the lateral spine can be obtained, making it possible to search for vertebral body fractures while a subject is evaluating the BMD. This imaging can be referred to as morphometric x-ray absorptiometry (MXA). However, manufacturers of DEXA technology have also referred to this procedure as "Instant Vertebral Assessment" (IVA) or "Lateral Vertebral Assessment" (LVA).

BMD measurement reports

BMD measurement reports typically include the following:

a) An image of the bone within the scanned area. The image was checked to see if there were any confusing artifacts. When comparing the study to a previous one, it is essential that the same anatomical location is evaluated (i.e. the same region of the hip).

b) The BMD expressed in g / cm^2. The BMC of the scanned region of interest (ROI).

c) Normal values depend on the reference database.

d) The T and Z score values are based on a comparison of the patient's BMD with the reference database. The results can also be available as a percentage of the mean of the reference population.

Possible pitfalls in BMD measurements at different locations [1]

Lumbar spine (antero-posterior view)	• Osteoarthritis (posterior elements, vertebral body, osteophytes) • Fracture • Hyperlordosis/scoliosis • Calcified aorta • Radio-opaque material (e.g., Thorotrast) • Size of the region of interest • Presence of barium • Fat tissue (differences between individuals and redistribution overtime)
Lumbar spine (lateral view)	• Ribs, pelvis overlying the region of interest • Scoliosis • Fat tissue (differences between individuals and redistribution overtime)
Proximal femur	• Size of the region of interest • Location of the region of interest • Leg position (rotation, abduction)

OSTEOPOROSIS - PREVENTION AND TREATMENT

Identify patients who are prone to fractures and optimize pharmacotherapy. A comprehensive risk factor analysis was carried out.

Two important areas of risk factor assessment that call for attention especially for the elderly include evaluating bone-related risk factors related to BMD and non-bone-related risk factors that are variables related to fracture risk but not bone density and increases the risk of fracture regardless of the diagnosis of osteoporosis. Age is

not a disadvantage for starting osteoporosis prevention strategies. The health care professional must also educate and evaluate clients about risk factors, nutritional recommendations, physical activity and pharmacological options, and other lifestyle changes that are necessary to achieve and maintain optimal bone density and health. Diagnosed osteoporotic individuals can also benefit from using the same evaluation and educational principles to reduce or stop further bone loss.

There are now several treatments for osteoporosis that improve BMD and ameliorate the incidence of fractures. Important pharmacological interventions.

3.1. Calcium & Vitamin D

Both are usually administered as a combination treatment of both substances. It is not entirely clear to what extent vitamin D and calcium alone decrease chances of osteoporotic fractures. Therefore, they are often administered as an additive to other osteoporotic drugs. [152]Clinical efficacy can be improved by taking 1000-1200 mg calcium / day with vitamin D 700-800 IU / day.

3.2. Bisphosphonates

The bisphosphonates are analogues of inorganic pyrophosphate and inhibit bone resorption. Products currently available in the market are etidronate, alendronate and risedronate. Etidronate, the oldest bisphosphonate, has been established to reduce chances of vertebral fractures, while alendronate and risedronate also show a risk reduction for hip and wrist fractures. [153,154]

3.3. Hormone replacement therapy (HRT)

It stops bone loss by inhibiting bone resorption and subsequently fractures. HRT has however recently been shown to have several extra-skeletal effects.[155] The combination of estrogen and progesterone to treat women who have not undergone hysterectomy has shown to increase the risk of cardiovascular disease and breast cancer and to lower the risk of colon cancer. According to a government-sponsored study known as the Women's Health Initiative (WHI), hormone therapy can reduce the risk of hip fractures and symptomatic fractures of the spine by 34% and all other fractures by 24%. [156]

3.4. Selective Estrogen Receptor Modulators (SERMs)

They are hormone-like drugs that affect multiple tissues. These drugs can selectively block estrogen from certain tissues, namely the breast, while increasing availability in other areas such as bones. In the simplest sense, the goal of these drugs is to maximize the positive actions of estrogen on the bones and to minimize the harmful effects of the hormone on the breast and endometrium. Two SERMS, tamoxifen and raloxifene (Evista) are currently used in postmenopausal women. Raloxifene is FDA approved for the treatment and prevention of osteoporosis, while tamoxifen is used for the same in breast cancer. Currently the only SERM on the market, raloxifene has been shown to reduce the risk of vertebral fractures and breast cancer in postmenopausal women with low BMD. [157] Additionally, raloxifene has been shown in a study to reduce the risk of coronary heart disease (CAD) in patients at high risk for cardiovascular disease. On the downside, raloxifene shows a risk of thromboembolic events.

3.5. Calcitonin

Calcitonin, an endogenous polypeptide hormone reduces osteoclastic bone resorption. Salmon calcitonin, 200 IU daily intranasally, is approved for the treatment of established osteoporosis to control vertebral fracture incidence. The recommended dose has been shown to reduce vertebral fractures in people with osteoporosis, but there is no reliable evidence to reduce non-vertebral and hip fractures. Calcitonin is contraindicated in patients with hypocalcemia and patients with nasal ulcers. The most commonly observed side effects are local reactions, such as rhinitis and nasal problems.[158]

3.6. Peptides of parathyroid hormone

Peptides have intermittent anabolic skeletal effects with increased bone formation. The effects are most pronounced on cancellous bone and can vary between cortical areas.[159] Teriparatide (recombinant human PTH 1-34) is also approved for the treatment of osteoporosis in men predisposed to fracture and is administered subcutaneously at 20 µg / day. Teriparatide is also used for the treatment of osteoporosis associated with systemic glucocorticoid therapy. The recommended treatment duration is 18 months. Vertebral and non-vertebral fractures have been reported to decrease in people with osteoporosis, but no data is available for hip fractures. It is however not advisable to be used in hypercalcemic patients, metabolic bone disorders other than osteoporosis, severe renal dysfunction, previous skeletal radiation, and malignancies affecting the skeleton. It should be used cautiously in patients with moderate renal impairment. Side effects include headache, nausea, dizziness, and postural hypotension.

3.7 strontium ranelate

Reduces bone resorption and simultaneously allows more bone formation.[160] Clinical studies show strontium ranelate minimizes the overall risk of fractures in postmenopausal women with low BMD. Strontium ranelate has not identified any serious adverse events. The most common side effects were diarrhea and nausea, which generally occurred in the first three months of treatment. A less common adverse event was venous thromboembolism, which was somewhat more common among strontium ranelate users. [161]

3.8 Denosumab [162]

Denosumab is a fully humanized monoclonal antibody against the ligand central factor receptor Kappa B (RANKL), a significant regulator of osteoclast development and activity. It is approved for the treatment of osteoporosis in postmenopausal women predisposed to fracture and is administered subcutaneously 60 mg every six months.

The incidence of vertebral, non-vertebral, and hip fractures evidently decreases in men with osteoporosis. Denosumab is generally contraindicated in hypocalcaemia or hypersensitivity to any of the components. It is contraindicated in pregnancy or pediatric population of 18 years old. No modification of dose is required in patients with renal pathology. Hypocalcemia should be corrected with an adequate intake of calcium and vitamin D before starting therapy. Side effects are skin infections, mainly cellulitis and hypocalcemia. Osteonecrosis has rarely been reported in clinical trials in patients receiving denosumab 60 mg every six months for osteoporosis.

Table 3: Exercises for Osteoporosis to Increase Bone Strength

Subjects and Reference	Physical exercise program	Effects on bone and other effects
Premenopausal women	Upper and lower body resistance exercise + Jump training	Increase Hip and Spine BMD
Post-menopausal women	Strength training (Hip muscles, abdominal muscles, back extensors	Maintenance of lumbar spine and femoral neck BMD
Post-menopausal women	Upper and lower body resistance exercise+ jump training	Maintenance of lumbar spine and Decrease incidence of vertebral fractures
Post-menopausal women	High impact exercise + Strength training	Increase Lumbar spine and Hip BMD

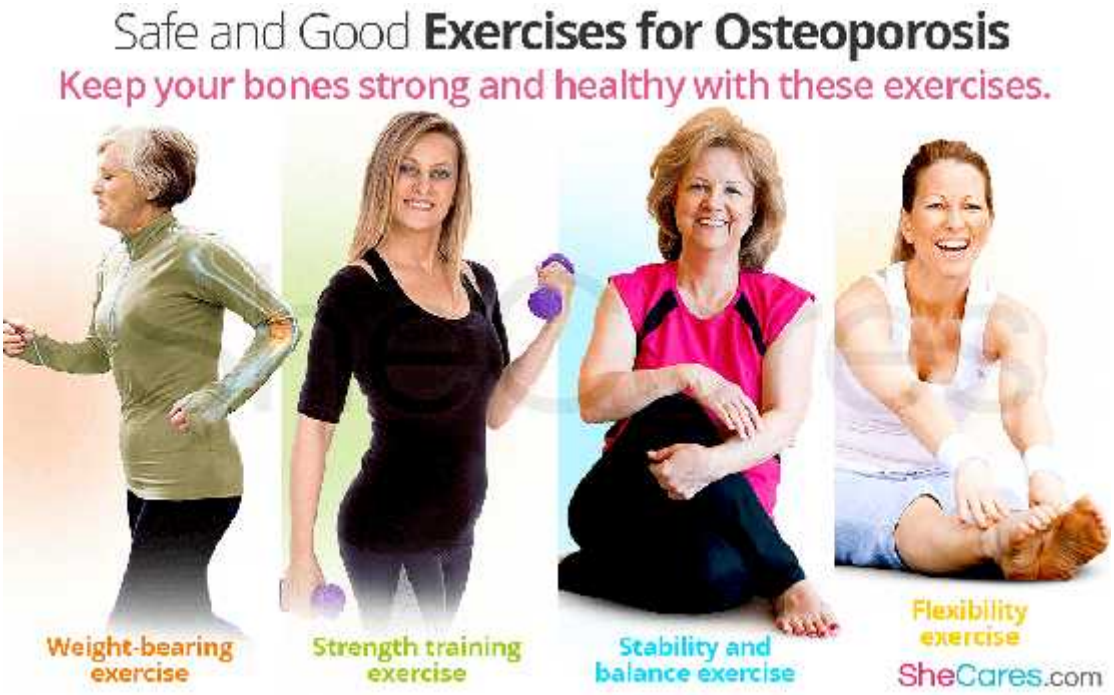


Fig 6: Exercises for osteoporosis

Thyroid Introduction

TH 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. 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. Hyper- and hypothyroidism are the most common pathologies in daily practice. 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.

Pathophysiology of thyroid effects on bone metabolism

One of the key elements for determining bony changes in any thyroid related disease stems from the remodeling cycle of the bone. This states that the bone undergoes a continuous process of formation and resorption throughout its life. It starts with osteoclasts, which are cells derived from OBs. They are connected by a dendritic network and begin bone resorption. After the OCs have finished the osteolytic process, the OBs will be observed at the site. In addition to local factors, it is also affected by systemic conditions such as the level of calcitonin, parathyroid hormone, vitamin D3, estrogen, T3, T4, glucocorticoids and growth hormones.

The hormone T3 acts on the TRa receptors in both OBs and OCs. It is believed to accelerate synthesis of OBs (bone formation) as well as osteoclasts (bone resorption), thereby increasing the turnover rate of the bone.

It still remains to be asserted whether T3 acts directly or indirectly on the osteoclast using the osteoprotegerin activator / receptor of the kappa B ligand pathway (OPG / RANKL) of the central factor (Figure 7). The pituitary gland, like T3, might also act directly on bone cells through the action of the thyroid stimulating hormone (TSH) on TSHR present in OBs and osteoclasts. / Figure 7.

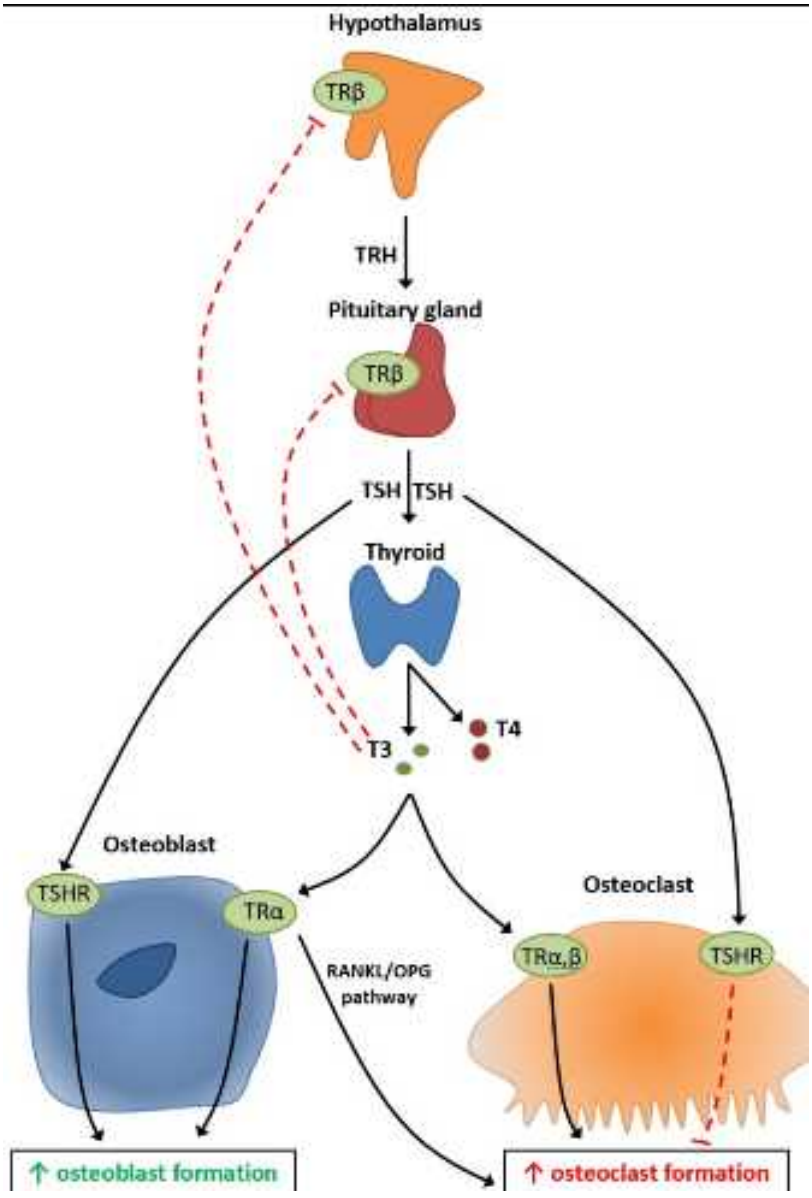


Fig 7: Physiopathology of the hypothalamic–pituitary–thyroid axis on bone metabolism.

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. 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, subclinical hypothyroidism, subclinical hyperthyroidism and their treatments in bone pathology.

Thyroid disorders can lead to osteoporosis is a presumption at present. Associated fractures involving the hip joint are a major cause of emergency room presentations worldwide not just functionally, but also financially. More importantly, hip fractures lead to decreased mobility, and decreased social independence. They also carry an increased predisposition to mortality, which is between 9.7% and 34.8% one-year post-trauma. Therefore, any attempt to reduce the incidence of hip fractures by limiting the reduction in BMD is important for patients with associated thyroid disorders.

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 paraTH in the bones. Increase in the secretion of thyroid hormones causes a negative calcium balance owing to hypercalcemia and hypercalcuria. This has a direct involvement in causing osteoporosis in such patients who are already at risk.

Tuchendler D, Bolanowski M carried out an evaluation of bone metabolism in 38 premenopausal patients aged 18 to 52 years with hyperthyroidism and compared it with the control group. The initial evaluation found a significant reduction in BMD in the lower femoral neck (as expressed by the Z-Score), in women having hyperthyroidism.[163] All these patients with hyperthyroid were managed using an oral thyrostatic agent: methimazole (dose 60 to 5 mg per day) during the 12 months of the study. At 1 year follow-up, a persistently statistically significant lower bone density of the NoF, expressed by the Z-Score, was noted in this hyperthyroid group. [163]

Ale and colleagues conducted a study in premenopausal women aged 21 to 45 with hyperthyroidism to assess the prevalence, predictive factors, and characteristics of osteoporosis and compared it with the control group. The average age of the patients was 36.16±8.43 years. A high rate of prevalence i.e 45% was found in this study.[164] This study confirmed that thyroid hormones and TSH are pivotal in the appearance of osteoporosis in hyperthyroidism.

Subclinical hyperthyroidism and Osteoporosis

Subclinical hyperthyroidism is said to manifest when there are low or undetectable levels of thyroid-stimulating hormone (TSH) in the serum, while the level of T3 and T4 are unaffected. This is associated with a rise in the level of bone turnover markers, more so in women before and after menopause. The literature suggests that subclinical hypothyroidism has some association with reduced BMD and higher fracture rate, especially in women.

J. Foldes et al. Conducted a cross-sectional study, wherein BMD was calculated at various sites using DEXA in 37 premenopausal women with subclinical hyperthyroidism. This study concluded that no significant effect was observed in premenopausal women with subclinical hyperthyroidism, but that the median and femoral wave radius was often below the mean of the reference population.[165]

Gurlek and Gedik et al. A cross-sectional study of 15 premenopausal women with subclinical hyperthyroidism was performed to assess whether bone turnover increased and BMD decreased due to endogenous subclinical hyperthyroidism.[166] This study suggests that endogenous subclinical hyperthyroidism plays no role in increased bone turnover and that BMD in premenopausal women does not decrease, at least in the short term. [166]

Tauchmanova L, et al. performed a cross-sectional evaluation of 30 premenopausal women with subclinical hyperthyroidism to assess the effect of endogenous SH on bone relative to menopausal status. They concluded that in such women, there was a marked reduction in bone mass, mainly in the cortical region, while the rate of bone turnover markers increased significantly. [167]

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. 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.

Tuchendler D et al examined the relationship of bone metabolism in 40 premenopausal patients aged 18 to 52 with hypothyroidism and compared it with the control group. At the initial examination, there was no decrease in BMD in hypothyroidism patients. [163] Women with hypothyroidism were treated with levothyroxine (doses of 25 to 150 µg per day). There was no significant difference to be found between the femoral neck and the lumbar spine BMD in the hypothyroid group, expressed by the Z-Score. However, a higher number of fractures were found in this group of patients, which is why it is still a mystery. [163]

In 2017, Supriti Bhatnaga et al. [168]A cross-sectional study of hypothyroid patients aged 20 to 60 evaluated the effects of hypothyroidism on BMD. They confirmed that these hormones have a crucial role in strengthening and remodeling of the bone. Furthermore, an increase in BMD in hypothyroidism patients leads to bone stiffness. The same study confirmed that women with subclinical hypothyroidism had reduced femoral neck (BMD) and that serum calcium levels in subclinical hypothyroidism were also lower than in the normal group. [168]

Greenspan S.L .; Greenspan F.S. et al. undertook a study of 28 premenopausal women with hypothyroidism that measured skeletal integrity treated with long-term L-thyroxine therapy.[169] 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.

In 2017, Supriti Bhatnaga et al. A cross-sectional study conducted on hypothyroid patients aged 20 to 60 to assess the relationship between hypothyroidism, BMD status, Vitamin D levels and the level of serum calcium. This study confirmed that thyroid hormones are a major determinant in strengthening and remodeling

bones, as a decrease in TH levels has a direct correlation with the level of serum calcium and vitamin D. [168]In addition, an increase in BMD in hypothyroidism patients leads to bone stiffness.

Furthermore, they state that a baseline assessment of serum calcium, vitamin D and BMD needs to be done to prevent the risk of pathological fractures in these patients.[168]

METHODOLOGY

The present study was conducted at KLES Dr.Prabhakar Kore Hospital and Medical Research Centre, Belagavi.

Study Design:

Hospital based one year Cross Sectional Study.

Study Period:

The study is being conducted from January 2019 to December 2019

Source of Data:

Data was collected from thyroid disorder patients undergoing DEXA scan, attending outpatient, department of Orthopaedics and department of Endocrinology, KLES Dr. Prabhakar Kore Hospital and Medical Research Center, Belagavi, Karnataka.

Sample Size

53 patients suffering from thyroid disorder who were willing to undergo a DEXA Scan.

Sampling procedure:

Sample size was calculated by using the formula

4pq/d2 where

p = prevalence of Osteoporosis in % (In a similar study done) & was 50%,

q = (100-p) %, and d = absolute error taken as 10%.

SELECTION CRITERIA

Inclusion criteria:

1. Women suffering from thyroid disorder both hypo and hyperthyroidism (female, old and new cases)
2. Patient age from 18 to 45 years.
3. Women who have not undergone partial or total hysterectomy or oophorectomy
4. Not associated with any life threatening diseases like MI , CVA.

Exclusion criteria:

1. Participants on following medication which are known to affect calcium metabolism
    a. Long term steroids
    b. Phenytoin
    c. Eltroxin
    d. Heparin
    e. Thiazide diuretics
    f. Oestrogen
    g. thiazolidinediones (TZDs)
2. Patients with following long term diseases
    a. Chronic liver/ kidney diseases
    b. Chronic skin disease
    c. Malignancy
    d. Rheumatoid Arthritis
3. Women who are on active treatment for fracture.

PROCEDURE:

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 (Annexure–I). The consented patients were enrolled in the present study. Further, descriptive data of the participants & risk factors were evaluated through a Questionnaire. (Annexure II).

PROFORMA:

The patients were evaluated through a proforma & after ruling out the patients in the exclusion criteria, the rest were enrolled in the study.

OPD No:

Name: To identify the patient

Age: Age is an important factor to be noted, as the study was focused on patients aged more than or equal to 18 years.

Sex: Female were included in the study

Address: Address was noted to communicate with the patient for treatment purposes if found osteoporotic & osteopenic.

Socioeconomic Status: Was classified into upper, middle and lower status based on income.

Questionnaire:

1. Occupation:

Occupation of the individual was asked and it was further classified into manual labour, sedentary work and other group. This was to assess the amount of physical activity a patient involves in as increased bone mass is seen with higher physical activity.

2. Complaints of the individual

To know the reason for attending the department of orthopaedics or department of Endrocrinology for evaluation & treatment.

3. Medication History.

To rule out all the exclusion criteria. To advice the patient to stop calcium supplementations 48 hours prior to the scan.

4. History suggestive of following chronic diseases.

a. Chronic liver disease b. Chronic kidney disease

c. Chronic skin disease d. Rheumatoid arthritis

e. Hypertension f. Malignant conditions

5. History of alcohol Consumption.

Alcohol consumption was asked as it leads to fall in bone mineral density. If the patient consumed alcohol, quantity of intake was assesed.

6. History of smoking cigarettes.

Cigarette smoking was asked as it leads to fall in bone mineral density. If the the patient smoked cigarettes, the number of cigarettes smoked per day was asked.

7. History of consumption of milk & milk products.

To assess whether the patient is on calcium rich diet as it leads to increase in BMD.

8. Diet

Patients diet was assessed whether the patient is a vegeterian or a non vegetarian.

9. Sunlight exposure

A history of exposure to sunlight (number of hours per day) was solicited. Exposure more than 1.5 hours per day to sunlight was considered adequate.

10. Family history of fractures

Family history of fractures after trivial fall or deformity of the back (Hunch Back) was asked.

11. Body mass index was calculated after determining the Height & weight

BMI: - wt(kg)/Ht(m2)_____

12. Duration since suffering from thyroid disorders.

To ascertain whether duration of thyroid disorders has any relationship with osteoporosis

Investigations:

1. Thyroid function test.

2. BMD (Bone mineral density) measurement was done using DEXA Scan of make GE Wipro and 2008 Lunar model.

DEXA Scan Evaluation technique:

A dual energy X-ray absorptiometry (DEXA) scan uses X-ray equipment and a computer to measure bone density. Bone mineral density is the most important tool in the diagnosis of osteoporosis. It allows for accurate, precise and reproducible assessment of bone mineral density and enables the detection of osteoporosis before the occurrence of fractures. DEXA Scan is the gold standard in the assessment of BMD.

Pre Scan Requisites

1. Completion of the questionnaire

2. Selection of the study group after ruling out the exclusion criteria

3. Filling of the informed consent.

Instructions prior to the scan

1. Stoppage of calcium supplements 48 hours before the scan.
2. Removal of clothes that have metal buttons or other metal accessories & change to a gown if necessary.
3. To remain still during the procedure.

Procedure:

The procedure was quick, painless and time taken was about 10 minutes. It involved exposing the body to a small dose of X-ray radiation. Patient was taken to the X-ray room and asked to lie down on an X-ray table. A radiographer operated the scanning equipment.

Scan was carried out at two sites the lumbar spine followed by the hip joint. Patient legs were flexed & placed over a large block for scanning of the lumbar spine. This was done to achieve straightening of the spine. For scanning of the hip joints patient was made to lie supine only. The scanning apparatus was then passed over the patient's lumbar spine & the hip joints respectively and it will project X rays beam. Some of this radiation travels straight through the bones and a certain amount is absorbed by them - how much depends on how dense the bones are.

A detector measured how much radiation passes through the bones and sends the information to a computer. A printed report was then obtained stating the BMD, 'T' & 'Z' scores.

Assessment of data:

The Bone Mineral Density (BMD in g/cm2) and 'T' and 'Z' scores were determined. '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 was analyzed as follows.

- Normal BMD: T scores not more than 1 SD below the adult mean.
- Osteopenia: T score between -1.0 and- 2.5.
- Osteoporosis: T score <- 2.5 with or without fragility fracture.

Data was collected and recorded and diagnosis based on the BMD score was done. BMD data was correlated with the data of various risk factors obtained through the questionnaire and correlations were derived.

RESULTS

In these series 53 patients were evaluated. Following are the observations made.

Table 1: BMD Distribution

BMD	Number	Percent
Normal	31	58.49
Osteopenia	11	20.75
Osteoporosis	11	20.75
Total	53	100.00

The overall prevalence of osteoporosis was 20.75% out of which hyperthyroid osteoporotic were 41.18% and hypothyroid osteoporotic were 11.11%, osteopenia 20.75% and thyroid patients with normal BMD were 58.49%

Graph 1: BMD wise distribution of patients

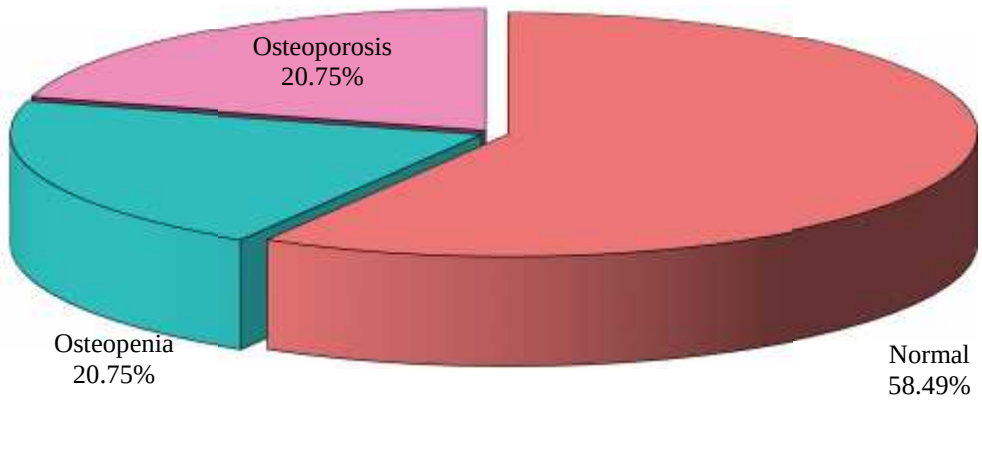


Table 2: Osteoporosis and Osteopenia at Various Anatomical Sites

BMD	Spine	%	Femur	%
Normal	31	58.49	32	60.38
Osteopenia	11	20.75	13	24.53
Osteoporosis	11	20.75	8	15.09
Total	53	100.00	53	100.00

Determination of BMD at Lumbar spine was more sensitive for osteoporosis with 11 thyroid patients having osteoporosis, where as dual hip joint DEXA evaluation showing 8 thyroid patients with osteoporosis.

Graph 2: Osteoporosis and Osteopenia at Various Anatomical Sites

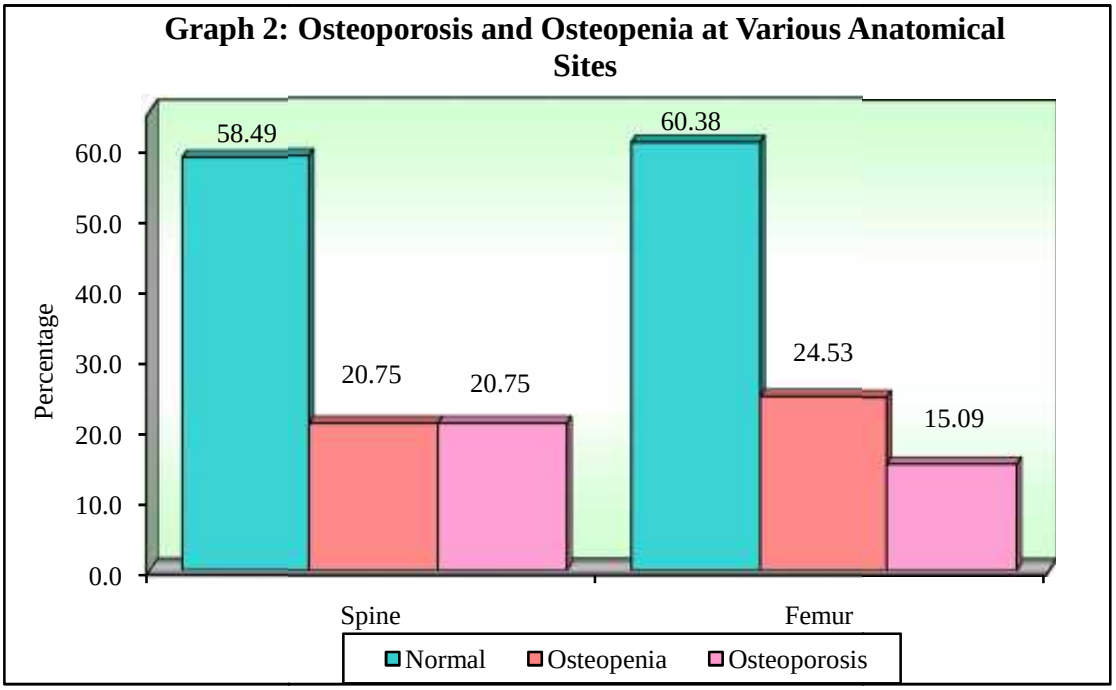


Table 3: BMD with status of Hyperthyroidism and Hypothyroidism

BMD	Hyperthyroidism	%	Hypothyroidism	%	Total	%
Normal	6	35.29	25	69.44	31	58.49
Osteopenia	4	23.53	7	19.44	11	20.75
Osteoporosis	7	41.18	4	11.11	11	20.75
Total	17	100.00	36	100.00	53	100.00
Chi-square=7.4242 P = 0.0240*						

*p<0.05

Total number of patients assessed in these study were 53 thyroid dysfunction patients. Out of which 17 were Hyperthyroidism and 36 were Hypothyroidism patients. Out of 17 hyperthyroidism patients 7 (41.2%) were osteoporotic and among 36 hypothyroidism patients 4 (11.11%) were osteoporotic. 25 (69.44%) among hypothyroidism group have normal BMD.

Graph 3: BMD with status of Hyperthyroidism and Hypothyroidism

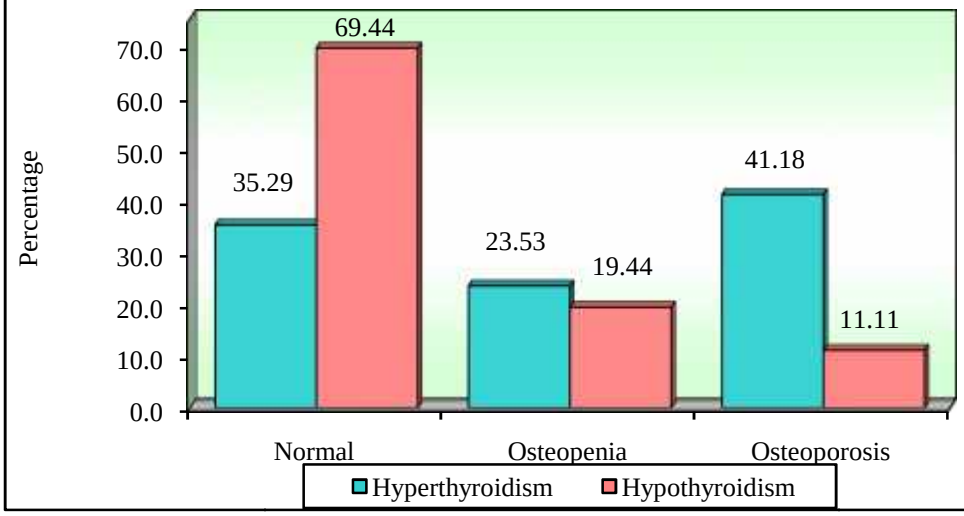


Table 4: Age groups with BMD

Age groups	Normal	%	Osteopenia	%	Osteoporosis	%	Total	%
<=25yrs	6	100.00	0	0.00	0	0.00	6	11.32
26-30yrs	18	85.71	1	4.76	2	9.52	21	39.62
31-35yrs	6	31.58	5	26.32	8	42.11	19	35.85
>=36yrs	1	14.29	5	71.43	1	14.29	7	13.21
Total	31	58.49	11	20.75	11	20.75	53	100.00
Mean age	27.87		35.00		32.55		30.32	
SD age	3.51		5.04		2.50		4.74	

Age of all the patients in this study was above 18 years. Majority of the patients were aged > 26 years. 6 patients (11.32%) were in the ge group <= 25 years. There were 21 patients (39.62%) in the age group of 26-30 years, 19 patients (35.85%) in the age group 31-35 years, 7 patients (13.21%) in the ag group >= 36 years. Highest number of osteoporotic individuals 9 were aged more than 31 years.

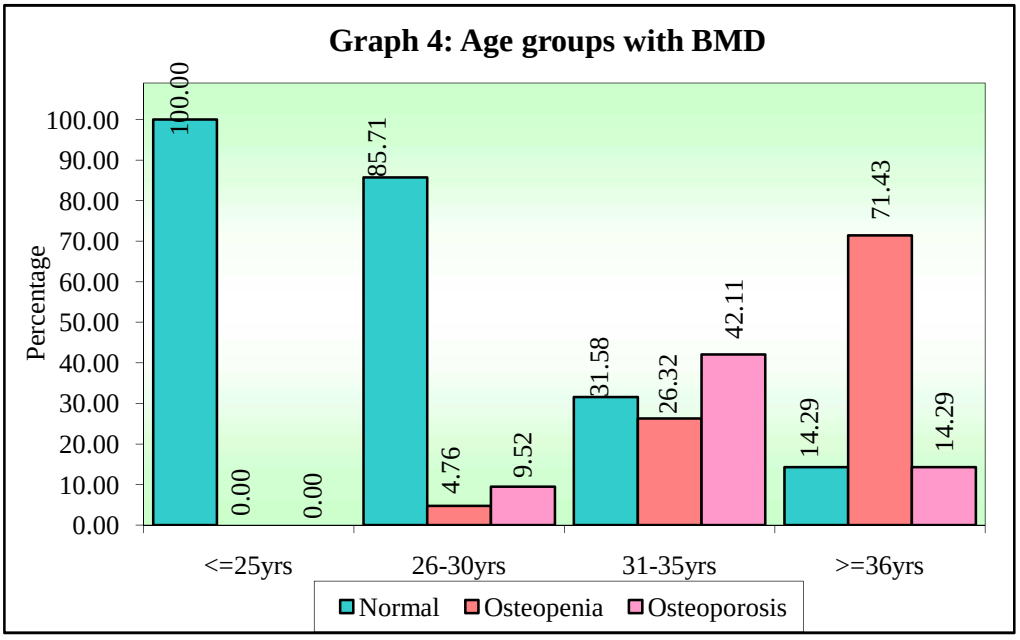


Table 5: Treatment for Hyper and Hypothyroidism with BMD

Treatment	Normal	%	Osteopenia	%	Osteoporosis	%	Total	%
No	11	68.75	5	31.25	0	0.00	16	30.19
Yes	20	54.05	6	16.22	11	29.73	37	69.81
Total	31	58.49	11	20.75	11	20.75	53	100.00
Chi-square=6.386 P = 0.0410*								

*p<0.05

Patients who are on treatment for hyperthyroidism for less than 6m- 1year had high chances of osteoporosis and patients who are on treatment for hypothyroidism for more than 2 years had high chances of osteoporosis. Patients who are on long term treatment(> 1 year) for hyperthyroidism had normal BMD values(58.49%).

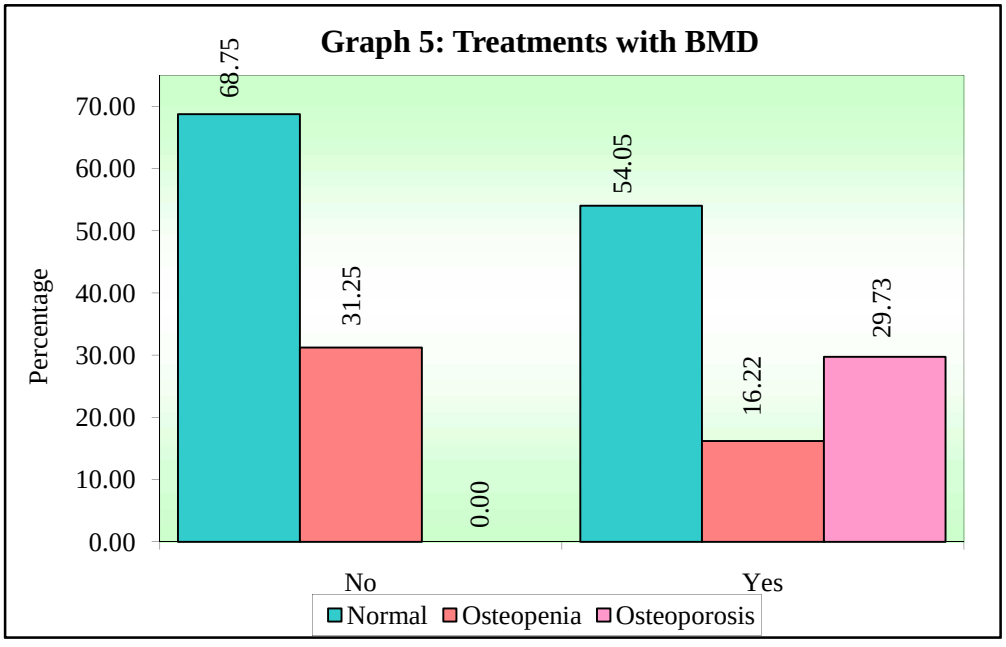


Table 6: Socio Economic status with BMD

SES	Normal	%	Osteopenia	%	Osteoporosis	%	Total	%
Lower SES	8	44.44	3	16.67	7	38.89	18	33.96
Middle SES	18	64.29	6	21.43	4	14.29	28	52.83
Upper SES	5	71.43	2	28.57	0	0.00	7	13.21
Total	31	58.49	11	20.75	11	20.75	53	100.00
Chi-square=6.1948, p=0.1850								

The percentage of osteoporotic thyroid patients in the lower socio economic group was the highest 7 (38.89%). Percentage of thyroid patients having normal BMD was highest in the upper socio economic group (71.43%)

Graph 6: Socio Economic status with BMD

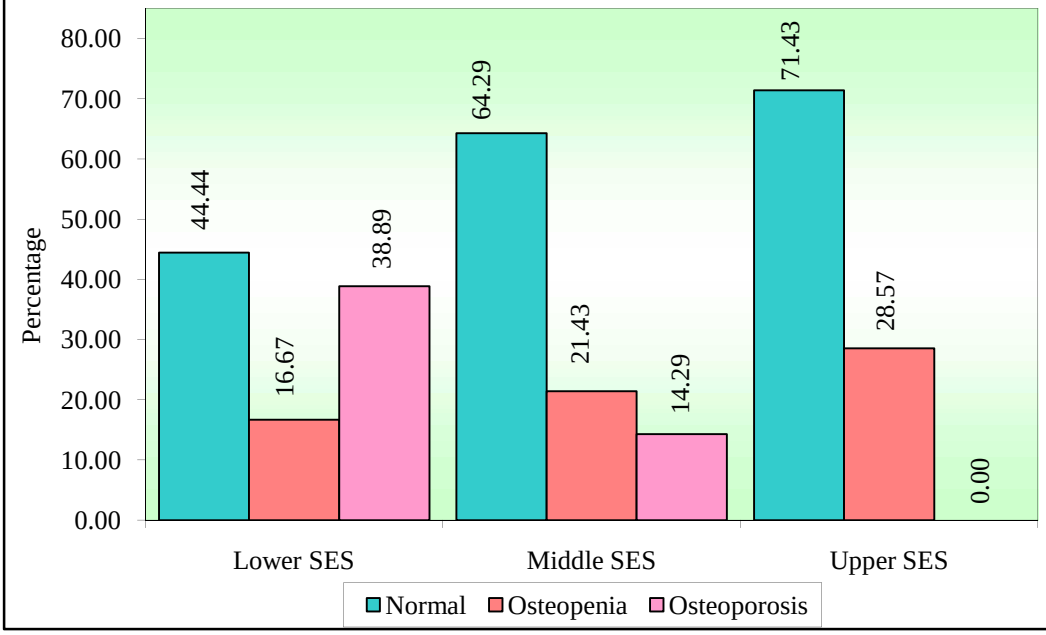


Table 7: BMI with BMD

BMI	Normal	%	Osteopenia	%	Osteoporosis	%	Total	%
Under weight	0	0.00	0	0.00	1	100.00	1	1.89
Normal	3	23.08	5	38.46	5	38.46	13	24.53
Over weight	16	64.00	4	16.00	5	20.00	25	47.17
Obese	12	85.71	2	14.29	0	0.00	14	26.42
Total	31	58.49	11	20.75	11	20.75	53	100.00
Chi-square=10.9422, p=0.0902								

Body Mass Index ranged from 18.2 to 34.7. Thyroid patients with higher body mass index had lower incidence of osteoporosis. Highest number of thyroid patients with osteoporosis had normal BMI values.

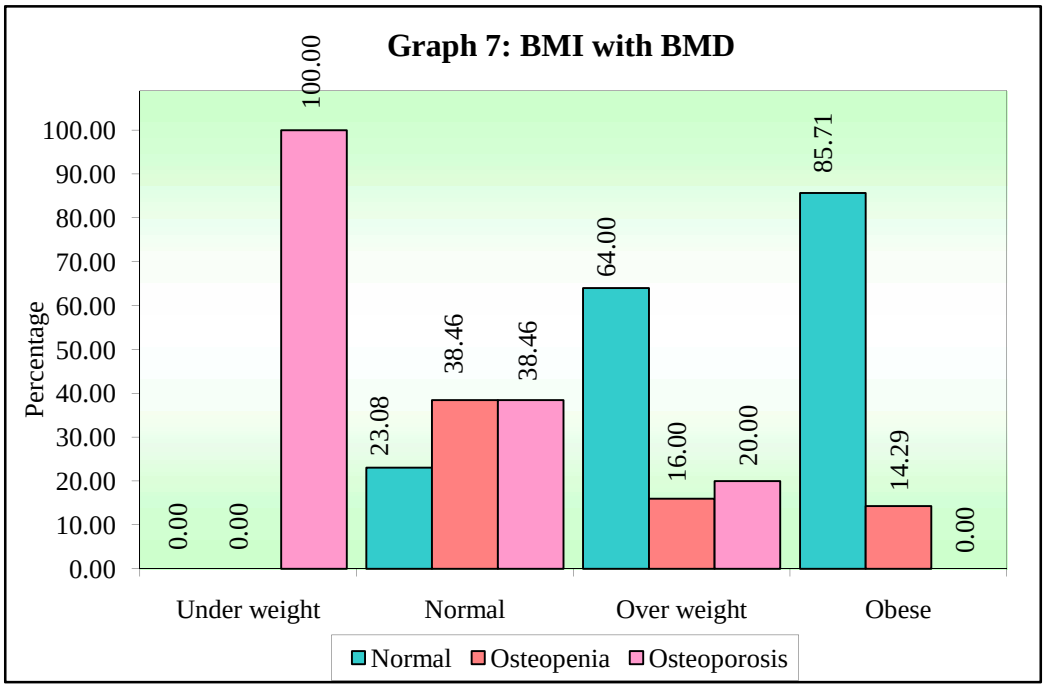


Table 8: Diet with BMD

Diet	Normal	%	Osteopenia	%	Osteoporosis	%	Total	%
Mixed	22	70.97	4	12.90	5	16.13	31	20.26
Vegetarian	9	40.91	7	31.82	6	27.27	22	14.38
Total	31	58.49	11	20.75	11	20.75	53	34.64
Chi-square=4.9760 P = 0.0830								

Highest percentage of patients with osteoporosis was in the vegetarian group (27.27%)

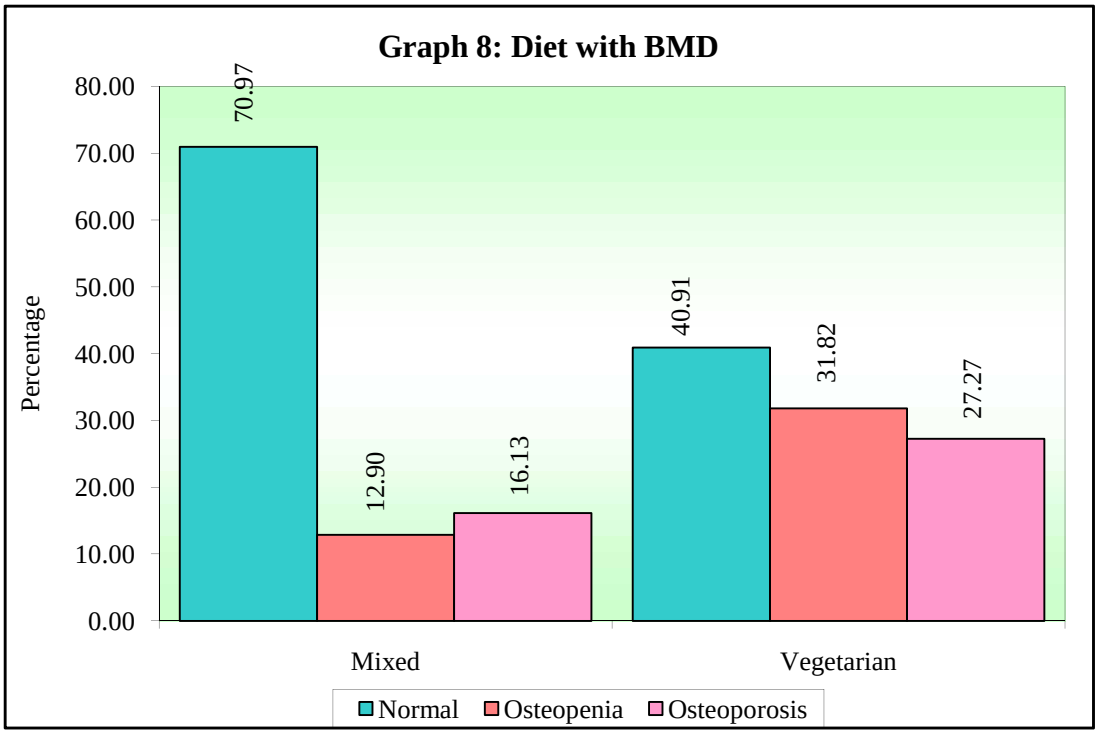


Table 9: Alcohol habit with BMD

Alcohol habit	Normal	%	Osteopenia	%	Osteoporosis	%	Total	%
No	23	60.53	6	15.79	9	23.68	38	24.84
Yes	8	53.33	5	33.33	2	13.33	15	9.80
Total	31	58.49	11	20.75	11	20.75	53	34.64
Chi-square=2.2452 P = 0.3251								

Out of the 53 thyroid patients scanned 15 (28.3% females had a history of alcohol consumption and 38 (71.69) did not have a history of alcohol consumption. 2(13.33%) of patients with positive history of alcohol intake were found to be osteoporotic.

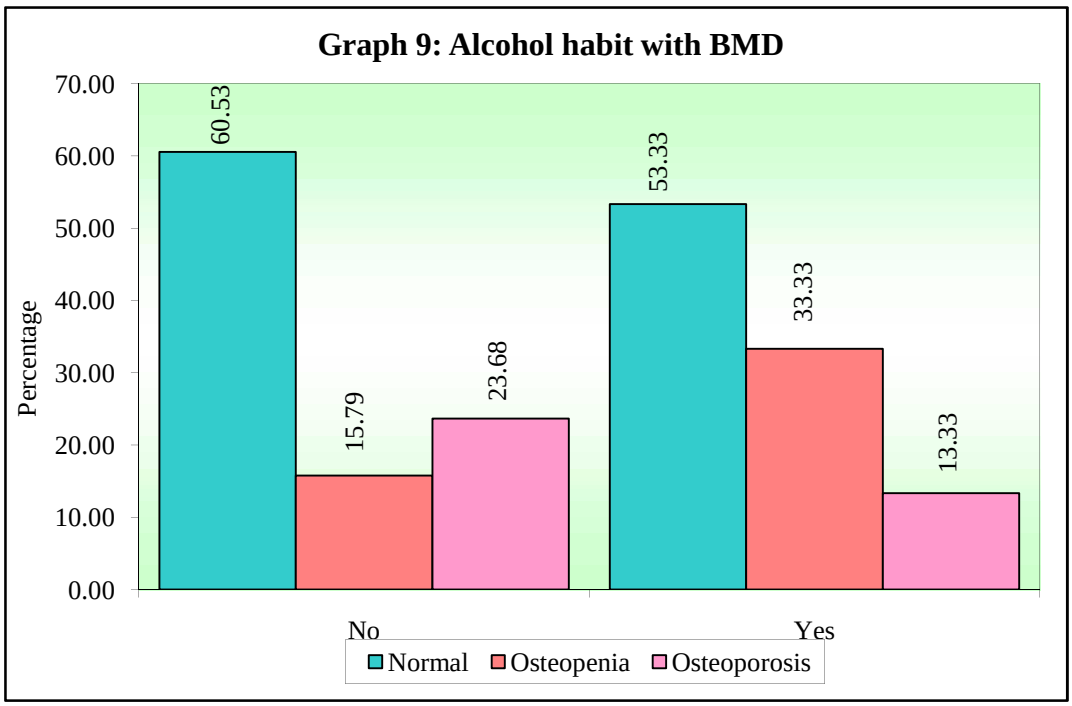


Table 10: Smoking habit with BMD

Smoking habit	Normal	%	Osteopenia	%	Osteoporosis	%	Total	%
No	31	58.49	11	20.75	11	20.75	53	34.64
Yes	0	0.00	0	0.00	0	0.00	0	0.00
Total	31	58.49	11	20.75	11	20.75	53	34.64
Chi-square=0.0000, p=1.0000								

Among the 53 thyroid patients none of them were smokers. In non smokers category,11 (20.75%) were osteoporotic.

Table 11: Sunlight exposure with BMD

Sunlight exposure	Normal	%	Osteopenia	%	Osteoporosis	%	Total	%
Adequate	22	70.96	6	19.35	3	11.11	31	58.49
Inadequate	9	29.03	5	16.12	8	30.77	22	41.50
Total	31	58.49	11	20.75	11	20.75	53	100.00
Chi-square=5.6871 P = 0.03								

The number of thyroid patients with adequate amount of exposure of sunlight (11/2- 2 hrs) were 31 and with inadequate exposure of sunlight (< 11/2 hrs) was 22. The prevalence of osteoporosis was higher among the group with inadequate exposure to sunlight. The number of thyroid patients with normal BMD was highest among the group with adequate exposure to sunlight.

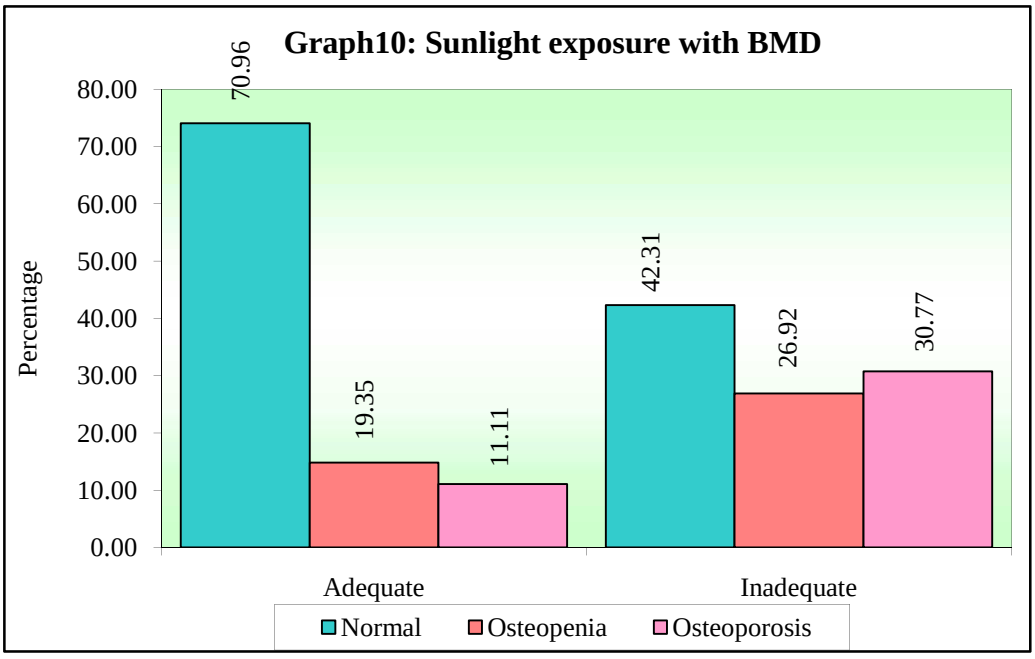


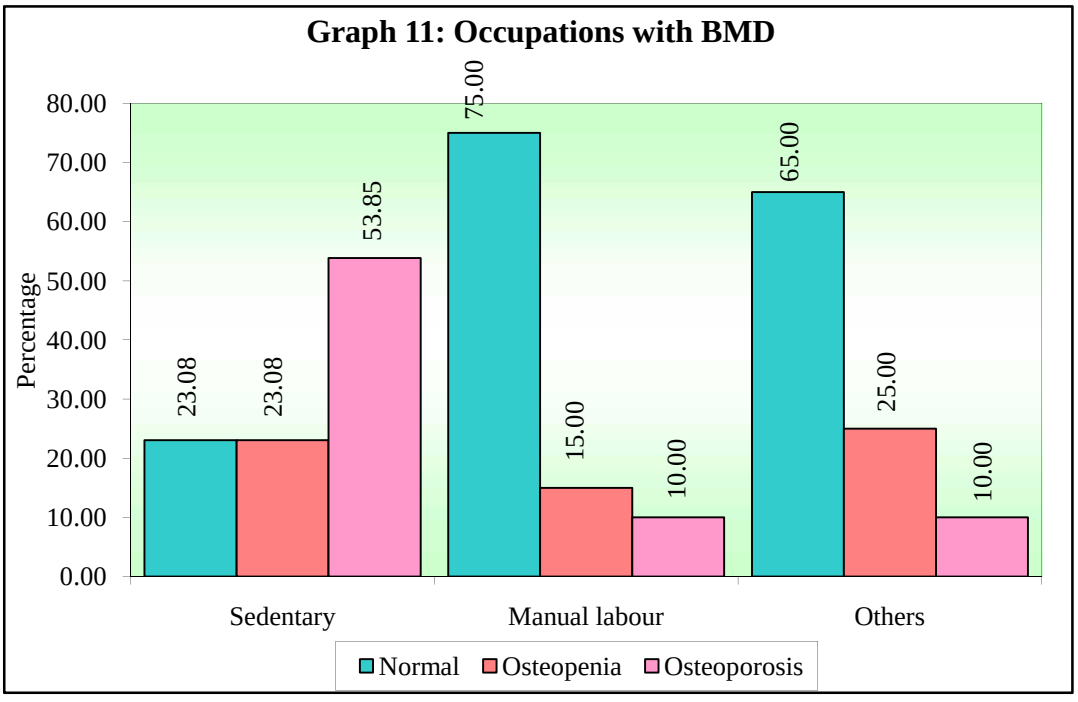
Table 12: Occupation with BMD

Occupations	Normal	%	Osteopenia	%	Osteoporosis	%	Total	%
Sedentary	3	23.08	3	23.08	7	53.85	13	24.53
Manual labour	15	75.00	3	15.00	2	10.00	20	37.74
Others	13	65.00	5	25.00	2	10.00	20	37.74
Total	31	58.49	11	20.75	11	20.75	53	100.00
Chi-square=13.4790, P = 0.0091*								

*p<0.05

The prevalence of osteoporosis was highest among those involved in sedentary work (house wives, office work) where in 7 thyroid patients (53.85%) had osteoporosis. 75% of patients among manual labour and other group had normal BMD. Thyroid patients who had other occupation had 20% osteoporosis prevalence.

Graph 11: Occupations with BMD



DISCUSSION

Thyroid disorders is 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 53 patients with thyroid dysfunction were studied using DEXA scan. The existence of osteoporosis in every patient was checked and matched against any TH pathology. Additionally, all, if any, risk factors too were evaluated. 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 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%.

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.

Tuchendler D, Bolanowski M carried out an evaluation of bone metabolism in premenopausal women with hyperthyroidism and compared it with the control group.

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. Many other publications have stated a decreased BMD and increased prevalence of osteoporosis in patients with hyperthyroidism. [163]

J. Foldes et al. Conducted a cross-sectional a study, wherein the BMD was calculated at various sites using DEXA in premenopausal women with subclinical hyperthyroidism. This study concluded that no significant effect was observed in premenopausal women with subclinical hyperthyroidism, but that the median and femoral wave radius was often below the mean of the reference population. Many other studies also concluded same in subclinical hyperthyroidism. [165]

The prevalence of hypothyroidism in India is 10-11%. The state of inadequate formation of T3 and T4 is termed hypothyroidism. Thyroid hormones 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.

Tuchendler D et al examined the relationship of bone metabolism in premenopausal with hypothyroidism and compared it with the control group. 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. However, a higher number of fractures were found in this group of patients, which is why it is still a mystery.[163]

Greenspan S.L .; Greenspan F.S. et al. undertook a study of 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.[169]

Chen J-H, et al., conducted a cross sectional study included 1350 (1091 premenopausal and 259 postmenopausal) women. Among the women, 108 (8% had osteopenia and 1242 (92%) had normal spinal BMD.[171] My study correlates with literature.

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 11 (20.75% in that Hyperthyroid osteoporotic were 41.18% and Hypothyroid osteoporotic were 11.11%) were greater than 30 years of age.

In comparative studies they concluded that age correlated inversely with BMD values and fracture threshold reduces with age. Anburanjan et al prospectively studied the rate of loss of BMD per annum. They concluded that rates of BMD loss were 0.91%, 0.84%, 0.72%, 0.78%, 1.66%. per annum respectively for the neck of femur, trochanter, intertrochanteric region, total hip and Ward's triangle.

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.

In our study majority of the osteoporotic patients among thyroid disorders gave a history of sedentary work (housewives, office work, and retired men) where in 7 patients (53.85%) had osteoporosis. 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. Thus lower rate of osteoporosis prevalence was seen among thyroid involved in laborious professions in our study.

There have been many cohort research publications that have stated the role of sedentary lifestyle in populations as being a major determinant for the decline of bone mass and consequently osteoporotic fractures.

Osteoporosis itself has no specific symptoms or signs; its primary debilitating complication is the increased risk of fractures. Osteoporotic fractures are those that "occur in situations where healthy people would not normally break a bone"; and hence have been named as fragility fractures. The commonest site of occurrence of such fractures is the vertebral column, rib, hip and wrist. The most common complaints reported in our study was backache .Rest had complaints of sciatica, knee joint pain, generalized weakness & diffuse body ache.

The number of osteoporotic thyroid patients in the lower strata of socio economic group was the highest 7 (38.89%). This was an essential finding as it was congruous with a separate study which was done to assess status of Indian women that hailed from a similarly lower socioeconomic status. The 289 women in the 30-60 year age group assessed using DEXA Scan concluded that the presence of osteoporosis at

the level of femoral neck was seen in 29% of the study patients.

Kadam et al showed that this density measured at all the three sites namely Spine, Hip & wrist was remarkably less in post menopausal than in pre menopausal women ($p< 0.001$) . It was at an epoch in the lumbar spine (25.8%) in pre menopausal women, while osteopenia was high in pre-menopausal women (44.3%).[170] Unni et al in their study found that increasing age was directly proportional to the presence of osteoporosis.

Many risk factors have been implicated in the causation of this condition. These are comprised of non-modifiable factors such as female gender, elderly age, small thin built. Caucasian / Asians and a significant family history of unusually higher incidence of fractures. Ethnic differences in BMD are strongly affected by body weight. Behavioral risk factors include functional impairment, physical inactivity, sedentary work, low calcium intake, high caffeine intake, smoking, and high alcohol intake.

Body Mass Index of the patients in our study ranged from 18.2 to 34.7. Thyroid patients with higher body mass index (BMI) had lower incidence of osteoporosis.

Literature shows low BMI, a measure of body composition, may be associated more number of osteoporotic fractures, and osteoporosis. Epidemiological studies show low body weight is one of the main determinant and risk factor for hip fractures.

It was found that highest percentage of thyroid patients with osteoporosis were in the vegetarian group (27.2%). Various Indian studies say that vegetarians have 50% risk of osteoporosis and 98.82% are osteopenic.

Important modifiable risk factors include calcium and vitamin D deficiency, sedentary life style, smoking, unusually high levels of alcohol and caffeine intake.

Only 15 thyroid patients in this study gave history of drinking alcohol of which 2 were osteoporotic. No thyroid patient gave history of smoking in our study.

Prospective and cohort studies show smoking decreases the BMD leading to an increased risk of osteoporosis and related insufficiency fractures in both the genders. Some have also reported demonstrating that quitting smoking may help to reduce fractures

Osteoporosis was higher among the group with inadequate exposure to sunlight (30.77%). The number of xzthyroid patients with normal BMD was highest among the group with adequate exposure to sunlight. Certain multi-centric (remove) studies conclude that there is a strong association between less exposure to sun light leading to the hip fractures in the age groups of above 50 years.

Genetic factors have a role on BMD. Fifty percent of the epoch bone mass, geometry, strength, bone architecture depends on genetic predisposition.

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.

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. Regular exercise, adequate dietary calcium along with supplemental (1000 mg/day), adequate vitamin D (600 IU/day) and cessation of smoking and alcohol intake.

Osteoporosis (and osteoporotic fractures) have a multi-factorial genesis; as a result of which their management is complex. The primary goals of treatment of established disease are arresting the declining BMD, maintaining skeletal integrity and prevent fragility fracture. All this necessitate early intervention.

CONCLUSION

In this cross sectional study 53 patients with thyroid disorders were evaluated with a DEXA scan for the presence of osteoporosis and the association of various risk factors. All the participants were women in this study.

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 %.

Age of the participants in our study ranged from 18 to 45 years. Average age was 33 years. A major number of osteoporotic thyroid patients 11 (20.75% in that Hyperthyroid osteoporotic were 41.18% and Hypothyroid osteoporotic were 11.11%) were greater than 30 years of age.

In our study the prevalence of osteoporosis was highest among thyroid patients involved in sedentary work (housewives, office work, and retired women) where in 7 patients (53.85%) had osteoporosis.

The most common complaints the patients presented with was backache (56%). Rest had complaints of knee pain, sciatica, generalized weakness & diffuse body ache.

Body Mass Index of the patients in our study ranged from 18.2 to 34.7. Thyroid patients with higher BMI had lower incidence of osteoporosis.

It was found that highest percentage of thyroid patients with osteoporosis were in the vegetarian group (27.2%).

The number of thyroid patients with adequate amount of exposure of sunlight (11/2- 2 hrs) were 31 and with inadequate exposure of sunlight (< 11/2 hrs) was 22. Inadequate exposure to sunlight was also found to be a major contributing factor to osteoporosis . The number of thyroid patients with normal BMD was highest among the group with adequate exposure to sunlight.

Out of the 53 thyroid patients scanned 15 (28.3% females had a history of alcohol consumption and 38 (71.70) did not have a history of alcohol consumption. 2(13.33%) patients with positive history of alcohol intake were found to be osteoporotic.

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 geriatric population, 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.

SUMMARY

In this study 53 patients suffering from thyroid disorders were evaluated to determine the prevalence of osteoporosis using a DEXA scan. All of them were women's in this study.

A structured questionnaire was used to assess the risk factors leading to osteoporosis. 11 patients were found to be osteoporotic, 11 were osteopenic and 31 patients had normal BMD values.

Majority osteoporotic patients were above 30 years of age. Thus increasing age was significant risk factor for osteoporosis.

Other significant risk factors were increase in time since sedentary work, lower socio-economic group, inadequate exposure to sunlight and duration of treatment.

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ANNEXURE I – CONSENT FORM

"CROSS-SECTIONAL STUDY OF OSTEOPOROSIS AMONG WOMEN WITH THYRIOD DYSFUNCTION"

Mrs/Ms_____

You are invited to participate in this study.

Principal Investigator: _____

OBJECTIVES AND PURPOSE OF THE STUDY:

1. To know the prevalence of osteoporosis in thyroid disorder patients using dual energy x ray absorptiometry scan (DEXA).
2. To assess the risk factors associated with osteoporosis.

You are invited to participate in this research as you are a patient suffering from thyroid disorder. The study is being done to find out the prevalence rate and factors affecting osteoporosis as mentioned in the objectives.

PROCEDURE

If you consent to be in this study, the relevant data is collected as per the proforma provided to you. BMD (Bone mineral density) measurement would be done using Central DEXA Scan (Dual energy X ray absorptiometry) of make GE Wipro and 2008 Lunar model. You will undergo a DEXA Scan and after ruling out all the exclusion criteria. This test is painless and can be performed within 5 to 15 minutes. You will be asked to undergo this procedure only once.

BENEFITS

To the patient in the study.

1. It will act as a diagnostic tool for the patients in the study by providing information regarding the presence of the disease.

2. Will help to initiate therapy for osteoporosis once the diagnosis is confirmed.

3. As a prognostic tool it will help to determine future probability of osteoporosis.

To the community at large.

1. The data obtained from the study will help to provide information on the epidemiology of the thyroid disorders which will be then basis for initiation for various programs for osteoporosis prevention.

2. It will help create awareness regarding osteoporosis.

RISKS

There are no risks associated with this study.

ALTERNATIVES

If you decline to participate decision it will not change the present or future health care or other services that you will receive. The treatment given out to you will be the standard treatment for your condition.

WITHDRAWING / REMOVAL FROM THE STUDY:

You can withdraw from the study during anytime you want and you will not be penalized for the same. You can be removed from the study if you do not fulfil the inclusion criteria.

PRIVACY AND CONFIDENTIALITY:

All information about the subject during the course of the study will be kept confidential to the extent permitted by law. The code numbers will identify the subject in this research record. Information from this study may be published but the subject's identity will be confidential in any publication.

COSTS

Cost of each DEXA Scan will cost around Rs 1200/- and Thyroid function test will be Rs 800/-. There will be no reimbursement for your expenses.

QUESTION

If any enquiries in the future or in case of study related problems you may contact

If you still have any queries please contact:

Dr. Roopa M Bellad
Professor,
Department of Peadiatrics,
Chairperson,
Institutional Ethics Committee for Human Subjects Research,
KAHER, J.N. Medical College,
Belagavi -590010

STATEMENT OF CONSENT:

The details of the research study in which I am expected to participate, for which I have to undergo DEXA Scan and Thyroid function test have been explained to me. I willingly, under no pressure from the researcher agree to take part in this study, and agree to participate in all investigations. I may withdraw at any time. I am not giving up any of my legal rights by signing this form.

My signature below indicates that I have read this entire consent form or it has been read to me, and had all my questions answered. I will be given a copy of this consent form.

Signature of the participant or legally authorized representative

Participants Name : Signature :

Name of the legally : Signature :

authorized representative

Witness's name : Signature :

Investigators Name : Signature :

Date:

Place:

ANNEXURE-II

PROFORMA

STUDY TITLE: "CROSS SECTIONAL STUDY OF OSTEOPOROSIS AMONG WOMEN WITH THYROID DYSFUNCTION"

I.P/ OPD NO:

Name: Age:

Address: Phone no:

1. What kind of work do you do on a daily basis? Name of the activity ☐

a. Household (retired) b. Manual labour c. Office work

d. Others specify_____

2. Do you have any of the following complaints? ☐

a. Backache b. Easy fatigability c. Fractures after trivial fall.

d. Others specify._____

3. Do you use any medications? ☐

a. Yes b. No

If yes then specify _____

4. How long have you been suffering from thyroid ? ☐

a. 0-6 months b. 6-1 years

c. 1-2 years d. 2-4 years

e. More than 4 years

5. What are your current thyroid function test levels T_3, T_4, TSH ☐

_____

6. Have you been diagnosed with any of the following conditions? ☐

a. Chronic liver disease b. Chronic kidney disease

c. Chronic skin disease d. Rheumatoid arthritis

e. Hypertensionf. Malignant conditions

g. Endocrine disorder h. Epilepsy

7. Whether you consume alcohol? ☐

a. Yes b. No

If yes then how much quantity per day? _____

8. Do you smoke cigarettes? ☐

a. Yes b. No

If yes then how many per day? _____

9. Do you consume milk and milk products daily? ☐

a. Yes b. No

If yes then quantity per day._____

10. Do you take any calcium / Vitamin D supplements? ☐

a. Yes b. No

If yes then dosage per day._____

11. Are you a vegetarian or non vegetarian? ☐

a. Vegetarian b. Non Vegetarian

12. Have either of your parents been diagnosed with osteoporosis or broken a bone after a minor fall (a fall from standing height or less)? ☐

a. Yes b. No

13.Do you have very little physical activity

a. Yes b. No

14. Determination of Body Mass Index

Height: - _____

Weight: - _____Kg.

BMI: - wt (kg)/Ht (m^2)_____.

ANNEXURE-III-ETHICAL CLEARANCE LETTER

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/41 **Date: 24/11/2018**

To,

REGISTRATION NO. BL0118002

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"CROSS SECTIONAL STUDY OF OSTEOPOROSIS AMONG WOMEN WITH THYRIOD DYSFUNCTION",** is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Arathi Darshan)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

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ANNEXURE IV – PHOTOGRAPHS

PHOTOGRAPH 1: DEXA EVALUATION APPARATUS

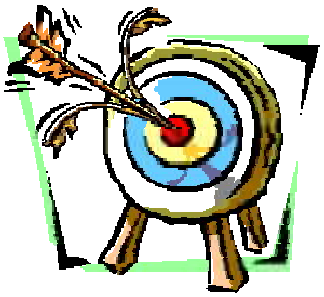
PHOTOGRAPH 2: DEXA SCAN OF LUMBAR SPINE

ANNEXURES V - MASTER CHART

S.NO.	PT No	AGE	SEX	NO OF YEARS SINCE THYRIOD	INFERENCE	TREATMENT	SOCIOECONOMIC STATUS	BMI	BMD	T SCORE	SITE	INFERENCE	BMD	T SCORE	SITE	INFERENCE	DIET	ALCOHOL	SMOKING
1	6787	24	FEMALE	NEWLY DIAGNOSED	HYPOTHYROIDISM	NO	MIDDLE	30.1	1.58	3.4	SPINE	NORMAL	1.501	4.2	FEMUR	NORMAL	VEG	NEGATIVE	NEGATIVE
2	6898	28	FEMALE	2 MONTHS	HYPOTHYROIDISM	YES	MIDDLE	30.5	1.673	4.1	SPINE	NORMAL	1.175	1.5	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE
3	6932	26	FEMALE	4 MONTHS	HYPOTHYROIDISM	YES	LOWER	34.7	1.382	1.7	SPINE	NORMAL	1.239	2	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE
4	6944	26	FEMALE	3 MONTHS	**HYPERTHYROIDISM**	YES	MIDDLE	26	0.9	-2.3	SPINE	OSTEOPENIA	0.972	-0.2	FEMUR	NORMAL	MIXED	POSITIVE	NEGATIVE
5	7129	28	FEMALE	2 YEARS	HYPOTHYROIDISM	YES	LOWER	21.6	0.825	-3	SPINE	OSTEOPOROSIS	0.701	-2.5	FEMUR	OSTEOPOROSIS	VEG	POSITIVE	NEGATIVE
6	7183	23	FEMALE	NEWLY DIAGNOSED	HYPOTHYROIDISM	NO	MIDDLE	26	1.12	-0.5	SPINE	NORMAL	1.071	0.6	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE
7	7261	26	FEMALE	NEWLY DIAGNOSED	HYPOTHYROIDISM	NO	MIDDLE	26.1	1.174	0	SPINE	NORMAL	1.046	0.4	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE
8	7258	32	FEMALE	6 MONTHS	HYPOTHYROIDISM	YES	UPPER	28.6	0.823	-1.5	SPINE	OSTEOPENIA	0.509	-1.8	FEMUR	OSTEOPENIA	VEG	NEGATIVE	NEGATIVE
9	7241	36	FEMALE	4 MONTHS	HYPOTHYROIDISM	YES	MIDDLE	21.9	0.889	-2.4	SPINE	OSTEOPENIA	0.748	-2.1	FEMUR	OSTEOPENIA	VEG	POSITIVE	NEGATIVE
10	7360	35	FEMALE	6 MONTHS	**HYPERTHYROIDISM**	YES	MIDDLE	29	0.85	-2.8	SPINE	OSTEOPOROSIS	0.85	-2.8	FEMUR	OSTEOPOROSIS	MIXED	NEGATIVE	NEGATIVE
11	7283	28	FEMALE	NEWLY DIAGNOSED	HYPOTHYROIDISM	NO	MIDDLE	28.9	1.153	-0.2	SPINE	NORMAL	1.042	0.4	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE
12	7595	32	FEMALE	6 MONTHS	HYPOTHYROIDISM	YES	LOWER	26.8	1.099	-0.7	SPINE	NORMAL	0.908	0.8	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE
13	7587	27	FEMALE	4 MONTHS	HYPOTHYROIDISM	YES	MIDDLE	35.6	1.135	-0.4	SPINE	NORMAL	1.047	0.4	FEMUR	NORMAL	VEG	POSITIVE	NEGATIVE
14	7580	28	FEMALE	5 MONTHS	**HYPERTHYROIDISM**	YES	UPPER	24.2	1.264	0.9	SPINE	NORMAL	1.157	1.3	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE
15	7583	42	FEMALE	NEWLY DIAGNOSED	HYPOTHYROIDISM	NO	LOWER	25.5	0.988	-1.6	SPINE	OSTEOPENIA	0.873	-1.1	FEMUR	OSTEOPENIA	VEG	NEGATIVE	NEGATIVE
16	7552	34	FEMALE	7 MONTHS	**HYPERTHYROIDISM**	YES	LOWER	25.6	0.864	-2.6	SPINE	OSTEOPOROSIS	0.672	-2.7	FEMUR	OSTEOPOROSIS	VEG	POSITIVE	NEGATIVE
17	7534	29	FEMALE	2 MONTHS	**HYPERTHYROIDISM**	YES	LOWER	25.5	0.785	-3.3	SPINE	OSTEOPOROSIS	0.72	-2.3	FEMUR	OSTEOPENIA	MIXED	NEGATIVE	NEGATIVE
18	7649	44	FEMALE	8 MONTHS	HYPOTHYROIDISM	YES	UPPER	27.4	0.997	-1.5	SPINE	OSTEOPENIA	0.865	-1.1	FEMUR	OSTEOPENIA	MIXED	NEGATIVE	NEGATIVE
19	7646	38	FEMALE	3 MONTHS	HYPOTHYROIDISM	YES	MIDDLE	22.6	0.941	-2	SPINE	OSTEOPENIA	0.747	-2.1	FEMUR	OSTEOPENIA	VEG	POSITIVE	NEGATIVE
20	7708	33	FEMALE	NEWLY DIAGNOSED	HYPOTHYROIDISM	NO	UPPER	28.2	1.223	0.4	SPINE	NORMAL	1.059	0.5	FEMUR	NORMAL	VEG	NEGATIVE	NEGATIVE
21	7706	34	FEMALE	3 YEARS	HYPOTHYROIDISM	YES	MIDDLE	28.6	0.815	-2.8	SPINE	OSTEOPOROSIS	0.873	-1.1	FEMUR	OSTEOPENIA	MIXED	NEGATIVE	NEGATIVE
22	7700	31	FEMALE	9 MONTHS	**HYPERTHYROIDISM**	YES	LOWER	22.8	0.723	-3.8	SPINE	OSTEOPOROSIS	0.7	-2.5	FEMUR	OSTEOPOROSIS	MIXED	NEGATIVE	NEGATIVE
23	7693	28	FEMALE	NEWLY DIAGNOSED	HYPOTHYROIDISM	NO	UPPER	32.9	1.163	-0.1	SPINE	NORMAL	0.931	-0.6	FEMUR	NORMAL	MIXED	POSITIVE	NEGATIVE
24	7682	33	FEMALE	4 MONTHS	**HYPERTHYROIDISM**	YES	LOWER	18.2	0.597	-4.9	SPINE	OSTEOPOROSIS	0.666	-2.8	FEMUR	OSTEOPOROSIS	MIXED	NEGATIVE	NEGATIVE
25	7160	27	FEMALE	4 MONTHS	HYPOTHYROIDISM	YES	MIDDLE	23.8	1.519	2.8	SPINE	NORMAL	1.218	1.8	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE
26	7709	32	FEMALE	NEWLY DIAGNOSED	**HYPERTHYROIDISM**	NO	MIDDLE	33.8	0.896	-2.3	SPINE	OSTEOPENIA	0.78	-1.8	FEMUR	OSTEOPENIA	VEG	NEGATIVE	NEGATIVE
27	7759	23	FEMALE	NEWLY DIAGNOSED	HYPOTHYROIDISM	NO	MIDDLE	30.3	1.072	-0.9	SPINE	NORMAL	1.083	0.7	FEMUR	NORMAL	MIXED	POSITIVE	NEGATIVE
28	7699	33	FEMALE	7 MONTHS	HYPOTHYROIDISM	YES	UPPER	29.4	1.244	0.5	SPINE	NORMAL	1.16	1.3	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE
29	7724	33	FEMALE	4 MONTHS	**HYPERTHYROIDISM**	YES	LOWER	21.6	0.88	-2.7	SPINE	OSTEOPOROSIS	0.846	1.3	FEMUR	OSTEOPENIA	VEG	NEGATIVE	NEGATIVE
30	7738	36	FEMALE	3 MONTHS	**HYPERTHYROIDISM**	YES	MIDDLE	24.7	0.621	-2.9	SPINE	OSTEOPOROSIS	0.616	-2.5	FEMUR	OSTEOPOROSIS	VEG	NEGATIVE	NEGATIVE
31	7720	24	FEMALE	NEWLY DIAGNOSED	HYPOTHYROIDISM	NO	LOWER	29.4	1.244	0.5	SPINE	NORMAL	1.16	0.9	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE

32	7431	29	FEMALE	7 MONTHS	**HYPERTHYROIDISM**	YES	LOWER	27.3	1.163	-0.7	SPINE	NORMAL	0.931	-0.6	FEMUR	NORMAL	MIXED	POSITIVE	NEGATIVE
33	7710	27	FEMALE	6 MONTHS	HYPOTHYROIDISM	YES	MIDDLE	27.4	1.073	-0.9	SPINE	NORMAL	0.869	-1.1	FEMUR	NORMAL	VEG	NEGATIVE	NEGATIVE
34	7292	31	FEMALE	3 YEARS	HYPOTHYROIDISM	YES	LOWER	25.6	0.864	-2.6	SPINE	OSTEOPOROSIS	0.672	-2.8	FEMUR	OSTEOPOROSIS	VEG	NEGATIVE	NEGATIVE
35	7357	36	FEMALE	NEWLY DIAGNOSED	HYPOTHYROIDISM	NO	LOWER	24.7	0.925	-2.1	SPINE	OSTEOPENIA	0.797	-1.7	FEMUR	OSTEOPENIA	VEG	POSITIVE	NEGATIVE
36	7399	24	FEMALE	3 MONTHS	HYPOTHYROIDISM	YES	UPPER	26.6	1.105	-0.6	SPINE	NORMAL	1.089	0.8	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE
37	7346	26	FEMALE	2 MONTHS	**HYPERTHYROIDISM**	YES	MIDDLE	30.7	0.99	-0.6	SPINE	NORMAL	1.023	0.2	FEMUR	NORMAL	MIXED	POSITIVE	NEGATIVE
38	7742	32	FEMALE	NEWLY DIAGNOSED	**HYPERTHYROIDISM**	NO	MIDDLE	21.6	0.919	-2.2	SPINE	OSTEOPENIA	0.72	-2.3	FEMUR	OSTEOPENIA	VEG	NEGATIVE	NEGATIVE
39	7066	30	FEMALE	7 MONTHS	HYPOTHYROIDISM	YES	LOWER	23.7	0.99	-0.6	SPINE	NORMAL	1.023	0.2	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE
40	7071	26	FEMALE	NEWLY DIAGNOSED	HYPOTHYROIDISM	YES	MIDDLE	25.6	1.133	-0.4	SPINE	NORMAL	1.029	0.2	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE
41	7078	26	FEMALE	3 MONTHS	**HYPERTHYROIDISM**	YES	MIDDLE	28.5	1.281	0.8	SPINE	NORMAL	1.039	0.3	FEMUR	NORMAL	VEG	NEGATIVE	NEGATIVE
42	7086	35	FEMALE	4 MONTHS	HYPOTHYROIDISM	YES	MIDDLE	26.4	1.073	-0.9	SPINE	NORMAL	0.869	-1.1	FEMUR	NORMAL	VEG	POSITIVE	NEGATIVE
43	7089	33	FEMALE	2 MONTHS	**HYPERTHYROIDISM**	YES	LOWER	34.6	1.027	-1.3	SPINE	OSTEOPENIA	0.874	-1.1	FEMUR	OSTEOPENIA	MIXED	POSITIVE	NEGATIVE
44	7087	28	FEMALE	6 MONTHS	HYPOTHYROIDISM	YES	LOWER	28.5	1.281	0.8	SPINE	NORMAL	1.039	0.3	FEMUR	NORMAL	VEG	NEGATIVE	NEGATIVE
45	7093	23	FEMALE	NEWLY DIAGNOSED	HYPOTHYROIDISM	NO	MIDDLE	31.2	1.239	0.5	SPINE	NORMAL	1.275	2.3	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE
46	7097	34	FEMALE	4 YEARS	HYPOTHYROIDISM	YES	MIDDLE	20.7	0.836	-2.9	SPINE	OSTEOPOROSIS	0.278	-2.7	FEMUR	OSTEOPOROSIS	VEG	NEGATIVE	NEGATIVE
47	70136	32	FEMALE	NEWLY DIAGNOSED	HYPOTHYROIDISM	NO	LOWER	25.7	1.281	0.8	SPINE	NORMAL	0.951	-0.4	FEMUR	NORMAL	VEG	POSITIVE	NEGATIVE
48	7762	26	FEMALE	4 MONTHS	**HYPERTHYROIDISM**	YES	LOWER	31.3	1.239	0.5	SPINE	NORMAL	1.113	0.9	FEMUR	NORMAL	MIXED	POSITIVE	NEGATIVE
49	8042	32	FEMALE	8 MONTHS	**HYPERTHYROIDISM**	YES	MIDDLE	33.3	1.133	-0.4	SPINE	NORMAL	1.182	1.5	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE
50	8057	26	FEMALE	2 MONTHS	HYPOTHYROIDISM	YES	MIDDLE	32.5	1.281	0.9	SPINE	NORMAL	0.998	0.3	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE
51	8053	34	FEMALE	NEWLY DIAGNOSED	HYPOTHYROIDISM	NO	MIDDLE	21.3	0.962	-1.8	SPINE	OSTEOPENIA	0.834	-1.4	FEMUR	OSTEOPENIA	MIXED	NEGATIVE	NEGATIVE
52	8013	36	FEMALE	4 MONTHS	HYPOTHYROIDISM	YES	MIDDLE	28.9	1.133	-0.4	SPINE	NORMAL	0.995	0	FEMUR	NORMAL	VEG	NEGATIVE	NEGATIVE
53	8024	28	FEMALE	NEWLY DIAGNOSED	HYPOTHYROIDISM	NO	MIDDLE	38.5	1.239	0.5	SPINE	NORMAL	0.968	-0.3	FEMUR	NORMAL	MIXED	NEGATIVE	NEGATIVE

Introduction



Objectives

Review of Literature

Methodology

Results

Discussion

Conclusion

Summary

Bibliography

Annexure-I

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Annexure-II

Annexure-III

Annexure-IV

Annexure-V