A STUDY OF THYROID DYSFUNCTION IN T2DM

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LIST OF ABBREVIATIONS

T2DM	Type 2 Diabeties Mellitus
TSH	Thyroid Stimulating Harmone
T3	Tri iodothyronine
T4	Tetra iodothyronine
FT3	Free Tri iodothyronine
FT4	Free Tetra iodothyronine
TRH	Thyro Tropin Releasing Harmone
TPO	Thyro Peroxidase
TBG	Thyroxine Binding Globulin
TG	Thyro Globuin
LDL	Low Density Lipoprotein
HDL	High Density Lipoprotein
VLDL	Very Low Density Lipoprotein
FBS	Fasting Blood Sugar
PPBS	Post Prandial Blood Sugar
HbA1C	Glycated Haemoglobin
ADA	American Diabetic Association
HLA	Human Leukocyte Antigen
MODY	Maturity Onset Diabetes of Young
OHA	Oral Hypoglycaemic Agent
BMI	Body Mass Index
WHO	World Health Organisation
CRF	Chronic Renal Failure
DKA	Diabetic Ketoacidosis

ABSTRACT

Introduction

The association between diabetes and thyroid dysfunction were first published in 1979. Type 2 diabetes mellitus (T2DM) has an intersecting underlying pathology with thyroid dysfunction. The literature is punctuated with evidence indicating a contribution of abnormalities of thyroid hormones toT2DM.

Hyperthyroidism and hypothyroidism have been associated with insulin resistance which has been reported to be the major cause of impaired glucose metabolism in T2DM. The state-of-art evidence suggests a pivotal role of insulin resistance in underlining the relation between T2DM and thyroid dysfunction

Materials and Methods

The information for the study will be collected from T2DM patients attending diabetic clinic and admitted in BLDEU'S Shri B.M Patil Medical college Hospital and Research centre,VIJAYAPUR between October 2014 to June 2017. Early morning fasting blood sample of a known T2DM patient was collected and was sent for thyroid profile. TSH, T3 and T4 will be estimated by using competitive binding ELISA method.

Inclusion Criteria

All T2DM patients attending diabetic clinic OPD and admitted in Shri B.M Patil Medical college Hospital and Research Centre, will be included in study group.

Exclusion Criteria

Those cases with known thyroid disorders, history of other illness which alter thyroid hormone levels like physiological Stress and CRF, DKA, patients on drugs like lithium and amiodarone.

Results

A hospital based prospective study among 200 T2DM patients was conducted in Shri B M Patil Medical College to evaluate the prevalence of thyroid dysfunction in patients with T2DM. 78 (39%) patients were in the age group of 51-60 years. Majority of patients were females (73%) as compared to males (27%). 25% patients had thyroid disorder and it was absent in 75% patients. Prevalence of thyroid disorder was more in female patients than in male and this association was significant.

Discussion

Thyroid diseases and diabetes mellitus are the two most common endocrine disorders encountered in clinical practice. Thyroid hormones contribute to the regulation of carbohydrate metabolism and pancreatic function and on the other hand, diabetes also affects thyroid function tests to a variable extent. It was observed that there was high prevalence of thyroid disorders in diabetic patients with advancing age. Prevalence of thyroid disorder was also significantly more in females (73%) as compared to males (27%).

Conclusion

In our study of 200 patients, 40.5% patients had diabetes for 6-10 years while 37% patients had diabetes for >1-5 years. Hence, We conclude that there is a high prevalence of thyroid disorders in patients of type 2 diabetes mellitus which was further found to be more in females, elderly patients, patients with uncontrolled diabetes i.e. HbA1C≥7, patients who were on both oral hypoglycaemic agents and insulin and patients with BMI>30. So regular screening of thyroid function in all type 2 diabetic patients should be done, especially in those with uncontrolled diabetes.

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INTRODUCTION

The association between diabetes and thyroid dysfunction were first published in 1979.[1] Thyroid dysfunction is a disorders of the thyroid gland which manifests either as hyper - or hypothyroidism and is reflected in the levels of thyroid stimulating hormone (TSH).[2]Diabetes Mellitus is the commonest endocrine disorder, leading cause of death worldwide.[3] The WHO estimated diabetes prevalence was 2.8% in 2000 and will be 4.4% by 2030. The total no. of people with diabetes is projected to rise from 171 million in 2000 to 366 million in 2030.[4] Thyroid disorders are also most common endocrine disorder in the general population after diabetes.[5] After ,1979 a number of studies estimated prevalence of thyroid dysfunction among diabetes patients ranging from 2.2-17 %.[6,7,8] However, fewer studies have estimated higher prevalence of thyrodiabetics i.e. 31% and 46.5% respectively.[9,10] Defective insulin secretion leads to various metabolic aberrations in T2DM, spanning from hyperglycemia due to defective insulin-stimulated glucose uptake and up regulated hepatic glucose production, along with dyslipidaemia, which includes impaired homeostasis of fatty acids, triglycerides, and lipoproteins.[11] DM appears to influence thyroid function at two sites; 1stly at the level of hypothalamic control of TSH release and 2ndly at peripheral tissue for converting T4 to T3. Hyperglycemia causes reduction in hepatic concentration of T4-5 deiodinase, low serum concentration of T3,raised levels of reverse T3 and low, normal, or high level of T4.

Thyroid hormone regulates metabolism and diabetes can alter metabolism. [12] The aim of this study is to evaluate the prevalence of thyroid dysfunction in T2DM.

As such a study has not been conducted in this part of country, we would like to study about thyroid dysfunction in patients with T2DM and to correlate the values of thyroid profile and diabetic profile and the risk factors associated with them.

AIMS AND OBJECTIVES

AIM:

To study the prevalence of thyroid dysfunction in patients with T2DM.

OBJECTIVE:

To correlate the values of thyroid profile and diabetic profile.

To estimate the risk factors associated between both.

REVIEW OF LITERATURE

Diabetes Mellitus

Diabetes is a syndrome of hyperglycemia and disturbances of carbohydrate, fat and protein metabolism associated with absolute or relative deficiencies in insulin secretion.

Epidemiology

In 2000 it was estimated that 171 million(2.8%) people globally suffered from diabetes. [4] Type-2 diabetes is the most common type of endocrine disorder worldwide. According to the year 2007 show that the 5 countries with the largest amount of people diagnosed with diabetes were India (40.9 million)**,** China(38.9 million), US (19.2 million), Russia(9.6 million), and Germany (7.4 million). [13]

Currently, India is the diabetes capital of the world. The world prevalence of diabetes among adults (aged 20–79 years) was found to be 6.4%, affecting 285 million adults, in2010, and it might increase to 7.7%, affecting 439 million adults by 2030. Between 2010and 2030, [14] there might be a 69% increase in numbers of adults with diabetes in developing countries and a 20% increase in developed countries.

It is estimated that over 40 million of those with diabetes are currently in India and that by 2025 that number will grow to 70 million. Another 30 million Indians will have pre-diabetes and are at high risk of developing type II diabetes mellitus (T2DM). T2DM is an economically costly disease and a major cause of mortality and morbidity. [15]

History [16]

The history of diabetes dates back to centuries before Christ, as early as 1550 BC, when the Egyptian Papyrus Ebers described an illness associated with the passage of much urine. Later, in the 2nd century A.D., Areatus of Cappadoica, a renowned Greek Physician, named the illness, "Diabetes" meaning " to run through" or a "siphon" . It was he who described it as "a melting down of the flesh and limbs into urine". Subsequently in the 5th to 6 century A.D, Susruta and Charaka, 2 notable Indian Physicians reported the association of polyuria and sweet urine. In 1674, Thomas Willis observed that diabetic urine was "as if imbued with honey and sugar". Thus, the name Diabetes mellitus was established as 'Mellitus' meaning honey. A century later, Willis Dolison demonstrated that the sweetness was indeed due to sugar. In 1869, Paul Langerhans, a medical student described the pancreatic histology in which he reported an unknown cell type in pancreas occurring as islands, 20 yrs. later, these were named, "Islets of Langerhans"by Laguesse. In 1910, Jeen De Meyer pointed out that, in diabetes, there is a lack of pancreatic secretion. A decade later, in 1921, the collaborated efforts of Frederick G. Banting, Charles H. Best, James B. Collip and JJR Macleod, led to the discovery of insulin, which could be used successfully in the treatment of diabetes. Recombinant DNA insulin became available in 1982. From diet regimens and insulin to transplantation of pancreas and islet cell, there have been considerable advances in the treatment of diabetes.

Type 2 Diabetes:

Patients are often asymptomatic. However, they may present with classical hyperglycemic symptoms of thirst, polyuria and weight loss. But they differ from type 1 diabetes, in that despite hyperglycemia, ketone bodies are present only in low

concentrations in blood and urine. Coma in these patients, if at all present, is due to hyperosmolar nonketotic coma. Lactic acidosis can occur in fulminant infections due to an acute increase in the insulin requirement. But, spontaneous ketosis does not occur.

Pancreatic pathology

The changes in both endocrine and exocrine tissue of the pancreas in type 2 Diabetes are more subtle than in type 1 diabetes. Quantitative changes include,

1) The mean total islet cell volume is moderately diminished to 50-60% of mean normal value.

2) The volume of β-cells in type 2 diabetes is decreased to about 50% of the normal value.

3) The α-cells may be increased and no change is noted in D or PP cells.

Morphologic Changes

The characteristic islet cell alteration is deposition of amyloid. Islet amyloid is not a qualitative, but is a quantitative marker of Type 2 DM. Amyloid is found in more than 25% of the islets in the body and tail of pancreas. The amyloid comes from islet amyloid polypeptides which are regulatory peptides in the central and peripheral nervous systems. There are evidences to suggest that it may induce in-vivo insulin resistance [17].

PATHOGENESIS

Although, known to run in families, modes of inheritance are not known, except for the variant known as MODY.

Markers of type 2 Diabetes Mellitus:

Molecular genetic studies in Type 2 DM have been disappointing so far. The most widely studied genes have been those for insulin and insulin receptor.

INSULIN GENE

It is located on the short arm of chromosome 11 between insulin-like growth factor-2 gene and the Harvey ras oncogene. This gene is polymorphic in regard to varying number and arrangement of tandemly repeated nucleotides beginning some 363 base pairs before transcription site. It was initially thought that homozygosity for a long insert (more than 1500 base pairs) correlated with the presence of type 2 Diabetes, but subsequent studies failed to confirm this.

The Insulin Receptor gene [18]

It is located on chromosome number 19. Three receptor mutations have been described so far, associated with diabetes mellitus and type A insulin resistance.

HLA Gene:

In contrast to type 1 DM, there are no strong association of HLA Gene with type 2 DM. However it is linked with some ethnic groups and none in Caucasian races. In South-Indian subjects, it was found that, there is an association of Type 2 DM with the fourth component, a decrease of C4B2 [19]. Among the South African and Indians, HLA Bw4 was significantly more in type 2 Diabetes than in non-diabetes subjects [20].

Etiology of type 2 diabetes mellitus:

There are 2 physiologic defects in type 2 Diabetes Mellitus. First, there is an abnormal insulin secretion. Secondly, resistance to action of insulin in the target tissues. Most of the type 2 diabetes mellitus patients are obese, and it is thought that

obesity induced secretory defect is secondary. On the other hand, many massively built individuals are not diabetic nor have glucose intolerance. It suggests that obesity doesn't lead to diabetes mellitus in the presence of normal β-cell secretory function and that relative insulin deficiency can cause insulin resistance with intact β-cell mass in type 2 diabetes mellitus, causing an increase in α and β cell ratio which accounts for excess of glucagon relative to insulin that is characteristic of Type 2 DM.

Role of Insulin Resistance

The biological response to insulin is reduced by about 40% in Type 2 DM. Insulin resistance in Type 2 DM involves effects on both hepatic glucose output and peripheral glucose uptake. Mechanism of insulin resistance is not known. It could possibly be due to decrease in number of insulin receptors, reduced tyrosine kinase activity of insulin receptors and abnormalities distal to the receptor. Fasting and Postprandial sugars and glucose tolerance tests were the tests used initially in establishing diabetic status, besides glucose estimation in urine. But all these tests have major drawbacks of being subjected to numerous fluctuations and they reflect only the current glycemic status of the patient. Glycosylated hemoglobin levels are now used extensively to determine the control of glycemic status, as it overcomes the above mentioned drawbacks of the other tests. It was in 1962, that Hursman and Dozy reported an association between glycated hemoglobin HbA1C and diabetes mellitus. The glycosylated hemoglobin levels that distinguish between the latest and overt diabetics strongly correlated with the values of fasting blood sugar values and glucose tolerance tests and reflected accurately, the changes in the control of diabetes.

Diagnosis of Diabetes Mellitus

American Diabetes Association Recommendations [21]

As per the International Expert Committee working under the sponsorship of ADA, established in May 1995.

1. Symptoms of diabetes plus random blood glucose concentration ≥ 200mg/dl. Random, means any time of the day without regard to time since the last meal.
2. Fasting blood sugar > 126mg/dl. Fasting means no caloric intake for at least 8 hours.
3. 2 hr. plasma glucose >200mg/dl during an oral glucose tolerance test, which is performed, as described by WHO, with a glucose load containing equivalent of 75gm of anhydrous glucose dissolved in water. If any of the 3 is positive, it has to be confirmed with any one of the three tests on a subsequent day.

Decode study group[21], on behalf of the European Diabetes Epidemiology group has compared the WHO and ADA diagnostic criteria in 1999. They opined that, high post-prandial sugar values are associated with an increased risk of death, independent of fasting sugar values. Hence fasting sugar values are not as satisfactory as 2 hr. glucose for the prediction of mortality. Thus, diagnostic criteria for diabetes based solely on fasting sugars are not appropriate to predict prognosis.

STRUCTURE AND FUNCTION OF THE THYROID GLAND

STRUCTURE

The thyroid gland is the largest endocrine gland in adults. It is situated in the neck, anterior and caudal to the larynx[22]. Thyroid development commences at day 24 in the human embryo as a midline thickening in the pharyngeal floor. The foetal thyroid gland can synthesize thyroxine by 11 weeks of gestation, but does not respond to pituitary secretion of TSH until about gestational week 22[23].

The naming of the thyroid gland (Greek thyreos, shield, plus eidos, form) is credited to Thomas Wharton (1614-1673), a physician at St Thomas Hospital, who described its shield shape; a structure consisting of two lobes that are connected by an isthmus. However, this shape relates more to the nearby thyroid cartilage[24]. The two lobes of the thyroid are asymmetrical with the right lobe often larger than the left. A normal thyroid weighs about 12-20gm dependent on body size and iodine supply[25]. In addition, women usually have larger thyroid glands than men. The thyroid gland enlarges during puberty, in pregnancy, during lactation, and in the latter part of the menstrual cycle. There is an apparent seasonal variation with an increase in thyroid volume (up 23%) during winter compared with summer.

FUNCTION

Thyroid hormones have effects on almost all tissues in the body. The primary function of the thyroid gland is to secrete an appropriate amount of T4 and to a lesser degree T3 to maintain the thyroid hormone levels within the body which are sufficient to meet the metabolic demands of the body. The levels of thyroid hormones are controlled by means of a negative feedback loop on the anterior pituitary and hypothalamus, whereby if the level of T3 is low, the pituitary secretes TSH, which stimulates further thyroid hormone production and secretion. Conversely if the level of T3 is raised, TSH secretion is inhibited and thyroid hormone synthesis by the thyroid gland decreases (Figure 1).

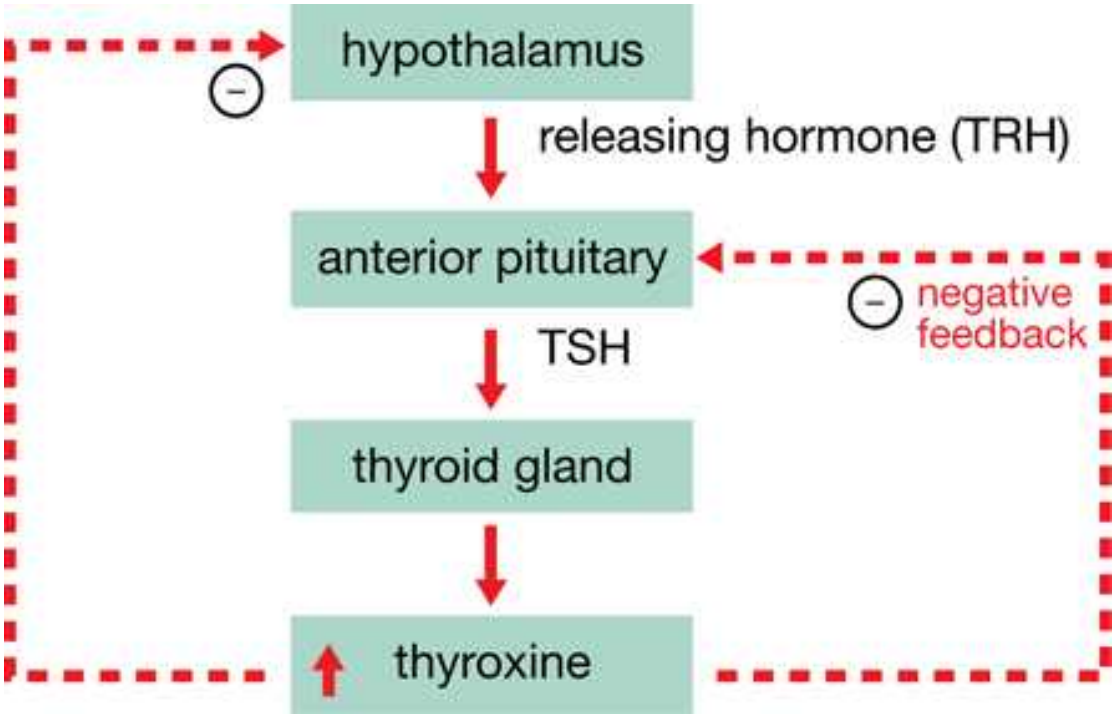


FIGURE 1: HYPOTHALAMIC-PITUITARY-THYROID AXIS

Dietary iodine is essential for thyroid hormone production. Iodide, ionized form of iodine, is bound to serum proteins, particularly albumin for transportation to the thyroid gland[26]. The thyroid gland has the capability of concentrating iodide against an electro-chemical gradient, about 20 to 40-fold above its level in the plasma[27]. Iodide is taken up by active transport into the thyroid cell by the sodium/iodide symporter, a membrane bound protein which is the first and vital step in the process of iodide supply for thyroid hormone synthesis. The sodium/iodide symporter is highly regulated, allowing for adaptation to variation in dietary supply.

The pituitary gland secretes trophic hormones which control the production of hormones by its various target organs. The thyroid gland is regulated by TSH, which itself is produced in the anterior pituitary gland. The pituitary secretes TSH which is a trophic hormone regulating both the secretion of thyroid hormones and the growth of the thyroid gland. Under normal circumstances the production of TSH is itself

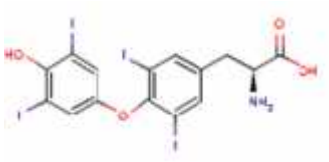
controlled by negative feedback inhibition of thyroid hormones such that, in the absence of hypothalamic or pituitary disease, illness or drugs, when the thyroid gland is overproducing thyroid hormones, the TSH level is suppressed; the converse occurs when the thyroid gland is not producing sufficient thyroid hormone (primary hypothyroidism)[28].

The synthesis and release of TSH is stimulated by thyrotropin-releasing hormone (TRH) a tripeptide released into the hypothalamic-hypophyseal portal system from the hypothalamus. TSH is inhibited by thyroid hormone by a negative feedback system. TSH is a glycoprotein composed of α and β subunits, the α subunit being common to the other glycoprotein hormones (Luteinising Hormone, Follicle Stimulating Hormone and Human Chorionic Gonadotropin), whereas the β subunit is unique to TSH. The set-point for the TRH/TSH axis (also referred to as the hypothalamic-pituitary axis) is established by TSH. This is more evident in older adults where the TSH response to decreasing FT4 levels are considered inappropriately low, suggestive of a resetting of the thyroid hormone feedback regulation threshold of TSH secretion[29] .

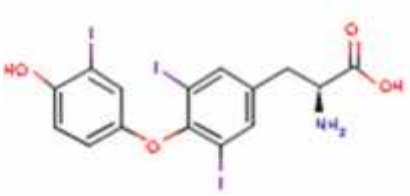
Thyroid hormone synthesis begins with uptake of iodide molecules from the plasma by the sodium/iodide symporter in the basolateral aspect of the follicular cell by the so called 'trapping'. Trapping of iodide requires active transport because the concentration gradient of iodide is higher within the cell than in the extracellular fluid. Once inside the follicle, the iodide molecules are oxidised to an 'activated' form of reactive iodine by TPO , which is attached to the apical or luminal side of the follicular cell. The amino acid, tyrosine, plays a key role in thyroid hormone synthesis. The 'activated' iodide molecules are then catalysed by TPO to bind to various tyrosine residues within the thyroglobulin molecule by a process called

‘organification’. This results in the production of monoiodotyrosine (T1) and di-iodotyrosine (T2) which, when also catalysed by TPO, couple together to form the thyroid hormones, 3,5,3‟,5‟-L-tetraiodothyronine (T4, thyroxine) and 3,5,3‟-L-triiodothyronine (T3) (Figure 2).

FIGURE 2: MOLECULAR CONFIGURATION OF T4 AND T3



3,5,3’,5’-L-tetraiodothyronine (T4)



3,5,3’- L-triiodothyronine (T3)

Thyroid hormones are stored within the colloid of the follicles. The thyroid is unique amongst endocrine glands, in that it can store its hormones for weeks at a time, in contrast to most other endocrine glands which synthesize and secrete their hormone as needed. It is estimated that thyroglobulin can store approximately a two-month supply of preformed T4 in the normal human thyroid, allowing an individual to remain euthyroid should conditions of iodine deficiency occur. The unutilised T1 and T2 are rapidly de-iodinated and the iodide thus liberated is excreted in urine or returned to the follicle via the iodide trapping mechanism. The newly synthesized T4 and T3 are released from the thyroglobulin complex, which is subsequently absorbed (through endocytosis) from the colloid, back into the follicular cells. From here, thyroid hormone is cleaved in lysozomes, yielding T4 and T3 molecules which are then secreted into the bloodstream.

The amount of reactive iodine incorporated into thyroglobulin is directly related to the concentration of iodide reaching the thyroid gland from the circulation.

Under normal conditions of dietary iodine sufficiency, thyroglobulin will contain more T2 than T1. When the T2 levels are high, thyroglobulin will contain two to four molecules of T4 with little T3 formed within it, due to insufficient availability of T1 to couple with T2. However, when dietary iodine is limited or deficient, there will be less iodide incorporated into thyroglobulin. This results in thyroglobulin containing more T1 than T2. As a consequence, under these circumstances thyroglobulin will contain more T3 than T4.

In conditions of iodide deficiency TSH is secreted and binds to the TSH receptor, stimulating both the function and growth of the thyroid gland. TSH increases the rate of iodide uptake (trapping) as well as the synthesis and release of thyroid hormone by the thyroid gland. In addition, TSH stimulates the growth of the thyroid gland which may result in a clinically enlarged thyroid (goitre). This causes compensatory change in adults where the thyroid secretes T3 in preference to T4[26]. Furthermore, changes in thyroid function can be observed in the shape of the follicular cells which alter according to the level of thyroid function. Under the microscope, depending on TSH stimulation, where there is less T4 as in hypothyroidism, the follicles are distended with colloid and the follicular cells appear flatter; in hyperthyroidism, the follicular cells become columnar[30].

T3 is the biologically active form of thyroid hormone that binds to the thyroid hormone receptor, which acts as a ligand-bound transcription factor. T4 is a prohormone which must be converted in the peripheral tissues to T3 to be biologically active. The ratio in serum of T4:T3 is usually about 20:1. T4 is recognised to be a prohormone or precursor to T3 as most of the thyroid hormone bound to receptors is in the form of T3. T4 is converted into T3 by deiodinase enzymes of which there are three types (Type I, II and III)[34]. These deiodonases are tissue specific – Type I

located primarily in the thyroid, liver and kidney has a low affinity for T4, Type II has a higher affinity for T4 and is found primarily in the pituitary gland, thyroid, brain and brown fat. Its presence allows regulation of T3 concentrations locally, that is, T4 is deiodinated peripherally to biologically active T3. The thyroid gland allows for storage of T4; acting as a reservoir for later conversion to T3. 90% of T3 available to the tissues is produced by the peripheral deiodination of T4, with only approximately 10% of T3 being directly secreted by the thyroid gland. The serum half-life of T3 is approximately one day compared to seven days for T4[31]. Type III deiodinase inactivates bothT4 and T3, converting T4 to reverse T3, a biologically inactive iodothyronine, and converting T3 to T2.

Thyroid hormones are poorly soluble in water and are transported in the bloodstream by specific carrier proteins (thyroxine binding globulin (TBG), transthyretin and albumin). Of these, TBG has the highest affinity to T4 and T3, although has the lowest concentration in serum, whereas albumin has relatively low binding affinity for T4 and T3 but is present in high concentration. T4 bound to a carrier protein is in an inactive state, that is, it is not available to be taken up by the tissue and exert its effects. A small portion of thyroid hormone is unbound (free) and considered biologically active. The free and bound hormones are in equilibrium, such that removal of the free form leads to increased dissociation of the bound hormone from its binding protein until the equilibrium and concentration of free hormone is re-established. Some conditions or medications (e.g. oestrogen containing contraceptives) affect the concentration or the ability of carrier proteins to bind to thyroid hormones. This does not affect the level of free hormones but can have a direct effect on measurement of total thyroxine (free + bound).

SUBCLINICAL HYPOTHYROIDISM:

Subclinical hypothyroidism is defined as a serum TSH concentration above the statistically defined upper limit of the reference range when serum free T4 (FT4) concentration is within its reference range[32]. Other causes of an elevated serum TSH must be excluded, for example: recent adjustments in levothyroxine dosage with failure to reach a steady state,[33] particularly in poorly compliant patients; transient increase in serum TSH in hospitalized patients during recovery from severe illness[34,35] or during recovery from destructive thyroiditis, including post viral subacute thyroiditis and postpartum thyroiditis; untreated primary adrenal insufficiency[36,37] ; patients receiving recombinant human TSH injections[38]; and the presence of heterophile antibodies against mouse proteins, which cause falsely high TSH concentrations in some assays[39]. Although central hypothyroidism (usually hypothalamic) may cause a mildly elevated serum TSH concentration (due to a circulating bioinactive TSH molecule)[40], the serum FT4 concentration is generally clearly low in these patients.

Serum TSH concentrations in a healthy population have a skewed distribution with a "tail" toward higher TSH concentrations. Because of the relatively high prevalence of subclinical hypothyroidism in the general population, it is likely that some of the skew in the upper limits of normal is a result of inclusion of patients with subclinical disease.

The third National Health and Nutrition Examination Survey (NHANES III)[41]examined serum TSH values in a "disease-free" subset (n = 13. 344) of an ethnically diverse reference population, aged 12 years and older (excluding pregnant women, individuals taking estrogens, androgens, or lithium, and those with detectable antithyroid antibodies to thyroid peroxidase [TPO] or laboratory evidence of

hypothyroidism or hyperthyroidism). In this selected population, the reference range of TSH concentration (2.5th-97.5th percentile) was 0.45 to 4.12 mIU/L, and the geometric mean TSH concentration was 1.4 mIU/L. The reference range varied as a function of age, sex, and ethnic group, but because the differences are relatively small, it is not considered necessary to adjust the reference range for these factors in clinical practice.

Some investigators suggest that the upper limit of normal for serum TSH concentration should be 2.5mIU/L[42] in a population rigorously screened to exclude thyroid disease or drugs that influence thyroid function. In support of this position is a higher rate of progression to overt hypothyroidism and a higher prevalence of antithyroid antibodies in individuals with serum TSH higher than 2.5 mIU/L compared with those with serum TSH between 0.5 and 2.5 mIU/L[43].

Possible consequences of subclinical hypothyroidism include cardiac dysfunction[44] or adverse cardiac end points (including atherosclerotic disease and cardiovascular mortality),elevation in total and low-density lipoprotein (LDL)[45] cholesterol, systemic hypothyroid symptoms or neuropsychiatric symptoms, and progression to overt, symptomatic hypothyroidism .[46]

Assessment of Evidence: The literature on subclinical hypothyroidism often arbitrarily separates patients into 2 groups determined by the degree of serum TSH elevation. To allow ease of comparison with the published literature, the evidence is divided into evidence for individuals with serum TSH concentrations between 4.5 and 10 mIU/L and those with a serum TSH higher than 10 mIU/L, when such data is available. Various studies examined the quality of the evidence for the strength of an association with certain adverse consequences of subclinical hypothyroid disease and the quality of the evidence addressing the risks and benefits of treatment.

SUBCLINICAL HYPERTHYROIDISM

It is characterised by low or undetectable levels of TSH with normal levels of free T3 and free T4.

On evaluation of the strength of the evidence for the association of untreated subclinical hyperthyroidism and the following clinical outcomes: progression to overt hyperthyroidism, adverse cardiac end points, atrial fibrillation, cardiac dysfunction, systemic and neuropsychiatric symptoms, reduced bone mineral density, and fractures[47]. A study also assessed the strength of the association between the TSH level and the risks and benefits of treatment. The body of evidence thus gathered classified patients with subclinical hyperthyroidism into 2 categories: those with mildly low but detectable serum TSH (0.1-0.45 mIU/L) and those with a clearly low serum TSH (<0.1 mIU/L).

THE INDIAN SCENARIO OF THYROID DISORDERS

Prevalence of subclinical hypothyroidism was also high in some Indian studies, the value being 9.4%. In women, the prevalence was higher, at 11.4%, when compared with men, in whom the prevalence was 6.2%. The prevalence of subclinical hypothyroidism increased with age. About 53% of subjects with subclinical hypothyroidism were positive for anti-TPO antibodies. This was a population-based study, which used cluster sampling strategy[48]. In this study, Urinary Iodine Status was studied in 954 subjects from the same population sampled, and the median value was 211 μg/l; this suggested that this population was iodine sufficient.

Population studies have suggested that about 16.7% of adult subjects have anti-thyroid peroxidase (TPO) antibodies and about 12.1% have anti-thyroglobulin (TG) antibodies. In this same study of 971 subjects, when subjects with abnormal

thyroid function were excluded, the prevalence of anti-TPO and anti-TG antibodies was 9.5% and 8.5%.

- Congenital Hypothyroidism in India

Studies from Mumbai have suggested that congenital hypothyroidism is common in India, the disease occurring in 1 out of 2640 neonates, when compared with the worldwide average value of 1 in 3800 subjects.There is often a delay in the diagnosis of congenital hypothyroidism in the country. This delay is attributable to the lack of awareness about the illness, as well as the lack of facilities available or screening program in place to comprehensively screen and test newborns for this illness.

- Overt Hypothyroidism in India (Goitre & Iodine Deficiency)

Recent population studies have shown that about 12% of adults have a palpable goiter[48]. Autoimmune thyroid disease is probably commoner than iodine deficiency as a cause of goiter in areas that are now iodine sufficient. However, given that iodine deficiency is a problem in India, the importance of iodine deficiency cannot be underestimated in the Indian context.

The link between endemic goiter and iodine deficiency has been researched in India by several eminent researchers, and this has led to the publication of several important reports[49]. Critical research has resulted in endemic goiter being reported from all over the country and not just from the Himalayan and Sub-Himalayan regions[50]. Researchers from New Delhi had shown that this was linked to iodine deficiency and that this resulted in decompensated hypothyroidism in many cases. This led to landmark studies which showed that iodine deficiency was associated with hypothyroidism in neonates, setting the scene for the now legendary salt iodization program supported by the Government of India. Subsequent to this program, it was

shown that in selected regions of Uttar Pradesh, the prevalence of congenital hypothyroidism had come down from 100/1000 to 18/1000. Several landmark studies have been carried out in the area of iodine deficiency disorders in the country[51,52].

In the post iodization phase, what happens to the prevalence of goiter? This very important question was answered in an elegantly conducted study[53]. About 14,762 children from all over India were studied for the following characteristics: goiter prevalence, urinary iodine and thiocyanate excretion, functional status of the thyroid, as well as serological and cytopathological markers for thyroid autoimmunity. About 23% of subjects had a goiter. A significantly higher level of median urinary thiocyanate (USCN) excretion was noted in goitrous subjects (0.75 mg/dl) when compared with controls (0.64 mg/dl; $P < 0.001$). The authors suggested that despite iodization, the prevalence of goiter has not dramatically declined. The researchers noted that thyroid autoimmunity could only partly explain the goiter and concluded that the role of goitrogens is an area that deserves further study.

LITERATURE REVIEW

Díez JJ et al in 2011 Assessed the prevalence of thyroid dysfunction in patients with type 2 diabetes. 318 patients (191 women, aged 29-89 yr, median duration of diabetes 8 yr) attended the diabetes clinic. The number of patients with thyroid dysfunction and their respective prevalences were: overt hyperthyroidism, 11 (3.5%); subclinical hyperthyroidism, 10 (3.1%); overt hypothyroidism, 48 (15.1%), and subclinical hypothyroidism, 34 patients (10.7%). The screening program detected the following cases of newly diagnosed thyroid dysfunction: subclinical hyperthyroidism, 5 (1.6%); overt hypothyroidism, 6 (1.9%), and subclinical hypothyroidism, 20 patients (6.3%). Therefore, total thyroid dysfunction was present in 32.4% (95% CI, 27.3-37.5%), and newly diagnosed thyroid dysfunction was present 9.7% (95% CI, 6.5-13.0%) of the patients. Logistic regression analysis showed that there were no significant relationships between the presence of thyroid dysfunction and duration of diabetes, hemoglobin A1c levels, and the presence of diabetic complications. The authors concluded that screening program detected new cases of thyroid dysfunction in ∼10% of diabetic subjects. However, they could not identify any diabetes-related clinical parameter with predictive value on the presence of thyroid dysfunction.[54]

Demitrost L et al in 2012 did a retrospective study for prevalence of thyroid dysfunction in type 2 DM in Manipur, India, data of 202 Type 2 DM patients who attended the diabetic clinic of the Regional Institute of Medical Sciences, Imphal from January 2011 to July 2012, and whose thyroid stimulating hormone (TSH) level was investigated were included. Out of the 202 type 2 DM patients for the study of which 61 were males and 141 were females, 139 (68.8%) were euthyroid, 33 (16.3%) had subclinical hypothyroidism (10 males and 23 females), 23 (11.4%) had

hypothyroidism (6 males and 17 females), 4 (2%) had subclinical hyperthyroidism and 3 (1.5%) were hyperthyroidism cases. Maximum cases were of hypothyroidism (subclinical and clinical) seen in the age group of 45-64 years. Patients with BMI > 25 were at increased risk of having hypothyroidism (P < 0.016). The study concluded that prevalence of hypothyroidism is quite high in type 2 DM patients above 45 years and more so if their BMI is over 25.[55]

Ravishankar S.N et al in 2013 studied thyroid functions in Type 2 diabetics and to know the spectrum of thyroid dysfunction in Type 2 DM. A total of 100 patients with Type 2 DM who were diagnosed on the basis of ADA criteria or who were taking treatment for Diabetes were included in the study. All patients in the study underwent thyroid profile tests for the thyroid status and also target organ evaluation for Diabetes. TPO-Ab, thyroid USG and FNAC where done where ever required. A detailed history and examination was done on these patients. A total of 100 Type 2 DM patients were included in the study. Thyroid disorders were present in 29%. Hypothyroidism in 1, hyperthyroid in 13 and subclinical hypothyroidism in 15 cases. In this study 50 patients were males and 50 were females. Females (36%) had high incidence of thyroid disorders than males (22%). Sub- clinical hypothyroidism was more common among elderly (31.25%). Elderly females had high incidence of sub- clinical hypothyroidism (18.2%). Clinical features were present in 8 patients, all of them were diagnosed hyperthyroid. Other patients did not have any signs and symptoms. Patients with hyperthyroidism had a poor glycemic control 55.5%. Duration of diabetes had no relation with incidence of thyroid disorders. Patients with severe diabetic micro vascular complication had sub- clinical hypothyroidism. The study concluded that prevalence of thyroid disorders in Diabetics was 29%. Elderly

population had more incidence than those below sixty. Sub-clinical hypothyroidism was more common among females. Diabetics with hyperthyroidism had poor glycemic control. Severe diabetic complications were noted in patients with sub-clinical hypothyroidism. Duration of Diabetes had no impact on thyroid dysfunction.[56]

Vikhe VB et al in 2013 studied to find the prevalence of thyroid dysfunction in patients with type 2 diabetes mellitus (type 2 DM) attending an outpatients department and medical wards in Dr D Y Patil Medical College and Hospital. Data of 50 diabetic and 50 non diabetic patients who attended OPD and admitted in medical wards of Dr D Y Patil Medical College and Hospital, pimpri, pune from September 2012 to September 2013. These subjects were investigated for total triidothyronine (T3), total thyroxin (T4), thyroid stimulating hormone (TSH), Fasting Blood Sugar(FBS), glycosylated hemoglobin (HbA1c), serum cholesterol, serum triglycerides, high density lipoprotein(HDL), low densit lipoprotein(LDL), very low density lipoprotein(VLDL), blood urea, serum creatinine. The level of T3and T4were significantly lower while the level of TSH was significantly higher in type 2 diabetics as compared to non-diabetics. From the 50diabetic subjects studied, 30% showed abnormal thyroid hormone levels (22 % had hypothyroidism and 8 % had hyperthyroidism). Significantly higher levels of FBS, HbA1c, serum cholesterol, serum triglyceride, LDL, VLDL, blood urea, creatinine, and significantly lower level of HDL was observed in diabetics as compared to non-diabetics subjects. The study concluded that prevalence of thyroid dysfunction among type 2 DM patients is very high (30 %) with subclinical hypothyroidism being most common. Concluding that, all patients with type 2 DM should be screened for thyroid dysfunction to reduce the mortality rate.[57]

Kumar RA et al in 2013 conducted a study to evaluate prevalence of thyroid dysfunction among south Indian type 2 diabetes individuals. Four hundred male and female type 2 diabetes patients aged between 25-75 years attending out-patient department of Karnataka institute of diabetology were randomly included in the study. Two hundred matched relatives who accompanied diabetic subjects were recruited as controls. Body mass index, waist circumference, blood pressure, Fasting plasma glucose, post prandial plasma glucose, HBA1c, lipid profile, triiodothyronin (T3),tetraiodothyronin (T4), and thyrotropin (TSH) levels were measured for all diabetes subjects and controls. Study population consisted of 59% males and 41% females in both the groups. Mean age of diabetes subjects was 55.85±9.6 years and that of controls was 55.1±8.9 years. Thyroid dysfunction was present in 24% of type 2 diabetes patients as compared to 13% of controls. Subclinical hypothyroidism was present in 11.25% vs. 7%, overt hypothyroidism in 12% vs. 5% and hyperthyroidism in 0.75% vs. 1% in diabetic subjects vs. controls.
The study concluded that prevalence of thyroid dysfunctions is high among type 2 diabetes subjects and warrants screening of diabetes subjects for thyroid functions. Failure to recognize the presence of abnormal thyroid hormone levels may be one of the reasons of poor outcome of type 2 diabetes management.[58]

Fleiner HF et al in 2016 did a cross-sectional, population-based study of adults in two surveys to investigate associations of autoimmune and type 2 diabetes with the prevalence of hypo- and hyperthyroidism. In HUNT2(a Norwegian study), autoimmune diabetes was associated with a higher age-adjusted prevalence of hypothyroidism among both women (prevalence ratio 1.79, 95% confidence interval [CI] 1.30-2.47) and men (prevalence ratio 2.71, 95% CI 1.76-4.19), compared with

having no diabetes. For hyperthyroidism, the corresponding cumulative prevalence ratios were 2.12 (95% CI 1.36-3.32) in women and 2.54 (95% CI 1.24-5.18) in men with autoimmune diabetes. The age-adjusted excess prevalence of hypothyroidism (~6 percentage points) and the presence of thyroid peroxidase antibodies (8-10 percentage points) associated with autoimmune diabetes was similar in women and men. The authors concluded that autoimmune diabetes, but not type 2 diabetes, was strongly and gender neutrally associated with an increased prevalence of hypo- and hyperthyroidism and the presence of thyroid peroxidase antibodies. Increased surveillance for hypothyroidism appears not necessary in patients with type 2 diabetes.[59]

Sahu S et al in 2015 studied 120 cases of type2 diabetes mellitus patients satisfying WHO criteria without pre-existing thyroid disease which were included in the study. Thyroid function test, fasting serum insulin was done.HOMA-IR & HOMA-B (HOMA-Homeostatic model assessment) was calculated. Serum antithyroid peroxidase anibody (anti-TPO) and antithyroglobulin antibodies (anti-TG) and ANA were done. Prevalence of thyroid dysfunction in type2 diabetes mellitus was 28.33% according to their study, which included overt hypothyroidism (15%), subclinical hypothyroidism (8.33%), secondary hypothyroidism (0.83%), overt hyperthyroidism (1.67%) and subclinical hyperthyroidism (2.5%). Anti TPO and anti TG antibodies were elevated in 62.07% cases of hypothyroidism, 40% cases of hyperthyroidism and 6.9% euthyroid cases of type2 DM. Anti TPO and antiTG antibodies were significantly raised in type2DM patient with hypothroidism than that of euthyroid (p value<0.0001). Compared to euthyroid diabetics, hypothyroid cases had lower values of insulin resistance markers like fasting insulin, HOMA-IR and

HOMA-B. Hyperthyroid cases had higher values. The study concluded that hyperthyroid diabetics have higher insulin resistance as fasting insulin, HOMA-IR, HOMA-B showed negative correlation with TSH. (p value<0.05).[60]

Moslem F et al in 2015 did a cross sectional study to explore the prevalence of thyroid dysfunction among type-2 diabetes patients attending a specialist diabetes centre in urban Dhaka. Prevalence rate of thyroid dysfunction was 10%. Females were found with higher rate of thyroid dysfunction (78.3%) with
male (21.7%). The majority of the patients had diabetes for more than a five-year duration (52%). Patients aged between 41-50years were found to be more affected with thyroid dysfunction (34.8%). The authors concluded that prevalence of thyroid disorder among female diabetes patients were higher. All patients had hypothyroidism. The approach of using a screening test to explore thyroid disorders was not effective, rather routine screening is recommended for all type 2 diabetes patients to reduce the burden of the disease.[61]

Khurana A et al in 2016 conducted a study to find out the prevalence of thyroid disorders in patients of type 2 diabetes mellitus.200 patients of type 2 diabetes mellitus aged between 40 - 70 years. All the patients were evaluated for thyroid dysfunction by testing thyroid profile (T3, T4 and TSH). The correlation of prevalence of thyroid disorder with gender distribution, age distribution, HbA1C, duration of diabetes, hypertension, family history of thyroid disorder, BMI, usage of OHAs and insulin, and dyslipidaemia was then done. The observations and interpretations were recorded and results obtained were statistically analysed. The results showed there was a high prevalence (16%) of thyroid disorders in patients of type 2 diabetes mellitus. Most common was subclinical hypothyroidism (7.5%) which

was further found to be more in females, elderly patients, patients with uncontrolled diabetes, i.e.,HbA1C values > 7or patients on insulin and patients with BMI > 30.

The study concluded that screening of thyroid dysfunction should be done in all diabetic patients – especially in patients with poor diabetic control.[62]

AIMS AND OBJECTIVES OF THE STUDY:

AIM:

To study the prevalence of thyroid dysfunction in patients with T2DM.

OBJECTIVE:

To correlate the values of thyroid profile and diabetic profile.

To estimate the risk factors associated between both.

MATERIALS AND METHODS

1. SOURCE OF DATA:

The information for the study will be collected from known or newly diagnosed T2DM patients, as per WHO criteria, attending diabetic clinic and admitted in BLDEU'S Shri B.M Patil Medical college Hospital and Research centre,VIJAYAPUR between October 2014 to June 2017.

2. METHOD OF COLLECTION OF DATA:

A detailed history and clinical examinations of patients will be done according to preforma and a total of 4 ml of venous blood from cubital vein will be collected after overnight fasting. 2m1 of blood will be collected in the fluoride vial for estimation of the fasting blood glucose and other 2 ml will be collected in plain vial and will be sent for thyroid hormone estimation by ELISA.

Two ml of blood will be collected again in fluoride vial two hours after the patient has taken his regular meals for estimation of post prandial blood glucose levels. Blood glucose will be estimated by glucose oxidase peroxidase method. TSH

and T3 and T4 will be estimated by using competitive binding ELISA . Classification of the values will be based on following criteria:

- Normal when total T4 and TSH are in normal range(i.e., TSH=0.69-2.02ng/ml, T4=4.4-10mcg/d1 for males and for females 4.8-11.6mcg/d1.
- Hypothyroidism when total T4<4.4ng/d1 and TSH >6.2m1U/1
- Subclinical hypothyroidism when T4 is within normal limit and TSH>6.2m1U/1
- Hyperthyroidism when serum TSH.4m1U/1.
- Investigations required in this study are routine standardized procedures like,
- Complete blood count
- FBS, PPBS, HBA1C
- Thyroid profile
- Lipid profile
- Renal profile

Inclusion Criteria:

a. All T2DM patients attending diabetic clinic OPD and admitted in Shri B.M Patil Medical college Hospital and Research Centre, will be included in study group.

Exclusion criteria:

1. Those cases with known thyroid disorders, history of other illness which alter thyroid hormone levels like physiological Stress and CRF, DKA, patients on drugs like lithium and amiodarone.

2. TYPE OF STUDY- prospective study.

Sample size calculation:

Considering a confidence level of 95% and confidence interval of 7 the number of patients in our study to achieve statistical significance is 196. This was calculated by Survey System (http://www.surveysystem.com/sscalc.htm#one). The Survey System ignores the population size when it is "large" or unknown. Population size is only likely to be a factor when you work with a relatively small and known group of people (e.g., the members of an association). Hence a sample size of 200 was considered adequate for our study.

STATISTICAL ANALYSIS

Quantitative data is presented with the help of Mean and Standard deviation. Comparison among the study groups is done with the help of unpaired t test as per results of normality test. Qualitative data is presented with the help of frequency and percentage table. Association among the study groups is assessed with the help of Fisher test, student 't' test and Chi-Square test. 'p' value less than 0.05 is taken as significant**.**

Pearson's chi-squared test

$$\mathrm{X}^2 = \sum_{i=1}^{n} \frac{(O_i - E_i)^2}{E_i}$$

Where X2 = Pearson's cumulative test statistic.

Oi = an observed frequency;

Ei = an expected frequency, asserted by the null hypothesis;

n = the number of cells in the table.

Results were graphically represented where deemed necessary.

Appropriate statistical software, including but not restricted to MS Excel, SPSS ver. 20 will be used for statistical analysis. Graphical representation will be done in MS Excel 2010.

OBSERVATIONS AND RESULTS

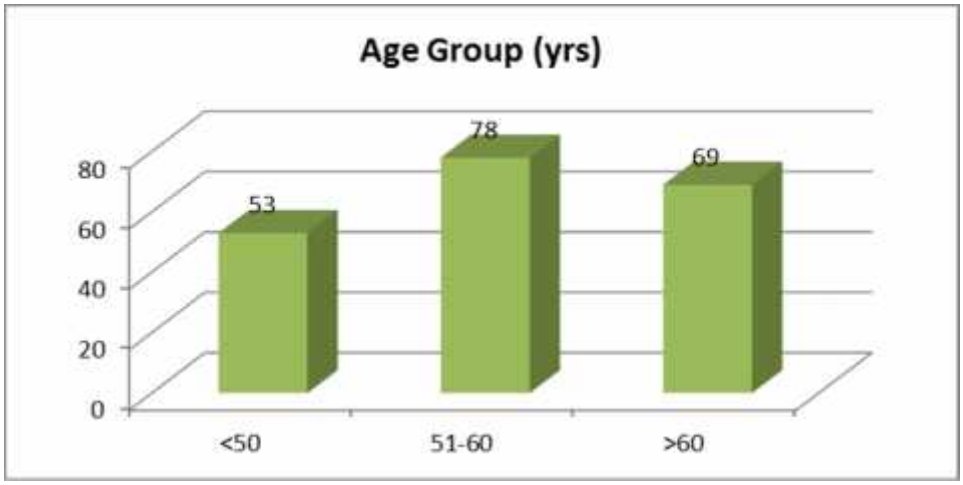
A hospital based prospective study among 200 patients was conducted to evaluate the prevalence of thyroid dysfunction in patients with T2DM.

Distribution of patients according to Age

78 (39%) patients were in the age group of 51-60 years had, followed by 69 (34.5%) patients in the age group of >60 years and 53 (26.5%) patients in the age group of <50 years.

Table 1: Distribution of patients according to Age

Age	F	%
<50	53	26.5%
51-60	78	39%
>60	69	34.5%
Total	200	100%



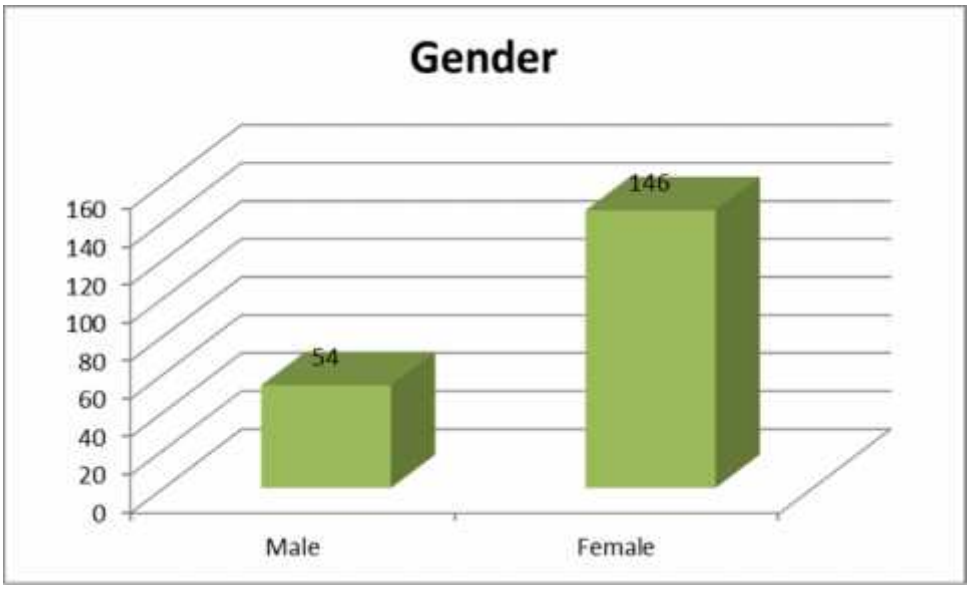
Graph 1: Distribution of patients according to Age

Distribution of patients according to Gender

Majority of patients were females (73%) as compared to males (27%).

Table 2: Distribution of patients according to Gender

Gender	N	%
Male	54	27%
Female	146	73%
Total	200	100%



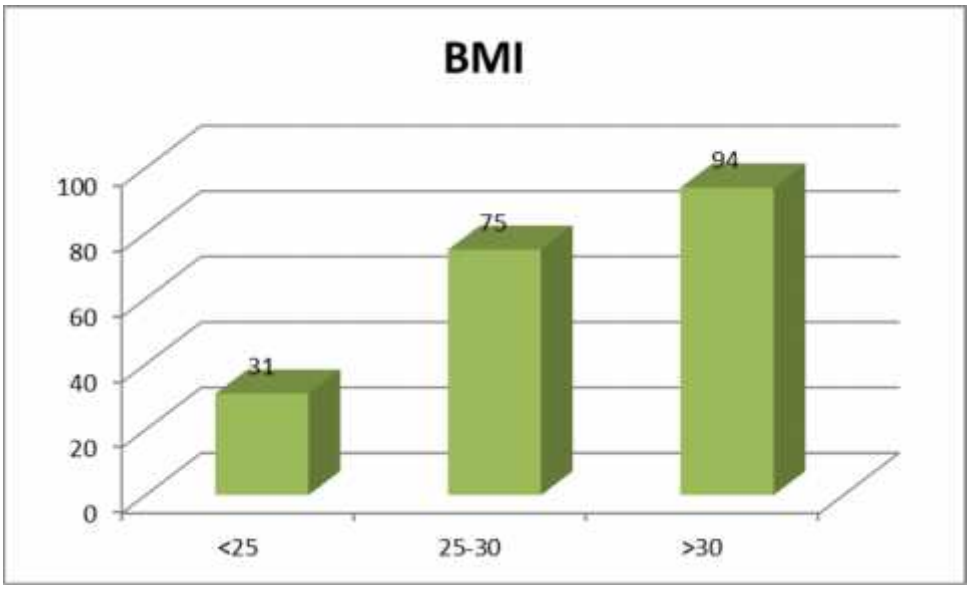
Graph 2: Distribution of patients according to Gender

Distribution of patients according to BMI

Majority of patients had BMI >30 (47%) followed by patients with BMI in the range of 25-30 (37.5%) and in the range of <25 (15.5%)

Table 3: Distribution of patients according to BMI

BMI	N	%
<25	31	15.5%
25-30	75	37.5%
>30	94	47%
Total	200	100%



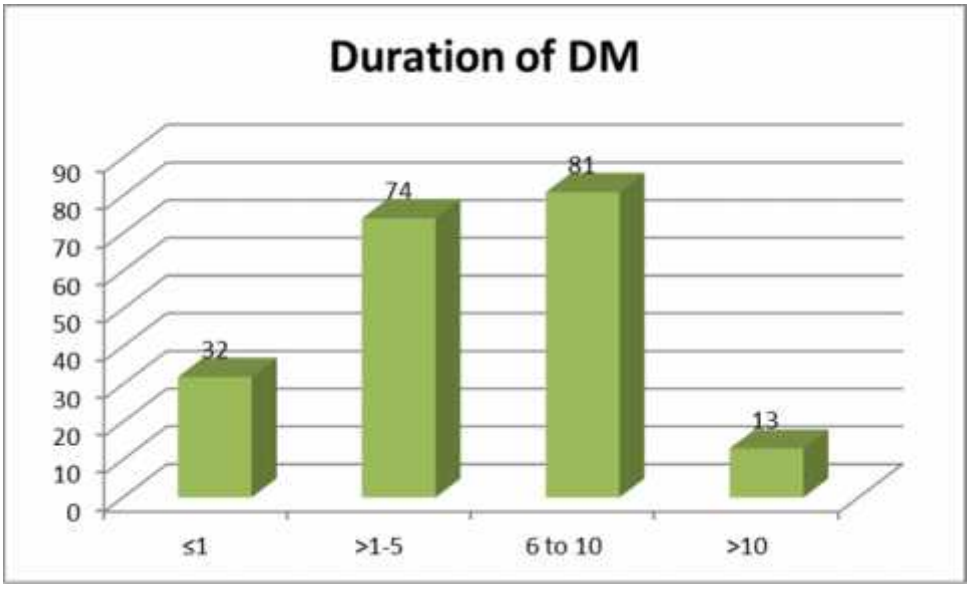
Graph 3: Distribution of patients according to BMI

Distribution of patients according to Duration of DM

40.5% patients had diabetes for 6-10 years while 37% patients had diabetes for >1-5 years. 16% and 6.5% patients had diabetes for ≤1 and >10 years respectively.

Table 4: Distribution of patients according to Duration of DM

Duration of DM	N	%
≤1	32	16%
>1-5	74	37%
6-10	81	40.5%
>10	13	6.5%
Total	200	100%



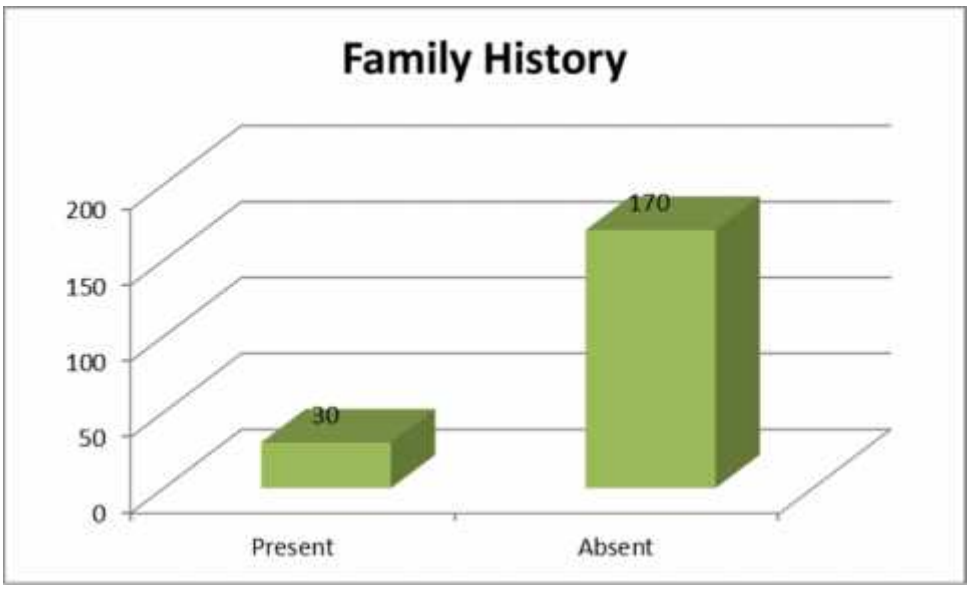
Graph 4: Distribution of patients according to Duration of DM

Distribution of patients according to Family History

15% patients had a family history of thyroid disorder whereas 85% patients had no family history of thyroid disorder.

Table 5: Distribution of patients according to Family History

Family History	N	%
Present	30	15%
Absent	170	85%
Total	200	100%



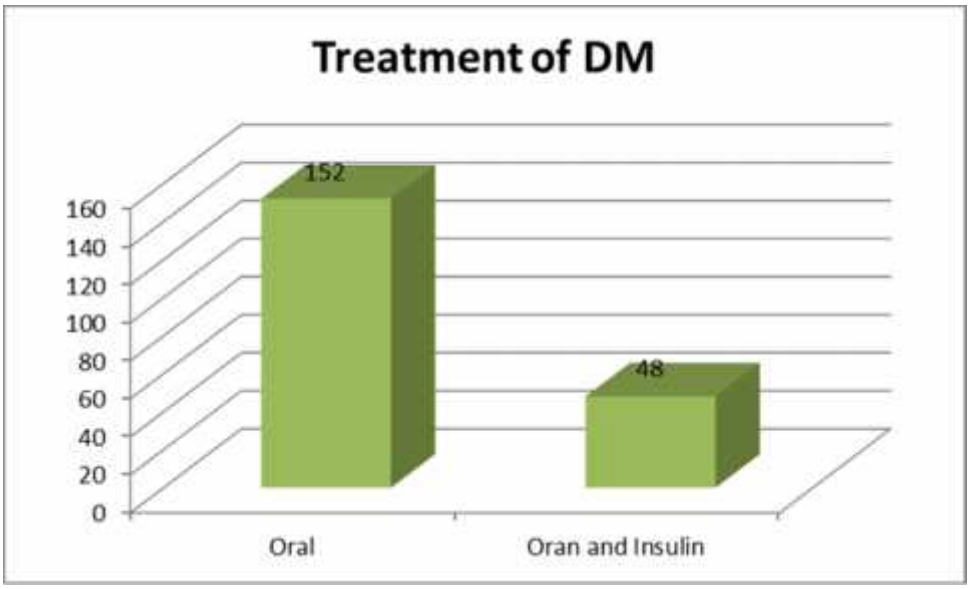
Graph 5: Distribution of patients according to Family History

Distribution of patients according to Treatment of DM

48 (24%) patients were on both oral hypoglycaemic agents and insulin while 152 (76%) patients were on oral treatment only.

Table 6: Distribution of patients according to Treatment of DM

Treatment of DM	N	%
Oral	152	76%
Oral and Insulin	48	24%
Total	200	100%



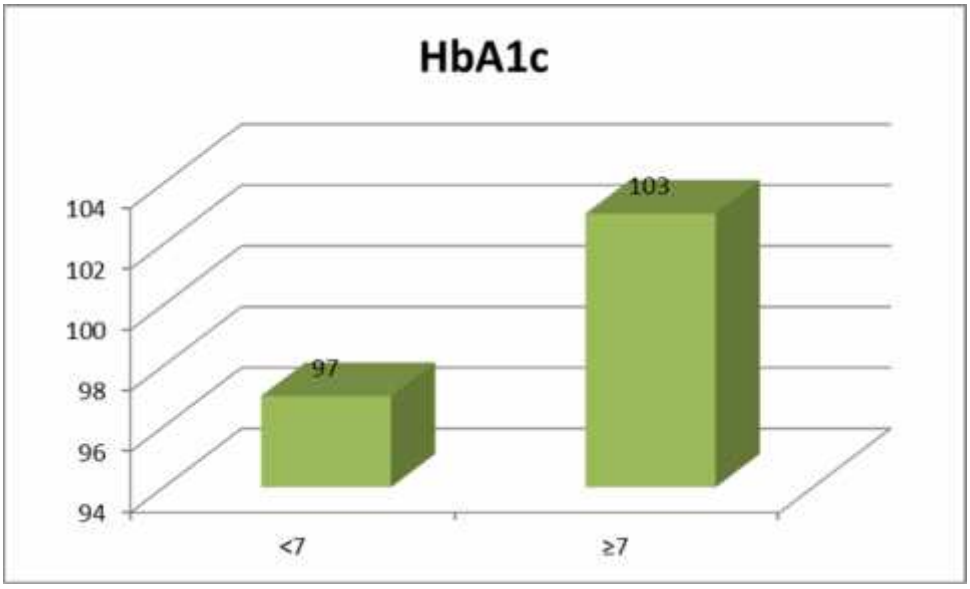
Graph 6: Distribution of patients according to Treatment of DM

Distribution of patients according to HbA1c

Majority of patients had HbA1c ≥7 i.e. in uncontrolled diabetes.

Table 7: Distribution of patients according to HbA1c

HbA1c	N	%
<7	97	48.5%
≥7	103	51.5%
Total	200	100%



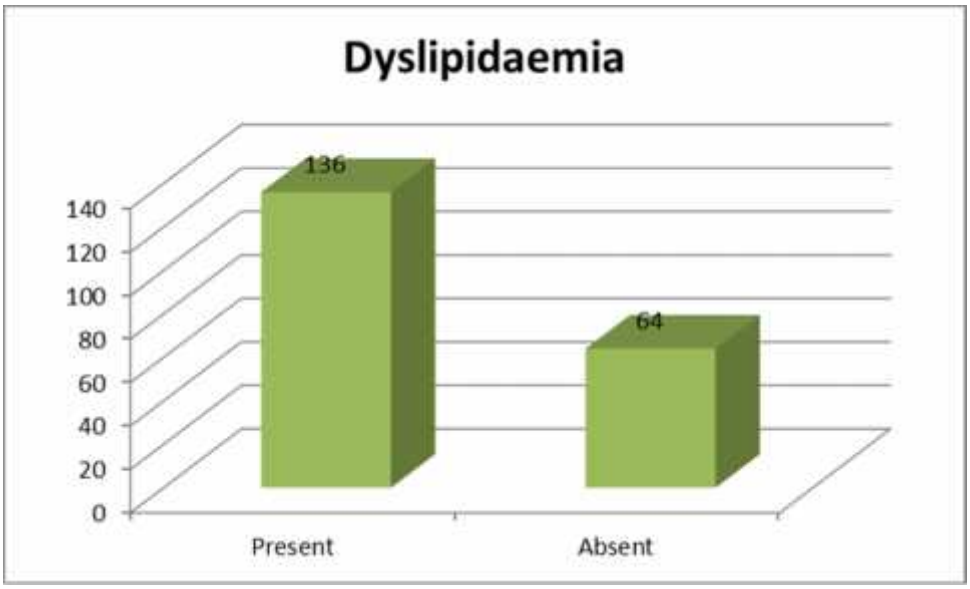
Graph 7: Distribution of patients according to HbA1c

Distribution of patients according to Dyslipidaemia

136 (68%) patients had dyslipidaemia whereas 64 (32%) patients did not have dyslipidaemia.

Table 8: Distribution of patients according to Dyslipidaemia

Dyslipidaemia	N	%
Present	136	68%
Absent	64	32%
Total	200	100%



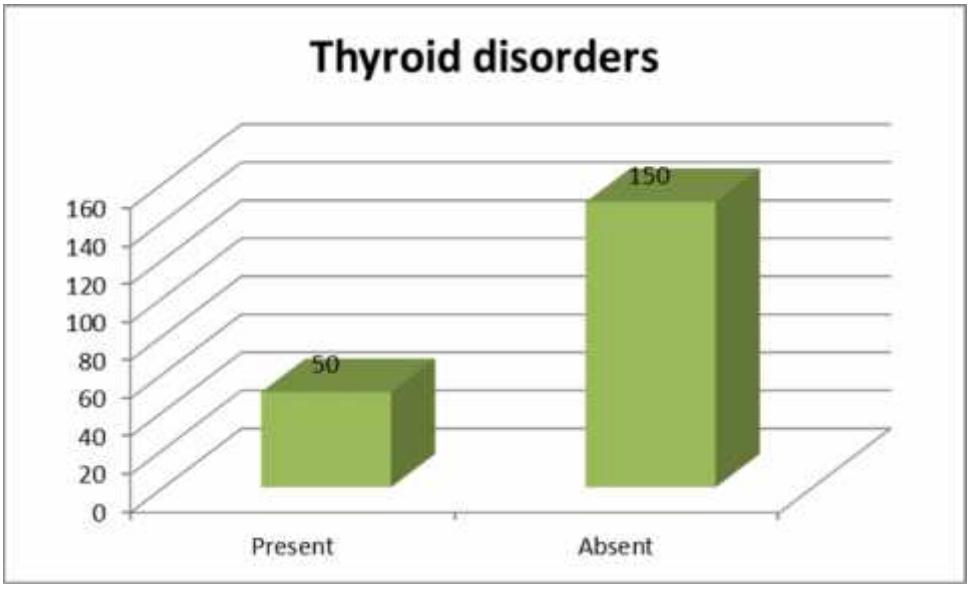
Graph 8: Distribution of patients according to Dyslipidaemia

Distribution of patients according to Thyroid disorders

25% patients had thyroid disorder and it was absent in 75% patients.

Table 9: Distribution of patients according to Thyroid disorders

Thyroid disorder	N	%
Present	50	25%
Absent	150	75%
Total	200	100%



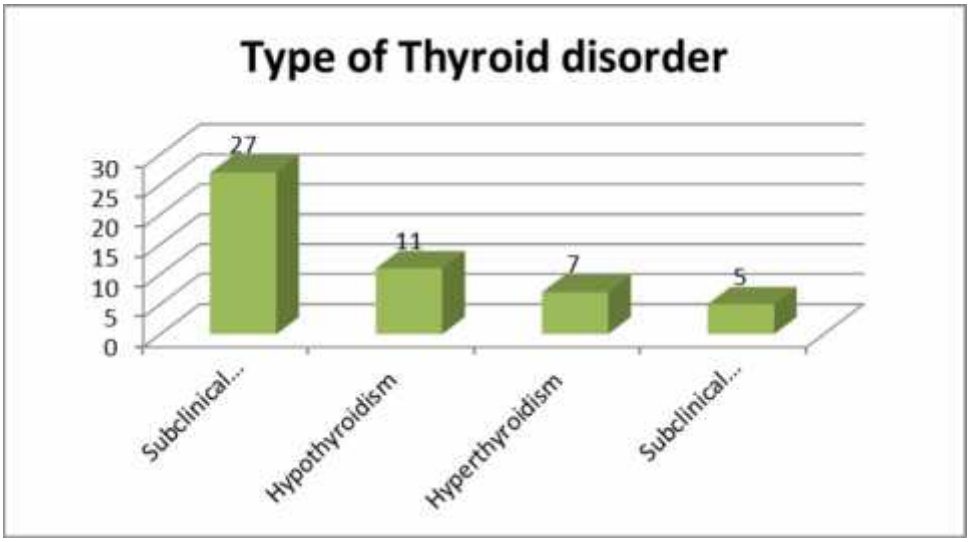
Graph 9: Distribution of patients according to Thyroid disorders

Distribution of patients according to Type of Thyroid disorder

The most common thyroid disorder was subclinical hypothyroidism (54%) followed by hypothyroidism (22%), hyperthyroidism (14%) and subclinical hyperthyroidism (10%).

Table 10: Distribution of patients according to Type of Thyroid disorder

Type of Thyroid disorder	N	%
Subclinical Hypothyroidism	27	54%
Hypothyroidism	11	22%
Hyperthyroidism	7	14%
Subclinical Hyperthyroidism	5	10%
Total	50	100%



Graph 10: Distribution of patients according to Type of Thyroid disorder

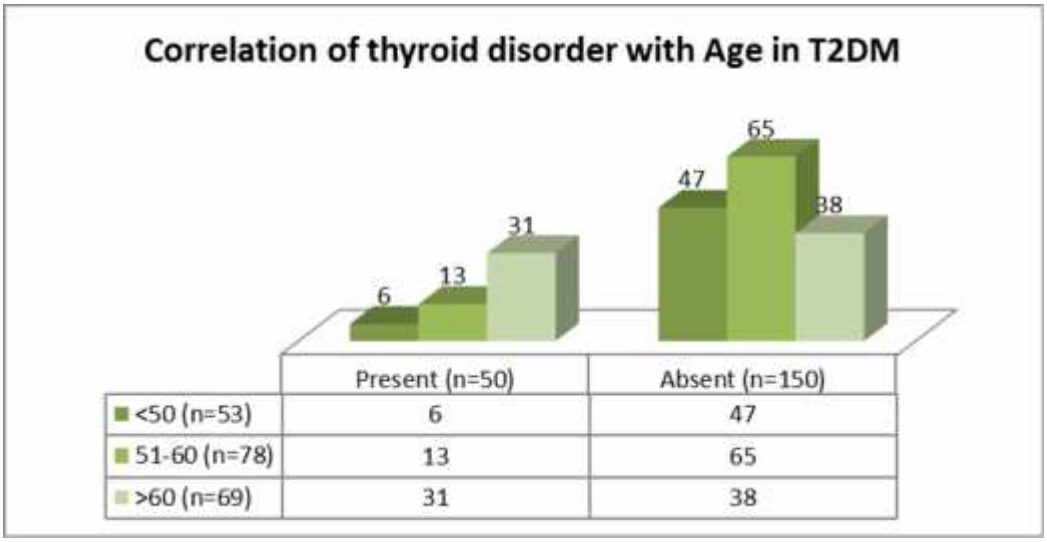
Correlation of thyroid disorder with Age in T2DM

There was significant correlation of thyroid disorder with age. It was observed that there was high prevalence of thyroid disorders in diabetic patients with advancing age.

Table 11: Correlation of thyroid disorder with Age in T2DM

Thyroid disorder	Age in years						p Value
	<50 (n=53)		51-60 (n=78)		>60 (n=69)		
	N	%	N	%	N	%	
Present (n=50)	6	12%	13	26%	31	62%	p<0.05*
Absent (n=150)	47	31.3%	65	43.3%	38	25.3%	

*** p<0.05 – Statistically Significant**



Graph 11: Correlation of thyroid disorder with Age in T2DM

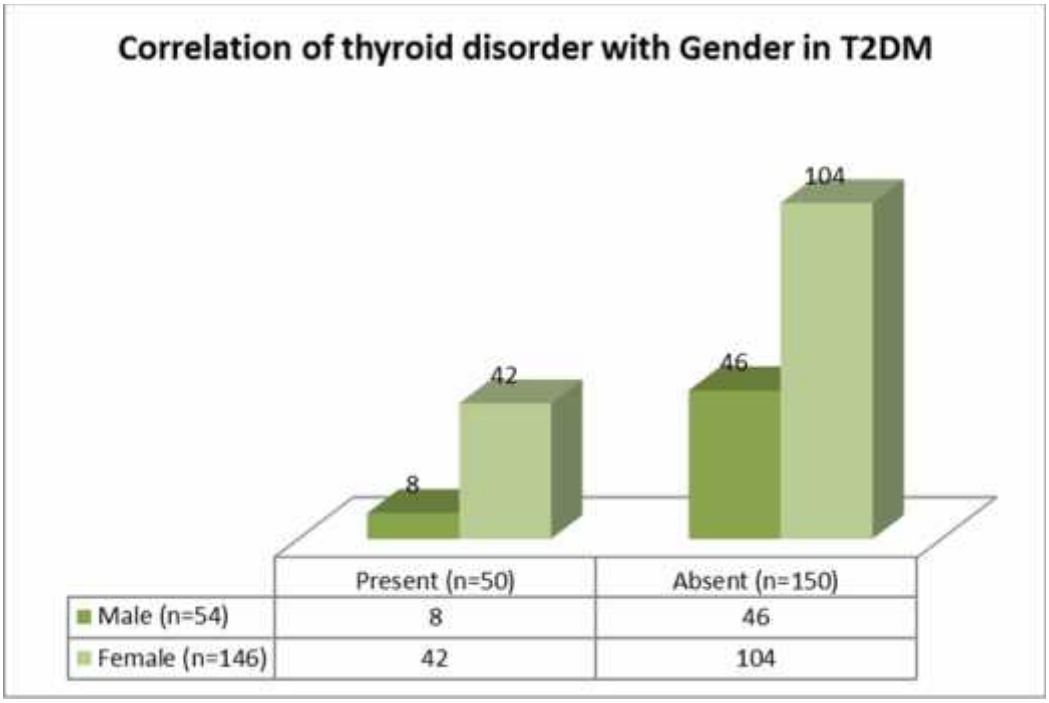
Correlation of thyroid disorder with Gender in T2DM

Prevalence of thyroid disorder was more in female patients than in male and this association was significant.

Table 12: Correlation of thyroid disorder with Gender in T2DM

Thyroid disorder	Male (n=54)		Female (n=146)		p Value
	N	%	N	%	
Present (n=50)	8	16%	42	84%	p<0.05*
Absent (n=150)	46	30.6%	104	69.4%	

*** p<0.05 – Statistically Significant**



Graph 12: Correlation of thyroid disorder with Gender in T2DM

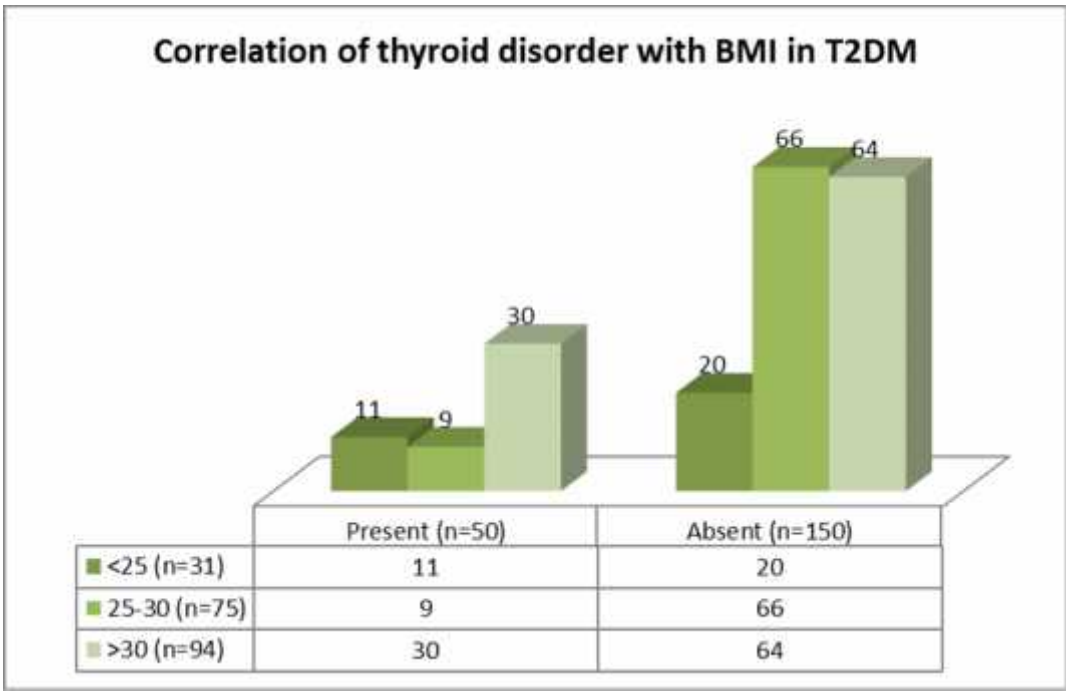
Correlation of thyroid disorder with BMI in T2DM

Out of 50 diabetic patients who had thyroid disorders, 11 (22%) had BMI <25, 9 (18%) had BMI between 25-30 and 30 (60%) had BMI >30. Thus the prevalence of thyroid disorders was found to be more in patients who had BMI >30 and this association were statistically significant.

Table 13: Correlation of thyroid disorder with BMI in T2DM

Thyroid disorder	BMI						p Value
	<25 (n=31)		25-30 (n=75)		>30 (n=94)		
	N	%	N	%	N	%	
Present (n=50)	11	22%	9	18%	30	60%	p<0.05*
Absent (n=150)	20	13.3%	66	44%	64	42.6%	

*** p<0.05 – Statistically Significant**



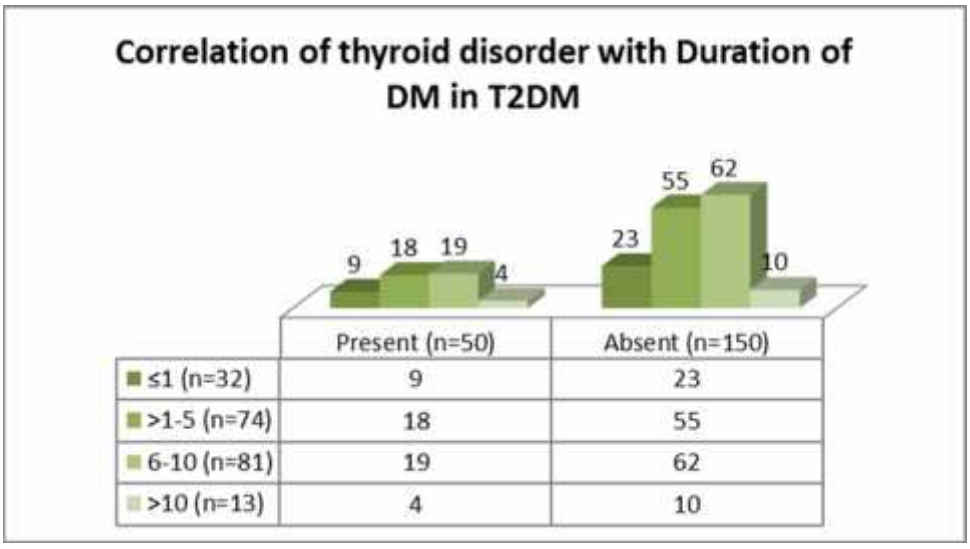
Graph 13: Correlation of thyroid disorder with BMI in T2DM

Correlation of thyroid disorder with Duration of DM in T2DM

Out of 50 diabetic patients who had thyroid disorders, 9 (18%) had duration of diabetes ≤1 yr, 18 (36%) had duration of diabetes >1 - 5 years, 19 (38%) had duration of diabetes 6 - 10 years and 4 (8%) had duration of diabetes > 10 years. However, this difference was statistically not significant

Table 14: Correlation of thyroid disorder with Duration of DM in T2DM

Thyroid disorder	Duration of DM (years)								p Value
	≤1 (n=32)		>1-5 (n=73)		6-10 (n=81)		>10 (n=14)		
	N	%	N	%	N	%	N	%	
Present (n=50)	9	18%	18	36%	19	38%	4	8%	p>0.05
Absent (n=150)	23	15.3%	55	36.7%	62	41.3%	10	6.7%	



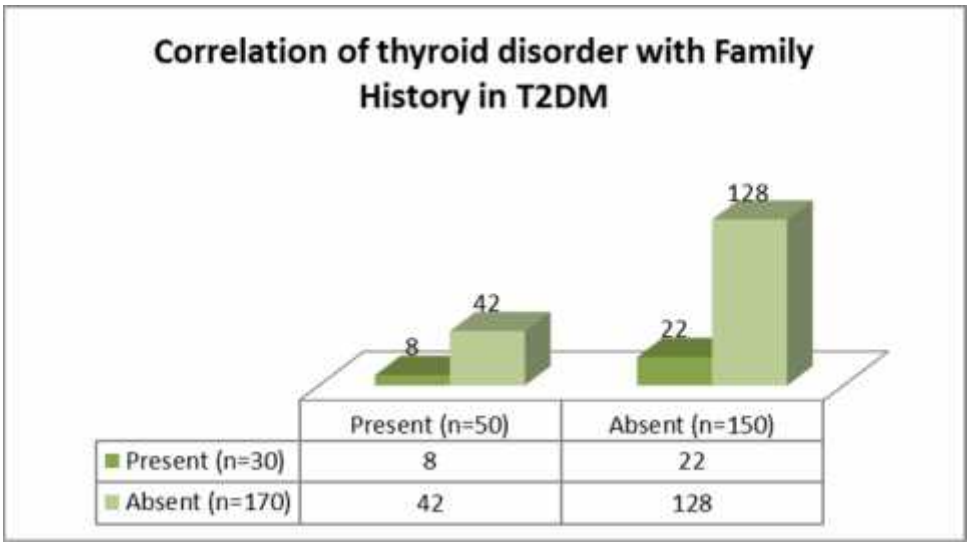
Graph 14: Correlation of thyroid disorder with Duration of DM in T2DM

Correlation of thyroid disorder with Family History in T2DM

8 (16%) had family history of thyroid disorder whereas 42 (84%) patients had no family history of thyroid disorder. The correlation of thyroid disorder with family history was statistically not significant.

Table 15: Correlation of thyroid disorder with Family History in T2DM

Thyroid disorder	Present (n=30)		Absent (n=170)		p Value
	N	%	N	%	
Present (n=50)	8	16%	42	84%	p>0.05
Absent (n=150)	22	14.7%	128	85.3%	



Graph 15: Correlation of thyroid disorder with Family History in T2DM

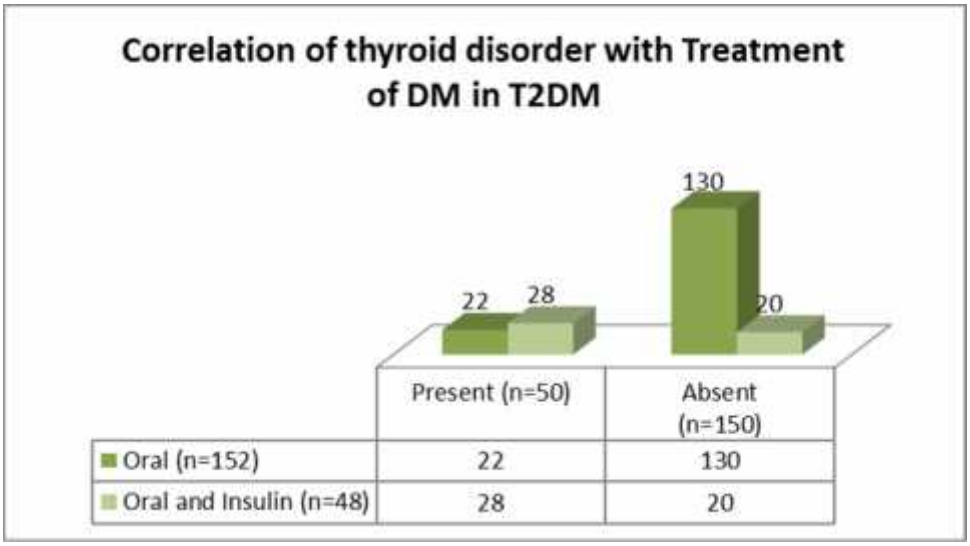
Correlation of thyroid disorder with Treatment of DM in T2DM

Among the patients with thyroid disorder in T2DM, 22 (44%) were on oral hypoglycaemic agents and 28 (56%) were on both oral hypoglycaemic agents and insulin. The prevalence of thyroid disorders was found to be more in patients who were on both oral hypoglycaemic drugs and insulin. This difference was found to be statistically significant.

Table 16: Correlation of thyroid disorder with Treatment of DM in T2DM

Thyroid disorder	Oral (n=152)		Oral and Insulin (n=48)		p Value
	N	%	N	%	
Present (n=50)	22	44%	28	56%	**p<0.05***
Absent (n=150)	130	86.7%	20	13.3%	

*** p<0.05 – Statistically Significant**



Graph 16: Correlation of thyroid disorder with Treatment of DM in T2DM

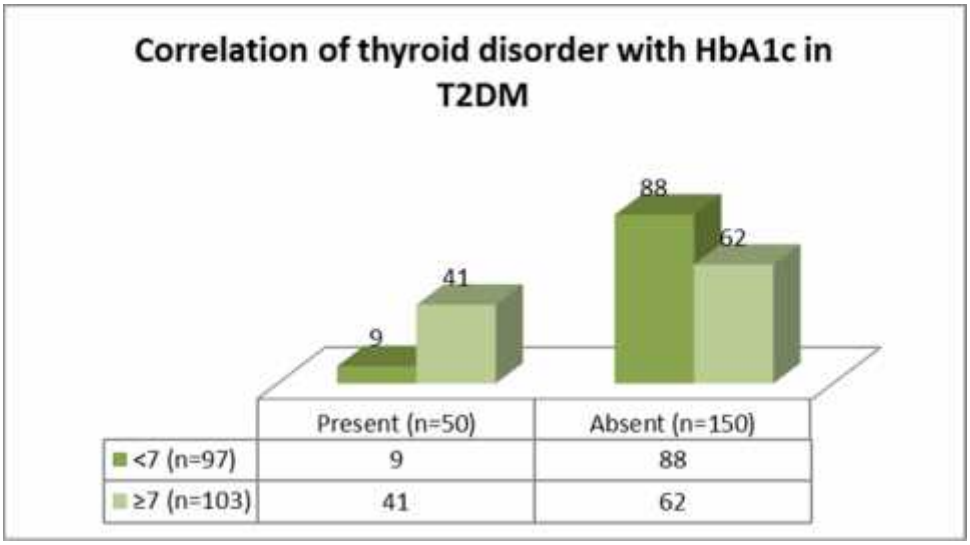
Correlation of thyroid disorder with HbA1c in T2DM

82% patients with thyroid disorder had HbA1c≥7 while 18% patients with thyroid disorder had HbA1c<7. This association of thyroid disorder with HbA1c was statistically significant.

Table 17: Correlation of thyroid disorder with HbA1c in T2DM

Thyroid disorder	<7 (n=97)		≥7 (n=103)		p Value
	N	%	N	%	
Present (n=50)	9	18%	41	82%	p<0.05*
Absent (n=150)	88	58.7%	62	41.3%	

*** p<0.05 – Statistically Significant**



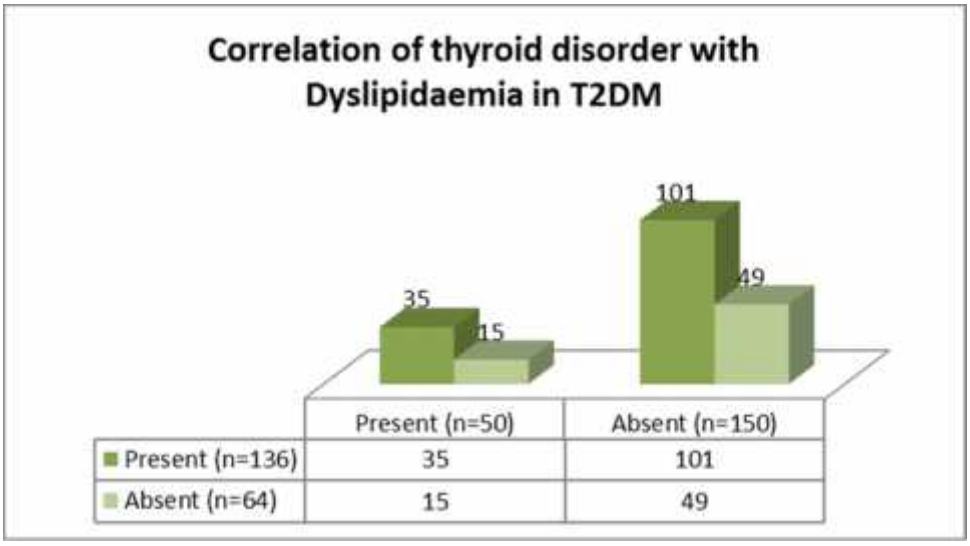
Graph 17: Correlation of thyroid disorder with HbA1c in T2DM

Correlation of thyroid disorder with Dyslipidaemia in T2DM

Out of 50 diabetic patients who had thyroid disorders, 35 (70%) had dyslipidaemia and 15 (30%) had no dyslipidaemia. The prevalence of thyroid disorders was found to be more in patients who had dyslipidaemia as compared to patients in whom dyslipidaemia was not present. However, this difference was statistically insignificant.

Table 18: Correlation of thyroid disorder with Dyslipidaemia in T2DM

Thyroid disorder	Present (n=136)		Absent (n=64)		p Value
	N	%	N	%	
Present (n=50)	35	70%	15	30%	p>0.05
Absent (n=150)	101	67.3%	49	32.7%	



Graph 18: Correlation of thyroid disorder with Dyslipidaemia in T2DM

DISCUSSION

Diabetes mellitus is one of the modern pandemics and an important health problem worldwide. Thyroid diseases and diabetes mellitus are the two most common endocrine disorders encountered in clinical practice. Thyroid hormones contribute to the regulation of carbohydrate metabolism and pancreatic function and on the other hand, diabetes also affects thyroid function tests to a variable extent. However, underlying thyroid disorders may go undiagnosed because the common signs and symptoms of thyroid disorders are similar to those for diabetes and can be overlooked or attributed to other medical disorders.

The recognition of this interdependent relationship between thyroid disease and diabetes is of importance to guide clinicians on the optimal management of both these conditions. A hospital based prospective study among 200 patients was conducted to evaluate the prevalence of thyroid dysfunction in patients with T2DM.

39% patients in the age group of 51-60 years had thyroid disorder followed by 34.5% patients in the age group of >60 years and 26.5% patients in the age group of <50 years. There was significant correlation of thyroid disorder with age. It was observed that there was high prevalence of thyroid disorders in diabetic patients with advancing age. This is similar to the studies of Vondra K et al[63], Michalek AM et al[64], Whitehead C et al[65], Feely J et al[1], Moulik PK et al[66] and Johnson JL et al[67] who also found high prevalence of thyroid disorders in diabetic patients with advancing age.

Prevalence of thyroid disorder was also significantly more in females (73%) as compared to males (27%). Our results are consistent with studies of Papazafiropoulou A et al[68], Celani MF et al[9], Vondra K et al[63], Pimenta WP et al[69], Babu K et al[70] and Michalek AM et al[64] in which they also reported prevalence of thyroid disorders

higher in diabetic females as compared to diabetic males. This was also in agreement to the study of Moslem F et al[238].

Moslem F et al[61] did a cross sectional study to explore the prevalence of thyroid dysfunction among type- 2 diabetes patients. Prevalence rate of thyroid dysfunction was 10%. Females were found with higher rate of thyroid dysfunction (78.3%) with male (21.7%). The majority of the patients had diabetes for more than five years (52%). Patients aged between 41-50 years were found to be more affected with thyroid dysfunction (34.8%). The authors concluded that prevalence of thyroid disorder among female diabetes patients were higher.

Thyroid disorder was more in patients with BMI >30 (47%) followed by patients with BMI in the range of 25-30 (37.5%) and in the range of <25 (15.5%). The prevalence of thyroid disorders was found to be more in patients who had BMI >30 and this association were statistically significant. This is similar to the studies of Papazafiropoulou A et al[68] and Proces S et al[71] who also found prevalence of thyroid disorders to be significantly more in patients who had higher BMI.

40.5% patients had diabetes for 6-10 years while 37% patients had diabetes for >1-5 years. 16% and 6.5% patients had diabetes for ≤1 and >10 years respectively. The correlation of thyroid disorder with duration of diabetes was statistically not significant. This is in agreement to the study of Diez JJ et al[54] who also found no significant relationship between presence of thyroid dysfunction and duration of diabetes.

Among 200 T2DM patients, 24% patients were on both oral hypoglycaemic agents and insulin while 76% patients were on oral treatment only. The prevalence of

thyroid disorders was found to be significantly more in patients who were on both oral hypoglycaemic agents and insulin. This is in agreement to the study of Celani et al[9] who also found prevalence of thyroid disorders significantly more in patients who were on insulin therapy.

82% patients with thyroid disorder had HbA1c≥7 while 18% patients with thyroid disorder had HbA1c<7. This association of thyroid disorder with HbA1c was statistically significant. This is comparable to study of Schlienger JL et al[72] where it was observed that patients with poorly controlled diabetes, i.e., HbA1C ≥12 were having low T3 levels, Bazrafshan HR et al[73] found significant positive correlation between HbA1C and TSH levels (Table I, p value < 0.01), Ardekani MA et al[74] found HbA1C significantly higher in diabetic patients having thyroid disorders as compared to euthyroid patients. This is also in agreement to the studies of Khurana A et al[62].

Khurana A et al[62] conducted a study to find out the prevalence of thyroid disorders in patients of type 2 diabetes mellitus. The study had 200 patients of type 2 diabetes mellitus aged between 40 - 70 years. All the patients were evaluated for thyroid dysfunction by testing thyroid profile (T3, T4 and TSH). The correlation of prevalence of thyroid disorder with gender distribution, age distribution, HbA1C, duration of diabetes, hypertension, family history of thyroid disorder, BMI, usage of OHAs and insulin, and dyslipidaemia was then done. The results showed there was a high prevalence (16%) of thyroid disorders in patients of type 2 diabetes mellitus. Most common was subclinical hypothyroidism (7.5%) which was further found to be more in females, elderly patients, patients with uncontrolled diabetes, i.e., HbA1C values ≥ 7or patients on insulin and patients with BMI > 30. The study concluded that

screening of thyroid dysfunction should be done in all diabetic patients – especially in patients with poor diabetic control.

68% patients had dyslipidaemia whereas 32% patients did not have dyslipidaemia. The prevalence of thyroid disorders was found to be more in patients who had dyslipidaemia as compared to patients in whom dyslipidaemia was not present. However, this difference was statistically insignificant. Our results are in concordance with Nobre et al[75] who also found no significant difference between lipid profile of euthyroid patients and patients having thyroid disorders in type 2 diabetes mellitus.

25% patients had thyroid disorder and it was absent in 75% patients. This is in agreement to the studies of Papazafiropoulou A et al[68] (overall prevalence of thyroid disorders was 12.3%), Nobre EL et al[75] (overall prevalence of thyroid disorders was 12.7%) and Radaiedeh AR et al[76] (overall prevalence of thyroid disorders was 12.5%).

The most common thyroid disorder was subclinical hypothyroidism (54%) followed by hypothyroidism (22%), hyperthyroidism (14%) and subclinical hyperthyroidism (10%). This correlates to the finding of Vikhe VB et al[57], Ravishankar SN et al[56], Demitrost L et al[55] and Díez JJ et al[54].

Vikhe VB et al[57] studied to find the prevalence of thyroid dysfunction in patients with type 2 diabetes mellitus (type 2 DM). Data of 50 diabetic and 50 non diabetic patients were investigated for total triidothyronine (T3), total thyroxin (T4), thyroid stimulating hormone (TSH), Fasting Blood Sugar(FBS), glycosylated hemoglobin (HbA1c), serum cholesterol, serum triglycerides, high density lipoprotein(HDL), low densit lipoprotein(LDL), very low density lipoprotein(VLDL),

blood urea, serum creatinine. The level of T3 and T4 were significantly lower while the level of TSH was significantly higher in type 2 diabetics as compared to non-diabetics. From the 50 diabetic subjects studied, 30% showed abnormal thyroid hormone levels (22% had hypothyroidism and 8% had hyperthyroidism). Significantly higher levels of FBS, HbA1c, serum cholesterol, serum triglyceride, LDL, VLDL, blood urea, creatinine and significantly lower level of HDL was observed in diabetics as compared to non-diabetics subjects. The study concluded that prevalence of thyroid dysfunction among type 2 DM patients is very high (30%) with subclinical hypothyroidism is being most common.

Ravishankar SN et al[56] studied thyroid functions in Type 2 diabetics to know the spectrum of thyroid dysfunction in Type 2 DM. A total of 100 patients with Type 2 DM who were diagnosed on the basis of ADA criteria or who were taking treatment for Diabetes were included in the study. All patients in the study underwent thyroid profile tests for the thyroid status and also target organ evaluation for Diabetes. Thyroid disorders were present in 29% - Hypothyroidism in 1, hyperthyroid in 13 and subclinical hypothyroidism in 15 cases. In this study 50 patients were males and 50 were females. Females (36%) had high incidence of thyroid disorders than males (22%). Sub- clinical hypothyroidism was more common among elderly (31.25%). The study concluded that prevalence of thyroid disorders in Diabetics was 29%. Elderly population had more incidence than those below sixty. Sub-clinical hypothyroidism was more common among females.

Demitrost L et al[55] did a retrospective study for prevalence of thyroid dysfunction in type 2 DM. Data of 202 T2DM patients were included. Out of the 202 T2DM patients for the study, 61 are males and 141 are females, 139 (68.8%) were

euthyroid, 33 (16.3%) have subclinical hypothyroidism (10 males and 23 females), 23 (11.4%) have hypothyroidism (6 males and 17 females), 4 (2%) have subclinical hyperthyroidism and 3 (1.5%) are hyperthyroidism cases. Maximum cases were of hypothyroidism (subclinical and clinical) were seen in the age group of 45-64 years. Patients with BMI > 25 were at increased risk of having hypothyroidism ($P < 0.016$). The study concluded that prevalence of hypothyroidism is quite high in type 2 DM patients above 45 years and more so if their BMI is over 25.

Díez JJ et al[54] assessed the prevalence of thyroid dysfunction in patients with type 2 diabetes. 318 patients (191 women, aged 29-89 yr, median duration of diabetes 8 yr) attended the diabetes clinic. The number of patients with thyroid dysfunction and their respective prevalence were: overt hyperthyroidism, 11 (3.5%); subclinical hyperthyroidism, 10 (3.1%); overt hypothyroidism, 48 (15.1%), and subclinical hypothyroidism, 34 patients (10.7%). The screening program detected the following cases of newly diagnosed thyroid dysfunction: subclinical hyperthyroidism, 5 (1.6%); overt hypothyroidism, 6 (1.9%), and subclinical hypothyroidism, 20 patients (6.3%). Therefore, total thyroid dysfunction was present in 32.4% and newly diagnosed thyroid dysfunction was present 9.7%.

SUMMARY

A hospital based prospective study among 200 patients was conducted to evaluate the prevalence of thyroid dysfunction in patients with T2DM. The following observations were drawn:

1. Out of 200 patients, 25% patients had thyroid disorder.
2. Among 25% of thyroid dysfunction patients, the most common thyroid disorder was subclinical hypothyroidism (54%) followed by hypothyroidism (22%), hyperthyroidism (14%) and subclinical hyperthyroidism (10%).
3. 39% patients in the age group of 51-60 years had thyroid disorder followed by 34.5% patients in the age group of >60 years and 26.5% patients in the age group of <50 years. There was significant correlation of thyroid disorder with increasing age.
4. Prevalence of thyroid disorder was also significantly more in females (73%) as compared to males (27%).
5. Thyroid disorder was more in patients with BMI >30 (47%) followed by patients with BMI in the range of 25-30 (37.5%) and in the range of <25 (15.5%). The prevalence of thyroid disorders was found to be more in patients who had BMI >30 and this association were statistically significant.
6. 40.5% patients had diabetes for 6-10 years while 37% patients had diabetes for >1-5 years. 16% and 6.5% patients had diabetes for ≤1 and >10 years respectively. The correlation of thyroid disorder with duration of diabetes was statistically not significant.
7. 24% patients were on both oral hypoglycaemic agents and insulin while 76% patients were on oral treatment only. The prevalence of thyroid disorders was found to be

significantly more in patients who were on both oral hypoglycaemic agents and insulin.

8. 82% patients with thyroid disorder had HbA1c≥7 while 18% patients with thyroid disorder had HbA1c<7. This association of thyroid disorder with HbA1c was statistically significant.
9. In our study, 68% patients had dyslipidaemia, suggesting Dyslipidemia was more common in patients with T2DM with Hypothyroidism.

CONCLUSION

Thyroid diseases and diabetes mellitus are the two most common endocrine disorders encountered in clinical practice. The recognition of this interdependent relationship between thyroid disease and diabetes is of importance to guide clinicians on the optimal management of both these conditions.

1. In our study, 40.5% patients had diabetes for 6-10 years while 37% patients had diabetes for >1-5 years. 16% and 6.5% patients had diabetes for ≤1 and >10 years respectively. The correlation of thyroid disorder with duration of diabetes was statistically not significant.

2. 24% patients were on both oral hypoglycaemic agents and insulin while 76% patients were on oral treatment only. The prevalence of thyroid disorders was found to be significantly more in patients who were on both oral hypoglycaemic agents and insulin.

   Hence, We conclude that there is a high prevalence of thyroid disorders in patients of type 2 diabetes mellitus which was further found to be more in females, elderly patients, patients with uncontrolled diabetes i.e. HbA1C≥7, patients who were on both oral hypoglycaemic agents and insulin and patients with BMI>30. So regular screening of thyroid function in all type 2 diabetic patients should be done, especially in those with uncontrolled diabetes.

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ANNEXURES

INFORMED CONSENT FOR PARTICIPATION DISSERTATION/ RESEARCH

I, the undersigned _____ S/O.D/O.W/O _____,aged ____years ordinarily resident of _____do here by state/declare that Dr Avinash V Jugati of Shri B.M.Patil Medical College and Hospital has examined me thoroughly on _____at_____(place) and has explained to me in my own language _____that I am suffering from _____ disease (condition) and this disease/condition mimic following diseases _____Further Dr Avinash V Jugati informed me that she is conducting dissertation/research titled "A STUDY OF THYROID DYSFUNCTION IN T2DM" of Shri B.M.Patil Medical College, Bijapur. Under the guidance of Dr M S Biradar requesting my participation in the study.

Apart from routine treatment procedure, the pre operative, operative, post operative and follow up observations will be utilized for the study as the reference data.

Doctor has also informed me that during conduct of this procedure _____ like adverse results may be encountered. Among the above complications most of them are treatable but are not anticipated hence there is chance of aggravation of my condition and in rare circumstances it may prove fatal in spite of anticipated diagnosis and best treatment made available.

Further Doctor has informed me that my participation in this study help in evaluation of results of the study which is useful reference for treatment of other similar cases in near future, and also I may be benefited in getting relieved of suffering or cure of the disease I am suffering.

The Doctor has also informed me that information given by me, observations made/ photographs/video graphs taken upon me by the investigator will be kept secret

and not accessed by the person other than me or my legal hirer except for academic purposes.

The Doctor did inform me that though my participation is purely voluntary based on information given to me, I can ask any clarification during the course of treatment/study related to Diagnosis, Procedure of treatment, result of treatment or prognosis. At the same time I have been informed that I can withdraw from my participation in this study at any time if I want or investigator can terminate me from the study at any time from the study but not the procedure of treatment & follow up unless I request to discharge.

In the view of anticipated or unexpected complications during the course of study, that I will be treated free of cost, as explained by the investigator.

After understanding the nature of dissertation or research, Diagnosis made, mode of treatment I the under signed Shri/Smt _____under my full conscious state of mind I agree to participate in the said research/Dissertation .

Signature of patient:

Signature of Doctor:

Witness 1

Witness 2

Date: Place:

PROFORMA

PROFORMA

Name: Age/Sex: I.P. No.

Occupation:

Address:

Date of Admission:

Chief Complaints:

History of Present IIIines:

Past history:

- History of type 2 DM
- History of IHD
- History of any thyroid disorder

Personal History

1. Smoking
2. Alcoholism
3. Diet
4. Marital status
5. Number of Children

PHYSICAL EXAMINATION

Weight

Height

BMI

Pallor

Icterus

Cyanosis

Clubbing

Vital Signs:

Pulse Rate :

B.P. :

R.R. :

Temperature :

INVESTIGATIONS

DIABETIC PROFILE:

FBS	
PPBS	
HbA1C	

LIPID PROFILE:

HAEMATOLOGY

Haemoglobin	gm %
Total WBC counts	Cells/mm3
Differential counts	%
Neutrophils	%
Lymphocytes	%
Eosinophils	%
Monocytes	%
Basophils	%
ESR	mm after 1 hour

BIOCHEMISTRY

Blood Urea	
Serum creatinine	

URINE EXAMINATION –

Albumin	
Sugar	
Microscopy	

Thyroid Profile

T3	
T4	
TSH	

MASTER CHART

Sr.No.	Name	I.P.no	Age	Gender	Weight	Height	BMI	Duration of DM	FH	Treatment	HbA1c	TOTAL CHOLESTEROL	LDL	TRIGLYCERIDES	HDL	T3	T4	TSH	Type of Thyroid disorder
1	Jagadish Adaki	16869	40	M	54	163	20.3	0.8	N	Oral	5.60	148	87	166	30	135.32	7.35	5.11	-
2	Sundar Mane	16871	38	M	71	162	27.1	6	N	Oral	8.58	155	105	50	40	97.54	13.54	2.11	-
3	Gurupadayya Mangi	16883	37	M	72	167	25.8	9	N	Oral	9.37	246	173	233	38	108.00	8.47	1.19	-
4	Mohammad Hanif	16966	37	M	51	150	22.2	0.6	N	Oral	6.45	202	125	90	59	93.49	6.54	2.08	-
5	Adagondappa	17000	48	M	56	156	23	0.8	N	Oral	6.17	156	92	211	32	104.03	10.13	1.52	-
6	Paramanand	17060	38	M	60	142	29.8	1	N	Oral	5.53	145	81	85	47	119.86	13.27	5.69	-
7	Tukaram Rajput	15134	35	M	56	157	22.7	0.9	Y	Oral	5.79	195	122	68	59	89.06	6.35	5.21	-
8	Shivaputrappa Bandi	15140	44	M	70	159	27.7	1	N	Oral	8.06	330	256	131	48	129.96	10.95	1.59	-
9	Hanamanth Pawar	15161	39	M	54	156	22.2	0.8	Y	Oral	5.53	210	152	57	47	138.10	7.82	1.19	-
10	Sidanna Hugar	15369	47	M	80	164	29.7	9	N	Oral+Insulin	7.57	162	91	135	44	127.32	8.85	2.03	-
11	Ravindranath Madyal	15542	42	M	65	158	26	2	N	Oral	6.55	205	121	138	56	94.42	11.25	2.56	-
12	Parshuram Pawar	15564	38	M	63	155	26.2	1	N	Oral	5.61	250	167	131	37	125.71	6.16	5.11	-
13	Mahibub Manyar	15589	47	M	77	180	23.8	7	N	Oral	9.76	188	134	80	38	82.45	9.61	1.85	-
14	Rudrappa Mudhol	12969	47	M	66	175	21.6	5	N	Oral	6.17	222	150	122	48	135.84	6.66	8.76	-
15	Shivappagouda Biradar	13059	42	M	64	161	24.7	2	N	Oral	5.55	189	132	63	44	127.39	7.34	5.69	-
16	B Rajendra	13143	42	M	74	168	26.2	9	N	Oral	9.79	276	201	157	44	112.27	6.38	1.8	-
17	Hanamanth Pareet	11192	43	M	78	162	29.7	8	N	Oral+Insulin	7.44	194	141	96	34	124.10	7.76	4.5	-
18	Thippanna Indi	9709	46	M	59	150	26.2	5	Y	Oral	5.85	217	153	114	49	137.37	9.31	1.95	-
19	Sangappa Indi	9851	43	M	58	149	26.1	5	Y	Oral	6.35	188	108	120	56	113.26	5.89	1.49	-
20	Vittal Chalawadi	9981	39	M	70	162	26.7	1	N	Oral	6.29	328	250	119	54	125.14	10.35	2.46	-
21	Ramappa Bandiwaddar	10139	48	M	80	166	29	8	N	Oral+Insulin	9.73	222	150	122	48	140.14	7.97	1.01	-
22	Shankareppa Dashwanth	8273	39	M	79	171	27	9	N	Oral+Insulin	8.43	238	159	316	41	133.92	13.10	2.99	-
23	Mallappa Kumbar	8352	42	M	70	157	25.3	5	N	Oral	8.64	314	217	500	51	140.05	5.77	2.37	-
24	Shankerappa Sondur	8457	37	M	72	155	30	6	N	Oral	9.30	177	89	154	57	116.19	9.40	5.11	-
25	Shankargouda Biradar	5793	36	M	57	165	20.9	2	Y	Oral	6.35	217	132	123	60	137.00	9.12	1.71	-
26	Sangappa Biradar	5991	43	M	65	155	27.1	4	N	Oral	5.76	260	185	189	43	83.72	10.15	2.22	-
27	Siddanna Mannur	6078	38	M	66	152	28.6	2	N	Oral	5.69	162	91	135	44	86.22	9.62	1.23	-
28	Iranna Hadapad	6083	48	M	73	172	24.7	7	N	Oral	7.24	247	177	80	54	120.58	12.20	1.98	-
29	Irayya Allalmath	6256	38	M	66	165	24.2	1	N	Oral	6.58	224	147	249	42	100.75	7.28	2.05	-
30	Laxman Biradar	5166	48	M	77	161	29.7	8	N	Oral	9.31	246	175	147	42	94.00	12.66	1.04	-
31	Shivappa Kumbar	5196	47	M	62	160	24.2	4	N	Oral	6.68	170	91	160	48	102.61	11.55	5.7	-
32	Shankar Halagani	3940	41	M	53	162	20.2	0.7	Y	Oral	6.05	181	111	57	59	137.67	7.69	5.11	-
33	Kantappa Jadar	2541	49	M	74	178	23.4	7	N	Oral	9.16	253	190	74	42	105.35	6.81	5.11	-
34	Kallappa Shimpi	2607	41	M	71	170	24.6	9	N	Oral	9.57	186	119	122	54	113.88	9.95	1.01	-
35	Rajaram Chavan	2881	42	M	80	168	28.3	8	N	Oral+Insulin	7.37	223	156	214	34	96.45	10.61	2.64	-
36	Bhimanna Thodalbhagi	2971	37	M	58	155	24.1	5	N	Oral	5.83	247	177	80	54	105.44	11.17	3.41	-
37	Siddaraya Metri	1532	41	M	61	166	22.1	3	N	Oral	6.39	196	136	87	46	134.35	7.97	1.9	-
38	Shivanand Khabade	1537	35	M	74	163	27.9	9	N	Oral	9.56	217	153	114	49	134.92	10.14	5.7	-
39	Hanamanth Papadi	72	40	M	80	164	29.7	6	N	Oral+Insulin	8.18	208	137	71	57	130.20	9.67	3.02	-
40	Dhondiram Chavan	249	44	M	71	162	27.1	8	N	Oral	7.35	121	65	51	46	84.44	10.81	1.5	-
41	Bagappa Banikol	276	43	M	68	155	28.3	3	N	Oral	5.72	211	138	329	35	85.81	12.13	6.5	-
42	Ravindra Jigajini	448	42	M	74	158	29.3	8	N	Oral	9.74	196	127	84	52	114.34	12.10	1.03	-
43	Veerendra Patil	453	45	M	68	165	25	1	N	Oral	5.93	220	149	71	57	129.00	7.27	1.33	-
44	Husensab Mulla	40166	44	M	47	153	20.1	0.8	Y	Oral	6.18	184	120	78	48	90.69	9.27	1.99	-
45	Manikchand Bogar	41289	44	M	68	163	25.6	2	N	Oral	6.06	179	104	358	35	93.62	5.51	1.01	-
46	Mumtaz Benakanahalli	41321	47	F	80	183	23.9	9	N	Oral+Insulin	9.30	224	147	249	42	112.04	8.77	2.08	-
47	Surayya Gulburga	27704	47	F	80	175	86.1	8	N	Oral+Insulin	8.36	233	139	333	38	97.37	7.02	2.11	-
48	Nagamma Hadapad	27895	45	F	66	161	25.5	3	N	Oral	5.81	233	139	333	38	104.41	11.99	4.5	-
49	Sharadabai Chavan	26135	36	F	45	162	17.1	0.6	Y	Oral	6.25	240	155	119	61	82.62	8.70	2.99	-

50	Raziyabee Loni	26434	47	F	54	142	26.8	0.8	Y	Oral	6.19	124	61	206	30	129.11	8.99	1.19	-
51	Devibai Naik	26085	43	F	74	166	26.9	7	N	Oral	8.10	145	81	85	47	108.86	13.19	2.83	-
52	Sonabai Kokatnur	26298	48	F	53	158	21.2	0.7	N	Oral	6.10	249	173	81	60	99.23	7.73	2.07	-
53	Mahananda Guddodagi	26404	49	F	80	183	23.9	7	N	Oral+Insulin	7.36	259	188	187	39	102.99	7.07	1.5	-
54	Shalini Hanchate	26501	69	F	55	165	20.2	0.6	N	Oral	5.57	111	54	103	36	108.41	8.25	1.72	-
55	Kasturi Sakri	26554	66	F	66	161	25.5	1	N	Oral	6.58	194	112	601	31	124.01	6.63	1.42	-
56	Parvathi Revanekar	26590	72	F	50	152	21.6	0.6	Y	Oral	5.55	151	83	186	36	92.97	9.21	2.03	-
57	Dodawwa Pujari	21966	67	F	49	151	21.5	0.8	Y	Oral	5.76	195	130	120	41	109.60	7.96	3.02	-
58	Shivabai Vastrad	22214	65	F	70	159	27.7	6	N	Oral	8.38	202	125	90	59	95.86	10.07	3.02	-
59	Shankeramma Jamakhandi	22249	66	F	59	150	26.2	4	Y	Oral	5.64	196	127	84	52	98.93	6.34	1.97	-
60	Sharanawwa Dashwant	22209	65	F	71	162	27.1	9	N	Oral	8.61	148	87	166	30	117.42	8.15	5.11	-
61	Gangabai Bannur	16835	70	F	80	160	31.2	13	N	Oral+Insulin	7.75	177	107	56	59	84.99	8.54	1.73	-
62	Soumya Kamble	16880	65	F	47	153	20.1	0.6	N	Oral	6.53	211	135	56	65	118.83	10.13	2.03	-
63	Kamala Yendigeri	17142	70	F	64	147	29.6	3	N	Oral	5.64	205	138	84	50	93.75	12.60	5.11	-
64	Kasturi Degadi	17289	74	F	64	153	27.3	1	N	Oral	6.17	222	154	62	56	124.13	13.55	1.42	-
65	Mahadevi Mulwad	17305	66	F	80	164	29.7	10	N	Oral+Insulin	7.53	328	250	119	54	133.88	11.42	1.38	-
66	Savita Bomanahalli	17536	72	F	63	180	19.4	2	Y	Oral	6.38	230	153	98	48	90.12	11.37	1.35	-
67	Sakkubai	14781	62	F	64	161	24.7	4	N	Oral	6.15	214	155	93	40	135.36	7.77	6.34	-
68	Kasturibai	14828	63	F	74	158	29.3	10	N	Oral	9.74	278	185	522	32	136.99	6.28	2.45	-
69	Shalubai	14845	71	F	66	162	25.1	2	N	Oral	5.76	190	114	217	44	131.18	9.08	4.07	-
70	Vachala Jhadhav	14847	70	F	77	183	23	6	N	Oral+Insulin	8.21	230	153	98	48	118.55	12.29	2.22	-
71	Laxmi Balundagi	14943	73	F	67	170	23.2	3	N	Oral	5.72	263	179	317	46	104.52	11.57	2.05	-
72	Madevi Kambar	14944	65	F	68	163	25.6	1	N	Oral	6.38	288	206	248	47	110.72	6.75	1.01	-
73	Awamma Indi	14965	70	F	63	150	28	4	N	Oral	5.69	198	116	130	56	105.74	10.84	2.45	-
74	Suslabai Bhusanur	13868	61	F	72	160	28.1	6	N	Oral	9.67	156	92	211	32	107.43	12.33	1.7	-
75	Shamla	14313	71	F	73	158	29.2	9	N	Oral	8.22	218	149	74	54	125.19	6.26	1.65	-
76	Danamma	14327	66	F	77	180	23.8	8	N	Oral+Insulin	8.78	198	116	130	56	114.42	10.39	2.56	-
77	Rukmabai	14418	68	F	39	142	19.3	0.6	N	Oral	6.44	258	183	88	57	127.86	10.70	1.52	-
78	Bandakka Patil	12401	64	F	65	155	27.1	4	N	Oral	5.82	223	156	214	34	122.76	10.32	6.5	-
79	Sonabai Kattimani	12445	71	F	72	175	23.5	6	N	Oral	7.63	156	101	54	44	120.63	12.46	1.72	-
80	Mala Sheik	12454	66	F	66	175	21.6	1	N	Oral	5.53	174	107	104	46	88.13	6.85	2.31	-
81	Ilachibai	4811	68	F	70	164	26	8	N	Oral	9.29	211	125	96	52	123.34	5.40	2.03	-
82	Lata	4886	67	F	64	165	23.5	4	N	Oral	6.16	238	159	316	41	118.61	12.79	3.27	-
83	Dundawwa	5370	71	F	66	163	24.8	1	N	Oral	5.88	260	186	203	41	114.68	7.64	5.21	-
84	Dhamabai	5407	74	F	45	162	17.1	0.8	N	Oral	6.02	190	131	52	59	113.12	11.98	1.82	-
85	Shantabai Kadani	5443	61	F	71	170	24.6	9	N	Oral	9.50	210	152	57	47	109.67	11.93	5.7	-
86	Mahubi Honnutagi	5448	67	F	80	168	28.3	10	N	Oral+Insulin	9.51	178	107	99	51	100.29	7.16	1.49	-
87	Anasuya Pujari	3981	64	F	68	155	28.3	3	N	Oral	5.62	228	161	168	36	112.94	8.45	1.23	-
88	Dandawwa Biradar	3993	61	F	61	166	22.1	5	N	Oral	6.33	125	60	86	48	131.46	6.14	4.5	-
89	Sidamma	3999	62	F	74	168	26.2	10	N	Oral	7.65	168	115	68	39	137.82	10.31	1.24	-
90	Ratnabai	4265	72	F	51	135	28	0.6	N	Oral	5.53	155	105	50	40	82.49	5.41	2.5	-
91	Shankerwwa Hadapad	4404	67	F	79	178	24.9	10	N	Oral+Insulin	9.33	205	121	138	56	139.57	9.43	5.7	-
92	Chandrawwa Loni	4627	64	F	74	166	26.9	6	N	Oral	8.90	196	136	87	46	96.68	6.30	1.35	-
93	Chandramma Dhabalagundi	3705	68	F	80	170	27.7	12	N	Oral+Insulin	8.76	184	120	78	48	92.51	5.57	2.01	-
94	Sattawa Kotyal	3818	63	F	78	162	29.7	7	N	Oral+Insulin	8.38	214	155	93	40	111.45	6.77	1.52	-
95	Shanta Soudi	3958	65	F	68	165	25	5	N	Oral	6.12	251	190	223	27	118.34	7.32	2.32	-
96	Kasturibai	3977	72	F	70	180	21.6	10	N	Oral	9.32	221	154	54	56	97.54	9.31	2.08	-
97	Anasuya Pujari	3981	68	F	61	155	25.4	3	Y	Oral	6.54	254	200	107	33	122.34	8.08	1.65	-
98	Siddamma Shinde	3999	67	F	70	169	24.5	10	N	Oral	8.17	250	175	80	59	95.29	12.52	2.64	-
99	Nandamma Alayagol	3428	68	F	55	172	18.6	0.5	Y	Oral	5.72	237	165	251	37	117.92	10.98	1.7	-
100	Sharanawwa Burukule	3474	66	F	62	160	24.2	4	Y	Oral	6.34	175	103	59	56	101.83	5.78	2.83	-
101	Kamalamma Biradar	3615	62	F	70	157	25.3	1	N	Oral	9.79	151	83	186	36	103.51	11.15	5.7	-
102	Chandramma Dhabalagundi	3805	67	F	67	154	28.3	5	N	Oral	5.56	201	130	214	38	106.97	8.39	2.26	-
103	Sitabai Maske	3042	62	F	80	168	28.3	12	N	Oral+Insulin	8.22	210	139	279	35	120.27	7.84	5.7	-
104	Sonayya Doddamani	3071	68	F	60	168	21.3	1	Y	Oral	5.78	278	185	522	32	138.36	7.43	1.24	-
105	Shivabai Ronihal	3196	71	F	80	182	24.2	13	N	Oral+Insulin	8.97	259	188	96	52	126.63	8.79	1.97	-

106	Rukmini	2037	68	F	64	177	20.4	5	N	Oral	5.92	155	94	60	49	112.28	8.77	2.02	-
107	Satavva Kotyal	2240	74	F	72	167	25.8	8	N	Oral	7.41	277	225	98	32	125.45	11.58	1.24	-
108	Sorojini Reddy	2264	66	F	80	183	23.9	10	N	Oral+Insulin	8.15	314	217	500	51	105.09	9.97	1.71	-
109	Siddubai	2394	73	F	79	178	24.9	6	N	Oral+Insulin	8.14	260	185	189	43	82.64	12.14	6.34	-
110	Mandakini Kurudi	2539	70	F	77	161	29.7	6	N	Oral+Insulin	8.39	189	132	63	44	110.63	8.28	1.53	-
111	Ilabai	2785	63	F	58	149	26.1	2	N	Oral	6.21	218	149	74	54	136.74	7.74	2.01	-
112	Maimunisa Sarwadkar	1750	69	F	71	165	26.1	6	N	Oral	8.94	124	61	206	30	107.02	5.51	2.07	-
113	Prema	1752	71	F	80	165	29.4	12	N	Oral+Insulin	9.43	195	130	120	41	111.70	5.68	6.34	-
114	Gangabai Patil	1845	71	F	80	165	29.4	11	N	Oral+Insulin	8.38	211	142	101	49	111.04	7.16	1.5	-
115	Kashibai Hiremath	1992	68	F	56	149	25.2	0.9	N	Oral	6.47	277	225	98	32	127.54	10.68	1.38	-
116	Lalima Sughandi	867	68	F	80	162	30.5	10	N	Oral+Insulin	9.28	211	135	56	65	84.84	5.88	1.95	-
117	Shankuntala Khanpure	878	61	F	65	158	26	4	N	Oral	6.35	208	137	71	57	112.03	10.40	1.53	-
118	Shakuntala Powar	1107	70	F	51	162	19.6	0.8	Y	Oral	6.13	221	154	54	56	132.46	8.07	1.5	-
119	Bhavani Kamble	1421	66	F	80	175	86.1	11	N	Oral+Insulin	8.89	330	256	131	48	101.59	5.55	3.41	-
120	Uma Alagavi	1712	72	F	75	160	29.3	10	N	Oral	7.43	254	200	107	33	119.13	7.67	1.33	-
121	Shailaja Bentoor	763	74	F	51	150	22.2	0.8	Y	Oral	5.79	186	119	122	54	113.40	13.09	2.61	-
122	Bhuvaneshwari	11547	74	F	72	155	30	10	N	Oral	8.44	217	132	123	60	94.72	6.29	1.9	-
123	Ningayya Hiremath	1251	51	M	63	172	21.3	2	Y	Oral	5.64	214	143	158	40	105.17	8.70	3.99	-
124	Girija K	1145	57	F	80	162	30.5	6	N	Oral+Insulin	7.70	263	179	317	46	102.97	8.20	2.71	-
125	Roopa Sarwad	25478	58	F	73	158	29.2	7	N	Oral	8.82	188	108	120	56	132.43	9.55	5.11	-
126	Savita S	14569	54	F	52	160	20.3	1.0	N	Oral	6.51	121	65	51	46	134.04	9.47	5.21	-
127	Basamma	22145	59	F	80	188	22.6	12	N	Oral+Insulin	9.52	251	190	223	27	100.09	9.82	1.52	-
128	Sujata	22456	55	F	79	163	29.7	9	N	Oral+Insulin	9.27	155	94	60	49	89.91	7.77	1.82	-
129	Vanajakshi Patil	36547	51	F	72	176	23.2	8	N	Oral	8.09	210	142	73	53	137.96	10.21	1.19	-
130	Mahadevi Patil	11254	51	F	80	164	29.7	10	N	Oral+Insulin	8.72	211	144	77	43	116.22	9.56	2.5	-
131	Rajeshwari	25413	51	F	70	153	29.9	2	N	Oral	9.16	178	107	99	51	102.65	12.34	1.77	-
132	Preeti	221	55	F	76	160	29.7	7	N	Oral	7.79	170	91	160	48	87.20	7.69	2.09	-
133	Munni Sheik	115	59	F	77	170	26.6	9	N	Oral	8.77	140	80	70	46	127.42	9.21	1.42	-
134	Yallawwa Kirraddi	4436	55	F	60	157	24.3	4	N	Oral	5.93	229	163	120	42	121.03	6.48	6.34	-
135	Jayakka Golasangi	8452	56	F	70	169	24.5	4	N	Oral	9.36	259	188	96	52	109.59	10.46	1.99	-
136	Ambika Salunke	11487	53	F	80	170	27.7	6	N	Oral+Insulin	7.67	194	112	601	31	89.74	13.07	5.7	-
137	Basamma J	6541	55	F	60	148	27.4	1	Y	Oral	5.59	224	144	297	43	123.73	10.48	2.65	-
138	Gangubai	3654	58	F	72	175	23.5	7	N	Oral	9.67	195	122	68	59	132.42	10.76	1	-
139	Dhannamma Bommanahalli	2589	59	F	70	180	21.6	3	N	Oral	8.30	211	142	101	49	81.81	6.87	5.7	-
140	Rukkamma	4569	56	F	67	154	28.3	3	N	Oral	6.36	251	174	190	45	129.18	7.10	1.85	-
141	Suslabai Honnur	7854	53	F	78	172	26.4	9	N	Oral+Insulin	7.79	168	97	208	48	94.55	10.72	5.11	-
142	Jayadevi Kotyal	11547	58	F	66	163	24.8	5	N	Oral	6.57	135	61	376	33	134.47	7.87	1.2	-
143	Basheera Mula	3369	57	F	55	165	20.2	0.8	N	Oral	6.13	210	142	73	53	140.21	9.18	5.7	-
144	Balabai	5478	57	F	71	165	26.1	8	N	Oral	7.78	249	173	81	60	138.83	9.38	2.61	-
145	Preeti Gumaste	21456	57	F	80	160	31.2	10	N	Oral+Insulin	7.12	211	138	329	35	87.68	11.86	1	-
146	Sharadha Hanchinal	22547	51	F	72	175	23.5	7	N	Oral	8.00	237	165	251	37	130.06	7.43	5.7	-
147	Bourawakka	6589	60	F	49	153	20.9	0.9	N	Oral	6.43	259	188	96	52	138.61	9.29	5.21	-
148	Ningawwakka	12569	51	F	78	181	23.8	8	N	Oral+Insulin	8.50	189	118	219	38	104.26	11.79	1.23	-
149	Sheelawwa	145	58	F	80	182	24.2	7	N	Oral+Insulin	7.46	179	104	358	35	106.09	11.96	1.7	-
150	Drupadi	2548	52	F	62	178	19.6	4	Y	Oral	6.02	188	134	80	38	109.97	13.59	1.8	-
151	Bandabai Patil	16911	56	F	39	142	19.3	0.8	Y	Oral	6.10	153	81	80	51	105.06	7.62	8.76	Hyperthyroidism
152	Mynabai	17078	51	F	66	151	28.9	2	N	Oral	5.73	259	188	187	39	137.04	10.47	4.5	Hyperthyroidism
153	Shanta Thotad	17196	54	F	64	155	26.6	3	N	Oral	6.36	194	141	96	34	140.50	6.62	1.72	Subclinical Hyperthyroidism
154	Sharawwa Ganger	15659	55	F	62	170	21.5	3	N	Oral	6.67	246	175	147	42	125.85	12.34	1.03	Subclinical Hypothyroidism
155	Shantawwa Pujari	12934	56	F	70	164	26	1	N	Oral	9.17	195	130	120	41	111.36	6.74	2.03	Subclinical Hypothyroidism
156	Ishwarrappa	11126	53	M	54	148	24.7	0.9	N	Oral	6.11	246	173	233	38	93.23	9.94	5.7	Subclinical Hypothyroidism
157	Chandrakka	12960	52	F	80	178	25.2	10	N	Oral+Insulin	9.58	135	61	376	33	122.32	8.40	2.61	Hyperthyroidism
158	Kalavati	12997	54	F	70	155	29.1	4	N	Oral	7.39	177	107	56	59	111.94	11.51	1.82	Subclinical Hypothyroidism
159	Borawwa	13067	55	F	77	162	29.3	6	N	Oral	9.54	250	167	131	37	123.51	10.46	3.27	Subclinical Hypothyroidism
160	Prakash Kulkarni	11276	55	M	49	151	21.5	0.7	Y	Oral	6.02	177	107	56	59	100.34	8.15	5.21	Subclinical Hyperthyroidism
161	Kamalakka	12937	58	F	80	165	29.4	8	N	Oral+Insulin	7.96	220	149	71	57	85.67	5.81	1.72	Subclinical Hypothyroidism

162	Mahabubi Algur	13076	55	F	64	165	23.5	5	N	Oral	6.54	189	118	219	38	120.93	8.57	1.33	Subclinical Hypothyroidism
163	Shantabai Kalburgi	11142	56	F	80	170	27.7	8	N	Oral+Insulin	7.80	201	130	214	38	102.25	6.96	6.34	Subclinical Hypothyroidism
164	Chanamma Navadagi	11169	59	F	75	160	29.3	7	N	Oral	8.53	125	60	86	48	88.37	11.42	1.72	Subclinical Hypothyroidism
165	Abayawwa Nadageri	11172	57	F	64	147	29.6	3	N	Oral	6.03	168	97	208	48	135.13	6.34	1.85	Subclinical Hypothyroidism
166	Suvarna	11203	54	F	80	167	28.7	6	N	Oral+Insulin	7.49	260	186	203	41	135.99	9.99	1.19	Subclinical Hypothyroidism
167	Mallamma	11300	62	F	72	164	26.8	7	N	Oral	8.40	111	54	103	36	101.76	7.64	2.71	Subclinical Hypothyroidism
168	Gouramma	11551	56	F	61	155	25.4	5	Y	Oral	5.96	202	136	144	37	113.47	7.58	1.98	Subclinical Hyperthyroidism
169	Irappa Hiparagi	11233	59	M	67	170	23.2	5	N	Oral	5.69	170	101	160	38	113.17	10.18	1.91	Subclinical Hypothyroidism
170	Saroja Patil	9882	57	F	77	183	23	7	N	Oral	9.34	214	143	158	40	109.06	6.46	2.02	Subclinical Hypothyroidism
171	Jainbee Girani	10023	57	F	63	150	28	2	N	Oral	6.35	198	132	108	44	126.14	7.35	2.68	Subclinical Hypothyroidism
172	Girija Belagali	10138	53	F	74	175	24.2	8	N	Oral	7.35	224	144	297	43	80.73	10.95	2.68	Subclinical Hypothyroidism
173	Gangabai Hatti	8086	56	F	55	172	18.6	0.7	Y	Oral	6.16	177	89	154	57	114.92	6.46	2.71	Subclinical Hypothyroidism
174	Gireppa Ballappanavar	11282	55	M	80	168	28.3	7	N	Oral+Insulin	8.78	170	101	160	38	139.89	6.68	1.19	Hypothyroidism
175	Ashadevi Rathod	8094	55	F	79	178	24.9	9	N	Oral+Insulin	8.93	205	138	84	50	114.71	10.64	1.99	Hypothyroidism
176	Vidya Kabbin	8211	54	F	80	167	28.7	8	N	Oral+Insulin	9.22	251	174	190	45	126.70	12.48	5.11	Hypothyroidism
177	Bhagirathi	8390	53	F	69	152	29.9	4	N	Oral	5.70	210	139	279	35	101.51	11.95	5.69	Hypothyroidism
178	Kasturibai	8434	53	F	80	172	27	6	N	Oral+Insulin	9.84	174	107	104	46	117.76	7.09	5.11	Subclinical Hypothyroidism
179	Kashibai Hiremath	6566	52	F	78	162	29.7	9	N	Oral+Insulin	7.13	229	161	194	36	108.90	6.63	2.05	Subclinical Hyperthyroidism
180	Gurubai Aimel	6579	56	F	60	168	21.3	5	N	Oral	6.46	168	115	68	39	95.40	9.09	3.3	Subclinical Hypothyroidism
181	Bouramma	6694	58	F	80	165	29.4	10	N	Oral+Insulin	8.31	228	161	168	36	119.32	12.43	5.69	Subclinical Hypothyroidism
182	Krishnappa Wagmore	9702	51	M	75	169	26.3	6	N	Oral	8.90	202	136	144	37	96.79	5.41	5.7	Subclinical Hypothyroidism
183	Kashibai Dalawai	6911	58	F	50	152	21.6	0.6	N	Oral	6.43	211	142	101	49	117.76	9.81	2.64	Subclinical Hypothyroidism
184	Sharanayya Hiremath	3608	51	M	59	178	18.6	4	N	Oral	6.62	276	201	157	44	130.27	12.75	1.73	Subclinical Hypothyroidism
185	Ratnabai Muttagi	6914	51	F	71	189	19.9	6	N	Oral	8.54	181	111	57	59	109.15	5.65	2.5	Hypothyroidism
186	Annapurna Shivasharan	6983	56	F	64	177	20.4	5	N	Oral	5.54	229	161	194	36	89.67	12.85	2.09	Hypothyroidism
187	Neelamma Basanur	4978	51	F	74	165	27.2	7	N	Oral	9.52	229	163	120	42	118.04	8.38	3.99	Hypothyroidism
188	Sundrabai Gadage	3680	57	F	77	161	29.7	9	N	Oral	8.65	198	132	108	44	94.59	8.03	5.11	Hypothyroidism
189	Mahadevi Kumbar	3476	58	F	63	180	19.4	2	Y	Oral	6.70	140	80	70	46	89.90	8.39	1.24	Hyperthyroidism
190	Parvati Gangashetti	3389	53	F	70	158	28	2	N	Oral	8.70	211	135	56	65	107.33	6.89	2.99	Hyperthyroidism
191	Sharanamma Ganiger	3401	53	F	78	168	27.6	6	N	Oral+Insulin	9.74	228	151	172	46	93.27	9.52	6.5	Subclinical Hypothyroidism
192	Kantabai Hugar	3216	51	F	77	180	23.8	9	N	Oral	7.90	175	103	59	56	123.58	8.20	8.76	Subclinical Hypothyroidism
193	Parvati Kannur	3261	57	F	51	162	19.6	0.5	Y	Oral	6.51	250	175	80	59	83.40	9.66	2.11	Subclinical Hypothyroidism
194	Satayya Hiremath	4297	69	M	51	135	28	0.7	N	Oral	6.13	211	125	96	52	125.89	8.41	1.01	Hypothyroidism
195	Bandakka Patil	12401	59	F	79	178	24.9	9	N	Oral+Insulin	7.59	222	154	62	56	108.24	7.23	2.03	Hypothyroidism
196	Lata Kore	4886	59	F	64	153	27.3	2	N	Oral	6.54	228	151	172	46	124.71	12.05	1.04	Hypothyroidism
197	Shantabai Kadani	5443	53	M	80	164	29.7	6	N	Oral+Insulin	9.53	190	114	217	44	135.21	12.32	2.07	Hyperthyroidism
198	Dhamabai	5407	54	F	60	150	26.7	5	Y	Oral	5.68	253	190	74	42	125.35	13.40	1.5	Hyperthyroidism
199	Shanta Soudi	3958	56	F	56	149	25.2	0.6	Y	Oral	6.12	156	101	54	44	110.46	7.20	5.7	Subclinical Hyperthyroidism
200	Siddubai	2394	57	F	66	162	25.1	1	N	Oral	6.69	211	144	77	43	94.19	11.42	5.69	Subclinical Hypothyroidism