ESTIMATION OF THYROID PROFILE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS
A COMPARATIVE CROSS-SECTIONAL STUDY

Dissertation submitted to BLDE (Deemed to be University)

Vijayapura Karnataka.

In partial fulfilment of the requirements for the award of the degree of

DOCTOR OF MEDICINE IN GENERAL MEDICINE

BY DR. ANMOL ATTRI

POST-GRADUATE IN GENERAL MEDICINE

UNDER THE GUIDANCE OF

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DR. ANMOL ATTRI

ABSTRACT

BACKGROUND

Diabetes mellitus and thyroid abnormalities are two most prevalent endocrinopathies. diabetes is one of the fastest growing noncommunicable metabolic syndrome.(11)

Thyroid dysfunction is a significant contributing factor to the poor glycaemic control observed in diabetes. In order to lower the risks of diabetes in the present and the future, it is imperative to improve thyroid function in both diabetic and non-diabetic patients and increase their resistance to the negative effects of thyroid diseases.(11)

Compared to non-diabetics, thyroid disease is more common in those with diabetes. Poor diabetes management is caused by aberrant thyroid hormone levels and abnormalities in thyroid function in diabetic patients. This is demonstrated in certain treated diabetics because it affects insulin resistance.(11)

AIM

To Estimate thyroid profile in Type 2 Diabetes mellitus patients.

OBJECTIVES OF THE STUDY

To determine relationship between type 2 diabetes mellitus and thyroid disorders and comparasion of their prevalance in both sexes.

MATERIALS AND METHODS

SOURCE OF DATA

Patients admitted in the medicine ICU/WARDS OF BLDEUS Shri BM Patil medical college and Research Centre, Vijayapura and who fulfil the inclusion criteria.
All investigations will be performed at the central lab of BLDEU'S Shri M Patil Medical College , Hospital and Research Centre, Vijayapura.

METHOD OF COLLECTION OF DATA

Type of study: COMPARATIVE CROSS-SECTIONAL STUDY DESIGN

Sample size: 84 patient

Using Statulator software for sample size calculation, Asuming the expected population standard deviation to be 11.9, and employing t-distribution to estimate sample size, the study would require a sample size of 84 to estimate a mean with 95% confidence and a precision of 2.6.

CONCLUSION

Serum T3, T4, and TSH levels were noticeably aberrant, and there was inadequate glycaemic regulation, according to our observations and results as well as comparisons with other research of a similar nature. Type 2 diabetics are more prone to have altered thyroid hormone levels, particularly in individuals with poor glycaemic control. If not adequately recognised, these changes in thyroid hormones may play a major role in the inadequate management of diabetes. A greater incidence of hypothyroidism was discovered by our investigation, especially in females. There was shown to be a moderate but substantial association between

TSH and HbA1c. In order to improve medical care and lower morbidity, type 2 diabetic patients must have routine thyroid hormone testing.

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List of Abbreviation

AGE	-	Advanced glycation end-products
DM	-	Diabetes Mellitus
DN	-	Diabetic Nephropathy
ESS	-	Euthyroid Sick Syndrome
HbA1c	-	Glycosylated Hemoglobin
HDL	-	High Density Lipoprotein
HPT	-	Hypothalamic-pituitary-thyroid
LDL	-	Low Density Lipoprotein
TBG	-	Thyroid-binding globulin
T2DM	-	Type 2 Diabetes Mellitus
TFT	-	Thyroid Function Test
TSH	-	Thyroid Stimulating hormone
TRH	-	Thyrotropin-releasing hormone

Introduction

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disease characterized by rising β-cell dysfunction, insulin resistance, and hyperglycemia.

It is one of the most common endocrine disorders, affecting millions of people worldwide. The prevalence of T2DM has been increasing rapidly, particularly in developing countries, due to factors such as urbanization, unhealthy diet, physical inactivity, and obesity. Numerous consequences, such as cardiovascular disease, neuropathy, nephropathy, and retinopathy, are linked to this chronic condition and have a substantial negative influence on the quality of life and death rates of those who are affected.

Another important part of the endocrine system that is critical for controlling growth, development, and metabolism is the thyroid gland. It generates hormones that are essential for controlling a number of physiological functions, such as energy expenditure, protein synthesis, and enzymatic activity. These hormones include thyroxine (T4) and triiodothyronine (T3). [(1)]

Thyroid-stimulating hormone (TSH), which is produced by the pituitary gland in response to signals from the hypothalamus, strictly regulates the release of these chemicals. [(1)]

The interaction between diabetes and thyroid disorders has been a subject of extensive research. It has been established that thyroid hormones influence carbohydrate metabolism and pancreatic function, while insulin and insulin resistance can affect thyroid gland activity. Thus, it should come as no surprise that people with type 2 diabetes have an increased risk of thyroid dysfunction, including hyperthyroidism and hypothyroidism.

Hypothyroidism, characterized by reduced thyroid hormone levels, is more prevalent among diabetic patients and can exacerbate the metabolic challenges associated with T2DM. On the other hand, hyperthyroidism, marked by excessive production of thyroid hormones, can lead to increased insulin resistance and worsened glycemic control.

The bidirectional relationship between T2DM and thyroid disorders underscores the importance of monitoring thyroid function in diabetic patients. Understanding the prevalence and impact of thyroid dysfunction in individuals with T2DM can provide valuable insights for improving clinical management and patient outcomes[2].

Numerous epidemiological studies have investigated the prevalence of thyroid disorders in T2DM patients, with varying results depending on the population and diagnostic criteria used. For instance, studies conducted in different regions have reported prevalence rates of hypothyroidism ranging from 10% to 30% in T2DM patients, while hyperthyroidism has been found in approximately 2% to 5% of these individuals. The higher prevalence of thyroid dysfunction in diabetic patients compared to the general population suggests a possible link between these two conditions.[3]

Several factors may contribute to the observed variations in prevalence rates, including genetic predisposition, environmental influences, and differences in healthcare practices. For example, iodine deficiency or excess, which affects thyroid function, may vary geographically, impacting the prevalence of thyroid disorders. Additionally, the criteria for diagnosing thyroid dysfunction, such as the cut-off values for TSH, T3, and T4 levels, can differ between studies, further contributing to the discrepancies in reported prevalence rates.[4]

Thyroid hormone levels in T2DM patients have been a focal point of research due to their potential impact on diabetes management and outcomes. Studies have shown that T2DM patients often exhibit altered thyroid hormone levels compared to non-diabetic individuals. For instance, some studies have reported higher TSH levels in T2DM patients, indicating a higher prevalence of subclinical hypothyroidism. Elevated TSH levels in diabetic patients may result from insulin resistance and hyperinsulinemia, which can influence the hypothalamic-pituitary-thyroid axis.[5]

On the other hand, abnormalities in T3 and T4 levels have also been observed in T2DM patients. Reduced T3 levels, known as low T3 syndrome, have been linked to poor glycemic control and increased cardiovascular risk in diabetic patients. The relationship between thyroid hormone levels and glycemic control is complex and multifaceted, involving interactions between insulin resistance, inflammation, and metabolic stress.[6]

Thyroid dysfunction can significantly affect the management and progression of T2DM. Hypothyroidism, for example, can exacerbate insulin resistance, making it more challenging to achieve optimal glycemic control. The slowing of metabolic processes in hypothyroidism can lead to weight gain, dyslipidemia, and increased cardiovascular risk, all of which complicate diabetes management. Additionally, hypothyroidism can affect drug metabolism, altering the pharmacokinetics of antidiabetic medications and necessitating adjustments in treatment regimens.(5)

Hyperthyroidism, although less common in T2DM patients, can also have detrimental effects on diabetes management. The hypermetabolic state induced by excess thyroid hormones can lead to increased insulin resistance, hyperglycemia, and a higher risk of diabetic complications. Moreover, the symptoms of hyperthyroidism, such as increased appetite and weight loss, can mask or mimic the clinical presentation of uncontrolled diabetes, complicating diagnosis and treatment.(5)

Given the intertwined pathophysiology of T2DM and thyroid dysfunction, there is a compelling need to systematically evaluate thyroid function in diabetic patients. Previous studies have reported varying prevalence rates of thyroid disorders in T2DM populations, highlighting the necessity for further investigation. Moreover, the clinical implications of thyroid dysfunction in T2DM are profound, affecting glycemic control, lipid metabolism, and overall disease management.(6)

This study aims to estimate the thyroid profile in patients with T2DM, exploring the prevalence of thyroid dysfunction and its association with glycemic control. By doing so, we seek to provide a comprehensive understanding of the interplay between these two endocrine disorders, ultimately contributing to the development of targeted clinical guidelines for the integrated management of T2DM and thyroid dysfunction.(6)

Aim and Objectives

❖ Aim

To Estimate thyroid profile in Type 2 Diabetes mellitus patients.

❖ Objectives

To determine relationship between type 2 diabetes mellitus and thyroid disorders and comparasion of their prevalance in both sexes.

Review of Literature

❖ Thyroid Gland

In the front of the neck, the thyroid gland is a centrally positioned organ. It is an endocrine gland that produces calcitonin and thyroid hormones, which are vital for controlling growth, metabolism, and blood levels of electrolytes like calcium.(7)

A number of illnesses can impact the thyroid, changing how hormones are produced and causing disorders including hyperthyroidism or hypothyroidism.

Conditions that cause inflammation may be related to the thyroid gland.

(such as thyroiditis), autoimmune disorders (like Graves' disease), and cancers (including papillary thyroid carcinoma, medullary thyroid carcinoma, and follicular carcinoma). The

thyroid gland is noteworthy because of its physical location and proximity to critical structures like the parathyroid glands, recurrent laryngeal nerves, and particular blood arteries, in addition to its crucial function in metabolism, growth, and electrolyte balance.[7]

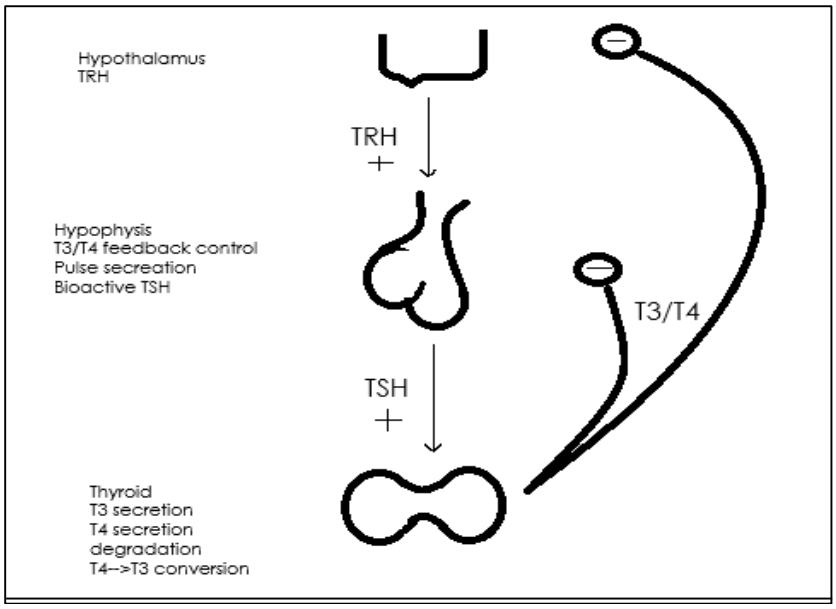


Figure 1: Diagramatic representation of hypothalamic,pituitary,thyroid axis

Anatomy overview

The thyroid gland is made up of two lobes that are joined by an isthmus that runs between the level of the second and third tracheal rings in the upper trachea.

Anatomically, the thyroid gland surrounds the cricoid cartilage and tracheal rings and is situated behind the sternohyoid and sternothyroid muscles.

It normally lines up with vertebral levels C5–T1 and is located beneath the laryngeal thyroid cartilage.[8]

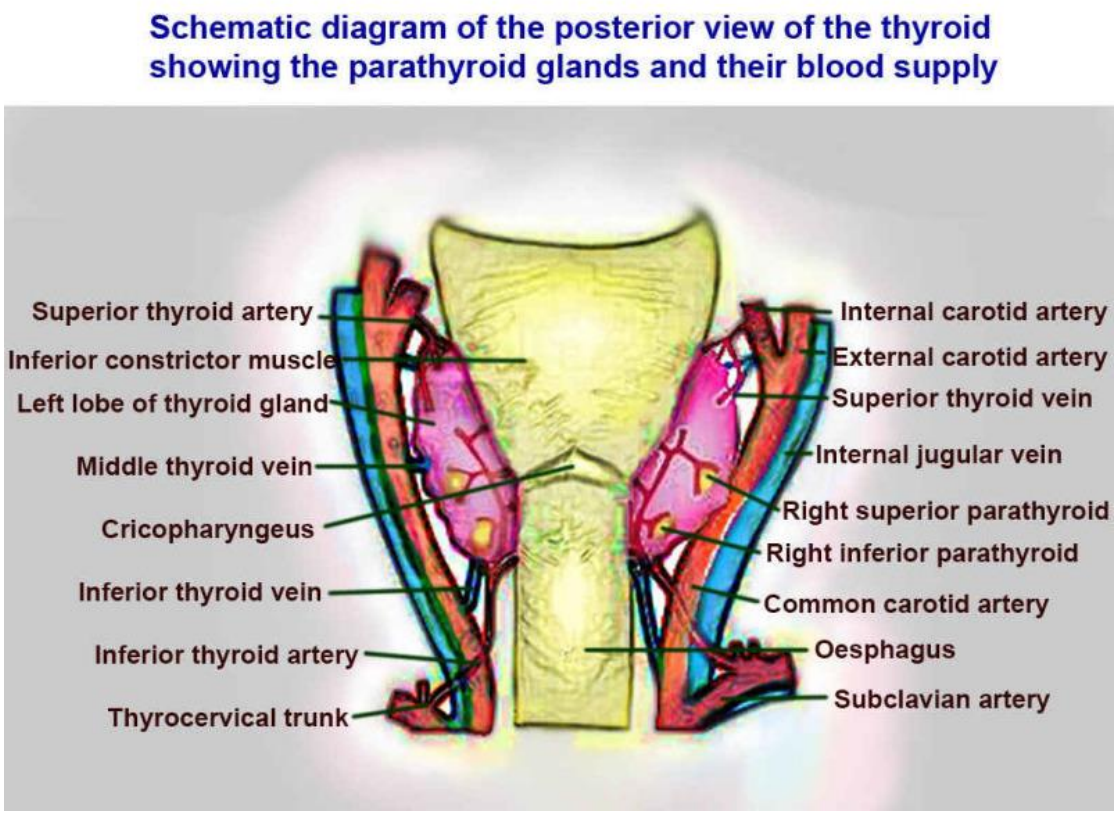
The lateral suspensory ligament, sometimes referred to as Berry's ligament, connects each thyroid lobe to the trachea and connects the thyroid to it.

Pretracheal fascia encloses the thyroid gland, which is housed in the visceral compartment of the neck together with the pharynx, oesophagus, and trachea. A well-defined central isthmus

and symmetric lateral lobes characterise a "normal" thyroid gland.[7]

The tubercle of Zuckerkandl, a pyramidal lobe on the posterior aspect of each lobe, is typically present as well. On the other hand, the thyroid gland may display a variety of morphological variations. The location of the gland and its close proximity to other structures give rise to numerous crucial surgical considerations that carry significant therapeutic implications.[9]

Figure 2 :



Diagramatic representation of thyroid gland

- **Blood Supply and Lymphatics**

  Being three to four times more vascular than the brain and six times more vascular than the kidney, the thyroid gland has an incredibly rich blood supply.[10]

The superior and inferior thyroid arteries, which supply the gland's upper and lower portions, respectively, give blood to it. The superior thyroid artery emerges close to the superior horn of the thyroid cartilage and is the initial outgrowth of the external carotid artery. [10]

After then, it travels behind the sternothyroid muscle in an anterior, inferior, and midline direction until reaching the upper pole of the thyroid lobe, where it branches. The superficial branch supplies the sternohyoid muscle and the thyrohyoid and sternothyroid muscles, whereas the other branch travels along the dorsal side of the thyroid gland.[10]

The isthmus, inner sides of the lateral lobes, and the pyramidal lobe, if existent, are supplied by the superficial branch, which also gives rise to the cricothyroid branch.

The inferior thyroid artery is produced by the thyrocervical trunk, which emerges from the

anterosuperior surface of the subclavian artery. At the inner edge of the anterior scalene muscle, this artery splits off from the thyrocervical trunk and travels medially to the thyroid gland. (10)

It meets the lower third of the outer border and the upper two thirds of the outer border at the posterior surface of the thyroid gland's lateral lobe. The ascending cervical branch, which is the biggest branch of the inferior thyroid artery, is not to be confused with the inferior thyroid artery. (10)

The thyroid ima artery is an extra artery present in 10% of the population. There are several possible sources of its origin, such as the internal thoracic artery, pericardiacophrenic artery, subclavian, brachiocephalic trunk, aortic arch, right common carotid, or transverse scapular. The thyroid ima, which supplies the anterior thyroid gland and isthmus, most frequently arises from the brachiocephalic trunk.(10)

The inferior, middle, and superior thyroid veins drain the thyroid gland. The internal jugular vein on either side of the neck is eventually the destination of the middle and superior thyroid veins, which have a convoluted path.
The inferior thyroid vein can empty into the subclavian or brachiocephalic veins, which are situated directly behind the manubrium. (10)

> The prelaryngeal, pretracheal, paratracheal, and lower deep cervical nodes are all involved in the thyroid gland's lymphatic outflow. In particular, lymphatic drainage from the isthmus and inferior lateral lobes reaches the paratracheal and lower deep cervical nodes, while lymphatic drainage from the superior sections of the thyroid gland reaches the superior pretracheal and cervical nodes. (10)

- **Nerves**

  The thyroid gland is mainly innervated by the autonomic nervous system. The vagus nerve supplies the primary parasympathetic fibers, whereas sympathetic fibers come from the inferior, middle, and superior ganglia of the sympathetic trunk. The autonomic nervous system primarily affects the gland's blood vessels and does not control hormone production or secretion.[11]

- **Physiologic Variant**

  Anywhere along its migration route, ectopic thyroid tissue can be found; reports have indicated that it can be found anywhere from the mouth to the diaphragm. Between one in 100,000 and one in 300,000 individuals have an ectopic thyroid gland; the most common location is an ectopic lingual thyroid at the base of the tongue.[7]

An additional feature of the thyroid gland may be a pyramidal lobe that rises from the isthmus. About 28% to 55% of people have this pyramidal lobe, which is a typical vestigial remnant of the thyroglossal duct and typically originates on the left side of the thyroid. There is evidence that the pyramidal lobe can exist on both sides and can be fully isolated from the main thyroid gland. [7]

Apart from the pyramidal lobe, the thyroid gland exhibits multiple morphological variants. The lateral lobes on the right and left sides may have different sizes and symmetric patterns, while the isthmus may be wide, constricted, or nonexistent. [7]

❖ Thyroid Dysfunction

The term "thyroid dysfunction" refers to a variety of conditions that impact the anatomy or physiology of the thyroid gland, leading to an irregular release of thyroid

hormones. The thyroid gland, situated at the base of the neck, secretes hormones like triiodothyronine (T3) and thyroxine (T4) that are essential for controlling growth, metabolism, and development.[7]

The thyroid gland's normal action is crucial for preserving the body's metabolic equilibrium, and any disruption in this process can have serious health consequences.[7]

❖ **Types of Thyroid Dysfunction**

1. **Hypothyroidism**
    - An underactive thyroid gland that produces insufficient thyroid hormones is a defining feature of hypothyroidism. The most prevalent thyroid condition is this one.
    - **Primary Hypothyroidism**: Caused by intrinsic thyroid gland failure. The most common cause is Hashimoto's thyroiditis, an autoimmune disorder where the body's immune system attacks the thyroid gland. [12]
    - **Secondary Hypothyroidism**: Results from a failure of the pituitary gland to secrete adequate thyroid-stimulating hormone (TSH), which regulates thyroid function. [12]
    - **Tertiary Hypothyroidism**: Caused by a hypothalamic dysfunction that leads to insufficient secretion of thyrotropin-releasing hormone (TRH). [12]
2. **Hyperthyroidism**
    - Hyperthyroidism, or thyrotoxicosis, occurs when the thyroid gland is overactive and produces excessive amounts of thyroid hormones.[13]
    - **Graves' Disease**: The most common cause of hyperthyroidism, it is an autoimmune disorder where antibodies stimulate the thyroid gland to produce too much hormone.
    - **Toxic Multinodular Goiter**: Caused by multiple overactive thyroid nodules.
    - **Thyroiditis**: Inflammation of the thyroid gland, which can lead to temporary hyperthyroidism.[14]

- **Causes of Thyroid Dysfunction**

1. **Autoimmune Diseases**
    - Autoimmune thyroid diseases, such as Hashimoto's thyroiditis and Graves' disease, are leading causes of thyroid dysfunction. Genetic predisposition and environmental factors contribute to these conditions.[15]
2. **Iodine Deficiency or Excess**
    - Iodine is essential for thyroid hormone synthesis. Deficiency can lead to goiter and hypothyroidism, while excess iodine can trigger hyperthyroidism or hypothyroidism.[16]
3. **Medications**
    - Certain medications, such as lithium, amiodarone, and interferons, can affect thyroid function and lead to either hypo- or hyperthyroidism.[17]
4. **Radiation Therapy**
    - Radiation treatment for cancers of the head, neck, or chest can damage the thyroid gland, leading to hypothyroidism.[18]
5. **Thyroid Surgery**
    - Surgical removal of the thyroid gland or parts of it can result in hypothyroidism.[19]
6. **Genetic Mutations**
    - Congenital hypothyroidism can result from genetic mutations affecting thyroid development or hormone synthesis.[20]

❖ **Type 2 Diabetes Mellitus (T2DM)**

Chronic metabolic disease known as type 2 diabetes mellitus (T2DM) is typified by insulin resistance, hyperglycemia, and increasing beta-cell dysfunction.[21]

Unlike type 1 diabetes, which is primarily an autoimmune condition resulting in the destruction of insulin-producing beta cells in the pancreas, T2DM develops due to a combination of genetic and environmental factors

that lead to impaired insulin action and secretion. At about 90–95% of all instances of diabetes worldwide, it is the most prevalent kind of the disease.

The prevalence of T2DM has been increasing at an alarming rate, making it a significant public health concern globally. (21)

The International Diabetes Federation estimates that 463 million persons worldwide had diabetes in 2019, and that figure might increase to 700 million by 2045. The increase in T2DM prevalence is closely linked to rising rates of obesity, physical inactivity, and unhealthy dietary habits, particularly in urbanized and developing regions.(22)

Pathophysiology

The pathophysiology of T2DM involves a complex interplay between genetic predisposition and environmental factors. Two main defects characterize T2DM: insulin resistance and beta-cell dysfunction.(23)

1. **Insulin Resistance**

Insulin resistance is a condition where peripheral tissues, such as muscle, fat, and liver, become less responsive to insulin. Because of this resistance, cells are unable to absorb glucose as well, which raises blood glucose levels. When this compensatory mechanism wears out over time, the pancreas initially produces more insulin, which causes hyperglycemia. (23)

2. **Beta-Cell Dysfunction**

T2DM is characterized by a progressive decrease of beta-cell function. The pancreatic beta cells that manufacture and secrete insulin are triggered by blood glucose levels.

In T2DM, these cells become dysfunctional and cannot secrete sufficient insulin to overcome insulin resistance. This dysfunction is exacerbated by factors such as glucotoxicity (high blood glucose levels) and lipotoxicity (high levels of fatty acids).(24)

- **Risk Factors**

T2DM develops as a result of certain risk factors, such as:

1. **Genetic Factors**

A family history of diabetes increases the risk of developing T2DM. Specific genetic mutations and polymorphisms have been identified that affect beta-cell function and insulin sensitivity.[25]

2. **Obesity and Physical Inactivity**

Excess body fat, particularly visceral fat, is a significant risk factor for T2DM. Obesity induces insulin resistance through mechanisms involving inflammatory cytokines, adipokines, and free fatty acids. Physical inactivity further exacerbates insulin resistance and impairs glucose metabolism. [25]

3. **Diet and Nutrition**

Diets high in refined carbohydrates, sugars, and saturated fats contribute to the development of insulin resistance and T2DM. Conversely, diets rich in fiber, whole grains, and healthy fats are protective against the disease. [25]

4. **Age and Ethnicity**

With advancing age comes an increased risk of type 2 diabetes, especially beyond 45.

T2DM is more common in some ethnic groups, including Native Americans, African Americans, Hispanic Americans, and South Asians. [25]

- **Clinical Manifestations**

T2DM often develops gradually, and individuals may remain asymptomatic for years. Common symptoms include:

- Increased thirst and frequent urination (polyuria)
- Excessive hunger (polyphagia)
- Unexplained weight loss
- Fatigue and weakness
- Blurred vision

- Slow-healing sores or frequent infections

- **Mechanisms Linking Diabetes and Thyroid Dysfunction**

The interplay between diabetes and thyroid dysfunction is complex and multifaceted, involving various physiological, biochemical, and immunological mechanisms. Both Type 2 diabetes mellitus (T2DM) and thyroid disorders significantly impact metabolism, and their co-occurrence can exacerbate complications and alter disease management. Understanding the mechanisms linking these two endocrine disorders is essential for optimizing patient care.[26]

1. **Insulin Resistance and Thyroid Function**

- **Insulin Resistance:** Insulin resistance, a defining feature of type 2 diabetes, is a condition in which the body's cells lose their sensitivity to insulin, raising blood glucose levels. Additionally, this illness affects thyroid function in a number of ways.[26]

- **Thyroid Hormone Production**: Insulin resistance can influence the hypothalamic-pituitary-thyroid (HPT) axis, potentially altering the production of thyroid hormones. Studies have shown that insulin can stimulate the growth and function of thyroid cells, suggesting that hyperinsulinemia (excess insulin in the blood) might affect thyroid hormone synthesis. [26]
- **Thyroid Hormone Metabolism**: Insulin resistance may impair the peripheral conversion of thyroxine (T4) to the more active triiodothyronine (T3), affecting metabolic regulation and potentially leading to altered thyroid hormone levels in diabetic patients. [26]

2. Autoimmune Mechanisms

- **Autoimmune Disorders**: Autoimmune processes can be involved in both type 2 diabetes and several thyroid conditions, including Graves' disease and Hashimoto's thyroiditis. The immune system assaults healthy tissues by mistake under these

circumstances.[15]

- **Common Autoimmune Pathways**: The presence of autoimmune thyroid disease is more common in individuals with Type 1 diabetes, an autoimmune disorder. However, the shared genetic and immunological pathways in autoimmune diseases can also predispose individuals with T2DM to thyroid dysfunction. [15]
- **Cytokine Influence**: Chronic inflammation associated with T2DM involves the release of various cytokines, which can affect thyroid tissue, leading to autoimmune thyroiditis. This can result in hypothyroidism (Hashimoto's thyroiditis) or hyperthyroidism (Graves' disease). [15]

3. Impact of Hyperglycemia

- **Hyperglycemia**: Chronic high blood glucose levels can have direct and indirect effects on thyroid function. [27]
- **Oxidative Stress**: Hyperglycemia induces oxidative stress, which can damage thyroid cells and alter their function. Oxidative stress affects the HPT axis, potentially leading to thyroid hormone imbalances. [27]
- **Glycation of Proteins**: Advanced glycation end-products (AGEs) resulting from prolonged hyperglycemia can affect thyroid cell function and hormone synthesis. [27]

4. Thyroid Hormones and Glucose Metabolism

- **Thyroid Hormones**: Thyroid hormones (T3 and T4) play a crucial role in regulating metabolism, including glucose metabolism. [28]
- **Glucose Absorption and Utilization**: Thyroid hormones enhance glucose absorption in the intestines and facilitate glucose uptake by cells. They also stimulate hepatic gluconeogenesis and glycogenolysis, processes that affect blood glucose levels. [28]

- **Insulin Sensitivity**: Thyroid hormones can influence insulin sensitivity. Hypothyroidism is associated with reduced insulin sensitivity (insulin resistance), while hyperthyroidism can increase insulin sensitivity but also raise glucose production, leading to hyperglycemia. [28]

5. Dyslipidemia and Lipid Metabolism

- **Lipid Metabolism**: Both diabetes and thyroid dysfunction are associated with dyslipidemia, which involves abnormal lipid levels in the blood.[29]
- **Thyroid Hormones and Lipids**: Thyroid hormones regulate lipid metabolism by influencing the synthesis, mobilization, and degradation of lipids. Hypothyroidism is typically associated with elevated levels of cholesterol and triglycerides, contributing to cardiovascular risk. [29]
- **Insulin Resistance and Lipids**: T2DM often involves dyslipidemia characterized by high triglycerides, low HDL cholesterol, and small dense LDL particles. These lipid abnormalities can further complicate thyroid function, particularly in hypothyroid patients, by exacerbating cardiovascular risks. [29]

6. Hormonal Interactions

- **Interactions Between Hormones**: The intricate balance of hormones in the body means that changes in thyroid hormones can affect other hormonal systems, including those regulating glucose.[30]
- **Cortisol and Stress Response**: Thyroid hormones influence the body's stress response, including the production and action of cortisol, which can affect glucose metabolism and insulin sensitivity. [30]
- **Sex Hormones**: Changes in thyroid function can influence levels of sex hormones, which are also involved in glucose and lipid metabolism, further linking the metabolic effects of T2DM and thyroid dysfunction.[30]

7. Clinical Implications and Management

- **Clinical Implications**: The bidirectional relationship between T2DM and thyroid dysfunction necessitates careful monitoring and management of both conditions.[31]

- **Thyroid Screening in Diabetic Patients**: Routine screening for thyroid function in patients with T2DM is recommended, especially in those with poor glycemic control or symptoms suggestive of thyroid disease.[31]
- **Integrated Management**: Treating thyroid dysfunction in diabetic patients can improve metabolic control and reduce complications. For instance, addressing hypothyroidism can enhance insulin sensitivity and improve lipid profiles, while managing hyperthyroidism can stabilize blood glucose levels.[31]
- **Prevalence of Thyroid Disorders in T2DM Patients**

  The prevalence of thyroid disorders among patients with Type 2 Diabetes Mellitus (T2DM) has been extensively studied, revealing a significant association between these two endocrine disorders. Various research findings highlight the elevated occurrence of thyroid dysfunction in individuals with T2DM compared to the general population.[31]

- **Hypothyroidism**

  Hypothyroidism, characterized by insufficient production of thyroid hormones, is notably prevalent in T2DM patients. Several studies have reported a higher incidence of both overt and subclinical hypothyroidism in this population:

  1. **General Population vs. T2DM Patients**: In the general population, the prevalence of hypothyroidism ranges from 4% to 10%, while among T2DM patients, it has been found to range between 10% and 30%. This increased prevalence suggests a strong link between T2DM and hypothyroidism.[32]
  2. **Subclinical Hypothyroidism**: Patients with type 2 diabetes are more likely to have subclinical hypothyroidism which is characterised by high thyroid-stimulating hormone (TSH) levels with normal free thyroxine (T4) levels.[32]

Hyperthyroidism

Hyperthyroidism, a condition characterized by excessive production of thyroid hormones, is less common than hypothyroidism but still occurs more frequently in T2DM patients than in the general population:

1. **Prevalence in T2DM Patients**: The prevalence of hyperthyroidism in T2DM patients ranges from 1% to 5%. Although less prevalent than hypothyroidism, its occurrence is still significant due to the metabolic challenges it imposes on diabetic patients.(33)
2. **Graves' Disease**: Graves' disease is the most common cause of hyperthyroidism and has been observed more frequently in T2DM patients. The autoimmune nature of both T2DM and Graves' disease may contribute to their co-occurrence.(33)

Thyroid Nodules and Cancer

Thyroid nodules and thyroid cancer also show a higher prevalence in T2DM patients:

1. **Thyroid Nodules**: Studies have demonstrated that T2DM patients have an increased risk of developing thyroid nodules. The prevalence of thyroid nodules in T2DM patients can be as high as 50%, compared to approximately 20-30% in the general population.(34)
2. **Thyroid Cancer**: Research suggests that the risk of thyroid cancer is elevated in T2DM patients. A meta-analysis indicated that T2DM patients have a 25% higher risk of developing thyroid cancer compared to non-diabetic individuals.(34)

❖ **Thyroid Hormone Levels in T2DM**

The relationship between thyroid hormone levels and Type 2 Diabetes Mellitus (T2DM) is complex and bidirectional. Thyroid hormones play a crucial role in regulating metabolism, growth, and development, and any abnormalities in these hormones can significantly impact glucose metabolism and insulin sensitivity. Conversely, the metabolic disturbances inherent in T2DM can influence thyroid function and hormone levels. Understanding the patterns of thyroid hormone levels in T2DM patients is essential for effective diagnosis, management, and treatment of both conditions.(35)

- **Thyroid Hormones: An Overview**

Thyroxine (T4) and triiodothyronine (T3) are the two main hormones produced by the thyroid gland. The predominant type of thyroid hormone in the bloodstream is T4, whereas the active form, T3, affects tissue metabolism.(36)

Thyroid-stimulating hormone (TSH), which is produced by the pituitary gland, controls the release of these hormones. Thyrotropin-releasing hormone (TRH) from the hypothalamus in turn controls TSH levels. (36)

- **Thyroxine (T4) :** T4 is converted to T3 in peripheral tissues, a process that is crucial for maintaining normal metabolic functions. (36)
- **Triiodothyronine (T3) :** Almost all physiological processes in the body, including growth and development, metabolism, body temperature, and heart rate, are influenced by T3, which has a greater metabolic impact than T4.(36)
- **Thyroid-Stimulating Hormone (TSH) :** TSH stimulates the thyroid gland to produce T4 and T3. Elevated TSH levels usually indicate hypothyroidism, while suppressed TSH levels suggest hyperthyroidism.(36)

- **Altered Thyroid Hormone Levels in T2DM**

Studies have shown that T2DM patients often exhibit altered thyroid hormone levels compared to non-diabetic individuals. These alterations can manifest as changes in TSH, T4, and T3 levels, reflecting different thyroid dysfunctions such as hypothyroidism, hyperthyroidism, and subclinical thyroid disorders.(36)

1. **Hypothyroidism in T2DM**
    - **Elevated TSH Levels**: Hypothyroidism is characterized by elevated TSH levels due to the decreased production of thyroid hormones (T4 and T3). In T2DM patients, hypothyroidism is commonly observed, with studies indicating a prevalence rate of up to 30%.(36)

- **Reduced T4 and T3 Levels**: Low levels of T4 and T3 are indicative of overt hypothyroidism. Subclinical hypothyroidism, where T4 and T3 levels remain normal but TSH is elevated, is also prevalent in T2DM patients. (36)

2. **Hyperthyroidism in T2DM**
   - **Suppressed TSH Levels**: Hyperthyroidism, though less common than hypothyroidism in T2DM patients, is characterized by suppressed TSH levels due to excessive production of thyroid hormones.(36)
   - **Elevated T4 and T3 Levels**: Increased levels of T4 and T3 are indicative of hyperthyroidism. This condition can exacerbate metabolic disturbances in T2DM patients, leading to poor glycemic control.(36)
3. **Euthyroid Sick Syndrome (ESS)**
   - T2DM patients, especially those with poorly controlled diabetes or severe illness, may experience Euthyroid Sick Syndrome. ESS is characterized by abnormal thyroid hormone levels without intrinsic thyroid disease. Common findings include low T3 levels, normal or low T4 levels, and variable TSH levels. ESS is thought to result from the body's adaptive response to chronic illness and metabolic stress. (36)
4. **Low T3 Syndrome**
   - A subset of T2DM patients may exhibit Low T3 Syndrome, characterized by reduced levels of T3 with normal levels of T4 and TSH. This syndrome is associated with poor glycemic control and increased cardiovascular risk. The decreased conversion of T4 to T3 in peripheral tissues is believed to be a contributing factor. (36)

❖ **Impact of Thyroid Dysfunction on Diabetes Management**

Thyroid dysfunction significantly affects the management of Type 2 Diabetes Mellitus (T2DM) due to the intricate relationship between thyroid hormones and glucose metabolism. Both hypothyroidism and

hyperthyroidism can complicate the management of diabetes, influencing glycemic control, insulin sensitivity, and the risk of complications. Understanding these impacts is crucial for optimizing treatment strategies and improving patient outcomes. (37)

- **Hypothyroidism and Diabetes Management**

Hypothyroidism, characterized by insufficient production of thyroid hormones, is common in patients with T2DM. Its presence can complicate diabetes management in several ways:

1. **Insulin Resistance**

Hypothyroidism is associated with increased insulin resistance. Reduced levels of thyroid hormones slow down metabolism, which can impair insulin action and glucose uptake by cells. This increased insulin resistance can make it more challenging to achieve glycemic control in diabetic patients. (37)

2. **Glycemic Control**

The metabolic slowdown caused by hypothyroidism can lead to higher blood glucose levels and make it difficult to manage diabetes effectively. Patients with coexisting hypothyroidism and T2DM may require higher doses of insulin or oral hypoglycemic agents to maintain target glucose levels. (37)

3. **Lipid Metabolism**

Dyslipidaemia is a prevalent issue among T2DM patients, and hypothyroidism can make it worse. The risk of cardiovascular disease is increased by elevated levels of triglycerides and low-density lipoprotein (LDL) cholesterol. Managing cholesterol levels becomes more difficult in the presence of hypothyroidism and diabetes. (37)

4. **Weight Gain**

Hypothyroidism can lead to weight gain, further worsening insulin resistance and complicating diabetes management. Weight gain in hypothyroid patients is often due to fluid retention and a decrease in basal metabolic rate.[37]

5. **Medication Adjustments**

Treating hypothyroidism with levothyroxine (synthetic T4) can improve insulin sensitivity and glycemic control. However, the initiation or adjustment of thyroid hormone replacement therapy requires careful monitoring of blood glucose levels and potential adjustments in diabetes medications.[37]

- **Hyperthyroidism and Diabetes Management**

Hyperthyroidism, characterized by excessive production of thyroid hormones, also poses challenges in managing T2DM:

1. **Increased Glucose Production**

Hyperthyroidism accelerates metabolism, leading to increased hepatic glucose production and glycogenolysis. This can result in hyperglycemia, making it difficult to maintain glycemic control in diabetic patients.[38]

2. **Insulin Sensitivity**

While hyperthyroidism can increase insulin sensitivity, the overall effect is often overshadowed by the increased glucose production and rapid absorption of carbohydrates from the gastrointestinal tract. The net effect can be poor glycemic control. [38]

3. **Weight Loss**

Hyperthyroidism often leads to weight loss and muscle wasting, which can mask the typical presentation of poorly controlled diabetes. Unintended weight loss in diabetic patients should prompt evaluation for hyperthyroidism. [38]

4. **Cardiovascular Risks**

Hyperthyroidism is associated with increased cardiovascular risks, including atrial fibrillation, hypertension, and heart failure. These risks are particularly concerning in T2DM patients, who are already at a higher risk for cardiovascular disease. (38)

5. **Medication Interactions**

Treating hyperthyroidism typically involves antithyroid medications, radioactive iodine therapy, or surgery. These treatments can interact with diabetes medications and affect blood glucose control. For example, achieving a euthyroid state can alter insulin requirements. (38)

- **Subclinical Thyroid Dysfunction**

Subclinical thyroid dysfunction, characterized by abnormal TSH levels with normal T4 and T3 levels, also affects diabetes management:

1. **Subclinical Hypothyroidism**

Subclinical hypothyroidism, with elevated TSH levels but normal thyroid hormone levels, is common in T2DM patients. It may not cause overt symptoms but can still contribute to insulin resistance and dyslipidemia. Regular monitoring and, in some cases, treatment with levothyroxine may be necessary.(39)

2. **Subclinical Hyperthyroidism**

Subclinical hyperthyroidism, with suppressed TSH levels but normal thyroid hormone levels, can increase the risk of atrial fibrillation and osteoporosis. It may also affect glycemic control, necessitating careful monitoring and management. (39)

❖ **Pathophysiological Mechanisms (Insulin resistance and its impact on thyroid function)**

Insulin resistance, a hallmark of Type 2 Diabetes Mellitus (T2DM), plays a significant role in the development and progression of thyroid dysfunction. The relationship between insulin resistance and thyroid function is complex and bidirectional, with each condition potentially influencing the other through various physiological and biochemical mechanisms.[40]

- **Definition and Mechanism**

Insulin resistance occurs when cells in the body, particularly in the muscles, fat, and liver, become less responsive to the actions of insulin. As a result, higher levels of insulin are required to achieve the desired effects of glucose uptake and metabolism. The pancreas compensates by producing more insulin, leading to a state of hyperinsulinemia. Over time, the beta cells in the pancreas may become unable to sustain this increased insulin production, resulting in elevated blood glucose levels and the onset of T2DM.[41]

- **Contributing Factors**

Several factors contribute to the development of insulin resistance, including:

1. **Genetic Predisposition**: Genetic factors can influence insulin sensitivity and the risk of developing insulin resistance.[40]
2. **Obesity**: Excess body fat, especially visceral fat, produces adipokines and inflammatory cytokines that interfere with insulin signaling. [40]
3. **Physical Inactivity**: Lack of exercise reduces insulin sensitivity and glucose uptake by muscles. [40]
4. **Diet**: Diets high in refined carbohydrates, sugars, and saturated fats contribute to insulin resistance. [40]
5. **Chronic Inflammation**: Low-grade inflammation, often associated with obesity and metabolic syndrome, exacerbates insulin resistance. [40]

Impact of Insulin Resistance on Thyroid Function

- **Thyroid Hormone Metabolism**

1. **Peripheral Conversion of T4 to T3**: Insulin resistance can hinder the thyroid hormone's ability to change from thyroxine (T4) to triiodothyronine (T3), which is more active. This conversion primarily occurs in the liver and kidneys, and insulin

resistance can affect these organs' ability to produce sufficient T3. Consequently, patients may exhibit lower levels of T3, contributing to symptoms of hypothyroidism despite normal T4 levels.[42]

2. **Thyroid Hormone Binding**: Insulin resistance can alter the levels of thyroid hormone-binding proteins, such as thyroid-binding globulin (TBG), transthyretin, and albumin. Changes in the binding capacity of these proteins can affect the availability of free (active) thyroid hormones, potentially leading to thyroid dysfunction.[42]

- **Thyroid Cell Growth and Function**

1. **Hyperinsulinemia**: Elevated insulin levels, a common feature of insulin resistance, can stimulate the proliferation of thyroid cells. Insulin acts as a growth factor, and its excess can lead to thyroid gland enlargement (goiter) and an increased risk of developing thyroid nodules. Hyperinsulinemia has been associated with a higher prevalence of thyroid nodules and thyroid cancer in patients with T2DM.[43]
2. **Insulin Receptors on Thyroid Cells**: Thyroid cells possess insulin receptors, and insulin signaling can influence their function. Insulin resistance may disrupt the normal regulatory mechanisms of thyroid cells, leading to altered thyroid hormone production and secretion.[43]

- **Inflammatory and Metabolic Stress**

1. **Chronic Inflammation**: Insulin resistance is often accompanied by chronic low-grade inflammation, characterized by elevated levels of inflammatory cytokines such as TNF-alpha, IL-6, and CRP. These cytokines can interfere with thyroid hormone synthesis and secretion, contributing to thyroid dysfunction. Inflammation can also affect the hypothalamic-pituitary-thyroid (HPT) axis, leading to alterations in TSH secretion.[44]
2. **Oxidative Stress**: Hyperglycemia and insulin resistance generate oxidative stress, which can damage thyroid cells and impair their function. Oxidative stress affects the thyroid gland's ability to produce hormones and can lead to structural changes within the gland.[44]

- **Clinical Implications**

- **Hypothyroidism**

1. **Increased Insulin Resistance**: Hypothyroidism can exacerbate insulin resistance, creating a vicious cycle that complicates diabetes management. Reduced thyroid hormone levels slow down metabolism, further impairing glucose utilization and increasing blood glucose levels.(36)
2. **Worsening of Metabolic Parameters**: Hypothyroidism can worsen lipid profiles, leading to elevated LDL cholesterol and triglycerides. This exacerbates cardiovascular risk in T2DM patients, necessitating careful management of both conditions.(36)

- **Hyperthyroidism**

1. **Increased Insulin Sensitivity but Poor Glycemic Control**: Hyperthyroidism may increase insulin sensitivity; however, the heightened metabolic rate and increased glucose production can lead to hyperglycemia. Managing blood glucose levels in hyperthyroid patients with T2DM can be challenging due to these opposing
   effects. (36)
2. **Weight Changes**: Weight loss associated with hyperthyroidism can mask the clinical presentation of diabetes, complicating diagnosis and treatment. Unintended weight loss in diabetic patients should prompt evaluation for thyroid dysfunction.(36)

❖ **Role of thyroid hormones in lipid metabolism and cardiovascular risk in T2DM patients**

Thyroid hormones play a critical role in regulating lipid metabolism, and their dysregulation can significantly impact cardiovascular health, particularly in patients with Type 2 Diabetes Mellitus (T2DM). The interplay between thyroid function, lipid metabolism, and cardiovascular risk is complex and multifaceted, necessitating a comprehensive understanding to optimize patient management.(45)

- **Thyroid Hormones and Lipid Metabolism**

Thyroid hormones, primarily thyroxine (T4) and triiodothyronine (T3), influence various aspects of lipid metabolism, including lipid synthesis, mobilization, and degradation. Their effects on lipid metabolism

can have significant implications for patients with T2DM, who are already at an increased risk for dyslipidemia and cardiovascular disease. (45)

1. **Regulation of Lipid Synthesis and Breakdown**
   - **Lipogenesis and Lipolysis**: Thyroid hormones stimulate lipolysis (the breakdown of lipids) and inhibit lipogenesis (the synthesis of lipids) in adipose tissue. T3, the more active thyroid hormone, enhances the expression of enzymes involved in lipolysis, such as hormone-sensitive lipase, and reduces the activity of enzymes involved in lipogenesis, such as acetyl-CoA carboxylase.(46)
   - **Cholesterol Metabolism**: Thyroid hormones promote the conversion of cholesterol to bile acids in the liver, a process essential for cholesterol homeostasis. They increase the expression of LDL receptors on hepatocytes, enhancing the clearance of low-density lipoprotein (LDL) cholesterol from the bloodstream.(47)
2. **Influence on Plasma Lipid Levels**
   - **LDL Cholesterol**: Hypothyroidism is often associated with elevated LDL cholesterol levels due to reduced LDL receptor activity and decreased cholesterol clearance. Hyperthyroidism, on the other hand, can lower LDL cholesterol levels by increasing LDL receptor activity.(5)
   - **High-Density Lipoprotein (HDL) Cholesterol**: The effect of thyroid hormones on HDL cholesterol is more variable. Hyperthyroidism may increase HDL cholesterol levels, while hypothyroidism can either decrease or have no significant effect on HDL levels.(48)
   - **Triglycerides**: Hypothyroidism is associated with elevated triglyceride levels due to impaired lipolysis and increased lipogenesis. Hyperthyroidism generally leads to reduced triglyceride levels.(49)

❖ **Impact on Cardiovascular Risk in T2DM Patients**

The alterations in lipid metabolism caused by thyroid dysfunction can significantly impact cardiovascular risk in T2DM patients. Given that T2DM itself is a major risk factor for cardiovascular disease, the additional burden of thyroid dysfunction can exacerbate this risk.[50]

1. **Hypothyroidism and Cardiovascular Risk**
    - **Atherosclerosis**: Elevated LDL cholesterol and triglycerides in hypothyroidism contribute to the development of atherosclerosis, characterized by the buildup of plaques in arterial walls. This increases the risk of coronary artery disease, myocardial infarction, and stroke. [50]
    - **Hypertension**: Hypothyroidism is associated with diastolic hypertension due to increased systemic vascular resistance. This can further elevate cardiovascular risk in T2DM patients, who are already prone to hypertension. [50]
        - **Cardiac Function**: Hypothyroidism can lead to bradycardia (slow heart rate), reduced cardiac output, and diastolic dysfunction, all of which can impair cardiovascular function. [50]
2. **Hyperthyroidism and Cardiovascular Risk**
    - **Cardiac Output and Arrhythmias**: Hyperthyroidism increases cardiac output and heart rate, leading to conditions such as atrial fibrillation, which is a significant risk factor for stroke and heart failure. The increased metabolic demand on the heart can exacerbate cardiovascular conditions. [50]
    - **Endothelial Dysfunction**: Hyperthyroidism can impair endothelial function, promoting vascular inflammation and atherogenesis, which can heighten cardiovascular risk in T2DM patients. [50]

❖ Previous Literature

Asuti, et al[2023][51] Studied, thyroid disease in people with type 2 diabetes In this survey,there were more women (55.6%) than men (44.4%). Thyroid dysfunction was present in 23.6% of cases (95% CI 0.184 to 0.293). Of the subjects, 27.11% (95% CI 0.163 to 0.402) had overt hypothyroidism, 5.10% (95% CI 0.010 to 0.141) had hyperthyroidism, and 67.79% (95% CI 0.543 to 0.793) had subclinical hypothyroidism. Compared to men (15.4%), women

(84.6%) had a substantially greater prevalence of anti-TPO positive (p=0.013). Thyroid dysfunction is more common in T2DM patients, with subclinical hypothyroidism predominating. Therefore, this study highlights how crucial it is to check TSH levels annually in all T2DM patients. This study aligns with the existing research.

Ahmed Kayode Jimoh et al [2022][52] Seventy-eight patients made up of 56 T2DM and 22 NDM were evaluated in this study. Out of the participants, There were 27 females and 51 males; 59.1% of the females had NDM and 67.9% of the females had T2DM. Participants with T2DM had a substantially higher WHR than those with NDM (0.92 ± 0.05 versus 0.88 ± 0.06). Additionally, compared to NDM, TSH levels were greater in T2DM. Twelve (21.4%) of the T2DM patients had an aberrant biochemical pattern, which included subclinical hyperthyroidism, subclinical hypothyroidism, and euthyroid sick syndrome, whereas forty-four (78.6%) had a euthyroid (normal) biochemical pattern. Furthermore, 30 (53.6%) of the individuals with type 2 diabetes received their diagnosis during the last five years. The length of diabetes did raise TSH levels, but this rise was not statistically significant. There was no overt hypothyroidism or hyperthyroidism among the T2DM individuals.This study aligns with the findings of the current research.

Anveetha et al[2021][53] studied, total of 120 people were studied, with 60 patients with type 2 diabetes mellitus (Cases group) and 60 healthy people (Control group). hyroid abnormalities were significantly more common in diabetic patients compared to healthy individuals, especially among those with poor glycemic control. Diabetic patients had significantly higher mean serum TSH levels and significantly lower mean serum T3 and T4 levels compared to the control group. The abnormal thyroid hormone levels in type 2 diabetics result from changes in the hypothalamic–pituitary–thyroid axis, leading to notable metabolic disturbances, particularly in those with poor glycemic control. Routine thyroid function screening in diabetics can help prevent complications, contributing to better treatment outcomes, reduced morbidity, and improved quality of life. This study supports the conclusions of the current research.

Pangajam P et al[2021][(54)] ,Patients with type 2 diabetes have higher blood levels of triglycerides, LDL cholesterol, and total cholesterol. Furthermore, T2DM patients have significantly decreased T3 and T4 levels and higher TSH levels. TSH and TC, LDL-C, and TGL also significantly positively correlate, whereas T3 and T4 levels significantly negatively correlate with TC, LDL-C, and TGL. Patients with type 2 diabetes have lipid imbalance and thyroid dysfunction. In patients with type 2 diabetes, early and regular screening for thyroid abnormalities can help lower the morbidity linked to dyslipidaemia. This study parallels the findings of the current research.

Abdur Rahim Abidia et al[2020][(55)] ,out of 100 diabetic subjects 41.03% male and 58.97% female were faced with euthyroid and the remaining 22% had thyroid dysfunctions. The prevalence of hypothyroidism was higher in women (78.97%) than in men (21.05%). There was no statistically significant variation in the mean values for TSH, FT3, and FT4 ($p>0.05\%$). Thyroid problems and diabetes, however, had a very distinct connection, suggesting that these two illnesses are interdependent. In the early stages of diabetes, it is advised to assess thyroid hormone status in addition to the diabetic profile. In individuals with uncontrolled diabetes, this method can assist postpone or stop the development of secondary problems and enhance diabetes management techniques. This study is congruent with current study.

Dr Asha Khubchandani et al [2020][(56)] research found that the percentage of diabetics with thyroid dysfunction was higher than that of the control group (24% versus 5%). P value of less than 0.05 indicated statistical significance for this result. The diabetic group had a higher prevalence of subclinical hypothyroidism (12% versus 3%, P value <0.01) than the control group. In a similar vein, the diabetic group had a higher prevalence of hypothyroidism (9%

versus 1%, P value <0.02) than the control group. Thyroid dysfunction was discovered to be highly prevalent in those with type 2 diabetes mellitus. This study aligns with the findings of the current research.

Dave M et al[2019][(57)] studied, 13% of patients with type 2 diabetes mellitus had abnormal thyroid profile. Subclinical hypothyroidism was the most prevalent presentation, occurring in 9.25% of cases. Overt hypothyroidism and subclinical hyperthyroidism were observed in 1.9% and 1.9% of cases, respectively. Males made up 14.3% and females 85.7% of those with abnormal thyroid profiles, a statistically significant difference. According to the study's findings, thyroid dysfunction is prevalent in T2DM patients, with a higher incidence in women than in men. Age, diabetes control, family history, treatment type, and HbA1c levels in diabetic patients did not significantly correlate with thyroid dysfunction.This study supports the conclusions of the current research.

Suresh Nayak B et al[2019][(58)] Random blood sugar and HbA1c values were found to be substantially higher in T2DM participants (group II) as compared to control subjects (group I). When comparing T2DM subjects to controls, TSH levels were considerably higher and T3 levels were significantly lower. TSH and HbA1c have a positive correlation. They came to the conclusion that type 2 diabetics have aberrant thyroid hormone levels. Thus, in order to enhance quality of life and lower morbidity in people with type 2 diabetes, frequent thyroid hormone testing is required. This study parallels the findings of the current research.

Wei Zhao et al[2018][(59)] Researchers found that, compared to those without DN, patients with DN had lower levels of free T3 (FT3) and greater levels of thyroid stimulating hormone (TSH) ($p < 0.01$). In patients with diabetic nephropathy (DN), the prevalence of low FT3 syndrome and subclinical hypothyroidism (SCH) was 20.9% and 10.8%, respectively, greater than in controls and patients without DN ($p < 0.05$). Studies employing Pearson correlation and Spearman rank correlation found positive relationships between TSH and serum creatinine ($r = 0.363$, $p = 0.013$) and between

TSH and the urinary albumin-to-creatinine ratio ($r = 0.337$, $p = 0.004$) in people with DN. Furthermore, there was a statistically significant positive correlation between FT3 and estimated glomerular filtration rate (eGFR) ($r = 0.560$, $p < 0.001$). According to the study, TSH levels were high and FT3 levels were low in T2DM patients with DN. As a result, treating thyroid dysfunction may be a viable treatment approach for DN, and patients with DN require frequent monitoring of their thyroid function. The outcomes of this study are in harmony with the current study.

Chutia, et al [2018][(60)] discovered that, out of 80 diabetes patients, 20 had hypothyroidism, 4 had hyperthyroidism, and 56 had euthyroidism. It was discovered that hypothyroid patients had significantly higher insulin resistance (IR) than euthyroid patients. Among hypothyroid patients, TSH and IR showed a favorable connection ($r = 0.230$), however it was not statistically significant. TSH and IR showed a high negative connection ($r = -0.94933$) in hyperthyroid patients, whereas no link was observed in euthyroid ones. The study came to the conclusion that inadequate management of type 2 diabetes mellitus (DM) may be mostly due to a failure to identify thyroid hormone abnormalities. Thyroid hormone monitoring should therefore be done on a regular basis in type 2 diabetic patients in order to enhance medical care and lower morbidity. This study parallels the findings of the current research.

Materials and Method

Study Design :

This is comparative cross-sectional study

Source of data:

Patients admitted in the medicine ICU/WARDS OF BLDEUS Shri BM Patil medical college and Research Centre, Vijayapura and who fulfil the inclusion criteria.

Study Duration and Place of Study :

The study was conducted period of one and Half year

Sample Size :

Using Statulator software for sample size calculation, Asuming the expected population standard deviation to be 11.9, and employing t-distribution to estimate sample size, the study would require a sample size of 84 to estimate a mean with 95% confidence and a precision of 2.6.

Inclusion and Exclusion Criteria

Inclusion Criteria

Patients with Type2 Diabetes mellitus (age groups 18-80 years)

Exclusion Criteria

- Seriously ill patients
- Adults who are previously diagnosed as type 1 diabetes mellitus
- Specific type of Diabetes Mellitus
- Gestational Diabetes Mellitus
- Known case of thyroid disease
- Known case of pancreatitis

Ethical Committee Approval:

The present study was approved by institutional ethics committee of our tertiary care centre (B.L.D.E.U.'s) committee with letter number (IEC/736/2022-23).

Method

- All 84 patients enrolled in this study undergone detail history regarding duration, severity Family history, type of treatment, compliance, control of glycemic status with co morbid condition like coronary artery disease, hypertension and cerebrovascular accident were noted.
- A thorough general and systemic examination was done. All these patients were investigated for routine investigation including complete blood count, renal function test, liver function test, lipid profile.
- These patients underwent plasma glucose estimation at fasting and 2 hours after meal. Plasma glucose estimation was done by trinder's (Glucose oxidase) method and HbA1C was estimated by HPLC method.
- All these 84 patients underwent thyroid estimation which includes T3, T4, TSH, by Ultrasensitive sandwich chemiluminescent immunoassay with fasting serum sample.

❖ **Statistical Analysis**

- The data obtained were entered in a Microsoft Excel sheet, and statistical analysis were performed using a statistical package for the social sciences (SPSS Version 25).
- Categorical data were presented with frequency and proportion while quantitative data were presented with mean and standard deviation.
- Normality of the data were tested with the help of Shapiro-Wilk test
- For a normally distributed continuous variables between two groups were compared using an Independent t-test, for not normally distributed variables Mann Whitney U test was used.
- Association between two groups were assessed by using the Chi-square test.
- $p<0.05$ will be considered statistically significant. All statistical tests will be performed two-tailed.

Observation and Results

Table 1 : Age distribution among study population

Age	Frequency	Percentage
≤ 40 Years	7	8.3
41 -50 years	14	16.7
51 -60 years	43	51.2
> 60 years	20	23.8
Total	84	100

Majority of the patients were form the age group of 51-60 years followed by > 60 years, 41-50 years and less than or equal to 40 years as shown in above table.

Figure 1 : Bar chart showing Age distribution among study population

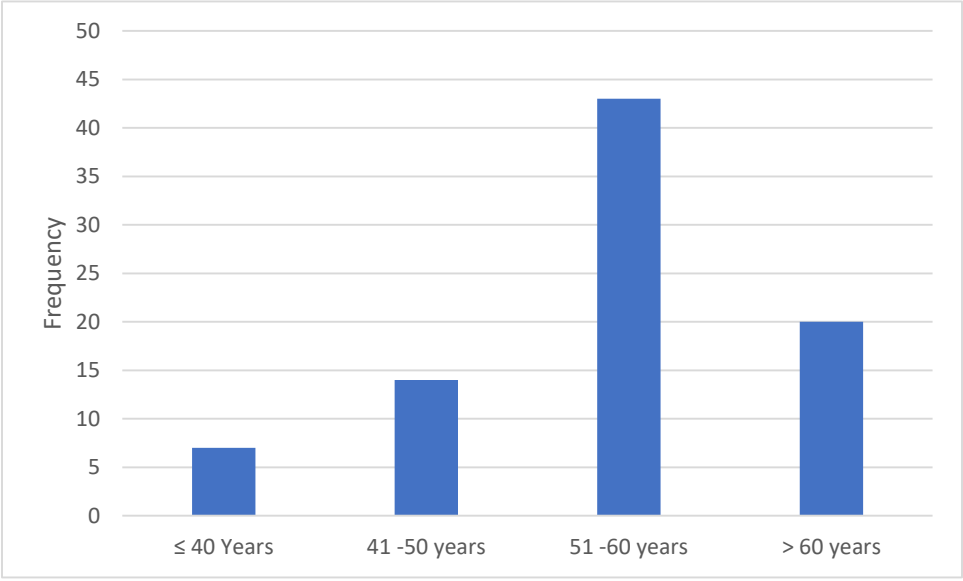


Table 2 : Gender distribution among study population

Gender	Frequency	Percentage
Male	27	32.1
Female	57	67.9
Total	84	100

Majority of the patients were females compared to males, male is to female ratio was 1 : 2.11

Figure 2 :Pie chart showing gender distribution among study population

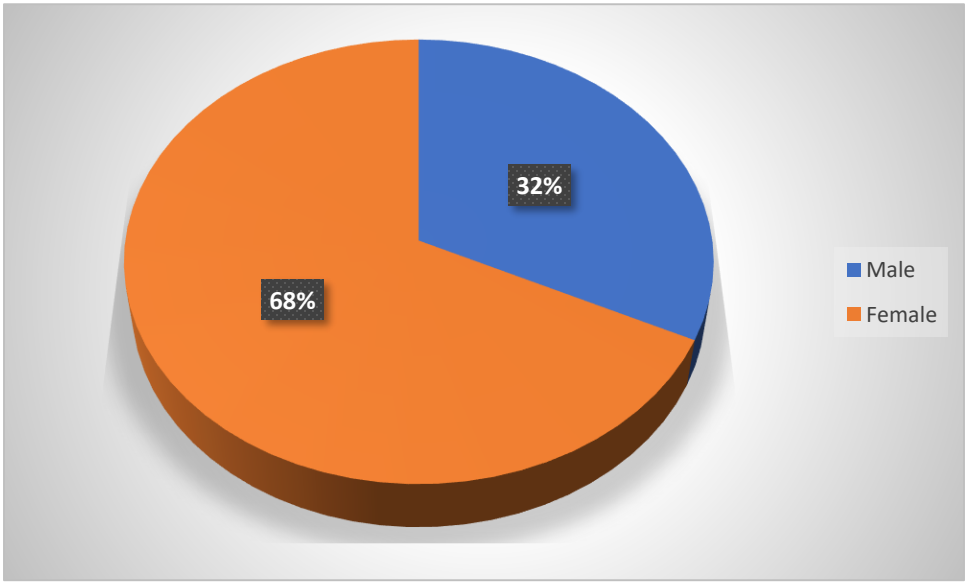


Table 3 : Distribution of types of thyroid disease among study population

Type of Thyroid Disease	Frequency	Percentage
Hypothyroid	25	29.8
Hyperthyroidism	13	15.5
Subclinical Hypothyroidism	7	8.3
Euthyroid	39	46.4

It was observed that, among all patients, 46.4% of the patients were observed with euthyroid, followed by hypothyroid, hyperthyroid and subclinical hypothyroidism as shown in above table.

Table 3 : Pie chart showing distribution of types of thyroid disease among study population

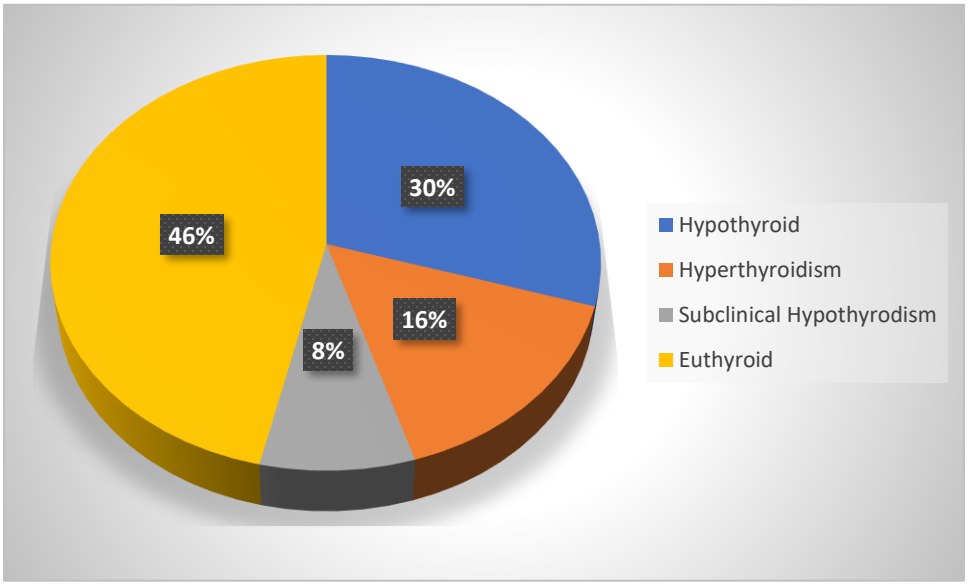


Table 4 : Mean distribution of T3 among different types of thyroid diesease

Type of Thyroid Disease	T3(ng/dl)		F-value	p-value
	Mean	SD		
Hypothyroid	0.28	0.044	94.08	<0.001
Hyperthyroidism	0.8	0.026		
Subclinical Hypothyroidism	0.459	0.11		
Euthyroid	0.47	0.118		

Mean distribution of T3 levels among different type of thyroid disease, were found statistically significant as shown in above table.

Figure 4 : Graph showing mean distribution of T3 among different types of thyroid disease.

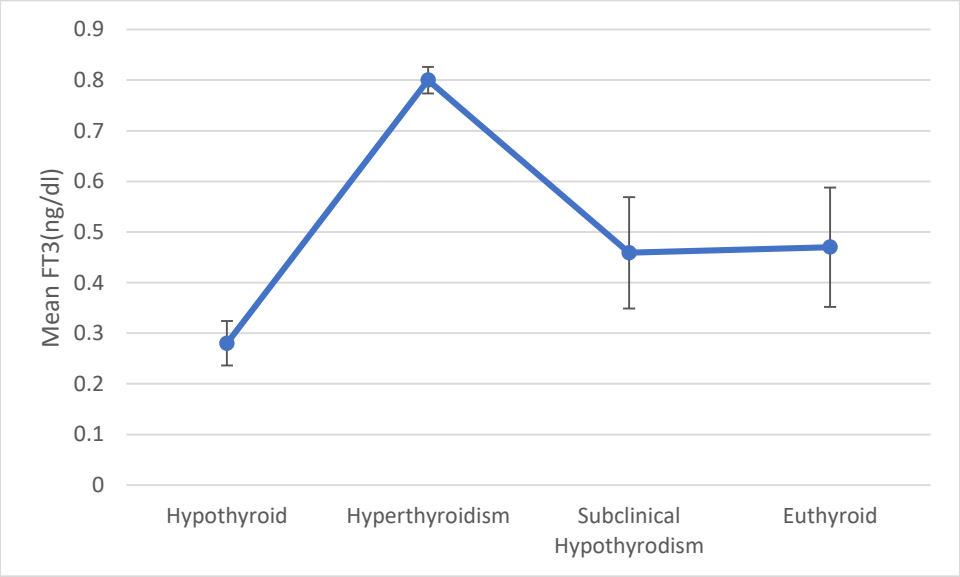


Table 5 : Mean distribution of T4 among different types of thyroid disease

Type of Thyroid Disease	Mean T4(µg/dl)		F-value	p-value
	Mean	SD		
Hypothyroid	0.125	0.074	211.885	<0.001
Hyperthyroidism	1.06	0.029		
Subclinical Hypothyroidism	0.84	0.25		
Euthyroid	0.73	0.133		

Mean distribution of T4 levels among different type of thyroid disease, were found statistically significant as shown in above table.

Figure 5 : Graph showing mean distribution of T4 among different types of thyroid disease.

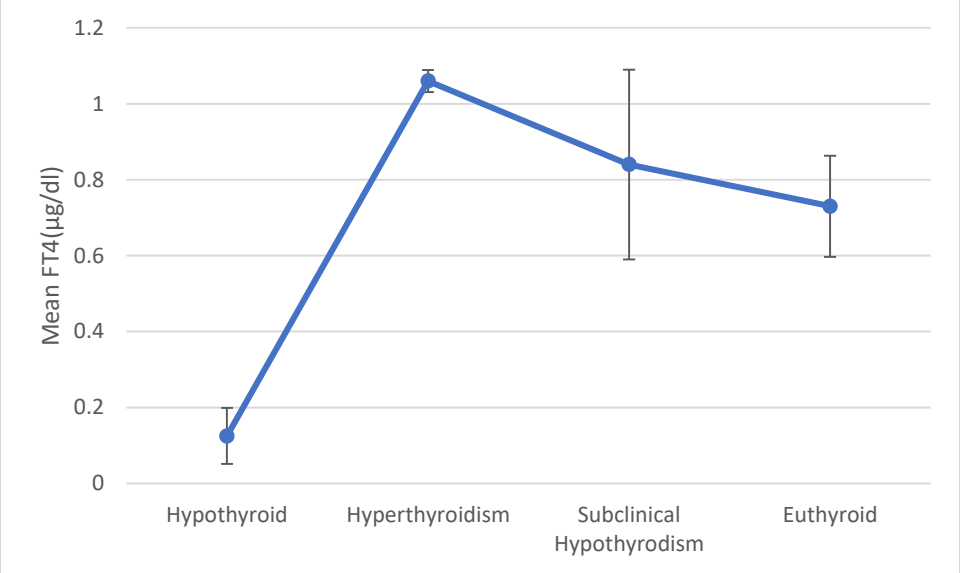


Table 6 : Mean distribution of TSH among different types of thyroid disease

Type of Thyroid Disease	TSH(µIU/l)		F-value	p-value
	Mean	SD		
Hypothyroidism	18.97	3.57	399.145	<0.001
Hyperthyroidism	0.051	0.064		
Subclinical Hypothyroidism	11.64	1.07		
Euthyroid	2.8	0.72		

Mean distribution of TSH levels among different type of thyroid disease, were found statistically significant as shown in above table.

Figure 6 : Graph showing mean distribution of TSH among different types of thyroid disease

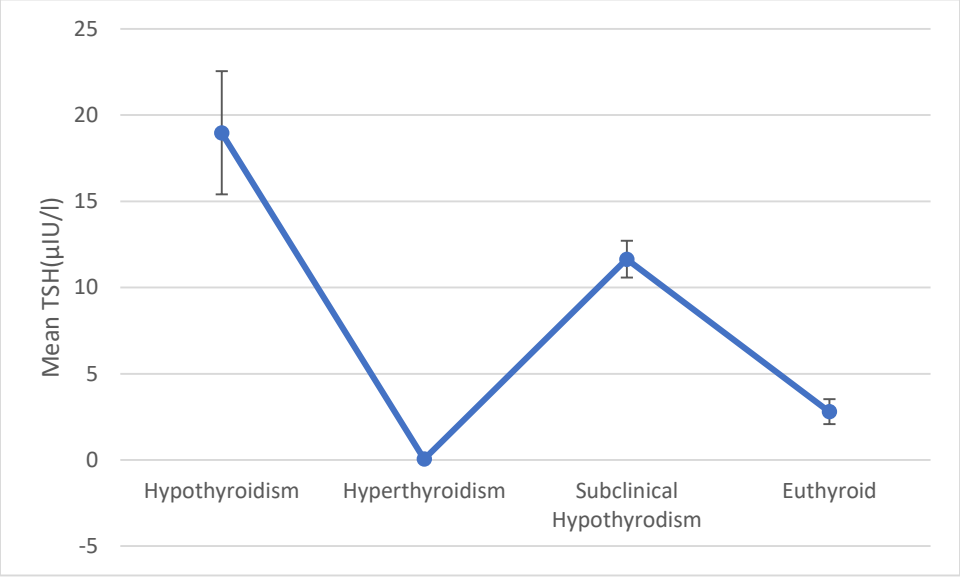


Table 7 : Mean distribution of T3, T4 and TSH between poor control and good control of diabetes

Type of Thyroid Disease	HbA1C		t-value	p-value
	HbA1c < 7.0%(n=33)	HbA1c > 7.0%)(n=51)		
T3(ng/dl)	1.64±0.49	0.98±0.34	7.29	<0.001
T4(µg/dl)	7.97±3.46	5.84±2.17	3.47	0.0008
TSH(µIU/l)	5.42±2.04	11.42±3.64	8.62	<0.001

Mean T3 among patients with HbA1C less than 7% was higher compared to >7%, similarly T4 with HbA1C less than 7% was higher compared to >7%, and mean TSH among the patients with HbA1C > 7% was higher compared to <7%, and this difference between them statistically highly significant.

Figure 7 : Graph showing mean distribution of T3, T4 and TSH between poor control and good control of diabetes.

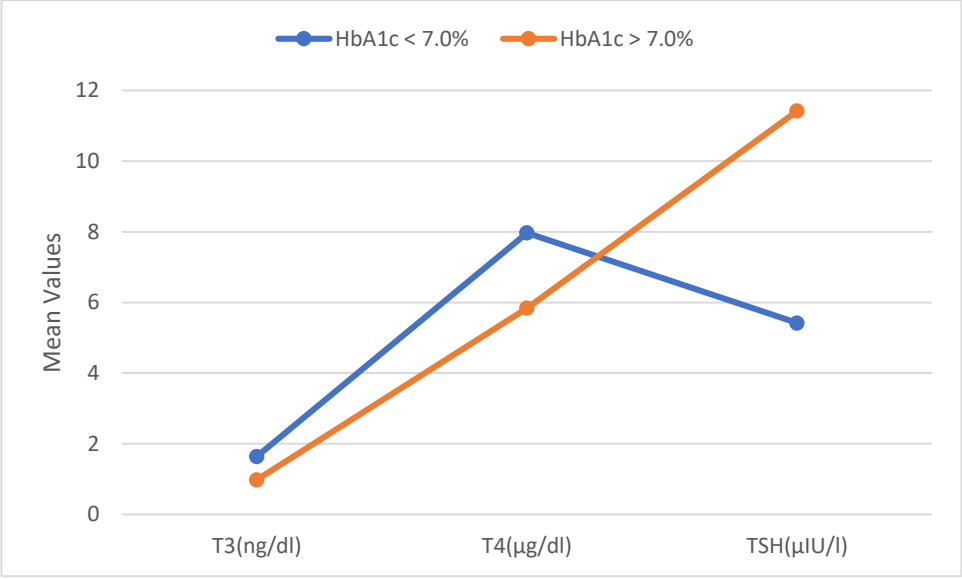


Table 8 : Prevalence of different types of thyroid disorder in gender

Type of Thyroid Disease	Gender	Total		p-value

	Male	Female		Chi-square	
Hypothyroid	7(28%)	18(72%)	25(100%)	0.84	0.359
Hyperthyroidism	4(30.8%)	9(69.2%)	13(100%)	0.013	0.9
Subclinical Hypothyroidism	2(28.6%)	5(71.4%)	7(100%)	0.044	0.832
Euthyroid	14(28.6%)	25(71.4%)	39(100%)	0.47	0.492

It was observed hypothyroid among male was 28%, while among female it was 72%, hyperthyroidism it was more among female, also subclinical hypothyroid more among females, and this difference in the prevalence of different types of hypothyroidism were statistically not significant.

Figure 8 : Bar chart showing prevalence of different types of thyroid disorder in gender

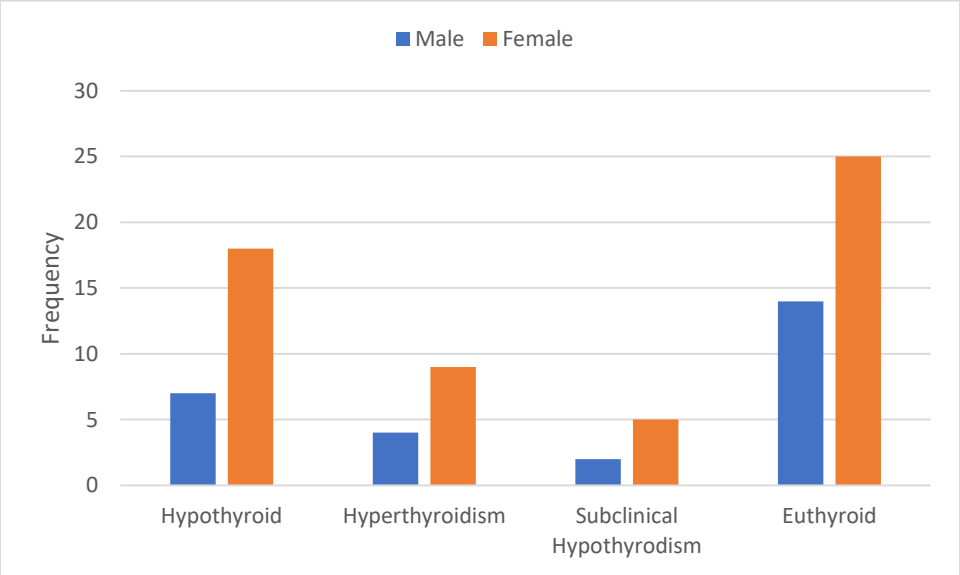


Table 9 : Correlation between HbA1C and thyroid parameters

HbA1C	Thyroid Parameter	R-value	P-value

	T3	0.184	0.132
	T4	-0.004	0.964
	TSH	0.496**	<0.001

**p-value<0.01, highly significant at 1% level of significance.

Correlation between thyroid parameters like T3 and T4 were found not correlated with HbA1C but TSH was significantly moderately positively correlated to HbA1C as shown in above table

Discussion

Among endocrine metabolic diseases, diabetes mellitus (DM) holds the largest share. India notably has the highest number of individuals with diabetes in the world, which contributes to significant mortality and morbidity due to complications.(63) Both DM and thyroid diseases are prevalent endocrinopathies in the population, and there is a mutual influence between insulin and thyroid hormones necessary for normal cellular metabolism.(63) Thyroid hormones control pancreatic and glucose metabolism, and diabetes can have varying effects on thyroid function tests (TFT).Research indicates that diabetes and thyroid disorders frequently coexist in many patients. Thyroid disorders can significantly impact glucose control, and untreated thyroid issues can complicate diabetes management. Therefore, a systematic approach to thyroid testing in diabetic patients is recommended to prevent cardiovascular complications and other diabetes-related complications such as nephropathy and retinopathy.(64) Early detection of abnormal hormone levels, along with other biochemical variables, in diabetes patients can improve health outcomes and reduce morbidity and mortality.

Numerous studies with different designs and objectives were conducted to determine the prevalence and contributing factors of thyroid dysfunction in patients with type 2 diabetes.

The focus of these studies were prevalence of hypothyroidism which has connection with pathophysiology, pathogenesis, co morbidities, and complications of type-2 diabetes mellitus.(65)
Insulin resistance and comparatively low insulin production might coexist to cause type 2 diabetes mellitus (DM). Genetics and factors related to lifestyle are the main causes of this illness. According to earlier research, hypothyroidism is brought on by insulin resistance, which lowers the peripheral muscles' sensitivity to thyroid hormones.

On the other hand, abnormality of thyroid hormones level attributed to insulin resistance, which decrease conversion of T4 to active T3, also reduced hypothalamus thyrotropin releasing hormone (TRH) in DM patients.(66)

Thyroid hormones act as insulin antagonists and also indirectly enhance insulin action. TRH synthesis decreases in diabetes mellitus, which might explain the low thyroid hormone levels observed in some diabetics. In our study, TSH levels were clinically significant in subjects

with type 2 diabetes mellitus compared to healthy non-diabetic subjects. The results indicate that hypothyroidism is frequently observed in individuals with type 2 diabetes mellitus. The results of present study were in accordance with the reports of Vinu vij et al,[67] Gurjeet singh et al,[68] Swamy RM et al,[69] Suzuki et al,[70] Celani et al,[71] Demitrost L et al,[72] Valerie Witting et al,[73] who in separate study found altered thyroid profi0le in a diabetic patient. One major contributing factor to the poor management frequently observed in some treated diabetes patients may be the failure to identify aberrant thyroid hormone levels in diabetics. This study aims to investigate the hyperglycaemic effect by establishing a correlation between thyroid profile parameters and fasting serum glucose, as well as to evaluate the relationship between thyroid dysfunction and the diabetes process. Patients with clinical suspicions of thyroid problems or those experiencing unexpected changes in weight gain, serum cholesterol, or diabetes metabolic control should have thyroid function testing in particular. Improving the management of related co-morbidities can result from treating hypothyroidism.

Diagnosing and treating subclinical hypothyroidism in these patients may significantly enhance their quality of life. Therefore, it is essential to detect cases where hypothyroidism contributes to morbidity and poor control of associated conditions.

- **Demographic Profile**

Demographic parameters like age observed that**,** majority of the patients were form the age group of 51-60 years followed by > 60 years, 41-50 years and less than or equal to 40 years. Male is to female ratio was 1 : 2.11, it showed that, females were more affected by thyroid dysfunction compared to males with T2DM. in the present study It was observed that, among all patients, 46.4% of the patients were observed with euthyroid, followed by hypothyroid, hyperthyroid and subclinical hypothyroidism

Study conducted by Reeta Taksali et al[74] observed that,
females were affected by thyroid dysfunction more than men In addition, diabetic women were more frequently affected than men and hypothyroidism is more than hyperthyroidism. Another study by Dave M et al[57] The results showed that 78 patients (72.2%) were between the ages of 41 and 60, 16 patients (14.8%) were over 61, and 14 patients (13%) were under

the age of 40. 64 patients (59.3%) and 44 patients (40.7%) represented the two sex groups in terms of distribution. Dr. Asha Khubchandani[56] conducted another study in which it was shown that the mean ages of patients with type 2 diabetes mellitus were 36.86 ± 7.21 years and 32.54 ± 6.68 years, respectively, for the control group (P<0.005). Between the two research groups, the sexes were comparable. In patients with type 2 diabetes mellitus, the male-to-female ratio was 56/44, while in healthy controls it was 51/49.

Study by Vaghasiya K et al.[75] observed that, Thyroid dysfunction was present in 14 (28%) of the 50 diabetes patients.Five (10%) had hyperthyroidism, eight (16%) had subclinical hypothyroidism, and one (3%) had hypothyroidism. Another study conducted by

Asuti et al.[51] found that participants' mean age was 54.02 ± 8.7 years, and that 55.6% of them were female. Thyroid dysfunction was present in 23.6% of the participants in this study (95% CI 0.184 to 0.293). Of this group, 67.79% (95% CI 0.543 to 0.793) had subclinical hypothyroidism, 5.1% (95% CI 0.010 to 0.141) had overt hypothyroidism, and 27.11% (95% CI 0.163 to 0.402) had overt hyperthyroidism. Another study by

Khassawneh A H et al.[76], Kumar et al.[77], and Demitrost L et al.[78] who reported the prevalence rates of 26.7%, 24%, and 31.2%, respectively.

Study conducted by Abdur Rahim Abidia et al[55] noted that, in a study including 100 diabetes people, the proportion of thyroid problems in the subjects was assessed. Of these, 22% had thyroid dysfunction while the remaining 78% were euthyroid. In particular, 3% of people had hyperthyroidism and 19% had hypothyroidism. In participants with diabetes, hypothyroidism was more common (19%) than hyperthyroidism (3%).

❖ Mean Values of T3, T4 and TSH

In the present study, we have observed that, mean T3 level was 0.50±0.074 ng/dl, while T4 was observed, 0.68±0.12µg/dl and TSH level was8.36±1.35 µUI/dl. Study conducted by Anveetha et al[53] observed that mean T3 level was 1.08±0.46 ng/dl, while T4 was observed, 6.52±3.14µg/dl and TSH level was 8.52±3.42 µUI/dl. Study by Abdur Rahim Abidia et al[55] **,** observed that, Despite not being statistically significant (p > 0.05), the mean TSH value in diabetic patients was 2.87 ± 2.92 µIU/ml, greater than that of non-

diabetic healthy subjects at 2.08 ± 1.19 µIU/ml. The mean T3 value was 3.87 ± 3.81 pmol/l in diabetic patients and 4.25 ± 0.90 pmol/l in non-diabetic healthy volunteers; however, the difference was not statistically significant ($p > 0.05$). The average FT4 value in patients with diabetes was 17.15 ± 8.98 pmol/l, marginally more than that of healthy non-diabetic persons at 16.35 ± 2.09 pmol/l; however, this difference did not reach statistical significance ($p > 0.05$). Dr Asha Khubchandani et al[56] found that the patients' serum TSH levels (6.2 ± 3.1 mIU/L) were substantially higher than those of the control group (3.1 ± 2.1 mIU/L) ($p < 0.001$). On the other hand, patients with type 2 diabetes mellitus had serum Total T3 (TT3) that was substantially lower (0.4 ± 0.2 ng/ml) than that of healthy people (0.9 ± 0.3 ng/ml) ($p < 0.05$).

Furthermore, there was a significant decrease in serum Total T4 levels (2.4 ± 1.2 µg/dl) between patients with type 2 diabetes mellitus and healthy individuals (6.2 ± 1.1 µg/dl) ($p < 0.05$).

Serum levels of T3, T4, and TSH were generally significantly higher in the diabetic group than in the control group, whereas serum levels of TSH were noticeably lower overall.

Mean Values of T3, T4 and TSH among HbA1c

Present study observed that, Mean T3 among patients with HbA1C less than 7% was higher compared to >7%, similarly T4 with HbA1C less than 7% was higher compared to >7%, and mean TSH among the patients with HbA1C > 7% was higher compared to <7%, and this difference between them statistically highly significant. Anveetha et al[53] observed that, the mean serum T3 and T4 levels in patients with good glycemic control were 1.24 ± 0.32 ng/ml and 7.74 ± 3.14 µg/ml, respectively, whereas in patients without good glycemic control, the mean levels were 0.92 ± 0.28 ng/ml and 5.30 ± 1.84 µg/ml. Serum T3 and T4 levels were considerably lower in those without adequate glycemic control than in those with good glycemic control ($p < 0.001$). The mean serum TSH level was 7.62 ± 1.92 µIU/ml in patients with good glycemic control and 9.42 ± 2.96 µIU/ml in those lacking good glycemic control. Patients with poor glycemic control had significantly higher serum TSH levels than those with adequate glycemic control ($p < 0.001$). Thyroid parameters differed noticeably between cases and controls, with individuals with poor glycemic control exhibiting more severe abnormalities.Other than the pancreas, both endocrine and non-endocrine organs have an impact on diabetes. Patients with diabetes may

occasionally have certain endocrine problems, such as changed thyroid hormone levels. Diabetes patients in this study may have both high and low thyroid hormone levels because of alterations in the synthesis and release of thyroid releasing hormone (TRH), which may be influenced by the glycemic state of the patients.Insulin, which is believed to modulate the levels of TRH and TSH, influences glycemic status.[79-80]

❖ **Mean Values of FT3, FT4 and TSH among HbA1c**

Correlation between thyroid parameters like T3 and T4 were found not correlated with HbA1C but TSH was significantly moderately positively correlated to HbA1C. study by Suresh Nayak B et al[58] observed that, there was positive and moderate correlation between HbA1c and levels of TSH among T2DM also they observed that deceased T3 levels with significantly increased random blood glucose and HbA1c in type 2 diabetics. The level of TSH was significantly elevated in type 2 diabetics. The interaction between thyroid disorders and diabetes mellitus is a complex process. Low T3 state is described as low serum total and free T3 levels but near normal serum T4 and TSH concentrations [81]. Low serum T3 is due to reduced peripheral conversion of T4 to T3 [82]. It is well known that insulin, an anabolic hormone enhances the levels of FT4 while it suppresses the levels of T3 by inhibiting hepatic conversion of T4 to T3 [83]. TRH synthesis decreases in diabetes mellitus and also there is loss of nocturnal TSH peak which is responsible for the occurrences of low thyroid hormone levels in some diabetics. Another study by Abdur Rahim Abidia et al[55], Pearson's correlation was used to find the relationship between HbA1c and thyroid profile. In diabetic patients, HbA1c showed a negative correlation with TSH ($r = -0.055$, $p = 0.585$), which was not significant as the p-value was greater than 0.05. HbA1c also showed a positive correlation with FT3 ($r = 0.165$, $p = 0.101$), but this was not significant since the p-value exceeded 0.05. Additionally, HbA1c was negatively correlated with FT4 ($r = -0.005$, $p = 0.976$), and this correlation was also not significant due to the p-value being greater than 0.05., these results are contradicting our results.

SUMMARY AND CONCLUSION

❖ Summary

Majority of the patients were form the age group of 51-60 years.Male is to female ratio was 1 : 2.11. Among all patients, 46.4% of the patients were observed with euthyroid, followed by hypothyroid (30%), hyperthyroid (16%) and subclinical hypothyroidism (8%). Mean distribution of T3 levels among different type of thyroid disease, were found statistically significant (p-value<0.001). Mean distribution of T4 levels among different type of thyroid disease, were found statistically significant(p-value<0.001). Mean distribution of TSH levels among different type of thyroid disease, were found statistically significant(p-value<0.001). Mean T3, T4 and TSH were statistically highly significant between poor and good control of diabetes (p-value<0.05). Prevalence of different type of thyroid were statistically not significant in gender. There was no statistically significant correlation between HbA1C and T3 and T4 while, TSH is positively significantly correlated with HbA1C(p-value<0.05).

❖ Conclusion

Based on our observations and results, and after comparing with similar studies, we conclude that glycaemic regulation was poor, and there were notable abnormalities in serum T3, T4, and TSH levels. Altered thyroid hormone levels are more common in type 2 diabetics, especially those with poor glycaemic control. These thyroid hormone abnormalities can be a significant factor in the poor management of diabetes if not properly understood. Our study found a higher prevalence of hypothyroidism, particularly among females. A significant and moderate correlation between HbA1c and TSH was observed. Therefore, routine thyroid hormone assays in type 2 diabetic patients are necessary to enhance medical management and reduce morbidity.

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PERFORMA OF CASE TAKING

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DEPARTMENT OF MEDICINE

“ESTIMATION OF THYROID PROFILE IN PATIENTS WITH TYPE 2 DIABETES MELLITUS A COMPARATIVE CROSS-SECTIONAL STUDY”

NAME : **IP NUMBER :**

AGE/SEX : **DOA :**

ADDRESS : **OCCUPATION :**

CHIEF COMPLAINTS :

HISTORY OF PRESENT ILLNESS :

PAST HISTORY :

FAMILY HISTORY :

PERSONAL HISTORY :

1. DIET

2. APPETITE

3. SLEEP

4. BOWEL / BLADDER

5. HABITS

TREATMENT HISTORY :

ON EXAMINATION

PATIENT IS CONSCIOUS-ORIENTED /DROWSY/STUPOR/COMATOSE

VITALS

PULSE RATE : TEMPERATURE :

BLOOD PRESSURE : RESPIRATORY RATE :

PALLOR : YES/ NO. CLUBBING : YES/NO

EDEMA : YES/NO LYMPHADENOPATHY : YES/NO

ICTERUS : YES/NO. CYNOSIS : YES/NO

WEIGHT : KG HEIGHT : M^2 BMI :

SYSTEMIC EXAMINATION:

PER ABDOMEN EXAMINATION:

1.INSPECTION :

2. PALPATION :

3. PERCUSSION :

4. AUSCULTATION :

CARDIOVASCULAR SYSTEM :

RESPIRATORY SYSTEM :

CENTRAL NERVOUS SYSTEM :

PROVISIONAL DIAGNOSIS :

INVESTIGATIONS :

1. COMPLETE BLOOD COUNT :

TOTAL COUNT	MCV- MCH-
HAEMOGLOBIN	
PLATELET COUNT	

2. LIVER FUNCTION TEST :

TOTAL BILIRUBIN	SGOT
DIRECT BILIRUBIN	SGPT
INDIRECT BILIRUBIN	ALP
ALBUMIN	

3. RENAL FUNCTION TEST :

CREATININE	CALCIUM
UREA	PHOSPHORUS
SODIUM	URIC ACID
POTASSIUM	

4. URINE ROUTINE :

PUS CELLS	RBCS
ALBUMIN	SUGAR

5. BLOOD GLUCOSE LEVELS :

FBS	PPBS
HBA1C	

6. THYROID PROFILE :

T3	T4
TSH	

FINAL DIAGNOSIS :

CONSENT

To voluntarily agree to take part in this study I must sign on the line below: If chose to take part in this study I may withdraw at any time I am not giving up any of my legal rights, by signing this form. My signature below indicates that I have read or have read to me this entire consent form including the risks and benefits and had all questions answered, I will be given a copy of this consent form.

Signature or thumb impression of the subject:
Name:
Date:

Signature or thumb impression of authorized representative:
Name:
Relation to the subject:
Date:

Signature or thumb impression of the witness:
Name:
Date:

Signature of the investigator:
Name:
Date:

	NAME	AGE	SEX	IP NO	FBS	PPBS	HBA1C	T3	T4	TSH
1	SARITA RATHOD	39	F	90296	109	205	6.8	0.48	0.76	6.6
2	SAMSUDDIN	51	M	20307	210	250	10.4	0.97	11.09	5.758
3	BASAGOND NINGAPPA	55	M	004477	206	256	10.6	0.66	16.54	0.479
4	BALAPPA SHIVAAPPA	55	M	69135	141	240	7.2	0.39	9.54	1.534
5	RAYAWWA	37	F	312058	94	210	6.9	0.48	6.45	4.289
6	NINGAPPA	70	M	30080	210	245	7.6	0.59	13.03	0.248
7	BASANGOUDA	42	M	60795	145	260	6.1	0.79	8.53	1.719
8	NABHISAHEB	75	M	30727	184	311	10.2	0.78	9.80	2.532
9	BANGERAVVA SINGHE	57	F	85848	53	205	9.7	0.92	16.12	5.859
10	IRASANGAYYA MALEYA	65	M	30009	203	265	7.4	0.74	14.25	3.237
11	NEJAGANI YAMANAVVA	37	M	130615	122	210	10.6	0.90	9.18	0.366
12	RUKAMAYYA	48	F	241202	150	180	8.3	2.72	1.06	0.465
13	RACHAPPA MORTAGI	32	M	90718	150	220	8.2	0.48	11.34	0.803
14	NIZAM KALMANI	51	M	41725	130	248	9.1	0.95	14.11	2.045
15	PYARANABI WALIKAR	54	F	005054	190	245	7.6	0.38	8.88	0.356
16	VIDYA DEEPAK GENNUR	43	F	003372	250	310	9.8	0.41	12.44	16.958
17	SOMLING	56	M	10154	102	210	7	1.03	9.3	2.089
18	ANITA	40	F	51657	172	220	13.1	1.08	16.73	1.526
19	SHAKUNTALA NAYKODI	50	F	40229	151	210	9.3	1.45	0.44	5.376
20	BISMILLA	56	F	276800	32	152	9.5	0.42	11.88	0.863
21	CHANDRASHEKHAR	62	M	281533	115	350	13.7	0.56	12.05	0.671
22	SURESH	68	M	251258	155	157	9.4	0.54	13.31	0.383
23	KHAJABHAI CHANDSAB	57	M	288394	184	301	8.5	0.35	9.58	0.832
24	AMMOGI HIREKURABAR	60	M	282080	105	169	10.9	0.23	10.09	2.329
25	BOURAMALA	60	F	006142	105	310	8.1	0.55	12.16	3.876
26	KORABU SANGANA	63	M	289357	155	148	10.8	0.54	9.14	3.670
27	NARENDRA	65	M	15735	128	246	8.4	0.74	11.86	21.674
28	BHIMANNA CHALAVADI	80	M	158088	147	215	6.7	0.81	13.40	4.190
29	ATARATHBI KOKANI	80	F	71107	72	183	4.3	0.38	10.69	2.487
30	BASAVRAJ	52	M	30173	308	465	12.9	0.73	10.48	1.457
31	SHIVAJI BABURAO	76	M	271206	68	118	6.7	0.78	12.17	3.758
32	RACHAYYA GANACHARI	51	M	31343	131	248	8.3	0.82	12.43	2.884
33	SURESH	72	M	82058	82	250	6.3	0.60	10.38	4.492
34	RAMACHANDRA	81	M	147736	139	230	7.3	0.80	13.39	1.201
35	KALLAPPA	40	M	60005	82	168	6.8	0.36	7.58	4.579
36	IMAMSAB	63	M	70209	135	213	8.7	0.26	3.67	3.163
37	CHANDRASHEKHAR T	62	M	281533	115	350	13.7	0.56	12.05	0.671
38	BHIMARAYA HUDDAR	45	M	231003	158	212	7.5	0.56	11.89	5.045
39	SHANMUKHAYYA ARAKERI	62	M	221728	138	256	8.5	0.55	11.66	3.37
40	MAHEBOOBSAB	62	M	60650	210	500	11.2	0.66	5.39	31.836
41	NANDA SURYAVANSHI	56	F	11476	82	169	12.5	0.36	9.54	3.25
42	SURESH	60	M	260632	92	210	8.1	0.70	8.64	4.899
43	SHIVAPPA CHANDRAKAVA	52	M	170628	190	225	7.2	0.76	9.61	8.048
44	SHARANAPPA	30	M	30056	104	158	7.2	1.05	13.61	2.203

45	SHANTAMMA INGALAGI	70	F	31222	83	210	7	0.77	10.52	1.774
46	HANAMNTH KOLKAR	48	M	41217	232	107	9.7	0.71	14.34	1.674
47	GANGABAI AGASAR	57	F	81456	152	210	7	0.44	3.77	50.860
48	SANTOSH KAMBAR	44	M	51446	175	316	11.5	0.61	13.03	3.288
49	NANAGOUDA CHANNAPA	38	M	60006	492	501	12.0	0.39	10.22	1.799
50	SHIVUBAI VALLAMDESHI	58	F	30606	146	269	12	1.12	8.81	8.570
51	SHARDABAI	57	F	00647	128	251	6.4	0.52	0.77	9.9
52	NOORAND	54	F	346530	136	173	6	0.49	0.74	6.1
53	FAREEDA	50	F	267380	141	273	12	0.49	0.76	9.3
54	LAGAMAWWA	47	F	194823	128	223	5	0.53	0.76	10.6
55	RENUKA	32	F	123641	142	164	5	0.48	0.71	8.2
56	SANGAMMA	65	F	144253	130	178	7	0.5	0.68	6.8
57	GANGAYYA GANACHARI	43	M	50255	141	214	7	0.52	0.67	7
58	TUKARAM	80	M	43388	185	215	6.5	0.53	7.64	1.0533
59	SUMANGALA	68	F	50330	103	328	8.5	0.65	6.93	1.903
60	SHIVALINGAPPA	68	M	34982	140	287	9	0.47	0.6	7.6
61	JINNU	65	M	83694	354	312	8.7	0.38	7.92	1.870
62	RABANABEE	66	F	115319	169	231	9.1	0.35	9.12	0.873
63	GOURAMMA KAMBAR	58	F	139963	162	210	6.9	0.19	4.70	3.664
64	SHRISHAIL	56	M	169814	180	238	7.1	0.80	8.57	0.414
65	PIRAPPA HARIJAN	65	M	179294	138	218	9.1	0.73	9.39	2.183
66	ANIL CHAVAN	63	M	128211	162	185	9.1	0.77	8.08	0.496
67	MEENAKSHI	60	F	134465	128	163	6.5	0.51	0.69	7.7
68	AMBADAS JOSHI	65	M	128620	182	236	6.8	0.79	8.79	2.448
69	MANU LAMANI	66	M	400388	160	246	7.8	0.82	9.75	2.669
70	TOTABAI CHAVAN	62	F	389435	124	190	5.9	0.62	8.02	0.656
71	BASAVRAJ VALAKOTI	63	M	378573	186	235	7.1	0.32	12.61	1.299
72	SIDDAPPA PUJARI	76	M	391320	140	194	13	0.47	0.62	9.1
73	ROSHANTI	70	F	135845	134	190	6	0.51	0.68	8
74	BANUBAI ADESAB	65	F	361283	109	225	7.1	1.79	15.80	1.389
75	SIDDAPPA VITOBA	65	M	77609	397	400	11.1	1.10	10.71	2.482
76	CHANDRASHEKHRA	77	M	375884	302	350	7.9	0.83	10.65	1.698
77	DELIP PARANAKAR	64	M	386369	185	214	6.3	0.32	10.63	1.817
78	BHIMARAY BIRADAR	84	M	401386	142	230	6.3	1.21	11.82	1.840
79	ANASUYAMMA BN	68	F	375946	148	210	10.3	0.57	9.16	1.703
80	GOLAPPA DEGINAL	78	M	391991	182	328	6.4	0.72	11.23	1.296
81	CHANDRAWWA AWATI	65	F	190544	105	180	5.6	0.68	13.29	2.628
82	ESUBAI	79	F	168398	127	155	9	0.48	0.62	7.5
83	SAVITA PAWAR	37	F	147058	144	267	7	0.49	0.79	7.8
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INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Friday, 26th August, 2022 at 3.30 p.m. in the Department of Pharmacology** scrutinizes the Synopsis of Post Graduate Student of BLDE (DU)'s Shri B.M.Patil Medical College Hospital & Research Centre from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "Estimation of Thyroid profile in Patients With Type 2 Diabetes Mellitus – A comparative cross sectional study".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: Dr. Anmol Attri

NAME OF THE GUIDE: Dr.R.C.Bidri, Professor, Dept. of General Medicine

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Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

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