"A STUDY ON THYROID PROFILE STATUS IN TYPE 2 DIABETES MELLITUS"

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the regulations

For the award of the degree of

M.D. BIOCHEMISREY- BRANCH – XIII

CHENNAI MEDICAL COLLEGE AND RESEARCH CENTRE,

IRUNGALUR, TRICHY- 621 105

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

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APRIL - 2017

CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY ON THYROID PROFILE STATUS IN TYPE 2 DIABETES MELLITUS"** is a bonafide original work of **Dr.V.KAYALVIZHI** in partial fulfillment of the requirements for M.D Branch –XIII (Biochemistry) Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in APRIL - 2017. The period of study was from 2014 – 2017.

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The work done by **Dr.V.KAYALVIZHI** on titled "**A STUDY ON THYROID PROFILE STATUS IN TYPE 2 DIABETES MELLITUS"** is under my supervision and I assure that this candidate has abide by the rules of the Ethical Committee.

GUIDE: Dr.Kalavathy Ponniraivan.M.D.,
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DECLARATION

I, **Dr.V. KAYALVIZHI**, solemnly declare that the dissertation titled **"A STUDY ON THYROID PROFILE STATUS IN TYPE 2 DIABETES MELLITUS"** was a bonafide work done by me at Chennai Medical College and Research Centre,Irungalur, Trichy during January 2015 - June 2016 under the guidance of my Professor and Head of the Department Dr.Kalavathy Ponniraivan,M.D.

This dissertation is submitted to TamilNadu Dr.MGR Medical University, towards partial fulfillment of requirement for the award of M.D Degree (Branch-XIII) in Biochemistry.

Place: Irungalur,
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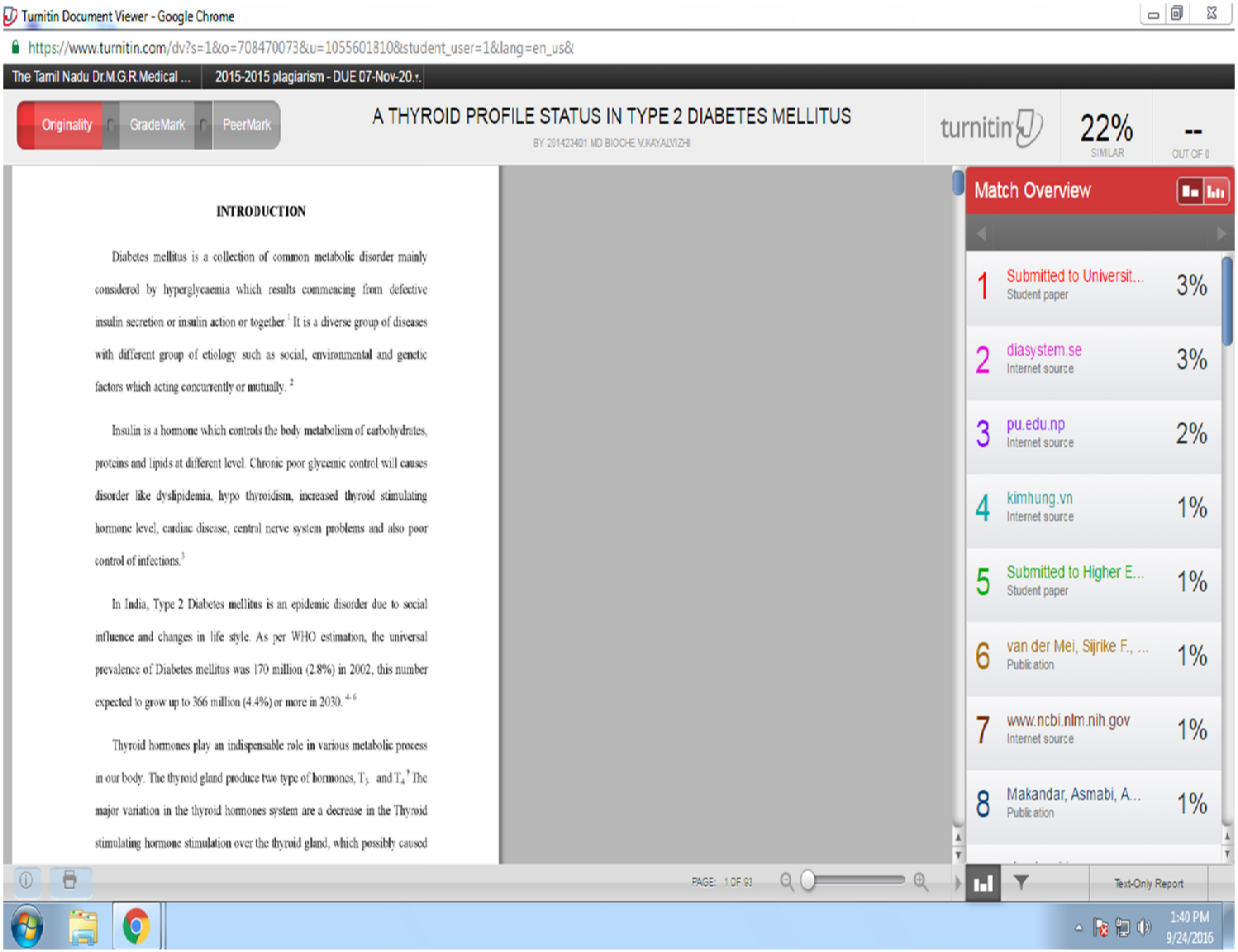
INTRODUCTION

Diabetes mellitus is a collection of common metabolic disorder mainly considered by hyperglycaemia which results commencing from defective insulin secretion or insulin action or together.[1] It is a diverse group of diseases with different group of etiology such as social, environmental and genetic factors which acting concurrently or mutually.[2]

Insulin is a hormone which controls the body metabolism of carbohydrates, proteins and lipids at different level. Chronic poor glycaemic control will causes disorder like dyslipidemia, hypo thyroidism, increased thyroid stimulating hormone level, cardiac disease, central nerve system problems and also poor control of infections.[3]

In India, Type 2 Diabetes mellitus is an epidemic disorder due to social influence and changes in life style. As per WHO estimation, the universal prevalence of Diabetes mellitus was 170 million (2.8%) in 2002, this number expected to grow up to 366 million (4.4%) or more in 2030.[4,5]

Thyroid hormones play an indispensable role in various metabolic process in our body. The thyroid gland produce two type of hormones, T_3 and T_4.[6] The major variation in the thyroid hormone system are a decrease in the Thyroid stimulating hormone stimulation over the thyroid gland, which possibly caused by central hypothyroidism and in the local production of T_3 and T_4.[6,7] These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Successful completion of my study

CONTENTS

ABBREVIATIONS

- ADA – American Diabetic association
- DM– Diabetes Mellitus
- IDF – International Diabetic Federation
- HbA1c – Haemoglobin A1c (Glycated Hemoglobin)
- FT_3 – Free Triiodothyronine
- F T_4 – Free Thyroxine
- TSH – Thyroid Stimulating Hormone
- TRH - Thyrotropin-Releasing Hormone
- GDM – Gestational Diabetic Mellitus
- SCH – Subclinical hypothyroidism
- T.Cholesterol – Total cholesterol
- TGL – Triglycerides
- HDL – High density lipoprotein
- LDL – Low density lipoprotein
- VLDL – Very Low Density Lipoprotein
- WHO - World Health Organization

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In addition, they have a crucial role in maintaining cellular homeostasis; when Thyroid hormones levels in the body are out of balance, they can cause multiple disorders, which include diabetes mellitus, cardiovascular disease, and chronic liver disease.[12, 13]

Thyroid diseases in Diabetes mellitus patients are regularly encountered. The clinical relationships between them are more commonly recognized with hypothyroidism among Diabetes patients. Thyroid hormones are insulin antagonists.Iodothyronines with high levels act as diabetogenics while low levels of iodothyronines inhibits the development of Diabetes.[14, 15]

Diabetes and thyroid disease are the generally two common and important endocrine disorders seen within adults population. Excess or deficiency of either insulin or thyroid hormones can result in functional abnormalities of one another, as both of them are closely involved in cellular metabolism.[16]

Glycemic control is influenced by Thyroid hormones through a range of actions on intermediary metabolism. Hyperglycaemia is promoted by Excess thyroid hormones levels through facilitating glucose intestinal absorption, enhancing glycogenolysis and Gluconeogenesis, and increasing insulin clearance.[17,18] In diabetes patients, hypothyroidism may control the glucose metabolism by various levels. These effects consist of decreases in hepatic glucose production, Gluconeogenesis and increased peripheral glucose consumption. The general effects of these processes are a progress to

hypoglycaemia. Recurrent hypoglycaemic attacks were recognized in children and adolescents who have diabetes and subclinical hypothyroidism.[19, 20]

A numerous studies were reported the occurrence of thyroid disease in diabetes patients changing from 2.2% to 17 %. [21, 22] However few studies shows even higher up to 46.5% [21, 23.] The relationship between type 1 DM and thyroid dysfunctions are proved one and may be an autoimmune process. [24] But in India, inadequate data is available on thyroid diseases in type 2 diabetes patients. So our study designed to evaluate incidence of thyroid dysfunction among type 2 diabetes mellitus subjects residing in south Indian region.

With these background, the present study aims to focus on to find out the prevalence of thyroid dysfunction in Type 2 DM population. An effort was made to compare and correlate these two metabolic disorders by taking into consideration of various biochemical parameters.

AIM:

Our research focused on to investigate the relationship between diabetes mellitus and thyroid profile status in Type 2 Diabetes mellitus patients.

OBJECTIVES

1. To study the correlation between Diabetes mellitus and Thyroid dysfunction by estimating F T_3, F T_4 and TSH in type 2 diabetic subjects.
2. To estimate the thyroid hormones level in Type2 Diabetes mellitus.
3. To study the relationship between various parameters like lipid profile, renal profile in various status of thyroid dysfunction in Diabetic mellitus patients.
4. To determine the prevalence and degree of various Thyroid dysfunction between type 2 diabetes mellitus patients.

REVIEW OF LITERATURE

DIABETES MELLITUS:

Diabetes is a major health problem in the world. It produces serious health - related and socioeconomic impact on individual person and also on populations. In addition, the pandemic increase of diabetes is spurred on by transitioning demographic like Population aging, socioeconomic, nutritional and Lifestyle patterns and migratory cause and a joined proliferation in overweight and obese adults and in children. [25, 26]

Diabetes is a common endocrine metabolic disorder. It is characterized by increased glucose level from a multiple interaction of hereditary and environmental factors due to decreased insulin secretions or resistance or both.[27]

HISTORY:

Polyuric diseases have been described for over 3500 years. The Hindu physicians, Charak and Sushrut, who wrote between 400 and 500 bc were probably the first to recognize the sweetness of diabetic urine .The word "diabetes" came from the Greek word meant for a syphon; and the sweet taste of diabetic urine was documented at beginning of the first millennium, but the adjective "mellitus " (honeyed) was further added by Rollo in the late 18th century., British physiologist Matthew Dobson was the first person, who showed in his Experiments that the sweet-tasting substance in the urine of

diabetic patients was sugar in 1776. The British Army surgeon John Rollo (1749–1809), further added the term "mellitus" (Greek word- meant for honey) to "diabetes" to differentiate it from diabetes insipidus. [25, 27]

PREVALENCE:

Diabetes is a major health problem affecting large population worldwide .WHO projected that the total figure of people among DM has risen from 108 million in 1980 to 422 million in 2014. The general predominance of DM in adults above 18 yrs of age was since 4.7% in 1980 which increased to 8.5% in 2014. It increases with age and approximately half of the cases are occur in people older than 55 years. [28, 74]

As per the International Diabetic Federation (IDF) 2015 , INDIA is one of the 6 main countries of the IDF SOUTH EAST ASIA (SEA) region.415 million people have diabetes in the globe and 78 million people in the SEA region. By 2040 this will get higher to 140 million in SEA region. In India, there were 69.1 million cases of DM in 2015 with prevalence of 8.7% of adult population (20-79 years). Most of the diabetics live in underdeveloped and developing countries (up to 80%). [29]

The prevalence of Type 2 DM is increasing rapidly due to reduced activity because of more industrialization.[30, 31] Many factors such as dietary habits, sedentary life style, ethnicity, obesity and hypertension and genetic predisposition to the disease are the major causes to this epidemic. [32].Uncontrolled DM is the major cause of micro and macro vascular complications like blindness, kidney failure, heart attacks, stroke, and lower

FIGURE 1

WORLDWIDE PREVALENCE OF DIABETES MELITUS[30]

Worldwide prevalence of diabetes mellitus. Global estimate is 382 million individuals with diabetes. Regional estimates of the number of individuals with diabetes (20–79 yearsof age) are shown (2013).

IDF Diabetes Atlas, the International Diabetes Federation, 2013.

limb amputation. Because of these long term complications, there are increased mortality and morbidity among diabetic subjects. As per WHO in 2012, 1.5 million deaths were directly due to DM and another 2.2 million deaths were related to high blood glucose.[33]

In absolute numbers, India will continue to be the country with the most individuals living with diabetes, projected in 2030 to have nearly 80 million people with diabetes.[34] The greatest increases in diabetes prevalence will be in India. Diabetes is the common heterogeneous endocrine disorder rising up to approximately 20% in urban and 10% in rural population. [35]

CLASSIFICATION OF DIABETES MELLITUS:

DM is classified based on the pathogenesis of hyperglycemia. The American diabetes association (ADA) classified DM as type 1 DM , Type 2 DM and other specific types of diabetic whi,ch include MODY , Endocrinopathies, IGT & IFG and GDM others.[36]

Type 2 DM:

PATHOGENESIS:

The Type-2 diabetes is accounts for just about 90% of all cases of diabetes. It is a heterogeneous, complex, interrelated disease involving multiple etiologies.[37] It is characterized through a combination of both insulin resistance and progressive beta cell worsening leads to altered insulin secretion and release, increased hepatic glucose synthesis as the outcome of increased glycogenolysis and gluconeogenesis .[38] There are two major specific

pathological deficiency reported in patients with type 2 DM one is decreased biological action of insulin on peripheral tissues, this is insulin resistance. The other one is beta cell dysfunction, which is lack of ability of the pancreas to turn out sufficient insulin to compensate insulin resistance. [39]

T2DM patients presenting with a few symptoms and not prone for ketosis because they are independent on insulin to avoid ketonuria. Obesity is more commonly associated with this type and weight reduction alone usually improves hyperglycaemia in these patients. Most people acquire this disease after the 40 years of age but it may also develop in younger people. Type2 DM in children and adolescent is an emerging, significant health problem. [40]

The overall pathological features of type 2 diabetes include increased absorption of intestinal glucose, reduced insulin secretion and obvious changes in the beta cells mass which include insulin degradation and enhanced catecholamine.[41]

THYRIOD H0RMONES:

Thyroid hormone plays an important role in various metabolic processes like carbohydrate, lipid metabolism and pancreatic functions. Alteration of thyroid hormone levels directly affects the basal metabolic rate. [42]

PREVALENCE:

The prevalence of the thyroid disease in general population has a great variability varying from 6.6% to 13.4%.[43, 44] This difference may be due to difference in diagnostic criteria of thyroid disease, degree of iodine intake among various regions, difference in sensitivities of the TSH assays and the

large population diversity.[45] Thyroid dysfunction is more common in female than male and this may be due to inhibition of disease activity by androgens and also exacerbation by estrogens.[46]

THYROID GLAND ANATOMY AND PHYSIOLOGY:

The thyroid is the largest endocrine gland, weighing approximately 20 gm. It is butterfly shaped and placed over the front of the neck. [47] The thyroid gland has two important physiological endocrine systems. Among this, the 1st system, which has most of the thyroid, is accountable for the making of the thyroid hormones triiodothyronine (T_3) and thyroxine (T_4). The 2nd endocrine cell system is responsible for the production of the peptide hormone Calcitonin. Thyroid hormones secretion is controlled by Thyroid stimulating hormone (TSH) from the anterior pituitary gland which is upregulated by thyrotropin regulating hormone (TRH) from hypothalamus. There is a fine control of hypothalamus- pituitary-Thyroid axis. [47]

FUNCTION OF THYROID:

Thyroid hormones has influence on numerous body systems which include growth & development, muscular function, cardiovascular system, sympathetic nervous system, and carbohydrate metabolism.[48]

Thyroid hormones are important for maturation and differentiation during development. They have metabolic functions to control the basic metabolic

FIGURE 2

HOMEOSTASIS IN THE HYPOTHALAMUS-PITUITARY - THYROID AXIS[84]

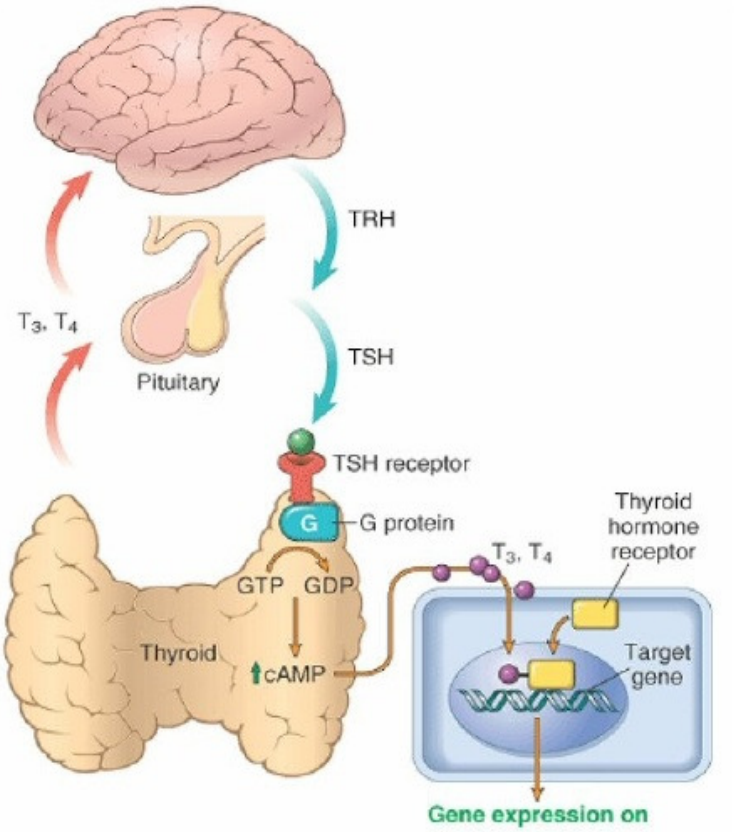


Figure 24-7 Homeostasis in the hypothalamus-pituitary-thyroid axis and mechanism of action of thyroid hormones. Secretion of thyroid hormones (T3 and T4) is controlled by trophic factors secreted by both the hypothalamus and the anterior pituitary. Decreased levels of T3 and T4 stimulate the release of thyrotropin-releasing hormone (TRH) from the hypothalamus and thyroid-stimulating hormone (TSH) from the anterior pituitary, causing T3 and T4 levels to rise. Elevated T3 and T4 levels, in turn, suppress the secretion of both TRH and TSH. This relationship is termed a negative-feedback loop. TSH binds to the TSH receptor on the thyroid follicular epithelium, which causes activation of G proteins, and cyclic AMP (cAMP)-mediated synthesis and release of thyroid hormones (T3 and T4). In the periphery, T3 and T4 interact with the thyroid hormone receptor (TR) to form a hormone-receptor complex that translocates to the nucleus and binds to so-called thyroid response elements (TREs) on target genes initiating transcription.

rate. It influences the metabolic pathways and it has either catabolic or anabolic action.[49]

There are 2 main mode of action in the body: 1. Increase in the overall metabolism 2. Promote growth of children. Some of the mechanisms of actions of thyroid hormones are

1. Increase protein synthesis 2. Increase the amount and activity of enzyme the system 3. Increase the volume and number of mitochondria 4. Their effect on the active transport of ions.[50, 51.]

The common thyroid problems involve abnormal production of thyroid hormones either increased production which leads to hyperthyroidism or insufficient production leads to hypothyroidism.

The causes for the above said statement is due to dysfunction of [52]

1. Thyroid hormone synthesis
2. Thyroid hormone transport and metabolism
3. TSH action
4. Thyroid Hormone binding protein.

THYRIOD AND DIABETES:

Both diabetes and Thyroid diseases are common endocrinopathies. There is a deep relationship between them. Thyroid hormones affect the carbohydrate metabolism and pancreatic function. At the same time, diabetes affects the thyroid function to a large extent. Thyroid dysfunction is much common in diabetic population compared to non-diabetic population. The relationship between DM and thyroid disorder is a complex interdependent

interaction. [53] This relationship was first investigated by coller and huggins in 1927.they proved the worsening of DM in hyperthyroidism. [56]

Prevalence of thyroid dysfunction is higher in diabetics than in normal population, estimation varying from 2.2% to 15 % [67] in DM compared to 6% in non DM population. Nevertheless, many studies have found out even higher prevalence of thyroid disease in diabetics, which was 31% and 46.5% respectively. [54, 23, 14]

The case control study by Vinu Vji et al, found the prevalence of thyroid disease among type2 DM was to be 28.75%. Udiong et al study found a higher incidence of abnormal thyroid hormone levels up to(46.5%) among diabetics in Nigeria (Hypothyroidism was 26% and hyperthyroidism was19.9%). Gurjeet Singh et al conducted study in Punjabi population comprising of 80 DM and 80 controls, found that 30% of patients had abnormal thyroid hormone levels and the TSH level was significantly higher.

EFFECTS OF THYROID HORMONES OVER DM:

Thyroid hormones are insulin antagonists, because of both insulin resistance and beta cell dysfunction are inversely related to thyroid stimulating hormone. [55] Whenever TSH increased, thyroid hormones were decreased and also antagonistic role of insulin are weakened.[57] Both are involved in cellular metabolism, excess and deficit of any one can result in functional derangement of the other.[21]

Thyroid hormones have direct control on insulin secretion. Both hyper and hypothyroidism, have a direct relation to insulin resistance.[58] There is a reduction of insulin secretions by the beta cells. In hyperthyroidism glucose induced insulin secretion is reduced and increased in hypothyroidism.and it has increased susceptibility to hypoglycemia, that's why complicating DM management. It also influences a diversity of abnormalities in blood lipid metabolism which include increased serum triglyceride, low-density lipoprotein (LDL) and total cholesterol concentration[59]. Subclinical hypothyroidism can also exacerbate the coexisting dyslipidemia which is frequently found in type 2diabetes and further increase the possibility of cardiovascular diseases. Sufficient thyroid hormone replacement therapy will repeal the lipid abnormalities.[60, 83]

The relationship between cholesterol and TSH was customized via insulin resistance. So, elevated serum TSH level and comparative insulin resistance are the furthermost risk for the development of increased dyslipidemia.[65]

Palma et al concluded the necessary for screening of DM patients for thyroid disease to reduce the possibility of aggravation of common risk factors like hypertension and dyslipidemia.

RELATIONSHIP BETWEEN THYROID DISORDER AND GLYCEMIC STATUS [69]

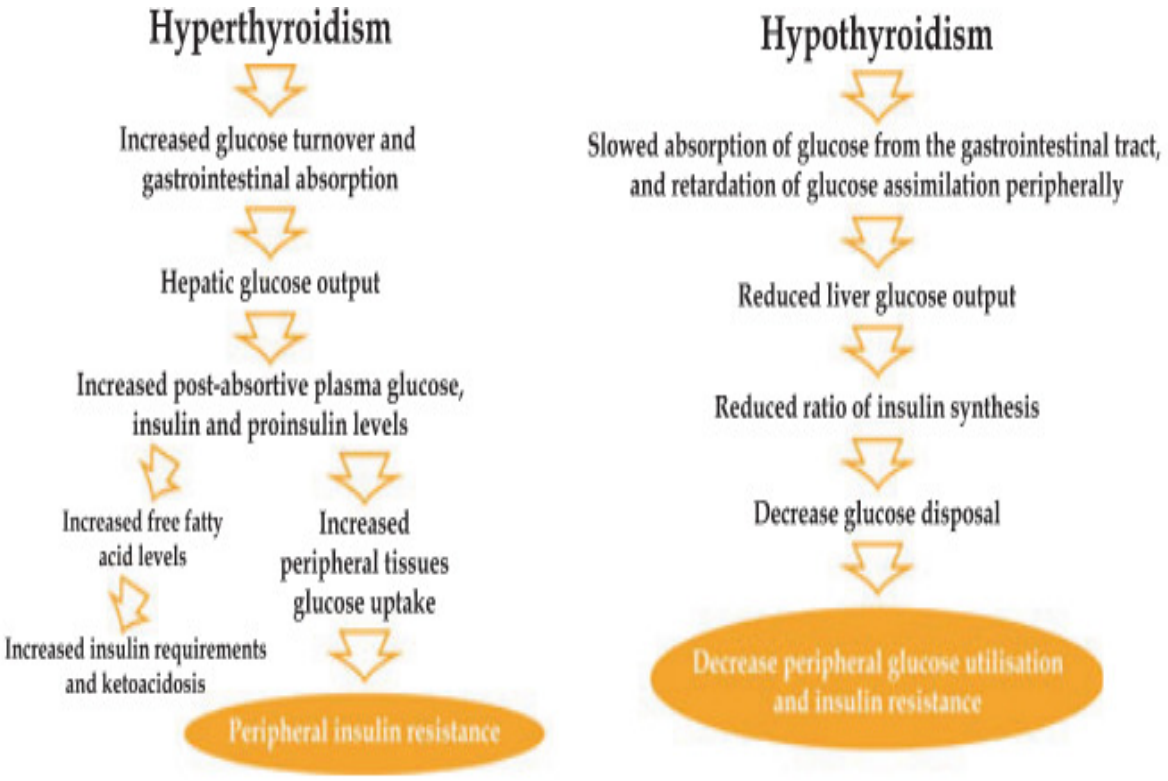
Thyroid dysfunction in DM mainly depends on glycaemic status which is mainly modulated by TRH and TSH level. It is also influenced by the occurrence of thyroid hormone binding inhibitor (THBI) which decreased the extra thyroidal conversion of T_4 to T_3. [70, 71]

In hyperthyroidism, the response of beta cells to glucose or catecholamine is increased due to increased beta cell mass. The liver endogenous glucose production is elevated and hepatic insulin sensitivity is reduced due to glycogenesis and glycogenolysis. Due to increased lipolysis in hyperthyroidism, there is an increase in FFA levels which stimulate hepatic gluconeogenesis. FFA increase also due to catecholamine stimulatied lipolysis by excess thyroid hormone level.[73]In the muscles there is an increase in skeletal muscle glucose utilization which is mediated by insulin stimulated glucose oxidation rate. [61]

The clinical diagnosis of thyroid disease in DM patients is difficult. Because poor DM control symptoms are similar with hyperthyroidism (weight loss, increased appetite and fatigue) and also severe DM nephropathy may be mistaken for hypothyroidism (edema, weight gain, and pallor).[62, 63]

FIGURE 3

RELATIONSHIP BETWEEN INSULIN RESISTENCE AND THYROID DISORDERS: [75]



EFFECTS OF DM OVER THYROID HORMONES:

Diabetes mellitus control the thyroid function in two sites: one at the stage of hypothalamic control of TSH release and secondly at peripheral tissue level by converting T_4 to T_3[64]. Hyperglycemia may cause reduction in hepatic conversion of T_4- T_3, decreased serum concentration of T_3, and raised intensity of reverse T_3.[68]

There is an alteration of thyroid hormone level in diabetic patients with poor glycemic control. In DM patients, nocturnal TSH level is decreased and the response to TRH is impaired. Insulin resistance and beta cell functions inversely co-related to TSH, that's may be by insulin antagonistic effects of thyroid hormone along with an increase in TSH. The higher serum TSH usually corresponds to lower thyroid hormones via negative feedback mechanism.[72]

MATERIALS AND METHODS

STUDY DESIGN : Case control study

PLACE OF STUDY : CMCH&RC, Irungalur, Trichy.

PERIOD OF STUDY : JANUARY 2015- JUNE 2016

SAMPLE SIZE : 170Cases and 50 Controls.

(Cases- Type 2 Diabetes mellitus patients) (Controls – non-diabetic patients)

AGE : 30-80 years

SEX : Both females (n=97) and males (n=123).

GEOGRAPHICAL DISTRIBUTION: Both urban and rural areas.

ETHICAL CONSIDERATIONS:

The necessary approval was obtained to conduct the study from the Chennai medical college hospital and research centre, ethical committee, Irungalur, Trichy. Patients were given an explanation regarding the intention of the study and informed written consent was obtained, confidentiality about their results was assured. Their participation was optional.

SELECTION OF CASES AND CONTROLS:

170 Type 2 Diabetic patients and 50 non diabetic patients between the age group of 30-80 years who were all attending in medicine, surgery and Endocrinology department OPD at CMCH&RC, TRichy , were selected for

the study. All the patients were included as cases evaluated and diagnosed as Type 2 Diabetes mellitus on the basis of history and Biochemical investigations.

Inclusion Criteria:

- 170 Patients with Type2 Diabetes mellitus (age groups 30-80 years) and 50 non diabetic were also included as controls

Exclusion Criteria:

- Seriously ill patients
- Adults who are previously diagnosed as cases of Type 1 diabetes mellitus
- Specific types of Diabetes Mellitus
- Gestational Diabetes mellitus.(GDM)
- Known case of thyroid disease.
- Cancer patients.

STUDY PROTOCOL:

After obtaining the informed consent, all patients were subjected to detailed history taking and clinical examination.

History:

A detailed history of duration and severity of the disease, clinical symptoms , and also family history, personal history and drug history and Co-morbid diseases was also obtained .

CLINICAL EXAMINATION:

A thorough physical examination was done to look for local and systemic features.

ANTHROPOMETRIC MEASUREMENTS:

Measurement of weight, height, and blood pressure were done.

The Body Mass Index was determined by wt and height calculations using the following equation:

BMI = Weight in Kg / Square of height in meters.

According to Indian guidelines, a BMI - 23 to 24.9 is defined as overweight, a BMI- $\geq$ 25 is moderate obesity and a BMI $\geq$ 30 is severe obesity.

The blood pressure was taken in the sitting posture and the average of two measurements was recorded in right arm.

COLLECTION OF SPECIMENS:

5ml of fasting venous blood samples were collected in clot activator coated polypropylene tubes by venupuncture under strict aseptic precaution as soon as the subjects got admitted as per the inclusion criteria.Similar way 2 hours post prandial also collected. Blood samples were centrifuged at 3500 rpm used for 10 minutes and serum was separated. 8-12 hours fasting samples, 2 hours post prandial samples were collected from all subjects during their hospital visit and analysis of below said parameters were done

INVESTIGATIONS:

- Fasting and Post prandial blood glucose, and HbA1c
- Renal parameters (serum urea, creatinine)
- Fasting Serum Lipid parameters which include (Total cholesterol, Triglycerides, LDL-cholesterol, HDL- cholesterol)

  Above mentioned test were analyzed in BS-420 Fully auto analyzer
- Serum Thyroid profile - FT3 , FT4 and TSH was done in ELISA reader.

SAMPLE STORAGE:

The specimens were freezed at -20°C for storage until analysis for thyroid profile.

ESTIMATION OF FASTING AND POST PRANDIAL BLOOD GLUCOSE: [85]

METHODOLOGY: Glucose Oxidase peroxidise method (END POINT)

PRINCIPLE:

The serum /plasma Glucose was first oxidized (GOD) to gluconic acid with the release of hydrogen peroxide by the enzyme glucose oxidase, which is further transformed to water and nascent oxygen by the action of enzyme peroxidase (POD). 4- Aminoantipyrine is an oxygen acceptor takes up the oxygen and simultaneously with phenol forms a pink colored chromogen which is measured at 505 nm.

Glucose + o_2 + H_2O $\xrightarrow{GOD}$ Gluconic acid + H_2O_2

H_2O_2 + phenol + 4-Aminoantipyrine $\xrightarrow{POD}$ (Red) quinoneimine complex + H_2O

GLUCOSE REAGENTS:

Phosphate buffer (Ph 7.5) : 0.1 mol/L

4-Aminoantipyrine : 5.0 mmol/L

Peroxidase : >1.5 KU/L

Glucose Oxidase : >15 KU/L

Phenol : 5.0 mmol/L

Glucose Standard (concentration: 100 mg /dl)

PROCEDURE:

Take 3 test tubes and labelled them as Blank (B), Std. (S) and (T) as follows:

S.NO	REAGENT	BLANK	STANDARD	TEST
1.	GLUCOSE REAGENT	1.0 ml	1.0 ml	1.0 ml
2.	GLUCOSE STANDARD	_	10 µl	_
3.	SPECIMEN	_	_	10 µl

Incubation period 10min & Reaction temperature 37◦C.

Mix up well and read absorbance of Std. (S) and Test (T) alongside Blank (B) on 505 nanometer or through green filter (500- 540 nm).

CALCULATION:

$$\text{Glucose conc (mg/dl)} = \frac{\Delta \text{ Abs for Test}}{\Delta \text{ Abs for Standard}} \times 100$$

Reference value:

Serum / Plasma (fasting) Glucose : 70- 100 mg/dl

Serum / Plasma (post prandial) : < 140 mg/dl

ESTIMATION OF HbA1c:[89]

METHODOLOGY:

PARTICLE ENHANCED IMMUNO TURBIDIMETRIC TEST

PRINCIPLE:

Total Hemoglobin and HbA1c in haemolysed blood is combining with the equal affinity to particle in R1. The amount of binding is proportional to the comparative concentrations of the both substances in the blood.

Mouse antihuman HbA1c monoclonal antibody (R2) binds to particle bound HbA1c.Goat antimouse IgG polyclonal antibody (R3) is interact with R2 also agglutination takes place. The calculated absorbance be proportional to the HbA1c bound to particles, which consecutively proportional to the % of HbA1c in the sample**.**

REAGENTS:

R1:Buffer : 20m mol/L Latex : 0.14%

R2: Buffer: 10mmmol/L & Mouse antihuman HbA1c monoclonal

Antibody: 5.5 mg/dL

R3: Buffer : 10mmol/L & Goat antimouse IgG polyclonal

Antibody: 67mg/dL.

ASSAY PROCEDURE:

Wavelength: 660nm Optical path :1cm Temperature: 31◦c

Sample	20µL
Reagent1	750µL
Mix and incubate for 2 min , then add	
Reagent 2	250µL
Mix and incubate for 3 min , then add	
Reagent 3	125 µL
Mix - read absorbance after exactly 2 min	

CALCULATION

The conc. of the HbA1c in unknown sample is derived from a calibration curve by appropriate mathematical models.

LIMIT OF DECTECTION:

Limit of detection is 10mmol/mol HbA1c

REFERENCE RANGE:

HbA1c	mmol/mol	%
Non -diabetic	20-42	4-6
Target of treatment	Less than 53	Less than 7
Change of treatment	Greater than 64	Greater than 8

According to ADA >6.5% - Diabetic

5.7-6.4% - Pre- Diabetic.

LIPID PROFILE

ESTIMATION TOTAL CHOLESTEROL[88]

METHODOLOGY: Cholesterol oxidase / peroxidase

PRINCIPLE:

Cholesterol esters are hydrolyzed to produce cholesterol. Then, free cholesterol takes part in two coupled reactions that permit to measure cholesterol photometrically.

The reaction sequence is as follows:

$$\text{Cholesterol ester} + H_2O \xrightarrow{\text{Cholesterol esterase}} \text{Cholesterol} + \text{Fatty acid}$$

$$\text{Cholesterol} + \tfrac{1}{2}\,O2 + H2O \xrightarrow{\text{Cho. Oxidase}} \text{Cholestenone} + H_2O_2$$

$$2H_2O_2 + \text{4- Aminoantipyrine} + \text{Phenol} \xrightarrow{\text{POD}} \text{Quinoneimine} + 4H2O$$

REAGENTS:

Cholesterol Reagent:

Mes buffer pH6.5	: 75 mmol/L
Phenol	: 6mmol/L
3,5 Dichlorophenol	: 0.2mmol/L
4 amino antipyrine	: 0.5 mmol/L
Cholesterol esterase	≥ 500KU/L

Cholesterol oxidase ≥ 300 KU/L

Peroxidise ≥ 1200 KU/L

Standard (5ml): Cholesterol 200mg/dl

The reagents were stored at 2ºC-8ºC.

PREPARATION OF WORKING SOLUTION:

The reagents are allowed to attain room temperature. Both the reagent and the std. are supplied for instant use.

PROCEDURE:

Reagent / Test	**Blank**	**Standard**	**Test**
Distilled water	10µL	--	
Reagent	1mL	1mL	1ml
Standard	--	10µL	--
Sample	--	--	10µL

Mixed and incubated for 10 min at 37^0C and read it by 505 nanometer**.**

CALCULATIONS:

$$\frac{\text{Sample absorbance}}{\text{Standard absorbance}} \times 200 = \text{Sample concentration (mg/dl)}$$

LINEARITY: This method is linear up to 1000mg/dl.

REFERENCE VALUES:

Serum Cholesterol (Total)

Desirable value : up to 200 mg/dl

Borderline High : 200 - 239 mg/dl

High value : > 240 mg/dl.

QUANTITATIVE ESTIMATION OF SERUM TRIGLYCERIDES[86]

METHODOLOGY: Glycerol-3- phosphate oxidase (GPO)

PRINCIPLE:

Quantification of triglycerides by enzymatic separation with lipoprotein lipase. Quinoneimine which is separated from 4-aminoantipyrine and 4-Chlorophenol by hydrogen peroxide of peroxidase act as indicator.

The reaction sequence is as follows:

$$\text{Triglycerides} \xrightarrow{\text{LPL}} \text{Glycerol + Fatty acid}$$

$$\text{Glycerol + ATP} \xrightarrow{\text{GK}} \text{Glycerol -3-phosphate + ADP}$$

$$\text{Glycerol -3-phosphate + oxygen} \xrightarrow{\text{GPO}} \text{Dihydroxyacetone phosphate + } H_2O_2$$

$$2H_2O_2 + \text{Aminoantipyrine + 4- Chlorophenol} \xrightarrow{\text{POD}} \text{Quinoeimine + HCl+ } H_2O$$

REAGENTS:

four-chlorophenol	: 4 mmol / L
ATP	: 2 mmol / L
Mg^{2+}	: 15mmol/L
Glycerolkinase	: ≥0.4 kU/L
Lipoprotein lipase	: ≥ 2 kU/L

Peroxidase : ≥ 2 kU/L

4-Aminoantipyrine : 0.5mmol/l

Glycerol -3- phosphate – oxidase : ≥0.5 kU /L

Good's buffer at pH 7.2 :50 mmol / l

Standard: Triglycerides 200mg/dl

The reagents were stored at 2ºC-8ºC

PREPARATION OF WORKING SOLUTION:

The reagents are allowed to attain room temperature. Both the reagent and the std. are supplied for ready to utilize.

PROCEDURE:

Reagent / Test	**Blank**	**Standard**	**Test**
Distilled water	**10µl**	**--**	**--**
Reagent	**1ml**	**1ml**	**1ml**
Standard	**--**	**10µl**	**--**
Sample	**--**	**--**	**10µl**

It was gently mixed, incubated the tubes for ten minutes by room temperature and measured by 505 nanometre.

CALCULATIONS:

$$\frac{\text{Sample absorbance}}{\text{Standard absorbance}} \times 200 = \text{Sample concentration (mg/dl)}$$

To correct for free glycerol, subtract 10mg/dl from the triglycerides value calculated above.

LINEARITY: 2-1000mg/dl.

REFERENCE VALUES:

Desirable value : < 150 mg/dl

ESTIMATION OF SERUM LOW DENSITY LIPOPROTEIN CHOLESTEROL[86] (LDL-c)

METHODOLOGY:

Direct enzymatic method

PRINCIPLE:

In the first step, LDL is secluded when non-LDL-lipoprotein is enzymatically processed. In the 2nd step, LDL is unconfined and LDL-cholesterol is selectively determined by a enzymatic reaction which produce colour change.

1. LDL + Reagent 1 $\longrightarrow$ Protected LDL

$$\text{Chylomicrons , HDL, VLDL} \xrightarrow{\text{CHE \&CHO}} \text{Cholestenone} + H_2O_2$$

$$H_2O_2 \xrightarrow{\text{Catalase}} H_2O$$

2. Protected LDL + Reagent 2 $\longrightarrow$ LDL

$$\text{LDL-C} \xrightarrow{\text{CHE \&CHO}} \text{Cholestenone} + H_2O_2$$

$$H_2O_2 + \text{4- Aminoantipyrine} + \text{H-DAOS} \xrightarrow{\text{POD}} \text{Color}$$

REAGENTS:

Reagent 1:

Cholesterol esterase	: ≥2.5 kU/L
Cholesterol oxidase	: ≥2.5 kU/L
N-(2-hydroxy-3-sulfopropyl)-	: 0.5mmol/L
3,5- Dimethoxyaniline, (H-DAOS)	
Good's buffer pH 6.8	: 20 mmol / L
Catalase	: ≥500 kU/

Reagent 2:

Good's buffer (pH 7.0)	:25 mmol / L
Peroxidase	: ≥15 kU/L
4-Aminoantipyrine	: 3.4mmol/L

Calibrator: LDL- Cholesterol 132 mg/dL

The reagents were stored at 2ºC-8ºC

PREPARATION OF WORKING SOLUTION:

The reagents are allowed to attain room temperature. Both of the reagent and the std. are supplied as ready to use.

PROCEDURE:

The sample and the working solution were brought to room temperature prior to use.

Reagent / Test	BLANK	CALIBRATOR	TEST
CALIBRATOR	_	3.0µl	-
SAMPLE	-	-	3.0µl
DISTILLED WATER	3.0µl	_	
REAGENT 1	280µl	280µl	280µl
Mix, incubate 5min,at 37◦C , read absorbance (A1), then add:			
REAGENT 2	70 µl	70 µl	70 µl

Mixed well and incubated up to 5 min, at 37◦C, read absorbance (A2) again Blank

$\Delta A = [(A_2 - A_1)$ sample or calibrator] - [(A2-A1)Blank]

CALCULATIONS:

$\frac{\Delta A \text{ Sample}}{\Delta A \text{ calibrator}}$ X Conc.calib = Sample concentration(mg/dl)

LINEARITY: This method is to determine LDL-C concentration within a measuring range from 1- 400mg/dl.

REFERENCE VALUES:

Serum LDL-C:

Desirable value : ≤ 130 mg/dl

Borderline High : 130 - 160 mg/dl

High value : above 160 mg/dl

HIGH DENSITY LIPOPROTEIN CHOLESTEROL[86] (HDL-c)

METHODOLOGY: Direct enzymatic method

PRINCIPLE OF THE METHOD:

Antibodies beside the human lipoproteins are used to form antigen-antibody complexes with VLDL, LDL and chylomicrons in a way that only HDL-cholesterol is determined by an enzymatic cholesterol measurement.

The reaction sequence is as follows:

$$\text{LDL, VLDL, Chylomicrons} \xrightarrow{\text{Anti-human } \beta \text{ – lipoprotein antibodies}} \text{Antigen – antibody complexes + HDL}$$

$$\text{HDL –C} + H_2O + O_2 \xrightarrow{\text{CHE \&CHO}} \text{Cholest-4-en-3-one} + \text{Fatty acid} + H_2O_2$$

$$H_2O_2 + \text{F- DAOS} + \text{4- Aminoantipyrine} \xrightarrow{\text{POD}} \text{Blue Complex} + H_2O$$

REAGENTS:

Reagent 1:

Ascorbate oxidase	: 2250 U/ L
Anti-human β - lipoprotein Antibody (sheep)	
Peroxidase	: 2000 U/L
4-Aminoantipyrine	: 0.75mmol/l
Good's buffer (pH 7.0)	: 25 mmol / l

Reagent 2:

Good's buffer (pH 7.0) : 30 mmol / l

Cholesterol esterase enzyme : 4000 U/L

Cholesterol oxidase enzyme : 20000U/L

N-Ethyl N (2-hydroxy 3-sulfopropyl) - :0.8mmol/L

3,5- dimethoxy-4-fluoroaniline,

Na. salt

Calibrator: HDL-Cholesterol : 50.6 mg/dl

PREPARATION OF WORKING SOLUTION:

The reagents were allowed to attain room temperature. Both reagent and std. were supplied ready to use.

PROCEDURE:

The sample and the working solution were brought to room temperature prior to use.

Reagent / Test	**BLANK**	**CALIBRATOR**	**TEST**
CALIBRATOR	_	2.4µl	-
SAMPLE	-	-	2.4µl
DISTILLED WATER	2.4µl	_	
REAGENT 1	240µl	240µl	240µl
Mix and incubate for 5min - 37◦C, take reading of absorbance (A1, afterwards add:			
REAGENT 2	60 microlit	60 microlit	60 microlit

$\Delta A = (A_2 - A_1)$ sample or calibrator.

It was mixed and incubated the test tubes for five minutes by room temperature. Take reading of the absorbance A_2.

CALCULATIONS: $\frac{\Delta A \text{ Sampl}}{\Delta A \text{ Calibrator}}$ X conc. Calib = Sample concentration (mg/dl)

LINEARITY: From 1-180 mg/dl.

REFERENCE VALUES:

Serum HDL-C: Males : 30-60 mg/dl

Females : 35-75 mg/dl

ESTIMATION OF UREA[74]

PRINCIPLE:

Urea is hydrolysed by enzyme urease to give ammonia and CO_2. This ammonia reacts with 2-oxoglutarate and NADH with glutamate-dehydrogenase (GLDH) enzyme to obtain glutamate & NAD+. The test is optimized so that GLDH is the rate limiting enzyme. The decrease in the absorbance is proportional to the urea concentration in the given time intervals.

REAGENTS:

ENZYMES:

Tris buffer (pH7.8) : 125 mmol/l

ADP : 0.88 mmol/l

Urease : >20kU/l

GLDH : >0.3kU/l

Sodium Azide : 0.095%

SUBSTRATES:

2-oxoglutrate : 25mmol/l

NADH : 1.25mmol/l

Sodium Azide : 0.095%

STANDARD:

Urea 80mg/dl or 13.3mmol/l Or

Sodium Azide 0.095%

PREPARATION OF REAGENT:

Reagent is prepared by mixing 4 parts of enzymes with 1 part of substrates.

ASSAY:

Wavelength: 340nm, 334nm, 365nm

Optical path: 1cm

Temperature: 25°C, 30°C to 37°C

Measurement : Adjacent to reagent blank (RB). 1 reagent blank for each series is required.

PROCEDURE:

REAGENT START PROCEDURE:

Reagent / Test	**Reagent Blank**	**Standard**
Sample /standard	-	10µL
Enzyme	1000µL	1000 µL
Mix, incubate for approx.1 minute.		
Substrate	250µL	250µL
Mix , read absorbance of Sample /standard after 30 seconds(A1), start timer simultaneously & read after exactly 1 minute(A2).calculate the absorbance difference: ΔA Sample /standard= (A2-A1)-ΔARB.		

SAMPLE START PROCEDURE:

Reagent / Test	Reagent blank(RB)	Sample / standard
Sample / standard	-	10µl
Working reagent	1000µl	1000µl
Mix , read absorbance of Sample /standard after 30 seconds(A1), start timer simultaneously & read after exactly 1 minute(A2).calculate the absorbance difference: ΔA Sample /standard= (A2-A1)-ΔARB		

CALCULATION:

C= 80.0× ΔA Sample/ ΔA standard [mg/dl] or

C=13.3× ΔA Sample/ ΔA standard [mmol/l]

LINEARITY: The test is linear up to 300mg/dl or 50mmol/l.

REFERENCE VALUES:

SERUM UREA value: 15-45 mg/dl or 1.7 -8.3 mmol/l

DETERMINATION OF SERUM CREATININE[74]

METHOD : JAFFE'S KINETIC METHOD

PRINCIPLE OF THE METHOD**:**

Creatinine present in the sample combines with picric acid in presence of alkaline medium producing orange-red coloured complex - creatinine picrate. The difference in absorbance at predetermined time during conversion is proportional to the concentration of creatinine in the sample.

Creatinine + Picric acid → Creatinine picrate color complex

REAGENTS:

Reagent 1: NAOH : o.2 mol / L

Reagent 2: Picric acid : 20 mmol/L

Standard : (Creatinine 2 mg/dL):
Creatinine 177µmol/L

PROCEDURE:

The sample and the working solution are brought to room temperature prior to use.

GENERAL SYSTEM PARAMETERS:

Reaction Type : Fixed Time

Reaction slope : Increasing

Wavelength : 500 nm (490-510 nm)

Flow cell Temp. : 20°C- 25°C or 37°C

Path length : 1 cm

Substrate start

Reagent / Test	**Blank**	**Sample or standard**
Sample or standard	-	5oµL
Dist.Water	5oµL	-
Reagent 1	1000µL	1000µL
Mix and incubate 0-5 mins, then add,		
Reagent 2	250µL	250µL
Mix and read absorbance A1 after 60 sec, read absorbance A2 after further 120 sec		

ΔA=(A2-A1) sample or standard

Sample start

Reagent / Test	**Blank**	**Sample or standard**
Sample or standard	-	5oµL
Dist.Water	5oµL	-
Mono reagent	1000µL	1000µL
Mix and read absorbance A1 after 60 sec, read absorbance A2 after further 120 sec		

ΔA=(A2-A1) sample or standard

Calculation:

Creatinine (mg/dL) = ΔA sample / ΔA (std/cal) × conc. Std/cal (mg/dL)

Measurement range: 0.2 - 15 mg/dL**.**

Limit of Detection: Above 0.2 mg/dL.

REFERENCE VALUES:

Serum Creatinine:

Males : 0.7-1.3 mg/dL

Females : 0.6-1.1 mg/dL

ESTIMATION OF THYROID PROFILE[87]

ESTIMATION OF FT3 (FREE T3)[87]

METHODOLOGY: Enzyme linked immunosorbent assay (**ELISA**)

PRINCIPLE:

Principle of competitive binding of Free T3 in a test sample and T3 – Peroxidase conjugate for a narrow number of binding sites present on the anti – T3 (Sheep) coated well. So the quantity of T3 –Peroxidase conjugate bound to the well is inversely proportional to the concentration of FT3 in the specimen.

After incubation, specimen and T3 –Peroxidase unbound enzyme conjugate is separated by washing in the equilibrium state. TMB /Substrate solution is mixed and a blue colour forms. The intensity of this colour, which changes to yellow after stopping the response is inversely proportional to the quantity of FT3 in the sample.

The ELISA micro plate readers is used to determine the absorbance. A dose response curve is used to extrapolate Specimen's concentration by using serum calibrators of known antigen concentrations.

KIT INSIDE:

1. MICROTITER STRIPS

   Eight - Well snap off strips, coated by anti –T3 sheep antibiody)

2. CALIBRATORS: (6 × 2 ml) instant use, human serum

FT3 CONCNTRATION	**Pg/ml**
Calibrator 1	O (A)
Calibrator 2	1 (B)
Calibrator 3	3 (C)
Calibrator 4	5 (D)
Calibrator 5	8 (E)
Calibrator 6	16 (F)

3. ENZYME – ANTIGEN CONJUGATE: (13 ml)

Ready for use, coloured red T3 – HRP Conjugate in a protein stabilising matrix. - 1%.

4. WASH SOLUTION : (20 ml)

Concentrated used for ca.1000ml . Tris buffer saline. - 250 mmol/l

5. SUBSTRATE REAGENT :(14ml)

3, 3', 5,5', tetramethylbenzidine - .5 g/l

Sodium acetate(buffer) - .05mol/l

Urea hydrogen peroxide - .03%

6. STOP SOLUTION : (7.5ml)

H2SO4 - .5 mol/l

PRESERVATIVES: Total concentration < .04%.

SPECIMEN:

- Serum
- Specimens stored up to 5 days – (2-8◦C), for 30 days at -20◦C.

PROCEDURE:

STEP 1	**WELL (µl)**	
	A1…..D2 CALIBRATORS	E2 SPECIMEN
CAL –A-F; in duplicate	50	_
SPECIMENS, CONTROLS; in copy	_	50
CONJUGATE	100	100

Gently shake and cover MIC with Adhesive strip

Incubate for 60 min at 20….25◦C

Wash up to 3 times

WASH	300	300
2 nd **STEP**		
SUBSTRATE	100	100
Don't vibrate MIC after SUB addition		
Incubate for 15 min at 20-25◦C		
STOP	50	50
mix up cautiously		

Read the absorbance immediately or within 30 mins by 450nm, via a reference wavelength of **630- 690nm**.

VALIDATION OF TEST:

The test results are accessible if calibration of highest absorbance CAL- **A ≥ 1.3.**

CALCULATION:

In a lin –lin graph, Plot absorbance is calculated in opposition to calibrator. Suitable interpolations of plotted measuring points are produced in a calibration curve, from which the analyte concentration in the sample can be measured.

REFERENCE VALUE:

	ADULT pg /dl	**PREGNANT** pg / dl
MEAN	2.8	3.0
STANDARD DEVIATION (S.D)	0.7	0.6
EXPECTED RANGE (≥2 S.D)	1.4-4.2	1.8-4.2

ESTIMATION OF FT4 (FREE T4)[87]

METHODOLOGY : ENZYME LINKED IMMUNOSORBENT ASSAY.

PRINCIPLE:

The ELISA is based on the rule of competitive binding between FT4 in a test sample and T4 –Peroxidase conjugate for a narrow number of binding sites on the anti –T4(Sheep) coated well. Thus the quantity of binding is inversely proportional to the concentration of FT4 in the specimen.

Following incubation of specimen and T4 –Peroxidase conjugate unbound enzyme conjugate is separated in the equilibrium state by washing. TMB /Substrate solution is added and a blue colour forms. The intensity of the colour , which changes to yellow after stopping the reaction , is inversely proportional to the quantity of FT4 in the sample.

ELISA micro plate reader used to read the absorbance of 630 nanaometer. The dose response curve developed by using serum calibrators of known antigen concentrations is used to extrapolate Specimen's concentration.

KIT CONTENTS:

7. MICROTITER STRIPS (in 1n strip holder)

8 - Well snap –off strips, coated with anti –T4sheep)

8. CALIBRATORS: (6 × 2 ml) for instant use, in human serum

FT4 CONCNTRATION	**ng/ml**
Calibrator 1	0 (A)
Calibrator 2	0.40 (B)
Calibrator 3	1.25 (C)
Calibrator 4	2.10 (D)
Calibrator 5	5.00 (E)
Calibrator 6	7.40 (F)

9. ENZYME – ANTIGEN CONJUGATE: (13 ml)

Instant use coloured green T4-HRP Conjugate in a protein stabilising matrix.- 1%.

10. WASH SOLUTION : (20 ml)

Concentrated for ca.1000ml

Tris buffered saline. - 250 mmol/l

11. SUBSTRATE REAGENT :(14ml)

3, 3', 5,5', tetramethylbenzidine – 0.5 g/l

Sodium acetate buffer - 0.05mol/l

Urea hydrogen peroxide - 0.03%

12. STOP SOLUTION : (7.5ml)

Sulphuric acid -0.5 mol/l

PRESERVATIVES: Total concentration < 0.04%.

SPECIMEN:

- Serum
- Specimens stored up to 5 days at 2-8◦C, for 30 days at -20◦C.

PROCEDURE:

STEP 1	**WELL (µl)**	
	A1…..D2 CALIBRATORS	E2 SPECIMEN
CAL –A-F; in duplicate	50	_
SPECIMENS, CONTROLS; in duplicate	_	50
CONJUGATE	100	100

shake gently and cover MIC with Adhesive strip, Incubate for 60 min at 20….25◦C, Wash 3 times

WASH	300	300
STEP -2		
SUBSTRATE	100	100
Do no rock MIC after SUB addition		
Incubate 15 min at 20…25◦C		
STOP	**50**	**50**
Mix cautiously		

Note the absorbance at **450nm** as soon as possible or within 30min, after terminating of reaction, using a reference wavelength of **630- 690nm**.

VALIDATION OF THE TEST:

The test results are accessible if calibration of highest absorbance CAL- **A ≥ 1.3.**

CALCULATION:

In a lin –lin graph, Plot absorbance is calculated in opposition to calibrator. Suitable interpolations of plotted measuring points are produced in a calibration curve, from which the analyte concentration in the sample can be measured.

REFERENCE VALUE:

	ADULT	**REGNANT**
MEAN	1.4 ng /ml	1.5 ng / ml
STANDARD DEVIATION (S.D)	0.3 ng /ml	0.37ng /ml
EXPECTED RANGE (≥ 2 S.D)	0.8-2.0 ng /ml	0.8-2.2 ng/ ml

ESTIMATION OF TSH[87]

METHODOLOGY: ENZYME LINKED IMMUNOSORBENT ASSAY **(ELISA)**

PRINCIPLE:

The ELISA is based on the principle of competitive binding between TSH in a test specimen and TSH –Peroxidase conjugate for a limited number of binding sites on the anti –TSH (Sheep) coated well. Thus the amount of TSH – Peroxidase conjugate bound to the well is inversely proportional to the concentration of TSH in the specimen.

After incubation of sample and TSH –Peroxidase unbound enzyme conjugate is removed in the stability state by washing. TMB /Substrate solution is mixed and a blue colour develops. The intensity of this colour is inversely proportional to the quantity of TSH in the sample.

ELISA micro plate readers or programmed ELISA systems (HUMAN'S Huma - Reader or ELISYS line) are used to read the absorbance.The dose response curve developed by using serum calibrators of known antigen concentrations is used to extrapolate Specimen's concentration

KIT CONTENTS:

1. **MICROTITER STRIPS (** in 1n strip holder**)**

   Eight Well snap –off strips , with anti –TSH sheep coating)

2. CALIBRATORS: (6 × 2 ml) all set for use, human serum

TSH CONCNTRATION	mIU/ml
Calibrator 1	0 (A)
Calibrator 2	0.5 (B)
Calibrator 3	3.0 (C)
Calibrator 4	6.0 (D)
Calibrator 5	15.0 (E)
Calibrator 6	30.0 (F)

3. ENZYME – ANTIGEN CONJUGATE: (13 ml)

For instant use, red anti-TSH (goat), HRP - labelled.

4. WASH SOLUTION : (50 ml)

Concentrated for ca.1000ml – pH 6.25 ± 0.1

Tris buffered. - 10mmol/l

NaCl - 8gm/l

5. SUBSTRATE REAGENT :(13ml)

3, 3', 5,5', tetramethylbenzidine (TMB) – 1.2mmol/l

Hydrogen peroxide - ≤ 6.0 mmol/l

STOP SOLUTION: (15ml)

H2so4 -0.5 mol/l

PRESERVATIVES: Total concentration < 0.1%.

SPECIMEN:

- Serum
- Specimens stored up to 5 days at 2-8◦C, for 30 days at -20◦C.

PROCEDURE:

Keep Reagents and specimens at room temperature prior to use

STEP 1	**WELL (µl)**	
	A1…..D2 CALIBRATORS	E2 SPECIMEN
CAL –A-F; in copy	50	_
SPECIMENS, CONTROLS; in copy	_	50
CONJUGATE	100	100

shake gently and cover MIC with Adhesive strip, Incubate for 60 min at 20--.25◦C, Wash up to 3 times

WASH	300	300
STEP -2		
SUBSTRATE	100	100
Don't shake MIC after SUB addition		
Incubate for 15 min at 20…25◦C		
STOP	100	100
Mix cautiously		

Note absorbance at **450nanometre** in 30min, after reaction at **630-690nanometre**

TEST VALIDATION :

If The following criteria are met, The test results are valid.

CALIBRATOR	ACCEPTED RANGE (OD)
A	< .05
B	>2.0 × absorbance CAL(A)
C	>3.0 × absorbance CAL(B)
D	>1.4 × absorbance CAL(C)
E	>1.9 × absorbance CAL(D)
F	>1.5 × absorbance CAL(E)
F	>1.2

CALCULATION:

In a lin –lin graph, Plot absorbance is calculated in opposition to calibrator. Suitable interpolations of plotted measuring points are produced in a calibration curve, from which the analyte concentration in the sample can be measured.

REFERENCE VALUE:

NORMAL RANGE: 0.3 - 4.0 mIU/L

STATISTICAL ANALYS:

All the data was initially entered to Microsoft Excel 2010 and later these spreadsheets were used for analysis. Statistical analysis was done by using SPSS version 20.0.

- Descriptive statistics were calculated as frequency, percentage, mean and standard deviation. Descriptive data were represented using various tables, graphs, diagrams etc.
- For all the statistical tests of significance, p value of <0.05 was considered to reject the null hypothesis.
- After the normality tests (Kolmogorov-Smirnov and Shapiro-Wilk) showed normal distribution of continuous variables, Student "t" test was done to test the difference in means between the study group and the control group.
- ANOVA test was done to test the difference in means between more than 2 groups.
- For categorical nominal variables, Chi-square test was done to test the association between the variables.

RESULTS

Table 1 Age distribution of the study population (n=220)

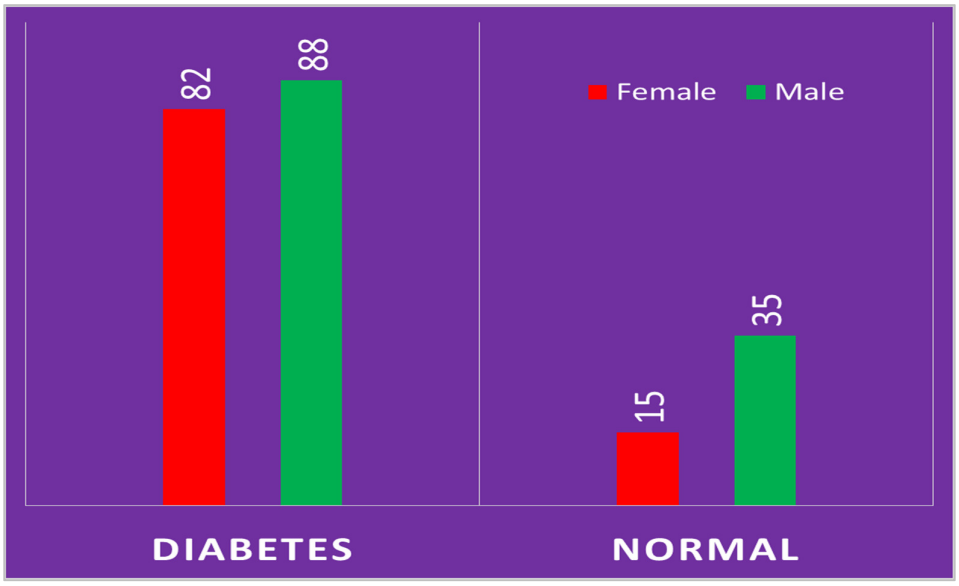
Age group	T_2DM Cases N (%)	Control N (%)	Total N (%)
31-40 years	31 (18.2)	8 (16)	39 (17.7)
41-50 years	57 (33.5)	25 (50)	82 (37.3)
51-60 years	50 (29.4)	10 (20)	60 (27.3)
61-70 years	30 (17.6)	5 (10)	35 (15.9)
71-80 years	2 (1.2)	2 (4)	4 (1.8)
Total	170 (100)	50 (100)	220 (100)

Chi-square p value: 0.121

Mean age (± S.D): 50.65 (9.92) years, minimum: 32 years, maximum: 80 years.

Comments: About 37% of the study subjects were in the age group of 41-50 years while 27% were aged 51-60 years. The study group is not significantly different from control group in age distribution.

Fig. 4. Bar chart showing gender distribution of the study population (n=220)



Chi-square p value: 0.022

Comments:

Majority of the study subjects were males (56%) while the remaining 44% were females. The control group had more males than the study group and this difference in gender distribution was statistically significant.

Table 2 Distribution of BMI across the groups (n=220)

BMI group	Group		Total
	Case	Control	
Underweight	1	0	1
(<18.5)	0.6%	0%	0.5%
Normal	37	19	56
(18.5-22.9)	21.8%	38%	25.5%
Overweight	35	9	44
(23 - 24.9)	20.6%	18%	20.0%
Obesity	97	22	119
(≥ 25)	57.1%	44%	54.1%
Total	170	50	220
	100.0%	100.0%	100.0%

Chi square p value: 0.130

Comments:

The differences in the distribution of overweight and obese individuals across the groups were not statistically significant.

Fig 5 Bar chart showing distribution of BMI across the groups (n=220)

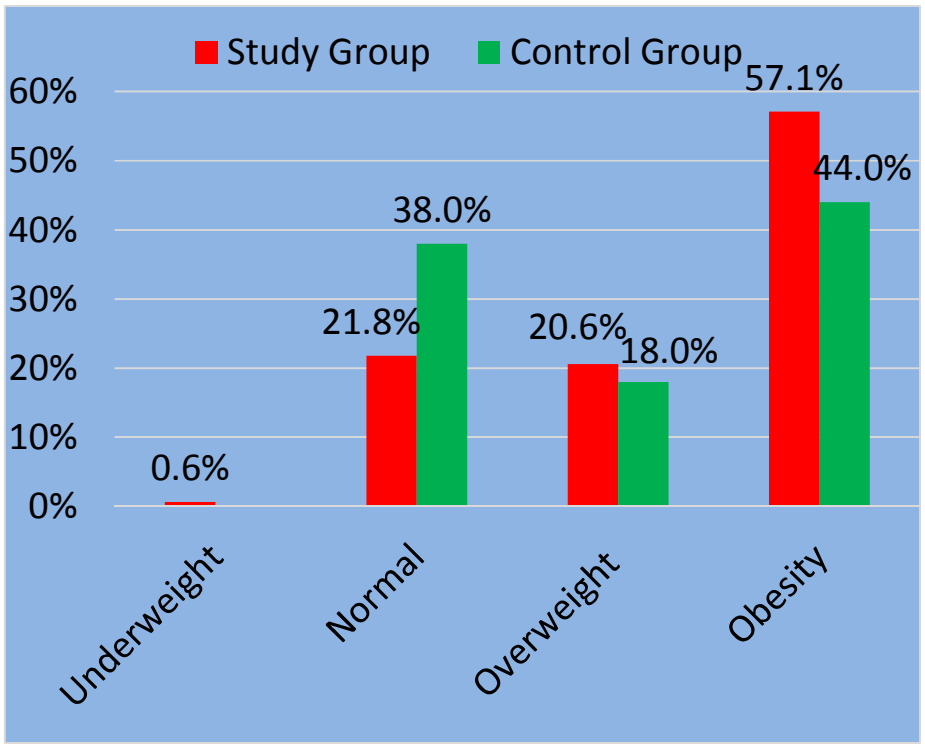


Table 3 Distribution of blood glucose and glycated haemoglobin values between the T2DM group and control group (n=220)

	Group	N	Mean	Mean difference	Student "t" test p value
Fasting Blood Glucose (mg/dl)	T2DM	170	156.45	64.573	**<0.001**
	Control	50	91.88		
Post-prandial Blood Glucose (mg/dl)	T2DM	170	246.98	135.476	**<0.001**
	Control	50	111.50		
HbA_1c (%)	T2DM	170	7.094	2.0441	**<0.001**
	Control	50	5.050		

Comments:

Subjects in the study group had higher mean fasting and post-prandial blood glucose levels and HbA_1clevels than controls as they are diabetics and this mean difference was statistically significant ($p<0.05$).

Table 4 Distribution of renal function tests between the T2DM **group and control group (n=220)**

	Group	**N**	**Mean**	**Mean difference**	**Student "t" test p value**
Blood Urea (mg/dl)	T2DM	170	21.81	0.866	0.402
	Control	50	20.94		
Serum creatinine (mg/dl)	T2DM	170	0.905	-0.0287	0.302
	Control	50	0.934		

Comments:

Subjects in the study group had higher mean blood urea and serum creatinine levels than controls but this mean difference was not statistically significant ($p>0.05$).

Table 5 Distribution of total cholesterol levels between the T2DM **group and control group (n=220)**

	Group	N	Mean	Mean difference	Student "t" test p value
Total cholesterol (mg/dl)	Study	170	187.64	5.855	0.342
	Control	50	181.78		

Comments:

Subjects in the study group had higher mean total cholesterol levels than controls but this mean difference was not statistically significant ($p>0.05$).

Table 6 Distribution of serum triglyceride levels between the T2DM **group and control group (n=220)**

	Group	N	Mean	Mean difference	Student "t" test p value
Serum triglycerides (mg/dl)	Study	170	144.81	27.552	**0.003**
	Control	50	117.26		

Comments:

Subjects in the study group had higher mean serum triglyceride levels than controls and this mean difference was statistically significant ($p<0.05$).

Table7 Distribution of serum HDL cholesterol levels between the T2DM **group and control group (n=220)**

	Group	**N**	**Mean**	**Mean difference**	**Student "t" test p value**
Serum HDL cholesterol (mg/dl)	Study	170	45.38	-0.084	0.959
	Control	50	45.46		

Comments:

Subjects in the T2DM group had mean serum HDL cholesterol levels similar to that of controls and this mean difference was not statistically significant (p>0.05).

Table 8 Distribution of serum LDL cholesterol levels between the T2DM **group and control group (n=220)**

	Group	**N**	**Mean**	**Mean difference**	**Student "t" test p value**
Serum LDL cholesterol (mg/dl)	Study	170	109.01	-0.568	0.895
	Control	50	109.58		

Comments:

Subjects in the study group had mean serum LDL cholesterol levels similar to that of controls and the minor mean difference was not statistically significant (p>0.05).

Fig 6 Bar chart showing distribution of lipid parameters across the groups (n=220)

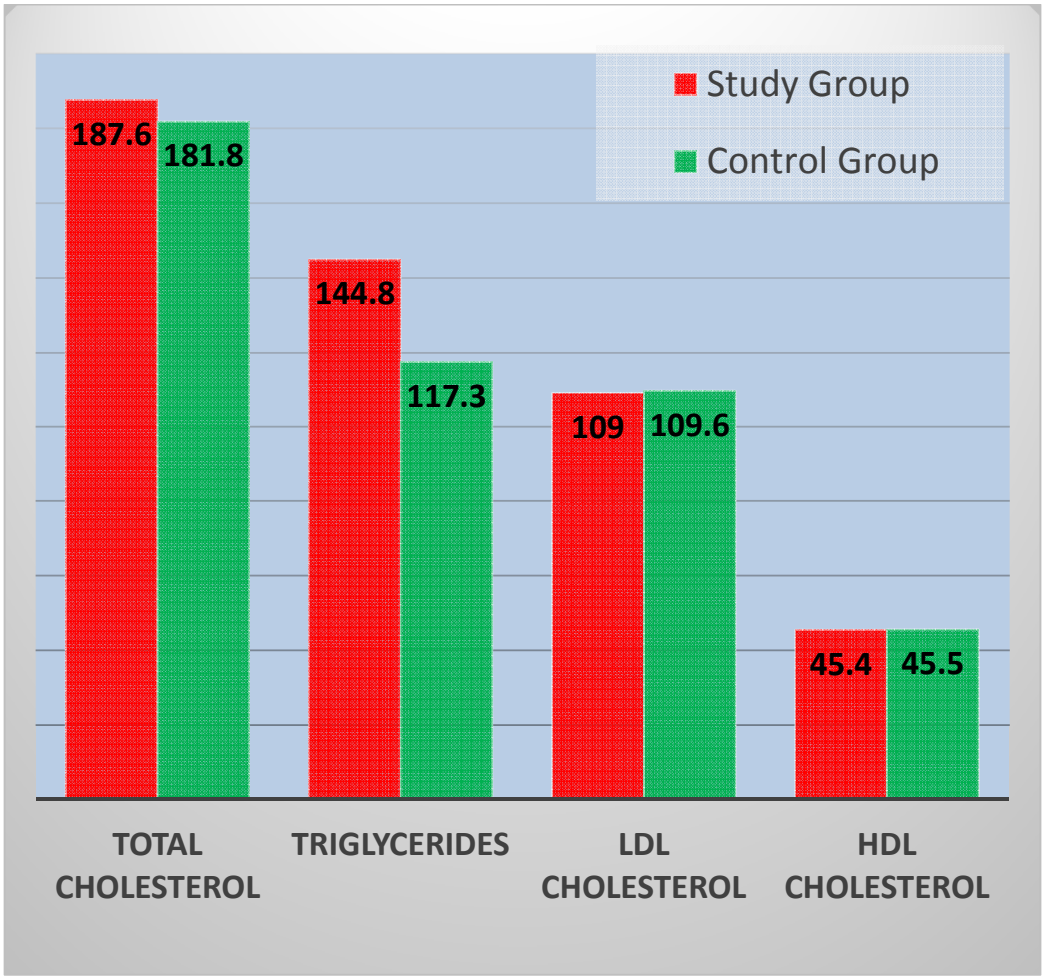


Table 9 Descriptive statistics of various thyroid parameters in the T2DM **group and control group (n=220)**

Thyroid biochemical parameters	Study group (n=170)		Control group (n=50)		Student 't' test p value
	Mean	Standard Deviation	Mean	Standard Deviation	
Free T3 (pg/ml)	2.46	0.89	2.40	0.49	0.690
Free T4 (ng/ml)	1.38	0.43	1.32	0.29	0.385
TSH (µU/L)	4.36	6.03	2.85	2.33	0.085

Comments:

Subjects in the study group who had diabetes had similar mean free T3 and T4levels compared to the controls but had a higher mean TSH levels than controls but this mean difference was not statistically significant ($p>0.05$).

Table 10 Distribution of various thyroid parameters in the study group and control group (n=220

Thyroid biochemical parameters		Study group (n=170)		Control group (n=50)		'z' test p value
		n	%	n	%	
Free T3 (pg/ml)	Low (<1.4)	7	4.1%	1	2 %	>0.05
	Normal (1.4 to 4.2)	161	94.7%	49	98 %	
	High (>4.2)	2	1.2%	0	0 %	
Free T4 (ng/ml)	Low (<0.8)	11	6.5%	1	2 %	>0.05
	Normal (0.8 to 2)	156	91.8%	49	98 %	
	High (>2)	3	1.8%	0	0 %	
TSH (µU/L)	Low (<0.3)	3	1.8%	0	0 %	*<0.05**
	Normal (0.3 to 4)	119	70.0%	42	84 %	
	High (>4.00)	48	28.2%	8	16 %	

Comments:

1. Subjects in the study group who had diabetes had higher proportion of subjects in the low and high free T3 and T4group compared to the controls but this difference was not statistically significant (p>0.05)

2. Subjects in the study group had a higher proportion of subjects in the low and high TSH group than controls and this difference was statistically significant (p<0.05).

Fig 7 Bar chart showing distribution of thyroid parameters across the groups (n=220)

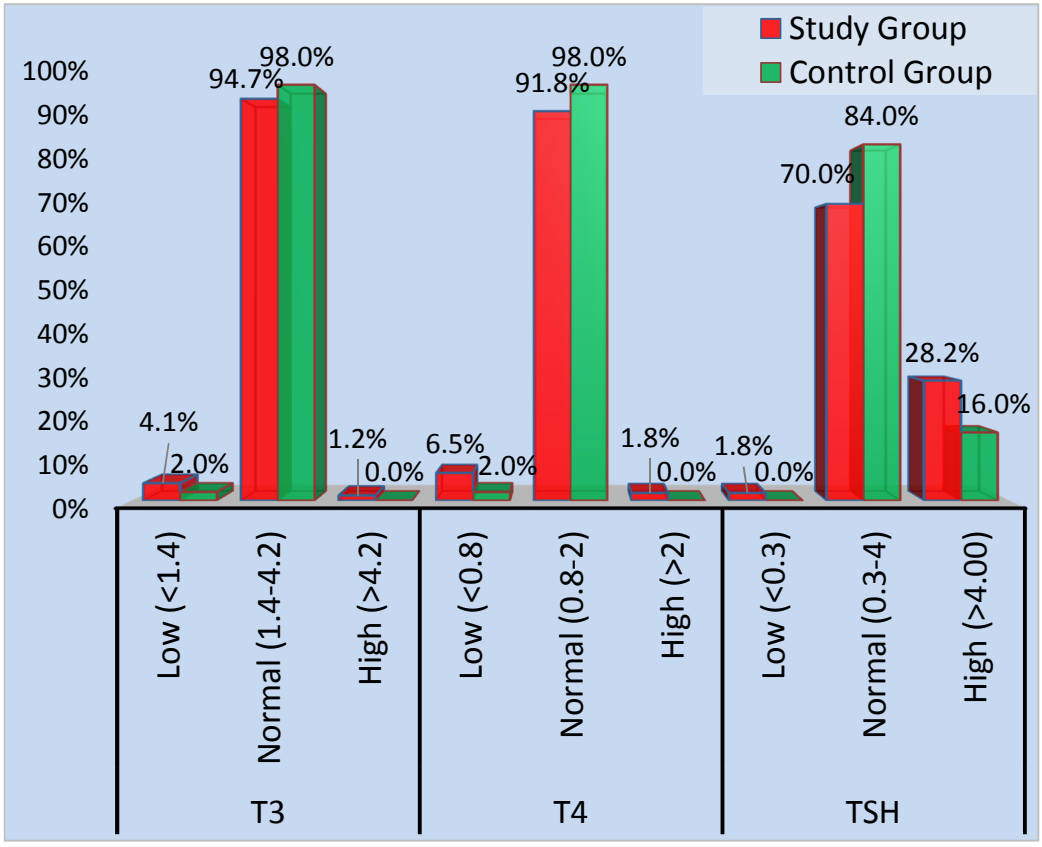


Table 11 Distribution of thyroid dysfunction in the study population (n=220)

Classification	Frequency	Percent
Normal	155	70.5
Hypothyroidism	17	7.7
Hyperthyroidism	6	2.7
Subclinical Hypothyroidism	42	19.1
Total	220	100.0

Comments:

About 19% of the subjects had subclinical Hypothyroidism while roughly 8% and 3% of the subjects had hypo and hyperthyroidism, respectively.

Fig 8 Pie chart showing distribution of thyroid dysfunction across the groups (n=220)

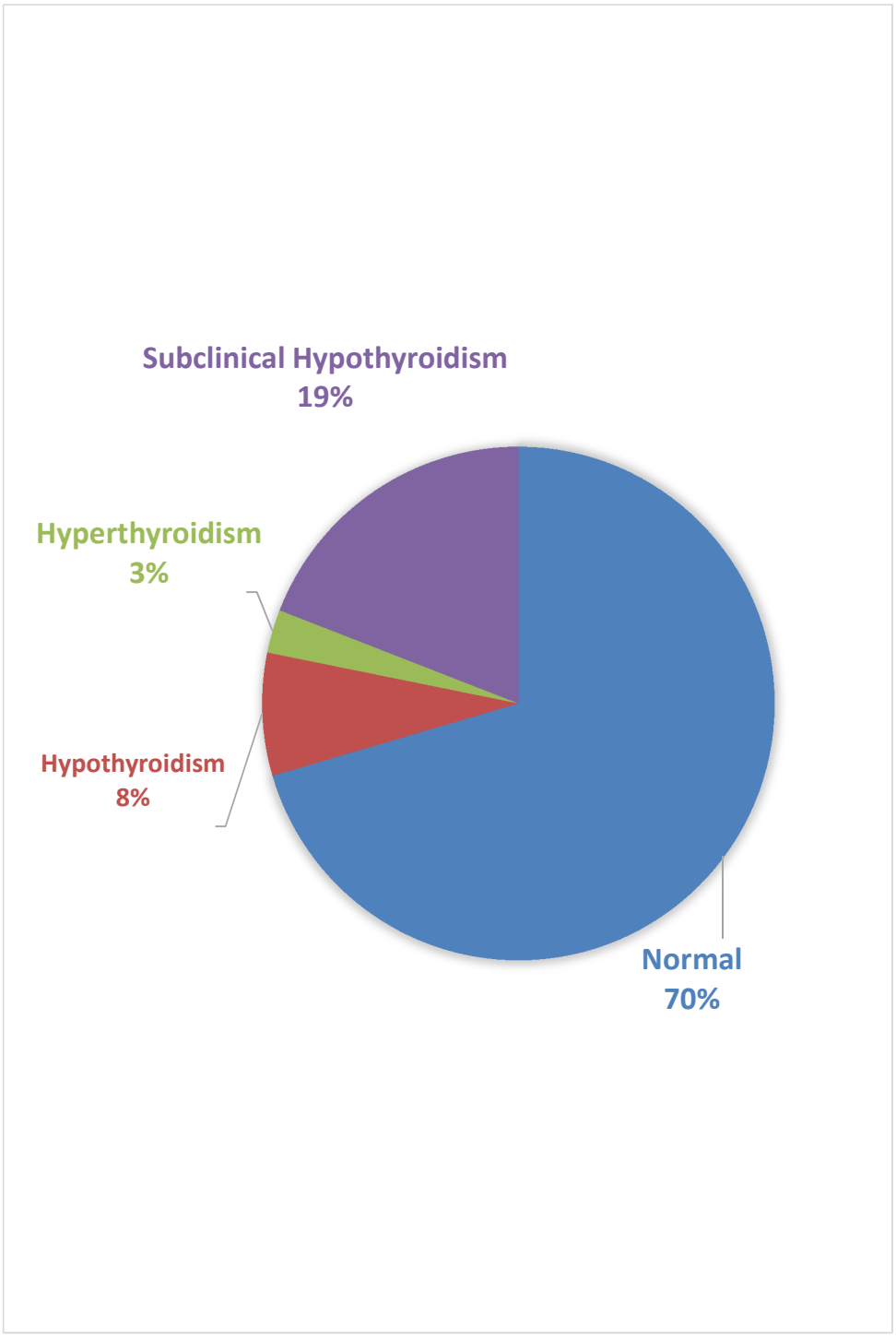


Table 12 Distribution of thyroid dysfunction in the study group and control group (n=220)

Classification	Study group (n=170)		Control group (n=50)	
	n	%	n	%
Normal	113	66.5%	42	84.0%
Thyroid dysfunction	57	33.5%	8	16.0%
Total	170	100.0%	50	100.0%

Chi-square value: 5.703 df=1 p value= ***0.017****

Comments:

1. The prevalence of thyroid dysfunction among subjects in the study group who had diabetes was 33.5% while it was 16% in the control group.
2. Considering the prevalence of thyroid dysfunction among subjects in the study group who had diabetes than the controls, the difference in prevalence (33.5% vs 16%) was statistically significant ($p<0.05$).

Table 13. Distribution of thyroid dysfunction in the study group and control group (n=220)

Classification Normal	Study group (n=170)		Control group (n=50)		'z' test p value
	n	%	n	%	
Normal	113	66.5%	42	84.0%	**<0.05***
Hypothyroidism	15	8.8%	2	4.0%	>0.05
Hyperthyroidism	6	3.5%	0	0.0%	>0.05
Subclinical Hypothyroidism	36	21.2%	6	12.0%	>0.05
Total	170	100.0%	50	100.0%	>0.05

Comments:

1. Subjects in the study group who had diabetes had higher proportion of subjects with hypothyroidism, hyperthyroidism and subclinical Hypothyroidism compared to the controls but this difference was not statistically significant ($p>0.05$).
2. But as a whole when considering the prevalence of thyroid dysfunction among subjects in the study group who had diabetes than the controls, the difference in prevalence (33.5% vs 16%) was statistically significant ($p<0.05$).

Fig 9. Bar chart showing distribution of thyroid dysfunction in the study group and control group (n=220)

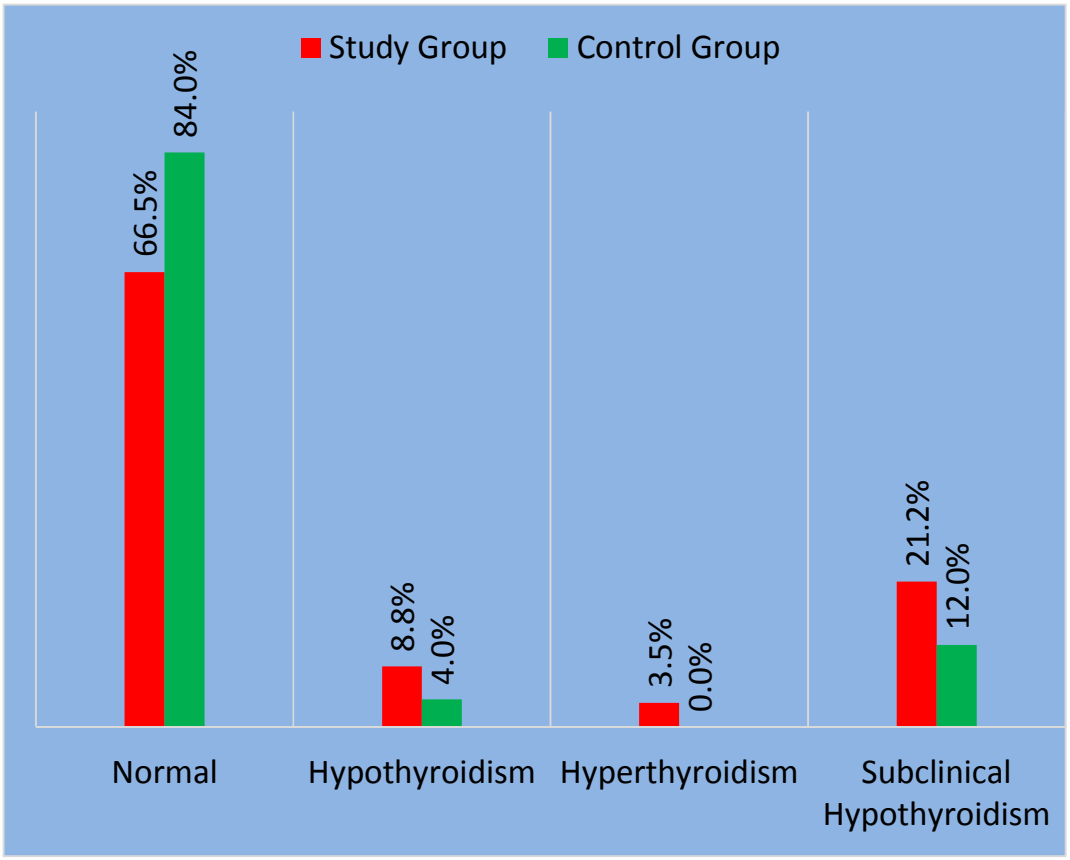


Table 14. Distribution of total cholesterol according to the thyroid status among diabetic subjects in the study group (n=170)

Thyroid status	Total cholesterol (mg/dl)					
	Normal (<200)		Elevated (>200)		Total	
Normal	84	74.3%	29	25.7%	113	100.0%
Subclinical and clinical Hypothyroidism	28	54.9%	23	45.1%	51	100.0%
Hyperthyroidism	5	83.3%	1	16.7%	6	100.0%
Total	117	68.8%	53	31.2%	170	100.0%

Chi-square value: 6.796 df = 2 p value:***0.033***

Comments:

Among the study subjects who had diabetes, the subjects with Subclinical and clinical Hypothyroidism had higher proportion of elevated total cholesterol levels (45% vs. 26% and 17%) in comparison to euthyroid and hyperthyroid subjects and this difference was statistically significant ($p<0.05$).

Fig. 10. Bar chart showing distribution of elevated total cholesterol according to the thyroid status among diabetic subjects (n=170)

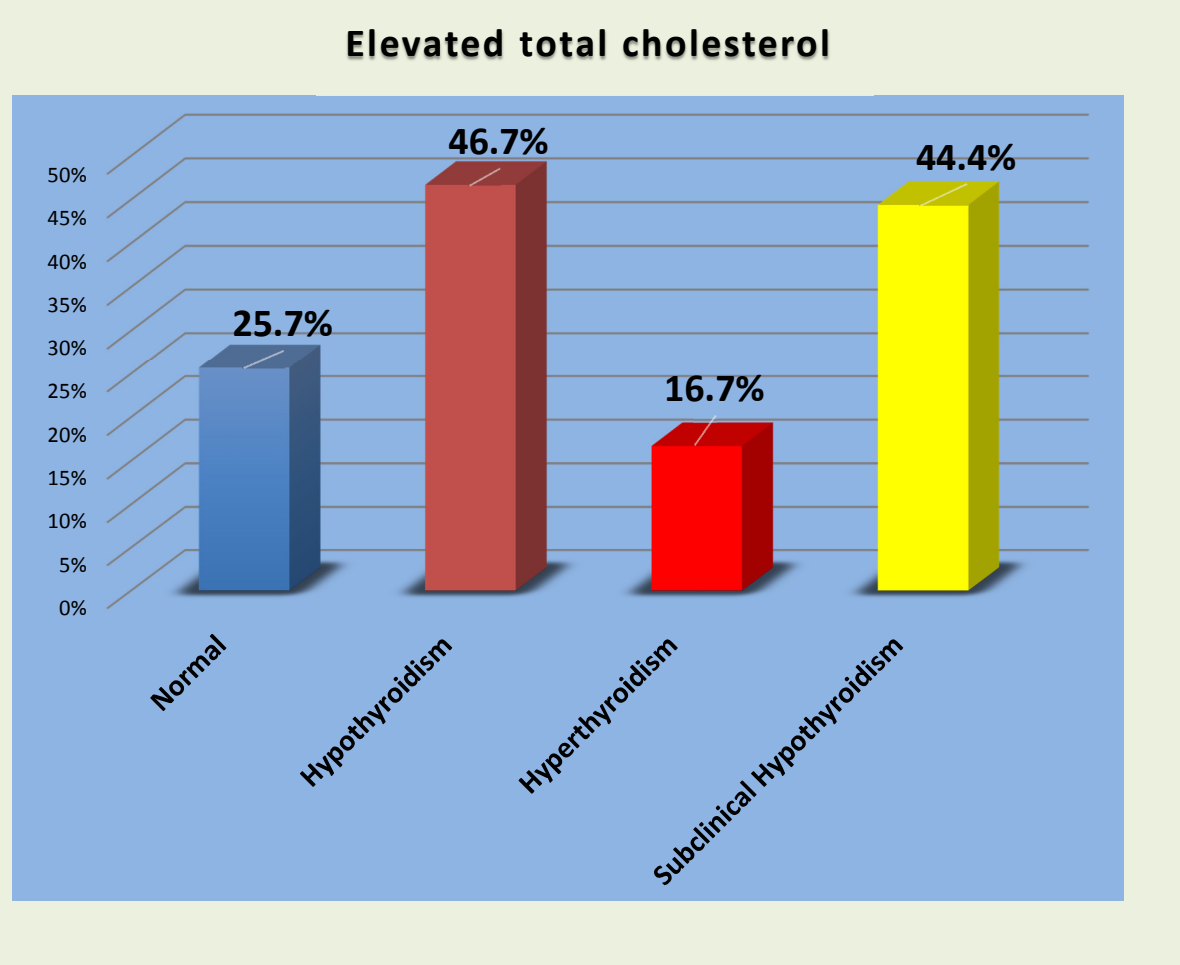


Table 15. Distribution of triglyceride according to the thyroid status among diabetic subjects in the study group (n=170)

Thyroid status	Triglycerides (mg/dl)					
	Normal (<150)		Elevated (>150)		Total	
Normal	78	69.0%	35	31.0%	113	100.0%
Subclinical and clinical Hypothyroidism	21	41.2%	30	58.8%	51	100.0%
Hyperthyroidism	4	66.7%	2	33.3%	6	100.0%
Total	103	60.6%	67	39.4%	170	100.0%

Chi-square value: 11.510 df = 2 p value: ***0.003***

Comments:

Among the study subjects who had diabetes, the subjects with Subclinical and clinical Hypothyroidism had higher proportion of elevated triglycerides levels (59% vs 31% and 33%) in comparison to euthyroid and hyperthyroid subjects and this difference was statistically significant ($p<0.05$).

Fig 11. Bar chart showing distribution of elevated triglyceride according to the thyroid status among diabetic subjects (n=170)

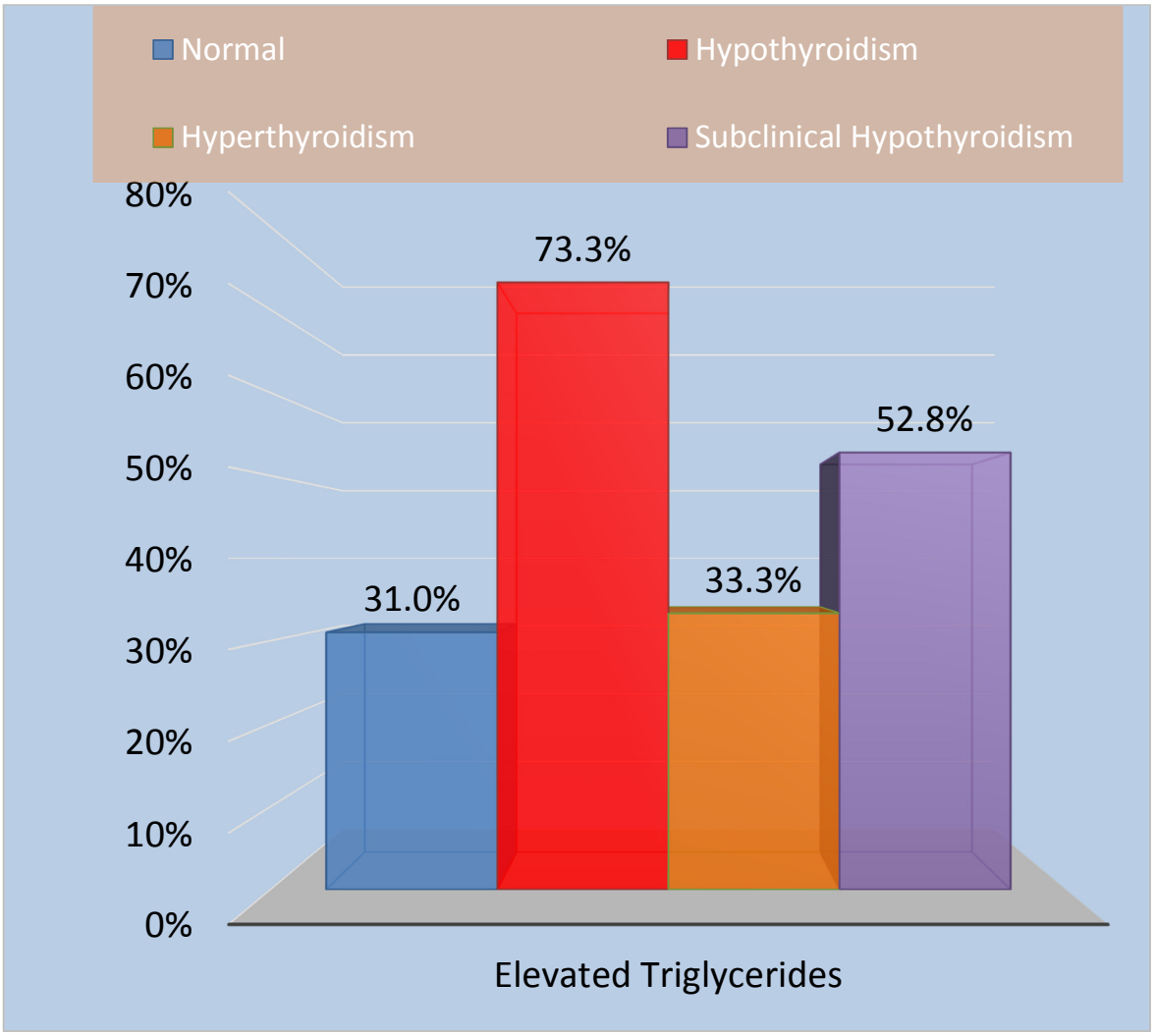


Table 16. Distribution of LDL CHOLESTEROL according to the thyroid status among diabetic subjects in the study group (n=170)

Thyroid status	LDL cholesterol (mg/dl)					
	Normal (<130)		Elevated (>130)		Total	
Normal	97	85.8%	16	14.2%	113	100.0%
Subclinical and clinical Hypothyroidism	31	60.8%	20	39.2%	51	100.0%
Hyperthyroidism	6	100.0%	0	0.0%	6	100.0%
Total	134	78.8%	36	21.2%	170	100.0%

Chi-square value: 14.888 df = 2 p value:***0.001***

Comments:

Among the study subjects who had diabetes, the subjects with Subclinical and clinical Hypothyroidism had higher proportion of elevated LDL cholesterol levels (39% vs 14% and 0%) in comparison to euthyroid and hyperthyroid subjects and this difference was statistically significant ($p<0.05$).

Fig 12. Bar chart showing distribution of elevated LDL cholesterol according to the thyroid status among diabetic subjects (n=170)

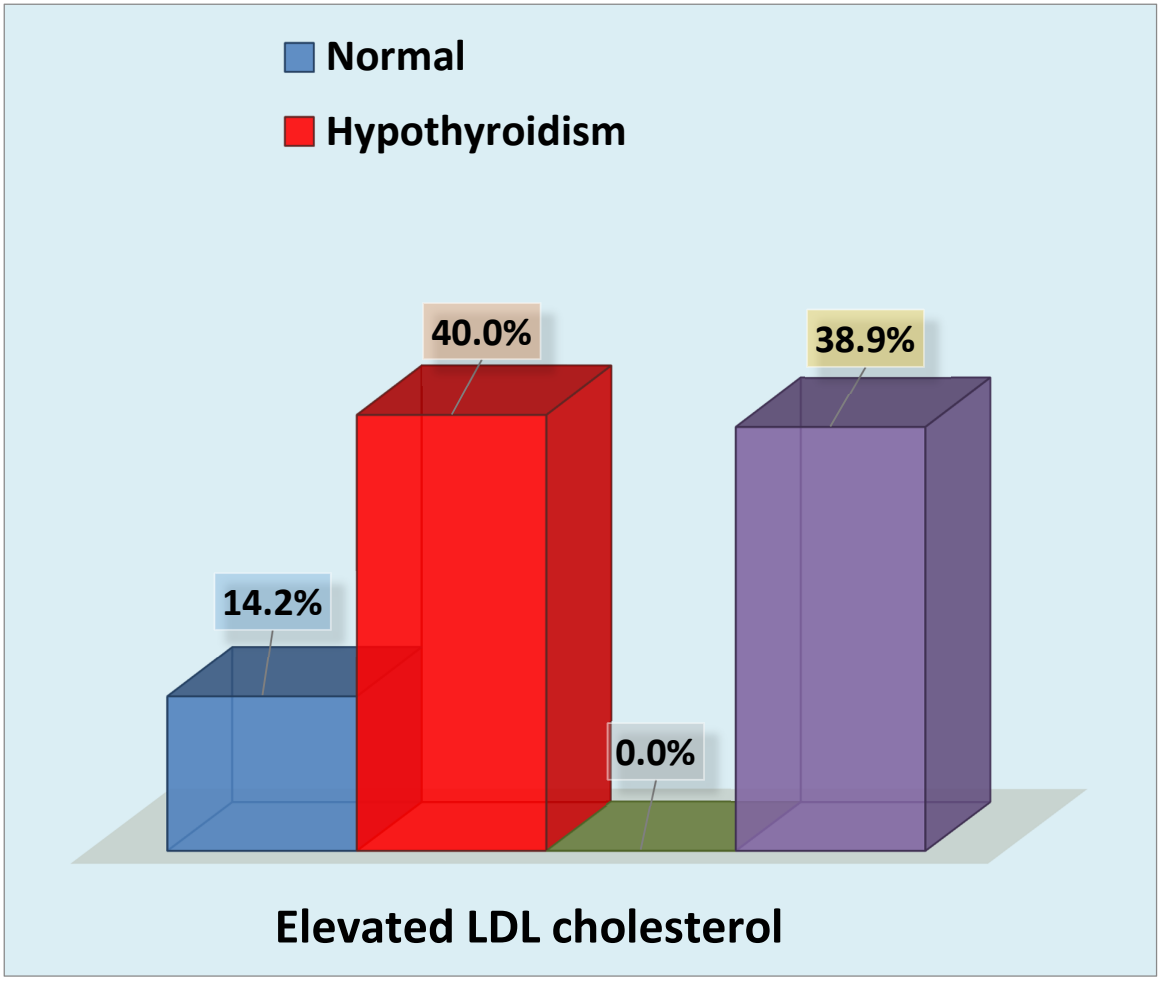


Table 17. Distribution of HDL cholesterol according to the thyroid status among diabetic subjects in the study group (n=170)

Thyroid status	HDL cholesterol (mg/dl)					
	Normal (>40 in males,>50 in females)		Reduced (<40 in males,<50 in females)		Total	
Normal	65	57.5%	48	42.5%	113	100.0%
Subclinical and clinical Hypothyroidism	16	31.4%	35	68.6%	51	100.0%
Hyperthyroidism	1	16.7%	5	83.3%	6	100.0%
Total	82	48.2%	88	51.8%	170	100.0%

Chi-square value: 12.106 df = 2 p value:***0.002***

Comments:

Among the study subjects who had diabetes, the subjects with Subclinical and clinical Hypothyroidism had higher proportion of reduced HDL cholesterol levels (69% vs 42%) in comparison to euthyroid but hyperthyroid subjects had even higher proportion of reduced HDL cholesterol levels (83%) and this difference was statistically significant ($p<0.05$).

Fig 13.Bar chart showing distribution of reduced HDL cholesterol according to the thyroid status among diabetic subjects(n=170)

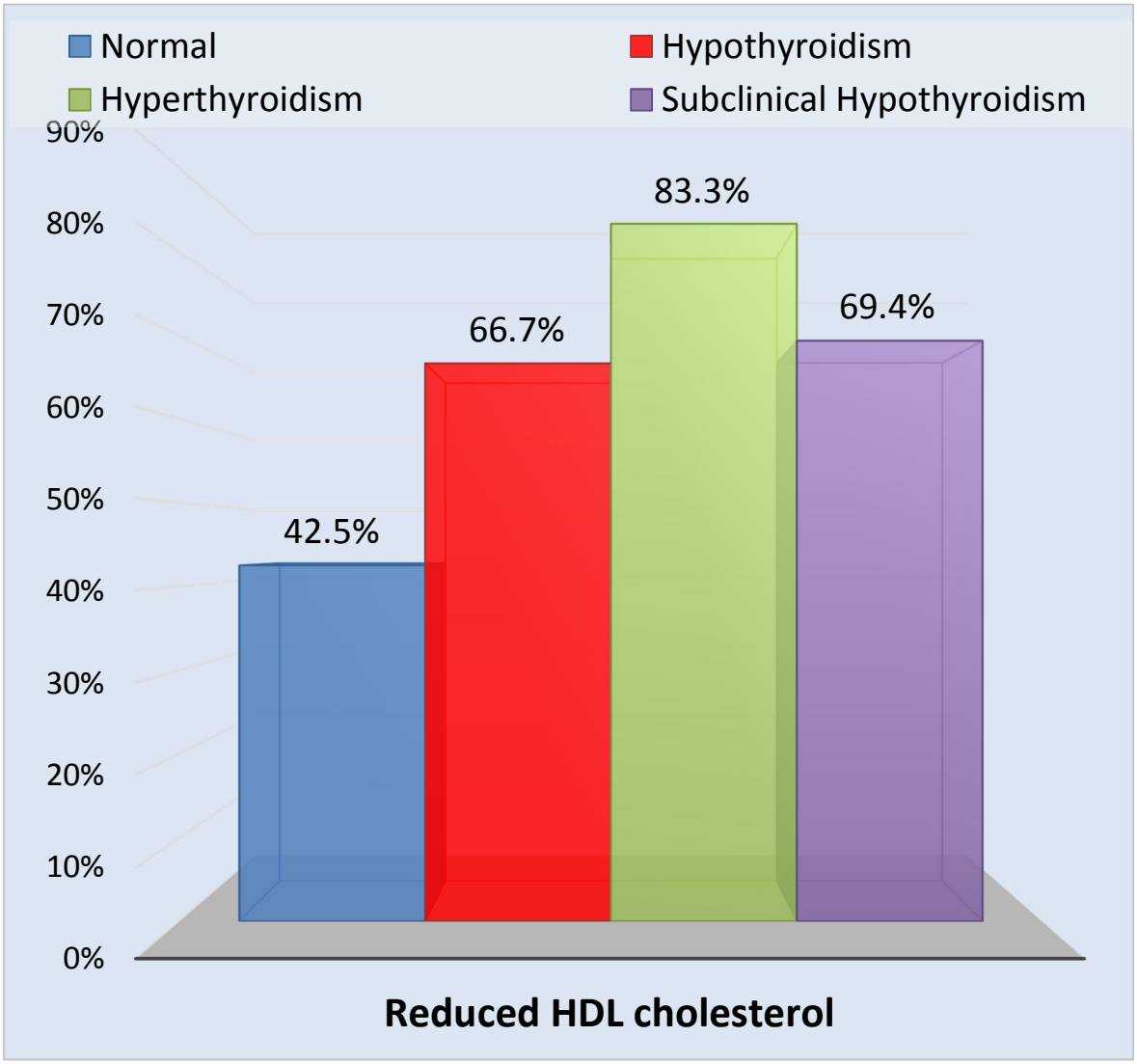


Table 18. Distribution of BMI according to the thyroid status among diabetic subjects in the study group (n=170)

Thyroid status	N	Mean BMI	Std. Deviation	95% Confidence Interval for Mean	
				Lower	Upper
Normal	113	25.4462	2.90318	24.9051	25.9873
Subclinical and clinical Hypothyroidism	51	25.4029	3.20670	24.5010	26.3048
Hyperthyroidism	6	25.0912	2.75206	22.2031	27.9794
Total	170	25.4207	2.97598	24.9701	25.8713

ANOVA test was done to test the difference in mean BMI levels between the three groups.

ANOVA test

p value	0.959
F statistic	0.041
Degree of freedom	2

Comments:

1) ANOVA test showed that there was no statistically significant difference in mean BMI between the three groups.
2) Hence the distribution of obesity and overweight was almost similar among subjects with and without thyroid dysfunction. This may be due to the fact that they were all diabetics and obesity may be present as a pre-existing and pre-disposing factor for diabetes and thyroid dysfunction.

Table 19. Distribution of HbA_{1c} according to the thyroid status among diabetic subjects in the study group (n=170)

Thyroid status	N	Mean HbA_{1c}	Std. Deviation	95% Confidence Interval for Mean	
				Lower	Upper
Normal	113	7.204	1.5664	6.912	7.496
Subclinical and clinical Hypothyroidism	51	6.939	1.7760	6.440	7.439
Hyperthyroidism	6	6.350	0.6535	5.664	7.036
Total	170	7.094	1.6145	6.850	7.339

ANOVA test was done to test the difference in mean HbA1c levels between the three groups.

ANOVA test

p value	0.324
F statistic	1.133
Degree of freedom	2

Comments:

1) ANOVA test showed that there was no statistically significant difference in mean HbA_{1c} between the three groups.
2) Hence the distribution of HbA_{1c} was almost similar among subjects with and without thyroid dysfunction.

Table 20. Gender distribution of thyroid status among diabetic subjects in the study group (n=170)

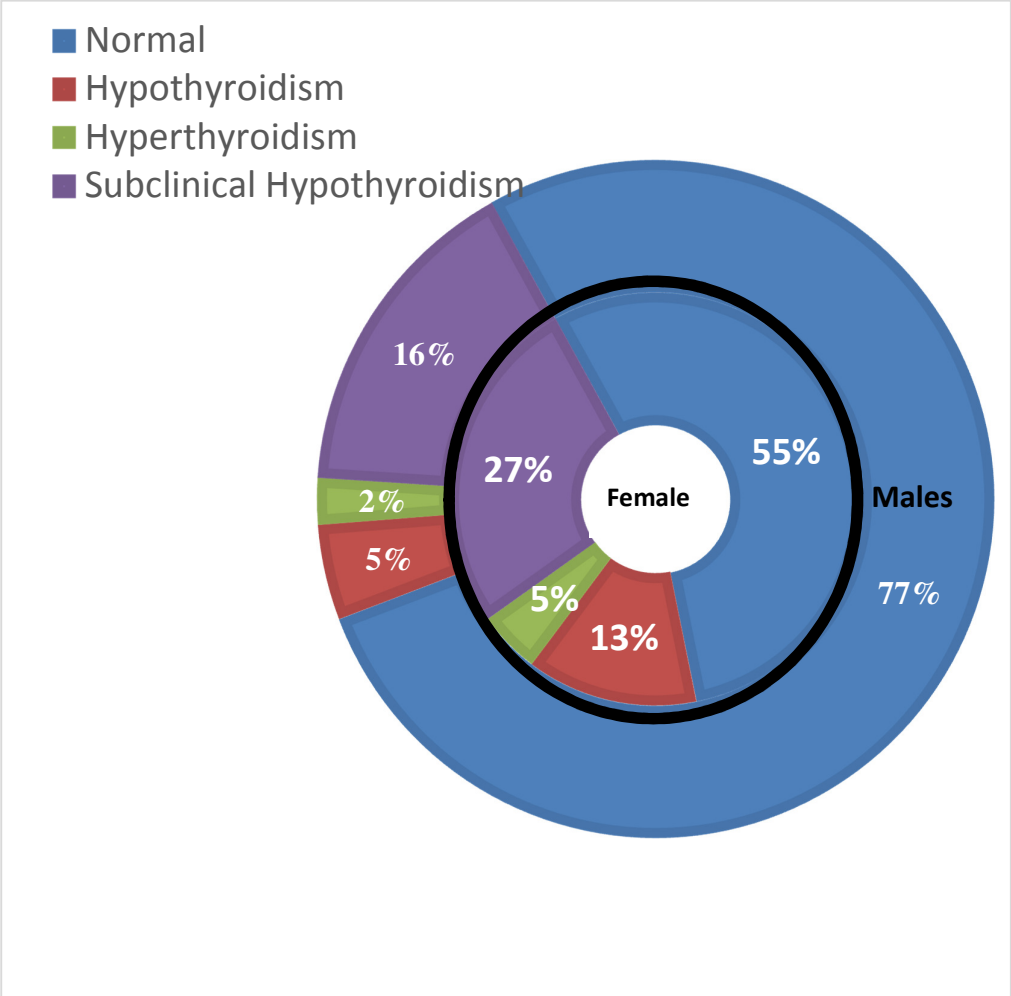
Thyroid status	Gender			
	Females		Males	
Normal	45	54.9%	68	77.3%
Hypothyroidism	11	13.4%	4	4.5%
Hyperthyroidism	4	4.9%	2	2.3%
Subclinical Hypothyroidism	22	26.8%	14	15.9%

Chi-square value: 10.193 df = 3 p value: ***0.017***

Comments:

Among the study subjects who had diabetes, female subjects had higher proportion of subclinical and clinical Hypothyroidism (combined: 40.2% vs 20.5%) in comparison to euthyroid and hyperthyroidism was also more common in females than males (4.9% vs 2.3 %) and this difference was statistically significant ($p<0.05$).

Fig 14. Doughnut chart showing gender distribution of thyroid status among diabetic subjects in the study group (n=170)



DISCUSSION

Diabetes mellitus is the most important health problem in populations worldwide and inspite of advances in treatment, a huge number of patients present with complications owing to poor glycaemic control. One of the vital factors that contribute to deprived glycaemic control is thyroid dysfunction, which tends to happen down with diabetes mellitus. This study sought to find out the prevalence of thyroid dysfunction in people with type 2 diabetes mellitus in our region.

DM is a multi-factorial disorder. There is a complex interaction between DM and thyroid disorder. Because of insulin and thyroid hormones are closely involved in cellular metabolism, any abnormal levels of one of them may result in the functional derangement of other.

In our study, we demonstrated a 33.5% prevalence of thyroid disease among 170 DM subjects compared to 16% in control group. Among this, 21.2% had subclinical hypothyroidism.it is similar to many studies like Palma et al, vinu vij et al, celani et al, singh et al and udiong et al.

Next to SCH, hypothyroidism was common (8.8%) followed by hyperthyroidism (3.5%).So total hypothyroidism is more common (30%) than hyperthyroidism. This also supported by various study like moghetti et al [76] which showed 89% hypothyroidism and 11% hyperthyroidism.

The reasons for both high and low level of thyroid hormones in diabetes are the modified TRH synthesis and release. This also due to various medications used for DM. Many studies concluded that the treatment of DM by sulfonylurea leads to an increase in occurrence of goitre and hypothyroidism . [77, 78]

SCH is defined as an above normal level of serum TSH with normal free thyroxine level.

SCH can causes outbreaks of sensitive functions like left ventricular diastolic dysfunction, lack of ovulation, increased expression of LDL receptors, and decreased HDL receptors. It is a threat indicator used for cardiovascular disease especially for atherosclerosis and coronary heart disease.SCH also independently increase the risk of insulin resistance mainly in muscles and adipose tissue.

So normalization of TSH level will decrease the post prandial blood glucose, HbA1c and lipids level. Timely diagnosis of SCH in DM patients and adequate treatment is very important to prevent complications.

In our study, serum lipid levels were elevated in DM subjects compared to normal subjects. A linear raise in serum total cholesterol, LDL Cholesterol and triglycerides, and linear decrease in serum HDL Cholesterol were observed. This also similar to Sawant et al [79] and Saha HR et al [80] studies.

The study population was predominantly male, but the prevalence of hypothyroidism was high in female at 40.2% and males at 20.5%. This is comparable to studies of Papazafiropoulou et al [81], Celani et al as well as Michalek et al [82] in which they also reported prevalence of thyroid disorders higher in diabetic females as compared to diabetic males, who found female predominance of 60%.This could be partly explained by the fact that autoimmune diseases tend to occur predominantly in females.

SUMMARY

The review of literature of many studies all over the world concluded that thyroid dysfunction and DM co-exists in significant number of patients. A recent finding shows that subclinical hypothyroidism and DM add to major problems such as retinopathy and neuropathy.

In our study overall 33.5% occurrence of thyroid dysfunction was found in Diabetic patients. Among this, highest prevalence was 21.2% of subclinical hypothyroidism followed by8.8% of hypothyroidism and 3.5% of hyperthyroidism. There was a positive association of TSH with LDL-c and TC. Further investigations are needed to understand the mechanism of lipid metabolism in Type 2 DM with respect to Thyroid function.

The unrevealed thyroid dysfunction may harmfully impact on Diabetes & its complications. Hypo Thyroidism has most important negative effect on glyceamic control and so potentially influence the complications.

Our results demonstrate that finding of thyroid dysfunction level especially sub clinical hypo thyroidism and its association with biochemical abnormalities like poor glycemic control, dyslipidemia, early will enable better treatment and prevention or delay the onset of complications. The American thyroid association guidelines recommend thyroid test in Type2 DM patients at the time of diagnosis and to be repeated at least every five years Hence it is suggested for routine screening for thyroid function in all type 2 DM patients.. A sensitive TSH assay is the screening test of choice.

CONCLUSION

Our study have proved that increased prevalence of hypothyroidism especially subclinical hypothyroidism in T2DM patients which is consistent with many previous studies, hence it may be advisable to check thyroid status in every T2DM patients for the better management of T2DM and to reduce its complications.

LIMITATION OF THE STUDY

Due to financial constraints: Unable to do thyroid autoantibody assays in this population.

Further research needs to be done with regards to thyroid antibodies bearing in mind strong family history and high prevalence of thyroid dysfunction in this population.

Our sample size was small and the limited observation period do not allow definite conclusion from our data. So we need more comprehensive study with large sample population and long period. That would be more informative.

REFERENCES

1. Shekhar Chandra Yadav, Alwin Saldhana, Biswajit Majumdar. Status of Thyroid profile In Type-2 Diabetes Mellitus. Journal of Nobel Medical College. 2012; 2: 64.
2. Banarasidas Bhanot, Park K. Textbook of preventive and social medicine. 19th ed. Jabalpur: India; 2007; 327-32.
3. WHO 1985. Diabetes mellitus. Tech. Rep. Ser.No.646.
4. Wild, S., Roglic, G., Green, A., Sicree, R. and King, H. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. Diabetes Care. 2004; 27: 1047–1053.
5. David R. Whiting A, Leonor Guariguata A , Clara Weil A , Jonathan Shaw B IDF Diabetes Atlas: Global estimates of the prevalence of diabetes for 2011 and 2030.
6. Sathisha TG, Cariappa KB, Nirmal Kumar , P Kanagavalli, and Pavithra V. The impact of T2DM on Thyroid Profile and Outcomes in a Female Population. RJPBCS. 2014; 5(2): 1554.
7. William's text book of endocrinology, Chapter 11.
8. Basha S.J, Raju DSSK, Anil Kumar M. A Study on the Association of Diabetes Mellitus Type-2 and Hypothyroidism. IOSR-JDMS.2016; 15(3): 39-42.
9. Jenna L.J, Diabetes Control in Thyroid Disease Diabetes. Spectrum. 2006; 19(3): 148-153.
10. Jameson J. L, Weetman A.P. Disorders of the thyroid gland. In: Fauci A S, Longo D L, Kasper D L,Hauser S L, Jameson J L, Loscalzo J, editors. Harrison's principles of internal medicine. 18th ed. NewYork: McGraw Hill; 2008. p. 2911.

11. Gagandeep Kaur Sidhu, Rahima Malek, Asha Khubchandani, Sohil.H.Mansuri, Miku Patel, Ruhan Oza. A Study of Serum Urea, Creatinine and Uric Acid Levels In Hypothyroid Patients**.** Int. Jr. Res. Med. 2016; 5(2): 115-118.
12. Hsiang Cheng Chi, Cheng-Yi Chen, Ming-Ming Tsai, Chung-Ying Tsai, and Kwang-Huei Lin. Molecular Functions of Thyroid Hormones and Their Clinical Significance in Liver-Related Diseases. Bio Med Research International. 2013; Article ID 601361.
13. T. Kano, T. Kojima, T. Takahashi, and Y. Muto. Serum thyroid hormone levels in patients with fulminant hepatitis: usefulness of rT3 and the rT3/T3 ratio as prognostic indices. Gastroenterologia Japonica. 1987; 22(3): 344–353.
14. Udoing CEJA, Udoh E, Etukudoh ME. Evaluation of thyroid function in diabetes mellitus in Calabar, Nigeria. Indian J. Clin. Biochem. 2007; 22: 74-7814.
15. Granner DK. Thyroid hormones. In Murray R.K,Granner DK, Mayes PA, Rodwell VW. Harper's Biochemistry, 25th ed. London: Prentice-Hall International Inc. 2000; 533- 38.
16. Sathish R., Mahan V. Diabetes & thyroid disease a review. Int. J. Diab. Dev. countries. 2003; 23(120): 1-3.
17. Chaoxun Wang, The Relationship between Type 2 Diabetes Mellitus and Related Thyroid Diseases. Journal of Diabetes Research. 2013; Article ID 390534.
18. Dimitriadis, Raptis. Thyroid hormone excess and glucose intolerance. Experimental and Clinical Endocrinology and Diabetes. 2001; 109(2): S225–S239.
19. Ueckermann V, Van Zyl DG. The prevalence of subclinical hypothyroidism among patients with diabetes mellitus at the Kalafong Hospital Diabetes Clinic: a

cross-sectional study. Journal of Endocrinology, Metabolism and Diabetes of South Africa. 2013; 18(2): 106-110.

20. Kadiyala R, Peter R, Okosieme OE. Thyroid dysfunction in patients with diabetes: clinical implications and screening strategies. Int. J. Clin. Pract. 2010; 64(8):1130-1139.
21. Singh G, Gupta V, Sharma AK, Gupta N. Evaluation of thyroid dysfunction among type 2 diabetic Punjabi population. Adv Biores. 2011; 2(2): 3-9.
22. Perros, P., Mc Crimmon, R.J., Shaw, G. and Frier, B.M. Frequency of thyroid dysfunction in diabetics patients: value of annual screening. Diabet Med. 1995; 12 (7): 622-627.
23. Celani M.F, Bonati M.E, Stucci N. Prevalence of abnormal thyrotropin concentrations measured by a sensitive assay in patients with Type 2 diabetes mellitus. Diabete Res. 1994; 27(1):15-25.
24. Radetti, Paganini, Gentill et al. Frequency of Hashimoto's thyroiditis in children with type 1 diabetes mellitus. Acta Diabetologica. 1995; 32(2): 121–124.
25. Richard I. G. Holt, Clive Cockram, Allan Flyvbjerg, Barry J. Goldstein. Textbook of Diabetes. 4th ed. p - 69.
26. A Nicholas. Type 2 diabetes mellitus and thyroid dysfunction: an intertwined duo. African Journal of Diabetes Medicine. 2014; 22(2):
27. World Health Organization. Report of WHO study group. WHO Technical Report Series. 1985: No 727.
28. Global report on diabetes. World Health Organization, Geneva, 2016.
29. International Diabetes Federation, 2015. https://issuu.com/int._diabetes_federation/docs/idf_atlas_2015_uk.

30. Dan L. Longo et al. Harrison's Principles Of Internal Medicine. 19th ed. 2015, chapter 417, page no: 2399-2406.
31. Ratna Manjula Songa, Viswabarathi, Siddhartha K. Comparative study of lipid profile in obese type 2 diabetes mellitus and obese non diabetes. 2015; 14(11):87-91.
32. Zimmat P, Alberti G, Shaw J. Global and societal implications of the diabetes epidemic. Nature. 2001; 414 (6865):782-7.
33. Onovughe Joseph Akporea et al. Prevalence of Hypertension and Diabetes Mellitus among Individuals Attending a Health Fair in Dominica. ASRJETS. 2016; 19(1): 74-84.
34. Chamany, Tabaei. Text book of Principles of Diabetes Mellitus. 2nd ed. p118.
35. Peeyush Yadav, Kaushik, Sonali Sharma. Importance of Screening Type-II Diabetics for Thyroid Dysfunction and Dyslipidemia. International Journal of Biochemistry and Biophysics. 2015; 3(2): 7-12.
36. American Diabetes Association. Diagnosis and Classification of Diabetes Mellitus. 2010; 33(1): S62–S69.
37. Ozougwu C, Obimba C, Belonwu D, Unakalamba B. The pathogenesis and pathophysiology of type 1 and type 2 diabetes mellitus Journal of Physiology and Pathophysiology. 2013; 4(4): 46-57.
38. Fuad AlSaraj. Pathogenesis of Type 2 Diabetes Mellitus http://dx.doi.org/10.5772/59183.
39. Gisela Wilcox. Insulin and Insulin Resistance. Clin Biochem Rev. 2005;26:
40. Carl Burtis, Edward Ashwood, David Bruns. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics. 5th ed. page 1416.

41. Mitrou, Raptis, Dimitriadis. Insulin action in hyperthyroidism: a focus on muscle and adipose tissue. Endocrine Reviews. 2010; 31(5): 663–679.
42. Fredric Wondisford, Sally Radovick. Clinical Management of Thyroid Disease. 1st ed. 2009, ch:4, pg 44-51.
43. Radaideh, Nusier, Amari et al. Thyroid dysfunction in patients with Type 2 diabetes mellitus in Jordan. Saudi Medical Journal. 2004; 25(8):1046–1050.
44. Anuradha Deuri, Thakuria, Kalita. A Prospective Study of Thyroid Dysfunction in Patients with Type2 Diabetes Mellitus in A Tertiary Care Hospital. Assam, India. e-ISSN: 2279-0853, p-ISSN: 2279-0861. 2016; 15(7): 21-25.
45. Vergara Palma C, Pavesi M, Nogueira G, Clemente EL, Pereira Vasconcellos M, Carlos Pereira L, et al. Prevalence of thyroid dysfunction in patients with diabetes mellitus. Diabetology and metabolic syndrome. 2013; 5(58): 1-2.
46. Cutolo M, Sulli A, Capellino S, et al. Sex hormones influence on the immune system: basic and clinical aspects in autoimmunity. Lupus. 2004; 13(9): 635-638.
47. Capen CC: Anatomy. In Werner and Ingbar's. The Thyroid. 7th ed. Braverman; Utiger RD, Eds. Philadelphia, Lippincott-Raven. 1996: p.19–38.
48. Louis, Mo, Mosby. Porterfield SP: The thyroid gland. In Endocrine Physiology. 1997: p.57–81.
49. Jennal Johnson. Diabetes Control in Thyroid Disease. Diabetes Spectrum. 2006; 19(3): 148-153.
50. Edina Bilic-Komarica, Amela Beciragic, Dzelaludin Junuzovic. The Importance of HbA1c Control in Patients with Subclinical Hypothyroidism. Mat Soc Med. 2012; 24(4): 212-219.

51. Diez J, Iglesias P, Burman D. Spontaneous normalization of thyrotropin concentations in patients with subclinical hypothyreoidism. J Clin Endocrinol Metab. 2005; 90: 4124-4127.
52. Dan, Longo et al. Harrison´s Principles of Internal Medicine. 19th ed. 2015. Chapter 405, pages 2283-2287.
53. Venkatachalam Ramesh, Rajagopalan Geetha, Devaraj Anitha, NRVK Swamy Thangarajan Thanga Panneerselvam. The Study of Thyroid Dysfunction among Type 2 Diabetic Patients. 2015; 3(9): 14-18.
54. Vikram Vikhe, Shubhangi Kanitkar, Krunal Tamakuwala, Anu Gaikwad, Meenakshi Kalyan, Rajani Agarwal. Thyroid Dysfunction In Patients With Type 2 Diabetes Mellitus At Tertiary Care Centre. National Journal Of Medical Research. 2013; 3(4):
55. Vibha Uppal, Chittranjan Vij, Gurdeep Kaur Bedi, Anil Vij, and Basu Dev Banerjee Thyroid Disorders in Patients of Type 2 Diabetes Mellitus. Indian J Clin Biochem. 2013; 28(4):
56. Collar FA, Huggeri CB. Effects of Hyperthyroidism upon Diabetes Mellitus Striking Improvement in Diabetes Mellitus from Thyroidectomy. Am Surg. 1927; 86(6): 877-84.
57. Chen J, Wu Y, Lin et al. Associations between cardiovascular risk, insulin resistance, β-cell function and thyroid dysfunction: a cross-sectional study in She ethnic minority group of Fujian Province in China. European Journal of Endocrinology. 2010; 163(5): 775–782.
58. Brenta S, Celi M, Pisarev M, Schnitman I, Sinay P. Arias A. Acute thyroid hormone withdrawal in athyreotic patients results in a state of insulin resistance. Thyroid. 2009; 19(6): 665–669.

59. Yun Zhang,Ping Lu, Ling Zhang, And Xinhua Xiao, Association between lipids profile and thyroid parameters in euthyroid diabetic subjects: a cross-sectional study. BMC Endocrine Disorders. 2015; 15:12.
60. Hyden J, Reaven P. Cardiovascular disease in diabetes mellitus type (2). J Lipid Research. 2000; 11: 519-528.
61. Dimitriadis S, Raptis A. Thyroid hormone excess and glucose intolerance. Experimental and Clinical Endocrinology and Diabetes. 2001; 109(2): S225–S239.
62. Asmabi Makandar, Amit D Sonagra, Nadia Shafi. Study Of Thyroid Function In Type 2 Diabetic And Non-Diabetic Population. International Journal of Medical Science and Public Health. 2015; 4(6):
63. Wu P. Thyroid disease and diabetes. Clin Diab. 2000; 18(1): 1–10.
64. Vikram Vikhe, Shubhangi Kanitkar, Krunal Tamakuwala, Anu Gaikwad, Meenakshi Kalyan, Rajani Agarwal. Thyroid Dysfunction in Patients With Type 2 Diabetes Mellitus At Tertiary Care Centre. National Journal Of Medical Research. 2013; 3(4).
65. Chubba A, Davis A, Inman Z, Davis M. Prevalence and progression of subclinical hypothyroidism in women with type 2 diabetes. The Fremantle Diabetes Study Cli Endocrinol (oxf). 2005; 62(4): 480-486.
66. Bakker L, Ter Maaten C, Popp-Snijders C, Slaets J, Heine J, Gans B. The relationship between thyrotropin and low density lipoprotein cholesterol is modified by insulin sensitivity in healthy euthyroid subjects. J Clin Endocrinol Metab. 2001; 86: 1206–1211.

67. Bharadiya Amit A, Swati C, Aundhkar, Jaju Jyotsna B,Bharadiya Rahi R. Evaluation of Thyroid Functions Test in Patients with Uncontrolled Type 2 Diabetes Mellitus. IJHSR. 2014; 4(5):
68. Shanmugam S, Damodharan S, Thomas Jacob J. Prevalence of thyroid dysfunction in patients with diabetes mellitus. Int J Res Med Sci. 2015; 3: 3629-33.
69. Potenza, Via, Yanagisawa. Excess thyroid hormone and carbohydrate metabolism. Endocrine Practice. 2009; 15(3): 254–262.
70. Devi A, Singh S, Singh S. Thyroid hormone dysfunctions in type 2 diabetic patients in urban areas of Manipur. International Journal of Pharmaceutical Science Invention. 2013; 2(10): 7-9.
71. Palanisamy Pasupathi, Govindaswamy Bakthavathsalam, Ganesan Saravanan, Ramachandran Sundaramoorthi. Screening for Thyroid Dysfunction in the Diabetic/Non-Diabetic Population. Thyroid Science. 2008; 3(8): 1-6.
72. Gursoy NT, Tuncel E. The relationship between the glycemic control and the hypothalamus-pituitary-thyroid axis in diabetic patients. Turkish Journal of Endocrinology and Metabolism. 1999; 4:163–168.
73. Mirella Hage, Mira S. Zantout, Sami Azar. Thyroid Disorders and Diabetes Mellitus. Journal of Thyroid Research. 2011; Volume 2011, Article ID 439463.
74. Carl Burtis, Edward Ashwood, David Bruns. Tietz Textbook of Clinical Chemistry and Molecular Diagnostics. 5th ed.
75. Leonidas Duntas, Jacques Orgiazzi, Georg Brabant. The Interface between Thyroid and Diabetes Mellitus. Clinical Endocrinology. 2011; 75: 1–9.
76. Moghetti P, Castello R, Tosi F, Zenti G, Magnani C, Bolner A, et al. Glucose counter regulatory response to acute hypoglycemia in hyperthyroid human subjects. J Clin Endocrinol Metab. 1994; 78: 169–173.

77. Bassyouni A, Ebrashy IE, Ismiel A, Amara I, Mahfouz M, Halmy N. Profile of the thyroid function and ultrasound among patients with type-2 diabetes mellitus. Sci Med J 2010; 22(2): 15–28.

78. Shaikh AW, Memon AS, Sirichand. Frequency of Hypothyroidism in Type 2 Diabetic Patients. Pakistan J Med Health Sci. 2009; 2(4).

79. Sawant M, Shetty D, Mankeshwar R, Ashavaid F. Prevalence of Dyslipidemia in Young Adult Indian Population. JAPI. 2008; 56: 99-102.

80. Saha HR, Sarkar BC, Khan SA, Sana NK, Choudhury S. A Comparative Study of Thyroid Hormone and Lipid Status in Diabetic and Non Diabetic Adults.2012; 1:450.

81. Papazafiropoulou A, Sotiropoulos A, Kokolaki A et al. Prevalence of Thyroid Dysfunction Among Greek Type 2 Diabetic Patients Attending an Outpatient Clinic. J Clinical Medicine Research. 2010; 2 (2): 75-78.

82. Michalek M, Mahoney C, Calebaugh D. Hypothyroidism and diabetes mellitus in an American Indian population. J Family Practice. 2000; 49(7): 53-5.

83. Rizos, Elisaf, Liberopoulos. Effects of Thyroid Dysfunction on Lipid Profile. The Open Cardiovascular Medicine Journal. 2011; 5: 76-84.

84. Kumar V, Abbas, Schoen J. Pathology Basics of Disease. Chapter 19. 11th ed.

85. Trinder P . Annals.Bio Chem. 6, 24.

86. Guder WG, Zawta B et al. The Quality Of Diagnostic Samples.1st ed. Darmstadt: GID Verlag: 2001:p. 22-3

87. Sterling L. Diagnosis and treatment of thyroid disease. Cleveland: CRC Press; 1975: 19-51.

88. Friedewald , W.T.,Levy, R.I., Fredsickson D.S.(1972), Clin.Chem,18,499-502.

CASE RECORD FORM

A Study on Thyroid Profile Status in Type 2 Diabetes Mellitus

Case no **:** Date :

Name :

Op/ Ip no : unit :

Age :

Sex :

Location : Urban / Rural

Occupation :

Height : Weight: BMI:

Complaints :

H/o illness :

Duration of DM :

Under Medication or Not :

If, Yes - Drug Details :

Past History :

Personal History :

Family History :

General Examination :

Systemic Examination :

Date of sample collection :

Date of Analysis :

INVESTIGATION :

Diabetic profile : (F) Glucose (PP) Glucose
HbA1c :

Renal profile : Urea: Creatinine :

Lipid profile : Tc HDLc : LDLc :
Triglycerides:

Thyroid profile : FT_3: FT_4 :
TSH:

Signature of Investigator

A Thyroid Profile status in type 2 Diabetes Mellitus - Case Group

S.NO	AGE	SEX	HEIGHT cm	WEIGHT kg	BMI	Syst.BP mm Hg	Dias.BP mm Hg	FBS mg/dl	PPBS mg/dl	HbA1C %	UREA mg/dl	CREATININE mg/dl	T.CHO mg/dl	TRIGLY mg/dl	LDL mg/dl	HDL mg/dl	T3 pg/ml	T4 ng/dl	TSH m IU/ml
1	57	M	165	70	25.711662	120	80	150	356	6	28	1.3	160	87	78	57	1.5	1.6	1.3
2	60	F	160	78	30.46875	136	90	233	341	9.6	15	0.9	189	73	163	39	4	1.9	8.1
3	45	M	175	65	21.22449	120	80	83	188	5.9	24	1	165	111	84	47	3.9	1.5	1
4	38	F	142	60	29.756001	110	70	341	486	10.4	15	0.7	190	234	118	35	2	1.7	1.7
5	60	M	165	80	29.384757	140	78	160	241	6.4	18	0.8	153	84	87	47	2.8	1.4	1.2
6	58	F	150	60	26.666667	140	86	110	153	5.9	19	0.7	218	117	139	51	2.9	1.4	1.2
7	59	M	164	70	26.026175	130	80	100	130	6	16	0.8	156	64	91	39	2.2	1.6	1.2
8	55	F	155	54	22.476587	130	86	95	119	5.2	23	0.7	185	127	112	40	2.2	1.4	18.9
9	58	F	152	60	25.969529	140	94	99	174	6.3	29	0.9	284	222	105	51	3.2	1.4	1.3
10	44	F	145	65	30.915577	140	90	123	150	5.6	17	0.8	192	124	113	41	3.1	0.9	13.3
11	49	F	152	64	27.700831	130	90	138	160	6.2	20	0.8	190	136	109	40	2.8	1.2	4
12	47	F	156	70	28.763971	120	80	218	378	10.9	35	0.9	208	265	105	69	2.5	1.7	5.8
13	60	M	172	50	16.901028	146	80	131	266	6.8	27	0.9	166	112	95	38	3	1.3	4.5
14	53	M	165	72	26.446281	130	96	168	260	8.6	29	1	220	116	136	50	1.4	1.4	2.4
15	57	F	150	62	27.555556	130	70	172	301	10.8	17	0.7	281	98	105	52	2.1	1.5	1.8
16	52	M	180	78	24.074074	140	90	145	315	10.6	22	0.6	169	80	96	49	3.1	1.5	1.8
17	61	M	163	70	26.346494	130	90	141	271	6.5	31	0.9	127	366	73	18	1.9	1.6	4.4
18	65	M	165	72	26.446281	140	90	225	463	9.8	42	1	202	103	136	34	2.7	1.7	4.3
19	37	M	172	75	25.351541	120	80	85	161	5.7	18	1	192	168	108	44	3	1.6	3.9
20	43	M	165	70	25.711662	110	70	122	214	6.2	19	1	179	182	108	54	3.4	1.5	0.7
21	46	F	165	72	26.446281	160	74	215	339	9.2	15	0.7	163	82	96	51	2.3	1.1	3.6
22	65	M	160	64	25	130	90	104	249	6.6	26	1	139	157	74	35	2.3	2.1	3.6
23	45	M	174	65	21.46915	130	86	125	230	6.9	16	1.1	190	117	110	45	4.5	1.3	1.2
24	62	M	162	55	20.957171	110	70	298	400	10.2	20	1.2	229	132	144	36	2.1	1.3	1.7
25	60	F	165	75	27.548209	156	94	138	229	7	25	0.9	158	154	76	46	2.2	1.4	2.3
26	63	F	155	48	19.979188	110	80	163	322	7.6	16	0.8	214	93	124	59	1.9	0.9	2.4
27	37	F	154	60	25.299376	110	70	235	345	9.6	19	0.7	204	118	146	46	1.3	1.2	4.1
28	44	M	175	65	21.22449	136	80	293	427	9	23	1.1	232	212	137	38	2.7	1.7	2.3
29	37	M	160	71	27.734375	146	84	316	454	10.2	25	1	166	84	82	57	1.8	1	21.5
30	70	M	160	48	18.75	160	90	144	194	5.7	21	1.6	235	80	145	49	2.2	1.4	2
31	58	M	160	64	25	130	80	106	164	5.8	28	1.3	249	181	151	38	2.4	1.5	6.9

32	43	F	156	60	24.654832	130	86	94	168	5.9	15	0.6	149	134	75	38	2.9	1.4	1.1
33	38	F	158	57	22.832879	110	70	93	185	5.7	25	0.8	224	127	169	58	1.5	0.5	35.5
34	78	M	170	70	24.221453	140	90	98	162	5.8	23	1	143	66	81	38	2.9	1.4	4
35	36	M	165	65	23.875115	150	90	101	162	5.8	15	0.9	307	102	196	51	2.6	1.6	4.5
36	33	M	164	57	21.192742	110	70	204	294	6.8	23	0.9	183	193	101	53	2.6	1.5	1.6
37	53	M	162	74	28.196921	130	90	138	276	6	20	1.2	137	128	76	37	2.3	1.3	3.6
38	38	M	165	72	26.446281	126	80	191	373	6.9	16	0.8	163	172	93	47	2.7	1.8	4
39	60	F	150	65	28.888889	140	90	312	424	10.3	28	1	182	149	110	39	2.2	1.4	0.8
40	62	F	152	68	29.432133	130	80	117	316	5.5	24	0.8	202	139	96	42	11.6	4.7	0.05
41	57	F	150	65	28.888889	150	84	125	196	5.4	17	0.7	190	103	108	46	2.8	1.3	2.7
42	46	M	170	65	22.491349	130	80	237	366	10.3	24	0.9	182	94	116	35	2.3	1.3	0.8
43	63	M	164	62	23.051755	130	90	127	252	6.3	25	0.9	157	273	94	32	3	1.2	7.2
44	52	F	160	68	26.5625	130	96	113	123	5.8	15	0.7	220	74	162	65	2.7	1.4	9.7
45	59	F	150	64	28.444444	110	70	135	199	6.2	15	0.8	136	210	72	58	2.4	1	3
46	49	F	150	48	21.333333	120	70	106	167	5.7	19	0.7	169	235	103	45	2.6	1.5	1.5
47	45	M	168	65	23.030045	146	90	139	164	5.6	25	1	211	243	118	39	2.7	1.6	1.5
48	50	F	150	62	27.555556	130	70	221	375	9.2	15	0.7	156	95	84	44	2.3	1.5	3.6
49	67	F	145	60	28.537455	110	60	120	145	6.1	16	1.1	147	152	71	36	1.6	0.7	4.5
50	65	F	150	62	27.555556	140	90	104	145	5.7	20	0.8	245	243	149	39	2.2	0.3	2.7
51	55	M	170	67	23.183391	130	85	118	142	5.4	19	1.4	102	210	46	30	0.5	0.2	37.5
52	42	M	160	75	29.296875	120	70	166	290	6.3	15	1	195	173	114	39	2.4	1.6	4.1
53	35	F	150	62	27.555556	150	80	388	394	8.4	28	0.9	266	78	96	41	2.6	1.5	0.9
54	47	F	147	60	27.766209	130	70	128	219	7.2	26	0.9	225	160	131	44	2.2	1.2	4
55	56	F	150	63	28	130	90	119	264	7.4	15	0.9	138	125	73	41	2.6	1.5	1.2
56	40	M	165	69	25.344353	126	86	87	162	5.6	21	1.1	208	117	166	48	1.9	0.7	3.5
57	49	F	162	58	22.10029	110	70	115	120	5.3	16	0.8	190	176	106	40	2.6	1	10.3
58	38	M	167	76	27.250887	150	80	83	155	5.7	25	0.8	249	93	92	36	2.4	1.6	1.5
59	66	M	176	90	29.054752	140	90	103	196	6.2	22	1	134	97	72	36	2.8	1.1	2.1
60	54	F	147	50	23.138507	140	70	114	136	5.9	29	0.7	162	151	82	44	2.9	1.9	0.05
61	50	F	152	67	28.999307	150	86	132	126	5.7	19	0.8	315	187	188	58	3.1	0.4	6.7
62	43	F	146	52	24.394821	110	70	91	102	5.2	15	0.7	186	134	111	42	2.3	1.3	1.8
63	38	F	160	80	31.25	110	80	119	167	7.8	16	0.7	193	119	113	40	2.7	1.8	0.6
64	58	F	150	57	25.333333	120	80	129	226	11.6	27	0.8	147	271	111	43	2.7	0.7	20.8
65	45	F	152	60	25.969529	120	70	176	256	8.4	21	0.7	161	132	93	38	2	1	2.4
66	60	M	170	76	26.297578	130	86	90	182	6.3	15	0.9	186	97	118	37	2.4	0.9	3
67	35	M	158	60	24.03461	140	80	371	564	10.8	18	0.9	151	101	79	46	1.9	1.8	2.5
68	47	M	170	72	24.913495	120	90	229	355	9.6	16	0.9	177	174	100	36	2	1.4	2.6
69	52	F	140	52	26.530612	140	90	153	201	7.2	15	0.8	162	73	80	47	2.3	1.8	0.1
70	33	M	157	65	26.370238	126	80	101	217	7	17	1.2	172	222	92	49	2.2	1.3	2.9

71	70	M	165	60	22.038567	128	76	103	248	7.4	24	1	148	62	83	39	2.5	1.3	1.7
72	49	M	165	58	21.303949	140	80	569	674	11.6	29	1.1	278	74	100	47	2.5	1.9	3.3
73	40	F	154	63	26.564345	120	80	174	369	8.6	17	0.8	200	199	95	44	2.2	1.2	2.4
74	63	F	150	60	26.666667	140	96	93	302	8	15	0.9	155	227	70	48	2.8	1.5	4.8
75	45	F	149	67	30.178821	110	70	145	179	7.2	28	0.7	276	187	95	54	2.2	1.4	1.4
76	42	F	158	65	26.037494	120	86	222	437	8.6	16	0.8	145	160	94	40	3.4	1.3	5.8
77	57	F	160	58	22.65625	140	80	91	171	5.7	33	0.9	277	321	141	62	2.1	1.3	5
78	46	F	154	75	31.62422	120	76	160	201	5.9	23	1	230	330	170	46	0.9	0.6	37.2
79	65	M	168	74	26.218821	140	80	169	294	8.5	36	0.9	181	189	96	37	1.2	1.4	5.9
80	67	M	165	68	24.977043	130	86	145	196	6	25	0.8	122	69	56	40	2.5	2.1	1.2
81	48	M	158	64	25.636917	130	90	99	162	5.8	16	1	211	127	118	41	2.3	1.6	2.3
82	67	F	155	63	26.222685	110	80	126	250	6.3	24	0.8	154	170	84	39	2.6	1.1	0.9
83	56	M	165	60	22.038567	136	90	125	220	6.7	15	1	177	98	104	37	3	1.5	1.7
84	43	F	145	63	29.964328	120	80	110	193	6.2	16	0.8	241	224	142	45	3.1	1.2	1.2
85	66	M	160	72	28.125	140	96	100	160	5.8	18	1.2	185	213	99	36	1.3	0.3	36.5
86	55	F	148	60	27.392257	120	90	229	363	8.3	21	0.7	177	135	99	51	2.2	1.8	1.1
87	33	M	174	68	22.460034	130	70	139	212	7.2	22	0.8	198	92	119	38	2.1	1.6	2.6
88	65	M	156	54	22.189349	110	70	173	422	8.8	44	1	172	73	83	62	1.7	1.8	1.1
89	63	F	154	75	31.62422	150	96	169	198	6.2	29	0.9	194	129	98	71	3	1.9	1.6
90	48	F	160	74	28.90625	130	80	217	402	8.9	19	0.7	208	138	134	62	2.1	1.5	1.8
91	37	F	164	58	21.564545	110	70	160	350	9.4	23	0.6	180	119	105	42	2.6	2	6.4
92	46	M	162	62	23.624447	130	80	122	265	6.4	20	0.9	172	82	110	35	2.4	1.2	6.2
93	51	M	170	74	25.605536	130	94	102	184	5.8	22	1	171	117	127	39	2.3	1.2	3
94	50	F	154	63	26.564345	130	80	278	349	8.4	24	0.8	211	87	130	41	2	1.2	3.9
95	58	M	168	78	27.636054	140	96	123	252	6.8	21	1	160	113	98	50	2.1	1.7	1.9
96	68	M	164	73	27.141582	130	70	104	233	6.2	37	0.9	190	150	108	50	2.8	1.6	1.1
97	38	F	150	64	28.444444	120	80	84	162	5.8	17	0.8	115	108	78	54	2	1.7	4
98	46	F	168	54	19.132653	130	80	428	705	10.4	28	1.3	246	187	174	35	2.3	1.7	4.9
99	57	M	168	64	22.675737	120	86	487	629	10.6	35	1.3	274	194	105	53	2.1	1.4	1.6
100	58	M	160	68	26.5625	130	80	94	160	5.8	46	1.2	131	65	69	30	1.7	1.2	4.5
101	59	M	162	65	24.767566	130	80	115	128	5.7	25	0.9	149	103	84	38	2.5	1.2	3.9
102	61	F	157	60	24.341758	120	80	84	153	5.7	17	0.7	219	124	132	58	2.3	1.4	1.1
103	56	F	160	65	25.390625	110	70	97	171	5.9	19	0.7	198	166	124	65	2.7	0.8	2.2
104	55	M	160	62	24.21875	110	70	176	200	8.2	35	0.9	221	189	136	36	2.5	1.4	1.3
105	48	F	158	63	25.23634	120	76	87	143	6	19	0.7	183	129	93	54	2.2	1.3	2.6
106	46	M	175	68	22.204082	140	80	229	355	8.2	16	0.9	177	174	100	36	2	1.4	2.6
107	56	F	157	68	27.587326	100	74	108	162	6	30	0.8	171	134	114	38	2.3	1.1	2.3
108	61	F	150	69	30.666667	120	70	125	214	6.2	25	0.9	194	150	108	54	2.7	1.3	2.4
109	42	M	174	76	25.102391	138	60	137	255	6.4	15	1	208	157	131	41	1.9	2	8

110	41	M	165	64	23.507805	130	90	117	232	6.4	24	1.1	172	176	114	41	3.1	1.7	0.7
111	33	F	155	58	24.141519	130	70	119	232	6.6	15	0.8	189	98	97	41	3.1	1.5	3.1
112	43	M	170	65	22.491349	140	80	112	192	6.8	21	0.9	101	130	129	40	2	1.2	2.1
113	45	M	174	70	23.120624	100	70	212	318	9.3	18	0.8	239	261	151	42	2	1.4	4.4
114	38	M	160	65	25.390625	140	90	263	382	10.4	17	0.9	205	138	132	45	2.3	1.8	3.1
115	46	M	168	62	21.96712	120	90	176	357	9.6	17	0.9	191	100	124	42	2.4	1.5	3.2
116	55	M	165	58	21.303949	130	86	172	222	6.7	22	1	279	297	168	43	2.3	1.9	10.4
117	39	M	165	74	27.1809	136	90	113	157	5.8	20	1	218	162	143	46	2.2	1.2	2.8
118	61	M	160	63	24.609375	130	78	122	168	5.2	19	0.9	119	244	72	40	2.4	1.5	8
119	49	M	168	62	21.96712	130	68	154	310	6.8	17	1	181	161	116	43	2.4	1.3	1.2
120	57	M	165	70	25.711662	130	86	168	363	8.1	22	0.8	167	164	113	38	2.1	1.7	2.6
121	44	F	145	60	28.537455	130	80	138	330	7.3	16	0.8	165	97	110	44	2.4	1.1	3.4
122	44	M	165	65	23.875115	130	80	101	141	5.4	20	1.1	160	118	100	50	1.3	0.9	4.6
123	66	F	147	57	26.377898	120	80	115	201	5.7	16	0.9	169	128	95	45	2.2	1.3	2.4
124	80	M	168	60	21.258503	146	94	100	143	5.4	25	1.1	166	74	97	51	1.9	1.4	3.7
125	65	M	170	64	22.145329	140	76	98	155	6	18	0.9	228	123	132	52	2.4	1.1	1.4
126	60	F	145	60	28.537455	130	90	151	187	6.5	22	0.9	183	128	165	43	2.1	1	6.9
127	39	M	175	64	20.897959	120	76	95	146	6	29	1.1	160	111	103	47	1.7	1.6	1.3
128	53	F	145	60	28.537455	140	94	92	151	6.2	24	0.8	195	131	132	57	2.5	1	2.8
129	55	M	168	74	26.218821	140	80	107	225	6.1	27	0.8	172	94	130	41	2.5	1.4	1
130	64	M	160	74	28.90625	130	90	173	283	7.2	23	0.9	126	95	76	52	2.3	1.6	2.7
131	46	M	164	60	22.30815	140	84	130	192	5.8	27	0.9	169	108	109	46	2.8	1.8	2.8
132	52	F	147	63	29.154519	140	80	213	340	7.4	17	0.8	182	244	104	40	2.5	1.8	1.7
133	52	M	164	57	21.192742	136	80	114	176	6.1	19	0.9	177	178	98	54	2.6	1.5	2.2
134	39	F	153	62	26.48554	120	70	114	134	6.2	16	0.8	169	164	96	58	2.2	1.3	2.2
135	67	F	155	50	20.811655	140	80	200	310	7.9	18	0.7	135	209	76	58	2.3	1.6	2.5
136	36	M	170	68	23.529412	110	70	253	329	8.5	23	0.8	194	184	129	47	2.6	1.3	2.6
137	59	M	160	53	20.703125	130	80	110	186	5.5	16	0.9	142	110	91	39	2.5	1.4	17.1
138	48	F	150	60	26.666667	120	80	297	438	7.8	17	0.7	177	97	97	67	2.4	1.4	2.8
139	44	M	165	68	24.977043	130	84	109	161	5.8	16	0.9	155	87	109	49	2.6	0.9	3.5
140	43	M	168	72	25.510204	130	76	281	522	9.2	28	1.1	169	126	119	39	2.4	1.5	3.5
141	48	M	158	60	24.03461	140	80	100	110	5.5	22	0.8	204	98	142	48	2.4	1.3	6.1
142	43	M	171	68	23.255019	130	90	90	154	5.9	17	1.1	177	135	122	43	2.9	1.1	2.7
143	40	M	165	68	24.977043	110	80	146	197	6.1	17	1	169	224	103	46	2.2	1	1.7
144	49	M	174	63	20.808561	130	70	191	248	7.1	16	1.1	184	76	132	42	2.5	1.5	1.6
145	61	M	160	59	23.046875	160	94	127	136	5.5	33	0.8	163	98	115	31	2.7	1.7	7.7
146	52	M	166	74	26.854406	140	90	262	370	7.8	28	1.2	136	105	82	46	2.8	1.2	2
147	47	F	148	64	29.218408	120	84	124	161	7.1	20	0.9	196	154	97	40	2.5	1.5	1.7
148	60	F	154	68	28.672626	130	94	92	133	5.9	21	1	210	170	105	39	2.5	1.4	5.1

149	40	F	150	70	31.111111	140	80	168	247	8.7	24	0.9	196	172	86	40	2.5	0.7	4.1
150	62	M	170	60	20.761246	130	94	208	260	6.8	15	1	176	180	90	36	2.6	1.4	2.1
151	39	F	160	74	28.90625	140	86	120	136	5.7	15	1	211	78	110	67	2	1.6	6.1
152	45	F	144	64	30.864198	120	80	102	146	5.3	29	1	226	124	127	57	2	1.3	2
153	42	M	168	70	24.801587	140	90	202	223	10.9	16	0.9	89	82	48	47	2.9	1.7	3.1
154	60	F	163	65	24.464602	130	76	410	572	9.8	25	1	249	152	160	41	2.3	1.1	6.4
155	40	F	155	63	26.222685	130	74	100	192	5.7	15	0.8	254	174	151	51	1	0.9	1.9
156	48	F	145	63	29.964328	110	70	124	153	5.9	15	0.8	157	110	80	43	2.6	1.3	2.5
157	34	F	160	49	19.140625	120	70	110	147	6.3	20	0.8	167	245	74	40	1.7	0.5	6.8
158	50	F	160	73	28.515625	140	80	130	182	7	26	0.8	244	120	99	61	2.6	1.3	3.7
159	56	F	156	60	24.654832	130	80	137	161	8.1	20	0.8	240	139	98	67	2.2	1	1.9
160	35	F	158	64	25.636917	110	70	169	204	7.4	24	0.8	210	164	98	42	4.2	1.5	5.1
161	32	F	152	65	28.133657	120	80	110	142	6.3	22	0.8	173	154	76	42	2.2	1.2	6
162	44	F	154	60	25.299376	130	80	148	196	5.9	25	0.8	220	111	192	52	2.7	1.3	5.4
163	45	F	154	56	23.612751	120	80	218	294	9.3	16	0.8	139	89	66	67	2.4	1.3	3.4
164	43	F	160	56	21.875	130	90	95	160	5.2	15	0.8	186	114	94	43	3	1.3	1.4
165	58	M	162	64	24.386526	120	80	98	162	6.4	16	1	132	279	70	29	2.9	1.3	9.4
166	47	F	154	70	29.515939	140	90	82	223	6.7	23	0.8	159	62	74	50	2.3	1.6	2.6
167	46	M	175	72	23.510204	130	90	120	210	7	27	1	240	124	146	44	2.8	1.7	1.6
168	55	F	154	60	25.299376	130	80	160	242	7.2	21	0.7	213	126	127	59	2.3	1	2.7
169	66	M	163	62	23.335466	120	80	114	171	6.2	35	1.2	198	208	127	51	2.7	1.9	1.6
170	55	M	158	60	24.03461	110	80	160	202	6.8	43	1.5	200	148	125	50	1.8	1.6	1.6

A Thyroid Profile status in type 2 Diabetes Mellitus-Control Group

S.NO	AGE	SEX	HEIGHT cm	WEIGHT kg	BMI	Syst.BP mm Hg	Dias.BP mm Hg	FBS mg/dl	PPBS mg/dl	HbA1C %	UREA mg/dl	CREATININE mg	T.CHO mg/dl	TRIGLY mg/dl	LDL mg/dl	HDL mg/dl	T3 pg/ml	T4 ng/dl	TSH m IU/ml
1	46	F	154	46	19.396188	110	70	92	123	5.3	18	0.7	210	123	120	41	2.1	1.5	2.9
2	40	M	160	64	25	120	86	98	103	5.2	21	0.8	114	142	80	43	3.1	1.5	2.8
3	40	F	158	54	21.631149	110	70	87	112	4.8	15	0.9	152	138	75	43	2.8	1.2	3.5
4	66	M	162	68	25.910684	140	80	92	107	5	30	1.4	161	87	103	29	2.5	1.4	1.3
5	47	F	156	58	23.833005	110	70	81	98	4.8	15	0.8	182	84	100	38	1.6	1.3	4.7
6	35	M	164	60	22.30815	110	80	70	94	5.4	21	0.8	240	109	130	57	3	1.3	1.5
7	49	M	168	63	22.321429	140	90	83	101	5	27	0.9	206	190	120	37	3.4	0.8	7.7
8	45	M	158	58	23.233456	130	80	84	107	5.1	21	1	203	148	122	40	2.7	1.1	3.4
9	46	M	157	64	25.964542	110	70	85	129	4.9	18	1	170	79	88	43	2.4	1.2	1
10	35	F	152	66	28.566482	110	80	91	124	4.9	15	1	195	88	106	43	3	1.6	2.3
11	38	F	160	72	28.125	110	80	73	106	5.1	15	0.7	181	117	91	49	2.6	1.6	0.9
12	46	M	168	70	24.801587	130	80	71	94	4.5	15	1.2	203	257	113	35	1.7	1.2	2.3
13	53	F	164	59	21.936347	120	80	93	104	5.7	19	0.6	188	114	119	33	2.7	0.9	1.7
14	75	M	160	70	27.34375	140	90	95	105	5	25	1	156	59	99	37	1.8	1.6	3.5
15	41	M	148	60	27.392257	110	70	102	113	5.5	23	1	214	77	126	60	2.2	1.9	1.4
16	54	M	156	68	27.942143	110	70	88	99	4.8	16	0.6	170	84	99	116	2	1.4	6.4
17	39	M	162	58	22.10029	120	80	97	109	5.3	21	1	175	129	95	48	2.9	1.6	1.6
18	46	M	154	60	25.299376	120	70	99	106	5.2	24	0.8	181	64	105	46	2.7	1.5	1
19	39	M	152	68	29.432133	120	70	94	103	5	16	1.1	188	248	105	40	1.6	1.9	1.9
20	45	F	152	58	25.103878	110	70	90	104	5.2	16	0.7	168	117	98	41	2.3	1.2	2
21	48	M	172	64	21.633315	110	70	94	107	5.4	17	0.8	209	116	128	49	2.2	1.7	1.9
22	45	M	162	62	23.624447	120	70	91	100	5	20	1	181	116	119	42	2.6	1.2	2.4
23	48	F	151	63	27.630367	130	80	96	102	4.8	22	0.8	161	68	109	41	2	1.2	4.4
24	48	M	176	59	19.047004	130	90	90	134	5.2	19	0.9	216	141	153	43	2.1	1.3	4.9
25	36	F	150	57	25.333333	110	70	98	114	5	16	0.7	214	205	125	40	2.2	1.5	1.9

26	55	M	168	72	25.510204	140	80	100	111	5.3	53	1.4	156	75	98	44	2.8	1	2.5
27	64	M	165	63	23.140496	130	90	100	131	5.1	33	1.1	155	82	90	46	2.4	1.9	1.4
28	44	M	160	58	22.65625	110	70	101	141	5.4	20	1.1	160	118	100	50	1.3	0.9	4.6
29	63	M	164	68	25.28257	140	80	99	126	4.9	28	1	156	182	103	35	2.4	1.3	0.7
30	61	F	148	60	27.392257	130	90	104	125	5.4	18	0.8	142	106	86	47	2.1	1.2	2.2
31	49	M	170	68	23.529412	110	80	97	131	4.9	19	1.1	185	111	111	61	2.5	1.2	3.8
32	52	M	172	70	23.661439	120	80	88	108	5.4	31	1.2	135	68	90	34	3.2	1.6	3.7
33	45	F	162	58	22.10029	100	70	90	104	5.2	16	0.7	168	117	98	41	2.3	1.2	2
34	49	M	168	65	23.030045	110	90	94	103	5	16	1.1	188	248	108	40	1.6	1.9	1.9
35	47	F	150	50	22.222222	130	70	89	137	5.2	24	0.8	189	104	97	70	1.6	1.4	15
36	52	M	168	64	22.675737	130	76	82	112	4.9	19	0.9	201	136	119	42	2.7	1.3	1.5
37	49	M	174	68	22.460034	110	80	97	109	5.3	21	1	175	127	95	48	2.9	1.6	1.6
38	45	M	164	70	26.026175	120	86	90	99	4.7	17	0.9	219	129	147	48	2.3	1	1.3
39	80	M	170	62	21.453287	130	80	100	133	5.4	25	1.1	166	74	97	51	1.9	1.4	3.7
40	48	M	162	59	22.481329	120	76	85	103	4.8	21	1.1	218	81	155	37	2.3	1.2	3.2
41	48	M	166	69	25.039919	130	84	91	99	4.4	15	0.9	151	66	94	42	2.6	1.3	0.8
42	53	F	150	62	27.555556	120	80	101	134	5.1	15	0.7	242	83	144	59	3.1	1.3	2.5
43	52	M	172	68	22.985398	140	84	85	100	4.5	19	1.1	254	84	177	50	2.6	1.4	1.7
44	61	M	154	60	25.299376	130	80	104	112	5	24	1.1	192	91	119	38	2.4	0.8	3.2
45	54	M	170	66	22.83737	120	84	100	110	5.1	15	0.9	145	135	96	34	2.3	1.1	2
46	45	M	164	58	21.564545	130	76	96	101	4.9	28	0.9	142	59	92	38	2.8	1	2
47	58	F	152	60	25.969529	110	80	105	120	5.1	15	0.7	185	93	102	47	3.4	1.5	3
48	51	M	164	62	23.051755	130	76	84	122	4.7	21	1	161	67	80	67	1.8	1	1.4
49	49	F	154	62	26.142688	110	80	82	96	4.3	30	0.8	221	248	151	33	2.1	0.6	6.6
50	50	M	168	60	21.258503	130	84	96	110	5.4	19	1.1	145	79	102	37	2.6	1.5	0.9

சென்னை மருத்துவக்கல்லூரி மருத்துவமனை மற்றும் ஆராய்ச்சி மையம், இருங்களூர், திருச்சிராப்பள்ளி – 621 105

ஒப்புதல் படிவம்

சென்னை மருத்துவக்கல்லூரி மருத்துவமனை மற்றும் ஆராய்ச்சி மையத்தின் உயிர் வேதியியல் துறையில் நடத்தப்படும் ''நீரிழிவு நோயாளிகளில் தைராய்டு ஹார்மோன் பரிசோதனை'' – ல் பங்கேற்குமாறு உங்களை கேட்டுக் கொள்கிறோம்.

- இப்பரிசோதனைக்கு சம்மதிப்பது உங்கள் விருப்பத்தைப் பொறுத்தது.
- இச்சோதனைக்கு கட்டணம் கிடையாது.
- கட்டாயம் ஏதும் இல்லை.
- பரிசோதனையிலிருந்து எந்நேரமும் விலக தங்களுக்கு முழு உரிமை உண்டு.

இந்த ஆய்வின் முடிவுகள் மருத்துவம் மற்றும் விஞ்ஞான முன்னேற்றத்திற்கு உதவும் என்று கருதுகின்றோம். இவைகளை வேறு எதற்கும் பயன்படுத்தப்பட மாட்டாது என உறுதியளிக்கிறோம்.

ஒப்புதல்

நான் திரு/திருமதி/செல்வி/ _____

முகவரி_____

_____நாள்_____

அன்று மேற்கண்ட ஆய்வுக்காக தகவல் படிவத்தினை படித்து, கேட்டு புரிந்து கொண்டு இந்த ஆராய்ச்சிக்கு தேவையான சோதனைக்கு என்னிடம் இருந்து 4மிலி. இரத்தம் எடுத்துக் கொள்ள அனுமதிக்கிறேன். என் மனப்பூர்வமான சம்மதத்தை அளிப்பதோடு இந்த ஆய்வின் முடிவுகளை மருத்துவம் மற்றும் விஞ்ஞான நோக்கத்திற்கு பயன்படுத்த ஒப்புதல் அளிக்கிறேன்.

நடுநிலை சாட்சியின் கையொப்பம்

பங்கேற்பாளர் கையொப்பம்

ஆய்வாளர் / சம்மதம் பெறுபவர் கையொப்பம்

Chennai Medical College Hospital & Research Centre
Irungalur, Trichy – 621 105.

Consent Form

You are requested to participate in a study conducted in the Department of Biochemistry, Chennai Medical College Hospital & Research Centre, Irungalur, Trichy, Tamilnadu titled "A study on Thyroid profile status in Type 2 Diabetes mellitus". Your participation in the study is voluntary

- There will be no cost for participating in the study
- Your participation is not a compulsion
- You have the right to withdraw from the study at any time.

Nature of Study:

- ✓ If any abnormalities are identified, you will be informed for further consultation.
- ✓ The results of this study will be kept confidential

We believe that the results of this study will be beneficial for advancements in medicine & Science. We assure you that we will not use these result for any other purpose.

Consent

I Mr /Mrs / Ms_____ residing at _____ _____on this day _____ after having read the consent form carrying information for the above mentioned study and I hereby give my consent to take 4ml of my blood sample for the purpose of doing serum electrolytes. I was explained about the procedure in detail and give my consent for participating in the study and for using the results for Medical & Scientific purposes.

Signature of the participant Signature of Witness

Signature of the Investigator