A CLINICAL, BIOCHEMICAL PROFILE OF TYPE 2 DIABETES IN WOMEN WITH SPECIAL REFERENCE TO VITAMIN D STATUS IN OBESE AND NON-OBESE PERSON

DISSERTATION SUBMITTED TO

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THANJAVUR- 613004.

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY CHENNAI, TAMILNADU, INDIA

CERTIFICATE FROM THE DEAN

THIS IS TO CERTIFY THAT THIS DISSERTATION ENTITLED **"A CLINICAL, BIOCHEMICAL PROFILE OF TYPE 2 DIABETES IN WOMEN WITH SPECIAL REFERENCE TO VITAMIN D STATUS IN OBESE AND NON-OBESE PERSON"** IS THE BONAFIDE WORK DONE BY **DR.PRAVEEN KUMAR V C**, IN PARTIAL FULFILMENT OF THE UNIVERSITY REGULATIONS OF THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI, FOR **M.D GENERAL MEDICINE** EXAMINATION OF THE TAMILNADU DR M.G.R MEDICAL UNIVERSITY TO BE HELD IN **MAY 2023.**

PROF.DR.R. BALAJINATHAN, M.D.

THE DEAN,

THANJAVUR MEDICAL COLLEGE AND HOSPITAL,

THANJAVUR.

DEAN
Thanjavur Medical College
Thanjavur

CERTIFICATE FROM THE HOD

THIS IS TO CERTIFY THAT THIS DISSERTATION ENTITLED **"A CLINICAL, BIOCHEMICAL PROFILE OF TYPE 2 DIABETES IN WOMEN WITH SPECIAL REFERENCE TO VITAMIN D STATUS IN OBESE AND NON-OBESE PERSON"** IS THE BONAFIDE WORK DONE BY **DR. PRAVEEN KUMAR V C**, IN PARTIAL FULFILMENT OF THE UNIVERSITY REGULATIONS OF THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI, FOR **M.D GENERAL MEDICINE EXAMINATION OF THE TAMILNADU DR M.G.R MEDICAL UNIVERSITY** TO BE HELD IN **MAY 2023.**

14/12/22

PROF.DR. C. PARANTHAKAN, M.D.

Dr.C.PARANTHAKAN, M.D.,
Reg. No: 43149
HOD & Professor
Department of Medicine
Thanjavur Medical College & Hospital
Thanjavur - 613 004.

THE HOD,

DEPARTMENT OF GENERAL MEDICINE,

THANJAVUR MEDICAL COLLEGE AND HOSPITAL,

THANJAVUR.

CERTIFICATE FROM THE GUIDE

THIS IS TO CERTIFY THAT THIS DISSERTATION ENTITLED "**A CLINICAL, BIOCHEMICAL PROFILE OF TYPE 2 DIABETES IN WOMEN WITH SPECIAL REFERENCE TO VITAMIN D STATUS IN OBESE AND NON-OBESE PERSON**" IS THE BONAFIDE WORK DONE BY **DR.PRAVEEN KUMAR V C**, IN PARTIAL FULFILMENT OF THE UNIVERSITY REGULATIONS OF THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY, CHENNAI, FOR **M.D GENERAL MEDICINE** EXAMINATION TO BE HELD IN **MAY 2023.**

PROF.DR.V.P. KANNAN, M.D.

Dr. V.P. KANNAN, M.D.
Professor
Regd. No : 58217,
TMCH, Thanjavur

UNIT CHIEF,

DEPARTMENT OF GENERAL MEDICINE,

THANJAVUR MEDICAL COLLEGE AND HOSPITAL,

THANJAVUR.

DECLARATION

I, **DR. PRAVEEN KUMAR V C**, SOLEMNLY DECLARE THAT THIS DISSERTATION TITLED "**A CLINICAL, BIOCHEMICAL PROFILE OF TYPE 2 DIABETES IN WOMEN WITH SPECIAL REFERENCE TO VITAMIN D STATUS IN OBESE AND NON-OBESE PERSON**" IS A BONAFIDE RECORD OF WORK DONE BY ME AT THE DEPARTMENT OF GENERAL MEDICINE, THANJAVUR MEDICAL COLLEGE AND HOSPITAL, UNDER THE GUIDANCE OF **PROF.DR.V.P. KANNAN, MD**, UNIT CHIEF, DEPARTMENT OF GENERAL MEDICINE, THANJAVUR MEDICAL COLLEGE AND HOSPITAL, THANJAVUR.

THIS DISSERTATION IS SUBMITTED TO THE TAMIL NADU DR. M.G.R MEDICAL UNIVERSITY, CHENNAI IN PARTIAL FULFILLMENT OF THE RULES AND REGULATIONS FOR THE AWARD OF **M.D DEGREE GENERAL MEDICINE** EXAMINATION TO BE HELD IN **MAY 2023**.

PLACE: THANJAVUR

DATE: 16.12.2022

V. C. Praveen Kumar

DR PRAVEEN KUMAR V C

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Submitter email	valasingam.vcpk@gmail.com
Similarity	17%
Analysis address	valasingam.vcpk.tnmg@analysis.urkund.com

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Dr. V.P. KANNAN, M.D.
Professor
Regd. No : 58217.
TMCH, Thanjavur

LIST OF ABBREVATIONS:

DM- Diabetes Mellitus

T2DM-Type II Diabetes Mellitus

HLA-Human Leucocyte Antigen

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1 ABSTRACT:

Introduction:

Diabetes mellitus type II (DM II) is a condition that affects multiple body systems and has a rapidly rising prevalence. Therefore, it is essential to adequately manage DM II in order to prevent complications such diabetic nephropathy, peripheral neuropathy, and retinal. Among its many advantages, vitamin D helps regulate haemoglobin A1c (HbA1c). It improves insulin sensitivity and secretion.

Objectives:

To study the clinical, biochemical profile of type2 diabetic women

2. To assess the vitamin D status in type2 diabetic obese and non-obese women

3. To determine the influence of glycaemic status of a patient in vitamin D level

4. To determine the correlation of duration of diabetes on vitamin D level

5.To establish the relationship between anthropometric measurement with vitamin D levels

Methodology:

100 patients were selected according to inclusion and exclusion criteria and written informed consent was taken for their participation. Patient history in relation to the

duration of type2 diabetes was also taken. Patients were evaluated for anthropometric measures like height, weight, waist/hip ratio, glycaemic value, renal function test, urine routine and vitamin D levels. Vitamin D levels were correlated with the anthropometric measure and glycaemic control. All data were entered I to Microsoft excel analysed using SPSS version 16.

Results:

The mean Age (years) among the subjects was 49.94 (± 6.69) years ranging from 38 to 64 years. The mean Weight (kg) among the subjects was 72.43 (± 8.06) kg , mean height in cm was 160.04 (± 4.23), mean BMI was 28.4 (± 3.79) kg/m2 Among the subjects, 90 (90%) had > 1 and 10 (10%) had < 1 Waist Hip ratio

The mean Duration of Diabetes (years) among Obese was 10.7 (± 3.99) which is higher by 2.66 and statistically significant compared to 8.04 (± 3.85) in Non-Obese. The mean Fasting Blood Sugar (mg/dl) among Obese was 248.42 (± 29.83) which is higher by 36.68 and statistically significant compared to 211.74 (± 19.77) in Non-Obese.The mean Post prandial blood sugar (mg/dl) among Obese was 296.42 (± 41.52) which is higher by 48.28 and statistically significant compared to 248.14 (± 21.36) in Non-Obese. The mean Total Cholesterol (mg/dl) among Obese was 233.52 (± 16) which is higher by 14.86 and statistically significant compared to 218.66 (± 12.84) in Non-Obese.The mean Blood Urea (mg/dl) among Obese was 43 (± 5.86) which is higher by 3.7 and statistically significant compared to 39.3 (± 6.73) in Non-Obese.

The mean Serum Vitamin D (ng/ml) among Obese was 24.21 (± 3.83) which is lower by 7.17 and statistically significant compared to 31.38 (± 2.53) in Non-Obese. Comparing the Vitamin D level with Obesity distribution, Obese group had higher proportion of

Inadequate Vitamin D level with 78% followed by Deficient Vitamin D level with 22% and least in Adequate Vitamin D level with 0% compared to Non-Obese group which had higher proportion of Adequate Vitamin D level with 72% followed by Inadequate Vitamin D level with 28% and least in Deficient Vitamin D level with 0%. The difference in Vitamin D level distribution between Obese and Non-Obese was statistically significant ($p < 0.05$).

The mean Duration of Diabetes (years) among Deficient Vitamin D level was 13.64 which is higher than mean among Inadequate Vitamin D level which was 10.45 followed by Adequate Vitamin D level with a mean of 6.47 and the difference was statistically significant ($p < 0.05$). The mean Fasting Blood Sugar (mg/dl) among Deficient Vitamin D level was 287.64 which is higher than mean among Inadequate Vitamin D level which was 232.36 followed by Adequate Vitamin D level with a mean of 209.14 and the difference was statistically significant ($p < 0.05$). The mean Post prandial blood sugar (mg/dl) among Deficient Vitamin D level was 344.55 which is higher than mean among Inadequate Vitamin D level which was 276.25 followed by Adequate Vitamin D level with a mean of 244.36 and the difference was statistically significant ($p < 0.05$). The mean Total Cholesterol (mg/dl) among Deficient Vitamin D level was 250.55 which is higher than mean among Inadequate Vitamin D level which was 227.75 followed by Adequate Vitamin D level with a mean of 216.17 and the difference was statistically significant ($p < 0.05$).The mean Blood Urea (mg/dl) among Deficient Vitamin D level was 46.27 which is higher than mean among Inadequate Vitamin D level which was 42.32 followed by Adequate Vitamin D level with a mean of 37.86 and the difference was statistically significant ($p < 0.05$).

The model with these predictors explains 78.53% variability (predictability) of Serum Vitamin D (ng/ml). Serum Vitamin D (ng/ml) decreases -0.03 times for each unit increase in Post prandial blood sugar (mg/dl) and is statistically significant. Serum Vitamin D (ng/ml) decreases -0.04 times for each unit increase in Total Cholesterol (mg/dl) and is statistically significant. Serum Vitamin D (ng/ml) decreases -0.09 times for each unit increase in Blood Urea (mg/dl) and is statistically significant. Serum Vitamin D (ng/ml) decreases -0.3 times for each unit increase in Duration of Diabetes (years) and is statistically significant. Serum Vitamin D (ng/ml) decreases -0.46 times for each unit increase in BMI (kg/m2) and is statistically significant.

Conclusion:

There is a high prevalence of hypo-vitaminosis D among patients with type-2 diabetes, particularly among patients with poor glycaemic control and in those with longer diabetes durations. Therefore, every diabetes patient needs lifestyle changes, prompt glycaemic management, and early vitamin D treatment.

Keywords:

Type II Diabetes Mellitus, Vitamin D, Obese

2 INTRODUCTION

Diabetes mellitus (DM) is a chronic-metabolic-disorder characterized by constant and persistent hyper-glycaemia. Impaired insulin secretion, resistance to peripheral actions of insulin, or both can be a reason for this.[1] The global prevalence of diabetes among adults in 2011 as 8.3% which is projected to increase to 9.9% by 2030 as reported by International Diabetes Federation.[2] The prevalence rate of diabetes mellitus among the adult population ranges from 3% in rural areas to 9% in urban areas in India.[3]

A metabolic condition with numerous aetiologies, diabetes mellitus (DM) is defined by persistent hyperglycaemia and disturbances in the metabolism of carbohydrates, proteins, and fats as a result of decreased insulin production, insulin action, or both. The long-term damage, malfunction, and destruction of different organs are all effects of diabetes mellitus.. [4] The development of incapacitating and life-threatening health complications, most prominent of which are microvascular (retinopathy, nephropathy, and neuropathy), and macrovascular complications leading to a 2- to 4-fold increased risk of cardiovascular diseases, can result from chronic hyperglycaemia in patients with diabetes mellitus working in synergy with the other metabolic aberrations.[5]

A complex interaction between genes and environment results in type 2 diabetes, a metabolic illness. Defects in insulin secretion and action, which cause hyperglycaemia, are its defining characteristics. A fasting hyperglycaemia (126 mg/dl), glucose levels greater than or equal to 200 mg/dl at 120 minutes following an oral glucose tolerance challenge, or two or more instances of higher-than-200 mg/dl results on a random glucose test are used to identify diabetes. Diabetes is frequently preceded by impaired fasting glucose (IFG), which is identified by fasting blood glucose levels between 100 and 125 mg/dl.[6]

The pathogenic mechanisms range from abnormalities that lead to resistance to insulin action to autoimmune destruction of the pancreatic beta-cells with subsequent insulin shortage. The anomalies in protein, lipid, and carbohydrate metabolism are caused by insulin's ineffective action on its target tissues. Reduced insulin action is caused by inadequate insulin secretion and/or impaired tissue insulin responses at one or more locations along the intricate hormonal action pathways.[5,7]

Polyuria, polydipsia, weight loss, occasionally coupled with polyphagia, and blurred vision are signs of severe hyperglycaemia. There may also be growth impairment and an increased risk of contracting certain infections in addition to chronic hyperglycaemia. The nonketotic hyperosmolar syndrome or hyperglycaemia with ketoacidosis are immediate, potentially fatal effects of untreated diabetes. Diabetes can cause long-term complications such as retinopathy, which can cause blindness, nephropathy, which can cause renal failure, peripheral neuropathy, which increases the risk of foot ulcers, amputations, and Charcot joints, and autonomic neuropathy, which causes symptoms of the gastrointestinal, genitourinary, cardiovascular, and sexual systems as well as infertility. Diabetes patients are more likely to get peripheral artery, cerebral, and cardiovascular atherosclerosis. Diabetes patients often have hypertension and problems in lipoprotein metabolism.[5,8,9]

Need for the study / Justification of the study:

Vitamin D have found out to play an important role in progression of diabetes. Its role in diabetes is attributed to improved beta cell function by enhanced insulin release, increases insulin sensitivity by stimulating the expression of insulin receptors and inhibition of beta cell apoptosis by inhibition of cytotoxic gene expression. In this study vitamin D levels in obese and non-obese type 2 diabetic women is evaluated and its association with anthropometry, glycaemic control, duration of diabetes was assessed.

3 AIM AND OBJECTIVES

3.1 **AIM:**

To find out the correlation of Vitamin D levels and diabetic profile among patients admitted in inpatient wards , Department of General Medicine, Thanjavur Medical College.

3.2 **OBJECTIVES:**

1. To study the clinical, biochemical profile of type2 diabetic women

2. To assess the vitamin D status in type2 diabetic obese and non-obese women

3. To determine the influence of glycaemic status of a patient in vitamin D level

4. To determine the correlation of duration of diabetes on vitamin D level

5.To establish the relationship between anthropometric measurement with vitamin D levels

4 REVIEW OF LITERATURE

Review of Literature of this study is discussed under the following heads:

1. Diabetes Mellitus

a. Epidemiology

b. Classification and Risk factors

c. Aetiopathogenesis

d. Complications

2. Role of Vitamin D in Diabetes Mellitus

3. Reviews of articles depicting the association between Vitamin D levels and Diabetes Mellitus

i. DIABETES MELLITUS

The term Diabetes mellitus was derived from the Latin term mellitus, which means sweet, and the Greek word diabetes, which means to syphon or pass through. According to a historical research, Apollonius of Memphis coined the name "diabetes" sometime between 250 and 300 BC. The sweet character of the urine in this condition was discovered by ancient Greek, Indian, and Egyptian civilizations, which led to the spread of the term "Diabetes Mellitus." In 1889, Mering and Minkowski made the discovery that the pancreas plays a role in the pathophysiology of diabetes. At the University of Toronto, Banting, Best, and Collip removed the hormone insulin from the pancreas of cows in 1922, making a cure for diabetes available to the public. Over the years, incomparable work has taken place, and multiple discoveries, as well as management strategies, have been created to tackle this growing problem. Diabetes is one of the most common chronic diseases in worldwide. [5,8,10]

Epidemiology of Diabetes Mellitus

8.8% of the population has diabetes, with men having slightly higher rates (9.6%) than women (9.0%), according to the International Diabetes Federation (IDF). According to the most recent data available, there are 463 million and 374 million people worldwide who suffer from diabetes and impaired glucose tolerance (IGT), a prediabetic disease. By 2045, it is predicted that there would be 548 million people with IGT and 700 million people with diabetes, a 51% rise from 2019.[11,12]

Table 1: Burden of diabetes/prediabetes in India

	YEAR	
	2019	**2045**
Impaired glucose tolerance (estimates) [20-79 years]		
Number of people (million)	25.2	35.7
Rank	4	3
Diabetes (estimates) [20-79 years]		
Prevalence (%)	8.9	-
Age adjusted prevalence (%)	10.4	-
Number of people (million)	77.0	134.2
Rank	2	2
Diabetes (estimates) [>65 years]		
Number of people (million)	12.1	27.5
Rank	3	2
Undiagnosed diabetes (estimates)		
Prevalence (%)	57.0	-
Number of people (million)	43.9	-
Rank	2	
Healthcare expenditure on diabetes		
Mean expenditure per person with diabetes (USD)	92.0	-
Deaths related to diabetes		
Total deaths (million)	1.0	-

The data from 15 states/UT of the country were used in the Indian Council of Medical Research-India DIABetes study, the largest nationally representative epidemiological survey on diabetes and prediabetes ever conducted in India. The prevalence of diabetes varied from 4.3% in Bihar to 13.6% in Chandigarh, and from 3.5 to 8.7% in rural areas. Compared to rural areas (5.2%), urban areas had a greater prevalence of diabetes (11.2%). In rural and urban areas, respectively, the prevalence of prediabetes ranged from 5.8 to 14.7% and 7.2 to 16.2%.[13,14]

A series of metabolic illnesses characterised by chronic hyperglycaemia brought on by deficiencies in insulin secretion, insulin action, or both is called Diabetes Mellitus (DM). These metabolic abnormalities are caused by reduced insulin levels to achieve a proper response and/or insulin resistance of target tissues, primarily skeletal muscles, adipose tissue, and to a lesser extent, liver, at the level of insulin receptors, signal transduction system, and/or effector enzymes or genes. The type and duration of diabetes are to blame for the severity of symptoms and development of complications. Patients with diabetes can experience polyuria, polydipsia, polyphagia, weight loss, and blurred vision. Some patients with diabetes are asymptomatic, particularly those with type 2 diabetes in the early stages of the disease. However, patients with severe hyperglycaemia and, particularly in children, those with absolute insulin deficiency, can experience these symptoms. If untreated, uncontrolled diabetes can cause coma, stupor, and, in rare cases, death from nonketotic hyperosmolar syndrome or ketoacidosis.[15]

Diabetes Mellitus (DM) is a metabolic disease, involving inappropriately elevated blood glucose levels.

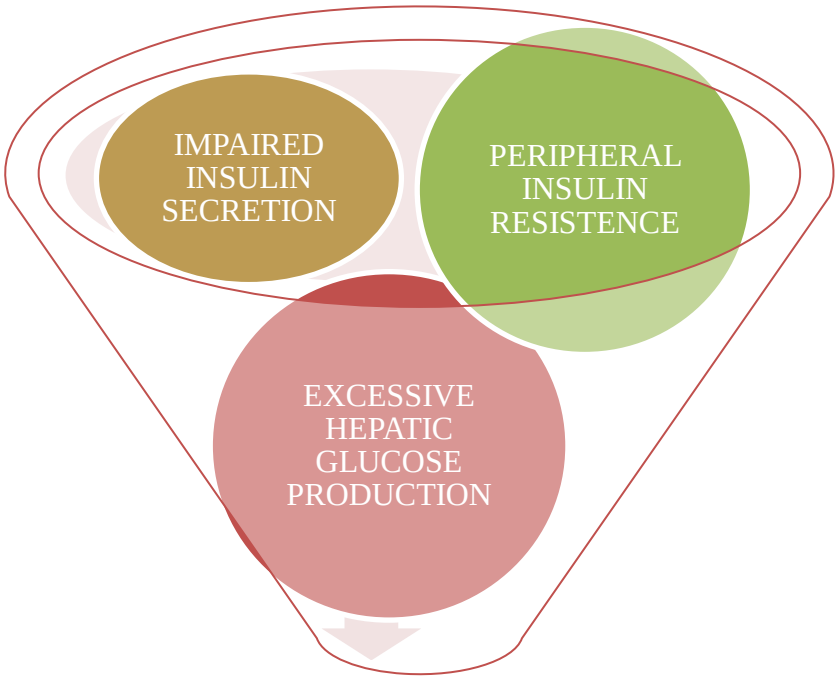
The classification of diabetes includes four clinical classes:[6]

- Type 1 diabetes (outcome of β-cell destruction, usually leading to absolute insulin deficiency)
- Type 2 diabetes (outcome of from a progressive insulin secretory defect on the background of insulin resistance)
- Other specific types of diabetes due to other causes, e.g., genetic defects in β-cell function, genetic defects in insulin action, diseases of the exocrine pancreas (such as cystic fibrosis), and drug- or chemical-induced (such as in the treatment of HIV/AIDS or after organ transplantation)
- Gestational diabetes mellitus (GDM) (diabetes diagnosed during pregnancy that is not clearly overt diabetes)

Type 1 Diabetes Mellitus (T1DM) and Type 2 Diabetes Mellitus (T2DM) are the two primary subtypes of DM, and both are typically brought on by faulty insulin secretion (T1DM) and/or action (T2DM). T2DM is expected to affect middle-aged and older individuals who have chronic hyperglycaemia as a result of poor lifestyle and nutritional choices, whereas T1DM is thought to manifest in children or teenagers. Because the pathogenesis of T1DM and T2DM is so dissimilar, each type has a unique aetiology, presentation, and course of treatment.

a. AETIOPATHOGENESIS

Type 2 diabetes mellitus is characterized by three pathophysiologic abnormalities



TYPE II DIABETES MELLITUS

Due to the compensatory increase in insulin secretion by pancreatic beta cells during the early stages of the illness, glucose tolerance is maintained despite insulin resistance. As compensatory hyperinsulinemia and insulin resistance develop, the pancreatic islets lose their ability to maintain the hyper-insulinemic condition. Impaired glucose tolerance leads to increases in post-meal glucose. As insulin secretion declines and hepatic glucose synthesis rises, overt diabetes with fasting hyperglycemia results. Beta cell failure may ensue in the last stage.

There are two primary subclasses of endocrine cells in the pancreatic islets of Langerhans: beta cells that produce insulin and alpha cells that secrete glucagon. Based on the glucose environment, beta and alpha cells continuously adjust the amount of hormones they excrete. T he glucose levels go off-balance without the balance between insulin and glucagon. Insulin in people with DM is either inconsistent or has impaired activity (insulin resistance), which causes hyperglycaemia.

Beta cell apoptosis in the pancreas, which is typically a result of autoimmune destruction of beta cells, is a hallmark of T1DM. As a result, beta cells are completely destroyed, and as a result, insulin is either completely missing or very low.

T2DM has a more gradual start and is characterised by an imbalance between insulin sensitivity and levels of insulin that results in an insulin functional deficit. Insulin resistance has several causes, but it often arises as people get older and more obese.

A significant risk factor for both types is the genetic background. As the human genome is studied more thoroughly, several loci that increase the risk of DM are discovered. Major Histo-Compatibility Complex (MHC) and Human Leukocyte Antigen polymorphisms have been known to affect the risk for T1DM (HLA).[16]

Genetics and lifestyle play a more intricate role in T2DM. There is strong evidence suggesting that T2DM has a more robust genetic profile than T1DM. Most illness sufferers have at least one parent who also has type 2 diabetes.[17] Beta-cell dysfunction is a component of T2DM, an insulin-resistance syndrome. Initial compensation involves a rise in insulin release, which keeps blood sugar levels within the usual range. As the illness worsens, beta cells alter, making it impossible for the insulin secretion to keep glucose homeostasis, leading to hyperglycaemia. The majority of T2DM patients are obese or have a greater body fat percentage, which is primarily distributed in the abdominal area. This adipose tissue promotes insulin resistance by upregulating adipokines and increasing FFA release, among other inflammatory processes. The chance of developing T2DM is further increased by a lack of physical exercise, history of GDM, in people with hypertension, or dyslipidaemia. Adipokine dysregulation, inflammation, abnormal incretin biology, decreased incretins like glucagon-like peptide-1 (GLP-I) or incretin resistance, hyperglucagonemia, amplified renal

glucose reabsorption, and abnormalities in gut microbiota are all suggested as contributing factors by emerging research.

90% of monozygotic twins who have one diseased twin will eventually develop T2DM in their lives.[18] 50 polymorphisms have been identified as contributing to T2DM risk or protection. These genes produce proteins that play important roles in a number of DM-related pathways, such as pancreatic development, insulin synthesis, secretion, and development, amyloid deposition in beta cells, insulin resistance, and gluconeogenesis control issues. Genetic loci for the transcription factor 7-like 2 gene (TCF7L2), which raises the risk for type 2 diabetes, were discovered by a genome-wide association study (GWAS).[19,20] NOTCH2, JAZF1, KCNQ1, and WFS1 are some of the other loci that have been linked to the development of T2DM. According to reports, the human leukocyte antigens (HLA), also known as the major histocompatibility complex (MHC), are responsible for 40% to 50% of the familial aggregation of T1DM. Significant determinants include polymorphisms in the class II HLA genes encoding DQ and DR4-DQ8, as well as DR3-DQ2, which are present in 90% of T1DM patients.[21,22]

MODY is a complex condition characterised by early-onset non-insulin dependent diabetes (usually under 25 years). It does not involve autoantibodies like T1DM and has an autosomal dominant mode of transmission. Hepatocyte nuclear factor-1-alpha (HNF1A) and glucokinase (GCK) gene mutations, which are present in 52–65 % and 15–32% of cases of MODY, respectively, have an impact on this disease. Since some people have mutations but never progress to the disease and others get clinical symptoms of MODY but have no identified mutation, the genetics of this condition is still unknown.[23,24]

Gestational diabetes is essentially diabetes that manifests during pregnancy. Although the cause of its development is yet unknown, some suggest that HLA antigens, notably HLA

DR2, 3, and 4, may be involved. Additionally, it's assumed that high levels of proinsulin contribute to gestational diabetes, and some research suggests that proinsulin may cause beta-cell stress. Others think that elevated levels of hormones such progesterone, cortisol, prolactin, human placental lactogen, and oestrogen may have an impact on peripheral insulin sensitivity and beta-cell activity.[25]

Due to the intrinsic glucogenic action of the endogenous hormones that are excessively secreted in a number of endocrinopathies, including acromegaly, Cushing syndrome, glucagonoma, hyperthyroidism, hyperaldosteronism, and somato-statinomas, these conditions have been linked with glucose intolerance and diabetes mellitus. Due to excessive pancreatic iron deposition and beta cell death, conditions like idiopathic hemochromatosis are linked to diabetes mellitus.

Adults with latent autoimmune diabetes is another type of T1DM (LADA). It happens in maturity, frequently with a slower onset.[5,10] In general, destruction happens quickly in youngsters and much quicker in adults. These patients' serum may show autoantibodies against islet cells, insulin, glutamic acid decarboxylase-65 (GAD-65), and zinc transporter 8 (Zn T8). After the first year, in particular, these antibodies start to lose their potency and lack the requisite diagnostic precision to be employed frequently for diagnosis. There is little to no release of insulin due to the extensive destruction of beta cells. Most of these patients do not have obesity. They are more likely to develop autoimmune diseases such celiac disease, Addison disease, Graves' disease, and Hashimoto thyroiditis. Idiopathic T1DM refers to a subset of T1DM that is not connected to either the aforementioned HLA or insulin autoimmunity.It is commoner in African and Asians and presents with episodic diabetic ketoacidosis (DKA).

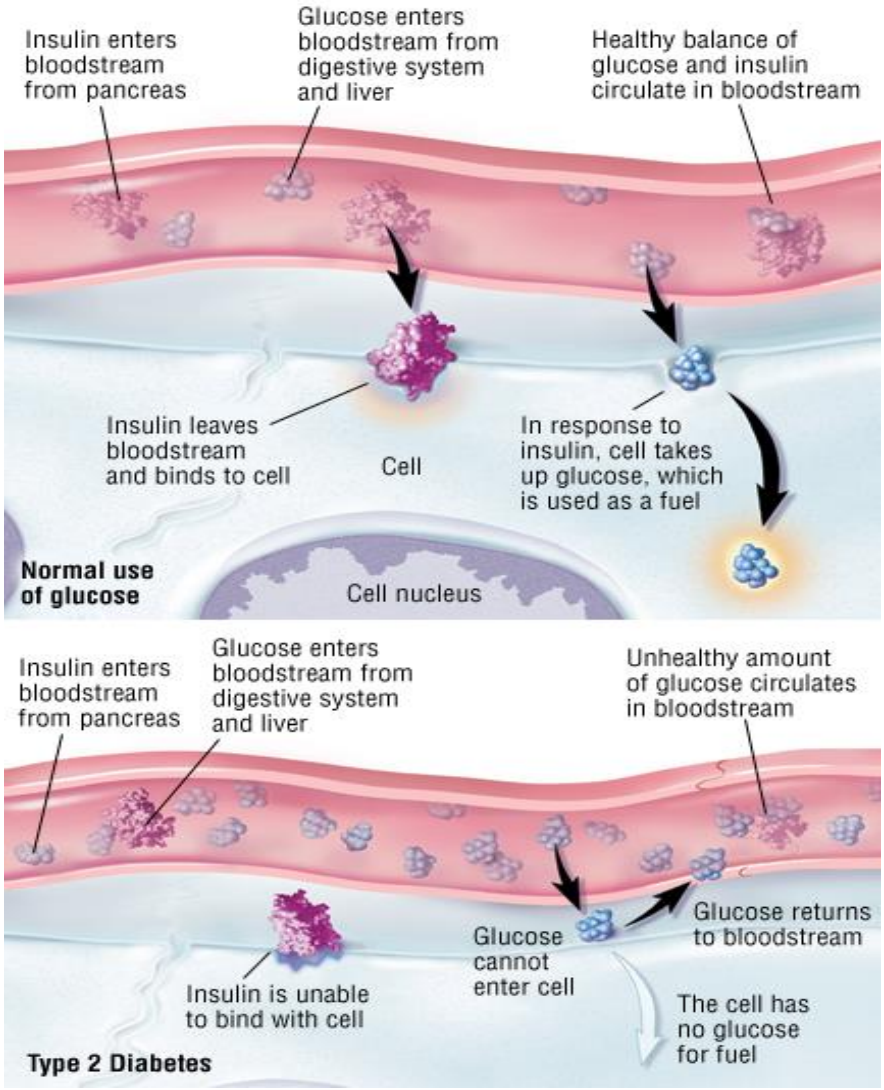


Figure 1: Pathogenesis of Diabetes Mellitus

Stages / Types	Normoglycemia	Hyperglycemia			
	Normal Glucose Regulation	Impaired Glucose Tolerance or Impaired Fasting Glucose (Prediabetes)	Diabetes Mellitus		
			Not insulin requiring	Insulin requiring for control	Insulin requiring for survival
Type 1*	←	←	←	←	→
Type 2	←	←	←	→	
Other Specific Types**	←	←	←	→	
Gestational Diabetes**	←	←	←	→	

Figure 2: Distribution of glucose levels in types of DM

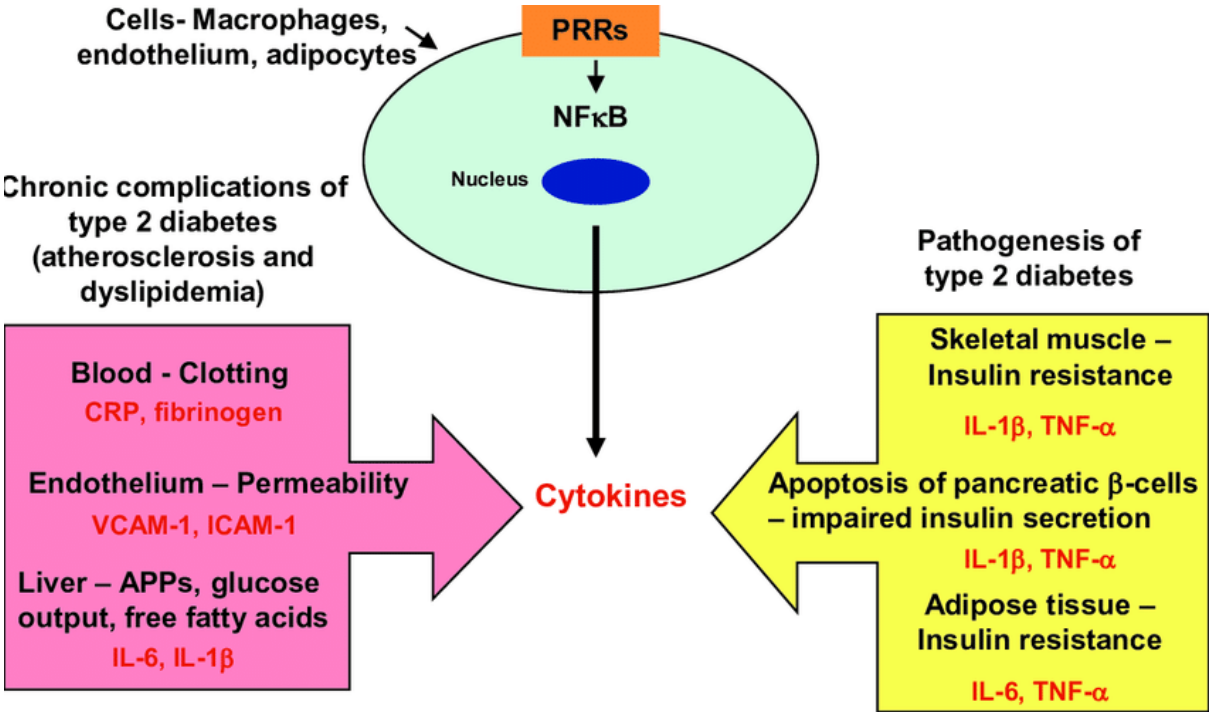
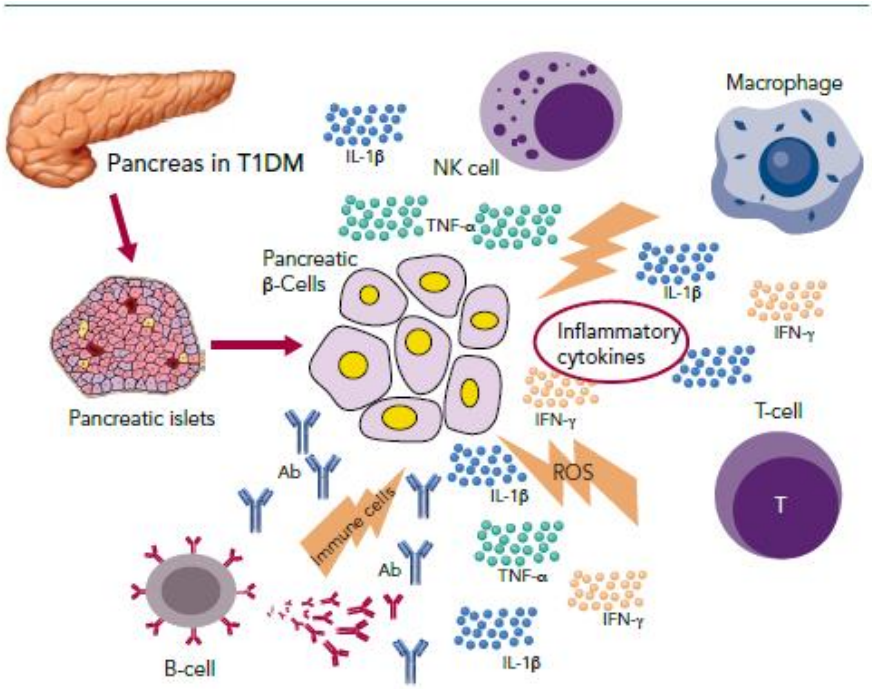


Figure 3: Role of inflammatory mediators in DM



Inflammation has a key role in the pathophysiology of type 2 diabetes and its associated metabolic abnormalities.

Figure 4: Inflammation in DM

b. RISK FACTORS and CLINICAL FEATURES OF DIABETES MELLITUS[6,26]

Risk Factors of Diabetes Mellitus

1. Positive family history of diabetes
2. Age >35 yr
3. Overweight (Body mass index ≥23 kg/m^2) and obesity (Body mass index ≥25 kg/m^2)
4. Enlarged waist or upper body adiposity (>90 cm for men and >80 cm for women)
5. Presence of hypertension
6. Recent weight gain
7. Sedentary lifestyle
8. Gestational diabetes

Figure 5: Risk factors for DM

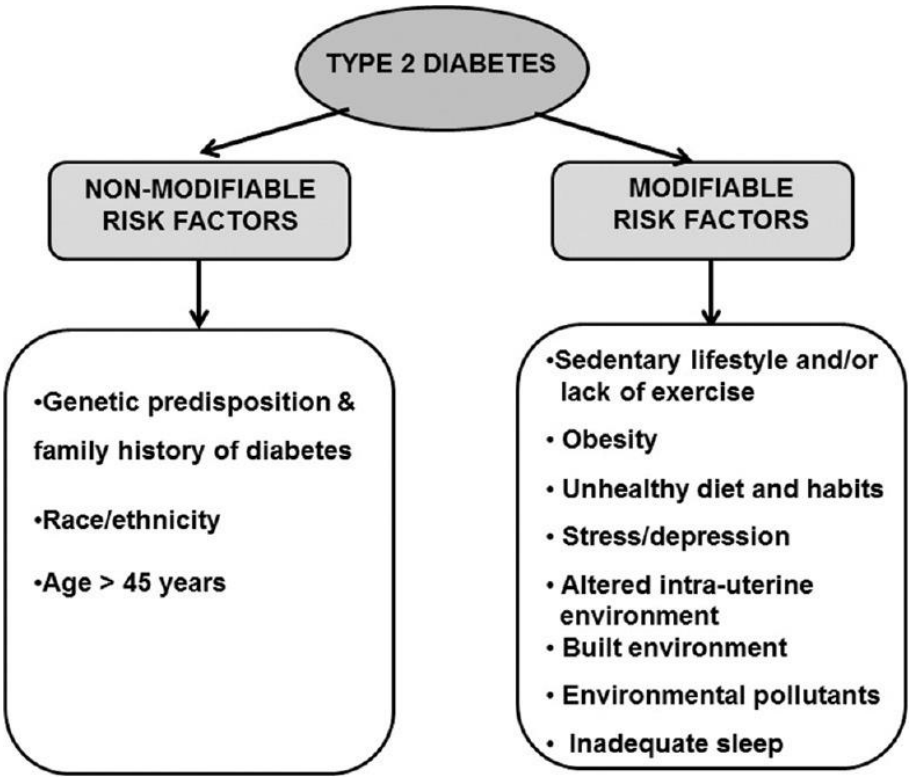


Figure 6: Modifiable and Non-modifiable risk factors of DM

Warning signs of Diabetes Mellitus[26]

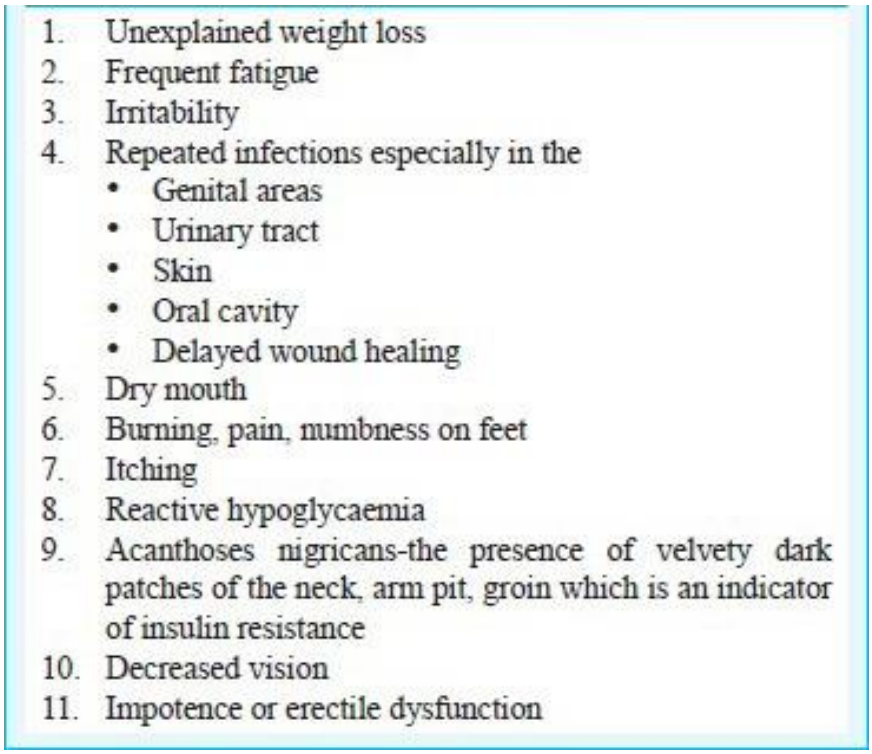


Figure 7: Warning signs of progression of DM

Type 1 diabetes, which develops rapidly into severe hyperglycaemia, as well as type 2 diabetes, which has very high levels of hyperglycaemia, typically present with the classic symptoms of diabetes, such as polyuria, polydipsia, and polyphagia. Only in type 1 diabetes or when type 2 diabetes goes undiagnosed for a long time does severe weight loss occur frequently. Other typical symptoms of undiagnosed diabetes include unexplained weight loss, exhaustion, restlessness, and physical pain.

COMPLICATIONS OF DIABETES MELLITUS

The complications can be acute or chronic. Acute complications include Hypoglycaemia, Diabetic Keto-acidosis, Hyperosmolar hyperglycaemic state (metabolic complication of diabetes mellitus (DM) characterized by severe hyperglycaemia, extreme dehydration, hyperosmolar plasma, and altered consciousness**).**

Chronic complications can be microvascular and macrovascular complications.[9]

General
Infections
Joint contractures
Hypoglycaemia/hyperglycaemia
Diabetic ketoacidosis
Non-ketotic hyperosmolar state

Macrovascular
Ischaemic heart disease
Hypertension
Stroke and peripheral vascular disease

Microvascular
Peripheral vascular disease
Nephropathy and renal failure
Retinopathies
Neuropathies (autonomic and somatic)

Figure 8: Classification of Complications of DM

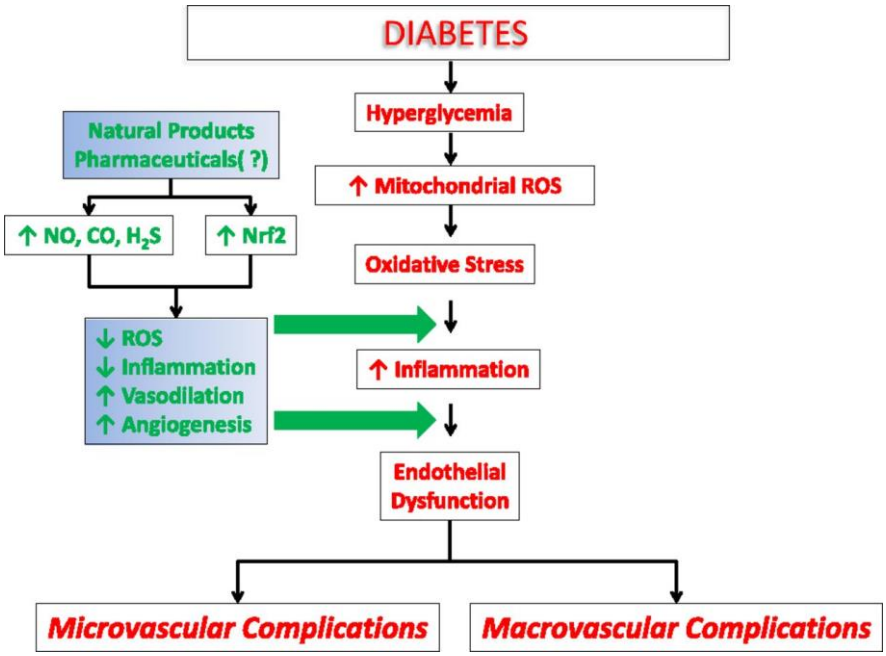


Figure 9: Aetiopathogenesis of complications of DM

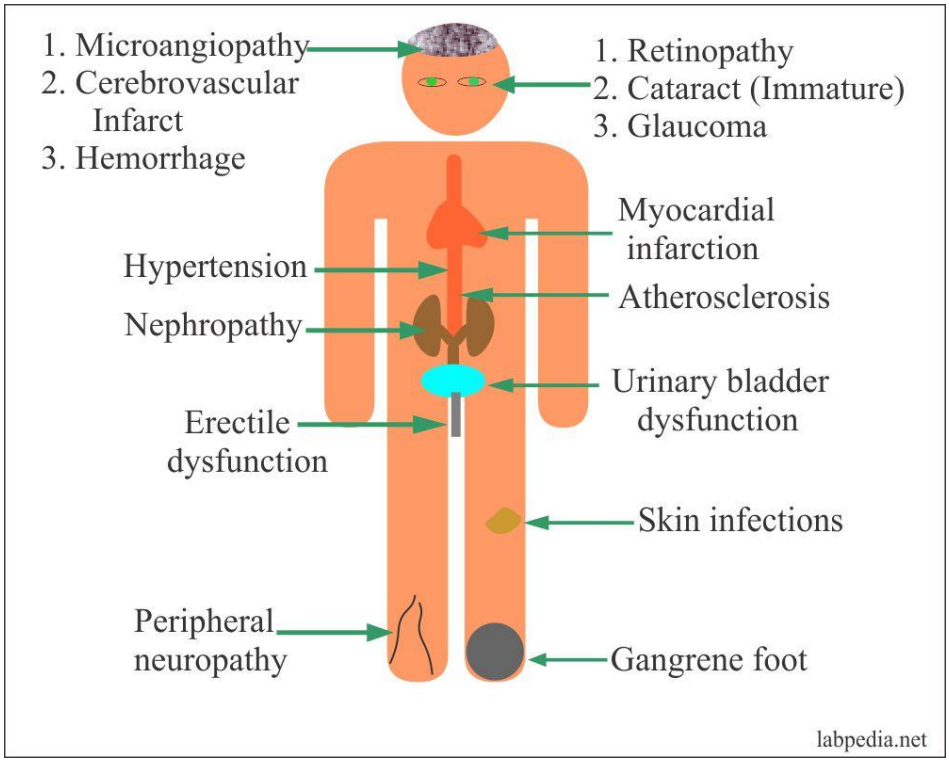


Figure 10: Complications of DM

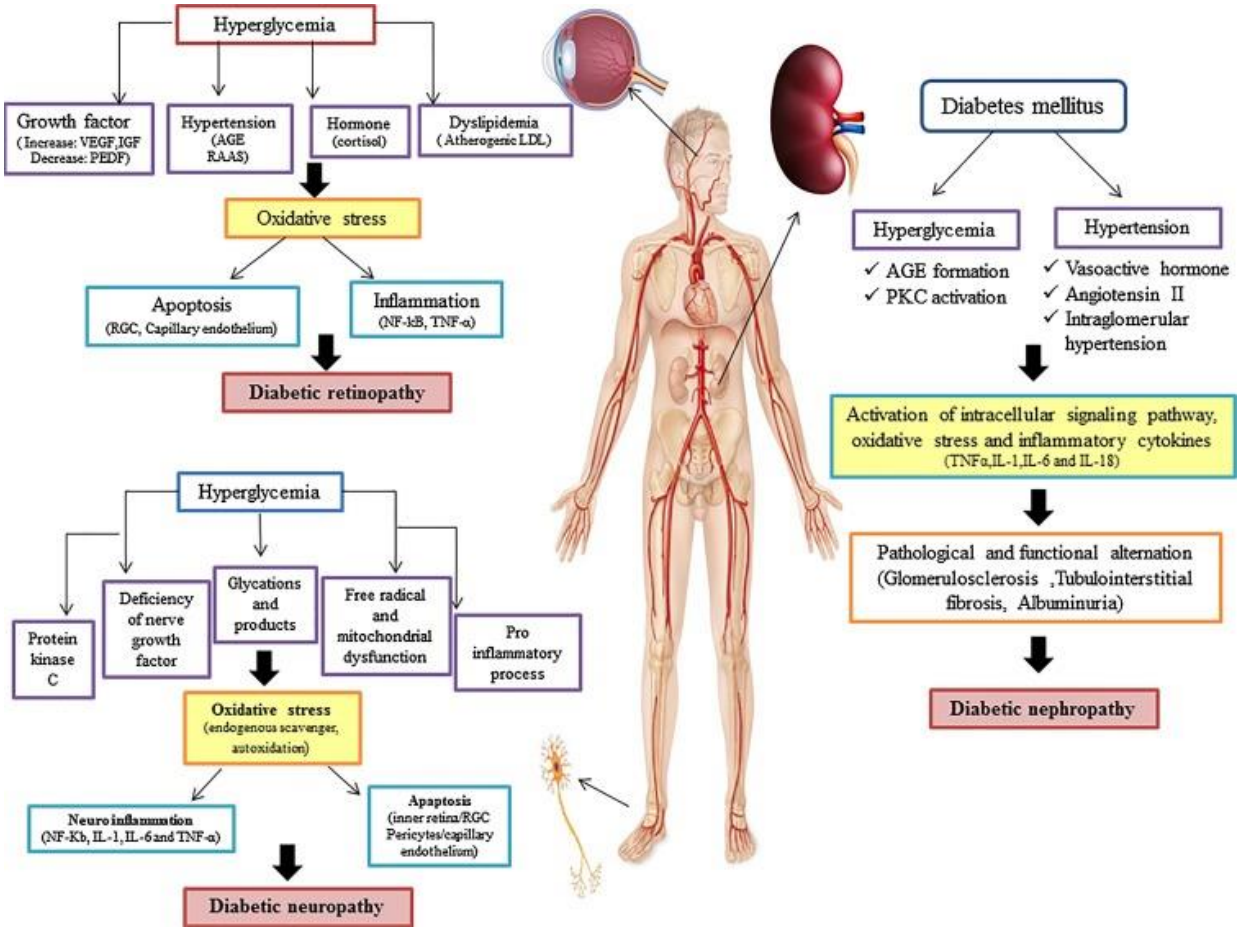


Figure 11: Factors involved in development of complications

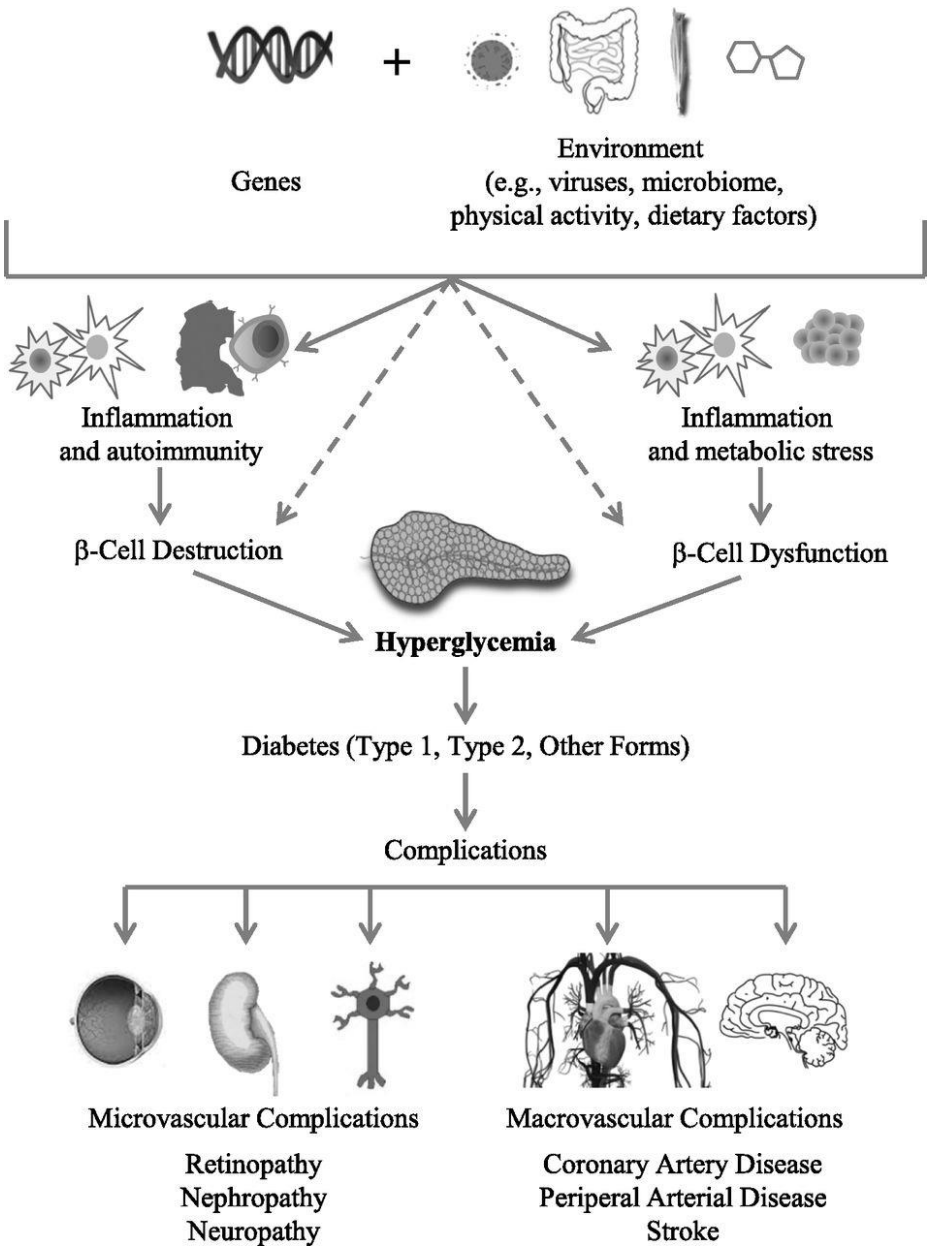


Figure 12: Risk factors and complications of DM

MONITORING OF BLOOD GLUCOSE LEVEL

Continuous monitoring of the blood sugar is required for preventing the complications of diabetes. Modern technology and electronics have made this probable by introducing test apparatus that can give digital displays on portable palm top devices. Urine and blood sugar measures are the most popular. [5,6,8,27]

1. Urine Testing: It is the simplest possible test for diabetes. The major limitation is that urine sugar will be positive only when the blood glucose level exceeds the kidney threshold. For most people this is when the blood sugar value is above 180 mg/dl and is much higher than the desired value between 60-120 mg/dl (optimal values vary) before meal. It thus helps only to monitor whether the blood glucose level exceeds the warning level. Hypoglycaemia cannot be detected by testing urine sugar.
2. Blood Testing: Blood tests give more accurate estimates for blood sugar. Two different methods are used to develop handy instruments for measuring blood sugar. Photometric meters use colour reflectance measures to detect the colour change on a strip pad caused by glucose in the blood. Electrochemical detection is an alternative method in that glucose in the blood causes a reaction on the test strip producing a tiny current. The meter detects the current and reports a digital test result. The optimal blood sugar (plasma glucose) values ranges from 60-120 mg/dl before meal and from 110-140 mg/dl after food for most people. Normal control is targeted to stabilise fasting blood sugar values around 95 mg/dl to avoid possible risk due to hypoglycaemia.
3. C-peptide Measure: c-peptide is a substance that the pancreas releases into the bloodstream in equal amounts to insulin. Unlike insulin, it is not absorbed by the

body and is washed out with urine. C-peptide levels show exactly how much insulin the body is making. Currently, tests based on the competition principle and the microtiter plate separation are available in kit form.

4. Glyco-haemoglobin (HbA1c) Measure[28–30]: Glyco-haemoglobin measure is one of the most decisive measures indicating possible microvascular complications of diabetes. The procedure (HPLC method) uses handheld devices. Monoclonal antibody method is used in these machines to quantitatively determine the glycol-haemoglobin contained in a drop of blood. A test that reflects long-term blood glucose control in diabetics is the concentration of haemoglobin A1c. When hemolysates of red cells are chromatographed, three or more small peaks named haemoglobin A1a, A1b, and A1c are eluted before the main haemoglobin A peak. These "fast" haemoglobins are formed by the irreversible attachment of glucose to the haemoglobin in a two-step reaction. The percentage of haemoglobin glycosylated depends on the average glucose concentration the red cell is exposed to over time. Since the average life of the red cell is 120 days, the percentage of glycosylated haemoglobin gives a good indication of the degree of blood sugar control over the preceding weeks. Numerous biochemical methods are used including electrophoresis, mini columns, radioimmunoassay, and high-pressure liquid chromatography.
5. Home Glucose monitoring: Glucose oxidase and reagents to measure the generation of hydrogen peroxide can be bonded to filter paper and the system used to measure glucose concentrations in a drop of capillary blood. This has resulted in the most important change in diabetes management since the introduction of insulin. Patients are instructed to obtain a blood sample by pricking their fingertip with a lancet. Spring-loaded lancets are available. They are easy to use and cause minimal

discomfort. Surprisingly, many patients consider the discomfort of the finger stick preferable to the inconvenience and aesthetic unpleasantness of obtaining a urine sample for testing. A drop of whole capillary blood is then placed on the reagent bonded to the paper strip. The blood reacts with glucose oxidase enzyme for a finite time period of 1 minute. The excess blood is removed by washing or wiping and the colour is allowed to develop. The concentration is then estimated by comparing to a colour chart, or by using a portable reflectance meter specific to the reagent strip, to measure the developed colour. Reflectance meters for measuring blood glucose are becoming increasingly sophisticated, compact, and reliable. Shirt-pocket-size models are now available, and prototype models that store the time, date, result, and insulin doses for later graphic printing at the patient's home or physician's office have been developed.[8]

DIAGNOSIS[6,8,10]

Diabetes can be diagnosed either by the haemoglobin A1C criteria or plasma glucose concentration (fasting or 2-hour plasma glucose).

Fasting Plasma Glucose (FPG)

A blood sample is taken after an 8 hour overnight fast. As per ADA, fasting plasma glucose (FPG) level of more than 126 mg/dL (7.0 mm/L) is consistent with the diagnosis.

Two-Hour Oral Glucose Tolerance Test (OGTT)

In this test, the plasma glucose level is measured before and 2 hours after the ingestion of 75 gm of glucose. DM is diagnosed if the plasma glucose (PG) level in the 2-hour sample is more than 200 mg/dL (11.1 mmol/L). It is also a standard test but is inconvenient and more

costly than FPG and has major variability issues. Patients need to consume a diet with at least 150 g per day of carbohydrate for 3 to 5 days and not take any medications that can impact glucose tolerance, such as steroids and thiazide diuretics.

Glycated Haemoglobin (Hb) A1C

This test gives an average of blood glucose over the last 2 to 3 months. Patients with an Hb A1C greater than 6.5% (48 mmol/mol) are diagnosed as having DM. Hb A1C is a convenient, rapid, standardized test and shows less variation due to pre-analytical variables. It is not much affected by acute illness or stress.

Hb A1C is costly and has many issues, as discussed below, including lower sensitivity. Hb A1C should be measured using the National Glyco-haemoglobin Standardization Program (NGSP) certified method standardized to Diabetes Control and Complications Trial (DCCT) assay. It is affected by numerous conditions such as sickle cell disease, pregnancy, haemodialysis, blood loss or transfusion, or erythropoietin therapy. It has not been well validated in non-Caucasian populations.

Anaemia due to deficiency of iron or vitamin B12 leads to spurious elevation of Hb A1C, limiting its use in countries with a high prevalence of anaemia. Also, in children and the elderly, the relation between Hb A1C and FPG is suboptimal.

In patients with classic symptoms of hyperglycaemia (increased thirst, increased hunger, increased urination), random plasma glucose more than 200 mg/dL is also sufficient to diagnose DM.FPG, 2-hour PG during 75-g GTT, and Hb A1C are equally appropriate for the diagnosis of DM.

VITAMIN D[31,32]

Because it is created in the skin when exposed to sunlight, vitamin D is known as the "sunshine vitamin." To keep serum calcium levels within the typical physiological range for musculoskeletal health, vitamin D is necessary. Vitamin D deficiency is defined by the Endocrine Society, the National and International Osteoporosis Foundation, and the American Geriatric Society as a 25-hydroxyvitamin (25 OH D) level of less than 30 ng/Ml. The recommended range, according to the Endocrine Society, is 40 to 60 ng/mL.

A hormone called vitamin D can be gained through diet and skin synthesis. 7-dehydrocholesterol in the skin is converted to pre-vitamin D by ultraviolet B (UVB) radiation with a wavelength of 290 to 315 nm. Heat isomerization transforms this pre-vitamin D into vitamin D. The liver converts dietary and skin-derived vitamin D into 25-hydroxyvitamin D (25 OH D), which can be used to determine one's vitamin D level. 25 hydroxyvitamin D-1 alpha-hydroxylase transforms 25 hydroxyvitamin D to 1,25-dihydroxyvitamin D (1,25 (OH)), which is physiologically active (CYP27B1) in the kidneys. The levels of parathyroid, calcium, and phosphorus control the renal synthesis of 1,25-dihydroxyvitamin. CYP27B1 gene expression in the kidney is mediated by various factors. Parathyroid hormone (PTH), hypocalcaemia, hypophosphatemia, and calcitonin affect the activation of CYP27B1 and can increase 1,25-(OH)2D levels. On the other hand, 1,25-(OH)2D and fibroblast growth factor-23 (FGF-23) inhibit CYP27B1 and can decrease 1,25-(OH)2D levels.[33,34]

A physiological role of vitamin D exists independent of calcium metabolism. The small intestine, colon, T and B lymphocytes, mononuclear cells, brain, and skin all contain vitamin D receptors. It increases the generation of insulin, controls the activity of T and B

lymphocytes that have been activated, guards against inflammatory bowel illnesses, and influences cardiac contractility.

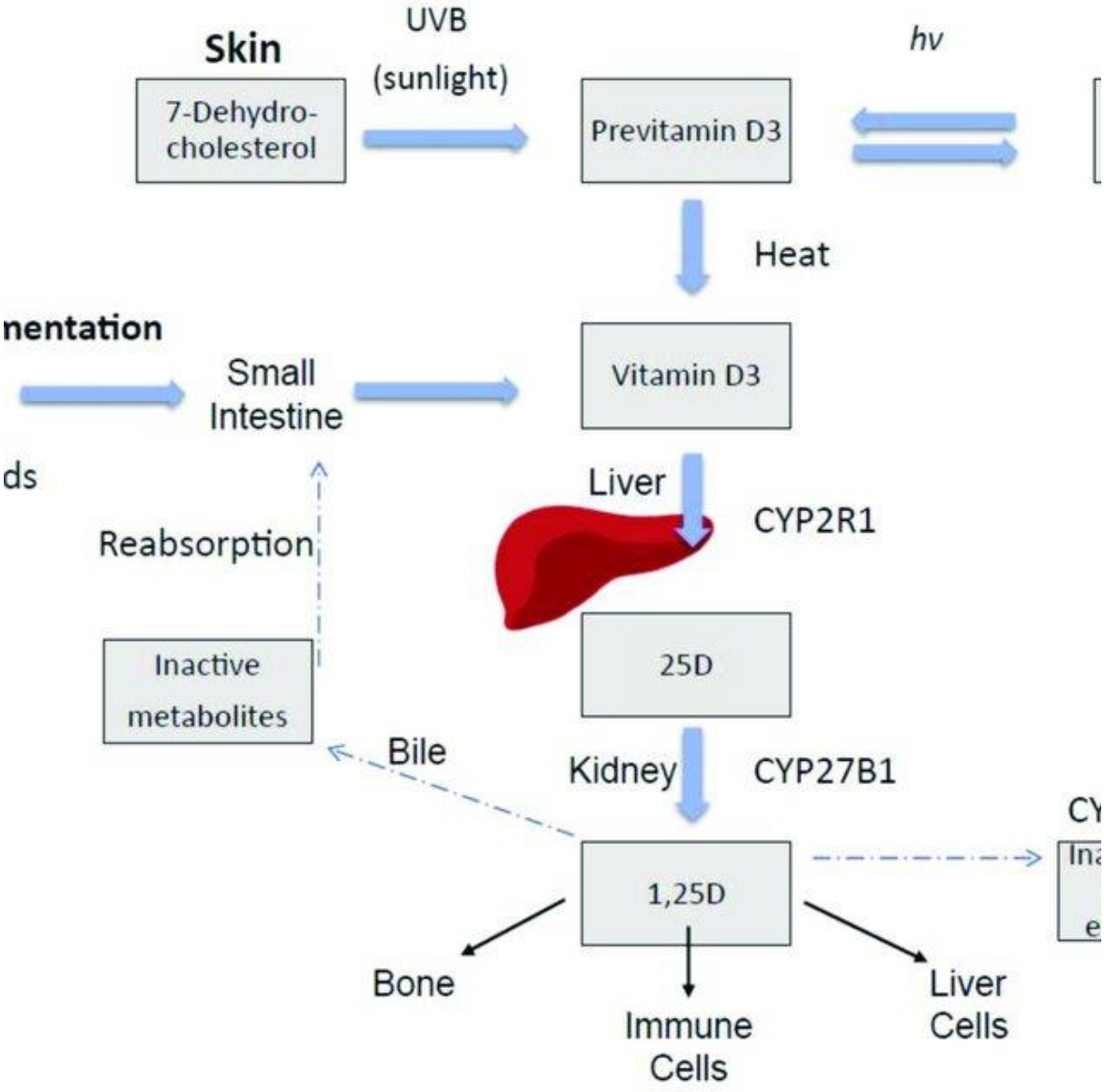
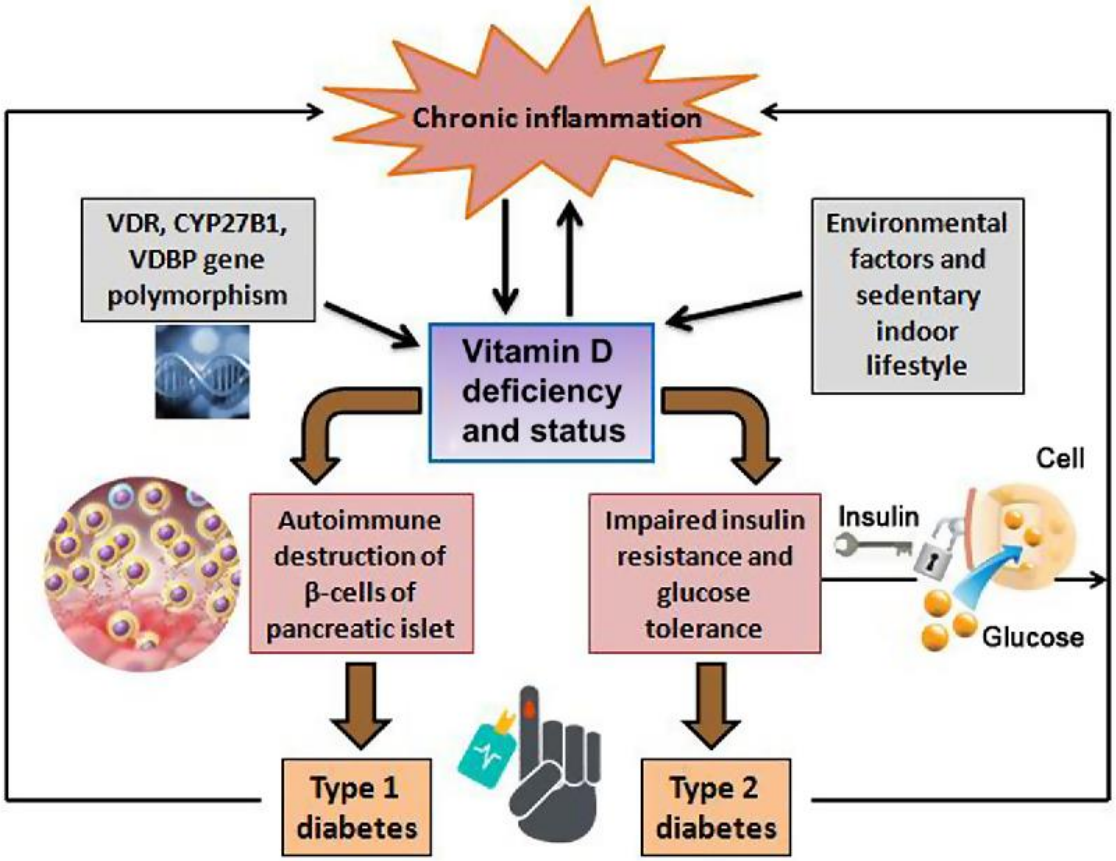


Figure 13: Synthesis of Vitamin D

Role of Vitamin D in Diabetes Mellitus[35–42]

There is emerging evidence that suggests vitamin D insufficiency may have a role in both type 1 and type 2 diabetes development. First, it has been established that VDRs and the 1 alpha hydroxylase enzyme are present in the pancreatic -cell, which secretes insulin. There is evidence that vitamin D supplementation improves insulin resistance and glucose tolerance. Reduced insulin secretion is a consequence of vitamin D insufficiency. Animal studies have demonstrated that vitamin D supplementation can restore insulin secretion. 49 Additionally, scientists have discovered a possible indirect effect of calcium on insulin secretion. Low vitamin D may reduce calcium's capacity to alter insulin secretion because vitamin D helps to normalise extracellular calcium, guaranteeing proper calcium flux through cell membranes. The improvement of insulin action by promoting insulin receptor expression, improving insulin responsiveness for glucose transport, having an indirect effect on insulin action possibly via a calcium effect on insulin secretion, and reducing systemic inflammation by a direct effect on cytokines are additional potential mechanisms linked to vitamin D and diabetes. Vitamin D also acts as a potent immunosuppressor. It tends to down-regulate the transcription of various proinflammatory cytokine genes like Interleukin-2, Interlukin-12 and Tumour Necrosis Factor-alpha. It promotes the induction of regulatory T-lymphocytes, the production of anti-inflammatory cytokines and protects beta-cell from destruction.[43,44]



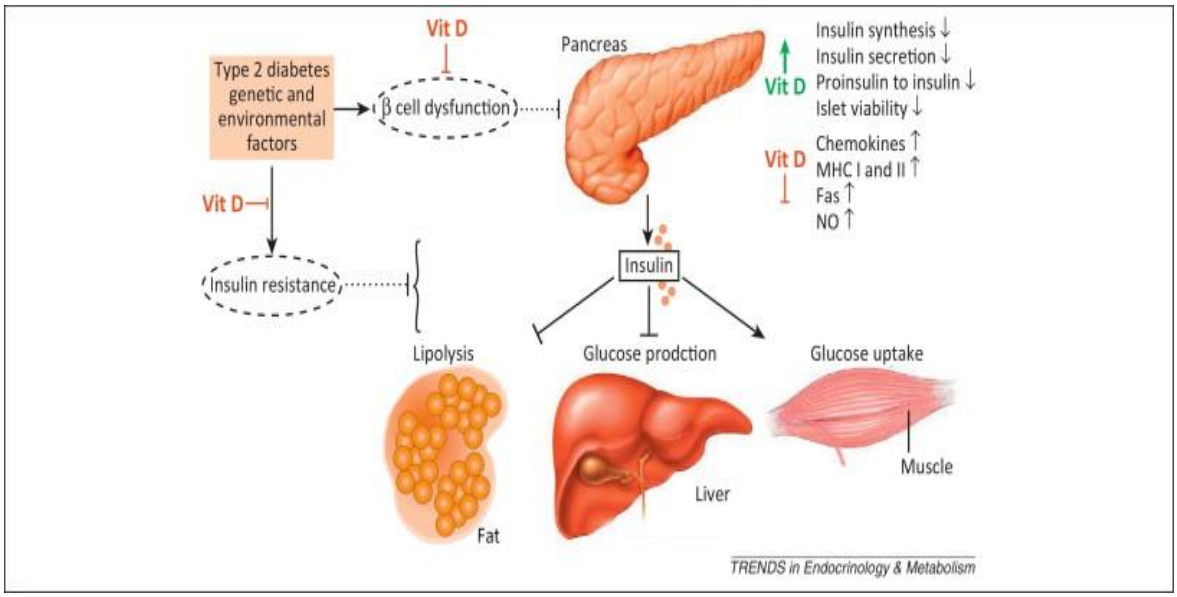


Figure 14 & Figure 15: Vitamin D and Diabetes Mellitus

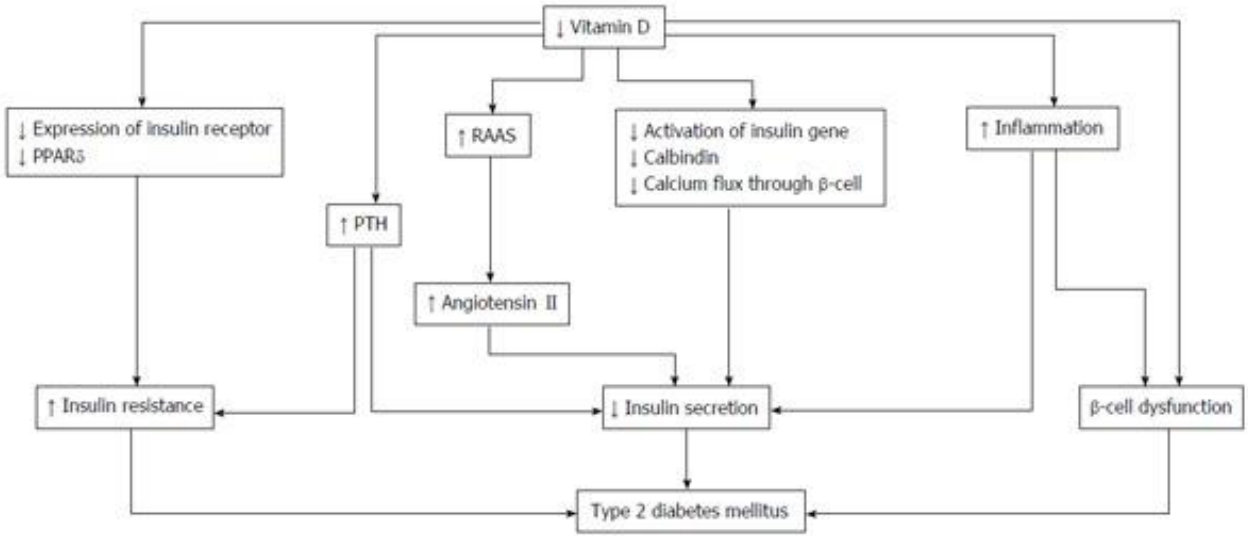


Figure 16: Vitamin D and Type II DM

Vitamin D insufficiency is linked to insulin secretion, insulin resistance, and pancreatic -cell malfunction because VDRs in pancreatic -cells are crucial in the development of type 2 DM. By controlling intracellular calcium, vitamin D modulates the activity of calbindin, a systolic calcium-binding protein present in pancreatic beta-cells, and inhibits the release of insulin in response to depolarization. Vitamin D is linked to insulin sensitivity as well. Vitamin D controls insulin sensitivity by promoting the expression of insulin receptors. A further way that vitamin D improves insulin sensitivity is by encouraging the expression of the nuclear receptor fatty acid sensor known as peroxisome proliferator-activated receptor (PPAR) delta, which is a member of the PPAR family and controls fatty acid levels in skeletal muscle and adipose tissue. Peripheral insulin resistance is significantly influenced by intracellular calcium through a dysfunctional signal transduction pathway that results in reduced glucose transporter activity.[38,45–50]

Reviews of articles depicting the association between Vitamin D levels and Diabetes Mellitus

1. **Kabadi and Patil (2021)**[51] did a study to evaluate the clinical profile of newly-detected-type-II-DM patients in relation to vitamin D levels among 150 newly-detected-type-II-DM patients. The mean age was 49.17 ± 12.72. One hundred and eleven (74%) of them had vitamin D levels <30 ng/dL. Mean vitamin D levels were 24.24 ± 11.20. Mean HbA1c was 10.96 ± 1.78, 9.66 ± 1.37, and 7.05 ± 0.65, among patients having their vitamin D levels ranging <20, 20–30, and >30 ng/dL, respectively, showing p value < 0.001. Mean BMI was 29.90 ± 2.18, of 111 of them who had vitamin D levels <30 ng/dL, 74 of them had BMI (18.5–22.9), 20 had BMI (23–24.9), and 17 of them had BMI >25. Vitamin D levels can be independent risk factors for the development of DM and obesity and hence must be treated promptly.
2. **Zakhary et al (2021)**[52] systematically screened four databases for relevant information; PubMed, Medline, PMC, and Google scholar. Data were collected from 14 articles, of which eight are systematic reviews and meta-analysis, one is a narrative review, five are randomized controlled trials and three are general information about DM II and Vitamin D. In addition, this article evaluates the clinical significance of Vitamin D administration in DM II from a glucose homeostasis perspective, and complications such as nephropathy, neuropathy, and retinopathy. Vitamin D had a clinical positive impact on glucose level, particularly on hemoglobin A1c (HbA1c) reduction, alleviation of diabetic neuropathy and nephropathy symptoms, and hyperglycemia induced-oxidative stress on the retinal cells.
3. **Salih et al (2021)**[37] did a study to evaluate impact of Vit D on DM.The mean age of patients was 49.94 ± 9.36, while the mean age the controls was 48.95 ± 10.56.

Females constituted 56.1% and males 43.9% in the cases group, while for the control group females were 54.8% and males were 43.9%. Low vitamin D levels were detected in 110 (71%) of cases and 63 (40.6%) of controls. There was a significant difference in vitamin D levels among cases and controls ($p < 0.001$), vitamin D level was lower among females compared to males, $p < 0.001$ and those living in urban areas compared to rural areas, $p < 0.001$, BMI and dyslipidemia had a significant effect on vitamin D levels among diabetics, p values 0.002 and < 0.001 respectively. The serum 25(OH)-D level was significantly lower in patients with poor glycaemic control compared to those with good glycaemic control and in patients with a diabetes duration greater than 5 years, p values < 0.001 and 0.002 respectively. No significant correlation was detected with age and smoking, p values 0.181 and 0.260 respectively.

4. **Ahmed et al (2020)**[53] did a study to determine if vitamin D2 and D3 levels differed between those with and without T2DM among 274 with DM and 222 without T2DM, and the relationship between diabetic microvascular complications and vitamin D2 and vitamin D3 levels in subjects with T2DM. All subjects were taking vitamin D2 and none were taking D3 supplements. Vitamin D2 levels were higher in diabetics, particularly in females, and higher levels were associated with hypertension and dyslipidemia in the diabetic subjects ($p < 0.001$), but were not related to diabetic retinopathy or nephropathy. Vitamin D3 levels measured in the same subjects were lower in diabetics, particularly in females ($p < 0.001$), were unrelated to dyslipidemia or hypertension, but were associated with retinopathy ($p < 0.014$). Neither vitamin D2 nor vitamin D3 were associated with neuropathy. For those subjects with hypertension, dyslipidemia, retinopathy or neuropathy, comparison of highest with lowest tertiles for vitamin D2 and vitamin D3 showed no difference.

5. **Zhao et al (2020)**[54] did a study to determine the relationship between 25-hydroxyvitamin D [25(OH) D] and glycated hemoglobin (HbA1c) levels in male and female patients with type 2 diabetes mellitus (T2DM). HbA1c levels in the vitamin D deficiency group were significantly higher than those in the no vitamin D deficiency group for all subjects. The same was true for female patients but not for male patients. There was no difference in HbA1c levels between male and female patients with T2DM, regardless of 25(OH) D deficiency. A negative correlation existed between 25(OH) D and HbA1c in all subjects, as well as in the male-only and female-only subgroups. Vitamin D deficiency was associated with high HbA1c levels before and after adjusting for confounding factors in all participants and in the female-only subgroup, but not in the male-only subgroup.
6. **Anyanwu et al (2020)**[55] did a study to determine Vit D levels inT2DM among 114 eligible type 2 diabetes mellitus participants and 60 healthy controls. The mean age was 52 ± 7.6 years in the T2DM group and 50 ± 8.4 in the control group, (p = 0.9). The female to male ratio in both T2DM and healthy control subjects was 1.5:1. Majority of the study subjects had vitamin D deficiency with prevalence of 72 (63.2%) in T2DM subjects and 32 (53.3%) in the controls (p = NS). There was no significant difference in the distribution of Vitamin D3 deficiency status by age or sex in both T2DM and Control groups. The mean serum vitamin D level in T2DM subjects with vitamin D deficiency was 9.2 ± 1.1 ng/dl and 21.5 ± 0.7 ng/dl in the sufficient group (t = 11.9, p = 0.0001). The mean HbA1c and Fasting plasma glucose were higher in the vitamin D deficient group compared to the sufficient group (7.5 ± 1.9% and 148 ± 60.9 mg/dl vs. 6.8 ± 1.6% and 134 ± 43.5 mg/dl respectively, p NS). The proportion of subjects with good glycaemic control (HbA1C f 7.0%) was

significantly higher in the vitamin D sufficient group 19 (73.1%) compared to the vitamin D deficient group, 33 (45.8%), Z = -2.39, p = 0.01).

7. **Santhoshkumar et al (2019)**[56] studied Eighty patients diagnosed with type 2 DM Vitamin D deficiency was observed in 52.5% of the patients. Vitamin D levels were not associated with markers of glycaemic control or insulin resistance. Hypovitaminosis D was observed in more than half of the patients with type 2 diabetes. suggesting a potential for vitamin D supplementation in type 2 DM patients.
8. **Pittas et al (2019)**[42] did a study to see whether vitamin D supplementation lowers the risk of diabetes among randomly assigned adults who met at least two of three glycaemic criteria for prediabetes (fasting plasma glucose level, 100 to 125 mg per deciliter; plasma glucose level 2 hours after a 75-g oral glucose load, 140 to 199 mg per deciliter; and glycated hemoglobin level, 5.7 to 6.4%) and no diagnostic criteria for diabetes to receive 4000 IU per day of vitamin D3 or placebo, regardless of the baseline serum 25-hydroxyvitamin D level. A total of 2423 participants underwent randomization (1211 to the vitamin D group and 1212 to the placebo group). By month 24, the mean serum 25-hydroxyvitamin D level in the vitamin D group was 54.3 ng per milliliter (from 27.7 ng per milliliter at baseline), as compared with 28.8 ng per milliliter in the placebo group (from 28.2 ng per milliliter at baseline). After a median follow-up of 2.5 years, the primary outcome of diabetes occurred in 293 participants in the vitamin D group and 323 in the placebo group (9.39 and 10.66 events per 100 person-years, respectively). The hazard ratio for vitamin D as compared with placebo was 0.88 (95% confidence interval, 0.75 to 1.04; P=0.12). The incidence of adverse events did not differ significantly between the two groups.

9. **Mamatha B Patil and Raghav (2018)**[57] did a study to determine the role of Vitamin D in diabetic patient and in insulin regulation among 156 female diabetic patients aged 30 to 60 years. Mean age of study group: 48.47±9.56 years. Mean duration of diabetes in the study population: ± SD: 5.10±4.36 years mean BMI was 24.97±4.16.Mean waist circumference: 98.93±12.18 cm, Mean Waist-Hip ratio: 0.98±0.08. Mean FBS: 202.73±81.73 mg/dl, Mean PPBS: 280.99±94.14 mg/dl, Mean HbA1C: 9.33±1.83 %. Almost all diabetic females (92.5%) have Vitamin-D deficiency. Mean Vitamin-D levels: 16.19±8.97 ng/ml. Duration of diabetes Poor Glycaemic control and increased BMI had significant role in causing Vitamin-D deficiency.
10. **Liu et al (2015)**[58] **did a review** to assess the relationship between serums vitamin D level and type 1 diabetes mellitus (T1DM) in children. A total of 10 studies were included in this study. Our results showed that serum vitamin D was significantly lower in children with T1DM than in healthy controls (MD = -0.60, P <0.05)
11. **Mauss et al (2015)**[53] This cross-sectional study (2009-2011) involved 1821 employees of a German engineering company (83.1% male, mean age 51.9 ±5.6 years. Severe vitamin D deficiency (<10 ng/ml) was associated with increasing FPG ($p \leq 0.01$) and HbA1c ($p \leq 0.001$) values in adjusted linear regression models. In multivariable models, severe vitamin D deficiency was associated with DM ($p \leq 0.05$) after controlling for potential confounders**.**
12. **Ifigenia Kostoglou-Athanassiou et al (2013)**[59] did a study to determine levels of 25-hydroxy vitamin D3 [25(OH)D3] and the relationship between 25(OH)D3 levels and glycaemic control in 120 patients with diabetes mellitus type 2 and 120 controls. 25(OH)D3 levels were lower in the diabetes mellitus type 2 patients than in the

control group, being 19.26 ± 0.95 ng/ml and 25.49 ± 1.02 ng/ml, in the patient and control groups, respectively ($p < 0.001$, Student's t-test). 25(OH)D3 levels were found to be inversely associated with HbA1c levels in the diabetic patients ($p = 0.008$, $r2 = 0.058$, linear regression). 25(OH)D3 levels were found to be inversely associated with HbA1c when the patient and control groups were analysed together ($p < 0.001$, $r2 = 0.086$).

5 RESEARCH QUESTION OR HYPOTHESIS

5.1 **RESEARCH QUESTION:**

Whether there is any association between Vitamin D levels and Diabetes Mellitus?

5.2 **NULL HYPOTHESIS:**

There is no association between Vitamin D levels and Diabetes Mellitus

5.3 ALTERNATE HYPOTHESIS:

There is association between Vitamin D levels and Diabetes Mellitus

6 METHODOLOGY

6.1 **STUDY SUBJECTS:**

Patients with Diabetes Mellitus admitted in Inpatient wards , Department of General Medicine, Thanjavur Medical College.

6.2 **STUDY DESIGN:**

Cross sectional observational study.

6.3 **STUDY SETTING:**

Inpatient wards , Department of General Medicine, Thanjavur Medical College.

6.4 **SAMPLING PROCEDURE:**

Patients with Diabetes Mellitus admitted in Inpatient wards , Department of General Medicine, Thanjavur Medical College were included in the study

6.5 INCLUSION CRITERIA:

1. Women age more than 18 years

2. diabetic normal and obese women

3. Patients who are willing to give written, informed consent

6.6 **EXCLUSION CRITERIA:**

1. Patient who are type1 diabetes mellitus

2. Patient with known renal disease, Crohn's disease, cystic fibrosis, celiac disease, malabsorption syndrome

3. Patients who have gestational diabetes mellitus

4 Patient with history of steroid intake and oral contraceptives pills for more than 6 months

5. Patient who are on calcium supplements

6. Patient who are on vitamin D supplement

7. Patient who have history of any types of carcinoma

6.7 **SAMPLE SIZE:**

According to Mamatha B patil et al[57] study, considering the prevalence of vitamin D deficiency in Diabetes as 92.5% with a precision of 6% and 95% confidence interval, the sample size is calculated as

$N = Z21\text{-}\alpha/2 * p * (1 - p) / d2$

Z1-α/2 - two tailed probability for 95% confidence interval = 1.96

p (%) - prevalence of vitamin D deficiency in Diabetes = 0.925

d (%) - precision or allowable error for vitamin D deficiency in Diabetes = 0.06

$N = 1.96^2 * 0.925 * (1 - 0.925) / 0.06^2$

N = 74.03

Thus the total sample size required for the study is 100 after addition of 20% non responsive error

6.8 **STUDY PROCEDURE:**

100 patients were selected according to inclusion and exclusion criteria and written informed consent was taken for their participation. Patient history in relation to the duration of type2 diabetes was also taken. Patients were evaluated for anthropometric measures like height, weight, waist/hip ratio, glycaemic value, renal function test, urine routine and vitamin D levels. Vitamin D levels were correlated with the anthropometric measure and glycaemic control

PARAMETERS ANALYSED

- Anthropometric measures (Height, Weight, waist hip ratio)
- Fasting and post prandial blood sugars,
- Fasting lipid profile
- Renal function tests
- Urine routine
- Vitamin D levels

6.9 **ETHICAL CONSIDERATION:**

Institutional Ethical Committee approval was obtained before the start of the study.

Informed written consent was obtained from each participant.

Source of Funding: NIL

Conflict of Interest: NIL

6.10 STUDY PERIOD:

2 years

6.11 STATISTICAL METHODS:

Descriptive Statistics:

1. Numerical variables like age are represented in mean, median, mode and standard deviation.
2. Categorical variables like gender are represented in frequencies and percentages. Pie-charts and bar diagrams are used as appropriate.

Inferential Statistics:

3. When a Numerical variable is associated with the Numerical variables such as Pearson's correlation test is used after checking for normality. The Pearson product-moment correlation coefficient (Pearson's correlation, for short) is a measure of the strength and direction of association that exists between two variables measured on at least an interval scale.
4. When a Categorical Variable is associated with a categorical variable, the variables are represented in both by tables and bar diagrams. For test of significance, chi-square test is used. The chi-square test for independence, also called Pearson's chi-square test or the chi-square test of association, is used to discover if there is a relationship between two categorical variables. Fisher's exact test is used when more than 20% of the cell values have expected cell value less than 5.
5. The independent-samples t-test (or independent t-test, for short) compares the means between two unrelated groups on the same continuous, dependent variable.
6. The one-way analysis of variance (ANOVA) is used to determine whether there are any statistically significant differences between the means of three or more independent (unrelated) groups
7. P-values less than 0.05 were considered statistically significant.
8. Data was entered in MS excel sheet and analysed using SPSS software version 16.

7 RESULTS

Results of the study is discussed under the following headings:

I. Age (years)

II. Age group

III. Weight (kg)

IV. Height (cm)

V. BMI (kg/m2)

VI. Obesity

VII. Waist Hip ratio

VIII. Duration of Diabetes (years) with Obesity

IX. Fasting Blood Sugar (mg/dl) with Obesity

X. Post prandial blood sugar (mg/dl) with Obesity

XI. Total Cholesterol (mg/dl) with Obesity

XII. Blood Urea (mg/dl) with Obesity

XIII. Comparision of Urine sugar with the Obesity

XIV. Serum Vitamin D (ng/ml) with Obesity

XV. Comparision of Vitamin D level with the Obesity

XVI. Duration of Diabetes (years) with Vitamin D level

XVII. Fasting Blood Sugar (mg/dl) with Vitamin D level

XVIII. Post prandial blood sugar (mg/dl) with Vitamin D level

XIX. Total Cholesterol (mg/dl) with Vitamin D level

XX. Blood Urea (mg/dl) with Vitamin D level

XXI. Linear Regression for predicting Serum Vitamin D (ng/ml)

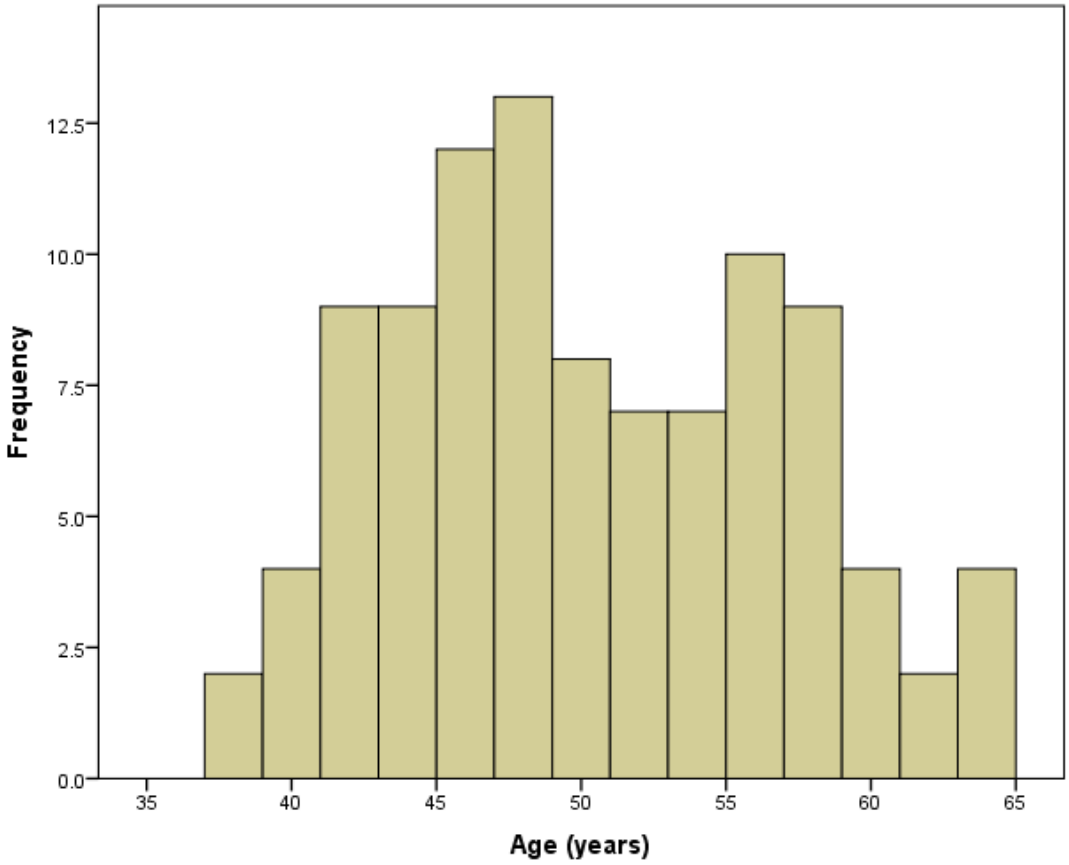
I. Age (years)

The mean Age (years) among the subjects was 49.94 (± 6.69) years ranging from 38 to 64 years

Table 2. Age (years)

Age (years)	
Mean	49.94
Median	49
Std. Deviation	6.69
Range	26
Minimum	38
Maximum	64

Figure 17. Age (years)



II. Age group

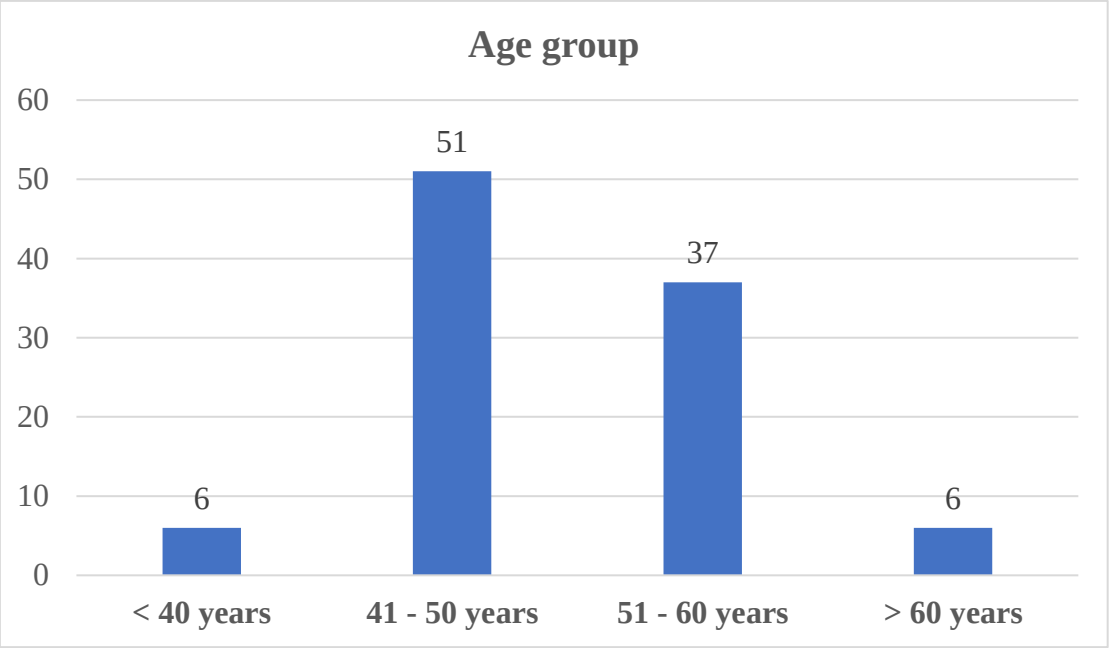
Among the subjects, 51 (51%) were in 41 - 50 years, 37 (37%) were in 51 - 60 years, 6 (6%) were in > 60 years and 6 (6%) were in < 40 years.

Table 3. Age group

Age group	Frequency	Percent
< 40 years	6	6.00
41 - 50 years	51	51.00
51 - 60 years	37	37.00

> 60 years	6	6.00
Total	100	100.00

Figure 18. Age group



III. Weight (kg)

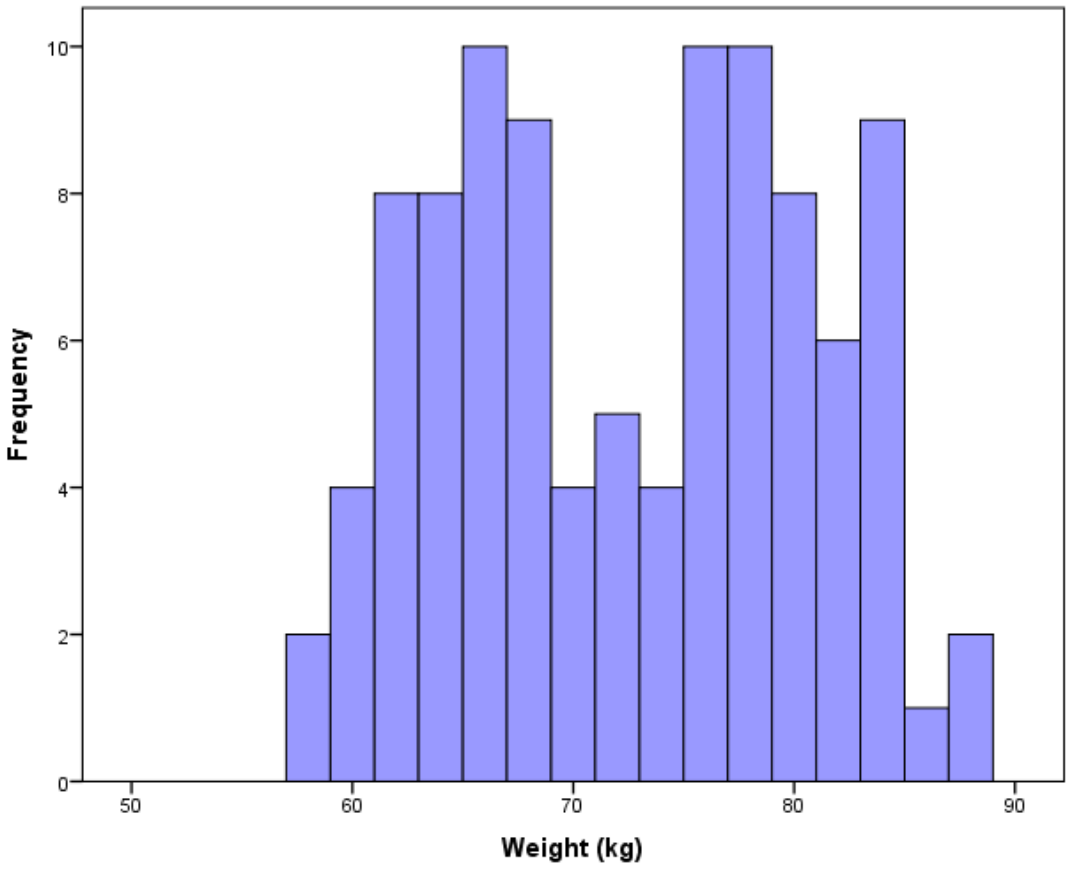
The mean Weight (kg) among the subjects was 72.43 (± 8.06) kg ranging from 58 to 88 kg.

Table 4. Weight (kg)

Weight (kg)	
Mean	72.43
Median	73

Std. Deviation	8.06
Range	30
Minimum	58
Maximum	88

Figure 19. Weight (kg)



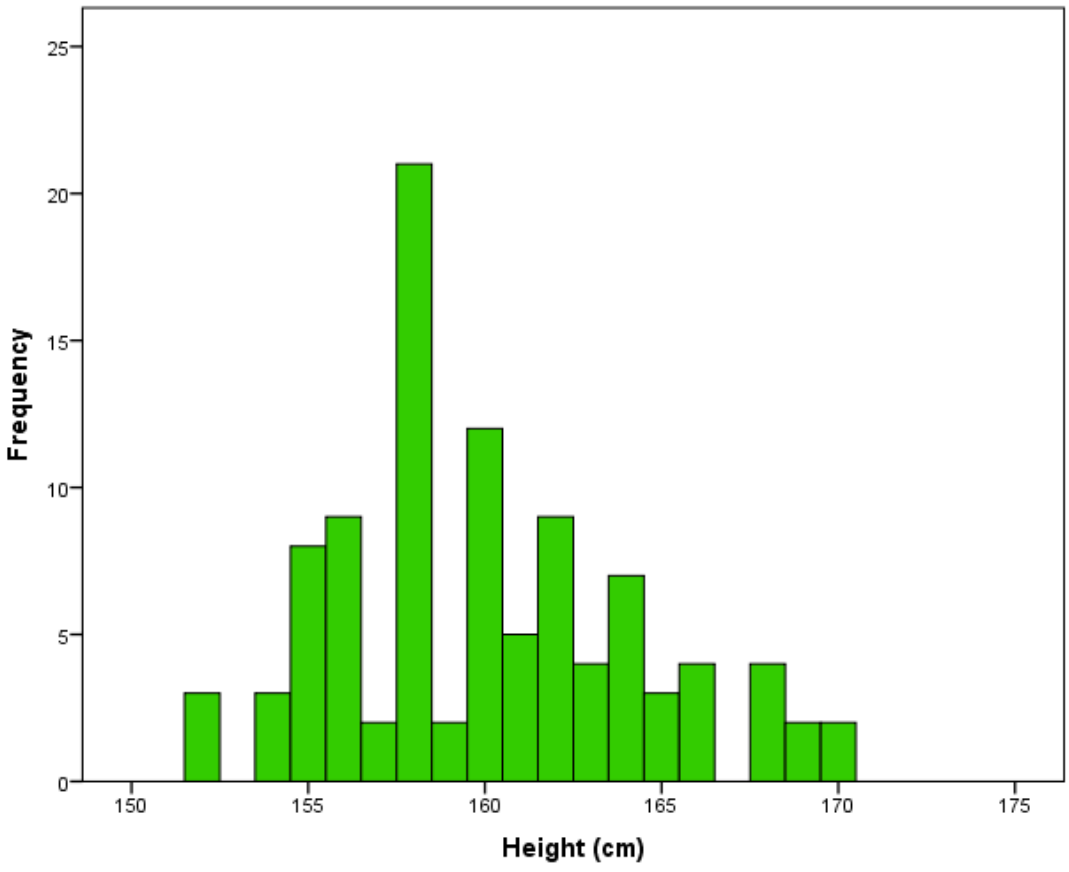
IV. Height (cm)

The mean Height (cm) among the subjects was 160.04 (± 4.23) cm ranging from 152 to 170 cm.

Table 5. Height (cm)

Height (cm)	
Mean	160.04
Median	160
Std. Deviation	4.23
Range	18
Minimum	152
Maximum	170

Figure 20. Height (cm)



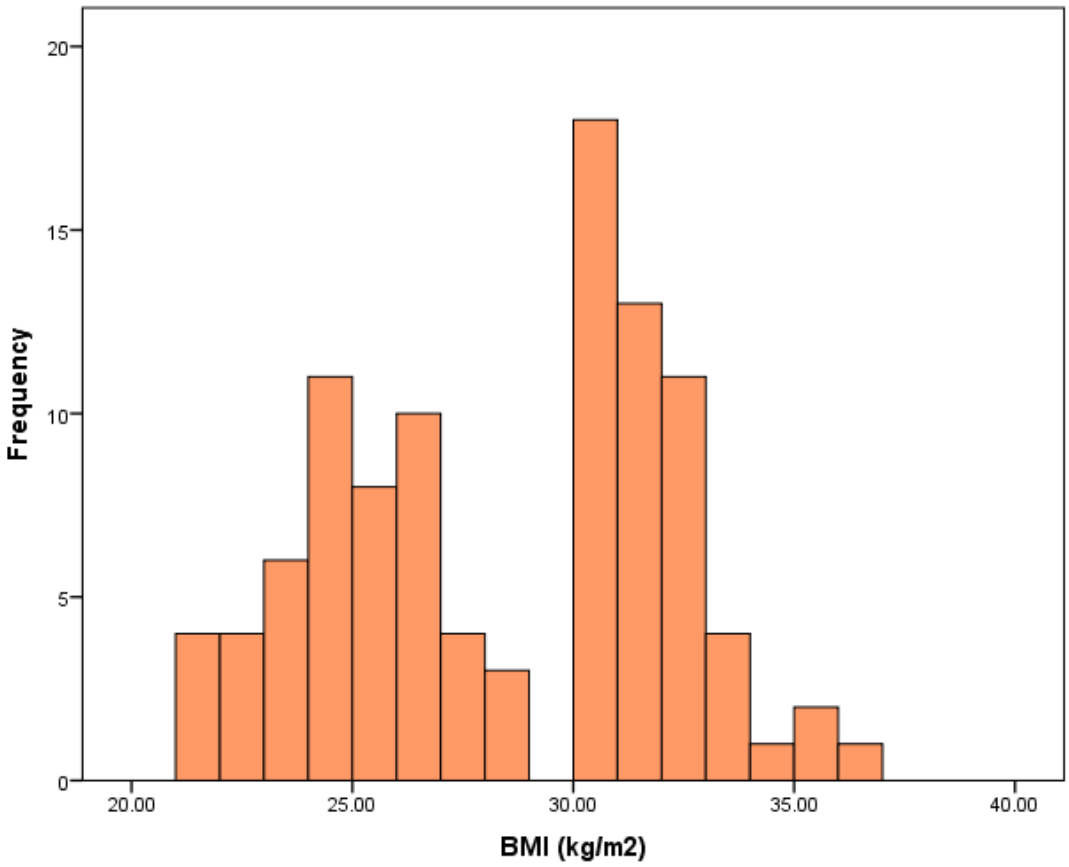
V. BMI (kg/m2)

The mean BMI (kg/m2) among the subjects was 28.4 (± 3.79) kg/m2 ranging from 21.3 to 36.35 kg/m2

Table 6. BMI (kg/m2)

BMI (kg/m2)	
Mean	28.40
Median	29.17
Std. Deviation	3.79
Range	15.05
Minimum	21.3
Maximum	36.35

Figure 21. BMI (kg/m2)



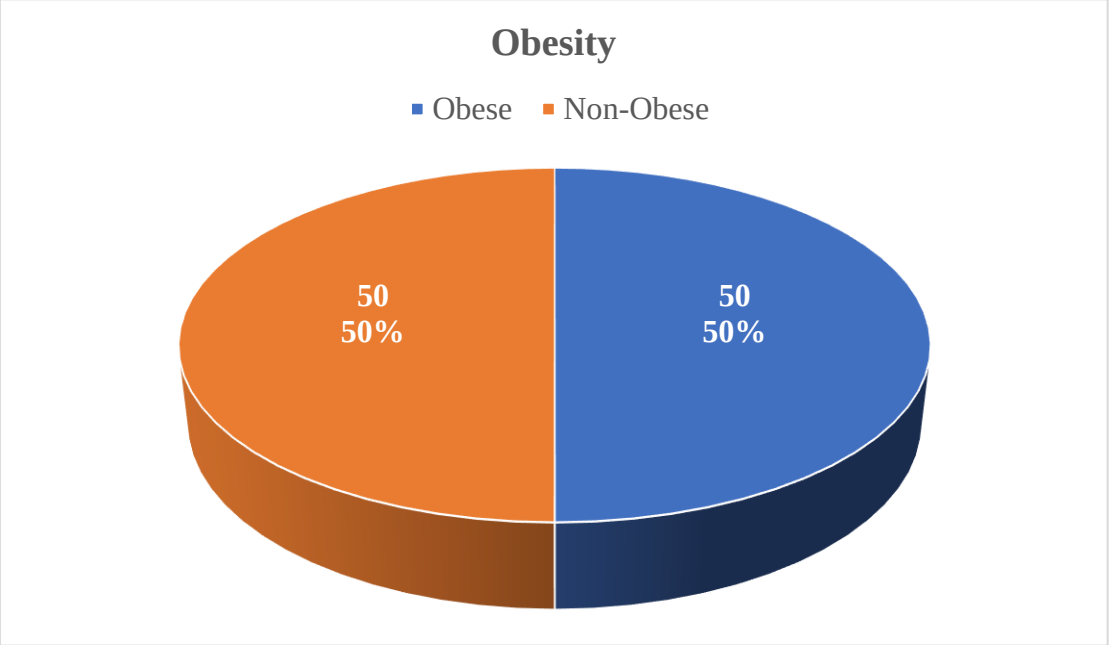
VI. Obesity

Among the subjects, 50 (50%) were Obese and 50 (50%) were Non-Obese

Table 7. Obesity

Obesity	Frequency	Percent
Obese	50	50.00
Non-Obese	50	50.00
Total	100	100.00

Figure 22. Obesity



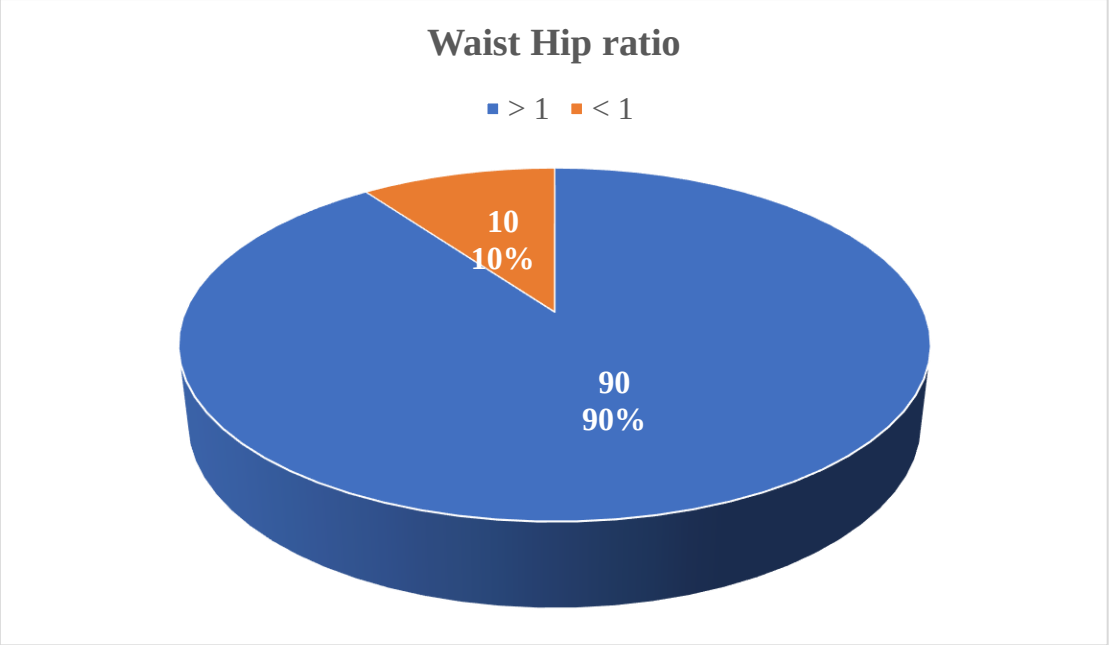
VII. Waist Hip ratio

Among the subjects, 90 (90%) had > 1 and 10 (10%) had < 1 Waist Hip ratio

Table 8. Waist Hip ratio

Waist Hip ratio	Frequency	Percent
> 1	90	90.00
< 1	10	10.00
Total	100	100.00

Figure 23. Waist Hip ratio



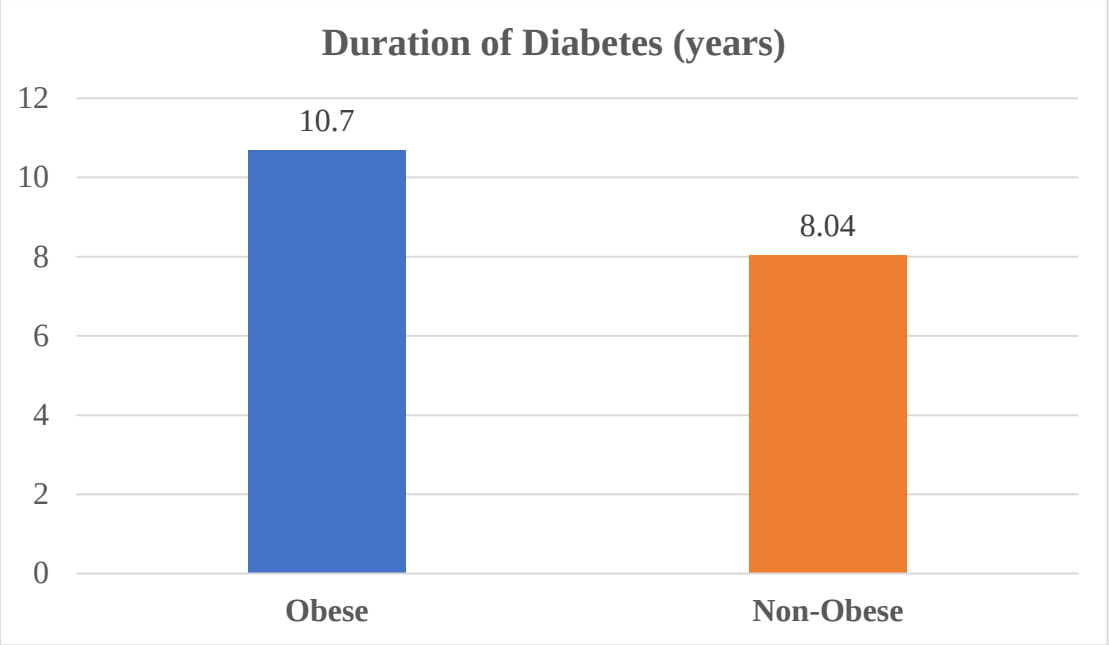
VIII. Duration of Diabetes (years) with Obesity

The mean Duration of Diabetes (years) among Obese was 10.7 (± 3.99) which is higher by 2.66 and statistically significant compared to 8.04 (± 3.85) in Non-Obese

Table 9. Duration of Diabetes (years) with Obesity

	Obesity	N	Mean	Std. dev.	Mean diff.	p value by 't' test
Duration of Diabetes (years)	**Obese**	50	10.70	3.99	2.660	0.001
	Non-Obese	50	8.04	3.85		

Figure 24. Duration of Diabetes (years) with Obesity



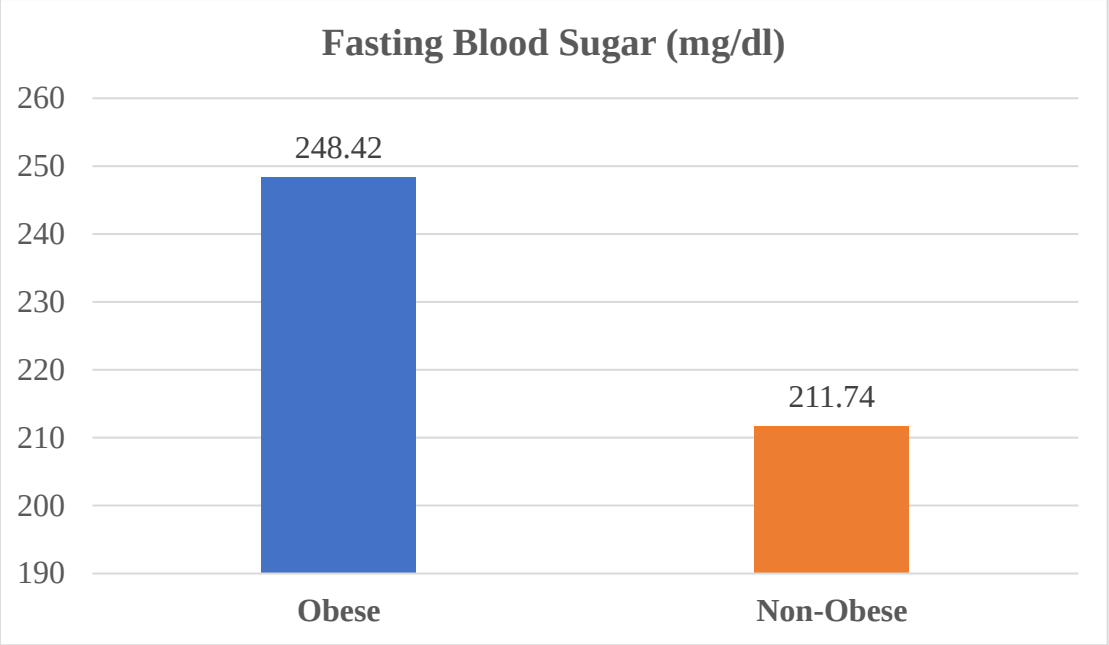
IX. Fasting Blood Sugar (mg/dl) with Obesity

The mean Fasting Blood Sugar (mg/dl) among Obese was 248.42 (± 29.83) which is higher by 36.68 and statistically significant compared to 211.74 (± 19.77) in Non-Obese

Table 10. Fasting Blood Sugar (mg/dl) with Obesity

	Obesity	N	Mean	Std. dev.	Mean diff.	p value by 't' test
Fasting Blood Sugar (mg/dl)	**Obese**	50	248.42	29.83	36.680	0.001
	Non-Obese	50	211.74	19.77		

Figure 25. Fasting Blood Sugar (mg/dl) with Obesity



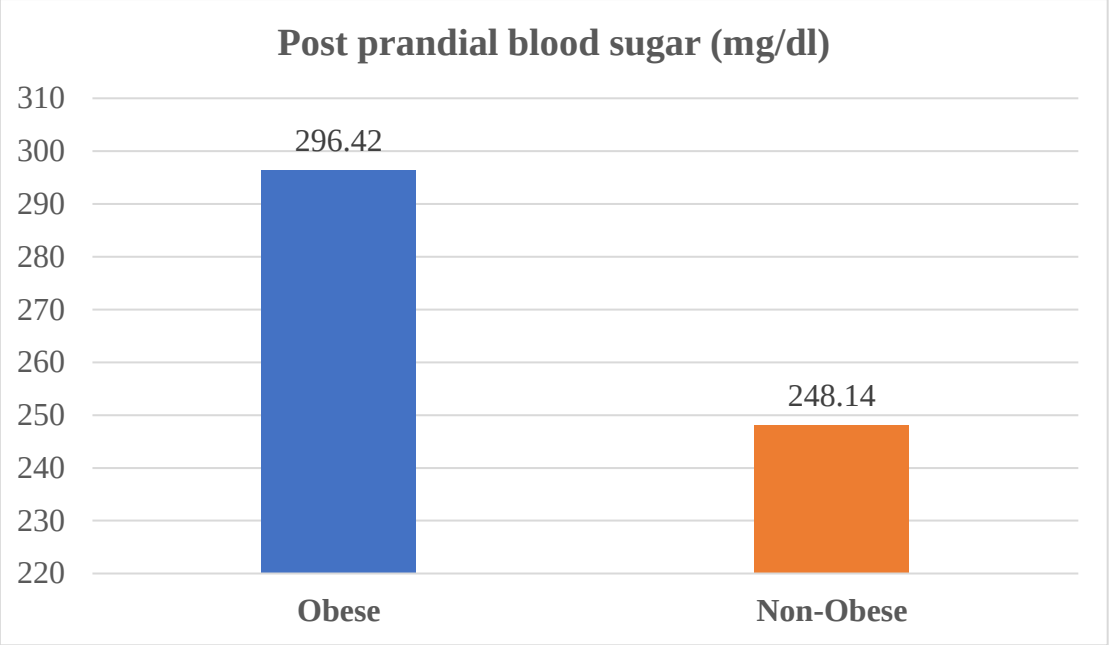
X. Post prandial blood sugar (mg/dl) with Obesity

The mean Post prandial blood sugar (mg/dl) among Obese was 296.42 (± 41.52) which is higher by 48.28 and statistically significant compared to 248.14 (± 21.36) in Non-Obese.

Table 11. Post prandial blood sugar (mg/dl) with Obesity

	Obesity	N	Mean	Std. dev.	Mean diff.	p value by 't' test
Post prandial blood sugar (mg/dl)	**Obese**	50	296.42	41.52	48.280	0.001
	Non-Obese	50	248.14	21.36		

Figure 26. Post prandial blood sugar (mg/dl) with Obesity



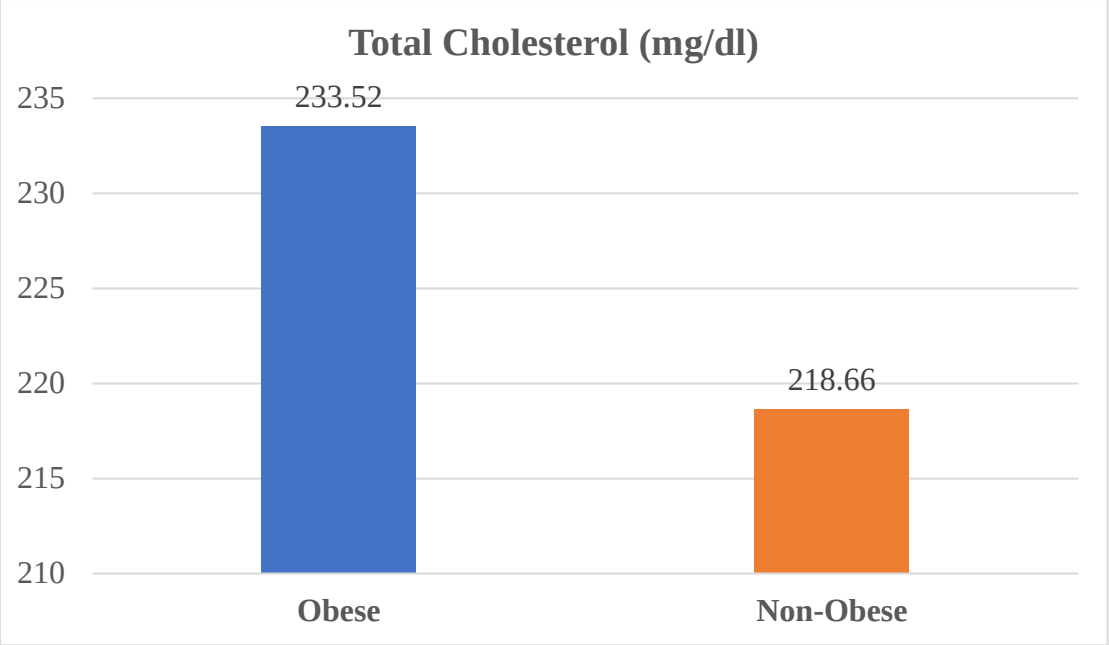
XI. Total Cholesterol (mg/dl) with Obesity

The mean Total Cholesterol (mg/dl) among Obese was 233.52 (± 16) which is higher by 14.86 and statistically significant compared to 218.66 (± 12.84) in Non-Obese

Table 12. Total Cholesterol (mg/dl) with Obesity

	Obesity	N	Mean	Std. dev.	Mean diff.	p value by 't' test
Total Cholesterol (mg/dl)	**Obese**	50	233.52	16.00	14.860	0.001
	Non-Obese	50	218.66	12.84		

Figure 27. Total Cholesterol (mg/dl) with Obesity



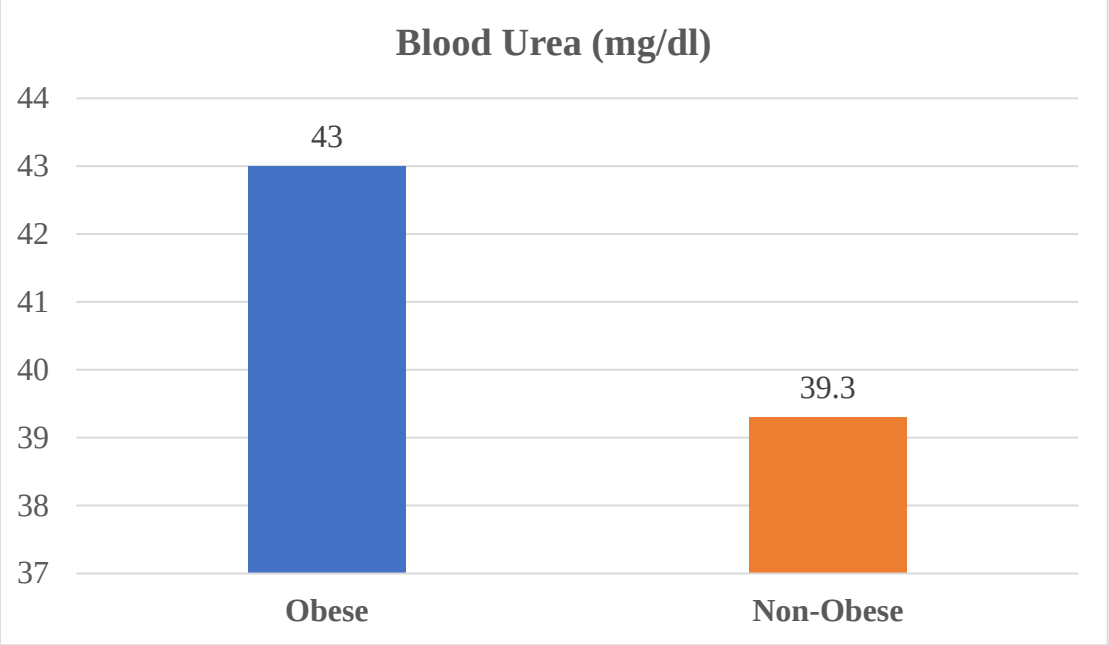
XII. Blood Urea (mg/dl) with Obesity

The mean Blood Urea (mg/dl) among Obese was 43 (± 5.86) which is higher by 3.7 and statistically significant compared to 39.3 (± 6.73) in Non-Obese

Table 13. Blood Urea (mg/dl) with Obesity

	Obesity	N	Mean	Std. dev.	Mean diff.	p value by 't' test
Blood Urea (mg/dl)	**Obese**	50	43.00	5.86	3.700	0.004
	Non-Obese	50	39.30	6.73		

Figure 28. Blood Urea (mg/dl) with Obesity



XIII. Comparision of Urine sugar with the Obesity

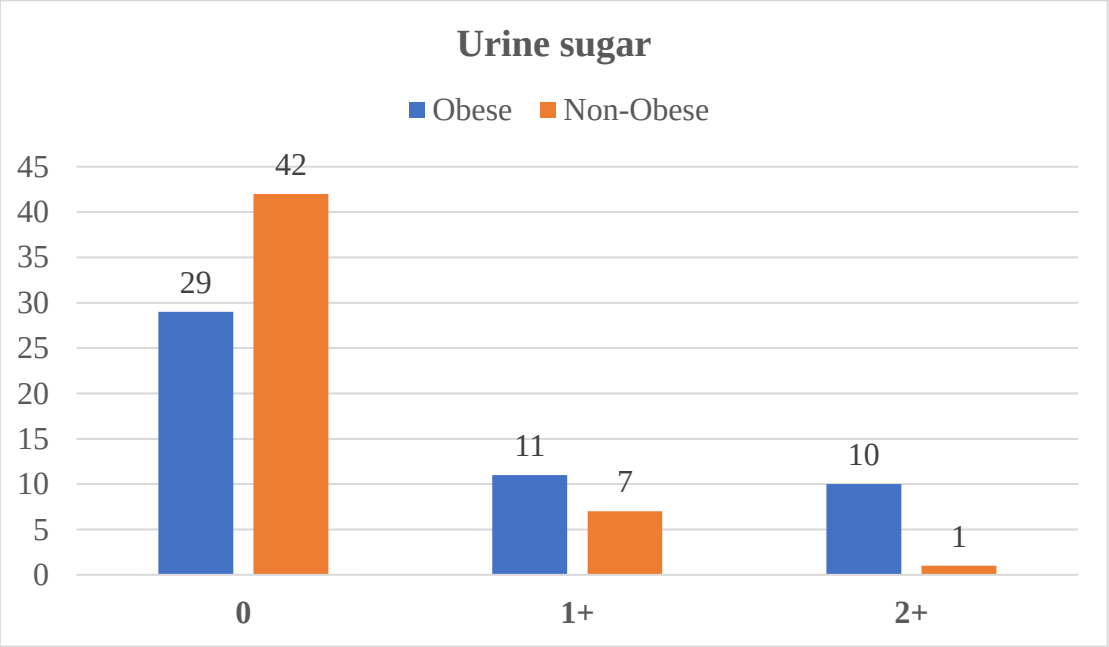
Comparing the Urine sugar with Obesity distribution, Obese group had higher proportion of nil urine sugar with 58% followed by 1+ with 22% and least in 2+ with 20% compared to Non-Obese group which had higher proportion of nil urine sugar with 84% followed by 1+ with 14% and least in 2+ with 2%. The difference in Urine sugar distribution between Obese and Non-Obese was statistically significant ($p < 0.05$)

Table 14. Comparision of Urine sugar with the Obesity

Urine sugar	Obesity		Total	Chi sq. p value
	Obese	Non-Obese		
0	29 (58%)	42 (84%)	71 (71%)	0.005
1+	11 (22%)	7 (14%)	18 (18%)	

2+	10 (20%)	1 (2%)	11 (11%)	
Total	50 (100%)	50 (100%)	100 (100%)	

Figure 29. Comparision of Urine sugar with the Obesity



XIV. Serum Vitamin D (ng/ml) with Obesity

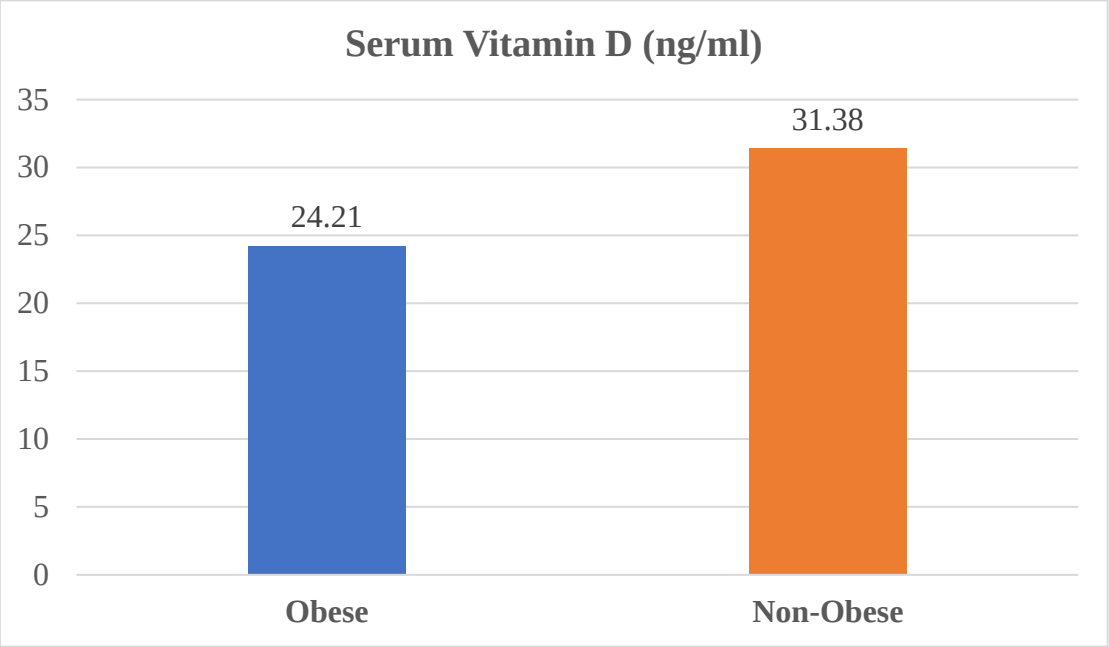
The mean Serum Vitamin D (ng/ml) among Obese was 24.21 (± 3.83) which is lower by 7.17 and statistically significant compared to 31.38 (± 2.53) in Non-Obese

Table 15. Serum Vitamin D (ng/ml) with Obesity

	Obesity	**N**	**Mean**	**Std. dev.**	**Mean diff.**	**p value by 't' test**

Serum Vitamin D (ng/ml)	**Obese**	50	24.21	3.83	7.172	0.001
	Non-Obese	50	31.38	2.53		

Figure 30. Serum Vitamin D (ng/ml) with Obesity



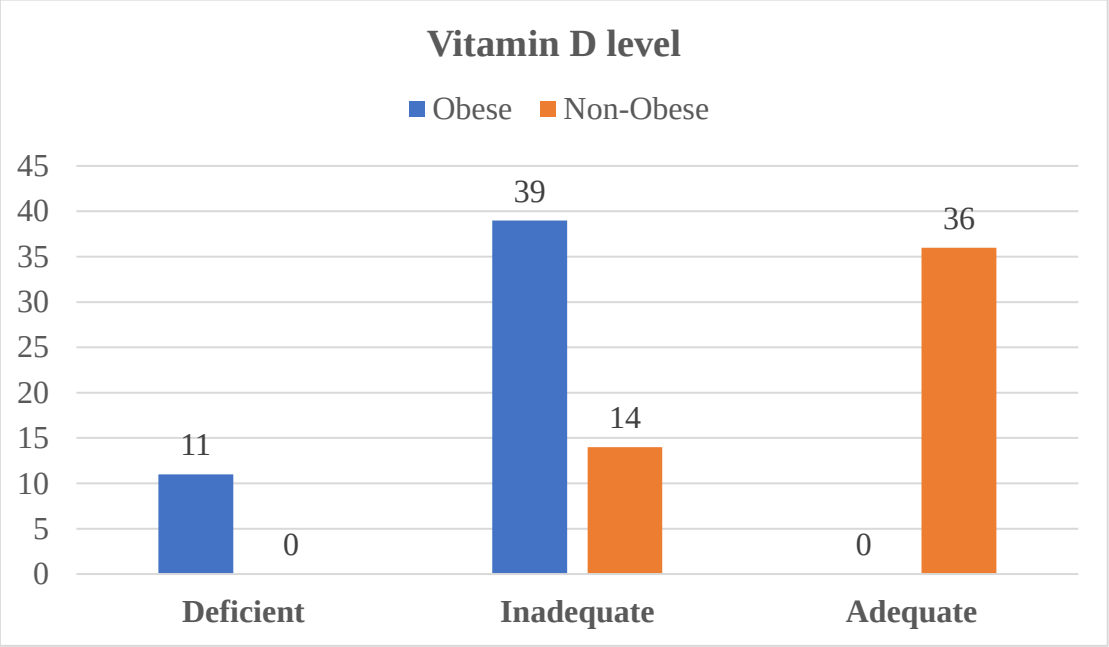
XV. Comparision of Vitamin D level with the Obesity

Comparing the Vitamin D level with Obesity distribution, Obese group had higher proportion of Inadequate Vitamin D level with 78% followed by Deficient Vitamin D level with 22% and least in Adequate Vitamin D level with 0% compared to Non-Obese group which had higher proportion of Adequate Vitamin D level with 72% followed by Inadequate Vitamin D level with 28% and least in Deficient Vitamin D level with 0%. The difference in Vitamin D level distribution between Obese and Non-Obese was statistically significant ($p < 0.05$)

Table 16. Comparision of Vitamin D level with the Obesity

Vitamin D level	Obesity		Total	Chi sq. p value
	Obese	Non-Obese		
Deficient	11 (22%)	0 (0%)	11 (11%)	0.001
Inadequate	39 (78%)	14 (28%)	53 (53%)	
Adequate	0 (0%)	36 (72%)	36 (36%)	
Total	50 (100%)	50 (100%)	100 (100%)	

Figure 31. Comparision of Vitamin D level with the Obesity



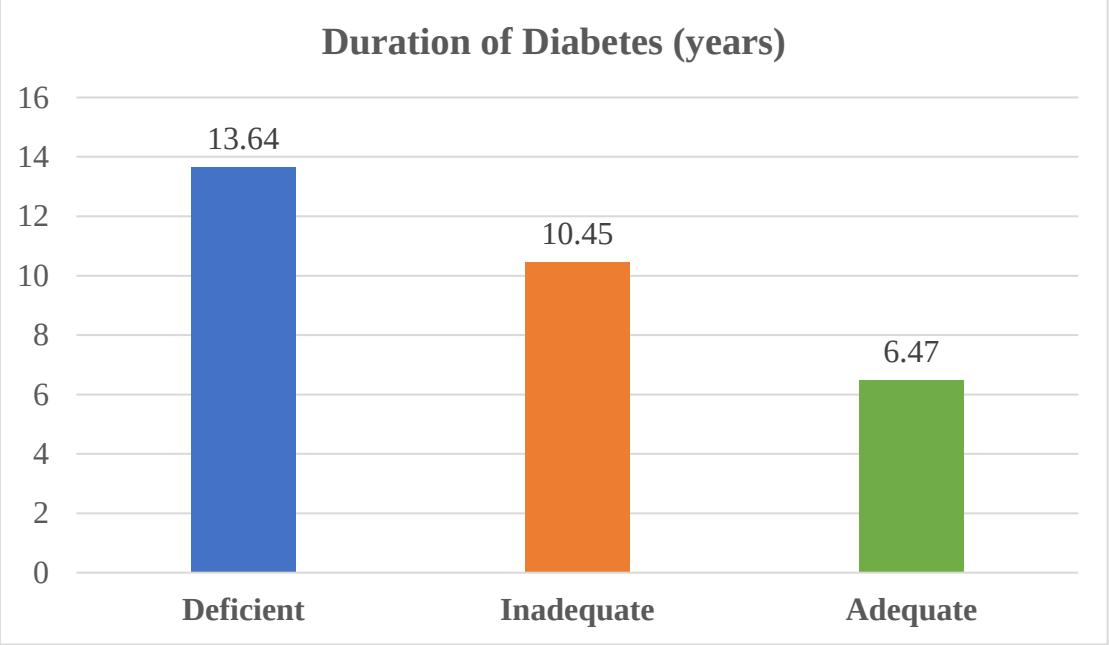
XVI. Duration of Diabetes (years) with Vitamin D level

The mean Duration of Diabetes (years) among Deficient Vitamin D level was 13.64 which is higher than mean among Inadequate Vitamin D level which was 10.45 followed by Adequate Vitamin D level with a mean of 6.47 and the difference was statistically significant ($p < 0.05$).

Table 17. Duration of Diabetes (years) with Vitamin D level

	Vitamin D level	N	Mean	Std. Deviation	ANOVA p value
Duration of Diabetes (years)	**Deficient**	11	13.64	4.70	0.001
	Inadequate	53	10.45	3.49	
	Adequate	36	6.47	2.76	

Figure 32. Duration of Diabetes (years) with Vitamin D level



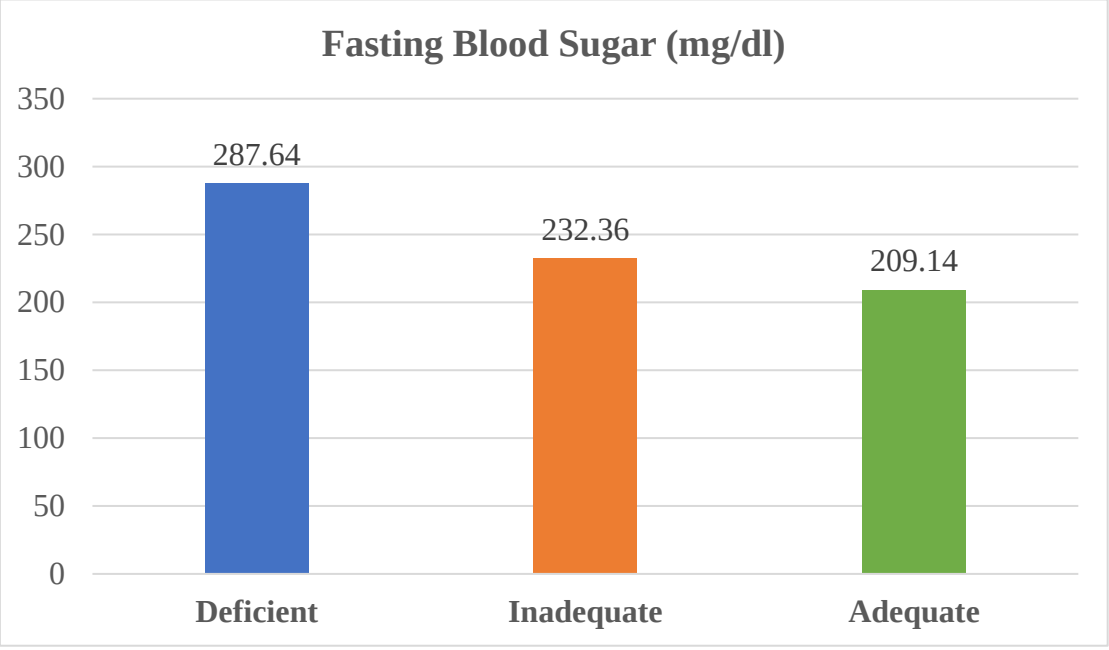
XVII. Fasting Blood Sugar (mg/dl) with Vitamin D level

The mean Fasting Blood Sugar (mg/dl) among Deficient Vitamin D level was 287.64 which is higher than mean among Inadequate Vitamin D level which was 232.36 followed by Adequate Vitamin D level with a mean of 209.14 and the difference was statistically significant ($p < 0.05$).

Table 18. Fasting Blood Sugar (mg/dl) with Vitamin D level

	Vitamin D level	N	Mean	Std. Deviation	ANOVA p value
Fasting Blood Sugar (mg/dl)	**Deficient**	11	287.64	16.94	0.001
	Inadequate	53	232.36	22.11	
	Adequate	36	209.14	21.10	

Figure 33. Fasting Blood Sugar (mg/dl) with Vitamin D level



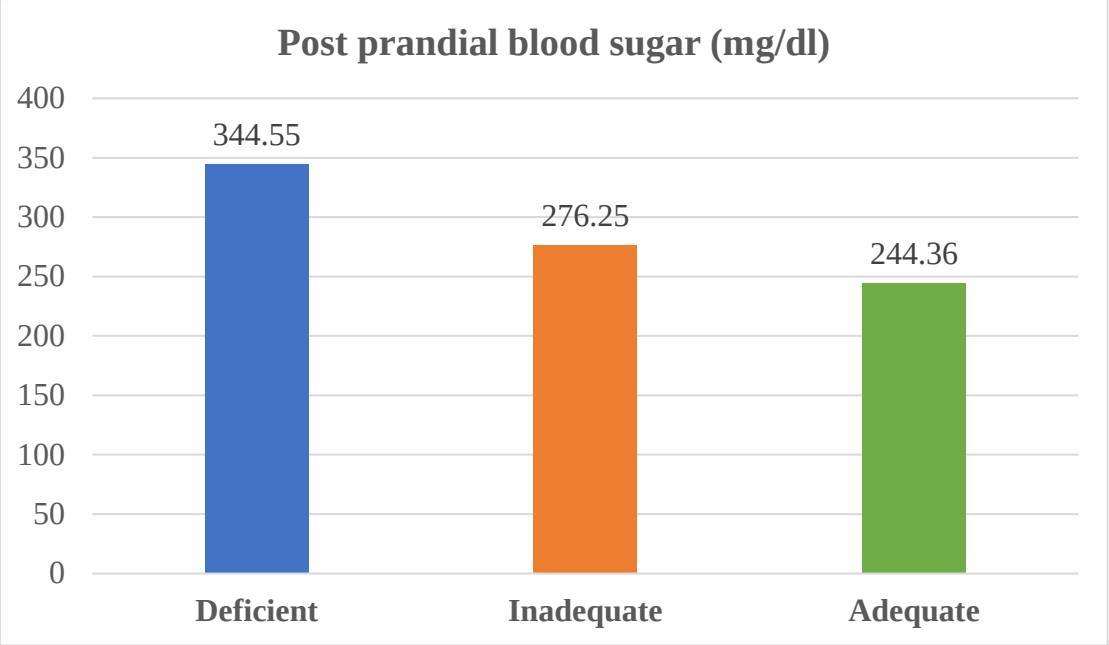
XVIII. Post prandial blood sugar (mg/dl) with Vitamin D level

The mean Post prandial blood sugar (mg/dl) among Deficient Vitamin D level was 344.55 which is higher than mean among Inadequate Vitamin D level which was 276.25 followed by Adequate Vitamin D level with a mean of 244.36 and the difference was statistically significant ($p < 0.05$).

Table 19. Post prandial blood sugar (mg/dl) with Vitamin D level

	Vitamin D level	**N**	**Mean**	**Std. Deviation**	**ANOVA p value**
Post prandial blood sugar (mg/dl)	**Deficient**	11	344.55	38.43	0.001
	Inadequate	53	276.25	30.36	
	Adequate	36	244.36	21.45	

Figure 34. Post prandial blood sugar (mg/dl) with Vitamin D level



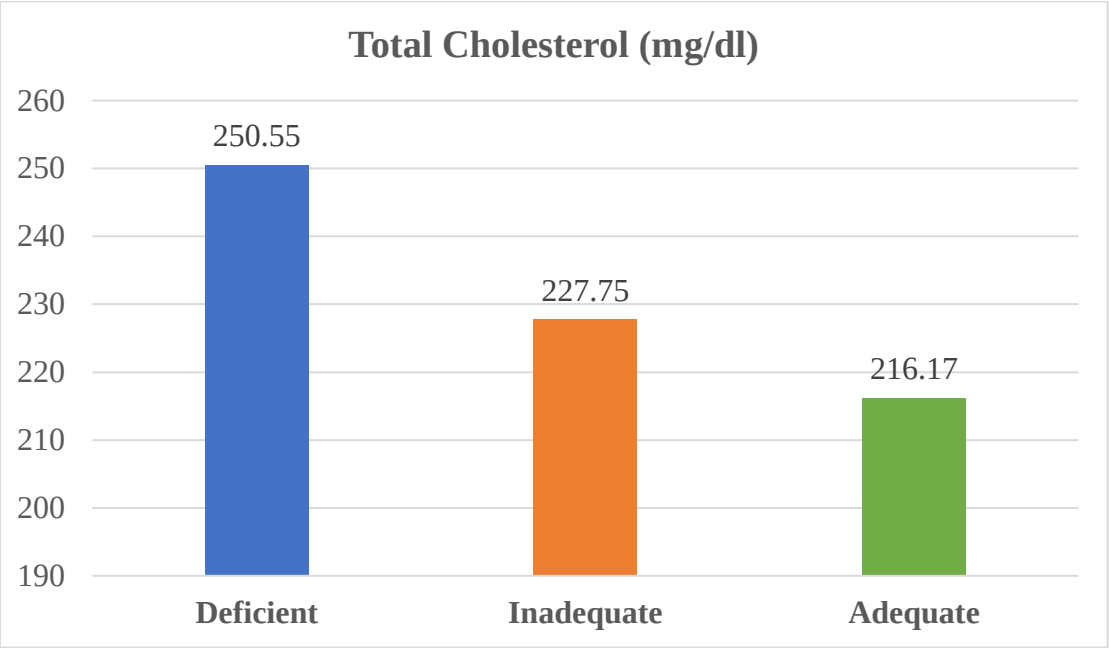
XIX. Total Cholesterol (mg/dl) with Vitamin D level

The mean Total Cholesterol (mg/dl) among Deficient Vitamin D level was 250.55 which is higher than mean among Inadequate Vitamin D level which was 227.75 followed by Adequate Vitamin D level with a mean of 216.17 and the difference was statistically significant ($p < 0.05$).

Table 20. Total Cholesterol (mg/dl) with Vitamin D level

	Vitamin D level	N	Mean	Std. Deviation	ANOVA p value
Total Cholesterol (mg/dl)	**Deficient**	11	250.55	8.72	0.001
	Inadequate	53	227.75	13.81	
	Adequate	36	216.17	12.19	

Figure 35. Total Cholesterol (mg/dl) with Vitamin D level



XX. Blood Urea (mg/dl) with Vitamin D level

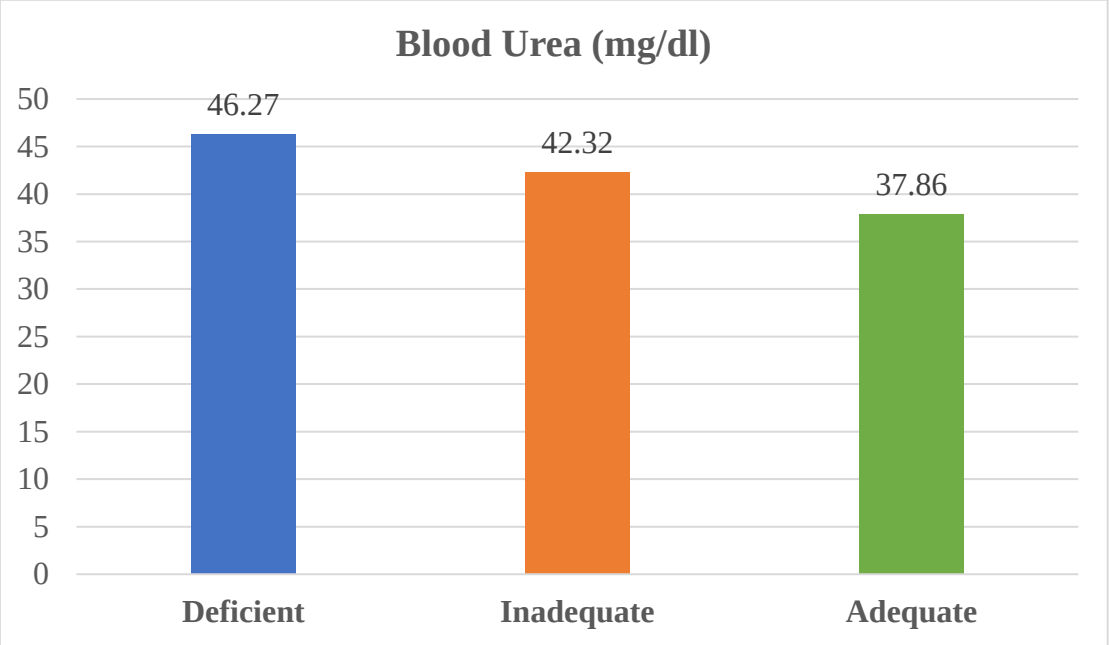
The mean Blood Urea (mg/dl) among Deficient Vitamin D level was 46.27 which is higher than mean among Inadequate Vitamin D level which was 42.32 followed by Adequate Vitamin D level with a mean of 37.86 and the difference was statistically significant ($p < 0.05$).

Table 21. Blood Urea (mg/dl) with Vitamin D level

	Vitamin D level	N	Mean	Std. Deviation	ANOVA p value
Blood Urea (mg/dl)	**Deficient**	11	46.27	6.78	0.001
	Inadequate	53	42.32	4.91	

	Adequate	36	37.86	7.13	

Figure 36. Blood Urea (mg/dl) with Vitamin D level



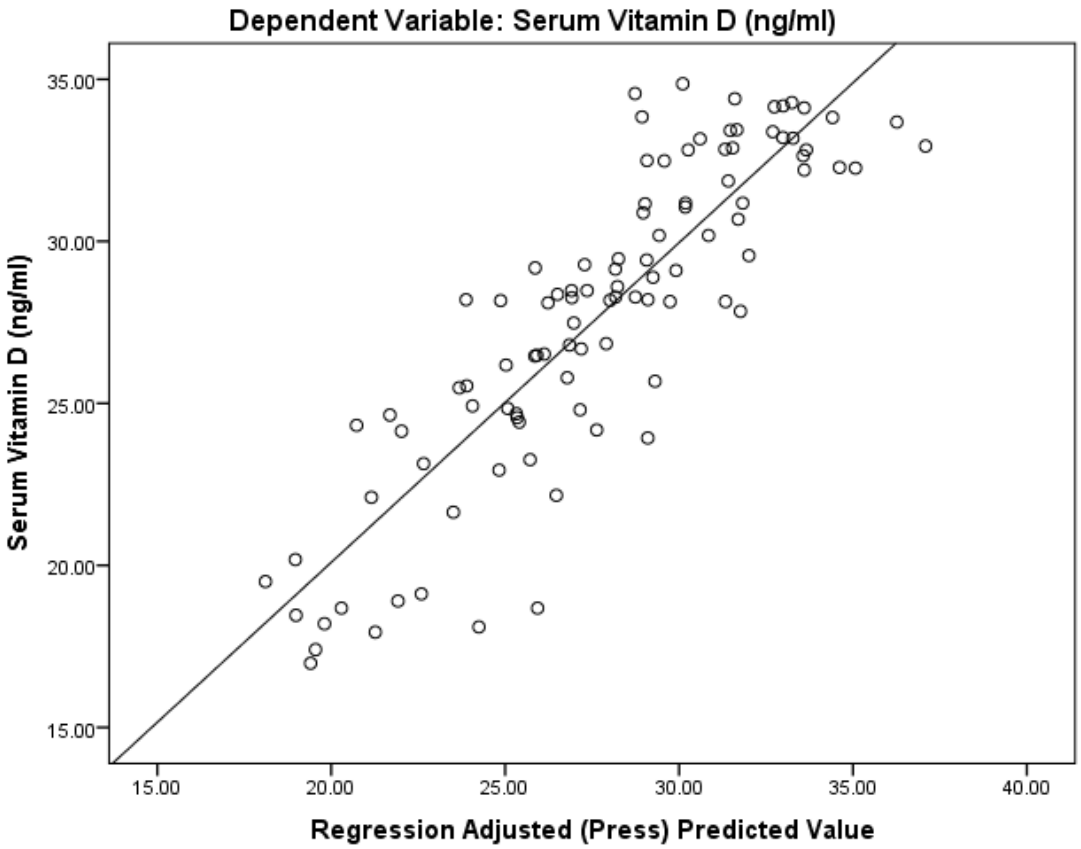
XXI. Linear Regression for predicting Serum Vitamin D (ng/ml)

The model with these predictors explains 78.53% variability (predictability) of Serum Vitamin D (ng/ml). Serum Vitamin D (ng/ml) decreases -0.03 times for each unit increase in Post prandial blood sugar (mg/dl) and is statistically significant. Serum Vitamin D (ng/ml) decreases -0.04 times for each unit increase in Total Cholesterol (mg/dl) and is statistically significant. Serum Vitamin D (ng/ml) decreases -0.09 times for each unit increase in Blood Urea (mg/dl) and is statistically significant. Serum Vitamin D (ng/ml) decreases -0.3 times for each unit increase in Duration of Diabetes (years) and is statistically significant. Serum Vitamin D (ng/ml) decreases -0.46 times for each unit increase in BMI (kg/m2) and is statistically significant.

Table 22. Linear Regression for predicting Serum Vitamin D (ng/ml)

Predictors for Serum Vitamin D (ng/ml)	Adjusted B (95% C.I.)	p value
Post prandial blood sugar (mg/dl)	-0.033 (-0.051 to -0.015)	0.001
Total Cholesterol (mg/dl)	-0.044 (-0.084 to 0.005)	0.029
Blood Urea (mg/dl)	-0.086 (-0.171 to 0.0004)	0.049
Duration of Diabetes (years)	-0.302 (-0.45 to -0.15)	0.001
BMI (kg/m2)	-0.462 (-0.62 to -0.3)	0.001

Figure 37. Linear Regression for predicting Serum Vitamin D (ng/ml)



8 DISCUSSION

The main objective of the study is to find out the correlation of Vitamin D levels and diabetic profile among patients admitted in inpatient wards , Department of General Medicine, Thanjavur Medical College.

The mean Age (years) among the subjects was 49.94 (± 6.69) years ranging from 38 to 64 years. Among the subjects, 51 (51%) were in 41 - 50 years, 37 (37%) were in 51 - 60 years, 6 (6%) were in > 60 years and 6 (6%) were in < 40 years. Among the subjects, 50 (50%) were Obese and 50 (50%) were Non-Obese. Among the subjects, 90 (90%) had > 1 and 10 (10%) had < 1 Waist Hip ratio.

The mean Duration of Diabetes (years) among Obese was 10.7 (± 3.99) which is higher by 2.66 and statistically significant compared to 8.04 (± 3.85) in Non-Obese. The mean Fasting Blood Sugar (mg/dl) among Obese was 248.42 (± 29.83) which is higher by 36.68 and statistically significant compared to 211.74 (± 19.77) in Non-Obese. The mean Post prandial blood sugar (mg/dl) among Obese was 296.42 (± 41.52) which is higher by 48.28 and statistically significant compared to 248.14 (± 21.36) in Non-Obese. The mean Total Cholesterol (mg/dl) among Obese was 233.52 (± 16) which is higher by 14.86 and statistically significant compared to 218.66 (± 12.84) in Non-Obese. The mean Blood Urea (mg/dl) among Obese was 43 (± 5.86) which is higher by 3.7 and statistically significant compared to 39.3 (± 6.73) in Non-Obese. According to Kamath et al[60] 86.6% of diabetic patients had waist circumference of > 80 cm and Chamukuttan et al[61] demonstrates that Asian Indians have higher upper body adiposity and higher visceral fat for a given BMI when compared with the Western population. BMI for an urban Indian is 23 kg/m2, and cut off values for Waist Circumference were 80 cm for women, and for Waist-Hip Ratio were

0.81 for women. Mamatha B Patil and Raghav [57] showed that the mean age of study group: 48.47±9.56 years. Mean duration of diabetes in the study population: ± SD: 5.10±4.36 years mean BMI was 24.97±4.16.Mean waist circumference: 98.93±12.18 cm, Mean Waist-Hip ratio: 0.98±0.08. Mean FBS: 202.73±81.73 mg/dl, Mean PPBS: 280.99±94.14 mg/dl, Mean HbA1C: 9.33±1.83 %. Almost all diabetic females (92.5%) have Vitamin-D deficiency. Mean Vitamin-D levels: 16.19±8.97 ng/ml. Duration of diabetes, Poor Glycaemic control and increased BMI had significant role in causing Vitamin-D deficiency.

Comparing the Urine sugar with Obesity distribution, Obese group had higher proportion of nil urine sugar with 58% followed by 1+ with 22% and least in 2+ with 20% compared to Non-Obese group which had higher proportion of nil urine sugar with 84% followed by 1+ with 14% and least in 2+ with 2%. The difference in Urine sugar distribution between Obese and Non-Obese was statistically significant ($p < 0.05$)

The mean Serum Vitamin D (ng/ml) among Obese was 24.21 (± 3.83) which is lower by 7.17 and statistically significant compared to 31.38 (± 2.53) in Non-Obese. Comparing the Vitamin D level with Obesity distribution, Obese group had higher proportion of Inadequate Vitamin D level with 78% followed by Deficient Vitamin D level with 22% and least in Adequate Vitamin D level with 0% compared to Non-Obese group which had higher proportion of Adequate Vitamin D level with 72% followed by Inadequate Vitamin D level with 28% and least in Deficient Vitamin D level with 0%. The difference in Vitamin D level distribution between Obese and Non-Obese was statistically significant ($p < 0.05$). The mean Duration of Diabetes (years) among Deficient Vitamin D level was 13.64 which is higher than mean among Inadequate Vitamin D level which was 10.45 followed by Adequate Vitamin D level with a mean of 6.47 and the difference was statistically significant ($p < 0.05$).

The mean Fasting Blood Sugar (mg/dl) among Deficient Vitamin D level was 287.64 which is higher than mean among Inadequate Vitamin D level which was 232.36 followed by Adequate Vitamin D level with a mean of 209.14 and the difference was statistically significant ($p < 0.05$). The mean Post prandial blood sugar (mg/dl) among Deficient Vitamin D level was 344.55 which is higher than mean among Inadequate Vitamin D level which was 276.25 followed by Adequate Vitamin D level with a mean of 244.36 and the difference was statistically significant ($p < 0.05$). The mean Total Cholesterol (mg/dl) among Deficient Vitamin D level was 250.55 which is higher than mean among Inadequate Vitamin D level which was 227.75 followed by Adequate Vitamin D level with a mean of 216.17 and the difference was statistically significant ($p < 0.05$). The mean Blood Urea (mg/dl) among Deficient Vitamin D level was 46.27 which is higher than mean among Inadequate Vitamin D level which was 42.32 followed by Adequate Vitamin D level with a mean of 37.86 and the difference was statistically significant ($p < 0.05$). Anyanwu et al[55] found that the mean serum vitamin D level in T2DM subjects with vitamin D deficiency was 9.2 ± 1.1 ng/dl and 21.5 ± 0.7 ng/dl in the sufficient group ($t = 11.9$, $p = 0.0001$). The mean HbA1c and Fasting plasma glucose were higher in the vitamin D deficient group compared to the sufficient group (7.5 ± 1.9% and 148 ± 60.9 mg/dl vs. 6.8 ± 1.6% and 134 ± 43.5 mg/dl respectively, p NS). The proportion of subjects with good glycaemic control (HbA1C f 7.0%) was significantly higher in the vitamin D sufficient group 19 (73.1%) compared to the vitamin D deficient group, 33 (45.8%), $Z = -2.39$, $p = 0.01$).

The linear regression model with these predictors explains 78.53% variability (predictability) of Serum Vitamin D (ng/ml). Serum Vitamin D (ng/ml) decreases -0.03 times for each unit increase in Post prandial blood sugar (mg/dl) and is statistically significant. Serum Vitamin D (ng/ml) decreases -0.04 times for each unit increase in Total Cholesterol (mg/dl) and is statistically significant. Serum Vitamin D (ng/ml) decreases -0.09

times for each unit increase in Blood Urea (mg/dl) and is statistically significant. Serum Vitamin D (ng/ml) decreases -0.3 times for each unit increase in Duration of Diabetes (years) and is statistically significant. Serum Vitamin D (ng/ml) decreases -0.46 times for each unit increase in BMI (kg/m2) and is statistically significant. Mauss et al[53] showed that severe vitamin D deficiency (<10 ng/ml) was associated with increasing FPG ($p \leq 0.01$) and HbA1c ($p \leq 0.001$) values in adjusted linear regression models. Mamatha et al[57] showed that glycaemic status has a significant association with Vitamin D levels. This finding could infer that glycaemic control in a given diabetic patients is one of the most important factor in determining Vitamin-D levels. Better the glycaemic control, the more the Vitamin-D level in a given patient. Or, it could be also interpreted that the patients having poor glycaemic control would need the addition of Vitamin-D supplements due to more probability of Vitamin-D deficiency in them.

Kabadi and Patil[51] showed that mean HbA1c was 10.96 ± 1.78, 9.66 ± 1.37, and 7.05 ± 0.65, among patients having their vitamin D levels ranging <20, 20–30, and >30 ng/dL, respectively, showing p value < 0.001. Mean BMI was 29.90 ± 2.18, of 111 of them who had vitamin D levels <30 ng/dL, 74 of them had BMI (18.5–22.9), 20 had BMI (23–24.9), and 17 of them had BMI >25. Vitamin D levels can be independent risk factors for the development of DM and obesity and hence must be treated promptly. Zakhary et al[52] showed that Vitamin D had a clinical positive impact on glucose level, particularly on hemoglobin A1c (HbA1c) reduction, alleviation of diabetic neuropathy and nephropathy symptoms, and hyperglycaemia induced-oxidative stress on the retinal cells. Salih et al [37] showed that low vitamin D levels were detected in 110 (71%) of cases and 63 (40.6%) of controls. There was a significant difference in vitamin D levels among cases and controls ($p < 0.001$), vitamin D level was lower among females compared to males, $p < 0.001$ and those living in urban areas compared to rural areas, $p < 0.001$, BMI and dyslipidaemia had a significant effect on

vitamin D levels among diabetics, p values 0.002 and < 0.001 respectively. The serum 25(OH)-D level was significantly lower in patients with poor glycaemic control compared to those with good glycaemic control and in patients with a diabetes duration greater than 5 years, p values < 0.001 and 0.002 respectively. Ahmed et al [53] found that vitamin D3 levels were lower in diabetics, particularly in females ($p < 0.001$), and also associated with retinopathy ($p < 0.014$). Neither vitamin D2 nor vitamin D3 were associated with neuropathy. Zhao et al [54] found that HbA1c levels in the vitamin D deficiency group were significantly higher than those in the no vitamin D deficiency group for all subjects. Santhosh kumar et al[56] found hypovitaminosis D in more than half of the patients with type 2 diabetes. suggesting a potential for vitamin D supplementation in type 2 DM patients. Liu et al[58] showed that serum vitamin D was significantly lower in children with T1DM than in healthy controls. Ifigenia Kostoglou-Athanassiou et al (2013)[59] showed that 25(OH)D3 levels were lower in the diabetes mellitus type 2 patients than in the control group, being 19.26 ± 0.95 ng/ml and 25.49 ± 1.02 ng/ml, in the patient and control groups, respectively. 25(OH)D3 levels were found to be inversely associated with HbA1c when the patient and control groups were analysed together ($p < 0.001$, $r2 = 0.086$). The potential mechanisms of vitamin D's beneficial effects in type 2 diabetes include: i) improved b-cell function via direct vitamin D action or by increase in intracellular ionised calcium, which would result in enhanced insulin release; (ii) increased insulin sensitivity related to insulin receptor expression or via calcium dependent pathways in target cells leading to increase in the utilisation of glucose; and (iii) inhibition of b-cell apoptosis due to VDR. [62]

9 LIMITATIONS

Confounding factors like HbA1c, Systolic Blood Pressure, Diastolic Blood Pressure and history of cardiovascular disease was not studied

Smaller sample size might have decreased the generalisability of study

Hospital based study in a tertiary care setting might have led to bias due to the fact that complicated diabetes mellitus patients of prolonged duration usually seek treatment from tertiary care hospital

10 RECOMMENDATIONS

Further studies with increased sample size matched for confounding factors done also in other settings such as primary and secondary care will represent the true nature of study findings. Adequate Vitamin D supplementation for all patients with Diabetes Mellitus and regular follow up is required. Multicentric larger sample size studies of patients with Diabetes Mellitus to assess correlation between Vitamin D and glycaemic status is required

11 CONCLUSION

Vitamin-D is currently the topic of interest for many experts in the field of medicine and its various functions is being found in ongoing researches. According to this study, poor glycaemic control and obesity are the most likely causes of vitamin-D deficiency, which is present in nearly all diabetic patients. There is a high prevalence of hypo-vitaminosis D among patients with type-2 diabetes, particularly among patients with poor glycaemic control and in those with longer diabetes durations. In this study, Among obese women with type2 diabetes mellitus, 22% were Deficient and 78% were Inadequate for vitamin-D levels. Among Non-obese women 28% were Inadequate and 72% were Adequate for vitamin-D. Vitamin D levels correlates with the duration of diabetes mellitus. In this study mean duration of diabetes mellitus for Deficient, Inadequate, Adequate vitamin D levels were 13.64years, 10.45years and 6.47years respectively. Vitamin D levels also correlates with the fasting blood sugar and post prandial blood sugars levels. Mean fasting blood sugar levels among Deficient-287.mg/dl, Inadequate-232.36 mg/dl, Adequate person- 209.14 mg/dl. Mean post prandial blood sugar levels among deficient-344.55 mg/dl, Inadequate-276.25 mg/dl, Adequate- 244.36 mg/dl respectively. Therefore, every diabetes patient needs lifestyle changes, prompt glycaemic management, and early vitamin D treatment.

12 SUMMARY OF RESULTS

- The mean Age (years) among the subjects was 49.94 (± 6.69) years ranging from 38 to 64 years
- Among the subjects, 51 (51%) were in 41 - 50 years, 37 (37%) were in 51 - 60 years, 6 (6%) were in > 60 years and 6 (6%) were in < 40 years.
- The mean Weight (kg) among the subjects was 72.43 (± 8.06) kg ranging from 58 to 88 kg.
- The mean Height (cm) among the subjects was 160.04 (± 4.23) cm ranging from 152 to 170 cm.
- The mean BMI (kg/m2) among the subjects was 28.4 (± 3.79) kg/m2 ranging from 21.3 to 36.35 kg/m2.
- Among the subjects, 50 (50%) were Obese and 50 (50%) were Non-Obese
- Among the subjects, 90 (90%) had > 1 and 10 (10%) had < 1 Waist Hip ratio
- The mean Duration of Diabetes (years) among Obese was 10.7 (± 3.99) which is higher by 2.66 and statistically significant compared to 8.04 (± 3.85) in Non-Obese
- The mean Fasting Blood Sugar (mg/dl) among Obese was 248.42 (± 29.83) which is higher by 36.68 and statistically significant compared to 211.74 (± 19.77) in Non-Obese.
- The mean Post prandial blood sugar (mg/dl) among Obese was 296.42 (± 41.52) which is higher by 48.28 and statistically significant compared to 248.14 (± 21.36) in Non-Obese.
- The mean Total Cholesterol (mg/dl) among Obese was 233.52 (± 16) which is higher by 14.86 and statistically significant compared to 218.66 (± 12.84) in Non-Obese.

- The mean Blood Urea (mg/dl) among Obese was 43 (± 5.86) which is higher by 3.7 and statistically significant compared to 39.3 (± 6.73) in Non-Obese.
- Comparing the Urine sugar with Obesity distribution, Obese group had higher proportion of nil urine sugar with 58% followed by 1+ with 22% and least in 2+ with 20% compared to Non-Obese group which had higher proportion of nil urine sugar with 84% followed by 1+ with 14% and least in 2+ with 2%. The difference in Urine sugar distribution between Obese and Non-Obese was statistically significant ($p < 0.05$)
- The mean Serum Vitamin D (ng/ml) among Obese was 24.21 (± 3.83) which is lower by 7.17 and statistically significant compared to 31.38 (± 2.53) in Non-Obese.
- Comparing the Vitamin D level with Obesity distribution, Obese group had higher proportion of Inadequate Vitamin D level with 78% followed by Deficient Vitamin D level with 22% and least in Adequate Vitamin D level with 0% compared to Non-Obese group which had higher proportion of Adequate Vitamin D level with 72% followed by Inadequate Vitamin D level with 28% and least in Deficient Vitamin D level with 0%. The difference in Vitamin D level distribution between Obese and Non-Obese was statistically significant ($p < 0.05$).
- The mean Duration of Diabetes (years) among Deficient Vitamin D level was 13.64 which is higher than mean among Inadequate Vitamin D level which was 10.45 followed by Adequate Vitamin D level with a mean of 6.47 and the difference was statistically significant ($p < 0.05$).
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- The linear regression model with these predictors explains 78.53% variability (predictability) of Serum Vitamin D (ng/ml). Serum Vitamin D (ng/ml) decreases -0.03 times for each unit increase in Post prandial blood sugar (mg/dl) and is statistically significant. Serum Vitamin D (ng/ml) decreases -0.04 times for each unit increase in Total Cholesterol (mg/dl) and is statistically significant. Serum Vitamin D (ng/ml) decreases -0.09 times for each unit increase in Blood Urea (mg/dl) and is statistically significant. Serum Vitamin D (ng/ml) decreases -0.3 times for each unit increase in Duration of Diabetes (years) and is statistically significant. Serum Vitamin D (ng/ml) decreases -0.46 times for each unit increase in BMI (kg/m2) and is statistically significant.

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14 ANNEXURES

I – Questionnaire

Name:

Age/sex:

Occupation:

Education:

Address:

Date of admission: date of discharge:

Complaints:

History of presenting illness:

Past history:

General examination:

Vitals:

Height:

Weight:

BMI:

Waist and Hip ratio:

Duration of Diabetes:

System examination:

Lab investigations:

PARTICIPANT CONSENT FORM

Participants name :

Address :

Title of the study:

'A CLINICAL, BIOCHEMICAL PROFILE OF TYPE 2 DIABETES IN WOMEN WITH SPECIAL REFERENCE TO VITAMIN D STATUS IN OBESE AND NON-OBESE PERSON'

The details of the study have been provided to me in writing and explained to me in my own language. I confirm that I have understood the above study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason, without the medical care that will normally be provided by the hospital being affected. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purposes.i have been given an information sheet giving details of the study. I fully consent to participate in the above study

Signature of the participant : *Date :*

Signature of the witness : *Date :*

Signature of the investigator : *Date :*

நோயாளியின் ஒப்புதல் படிவம்

நோயாளியின் பெயர் :

முகவரி :

1 .மேற்கூறிய ஆய்வினை பற்றி எழுத்து மூலமாகவும் , என் சொந்த மொழியிலும் முழுவிவரம் அறிந்துகொண்டேன்.

2.இந்த ஆய்வில் நான் பங்கேற்பதற்கான தேவையை பற்றியும், எந்த சமயத்திலும் ஆய்வில் இருந்து வெளியேற முடியும் என்றும் தெரிந்து கொண்டேன்.

3.ஆய்வின்போது பெறப்படும் எனது தகவல்கள் மற்றும் மருத்துவக்குறிப்புகள் அனைத்தையும் எந்தவித தடையும் இன்றி ஆராய்ச்சிக்கு பயன்படுத்தி கொள்ளலாம்.

4. மேற் கூறிய ஆய்விற்கு எனது முழு சம்மந்தம் தெரிவிக்கிறேன்.

நோயாளியின் கையொப்பம்: தேதி :

சாட்சியின் கையொப்பம் : தேதி:

ஆராய்ச்சியாளரின் கையொப்பம் : தேதி:

PATIENT INFORMATION SHEET

I , Dr. PRAVEEN KUMAR V C(Mobile:9361975279) am conducting a study ' A CLINICAL, BIOCHEMICAL PROFILE OF TYPE 2 DIABETES IN WOMEN WITH SPECIAL REFERENCE TO VITAMIN D STATUS IN OBESE AND NON-OBESE PERSON' and assessing correlation with vitamin D levels and glycemic control, duration of diabetes, and anthropometric measures. In this study after obtaining consent your blood and urine sample is taken for analysis. You are expected to participate in the study from the time of admission at GOVERNMENT THANJAVUR MEDICAL COLLEGE until the day of discharge/expiry. At any given point of time during the study the confidentiality of the patient is assured . In participants who suffer direct physical, psychological , social, legal or economic harm as a result of their participation are entitled, after due assessment to financial or other assistance to compensate them equitably for any temporary or permanent impairment or disability. In case of injury caused due to the research you are entitled to the utmost care and free treatment. You are free to withdraw from the study by refusing for the procedures to be done without the loss of benefits that the participant would otherwise be entitled.

Dr. PRAVEEN KUMAR V C

(Mobile:9361975279)

Thanjavur Medical college,

Thanjavur

நோயாளியின் தகவல் படிவம்

மரு. பிரவீன் குமார் வ ச ஆகிய நான் பெண்நீரிழிவு நோயாளிகளுள் உடல் பருமன் அதிகம் மற்றும் உடல் பருமன் அதிகம் அல்லாதோர் இடையே வைட்டமின் டி அளவிற்கும், இரத்தசர்க்கரைஅளவு,சர்க்கரை நோயின் காலவரையறை உடல் எடை,உயரம், இடுப்பளவு ஆகியனவற்றுக்கு உள்ள தொடர்பை பற்றி ஒரு ஆய்வறிக்கை மேற் கொள்கிறேன். இதனால் நீரிழிவு நோய் வைட்டமின் டி அளவில் ஏற்படுத்தும் பாதிப்பையும், இரண்டிற்கும் உள்ள தொடர்பையும் அறிய முடியும்.இந்த ஆய்வில் தேவையான இரத்த மாதிரிகளும் மருத்துவ பரிசோதனைகளும் உங்களின் முழுமையான சம்மநம் பெற்ற பின்பே மேற்கொள்ளப்படும்.நீங்கள் இந்த தஞ்சாவூர் மருத்துவக்கல்லூரியில் உள்நோயாளியாக சேர்ந்த நாள் முதல் குணமடைந்து வீட்டுக்கு செல்லும் வரை இந்த ஆய்வு மேற்கொள்ளப்படும். எந்த ஒரு தருணத்திலும் உங்களின் ரகசியத்தன் மை உறுதி செய்யப்படும். இந்த ஆய்வில் பங்கு பெறும் நோயாளிகளுக்கு நேரடியான பிரச்சனைகள் உடல், மனம், சட்ட , சமூக மற்றும் பொருளாதார ரீதியில் ஏற்பட்டால் அதனை உடனடியாக கண்டறிந்து அவற்றை நிவர்த்திசெய்யப் படும். இந்த ஆய்வில் இருத்து வெளியேறவோ அல்லது மருத்துவ பரிசோதனைகளுக்கு மறுப்பு தெரிவிக்கவோ முழு சுதந்திரம் உண்டு.

மரு. பிரவீன் குமார் வ ச

தஞ்சாவூர் மருத்துவக்கல்லூரிமருத்துவமனை.

IV- Institutional Ethical Committee Clearance

THANJAVUR MEDICAL COLLEGE
INSTITUTIONAL ETHICAL
COMMITTEE FOR HUMAN STUDIES

Registered under National Ethics Committee Registry for Biomedical and Health Research (NECRBHR), Ministry of Health and Family Welfare, Govt of India
(Reg. No: EC/NEW/INST/2020/1058)

Chairman
Dr. J. Venkatesan M.D.,
Rtd Professor of Psychiatry,
2956, South Rampart road,
Thanjavur

Member Secretary
Dr. N. Arumugam M.D.,
Professor of Pathology,
Thanjavur Medical College

Members

Dr. S.Kumaravel M.S. DNB, Ph.D.
Professor of Orthopedics, TMC,

Dr. Shanthi Paulraj M.D.,
Professor of Anesthesiology, TMC,

Dr. Udaya Aruna M.S.,
Associate Professor of Obs & Gyn,
TMC,

Dr. B. Jayapriya D.Ch., M.D.
Associate Professor of
Pharmacology, TMC,

Dr. M. Senthilkumar M.D.,
Associate Professor of
Pathology, TMC

Dr. Eunice Swarna Jacob M.D
Associate Professor of
Microbiology, TMC

Dr. A. Vinoth M.D
Assistant Professor t of Internal
Medicine, TMC

Dr. G. Karthikeyan M.S.,
Assistant Professor of General
Surgery, TMC

Dr. L. Mageshwaran M.D.,
Assistant Professor of
Pharmacology, TMC

Dr. S. Sangeeta M.A., PhD.,
Associate Professor
Dept. of social science,
Tamil University, Thanjavur

Mr. A. Kuppusami B.Sc., B.L.,
Public Prosecutor,
Thanjavur.

Mr. S. Prince
Senior Telephone Supervisor,
BSNL, Thanjavur.

CERTIFICATE No: 843/2021

TITLE OF THE STUDY:

A clinical, biochemical profile of type 2 diabetes in women with special reference to vitamin D status in obese and non-obese person

PRINCIPAL INVESTIGATOR:

Dr. V.C. Praveen kumar, Postgraduate in Department of General Medicine, Thanjavur Medical College.

This to certify that the protocol submitted by the principal investigator of the above mentioned study has been reviewed as per standard ethical guidelines and the same has been **APPROVED** by the members of the Institutional ethical committee at its meeting held on **21.01.2021**.

21.1.21

Dr. N. Arumugam M.D.,
Member Secretary of IEC
Vice Principal & Professor of Pathology,
Thanjavur Medical College
Thanjavur

MASTER CHART

S.NO	NAME	AGE	WEIGHT	HEIGHT	BMI	W/H RATIO	FBS	PPBS	TOTAL CHL	URINE SUGAR	DUATION OF DM	B.UREA	VITD
1	sundarambal	60	78	156	32.05	>1	248	266	224	Nil	15	48	28.2
2	Umarani	58	72	152	31.06	>1	216	298	248	2+	10	36	28.17
3	Selvi	45	82	160	32.03	>1	206	236	236	Nil	8	40	24.18
4	Narreen	48	88	158	35.34	>1	256	324	258	2+	10	42	22.1
5	Rekha	44	76	158	30.52	>1	210	246	216	Nil	5	36	29.1
6	Ruckmani	54	79	155	32.88	>1	288	356	264	2+	16	44	20.18
7	Chellammal	64	76	158	30.44	>1	278	328	254	2+	20	52	18.2
8	Ranganayaki	55	81	160	31.64	>1	244	284	232	1+	10	42	24.42
9	Kala	50	84	164	31.23	>1	196	232	220	Nil	7	38	28.20
10	Devi	48	82	158	32.84	>1	236	254	244	Nil	10	42	24.56
11	Yosadha	56	77	158	30.84	>1	266	292	236	Nil	8	44	18.68
12	Mumtaj	64	75	155	31.21	>1	232	278	224	1+	15	52	25.48
13	Chinnamal	48	84	163	31.61	>1	216	286	218	1+	10	38	22.16
14	Bhuvana	42	78	160	30.46	>1	244	294	242	1+	6	34	24.80

15	**Muttammal**	**56**	**83**	**154**	**34.99**	**>1**	**258**	**302**	**248**	**2+**	**14**	**56**	**18.68**
16	**Kamatchi**	**51**	**80**	**155**	**33.29**	**>1**	**244**	**288**	**216**	**1+**	**11**	**44**	**22.94**
17	**Jyothi**	**53**	**84**	**158**	**33.64**	**>1**	**238**	**278**	**228**	**Nil**	**13**	**48**	**21.64**
18	**Kavitha**	**59**	**82**	**152**	**35.49**	**>1**	**288**	**326**	**242**	**Nil**	**19**	**52**	**19.50**
19	**Arthy**	**44**	**77**	**156**	**31.64**	**>1**	**234**	**278**	**256**	**Nil**	**4**	**36**	**26.80**
20	**Umul**	**52**	**75**	**158**	**30.04**	**>1**	**227**	**269**	**232**	**Nil**	**10**	**46**	**28.10**
21	**Jasmine**	**47**	**84**	**152**	**36.35**	**>1**	**290**	**328**	**248**	**2+**	**7**	**38**	**19.12**
22	**Lakshmi**	**49**	**76**	**156**	**31.29**	**>1**	**252**	**289**	**228**	**1+**	**9**	**42**	**26.46**
23	**Radha**	**53**	**79**	**155**	**32.88**	**>1**	**244**	**298**	**216**	**Nil**	**13**	**44**	**25.54**
24	**Zahira**	**57**	**83**	**164**	**30.85**	**>1**	**258**	**304**	**240**	**Nil**	**17**	**48**	**24.14**
25	**Vimala**	**58**	**78**	**158**	**31.24**	**>1**	**222**	**268**	**228**	**Nil**	**15**	**38**	**26.18**
26	**Chitra**	**44**	**74**	**160**	**30.40**	**>1**	**288**	**316**	**268**	**1+**	**8**	**48**	**18.10**
27	**Saraswathi**	**48**	**78**	**158**	**31.84**	**>1**	**256**	**282**	**244**	**Nil**	**6**	**42**	**29.18**
28	**Jaya**	**52**	**79**	**162**	**30.10**	**>1**	**232**	**268**	**236**	**Nil**	**10**	**36**	**28.26**
29	**Charulatha**	**56**	**76**	**156**	**31.22**	**>1**	**216**	**254**	**214**	**Nil**	**8**	**38**	**29.46**
30	**Nandhini**	**58**	**80**	**163**	**30.11**	**>1**	**206**	**238**	**206**	**Nil**	**10**	**41**	**23.93**

31	**Rekha**	**54**	**78**	**156**	**32.05**	**>1**	**288**	**346**	**256**	**1+**	**12**	**47**	**17.94**
32	**Suganya**	**50**	**82**	**156**	**33.69**	**>1**	**262**	**294**	**228**	**Nil**	**8**	**40**	**24.84**
33	**Radhika**	**46**	**84**	**164**	**31.23**	**>1**	**226**	**258**	**236**	**Nil**	**8**	**36**	**29.28**
34	**Jessica**	**42**	**88**	**162**	**33.53**	**>1**	**214**	**264**	**216**	**Nil**	**4**	**38**	**28.18**
35	**Manimegalai**	**45**	**74**	**156**	**30.40**	**>1**	**228**	**279**	**224**	**1+**	**6**	**36**	**29.14**
36	**Jayalakshmi**	**48**	**78**	**160**	**30.46**	**>1**	**234**	**286**	**220**	**Nil**	**4**	**36**	**28.28**
37	**Mary**	**51**	**86**	**168**	**30.49**	**>1**	**268**	**306**	**218**	**1+**	**8**	**40**	**28.36**
38	**Andal**	**54**	**74**	**158**	**30.40**	**>1**	**284**	**366**	**254**	**2+**	**14**	**36**	**18.90**
39	**Jenifer**	**57**	**76**	**156**	**31.22**	**>1**	**232**	**304**	**216**	**Nil**	**10**	**46**	**24.68**
40	**Rangammal**	**60**	**80**	**160**	**31.25**	**>1**	**254**	**316**	**228**	**Nil**	**15**	**48**	**23.14**
41	**Ganga**	**64**	**76**	**158**	**30.44**	**>1**	**220**	**244**	**230**	**Nil**	**12**	**42**	**25.79**
42	**Baby**	**62**	**84**	**160**	**32.81**	**>1**	**284**	**358**	**248**	**2+**	**10**	**40**	**24.64**
43	**Narmadha**	**60**	**80**	**162**	**30.48**	**>1**	**298**	**384**	**258**	**2+**	**18**	**40**	**17.40**
44	**Lucy**	**58**	**82**	**158**	**32.03**	**>1**	**230**	**286**	**236**	**Nil**	**10**	**52**	**24.92**
45	**Varalakshmi**	**56**	**78**	**154**	**32.88**	**>1**	**214**	**248**	**208**	**Nil**	**8**	**40**	**26.84**
46	**Selvalakshmi**	**54**	**78**	**156**	**32.05**	**>1**	**288**	**354**	**214**	**2+**	**14**	**56**	**24.32**

47	**Sasirekha**	**55**	**80**	**162**	**30.48**	**>1**	**254**	**310**	**210**	**Nil**	**10**	**48**	**23.26**
48	**Niranjana**	**53**	**76**	**160**	**30.46**	**>1**	**310**	**408**	**244**	**Nil**	**18**	**42**	**16.98**
49	**Padmapriya**	**57**	**76**	**158**	**30.44**	**>1**	**228**	**256**	**218**	**Nil**	**10**	**48**	**28.48**
50	**Sheeren**	**51**	**84**	**160**	**32.81**	**>1**	**316**	**394**	**248**	**1+**	**12**	**54**	**18.46**
51	**Anandhi**	**55**	**64**	**155**	**26.63**	**>1**	**212**	**256**	**210**	**Nil**	**15**	**40**	**28.60**
52	**Kavitha**	**58**	**68**	**164**	**25.28**	**>1**	**228**	**264**	**214**	**Nil**	**18**	**46**	**27.48**
53	**Vennilla**	**45**	**72**	**161**	**27.77**	**>1**	**196**	**234**	**218**	**Nil**	**8**	**40**	**31.18**
54	**Bhuvaneshwari**	**38**	**62**	**158**	**24.83**	**>1**	**184**	**228**	**212**	**Nil**	**5**	**36**	**34.28**
55	**Kalaivani**	**39**	**60**	**162**	**22.86**	**<1**	**172**	**218**	**204**	**Nil**	**6**	**38**	**33.82**
56	**Bhavani**	**42**	**64**	**168**	**22.67**	**<1**	**192**	**228**	**216**	**Nil**	**8**	**32**	**32.20**
57	**Geetanjali**	**44**	**68**	**162**	**25.91**	**>1**	**204**	**242**	**224**	**Nil**	**7**	**42**	**33.16**
58	**Gokila**	**46**	**59**	**164**	**21.94**	**<1**	**176**	**210**	**198**	**Nil**	**8**	**36**	**32.26**
59	**Pavithra**	**48**	**66**	**158**	**26.43**	**>1**	**208**	**242**	**228**	**Nil**	**12**	**38**	**29.42**
60	**Angammal**	**45**	**64**	**166**	**23.22**	**>1**	**229**	**266**	**214**	**Nil**	**8**	**40**	**31.86**
61	**Dhrovpathi**	**52**	**62**	**160**	**24.21**	**>1**	**248**	**284**	**242**	**Nil**	**12**	**46**	**28.48**
62	**Seetha**	**55**	**58**	**157**	**23.53**	**>1**	**236**	**294**	**246**	**1+**	**15**	**48**	**26.52**

63	**Parvathi**	**41**	**68**	**166**	**24.67**	**>1**	**204**	**248**	**232**	**Nil**	**4**	**40**	**33.44**
64	**Ruckmani**	**43**	**72**	**169**	**25.20**	**>1**	**213**	**239**	**218**	**Nil**	**7**	**42**	**32.84**
65	**Nilofer**	**46**	**66**	**162**	**24.96**	**>1**	**218**	**256**	**238**	**Nil**	**9**	**46**	**30.88**
66	**Rangammal**	**47**	**64**	**168**	**22.67**	**<1**	**188**	**224**	**206**	**Nil**	**10**	**32**	**32.64**
67	**Saradha**	**49**	**66**	**158**	**26.43**	**>1**	**218**	**254**	**218**	**Nil**	**12**	**30**	**32.48**
68	**Ammu**	**41**	**61**	**154**	**25.72**	**<1**	**226**	**268**	**212**	**Nil**	**5**	**36**	**33.42**
69	**Alamelu**	**39**	**58**	**165**	**21.30**	**<1**	**184**	**214**	**198**	**Nil**	**4**	**38**	**33.68**
70	**Suseela**	**40**	**62**	**169**	**21.70**	**>1**	**178**	**210**	**186**	**Nil**	**3**	**40**	**32.94**
71	**Fathima**	**45**	**65**	**158**	**26.03**	**>1**	**242**	**288**	**204**	**2+**	**6**	**40**	**32.82**
72	**Violet**	**46**	**68**	**155**	**28.30**	**>1**	**234**	**274**	**216**	**1+**	**8**	**36**	**31.16**
73	**Vathsala**	**42**	**61**	**155**	**25.39**	**>1**	**218**	**258**	**214**	**1+**	**4**	**42**	**34.40**
74	**Jemima**	**38**	**60**	**161**	**23.14**	**>1**	**228**	**268**	**212**	**1+**	**3**	**40**	**33.20**
75	**Margret**	**39**	**64**	**160**	**25**	**>1**	**218**	**256**	**240**	**Nil**	**3**	**36**	**32.88**
76	**Bhaseera**	**49**	**66**	**166**	**23.95**	**>1**	**206**	**238**	**214**	**Nil**	**10**	**42**	**28.14**
77	**Ramani**	**46**	**64**	**163**	**24.08**	**>1**	**212**	**242**	**218**	**Nil**	**6**	**44**	**29.56**
78	**Charumati**	**45**	**65**	**161**	**25.07**	**>1**	**216**	**250**	**224**	**Nil**	**8**	**38**	**30.18**

79	**Nancy**	**43**	**62**	**159**	**24.52**	**>1**	**186**	**224**	**210**	**Nil**	**5**	**36**	**32.82**
80	**Anju**	**42**	**60**	**157**	**24.34**	**>1**	**198**	**236**	**218**	**Nil**	**4**	**36**	**33.18**
81	**Monika**	**44**	**68**	**161**	**26.23**	**>1**	**176**	**202**	**198**	**Nil**	**6**	**38**	**34.12**
82	**Janaki**	**42**	**66**	**165**	**24.24**	**>1**	**188**	**218**	**204**	**Nil**	**4**	**36**	**32.28**
83	**Visalatchi**	**49**	**69**	**158**	**27.63**	**>1**	**194**	**236**	**212**	**Nil**	**10**	**42**	**28.14**
84	**Abarna**	**44**	**72**	**170**	**24.90**	**>1**	**212**	**248**	**218**	**Nil**	**6**	**40**	**30.68**
85	**Divya**	**47**	**70**	**162**	**26.67**	**>1**	**218**	**254**	**228**	**Nil**	**8**	**42**	**30.18**
86	**Bhagyam**	**56**	**65**	**158**	**26.03**	**>1**	**228**	**276**	**242**	**Nil**	**15**	**46**	**26.48**
87	**Ambika**	**58**	**68**	**159**	**26.89**	**>1**	**214**	**258**	**228**	**Nil**	**12**	**40**	**28.28**
88	**Indrani**	**62**	**62**	**168**	**21.96**	**<1**	**198**	**234**	**222**	**Nil**	**15**	**44**	**31.06**
89	**Maheshwari**	**64**	**70**	**166**	**25.40**	**>1**	**210**	**242**	**216**	**1+**	**12**	**48**	**28.89**
90	**Anjana**	**52**	**68**	**170**	**23.52**	**<1**	**212**	**254**	**215**	**Nil**	**7**	**44**	**27.84**
91	**Zahreena**	**55**	**66**	**164**	**24.54**	**>1**	**243**	**276**	**238**	**Nil**	**15**	**42**	**26.68**
92	**Rajeshwari**	**50**	**72**	**162**	**27.43**	**>1**	**216**	**248**	**228**	**Nil**	**10**	**36**	**25.68**
93	**Usha**	**48**	**70**	**158**	**28.03**	**>1**	**228**	**256**	**218**	**Nil**	**8**	**44**	**33.84**
94	**Uma**	**46**	**68**	**155**	**28.30**	**>1**	**232**	**264**	**222**	**Nil**	**8**	**40**	**34.56**

95	**Rose**	**48**	**62**	**165**	**22.77**	**<1**	**195**	**228**	**206**	**Nil**	**10**	**50**	**31.18**
96	**Mariyam**	**50**	**74**	**163**	**27.85**	**>1**	**226**	**258**	**224**	**1+**	**8**	**40**	**32.49**
97	**Priyadharshini**	**42**	**64**	**161**	**24.69**	**>1**	**218**	**246**	**218**	**Nil**	**4**	**36**	**34.15**
98	**Renukambal**	**44**	**65**	**158**	**26.03**	**>1**	**248**	**278**	**242**	**1+**	**2**	**3**	**34.18**
99	**Victoria**	**48**	**63**	**164**	**23.42**	**<1**	**224**	**246**	**222**	**Nil**	**5**	**38**	**33.38**
100	**Nagammal**	**47**	**68**	**160**	**26.50**	**>1**	**244**	**272**	**228**	**Nil**	**4**	**40**	**34.86**

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