"CORRELATION OF POSTPRANDIAL DYSLIPIDEMIA AND GLYCEMIC CONTROL WITH CAROTID INTIMA MEDIA THICKNESS IN PATIENTS WITH TYPE 2 DIABETES MELLITUS"

By

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Dissertation submitted to

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE, KOLAR, KARNATAKA

In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE
IN
GENERAL MEDICINE

Under the guidance of

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TAMAKA, KOLAR

2022

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ACKNOWLEDGEMENT

I thank the almighty for showering his blessings on me.

With humble gratitude and great respect, I would like to thank my teacher, mentor and guide, **Dr. B. N. RAGHAVENDRA PRASAD,** Professor and Unit head, Department of General Medicine, Sri Devaraj Urs Medical College, Kolar, for his able guidance, constant encouragement, immense help and valuable advices which went a long way in moulding and enabling me to complete this work successfully. Without his initiative and constant encouragement this study would not have been possible. His vast experience, knowledge, able supervision and valuable advices have served as a constant source of inspiration during the entire course of my study.

I would sincerely like to express my deepest gratitude to my co guide Dr Anil Kumar Sakalecha, Professor and HOD of Department of Radiology for helping me throughout my study

I would like to express my heartfelt gratitude to **Dr. Lakshmaiah. V** for his immense support, motivation and guidance.

I would like to express my heartfelt gratitude to **Dr. Prabakar.K, Dr.Raveesha. A, Dr.Vidyasagar. C.R and Dr.Srinivasa.s.v** for their step-by-step guidance, support and constant encouragement throughout the study. Their valuable advice and experience helped me to complete this study successfully.

I owe deep felt gratitude to my dear parents **M. DHARMAN** and **D. JAYALAKSHMI** along with my brother **D. MANOJ** and my sister in law **KAMINEE** for their moral support and constant encouragement during this study

I thank **Dr Yashwanth Lakshmaiah.V,** Department of cardiology, for his constant guidance and advice.

I would like to express my sincere thanks to **Dr. Vishwanatha reddy.N, Dr.Anitha, Dr. Manjunath, Dr. Sindhu.B.R, Dr. Thanuj.K.V,** my teachers of Department of General Medicine, Sri Devaraj Urs Medical College and Research Institute, Kolar, for their constant guidance and encouragement during the study period.

I thank **Dr Rashmi,** Department of Nephrology, for his constant guidance and advice.
I am thankful to my fellow **postgraduates, Dr. Atishaya, Dr. Rakesh, Dr. Dheeraj, Dr. Javeria, Dr. Hemanth, and Dr. Aparna** for having rendered all their co-operation and help to me during my study.

I am thankful to seniors, Dr.Samba shivarao, Dr.Pujitha, Dr.Jithendhra, Dr.Minni Meka, Dr.Rumaissa, **Dr. Sambashiva, Dr. Manoj, Dr. Hamsa, Dr. Sasishekar, Dr. Kishore.V, Dr. Sanmitharam, Dr. Deepthi, Dr. Dhruva, Dr. Sreenath, Dr. Megha and Dr. Charchit** for their constant motivation and countless help.

I am thankful to juniors, Dr. Manohar, Dr. Kavya, Dr. Pavan, Dr. Poongulali, Dr. Amulya, Dr. Manasa, Dr. Sujitha and Dr. Praveen for their constant motivation and countless help.

I thank all my Interns and nurses of ICU, MICU and ward nursing staff for their support.

Last but not the least, I thank all my patients involved in this study, without whose co-operation, this study would not have been possible.

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LIST OF ABBREVIATIONS

GLOSSARY	ABBREVIATIONS
CCA	Common carotid artery
CCA-IMT	Common carotid artery intima-media thickness
CIMT	Carotid intima media thickness
CVD	Cardiovascular disease
DM	Diabetes mellitus
FBG	Fasting blood glucose
FFAs	Free fatty acids
FPG	Fasting plasma glucose
FRS	Framingham risk score
GDM	Gestational diabetes
HbA1C	Hemoglobin a1c
HDL	High-density lipoprotein
ICA-IMT	Intima-media thickness intima-media thickness
ICAM-1	Intercellular adhesion molecule 1
IDF	International diabetes federation
IMT	Intima–media thickness
LDL	Low-density lipoprotein
NO	Nitric oxide
NT	Nitro tyrosine
PKC	Protein kinase C
PPG	Postprandial hyperglycemia
RAGE	Receptor for advanced glycation endproducts
RNS	Reactive nitrogen species
ROS	Reactive oxygen
RPG	Random plasma glucose
T1DM	Type 1 diabetes
T2DM	Type 2 diabetes

TNF	Tumour necrosis factor
UKPDS	United Kingdom prospective diabetes study
VLDL	Very low-density lipoprotein
VSMCs	Vascular smooth muscle cells

ABSTRACT

Background: In diabetic patients, the macrovascular disease is a major cause of death, and many diabetics have numerous atherosclerosis risk factors. Carotid-Intima Media Thickness (CIMT) is measured by ultrasonography, a non-invasive and quantitative method of evaluating early atherosclerotic changes in the vasculature. The current study aims to measure postprandial lipid profile, one-hour postprandial plasma glucose, HbA1C, and correlate with CIMT.

Materials: This is an observational study conducted on type-2 diabetes mellitus patients attending RL Jalappa Hospitals, Kolar, from January 2020 to January 2021 selected in a randomized manner. Mean CIMT (in mm) is considered as the primary outcome variable. Post prandial blood sugar(mg/dl) and Hba1C (%) and triglycerides(mg/dl) are considered as primary explanatory variable.

Results: A total of 65 patients with a mean age of 56.86 ± 11.89 years with 73.85% males, mean BMI 29.73 ± 4.03kg/m2 are included in the study. The mean fasting blood pressure is 137.35 ± 48.82 mg/dl; the mean-post prandial blood sugar is 267.78 ± 84.45 mg/dl, and the mean HBA1C is 7.67 ± 1.83(%), in our study. The mean triglycerides are 259.98 ± 111.46 mg/dl, the mean HDL cholesterol is 39.19 ± 11.31 mg/dl, the mean LDL cholesterol is 99.97 ± 27.26 mg/dl, and the mean total cholesterol is 191.16 ± 37.23 mg/dl. Mean CIMT is 2.73 ± 3.56 mm.

Conclusion: There is a moderate positive correlation between postprandial triglycerides and CIMT in our study. There is a weak positive correlation between HBA1C, LDL cholesterol,

total cholesterol, and CIMT. There is a weak negative correlation between HDL cholesterol and CIMT. Only postprandial triglyceride (r-value 0.651, p-value: <0.001) has a better association with CIMT compared to other lipid parameters in our study.

Key words: Type 2 diabetes mellitus, Carotid intima-media thickness, Postprandial triglyceride, Low-density Lipoprotein (LDL), High-density lipoprotein (HDL), total cholesterol.

INTRODUCTION

INTRODUCTION:

The term diabetes describes a group of metabolic disorders with hyperglycemia. The heterogeneous etio-pathology includes impaired insulin secretion, insulin action, or both, and disturbances of carbohydrate, fat, and protein metabolism.[1] Diabetes comprises many disorders characterized by hyperglycemia. There are two major types: type 1 diabetes (T1DM) and type 2 diabetes (T2DM). The differentiating factors between the two types have historically been based on age at onset, degree of loss of β cell function, degree of insulin resistance, presence of diabetes-associated autoantibodies, and insulin requirement for survival.[2] Characteristically, diabetes can appear with symptoms such as thirst, polyuria, blurring of vision, and weight loss. However, in T2DM, symptoms are often not severe or may be absent because of the slow pace at which the hyperglycemia is worsening.[3]

According to the International-Diabetes Federation (IDF), 451 million people worldwide have diabetes in 2017, with that number expected to climb to 693 million by the year 2045 if no effective prevention strategies are adopted.[4] Diabetes caused 4.2 million deaths and at least USD 760 billion dollars in health expenditure in 2019 – 10% of total spending on adults.[5] India has 6.51 crore diabetes cases which are the second-highest number of diabetics in the world and is projected to have 10.9 crore affected persons by 2035. The prevalence of this disease in the country is 9%. 3.5 crore undiagnosed cases of diabetes are estimated to be present in India.[6]

Diabetics are more vulnerable to many complications, which include macrovascular diseases (hypertension, coronary artery disease, strokes, and peripheral vascular disease), microvascular diseases (retinopathy, nephropathy, and neuropathy), and cancers.[7] Diabetic

macroangiopathy, or atherosclerosis caused by diabetes mellitus (DM), leads to cardiovascular diseases and stroke, causing the main cause of mortality and have a substantial impact on their quality of life. Endothelial as well as vascular smooth muscle cell dysfunction causing a change in vascular homeostasis is the main feature of diabetic macroangiopathy. Among the many metabolic abnormalities which are associated with the progression of atherosclerosis in diabetic patients, it can be said that prolonged hyperglycemia and insulin resistance, and also risk factors like obesity, arterial hypertension, and dyslipidemia, play a crucial role in the progression of atherosclerosis.[8]

A prolonged and intensified postprandial dysmetabolism, most notably hyperglycemia and hypertriglyceridemia, which promote endothelial dysfunction and oxidative stress, is thought to be the source of the higher prevalence of cardiovascular impairment in type 2 diabetes.[9] Thus, postprandial dyslipidemia is as significant as fasting dyslipidemia in causing atherosclerotic complications in type 2 DM.[10] Recent studies by Robert Hoffman published in the Journal of thoracic disease indicate that even acute hyperglycaemia can cause profound endothelial dysfunction affecting the mainly macrovascular system. This can be identified by the acute rise of ICAM-1 levels after a glucose challenge which is an indicator of endothelial activation.[11] Recently, in 2017 in an article published strongly suggesting the role of significant postprandial dysmetabolism in diabetic patients.[12] A prospective case-control study on 14916 patients indicates that non-fasting triglyceride levels appear to be a strong and independent predictor of future risk of MI. [13] HbA1c was likewise highly related with risks of cardiovascular complications and all-cause mortality when compared to fasting blood glucose (FBG).[14]

The measurement of intima-media thickness (IMT) of the carotid arteries is profoundly used as a diagnostic marker for atherosclerosis around the world. The carotid IMT can be simply, noninvasively, and reproducibly measured through B-mode carotid ultrasound. The carotid IMT is also a predictor of cerebral and cardiovascular events. Also, regressions of increased carotid IMT by drugs used for lipid-lowering and hypertension have been reported.[15] Thus, in order to explore the function of postprandial lipid profile, one-hour postprandial plasma glucose, and HbA1C in early atherosclerosis, these data must be related with carotid intima-media thickness.

A prospective longitudinal study to correlate between glycaemic and lipid levels with CIMT among patients with T2DM reported that CIMT measurement can be utilized as a means to detect atherosclerosis at an early stage.[16] The CIMT values were substantially linked with the fasting and the postprandial cholesterol, fasting and postprandial triglycerides, fasting as well as postprandial blood glucose values, and HbA1c values (p-value < 0.001) in a cross-sectional study. [17]

NEED OF THE STUDY:

Diabetes is a major contributor to atherosclerosis, predisposing people with T2DM to a higher chance for developing stroke as well as cardiovascular diseases. Persistent high blood sugar levels can damage the blood vessels. Diabetes results in oxidative stress on the endothelial walls causing endothelial dysfunction and consequently leading onto the thickening of carotid intima-media. Postprandial dyslipidemia and hyperglycemia act as independent and cumulative factors in causing postprandial endothelial dysfunction. It's considered that CIMT is a significant indicator of subclinical atherosclerosis. Hence by correlating post prandial lipids, postprandial hyperglycaemia, and HbA1c with CIMT, one can find their influence over atherosclerosis. The American-Heart Association currently recommends arterial intima-media thickness measurement by B mode ultrasound as a relatively safe, non-invasive, and inexpensive method of assessing sub-clinical atherosclerosis, forming an individual predictor of atherosclerotic events, among the various non-invasive imaging methods available. Timely screening of atherosclerosis utilizing CIMT is useful in slowing the progression of the disease and allowing for early intervention. There are only a few studies which relate CIMT and post prandial dyslipidemia, post prandial hyperglycemia, and HbA1c. The current study is conducted to assess and correlate the postprandial glycemic levels, postprandial dyslipidemia, and HbA1c with CIMT values.

AIMS & OBJECTIVES

AIMS AND OBJECTIVES:

To measure post prandial lipid profile, one-hour post prandial plasma glucose, and HbA1C and correlating with carotid intima-media thickness.

REVIEW OF

LITERATURE

REVIEW OF LITERATURE:

Type 2 Diabetes Mellitus:

Diabetes mellitus is a term used to describe a set of metabolic illnesses characterized by chronic hyperglycemia. It results from either impaired insulin secretion or impaired insulin efficacy, or, most often, both. Currently, the measurement of glucose in venous plasma is the standard method to diagnose diabetes.[18]

Diagnostic criteria for diabetes mellitus[18]

- HbA1c ≥6.5% (≥48 mmol/mol).
- Random plasma glucose ≥200 mg/dl (≥11.1 mmol/l).
- Fasting plasma glucose ≥126 mg/dl (≥7.0 mmol/dl).
- OGTT 2-hour glucose in venous plasma ≥200 mg/dl (≥11.1 mmol/l).

Classification:

Diabetes is divided into the following broad categories:

1. Type 1 diabetes mellitus caused due to autoimmune beta-cell destruction, usually leading to absolute insulin deficiency.
2. Type 2 diabetes mellitus is caused by a gradual decrease of sufficient beta-cell insulin production, which is typically accompanied by insulin resistance.
3. Diabetes detected in the second or third trimester of pregnancy that was not plainly overt previous to pregnancy is known as gestational diabetes mellitus.
4. Other types of diabetes, such as monogenic diabetes syndromes (such as neonatal diabetes and young-onset diabetes), diseases of the exocrine pancreas (such as cystic

fibrosis and pancreatitis), and drug- or chemical-induced diabetes (such as with glucocorticoids or after organ transplantation).[19]

Epidemiology:

T2DM accounts for between 90% and 95% of diabetes, with the highest proportions in low- and middle-income countries. It is a common and serious global health problem that has evolved in association with rapid cultural, economic and social changes, aging populations, increasing and unplanned urbanization, dietary changes such as increased consumption of highly processed foods and sugar, sweetened beverages, obesity, reduced physical activity, unhealthy lifestyle and behavioral patterns, fetal malnutrition, and increasing fetal exposure to hyperglycemia during pregnancy. T2DM is most common in adults, but an increasing number of children and adolescents are also affected.[20]

People with diabetes rose from 108 million in 1980 to 422 million in 2014. Prevalence has been rising more rapidly in low- and middle-income countries than in high-income countries. Diabetes is a major cause of blindness, kidney failure, stroke, and lower limb amputation. Between 2000 and 2016, there was a 5% increase in premature mortality from diabetes Diabetes was directly responsible for an estimated 1.5 million fatalities in 2019. In 2012, elevated blood glucose caused an additional 2.2 million fatalities. The total prevalence of diabetes in all 15 Indian states was 73%, according to a study conducted by the Indian Council of Medical Research-India Diabetes (95% CI 7·0-7·5). Diabetic prevalence varied from 4·3% in Bihar (95% CI 3·7-5·0) to 10·0% (8·7-11·2) in Punjab and was higher in urban areas (11·2%, 10·6-11·8) than in rural areas (5·2%, 4·9-5·4; $p<0·0001$) and higher in mainland states (8·3%, 7·9-8·7) than in the northeast (5·9%, 5·5-6·2; $p<0·0001$).[21]

Etiology, risk factors, pathophysiology:

Autoimmune destruction of beta-cells is not a feature of type 2 diabetes patients, and they do not have any of the other known causes of diabetes. The majority of the patients are overweight or obese, which can further cause insulin resistance. Patients who may not meet the typical weight criteria for obesity or overweight may have a higher percentage of body fat distributed primarily in the abdominal area.[22]

Many factors are responsible for T2DM, including age, obesity, unhealthy lifestyles, and prior gestational diabetes (GDM). The frequency of T2DM also varies between different racial and ethnic subgroups, especially in young and middle-aged people. There are particular populations that have more people with type 2 diabetes, for example, Native Americans, Pacific Islanders, and populations in the Middle East and South Asia.[3,23] It is also often associated with strong familial, likely genetic, or epigenetic predisposition.[3] However, the genetics of T2DM are complicated and not clearly defined, though studies suggest that some common genetic variants of T2DM occur among many ethnic groups and populations.[24]

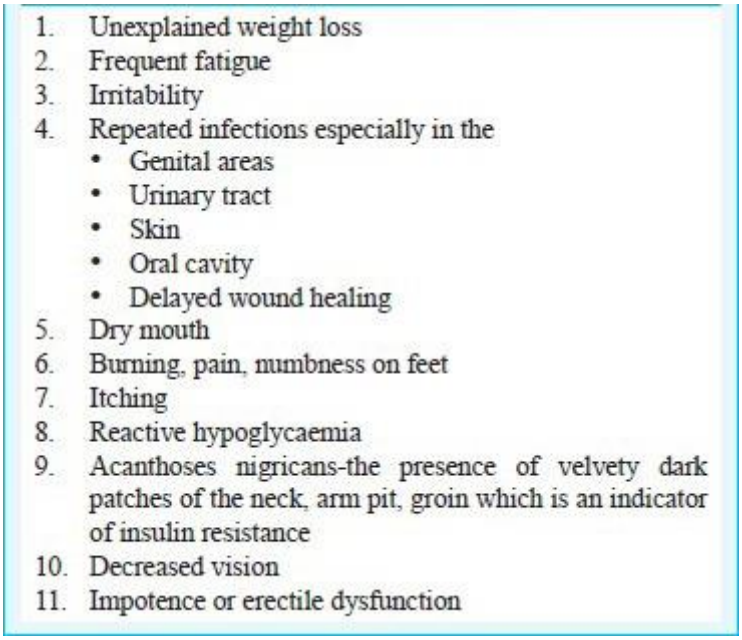
Peripheral insulin resistance decreased hepatic glucose production control, and diminishing beta-cell function characterizes the pathophysiology of this disease, finally leading to beta-cell failure. Initial insulin secretion deficits and, in many individuals, relative insulin shortage in conjunction with peripheral insulin resistance are thought to be the key occurrences.[25,26] Beta-cell dysfunctions is characterized by a decrease in the first phase of insulin production in response to glucose stimulation, which occurs prior to the onset of glucose intolerance in the patients with type 2 diabetes mellitus.[27]

Later, the second phase release of newly synthesized insulin is impaired, an effect that can be reversed, in part at least in some patients, by restoring strict control of glycemia. Desensitization, also known as beta-cell glucotoxicity, is the outcome of glucose's paradoxical inhibitory impact on insulin release and may be due to the accumulation of glycogen within the b-cell due to prolonged hyperglycemia.[28] Insulin resistance has been thought to have a crucial role in the development of type-2 diabetes since Yalow and Berson's study observed hyper insulinism in type-2 diabetes patients.[29] Some argue that insulin resistance in type-2 diabetes is solely attributable to the coexistence of increased adiposity and diabetes because obesity or an increase in intra-abdominal adipose tissue is related to insulin resistance even without diabetes.[30]

Clinical presentation:

The classic symptoms of diabetes such as polyuria, polydipsia, and polyphagia occur commonly in type 1 diabetes, which has a rapid development of severe hyperglycemia and also in type-2 diabetes with very high levels of hyperglycemia. Severe weight loss is common in type-1 diabetes or if type 2 diabetes remains undetected for a long period. Unexplained weight loss, fatigue, and restlessness, and body pain are also common signs of undetected diabetes. Symptoms that are mild or have gradual development could also remain unnoticed.[31]

Figure 1: Warning signs of diabetes:[31]



Diagnosis:

Health care professionals most often use the fasting blood sugar (FBS) test or the A1C test to diagnose diabetes. In some cases, they may use a random plasma glucose (RPG) test. The FBS blood test measures blood glucose levels at a single point in time. For the most reliable results, it is best to have this test in the morning, preferably after 8 hours of fasting. The A1C test is a blood test that provides average levels of blood glucose over the past 3 months. Other names for the A1C test are hemoglobin A1C, HbA1C, glycated hemoglobin, and glycosylated hemoglobin test. Sometimes health care professionals use the RPG test to diagnose diabetes when diabetes symptoms are present and need an immediate diagnosis.[32]

Complications:

Many problems are more common in these people, owing to complicated and linked mechanisms such as hyperglycemia, insulin resistance, low-grade inflammation, and accelerated atherogenesis. Cardiac and cerebrovascular illnesses, particularly coronary vascular disease, stroke, and heart failure, are frequently associated with diabetes and can be

fatal. Their clinical picture can be aberrant, and they can be silent for a long time. Hence type-2 diabetes mellitus must be taken as a major cardiovascular risk factor. Diabetic nephropathy is now the most common cause of end-stage renal failure. Retinopathy is another major complication of diabetic patients in whom regular screening should be done, starting from the time of diagnosis of the disease as it can be slowly progressive. Diabetic foot is a serious condition that occurs due to microangiopathy, macroangiopathy, and neuropathy in diabetics. Some cancers, as well as cognitive loss, sleep apnea syndrome, mental disorders, and bone metabolism impairments, may occur as a complication of this disease. A strategy combining systematic screening and multi-interventional therapy may be able to prevent the majority of these problems.[33]

Atherosclerosis in Type 2 Diabetes Mellitus:

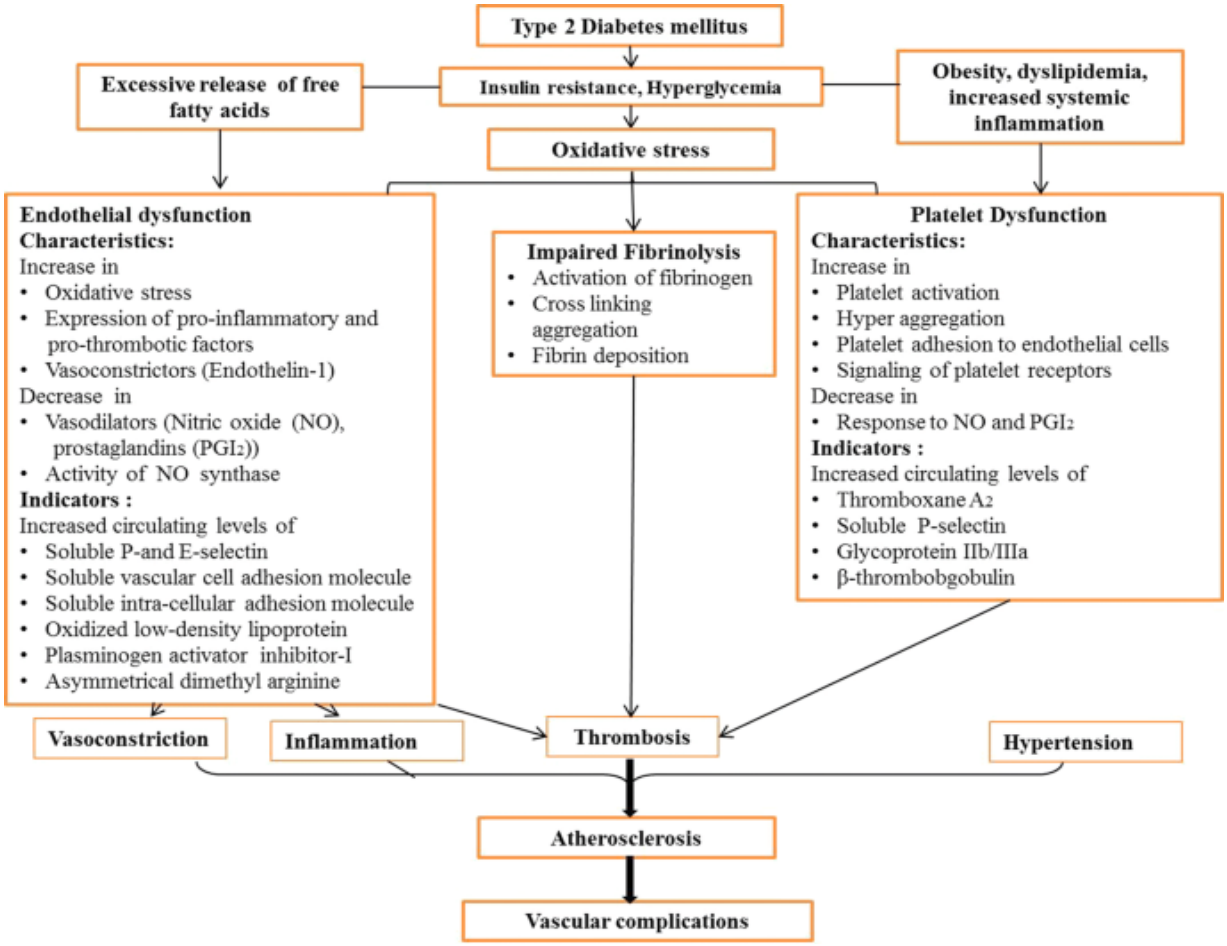
Insulin resistance which occurs in the liver and muscle is a major contributor to the establishment and evolution of diabetes mellitus, as well as other atherosclerotic diseases, including hypertension and dyslipidemia. Accordingly, patients with insulin resistance often have multiple risk factors that induce the progression of atherosclerosis through various mechanisms.[34]

Endothelial cells can synthesize and release a variety of bioactive substances which can regulate as well as maintain the function and structure of intact vessels through balancing between oxidation and anti-oxidation, and inflammation and anti-inflammation in the vascular wall; proliferation and anti-proliferation of the vascular smooth muscle cells; dilation and contraction of vessels; and coagulation and fibrinolysis of blood.[35] Accordingly, elevated low-density lipoprotein (LDL)- cholesterol levels, hyperglycemia, oxidative stress, and smoking may cause vascular endothelial dysfunction that may result in atherosclerosis.[35]

Hyperglycemia is hypothesized to cause vascular injury by causing an imbalance in NO bioavailability and the accumulation of reactive oxygen species and reactive nitrogen species (ROS and RNS), resulting in endothelial dysfunction. Furthermore, hyperglycemia damages the vascular bed through a number of cellular mechanisms, including increased intracellular AGE production, increased expression of AGE receptors (RAGE) and ligands, increased polyol and hexosamine flux, activation of protein kinase C (PKC), and overactivation of the hexosamine pathway.[36]

Insulin resistance, a key feature of T2DM, is the reduced ability of insulin to promote glucose uptake in multiple organs, including skeletal muscle, adipose tissue, and heart, and to restrain hepatic glucose output.[25,37] Insulin resistance also increases oxidative stress and PKC activation by stimulating the proliferation of the vascular-smooth muscle cells (VSMCs) and the excessive release of free fatty acids (FFAs) in adipose tissue.[38] Excess FFAs can create lipotoxicity, which can impede normal endothelium function in the same way as glucotoxicity does.[36] Insulin-resistant visceral adipocytes increase FFA inflow in arterial endothelial cells, further activating metabolite-sensitive vascular damage pathways. This could explain the link between cardiovascular diseases and insulin resistance.[36,39] Insulin resistance also increases fatty acid oxidation, resulting in elevated oxidative stress in diabetic macro vasculature and increased hyperglycemia-derived ROS generation in diabetic microvascular disorders. Thus, in both forms of diabetic vascular problems, oxidative stress is found to be the shared mechanism for causing vascular dysfunction.[40,41]

Figure 2: Pathophysiological events leading to vascular complications in T2DM patients.[36]



Diabetes patients had a higher atherosclerotic plaque burden, a larger atheroma volume, and a smaller coronary artery lumen diameter than people without diabetes.[42] Insulin resistance begins before the beginning of pre-diabetes or diabetes and worsens with the progression of diabetes, whereas hyperglycemia develops in pre-diabetes and worsens with the progression of diabetes. Insulin resistance with impaired insulin signaling, hyperinsulinemia, and hyperglycemia contributes to a number of processes, including increased free fatty acids, advanced glycated end products production, protein kinase C activation, oxidative stress, mitochondrial dysfunction, and epigenetic modifications, all of which contribute to endothelial dysfunction and inflammation, resulting in vascular smooth muscle (VSMC) and endothelial cells activation. Diabetes results in increased levels of modified (oxidized)

LDL, which is kept in the subendothelial layer of susceptible parts of the vasculature. Circulating leukocytes adhere to the endothelium wall as well as move into the intimal media's VSMC layer. These monocytes ingest lipoproteins and change into lipid-laden foam cells/macrophages, which produce proteinases and inflammatory mediators such as tumor necrosis factor-alpha (TNF-) and interleukins. Formation of Inflammasome complex as well as endoplasmic reticulum stress cause macrophage proliferation and inflammatory activation, resulting in a phenotypic transition in macrophages and VSMCs (proliferation, migration, and dedifferentiation). VSMC releases collagen in response to vascular damage, forming a fibrous cap that helps to keep atherosclerotic plaques stable. Progressive stenosis of the arteries occurs when stable lesions remodel inward. Plaques can become vulnerable as the fibrous cap thins and macrophages apoptose in late atherosclerotic lesions, where inadequate efferocytosis (phagocytic clearance) of lipid-laden macrophages leads to the creation of a necrotic core, speeding up vascular inflammation, necrosis, and thrombosis. The ensuing unstable atherosclerotic lesion complex is vulnerable to abrupt growth due to acute thrombus development, which creates a nidus for platelet thrombosis, bleeding of atherosclerotic plaque micro vessels, and fibrous cap rupture.[43]

Figure 3: Development and progression of atherosclerosis in diabetes.[43]

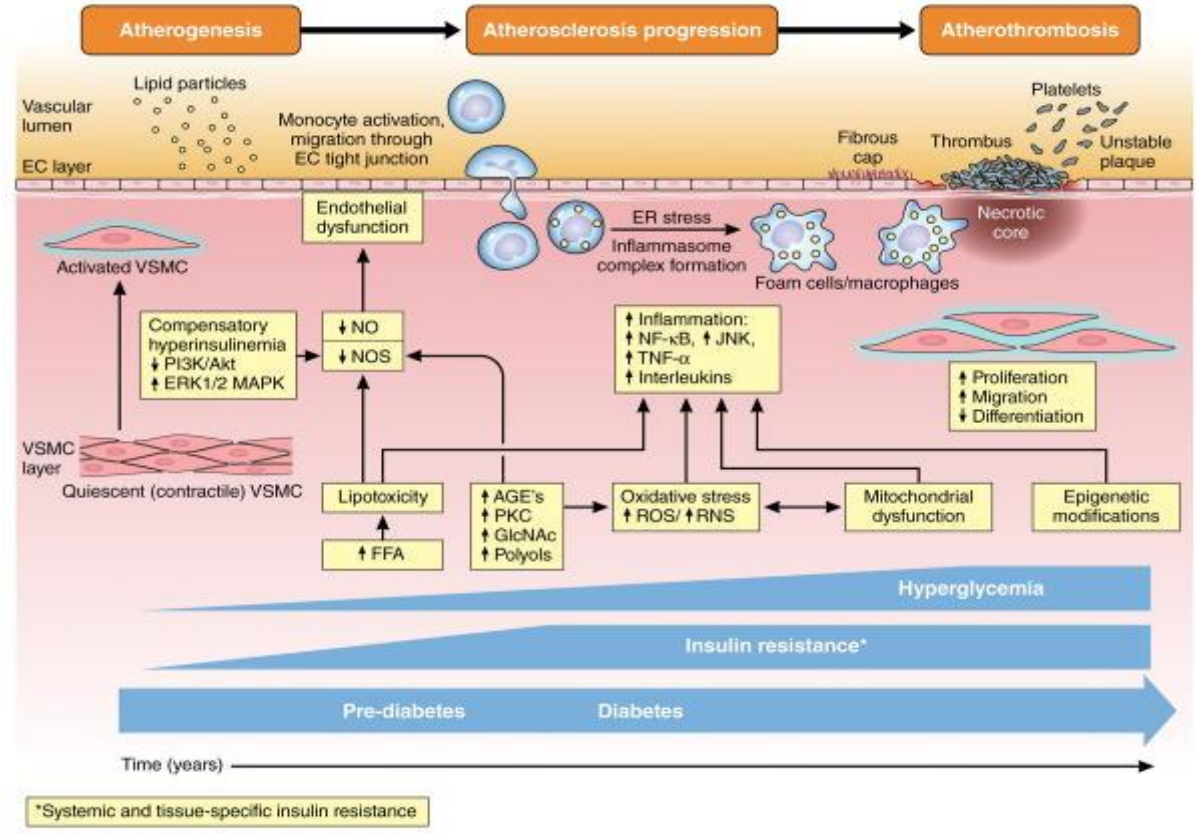
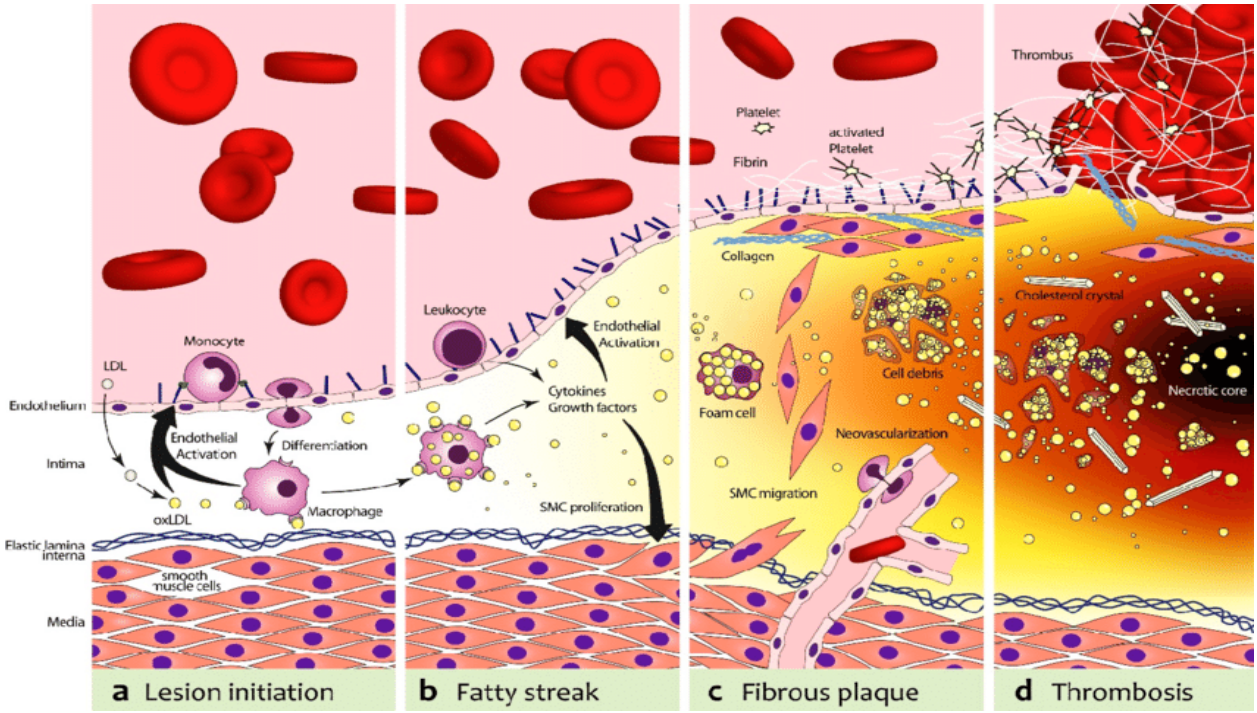


Figure 4: pathogenesis of atherosclerosis



Role of carotid intima thickness in atherosclerosis:

Carotid intima-media thickness (CIMT), recorded with B-mode sonography, is an important marker to quantify atherosclerotic burden in the common carotid artery (CCA). A long-term thesis in the development of atherosclerosis was the "response-to-injury" theory[44], in which a physical injury of the endothelium was considered to be responsible for atherosclerotic changes of vessel walls. This view was completed in the last three decades since endothelial dysfunction was considered to be a functional trigger.[45,46] Briefly, the infiltration of LDL-cholesterol through the endothelium, LDL-deposition in the intima, and the following oxidative and enzymatic processes have been described.[47] Hence, the intima-media complex of arterial walls plays a pivotal role in the development of atherosclerosis as well as may reflect different stages in the progression of the disease: a hypertensive hypertrophic response of medial cells can be observed in early phases of atherosclerosis (quantified by CIMT-measurement), while carotid plaque formation is often seen in further stages of atherosclerosis, which may be caused by inflammation, oxidation, endothelial dysfunction, and/or smooth muscle cell proliferation.[48]

The carotid artery wall is affected by the majority of significant cardiovascular risk factors. CCA-IMT and ICA-IMT had identical causative factors in a study of 3316 Framingham offspring cohort participants, except total cholesterol, which was not connected with CCA-IMT. The CCA-IMT increased by 0.007 mm, and the ICA-IMT increased by 0.037 mm each year. So, the age of the subjects has a good predictive value for CCA-IMT and ICA-IMT. Gender had a high correlation with CIMT after age. Furthermore, HDL cholesterol, smoking, hypertension, and diabetes were.[49]

Carotid intima thickness as a marker of subclinical atherosclerosis in Type 2 DM:

The CIMT (carotid intima-media thickness) is an ultrasonography biomarker of atherosclerosis that is used to detect preclinical organ damage. When compared to individuals without diabetes, those with diabetes had a higher CIMT[50],[51] on average, common CIMT was 0.13 mm higher in diabetic subjects.[51] CIMT appears to rise from persons without diabetes to those with impaired glucose tolerance, newly diagnosed diabetes, and established diabetes, though to a lesser level. Internal carotid artery intima-media thickness appears to rise faster than common carotid artery intima-media thickness (CCA-IMT).[50]

In asymptomatic patients with type-2 diabetes, CIMT was demonstrated to offer incremental predictive information to established risk variables for the prediction of coronary events. It's worth noting that there's a slight positive association of CIMT with obstructive CAD in both non-diabetic and diabetic people.[54] This indicates CIMT can be seen as a marker of atherosclerotic burden. In non-diabetes, insulin resistance has been linked to a higher CIMT. Diabetes is an independent predictor of CIMT development, according to sequential CIMT assessments.[52]

Measurement of Carotid intima-media thickness:

Classification of CIMT-values in healthy and pathological values varies considerably, depending on the CIMT measurement protocol. Therefore, conclusions from measured CIMT must be drawn by consideration of the underlying protocol. Attention should be given that the same measurement value in two different studies may be taken as normal on one and as pathologic on the other hand.[53]

CIMT could be measured with B-mode ultrasonography in a simple, safe, reliable, and cost-effective manner, and the accuracy of this test is boosted when the CIMT is recorded at multiple extracranial carotid locations.[54,55] Both the near-wall and far-wall of the carotid artery can be used in the calculation of CIMT. Media-adventitia interface is frequently simple to find on the far wall, and this site has been considered as being more accurate.[55]

Current ultrasound instrumentation with transducers 4-8 MHz is most capable of identifying the 2 arterial interfaces (lumen-intima and media-adventitia) necessary for measuring IMT. The screening examination is performed bilaterally on the carotid artery segments extracranially. These segments are the distal straight 1 cm of the common carotid arteries, the carotid bifurcations, and the initial 1 cm of the internal carotid arteries. Circumferential, longitudinal scans can identify IMTs on the near and far walls of each segment (total of 6 walls per side). The actual thickness of each lesion is measured with ultrasound instrument calipers. IMT is an operational measurement definition of a single characteristic of atherosclerosis based on considerable information documenting that both the media and intima are involved in atherogenesis as well as the anatomical progression of lesions.[56]

The normal thickness of the intima-medial layer of the common carotid artery as evaluated by B-mode ultrasound imaging was 0.74 ± 0.14 mm.[57] Some studies also indicated that CIMT <0.8 mm is associated with normal healthy individuals, and a value of CIMT at or above 1 mm is associated with atherosclerosis and significantly increased cardiovascular complications in any age group.[54,55]

Age, race, gender, habits like smoking and alcohol use, any regular endurance activity, blood pressure, dyslipidemia, nutrition patterns, risk-lowering medication use, glycemia, obesity-

related anthropometric parameters, hyperuricemia, obesity, and its related illnesses are all connected with CIMT. It has been associated to many factors and diseases, including genetic inheritance, genotypic indices, body composition, infections, rheumatoid arthritis, immunological diseases, lipid peroxidation, anthropometric hemocyte variables, vitamin D, inflammatory cytokines matrix metalloproteinases, cardiovascular parameters, and other novel aspects and conditions.[58]

Correlation of post prandial Dyslipidemia with CIMT in Type 2 DM:

A huge risk factor that corresponded most with cardiovascular disease occurrence in diabetics was blood TG levels, as per the WHO global study. High TG levels (>203 mg/dl) were related with a double the risk of Coronary vascular disease events in diabetics after a 7-year prospective analysis. Postprandial hypertriglyceridemia is thought to develop about 3-6 hours after a meal. Further, it rises after the following meal, exposing the blood vessels to this postprandial event, which leads to atherosclerosis. As a result, in diabetic patients, measuring triglyceridemia in the postprandial stage is critical. Due to the asymptomatic nature of atherosclerosis in its milder forms, using CIMT as a proxy non-invasive marker is typically reliable.[59]

In patients with high triglyceride levels in the postprandial state, mean CIMT was substantially higher than in individuals with standard FTG and PPTG levels (2.06 mm vs. 0.52 mm, $p<0.001$), according to a case-control study which demonstrated a substantial link between increased fasting triglyceride level (FTG) and CIMT, but a stronger correlation between increased triglyceride levels in post prandial state and CIMT.[60]

In diabetic patients, a positive and statistically significant correlation was noted between postprandial cholesterol, postprandial HDL, postprandial LDL, postprandial VLDL, and postprandial triglyceride and CIMT indicating that postprandial dyslipidemia is an independent factor causing endothelial dysfunction.[61] According to a cross-sectional study, CIMT levels rose in tandem with PPTG levels, indicating that CIMT and PPTG have a significant relationship (p0.01). Postprandial triglyceride levels and carotid intima-media thickness have a stronger correlation than fasting triglycerides and CIMT.[62]

A positive correlation was found of CIMT, and hence of atherosclerosis with PPTG and FTG. PPTG levels correlated best with CIMT, and the level of correlation was significant at all values of PPTG. On the other hand, FTG correlated very poorly at FTG values of less than 150 mg/dl (r = 0.1039).[63] A case-control study reported a significant association between triglycerides and average CIMT in both cases and controls (r=0.194, 95% C.I. 0.001-0.376, p=0.05). There was a weak non-significant association between LDL and average CIMT in cases (r=0.133, 95% C.I. -0.08-0.343, p=0.05).[64]

When Pearson's correlation was applied, it was reported that a higher association was detected between PPTG and CIMT (r = 0.429) as compared to that between FTG and CIMT (r = 0.258). It has been proposed that postprandial hypertriglyceridemia is an individual risk factor for the development of early development of atherosclerosis in diabetic patients. The measurement of postprandial triglyceride levels can thus be used to assess diabetic patients for the risk of atherosclerosis and other complications.[65]

Correlation of post prandial hyperglycemia with CIMT in Type 2 DM:

Postprandial hyperglycemia is one of the first anomalies of glucose homeostasis associated with type-2 diabetes, and it is exacerbated in diabetics with fasting hyperglycemia. PPG is defined as a plasma glucose level greater than 140 mg/dL after a meal. Impairment in first-phase insulin response reduced insulin sensitivity and suppressed hepatic glucose output secondary to deficiency of insulin are linked to the development of postprandial high triglyceride levels.[66]

According to a meta-analysis, there is a link between glucose levels and intima medial thickness. Overall, the correlation was positive (r = 0.082), and the confidence interval did not contain zero. A small positive association between CIMT and post-prandial glucose levels was identified by regression analysis.[67]

In a case-control study, post prandial plasma sugar was correlated with average CIMT (r=0.300, 95% C.I. 0.086-0.488, p=0.00677). A population-based prospective study showed a significantly increased thickness of the carotid wall when correlated with both fasting and postprandial glycemia (p=0.000).[68] In a prospective study, male patients with Postprandial hyperglycemia had significantly higher CIMT values (0.92) than controls (0.59), and similar observations were found in females where cases and controls had CIMT 0.80 and 0.49, respectively. The study reported no significant gender bias in CIMT in that both males and females having postprandial hyperglycemia had high CIMT values. Thus post prandial hyperglycemia is the main reason for the increase in CIMT, and as the level of post prandial plasma glucose increases, there is a proportionate increase in the CIMT.[69]

Compared with diabetic patients who had CIMT $\geq P25$, CIMT $\geq P75$ patients exhibited significantly increased plasma glucose variables (except for FPG and PG30) PG60, PG120,

PG180, PGS, HbA1C. A multivariate logistic regression analysis was performed to establish which were independently related with carotid IMT, and the results showed the post-challenge glucose spikes (PGS) were identified as the strongest determinant of CIMT from all the atherosclerosis risk factors. PGS is significantly correlated to a variety of atherosclerosis risk factors.[70]

Correlation of HbA1c with CIMT in Type 2 Diabetes Mellitus:

In a case-control study, there was a positive correlation between Hba1c and CIMT in cases and controls, but it was not significant. (r=0.209, 95% C.I = -0.110-0.410, p=0.062). [64] A study to find the association between the calculated CVD risk scores using Framingham Risk Score (FRS), United Kingdom Prospective Diabetes Study (UKPDS) risk engine and the World Health Organization (WHO) risk score and CIMT, a subclinical marker of atherosclerosis, in Type 2 diabetes (T2DM) patients, among all the variables, CIMT had a significant and positive association with duration of T2DM and HbA1c level.[71]

A population-based prospective study showed a significantly increased thickness of the carotid wall when correlated with glycated Hb and both fasting and postprandial glycemia. HbA1c more than 7 was associated with significantly increased CIMT (mean CIMT 1.113mm as compared to 0.94mm in subjects with less than 7 HbA1c).[68] Pearson correlation analysis revealed a moderate positive connection between HbA1c (log transformation) and CIMT (r=0.567, p 0.001) in a cross-sectional research.[72]

A non-interventional, cross-sectional study comparing CIMT and HbA1c levels in newly diagnosed diabetic patients found on bivariate correlation analyses found that the correlation between HbA1C with CIMT was significant ($P < 0.05$) with $r = 0.546$. Diabetic patients had

a significant increase in the intima medial thickness, which correlated with their HbA1c level.[73]

Postprandial dyslipidemia and hyperglycemia as independent and cumulative factors in causing postprandial endothelial dysfunction:

Meal absorption is a complex phenomenon, and postprandial hyperlipidemia and hyperglycemia are simultaneously present in the post-absorptive phase, particularly in diabetics and in subjects with impaired glucose tolerance. Therefore, a specific and direct role of hyperglycemia, independent of the concomitant hyperlipidemia, has been frequently questioned. When hyperglycemia and hypertriglyceridemia were simultaneously present, there was a major impairment of endothelial function compared with that observed during either hyperglycemia or hypertriglyceridemia alone, suggesting that they have an independent but cumulative effect on endothelial cells.[74]

The mechanism through which postprandial hyperglycemia and hypertriglyceridemia produce endothelial dysfunction has been proposed to be the production of oxidative stress. The process may involve the overgeneration of superoxide anion (O2−), which in turn inactivates nitric oxide (NO).[75] NO and O2− react by producing peroxynitrite, a potent, long-lived oxidant.[75] The peroxynitrite anion is cytotoxic because it oxidizes sulfhydryl groups in proteins, initiates lipid peroxidation, and nitrates amino acids such as tyrosine, which affects many signal transduction pathways.[75] The production of peroxynitrite can be indirectly inferred by nitrotyrosine (NT), and NT has been found in the plasma of diabetic patients. There is also evidence that an acute increase of glycemia simultaneously induces an increase of NT and endothelial dysfunction in healthy subjects, suggesting that peroxynitrite may be involved in the generation of endothelial abnormalities during hyperglycemia.[74,76] Also, a nitrosylation of VLDL has been found in experimental

atherosclerosis, suggesting that increased production of peroxynitrite may also be associated with altered lipid metabolism. Hyperglycemia and hypertriglyceridemia, alone or in combination, produce an increase of NT. Interestingly, NT plasma levels increase even more during combined hyperglycemia and hypertriglyceridemia.[74,77]

Hypertriglyceridemia and low HDL cholesterol levels constitute the most common dyslipidemic pattern in these patients. Postprandial lipid measurements provide a more meaningful insight regarding the lipemic status of an individual. HbA1c level is a reliable measurement of chronic hyperglycemia in a given patient. Thus postprandial dyslipidemia and hyperglycemia act as independent and cumulative factors in causing postprandial endothelial dysfunction.[17]

MOST RELEVANT STUDIES:

Singh Praliya et al.[61] **(2020)** conducted a study in type-2 diabetes mellitus patients, correlating Postprandial Lipid Profile and Carotid Intima-Media Thickness. Diabetic participants had a greater mean CIMT value (1.00±0.489) than normal subjects (0.43±0.02), indicating that Type 2 DM is linked to higher CIMT values. In diabetic patients, raised CIMT (>0.8mm) was reported in 46.7 percent, compared to 10% in non-diabetic people. The researchers found that in Type 2 DM patients with aberrant fasting and postprandial dyslipidemia, CIMT is elevated.

Kumar et al.[64] **(2020)** in their study to correlate CIMT with glycemic control and hsCRP in Statin Naïve Diabetics reported average CIMT in cases was higher in Diabetic cases than Control (0.66 + 0.14 vs 0.56±0.05, difference = 0.10 mm, 95% C.I. (0.1 - 0.17), $p < 0.0001$). HsCRP was correlated significantly with average CIMT in Diabetic cases. (r=0.512, 95% C.I.

0.33-0.658, p<0.0001). Fasting Blood Sugar was correlated with average CIMT in Diabetic cases. (r=0.234, 95% C.I. 0.015-0.432, p=0.0366). Post Prandial plasma sugar levels was also correlated with average CIMT (r=0.300, 95% C.I. 0.086-0.488, p=0.00677). The study concluded that risk factors of cardiovascular diseases like glycaemic control and inflammatory markers like hsCRP are significantly associated with CIMT even in non-smoking and statin naïve Diabetics.

Raja, Vivek et al.[78] **(2020)** did a study on the relation between triglycerides levels during fasting and postprandial states and CIMT in type-2 diabetic patients. The researchers found an association between postprandial hypertriglyceridemia and carotid intima-media thickness (CIMT).

Pramayudha et al.[72] **(2019**) conducted a cross-sectional study to find out the correlation between HbA1c in newly diagnosed T2DM and CIMT. This study involved 32 subjects with a median age of 52 (40 - 60) years. The mean value of CIMT was 0.77±0.22 mm, while the median value of HbA1c was 6.7 (5.2- 12.3). Bivariate analysis showed a moderate positive correlation between HbA1c and CIMT in newly diagnosed patients with T2DM. (r= 0.567, p < 0.001). The study demonstrated a positive correlation between HbA1c in newly diagnosed T2DM and CIMT.

Pathak et al.[62] **(2018)** did a cross-sectional study to correlate CIMT quantification as a reliable marker of atherosclerotic burden in diabetes patients. Subjects with a fasting triglyceride level less than 150 mg/dL had a mean CIMT of 13.2±0.48 mm, ranging from 0.6 to 2.4 mm. The researchers discovered that fasting and postprandial levels of triglyceride are linked to CIMT. However, because triglyceride levels in the postprandial state correlate better

with CIMT than fasting values, it can be considered an individual risk factor for atherosclerosis in type-2 diabetes mellitus.

Salla et al.[79] **(2017**) conducted a study to evaluate the intimal media thickness in 100 patients of diabetes mellitus with its duration, hypertension, age, and sex distribution, and also lipid profile abnormalities, and study the association of CIMT with dyslipidemia. The majority (74.4 percent) of the 47 patients with atherosclerotic risk factors such as diabetes mellitus had CIMT > 0.9 mm, according to this study. Also, the majority (60.3 percent) of the 53 patients with only risk factors for atherosclerotic events but no incidents had CIMT <= 0.9 mm. The study found that the CIMT in diabetic patients with atherosclerosis was considerably greater than the intimal thickness in diabetic patients with atherosclerosis risk factors but no events, with a $p < 0.01$.

Rao et al.[60] **(2016)** investigated the effect of triglyceride levels in postprandial state and their relationship with intimal thickness in patients with type-2 diabetes mellitus in a case-control study. The relationship between CIMT and fasting triglyceride and postprandial triglyceride levels was significant statistically (P0.001). The researchers found that postprandial hypertriglyceridemia in type-2 diabetes could be an important risk factor for early atherosclerosis.

Rajput et al.[63] **(2016)** have undertaken a study to find the correlation of postprandial hypertriglyceridemia with CIMT in North Indian patients with type-2 Diabetes Mellitus. Both PPTG and FTG showed a strong correlation with CIMT, the correlation coefficients being 0.879 and 0.764, respectively. If the subjects with normal FTG were taken to calculate the correlation between PPTG and CIMT, correlation coefficient was 0.848, and if only the subjects with raised FTG were taken, then the correlation coefficient was 0.735; suggesting a

stronger correlation of PPTG with CIMT at normal levels of fasting triglyceride. The study concluded that both increased postprandial levels of triglyceride and fasting triglyceride levels are determining factors for increasing CIMT in T2DM patients.

Zaidi et al.[68] **(2016)** in a population-based prospective investigation, discovered that the diabetes groups' mean CIMT is greater in more than 70% of the cases. Diabetic duration and duration of hypertension had a significant impact on wall thickness; the longer each was present, the thicker the wall was. Thickness was positively connected with glycemic management, particularly HbA1c. Although glycemic control was linked to wall thickness, there was a stronger link between systolic blood pressure and intimal thickness.

Olt et al.[80] **(2016)** In type 2 diabetic patients, researchers investigated the connection between HbA1c and CIMT, a noninvasive measure of atherosclerosis. The patients' mean HbA1c levels were 8.6±2.03%. The patients' mean CIMT readings were 0.74±0.22 mm. The HbA1c readings were not different across the groups ($p > 0.05$) according to the Student T-test and ROC curve analysis. HbA1c could not be employed as a predictor for diagnosing atherosclerosis in early stages in diabetes patients, according to the findings.

Malthesh et al.[73] **(2016)** conducted research to find the CIMT and HbA1c values in diabetes patients who were recently diagnosed and correlate between the two and to see whether diabetes manifests as an increase in the thickness of Carotid walls as early as diagnosis and to determine the usefulness of the CIMT estimation as a diagnostic tool in diabetics. Independently, HbA1c significantly positively correlates with CIMT ($p < 0.05$). The significant correlation between HbA1c and CIMT here showed that the onset of the subclinical atherosclerotic vascular changes in young patients with diabetes target population can be screened by measuring CIMT.

Bushra et al.[65] **(2015)** In type 2 diabetes patients, the researchers looked into the link between CIMT and postprandial triglyceride levels. Based on fasting and postprandial triglyceride levels, the study population (Type 2 diabetics) was separated into three groups. Postprandial hypertriglyceridemia was found to have a stronger and more significant relationship with CIMT than fasting triglycerides, suggesting that it could be a major risk factor for early atherosclerosis despite normal levels of fasting triglyceride.

Nilofer, Samed et al.[17] **(2014)** The link between postprandial lipid levels as well as HbA1c levels with carotid IMT values in diabetic persons was investigated in a cross-sectional study to explore for the function of postprandial dyslipidemia and hyperglycemia in accelerating atherosclerosis. Fasting and postprandial cholesterol, fasting and postprandial triglycerides fasting and postprandial plasma sugars, and HbA1c values all linked strongly with CIMT values (p-value 0.001), indicating that these factors have an impact on CIMT variation and hence their atherogenic potential. The study came to the following conclusion: 1. In persons with postprandial dyslipidemia, the thickness of the intima-media is dramatically enhanced (especially postprandial hypertriglyceridemia and postprandial hypercholesterolemia). Postprandial triglycerides had the greatest impact on CIMT variation among the dyslipidemias. The thickness of the Carotid intima-media is similarly influenced by HbA1c levels. The CIMT is affected by chronic hyperglycemia and postprandial dyslipidemia, both individually and cumulatively.

Jami, Swathi et al.[81] **(2012**) did a comparative investigation in type-2 diabetic patients to see the relation between fasting and postprandial triglyceride levels and carotid intimal thickness. Based on their fasting and postprandial triglyceride levels, the researchers discovered a link between postprandial hypertriglyceridemia and CIMT.

Sainani et al.[69] **(2012)** in a prospective study to compare CIMT of diabetic patients with postprandial hyperglycemia with that of non-diabetics, found that post prandial hyperglycemia is accountable for an increase in CIMT, and as the level of post prandial plasma sugar increases, there is a proportionate increase in the IMT.

Einarson et al.[67] **(2010)** searched Medline, EMBASE, Scopus, and Cochrane databases for original studies from inception to 2009, reporting both postprandial glucose levels and CIMT measurements in order to investigate the association between glucose and CIMT. Postprandial glucose levels and CIMT, both of which have been linked to poor cardiovascular outcomes, exhibit a tiny but significant association.

Hu et al.[70] **(2010)** In type-2 diabetes people, researchers looked at the links between post-challenge glucose spikes (PGS), carotid intima-media thickness (IMT), and established atherosclerotic risk factors. The study found multiple significant links between PGS and recognized atherosclerosis risk variables, implying that PGS is linked to carotid IMT independently. Independent of other risk variables, large post-challenge glucose elevations may contribute to the development of atherosclerosis in people with type 2 diabetes.

LACUNAE IN LITERATURE:

CIMT is suggested to be an important biomarker of subclinical atherosclerosis. Endothelial dysfunction is believed an important link between the postprandial state, atherosclerosis, and CVD. Postprandial dysmetabolism is associated with increased inflammation, endothelial dysfunction. Hence by correlating post prandial lipids, hyperglycemia, and HbA1c with CIMT, we can suggest the influence of postprandial dysmetabolism over atherosclerosis.

There is a dearth of literature in this area, and this study is attempted to bridge this gap in the literature in the Indian context.

MATERIALS & METHODS

MATERIALS AND METHODS:

Study site: This study was conducted in the department of General Medicine at Sri Devaraj Urs Medical College, Tamaka, Kolar

Study population: Type 2 Diabetes mellitus patients treated at RL JALAPPA HOSPITALS; KOLAR were considered as the study population.

Study design: The current study was an observational study

Sample size:

The sample size was estimated by using the correlation coefficient (r) of Post Prandial lipids with CIMT as 0.357 (i.e., r = 0.357) from the study by Kavita Bendwal et al.[82] Using these values at 95% confidence level and 80% power and substituting in the below formula[83], a sample size of 59 was obtained. Considering a 10% Non-response rate, a sample size of 59 + 5.9 = 65 subjects with Type 2 DM and Dyslipidemia will be included in the study.

Total sample size = $N = [(Z_\alpha + Z_\beta)/C]^2 + 3$

The standard normal deviate for $\alpha = Z_\alpha = 1.960$

The standard normal deviate for $\beta = Z_\beta = 0.842$

r = Correlation coefficient = 0.357

$C = 0.5 * \ln[(1+r)/(1-r)] = 0.373$

N = 59

Sampling method: All the eligible subjects were recruited into the study consecutively by convenient sampling till the sample size is reached.

Study duration: The data collection for the study was done between January 2020 to December 2021 for a period of 1 year.

Inclusion Criteria:

All Type 2 Diabetes mellitus patients above 18 years of age treated at RL Jalappa hospitals Kolar selected in a randomizer manner.

Exclusion criteria:

- Patients with known macrovascular complications like Cerebro vascular accidents, coronary artery diseases, and Peripheral vascular diseases.
- Patients on drugs causing dyslipidemia and dysglycemia like Diuretics, fluoroquinolones, beta-blockers, steroids, anti psychotics
- Known chronic kidney disease patients
- Conditions affecting hemoglobin- methemoglobinemia, anemia, polycythemia**.**

Ethical considerations: The study was approved by the institutional human ethics committee. Informed written consent was taken from all the study participants, and only those participants willing to sign the informed consent were included in the study. The risks and benefits involved in the study and the voluntary nature of participation were explained to the participants before obtaining consent. The confidentiality of the study participants was maintained.

Data collection tools: All the relevant parameters were documented in a structured study proforma.

Methodology:

- Patients were selected as per the inclusion and exclusion criteria.
- Patients were diagnosed as Type-2 Diabetes Mellitus as per American Diabetes Association Diagnosis criteria

- They were explained about the procedure, and their consent was taken, and they were subjected to blood investigations and carotid Doppler.
- Clinical, laboratory and sociodemographic data were elicited and recorded in a predefined proforma.

1. Socio demographic data

- Age
- Sex

2. Clinical data

- Height
- Bodyweight
- Clinical examination
- BMI

3. Laboratory data

- The patients were selected, and their age, sex, BMI matched, and their results were correlated.
- In these patients, postprandial lipid profile and one-hour post prandial blood glucose were correlated with carotid intima-media thickness, and the results were analyzed.

Investigations done:

- 4 HOURS POST PRANDIAL LIPID PROFILE
- ONE HOUR POST PRANDIAL PLASMA GLUCOSE
- HbA1C
- CAROTID DOPPLER MEASURING CAROTID INTIMA MEDIA THICKNESS
- CBC
- RENAL FUNCTION TESTS

STATISTICAL METHODS:

Mean CIMT (in millimeter) was considered as the primary outcome variable. post prandial blood sugar(mg/dl) and Hba1C (%) and triglycerides(mg/dl) were considered as Primary explanatory variable. Descriptive statistics were used to analyze data in accordance with the study's objectives. Data were expressed as the mean, 95% confidence interval (CI; lower and upper bounds), median, minimum and maximum, and percentage, where appropriate. Data was also represented using appropriate diagrams like bar diagrams and pie diagrams. Association between quantitative explanatory and outcome variables was assessed by calculating the Pearson correlation coefficient (R), and the data was represented in a scatter diagram P-value < 0.05 was considered statistically significant. Data were analyzed by using SPSS software, V.22.[84]

OBSERVATIONS AND RESULTS

RESULTS:

A total of 65 subjects were included in the final analysis.

Table 1: Descriptive analysis of age (in years) in study population (N=65)

Parameter	Mean ± SD	Median	Minimum	Maximum
Age (in years)	56.86 ± 11.89	58.00	34.00	90.00

The mean age was 56.86 ± 11.89 years, ranged from 34 to 90 years. (Table 1)

Table 2: Descriptive analysis of gender in the study population (N=65)

Gender	Frequency	Percentages
Male	48	73.85%
Female	17	26.15%

Among the study population, 48(73.85%) were male, and the remaining 17(26.15%) were female. (Table 2 & Figure 5)

Figure 5: Pie chart of gender in the study population (N=65)

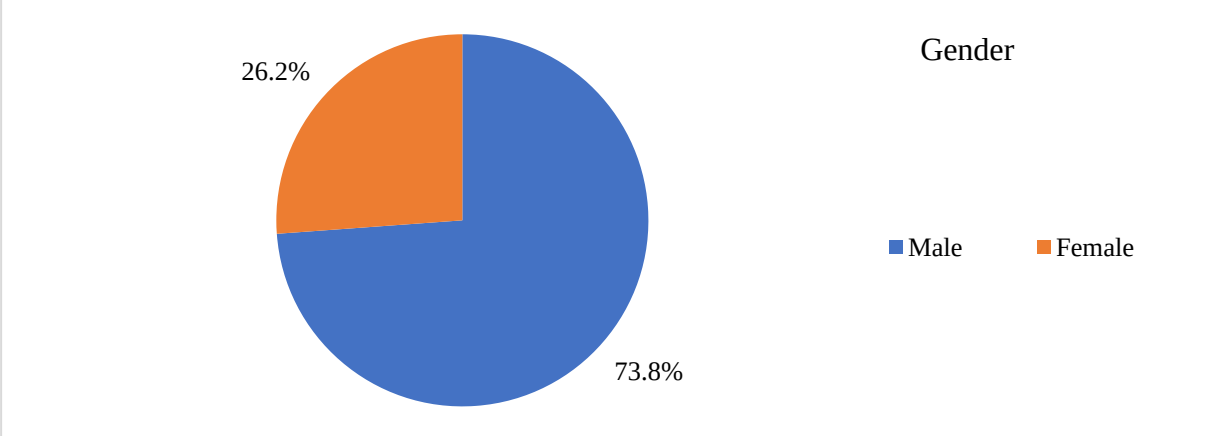


Table 3: Descriptive analysis of BMI in study population (N=65)

Parameter	Mean ± SD	Median	Minimum	Maximum
BMI (in kg/m2)	29.73 ± 4.03	29.00	22.00	39.00

The mean BMI was 29.73 ± 4.03kg/m2, ranged from 22 to 39. (Table 3)

Table 4: Descriptive analysis of diagnosis in the study population (N=65)

Diagnosis	Frequency	Percentages
COPD	10	15.38%
Dengue fever	8	12.31%
UTI	6	9.23%
Viral fever	6	9.23%
urosepsis	5	7.69%
DKA	4	6.15%
Others	26	40%

Among the diagnosis, 10(15.38%) had COPD, 8(12.31%) had dengue fever, 6(9.23%) had UTI and viral fever for each, 5(7.69%) had urosepsis, 4(6.15%) had DKS, and 26(40%) had others. (Table 4)

Table 5: Descriptive analysis of hypertension in the study population (N=65)

Hypertension	Frequency	Percentages
Yes	31	47.69%
No	34	52.31%

Out of 65 participants, 31(47.69%) had hypertension. (Table 5 & Figure 6)

Figure 6: Pie chart of hypertension in the study population (N=65)

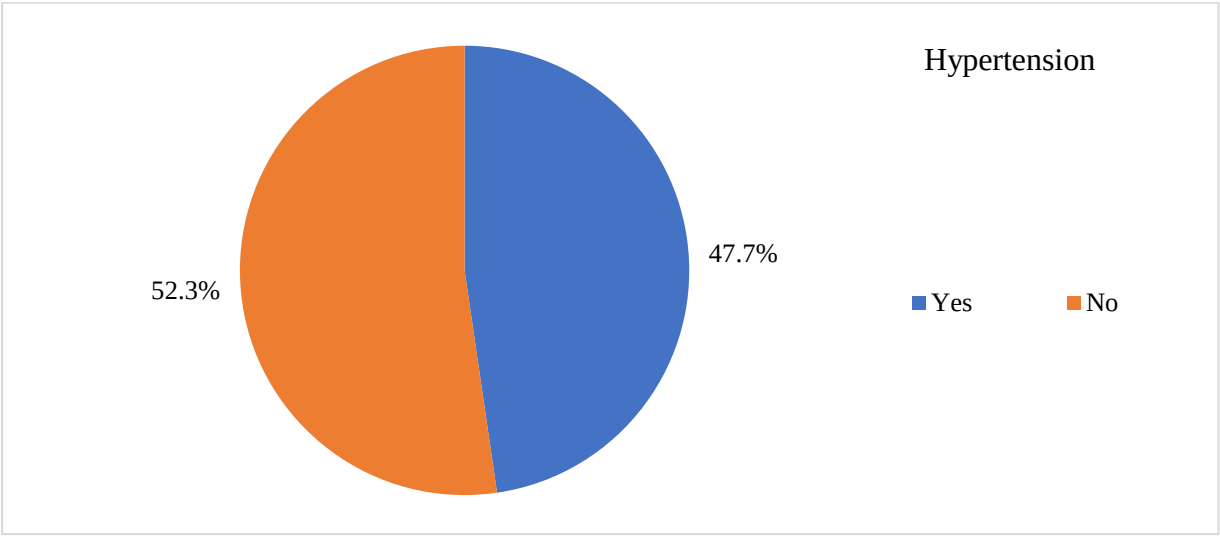


Table 6: Descriptive analysis of smokers in the study population (N=65)

Smoker	Frequency	Percentages
Yes	17	26.15%
No	48	73.85%

Out of 65 participants, 17(26.15%) were smokers. (Table 6 & Figure 7)

Figure 7: Pie chart of smokers in the study population (N=65)

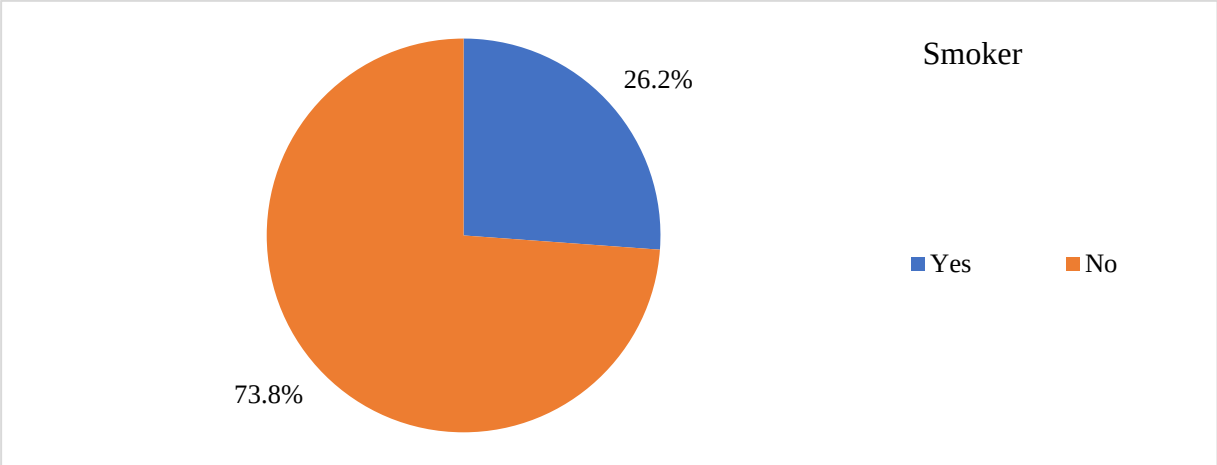


Table 7: Descriptive analysis of alcohol in the study population (N=65)

Alcohol	Frequency	Percentages
Yes	4	6.15%
No	61	93.85%

Out of 65 participants, 4(6.15%) were alcoholics. (Table 7 & Figure 8)

Figure 8: Bar chart of alcohol in the study population (N=65)

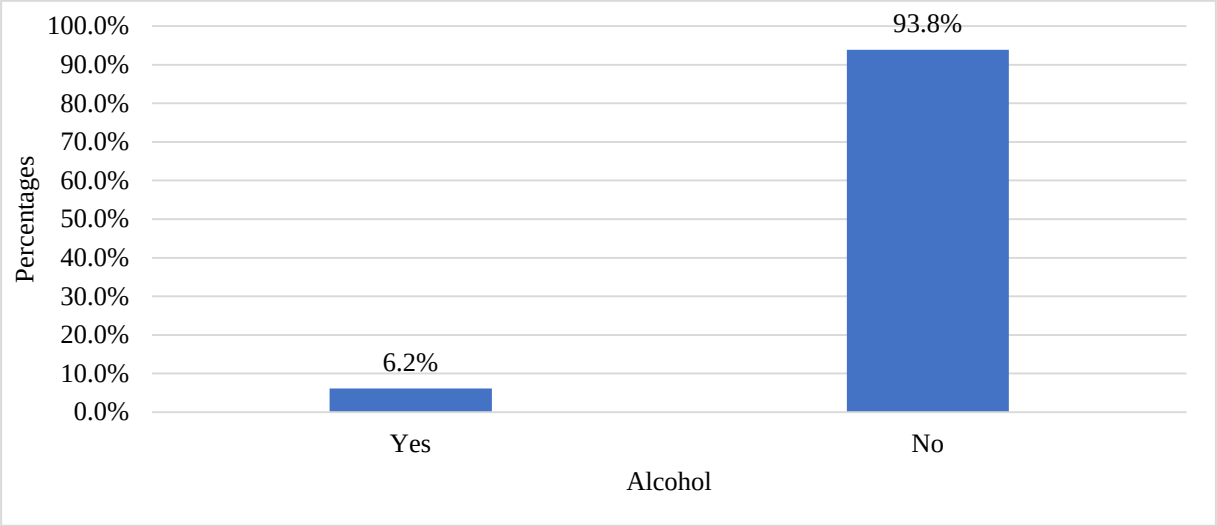


Table 8: Descriptive analysis of vital signs in study population (N=65)

Parameter	Mean ± SD	Median	Minimum	Maximum
Pulse rate (in bpm)	80.4 ± 9.64	80.00	60.00	120.00
Systolic Blood Pressure (mmhg)	121.38 ± 17.4	120.00	80.00	170.00
Diastolic Blood Pressure (mmhg)	77.38 ± 12.03	80.00	40.00	120.00

The mean pulse rate, systolic blood pressure, and diastolic blood pressure were 80.4 ± 9.64 bpm, ranged from 60 to 120 beats per minutes121.38 ± 17.4 mmHg, ranged from 80 to 170 mmHg and 77.38 ± 12.03mmhg, ranged from 40 to 120 respectively. (Table 8)

Table 9: Descriptive analysis of other parameter in the study population (N=65)

Other parameter	Frequency	Percentages
CVS (s1s2+.)	65	100%
RS (B/LAE+)	65	100%
P/A (SOFT)	65	100%
CNS (NFND)	65	100%

Out of 65 participants in our study, all of them 65(100%) had CVS (s1s2+.), RS (B/LAE+), P/A (SOFT), and CNS (NFND) for each. (Table 9)

Table 10: Descriptive analysis of renal parameter in study population (N=65)

Parameter	Mean ± SD	Median	Minimum	Maximum
Blood urea (in mg/dl)	23.32 ± 9.27	20.00	2.00	51.00
Serum creatinine (in mg/dl)	1.01 ± 1.04	0.90	0.20	9.00
Sodium (in mEq/L)	136.31 ± 4.1	136.00	127.00	147.00
Potassium (in mmol/L)	4.05 ± 0.54	4.00	3.00	5.48

Among the renal parameter, the mean blood urea was 23.32 ± 9.27 mg/dl, ranged from 2 to 51, serum creatinine was 23.32 ± 9.27mg/dl, ranged from 0.20 to 9. the mean sodium was 136.31 ± 4.1 mEq/L, ranged from 127 to 147. the mean potassium was 4.05 ± 0.54 mmol/l, ranged from 3 to 5.48. (Table 10)

Table 11: Descriptive analysis of blood sugar parameter in study population (N=65)

Parameter	Mean ± SD	Median	Minimum	Maximum
Fasting Blood Pressure (in mg/dl)	137.35 ± 48.82	126.00	54.00	285.00
Post prandial blood sugar(mg/dl)	267.78 ± 84.45	242.00	94.00	482.00
Hba1C (%)	7.67 ± 1.83	7.20	5.10	14.40

The mean fasting blood pressure was 137.35 ± 48.82 mg/dl, ranged from 54 to 285, the mean post prandial blood sugar was 267.78 ± 84.45 mg/dl, ranged from 94 to 482, and the mean HBA1C was 7.67 ± 1.83(%), ranged from 5.10 to 14.40. (Table 11)

Table 12: Descriptive analysis of lipid profile in study population (N=65)

Parameter	Mean ± SD	Median	Minimum	Maximum
Triglycerides(mg/dl)	259.98 ± 111.46	240.00	103.00	581.00
HDL Cholesterol (mg/dl)	39.19 ± 11.31	37.00	23.10	79.00
LDL Cholesterol (mg/dl)	99.97 ± 27.26	98.00	11.00	193.00
Total Cholesterol (mg/dl)	191.16 ± 37.23	189.00	79.70	285.20

The mean triglycerides were 259.98 ± 111.46 mg/dl, ranged from 103 to 581, the mean HDL cholesterol was 39.19 ± 11.31 mg/dl, ranged 23.10 to 79, the mean LDL cholesterol was 99.97 ± 27.26 mg/dl, ranged 11 to 193 and the mean total cholesterol was 191.16 ± 37.23 mg/dl, ranged 79.70 to 285.20 in the study population. (Table 112)

Table 13: Descriptive analysis of the carotid intima-media thickness test in study population (N=65)

Parameter	Mean ± SD	Median	Minimum	Maximum
Right CIMT (in Millimetre)	2.72 ± 3.65	0.80	0.40	17.00
Left CIMT (in Millimetre)	2.75 ± 3.57	0.80	0.40	13.00
Mean CIMT (in Millimetre)	2.73 ± 3.56	0.80	0.40	14.50

Among the carotid intima-media thickness test, the mean right side was 2.72 ± 3.65 millimetre, ranged 0.40 to 17, the ema left side was 2.75 ± 3.57 millimetre, ranged 0.40 to 13, and the mean CIMIT was 2.73 ± 3.56 millimetre, ranged 0.40 to 14.50. (Table 13)

Table 14: Correlation between post prandial blood sugar(mg/dl) and mean CIMT (millimeter) (N=65)

Parameter	Pearson Correlation	P value
Post prandial blood sugar(mg/dl)	0.397	0.001

There was a weak positive correlation between Post prandial blood sugar(mg/dl) and mean CIMT (millimeter) **(**R value: 0.397, P value: 0.001). (Table 14 & Figure 9)

Figure 9: Scatter plot diagram of a correlation between post prandial blood sugar(mg/dl) and mean CIMT (millimeter) (N=65)

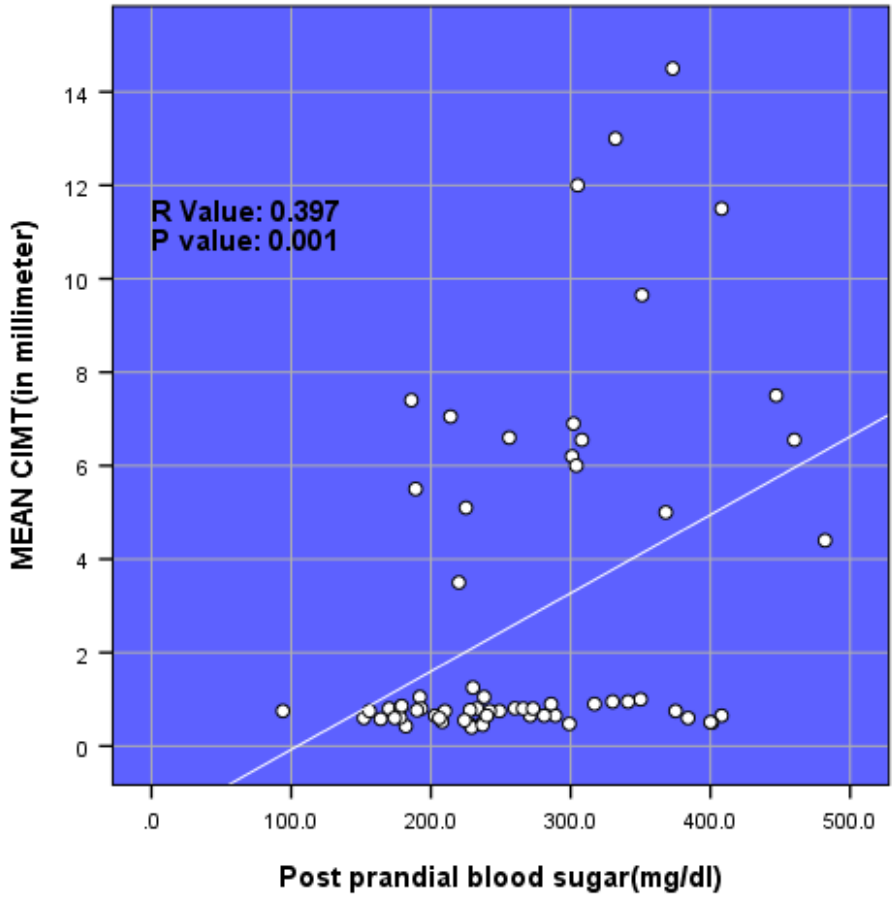


Table 15: Correlation between HBA1C (%) and mean CIMT (millimeter) (N=65)

Parameter	Pearson Correlation	P value
HBA1C (%)	0.265	0.033

There was a weak positive correlation between HBA1C (%) and mean CIMT (millimeter) (R-value: 0.265, P value: 0.033). (Table 15 & Figure 10)

Figure 10: Scatter plot diagram of a correlation between HBA1C (%) and mean CIMT (millimeter) (N=65)

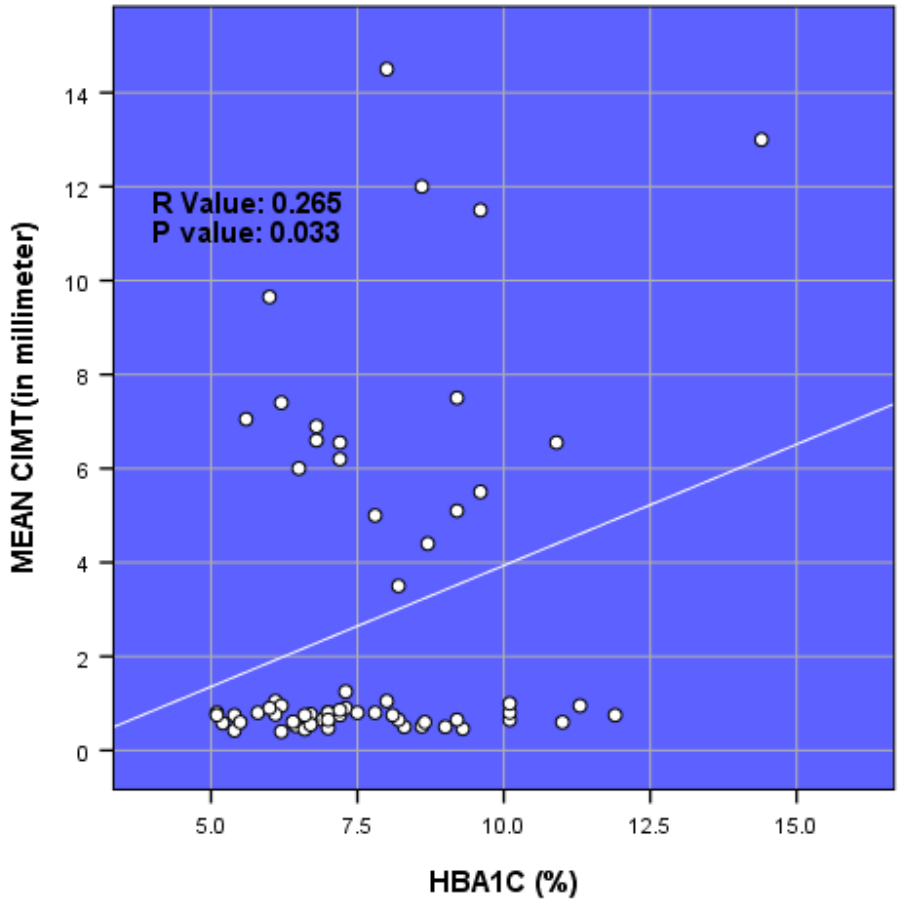


Table 16: Correlation between triglycerides (mg/dl) and mean CIMT (millimeter) (N=65)

Parameter	Pearson Correlation	P value
Triglycerides(mg/dl)	0.651	<0.001

There was a moderate positive correlation between Triglycerides(mg/dl) and mean CIMT millimeter (R value: 0.651, P value: <0.001). (Table 16 & Figure 11)

Figure 11: Scatter plot diagram of a correlation between triglycerides (mg/dl) and mean CIMT (millimeter) (N=65)

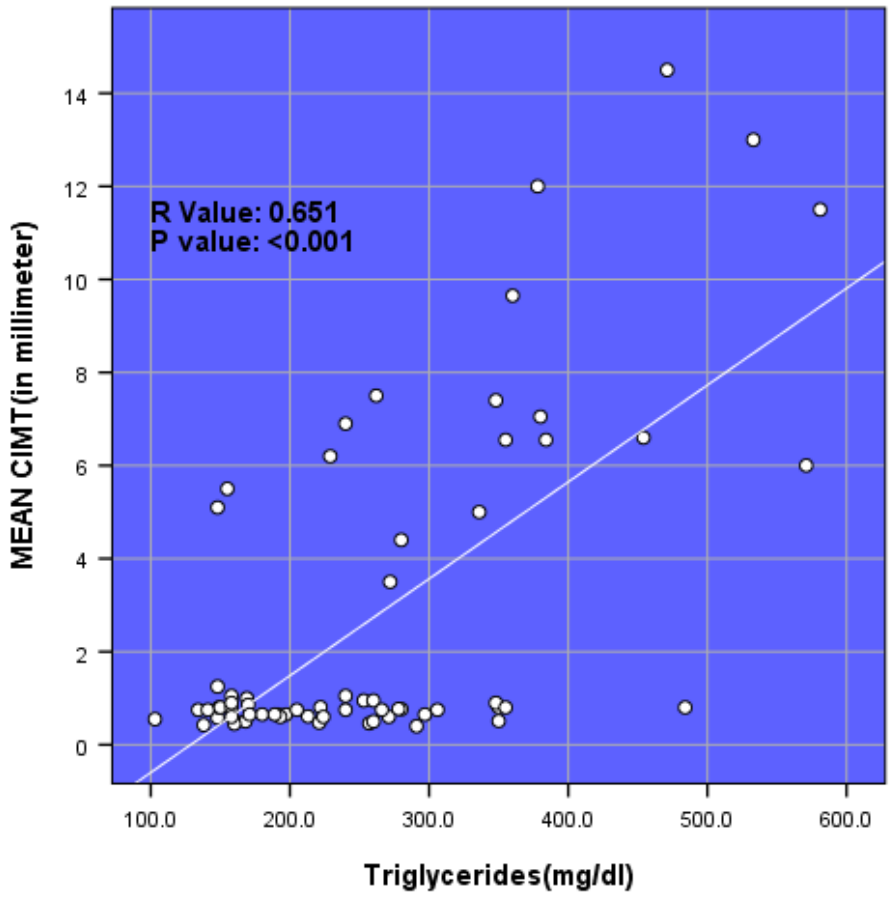


Table 17: Correlation between HDL cholesterol (mg/dl) and mean CIMT (millimeter) (N=65)

Parameter	Pearson Correlation	P-value
HDL cholesterol **(**mg/dl)	-0.120	0.340

There was a weak negative correlation between HDL cholesterol (mg/dl) and mean CIMT millimeter (R-value: -0.120, P value: 0.340). (Table 17 & Figure 12)

Figure 12: Scatter plot diagram of a correlation between HDL cholesterol (mg/dl) and mean CIMT (millimeter) (N=65)

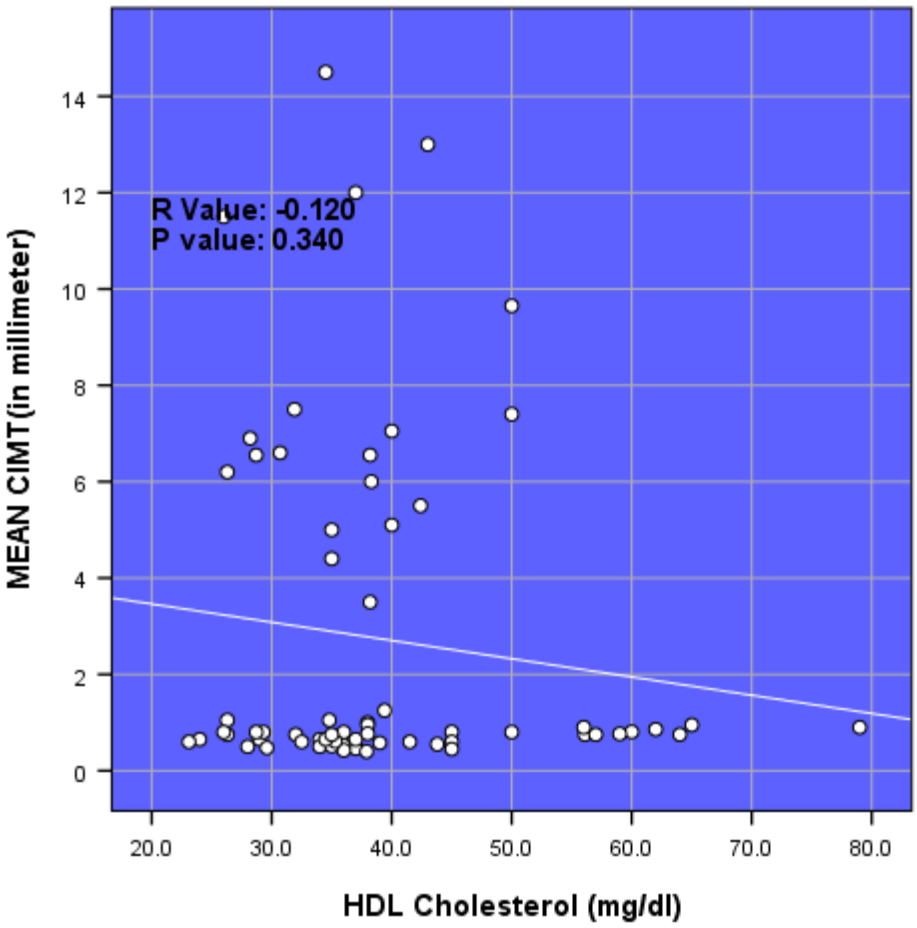


Table 18: Correlation between LDL cholesterol (mg/dl) and mean CIMT (millimeter) (N=65)

Parameter	Pearson Correlation	P-value
LDL cholesterol **(**mg/dl)	0.038	0.765

There was a weak positive correlation between LDL cholesterol (mg/dl) and mean CIMT millimeter (R value: 0.038, P value:0.765). (Table 18 & Figure 13)

Figure 13: Scatter plot diagram of a correlation between LDL cholesterol (mg/dl) and mean CIMT (millimeter) (N=65)

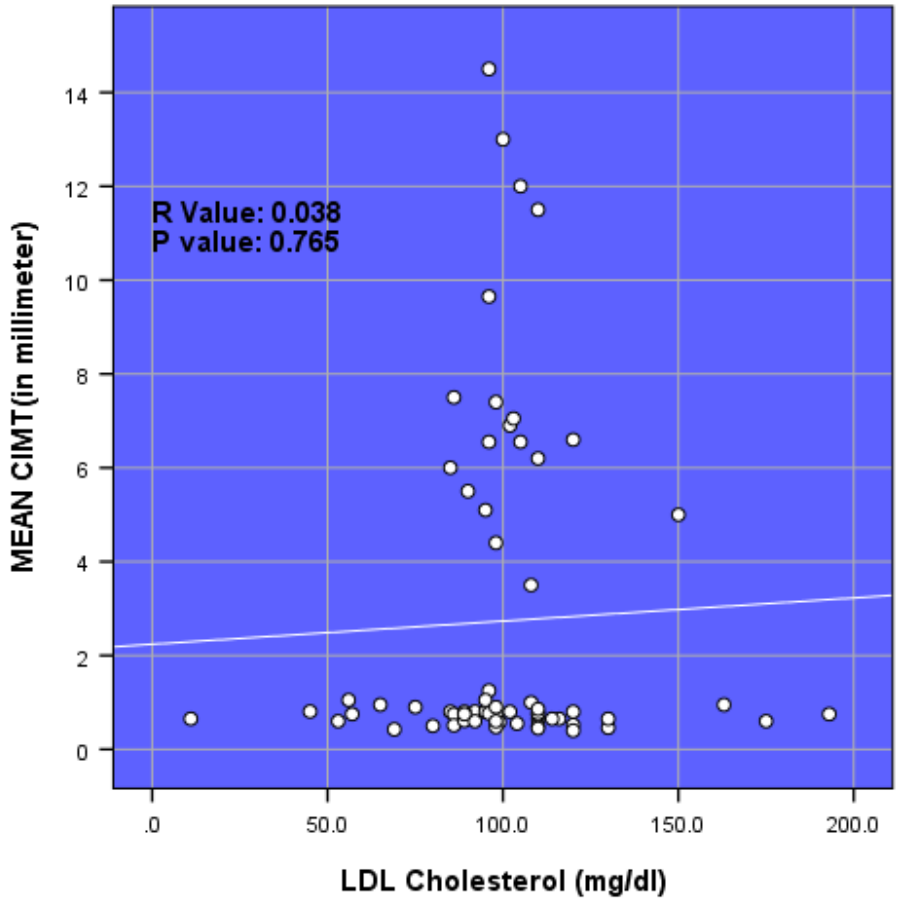
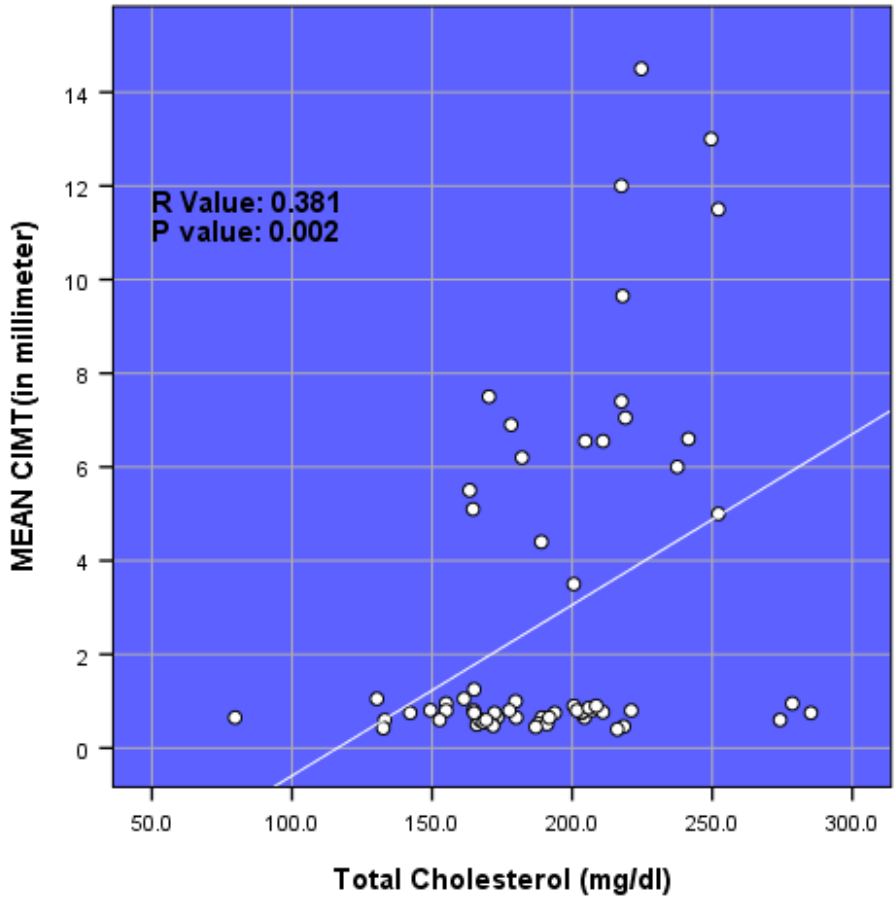


Table 19: Correlation between total cholesterol (mg/dl) and mean CIMT (millimeter) (N=65)

Parameter	Pearson Correlation	P-value
Total cholesterol **(**mg/dl)	0.381	0.002

There was a weak positive correlation between total cholesterol (mg/dl) and mean CIMT millimeter (R value: 0.381, P value: 0.002). (Table 19 & Figure 14)

Figure 14: Scatter plot diagram of a correlation between total cholesterol (mg/dl) and mean CIMT (millimeter) (N=65)



DISCUSSION

DISCUSSION:

Diabetes and related complications are associated with long-term damage and failure of various organ systems. Diabetes induces changes in the microvasculature, causing extracellular matrix protein synthesis and capillary basement membrane thickening, which are the pathognomic features of diabetic microangiopathy. These changes in conjunction with advanced glycation end products, oxidative stress, low-grade inflammation, and neovascularization of vasa vasorum can lead to macrovascular complications.[85] Type 2 DM is increasingly linked with the development of premature atherosclerosis and a high risk of cardiovascular mortality and morbidity. Postprandial lipid measurements provide a more meaningful insight regarding the lipemic status of an individual as we are in post-prandial in most of the day. Postprandial dyslipidemia and hyperglycemia act as independent and cumulative factors in causing postprandial endothelial dysfunction. CIMT is considered to be an important marker of subclinical atherosclerosis. Hence by correlating post prandial lipids and hyperglycemia with CIMT, we can suggest the influence of postprandial dysmetabolism over atherosclerosis. This is an observational study to measure post prandial lipid profile, one-hour post prandial plasma glucose, and HbA1C and correlate with CIMT in type-2 diabetes mellitus patients above 18 years of age treated at RL Jalappa Hospitals, Kolar selected in a randomizer manner. Mean CIMT (in mm) is considered as the primary outcome variable. post prandial blood sugar(mg/dl) and Hba1C (%) and triglycerides(mg/dl) are considered as primary explanatory variable.

A total of 65 subjects with a mean age of 56.86 ± 11.89 years, meeting the inclusion as well as exclusion criteria, are included in the final analysis. The study population has a preponderance of male subjects, with 73.85% males and 26.15% females. Comparable to our

study, the mean age of the study population was 54.64±9.53 in Bendwal et al.[82] study and they had 60.83% males and 39.16% females. Praliya et al.[61] had a younger population with a mean age of 48.13±9.39 years, and 53.3% among participants were males with 46.7% females. In Olt et al.[80] study the mean age of the patients, was similar to ours at 58.4±10.7 years, and they had a predominantly female subjects, with 69.2% of the patients being female and 30.8% male. Kumar et al.[64] in an observational cross-sectional observational study of 80 cases suffering from type 2 DM and 20 non-diabetic controls had a younger age case with a mean age of 49.11±9.63.

The mean BMI was 29.73 ± 4.03kg/m2 in our study, comparable to that in Bendwal et al.[82] study at 28.75±10.25 and Kumar et al.[64] who had a BMI of 28.18±2.97 in the cases in their study. The prevalence of hypertension in this study is 47.69% which is much higher than that observed in Pramayuda et al.[72] study which was 28.1%. Only 16 % had hypertension in Raja et al.[78] study.

The mean fasting blood pressure is 137.35 ± 48.82 mg/dl, the mean post prandial blood glucose levels is 267.78 ± 84.45 mg/dl, and the mean HBA1C is 7.67 ± 1.83(%), in our study. The mean postprandial blood sugar in diabetic patients was 273.73 ± 35.402 in Praliya et al.[61] study. Bendwal et al.[82] had a mean HBA1c comparable with our study at 7.9±2.30. The mean HbA1c value was 8.6±2.03 % in Olt et al.[80] study. The mean HbA1C was higher than our study at 10.21% ±2.41 as reported by Mathesh et al.[73] The mean HbA1c was 7.461± 0.786 in Nilofer Samed's study.[17]

Table 20: Mean HbA1c across studies:

Study	**Mean HbA1c** (%)
Current study	7.67 ± 1.83
Bendwal et al.[82]	7.9±2.30
Olt et al.[80]	8.6±2.03
Mathesh et al.[73]	10.21% ±2.41
Nilofer Samed.[17]	7.461± 0.786

The mean triglycerides are 259.98 ± 111.46 mg/dl, the mean HDL cholesterol is 39.19 ± 11.31 mg/dl, the mean LDL cholesterol is 99.97 ± 27.26 mg/dl, and the mean total cholesterol is 191.16 ± 37.23 mg/dl, in our study population. The mean serum postprandial triglycerides in the NN, NH, and HH group were 152.52±31.15, 274.26±54.33, and 296.92±65.76 (mg/dl) respectively in Bushra et al.[86] study. Compared to our study, in Bendwal et al.[82] study the mean total cholesterol 297.21±128.76 mg/dL, mean triglycerides 415.02±280.32 and mean LDL 148.62±72.78 are all much higher, with only the mean HDL at 41.95±13.74 comparable to our study. In Raja et al.[78] study the mean serum total cholesterol in the normal fasting and normal postprandial triglyceride group was 147.50±11.08, mean serum LDL was 87.92±3.58, in the normal fasting and high postprandial group, the mean serum total cholesterol was 159.40±6.19, mean serum LDL was 92.90±5.135, and high fasting and high postprandial group mean serum total cholesterol was 177.52±10.91mg/dl, mean serum LDL was 97.33±6.88 mg/dl. The mean serum total cholesterol in the normal fasting and normal postprandial triglycerides, normal fasting and high postprandial triglycerides, and high fasting and high postprandial triglycerides group were 148.47±32.60, 174.0±37.63, 181.07±33.79 (mg/dl) respectively in Bushra et al.[86] study.

In healthy middle-aged adults, CIMT values between 0.6 and 0.7 mm have been considered normal, while CIMT of 1 mm or more has been associated with a significant increased absolute risk of coronary disease.[87] Mean right side CIMT is 2.72 ± 3.65 mm and on the left side is 2.75 ± 3.57 mm, giving a mean CIMT of 2.73 ± 3.56 mm in our study. Praliya et al.[61] found the mean CIMT was raised in diabetic at 1.00 ± 0.489 as compared to healthy subjects, which was 0.43 ± 0.02, signifying that Type-2 DM is associated with raised CIMT values. The mean CIMT in Olt et al.[80] study was 0.74±0.22 mm. Malthesh et al.[73] reported a mean CIMT of 0.082 ± 0.033 cm in their study. In Rajput et al.[63] study the mean CIMT was found to be most in high fasting and postprandial triglyceride group (1.52+ 0.33mm), lowest in normal fasting and postprandial triglyceride group (0.68 + 0.12mm) and intermediate in normal fasting and high postprandial triglyceride group (1.17 + 0.17mm). The mean value of CIMT was 0.77±0.22 mm in Pramayuda et al.[72] study.

Table 21: Mean CIMT across studies:

Study	Mean CIMT
Current study	2.73 ± 3.56 mm
Rajput et al.[63]	1.52+ 0.33mm
Malthesh et al.[73]	0.082 ± 0.033 cm
Praliya et al.[61]	1.00 ± 0.489 mm
Salla et al.[88]	0.75 ± 0.07 mm

There was a weak positive correlation between postprandial blood sugar(mg/dl) and mean CIMT (millimeter) (R-value: 0.397, P-value: 0.001). The postprandial blood sugar values showed a positive correlation with CIMT values which was statistically significant (p-value < 0.001) in Nilofer Samed's study.[17] Bendwal et al.[82] found a postprandial blood sugar has a significant impact on the CIMT ($r = 0.442$, $p<0.05$).

We noted a moderate positive correlation between postprandial triglycerides and mean CIMT (r = 0.651, p-value: <0.001) in concordance with Bendwal et al. and Praliya et al.'s studies. Bendwal et al. reported a statistically significant difference in the CIMT with increasing with serum triglyceride value as the group of patients with high fasting and high post prandial triglyceride level had higher CIMT compared to those with normal fasting and normal post prandial triglyceride and normal fasting and high postprandial triglycerides (p<0.005).[82] While both fasting and postprandial triglycerides were significantly correlating with CIMT, the postprandial triglycerides (r=0.67) were more significantly correlating with CIMT as compared to the fasting levels (r=0.45).[82] Raja et al.[78] also found that postprandial triglyceride levels (r = 0. 859; p<0.001) correlated better with CIMT when compared to fasting triglyceride levels (r= 0.819). Their study showed a stronger correlation than ours. The correlation between post prandial post prandial triglyceride levels and the CIMT for diabetic patients was found to be positive and significant statistically (r=0.588, p-value < 0.05) in Praliya et al.[61] study. In Bushra et al.[17] study CIMT exhibited a significant increase in the groups with elevated postprandial triglyceride levels. There was the highest association between postprandial triglyceride and CIMT (r = 0.429) as compared to that between fasting triglyceride and CIMT (r = 0.258). Nilofer Samed reported the post prandial triglycerides values showed a positive correlation with CIMT values which was significant statistically (p-value <0.001). Rajput et al.[63] study showed both postprandial triglycerides and fasting triglycerides had a strong correlation with CIMT, the correlation coefficients being 0.879 and 0.764, respectively. In our study, most of the abnormal CIMT corresponds to a higher triglyceride levels except two subjects who have triglyceride levels within the normal range. This can be attributed to their age and the severity of hypertension.

There is a weak positive correlation between HBA1C (%) and mean CIMT (mm) (R-value 0.265, p-value: 0.033). Olt et al.[80] found no profound relationship between CIMT and HbA1c values in their study. On bivariate correlation analyses, the correlation between HbA1C with CIMT was significant ($p < 0.05$) with $r = 0.546$ as noted by Malthesh et al.[73] The HbA1c and CIMT as glycemic parameters in Pramayuda et al.[72] study seem to have a moderate positive correlation ($r=0.567$; $p<0.001$).

There was a weak negative correlation between HDL cholesterol (mg/dl) and mean CIMT millimeter (R-value: -0.120, p-value: 0.340), as noted in the following studies as well. This negative correlation is attributed to the fact that HDL cholesterol levels are generally inversely associated with the risk for developing atherosclerosis.[89] Praliya et al.[61] reported a negative and statistically significant connection between postprandial HDL and CIMT in diabetes individuals ($r=- 0.176$, p value=0.353). HDL-Cholesterol had a negative correlation with CIMT in Salla et al.[88] study. The postprandial HDL values did not show a statistically significant correlation with CIMT values (p value=0.551) in Nilofer Samed's study.[17] Bendwal et al.[82] also found no significant correlation between postprandial HDL and CIMT ($r= -0.241$ $p>0.05$).

There was a weak positive correlation between LDL cholesterol (mg/dl) and mean CIMT millimeter (R-value: 0.038, P value:0.765). Salla et al.[88] study showed a significant positive correlation ($p < 0.01$) between LDL-C and CIMT. Nilofer Samed found that even though the mean CIMT values increased in magnitude with the rise in LDL values, the postprandial LDL did not show a statistically significant correlation with CIMT value (p value=0.194).[17]

There is a weak positive correlation between total cholesterol (mg/dl) and mean CIMT millimeter (R-value: 0.381, P-value: 0.002). Compared to our study, Salla et al.'s study found

that total cholesterol and CIMT showed a statistically significant positive correlation (correlation coefficient 'r'= 0.606, $p < 0.01$) in the group with CIMT < 0.9 mm and the group with CIMT ≥ 0.9 mm also had significant positive correlation (correlation coefficient 'r'= 0.676, $p < 0.01$).[88] Bendwal et al.[82] reported no significant association between postprandial total cholesterol and CIMT ($p>0.05$).

Raja et al.[78] study came to the conclusion that postprandial hypertriglyceridemia can be labelled as an important independent marker of accelerated early atherosclerotic changes in diabetics. Despite normal fasting triglyceride levels, Bendwal et al.[82] concluded that postprandial hypertriglyceridemia may be an independent risk factor for early atherosclerosis in T2DM. Praliya et al.[87] discovered that CIMT levels are elevated in patients with type 2 diabetes mellitus, particularly those with abnormal fasting and postprandial dyslipidemia. Postprandial hypertriglyceridemia, according to Bushra et al.[86] is a crucial risk factor for the development of early atherosclerosis in type-2 diabetes mellitus patients. Olt et al.[80] demonstrated that while HbA1c values were not correlated with subclinical atherosclerosis, increased age is the major deciding factor for the macrovascular complications in diabetic patients. The postprandial cholesterol, post prandial triglycerides, post prandial plasma glucose values, the HbA1c values all correlated significantly with the CIMT values (p-value < 0.001) in Nilofer Samed's study.[17] Pramayuda et al.[72] reported a positive correlation between HbA1c level with CIMT in newly diagnosed T2DM patients. Our study found a moderate positive correlation between postprandial triglycerides and CIMT but a weak positive correlation between HBA1C, LDL cholesterol, total cholesterol, and CIMT. There is a weak negative correlation between HDL cholesterol and mean CIMT. Only postprandial triglyceride (R-value 0.651, p value: <0.001) has a better association with CIMT compared to other lipid parameters in our study.

SUMMARY

SUMMARY:

Diabetes is a crucial risk factor for atherosclerosis as well as its complications, including myocardial infarction (MI), stroke, and vascular death. Compared with subjects without diabetes, diabetes patients have a twofold higher risk of cardiovascular complications.[90] CIMT is an ultrasound biomarker of atherosclerosis, considered as a tool of subclinical organ damage. Measurement of CIMT by non-invasive B mode ultrasound can detect atherosclerosis at an early subclinical stage and help in the detection of asymptomatic cardiovascular disease. This is an observational study conducted on type-2 diabetes mellitus patients attending RL Jalappa Hospitals, Kolar, from January 2020 to January 2021 selected in a randomizer manner. We measured post prandial lipid profile, one-hour post prandial plasma sugar, and HbA1C and correlated with carotid intima-media thickness. Our study found a moderate positive correlation between postprandial triglycerides and CIMT but a weak positive correlation between HBA1C, LDL cholesterol, total cholesterol, and CIMT. There is a weak negative correlation between HDL cholesterol and mean CIMT. Only postprandial triglyceride (r-value 0.651, p-value: <0.001) has a better association with CIMT compared to other lipid parameters in our study.

CONCLUSIONS

CONCLUSIONS:

A total of 65 subjects with a mean age of 56.86 ± 11.89 years, meeting the inclusion and exclusion criteria, are included in the final analysis. The study population has a preponderance of male subjects, with 73.85% males and 26.15% females. The mean BMI was 29.73 ± 4.03kg/m2 in our study.The mean fasting blood pressure is 137.35 ± 48.82 mg/dl, the mean post prandial blood sugar is 267.78 ± 84.45 mg/dl, and the mean HBA1C is 7.67 ± 1.83(%), in our study.

The mean triglycerides are 259.98 ± 111.46 mg/dl, the mean HDL cholesterol is 39.19 ± 11.31 mg/dl, the mean LDL cholesterol is 99.97 ± 27.26 mg/dl, and the mean total cholesterol is 191.16 ± 37.23 mg/dl, in our study population. The mean right side CIMT is 2.72 ± 3.65 mm and on the left side is 2.75 ± 3.57 mm, giving a mean CIMT of 2.73 ± 3.56 mm in our study.

There was a weak positive correlation between Post prandial blood sugar(mg/dl) and mean CIMT (millimeter) **(r** value: 0.397, P value: 0.001).There is a moderate positive correlation between postprandial triglycerides and mean CIMT (r = 0.651, p value: <0.001).There was a weak positive correlation between HBA1C (%) and mean CIMT (millimeter) (R-value: 0.265, p value: 0.033).There was a weak negative correlation between HDL cholesterol (mg/dl) and mean CIMT millimeter (R-value: -0.120, p value: 0.340).

There was a weak positive correlation between LDL cholesterol (mg/dl) and mean CIMT millimeter (R-value: 0.038, p value:0.765). There was a weak positive correlation

between total cholesterol (mg/dl) and mean CIMT millimeter (R-value: 0.381, p value: 0.002).

Our study found a moderate positive correlation between postprandial triglycerides and CIMT but a weak positive correlation between HBA1C, LDL cholesterol, total cholesterol, and CIMT. There is a weak negative correlation between HDL cholesterol and mean CIMT. Only postprandial triglyceride (R-value 0.651, p-value: <0.001) has a better association with CIMT compared to other lipid parameters in our study.

LIMITATIONS AND RECOMMENDATIONS:

The study sample is very small, consisting of only 65 subjects. Increasing the study population would provide a better insight into the correlations. The CIMT measurements are subject to observer variation, due to which standardization is not possible. Duration of diabetes in not known. There is no specific cut-off for post-prandial triglyceride levels mentioned in the previous literature. Our study attempts to give a maximum acceptable triglyceride level as within 230mg/dl, but still, studies are needed to justify this recommendation. And postprandial dyslipidemia can be used as an early marker of atherosclerosis as it is correlating positively with CIMT

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ANNEXURES

STUDY PROFORMA

Name:

Age / Sex:

Residential Address:

Mobile No:

Case History:

Other known Illness:

BP: Pulse rate:
CVS-
RS-
P/A-
CNS-

Outcome Measures:

Post prandial lipid profile	
1 hour post prandial plasma glucose	
HbA1c	
Carotid Doppler measuring carotid intima media thickness	

Signature

PATIENT INFORMATION SHEET

Study Title: **Correlation of post prandial dyslipidemia and glycemic control with carotid intima media thickness in type 2 diabetes meliitus patients**

Study site: R.L Jalappa hospital, Tamaka, Kolar.

- **Aim**: To correlate fasting lipid profile, one hour postprandial plasma glucose and HbA1C with carotid intima media thickness in type 2 diabetes patients.

Blood samples will be taken for post prandial lipid profile and one hour post prandial plasma glucose and HbA1c.Carotid Doppler will be done and carotid intima media thickness will be measured.. This information is intended to give you the general background of the study. Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study we will collect information (as per proforma) from you or a person responsible for you or both. Relevant history will be taken. This information collected will be used only for publication. Principal investigator will be paying for post prandial lipid profile,post prandial blood glucose, glycated hemoglobin and carotid Doppler.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee. There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

For any further clarification you can contact the study investigator:

Dr. DEEPAK D

Mobile no: 9751312242

EMAIL:d.deepak2608@gmail.com

CONSENT FORM

I ---------- participant, hereby give consent to participate in the study entitled "**Correlation of postprandial lipids and glycemic control with carotid intima media thickness in patients with Type 2 Diabetes Mellitus"**

I have been explained that;

1. I would have to provide a blood sample for the study purpose.
2. I have to answer the questionnaires related to project.
3. I do not have to incur any additional expenditure on my inclusion into the study.
4. The data generated from my clinical examination and laboratory tests and other reports will be used in the study (which may be subsequently published) without revealing my identity in any manner.

I affirm that I have been given full information about the purpose of the study and the procedures involved and have been given ample opportunity to clarify my doubts in my mother tongue. In giving my consent, I have not faced any coercion. I have been informed that, notwithstanding this consent given, I can withdraw from the study at any stage.

For any further clarification you can contact the study investigator:

Dr. DEEPAK.D

Mobile no: 9751312242

Email: d.deepak2608@gmail.com

Signature of participant: Place:

Name of participant: Date:

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಟೈಪ್ 2 ಡಯಾಬಿಟಿಸ್ ಮೆಲಿಟಸ್ ರೋಗಿಗಳಲ್ಲಿ ಶೀರ್ಷಧಮನಿ ಇಂಟಿಮಾ ಮೀಡಿಯಾ ದಪ್ಪದೊಂದಿಗೆ ಪೋಸ್ಟ್ ಪ್ರಾಂಡಿಯಲ್ ಡಿಸ್ಲಿಪಿಡೆಮಿಯಾ ಮತ್ತು ಗ್ಲೈಸೆಮಿಕ್ ನಿಯಂತ್ರಣದ ಪರಸ್ಪರ ಸಂಬಂಧ

ಅಧ್ಯಯನ ಸ್ಥಳ: ಆರ್.ಎಲ್ ಜಲಪ್ಪ ಆಸ್ಪತ್ರೆ, ತಮಾಕಾ, ಕೋಲಾರ.

ಗುರಿ: ಉಪವಾಸದ ಲಿಪಿಡ್ ಪ್ರೊಫೈಲ್ ಅನ್ನು ಪರಸ್ಪರ ಸಂಬಂಧಿಸಲು, ಟೈಪ್ 2 ಡಯಾಬಿಟಿಸ್ ರೋಗಿಗಳಲ್ಲಿ ಶೀರ್ಷಧಮನಿ ಇಂಟಿಮಾ ಮೀಡಿಯಾ ದಪ್ಪದೊಂದಿಗೆ ಒಂದು ಗಂಟೆ ಪೋಸ್ಟ್‌ಪ್ರಾಂಡಿಯಲ್ ಪ್ಲಾಸ್ಮಾ ಗ್ಲೂಕೋಸ್ ಮತ್ತು ಎಚ್‌ಬಿಎ 1 ಸಿ.

ಪೋಸ್ಟ್ ಪ್ರಾಂಡಿಯಲ್ ಲಿಪಿಡ್ ಪ್ರೊಫೈಲ್‌ಗಾಗಿ ರಕ್ತದ ಮಾದರಿಗಳನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುವುದು ಮತ್ತು ಒಂದು ಗಂಟೆ ಪೋಸ್ಟ್ ಪ್ರಾಂಡಿಯಲ್ ಪ್ಲಾಸ್ಮಾ ಗ್ಲೂಕೋಸ್ ಮತ್ತು ಎಚ್‌ಬಿಎ 1 ಸಿ. ಕ್ಯಾರೊಟಿಡ್ ಡಾಪ್ಲರ್ ಅನ್ನು ಮಾಡಲಾಗುವುದು ಮತ್ತು ಶೀರ್ಷಧಮನಿ ಇಂಟಿಮಾ ಮೀಡಿಯಾ ದಪ್ಪವನ್ನು ಅಳೆಯಲಾಗುತ್ತದೆ .. ಈ ಮಾಹಿತಿಯು ನಿಮಗೆ ಅಧ್ಯಯನದ ಸಾಮಾನ್ಯ ಹಿನ್ನೆಲೆಯನ್ನು ನೀಡುತ್ತದೆ. ದಯವಿಟ್ಟು ಈ ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನಕ್ಕೆ ಸಂಬಂಧಿಸಿದಂತೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಒಪ್ಪಿದರೆ ನಾವು ನಿಮ್ಮಿಂದ ಅಥವಾ ನಿಮ್ಮ ಅಥವಾ ಇಬ್ಬರ ಜವಾಬ್ದಾರಿಯುತ ವ್ಯಕ್ತಿಯಿಂದ ಮಾಹಿತಿಯನ್ನು (ಪ್ರೊಫಾರ್ಮಾದ ಪ್ರಕಾರ) ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಸಂಬಂಧಿತ ಇತಿಹಾಸವನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುವುದು. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರಕಟಣೆಗೆ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ. ಪೋಸ್ಟ್ ಪ್ರಾಂಡಿಯಲ್ ಲಿಪಿಡ್ ಪ್ರೊಫೈಲ್, ಪೋಸ್ಟ್ ಪ್ರಾಂಡಿಯಲ್ ಬ್ಲಡ್ ಗ್ಲೂಕೋಸ್, ಗ್ಲೈಕೇಟೆಡ್ ಹಿಮೋಗ್ಲೋಬಿನ್ ಮತ್ತು ಶೀರ್ಷಧಮನಿ ಡಾಪ್ಲರ್‌ಗಾಗಿ ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ ಪಾವತಿಸಲಿದ್ದಾರೆ.

ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಲಾದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸುವುದಿಲ್ಲ. ನಿಮ್ಮ ಗುರುತು ಬಹಿರಂಗಗೊಳ್ಳುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಂಸ್ಥಿಕ ನೈತಿಕ ಸಮಿತಿಯು ಪರಿಶೀಲಿಸಿದೆ ಮತ್ತು ನೀವು ಸಾಂಸ್ಥಿಕ ನೈತಿಕ ಸಮಿತಿಯ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ. ಈ ಅಧ್ಯಯನವನ್ನು ಒಪ್ಪಿಕೊಳ್ಳಲು ಯಾವುದೇ ಬಲವಂತವಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನೀವು ಪಡೆಯುವ ಕಾಳಜಿ ಬದಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಕೊಂಡರೆ ಮಾತ್ರ ನೀವು ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆಗೆ ಸಹಿ / ಒದಗಿಸುವ ಅಗತ್ಯವಿದೆ.

ಯಾವುದೇ ಹೆಚ್ಚಿನ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ನೀವು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಯನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು:

ಡಾ. ದೀಪಾಕ್ ಡಿ

ಮೊಬೈಲ್ ಸಂಖ್ಯೆ: 9751312242

ಇಮೇಲ್: d.deepak2608@gmail.com

ಒಪ್ಪಿಗೆ ಪತ್ರ

ನಾನು ---------- ಭಾಗವಹಿಸುವವರು, ‘‘ಪೋಸ್ಟ್‌ಪ್ರ್ಯಾಂಡಿಯಲ್ ಲಿಪಿಡ್‌ಗಳ ಪರಸ್ಪರ ಸಂಬಂಧ’’ ಎಂಬ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಈ ಮೂಲಕ ಸಮ್ಮತಿ ನೀಡುತ್ತೇನೆ ಮತ್ತು ಟೈಪ್ 2 ಡಯಾಬಿಟಿಸ್ ಮೆಲ್ಲಿಟಸ್ ರೋಗಿಗಳಲ್ಲಿ ಶೀರ್ಷಧಮನಿ ಇಂಟಿಮಾ ಮೀಡಿಯಾ ದಪ್ಪದೊಂದಿಗೆ ಗ್ಲೈಸೆಮಿಕ್ ನಿಯಂತ್ರಣ ’’

ನನಗೆ ಅದನ್ನು ವಿವರಿಸಲಾಗಿದೆ;

1. ಅಧ್ಯಯನದ ಉದ್ದೇಶಕ್ಕಾಗಿ ನಾನು ರಕ್ತದ ಮಾದರಿಯನ್ನು ಒದಗಿಸಬೇಕಾಗಿತ್ತು.

2. ಯೋಜನೆಗೆ ಸಂಬಂಧಿಸಿದ ಪ್ರಶ್ನಾವಳಿಗಳಿಗೆ ನಾನು ಉತ್ತರಿಸಬೇಕಾಗಿದೆ.

3. ನನ್ನ ಅಧ್ಯಯನಕ್ಕೆ ಸೇರ್ಪಡೆಗೊಳ್ಳಲು ನಾನು ಯಾವುದೇ ಹೆಚ್ಚುವರಿ ಖರ್ಚು ಮಾಡಬೇಕಾಗಿಲ್ಲ.

4. ನನ್ನ ಕ್ಲಿನಿಕಲ್ ಪರೀಕ್ಷೆ ಮತ್ತು ಪ್ರಯೋಗಾಲಯ ಪರೀಕ್ಷೆಗಳು ಮತ್ತು ಇತರ ವರದಿಗಳಿಂದ ಉತ್ಪತ್ತಿಯಾಗುವ ಡೇಟಾವನ್ನು ಯಾವುದೇ ರೀತಿಯಲ್ಲಿ ನನ್ನ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸದೆ ಅಧ್ಯಯನದಲ್ಲಿ ಬಳಸಲಾಗುತ್ತದೆ (ಅದನ್ನು ನಂತರ ಪ್ರಕಟಿಸಬಹುದು).

ಅಧ್ಯಯನದ ಉದ್ದೇಶ ಮತ್ತು ಒಳಗೊಂಡಿರುವ ಕಾರ್ಯವಿಧಾನಗಳ ಬಗ್ಗೆ ನನಗೆ ಸಂಪೂರ್ಣ ಮಾಹಿತಿ ನೀಡಲಾಗಿದೆ ಮತ್ತು ನನ್ನ ಮಾತೃಭಾಷೆಯಲ್ಲಿ ನನ್ನ ಅನುಮಾನಗಳನ್ನು ಸ್ಪಷ್ಟಪಡಿಸಲು ಸಾಕಷ್ಟು ಅವಕಾಶವನ್ನು ನೀಡಲಾಗಿದೆ ಎಂದು ನಾನು ದೃ irm ಪಡಿಸುತ್ತೇನೆ. ನನ್ನ ಒಪ್ಪಿಗೆ ನೀಡುವಲ್ಲಿ, ನಾನು ಯಾವುದೇ ಬಲಾತ್ಕಾರವನ್ನು ಎದುರಿಸಲಿಲ್ಲ. ಈ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡಿದ್ದರೂ, ನಾನು ಯಾವುದೇ ಹಂತದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು ಎಂದು ನನಗೆ ತಿಳಿಸಲಾಗಿದೆ.

ಯಾವುದೇ ಹೆಚ್ಚಿನ ಸ್ಪಷ್ಟೀಕರಣಕ್ಕಾಗಿ ನೀವು ಅಧ್ಯಯನ ತನಿಖಾಧಿಕಾರಿಯನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು:

ಡಾ. ದೀಪಾಕ್.ಡಿ

ಮೊಬೈಲ್ ಸಂಖ್ಯೆ: 9751312242

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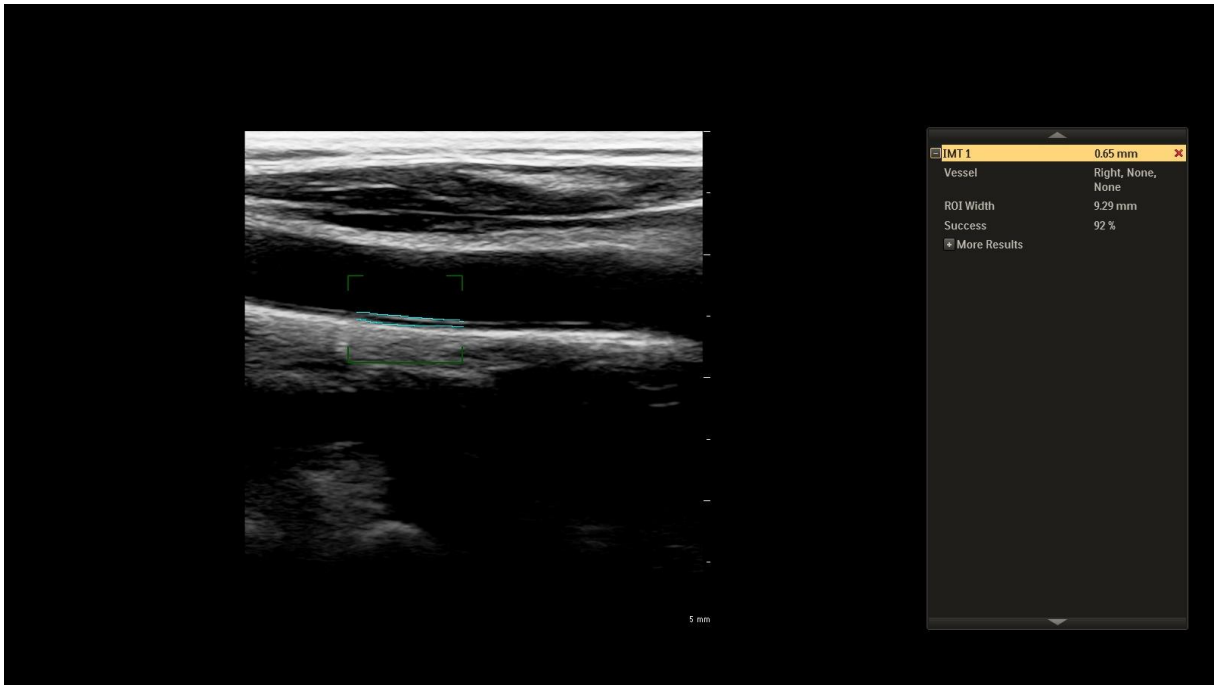
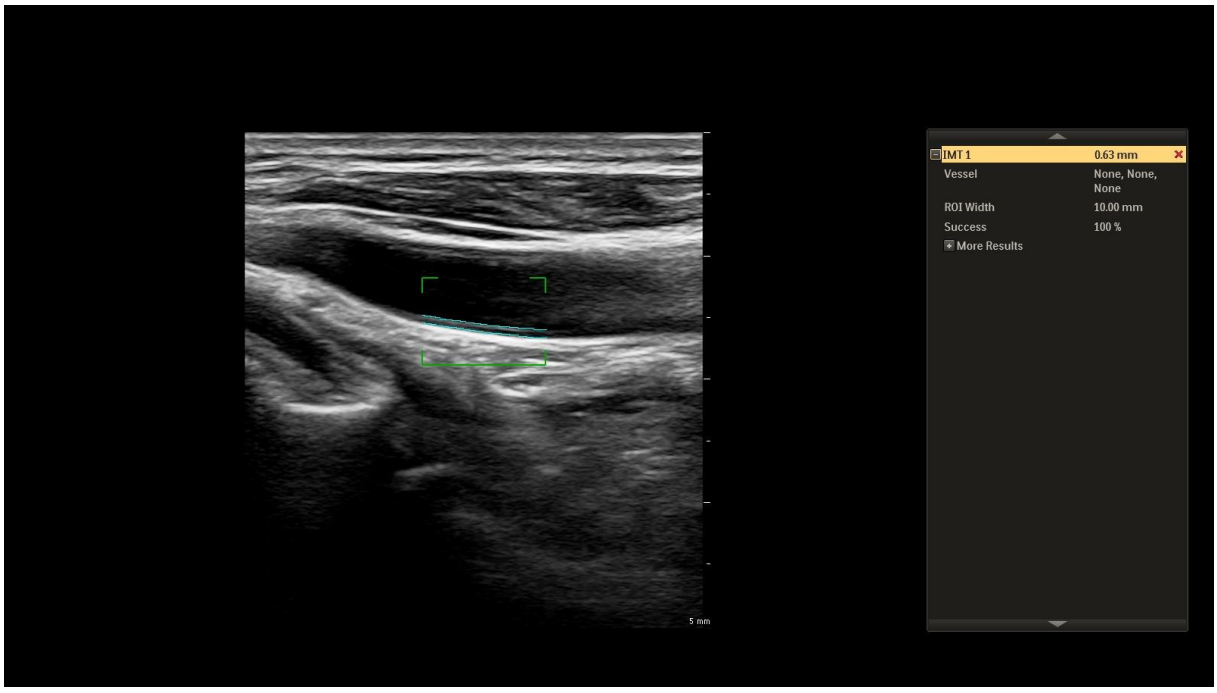
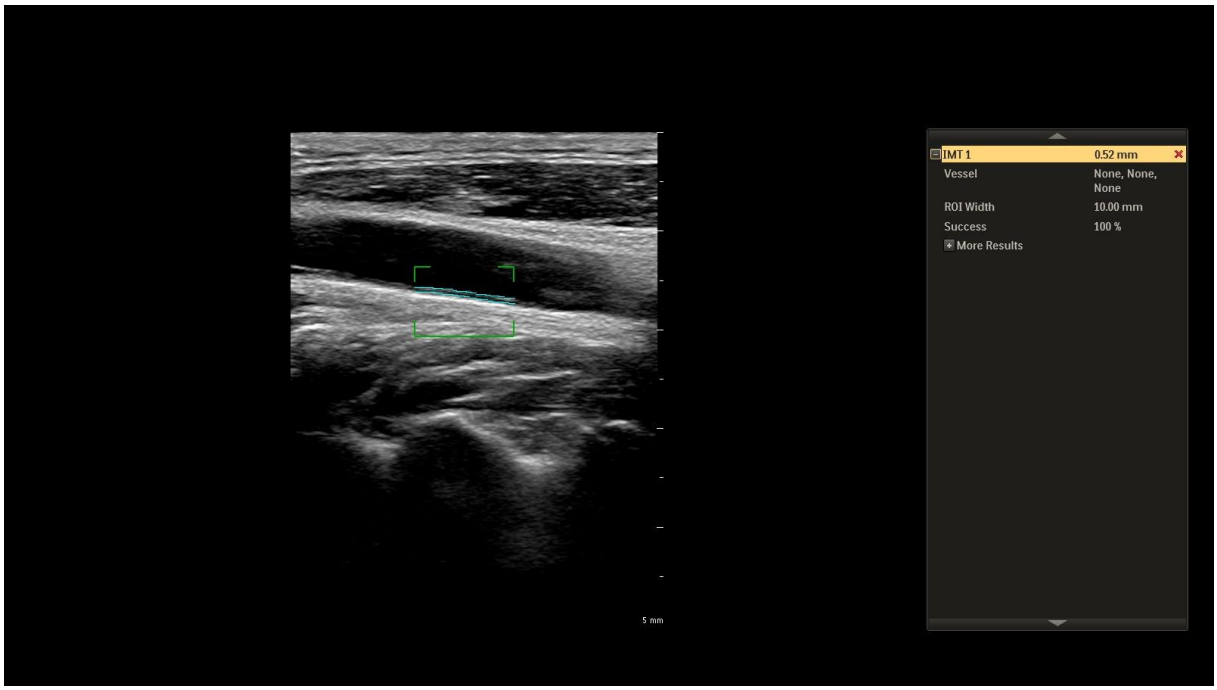
ಭಾಗವಹಿಸುವವರ ಸಹಿ: ಸ್ಥಳ:

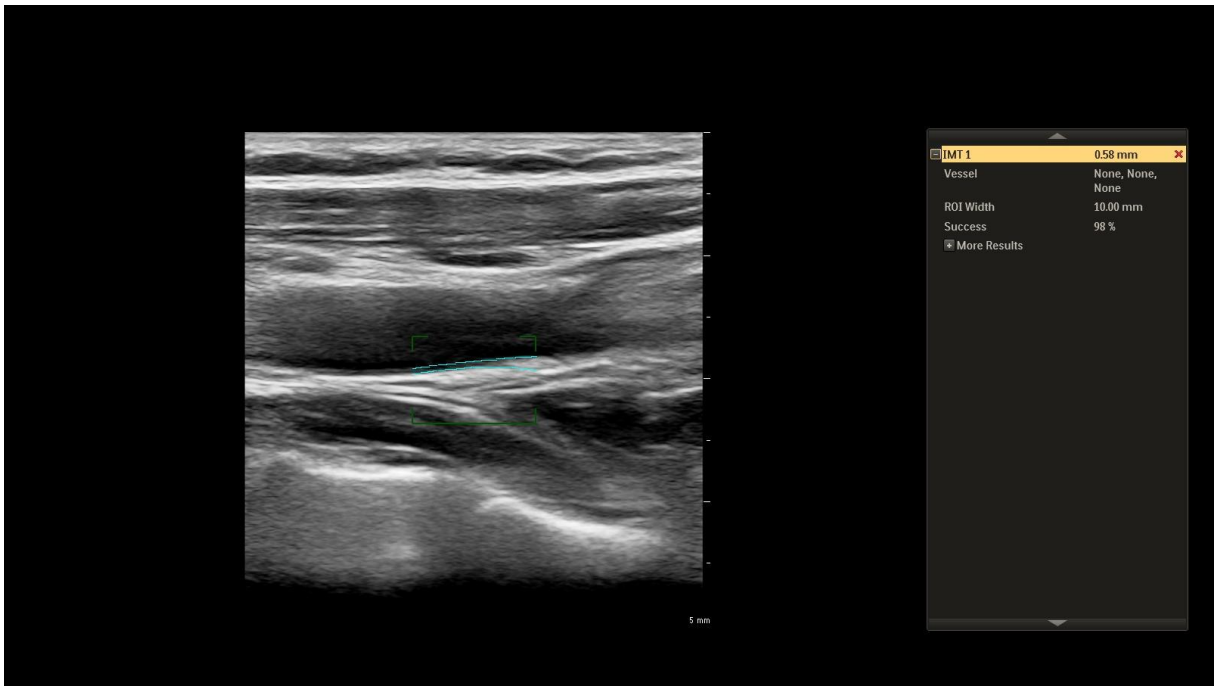
ಭಾಗವಹಿಸುವವರ ಹೆಸರು: ದಿನಾಂಕ:

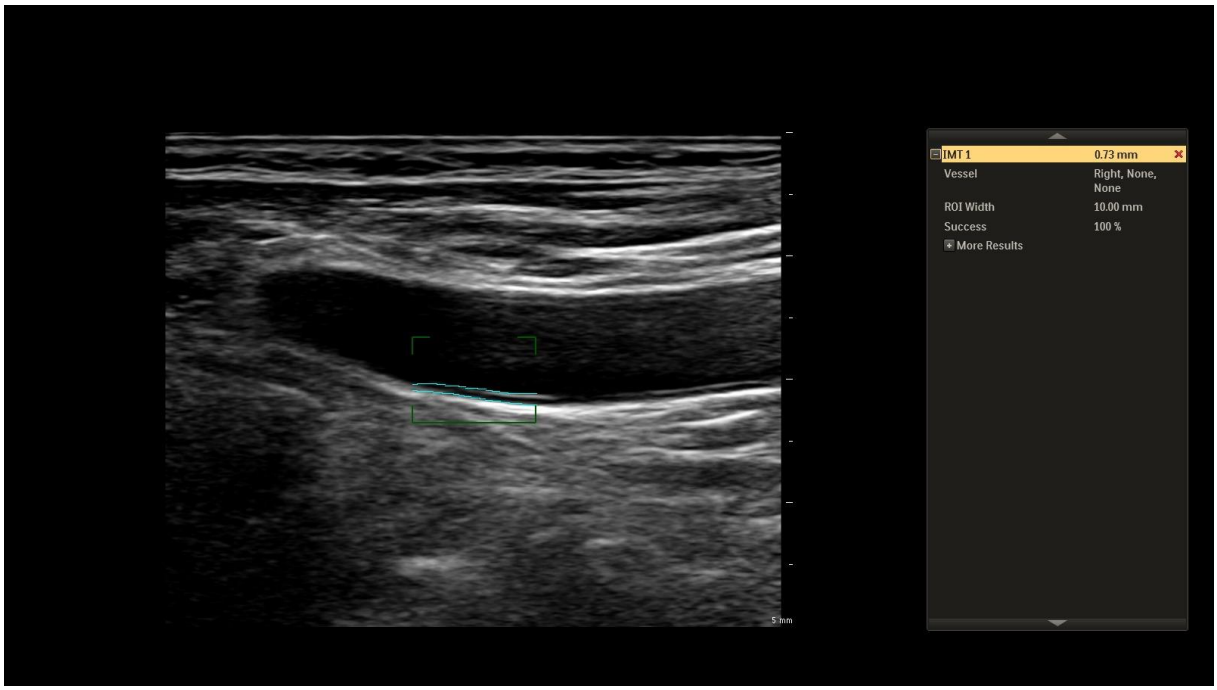
CLINICAL IMAGES

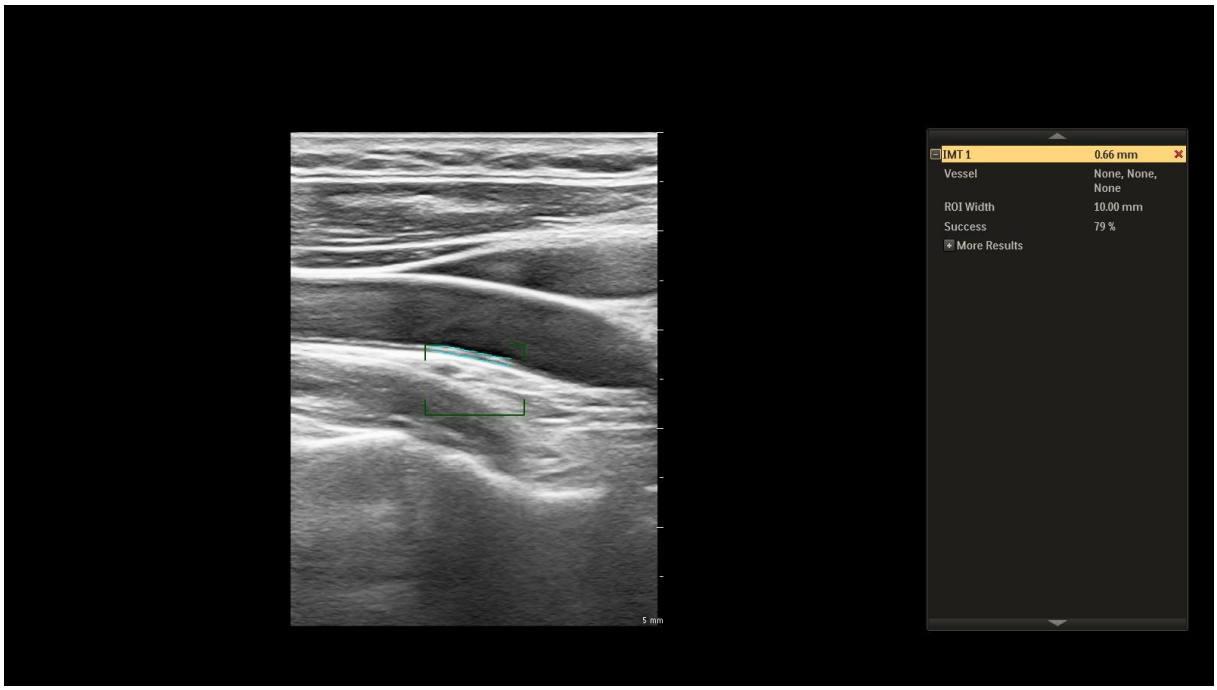





5 mm
IMT 1
0.52 mm
Vessel
None, None, None
ROI Width
10.00 mm
Success
100 %
More Results


5 mm
IMT 1
0.58 mm
Vessel
None, None, None
ROI Width
10.00 mm
Success
98 %
More Results


IMT 1
0.73 mm
Vessel
Right, None, None
ROI Width
10.00 mm
Success
100 %
More Results
5 mm


IMT 1
0.66 mm
Vessel
None, None, None
ROI Width
10.00 mm
Success
79 %
More Results
5 mm

MASTER SHEET

S.NO	UHID NO	Gender	Age(in years)	Diagnosis	Hypertension	BMI	Smoker	Alcohol	Pulse	Systolic Blood pressure	Diastolic blood pressure	CVS	RS	P/A	CNS
1	898807	Male	67	UTI	Yes	24	No	No	80	120	70	S1S2+.	B/LAE+	SOFT	NFND
2	900893	Female	60	DKA	Yes	32	No	No	90	130	80	S1S2+.	B/LAE+	SOFT	NFND
3	900957	Male	70	UTI	Yes	22	Yes	Yes	82	120	80	S1S2+.	B/LAE+	SOFT	NFND
4	900951	Female	53	BRONCHOPNEUMONIA	No	26	No	No	80	90	40	S1S2+.	B/LAE+	SOFT	NFND
5	858600	Male	51	urosepsis	Yes	36	No	No	72	130	80	S1S2+.	B/LAE+	SOFT	NFND
6	882128	Female	40	LEPTOSPIROSIS	Yes	34	No	No	71	130	80	S1S2+.	B/LAE+	SOFT	NFND
7	882980	Female	40	DENGUE FEVER	Yes	28	No	No	60	80	60	S1S2+.	B/LAE+	SOFT	NFND
8	887854	Male	68	BPH	No	30	Yes	No	82	130	90	S1S2+.	B/LAE+	SOFT	NFND
9	887879	Male	50	RENAL CALCULI	Yes	35	Yes	No	86	120	80	S1S2+.	B/LAE+	SOFT	NFND
10	888515	Male	44	DENGUE FEVER	Yes	28	No	No	72	170	100	S1S2+.	B/LAE+	SOFT	NFND
11	890110	Male	59	DENGUE FEVER	No	39	No	No	82	120	80	S1S2+.	B/LAE+	SOFT	NFND
12	890709	Male	45	RIGHT LOWER LOBE PNEUMONIA	No	33	No	No	86	120	80	S1S2+.	B/LAE+	SOFT	NFND
13	891458	Male	47	RIGHT LOWER LIMB CELLULITIS	Yes	28	No	Yes	82	120	80	S1S2+.	B/LAE+	SOFT	NFND
14	892355	Male	58	LEPTOSPIROSIS	No	36	No	No	64	130	80	S1S2+.	B/LAE+	SOFT	NFND
15	892798	Male	76	BPH	No	29	Yes	No	92	140	90	S1S2+.	B/LAE+	SOFT	NFND
16	888482	Female	55	UTI	No	28	No	No	80	120	80	S1S2+.	B/LAE+	SOFT	NFND
17	889963	Female	63	DKA	Yes	36	No	No	90	110	70	S1S2+.	B/LAE+	SOFT	NFND
18	893111	Male	40	DENGUE FEVER	No	28	No	No	92	110	70	S1S2+.	B/LAE+	SOFT	NFND
19	894485	Male	50	VIRAL FEVER	Yes	32	Yes	No	69	110	90	S1S2+.	B/LAE+	SOFT	NFND
20	894616	Male	73	urosepsis	Yes	28	Yes	No	70	150	100	S1S2+.	B/LAE+	SOFT	NFND
21	895212	Male	63	METABOLIC ENCEPHALOPATHY	Yes	32	No	No	90	100	70	S1S2+.	B/LAE+	SOFT	NFND
22	895296	Male	70	VIRAL FEVER	Yes	28	Yes	No	92	140	90	S1S2+.	B/LAE+	SOFT	NFND
23	895325	Female	61	urosepsis	Yes	35	No	No	100	130	80	S1S2+.	B/LAE+	SOFT	NFND
24	896057	Male	40	DKA	No	35	No	No	76	110	70	S1S2+.	B/LAE+	SOFT	NFND
25	895617	Male	51	PULMONARY TUBERCULOSIS	No	28	Yes	No	75	100	80	S1S2+.	B/LAE+	SOFT	NFND
26	877296	Male	42	VIRAL FEVER	No	36	No	No	86	110	70	S1S2+.	B/LAE+	SOFT	NFND
27	876818	Male	52	COPD	No	24	No	No	76	130	80	S1S2+.	B/LAE+	SOFT	NFND
28	867889	Female	55	IRON DEFICIENCY ANEMIA	No	32	Yes	No	80	130	90	S1S2+.	B/LAE+	SOFT	NFND
29	867559	Female	72	COPD	No	34	No	No	84	130	80	S1S2+.	B/LAE+	SOFT	NFND
30	866715	Male	58	COPD	Yes	29	No	No	86	100	70	S1S2+.	B/LAE+	SOFT	NFND
31	863763	Male	50	RICKETSSIAL FEVER	Yes	24	Yes	No	72	120	80	S1S2+.	B/LAE+	SOFT	NFND

32	879196	Male	60	HTN URGENCY	Yes	38	Yes	No	80	110	70	S1S2+.	B/LAE+	SOFT	NFND
33	876795	Male	60	COPD	Yes	32	No	No	80	160	120	S1S2+.	B/LAE+	SOFT	NFND
34	875645	Male	65	DENGUE FEVER	No	23	No	No	65	140	80	S1S2+.	B/LAE+	SOFT	NFND
35	874091	Male	60	BPH	No	35	Yes	No	70	110	70	S1S2+.	B/LAE+	SOFT	NFND
36	854592	Male	68	PYELONEPHRITIS	No	27	No	No	82	110	70	S1S2+.	B/LAE+	SOFT	NFND
37	872745	Male	70	BRONCHOPNEUMONIA	Yes	27	No	No	84	110	70	S1S2+.	B/LAE+	SOFT	NFND
38	872269	Male	44	RIGHT LOWER LOBE PNEUMONIA	No	29	Yes	No	65	130	80	S1S2+.	B/LAE+	SOFT	NFND
39	870556	Male	42	COPD	Yes	32	No	No	72	120	60	S1S2+.	B/LAE+	SOFT	NFND
40	869645	Male	64	LEFT LOWER LIMB CELLULITIS	Yes	30	No	No	86	140	70	S1S2+.	B/LAE+	SOFT	NFND
41	869611	Male	70	urosepsis	Yes	35	No	No	76	140	80	S1S2+.	B/LAE+	SOFT	NFND
42	869575	Male	35	VIRAL FEVER	No	28	No	No	80	140	90	S1S2+.	B/LAE+	SOFT	NFND
43	871451	Male	55	COPD	Yes	36	No	No	86	120	80	S1S2+.	B/LAE+	SOFT	NFND
44	936998	Male	60	VIRAL FEVER	No	26	No	No	89	130	90	S1S2+.	B/LAE+	SOFT	NFND
45	932700	Female	53	UTI	No	32	No	No	88	110	70	S1S2+.	B/LAE+	SOFT	NFND
46	930785	Male	55	MENINGOENCEPHALITIS	No	27	No	No	80	140	80	S1S2+.	B/LAE+	SOFT	NFND
47	937262	Male	34	DENGUE FEVER	No	25	No	No	60	110	70	S1S2+.	B/LAE+	SOFT	NFND
48	939054	Female	67	DKA	No	27	No	No	120	80	60	S1S2+.	B/LAE+	SOFT	NFND
49	938681	Male	59	HTN URGENCY	Yes	30	No	No	82	110	70	S1S2+.	B/LAE+	SOFT	NFND
50	937916	Male	54	COPD	No	29	Yes	Yes	72	150	110	S1S2+.	B/LAE+	SOFT	NFND
51	940429	Male	84	ACUTE GASTRO ENTERITIS	No	26	No	No	82	130	80	S1S2+.	B/LAE+	SOFT	NFND
52	932025	Male	48	UTI	Yes	29	Yes	Yes	86	110	60	S1S2+.	B/LAE+	SOFT	NFND
53	932788	Female	90	ACUTE GASTRO ENTERITIS	No	32	No	No	82	130	80	S1S2+.	B/LAE+	SOFT	NFND
54	929396	Female	60	COPD	No	28	No	No	78	160	90	S1S2+.	B/LAE+	SOFT	NFND
55	922351	Female	54	DENGUE FEVER	No	26	No	No	86	110	70	S1S2+.	B/LAE+	SOFT	NFND
56	921510	Female	35	PULMONARY TUBERCULOSIS	Yes	29	No	No	90	130	70	S1S2+.	B/LAE+	SOFT	NFND
57	928271	Male	50	RICKETSSIAL FEVER	No	25	No	No	80	110	70	S1S2+.	B/LAE+	SOFT	NFND
58	925166	Male	70	COPD	No	30	No	No	68	100	70	S1S2+.	B/LAE+	SOFT	NFND
59	924027	Male	48	COPD	Yes	33	No	No	80	110	70	S1S2+.	B/LAE+	SOFT	NFND
60	921551	Male	74	METABOLIC ENCEPHALOPATHY	No	27	Yes	No	80	110	70	S1S2+.	B/LAE+	SOFT	NFND
61	921316	Male	44	DENGUE FEVER	Yes	23	Yes	No	90	110	70	S1S2+.	B/LAE+	SOFT	NFND
62	919892	Female	52	VIRAL FEVER	No	27	No	No	80	110	70	S1S2+.	B/LAE+	SOFT	NFND
63	914669	Male	68	urosepsis	Yes	26	No	No	76	130	80	S1S2+.	B/LAE+	SOFT	NFND
64	913586	Male	60	HTN URGENCY	Yes	30	No	No	72	110	70	S1S2+.	B/LAE+	SOFT	NFND
65	875723	Female	61	UTI	No	26	No	No	80	120	80	S1S2+.	B/LAE+	SOFT	NFND

S.NO	BI urea	Sr. Creatinine	Sodium	Potassium	Fasting blood pressure	Post prandial blood sugar (mg/dl)	HBA1C (%)	Triglycerides (mg/dl)	HDL Cholesterol (mg/dl)	LDL Cholesterol (mg/dl)	Total Cholesterol (mg/dl)	RIGHT CIMT	LEFT CIMT	MEAN CIMT (in millimeter)
1	44	1.20	135.0	3.40	140	289	6.90	197	35.8	98	173.2	0.60	0.70	0.65
2	34	1.20	136.0	4.10	204	332	14.40	533	43.0	100	249.6	14.00	12.00	13.00
3	22	0.60	132.0	4.50	160	330	11.30	253	65.0	163	278.6	0.90	1.00	0.95
4	51	1.00	141.0	3.70	137	408	10.10	180	34.0	110	180.0	0.50	0.80	0.65
5	34	1.20	136.0	3.60	172	228	9.30	257	37.0	130	218.4	0.40	0.52	0.46
6	35	0.90	133.0	4.10	133	368	7.80	336	35.0	150	252.2	5.00	5.00	5.00
7	40	1.20	139.0	4.60	206	401	8.30	260	34.0	80	166.0	0.50	0.50	0.50
8	22	0.90	137.0	4.60	98	281	9.20	297	29.0	116	204.4	0.70	0.60	0.65
9	26	0.60	136.0	4.60	126	271	8.20	191	37.0	114	189.2	0.60	0.70	0.65
10	29	1.20	131.0	3.50	106	189	9.60	155	42.4	90	163.4	5.00	6.00	5.50
11	35	1.00	136.0	4.20	199	260	10.10	350	45.0	92	207.0	0.72	0.89	0.81
12	40	1.20	136.0	4.10	129	164	5.20	148	39.0	99	167.6	0.71	0.45	0.58
13	36	1.20	135.0	3.90	101	152	6.70	271	45.0	175	274.2	0.50	0.70	0.60
14	16	0.90	127.0	3.90	190	170	5.10	149	50.0	85	164.8	0.80	0.80	0.80
15	22	1.00	140.0	3.50	110	305	8.60	378	37.0	105	217.6	11.00	13.00	12.00
16	20	0.40	138.0	4.20	160	249	11.90	240	26.3	98	172.3	0.80	0.70	0.75
17	18	9.00	132.0	4.90	88	156	5.10	306	56.1	86	203.3	0.80	0.70	0.75
18	22	0.90	138.3	4.60	107	242	7.20	134	57.0	110	193.8	0.70	0.80	0.75
19	16	0.80	140.0	3.70	106	94	5.40	141	64.0	193	285.2	0.70	0.80	0.75
20	18	0.70	134.0	3.40	67	192	6.10	240	26.3	56	130.3	1.10	1.00	1.05
21	10	0.96	139.0	4.00	105	178	7.00	222	60.0	45	149.4	0.79	0.82	0.81
22	31	1.08	135.0	3.80	103	350	10.10	169	38.0	108	179.8	1.10	0.90	1.00
23	28	1.35	130.0	5.45	241	384	11.00	193	41.5	53	133.1	0.70	0.50	0.60
24	17	1.07	130.0	3.70	180	341	6.20	260	38.0	65	155.0	0.80	1.10	0.95
25	18	0.40	138.0	4.60	114	286	7.30	348	56.0	75	200.6	0.60	1.20	0.90
26	20	0.40	139.0	4.10	160	375	8.10	266	32.0	57	142.2	0.90	0.60	0.75
27	18	1.00	136.0	4.70	102	208	6.45	160	36.0	120	188.0	0.59	0.45	0.52
28	30	0.90	141.0	3.60	109	266	7.50	150	36.0	89	155.0	0.80	0.80	0.80
29	20	0.90	136.0	4.80	142	220	8.20	272	38.2	108	200.6	6.00	1.00	3.50
30	18	0.60	138.0	4.70	126	237	9.00	168	28.0	110	171.6	0.50	0.50	0.50
31	22	1.10	142.0	3.40	111	193	5.80	484	29.3	95	221.1	0.80	0.80	0.80
32	22	0.90	144.0	4.40	140	301	7.20	229	26.3	110	182.1	6.10	6.30	6.20

33	18	0.24	136.0	4.27	242	373	8.00	471	34.5	96	224.7	17.00	12.00	14.50
34	14	0.20	134.0	3.20	127	408	9.60	581	26.0	110	252.2	11.00	12.00	11.50
35	38	1.00	141.0	3.50	87	225	9.20	148	40.0	95	164.6	5.00	5.20	5.10
36	2	0.83	134.0	3.90	149	351	6.00	360	50.0	96	218.0	7.30	12.00	9.65
37	12	0.50	136.0	4.10	141	229	6.20	291	37.9	120	216.1	0.40	0.40	0.40
38	12	1.08	134.0	4.20	111	182	5.40	138	36.0	69	132.6	0.41	0.44	0.43
39	17	0.90	139.0	3.50	240	400	8.60	350	35.0	86	191.0	0.47	0.55	0.51
40	18	0.60	147.0	4.10	99	230	7.30	148	39.4	96	165.0	0.90	1.60	1.25
41	24	1.00	139.0	4.30	126	190	6.10	280	59.0	96	211.0	0.84	0.69	0.77
42	18	0.90	134.0	4.50	130	233	7.00	158	26.0	120	177.6	0.80	0.80	0.80
43	21	0.60	145.0	4.80	87	237	6.60	160	45.0	110	187.0	0.45	0.45	0.45
44	19	0.90	136.0	3.90	242	460	10.90	384	38.2	96	211.0	6.80	6.30	6.55
45	17	1.07	132.0	4.00	285	482	8.70	280	35.0	98	189.0	4.00	4.80	4.40
46	14	0.70	128.0	3.40	204	308	7.20	355	28.7	105	204.7	6.30	6.80	6.55
47	30	1.00	138.0	4.90	54	203	6.70	189	24.0	130	191.8	0.60	0.70	0.65
48	18	1.00	137.0	3.40	155	256	6.80	454	30.7	120	241.5	6.30	6.90	6.60
49	23	1.04	133.0	3.80	92	238	8.00	158	34.8	95	161.4	0.70	1.40	1.05
50	14	1.00	136.0	3.50	125	302	6.80	240	28.2	102	178.2	7.00	6.80	6.90
51	26	1.00	135.0	3.70	103	214	5.60	380	40.0	103	219.0	7.00	7.10	7.05
52	28	1.20	130.0	5.48	101	186	6.20	348	50.0	98	217.6	7.60	7.20	7.40
53	15	0.90	136.0	3.60	135	228	6.70	278	38.0	110	203.6	0.73	0.81	0.77
54	12	0.80	136.0	3.90	198	273	7.80	355	28.7	102	201.7	0.70	0.90	0.80
55	17	1.00	136.0	3.50	105	178	6.40	213	35.3	89	166.9	0.65	0.57	0.61
56	37	0.61	143.0	3.40	93	304	6.50	571	38.3	85	237.5	6.00	6.00	6.00
57	17	1.10	135.0	3.00	110	179	7.20	170	62.0	110	206.0	0.73	0.99	0.86
58	18	0.40	136.0	4.00	69	224	6.70	103	43.8	104	168.4	0.50	0.60	0.55
59	41	1.20	138.0	4.20	202	299	7.00	221	29.6	98	171.8	0.45	0.50	0.48
60	18	0.40	145.0	4.70	133	317	6.00	158	79.0	98	208.6	0.90	0.90	0.90
61	20	1.08	128.0	4.30	97	174	5.50	224	32.5	92	169.3	0.60	0.60	0.60
62	20	1.00	134.0	4.70	125	240	7.00	171	34.5	11	79.7	0.60	0.70	0.65
63	28	0.87	141.0	4.10	188	447	9.20	262	31.9	86	170.3	7.00	8.00	7.50
64	26	0.90	135.0	3.90	97	210	6.60	205	35.0	89	165.0	0.70	0.80	0.75
65	30	0.70	133.0	3.20	104	206	8.65	158	23.1	98	152.7	0.60	0.60	0.60