PRESENCE OF ASYMPTOMATIC CORONARY ARTERY DISEASE IN TYPE 2 DIABETES MELLITUS AND RISK FACTORS

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Dr. ABBAGONI RAHUL GOUD

ABBREVIATIONS

CAC – Coronary Artery Calcium

CAD – Coronary Artery Disease

CCTA – Coronary Computed Tomography Angiography

CHD – Coronary Heart Disease

CRP – C-Reactive Protein

CVD – Cardiovascular Disease

DM – Diabetes Mellitus

DM2 – Type 2 Diabetes Mellitus

ECG – Electrocardiogram

GBD – Global Burden of Disease

HLA – Human Leukocyte Antigen

IHD – Ischemic Heart Disease

MACE – Major Adverse Cardiovascular Events

MOD – Maturity-Onset Diabetes of the Young

MPI – Myocardial Perfusion Imaging

NIDDM – Non-Insulin Dependent Diabetes Mellitus

Ox-LDL – Oxidized Low-Density Lipoprotein

SAA – Serum Amyloid A

SIHD – Stable Ischemic Heart Disease

SSG – Split-Thickness Skin Grafting

T2DM – Type 2 Diabetes Mellitus

WHO – World Health Organization

ABSTRACT

Background: "Coronary artery disease (CAD) remains a significant cause of morbidity and mortality, particularly among individuals with type 2 diabetes mellitus (T2DM). Diabetic patients are at a higher risk of developing CAD, often presenting asymptomatically. Despite the global burden of CAD, limited studies exist on its prevalence among asymptomatic T2DM patients in the Indian population. This study aims to determine the proportion of asymptomatic CAD in patients with T2DM and identify associated risk factors.

Materials and Methods: A cross-sectional study was conducted at BLDE (DU) Shri B.M Patil Medical College Hospital and Research Centre between May 2023 and December 2024. 105 T2DM patients (aged 40-60 years, with diabetes for over five years) were included. Data collection involved detailed history, clinical examination, BMI assessment, and investigations such as electrocardiography (ECG), 2D echocardiography, lipid profile, treadmill test (TMT), and coronary angiography. Statistical analysis included descriptive and inferential statistics, with a p-value of <0.05 considered significant."

Results: CAD was detected in 53.3% of patients. The mean patient age was 52.46 ± 5.63 years, with a slight male predominance (60%). Smoking was significantly higher among CAD-positive patients (82.1%) compared to CAD-negative patients (55.2%) ($p<0.05$). CAD-positive individuals had significantly higher mean BMI, HbA1c, total cholesterol, triglycerides, and LDL cholesterol, along with lower HDL levels, indicating dyslipidemia as a critical risk factor. Triple-vessel disease (TVD) was the most common pattern, affecting 22.9% of cases.

Conclusion: The study highlights the substantial burden of asymptomatic CAD among T2DM patients, emphasising the need for early screening and intervention. Strict glycemic control, lifestyle modifications, and aggressive management of dyslipidemia and smoking cessation should be prioritised to mitigate cardiovascular risks. Future research should focus on cost-effective screening tools to enhance early detection and improve patient outcomes.

Keywords: Coronary artery disease, Type 2 diabetes mellitus, Asymptomatic CAD, Dyslipidemia, Risk factors, Screening.

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INTRODUCTION

Coronary artery disease continues to be one of the most considerable demands on healthcare resources and is a "significant cause of morbidity and mortality in individuals with diabetes mellitus[1]. Despite a steady decline in age-specific mortality from CAD over the past several decades, IHD is now the leading cause of death worldwide, and the rate of CAD is expected to only accelerate in the coming decades; contributory factors include the ageing of the population, increases in the worldwide prevalence of obesity and type 2 diabetes, and a rise in cardiovascular risk factors. According to the World Health Organization, global fatalities due to coronary artery disease (CAD) were projected to increase from 7.5 million in 2005 to 11.2 million by 2020. In India, the prevalence of coronary heart disease (CHD) has shown a significant rise over the past six decades, ranging from 1% to 9%-10% in urban populations and <1% to 4%-6% in rural populations. When stricter diagnostic criteria (clinical ± Q waves) are applied, the prevalence in rural populations is estimated at 1%-2% and 2%-4% in urban populations."[1]

"Patients with diabetes mellitus (DM) are at a higher risk of developing coronary artery disease (CAD) than non-DM patients because CAD develops slowly and sporadically takes a severe clinical course.[2]

Compared to non-diabetics, people with diabetes also have a more significant incidence of multi-vessel CAD (66 versus 46 per cent) and more diseased vessels.[3]

Myocardial ischemia is found in about 22% of asymptomatic patients with diabetes mellitus (DM). However, a large proportion of these individuals are low-risk and do not have myocardial ischemia, emphasising the diverse nature of this population, which consists of both those with and without coronary artery disease (CAD).[4] Coronary artery disease is the

primary contributor to both direct and indirect costs associated with diabetes, in addition to being a significant cause of morbidity and mortality.[5] Individuals with DM II have increased risk, faster progression and greater extent of CAD compared to non-diabetics.[6]

Cardiovascular death is the most common cause of death in diabetes, and outcomes of coronary artery disease are worst in diabetics."[7]

The Multiple Risk Factor Intervention Trial demonstrated that over 12 years, cardiovascular disease accounted for 9.7% of deaths in diabetic men, compared to 2.6% in non-diabetic men. This difference remained significant even after adjusting for age, cholesterol levels, systolic blood pressure, and smoking history.[7]

There is limited literature available on the Indian population. Hence, this study was designed to find the proportion of asymptomatic coronary artery disease in type 2 diabetes mellitus and risk factors.

REVIEW OF LITERATURE

"For over half a century, therapies to lower the risk of heart attack and stroke in people who do not have established heart disease have mostly been performed using a "two-step procedure" based on absolute risk. First, physicians stratified patients who are candidates for primary prevention into 'lower-, intermediate-, and higher risk subgroups', typically calculated over a 10-year time frame, using a global risk–estimating algorithm such as the 'Framingham risk score,' 'the Reynolds risk score,' or the European Systematic Coronary Risk Evaluation.

The leading cause of Stable Ischemic Heart Disease is the gradual constriction or occlusion of one or more Epicardial Coronary Arteries by an atheromatous plaque (SIHD). According to data from the Framingham Heart Study, the lifetime risk of developing symptomatic CAD after the age of 40 is roughly 49 per cent for men and around 32 per cent for women. IHD accounted for 48 per cent of all cardiovascular disease deaths in 2017."

"Despite a steady decline in age-specific mortality from CAD over the past several decades, IHD is now the leading cause of death worldwide, and the rate of CAD is expected to only accelerate in the coming decades; contributory factors include "ageing of the population, increases in the worldwide prevalence of obesity and type 2 diabetes, and a rise in cardiovascular risk factors." According to the World Health Organization, the global number of CAD fatalities will have grown from 7.5 million in 2005 to 11.2 million by 2020.

The idea that ischemic heart disease is identical to significant stenoses of the epicardial coronary arteries is "oversimplified." Ischemic heart disease can be caused by several causes that work together."

Historical review:

"The history of ischemic heart disease dates back to 1912 when James Brian Herrick first described the syndrome of prolonged, severe chest pain and attributed it to the development of blood clots in the vessels serving the heart. Thus, the term coronary thrombosis or coronary occlusion was used. The precise diagnosis was impossible until the electrocardiogram (ECG) was introduced into clinical practice in the 1920s.

In 1961, Julian[8] Put forth the concept of treatment of arrhythmias, cardio-pulmonary resuscitation with external ventricular defibrillation. This change in the approach caused a 50% reduction in admission mortality. By 1963, with the introduction of thrombolysis, acetylsalicylic acid, invasive cardiology and cardiac surgery, hospital mortality has reduced stepwise by almost 70%. Over the past 30 years, modern therapies and effective secondary prevention have significantly improved the two-year survival rate of patients after myocardial infarction, increasing it by 75%."[9]

"The 20th century saw a revolution in the understanding and treatment of ischemic heart disease, marked by key advancements such as the development of the electrocardiogram, echocardiography, and cardiac care units. Significant milestones also included the Framingham Heart Study, the lipid hypothesis of atherosclerosis, thrombolysis, heart catheterisation, percutaneous coronary interventions, open-heart surgery, and the introduction of implantable defibrillators."[10]

Diabetes mellitus

Diabetes is a worldwide epidemic. With 41 million diabetics in the country, India is the world's diabetes capital. "Diabetes prevalence for all age groups was expected to be 2.8%

in 2000 and 4.4% in 2030.[11,12] According to the International Diabetes Federation (2017), the prevalence of diabetes in 2017 and 2045 is expected to be 8.8 and 11.4%, respectively. Diabetic eye disease is getting more prevalent."[13] According to the most recent epidemiological estimates from 2019, 77 million people in India have diabetes, with the number anticipated to climb to nearly 134 million by 2045. With 77 million diabetics, India is second only to China in the worldwide diabetes epidemic. "Of these, 12.1 million are above the age of 65, expected to rise to 27.5 million by 2045. It is also estimated that about 57% of individuals with diabetes in India, or around 43.9 million people, are undiagnosed.[14]

Classification of diabetes mellitus

The four types of diabetes mellitus are identified as per the new classification.[15] Type 1, Type 2, "Other specific types" and gestational diabetes.

Type 1: autoimmune β-cell destruction leading to absolute insulin deficiency.

Type 2 (NIDDM / Adult onset) presents with insulin resistance in peripheral tissues and an insulin secretory defect of the β cells.

Other Specific Types: αβ-cell dysfunction (MODY or Maturity onset Diabetes Mellitus) or with defects of insulin action. Persons with diseases of the exocrine pancreas and dysfunction associated with endocrinopathies.

Diabetes that appears during pregnancy is known as gestational diabetes. Why it occurs is unknown; however, some suggest that HLA antigens, notably HLA DR2, 3, and 4, may play a role." Excess proinsulin is suspected to have a role in gestational diabetes, and some believe it may cause beta-cell stress. Others feel that excessive levels of hormones such as

progesterone, cortisol, prolactin, human placental lactogen, and oestrogen might interfere with beta-cell activity and peripheral insulin sensitivity.[16]

SECONDARY

- Pancreatic disease
- Hormonal abnormalities
- Genetic diseases
- Ingestion of certain drugs or chemical compounds.

Cushing syndrome, acromegaly, glucagonoma, hyperaldosteronism, hyperthyroidism, and somatostatinomas are all associated with glucose intolerance and diabetes mellitus due to the glucogenic effects of the hormones secreted excessively in these conditions. Additionally, diabetes mellitus is linked to disorders such as idiopathic hemochromatosis, where excessive iron accumulation in the pancreas leads to beta cell loss.

Criteria for the Diagnosis of Diabetes Mellitus[17]

1) Oral glucose tolerance test: ≥ 200 mg/dl at 2 hours plasma glucose

2) ≥126 mg/dl Fasting plasma glucose.

3) Symptoms associated with > 200 mg/dl plasma glucose concentration

Complications of diabetes mellitus

Diabetes is linked to several problems. Acute metabolic issues related to death include hypoglycemia, or low blood glucose, which causes unconsciousness, and diabetic ketoacidosis, which results from abnormally high blood glucose concentrations

(hyperglycemia). This analysis will concentrate on the long-term vascular problems of diabetes, which are undoubtedly its most destructive effect. These numerous issues are caused, at least in part, by persistently elevated blood glucose levels, which deteriorate blood vessels.

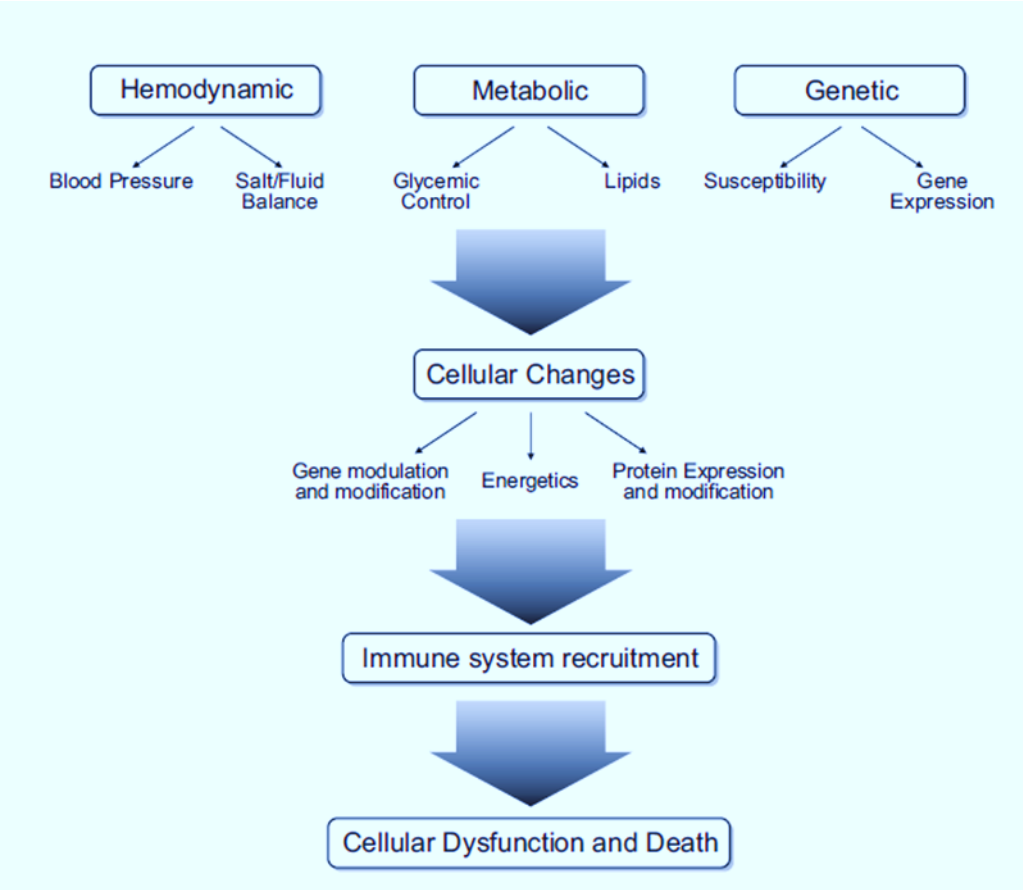

Figure 1: Major pathway showing the complications of diabetes mellitus

"The complications of diabetes are grouped as microvascular disease and macrovascular disease. The microvascular disease includes eye diseases such as retinopathy, kidney disease termed nephropathy, and damage to neural tissue called neuropathy.

The major macrovascular complications include accelerated cardiovascular disease, resulting in myocardial infarction and cerebrovascular disease, such as stroke. There is also

myocardial dysfunction associated with diabetes mellitus, which may appear at least in part independent of atherosclerosis.

Other complications of chronic origin include depression, sexual dysfunction, and dementia.

Coronary artery disease

Incidence of coronary artery disease:

Cardiovascular diseases (CVD) is the chief cause of mortality in a country like India, where a quarter of all mortality is due to CVD. The Global burden of disease study estimates a CVD death rate of 272/100000 population of India, which is higher than the global average of 235/100000 population. Premature mortality due to CVD was estimated to be 23.2 million in the year 1990 and has now increased by 57%, with 37 million lives lost in 2010.[18] CVDs, especially coronary Heart Disease (CHD), have a high epidemic proportion globally. Worldwide, CVD led to 17.5 million deaths in a survey of the year 2012. 75% of these occur in developing countries. The GBD study reported that deaths and disabilities from CHD have more than doubled in India in the last 30 years. Absolute number of deaths from CHD increased from 0.62 million (1990) to 0.78 million (1995), 0.95 million (2000), 1.01 million (2005) and 1.13 million (2010)."[19]

ATHEROSCLEROSIS:

"Atherosclerosis is a chronic immune-inflammatory, fibroproliferative disease, fueled by lipids, which primarily affects the intima of the medium-sized and large-sized arteries, resulting in intimal thickening and luminal narrowing and inadequate blood supply.[20]

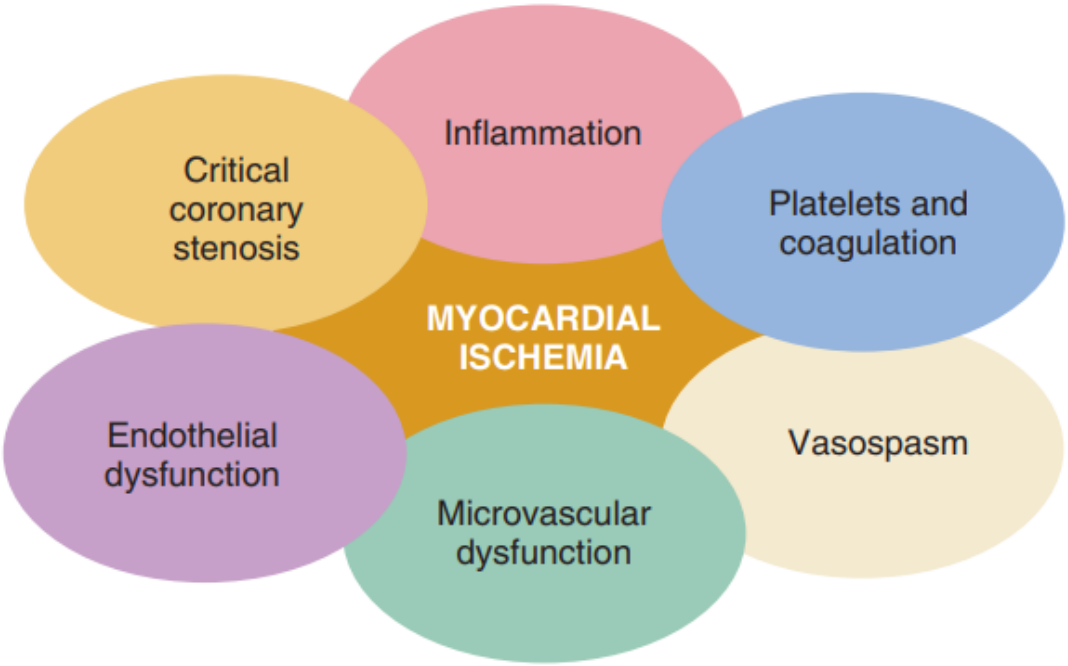

Figure 2: Interactions

The aetiology of atherosclerosis underwent significant change during the twentieth century. Atherosclerosis is no longer seen as an inanimate tube." Experiments conducted in the "early half of the twentieth century used dietary manipulation to generate fatty lesions in the arteries of animals, eventually identifying cholesterol as the reason. These findings, combined with identifying human lipoprotein particles in the "mid-twentieth century," pushed for the isolation of lipids as a cause of atherosclerosis. All of these elements play a role in atherogenesis.

RISK FACTORS FOR ATHEROSCLEROTIC CORONARY ARTERY DISEASE

Several prospective studies, like Framingham's study, established the coronary heart disease "risk" factors (i.e.) factors that make the occurrence of the disease more probable.[21]

NON-MODIFIABLE RISK FACTORS	MODIFIABLE RISK FACTORS
• AGE	• DIABETES MELLITUS
• MALE GENDER	• HYPERTENSION
• FAMILY HISTORY	• OBESITY
• GENETIC FACTORS	• SMOKING
• TYPE A PERSONALITY	• SEDENTARY LIFESTYLE
	• HYPERLIPIDEMIA • HIGH CARBOHYDRATE AND TRANS-UNSATURATED FAT INTAKE”

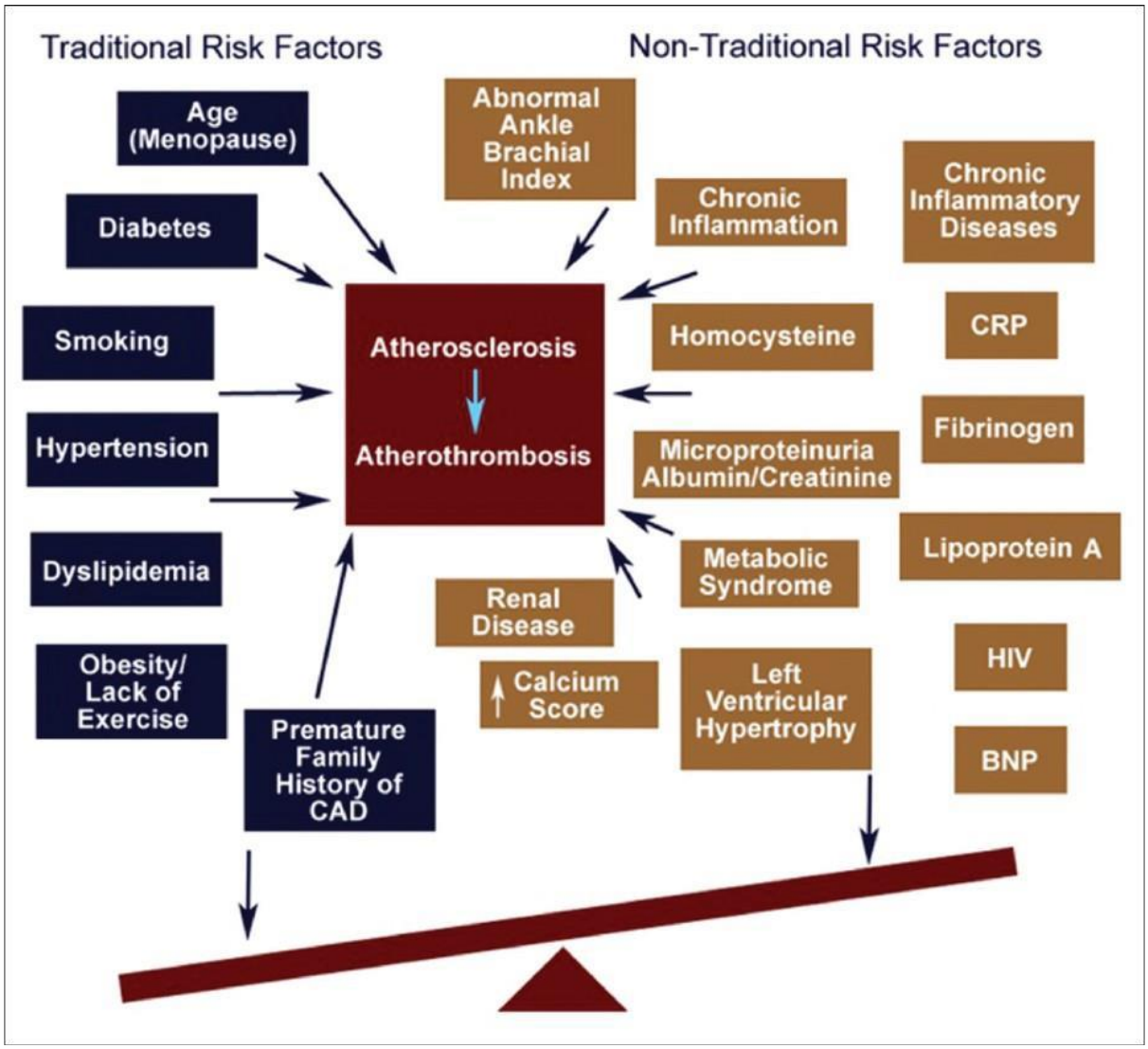

Figure 3: Showing the traditional and non-traditional risk factors

PATHOGENESIS OF ATHEROSCLEROSIS

The "response to injury" hypothesis considers "atherosclerosis to be a chronic, inflammatory response of the arterial wall initiated by injury to the endothelium.[22] Although the course of atherosclerotic disease progression is silent with a very long incubation period, the dreaded complications of this disease due to arterial lumen narrowing, like myocardial infarction, angina, and sudden cardiac death, have led to focusing much more attention towards the pathogenesis.

The role of inflammation in the atherosclerotic process has become increasingly recognised over the past decade.[23,24] From a pathological perspective, all stages of atherosclerotic plaque—initiation, growth, and complication—are influenced by inflammatory processes.[25,26]

The primary factors that drive atherogenesis include smoking, hypertension, atherogenic lipoproteins, and hyperglycemia. These risk factors create a range of harmful stimuli that trigger the release of leukocyte soluble adhesion molecules, which promote the attachment of monocytes to endothelial cells, as well as chemotactic factors that facilitate the migration of monocytes into the sub-intimal region."

"The transformation of monocytes into macrophages, followed by the uptake of cholesterol lipoproteins, is believed to initiate the formation of the fatty streak. This process, compounded by harmful stimuli, attracts further macrophages, mast cells, and activated T cells into the growing atherosclerotic lesion. Oxidised LDL (Ox-LDL) may play a key role in preventing the apoptosis of smooth muscle cells in the atherosclerotic plaque cap. Additionally, releasing metalloproteinases and other connective tissue enzymes by activated macrophages can degrade collagen, making the plaque more vulnerable to rupture." This rupture exposes the

necrotic core of the plaque to arterial blood, triggering thrombosis.

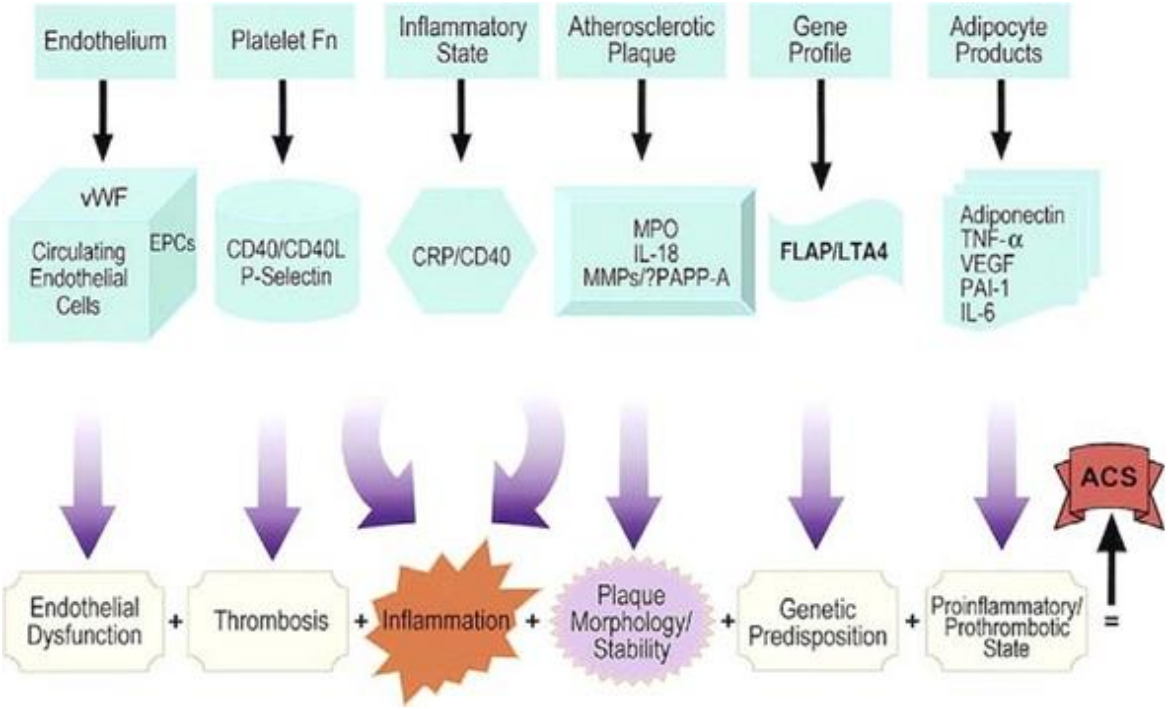

Figure 4: Various inflammatory markers.

INFLAMMATORY MARKERS IN ATHEROGENESIS.

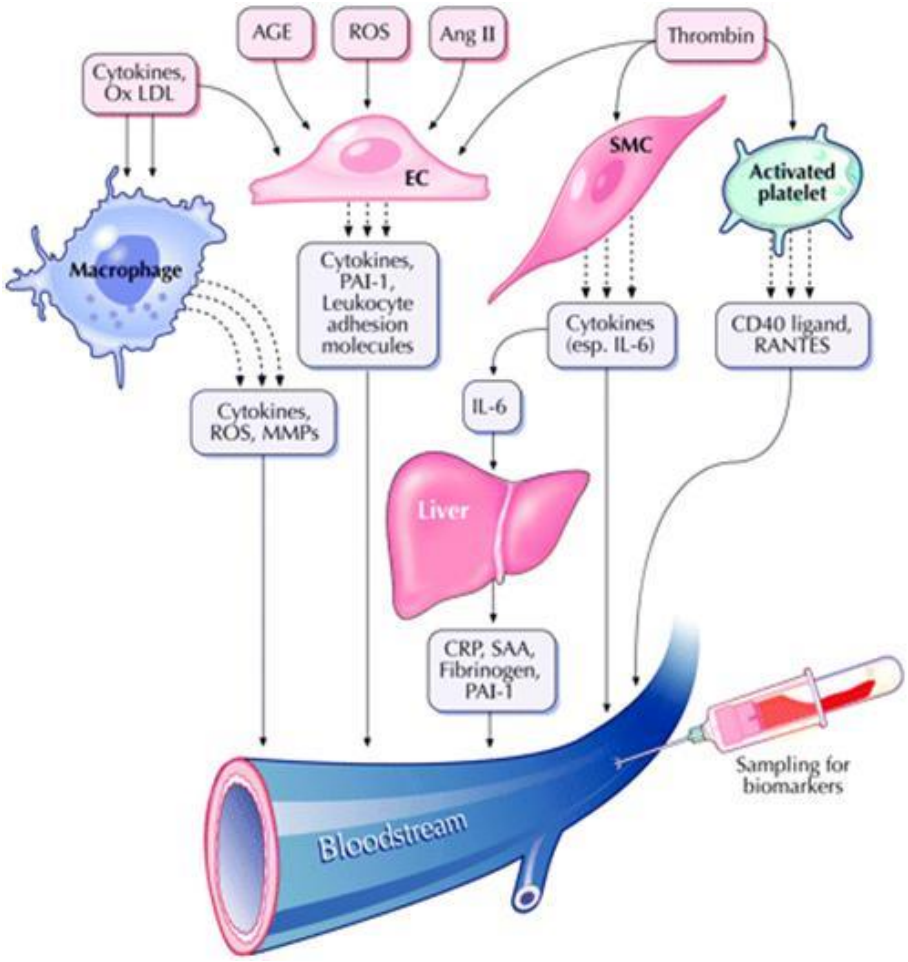

Figure 5: Inflammatory markers in atherogenesis

It is well-established that the inflammatory cascade has sources beyond the atherosclerotic coronary arteries, "including atherosclerosis in other vessels, systemic inflammation (such as in connective tissue diseases), and local infections (such as gingivitis, prostatitis, bronchitis, urinary tract infections, and gastric inflammatory conditions). These systemic inflammatory processes can lead to elevated levels of inflammatory markers, which may be mistakenly attributed to atherosclerotic cardiovascular disease.

The growing understanding of the inflammatory component in atherogenesis provides biological plausibility for using inflammation markers as effective indicators of atherosclerosis or as predictors of its complications. These pathophysiological insights open up potential targets for measuring ongoing inflammation and identifying its role in the disease process. Potential markers for measurement include pro-inflammatory risk factors such as oxidised low-density lipoprotein (Ox-LDL), pro-inflammatory cytokines (e.g., interleukin-1, TNF), adhesion molecules (e.g., ICAM-1, selectins), and inflammatory stimuli with hepatic effects (e.g., interleukin-6), as well as other products of hepatic stimulation like serum amyloid A (SAA) and C-reactive protein (CRP), along with other acute-phase proteins." Recognising the inflammatory cascade's role highlights these markers' potential as positive predictors of prevalent or emerging cardiovascular disease (CVD). "However, their clinical utility will depend on their ability to provide additional relevant information for practical application.

These include:

(1) Ease to standardise the assay and to control the variability of the measurement;

(2) established risk-factors;

(3) Association with CVD

(4) Presence of guide to interpreting results;

(5) Ability to improve the overall

(6) Generalization of results

(7) acceptable cost of the assays.

The use of inflammatory markers is influenced by their relationship with cardiovascular disease (CVD), whether linear, nonlinear, or dichotomous. These different relationships must be carefully examined when evaluating the effectiveness and relevance of the inflammatory markers currently under consideration.

Table 1. Assays of Inflammatory Markers for Potential Clinical Use

“

Complications of atherosclerosis.”

Growth typically occurs “discontinuously during the chronic asymptomatic or stable phase of lesion evolution, with periods of relative quiescence broken by periods of fast progression.

The stenoses may eventually advance to the point where they obstruct blood flow via the artery. Lesions with ‘more than 60% stenoses can induce flow limits under high demand conditions.’ This type of lesion causes the most common presenting symptoms of ‘chronic stable angina pectoris’ or intermittent claudication with increased activity or demand. As can be seen, the symptomatic phase of atherosclerosis frequently begins many decades after initiating the lesion. However, in many cases of ‘myocardial infarction,’ there is no prior history of stable angina.” Several types of imaging data imply that many episodes of Acute coronary syndrome are caused by lesions that do not restrict flow rather than ‘high-grade stenoses.’ Acute coronary syndromes are typically caused by thrombi that form as a result of “disruption of plaques that does not result in a critical stenosis.”

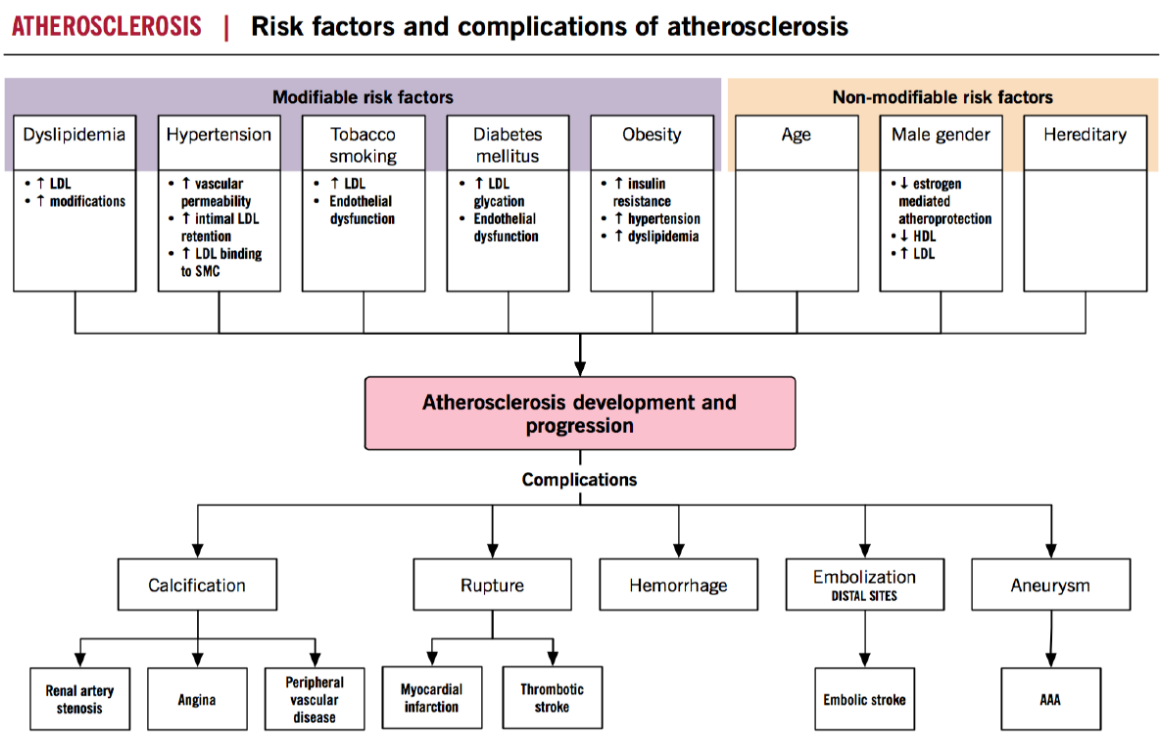

Figure 6: Risk factors and complications of atherosclerosis

Various articles discussing the coronary artery disease in diabetes mellitus patients;

A study conducted by Raggi P et al. in 2004 involving 10,377 individuals, including 903 diabetic patients, found that individuals with diabetes had a higher coronary artery calcium (CAC) score and mortality compared to non-diabetic individuals over a five-year follow-up period.[27]

In a 2006 study by Anand DV et al., 33% of patients had a myocardial calcium score of 400 or higher, with 28% also exhibiting inducible ischemia. The Diabetes Heart Study found that coronary artery calcium (CAC) was a reliable predictor of mortality, with higher levels of CAC significantly increasing the likelihood of mortality.[28]

A 2006 study by Scognamiglio R et al. revealed that 60% of asymptomatic diabetic patients had abnormal echocardiography findings, indicating the presence of ischemia. The study highlighted that significant coronary artery disease (CAD) existed in asymptomatic diabetics, and this was independent of their risk factor profile.[29]

In a study by Yoo WS et al. (2009) to "assess the clinical parameters to predict CAD in asymptomatic type 2 diabetic patients. In group A, the prevalence of coronary artery disease (CAD) was 41.0%, while in group B, it was 16.7%. A strong correlation was observed between the frequency of CAD and the number of risk factors, but no significant correlation was found with the severity of CAD. Multivariate analysis identified that in asymptomatic individuals, having diabetes for at least ten years and a family history of CAD were independent risk factors for CAD." The study recommends regular screening for CAD in individuals with type 2 diabetes who have had the condition for 10 or more years or who have a family history of the disease, even if they are asymptomatic.[30]

A 2010 study by Hadamitzky M et al. involving 140 asymptomatic diabetic patients who underwent

coronary computed tomography angiography (CCTA) found that the prevalence of coronary artery disease (CAD) was higher in type 2 diabetes mellitus compared to non-diabetics. The study concluded that diabetics had a three-fold increased rate of cardiac events and suggested that CCTA when combined with traditional risk factors, offers better predictive value for cardiac events in diabetics.[31]

A 2010 study by Yamasaki Y et al. evaluated myocardial perfusion imaging (MPI) in asymptomatic Japanese individuals with type 2 diabetes mellitus (DM2). The study found that patients with summed stress scores greater than or equal to 9 had a 1.9-fold increase in the rates of cardiovascular events or death. This suggests that abnormal myocardial perfusion imaging (MPI) may be a valuable prognostic tool for predicting an increased risk of cardiovascular disease in asymptomatic diabetic patients.[31]

In a cross-sectional study conducted by Tsujimoto T et al. (2011) to assess the asymptomatic coronary heart disease in patients with type 2 diabetes with vascular complications. The study documented that 53% of individuals with type 2 diabetes had asymptomatic coronary heart disease (CHD), with more than 50% of these patients showing significant coronary artery stenosis. Additionally, 31% had multivessel disease or left primary disease, and 38% had more than 75% diameter stenosis in their coronary arteries. The findings concluded that asymptomatic coronary heart disease (CHD), marked by more than 50% diameter stenosis and myocardial perfusion abnormalities, was identified in over half of the patients with type 2 diabetes, particularly those with vascular complications.[32]

In a study by Gazzaruso C et al. (2012) to evaluate the impact of screening for asymptomatic coronary artery disease (CAD) in type 2 diabetes mellitus (T2DM), multivariate Cox regression analysis revealed a significant protective effect of CAD screening against the incidence of major adverse cardiovascular events (MACE). According to our results, people with type 2 diabetes may see a substantial decrease in cardiovascular morbidity and death by screening for asymptomatic CAD. This

could result from specific therapeutic and diagnostic measures used in diabetic individuals whose CAD was confirmed at screening.[33]

In a study conducted by Sarkar NC et al. (2015) to assess the early detection of coronary artery disease (CAD) in asymptomatic type 2 diabetes mellitus, the duration of diabetes and the presence of multiple risk factors were found to be strongly correlated with the occurrence of CAD, as well as with the involvement of multiple coronary arteries.[34]

In a 2016 review by Tavares C et al., evaluating methods for screening asymptomatic coronary artery disease in individuals with type 2 diabetes mellitus, it was concluded that myocardial perfusion scintigraphy and stress echocardiography are the most effective screening techniques. These methods offer superior sensitivity and specificity compared to traditional exercise tests. But thanks to technological advancements, new imaging diagnostic techniques that are less invasive than conventional coronary angiography have been developed. These techniques are becoming increasingly important for diagnosing coronary artery disease because they demonstrate higher efficacy at a lower risk and invasiveness.[35]

In a 2020 study by Kadam S et al., aimed at assessing the prevalence of coronary artery disease in asymptomatic type 2 diabetes mellitus, it was found that 18.64% of the patients showed signs of CAD on ECG. The study emphasised the significance of recording the ECG in all the patients, even asymptomatic T2DM patients, during frequent intervals irrespective of the presence or absence related to CAD. The study observed that ECG changes usually do not match clinical symptoms and may be silent CAD is common. There is a weak correlation between the Rose Angina questionnaire and ECG in patients with asymptomatic CAD.[36]

In a 2022 study by Takamura K et al., which aimed to assess the risk factors for coronary artery disease

in asymptomatic patients with type 2 diabetes mellitus, it was concluded that this group was diverse, with some individuals showing no evidence of CAD. The study identified smoking and the Agatston score as key independent predictors of obstructive CAD. Specifically, current smoking and an Agatston score greater than 100 were found to be significant predictors of blocked coronary arteries.[37]

In a 2023 study by Kumar V et al., which assessed the prevalence of coronary artery disease in asymptomatic patients with type 2 diabetes mellitus, it was found that silent CAD was highly prevalent among these individuals. The study emphasised the importance of regular screening in such patients to detect CAD early and prevent the associated morbidity and mortality of overt coronary artery disease.[38]

AIMS & OBJECTIVES

Aim:

"To Find the Proportion of Asymptomatic Coronary Artery Disease in Type 2 Diabetes Mellitus and Risk Factors

Objective

- To detect the presence of coronary artery disease in type 2 diabetes mellitus patients
- To find any risk factors associated with coronary artery disease in type 2 diabetes mellitus patients."

MATERIAL & METHOD

Study design: Cross-Sectional Study.

Source of Data: "The study included patients attending outpatients and inpatients of BLDE (DU) Shri B.M Patil Medical College Hospital and Research Centre.

Study period: The period of study will be from May 2023 to December 2024

Inclusion Criteria:

- History of type 2 diabetes mellitus of more than 5 years
- History of type 2 diabetes mellitus according to ADA criteria.
- According to two or more risk factors.
- Age between 40-60 years.

Exclusion Criteria

- Patients having an absolute contraindication to TMT.
- Uncontrolled hypertension (BP>200/110 mm Hg),
- LVEF <30%

Sample Size:105

Baweja PS, Sandesara PB, Ashraf MJ. Asymptomatic coronary artery disease in type II diabetes. Missouri Medicine. 2014 Jan;111(1):73.[39]

With anticipated Sensitivity and specificity of ECG of Type 2 Diabetic patients at 50.0% and 80%, respectively, considering the prevalence of sepsis at 40% (Paramdeep et al.), at the precision of 1% and 95% confidence, the required minimum sample size is 105.

- Formula used is -

$$N=\frac{Z^2P(1-p)}{\Delta^2}$$

N will be (a+c) if we use sensitivity as p

N= (a+c)/Prevalence

Methodology

- Patients diagnosed with diabetes mellitus presenting to the OPD and IPD attending B.L.D.E (D U) Shri B.M Patil Medical College Hospital and Research Centre, Vijayapura, will be assessed with FBS, PPBS, HBA1C

- Then cases of Type 2 DM without any clinical and electrocardiographic evidence of coronary artery disease. The data were collected according to proforma in terms of detailed history, clinical examination, BMI, systemic examination and necessary investigations like" -

    - Electrocardiography (ECG)

    - FBS

    - PPBS

- HBA1C
- 2D Echo will be carried out, and vascular ageing will be assessed.
- Lipid profile
- TMT
- Coronary angiography

STATISTICAL ANALYSIS

"All the patient's data were collected in predesigned proforma and entered in an Excel sheet. The collected data were summarised into mean, standard deviation, frequency, and percentage. The summarised data were represented using tables, figures, bar diagrams and pie charts. The mean difference between continuous data was compared using an unpaired t-test and categorical data using a chi-square test. A p-value of <0.05 was considered statistically significant for all statistical purposes."

RESULTS

The present study included 105 patients, fulfilling the inclusion criteria. The mean age of patients was found to be 52.46±5.63yrs.

Table 1: Showing the mean age of patients.

	N	Minimum	Maximum	Mean	SD
Age in yrs	105	40	60.0	52.46	5.63

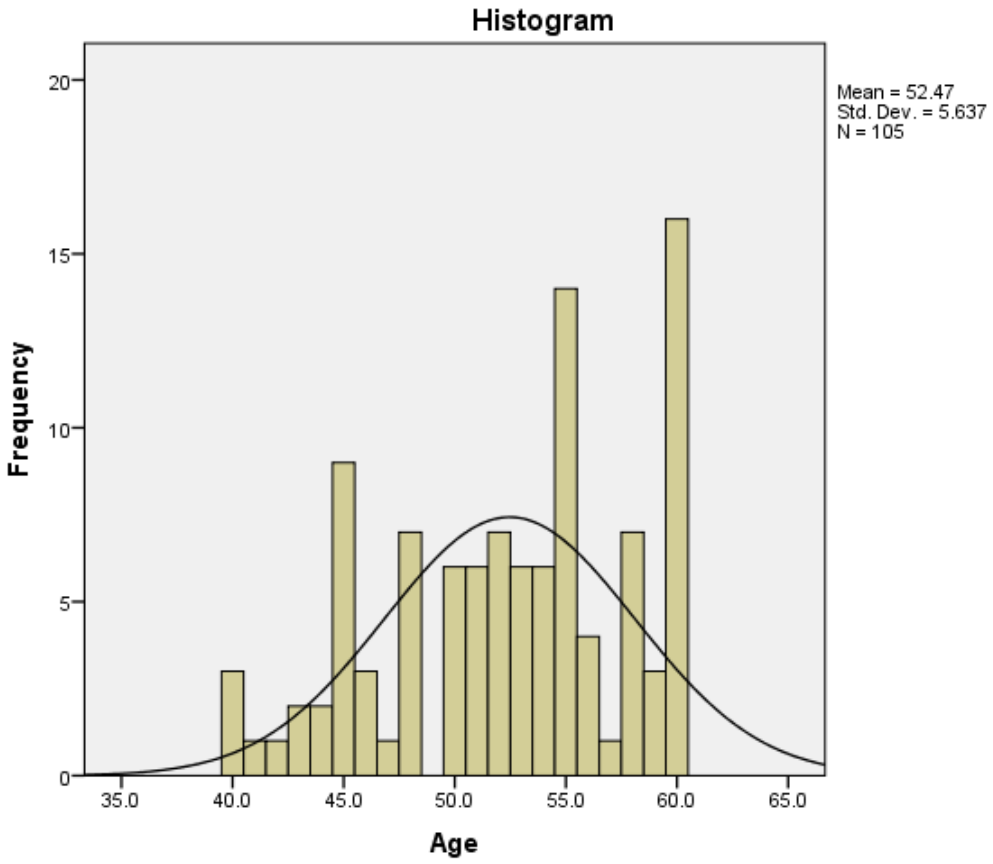

Figure 7: Showing the mean age of patients

Table 2: Comparison of mean age with CAD

	CAD				p-value
	Absent		Present		
	Mean	SD	Mean	SD	
Age	51.6	5.4	53.3	5.8	0.37

The mean age between the groups was comparable, with no significant difference. The mean age of patients was 51.6 years in CAD absent and 53.3 years in CAD present cases.

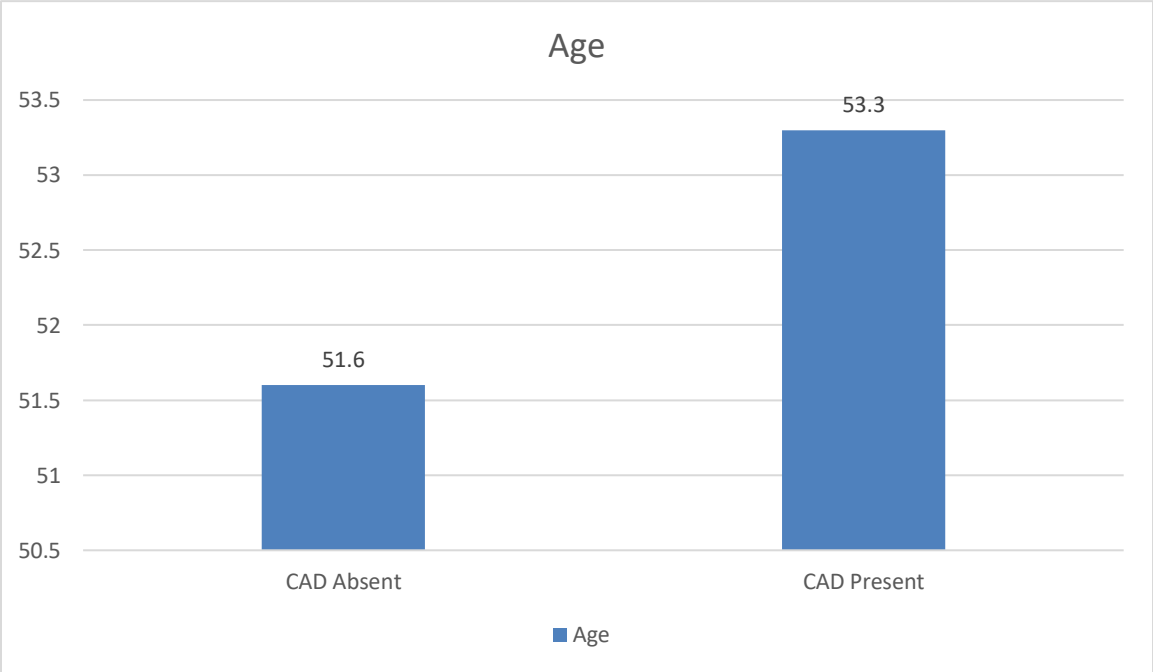

Figure 8: Comparison of mean age with CAD

Table 3: Gender distribution

		Count	N %
Gender	Female	42	40.0%
	Male	63	60.0%

Among them, 60% were male and 40% were female patients, with marginal male preponderance.

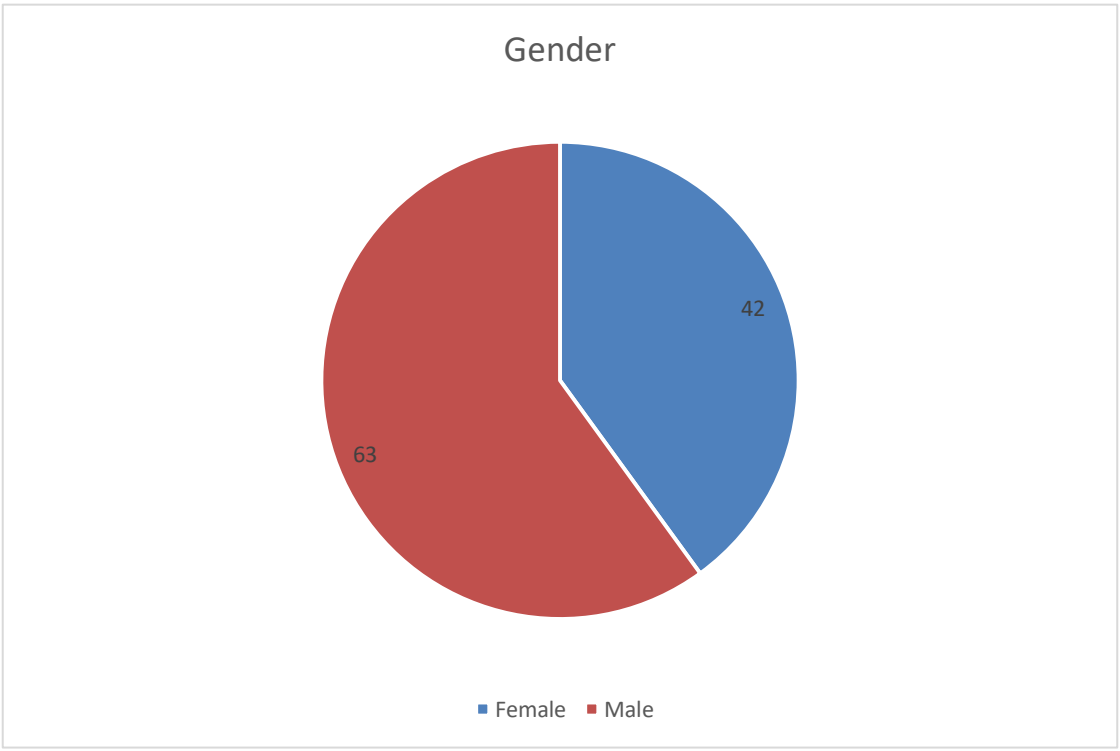

Figure 9: Gender distribution

Table 4: Comparison of gender with CAD

		CAD			
		Absent		Present	
		Count	N %	Count	N %
Gender	Female	16	32.7%	26	46.4%
	Male	33	67.3%	30	53.6%

Gender distribution between the groups was comparable, with no significant difference noted.

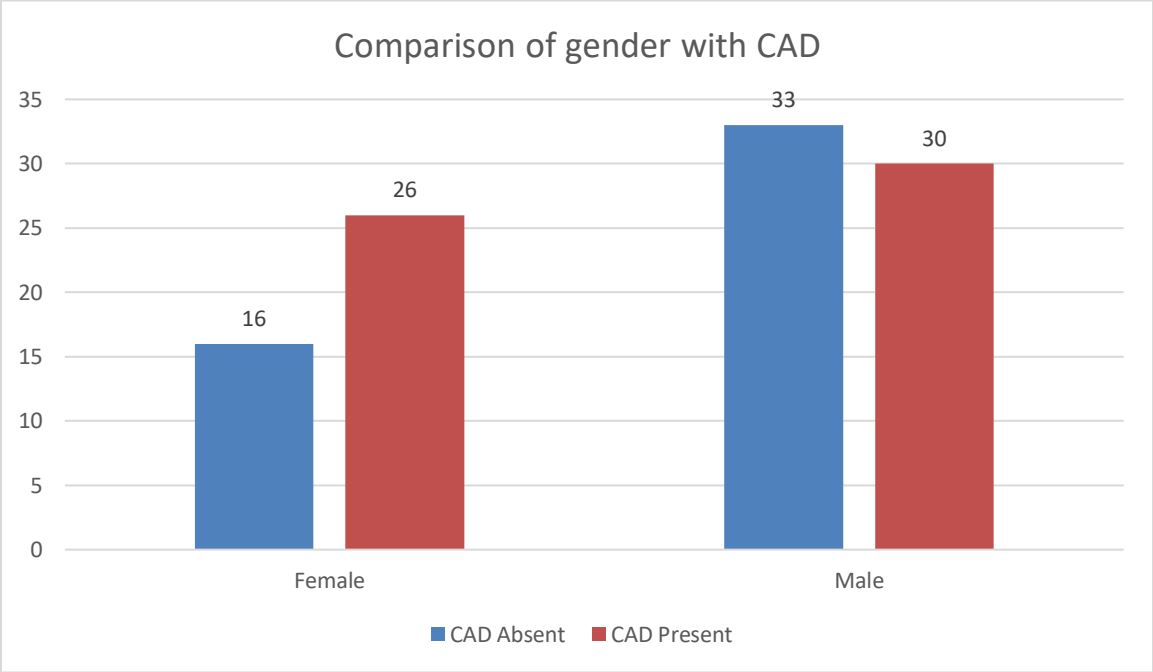

Figure 10: Comparison of gender with CAD

Table 5: Distribution according to occupation

		Count	N %
Occupation	Army	1	1.0%
	Bank officers	1	1.0%
	Business	15	14.3%
	Employee	8	7.6%
	Farmer	33	31.4%
	Homemaker	34	32.4%
	Police	1	1.0%
	Sportsman	1	1.0%
	Worker	11	10.5%

The majority were homemakers by occupation (32.4%), followed by farmers (31.4%), businesspeople (14.3%), and 10.5% of workers.

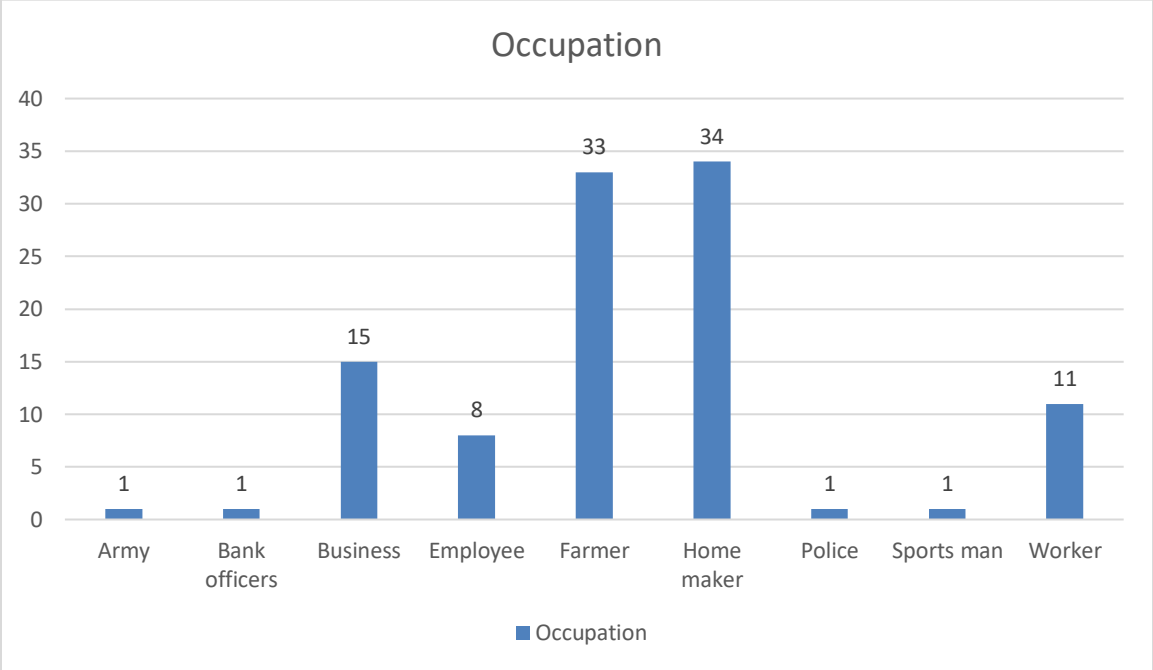

Figure 11: Distribution according to occupation

Table 6: Showing distribution of smoking, alcohol and hypertension

		Count	N %
Smoking	Absent	14	13.3%
	Present	91	86.7%
Alcohol	Absent	45	42.9%
	Present	60	57.1%
HTN	Absent	44	41.9%
	Present	61	58.1%

Among the participants, 86.7% were smokers, 57.1% were alcoholics, and 58.1% were with hypertension.

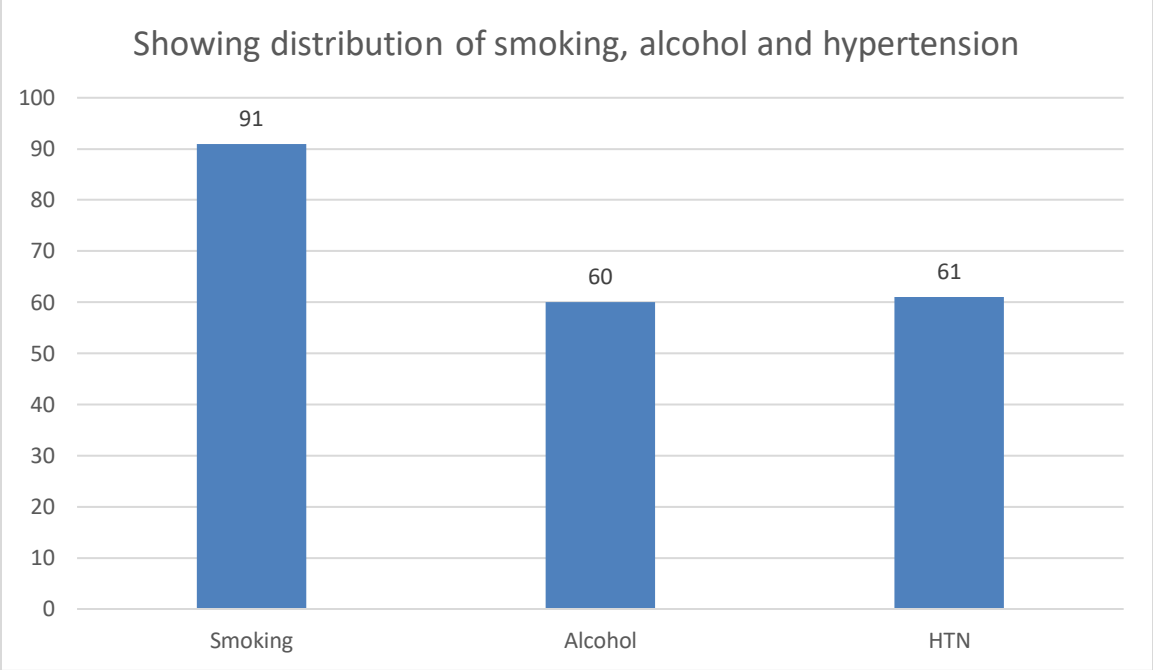

Figure 12: Showing distribution of smoking, alcohol and hypertension

Table 7: Showing findings on ECG and 2D ECHO

		Count	N %
ECG finding	Normal	49	46.7%
	NSTEMI	16	15.2%
	STEMI	37	35.2%
	T-waves inversions	3	2.9%

2D ECHO finding	IHD	56	53.3%
	Normal	49	46.7%

ECG findings show the Presence of STEMI in 35.2%, NSTEMI in 15.2% and 2.9%, with T-wave inversion—2D ECHO showing IHD in 53.3% of the cases.

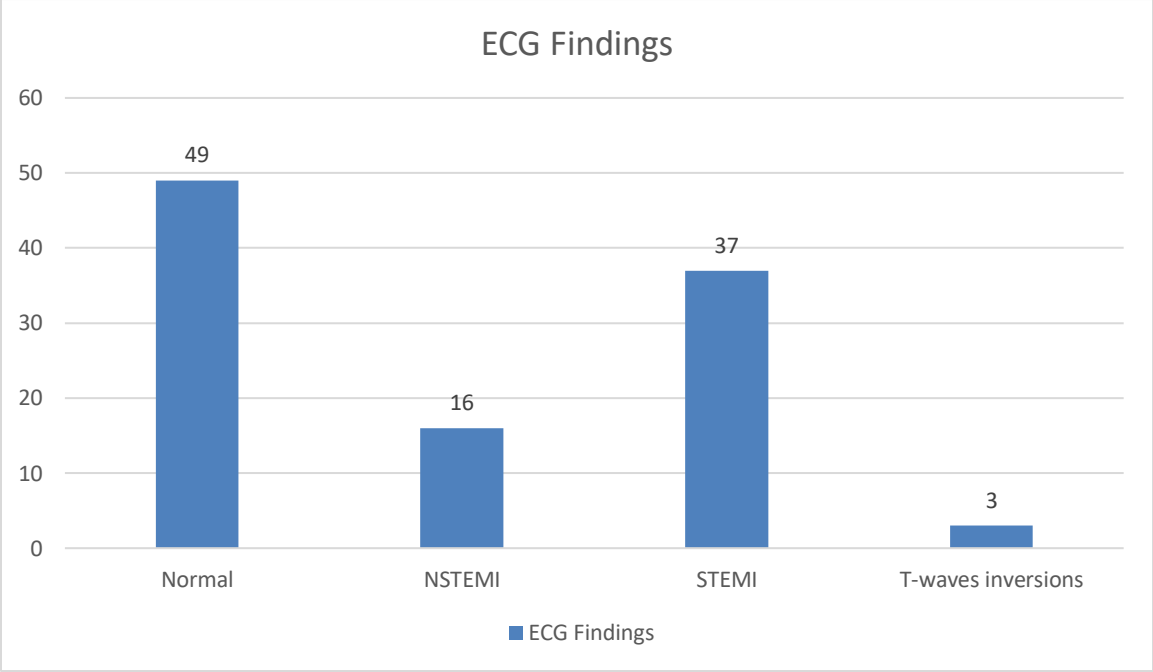

Figure 13: Showing findings on ECG

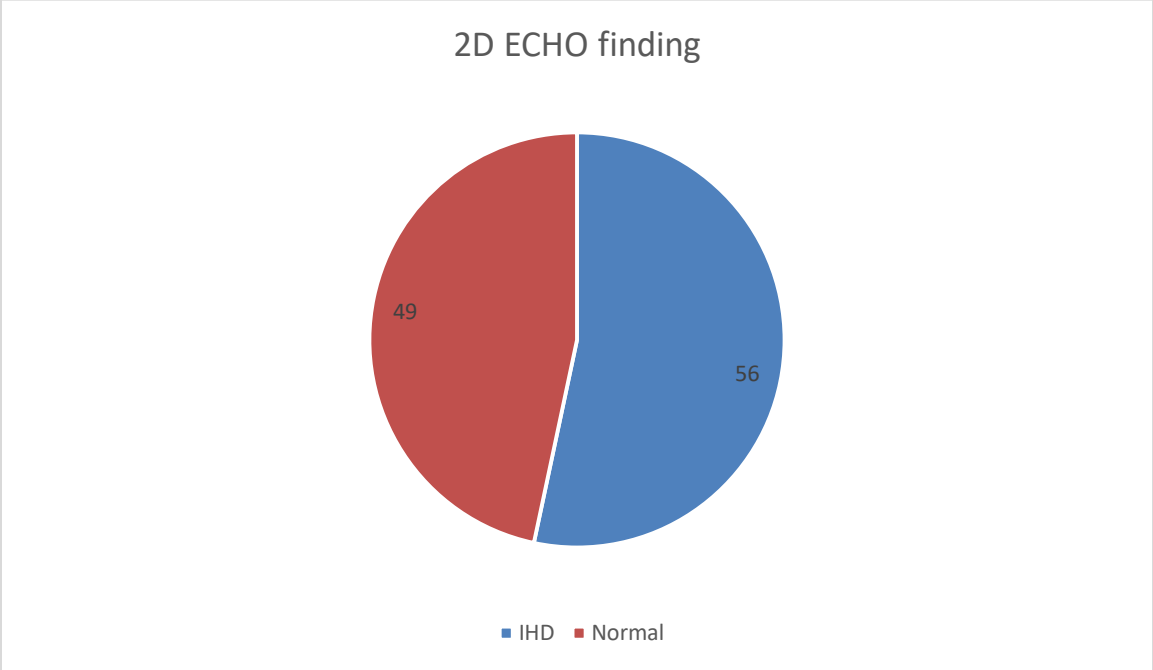

Figure 14: Showing findings on 2D ECHO

Table 8: Showing the coronary angiography findings

		Count	N %
Coronary angiography	Normal	49	46.7%
	SVD	14	13.3%
	DVD	18	17.1%
	TVD	24	22.9%

The coronary angiography shows the pattern of TVD in 22.9%, DVD in 17.1% and SVD in 13.3% of the cases.

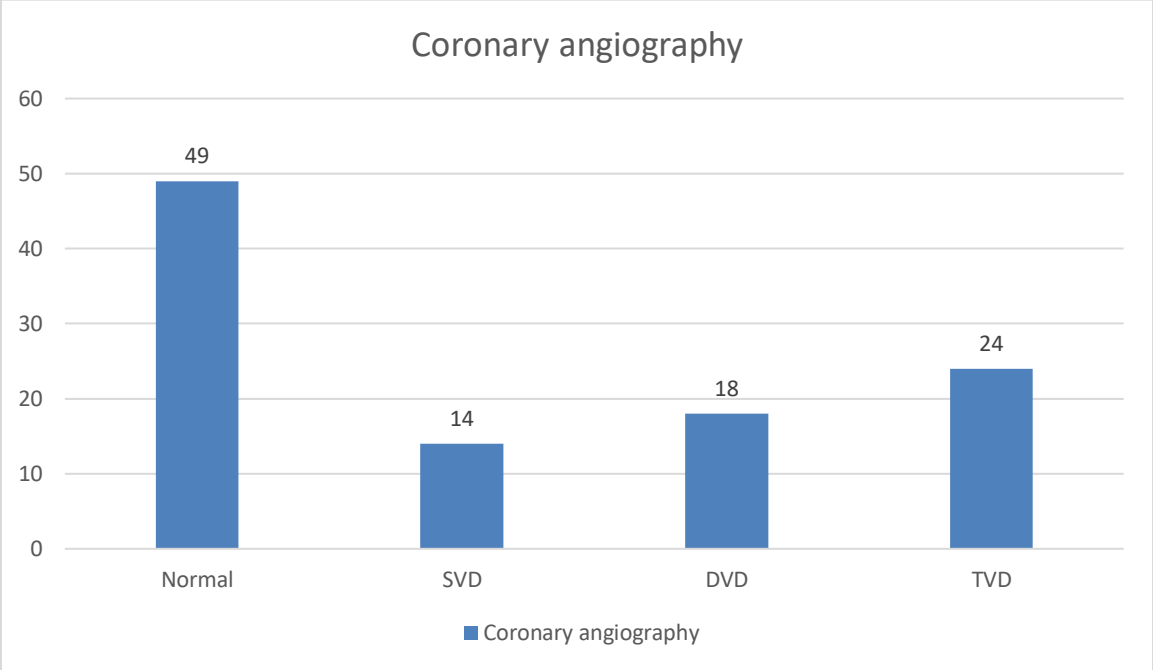

Figure 15: Showing the coronary angiography findings

Table 9: Showing the presence of CAD among patients

		Count	N %
CAD	Absent	49	46.7%
	Present	56	53.3%

Coronary artery disease was present in 53.3% of the cases with diabetes mellitus in the present study.

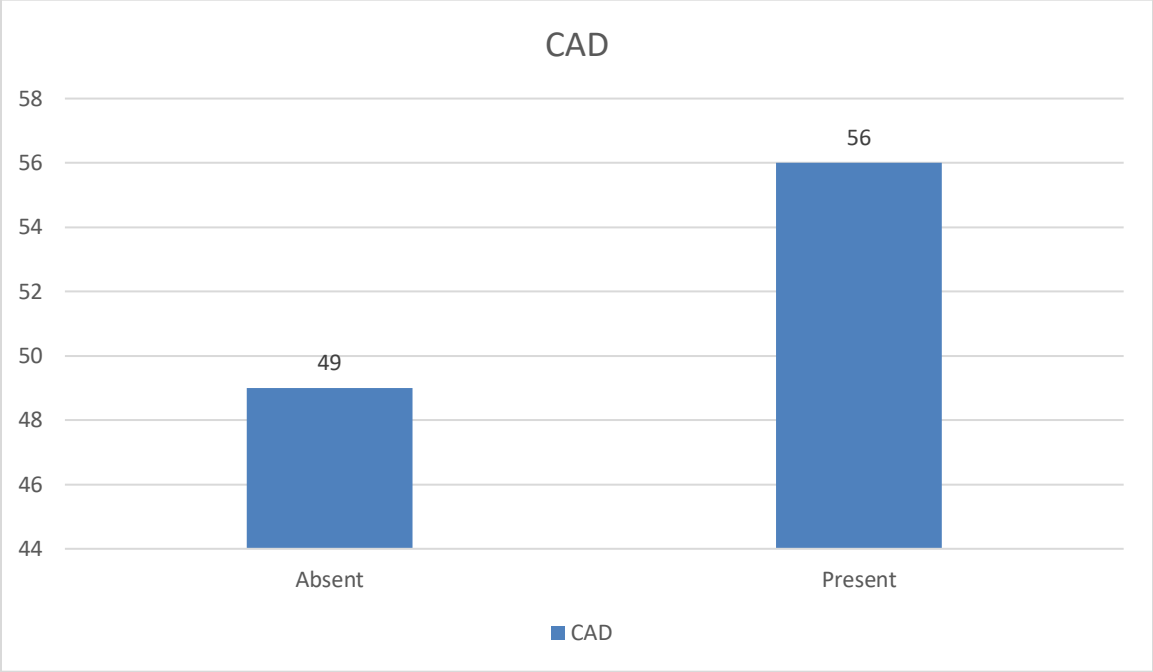

Figure 16: Showing the presence of CAD among patients

Table 10: Comparison of smoking with CAD

		CAD				Chi-square (p-value)
		Absent		Present		
		Count	N %	Count	N %	
Smoking	Absent	22	44.8%	10	17.9%	3.21 (0.01)*
	Present	27	55.2%	46	82.1%	

Smoking was found to be significantly higher among patients with CAD (82.1%) compared to patients without CAD (55.2%). ($p<0.05$)

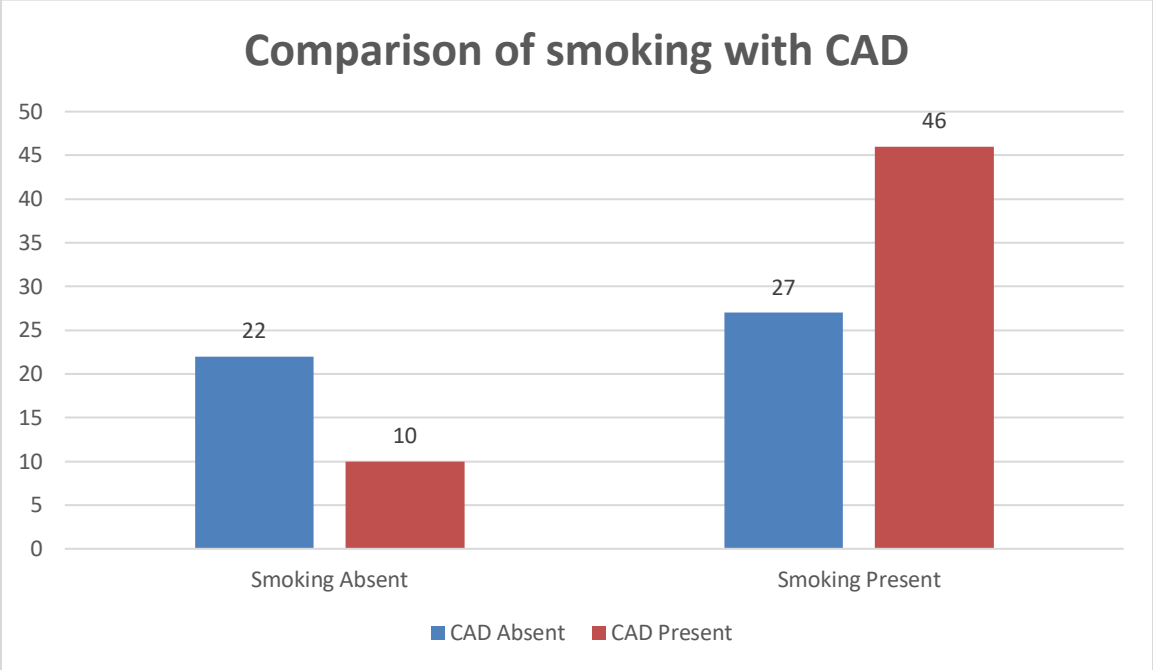

Figure 17: Comparison of smoking with CAD

Table 11: Comparison of alcohol consumption with CAD.

		CAD				Chi-square (p-value)
		Absent		Present		
		Count	N %	Count	N %	
Alcohol	Absent	18	36.7%	15	26.7%	1.24 (0.32)
	Present	31	63.3%	41	73.3%	

There is a higher incidence of alcohol consumption among the CAD present (73.3%) patients compared to CAD absent (63.3%). However, this was not statistically significant.

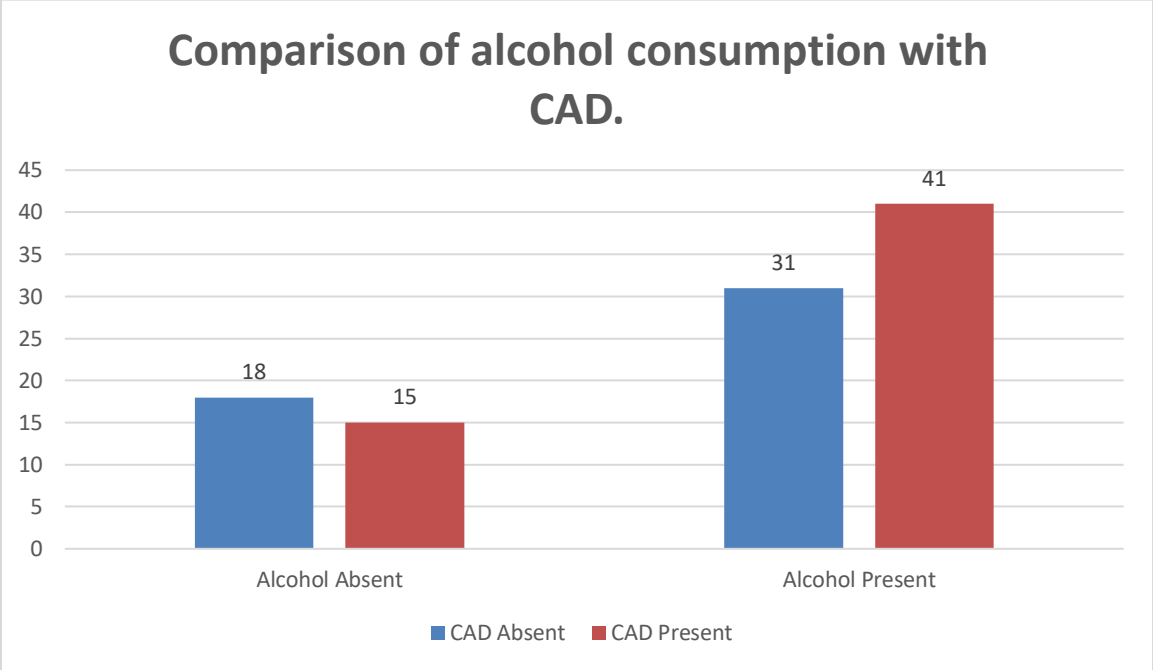

Figure 18: Comparison of alcohol consumption with CAD.

Table 12: Comparison of mean physical characters with CAD.

	CAD				p-value
	Absent		Present		
	Mean	SD	Mean	SD	
Weight	74.1	8.1	78.6	8.7	0.14
Height	163.7	6.4	166.3	6.2	0.22
BMI	23.69	1.97	26.73	1.79	0.05*

There is a significantly higher mean BMI among the cases with CAD than patients without CAD.

However, the mean weight and height were comparable in the two groups.

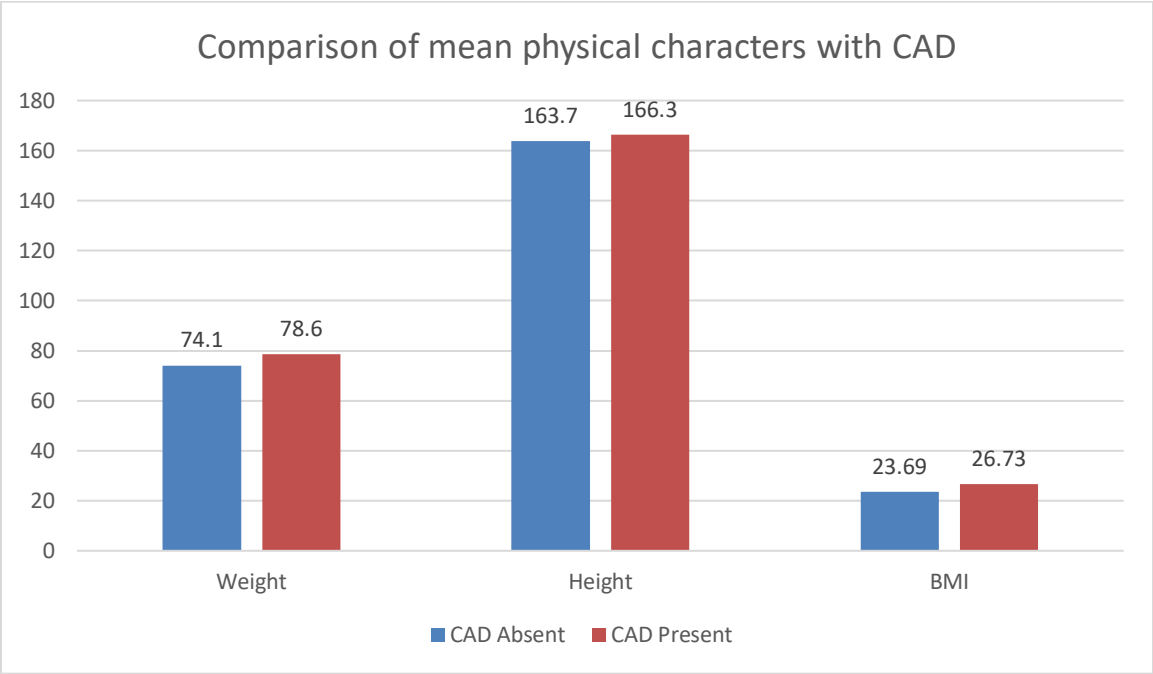

Figure 19: Comparison of mean physical characters with CAD

Table 13: Comparison of vital parameters with CAD

	CAD				p-value
	Absent		Present		
	Mean	SD	Mean	SD	
PR (min)	79.5	7.5	82.1	7.0	0.39
RR (min)	16.4	1.4	16.6	1.4	0.84
SBP (mmHg)	125.4	14.9	138.9	13.8	0.21

DBP (mmHg)	79.5	11.1	82.4	10.1	0.3

The vital parameters such as pulse rate, respiratory rate, and blood pressure were not significantly different between the groups. However, the mean blood pressure among the patients with CAD was higher than those without CAD.

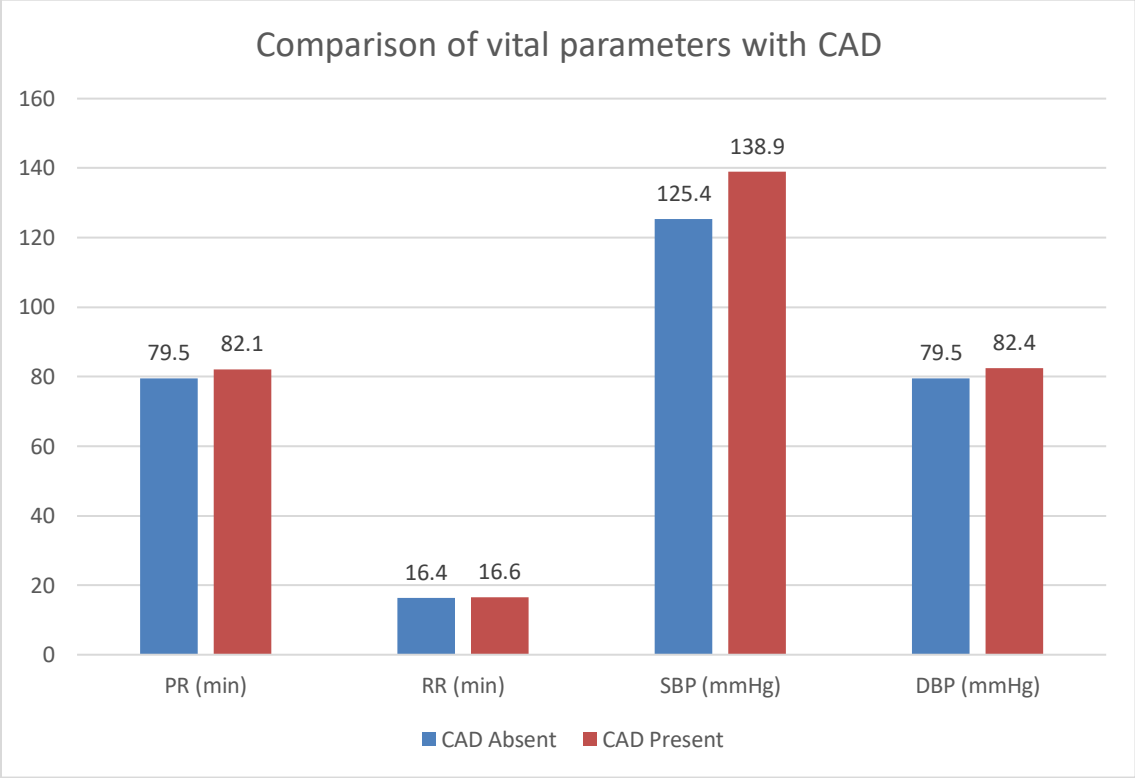

Figure 20: Comparison of vital parameters with CAD

Table 14: Comparison of glycemic status with CAD

	CAD		p-value
	Absent	Present	

	Mean	SD	Mean	SD	
FBS (mg/dL)	191.1	46.0	221.4	39.9	0.21
PPBS (mg/dL)	228.5	58.1	284.6	57.9	0.05*
HbA1c (%)	7.7	1.6	8.3	1.3	0.05*

On the assessment of glycemic status, there is a significantly higher mean HbA1c among the cases with CAD (8.3±1.3) compared to cases without CAD (7.7±1.6).

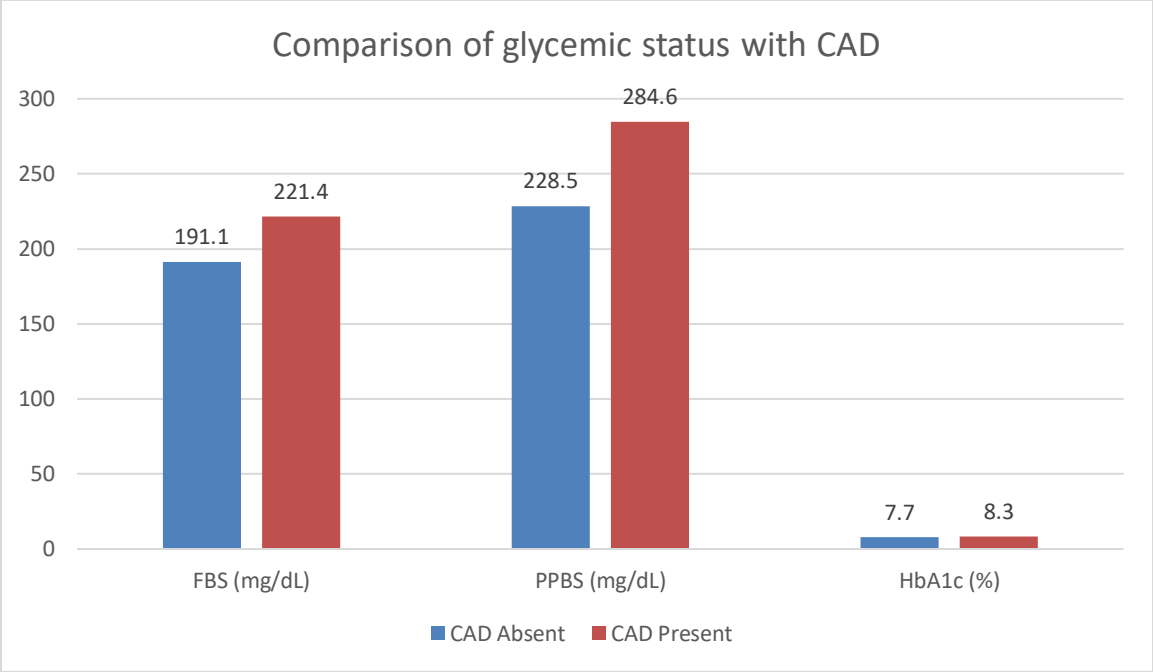

Figure 21: Comparison of glycemic status with CAD

Table 15: Comparison of lipid profile parameters with CAD

	CAD	p-value

	Absent		Present		
	Mean	SD	Mean	SD	
T. Cholesterol (mg/dl)	191.1	31.5	224.4	29.2	0.01*
TG (mg/dl)	180.4	42.0	236.3	50.3	0.01*
LDL Cholesterol (mg/dl)	95.6	15.2	121.7	22.9	0.01*
HDL Cholesterol (mg/dl)	36.6	4.6	31.4	5.0	0.01*

On lipid profile assessment, "there is a significant difference between the groups' mean lipid parameters. The mean total cholesterol, triglycerides, and LDL cholesterol were significantly higher in cases with CAD than those without CAD. Similarly, the mean HDL was significantly lower in cases with CAD than those without CAD.($p<0.05$) showing significant dyslipidemia among the cases with CAD compared to cases without CAD.

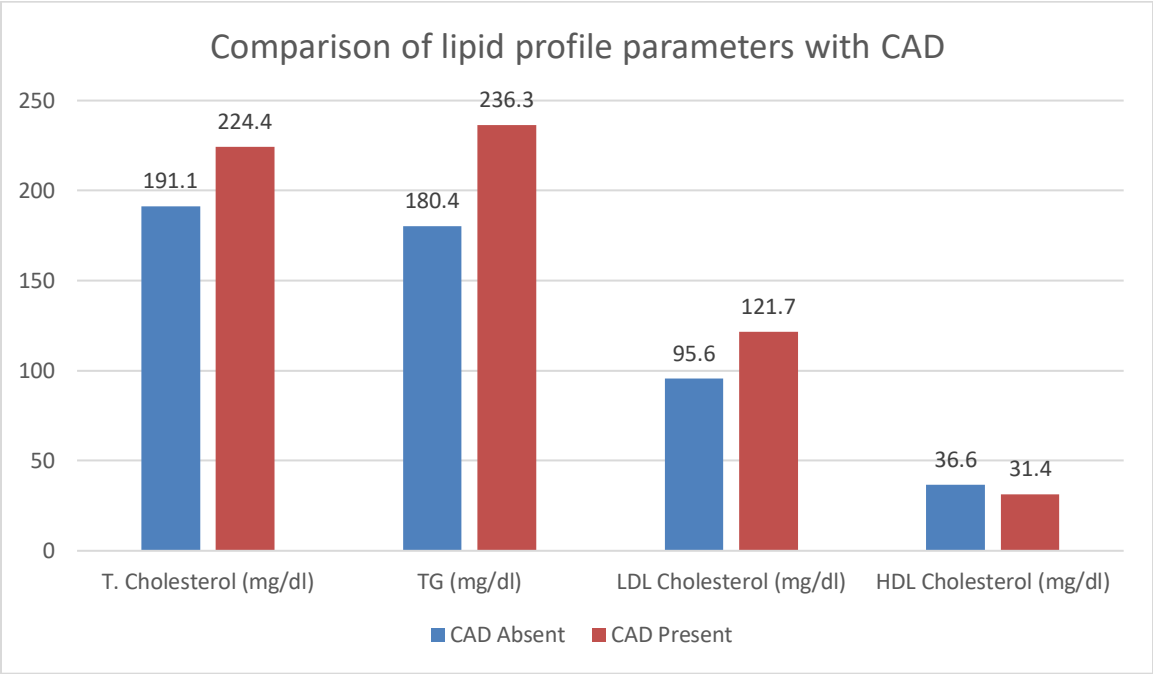

Figure 22: Comparison of lipid profile parameters with CAD

DISCUSSION

Coronary artery disease (CAD) is a significant complication in individuals with type 2 diabetes mellitus (T2DM), often remaining asymptomatic until severe cardiovascular events occur. The presence of asymptomatic CAD in patients with T2DM poses a significant clinical challenge, as the lack of overt symptoms may delay diagnosis and timely intervention." This discussion aims to interpret the study's findings, analyse the association between risk factors and the presence of silent CAD, and compare the results with existing literature.[40]

The high prevalence of asymptomatic CAD in T2DM patients underscores the role of metabolic and cardiovascular risk factors in disease progression. Key risk factors such as dyslipidemia, hypertension, obesity, and chronic hyperglycemia contribute to the development of atherosclerosis and endothelial dysfunction, accelerating the onset of CAD even in the absence of symptoms. Moreover, insulin resistance, a hallmark of T2DM, further exacerbates vascular complications by promoting inflammation, oxidative stress, and plaque formation.[41,42]

Studies have consistently demonstrated that traditional risk factors, such as age, smoking, and family history of cardiovascular disease, play a significant role in CAD development among diabetic patients. However, emerging evidence suggests that novel biomarkers may provide additional predictive value in identifying high-risk individuals, including high-sensitivity C-reactive protein (hs-CRP) and homocysteine levels—subclinical inflammation and vascular calcification's role in asymptomatic CAD warrants further investigation.

"Early detection of asymptomatic CAD in T2DM patients is crucial for risk stratification and implementation of preventive measures. Non-invasive imaging modalities such as coronary artery

calcium (CAC) scoring, stress testing, and computed tomography angiography (CTA) have been explored as potential screening tools. However, their routine use remains controversial due to cost-effectiveness considerations and varying sensitivity and specificity in different patient populations."[43,44]

Overall, the presence of asymptomatic CAD in T2DM patients highlights the need for a proactive approach to cardiovascular risk assessment. Stringent glycemic control, lifestyle modifications, and pharmacological interventions, including statins and antihypertensive agents, are pivotal in reducing cardiovascular morbidity and mortality. Future research should focus on refining screening strategies and identifying high-risk subgroups to optimise early intervention efforts.

The present study included 105 patients, fulfilling the inclusion criteria. The mean age of patients was found to be 52.46±5.63yrs. The mean age between the groups was comparable, with no significant difference. The mean age of patients was 51.6 years in CAD absent and 53.3 years in CAD present cases. Among them, 60% were male and 40% were female patients, with marginal male preponderance. Among the participants, 86.7% were smokers, 57.1% were alcoholics, and 58.1% were with hypertension.

A similar study by Yoo WS et al. found the same age group between the groups. The mean age was 60.5 years in the CAD group and 55.5 years in cases without CAD. There was a preponderance of male patients compared to female.[30] In another study by Kadam et al., the mean age of patients was 47.8 years, with a female preponderance showing that there were 63 female and 35 male patients. [36]

Coronary artery disease was present in 53.3% of the cases with diabetes mellitus in the present study. The coronary angiography shows the pattern of TVD in 22.9%, DVD in 17.1% and SVD in 13.3% of

the cases. ECG findings show the Presence of STEMI in 35.2%, NSTEMI in 15.2% and 2.9%, with T-wave inversion—2D ECHO showing IHD in 53.3% of the cases.

In concordance, Kumar V. et al. found that silent CAD was highly prevalent among these individuals. The study emphasised the importance of regular screening in such patients to detect CAD early and prevent the associated morbidity and mortality of overt coronary artery disease.[38]

Smoking was found to be significantly higher among patients with CAD (82.1%) compared to patients without CAD (55.2%). ($p<0.05$) There is a significantly higher mean BMI among the cases with CAD than patients without CAD.

In concordance with the present study, Tsujimoto T et al. documented that 53% of individuals with type 2 diabetes had asymptomatic coronary heart disease (CHD), with more than 50% of these patients showing significant coronary artery stenosis. Additionally, 31% had multivessel disease or left primary disease, and 38% had more than 75% diameter stenosis in their coronary arteries. The findings concluded that asymptomatic coronary heart disease (CHD), marked by more than 50% diameter stenosis and myocardial perfusion abnormalities, was identified in over half of the patients with type 2 diabetes, particularly those with vascular complications.[32]

A study by Takamura K et al. identified smoking as a key independent predictor of obstructive CAD.[37] Another study by Kadam S et al. found that 18.64% of the patients showed signs of CAD on ECG. The study emphasised the significance of recording the ECG in all the patients, even asymptomatic T2DM patients, during frequent intervals irrespective of the presence or absence related to CAD. The study observed that ECG changes usually do not match clinical symptoms and may be silent CAD is common.[36]

Another study by Yoo WS et al. "found the prevalence of coronary artery disease in 41% of the cases with diabetes mellitus. The study recommends regular screening for CAD in individuals with type 2 diabetes who have had the condition for 10 or more years or who have a family history of the disease, even if they are asymptomatic.[30]

Lipid profile abnormalities are widespread in type 2 diabetes, and it has a great influence on CAD. On the assessment of glycemic status, there is a significantly higher mean HbA1c among the cases with CAD (8.3±1.3) compared to cases without CAD (7.7±1.6). The lipid profile assessment shows a significant difference in the mean lipid parameters between the groups. The mean total cholesterol, triglycerides, and LDL cholesterol were significantly higher in cases with CAD than those without CAD. Similarly, the mean HDL was significantly lower in cases with CAD than those without CAD. ($p<0.05$) showing significant dyslipidemia among the cases with CAD compared to cases without CAD."

Yamasaki Y et al. suggest that abnormal myocardial perfusion imaging (MPI) may be a valuable prognostic tool for predicting an increased risk of cardiovascular disease in asymptomatic diabetic patients.[31] Like the present study, Takamura K et al. documented higher mean total cholesterol, LDL cholesterol, and triglycerides and a lower mean HDL cholesterol level in patients with CAD positive compared to cases without CAD.[37] In Another study by Kumar V et al., the BMI and Waist circumference were significantly higher among the cases with CAD compared to patients without CAD. The HbA1c and lipid profile (cholesterol and LDL) were significantly higher among the patients with CAD.[38]

Recommendations

1. **Routine Screening for CAD:** Given the high prevalence of asymptomatic CAD in T2DM patients, routine screening using non-invasive tools such as ECG, 2D echocardiography, and coronary artery calcium (CAC) scoring should be considered, especially for high-risk individuals.
2. **Strict Glycemic Control:** Maintaining optimal HbA1c levels through proper diabetes management, including lifestyle modifications, oral hypoglycemic agents, and insulin therapy when necessary, can help reduce CAD risk.
3. **Lipid Management:** Early and aggressive treatment of dyslipidemia through statins, fibrates, and lifestyle changes should be emphasised to lower total cholesterol, LDL, and triglycerides while increasing HDL levels.
4. **Smoking Cessation Programs:** Since smoking is a significant risk factor, targeted smoking cessation programs should be integrated into routine diabetic care to reduce cardiovascular complications.
5. **Hypertension Control:** Regular monitoring and adequate blood pressure management should be prioritised through antihypertensive medications, dietary modifications, and physical activity.
6. **Weight and BMI Management:** Patients should be encouraged to achieve and maintain a healthy weight through a balanced diet and structured exercise programs to lower CAD risk.
7. **Alcohol Reduction Strategies:** Although not statistically significant in this study, alcohol consumption was higher among CAD-positive patients. Public health initiatives should promote awareness of the cardiovascular risks associated with alcohol intake.

8. **Patient Education and Lifestyle Counseling:** Awareness programs should be developed to educate diabetic patients about the risks of asymptomatic CAD and the importance of regular check-ups, medication adherence, and healthy lifestyle choices.
9. **Further Research:** More extensive longitudinal studies should be conducted better to understand the predictive factors of asymptomatic CAD in T2DM and develop cost-effective screening strategies for early detection.

Implementing these recommendations can help reduce the burden of CAD in diabetic populations and improve long-term cardiovascular outcomes.

SUMMARY

- The present study included a total of 105 patients fulfilling inclusion criteria. The mean age of patients was found to be 52.46±5.63yrs.
- "The mean age between the groups was comparable, with no significant difference. The mean age of patients was 51.6 years in CAD absent and 53.3 years in CAD present cases.
- Among them, 60% were male and 40% were female patients, with marginal male preponderance.
- Gender distribution between the groups was comparable, with no significant difference noted.
- The majority were homemakers by occupation (32.4%), followed by farmers 31.4%, business people 14.3% and 10.5% workers.
- Among the participants, 86.7% were smokers, 57.1% were alcoholics, and 58.1% were with hypertension.
- ECG findings show the Presence of STEMI in 35.2%, NSTEMI in 15.2% and 2.9% with T-wave inversion. 2D ECHO showed IHD in 53.3% of the cases.
- The coronary angiography shows the pattern of TVD in 22.9%, DVD in 17.1% and SVD in 13.3% of the cases.
- Coronary artery disease was present in 53.3% of the cases with diabetes mellitus in the present study.
- Smoking was found to be significantly higher among the patients with CAD (82.1%) compared

to patients without CAD (55.2%). ($p<0.05$)

- There is a higher incidence of alcohol consumption among the CAD present (73.3%) patients compared to CAD absent (63.3%). However, this was not statistically significant.

- There is a significantly higher mean BMI among the cases with CAD than patients without CAD. However, the mean weight and height were comparable in the two groups.

- The vital parameters such as pulse rate, respiratory rate, and blood pressure were not significantly different between the groups. However, the mean blood pressure among the patients with CAD was higher than those without CAD.

- On glycemic status assessment, there is significantly higher mean HbA1c among cases with CAD (8.3±1.3) than cases without CAD (7.7±1.6).

- On the lipid profile assessment, there is a significant difference in the mean lipid parameters between the groups. The mean total cholesterol, triglycerides, and LDL cholesterol were significantly higher in cases with CAD than those without CAD. Similarly, the mean HDL was significantly lower in cases with CAD than those without CAD.($p<0.05$) showing significant dyslipidemia among the cases with CAD compared to cases without CAD."

CONCLUSION

“The present study, which included 105 patients with type 2 diabetes mellitus, highlights the significant burden of asymptomatic coronary artery disease. The findings reveal that CAD was present in 53.3% of the cases, with a mean patient age of 52.46 years and a slight male predominance. Smoking emerged as a significant risk factor, with a higher prevalence among CAD-positive patients (82.1%) compared to CAD-negative patients (55.2%). Additionally, CAD-positive individuals exhibited significantly higher mean BMI and HbA1c levels, indicating the role of poor glycemic control and obesity in disease progression.

Lipid profile assessment revealed that CAD-positive cases had significantly higher total cholesterol, triglycerides, and LDL cholesterol levels and lower HDL levels, confirming the presence of dyslipidemia as a critical risk factor. While alcohol consumption and blood pressure were higher among CAD-positive patients, these differences were not statistically significant. Furthermore, coronary angiography findings demonstrated that triple-vessel disease (TVD) was the most common pattern, affecting 22.9% of cases.

Overall, the study underscores the importance of early screening and intervention in T2DM patients to identify asymptomatic CAD and mitigate associated cardiovascular risks. Strategies such as lifestyle modifications, strict glycemic control, aggressive management of dyslipidemia and smoking cessation should be prioritised. Future research should explore more cost-effective and reliable screening tools to facilitate early detection and improve clinical outcomes in this high-risk population.”

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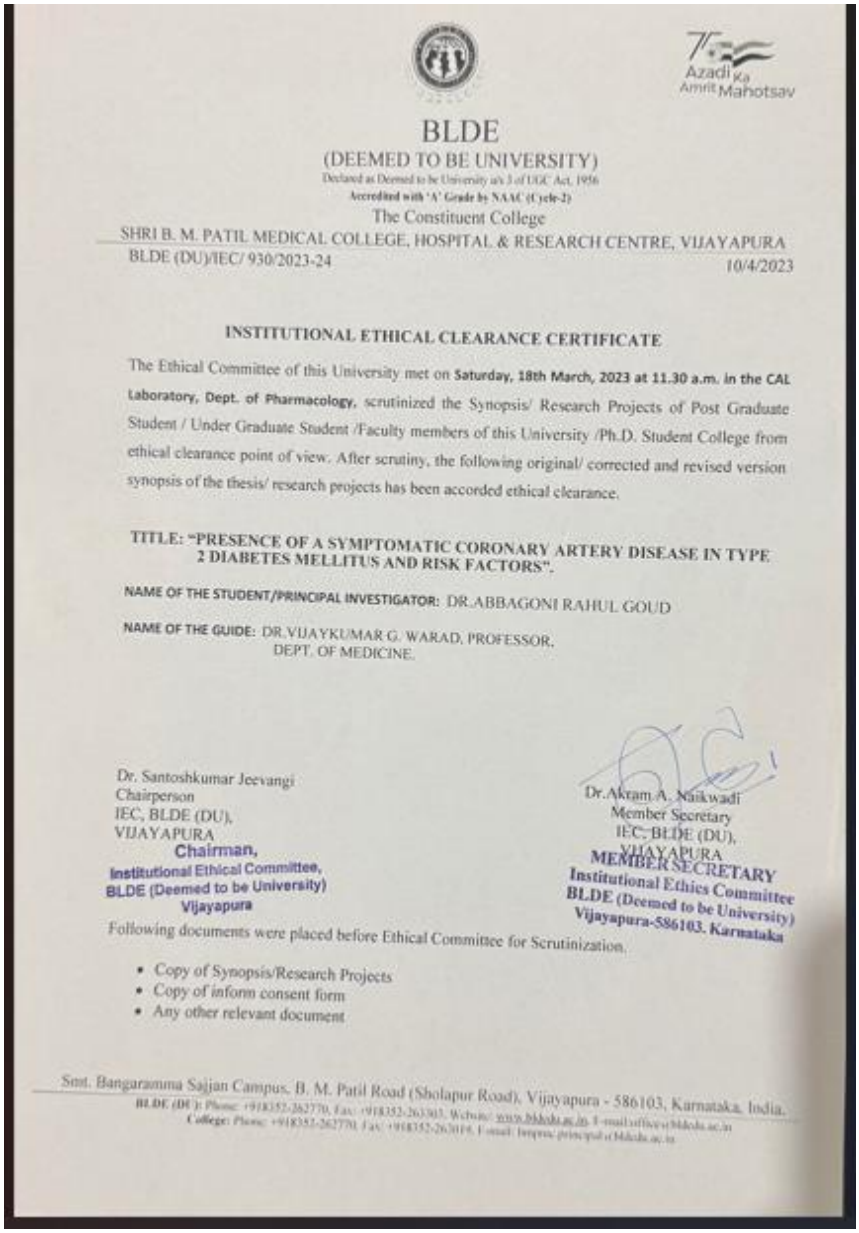
ANNEXURE- I

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE



BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 930/2023-24 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinized the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "PRESENCE OF A SYMPTOMATIC CORONARY ARTERY DISEASE IN TYPE 2 DIABETES MELLITUS AND RISK FACTORS".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.ABBAGONI RAHUL GOUD

NAME OF THE GUIDE: DR.VIJAYKUMAR G. WARAD, PROFESSOR, DEPT. OF MEDICINE.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA
Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University)
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA
MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103, Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

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

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.
BLDE (DU): Phone: +918352-262770, Fax: +918352-263303, Website: www.bldedu.ac.in, E-mail:office@bldedu.ac.in
College: Phone: +918352-262770, Fax: +918352-263019, E-mail: hmpmc.principal@bldedu.ac.in

ANNEXURE- II

INFORMED CONSENT FORM

BLDE (DEEMED TO BE UNIVERSITY),

SHRI B.M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA, KARNATAKA - 586103.

TITLE OF THE PROJECT – PRESENCE OF ASYMPTOMATIC CORONARY ARTERY DISEASE IN TYPE 2 DIABETES MELLITUS AND RISK FACTORS

PRINCIPAL INVESTIGATOR**: Dr ABBAGONI RAHUL GOUD**

P.G. GUIDE: Dr **VIJAYKUMAR G WARAD**

PROFESSOR OF MEDICINE

CHAIRMAN ETHICAL COMMITTEE-

All aspects of this consent form are explained to the patient in the language they understand.

1) PURPOSE OF RESEARCH:

I have been informed about this study. I have also been given a free choice of participation in this study.

2) PROCEDURE:

I am aware that in addition to receiving routine care, the investigator will ask me a series of questions. I have been asked to undergo the necessary investigations and treatment to help investigate this study.

3) RISK AND DISCOMFORTS:

I understand, and I am aware that I may experience some pain and discomfort during the examination or my treatment. This is mainly the result of my condition, and the procedure of this study is not expected to exaggerate these feelings associated with the usual course of treatment.

4) BENEFITS:

I understand that my participation and association with this study will help patients' survival and better outcomes.

5) CONFIDENTIALITY:

I understand that the medical information produced by this study will become a part of Hospital records and will be subject to confidentiality and privacy regulations. Information of a sensitive personal nature will not be a part of the medical records. However, it will be stored in the investigator's research file and identified only by a code number. The code key connecting names to numbers will be kept separately. If the data are used for publication in the

No name will be used for medical literature or teaching purposes, and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand that I may see the pictures and videotapes and hear the audiotapes before giving this permission.

6) REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study when **Dr ABBAGONI RAHUL GOUD** is available to answer my questions or concerns. I know I will be informed of any significant new findings discovered during the study, which might influence my continued participation. If, during the study or later, I wish to discuss my involvement in or concerns regarding this study with someone not directly involved, I am aware that the hospital's social worker is available to talk with me. I will be given a copy of this consent form to keep for careful reading.

7) REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or withdraw my consent and discontinue participating in the study at any time without prejudice to my present or future care at this hospital. I also understand that ***DR.*** **ABBAGON RAHUL GOUD** may terminate my participation in the study after he has explained the reasons for doing so and has helped arrange for my continued care by my physician or physical therapist if appropriate.

8) INJURY STATEMENT:

I understand that in the unlikely event of injury to me resulting directly from my participation in this study if such injury were reported promptly, the appropriate treatment would be available, but no further compensation would be provided. I understand my agreement to participate in this study. I am not waiving any of my legal rights. In the patient's language, I explained the purpose of this research, the procedures required, and the possible risks and benefits to the best of my ability.

Participant **Dr ABBAGONI RAHUL GOUD**

(Investigator)

Date

I) STUDY SUBJECT CONSENT STATEMENT:

I confirm that **Dr ABBAGONI RAHUL GOUD** has explained the purpose of the research, the study procedures I will undergo, and the possible risks and discomforts as well as benefits that I may experience in my own language. I have read and understand this consent form. Therefore, I agree to give consent to participate as a subject in this research project.

Participant / Guardian Date:

Witness to signature Date:

ANNEXURE –III

BLDE DEEMED TO BE UNIVERSITY,

SHRI B M PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE, VIJAYAPURA, KARNATAKA - 586103.

SCHEME OF CASE TAKING

Name: CASE NO:

Age: OP/IP NO:

Sex: DOA:

Religion: DOD:

Occupation:

Address:

Presenting complaints with duration:

History of presenting complaints:

Past History:

Family History:

Personal History:

Treatment History:

General Physical Examination

Pallor: present/absent

Icterus: present/absent

Cyanosis: present/absent

Clubbing: present/absent

Generalised lymphadenopathy: present/absent

Edema: present/absent

VITALS:

PR:

BP: in mm of mercury (mm hg)

RR:

Temp:

SYSTEMIC EXAMINATION

Cardiovascular system

Respiratory system

Per abdomen

Central nervous system

INVESTIGATIONS:

1. ELECTROCARDIOGRAPHY
2. FASTING BLOOD GLUCOSE
3. POSTPRANDIAL BLOOD GLUCOSE
4. GLYCOSYLATED HAEMOGLOBIN
5. LIPID PROFILE
6. 2D ECHO
7. TMT
8. CORONARY ANGIOGRAPHY

Other relevant investigations will be done when required.

CONCLUSION:

DATE: **SIGNATURE**

ANNEXURE-IV

MASTERCHART

Sl No	IP/OP No	Name	Age	Gender	Occupation	Smoking	Alcohol	HTN duration	DM duration	Family H/O CAD	Weight	Height	BMI	PR min	RR min	SBP mmHg	DBP mmHg
1	206487	Ashok	58.00	Male	POLICE	P	P	A	5.00	A	78.00	173.00	23.70	80.00	16.00	120.00	80.00
2	204903	Nagappa	54.00	Male	Farmer	P	P	A	6.00	A	65.00	169.00	22.00	78.00	16.00	110.00	80.00
3	211313	Nilappa	60.00	Male	Farmer	P	P	A	5.00	A	66.00	170.00	22.00	76.00	16.00	110.00	80.00
4	212725	Amjad	53.00	Male	SPORTSMAN	P	P	P	5.00	A	65.00	178.00	23.00	87.00	18.00	140.00	89.00
5	219631	Ramana	54.00	Male	Farmer	P	A	A	5.00	A	68.00	173.00	22.00	78.00	16.00	110.00	80.00
6	216206	Rayawwa	45.00	Female	Home Maker	A	A	A	5.00	A	76.00	155.00	25.00	76.00	18.00	130.00	90.00
7	4373	Rauf Ansari	52.00	Male	Worker	P	P	P	8.00	A	70.00	169.00	23.00	88.00	18.00	180.00	100.00
8	4472	Gayatri	45.00	Female	Home Maker	P	A	P	15.00	A	66.00	165.00	24.00	86.00	16.00	140.00	90.00
9	4791	Dayanandh	54.00	Male	Farmer	P	P	P	10.00	A	68.00	168.00	23.00	80.00	18.00	140.00	90.00
10	5191	Mhomed Nooralam	52.00	Male	Farmer	P	P	A	12.00	A	70.00	174.00	24.00	76.00	18.00	90.00	60.00
11	58505	Malikarjun	46.00	Male	EMPOLYE	P	P	A	5.00	A	76.00	178.00	23.00	80.00	16.00	130.00	90.00
12	6037	Laxmi	50.00	Female	Home Maker	P	A	P	10.00	A	78.00	166.00	26.00	87.00	18.00	110.00	70.00

13	209188	Bapuray	48.00	Male	Farmer	P	P	P	15.00	A	57.00	159.00	24.00	92.00	14.00	120.00	80.00
14	201948	Basamma	60.00	Female	Home Maker	P	A	P	15.00	A	53.00	165.00	28.00	90.00	15.00	110.00	90.00
15	224565	Sada shivayya	52.00	Male	Farmer	P	P	P	6.00	A	59.00	168.00	22.00	96.00	18.00	130.00	60.00
16	240238	Indubai	55.00	Female	Home Maker	A	A	A	10.00	A	60.00	164.00	21.00	88.00	16.00	110.00	60.00
17	268289	Iraj Hottagi	42.00	Male	Worker	P	P	P	5.00	A	64.00	162.00	24.00	89.00	14.00	130.00	80.00
18	287821	Jaibuniga	55.00	Female	Home Maker	P	A	P	15.00	A	80.00	166.00	22.60	88.00	14.00	100.00	60.00
19	232997	Santhabai	53.00	Female	Worker	P	A	P	15.00	A	87.00	170.00	22.50	78.00	16.00	140.00	100.00
20	18247	Yellavva	60.00	Female	Home Maker	P	A	P	7.00	A	74.00	174.00	22.30	76.00	16.00	139.00	60.00
21	356960	Shekavva	55.00	Female	Home Maker	P	A	P	5.00	A	77.00	175.00	22.70	77.00	17.00	140.00	80.00
22	377319	Nannabai	48.00	Female	Worker	A	A	P	6.00	A	79.00	156.00	22.70	74.00	18.00	130.00	90.00
23	206476	Jaganadh Poojari	55.00	Male	Business	P	P	P	10.00	A	74.00	168.00	22.30	80.00	17.00	140.00	80.00
24	105588	Kamalavva	60.00	Female	Farmer	P	A	P	8.00	A	58.00	153.00	22.10	80.00	16.00	110.00	80.00
25	103921	Mairunabee	40.00	Female	Home Maker	P	A	P	6.00	A	56.00	156.00	22.60	82.00	16.00	130.00	80.00
26	125570	Nagamma	60.00	Female	Home Maker	A	A	A	16.00	A	54.00	165.00	22.82	80.00	15.00	110.00	90.00
27	138135	Ramappa	55.00	Male	Farmer	P	P	A	5.00	A	46.00	163.00	21.00	64.00	14.00	140.00	90.00
28	207180	Rudra Gowda	60.00	Male	Farmer	P	P	A	12.00	A	54.00	157.00	24.00	66.00	14.00	130.00	70.00
29	7893	Mahadevi Pujari	58.00	Female	Home Maker	P	A	P	10.00	A	56.00	158.00	23.00	68.00	16.00	135.00	90.00
30	10705	Siddappa Gubbi	48.00	Male	Home Maker	P	P	P	10.00	A	65.00	168.00	23.00	76.00	15.00	125.00	70.00
31	10700	Bibanabi Babusab	50.00	Female	Farmer	P	A	A	10.00	A	78.00	162.00	24.00	86.00	18.00	140.00	90.00

32	280662	Arjun	45.00	Male	EMPOLYE	P	P	A	6.00	A	73.00	170.00	22.00	80.00	17.00	130.00	90.00
33	282113	Bhimappa	59.00	Male	Farmer	P	P	A	6.00	A	65.00	161.00	24.00	80.00	16.00	130.00	80.00
34	15143	Malkappa	54.00	Male	ARMY	P	P	A	5.00	A	66.00	172.00	22.40	78.00	16.00	130.00	90.00
35	112157	Kallanna Gowda	50.00	Male	Business	P	P	P	10.00	A	66.00	162.00	23.50	76.00	16.00	130.00	80.00
36	377319	Nannabai Poojari	48.00	Female	Worker	P	A	P	5.00	A	75.00	156.00	25.20	82.00	19.00	170.00	100.00
37	172180	KamalaBai	43.00	Female	Farmer	P	A	P	5.00	A	62.00	158.00	24.10	72.00	16.00	130.00	80.00
38	89674	Ramanna Gowda	51.00	Male	Farmer	P	P	A	10.00	A	76.00	165.00	23.20	76.00	16.00	110.00	70.00
39	298849	Dandamma	55.00	Female	Home Maker	P	A	A	10.00	A	62.00	163.00	24.60	80.00	17.00	110.00	70.00
40	SSH00037	Banumma	45.00	Female	Home Maker	P	A	P	15.00	A	68.00	167.00	25.60	87.00	18.00	140.00	90.00
41	SSH00055	Shanthabai	58.00	Female	Home Maker	A	A	A	8.00	A	74.00	164.00	28.00	88.00	18.00	140.00	90.00
42	SSH00313	Govindh Roa	59.00	Male	Business	P	P	P	15.00	A	76.00	170.00	24.00	80.00	16.00	130.00	90.00
43	SSH00066	Honomavva	45.00	Female	Home Maker	P	A	A	10.00	A	80.00	165.00	27.00	86.00	18.00	110.00	80.00
44	25001055	Jainanna	45.00	Female	Worker	P	A	P	5.00	A	66.00	156.00	23.00	80.00	16.00	110.00	60.00
45	3626	LaxmiBai	55.00	Female	Home Maker	P	A	A	10.00	A	85.00	166.00	27.00	89.00	18.00	130.00	90.00
46	3691	Halema	60.00	Female	Home Maker	A	A	P	5.00	A	82.00	165.00	26.70	98.00	18.00	130.00	90.00
47	4062	SugalaBai	40.00	Female	Home Maker	P	A	P	5.00	A	76.00	166.00	24.00	96.00	18.00	100.00	60.00
48	4212	Zahidanasreen	44.00	Female	Home Maker	A	A	A	5.00	A	83.00	168.00	28.00	92.00	18.00	130.00	90.00
49	4068	LaxmiBai	51.00	Female	Home Maker	P	A	A	6.00	A	65.00	163.00	22.00	90.00	16.00	130.00	90.00
50	25001007	Shivappa	58.00	Male	Business	P	P	P	10.00	A	78.00	166.00	25.00	78.00	17.00	130.00	90.00

51	13910	Balachandra	56.00	Male	Business	P	P	P	15.00	A	78.00	156.00	22.00	72.00	15.00	135.00	85.00
52	13946	Pasha Makandar	55.00	Male	Farmer	P	P	A	10.00	A	75.00	152.00	25.00	80.00	17.00	130.00	80.00
53	16060	Dundappa	40.00	Male	EMPOLYE	P	P	P	5.00	A	68.00	158.00	26.00	78.00	15.00	110.00	60.00
54	17403	Gangappa	47.00	Male	EMPOLYE	P	P	A	6.00	A	75.00	166.00	23.00	78.00	18.00	130.00	70.00
55	18179	Vishnu	58.00	Male	Business	P	P	P	11.00	A	77.00	168.00	26.00	76.00	14.00	115.00	60.00
56	18902	Makutam saab	48.00	Male	Worker	P	P	P	8.00	A	88.00	170.00	22.00	82.00	15.00	140.00	80.00
57	18895	Ratna Singh	54.00	Male	Farmer	P	P	A	15.00	A	73.00	168.00	23.00	66.00	17.00	135.00	90.00
58	12162	Muneer Mulla	60.00	Male	Farmer	P	P	P	20.00	A	88.00	169.00	26.00	88.00	17.00	130.00	80.00
59	11753	Mallikarjun	55.00	Male	Farmer	P	P	P	10.00	A	82.00	165.00	22.00	82.00	14.00	125.00	60.00
60	12821	Ms Doulakoti	55.00	Male	Farmer	P	P	A	12.00	A	70.00	155.00	23.00	80.00	16.00	110.00	100.00
61	13123	Iprayabyi	52.00	Female	Home Maker	P	A	P	20.00	A	66.00	158.00	28.00	80.00	14.00	115.00	60.00
62	13710	Maruthi	51.00	Male	EMPOLYE	P	P	P	10.00	A	78.00	162.00	21.00	68.00	16.00	130.00	80.00
63	5151	Anil Kamble	53.00	Male	Business	P	P	A	8.00	A	77.00	162.00	28.00	88.00	18.00	140.00	90.00
64	8423	Jaibunniga	48.00	Female	Home Maker	P	A	P	14.00	A	68.00	168.00	24.00	68.00	17.00	135.00	80.00
65	8173	Fayyazammad	41.00	Male	Business	P	P	A	5.00	A	72.00	166.00	22.00	78.00	16.00	130.00	60.00
66	8405	Kamrunnisa	60.00	Female	Home Maker	P	A	A	20.00	A	75.00	164.00	22.00	66.00	18.00	120.00	90.00
67	9437	Anusaya Shahagr	54.00	Female	Farmer	A	A	A	12.00	A	80.00	180.00	28.00	88.00	15.00	120.00	80.00
68	9778	Bhimsigh Rathod	55.00	Male	Farmer	P	P	P	8.00	A	89.00	156.00	21.00	92.00	14.00	140.00	90.00
69	18335	Shanthabai	53.00	Female	Home Maker	P	A	P	8.00	A	79.00	168.00	22.00	78.00	18.00	120.00	85.00

70	251039	Baburai	57.00	Male	Worker	P	P	A	12.00	A	83.00	166.00	21.00	76.00	14.00	130.00	70.00
71	250112042	Prabakar	51.00	Male	Business	P	P	P	10.00	A	88.00	170.00	21.00	88.00	17.00	130.00	70.00
72	250113148	Kashimsaab	48.00	Male	Worker	P	P	P	6.00	A	79.00	158.00	22.00	64.00	15.00	140.00	80.00
73	283568	Basanna Gowda	52.00	Male	Farmer	P	P	P	5.00	A	68.00	168.00	22.50	76.00	18.00	130.00	80.00
74	290105	Kasinath	51.00	Male	Farmer	P	P	P	10.00	A	70.00	165.00	22.60	86.00	18.00	130.00	90.00
75	295755	Sidray	52.00	Male	BANK OFFICERS	P	P	P	5.00	A	72.00	166.00	24.00	82.00	16.00	110.00	70.00
76	SSH00071	Dodappa	60.00	Male	Farmer	A	A	A	20.00	A	78.00	168.00	24.00	88.00	18.00	130.00	90.00
77	SSH00070	Neelavva	59.00	Female	Home Maker	A	A	A	15.00	A	74.00	167.00	24.80	80.00	16.00	130.00	90.00
78	26381	ch Suryanarayana	58.00	Male	Farmer	P	P	P	10.00	A	76.00	170.00	23.00	86.00	18.00	110.00	90.00
79	SSH00285	Faradh Khan	52.00	Male	Business	P	P	P	8.00	A	72.00	175.00	24.00	80.00	18.00	130.00	90.00
80	2857/34682	KasthuriBai	55.00	Female	Home Maker	P	A	A	5.00	A	78.00	165.00	25.80	82.00	16.00	110.00	80.00
81	225541	Ramappa	45.00	Male	EMPOLYE	P	P	P	5.00	A	78.00	178.00	24.00	78.00	16.00	150.00	90.00
82	139568	Abdhul Razak	53.00	Male	Worker	P	P	P	10.00	A	76.00	176.00	23.00	86.00	18.00	90.00	70.00
83	8926	Govind Rao Hanamath	60.00	Male	Farmer	P	P	P	13.00	A	79.00	178.00	23.00	82.00	14.00	110.00	85.00
84	269737	Ambawwa	53.00	Female	Farmer	P	A	P	10.00	A	75.00	154.00	24.30	86.00	18.00	150.00	90.00
85	271333	Rayibai	51.00	Female	Home Maker	P	A	A	10.00	A	78.00	159.00	22.50	86.00	18.00	110.00	80.00
86	391721	Sadha Shiv	56.00	Male	Business	P	P	A	10.00	A	75.00	157.00	24.00	79.00	14.00	135.00	85.00
87	397014	Sidhi Ram	46.00	Male	Farmer	P	P	A	8.00	A	67.00	155.00	22.00	74.00	18.00	140.00	80.00
88	156433	Ashwini	56.00	Female	Home Maker	A	A	P	8.00	A	69.00	167.00	24.00	76.00	16.00	150.00	90.00

89	347322	F.I karajayi	60.00	Male	Farmer	A	A	A	10.00	A	53.00	154.00	22.10	82.00	18.00	140.00	90.00
90	5813	Shob Ramsing Rathod	43.00	Female	Home Maker	A	A	A	5.00	A	68.00	156.00	28.00	85.00	15.00	120.00	80.00
91	6298	Husainsab	58.00	Male	Farmer	P	P	P	10.00	A	72.00	167.00	26.00	88.00	18.00	130.00	90.00
92	6144	Bupalal Jumadar	60.00	Male	Farmer	P	P	P	12.00	A	75.00	168.00	23.00	80.00	16.00	125.00	70.00
93	6822	Putalabai Rathod	46.00	Female	Home Maker	P	A	P	15.00	A	66.00	162.00	22.00	88.00	18.00	130.00	80.00
94	7382	Shankar Gowdasami	50.00	Male	Farmer	P	P	P	17.00	A	78.00	169.00	23.00	80.00	18.00	140.00	70.00
95	13690	Shantabai	45.00	Female	Home Maker	P	A	A	12.00	A	75.00	168.00	21.00	64.00	18.00	130.00	90.00
96	293879	Gurulingappa	60.00	Male	EMPOLYE	P	P	A	5.00	A	70.00	170.00	23.20	80.00	18.00	120.00	70.00
97	294569	Vasudev	60.00	Male	EMPOLYE	P	P	P	10.00	A	65.00	171.00	22.50	86.00	16.00	110.00	80.00
98	298444	Badangi Sab	60.00	Male	Farmer	P	P	P	10.00	A	66.00	174.00	23.50	82.00	16.00	110.00	80.00
99	250113041	Bhagya Shree	50.00	Female	Home Maker	A	A	A	6.00	A	87.00	162.00	24.00	66.00	17.00	120.00	90.00
100	18911	Sidduaanna	45.00	Male	Worker	P	P	A	5.00	A	74.00	166.00	23.00	76.00	17.00	125.00	90.00
101	19597	Kauthuri Bhai	55.00	Female	Home Maker	P	A	P	10.00	A	76.00	164.00	24.00	72.00	16.00	110.00	70.00
102	19593	Prakash	56.00	Male	Business	P	P	P	10.00	A	77.00	154.00	22.00	80.00	17.00	130.00	80.00
103	20094	Goutham	44.00	Male	Business	P	P	A	5.00	A	79.00	156.00	26.00	92.00	16.00	140.00	90.00
104	20373	Prabhu Gowda	55.00	Male	Business	P	P	P	8.00	A	72.00	166.00	28.00	90.00	17.00	130.00	85.00
105	20626	Subash	50.00	Male	Business	P	P	P	6.00	A	80.00	168.00	26.00	88.00	15.00	135.00	75.00

SlNo	FBS mg/dl	HbA1c	TCholesterol mg/dl	TG mg/dl	LDLChol mg/dl	HDLChol mg/dl	ECG finding	ECG Interpret	2DECHO finding	2DEcho interpret	LVEF	Coronary angiography	CAG Interpret	TMT interpret	CAD (0=absent, 1=Present)
1	186.00	6.90	180.00	172.00	87.00	34.00	STEMI	Abnormal	IHD	Abnormal	40.00	DVD	Abnormal		1.00
2	210.00	7.80	146.00	132.00	83.00	36.00	Normal	Normal	Normal	Normal	40.00	Normal	Normal		0.00
3	216.00	6.80	216.00	260.00	120.00	40.00	Normal	Normal	Normal	Normal	65.00	Normal	Normal		0.00
4	130.00	6.40	177.00	165.00	65.00	34.00	STEMI	Abnormal	IHD	Abnormal	50.00	SVD	Abnormal		1.00
5	206.00	6.60	95.00	208.00	78.00	36.00	Normal	Normal	Normal	Normal	45.00	Normal	Normal		0.00
6	260.00	8.20	260.00	300.00	150.00	40.00	STEMI	Abnormal	IHD	Abnormal	40.00	TVD	Abnormal		1.00
7	160.00	7.20	160.00	190.00	80.00	34.00	NSTEMI	Abnormal	IHD	Abnormal	60.00	SVD	Abnormal		1.00
8	220.00	7.80	250.00	165.00	180.00	33.00	NSTEMI	Abnormal	IHD	Abnormal	35.00	DVD	Abnormal		1.00
9	175.00	6.80	114.00	158.00	54.00	28.00	Normal	Normal	Normal	Normal	40.00	Normal	Normal		0.00
10	156.00	14.10	177.00	94.00	85.00	33.00	Normal	Normal	Normal	Normal	55.00	Normal	Normal		0.00
11	177.00	8.30	211.00	256.00	102.00	35.00	STEMI	Abnormal	IHD	Abnormal	40.00	TVD	Abnormal		1.00
12	156.00	7.40	180.00	150.00	80.00	35.00	Normal	Normal	Normal	Normal	55.00	Normal	Normal		0.00
13	143.00	6.70	148.00	176.00	64.00	49.00	STEMI	Abnormal	IHD	Abnormal	45.00	SVD	Abnormal		1.00
14	154.00	7.60	220.00	280.00	136.00	34.00	STEMI	Abnormal	IHD	Abnormal	35.00	TVD	Abnormal		1.00
15	145.00	8.60	210.00	177.00	91.00	36.00	STEMI	Abnormal	IHD	Abnormal	50.00	DVD	Abnormal		1.00

16	176.00	9.00	205.00	178.00	164.00	40.00	NSTEMI	Abnormal	IHD	Abnormal	35.00	TVD	Abnormal	1.00
17	201.00	9.70	195.00	225.00	111.00	37.00	STEMI	Abnormal	IHD	Abnormal	50.00	SVD	Abnormal	1.00
18	136.00	7.70	162.00	171.00	120.00	38.00	STEMI	Abnormal	IHD	Abnormal	35.00	TVD	Abnormal	1.00
19	130.00	6.00	186.00	206.00	112.00	36.00	STEMI	Abnormal	IHD	Abnormal	50.00	TVD	Abnormal	1.00
20	176.00	6.70	180.00	220.00	110.00	30.00	STEMI	Abnormal	IHD	Abnormal	45.00	DVD	Abnormal	1.00
21	140.00	6.00	186.00	192.00	92.00	34.00	NSTEMI	Abnormal	IHD	Abnormal	50.00	SVD	Abnormal	1.00
22	176.00	8.90	177.00	156.00	82.00	40.00	STEMI	Abnormal	IHD	Abnormal	35.00	SVD	Abnormal	1.00
23	175.00	6.80	245.00	280.00	150.00	40.00	TWAVES INVERSIONS	Abnormal	IHD	Abnormal	60.00	DVD	Abnormal	1.00
24	146.00	8.60	220.00	176.00	111.00	32.00	STEMI	Abnormal	IHD	Abnormal	45.00	DVD	Abnormal	1.00
25	150.00	6.20	242.00	256.00	110.00	30.00	NSTEMI	Abnormal	IHD	Abnormal	55.00	DVD	Abnormal	1.00
26	157.00	6.20	180.00	157.00	97.00	31.00	STEMI	Abnormal	IHD	Abnormal	35.00	DVD	Abnormal	1.00
27	146.00	6.10	280.00	250.00	130.00	32.00	NSTEMI	Abnormal	IHD	Abnormal	50.00	DVD	Abnormal	1.00
28	136.00	6.10	184.00	210.00	110.00	30.00	STEMI	Abnormal	IHD	Abnormal	45.00	DVD	Abnormal	1.00
29	210.00	8.20	180.00	176.00	112.00	31.00	Normal	Normal	Normal	Normal	60.00	Normal	Normal	0.00
30	190.00	7.90	216.00	251.00	110.00	38.00	NSTEMI	Abnormal	IHD	Abnormal	35.00	TVD	Abnormal	1.00
31	166.00	8.20	210.00	175.00	82.00	40.00	Normal	Normal	Normal	Normal	55.00	Normal	Normal	0.00
32	209.00	7.80	201.00	156.00	101.00	44.00	STEMI	Abnormal	IHD	Abnormal	50.00	DVD	Abnormal	1.00
33	184.00	9.40	246.00	316.00	110.00	40.00	Normal	Normal	Normal	Normal	50.00	Normal	Normal	0.00
34	400.00	14.10	181.00	130.00	111.00	42.00	Normal	Normal	Normal	Normal	45.00	Normal	Normal	0.00
35	226.00	8.20	225.00	176.00	96.00	38.00	STEMI	Abnormal	IHD	Abnormal	45.00	TVD	Abnormal	1.00

36	178.00	8.90	192.00	156.00	75.00	34.00	Normal	Normal	Normal	Normal	55.00	Normal	Normal		0.00
37	130.00	8.20	210.00	176.00	92.00	32.00	Normal	Normal	Normal	Normal	45.00	Normal	Normal		0.00
38	130.00	6.10	173.00	151.00	101.00	36.00	Normal	Normal	Normal	Normal	55.00	Normal	Normal		0.00
39	155.00	6.60	260.00	185.00	120.00	50.00	Normal	Normal	Normal	Normal	55.00	Normal	Normal		0.00
40	218.00	8.20	256.00	312.00	150.00	35.00	STEMI	Abnormal	IHD	Abnormal	30.00	TVD	Abnormal		1.00
41	220.00	9.40	190.00	211.00	96.00	40.00	STEMI	Abnormal	IHD	Abnormal	40.00	SVD	Abnormal		1.00
42	190.00	9.10	187.00	216.00	85.00	36.00	STEMI	Abnormal	IHD	Abnormal	35.00	TVD	Abnormal		1.00
43	156.00	6.30	176.00	210.00	80.00	40.00	NSTEMI	Abnormal	IHD	Abnormal	60.00	SVD	Abnormal		1.00
44	190.00	7.60	210.00	186.00	104.00	40.00	Normal	Normal	Normal	Normal	40.00	Normal	Normal		0.00
45	211.00	8.10	172.00	156.00	92.00	36.00	NSTEMI	Abnormal	IHD	Abnormal	35.00	SVD	Abnormal		1.00
46	220.00	8.60	194.00	210.00	110.00	45.00	STEMI	Abnormal	IHD	Abnormal	45.00	TVD	Abnormal		1.00
47	160.00	6.10	183.00	181.00	117.00	30.00	Normal	Normal	Normal	Normal	60.00	Normal	Normal		0.00
48	329.00	7.20	273.00	115.00	110.00	26.00	Normal	Normal	Normal	Normal	55.00	Normal	Normal		0.00
49	166.00	6.80	186.00	196.00	112.00	45.00	STEMI	Abnormal	IHD	Abnormal	45.00	DVD	Abnormal		1.00
50	180.00	6.80	196.00	170.00	96.00	34.00	Normal	Normal	Normal	Normal	60.00	Normal	Normal		0.00
51	240.00	8.40	230.00	186.00	110.00	39.00	Normal	Normal	Normal	Normal	50.00	Normal	Normal		0.00
52	160.00	7.80	172.00	192.00	92.00	36.00	Normal	Normal	Normal	Normal	55.00	Normal	Normal		0.00
53	246.00	9.90	216.00	250.00	110.00	36.00	Normal	Normal	Normal	Normal	50.00	Normal	Normal		0.00
54	180.00	7.20	210.00	196.00	95.00	39.00	Normal	Normal	Normal	Normal	40.00	Normal	Normal		0.00
55	206.00	7.40	180.00	191.00	112.00	40.00	Normal	Normal	Normal	Normal	45.00	Normal	Normal		0.00
56	192.00	7.10	160.00	182.00	92.00	34.00	Normal	Normal	Normal	Normal	60.00	Normal	Normal		0.00
57	166.00	6.80	208.00	180.00	104.00	40.00	Normal	Normal	Normal	Normal	55.00	Normal	Normal		0.00
58	196.00	9.40	210.00	256.00	112.00	40.00	Normal	Normal	Normal	Normal	65.00	Normal	Normal		0.00
59	212.00	9.60	182.00	240.00	110.00	35.00	Normal	Normal	Normal	Normal	45.00	Normal	Normal		0.00
60	210.00	7.80	210.00	270.00	121.00	38.00	Normal	Normal	Normal	Normal	45.00	Normal	Normal	Positive	0.00
61	177.00	6.80	174.00	210.00	94.00	36.00	Normal	Normal	Normal	Normal	60.00	Normal	Normal		0.00

62	211.00	7.60	210.00	156.00	96.00	35.00	Normal	Normal	Normal	Normal	50.00	Normal	Normal		0.00
63	156.00	6.80	192.00	210.00	86.00	32.00	NSTEMI	Abnormal	IHD	Abnormal	55.00	SVD	Abnormal		1.00
64	186.00	7.60	176.00	216.00	112.00	34.00	NSTEMI	Abnormal	IHD	Abnormal	55.00	TVD	Abnormal		1.00
65	210.00	7.70	198.00	216.00	110.00	36.00	STEMI	Abnormal	IHD	Abnormal	35.00	TVD	Abnormal		1.00
66	216.00	8.60	216.00	200.00	90.00	36.00	STEMI	Abnormal	IHD	Abnormal	45.00	DVD	Abnormal		1.00
67	178.00	7.20	210.00	186.00	90.00	36.00	STEMI	Abnormal	IHD	Abnormal	30.00	TVD	Abnormal		1.00
68	192.00	8.10	216.00	186.00	92.00	36.00	Normal	Normal	Normal	Normal	60.00	Normal	Normal		0.00
69	170.00	6.80	192.00	161.00	84.00	39.00	Normal	Normal	Normal	Normal	45.00	Normal	Normal		0.00
70	157.00	6.80	166.00	140.00	100.00	40.00	Normal	Normal	Normal	Normal	60.00	Normal	Normal	Positive	0.00
71	196.00	8.00	216.00	180.00	90.00	30.00	Normal	Normal	Normal	Normal	50.00	Normal	Normal		0.00
72	170.00	6.50	196.00	180.00	94.00	40.00	Normal	Normal	Normal	Normal	60.00	Normal	Normal		0.00
73	256.00	8.60	205.00	147.00	140.00	44.00	TWAVES INVERSIONS	Abnormal	IHD	Abnormal	45.00	SVD	Abnormal		1.00
74	150.00	6.20	156.00	120.00	100.00	48.00	TWAVES INVERSIONS	Abnormal	IHD	Abnormal	50.00	DVD	Abnormal		1.00
75	186.00	7.40	194.00	101.00	83.00	44.00	STEMI	Abnormal	IHD	Abnormal	45.00	SVD	Abnormal		1.00
76	168.00	6.20	206.00	280.00	90.00	30.00	NSTEMI	Abnormal	IHD	Abnormal	45.00	DVD	Abnormal		1.00
77	160.00	6.10	170.00	210.00	110.00	40.00	STEMI	Abnormal	IHD	Abnormal	45.00	DVD	Abnormal		1.00
78	168.00	6.60	174.00	187.00	98.00	45.00	STEMI	Abnormal	IHD	Abnormal	50.00	TVD	Abnormal		1.00
79	212.00	9.40	180.00	230.00	122.00	40.00	STEMI	Abnormal	IHD	Abnormal	40.00	TVD	Abnormal		1.00
80	186.00	11.60	170.00	286.00	110.00	45.00	STEMI	Abnormal	IHD	Abnormal	30.00	TVD	Abnormal		1.00
81	188.00	6.60	178.00	143.00	76.00	35.00	Normal	Normal	Normal	Normal	60.00	Normal	Normal		0.00
82	136.00	6.50	156.00	111.00	80.00	36.00	Normal	Normal	Normal	Normal	60.00	Normal	Normal		0.00

83	181.00	7.90	186.00	271.00	130.00	30.00	NSTEMI	Abnormal	IHD	Abnormal	30.00	TVD	Abnormal		1.00
84	345.00	11.80	267.00	346.00	120.00	30.00	STEMI	Abnormal	IHD	Abnormal	35.00	TVD	Abnormal		1.00
85	135.00	8.10	176.00	211.00	90.00	32.00	STEMI	Abnormal	IHD	Abnormal	45.00	SVD	Abnormal		1.00
86	170.00	7.20	194.00	149.00	65.00	49.00	Normal	Normal	Normal	Normal	55.00	Normal	Normal		0.00
87	237.00	7.70	225.00	216.00	106.00	42.00	NSTEMI	Abnormal	IHD	Abnormal	60.00	TVD	Abnormal	Positive	1.00
88	230.00	8.50	185.00	206.00	94.00	39.00	NSTEMI	Abnormal	IHD	Abnormal	50.00	DVD	Abnormal		1.00
89	258.00	6.60	148.00	193.00	70.00	34.00	Normal	Normal	Normal	Normal	45.00	Normal	Normal		0.00
90	170.00	7.60	180.00	216.00	92.00	30.00	Normal	Normal	Normal	Normal	60.00	Normal	Normal		0.00
91	210.00	8.10	210.00	246.00	122.00	39.00	STEMI	Abnormal	IHD	Abnormal	55.00	TVD	Abnormal		1.00
92	186.00	7.60	186.00	290.00	116.00	36.00	STEMI	Abnormal	IHD	Abnormal	35.00	TVD	Abnormal		1.00
93	230.00	7.40	191.00	216.00	110.00	36.00	Normal	Normal	Normal	Normal	65.00	Normal	Normal		0.00
94	256.00	7.80	220.00	290.00	120.00	39.00	STEMI	Abnormal	IHD	Abnormal	45.00	TVD	Abnormal		1.00
95	188.00	7.60	177.00	156.00	86.00	32.00	Normal	Normal	Normal	Normal	60.00	Normal	Normal		0.00
96	178.00	7.00	240.00	286.00	106.00	44.00	NSTEMI	Abnormal	IHD	Abnormal	50.00	TVD	Abnormal		1.00
97	180.00	6.40	180.00	240.00	110.00	45.00	STEMI	Abnormal	IHD	Abnormal	45.00	SVD	Abnormal		1.00
98	186.00	7.20	186.00	216.00	90.00	35.00	STEMI	Abnormal	IHD	Abnormal	50.00	DVD	Abnormal		1.00
99	161.00	6.40	202.00	180.00	101.00	40.00	Normal	Normal	Normal	Normal	55.00	Normal	Normal		0.00
100	171.00	6.90	210.00	190.00	111.00	42.00	Normal	Normal	Normal	Normal	45.00	Normal	Normal		0.00
101	178.00	7.40	216.00	190.00	96.00	32.00	Normal	Normal	Normal	Normal	55.00	Normal	Normal		0.00
102	156.00	6.50	180.00	156.00	92.00	40.00	Normal	Normal	Normal	Normal	50.00	Normal	Normal		0.00
103	192.00	7.40	190.00	150.00	92.00	36.00	Normal	Normal	Normal	Normal	55.00	Normal	Normal		0.00
104	181.00	7.10	190.00	161.00	94.00	39.00	Normal	Normal	Normal	Normal	40.00	Normal	Normal		0.00
105	172.00	7.00	160.00	156.00	80.00	36.00	Normal	Normal	Normal	Normal	55.00	Normal	Normal		0.00