“A COMPARATIVE STUDY OF CLINICAL FEATURES AND ANGIOGRAPHIC PROFILE IN DIABETICS AND NON DIABETICS”

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ABBREVIATIONS

ALWMI	-	Anterolateral Wall Myocardial Infarction
ASWMI	-	Anteroseptal Wall Myocardial Infarction
AWMI	-	Anterior Wall Myocardial Infarction
CAD	-	Coronary Artery Disease
CAG	-	Coronary Angiography
DM	-	Diabetes Mellitus
DVD	-	Double Vessel Disease
ECG	-	Electrocardiogram
EF	-	Ejection Fraction
HbA1c	-	Glycated Hemoglobin
ILWMI	-	Inferolateral Wall Myocardial Infarction
IPWMI	-	Inferoposterior Wall Myocardial Infarction
IWMI	-	Inferior Wall Myocardial Infarction
LAD	-	Left Anterior Descending Artery
LCX	-	Left Circumflex Artery
LWMI	-	Lateral Wall Myocardial Infarction
LV	-	Left Ventricle/Left Ventricular
NSTEMI	-	Non-ST Elevation Myocardial Infarction
RCA	-	Right Coronary Artery
RWMA	-	Regional Wall Motion Abnormality
SD	-	Standard Deviation
STEMI	-	ST Elevation Myocardial Infarction
SVD	-	Single Vessel Disease
TVD	-	Triple Vessel Disease

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ABSTRACT

Background:

Diabetes mellitus is a major risk factor for coronary artery disease (CAD) and is associated with accelerated atherosclerosis, leading to significant cardiovascular morbidity and mortality. The present study aimed to compare the clinical features and angiographic profiles of CAD between diabetic and non-diabetic patients.

Methods:

This cross-sectional comparative study included 126 patients (63 diabetics and 63 non-diabetics) who underwent coronary angiography. Demographic characteristics, clinical presentations, electrocardiographic findings, echocardiographic parameters including ejection fraction and regional wall motion abnormalities, and detailed angiographic profiles were analyzed and compared between the two groups.

Results:

The majority of patients in both groups were in the 51-60 years age range with male preponderance. Chest pain was the predominant symptom in both groups, but diabetic patients showed a significantly higher prevalence of giddiness ($p=0.02$) and other atypical symptoms ($p=0.04$). Glycemic control assessment revealed that 49.2% of diabetic patients had HbA1c levels between 8.6-11%, with a significant association between higher HbA1c levels and CAD severity ($p=0.009$). Left ventricular dysfunction was more pronounced in diabetics, with a higher prevalence of reduced ejection fraction ($p=0.03$) and regional wall motion abnormalities ($p=0.05$). Angiographic findings demonstrated that diabetic patients had a significantly higher prevalence of triple-vessel disease (42.9% vs 23.8%) and double-vessel disease (27% vs 15.9%) compared to non-diabetics ($p<0.001$).

The left circumflex (63.5% vs 34.9%, p=0.001) and right coronary arteries (55.6% vs 36.5%, p=0.03) were more frequently involved in diabetics, suggesting a predilection for multi-vessel and diffuse CAD.

Conclusion:

Diabetic patients with CAD exhibit more atypical clinical presentations, more pronounced left ventricular dysfunction, and significantly more extensive, diffuse, and complex coronary artery involvement compared to non-diabetics. Poor glycemic control correlates with increased CAD severity. These findings emphasize the importance of early screening, aggressive risk factor modification, and appropriate revascularization strategies in diabetic patients with CAD.

Keywords:

Coronary artery disease, Diabetes mellitus, Clinical presentation, Angiographic profile, Multi-vessel disease, Glycemic control, Left ventricular dysfunction

INTRODUCTION

Diabetes mellitus represents a complex metabolic disorder characterized by profound systemic implications, particularly concerning cardiovascular pathophysiology.[1] The intricate relationship between diabetes and vascular alterations has emerged as a critical area of medical research, with significant implications for diagnostic and therapeutic strategies.[2]

Epidemiological data consistently demonstrate the escalating global prevalence of diabetes, highlighting its status as a major public health challenge. The World Health Organization estimates that diabetes affects approximately 463 million adults worldwide, with projections indicating potential increases to 700 million by 2045.[3] This dramatic surge underscores the urgent need for comprehensive understanding of diabetic pathophysiological mechanisms.

Cardiovascular complications represent the predominant cause of morbidity and mortality among diabetic patients. The intricate pathogenetic processes involve:

- Accelerated atherosclerosis
- Endothelial dysfunction
- Inflammatory cascade activation
- Oxidative stress enhancement
- Altered lipid metabolism[4]

Angiographic investigations provide critical insights into vascular structural and functional alterations associated with diabetes. Comparative analyses between diabetic and non-diabetic populations reveal distinctive morphological and hemodynamic characteristics that significantly impact clinical management strategies.[5]

Pathophysiological mechanisms underlying diabetic vascular alterations involve multiple complex interactions:

- Hyperglycemia-induced endothelial damage
- Advanced glycation end-product formation
- Inflammatory mediator dysregulation
- Oxidative stress amplification
- Impaired vascular repair mechanisms[6]

Clinical manifestations of diabetic vascular disease demonstrate substantial heterogeneity, necessitating comprehensive diagnostic and analytical approaches. Angiographic profiles offer invaluable information regarding:

- Vessel wall modifications
- Plaque morphology
- Stenosis progression
- Collateral circulation development[7]

The multifaceted nature of diabetic vascular pathology demands sophisticated investigative strategies. Comparative studies between diabetic and non-diabetic populations provide crucial insights into:

- Disease progression mechanisms
- Risk stratification
- Potential therapeutic interventions[8]

Emerging research suggests that early detection and comprehensive understanding of vascular alterations can significantly modify disease trajectory and patient outcomes. Detailed angiographic and clinical characterizations offer unprecedented opportunities for personalized medical approaches.[9,10]

AIM & OBJECTIVES

Objective:

1. To study clinical presentation and angiographic characteristics of coronary artery disease in diabetic patients compared to that of non-diabetics.

REVIEW OF LITERATURE

The “term diabetes mellitus is derived from the Latin word mellitus, which means sweet, and the Greek word diabetes, which means siphon, which means to pass through. According to a historical analysis, Apollonius of Memphis coined the term "diabetes" in the years 250–300 BC. When the ancient Greek, Indian, and Egyptian civilizations realized that urine in this condition was sweet, the term "diabetes mellitus" was born. In 1889, Mering and Minkowski made the discovery that the pancreas plays a part in the development of diabetes”.[11]

DEFINITION

The “term diabetes mellitus describes a metabolic disorder of multiple etiology characterized by chronic hyperglycemia with disturbances in carbohydrate, protein and fat metabolism resulting from defects in insulin secretion, insulin action or both”.[12]

HISTORY

Diabetes has a long history, as evidenced by the Ebers Papyrus, which dates to around 1550 B.C.

In the second century, Arateus of Cappadocia used the name "diabetes," which comes from the Ionian Greek word "to pass through," to refer to a condition that caused excessive urine production. He found that the diabetes was caused by a kidney condition, and he was unable to differentiate it from other non-diabetic conditions that manifested as polyuria.

Other names for diabetes, such as "diarrhea urinosa" and "dipsakos," were used by the Roman physician Galen (131–201), who later emphasized the cardinal symptoms of excessive thirst and drinking.[13]

“The link between polyuria and a sweet-tasting substance in the urine was originally noted by Sushruta and Charaka in the fifth and sixth centuries. The substance was described as having the flavor of lime honey (madhu meha), which was sticky to the touch and highly attracted to ants.

When Paul Langerhans (1847–1888) observed cell clusters in a pancreatic preparation that could be isolated from the surrounding exocrine and ductal tissue, he discovered the first hint of the hypoglycemic chemical.

In 1879, Oscar Minkowaski created diabetes in a dog by extracting an antidiabetic chemical from the pancreas.

These cell clusters were dubbed the Islet of Langerhans by Languesce (1893), and they make up the pancreatic endocrine tissue.[14] The second half of the 19th century saw the beginning of the clarification of the pancreas' ability to reduce blood glucose. The presence of the glucose-lowering hormone was still speculative when Jean de Meyer (1909) named it "insulin" (insula = island in Latin).[13]

After reading about the link between diabetes and pancreatic destruction, Canadian scientist Frederick Banting (1921), a failed orthopedic surgeon, became confident that he might discover an antidiabetic chemical. He eventually extracted it and tested it on a patient with remarkable results.

The main structure of insulin was described by Fredrick Sanger in 1955. Important discoveries about insulin secretion and some of the distinctions between IDDM and NIDDM were made possible by radio immunoassay in the 1960s. In 1977, the insulin gene was cloned, enabling the clinical use of human insulin and introducing molecular biology as a crucial new tool for diabetes research”.[15]

EPIDEMIOLOGY

An iceberg disease is diabetes. Ninety percent of adults worldwide have type 2 diabetes. T1DM begins to develop gradually after birth and peaks between the ages of 4 and 6 and again between the ages of 10 and 14.[16] About forty-five percent of children arrive before the age of ten. In those under 20, the prevalence is roughly 2.3 per 1000.[17] "There are no discernible gender differences in the incidence of childhood type 1 diabetes, despite the fact that most autoimmune disorders are more prevalent in women. Globally, the prevalence of type 1 diabetes has been rising. Rates are increasing by 2% to 5% each year in the Middle East, Europe, and Australia[18] T1DM rates in the US increased by almost 2% per year for the majority of age and ethnic groups, with rates higher among Hispanic youngsters. This pattern's precise cause is still unknown.[19]Although obesity in teenagers has increased the prevalence of type 2 diabetes in younger groups, the disease often manifests later in life. roughly 9% of Americans overall have type 2 diabetes, whereas roughly 25% of people over 65 have the disease.

According to the International Diabetes Federation, 1 in 11 persons worldwide between the ages of 20 and 79 had diabetes mellitus in 2015. By 2040, experts predict that the number of people with diabetes mellitus will rise from 415 to 642 million, with the largest increase occurring in those moving from low to middle income levels.15. In the US, Blacks, Native Americans, Pima Indians, and Hispanic Americans are 2–6 times more likely than Whites to have type 2 diabetes, though the prevalence varies by ethnic group. Environmental variables can significantly increase the incidence of type 2 diabetes, even if ethnicity alone is a significant contributor".[20]

Indian Scenario

"Nearly 25% of the world's population lives in South Asia, which is currently experiencing an epidemiological transition due to a sharp rise in the frequency of non-communicable diseases (NCDs). The biggest contributor to the burden of NCDs is India, the largest nation in the region. India has a high overall burden of diabetes, hypertension, and dyslipidemia, according to numerous research done over the past 20 years. According to a 2023 study by the Indian Council of Medical Research-India Diabetes (ICMR INDIAB), 10.1 crore people have diabetes.[21] An estimated 77 million adults over the age of 18 in India have type 2 diabetes, and almost 25 million are prediabetics, meaning they have a higher chance of getting the disease in the near future. Over 50% of people are not aware that they have diabetes, which can cause health issues if it is not identified and treated in a timely manner".[22]

CLASSIFICATION OF DIABETES MELLITUS (ETIOLOGICAL)[23]

I. "Type 1 Diabetes

Beta cell destruction, usually leading to absolute insulin deficiency

A- Immune mediated

B- Idiopathic.

II. Type 2 DM

May be ranging from predominantly insulin resistance with relative insulin deficiency to a predominant insulin secretory defect with insulin resistance.

III. Other specific types of diabetes

A. Genetic defect of beta cell function characterised by mutation in:

1- Hepatocyte nuclear transcription factor(HNF- 4) (Maturity Onset Diabetes Mellitus1)

2- Glucokinase (MODY 2)

3- HNF- 1 (MODY 3)

4- Insulin promoter factor -1 (PF- 1; MODY 4)

5- HNF-1(MODY-5)

6- NEURO D1(MODY SIX)

7- MITOCHONDRIAL(DNA)

8- Subunits of ATP- SENSITIVE potassium channel

9- Proinsulin or insulin

B. Genetic defect in insulin action

1- Type A insulin resistance

2- Leprechaunism

3- Rabson Mendensall syndrome

4- Lipodystrophy syndromes

C. Diseases of the exocrine pancreas – pancreatitis, pancreatectomy, neoplasia etc.

D. Endocrinopathies- acromegaly, cushing syndrome, glucagonoma, pheochromocytoma etc.

E. Drug induced DM- glucocorticoids , pentanamidine, nicotinic acid , diazoxide,

F. Infection – congenital rubella , cytomegalo virus

G. Uncommon form of immune mediated diabetes “stiff person syndrome” anti insulin receptor antibodies

H. Other genetic syndrome sometimes associated with diabetes

IV. Gestational Diabetes Mellitus”

ETIOPATHOGENESIS

- **Type 1 DM:**

Genetic, environmental, and immunologic variables interact to cause type 1 diabetes, which ultimately results in insulin insufficiency and the death of pancreatic beta cells. In identical twins, type 1 DM concordance ranges from 30% to 70%.Most people with type 1diabetes have signs of islet-direct autoimmunity, although not all do. Type 1 diabetes is caused by autoimmune beta cell death.

It is believed that an infectious or environmental stimulation initiates this autoimmune process, which is then maintained by a protein unique to beta cells. Most often, immunologic indicators show up after the triggering event but before diabetes manifests itself clinically. Diabetes symptoms don't show up until 80% of the beta cells have been damaged. Although there are still functional beta cells present at this stage, there are not enough of them to sustain glucose tolerance. Infections or puberty are the conditions that cause the shift from glucose tolerance to frank diabetes.Following the first clinical manifestation of type 1 diabetes, there may be a honeymoon period during which modest insulin dosages are sufficient to achieve glycemic control or, in rare cases, insulin is not required. “When the autoimmune process kills the remaining beta cells, the person becomes totally insulin deficient, ending the brief period of endogenous insulin production from remnant beta cells”.[23]

Autoimmunity:

“T lymphocytes reacting against as-yet-ill-defined beta-cell antigens are the main source of islet destruction in type 1 diabetes, an autoimmune illness that reduces beta cell mass. T lymphocytes harm cells by reacting to the antigens of beta cells. These T cells comprise CD8+ cytotoxic T lymphocytes, which destroy beta cells directly and also release cytokines that trigger macrophage activation, and CD4+ T

cells of the TH1 subtype, which damage tissue by stimulating macrophages. The islet exhibits cellular necrosis and lymphocytic infiltration in the rare instances where the pancreatic lesions were seen during the early, active phases of the illness. We refer to this lesion as insulitis. Beta cells are harmed by cytokines generated locally. IFN-δ, which is produced by T cells, and interleukin-1 and tumor necrosis factor, which are produced by macrophages that are activated during the immune response, are among the cytokines linked to cell injury. Seventy to eighty percent of patients also have autoantibodies against several beta cell antigens, such as insulin and glutamic acid decarboxylase, which can lead to islet destruction".[24]

Genetic Considerations:

Type 1 diabetes is caused by a combination of genes. The HLA region on chromosome 6 contains the primary susceptibility gene for type 1 diabetes. The HLA DR3 and DR4 halo types are present in the majority of people with type 1 diabetes. "The halo types DQA1*0301, DQB1*0302, and DQB1*0201 are most significantly linked to type 1 diabetes, according to improvements in HLA locus genotyping. Compared to 2% of the general population, 40% of children with type 1 diabetes had these halo kinds".

At least ten distinct genetic loci, in addition to MHC class II connections, contribute to the risk of "type 1 diabetes. These loci most recently include polymorphisms in the insulin gene's promoter region, the CTLA-4 gene, the interleukin-2 receptor, IFIH1, and PTPN22".[23]

Environmental Factors:[25]

Putative environmental triggers include viruses (coxsackie and rubella most prominently), bovine milk proteins, and nitrosourea compounds.

Pancreatic Toxins:[23]

Beta cell necrosis was observed within 48 hours after the injection of chemical agents such as "alloxan, dehydroascorbic acid, and different chelating agents such ethylene diamine tetracetic acid (EDTA) and 8-hydroxyquinoline. Benzothiadiazine ingestion may cause temporary hyperglycemia".

Viruses and Diabetes:[23]

In "animals at least two viruses have been described which directly damage the beta cells. A strain of foot and mouth virus and EMC virus are identified. In man there is an association of diabetes with mumps but this is rare".

Immunological Markers:[23]

"Islet cell auto antibodies (ICAs) are a composite of several different antibodies directed at pancreatic islet molecules such as GAD, insulin, and IA-2/ICA-512 and serve as a marker of the autoimmune process of type1 DM".

❖ **Type 2 DM:**[23]

"Type 2 diabetes is primarily caused by insulin resistance and aberrant insulin production. The majority of research backs up the theory that diabetes only arises when insulin secretion is insufficient, whereas insulin resistance occurs before an insulin secretory deficiency. Impaired insulin secretion, insulin resistance, increased hepatic glucose production, and normal fat metabolism are the hallmarks of type 2 diabetes. Type 2 diabetes is characterized by a high prevalence of obesity, especially visceral or central obesity as indicated by the hip-waist ratio.

Because the pancreatic beta cells adjust by producing more insulin, glucose tolerance remains close to normal in the early stages of the illness, even in the face of insulin resistance.In certain people, the pancreatic islets can no longer maintain the hyperinsulinemia as insulin resistance and compensatory hyperinsulinemia worsen".

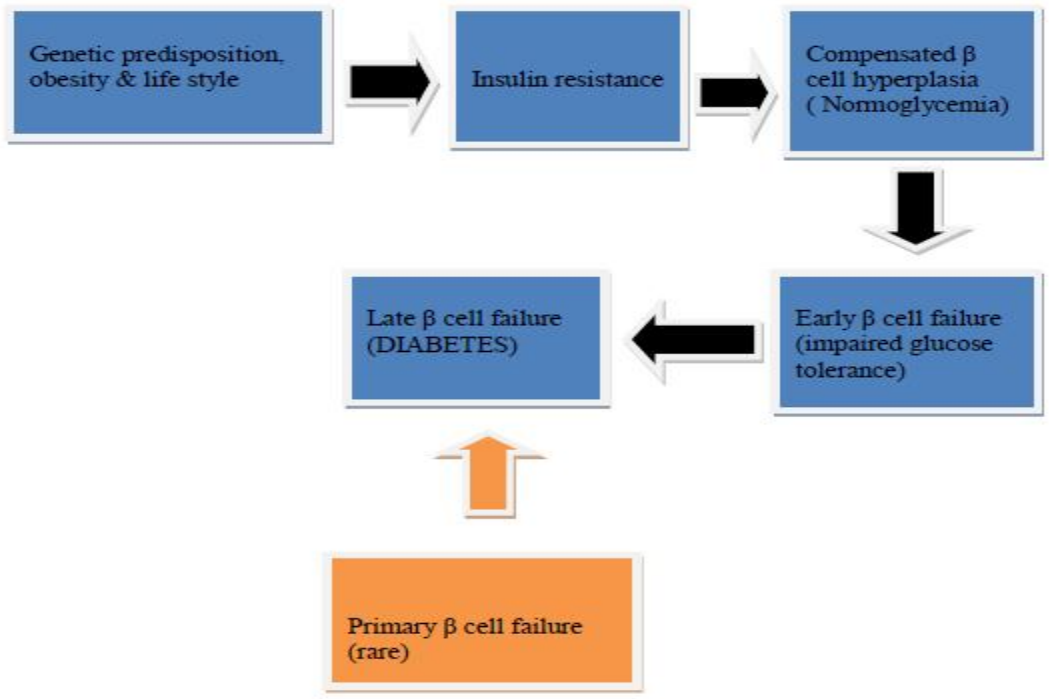
Genetic consideration:

There is a significant hereditary component to type 2 DM. In identical twins, type 2 DM concordance ranges from 70% to 90%. People who have a parent with type 2 diabetes are more likely to get diabetes themselves. Because environmental factors (such as obesity, nutrition, and physical activity) influence the phenotypic in addition to genetic predisposition, the disease is polygenic and multifactorial. "Type 2 diabetes has primarily been linked to a variation of the transcription factor 7-lie 2 genes. Genes encoding the peroxisome proliferators-activated receptor, inward rectifying potassium channel expressed in beta cells, zinc transporter expressed in beta cells, IRS, and calpain 10 have also been linked to genetic variants linked to type 2 diabetes".

Obesity and Insulin Resistance:

"Insulin resistance is the link between obesity and diabetes. The risk for diabetes increases as the body mass index (a measure of body fat content) increases, suggesting a dose- response relationship between fat and insulin resistance".[23]

Figure 1: Pathogenesis of Type 2 Diabetes Mellitus



Risk factors for type 2 Diabetes Mellitus:[23]

- "Family history of diabetes (i.e., patient or sibling with type 2 diabetes)
- Obesity (B I≥25 kg/m2)
- Habitual physical inactivity
- Race ethnicity (e.g., African American ,Hispanic American, Native American, Asian American, Pacific Islander)
- Previously identified IFG or IGT
- History of GDM or delivery of baby > 4 kg(>9lb)
- Hypertension (blood pressure ≥140/90 mmHg)
- HDL cholesterol level ≤ 35mg/dl(0.90mmol/L) and /or a triglyceride level≥250 mg/dl(282mmol/L)
- Polycystic ovary syndrome or acanthosis migrans
- History of vascular disease".

CLINICAL FEATURES

HISTORY[11, 26]

There are numerous symptoms associated with hyperglycemia. The table below compares the clinical characteristics of the two primary forms of diabetes. Patients with type 2 diabetes, many of whom are asymptomatic or have non-specific complaints like persistent fatigue and malaise, frequently do not exhibit the characteristic signs of thirst, polyuria, nocturia, and fast weight loss that are common in type 1 diabetes. Inquiring about autoimmune disorders, insulin resistance, and family history is essential for diagnosing diabetes mellitus.

Table 1: "Comparative Clinical Features of Type1 and Type 2 Diabetes mellitus"

	TYPE I	TYPE II
"Age at onset	< 40 years	>50 years
Duration of symptoms	Weeks	Months to years
Body weight	Normal or low	Obese
Ketonuria	Yes	No
Rapid death without treatment with insulin	Yes	No
Auto antibodies	Yes	No
Diabetes complications at diagnosis	No	25%
Family history diabetes	Uncommon	Common
Other autoimmune disease	Uncommon	Uncommon"

PHYSICAL EXAMINATION[11]

When a person with hyperglycemia is physically examined, they may exhibit poor skin turgor due to dehydration and a characteristic fruity breath odor, which is a sign of ketosis.

- Clinicians may see lethargy, nausea, vomiting, and Kussmaul respirations in patients with diabetic ketoacidosis (DKA).
- Hemorrhages or exudates on the macula may be seen during a funduscopy examination in a patient with diabetes mellitus. The retinal venules may seem dilated or obstructed in cases of frank diabetic retinopathy. Ophthalmologists are also concerned about the growth of new blood vessels because they can accelerate retinal hemorrhages and macular edema, which can lead to blindness.

- Despite the possibility of “similar presentations, T1DM and T2DM can be differentiated by clinical history and examination. Typically, people with type 2 diabetes are overweight or obese and exhibit symptoms of insulin resistance, such as acanthosis nigricans, which are velvety, hyperpigmented patches on the skin of the neck, axilla, or inguinal folds. Long-term hyperglycemia patients may experience numbness, neuropathic discomfort, repeated yeast infections, or blurred vision. At every appointment, the clinicians must inquire about any recent changes to the patient's foot skin”. The standard physical examination should include the diabetic foot examination, which includes the monofilament test.

DIAGNOSIS

- History
- Physical examination
- Elevated “serum glucose levels (fasting glucose >126 mg/dL, random glucose >200 mg/dL, or hemoglobin A1C >6.5%), with or without antibodies to insulin and glutamic acid decarboxylase (GAD”), are indicative of this history.
- “HbA1c tests and fasting glucose levels are helpful in the early detection of type 2 diabetes. If borderline, a glucose tolerance test can be used to assess blood glucose response to an oral glucose tolerance test (OGTT) as well as fasting glucose levels. A fasting blood glucose level of 100 to 125 mg/dL or a 2-hour post-oral glucose tolerance test (post-OGTT) glucose level of 140 to 200 mg/dL are indicative of prediabetes, which frequently precedes type 2 diabetes.[27]

- The American Diabetes Association (ADA) states that any of the following methods can be used to diagnose diabetes: a HbA1c reading of at least 6.5%; 126 mg/dL (7.0 mmol/L) or above in fasting plasma glucose (no food for at least 8 hours); A random plasma glucose level of 11.1 mmol/L or 200 mg/dL or higher in a patient exhibiting signs of hyperglycemia (polyuria, polydipsia, polyphagia, weight loss) or hyperglycemic crisis; a two-hour plasma glucose level of 11.1 mmol/L or 200 mg/dL or higher during a 75-g OGTT. The United States Preventative Service Task Force recommends screening overweight people between the ages of 40 and 70, but the American Diabetes Association advises screening everyone 45 and older regardless of risk.[28]

Trends in hyperglycemia and hypoglycemia can be detected by home glucose testing. Over a period of three months (the life of a red blood cell), the HbA1c test shows the degree of glycation caused by hyperglycemia. Diabetic nephropathy can be detected in its early stages by urine albumin testing. Serum lipid monitoring is recommended at the time of diagnosis since individuals with diabetes are also at risk for cardiovascular disease. Similarly, because hypothyroidism is more common, some advise checking thyroid health every year by measuring thyroid-stimulating hormone levels".[29]

Figure 2: Guidelines for Diagnosis of Diabetes Mellitus

Parameter	Normoglycemia(mg/dl)		Prediabetes (mg/dl)		Diabetes (mg/dl)
	WHO	ADA	WHO	ADA	
FPG	< 110	< 100	110-125 (IFG)	100-125 (IFG)	≥ 126
2-h PG	< 140		140-199 (IGT)		≥ 200
HbA1c	< 5.7%		5.7-6.4%		≥ 6.5%
Random plasma glucose*					≥ 200 (with symptoms of diabetes)

***WHO - World Health Organization, *ADA - American Diabetic Association**

COMPLICATIONS OF DIABETES MELLITUS [23]

I. "Acute complications of DM:

a) Diabetic Ketoacidosis

b) Hyperglycaemia hyperosmolar state"

II .Chronic complications of DM:

"Chronic complications of DM affect many organ systems and are responsible for the majority of morbidity and mortality associated with disease. Chronic complications can be divided into vascular and non vascular complications".

Microvascular:

Eye disease**:** Retinopathy (non proliferative/proliferative), macular edema,

Neuropathy: sensory and motor (mono and polyneuropathy) Autonomic

Nephropathy

Macrovascular:

Coronary artery disease

Peripheral vascular disease,

Cerebrovascular disease

Nonvascular:

Gastrointestinal (gastroperesis, diarrhea)

Genitourinary (uropathy, sexual dysfunction)

Dermatological Infections”

MECHANISM OF COMPLICATIONS:

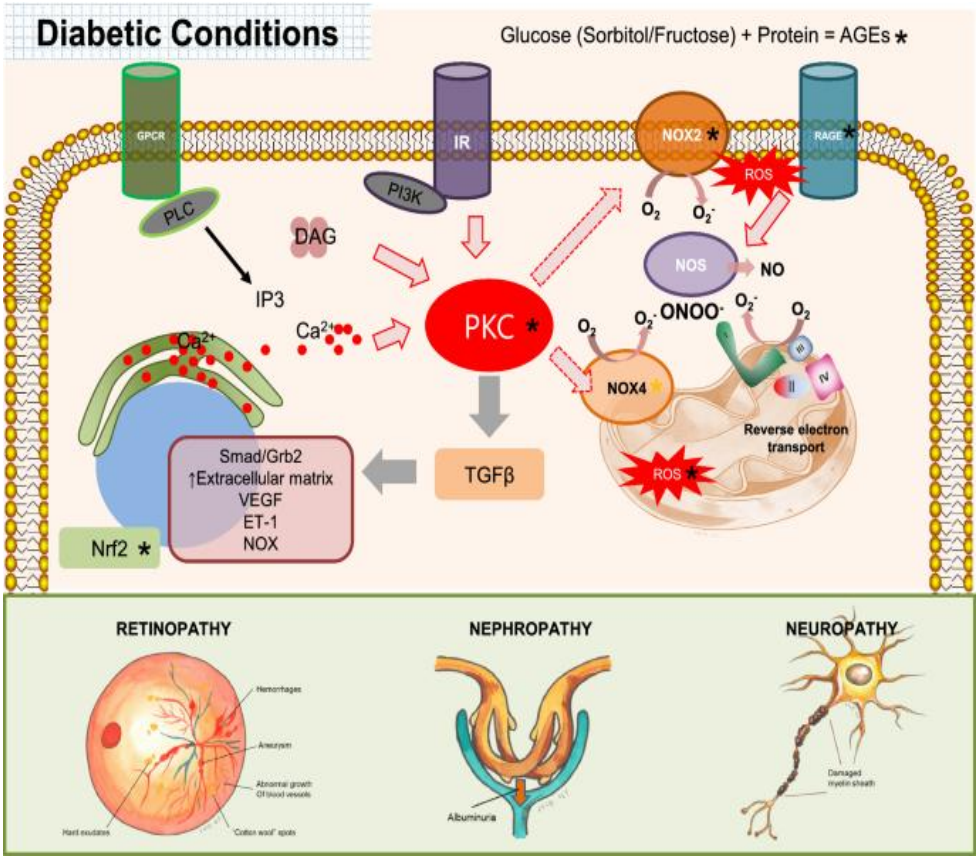
“Chronic hyperglycemia is a significant contributing factor to the complications of diabetes mellitus, however it is unclear what mechanism or mechanisms cause such a wide range of cellular and organ dysfunction. Four well-known theories have been put out to explain how hyperglycemia may contribute to the long-term consequences of diabetes mellitus; these theories are not exclusive of one another.

- The first theory states that non-enzymatic glycation of intracellular and extracellular proteins causes increased intracellular glucose to produce Advanced Glycation End products (AGEs).
- Using the sorbitol route, hyperglycemia raises glucose metabolism, according to the second idea.
- Third theory: Protein kinase C (PKC) is activated when diacylglycerol is formed more frequently due to hyperglycemia.
- The fourth explanation suggests that hyperglycemia increases the flow of fructose-6-phosphate, a substrate for O-linked glycosylation and the synthesis of proteoglycans, through the hexosamine pathway”.

The etiology of "diabetes and its consequences has been linked to excess free iron. Increased glycation of hemoglobin, which liberates the iron in its free state from heme, is the result of a poor glycemic regulated state". This creates a vicious cycle of elevated amounts of free iron, glycation of hemoglobin, and hyperglycemia. The oxidation of proteins and lipids as well as the production of reactive oxygen species are catalyzed by free iron (Fe3+, Fe+2).[30]

The harmful effects of transition metals, including as insulin resistance, are intensified when Advanced Glycated End (AGE) products attach to them. Reactive oxygen species (ROS) disrupt insulin signaling on multiple levels, preventing glucose transporter (GLUT-4) from translocating to the plasma membrane and affecting absorption directly through the insulin receptor. By lowering this chain of events, decreasing iron reserves might improve insulin resistance.[31, 32]

Figure 3: Mechanism of Diabetic Complications



CORONARY ARTERY DISEASES IN DIABETES

Epidemiology

Notably, T2DM makes for "over 90% of all instances of diabetes, and cardiovascular (CV) events in T2DM patients are a leading contributor to their elevated risk of dying young and are becoming a growing global health concern. Ischemic heart disease, heart failure (HF), stroke, coronary artery disease (CAD)", and peripheral artery disease are the main cardiovascular illnesses (CVDs) linked to type 2 diabetes. At least 50% of people with T2DM may die as a result of these complications. Data indicate a declining trend in the global prevalence of CVD due to "type 2 diabetes, despite the fact that the incidence of CVD among individuals with T2DM is 2-3 times higher than that of those without the disease.[33] Prior to 2016, the prevalence of all CVDs in people with type 2 diabetes varied between 14.3 and 46.9, but a meta-analysis conducted between 2007 and 2017 showed that the prevalence was 32%.3375% to 85% of persons with diabetes mellitus have hypertension, 70% to 80% have increased LDL, and 60% to 70% are obese. DM is linked to a 2–4 times higher risk of dying from heart disease, and CAD is the leading cause of death in both type 1 and type 2 DM. More than 70% of diabetics over 65 will pass away from heart disease or stroke".[34]

The Urban Population Study of Chennai (CUPS): In the CUPS trial, the overall population reported an 11% prevalence of CAD, "with 1.2% of patients experiencing a MI, 1.3% having Q-wave abnormalities, 1.5% having ST-segment abnormalities, and 7.0% having T-wave abnormalities. Since 1970, the prevalence of CAD in urban India has increased tenfold, to 11%, and is now on par with that of migratory Indians. The prevalence of CAD among patients with diabetes in the same study was 21.4% (known diabetes, 25.3%, and newly diagnosed diabetes, 13.1%).

This was significantly higher than the rates among subjects with IGT and NGT, which were 14.9% and 9.1%, respectively. In participants with diabetes, the prevalence of known MI was three times greater. Nonetheless, this investigation demonstrated that even during the IGT stage, the risk for CAD rose".[35]

In India, type 2 diabetes is about to spread like wildfire.[36] Since coronary artery disease (CAD) and type 2 diabetes share a number of risk factors, including age, hypertension, dyslipidemia, obesity, stress, and physical inactivity, a rise in the prevalence of diabetes also indirectly indicates a rise in the risk of CAD. "CAD has been found to arise two to three decades earlier in diabetic participants than in their nondiabetic counterparts, and diabetic subjects are known to have a two to four times higher chance of developing CAD".[37]

Due to higher mortality, individuals with diabetes have an almost eight-year shorter life expectancy. More than 80% of all deaths and 75% of all hospitalizations in diabetic people are caused by coronary artery disease. Additionally, it has been found that those with diabetes have a higher risk of plaque rupture.[38]

Pathophysiology of CAD in DM

Hyperglycemia

T2DM is seen as a complex illness including anomalies in the metabolism of fats and carbohydrates. The most obvious sign of it is persistently high blood sugar. Epigenetic and post-translational changes in the vascular architecture are defective in T2DM patients, according to recent data. Diabetes's numerous endometabolic abnormalities affect "glucose and lipid metabolism, including cellular toxicity and the effects of palmitic acid and other saturated fatty acids on the insulin receptor (IR), as well as behaviors like overfeeding (leptin resistance or deficiency)".[39] White adipocytes release the protein leptin, which increases energy expenditure by binding

to its corresponding receptor (leptin receptor B) and promotes postprandial satiety. Overfeeding behaviors are caused by "mutations in either the leptin protein (biologically inactive) or its receptor (defective activation), which results in significant obesity phenotypes linked to peripheral insulin resistance and hyperglycemia".[40]

Similarly, it has been suggested that saturated fatty acids, including palmitic acid, desensitize to insulin in both the brain and peripheral organs. Both central and peripheral insulin resistance are brought on by this dual effect, which causes hyperglycemia and a disruption of the body's energy balance. Islet β cells undergo apoptosis as a result of chronic insulin resistance, as well as the effects of lipoproteins, saturated fatty acids, leptin, and circulating proinflammatory cytokines.[41] Postprandial glucose levels stay continuously elevated for extended periods of time due to the organism's cells' poor absorption of glucose, "which leads to glucose-related tissue toxicity [production of receptor for advanced glycation end-products (RAGE), endothelial dysfunction, histone hyperacetylation, DNA methylation, etc.].

This toxicity has detrimental clinical effects on the nephrons (microalbuminuria and impaired glomerular filtration), nerves (peripheral neuropathy), and microvessels and macrovessels (retinopathy, coronary artery disease, etc.). In individuals with peripheral insulin resistance, IR agonists including chaetochromin derivatives and monoclonal antibodies with agonist action on the IR have been shown to enhance Akt activations and IR responsiveness, respectively. This improves glucose metabolism at the cellular level".[42]

Chronic inflammation and thrombosis

Tumor necrosis factor-α, interleukin (IL)-6, IL-12, IL-10, and other proinflammatory cytokines are produced and released as a result of "glucose toxicity

caused by aldose reductase activation, which also triggers subsequent PKC-dependent nonosmotic nuclear factor (NF)-κB activation. Similarly, adipocytokines such adipsin, adiponectin, leptin, tumor necrosis factor-α, and plasminogen activator inhibitor I are released when inflammation occurs in adipose tissue. Transduction signals from pathways linked to inflammation, obesity, and insulin have an impact on the vascular redox state. Crucially, when adipose tissues detect paracrine indicators of cardiovascular (CV) oxidative stress or injury, they can alter the secretory profile.[43] Through toll-like receptor (TLR) signaling, these inflammatory signals can transduce cellular signals in tissues such fat, liver, muscle, heart, endothelium, etc. This, in turn, activates inflammatory nuclear factors (NF-κB), which feeds the chronic cycle of persistent inflammation. TLR-2 and TLR-4 specifically influence the expression of inflammatory genes and cytokines (IL-12, monocyte chemoattractant protein-1, etc.) as well as the frequency, size, and lipid content of atherogenic plaque. Another excellent example of an anti-inflammatory medication that lowers recurrent CV events regardless of lipid levels is canakinumab, a monoclonal antibody against IL-1b, as demonstrated in the CANTOS research".[44]

For example, by activating platelets of both traditional and alternative routes, circulating inflammatory substances might trigger potentially fatal cell signaling, including thrombosis. In reaction to these circulating cytokines, platelets are readily activated and have the ability to aggregate quickly, particularly in low-flow regions like the brain, lower extremities, and coronaries. The risk of stroke and MIs is increased when these vessels are blocked or partially blocked, which can cause infarctions or necrosis of vital organs like the heart and brain.[45]

Local signals, plaque erosion, partial or complete rupture, and circulating inflammatory signals can all cause "thrombosis in the atherogenic suboccluded area

or distant regions on that arterial territory in vessels with an atherogenic lesion. Even though immune cells can repair and replace tissue, their presence and the release of inflammatory chemoattractive substances exacerbate the thrombotic state and raise the risk of additional plaque core necrosis and plaque instability, which can lead to the release of debris into the distal portions of the artery lesion. This condition exacerbates under low shear stress conditions".[46]

Dyslipidemia and atherogenesis

Patients with diabetes mellitus frequently have dyslipidemia and obesity, which can promote atherogenesis and atherosclerosis. In essence, the "packed energy" found in fat droplets in cells—particularly adipocytes—is what our bodies may use as fuel during periods of fasting or when increased physical activity is required. On the other hand, either anaerobic or aerobic mitochondrial pathways are used to convert carbohydrates into energy. "De-novo lipogenesis" is the process by which lipids are created from carbohydrates when all of the cell's basic energy needs are satisfied. Within the cell, lipids can be stored and transformed back into pyruvate, a substance that can be burned.[47] Because of their great affinity for cell membranes and ability to enter cells with little effort (via vectors-exosomes), lipids can themselves serve as a "source of energy" during periods of fasting. However, proteins help distribute lipids because of their physicochemical characteristics, which prevent early absorption and allow them to stay in the circulatory system. The liver is primarily responsible for coordinating the synthesis of those proteins. These proteins are divided into three groups based on their molecular density: high-density lipoprotein, LDL, and very low-density lipoprotein (LDL).

The circulatory system carries lipoproteins to distant organs and tissues together with triglycerides, which are collections of lipids.[48]

The development of atherogenesis has been linked to persistently elevated levels of atherogenic “LDL cholesterol as well as elevated non-high-density lipoprotein C and ApoB levels in patients. Low-density lipoprotein (oxLDL) oxidation is a significant condition that reflects oxidative stress and enhances the inflammatory and atherogenic characteristics of LDL. Furthermore, there is a correlation between the occurrence of coronary disease and elevated serum levels of oxLDL.[49] Thus, lowering LDL cholesterol with statins or new proprotein convertase subtilisin/kexin type 9 inhibitors makes sense as a therapeutic goal. By preventing the conversion of hydroxymethylglutaryl-coenzyme A into mevalonic acid (primitive fatty acid), statins reduce the synthesis of cholesterol. On the other hand, as the GLAGOV study recently shown, proprotein convertase subtilisin/kexin type 9 inhibitors enhance the density of LDL-receptors on the cell surface, which facilitates the cell's absorption of LDL and reduces circulating LDL, thus promoting plaque regression”.[50]

At the same time, vascular stiffness and atherosclerosis are facilitated by hyperglycemia. The development and stability of the plaque are significantly influenced by endothelial degradation throughout time as well as the impact of inflammatory cytokines on the endothelium. Epigenetic mechanisms and posttranslational modifications control “the cellular mechanisms of media thickening and proliferation, the presence of endothelium-adhesion molecules (vascular cell adhesion molecule 1 and intercellular adhesion molecule 1), and macrophage infiltration in the subintima. Among other conditions, hyperglycemia causes hyperacetylation of histone H3K9/K14 in 88 genes that code for diabetes, 52 genes that code for hypertension, and 84 genes that code for cardiovascular problems. Important glucose metabolism and metalloproteinases that regulate genes like heme

oxygenase 1 (HMOX1), IL-8 precursor, matrix metalloproteinase (MMP) protein-10, cysteine/glutamate transporter (SLC7A11), and MMP1 are expressed in the endothelium when the histone H3K9/K14 is hyperacetylated. Because they both participate in vascular remodeling, specifically in the growth and instability of plaque, and are regulated by proinflammatory signals, metalloproteinases and ILs are closely connected. Although MMP inhibitors have been used to stabilize plaques, more precise targeting of MMPs is required because broad-spectrum inhibitors affect the plaque in two ways".

"Hyperglycemia also induces DNA methylation of important genes for glucose metabolism, G-coupled protein receptors (GPRs), and insulin growth factor proteins such as ABCC11, ADAD1, ADAM8, BCL3, CCDC61, CEP120, CSF1R, CSTL1, CTTNBP2NL, EGLN3, ENOX1, ERAS, FAM107A, FASLG, GADD45B, GNG2, GPR39, GPR62, GRK7, HMGB2, HNRNPL, HYOU1, and IGFBPL1. Gene expression and suppression persist for up to 6 days in the endothelium after the hyperglycemic episode in vitro. Here is the importance of novel GPR agonists which currently are underway in an effort to improve GPR signaling in tissues and its metabolic benefits in patients with diabetes".

"Other epigenetic mechanisms such as microRNAs (miR) can regulate gene expression post-transcriptionally, directly exert their effects in signal pathways, and reach other cells when included in extracellular vesicles called 'exosomes'. miR-941, miR-208b, miR-197, and miR-223 have been found to have diagnostic value in predicting CV events or CV death. miR-126-5p has been associated inversely with the complexity of CAD with low serum levels in multivessel disease and high SYNTAX scores in patients with stable angina. Some epigenetic therapies are underway as potential antithrombotics such as miR-19b for use in patients with unstable angina.

Also, a bigger epigenetic factor, long noncoding RNAs in exosomes, such as exosomal long noncoding RNA-growth arrest-specific 5 (long noncoding RNA GAS5), can increase the apoptosis of macrophages and endothelial cells in atherosclerosis".[51]

Hypertension

"Since the origin of initial hypertension is uncertain, the renin-angiotensin-aldosterone system has been suggested as a viable model to explain secondary hypertension. By impairing energy metabolism (mitochondrial dysfunction) and the release of endothelial nitric oxide synthase, a crucial vasodilator, inflammatory cytokines significantly alter the endothelium, influencing vascular relaxation and causing arterial stiffness. These inflammatory cytokines chemoattract lymphocytes and macrophages, which can release angiotensin II (AngII) and reactive oxygen species. AngII increases blood flow by causing constriction of the artery medium, which raises blood pressure, while reactive oxygen species trigger NF-κB activation, intensifying the vicious cycle of the local inflammatory response. Because of the chronic nature of this inflammatory stage, AngII can continuously and reliably raise blood pressure. The development of media hypertrophy brought on by this high-flow system further shrinks the arterial lumen, raising resting blood pressure readings. If left untreated, this condition can lead to secondary hypertension and necessitate medical intervention with antihypertensives and lifestyle modifications".

"The risk of atherogenic plaque erosion or rupture, bleeding (particularly microcirculation), and thrombosis is increased when a high-flow system persists in conjunction with inflammation, dyslipidemia, and hyperglycemia.

Catecholamines' sympathetic control of blood pressure is also crucial for the occurrence and maintenance of hypertension. Targeting the renin-angiotensin-

aldosterone system may be more clinically useful than the sympathetic pathway, which is why renal denervation was suggested as a treatment for uncontrolled hypertension. However, the SYMPLICITY HTN-3 study did not yield relevant and consistent findings".[51]

PATTERN OF CAD IN DIABETIC PATIENTS

Vessel calibre

Women typically have smaller coronary arteries, which are correlated with body mass index. Thirteen diabetic patients with normal angiograms also had considerably smaller arteries than controls, according to a small study. Following CABG, a small vessel size is highly linked to an elevated risk of in-hospital death. Additionally linked to a higher risk of restenosis and the requirement for repeat revascularization following PCI is a smaller target vessel size. After controlling for sex and other clinical factors, it has been suggested that this connection explains why women and smaller patients have a higher procedural risk.

Vessel involvement

"The number of diseased vessels predicts future cardiac morbidity and mortality. There is convincing evidence that diabetic patients have a higher incidence of multivessel disease".

Location of lesions

Prognostically significant proximal segments and ostial disease are linked to a higher incidence of major adverse cardiac events (MACE) following PCI and a decreased risk of operative success. Whether these lesions are more prevalent in NIDDM patients remains unknown. NIDDM is linked to a higher incidence of left main stem illness.

Type of lesions

A higher incidence of MACE is linked to “lesions at the bifurcation of two epicardial veins, which pose a technical difficulty to the interventionist. Whether diabetic persons are more likely to have these lesions is unknown”. Similarly, higher rates of MACE and poorer procedural outcomes are linked to total occlusions. According to certain research, patients with diabetes had more total occlusions.

Collateral circulation

One crucial cardioprotective mechanism that the endothelium is believed to mediate in response to the development of substantial myocardial ischaemia is the establishment of a collateral coronary circulation. It has been demonstrated that DM impairs the formation of collateral vessels. It is uncertain, therefore, how this affects results or whether endothelial activation and inflammatory indicators are related.

Coronary artery calcification

The development of calcification coincides with the onset of coronary atherosclerosis. “Increased coronary artery calcification scores as assessed by electron beam computed tomography are linked to both insulin resistance and NIDDM. There is debate on the usefulness of measuring coronary artery calcification in risk stratification”. However, there is a higher chance of MACE following PCI and a lower chance of procedural success when PCI is performed on a calcified lesion.

SEVERITY OF CAD IN DIABETIC PATIENTS

Few would contest that CAD is more severe and diffuse in diabetic individuals, but how thoroughly has this been described, and what can we infer from it?The frequency of major stenoses and the degree of atheroma impacting the coronary tree are useful indicators of disease severity. “According to a multivariate analysis of more than 15,000 patients in the Coronary Artery Surgery Study (CASS), there is a slight

independent correlation between having diabetes mellitus and having more severe CAD. The majority of angiographic and postmortem investigations concur that patients with NIDDM have more severe CAD. Some research, though, has not discovered any distinction. Low patient numbers, inadequate study design, and technical difficulties in measuring the severity of the condition could all contribute to these conflicting findings. The accuracy of quantitative coronary angiography as a tool for assessing coronary severity has been established." Quantitative coronary angiography has only been utilized in two studies to assess the severity of CAD in NIDDM patients. Both discovered that the diabetes groups had more severe CAD.It has also been demonstrated that IDDM is linked to more severe illness.The development of atherosclerotic plaque that blocks over 70% of the coronary channel lumen is typically the cause of stable angina symptoms, which are plainly seen during coronary angiography. The severity of stenosis is linked to an increased risk of coronary heart disease. However, because to their far higher frequency, mild to moderately severe plaques are more commonly linked to acute coronary syndromes. In contrast to less extensive disease with more severe stenoses, this implies that diffuse and extensive disease may have more prognostic importance. The claim that diabetes people have more lesions producing severe obstruction is supported by the majority of the data. Additionally, diabetic CAD has a higher atheroma burden and is more diffuse. Higher mortality is correlated with more severe CAD in diabetes patients.35 Given these findings, it is reasonable to assume that a significant contributing factor to the worse outcomes following PCI is the advancement of the disease. Therefore, it stands to reason that any pharmacological interventions that lessen the severity of the condition would result in better outcomes.

FACTORS IMPLICATED IN MODULATING DIABETIC CAD SEVERITY

Factors that modulate the severity of CAD

- “Sex
- Ethinicity
- Lipids
- Insulin resistance
- Inflammation
- Hyperglycaemia

Sex

This was especially noticeable in female diabetic patients, according to several studies documenting increased disease severity. It is hypothesized that the cardioprotection shown in premenopausal women may be lost. Although it is uncertain if this is significant, it has been demonstrated that insulin influences the release of sex hormones.

Ethnicity

Compared to white people, the mortality rate from CAD is 40% greater in several ethnic groups. “Diabetic patients from the Middle East and India were the subjects of two research. They provide no conclusive evidence on the existence of a distinct pattern, but they do support the idea of elevated CAD. Although there was no indication of a more diffuse pattern of disease, diabetic patients had higher coronary artery scores, indicating increased severity. Three vessel disease was more prevalent in them.Asians and whites have the same CAD score, according to a tiny study. To find out how ethnic variance affects the severity of CAD, more research is needed”.

Lipids

The quantity of "triglyceride-rich lipoprotein particles and the concentration of plasma Lp(a) lipoprotein in NIDDM patients are associated with the severity of angiographic CAD. Angiographic disease severity was found to be negatively correlated with a subtype of high density lipoprotein and favorably correlated with intermediate density lipoprotein" in a different study that looked at patients with NIDDM. Numerous previous research may have been confounded because they failed to consider lipid profiles.

Insulin resistance

A small study conducted in Japan found a link between the severity of CAD in non-diabetic patients and a biochemical indicator of insulin resistance. The relationship between insulin resistance and the severity of the disease in NIDDM patients is uncertain.

Inflammation

Elevated serum C reactive protein indicates the degree of carotid artery atherosclerosis and is linked to increased coronary risk. Given the contradictory findings of two investigations, the relationship between the concentration of C reactive protein and the severity of CAD is still unknown. Atherogenesis is linked to the upregulation of endothelial adhesion molecules like VCAM-1. It is unknown, therefore, whether soluble VCAM-1 concentration and CAD severity are related.

Hyperglycaemia

Increased disease severity is linked to rising hyperglycemia, as indicated by the proportion of glycosylated hemoglobin A1c. Despite this, there is ongoing debate on the significance of glucose-lowering therapies in lowering cardiovascular risk and their impact on post-PCI outcomes.According to preliminary findings from the

UKPDS (UK prospective diabetes study), strict glycaemic management had very little effect on macrovascular disease. The risk of myocardial infarction decreased by 14% for every 1% decrease in hemoglobin A1c. However, the study's ability to identify a statistical decrease was limited by the comparatively small number of macrovascular events that were documented. It has been demonstrated that in individuals with diabetes mellitus, poor glycaemic management as indicated by glycosylated hemoglobin A1c levels at the time of PCI is an independent predictor of restenosis. Further research is necessary to fully understand the significance of strict glycaemic control following PCI, but it is probably linked to better results. This problem will be resolved via BARI 2D (bypass angioplasty revascularization investigation).

Diabetic CAD may also be influenced by some poorly understood criteria in addition to the more well-known risk factors. The relationship between insulin resistance, inflammation, and the severity of CAD needs further research. It is commonly known that decreasing cholesterol might lead to plaque regression and better results. Similarly, pharmacologically modifying insulin resistance and inflammation may lessen the severity of the condition in these individuals.

REVIEW OF RELATED STUDIES

Melidonis A et al (1999)[52] "Two groups of individuals with angiographically proven CAD were included in this randomized research. There were 463 diabetics in Group A, who were 60.3 years old, and 210 non-diabetic patients in Group B, who were 58.5 years old. Age, sex, weight, and traditional risk variables were all matched between the two groups. The authors assessed coronary artery lumen width, left ventricular (LV) function, and the geographical localization of CAD. One-vessel disease was less common ($p<0.001$) and three-vessel disease was more common ($p<0.001$) in the diabetics. In Group A, the CAD was more widespread (mean 2.2 vessels, $p<0.01$) than in Group B (mean 1.8 vessels). Both the anterior descending artery with three-vessel disease ($p<0.05$) and the right coronary artery ($p<0.01$) were more frequently impacted in diabetics. While the female diabetics had a greater LV ejection fraction ($p<0.05$), the male diabetics had the same angiographic CAD severity. The CAD findings of the female diabetics under the age of 55 were similar to those of the women in Group B who were 4 years older. Compared to the general population, diabetics exhibit more severe and diffuse CAD. The severity of CAD is not influenced by a person's sex".

Gui MH et al (2009)[53] "The goal of the current investigation was to compare the coronary artery angiographic profiles of diabetic CAD patients to those of nondiabetics. With a significantly lower insulin secretion index (Homa-IS) level ($p<0.001$), diabetic CAD patients had significantly higher waist to hip ratios (WHR) ($p=0.016$), fasting plasma glucose (FPG), 2h plasma glucose (2hPG), glycosylated hemoglobin (HbA1c) ($p<0.001$), insulin resistance index (Homa-IR) ($p=0.001$), and apolipoprotein A (ApoA) ($p=0.008$). In addition to having a substantially higher cumulative coronary atherosclerosis score (CAS) ($p=0.003$), diabetic patients were

more likely to have three-vessel disease (35.2% vs. 24.0%, p=0.009) and less likely to have one-vessel disease (28.8% vs. 46.2%, p<0.001). In diabetics, the right coronary artery was implicated much more frequently (66.4% vs. 52.6%, p=0.002), and the CAS was also noticeably greater (p=0.002). They came to the conclusion that, in comparison to nondiabetics, diabetics had more severe and diffuse coronary artery disease as seen by angiograms. In the diabetics, the right coronary artery was implicated far more frequently. In the current investigation, ApoA was the protective factor for CAD, whereas Homa-IR, diabetes mellitus, and duration of CAD were the independent risk factors".

Chu, Zg et al (2010)[54] "The purpose of this study is to use coronary CT angiography (CTA) to detect the characteristics of CAD in diabetic patients. Plaques were discovered in 470 segments (4.2 ± 2.8 per patient) and 287 coronary vessels (2.5 ± 1.1 per patient). The left anterior descending (LAD) artery (35.8%) and its proximal portion (19.1%) were most commonly affected (all $p < 0.001$), and multi-vessel disease was more common than single vessel disease ($p < 0.001$). The most prevalent type was calcified plaques (48.8%; $p < 0.001$), which were followed by mixed plaques (38.1%). Mild narrowing (36.9%) was the most prevalent of the various stenosis degrees ($p < 0.001$), although there was no discernible difference between obstructive and non-obstructive stenosis (50.4% vs. 49.6%, $p = 0.855$). Male and female diabetes patients did not differ significantly in terms of the extent of CAD, plaque types, or luminal narrowing. They came to the conclusion that patients with type 2 diabetes had a high plaque burden on coronary CT scans. The proximal portion of the LAD artery had a higher frequency of plaques, which were primarily calcified, and the high incidence of obstructive stenosis warrants additional attention. Furthermore, DM decreased the sex difference in CAD CT results".

“The prevalence, extent, severity, and prognosis of coronary artery disease (CAD) among people with and without diabetes (DM) who have similar CAD risk factors were investigated by **Rana JS et al. (2012)**[55]. 108 (3.2%) and 115 (1.7%) fatalities among DM and non-DM persons, respectively, occurred during a 2.2-year follow-up. DM patients had lower rates of having normal arteries (28 vs. 36%) and higher rates of obstructive CAD (37 vs. 27%) than non-DM patients ($P < 0.0001$). Per-segment stenosis in the proximal and mid-segments of every coronary artery was higher in DM patients than in non-DM patients for obstructive one-vessel disease (19 vs. 14%), two-vessel disease (9 vs. 7%), and three-vessel disease (9 vs. 5%) ($P < 0.0001$ for comparison). A higher risk of death was associated with non-obstructive CAD (5.25 [2.56-10.8]; $P < 0.001$), one-vessel disease (6.39 [2.98-13.7]; $P < 0.0001$), two-vessel disease (12.33 [5.622-27.1]; $P < 0.0001$), three-vessel disease (13.25 [6.15-28.6]; $P < 0.0001$), and non-DM individuals without CAD (hazard ratio 3.63 [95% CI 1.67-7.91]; $P = 0.001$). They came to the conclusion that DM people have a higher prevalence, severity, and extent of CAD than matched non-DM people. People with diabetes mellitus have a greater probability of dying at similar levels of CAD than people without the disease”.

Hegde SS et al. (2014)[56] “examined the coronary artery and its branches' angiographic extents, types, and numbers of vessels in individuals with acute coronary syndrome (ACS).ACS patients with and without diabetes were compared in the same way. Compared to 8 (16%) of 50 non-diabetic patients, 22 (44%) of 50 diabetic patients in our study had triple or multi-vessel disease. Of the 199 vessels implicated in the 100 ACS patients, 61.3% were diabetics and 38.6% were non-diabetics. Of the 50 diabetic patients, 23 (46%) needed CABG as a therapy outcome. Of the 23 patients who had to have CABG, 19 (73.1%) had HbA1c values >8.5%,

69.2% had triple or multi-vessel disease, and 24% of diabetics were in their third or fourth decade, compared to 10% and 26% of non-diabetics in the same age group. This study shown that, in comparison to nondiabetics with ACS, diabetics had a much higher incidence of triple/multi vessel disease, a significantly higher severity and extent of CAD, and ACS that manifested much earlier in life. High HbA1c diabetics had higher coronary artery involvement, and CABG was the necessary treatment for them".

"The angiographic severity of coronary artery disease in patients with diabetes was compared to that of those without the condition by **Parvin, Tanjima et al. (2015)**[57]. 102 participants with coronary artery disease on coronary angiography were included in this observational study. A total of 78 (76.5%) non-diabetic individuals and 24 (23.5%) diabetic patients made up the two groups of two patients. Ninety-four (92%) of the study participants were male, and their mean age±SD was 52.8±9.5 years. Compared to non-diabetic patients, diabetic patients were older (mean age±SD; 57.6±9.5 versus 51.3±9.9 years; p 004) and had lower rates of smoking (42% versus 67%, p 0.034) and acute coronary syndrome (29% versus 59%; p 0.018), as well as higher rates of hypertension (75% versus 50%, p 0.036) and chronic stable angina (71% versus 41%, p 0.018). Patients with diabetes were more likely to have three-vessel disease (50% versus 31%, p 0.094) and left main stem disease (21% against 5%, p 0.031). Diabetics had a considerably higher prevalence of severe coronary artery stenosis than non-diabetics (Gensini score, 50.9±29.9 versus 32.6±21.9, p 0.001). They came to the conclusion that those with diabetes have a higher risk of developing severe and widespread coronary artery disease. Patients with diabetes are more likely than those without the condition to have left main stem and triple vessel disease".

Rajiv G et al. (2018)[58] found "that both groups had positive angiographic lesions when comparing the average percentage of stenosis and the site and number of vessels involved. There was no discernible difference in the two populations' levels of coronary risk factors. In both groups, there were 131 (65.5%) positive angiographic lesions overall. 61 (81.33%) of the individuals with diabetes mellitus had positive CAG results. Diffuse lesions 6 (9.8%), average vessel stenosis 82.63%, single vessel disease (SVD) 16 (26.24), double vessel disease (DVD) 25 (40.98%), and triple vessel disease (TVD) 20 (32.78%) were the lesions identified. In contrast, the non-diabetic group had 70 (56%) positive angiographic lesions, of which 23 (32.85%) had single vessel disease (SVD), 28 (40%) had double vessel disease (DVD), and 19 (27.15%) had triple vessel disease (TVD). There were no diffuse lesions detected, and the average vessel stenosis was 78.03%. According to the angiographic data, persons with diabetes are more likely than those without the condition to have diffuse lesions, significant stenosis of the coronary arteries, coronary heart disease (CHD), DVD, and TVD. They came to the conclusion that the percentage of severe and unanticipated presentations of CAD in diabetics was significantly higher. It is more challenging to address this increased frequency of complicated lesion shape with definitive interventions such as coronary artery bypass grafting (CABG) and percutaneous transluminal coronary angioplasty (PCI). Diabetics have poorer clinical outcomes and a greater risk factor profile. The likelihood of complications following the development of the disease will be decreased by early identification and suitable care".

The "clinical and angiographic profiles of individuals under 45 years old who presented with Acute Coronary Syndrome (ACS) and Diabetes Mellitus (DM) and those who did not were compared by **Narayanan B L, et al. (2020)**[59]. A total of 80

ACS patients were examined. The average age was 41.2±4.01 years, and the average Glycated Haemoglobin (HbA1c) level was 8.65±3.3%. Younger CAD patients had varied clinical profiles and patterns of coronary artery involvement as determined by coronary angiography; the most common type of ACS was ST-Elevation Myocardial Infarction (STEMI). Echocardiography (ECHO) revealed normal Left Ventricular (LV) function, but Single Vessel Disease (SVD) was the most frequent angiographic finding. Diabetics with greater HbA1c were more likely to have multiple artery disease and atypical chest discomfort (p=0.001). They came to the conclusion that ACS patients who are younger and do not have diabetes have a lower disease burden than those who have diabetes or are older. This discovery may aid in disease prognosis."

Sareddy P. and associates (2021)[60] **"**In this case control research, 142 individuals with angiographically confirmed CAD participated. The cases and remaining controls consisted of 71 patients who had diabetes or had recently been diagnosed with the disease. The mean age at which CAD occurred was 52.15+6.81, and there was no discernible variation in mean age across the groups. Female diabetics had a higher prevalence of CAD. Obesity, smoking, dyslipidemia, and hypertension were identified as the main risk factors for CAD. The most prevalent conditions among diabetic CAD patients were silent ischemia and atypical chest discomfort. Diabetics are more likely to have multiple vascular involvement (47.9% vs. 18.3%, p <0.01). Long-term diabetes and inadequate glycemic control were linked to more severe and widespread CAD (p<0.05) and the result of CABG treatment (p<0.01). They came to the conclusion that severe, widespread coronary artery involvement was more common in diabetic CAD patients. The necessity of a thorough cardiac examination at an early stage is shown by the significant percentage

of diabetics who had asymptomatic ischemia with normal ECG and 2D echo. Uncontrolled and extended diabetes, female gender, hypertension, and dyslipidemia all contributed to more severe forms of CAD and poor CABG treatment outcomes".

Nambirajan et al. (2022)[61] "compared the angiographic profiles of patients in a South Indian community who had diabetes and those who did not. In our study, female patients with diabetes had a higher probability of having ACS than those without the condition; 26 (51%) out of 51 diabetic patients had multi-vessel disease, compared to 12 (23%) out of 51 non-diabetics. Compared to 47.05% of non-diabetics, 72.55% of diabetics had stenosis severity ranging from grade 4-5. Diabetics have significantly higher levels of total occlusion or grade 5 stenosis than non-diabetics. This study shown that, in comparison to non-diabetics with ACS, diabetics had a much higher incidence of triple/multi vascular disease and a significantly higher severity and extent of CAD".

When examining the place and number of vessels involved, as well as the average percentage of stenosis, "**Al Baker, S. M. E. et al. (2023)**[62] found angiographic lesions in both groups. There was no discernible difference in the two populations' levels of coronary risk factors. In both groups, the overall percentage of positive angiographic lesions was 61.5%. 69.3% of patients with diabetes mellitus had positive CAG results. As a percentage of vascular stenosis, the lesions that were identified were triple vessel disease (TVD) 24 (27.3%), double vessel disease (DVD) 14 (15.9%), and single vessel disease (SVD) 23 (26.1%). In contrast, the non-diabetic group had a total of 70 (53.4%) positive angiographic lesions, of which 30 (24.9%) were single vessel disease (SVD), 15 (12.9%) were double vessel disease (DVD), and 17 (14.7%) were triple vessel disease (TVD). The average vessel stenosis was 78.03%, and no widespread lesions were discovered. According to the angiographic data,

persons with diabetes are more likely than those without the condition to have diffuse lesions, significant stenosis of the coronary arteries, coronary heart disease (CHD), DVD, and TVD. Conclusion: The percentage of severe and unpredictable presentations of CAD was significantly greater in diabetics. It is more challenging to address this increased frequency of complicated lesion shape with definitive interventions such as coronary artery bypass grafting (CABG) and percutaneous transluminal coronary angioplasty (PCI). Diabetics have poorer clinical outcomes and a larger profile of risk factors. The likelihood of complications following the development of the disease will be decreased by early identification and suitable care."

MATERIAL AND METHODS

- **Study design:** An Observational Case Control study
- **Study area: "**Department of General Medicine, B.L.D.E. (D.U.) Shri B.M.Patil Medical College hospital and research center, Vijayapura, Karnataka, India.
- **Study period:** Research study was conducted from May 2023 to December 2024. Below is the work plan.

Table 2: Work plan of the study with percentage of allocation of study time and duration in months"

Work plan	% of allocation of study time	Duration in months
Understanding the problem, preparation of questionnaire.	5-10%	May 2023 to July 2023
Pilot study, Validation of questionnaire, data collection and manipulation	Upto 80%	August 2023 to June 2024
Analysis and interpretation	5-10%	July 2024 to September 2024
Dissertation write-up and submission	5-10%	October 2024 to December 2024

- **Sample size**: This study requires a total sample size of 126
  - ✓ 63CADpatients who are diabetic will be taken as cases and
  - ✓ 63CADpatients who are non-diabetic were taken with age and sex matched controls.
- **Sampling method**: Convenient sampling method\

- **Inclusion criteria:**

1. Patients previously known diabetic or first time detected diabetic by World health organization (WHO) criteria and having coronary artery disease who underwent coronary angiography are included as cases.
2. Patients diagnosed as having coronary artery disease who underwent coronary angiography and are non-diabetic are included as controls.

- **Exclusion criteria:**

1. Patients with Valvular heart disease
2. Patients with congenital heart disease
3. Patients with Type 1 Diabetes Mellitus

METHODOLOGY:

The study was conducted at B.L.D.E. (D.U.) Shri B.M. Patil Medical College Hospital and Research Center in Vijayapura. Patients undergoing coronary angiography and diagnosed with coronary artery disease (CAD) were enrolled after obtaining written informed consent.

The research adopted a comparative study design with two primary groups:

- Cases: CAD patients with diabetes
- Controls: CAD patients without diabetes

Patient Recruitment and Consent

A comprehensive explanation of the study's nature and purpose was provided to potential participants. A structured format was used to record personal details for each subject. Only patients willing to participate were included in the study.

Examination and Data Collection

A detailed methodology was implemented for comprehensive patient assessment:

1. **“Patient Evaluation**

- Detailed medical history collection
- Comprehensive general physical examination
- Systematic clinical examination

2. **Diagnostic Investigations Laboratory Investigations:**

- Complete blood count
- Fasting blood glucose
- Postprandial blood sugar
- HbA1c (glycosylated hemoglobin)
- Lipid profile
- Renal function tests”

Cardiovascular Assessments:

- Electrocardiogram (ECG)
- 2D Echocardiography (2DECHO)
- Coronary Angiography

Ethical Considerations

The study was conducted after obtaining appropriate institutional ethical committee approval, ensuring patient confidentiality and informed consent.

STATISTICAL ANALYSIS

"Data was entered in excel sheet and analyzed using SPSS version 21. Results were presented in tabular and graphical forms Mean, median, standard deviation and ranges were calculated for quantitative data. Qualitative data were expressed in terms of frequency and percentages. Student t test (Two Tailed) was used to test the significance of mean and P value <0.05 was considered significant".

RESULTS

The “present observational case-control study was conducted among 126 diabetic and non-diabetic patients of B.L.D.E. (D.U.) Shri B.M.Patil Medical College hospital and research center, Vijayapura. from May 2023 to December 2024 to study clinical presentation and angiographic characteristics of coronary artery disease in diabetic patients compared to that of non-diabetics”.

Following are the results of the study.

Table 3: Comparison of age among groups

Age (in years)	Diabetics	Non-diabetics	p-value
30-40	1 (1.6%)	6 (9.5%)	0.09
41-50	11 (17.5%)	4 (6.3%)	
51-60	29 (46%)	26 (41.3%)	
61-70	13 (20.6%)	19 (30.2%)	
71-80	9 (14.3%)	8 (12.7%)	
Total	**63 (100%)**	**63 (100%)**	

This table shows the age distribution of patients in both groups. In the diabetic group, most patients (46%) were between 51-60 years, followed by 61-70 years (20.6%). In the non-diabetic group, the highest percentage was also in the 51-60 age range (41.3%), followed by 61-70 years (30.2%). While non-diabetics had more patients in the younger 30-40 age group (9.5% vs 1.6%), diabetics had more in the 41-50 range (17.5% vs 6.3%). The p-value of 0.09 indicates that these differences in age distribution between the groups were not statistically significant.

Figure 4: Comparison of age among groups

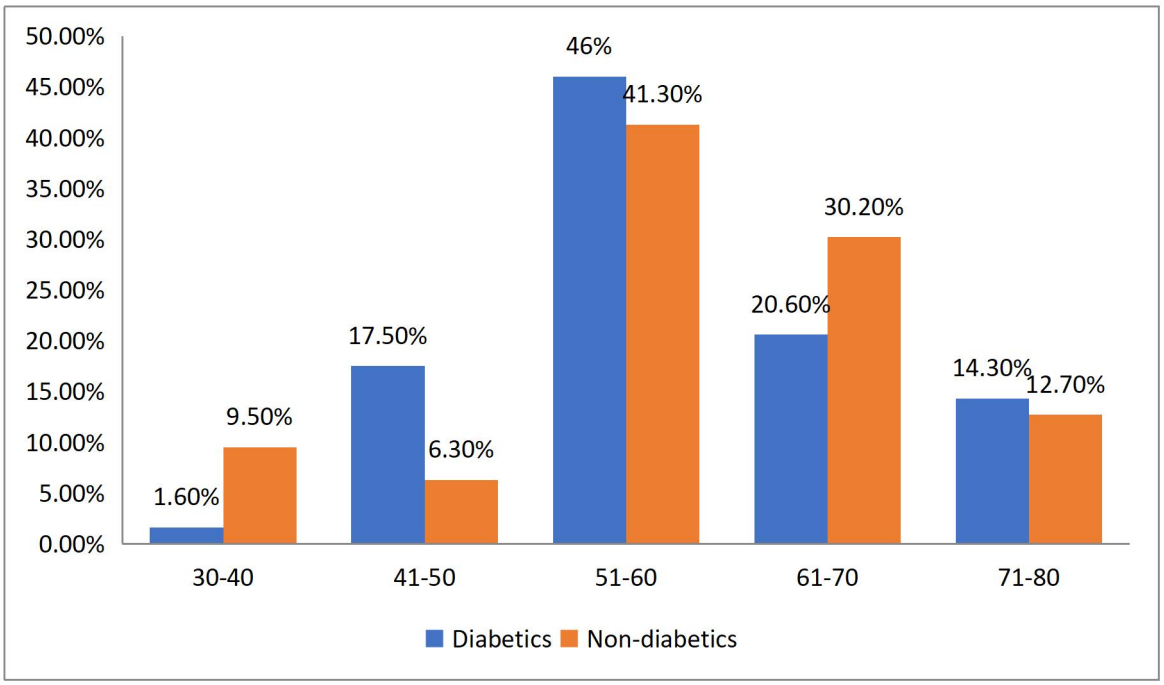


Table 4: Comparison of gender among groups

Gender	Diabetics	Non-diabetics	p-value
Female	27 (42.9%)	23 (36.5%)	0.46
Male	36 (57.1%)	40 (63.5%)	
Total	**63 (100%)**	**63 (100%)**	

This table shows the gender distribution in both groups. The diabetic group had 57.1% males and 42.9% females, while the non-diabetic group had 63.5% males and 36.5% females. With a p-value of 0.46, this difference in gender distribution was not statistically significant.

Figure 5: Comparison of gender among groups

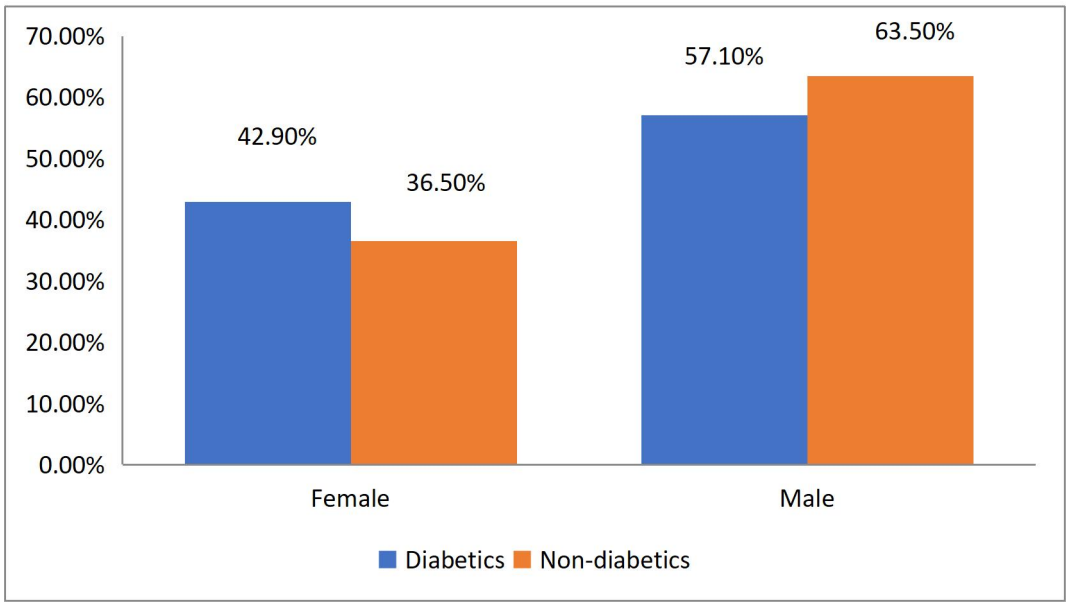


Table 5: Comparison of symptoms among groups

Symptoms	Diabetics	Non-diabetics	p-value
Chest pain	55 (87.3%)	52 (82.6%)	0.41
Breathlessness	28 (44.4%)	29 (46%)	0.1
Giddiness	3 (4.8%)	3 (4.8%)	**0.02**
Palpitation	15 (23.8%)	11 (14.3%)	0.37
Sweating	28 (44.4%)	17 (26.9%	0.509
Others	29 (46.03%)	22 (34.9%)	**0.04**

This table "compares the symptoms presented by diabetic and non-diabetic patients. Chest pain was the most common symptom in both groups (87.3% in diabetics vs 82.6% in non-diabetics)". Breathlessness was similar in both groups (44.4% vs 46%). Interestingly, giddiness showed statistical significance (p=0.02) though the percentages appear identical (4.8% in both groups). Sweating was more common in diabetics (44.4% vs 26.9%), and "Others" symptoms also showed a significant difference (p=0.04) with 46.03% in diabetics compared to 34.9% in non-diabetics.

Figure 6: Comparison of symptoms among groups

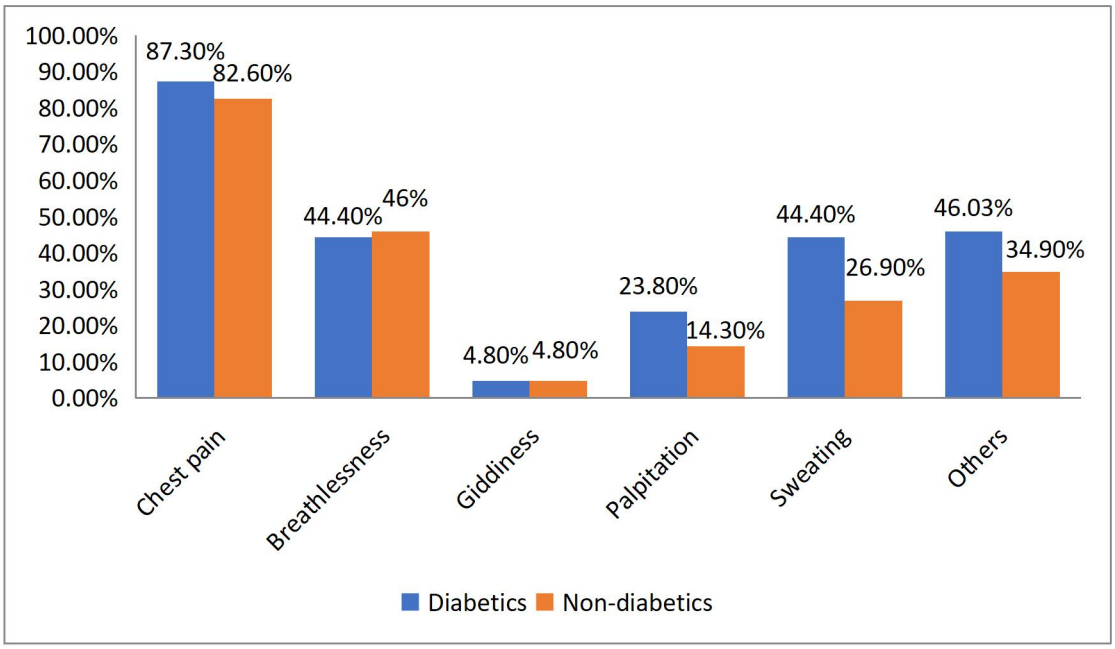


Table 6: Duration of diabetes among diabetics

Duration of diabetes (years)	
Mean	3.42
SD	5.1

This table shows that in the diabetic group, the mean duration of diabetes was 3.42 years with a standard deviation of 5.1 years, indicating considerable variation in how long patients had been diagnosed with diabetes.

Figure 7: Duration of diabetes among diabetics

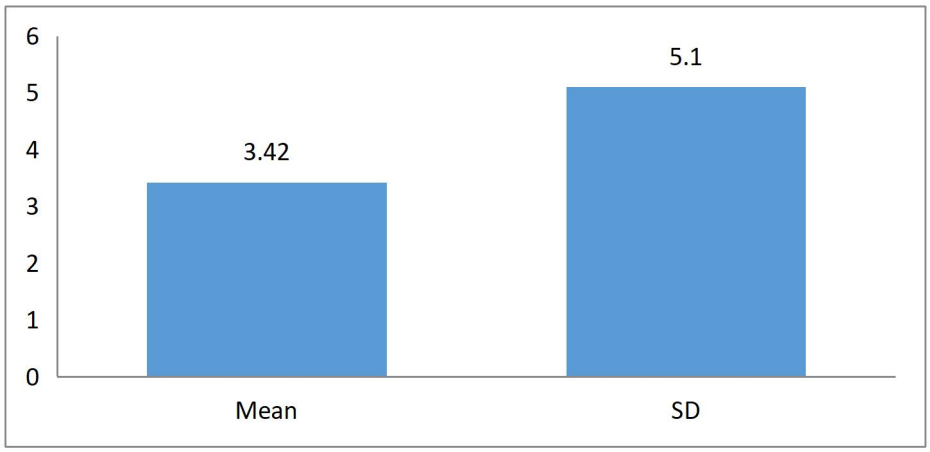


Table 7: Comparison of HbA1c among groups

HbA1c	Diabetics	Non-diabetics	p-value
<5.7	0	63 (100%)	**<0.001**
5.7-6.5	2 (3.2%)	0	
6.6-8.5%	20 (31.7%)	0	
8.6-11%	31 (49.2%)	0	
>11	10 (15.8%)	0	
Total	**63 (100%)**	**63 (100%)**	

This table shows the glycemic control in both groups. All non-diabetic patients (100%) had HbA1c levels <5.7%, which is expected. In the diabetic group, almost half (49.2%) had poor control with HbA1c between 8.6-11%, 31.7% had moderate control (6.6-8.5%), and 15.8% had very poor control (>11%). Only 3.2% had borderline values (5.7-6.5%). The p-value <0.001 indicates this distribution is highly statistically significant.

Figure 8: Comparison of HbA1c among groups

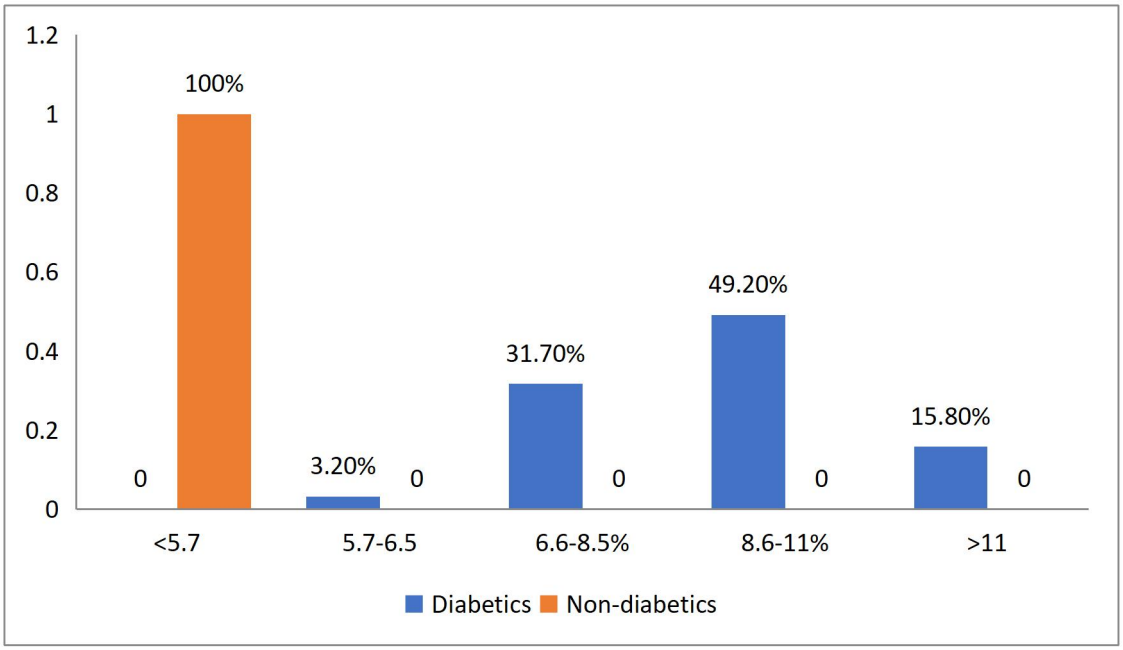


Table 8: Comparison of ECG findings among groups

ECG findings	Diabetics	Non-diabetics	p-value
ALWMI	1 (1.6%)	1 (1.6%)	0.71
ASWMI	4 (6.3%)	4 (6.3%)	
AWMI	19 (30.2%)	17 (27%)	
ILWMI	2 (3.2%)	1 (1.6%)	
IPWMI	2 (3.2%)	3 (4.8%)	
IWMI	12 (19%)	8 (12.7%)	
LWMI	2 (3.2%)	5 (7.9%)	
NSTEMI	15 (23.8%)	11 (17.5%)	
Sinus tachycardia	0	1 (1.6%)	
Unstable angina	6 (9.5%)	9 (14.3%)	
Normal	0	3 (4.8%)	
Total	**63 (100%)**	**63 (100%)**	

This table details ECG findings in both groups. Anterior Wall Myocardial Infarction (AWMI) was the most common finding in both groups (30.2% in diabetics vs 27% in non-diabetics), followed by Non-ST Elevation Myocardial Infarction (NSTEMI) (23.8% vs 17.5%). Inferior Wall Myocardial Infarction (IWMI) was more common in diabetics (19% vs 12.7%). Notably, 4.8% of non-diabetics had normal ECGs, while no diabetics did. The p-value of 0.71 suggests these differences were not statistically significant.

Figure 9: Comparison of ECG findings among groups

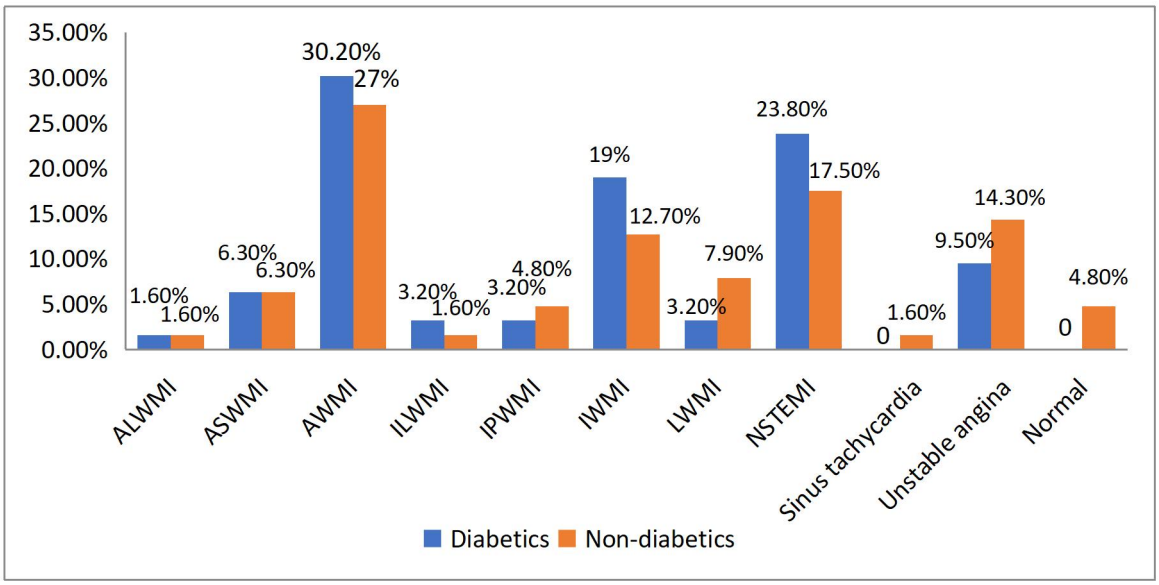


Table 9: Comparison of RWMA among groups

RWMA	Diabetics	Non-diabetics	p-value
Present	59 (93.7%)	49 (77.8%)	**0.05**
Absent	4 (6.3%)	14 (22.2%)	
Total	**63 (100%)**	**63 (100%)**	

This table examines Regional Wall Motion Abnormality (RWMA) on echo-cardiography.RWMA was significantly more common in diabetics (93.7%) compared to non-diabetics (77.8%), with a p-value of 0.05, indicating statistical significance.

Figure 10: Comparison of RWMA among groups

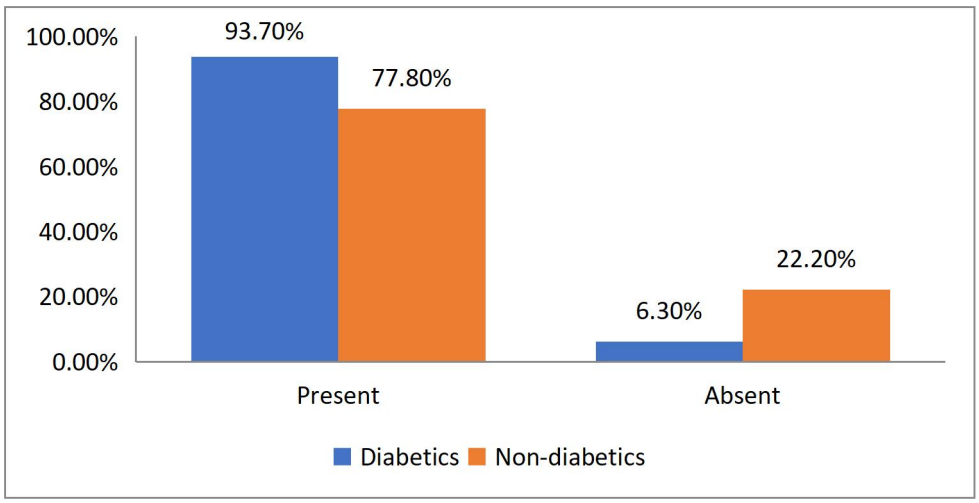


Table 10: Comparison of ejection fraction among groups

Ejection fraction	Diabetics	Non-diabetics	p-value
<30%	7 (11.7%)	6 (9.5%)	**0.03**
30-39%	13 (20.6%)	3 (4.8%)	
40-49%	23 (36.5%)	22 (34.9%)	
>50%	20 (31.7%)	32 (50.8%)	
Total	**63 (100%)**	**63 (100%)**	

This table compares left ventricular ejection fraction between groups. More diabetics had severely reduced (<30%) and moderately reduced (30-39%) ejection fractions (11.7% and 20.6% respectively) compared to non-diabetics (9.5% and 4.8%). Correspondingly, more non-diabetics (50.8%) had preserved ejection fraction (>50%) compared to diabetics (31.7%). With a p-value of 0.03, these differences were statistically significant.

Figure 11: Comparison of ejection fraction among groups

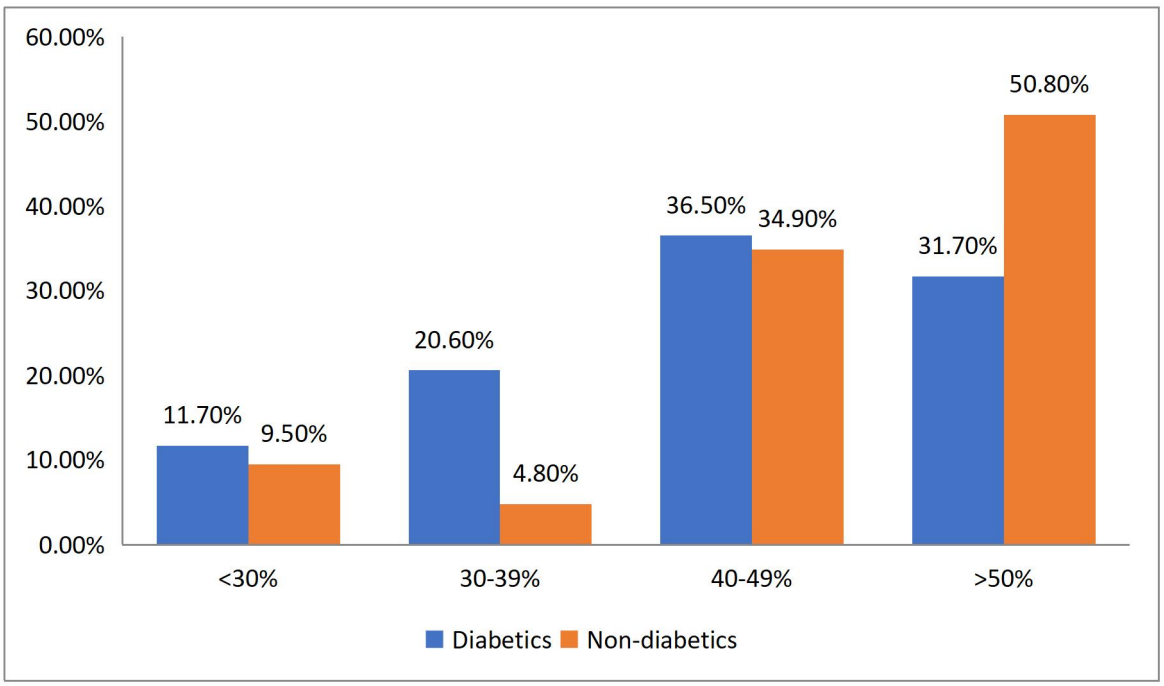


Table 11: Comparison of CAG among groups

CAG findings	Diabetics	Non-diabetics	p-value
Triple vessel disease	27 (42.9%)	15 (23.8%)	
Double vessel disease	17 (27%)	10 (15.9%)	
Single vessel disease	15 (23.8%)	22 (34.9%)	**<0.001**
Minor CAD	4 (6.3%)	12 (19.04%)	
Normal	0	4 (6.3%)	
Total	**63 (100%)**	**63 (100%)**	

This table shows coronary angiography findings. Triple vessel disease was significantly more common in diabetics (42.9% vs 23.8%), while single vessel disease was more common in non-diabetics (34.9% vs 23.8%). Non-diabetics also had more minor coronary artery disease (19.04% vs 6.3%) and normal coronaries (6.3% vs 0%). The p-value <0.001 indicates these differences were highly statistically significant.

Figure 12: Comparison of CAG among groups

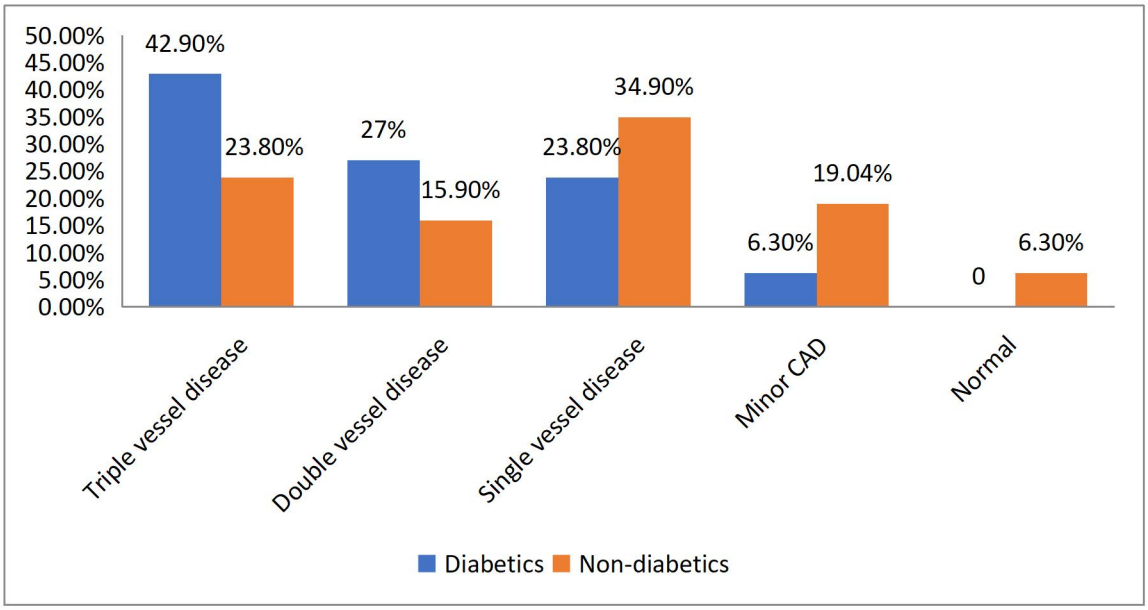


Table 12: Comparison of vessel involved among groups

Vessel involved	Diabetics	Non-diabetics	p-value
LAD	57 (90.5%)	52 (82.5%)	0.19
LCX	40 (63.5%)	22 (34.9%)	**0.001**
RCA	35 (55.6%)	23 (36.5%)	**0.03**

This table details which specific coronary vessels were involved. Left Anterior Descending (LAD) artery involvement was common in both groups (90.5% in diabetics vs 82.5% in non-diabetics) without significant difference (p=0.19). However, Left Circumflex (LCX) and Right Coronary Artery (RCA) involvement showed significant differences (p=0.001 and p=0.03), with diabetics having significantly higher involvement of both vessels (63.5% vs 34.9% for LCX and 55.6% vs 36.5% for RCA).

Figure 13: Comparison of vessel involved among groups

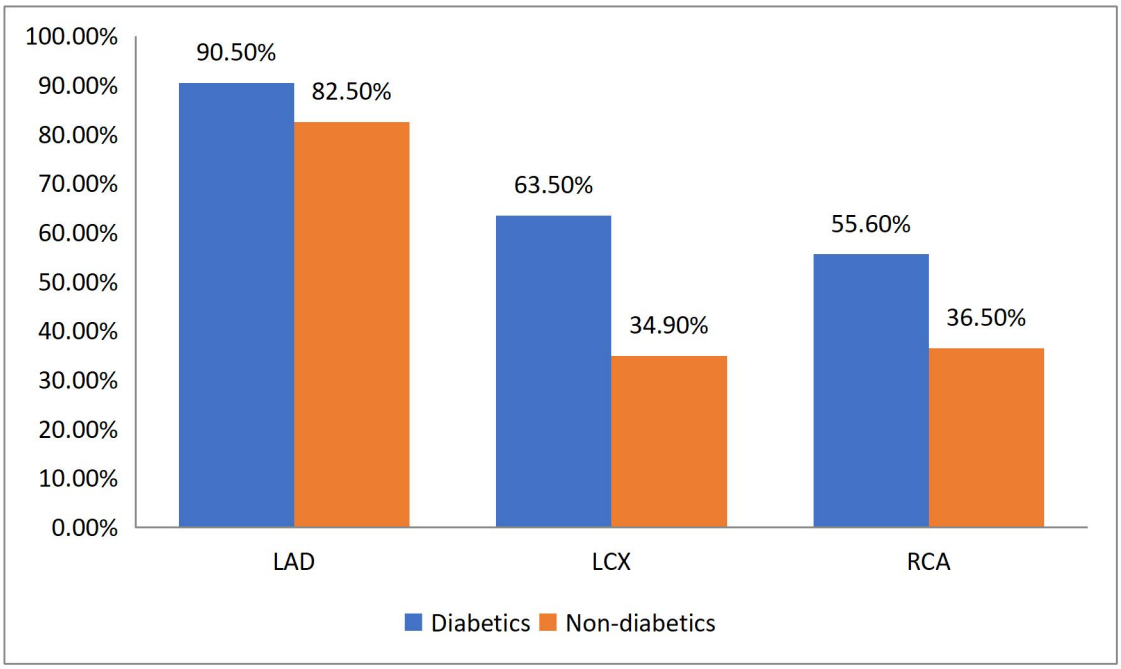


Table 13: Association of CAG findings with duration of diabetes

duration of diabetes (years)	CAG findings					p-value
	Single vessel	Double vessel	Triple vessel	Minor CAD	Normal	
0-5	30 (81.1%)	18 (66.7%)	28 (66.7%)	15 (93.8%)	4 (100%)	0.09
5-10	5 (13.5%)	7 (25.9%)	6 (14.3%)	1 (6.3%)	0	
>10	2 (5.4%)	2 (7.4%)	8 (19%)	0	0	
Total	**37 (100%)**	**27 (100%)**	**42 (100%)**	**16 (100%)**	**4 (100%)**	

This table examines the relationship between diabetes duration and extent of coronary disease. While there appears to be a trend toward more triple vessel disease with longer diabetes duration (19% of patients with >10 years of diabetes had triple vessel disease), the p-value of 0.09 suggests this association did not reach statistical significance.

Figure 14: Association of CAG findings with duration of diabetes

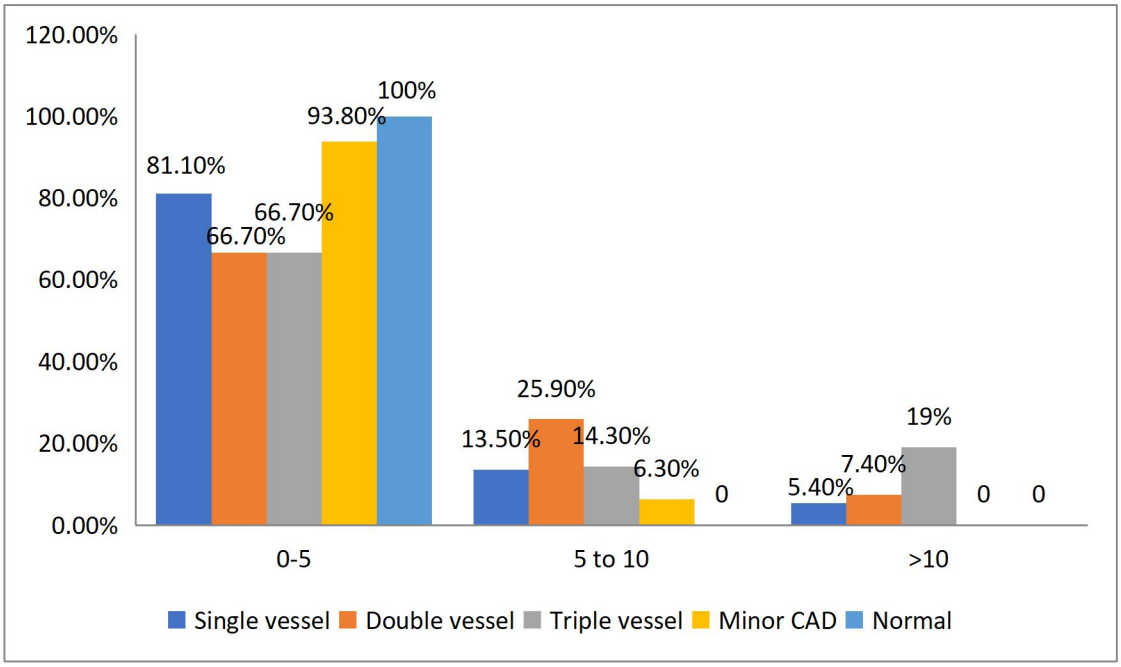
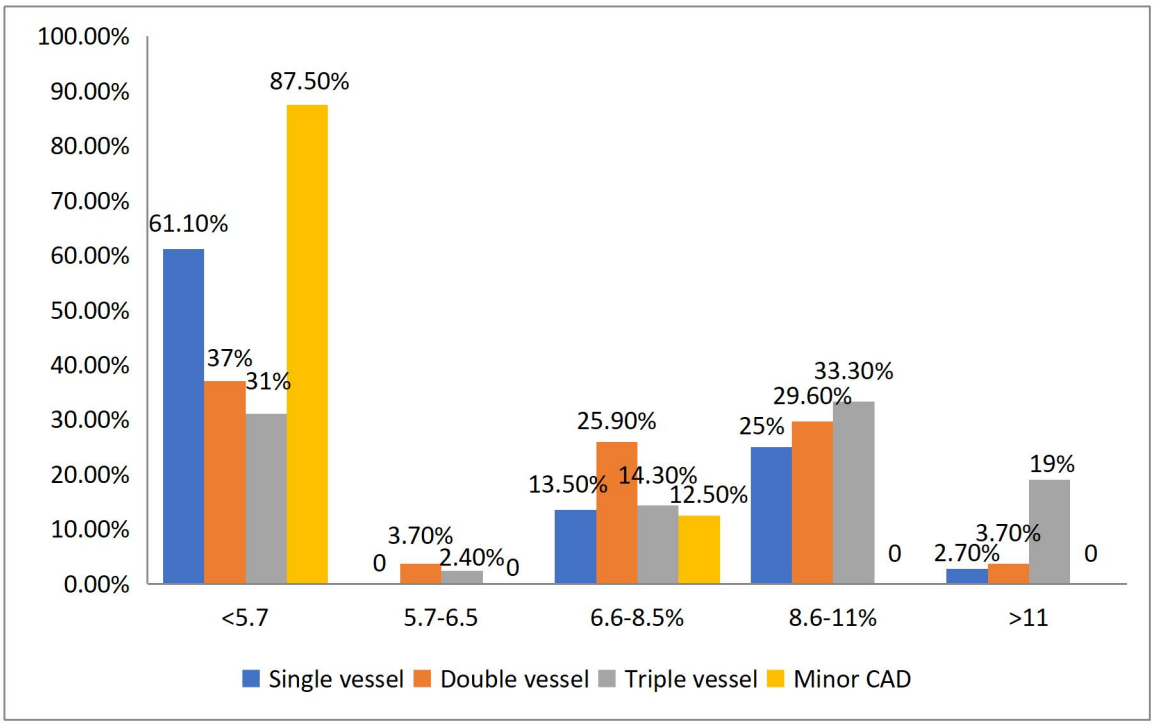


Table 14: Association of CAG findings with HbA1c

HbA1c (%)	CAG findings					p-value
	Single vessel	Double vessel	Triple vessel	Minor CAD	Normal	
<5.7	22 (61.1%)	10 (37%)	13 (31%)	14 (87.5%)	4 (100%)	**0.009**
5.7-6.5	0	1 (3.7%)	1 (2.4%)	0	0	
6.6-8.5%	5 (13.5%)	7 (25.9%)	6 (14.3%)	2 (12.5%)	0	
8.6-11%	9 (25%)	8 (29.6%)	14 (33.3%)	0	0	
>11	1 (2.7%)	1 (3.7%)	8 (19%)	0	0	
Total	**37 (100%)**	**27 (100%)**	**42 (100%)**	**16 (100%)**	**4 (100%)**	

This table shows the relationship between glycemic control and coronary disease severity. Patients with higher HbA1c values (particularly >8.6%) were more likely to have multi-vessel disease. Notably, 19% of patients with HbA1c >11% had triple vessel disease. The p-value of 0.009 indicates this association was statistically significant, suggesting poorer glycemic control correlates with more extensive coronary disease.

Figure 15: Association of CAG findings with HbA1c



DISCUSSION

Coronary artery disease (CAD) remains a major cause of morbidity and mortality worldwide, with diabetes mellitus significantly contributing to its pathogenesis and progression. Diabetes accelerates the atherosclerotic process, leading to more extensive, diffuse, and complex coronary lesions. The present study was designed to compare the clinical presentation and angiographic profile of CAD in diabetic and non-diabetic patients. Our findings provide valuable insights into the differences in disease patterns, which could help in tailoring management strategies for these two distinct populations.

Demographic Characteristics

In our study, we analyzed 126 patients who underwent coronary angiography, with equal distribution of diabetic (n=63) and non-diabetic (n=63) subjects. The age distribution showed that the majority of patients in both groups fell within the 51-60 years age bracket (46% of diabetics and 41.3% of non-diabetics), followed by the 61-70 years age group. This age distribution is consistent with the findings reported by "Hegde SS et al., who observed that among diabetics 24% of the cases were in their third decade and 40% of the cases were in the fourth decade as compared to 10% and 26% of non-diabetics of similar age group".[56]

Our data revealed a male preponderance in both groups (57.1% in diabetics and 63.5% in non-diabetics), although this gender difference was not statistically significant (p=0.46). This male predominance aligns with the observations by Panduranga et al., who reported that 79% of their CAD patients with diabetes were males.[63] "The higher prevalence of CAD in males may be attributed to the protective effect of estrogen in premenopausal women, which delays the onset of atherosclerotic changes".

Clinical Presentation

The clinical presentation of CAD demonstrated some similarities and differences between the two groups. Chest pain was the most common presenting symptom in both diabetics (87.3%) and non-diabetics (82.6%), with no significant difference between the groups (p=0.41). This finding is consistent with that of Hasin et al., who reported chest pain as the predominant symptom in both diabetic and non-diabetic CAD patients.[64]

Interestingly, our study found a significantly higher prevalence of giddiness as a presenting symptom in diabetic patients compared to non-diabetics (p=0.02). This could be attributed to autonomic neuropathy in diabetics, which may alter the perception of anginal symptoms and lead to atypical presentations. The category of 'other symptoms' was significantly more common in diabetics (46.03% vs 34.9%, p=0.04). This is in line with the study by Juneja et al., who reported that diabetic patients often present with atypical symptoms or silent ischemia due to cardiac autonomic neuropathy.[65]

The mean duration of diabetes in our study population was 3.42 ± 5.1 years. This relatively short duration suggests that coronary atherosclerosis may begin early in the course of diabetes, or even during the prediabetic state. This finding underscores the importance of early screening and aggressive risk factor modification in patients with newly diagnosed diabetes or impaired glucose tolerance.

Glycemic Control and CAD

Our study demonstrated a significant difference in glycemic control between the diabetic and non-diabetic groups, as evidenced by HbA1c levels. All non-diabetic patients had HbA1c levels <5.7%, whereas the majority of diabetic patients had HbA1c levels between 8.6-11% (49.2%), followed by 6.6-8.5% (31.7%) and >11% (15.8%). This suggests that most of our diabetic patients had suboptimal glycemic control, which might have contributed to the development and severity of their CAD.

The association between HbA1c levels and the severity of CAD was statistically significant (p=0.009). We observed that patients with higher HbA1c levels (>8.6%) had a greater likelihood of having triple-vessel disease (TVD) or double-vessel disease (DVD). These findings are consistent with those reported by Ravipati et al., who demonstrated a direct correlation between HbA1c levels and the severity of CAD.[66] Similarly, a study by Saleem et al. found that patients with poor glycemic control (HbA1c ≥7%) had significantly higher incidence of multi-vessel disease compared to those with good glycemic control (HbA1c <7%).[67]

The UK Prospective Diabetes Study (UKPDS) established that intensive blood glucose control significantly reduces the risk of microvascular complications but has a less pronounced effect on macrovascular outcomes.[68] However, long-term follow-up data from the same study suggested that early and sustained glycemic control may translate into reduced cardiovascular events over time, a phenomenon termed as "metabolic memory" or "legacy effect".[69] Our findings reinforce the importance of achieving optimal glycemic control in diabetic patients to potentially mitigate the severity of CAD.

ECG Findings

The electrocardiographic patterns observed in our study revealed a diverse spectrum of findings across both groups. Anterior wall myocardial infarction (AWMI) was the most common presentation in both diabetics (30.2%) and non-diabetics (27%), followed by non-ST-elevation myocardial infarction (NSTEMI) (23.8% in diabetics vs 17.5% in non-diabetics) and inferior wall myocardial infarction (IWMI) (19% in diabetics vs 12.7% in non-diabetics). Although the distribution of ECG findings did not differ significantly between the two groups (p=0.71), it is noteworthy that normal ECG findings were observed in 4.8% of non-diabetics but were absent in the diabetic group.

The presence of normal ECG findings in some non-diabetic patients despite angiographically proven CAD highlights the limitations of ECG as a screening tool for CAD. This is particularly relevant in the context of minor CAD, where the ischemic burden may not be sufficient to cause ECG changes. Conversely, the absence of normal ECG findings in the diabetic group may reflect the more extensive and severe nature of CAD in these patients, as corroborated by our angiographic findings.

Similar "patterns were reported by Uddin et al., who found a higher prevalence of acute coronary syndromes in diabetic patients compared to non-diabetics".[70] They attributed this to the pro-inflammatory and pro-thrombotic state associated with diabetes, which predisposes to plaque rupture and thrombosis.

Left Ventricular Function

Assessment of left ventricular (LV) function is crucial in patients with CAD, as it provides prognostic information and guides therapeutic decisions. Our study found a statistically significant difference in the distribution of ejection fraction (EF) between diabetic and non-diabetic patients (p=0.03). Notably, a higher proportion of diabetic patients had moderate to severe LV dysfunction (EF <40%) compared to non-diabetics (32.3% vs 14.3%). Conversely, a significantly higher percentage of non-diabetic patients had preserved LV function (EF >50%) compared to diabetics (50.8% vs 31.7%).

The more pronounced LV dysfunction in diabetic patients can be attributed to several factors. Firstly, the more extensive & severe CAD in diabetics, as evidenced by our angiographic findings, leads to a greater area of myocardium at risk. Secondly, diabetic cardiomyopathy, characterized by myocardial fibrosis, hypertrophy, and impaired contractility, can contribute to LV dysfunction independent of epicardial coronary artery stenosis.Thirdly,the synergistic effect of hypertension, which is associated with diabetes,can increase afterload and consequent LV remodeling.

These findings are in agreement with those of Bax et al., who demonstrated that diabetic patients with CAD had significantly lower EF compared to their non-diabetic counterparts.[71] Similarly, Kamalesh et al. found that diabetic patients had a higher prevalence of heart failure with reduced ejection fraction (HFrEF) despite similar infarct sizes, suggesting that factors beyond ischemia contribute to LV dysfunction in diabetes.[72]

Regional wall motion abnormalities (RWMA) were significantly more prevalent in diabetic patients compared to non-diabetics (93.7% vs 77.8%, p=0.05). This higher prevalence of RWMA in diabetics may reflect a greater area of infarcted or ischemic myocardium due to more extensive CAD. Additionally, diabetic patients may have impaired coronary microcirculation, which can lead to myocardial perfusion defects and consequent RWMA despite patent epicardial coronary arteries.

Angiographic Profile

The angiographic findings in our study revealed striking differences between diabetic and non-diabetic patients. The prevalence of multi-vessel disease was significantly higher in diabetics compared to non-diabetics ($p<0.001$). Triple-vessel disease was observed in 42.9% of diabetic patients compared to 23.8% of non-diabetics, while double-vessel disease was present in 27% of diabetics versus 15.9% of non-diabetics. Conversely, single-vessel disease was more common in non-diabetics than in diabetics (34.9% vs 23.8%). Moreover, minor CAD and normal coronary arteries were significantly more prevalent in non-diabetics (19.04% and 6.3%, respectively) compared to diabetics (6.3% and 0%, respectively).

These findings are consistent with those reported by Natali et al., who found a higher prevalence of multi-vessel disease in diabetic patients (63% vs 50%) in their large multi-center study.[73] Similarly, Shah T et al.[74] reported that compared to “non-

diabetics,diabetics had higher syntax score, 23-32 30(30%) were diabetics, while 13(13%)were nondiabetics, and score>33 9, (9%) diabetics as compared to nondiabetic has 1%,p value<0.001)".

The more extensive and "diffuse nature of CAD in diabetic patients can be attributed to the pro-atherogenic milieu associated with diabetes. Hyperglycemia induces endothelial dysfunction, increases oxidative stress, promotes inflammation, and enhances platelet aggregation, all of which contribute to accelerated atherosclerosis. Additionally, insulin resistance and the associated dyslipidemia (characterized by high triglycerides, low HDL-cholesterol, and small, dense LDL particles) further exacerbate the atherosclerotic process".

Our analysis of the "vessels involved revealed significant differences in the distribution pattern between the two groups. While the left anterior descending artery (LAD) was the most commonly involved vessel in both groups (90.5% in diabetics vs 82.5% in non-diabetics, p=0.19), the involvement of the left circumflex artery (LCX) and right coronary artery (RCA) was significantly higher in diabetics compared to non-diabetics (LCX: 63.5% vs 34.9%, p=0.001; RCA: 55.6% vs 36.5%, p=0.03)".

This pattern of multi-vessel involvement in diabetics has been consistently reported in the literature. Varghese K et al. found that diabetic patients had a higher prevalence of LCX and RCA involvement compared to non-diabetics, although the difference did not reach statistical significance in their study.[75] Similarly, Srinivasan et al. reported that diabetic patients with acute coronary syndrome had a higher prevalence of multi-vessel disease and diffuse lesions compared to non-diabetics.[77]

The predilection for multi-vessel involvement in diabetics may be related to the systemic nature of the disease, which affects all vascular beds. Additionally, the increased prevalence of diffuse atherosclerosis in diabetics, as opposed to focal lesions

in non-diabetics, may explain the higher involvement of multiple vessels. This pattern has important implications for revascularization strategies, as diabetic patients may require more extensive revascularization or coronary artery bypass grafting (CABG) rather than percutaneous coronary intervention (PCI).

Duration of Diabetes and CAD Severity

"We analyzed the association between the duration of diabetes and the severity of CAD, although this relationship did not reach statistical significance in our study ($p=0.09$). However, there was a trend towards a higher prevalence of triple-vessel disease in patients with a longer duration of diabetes (>10 years) compared to those with a shorter duration. This observation aligns with the pathophysiological understanding that prolonged exposure to hyperglycemia and other metabolic derangements associated with diabetes can lead to progressive vascular damage and atherosclerosis.

Fox et al. reported a significant association between the duration of diabetes and the severity of CAD in their study, with each 10-year increase in diabetes duration corresponding to a 25% increase in the risk of multi-vessel disease.[77] Similarly, Serruys PW et al. found that patients with a diabetes duration >10 years had a significantly higher SYNTAX score compared to those with a shorter duration.[78]

The absence of a statistically significant association in our study may be attributed to the relatively short mean duration of diabetes (3.42 years) in our cohort, which might not have been sufficient to demonstrate a clear dose-response relationship. Additionally, other factors such as glycemic control, lipid profile, blood pressure, and genetic predisposition may have confounded this relationship".

Clinical Implications

"The findings of our study have several important clinical implications. Firstly, the more extensive and severe nature of CAD in diabetic patients underscores the importance of aggressive risk factor modification, including optimal glycemic control, lipid management, blood pressure control, and lifestyle modifications. Current guidelines recommend a target HbA1c of <7% for most diabetic patients, with more stringent targets (<6.5%) for selected individuals who can achieve it without significant hypoglycemia.[79] However, the benefits of intensive glycemic control for macrovascular outcomes may take several years to manifest, highlighting the importance of early intervention.

Secondly, the higher prevalence of multi-vessel disease in diabetics has implications for revascularization strategies. The FREEDOM trial demonstrated a significant reduction in the composite endpoint of death, myocardial infarction, and stroke with CABG compared to PCI in diabetic patients with multi-vessel disease. [80]Therefore, CABG may be the preferred revascularization strategy for diabetic patients with multi-vessel CAD, especially those with complex lesions or those who are suitable candidates for surgery.

Thirdly, the higher prevalence of LV dysfunction in diabetic patients emphasizes the importance of routine assessment of LV function in these individuals. Early identification of LV dysfunction can guide the initiation of therapies that have been shown to improve prognosis, such as angiotensin-converting enzyme inhibitors (ACEIs), angiotensin receptor blockers (ARBs), beta-blockers, and mineralocorticoid receptor antagonists (MRAs).

Lastly, the significantly higher prevalence of triple-vessel disease in diabetic patients, even with a relatively short duration of diabetes, suggests that coronary atherosclerosis may begin early in the course of diabetes or even during the prediabetic state. This highlights the importance of early screening for CAD in patients with newly diagnosed diabetes or impaired glucose tolerance, especially those with other cardiovascular risk factors".

Limitations and Future Directions

"Our study has several limitations that warrant consideration. Firstly, the cross-sectional design precludes the establishment of causal relationships. Longitudinal studies would be better suited to evaluate the temporal relationship between diabetes and the development and progression of CAD. Secondly, we did not account for other cardiovascular risk factors such as hypertension, dyslipidemia, smoking, and obesity, which may have confounded the observed relationships. Thirdly, we did not assess the impact of different antidiabetic medications on CAD patterns, which could have provided insights into the potential cardioprotective effects of certain drugs, such as GLP-1 receptor agonists and SGLT2 inhibitors.

Future research should focus on elucidating the mechanisms underlying the accelerated atherosclerosis in diabetes, particularly the role of inflammation, oxidative stress, endothelial dysfunction, and epigenetic modifications. Additionally, studies evaluating the impact of different glycemic targets and antidiabetic medications on CAD outcomes in diabetic patients would provide valuable information for clinical decision-making. Furthermore, the development of personalized risk assessment tools that incorporate clinical, biochemical, and genetic factors could help in the early identification of high-risk individuals who may benefit from more aggressive preventive strategies".

Conclusion

In conclusion, our study demonstrates significant differences in the clinical presentation and angiographic profile of CAD between diabetic and non-diabetic patients. Diabetic patients have a higher prevalence of atypical symptoms, multi-vessel disease, and LV dysfunction compared to non-diabetics. The left circumflex and right coronary arteries are more commonly involved in diabetics, suggesting a predilection for multi-vessel disease. Poor glycemic control is associated with more severe CAD, highlighting the importance of optimal diabetes management. “These findings have important implications for the screening, diagnosis, and management of CAD in diabetic patients, and underscore the need for a multifaceted approach to reduce cardiovascular morbidity and mortality in this high-risk population”.

CONCLUSION

This comparative study demonstrates significant differences in the clinical features and angiographic profiles between diabetic and non-diabetic patients with coronary artery disease. Diabetic patients presented with more atypical symptoms, including a significantly higher prevalence of giddiness and other non-specific complaints, which underscores the importance of maintaining a high index of suspicion for CAD in this population, even in the absence of typical anginal symptoms.

The angiographic findings revealed a strikingly different pattern of coronary artery involvement between the two groups. Diabetic patients exhibited a significantly higher prevalence of multi-vessel disease, with triple-vessel disease being almost twice as common in diabetics compared to non-diabetics. The left circumflex and right coronary arteries were more frequently involved in diabetic patients, suggesting a predilection for diffuse atherosclerosis rather than focal lesions. Furthermore, the absence of normal coronary arteries in the diabetic group highlights the accelerated atherosclerotic process associated with diabetes.

The correlation between poor glycemic control and the severity of CAD was evident in our study, with patients having higher HbA1c levels showing a greater likelihood of multi-vessel disease. This emphasizes the importance of optimal glycemic control in reducing the burden of CAD in diabetic patients. Additionally, the more pronounced left ventricular dysfunction observed in diabetics, as evidenced by lower ejection fractions and higher prevalence of regional wall motion abnormalities, suggests that diabetes negatively impacts myocardial function beyond the effects of epicardial coronary artery stenosis.

In conclusion, diabetes mellitus significantly alters the clinical presentation and angiographic profile of coronary artery disease, leading to more extensive, diffuse, and complex coronary lesions, as well as more pronounced myocardial dysfunction. These findings have important implications for the screening, diagnosis, and management of CAD in diabetic patients. A multifaceted approach including early detection, aggressive risk factor modification, optimal glycemic control, and appropriate revascularization strategies is essential to reduce the cardiovascular morbidity and mortality in this high-risk population.

SUMMARY

INTRODUCTION

Diabetes mellitus is a major risk factor for coronary artery disease (CAD) and is associated with accelerated atherosclerosis, leading to significant cardiovascular morbidity and mortality. The present study aimed to compare the clinical features and angiographic profiles of CAD between diabetic and non-diabetic patients.

AIMS AND OBJECTIVES

To study clinical presentation and angiographic characteristics of coronary artery disease in diabetic patients compared to that of non-diabetics.

MATERIAL AND METHODS

This cross-sectional comparative study included 126 patients (63 diabetics and 63 non-diabetics) who underwent coronary angiography. Demographic characteristics, clinical presentations, electrocardiographic findings, echocardiographic parameters including ejection fraction and regional wall motion abnormalities, and detailed angiographic profiles were analyzed and compared between the two groups.

RESULTS

- This study compared the clinical features and angiographic profiles of 63 diabetic and 63 non-diabetic patients with coronary artery disease. The demographic analysis revealed a predominance of patients in the 51-60 years age group in both diabetic (46%) and non-diabetic (41.3%) populations, with no significant difference in age distribution (p=0.09). Male preponderance was observed in both groups (57.1% in diabetics and 63.5% in non-diabetics), although this gender difference was not statistically significant (p=0.46).

- The clinical presentation showed that chest pain was the most common symptom in both diabetics (87.3%) and non-diabetics (82.6%). However, diabetic patients had a significantly higher prevalence of giddiness (p=0.02) and other atypical symptoms (p=0.04), suggesting altered perception of anginal symptoms possibly due to autonomic neuropathy. The mean duration of diabetes in our study population was 3.42 ± 5.1 years.
- Glycemic control assessment revealed that the majority of diabetic patients had suboptimal control, with 49.2% having HbA1c levels between 8.6-11%, 31.7% between 6.6-8.5%, and 15.8% above 11%. The association between HbA1c levels and the severity of CAD was statistically significant (p=0.009), with higher HbA1c levels correlating with more extensive coronary artery involvement.
- Electrocardiographic findings showed anterior wall myocardial infarction as the most common presentation in both groups (30.2% in diabetics vs 27% in non-diabetics), followed by NSTEMI and IWMI. Although the distribution of ECG findings did not differ significantly between the groups (p=0.71), normal ECG findings were observed in 4.8% of non-diabetics but were absent in diabetics.
- Left ventricular function assessment demonstrated that diabetic patients had a higher prevalence of moderate to severe LV dysfunction (EF <40%) compared to non-diabetics (32.3% vs 14.3%), with this difference being statistically significant (p=0.03). Regional wall motion abnormalities were also significantly more prevalent in diabetics compared to non-diabetics (93.7% vs 77.8%, p=0.05).

- The angiographic profile revealed striking differences between the two groups. Triple-vessel disease was significantly more common in diabetics (42.9% vs 23.8%), while single-vessel disease, minor CAD, and normal coronary arteries were more prevalent in non-diabetics ($p<0.001$). The left anterior descending artery was the most commonly involved vessel in both groups, but the left circumflex ($p=0.001$) and right coronary artery ($p=0.03$) were significantly more involved in diabetics, suggesting a predilection for multi-vessel disease.
- Analysis of the relationship between diabetes duration and CAD severity showed a trend towards a higher prevalence of triple-vessel disease in patients with longer diabetes duration (>10 years), although this did not reach statistical significance ($p=0.09$). Overall, these findings highlight the more extensive, diffuse, and complex nature of coronary artery disease in diabetic patients, emphasizing the need for aggressive risk factor modification and appropriate revascularization strategies in this high-risk population.

CONCLUSION:

Diabetic patients with CAD exhibit more atypical clinical presentations, more pronounced left ventricular dysfunction, and significantly more extensive, diffuse, and complex coronary artery involvement compared to non-diabetics. Poor glycemic control correlates with increased CAD severity. These findings emphasize the importance of early screening, aggressive risk factor modification, and appropriate revascularization strategies in diabetic patients with CAD.

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ANNEXURES

RESEARCH INFORMED CONSENT FORM

BLDE (DEEMED TO BE UNIVERSITY),

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

TITLE OF RESEARCH: **" A Comparative study of clinical features and angiographic profile in diabetics and non-diabetics "**

GUIDE : **Dr. SHASHIDHAR S DEVARMANI**
M.D GENERAL MEDICINE

CO-GUIDE : **Dr. SANJEEV SAJJANAR**
MD,DM CARDIOLOGY

P.G.STUDENT : **Dr. SAMUDRALA SNEHA**

All aspects of this consent form are explained to the patient in the language understood by him or her.

PURPOSE OF STUDY:

I have been informed that the purpose of this study is to compare the clinical and angiographic features in diabetics and non diabetics.

PROCEDURE:

I understand that I will undergo a detailed history and clinical examination and investigations.

BENEFITS:

I understand that my participation in this study will have no direct benefit to me other than the potential benefit of treatment, which is planned to prevent further morbidity and mortality in me.

CONFIDENTIALITY:

I understand that the medical information produced by the study will become a part of hospital records and will be subjected to confidentiality and privacy regulation of the hospital. If the data is used for publication, the identity will not be revealed.

REQUEST FOR MORE INFORMATION:

I understand that I may ask for more information about the study at any time.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary, and I may refuse to participate or withdraw from the study at any time.

(Signature of Guardian) (Signature of patient)

PROFORMA

Name: DOA:

I.P.No.: DOD:

Age: Hospital:

Sex: Address:

HISTORY

Chief complaint:

BRIEF HISTORY OF PRESENTING ILLNESS:

PAST AND ASSOCIATED ILLNESS:

FAMILY HISTORY:

PERSONAL HISTORY:

Diet

Appetite

Sleep

Bowel and bladder

General physical examination

Pulse- BP-

Temp.- RR-

Height- Weight-

SYSTEMIC EXAMINATION

Cardiovascular system:-

Central nervous system:-

Respiratory system:-

Per abdomen examination:-

PROVISIONAL DIAGNOSIS

Treatment detail:

INVESTIGATION:

FBS: mg/dL

PPBS: mg/dL

HbA_1c: %

Urea: mg/dL

Creatinine: mg/dL

Urine Analysis:

Lipid Profile: Total Cholesterol: mg/dL

Triglyceride: mg/dL

HDL Chol.: mg/dL

VLDL Chol.: mg/dL

LDL Chol.: mg/dL

Other Investigations**:**

Hematological

Hemoglobin:

TLC/DLC:

Electrocardiogram::

2D ECHO:

Coronary Angiogram

CONCLUSION:

DATE:

SIGNATURE:

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/ 888/2022-23 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,** scrutinizes 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: "A COMPARATIVE STUDY OF CLINICAL FEATURES AND ANGIOGRAPHIC PROFILE IN DIABETICS & NON-DIABETICS".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.SAMUDRALA SNEHA

NAME OF THE GUIDE: DR.SHASHIDHAR S.DEVARMANI, PROFESSOR, DEPT. OF GENERAL 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: bmpmc.principal@bldedu.ac.in

MASTER CHART

Sl no.	Name	Age	Sex	IP no.	Clinical symptoms				T2DM		ECG	2D-ECHO		CAG	Stenosis(%)
					Chest pain	Breathlessness	Giddiness	Others	Duration	HBA1C		RWMA	LVEF		
1	Rachavva Anagavadi	72y	F	2024-27928	+	+	-	-			LWMI	+	55%	SVD	LAD-80-90%
2	Gurubai Sudam	55y	F	2024-38524	+	-	-	Vomitings			IWMI	-	60%	SVD	RCA-90%
3	Shivappa M Kambogi	65y	M	2024-30301	+	-	-	-			AWMI	+	35%	SVD	LAD-80%
4	Shantabai Malli	65y	F	2024-26542	-	+	+	-			NSTEMI	+	40%	Recanalised LAD	
5	Ganapati Madar	65y	M	2023-46496	+	-	-	-			AWMI	+	40%	TVD	LAD-100% RCA-99% LCX-80%
6	Phulsing Rajaput	74y	M	2023-43635	-	+	-	-			AWMI	+	40%	SVD	LAD-905
7	Shekhavva Kattimani	55y	F	2023-50101	+	-	-	-			NSTEMI	+	50%	SVD	LAD-95%
8	Laxmibai Nidani	75y	F	2023-30200	+	-	-	sweating,palpitations			AWMI	+	45%	SVD	LAD-90%
9	Mahadevi Hatti	60y	F	2023-47341	+	+	-	-			LWMI	+	40%	SVD	LAD-100%
10	Imamsab Bandal	60y	F	2023-46862	+	-	-	-			NSTEMI	+	50%	SVD	LAD-60%
11	Kasturibai Donur	70y	F	2024/9129	+	+	-	-			ASWMI	+	30%	TVD	LCX-90%LAD-100% RCA-50-60%
12	AnandKumar Gudagennavar	61y	M	2023-33995	+	-	-	sweating			NSTEMI	+	30%	DVD	LCX-100% LAD-50% RCA-40%
13	Premanand Shatagar	54y	M	2024/2816	+	-	-	-			IWMI	+	45%	TVD	LAD-70% LCX-60-70% RCA-100%
14	Sharanayya Hiremath	70y	M	2024/2866	+	+	+	-			ASWMI	+	30%	TVD	LAD-70% LCX-30-40% RCA-80-90%
15	Pandit Koli	70y	M	2023-46126	-	+	-	-			S.TACHY	-	60%	MINOR CAD	RCA-40%
16	Rachappa Hadapad	62y	M	2023-48132	-	+	-	-			AWMI	+	40%	TVD	LAD-90% LCX-90% RCA-90%
17	Ravi Mane	41y	M	2024-40077	+	+	-	Palpitations			IWMI	+	35%	TVD	LAD-100% LCX-90% RCA-80%
18	Hanumanth Okali	52y	M	2023-34662	+	-	-	sweating			Normal	-	60%	MINOR CAD	LM- MINOR PLAQUE
19	Dundappa Khed	74y	M	2023-48395	+	+	-	-			AWMI	+	35%	TVD	LAD-95% LCX-90% RCA-70%
20	Somakka Lamani	55y	F	2024-29943	+	-	-	sweating,palpitations			NSTEMI	-	55%	MINOR CAD	LAD-MINOR PLAQUE
21	Chidanand Mathapati	55y	M	2024-33200	+	-	-	sweating,palpitations			UA	-	60%	MINOR CAD	LAD/LCX-MINOR PLAQUE

22	Babu Jadhav	53y	M	2024-22040	+	-	-	-			ILWMI	+	45%	DVD	LAD-90% LCX-90%
23	Maiboob Gachan	34y	M	2024-32900	+	-	-	-			AWMI	+	55%	Recanalised LAD	LAD-MINOR PLAQUE
24	Basagond Biradar	72y	M	2023-49232	-	+	-	-			LWMI	+	40%	SVD	LCX-100%
25	Sangappa Kademani	60y	M	2023-48389	-	+	-	-			NSTEMI	+	45%	TVD	LAD-90% LCX 80% RCA 100%
26	Guralingappa Goranal	70y	M	2024-29419	+	+	-	-			UA	-	60%	MINOR CAD	RCA-MINOR PLAQUE
27	Sumitra Soudi	54y	F	2024-29291	-	+	-	-			UA	-	60%	NORMAL	
28	Nilappa Halli	60y	M	2024-29411	+	-	-	Sweating			ASWMI	+	30%	TVD	LAD-100% LCX-90% RCA-80%
29	Sumitra Hebberi	44y	F	2024-26940	+	-	-	-			UA	-	60%	NORMAL	
30	Channappa Dapli	55y	M	2023=25737	+	-	-	sweating,vomitings			IWMI	+	50%	DVD	LCX-50% RCA-80%
31	Bapuray Sarawad	61y	M	2023-27818	+	-	-	sweating			AWMI	+	50%	SVD	LAD-50-60%
32	Kanchana Tambhat	65y	F	2023-36485	+	+	+	-			IPWMI	+	45%	DVD	LCX-90% LAD-MINOR PLAQUE
33	Sharanappa Kharath	40y	M	2023-41453	+	-	-	vomitings			NSTEMI	-	55%	NORMAL	LAD-MINOR PLAQUE
34	Shivalingavva Yalawar	68y	F	2023-43109	-	+	-	Palpitations			AWMI	+	45%	SVD	LAD-95%
35	Suvarna Bilagi	65y	F	2023-43806	-	+	-	-			IWMI	+	60%	MINOR CAD	LAD-MINOR PLAQUE
36	Chandubai Pawar	67y	F	2023-54232	+	-	-	-			NSTEMI	+	45%	MINOR CAD	LAD-MINOR PLAQUE
37	Ashok Araballi	67y	M	2024-33556	+	+	-	Sweating			UA	+	30%	DVD	LAD-99% LCX-45% RCA-50%
38	Shankar Katate	55y	M	2024-31335	+	+	-	-			AWMI	+	30%	DVD	LAD-70% RCA-60%
39	Ambavva Pawar	55y	F	2024-28030	+	-	-	-			AWMI	+	50%	SVD	LAD-99%
40	Shivappa Maddi	62y	M	2024-28252	+	+	-	sweating,palpitations			AWMI	+	60%	MINOR CAD	LAD-50%
41	Sunanda Mirajkar	54y	F	2024-28006	+	+	-	-			ASWMI	+	40%	DVD	LAD-90% RCA-90%
42	Suresh Koti	52y	M	2023-51547	+	-	-	-			AWMI	+	45%	SVD	LAD-90%
43	Ambadas Kinnur	34y	M	2023-44668	+	-	-	sweating,palpitations			AWMI	+	45%	SVD	LAD-100%
44	Sangavva Mogali	73y	F	2023-48925	+	+	-	-			LWMI	+	45%	TVD	LAD-100% LCX-90% RCA-100%
45	Basappa Narali	75y	M	2024/2034	+	-	-	sweating			IWMI	+	45%	SVD	LAD-50% RCA-100%
46	Devanand Khade	36y	M	2025101221	+	-	-	-			NORMAL	-	60%	MINOR CAD	LAD/LCX-50%
47	Vitthal Munjanni	55y	M	2025110206	+	-	-	-			NORMAL	+	60%	TVD	LAD-100% LCX-100%
48	Nagappa Malloli	55y	M	2025/994	+	-	-	-			AWMI	+	60%	DVD	LAD-100% LCX-60%
49	Dundavva Bolegaon	74y	F	2024-26840	+	-	-	Vomitings			IPWMI	+	40%	TVD	LAD-90% RCA-90% LCX-70%
50	Supurabegam Jamadar	54y	F	2025000889	+	-	-	-			UA	-	60%	MINOR CAD	LAD-MINOR PLAQUE
51	Dareppa Harijan	58y	M	2024-30809	-	+	-	-			IWMI	+	40%	DVD	LAD-50% RCA-100%
52	Shreekar Hiremath	55y	M	2501140048	+	-	-	-			NSTEMI	+	50%	MINOR CAD	LAD-40%
53	Mataji Handi	37y	F	2024-27566	+	+	-	-			AWMI	-	60%	SVD	LAD-90%

54	Chandsab Devar	60y	M	2024-11484	+	+	-	sweating			IPWMI	+	50%	TVD	LAD-50% LCX-80% RCA-80%
55	Bhimanna Kumbar	68y	M	2024-39373	+	-	-	sweating,palpitations			UA	+	50%	SVD	LAD-90%
56	Savita Bangaratali	35y	F	2024-15137	+	+	-	sweating			NSTEMI	+	50%	SVD	RCA-60%
57	Ramesh Hiregana	50y	M	2024-17917	-	+	-	-			NSTEMI	+	55%	NORMAL	
58	Ningayya Matapati	69y	M	2024-17407	+	+	-	sweating			ALWMI	+	40%	DVD	LAD-95% RAMUS-90% RCA-90%
59	Ramesh Radder	58y	M	2024-17755	+	-	-	-			AWMI	+	40%	SVD	LAD-70% LCX-40%
60	Parashuram Mulimani	52y	M	2024-20543	+	+	-	-			LWMI	+	50%	SVD	LAD-95%
61	Chandappa Harijan	58y	M	2024-20542	+	-	-	-			UA	-	60%	MINOR CAD	LAD-40%
62	Muneer Jath	50y	M	2024-00020	+	-	-	sweating,palpitations			IWMI	+	50%	SVD	LAD-30% RCA-100%
63	Laxmi Sonar	61y	F	2024-4044	+	-	-	-			UA	-	60%	SVD	LAD-95%
64	Rauf Ansari	52y	M	2024/4373	-	+	-	-	14y	9.1	NSTEMI	+	60%	SVD	LCX-90%
65	Laxmibai Patil	55y	F	2024/3626	+	-	-	-	7y	7.8	UA	-	35%	SVD	LAD-80%
66	Halima Hattaraki	61y	F	2024/3691	+	-	-	-	20y	8.7	LWMI	+	45%	TVD	LAD-80% LCX/RCA-100%
67	Banuma Mulla	66y	F	2024-00069	+	+	-	sweating,palpitations	5y	7.9	ASWMI	+	35%	TVD	LAD/LCX/RCA-90%
68	Kashinath Baichabai	61y	M	2024-39344	+	-	-	-	10y	8.4	NSTEMI	-	60%	DVD	LAD-90% RCA-80%
69	Gopu Chawan	72y	M	2024-39072	+	-	-	sweating,palpitations	8y	10.2	AWMI	+	40%	SVD	LAD-100% LCX-40%
70	Laxmibai Waliker	59y	F	2023-46130	+	-	+	-	ND	11.3	AWMI	+	50%	SVD	LAD-95% RCA-40%
71	Ramappa Bilur	45y	M	2024-225541	+	+	-	-	5m	9.2	LWMI	+	30%	SVD	LAD-100%
72	Malakappa Waddar	54y	M	2024-38511	+	-	-	-	4y	13.4	NSTEMI	+	40%	TVD	LAD/RCA-90% LCX-80%
73	Nagappa Shahpur	54y	M	2024-28664	+	+	-	-	3y	9.3	IWMI	+	30%	TVD	LAD/RCA-100% LCX-90%
74	Sharanappa Doddamani	60y	M	2024-27768	-	+	-	-	2y	9.1	ASWMI	+	30%	DVD	LAD-100% LCX-90%
75	Aishyabi Tambe	72y	F	2024-27048	+	+	-	-	4y	7.6	NSTEMI	+	50%	DVD	LAD-50% LCX-90%
76	Shantabi Gadave	53y	F	2023-40569	+	+	-	-	15y	10.2	AWMI	+	50%	TVD	LAD-80% LCX-70% RCA-90%
77	Golappa Deginal	78y	M	2023-54767	+	-	-	sweating,palpitations	5y	6.4	AWMI	+	40%	DVD	LAD-90% LCX-40% RCA-60%
78	Basavanand Kumbar	64y	M	2024-39612	+	-	-	sweating	ND	10.1	UA	+	55%	DVD	LCX-95% RCA-80%
79	Meenakshi Kattimani	55y	F	2024-29750	+	+	-	-	16y	8.5	NSTEMI	+	60%	TVD	LAD-80% LCX-50% RCA-80%
80	Nanabai Thorbole	48y	F	2023-52652	+	+	-	sweating	5y	8.9	AWMI	+	35%	SVD	LAD-100%
81	Dhareppa Kilari	50y	M	2024-34253	+	-	-	sweating,palpitations	2y	8.6	IWMI	+	45%	SVD	LCX-90%
82	Shrishail Gadade	53y	M	2024-34472	+	-	-	sweating,palpitations	2y	8.2	UA	+	45%	DVD	LAD-100% LCX-95% RCA-90%
83	Ambawwa Mali	53y	F	2023-37165	+	-	-	-	10Y	11.8	AWMI	+	35%	TVD	LAD-90% LCX-100% RCA-100%
84	Iraj Hottagi	42y	M	2023-36935	+	-	-	-	ND	9.7	IWMI	+	50%	SVD	RCA-80%
85	Sharanagouda Biradar	52y	M	2024-38520	+	+	-	sweating	8y	12.4	AWMI	+	45%	SVD	LAD-80% RCA-40%

86	Iranna Hadapad	39y	M	2024-12821	+	-	-	-	2y	9.9	AWMI	+	40%	TVD	LAD-50% LCX-90% RCA-95%
87	Dastagiri Moulasab	65y	M	2024-12279	+	-	-	sweating	4y	8.3	AWMI	+	35%	RECANALISED LAD	
88	Muneer Ahmed Mulla	60y	M	2024-12162	+	+	+	sweating,palpitations	3y	6.8	ILWMI	+	30%	TVD	LAD-70% LCX-70% RCA-60%
89	Kudaratali Attar	56y	M	2024-38912	+	-	-	sweating,palpitations	2y	8.6	NSTEMI	+	40%	TVD	LAD-90% LCX-90% RCA-90%
90	Siddappa Pujari	75y	M	2024-91320	+	-	-	sweating,palpitations	1y	8.3	IWMI	+	45%	DVD	LAD-90% LCX-50% RCA-100%
91	Bapulal Jamadar	60y	M	2024-06144	+	+	-	sweating,palpitations	5y	8.6	AWMI	+	35%	TVD	LAD-70% LCX-70% RCA-60%
92	Basamma Ulkal	65y	F	201948	-	+	-	-	2y	7.6	AWMI	+	35%	TVD	LAD-100% LCX-30% RCA-90%
93	Rudragouda Biradar	62y	M	207180	+	-	-	-	10y	8.8	AWMI	+	45%	DVD	LAD-100% LCX-60% RCA-90%
94	Sadashivayya Mathad	60y	M	2023-30187	-	+	-	-	4y	8.6	ILWMI	+	50%	TVD	LAD-90% LCX-50% RCA-100%
95	Indubai Shahpur	55y	F	2023-32598	+	+	-	-	10y	9	NSTEMI	+	35%	TVD	LAD-100% LCX-90% RCA-90%
96	Kallappa Budihal	78y	M	2023-38597	+	+	-	-	10y	6.2	NSTEMI	+	55%	TVD	LAD-70% LCX-90% RCA-%
97	Jaibunissa Fouji	63y	F	2023-39838	-	-	-	Vomitings,loose stools	23y	7.7	IWMI	+	35%	TVD	LAD-90% LCX-100%
98	Laxmibai Gangasetti	51y	F	2024-4068	+	+	-	-	7y	8.2	ASWMI	+	25%	DVD	LAD-95% LCX-80% RCA-40%
99	Mallikarjun Teggihalli	46y	M	2024-58505	+	+	-	-	2y	12.8	AWMI	+	40%	TVD	LAD-80% LCX-90% RCA- 50%
100	Rayawwa Nimangri	45y	F	2024-30064	-	-	-	sweating,vomitings	8yr	12.4	AWMI	+	40%	TVD	LAD-70% LCX-50% RCA-80%
101	Kamalabai Hiremath	43y	F	2024-24430	+	-	-	sweating,palpitations	ND	8.2	NSTEMI	+	45%	SVD	LAD-90%
102	Shantabai Jadhav	74y	F	2024-00055	+	-	-	sweating,palpitations	10y	8.1	IWMI	+	40%	SVD	RCA-95%
103	Kasturibai Navi	55y	F	2024-34682	+	+	-	-	12y	8.5	AWMI	+	25%	TVD	LAD-90% LCX-90% RCA-60%
104	Nilavva Hegadi	59y	F	2024-0070	+	-	-	sweating,palpitations	7y	9.6	AWMI	+	40%	DVD	LAD-90% LCX-90% RCA-40%
105	Danamma Savalagi	62y	F	2501131409	+	-	-	-	7y	8.2	NSTEMI	+	60%	DVD	LAD-60% LCX-90% RCA-50%
106	Umar Mulla	76y	M	250110160	-	+	-	-	15y	9.2	NSTEMI	+	40%	TVD	LAD-90% LCX-90% RCA-90%
107	Parvati Misal	77y	M	2024-00339	+	-	-	sweating,palpitations	6y	8.1	IWMI	+	50%	DVD	LAD-90% RCA-90%
108	Shantabai Jummanagol	53y	F	2024-18335	+	-	-	-	11y	12.9	UA	+	45%	TVD	LAD-90% LCX-90% RCA-100%
109	Ramangouda Patil	66y	M	2024-16456	+	+	-	-	3y	10.3	NSTEMI	+	35%	TVD	LAD-90% LCX-95% RCA-100%
110	Hamedabi Nadaf	60y	F	2024-15291	+	+	-	-	10y	12.1	UA	+	60%	TVD	LAD-90% LCX-70% RCA-100%
111	Pasha Makandar	55y	F	2024-13946	+	-	-	sweating	4y	9.2	UA	+	55%	TVD	LAD-90% LCX-90% RCA-100%
112	Bibanabi Attar	50y	F	2024-10700	+	-	-	sweating,palpitations	7y	7.5	AWMI	+	35%	SVD	LAD-100% RCA-60%
113	Vishnu Pawar	58y	M	2024-18179	+	-	-	sweating	11y	9.2	IPWMI	+	45%	DVD	LAD-90% RCA-90%
114	Gangappa Himakar	47y	M	2024-17403	+	-	-	sweating	4y	8.2	AWMI	+	40%	SVD	LAD-100%
115	Mahipati Thangale	70y	M	2024-17169	+	+	-	sweating	2y	9.1	IWMI	+	50%	DVD	LAD-70% LCX-50 RCA-90%
116	Mahadev Kori	60y	F	2024-16830	+	-	-	sweating	15y	8.8	ASWMI	+	35%	SVD	LAD-80%
117	Siddappa Harijan	62y	M	2024-16244	-	+	+	-	1y	9.7	IWMI	+	30%	DVD	LAD-70% LCX-40% RCA-60%

118	Mahadevi Navadagi	65y	F	2024-11324	+	-	-	sweating,palpitations	13y	10.4	IWMI	+	45%	TVD	LAD-100% LCX-90% RCA-95%
119	Shoba Rathod	43y	F	2024-11711	+	-	-	-	5y	7.9	NSTEMI	-	60%	RECANALISED PLV	LAD-40%
120	Mallikarjun Tallolli	55y	M	2024-11753	+	-	-	sweating	9y	11.3	IPWMI	+	45%	TVD	LAD-90% LCX-95% RCA-90%
121	Gurulingappa Bhavikatti	60y	M	2024-39830	+	+	-	-	1m	9	IWMI	+	50%	TVD	LAD-90% LCX-95% RCA-100%
122	Aziz Patel	56y	M	2024-13940	+	-	-	-	1y	12.4	IWMI	+	50%	DVD	LAD-90% RCA-60%
123	Basappa Mashyal	75y	M	2024-19769	+	+	-	sweating	20y	11.9	AWMI	+	35%	TVD	LAD-90% LCX-60% RCA-100%
124	Kasturibai Singe	55y	F	2024-19597	+	+	-	-	8y	8.6	NSTEMI	+	55%	DVD	LAD-90% RCA-50%
125	Gautham Bhade	44y	M	2024-20094	+	-	-	-	5y	11.7	ALWMI	-	55%	TVD	LAD-90% LCX-100% RCA-100%
126	Prabhugouda Biradar	55y	M	2024-20373	+	+	-	sweating	6m	8.6	NSTEMI	+	40%	SVD	LAD-100%

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