"CROSS SECTIONAL STUDY COMPARING IMMATURE PLATELET FRACTION WITH DEGREES OF HYPERGLYCEMIA IN TYPE 2 DIABETES MELLITUS IN POST CORONARY ANGIOGRAPHY PATIENTS AT KLES DR. PRABHAKAR KORE HOSPITAL"

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65

ABBREVIATIONS

ACS	-	Acute Coronary Syndrome
ALP	-	Alkaline Phosphatase
aPTT	-	Activated Partial Thromboplastin Time
CAD	-	Coronary Artery Disease
CVD	-	Cardiovascular Disease
DALY	-	Disability Adjusted Life Years
ECG	-	Electrocardiography
ECHO	-	Echocardiography
HDL	-	High-Density Lipoprotein
HbA1C	-	Glycated Hemoglobin
INR	-	International Normalized Ratio
IPF	-	Immature Platelet Fraction
LBBB	-	Left Bundle Branch Block
LDL	-	Low-Density Lipoprotein
LVH	-	Left Ventricular Hypertrophy
MI	-	Myocardial Infarction
MPV	-	Mean Platelet Volume
NCDs	-	Non-Communicable Diseases
PDW	-	Platelet Distribution Width
RBBB	-	Right Bundle Branch Block
RP	-	Reticulated Platelets
SGOT	-	Serum Glutamic Oxaloacetic Transaminase
SGPT	-	Serum Glutamic Pyruvic Transaminase
TNF-α	-	Tumor Necrosis Factor-α
TPO	-	Thrombopoietin
vWF	-	von Willebrand Factor

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ABSTRACT

Title: "Cross Sectional Study Comparing Immature Platelet Fraction with Degrees of Hyperglycemia in Type 2 Diabetes Mellitus in Post Coronary Angiography Patients at KLES Dr. Prabhakar Kore Hospital"

Background: Diabetes mellitus is among the primary causes of morbidity and mortality globally. Cardiovascular causes account for maximum morbidity and mortality in the diabetic population. Platelets are a key component in development of coronary artery disease. Diabetes is associated with accelerated platelet turnover and high reticulated platelets (RPs) or immature platelets. These are larger and more sensitive, with hyperactivity and low response to antiplatelet therapy. Immature platelet fraction is a marker which detects the turnover of circulating platelets in the blood. Immature platelet fraction (IPF) and MPV can be added to screening methods for coronary artery disease. They can serve as a risk indicator for patients by acting as an early hematological marker.

Methods: In this cross-sectional study we included 130 patients who were undergoing coronary angiography and were either newly diagnosed or known cases of diabetes mellitus with uncontrolled sugars or on medication. Patients with platelet count <1 lakh or >4.5 lakhs were excluded from the study. Patients with abnormal coronary angiography findings were further evaluated. Random blood sugar, fasting blood sugar and HbA1C were checked. Immature platelet fraction (IPF) and platelet indices like platelet count, mean platelet volume (MPV) was checked. These values were compared and correlated to evaluate immature platelet fraction and degree of diabetes mellitus in post coronary angiography patients. Mean platelet values was also compared with diabetic status and coronary angiography findings.

Result: In our study, a total of 130 participants were enrolled, of which 87 (66.9%) were males and 43 (33.1%) were females. The most common age bracket was 51 to 60 years old with 49 (37.7%) persons. In our study, we compared MPV and IPF with HbA1C, fasting blood glucose and random blood glucose, to observe association with diabetic status. Higher HBA1C was associated with increased mean platelet volume ($p = 0.006$). There was also a statistically significant positive correlation between Fasting Sugars and Mean Platelet Volume ($p = 0.007$). Mean platelet volume was more for patients with advanced coronary angiography findings (triple vessel disease) ($p = 0.011$). In our study, there was no statistically significant correlation between HbA1C and IPF ($p = 0.706$). IPF weakly correlated with fasting sugars ($p = 0.046$). Mean platelet volume was significantly correlated with IPF ($p = <0.001$). The median Mean Platelet Volume was higher in the group on antiplatelet therapy ($p = 0.017$). Total cholesterol was increased in patients with higher IPF values ($p = 0.027$).

Conclusion: Our study shows MPV to be a useful, inexpensive and rapid hematological marker for correlating diabetes and coronary artery disease outcome. There was a strong correlation between MPV and IPF values, indicating most larger platelets are also reticulated and newly produced from the marrow. There was a weak correlation between fasting sugars and IPF, indicating the need for a larger sample size to evaluate the true relationship.

Keywords: Immature platelet fraction (IPF), Mean platelet volume (MPV), Diabetes mellitus (DM), atherosclerotic burden, coronary angiography (CAG)

INTRODUCTION

Diabetes mellitus is among the primary causes of morbidity and death globally. The number of people suffering from diabetes is expected to gradually increase, with a predicted population growth rate of 20% and diabetes growth rate of 46%. India ranks second in number of people suffering from diabetes with estimated prevalence of 74.2 million in 2021[1].

Obesity and prediabetes are highly prevalent in India, which indicates an upcoming growth in the prevalence of diabetes in India[2]. Uncontrolled diabetes causes increased risk of vascular diseases including macrovascular and microvascular complications. Macrovascular complications encompass cerebrovascular, cardiovascular and peripheral artery disease. Microvascular complications encompass diabetic retinopathy, neuropathy and nephropathy[3].

Only 7% of Indian population meets the treatment guidelines for blood glucose, blood pressure and lipids when on medication[2].

Cardiovascular diseases are another major cause morbidity and mortality worldwide[4]. India accounts for the maximum number of ischemic heart disease and ST elevated myocardial infarction worldwide[5]. Cardiovascular causes also account for maximum morbidity and mortality in the diabetic population[6].

Diabetes mellitus and obesity can lead to insulin resistance which causes an increase in the cytokines released by the adipose tissue. This increase in cytokine expression causes inflammation and lipid accumulation in blood vessels, which leads to endothelial dysfunction, myocardial infarction and cardiomyopathies. Diabetes mellitus also increases C reactive protein expression, increasing low density lipoprotein (LDL) uptake in coronary vessels. This increases endothelial dysfunction and atherosclerotic plaque formation[7].

Insulin resistance also increases free fatty acid release from adipose tissue. This promotes triglyceride production, which increases the risk of atherosclerosis and cardiovascular disease. Hyperglycemia increases glycosylation and oxidation of lipoproteins, decreases vascular compliance and increases rate of atherosclerosis[8].

Diabetes also causes increase in coagulation, impaired fibrinolysis, endothelial dysfunction and increased platelet activity, resembling a prothrombotic state. This is a crucial mechanism in the formation of atherothrombosis in diabetic individuals, increasing the risk of coronary events. Platelet activity increase is seen early in diabetes and precedes development of cardiovascular disease[9].

Elevated triglycerides (TG) and reduced high-density lipoprotein (HDL) are seen in dyslipidemia associated with diabetes mellitus. This combination enhances platelet activation and leads to endothelial dysfunction, which increases the risk of atherosclerosis[10].

Platelets are a key component in development of coronary artery disease. Diabetes is associated with accelerated platelet turnover and high reticulated platelets (RPs) or immature platelets. These are larger and more sensitive, with hyperactivity and low response to antiplatelet therapy[11].

Immature platelet fraction is a marker which detects the turnover of circulating platelets in the blood[12]. Platelets which are released from the bone marrow have larger size with increased protein synthesis and aggregating capacity[13]. Larger number of reticulated platelets are also a marker for inadequate response to antiplatelet therapy[14].

Mean platelet volume (MPV) increases in myocardial infarction is linked to unfavorable results, reduced angiographic reperfusion, increase rate of reinfarction and increased residual thrombosis post fibrinolysis. Immature platelet fraction (IPF) and MPV can be added to screening methods for coronary artery disease. They can serve as a risk indicator for patients by acting as an early hematological marker[13].

Immature platelet fraction increases in diabetic patients and shows correlation with failure of glycemic control and presence of cardiovascular disease in the general population[15].

OBJECTIVE

- To compare mean platelet volume (MPV) with hyperglycemia in diabetes mellitus patients and coronary angiography findings
- To compare immature platelet fraction (IPF) levels with hyperglycemia in diabetes mellitus patient and coronary angiography finding
- To correlate immature platelet fraction (IPF) with mean platelet volume (MPV) levels
- To correlate mean platelet volume (MPV) levels with antiplatelet use and coronary atherosclerotic burden

REVIEW OF LITERATURE

Review of Literature

Burden of Diabetes

One of the top ten causes of death worldwide and one of the biggest health issues of the century is diabetes[1]. The World Health Organization (WHO) reports that non-communicable diseases (NCDs) accounted for 74% of all deaths worldwide in 2019. Among them, diabetes mellitus resulted in 1.6 million deaths, making it the ninth most common reason behind mortality globally[16].

Current global statistics show 536.6 million people suffer from diabetes and 541 million people have impaired glucose tolerance (IGT), a prediabetic condition. By 2045, it is anticipated that there would be 730.3 million IGT patients and 783.2 million diabetic patients. While world population growth rate is expected to be 20%, the rate of diabetes growth is projected at 46%[1]. Figure 1 illustrates the changing trend in prevalence of diabetes worldwide.

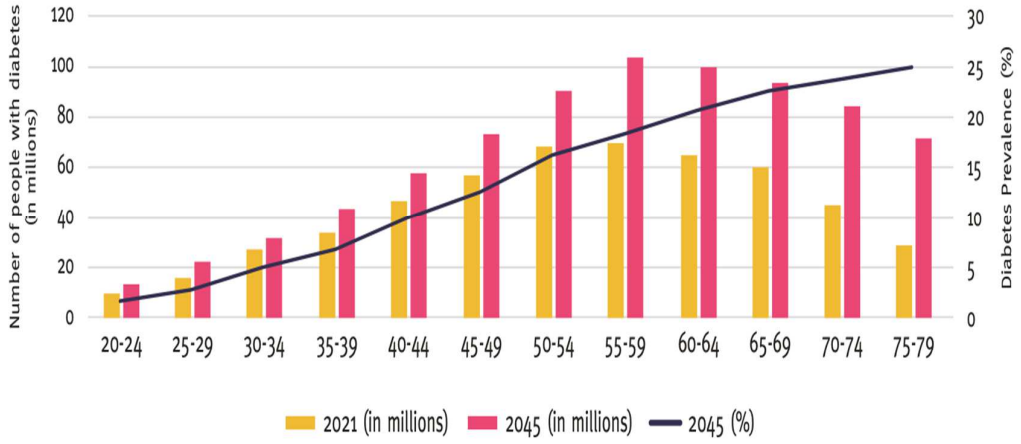

Figure 1. Prevalence of Diabetes across age groups in 2021 and estimated prevalence in 2045

The prevalence of diabetes is highest among adults aged 50 to 60 years old in 2021. With increase in the aging world population, an increase in those with diabetes over the age of 60 years will be seen. In 2021, there are 17.7 million more men than women with diabetes[1].

According to WHO, diabetes mellitus is becoming more and more common in low- and middle-income nations[17]. According to data from 2021, China (140.9 million), India (74.2 million), and Pakistan (33 million) have the highest rates of adult diabetes[1]. Evaluation of incidence is a key element in tracking the progress of chronic diseases such as diabetes and monitoring efficacy of treatment.

Another lacuna in the prevalence of diabetes is the proportion of undiagnosed diabetic individuals. Based on the World bank income classification, 28.8% (29.9 million) remain undiagnosed in high-income countries, 48.4% (200.4 million) in middle-income countries and 50.5% (9.5 million) in low-income countries[1]. These numbers reflect an urgent need for improvement in diabetes screening. Timely diagnosis and treatment or diabetes can prevent development of fatal complications.

India is seeing a surge in the prevalence of non-communicable diseases as part of an epidemiological shift. Several studies over the past few years have shown an increase in prevalence of diabetes, hypertension, and dyslipidemia, with a substantial amount of the global load falling on India[2].

The ICMR-INDIAB trial conducted in 2023 across India has estimated prevalence of diabetes to be 101 million and prediabetes to be 136 million[2]. The International Diabetes Federation (IDF) projects that 8.3% of Indians have been diagnosed with diabetes in 2021, 5.4% have impaired glucose tolerance and 7.8% have impaired fasting glucose[1]. Figure 2 illustrates the prevalence of diabetes and

other parameters of metabolic syndrome, including hypertension, obesity, and dyslipidemia, in India according to the ICMR-INDIAB trial.

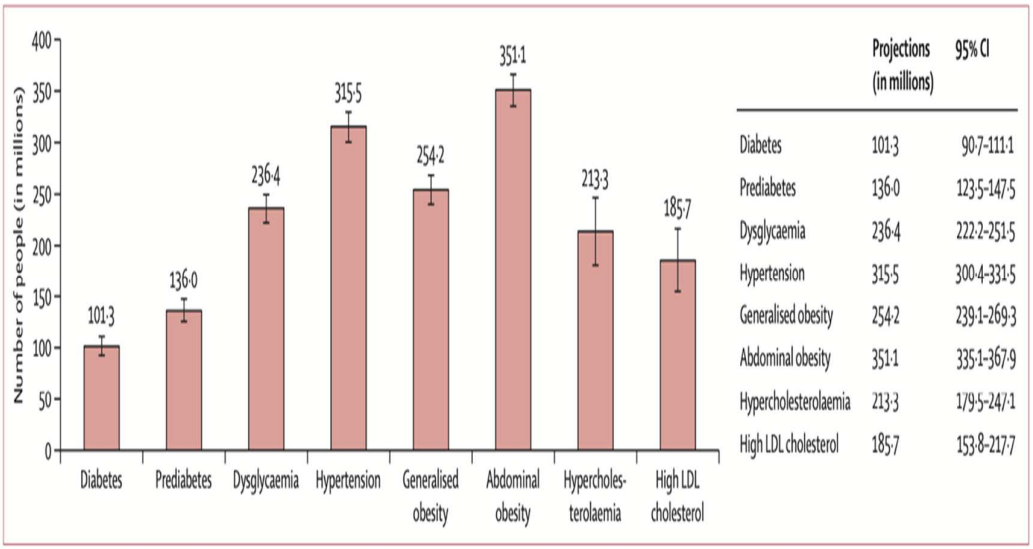

Figure 2. Diabetes prevalence and metabolic syndrome indicators

Even in areas of India where the incidence of diabetes is now low, high rates of obesity and prediabetes indicate that the prevalence will only rise. Lower obesity levels (body mass index) and a quick transition from prediabetes to diabetes are common in Indian Asians[2].

The incidence of diabetes is continuously rising in India along with the prevalence of the disease, and the disease is rapidly progressing from euglycemia to prediabetes and diabetes[18]. The rates of occurrence of diabetes, prediabetes, and dysglycemia were 22.2, 29.5, and 51.7 per 1000 person-years, respectively, in the Chennai Urban Rural Epidemiological Study (CURES) cohort. In individuals with normal blood glucose, the conversion rate of diabetes was 19.4%, whereas in those with prediabetes, it was 58.9%. Diabetes incidence was 78.9 per 1000 person-years in those with prediabetes[3].

The mean healthcare expenditure on diabetes diagnosis, treatment, and related complications per person in India is approximately 114.4 USD per year. The total deaths which are attributable to diabetes and its complications are 647,831 per year in India[1].

Diabetic retinopathy, nephropathy, and neuropathy are examples of microvascular problems that are brought on by uncontrolled diabetes. There is an increased risk of macrovascular disorders such as peripheral artery disease, heart disease, and cerebrovascular accidents[3].

Merely 7% of individuals diagnosed with diabetes in India achieve the prescribed levels of blood pressure, lipids, and blood glucose when taking treatment. For people with undetected illnesses, the percentage is much lower[2].

Diagnosis of Diabetes, Risk Factors and Complications

The WHO describes diabetes mellitus as "a chronic metabolic condition marked by increased blood glucose levels. Over time, this disease can cause damage to the heart, blood vessels, eyes, kidneys, and nerves"[19]. Type 2 diabetes mellitus is the commonest subtype and accounts for 90% cases[1].

Peripheral tissue resistance to insulin and impaired pancreatic β-cell insulin production are the two main mechanisms of type 2 diabetes. Diabetes involves the pancreas, liver, skeletal muscles, kidneys, brain, small intestine, and adipose tissue. Additionally, recent research indicates that inflammation, aberrant gut microbiota, and deregulation of adipokines are significant contributors to the pathogenesis[19].

Figure 3 illustrates the International Diabetes Federation guidelines for diagnosis of diabetes.

National recommendations advise against using HbA1c as the only diagnostic marker for diabetes and prediabetes because of the significant prevalence of iron deficiency anemia in India[2].

Figure 3. Modified diagnostic criteria for diabetes.

The etiology of diabetes is multifactorial. Non-modifiable risk variables at the individual level include family history, age, ethnicity, and genetics. Modifiable risk factors have been the principal cause of the significant rise in the prevalence of diabetes, including absence of exercise, sedentary lifestyle, increasing prevalence of

obesity, unhealthy diet and beverages, habits (smoking and alcohol consumption), environmental pollutants and mental health (stress, depression, lack of sleep)[1].

According to the Global Burden of Disease Study 2016, the major risk factors for disability adjusted life years (DALYs) and diabetes-related mortality include tobacco use, poor consumption of fruits, nuts, seeds, and whole grains in the diet, and obesity[18].

Traditional complications of diabetes include microvascular and macrovascular conditions including retinopathy, nephropathy, neuropathy, coronary artery disease, cerebrovascular disease, and peripheral vascular disease. Diabetes also causes impairment of musculoskeletal, hepatic, and gastric systems, decline in cognitive function and mental health[20].

Over the past ten years, studies conducted in high-income nations have demonstrated that diabetic patients have at least ten to twenty times the morbidity from microvascular problems compared to the non-diabetic population. In diabetics, the rates of macrovascular problems were two to four times higher than in non-diabetics[20]. In comparison to the Western population, the CURES study carried out in Southern India revealed a higher prevalence of coronary artery disease (CAD) and a lower prevalence of peripheral vascular disease, nephropathy, neuropathy, and retinopathy[3].

There is a higher risk of complications seen with developing economies (middle- and low-income countries) like India. This may be attributed to delayed diagnosis of diabetes mellitus and its associated complications, any concurrent medical conditions, inadequate systems of health care, and increased cost of diagnosis and treatment which leads to poor control[18].

The total deaths attributable to diabetes and its complications in India are 647,831 per year[1]. A study from Srinagar showed 16,690 mortalities amongst 234,776 total enrolled. The primary reasons of mortality were infections (41%), chronic renal failure (33.6%), coronary artery disease like myocardial infarction (16.9%), cerebrovascular disease like stroke (13.2%) and chronic obstructive pulmonary disease (6.9%)[21]. According to the CURES study, the death rate for individuals with diabetes was four times greater than that of those without the disease (27.9 per 1000 person-years against 8 per 1000 person-years). Furthermore, research showed that the two conditions with the highest population-attributable risk for all causes of death were diabetes and ischemic heart disease[3].

Burden of Coronary Artery Disease

Cardiovascular diseases (CVDs) are the leading cause of mortality and morbidity worldwide[4]. In India, cardiovascular disease contribution to death and disease burden has almost doubled since 1990. Owing to the increasing burden of diseases, India's National Health Policy 2017 sought to lower 25% of cardiovascular disease-related premature deaths and treat 80% of patients with hypertension through screening by the year 2025[4].

According to the 2010 Global Burden of Disease survey, cardiovascular illnesses account for 24.8% of fatalities in India[22]. Figure 4 shows a comparison of cardiovascular mortality in various regions based on prospective studies conducted[22]. Three prospective trials conducted in India, in Mumbai, Kerela and Andhra Pradesh show higher proportion of mortality attributable to cardiovascular diseases (30% to 42%) compared with Global Burden of Disease study[22].

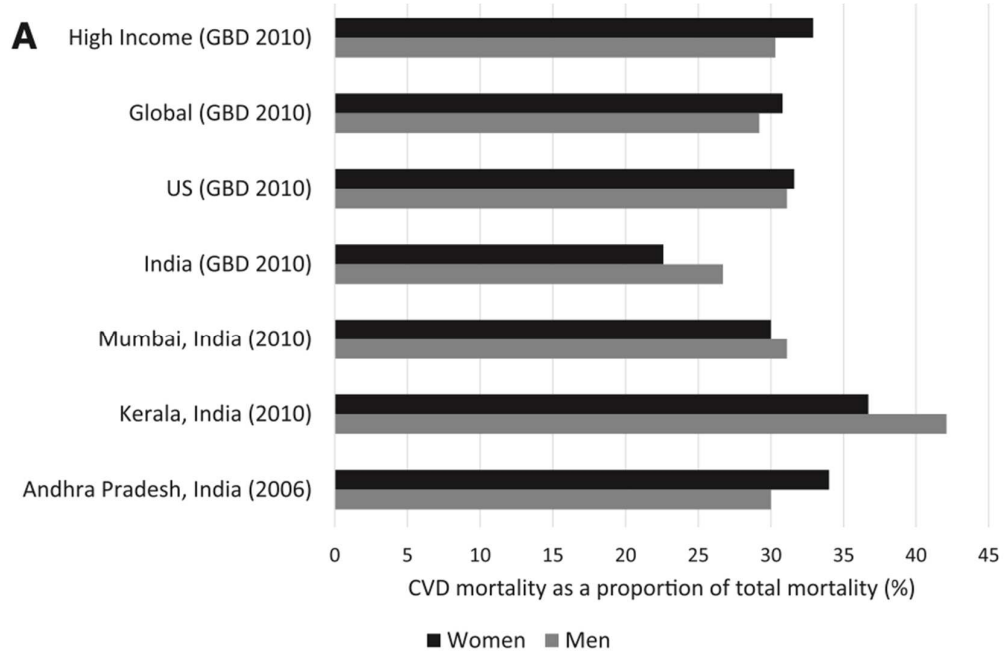

Figure 4. Prevalence of mortality related to cardiovascular diseases, comparison of various prospective studies.

Cerebrovascular disease (stroke) and cardiovascular disease (ischemic heart disease) represent 83% of all deaths in India from CVD, with IHD being more frequent. In India, both causes over one-fifth (21.1%) of all deaths and one-tenth of all years of life lost[22]. Indians currently contribute highest to the number of acute coronary syndrome and ST elevated myocardial infarction cases[5].

Hospitalisation for complications of coronary artery disease (CAD) are two-to-four-fold more in the Indian population, with a five-to-ten-fold admission rate for population younger than 40 years old. The prevalence of CAD in India is 21.4% for diabetics and 11% for nondiabetics. The prevalence is nearly half in rural areas compared to urban regions of India[5].

Risk factors frequently linked to a higher incidence of coronary artery disease in India are hypertension, diabetes, dyslipidemias, smoking and obesity. Nine common risk factors (including low fruits and vegetable, intake physical inactivity,

psychosocial stressors) contributed to more than 90% of acute myocardial infarctions (acute MI) in South Asians according to the INTERHEART study[23].

According to the Global Burden of Disease India, the crude disability adjusted life years (DALYs) of ischemic heart disease from 1990 to 2016 increased by 33%, and prevalence increased by 53%[4]. Figure 5 illustrates the DALYs in ischemic heart disease patients with percentage contribution of each risk factor.

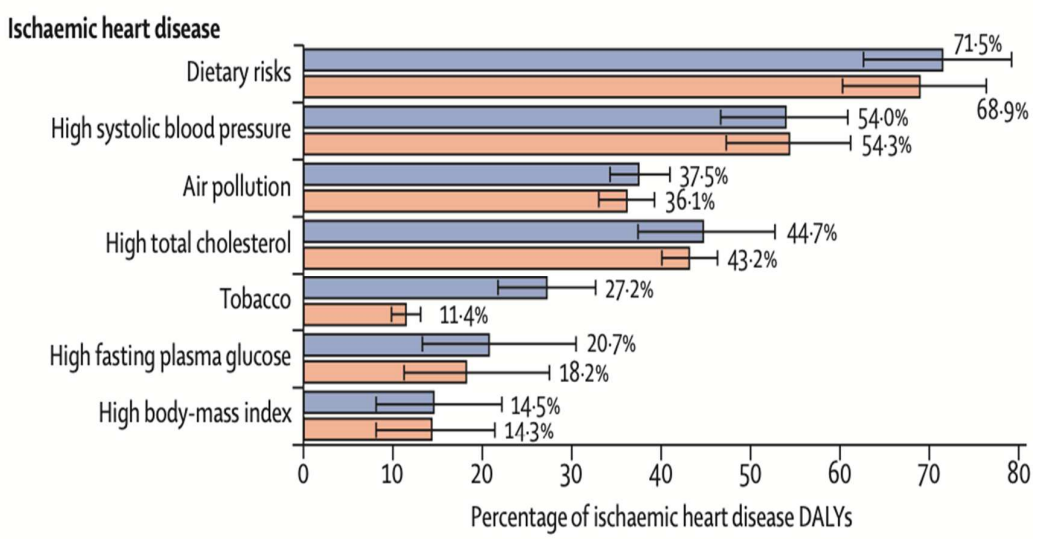

Figure 5. Percentage of key risk factors that contribute to ischemic heart disease

Mortality due to coronary artery disease is 20-50% higher in Asian population compared to worldwide statistics[5]. In India, immediate action is needed to reduce the number of premature deaths from cardiovascular illnesses, particularly among the economically productive age group.

Correlation of Diabetes with Coronary Artery Disease

The primary reason for mortality and morbidity among diabetic individuals is cardiovascular disease[6]. Deaths attributable to cardiovascular disease in the United States are 1.7 times higher in diabetic adults, mostly secondary to myocardial

infarction and stroke[8]. Thus, the main objective of treating diabetic mellitus should include modification of cardiovascular risk.

Most common risk factors linking cardiovascular diseases with diabetes mellitus include obesity, hypertension, and dyslipidemia. Further research has indicated that the development of cardiovascular disease in diabetics is associated with elevated oxidative stress, endothelial dysfunction, increased coagulability and autonomic neuropathy[6].

Insulin resistance in diabetes and obese individuals causes increase in cytokine release by adipose tissues including tumor necrosis factor-α (TNFα), interleukin (IL)-1, IL-6, leptin, resistin MCP-1, PAI-1, angiotensin and fibrinogen[7]. Increased expression of these cytokines causes inflammation and lipid build-up in the arteries and veins, which results in endothelial dysfunction, myocardial infarction, along with cardiomyopathies. Increased IL-1 can also cause destabilization of atheromatous plaques and resultant myocardial infarction[8].

C-reactive protein (CRP) in diabetics increases oxidized LDL uptake in coronary vessels which may cause the malfunction of endothelium and development of atherosclerotic plaques[8]. Decreased adiponectin also contributes to endothelial dysfunction and reduced LDL oxidation[7].

According to current guidelines, diabetic individuals who are overweight or obese should lose 5% of their body weight during a 4-year period. This is associated with increased high-density lipoproteins, decreased TG, and reduction in recently given drugs to decrease cholesterol in people with diabetes[8]. By enhancing glycemic control, blood pressure, and cholesterol profile, bariatric surgery, weight loss drugs, and intensive lifestyle adjustment helped diabetic individuals lose weight and strengthen their cardiovascular risk profile[24].

The SCOUT trial demonstrated that minimal loss of weight had the potential to improve 5-year cardiovascular mortality rates in diabetic individuals versus the Look AHEAD trial that did not show any effect of weight loss on cardiovascular mortality, MI, stroke or angina[8].

Hypertension is another major risk factor in diabetes mellitus and cardiovascular diseases. Diabetic nephropathy can progress to nephrotic syndrome, which comprises of proteinuria, hyperlipidemia and hypercoagulable state resulting from loss of AT3. This result in cardiovascular disease in patients with diabetic kidney disease[8].

Guidelines recommend initiating pharmacological treatment if systolic BP >140mmHg and diastolic BP >90mmHg for diabetic adults. Many trials including ALLHAT trial, discovered that there was no discernible change in the risk of coronary artery disease, myocardial infarction, or mortality in diabetes patients related to the initial antihypertensive medication therapy[25].

The UKPDS 38 trial compared significance of tight BP control (<150/85mmHg) with normal control of BP (<180/105mmHg) on macro and microvascular complications in diabetes mellitus. There was a 34% decrease in macrovascular disorders, such as myocardial infarction, and a 37% decrease in microvascular diseases in the group under stricter control of diabetes[26].

Insulin resistant adipose cells cause increased free fatty acid release. These free fatty acids encourage the synthesis of triglycerides, which increases apolipoprotein B and very low-density lipoprotein (VLDL) cholesterol. Both these factors have been linked to increase cardiovascular diseases. Hyperglycemia also increases glycosylation and oxidation of lipoproteins, decreases vascular compliance, and facilitates atherosclerosis[8].

Diabetics less than 40 years old with clinical evidence of atherosclerotic coronary vascular disease or low-density lipoprotein (LDL) more than 189mg/dL, are recommended to consume high intensity statins. 40-year-old individuals with a 10-year ASCVD risk more than 7.5% are advised to take high-dose statins and individuals with a risk less than 7.5% are treated with moderate-dose statins[27].

Lipid lowering agents other than statins, such as fenofibrates, have not shown similar efficacy in reducing cardiovascular events. Agents to increase high-density lipoproteins may provide minimal benefit[28].

Diabetes mellitus increases the risk of myocardial infarction in patients, and it is an important contributor to the incidence of cardiovascular events[8]. Patients with diabetes have increased risk of morbidity, mortality, and re-infarction than people without the disease; the one-year mortality rate is around 50%[29].

Diabetes mellitus is associated with increased coagulability, which could be a mechanism for increased incidence of myocardial infarction. Diabetes mellitus results in increased expression of glycoprotein 2B/3A receptors and von Willebrand factor (vWF), which cause platelet activation. There is also elevated expression of plasminogen activator inhibitor type-1, which increases thrombus formation, decreases fibrinolysis, and accelerates plaque formation[8].

Diabetic nephropathy patients have low levels of circulating anti-coagulants factors like protein-C and antithrombin-3, attributed to proteinuria[30].

Silent ischemia is commonly observed in patients with diabetes (10-20%) than non-diabetics (1-4%). This has been observed by comparing angiographic studies, which are more advanced in diabetic patients. The disparity could be explained by development of diabetic neuropathy[8].

In diabetes mellitus most common cause of mortality and death is cardiovascular disease. Effective treatment of Diabetes Mellitus can reduce risk of cardiovascular diseases like myocardial infarction, coronary artery disease and cardiomyopathies also . Poor glycemic management has been associated in numerous studies with inferior clinical outcomes, thus close monitoring and controlling glycemic levels is important. Diabetic patients who manage to achieve tighter glycemic control in early phase of disease, before advancement of other cardiovascular risk factors, may have maximum gain from strict glycemic management[8].

Platelet Dysfunction in Diabetes and its relation to coronary artery disease

Diabetes is considered a prothrombotic state with increased coagulation, endothelial dysfunction, impaired fibrinolysis and platelet hyperactivity. Platelets are crucial in development of atherothrombosis in diabetes and atherothrombosis result in morbidity and mortality[9].

Hyperactive platelets with higher adhesiveness, aggregation, and thrombus formation are a consequence of diabetes[31]. Platelets activity is enhanced early in diabetes and may precede development of cardiovascular diseases[9]. Figure 6 illustrates all mechanisms which lead to platelets dysfunction in diabetes patients.

MECHANISMS CONTRIBUTING TO PLATELET DYSFUNCTION IN PATIENTS WITH DM

Hyperglycaemia	Deficient Insulin Action	Associated Metabolic Conditions	Other Cellular Abnormalities	
			Platelet	Endothelial Dysfunction
Increased P-select in expression	Impaired response to NO and PGI_2	Obesity	Increased platelets turnover	Increased production of TF
Osmotic effect	IRS-dependent factors: - increased intracellular Ca^{++} - degranulation	Dyslipidaemia	Increased intracellular Ca^{++}	Decreased NO and PGI_2 production
Activation of PKC		Inflammation	Upregulation of $P2Y_{12}$ signalling	
Decreased membrane fluidity by glycation of surface protein			Oxidative stress	
			Increased	
			P-selectin and GP expression	

Figure 6. Mechanisms of platelet dysfunction in diabetes mellitus

In Diabetes mechanisms of platelet dysfunction which results in the formation of 'diabetic platelet' are categorized as: a) deranged blood sugar, b) insulin resistance and deficiency, c) associated metabolic conditions, and d) other cellular abnormalities[32].

Hyperglycemia increases platelet reactivity by causing non enzymatic glycation of proteins on surface of platelets, increasing platelet activity[31]. It also results in activation of platelet GP IIb/IIIa receptor and expression of P-selectin, which binds von Willebrand factor. This causes increased platelet-fibrin interaction in diabetics[32]. Hyperglycemia also activates protein kinase C, which plays an important role in platelet activation[31]. Low-density lipoproteins (LDL) are glycated, which increases the production of nitric oxide (NO) and intracellular calcium[33].

Overall, hyperglycemia increases procoagulant factors and inhibits fibrinolysis by elevating plasminogen activator inhibitor factor[32]. Figure 7 demonstrates the impact of hyperglycemia on platelets.

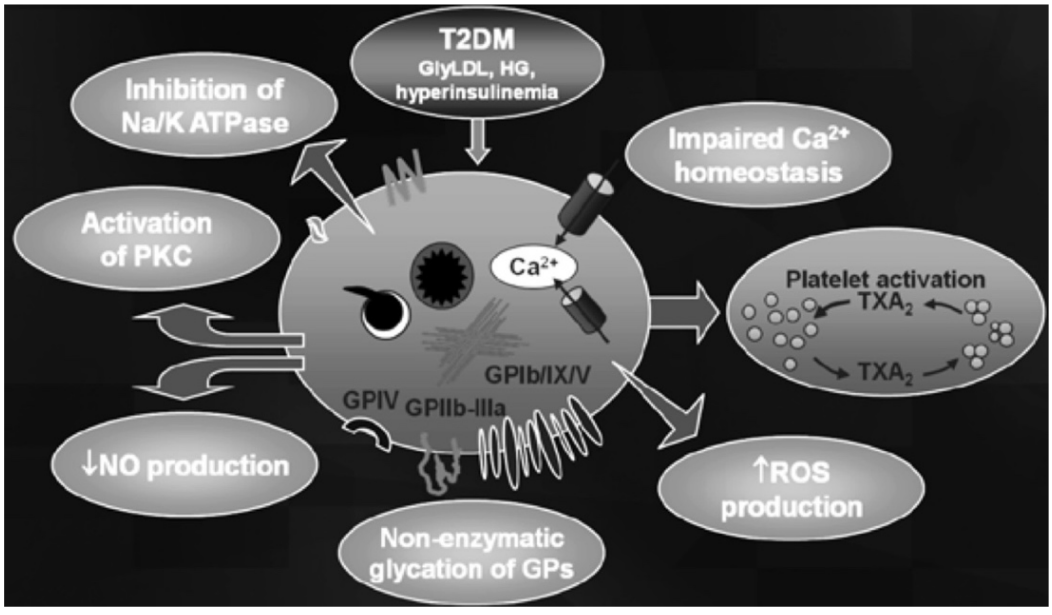

Figure 7. Impact of hyperglycemia on platelets

Insulin deficiency and resistance causes increase Insulin Receptors (IRs) and Insulin-like Growth Factor-1 (IGF-1) on platelets. IGF-1 stimulation results in dose-dependent phosphorylation of IGF receptors , Insulin Receptor Substrate-1 (IRS-1) and IRS-2. Binding of these receptors with p85 subunit of phosphoinositide-3 kinase (PI3K) results phosphorylation of protein kinase B. This causes modulation of platelet reactivity[32].

Insulin resistance also leads to increased intracellular calcium levels, which augments platelet degranulation and aggregation[34]. Nitric oxide (NO) and prostacyclin (PGI2) are released by the endothelium and delay platelet activation. This response is impaired in insulin resistance and leads to increased platelet reactivity[35].

Obesity is commonly seen in diabetic individuals. Insulin resistance, which impacts platelet function, is linked to obesity. Persons with high body mass index also have altered response to anti platelet drugs, mainly clopidogrel[36].

Dyslipidemia including low High-density lipoprotein (HDL) cholesterol and elevated triglycerides is common in diabetes mellitus. Hypertriglyceridemia enhances platelet activation[10]. Triglyceride predominant, Very-low-density Lipoprotein (VLDL) particles alter fibrinolysis and increase risk of atherothrombosis. There is a high content of apoE on VLDL which contacts platelet LDL receptors[32].

Low HDL in diabetes is secondary to endothelial dysfunction and it increases atherothrombotic risk. Intravenous reconstituted HDL raises level of nitric oxide (NO) which rapidly controls the endothelial function in hypercholesterolemia[37].

Diabetes is also associated with increased reactive oxygen (ROS) and nitrogen (RNS) species, which augment platelet activation[32]. This increases advanced glycation end products (AGEs) which leads to atherosclerotic complications[38]. High glucose triggers neutrophil release of S100 calcium binding protein, which binds AGE

receptors on Kupffer cells in liver, increasing Thrombopoietin (TPO) production. TPO causes megakaryocyte proliferation and thrombocytosis[39].

Local platelet activation promotes acute thrombus formation and is essential in the pathogenesis of acute coronary syndrome (ASC). Platelet alpha granules activation leads to circulating leukocyte adherence via P-selectin binding to P-selectin glycoprotein ligand-1 (PSGL-1) receptor. Increased platelet leukocyte complex is seen in acute coronary syndromes and chronic stable angina. Monocyte-platelet and neutrophil-platelet aggregates have been demonstrated in coronary sinus of patients who undergo coronary angiography[40].

Platelets play a pivotal part in the pathophysiology of coronary artery disease. They result in endothelial dysfunction, development of atherosclerotic lesions and thrombotic complications[12]. Diabetes is associated with accelerated platelet turnover, with high reticulated platelets (RP). Due to their increased sensitivity and size, these platelets become hyperactive and fail to respond well to antiplatelet medications like clopidogrel and aspirin[11].

Immature Platelet Fraction (IPF) and Mean Platelet Volume (MPV)

IPF is a parameter which assesses the turnover of circulating platelets[12]. These immature platelets are released from the bone marrow and mature in the peripheral blood in approximately 24 hours. This time lag can be an indirect marker of bone marrow activity[41].

Immature or reticulated platelets contain increased ribonucleic acid (RNA), which are more reactive than mature platelets and contribute significantly to thrombus formation[14]. Bone marrow releases platelet fraction which has elevated aggregating potential because of increased size and protein synthesis capability[13]. Larger

aggregating surface for adhesion molecules and glycoprotein receptors, increased thromboxane A2 and pro thrombotic mediators release are seen in immature platelets[42]. Reticulated platelets are also a predictor of inadequate response to antiplatelet therapy[14].

MPV is a measurement of platelet size and reactivity. Studies suggest that increased mean platelet volume in myocardial infarction is connected to unfavorable clinical results, poor angiographic reperfusion, increased rate of reinfarction and more residual thrombosis post fibrinolysis[13].

Immature platelet fraction (IPF) and mean platelet volume (MPV) can be designed as a screening tool for coronary artery disease. A positive correlation has been seen with high IPF and MPV values and left anterior descending artery (LAD) involvement and triple vessel disease[13]. Previous studies have linked levels of reticulated platelets with platelet reactivity and cardiovascular disorders[42].

Direct assessment of immature platelet fraction is an easy and inexpensive method to evaluate the number of reticulated platelets in blood[14]. In identifying patients with risk of coronary artery disease hematological markers such as IPV and MPC can be added in stratification scores as they rise early[13].

Diabetes mellitus has been linked to accelerated platelet turnover and thus show a blunted response to antiplatelet therapy[15]. It is associated with systemic chronic inflammation, increased hepatic thrombopoietin (TPO) production by interleukin (IL) 6 and acute phase response, thus increasing immature platelet production[42]. It is an important contributor to cardiovascular diseases development. IPF is elevated in diabetic patients and shows correlation with failure of glycemic control and presence of cardiovascular diseases in this population[15].

RESULTS

A total of 130 consecutive patients undergoing coronary angiography, who are either newly diagnosed or known cases of diabetes mellitus, with abnormal glucose parameters or on antidiabetic medications, who visited either the medicine or cardiology department of Dr. Prabhakar Kore Hospital and Research centre, Belagavi, from January 2023 to December 2023, fulfilling the selection criteria, were enrolled in the study. Informed consent was taken from all patients.

Age/Gender	**Mean ± SD ‖ Median (IQR) ‖ Min-Max OR N (%)**
Age (Years)	61.50 ± 8.80 ‖ 60.50 (55.25-67.00) ‖ 41.00 - 85.00
Age	
41-50 Years	16 (12.3%)
51-60 Years	49 (37.7%)
61-70 Years	46 (35.4%)
71-80 Years	16 (12.3%)
81-90 Years	3 (2.3%)
Gender	
Male	87 (66.9%)
Female	43 (33.1%)

Table 1 showed the mean Age (Years) was 61.50 ± 8.80.

16 (12.3%) of the participants had Age: 41-50 Years. 49 (37.7%) of the participants had Age: 51-60 Years. 46 (35.4%) of the participants had Age: 61-70 Years. 16 (12.3%) of the participants had Age: 71-80 Years. 3 (2.3%) of the participants had Age: 81-90 Years.

87 (66.9%) of the participants had Gender: Male. 43 (33.1%) of the participants had Gender: Female.

Table 1- Age and Gender distribution

The most common chief complaint was chest pain, seen in 101 (77.7%) patients followed by dyspnea on exertion in 66 (50.8%) patients and sweating in 13 (10%) patients. Least common symptoms seen included nausea, vomiting, orthopnea and swelling of lower limbs as depicted in table 2.

Chief Complaints	**Yes**
Chest Pain	101 (77.7%)
Dyspnoea On Exertion	66 (50.8%)
Sweating	13 (10.0%)
Palpitations	9 (6.9%)
Retrosternal Burning	4 (3.1%)
Giddiness	3 (2.3%)
PND	2 (1.5%)
Dyspnoea At Rest	2 (1.5%)
Nausea	1 (0.8%)
Limb Claudication	1 (0.8%)
Orthopnea	1 (0.8%)
Swelling Of Legs	1 (0.8%)
Vomiting	1 (0.8%)
Weakness Of Left Upper and Lower Limb	1 (0.8%)

Table 2 – Frequency of chief complaints

Previous known comorbidities were present in 119 (91.5%) of the total study population. Of most significance is history of diabetes mellitus in 110 (84.6%) and rest 20 (15.4%) were newly diagnosed diabetes patients.

Figure 1 shows the percentage of the study population with other known comorbidities, including hypertension 80 (61.5%), ischemic heart disease (IHD) in 15 (11.5%) and hypothyroidism in 7 (5.4%).

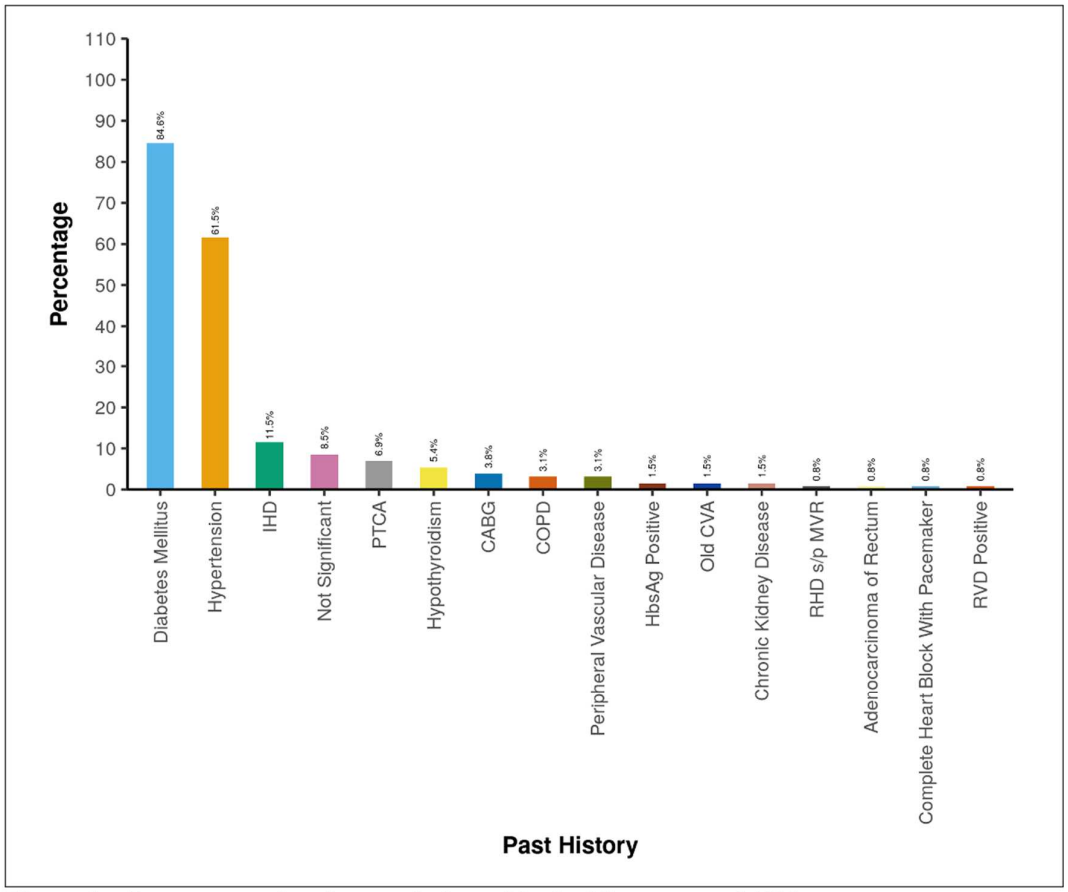

Figure 1 – Percentage of study population with significant comorbidities.
IHD: ischemic heart disease, PTCA: percutaneous transluminal coronary angiography, CABG: coronary artery bypass grafting, COPD: chronic obstructive pulmonary disease, CVA: cerebrovascular accident, RHD: rheumatic heart disease, RVD: retroviral disease

Prior medication history was observed in 119 (91.5%) patients. Common medications included antidiabetics in 107 (82.3%) persons, antihypertensives in 82 (63.1%) persons, lipid lowering agents in 44 (33.8%) persons, antiplatelets in 43 (33.1%) persons, beta blockers in 12 (9.2%) persons, thyroid supplements in 7 (5.4%) persons, inhalers including beta agonists, muscarinic agonists and corticosteroids in 4 (3.1%) persons and antiretroviral (ART) drug regimen by 1 (0.8%) person. Figure 2 demonstrates the distribution of medication history.

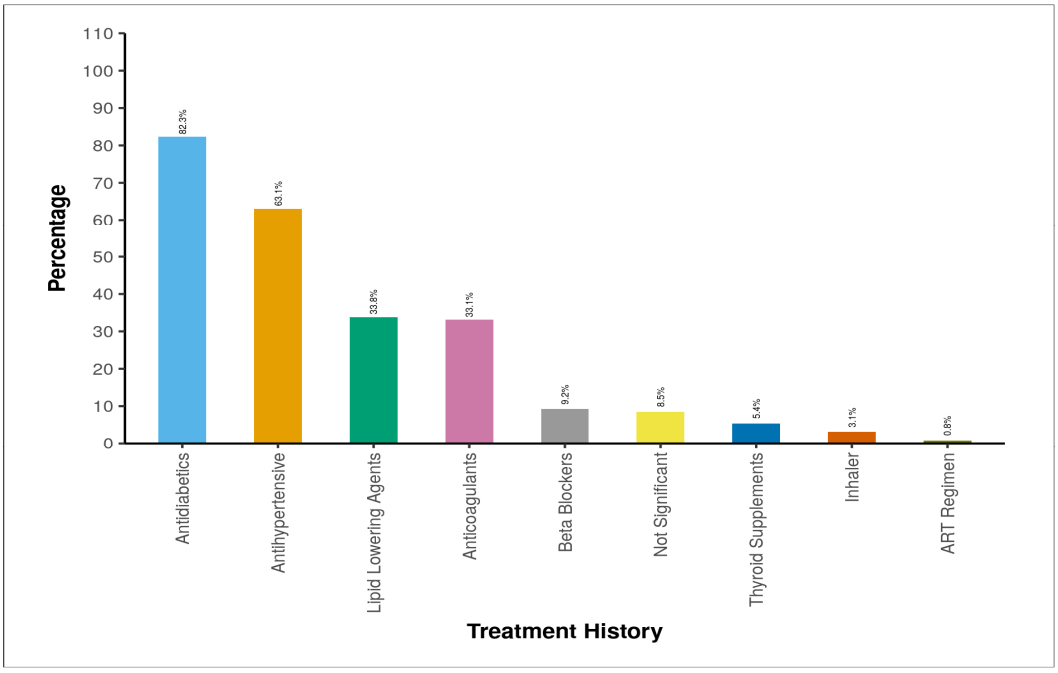

Figure 2 – Distribution of medication/treatment history

Table 3 illustrates a summary of all laboratory parameters of the patients in the study. The mean HbA1C (%) was 8.39 ± 2.19. The mean Fasting Sugars (mg/dL) was 146.95 ± 65.76. The mean Random Sugars (mg/dL) was 202.88 ± 86.97. The mean IPF (%) was 4.44 ± 0.68. The mean Mean Platelet Volume was 10.46 ± 1.22. The mean Total Cholesterol (mg/dL) was 168.89 ± 64.57. The mean LDL (mg/dL) was 112.85 ± 48.03. The mean HDL (mg/dL) was 34.49 ± 8.58. The mean Triglycerides (mg/dL) was 202.77 ± 109.68.

Investigations	**Mean ± SD**	**Median (IQR)**	**Min - Max**
HbA1C (%)	8.39 ± 2.19	7.90 (6.70-9.88)	4.7 - 16.2
Fasting Sugars (mg/dL)	146.95 ± 65.76	126.00 (98.00-186.00)	67.0 - 398.0
Random Sugars (mg/dL)	202.88 ± 86.97	185.00 (145.00-231.50)	80.0 - 545.0
IPF (%)	4.44 ± 0.68	4.44 (3.91-5.10)	3.0 - 5.5
Platelet Count (X10/mm^3)	265.72 ± 81.90	264.50 (205.25-302.50)	115.0 - 448.0
Mean Platelet Volume	10.46 ± 1.22	10.30 (9.60-11.00)	8.2 - 14.9
Hemoglobin (g/dL)	12.48 ± 2.10	12.30 (11.03-13.70)	7.4 - 17.2
Leucocyte Count (/mL)	9053.85 ± 3184.51	8750.00 (7200.00-10475.00)	3500.0 - 30200.0
Blood Urea (mg/dL)	26.91 ± 13.01	23.10 (17.72-31.70)	10.4 - 90.4
S. Creatinine (mg/dL)	1.02 ± 0.38	0.94 (0.76-1.19)	0.4 - 2.3
S. Sodium (mEq/L)	136.10 ± 3.69	136.00 (134.00-138.00)	126.0 - 144.0
S. Potassium (mEq/L)	4.20 ± 0.45	4.20 (3.93-4.50)	3.0 - 5.3

Investigations	Mean ± SD	Median (IQR)	Min - Max
Chloride (mEq/L)	101.21 ± 4.20	102.00 (99.00-104.00)	88.0 - 112.0
Total Bilirubin (mg/dL)	0.67 ± 0.38	0.60 (0.40-0.90)	0.1 - 2.1
Direct Bilirubin (mg/dL)	0.36 ± 0.22	0.30 (0.18-0.46)	0.1 - 1.3
SGPT (U/L)	38.55 ± 82.07	24.50 (17.25-33.00)	4.0 - 705.0
SGOT (U/L)	41.60 ± 90.45	24.50 (18.00-33.75)	10.0 - 705.0
ALP (U/L)	86.55 ± 26.66	79.00 (70.25-98.00)	51.0 - 275.0
Total Protein (g/dL)	6.89 ± 0.54	6.90 (6.60-7.20)	5.0 - 8.2
S. Albumin (g/dL)	3.82 ± 0.45	3.80 (3.50-4.20)	2.8 - 4.8
aPTT (s)	1.08 ± 0.19	1.04 (0.97-1.16)	0.7 - 2.1
INR	1.06 ± 0.14	1.03 (0.96-1.12)	0.8 - 1.7
Total Cholesterol (mg/dL)	168.89 ± 64.57	162.00 (110.25-216.50)	73.0 - 321.0
LDL (mg/dL)	112.85 ± 48.03	101.50 (74.00-157.00)	27.0 - 212.0
HDL (mg/dL)	34.49 ± 8.58	32.00 (29.00-38.00)	17.0 - 63.0
Triglycerides (mg/dL)	202.77 ± 109.68	169.00 (121.00-227.00)	57.0 - 543.0

Table 3 – Distribution of laboratory parameters

IPF: immature platelet fraction, LDL: low density lipoprotein, HDL: high density lipoprotein

The mean (SD) of HbA1C (%) was 8.39 (2.19). The median (IQR) of HbA1C (%) was 7.90 (6.7-9.88). The HbA1C (%) ranged from 4.7 - 16.2.

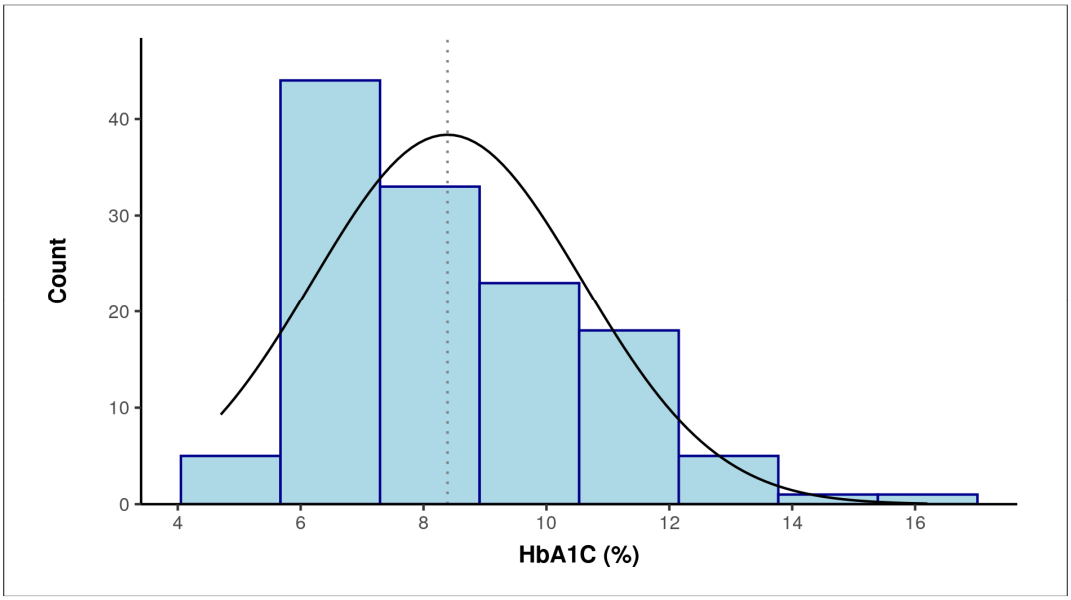

Figure 3 – Distribution of HbA1C in study population

The mean (SD) of IPF (%) was 4.44 (0.68). The median (IQR) of IPF (%) was 4.44 (3.91-5.1). The IPF (%) ranged from 3 - 5.53.

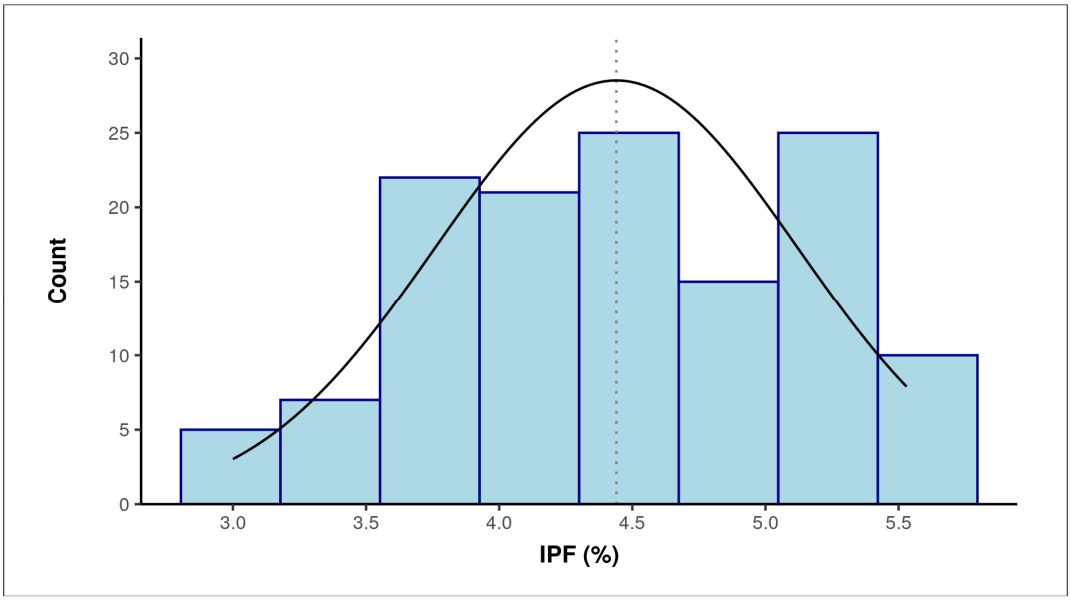

Figure 4 – Distribution if immature platelet fraction (IPF) in study population.

The mean (SD) of Mean Platelet Volume was 10.46 (1.22). The median (IQR) of Mean Platelet Volume was 10.30 (9.6-11). The Mean Platelet Volume ranged from 8.2 - 14.9.

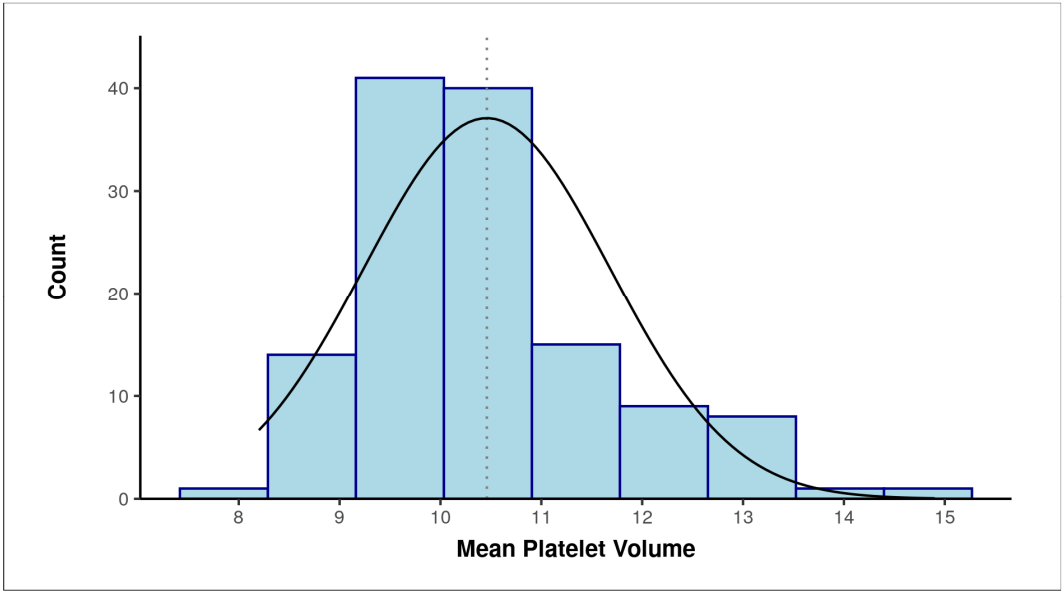

Figure 5 – Distribution of mean platelet volume (MPV) in study population.

Electrocardiography (ECG) findings were recorded in all participants. The most common ECG finding was normal sinus rhythm in 34 (26.2%) persons, followed by strain pattern in 23 (17.7%) persons. Most common myocardial infarction seen was evolved anterior wall in 21 (16.2%) persons followed by anterior wall and evolved inferior wall, both with 16 (12.3%) persons. Least common were complete heart block, inferolateral wall myocardial infarction, poor R wave progression and sinus bradycardia, each in 1 (0.8%) person.

ECG	
Normal Sinus Rhythm	34 (26.2%)
Strain Pattern	23 (17.7%)
Evolved Anterior Wall MI	21 (16.2%)
Anterior Wall MI	16 (12.3%)
Evolved Inferior Wall MI	16 (12.3%)
RBBB	9 (6.9%)
LBBB	6 (4.6%)
Sinus Tachycardia	5 (3.8%)
Heart Block	4 (3.1%)
LVH	4 (3.1%)
Posterior Wall MI	3 (2.3%)
Atrial Fibrillation	2 (1.5%)
Inferior Wall MI	2 (1.5%)
Complete Heart Block	1 (0.8%)
Inferolateral Wall MI	1 (0.8%)
Poor R Wave Progression	1 (0.8%)
Sinus Bradycardia	1 (0.8%)

Table 4 – Distribution of echocardiography (ECG) findings

MI: myocardial infarction, RBBB: right bundle branch block, LBBB: left bundle branch block, LVH: left ventricular hypertrophy

Echocardiography (ECHO) findings were compared in all patients. Normal results were in 52 (40%) persons. Most common anomaly was akinesia of either free wall in 56 (43.1%) persons and hypokinesia was seen in 29 (22.3%) persons. Mild LV dysfunction with ejection fraction between 41 to 60% was seen in 45 (34.6%) persons, moderate LV dysfunction with ejection fraction between 31 to 40% in 27 (20.8%) persons and severe LV dysfunction with ejection fraction less than 30% in 6 (4.6%) persons.

ECHO	
Akinesia	56 (43.1%)
Normal	52 (40.0%)
Mild LV Dysfunction	45 (34.6%)
Hypokinesia	29 (22.3%)
Moderate LV Dysfunction	27 (20.8%)
Valvular Pathology	26 (20.0%)
Severe LV Dysfunction	6 (4.6%)
Asynchronous LV Movement	4 (3.1%)
Asymmetrical Septal Hypertrophy	1 (0.8%)
Atrial Fibrillation	1 (0.8%)

Table 5 – Distribution of echocardiography findings

LV: left ventricular

36.2% of the participants had Coronary Angiography: Single Vessel Disease. 29.2% of the participants had Coronary Angiography: Double Vessel Disease. 34.6% of the participants had Coronary Angiography: Triple Vessel Disease.

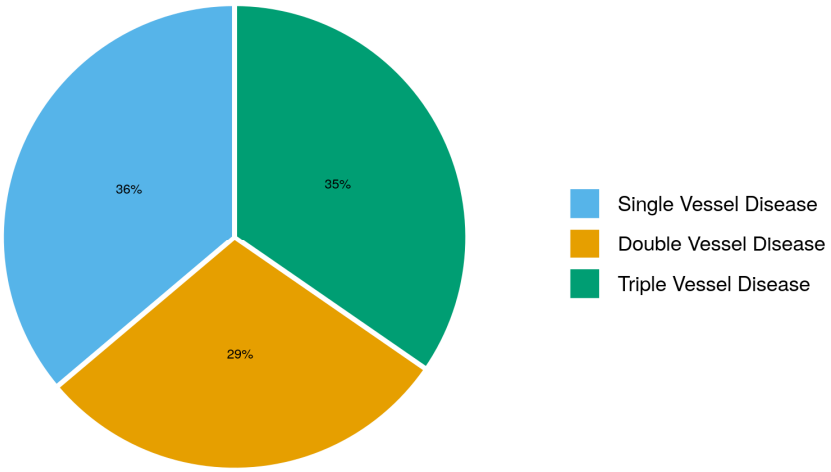

Figure 6 – Pie graph showing distribution of coronary angiographic findings

Association between HbA1C and mean platelet volume (MPV)

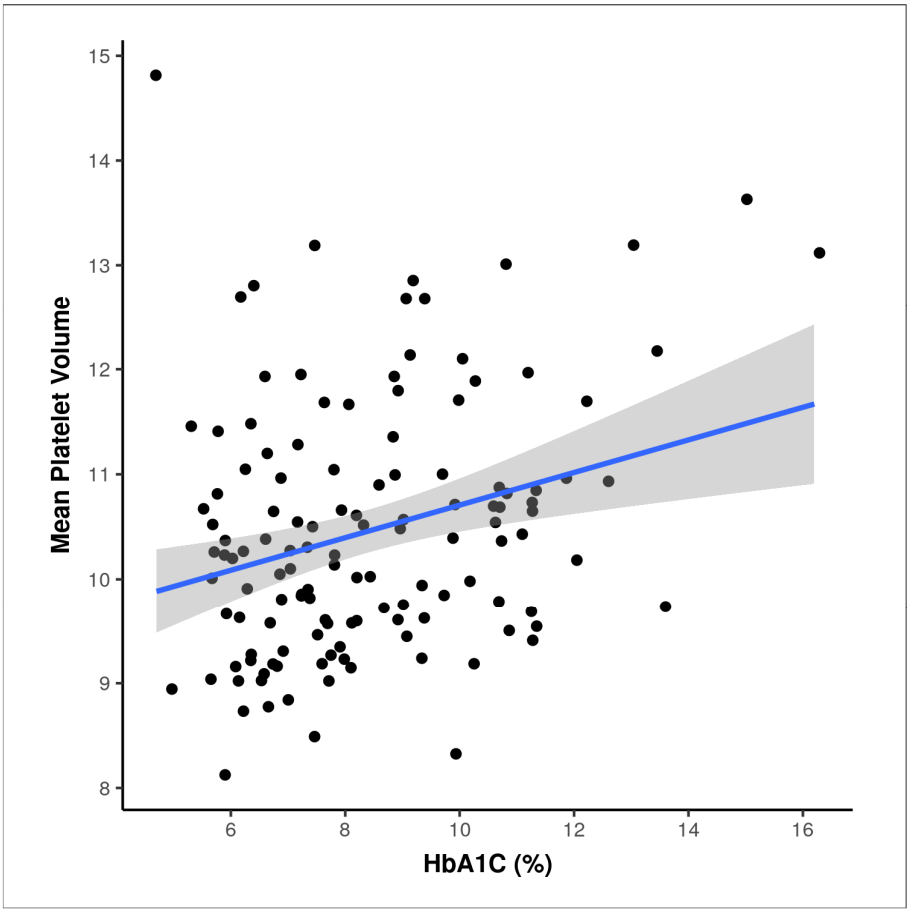

Figure 7 - Scatterplot depicting correlation between HbA1C and MPV

Correlation	Spearman Correlation Coefficient	P Value
HbA1C (%) vs Mean Platelet Volume	0.2	0.006

Table 6 – Association of HbA1C and MPV

The above scatterplot depicts the correlation between HbA1C (%) and Mean Platelet Volume. Individual points represent individual cases. The overall trend of the correlation between the two variables is shown by the blue trendline. The trendline's 95% confidence interval is shown by the shaded grey area.

Due to the non-normal distribution of at least one of the variables, non-parametric tests (Spearman Correlation) were employed to investigate the correlation between the two.

There was a positive correlation between HbA1C (%) and Mean Platelet Volume, and this correlation was statistically significant (rho = 0.24, p = 0.006).

The Mean Platelet Volume rises by 0.16 units for every unit increase in HbA1C (%).

On the other hand, the HbA1C (%) increases by 0.50 units for every unit increase in Mean Platelet Volume

Association between fasting sugars and mean platelet volume (MPV)

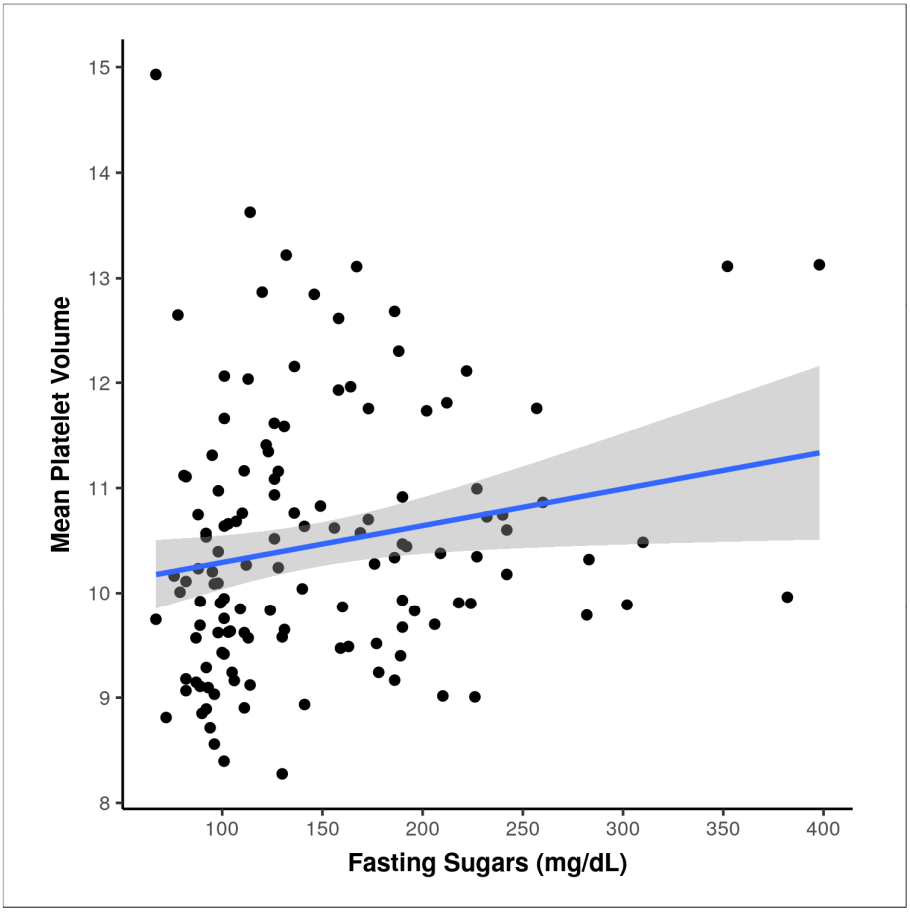

Figure 8 - Scatterplot depicting correlation between fasting sugars and MPV

Correlation	Spearman Correlation Coefficient	P Value
Fasting Sugars (mg/dL) vs Mean Platelet Volume	0.2	0.007

Table 7 – Association between fasting sugars and MPV

The scatterplot above depicts the correlation between Fasting Sugars (mg/dL) and Mean Platelet Volume. Individual points represent individual cases. The overall correlation trend between the two variables is shown by the blue trendline. The trendline's 95% confidence interval is visualized as a shaded grey region.

Since at least one of the variables was not normally distributed, non-parametric tests (Spearman Correlation) were employed to investigate the correlation between the two variables.

There was a positive correlation between Fasting Sugars (mg/dL) and Mean Platelet Volume, and this correlation was statistically significant (rho = 0.24, p = 0.007).

There is a 0.00 unit increase in Mean Platelet Volume for every unit increase in Fasting Sugars (mg/dL).

Conversely, the Fasting Sugars (mg/dL) increase by 10.19 units for every unit increase in Mean Platelet Volume.

Association between random sugars and mean platelet volume (MPV)

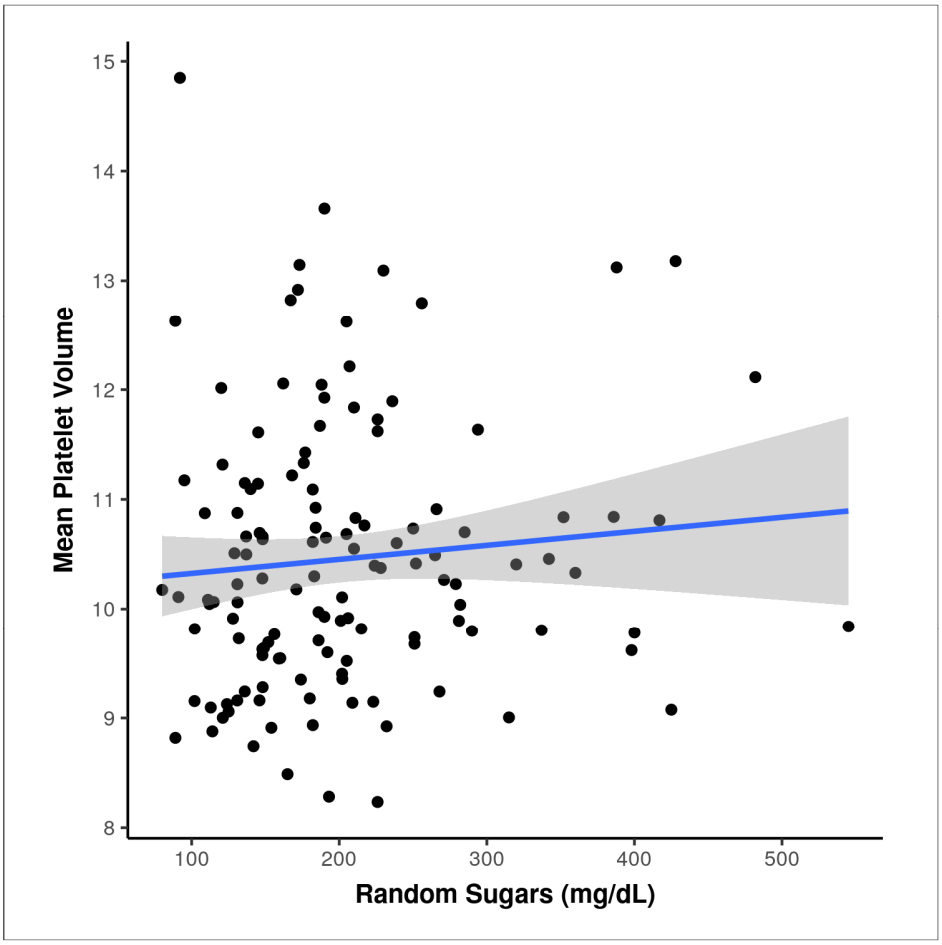

Figure 9 - Scatterplot depicting correlation between random sugars and MPV

Correlation	Spearman Correlation Coefficient	P Value
Random Sugars (mg/dL) vs Mean Platelet Volume	0.1	0.162

Table 8 – Association between random sugars and MPV

The above scatterplot depicts the correlation between Random Sugars (mg/dL) and Mean Platelet Volume. Individual points represent individual cases. The two variables' overall trend of correlation is shown by the blue trendline. The 95% confidence interval for this trendline is shown by the shaded grey area.

The correlation between the two variables was examined using non-parametric tests (Spearman Correlation) because at least one of the variables did not follow a normal distribution.

There was no statistically significant correlation between Random Sugars (mg/dL) and Mean Platelet Volume (rho = 0.12, p = 0.162).

Association between coronary angiography findings and mean platelet volume (MPV)

Mean Platelet Volume	Coronary Angiography			Kruskal Wallis Test	
	Single Vessel Disease	Double Vessel Disease	Triple Vessel Disease	$\chi 2$	p value
Mean (SD)	10.14 (1.19)	10.35 (0.99)	10.88 (1.33)	9.078	0.011
Median (IQR)	9.9 (9.2-10.7)	10.3 (9.7-10.78)	10.6 (9.8-11.9)		
Min - Max	8.5 - 14.9	8.3 - 13.2	8.2 - 13.6		

Table 9 - Association of coronary angiography findings with MPV

The three subgroups of the variable Coronary Angiography did not exhibit a normal distribution of the variable Mean Platelet Volume. To compare groups, non-parametric tests like the Kruskal Wallis Test were employed.

The mean (SD) of Mean Platelet Volume in the Coronary Angiography: Single Vessel Disease group was 10.14 (1.19). The mean (SD) of Mean Platelet Volume in the Coronary Angiography: Double Vessel Disease group was 10.35 (0.99). The mean (SD) of Mean Platelet Volume in the Coronary Angiography: Triple Vessel Disease group was 10.88 (1.33). The median (IQR) of Mean Platelet Volume in the Coronary Angiography: Single Vessel Disease group was 9.9 (9.2-10.7). The median (IQR) of Mean Platelet Volume in the Coronary Angiography: Double Vessel Disease group was 10.3 (9.7-10.78). The median (IQR) of Mean Platelet Volume in the Coronary Angiography: Triple Vessel Disease group was 10.6 (9.8-11.9). The Mean Platelet Volume in the Coronary Angiography: Single Vessel Disease ranged from 8.5 - 14.9.

The Mean Platelet Volume in the Coronary Angiography: Double Vessel Disease ranged from 8.3 - 13.2. The Mean Platelet Volume in the Coronary Angiography: Triple Vessel Disease ranged from 8.2 - 13.6.

There was a significant difference between the 3 groups in terms of Mean Platelet Volume ($\chi 2 = 9.078$, $p = 0.011$), with the median Mean Platelet Volume being highest in the Coronary Angiography: Triple Vessel Disease group.

Strength of Association (Kendall's Tau) = 0.21 (Small Effect Size)

The distribution of Mean Platelet Volume amongst the three groups is shown in the Box-and-Whisker graphic underneath. The median mean platelet volume is represented by the middle horizontal line; the 75th and 25th centiles of mean platelet volume are represented by the upper and lower bounds of the box; and the Tukey limits for mean platelet volume in each group are indicated by the upper and lower extent of the whiskers.

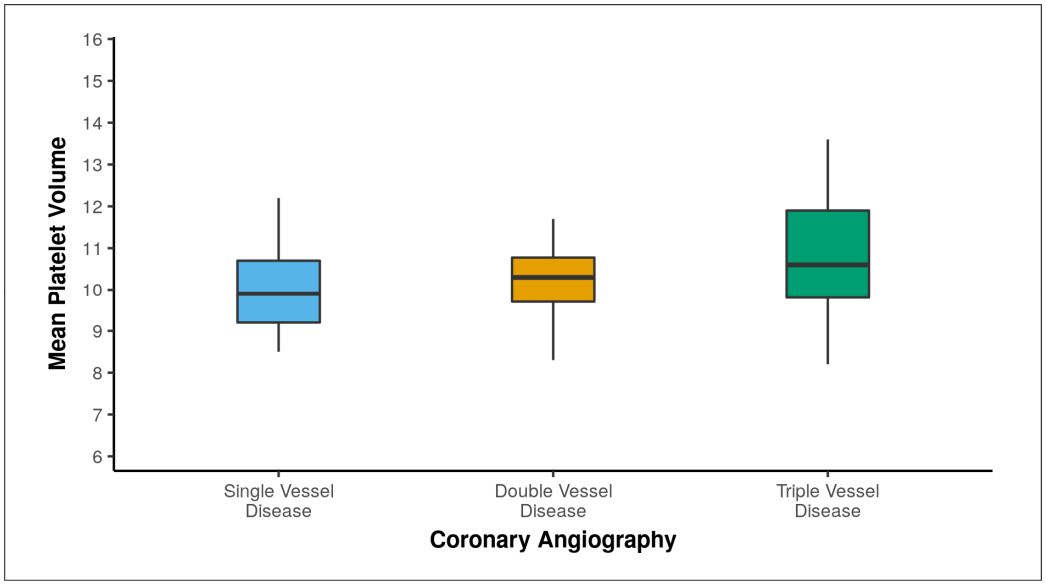

Figure 10 - Box and Whisker plot depicting MPV correlation with coronary angiography findings

The density plot below depicts the distribution of Mean Platelet Volume in the 3 different groups of the variable Coronary Angiography.

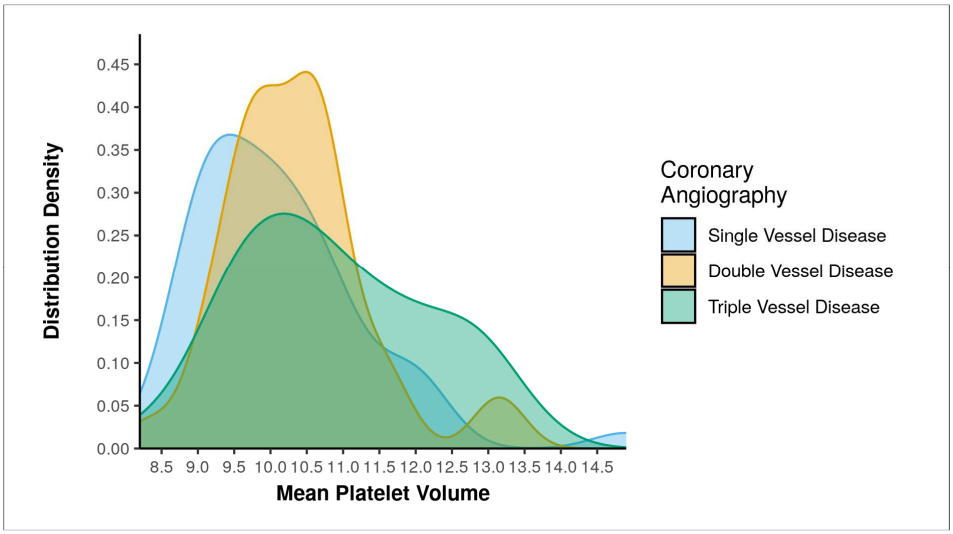

Figure 11 - Distribution density of MPV value in 3 different coronary angiography groups. As severity of coronary occlusion is increasing, the MPV value is larger.

Association between HbA1C and immature platelet fraction (IPF)

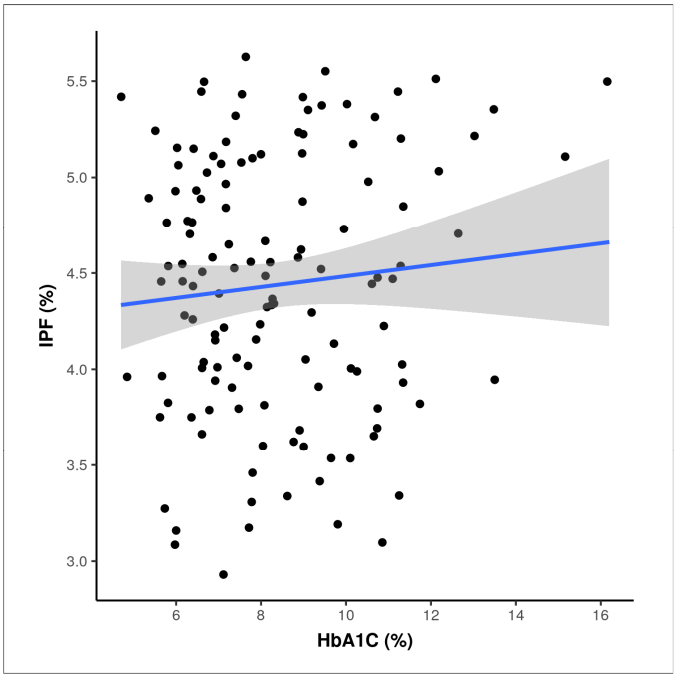

Figure 12 – Scatterplot depicting correlation between HbA1C and IPF

Correlation	Spearman Correlation Coefficient	P Value
HbA1C (%) vs IPF (%)	0.0	0.706

Table 10 – Association of HbA1C and IPF

The scatterplot above depicts the correlation between HbA1C (%) and IPF (%). Individual points represent individual cases. The overall trend of the correlation between the two variables is shown by the blue trendline. The trendline's 95% confidence interval is shown by the shaded grey area.

Non-parametric tests (Spearman Correlation) were used to examine the correlation between the two variables since at least one of the variables was not normally distributed.

There was no statistically significant correlation between HbA1C (%) and IPF (%) (rho = 0.03, p = 0.706).

Association between Fasting sugars and immature platelet fraction (IPF)

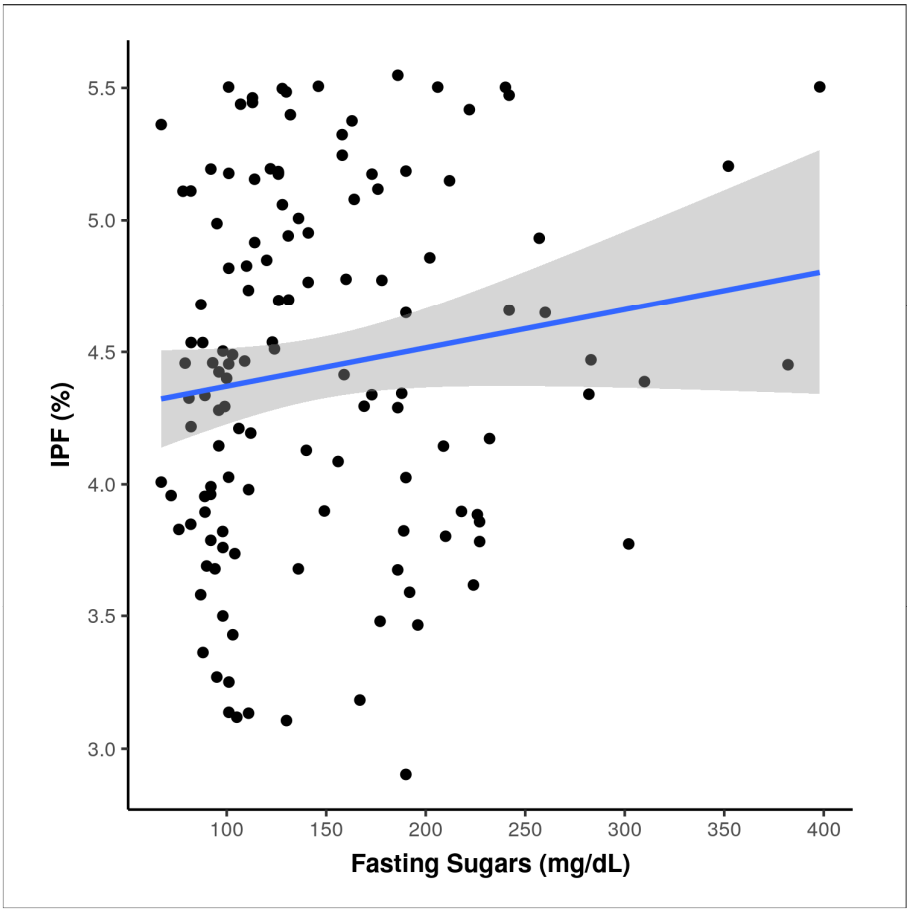

Figure 13 - Scatterplot depicting correlation between fasting sugars and IPF

Correlation	Spearman Correlation Coefficient	P Value
Fasting Sugars (mg/dL) vs IPF (%)	0.2	0.046

Table 11 – Association of fasting sugars and IPF

The scatterplot above, depicts the correlation between Fasting Sugars (mg/dL) and IPF (%). Individual points represent individual cases. The overall trend of the correlation between the two variables is shown by the blue trendline. The trendline's 95% confidence interval is shown by the shaded grey area.

The correlation between the two variables was examined using non-parametric tests (Spearman Correlation) because at least one of the variables did not follow a normal distribution.

There was a positive correlation between Fasting Sugars (mg/dL) and IPF (%), and this correlation was statistically significant (rho = 0.18, p = 0.046).

There is a 0.00 unit rise in the IPF (%) for every unit increase in Fasting Sugars (mg/dL).

On the other hand, the Fasting Sugars (mg/dL) increase by 13.74 units for every unit increase in IPF (%).

Association between random sugars and immature platelet fraction (IPF)

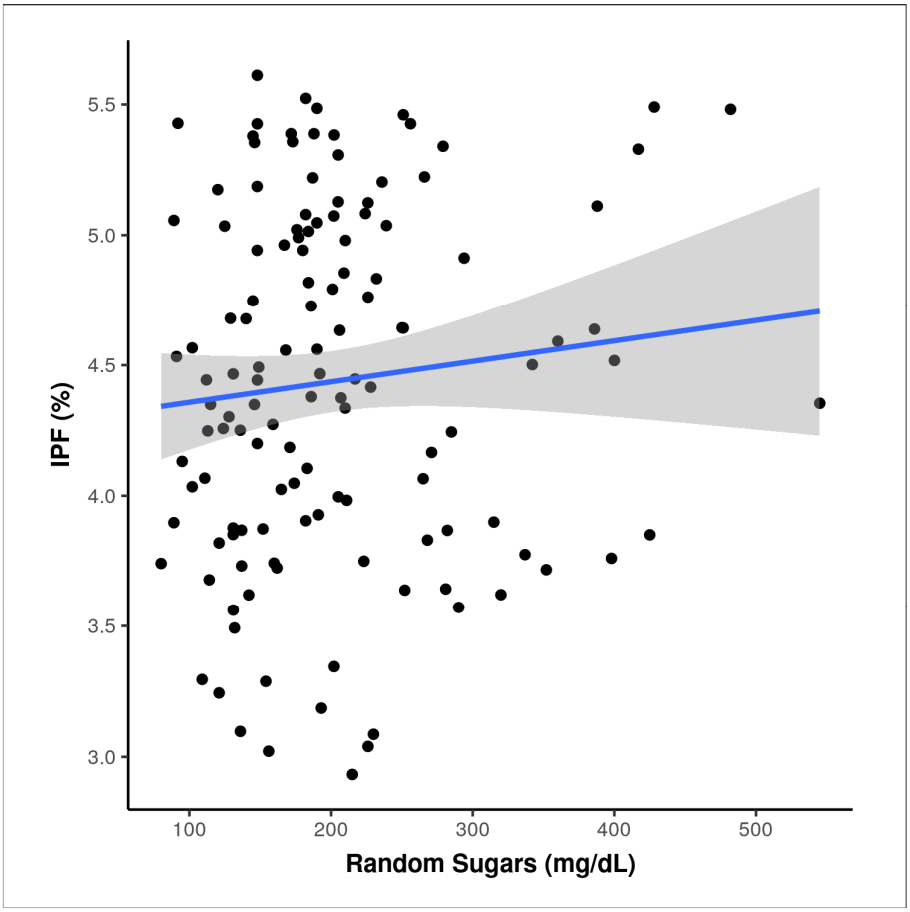

Figure 14 - Scatterplot depicting correlation between random sugars and IPF

Correlation	Spearman Correlation Coefficient	P Value
Random Sugars (mg/dL) vs IPF (%)	0.1	0.233

Table 12 - Association of random sugars and IPF

The correlation between Random Sugars (mg/dL) and IPF (%) is seen in the scatterplot above. Individual points represent individual cases. The overall trend of the correlation between the two variables is shown by the blue trendline. The trendline's 95% confidence interval is shown by the shaded grey area.

The correlation between the two variables was examined using non-parametric tests (Spearman Correlation) because at least one of the variables did not follow a normal distribution.

There was no statistically significant correlation between Random Sugars (mg/dL) and IPF (%) (rho = 0.11, p = 0.233).

Association of Coronary Angiography findings with immature platelet fraction (IPF)

IPF (%)	Coronary Angiography			One-Way ANOVA	
	Single Vessel Disease	Double Vessel Disease	Triple Vessel Disease	F	p value
Mean (SD)	4.30 (0.64)	4.53 (0.65)	4.51 (0.72)	1.458	0.237
Median (IQR)	4.25 (3.89-4.72)	4.5 (3.97-5.12)	4.65 (3.81-5.12)		
Min - Max	3 - 5.53	3.12 - 5.43	3.1 - 5.47		

Table 13 – Association of coronary angiography findings with IPF

In each of the three subgroups of the variable Coronary Angiography, the variable IPF (%) had a normal distribution. To compare groups, parametric tests (One-Way ANOVA) were employed.

The mean (SD) of IPF (%) in the Coronary Angiography: Single Vessel Disease group was 4.30 (0.64). The mean (SD) of IPF (%) in the Coronary Angiography: Double Vessel Disease group was 4.53 (0.65). The mean (SD) of IPF (%) in the Coronary Angiography: Triple Vessel Disease group was 4.51 (0.72). The median (IQR) of IPF (%) in the Coronary Angiography: Single Vessel Disease group was 4.25 (3.89-4.72). The median (IQR) of IPF (%) in the Coronary Angiography: Double Vessel Disease group was 4.5 (3.97-5.12). The median (IQR) of IPF (%) in the Coronary Angiography: Triple Vessel Disease group was 4.65 (3.81-5.12). The IPF (%) in the Coronary Angiography: Single Vessel Disease ranged from 3 - 5.53. The IPF (%) in the Coronary Angiography: Double Vessel Disease ranged from 3.12 -

5.43. The IPF (%) in the Coronary Angiography: Triple Vessel Disease ranged from 3.1 - 5.47.

There was no significant difference between the groups in terms of IPF (%) ($F = 1.458$, $p = 0.237$).

Strength of Association (Kendall's Tau) = 0.1 (Small Effect Size)

Below is a Box-and-Whisker figure showing the IPF (%) distribution among the three groups. The median IPF (%) is represented by the main horizontal line; the Tukey limits for IPF (%) in each group are represented by the upper and lower extend of the whiskers; and the upper and lower bounds of the box, respectively, by the 75th and 25th centiles of IPF (%).

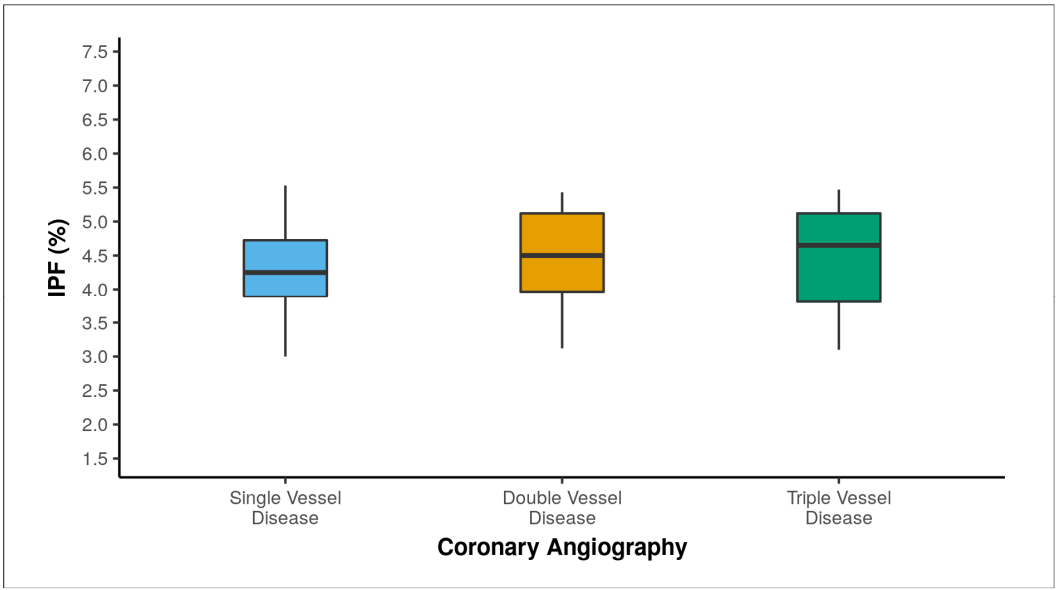

Figure 15 – Box and Whisker plot depicting IPF correlation with coronary angiography findings

The distribution of IPF (%) among the three distinct groups of the variable Coronary Angiography is shown in the density map below.

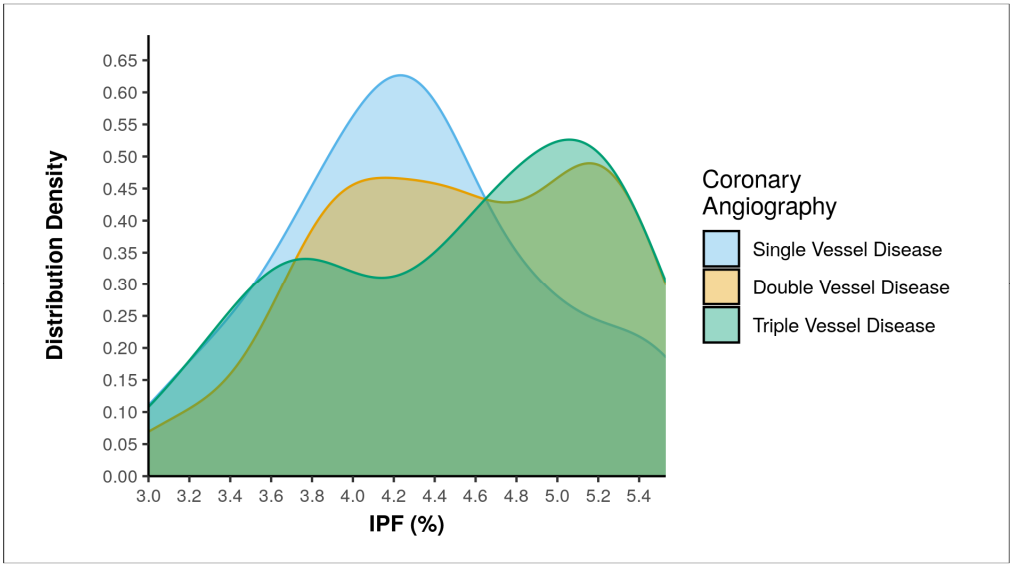

Figure 16 – Distribution density of IPF value in 3 different coronary angiography groups. As severity of coronary occlusion is increasing, the IPF value is larger.

Association of immature platelet fraction (IPF) with mean platelet volume (MPV)

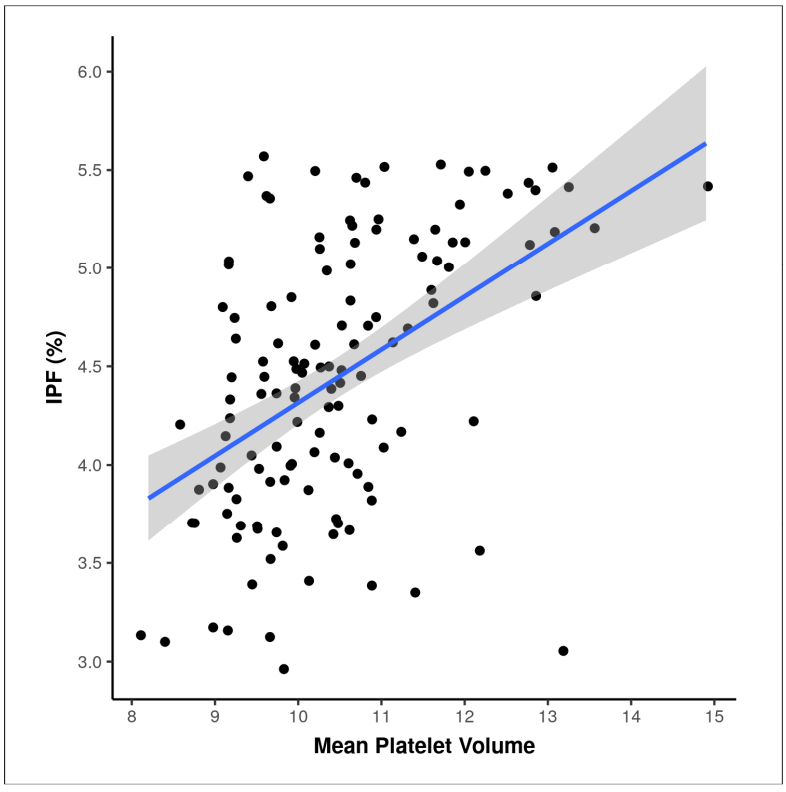

Figure 17 – Scatterplot depicting correlation between IPF and MPV

Correlation	Spearman Correlation Coefficient	P Value
Mean Platelet Volume vs IPF (%)	0.5	<0.001

Table 14 – Association of IPF with MPV

The link between Mean Platelet Volume and IPF (%) is shown in the scatterplot above. Individual points represent individual cases. The overall trend of the correlation between the two variables is shown by the blue trendline. The trendline's 95% confidence interval is shown by the shaded grey area.

The correlation between the two variables was examined using non-parametric tests (Spearman Correlation) because at least one of the variables did not follow a normal distribution.

There was a positive correlation between Mean Platelet Volume and IPF (%), and this correlation was statistically significant (rho = 0.47, p = <0.001).

The IPF (%) rises by 0.27 units for every unit increase in Mean Platelet Volume..

In contrast, the Mean Platelet Volume rises by 0.87 units for every unit increase in IPF (%).

Association between treatment history with antiplatelets and mean platelet volume (MPV)

Mean Platelet Volume	**Treatment History: Antiplatelets**		**Wilcoxon-Mann-Whitney U Test**	
	Yes	**No**	**W**	**p value**
Mean (SD)	10.84 (1.29)	10.27 (1.14)	2351.500	0.017
Median (IQR)	10.7 (9.85-11.7)	10.2 (9.55-10.75)		
Min - Max	8.8 - 13.6	8.2 - 14.9		

Table 15 – Association between antiplatelet history and MPV

The variable Mean Platelet Volume was not regularly distributed in the 2 subgroups of the variable Treatment History: Antiplatelets. To compare groups, non-parametric tests (Wilcoxon-Mann-Whitney U Test) were employed.

The mean (SD) of Mean Platelet Volume in the Treatment History: Antiplatelets: Yes group was 10.84 (1.29). The mean (SD) of Mean Platelet Volume in the Treatment History: Antiplatelets: No group was 10.27 (1.14). The median (IQR) of Mean Platelet Volume in the Treatment History: Antiplatelets: Yes group was 10.7 (9.85-11.7). The median (IQR) of Mean Platelet Volume in the Treatment History: Antiplatelets: No group was 10.2 (9.55-10.75). The Mean Platelet Volume in the Treatment History: Antiplatelets: Yes ranged from 8.8 - 13.6. The Mean Platelet Volume in the Treatment History: Antiplatelets: No ranged from 8.2 - 14.9.

There was a significant distinction noted between the 2 groups in terms of Mean Platelet Volume (W = 2351.500, p = 0.017), with the median Mean Platelet Volume being maximum in the Treatment History: Antiplatelets: Yes group.

Strength of Association (Point-Biserial Correlation) = 0.22 (Small Effect Size)

This box-and-whisker graphic below shows how the mean platelet volume was distributed between the two groups. The Tukey limits for Mean Platelet Volume in each group are represented by the upper and lower extent of the whiskers, the upper and lower bounds of the box indicate the 75th and 25th centiles of Mean Platelet Volume, respectively, and the median is represented by the middle horizontal line.

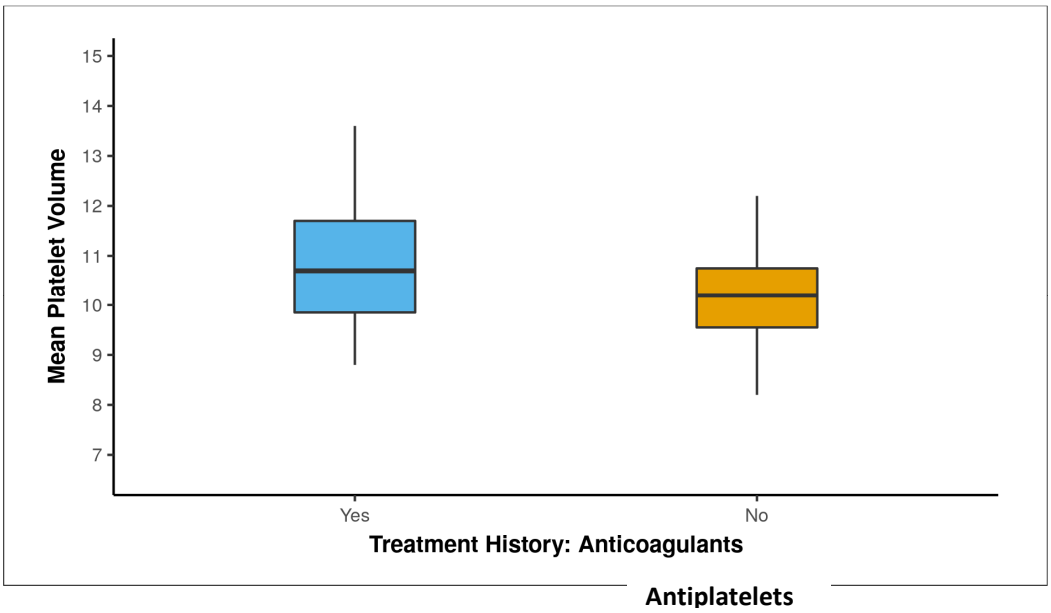

Figure 18 - Box and Whisker plot depicting MPV correlation with treatment history with antiplatelet medication

Association between treatment history with antiplatelets and immature platelet fraction (IPF)

IPF (%)	Treatment History: Antiplatelets		Wilcoxon-Mann-Whitney U Test	
	Yes	No	W	p value
Mean (SD)	4.57 (0.71)	4.38 (0.65)	2162.500	0.149
Median (IQR)	4.47 (3.9-5.26)	4.41 (3.92-4.93)		
Min - Max	3 - 5.47	3.1 - 5.53		

Table 16 – Association between antiplatelet history and IPF

The variable IPF (%) was not normally distributed in the 2 subgroups of the variable Treatment History: Antiplatelets. To compare groups, non-parametric tests (Wilcoxon-Mann-Whitney U Test) were employed.

The mean (SD) of IPF (%) in the Treatment History: Antiplatelets: Yes group was 4.57 (0.71). The mean (SD) of IPF (%) in the Treatment History: Antiplatelets: No group was 4.38 (0.65). The median (IQR) of IPF (%) in the Treatment History: Antiplatelets: Yes group was 4.47 (3.9-5.26). The median (IQR) of IPF (%) in the Treatment History: Antiplatelets: No group was 4.41 (3.92-4.93). The IPF (%) in the Treatment History: Antiplatelets: Yes ranged from 3 - 5.47. The IPF (%) in the Treatment History: Antiplatelets: No ranged from 3.1 - 5.53.

There was no significant difference noted between the groups in terms of IPF (%) ($W = 2162.500$, $p = 0.149$).

Strength of Association (Point-Biserial Correlation) = 0.13 (Small Effect Size)

Association between total cholesterol and immature platelet fraction (IPF)

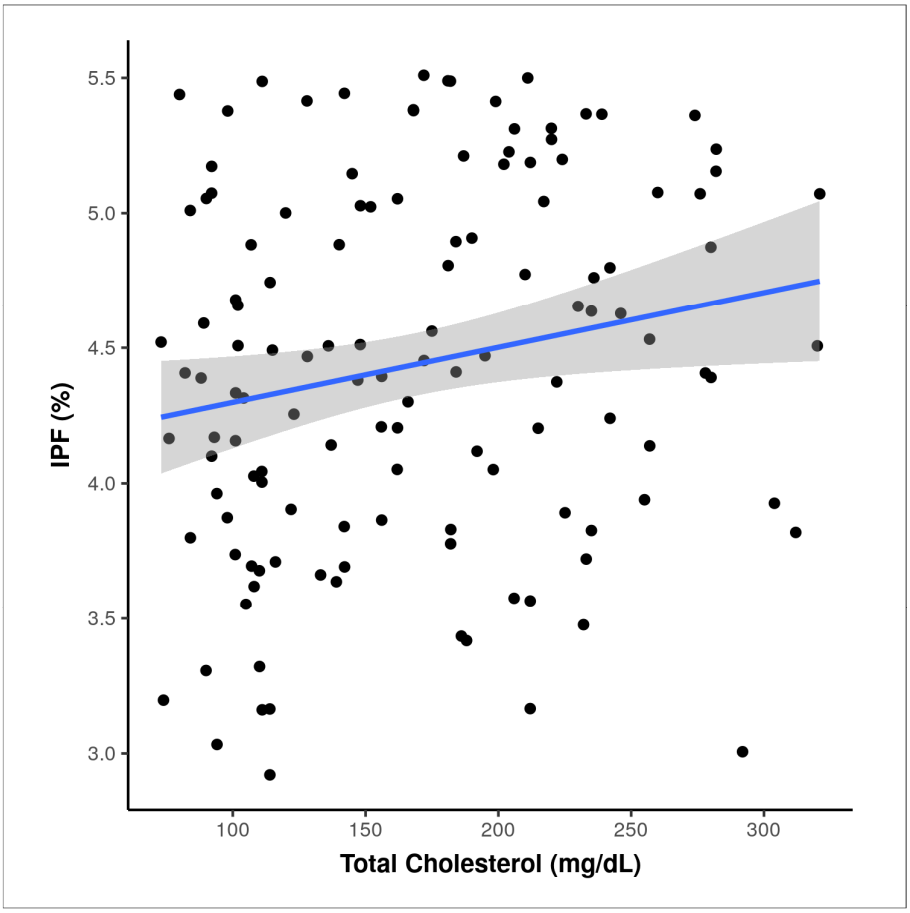

Figure 19 – Scatterplot depicting correlation between total cholesterol and IPF

Correlation	Pearson's Correlation Coefficient	P Value
Total Cholesterol (mg/dL) vs IPF (%)	0.2	0.027

Table 17 – Association between total cholesterol and IPF

The scatterplot above shows the relationship between IPF (%) and total cholesterol (mg/dL). Each point corresponds to a single instance case. The overall trend of the correlation between the two variables is shown by the blue trendline. The trendline's 95% confidence interval is shown by the shaded grey area.

Parametric tests (Pearson's Correlation) were employed to investigate the relationship between the two variables, as both the variables were normally distributed.

There was a positive correlation between Total Cholesterol (mg/dL) and IPF (%), and this was statistically significant ($r = 0.19$, $p = 0.027$).

IPF (%) rises by 0.00 units for every unit increase in total cholesterol (mg/dL).

On the other hand, the total cholesterol (mg/dL) rises by 18.52 units for every unit increase in IPF (%).

MATERIALS AND METHODS

Source of data - Patients in the department of internal medicine and cardiology at Dr. Prabhakar Kore Hospital and Research Centre, Belagavi

Study design – Cross sectional study

Study period - January 2023 to December 2023

Sample size - The prevalence rate-based minimum sample size formula is:

$$n = \frac{z_\alpha^2 P(1-P)}{d^2}$$

where P is the prevalence rate of cardiovascular morbidity among diabetic patients and d is the percentage likely difference in the prevalence.

z_α is linked with the level of significance. For 5% level of the significance $z_\alpha = 1.96$.

Ref:

With P = 32.2% and d = 25% of P = 8.05%, the sample size is 129.

To round off the sample size will be raised to 130.

Sampling technique - This is a cross-sectional study that will include all consecutive patients who meet the inclusion criteria. Descriptive analysis, ANOVA, and the chi-square test will be used in the statistical analysis performed by SPSS.

Inclusion criteria:

- Patients undergoing coronary angiography studies
- New or old diagnosed case of Diabetes Mellitus, with abnormal glucose parameters or on anti-diabetic medication.
- Age >18 years

Exclusion criteria:

- Any patient with platelet count <1 lakh and >4.5 lakhs
- Lactating and pregnant females

Study protocol

Every patient who meets the requirements for inclusion and agrees to take part in the research will be included. The protocol will be explained to the patient and informed consent will be taken.

Patients with abnormal coronary angiography findings will be further evaluated. Random blood sugar, fasting blood sugar and HbA1C will be evaluated.

Immature platelet fraction (IPF) and platelet indices like platelet count, mean platelet volume will be checked. Additional testing will include Hb, total leucocyte count, urea, creatinine, sodium, potassium, chloride, total and direct bilirubin, SGOT, SGPT, ALP, total protein, albumin, aPTT, PT INR, ECG, 2D ECHO and fasting lipid profile.

These values will be compared and correlated to evaluate immature platelet fraction and degree of diabetes mellitus in post coronary angiography patients. Mean platelet values will also be compared with diabetic status and coronary angiography findings.

Statistical Analysis

Given that this is an observational study, the following will be the analysis plan. For the continuous quantitative variables, the mean and standard deviation will be calculated. When comparing data that has been split into two groups based on a particular qualitative attribute, appropriate statistical methods such as the student's unpaired t test will be used to compare the continuous variables. The pre- and post-therapy measures will be compared using the student's paired t-test results.

Discrete variables will be represented by the median. The categorical data will be expressed using percentages, ratios, and rates. Fisher's exact test, the test of proportion, or the Chi-square test will be used to determine the relationship between the outcome, clinical, and demographic factors. Discrete variables will be tested using nonparametric methods.

In addition to the methods mentioned above, appropriate tools such as ANOVA, correlation, regression, etc., will be employed based on the situation. We'll use the appropriate graphs to show the comparison. For every test, a p-value of less than 5% (0.05) will be considered significant.

DISCUSSION

The study was a hospital based descriptive, observational, cross-sectional study conducted in a tertiary care hospital in Belgaum. 130 consenting individuals were enrolled and investigated as per a pre-defined proforma in the study period from January 2023 to December 2023. Data of the study was compiled in a master chart prepared in MS Excel version 16.84. Statistical analysis was performed using SPSS version 21.0 software. In this study, we analysed immature platelet fraction (IPF) and mean platelet volume (MPV) and its relationship with hyperglycemia in diabetic patients post coronary angiography.

Descriptive Parameters

In our study, a total of 130 participants were enrolled, of which 87 (66.9%) were males and 43 (33.1%) were females. This was consistent with the results of a study conducted by Paramita P. et al where males (73.1%) were more commonly affected than females (26.9%)[43]. The most common age bracket was 51 to 60 years old with 49 (37.7%) persons and second was 61 to 70 years old with 46 (35.4%) persons. This was consistent with findings seen in the study by Rauf S et al, where the most common age group affected by acute coronary syndrome was 51 to 60 years (39%)[13].

A study conducted by King-Shier K et al. showed chest pain (89.3%) as the most common clinical finding bringing a patient of myocardial infarction to the hospital, which was consistent with our study, showing frequency of chest pain in 101 (77.7%) persons, followed by dyspnea on exertion in 66 (50.8%) persons and sweating in 13 (10%) persons[44].

Mean Platelet Volume

Mean platelet volume (MPV), a frequently used indicator of platelet size, is currently being researched as a possible indicator of platelet reactivity.

Diabetic individuals have larger platelets and more reactive platelets as reflected by increase in mean platelet volume. The increased reactivity predisposes diabetic individuals to development of microvascular and macrovascular complications including coronary artery disease. Kodiatte, Thomas Alex et al. conducted a study demonstrating increased MPV (p=0.003) in uncontrolled diabetics[46].

In our study, we compared MPV with HbA1C, fasting blood glucose and random blood glucose, to observe association with diabetic status. There was a positive correlation between HbA1C (%) and Mean Platelet Volume, and this was a statistically significant (p = 0.006) correlation. For every 1 unit increase in Mean Platelet Volume, the HbA1C (%) increases by 0.50 units. There was a statistically significant positive correlation between Fasting Sugars (mg/dL) and Mean Platelet Volume (p = 0.007). For every 1 unit increase in Mean Platelet Volume, the Fasting Sugars (mg/dL) increases by 10.19 units. There was no statistically significant association between Random Sugars (mg/dL) and Mean Platelet Volume (p = 0.162).

Mean platelet volume (MPV) was shown to be higher in individuals with acute myocardial infarction and coronary artery occlusion in a study by Chu, S. G. et al.[45]. Another meta-analysis conducted by Sansanayudh N et al. showed patients with coronary artery disease to have higher MPV[47]. In our study, there was a significant association between mean platelet volume and coronary angiography findings (p = 0.011), with the median Mean Platelet Volume being maximum in the Coronary Angiography: Triple Vessel Disease group.

A study conducted by Huang et al. showed patients with acute coronary syndrome having higher MPV, and the study suggested using MPV as an independent biomarker for ACS diagnosis. They also came to the conclusion that a higher MPV was linked to a higher risk of thrombosis[48]. Our study found the mean (SD) of Mean Platelet Volume in the Coronary Angiography: Single Vessel Disease group was 10.14, in the Coronary Angiography: Double Vessel Disease group was 10.35 and in Coronary Angiography: Triple Vessel Disease group was 10.88. Thus a higher MPV value was seen with worsening coronary artery occlusion severity.

Immature Platelet Fraction

Immature platelet fraction (IPF) is a marker of reticulated or immature platelets in the blood steam and reflect platelet turnover. IPF values have a positive correlation with MPV according to multiple meta-analysis. Thus IPF has been proposed as a marker for platelet reactivity in diabetic individuals, where platelet hyperactivity is one proposed mechanism for increased risk of ischemic events[42].

A study conducted by Verdoia, Monica et al. found no correlation between diabetes status or glycaemic control on IPF levels. There was no correlation noted between the coronary angiography findings and IPF levels in diabetic individuals[42]. In another study conducted by Huang et al. they showed that IPF could not be used as an independent marker to assess risk of acute coronary syndrome[48].

In our study, there was no statistically significant correlation between HbA1C (%) and IPF (%) ($p = 0.706$). There was a statistically significant positive correlation between Fasting Sugars (mg/dL) and IPF (%) ($p = 0.046$). For every 1 unit increase in IPF (%), the Fasting Sugars (mg/dL) increases by 13.74 units. There was no statistically significant association between Random Sugars (mg/dL) and IPF (%) ($p = 0.233$).

According to a study conducted by Verdoia, Monica et al. there was no impact of IPF on coronary artery disease or triple vessel disease. Another study conducted by Larsen, Sanne Bøjet et al. showed that increase IPF value is related to low grade inflammation in stable coronary artery disease and related to atherosclerosis progression[49].

The results of the coronary angiography in the single vessel, double vessel, and triple vessel groups did not differ significantly in our investigation, in terms of correlation with IPF (%) ($F = 1.458$, $p = 0.237$). The strength of association was 0.1, indicating small effect size.

Correlation between markers of platelet activity

Platelet volume and immature platelets are related to platelet activity. However, not every platelet with a larger volume is an immature platelet[50].

When platelets are lost quickly, megakaryocytes respond by producing immature platelets, which is why a high IPF count is observed. In acute coronary syndrome, there is endothelial cell injury with atherosclerotic plaque formation, leading to steady platelet consumption. Slow platelet consumption activates polyploid megakaryocytes, which produces mature platelets with increased volume[51]. Thus increase in MPV is seen with normal IPF levels in acute coronary syndrome patients.

MPV reflects 4 month status of platelets in individuals and can predict the onset of acute coronary syndrome in chronic stable underlying disease. Immature platelets have a lifespan of 24 hours in periphery. Sudden increase in IPF is seen with increased megakaryocyte activation, increased turnover and increased platelet consumption in stable atherosclerosis. Thus IPF can be used to correlate with major cardiac adverse events and risk of mortality[48].

A study conducted by Miyazaki, Koji et al. suggested that IPF values correlate with reticulated platelets and are influenced by platelet size[52]. Measurement of platelet size and volume is done by MPV. In our study, there was a statistically significant positive correlation between Mean Platelet Volume and IPF (%) ($p = <0.001$). For every 1 unit increase in Mean Platelet Volume, IPF (%) increased by 0.27 units. Conversely, for every 1 unit increase in IPF (%), Mean Platelet Volume increased by 0.87 units.

Treatment history with antiplatelets

In patients with previous diagnosis of coronary artery disease, continued use of antiplatelet drugs including aspirin and clopidogrel is given to prevent future coronary thrombotic events. Despite this use, many patients develop restenosis of coronary vasculature. A study conducted by Wu, Zhao-Ke et al. showed significant clopidogrel resistance to be associated with obesity, diabetes and hyperuricemia[53].

A study conducted by Guthikonda, Sasidhar et al. showed that larger and reticulated platelets have increased reactivity irrespective of dual antiplatelet therapy. Larger platelets were evaluated using MPV and reticulated platelets using flow cytometry[54].

In our study, there was a significant difference between the 2 groups with relation to Mean Platelet Volume ($p = 0.017$), with the median Mean Platelet Volume being higher in the group on antiplatelet therapy. There was no significant difference between the groups when comparing antiplatelet use history in terms of IPF (%) ($p = 0.149$). Strength of association was 0.13, reflecting small size effect.

Thus, Guthikonda, Sasidhar et al. showed that in chronic stable coronary artery disease, with elevated MPV and increase reticulated platelets, there is platelet aggregation and activation irrespective of antiplatelet status[54]. Thus platelet size can be used as a measure to evaluate risk of future coronary artery disease irrespective of medication status.

Relation with cholesterol

Hyperlipidaemia is associated with increase in platelet size and activity. A study conducted by Singh, Anurag et al. showed increase in platelet distribution width (PDW) and aggregation in dyslipidaemia patients[55]. Another study conducted by Ravindran, Resmi, and Lissy K Krishnan showed that hyperlipidaemia increases the platelet lipid content and increases platelet activity, thus increasing risk of coronary artery disease[56].

In our study, there was a positive correlation between Total Cholesterol (mg/dL) and IPF (%), and this was a statistically significant ($p = 0.027$) correlation. For every 1 unit increase in IPF (%), the Total Cholesterol (mg/dL) increases by 18.52 units.

CONCLUSION

Mean platelet volume (MPV) and immature platelet fraction (IPF) are simple inexpensive tests that can be used to evaluate larger, reticulated platelets which are metabolically more active. Diabetes mellitus leads to larger platelets with accelerated turnover time.

Our study shows MPV to be a useful, inexpensive and rapid hematological marker for correlating diabetes and coronary artery disease outcome. MPV directly correlates with severity of coronary artery occlusion, where larger MPV values were noted in patients with triple vessel disease. MPV correlation is an independent indicator of coronary artery disease.

There was a strong correlation between MPV and IPF values in our study, indicating most larger platelets are also reticulated and newly produced from the marrow.

Our study noted no correlation between IPF and coronary artery disease, probably because these immature platelets are only seen within 24 hours of release from marrow in patients with major cardiac adverse effects. Chronic platelet activation leads to formation of mature large platelets which have a high MPV and normal IPF values, as seen with chronic atherosclerosis.

There was a weak correlation between fasting sugars and IPF in our study, indicating the need for a larger sample size to evaluate true relationship between diabetes mellitus status and IPF as a marker for increased platelet activity.

Many patients on antiplatelet treatment have a risk for development of recurrent coronary event. This has been proposed because of clopidogrel and aspirin resistance. One factor for development of this resistance is larger, active platelets as demonstrated in the study we conducted. There was a significant correlation noted between antiplatelet use with MPV and occurrence of a coronary event.

SUMMARY

The study titled "Cross Sectional Study Comparing Immature Platelet Fraction with Degrees of Hyperglycemia in Type 2 Diabetes Mellitus in Post Coronary Angiography Patients at KLES Dr. Prabhakar Kore Hospital" was conducted over a period of one year from January 2023 to December 2023, in the Departments of Medicine and Cardiology at Dr. Prabhakar Kore Hospital, Belgaum, Karnataka. It was a cross sectional, observational study with total number of 130 participants enrolled.

The objectives were to compare mean platelet volume (MPV) and immature platelet fraction (IPF) with hyperglycemia in diabetes mellitus patients with coronary angiographic findings. Other objectives were to correlate MPV with IPF and correlate MPV with antiplatelet use and coronary atherosclerotic burden.

Out of 130 patients included in the study, 87 were males and 43 were females. The mean age of included participants was 61.50 ± 8.80, with the most common age group being 51 to 60 years, including 49 (37.7%) patients. The most common chief complaint was chest pain, reported in 101 (77.7%) patients, followed by dyspnoea on exertion in 66 (50.8%) patients and sweating in 13 (10%) patients.

Previously known comorbidities were present in 119 (91.5%) study participants, all of whom were on medication for the same. Previously known diabetics were 110 (84.6%) patients and rest 20 (15.4%) were newly diagnosed diabetics. Other common comorbidities were hypertension in 80 (61.5%) patients and IHD in 15 (11.5%) patients. Prior medication history included antidiabetic medication in 107 (82.3%) patients, antihypertensive agents in 82 (63.1%) patients, lipid lowering agents in 44 (33.8%) patients and antiplatelets in 43 (33.1%) patients.

The mean HbA1C (%) was 8.39 ± 2.19. The mean Fasting Sugars (mg/dL) was 146.95 ± 65.76. The mean Random Sugars (mg/dL) was 202.88 ± 86.97. The mean IPF (%) was 4.44 ± 0.68. The mean Mean Platelet Volume was 10.46 ± 1.22. The mean Total Cholesterol (mg/dL) was 168.89 ± 64.57. The mean LDL (mg/dL) was 112.85 ± 48.03. The mean HDL (mg/dL) was 34.49 ± 8.58. The mean Triglycerides (mg/dL) was 202.77 ± 109.68.

Cardiac parameters were recorded in all patients. The most common ECG finding was normal sinus rhythm in 34 (26.2%) persons, followed by strain pattern in 23 (17.7%) persons. Most common myocardial infarction seen was evolved anterior wall in 21 (16.2%) persons. Echocardiography (ECHO) findings were compared in all patients. Normal results were in 52 (40%) persons. Most common anomaly was akinesia of either free wall in 56 (43.1%) persons and hypokinesia was seen in 29 (22.3%) persons. Coronary angiography findings were compared to evaluate coronary atherosclerotic burden. 36% patients has single vessel disease, 29% patients had double vessel disease and 35% patients were triple vessel disease.

In our study, we compared MPV with HbA1C, fasting blood glucose and random blood glucose, to observe association with diabetic status. The results showed a statistically significant ($p = 0.006$) positive connection between Mean Platelet Volume and HbA1C (%). An increase of 1 unit in Mean Platelet Volume results with a 0.50 unit rise in HbA1C (%). Mean Platelet Volume and Fasting Sugars (mg/dL) correlated positively, and this relationship was statistically significant ($p = 0.007$). There is a 10.19 unit rise in fasting sugars (mg/dL) for every unit increase in mean platelet volume. There was no statistically significant correlation between Random Sugars (mg/dL) and Mean Platelet Volume ($p = 0.162$). There was also a significant association between mean platelet volume and coronary angiography findings ($p =$

0.011), with the median Mean Platelet Volume being highest in the Coronary Angiography: Triple Vessel Disease group.

In our study, there was no statistically significant correlation between HbA1C (%) and IPF (%) ($p = 0.706$). Between Fasting Sugars (mg/dL) and IPF (%), there was a positive connection that was statistically significant ($p = 0.046$). The Fasting Sugars (mg/dL) rise by 13.74 units for every unit increase in IPF (%). There was no statistically significant correlation between Random Sugars (mg/dL) and IPF (%) ($p = 0.233$). The results of the coronary angiography in the single vessel, double vessel, and triple vessel groups did not differ significantly in our investigation, in terms of correlation with IPF (%) ($F = 1.458$, $p = 0.237$). The strength of association was 0.1, indicating small effect size.

Our study also showed a positive correlation between Mean Platelet Volume and IPF (%), and this was a statistically significant ($p = <0.001$) correlation. IPF (%) rose by 0.27 units for every unit increase in Mean Platelet Volume. On the other hand, Mean Platelet Volume increased by 0.87 units for every unit rise in IPF (%).

When comparing MPV and antiplatelet drug history, there was a significant difference between the 2 groups ($p = 0.017$), with the median Mean Platelet Volume being higher in the group on antiplatelet therapy. There was no significant difference between the groups when comparing antiplatelet use history in terms of IPF (%) ($p = 0.149$). Strength of association was 0.13, reflecting small size effect.

Lastly, between Total Cholesterol (mg/dL) and IPF (%), we found a positive connection that was statistically significant ($p = 0.027$). Total Cholesterol (mg/dL) rises by 18.52 units for every unit increase in IPF (%).

LIMITATIONS

Automated machines were used to calibrate MPV and IPF values, which are faster and relatively less expensive, but can have variable results. More accurate for measurement is flow cytometry analysis and smear evaluation of reticulated platelets. Another lacuna in the study was the lack of proper timing for collection of MPV and IPF samples. MPV rises with the duration of anticoagulant administration and falls with cytoplasmic content dilution. Due to small sample size, single centre for patient recruitment and cross sectional data collection, follow up period could not be established. Additional correlations and long term data including effect on mortality could not be evaluated. Thus, effect of these biomarkers may be possibly explored in greater detail by increasing the sample size, conducting multi-centre research and adding a follow up period for patients recruited.

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ANNEXURES – I

INFORMED CONSENT FORM

Dear Mr. /Mrs. /Dr. _____, you are kindly requested to enroll yourself in a research study tilted, "Cross Sectional Study Comparing Immature Platelet Fraction with Degrees of Hyperglycemia in Type 2 Diabetes Mellitus in Post Coronary Angiography Patients at KLES Dr. Prabhakar Kore Hospital" being conducted by BG0121002, a post graduate student in M.D. General Medicine and the study will be carried out under the direct supervision and guidance of, Professor, Department of General Medicine, Jawaharlal Nehru Medical College, Belgaum.

You have been requested to participate in this as you fit into the laid-out criteria for a study 'subject'/ participant.

Your participation in study is voluntary. During the study you will be asked some questions and you are supposed to answer to the best of your knowledge. Your decision whether to participate in the study will not affect your treatment in any form. If you decide to participate you are free to withdraw at any time.

TITLE OF THE STUDY:

"Cross Sectional Study Comparing Immature Platelet Fraction with Degrees of Hyperglycemia in Type 2 Diabetes Mellitus in Post Coronary Angiography Patients at KLES Dr. Prabhakar Kore Hospital"

PURPOSE OF THE STUDY: To compare immature platelet fraction (IPF) and degree of diabetes mellitus in post coronary angiography patients.

PROCEDURES INVOLVED: If you agree to enroll yourself in my study, you will be interviewed regarding your present, past and family history then you will be clinically examined in detail and investigated accordingly.

Then you will be subjected to a few blood investigations, namely Immature platelet fraction (IPF) and platelet indices including platelet count, mean platelet volume will be done.

Additional testing will include Hb, total leucocyte count, urea, creatinine, sodium, potassium, chloride, total and direct bilirubin, SGOT, SGPT, ALP, total protein, albumin, aPTT, PT INR, ECG, 2D ECHO, HbA1C and fasting lipid profile.

RISKS AND BENEFITS: There are no potential risks involved in this study.

Benefits of taking part in this research: By taking part in this study, you will be able to compare immature platelet fraction and degree of diabetes mellitus in post coronary angiography patients.

VOLUNTARY PARTICIPATION / WITHDRAWAL FROM THE STUDY: Taking part in the study is voluntary. You may choose not to enroll yourself in this study and may choose to leave the study anytime in between.

ALTERNATIVES: Your decision regarding participation in study will not change present or future health care services offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. You would simply be excluded from the study if you wish to, and all your details shall be kept confidential, and you will get the routine line of management.

PRIVACY AND CONFIDENTIALITY: All data collected or disclosed by you during participation of study, will be kept fully confidential. If however during the course it becomes necessary for the progress of the course to disclose the identity, it would be done so only after your informed & written consent. The only people to know that you are a research subject are members of the research team. No information about you will be disclosed to other without your written permission except:

In emergency to protect your rights AND welfare.

If required by law.

AUTHORIZATION TO PUBLISH RESULT: The results of the study may be used to publish an article. When the results of research published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information obtained in connection with this study and that can be identified with you will remain confidential.

FINANCIAL INCENTIVES FOR PARTICIPATION: No additional costs shall be incurred upon you for the purpose of this study. It is purely being done with the idea of research and all the cost of study will be borne by the investigator.

COMPENSATION: In the event that you become injured as a result of taking part in this study, treatment will be offered to you at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum, or you will be given information about where to receive medical care. However, no reimbursement, compensation or free medical care will be given.

QUESTIONS/CONTACT DETAILS: You shall be free to contact the below mentioned name & addresses anytime during the study period for any clarification or help as you may desire for.

CONSENT FORM

I, voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read this consent form, or it has been read to me, this consent form and have had all the questions answered.

……..……..

…..……

Name of the Participant

Signature of the participant

or Left-Hand Thumb impression

……

..……

Name of Investigator

Signature of investigator

or Left-Hand Thumb impression

……

……..

Name of Witness

Signature of Witness

or Left-Hand Thumb impression

Date:

Place:

ANNEXURES II-PROFORMA

Hospital Based Study Comparing Immature Platelet Fraction with Degrees of Hyperglycemia in Type 2 Diabetes Mellitus in Post Coronary Angiography Patients

PROFORMA

IP Number	
Name	
Age	
Sex	

Present Complaints	
Past History	
Family History	
Personal History	
Treatment History	

General Examination:

Pallor	
Icterus	
Cyanosis	
Clubbing	
Edema	
Lymphadenopathy	
Pulse	
Blood Pressure	
Temperature	
Respiratory Rate	

Systemic Examination:

Cardiovascular System	
Respiratory System	
Per Abdomen	
Central Nervous System	

Investigations:

HbA1C	
Fasting Sugars	
Random Sugars	
Immature Platelet Fraction (IPF)	
Platelet Count	
Mean Platelet Volume (MPV)	
Hemoglobin	
Total Leucocyte Count	
Urea	
Creatinine	
Sodium	
Potassium	
Chloride	
Total Bilirubin	
Direct Bilirubin	
SGPT	
SGOT	
ALP	
Total Proteins	
Albumin	
aPTT/PT INR	

Fasting Lipid Profile	
ECG	
2D ECHO	
Coronary Angiography	

Number	IP Number	Age	Sex	Chief complaints	Past history	Personal history	Treatment history	General examination	Pulse	Blood Pressure	Respiratory rate	Cardiovascular system	Respiratory system	Per Abdomen	Central nervous system
1	10003895	67	Male	Dyspnoea on exertion, giddiness and palpitations	Hypertension, Diabetes mellitus and complete heart block with pacemaker	-	Antihypertensive, antidiabetics and lipid lowering agents	Normal	78/min	130/80 mmHg	14/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
2	10003652	71	Male	Dyspnoea on exertion, chest pain on exertion	IHD - IWMI, TVD s/p CABG, hypertension, Diabetes mellitus	Tobacco chewer	Antihypertensive, antidiabetics, beta blockers, diuretics and lipid lowering agents	Pallor, edema	88/min	140/80 mmHg	14/min	S1S2 heard, no murmur	B/L basal crepitations	Soft, non tender	No focal deficits
3	10003069	70	Female	Chest pain radiating to back, nausea, sweating	Hypertension, Diabetes mellitus	-	Antihypertensives	Normal	98/min	110/80 mmHg	26/min	Loud S2, no murmur	B/L basal crepitations	Soft, non tender	No focal deficits
4	10002561	47	Male	Dyspnoea on exertion, chest pain on exertion	-	Tobacco chewer	-	Edema	74/min	110/70 mmHg	16/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
5	10005585	62	Male	Chest and epigastric pain, sweating	Diabetes mellitus	Tobacco chewer	Antidiabetics	Normal	80/min	110/80 mmHg	20/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
6	10007570	59	Male	Chest pain radiating to back	-	Smoker	-	Normal	80/min	130/70 mmHg	16/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
7	10005080	61	Female	Chest pain radiating to back, dyspnoea on exertion	Diabetes mellitus, IHD - AWMI	-	Antidiabetics, antiocoagulants and lipid lowering agents	Normal	60/min	150/90 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
8	10004473	66	Male	Chest pain radiating to back, dyspnoea on exertion	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Pallor, edema	150/min	100/60 mmHg	38/min	S1S2 heard, no murmur	B/L coarse crepitations	Soft, non tender	No focal deficits
9	10004447	47	Male	Chest pain	Hypertension, Diabetes mellitus	Alcoholic	Antihypertensives, antidiabetics	Normal	90/min	130/80 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
10	10002731	55	Female	Dyspnoea on exertion, orthopnea, PND	Diabetes mellitus	-	Antidiabetics	Edema	80/min	120/80 mmHg	20/min	S1S2 heard, no murmur	B/L basal crepitations	Soft, non tender	No focal deficits
11	10006044	55	Female	Dyspnoea on exertion, chest pain radiating to back	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	90/min	170/100 mmHg	20/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
12	10006106	60	Male	Retrosternal chest pain	-	-	-	Normal	70/min	100/80 mmHg	14/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
13	10006699	59	Female	Chest pain radiating to back	-	-	-	Edema	72/min	96/60 mmHg	16/min	Pansystolic murmur in mitral area	Air entry equal	Soft, non tender	No focal deficits
14	10004467	58	Male	Dyspnoea on exertion, chest pain radiating to back	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	65/min	150/90 mmHg	14/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
15	10041225	47	Male	Chest pain, sweating, palpitations	Diabetes mellitus	Tobacco chewer	Antidiabetics	Normal	70/min	130/80 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
16	10040758	55	Female	Chest pain radiating to back	Diabetes mellitus	-	Antidiabetics	Normal	88/min	110/70 mmHg	16/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
17	10039208	55	Male	Dyspnoea on exertion	Hypertension, Diabetes mellitus, Chronic kidney disease	-	Antihypertensives, antidiabetics	Pallor, edema	104/min	110/70 mmHg	30/min	S1S2 heard, no murmur	B/L coarse crepitations	Soft, non tender	No focal deficits
18	10038939	60	Male	Dyspnoea on exertion, chest pain on exertion	Hypertension	Smoker, alcoholic	Antihypertensives	Clubbing	102/min	90/60 mmHg	32/min	S1S2 heard, no murmur	B/L coarse crepitations	Soft, non tender	No focal deficits
19	10039636	63	Male	Dyspnoea on exertion, chest pain on exertion	Hypertension, Diabetes mellitus	Smoker	Antihypertensives, antidiabetics	Normal	72/min	140/70 mmHg	24/min	Early systolic murmur in aortic area	B/L basal crepitations	Soft, non tender	No focal deficits
20	10038559	62	Male	Dyspnoea on exertion, palpitations	Hypertension	-	Antihypertensives	Normal	70/min	140/90 mmHg	30/min	S1S2 heard, no murmur	B/L basal crepitations	Soft, non tender	No focal deficits
21	10039161	57	Female	Chest pain on exertion	Hypertension	-	Antihypertensives	Normal	76/min	130/90 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
22	10014856	73	Male	Dyspnoea on exertion, chest pain radiating to back	Diabetes mellitus	-	Antidiabetics	Pallor	83/min	110/80 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
23	10014842	58	Male	Dyspnoea on exertion	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	118/min	120/70 mmHg	30/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
24	10013599	52	Male	Dyspnoea on exertion, palpitations	-	Tobacco chewer	-	Normal	130/min	120/70 mmHg	16/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
25	10013838	65	Female	Chest pain radiating to back	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	120/min	120/70 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
26	10012579	75	Male	Dyspnoea on exertion, PND	Diabetes mellitus, Adenocarcinoma of rectum	-	Antidiabetics	Normal	90/min	110/70 mmHg	18/min	S1S2 heard, no murmur	B/L basal crepitations	Soft, non tender	No focal deficits
27	10013644	59	Male	Dyspnoea on exertion, sweating	Diabetes mellitus	-	Antidiabetics	Normal	76/min	120/70 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
28	10038654	58	Female	Chest pain radiating to back	-	-	-	Normal	94/min	130/80 mmHg	16/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
29	10038578	49	Male	Chest pain radiating to back	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	70/min	110/70 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
30	10037979	53	Male	Dyspnoea on exertion, chest pain on exertion	IHD s/p CABG	Smoker	Anticoagulants, beta blockers and lipid lowering agents	Normal	88/min	120/80 mmHg	16/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
31	10037259	73	Male	Dyspnoea on exertion	-	Smoker, alcoholic	-	Normal	72/min	120/80 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
32	10037506	60	Male	Chest pain on exertion	-	Tobacco chewer, alcoholic	-	Normal	100/min	120/70 mmHg	16/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
33	10036609	55	Male	Giddiness and palpitations	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	120/min	120/70 mmHg	22/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
34	10036646	65	Male	Retrosternal chest pain	Hypertension, Diabetes mellitus	Smoker	Antihypertensives, antidiabetics	Normal	65/min	150/90 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
35	10036685	58	Female	Dyspnoea on exertion	Hypertension	Tobacco chewer	Antihypertensives	Normal	68/min	110/70 mmHg	16/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
36	10036784	70	Female	Chest pain radiating to back	Hypertension, Diabetes mellitus and uterine prolapse	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	90/min	130/70 mmHg	14/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
37	10036051	79	Male	Dyspnoea on exertion, chest pain on exertion	Hypertension, Diabetes mellitus and IHD s/p PTCA	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	80/min	130/80 mmHg	14/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
38	10035629	55	Female	Chest pain and sweating	-	-	-	Normal	88/min	110/70 mmHg	20/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
39	10035404	74	Male	Chest pain radiating to back	Hypertension, Diabetes mellitus and old CVA	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	120/min	100/60 mmHg	30/min	S1S2 heard, no murmur	B/L coarse crepitations	Soft, non tender	No focal deficits
40	10032411	57	Male	Dyspnoea on exertion, chest pain on exertion	Diabetes mellitus, IHD s/p CABG	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	76/min	110/70 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
41	10032431	83	Male	Retrosternal chest pain	Hypertension, Diabetes mellitus and CA prostate	-	Antihypertensives, antidiabetics	Normal	84/min	130/70 mmHg	16/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
42	10035928	67	Female	Chest pain	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	82/min	110/80 mmHg	17/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
43	10041883	67	Male	Dyspnoea on exertion, chest pain	Diabetes mellitus and COPD	-	Antidiabetics and inhaler	Normal	76/min	150/90 mmHg	26/min	S1S2 heard, no murmur	B/L wheeze	Soft, non tender	No focal deficits
44	10041794	67	Female	Dyspnoea on exertion, chest pain on exertion	Hypertension, Diabetes mellitus and rheumatoid arthritis	-	Antihypertensives, antidiabetics and disease modifying drugs	Normal	70/min	120/80 mmHg	16/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
45	10041690	79	Female	Chest pain radiating to back	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	70/min	100/60 mmHg	16/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
46	10041225	47	Male	Chest pain, sweating, palpitations	Diabetes mellitus	Smoker, alcoholic	Antidiabetics	Normal	70/min	130/80 mmHg	14/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
47	10040758	58	Female	Dyspnoea on exertion, chest pain on exertion	Diabetes mellitus	-	Antidiabetics	Normal	88/min	110/70 mmHg	12/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
48	10039208	55	Male	Dyspnoea on exertion	Hypertension, Diabetes mellitus, Chronic kidney disease and old CVA	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Pallor, edema	104/min	110/80 mmHg	30/min	Pansystolic murmur in mitral area	B/L coarse crepitations	Soft, non tender	Right hemiplegia
49	10039636	63	Male	Dyspnoea on exertion, chest pain on exertion	Hypertension, Diabetes mellitus	Smoker	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	72/min	140/70 mmHg	26/min	Early systolic murmur in aortic area	B/L coarse crepitations	Soft, non tender	No focal deficits
50	10038559	62	Male	Dyspnoea on exertion, palpitations	Hypertension, B/L knee osteoarthritis	Tobacco chewer	Antihypertensives, calcium supplements	Normal	70/min	140/90 mmHg	24/min	S1S2 heard, no murmur	B/L basal crepitations	Soft, non tender	No focal deficits
51	10041196	56	Male	Chest pain, sweating, palpitations	Diabetes mellitus	-	Antidiabetics	Normal	98/min	120/80 mmHg	18/min	Pansystolic murmur in mitral area	Air entry equal	Soft, non tender	No focal deficits
52	10039197	84	Male	Dyspnoea on exertion, chest pain radiating to back	Hypertension, Diabetes mellitus	Alcoholic	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	76/min	130/100 mmHg	15/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
53	10039391	64	Male	Retrosternal burning	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	84/min	130/80 mmHg	12/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
54	10036399	57	Male	Chest pain on exertion	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	62/min	130/80 mmHg	13/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
55	10035905	55	Male	Dyspnoea on exertion, chest pain	Diabetes mellitus	Tobacco chewer, alcoholic	Antidiabetics	Pallor	126/min	110/70 mmHg	26/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
56	10034344	66	Male	Dyspnoea on exertion, chest pain radiating to back	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	82/min	130/80 mmHg	15/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
57	10041529	64	Male	Dyspnoea on exertion, chest pain	Hypertension, Diabetes mellitus and COPD	Smoker, alcoholic	Antihypertensives, antidiabetics and inhalers	Normal	136/min	160/90 mmHg	28/min	Pansystolic murmur in mitral area	B/L basal crepitations and rhonchi	Soft, non tender	No focal deficits
58	10042165	65	Male	Dyspnoea on exertion, chest pain	Diabetes mellitus	-	Antidiabetics	Normal	64/min	120/70 mmHg	14/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
59	10040191	68	Male	Dyspnoea on exertion	Diabetes mellitus and hypothyroidism	-	Antidiabetics, thyroid supplements	Edema	84/min	140/64 mmHg	24/min	S1S2 heard, no murmur	B/L basal crepitations	Soft, non tender	No focal deficits
60	10039186	53	Male	Chest pain on exertion	Diabetes mellitus	Alcoholic	Antidiabetics	Normal	80/min	160/90 mmHg	16/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
61	10045483	70	Female	Dyspnoea on exertion, chest pain on exertion	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	68/min	140/70 mmHg	14/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
62	10045014	75	Male	Dyspnoea on exertion	Hypertension, Diabetes mellitus	Smoker, alcoholic	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	72/min	120/80 mmHg	12/min	Midsystolic murmur in aortic area	Air entry equal	Soft, non tender	No focal deficits
63	10043745	65	Male	Dyspnoea on exertion, chest pain	Hypertension, Diabetes mellitus	Tobacco chewer	Antihypertensives, antidiabetics	Normal	90/min	150/90 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
64	10045553	65	Male	Chest pain	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	78/min	150/90 mmHg	14/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
65	10043789	41	Female	Chest pain on exertion	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	80/min	150/90 mmHg	12/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
66	10041583	63	Male	Dyspnoea on exertion, chest pain	Diabetes mellitus	-	Antidiabetics	Normal	118/min	150/90 mmHg	28/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
67	10042126	69	Male	Dyspnoea on exertion, chest pain	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	68/min	140/80 mmHg	14/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
68	10041750	45	Male	Chest pain on exertion	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	100/min	130/90 mmHg	14/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
69	10041128	48	Male	Dyspnoea on exertion, chest pain	Hypertension, IHD s/p PTCA	-	Antihypertensive, anticoagulants and lipid lowering agents	Normal	86/min	100/60 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
70	10039466	62	Female	Chest pain	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	118/min	130/80 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
71	10039950	42	Female	Chest pain, weakness of left upper and lower limb	Hypertension, Diabetes mellitus	Alcoholic	Antihypertensives, antidiabetics	Normal	100/min	210/100 mmHg	32/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	Left hemiparesis
72	10040354	62	Female	Chest pain on exertion	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	118/min	130/80 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
73	10039864	58	Female	Chest pain radiating to back	Hypertension, Diabetes mellitus, IHD s/p CABG and hypothyroidism	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents, thyroid supplements	Normal	68/min	120/70 mmHg	14/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
74	10052680	73	Male	Dyspnoea on exertion, chest pain	Hypertension, Diabetes mellitus	Alcoholic	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	78/min	150/90 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
75	10050588	65	Male	Dyspnoea on exertion, swelling of legs	Diabetes mellitus, peripheral vascular disease and hypothyroidism	Smoker	Antidiabetics, thyroid supplements, anticoagulants	Pallor, edema	76/min	110/70 mmHg	22/min	S1S2 heard, no murmur	B/L coarse crepitations	Soft, non tender	No focal deficits
76	10049487	62	Female	Chest pain radiating to back	-	-	-	Normal	70/min	120/70 mmHg	28/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
77	10050790	60	Male	Chest pain radiating to back	Hypertension, Diabetes mellitus and hypothyroidism	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents, thyroid supplements	Normal	76/min	120/70 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
78	10050939	74	Male	Retrosternal burning	Diabetes mellitus	-	Antihypertensive, anticoagulants and lipid lowering agents	Normal	72/min	130/80 mmHg	15/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
79	10051102	60	Female	Chest pain on exertion	Diabetes mellitus	-	Antidiabetics	Normal	76/min	160/90 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
80	10051989	55	Female	Dyspnoea on exertion	Hypertension, Diabetes mellitus and hypothyroidism	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents, thyroid supplements	Normal	78/min	110/70 mmHg	20/min	S1S2 heard, no murmur	B/L basal crepitations	Soft, non tender	No focal deficits
81	10052007	67	Male	Dyspnoea on exertion	Diabetes mellitus	-	Antidiabetics	Normal	100/min	110/70 mmHg	24/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
82	10052016	49	Male	Dyspnoea on exertion, chest pain on exertion	Diabetes mellitus	Alcoholic	Antidiabetics	Normal	72/min	100/70 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
83	10049992	61	Male	Chest pain on exertion, giddiness	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	50/min	130/90 mmHg	20/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits
84	10051966	49	Female	Retrosternal burning	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	90/min	180/100 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits

Number	HbA1C	Fasting sugars	Random sugars	IPF	Platelet count	Mean platelet volume	Hemoglobin	Leucocyte count	Urea	Creatinine	Sodium	Potassium	Chloride	Total Bilirubin	Direct Bilirubin	SGPT	SGOT	ALP	Total proteins	Albumin	aPTT/PT INR	Cholesterol	LDL	HDL	TG	ECG	ECHO	Coronary angiography
1	6.9	67	102	4.04	318	9.8	14.3	8900	21.8	0.81	142	4.49	104	0.59	0.3	15	22	74	7.4	4.4	1.28/1.01	192	96	52	142	Complete heart block with paced rhythm	Normal resting LV systolic function. Trivial MR, sclerotic AV valve, mild PAH, EF 60%	Double Vessel Disease
2	5.9	130	226	3.1	235	8.2	8.3	3500	25.7	0.93	127	4.01	97	0.4	0.21	12	17	77	6.2	4	1.01/1.21	292	164	42	202	LVH with strain pattern	Akinesia of inferior, inferolateral, basal and inferoseptal wall, moderate LV dysfunction, EF 40%, moderate MR, moderate PAH, LA dilated	Triple Vessel Disease
3	5.7	101	129	4.76	352	10.6	10.6	14,000	15	0.71	134	4.34	99	0.84	0.46	24	29	72	7.8	3.6	2.14/1.31	236	158	59	146	Inferior, posterior wall and right ventricular MI	Hypokinesia of inferior, inferoseptum, inferolateral wall, mild LV dysfunction, EF 45%, moderate MR, grade 2 TR, moderate PAH	Triple Vessel Disease
4	8.2	96	115	4.41	276	10	12.1	6300	35	1.19	129	4.46	98	0.66	0.4	37	38	83	7.4	3.7	1.16/1.49	128	92	58	142	Rbbb, strain pattern in lateral leads	Global hypokinesia of LV, EF 30%, LA LV dilated, grade 2 MR	Double Vessel Disease
5	9.9	101	193	3.2	187	8.3	13	8700	23	1.39	131	3.92	95	0.32	0.12	12	18	72	7.1	3.4	1.12/1.09	114	56	17	287	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Double Vessel Disease
6	6.7	82	95	4.12	265	11.1	15.8	8200	16	0.94	139	4.81	105	1.1	0.56	28	24	62	6.9	3.2	1.12/0.91	257	190	42	205	Normal sinus rhythm	Akinesia of anterior wall, anteroseptum, apex and hypokinesia of apical septum, EF 46%	Triple Vessel Disease
7	5.8	76	80	3.78	288	10.2	11.8	8000	12.7	0.67	142	3.64	104	0.78	0.38	21	27	71	7.2	4.2	1.47/0.95	312	192	31	190	Strain pattern in anterior leads	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%, thickened aortic valve	Triple Vessel Disease
8	6.5	120	167	4.91	270	12.8	15.4	30,200	34.5	1.46	140	4.64	97	2.1	1.1	85	465	92	6.2	3.2	1.16/1.11	107	55	34	165	Anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum, apicolateral septum. Apex thinned out, severe LV dysfunction, EF 30%	Triple Vessel Disease
9	4.7	67	92	5.42	115	14.9	13.8	10,600	13	0.83	139	3.78	104	0.92	0.45	32	31	78	7	3.5	1.27/1.20	199	134	44	121	Evolved anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum and apex, moderate LV dysfunction, EF 40%	Single Vessel Disease
10	10.2	210	425	3.9	269	9.1	13.2	10,200	29.6	0.69	126	4.86	88	1.22	0.42	32	38	72	7.6	3.6	0.91/0.95	304	202	42	159	Lbbb	Asynchronous LV movement, global hypokinesia of LV with severe LV dysfunction, EF 30%	Double Vessel Disease
11	6.9	140	171	4.15	272	10.1	12.3	10,700	22	0.71	138	4.19	103	1.02	0.52	31	28	68	7.1	4.1	1.25/0.82	162	92	51	192	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Single Vessel Disease
12	5.8	82	91	4.48	183	10.2	10.3	6,000	22.9	1.02	138	4.54	105	0.92	0.5	22	18	76	5	2.8	0.97/0.92	172	96	52	142	Anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum and apex, mild LV dysfunction, EF 45%	Single Vessel Disease
13	6.1	78	89	5.1	194	12.7	11.1	5,800	24	0.86	135	3.61	98	1.02	0.58	12	18	82	7.2	4.6	0.94/1.02	202	196	38	262	Anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum, apicolateral segment and apex, moderate LV dysfunction, EF 40%, moderate MR	Triple Vessel Disease
14	4.9	72	89	3.93	282	8.9	15.2	8,300	19.9	0.98	136	4.46	100	1.02	0.32	22	28	67	7.2	3.6	0.72/0.98	156	132	56	192	Evolved anterior wall MI	Hypokinesia of anterior wall, anteroseptum, apical septum and apex, mild LV dysfunction, EF 45%	Single Vessel Disease
15	9	190	251	4.72	305	9.7	17.2	12,900	16.4	1.04	137	4.39	99	1.1	0.9	32	30	96	6.2	3.2	1.0/1.10	181	131	26	201	Evolved inferior wall MI	Akinesia of inferior wall, inferoseptum, inferolateral wall, mild LV dysfunction, EF 45%, pericardial effusion	Double Vessel Disease
16	11.3	98	149	4.53	452	9.6	11	10,600	15.2	0.52	135	4.22	102	0.38	0.23	14	22	71	7.2	4.1	1.14/1.01	320	182	30	300	Evolved inferior wall MI	Hypokinesia of mid and basal inferior wall, EF 55%	Double Vessel Disease
17	7.2	160	201	4.8	277	9.9	10.7	13,900	90.4	2.18	142	3.98	105	0.54	0.41	705	638	117	7	3.8	1.02/1.05	280	200	40	262	Lbbb	Akinesia of inferior wall, inferoseptum, inferolateral wall, moderate LV dysfunction, EF 35%, dilated LV	Single Vessel Disease
18	7	92	182	3.92	282	8.9	11	8,500	58.2	1.17	140	3.31	104	0.4	0.3	22	26	94	6.8	4	0.99/1.38	235	161	47	223	Anterior wall MI	Akinesia of anterior wall, anteroseptum, apical wall, mild LV dysfunction EF 45%, moderate MR, moderate PAH	Single Vessel Disease
19	8.2	100	159	4.35	244	9.5	11.5	4,600	22.3	0.98	142	4.6	106	0.42	0.22	30	28	92	7.2	3.2	1.05/1.13	280	142	38	192	Normal sinus rhythm	Bicuspid aortic valve, normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Triple Vessel Disease
20	5.6	92	148	5.2	193	10.6	13.8	8,400	45.9	1.69	138	3.88	104	0.2	0.12	22	24	68	6.2	3.2	1.23/1.02	224	108	48	200	Evolved inferior wall MI	Akinesia of mid and basal inferoseptum, inferior wall and inferolateral wall, EF 45%	Double Vessel Disease
21	6.8	114	180	4.96	291	9.2	12.3	9,000	33.9	0.84	140	4.16	105	0.43	0.22	4	17	81	7.3	4.3	1.04/1.12	120	74	37	115	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Single Vessel Disease
22	7.3	124	206	4.54	358	9.9	17.1	10,800	44.6	1.79	137	4.27	100	1.1	0.67	28	36	100	6.8	3.9	0.87/1.23	115	47.5	21	147	Evolved anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum, apex, inferior wall, inferoseptum, moderate LV dysfunction, EF 35%	Single Vessel Disease
23	10.7	240	417	5.41	304	10.8	11.7	10,400	40.4	1.31	134	4.78	102	0.8	0.46	35	37	94	7.2	3.8	0.94/0.94	239	162	37	160	Sinus tachycardia	Akinesia of anterior wall, anteroseptum, apical septum, apicolateral segment and apex, moderate LV dysfunction, EF 40	Double Vessel Disease
24	6.6	113	190	5.5	197	12	14.6	5,900	14.1	0.84	139	4.3	103	0.36	0.2	20	28	74	6.4	3.6	1.10/1.03	182	117	37	107	Atrial fibrillation	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%, atrial fibrillation	Single Vessel Disease
25	12.6	260	386	4.73	324	10.9	12.3	11,900	13.8	0.57	134	4.21	98	0.78	0.4	41	36	68	6.2	3.6	1.24/0.95	235	165	34	148	Sinus tachycardia	Hypokinesia of inferolateral segment, mild LV dysfunction, EF 50%	Triple Vessel Disease
26	7.3	283	360	4.55	158	10.3	15	7,000	19.6	0.92	136	3.14	101	0.9	0.47	13	20	116	7.9	4	1.01/0.94	257	178	24	288	Evolved anterior wall MI	Hypokinesia of anterior wall, anteroseptum, apical septum, inferior wall and apex, moderate LV dysfunction, EF 40%	Single Vessel Disease
27	10.7	227	320	3.71	262	10.4	12.3	8,200	26.4	1.08	132	4.21	99	0.96	0.41	27	34	68	6.3	3.2	1.08/0.88	133	74	44	74	Lbbb with bifascicular block	Asynchronous LV movement, borderline resting LV systolic function, EF 50%	Double Vessel Disease
28	7.2	164	210	4.98	404	11.9	10	14,600	29.7	0.82	135	4.02	100	0.99	0.48	88	65	165	6.6	3.4	1.15/1.32	190	102	30	168	Evolved anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum, inferoseptum, moderate LV dysfunction, EF 45%	Single Vessel Disease
29	8.8	123	168	4.6	298	11.3	11.6	9,200	12.6	0.83	141	4.74	105	0.62	0.28	34	27	70	6.1	3.1	1.16/0.93	73	27	30	157	Evolved inferior wall MI	Akinesia of inferior wall, inferolateral segment, EF 50%, muscle mass preserved	Triple Vessel Disease
30	6.7	94	142	3.71	226	8.8	13.7	9,400	25.4	1.01	135	3.65	103	1.49	0.66	33	50	75	6.9	4.3	1.31/1.01	116	57	36	168	Evolved inferior wall MI, lateral strain pattern	Akinesia of apical septum, apex, borderline resting LV systolilc function, EF 50%, mild PAH	Single Vessel Disease
31	7.1	128	202	5.15	401	10.2	10.8	11,100	54.3	1.33	135	3.85	95	0.82	0.42	20	12	74	6.2	3.2	0.95/0.96	204	106	28	154	Evolved inferior wall MI	Akinesia of apical septum, apex, hypokinesia of anterior wall, anterior septum, EF 45%	Triple Vessel Disease
32	6.6	141	232	4.83	239	9	11.5	9,300	23.1	0.99	133	3.62	102	2.07	1.34	22	22	275	6.7	3	1.05/1.33	242	130	36	102	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Single Vessel Disease
33	6.3	109	190	4.48	376	9.9	13.4	9,700	29.4	1.21	141	4.5	103	0.69	0.45	20	15	86	7.7	4.8	1.07/0.98	147	84	55	119	Evolved inferior wall MI	Akinesia of inferiolateral, inferior, inferoseptum and lateral wall, moderate LV dysfunction, EF 40%, RV dysfunction	Single Vessel Disease
34	8.9	190	266	5.17	120	10.9	16.8	5,600	19.3	0.85	136	4.22	103	0.7	0.38	26	20	74	7.2	3	1.00/0.90	260	146	32	282	Strain pattern in lateral leads	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Double Vessel Disease
35	6.6	93	146	4.41	275	9.1	10.9	10,900	29.2	0.72	139	4.11	105	0.58	0.22	32	27	82	7.8	4.5	1.17/0.95	102	78	48	112	Strain pattern in lateral leads	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Single Vessel Disease
36	9.4	186	256	5.47	234	12.7	11.6	10,100	18.6	0.84	136	3.39	103	0.9	0.52	26	18	70	6.2	3.2	0.95/1.01	211	154	32	192	Evolved inferior wall MI, posterior wall MI	Akinesia of basal and inferior wall, lateral wall, borderline resting LV systolic function, EF 50%	Triple Vessel Disease
37	6.9	101	174	4.12	221	9.4	13.7	7,300	16	0.79	140	4.08	104	0.42	0.24	13	18	68	7.4	4.1	0.92/0.83	108	68	31	83	Incomplete Rbbb with strain pattern in anterolateral leads	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%, concentric LVH	Single Vessel Disease
38	11.3	149	211	3.94	452	10.8	11	9,600	15.2	0.53	135	4.22	102	0.38	0.23	14	22	71	7.2	4.2	1.14/1.01	111	72	38	92	Evolved inferior wall MI	Hypokinesia of mid and basal inferior wall, preserved LV systolic function, EF 55%	Double Vessel Disease
39	5.6	101	186	4.47	184	10	12.3	9,200	72.4	2.2	132	4.78	98	1.1	0.64	29	64	91	6.5	3.5	0.99/1.20	148	95	42	81	Anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum, apex, inferior wall, inferoseptum, moderate LV dysfunction, EF 30%	Double Vessel Disease
40	9.7	224	281	3.61	180	9.8	13.7	6,800	29.5	0.92	135	4.68	102	0.82	0.38	17	10	82	6.1	3.1	1.15/0.84	139	83	30	161	Normal sinus rhythm	Hypokinesia of inferior wall, inferoseptum, borderline resting LV systolic function, EF 50%	Triple Vessel Disease
41	9.1	190	265	4.12	259	10.5	11.8	6,700	37.7	1.42	127	3.86	94	1	0.7	26	22	58	7.8	4.1	1.21/0.95	215	141	63	148	Left anterior hemi block	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Triple Vessel Disease
42	10.8	167	230	3.12	264	13.1	8.8	6,400	27.4	0.67	138	3.97	104	0.81	0.41	20	14	51	6.5	4.1	0.93/0.96	94	44	40	88	Lbbb	Asynchronous LV movement, normal LV systolic function, no regional wall motion anomaly, EF 60%	Triple Vessel Disease
43	11.3	110	184	4.83	379	10.7	11.6	10,300	34.8	1.47	138	4.79	106	0.41	0.15	21	25	84	8	4.6	0.98/1.05	184	122	37	124	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Single Vessel Disease
44	7.6	111	205	4	295	9.6	10.2	7,800	32.2	1.15	136	4.86	105	0.5	0.26	17	20	69	6.3	4.1	0.96/0.95	198	88	32	210	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Triple Vessel Disease
45	7.1	176	224	5.15	292	10.3	12.1	6,000	47.1	1.52	132	4.52	100	0.52	0.27	33	24	98	5.9	3.4	1.00/1.12	145	90	29	224	Inferolateral wall MI	Akinesia of anterior wall, anteroseptum, apical septum, apex, mild PAH, EF 30%	Triple Vessel Disease
46	9	206	251	5.41	305	9.7	17.2	12,900	16.4	1.04	137	4.36	99	0.82	0.58	22	20	64	6.6	3.6	1.00/1.10	181	131	26	201	Evolved inferior wall MI	Akinesia of inferior wall, inferoseptum, inferolateral segment, mild LV dysfunction, EF 45%	Double Vessel Disease
47	11.3	113	148	5.35	452	9.6	11	10,600	16.2	0.57	137	4.78	103	0.38	0.23	22	14	71	7.2	4.2	0.94/0.96	220	186	21	168	Evolved inferior wall MI	Hypokinesia of mid and basal inferior wall, preserved LV systolic function, EF 55%	Double Vessel Disease
48	7.2	190	215	3	370	9.9	7.6	10,600	47.4	2.26	139	3.8	102	0.54	0.41	638	705	117	7	3.8	1.02/1.05	114	74	36	132	Lbbb	Akinesia of inferior wall, inferoseptum, inferolateral, anteroseptum wall, moderate LV dysfunction, EF 35%, moderate MR, moderate PAH	Single Vessel Disease
49	8.2	159	192	4.47	244	9.5	11.5	4,600	22.3	0.98	142	4.6	106	1.2	0.72	38	32	64	7.6	4.2	1.05/1.13	184	112	22	222	Normal sinus rhythm	Bicuspid aortic valve, normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Triple Vessel Disease
50	7.5	126	182	5.15	181	10.6	12.1	7,700	29.4	1.45	143	4.07	110	0.6	0.34	18	22	70	8.2	4.8	1.23/1.02	282	146	28	240	Evolved inferior wall MI	Akinesia of basal and mid inferoseptum, inferior wall, inferolateral wall, mild LV dysfunction, EF 45%	Double Vessel Disease
51	11	186	228	4.38	291	10.4	9.6	9,200	64.5	1.24	131	5.33	100	0.33	0.12	29	31	99	7.1	4.1	1.17/1.05	156	106	32	182	Normal sinus rhythm	Akinesia of anteroseptum, apical septum, anterior wall, apex, moderate LV dysfunction, EF 35%, Grade 3 MR	Triple Vessel Disease
52	8.1	202	226	4.76	233	11.7	11.2	7,000	37.1	1.53	134	4.5	102	0.72	0.36	18	20	66	6.8	3.8	1.02/1.01	210	182	46	224	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%, concentric LVH	Triple Vessel Disease
53	9.6	128	182	5.47	206	11.1	12.5	8,400	21.6	0.72	136	4.22	101	0.62	0.23	20	12	123	7.4	4.4	0.98/1.07	128	83	39	103	Evolved anterior wall MI	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Single Vessel Disease
54	7.7	130	148	5.53	271	9.6	16.2	8,400	21.2	0.78	140	4.24	100	0.42	0.2	26	32	68	6.6	3.6	0.95/0.88	111	76	20	132	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Single Vessel Disease
55	9.3	103	132	3.43	154	9.7	17	7,300	22.6	0.67	144	3.97	102	2.06	0.86	94	29	113	6.3	3.3	1.42/1.05	186	112	35	184	Evolved anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum, apex and apicolateral wall, moderate LV dysfunction, EF 40%	Single Vessel Disease
56	6.2	131	186	4.68	285	9.7	11.8	10,400	28	1.55	134	4.13	102	0.8	0.36	32	26	72	6.9	3.7	0.98/0.92	230	161	30	142	LVH with strain pattern	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Triple Vessel Disease
57	5.8	95	121	3.31	230	11.4	8.2	8,100	48.4	1.46	140	3.99	99	0.22	0.15	91	33	81	5.9	3.1	0.93/1.08	110	72	24	98	Sinus tachycardia	Akinesia of inferior wall, inferoseptum, inferolateral wall, mild LV dysfunction, EF 45%, grade 3 MR, moderate PAH	Triple Vessel Disease
58	8.4	99	128	4.25	245	10	12.5	6,700	20	1.24	138	4.08	103	0.3	0.21	22	20	74	7.2	4	1.05/1.12	76	56	26	120	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Single Vessel Disease
59	10	136	162	3.63	289	12.1	12.6	5,900	20.9	0.62	134	4.12	100	0.88	0.4	34	30	110	6.8	3.6	0.94/1.00	206	182	18	222	Strain pattern in lateral leads	Akinesia of anterior wall, anteroseptum, apex, inferior and inferolateral wall, severe LV dysfunction, EF 30%, dilated chambers	Triple Vessel Disease
60	7.8	105	136	3.16	192	9.2	12.5	6,900	14.7	0.94	139	3.95	102	0.28	0.16	12	18	66	7	4.1	1.05/1.17	111	72	31	101	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Single Vessel Disease
61	8.3	169	210	4.27	313	10.5	11.2	8,200	15.9	0.71	137	4.48	103	0.4	0.22	22	20	98	6.1	3.4	1.13/0.97	166	101	38	184	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Single Vessel Disease
62	8.8	158	188	5.31	153	12	14.9	4,600	29.4	1.09	143	4.2	109	0.22	0.14	34	30	67	6.5	3.8	1.11/1.17	220	142	30	146	Evolved anterior wall MI with Rbbb	Akinesia of anterior wall, anteroseptal, apical septum, apex, mild LV dysfunction, EF 45%, moderate calcific AS	Triple Vessel Disease
63	8.1	186	223	3.67	197	9.2	12	7,100	15.9	1.04	138	4.18	105	1.2	0.66	58	40	82	7.1	4.4	0.94/0.93	142	96	28	57	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%, concentric LVH	Single Vessel Disease
64	13.6	302	337	3.87	298	9.8	10.8	8,200	25.2	0.85	138	4.55	103	0.27	0.18	13	19	113	6.9	4	1.03/1.02	94	50	35	102	Evolved anterior wall MI	Hypokinesia of anterior wall, anteroseptum, apical septum and apex, apicolateral segments, mild LV dysfunction, EF 45%	Triple Vessel Disease
65	10.8	232	285	4.19	265	10.8	10.1	6,800	22.6	0.6	135	4.03	100	0.92	0.56	26	22	64	5.8	3.3	0.85/0.95	137	101	27	103	Strain pattern in anterior leads	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Double Vessel Disease
66	10.6	310	342	4.41	274	10.5	12.3	14,700	37.9	0.84	127	3.83	88	0.32	0.1	28	24	112	7.2	4.1	1.43/1.16	136	86	26	190	Anterior wall MI with Rbbb	Akinesia of anterior wall, anteroseptum, apical septum, apex, inferior wall, inferoseptum, moderate LV dysfunction, EF 35%	Single Vessel Disease
67	6.8	126	184	5.1	218	11	13	9,800	11.6	0.65	134	4.3	98	0.21	0.1	18	14	62	6.6	3.6	1.09/0.92	276	114	31	184	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%, concentric LVH	Triple Vessel Disease
68	9	257	294	4.95	195	11.7	13.5	7,200	17.7	0.66	135	4.18	102	1.12	0.67	72	52	78	7.1	4.2	1.00/1.13	84	47	25	125	Anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum and apex, moderate LV dysfunction, EF 40%	Triple Vessel Disease
69	9.4	92	131	3.87	361	9.2	15.5	9,800	25	0.87	133	4.14	94	0.88	0.52	16	21	64	6.7	3.6	1.18/1.01	122	66	38	120	Evolved anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum, apex and hypokinesia of lateral wall, moderate LV dysfunction EF 35%	Single Vessel Disease
70	8.1	173	217	4.41	397	10.7	13.9	13,100	17.5	0.76	133	5.2	15.2	0.51	0.38	46	32	89	6.6	3.7	1.70/1.17	278	191	42	422	Anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum and apex, moderate LV dysfunction, EF 40%	Single Vessel Disease
71	6.1	82	125	5.12	289	9.1	15.3	11,800	62.8	1.48	137	3.61	100	0.66	0.28	157	222	87	8	3.4	0.84/1.04	212	167	31	328	Anterior wall MI with Rbbb	Akinesia of anterior wall, anteroseptum, apical septum and apex, moderate LV dysfunction, EF 40%	Triple Vessel Disease
72	7.7	111	154	3.23	362	9	14.1	9,100	22.6	1.18	136	3.95	101	0.2	0.12	21	22	72	7.4	4.1	1.21/1.01	212	164	40	386	Anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum and apex, moderate LV dysfunction, EF 40%	Single Vessel Disease
73	9.1	87	160	3.67	251	9.5	12.1	7,800	27	0.66	141	4.14	107	0.46	0.22	14	15	71	7	4.4	1.30/1.39	108	53	34	198	Normal sinus rhythm	Hypokinesia of inferior wall, inferolateral and inferoapical wall, normal resting LV systolic function, EF 50%	Triple Vessel Disease
74	10.2	89	111	4.01	145	10	13	5,700	16.8	0.91	132	4.34	101	0.5	0.2	28	24	118	7.3	3.8	1.18/1.06	92	71	27	212	Lbbb with first degree heart block	Asynchronous LV movement, normal LV systolic function, no regional wall motion anomaly, EF 60%, sclerotic aortic valve	Single Vessel Disease
75	5.7	82	121	3.76	326	9.1	14.2	6,400	23.7	0.41	138	3.82	112	0.42	0.16	46	52	132	6.8	4.1	1.04/1.12	182	131	36	421	Normal sinus rhythm	Akinesia of anterior wall, anteroseptum, apical septum, apex, mild LV dysfunction EF 45%, thickened aortic valve	Single Vessel Disease
76	5.4	131	177	4.98	216	11.5	12.2	13,300	35.3	0.82	138	4.71	108	1.1	0.62	21	26	88	6.9	3.9	1.45/1.05	140	108	30	95	Anterior wall MI	Akinesia of inferior wall, inferolateral and lateral wall, mild LV dysfunction, EF 45%, severe PAH, grade 3 TR	Triple Vessel Disease
77	11.3	156	191	4.01	274	10.6	14.6	8,400	21	1.15	135	3.67	100	0.61	0.06	25	69	53	6.9	3.9	1.09/1.10	255	182	28	321	Evolved anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum, apex, mild LV dysfunction EF 45%	Double Vessel Disease
78	10	101	145	5.43	193	11.7	14	8,400	23.8	1.57	136	4.55	103	1.21	0.64	43	47	92	7.2	4.4	1.03/0.95	168	88	27	543	LVH with strain pattern	Asymmetrical septal hypertrophy, normal resting systolic function, no regional wall motion anomaly, EF 60%	Double Vessel Disease
79	8.6	88	109	3.35	154	10.8	12.3	10,500	22.1	0.64	139	4.5	102	0.62	0.49	22	27	62	6.8	3.2	1.00/0.98	90	40	45	76	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Triple Vessel Disease
80	6.9	87	102	4.65	280	9.2	12.2	7,200	17.2	0.54	138	3.9	102	0.92	0.37	43	44	106	7.1	4.2	0.97/0.99	102	88	31	152	Strain pattern in lateral leads	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Triple Vessel Disease
81	12.1	242	279	5.43	406	10.2	14.6	12,200	25.2	1.17	132	4.86	96	1.32	0.78	71	58	114	6.5	3.1	1.03/1.19	172	101	27	422	Poor R wave progression	Akinesia of inferior wall, inferoseptum, inferolateral segment, anterior wall, severe LV dysfunction, EF 30%	Triple Vessel Disease
82	8.9	192	252	3.65	160	10.5	16.2	6,900	21.5	0.75	132	4.14	99	0.12	0.07	12	16	78	7	3.9	0.85/0.94	212	168	40	322	Evolved anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum, apex, mild LV dysfunction EF 45%	Double Vessel Disease
83	6.1	96	113	4.22	260	9.11	13.5	9,100	33.3	0.98	137	4.72	104	0.77	0.38	24	28	72	6.9	2.9	0.92/0.94	104	87	29	110	Sinus bradycardia	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Single Vessel Disease
84	9.9	242	250	4.65	308	10.7	13.5	9,900	25.5	0.5	133	3.88	96	0.43	0.28	31	37	142	7.2	4.2	1.16/0.98	89	62	37	111	Strain pattern in anterior leads	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Single Vessel Disease

85	10051015	54	Male	Chest pain radiating to back	Hypertension, Diabetes mellitus	Smoker	Antihypertensives, antidiabetics	Normal	83/min	110/70 mmHg	16/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	7.4	218	282	3.95	644	10	13.1	9,800	30.7	1.19	131	4.73	92	0.92	0.66	26	22	72	6.6	3.6	0.91/1.40	111	72	32	109	Evolved anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum, apex and apicolateral wall, moderate LV dysfunction, EF 40%	Double Vessel Disease
86	10052078	60	Male	Dyspnoea on exertion, chest pain on exertion	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	72/min	100/70 mmHg	26/min	Ejection systolic murmur in aortic area	Air entry equal	Soft, non tender	No focal deficits	6.1	79	112	4.44	266	10.1	13	11,000	18.8	0.81	135	4.03	101	0.59	0.3	8	14	80	7.2	3.9	0.96/1.02	82	66	29	132	LVH with strain pattern	Severe calcific AS, concentric LVH, normal LV systolic function, no regional wall motion anomaly, EF 60%	Triple Vessel Disease
87	10056104	46	Female	Chest pain and dyspnoea on rest	Hypertension, Diabetes mellitus and obstructive sleep apnea	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	95/min	110/70 mmHg	32/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	6.7	107	146	5.45	210	10.7	7.9	11,900	29.6	0.49	142	3.8	104	0.21	0.1	12	16	62	7	4.2	1.04/1.68	168	126	27	190	Anterior wall MI with Rbbb	Akinesia of anterior wall, anteroseptum, apical septum, apex, hypokinesia of inferoseptum, moderate LV dysfunction EF 40%, mild PAH	Single Vessel Disease
88	10056099	58	Male	Chest pain radiating to back	Hypertension, Diabetes mellitus	Smoker	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	100/min	120/80 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	8	189	268	3.75	330	9.32	13.4	11,200	22.8	1.01	136	4.48	102	0.28	0.16	13	21	73	6.6	3.5	0.98/1.08	107	62	29	170	Normal sinus rhythm	Hypokinesia of anteroseptum, preserved LV systolic function, EF 50%, preserved muscle mass	Doudle Vessel Disease
89	10055101	64	Male	Chest pain radiating to back	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	110/min	140/100 mmHg	14/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	5.7	98	131	3.91	281	10.9	14	7,800	10.4	0.99	140	3.71	108	0.52	0.36	25	27	72	6	3.2	1.34/1.05	98	75	31	164	Anterior wall MI	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Single Vessel Disease
90	10056412	61	Female	Chest pain, retrosternal burning	Diabetes mellitus	-	Antidiabetics	Normal	70/min	140/90 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	10.2	212	236	5.12	206	11.9	12.5	8,000	27.8	0.68	131	3.58	101	0.8	0.24	24	30	140	6.7	4	0.88/1.03	217	176	28	414	Evolved inferior wall MI	Akinesia of inferior wall and inferoseptum, inferolateral and lateral wall, moderate LV dysfunction, EF 40%	Triple Vessel Disease
91	10055333	59	Male	Chest pain and sweating	Diabetes mellitus	-	Antidiabetics	Normal	104/min	170/100 mmHg	30/min	S1S2 heard, no murmur	B/L basal crepitations	Soft, non tender	No focal deficits	10.7	282	400	4.43	290	9.8	15.8	9,800	32	0.85	128	4.93	95	0.41	0.27	21	25	82	6.9	3.8	1.07/0.96	195	152	32	212	Strain pattern in anterior leads	Akinesia of anterior wall, anteroseptum, apical septum, apex, mild LV dysfunction, EF 45%	Doudle Vessel Disease
92	10052915	55	Female	Chest pain and sweating	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	92/min	110/70 mmHg	22/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	9.1	188	207	4.32	179	12.2	10.9	8,900	13.7	0.58	134	4.16	98	0.22	0.1	12	10	56	7.2	4.2	0.99/1.07	88	71	29	398	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Single Vessel Disease
93	10049949	65	Female	Dyspnoea on exertion, chest pain	Hypertension	-	Antihypertensives	Normal	77/min	130/80 mmHg	16/min	Ejection systolic murmur in mitral area	Air entry equal	Hepatomegaly	No focal deficits	10.8	104	398	3.75	473	9.6	11.4	8,800	21.6	1.02	133	3.52	98	0.41	0.27	48	32	116	6.7	3.4	1.25/1.07	142	110	36	121	Evolved inferior wall MI	Hypokinesia of inferior wall, inferolateral segment, borderline resting LV systolic function, EF 50%, AML prolapse	Single Vessel Disease
94	10049326	50	Male	Chest pain	Hypertension, Diabetes mellitus and HbsAg positive	Alcoholic	Antihypertensives, antidiabetics	Normal	106/min	170/110 mmHg	20/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	9.8	209	271	4.23	217	10.3	17	7,600	12.5	0.8	138	3.71	102	0.82	0.35	26	42	88	7.1	4.4	1.10/1.09	162	122	35	105	Anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum, apex, mild LV dysfunction, EF 45%	Single Vessel Disease
95	10048342	60	Male	Dyspnoea on exertion, chest pain on exertion	Diabetes mellitus, IHD s/p CABG	-	Antidiabetics, beta blockers, antiocoagulants and lipid lowering agents	Normal	85/min	140/80 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	13.1	352	388	5.21	187	13.1	12.2	6,500	21.4	0.82	136	4.02	104	0.32	0.16	17	15	103	6.8	3.8	1.02/0.82	282	200	32	526	Normal sinus rhythm	Hypokinesia of anterolateral, inferolateral segment, borderline resting LV systolic function, EF 50%, muscle mass preserved	Triple Vessel Disease
96	10045801	65	Female	Chest pain	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	117/min	120/80 mmHg	25/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	7.6	163	202	5.43	368	9.4	10.9	9,000	14.9	0.69	134	4.25	98	0.41	0.21	11	14	62	7.1	4	0.90/1.05	98	65	30	182	Sinus tachycardia	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Triple Vessel Disease
97	10055399	57	Male	Dyspnoea on exertion	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	80/min	130/80 mmHg	20/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	11.2	177	202	3.39	345	9.5	12.7	7,900	32.5	0.79	138	4.19	101	0.26	0.15	9	11	72	7	4.2	1.05/0.99	188	172	34	414	Normal sinus rhythm	Hypokinesia of anterior wall, anteroseptum, apical septum and apex, mild LV dysfunction, EF 45%	Single Vessel Disease
98	10052162	53	Female	Dyspnoea on exertion, chest pain	Hypertension, Diabetes mellitus and anemia	-	Antihypertensives, antidiabetics	Pallor	76/min	120/80 mmHg	22/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	6.3	89	124	4.32	348	9.2	9.7	8,400	48.2	1.11	136	4.23	105	1.1	0.78	43	36	112	6.7	3.6	1.27/1.20	242	198	28	244	Evolved inferior wall MI	Hypokinesia of inferior wall, inferior septum, borderline resting LV systolic function, EF 50%	Single Vessel Disease
99	10052912	67	Male	Dyspnoea on exertion, chest pain on exertion	Diabetes mellitus	Tobacco chewer	Antidiabetics	Normal	70/min	110/70 mmHg	17/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	11.2	101	120	5.14	138	12	13.6	4,200	45.7	1.25	130	3.99	95	0.34	0.18	18	15	67	6.9	4.1	1.21/1.08	187	110	30	321	Evolved anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum, apex, hypokinesia of inferoseptum, moderate LV dysfunction EF 38%, dilated LV	Single Vessel Disease
100	10051695	58	Male	Dyspnoea on exertion	Diabetes mellitus	-	Antidiabetics	Normal	83/min	140/90 mmHg	16/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	7.8	111	145	4.65	129	11.1	14.7	6,900	17.8	0.91	138	4.61	101	0.78	0.45	25	18	116	6.8	3.9	1.13/1.16	101	85	30	164	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Double Vessel Disease
101	10050569	56	Male	Chest pain and sweating	Diabetes mellitus, IHD s/p PTCA and peripheral vascular disease	Smoker	Antidiabetics, beta blockers, antiocoagulants and lipid lowering agents	Normal	72/min	100/70 mmHg	25/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	7.9	98	131	3.47	238	10.1	10.1	3,900	26.7	1.04	138	4.72	109	0.46	0.22	18	14	85	7.1	4.4	1.03/1.09	105	58	39	118	Evolved anterior wall MI with Rbbb	Akinesia of anterior wall, anteroseptum, apical septum, apex, moderate LV dysfunction, EF 40%	Single Vessel Disease
102	10050449	74	Female	Dyspnoea on exertion, palpitations	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	100/min	140/80 mmHg	21/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	7.4	132	173	5.32	192	13.2	12.8	8,400	25.3	0.69	139	4.02	104	0.52	0.26	22	15	111	6.9	3.8	1.10/1.00	206	151	39	343	Sinus tachycardia	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Double Vessel Disease
103	10048828	60	Male	Chest pain and limb claudication	Hypertension, Diabetes mellitus and peripheral vascular disease	Smoker	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	88/min	110/80 mmHg	26/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	9.4	382	545	4.44	238	9.9	11	9,400	35.3	1.1	140	4.83	106	0.21	0.12	25	12	98	6.6	3.4	0.96/1.14	222	184	28	534	Strain pattern in lateral leads	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Double Vessel Disease
104	10044878	59	Male	Dyspnoea on exertion, chest pain on exertion	Diabetes mellitus, IHD s/p PTCA	-	Antidiabetics, beta blockers, antiocoagulants and lipid lowering agents	Normal	70/min	110/70 mmHg	16/min	Ejection systolic murmur in aortic area	Air entry equal	Soft, non tender	No focal deficits	9.1	146	172	5.43	298	12.9	11.5	7,400	24	1.05	138	4.28	104	0.67	0.32	15	21	116	7.2	4.4	0.98/1.05	80	32	35	87	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%, moderate calcific AS	Triple Vessel Disease
105	10043230	70	Female	Dyspnoea at rest	Hypertension, Diabetes mellitus and COPD	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents, inhalers	Pallor	60/min	140/80 mmHg	32/min	S1S2 heard, no murmur	B/l diffuse wheeze	Soft, non tender	No focal deficits	6.4	122	176	5.12	183	11.4	11.3	11,200	19.9	0.72	138	4.18	104	0.39	0.16	25	21	87	6.6	4.2	1.15/0.99	92	46	27	164	Evolved inferior wall MI	Akinesia of inferiolateral and inferior wall, mild LV dysfunction, EF 45%	Double Vessel Disease
106	10055672	75	Male	Dyspnoea on exertion, chest pain	Hypertension, Diabetes mellitus and Neurofibromatosis 1	-	Antihypertensives, antidiabetics	Pallor	75/min	120/70 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	5.9	95	148	4.98	344	10.3	10.4	10,100	16.9	1.11	136	3.35	99	0.46	0.24	34	36	68	5.2	3.3	1.05/1.08	162	84	29	176	Rbbb, strain pattern in lateral leads	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%, concentric LVH	Double Vessel Disease
107	10054238	66	Male	Dyspnoea on exertion, chest pain	Diabetes mellitus, old TB and anemia	-	Antidiabetics	Pallor	120/min	110/70 mmHg	26/min	Ejection systolic murmur in mitral area	Air entry equal	Soft, non tender	No focal deficits	7.5	226	315	3.87	261	9.1	7.4	13,000	36.4	1.07	128	4.33	92	0.26	0.11	14	21	106	6.3	3.6	0.97/1.26	84	43	35	101	Evolved anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum, apex, mild LV dysfunction EF 45%, grade 3 MR	Double Vessel Disease
108	10057082	66	Male	Dyspnoea on exertion, chest pain	Hypertension, Diabetes mellitus and COPD	Tobacco chewer	Antihypertensives, antidiabetics and inhalers	Normal	80/min	100/60 mmHg	16/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	8.7	196	290	3.56	230	9.82	9.8	9,800	57	2.17	136	5.09	105	1.21	0.67	35	44	82	7.1	4.5	0.88/1.21	232	175	41	366	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Triple Vessel Disease
109	10055977	60	Male	Dyspnoea on exertion, chest pain on exertion	Diabetes mellitus	-	Antidiabetics	Normal	72/min	140/80 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	10.5	141	239	5.05	199	10.7	14.4	9,100	13.9	0.89	140	4.13	103	0.94	0.57	26	16	76	6.6	4.2	1.42/0.97	148	110	34	174	Evolved anterior wall MI	Akinesia of inferolateral wall, hypokinesia of anterior wall, anteroseptum, apical septum and apex, mild LV dysfunction, EF 45%	Double Vessel Disease
110	10054601	70	Female	Dyspnoea on exertion, chest pain and sweating	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	98/min	160/80 mmHg	28/min	S1S2 heard, no murmur	B/L basal crepitations	Soft, non tender	No focal deficits	13.4	222	482	5.45	260	12.2	10.4	9,500	33	0.87	134	2.97	95	0.55	0.22	10	27	81	6.7	3.6	0.71/1.08	142	92	37	143	Evolved anterior wall MI	Akinesia of anteroseptum, apical septum, hypokinesia of anterior wall, mild LV dysfunction, EF 45%	Triple Vessel Disease
111	10053518	45	Male	Chest pain	Hypertension, Diabetes mellitus, hypothyroidism and IHD s/p PTCA	Smoker, alcoholic	Antidiabetics, antidiabetics, beta blockers, antiocoagulants and lipid lowering agents, thyroid summplements	Normal	80/min	150/90 mmHg	26/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	15.1	114	190	5.12	266	13.6	12.3	8,200	12.6	0.89	139	3.76	104	0.26	0.11	64	37	62	7.2	4.4	1.10/1.15	321	212	32	528	Normal sinus rhythm	Hypokinesia of inferior wall, inferoseptum, borderline resting LV systolic function, EF 50%	Triple Vessel Disease
112	10052952	63	Male	Chest pain on exertion	-	-	-	Normal	98/min	140/80 mmHg	32/min	Ejection systolic murmur in tricuspid area	B/L basal crepitations and rhonchi	Soft, non tender	No focal deficits	6.2	88	131	4.54	229	10.3	12.6	8,900	20.4	0.64	142	4.2	104	0.45	0.16	14	12	89	6.7	3.2	0.99/1.18	175	120	53	100	Strain pattern in anterior leads	Akinesia of inferior wall and inferolateral segment, mild LV dysfunction, EF 45%, grade 3 TR	Double Vessel Disease
113	10053462	74	Male	Chest pain on exertion	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	72/min	150/80 mmHg	15/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	6.4	178	209	4.76	295	9.2	10.6	5,800	35.8	1.41	136	4.31	105	0.21	0.12	18	14	65	6.8	3.2	0.79/0.96	114	96	30	148	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%, concentric LVH	Double Vessel Disease
114	10052051	70	Female	Chest pain, vomiting	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	96/min	100/70 mmHg	18/min	Ejection systolic murmur in aortic area	Air entry equal	Soft, non tender	No focal deficits	7.7	126	187	5.12	280	11.6	13.4	11,600	26.1	1.23	136	3.01	90	0.87	0.45	36	28	87	6.9	4.1	1.28/0.97	90	54	29	111	Postero lateral wall MI with Rbbb	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%, MVP AML prolapse, grade 2 MR	Double Vessel Disease
115	10052058	60	Female	Chest pain radiating to back	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	92/min	170/80 mmHg	25/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	10.7	92	137	3.74	309	10.6	10.2	8,800	17	0.76	136	3.45	98	0.3	0.17	42	51	78	7.6	4.2	1.03/1.00	233	176	33	365	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%, concentric LVH	Triple Vessel Disease
116	10051324	49	Male	Dyspnoea on exertion	Diabetes mellitus and diabetic nephropathy	-	Antidiabetics	Pallor	64/min	140/80 mmHg	20/min	S1S2 heard, no murmur	B/L basal crepitations	Soft, non tender	No focal deficits	5.9	101	156	3.12	240	9.7	16.5	10,800	13.79	2.15	138	4.68	103	1.1	0.64	26	29	101	7	4.1	1.20/0.93	74	48	35	212	Strain pattern in anterior leads	Global hypokinesia of LV, mild LV dysfunction, EF 45%	Double Vessel Disease
117	10051803	67	Female	Dyspnoea on exertion, chest pain	Hypertension, Diabetes mellitus and hypothyroidism	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents, thyroid supplements	Normal	86/min	150/80 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	7.1	81	136	4.23	227	11.2	12	8,700	23.1	0.72	137	4.8	102	0.32	0.17	14	12	75	6.5	3.7	0.90/0.96	156	101	31	200	Evolved anterior wall MI	Hypokinesia of apical septum, apex, borderline resting LV systolic function, EF 50%, muscle mass preserved	Single Vessel Disease
118	10050571	73	Male	Dyspnoea on exertion, chest pain	Hypertension, IHD s/p PTCA	-	Antihypertensive, beta blockers, anticoagulants and lipid lowering agents	Normal	100/min	100/70 mmHg	24/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	6.3	126	140	4.65	327	11	16.9	19,100	28.8	1.26	135	3.66	96	0.75	0.27	59	77	89	6.8	3.8	1.21/1.44	246	190	48	94	Anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum, apex, apicolateral segment, moderate LV dysfunction, EF 35%	Triple Vessel Disease
119	10050716	69	Male	Chest pain and sweating	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics, anticoagulants and lipid lowering agents	Normal	70/min	140/90 mmHg	18/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	11.8	227	352	3.79	205	10.9	11.1	6,400	30.8	1.71	134	4.91	102	0.44	0.23	10	40	95	5.9	3.6	0.96/0.99	182	148	38	94	Inferior wall MI	Hypokinesia of inferior wall, inferolateral segment, borderline resting LV systolic function, EF 50%	Single Vessel Disease
120	10050820	55	Male	Dyspnoea on exertion	Diabetes mellitus and RHD s/p MVR	-	Antidiabetics, beta blockers, antiocoagulants and lipid lowering agents	Normal	170/min	130/90 mmHg	28/min	S1S2 heard, no murmur	B/L basal crepitations	Soft, non tender	No focal deficits	16.2	398	428	5.42	159	13.1	10.5	4,400	20.6	0.8	131	3.67	95	0.8	0.32	21	36	114	7.8	4.3	1.29/1.15	233	176	41	367	Atrial fibrillation	Post MVR, LA dilated, global hypokinesia of LV, moderate LV dysfunction, EF 35%, RV dysfunction with moderate AR	Double Vessel Disease
121	10050653	85	Female	Dyspnoea and chest pain at rest	Diabetes mellitus	-	Antidiabetics	Normal	102/min	140/90 mmHg	30/min	Ejection systolic murmur in mitral area	Air entry equal	Soft, non tender	No focal deficits	7.4	96	165	4.12	278	8.5	11.5	9,500	17.6	0.8	136	4.25	102	0.56	0.32	18	15	62	6.6	3.2	1.05/1.05	101	78	30	211	Evolved anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum, apex and apicolateral wall, moderate LV dysfunction, EF 40%, grade 3 MR	Single Vessel Disease
122	10050699	72	Female	Chest pain	Diabetes mellitus	-	Antidiabetics, antiocoagulants and lipid lowering agents	Normal	62/min	110/70 mmHg	14/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	9	158	205	5.32	225	12.6	11	11,000	37.2	1.37	133	4.24	102	0.67	0.28	11	19	99	7.4	4.2	1.14/0.96	274	178	29	488	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Triple Vessel Disease
123	10047930	69	Female	Dyspnoea on exertion	Hypertension, Diabetes mellitus	Tobacco chewer	Antihypertensives, antidiabetics	Normal	110/min	110/70 mmHg	22/min	Pansystolic murmur in mitral area	Air entry equal	Soft, non tender	No focal deficits	6.7	98	137	3.81	396	10.4	10.1	13,100	21.9	1.38	131	3.82	92	0.36	0.14	42	36	78	7.5	4.1	0.83/1.14	101	78	33	94	Evolved anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum, apex, apicolateral segment, severe LV dysfunction, EF 30%, severe MR	Triple Vessel Disease
124	10049313	58	Male	Chest pain	Hypertension, Diabetes mellitus	Smoker, alcoholic	Antihypertensives, antidiabetics	Normal	102/min	140/80 mmHg	26/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	8	136	205	5.1	204	10.7	12.9	15,100	30.6	1.54	133	3.73	96	1.32	0.78	26	22	101	6.9	3.8	1.88/1.29	152	107	35	151	Anterior wall MI	Akinesia of anterior wall, anteroseptum, apical septum and apex, mild LV dysfunction, EF 45%	Double Vessel Disease
125	10049689	58	Male	Chest pain radiating to back	Hypertension, HbSAg and RVD positive	Smoker, alcoholic	Antihypertensives, ART regimen	Normal	70/min	160/90 mmHg	20/min	S1S2 heard, no murmur	Air entry equal	Hepatomegaly	No focal deficits	7.9	106	148	4.16	251	9.2	13.9	7,900	20.2	0.9	137	4.41	102	0.41	0.16	12	11	78	7.8	4.5	1.06/0.94	123	78	21	334	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Single Vessel Disease
126	10058925	63	Female	Dyspnoea on exertion	Hypertension, Diabetes mellitus and IHD s/p PTCA	-	Antihypertensives, antidiabetics, beta blockers, anticoagulants and lipid lowering agents	Normal	64/min	160/100 mmHg	26/min	Ejection systolic murmur in tricuspid area	Air entry equal	Soft, non tender	No focal deficits	12.2	173	226	5.1	124	11.7	11.5	4,800	24.5	0.94	139	4.32	105	0.89	0.53	45	38	132	6.6	3.8	1.06/1.10	92	74	30	228	Strain pattern in anterior leads	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%, Grade 3 TR, moderate PAH	Single Vessel Disease
127	10056012	53	Female	Chest pain on exertion	Hypertension, Diabetes mellitus	-	Antihypertensives, antidiabetics	Normal	95/min	160/90 mmHg	28/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	6.3	90	114	3.72	334	8.8	11.4	7,600	15.6	0.71	138	4.07	103	0.34	0.15	12	19	123	7	4.2	1.02/0.93	110	82	26	339	Strain pattern in inferior leads	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Triple Vessel Disease
128	10051934	58	Male	Chest pain on exertion	Hypertension, Diabetes mellitus	Smoker, alcoholic	Antihypertensives, antidiabetics, beta blockers, anticoagulants and lipid lowering agents	Normal	66/min	110/70 mmHg	16/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	6.7	89	152	3.93	185	9.6	11.8	4,300	18	1.19	136	4	102	0.22	0.09	16	28	72	6.5	3.2	0.85/1.00	225	173	46	384	Normal sinus rhythm	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Double Vessel Disease
129	10058533	49	Male	Chest pain on exertion	Diabetes mellitus, IHD s/p PTCA and peripheral vascular disease	Smoker, alcoholic	Antidiabetics, beta blockers, antiocoagulants and lipid lowering agents	Pallor	88/min	120/80 mmHg	14/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	7.1	103	148	4.4	260	10.6	13	7,400	17.6	1.1	131	4.36	96	0.51	0.18	61	40	105	7.3	4.4	1.03/1.14	101	82	28	252	Strain pattern in anterior leads	Akinesia of anterior wall, anteroseptum, apical septum, apex, hypokinesia of apicolateral, anterolateral segment, moderate LV dysfunction, EF 35%	Double Vessel Disease
130	10058557	73	Male	Chest pain and sweating	Hypertension, Diabetes mellitus and IHD s/p PTCA	-	Antihypertensives, antidiabetics, beta blockers, anticoagulants and lipid lowering agents	Normal	130/min	140/90 mmHg	30/min	S1S2 heard, no murmur	Air entry equal	Soft, non tender	No focal deficits	7.9	112	183	4.12	200	10.2	11.4	11,700	40.5	1.46	137	5.04	104	0.75	0.46	26	12	62	7.6	4.3	1.07/0.87	93	44	34	158	Strain pattern in lateral leads, trifascicular block	Normal resting LV systolic function, no regional wall motion anomaly, EF 60%	Double Vessel Disease