“COMPARATIVE STUDY ON PLATELET INDICES IN PATIENTS WITH CARDIAC AND NON CARDIAC CHEST PAIN”

By

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Dissertation submitted to BLDE University, Vijayapur

In partial fulfilment of the requirements for the award of the degree of

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

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Dr. SHIVANAND MULAKURI

Abstract

Platelets play a central role in the pathogenesis of acute coronary syndromes (ACS) and high MPV has been associated to more reactive platelets are regarded as an independent risk factor for myocardial infarction. The aim of the study is to study the role of platelet indices in patient with Acute Coronary Syndrome (ACS).

Materials and methods:

In the present study 100 patients who presented to ShriB.M.Patil Hospital with Acute Coronary Syndromes (ACS) including Acute STEMI, NSTEMI and Unstable angina were included. Platelet indices including Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW) were calculated in all these patients at the time of admission through automated analyzer. These parameters were compared with 100 age and sex matched controls.

Results:

The mean age of the study population is 56.3 years. The MPV for the control group in our study was 10.3±1.5fL, whereas for STEMI 11.5±2.5fL, NSTEMI 10.8±1.3fL and for Unstable Angina it was 10.6±1.7fL.The mean of PDW for the control group in our study was 10.2±1.7fL, whereas for STEMI 11.7±2.1fL, NSTEMI 11.0±1.4fL and for Unstable Angina it was 11.1±2.0fL. There was a significant increase in MPV & PDW in patients with ACS. Patients with ACS had higher values of MPV and PDW when compared to age matched controls. (p=0.0001).

Conclusion:

Larger platelets are haemostatically more active and are a risk factor for developing coronary thrombosis and subsequent acute coronary event (Acute STEMI/NSTEMI). Platelets with higher MPV and PDW were at higher risk of ACS. These patients can easily be identified during routine haematological analysis and could possibly benefit from preventive treatment.

ABBREVATIONS

ACS	Acute coronary syndrome
ATP	Adenosine Triphosphate
ADP	Adenosine Diphosphate
AKI	Acute kidney injury
AMI	Acute myocardial infarction
ARR	Absolute risk reduction
CAD	Coronary artery disease
CI	Confidence interval
CK	Creatine kinase
CKD	Chronic kidney disease
COX-1	Cyclooxygenase 1
CRP	C-reactive protein
CV	Coefficient of variation
CVD	Cardiovascular disease
ECG	Electrocardiography
ECHO	Echocardiography
ECM	Extracellular matrix
GFR	Glomerular filtration rate
GP	Glycoprotein
HR	Hazard ratio
ICAM-1	Intercellular adhesion molecule-1
ICU	Intensive care unit
IL	Interleukin
LDL	Low density lipoprotein
LMWH	Low molecular weight heparin
MPV	Mean Platelet Volume

mRNA	messenger ribonucleic acid
NO	Nitric oxide
NSTEMI	Non ST-elevation myocardial infarction
OR	Odds ratio
PCI	Percutaneous coronary intervention
PAR	Protease activated receptor
PDW	Platelet Distribution Width
PPP	Platelet poor plasma
PRP	Platelet rich plasma
STEMI	ST-elevation myocardial infarction
TXA2	Thromboxane A2
UA	Unstable angina
vWF	von Willebrand factor
VCAM-1	Vascular cell adhesion molecule-1

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INTRODUCTION

INTRODUCTION

Chest pain is one of the most common complaints among the patients presenting to emergency department. The acute chest pain is most commonly caused due to cardiac, gastroesophageal, musculoskeletal, and pulmonary conditions. History and physical examination are the most important in assessing a patient presenting with acute chest pain. It is essential to obtain the characteristics of the pain such as site, onset, duration, radiation, quality and other associated signs.

Cardiac causes of acute chest pain are classified as ischemic and non-ischemic. Most common ischemic cause is coronary artery disease. Other causes include coronary artery spasm, aortic stenosis, and hypertrophic cardiomyopathy. Pericarditis andValvular diseasesare common non-ischemic causes.

In the patients undergoing evaluation for acute chest pain in the emergency departments, about 10% to 30% have acute Myocardial infarction or unstable angina. Life-threatening problems include acute aortic dissection and pulmonary embolism, but most of the patients remain undiagnosed or leave the emergency department with a diagnosis of a non-cardiac chest pain.[1]

Angina is the classic manifestation of ischemia; it is described as chest pain with heaviness or squeezing, crushing, burning sensation, or difficulty in breathing. The pain often radiates to the neck, left shoulder or arm. It increases in intensity over few minutes. Onset of pain may be associated with physiological stress or exercise but often there is no obvious precipitating factor.

Angina pectoris is classically defined as chest pain of cardiac origin due to imbalance in the myocardial oxygen demand and supply. Myocardial ischemia

commonly occurs in the presence of coronary atherosclerosis. Spasm of the coronary vessels can occur even in normal coronary arteries. Other causes of impaired coronary blood flow include coronary arteritis, coronary emboli from thrombus in the left atrium or left ventricleor a congenital abnormality of the coronary arteries.

Atypical descriptions of chest pain decrease the possibility of myocardial ischemia.“The American College of Cardiology (ACC) and American Heart Association (AHA) guidelines” suggest the following descriptions of pain as non-characteristic of myocardial ischemia:[2]Pleuritic pain (sharp pricking or stabbing pain increases on inspiration or coughing).Pain or discomfort in the hypogastric region.Pain that is well localized by the tip of the finger.Pain occurring with movement or on palpation of the chest wall or arms.Short episodes of pain lasting a few seconds or less.Pain radiating into the lower extremities.

The “angina equivalents” such as jaw or shoulder pain in the absence of chest pain or dyspnoea, nausea or vomiting, and diaphoresis may be associated with cardiac causes.Individuals with diabetes,women, and elderly individuals may present with atypical symptoms.

Pericardial Disease –Most of the parietal and whole of the visceral pericardium is insensitive to pain. Pericarditis almost always involves the surrounding pleura, and patients may present with characteristic pleuriticpain. Pericarditis may produce a substernal painwhich is crushing type and similar to that of acute MI.[3]

Vascular Disease - Sudden onset of excruciating tearing chestpainis seen in acute aortic dissection, the location of painreflects the type of dissection. Dissectionof the ascending aortic manifests as pain in the anterior aspect of the chest, and posterior descending aortic dissection causes pain in the back. Pulmonary embolismcauses a

sudden onset of pleuritictype of chest pain and dyspnoea. Massive pulmonary embolism causes severesubsternal pain, which is due to the pulmonary arterydistention. Smaller emboli can cause pulmonary infarction and present with pleuritic chest pain over lateral aspect and haemoptysis.

Pulmonary Conditions- Pulmonary conditions causing chest pain generally produce dyspnoea and pleuritic symptoms. Pneumonia causes pain over the involved lung. In pneumothorax pain begins suddenly and is usually associated with dyspnoea.

Gastrointestinal Conditions - Irritation of the oesophagus due to acid reflex produces burning sensation that increases with alcohol, aspirin, and some foods. Squeezing chest pain and similar to that of angina is seen in oesophageal spasm. Chest pain in peptic ulcer disease occurs usually one to two hours after meals and responds rapidly to antacid therapies. Most commonly pain is located in the epigastric region but can radiate to the chest and shoulders. Pancreatitis causes severe, aching epigastric pain that radiates to the back.

Musculoskeletal causes - Musculoskeletal disorders of chest wall such as costochondritis, or involvement of the nerves of the chest wall as seen in herpes zoster and Pancoasttumour, or following heavy exercise and trauma are most common causes of chest pain. Chest pain secondary to musculoskeletal causes is well localised and can be elicited by applying pressure over the affected area or by movement of the patient’s neck.

AIMS AND OBJECTIVES

AIM OF THE STUDY

To study association of Platelet Indices viz., Mean Platelet Volume, and Platelet Distribution Width in Patients with cardiac and non-cardiac chest pain.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

MYOCARDIAL ISCHEMIA:

Myocardial function is related to the coronary blood flow and oxygen delivery. For the normal functioning of the heart, balance between oxygen supply and demand is important. Diseases that affect the coronary blood flow result in balance and can lead to ischemia induced dysfunctionof the contraction and can cause hypotension which further worsens myocardial ischemia. The oxygen extraction by myocardium is maximal at rest, averaging about 60% to 85% of arterial oxygen content.[9]

As the oxygen supply to the heart is closely related to coronary blood flow, after a thrombotic coronary occlusion there is sudden cessation of perfusion immediately leads the onset of anaerobic glycolysis. As ischemia continues, acidosis develops in the tissue and there is an efflux of potassium into the extracellular space. The ATP levels fall below the required level necessary to maintain critical cell function, resulting in the onset of myocyte death.

The extents of irreversible tissue injury after coronary occlusion are variable and depend on location of thrombus, residual coronary flow, and theoxygen consumption. In the absence of significant collaterals after 20 minutes of coronary occlusionirreversible myocardial injury occurs. Irreversible injurystarts in the sub endocardium and progresses over time, from sub endocardial layers to the subepicardial layers.

ACUTE CORONARY SYNDROMES:

Ischemic Heart Disease is a condition associated with inadequate supply of blood and oxygen to the myocardium. The most common cause of myocardial ischemia is atherosclerosis of coronary vessels causing reduction in myocardial blood flow and decreased perfusion of myocardium.

In Acute Coronary Syndrome, the most important predisposing factor is the plaque disruption or acute plaque change in the atherosclerotic vessel which may present as

- Unstable angina
- ST Elevation Acute Myocardial Infarction
- Non ST Elevation Myocardial infarction.

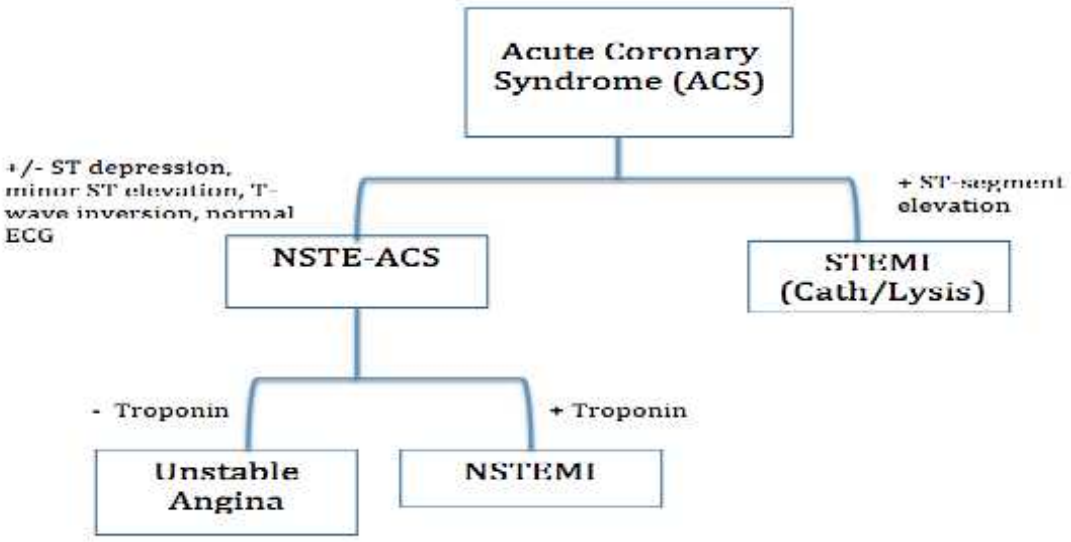

Figure 1: Classification of Acute Coronary syndrome

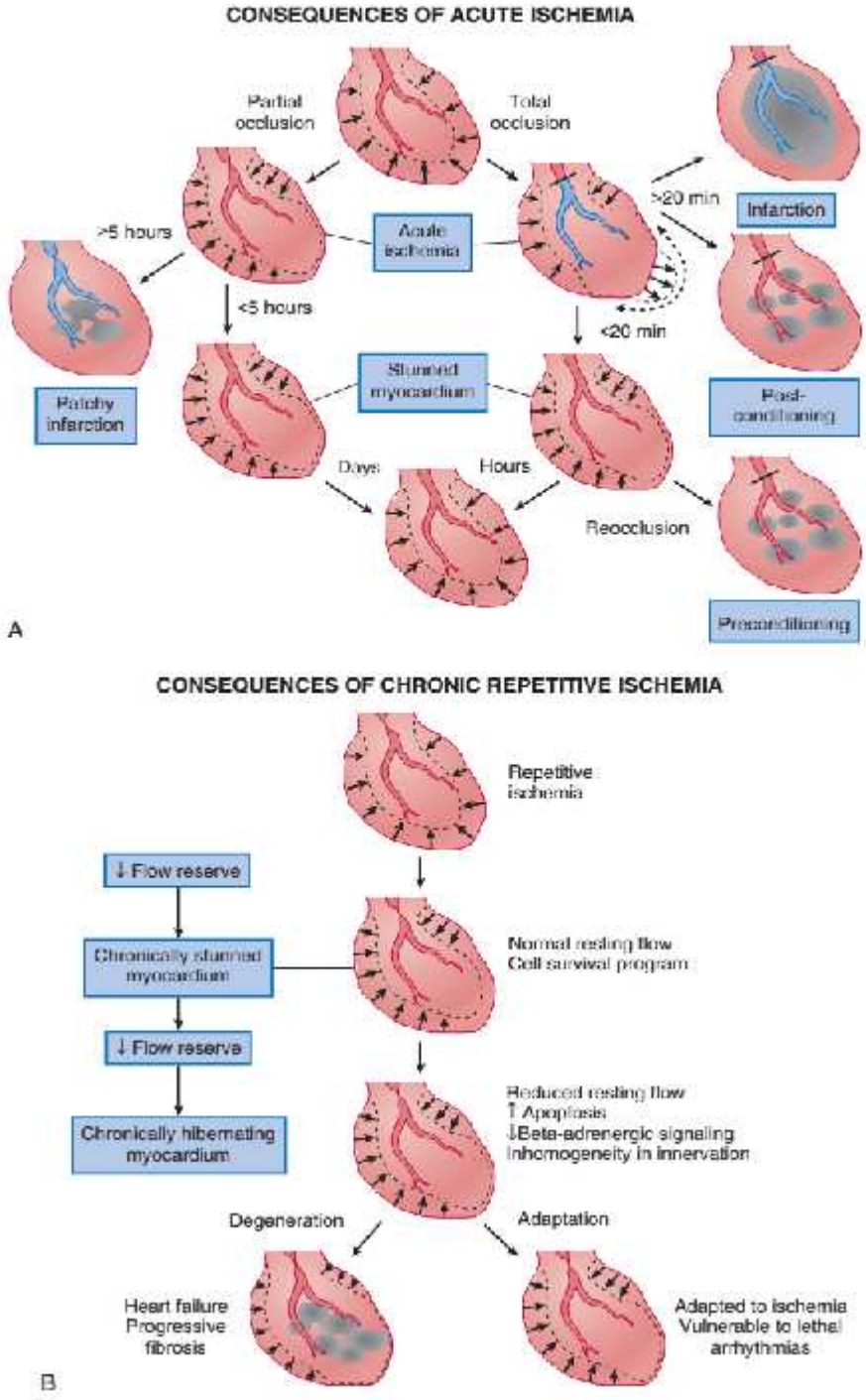

Figure 2: Consequences of Myocardial Ischemia

EFFECTS OF ACUTE ISCHEMIA:

A short period of complete occlusion or a prolonged partial occlusion leads to acute contractile dysfunction proportional to the reduction in blood flow. After 20 minutes of total occlusion irreversible injury begins but is delayed for up to 5 hours after a partial occlusion or in presence of significant collaterals.[10]Whenperfusion is re-established before the onset of irreversible injury, it leads to stunned myocardium, and the time required for recovery of function is proportional to the duration and severity of ischemia. With prolonged ischemia, stunning coexists with sub endocardial infarction and leads to for an increased amount of irreversible dysfunction. Experimental infarct size can be reduced by cardio protective mechanisms. Intermittent occlusion at the time of reperfusion can limit infarct size.[11]

Myocardial function returns to normal after single episode of ischemia lasting less than 2 minutes. As the duration and severity of ischemia increases, a delay in the recovery of function occurs despite the blood flow has been restored. Following a 15-minute occlusion in the absence of tissue necrosis regional myocardial function remains depressed for up to 6 hours even after resolution of ischemia, a phenomenon called myocardial stunning.[12]The characteristic feature of isolated myocardial stunning is that function remains depressed while resting myocardial perfusion is normal.[13]

RISK FACTORS FOR CORONARY HEART DISEASE:

The aetiology of coronary heart disease is multifactorial. Some risk factors are modifiable whereas others non modifiable. Presence of any one of the risk factors places an individual at risk of developing heart disease.

Modifiable risk factors	Non Modifiable risk factors
Overweight/Obesity Hypertension Hyperlipidaemia Smoking Stress Sedentary lifestyle	Increasing Age Gender Family history Genetic Menopause

Table 1: Risk factors for coronary artery diseases

AGE:

Age is a non-modifiable risk factor with a dominant influence on heart diseases. Atherosclerosis although progressive, rarely manifests clinicallyuntil middle age or later. The incidence of myocardial infarction increases between ages 40 to 60 years.[14]

GENDER:

Compared to age matched menpremenopausal women are relatively protected against atherosclerosis and its consequences. However after menopause the incidence of coronary heart diseases increases. A favourable influence of oestrogen has long been proposed to explain the protective effect.[15]

ATHEROSCLEROSIS:

It can affect any artery in the body. When it occurs in the heart, it causes angina myocardial infarction and sudden death. It begins early in life. Abnormal

arterial function has been found among high risk children and adolescents with familial hyperlipidaemia, smokers and those with hypertension.

SMOKING:

Smoking is one of the most important risk factor for coronary artery disease. Smokers lose at least one decade of life expectancy, as compared with never-smokers.[17]The risk of death from cigarette smoking continues to increase among women and the increased risks are now nearly identical for men and women.[18]Compared with non-smokers, smoking increases the risk of both coronary heart disease and stroke two- to fourfold. Ischemic heart disease underlies 35% to 40% of all smoking-related deaths, with an additional 8% attributable to second-hand smoke exposure. Second-hand smoke exposure also is associated with heart disease in non-smoking adults. Non-smokers exposed to second-hand smoke at home orwork is at increased risk for heart diseases by 25% to 30%. Second-hand smoking has harmful effects on the cardiovascular system that can increase the risk of heart attack, especially among those who already have heart disease.

HYPERTENSION:

Elevated blood pressure is a major risk factor for coronary heart disease, heart failure, cerebrovascular disease, peripheral arterial disease, renal failure, atrial fibrillation, and total mortality, the degree of blood pressure lowering relates linearly to risk reduction. Observational data indicate that death from both coronary heart disease and stroke increases progressively from blood pressure levels as low as 115mmHg systolic and 75mmHg diastolic. Among patients of 40 to 70 years of age, each increment of 20mmHg in systolic or 10mmHg in diastolic blood pressure doubles the risk of cardiovascular disease across a blood pressure range of 115/75 to

185/115mm Hg. Prehypertension, defined as systolic blood pressure between 120 to 139 mm Hg or diastolic blood pressure between 80 to 89 mm Hg, is associated with nearly twice the risk of MI and stroke in women compared with normal blood pressure.[19]

LOW-DENSITY LIPOPROTEIN (LDL) CHOLESTEROL:

Of plasma-based atherothrombotic risk factors, LDL cholesterol is the best-established risk factor causally linked to incident MI and cardiovascular death. Mutations in the LDL receptor that produce hypercholesterolemia leads to accelerated atherosclerosis as early as the first decade of life in patients with homozygous familial hypercholesterolemia, while those with heterozygous hypercholesterolemia develop disease approximately 10 to 15 years later. Other recently described mutations that affect LDL metabolism, such as those in the enzyme proproteinconvertasesubtilisin/kexin type 9 (PCSK9), result in life-long reductions in LDL cholesterol and reduced lifetime risks of events.[20]

HIGH-DENSITY LIPOPROTEIN (HDL) CHOLESTEROL:

Abundant prospective epidemiologic data demonstrates a strong inverse relationship between HDL cholesterol and vascular risk. In general, observational data suggest that each incremental increase in HDL cholesterol of 1 mg/dl is associated with a 2% to 3% decrease in risk of total cardiovascular disease. The process of reverse cholesterol transport contributes to the protective role of HDL against coronary death. HDL carries cholesterol from the vessel wall, augmenting peripheral catabolism of cholesterol. HDL also carries antioxidant enzymes that reduce the levels of oxidized phospholipids in atheromatous lesions. HDL also may have anti-inflammatory properties and promote cholesterol efflux from macrophages.[21]

TRIGLYCERIDES:

Plasma triglycerides are primarily produced in the intestines and within the liver. Metaanalyses suggest that the adjusted risk ratio for coronary disease among patients with triglyceride levels in the top third of reported values compared with those in the bottom third decreased from approximately 2.0 to 1.5 after accounting for HDL cholesterol.[22]

DIABETES MELLITUS:

Diabetes markedly increases the risk of atherosclerosis. Insulin resistance and diabetes rank among the major cardiovascular risk factors. As compared with age- and ethnically matched non-diabetic subjects patients with diabetes have twofold to eightfold increased rates of future cardiovascular events, and 75% of all deaths in diabetic patients result from coronary heart disease. Diabetic patients have a greater atherosclerotic burden in the major arteries, as well as of microvasculardisease as compared with unaffected persons. Insulin resistance alone leads to an increased risk of congestive heart failure. The analysis of data from the Nurses' Health Study among women who eventually developed type 2 diabetes, the relative risk of MI was elevated threefold than before the diagnosis of diabetes.[23]

HIGH-SENSITIVITY C-REACTIVE PROTEIN (Hs-CRP):

A series of large-scale prospective cohorts conducted worldwide indicated that CRP, when measured with high-sensitivity assays (hsCRP), independently predicts risk of MI, stroke,peripheral arterial disease, and sudden cardiac death among apparently healthy persons, even when LDL cholesterol levels are low.[24]

HOMOCYSTEINE:

Homocysteine is a sulfhydryl-containing amino acid derived from the demethylation of dietary methionine. In patients with rare inherited defects of methionine metabolism, severe hyperhomocysteinemia (plasma levels higher than 100 mmol/litre) can develop; such patients are at markedly elevated risk for premature atherothrombosis as well as venous thromboembolism.

CARDIAC BIOMARKERS

Patients presenting with chest pain or discomfort consistent with ACS should undergo measurement of biomarkers for myocardial injury. The preferred biomarker is cardiac troponin (cTnT or cTnI); creatine kinase MB isoenzyme (CK-MB) is less sensitive.[25]

TROPONINS:

Troponins I and T in cardiac muscleare encodedby different genes. The assays for cardiac troponins are more specific and sensitive than CK-MB for myocardial injury, and cardiac troponin is the preferred diagnostic biomarker.[26]False-positive elevations are rarely seen as it is highly specific for myocardium. Damage occurring with other forms of myocardial injury, such as in the setting of myocarditis, or extreme exercise; right ventricular strain from pulmonary embolism[27]; or other causes of acute pulmonary hypertension may be associated with elevation of enzymes. Elevated levels of cardiac troponins are also seen in patients with renal disease.[28]with testing for up to 12 hours after arrival to thehospital; cardiac troponins have sensitivity higher than 95% and a specificity of 90%. Single sample at initial

evaluation results in substantially worse performance, with a sensitivity of just 70% to 75%.

HIGH-SENSITIVITY CARDIAC TROPONIN:

The term high-sensitivity troponin (hsTn) is reserved for assays that can detect cardiac troponin in more than 50% of an apparently healthy population.Such assays are substantially more sensitive than previous generation assays but also have diminished clinical specificity for MI because they detect true myocardial injury in a variety of other clinical settings. The sensitivity for detecting MI using single sample at initial evaluation is approximately 90%, the specificity is approximately 90%, and the negative predictive value is approximately 97% to 99%.[29-30]In patients presenting within 3 hours of the onset of chest pain, such sensitive assays have a sensitivity of 80% to 85% versus approximately 55% for older assays. High-sensitivity assays with even lower limits of detection (e.g., <0.001 ng/mL), are currently in development, they allow at least 50% (some ≥95%) of healthy individuals below the 99th percentile to have a measurable level of troponin.[31] When such assays were performed in patients with non–ST-segment elevation MI (NSTEMI), 72% had circulating troponin levels at baseline above the 99th percentile and the other 28% had levels above the limit of detection. In patients with unstable angina, 44% had circulating troponin levels above the 99th percentile, and another 52% had levels above the limit of detection at baseline; 6 to 8 hours later, these values were 82% and 18%, respectively.[32]

CREATINE KINASE-MB:

CK-MB was the biomarker of choice for the diagnosis of MI before the use of cardiac troponin. It is found in skeletal muscle, tongue, diaphragm, small intestine,

uterus, and prostate. Its major limitation is its relative lack of specificity.The ratio of CK-MB to total CK (CK-MB relative index)increases sensitivity.If a cardiac-specific troponin assay is not available, CK-MB measured with a mass assay is the best alternative. Cardiac muscle contains both the MM and MB isoenzymes of CK. The advantage of CK-MB is it hasshorterhalf-life in the circulation, hence makes it useful for measuring the timing of an MI and for diagnosing reinfarction in a patient who has experienced an MI in the past week.

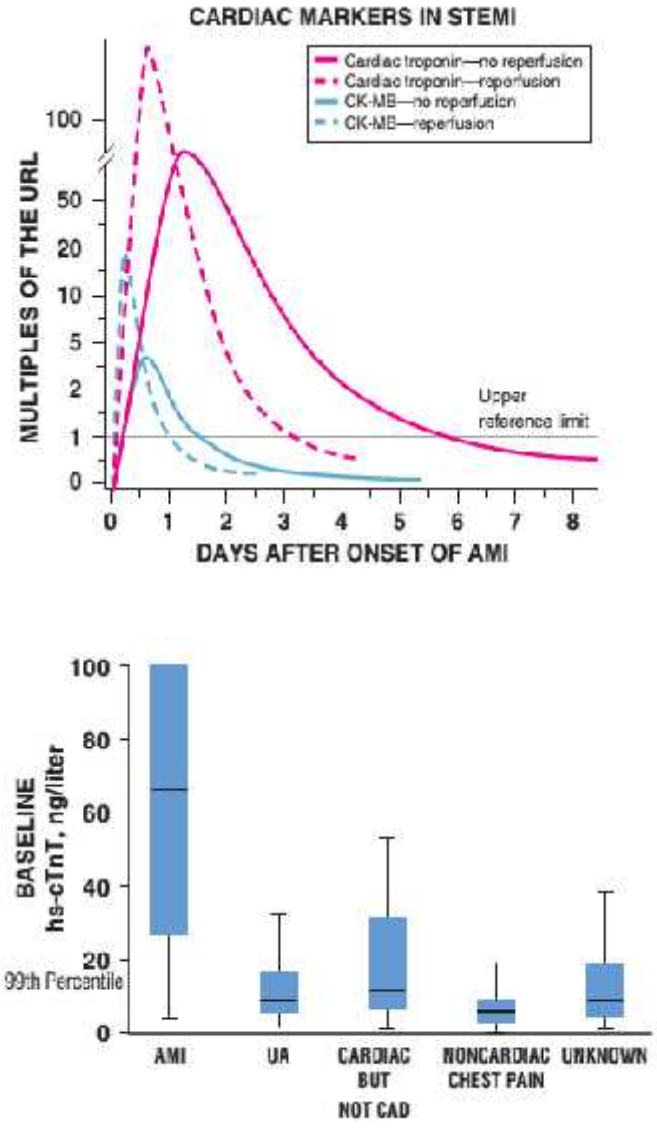

Figure 3: Cardiac Biomarkers CPK-MB and Troponin T

ELECTROCARDIOGRAPHY

An ECG should be done for patients within 10 minutes after arrival with ongoing chest discomfort and as soon as possible for patients with history of chest discomfort consistent with ACS but whose discomfort has resolved.[33]Aprehospital ECG decreases the door-to-diagnosis time and, for ST-segment elevation MI (STEMI), the door-to-balloon time. Transient or new persistent ST-segment abnormalities (≥0.05 mV) that develop during a symptomatic episode at rest and resolve when the symptoms subside strongly suggest acute ischemia and severe coronary disease. Lesser amounts of ST-segment deviation or T wave inversion of 0.2 mV or lessare usually defined as Nonspecific ST-segment and T wave.

Table 2: Electrocardiographic manifestations of Myocardial Infarction

Manifestation	Characteristics
ST Elevation	New ST elevation at the J point in two contiguous leads with the cut-points: ≥0.1mV in all leads other than leads V2-V3 where the following cut points apply: ≥0.2mV in men ≥40 years; ≥0.25mV in men <40 years, or ≥0.15mV in women.
ST depression and T wave Changes	New horizontal or down-sloping ST depression ≥0.05mV in two contiguous leads and/or T inversion ≥0.1mV in two contiguous leads with prominent R wave or R/S ratio >1.
Associated with prior Myocardial infarction	Any Q wave in leads V2-V3 ≥0.02s or QS complex in leads V2 and V3. Q wave ≥0.03s and ≥0.1mV deep or QS complex in leads I. II, aVL, aVF, or V4-V6 in any two leads. R wave ≥0.04s in V1-V2 and R/S ≥1 with a concordant positive T wave in the absence of conduction defect.

PLATELETS: OVERVIEW

Platelets are non-nucleate fragments, minute discs with a diameter of 1 to 4 mm that are released by the bone marrow megakaryocytes into the circulation and areprimarily responsible for the maintenance of vascular integrity and haemostasis. They are formed from megakaryocytes in the bone marrow, which are extremely large cells. Thesemegakaryocytes fragment into minute platelets either in the bone marrow or soon after entering the blood, as they squeeze through the capillaries. The normal concentration of platelets in the blood is between 1,50,000 and 3,00,000 per microliter. Glycoproteinspresent on its surface prevents adherence to normal endothelium but causes adherence to injured areas of the vessel wall, to injured endothelial cells or to exposed collagen from deep within the vessel wall. The platelet membrane also contains large amounts of phospholipids that play a role in various stages in the blood-clotting process.

Platelets play major role in haemostasisand providing rapid protection against bleeding and formation of stable blood clots via the coagulation cascade by forming a haemostatic plug that seals vascular defects, and by providing a surface that recruits and concentrates activated coagulation factors.They also protect from infection by phagocytosis and by secreting chemokines and attract leukocytes. Platelet function is commonly assessed by platelet count, bleeding time, mean platelet volume and platelet distribution width.

PLATELET STRUCTURE AND FUNCTIONS:

Platelet Adhesion: Platelet adhesion initiates clot formation and depends on von Willebrand Factor and platelet glycoprotein Gp1b. Under shear stress, vWFundergoes

a conformational change, assuming an extended shape that allows it to bind simultaneously to collagen in the ECM and to platelet Gp1b.

Platelet Activation: Platelet adhesion leads to an irreversible change in shape and secretion of granules —a process termed platelet activation. Calcium and ADP released from δ granules are especially important in subsequent events, since calcium is required for several coagulation factors and ADP is a potent activator of resting platelets. Activated platelets also synthesize thromboxane A2 (TxA2), a prostaglandin that activates additional nearby platelets and that also has an important role in platelet aggregation (described below). During activation, platelets undergo a change in shape from smooth discs to spheres with numerous long, spiky membrane extensions, as well as more subtle changes in the make-up of their plasma membranes. The shape changes enhance subsequent aggregation and increase the surface area available for interaction with coagulation factor.

Platelet Aggregation: Platelet aggregation follows platelet adhesion and activation, and is stimulated by some of the same factors that induce platelet activation, such as TxA2. Aggregation is promoted by bridging interactions between fibrinogen and GpIIb/IIIa receptors on adjacent platelets.

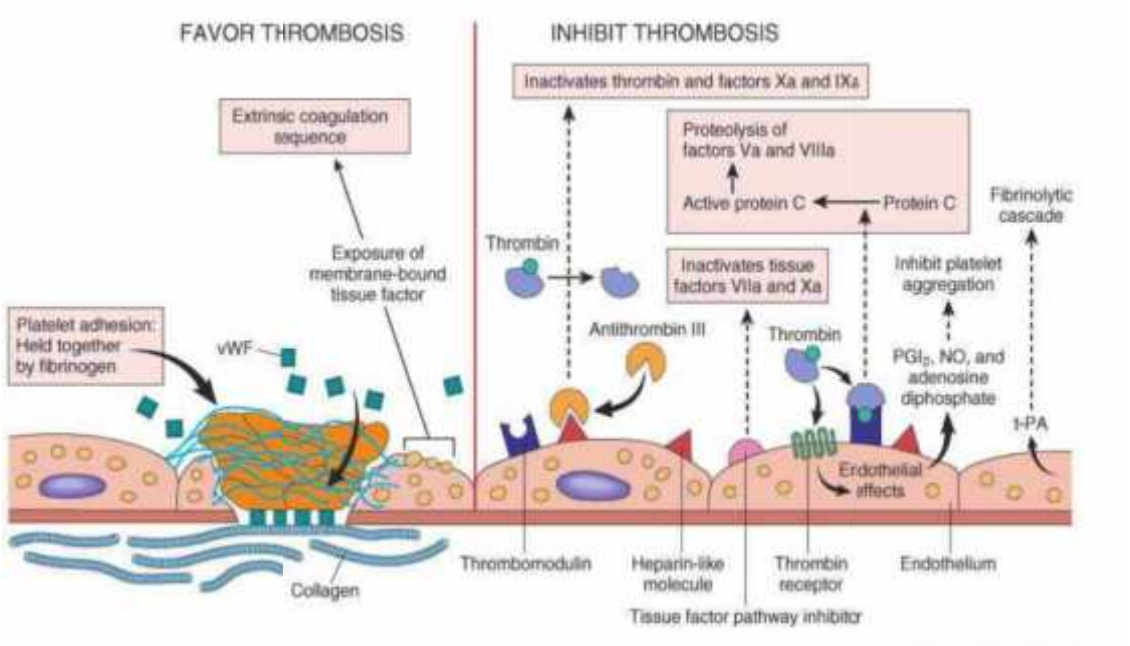

Figure 4: Role of platelets in thrombosis

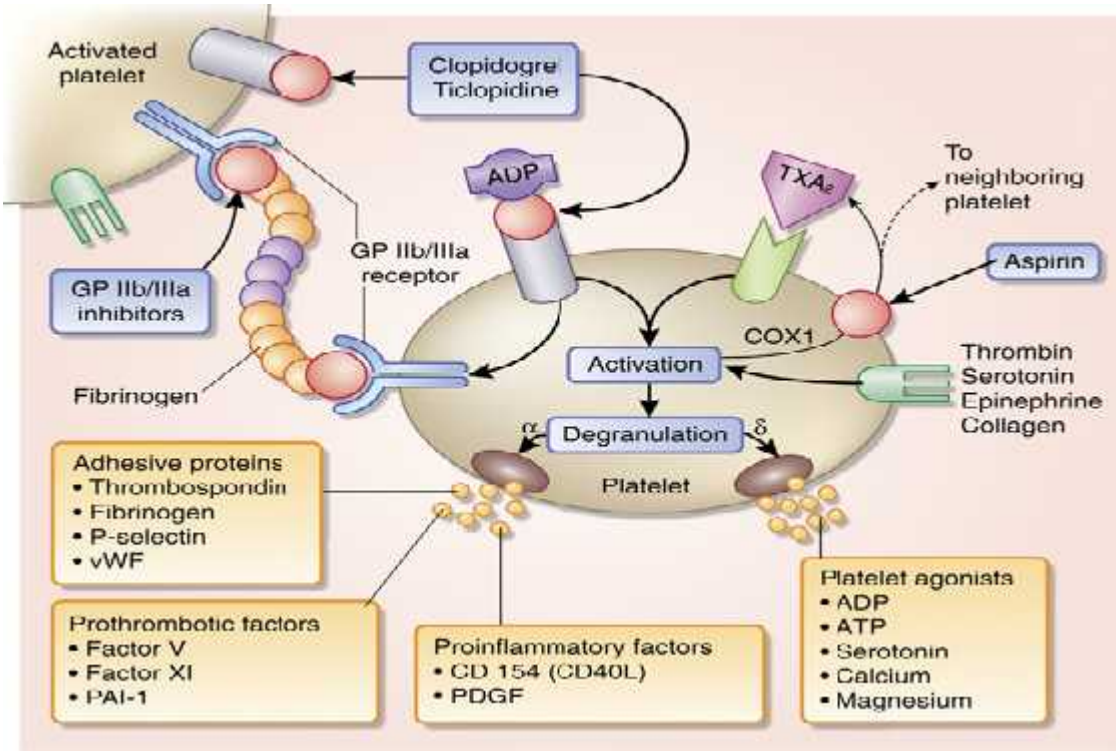

Figure 5: Structure and function of platelet

PRINCIPLE OF AUTOANALYSER:

Impedance measurement principle

The cells are passed one after the other through a capillary opening. The passing cells produce an electrical resistance and thus an electronic signal generated which is proportionate to its volume. Hence, the cells are identified based on their size and get represented in a volume distribution curve.[35]

PLATELET INDICES: Advances in automated blood cell analyser have made it possible to measure various blood cell parameters automatically. Platelet indices such as mean platelet volume, platelet distribution width and platelet large cell ratio provide important information but are not applied for routine clinical use.[36]

Mean Platelet Volume is calculated by the following formula,

MPV=(Plateletcrit/Platelet count)$\times 10^5$. Plateletcrit is the ratio of the platelet volume to the whole blood volume.[37]

Circulating platelets are different in size and functional activity. The larger platelets are more active and produce a greater quantity of thrombogenic factors.[38]

The Mean Platelet Volume increases with increased platelet turnover is mediated by several cytokines that affect megakaryocyte ploidy and result in the production of larger and more reactive platelets.[39]

Platelet Distribution Width is the distribution width on 20% frequency level with the peak considered as 100%.[40]The Platelet Distribution Width is useful to differentiate reactive thrombocytosis from other types, especially when it is combined with the MPV and platelet count to obtain a discriminant function.[41]

ROLE OF PLATELETS AND USE OF PLATELET PARAMETERS IN VARIOUS DISEASES

Mean platelet volume is significantly increased in patients with Iron deficiency Anaemia and Idiopathic Thrombocytopenic Purpura. It is also increased in Acute Post Streptococcal Glomerulonephritis, Renal Failure.

In patients with Rheumatic Heart Diseases and Diabetes Mellitus there is significant increase in Mean Platelet Volume compared with the control group.[42]

Patients with Ulcerative Colitis and inCrohn's Disease, there is a significant decrease in Mean Platelet Volume and Platelet Distribution Width when compared with healthy controls.[43]

Platelet count, Mean Platelet Volume and Platelet Distribution Width are higher in lung cancer patients compared with healthy subjects. Among patients with lung cancer Platelet distribution width in small cell lung cancer patients is higher than in non-small cell lung cancer patients.[44]

EVALUATION OF PLATELET INDICES IN ACUTE CORONARY SYNDROME

VitthalKhode et al. in 2011 studied in 128 patients, that the mean platelet volume was significantly higher in patients with Acute Myocardial Infection AMI (9.65 ± 0.96) as compared to Stable Coronary Artery Disease (SCAD) (9.37 ± 0.88) and controls (9.21 ± 0.58).

Mohammad Reza et al. in 2013,studied the importance of at admission platelet volume indices in patients with acute chest pain suggesting acute coronary syndrome at Department of Cardiology, Seyyed-al-Shohada Heart Center, Urmia University of

Medical Sciences, Urmia, Iran and showed that, An elevated admission Mean Platelet Volume and Platelet Distribution Width may be of benefit to detect chest pain resulting in MI from that of non-cardiac one, and also for risk stratification of patients who suffered from an acute chest discomfort.

Salim Ret al. in 2012, College of Medicine, Baghdad University, Baghdad, Iraq, study conducted in 50 patients showed that, platelet indices—mean platelet volume (MPV), platelet distribution width (PDW), were significantly raised in patients with Acute Myocardial Infarction and Unstable Angina.

JasminJasani et al., Studied on "Evaluation of platelet count and platelet indices in patients with coronary artery disease" and showed that Patients with acute coronary syndrome had higher platelet volume indices and lower platelet counts compared with those with stable angina and the normal population.

Abdullah S. Assiri et al studied on diagnostic importance of parameters in patients with coronary syndrome admitted to a tertiary care hospital in southwest region, Saudi Arabia among 212 patients with acute coronary syndrome and 49 matched controls. The MPV was significantly increased in MI cases compared to controls ($P < 0.009$); similarly, the MPV was significantly larger in UA cases compared to controls ($P < 0.001$). The PDW was significantly higher in MI cases compared to controls (15.88 ± 1.5 fl vs. 11.96 ± 1.8 fl, respectively, $P < 0.001$); similarly, the PDW as also significantly larger in UA cases compared to controls (18.1 ± 18 fl vs. 11.96 ± 1.8 fl, respectively, $P < 0.019$).

MATERIALSAND METHODS

MATERIALS AND METHODS

Source of data

A prospective hospital based study carried out on 200 patients attending emergency department or admitted in BLDE University ShriB.M.Patil Medical College, Hospital and Research Centre, Bijapur with the complaints of acute chest pain from October 2015 to September 2017.

All patients were evaluated for the cause of chest pain and were classified under cardiac and non-cardiac causes for chest pain .Patients with cardiac causes were compared with patients of non-cardiac causes of chest pain.

Method of collection of data

The study was carried out on patients presenting with acute chest pain to the emergency department or outpatient department.

All subjects were interviewed as per the prepared proforma and then complete clinical examination was done.

The blood samples of the patients were drawn from the antecubital vein using a 5ml syringe and immediately mixed in EDTA vacutainers.

The sample was run within two hours of venepuncture using the automated Hematoanalyzer and complete blood count analysis of the sample was made including the platelet indices.

The other relevant investigations like electrocardiogram and cardiac enzymes were also analysed for confirmation of the diagnosis.

Inclusion criteria

1. Patients presenting with acute onset of chest pain.
2. Patients diagnosed with unstable angina, ST segment elevation myocardial infarction(STEMI), non-ST segment elevation myocardial infarction (NSTEMI).
3. Patients more than 18 years of age.

Exclusion criteria

1. Patients with bleeding diathesis, previous stroke, major operations or significant trauma in past two weeks or hypertension (>180/110mm of hg).
2. Patients less than 18 years of age.

STATISTICAL METHODS EMPLOYED:

Data was analysed by using:

1) Diagrammatic presentation
2) Mean ± SD
3) 't' test
4) Chi square test

RESULTS

RESULTS

The study included 200 subjects with 100 cases of cardiac chest pain and 100 controls of non-cardiac chest pain.

Among the 100 cases of cardiac chest pain 22 were diagnosed as ST-Elevation Myocardial Infarction, 55 were of Non-ST-Elevation Myocardial Infarction and 23 were diagnosed as Unstable angina.

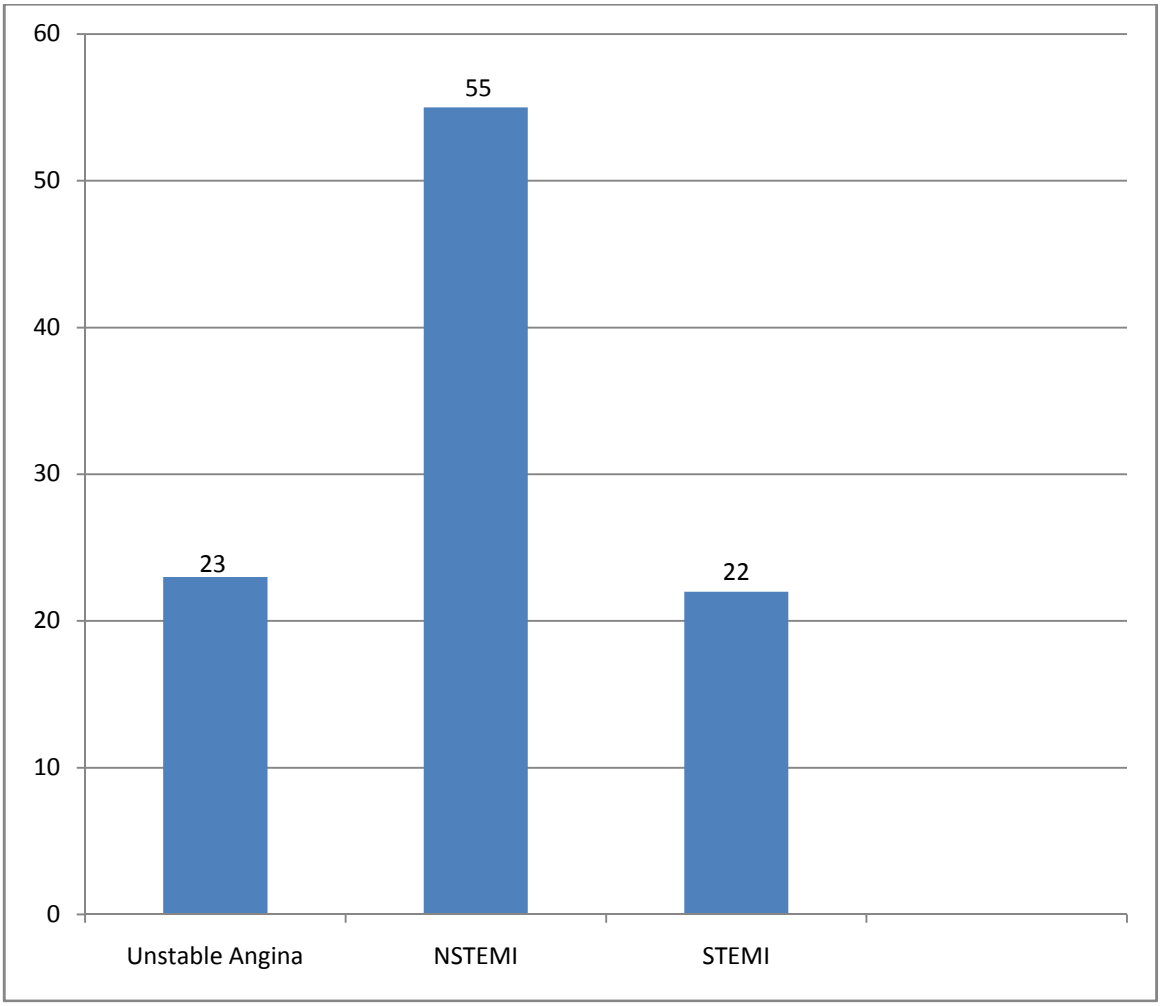

Figure 6: Classification of acute coronary syndrome among cases

Table 3: Disease distribution in different age groups

AGE(Years)	NUMBER	PERCENT
31-40	22	11.1%
41-50	49	24.6%
51-60	57	28.6%
61-70	54	27.1%
>70	18	8.5%
Total	**200**	**100%**

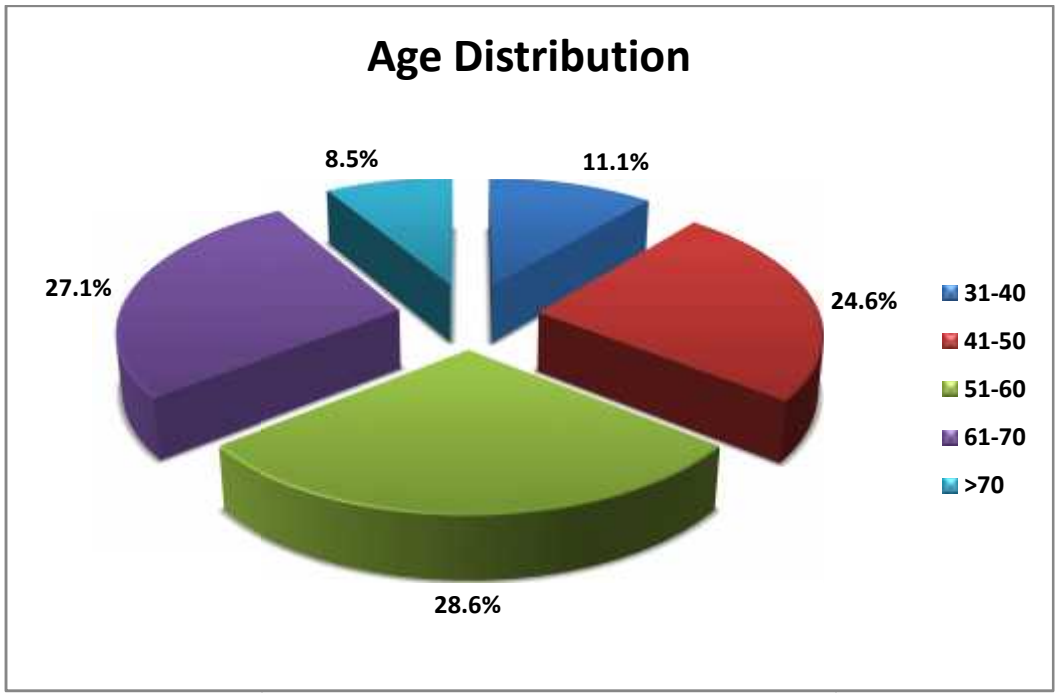

FIGURE 7: Distribution of cases according to age

In the present study, ages ranged from 30 to 85 years were registered. Majority of the cases belonged to the age group of 50 to 70 years (55.7%) followed by fifth decade of life (24.6%).

Table 4: Distribution of cases according to sex

SEX	NUMBER	PERCENT
Male	127	63.8%
Female	73	36.2%
Total	**200**	**100%**

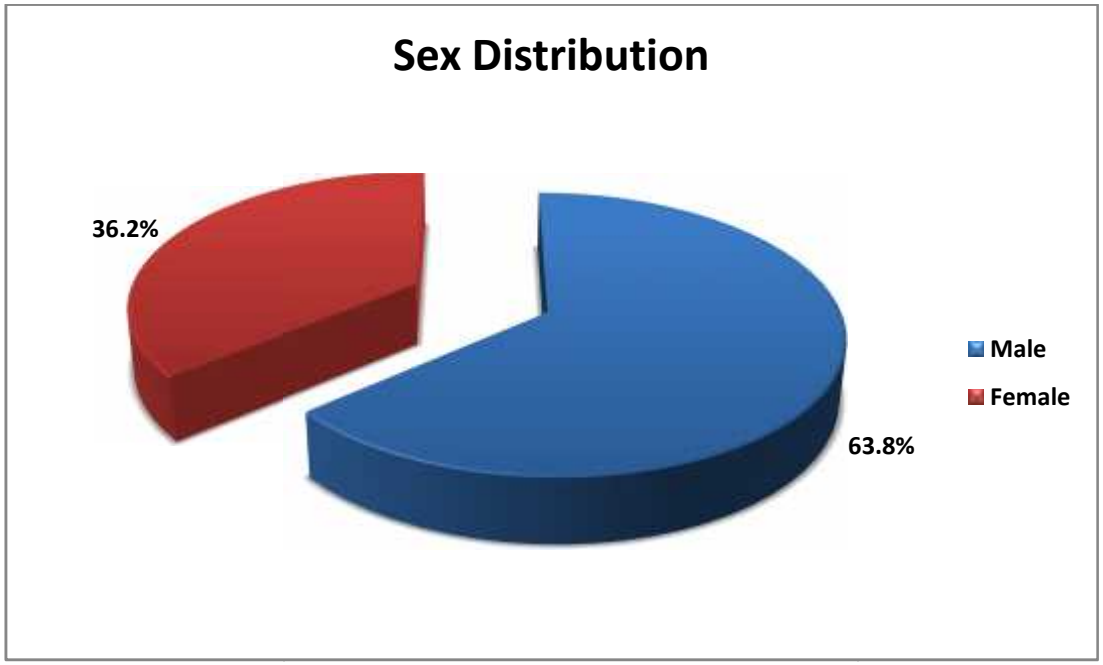

Figure 8: Distribution according to sex

In the study, total number of male including both cases and controls were 127 (63.8%) and total numbers of females were 73 (36.2%). The total number of males presenting with chest pain and were diagnosed with acute coronary syndrome were 58 (45.6%) and among females 42 (57.53%) patients were diagnosed as acute coronary syndrome. The male to female ratio was 1.7:1.

TABLE 5: Association of age and sex among cases and controls

AGE (Years)	MALE		FEMALE		p VALUE
	Number	Percent	Number	Percent	
31-40	16	12.6	6	8.3	0.26
41-50	36	28.3	13	18.1	
51-60	35	27.6	22	30.6	
61-70	29	22.8	25	34.7	
>70	11	8.7	6	8.3	
Total	127	100.0	72	100.0	

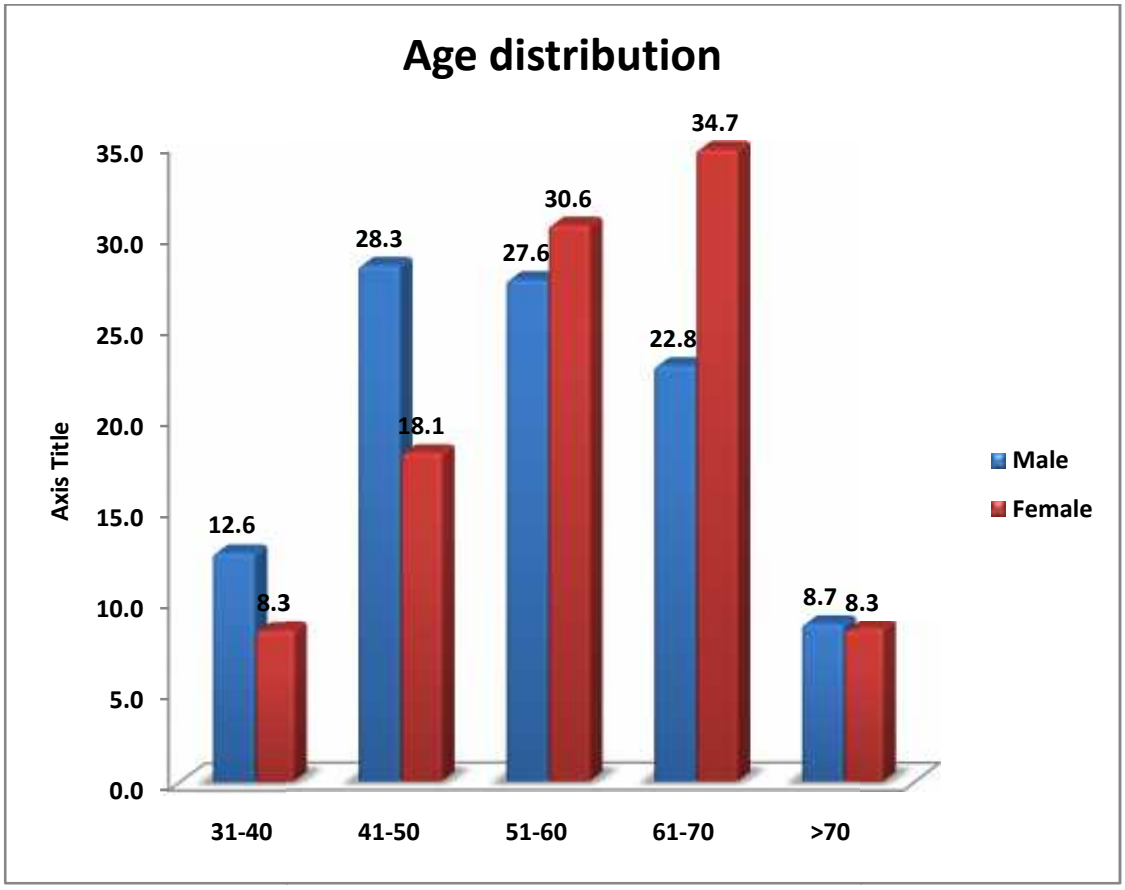

Figure 9: Association of Age and Sex

TABLE 6: Comparison of mean age among ACS and NON-ACS cases

Variables	Non Cardiac		NSTEMI		STEMI		Unstable Angina		p value
	N	%	N	%	N	%	N	%	
AGE	55.2	11.4	56.8	12.0	60.4	10.5	54.0	12.5	0.032*

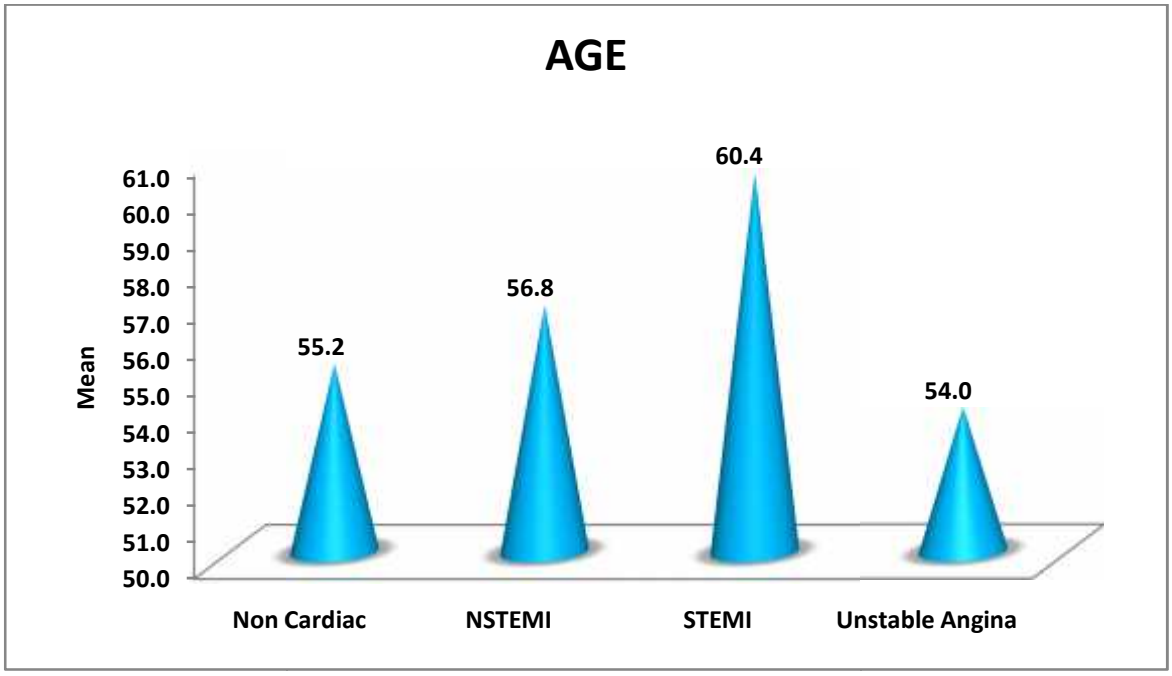

Figure 10: Comparison of Mean Age among ACS and Non ACS cases

In our study the incidence of Acute Coronary Syndrome was more common in males compared to females. Among total of 100 cases of acute coronary syndrome in the study group 58 were males and females were 42

The incidence of STEMI, NSTEMI and Unstable Angina was found to be statistically significant with more common in males compared to females.

Male predominance was seen among all the three groups of ACS.

TABLE 7: Association of acute coronary syndrome and sex

SEX	Non Cardiac		NSTEMI		STEMI		Unstable Angina		p value
	Nos	%	Nos	%	Nos	%	Nos	%	
Male	69	69.0	12	57.1	31	56.4	15	65.2	0.406
Female	31	31.0	9	42.9	24	43.6	8	34.8	
Total	100	100.0	21	100.0	55	100.0	23	100.0	

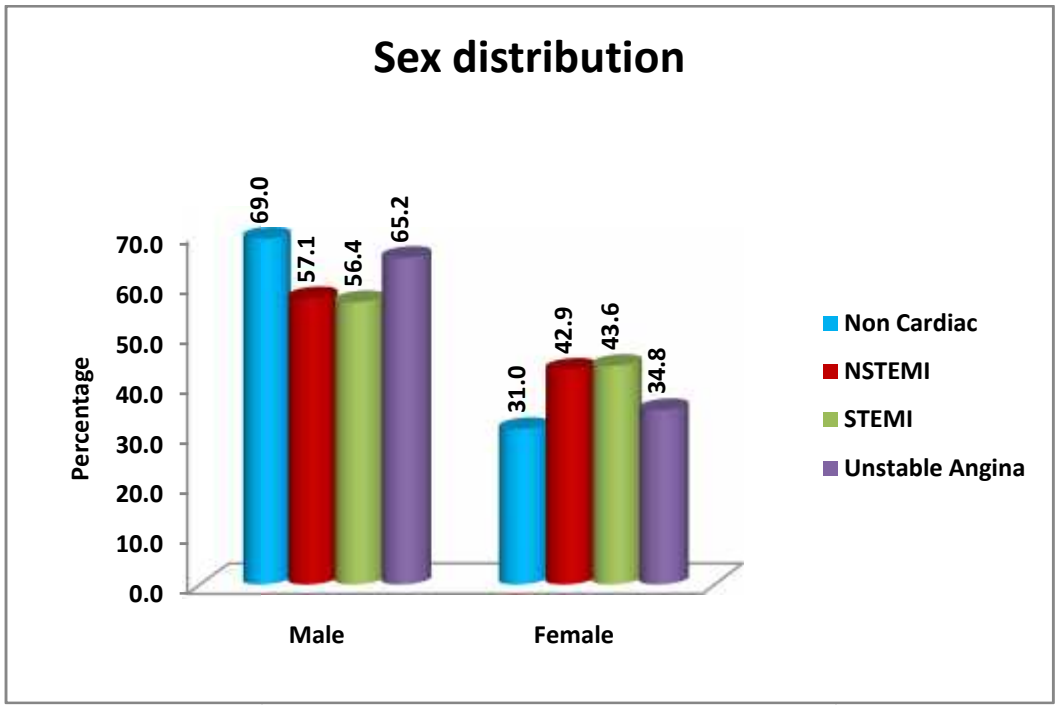

Figure 11: Association of AMI and Sex

RISK FACTORS

The risk factors for developing ischemic heart diseases such as Smoking, Hypertension, Diabetes Mellitus and Family history were analysed.

In our study family history of Ischemic Heart Disease was noted in 8% among cases and 2% among controls. Those with the Family history had 4 times increased risk of getting the disease as compared to controls with positive family history. But it was not statistically significant.

All the smokers in our study group were male. The prevalence of smoking in the study population was found to be 27.5%. The highest prevalence of smoking was noted among STEMI (50.9%) followed by NSTEMI (28.6%) and in unstable angina (21.7%). The prevalence among smoking in controls was (16.0%). The history of smoking was statistically significant in cases compared to controls (p= <00.1). The cigarette smoking was found to be risk factor for IHD in our study.

Similarly prevalence of hypertension in the study group was found to be 11% and was seen in 29.1% among cases with STEMI and in 14.3% of patients with NSTEMI. Whereas hypertension was seen in only 4.3% among cases with unstable angina and in 2% among controls.Hypertension is found to be a direct risk factor for ischemic heart disease in our study.

The prevalence of Diabetes in the study population was found to be 18%. The study showed prevalence of diabetes was maximum in NSTEMI with 38.1% followed by STEMI 32.7%. Prevalence of diabetes among unstable angina was found to be 13.0% and among controls 7.0%. Thus study shows diabetes as statistically significant risk factor and as an independent risk factor for ischemic heart disease.

Dyslipidaemia was found to be an important contributing risk factor among cases with acute coronary syndrome. Prevalence of dyslipidaemia was seen in 53% among cases of acute coronary syndrome, whereas dyslipidaemia was found in only 10% of controls. Thus in our study dyslipidaemia was found to be significant risk factor for development of ACS.

TABLE 8: Association of acutecoronary syndrome and risk factors

PAST HISTORY	Non Cardiac		NSTEMI		STEMI		Unstable Angina		p value
	N	%	N	%	N	%	N	%	
Smoking	16	16.0	6	28.6	28	50.9	5	21.7	<0.001*
Diabetes	7	7.0	8	38.1	18	32.7	3	13.0	<0.001*
Hypertension	2	2.0	3	14.3	16	29.1	1	4.3	<0.001*

Note: *means significant at 5% level of significance (p<0.05)

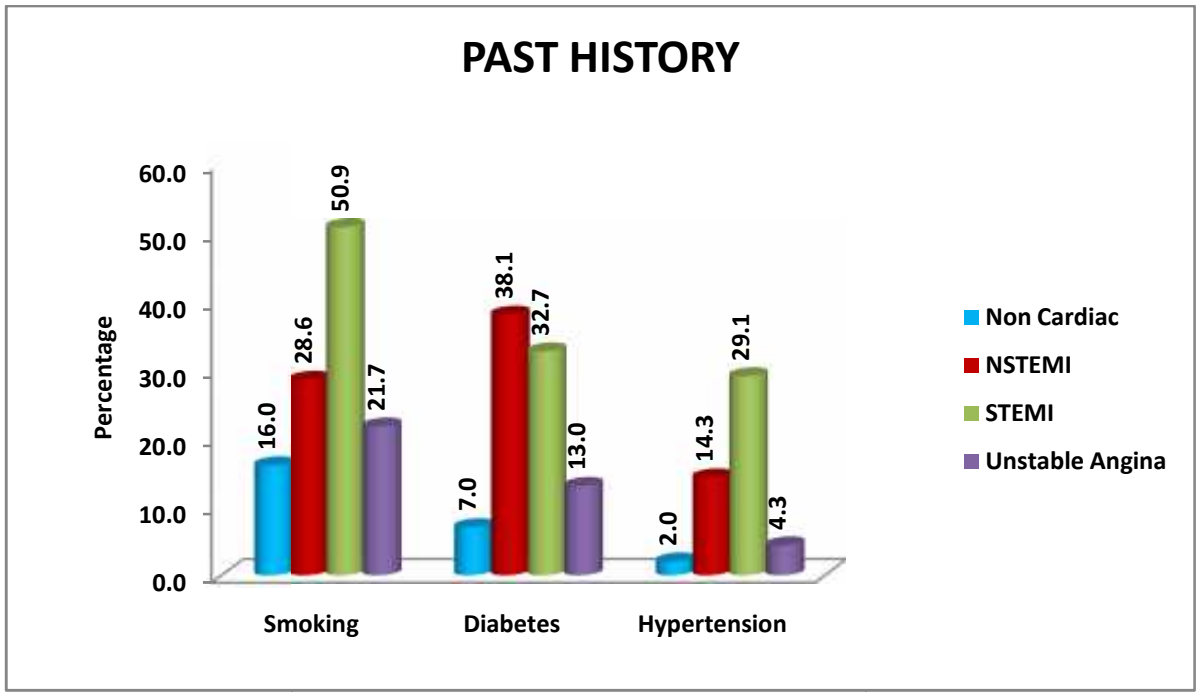

Figure 12: Association of Acute Coronary Syndrome and Risk Factors

TABLE 9: Association of acute coronary syndrome and lipid profile

LIPID PROFILE	Non Cardiac		NSTEMI		STEMI		Unstable Angina		p value
	N	%	N	%	N	%	N	%	
Normal	90	90.0	12	57.1	23	41.8	12	50.0	
Dyslipidaemia	10	10.0	9	42.9	32	58.2	12	50.0	<0.001*
Total	100	100.0	21	100.0	55	100.0	24	100.0	

Note: *means significant at 5% level of significance (p<0.05)

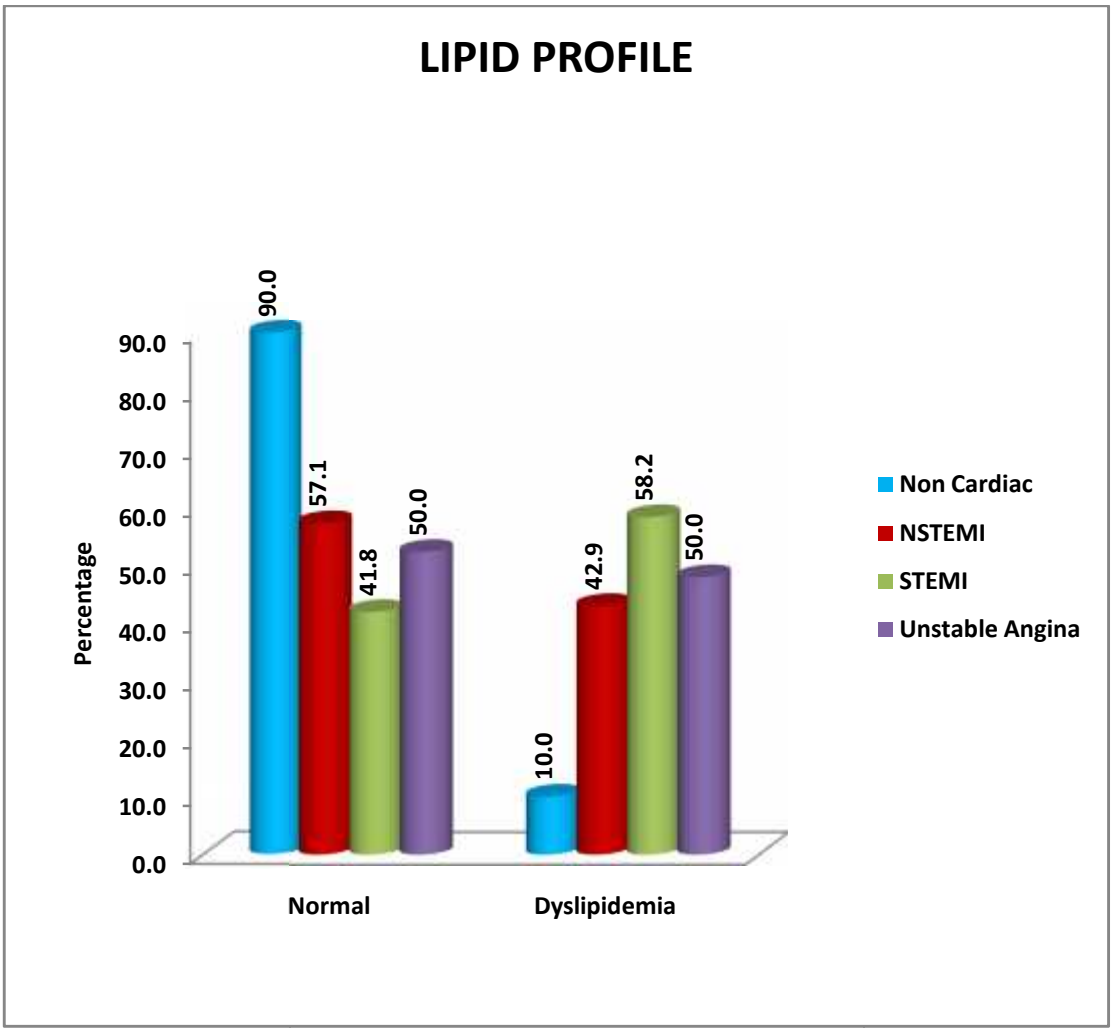

Figure 13: Association of Acute Coronary Syndrome and Lipid Profile

CARDIAC BIOMARKERS

Cardiac biomarker Troponin T was found positive in 61 among the cases with 49 among STEMI and among 10 and 2 in NSTEMI and Unstable angina respectively.

Among STEMI cases Troponin T was found to be positive in 89.1% and was negative in 10.9%. Similarly Troponin T was found positive in 47.6% of NSTEMI.

In our study the CPK-MB was significantly increased in cases with STEMI with mean valve of 92.9±77.8 followed by NSTEMI 62.5±60.2, whereas in non-cardiac controls CPK-MB was 20.3±12.2.Considering CPK-MB valve of more than 30 as significant association with Acute Coronary Syndrome, in our study it was found that CPK-MB valve was significant in STEMI (96.4%). In NSTEMI, CPK-MB was significantly increased in 71.4% and in Unstable Angina 43.5% compared to controls (12.0%).

Table 10: Association of Acute Coronary Syndrome and Troponin T

TROP T	Non Cardiac		NSTEMI		STEMI		Unstable Angina		p value
	N	%	N	%	N	%	N	%	
Negative	100	100.0	11	52.4	6	10.9	24	100.0	
Positive	0	0.0	10	47.6	49	89.1	0	0.0	<0.001*
Total	100	100.0	21	100.0	55	100.0	24	100.0	

Note: *means significant at 5% level of significance (p<0.05)

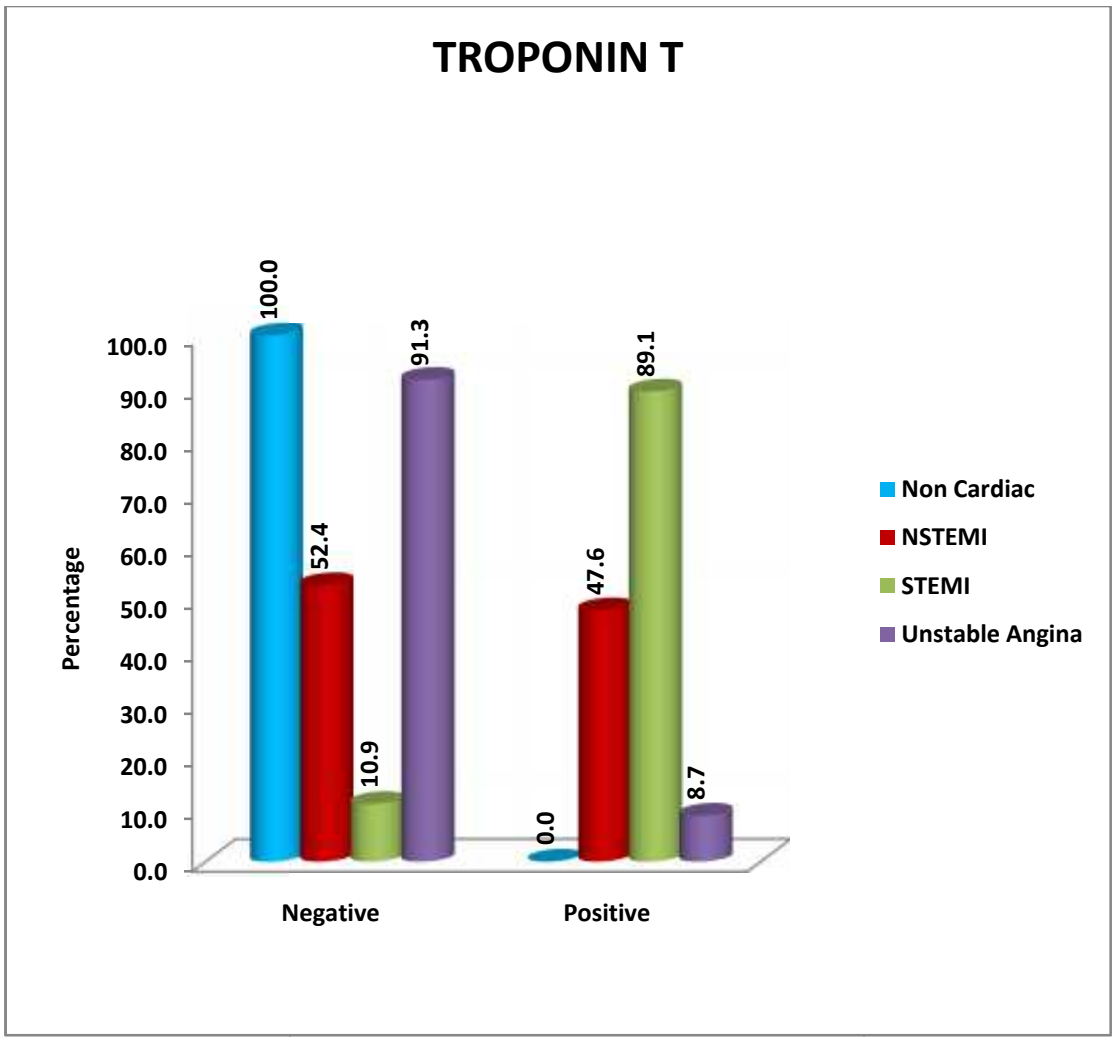

Figure 14: Association of Acute Coronary Syndrome and Troponin T

Table 11: Association of acute coronary syndrome and ECG.

ECG	Non Cardiac		NSTEMI		STEMI		Unstable Angina		p value
	N	%	N	%	N	%	N	%	
Normal	89	89.0	0	0.0	0	0.0	3	13.0	<0.001*
ST depression	11	11.0	21	100.0	0	0.0	20	87.0	
ST elevation	0	0.0	0	0.0	55	100.0	0	0.0	
Total	100	100.0	21	100.0	55	100.0	23	100.0	

Note: *means significant at 5% level of significance (p<0.05)

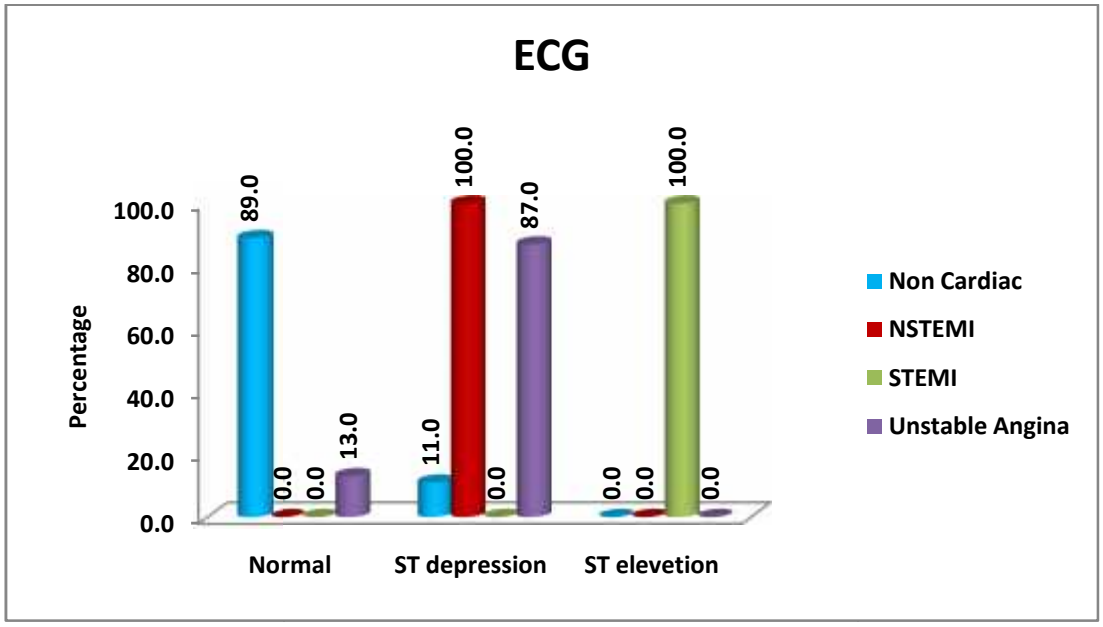

Figure 15: ECG changes among cases with ACS

In our study ST-Elevation was seen in 55 cases of which 26 cases had ST-Elevation in Inferior wall leads followed by 18 cases with Anteroseptal and 11 cases had ST-Elevation in lateral wall leads. ST-Depression was seen in 41 cases among which 21 cases were diagnosed with NSTEMI and 20 cases were diagnosed with Unstable Angina. Non Specific ST segment changes were seen among 11 patients with non-cardiac chest pain.

TABLE 12: Association of acute coronary syndrome and ECHO

ECHO	Non Cardiac		NSTEMI		STEMI		Unstable Angina		p value
	N	%	N	%	N	%	N	%	
IHD	2	2.0	21	100.0	55	100.0	11	47.8	<0.001*
Normal	98	98.0	0	0.0	0	0.0	12	52.2	
Total	100	100.0	21	100.0	55	100.0	23	100.0	

Note: *means significant at 5% level of significance (p<0.05)

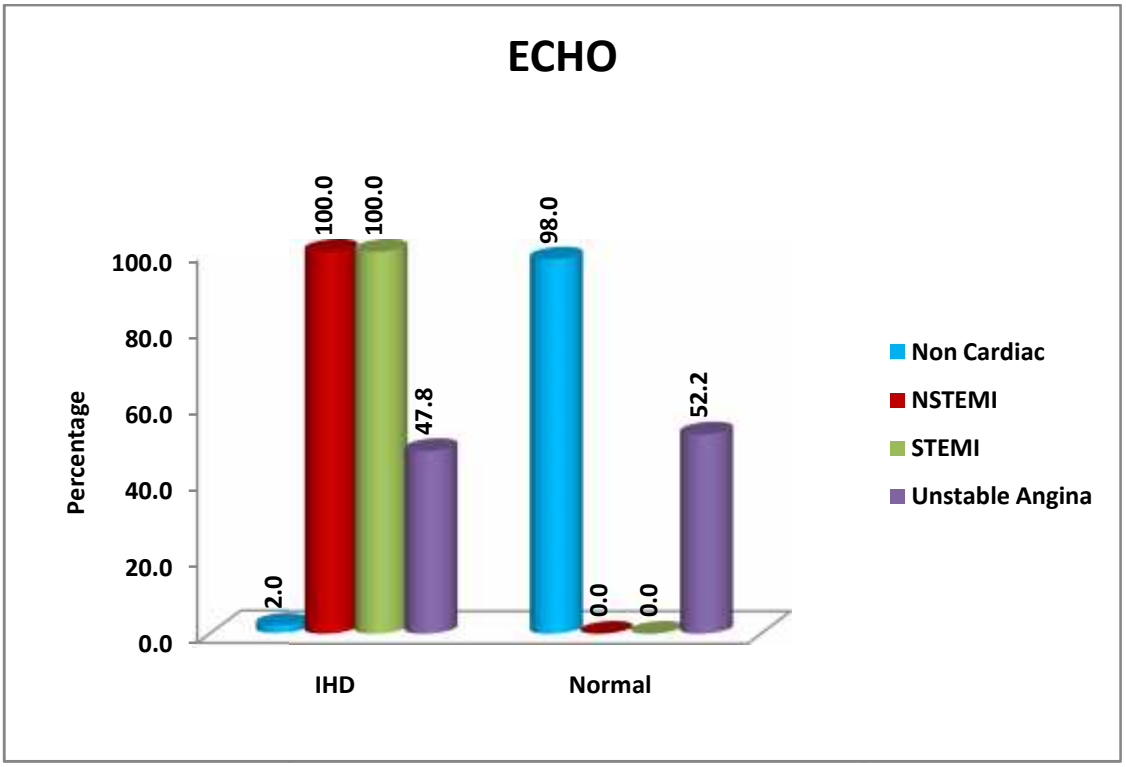

Figure 16: Association of Acute Coronary Syndrome and ECHO

Echocardiography was done in all patients presented with acute onset chest pain. Echocardiography was suggestive of Ischemic heart disease in 88 patients among which 55 were diagnosed as STEMI (62.5%), 21 cases as NSTEMI (23.8%) and 11 cases as Unstable Angina (12.5%).

HEMATOLOGICAL PARAMETERS:

Platelet Indices:

The platelet indices Mean platelet volume (MPV), Platelet distribution width (PDW) and Platelet count were studied among patients with acute coronary syndrome and compared with age and sex matched control groups.

MEAN PLATELET VOLUME:

Mean Platelet Volume of all the patients was noted. The mean of the MPV for the control group in our study was 10.3±1.5fL, whereas for STEMI 11.5±2.5fL, NSTEMI 10.8±1.3fL and for Unstable Angina it was 10.6±1.7fL The MPV was highest in ST-Elevation Myocardial Infarction group 11.5±2.5fL followed by Non-ST Elevation Myocardial Infarction 10.8±1.3fL. The MPV in Unstable Angina 10.6±1.7fL was close to the MPV values recorded in the control group.

TABLE 13: Comparison of platelet indices and haematological parameters among ACS and NON-ACS cases

Variables	Non Cardiac		NSTEMI		STEMI		Unstable Angina		p value
	MEAN	SD	MEAN	SD	MEAN	SD	MEAN	SD	
PC	2.4	0.7	2.7	0.7	2.5	0.6	2.5	0.7	0.204
PDW(fl)	10.2	1.7	11.0	1.4	11.7	2.1	11.1	2.0	<0.001*
MPV(fl)	10.3	1.5	10.8	1.3	11.5	2.5	10.6	1.7	0.001*
WBC	9.1	3.9	11.1	3.3	12.2	3.4	10.3	3.6	<0.001*
HBG	12.0	1.9	12.5	1.8	12.0	2.1	12.7	1.6	0.356

Note: *means significant at 5% level of significance (p<0.05)

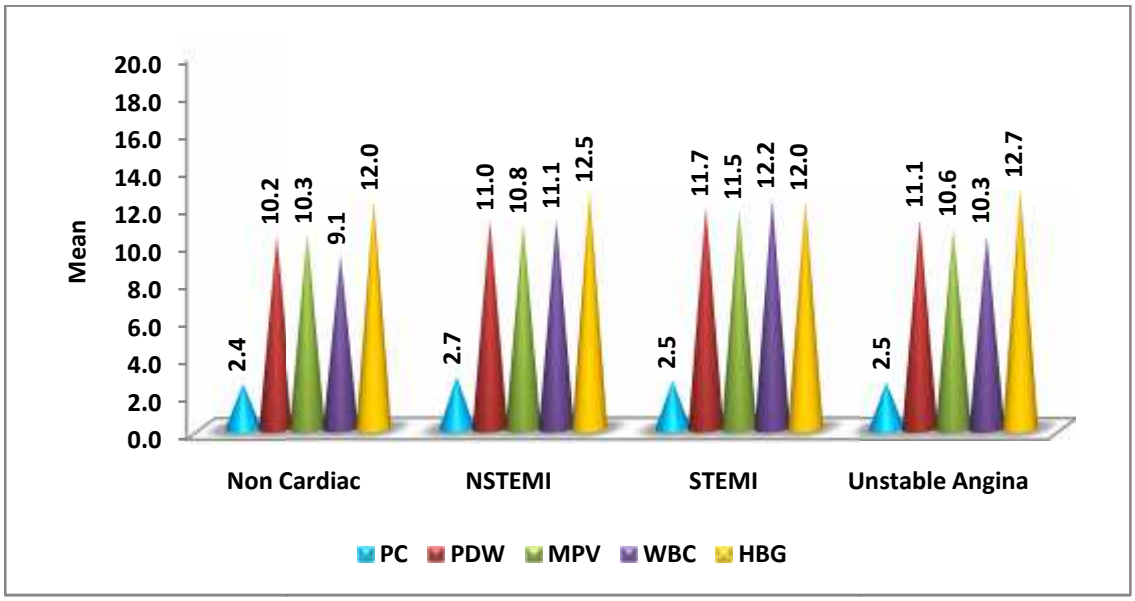

Figure 17: Comparison of Haematological Indices among ACS and Non ACS cases.

In the study we found MPV to be highly significant for both STEMI and NSTEMI with p value (p<0.001) at second degree of freedom and 95% Confidence level control in comparison to patients with unstable angina.

PLATELET DISTRIBUTION WIDTH:

Platelet Distribution Width of all the patients was noted. The mean of the PDW for the control group in our study was 10.2±1.7fL, whereas for STEMI 11.7±2.1fL, NSTEMI 11.0±1.4fL and for Unstable Angina it was 11.1±2.0fL The PDW was highest in ST-Elevation Myocardial Infarction group followed by Non-ST Elevation Myocardial Infarction and Unstable Angina. Our study showed significant increase in PDW among cases with ACS compared to controls.

Association between Mean Platelet Volume and Cardiac Troponin T

In our study it was observed that the association between Mean Platelet Volume and cardiac Troponin T was statistically significant ($p<0.001$) for STEMI and NSTEMI group as these group had noticed a larger value of MPV and had cardiac enzyme Troponin T positive. Among the cases with Troponin T positive the Mean Platelet Volume was 11.5±2.4fL whereas in Troponin T negative group MPV was 10.4±1.5fL.Thus Troponin T positive is associated with statistically significant ($p<0.001$) elevated values of Mean Platelet Volume compared to Troponin T Negative.

TABLE 14:Comparison of mean PDW and MPV according to TROPONIN T

Variables	TROP T +ve		TROP T -ve		p value
	Mean	SD	Mean	SD	
PDW	11.7	2.0	10.4	1.7	<0.001*
MPV	11.5	2.4	10.4	1.5	<0.001*

Note: *means significant at 5% level of significance (p<0.05)

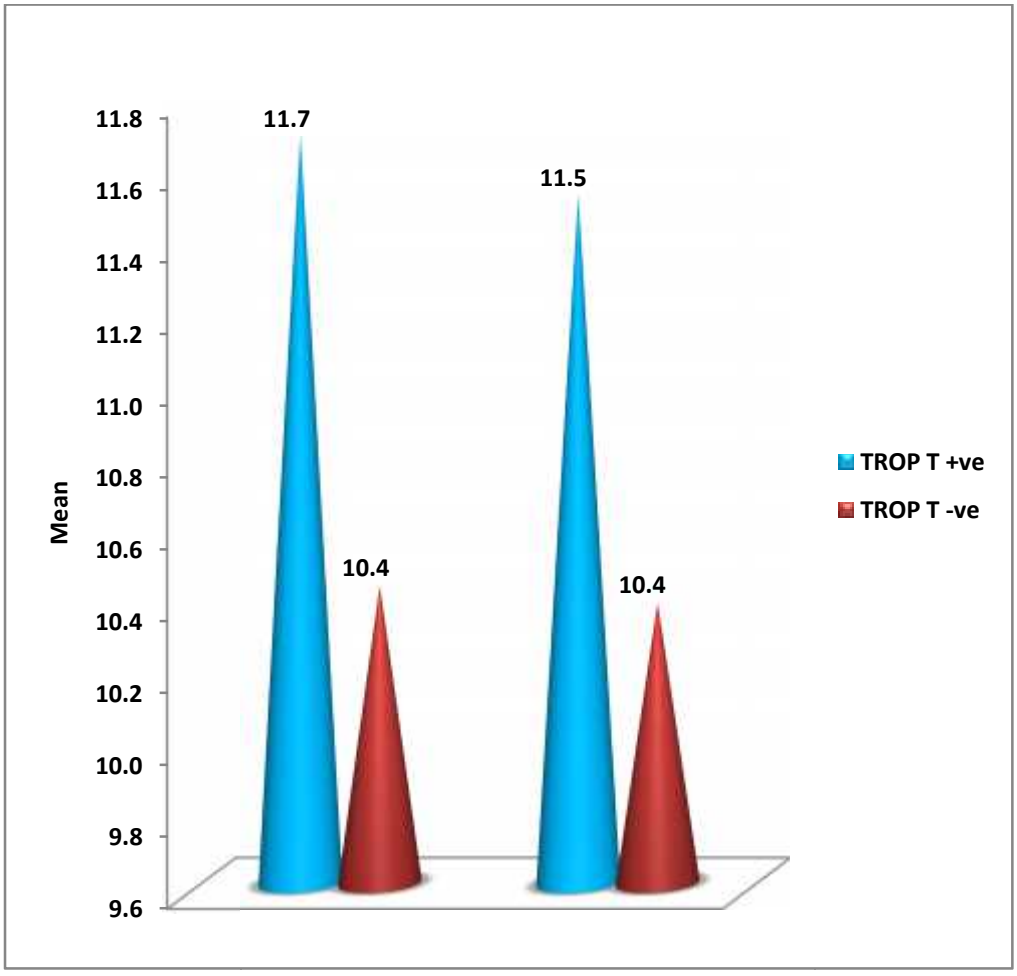

Figure 18: Comparison of Mean PDW and MPV according to TROPONIN T

Table 15: Comparison of Mean CPK MB among ACS and Non ACS cases

Variables	Non Cardiac		NSTEMI		STEMI		Unstable Angina		p value
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
CPK MB	20.3	12.2	62.5	60.2	92.9	77.8	33.5	15.8	<0.001*

Note: *means significant at 5% level of significance (p<0.05)

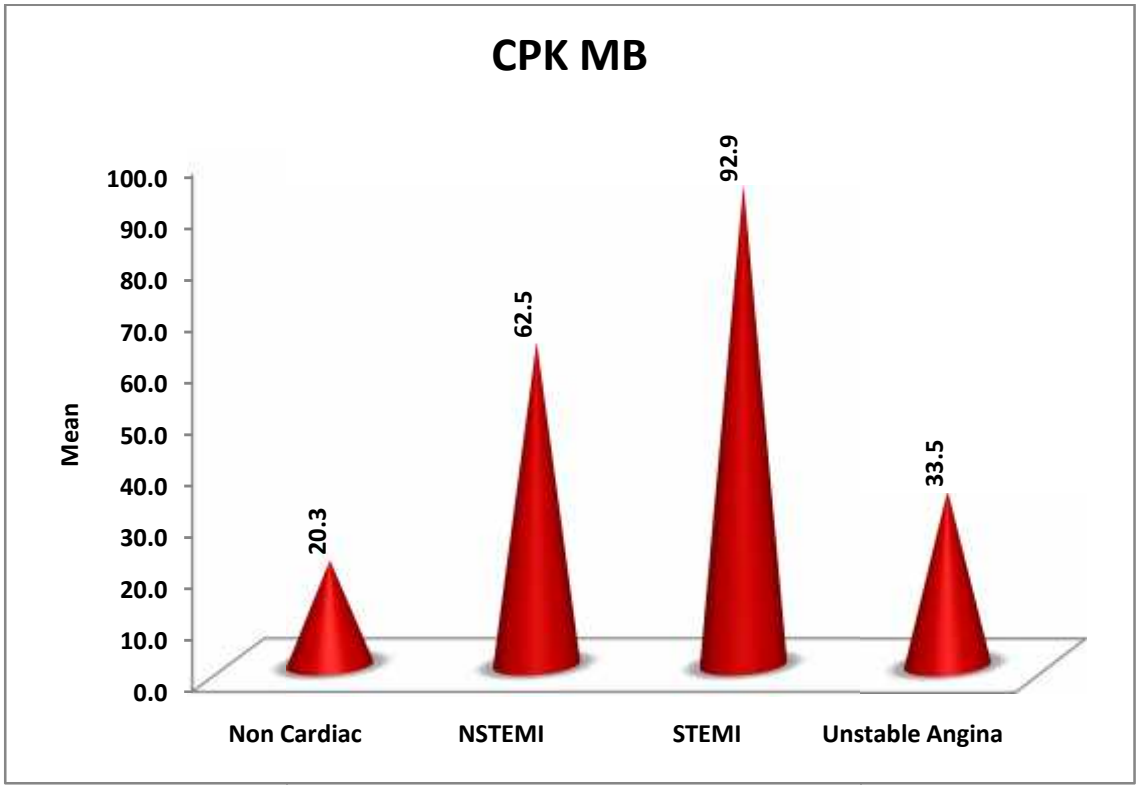

Figure 19: Comparison of Mean CPK MB among ACS and Non ACS cases

Table 16: Association of AMI and CPK MB

CPK MB	Non Cardiac		NSTEMI		STEMI		Unstable Angina		p value
	N	%	N	%	N	%	N	%	
CPK MB ≤30	88	88.0	6	28.6	2	3.6	13	56.5	<0.001*
CPK MB >30	12	12.0	15	71.4	53	96.4	10	43.5	
Total	100	100.0	21	100.0	55	100.0	23	100.0	

Note: *means significant at 5% level of significance (p<0.05)

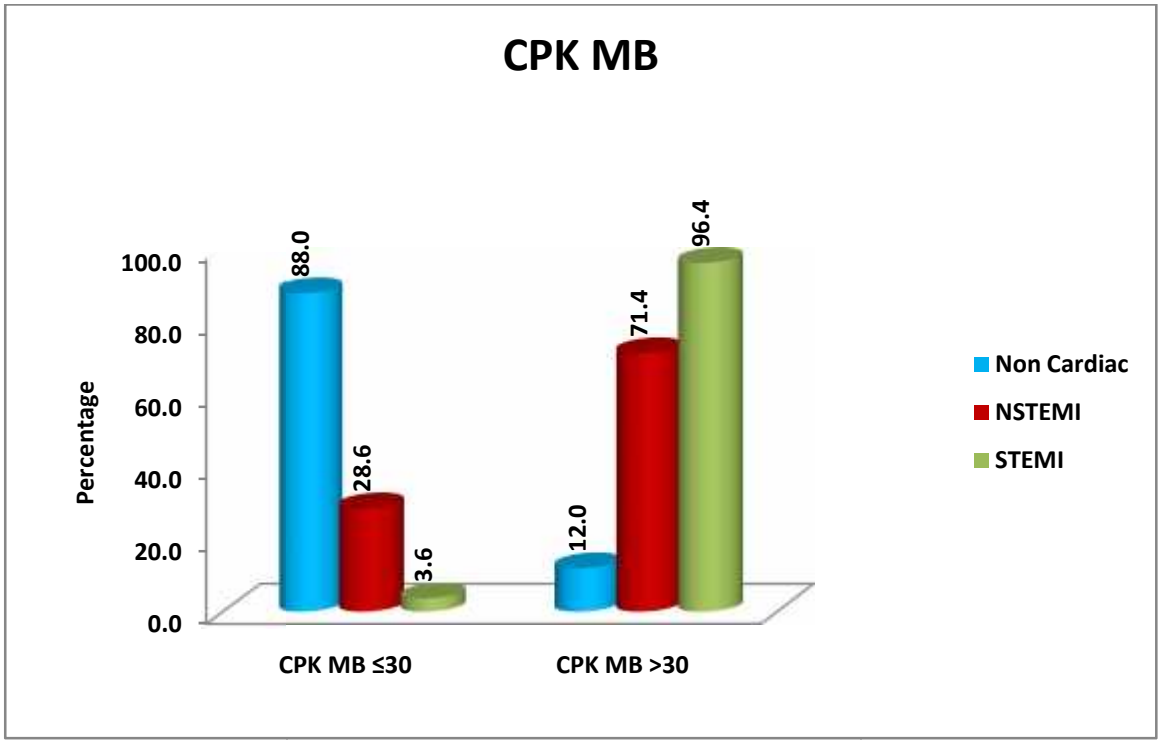

Figure 20: Association of AMI and CPK MB

Considering CPK-MB valve of more than 30 as significant association with Acute Coronary Syndrome, in our study it was found that CPK-MB valve was significant in STEMI (96.4%). In NSTEMI, CPK-MB was significantly increased in 71.4% and in Unstable Angina 43.5% compared to controls (12.0%).

Table 17:Comparison of Mean PDW and MPV according to CPK MB

Variables	CPK MB ≤30		CPK MB >30		p value
	Mean	SD	Mean	SD	
PDW	10.2	1.6	11.5	2.0	<0.001*
MPV	10.3	1.4	11.2	2.2	<0.001*

Note: *means significant at 5% level of significance (p<0.05)

Our study showed that MPV was significantly in cases with significant CPK-MB levels. The MPV among cases with CPK-MB>30 was 11.5±2.2 as compared to those with CPK-MB<30 (10.3±1.4). Thus MPV was significantly increased among cases with elevated CPK-MB levels.

Similarly PDW was also significantly increased among cases with CPK-MB>30 (11.5±2.0) compared to those with CPK-MB<30 (10.2±1.6).

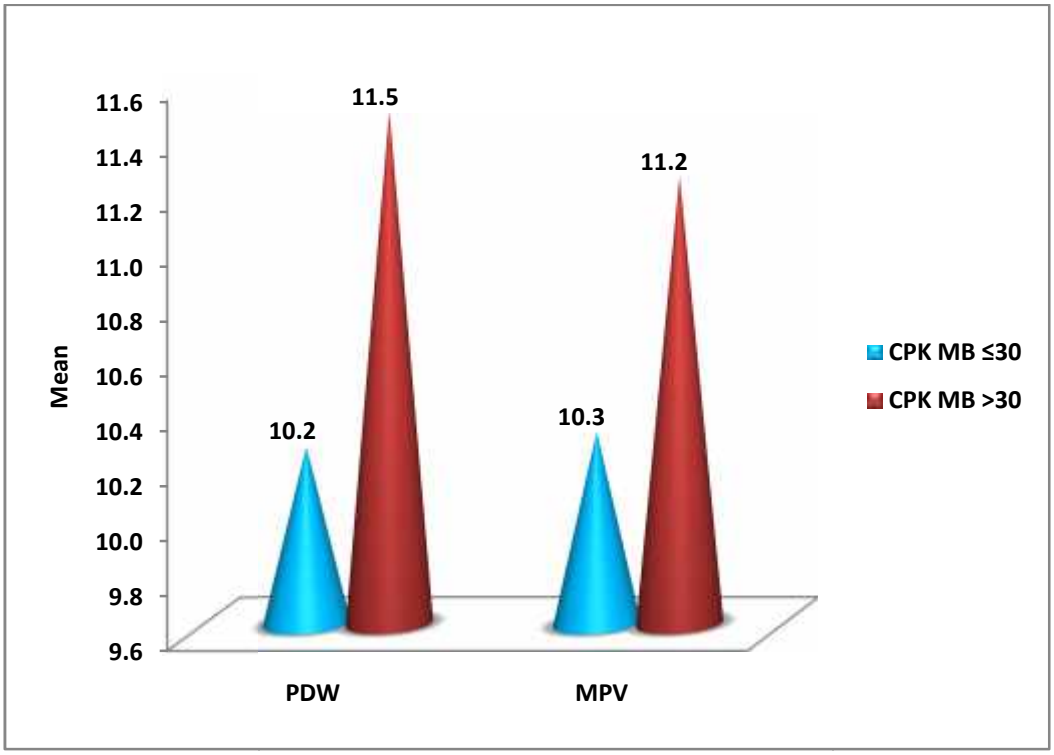

Figure 21: Comparison of Mean PDW AND MPV according to CPK MB

DISCUSSION

Acute coronary syndrome is a major cause of morbidity and mortality in industrialized countries. The aetiology of Ischemic Heart Disease is multifactorial. The study included 200 subjects with 100 cases of cardiac chest pain and 100 controls of non-cardiac chest pain. Among the 100 cases of cardiac chest pain 22 were diagnosed as ST-Elevation Myocardial Infarction, 55 were of Non-ST-Elevation Myocardial Infarction and 23 were diagnosed as Unstable angina.

AGE:

Cases of ages ranged from 30 to 85 years were registered. Majority of the cases belonged to the age group of 50 to 70 years (55.7%) followed by fifth decade of life (24.6%). This is similar with the Asian population at risk for an Ischemic Heart Disease, occurring most commonly in the 6th decade of life.[60] More than 50% of coronary artery disease related deaths in India occur among people <70 years of age.[61]

GENDER:

The prevalence of cardiovascular disease is higher in males compared to females but the mortality is high among females compared to males. The Framingham study showed that women a lower incidence of coronary artery disease than men till the age of 75. In the present study total number of male including both cases and controls were 127 (63.8%) and total numbers of females were 73 (36.2%). This percentage was almost similar to other studies on the correlation between MPV and ACS. The male to female ratio in our study was 1.7:1.

ASSESSMENT OF RISK FACTORS:

The prevalence of coronary artery disease in urban Indian population is 6.5% to 7.4%. In our study family history of Ischemic Heart Disease was noted in 8%

among cases and 2% among controls. In our study the distribution of risk factors was similar to that of other studies showing relationship between ACS and MPV.

SMOKING:

Smoking has been identified as one of the important risk factor for all cardiovascular diseases. Smokers have an approximately two fold higher risk of coronary artery disease compared with non-smokers.[65] The prevalence of smoking in the study population was found to be 27.5%. The highest prevalence of smoking was noted among STEMI (50.9%) followed by NSTEMI (28.6%) and in unstable angina (21.7%). The prevalence among smoking in controls was (16.0%). The history of smoking was statistically significant in cases compared to controls (p= <00.1).

A study done by Joshi et.al in 2007 showed higher prevalence of smoking in patients presenting with ACS. Another similar study done by Khandekar MM et al, showed prevalence of smoking in patients presenting with ACS (21.4%) as compared to controls (5%).

HYPERTENSION:

Prevalence of hypertension in the study group was found to be 11% and was seen in 29.1% among cases with STEMI and in 14.3% of patients with NSTEMI. Whereas hypertension was seen in only 4.3% among cases with unstable angina and in 2 % among controls. Hypertension is found to be a direct risk factor for ischemic heart disease in our study.

DIABETES MELLITUS:

Diabetes is a major risk factor for coronary artery diseases. Prevalence of Diabetes in the study population was found to be 18%. The study showed prevalence of diabetes was maximum in NSTEMI with 38.1% followed by STEMI 32.7%. Prevalence of diabetes among unstable angina was found to be 13.0% and among controls 7.0%.

A similar study conducted by Jaya Manchand et al showed prevalence of Diabetes in 19.4% in patients with ACS and in 6.7% among controls, which was similar to our study results. The study done by Kodiatte et al showed more male diabetics compared to females which is similar to our study results.

DYSLIPIDEMIA:

Dyslipidaemia was found to be an important contributing risk factor among cases with acute coronary syndrome. Prevalence of dyslipidaemia was seen in 53% among cases of acute coronary syndrome, whereas dyslipidaemia was found in only 10% of controls. Thus in our study dyslipidaemia was found to be significant risk factor for development of ACS.

MEAN PLATELET VOLUME

The mean of the MPV for the control group in our study was 10.3±1.5fL, whereas for STEMI 11.5±2.5fL, NSTEMI 10.8±1.3fL and for Unstable Angina it was 10.6±1.7fL The MPV was highest in ST-Elevation Myocardial Infarction group 11.5±2.5fL followed by Non-ST Elevation Myocardial Infarction 10.8±1.3fL. The MPV in Unstable Angina 10.6±1.7fL was close to the MPV values recorded in the control group. In the study we found MPV to be highly significant for both STEMI and NSTEMI with p value ($p<0.001$) at second degree of freedom and 95% Confidence level control in comparison to patients with unstable angina.

Table 18: Comparison of MPV in AMI and controls in different studies.

PUBLICATION	CASES	MPV(fl)	CONTROLS	MPV(fl)	p VALUE
Agrawal et al[45]	50	11.04±2.2	50	7.81±1.28	0.0001
ManjuPandey et al[46]	110	10.05±1.01	110	8.14±0.72	<0.001
Assiri et al[47]	212	8.99±1.5	48	8.38±0.51	0.009
Jasminjasani et al[48]	40	11.02±2.38	40	7.98±2.57	0.001
Khandekar et al[49]	94	10.43	30	9.2	<0.001

In our study it was observed that the association between Mean Platelet Volume and cardiac Troponin T was statistically significant (p<0.001) for STEMI and NSTEMI group as these group had noticed a larger value of MPV and had cardiac enzyme Troponin T positive. Among the cases with Troponin T positive the Mean Platelet Volume was 11.5±2.4fL whereas in Troponin T negative group MPV was 10.4±1.5fL

In patients with ACS there is rupture of unstable atherosclerotic plaque triggering a thrombogenic cascade leading to clinical events. The role of platelets and platelet reactivity is critically important in the formation and propagation of intracoronary thrombus. MPV is one of the marker indicating the function of platelets and activation of platelets.

The MPV was significantly raised in patients with ACS in comparison to NON-ACS in our study. This is in agreement with the results of similar studies conducted.

PLATELET DISTRIBUTION WIDTH

The PDW in our study was significantly increased in cases compared with controls. The mean PDW among controls was 10.2 ±1.7 as compared to cases with ACS 11.7±2.1. The results were similar to other studies which showed significant increase in PDW among cases compared to controls.PDW was also significantly increased among cases with CPK-MB>30 (11.5±2.0) compared to those with CPK-MB<30 (10.2±1.6).

Table 19: Comparison of PDW in AMI and controls in different studies.

PUBLICATION	CASES	PDW(fl)	CONTROLS	PDW(fl)	P VALUE
ManjuPandey et al	110	14.68±1.02	110	10.71±0.48	<0.001
Khandekar et al	94	13.19	30	10.75	<0.001
Assiri et al	212	15.88±1.5	48	11.96±1.8	0.009
Jasminjasani et al	40	17.85±3.30	40	10.70±2.07	0.001

The PDW in our study was significantly increasedin cases compared with controls. The mean PDW among controls was 10.2 ±1.7 as compared to cases with ACS 11.7±2.1. The results were similar to other studies which showed significant increase in PDW among cases compared to controls.

Study done by ManjuPandey et al showed PDW of 14.68±1.02 in cases compared to controls 10.71±0.48. Similar study conducted by Assiri et al also showed significant increase in PDW among cases (15.88±1.5) compared with controls (11.96±1.8). Similarly PDW was also significantly increased among cases with CPK-MB>30 (11.5±2.0) compared to those with CPK-MB<30 (10.2±1.6).

SUMMARY AND CONCLUSIONS

- This study was undertaken in ShriB.M.Patil Medical College, Bijapur, to study the efficacy and role of platelet parameters in Acute Coronary syndromes.
- A total of 200 cases were studied and were divided further into two groups of cardiac and non-cardiac depending upon characteristics of chest pain and the investigations.
- Among the 100 cases of cardiac chest pain 22 were diagnosed as ST-Elevation Myocardial Infarction, 55 were of Non-ST-Elevation Myocardial Infarction and 23 were diagnosed as Unstable angina.
- Majority of the cases belonged to the age group of 50 to 70 years (55.7%) followed by fifth decade of life (24.6%).
- In the study, total number of male including both cases and controls were 127 (63.8%) and total numbers of females were 73 (36.2%).
- Among total of 100 cases of acute coronary syndrome in the study 58 were males and females were 42.
- The prevalence of smoking in the study population was found to be 27.5%. The highest prevalence of smoking was noted among STEMI (50.9%) followed by NSTEMI (28.6%) and in unstable angina (21.7%).
- Prevalence of hypertension in the study group was found to be 11% and was seen in 29.1% among cases with STEMI and in 14.3% of patients with NSTEMI. Whereas hypertension was seen in only 4.3% among cases with unstable angina and in 2 % among controls.
- The prevalence of Diabetes in the study population was found to be 18%. The study showed prevalence of diabetes was maximum in NSTEMI with 38.1%

followed by STEMI 32.7%. Prevalence of diabetes among unstable angina was found to be 13.0% and among controls 7.0%.

- Prevalence of dyslipidaemia was seen in 53% among cases of acute coronary syndrome, whereas dyslipidaemia was found in only 10% of controls.
- In our study ST-Elevation was seen in 55 cases of which 26 cases had ST-Elevation in Inferior wall leads followed by 18 cases with Anteroseptal and 11 cases had ST-Elevation in lateral wall leads. ST-Depression was seen in 41 cases among which 21 cases were diagnosed with NSTEMI and 20 cases were diagnosed with Unstable Angina.
- In the study we found MPV to be highly significant for both STEMI and NSTEMI with p value ($p<0.001$) control in comparison to patients with unstable angina.
- In our study it was observed that the association between Mean Platelet Volume and cardiac Troponin T was statistically significant ($p<0.001$) for STEMI and NSTEMI group as these group had noticed a larger value of MPV and had cardiac enzyme Troponin T positive.
- In our study the CPK-MB was significantly increased in cases with STEMI with mean valve of 92.9±77.8 followed by NSTEMI 62.5±60.2, whereas in non-cardiac controls CPK-MB was 20.3±12.2.
- Our study showed that MPV was significantly in cases with significant CPK-MB levels. The MPV among cases with CPK-MB>30 was 11.5±2.2 as compared to those with CPK-MB<30 (10.3±1.4). Thus MPV was significantly increased among cases with elevated CPK-MB levels.
- PDW was also significantly increased among cases with CPK-MB>30 (11.5±2.0) compared to those with CPK-MB<30 (10.2±1.6).

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ANNEXURE I

ETHICAL COMMITTEE CERTIFICATE

B.L.D.E.UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR – 586103
INSTITUTIONAL ETHICAL COMMITTEE

No/58/2015
20/11/15

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 17-11-2015 *at* 03 pm *scrutinize the Synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has accorded Ethical Clearance.*

Title "Comparative study on platelet indices in patients with cardiac and non cardiac chest pain."

Name of P.G. Student : Dr Shivanand. S. Mulakuri
Dept of Medicine

Name of Guide/Co-investigator : Dr. Vijaykumar G. Warad
Professor.

DR.TEJASWINI VALLABHA
CHAIRMAN
CHAIRMAN
Institutional Ethical Committee
BLDEU's Shri B.M. Patil
Medical College,BIJAPUR-586103.

Following documents were placed before E.C. for Scrutinization:
1)Copy of Synopsis/Research Project
2)Copy of informed consent form.
3)Any other relevant documents.

ANNEXURE II

INFORMED CONSENT FORM:

TITLE OF RESEARCH: **"Comparative study of platelet indices in patients with cardiac and non cardiac chest pain"**

GUIDE : DR VIJAYKUMAR G WARAD

M.D GENERAL MEDICINE

P.G.STUDENT : DR SHIVANAND MULAKURI

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

PURPOSE OF STUDY:

I have been informed that the purpose of this study is to study diagnostic importance of platelet volume indices in patients with acute chest pain suggesting acute coronary syndrome.

PROCEDURE:

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

BENEFITS:

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

CONFIDENTIALITY:

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

REQUEST FOR MORE INFORMATION:

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

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

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

(Signature of Guardian) (Signature of patient)

ANNEXURE III

B.L.D.E UNIVERSITY

SHRI B. M. PATIL MEDICAL COLLEGE VIJAYAPURA, KARNATAKA

PROFORMA

SCHEME OF CASE TAKING

Name: CASE NO:

Age: OP/IP NO:

Sex: DOA:

Religion: DOD:

Occupation:

Address:

Presenting complaints with duration:

History of presenting complaints:

Past History:

Family History:

Personal History:

Treatment History:

General Physical Examination

Pallor: present/absent

Icterus: present/absent

Cyanosis: present/absent

Clubbing: present/absent

Generalized lymphadenopathy: present/absent

Odema: present/absent

VITALS:

PR:

BP: in mm of mercury (mm hg)

RR:

Temp:

SYSTEMIC EXAMINATION:

- Cardiovascular system
- Respiratory system
- Per abdomen
- Central nervous system

INVESTIGATIONS

PATHOLOGY:

1.)Complete blood count:	
Hb	gm/dl
Total count	Cells/cumm
Differential count Neutrophils	%
Lymphocytes	%
Eosinophils	%
Basophils	%
Monocytes	%
2.) ESR	At the end of 1st hour.
3.)Platelet Indices	
Platelet Count	
Mean Platelet Volume	
Platelet Distribution Width	

4.) Urine Routine	
Sugar	
Albumin	
Cell type	
Cell count	
5.) Peripheral Smear Study	

BIOCHEMISTRY:

1)Troponins	
2)CPK-MB	
3)Fasting Blood sugar	
4)Postprandial Blood sugar	
5)LIPID PROFILE	
Total Cholesterol	
Triglycerides	
HDL-Cholesterol	
LDL-Cholesterol	
VLDL-Cholesterol	

ELECTROCARDIOGRAPHY:

Other relevant investigations will be done when required.

CONCLUSION:

DATE: SIGNATURE

KEY TO MASTERCHART

LIPID PROPILE

1	NORMAL
2	DYSLIPIDAEMIA

ECHOCARDIOGRAPHY

NOR	NORMAL STUDY
IHD	ISCHEMIC HEART DISEASE

ELECTROCARDIOGRAPHY

NOR	NORMAL ECG
STE	ST SEGMENT ELEVATION
STD	ST SEGMENT DEPRESSION

DIAGNOSIS

NC	NON CARDIAC CHEST PAIN
UA	UNSTABLE ANGINA
NSTEMI	NON-ST ELEVATION MYOCARDIAL INFARCTION
STEMI	ST ELEVATION MYOCARDIAL INFARCTION

PAST HISTORY

S	SMOKING
D	HYPERTENSION
H	DIABETIC

MASTER CHART

S.No	IP NO	NAME	SEX	AGE	PC	PDW	MPV	WBC	HBG	TROP T	CPK MB	LIPID PROFILE	FBS	PPBS	ECG	ECHO	DIAGNOSIS	PAST HISTORY
1	26720	PRABHU PARANDE	M	52	2.15	10.8	9.7	8.1	14.4	POS	181	2	129	145	STE	IHD	STEMI	S
2	25633	BHIMARAYA AVARADI	M	48	2.37	15	11.8	9.8	15.3	NEG	33	2	167	224	NOR	NOR	UA	S,D
3	27155	GULAMBI HORAKERI	F	75	3.7	10.2	9.5	10.5	11.6	POS	174	2	130	146	STE	IHD	STEMI	S,H
4	27306	SHIVRAJ HOSANUR	M	47	2.56	11.3	9.9	7.9	11.9	NEG	10	1	106	140	NOR	NOR	NC	S
5	27131	MALAKUBAI INDI	F	62	2.43	9.4	9	11.2	11.5	NEG	73	2	108	165	STD	IHD	UA	S,H
6	23973	SHANTA KHYADI	F	70	1.89	13.6	11.2	13.7	12.8	POS	56	2	123	142	STE	IHD	STEMI	S
7	23991	WALABAI LAMANI	F	60	3.75	12.7	10.9	10.7	9.7	NEG	27	2	116	150	NOR	NOR	UA	
8	23865	KALAVATI PATIL	F	65	2.93	10.1	9.7	15.3	12.1	POS	46	2	168	260	STE	IHD	STEMI	S,D
9	24116	SHARADA HONAMATTI	F	36	2.01	10.1	9.8	7.6	12.9	NEG	20	2	230	269	STD	IHD	UA	
10	26722	RAJU BADIGER	M	38	1.6	9.4	8.5	9.7	12.4	NEG	16	1	96	110	NOR	NOR	NC	
11	27070	GOLLANAGOUDA PATIL	M	40	3.2	11.3	9.9	7.1	13.2	NEG	34	1	143	168	STD	IHD	UA	S
12	26903	BALAWWA PATED	F	68	2.6	11.9	9.9	11.2	11.1	POS	112	1	90	126	STE	IHD	STEMI	S,H
13	27347	DHAMOJI KADAM	M	70	2.18	10.7	9.1	11.9	11.2	NEG	37	1	98	130	NOR	NOR	NC	S
14	15015	KASHIMSAB DODAMANI	M	85	2.6	12.4	10.1	12.4	10.4	POS	48	1	125	152	STD	IHD	NSTEMI	
15	25577	ABDULRAZAQ GANI	M	67	3.09	17.2	12.5	11.9	15.3	POS	47	2	138	265	STE	IHD	STEMI	S,D
16	15379	ISHWARAMMA SEETARAM	F	48	3.02	13.9	11.3	13.7	13.1	POS	45	2	260	354	STE	IHD	STEMI	S,D,H
17	23994	UMESH SAJJANAR	M	57	2.5	13.4	10.8	16.1	13.1	POS	67	2	185	350	STE	IHD	STEMI	S
18	15612	BADESAB MASARKAR	M	65	2.82	11.3	10.8	15.2	13.1	NEG	13	1	110	120	NOR	NOR	UA	S,H
19	15548	SURESH SINDUR	M	52	1.98	11.1	10.1	6.1	13.3	NEG	47	2	106	117	NOR	NOR	UA	
20	15559	MAKAPPA METRI	M	48	2.7	10.9	8.9	11.9	11.2	NEG	35	2	102	135	NOR	NOR	NC	S
21	16221	BASAPPA JAMAKANDI	M	48	2.94	8.8	8.9	5.6	13.7	NEG	58	1	137	145	STE	IHD	STEMI	S
22	23649	KHITAB SHAIKH	M	65	1.57	11.6	10.5	13.1	10.6	POS	218	2	196	160	STE	IHD	STEMI	S,H
23	16218	NINGANNA HUGAR	M	40	2.63	9.4	8	14.5	10.4	NEG	39	1	90	135	STD	NOR	UA	
24	15396	SIDDAPPA JIDDIMANI	M	60	3.3	9.7	9.3	9.3	10.7	NEG	31	1	90	110	NOR	NOR	NC	
25	24046	MAKTAMSAB KALADAGI	M	60	1.6	14.8	11.4	8.1	15.5	NEG	28	2	310	350	NOR	NOR	NC	S,D
26	16186	SHANTAGOUDA BIRADAR	M	40	1.5	9.7	10.4	14.1	12.9	POS	60	1	190	162	STE	IHD	STEMI	S
27	16193	GANGABAI BADNI	F	78	2.4	12.2	10.5	10.8	10.7	NEG	31	2	260	320	STD	NOR	UA	
28	15210	NAGAPPA NYAMGOUDAR	M	75	1.61	10.1	13.4	19.2	13.5	NEG	16	1	104	125	NOR	NOR	NC	S
29	27336	RAFIQ NIMBAL	M	48	1.39	11	8.9	14.4	15.6	NEG	41	2	140	285	STD	IHD	UA	S
30	27444	MAHADEVI PATIL	F	64	3.2	9.3	10.1	12.1	11.8	POS	77	2	124	135	STE	IHD	STEMI	
31	25796	KRISHNA BHIMU CHAUVAN	M	55	1.1	9.3	9.2	4.6	14.5	NEG	12	2	148	239	NOR	NOR	NC	D
32	25749	SHANTABAI DUNDAPPA	F	65	2.31	11.1	10.3	9.2	11.5	NEG	40	1	106	140	STE	IHD	STEMI	
33	25663	BABU BANERJEE	M	80	1.5	9.1	9.2	4.9	9.2	NEG	15	1	115	105	NOR	NOR	NC	
34	25633	BASAPPA RUGI	M	50	2.2	9.8	9.3	18.4	14.9	NEG	48	1	110	123	NOR	NOR	NC	
35	25627	NINGAWWA MUDAKALLI	F	70	3.8	12.7	11.1	5.8	6.7	POS	35	1	130	156	STE	IHD	STEMI	
36	25530	MAHADEVI DALAWAI	F	50	2.11	10.5	10.4	6.5	10.5	NEG	15	1	120	135	NOR	NOR	NC	
37	25353	JAKKAWWA JAKATI	F	65	2.9	11.7	10.4	7.6	10.7	POS	60	2	180	216	STD	IHD	NSTEMI	

38	26414	KASTURI HUBBALLI	F	62	1.72	18.1	16.2	11.3	13.3	POS	35	2	210	327	STE	IHD	STEMI	D
39	26378	KALAVATI BANGERI	F	45	1.13	11.7	10.2	12.5	11.6	NEG	52	1	116	142	STD	IHD	NSTEMI	
40	26388	BASHASAB USTAD	M	60	2.7	18.3	15.2	10.2	14.3	NEG	63	2	104	132	STD	IHD	UA	
41	20852	SHREKANTH MALI	M	45	1.16	10.4	10.6	9.6	10.2	POS	75	1	142	156	STE	IHD	STEMI	
42	20845	KAMALABAI POWAR	F	42	2.15	9.2	9	5.6	12.1	NEG	10	1	110	116	NOR	NOR	NC	
43	19642	PAVITRAMMA PATIL	F	63	2.19	14.1	11.1	11.8	12.9	POS	90	1	149	156	STD	IHD	NSTEMI	D
44	8011	GIRIMALLAPPA UMAGAR	M	65	2.59	8.8	9.1	12.8	12.4	POS	41	1	86	112	STE	IHD	STEMI	
45	8132	SURESH PATIL	M	40	2.2	11.4	10.2	5.4	14.1	NEG	10	1	112	130	NOR	NOR	NC	
46	8166	SHIVAJI SHINDE	M	36	2.6	10.1	9.8	9.4	12.9	NEG	30	1	110	116	STD	IHD	UA	
47	26581	HANUMANTHRAYA M	M	56	2.6	12.4	10.4	12.6	13.5	NEG	34	2	105	136	STE	IHD	STEMI	
48	24220	DAYANAND PATIL	M	62	2.2	9.2	9.1	7.5	10.5	NEG	15	1	110	139	NOR	NOR	NC	D
49	23888	LAKAPPA MADANALLI	M	59	1.7	9.4	9.2	10.2	12.4	NEG	30	1	130	152	NOR	NOR	NC	
50	23854	BHIMANAGOUDA PATIL	M	46	2.5	9.8	9.2	4.6	10.8	NEG	25	1	90	96	NOR	NOR	NC	
51	23880	ARUSUBAI TALWAR	F	60	2.4	10.1	9.4	8.6	11.4	NEG	10	1	125	110	NOR	NOR	NC	
52	8409	SHANTABAI JIGAJINI	F	52	2.51	9.8	9.5	11.9	9.9	POS	40	1	149	152	STE	IHD	STEMI	
53	8241	VITAL GUNNAPUR	M	75	2.7	14.5	11.5	7.2	12.9	NEG	19	1	120	116	STD	IHD	UA	
54	85561	SAROJA PATIL	F	38	2.65	9.8	9.5	12.1	9.9	NEG	15	1	142	130	NOR	NOR	NC	
56	8829	SHIVANAND MADHABHAVI	M	56	3.9	10.7	9.7	15.5	14.1	NEG	26	1	98	136	STD	IHD	UA	
57	24221	SIDDAMMA AGALIMATH	F	75	1.7	10.6	9.7	6.6	12.1	NEG	20	2	146	270	STD	IHD	UA	D
58	10721	SHARANAPPA KURBAR	M	36	2.9	10.4	10.2	7.9	13.4	NEG	29	1	119	135	NOR	NOR	NC	
59	10420	BHEEMABAI WALIKAR	F	65	3.4	9.5	9.5	14.6	11.6	POS	300	2	105	130	STE	IHD	STEMI	
60	12668	MUTTURAJ TELAGI	M	44	3.3	10.1	9.4	6.8	14.7	NEG	26	2	205	256	STD	IHD	NSTEMI	D
61	11600	MALLANAGOUDA PATIL	M	70	1.5	10.2	10.4	8.1	12.2	POS	25	1	136	160	STD	NOR	UA	
62	14485	SUSHILABAL NATEKAR	F	60	3.61	10.5	10.4	12.5	11.5	POS	55	2	126	140	STE	IHD	STEMI	
63	11075	YALLAPA WALIKAR	M	50	2.25	11.2	10.9	9.1	13.9	POS	60	1	122	130	STE	IHD	STEMI	
64	9406	SIDDAPPA PUJERI	M	60	2.3	18.5	12.7	15.8	10.9	POS	46	2	312	245	STE	IHD	STEMI	D,S
65	10754	KAMALABAI GUDALE	F	60	2.9	10.2	11.2	12.1	11.5	POS	35	2	205	269	STE	IHD	STEMI	D
66	13503	SHANKAREMMA BIRADAR	F	60	2.25	14.1	11.2	10.8	11.5	NEG	31	1	138	178	STE	IHD	STEMI	
67	12996	MALLANAGOUDA PATIL	M	63	2.71	9.2	9.6	8.7	13.2	NEG	29	1	116	128	NOR	NOR	NC	S
68	11399	GOLAPPA NARAGUND	M	87	2.64	12.1	10.6	12.2	10.5	POS	114	1	104	116	STE	IHD	STEMI	S
69	11608	MAHENDRA JOLLI	M	50	2.5	10.3	9.4	11.6	14.1	POS	225	1	112	130	STE	IHD	STEMI	
70	11881	ANNAPURNA BARDOL	F	58	2.88	10.4	12.6	12.5	8.5	POS	52	1	140	160	STE	IHD	STEMI	
71	13046	RANGOND KOLKAR	M	41	2.21	11.5	10.4	9.1	14.1	NEG	34	2	96	130	STD	IHD	NSTEMI	
72	13107	MALLANAGOUDA PATIL	M	40	3.39	11.6	10.1	5.5	12.8	NEG	12	2	90	106	NOR	NOR	NC	
73	15333	RAYAMMA NAGANUR	F	65	3.4	11.8	10.5	11.5	12.1	NEG	29	1	104	110	STD	IHD	NSTEMI	
74	15173	MALLANAGOUDA BIRADAR	M	60	2.16	11.5	10.8	9.1	10.4	NEG	41	1	126	140	STD	IHD	UA	
75	15095	VIJAYKUMAR HOSMANI	M	54	2.56	15.9	11.8	11.1	12.3	NEG	29	2	346	446	STD	IHD	UA	D
76	16147	MAHAMED AIGALI	M	48	2.02	12.8	10.6	16.4	11.3	POS	50	1	156	120	STE	IHD	STEMI	
77	15347	SAKKUBAI PAKIR	M	60	3.4	11.8	10.5	11.5	10.2	NEG	17	1	102	145	NOR	NOR	NC	
78	15343	SHRISHAIL HOSMANI	M	42	3.1	10.2	9.6	9.1	13.8	NEG	13	1	121	125	STD	NOR	UA	
79	19947	PRAKASH KORALI	M	35	1.7	9.4	9.1	8.6	10.4	NEG	15	1	116	140	NOR	NOR	NC	
80	18523	PARVATI SHETTY	F	60	2.8	11.7	10.5	11.4	8.8	POS	57	2	438	480	STD	IHD	NSTEMI	D

81	26808	BABUSAB BADEMANGOL	M	47	2.9	10.9	9.9	12.4	15.5	NEG	10	1	87	106	NOR	NOR	NC	S
82	18502	MAYAPPA HALASANGI	M	52	2.35	13.1	11.1	12.9	14.5	POS	109	2	125	216	STE	IHD	STEMI	D,S
83	18108	MAHALINGAYYA PUJERI	M	55	3.12	10.8	9.8	13.6	12.7	POS	42	1	150	176	STE	IHD	STEMI	D,S,H
84	18534	DEVAPPAGOUDA KANTA	M	56	2.5	12.6	11.9	11.5	13.4	POS	60	1	202	246	STE	IHD	STEMI	D,S,H
85	18446	PUJAPPA PUJERI	M	64	2.3	8.3	8.6	7.4	13.9	NEG	19	1	116	150	NOR	NOR	NC	S
86	18855	CHANDABEE NADAF	F	60	2.53	9.4	8.6	10.8	12.9	NEG	17	1	165	140	STD	IHD	NSTEMI	
87	29087	BORAWWA KOLEKAR	F	70	1.84	12.4	10.5	6.9	8.9	POS	59	2	146	180	STE	IHD	STEMI	
88	28271	SHERANU ASKI	M	38	1.64	9.2	9.4	7.5	10.7	NEG	10	1	118	132	NOR	NOR	NC	
89	27521	YALLAPPA MANE	M	42	3.09	9.7	9.4	8.7	14.4	NEG	30	1	268	253	STD	NOR	UA	S,D
90	27595	SIDDARAM JAGADISH	M	42	2.07	12.1	10.4	8.1	11.4	NEG	14	1	105	107	NOR	NOR	NC	
91	27576	MALLAMMA WATAGOD	F	60	2.4	12.3	11	14.8	10.2	NEG	12	1	109	116	NOR	NOR	NC	
92	27617	AFSARA MEHABOOBKADAR	F	60	2.89	9.9	9.5	7.2	12	NEG	24	1	115	124	NOR	NOR	NC	
93	27619	PARAPPA BIRADAR	M	56	2.35	14.3	16.5	12.6	10.7	POS	31	2	154	126	STE	IHD	STEMI	S,H
94	27679	SATTEWWA MANGULI	F	40	2.6	18.9	15.1	7.6	12.7	NEG	22	1	110	132	STD	NOR	UA	
95	27690	BASAYYA SARAWARMATH	M	42	3.16	14	12.5	14.3	14.5	POS	57	2	110	162	STE	IHD	STEMI	S
96	27670	DANAMMA RAICHUR	F	46	2.26	9.2	9.4	9.6	11.4	NEG	10	1	156	210	NOR	NOR	NC	D
97	25748	SHANTABAI PUJERI	F	65	2.31	11.1	10.3	9.2	11.5	POS	40	2	125	106	STE	IHD	STEMI	
98	29104	GURULINGAPPA BIRADAR	M	84	3.44	10.3	11.4	12.4	13.5	POS	300	1	140	135	STE	IHD	STEMI	S,D
99	29130	MALLIKARJUN BIRADAR	M	47	2.09	11.8	10.3	4.6	13.3	NEG	15	1	126	112	STE	IHD	STEMI	S
100	29051	SHANKARAPPA KALAPPA	M	61	1.98	14.4	11.7	17.8	11.1	POS	300	2	145	206	STE	IHD	STEMI	S,D,H
101	28886	RAJU RATHOD	M	40	1.65	9.5	8.7	5.4	12.6	NEG	16	1	109	130	NOR	NOR	NC	
102	28733	LAXMAWWA NERIKAT	F	70	4.3	12.5	10.6	13.4	9.7	NEG	10	1	128	170	NOR	NOR	NC	
103	28728	CHINAWWA JINJARWADI	F	65	2.54	13.5	10.9	7.8	12.7	POS	45	1	105	124	STD	IHD	NSTEMI	
104	28716	SAHEBGOUDA ELAGI	M	42	3.14	9.7	9.4	9.2	10.4	NEG	10	1	104	125	NOR	NOR	NC	S
105	28723	SHIVABAI HOSAMANI	F	78	2.1	9.4	10.2	4.9	12.7	NEG	22	1	76	118	NOR	NOR	NC	
106	28667	MAMU RATHOD	M	68	1.91	9.5	11.1	2.8	7.7	NEG	25	1	112	105	NOR	NOR	NC	
107	28584	BASAPPA HADAGALI	M	65	2.4	15	17.6	7.2	13.6	NEG	25	1	116	105	STD	NOR	UA	S
108	28409	RAMJAN HADRI	M	55	2.7	10.6	11.1	17.3	14.7	NEG	31	1	210	250	STD	NOR	UA	S,D
109	6033	MALLIKARJUN JAGATI	M	65	2.36	10.1	10.3	9.7	15.7	POS	70	1	81	110	STE	IHD	STEMI	S,H
110	6111	KALLAPPA BIRADAR	M	58	1.76	9.4	9.2	8.9	12.4	NEG	20	1	116	150	NOR	NOR	NC	
111	2969	KAMALABAI TABBANNAVAR	F	62	2.35	8.9	8.1	14.3	7.8	POS	57	1	121	185	STE	IHD	STEMI	D,H
112	5118	BASAPPA NINGAPPA	M	55	2.45	11.1	11.3	10.4	11	POS	45	2	154	112	STE	IHD	STEMI	
113	24110	ANAND RATHOD	M	45	2.4	8.6	9.9	8.6	10.4	NEG	12	1	104	140	NOR	NOR	NC	
114	19069	MAHADEVAPPA MADAR	M	55	1.4	9.2	9.7	11.2	9.8	NEG	16	1	98	125	NOR	NOR	NC	
115	15360	GADDAPPA GULED	M	60	2.15	10.4	11.6	4.7	12.3	NEG	48	1	110	134	NOR	NOR	NC	
116	7989	ALLISAB DALAL	M	65	3.8	12.6	10.6	11.5	13.4	POS	95	2	120	136	STD	IHD	NSTEMI	S,D,H
117	20844	SIDDAPPA HOSAMANI	M	76	2.4	11.8	12.6	6.7	10.2	POS	110	2	114	128	STE	IHD	STEMI	S,H
118	25852	RATNABAI CHAVAN	F	65	3.6	12	10.7	13.3	8.6	NEG	30	1	90	106	NOR	NOR	NC	
119	25933	BABU BIRADAR	M	45	1.7	8.6	9.2	4.5	10.5	NEG	10	1	104	110	NOR	NOR	NC	
120	26088	SHIVABASAPPA BIRADAR	M	76	1.46	9.2	9.1	7.8	10.2	NEG	16	1	110	136	NOR	NOR	NC	
121	26214	SANGANAGOUDA	M	64	1.76	12.1	14.2	10.2	11.6	POS	88	2	82	135	STE	IHD	STEMI	S,H
122	25336	SHRIMANTH DURGI	M	50	2.5	9.4	9.8	4.6	12.5	NEG	12	1	120	132	NOR	NOR	NC	

123	25213	SEETAWWA MADAR	F	42	3.7	11.5	10.4	15.1	9.8	NEG	21	1	97	140	STD	NOR	UA	
124	15173	MALLANGOUDA BIRADAR	M	60	2.16	10.8	11.5	9.1	10.4	NEG	41	1	126	140	STD	IHD	NSTEMI	S,D
125	25796	KRISHNA CHAUVAN	M	55	1.18	9.1	9.5	5.6	11.5	NEG	12	2	239	148	NOR	NOR	NC	D
126	24851	KASIBAI	F	65	2.4	10.1	11.4	9.7	12.5	NEG	16	1	124	150	NOR	NOR	NC	
127	18796	CHANDRABAI	F	60	2.6	11.2	12.1	8.6	11.5	NEG	10	1	116	124	NOR	NOR	NC	
128	17256	MALAKAPPA	M	55	1.7	7.4	8.2	6.4	10.5	NEG	11	1	135	122	NOR	NOR	NC	
129	21359	TIPPANNA	M	65	2.5	9.6	9.7	9.6	12.2	NEG	22	1	120	103	NOR	NOR	NC	
130		JAYASHREE	F	70	2.9	10.4	10.8	11.5	10.6	POS	56	1	126	110	STE	IHD	STEMI	
131		RATNABAI CHAVAN	F	64	3.4	11.4	11.6	10.1	12.8	NEG	45	1	90	130	STD	NOR	UA	
132	24301	HANUMANTH KESU	M	70	2.2	9.4	9.2	5.4	14.6	NEG	10	1	115	120	NOR	NOR	NC	
133	26021	PRABHAVATI	F	56	1.4	10.2	10.4	8.5	11.2	NEG	20	1	102	119	NOR	NOR	NC	S
134	29130	MALLIKARJUN	M	47	1.7	11.2	10.4	8.6	10.6	NEG	18	1	110	140	NOR	NOR	UA	
135	29051	SHANKARAPPA NETIKAR	M	61	2.5	11.5	11.9	7.6	12.5	NEG	15	1	106	132	STD	NOR	UA	
136	17084	PRABHAVATI GOUNDI	F	40	3.14	8.5	8.4	13.7	15.2	NEG	14	1	126	145	NOR	NOR	NC	
137	17263	ARANABAI KALE	F	46	3.21	10.1	10.6	6.8	12.2	NEG	21	1	139	145	STD	NOR	UA	
138	17266	DEEPAK SHINTRE	M	55	2.68	9.1	10.1	6.7	16.6	NEG	18	1	102	132	NOR	NOR	NC	
139	17477	VENKAPPA GADYAL	M	65	2.07	9.3	9.5	5.5	12.4	NEG	10	1	114	120	STD	NOR	UA	
140	17793	MOHAMMAD AIGALI	M	48	4.7	9.6	10.5	19.4	12.6	NEG	29	1	498	192	STD	NOR	UA	S,D
141	17831	YANKAWWA HANCHINAL	F	80	3.07	10.8	12.3	17.6	8.2	POS	234	2	89	106	STE	IHD	STEMI	
142	17878	RAMANAGOUDA PATIL	M	72	2.39	10.2	11.4	9.6	14.2	NEG	17	1	128	135	STD	IHD	NSTEMI	S,H
143	17960	ADIVEPPA MAVINGIDAD	M	48	3.18	10.8	13.2	9.5	13.2	NEG	30	2	310	453	STD	IHD	NSTEMI	S,D
144	18082	VENKATACHALAPATI	M	62	2.54	9.6	9.8	5.6	12.5	NEG	13	1	292	124	NOR	NOR	NC	S,D
145	18085	RAM ASANGI	M	70	2.32	9.8	9.8	6.8	10.4	NEG	14	1	91	153	NOR	NOR	NC	S
146	18348	BANDABAI PATIL	F	70	3.5	10.4	11.6	5.4	11.5	NEG	14		83	120	NOR	NOR	NC	
147	18635	NABILAL MAKANDAR	M	48	2.21	10.1	11.2	8.2	14.5	NEG	23	1	108	125	STD	NOR	UA	
148	18689	RUKAMAWWA GUGARI	F	50	2.5	9.1	9.4	6.7	12.5	NEG	10	1	86	142	NOR	NOR	NC	
149	18911	SHEKAPPA MADABHAVI	M	45	2.25	9.3	9.5	7.5	13.9	NEG	42	1	106	110	STD	IHD	NSTEMI	
150	19233	SHIVABAI BIRADAR	F	65	3.05	9.9	10.6	16.2	10.9	NEG	19	1	116	113	NOR	NOR	NC	
151	19235	MEHABOOBSAB MULLA	M	70	2.04	9.9	11.3	13.8	12.5	NEG	21	1	93	124	NOR	NOR	NC	S,H
152	19245	HANAMANTH PALASYAGOL	M	40	3.03	10.3	11.7	7.3	16.5	NEG	27	1	320	165	STE	IHD	STEMI	S,D
153	19380	BASAWWA KOTYAL	F	55	2.89	9.8	10.3	10.6	11.9	NEG	23	1	158	134	STD	NOR	UA	
154	19443	NANAGOUDA PATIL	M	55	1.33	13.1	20.7	16.1	15.9	POS	85	2	125	145	STE	IHD	STEMI	S,H
155	19264	JAGADEVI KUDURE	F	76	1.45	12.5	10.5	14.2	11.6	NEG	95	1	110	126	NOR	NOR	UA	
156	19815	SANGAWWA MALLAPURI	F	70	2.72	10.3	11.4	15.6	11.3	POS	126	2	106	145	STE	IHD	STEMI	H
157	19881	CHANNAMMA BHAVUR	F	60	2.49	10.8	12.2	10.8	12.3	POS	45	2	190	160	STE	IHD	STEMI	D,H
158	19930	BALU PUJERI	M	50	2.45	11.5	10.4	12.5	11.6	NEG	24	1	105	110	NOR	NOR	UA	
159	20140	GURULINGAYYA HIREMATH	M	38	2.3	11.6	14.4	17.6	13.5	NEG	32	2	181	126	STD	IHD	UA	
160	20141	MAHADEVI AVAREKAL	F	50	1.73	10.2	10.5	11.4	6.5	NEG	17	1	110	137	NOR	NOR	NC	
161	20212	MALLAPPA NATEKAR	M	65	2.68	10.6	12.3	14.2	14.2	NEG	17	2	194	225	STD	NOR	UA	
162	20232	BALU RATHOD	M	40	2.7	9.8	11.4	6.2	14.8	NEG	24	1	97	119	NOR	NOR	NC	
163	20345	SONAM BIDRI	F	36	4.1	9.9	11.1	21.1	9.4	NEG	10	1	110	110	NOR	NOR	NC	
164	20387	MALLIKARJUN BIJAPUR	M	65	1.76	11.1	13.6	22.7	15.8	POS	258	2	195	225	STE	IHD	STEMI	D.S

165	20558	KAMALA SHINDE	F	55	3.04	9.8	10.3	10.9	11.6	POS	23	1	180	210	STD	IHD	NSTEMI	
166	20657	KEMPAYYA HIREMATH	M	65	1.9	10.7	12.7	9.6	13.6	NEG	64	2	268	336	STD	IHD	NSTEMI	S,D
167	20668	MAHAVEER SAHAPUR	M	45	3.4	10.9	12.5	14.7	13.7	POS	78	2	124	110	STE	IHD	STEMI	
168	20731	SHARAWWA TALAKERI	F	60	1.82	11.9	16.4	9.6	12.9	POS	79	2	180	136	STE	IHD	STEMI	D,H
169	20852	SHREEKANTH MALI	M	40	2.69	8.3	8.7	18.1	14.8	NEG	287	2	113	120	STD	IHD	NSTEMI	S,H
170	20972	IRAPPA MADEGAR	M	54	1.28	11.9	14.1	10.7	10.2	NEG	38	1	124	127	NOR	NOR	NC	
171	20989	ANWARHUSSAIN YAKEEN	M	49	3.4	8.4	10.2	7.7	13.3	NEG	20	1	110	120	NOR	NOR	NC	S
172	21127	SHIVAPPA MADAR	M	55	1.5	10.1	10.8	7.4	12.9	NEG	35	1	116	167	NOR	NOR	NC	
173	21141	NAGAMMA AMARAPPAGOL	F	60	1.6	9.1	9.8	5.6	10.5	NEG	13	1	130	124	NOR	NOR	NC	
174	21186	BASAMMA METI	F	60	2.85	9.2	9.2	11.5	11.7	POS	249	2	150	338	STE	IHD	STEMI	D
175	21453	VITHAL KEERTI	M	50	2.49	11.1	14.2	10.1	12.6	POS	85	1	115	125	STD	IHD	NSTEMI	
176	21517	PRAJAVATI ANKALAGI	F	55	3.5	9.9	10.4	10.7	11.6	POS	157	1	144	152	STD	IHD	NSTEMI	
177	21537	MALLAMMA PUJERI	F	50	3.5	10.1	11.3	10.6	12.8	NEG	22	1	110	113	NOR	NOR	NC	
178	21535	VAIRABAI BHAIRKADOR	F	60	3.7	8.1	7.6	5.7	11.4	NEG	25	1	110	146	NOR	NOR	NC	
179	21722	JITENDRA HAJERI	43	M	2.4	8.6	9.4	6.8	10.5	NEG	37	1	126	127	NOR	NOR	NC	
180	21729	RAJU LONI	M	40	1.13	10.1	10.6	7.8	12.6	NEG	12	1	130	165	NOR	NOR	NC	
181	21731	BHEEMAGOUDA HUGGI	M	60	2.1	8.5	8.5	9.4	13.6	NEG	15	1	110	127	NOR	NOR	NC	
182	21740	ADIVEPPA WANGADI	M	63	2.5	8.6	9.2	9.4	10.2	NEG	18	1	117	153	NOR	NOR	NC	S
183	21755	REVANSIDDA KOLHAR	M	55	2.4	9.2	10.1	12.1	14.8	NEG	41	2	143	124	NOR	NOR	NC	
184	21872	ANIL NANDRAL	M	46	1.61	10.4	11.8	7.1	13.5	NEG	97	1	116	156	NOR	NOR	NC	
185	21880	KANTAPPA SHIVASHARAN	M	50	2.5	9.6	10.2	11.7	13	POS	60	2	135	146	STD	IHD	UA	
186	21956	PONAMMA SANGOLI	F	65	3.2	11.2	13.1	21.5	11.1	NEG	23	1	170	120	NOR	NOR	NC	
187	22036	SHEKAWWA KALADAGI	F	50	1.5	10.5	11.2	10.6	14.2	NEG	16	1	114	125	NOR	NOR	UA	
188	22217	KAMALABAI BAGEWADI	F	58	2.7	9.6	10.4	12.9	12.5	NEG	20	2	160	225	NOR	NOR	NC	D,H
189	22419	NAGAPPA BALLARI	M	65	2.5	9.2	9.1	6.2	10.8	NEG	15	1	106	110	NOR	NOR	NC	
190	22552	SAVITRI RAVI	F	44	3.04	10	10.9	8.4	12.3	NEG	36	1	95	115	NOR	NOR	NC	
191	22558	BASAVARAJ GOURAGOND	M	42	2.6	9.1	9.5	8.1	10.5	NEG	20	1	120	102	NOR	NOR	NC	
192	22913	MEHABOOBSAB GAGINAL	M	60	2.38	9.9	10.6	5.6	13.1	NEG	36	1	95	136	NOR	NOR	NC	
193	22998	SUREKHA KANCHINAKOTI	F	37	2.7	10.3	11.9	6.1	12.1	NEG	39	1	96	102	STD	NOR	UA	
194	23178	SEETU LAMANI	M	65	3.2	10.6	11.8	13.8	15.1	POS	66	2	126	130	STD	IHD	NSTEMI	D
195	23180	KHUBU POWAR	M	48	2.4	10.1	11.1	9.5	13.7	NEG	26	1	110	140	NOR	NOR	NC	
196	23186	MAHADEV TONSYAL	M	55	2.08	9.2	10.4	11.9	12.7	NEG	24	1	83	125	NOR	NOR	NC	
197	23330	LARASINGH RATHOD	M	75	2.4	12.8	20.2	18.4	7.7	POS	83	1	188	215	STE	IHD	STEMI	S,D,H
198	23456	DANAYYA GANACHARI	M	68	2.6	11	13.2	12.5	14.2	NEG	20	1	130	146	NOR	NOR	NC	
199	23482	BASAVARAJ TALAWAR	M	56	3.9	9.4	10.2	10.3	12.7	NEG	28	1	126	128	NOR	NOR	NC	
200	17908	JAGADEVRAO INAMDAR	M	84	3.1	10.6	11.1	22.7	8.9	NEG	10	1	114	127	NOR	NOR	NC	S