STUDY OF PLATELET INDICES IN PATIENTS WITH ACUTE CORONARY SYNDROMES

By

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Dissertation submitted to the

B.L.D.E. UNIVERSITY BIJAPUR, KARNATAKA

In partial fulfillment of the requirements for the degree of

DOCTOR OF MEDICINE

In

PATHOLOGY

Under the guidance of

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ACKNOWLEDGMENT

This study has been accomplished with the grace of almighty God. It gives me immense pleasure to express my heartfelt gratitude to all. I dedicate this page to each and everyone who have helped me to explore the expanses of knowledge.

A line from Sanskrit Shlokha Says “Guru r brahma guru r vishnu gurudevo maheshwaraha, guru ssakshaat parabrahma tasmay shri gurave namaha” - meaning a teacher is next to god and without him knowledge is always incomplete

I wish to take this opportunity to express my indebtedness to my guide **Dr. R.M.Potekar,** Professor Department of Pathology, for his resolute guidance, precise approach, constructive criticism and meticulous supervision throughout the course of my work and preparation of manuscripts that have been a valuable part of my learning experience

I also wish to thank my Co-guide **Dr. Sharanabasawappa Badiger**, Professor Department of Medicine, for his immense guidance, supervision & approach throughout the study.

I express my sincere gratitude to **Dr. B.R.Yelikar**, Professor and Head, Department of Pathology for his valuable suggestions, indispensable guidance and critical appreciation in pursuit of this study.

A sincere and heartfelt thanks to all the esteemed teachers of the Department of Pathology for their congenial supervision, assiduous concern and positive feedback, which has made it conceivable for me to expedite this dissertation.

I am very grateful to all the non teaching staff of Department of Pathology, who have helped me during this work.

It is with great pleasure I express my sincere gratitude to my parents for their constant encouragement, inspiration and sacrifices.

Last but not the least**,** My sincere gratitude to all my study subjects whose cooperation has contributed to this study.

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ABSTRACT

BACKGROUND: Platelets have been implicated in the pathogenesis of cardiovascular disorders including atherosclerosis and its complications such as acute myocardial infarction, unstable angina and sudden cardiac death. Platelet indices correlates with functional status of platelets and is an emerging risk marker for atherothrombosis **.**

AIM*:* To study efficacy of platelet parameters in Acute Coronary Syndromes**.**

MATERIAL AND METHODS: A prospective hospital based study was carried out on 175 cases diagnosed with Acute Coronary Syndromes and 175 controls from October 2011 to March 2013 considering the inclusion and exclusion criteria**.**

RESULTS*:* The incidence of ACS in males (62.86%) was more as compared to females (37.14%).The average age with which the patient presented with ACS was 57.76 ± 13.19 years. The commonest manifestation of ACS was ST elevation MI.Analysis of PVI indicated MPV & PDW as significant risk factor for developing a myocardial infarction. This was in concordance with the elevated cardiac enzymes levels.

CONCLUSION*:* The study concludes that Platelet Indices especially MPV & PDW is raised in patients who have suffered STEMI & NSTEMI as compared with patients diagnosed with unstable angina

Key words: Acute Coronary Syndrome ,Platelet Indices, Mean Platelet Volume.

LIST OF ABBREVIATIONS USED (In alphabetical order)

ACS	Acute Coronary Syndromes
AMI	Acute Myocardial Infarction
BNP	Brain Natriuretic Peptide
CAD	Coronary Artery Disease
CHD	Coronary Heart Disease
CVD	Cardiovascular Disease
CPK-MB	Creatine Phosphokinase
CRP	C-Reactive Protein
ECG	Electrocardiogram
HDL	High Density Lipoprotein
IHD	Ischaemic Heart Disease
ITP	Idiopathic Thrombocytopenic Purpura
LDL	Low Density Lipoprotein
MPV	Mean Platelet Volume
NSTEMI	Non-ST Elevation Myocardial Infarction
P-LCR	Platelet Large Cell Ratio
PDW	Platelet Distribution Width
PVI	Platelet Volume Indices
STEMI	ST-Elevation Myocardial Infarction
Tr	Troponin
UA	Unstable Angina

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Ischaemic Heart Disease is defined as myocardial impairment due to imbalance between coronary blood flow and myocardial requirement. Cardiovascular diseases accounts for approximately 12 million deaths annually and is the commonest cause of death globally.[1]

Patients with Ischaemic Heart Disease fall into two large groups:

- Patients with stable angina secondary to Coronary Artery Disease
- Patients with Acute Coronary Syndromes

The latter group, in turn, is composed of patients with Acute Myocardial Infarction with ST-segment elevation on their presenting ECG and those with Unstable Angina and Non-segment elevation MI.[2]

Conventional risk factors for atherosclerosis include smoking, diabetes-mellitus, hypertension, hyperlipidemia, obesity and stress which either acting singly or in combination increase the chances of developing coronary atherosclerosis. However, they only explain part of the cases and other relevant risk factors need to be identified for an accurate calculation of an individual's risk for myocardial infarction.

Platelet indices viz – Mean platelet volume (MPV), Platelet distribution width (PDW) and Platelet large cell ratio (P-LCR) have been well utilized for certain conditions like Idiopathic Thrombocytopenic Purpura (ITP), Aplastic anemia and other haemotological and myeloproliferative disorders to assess the prognosis but are underutilized for cardiovascular disorders.[3]

Platelets have been implicated in the pathogenesis of cardiovascular disorders including atherosclerosis and its complications such as acute myocardial infarction, unstable angina and sudden cardiac death.

Platelet hyperactivity and local platelet activation have been suggested to play a role in acute coronary events. Platelet size have been shown to reflect platelet activity which is indirectly measured by the parameters. Larger platelets are metabolically and enzymatically more active than small platelets.[4]

Platelet indices correlates with functional status of platelets and is an emerging risk marker for atherothrombosis.[5]

The most sensitive and specific biomarkers of myocardial damage are Troponin I and Troponin T, levels of both begin to rise at 2 to 4 hours and peak at 48 hours. Creatine Kinase enzymes begins to rise within 2 to 4 hours of the onset of MI,peaks at about 24 hours and returns to normal within approximately 72 hours.[6]

Platelet parameters can be detected earlier as compared to specific and non specific markers of Myocardial Infarction. Platelet indices are easily recorded by automated cell counter and are routinely available in most clinical laboratories. There is scope to make better use of the platelet parameters generated, as patients with larger platelets can easily be identified during routine haematologial analysis and could possibly benefit from timely treatment.[4]

AIM OF THE STUDY

-To study efficacy of platelet parameters in Acute Coronary Syndromes.

REVIEW OF LITERATURE

ISCHEMIC HEART DISEASE : *Ischemic heart disease* (IHD) is a condition in which there is an inadequate supply of blood and oxygen to a portion of the myocardium, it typically occurs when there is an imbalance between myocardial oxygen supply and demand. The most common cause of myocardial ischemia is atherosclerotic disease of an epicardial coronary artery (or arteries) sufficient to cause a regional reduction in myocardial blood flow and inadequate perfusion of the myocardium supplied by the involved coronary artery[2]

Ischemic heart disease is classified as[6]:

- Angina pectoris
- Myocardial infarction (MI)
- Chronic IHD with heart failure
- Sudden cardiac death

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

- Unstable angina
- Acute myocardial infarction
- Sudden cardiac death.

Platelets play a pivotal role in atherothrombosis, the major cause of most unstable coronary syndromes.[7]

Epidemiology of cardiovascular disease :

IHD causes more deaths and disability and incurs greater economic costs than any other illness in the developed world[2]. Each year, more than 17 million people die from cardiovascular disease worldwide[10]. Lifestyles of populations across the world have changed dramatically in the 20th century. These change (collectively termed as epidemiological transition) have been brought about by a number of developments in science and technology that now affect every facet of human existence. In developed nations the rise in the burden of CVD occurred over several decades due to a long period of epidemiological transition. In India, perhaps because of the rapid pace of economic development, epidemiological changes have spanned a much shorter time. As a consequence, cardiovascular disease (CVD) has emerged as the leading cause of death all over India[8]

The pattern of CHD in India has been reported to be as follows[9]-

- CHD appears a decade earlier compared with age incidence in developed countries
- The peak period is attained between 51-60 years
- Males are affected more than females
- Hypertension & Diabetes account for about 40% of all cases.
- Heavy smoking is responsible aetiologically in a good number of cases.

Current estimates from disparate cross-sectional studies indicate the prevalence of CHD to be between 7-13 per cent in urban and 2-7 per cent in rural India[8]. Traditional risk factors play an important role in the excess risk for cardiovascular disease in developing countries, thereby emphasizing the urgent need to develop cost-effective programs to control these risk factors in these settings with limited resources. [10]

PATHOGENESIS OF ACS

The pathogenesis for ACS among patients can be divided into four groups:[11]

1. Atheromatous CHD
2. Non-atheromatous CHD
3. Hypercoagulable states.
4. MI related to substance misuse.

These conditions are characterized by an imbalance between myocardial oxygen supply and demand. The most common mechanisms involve an imbalance that is caused primarily by a reduction in oxygen supply to the myocardium, the imbalance due to increased myocardial oxygen requirements, usually in the presence of a fixed, restricted oxygen supply.

There is reduced myocardial perfusion that results from coronary artery narrowing caused by a thrombus that developed on a disrupted atherosclerotic plaque and is usually non-occlusive.

Microembolization of platelet aggregates and components of the disrupted plaque are believed to be responsible for the release of myocardial markers in many of these patients.

VASCULAR INJURY AND CORONARY ATHEROGENESIS

Vascular injury and thrombus formation are key events in the origin and progression of atherosclerosis and in the pathogenesis of Acute Coronary Syndromes.

The proposed pathophysiologic classification of vascular injury divides it into 3 types-

Type I- Functional alterations of endothelial cells with no morphological change

Type II -Endothelial denudation and intimal damage with intact internal elastic lamina

Type III-Endothelial denudation with damage to intima and media

The chronic minimal injury to the arterial endothelium in spontaneous atherosclerosis is caused mainly by a disturbance in the pattern of blood flow at bending points and areas near branching vessels, hypercholesterolemia, circulating vasoactive amines, immunocomplexses, infections & chemical irritants like tobacco smoke which cause Type I injury leading to accumulation of lipids and monocytes (macrophages)[12].

Release of toxic products by macrophages leads to Type II damage characterized by adhesions of platelets. Macrophages and platelets together release mitogens from alpha-granules which act as growth factors for fibroblasts leading to migration and proliferation of smooth muscle cells. These cells form collagen fibrils,

proteoglycans, mucopolysaccharides and elastin fibres leading to a fibrointimal lesion, or occasionally an outer capsule on a predominantly lipid lesion[13].

A lipid lesion surrounded by a thin capsule may be easily disrupted, leading to Type III damage with a thrombus formation. When thrombi are small, they can become organized and contribute to the growth of the atherosclerotic plaque. When thrombi are large and occlusive, they contribute to Acute Coronary Syndromes such as unstable angina, myocardial infarction and sudden death[14].

MORPHOLOGY OF CORONARY ATHEROSCLEROSIS

Stary has classified the sequential changes in atherosclerosis. Presumably, as a result of Type I injury of endothelial cells, isolated macrophages or foam cells in the intima were the earliest sign of lipid retention (Stary I lesion).With progression, a more substantial number of macrophages or foam cells were found accompanied by smooth muscle cells also containing lipid droplets and by minimal, scattered extracellular lipid (Stary II lesion) appearing macroscopically on Sudan IV staining as a flat or slightly raised fatty streak. This on progression showed multiple extracellular lipid cores (Stary III lesions) appearing as a raised fatty streak or an atheroma, characterized by a single confluent extracellular lipid core (Stary IV lesion).The Stary III & IV lesions are not surrounded by a fibrotic cap. On further progression, some of these lesions become predominantly fibromuscular whereas others become fibrolipid and are characterized by a cap of smooth muscles and collagen surrounding multiple lipid cores or a single lipid core (Stary V lesion). [15]

PROGRESSION OF ATHEROSCLEROSIS

Endothelial Dysfunction in Atherosclerosis.:

The earliest changes that precede the formation of lesions of atherosclerosis take place in the endothelium. These changes include increased endothelial permeability to lipoproteins and other plasma constituents which is mediated by nitric oxide, prostacyclin, platelet-derived growth factor, angiotensin II endothelin up-regulation of leukocyte adhesion molecules including L-selectin, integrins, and platelet endothelial cell adhesion molecule-I(PECAM-1), the up-regulation of endothelial adhesion molecules which include E-selectin, P-selectin, Intercellular adhesion molecule-I (IAM-1), Vascular cell adhesion molecule-1(V-CAM-I) and migration of leukocytes into the artery wall which is mediated by oxidized Low-density lipoprotein, Monocyte chemotactic protein-1(MCP-I), Interleukin-8, Platelet-derived growth factor, Macrophage colony stimulating factor and osteopontin.

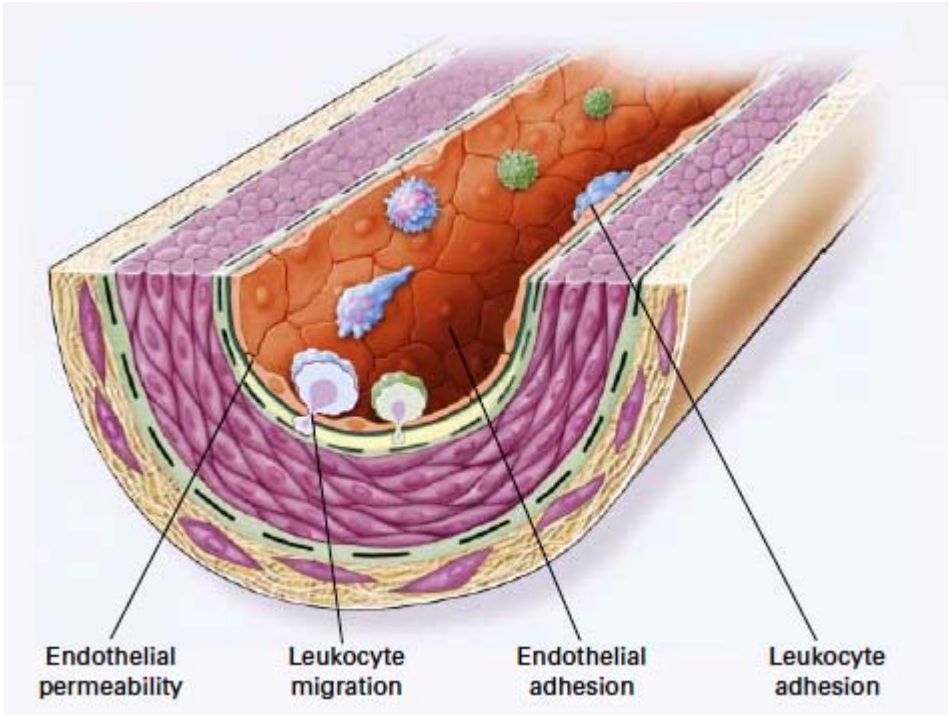


FIG 1: ENDOTHELIAL DYSFUNCTION IN ATHEROSCLEROSIS.

Fatty-Streak Formation in Atherosclerosis.

Fatty streaks initially consist of lipid-laden monocytes and macrophages (foam cells) together with T lymphocytes. Later they are joined by various numbers of smooth-muscle cells. The steps involved in this process include smooth-muscle migration which is stimulated by Platelet-derived growth factor, Fibroblast growth factor-2 and Transforming growth factor -β. T-cell activation which is mediated by Tumor Necrosis Factor- α, Interleukin-2 and Ganulocyte–macrophage colony-stimulating factor. Foam cell formation which is mediated by oxidized Low-density lipoprotein, Macrophage colony-stimulating factor, Tumor necrosis factor -α, Interleukin-1 and platelet adherence and aggregation which are stimulated by integrins, P-selectin, fibrin & Thromboxane A2 tissue factor which is responsible for the adherence and migration of leukocytes.

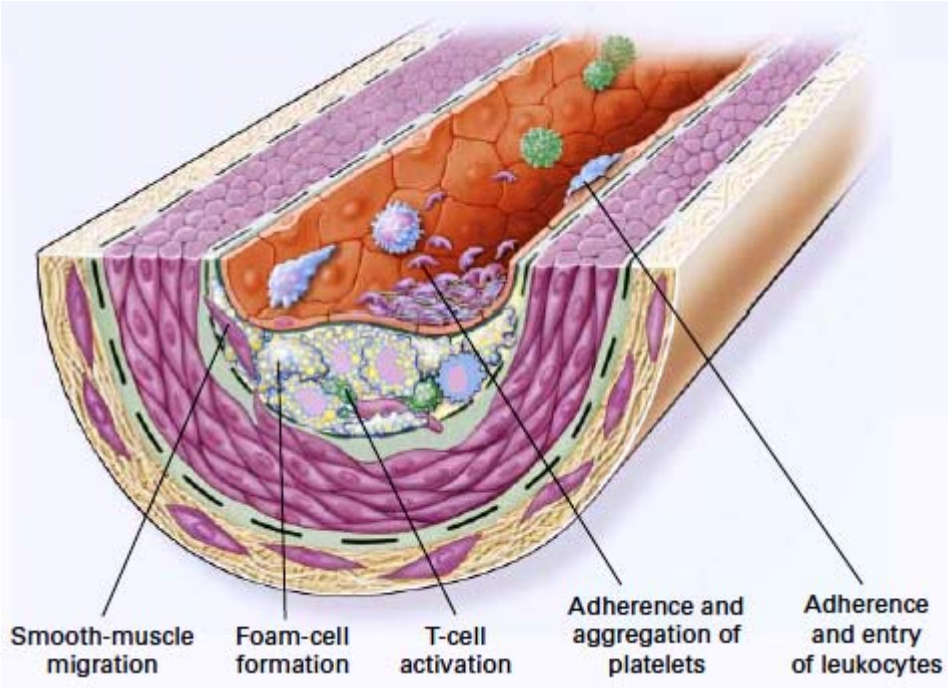


FIG2: FATTY-STREAK FORMATION IN ATHEROSCLEROSIS.

Formation of an Advanced, Complicated Lesion of Atherosclerosis:

As fatty streaks progress to intermediate and advanced lesions, they tend to form a fibrous cap that walls off the lesion from the lumen. This represents a type of healing or fibrous response to the injury. The fibrous cap covers a mixture of leukocytes, lipid and debris, which may form a necrotic core. These lesions expand at their shoulders by means of continued leukocyte adhesion and entry. The principal factors associated with macrophage accumulation include Macrophage colony-stimulating factor, Monocyte chemotactic protein-I (MCP-I) and oxidized Low-density lipoprotein. The necrotic core represents the results of apoptosis and necrosis, increased proteolytic activity and lipid accumulation. The fibrous cap forms as a result of increased activity of Platelet-derived growth factor, Transforming growth factor- β, Interleukin- 1, Tumor necrosis factor *α,* osteopontin and decreased connective-tissue degradation.

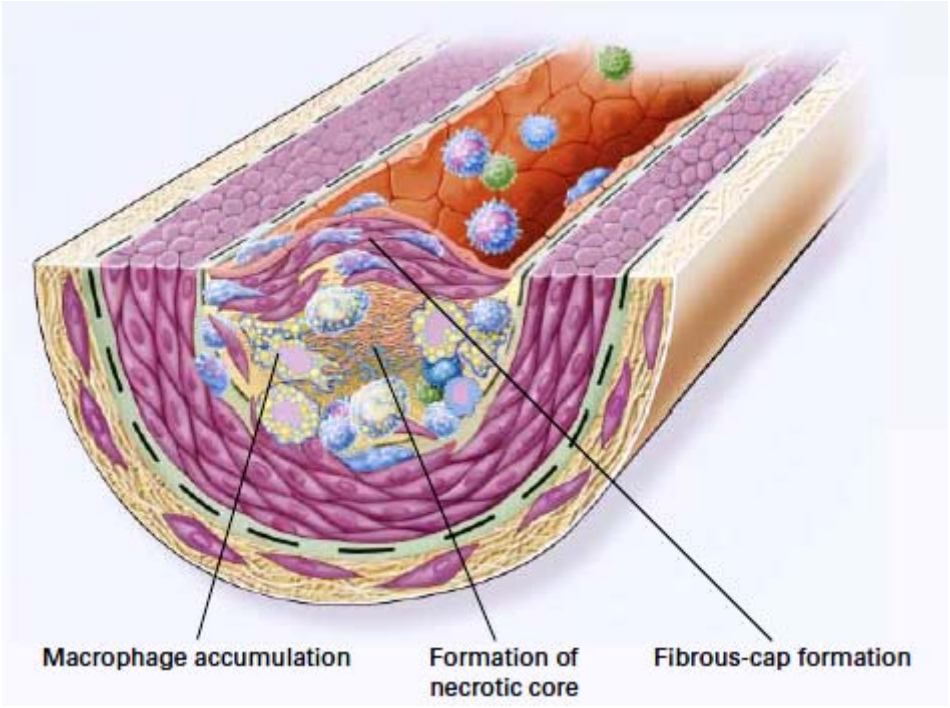


FIG 3: FORMATION OF AN ADVANCED, COMPLICATED LESION OF ATHEROSCLEROSIS

Unstable Fibrous Plaques in Atherosclerosis.

Rupture of the fibrous cap or ulceration of the fibrous plaque can rapidly lead to thrombosis and usually occurs at sites of thinning of the fibrous cap that covers the advanced lesion. Thinning of the fibrous cap is apparently due to the continuing influx and activation of macrophages which release metalloproteinases and other proteolytic enzymes at these sites. These enzymes cause degradation of the matrix which can lead to hemorrhage from the vasa vasorum or from the lumen of the artery and can result in thrombus formation and occlusion of the artery.[16]

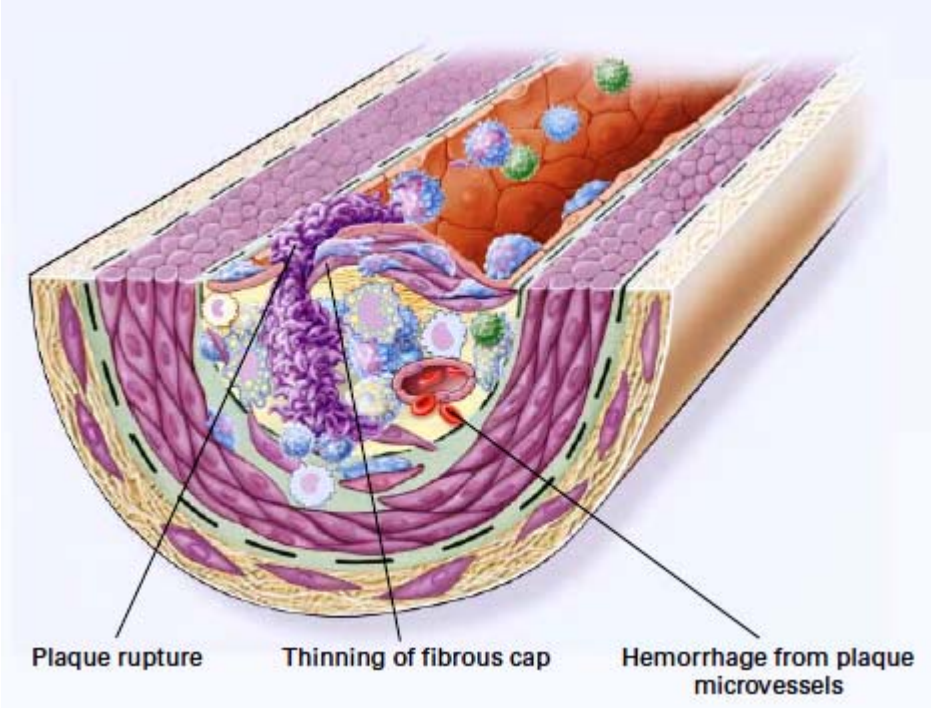


FIG 4: UNSTABLE FIBROUS PLAQUES IN ATHEROSCLEROSIS.

RISK FACTORS FOR CARDIOVASCULAR DISEASE:

WHO has drawn attention to the fact that CHD is our "modern epidemic". The aetiology of CHD is multifactorial. Some risk factors are modifiable, others immutable. Presence of any one of the risk factors places an individual in a high risk category for developing CHD.[9]

TABLE 1:MAJOR RISK FACTORS FOR CHD[6,9]

NON MODIFIABLE	MODIFIABLE
Age	Hyperlipidemia
Sex	Hypertension
Family History	Cigarette smoking
Genetic Factors	Diabetes
	Obesity
	Sedentary Habits
	Stress
	C-reactive Protein

AGE:

Age is a dominant influence. Although atherosclerosis is typically progressive, it usually does not become clinically manifested until middle age or later. Between ages 40 and 60 the incidence of myocardial infarction increases fivefold. Death rates from IHD rise with each decade even into advanced age[6]

GENDER :

Other factors being equal, premenopausal women are relatively protected against atherosclerosis and its consequences compared to age-matched men **.** After menopause, however, the incidence of atherosclerosis-related diseases increases and at older ages actually exceeds that of men. Although a favorable influence of estrogen has long been proposed to explain the protective effect, some clinical trials have failed to demonstrate any utility of hormonal therapy for vascular disease prevention.[17]

GENETIC FACTORS:

Family history is the most significant independent risk factor for atherosclerosis. Many Mendelian disorders associated with atherosclerosis, such as Familial Hypercholesterolemia, have been characterized. Nevertheless, these genetic diseases account for only a small percentage of cases. The well-established familial predisposition to atherosclerosis and IHD is usually multifactorial, relating to inheritance of various genetic polymorphisms, and familial clustering of other established risk factors, such as hypertension or diabetes[18]

HYPERLIPIDEMIA:

More specifically *Hypercholesterolemia*—is a major risk factor for atherosclerosis. Even in the absence of other factors, hypercholesterolemia is sufficient to stimulate lesion development.[19]

The major component of serum cholesterol associated with increased risk is low-density lipoprotein (LDL) cholesterol ("bad cholesterol").LDL cholesterol is the form of cholesterol that is delivered to peripheral tissues. In contrast, high-density lipoprotein (HDL, "good cholesterol") mobilizes cholesterol from tissue and

transports it to the liver for excretion in the bile. Consequently, higher levels of HDL correlate with reduced risk.[6]

With newer techniques, high-density and low-density lipoproteins have been further subdivided into sub-fractions. Recent evidence indicates that levels of plasma apolipoprotein-A-1(the major HDL protein) are better predictors of CHD than HDL cholesterol or LDL cholesterol respectively.[9]

HYPERTENSION :

Hypertension, defined as systolic/diastolic blood pressure of 140/90 mm Hg or higher, is the most prevalent modifiable risk factor for coronary heart disease, stroke, and heart failure[20]. Hypertension increases the risk of IHD by approximately 60%[6]. Systolic pressure is a stronger risk predictor than diastolic blood pressure, and each 20-mm Hg increase in systolic blood pressure is associated with two-fold increased risk of coronary heart disease in middle-aged populations.[20] Hypertension is the most important cause of left ventricular hypertrophy and hence the latter is also related to IHD[6]

CIGARETTE SMOKING:

Smoking is a well-established risk factor in men and probably accounts for the increasing incidence and severity of atherosclerosis in women. Prolonged (years) smoking of one pack of cigarettes or more daily, doubles the death rate from IHD. Smoking cessation reduces the risk substantially.[6]

DIABETES MELLITUS:

Diabetes induces hypercholesterolemia and markedly increases the risk of atherosclerosis. Other factors being equal, the incidence of myocardial infarction is twice as high in diabetics as in non diabetics. There is also an increased risk of strokes and a 100-fold increased risk of atherosclerosis-induced gangrene of the lower extremities.[6]

CHD is responsible for 30-50% of deaths in diabetics over the age of 40 years in industrialized countries.[9]

OBESITY :

Waist-to-hip ratio shows a graded and highly significant association with myocardial infarction risk worldwide. Redefinition of obesity based on waist-to-hip ratio(24.3%cases) instead of BMI (7.7% cases) increases the estimate of myocardial infarction attributable to obesity in most ethnic groups.[21]

PHYSICAL INACTIVITY :

Sedentary life-style is associated with a greater risk of the development of early CHD. There is evidence that regular physical exercise increases the concentration of HDL & decreases both body weight and blood pressure which are beneficial to cardiovascular health.[9]

ALCOHOL :

High alcohol intake defined as 75gm or more per day is an independent risk factor for CHD, hypertension & all cardiovascular diseases.[9]

ADDITIONAL RISK FACTORS:

INFLAMMATION :

Inflammation is present during all stages of atherogenesis and is intimately linked with atherosclerotic plaque formation and rupture. With increasing recognition that inflammation plays a significant causal role in IHD, assessment of systemic inflammation has become important in overall risk stratification. While a number of circulating markers of inflammation correlate with IHD risk, C-reactive protein (CRP) has emerged as one of the simplest and most sensitive.[22]

CRP is an acute-phase reactant synthesized primarily by the liver. It is downstream of a number of inflammatory triggers and plays a role in the innate immune response by opsonizing bacteria and activating complement. When CRP is secreted from cells within the atherosclerotic intima, it can activate local endothelial cells and induce a prothrombotic state and also increase the adhesiveness of endothelium for leukocytes. Most importantly, it strongly and independently predicts the risk of myocardial infarction, stroke, peripheral arterial disease, and sudden cardiac death, even among apparently healthy individuals(Fig 5).Indeed, CRP levels have recently been incorporated into risk stratification algorithms.[23]

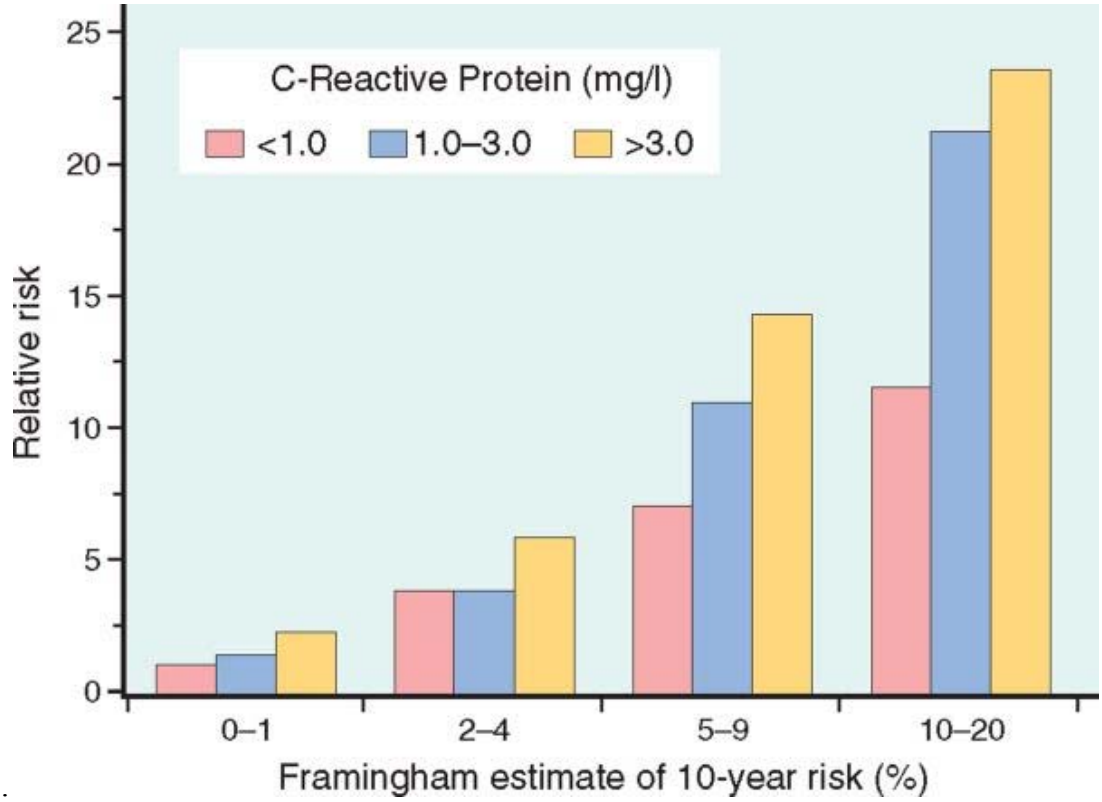


FIG 5:C-REACTIVE PROTEIN ADDS PROGNOSTIC INFORMATION TO ALL LEVELS OF TRADITIONAL RISK IDENTIFIED FROM FRAMINGHAM HEART STUDY.

HYPERHOMOCYSTINEMIA. :

Clinical and epidemiologic studies show a strong relationship between total serum homocysteine levels and coronary artery disease, peripheral vascular disease, stroke, and venous thrombosis.[24]Elevated homocysteine levels can be caused by low folate and vitamin B_{12} intake, although the jury is still out on whether supplemental folate and vitamin B_{12} ingestion can reduce the incidence of cardiovascular disease. *Homocystinuria*, due to rare inborn errors of metabolism, results in elevated circulating homocysteine (>100 μmol/L) and premature vascular disease.[6]

THROMBOTIC AND FIBRINOLYTIC FACTORS :

Several markers of hemostatic and/or fibrinolytic function (e.g., elevated plasminogen activator inhibitor 1) are predictors of risk for major atherosclerotic events, including myocardial infarction and stroke. Thrombin, through both its procoagulant and proinflammatory effects as well as platelet-derived factors are increasingly recognized as major contributors to local vascular pathology.[25,26]

BIOMARKERS OF SUBCLINICAL DAMAGE OR DISEASE :

The diagnostic approach to ACS remains one of the most difficult and controversial challenges facing emergency physicians. In recent years, cardiac troponins have emerged as the biochemical "gold standard" for diagnosis of patients with acute chest pain, enhancing our ability to recognize ACS. Early diagnosis and treatment of myocardial ischemia improve patient outcomes but conventional markers are often non-diagnostic at the time of arrival at the emergency department. Promising new biomarkers, which appear earlier after the onset of ischemia, are being studied and integrated into clinical practice. Some are markers of myocyte necrosis, but others, including ischemia-modified albumin and natriuretic peptides, detect myocardial ischemia and myocardial dysfunction.[27]

Ideal markers are not normally present in serum, become rapidly and markedly elevated during acute MI and are not released from other injured tissues. The increasing sensitivity and specificity of serum cardiac markers which are macromolecules (proteins) released from myocytes undergoing necrosis, have made them the "gold standard" for detection of myocardial necrosis.

What we have now are :

1. Troponins
2. Creatine kinase – MB isoenzyme
3. Myoglobin
4. Brain Natriuretic Peptide (BNP)
5. Pro BNP
6. Lactate Dehydrogenase

All are markers of myocardial injury or necrosis but what has always been used are the cardiac Troponins and Creatine kinase.

TROPONINS:

The troponins together form a complex of three proteins. This complex consists of troponin T (TnT; Tropomyosin binding), troponin I (TnI, Inhibitory component) and troponin C (TnC, Calcium binding component). Upon cell death, these and all other proteins constituting the cell are released into the circulation. After acute myocardial infarction it has been shown in serum of patients with AMI that the predominant forms in blood are free cTnT and the cTnI-TnC binary complex. Troponins have replaced other markers because they are more specific in the setting of injuries to skeletal muscle or other organs and also are more sensitive in the setting of minimal myocardial injury.

Cardiac-derived TnI (cTnI) and TnT (cTnT), proteins of the sarcomere, are not normally present in the blood with standard sensitivity assays and have amino acid sequences distinct from their skeletal muscle isoforms.

Troponin T is a cardio-specific polypeptide mostly bound to contractile elements of myocardial cells, but with small amounts also present free in the cytoplasm. Cytosolic cardiac troponin T is released within the first few hours after infarction. Release of myofibrillar cTnT occurs more slowly, over a period of days. This biphasic release results in an early rise in serum levels (3-4 hours after the infarct) which is sustained for 10 days or more. This makes it a very useful marker. Minor elevations occur in unstable angina.

With even small acute MIs, troponins increase to 20-fold or more above the lower limits of the assay, and elevations persist for several days.

The troponins generally are first detectable 2 to 4 hours after the onset of acute MI, are maximally sensitive at 8 to 12 hours, peak at 10 to 24 hours, and persist for 5 to 14 days. Their long persistence has allowed them to replace other markers for the diagnosis of acute MI in patients presenting late (>1 to 2 days) after symptoms. However, this persistence can obscure the diagnosis of an early recurrent MI, for which more rapidly cleared markers (i.e. CK-MB) are more useful.[28]

CREATINE KINASE AND ISO-ENZYMES :

Creatine kinase is a cytosolic enzyme (81 kDa) expressed in various tissue types. The two subunits of this dimeric enzyme can either be B-type (brain) or M-type (muscle), yielding three possible isoenzymes (MM, BB and MB), which are all present in tissue, but the composition varies. Skeletal muscle expresses CK-MM at high levels (99%) and CK-MB at low levels (1.1%), whereas cardiac muscle, in contrast, expresses CK-MM at 79% and CK-MB at 20%.46 It is important to realize that the total CK activity in skeletal muscle is about 5-10 fold higher than in cardiac

muscle, so that in absolute values (activity per gram wet weight tissue), skeletal muscle contains approximately equal amounts of CK.MB.

However, because cardiac muscle contains the largest proportion of CK-MB, this was the first biochemical marker for AMI that was relatively specific for necrotic myocardium. [28,29]

Total CK starts to rise within 3 to 8 hours after MI, peaks at 10 – 24 hours and returns to normal by 3 – 4 days. It can be markedly elevated with skeletal muscle trauma or brain injury.

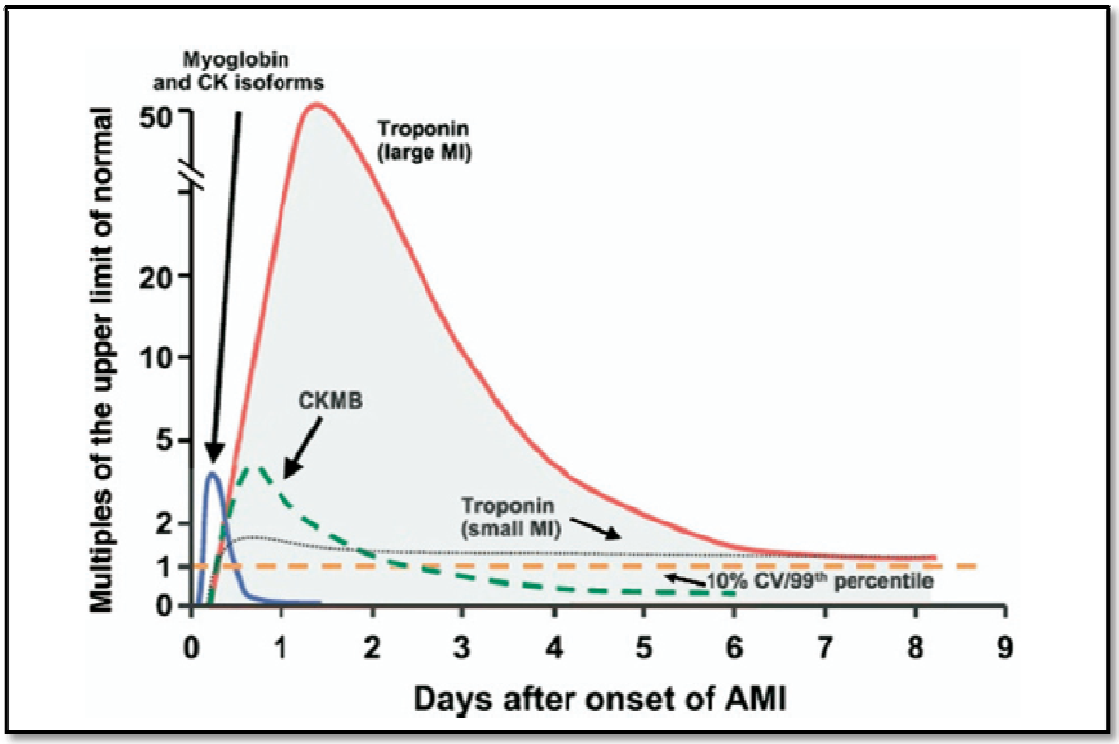


FIG 6 : ELEVATION OF CARDIAC MARKERS AFTER MI.[30]

PLATELETS : GENERAL OVERVIEW

Platelets are anucleate fragments with a diameter of 1 to 4 mm that are released by bone marrow megakaryocytes into the circulation and are thought to be primarily responsible for the maintenance of vascular integrity and hemostasis.[31]The megakaryocyte surface membrane forms protoplatelet extensions from which platelets "bud off" and are emitted into the circulation, where they number approximately 200,000 to 400,000 per microliter of blood.[32]

Blood platelets play critical roles in haemostasis, providing rapid protection against bleeding and catalyzing slower formation of stable blood clots via the coagulation cascade. They are also involved in protection from infection by phagocytosis of pathogens and by secreting chemokines that attract leukocytes. Platelet function is commonly assessed by platelet count, bleeding time, and platelet aggregation or activation. However, defining and measuring *in vivo* platelet function remains a challenge.[8]

PLATELET ULTRASTRUCTURE & FUNCTIONS:

Human platelets circulate in the blood as discs that lack the nucleus found in most cells. Platelets are heterogeneous in size, exhibiting dimensions of 0.5× 3.0μm. The surface of the platelet plasma membrane is smooth except for periodic invaginations that delineate the entrances to the open canalicular system (OCS), a complex network of interwinding membrane tubes that permeate the platelet's cytoplasm. The lipid bilayer of the resting platelet contains a large concentration of transmembrane receptors that include the glycoprotein receptor for von Willebrand factor (vWF), the major serpentine receptors for ADP, thrombin, epinephrine and

thromboxane A2, the Fc receptor Fcγ RIIA and the β3 and β1 integrin receptors for fibrinogen and collagen.[34]

The anatomy of platelet is divided into into three major Regions:

The peripheral zone consists of the external and internal membrane systems that provide the exposed surface of the platelet and walls of the tortuous channels making up the surface-connected open canalicular system (OCS). An exterior coat or glycocalyx, rich in glycoproteins, constitutes the outermost covering of the peripheral zone. Its chemical constituents provide the receptors for stimuli triggering platelet activation and the substrates for adhesion–aggregation reactions. The middle layer of the peripheral zone is a typical unit membrane.It is rich in asymmetrically distributed phospholipids that provide an essential surface for interaction with coagulant proteins.The area lying just inside the unit membrane represents the third component of the peripheral zone. It is closely linked to the unit membrane and translates signals received on the outside surface into chemical messages and physical alterations required for platelet activation.

The internal membrane systems include the OCS, even though it is continuous with, and part of, the external membrane system. Channels of the dense tubular system (DTS) and the membrane complexes (MC) formed by elements of the OCS and DTS are internal membrane systems, but function with and are considered part of the peripheral zone.

The sol–gel zone is the matrix of the platelet cytoplasm. It contains several fibre systems in various states of polymerization that support the discoid shape of unaltered platelets and provide a contractile system involved in shape change, pseudopod extension, internal contraction, and secretion.Elements of the contractile

system appear to be major components, since they constitute approximately 30–50% of the total platelet protein. Masses as well as discrete particles of glycogen are distributed in the sol–gel matrix.

The organelle zone consists of granules, electron-dense bodies, peroxisomes, lysosomes, glycosomes and mitochondria randomly dispersed in the cytoplasm. It serves in metabolic processes and for the storage of enzymes, nonmetabolic adenine nucleotides, serotonin, a variety of protein constituents and calcium destined for secretion.[35]

TABLE 2:PLATELETS ULTRASTRUCTURE AND FUNCTIONS

Zone & Component	Function
Peripheral zone	
Glycocalyx – proteins, phospholipids, Mucopolysaccharides	Adhesion & Aggregation
Phospholipid bilayer Phospholipids	Source of arachidonic acid
Integral proteins Glycoproteins Ib/IX, IIb/IIIa Enzymes	Adhesion & aggregation, activation
Structural zone	
Microtubules Cytoskeletal network Cytoplasmic network – actin, myosin Actin binding protein	

Organelle zone	
Granules	Non protein mediators
Dense bodies	Protein mediators
Alpha granules	Enzymes
Lysosomes	Break down H2O2
Microperoxisomes	
Membrane systems	
Open canalicular system	Secretion of granule contents
Dense tubular system	Calcium storage site

Principle of Autoanalyzer: Impedance measurement principle

In impedance measurement (resistance measuring principle), cells are passed one after the other through a capillary opening. The passing cell produces an electrical resistance and thus an electronic signal which is proportionate to its volume. Hence, the cells are identified based on their size and get represented in a volume distribution curve [37].

Platelet indices: Recent advances in automated blood cell analysers have made it possible to measure various blood cell parameters automatically. Among these parameters, platelet indices, such as mean platelet volume (MPV), platelet distribution width (PDW) and platelet large cell ratio (PLCR) provide some important information but are not accepted for routine clinical use [38]. If these indices really are informative regarding platelet kinetics, they might become very useful laboratory measures for thrombocytopenia.[39]

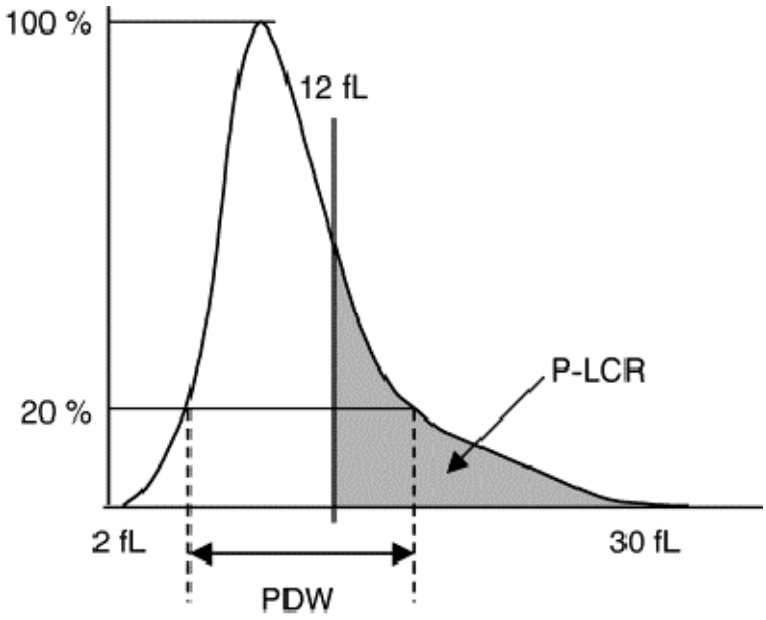


FIG 7: PLATELET HISTOGRAM

MPV: a measurement of the average size of platelets.

MPV was calculated by the following formula, MPV (fL) = [(plateletcrit (%)/ platelet count ($x10^9$/l)] x 10^5. Plateletcrit is the ratio of the platelet volume to the whole blood volume.[40]

Circulating platelets are very different in size, metabolism, and functional activity. The largest are more reactive and produce a greater quantity of thrombogenic factors[41]

The increase of MPV in conditions with increased platelet turn over is probably mediated by several cytokines (interleukins 6 and 11 and thrombopoietin) that affect megakaryocyte ploidy and result in the production of larger and more reactive platelets.[42]

PDW: PDW is the distribution width on 20% frequency level with the peak taken as 100% .[40]

The PDW is useful in differentiating reactive thrombocytosis from the essential type, especially when it is combined mathematically with the MPV and platelet count to obtain a discriminant function.[43]

P-LCR: This is the ratio of large platelets exceeding 12 fL discriminator and is calculated as the ratio of the particle count between the 12-fL fixed discriminator and Upper discriminator (UD) to the particle count between Lower discriminator (LD) and Upper discriminator (UD) .[40]

UTILITY OF PLATELET PARAMETERS IN VARIOUS DISEASES:

MPV is significantly increased in patients with Idiopathic Thrombocytopenic Purpura and Iron Deficiency Anemia whereas in those with Aplastic Anemia and Leukemia it is normal. The mean platelet volume of the patient with Idiopathic Thrombocytopenic Purpura decreases as the platelet count increases and it becomes normal when the patient's platelet count reaches the normal range.

In Acute Post Streptococcal Glomerulonephritis, Renal Failure & Congenital Cyanotic Heart Disease the mean platelet volume is significantly increased.

In pregnant women with pre-eclampsia, the mean platelet volume showed a significantly higher value than in normal spontaneous vaginal delivery (NSVD), Spontaneous pre-mature rupture of membrane (SPRM) & abortion.

In adults with Rheumatic Heart Disease and Diabetes Mellitus the Mean Platelet Volume is significantly increased over that of the control group[44].

Increased MPV value is associated with a worse outcome in patients suffering an acute ischemic cerebrovascular event. Patients within the highest quintile of MPV

have a two fold risk of suffering a severe stroke compared with patients within the lowest quintile[45].

MPV changes have been observed in some but not all studies in Rheumatoid Arthritis[46] and systemic lupus erythematosus (SLE)[47]. It has been observed that MPV of active Rheumatoid Arthritis patients is significantly lower than that of patients with osteoarthritis and healthy subjects. This finding was accompanied by increased disease activity, measured by Disease Activity Score 28 (DAS28), platelet count and biomarkers of inflammation which suggested that platelet activation in RA is associated with reactive megakaryocytopoiesis as part of active inflammation. Small MPV may also reflect accelerated maturation and short lifespan of platelets in active RA[46].

In patients with active Ulcerative Colitis and Crohn's Disease, there is a statistically significant decrease in MPV, PDW levels and increase in PCT levels when compared to healthy controls. In remission phase of Inflammatory Bowel Disease while MPV levels were lower, PDW and PCT levels were higher than control group[48].

Platelet count, Mean platelet volume and Platelet distribution width are higher in lung cancer patients compared with healthy subjects. Among patients with lung cancer, PDW in small cell lung cancer (SCLC) patients is higher than in non-small cell lung cancer (NSCLC) patients. However, there is no difference in Platelet indices in between stage III and IV in NSCLC patients and in between limited and extensive disease in SCLC[49].

In the diagnosis of bone marrow metastasis in patients with solid tumor, MPV in patients with marrow metastasis is lower than in patients without metastasis[50]

ROLE OF PLATELETS IN HAEMOSTASIS:

Blood platelets act as the first defense of the body against haemorrhage. When stimulated usually by a break in the endothelial lining of a blood vessel, they are attracted to the defect, they round up, develop pseudopods, become sticky and adhere to the abnormal area.

Platelets adhere to the injured blood vessel to prevent blood loss through a discrete series of steps involving platelet adhesion to the wounded area and platelet activation i.e. generation of intracellular chemical signals that are initiated by platelet adhesion. These signals cause rapid morphological changes such as extension of pseudopodia, platelet-platelet aggregation and granule secretion.

PLATELET ADHESION: Vascular injury disrupts the single layer of endothelial cells that line blood vessel walls exposing a rich matrix of subendothelial proteins by means of adhesion receptors on platelets like GP Ib-V-IX complex, Integrins especially $\alpha IIb\beta_3$ (GP IIb-IIIa) and $\alpha_2\beta_1$, GPVI, GPIV etc.

PLATELET AGGREGATION-Platelets circulate as disc shaped cells but when they come into contact with exposed subendothelium, agonists that activate platelets are exposed, generated or released .These agonists cause platelets to change shape such that they form pseudopodia because of changes in the polymerization of the actin cytoskeleton followed by aggregation of platelets. The platelet agonists induce signal transduction in platelets resulting in activation of Platelet Integrin Adhesion Receptor $\alpha_{2b}\beta_3$ which binds to fibrinogen or vWF and links adjacent platelets in an aggregate. The agonists include subendothelial collagen, thrombin, ADP, circulating epinephrine and the arachidonic acid metabolite-Thromboxane2(TXA2)

PLATELET RELEASE REACTION:

Upon activation of platelets by agonists, platelets undergo a release reaction thereby secreting its granular contents-ATP, ADP, Calcium, Serotonin, PF4, β-Thromboglobulin, PDGF, Fibrinogen, Fibronectin, Thrombospondin, vWF and the production of TXA2. Weak agonists (ADP and epinephrine) require both cyclooxygenase activity and primary aggregation to induce secretion whereas strong agonists(collagen and thrombin)at high concentrations induce platelet aggregation and secretion that is independent of cyclooxygenase activity.

CLOT FORMATION-

This is the normal, physiologically important outcome of primary haemostasis. Platelets adhere to the wound site, secrete factors that further activate local platelets and aggregates to form a platelet plug. Platelets also contribute to clot formation by enhancing the formation of fibrin and immobilizing it on their surface.[51]

EVALUATION OF PLATELET INDICES IN ACUTE CORONARY SYNDROME:

Traditionally, platelet function and size correlate because larger platelets, produced from activated megakaryocytes in the bone marrow, as explained are likely to be more reactive than normal platelets. Consequently, larger and hyperactive platelets play a pivotal role in accelerating the formation and propagation of intracoronary thrombus, leading to the occurrence of acute thrombotic events. These observations led to the hypothesis that increased consumptions of platelets thereby reducing the platelet count and increased MPV, which is an index of platelet size that

acts as a reliable index of platelet activation, may be a potentially useful marker in cardiovascular risk stratification. [52]

Khandekar M M et al observed 210 patients over a period of one year and found that platelet indices are raised in patients who have suffered an acute coronary event in comparison with controls and those with stable CAD. The data also suggested that MPV value of >9.6 fl was associated with significant risk for developing MI in patients with Coronary Artery Disease. This reflects that increased MPV contributes to pre-thrombotic stage in acute ischaemic syndromes and that larger platelets may play a specific role in infarction.[4]

Lippi G et al included a total of 456 patients with Acute Coronary Syndromes. These patients ,all having cardiac Troponin T levels of 0.03ng/ml or greater in addition to ischaemic electrocardiogram changes, had higher MPV values than non-Acute Coronary Syndromes patients with normal cardiac Troponin T levels .At 9.0 fl cut off, the negative and positive predictive values of MPV were 83% and 43% respectively.[5]

Chu S G et al analysed 24 different studies of over 6000 subjects and observed significant estimated mean difference in MPV between AMI and non-AMI populations. The data also suggested that elevated MPV was associated with increased mortality following a Myocardial Infarction.[7]

Endler G et al compared 185 patients with stable CAD with 188 individuals who had suffered myocardial infarction and the findings indicated that increased mean platelet volume (MPV≥11.6fl) may represent an independent risk factor for MI in patients with CAD.[53]

Pizzulli L et al studied 981 patients and found that patients with unstable angina requiring immediate PTCA had a lower platelet count and higher mean platelet volume (10.4±1.03fl) than the rest of the population with unstable angina.[54]

Khode V et al compared 39 patients with AMI & 24 patients with Stable CAD with 65 controls and observed that the best cut-off values for MPV when predicting AMI and Stable CAD in patients were 9.25fl(sensitivity 56.4% and specificity 45.9%) and 9.15fl(sensitivity 54.2% and specificity 42.23%) respectively[55].

The frequently described inverse relationship between platelet count and MPV in physiological and some pathological conditions reflects the tendency to maintain hemostasis by preserving a constant platelet mass. This inverse relationship is often seen in inflammatory disorders, where enhanced thrombopoiesis increases the quantity of circulating platelets and large amount of highly reactive large-sized platelets migrate to inflammatory sites, where they are intensely consumed. Importantly, defective thrombopoiesis and enhanced destruction and swelling of circulating platelets in an environment rich in activating agents can affect the relationship between platelet count and MPV. Circulating platelets contain matrix ribonucleic acid, mitochondria, alpha and dense granules which provide mechanisms of self regulation by shape-change and release of biologically active substances . Rapid (minutes-hours) shifts in platelet indices, including an increase of MPV, may take place as a result of the synthesis of prothrombotic and pro-inflammatory agents in platelets, degranulation of alpha-granules, and release of highly reactive platelets from stores (the spleen).[56]

MPV is measured by cell counters employing impedance and optical effects. The discordance between the results of different and even the same cell counters

limits the interchangeable use of MPV. This can explain, at least partly, why haematological laboratories sometimes do not display the MPV and some other indices of platelet function.

Understanding of the role of platelets in a variety of thrombotic and inflammatory disorders has substantially improved, owing to the recent advances in the quantification of laboratory markers of platelet function. MPV has emerged as a relatively reliable marker of thrombopoiesis and platelet function. PDW, P-LCR and Plateletcrit are yet to be explored fully with respect to its significance.[57]

MATERIALS AND METHODS

Source of data

A prospective hospital based study was carried out on 175 patients admitted in BLDE University Shri B.M.Patil Medical College, Hospital and Research centre, Bijapur from October 2011 to March 2013 considering the inclusion and exclusion criteria.

All the patients diagnosed with Acute Coronary Syndromes were included in the study and compared with age and sex matched normal healthy controls having a normal electrocardiogram and no past history of Ischaemic heart disease.

Methods of collection of data.

- The study was carried out on patient presenting with Acute Coronary Syndromes within 24 hours.
- All subjects were interviewed as per the prepared proforma and then complete clinical examination was done
- The blood samples of the patients was drawn from the antecubital vein using a 5ml syringe and immediately mixed in EDTA vacuutainers.
- The sample was run within two hours of venepuncture using the 3 part differentiated automated Hematoanalyzer (Sysmex KX-21) and complete blood count analysis of the sample was made including the platelet indices (MPV, PDW, P-LCR).
- The peripheral smear slides of the samples were also made using Leishmann's stain to study platelet morphology obtained from the autoanalyser.

- Relevant investigations like electrocardiogram and cardiac enzymes were also analysed for confirmation of the diagnosis.

Inclusion criteria:

1. Patients diagnosed with unstable angina (UA),ST segment elevation myocardial infarction(STEMI), non-ST segment elevation myocardial infarction(NSTEMI).
2. Patients more than 18 years of age

Exclusion criteria:

1. Patients with bleeding diathesis, previous stroke, major operations or significant trauma in the past two weeks or hypertension(>180/110 mm of Hg)
2. Patients less than 18 years of age
3. Patients with non-cardiac chest pain

STATISTICAL METHODS EMPLOYED:

Data will be analysed by using :

1) Diagrammatic presentation
2) Mean $\pm$ SD
3) 't' test
4) Chi square test

RESULTS

175 cases and 175 control were included in the study. Of the 175cases, 79 cases (45.14%) were diagnosed as ST-Elevation Myocardial Infarction,58(33.14%) were of unstable angina followed by 38(21.71%) cases of Non-ST-Elevation Myocardial Infarction.(Fig 8)

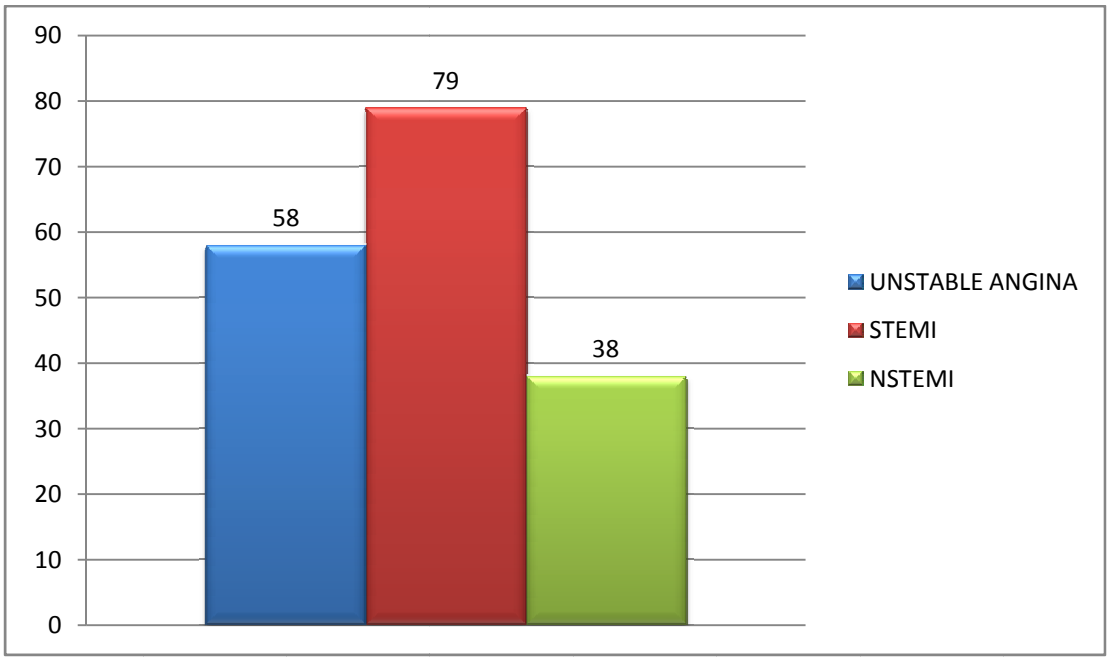


FIG 8:MANIFESTATION OF ACS AMONG CASES

In the present study, the ages ranged from 28 to 89 years. The mean age of patients in our study is 57.76 ±13.19 years. Majority of the patients diagnosed with Acute Coronary Syndrome belonged to the 5th decade of life (29.71%), followed by 6th decade (22.86%) and 4th decade (18.86%) of life.(Table 3)

TABLE 3: DISEASE DISTRIBUTION IN DIFFERENT AGE GROUPS

AGE(YEARS)	CATEGORY	FREQUENCY	PERCENT
11-20	1	0	
21-30	2	5	2.86%
31-40	3	16	9.14%
41-50	4	33	18.86%
51-60	5	52	29.71%
61-70	6	40	22.86%
71-80	7	26	14.86%
81-90	8	3	1.71%
TOTAL		175	100.00%

In the present study, total number of males including both cases and controls were 223 (63.71%) and number of females were 127 (36.28%).

The total number of males presenting with acute coronary syndrome among the cases were 110(62.86%) and females affected were 65(37.14%) (Table 4). The male to female ratio was 1.7:1.

TABLE 4: SEX DISTRIBUTION AMONG CASES

SEX RATIO (CASES)	FREQUENCY	PERCENT
Male	110	62.86%
Female	65	37.14%
Total	175	100.00%

TABLE 5: SEX DISTRIBUTION AMONG CONTROLS

SEX RATIO (CONTROL)	FREQUENCY	PERCENT
Male	113	64.57%
Female	62	35.42%
Total	175	100.00%

The risk factors for developing ischaemic heart diseases viz Smoking, Hypertension, Diabetes Mellitus, Alcohol & Family History were analysed. (Table 6)

In our study, family history of IHD was noted in only 2.3% patients among the cases and in 0.5% controls. Those with positive family history had 4 times higher odds of getting the disease as compared to controls with positive history (Odd's Ratio=4:1). But it was not significant statistically. (p=0.211).

All the smokers were male in our study group. The highest prevalence of smoking was among NSTEMI (21%) followed by STEMI (19%) with more smokers being among the cases (29.7%) compared to those with control (17%).This difference is statistically significant(p=0.005). Thus, cigarette smoking was found to be a risk factor for IHD in our study (Odd's Ratio=2:1).

Similarly alcohol consumption was observed in among 46.9% cases in our study and in only 7.2% controls with an odds ratio of 10.99. As the exact amount of alcohol intake was not available we could not stratify the patients into moderate or heavy alcohol intake groups. The difference came to be highly significant. (p=<0.0001).Thus, alcohol consumption was found to be a risk factor for IHD in our study.

History of diabetes mellitus was positive in 19.4% patients in our study & 6.7% in the control group with an odds ratio of 3.28.Male predilection was noted in our study with 71.73% males and 28.26% females being diabetic. The difference was found to be highly significant statistically. (p<0.005).Thus, diabetes was found to be a risk factor for IHD in our study.

Hypertension was seen in 17.7% patients, of which 62.61% were males and 37.38% were females. In our study, hypertension was seen more among the control group (24.3%) in comparison to the cases. The difference however is not significant.(p=0.118).Thus, hypertension was not found to be a direct risk factor for IHD in our study. This may be attributed to the multi factorial causation of CVD & higher number of undiagnosed cases of hypertension, in accordance to the rule of halves and the iceberg phenomenon.

TABLE 6 : DISTRIBUTION OF RISK FACTORS

RISK FACTOR	CASES	CONTROLS	Odds Ratio	P value
	n_1=175	n_2=175		
POSITIVE FAMILY HISTORY	4(2.3%)	1(0.5%)	4.07	0.211
CIGARETTE	52(29.7%)	30(17.0%)	2.04	**0.005**
ALCOHOL	82(46.9%)	13(7.2%)	10.99	**<0.0001**
DIABETES	34(19.4%)	12(6.7%)	3.28	**<0.005**
HYPERTENSION	31(17.7%)	43(24.3%)	0.66	0.118

HEMATOLOGICAL PARAMETERS :

PLATELET INDICES :

The platelet indices Mean platelet volume (MPV), Platelet distribution width (PDW) and Platelet large cell ratio (P-LCR) were studied among patients with ACS and compared with age and sex matched healthy control groups. (Table 7)

MEAN PLATELET VOLUME :

MPV of all patients was noted .The mean of the MPV for the control group in our study was 8.14 ± 0.67 fL, for unstable angina it was 8.53 ± 0.54fL, 9.67 ± 0.82 fL for STEMI & 9.54 ± 0.76 fL for NSTEMI (table 7). The MPV was highest in ST-Elevation Myocardial Infarction group 9.67 ± 0.82 fL followed by Non-ST-Elevation Myocardial Infarction 9.54 ± 0.76 fL when compared with patients diagnosed as unstable angina 8.53 ± 0.54 fL which was close to the MPV values recorded in the control group

TABLE 7: DISTRIBUTION OF PLATELET INDICES

CATEGORY	UNSTABLE ANGINA	STEMI	NSTEMI	CONTROLS	OVERALL (CASES)
Number (350)	58	79	38	175	175
Platelet Indices					
PDW (fL)	13.41 ± 4.02	13.66 ± 3.55	13.24 ± 3.46	12.23 ± 3.13	13.49±3.67
MPV (fL)	8.53 ± 0.54	9.67 ± 0.82	9.54 ± 0.76	8.14 ± 0.67	09.27±0.89
P-LCR (%)	18.57 ± 3.70	22.09 ± 4.89	22.36 ± 4.95	18.12 ± 3.54	20.99±4.83

Based on previous similar studies done on platelet indices & CVD, a cut-off value of 9.6 fl was taken to make it a dichotomous variable for calculating the statistical association between MPV recorded and the clinical diagnostic category.

We found it to be highly significant for both category (STEMI & NSTEMI) with p value ($p<0.0001$) at 2 degrees of freedom and 95% Confidence level control in comparison to patients diagnosed with unstable angina.(table 8)

TABLE 8: DISTRIBUTION OF CASES ACCORDING TO MPV VALUES

	CLINICAL DIAGNOSIS			
MPV RECORDED	**UNSTABLE ANGINA**	**STEMI**	**NSTEMI**	**Total**
<9.6	56(53.33%)	35(33.33%)	14(13.33%)	105
≥9.6	2(2.86%)	44(62.86%)	24(34.29%)	70
TOTAL	58	79	38	175

ASSOCIATION BETWEEN MEAN PLATELET VOLUME & CARDIAC TROPONIN T RESULT:

Our study showed that the association between Mean Platelet Volume and Cardiac Troponin T is statistically significant (p=0.031) at 1 degree of freedom and 95% confidence level for the STEMI group as 95% of cases here had larger value of MPV and had cardiac enzyme Troponin T positive, for the NSTEMI group about 87% cases had both the larger indices and positive Troponin Value but it was not statistically significant (p=0.603).(Table 9).

TABLE 9: ASSOCIATION BETWEEN MEAN PLATELET VOLUME & CARDIAC TROPONIN T

	STEMI			NSTEMI	
	TROPONIN			TROPONIN	
MPV RECORDED	POSITIVE	NEGATIVE	Total	POSITIVE	NEGATIVE
<9.6	28(80.0)	7(20.0)	35	12(92.9)	1(7.1)
≥9.6	42(95.4)	2(4.6)*	44	21(87.5)	2(12.5)
TOTAL	70	9	79	34	4
Chi-square	4.612	*p*=0.031		0.269	*p*=0.603

*Fischer Exact Test

ASSOCIATION BETWEEN MEAN PLATELET VOLUME AND CPK-MB LEVEL:

Our study also showed that 95 % cases of the STEMI group had larger value of MPV and were also positive for CPK-MB. However, the association was not significantly associated at 1 degree of freedom and 95% confidence level (P=0.065). In NSTEMI group about 87 % had both the variable on higher side, but here also p value was non-significant (p=0.875), emphasizing the non-specific nature of the marker CPK-MB. (Table 10)

TABLE 10: ASSOCIATION BETWEEN MEAN PLATELET VOLUME AND CPK-MB LEVEL:

	STEMI			NSTEMI		
	CPK-MB			CPK-MB		
MPV RECORDED	0-25 IU	>25 IU	Total	0-25 IU	>25IU	Total
<9.6	6(17.1)	29(82.9)	35	2(14.3)	12(85.7)	14
≥ 9.6	2(4.60	42(95.4)	44	3(12.5)	21(87.5)	24
TOTAL	8	71	79	5	33	38
Chi-square	3.399	p=0.065		0.0247	p=0.875	

PLATELET DISTRIBUTION WIDTH :

The PDW in our study for the control group was 12.23 ± 3.13 fL . The mean of the PDW values for unstable angina was 13.41 ± 4.02 fL, 13.66 ± 3.55 fL for STEMI & 13.24 ± 3.46 fL for NSTEMI . (Table 7)

Table 11 created using PDW and Clinical Diagnosis found these variable to be highly significant with Chi-squared=12.21, degree of freedom =2, p=0.002. Based on logical statistical reasoning, a cut-off value of 12.8 was taken, that is the average of the mean value of all case & mean value of control.(12.23+13.49)/2 = 12.8 .To convert it into a dichotomous variable for calculating the statistical association between PDW and clinical category & also with cardiac enzymes, (table 10) PDW with the value of <12.8 & PDW >=12.8 was taken (Table 11).

TABLE 11:DISTRIBUTION OF CASES ACCORDING TO PDW VALUES

	CLINICAL DIAGNOSIS			
PDW RECORDED	**UNSTABLE ANGINA**	**STEMI**	**NSTEMI**	**Total**
<12.8	21	51	24	97
≥12.8	37	28	14	78
TOTAL	58	79	38	175

ASSOCIATION BETWEEN PLATELET DISTRIBUTION WIDTH AND CARDIAC TROPONIN –T RESULTS

	STEMI			NSTEMI		
	TROPONIN-T			TROPONIN-T		
PDW RECORDED	POSITIVE	NEGATIVE	Total	POSITIVE	NEGATIVE	Total
<12.8	33(82.50)	7(17.5)	40	22(91.67)	2(8.33)	24
≥12.8	37(94.87)	2(5.13)*	39	12(85.71)	2(14.29)	14
TOTAL	70	9	79	34	4	38
Chi-square	4.87	*p*=0.027		5.6	*p*=0.018	

*Fischer Exact Test

TABLE 12:ASSOCIATION BETWEEN PLATELET DISTRIBUTION WIDTH AND CARDIAC TROPONIN-T

Here in the STEMI group 95 % cases were Troponin-T positive with PDW ≥ 12.8 and only 5% were Troponin-T negative which is statistically significant (p=0.027) . Among the NSTEMI group, there were 86% enzyme positive with PDW≥12.8 against 14% cases with Troponin-T negativity with PDW≥12.8.This association was again statistically significant (p=0.018), suggesting that PDW ≥12.8 with Troponin-T positivity is indicative of an impending acute coronary event.(Table 12).

PLATELET LARGE-CELL RATIO:

The mean P-LCR recorded in our study for STEMI was 22.09 ± 4.89 fL, for NSTEMI it was 22.36 ± 4.95fL,unstable angina 18.57 ± 3.70fL and for the control group it was recorded as 18.12 ± 3.54fL (Table 7). Again a cut-off value of 19.5 was taken to dichotomize the variable & show the association between P-LCR recorded and the clinical diagnostic category (STEMI & NSTEMI*)* it was highly significant *(*p<0.0001*)* at 2 degrees of freedom and 95% Confidence level in comparison to the patients diagnosed with unstable angina.(Table 13)

TABLE 13 : DISTRIBUTION OF CASES ACCORDING TO P-LCR VALUES

	CLINICAL DIAGNOSIS			
P-LCR RECORDED	**UNSTABLE ANGINA**	**STEMI**	**NSTEMI**	**Total**
<19.5	37(63.79%)	17(21.52%)	9(13.33%)	105
>=19.5	21(36.21%)	62(78.48%)	29(34.29%)	70
TOTAL	58	79	38	175

ASSOCIATION BETWEEN PLATELET LARGE CELL RATIO AND CARDIAC TROPONIN –T RESULTS

The results shows that 92 % were Troponin T positive when P-LCR ≥ 19.5 and only 8% cases were Troponin T negative among the STEMI group. However,

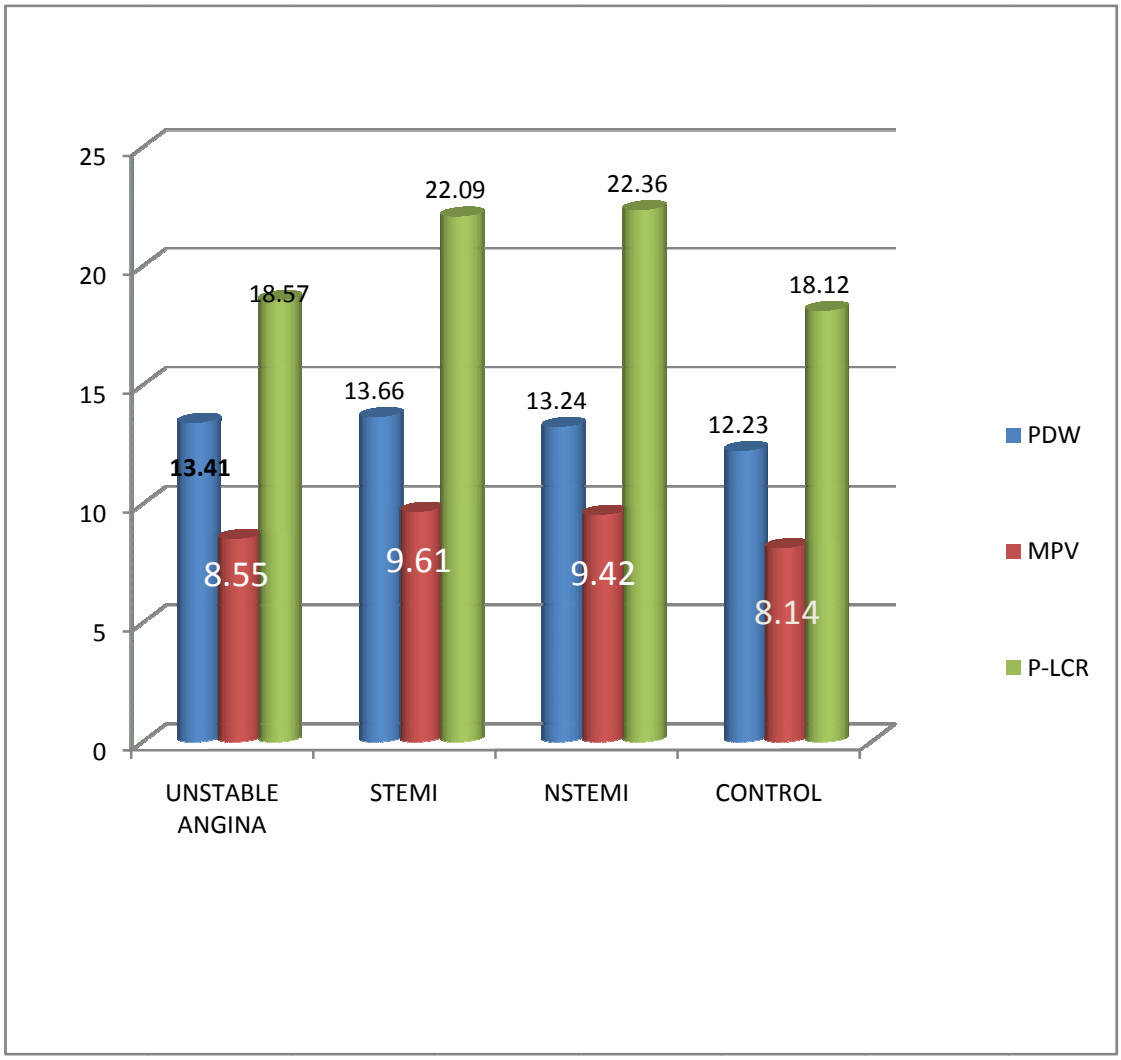
when P-LCR <19.5, 76% cases were Troponin-T positive & 24% were Troponin-T negative. This association was statistically insignificant(p=0.179).

Similarly for NSTEMI group about 55% cases were enzyme positive and had P-LCR value ≥19.5, as against 44 % cases who were enzyme negative. These findings were also statistically not significant (p=0.346) .Thus, according to our study, P-LCR cannot be considered as a supportive indices for evaluating cardiovascular patients profile. (Table 14)

	STEMI			NSTEMI		
	TROPONIN-T			TROPONIN-T		
P-LCR RECORDED	**POSITIVE**	**NEGATIVE**	**Total**	**POSITIVE**	**NEGATIVE**	**Total**
1(<19.5)	13(76.47)	4(23.53)	17	7(35.0%)	13(65.0%)	20
2(>=19.5)	57(91.94)	5(8.06)	62	10(55.6%)	8(44.4%)	18
TOTAL	70	9	79	17	21	38
Chi-square	1.81	*p*=0.179		0.89	*p*=0.346	

TABLE14: ASSOCIATION BETWEEN PLATELET LARGE CELL RATIO AND CARDIAC TROPONIN –T RESULTS

FIG 9:THE BAR DIAGRAM REPRESENTATION OF THE MEAN OF ALL THE PLATELET INDICES (VIZ.MPV,PDW,P-LCR) IN VARIOUS MANIFESTATIONS OF ACS AND THEIR COMPARISON WITH CONTROL GROUP



DISCUSSION

Myocardial Infarction is a major cause of morbidity & mortality in industrialized countries[2].Though a large number of risk factors are known, they explain only a part of the cases[1]. The aeitology of Ischaemic Heart Disease, is without doubt multifactorial[9]. It is likely that platelet activation plays a central role in the transformation of atherosclerotic cardiovascular disease (CVD) into its potentially major adverse clinical events, such as ischemic stroke and myocardial infarction.[58]

The present study included 175 patients diagnosed as Acute Coronary Syndrome.175 age & sex matched healthy controls were also included in this study. Patients with bleeding diathesis, previous stroke, major operations or significant trauma in the past two weeks[2] or hypertension (>180/110 mm of Hg)[2,20] were excluded as studies have shown persisting high platelet indices values in these cases. Of the 175 cases, 79 were diagnosed as STEMI, 58 cases were of Unstable Angina & 38 cases were of NSTEMI.

AGE

The average age of onset of CVD is younger (below 55 years) among Indians than in other populations around the world.[59,60.]

In the present study, the ages ranged from 28 to 89 years. The mean age in our study was 57.76 ±13.19 years. Majority of patients diagnosed as Acute Coronary Syndrome belonged to the 5th decade of life (29.71%), followed by 6th decade (22.86%) and 4th decade (18.86%) of life. This is in accordance with the Asian population at risk for an Ischaemic Heart Disease, occurring one decade earlier than in developed countries [60]. 50% of CHD-related deaths in India occur in people <70

years of age, whereas only 22% of CHD-related deaths in western countries occur in this age group[61].

GENDER :

The prevalence of cardiovascular disease is higher in males than females though the mortality due to CVD is higher in females. The Framingham study showed that women have a lower incidence of coronary artery disease than men do up until age 75 [59,60,62]

In the present study, total number of males including both cases and controls were 223 (63.71%) and number of females were 127 (36.28%).This percentage was almost similar to that in other studies on the correlation between MPV and ACS [4,60].

The total number of males presenting with acute coronary syndrome among the cases were 110(62.86%) and females affected were 65(37.14%). The male to female ratio was 1.7:1.

ASSESSMENT OF RISK FACTORS :

Prevalence of CVD in urban Indian population is between 6.5% to 13.2% and in the rural population between 1.6% to 7.4%. The prevalence in rural area is growing rapidly possibly due to changing life styles [63]. Asian Indian have been shown to manifest CVD at lower levels of these risk factors, as compared to other population[64]. In our study, distribution of risk factors was similar to that in another study carried out specifically on the relation between the risk factors of ACS and MPV [4].

SMOKING :

Smoking has been identified as one of the important risk factor for all CVD. Smokers have an approximately two-fold higher risk of cardiovascular disease compared with non-smokers.[65.]

Nevertheless the risk of cardiovascular disease is roughly proportional to cigarette consumption and the risk persists even at low level of smoking, that is, one to two cigarettes per day and recent studies have shown that environmental tobacco smoke is a risk factor for Ischemic Heart Disease. Passive smoking increases the coronary death rate among non smokers by 20% to 70%.[66]

TABLE 15: COMPARISON OF PREVALENCE OF SMOKING WITH OTHER STUDIES

	KHANDEKAR MM ET AL.[4] 2006	JOSHI ET.AL[67] 2007	YAGHOUBI ET.AL[60] 2013	PRESENT STUDY
PREVALENCE SMOKING	19.1%%	61.6%	30.5%	29.7%

A study done by Khandekar MM et al.[4] showed higher incidence of smoking in patients presenting with ACS (21.4%)as compared to controls (5%). These results are not in correlation to our study which showed 29.7% cases as compared to 17% controls. Our study was almost in concordance with Yaghoubi et al.[60] study which had 30.5% cases with positive history of smoking.

ALCOHOL CONSUMPTION :

Studies have shown that regular and moderate alcohol intake is associated with low risk of IHD and heavy or binge drinking was associated with high risk of IHD.[68,69] However , a study on acute MI patients revealed that alcohol consumption in south Asians was not protective against CHD[53] .

Alcohol consumption was observed in among 46.9% cases in our study and in only 7.2% controls. These was in contrast to study done by Joshi et al [67]which had only 13.3% cases. As the exact amount of alcohol intake was not available we could not stratify the patients into moderate or heavy alcohol intake groups

DIABETES MELLITUS :

Diabetes is a major risk factor for heart disease, and heart disease is responsible for substantial morbidity and mortality among people living with diabetes.[70]

Those with diabetes have 2-4 fold higher risk of developing coronary disease than people without diabetes.[71]

When CAD occurs in diabetics, the course of the disease is particularly aggressive and associated with worst outcomes than in non-diabetics. The risk of these complications is also greater for women than for men.[70]

In contrast ,the age and sex adjusted mortality risk in diabetic patients without pre-existing coronary artery disease has been found to be equal to that of non-diabetic individuals with prior MI**.** [71,72]

DM was seen in 19.4% patients in our study & 6.7% cases in the control group with an odd ratio of 3.28.Male predilection was noted in our study with 71.73% males

and 28.26% females being diabetic which was in contrast with the study by Linda et al[70] which showed greater risk among females.

Study done by Kodiatte T.A et al.[73] which studied platelets in Type-2 DM showed more male diabetics compared to females with 65% and 35% respectively in their study which are nearly similar to our findings.

Our study was also in accordance to studies like Gupta R et al [74] who quoted ICMR survey which studied risk factor prevalence of Non-communicable Disease among men and women in 8 Indian states showing higher male predilection of DM than female.

TABLE 16 : COMPARISON OF PREVALENCE OF DIABETES MELLITUS WITH OTHER STUDIES

	KHANDEKAR MM ET AL. [4] 2006	JOSHI ET AI [67] 2007	ALI MK ET AL[72] 2010	YAGHOUBI ET.AL[60] 2013	**PRESENT STUDY**
DIABETES MELLITUS	**14.8%**	**20.2%**	**30.4%**	**14.8%**	**19.4%**

Study by Ali MK[72] showed a higher prevalence of Diabetes mellitus(30.4%) while individual study done by Khandekar MM et al.[4] and Yaghoubi et al [60] on Indian population showed 14.8% diabetics in their study.

Our study had 19.4% diabetics which was nearly same as diabetics found in the studies by Khandekar MM et al.[4] and Yaghoubi et al[60]

HYPERTENSION

Hypertension was seen in 17.7% patients of which 62.61% were males and 37.38% were females. In our study hypertension was seen more among the control (24.3%) in comparison to the cases.

TABLE 17: COMPARISON OF PREVALENCE OF HYPERTENSION WITH OTHER STUDIES

	Khandekar MM et al. [4] 2006	JOSHI ET.AL [67] 2007	YAGHOUBI ET.AL [60] 2013	PRESENT STUDY
PREVALENCE HYPERTENSION	**24.4%**	**29.6%**	**42.4%**	**17.7%**

Yaghoubi et al.[60] study showed a higher prevalence of hypertension (42.4%) when compared to Khandekar et al.[4] which showed 24.4% cases and our study (17.7%). This variation can be attributed to the regional variation of hypertension as per Gupta et al.[63] which shows higher prevalence of hypertension in North Indian states compared to South India.

FAMILY HISTORY OF IHD:

Premature coronary heart disease in a first-degree relative (male relative <55 years and female <65 years or <60 years in both genders) is associated with increased risk of coronary heart disease.[65] Family history seemed to be slightly more important in young (14·8%) compared with old individuals (10·4%).[53]

However, our study showed only 2-3% cases associated with positive family history.

Among the risk factor, we found cigarette smoking, alcohol consumption & diabetes mellitus as significant risk factors for Ischaemic Heart Disease However,. it was observed that positive family history and hypertension were statistically not significant.

PLATELET INDICES:

Platelet Indices (MPV, PDW & P-LCR) were analysed in patients with Acute Coronary Syndrome & compared with healthy control group.

The MPV values evaluated in our study were 8.14 ± 0.67fl for the control group, 8.53 ± 0.54fl for unstable angina, 9.67 ± 0.82fl for STEMI & 9.54 ± 0.76 for NSTEMI.

We found that MPV was raised in patients who have suffered an acute coronary event when compared with controls and those with unstable angina. This is in agreement with the results of similar studies by other workers.

TABLE 18: COMPARISON OF MPV IN AMI AND CONTROLS IN DIFFERENT STUDIES:

PUBLICATION	CASES	MPV(fl)	CONTROLS	MPV(fl)	p Value
O'Brien et al (1973)	23	8.10	36	7.01	<0.001
Cameron et al (1983)	100	9.07	200	8.32	<0.001
Martin et al (1983)	15	7.3	22	6.32	0.05
Martin et al (1991)	126	10.09	1590	9.72	<0.001
Smyth et al (1993)	24	8.54	23	8.1	0.04
Pizulli et al (1998)	108	9.4	97	8.2	<0.001
Khandekar M M et al(2006)	94	10.43	30	9.2	<0.001
Lippi G et al(2009)	456	7.4	1848	8.0	<0.001
Chu S G et al(2010)	911	9.24	1898	8.48	<0.001
Khode et al(2012)	*39*	*9.65*	*65*	*9.21*	*0.018*
Present Study	175	9.605	175	8.33	<0.0001

In ACS, rupture of unstable atherosclerotic plaque triggers a thrombogenic cascade leading to clinical events. However, platelet reactivity is critically important in the formation and propagation of intracoronary thrombus.[41] MPV, one of the markers indicating the function of platelets, is a simple and easy measurement.[53]

The MPV was significantly ($p<0.0001$) raised in patients with MI in comparison to healthy control population in our study. This is in agreement with the results of similar studies by other workers.[4,5,7,27,45,53,54,55,57,58,60,75,76,77] In these studies increased MPV was found to be associated with coronary artery disease, acute MI, congestive heart failure and hypertensive patients with evidence of target organ

damage and cerebrovascular disease,[20] an important complication of atherosclerosis. Substantial evidence indicates that platelets and their interaction with the coronary arterial wall are of pathogenic importance in coronary atherosclerosis and its complications. After erosion or rupture of atherosclerotic plaques in coronary arteries, platelet activation plays a crucial role in the prothrombotic events leading to MI. Increased platelet reactivity is associated with increased platelet volume.[4,5,53] As mentioned earlier large platelets that contain more dense granules are metabolically and enzymatically more active than small platelets and have higher thrombotic potential[4]. The size of platelets has been found to associated with an increased number of megakaryocytes. The increased ploidy of megakaryocytes is correlated with megakaryocyte and platelet volume.[77,78,79,80] Elevated levels of CD40 ligands, which are expressed by activated platelets, have been found in atheromatous plaques.[81] Pizulli et al.[54] suggested that because platelets stay in the circulation for 7–11 days, they might be detected days before symptoms appear. Similarly, Martin et al.[75] have shown a correlation between higher MPV and recurrence or death after the first MI in their prospective study.

Chu H et al[82] showed MPV is significantly associated with ACS in patients with acute chest pain and is an early and independent predictor.

Chu SG et al.[7] review demonstrated that elevated MPV is associated with acute MI, mortality following MI, and restenosis following coronary artery intervention.

Mathur et al[83] studies have shown higher MPV values in patients with UA (10.7fl) than those with MI(9.8fl),but our study found no such difference which compares well with a study by Senaran et al[84] in which Mean platelet volume was found to be

elevated in patients with AM1 (8.2 ± 0.8 fl) and UA(7.7 ± 0.5fl) compared with control subjects (6.6 ± 0.6 fl)

PLATELET DISTRIBUTION WIDTH

The PDW was significantly higher in the patients diagnosed with ACS (13.66 fl) as compared to the control group. (12.23fl)

TABLE 19 : COMPARISON OF PLATELET DISTRIBUTION WIDTH WITH OTHER STUDIES

PLATELET DISTRIBUTN WIDTH(fl) (SD	Khandekar MM et al. [4] 2006	KHODE[55] ET.AL 2007	Obeidi ET.AL [58] 2009	PRESENT STUDY
MI GROUP	13.19	10.84	21.6	13.66
NON MI GROUP	11.35	10.65	21.1	13.41
CONTROLS	10.75	10.35	15	12.23
SIGNIFICANCE (p value)	<0.001	0.376	<0.007	0.027

The role of PDW specifically in patients with CAD and acute coronary events is yet to be explored.[4] Nevertheless our observations showed that PDW was

significantly elevated among the patients as compared to the non-MI patients and the healthy control group.

Similar results were noted by other studies like Khandekar et al.[4] Khode et al[55]. with higher PDW levels among MI patients but this levels were significant only in few studies [4].

Vagdatli E et al.[57] in their study on puerperas in different trimesters, MI patients and those with phlebothrombosis and healthy people concluded that PDW seemed to be more specific indicator of platelet activation than MPV, since it was not elevated during single platelet distention caused by platelet swelling. And the combined use of MPV and PDW could predict activation of coagulation more efficiently.

PLATELET-LARGE CELL RATIO

The PLCR parameter is generated by only a few machines, with the Sysmex analyser being one of them. It is not often quoted in the literature, probably because it is a relatively new PVI parameter.[4]

TABLE 20 : COMPARISON OF PLATELET LARGE CELL RATIO WITH OTHER STUDIES

PLATELET LARGE CELL RATIO	Khandekar MM et al. [4] 2006	KHODE[55] ET.AL 2007	Bhayana et al[79] 2009	PRESENT STUDY
MI GROUP	29.4	21.58	17.06	22.23
NON MI GROUP	22.55	20.92	17.06	18.57
CONTROLS	20.65	19.93	16.81	18.12
SIGNIFICANCE (p value)	<0.001	0.315	0.376	0.179

Our study show that P-LCR is not a reliable marker for predicting an acute coronary event. This is in agreement with the studies by Khode[55] et al. and Bhayana et al[79]. However these results are in contrast to study of Khandekar MM et al [4].

Our data suggest that the increased platelet volume indices contributes to the prethrombotic state in acute ischaemic syndromes and that larger platelets may play a specific role in infarction. Because larger platelets are haemostatically more active, the presence of larger platelets is probably a risk factor for developing coronary thrombosis and MI. Patients with larger platelets can easily be identified during routine haematological analysis because PVI are generated as a by product of automated blood counts. Thus, in conclusion, PVI provides an important, simple,

effortless, and cost effective tool, which can be useful in predicting an impending acute coronary event.

LIMITATIONS OF THE STUDY

- Follow up of the patients was not possible to examine the prognostic value of our findings.
- Patients with qualitative disorders and causes of reactive platelets were not assessed.
- Platelet function tests could not be conducted on the sample to substantiate our findings further.

SUMMARY & CONCLUSION

- This study was undertaken in Shri B.M.Patil Medical College, Bijapur, Karnataka to study efficacy of platelet parameters in Acute Coronary Syndromes
- A total of 350 cases were studied and were divided further into two groups of 175 patients each, who were patients diagnosed with ACS and age and sex matched healthy controls.
- Majority of patients diagnosed as Acute Coronary Syndrome belonged to the 5^{th} decade of life (29.71%), followed by 6^{th} decade (22.86%) and 4^{th} decade (18.86%) of life.
- In the present study, total number of males including both cases and controls were 223 (63.71%) and number of females were 127 (36.28%).
- The number of males in the MI group were 110 (62.86%) compared to non - MI group 113 (64.57%)
- The number of females in the MI group were 65(37.14%) compared to non - MI group 62 (35.42%)
- The total number of smokers were 52(29.7%) with more smokers being seen in the MI group.
- Alcohol consumption was observed only among 82 (46.9%) patients in our study. MI group had more alcoholics than non-MI group (7.2%).
- Our study had 34(19.4%) diabetics showing a male predilection with (71.73%) male diabetics.

- Hypertension was seen in 31(17.7%) patients, of which 62.61% were males and (37.38%) were females. Hypertension was more prevalent among non-MI (24.3%)than MI patients

- In our study family history of IHD was noted in only 4(2.3%) patients
- The MPV was highest in the MI group 9.67(±0.82)f L than non-MI patients with 8.53(± 0.54)fL and control group8.14(± 0.67) fL, which was statistically significant ($p<0.0001$).This was in accoradance with elevated Troponin –T levels.(95.4%)
- The PDW was significantly higher in the patients of MI group 13.66(± 3.55) fL than non-MI patients with 13.41*(*± 4.02)fL and the control group 12.23(± 3.13) fL. This was in accoradance with elevated Troponin –T levels. (94.87%)
- The P-LCR recorded in our study for MI group was higher and *(*22.36 ± 4.95fL), in comparison to the non-MI group 18.57 ± 3.70fL the control group*(*18.12 ± 3.54fL).The p-value for P-LCR was however not statistically significant for evaluating a cardiovascular patients profile.

CONCLUSION

The study was undertaken to determine whether an association exists between platelet indices - mean platelet volume (MPV), platelet distribution width (PDW), Platelet large cell ratio (P-LCR) in acute myocardial infarction and other ischemic cardiac events.

These indices are useful means of identifying larger and more active platelets which are a risk factor for developing coronary thrombus.

Such patients can easily be identified during routine hematological analysis and possibly benefit from preventive and anti-platelet treatment.

Our study concluded that among the platelet indices, mainly Mean Platelet Volume (MPV) and Platelet Distribution Width (PDW) are readily available, relatively inexpensive useful markers which were significantly raised among patients admitted with MI in our hospital. Platelet Large Cell Ratio did not show any significant association among the patients and healthy population. Thus these should be utilized with other investigative tools for timely management of patients diagnosed with acute coronary syndrome.

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PROFORMA FOR THE STUDY OF PLATELET INDICES

NAME**:** CASE NO:

AGE: IP NO:

SEX**:** DOA:

OCCUPATION: DOD:

RESIDENCE:

Chief complaints:

History of present illness:

Past history:

Family history:

Personal history:

General physical examination:

Vitals:

Pulse Rate:

Blood Pressure:

Respiratory Rate:

Systemic examination:

- Cardiovascular system
- Respiratory system
- Central Nervous System
- Per Abdomen Examination

Clinical diagnosis:

Hematological investigations: (Complete blood count)

Parameters	
WBC	
RBC	
HGB	
HCT	
MCV	
MCH	
MCHC	
PLATELETS	
LYMPHOCYTES(%)	
MIXED (%)	
NEUTROPHILS(%)	
RDW	
PDW	
MPV	
P-LCR	

Peripheral Smear Examination:

RBC:

WBC:

PLATELETS:

IMPRESSION:

Biochemistry:

i. Cardiac Troponin
ii. CPK-MB
iii. Triglycerides
iv. HDL Cholesterol

Other investigations : i) 12 LEAD ECG ii) 2D ECHO

Ethical Committee BIJAPUR-586103 OUT WARD No 27/2011 Date 21-10-11 BLDE's Shri B M. PATIL Medical College

B.L.D.E. UNIVERSITY'S

SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR-586 103

INSTITUTIONAL ETHICAL COMMITTEE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 20-10-2011 *at* 10-30 am *to scrutinize the Synopsis/Research projects of postgraduate/undergraduate student/Faculty members of this college from Ethical Clearance point of view. After scrutiny the following original/corrected & revised version synopsis of the Thesis/Research project has been accorded Ethical Clearance.*

Title "Study of platelet indices in patients with acute coronary syndrome"

Name of P.G./U.G. student/Faculty member Dr. Jaya Manchanda Dept of Pathology

Name of Guide/Co-investigator Dr. R.M. Patekar, Prof of Pathology

DR.M.S.BIRADAR,
CHAIRMAN
INSTITUTIONAL ETHICAL COMMITTEE
BLDEU'S, SHRI.B.M.PATIL
MEDICAL COLLEGE, BIJAPUR.

Chairman
Ethical Committee
BLDEA'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.

KEY TO MASTERCHART

Age:

AGE IN YEARS	ALLOTTED NUMBER
11-20	1
21-30	2
31-40	3
41-50	4
51-60	5
61-70	6
71-80	7
81-90	8

Sex-

Male-1,female-2

Past history-

PAST HISTORY	ALLOTTED NUMBER
DIABETES	1
HYPERTENSION	2
HISTORY OF DRUG INTAKE	3
NOTHING SIGNIFICANT	4
ALCOHOL	5
CIGARETTE SMOKING	6

Family history-

FAMILY HISTORY	ALLOTTED NUMBER
IHD	1
DIABETES	2
HYPERTENSION	3
NOTHING SIGNIFICANT	4

Clinical Diagnosis-

CLINICAL DIAGNOSIS	ALLOTTED NUMBER
UNSTABLE ANGINA	1
STEMI	2
NSTEMI	3

WBC - (4000-11,000)NORMAL-1 >11,000-2

RBC - NORMAL RANGE(4.5-5.5 million/cumm)-1,ABNORMAL-2

HBG - (0-5GM%)-1,(5-11GM%)-2,(12-15GM%)-3

HCT - NORMAL RANGE(38-50%)-1,ABNORMAL-2

MCV - NORMAL RANGE (80-100fL)-1,ABNORMAL-2

MCH - NORMAL RANGE(27-32pg)-1,ABNORMAL-2

MCHC- NORMAL RANGE(30-35gm/dl)-1,ABNORMAL-2

PLATELET- NORMAL RANGE(1.5-4LAKHS)-1,ABNORMAL-2

LYMPHOCYTES- NORMAL RANGE(UPTO 40%)-1,ABNORMAL-2

MIXED- NORMAL RANGE-1,ABNORMAL-2

NEUTROPHILS- NORMAL RANGE(UPTO 70%)-1,ABNORMAL-2

RDW - NORMAL RANGE(11-15%)-1,ABNORMAL-2

CARDIAC TROPONIN-POSITIVE-1,NEGATIVE-2

CPKMB- NORMAL(0-25)-1,ABNORMAL(>25)-2

ECG -WITHIN NORMAL LIMIT-1,STEMI-2,NSTEMI-3

2D-ECHO-NORMAL-1,ABNORMAL-2,NOT DONE-3

MASTER CHART

S NO	NAME	PDW	MPV	P-LCR	SEX	PAST HISTORY	FAMILY HISTORY	CLINICAL DIAGNOSIS	WBC	RBC	HBG	HCT	MCV	MCH	MCHC	PLATELETS	LYMPHOCYTES	MIXED	NEUTROPHILS	RDW	PDW	MPV	P-LCR	TROPONINCPK-MB		CHOLESTEROLHDL	ECG	ECHO	AGE
1	SUSULABAI	10.1	8.4	14.5	2	2	4	2	2	1	3	1	1	1	1	1	1	1	2	1	1	1	1	2	2	2	2	3	65
2	MAMTAJ	10.9	9.9	18.1	2	12	4	2	2	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	2	2	3	63
3	SIDDAMMA	12	9.5	22.6	1	5	4	1	1	1	3	1	1	1	1	1	1	1	2	1	1	2	1	2	1	1	1	3	56
4	YAMUPPA	11.5	9.2	21.1	1	2	4	2	1	1	3	2	1	1	1	1	1	1	2	1	1	2	1	1	2	1	2	3	55
5	KHAZABEE	10.5	9	16.3	2	12	1	2	2	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	1	2	2	60
6	VIJAY	10	9.4	16.6	1	5	4	2	2	1	3	1	2	1	1	1	1	1	1	1	1	2	1	1	2	3	2	3	59
7	SHIVAPPA	13.6	8.1	27.4	1	256	4	1	2	1	2	1	1	1	1	1	1	1	2	1	1	1	2	2	2	1	1	3	51
8	CHANDRABAI	11.2	12.1	26.1	2	2	4	2	1	1	3	1	1	1	1	1	1	1	2	1	1	4	2	1	1	1	2	3	60
9	ABDUL	16.1	10.2	16.1	2	256	4	1	1	1	2	1	1	1	1	1	1	1	1	1	2	3	1	2	1	1	1	3	80
10	SANGANNA	15.4	9	20.4	1	26	4	2	2	1	2	1	1	1	1	1	1	1	2	1	2	2	1	1	2	3	2	3	50
11	TIPPANNA	15	9.2	21.2	1	5	4	2	1	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	3	2	3	60
12	KASIBAI	11.6	9.4	21.2	2	2	4	2	1	1	2	1	1	1	1	1	1	1	2	1	1	2	1	1	1	3	2	3	80
13	SUKUMARI	10.1	8.5	15.8	2	2	4	2	2	1	3	1	1	1	1	1	1	1	2	1	1	1	1	1	1	1	2	3	78
14	MAHANGUDDAPPA	11	9.1	19	1	5	4	1	1	1	3	1	1	1	1	1	1	1	2	1	1	2	1	2	1	1	1	1	52
15	BHAHASAB	15.3	10.7	30.3	1	6	4	2	1	1	3	1	1	1	1	1	1	1	1	1	1	3	2	1	2	3	2	3	65
16	SHANKARAPPA	10.2	8.8	18.1	1	56	4	3	2	1	3	1	1	1	1	1	1	1	2	1	1	1	1	2	2	3	2	3	65
17	GURUNDAPPA	9.2	8.2	12.7	1	254	4	2	2	1	3	1	2	2	1	1	1	1	2	1	1	2	1	1	2	1	2	3	72
18	ARJUN	12.2	9.3	22.5	1	5	4	3	2	2	3	1	1	1	1	1	1	1	2	1	1	2	1	1	1	3	3	2	55
19	KUSUMA	19.5	12.2	42.3	2	2	4	2	2	1	2	1	1	1	1	1	1	1	2	1	1	4	3	1	2	1	2	3	48
20	GOPAL	14.6	9.6	24.9	1	5	4	2	2	1	1	1	1	1	1	1	1	1	2	2	1	2	1	1	2	1	2	3	55
21	MALAKAPPA	8.4	7.4	18.9	1	5	4	3	1	1	3	1	1	1	1	1	1	1	1	1	1	1	1	1	2	1	2	3	55
22	UMAKANT	8.5	9.8	18.6	1	2	4	3	1	1	3	1	1	1	1	1	1	1	1	1	1	2	1	2	2	1	3	3	56
23	VEERESH	10.7	8.9	17.9	1	6	4	2	1	1	3	1	1	1	1	1	2	1	1	1	1	1	1	3	1	1	2	3	47
24	MABISAB	9.8	8	13.3	1	5	4	2	2	2	3	1	1	1	1	1	1	1	2	1	1	2	1	2	1	3	2	3	75
25	KASTURIBAI	11.2	8.9	17.6	2	5	4	1	1	1	3	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	1	69
26	GUNDU	11.5	9.4	22.1	1	6	4	3	2	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	1	1	3	2	45

S NO	NAME	PDW	MPV	P-LCR	SEX	PAST HISTORY	FAMILY HISTORY	CLINICAL DIAGNOSIS	WBC	RBC	HBG	HCT	MCV	MCH	MCHC	PLATELETS	LYMPHOCYTES	MIXED	NEUTROPHILS	RDW	PDW	MPV	P-LCR	TROPONINCPK-MB		CHOLESTEROLHDL	ECG	ECHO	AGE
27	NISAR	13.5	9.4	24.6	1	5	4	2	2	1	2	1	1	1	1	1	1	1	2	1	1	2	1	1	2	3	2	3	74
28	MANAPPA	8.5	7.8	11.4	1	1	4	1	1	1	3	1	1	1	1	1	1	1	2	1	1	1	1	2	1	3	1	3	65
29	NABISAB	10.9	8.7	16.7	1	1	4	1	1	1	3	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	3	65
30	BASAVRAJ	11	9.4	21.2	1	5	4	2	2	1	3	1	2	1	1	1	1	1	2	1	1	2	1	2	2	1	2	3	30
31	RANGAPPA	15.4	9.8	24.6	1	2	4	2	1	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	1	2	3	70
32	SOMANNA	13.9	9.7	28.4	1	6	4	2	1	1	3	1	1	1	1	1	1	1	1	1	1	2	1	1	2	3	2	3	55
33	IMANSAB	19.6	11.6	37.7	1	16	4	2	2	1	3	1	1	1	1	1	1	1	1	2	1	1	3	1	2	3	2	2	74
34	KASHIBAI	9.4	8	12.1	2	5	4	3	2	1	2	1	1	1	1	1	1	1	2	1	1	2	1	1	2	1	3	3	58
35	KAIKILARI	9.4	8	12.1	2	5	4	3	2	1	2	1	1	1	1	1	1	1	2	1	1	2	1	1	2	1	3	3	58
36	JAYASHREE	13.3	9.4	22.9	2	5	4	1	1	1	2	1	1	1	1	1	1	1	2	1	1	2	1	2	1	1	1	1	45
37	BASAVRAJ	10	8.5	15.8	1	126	4	3	2	1	3	1	2	2	1	1	1	1	2	1	1	2	1	1	2	3	3	1	50
38	BAVANTRAY	12.2	9.6	23.9	1	5	4	2	2	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	3	2	2	74
39	MAYAWWA	9.5	8.4	12.2	2	2	4	2	1	1	2	1	1	1	1	1	1	1	2	1	1	2	1	1	2	1	2	3	60
40	PRABHAVATI	12.3	8.4	24	2	5	4	1	1	1	3	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	3	57
41	KAUVERY	9.6	12.3	22.2	2	5	4	2	1	1	3	1	1	1	1	1	1	1	2	1	1	4	1	1	2	3	2	3	28
42	SIDARAMPA	14.1	8	18.1	1	2	4	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	2	1	1	1	3	55
43	VITTAL	11.7	9.5	22.1	1	5	4	2	1	1	1	1	1	1	1	1	1	1	1	1	1	2	1	1	2	3	2	3	60
44	MALAPPA	12.8	10.1	26.6	1	2	4	2	1	1	3	1	1	1	1	1	1	1	1	1	1	3	2	2	2	3	2	3	79
45	KESU	12.6	9.8	24.2	1	56	4	3	2	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	1	1	3	3	60
46	MADAPPA	11.4	9.1	19.1	1	4	4	2	2	1	3	1	1	1	1	1	1	1	2	1	1	2	1	2	2	3	2	3	50
47	SUHAKAR	15.8	8.1	18.2	1	4	4	1	2	1	3	1	1	1	1	1	1	1	1	1	1	1	1	2	1	1	1	1	32
48	SHIVASANGRAM	10.7	9.3	20	1	4	4	2	2	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	1	2	3	28
49	RATNABAI	11.8	10	22.6	2	2	4	2	2	1	3	1	1	1	1	1	1	1	2	1	1	3	1	1	2	3	2	3	62
50	SAYABHAVA	13.1	9.6	25.1	2	4	4	3	2	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	1	2	3	48
51	RANAPPA	12.2	9.9	22.3	1	4	4	3	1	1	3	1	1	1	1	1	1	1	2	1	1	3	1	1	2	1	2	3	75
52	SHARANAPPA	11	9.8	22	1	56	4	2	1	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	1	2	3	57
53	HANUMANTH	11.8	9.4	22	1	5	4	3	2	1	2	1	1	1	1	1	1	1	2	1	1	2	1	1	2	3	3	3	60
54	NINGAPPA	8.9	7.7	11.3	1	4	4	2	2	1	3	1	1	1	1	1	1	1	2	1	1	1	1	2	2	3	2	3	48
55	VITTAL	12	8.4	21.6	1	4	4	1	1	1	3	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	3	54

S NO	NAME	PDW	MPV	P-LCR	SEX	PAST HISTORY	FAMILY HISTORY	CLINICAL DIAGNOSIS	WBC	RBC	HBG	HCT	MCV	MCH	MCHC	PLATELETS	LYMPHOCYTES	MIXED	NEUTROPHILS	RDW	PDW	MPV	P-LCR	TROPONINCPK-MB		CHOLESTEROLHDL	ECG	ECHO	AGE
56	CHANDRAKANT	11.2	10	22.1	1	4	4	3	2	1	3	1	1	1	1	1	1	1	2	1	1	3	1	1	2	1	3	2	45
57	NIZAMUDDIN	11.5	9.3	20.3	1	125	4	3	1	1	1	1	1	1	1	1	1	1	1	1	1	2	1	1	2	3	3	3	51
58	MAINABAI	11.1	8.2	19.1	2	2	4	1	1	1	3	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	3	70
59	SAYABANNA	13.1	9.6	25.1	1	4	4	3	2	1	1	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	3	3	48
60	ITABAI	13	9.8	22.9	2	4	4	2	1	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	1	2	3	70
61	ISHWARAMMA	13.8	9.8	22.3	2	2	4	3	2	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	2	3	2	75
62	SAROJNI	12.5	8.8	21.9	2	2	4	1	1	1	3	1	1	1	1	1	1	1	2	1	1	1	1	2	1	2	1	3	72
63	MAHADEVAPPA	11.4	9.4	21.2	1	4	4	3	2	1	1	1	1	1	1	1	1	1	2	1	1	2	1	1	2	2	3	3	65
64	SIDAPPA	13.1	10	26.3	1	4	4	2	1	1	1	1	1	1	1	1	1	1	1	1	1	3	2	1	2	3	2	3	60
65	GANGAMMA	15.4	7	19	2	4	4	1	2	1	2	1	1	1	1	1	1	1	2	1	1	1	1	2	1	3	1	3	72
66	YEMENAPPA	14.3	11.2	34.6	1	1	4	3	1	1	3	1	1	1	1	1	1	1	2	1	1	4	2	1	2	3	3	3	65
67	MALLIKARJUN	9	8	12.3	1	4	4	1	1	1	2	1	1	1	1	1	1	1	1	1	1	1	1	2	2	3	1	3	48
68	PUNDAKKAPPA	15.8	8.7	18	1	4	4	1	1	1	2	1	1	1	1	1	1	1	1	1	1	1	1	2	1	1	1	3	60
69	YAMANAWWA	16.3	8.6	21	2	4	4	1	1	1	2	1	1	1	1	1	1	1	2	1	1	1	1	2	2	1	1	3	60
70	HAFISABAI	14.2	10	27	2	2	4	3	2	1	2	1	1	1	1	1	1	1	2	1	1	3	1	2	2	1	3	3	85
71	ABDUL	11.1	9.4	20.1	1	2	4	2	2	1	3	1	1	1	1	1	1	1	2	1	1	2	1	2	2	3	2	3	54
72	ASHOK	11.2	8.7	17.9	1	4	4	1	1	1	3	1	1	1	1	1	1	1	1	1	1	1	1	2	2	3	1	3	42
73	NASIR	10.7	9	18	1	56	4	2	1	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	1	2	3	50
74	YEMENAWWA	19	9.6	22.2	2	5	4	3	2	1	3	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	3	3	55
75	BANDENAWAZ	10.5	8.6	15.7	1	4	4	1	1	1	3	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	3	50
76	SRIYAPPA	12.1	9.4	21	2	5	4	2	2	1	3	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	2	3	45
77	SHARADHA	10.1	9.2	16.5	2	4	4	2	1	1	3	1	1	1	1	1	1	1	1	1	1	2	1	1	2	3	2	3	32
78	MALLAPPA	19	8.2	16.4	1	5	4	1	1	1	3	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	3	45
79	BAYAWWA	18.2	9.8	20	2	12	4	2	1	1	2	1	1	1	1	1	1	1	2	1	1	2	1	1	2	3	2	2	75
80	HANUMANTH	15.5	7.8	18.6	1	4	4	1	1	1	3	1	1	1	1	1	1	1	2	1	1	1	1	2	2	3	1	1	42
81	ABDUHUL	12.5	10.1	21.2	1	12	4	2	1	1	3	1	1	1	1	1	1	1	2	1	1	3	1	2	2	1	2	2	63
82	GURUBAI	13	10.4	18.1	2	4	4	2	1	1	3	1	1	1	1	1	1	1	1	1	1	3	1	1	2	3	2	2	68
83	YAMANAPPA	16	9.6	18.2	1	16	4	2	2	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	3	2	2	58
84	AMNA SAHEB	15.9	10.6	23.4	1	4	4	2	2	1	3	1	1	1	1	1	1	1	1	1	1	3	1	1	2	3	2	2	32
85	GAIBISAB	12.6	9.6	20.2	1	5	4	3	2	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	1	3	2	40
86	HUCHAPPA	17.2	9.8	23.2	1	4	4	3	1	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	1	3	2	37

S NO	NAME	PDW	MPV	P-LCR	SEX	PAST HISTORY	FAMILY HISTORY	CLINICAL DIAGNOSIS	WBC	RBC	HBG	HCT	MCV	MCH	MCHC	PLATELETS	LYMPHOCYTES	MIXED	NEUTROPHILS	RDW	PDW	MPV	P-LCR	TROPONINCPK-MB		CHOLESTEROLHDL	ECG	ECHO	AGE
87	RAMESH	11.9	9.4	24.2	1	4	4	1	1	1	3	1	1	1	1	1	1	1	1	1	1	2	1	2	1	3	1	3	43
88	SHANKARAPPA	11.7	9	16.2	1	4	4	1	1	1	3	1	1	1	1	1	1	1	1	1	1	2	1	2	2	3	1	3	50
89	ARUN KUMAR	15.4	9.9	23.4	1	2	4	2	1	1	2	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	2	3	46
90	SAROJINI	13.8	10	24.2	2	2	4	3	1	1	2	1	1	1	1	1	1	1	2	1	1	3	1	2	1	1	3	2	43
91	SARAJIRAO	13.8	9.7	24.6	1	4	4	2	2	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	3	2	3	66
92	SURYANKA	14.1	8.2	17.2	2	4	4	1	1	1	3	1	1	1	1	1	1	1	2	1	1	1	1	2	2	3	1	3	60
93	MOHAMMED	16.1	9.8	24.2	1	4	4	2	2	1	3	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	2	3	55
94	LAKSHMI BAI	15.7	9.2	21.9	2	2	4	2	2	1	2	1	1	1	1	1	1	1	2	1	1	2	1	1	2	1	2	2	80
95	BASAVARAJ	14.2	9.9	22.1	1	6	4	3	1	1	3	1	2	1	1	1	1	1	2	1	1	2	1	1	2	3	3	2	56
96	KASTURI BAI	11.2	8.9	17.6	2	4	4	1	1	1	3	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	1	59
97	SHAEB GOUDA	11	9.8	22	1	4	4	2	1	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	3	2	3	49
98	PARU BAI	10.3	8.6	23.4	2	4	4	2	1	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	3	2	2	48
99	HUSSAIN SAB	12.1	9.6	19.7	1	4	4	3	1	1	3	1	1	1	1	1	1	1	1	1	1	2	1	1	2	3	3	3	59
100	KASTURI BAI	9.8	8.4	18.6	2	4	4	1	2	1	2	1	1	1	1	1	1	1	2	1	1	1	1	2	1	3	1	3	69
101	JANATHBEE	22.2	10.1	24.6	2	4	4	3	2	1	3	2	1	1	1	1	1	1	2	1	2	3	1	1	2	3	3	3	60
102	SUNDARA	17.4	9.9	22.4	2	5	4	2	1	1	2	1	1	1	1	1	1	1	1	1	1	2	1	1	2	3	2	3	65
103	MAHADEVAPPA	18.4	9.4	20.2	1	5	4	2	2	1	3	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	2	1	40
104	SANGANGOUDA	14.6	8.2	19.2	1	5	4	1	1	1	3	1	1	1	1	1	1	2	2	1	1	1	1	2	1	3	1	3	54
105	SHANTABAI	16.4	8.2	20.2	2	1	4	1	1	1	2	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	1	45
106	KALLAPPAGOUDA	20	9.2	24.2	1	5	4	3	1	1	2	1	1	1	1	1	1	1	1	1	2	2	1	1	2	3	3	2	55
107	CHANAMMA	18.6	9.4	20.2	2	5	4	2	2	1	1	2	2	2	1	1	1	1	1	2	1	2	1	1	2	3	1	3	70
108	SHANTABAI	18.4	10	24.8	2	1	4	2	1	1	3	1	1	1	1	1	1	1	1	1	2	3	1	1	1	3	2	1	65
109	IRAMA	18.6	9	21.4	1	4	4	2	2	1	1	3	1	1	1	1	1	1	1	1	1	2	2	1	1	1	1	2	52
110	SUGALABAI	16.4	8.3	22.1	2	5	4	1	2	1	2	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	3	72
111	JAMUNADAS	12.7	8.2	24.3	1	4	4	1	2	1	3	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	3	60
112	SANGAMMA	9	9.5	22.2	2	5	4	2	1	1	2	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	2	3	65
113	ATHMARAM	14.2	10.5	29.2	1	5	4	3	2	1	3	1	1	1	1	1	1	1	1	1	1	3	2	1	2	3	3	3	36
114	MS PATIL	9.5	8.3	12.8	1	5	4	1	1	1	2	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	3	46
115	ALLABAKSHA	13.5	10.5	18.4	1	16	4	2	2	1	3	1	1	1	1	1	1	1	1	1	1	3	1	1	2	3	2	3	65
116	HANAMANTH	12	10.4	26.7	1	46	4	2	2	1	3	1	1	1	1	1	1	1	1	1	1	3	2	1	2	3	2	3	40
117	GURUBAI	11.5	9.8	24.2	2	1	4	3	1	1	2	1	1	1	1	1	1	1	1	1	1	2	1	1	2	3	3	3	75
118	MALLINATH	11.9	9.6	23.1	1	6	4	2	1	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	3	2	3	30

S NO	NAME	PDW	MPV	P-LCR	SEX	PAST HISTORY	FAMILY HISTORY	CLINICAL DIAGNOSIS	WBC	RBC	HBG	HCT	MCV	MCH	MCHC	PLATELETS	LYMPHOCYTES	MIXED	NEUTROPHILS	RDW	PDW	MPV	P-LCR	TROPONINCPK-MB		CHOLESTEROLHDL	ECG	ECHO	AGE
119	SAHESH	9.8	8.5	14.2	1	5	4	1	1	1	3	1	1	1	1	1	1	1	2	1	1	1	1	2	1	3	1	3	35
120	YAMANAWWA	19	9.6	22.2	2	5	4	3	2	1	3	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	3	3	65
121	SAHEBGOUDA	11	9.8	22	1	5	4	2	1	1	3	1	1	1	1	1	1	1	1	1	1	2	1	1	2	3	2	3	41
122	LAXMIBAI	20.2	9.5	19.9	2	1	4	2	2	1	3	1	1	1	1	1	1	1	1	1	1	2	1	2	1	3	2	3	60
123	MALLAPPA	16.4	9.8	24.8	1	6	4	2	1	1	3	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	2	3	45
124	RAJESA	17.4	9.4	23.1	1	6	4	2	2	1	2	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	2	2	65
125	SHANTABAI	26.2	8.6	22.4	2	5	4	1	1	1	2	1	1	1	1	1	1	1	2	1	1	1	1	2	1	3	1	1	68
126	BABASABGOUDA	18.6	8.5	20.2	1	1	4	1	1	1	3	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	1	68
127	NEELAMMA	24.8	10	25.4	2	5	4	1	1	1	2	1	1	1	1	1	1	1	1	1	1	2	1	2	1	3	1	1	68
128	DANDAMMA	18.9	8.4	24.3	2	5	4	1	1	1	3	1	1	1	1	1	1	1	2	1	1	1	1	2	1	3	1	1	55
129	KESAPPA	23.4	8.9	21.4	1	5	4	1	1	1	3	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	1	67
130	RUDRAGOUDA	24.6	9.1	22.4	1	2	4	2	2	1	2	1	1	1	1	1	1	1	1	1	1	2	1	1	2	3	2	2	78
131	AMEENAPPA	24.1	9.7	22.4	1	5	4	2	2	1	2	1	1	1	1	1	1	1	1	1	1	2	1	1	2	3	2	3	89
132	SURAGAPPA	17.4	8.2	19.6	1	6	4	1	1	1	2	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	3	75
133	GURAMMA	19	9.6	24.3	2	1	4	2	1	1	2	1	1	1	1	1	1	1	1	1	1	2	1	1	2	3	2	2	83
134	PARVATI	17.4	8	19.1	2	5	4	1	2	1	2	1	1	1	1	1	1	1	1	1	1	1	1	2	2	3	1	3	75
135	KAASH	10.5	8.7	16.5	1	5	4	1	1	1	3	1	1	1	1	1	1	1	2	1	1	1	1	2	1	1	1	3	27
136	BABURAY	9.6	8.3	13.3	1	5	4	1	1	1	2	1	1	1	1	1	1	1	2	1	1	1	1	2	1	1	1	3	59
137	SUGALABAI	10.1	8.8	17	2	5	4	1	2	1	2	1	1	1	1	1	1	1	1	1	1	1	1	2	2	1	1	3	72
138	USHA	11.2	9.4	20.4	2	5	4	2	1	1	2	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	2	3	77
139	MAKAKAJAYYA	12.1	10.3	26.5	1	5	4	2	2	1	3	1	1	1	1	1	1	1	1	1	1	3	1	1	2	1	2	3	48
140	BASAVARAJ	9.4	8.2	13.2	1	5	4	1	1	1	3	1	1	1	1	1	1	1	2	1	1	1	1	2	1	1	1	3	38
141	BHIMAGOUDA	10.9	9.3	20.2	1	5	4	2	2	1	3	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	2	3	65
142	SANGAPPA	19.7	10.6	22.1	1	5	4	2	1	1	3	1	1	1	1	1	1	1	1	1	1	3	1	1	2	1	2	3	77
143	NANJAPPA	18.6	10	22.2	1	5	4	3	1	1	3	1	1	1	1	1	1	1	1	1	1	3	1	1	2	1	2	2	77
144	HANUMANTH	20.1	10.8	23.2	1	5	4	2	1	1	2	1	1	1	1	1	1	1	1	1	1	3	1	1	2	1	2	3	70
145	GURUBAI	10.4	8.8	15.5	2	5	4	3	2	1	2	1	1	1	1	1	1	1	1	1	1	1	1	1	2	3	3	3	62
146	SURYAKANTH	16.2	8.7	24.2	1	6	4	1	2	1	2	1	1	1	1	1	1	1	1	1	1	1	1	2	1	1	1	1	59
147	RAJENDRA	11.8	9.7	23.1	1	6	4	3	1	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	2	1	2	3	57
148	SHRISHAIL	9.7	8.4	13.7	1	5	4	2	2	1	3	1	1	1	1	1	1	1	1	1	1	1	1	1	2	1	2	3	45

S NO	NAME	PDW	MPV	P-LCR	SEX	PAST HISTORY	FAMILY HISTORY	CLINICAL DIAGNOSIS	WBC	RBC	HBG	HCT	MCV	MCH	MCHC	PLATELETS	LYMPHOCYTES	MIXED	NEUTROPHILS	RDW	PDW	MPV	P-LCR	TROPONINCPK-MB		CHOLESTEROLHDL	ECG	ECHO	AGE
149	VITTAL	14.1	10.7	31.4	1	5	4	2	2	1	2	1	1	1	1	1	1	1	1	1	1	3	2	1	2	1	2	3	70
150	SABAWWA	10.6	9.1	18.1	2	1	4	3	1	1	2	1	1	1	1	1	1	1	2	1	1	2	1	1	2	1	3	3	68
151	YALLAPPA	11.3	9.3	20.7	1	5	4	2	2	1	3	1	1	1	1	1	1	1	1	1	1	2	1	1	2	3	2	3	60
152	BK KESNU	9.4	8.3	14.8	1	5	4	1	1	1	2	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	3	77
153	BASAVARAJ	16.5	9.9	21.2	1	5	4	2	2	1	2	1	1	1	1	1	1	1	1	1	1	2	1	1	2	3	2	1	65
154	MAHADEVI	13.5	10	27	2	5	4	2	1	1	2	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	2	2	57
155	LAXMIBAI P	12	9.9	25	2	1	4	3	1	1	3	1	1	1	1	1	1	1	2	1	1	1	2	1	2	1	2	3	58
156	BASAVARAJ	10.6	8.9	18.8	1	16	4	3	1	1	3	1	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	3	65
157	TUKARAM	15.3	8	19.2	1	5	4	1	2	1	3	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	3	60
158	LAXMIBAI	18.2	8.4	20.2	2	1	4	1	1	1	2	1	1	1	1	1	1	1	2	1	1	1	1	2	1	1	1	3	40
159	MALLAPPA	16.4	8.7	18.8	1	5	4	1	2	1	3	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	3	50
160	SHANTIBAI	11.3	9.2	20.1	2	5	4	1	1	1	2	1	1	1	1	1	1	1	2	1	1	2	1	2	1	1	1	3	53
161	SHARANABASSAPPA	8.9	8.1	13	1	16	4	1	1	1	3	1	1	1	1	1	1	1	2	1	1	1	1	2	1	1	1	3	45
162	MAHANUDA	9.7	8.1	12.3	2	5	4	1	1	1	2	1	1	1	1	1	1	1	2	1	1	1	1	2	1	1	1	3	36
163	GANESH	13.1	10.1	27.1	1	5	4	2	1	1	3	1	1	1	1	1	1	1	2	1	1	3	2	1	2	1	1	2	31
164	SIDDHU	11.2	8.3	19.9	1	16	4	1	1	1	3	1	1	1	1	1	1	1	2	1	1	1	1	2	1	1	1	3	80
165	MAHADEVI	11.7	9.8	23.7	2	5	4	2	1	1	2	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	2	3	62
166	GUNDARAO	9.3	8.2	12.6	1	4	4	1	1	1	2	1	1	1	1	1	1	1	1	1	1	1	1	2	1	3	1	3	65
167	PARIS	9.9	9	17.8	1	5	4	1	2	1	2	1	1	1	1	1	1	1	1	1	1	2	1	2	1	1	1	3	40
168	SOMAPPA	12.9	9.6	23.5	1	46	4	2	1	1	2	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	2	3	56
169	LAXMIBAI	10.3	9.2	18.4	2	1	4	1	1	1	2	1	1	1	1	1	1	2	2	1	1	2	1	2	1	3	1	3	70
170	MAHADEVI	10.1	8.3	13.5	2	1	4	1	1	1	2	1	1	1	1	1	1	1	2	1	1	1	1	2	1	1	1	3	62
171	ASOKUPPAR	13.6	9.9	27	1	6	4	3	2	1	3	1	1	1	1	1	1	1	2	1	1	2	1	1	1	1	3	3	38
172	MUTTASAB	13.5	9.9	26.1	1	46	4	2	1	1	2	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	2	3	65
173	HUSANABAI	20.8	11.4	37.6	2	1	4	3	1	1	2	1	1	1	1	1	1	1	1	1	1	4	2	1	2	1	3	3	50
174	VIJAYALAXMI	10.2	8.8	17.8	2	5	4	1	1	1	2	1	1	1	1	1	1	1	1	1	1	1	1	2	1	1	1	3	40
175	SANGAWWA	12.2	9.8	24.1	2	1	4	2	1	1	2	1	1	1	1	1	1	1	1	1	1	2	1	1	2	1	2	3	56