"THE CORRELATION BETWEEN DIFFERENT COMPLETE BLOOD COUNT PARAMETERS IN ACUTE CORONARY SYNDROMES"

By

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Dissertation submitted to the

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

DOCTOR OF MEDICINE

IN

PATHOLOGY

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ABBREVIATIONS

IHD	Ischemic Heart disease
CAD	Coronary artery disease
ACS	Acute coronary syndrome
MI	Myocardial infarction
ECG	Electrocardiograph
CK	Creatinine Kinase
SCD	Sudden Cardiac Death
CVD	Cardiovascular Disease
CHD	Coronary Heart Disease
LDL	Low-density Lipoprotein
HDL	High-density Lipoprotein
IL-1	Interleukin-1
CRP	C-reactive protein
CK-MB	Creatine Kinase - MB
C.Trop T	Cardiac Troponin T
TnT	Troponin T
TnI	Troponin I
AMI	Acute Myocardial Infarction
RBC	Red Blood cell
MCV	Mean corpuscular volume
MCHC	Mean corpuscular hemoglobin concentration
MCH	Mean corpuscular hemoglobin
RBC	Red blood cells
MPV	Mean platelet volume
PDW	Platelet distribution width
STEMI	ST-segment elevation myocardial infarction
WBC	White blood cells
PLR	Platelet to lymphocyte ratio

CBC	Complete Blood count
SPECT	single–photon emission computerized tomography scintigraphy
MPS	myocardial perfusion scintigraphy
NLR	Neutrophil lymphocyte ratio
IRA	infarct related artery
Hb	Hemoglobin
HCT	Hematocrit
ANC	Absolute Neutrophil Count
ALC	Absolute Lymphocyte Count
µm	micrometer
pg	picogram
fl	femtolitre
g/dl	gram per deciliter
SD	Standard Deviation

ABSTRACT

BACKGROUND: According to World Health Organization, coronary vascular disease represents one-third of all global deaths. In India, 1/4th of mortalities are due to cardiovascular diseases.Various cellular elements of blood have been implicated in the pathogenesis of cardiovascular disorders including atherosclerosis and its complications such as acute coronary syndrome. White blood cell count, Platelets and its indices correlates with increased risk and severity of acute coronary syndrome.

AIM: To study assess various hematological parameters like Total White Blood Cell count(WBC), Absolute Neutrophil count (ANC),Absolute Lymphocyte count (ALC), Neutrophil to Lymphocyte Ratio (NLR), Mean Corpuscular Hemoglobin Concentration (MCHC), Mean Platelet Volume (MPV), Platelet Distribution Width (PDW) in patients diagnosed with Acute Coronary Syndromes.

MATERIAL AND METHODS: A prospective hospital based study was carried out on 116 cases with acute coronary syndromes and 116 controls (age and sex matched) from 1st November, 2015 to 30th June, 2017 considering the inclusion and exclusion criteria.

RESULTS: The incidence of ACS in males (72.4%) was more in compared to females (27.6%). The most commonly affected age group was 5th decade. Another observation was made that as age increases, in females, the incidence of acute coronary syndrome also increases, possibly due to waning of hormonal effect. Total WBC count, ANC, ALC, NLR, MCHC, MPV and PDW showed significant association in ACS compared with healthy control group and in concordance with biochemical or electrocardiography results.

CONCLUSION: The study concludes that Total WBC count, ANC, ALC, NLR, MCHC and PDW are readily available in automated cell counters but underused parameters which can give clinician a helping hand in diagnosis, assessing severity and timely interventions in cases of Acute Coronary Syndrome admitted in intensive care units.

KEY WORDS: Acute Coronary Syndrome, Absolute Neutrophil Count, Absolute Lymphocyte Count, Neutrophil Lymphocyte Ratio

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INTRODUCTION

Ischemic heart disease (IHD) is a group of syndromes with similar pathophysiology resulting from imbalance between myocardial supply and demand.[1]Globally, cardiovascular diseases accounts for approximately 12 million deaths annually.[2] In India, 26% of mortality are due to cardiovascular disease, irrespective of the age and sex.[3] It presents largely into two groups: patients with chronic coronary artery disease (CAD) and patients with acute coronary syndrome (ACS).[4]

Acute coronary syndrome represents patients with ST-elevation myocardial infarction on their presenting electrocardiogram and non-ST elevation ACS. The latter includes non-ST elevation myocardial infarction and those with unstable angina.[4]

Conventional risk factors responsible for myocardial ischemia are coronary atherosclerosis, coronary emboli, myocardial vessel inflammation, or spasm.[1]

Atherosclerosis underlies the pathogenesis of coronary, cerebral and peripheral vascular diseases, and causes more morbidity and mortality (roughly 50% of all deaths) in the western world than any other disorder.[5] Clinically atherosclerosis manifest as stenosis or acute plaque rupture with thrombosis leading to compromised blood flow.[1,5] Atherosclerotic plaque formation and rupture involves inflammation at various stages. With the increasing recognition that inflammation plays a significant causal role in ischemic heart disease, assessment of systemic inflammation has become important in diagnosis in clinically limited resource setting and even in overall risk categorization.[1]

Total cell count, neutrophils, monocytes, platelet indices and various ratios have generated lot of interest in recent years among researchers involving various cardiovascular events. As this is indirectly related to inflammation leading to increase in total count, neutrophil count, platelet hypersensitivity and platelet activation, so changes in these parameters will invariably be helpful in diagnosis and risk stratification in ischemic heart disease.

The most sensitive and specific biomarkers of myocardial damage are Troponin T and I, level of both begin to rise within 2-4 hours and reaches peak value at 48 hours.[6] Creatinine kinase enzyme begins to rise within 2 to 4 hours of onset of event and peaks at about 24 hours and comes back to normal within approximately 72 hours.[1]

High blood cell counts, mainly neutrophil counts, platelet counts, their ratios and various other hematological parameters are strong and independent predictor of cardiovascular disease. These hematological parameters are easily recorded by automated cell counters which are available in most of the laboratories. So, there is a scope of further improvement of diagnosing cardiovascular events for treatment in limited resource setting, also which will lead to saving of enormous number of lives.[7]

AIM OF THE STUDY

To assess various hematological parameters like white blood cell count (WBC), Absolute Neutrophil Count (ANC), Absolute Lymphocyte Count (ALC), Neutrophil Lymphocyte ratio (NLR), Mean Platelet Volume (MPV), Platelet Distribution Width (PDW) and all Red Blood cell (RBC) parameters values in patients diagnosed with Acute Coronary Syndromes as risk predictors.

REVIEW OF LITERATURE

ISCHEMIC HEART DISEASE:

Ischemic heart disease (IHD) is an imbalance between myocardial perfusion and demand of oxygenated blood manifested as various clinico-pathological syndromes. Ischemia limits the nutrient availability, tissue oxygenation and removal of metabolic wastes. Ischemia due to obstructive atherosclerotic lesion in epicardial arteries in more than 90% of cases referred as coronary artery disease (CAD). [1]

Ischemic heart disease can manifest as:

1. Myocardial infarction (MI)
2. Angina pectoris
3. Chronic IHD with heart failure
4. Sudden cardiac death (SCD).[7]

Various clinical manifestations in IHD can partly be explained by number, structure and to degree of obstruction of plaque which holds true for acute coronary syndrome, which includes

1. Unstable angina
2. Acute myocardial infarction
3. Sudden death

These events are associated with various plaque changes which are associated with superficial erosion, ulceration, ruptureleading to formation of thrombus which occludes the artery.[1]

Granulocytes and platelets play an important role in the process of acute coronary syndrome.[4]

EPIDEMIOLOGY OF CARDIOVASCULAR DISEASE:

Cardiovascular diseases (CVDs) has caused more disability and death than any other life-threatening illness in world.[4] According to World Health Organization factsheet updated in May 2017, cardiovascular diseases are number one cause of global deaths. Around 17 million people died of CVDs in 2015, representing 31% of all global deaths.[3]Out of all these, an estimated 7.4 million were due to coronary heart disease. In India, 24% of mortality were due to cardiovascular disease.[8]

In 20th century, lifestyle across the globe is changing which iscollectively termed as epidemiological transition due to various developments in science and technology. From active lifestyle to fast foods, sedentary habits, combined with increasing tobacco use, obesity, diabetes, hypertension and dyslipidemia have all contributed to drastic increase in morbidity and mortality. In western nations, the rise occurred over several decades because of long period of epidemiological transition. In India, due to the rapid pace of economic development, epidemiological changes occurred over a much shorter time. As a consequence, cardiovascular disease (CVDs) are leading cause of death all over India. Indians are affected at least 5-6 years earlier than their western counterparts with coronary heart disease (CHD).[1,4,9,10]

In studies conducted in 1990s up to 2002 revealed coronary heart disease in urban areas to be around 6.4% and 2.5% in rural areas. In urban India, pooled estimates for females were 6.7% and for male it was 6.1% and in rural India, it stands at 2.7% for

females and 2.1% for males. According to medical certificate data, shows 25.1% of deaths are due to circulatory system involvement.[9]

NORMAL HEART:

Heart originates in embryonic form from splanchnopleuric mesoderm in the cardiogenic region.[11] Heart is a pyramidal shaped organ in the thoracic cavity with two principle coronary arteries arising from aortic sinuses. The right coronary artery with its branches supplies right atrium, right ventricle, the sino-atrial and atrio-ventricular nodes, postero-inferior one third of interventricular septum, a portion of left atrium and portion of posterior part of left ventricle. Left coronary artery with its branches like left anterior descending artery and left circumflex artery supplies left atrium, left ventricle and most of the interventricular septum.[12]

PATHOGENESIS OF ACUTE CORONARY SYNDROME (ACS)

Acute coronary syndrome can be divided among four major groups:

1. Atheromatous CHD
2. Non-atheromatous CHD
3. Hypercoagulable states
4. MI related to substance abuse.[13]

Central to understanding of myocardial ischemia is the concept of myocardial demand and supply. In normal conditions, myocardium controls the supply of oxygen rich blood to prevent ischemia and infarction. There are various determinants which dictates myocardial oxygen demand such as heart rate, myocardial contractility and myocardial wall stress.[4,14]

The normal coronary circulation is controlled by myocardial oxygen demand, which is met by ability of coronary vessels to change its resistance considerably while myocytes extract required amount of oxygen. For example, there is a changein oxygen demand of heart with emotional stress and exercise in a manner to regulate supply of oxygen and metabolites.[4]

Atherosclerosis limits the required increase in lumen of coronary arteries when demand for flow is there during excitement and exertion. When the lumen reduction is critical, myocardial perfusion is reduced to basal state.[4,10]

The main cause of acute coronary artery disease is atherosclerotic plaque formation and its complications, which acts as an inflammatory process, leading to increase in white blood cell count.[4]

An elevated white blood cell count along with red blood cells and platelets changes area well-known indicator of inflammation andplays a vital role in development of coronary heart disease.[5,9]

PATHOGENESIS:

Atherosclerosis is viewed as a chronic inflammatory and healing process of arterial wall due to endothelial injury. Progression of the lesion occurs through interactions between endothelial cells and smooth muscle cells of arterial walls with lipoproteins, monocyte-derived macrophages, and T-lymphocytes. Atherosclerosis progresses in sequential manner like:

- Endothelial injury and dysfunction
- Lipoproteins accumulation

- Monocyte adhesion to the endothelium
- Platelet adhesion to endothelium
- Smooth muscle cell proliferation, extracellular matrix production, and recruitment of the cells.
- Accumulation of lipids

Endothelial Injury: It is the cornerstone of response-to-injury hypothesis. Any kind of injury leading to endothelial loss which can be due to mechanical denudation, immune complex deposition, hemodynamic forces, chemicals or irradiation will further causeintimal thickening. Most of early human lesions begin at sites of morphologically intact endothelium. Thus, dysfunctional intact endothelial cells exhibit altered gene expression,enhanced leucocyte adhesion and increased endothelial permeability.[5,15]

Inflammatory cytokines released due to various etiological culprits can also stimulate pro-atherogenic gene expression. However, hemodynamic disturbance and hypercholesterolemia are two most important causes of endothelial dysfunction.[5]

Hemodynamic disturbance: Importance of hemodynamic flow can be observed by the fact that plaques occur at ostia of exiting vessels, branching points and along posterior wall of abdominal aorta. Some in vitro studies have shown that non-turbulent laminar flow will lead to induction of endothelial genes which protects against atherosclerosis.[5,14]

Lipids:

Lipids form lipoprotein complexes with specific apolipoproteins for transport in blood stream. Dyslipoproteinemias includes:[10,13]

1. Increased low-density lipoprotein (LDL) cholesterol levels.
2. Decreased high-density lipoprotein (HDL) cholesterol levels.
3. Increased levels of lipoprotein (a)

Evidence connecting hypercholesterolemia to atherogenesis include:

1. Cholesterol and cholesterol-esters are dominant lipids in atheromatous plaque.
2. Various genetic factors are associated with accelerated atherosclerosis which are involved in lipoprotein uptake and metabolism like defective LDL receptors and inadequate hepatic LDL uptake in familial hypercholesterolemia.
3. Other genetic and acquired disorders lead to premature atherosclerosis like diabetes and hypothyroidism.
4. Various epidemiological studies also demonstrated relationship between levels of total plasma cholesterol and severity of atherosclerosis.
5. Lowering serum cholesterol will lead to decrease in rate of progression of atheromatous plaque and reducing the risk of cardiovascular events.[5,10]

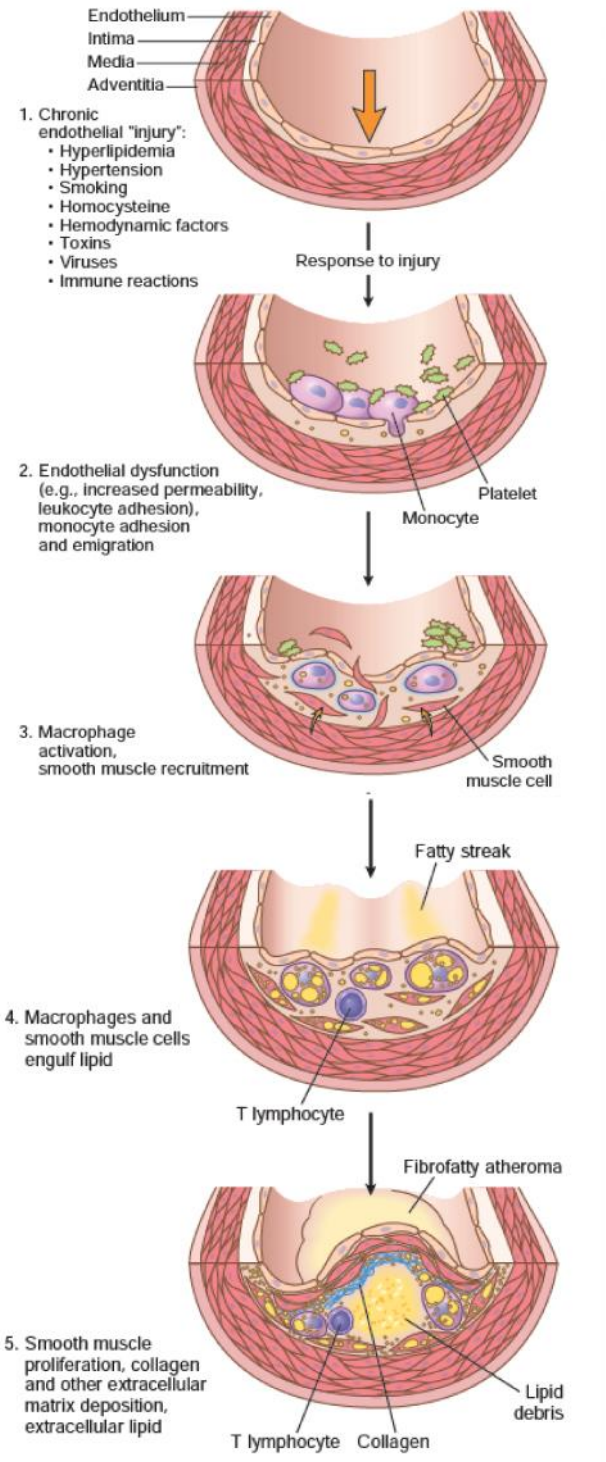


Figure 1: (Adopted from Robbins and Cortan 2014) Hypothesis depicting the evolution of arterial wall changes in response to injury

Mechanism by which hyperlipidemia is contributing to atherogenesis:

- Chronic hyperlipidemia, directly impairs endothelial dysfunction, membrane and mitochondrial dysfunction by increasing reactive oxygen species production leading to nitric oxide decay and further causing damping of vasodilator activity.[5,10]
- Chronic hyperlipidemia, will lead to lipoprotein accumulation in intimal layer where they form aggregates or oxidized by free radicals produced by inflammatory cells. These LDLs get accumulated in macrophages leading to formation of foam cells, even smooth muscle cells get transformed into lipid-laden foam cells by ingesting modified lipids. These modified lipoproteins are not only toxic to endothelial cells, smooth muscle cells and macrophages but also causes release of growth factors, chemokines and cytokines.[5,15]

Inflammation:

Chronic inflammation will cause initiation and progression of atherosclerotic lesion. Inflammation is triggered by accumulation of free fatty acid in macrophages and cholesterol crystals leading to stimulation of cytosolic innate immune receptors of inflammasome. This activation leads to production of pro-inflammatory cytokines like Interleukin-1 (IL-1), which causes recruitment of various leucocytes, including monocytes. T lymphocytes along with macrophages will stimulate local production of cytokines and chemokines which will lead to more recruitment and activation of inflammatory cells.Activated macrophage produces reactive oxygen species which further causes enhancement of LDL oxidation and growth factors which lead to smooth muscle proliferation. These leucocytes and vascular endothelial cells will release more

growth factors which further promotes proliferation of smooth muscles cells and synthesis of extracellular matrix proteins.[16,17]

Smooth muscle proliferation and matrix synthesis:

Intimal smooth muscle cell proliferation and extracellular matrix deposition converts fatty streak into mature atheroma leading to growth of atherosclerotic lesions. Several growth factors are involved in smooth muscle cell proliferation like platelet-derived growth factor which is released by macrophages, endothelial cells and smooth muscle cells, fibroblast growth factor and transforming growth factor-α. These factors stimulate smooth muscle cells to synthesize extracellular matrix which leads to atherosclerotic plaque stabilization. In contrast, there is increase in breakdown of extracellular matrix components due to activated inflammatory cells leading to unstable plaque.[5,15-17]

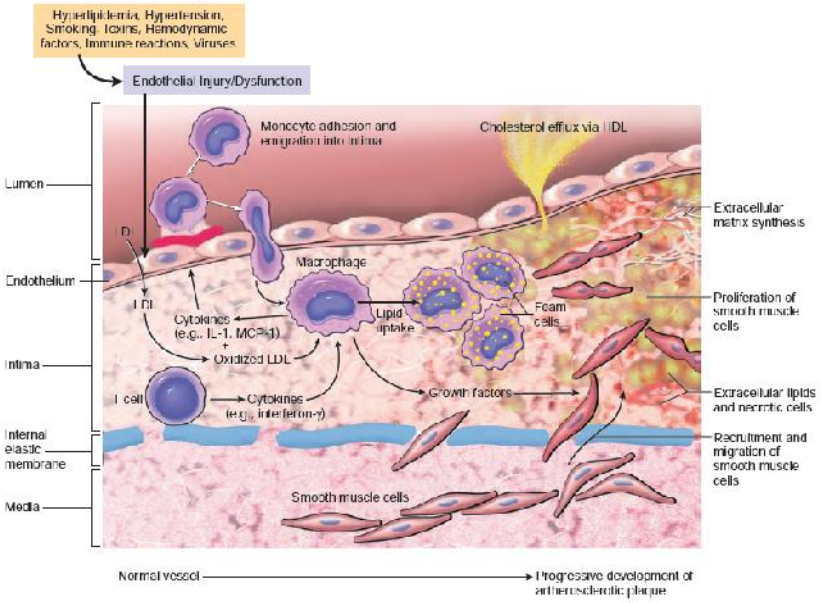


Figure 2: (Adopted from Robbins and Cortan 2014) Cellular interactions sequence in atherosclerosis.

RISK FACTORS FOR CORONARY HEART DISEASES:

WHO has declared coronary heart disease as our modern “epidemic”. The etiology of CHD is multifactorial. Majority of these risk factors are divided as modifiable and non-modifiable. Presence of even one of these will place an individual in a high risk categories.[1,9]

Table 1:RISK FACTORSFOR CORONARY HEART DISEASES:[1,9]

NON MODIFIABLE	MODIFIABLE
Age	Smoking
Gender	Hypertension
Family history	Hyperlipidemia
Genetic factors	Diabetes
Personality	Obesity
	Sedentary lifestyle
	Stress

NON-MODIFIABLE:

Age: Although development of plaque is a progressive process, it usually doesn’t manifest until it has reached a critical threshold in middle age. This explains the reason for five-fold increase in incidence of myocardial infarction between the age of 40-60 years.[1,10]

Gender: Premenopausal women are relatively less prone to atherosclerosis than age-matched men unless they are predisposed to diabetes, hyperlipidemia or severe hypertension. Clinical trials of estrogen replacement have not shown any significant effect rather some have shown to actually increase cardiovascular effect.[1,10]

Genetics: Family history shows a strong association as a risk factor for atherosclerosis. There are certain Mendelian disorders which have strong association with atherosclerosis but they account for very small number.[1,10]

Personality: Type A personality behavior people who are restless and have a sense of urgency and impatience are associated with CHD more than type B personality people who are calmer and philosophical.[9]

MODIFIABLE RISK FACTORS:

Smoking: It is a well-established and a major risk factor for CHD. Risk for developing CHD is directly related to number of cigarettes smoked per day and is not only a independent factor but also has a synergistic effect with other risk factors like hypertension and elevated serum cholesterol. Even one year of cessation of smoking will lead to decline in risk of CHD quite substantially. [1,9,10]

Hypertension: This has been identified as single most useful risk factor parameter for detection of CHD in an individual. Both systolic and diastolic pressures are generally accepted.[1,10]

Hyperlipidemia: Specifically, hypercholesterolemia even in the absence of other risk factors have been identified as risk factor which can initiate atherosclerotic process which can lead to CHD. Low-density lipoprotein in contrast to high density lipoprotein, which

is one of the major component of cholesterol is counted as major risk factor for atherosclerosis leading to CHD.[1,10]

Diabetes: People with diabetes have 2-3 times higher risk of CHD than individual without diabetes. CHD is responsible for 30-40% deaths of diabetics with age over 40 years.[1.9]

Physical activity: People with sedentary lifestyle are at greater risk of developing early CHD as there are numerous studies with evidence that regular physical exercise will lead to increase in high-density lipoproteins.[1,9]

Alcohol: High intake of alcohol, which is defined as 75gm or more per day is an independent risk factor for CHD and hypertension. [1,9,10]

ADDITIONAL RISK FACTORS:

Inflammation:

Inflammation is closely linked to all stages of atherogenesis and involved in plaque formation to rupture. Over the years, many markers of inflammation, were used to correlate ischemic heart disease but *C-reactive protein* (CRP) has emerged as simplest and most sensitive of all. It is a well-known that plasma CRP is a strong, independent marker for risk of myocardial infarction, stroke, peripheral arterial disease and sudden cardiac death. CRP levels have been incorporated even in risk stratification algorithms.[1,17]

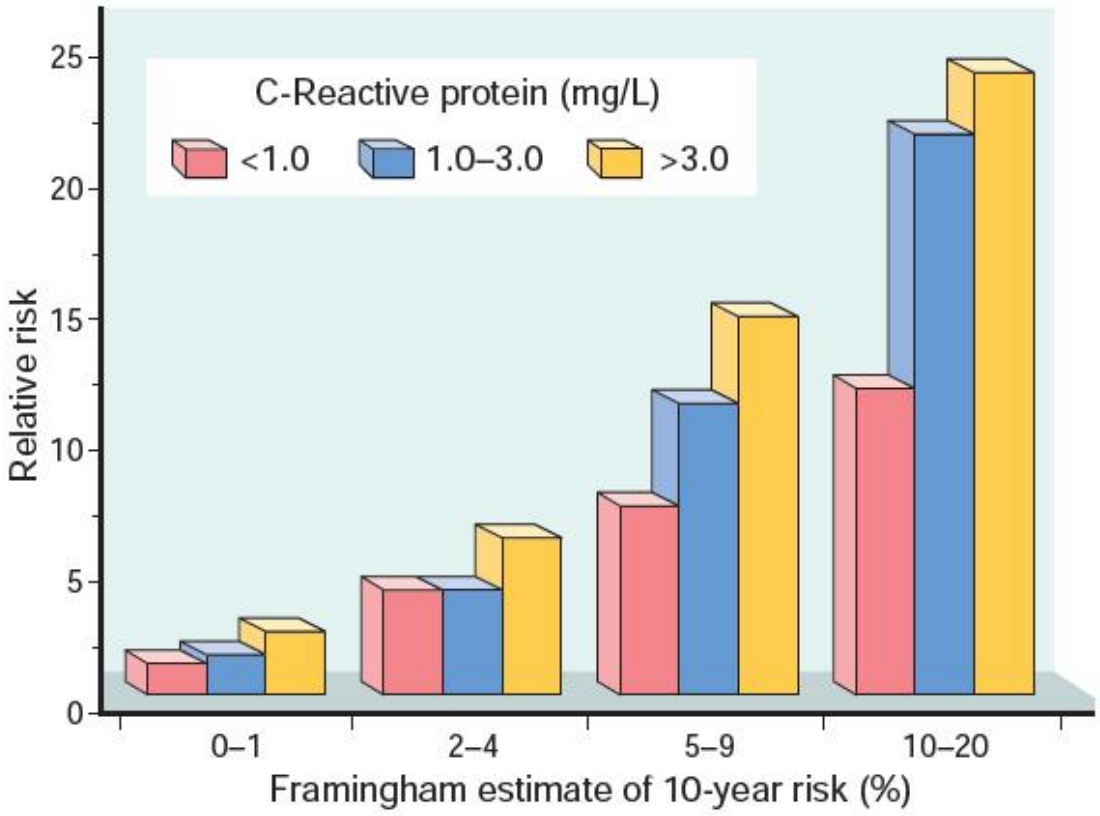


Figure 3: (Adopted from Robbins and Cortan 2014) C-reactive protein (CRP) predicts cardiovascular risk.

Hyperhomocystinemia: Serum homocysteine levels have been associated with peripheral vascular diseases, stroke, and coronary atherosclerosis and even with venous thrombosis. Although low folate and vitamin B12 levels can increase homocysteine levels, supplement vitamins would not have any effect on incidence of cardiovascular disease. Homocystinuria, a rare inborn error of metabolism, which results in elevated circulating homocysteine (>100 μmol/L) is associated with premature vascular disease.[1,5,10]

Lipoprotein a [Lp(a)]: altered form of LDL having apolipoprotein B-100 portion of LDL which is linked to apolipoprotein A (apo A). It is independent of total cholesterol or LDL levels and is related to coronary and cerebrovascular disease.[1,5]

Thrombotic and fibrinolytic factors: Several factors like plasminogen activator inhibitors 1, which is a thrombotic factor are predictors of major atherosclerotic events, leading to stroke and myocardial infarction. Thrombin, a platelet derived factor which acts as both procoagulant and proinflammatory is increasingly recognized as major contributors to local vascular pathology.[1,5,10]

BIOMARKERS OF CORONARY ARTERY DISEASES:

The diagnostic approach to acute coronary syndrome was one of the most challenging for emergency physicians. Markers for myocardial necrosis were evolved during 1980s and 1990s like lactate dehydrogenase isoenzymes and more cardiac-specific enzymes (CK-MB) became available. Subsequently, many new markers have become available like creatine kinase-MB isoforms, myoglobin, and cTnT and cTnI on commercial automated instruments. Although there are many biomarkers currently being used but none of the markers meet the features of 'Ideal" blood based biomarker. Currently CK-MB, Troponin T, Troponin I, Myoglobin are included in testing panel and few markers are under assessment for potential clinical use like high-sensitive CRP, B-type (formerly known as brain) natriuretic peptide.[18]

Current guidelines by European Society of Cardiology/American College of Cardiology of Acute, Evolving, or Recent Myocardial Infarction:

1. Typical rise and gradual fall (Troponin) or more rapid rise and fall (CK–MB) of biochemical markers with at least one of the following:

- Ischemic symptoms
- Development of pathologic Q waves on electrocardiogram
- Electrocardiographic changes indicative of ischemia (ST-segment elevation or depression)
- Coronary artery intervention (e.g. coronary angioplasty)

2. Pathologic (morphologic) findings of an acute myocardial infarction.[18,19]

Creatine Kinase and CK-MB:

Creatine kinase is present in many tissues, including myocardium and skeletal muscles. Creatine kinase has 3 isoenzymes: MM, MB, and BB. MM isoform is present in higher concentration in skeletal muscles, with MB isoform is in higher concentration in myocardium. CK-MB has been reported in muscle tissue of trained athletes, patients of renal failure and chronic myopathic skeletal muscle injury. Until last decade CK-MB levels were thought to be gold standard test for acute myocardial infarction. Following myocardial injury, initially CK-MB levels start to rise at 4-9 hours after onset of chest pain and reaches peak levels at 24 hours and returning to baseline within 48-72 hours. The biggest advantage for CK-MB remains till date is that it remains elevated for longer periods of time and easier to detect in serial measurement and however, CK-MB levels also rises with myocarditis, cardiac transplant rejection and cardiac surgery.[6,18]

Troponin T and Troponin I:

It is a regulatory complex of three protein subunits which are located on thin filament of myocardial contractile apparatus. Three subunits are, troponin C (calcium binding component), TnI (tropomyosin binding component) and TnT (inhibitory component). Skeletal and cardiac isoforms of both TnI and TnT varies in amino acid sequence to permit development of monoclonal antibody-based immunoassay. Both isoforms are stored in 2-compartment in myocytes, majority in sarcomere and minor part in cytosolic pool. Both of their serum levels increase within 4-9 hours, peaks at 12-24 hours after acute myocardial infarction and remains elevated up to 14 days. Significant elevation of their levels most likely reflects myocardial necrosis. Most authors have billed troponins as absolute cardiac specific assay.[6,18,19]

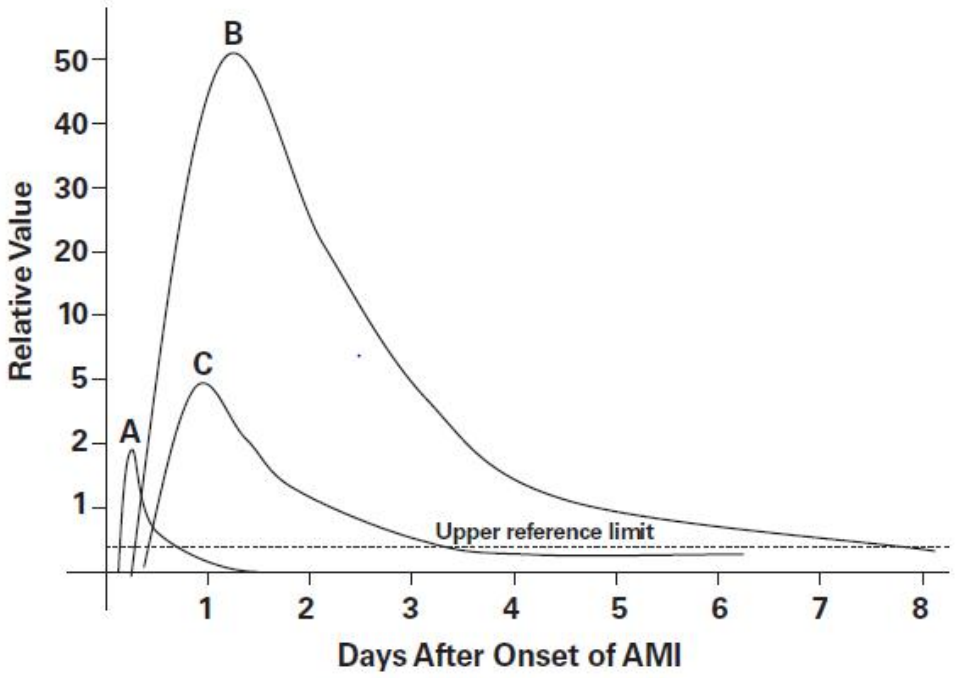


Figure 4: [adopted from (Libby P, 2002)] Timing of release of cardiac markers. A, myoglobin; B, troponin; C, creatine kinase–MB. AMI, acute myocardial infarction.

COMPLETE BLOOD CELL COUNT: GENERAL OVERVIEW

Physiological process of blood cell formation is known as hematopoiesis. It starts in 3rd week of life in yolk sac, as aggregates of blood islands. At around 4 months of fetal life, bone marrow starts producing blood cells.[20]

All the blood cells in peripheral blood circulation are derived from primitive mesenchymal cells known as pluripotent stem cell. They have an ability of self-renewal and differentiation. These cells are morphologically similar to lymphocytes. These cells can also differentiate along different pathways and can produce other tissue cells. As a part of proliferation, cells become committed to a lineage and are known as progenitor cells. These cells have restrictive multilineage potential.[21]

Two multilineage cell lines originating are: lymphoid and myeloid. All blood cells belong to either one lymphoid or myeloid linage of these two lineages. Myeloid lineage include erythrocytes, platelets, neutrophils, eosinophils, basophils and monocytes. Lymphoid lineage will give rise to B and T- lymphocytes.[21]

A mature erythrocyte is biconcave, non-nucleated disk of 7-8 μm. Its main function is to transport oxygen and carbon dioxide. The life span of red cell is around 120days, after which it is ingested and degraded by mononuclear phagocytes.[20]

Other myeloid lineage cells like neutrophils are 14-15 μm in size with 2-5 lobes joined by chromatin strands and having numerous specific granules in the cytoplasm. Main function of neutrophils is in acute inflammation.[20]

Eosinophils are similar to neutrophils with slightly larger size around 15-16 μm, usually bilobed and have numerous orange-red granules. These granules contain major basic protein that is toxic to many parasites.[20]

Basophils are also similar to neutrophils in maturation stages and are 9-12 μm in size with cytoplasm filled with coarse deep purple-black granules which even obscure the bi-trilobed nucleus. Its granules contain histamine and heparin which play a vital role in allergic and anaphylactic reactions.[20]

Monocytes are largest of all white blood cells on peripheral smear with 15-20 μm in size having irregular shape with oval or kidney shaped nucleus and fine reticular chromatin. Cytoplasm is blue-gray in color with ground glass appearance containing fine azurophilic granules and vacuoles. Their main function is phagocytosis and also act as antigen presenting cell along with production of variety of cytokines.[20,21]

Lymphocytes are mainly of three types: B-lymphocytes, T-lymphocytes and natural killer-cells.

B-lymphocytes are about 10-20% of lymphocytes in peripheral blood. They are located in superficial cortex, germinal centers, mantle zone of lymph node, follicles of spleen and in other sites too. T-lymphocytes are 60-70% of the population of circulating lymphocytes. They are located in thymus, paracortical region of lymph node and periarteriolar lymphoid sheaths in spleen. They are mainly responsible for cell mediated immunity. Natural killer cells comprise of around 10-15% of lymphocyte population in peripheral blood.[20]

Platelets are anucleated fragments with diameter of 1-4µm which are released by bone marrow megakaryocytes into the circulation. Theseare thought to be responsible for maintaining vascular integrity and homeostasis. Megakaryocyte surface membrane form proto-platelet extensions, from here platelets “bud-off” and are released in to circulation. At any giventime approximately 200,000 to 400,000 platelets/µl remain in peripheral blood.[20,21]

RED BLOOD CELL INDICES:

There are various red cell indices which measure the size and hemoglobin content of Red Blood Cells (RBC) which includes Mean Corpuscular Volume (MCV), Mean Corpuscular Hemoglobin Concentration (MCHC) and Mean Corpuscular Hemoglobin (MCH).[22]

Mean Corpuscular Volume (MCV): This is the expression of volume occupied by a single erythrocyte. It is measured in cubic micrometers. Normal value for an adult is 82-98cumm.[20,22]

Mean Corpuscular Hemoglobin Concentration (MCHC): It is an average concentration of hemoglobin in the RBCs. Normal value is between 32-36g/dl.[20]

Mean Corpuscular Hemoglobin (MCH): It is a measurement of average of weight of RBC of hemoglobin in RBC. Its normal value range from 26-34pg/cell.[20]

PLATELET INDICES:

There are various platelet indices which are used to measure the size and shape of an average platelet such as mean platelet volume (MPV), platelet distribution width (PDW), which provide important information.

Mean platelet volume (MPV): It is a mathematical average of size of platelets which are present in blood at a particular time. Normal value is between 9.7-12.8fl.

Platelet distribution width (PDW): It is a variation in size of platelets. It depends on how recently platelet has released from marrow. Activated platelets and patients with thrombocytopenia usually have large platelets.[20,21]

AUTOMATION IN HEMATOLOGY:

Variety of fully automated cell counters are now a days commercially available. The instruments are based on various technologies used in different ways:

1. Impedance measurement,
2. High frequency measurement,
3. Forward scatter at different angles,
4. Fluorescence flow-through cytometry.

The measurement technology of the systems of the Sysmex X-Class is based on fluorescence flow-through cytometry, optimized for specific requirements. Depending on the instrument specifications of the X-Class. This method is used for the analysis of White blood cells (WBC), reticulocytes, platelets and for the determination of exact erythroblast count.[23]

EVALUATION OF VARIOUS PARAMETERS IN ACUTE CORONARY SYNDROME

Naz, Ali and Akhtar[24] conducted a study in the department of Physiology, Lahore Medical and Dental College to evaluate the Neutrophil Lymphocyte Ratio (NLR) in diagnosis of coronary artery disease. The study was conducted on 40 cases with coronary artery disease and 20 normal healthy adult males with age group 35-55 years. All the participants were non-smokers and non-diabetic. Total and differential leukocyte count was checked by automated hem-analyzer and NLR was also calculated and data was statistically analyzed having highly significant p-value <0.001 in participants with coronary artery disease.

Nunez *et al*[25]conducted a study on 515 consecutive patients admitted with ST segment elevation myocardial infarction (STEMI). White blood cells and differential count were measured at the time of admission and daily for first 96 hours afterward. Patients with cancer, inflammatory diseases or premature death were excluded and a total of 470 patients were included in final analysis. Mean age group in their sample was 65 ± 13 years. The association between maximum value of NLR and maximum value of WBC with mortality was assessed by Cox regression analysis. They concluded Neutrophil Lymphocyte ratio is a useful marker to predict long term mortality in patients with ST segment elevation Myocardial infarction, with a discriminative ability than total WBC maximum count.

A study done by Yüksel *et al*[26]on 388 patients who underwent coronary angiography. According to Gensini score patients were categorized as mild and severe atherosclerosis. Control group had 80 patients with normal coronary arteries. Platelet to

lymphocyte ratio (PLR) of three groups was compared. They came out with the results that mean PLR of severe atherosclerosis group was higher than that of control group and mild atherosclerosis. PLR also showed significant correlation with Gensini score. They concluded that high PLR appears to be additive to conventional risk factors and commonly used biomarkers in predicting severe atherosclerosis

According to study done by Ozdemir *et al*[27]in 262 patients, out of which 113 patients with normal myocardial perfusion and 149 with myocardial infarction were selected. These patients were undergoing myocardial perfusion gated single–photon emission computerized tomography scintigraphy (SPECT) and also had CBC within 90 days of myocardial perfusion scintigraphy (MPS). In accordance with MPS results, patients were divided into two groups. Group 1: patients who had ischemia or infarction or both with positive MPS results and Group2: summed difference score, stress summed score, stress thickening score were compared with group 1 and significant values were found in group 1. They concluded that there is significant change in Neutrophil count and NLR but no significant change seen in platelet, lymphocyte and PDW values.

In continuation of his previous study with a wider sample size of 1030 consecutive patients studied by Nunez *et al*[28], who were admitted with activated troponin in emergency department were selected. CBC, Lymphocytes, Monocyte and Neutrophil were calculated by using an automated blood cell counter and all these measurements were within first 24 hrs of onset of symptoms. After a follow up of 36 months, out of 1030 patients 139 patients achieved complete end point of death or MI. They reported that lymphopenia is associated with an increased risk for developing the combined end

point of death or MI in patients with acute chest pain without enzymatic and electrocardiographic changes.

Brown, Giles and Croft[29] conducted a study to find out association between WBC count and CHD mortality with Cox regression analyses of data from 8914 adults of age group 30-75. Covariates included were age, sex, race, education, physical activity, smoking status, hypertensive status, total serum cholesterol, body mass index, hematocrit, and history of cardiovascular disease, stroke and diabetes. During the follow up period of 17 years, there were 548 deaths from CHD and 782 deaths from various diseases of heart. Mean WBC count was greater among persons who died from CHD. These results pointed that high WBC counts are a predictor of CHD mortality independent of effects of smoking and other CVD risk factors.

Wheeler *et al*[30], did a meta-analysis of seven long-term studies involving a total of 30,374 participants and indicated that Neutrophil count may be a stronger predictor of CAD risk than any other leukocyte components.

A prospective study was done by Selcuk *et al*[31] in 107 patients who underwent diagnostic coronary angiography. According to presence or absence of cardiovascular disease, these patients were categorized accordingly into two groups. Out of 107 patients 65 were diagnosed as having CAD. After correlation analysis, they found that NLR and lymphocyte count had a significant correlation with presence or absence of cardiovascular disease. After multivariate analysis, they suggested that Neutrophils are the prominent subtype of the leukocytes involved in acute coronary event.

Peter M. Sweetnam *et al*[32] conducted a study to find out the role of the individual type of leukocyte in prediction of ischemic heart disease. They recruited 4860 men aged 45-63 years between 1979 and 1983 in South Wales and the west of England, respectively. At the 10-year follow-up, the total leukocyte count predicted ischemic heart disease events after adjusting for classical risk factors, including smoking. Five year follow-up results were available for differential white cell counts. The main contributor to the increase in total count in the men who developed disease were the Neutrophil count along with significant increase in the eosinophil counts.

Munir and Afzal[33]conducted a study to assess the predictive ability of differential leukocyte subtypes in patients with acute coronary syndrome. The study was conducted in 69 healthy subjects (mean age 59.7± 14.3 years) and 133 patients of acute coronary syndrome (mean age 60.9± 11.9 years) and were followed up for one year. All patients were evaluated by taking detailed history and physical examination. The variables included in study were age, sex, diabetes mellitus, systolic and diastolic hypertension, hyperlipidemia, smoking, family history of ischemic heart disease, cardiac biomarkers, C- reactive protein, total and differential leukocyte counts. Diagnosis was based on clinical characteristics and laboratory data. The predictive ability for death by total count of leukocytes, Neutrophil, Lymphocytes and Monocyte was assessed using Cox regression analysis. They concluded that total and differential leukocyte counts are independent markers for the prediction of risk in patients with acute coronary syndrome.

Erkol *et al*[34]conducted a retrospective study on 1625 patients with acute ST-Segment elevation who underwent primary percutaneous coronary intervention <12 hour after onset of symptoms. The aim of study was to investigate whether the hemographic

parameters on admission are associated with spontaneous infarct related artery (IRA) patency. Angiography showed patent IRA in 160 patients. Neutrophil count on admission was significantly lower and lymphocyte count on admission was significantly higher in patent IRA group. Neutrophil lymphocyte ratio was found to be individual predictor of occluded IRA on initial angiography with a sensitivity of 62.75% and a specificity of 70%. They concluded that NLR on admission is significantly related to angiographic thrombus burden and spontaneous early IRA patency in patients with acute STEMI.

A study was done by Bajari and Tak[35]to find relationship between Neutrophil-Lymphocyte ratio at admission and patient outcome over a period of six months in subjects with acute coronary syndrome (ACS). The study was conducted in 435 consecutive patients presenting with acute coronary syndrome. 35 patients dropped out and only 400 completed the study. Patients were categorized into two groups according to cut off value (5.25) of NLR: NLR group 1(NLR ≤ 5.25) had 265 patients and NLR group 2 (NLR > 5.25) included 135 patients. During the study 47 patients died within 6 months of follow up. Higher mortality was seen in NLR group 2 as compared to NLR group 1 with p value <0.001. The study concluded that elevated NLR is independently associated with higher all –cause mortality rate up to 6 months period irrespective of ACS type.

A prospective cohort study was carried out by Adam *et al*[36]at cardiology department of civil hospital of Karachi. 297 patients with acute coronary syndrome were recruited in the study. The relationship of baseline White blood cell (WBC) to mean platelet volume(MPV) ratio with major adverse cardiac event and mortality was assessed during a 30 day follow up. The patients were divided into two groups: group A (ratio<1000) and group B (ratio>1000). WBC count, Platelet count, Platelet to

Lymphocyte ratio and Neutrophil to Lymphocyte ratio were significantly higher in MACE- positive group as compared to major adverse cardiac event- negative. WBC count (p=0.02), Neutrophil to lymphocyte ratio (p=0.03) were significantly higher in major adverse cardiac event- positive group as compared to major adverse cardiac event - negative group symptoms. The aim of the study was to investigate whether the hematological parameters on admission were associated with spontaneous infarct related artery (IRA) patency. Angiography showed patent IRA in 160 patients. Neutrophil count on admission was significantly lower and lymphocyte count on admission was significantly higher in patent IRA group. Neutrophil to Lymphocyte ratio (NLR) was significantly lower in the patent IRA group. Neutrophil lymphocyte ratio was found to be individual predictor of occluded IRA on initial angiography with a sensitivity of 62.75% and a specificity of 70%. They concluded that NLR on admission is significantly related to angiographic thrombus burden and spontaneous early IRA patency in patients with acute STEMI.

MATERIALS AND METHODS

Source of data:

A prospective hospital based study was carried on patients admitted in BLDEU'S Shri B.M. Patil Medical College, Hospital and Research Centre, Vijayapur considering the inclusion and exclusion criteria during 1st November,2015 to 30th June, 2017.

All the patients with acute coronary syndromes were included in the study and compared with normal healthy controls (age and sex matched) having a normal electrocardiogram and no past history of ischemic heart disease.

Methods of collection of data:

- The study was carried out on patients presenting with acute coronary syndrome within 24 hours.
- All patients were interviewed as per Performa prepared and then complete clinical examination will be done.
- The blood samples were collected from antecubital vein using aseptic precautions and analyzed by standard procedure using 6 part differentiated automated Hematology analyzer (Sysmex XN1000) (Figure No 5).
- Peripheral smear of all cases were studied (Figure No 6).
- Relevant investigations like electrocardiogram and cardiac enzymes were analyzed.

Inclusion criteria:

Patients diagnosed with

a. Myocardial infarction

b. Angina (Unstable Angina)

Exclusion criteria:

Patients of coronary artery disease with hematological malignancies, systemic inflammatory disease and infectious causes, will be excluded from study.

STATISTICAL ANALYSIS:

All characteristics were summarized descriptively. For continuous variables, the summary statistics of mean, standard deviation (SD) were used. For categorical data, the number and percentage were used in the data summaries. Chi-square ($\chi 2$)/ Freeman-Halton Fisher exact test was employed to determine the significance of differences between groups for categorical data. The difference of the means of analysis variables between two independent groups was tested by unpaired t test. If the p-value was < 0.05, then the results were considered to be statistically significant otherwise it was considered as not statistically significant. Data were analyzed using SPSS software v.23.0. and Microsoft office.

Figure No 5 : Sysmex XN 2100 Automated Hematology Analyzer

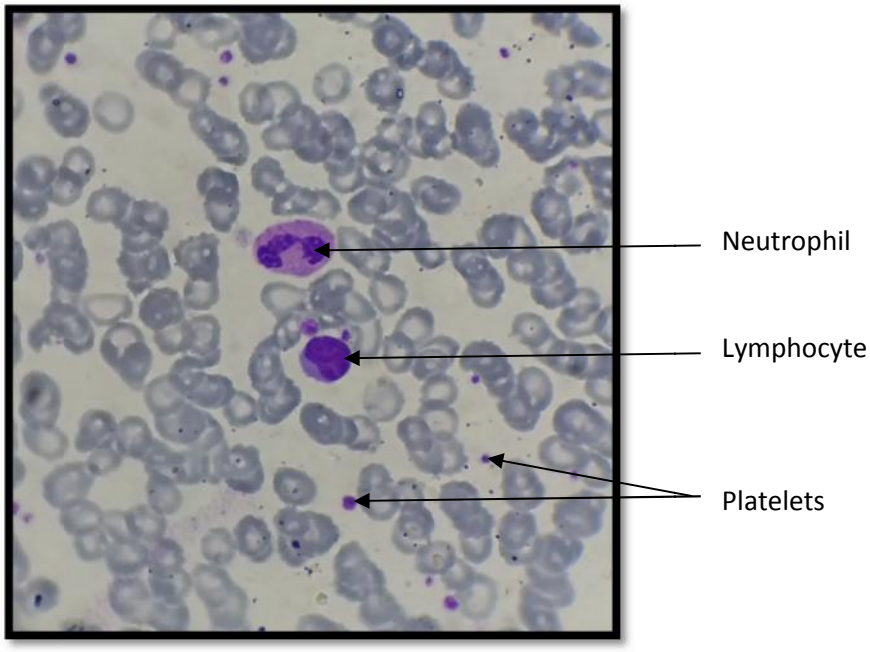


Figure No 6 : Microphotographs Peripheral Smear (1000 X)

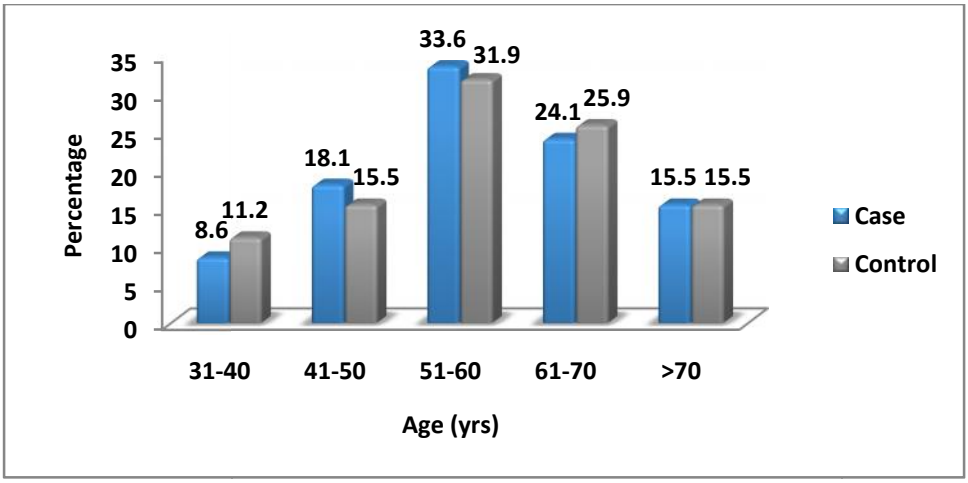
RESULTS

Total of 232 subjects were included in the study, out of which 116 were cases (study group) and 116 were controls (control group). Out of 116 cases which were taken up for study and were matched with control group for age and gender. Maximum number of patients (39 cases, 33.6%) were seen in 5th decade of life. Out of total 116 cases, 88 cases (75.8%) were in 5th, 6th and 7th decade of life (table 2). 18 cases (15.5% of cases) were in age group above 70 years and 10 cases (least of all) (8.6%) were there in 3rd decade of life (figure 7).

Table 2: Age Distribution by study groups

Age(yrs)	Case		Control	
	N	Percent	N	Percent
31-40	10	8.6	13	11.2
41-50	21	18.1	18	15.5
51-60	39	33.6	37	31.9
61-70	28	24.1	30	25.9
>70	18	15.5	18	15.5
Total	116	100	116	100

Figure 7: Bar Chart of Age Distribution by study groups



While comparing, male to females in both the groups, out of 116 cases 84 cases i.e. 72.4% were male and 32 cases i.e. 27.6% were females in both study group and control group (table 3, Figure 8). Male: Female ratio was calculated to be 2.6:1 (table 4).

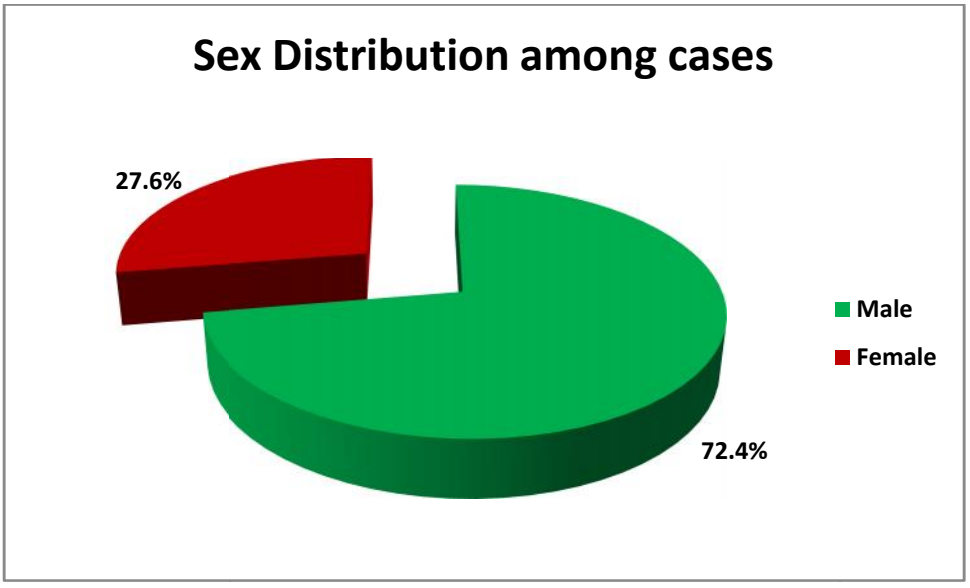
Table 3: Sex Distribution by study groups

Sex	Case		Control	
	N	Percent	N	Percent
Male	84	72.4	84	72.4
Female	32	27.6	32	27.6

Table 4: Sex ratio

M/F ratio	2.6:1

Figure 8: Sex Distribution among cases

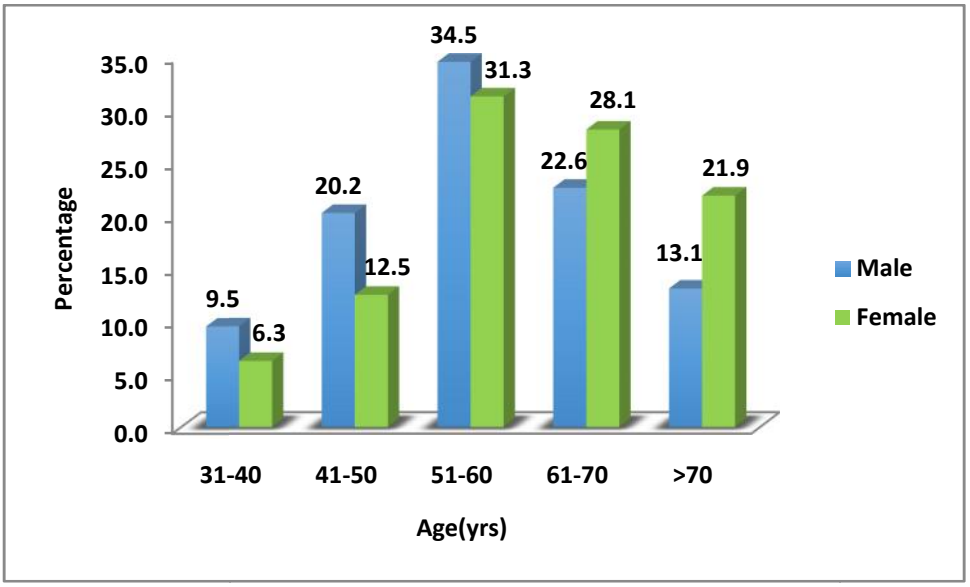


In the present study group, there were 84 males and 32 female subjects. Among those age distributions between male and females were calculated and shows as age increases male to female ratio is decreasing. In younger age group (3rd decade) it is 4:1 but as age increases ratio reduces from 2.90:1 to 2.11:1 in 5th decade and 6th decade, respectively (table 5, Figure 9).

Table 5: Age Distribution by Sex among cases

Age(yrs)	Male		Female		M:F ratio	p value
	N	%	N	%		
31-40	8	9.5	2	6.3	4.0	0.632
41-50	17	20.2	4	12.5	4.25	
51-60	29	34.5	10	31.3	2.90	
61-70	19	22.6	9	28.1	2.11	
>70	11	13.1	7	21.9	1.57	
Total	84	100.0	32	100.0	2.6	

Figure 9: Bar Chart of Age Distribution by Sex among cases (% of distribution)



In the present study, inclusion criteria of acute coronary syndrome was to include diagnosis based on clinical symptoms and electrocardiogram with either or both of biochemical changes like CK-MB and troponin T positivity. Tables indicates, out of 116 cases, 48 showed CK-MB positivity and 65 cases showed troponin T positivity and 35 cases showed both CK-MB positivity and troponin T positivity (table 6).

Table 6: Comparison of Age groups among study groups with biochemical test and ECG study.

Age(yrs)	CK-MB Positive		C.TropPositive		CK-MB &C.Trop both Positive		ECG Positive	
	N	%	N	%	N	%	N	%
31-40	3	6.3	4	6.2	1	2.9	10	8.6
41-50	8	16.7	11	16.9	7	20.0	21	18.1
51-60	16	33.3	22	33.8	11	31.4	39	33.6
61-70	15	31.3	17	26.2	11	31.4	28	24.1
>70	6	12.5	11	16.9	5	14.3	18	15.5
Total	48	100.0	65	100.0	35	100.0	116	100

Note: following results were of the patients who underwent biochemical test and ECG study.

HEMATOLOGICAL PARAMETERS:

The total White Blood Cell Count (WBC), Hemoglobin (Hb), Hematocrit (HCT), Mean Corpuscular Hemoglobin (MCH), Mean Corpuscular Hemoglobin Concentration (MCHC), Platelet Distribution Width (PDW), Mean Platelet Volume (MPV), Neutrophil to Lymphocyte Ratio (NLR) were the various parameters which were evaluated in thepresent study.

WBC PARAMETERS

The mean value for total WBC count ($x10^3$ per µl) in study group was 12.5±3.9 and in control group was 7.5±1.4 with p-value <0.001 at 5% level of significance.

Mean values for Absolute Neutrophil Count (ANC) ($x10^3$ per µl) was 10.2±4.0 in study group and 4.7±1.2 in control group. Mean values for Absolute Lymphocyte Count (ALC) ($x10^3$ per µl) was 1.9±0.9 and 2.4±0.6 in study and control group respectively, giving statistically significant p-value <0.001 in both ANC and ALC (table 7,figure 10).

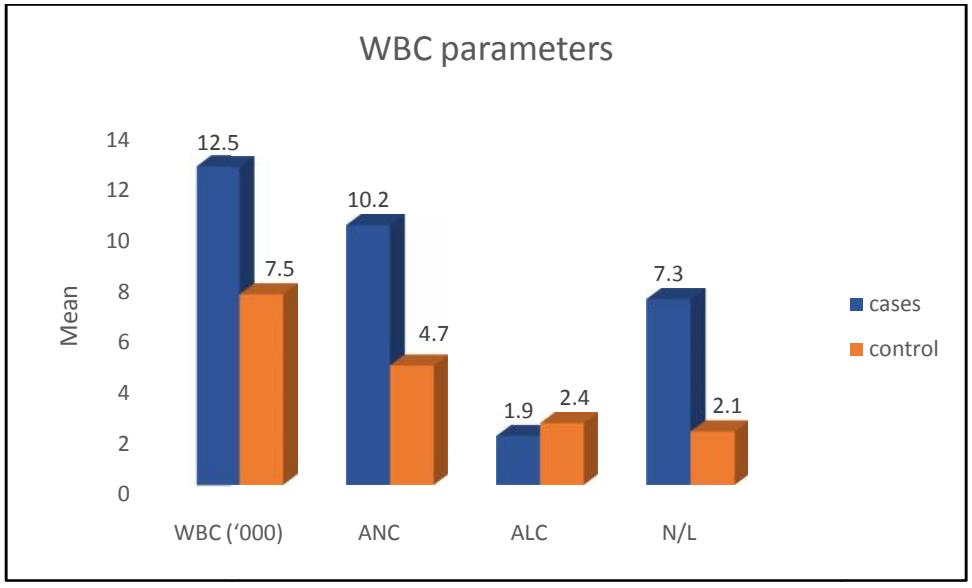
One of the important parameter which was derived in our study was Neutrophil to Lymphocyte ratio (NLR), which showed 7.3±5.8 in case group and in control group 2.1±0.7 and had significant p-value of <0.001 (table 7, figure 10).

Table 7: Comparison of mean WBC parameters among study groups

Parameters	Case		Control		p value
	Mean	SD	Mean	SD	
WBC ($x10^3$ per µl)	12.5	3.9	7.5	1.4	<0.001*
ANC ($x10^3$ per µl)	10.2	4	4.7	1.2	<0.001*
ALC ($x10^3$ per µl)	1.9	0.9	2.4	0.6	<0.001*
NLR	7.3	5.8	2.1	0.7	<0.001*

Note: *significantly distributed at 5% level of significance

Figure 10: Bar Chart of mean WBC parameters among study group and control group



RBC PARAMETERS

The mean value for Hemoglobin (Hb) (g/dl) is 13.1±2.7 in study group and in control group Hemoglobin was 13.1±1.7 with p-value 0.967 which was non-significant at 5% level of significance.

In present study, various other Red Blood Cell parameters which were also evaluated, like Hematocrit (%) where 39.2±7.2 and 40.6±4.9 mean in study and control group respectively. Another parameter of Red Blood Cell Count (10^6 per µl)was done with mean in study group was 4.6±0.8 and in control group 4.9±2.6. Other RBC parameters mean values like Mean Corpuscular Volume (MCV) (fl) was 86.1±9.7 in study group and 87.1±9.1 in control group and 28.6±4.0 in study group and 28.2±2.5 in control group was the value of Mean Corpuscular Hemoglobin (pg), p-value of all these parameters were statistically non-significant(table 8, figure 11).

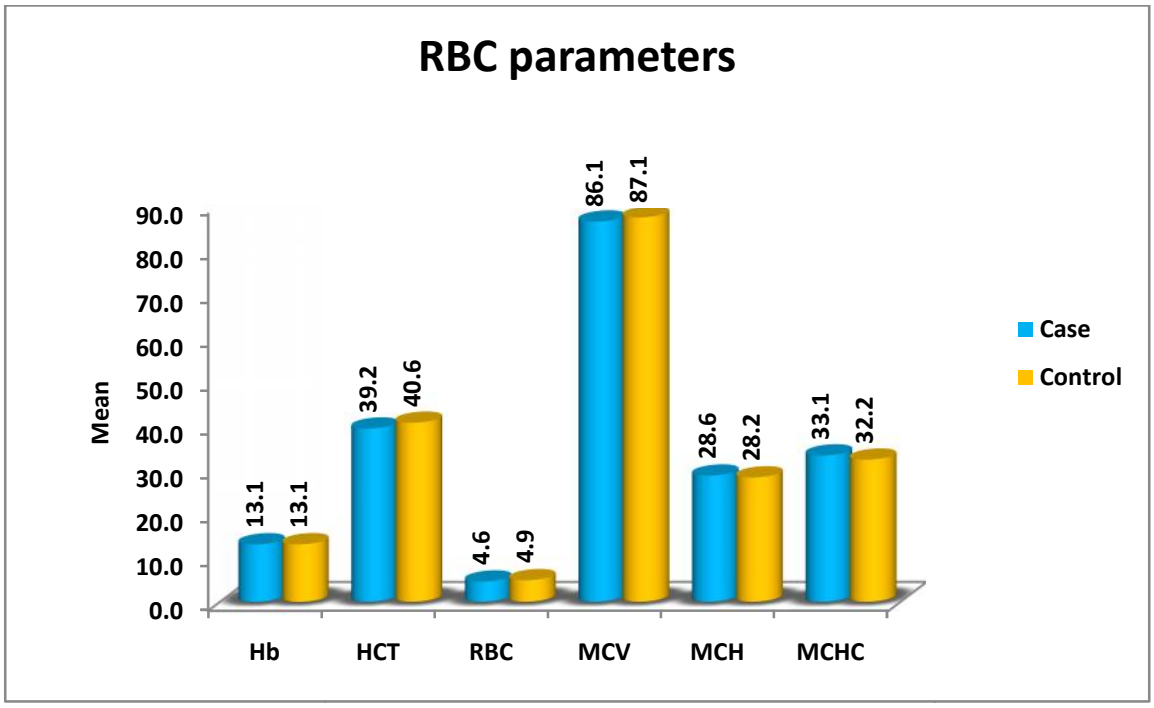
But out of all red blood cell parameters, a Mean Corpuscular Hemoglobin Concentration (MCHC) (g/dl)was significant with p-value <0.001 for which case group mean value were 33.1±2.0 and in control group mean value calculated to be 32.2±1.4 (table 8, figure 11).

Table 8: Comparison of mean RBC parameters among study groups

Parameters	Case		Control		p value
	Mean	SD	Mean	SD	
Hb (g/dl)	13.1	2.7	13.1	1.7	0.967
HCT (%)	39.2	7.2	40.6	4.9	0.103
RBC (10^6/µl)	4.6	0.8	4.9	2.6	0.238
MCV (fl)	86.1	9.7	87.1	9.1	0.448
MCH (pg)	28.6	4.0	28.2	2.5	0.344
MCHC (g/dl)	33.1	2.0	32.2	1.4	<0.001*

Note: *significantly distributed at 5% level of significance

Figure 11: Bar Chart of mean RBC parameters among study groups



PLATELET PARAMETERS

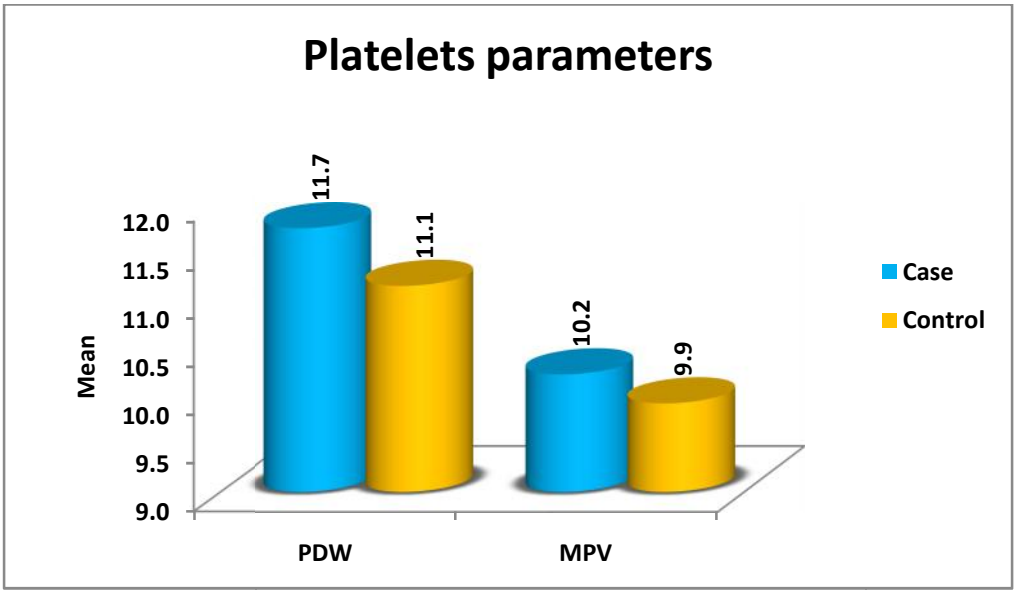
Platelet Distribution Width (PDW) (fl) and Mean Platelet Volume (MPV) (fl)were two of platelet parameters considered in our study. In study group, mean value for Platelet Distribution Width was 11.7±2.2 and in control group mean value was 11.1±2.1. Mean Platelet Volume (MPV) value 10.2±0.9 in study group and 9.9±0.9 in control group. The p-value of these both platelet parameters (MPV, PDW) were statistically significant (table 9, figure 12).

Table 9: Comparison of mean Platelets parameters among study groups

Parameters	Case		Control		p value
	Mean	SD	Mean	SD	
PDW (fl)	11.7	2.2	11.1	2.1	0.032*
MPV (fl)	10.2	0.9	9.9	0.9	0.014*

Note:*significantly distributed at 5% level of significance

Figure 12: Bar chart of mean Platelets parameters among study groups



DISCUSSION

Cardiovascular disease stands as one of the leading cause of mortality, worldwide. In this context cardiovascular disease can give rise to coronary heart disease which attribute to the involvement of myocardial infarction and may eventually lead to sudden death. Extensive research during the past decades has offered potential of new parameters for predicting coronary heart disease risk by establishing atherosclerosis as an inflammatory disease.[14,27,37,38] Although myocardial infarction occurs mainly in older patients which are above 45 years, younger men and women can also be affected.[14] There is an extensive increase in the prevalence of heart disease among specific ethnic groups like Asian Indian origin, these people are at risk of myocardial infraction at a much younger age as compared to rest of the world population and are associated with complex coronary artery abnormalities.[39] In this context, potentially 80% of cardiovascular disease cases are preventable due to modifiable risk factors but still mortality continues to rise, mainly because of ineffective and insufficient preventive measures.[19]

Atherosclerosis is the most common cause of cardiovascular disease. Coronary artery atherosclerosis leads to diminished blood supply to myocardium, which leads to myocardial infarction as blood flow reaches below critical levels, often accompanied by electrocardiogram changes.[19]

Elevated White Blood Cells (WBCs) count is a consistent indicator of inflammation. The total WBC number and its various cellular subtypes have been implicated as predictors of coronary heart disease. Almost all of the cellular elements of blood like WBCs, Red Blood Cells (RBCs) and Platelets are involved in pathogenesis of atherosclerosis and its complications. These cellular elements including platelets not only

plays a role in development of CHD, but can also help in predicting any recurring event and even death in previously diagnosed CHD patients.[38]

Table 10: Comparison of mean age (years) in both groups with various other studies.

	Control Age (years)	Case Age (years)
Selcuk *et al*[31]	57.7±10.8	60.7±10.5
Yuksel *et al*[26]	57.8±10.5	63.7±12.2
Ozdemir *et al*[27]	56.75±10.89	60.84±13.74
Present Study	57.99±11.64	57.91±11.36

An average age of onset of CVD is younger among Indians than the rest of the world. Affected age for the developed world is >45 years but in Indian setup affected age is between 35 to 64years, which correlates with our study where 75.8% of subjects were between 4^{th} to 6^{th} decades of life.[31] 15.5% of our study subjects were above 70years. In present study mean age for case group was 57.91±11.36 years and 57.99±11.64 years in control group (table 10).

In a study done by Selcuk *et al*[31],the mean age of patients which were affected were 60.7±10.5 years and mean age for control group was 57.7±10.8 years. The groups were indistinguishable in terms of age group.

In a study done by Yuksel *et al*[26], mean age for the study group with coronary artery disease was 63.7±12.2 years with mean age in control group having normal coronary artery disease was 57.8±10.5 years. The study group was comparable in terms

of traditional risk factors but patients with coronary artery disease were older as compared to control group.

In study done by Ozdemir *et al*[27], mean age for affected group was 60.84±13.74 with mean age in healthy control group was 56.75±10.89, both groups were similar.

Table 11: Comparison of gender within various studies.

	Males	Females
Demir *et al*[41]	49/75(65.3%)	26/75(34.7%)
Zhang *et al*[42]	59/76(77.6%)	17/76(22.4%)
Ozdemir *et al*[27]	101/149(67.8%)	48/149(32.2%)
Present Study	84/116(72.4%)	32/116(27.6%)

The prevalence of Cardiovascular disease is higher in males than females though the mortality due to CVD is higher among females. The Framingham study showed that women have a lower incidence of coronary artery disease than men do, until the age of 75years.[39,40]

In the present study, out of 116 total number cases, number of males were 84 (72.4%) and number of females were 32 (27.6%) (table 11, figure 7).

In the study done by Demir *et al*[41],49 (65.3%) cases out of total 75 patients affected with coronary artery disease were male and 26 (34.7%) were female, showing majority were males.

In a study done by Zhang *et al*[42], out of 76 cases of acute coronary syndrome under study, 59 (77.6%) were male patients and only 17 (22.4%) were female patients.

In a study conducted by Ozdemir *et al*[27], out of 149 cases with myocardial ischemia, 101 (67.8%) cases were males and 48 (32.2%) cases were only females.

In the present study, among the age distributions between males and females, as age increasedit was noted male to female ratio has decreased (table 5). In younger age group (3rd decade) it was 4:1 but as age increases ratio reduced to 1.57:1 in age above 70 years. The largest group of people which were affected were in 5th decade and then in 6th decade of life. The least affected group of all was 3rd decade with only 10 (8.2%) out of 116 cases (table 6). It is widely accepted that postmenopausal women lose the protective action of endogenous estrogen on vascular endothelium, which lead to increased risk of coronary artery disease which was comparable with the findings of present study.[43]

HEMATOLOGICAL PARAMETERS

The association between white blood cell count and coronary artery disease (CAD) has been observed in different populations with varying baseline risk. CAD and WBC count association is independent of other risk factors like smoking, obesity and many others.[37] Even in individuals with disease free baseline, high white blood cell count has helped in anticipating future cardiovascular events.[37]Multiple researches have shown an association between high white blood cell count and high mortality rates in patients with unstable angina pectoris, acute coronary syndrome and in patients who have underwent percutaneous coronary intervention or even in cases of coronary artery bypass graft.[44,45]

Table 12: Total White blood cell count ($x10^3$ per µl) comparison with various studies

	Control	Case	p-value
Demir *et al*[41]	7.4±1.84	7.93±1.96	<0.001
Selcuk *et al*[31]	7.65±1.67	8.21±2.09	0.145
Naz *et al*[24]	7.49±1.16	9.29±3.62	<0.001
Sweetnam *et al*[32]	7.48±2.12	8.18±2.10	<0.0001
Nilsson *et al*[46]	5.5±1.4	7.7±2.3	<0.01
Present study	7.51±1.44	12.55±3.95	<0.001

The leukocyte count has been correlated with Coronary heart disease since 1920. A number of studies have been conducted in CHD free populations over several decades to show positive correlation between leukocyte count and risk of CHD.[38]

In the present study, mean value of total WBC count was 12.55±3.95 in case group and 7.51±1.44 in control group, showing a significant correlation, which was further compared with studies done in by Demir *et al*[41], Naz *et al*[24], Sweetnam *et al*[32]and Nilsson *et al*[46] (table 12).

In the study done by Demir *et al*[41], showed total WBC count as 7.93±1.96 in coronary artery disease group and 7.4±1.84 in control group, showing as association in severity of coronary artery disease patients.

In a study done by Selcuk *et al*[31], total WBC count was 8.21±2.09 and 7.65±1.67 in case and control group, respectively. This study found no correlation between coronary artery disease and total WBC count.

In a study conducted by Naz *et al*[24], mean for total WBC count was 7.49±1.16 in control group and 9.29±3.62 in group affected by coronary artery disease which was significantly high.

In study done by Sweetnam *et al*[32] and Nilsson *et al*[46], total WBC count in case group was 8.18±2.10 and 7.7±2.3 along with 7.48±2.12 and 5.5±1.4 in control group respectively. These studies show comparable results of present study data showing strong correlation to total leucocyte count.

Table 13: Total Absolute Neutrophil Count (ANC) ($x10^3$ per µl) comparison with various studies

	Control	Case	p-value
Demir *et al*[41]	4.16±1.27	4.78±1.36	<0.01
Selcuk *et al*[31]	4.50±1.5	5.1±1.67	0.063
Erkol *et al*[34]	7.8±2.4	9.7±3.8	<0.001
Bajari *et al*[35]	7.68±3.38	10.30±2.81	<0.001
Nilsson *et al*[46]	3.0±1.0	4.5±1.7	<0.001
Present Study	4.7±1.2	10.2±4.0	<0.001

In a study done by Demir *et al*[41], ANC in study group was 4.78±1.36 and in control group it was 4.16±1.27 which show significant correlation.

In a study done by Selcuk *et al*[31], ANC in study group was 5.1±1.67 and 4.50±1.5 in control group showing no noteworthy difference.

In various other studies done by Erkol *et al*[34], Bajari *et al*[35]and Nilsson *et al*[46]showed remarkable correlation of ANC with coronary pathology. These results were comparable to the present study, in which mean value for absolute neutrophil count (ANC) in study group was 10.2±4.0 and 4.7±1.2 in control group (table 13).

This result representing a strong correlation with neutrophils due to secretion of proteolytic neutral proteases which promotes detachment of endothelial cells from vessel walls and adherence of platelets to subendothelial collagen. In activated stage neutrophils also release large amount of chemotactic agent Leukotriene B4and also many inflammatory mediators.[37]

Table 14: Total Absolute Lymphocyte Count (ALC) ($x10^3$ per µl) comparison with various studies

	Control	Case	p-value
Demir *et al*[41]	2.46±0.76	2.33±0.67	0.01
Selcuk *et al*[31]	2.39±0.61	2.03±0.74	0.010
Erkol *et al*[34]	2.4±1.0	1.9±1.1	<0.001
Munir *et al*[33]	1.61±0.05	1.95±0.07	0.001
Nilsson *et al*[46]	2.0±0.6	2.4±0.9	Not Significant
Present Study	2.4±0.6	1.9±0.9	<0.001

In study done by Demir *et al*[41], ALC in coronary artery disease (CAD) group was 2.33±0.67 and 2.46±0.76 in control group which showed a correlation between the two group.

In study done by Selcuk *et al*[31], ALC in CAD group was 2.03±0.74 and 2.39±0.61 in control group, they found an negative correlation with respect to lymphocyte count and severity of atherosclerosis showing lymphopenia is associated with increased risk of developing myocardial infarction in acute chest pain patients.

In a study conducted by Erkol *et al*[34], ALC in affected group was 1.9±1.1 and in control group it was 2.4±1.0, showing lymphopenia is related to progression of CAD, plaque venerability and increase mortality.

In study done by Munir *et al*[33], ALC was 1.95±0.07 in case group and 1.61±0.05 in control group, displaying stress presented by ACS.

In study done by Nilsson *et al*[46], showed no correlation between affected group having ALC as 2.4±0.9 and controls as 2.0±0.6.

In our present study, absolute lymphocyte count (ALC) in study group was 1.9±0.9 and in control group it was 2.4±0.6, showing significant correlation of lymphopenia to coronary pathology (table 14).

Table 15: Neutrophil Lymphocyte Ratio (NLR) comparison with various studies

	Control	Case	p-value
Demir *et al*[41]	1.79±0.57	2.95±1.29	<0.001
Zhang *et al*[42]	-	3.64±1.94	<0.001
Selcuk *et al*[31]	2.04±1.01	2.86±1.57	<0.001
Naz *et al*[24]	1.61	3.67	<0.001
Nunez *et al*[25]	-	-	<0.001
Erkol *et al*[34]	4.1±3.2	6.9±5.5	<0.001
Bajari *et al*[35]	4.10±3.39	7.92±3.46	<0.001
Munir *et al*[33]	2.7±0.11	3.2±0.15	0.006
Present Study	2.1±0.7	7.3±5.8	<0.001

The balance between various leucocyte subtypes modulates the inflammatory response. Not just high neutrophil count but also lymphocytopenia also contributes to worse clinical outcome with ST-elevation myocardial infarction. Thus, neutrophil to lymphocyte ratio (NLR) has emerged as effective indicator of inflammatory state in various clinical studies.[25,38]

NLR has also emerged as an indicator of early and late clinical outcomes in patients of ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention.[31,38]

In various studies done by Demir *et al*[41], Zhang *et al*[42], Selcuk *et al*[31], Naz *et al*[24], Nunez *et al*[25], Erkol *et al*[34], Bajari *et al*[35] and Munir *et al*[33], showed significant correlation between coronary pathology and NLR. These studies have shown that NLR is not only an

independent predictor of presence of coronary artery disease but also can be helpful in long term mortality prediction. Present study results were also indistinguishable from these studies[37,38](table 15).

RBC parameters:

Table 16: Hemoglobin (Hb) (gm/dl) comparison with various studies

	Control	Case	p-value
Demir *et al*[41]	14.2±1.6	13.7±1.7	0.16
Erkol *et al*[34]	14.0±1.7	14.0±1.8	0.18
Adam *et al*[36]	12.54±2.28	12.40±2.07	0.630
Tsai *et al*[47]	13.3±2.1	13.1±2.6	0.033
Present Study	13.1±1.7	13.1±2.7	0.967

In present study, Hemoglobin (Hb) levels were evaluated in study group which was 13.1±2.7 and 13.1±1.7 in control group, showing no significant relation in acute coronary syndrome patients and in normal healthy controls. Similar studies done by Demir *et al*[41], Erkol *et al*[34], Adam *et al*[36] and Tsai *et al*[47] showed similar finding as present study that there was no significant relation between acute coronary event and hemoglobin (Table 16).

Table 17: Hematocrit (HCT) (%) comparison with various studies

	Control	Case	p-value
Ozdemir *et al*[27]	39.91±4.29	40.57±5.04	0.068
Adam *et al*[36]	37.94±6.20	37.41±6.28	0.491
Ishida *et al*[48]	39.1±5.50	35.4±6.1	0.008
Tsai *et al*[47]	39.8±5.9	39.1±6.3	0.039
Present Study	40.6±4.9	39.2±7.2	0.103

Though the clinical usefulness of Hematocrit (HCT) alone is still unclear but there are many studies of different patient populations which show there may be an association between increased hematocrit and risk of CHD.[37,49]One of such studies was done by Ishida *et al*[48] showing mean HCT as 35.4±6.1 in study group and 39.1±5.50 in control group, showing significant association.

In the present study HCT for case group was 39.2±7.2 and in control group 40.6±4.9 with direct no significant correlation with acute coronary syndrome which was in harmony other studies done by Ozdemir *et al*[27], Adam *et al*[36] and Tsai *et al*[47] where difference between control group and study group were not significant (table 17).

Table 18: Red blood cell (RBC) count (10^6/µl) comparison with various studies

	Control	Case	p-value
Zhang *et al*[42]	-	4.25±0.57	0.537
Adam *et al*[36]	4.64±0.76	4.58±0.85	0.550
Tsai *et al*[47]	4.58±1.34	4.46±0.94	0.030
Present Study	4.9±2.6	4.6±0.8	0.238

Elevated RBC counts and hematocrit in association with increased viscosity is a risk factor for coronary events which causes release of adenosine diphosphate for RBCS resulting in increased platelet adhesion.[37,38]

In a study by Zhang *et al*[42] mean value of RBC was 4.25 ±0.57, showing non-significant association, which was similar to study by Adam *et al*[36] and Tsai *et al*[47]where mean values were 4.58 ±0.85 and 4.46±0.94 in study group and 4.64±0.76 and 4.58±1.34 in control group, respectively.

In our present study mean value of 4.6± 0.8 in study group and 4.9±2.6 in control group, which was non-significant which correlates with studies done by Zhang *et al*[42], Adam *et al*[36] and Tsai *et al*[47] (table 18).

Table 19: Mean Corpuscular Hemoglobin (MCH) (pg) comparison with various studies

	Control	Case	p-value
Ishida *et al*[48]	31.6±2.0	29.9±3.6	0.002
Tsai *et al*[47]	30.02±9.1	29.6±3.0	0.323
Present Study	28.2±2.5	28.6±4	0.344

The role of mean corpuscular hemoglobin (MCH) level in acute coronary syndrome is minimal. In percutaneous coronary interventions (PCI) duration of antiplatelet therapy is determined by various factors, the value of MCH plays an important role in decision discontinue the drug.[48]

In a study done by Ishida *et al*[48], in acute coronary patients found that and their mean was 29.9±3.6 and 31.6±2.0 in control group, showing significant association about duration of anti-platelet therapy relation to MCH levels.

In studies done by Tsai *et al*[47], mean corpuscular hemoglobin level in study group was 29.9±3.6 and 30.02±9.1 in control group, which is similar to present study with a mean value 28.6±4 in study group and 28.2±2.5 in control group showing no significant relation between acute coronary pathology and MCH levels (table 19).

Table 20: Mean Corpuscular Volume (MCV) (fl) comparison with various studies

	Control	Case	p-value
Ishida *et al*[48]	93.4±6.1	90.1±8.2	0.040
Tsai *et al*[47]	88.2±7.6	88.6±7.9	0.320
Present Study	87.1±9.1	86.1±9.7	0.448

In the present study, mean value for Mean Corpuscular Volume (MCV) in study group was 86.1±9.7 and 87.1±9.1 in control group, which showed no significant association. These findings were similar to studies done by Ishida *et al*[48] and Tsai *et al*[47], which also showed no notable relation between acute coronary syndrome and MCV (table 20).

In the study done by Ishida *et al*[48], mean value for MCV in study group was 90.1±8.2 and in control group it was 93.4±6.1, showing no notable relation between acute coronary syndrome and MCV.

In a study conducted by Tsai *et al*[47], value for MCV in study group was 88.6±7.9 and 88.2±7.6 in control group, with no significant correlation.

Table 21: Mean Corpuscular Hemoglobin concentration (MCHC) (g/dl) comparison with various studies

	Control	Case	p-value
Ishida *et al*[48]	33.9±1.7	33.1±1.4	0.052
Tsai *et al*[47]	33.5±1.2	33.3±1.4	<0.0001
Present Study	32.2±1.4	33.1±2.0	<0.001

In the study done by Ishida *et al*[48], mean value for MCHC in study group was 33.1±1.4 and in control group it was 33.9±1.7, showing no notable relation between acute coronary syndrome and MCHC.

In a study conducted by Tsai *et al*[47], value for MCHC in study group was 33.3±1.4 and 33.5±1.2 in control group, with significant correlation.

In the present study, mean value for Mean Corpuscular Hemoglobin concentration (MCHC) in study group was 33.1±2.0 and 32.2±1.4 in control group, with significant association (table 21).

The exact mechanism of increased MCHC in ACS is not well understood, one of the author has hypothesized that inflammation and iron status partially explains the association as inflammatory response which may leads to iron deficiency, further causing decrease in MCHC and affect prognosis but further studies are needed to completely understand the exact mechanism.[50]

Platelets:

Platelet indices were analyzed in patients with acute coronary syndrome and compared with healthy control group. The Mean Platelet Volume (MPV) value evaluated in our study group was 10.2±0.9 as compared to 9.9±0.9 in control group, showing an association of platelet function in case of coronary event.

In present study, MPV was raised in patients who had suffered acute coronary event when compared to controls. This is in agreement with results of study done by Jaya *et al*[13] where MPV was9.67±0.82fl in study group and 8.4±0.67fl in control group, which showed a significant relation between platelets contributing to prethrombotic state in acute ischemic syndrome.

Further in studies done by Chu *et al*[51] and Khode *et al*[52] showed MPV is significantly associated with acute coronary syndrome in patients with acute chest pain as an independent predictor. These studies also showed that elevated MPV is associated with acute myocardial infarction and also with restenosis following coronary artery intervention.

However, in a study done by Yuksel *et al*[26], MPV value was 8.4±1.4 and 8.3±1.3 in study group and control group respectively showed no relation to acute coronary syndrome.

Table 22: Mean Platelet Volume (MPV) (fl) comparison with various studies

	Control	Case	p-value
Jaya *et al*[13]	8.4±0.67	9.67±0.82	<0.001
Yuksel *et al*[26]	8.3±1.3	8.4±1.4	0.715
Chu *et al*[51]	8.48±1.42	9.24±1.69	<0.001
Khode *et al*[52]	9.21±0.6	9.65±0.9	0.018
Present Study	9.9±0.9	10.2±0.9	0.014

In ACS, rupture of unstable atherosclerotic plaque triggers a thrombotic cascade leading to the clinical event. However, platelet reactivity is critical for formation and propagation of intracoronary thrombus.[49] MPV, is one of the simple and easy marker which can be used. MPV was significantly raised in patients with acute MI in comparison to healthy control group in the present study which is in agreement with studies done by Jaya *et al*[13,]Khode *et al*[52] and Chu *et al*[51] (table 22).

Table 23: Platelet Distribution Width (PDW) (fl) comparison with various studies

	Control	Case	p-value
Jaya *et al*[13]	12.23±3.13	13.66±3.55	<0.0001
Ozdemir *et al*[27]	16.58±0.51	16.81±0.60	0.0120
Erkol *et al*[34]	16.0±1.8	16.0±1.4	0.98
Present Study	11.1±2.1	11.7±2.2	<0.032

Platelet distribution width (PDW) was also significantly higher in the patients diagnosed with ACS i.e. 11.7±2.2 as compared to control group with 11.1±2.1 showing similar trend to study done by Ozdemir *et al*[27]. In another study done by Jaya *et al*[13] which showed 13.66±3.55 and 12.23±3.13 in study and control group, respectively.

In study done by Erkol *et al*[34] mean value in study group was 16.0±1.4 and 16.0±1.8 in control group, showing no significant relationship between PDW in acute coronary event contradicting the results of present study.

Overall, in the present study Total White Blood Cell (WBC) counts, Absolute Neutrophil Count(ANC), Absolute Lymphocyte Count (ALC), Neutrophil Lymphocyte Ratio(NLR), were showing significant association with acute coronary syndrome.

In Red Blood Cell parameters, MCHC was the only affected parameter which was significantly correlating in the study group but the possible mechanism is not well understood, proposed mechanism which has been stated in literature, have considered MCHC as inflammatory marker.[50]

In platelet parameters, MPV and PDW both were statistically significant in acute coronary syndrome patients.

These results were comparable to other studies in literature. Possible mechanism is result of inflammatory cytokines and acute cellular response due to inflammation. Few studies done also showed contradicting results to the present study. Larger studies are required to further validate the results.

RECOMMENDATIONS:

1. For routineComplete blood count (CBC) hematology report in addition, we recommend toinclude ANC, ALC, NLR, MPV & PDW for the patients admitted in intensive care unit for reason of cardiovascular disease.
2. Awareness and utility of these new parameters to the residents and critical care specialists for timely intervention in ACS patient management for better outcome.
3. Neutrophil Lymphocyte Ratio (NLR) can also be used for risk stratification in cases of previously diagnosed Ischemic Heart Disease patients.

LIMITATIONS:

1. Larger sample size studies are required to substantiate the results.
2. The present study has not segregated smokers/non-smokers and parameters of lipid profile were not considered.
3. Follow up of patients and outcome has to be considered.
4. Platelets with qualitative disorders and causes of reactive platelets were not assessed.

SUMMARY

- This study was undertaken in Shri B.M. Patil Medical College, Vijayapur, Karnataka to study correlation of various complete blood count parameters in Acute Coronary Syndromes(ACS).
- A total of 232 subjects were studied and were divided further into two groups of 116 subjects each, who were diagnosed with ACS and age and sex matched healthy controls.
- Majority of patients diagnosed as ACS belonged to the 5^{th} decade of life (33.6%), followed by 6^{th} decade (24.1%) and 4^{th} decade (18.1%) of life.
- In the present study, total number of males including both cases and controls were 164 (72.4%) and number of females were 64 (27.6%) with male: female ratio being 2.6:1
- The total White blood cell (WBC) count ($x10^3$ per µl) was 12.5(±3.9) and in control group was 7.5(±1.4) with p-value <0.001 at 5% level of significance.
- Mean values for Absolute Neutrophil Count (ANC) ($x10^3$ per µl) was 10.2(±4.0) in study group and 4.7(±1.2) in control group. Mean values for Absolute Lymphocyte Count (ALC) ($x10^3$ per µl) was 1.9(±0.9) and 2.4(±0.6) in study and control group, which were statistically significant.
- The Neutrophil lymphocyte ratio (NLR) was higher in ACS patients 7.3(±5.8) than in healthy individuals with 2.1(±0.7), which was statistically significant (p<0.001).
- The Hemoglobin (Hb) recorded in our study for study group was 13.1(±2.7), in comparison to control group where it was 13.1(±1.7). The p-value for Hb was

however not statistically significant for evaluating a cardiovascular patients profile.

- Various Red blood cell parameters like Hematocrit (HCT), RBC count, Mean corpuscular volume (MCV), Mean Corpuscular hemoglobin (MCH) mean value were statistically non-significant compared to control group.
- The Mean Corpuscular Hemoglobin Concentration (MCHC) in ACS group was 33.1(±2.0) and in healthy group was 32.2(±1.4), which was statistically significant ($p<0.001$).
- Platelet indices studied were Mean Platelet Volume (MPV) and Platelet distribution width (PDW) of which both were statistically significant with p-value 0.014 and 0.032 respectively.

CONCLUSION

The study was undertaken to determine the association between various complete blood count parameters generated in automated cell counters (either 3-part or 5-part differential counters) like Total White Blood Cell (WBC) counts, Absolute Neutrophil Count (ANC), Absolute Lymphocyte Count (ALC), Neutrophil Lymphocyte Ratio (NLR), Hemoglobin (Hb), Hematocrit (HCT), Mean Corpuscular Volume (MCV), Mean Corpuscular Hemoglobin (MCH), Mean Corpuscular Hemoglobin Concentration (MCHC), Mean Platelet Volume (MPV), Platelet Distribution Width (PDW) in acute coronary events. These indices were analyzed in patients with acute coronary syndrome and found to be statistically significant when compared with healthy control subjects. Such patients can easily be identified during routine hematological investigations and possibly benefit for the prevention, treatment intervention at earliest and prognostication.

Present study concluded that among the CBC parameters generated in automated cell counters; Total WBC count, ANC, ALC, NLR, MCHC and PDW are readily available as part of routine hematology investigations, which are relatively inexpensive, are useful markers and were significantly raised among patients admitted with ACS in our hospital. Red cell parameters except MCHC didn't show any significant association among the patients and healthy population. We recommend: these CBC parameters should be utilized with other investigative tools for timely management of patients admitted in ICU diagnosed with acute coronary syndrome.

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ANNEXURES

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

No/58/2015
20/11/15

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

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

Title "The correlation between different complete blood count parameters in acute coronary syndromes"

Name of P.G. Student : Dr Abhey Chawla, Dept of Pathology

Name of Guide/Co-investigator : Dr. M. H. Karigoudar, Professor

DR.TEJASWINI VALLABHA
CHAIRMAN

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

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

RESEARCH INFORMED CONSENT FORM

TITLE OF THE PROJECT: THE CORRELATION BETWEEN DIFFERENT COMPLETE BLOOD PARAMETERS IN ACUTE CORONARY SYNDROMES.

PRINCIPAL INVESTIGATOR: Dr. ABHEY CHAWLA.
P.G.DEPARTMENT OF PATHOLOGY

P.G.GUIDE: Dr. MAHESH. H. KARIGOUDAR
PROFESSOR,
DEPARTMENT OF PATHOLOGY

P.G. CO-GUIDE: Dr. S. M. BIRADAR
ASSOCIATE PROFESSOR,
DEPARTMENT OF MEDICINE

PURPOSE OF RESEARCH:

I have been informed that this study is done to know the correlation between various complete blood count and acute coronary syndromes.

PROCEDURE:

I understand that I undergo detailed history and after which blood sample will be taken.

RISK AND DISCOMFORTS:

I understand that, there is no risk involved in the procedures performed.

BENEFITS:

I understand that my participation in the study will help to know the prognosis of Acute Coronary Syndromes.

CONFIDENTIALITY:

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

REQUST FOR MORE INFORMATION:

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

REFUSAL FOR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary and that I may refuse to participate or may withdraw from the study at any time without prejudice.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me during the study I will get medical treatment but no further compensations.

I have read and fully understood this consent form. Therefore I agree to participate in the present study.

_____ _____

Participant / Guardian Date:

_____ _____

Signature of Witness Date:

I have explained the patient the purpose of the study, the procedure required and possible risk and benefit to the best of my ability in the vernacular language.

_____ _____

Investigator / P.G. Date:

_____ _____

Witness to Signature Date

PROFORMA FOR STUDY

Demographic Details:

Name:

Age:

Sex: M/F

OP/IP No. :

Lab. No. /Sample No. :

Chief complaints:

History of present illness:

Past history: Diabetes Mellitus, Hypertension, History of any drug intake

Family history:

General physical examination:

Systemic examination:

Clinical diagnosis:

Hematological investigations: (Complete blood count)

Sample Comment:

Parameters	
WBC	
RBC	
HGB	
HCT	
MCV	
MCH	
MCHC	
PDW	
MPV	
NEUTROPHIL	
LYMPHOCYTE	
MONOCYTE	
EOSINOPHIL	
BASOPHIL	

N/L RATIO:

BIOCHEMISTRY:

Cardiac Troponin:

CPK-MB:

OTHER INVESTIGATIONS:

ELECTROCARDIOGRAM:

IMPRESSION:

KEY TO MASTER CHART

OP/IP no.	:	Outpatient/ inpatient number
Lab no.	:	Laboratory number
N	:	Neutrophils
ANC	:	Absolute Neutrophil Count
L	:	Lymphocytes
ALC	:	Absolute Lymphocyte Count,
M	:	Monocytes
E	:	Eosinophil
B	:	Basophils
WBC	:	White Blood Cell Count
Hb	:	Hemoglobin
HCT	:	Hematocrit
MCV	:	Mean Corpuscular Volume
RBC	:	Red Blood Cell Count
MCH	:	Mean Corpuscular Hemoglobin
MCHC	:	Mean Corpuscular Hemoglobin Concentration
PDW	:	Platelet Distribution Width

MPV	:	Mean Platelet Volume
NLR	:	Neutrophil Lymphocyte Ratio
CK-MB	:	Creatine Kinase - MB
C.Trop	:	Cardiac Troponin T
ECG	:	Electrocardiograph
+	:	Positive (Myocardial infarction)
-	:	Negative (Normal study)
1	:	Positive
0	:	Negative

MASTER CHART - TEST

Sl No	Name	Age	Sex	OP/IP No.	Lab No.	N		L		M	E	B	WBC	Hb	HCT	MCV	RBC	MCH	MCHC	PDW	MPV	NLR	CK-MB	C.Trop.	ECG
1	Amasidda	36	M	5046	24203	90	14.985	8	1.332	0	2	0	16650	15.2	45.2	90	5.02	30.3	33.6	12.7	11.7	11.25			+
2	Nisar	43	M	22628	104968	80	11.024	12	1.6536	4	4	0	13780	16	47.6	77.9	6.11	26.2	33.6	13.9	11	6.67	85	1	+
3	Bheemanagouda	45	M	4490	21693	76	9.7508	18	2.3094	3	3	0	12830	16.4	48.9	89.6	5.46	30	33.5	11.3	10.1	4.22			+
4	Ajij	45	M	11055	50585	86	16.8474	13	2.5467	0	1	0	19590	15.2	46.2	87.3	5.29	28.7	32.9	12.9	10.8	6.62	16	0	+
5	Musawali	45	M	11170	51163	83	9.0553	11	1.2001	2	4	0	10910	13.4	38.2	91.2	4.19	32	35.1	10.7	9.6	7.55	143	1	+
6	Lalitabai	45	F	21790	100647	92	16.8728	6	1.1004	0	2	0	18340	13.2	38.8	92.2	4.21	29.9	34	11.4	10.3	15.33			+
7	Chinnawwa	50	F	14588	66412	66	7.458	28	3.164	2	4	0	11300	8.8	29.9	70.9	4.22	20.9	29.4	15.5	12.3	2.36		0	+
8	Gourabai	55	F	10908	49754	60	6.66	37	4.107	2	1	0	11100	10.7	34.3	60.4	5.68	18.8	31.2	13.8	10.1	1.62	18	0	+
9	Gururaj	55	M	14880	69584	91	16.5347	7	1.2719	1	1	0	18170	10.3	32.8	88.9	3.69	27.9	31.4	12.3	10.3	13.00	49	0	+
10	Veerabhadrappa	55	M	15020	68120	77	10.01	20	2.6	1	2	0	13000	14.2	39.3	73	5.38	26.4	36.1	11.7	9.5	3.85	24	0	+
11	Boramma	55	F	22798	105869	82	12.8248	15	2.346	0	3	0	15640	13.6	39.1	90.7	4.31	31.6	34.8	11.9	10.4	5.47	26	1	+
12	Jangir	57	M	11365	51853	92	13.432	6	0.876	0	2	0	14600	12.7	36.6	90.4	4.05	31.4	34.7	11.2	10.1	15.33	24	0	+
13	Chenaveerayya	58	M	10936	49837	82	7.3144	16	1.4272	0	2	0	8920	15.9	44.1	85.1	5.18	30.7	36.1	11.4	10.2	5.13	20	0	+
14	Shivabasappa	59	M	10922	49821	72	6.8544	25	2.38	1	2	0	9520	14.6	43.1	85.5	5.04	29	33.9	12.2	10.3	2.88	27	1	+
15	Basavaraj	60	M	3713	17158	73	8.03	23	2.53	2	2	0	11000	14.1	39.2	82.5	4.75	29.7	36	10.3	8.9	3.17	43	1	+
16	Arjun	62	M	15803	72034	71	10.1956	23	3.3028	2	4	0	14360	13.8	41.3	90.2	4.58	30.1	33.4	9.9	9.8	3.09		1	+
17	Ramagound	63	M	15232	69261	76	7.3188	20	1.926	1	3	0	9630	14	41.7	90.3	4.62	30.5	33.6	12.5	10.6	3.80	52	0	+
18	Pirappa	65	M	4264	20344	78	7.9716	17	1.7374	1	4	0	10220	14.2	43.3	93.3	4.64	30.6	32.8	13.1	10.7	4.59			+
19	Yallappa	65	M	11713	53403	91	15.2334	8	1.3392	0	1	0	16740	16	46.7	83.8	5.57	28.7	34.3	12.6	10.5	11.38	61	1	+
20	Kashibai	65	F	15935	72627	78	12.1212	17	2.6418	2	3	0	15540	12.6	37.8	88.9	4.25	29.6	33.3	11.6	10.1	4.59	33	0	+
21	Parvati	68	F	15720	50307	82	12.8904	14	2.2008	0	4	0	15720	11.1	33.8	73.8	4.58	24.2	32.8	15	12.2	5.86			+
22	Devanagouda	68	M	16070	73512	93	14.7684	4	0.6352	1	2	0	15880	15	43	84.1	5.11	29.4	34.9	13.6	11.1	23.25	53	0	+
23	Nagappa	71	M	15210	69226	94	17.014	5	0.905	0	1	0	18100	13.8	40.2	88.4	4.55	30.1	34.1	15.6	11.6	18.80			+
24	Lakkavva	75	F	3436	16136	82	10.2992	17	2.1352	0	1	0	12560	12.4	37.4	83.5	4.48	27.7	33.2	12.7	10.9	4.82		1	+
25	Bhagirathi	84	F	8390	38718	88	17.0896	9	1.7478	0	3	0	19420	9.2	29.8	107.2	2.78	33.1	30.9	9.7	9.9	9.78	31	1	+
26	Mahadevappa	57	M	25535	119502	57	6.0933	35	3.7415	0	8	0	10690	14.5	42.2	89	4.74	30.6	34.4	11.8	11.1	1.63		1	+
27	Sharanappa	64	M	26111	122246	70	7.315	24	2.508	1	5	0	10450	12.1	35.5	83.5	4.25	28.5	34.1	12.5	10.7	2.92	78	1	+
28	Shrishail	56	M	26244	122862	60	6.3	35	3.675	1	4	0	10500	16	46.6	90.8	5.13	31.2	34.3	10.4	9.5	1.71	26	1	+
29	Danappa	57	M	26411	123565	77	7.1918	17	1.5878	0	6	0	9340	14.5	42.6	81.9	5.20	27.6	34	12.1	10.6	4.53			+
30	Shivappa	72	M	27783	129862	74	8.5988	23	2.6726	2	1	0	11620	14.4	43.4	89.9	4.83	29.8	33.2	9.1	9	3.22	139	1	+
31	Mahantapp	45	M	28213	131703	87	16.0863	10	1.849	0	3	0	18490	14.7	42.7	91.8	4.65	31.6	34.4	10.5	9.7	8.70	26		+
32	Siddalingawwa	75	F	29122	136013	91	18.1181	7	1.3937	0	2	0	19910	10.9	33.6	83.4	4.03	27	32.4	12.3	10.7	13.00	52	1	+
33	Subhasgouda	60	M	29123	136024	64	6.1824	31	2.9946	1	4	0	9660	15.2	44.6	93.5	4.77	31.9	34.1	12.1	10.9	2.06	74	1	+
34	Udapirao	62	M	29134	136036	82	12.915	15	2.3625	1	2	0	15750	16.7	48.5	87.2	5.56	30	34.4	8.4	8.5	5.47	19	0	+
35	Appasaheb	60	M	29327	137073	80	8.832	18	1.9872	0	2	0	11040	14.8	45.3	78.1	5.80	25.5	32.7	12.3	10.5	4.44	247	1	+
36	Rudragouda	75	M	29617	138650	59	6.4841	35	3.8465	2	4	0	10990	13.8	41.6	89.8	4.63	29.8	33.2	12.6	10.5	1.69		1	+
37	Ishwarappa	50	M	29860	139755	95	10.8205	4	0.4556	0	1	0	11390	14.5	43.9	87.6	5.01	28.9	33	9.7	9.7	23.75	174	1	+
38	Shivappa	45	M	30007	140489	89	10.3418	10	1.162	0	1	0	11620	15.5	44.7	101.8	4.39	35.3	34.7	10.1	9.5	8.90	168	0	+

40	Pandit	45	M	30460	143040	82	9.6596	14	1.6492	2	2	0	11780	15.3	45.8	88.9	5.15	29.7	33.4	17.5	12.7	5.86	19	0	+
41	Sharanappa	60	M	30621	143533	85	8.585	13	1.313	1	1	0	10100	15.6	44.2	86	5.14	30.4	35.3	10.1	8.9	6.54	32	0	+
42	Devu	76	M	30776	144402	77	7.1918	19	1.7746	1	3	0	9340	13	40.7	84.8	4.80	27.1	31.9	9.9	9.3	4.05	27	1	+
43	Shridhar	65	M	30891	144731	85	7.395	10	0.87	2	3	0	8700	12.3	34.5	78.9	4.37	28.1	35.7	14.1	12	8.50	31	0	+
44	Jawar	40	M	30964	145216	91	13.7501	6	0.9066	1	2	0	15110	15.8	46.4	86.6	5.36	29.5	34.1	13.4	10.6	15.17		0	+
45	Ninganagouda	64	M	31290	146867	83	6.7894	13	1.0634	1	3	0	8180	9.4	32.1	75.5	4.25	22.1	29.3	10.5	9.4	6.38	29		+
46	Kallawwa	65	F	32022	150612	86	17.1656	12	2.3952	1	1	0	19960	6.2	21.9	56.3	3.89	15.9	28.3	9.6	9.4	7.17	23	1	+
47	Ramachandra	63	M	32056	150717	85	14.4415	14	2.3786	0	1	0	16990	16	47.3	100	4.73	33.8	33.8	14.2	11.1	6.07	36	0	+
48	Goudappagouada	38	M	32533	153030	92	16.6796	5	0.9065	0	3	0	18130	14.9	44.2	82.8	5.34	27.9	33.7	9.6	9.1	18.40		1	+
49	Panchakshari	53	M	32651	153625	62	3.7014	23	1.3731	1	8	0	5970	14.2	40.4	90.2	4.48	31.7	35.1	9.4	9.2	2.70	18	0	+
50	Gorakanath	45	M	32687	153668	72	7.6608	25	2.66	0	3	0	10640	13.5	39.9	80.9	4.93	27.4	33.8	8.6	8.7	2.88	45	1	+
51	Manu	62	M	33140	155887	70	6.37	25	2.275	3	2	0	9100	15.5	41.6	81.9	5.08	30.5	37.5	11.3	9.6	2.80	50	0	+
52	Appasaheb	78	M	33454	157683	75	6.33	19	1.6036	2	4	0	8440	10.1	31	79.7	3.89	26	32.6	13	10.9	3.95			+
53	Shivaprasad	72	M	33378	157121	80	8.168	11	1.1231	5	4	0	10210	10.7	33.5	78.6	4.26	25.1	31.9	9.3	9.5	7.27		0	+
54	Shankreppa	54	F	33447	157680	70	9.73	27	3.753	1	2	0	13900	15.5	46.7	95.9	4.87	31.8	33.2	12.7	10.8	2.59		1	+
55	Mohamad	58	M	33561	158322	89	6.2567	8	0.5624	1	2	0	7030	6.8	24.6	63.2	3.89	17.5	27.6	10.5	9.5	11.13	24	0	+
56	Gouramma	40	F	33632	1588987	78	16.0914	18	3.7134	2	2	0	20630	11.5	35.9	84.5	4.25	27.1	32	10.7	9.7	4.33	42	0	+
57	Subas	52	M	33836	159458	75	9.9375	22	2.915	0	3	0	13250	14.2	42	84.5	4.97	28.6	33.8	10.8	9.9	3.41	186	1	+
58	Mallamma	60	F	33947	159896	83	7.3704	14	1.2432	0	3	0	8880	12	36.3	91.2	3.98	30.2	33.1	10	9.7	5.93	37	1	+
59	Topu	60	M	34592	162571	86	3.784	12	0.528	1	1	0	4400	16.5	44.3	81.4	5.44	30.3	37.2	12.4	9.7	7.17	22	1	+
60	Balaram	59	M	34725	163102	63	5.103	30	2.43	3	4	0	8100	16.2	45.1	85.6	5.27	30.7	35.9	11.8	9.7	2.10	45	0	+
61	Bagamabee	70	F	34808	163613	89	12.015	9	1.215	1	1	0	13500	12.6	35.2	80.2	4.39	28.7	35.8	13.7	10.2	9.89	49	1	+
62	Somanna	60	M	34885	163979	73	6.862	25	2.35	1	1	0	9400	13	38.4	74	5.19	25	33.9	14.2	10.1	2.92	27	0	+
63	Siddappa	56	M	35445	166626	91	11.648	7	0.896	0	2	0	12800	14.8	39.1	84.8	4.61	32.1	37.9	11	9.2	13.00	46	0	+
64	Khadirabasha	75	M	35548	167167	78	11.1618	18	2.5758	2	2	0	14310	13.8	39.6	85.5	4.63	29.8	34.8	8.9	8.9	4.33	70	1	+
65	Ningawwa	80	F	35620	167696	66	4.752	30	2.16	1	3	0	7200	5.2	15.8	96.3	1.64	31.7	32.9	7.9	9	2.20	30		+
66	Rachayya	35	M	35769	168120	95	12.407	5	0.653	0	0	0	13060	14.6	40.5	101.8	3.98	36.7	36	11	10.3	19.00		0	+
67	Putalabai	75	F	35895	168866	87	15.2076	10	1.748	1	2	0	17480	11	33.6	100	3.36	32.7	32.7	12.9	11	8.70		1	+
68	Basavaraj	40	M	36016	169347	64	5.9456	32	2.9728	1	3	0	9290	15.1	45.1	89.3	5.05	29.9	33.5	9.1	8.6	2.00	65	0	+
69	Sanganagouda	75	M	36173	170127	85	8.3385	11	1.0791	1	3	0	9810	12.7	40.9	78.7	5.20	24.4	31.1	15.1	11.9	7.73	32	0	+
70	Riyazahmad	56	M	36285	170528	85	17.3485	14	2.8574	0	1	0	20410	14.5	42.9	86	4.99	29.1	33.8	10.1	9.6	6.07	36	1	+
71	Irubai	75	F	36426	171110	83	8.3415	15	1.5075	0	2	0	10050	10.7	33.3	82.8	4.02	26.6	32.1	10.6	9.9	5.53			+
72	Boramma	55	F	36464	171203	65	4.2185	32	2.0768	1	2	0	6490	12.3	35.4	86.6	4.09	30.1	34.7	10.7	9.8	2.03	40	0	+
73	Mallanagouda	52	M	36863	173007	65	4.4655	28	1.9236	3	4	0	6870	16	46.5	98.5	4.72	33.9	34.4	14.4	11.4	2.32		1	+
74	Rayappa	63	M	37106	174248	86	7.3444	10	0.854	1	3	0	8540	14.2	41.9	86.9	4.82	29.5	33.9	12.3	10.5	8.60		1	+
75	Balappa	65	M	37279	175235	86	13.0376	12	1.8192	0	2	0	15160	11.8	35.1	82.6	4.25	27.8	33.6	11.8	10.5	7.17		1	+
76	Subhas	53	M	37316	175439	86	8.3678	12	1.1676	0	2	0	9730	12.5	36.5	90.3	4.04	30.9	34.2	9.7	9.3	7.17	112	1	+
77	Shreemant	64	M	37726	177704	78	7.5036	20	1.924	0	2	0	9620	12.5	37.3	101.9	3.66	34.2	33.5	8.7	8.9	3.90	207	1	+
78	Shashikala	46	F	37897	178393	94	14.5512	4	0.6192	1	1	0	15480	13.3	41	84.9	4.83	27.5	32.4	12.3	10.3	23.50			+
79	Bagirati	62	F	38294	180161	58	2.4302	40	1.676	1	1	0	4190	3.8	10.9	125.3	0.87	43.7	34.9	23	13.6	1.45	41	1	+
80	Husenbi	80	F	38803	182964	97	12.4451	3	0.3849	0	0	0	12830	11.3	33.2	78.9	4.21	26.8	34	10.3	10	32.33	300		+
81	Devibai	60	F	39312	185717	80	8.88	18	1.998	0	2	0	11100	12.9	37.8	74.4	5.08	25.4	34.1	11.1	11	4.44	300	1	+
82	pralhad	47	M	39561	186813	80	7.528	18	1.6938	0	2	0	9410	13.8	42.3	86	4.92	28	32.6	12.9	10.7	4.44		1	+

84	Amasidda	46	M	38913	183481	83	12.8899	14	2.1742	0	3	0	15530	14.3	42.4	103.4	4.10	34.9	33.7	9	9.5	5.93	138	1	+
85	Shivanand	48	M	39148	184679	80	8.728	16	1.7456	1	3	0	10910	13.9	42.2	83.9	5.03	27.6	32.9	12.9	11.4	5.00	37	1	+
86	Mahadev	55	M	40599	192150	79	9.7407	18	2.2194	0	3	0	12330	16.2	50.8	95.1	5.34	30.3	31.9	10.3	9.5	4.39	300		+
87	Ramabai	55	F	40585	192102	54	5.1462	40	3.812	3	3	0	9530	7.1	25.6	56.3	4.55	15.6	27.7	10.4	10.2	1.35	45	0	+
88	Kallappa	65	M	40317	190829	94	17.1644	4	0.7304	0	2	0	18260	13.2	38.4	86.7	4.43	29.8	34.4	14	11.2	23.50	47	0	+
89	Ramesh	34	M	40855	193559	94	17.4088	4	0.7408	0	2	0	18520	12.9	40.7	81.7	4.98	25.9	31.7	9.7	9.1	23.50	22	1	+
90	Iragantappa	45	M	41208	195081	81	12.6522	15	2.343	1	3	0	15620	16.8	51	87.3	5.84	28.8	32.9	10.9	9.7	5.40			+
91	Shankareppa	54	M	41367	196465	59	7.7939	35	4.6235	2	4	0	13210	14.1	41.8	92.9	4.50	31.3	33.7	13.1	10.9	1.69			+
92	Bharat	52	M	41571	197185	86	10.2856	11	1.3156	0	3	0	11960	14.4	42.8	80.5	5.32	27.1	33.6	13.4	10.8	7.82	62	1	+
93	Zameer	38	M	41858	198613	85	12.6565	14	2.0846	0	1	0	14890	16.9	49.5	80.1	6.18	27.3	34.1	18.3	12.7	6.07		1	+
94	Adiveppa	72	M	42049	199401	86	10.922	8	1.016	2	4	0	12700	12.2	34.6	79.9	4.33	28.2	35.3	12.5	10.2	10.75	23	1	+
95	Md ibrahim	44	M	42068	199840	78	11.2788	18	2.6028	0	4	0	14460	15.7	49.1	83.1	5.91	26.6	32	10.9	9.7	4.33	31	1	+
96	Malappa	75	M	42564	202304	89	1.4952	6	0.1008	1	4	0	1680	10.4	35.2	62.2	5.66	18.4	29.5	10.2	9.8	14.83	98	1	+
97	Kantabai	52	F	42870	203974	84	12.4908	15	2.2305	0	1	0	14870	9.1	30.2	76.8	3.93	23.2	30.1	10.4	10	5.60	71	1	+
98	Sharanappa	42	M	43168	205462	74	4.7582	22	1.4146	2	2	0	6430	13	38.4	95	4.04	32.2	33.9	10.4	9.6	3.36		1	+
99	Sadashiva	70	M	10986	56077	85	9.8685	13	1.5093	1	1	0	11610	10.8	33.8	105.6	3.20	33.8	32	11.6	10.2	6.54	227	1	+
100	Bheemabai	65	F	10420	53423	88	12.9096	9	1.3203	1	2	0	14670	11.6	37.5	84.8	4.42	26.2	30.9	9.5	9.5	9.78	300	1	+
101	Siddu	40	M	4754	24338	86	10.1996	10	1.186	1	3	0	11860	16.9	53.8	86.8	6.20	27.3	31.4	12.3	11	8.60	51	1	+
102	Subhaschandra	50	M	4496	22922	79	6.5491	16	1.3264	2	3	0	8290	14.3	46.5	81.4	5.71	25	30.8	14.2	11.3	4.94	100	1	+
103	Kamalabai	62	F	2969	148229	87	12.441	10	1.43	1	2	0	14300	7.8	26.5	76.4	3.47	22.5	29.4	8.9	8.1	8.70	57	1	+
104	Neelamma	50	F	2388	12109	92	16.9832	8	1.4768	0	0	0	18460	11.2	37.7	88.9	4.24	26.4	29.7	13.9	10.9	11.50	50	1	+
105	Krishna	55	M	2387	12103	88	17.5032	10	1.989	0	2	0	19890	15.3	47.3	89.2	5.30	28.9	32.3	11.1	9.6	8.80	246	1	+
106	Nagamma	62	F	2227	11252	84	10.2144	12	1.4592	1	3	0	12160	13.7	40	83	4.82	28.4	34.3	12	10.5	7.00		1	+
107	Raghavendra	55	M	2157	10798	66	7.2468	24	2.6352	6	4	0	10980	14.5	43.2	85	5.08	28.5	33.6	10.6	9.5	2.75	300	1	+
108	Anvarsab	70	M	6164	31309	83	18.2932	13	2.8652	1	3	0	22040	12.6	41.1	90.1	4.56	27.6	30.7	11.9	10.3	6.38	83	1	+
109	Shavakka	68	F	341	1597	92	7.9212	7	0.6027	0	1	0	8610	3.4	12.2	95.3	1.28	26.6	27.9	10	10.4	13.14	19	1	+
110	Sayada	66	M	4809	24739	78	10.257	18	2.367	0	4	0	13150	12.4	38.6	88.3	4.37	28.4	32.1	12.4	10.7	4.33	154	1	+
111	Mallappa	65	M	9508	48407	67	6.4856	27	2.6136	2	4	0	9680	13.7	41.2	89.8	4.59	29.8	33.3	12	10.2	2.48	151	1	+
112	Shankar	59	M	9608	49021	68	7.0244	25	2.5825	2	5	0	10330	12.3	38.1	82.3	4.63	26.6	32.3	11.3	10.4	2.72	36	1	+
113	Shivappa	76	M	9893	50604	72	9.1224	24	3.0408	2	2	0	12670	11.5	37.1	78.8	4.71	24.4	31	12.9	10.6	3.00	78	1	+
114	Ganga	54	F	9056	45944	88	11.968	9	1.224	1	2	0	13600	10.8	35.4	84.9	4.17	25.9	30.5	13.9	11.5	9.78	53	1	+
115	Shantabai	52	F	8409	44508	90	10.782	9	1.0782	0	1	0	11980	9.9	33.9	93.1	3.64	27.2	29.2	9.8	9.5	10.00		1	+
116	Shivanand	56	M	8829	45167	80	12.408	17	2.6367	1	2	0	15510	14.4	43.6	88.8	4.91	29.3	33	10.7	9.7	4.71	26	0	+

MASTER CHART - CONTROL

Sl No	Name	Age	Sex	OP/IP No.	Lab No.	N	ANC	L	ALC	M	E	B	WBC	Hb	HCT	MCV	RBC	MCH	MCHC	PDW	MPV	NLR	CK-MB	C.Trop.	ECG
1	Shivappa	40	M	22497	104273	52	3.26	39	2.44	5	4	0	6260	18.3	55.4	85.5	6.48	28.2	33	10.2	9.7	1.33			-
2	Kantappa	40	M	226991	98140	60	2.57	34	1.46	3	3	0	4290	12	37.3	79	4.72	25.4	32.2	15.5	11.4	1.76			-
3	Virendra	40	M	236168	103612	76	6.80	17	1.52	2	5	0	8950	12.9	38.6	78.8	4.90	26.3	33.4	9.4	9.3	4.47			-
4	Venkatesh	46	M	22633	104993	56	3.71	40	2.65	1	3	0	6620	15.2	42.5	86.7	4.90	31	35.8	13.7	11	1.40			-
5	Goudappagouda	44	M	237916	103576	60	3.81	32	2.03	2	6	0	6350	13.4	39.3	93.3	4.21	31.8	34.1	10.4	9.4	1.88			-
6	Shanubai	40	F	240309	104745	60	5.17	34	2.93	2	4	0	8610	13.2	40.1	85	4.72	28	32.9	10.6	9.7	1.76			-
7	Vidyawati	48	F	238037	103943	65	4.43	25	1.71	4	6	0	6820	12	36.1	83.4	4.33	27.7	33.2	13.3	11.1	2.60			-
8	Mahadevi	55	F	240516	104731	64	6.23	31	3.02	1	4	0	9730	13.8	41.8	84.1	4.97	27.8	33	9.9	9.4	2.06			-
9	Muregappa	52	M	22107	102547	55	5.51	34	3.41	7	4	0	10020	17	48.6	92.9	5.23	32	35	12.2	10.3	1.62			-
10	Shatagouda	55	M	22459	104041	58	4.37	38	2.86	2	2	0	7530	15.4	44.1	89.5	4.93	31.2	34.9	9	8.8	1.53			-
11	Shivamma	50	F	22615	104863	62	4.56	31	2.28	3	4	0	7360	11.5	35.5	86.4	4.11	28	32.4	9.5	9.1	2.00			-
12	Gangadhar	58	M	22551	104656	65	4.55	28	1.96	2	5	0	7000	13.2	40.6	86.4	4.70	28.1	32.5	9.1	9	2.32			-
13	Lalith	55	M	236483	102802	66	4.09	25	1.55	5	4	0	6190	15	44.7	89.9	4.97	30.2	33.6	10.7	9.4	2.64			-
14	Manohar	62	M	22303	103127	74	6.66	20	1.80	4	2	0	9000	9.4	25.5	90.1	2.83	33.2	36.9	12.8	9.8	3.70			-
15	Rudrappa	56	M	20413	101982	63	4.29	30	2.04	5	2	0	6810	12.8	43.2	75.3	5.74	22.3	29.6	13	10.5	2.10			-
16	Sanganagouda	64	M	236469	102796	65	4.02	29	1.79	1	5	0	6180	15.2	46.5	86	5.41	28.1	32.7	12.3	10.1	2.24			-
17	Bhimappa	64	M	240529	104787	60	2.39	34	1.35	6	0	0	3980	12.9	38.9	94.2	4.13	31.2	33.2	9.8	9.5	1.76			-
18	Siddappa	65	M	22390	103712	66	5.16	27	2.11	3	4	0	7820	14.3	41.2	99.5	4.14	34.5	34.7	8.8	8.9	2.44			-
19	Neelakanth	67	M	236787	103442	62	4.15	25	1.67	1	2	0	6690	11.1	34.8	81.3	4.28	25.9	31.9	10	9.6	2.48			-
20	Mahadevi	63	F	237789	103474	57	5.35	36	3.38	2	5	0	9380	10.8	33.6	76.2	4.41	24.5	32.1	9.3	8.9	1.58			-
21	Rukmini	66	F	23807	103400	60	4.90	34	2.77	2	4	0	8160	13.1	40.2	87.8	4.58	28.6	32.6	13.6	10.9	1.76			-
22	Nabilal	68	M	21245	97988	63	4.91	29	2.26	5	3	0	7800	13.1	37.2	90.5	4.11	31.9	35.2	16.7	11.3	2.17			-
23	Gurupadayya	70	M	22550	104716	62	2.64	34	1.45	1	3	0	4250	10.7	31.7	79.1	4.01	26.7	33.8	8.1	8.5	1.82			-
24	Jayappa	74	F	253625	126860	59	4.70	33	2.63	4	4	0	7970	12.2	40.4	85.2	4.74	25.7	30.2	10.8	10.1	1.79			-
25	Sonubai	86	F	22594	104811	68	5.11	26	1.95	2	4	0	7510	12.6	38.9	95.3	4.08	30.9	32.4	10.7	10	2.62			-
26	Mahadevappa	57	M	25535	119502	77	7.41	17	1.64	3	3	0	9620	12.5	38.4	89	4.31	30.8	32.6	12.5	10.6	4.53			-
27	Sharanappa	64	M	26111	122246	70	7.32	24	2.51	1	5	0	10450	12.1	35.5	83.5	4.25	28.5	34.1	12.5	10.7	2.92			-
28	Shrishail	56	M	26244	122862	60	6.30	35	3.68	1	4	0	10500	16	46.6	90.8	5.13	31.2	34.3	10.4	9.5	1.71			-
29	Danappa	57	M	26411	123565	77	7.19	17	1.59	0	6	0	9340	14.5	42.6	81.9	5.20	27.9	34	12.1	10.6	4.53			-
30	Shivappa	72	M	27783	129862	74	8.60	23	2.67	2	1	0	11620	14.4	43.4	89.9	4.83	29.8	33.2	9.1	9	3.22			-
31	Mahantapp	45	M	28213	131703	58	5.27	34	3.09	2	6	0	9090	14.2	46.1	91.8	5.02	25.4	30.8	10.7	9.3	1.71			-
32	Shankreppa	71	F	21489	107295	61	3.26	30	1.61	3	6	0	5350	12	38.1	97.2	3.92	30.6	31.5	10.4	9.8	2.03			-
33	Subhasgouda	60	M	29123	136024	64	6.18	31	2.99	1	4	0	9660	15.2	44.6	93.5	4.77	31.9	34.1	12.1	10.9	2.06			-
34	M P Naik	58	M	165167	80187	70	5.71	26	2.12	3	1	0	8160	14.4	44.3	79.2	5.59	25.8	32.5	9.8	9.1	2.69			-
35	Vittal	60	M	177711	86532	67	4.60	28	1.92	3	2	0	6860	13.4	37.3	94.4	3.95	33.9	35.9	8.6	9.1	2.39			-
36	Mallikarjun	75	M	260847	129519	59	2.95	35	1.75	2	4	0	5000	12.2	38.5	89.7	4.29	28.4	31.7	10.3	10.1	1.69			-
37	T Y Yadahalli	50	M	22981	110637	56	3.25	32	1.86	4	8	0	5800	15.4	46.4	89.9	5.16	29.8	33.2	8.8	8.6	1.75			-
38	Dashret	45	M	176493	85897	72	5.74	24	1.91	4	0	0	7970	15.9	46.7	101.3	4.61	34.5	34	10.6	9.7	3.00			-

40	Mallappa	45	M	255681	127512	54	3.53	38	2.49	3	5	0	6540	12.6	41.1	82.4	4.99	25.3	30.7	12.2	10.2	1.42			-
41	Shantgoud	60	M	18745	112241	60	4.53	31	2.34	3	6	0	7550	10.4	33.3	92.2	3.61	28.8	31.2	8.2	8.4	1.94			-
42	G R Kumbar	80	M	227293	112606	60	4.72	32	2.52	4	4	0	7860	12.5	39.5	89.2	4.43	28.2	31.6	10.1	9.5	1.88			-
43	Huvanna	65	M	225626	110909	53	3.14	38	2.25	4	5	0	5920	12.5	41.7	82.4	5.06	24.7	30	12.1	10.4	1.39			-
44	Shreeshail	40	M	20532	102585	63	5.34	30	2.54	4	3	0	8470	14	43.3	87.7	4.94	28.3	32.3	9.9	9.3	2.10			-
45	Chandramappa	68	M	3194	18110	63	4.96	32	2.52	4	1	0	7870	14.2	42.2	86.7	4.87	29.2	33.6	10.7	9.9	1.97			-
46	Shantabai	65	F	15848	81076	63	5.91	32	3.00	2	3	0	9380	12.3	39.2	12.3	31.87	30.8	31.4	10.9	9.9	1.97			-
47	Dhansing	67	M	22463	112765	67	5.08	27	2.05	4	2	0	7580	11.5	36.1	82	4.40	26.1	31.9	9.9	9.6	2.48			-
48	P A Biradar	38	M	224976	110629	52	3.81	46	3.37	1	1	0	7330	14.9	45.7	86.1	5.31	28.1	32.6	14.1	10.8	1.13			-
49	D R Bagawan	53	M	224980	110636	52	3.54	40	2.72	4	4	0	6810	12.9	40.1	81.7	4.91	26.3	32.2	12.5	10.5	1.30			-
50	Malakappa	45	M	16409	83461	72	4.98	25	1.73	3	0	0	6920	12.6	37	83.7	4.42	28.5	34.1	10.4	9.4	2.88			-
51	I G Badami	58	M	38792	18368	49	3.79	46	3.56	2	3	0	7740	12.8	42.3	79.2	5.34	24	30.3	11.4	9.9	1.07			-
52	Mohamad	78	M	255682	128023	59	3.97	35	2.36	2	4	0	6730	14.5	45.7	79.2	5.77	25.1	31.7	15.2	11.3	1.69			-
53	Mallappa	72	M	167183	81201	52	3.29	43	2.72	3	2	0	6330	14.3	44.7	94.3	4.74	30.2	32	14.2	11.6	1.21			-
54	Basalingavva	58	F	39426	18603	66	5.21	29	2.29	4	1	0	7900	12	39.5	88.4	4.47	26.8	30.4	11.3	10	2.28			-
55	Shushilendra	58	M	217256	106456	53	3.57	41	2.76	3	3	0	6730	13.7	43.4	90.2	4.81	28.5	31.6	11.8	10.6	1.29			-
56	Siddamma	40	F	228156	112402	69	5.38	28	2.18	2	1	0	7790	14.3	45.1	92.8	4.86	29.4	31.7	9.8	9.4	2.46			-
57	M S Nidoni	52	M	225172	110667	48	3.64	43	3.26	3	6	0	7580	14.3	43.2	88.3	4.89	29.2	33.1	9.2	8.9	1.12			-
58	Channamma	60	F	19881	102852	71	4.96	23	1.61	4	2	0	6990	11.3	35.5	96.7	3.67	30.8	31.8	11.5	10.5	3.09			-
59	Shivanand	60	M	255684	127906	59	4.05	36	2.47	2	3	0	6860	14.6	45.8	79.4	5.77	25.3	31.9	14.1	11.3	1.64			-
60	Ravindra	59	M	255685	128025	58	3.64	38	2.38	2	2	0	6270	12.7	40	91.5	4.37	29.1	31.8	10.8	10.2	1.53			-
61	Indumati	70	F	253677	126856	57	5.52	38	3.68	3	2	0	9680	12.8	40.4	83.6	4.83	26.5	31.7	11.5	10.2	1.50			-
62	Yaseen	60	M	253822	128236	64	4.24	31	2.05	2	3	0	6620	10.6	34.2	88.1	3.88	27.3	31	17.9	13.1	2.06			-
63	Shamrao	55	M	15775	80475	60	4.03	33	2.21	3	4	0	6710	12.4	37.3	107.8	3.46	35.8	33.2	9.1	9.3	1.82			-
64	K M Patil	77	M	236786	104548	72	4.51	23	1.44	1	4	0	6260	13.1	39.1	87.9	4.45	29.4	33.5	10.2	9.8	3.13			-
65	Sonubai	80	F	260860	129522	65	6.20	31	2.96	1	3	0	9540	12.9	38.6	97.7	3.95	32.7	33.4	11.1	10.2	2.10			-
66	Ramachandra	35	M	209687	102680	61	4.80	33	2.60	5	1	0	7870	14.1	43.3	89.8	4.82	29.3	32.6	9	8.7	1.85			-
67	Bagirathi	75	F	255695	127517	64	4.33	30	2.03	2	4	0	6760	10.1	32.2	79.3	4.06	24.9	31.4	9.2	9.2	2.13			-
68	Basavaraj	36	M	208490	102128	54	5.42	37	3.71	4	5	0	10030	15	45.3	87.8	5.16	29.1	33.1	9.3	9.1	1.46			-
69	Dashrat	75	M	255680	127511	55	3.58	39	2.54	2	4	0	6500	12.6	41.2	82.7	4.98	25.3	30.6	11.6	10.2	1.41			-
70	Shivappa	52	M	229177	112712	70	6.05	25	2.16	3	2	0	8640	14.6	45	91.1	4.94	29.6	32.4	12.2	10.4	2.80			-
71	Neelamma	74	F	253621	126863	58	4.78	33	2.72	4	5	0	8240	12.1	40.1	85	4.72	25.6	30.2	11	10.1	1.76			-
72	Prajavati	55	F	21517	107336	72	7.70	23	2.46	3	2	0	10700	11.6	36.2	93.1	3.89	29.8	32	10.4	9.9	3.13			-
73	Shivappa	52	M	229177	112712	70	6.05	25	2.16	3	2	0	8640	14.6	45	91.1	4.94	29.6	32.4	12.2	10.4	2.80			-
74	Channappa	64	M	260856	129521	61	3.29	32	1.73	3	4	0	5400	16.2	51.7	92	5.62	28.8	31.3	11.7	10.2	1.91			-
75	D S Janagound	65	M	166978	81042	59	5.89	34	3.39	3	4	0	9980	10.9	37.5	80.5	4.66	23.4	29.1	9.7	9.1	1.74			-
76	Ramappa	53	M	260850	129520	60	2.90	36	1.74	1	3	0	4830	12.2	38.3	89.9	4.26	28.6	31.9	11	10.1	1.67			-
77	Malleshappa	65	M	260861	129524	58	4.26	35	2.57	3	4	0	7340	11.3	37.1	90.3	4.11	27.5	30.5	17	12.7	1.66			-
78	Prathima	46	F	209679	102660	51	2.56	38	1.91	4	7	0	5020	10.7	36.3	88.3	4.11	26	29.5	9.2	9.3	1.34			-
79	Mallamma	62	F	177716	86538	67	4.91	26	1.91	4	3	0	7330	12.6	38.8	89.2	4.35	29	32.5	8.5	8.7	2.58			-
80	Ganga	85	F	255693	127515	63	4.14	31	2.04	1	5	0	6570	10.2	32.2	79.3	4.06	24.9	31.4	9.3	9.1	2.03			-
81	Madiwalamma	60	F	225525	110875	56	3.85	38	2.61	4	2	0	6880	12	38.4	85.1	4.51	26.6	31.3	11.2	10	1.47			-
82	M S Patil	43	M	217111	106890	72	4.64	25	1.61	3	0	0	6450	16.1	50.6	96.7	5.23	30.8	31.8	10.5	10.1	2.88			-

84	Aravind	46	M	177055	86117	64	6.64	30	3.11	2	4	0	10380	13.7	41.6	79.7	5.22	26.2	32.9	10	9.4	2.13			-
85	Mohan	48	M	177706	86531	56	4.92	38	3.34	3	3	0	8790	14.3	44.2	83.7	5.28	27.1	32.4	10.4	9.4	1.47			-
86	Shekugouda	55	M	229194	112820	67	6.32	25	2.36	3	5	0	9440	15.2	46.8	100.6	4.65	32.7	32.5	9.7	9	2.68			-
87	Shantabai	55	F	165006	80230	66	5.15	30	2.34	2	2	0	7800	11.8	38.3	81.8	4.68	25.2	30.8	10.2	9.6	2.20			-
88	Wasim	65	M	253543	126473	54	4.47	38	3.15	3	5	0	8280	13.6	41.4	92	4.50	30.2	32.9	11	9.7	1.42			-
89	Parashuram	30	M	21503	107297	67	4.35	25	1.62	4	4	0	6490	14.3	44.1	87.3	5.05	28.3	32.4	10.1	9.6	2.68			-
90	Alu	45	M	3536	17946	53	4.44	37	3.10	3	7	0	8380	14.4	43.3	80.8	5.36	26.9	33.3	12.4	10.5	1.43			-
91	B M Ramprasad	54	M	209677	102661	64	4.51	28	1.97	5	3	0	7040	14.6	45.2	89.9	5.03	29	32.3	14.2	11	2.29			-
92	Deepak	52	M	171423	83240	63	4.36	29	2.01	5	3	0	6920	11.8	35.9	83.7	4.29	27.5	32.9	14.3	11.3	2.17			-
93	Ganapati	42	M	171695	83458	49	3.68	40	3.00	4	7	0	7500	15.3	45.8	94.4	4.85	31.5	33.4	15	11.4	1.23			-
94	Malappa	72	M	167183	81201	52	3.29	43	2.72	3	2	0	6330	14.3	44.7	94.3	4.74	30.2	32	14.2	11.6	1.21			-
95	H M Nadaf	48	M	224983	110640	58	5.35	38	3.50	2	2	0	9220	14	42.4	85.8	4.94	28.3	33	9.2	9	1.53			-
96	Sangappa	71	M	253583	128868	67	5.25	29	2.27	1	3	0	7830	9.8	28.6	76.7	3.73	23.1	30.1	10.7	10.1	2.31			-
97	Shantawwa	52	F	217880	106858	60	3.10	32	1.65	3	5	0	5170	13.2	41.7	88.2	4.73	27.9	31.7	9.5	9.2	1.88			-
98	L D Bajantri	42	M	224985	110634	62	6.08	34	3.34	3	1	0	9810	13.3	39.3	88.7	4.43	30	33.8	8.2	8.3	1.82			-
99	Subash	70	M	22248	111138	72	5.33	23	1.70	3	2	0	7400	14	43.3	90.8	4.77	29.4	32.3	11.3	9.9	3.13			-
100	Shantabai	65	F	15848	81076	63	5.91	32	3.00	2	3	0	9380	12.3	39.2	98	4.00	30.8	31.4	10.9	9.9	1.97			-
101	Shrisail	40	M	218715	107250	54	4.56	34	2.87	4	8	0	8440	15.7	46.5	90.5	5.14	30.5	33.8	10.9	9.7	1.59			-
102	Subhas	50	M	15953	81223	56	3.04	35	1.90	4	5	0	5430	12.3	39.4	81.2	4.85	25.4	31.2	12.6	10.7	1.60			-
103	Fathima	64	F	253620	128869	50	3.14	42	2.63	3	5	0	6270	11.6	36.2	87.2	4.15	28	32	12.7	11.1	1.19			-
104	Basamma	50	F	209296	102380	68	7.10	28	2.92	1	3	0	10440	12.5	39.9	84	4.75	26.3	31.3	10.8	9.7	2.43			-
105	Gollalappa	55	M	16491	83638	61	3.95	35	2.27	4	0	0	6480	11.3	36.3	89	4.08	27.7	31.1	9.3	9.3	1.74			-
106	Vimalabai	59	F	254562	126427	65	5.10	28	2.20	3	4	0	7850	9.6	28.2	76.8	3.67	23.2	30.1	10.8	10.1	2.32			-
107	Rachappa	55	M	260866	129525	65	6.40	27	2.66	4	4	0	9840	11.2	34.8	79.6	4.37	25.6	32.2	9.9	9.4	2.41			-
108	Khema	70	M	228674	112482	62	4.45	30	2.15	4	4	0	7180	14.4	45.5	90.6	5.02	28.7	31.6	10.3	9.4	2.07			-
109	Kashibai	68	F	218417	107107	50	3.18	40	2.54	3	7	0	6360	10.8	34.7	89.4	3.88	27.8	31.1	9.7	9.5	1.25			-
110	Subash	70	M	22248	111138	72	5.33	23	1.70	3	2	0	7400	14	43.3	90.8	4.77	29.4	32.3	11.3	9.9	3.13			-
111	Appasaheb	60	M	255690	128232	58	3.67	36	2.28	2	4	0	6330	12.6	39.8	91.5	4.35	29	31.7	10.1	9.7	1.61			-
112	Shankar	59	M	9608	49021	56	3.84	35	2.40	2	7	0	6860	15.1	46.9	89	5.27	28.7	32.2	11.3	9.7	1.60			-
113	Shivappa	80	M	3647	18291	69	5.77	24	2.01	4	3	0	8360	14.1	43.9	90.3	4.86	29	32.1	12.1	9.9	2.88			-
114	Basalingavva	58	F	39426	18603	66	5.21	29	2.29	4	1	0	7900	12	39.5	88.4	4.47	26.8	30.4	11.3	10	2.28			-
115	S H Malghan	52	F	177199	86560	54	3.13	38	2.20	2	6	0	5790	14.5	45.1	86.9	5.19	27.9	32.2	11.8	10.5	1.42			-
116	Basu	58	M	253781	126854	65	5.01	28	2.16	3	4	0	7710	15.6	47.8	82.6	5.79	26.9	32.6	19.2	13.1	2.32			-