**DISSERTATION ON**

# A STUDY ON MEAN PLATELET VOLUME IN ACUTE CORONARY SYNDROME

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# CERTIFICATE

This is to certify that the dissertation entitled **"A STUDY ON MEAN PLATELET VOLUME IN ACUTE CORONARY SYNDROME"** is a bonafide work done by **DR.S.VENKATESAN,** Post Graduate Student, Institute of Internal Medicine, Madras Medical College, Chennai-3, in partial fulfillment of the University Rules and Regulations for the award of MD Branch – I Internal Medicine, under our guidance and supervision, during the academic year 2009 - 2012.

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# DECLARATION

I solemnly declare that the dissertation entitled "**A STUDY ON MEAN PLATELET VOLUME IN ACUTE CORONARY SYNDROME**" is done by me at Madras Medical College, Chennai-3 during May 2011 to November 2011 under the guidance and supervision of Prof. C. RAJENDIRAN, M.D., to be submitted to The Tamilnadu Dr M.G.R Medical University towards the partial fulfillment of requirements for the award of M.D DEGREE IN GENERAL MEDICINE BRANCH-I.

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# ABBREVIATIONS

| | | |
|---|---|---|
| CAD | - | Coronary artery disease |
| AP | - | Angina pectoris |
| ACS | - | Acute coronary syndrome |
| AMI | - | Acute myocardial infarction |
| UA | - | Unstable angina |
| STEMI | - | ST elevation MI |
| NSTEMI | - | Non ST elevation MI |
| HT | - | Systemic Hypertension |
| DM | - | Diabetes mellitus |
| WBC | - | White blood cell |
| TC | - | Total count |
| P | - | Polymorphs |
| L | - | Lymphocytes |
| E | - | Eosinophils |

TB - Total bilirubin

DB - Direct bilirubin

SGOT - Aspartate transferase

SGPT - Alanine transferase

MPV - Mean platelet volume

PDW - Platelet distribution width

P-LCR - Platelet – Large cell ratio

TC - Total cholesterol

TGL - Triglycerides

HDL - High density cholesterol

# CONTENTS


| S.NO | TITLE | PAGE NO |
|---|---|---|
| 1. | INTRODUCTION | 1 |
| 2. | AIMS AND OBJECTIVES | 3 |
| 3. | REVIEW OF LITERATURE | 4 |
| 4. | MATERIALS AND METHODS | 32 |
| 5. | OBSERVATION AND RESULTS | 36 |
| 6. | DISCUSSION | 64 |
| 7. | LIMITATIONS OF STUDY | 72 |
| 8. | CONCLUSION | 73 |
| 9. | BIBLIOGRAPHY | |
| 10. | ANNEXURES<br>• PROFORMA<br>• MASTER CHART<br>• ETHICAL COMMITTEE APPROVAL ORDER | |

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# INTRODUCTION

Acute coronary syndrome is a very important cause of morbidity and mortality despite the recent advances in its diagnosis and treatment.

Coronary heart disease may manifest as silent myocardial ischemia, stable angina pectoris (AP), unstable AP, myocardial infarction (STEMI and NSTEMI), heart failure, and sudden cardiac death.

It is important to identify the risk factors in coronary heart disease as they have a very important place in the prevention of acute coronary syndromes, and also in the follow-up and treatment of patients with coronary heart disease.

Platelets play a critical role in the formation of thrombus on the ruptured plaque and subsequent progression to myocardial infarction. Hence, acetyl salicylic acid, thienopyridine, and glycoprotein IIb/IIIa inhibitors, which all inhibit platelet functions, are used in the treatment of ACS.

Platelets are heterogeneous in size, density, and activity. Alterations of these parameters may be associated with pulling the

trigger of acute coronary syndrome and its spread. Large platelets are more adhesive and tend to aggregate more than smaller ones. Increase of platelet volume may contribute to increased prothrombotic tendency of atherosclerotic plaque in acute coronary syndrome and increased risk of intracoronary thrombus formation in AMI cases.

In this study, we aimed to investigate the significance of mean platelet volume and acute coronary syndrome, in comparison with control and stable AP group.

# AIMS AND OBJECTIVES OF THE STUDY

1) To study the significance of mean platelet volume and acute coronary syndrome.
2) To compare mean platelet volume in ACS, stable angina pectoris and healthy controls.

# REVIEW OF LITERATURE

CORONARY ARTERY DISEASE is a major cause of morbidity and mortality in any part of world. The better known risk factors are

1) Age

2) Family history

3) Cigarette smoking

4) Elevated LDL cholesterol

5) Hypertension

6) Diabetes mellitus which is accepted to be a coronary heart disease equivalent.

Apart from these,

a. Lipoprotein (a)

b. Endothelial dysfunction

c. Homocysteine

d. C Reactive protein are considered as new risk factors of coronary heart disease.

These risk factors account for only a part of ACS cases. Hence, it is essential to identify other related risk factors to predict individual risk in the development of ACS.

# ACUTE MYOCARDIAL INFARCTION[1]

***Aspects of Diagnosis of Myocardial Infarction by Different Techniques***

| TECHNIQUE | FEATURES |
|---|---|
| Pathology | Myocardial cell death |
| Biochemistry | Markers of myocardial cell death recovered from blood samples |
| Electrocardiography | Evidence of myocardial ischemia (ST and T wave abnormalities); evidence of loss of electrically functioning cardiac tissue (Q waves) |
| Imaging | Reduction or loss of tissue perfusion; cardiac wall motion abnormalities |

## REVISED DEFINITION OF MYOCARDIAL INFARCTION[1]

***Criteria for Acute, Evolving, or Recent MI***

Either of the following criteria satisfies the diagnosis for acute, evolving, or recent MI:

1) Typical rise and/or fall of biochemical markers of myocardial necrosis with at least one of the following:

   a. Ischemic symptoms

   b. Development of pathologic Q waves in the ECG

c. Electrocardiographic changes indicative of ischemia (ST-segment elevation or depression)

d. Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality

2) Pathologic findings of an acute myocardial infarction

## CRITERIA FOR HEALING OR HEALED MYOCARDIAL INFARCTION[1]

Any one of the following criteria satisfies the diagnosis for healing or healed myocardial infarction:

1) Development of new pathologic Q waves in serial ECGs. The patient may or may not remember previous symptoms. Biochemical markers of myocardial necrosis may have normalized, depending on the length of time that has passed since the infarction developed.

2) Pathologic findings of a healed or healing infarction

## CLASSIFICATION OF MYOCARDIAL INFARCTION[1]

| TYPE | FEATURES |
|---|---|
| 1 | Spontaneous myocardial infarction related to ischemia caused by a primary coronary event such as plaque erosion and/or rupture, fissuring, or dissection |

| TYPE | FEATURES |
|---|---|
| 2 | Myocardial infarction secondary to ischemia caused by increased oxygen demand or decreased supply (e.g., coronary artery spasm, coronary embolism, anemia, arrhythmias, hypertension, hypotension) |
| 3 | Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischemia, accompanied by presumably new ST-segment elevation, or new LBBB, or presumably new major obstruction in a coronary artery by angiography and/or pathology, but death occurring before blood samples could be obtained, or before the appearance of cardiac biomarkers in the blood |
| 4a | Myocardial infarction associated with PCI |
| 4b | Myocardial infarction associated with stent thrombosis, as documented by angiography or autopsy |
| 5 | Myocardial infarction associated with CABG |

CABG = coronary artery bypass grafting; LBBB = left bundle branch block.

## PATHOLOGY OF AMI[2]

STEMI usually occurs when coronary blood flow decreases abruptly after a thrombotic occlusion of a coronary artery previously affected by atherosclerosis. A slowly developing, high-grade coronary artery stenosis does not typically precipitate STEMI because of the development of a rich collateral network over time.

Instead, STEMI occurs when a coronary artery thrombus develops rapidly at a site of vascular injury. This injury is produced or facilitated by factors such as cigarette smoking, hypertension, and lipid accumulation[2].

STEMI occurs when the surface of an atherosclerotic plaque becomes disrupted (exposing its contents to the blood) and conditions (local or systemic) favor thrombogenesis. A mural thrombus forms at the site of plaque disruption, and the involved coronary artery becomes occluded. Histologic studies indicate that the coronary plaques prone to disruption are those with a rich lipid core and a thin fibrous cap[2].

After an initial platelet monolayer forms at the site of the disrupted plaque, various agonists (collagen, ADP, epinephrine, serotonin) promote platelet activation. After agonist stimulation of platelets, thromboxane $A_2$ (a potent local vasoconstrictor) is released, further platelet activation occurs, and potential resistance to fibrinolysis develops[2].

In addition to the generation of thromboxane $A_2$, activation of platelets by agonists promotes a conformational change in the glycoprotein IIb/IIIa receptor. Once it is converted to its functional

state, this receptor develops a high affinity for soluble adhesive proteins (integrins) such as fibrinogen. Since fibrinogen is a multivalent molecule, it can bind to two different platelets simultaneously, resulting in platelet cross-linking and aggregation[2].

The coagulation cascade is activated on exposure of tissue factor in damaged endothelial cells at the site of the disrupted plaque. Factors VII and X are activated, ultimately leading to the conversion of prothrombin to thrombin, which then converts fibrinogen to fibrin. Fluid-phase and clot-bound thrombin participate in an auto amplification reaction leading to further activation of the coagulation cascade. The culprit coronary artery eventually becomes occluded by a thrombus containing platelet aggregates and fibrin strands.[2]

# DEFINITION[1]

STABLE angina pectoris typically manifests as a deep, poorly localized chest or arm discomfort (rarely described as pain), reproducibly precipitated by physical exertion or emotional stress, and relieved within 5 to 15 minutes by rest or sublingual nitroglycerin.

UNSTABLE angina is defined as angina pectoris (or equivalent type of ischemic discomfort) with at least one of three features:

1) Occurring at rest (or minimal exertion) and usually lasting >20 minutes (if not interrupted by the administration of a nitrate or an analgesic)

2) Being severe and usually described as frank pain and of new onset (within 1 month).

3) Occurring with a crescendo pattern (i.e., pain that awakens the patient from sleep or that is more severe, prolonged, or frequent than previously).

Approximately two thirds of patients with unstable angina

have evidence of myocardial necrosis on the basis of elevated cardiac serum markers, such as cardiac-specific troponin T or I and creatine kinase isoenzyme (CK)–MB, and thus have a diagnosis of NSTEMI. As troponin measurements become progressively more sensitive, an increasing fraction of patients with NSTE-ACS exhibit some release of troponin, and therefore these should be considered cases of NSTEMI with a reciprocal reduction in the fraction with unstable angina.

**BRAUNWALD CLINICAL CLASSIFICATION OF UA/NSTEMI[1]**

| CLASS | DEFINITION |
|---|---|
| **Severity** | |
| Class I | New onset of severe angina or accelerated angina; no rest pain |
| Class II | Angina at rest within past month but not within preceding 48 hr (angina at rest, subacute) |
| Class III | Angina at rest within 48 hr (angina at rest) |
| **Clinical Circumstances** | |
| A) Secondary angina | Develops in the presence of extra cardiac condition that intensifies myocardial ischemia |
| B) Primary angina | Develops in the absence of extra cardiac condition |
| C) Post infarction angina | Develops within 2 wk after acute myocardial infarction |

| CLASS | DEFINITION |
|---|---|
| Intensity of treatment | Patients with unstable angina may also be divided into three groups according to whether unstable angina occurs: (1) in the absence of treatment for chronic stable angina, (2) during treatment for chronic stable angina, or (3) despite maximal anti-ischemic drug therapy. The three groups may be designated by subscripts 1, 2, and 3, respectively. |
| Electrocardiographic changes | Patients with unstable angina may be further divided into those with or without transient ST-T wave changes during pain. |

UA/NSTEMI = unstable angina/non–ST elevation myocardial infarction.

## PATHOLOGY OF UNSTABLE ANGINA/NSTEMI[2]

UA/NSTEMI is most commonly caused by a reduction in oxygen supply and/or by an increase in myocardial oxygen demand which is superimposed on a lesion that causes coronary arterial obstruction, usually an atherothrombotic coronary plaque.

Four pathophysiologic processes that may contribute to the development of UA/NSTEMI have been identified[2]:

1) Plaque rupture or erosion with a superimposed nonocclusive thrombus, believed to be the most common cause; in such patients,

NSTEMI may occur with downstream embolization of platelet aggregates and/or atherosclerotic debris

2) Dynamic obstruction e.g., coronary spasm, as in Prinzmetal's variant angina (PVA)

3) Progressive mechanical obstruction e.g., rapidly advancing coronary atherosclerosis or restenosis following percutaneous coronary intervention (PCI)

4) UA secondary to increased myocardial oxygen demand and/or decreased supply e.g., tachycardia, anemia

More than one of these processes may be involved.[2]

# CARDIAC BIOMARKERS

The ACC and AHA guidelines recommend cTnI or cTnT as the preferred first-line markers, but CK-MB (by mass assay) is an acceptable alternative. The preference for cardiac troponins reflects the greater specificity of these markers compared with CK-MB and the prognostic value of troponin elevations in the presence of normal CK-MB levels. If the initial set of markers is negative in patients who have presented within the first 6 hours of the onset of pain, the guidelines recommend that another sample be drawn in the time frame of 8 to 12 hours after symptom onset.

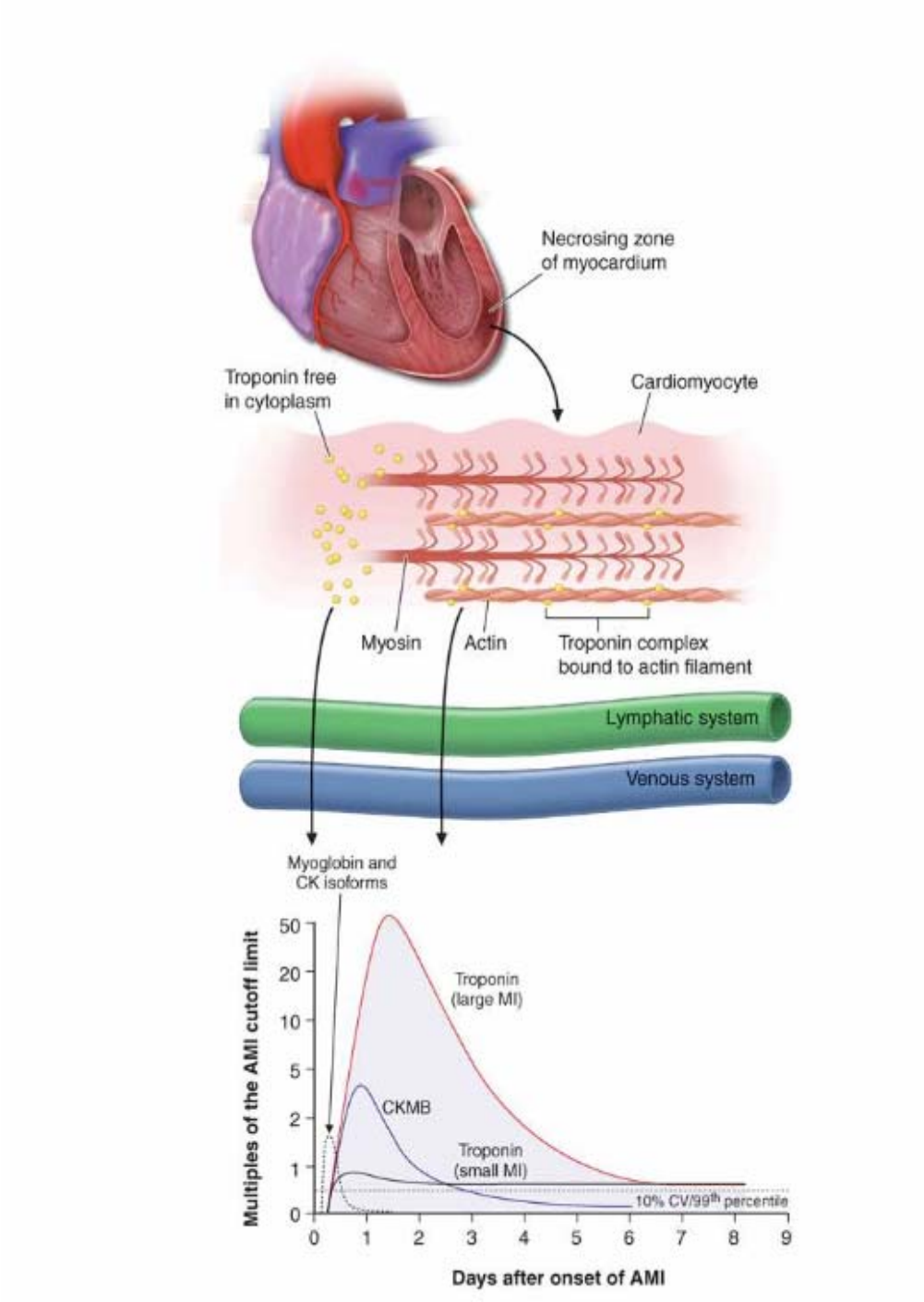
Cardiac-specific Troponin T (cTnT) and Cardiac specific Troponin I (cTnI) have amino-acid sequences different from those of the skeletal muscle forms of these proteins. These differences permitted the development of quantitative assays for cTnT and cTnI with highly specific monoclonal antibodies. Since cTnT and cTnI are not normally detectable in the blood of healthy individuals but may increase after STEMI to levels >20 times higher than the upper reference limit (the highest value seen in 99% of a reference population not suffering from MI), the measurement of cTnT or cTnI is of considerable diagnostic usefulness, and they are now the

preferred biochemical markers for MI. The cardiac troponins are particularly valuable when there is clinical suspicion of either skeletal muscle injury or a small MI that may be below the detection limit for creatine phosphokinase (CK) and its MB isoenzyme (CKMB) measurements, and they are, therefore, of particular value in distinguishing UA from NSTEMI. Levels of cTnI and cTnT may remain elevated for 7–10 days after STEMI.[2]

CK rises within 4–8 h and generally returns to normal by 48–72 h. An important drawback of total CK measurement is its lack of specificity for STEMI, as CK may be elevated with skeletal muscle disease or trauma, including intramuscular injection. The MB isoenzyme of CK has the advantage over total CK that it is not present in significant concentrations in extra cardiac tissue and, therefore, is considerably more specific. However, cardiac surgery, myocarditis, and electrical cardioversion often result in elevated serum levels of the MB isoenzyme. A ratio (relative index) of

CKMB mass: CK activity 2.5 suggests but is not diagnostic of a myocardial rather than a skeletal muscle source for the CKMB elevation.[2]

## BIOMARKERS IN ACS[2]



Many hospitals are using cTnT or cTnI rather than CKMB as the routine serum cardiac marker for diagnosis of STEMI, although any of these analytes remain clinically acceptable. It is not cost-effective to measure both a cardiac-specific troponin and CKMB at all time points in every patient.

While it has long been recognized that the total quantity of protein released correlates with the size of the infarct, the peak protein concentration correlates only weakly with infarct size. Recanalization of a coronary artery occlusion (either spontaneously or by mechanical or pharmacologic means) in the early hours of STEMI causes earlier peaking of biomarker measurements because of a rapid washout from the interstitium of the infarct zone, quickly overwhelming lymphatic clearance of the proteins.

The non-specific reaction to myocardial injury is associated with polymorphonuclear leukocytosis, which appears within a few hours after the onset of pain and persists for 3–7 days; the white blood cell count often reaches levels of 12,000–15,000/dL. The erythrocyte sedimentation rate rises more slowly than the white blood cell count, peaking during the first week and sometimes remaining elevated for one or two weeks[2].

Serum myoglobin and heart-type fatty acid binding protein (H-FABP) are smaller molecules and diffuse through interstitial fluids more rapidly after cell death than the larger CK and troponin molecules; they become abnormal as early as 30 minutes after myocardial injury. Because neither is specific to myocardial tissue, however, false-positive rates in Emergency Department populations are high.

Ischemia-modified albumin (IMA) has been approved by the U.S. Food and Drug Administration for clinical use. The albumin cobalt binding test for the detection of IMA is based on the observation that the affinity of the N-terminus of human albumin for cobalt is reduced in patients with myocardial ischemia. As with the other markers, however, the clinical specificity of IMA in the broad population of patients with chest pain and suspected ACS remains an area for further investigation.

B-type natriuretic peptides (BNP and N-terminal pro-BNP [NT-proBNP]) arise in the setting of increased ventricular wall stress. Natriuretic peptides are most commonly used to aid in the diagnosis of heart failure. BNP levels can be elevated in the setting of transient myocardial ischemia, and the magnitude of elevation in

ACS is correlated with prognosis. However, the lack of specificity of natriuretic peptide elevation for ACS limits its use as a diagnostic marker.

Many patients presenting with ACS, including those without evidence of myocyte necrosis, have elevated concentrations of inflammatory biomarkers such as C-reactive protein, serum amyloid A, myeloperoxidase, or interleukin-6 (IL-6). To date, no study has identified exact decision cut points or shown an incremental benefit on an admission or treatment strategy based on these new markers, so the clinical usefulness of these observations remains uncertain.

# LIPIDS AND MI

During the first 24 to 48 hours after admission, total cholesterol and high-density lipoprotein (HDL) cholesterol remain at or near baseline values but generally fall precipitously after that. The fall in HDL cholesterol after STEMI is greater than the fall in total cholesterol; thus, the ratio of total cholesterol to HDL cholesterol is no longer useful for risk assessment unless measured early after MI. A lipid profile should be obtained on all STEMI patients who are admitted within 24 to 48 hours of symptoms. The success of lipid-lowering therapy in primary and secondary prevention studies and evidence that hypolipidemic therapy improves endothelial function and inhibits thrombus formation indicate that early management of serum lipids in patients hospitalized for STEMI is advisable. For patients admitted beyond 24 to 48 hours, more accurate determinations of serum lipid levels are obtained approximately 8 weeks after the infarction has occurred.[2]

# PLATELETS[3]

Platelets are disc-shaped, anucleate cell fragments that are shed from megakaryocytes in the bone marrow into the blood stream. They play a critical role in normal hemostasis, by forming the hemostatic plug that initially seals vascular defects, and by providing a surface that recruits and concentrates activated coagulation factors. Their function depends on several glycoprotein receptors, a contractile cytoskeleton, and two types of cytoplasmic granules. α-*Granules* have the adhesion molecule P-selectin on their membranes and contain fibrinogen, fibronectin, factors V and VIII, platelet factor 4 (a heparin-binding chemokine), platelet-derived growth factor (PDGF), and transforming growth factor-β (TGF-β). *Dense (or δ) granules* contain ADP and ATP, ionized calcium, histamine, serotonin, and epinephrine[3].

After vascular injury, platelets encounter Extra cellular matrix constituents such as collagen and the adhesive glycoprotein vWF. On contact with these proteins, platelets undergo:

1) Adhesion and shape change,

2) Secretion (release reaction), and

3) aggregation

Platelet adhesion to Extra cellular matrix is mediated largely via interactions with vWF, which acts as a bridge between platelet surface receptors (e.g., glycoprotein Ib [GpIb]) and exposed collagen. Although platelets can also adhere to other components of the ECM (e.g., fibronectin), vWF-GpIb associations are necessary to overcome the high shear forces of flowing blood. Reflecting the importance of these interactions, genetic deficiencies of vWF or its receptor (Bernard-Soulier syndrome) result in bleeding disorders[3].

Secretion (release reaction) of both granule types occurs soon after adhesion. Various agonists can bind platelet surface receptors and initiate an intracellular protein phosphorylation cascade ultimately leading to degranulation. Release of the contents of dense-bodies is especially important, since calcium is required in the coagulation cascade, and ADP is a potent activator of platelet aggregation. ADP also begets additional ADP release, amplifying the aggregation process. Finally, platelet activation leads to the appearance of negatively charged phospholipids (particularly phosphatidylserine) on their surfaces. These phospholipids bind calcium and serve as critical nucleation sites for the assembly of complexes containing the various coagulation factors[3].

Platelet aggregation follows adhesion and granule release. In addition to ADP, the vasoconstrictor thromboxane $A_2$ is an important platelet-derived stimulus that amplifies platelet aggregation, which leads to the formation of the primary hemostatic plug. Although this initial wave of aggregation is reversible, concurrent activation of the coagulation cascade generates thrombin, which stabilizes the platelet plug via two mechanisms[3].

First, thrombin binds to a protease-activated receptor on the platelet membrane and in concert with ADP and $TxA_2$ causes further platelet aggregation. This is followed by platelet activation, an event that is dependent on the platelet cytoskeleton that creates an irreversibly fused mass of platelets, which constitutes the definitive secondary hemostatic plug[3].

Second, thrombin converts fibrinogen to fibrin in the vicinity of the platelet plug, functionally cementing the platelets in place.

The interplay of platelets and endothelium has a profound impact on clot formation. The endothelial cell-derived prostaglandin $PGI_2$ (prostacyclin) inhibits platelet aggregation and is a potent vasodilator; conversely, the platelet-derived prostaglandin $TxA_2$ activates platelet aggregation and is a

vasoconstrictor. Effects mediated by $PGI_2$ and $TxA_2$ are exquisitely balanced to effectively modulate platelet and vascular wall function: at baseline, platelet aggregation is prevented, whereas endothelial injury promotes hemostatic plug formation. The clinical utility of aspirin (an irreversible cyclooxygenase inhibitor) in persons at risk for coronary thrombosis resides in its ability to permanently block platelet $TxA_2$ synthesis. Although endothelial $PGI_2$ production is also inhibited by aspirin, endothelial cells can resynthesize active cyclooxygenase and thereby overcome the blockade. In a manner similar to $PGI_2$, endothelial-derived nitric oxide also acts as a vasodilator and inhibitor of platelet aggregation[3].

# MEAN PLATELET VOLUME

Platelets are heterogeneous in their size, density, and functional activity[19]. Alterations in these parameters may result as pulling the trigger of acute coronary syndrome and its spread[20] .

As atherosclerotic plaque rupture begins the thrombogenic phenomenon in ACS, the functional activity of circulating platelets plays vital role in the progression of thrombus formation[21].

Large platelets are more adhesive and tend to aggregate more than smaller platelets as assessed by in vitro aggregometry. They also exhibit increased levels of procoagulatory surface proteins such as P-selectin and glycoprotein IIIa. Increase in platelet volume may contribute to increased prothrombotic tendency of atherosclerotic plaque in acute coronary syndrome and increased risk burden of intracoronary thrombus formation in Acute myocardial infarction cases[5].

Platelet volume is an important indicator of platelet function and activation. Larger platelets have more secretory granules and mitochondria and are more active than the small platelets. As they lead to the formation and dissemination of intracoronary thrombus,

larger and hyperactive platelets accelerate the emergence of acute coronary syndrome.

It has been suggested that increased platelet volume measured after MI might be a determinant factor for future ischemic episodes and outcome.

Because MPV is a simple and inexpensive laboratory measurement, it might be considered a useful rule-out test along with other conventional cardiac biomarkers for the risk stratification of ACS patients admitted to the emergency departments.

A study showed MPV had great diurnal and nocturnal variation that can be attributed to alterations in the autonomic nervous system[46].

It has been show that there is a correlation between sympathetic activity and MPV in AMI patients. It can be explained by the effects of the adrenergic system on peripheral platelet activation and thrombopoiesis in bone marrow.

The effects of the adrenergic system occurs in two ways in the peripheral circulation.

1) Platelet activation via alpha$_2$-adrenoreceptor activation changes the shape and hence increases MPV.

2) Larger, activated platelets which are sequestered in the spleen can be released into the circulation following exercise or following administration of adrenaline, and leads to the increase in MPV following physical effort[46].

It has been shown that admission MPV in AMI patients is an independent factor for impaired reperfusion and related mortality. MPV measurement is an easy and feasible way to detect high-risk patients who need different approaches and treatments.

## MEAN PLATELET VOLUME[45]

**The mean platelet volume result** is calculated and produced by an automated analyzer, and is a calculation of the average size of platelets, whereas MCV is a calculation of the average size of individual red blood cells.

Normal Range7.5-11.5 fl

## If MPV is reduced

It implies the platelets are smaller than normal. They are known as Micro thrombocytes[45].

## CAUSES:-

**1. Aplastic Anemia -**

An acquired disorder in which the bone marrow stops producing new blood cells.

**2. Wiskott-Aldrich Syndrome -**

An inherited immune deficiency disease.

**3. Thrombocytopenia-absent radii (TAR Syndrome) -**

A rare inherited disorder in which there are low platelets and absence of the radius bones in the forearms.

**4. Storage Pool Disease -**

A mild bleeding disorder that causes bruising.

**If MPV is increased**

It implies the platelets are larger than normal. They are known as Macro thrombocytes[45].

**CAUSES:-**

**1. Idiopathic Thrombocytopenic Purpura -**

A bleeding disorder in which the blood does not clot.

**2. Bernard-Soulier Disease -**

A congenital disorder where the platelets lack receptors to adhere to the walls of the blood vessels.

**3. May-Hegglin Anomaly -**

A rare, inherited disorder characterized by abnormally large platelets.

**4 Greater risk of heart attacks and stroke-**

As there are increased numbers of large, hyperaggregable platelets there is increased risk of ischemic events.

MPV is known to be increased in patients at the time of admission for MI, and it is postulated that alterations in the entire megakaryocyte-platelet-hemostatic axis precede MI.

MPV increases before MI for three reasons[46]:

1) The life span of platelets is approximately eight days, and the increase in MPV is seen within the first 12 h of admission
2) The increase in MPV persists six weeks after discharge when the infarct would be largely healed
3) Log normality of platelet volume is preserved.

Recent studies have revealed the in vitro data on the therapeutic effects on platelet size of Losartan, an angiotensin II receptor antagonist, or Doxazosin, an alpha1-adrenoreceptor antagonist. These observations have not yet been confirmed. In a small study with 30 patients, found platelet aggregation inhibitors have no effect on MPV values[5].

# MATERIALS AND METHODS

## SETTING

This study is conducted in Cardiology department (CCU & OP) and Medicine department of Madras medical college in collaboration with Department of Pathology and Biochemistry.

## ETHICAL APPROVAL

Obtained.

## STUDY DURATION

This study was conducted over a period of 7 months from may 2011 to November 2011.

## STUDY POPULATION

Patients admitted with acute coronary syndromes in coronary care unit and medical wards, stable angina pectoris patients followed in cardiology op and medical op, and healthy controls in medical wards and medicine op in Rajiv Gandhi Govt General Hospital.

## CASE DEFINITION

***Acute myocardial infarction:***

Ischemic symptoms

Development of pathologic Q waves in the ECG

Electrocardiographic changes indicative of ischemia (ST-segment elevation or depression)

Imaging evidence of new regional wall motion abnormality

***Unstable angina:***

Angina pectoris (or equivalent type of ischemic discomfort) with at least one of three features:

1) Occurring at rest (or minimal exertion) and usually lasting >20 minutes (if not interrupted by the nitroglycerin administration.)

2) Being severe and usually described as frank pain, and of new onset (within 1 month).

3) Occurring with a crescendo pattern (i.e., pain that awakens the patient from sleep or that is more severe, prolonged, or frequent than previously).

***Stable angina pectoris:***

Deep, poorly localized chest or arm discomfort (rarely described as pain), reproducibly precipitated by physical exertion or emotional stress, and relieved within 5 to 15 minutes by rest or sublingual nitroglycerin in whom ECG may be normal or show ischemic changes.

## SAMPLING

Patients are allotted by convenience sampling in each group.

## TYPE OF STUDY

It is a hospital based cross sectional study.

## INCLUSION CRITERIA

Patients with

1. Acute myocardial infarction
2. Unstable angina
3. Stable angina pectoris
4. Healthy controls

## EXCLUSION CRITERIA

1. Sepsis, immune thrombocytopenia
2. Severe hepatic and renal disease
3. Patients on anti-inflammatory agents,
4. Anti-coagulation and anti-platelet agents except aspirin.

## SAMPLE SIZE

50 patients admitted with ACS, 50 patients with stable angina pectoris and 50 healthy controls.

## METHODS

Blood samples for Mean platelet volume were taken at the time of admission in all ACS patients which were examined within 1 hour. Blood samples from Stable AP patients and healthy controls were also taken randomly in OP and wards and examined within 1 hour. samples were also sent for CBC, RFT, LFT. All persons were subjected to ECG and ECHOCARDIOGRAPHY. Mean platelet volume was analysed by Automatic analyzer.

## STATISTICAL ANALYSIS

The tests used are

ANOVA

Chi-square test

Data were collected and statistical significance was analysed.

# RESULTS

## SEX AND ACS

DIAGRAM 1:

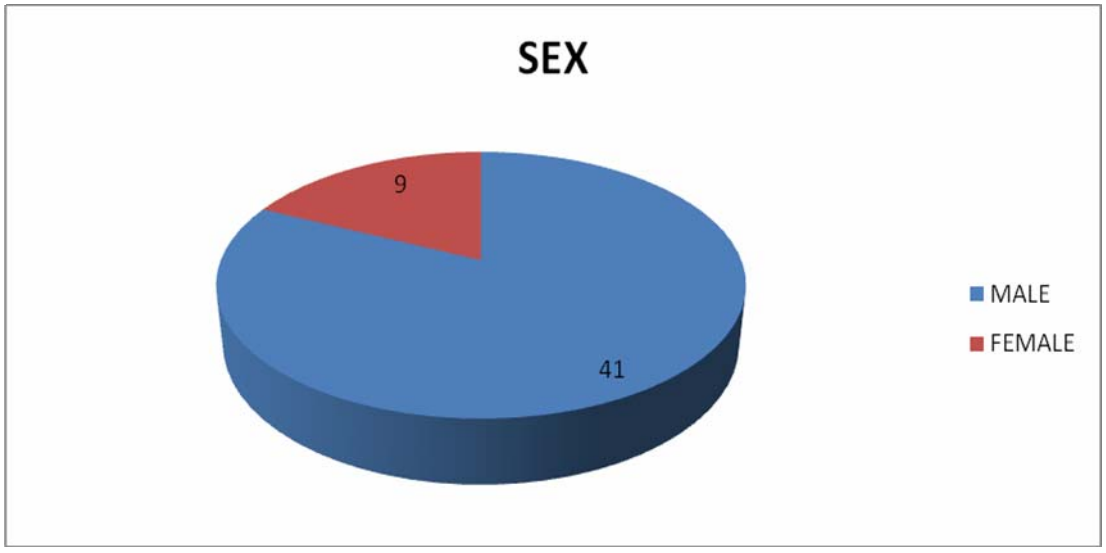



TABLE 1.1 :

| SEX | FREQUENCY | PERCENTAGE |
|---|---|---|
| MALE | 41 | 82% |
| FEMALE | 9 | 18% |
| TOTAL | 50 | 100% |

**TABLE 1.2 :**

| SEX | MEAN | N | STD DEVIATION | STD ERROR OF MEAN |
|---|---|---|---|---|
| MALE | 9.583 | 41 | .7450 | .1163 |
| FEMALE | 9.822 | 9 | .3232 | .1077 |
| TOTAL | 9.626 | 50 | .6919 | .0978 |

**TABLE 1.3:**

| MPV | Sum of squares | Df | Mean square | F | Sig p value |
|---|---|---|---|---|---|
| Between sexes | .423 | 1 | .423 | .881 | .353 |

# AGE GROUP AND ACS

TABLE 2.1

| AGE GROUP | FREQUENCY | PERCENTAGE |
|---|---|---|
| 30-40 | 13 | 26% |
| 41-50 | 20 | 40% |
| 51-60 | 13 | 26% |
| 61-70 | 3 | 6% |
| 71-80 | 1 | 2% |
| TOTAL | 50 | 100% |

DIAGRAM 2 :

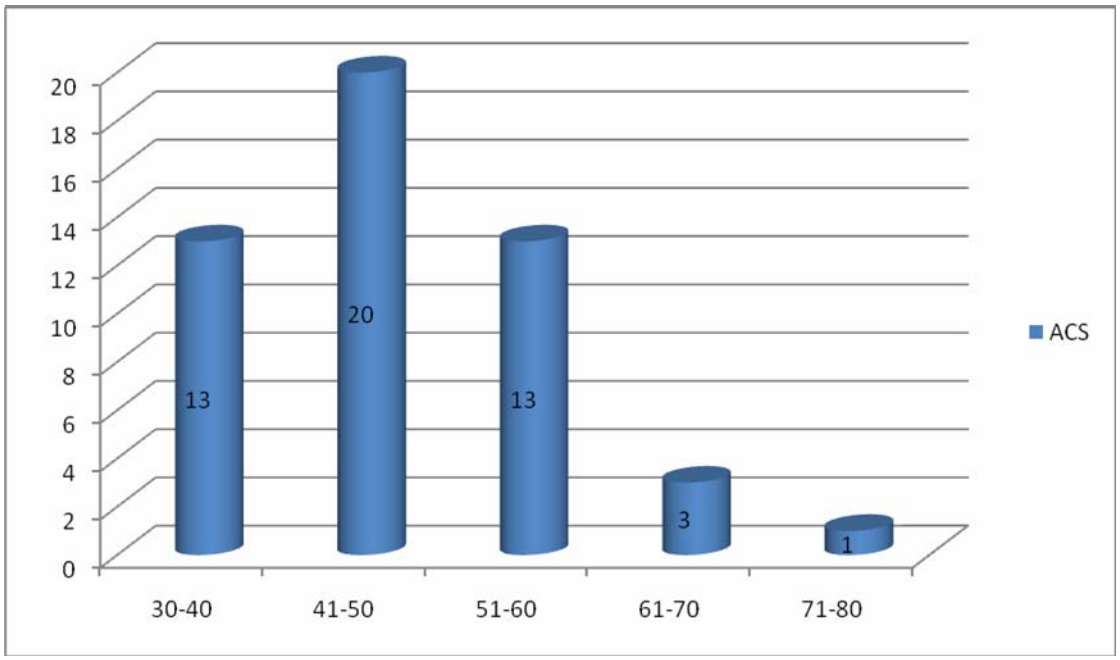


**TABLE 2.2 :**

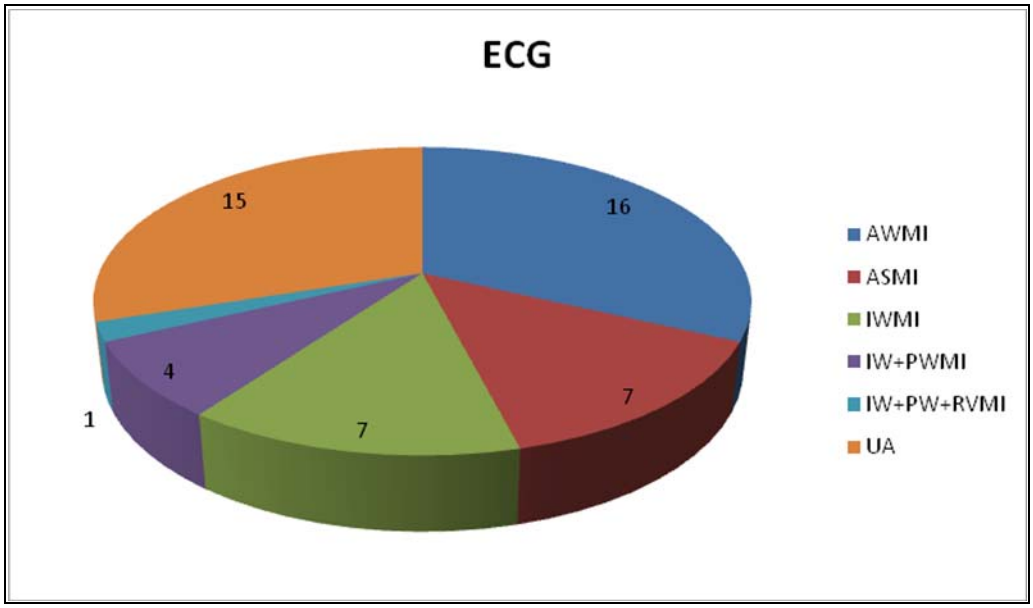
| | MPV | Sum of square | df | Mean square | F | Sig P value |
|---|---|---|---|---|---|---|
| | Between groups | 11.939 | 26 | .459 | .917 | .587 |

# TYPE OF ACS BASED ON ECG

**TABLE 3.1:**

| MI | FREQUENCY | PERCENTAGE |
|---|---|---|
| AWMI | 16 | 32% |
| ASMI | 7 | 14% |
| IWMI | 7 | 14% |
| IW + PWMI | 4 | 8% |
| IW+ PW+ RVMI | 1 | 2% |
| UA | 15 | 30% |

**DIAGRAM 3:**



# HYPERTENSION AND ACS

**TABLE 4.1:**

| HYPERTENSION | FREQUENCY | PERCENTAGE |
|---|---|---|
| YES | 29 | 58% |
| NO | 21 | 42% |

**DIAGRAM 4 :**

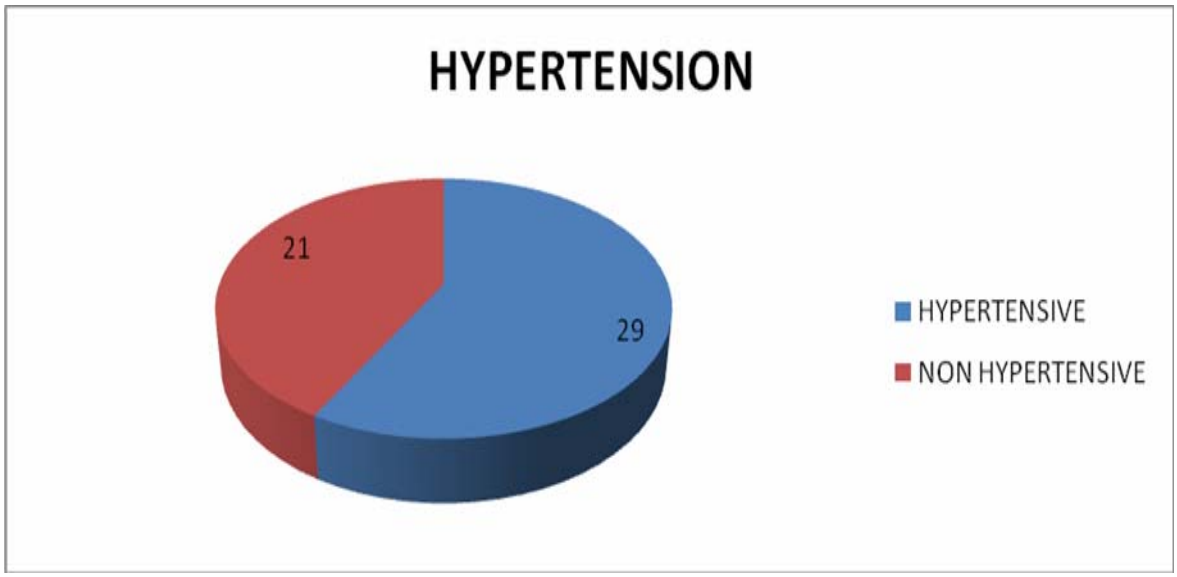


**TABLE 4.2 :**

| MPV | Sum of square | Df | Mean square | F | Sig P value |
|---|---|---|---|---|---|
| Between groups | .346 | 1 | .346 | .719 | .401 |

# DIABETES AND ACS

**TABLE 5.1:**

| DIABETIC | FREQUENCY | PERCENTAGE |
|---|---|---|
| YES | 26 | 52% |
| NO | 24 | 48% |

**DIAGRAM 5:**

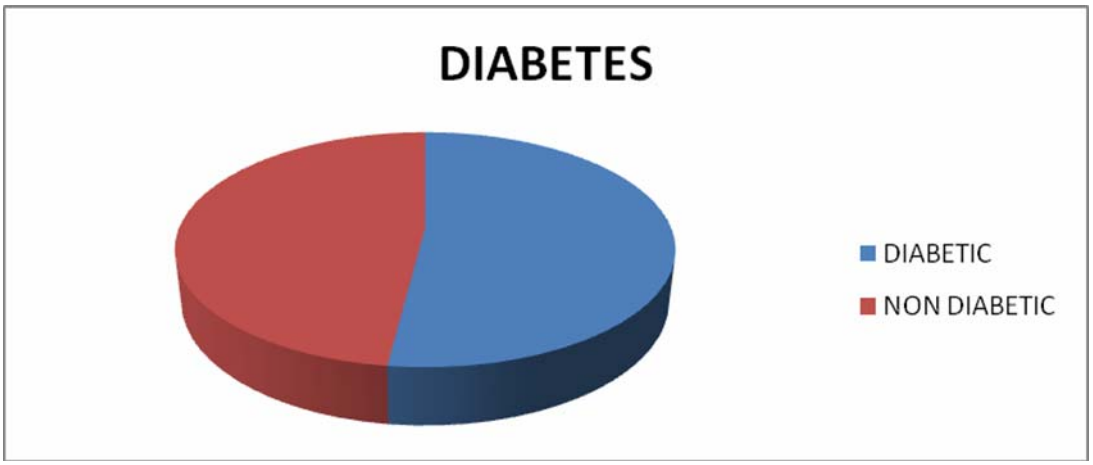


**TABLE 5.2:**

| MPV | Sum of square | Df | Mean square | F | Sig P value |
|---|---|---|---|---|---|
| Between groups | 1.430 | 1 | 1.430 | 3.116 | .084 |

# PRIOR CAD AND ACS

**TABLE 6.1:**

| PRIOR CAD | FREQUENCY | PERCENTAGE |
|---|---|---|
| YES | 6 | 12% |
| NO | 44 | 88% |

**DIAGRAM 6 :**

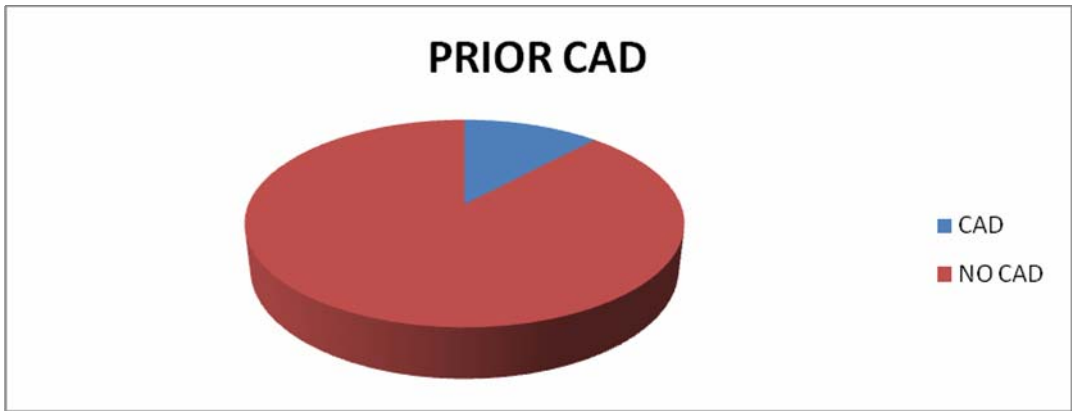



**TABLE 6.2 :**

| MPV | Sum of square | Df | Mean square | F | Sig P value |
|---|---|---|---|---|---|
| Between groups | .108 | 1 | .108 | .223 | .639 |

# SMOKING AND ACS

**TABLE 7.1:**

| SMOKING | FREQUENCY | PERCENTAGE |
|---|---|---|
| YES | 30 | 73% |
| NO | 20 | 27% |

**DIAGRAM 7 :**

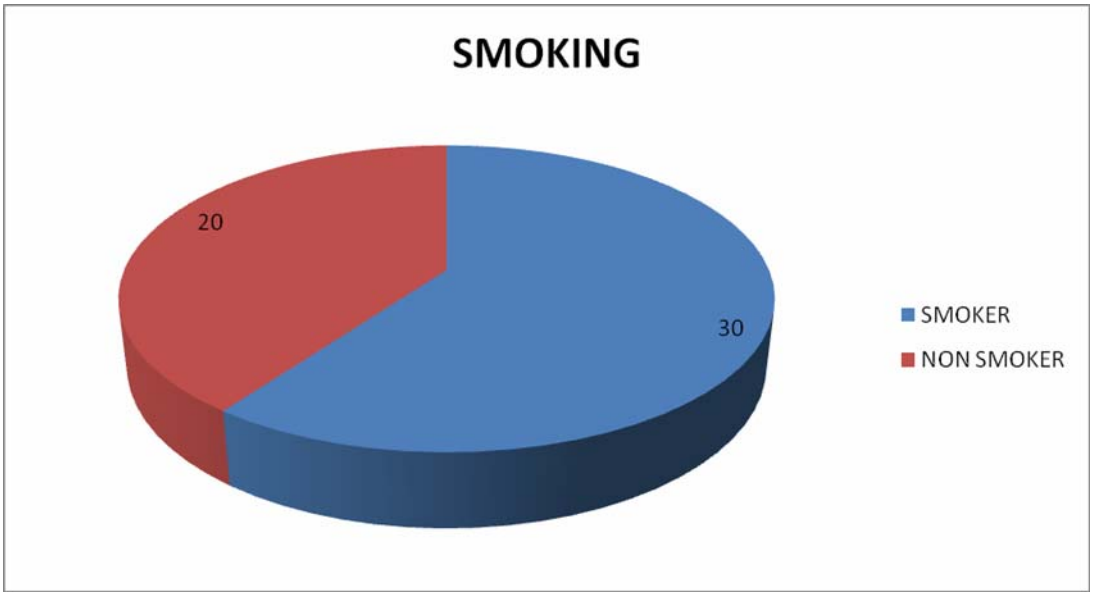


**TABLE 7.2 :**

| MPV | Sum of squares | Df | Mean square | F | Sig P value |
|---|---|---|---|---|---|
| Between groups | 1.255 | 1 | 1.255 | 2.712 | .106 |

# ALCOHOL AND ACS

**TABLE 8.1:**

| ALCOHOL | FREQUENCY | PERCENTAGE |
|---|---|---|
| YES | 34 | 82% |
| NO | 16 | 18% |

**DIAGRAM 8 :**

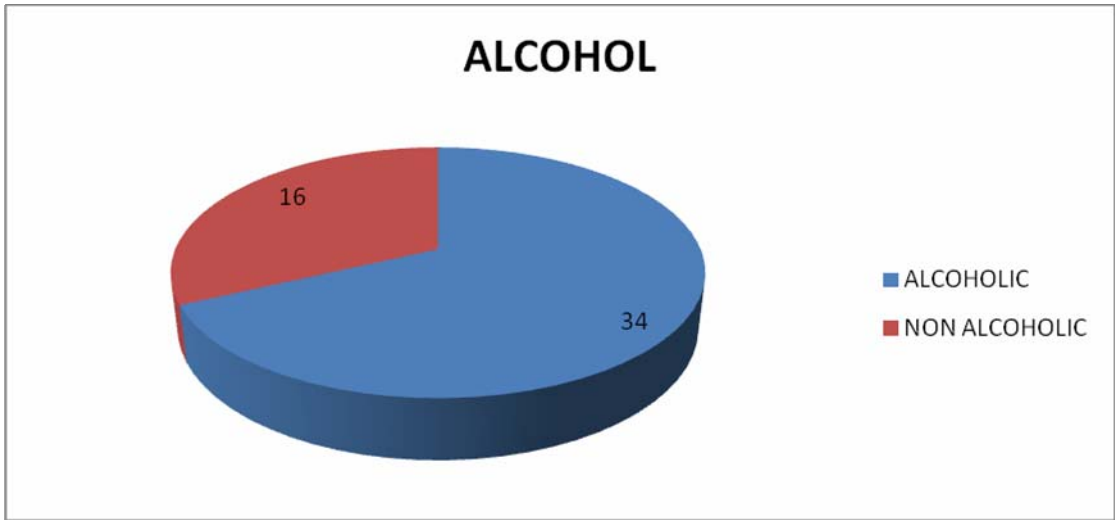


**TABLE 8.2 :**

| MPV | Sum of squares | Df | Mean square | F | Sig P value |
|---|---|---|---|---|---|
| Between groups | .003 | 1 | .003 | .006 | .937 |

# KILLIP CLASSIFICATION

**TABLE 9.1:**

| Killip class | Frequency | Percentage | Mortality | Percentage |
|---|---|---|---|---|
| 1 | 16 | 45% | 0 | 0% |
| 2 | 12 | 33% | 1 | 17% |
| 3 | 4 | 11% | 1 | 17% |
| 4 | 4 | 11% | 4 | 66% |
| **Total** | **35** | 100% | **6** | 100% |

**DIAGRAM 9:**

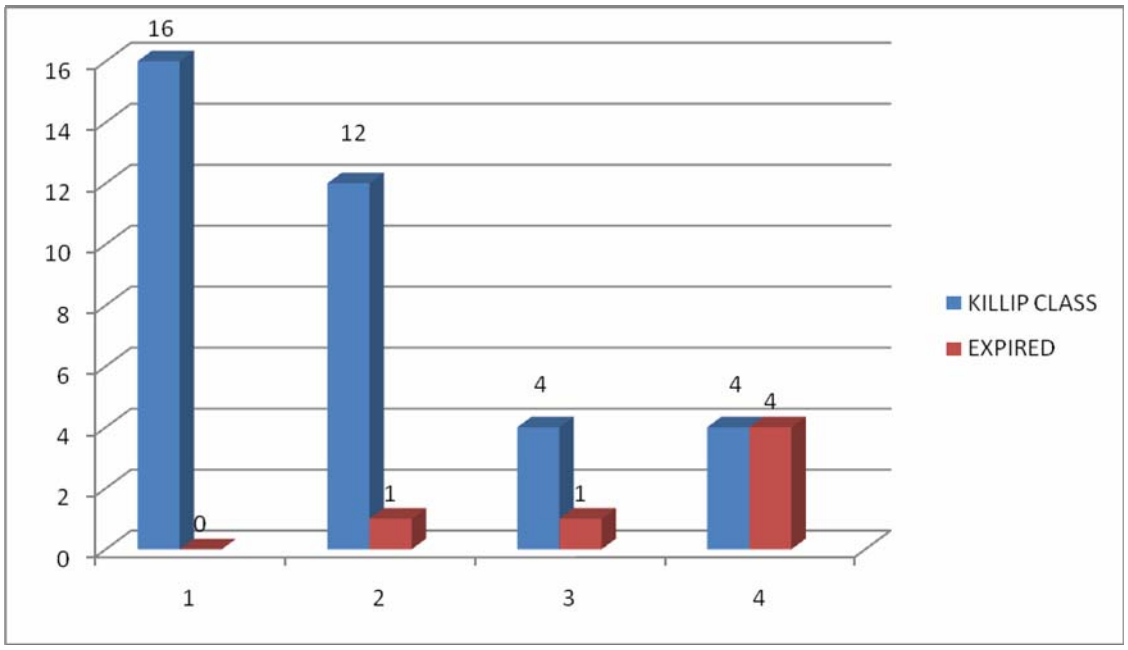


**TABLE 9.2 :**

| MPV | Sum of squares | Df | Mean square | F | Sig P value |
|---|---|---|---|---|---|
| Between groups | 2.968 | 3 | .989 | 2.225 | .104 |

# MEAN PLATELET VOLUME IN COMPARISON

**DIAGRAM 10:**

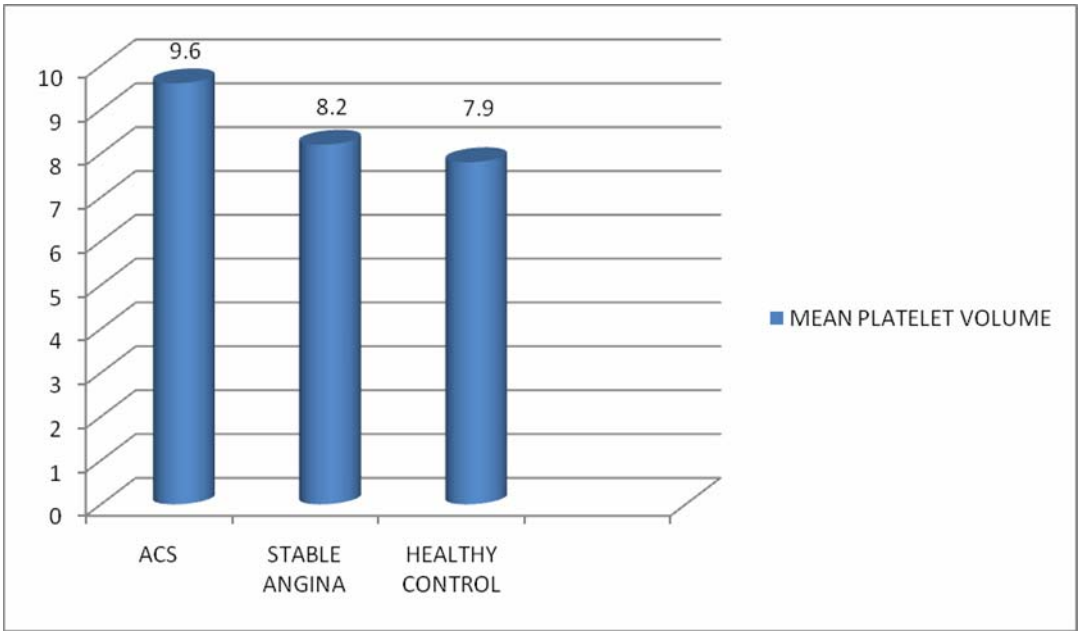



**TABLE 10 :**

| GROUP | MEAN PLATELET VOLUME (Femtolitre) |
|---|---|
| ACS | 9.6 |
| STABLE ANGINA | 8.2 |
| HEALTHY CONTROL | 7.9 |

# ECHOCARDIOGRAPHY FINDINGS IN ACS

**TABLE 11.1:**

| Echo | Frequency | Percentage |
|---|---|---|
| NO RWMA | 13 | 26% |
| RWMA | 11 | 22% |
| MILD LV DYSFUNCTION | 11 | 22% |
| MOD LV DYSFUNCTION | 8 | 16% |
| SEVERE LV DYSFUNCTION | 7 | 14% |
| TOTAL | 50 | 100% |

**RWMA- REGIONAL WALL MOTION ABNORMALITY**

**DIAGRAM 11 :**

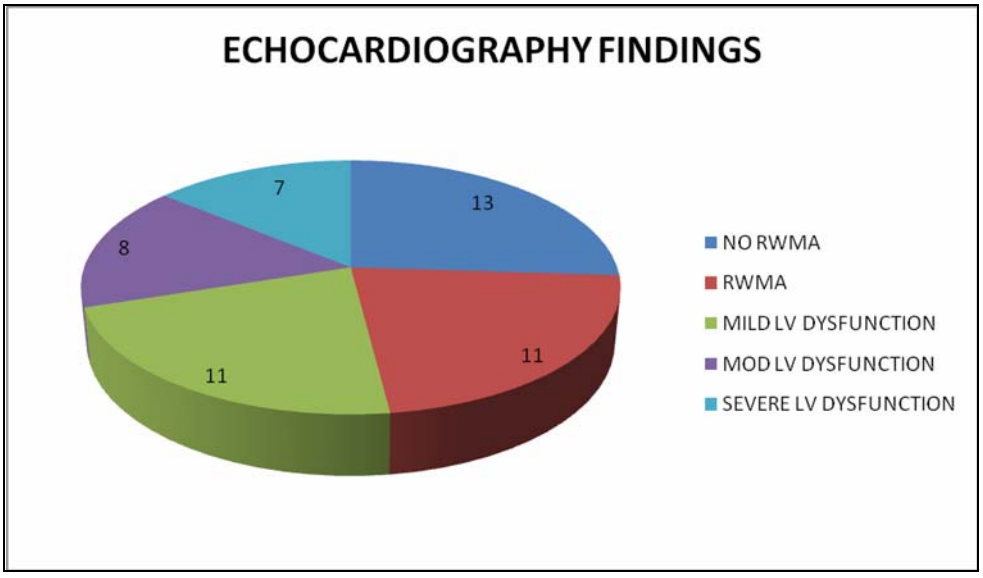


**TABLE 11.2 :**

| MPV | Sum of squares | Df | Mean square | F | Sig |
|---|---|---|---|---|---|
| Between groups | 4.326 | 4 | 1.081 | 2.544 | .052 |

# MEAN PLATELET VOLUME IN ACS

**TABLE 12.1:**

| GROUP | MEAN PLATELET VOLUME(FEMTOLITRE) |
|---|---|
| AWMI | 9.6 |
| ASMI | 9.6 |
| IWMI | 9.7 |
| IW+PWMI | 10.0 |
| IW+PW+RVMI | 9.8 |
| UA | 9.5 |

**DIAGRAM 12 :**

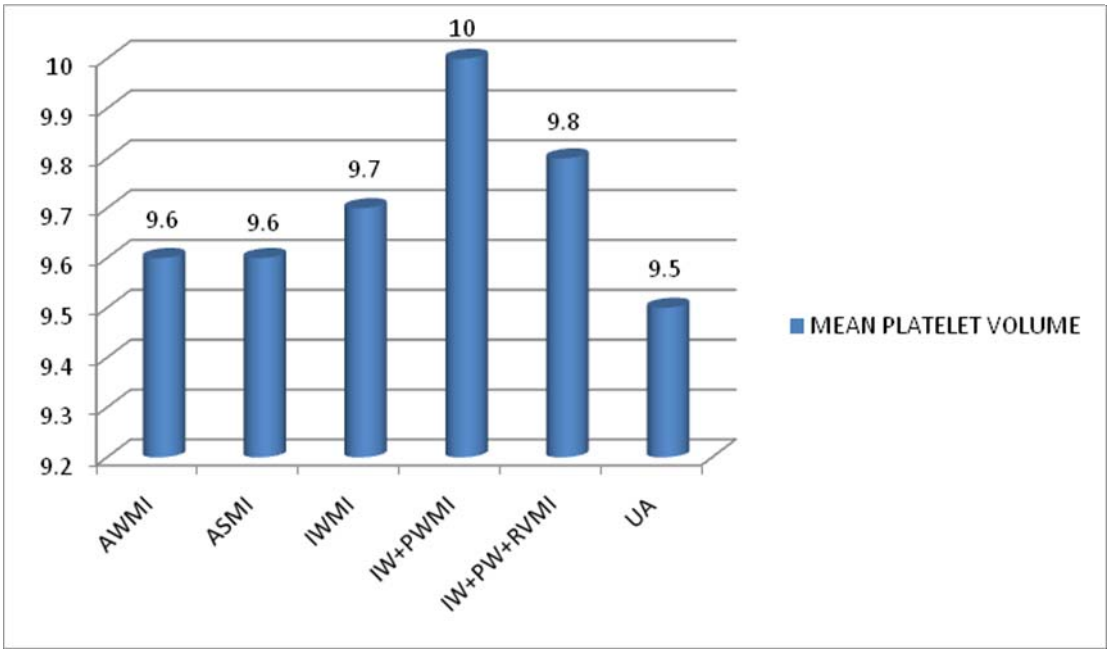


**TABLE 12.2 :**

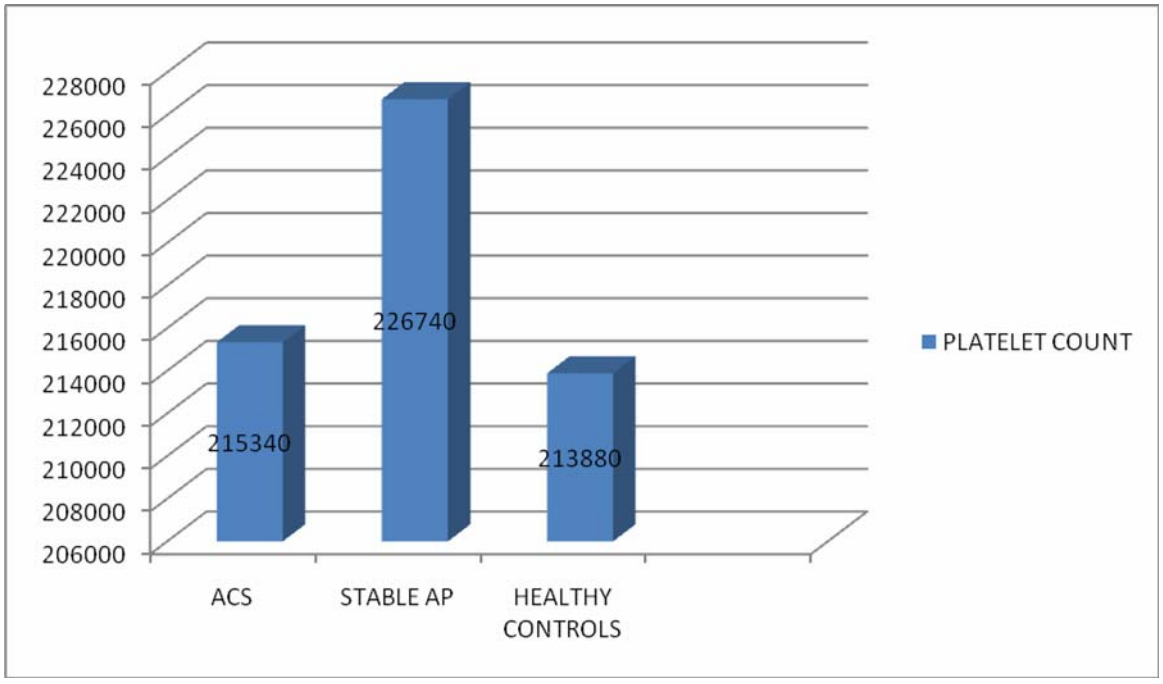
| MPV | Sum of squares | Df | Mean square | F | Sig |
|---|---|---|---|---|---|
| Between groups | .860 | 5 | .172 | .335 | .889 |

# PLATELET COUNT IN COMPARISON

**TABLE 13:**

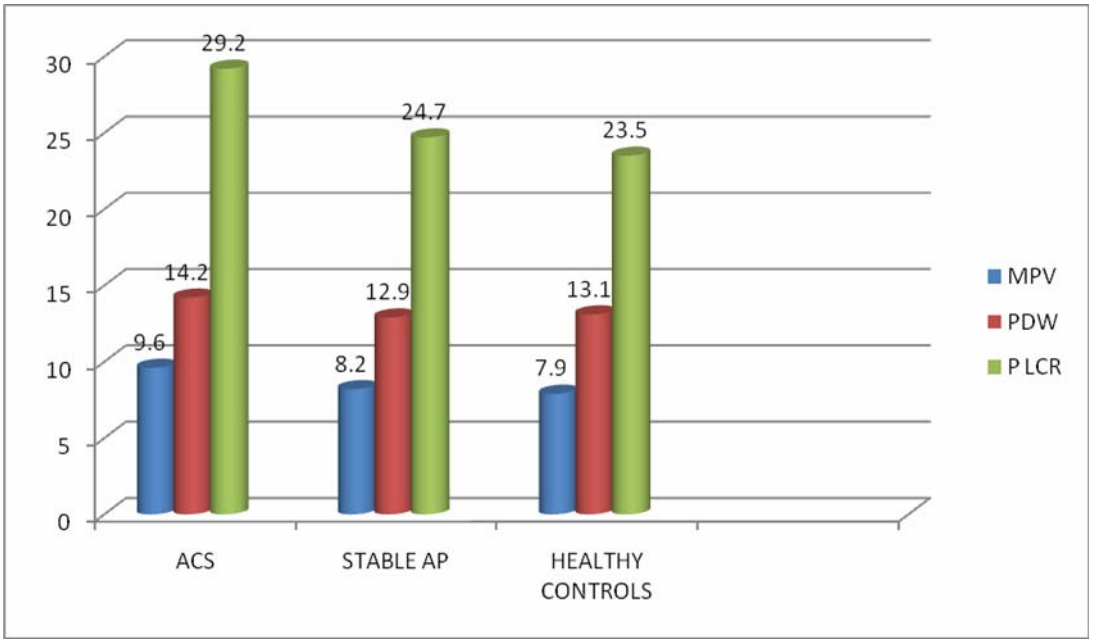
| PLATELET COUNT | MEAN cells/dl |
|---|---|
| ACS | 2,15,340 |
| STABLE ANGINA PECTORIS | 2,26,740 |
| HEALTHY CONTROLS | 2,13,880 |

**DIAGRAM 13 :**



# MPV PDW P-LCR IN COMPARISON

**TABLE 14:**




MPV – MEAN PLATELET VOLUME

PDW – PLATELET DISTRIBUTION WIDTH

P-LCR – PLATELET LARGE CELL RATIO

**DIAGRAM 14 :**

| GROUP | MPV(fl) | PDW% | P-LCR |
|---|---|---|---|
| ACS | 9.6 | 14.2 | 29.2 |
| STABLE AP | 8.2 | 12.9 | 24.7 |
| HEALTHY CONTROLS | 7.9 | 13.1 | 23.5 |

# OUTCOME AND DEATH

**TABLE 15.1:**

| OUTCOME | FREQUENCY | PERCENTAGE |
|---|---|---|
| IMPROVED | 44 | 88 |
| EXPIRED | 6 | 12 |

**DIAGRAM 15 :**

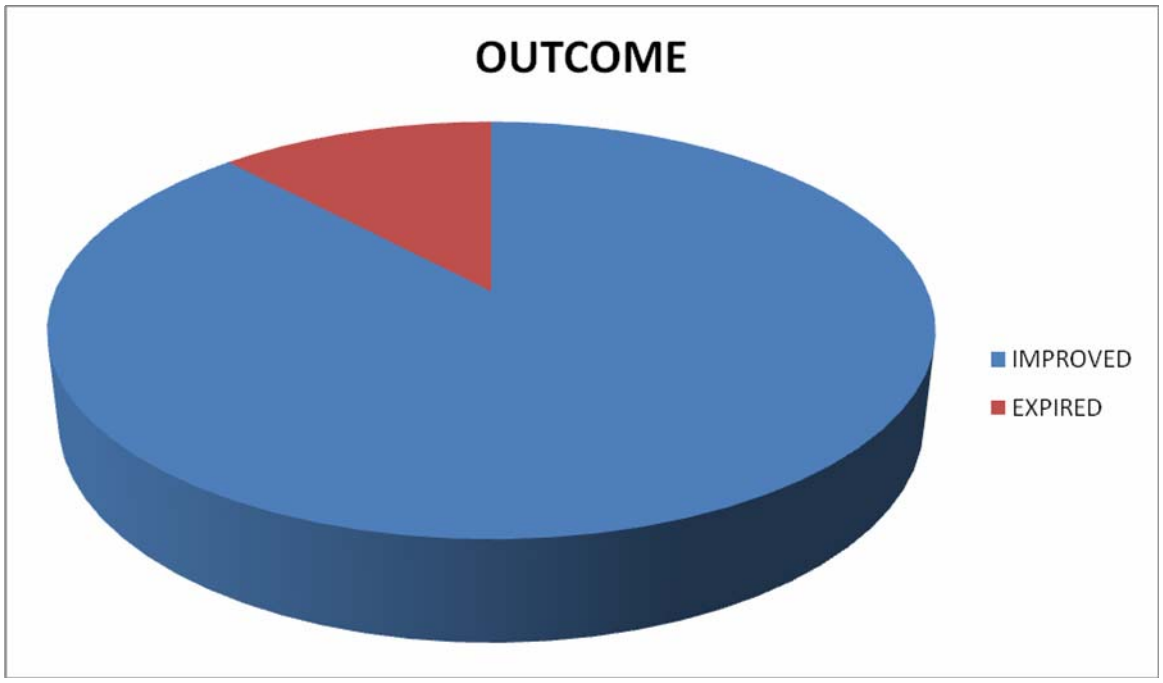


**TABLE 15.2 :**

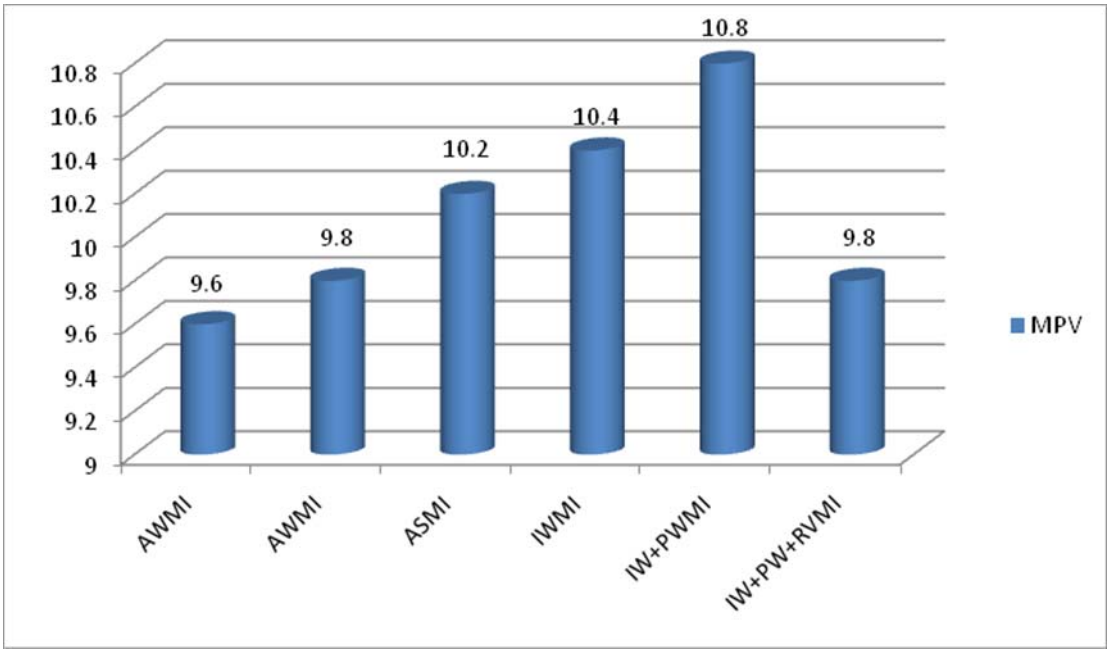
| MPV | Sum of squares | Df | Mean square | F | Sig |
|---|---|---|---|---|---|
| Between groups | 2.246 | 1 | 2.246 | 5.084 | .029 |

# MPV AND DEATH

**TABLE 16:**

| PATIENT NAME | DEATH | MPV(femtolitre) |
|---|---|---|
| MARIMUTHU | AWMI | 9.6 |
| BALAJI | AWMI | 9.8 |
| FATHIMA | ASMI | 10.2 |
| YUVACHANDRAN | IWMI | 10.4 |
| DEVAN | IW+PWMI | 10.8 |
| MUNUSAMY | IW+PW+RVMI | 9.8 |

**DIAGRAM 16 :**



## COMPARISON OF GROUPS BY MPV

**TABLE 17:**

| | | | P-value | Sig/ Not Sig |
|---|---|---|---|---|
| **MPV (Fl)** | ACS group 9.6 ± 0.7 | Stable AP group: 8.2 ± 0.6 | 0.000 | Sig |
| | Stable AP group 8.2 ± 0.6 | Control group: 7.9 ± 0.5 | 0.006 | Sig |
| | ACS group 9.6 ± 0.7 | Control group: 7.9 ± 0.5 | 0.000 | Sig |

**TABLE 18:**

## COMPARISON OF GROUPS BY MEAN PLATELET COUNTS

| | | | P-value | Sig/ Not Sig |
|---|---|---|---|---|
| **Platelet count** | ACS group 215340 ± 63491.5 | Stable AP group 226740 ± 49723.6 | 0.524 | Not Sig |
| | Stable AP group 226740 ± 49723.6 | Control group 213880 ± 41747.8 | 0.440 | Not Sig |
| | ACS group 215340 ± 63491.5 | Control group 213880 ± 41747.8 | 0.989 | Not Sig |

# DISCUSSION

In our study, three groups were taken into account. Acute coronary syndrome, stable angina pectoris and healthy controls respectively.

In our study the significance of mean platelet volume in acute coronary syndrome is the prime concern and they were compared with the other two groups.

Samples for Mean platelet volume were taken at the time of admission in ACS group. MPV was reported by automatic analyzer. Statistical significance in MPV was analysed in each group.

## AGE AND MPV IN ACS

Mean age of patients who sustained ACUTE CORONARY SYNDROME was 48 in my study. Mean age in male was 46.7% and female was 52.4%. Mean age in males is less than that of female. No statistical significance was detected in MPV between various age groups in relation to ACS (p value is .587 i.e. >.05).

## SEX DISTRIBUTION AND MPV IN ACS

Out of 50 patients, 41(82%) were males and 9(18%) were females. In a study[5] the 70.8% were males. No statistical

significance was detected in MPV between the two sexes in relation to ACS (p value is .353 i.e. >.05).

**TYPE OF ACS**

In my study, out of 50 ACS patients, 16(32%) had AWMI, 7(14%) had ASMI, 7(14%) had IWMI, 4(8%) had IW+PWMI, 1(2%) had IW+PW+RVMI and 15(30%) had UNSTABLE ANGINA.

**HYPERTENSION AND MPV IN ACS**

29(58%) were hypertensive and 21(42%) were non-hypertensive. In a similar study[5] et al 60% were males. No statistical significance was detected in MPV between the hypertensive and non-hypertensive in relation to ACS (p value is .401 i.e. >.05).

**DIABETES AND MPV IN ACS**

26 (52%) were diabetic and 24(48%) were non diabetic. In a similar study[5] 36.2% were diabetic. No statistical significance was detected in MPV between diabetics and non-diabetics in relation to ACS (p value is .084 i.e. >.05).

## SMOKING AND MPV IN ACS

30(73%) were smoker out of 41 males. None of the females were smokers. 20(82%) were non-smoker. No statistical significance was detected in MPV between smokers and non-smokers in relation to ACS (p value is .106 i.e. >.05).

## ALCOHOL AND MPV IN ACS

34 (68%) were consuming alcohol and 16(32%) did not consume alcohol. No statistical significance was detected in MPV between alcoholics and non-alcoholics in relation to ACS (p value is .937 i.e. >.05).

## KILLIP CLASSIFICATION AND MPV IN ACUTE MYOCARDIAL INFARCTION

Patients were divided into 4 based on this classification. 15 (45%) were in Class 1, 12 (33%) were in class 2, 4 (11%) were in class 3 and 4(11%) were in class 4. No statistical significance was detected in MPV between the various killip groups in relation to ACS (p value is .104 i.e. >.05).

## KILLIP CLASSIFICATION AND OUTCOME IN ACUTE MYOCARDIAL INFARCTION

Patients in each killip class were analysed for death as outcome. Out of the total 0 (0%) death in class 1, 1 (8%) death in class 2, 1 (25%) death in class 3 and 4 (100%) deaths in class 4 occurred.

## ECHO FINDINGS AND MPV IN ACS

In our study, echo findings were analysed. 13 (26%) had NO RWMA, 11(22%) had RWMA, 11(22%) had MILD LV DYSFUNCTION, 8(16%) had MODERATE LV DYSFUNCTION, 7(14%) had SEVERE LV DYSFUNCTION. No statistical significance was detected in MPV between various echo findings in relation to ACS (p value is .052 i.e. >.05).

## MEAN PLATELET VOLUME

Our study showed that mean platelet volume (Femtolitres) in ACS group was 9.6, STABLE AP group was 8.2 and HEALTHY CONTROLS was 7.9. Mean platelet volume were comparable between ACS and STABLE AP with statistically significant difference of 0.000(<0.5). Similarly mean platelet volume were

comparable between STABLE AP AND HEALTHY CONTROLS with significant difference of 0.006(<0.5), ACS and HEALTHY CONTROLS with significant difference of 0.000(<0.5).

In a similar study[4] which compared unstable AP and AMI patients, MPV in unstable AP was 9.0 ± 1.0 fl, and 8.9 ± 0.8 fl in AMI patients. MPV were similar between these two groups with no statistically significant difference p=0.999.

In stable AP patients, MPV as 7.5 ± 0.6 fl and 7.2 ± 0.6 fl in control group, respectively. MPV were comparable between these two groups with no statistically significant difference p=0.126.

When unstable angina pectoris and AMI cases were compared to each of stable angina pectoris and control groups, it was observed that MPV was increased.

Here in our study too, there is statistically significant difference between ACS and other two groups which is similar to the above cited study[4].

In the study by Endler et al[10], compared AMI patients to those with stable AP, found MPV to be increased which is consistent with our study.

In the study by Kishk et al[24]. which compared AMI patients to stable angina pectoris and control groups detected that the former group had lower platelet count and higher MPV than the latter groups. In our study, mean platelet volume was higher in AMI group than others but platelet count was not reduced.

The study by Puzzili et al[36]. found that MPV was higher in unstable angina pectoris patients, compared to stable angina and control groups which is consistent with our study.

**MEAN PLATELET VOLUME IN ACS**

In our study mean platelet volume in AWMI was 9.6, ASMI was 9.6, IWMI was 9.7, IW+PWMI was 10.0, IW+PW+RVMI was 9.8 and UA was 9.5. No statistical significance was detected in MPV between the various types of ACS (p value is .889 i.e. >.05).

**PLATELET COUNT AND ACS**

In our study, platelet counts in three groups were analysed. Mean value in ACS group was 2,15,340 cells/dl, STABLE AP was 2,26,740 cells/dl and HEALTHY CONTROLS was 2,13,880 cells/dl. While comparing between the groups there was no statistical difference between any groups.

In a similar study[4] unstable AP patients, mean platelet count was 239.6 ± 59.2 x 109 /L respectively; and 228.5 ± 74.1 x 109/L in AMI patients. Mean platelet count was similar between these two groups with no statistically significant difference p=0.791.

In stable AP patients, mean platelet count was detected as 268.3 ± 73.5 x

109/L, and 285.5 ± 80.9 x 109/L in control group, respectively. Mean platelet count was comparable between these two groups with no statistically significant difference p=0.586.

When unstable angina pectoris and AMI cases were compared to each of stable angina pectoris and control groups, it was observed that platelet counts were decreased.

Hence in our study the results were not convincing while comparing to the above study in terms of platelet count.

**PLATELET DISTRIBUTION WIDTH AND ACS**

In our study mean value of in ACS group was 14.2%, STABLE AP was 12.9% and HEALTHY CONTROLS was 13.1%.

**PLATELET-LARGE CELL RATIO AND ACS**

In our study mean value of P-LCR in ACS group was 29.2,

STABLE ANGINA was 24.7 and HEALTHY CONTROLS was 23.5.

**OUTCOME AND MPV IN ACS**

In our study, 6(12%) patients expired and 44(88%) survived out of 50 in the ACS group. Statistical significance was detected in MPV between the outcomes in relation to ACS (p value is .029 i.e. < .05).

It is revealed from the study that MPV is increased in ACS group in comparison with stable angina pectoris and healthy controls. Statistical difference has been showed in MPV between the groups.

# LIMITATIONS OF STUDY

1) MPV has been reported to be dependent on a number of variables

   a. Time of analysis after venipuncture,

   b. Method of analysis,

   c. Anticoagulant used

   d. Specimen storage temperature.

2) The population studied is small when compared to other studies.

3) Cardiac biomarkers were not done for all the patients.

4) Coronary angiography was not done for all the patients.

# CONCLUSION

1) Mean platelet volume was found to be increased in ACS group when compared to Stable angina pectoris and Healthy controls.
2) Statistically significant difference in mean platelet volume was found between the three groups respectively.

3) Mean platelet volume is an easy and feasible test, it can be used as along with other cardiac biomarkers in patients coming with chest pain to evaluate for acute coronary syndrome.
4) More comprehensive studies are required to further evaluate of the beneficial effects of platelet aggregation inhibitors or other drugs on patients with increased MPV.

| NAME | AGE | SEX | PRESENTATION | DURATION | HYPERTENSION | DIABETES | PRIOR CAD | SMOKING | ALCOHOL | BPmm hg | PULSE | KILLIP | THROMBOLYSIS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Loganathan | 30 | M | AWMI | 4 hrs | NO | NO | NO | YES | YES | 100/70 | 108 | 3 | YES |
| Vasu | 43 | M | ASMI | 12 hrs | YES | NO | NO | YES | YES | 130/80 | 102 | 2 | YES |
| Paramasivam | 46 | M | IWMI | 5 | NO | YES | NO | NO | YES | 120/80 | 96 | 2 | YES |
| Varadharaj | 55 | M | AWMI | 4 | YES | YES | NO | YES | YES | 150/90 | 96 | 1 | YES |
| Amulu | 62 | F | UA | 8 | NO | NO | YES | NO | NO | 110/80 | 102 | 1 | YES |
| Devan | 36 | M | IW+PWMI | 3 | NO | YES | NO | NO | NO | 100/80 | 40 | 3 | YES |
| Chittibabu | 68 | M | IWMI | 7 | YES | NO | NO | YES | YES | 120/80 | 72 | 1 | YES |
| Fathima | 54 | F | ASMI | 5 | YES | YES | NO | NO | NO | 80/? | ? | 4 | NO |
| Saravanan | 35 | M | AWMI | 4 | NO | NO | NO | YES | YES | 150/90 | 102 | 1 | YES |
| Gopal | 40 | M | UA | 8 | YES | NO | NO | NO | YES | 110/90 | 96 |  | NO |
| Ramadoss | 47 | M | UA | 4 | YES | YES | NO | YES | YES | 160/90 | 102 |  | NO |
| Saseeb | 52 | M | AWMI | 10 | NO | YES | NO | YES | YES | 140/80 | 120 | 2 | YES |
| Velu | 56 | M | AWMI | 6 | YES | YES | NO | YES | YES | 120/80 | 108 | 1 | YES |
| Ravi | 42 | M | IWMI | 4 | YES | NO | NO | YES | NO | 110/80 | 96 | 2 | YES |
| Mariammal | 45 | F | AWMI | 6 | YES | NO | YES | NO | NO | 100/70 | 102 | 1 | YES |
| Vimala | 54 | F | IW+PWMI | 4 | NO | YES | NO | NO | YES | 100/80 | 108 | 2 | YES |
| Raji | 50 | M | UA | 4 | YES | YES | NO | YES | YES | 160/100 | 96 |  | NO |
| Yuvachandran | 58 | M | IWMI | 5 | YES | YES | NO | YES | YES | 100/60 | 84 | 2 | YES |
| Vinayagam | 38 | M | UA | 7 | NO | NO | NO | YES | NO | 130/90 | 102 |  | NO |
| Thangadurai | 36 | M | UA | 4 | YES | NO | NO | YES | YES | 110/80 | 108 |  | NO |
| Veeramani | 40 | M | UA | 11 | YES | NO | NO | NO | NO | 120/80 | 96 |  | NO |
| Nidhi | 42 | M | AWMI | 6 | NO | NO | YES | YES | NO | 110/80 | 102 | 1 | YES |
| Panneer | 50 | M | ASMI | 5 | YES | YES | NO | YES | YES | 150/90 | 96 | 2 | YES |
| Raja | 65 | M | UA | 5 | NO | YES | NO | NO | YES | 110/90 | 102 |  | NO |
| Balaji | 39 | M | AWMI | 6 | NO | YES | NO | YES | YES | 60/? | ? | 4 | NO |
| Kani | 48 | M | AWMI | 8 | YES | YES | NO | NO | YES | 200/100 | 120 | 3 | NO |
| Parasuram | 56 | M | IW+PWMI | 6 | YES | NO | NO | YES | YES | 90/60 | 54 | 3 | YES |
| Prabakar | 40 | M | UA | 7 | YES | NO | NO | YES | YES | 160/90 | 108 |  | NO |
| Parameswari | 46 | F | ASMI | 5 | NO | YES | NO | NO | NO | 110/80 | 96 | 1 | YES |
| Rathinavel | 54 | M | UA | 4 | NO | NO | NO | NO | YES | 150/90 | 96 |  | NO |
| James | 50 | M | UA | 1 | YES | YES | NO | YES | YES | 140/90 | 102 |  | NO |
| Vetri | 34 | M | IWMI | 4 | NO | NO | NO | YES | YES | 100/80 | 96 | 1 | YES |
| Munusamy | 42 | M | I+P+RVMI | 3 | YES | YES | NO | YES | YES | 70/? | ? | 4 | NO |
| Vennila | 56 | F | AWMI | 6 | NO | NO | NO | NO | NO | 160/100 | 108 | 1 | YES |
| Sivasankaran | 48 | M | UA | 6 | YES | NO | YES | NO | YES | 150/100 | 102 |  | NO |
| Munivel | 44 | M | AWMI | 4 | NO | NO | NO | YES | YES | 140/90 | 96 | 1 | YES |
| Pankajam | 37 | F | ASMI | 5 | YES | YES | NO | YES | NO | 210/110 | 108 | 2 | NO |
| Ismail | 49 | M | AWMI | 3 | NO | YES | NO | YES | YES | 160/90 | 120 | 1 | YES |
| Veerammal | 74 | F | UA | 8 | YES | NO | NO | NO | NO | 160/100 | 102 |  | NO |
| Vendhan | 44 | M | UA | 12 | NO | YES | NO | YES | YES | 130/90 | 96 |  | NO |
| Karthik | 60 | M | AWMI | 5 | YES | NO | YES | NO | NO | 110/70 | 96 | 1 | YES |

| NAME | AGE | SEX | PRESENTATION | DURATION | HYPERTENSION | DIABETES | PRIOR CAD | SMOKING | ALCOHOL | BPmm hg | PULSE | KILLIP | THROMBOLYSIS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Mohammad | 54 | M | IWMI | 4 | YES | YES | NO | YES | NO | 100/70 | 72 | 2 | YES |
| Marimuthu | 48 | M | AWMI | 6 | NO | YES | NO | NO | YES | 70/? | 126 | 4 | NO |
| Palani | 56 | M | ASMI | 8 | YES | NO | NO | NO | YES | 170/100 | 64 | 2 | YES |
| Veeraragavan | 39 | M | IW+PWMI | 4 | NO | YES | NO | YES | YES | 130/90 | 102 | 1 | YES |
| Ayyadurai | 45 | M | UA | 4 | NO | YES | NO | YES | YES | 110/70 | 102 | | NO |
| Venda | 44 | F | ASMI | 3 | YES | NO | NO | NO | NO | 110/80 | 96 | 1 | YES |
| Periasamy | 52 | M | IWMI | 5 | YES | YES | NO | NO | YES | 110/80 | 108 | 1 | YES |
| Annamalai | 40 | M | AWMI | 10 | NO | YES | NO | YES | YES | 100/80 | 120 | 2 | YES |
| Prakash | 45 | M | AWMI | 7 | YES | NO | YES | YES | NO | 140/90 | 102 | 2 | YES |

ECHO

| | |
|---|---|
| NO RWMA | 1 |
| RWMA | 2 |
| MILD LV DYSFUNCTION | 3 |
| MODERATE | 4 |
| SEVERE | 5 |

| HEPARIN | TOTAL COUNT | P | L | E | PLATELET COUNT | SUGAR | UREA | CREATININE | TB | DB | OT | PT | TC | TGL | HDL | MPV | PDW | P-LCR | ECHO | HOSPITAL STAY | OUTCOME |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 12000 | 72 | 26 | 2 | 204000 | 84 | 25 | 0.8 | 1.1 | 0.3 | 42 | 46 | 158 | 124 | 46 | 10.9 | 15.6 | 37.6 | 3 | 7 | SURVIVED |
| | 9600 | 68 | 30 | 2 | 186000 | 96 | 30 | 0.9 | 1 | 0.1 | 44 | 45 | 240 | 164 | 48 | 9.2 | 14.2 | 29.2 | 2 | 5 | SURVIVED |
| | 9700 | 66 | 30 | 4 | 126000 | 128 | 32 | 1 | 0.9 | 0.1 | 45 | 44 | 168 | 96 | 66 | 11.3 | 17.6 | 40.2 | 3 | 6 | SURVIVED |
| | 8600 | 72 | 26 | 2 | 304000 | 192 | 28 | 0.9 | 0.9 | 0.1 | 45 | 46 | 170 | 130 | 64 | 8.9 | 12.8 | 26.2 | 4 | 5 | SURVIVED |
| | 11000 | 66 | 33 | 1 | 208000 | 102 | 30 | 0.8 | 0.8 | 0.1 | 44 | 45 | 220 | 140 | 32 | 10.2 | 14.8 | 35.6 | 1 | 7 | SURVIVED |
| | 9600 | 74 | 24 | 2 | 192000 | 284 | 40 | 1.1 | 1 | 0.2 | 42 | 45 | 186 | 100 | 30 | 10.8 | 13.6 | 36.6 | 5 | 1 | EXPIRED |
| | 7200 | 72 | 26 | 2 | 174000 | 106 | 26 | 0.8 | 1 | 0.1 | 42 | 46 | 240 | 124 | 56 | 9.6 | 13.3 | 28.8 | 3 | 6 | SURVIVED |
| YES | 8400 | 66 | 30 | 3 | 120000 | 204 | 32 | 0.9 | 0.8 | 0.1 | 40 | 42 | 154 | 108 | 45 | 10.2 | 14.8 | 36.8 | 5 | 1 | EXPIRED |
| | 9400 | 64 | 33 | 3 | 304000 | 104 | 28 | 0.6 | 0.9 | 0.1 | 46 | 44 | 120 | 120 | 34 | 8.4 | 12.6 | 26.2 | 2 | 6 | SURVIVED |
| YES | 7200 | 70 | 26 | 4 | 286000 | 84 | 28 | 0.8 | 0.8 | 0.1 | 46 | 44 | 189 | 165 | 60 | 9.2 | 14 | 28.2 | 1 | 5 | SURVIVED |
| YES | 8900 | 64 | 33 | 3 | 204000 | 236 | 30 | 1 | 1 | 0.2 | 45 | 43 | 224 | 148 | 42 | 8.6 | 12.8 | 27 | 2 | 5 | SURVIVED |
| | 7400 | 66 | 32 | 2 | 124000 | 164 | 40 | 1.2 | 1 | 0.1 | 44 | 40 | 174 | 130 | 64 | 9.8 | 14.2 | 29.2 | 3 | 6 | SURVIVED |
| | 9400 | 56 | 40 | 4 | 240000 | 198 | 42 | 1.2 | 0.8 | 0.1 | 42 | 45 | 154 | 142 | 60 | 10 | 14.8 | 32.2 | 2 | 5 | SURVIVED |
| | 7000 | 68 | 30 | 4 | 194000 | 90 | 26 | 0.8 | 0.8 | 0.1 | 44 | 47 | 220 | 180 | 40 | 9.2 | 13.6 | 28.6 | 4 | 6 | SURVIVED |
| | 8200 | 70 | 28 | 2 | 300000 | 110 | 22 | 0.6 | 0.9 | 0.1 | 46 | 42 | 185 | 156 | 46 | 9.4 | 13.2 | 26 | 4 | 7 | SURVIVED |
| | 9900 | 68 | 26 | 6 | 120000 | 140 | 28 | 0.6 | 0.8 | 0.2 | 45 | 44 | 140 | 174 | 48 | 9.8 | 14.4 | 28.2 | 4 | 6 | SURVIVED |
| YES | 7600 | 69 | 30 | 1 | 181000 | 264 | 34 | 1.2 | 1 | 0.2 | 48 | 46 | 185 | 125 | 40 | 10.2 | 16 | 30.2 | 1 | 5 | SURVIVED |
| | 8200 | 64 | 34 | 2 | 240000 | 240 | 34 | 1.4 | 0.8 | 0.1 | 45 | 48 | 235 | 158 | 43 | 10.4 | 15.4 | 31.2 | 5 | 14 HRS | EXPIRED |
| YES | 8600 | 60 | 34 | 6 | 288000 | 80 | 26 | 0.8 | 0.8 | 0.1 | 46 | 45 | 180 | 124 | 43 | 9.6 | 14.2 | 28.4 | 1 | 6 | SURVIVED |
| YES | 7400 | 68 | 30 | 2 | 150000 | 196 | 28 | 0.8 | 0.9 | 0.1 | 42 | 40 | 186 | 180 | 47 | 7.8 | 12.4 | 27.4 | 1 | 5 | SURVIVED |
| YES | 6200 | 66 | 30 | 4 | 186000 | 106 | 30 | 1 | 1.1 | 0.2 | 44 | 40 | 214 | 124 | 59 | 9.6 | 13.8 | 27.8 | 2 | 5 | SURVIVED |
| | 8000 | 64 | 32 | 4 | 240000 | 146 | 24 | 0.6 | 1.2 | 0.2 | 46 | 45 | 164 | 168 | 50 | 8.8 | 13.2 | 25.4 | 2 | 7 | SURVIVED |
| | 7400 | 80 | 16 | 4 | 186000 | 102 | 28 | 0.6 | 0.8 | 0.1 | 48 | 46 | 175 | 145 | 60 | 9.2 | 14.4 | 26.4 | 3 | 7 | SURVIVED |
| YES | 9400 | 69 | 28 | 3 | 360000 | 290 | 36 | 1.2 | 0.8 | 0.1 | 44 | 48 | 240 | 186 | 44 | 9.6 | 14.8 | 28.2 | 1 | 5 | SURVIVED |
| YES | 9800 | 54 | 40 | 6 | 172000 | 146 | 40 | 1.4 | 0.9 | 0.1 | 40 | 45 | 146 | 190 | 56 | 10.2 | 15.8 | 30.4 | 5 | 5 | EXPIRED |
| YES | 8800 | 68 | 29 | 3 | 268000 | 380 | 42 | 1.4 | 0.9 | 0.1 | 42 | 47 | 246 | 159 | 55 | 9.4 | 13.4 | 28.4 | 4 | 6 | SURVIVED |
| | 9000 | 62 | 32 | 6 | 142000 | 76 | 28 | 0.8 | 1.2 | 0.2 | 46 | 43 | 198 | 137 | 43 | 10.4 | 15 | 31.8 | 5 | 12 | SURVIVED |
| YES | 6800 | 72 | 22 | 6 | 246000 | 120 | 30 | 0.8 | 0.8 | 0.1 | 40 | 45 | 174 | 186 | 42 | 10.2 | 14.2 | 30.2 | 2 | 6 | SURVIVED |
| | 7200 | 66 | 32 | 2 | 190000 | 138 | 34 | 1.2 | 0.9 | 0.1 | 45 | 46 | 236 | 124 | 48 | 9.8 | 13.8 | 26.8 | 2 | 7 | SURVIVED |
| YES | 9200 | 69 | 28 | 3 | 320000 | 82 | 24 | 0.8 | 1 | 0.1 | 42 | 42 | 149 | 170 | 65 | 8.9 | 13.6 | 25.9 | 1 | 5 | SURVIVED |
| YES | 8600 | 64 | 32 | 4 | 178000 | 190 | 34 | 1 | 1 | 0.2 | 40 | 48 | 248 | 158 | 40 | 9.6 | 14.2 | 27.8 | 1 | 5 | SURVIVED |
| | 9400 | 71 | 26 | 3 | 240000 | 102 | 24 | 1 | 0.9 | 0.2 | 44 | 49 | 246 | 140 | 42 | 8.8 | 13.4 | 27.2 | 4 | 8 | SURVIVED |
| YES | 9200 | 70 | 28 | 2 | 181000 | 166 | 38 | 1.4 | 0.8 | 0.1 | 46 | 45 | 149 | 268 | 44 | 9.8 | 14.2 | 28.6 | 5 | 12 HRS | EXPIRED |
| | 8400 | 68 | 28 | 4 | 120000 | 88 | 22 | 0.8 | 0.8 | 0.1 | 44 | 42 | 154 | 124 | 50 | 10.2 | 14.8 | 31.8 | 2 | 6 | SURVIVED |
| YES | 8540 | 66 | 30 | 4 | 342000 | 92 | 24 | 0.8 | 1 | 0.1 | 42 | 40 | 248 | 182 | 54 | 9.6 | 13.9 | 27.8 | 1 | 6 | SURVIVED |
| | 7000 | 66 | 30 | 4 | 196000 | 110 | 30 | 1 | 1.1 | 0.2 | 40 | 46 | 196 | 196 | 54 | 8.8 | 13.2 | 25.4 | 2 | 5 | SURVIVED |
| YES | 9200 | 64 | 32 | 4 | 246000 | 168 | 34 | 1 | 1 | 0.2 | 42 | 45 | 224 | 145 | 40 | 9.4 | 14 | 28.2 | 3 | 6 | SURVIVED |
| | 8600 | 66 | 32 | 2 | 180000 | 124 | 40 | 1.4 | 1 | 0.1 | 44 | 42 | 184 | 158 | 42 | 10.8 | 15.4 | 30.4 | 3 | 5 | SURVIVED |
| YES | 9600 | 59 | 36 | 5 | 274000 | 102 | 28 | 0.8 | 0.8 | 0.1 | 43 | 48 | 154 | 120 | 65 | 9.8 | 14.6 | 28.2 | 1 | 5 | SURVIVED |
| YES | 8400 | 68 | 30 | 2 | 144000 | 254 | 36 | 1.4 | 0.9 | 0.1 | 42 | 49 | 169 | 142 | 44 | 10.2 | 14.8 | 30.2 | 1 | 5 | SURVIVED |
| | 8300 | 62 | 32 | 6 | 268000 | 84 | 28 | 0.8 | 0.7 | 0.1 | 46 | 42 | 154 | 280 | 42 | 9.2 | 14.2 | 28.4 | 1 | 5 | SURVIVED |

| HEPARIN | TOTAL COUNT | P | L | E | PLATELET COUNT | SUGAR | UREA | CREATININE | TB | DB | OT | PT | TC | TGL | HDL | MPV | PDW | P-LCR | ECHO | HOSPITAL STAY | OUTCOME |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 9500 | 64 | 32 | 4 | 172000 | 174 | 30 | 1 | 0.8 | 0.1 | 44 | 46 | 236 | 146 | 50 | 8.6 | 12.8 | 26.2 | 3 | 7 | SURVIVED |
| YES | 6900 | 66 | 33 | 1 | 189000 | 190 | 36 | 1.2 | 1 | 0.2 | 41 | 48 | 185 | 120 | 64 | 9.8 | 14.8 | 27.2 | 5 | 9 HRS | EXPIRED |
| | 6400 | 70 | 28 | 2 | 348000 | 106 | 26 | 0.8 | 0.9 | 0.1 | 42 | 46 | 174 | 100 | 44 | 9.6 | 13.6 | 26.8 | 3 | 6 | SURVIVED |
| | 9000 | 68 | 30 | 2 | 169000 | 280 | 30 | 0.8 | 0.9 | 0.1 | 40 | 45 | 145 | 126 | 48 | 8.9 | 13.2 | 25.6 | 4 | 7 | SURVIVED |
| YES | 8200 | 60 | 36 | 4 | 283000 | 254 | 34 | 1.2 | 1 | 0.1 | 44 | 42 | 280 | 124 | 42 | 9.6 | 14.6 | 28.4 | 1 | 5 | SURVIVED |
| | 6400 | 64 | 32 | 4 | 182000 | 78 | 28 | 0.6 | 0.7 | 0.1 | 45 | 47 | 184 | 158 | 48 | 9.6 | 14.4 | 27.6 | 3 | 5 | SURVIVED |
| | 7600 | 66 | 32 | 2 | 170000 | 102 | 40 | 1.2 | 0.8 | 0.2 | 44 | 41 | 149 | 224 | 42 | 10.4 | 15.4 | 29.8 | 4 | 6 | SURVIVED |
| | 8400 | 68 | 30 | 2 | 240000 | 220 | 42 | 1.2 | 0.7 | 0.1 | 42 | 48 | 210 | 124 | 40 | 9.2 | 14.8 | 28.8 | 3 | 7 | SURVIVED |
| | 8200 | 66 | 31 | 3 | 190000 | 108 | 28 | 0.8 | 0.8 | 0.1 | 44 | 46 | 168 | 140 | 64 | 9.8 | 15 | 29.2 | 2 | 5 | SURVIVED |

| NAME | AGE | SEX | HYPERTENSION | DIABETES | SMOKING | ALCOHOL | TOTAL COUNT | P | L | E |
|---|---|---|---|---|---|---|---|---|---|---|
| Vikram | 24 | M | NO | NO | NO | YES | 8000 | 56 | 40 | 4 |
| Nalan | 25 | M | NO | NO | YES | YES | 7000 | 66 | 32 | 2 |
| Saravanan | 29 | M | YES | NO | YES | NO | 8000 | 77 | 30 | 3 |
| Zaheer | 30 | M | NO | NO | YES | YES | 7700 | 74 | 22 | 4 |
| Velammal | 30 | F | YES | NO | NO | NO | 7600 | 65 | 33 | 2 |
| Saradha | 32 | F | NO | YES | NO | NO | 6800 | 62 | 36 | 2 |
| Ragavan | 32 | M | YES | NO | YES | YES | 6450 | 70 | 28 | 2 |
| Thiagu | 32 | M | YES | YES | YES | YES | 7800 | 62 | 26 | 2 |
| Ranjith | 32 | M | NO | NO | YES | NO | 6600 | 66 | 32 | 2 |
| Kannadasan | 33 | M | NO | NO | YES | NO | 7500 | 68 | 30 | 2 |
| Vivek | 34 | M | NO | NO | YES | YES | 6800 | 70 | 26 | 4 |
| Baby | 34 | F | NO | NO | NO | NO | 6400 | 64 | 32 | 4 |
| Bama | 34 | F | NO | NO | NO | NO | 5000 | 62 | 36 | 2 |
| Saradhammal | 34 | F | NO | NO | NO | NO | 8800 | 68 | 30 | 2 |
| Karuppusamy | 35 | M | NO | NO | YES | YES | 7800 | 74 | 22 | 4 |
| Basker | 35 | M | NO | YES | YES | YES | 5600 | 62 | 38 | 2 |
| Varadhan | 35 | M | NO | YES | YES | NO | 6800 | 64 | 32 | 4 |
| Jaiganesh | 35 | M | NO | NO | YES | YES | 8500 | 66 | 30 | 4 |
| Bakkiam | 35 | F | NO | NO | NO | NO | 6400 | 66 | 30 | 4 |
| Ramesh | 35 | M | YES | NO | YES | YES | 9900 | 68 | 30 | 2 |
| Rani | 36 | F | YES | YES | NO | NO | 8400 | 60 | 36 | 4 |
| Ellammal | 36 | F | NO | NO | NO | NO | 6600 | 64 | 32 | 4 |
| Veeramani | 36 | M | NO | NO | NO | YES | 10200 | 70 | 28 | 2 |
| Kali | 38 | M | NO | NO | NO | YES | 6200 | 70 | 26 | 4 |
| Ravi | 38 | M | NO | NO | NO | YES | 7200 | 62 | 36 | 2 |
| Mangammal | 38 | F | YES | NO | NO | NO | 6800 | 70 | 28 | 2 |
| Sulaiman | 38 | M | YES | NO | NO | NO | 7480 | 66 | 30 | 4 |
| Mani | 38 | M | YES | YES | YES | YES | 8100 | 70 | 28 | 2 |
| Lalitha | 38 | F | YES | NO | NO | NO | 5800 | 72 | 26 | 2 |
| Velu | 38 | M | NO | NO | YES | NO | 6600 | 62 | 36 | 2 |
| Nandakumar | 38 | M | NO | NO | YES | NO | 9000 | 70 | 28 | 2 |
| Sabari | 40 | M | NO | YES | YES | YES | 6800 | 64 | 32 | 4 |

| NAME | AGE | SEX | HYPERTENSION | DIABETES | SMOKING | ALCOHOL | TOTAL COUNT | P | L | E |
|---|---|---|---|---|---|---|---|---|---|---|
| Adhithan | 40 | M | NO | NO | YES | YES | 6000 | 72 | 26 | 2 |
| Esther | 40 | F | NO | NO | NO | NO | 7100 | 65 | 31 | 4 |
| Laila | 40 | F | NO | NO | NO | NO | 9000 | 70 | 26 | 4 |
| Dhandapani | 40 | M | YES | NO | YES | YES | 7800 | 68 | 30 | 2 |
| Vellaiyan | 40 | M | YES | NO | YES | YES | 7400 | 70 | 28 | 2 |
| Rajasekar | 42 | M | NO | NO | NO | YES | 5800 | 66 | 32 | 4 |
| Mayandi | 44 | M | YES | NO | NO | YES | 6500 | 60 | 36 | 4 |
| Akbar | 44 | M | NO | NO | NO | YES | 7000 | 72 | 26 | 2 |
| Soodamani | 45 | F | NO | YES | NO | NO | 5800 | 78 | 20 | 2 |
| Kannan | 45 | M | NO | NO | YES | YES | 6800 | 66 | 30 | 4 |
| Sankar | 45 | M | YES | NO | NO | YES | 7540 | 65 | 31 | 4 |
| Pandian | 45 | M | NO | YES | YES | YES | 7500 | 68 | 30 | 2 |
| Manikandan | 48 | M | YES | NO | YES | YES | 6500 | 72 | 24 | 4 |
| Kala | 50 | F | NO | NO | NO | NO | 8100 | 65 | 30 | 5 |
| Parvadham | 50 | F | YES | NO | NO | NO | 6800 | 64 | 32 | 4 |
| Veearapandi | 55 | M | NO | YES | YES | YES | 8000 | 68 | 30 | 2 |
| Sadasivam | 65 | M | NO | NO | YES | YES | 7000 | 62 | 36 | 2 |
| Sarala | 70 | F | NO | YES | NO | NO | 6450 | 60 | 36 | 4 |

ECHO

| | |
|---|---|
| NO RWMA | 1 |
| RWMA | 2 |
| MILD LV DYSFUNCTION | 3 |
| MODERATE LV DYSFUNCTION | 4 |
| SEVERE LV DYSFUNCTION | 5 |

| PLATELET COUNT | SUGAR | UREA | CREATININT | TB | DB | OT | PT | TGL | TC | HDL | MPV | PDW | P-LCR | ECHO |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 220000 | 102 | 34 | 0.8 | 0.8 | 0.2 | 45 | 45 | 142 | 134 | 54 | 7.2 | 12.4 | 22.5 | 1 |
| 180000 | 98 | 36 | 1.1 | 0.8 | 0.1 | 40 | 48 | 100 | 164 | 52 | 7.4 | 12.6 | 22.4 | 1 |
| 200000 | 102 | 40 | 0.9 | 1 | 0.2 | 45 | 46 | 120 | 126 | 64 | 7.3 | 12.4 | 22.1 | 1 |
| 250000 | 98 | 28 | 0.6 | 0.9 | 0.2 | 50 | 42 | 112 | 108 | 45 | 8.2 | 13.4 | 24.2 | 1 |
| 220000 | 100 | 30 | 0.6 | 0.8 | 0.1 | 44 | 42 | 144 | 153 | 59 | 8.4 | 13.6 | 25.1 | 1 |
| 178000 | 246 | 24 | 0.8 | 0.8 | 0.2 | 48 | 40 | 140 | 165 | 54 | 8.4 | 14 | 24.5 | 1 |
| 240000 | 140 | 24 | 0.6 | 0.8 | 0.1 | 48 | 43 | 133 | 150 | 69 | 8.1 | 13.6 | 24.6 | 1 |
| 300000 | 102 | 28 | 0.6 | 0.9 | 0.1 | 45 | 48 | 130 | 200 | 68 | 7.5 | 12.9 | 22.5 | 1 |
| 210000 | 90 | 28 | 0.8 | 0.8 | 0.1 | 48 | 44 | 124 | 166 | 60 | 8 | 12.8 | 21.8 | 1 |
| 240000 | 98 | 28 | 0.6 | 0.9 | 0.1 | 45 | 45 | 182 | 155 | 58 | 7.4 | 12.4 | 21.9 | 1 |
| 198000 | 104 | 30 | 0.8 | 1 | 0.2 | 42 | 46 | 112 | 123 | 48 | 7.4 | 12.8 | 23.1 | 1 |
| 164000 | 84 | 38 | 0.8 | 0.9 | 0.2 | 40 | 47 | 154 | 120 | 68 | 7.8 | 13 | 22.5 | 1 |
| 145000 | 102 | 26 | 0.8 | 0.8 | 0.1 | 46 | 42 | 120 | 162 | 68 | 7.7 | 12.9 | 23.1 | 1 |
| 220000 | 114 | 24 | 0.8 | 0.8 | 0.1 | 48 | 46 | 102 | 165 | 48 | 8.2 | 13.8 | 25.4 | 1 |
| 168000 | 89 | 24 | 0.8 | 1 | 0.1 | 45 | 48 | 135 | 184 | 65 | 7.6 | 12.6 | 22.4 | 1 |
| 240000 | 120 | 28 | 0.8 | 0.8 | 0.1 | 49 | 48 | 144 | 165 | 62 | 7 | 12.1 | 21.4 | 1 |
| 240000 | 182 | 34 | 0.9 | 0.9 | 0.2 | 42 | 48 | 144 | 124 | 60 | 7.6 | 12.4 | 23.4 | 1 |
| 190000 | 114 | 34 | 0.8 | 0.8 | 0.2 | 45 | 48 | 122 | 179 | 62 | 7.8 | 12.5 | 22.3 | 1 |
| 180000 | 96 | 22 | 0.9 | 1 | 0.1 | 45 | 42 | 120 | 157 | 66 | 7.9 | 13.4 | 23.7 | 1 |
| 204000 | 98 | 32 | 0.6 | 1 | 0.2 | 44 | 44 | 120 | 175 | 65 | 8.1 | 13.6 | 24.5 | 1 |
| 212000 | 96 | 32 | 0.9 | 0.9 | 0.1 | 48 | 42 | 135 | 168 | 56 | 7.8 | 12.8 | 22.9 | 1 |
| 168000 | 98 | 28 | 0.8 | 1 | 0.3 | 40 | 48 | 156 | 135 | 56 | 7.2 | 13.2 | 22.1 | 1 |
| 190000 | 86 | 28 | 0.8 | 0.9 | 0.1 | 42 | 45 | 130 | 168 | 62 | 8.5 | 14.1 | 25.6 | 1 |
| 260000 | 88 | 34 | 0.6 | 0.9 | 0.1 | 42 | 46 | 100 | 123 | 64 | 8.2 | 13.8 | 24.1 | 1 |
| 200000 | 84 | 30 | 0.8 | 1 | 0.1 | 40 | 49 | 142 | 120 | 54 | 7.2 | 12.1 | 22.4 | 1 |
| 200000 | 96 | 26 | 0.8 | 0.8 | 0.1 | 44 | 40 | 100 | 135 | 56 | 8.1 | 13.9 | 24.5 | 1 |
| 240000 | 100 | 28 | 0.8 | 0.9 | 0.1 | 48 | 42 | 130 | 168 | 68 | 8.2 | 13.5 | 24.2 | 1 |
| 148000 | 80 | 38 | 0.6 | 0.7 | 0.2 | 44 | 50 | 134 | 240 | 60 | 8.2 | 13.6 | 24.5 | 1 |
| 280000 | 104 | 28 | 0.8 | 0.8 | 0.1 | 46 | 40 | 120 | 158 | 68 | 7.9 | 13.1 | 23.1 | 1 |
| 146000 | 90 | 24 | 0.8 | 0.8 | 0.1 | 40 | 44 | 102 | 153 | 48 | 8.9 | 14 | 26.5 | 1 |
| 290000 | 88 | 38 | 0.6 | 0.8 | 0.1 | 40 | 44 | 134 | 148 | 64 | 7.8 | 13.2 | 23.5 | 1 |
| 198000 | 126 | 30 | 0.6 | 0.8 | 0.1 | 48 | 48 | 120 | 169 | 54 | 8.2 | 13.8 | 24.9 | 1 |

| PLATELET COUNT | SUGAR | UREA | CREATININT | TB | DB | OT | PT | TGL | TC | HDL | MPV | PDW | P-LCR | ECHO |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 180000 | 84 | 24 | 0.8 | 0.7 | 0.2 | 46 | 49 | 122 | 154 | 69 | 7.6 | 12.2 | 22.9 | 1 |
| 180000 | 89 | 34 | 0.6 | 0.8 | 0.1 | 44 | 48 | 124 | 158 | 54 | 7.9 | 12.4 | 22.6 | 1 |
| 340000 | 102 | 39 | 1 | 0.8 | 0.2 | 48 | 49 | 130 | 164 | 65 | 8.3 | 13.5 | 24.8 | 1 |
| 280000 | 88 | 26 | 0.8 | 1 | 0.2 | 44 | 45 | 110 | 189 | 60 | 7.4 | 12.7 | 22.7 | 1 |
| 240000 | 98 | 30 | 1 | 0.7 | 0.1 | 40 | 42 | 102 | 185 | 58 | 7.8 | 13.2 | 22.7 | 1 |
| 212000 | 102 | 30 | 0.6 | 0.9 | 0.1 | 45 | 42 | 150 | 124 | 68 | 7.6 | 12.4 | 22.8 | 1 |
| 190000 | 142 | 26 | 0.9 | 0.8 | 0.2 | 44 | 47 | 126 | 147 | 56 | 7.8 | 12.6 | 22.9 | 1 |
| 200000 | 76 | 28 | 0.6 | 0.8 | 0.1 | 48 | 42 | 132 | 186 | 67 | 8.2 | 13.8 | 24.4 | 1 |
| 200000 | 148 | 28 | 0.8 | 1 | 0.1 | 45 | 45 | 142 | 124 | 65 | 9.2 | 14.2 | 27.5 | 3 |
| 168000 | 98 | 34 | 0.8 | 0.9 | 0.1 | 42 | 45 | 142 | 195 | 64 | 7.5 | 12.7 | 22.1 | 1 |
| 200000 | 86 | 24 | 0.7 | 0.7 | 0.1 | 40 | 44 | 124 | 124 | 62 | 7.8 | 12.4 | 22.6 | 1 |
| 260000 | 140 | 30 | 0.8 | 0.9 | 0.1 | 42 | 40 | 100 | 146 | 56 | 8.1 | 13.4 | 24.8 | 1 |
| 250000 | 88 | 26 | 0.8 | 0.9 | 0.1 | 40 | 45 | 130 | 164 | 59 | 9.1 | 14.4 | 27.5 | 1 |
| 245000 | 142 | 35 | 1 | 1 | 0.1 | 46 | 49 | 100 | 154 | 54 | 6.8 | 11.8 | 21.4 | 3 |
| 180000 | 86 | 32 | 0.7 | 1 | 0.2 | 44 | 48 | 104 | 129 | 62 | 7.5 | 12.8 | 22.9 | 1 |
| 250000 | 86 | 30 | 0.9 | 1 | 0.2 | 45 | 46 | 122 | 124 | 58 | 8.5 | 14.6 | 25.1 | 1 |
| 180000 | 88 | 28 | 0.9 | 1 | 0.1 | 44 | 44 | 132 | 142 | 50 | 7.8 | 13.4 | 22.9 | 1 |
| 220000 | 124 | 32 | 1 | 0.9 | 0.2 | 42 | 45 | 120 | 168 | 68 | 7.6 | 12.8 | 21.8 | 3 |

| NAME | AGE | SEX | YPERTENSI | DIABETES | SMOKING | ALCOHOL | BP | PULSE | OTAL COUN | P | L | E | ATELET COU |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Subramani | 40 | M | YES | NO | YES | NO | 110/80 | 90 | 7400 | 66 | 32 | 2 | 250000 |
| Thirulokchander | 55 | M | NO | NO | NO | NO | 120/90 | 96 | 8400 | 64 | 34 | 2 | 246000 |
| Ettiappan | 34 | M | NO | YES | YES | YES | 140/90 | 102 | 7000 | 69 | 28 | 3 | 348000 |
| Sampath | 45 | M | YES | YES | NO | YES | 150/90 | 90 | 6800 | 72 | 26 | 2 | 246000 |
| Vanmathi | 58 | F | YES | NO | NO | NO | 120/80 | 90 | 6400 | 70 | 26 | 4 | 184000 |
| Pradeep | 60 | M | YES | NO | YES | YES | 110/90 | 84 | 7200 | 68 | 30 | 2 | 222000 |
| Madasamy | 38 | M | NO | NO | YES | NO | 110/90 | 96 | 7800 | 66 | 30 | 4 | 150000 |
| Kavitha | 50 | F | NO | YES | NO | NO | 120/80 | 96 | 6200 | 64 | 32 | 4 | 186000 |
| Seethammal | 42 | F | YES | YES | NO | NO | 160/100 | 90 | 9000 | 60 | 36 | 4 | 240000 |
| Rajavel | 40 | M | YES | NO | NO | YES | 140/90 | 90 | 5400 | 68 | 30 | 2 | 240000 |
| Kannappan | 56 | M | YES | NO | YES | NO | 120/90 | 102 | 8000 | 67 | 30 | 3 | 310000 |
| Patrik | 35 | M | NO | YES | YES | NO | 130/90 | 78 | 7400 | 66 | 31 | 3 | 180000 |
| Raja | 38 | M | YES | NO | NO | YES | 140/90 | 84 | 6800 | 58 | 36 | 6 | 168000 |
| Mumtaj | 45 | F | YES | NO | NO | NO | 110/90 | 84 | 7400 | 66 | 32 | 4 | 240000 |
| Makimairaj | 40 | M | NO | YES | NO | YES | 110/90 | 90 | 8000 | 69 | 30 | 1 | 256000 |
| Chandran | 30 | M | NO | YES | YES | YES | 120/80 | 78 | 7400 | 67 | 30 | 3 | 190000 |
| Parthasarathy | 36 | M | YES | NO | YES | YES | 130/90 | 72 | 7200 | 72 | 26 | 2 | 168000 |
| Kamatchi | 34 | F | NO | NO | NO | NO | 120/80 | 84 | 8000 | 71 | 26 | 3 | 280000 |
| Kanagaraj | 72 | M | NO | YES | YES | NO | 120/90 | 90 | 8100 | 68 | 30 | 2 | 220000 |
| Siva | 50 | M | YES | NO | YES | YES | 140/90 | 84 | 6900 | 69 | 28 | 3 | 196000 |
| Elavarasan | 45 | M | NO | NO | YES | NO | 110/80 | 78 | 8200 | 65 | 30 | 5 | 150000 |
| Mala | 38 | F | NO | NO | NO | NO | 120/90 | 96 | 8400 | 58 | 36 | 6 | 268000 |
| Latha | 54 | F | YES | NO | NO | NO | 160/90 | 84 | 6900 | 70 | 28 | 2 | 240000 |
| Stalin | 35 | M | YES | NO | YES | YES | 140/90 | 78 | 7500 | 70 | 28 | 2 | 300000 |
| Kalaivani | 48 | F | YES | NO | NO | NO | 140/90 | 72 | 7700 | 72 | 26 | 2 | 190000 |
| Veera | 46 | M | YES | YES | YES | NO | 110/90 | 90 | 8000 | 66 | 30 | 4 | 178000 |
| Karunakaran | 38 | M | YES | NO | NO | YES | 140/80 | 84 | 9200 | 68 | 30 | 2 | 250000 |
| Vanitha | 40 | F | YES | YES | NO | NO | 150/90 | 102 | 8600 | 64 | 32 | 4 | 286000 |
| Lingam | 44 | M | YES | YES | YES | YES | 110/80 | 96 | 8400 | 72 | 22 | 6 | 175000 |
| Elumalai | 38 | M | NO | NO | YES | YES | 110/80 | 90 | 7500 | 70 | 26 | 4 | 200000 |
| Thiruveni | 50 | F | NO | YES | NO | NO | 130/90 | 84 | 6800 | 66 | 30 | 4 | 280000 |

| NAME | AGE | SEX | YPERTENSI | DIABETES | SMOKING | ALCOHOL | BP | PULSE | OTAL COUN | P | L | E | ATELET COU |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Vetrivel | 52 | M | YES | YES | YES | YES | 150/90 | 90 | 8500 | 68 | 30 | 2 | 159000 |
| Ayyanar | 46 | M | YES | YES | YES | NO | 100/80 | 90 | 7900 | 68 | 30 | 2 | 180000 |
| Geetha | 38 | F | NO | YES | NO | NO | 100/80 | 78 | 5800 | 59 | 38 | 3 | 290000 |
| Parthiban | 45 | M | YES | YES | NO | YES | 140/90 | 78 | 7500 | 70 | 28 | 2 | 286000 |
| Raniammal | 40 | F | NO | YES | NO | NO | 110/80 | 72 | 8100 | 70 | 28 | 2 | 190000 |
| Imran | 37 | M | YES | YES | YES | YES | 150/80 | 84 | 6800 | 64 | 32 | 4 | 286000 |
| Janakiraman | 39 | M | YES | YES | YES | YES | 110/80 | 90 | 7400 | 66 | 32 | 4 | 290000 |
| Babu | 46 | M | NO | YES | NO | YES | 110/80 | 84 | 8000 | 66 | 30 | 4 | 195000 |
| Joseph | 67 | M | YES | NO | YES | YES | 150/90 | 78 | 5900 | 68 | 30 | 2 | 184000 |
| Vadivu | 60 | F | YES | NO | NO | NO | 160/100 | 90 | 6400 | 70 | 28 | 2 | 290000 |
| Rathinam | 54 | M | NO | NO | YES | NO | 120/80 | 90 | 6800 | 68 | 26 | 6 | 200000 |
| Velmurugan | 50 | M | YES | NO | YES | YES | 110/80 | 78 | 7200 | 70 | 28 | 2 | 185000 |
| Ramasamy | 38 | M | NO | NO | NO | YES | 120/80 | 72 | 8100 | 74 | 32 | 4 | 208000 |
| Karuthamma | 49 | F | NO | NO | NO | NO | 120/90 | 78 | 6800 | 69 | 30 | 1 | 195000 |
| Kalimuthu | 40 | M | NO | YES | YES | YES | 110/80 | 84 | 5700 | 66 | 30 | 4 | 280000 |
| Deenadayalan | 38 | M | YES | YES | YES | YES | 170/100 | 90 | 8200 | 64 | 32 | 4 | 200000 |
| Venkat | 56 | M | YES | NO | YES | NO | 140/90 | 78 | 7500 | 66 | 30 | 4 | 156000 |
| Parameswaran | 48 | M | YES | NO | YES | YES | 130/90 | 84 | 8400 | 60 | 36 | 4 | 286000 |
| Thenali | 40 | M | YES | YES | YES | YES | 120/90 | 90 | 7600 | 64 | 34 | 2 | 240000 |

ECHO

| | |
|---|---|
| NO RWMA | 1 |
| RWMA | 2 |
| MILD LV DYSFUNCTION | 3 |
| MODERATE LV DYSFUNCTION | 4 |
| SEVERE LV DYSFUNCTION | 5 |

| SUGAR | UREA | EATINI | TB | DB | OT | PT | TC | TGL | HDL | MPV | PDW | P-LCR | ECHO |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 96 | 28 | 0.6 | 0.9 | 0.1 | 34 | 36 | 186 | 120 | 64 | 7.8 | 11.8 | 23.2 | 1 |
| 102 | 30 | 0.8 | 0.9 | 0.2 | 36 | 38 | 154 | 140 | 66 | 8.2 | 12.4 | 25.6 | 1 |
| 110 | 26 | 0.9 | 0.8 | 0.1 | 40 | 40 | 200 | 112 | 54 | 7.6 | 11.8 | 22.9 | 1 |
| 90 | 30 | 1 | 1 | 0.1 | 39 | 35 | 240 | 162 | 68 | 8 | 12.6 | 23.5 | 1 |
| 80 | 32 | 0.6 | 0.7 | 0.1 | 38 | 36 | 186 | 140 | 44 | 9.2 | 13.8 | 27.2 | 1 |
| 109 | 22 | 0.5 | 0.8 | 0.2 | 35 | 38 | 154 | 100 | 40 | 8.8 | 12.8 | 25.8 | 1 |
| 120 | 28 | 0.8 | 0.8 | 0.2 | 34 | 39 | 170 | 114 | 58 | 7.6 | 12.2 | 22.8 | 1 |
| 180 | 22 | 0.4 | 0.9 | 0.1 | 36 | 35 | 164 | 125 | 44 | 8.4 | 12.8 | 25.5 | 1 |
| 102 | 26 | 0.6 | 0.8 | 0.1 | 39 | 34 | 200 | 142 | 52 | 8.2 | 11.9 | 24.9 | 3 |
| 99 | 30 | 1 | 1 | 0.2 | 35 | 38 | 140 | 105 | 44 | 9.4 | 14 | 28.2 | 1 |
| 140 | 32 | 0.9 | 1 | 0.1 | 38 | 35 | 184 | 134 | 48 | 7.4 | 11.6 | 22.5 | 1 |
| 110 | 28 | 0.8 | 0.8 | 0.1 | 37 | 35 | 154 | 110 | 62 | 8.2 | 12.6 | 24.6 | 1 |
| 98 | 25 | 0.8 | 0.9 | 0.1 | 35 | 38 | 164 | 142 | 42 | 8.6 | 12.8 | 25.2 | 1 |
| 108 | 22 | 0.6 | 0.7 | 0.1 | 36 | 39 | 174 | 186 | 40 | 7.9 | 11.8 | 22.4 | 1 |
| 240 | 26 | 0.8 | 1 | 0.2 | 40 | 42 | 180 | 120 | 48 | 8.4 | 12.6 | 24.4 | 1 |
| 98 | 30 | 0.8 | 1.1 | 0.3 | 42 | 40 | 240 | 135 | 58 | 9.1 | 13.9 | 27.2 | 1 |
| 134 | 32 | 0.9 | 0.9 | 0.2 | 44 | 48 | 148 | 140 | 56 | 8.6 | 12.5 | 25.4 | 1 |
| 86 | 32 | 1 | 0.9 | 0.1 | 41 | 40 | 168 | 120 | 66 | 7.9 | 11.8 | 22.8 | 1 |
| 145 | 24 | 0.8 | 0.8 | 0.1 | 40 | 38 | 148 | 100 | 68 | 7.6 | 12 | 22.4 | 3 |
| 84 | 22 | 0.8 | 0.9 | 0.2 | 35 | 39 | 164 | 124 | 59 | 8.2 | 12.6 | 24.8 | 1 |
| 90 | 28 | 0.8 | 1 | 0.1 | 36 | 38 | 200 | 135 | 67 | 8.8 | 13.4 | 25.2 | 1 |
| 90 | 30 | 1 | 0.8 | 0.1 | 38 | 38 | 154 | 142 | 45 | 9.2 | 14.2 | 28 | 1 |
| 110 | 30 | 0.9 | 1.1 | 0.2 | 36 | 40 | 186 | 108 | 68 | 8.6 | 13.8 | 25.4 | 1 |
| 85 | 24 | 0.6 | 0.8 | 0.1 | 34 | 40 | 248 | 154 | 56 | 8.8 | 14 | 25.1 | 1 |
| 80 | 24 | 0.8 | 0.9 | 0.2 | 40 | 42 | 174 | 120 | 68 | 7.6 | 12.4 | 22.4 | 1 |
| 148 | 22 | 0.8 | 0.8 | 0.1 | 42 | 39 | 164 | 220 | 64 | 9 | 14.2 | 27.4 | 1 |
| 125 | 29 | 1 | 0.8 | 0.1 | 35 | 38 | 180 | 145 | 50 | 8.4 | 13.6 | 25.2 | 1 |
| 220 | 21 | 0.6 | 0.9 | 0.2 | 36 | 38 | 142 | 168 | 40 | 7.8 | 12.8 | 22.6 | 3 |
| 96 | 20 | 0.6 | 0.9 | 0.2 | 38 | 40 | 175 | 120 | 54 | 8 | 12.2 | 24.8 | 1 |
| 142 | 28 | 0.8 | 1 | 0.1 | 34 | 40 | 164 | 109 | 64 | 8.2 | 12.6 | 24.8 | 1 |
| 102 | 25 | 0.8 | 1 | 0.2 | 42 | 38 | 234 | 120 | 48 | 8.6 | 13.4 | 25.4 | 1 |

| SUGAR | UREA | EATINI | TB | DB | OT | PT | TC | TGL | HDL | MPV | PDW | P-LCR | ECHO |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 245 | 23 | 0.6 | 0.8 | 0.1 | 40 | 38 | 182 | 107 | 44 | 8.2 | 13.4 | 25.6 | 3 |
| 140 | 22 | 0.8 | 0.7 | 0.1 | 42 | 42 | 168 | 132 | 46 | 7.4 | 12.2 | 22.8 | 1 |
| 96 | 28 | 1 | 0.7 | 0.1 | 40 | 39 | 124 | 110 | 40 | 7.2 | 11.9 | 22.4 | 1 |
| 186 | 26 | 0.8 | 0.9 | 0.2 | 30 | 35 | 169 | 122 | 56 | 9.2 | 14.2 | 27.5 | 1 |
| 120 | 24 | 0.8 | 1 | 0.1 | 35 | 40 | 174 | 114 | 58 | 8.4 | 12.8 | 25.6 | 1 |
| 155 | 21 | 0.6 | 0.9 | 0.2 | 36 | 40 | 242 | 158 | 45 | 8.6 | 13.2 | 25.2 | 1 |
| 84 | 28 | 1 | 0.9 | 0.1 | 35 | 38 | 154 | 164 | 44 | 7.2 | 12.1 | 22.6 | 4 |
| 98 | 34 | 1.2 | 0.8 | 0.1 | 34 | 38 | 280 | 100 | 64 | 8 | 13.2 | 24.8 | 1 |
| 80 | 28 | 0.9 | 1 | 0.2 | 40 | 42 | 146 | 175 | 40 | 7.8 | 12.4 | 22.6 | 1 |
| 140 | 36 | 0.8 | 0.8 | 0.1 | 42 | 38 | 225 | 122 | 48 | 7.4 | 12.2 | 23 | 1 |
| 90 | 29 | 0.8 | 0.9 | 0.2 | 42 | 36 | 146 | 104 | 68 | 8.2 | 13.2 | 24.4 | 1 |
| 79 | 21 | 0.6 | 0.8 | 0.1 | 35 | 40 | 154 | 112 | 50 | 8.6 | 13.4 | 25.2 | 3 |
| 110 | 30 | 0.8 | 0.7 | 0.1 | 36 | 42 | 220 | 145 | 52 | 9.4 | 14.2 | 28.2 | 1 |
| 84 | 34 | 1 | 0.9 | 0.2 | 38 | 40 | 188 | 119 | 44 | 7.6 | 11.9 | 22.4 | 1 |
| 190 | 27 | 0.6 | 1 | 0.1 | 48 | 38 | 168 | 124 | 58 | 8.2 | 13.3 | 24.8 | 1 |
| 154 | 25 | 0.8 | 0.9 | 0.2 | 42 | 40 | 169 | 104 | 40 | 8.4 | 13.9 | 25.2 | 1 |
| 98 | 34 | 1.1 | 1 | 0.1 | 42 | 38 | 245 | 144 | 42 | 7.8 | 12.8 | 22.8 | 1 |
| 88 | 26 | 0.8 | 0.9 | 0.1 | 40 | 38 | 180 | 186 | 64 | 8.2 | 13.4 | 25.8 | 1 |
| 124 | 28 | 0.9 | 0.8 | 0.2 | 47 | 46 | 165 | 180 | 58 | 8.4 | 13.8 | 25.6 | 1 |

# INSTITUTIONAL ETHICS COMMITTEE
# MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No: 04425305301
Fax : 044 25363970

## CERTIFICATE OF APPROVAL

To
Dr. Venkatesan. S
PG in MD General Medicine
Madras Medical College, Chennai -3.

Dear Dr. Venkatesan. S

The Institutional Ethics Committee of Madras Medical College reviewed and discussed your application for approval of the proposal entitled "A study on mean platelet volume in acute coronary syndrome" No. 12042011.

The following members of Ethics Committee were present in the meeting held on 21.04.2011 conducted at Madras Medical College, Chennai -3.

| | | |
|---|---|---|
| 1. | Prof. S.K. Rajan, MD | -- Chairperson |
| 2. | Prof. V. Kanagasabai MD<br>Dean, Madras Medical College, Chennai-3, | -- Deputy chairman |
| 3. | Prof. A. Sundaram, MD<br>Vice Principal , Madras Medical College, Chennai -3 | -- Member Secretary |
| 4. | Prof R. Sathianathan MD | -- Member |
| 5. | Prof R. Nandhini, MD<br>Director, Institute of Pharmacology, MMC, Ch-3 | -- Member |
| 6. | Prof. Pregna B. Dolia MD<br>Director , Institute of Biochemistry, MMC, Ch-3 | -- Member |
| 7. | Prof. C. Rajendiran .MD<br>Director , Institute of Internal Medicine, MMC, Ch-3 | -- Member |
| 8. | Thiru. A. Ulaganathan<br>Administrative Officer, MMC, Chennai -3 | -- Layperson |
| 9. | Thiru. S. Govindasamy . BA.BL | -- Lawyer |
| 10. | Tmt. Arnold Soulina | -- Social Scientist |

We approve the proposal to be conducted in its presented form.

Sd /. Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report

Member Secretary, Ethics Committee