A STUDY ON PLATELET COUNT AND THEIR INDICES-MEAN PLATELET VOLUME AND PLATELET DISTRIBUTION WIDTH AND THEIR PROGNOSTIC SIGNIFICANCE IN ACUTE CORONARY SYNDROME IN TERTIARY CARE HOSPITAL

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CERTIFICATE

This is to certify that the dissertation titled **"A STUDY ON PLATELET COUNT AND THEIR INDICES-MEAN PLATELET VOLUME AND PLATELET DISTRIBUTION WIDTH AND THEIR PROGNOSTIC SIGNIFICANCE IN ACUTE CORONARY SYNDROME IN TERTIARY CARE HOSPITAL"** is a bonafide work done **by Dr.M.ANITHA**, Post graduate student, Institute of Internal Medicine, Madras Medical College, Chennai -03, in partial fulfillment of the University Rules and Regulations for the award of Degree of MD General Medicine (Branch – I),Internal Medicine, under our guidance and supervision, during the academic year 2014– 2017.

Prof. Dr.S.MAYILVHANAN M.   **Prof.Dr.G.SUNDARAMURTHYM.D**

Director and Professor,   Professor of medicine,

Institute of Internal Medicine,   Institute of Internal Medicine

Madras Medical College &   Madras Medical College &

Rajiv Gandhi Govt General hospital   Rajiv Gandhi Govt General Hospital

Chennai – 600 003   Chennai – 600 003

Prof. Dr.M.K. MURALITHARAN MS, MCH

DEAN

Madras Medical College &

Rajiv Gandhi Government General Hospital

Chennai – 600 003

DECLARATION

I solemnly declare that the dissertation titled "**. A STUDY ONPLATELET COUNT AND THEIR INDICES-MEAN PLATELET VOLUMEAND PLATELET DISTRIBUTION WIDTH AND THEIR PROGNOSTIC SIGNIFICANCE IN ACUTE CORONARY SYNDROME IN TERTIARY CARE HOSPITAL** "is done by me at Madras Medical College Chennai –3 during the period April 2016 to September 2016 under the guidance and supervision of **Prof. Dr. G. SUNDARAMURTHY** submitted to the Tamilnadu Dr.M.G.RMedical University towards the partial fulfillment of requirements for the award of M.D. DEGREE IN GENERAL MEDICINE (BRANCH-I) .

Place : Chennai   **Dr.M.ANITHA**

Date   :   Post Graduate,

                                M.D. General Medicine,

                                Rajiv Gandhi Govt. General Hospital

                                Chennai – 600003

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Last but not the least, I wish to thank all the patients without whom the study would have been impossible.

CONTENTS

INTRODUCTION

INTRODUCTION

Cardiovascular disease is an epidemic of modern society. Myocardial infarction is a major cause of morbidity and mortality in the world. Despite the impressive strides in diagnosis and management over the past four decades, acute myocardial infarction continues to be a major public health problem in industrialized world and is becoming an increasingly important problem in developing countries. In United States, 13 million individuals have ischemic heart disease,>6 million have angina pectoris,>7 million have persistent myocardial infarction. Because acute myocardial infarction may strike an individual during productive years, it can have profoundly deleterious psychosocial and economic ramifications.

After rupture of an atherosclerotic plaque in a coronary artery, there is platelet adhesion, activation and aggregation leading to the formation of a thrombus and ultimately culminating in acute myocardial infarction. So the more reactive the platelet are, higher the chances for myocardial infarction.

Assiri et al (2011) stated that platelet parameters mainly MPV and PDW are inexpensive lab tests which we detected to be significantly

raised in patient suffered from acute coronary syndrome compared with controls.

Jasmine Jasani et al (2014) stated that acute coronary syndrome had higher platelet volume indices and lower platelet counts compared with those with stable angina and normal population. Measurements of platelet volume indices and platelet count benefit in detecting patients at higher risk for coronary events.

S.C.Costa et al(2015) states that platelet indices as additional and complementary marker as thrombotic risk in acute coronary syndrome. Correlates macro platelets with higher thrombotic potential.

So in this observational study, associations between platelet counts, mean Platelet volume and platelet distribution width as thrombotic risk in acute coronary syndrome were investigated in tertiary care hospital patient.

AIMS AND OBJECTIVES

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- To study the effect of platelet count and their indices-mean platelet volume and platelet distribution width and their prognostic significance in acute coronary syndrome.

- To assess outcome of coronary heart disease patient.

- To analyse the patient's treatment pattern and follow-up arrangement.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The role of platelet in acute coronary syndrome has been appreciated for several decades. But the last 10-15 years have seen dramatic increase in understanding, development, clinical evaluation and therapeutic application of platelet inhibitor therapy.

Platelet released from megakaryocytes in the bone marrow play an essential role in haemostasis, thrombosis and coagulation of blood. These tiny cells previously described as" sponges" are known to engage in a complex repertoire of biochemical and molecular activities designed to prevent haemorrhage. (Adelson et ai,1961)[1]

Platelet indices indicates differentiation dysfunction, hyperactivation, aggregation or adhesion express disease pathogenesis (Maitree Bhattacharya et al, 2013)[2]

Platelets have major role in the pathogenesis of acute coronary syndrome, where plaque rupture is followed by platelet activation and thrombus formation leading to coronary artery occlusion.[3],[4],[5].

Anti platelet agents have a cardinal role in management of patients with ACS by interrupt the pathologic cascade of thrombosis. Therefore,

acetylsalicylic acid, thenopyridine and glycoprotein 2b/3a inhibitors are platelet inhibitors used in the treatment of ACS patients.[6]

HISTORICAL ASPECTS

Historically, platelets were referred to as cellular dust.[7] The classification of platelet as blood corpuscles described by Bizzozero (1882).The importance of platelet to form haemostatic plug was reported by Eberth and scimmelbusch (1888).Another milestone in platelet involved in thrombogenesis identified by Aschoff(1925).

PLATELET STRUCTURAL AND FUNCTIONAL ANATOMY LIGHT MICROSCOPY

On Wright-Giemsa stained blood smears, platelets appear as small anucleate, ovoid or round cells, with a pale greyish blue cytoplasm that contains homogeneously distributed purple –red granules.

DIMENSIONS

The volumes of circulating platelets from a single individual are heterogeneous and exhibit a lognormal size distribution; and the platelet volume in a single individual (mean platelet volume, MPV) varies from 7 to 9 femtolitres.(paulus,1975 et al[8],Martin et al 1982[9];Stenberg and Lenin 1989[10]; corash, 1977)[11].

ELECTRON MICROSCOPY AND SUBCELLULAR ORGANELLES

By scanning electron microscopy, circulating platelet appear as flat discs, with smooth contours and rare spiny filopodia. Scanning electron microscopy also reveals random openings of a channel system, the surface connected cannalicular system,which invaginates throughout the platelet and is conduit by which granule contents exocytose after stimulation. Although the platelet is anucleate,transmission electron microscopy reveals a cytoplasm packed with a number of different organelles essential to the maintenance of normal haemostasis.

GLYCOCALYX

STRUCTURE: A glycocalyx 15 to 20 nm thick, is visualised by transmission electron microscopy and contains glycoprotein, glycolipids, mucopolysaccharides, and adsorbed plasma proteins.

FUNCTION: The glycocalyx has a net negative surface charge due to sialic acid residues on the proteins and lipids; the charge is thought to minimize attachment of circulating platelet to each other (coller ,1984)[12]. This structure is rich in carbohydrate moieties of membrane –associated glycoproteins, which serve as receptors to mediate transfer of signals by

stimulatory agents .The glycocalyx interacts with platelet activators to facilitate platelet adhesion and aggregation.

PLASMA MEMBRANE

STRUCTURE:

The platelet plasma membrane is typical trilaminar membrane with glycoproteins, glycolipids, and cholesterol embedded in a phospholipids bilayer.

FUNCTION:

The plasma membrane contains sodium and calcium ATPase pumps, which are important for maintaining ionic homeostasis. It has a specialized role in providing a surface for the acceleration of blood coagulation, in that specific platelet coagulant protein, platelet factor 3, resides in this lipoprotein –rich unit membrane

SURFACE –CONNECTED CANALICULAR SYSTEM

STRUCTURE:

The surface –connected canalicular system, also called the open canalicular system, weaves throughout the cell cytoplasm in a tortuous fashion.

FUNCTION:

The functions of the surface connected canalicular system are to provide a route entry and egress for molecules, an internal reservoir of membrane to facilitate platelet spreading and filopodia formation after adhesion and storage reservoir for membrane glycoprotein's that increase on the platelet surface after activation.

DENSE TUBULAR SYSTEM

STRUCTURE:

Unlike the surface –connected canalicular system, the dense tubular system is a closed –channel system consisting of narrow, membrane limited tubules, approximately 400 to 600 A in diameter. It is infact, residual smooth endoplasmic reticulum from megakaryocytic.

FUNCTION:

This channel system is involved in the regulation of intracellular calcium transport because it has been reported to selectively bind, sequester, and release divalent cations after activation. The dense tubular system is also the site of prostaglandin synthesis in platelets.

CYTOSKELETON:

General structure:

The platelet cytoskeleton contains 30 to 50 % of total platelet protein and is made up of three major structural components: an actin microfilament network present throughout the cytoplasm, a micro tubule coil localized at the platelet periphery and a membrane skeleton comprising a network of short actin filaments that underlies the inner surface of the plasma membrane. Although they are distinct structures, interconnections between these elements are present.

STRUCTURE AND FUNCTION OF SPECIFIC CYTOSKELETAL ELEMENTS

Actin Microfilaments:

Twenty to thirty percent of total platelet protein is made up of actin (pollard, 1990).Actin exist in two forms ,G-actin (actin monomers) and F-actin (polymerized actin). In the unstimulated platelet,30-40% of actin is polymerized into filaments; the balance of actin monomers are prevented from polymerizing by protein such as profiling or thymosin B4 that sequester nonnumeric actin, or by proteins that cap filaments in the intact cell, such as gelsolin.

Upon platelet activation, the proportion of filamentous actin rapidly increases to 60-70%.Actin monomers polymerize into filaments at platelet peripheries and bundles of new filaments form to fill developing filopodia.

MICROTUBULES:

A circumferential microtubule band that supports the discoid form of the platelet is made up of two nonidentical subunit protein such as alpha and beta tubulin associated with microtubule associated protein (MAP s).The 25nm diameter microtubule coil lies adjacent to, but does not touch, plasma membrane.

Microtubule is present primarily in their polymerized form in unstimulated platelets. Platelet activation results in microtubule disassembly, then reassembly; such alterations in marginal microtubule bundle result in platelet shape changes.

Membrane skeleton:

The short actin filaments of the membrane skeleton, which underlie the inner surface of the plasma membrane, together with the microtubule coil, are thought to help stabilize the platelet discoid shape.

Two major platelet membrane glycoprotein, GP IIb-IIIa and GP-IX are associated with the membrane skeleton.

GRANULES

Platelet contain four distinct population of granules

1. Alpha

2. Dense bodies

3. Lyosomes

4. Microperoxisomes.

After platelet stimulation by agonist, granules fuse with channels of the surface –connected canalicular system and extrude their contents (white, 1974).Internal contraction is required for this extrusion and ultimate changes into the surrounding medium.

α - Granules:

Structure: α Granules are the predominant granule type in the platelet. The α granule has been subdivided morphologically into three distinct zones by electron microscopy:

1. Electron dense nucleoid that occupies the bulk of the granule

2. Peripheral zone of lower electron density that lies adjacent to the granule membrane

3. There are 1 to 6 tubular structures that reside in the electron lucent peripheral zone.

Content: B- thromboglobulin and platelet factor 4 have been localized to the dense nucleoid.Von willebrand factor is present in the tubular structures of the granule peripheral zone .Thrombospondin,and fibrinogen are present in the granular matrix. Other proteins present in α

Granules include albumin, immunoglobin G (Ig G), fibronectin, platelet derived growth factor ,GP IIb ,IIIa ,Beta amyloid protein precursor, factor V, multimerin, factor V/Va binding protein, transforming growth factor β1 and a plasminogen activator similar to tissue plasminogen activator.

Proteins present on the α –granule membrane include P – selectin, GPIIb / IIIa, granule membrane protein-33 (GMP-33), CD9, platelet –endothelial adhesion molecule 1 (PECAM-1) and osteonectin.

Dense Bodies:

Structure: Ultrastructurally, dense granules have a bull's eye appearance .They are the most electron -dense organelles in platelets.

Contents: The principal constituents of dense granules are a non metabolic pool of adenine nucleotides (adenosine triphosphate and diphosphate, ATP and ADP), PPi, calcium and magnesium and serotonin (5-hydroxy trytamine). In addition, dense bodies contain guanosine triphophate and diphosphate (GTP and GDP).The dense granule membrane contains P-selectin and granulophysin.

Lysosomes:

Structure: Liposome's are small vesicles of approximately 175 to 200 nm.

Content: Lysosomes are the only platelet granules that contain acid hydrolases. platelet lysosomes contain a large variety of enzymes, including β hexosaminidase and β glycerophosphatase. Lysosomal membrane glycoprotein (LIMP-CD63) and lysosomal associated membrane proteins 1 and 2 (LAMP-1 and LAMP-2) become expressed on the plasma membrane activation.

Microperoxisomes:

Stucture: Microperoxisomes are small (90nm) granules that are relatively few in number in platelets and can be demonstrated only cytochemically.

Content: They are reactive with alkaline diaminobenzidine medium. The enzyme responsible for the cytochemical peroxidise activity in microperoxisomes is catalase.

Coated vesicles:

Structure: coated **vesicles** are 70 to 90 organelles

Content: The polyhedral coat on the surface of these vesicles is composed of clathrin .Coated pits and vesicles transfer plasma components to platelet granule (Behnke, 1989)[13]

Mitochondria:

Structure: Mitochondria in platelet are similar, with the exception of smaller size to those in other cell types. There are approximately seven per human platelet.

Content: Mitochondria are the site of activity for all components of the respiratory chain and all almost all enzymes in the citric acid cycle.

Glycogen:

Platelet contains small particles of glycogen or masses of closely associated glycogen particles; these play a essential role in platelet metabolism**.**

PLATELET STRUCTURE
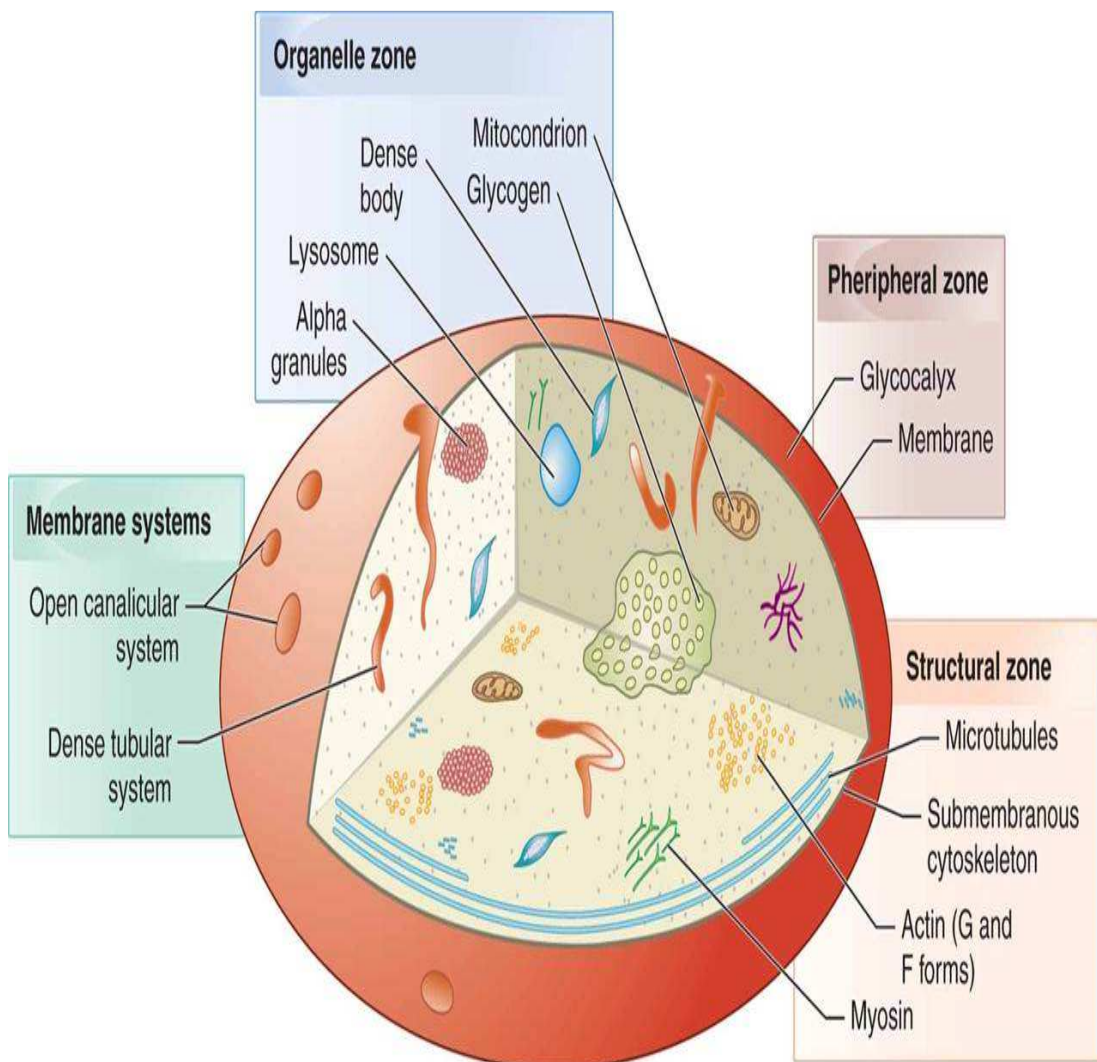
PLATELET PHYSIOLOGY

Platelet lipid and proteins:

Membrane lipids:

Phospholipids contributes about 80% of total platelet lipid, although smaller amounts of neutral lipids and glycolipids are also present. The five major phospholipids identified in human platelets are

1. phosphotidylcholine

2. phosphatidylethanolamine

3. spingomyelin

4. phosphatidylserine

5. phosphatidylinositol

Almost all platelet fatty acids are esterified in phospholipids, leaving only trace amounts of free fatty acids. Arachidonic acid, the precursor of prostaglandins and thromboxanes, is enriched in these phospholipids and the metabolism of arachidonic acid is critical for normal platelet function (Marcus,1976).[14]

Neutral lipids make up approximately 28% of total platelet lipids, the predominant neutral lipid being cholesterol.

Membrane Glycoproteins:

Platelet membrane glycoprotein's function mediates a wide number of adhesive cellular interactions. These glycoprotein's function as receptors that can receive signals from outside the platelet, facilitating cell-cell interactions; binding of specific ligands to these receptors results in distinct platelet responses to the external environment.

Glycoprotein IIb/IIIa :

Glycoprotein IIb-IIIa is the principal receptor on the platelet plasma membrane (Phillips et al,1988).[15]It is a member of the integrin family of proteins. A ca2+ dependent conformational change in GP IIa – IIIb after platelet agonist induced stimulation facilitates strong binding to fibrinogen and vWF resulting in cross linking of GP IIb-IIIa molecules on adjacent platelets and platelet aggregation.

Glycoprotein Ib-IX:

Glycoprotein Ib mediates the interaction of platelet with vWF. GPIb also functions as binding site for thrombin. GPIb is present on platelet surface in a 1:1 ratio with GP IX.

Other membrane Glycoprotein's:

Membrane glycoprotein GPI a-IIa,GPIc-IIa,mediate platelet adhesion to collagen, fibronectin, laminin and vitronectin.GP V forms a non covalent complex with GP IB-IX in the platelet membrane. PECAM -1 binds to heparin like molecules. GPIV is a receptor for thrombospondin.GP IV is also reported to bind collagen.

Other platelet proteins:

1. platelet factor 4

2.β thromboglobulin

3. thrombospondin

4. platelet derived growth factor

5. fibronectin

Platelet Factor 4:

platelet factor 4 binds with heparin neutralizes its anticoagulant activity also inhibits endothelial cell proliferation, migration, and angiogenesis, potentiate platelet aggregation.
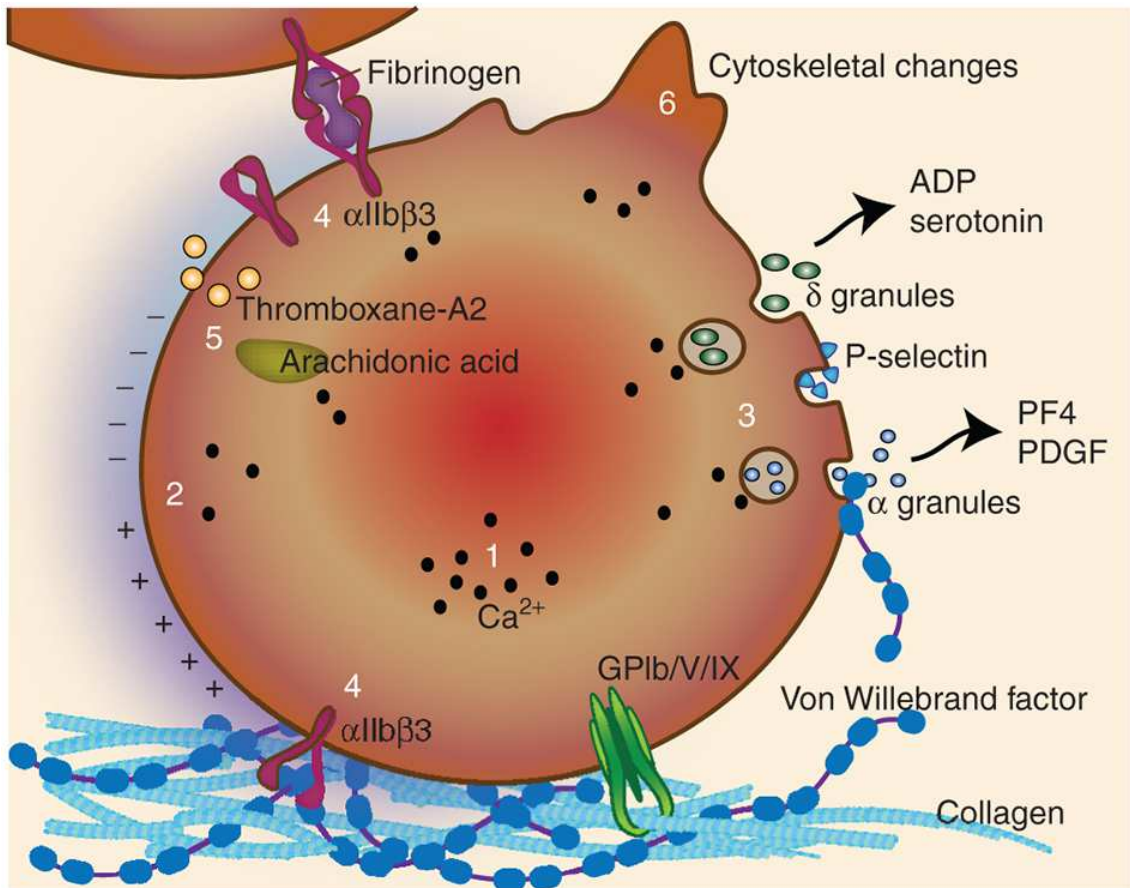
PLATELET BIOCHEMISTRY:

The platelet synthesize minimal protein because of less ability and also it contains low level of RNA and anucleate.As per its dry weight, platelet constitutes

- Carbohydrate -8%

- Protein -60%

- Lipid-15%

Minerals   -   Magnesium

              -      calcium

              -      potassium

              -      zinc

Vitamin-B12,folic acid, ascorbic acid.

PLATELET ENERGY METABOLISM:

Platelet has the energy metabolism almost similar to that of skeletal muscle. Both utilize glycolysis and synthesis and use excess amount of glycogen in both, the major mediator of intracellular energy use is an actomyosin-like adenosine triphsphatase. the platelet like that of muscle ,is metabolically active and expend excess energy rapidly during aggregation, the release, and clot retraction.

The major energy source for the platelet is glucose, which is rapidly taken up from the plasma.

Platelet energy metabolism is derived from glucose and to lesser extend from metabolism of fatty acid. Energy driven equal amount from glycolysis and citric acid cycle. Platelet energy reserve is provided by metabolic pool of platelet nucleotides that is continuous turnover.

Nucleotide metabolism:

Adenine nucleotides constitute 90% of free platelet nucleotides and are divided into two different pools. The metabolic or cytoplasmic pool constitutes 40 % of total adenine nucleotides. It is utilised for maintenance of various energy consuming cell functions and utilized during platelet release.

Arachidonate Metabolism:

Arachidonic acid is released from platelet membrane phospholipids after stimulation by enzymatic action of phospholipids A2 or phospholipase C and diglyceride lipase. After release, arachidonic acid can be acted by either lipoxygenase,results in production of peroxy and hydroxyl fatty acids or by cyclooxygenase ,which results in production of thromboxanes and prostaglandins.

Platelet "coagulation '' Factors;

Numerous platelet protein interact with plasma coagulation protein although the mechanism by which platelet membrane component become reorganized and capable of functioning as a catalytic surface for plasma protein are not known.

Plasma coagulation factors which are associated with platelets, including von Will brand factor, coagulation inhibitors, and factor XIII.

Numerous substance associated with or derived from platelet have been named as platelet factors 1 to 10 as Arabic numerals .The most important of these are PF4 and PF3.

Platelet Factor 3

PF 3 is needed in two process in blood coagulation system, which are interaction between factors IXa and VIIIa ,which results in activation a factor X, as well as interaction between Xa and factor Va which leads to formation of prothrombinase. These coagulation reaction are greatly accentuated on platelet surface.

PLATELET COUNT

The normal platelet count varies between 1,50,000 to 3,50,000/mm³

ORIGIN OF PLATELET FROM MEGAKARYOCYTES

The megakaryocyte is a large progenitor cells in the bone marrow, which is the source of platelets. Platelet released from megakaryocyte through a series of fascinating cell biological events. During maturation,

they become polypoid and accumulate massive amounts of protein and membrane.

Then, in a cytoskeletal –driven process, they extend long branching process, named as proplatelets, into sinusoidal blood vessels were they release platelets. The time required for megakaryocyte to complete polyploidization, mature, and release platelets is 5 days in human,2-3 days in rodents.(Ebbe and Stohlman ,1965; Odell and Jackson ,Odell et al.,1970)

FASCINATING CELL BIOLOGICAL EVENTS:

1. Megakaryocyte development in adult bone marrow

2. Endomitosis to create polypoid nucleus

3. cytoplasmic maturation

4. proplatelet formation and release

5. preplatelet to proplatelet interconversion

6. Platelet release

They yield about 1000 and 8000 platelets having volume of 7-9 femto litres each (Martin et al ,1982;Stenberg and Levin ,1989; Corash, 1989) Megakaryocytes are suicidal micro organs whose mission is to proliferate and then fragment their cytoplasm on demand to maintain blood platelets at steady level 1,50,000 to 3,50,000/mm³

Maintenance of platelet counts within this range represents a surplus of over 10 times that necessary to ensure routine haemostasis but providesa precautionary reserve for times of excess platelet loss or consumption.

THE INCREDIBLE JOURNEY: ORIGIN OF PLATELET FORMATION FROM MEGAKARYOCYTE

1. MK development in adult bone marrow
2. Endomitosis to create polyploid nucleus
3. Cytoplasmic maturation
4. Proplatelet formation and release
5. Preplatelet to proplatelet interconversion
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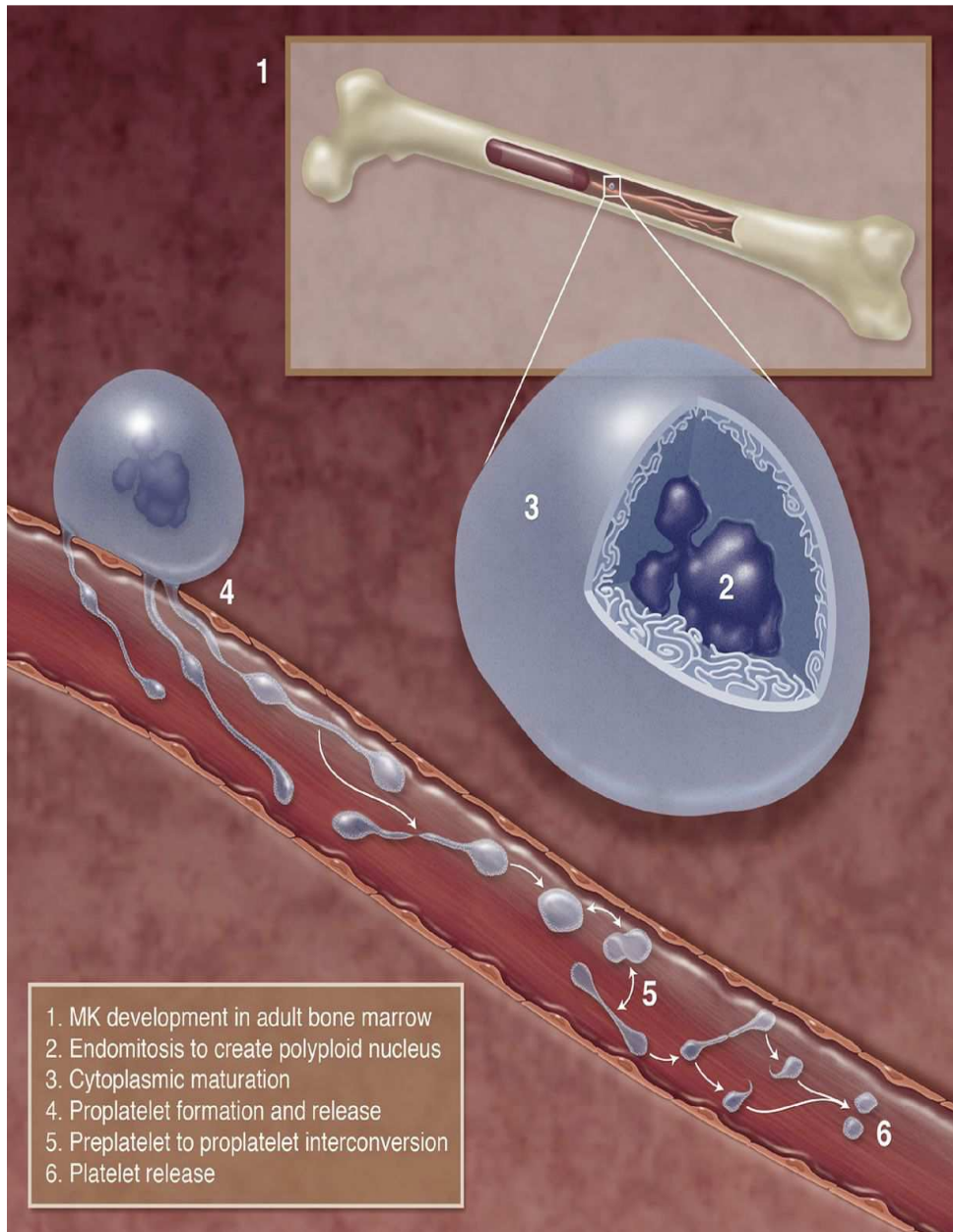
PLATELET LIFE SPAN, TURN OVER & REMOVAL:

Platelet life span, based on time needed to cleared from circulation, estimated approximately 8-12 days. The sites for platelet removal appear to be speen, liver and bone marrow. Degranulation and loss of density and platelet constituents has not been shown to decrease platelet life span indicating that number of haemostatic interactions may not be a key component.

PLATELET ADHESION, ACTIVATION & AGGREGATION:

The antithrombotic properties of intact vascular endothelium include potent platelet inhibitors. These inhibitors include PGI2, NO &CO which are labile molecules that are released by endothelial cells and act locally as autacoids and ADPase, an ectonucleotidase of endothelial membranes that break down platelet activating ADP.

ADHESION:

On vascular intimal injury, the antiplatelet properties of local endothelium diminished, while previously considered as cryptic, thrombogenic sub endothelial substance eg.collagen become exposed to flowing blood. Circulating platelet recognize vascular disruption site and undergo the adhesion process at the site of vascular injury.

Platelet adhesion is mediated by von willebrand factor which is present in the extracellular matrix of sub endothelial vessel wall. The receptor of von willebrand factor on the platelet surface is localized in membrane glycoprotein (Gp) Ib,part of the platelet membrane Gp Ib/ IX-V complex. Platelet adhesion is also facilitated by direct binding to sub endothelial collagen by means of specific platelet membrane collagen receptors.

ACTIVATION:

Adherent platelets then become activated. The platelet activation process results from integrative action of several agonists that bind to their respective membrane receptors on adherent platelets and transmit platelet activating intracellular signals. These platelet stimuli results from various mediators

1. Humeral mediators in plasma (epinephrine, thrombin)

2. Mediators from activated cell (ADP, serotonin)

3. Vessel wall extracellular matrix (collagen,von willebrand factor)

Activated platelet then undergo release reaction during which they secrete pre-packaged constituents of their cytoplasmic granules.

Dense granules release-ADP, ATP, serotonin

α -granule release

1. soluble adhesive protein-(fibrinogen, von willebrand factor, thrombospondin, fibronectin)

2. Growth factors-(PDGF, TGF α,TGF β)

3. procoagulants (platelet factor 4,Factor V)

Simultaneously, activated platelet synthesize denovo and release the potent platelet activator and vasoconstrictor thromboxane (TXA2)

AGGREGATION:

The products of the platelet release reaction, including secreted granule constituents and (TX2) mediate aggregation. During platelet aggregation, additional platelets are recruited from circulation to the site of vascular injury leading to the formation of occlusive platelet thrombus.

At the lower shear level (eg.in venous circulation), the molecular glue that mediates aggregation is fibrinogen, which can be derived either from plasma or from α – granule releasate of activated platelets. At higher level shear (eg.in arteries) von willebrand factor can substitute for fibrinogen as the ligand of aggregation.

Fibrinogen or von willebrand factor binds to the specific platelet membrane receptor that are located in the GP IIb/IIIa complexand mediates aggregation and finally platelet plug is formed. The platelet plug is anchored and stabilized by the fibrin mesh that develops simultaneously as the product of coagulation cascade.
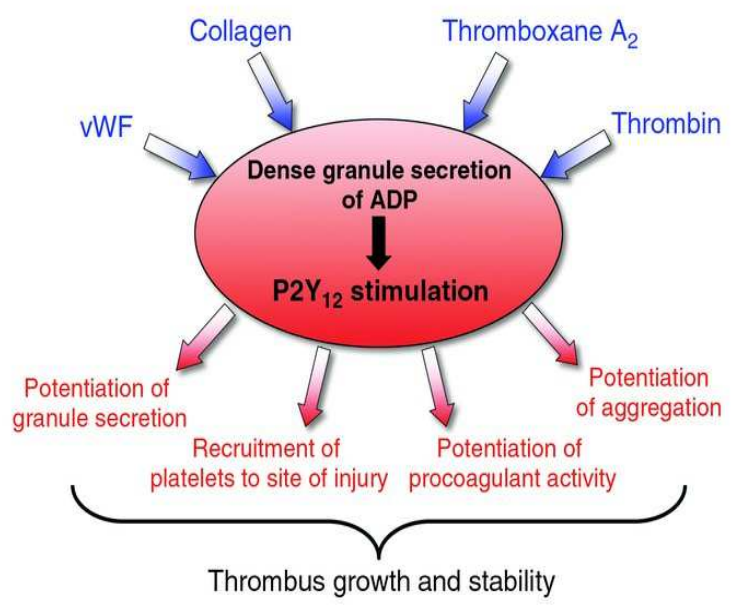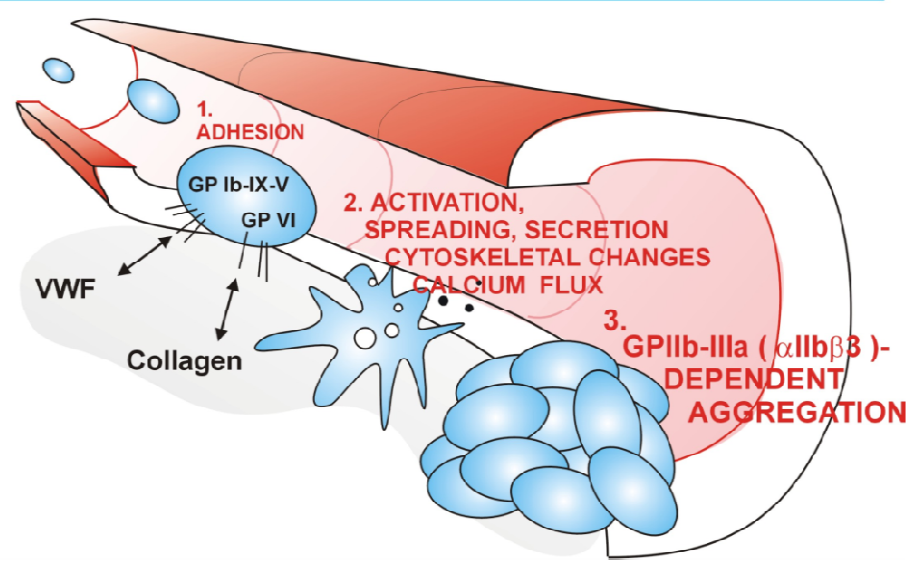
PATHOGENESIS OF ACUTE MYOCARDIAL INFARCTION

Almost all myocardial infarction result from coronary atherosclerosis, generally with superimposed coronary thrombosis. During the natural evolution of atherosclerotic plaques, especially those that are lipid laden, an abrupt and catastrophic transition may occur characterized by plaque rupture .After plaque rupture there is exposure of substance that promote platelet activation and aggregation, thrombin generation and ultimately thrombus formation. The resultant thrombus that is formed interrupts blood flow and leads to an imbalance between oxygen supply and demand and if this imbalance is severe and persistent, to myocardial necrosis.

PLATELET VOLUME AND CARDIOVASCULAR DISEASE

As the initial step in the pathogenesis of acute myocardial infarction is plaque erosion or rupture followed by platelet adhesion, activation & aggregation followed by thrombus formation, platelet with more activity will predispose to the occurrence of myocardial infarction. Mean platelet volume (MPV) correlates with platelet function and activation, whether measured as aggregation, thromboxane synthesis, β thromboglobulin release, procoagulant function or adhesion molecule expression. (Bath et al,1996).[16]

Increased platelet reactivity as well as shortened bleeding time are associated with increased platelet volume (Milner & Martin, 1985; Trowbridge & Martin, 1987)[17]. large platelets are metabolically and enzymatically more active than small platelet as assessed by invitro aggregometry(Corash et al 1977)[18] and they have higher thrombotic potential (karpatkin 1972)[19].they also express higher levels of procoagulatory surface proteins such as P-selectin (Mathur et al , 2001)[20] and glycoprotein III a(pathansali et al,2001)[21]

Large platelets are denser; they produce more thromboxane A2 per unit volume of platelet cytoplasm and decrease bleeding time more than control platelets. Larger platelets aggregate more rapidly upon collagen challenge, release more serotonin & other granule contents and express more receptors per unit area (pizzuli et al, 1998)[22].

Platelet morphology and physiology are determined during or even before fragmentation of their precursor cell,the megakaryocyte (Rabellino et at 1981)[23].Although the mechanism is still unclear, megakaryocyte plody seems to correlate closely with platelet volume(Hoffman and Long 1995)[24].Although ploidy and platelet volume are independent variables, alteration in both parameters usually occur in tandem(Trowbridge and Martin ,1987). Certain cytokines such as Interleukin -3,thrombopoetin and in particular interleukin 6(IL-6) seem to have major influence on

megakaryocyte ploidy leading to the production of larger and more reactive platelets 9 (Debilli et al 1993;Brown et al 1997).Recently a frequent G/C polymorphism in the promoter region of IL-6 at nucleotide position (-174) has been shown to influence IL-6 serum levels (Fishman et al 1998). In individual carrying the common G allele, higher IL-6 levels have been found compared with the levels in carriers of the C allele (Fishman et al 1998).

Large platelets are not necessarily young platelets (Martin et al, 1983) and there is now no convincing evidence that platelets appreciably change volume or density as they circulate (Penington, 1976).

Various studies found an association between mean platelet volume and coronary artery disease or the occurrence of an acute myocardial infarction (Assiri et al, 2012)[25] while others observed no effect (Halbmayer et al 1995)

Platelet volume and prognosis following acute myocardial infarction:

Hendra et al (1988)[26] observed that all patients with severe cardiac failure had larger platelet volumes than patients with mild or no failure.Osuna et al (1998)[27] stated that increase in mean platelet volume on admission was an independent risk factor for cardiac failure.

Increased MPV was found to be an independent risk factor for recurrent myocardial infarction by Martin et al (1982)[28]

Yilmaz et al(2004)[29] observed that in patients with dilated cardiomyopathy and in sinus rhythm, an increased MPV was associated with an increased incidence of left ventricular thrombus.

D.de Gonzalo-Calvo et al (2013)[30] states that platelet distribution width could be a predictor of 1 –year mortality in elderly population used as valuable tool for recognizing high risk individual so we can prevent by applying early stage preventive strategies.

In contrast to the above observations, Cameron et al (1983)[31] noted that the increase in MPV did not appear to provide any prognostic information after myocardial infarction. The value in patients who died was no different from that of the survivors. The MPV did not correlate with the more established factors determining prognosis after myocardial infarction such as size of the infarct.

AGE, SEX AND MPV

Funiak et al (1994)[32] observed increased MPV in patients advanced age. In contrast Bancroft et al (2000)[33] observed decreased MPV with advanced age. He observed no difference between genders.

MPVANDSMOKING

Smokers were found to have an increased MPV (Tschope et al, 1989[34]; Kario et al ,1992)[35].

MPVANDOTHERDISEASES

An increased MPV was observed in diabetics compared with non diabetic by sharpe et al (1993)[36]

Although Osuna et al (1998) [37]observed a higher MPV in those with systemic hypertension,Bathet al (1996)[38] observed no such effect.

Ford et al (1998)[39] observed that patients with hyperthyroidism and increased MPV.

Bansal et al (2002)[40] stated that MPV was increased in patients with chronic obstructive pulmonary disease and this could possibly contribute to an increased incidence of pulmonary embolism in these patients.

In chronic liver disease, MPV and platelet count are decreased (Jorgensen et al, 1984)[41].

MPV AND DRUGS

Aspirin has no effect on MPV (pizulli et al,1998).Recently ,invitro data on the therapeutic effects on platelet volume of losartan,an angiotensin II receptor antagonist or Doxasosin ,an α 1adrenoceptor antagonist, have been reported (Jagroop and mikhailidis 2001)[42].These observation have not been confirmed in vivo (jagroop and mikhailidis 2000).

MATERIALS AND METHODS

Setting   **:**   Dept of Medicine and Intensive coronary care unit, Rajiv Gandhi Govt, General Hospital Madras medical college, Chennai.

Collaborating

Department   :   Dept of cardiology, Govt.Rajiv Gandhi Hospital and Madras Medical college, Chennai.

Design of study   :   Observational prospective analytical study

Period of study   :   6 months

Sample size   :   65 patients and 35 controls

Ethical committee

approval   :   The present project was approved by the Ethical committee.

INCLUSION CRITERIA:

1. Age ranged from 18 to 80years.

2. Patient diagnosed as unstable angina,

STEMI-ST segment elevation myocardial infarction

NON –STEMI-non-ST segment elevation myocardial infarction

EXCLUSION CRITERIA

1. Patients with active hemorrhage

2. Patients with hematological disorders like leukemia, lymphoma and bone marrow disorders

3. Patients who have been transfused blood or platelet prior to admission – 8 days back

4. Patients who received chemotherapy / radiotherapy

5. Previous stroke

6. Blood pressure >180/110mmhg

7. Patient less than 18 years of age

8. Patient with non cardiac chest pain

CONTROLS:

Age and sex matched subjects who did not have angina pectoris, ECG evidence of coronary artery disease, history of previous coronary artery disease and who met the above exclusion criteria were kept as controls.

CONSENT:

Informed consent was obtained from all those who participated in the study or their relatives.

MATERIALS:

Thus a total of 65 cases who satisfied the inclusion and exclusion criteria stated above were taken up for subsequent study.35 age and sex matched subjects were kept as control

Definitions used for the study:

1. Acute myocardial infarction:

A patient was considered to have acute myocardial infarction if he/she gave a definite clinical history suggestive of acute myocardial infarction and ECG shows ST segment elevation.

> STEMI-ST segment elevation myocardial infarction >1mmin any two or more adjacent precordial or limb leads with elevated cardiac biomarkers

> NSTEMI-Non ST Elevation myocardial infarction-ST segment depression or T wave inversion with elevated cardiac biomarkers.

> Unstable angina-ST depression or T wave inversion without any elevation of cardiac biomarkers.

2. Smoking:

A subject was considered to be a smoker if he /she gave a history of tobacco smoking within the past 20 years. Person who had quit smoking completely before 20 years were not considered as smokers.

3. systemic hypertension;

A subject was considered to have systemic hypertension if he was already diagnosed to have systemic hypertension and was on anti hypertensive medication or if the systolic blood pressure during hospital stay was found to be more than or equal to 140mmHg and /or the diastolic blood pressure was more than or equal to 90mmHg according to JNC VIII report.

4. Diabetes mellitus:

A subject was considered to have diabetes mellitus if he/she was already diagnosed to have diabetes mellitus or during hospital Stay was found to have a Fasting plasma glucose of ≥ 126 mg/dl

Or

➢ 2 hour postprandial plasma glucose ≥ 200 mg/dl

Or

➢ Symptoms of diabetes mellitus plus random blood sugar ≥ 200 mg/dl.

5. Left ventricular dysfunction

Left ventricular dysfunction was divided to mild, moderate, severe

According to ejection fraction in ECHO

$\leq 29\%$ severe

30-39   moderate

40-49   mild

6. Short term outcome-outcome at the end of 7 days after acute

myocardial infarction was considered with respect to left ventricular dysfunction, left ventricular clot in ECHO, recurrence of angina, occurrence of arrhythmia, occurrence of death.

Methods:

Selected socio-demographic, clinical and laboratory data were collected from the patients and controls and recorded in a proforma (enclosed in Appendix-Appendix I)

I.socio –demographic data comprised of

- Age
- Sex
- History of smoking
- Family history of coronary artery disease

II.Clinical Data

- History
- Clinical examination

III.Laboratory Data

- Blood sugar

- ECG

- Echocardiogram

- Platelet count

- Mean platelet volume

- Platelet Distribution Width

ECG- 12 lead multi channel ECG was taken in all the patients. ECHO-Tran thoracic ECHO was done using ALOKO PRO S   100 in all the cases.2 D ECHO, M-MODE ECHO was done to analyse the regional wall motion abnormality, presence of clot and also to assess the left ventricular function. Colour Doppler evaluation was done to evaluate the presence of valvular regurgitation and also to assess the diastolic function.

For the above mentioned haematological parameters, 2ml of blood was withdrawn by venepuncture from the patients within 24 hours of admission to the hospital. The venepuncture site was properly cleaned and blood withdrawn and collected in EDTA containing disposable tubes available in the market. The sample was transported immediately to a quality controlled centre where the sample was analysed for platelet

volume. The instrument was started up. After start up, a pipette appeared from the instrument. Blood sample was fed to the instrument by the principal worker. The pipette drew the necessary amount of blood. From that moment a waiting time of 180 seconds appeared on the instrument. At the end of 180 seconds, a print out with the platelet count and mean platelet volume and platelet distribution width was ejected from the printer connected to the instrument. The instrument was repeatedly standardized for quality control.

Conflict of interest:

There was no conflict of interest.

Financial support:

Nil

Limitations:

1. Technical constraints and the cost factor of the investigation have led to limited number of cases.

2. Since the lipid levels were found to be an independent of mean platelet volume in earlier studies, it was neither considered for the present study nor taken for analytical purposes.

STATISTICAL ANALYSIS:

Data were entered in Microsoft excel spread sheet and analysed utilizing the software-Epidemiological information Package 2002(Epi Info 2002)- developed by the centre for disease control and prevention, Atlanta for World Health Organisation. Range, Median, Mean and Standard deviation and 'p' values were calculated using this package.

Chi –square test was done to find out the significance of relationship between the groups. Significance was considered if the 'p' value was below 0.05.

OBSERVATIONS AND RESULTS

The total number of subjects included in the study was 100. Among the 100 subjects, 65 cases and 35 controls and their profile is furnished below

AGE DISTRIBUTION :

The age of the cases ranged from 20-80 years and that of controls ranges from 28-75 years. The mean and standard deviation for the cases were 52.98±15.00 years and those for the controls were 49.03±10. 36 years. There was no significant difference with respect to age among them. The distribution of cases and controls with respect to age is given below in the table -1 given below.

TABLE -1

1. Distribution of case and control with respect to age

			AGE_GROUP				Total
			20-40	41-60	61-80	ABOVE 80	
GROUP	CASE MI	Count	16	25	22	2	65
		% within GROUP	24.6%	38.5%	33.8%	3.1%	100.0%
	CONTROL	Count	9	22	4	0	35
		% within GROUP	25.7%	62.9%	11.4%	0.0%	100.0%
Total		Count	25	47	26	2	100
		% within GROUP	25.0%	47.0%	26.0%	2.0%	100.0%
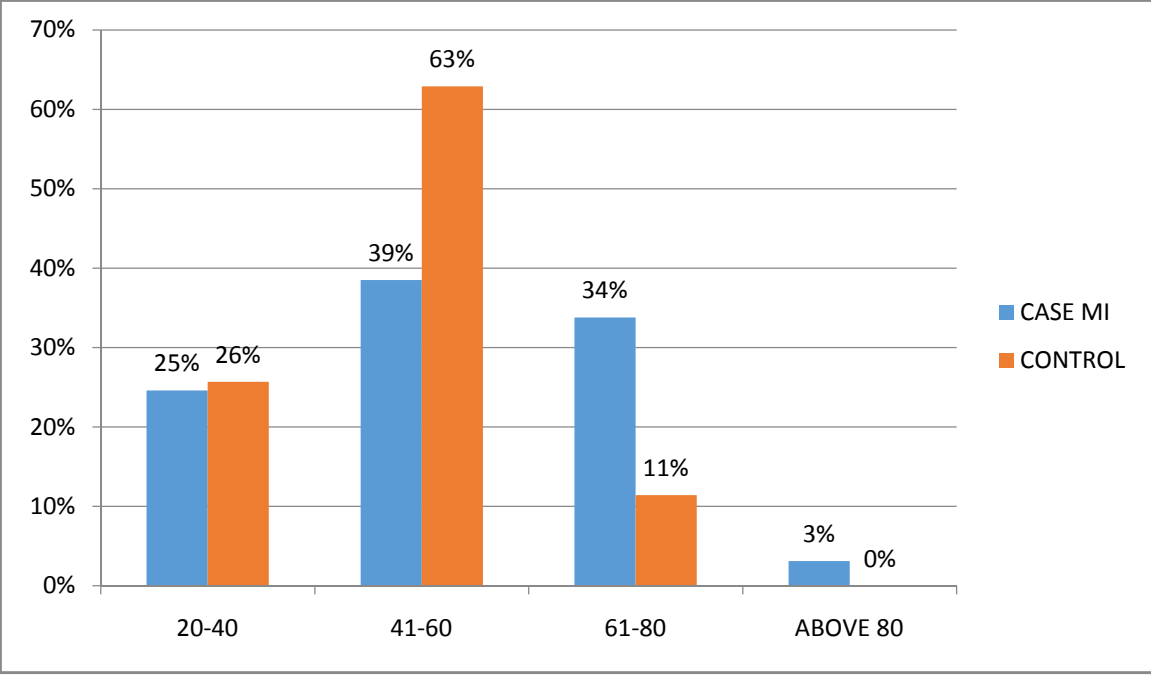
2. SEX DISTRIBUTION:

Among the 65 cases studied, there were 45 males and 20 females. Among the controls, there were 25 males and 10 females. The difference in the sex composition of the case and control was not statistically significant. The details are furnished in Table -2 depicted below.

TABLE 2

Distribution of case and control in relation to gender

			SEX		Total
			Male	Female	
GROUP	CASE MI	Count	45	20	65
		% within GROUP	69.2%	30.8%	100.0%
	CONTROL	Count	25	10	35
		% within GROUP	71.4%	28.6%	100.0%
	Total	Count	70	30	100
		% within GROUP	70.0%	30.0%	100.0%
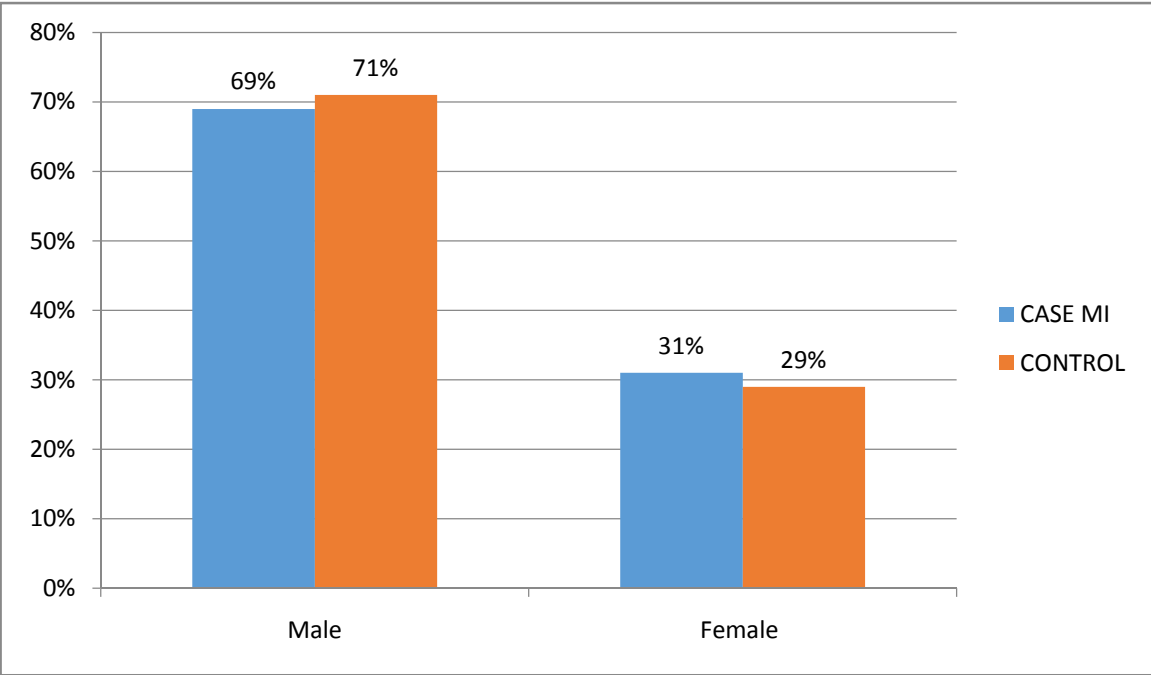
3.SMOKING:

Among the 65 cases 40 had the habit of smoking. Among the 35 controls, 13 had the habit of smoking. There were no female smokers in either group. There was no statistical difference in the habit of smoking between cases and controls. The details are depicted in the table-3 given below.

TABLE 3

Distribution of case and control with respect to habit of smoking

			SMOKING		Total
			Yes	No	
GROUP	CASE MI	Count	40	25	65
		% within GROUP	75.5%	53.2%	100.0%
	CONTROL	Count	13	22	35
		% within GROUP	24.5%	46.8%	100.0%
Total		Count	53	47	100
		% within GROUP	54.0%	46.0%	100.0%

Pearson Chi-Square =5.435,P=0.020
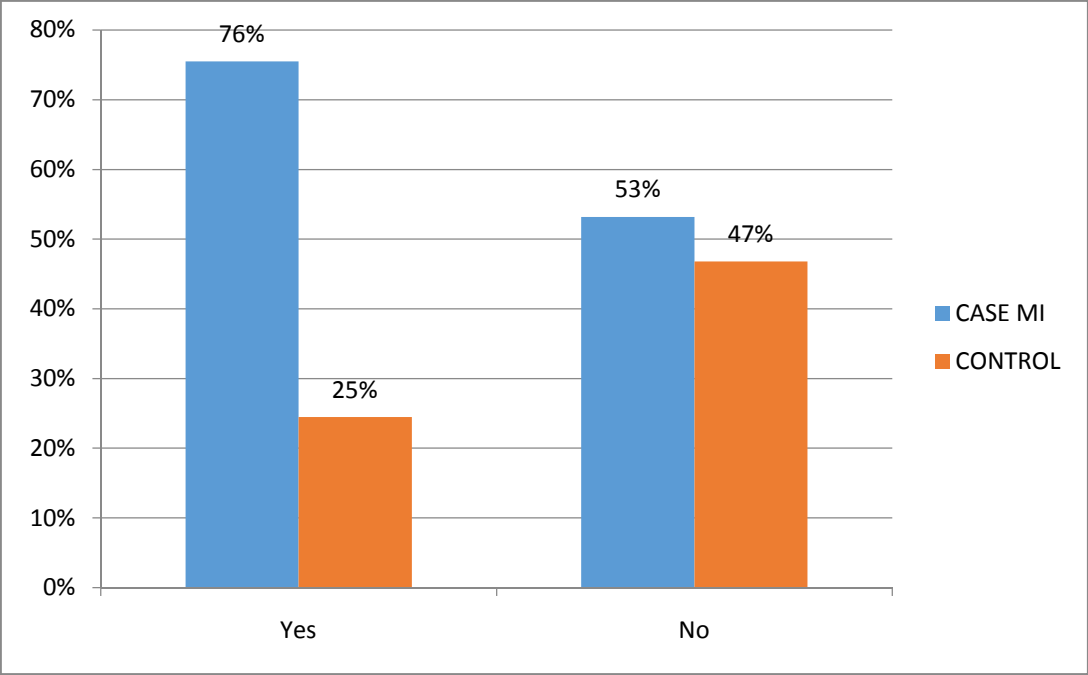
4.DIABETES MELLITUS:

Among the 65 cases, 29 had diabetes mellitus. Among the 35 controls, 8 had diabetes mellitus. There was statistically significant difference among cases and controls with respect to diabetes mellitus. The details are given in the table-4 shown below.

TABLE-4

Distribution of case and control with respect to diabetes

			DIABETES		Total
			Yes	No	
GROUP	CASE MI	Count	29	36	65
		% within GROUP	78.4%	57.1%	100.0%
	CONTROL	Count	8	27	35
		% within GROUP	21.6%	42.9%	100.0%
Total		Count	37	63	100
		% within GROUP	100.0%	100.0%	100.0%

Pearson Chi-Square =4.620 ,P=0.032
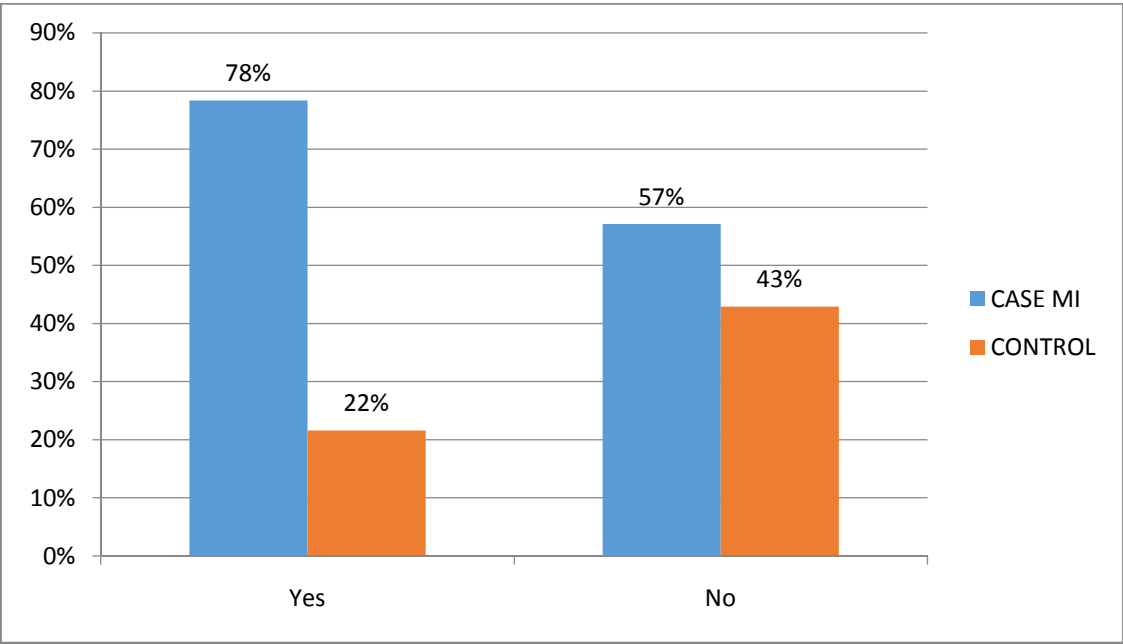
5.SYSTEMIC HYPERTENSION:

Among the 65 cases ,35 had systemic hypertension. Among the 35 controls, 19 had systemic hypertension. Systemic hypertension was present among cases and controls with statistically significant difference.

The details are provided in the table- 6 given below.

TABLE -5

Distribution of case and control with respect to hypertension

			HYPERTENSION		Total
			Yes	No	
GROUP	CASE MI	Count	35	30	65
		% within GROUP	76.1%	55.6%	100.0%
	CON TROL	Count	19	15	34
		% within GROUP	23.9%	44.4%	100.0%
Total		Count	47	52	99
		% within GROUP	100.0%	100.0%	100.0%

Pearson Chi-Square=4.603,P=0.032
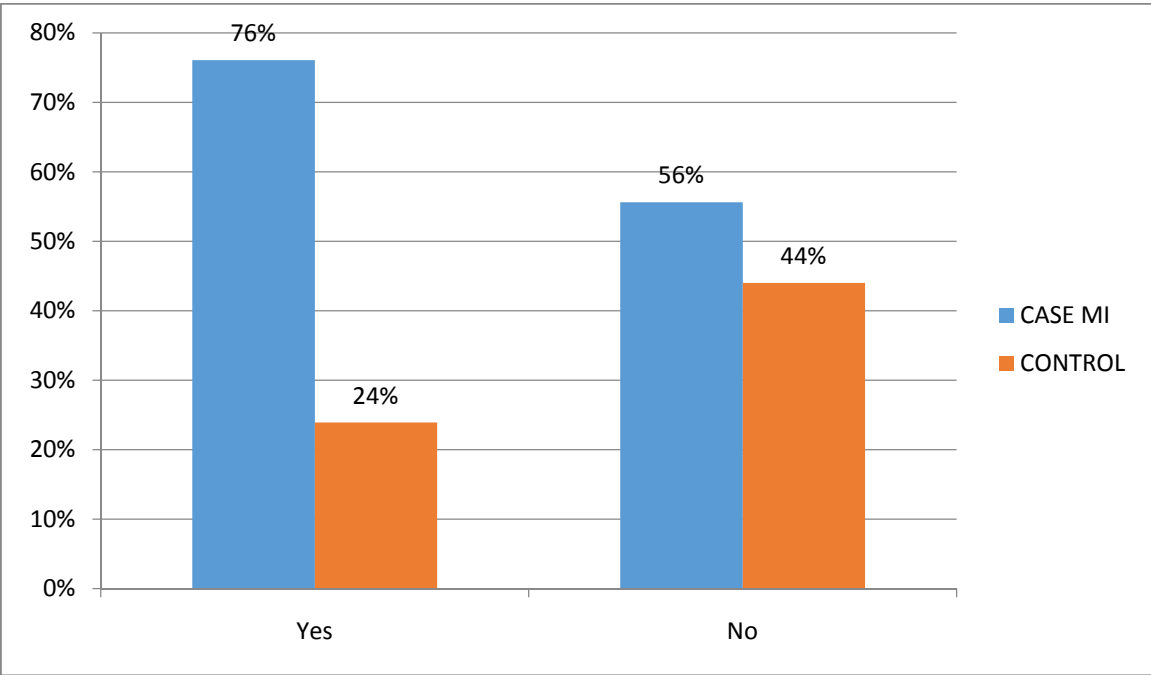
6.ASPIRIN INTAKE.

TABLE 6

Distribution of case with respect to aspirin intake

ON_ASPIRIN

		Frequency	Percent	Valid Percent	Cumulative Percent
	Yes	31	47.7	47.7	47.7
Valid	No	34	52.3	52.3	100.0
	Total	65	100.0	100.0	
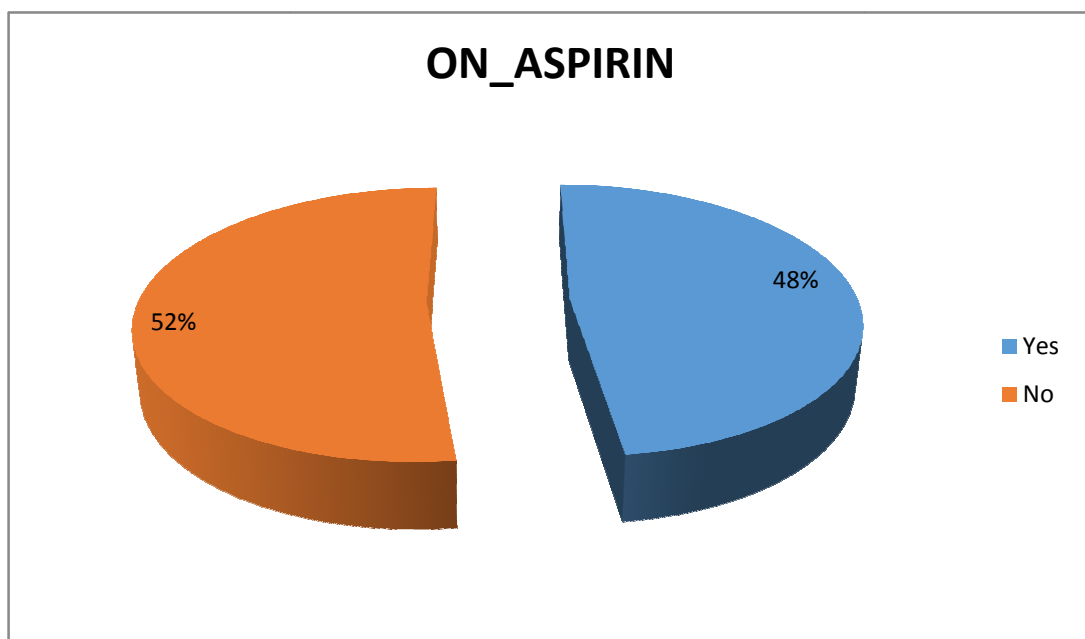
STEMI

		Frequency	Percent	Valid Percent	Cumulative Percent
	1.00	26	40.0	40.0	40.0
Valid	2.00	39	60.0	60.0	100.0
	Total	65	100.0	100.0	
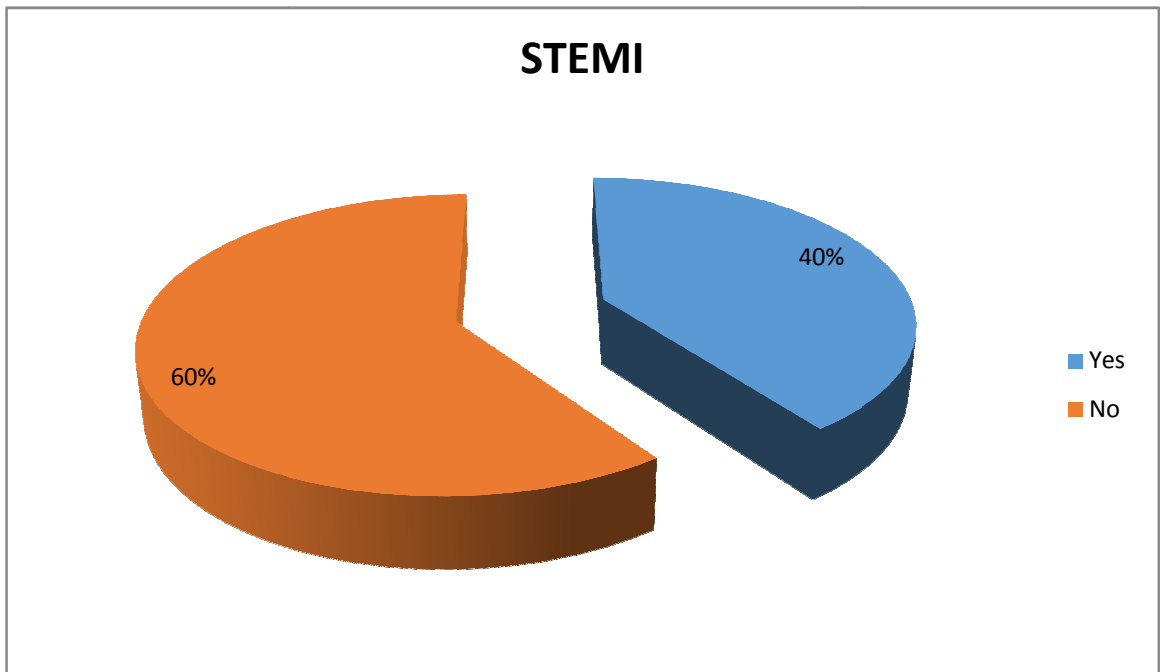
NSTEMI

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1.00	18	27.7	27.7	27.7
	2.00	47	72.3	72.3	100.0
	Total	65	100.0	100.0	
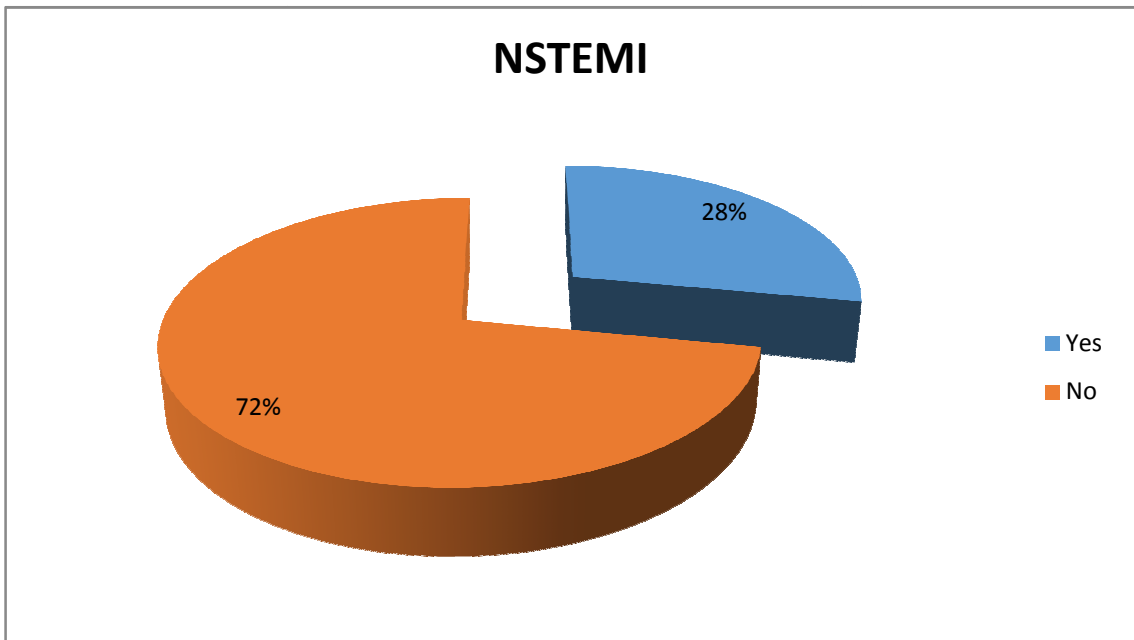
UNSTABLE_ANGINA

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1.00	20	30.8	30.8	30.8
	2.00	45	69.2	69.2	100.0
	Total	65	100.0	100.0	
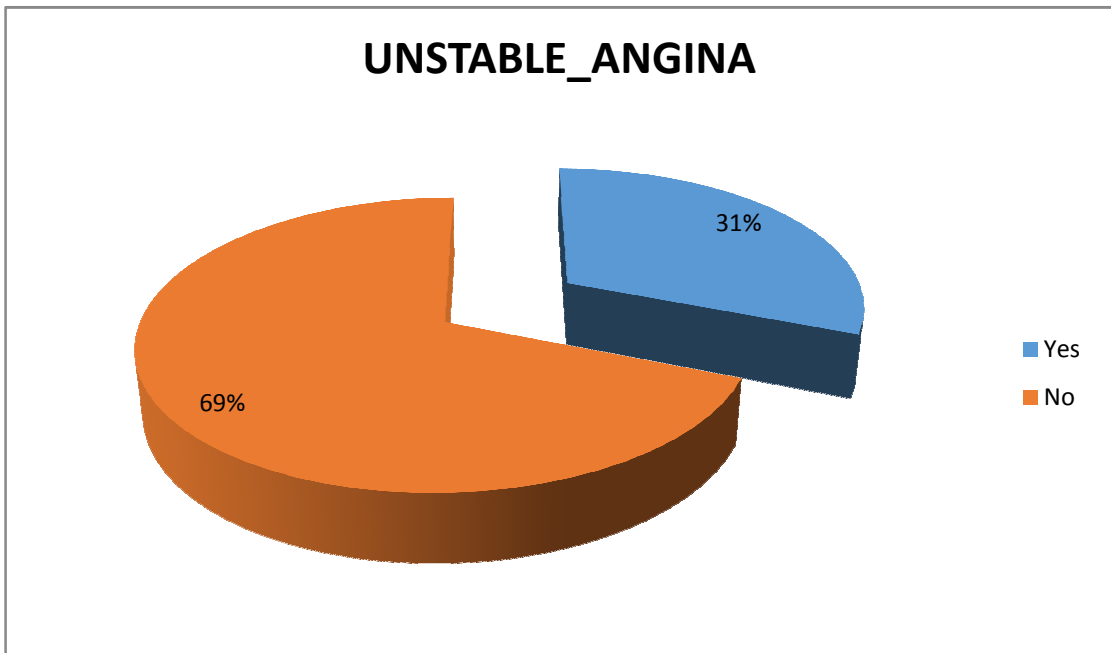
7.PLATELET COUNT:

The mean platelet count of the cases was4.98±0.23lakhs/mm³ and that of the controls was 1.29±0.42 Lakhs/mm³ (p value 0.050 statistically significant).

TABLE 7

platelet count in case and control

Group Statistics

	GROUP	N	Mean	Std. Deviation	Std. Error Mean
PLATELET_COUNT	CASE MI	65	495606.1538	1493656.28408	185265.26076
	CONTROL	35	132028.5714	29341.28362	4959.58214

t=1.962 p=0.05 significant

MEAN PLATELET_COUNT IN LAKHS

59
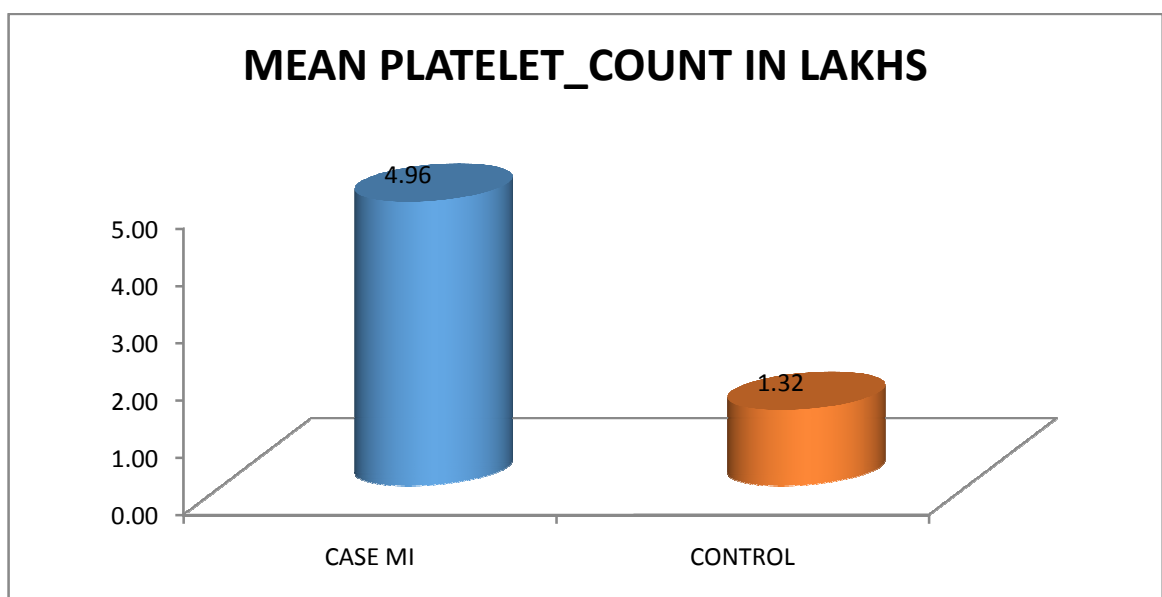
8. MEAN PLATELET VOLUME:

The MPV of the cases ranged from 8.4 to 12 femtolitres with a mean of 10.2 ± 1.1 femtolitres. The MPV of the controls ranged from 7 to 10.60 femtolitres with mean of 8.29 ± 0.9 femtolitres. The difference between the two was statistically significant.(p value 0.001 significant).

TABLE -8

MPV in case and control

	GROUP	N	Mean	Std. Deviation	Std. Error Mean
MPV	CASE MI	65	10.2662	1.13072	.14025
	CONTROL	35	8.2914	.92906	.15704

t=8.843** p<0.001 significant

MEAN MPV
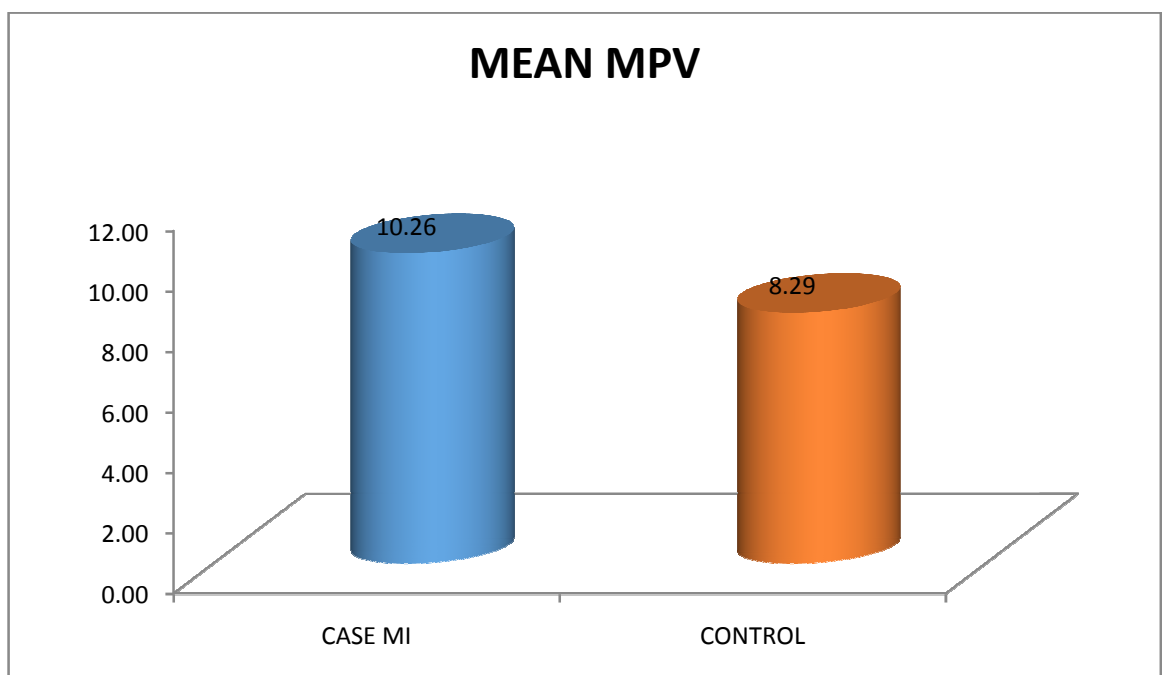
9.PLATELET DISTRIBUTION WIDTH

TABLE-9

PDW in case and control

	GROUP	N	Mean	Std. Deviation	Std. Error Mean
PDW	CASE MI	65	13.0646	2.23092	.27671
	CONTROL	35	10.7629	1.38438	.23400

t=5.548** p<0.001 significant

MEAN PDW

CASE MI: 13.07
CONTROL: 10.76
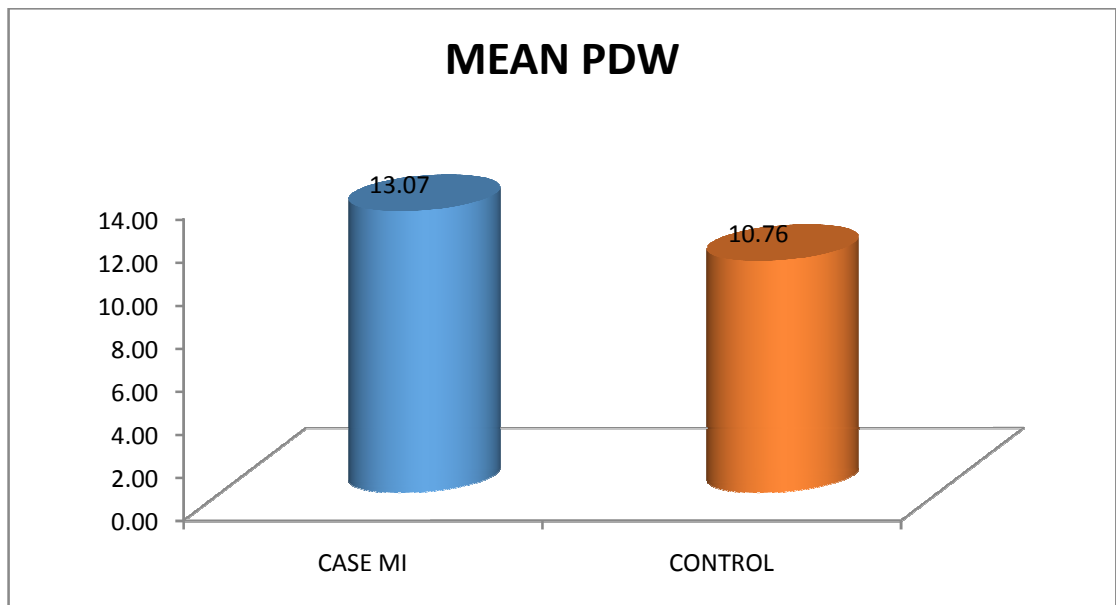
10.RELATIONSHIP BETWEEN PLATELET COUNT AND ACUTE CORONARYSYNDROME

TABLE -10

Platelet count in STEMI, NSTEMI, Unstable angina

		PLATELET_COUNT				
		Mean	Maximum	Minimum	Median	Standard Deviation
STEMI	PRESENT	887000	8800000	66000	250000	2331358
	ABSENT	234676.9	397000	80000	235000	83608.72
NSTEMI	PRESENT	206222.2	343000	98000	201000	66571.11
	ABSENT	606434	8800000	66000	255000	1748443.9
UNSTABLE _ANGINA	PRESENT	264520	397000	80000	282000	89071.34
	ABSENT	598311.1	8800000	66000	222000	1790703

MEAN PLATELET COUNT IN LAKHS FOR STEMI

8.87

2.35

PRESENT   ABSENT
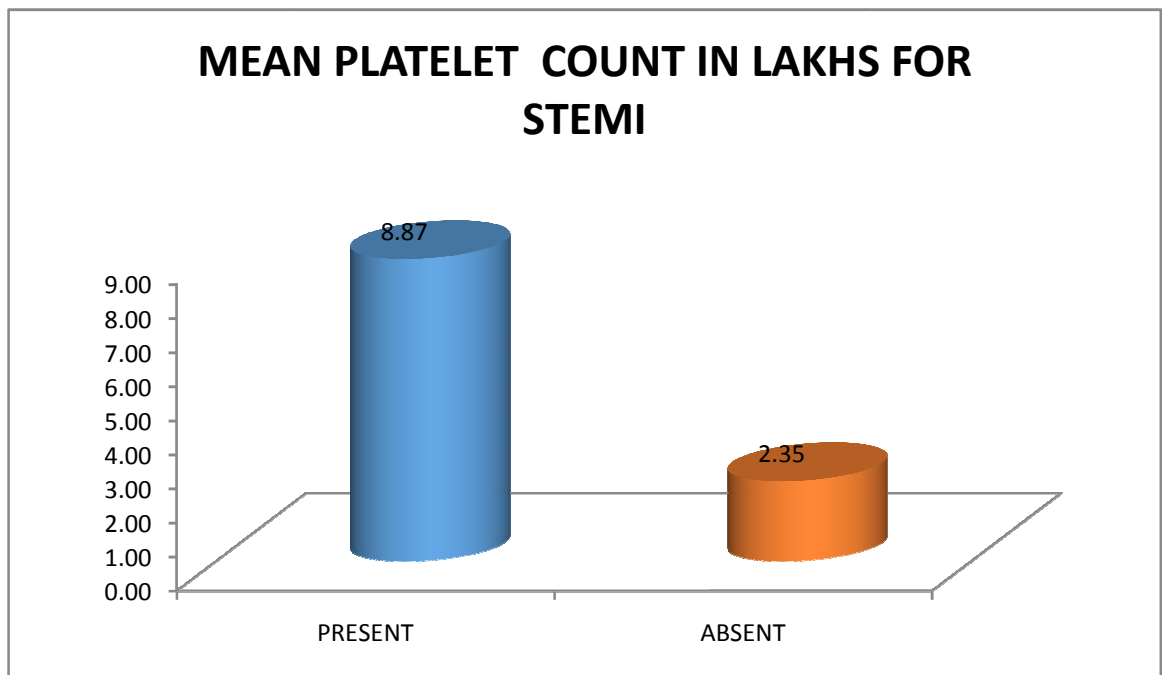
MEAN PLATELET COUNT IN LAKHS FOR NSTEMI
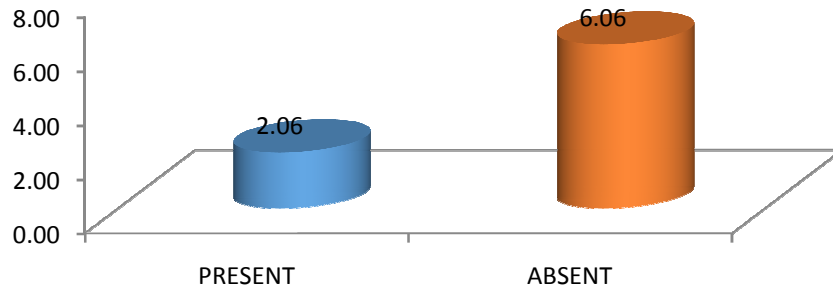
MEAN PLATELET COUNT IN LAKHS FOR UNSTABLE ANGINA
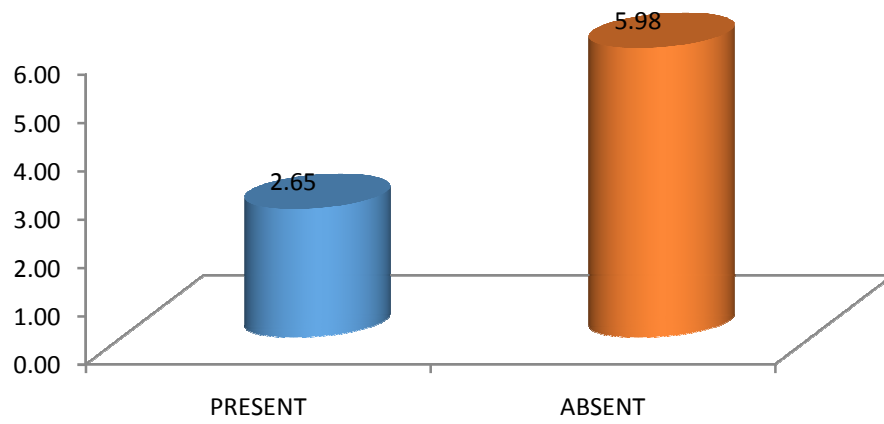
11.RELATIONSHIP BETWEEN MEAN PLATELET VOLUME AND ACUTE CORONARY SYNDROME

TABLE -11

MPV in STEMI, NSTEMI, unstable angina

		MPV				
		Mean	Maximum	Minimum	Median	Standard Deviation
STEMI	PRESENT	10.79	13.8	8.2	10.9	1.26
	ABSENT	9.92	11.7	8	9.9	0.89
NSTEMI	PRESENT	10.02	11.7	8.4	9.95	0.91
	ABSENT	10.36	13.8	8	10.3	1.2
UNSTABLE _ANGINA	PRESENT	9.75	11	8	9.8	0.82
	ABSENT	10.5	13.8	8.2	10.4	1.18

MEAN MPV

MEAN MPV IN NSTEMI

MEAN MPV IN UNSTABLE ANGINA
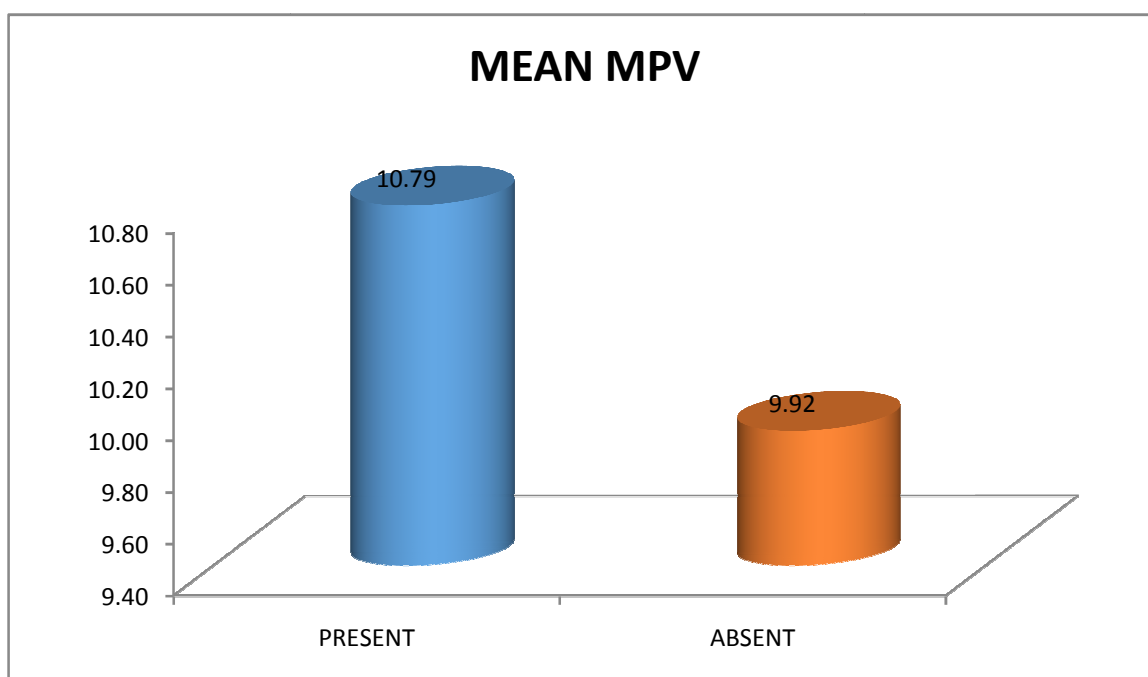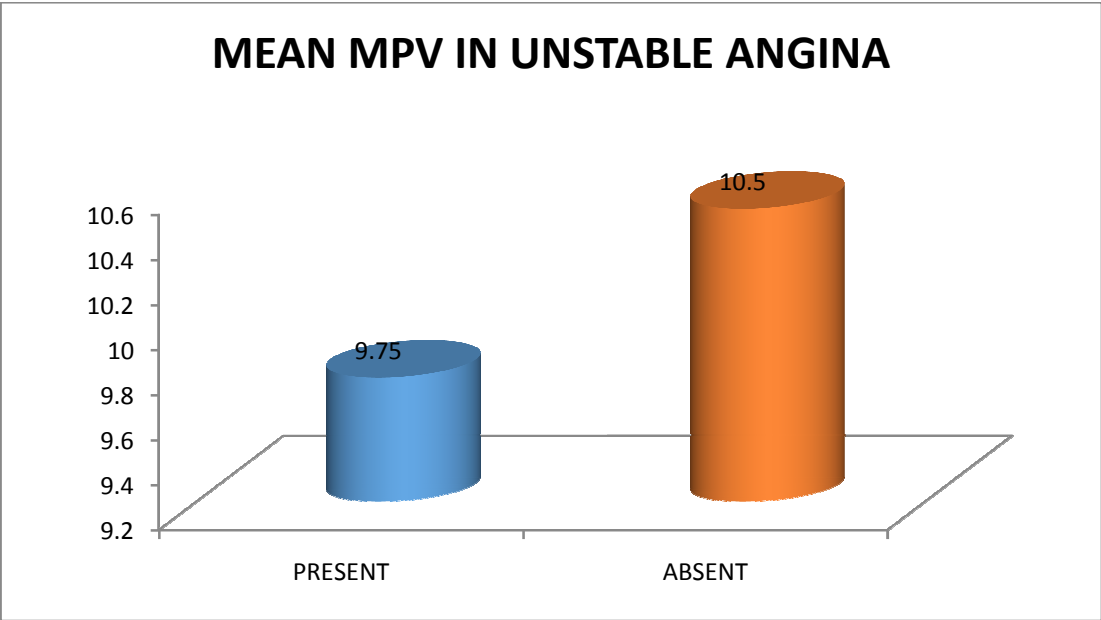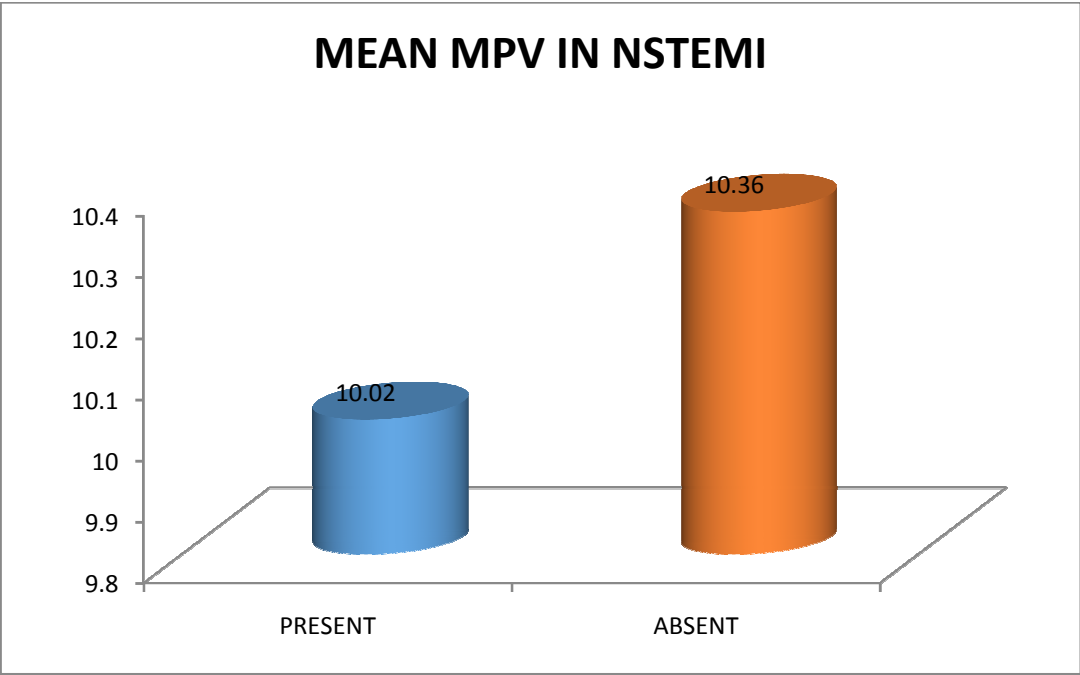
12. PLATELET DISTRIBUTION WIDTH IN ACUTE CORONARY SYNDROME

TABLE-12

PDW in STEMI, NSTEMI, unstable angina

		PDW				
		Mean	Maximum	Minimum	Median	Standard Deviation
STEMI	PRESENT	14.32	21.4	10.5	14.15	2.51
	ABSENT	12.23	15.6	8.8	12.3	1.56
NSTEMI	PRESENT	12.39	15.6	9.7	12.45	1.43
	ABSENT	13.32	21.4	8.8	13.4	2.43
UNSTABLE _ANGINA	PRESENT	11.99	15	8.8	11.95	1.66
	ABSENT	13.54	21.4	9.7	13.5	2.3

MEAN PDW IN STEMI

MEAN MPV IN NSTEMI

Bar chart showing mean MPV values. PRESENT: 10.02 (blue). ABSENT: 10.36 (orange). Y-axis ranges from 9.8 to 10.4.

MEAN MPV IN UNSTABLE ANGINA

Bar chart showing mean MPV values. PRESENT: 9.75 (blue). ABSENT: 10.5 (orange). Y-axis ranges from 9.2 to 10.6.
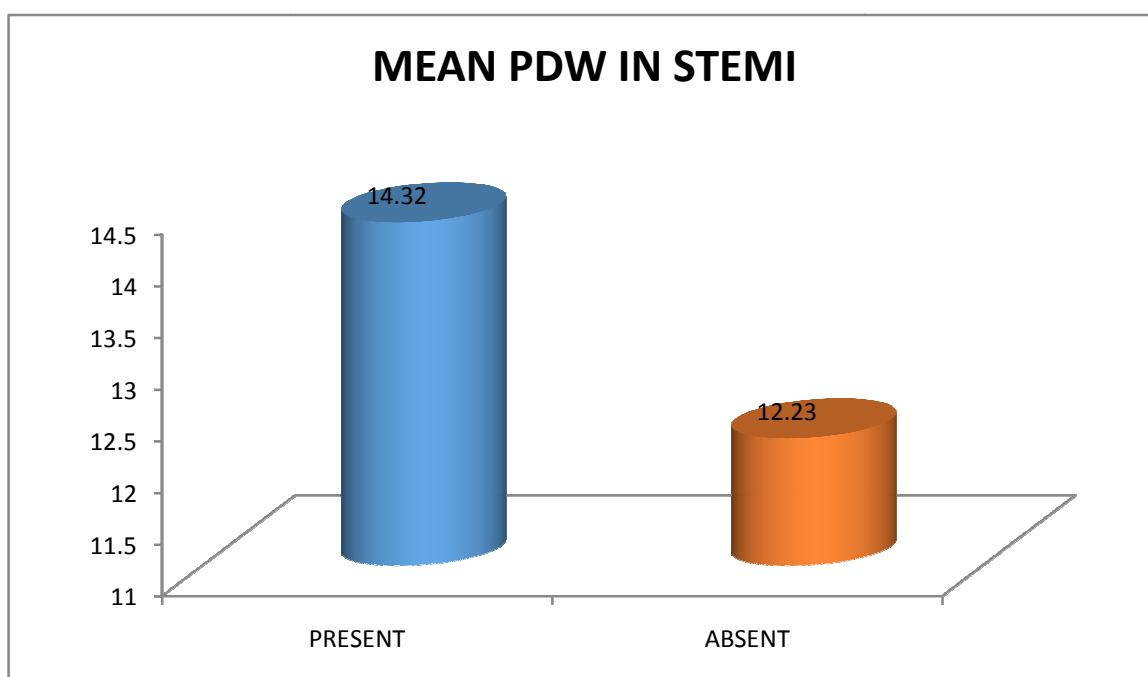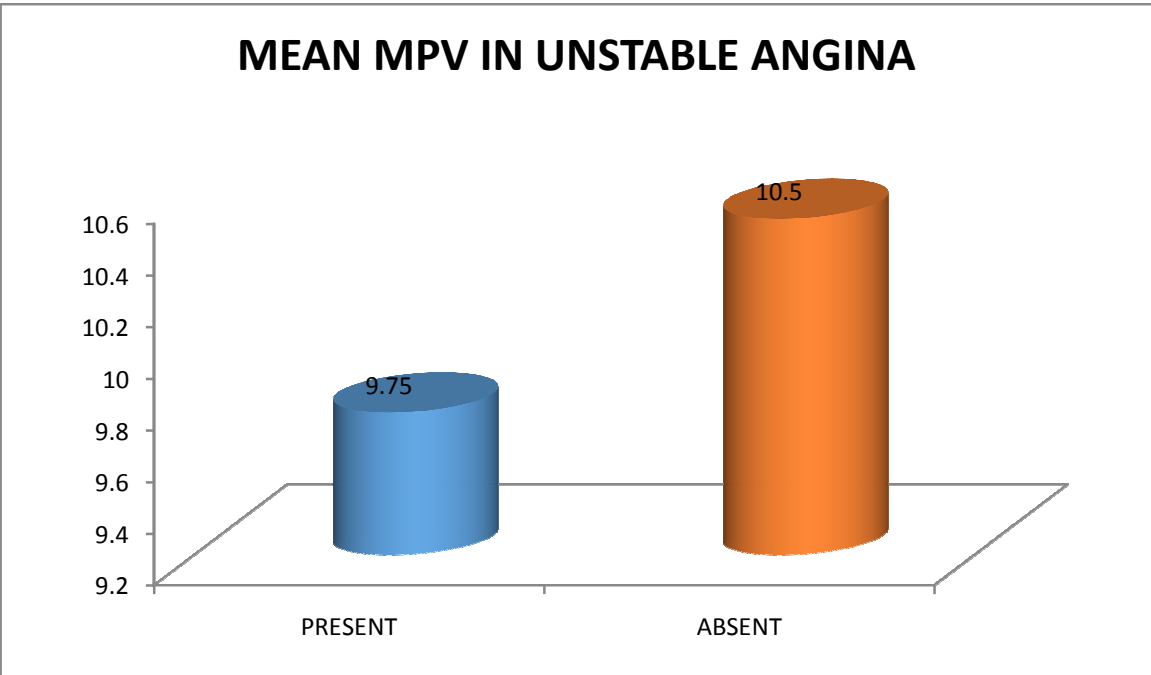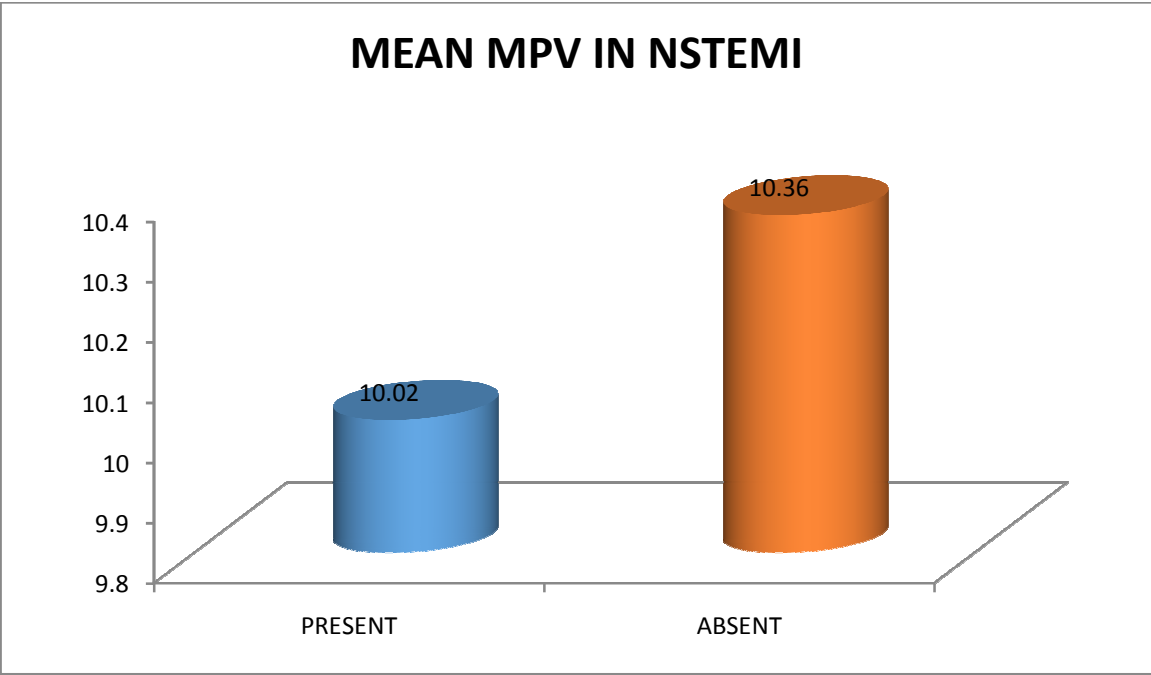
TABLE -13

Relation ship of platelet count, MPV, PDW with ejection fraction

Correlations

		PLATELET_COUNT	PDW	MPV
EJECTION_FRACTION	Pearson Correlation	-.033	-.377**	-.238
	Sig. (2-tailed)	.795	.002	.057
	N	65	65	65

**. Correlation is significant at the 0.01 level (2-tailed).

TABLE-14

Relationship of platelet count, MPV, PDW with complication

difference between two groups

	COMPLICATION	N	Mean	Std. Deviation	Std. Error Mean	t value	P value
PLATELET_COUNT	1.00	12	950500.0000	2472885.52468	713860.56168	1.172	0.246
	2.00	53	392611.32008	1180550.95965	162161.14559		
PDW	1.00	12	15.4333	2.94906	.85132	4.695 **	P<0.001
	2.00	53	12.5283	1.64251	.22562		
MPV	1.00	12	11.3083	1.29507	.37385	3.911 **	P<0.001
	2.00	53	10.0302	.95466	.13113		

			PLATELET _COUNT	PDW	MPV
Kendall's tau_b	COMPLICATI ON	Correlation Coefficient	-.069	-.368[**]	-.318[**]
		Sig. (2-tailed)	.504	.000	.002
		N	65	65	65
		Sig. (2-tailed)	.008	.000	.
		N	65	65	65
Spearman's rho	COMPLICATI ON	Correlation Coefficient	-.083	-.442[**]	-.381[**]
		Sig. (2-tailed)	.508	.000	.002
		N	65	65	65

Correlation is significant at the 0.05 level (2-tailed).

Correlation is significant at the 0.01 level (2-tailed).

TABLE -15

Relationship of platelet count, MPV, PDW with death difference between two groups

Group Statistics							
	DEATH	N	Mean	Std. Deviation	Std. Error Mean	t value	P value
PLATELET_COUNT	1.00	4	214000.0000	111513.82575	55756.91287	0.387	0.700
	2.00	61	514072.1311	1540612.79714	197255.25573		
PDW	1.00	4	16.3000	1.83848	.91924	3.203*	0.02
	2.00	61	12.8525	2.09671	.26846		
MPV	1.00	4	11.5750	.37749	.18875	2.484*	0.015
	2.00	61	10.1803	1.11128	.14228		

Correlations

			PLATELET_COUNT	PDW	MPV
	DEATH	Correlation Coefficient	.046	-.297[**]	-.269[**]
		Sig. (2-tailed)	.652	.004	.010
		N	65	65	65

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

TABLE -16

RELATIONSHIP OF MPV, PDW AND OTHER PARAMETERS

The relationship between MPV, PDW and other parameters are statistically significant.

		PDW	MPV
		Mean	Mean
AGE_GROUP	20-40	11.73	9.37
	41-60	12.20	9.52
	61-80	12.69	9.81
	ABOVE 80	14.70	10.35
SEX	Male	12.11	9.55
	Female	12.61	9.62
SMOKING	YES	12.54	9.79
	NO	11.95	9.33
DIABETES	YES	13.17	10.08
	NO	11.73	9.28
HYPERTENSION	YES	12.73	9.90
	NO	11.86	9.30
DEATH	YES	16.30	11.58
	NO	12.85	10.18

TABLE 17

RELATIONSHIP BETWEEN AGE WITH MPV, PDW

PDW chart showing values by age group: 20-40: 11.73, 41-60: 12.2, 61-80: 12.69, ABOVE 80: 14.7

MPV chart showing values by age group: 20-40: 9.37, 41-60: 9.52, 61-80: 9.81, ABOVE 80: 10.35

PDW

Male: 12.11
Female: 12.61

MPV

Male: 9.55
Female: 9.62

SMOKING IN PDW

SMOKING MPV

DM PDW

DM MPV

HTN PDW

12.73 — YES
11.86 — NO

SMOKING MPV

9.9 — YES
9.3 — NO

DEATH MPV

DEATH PDW
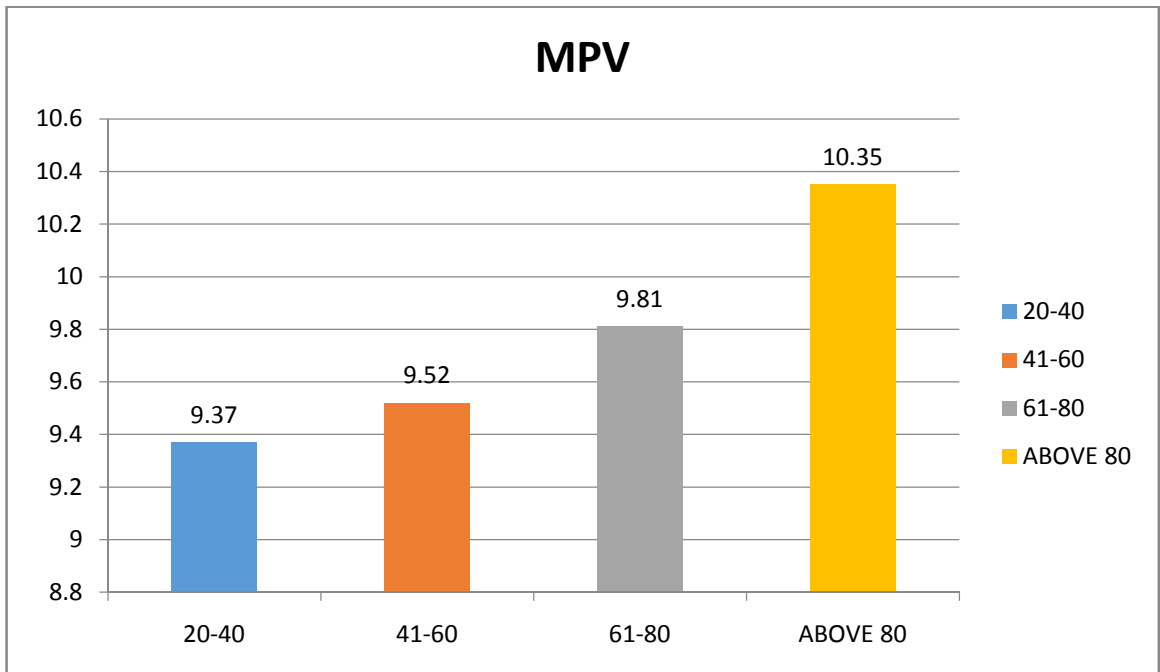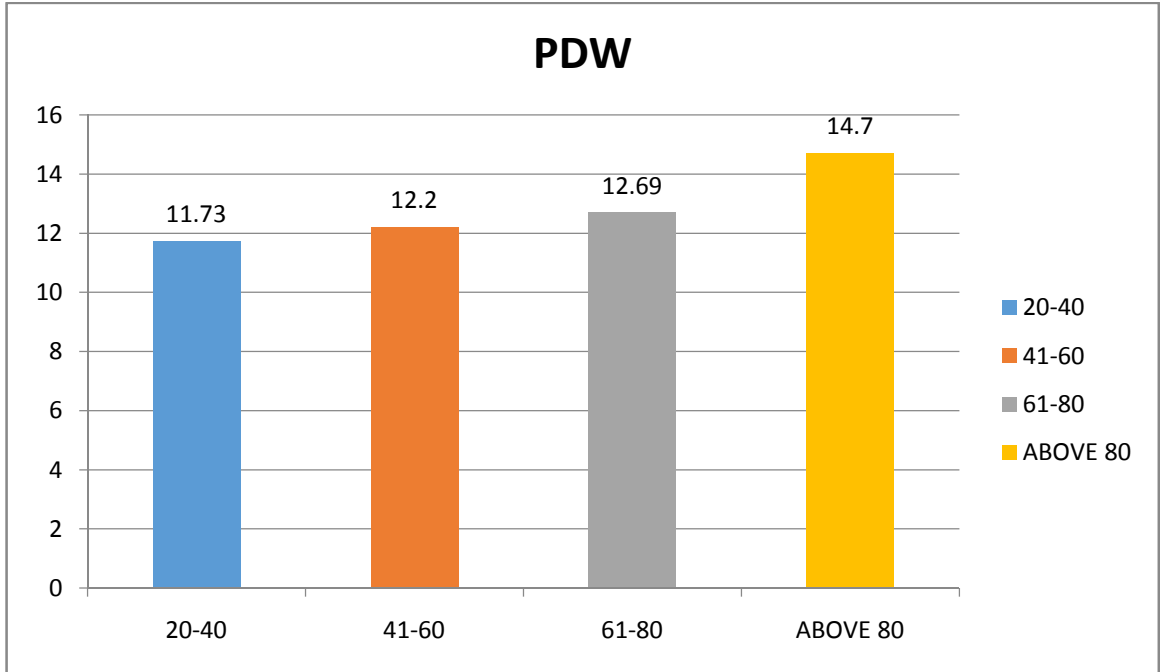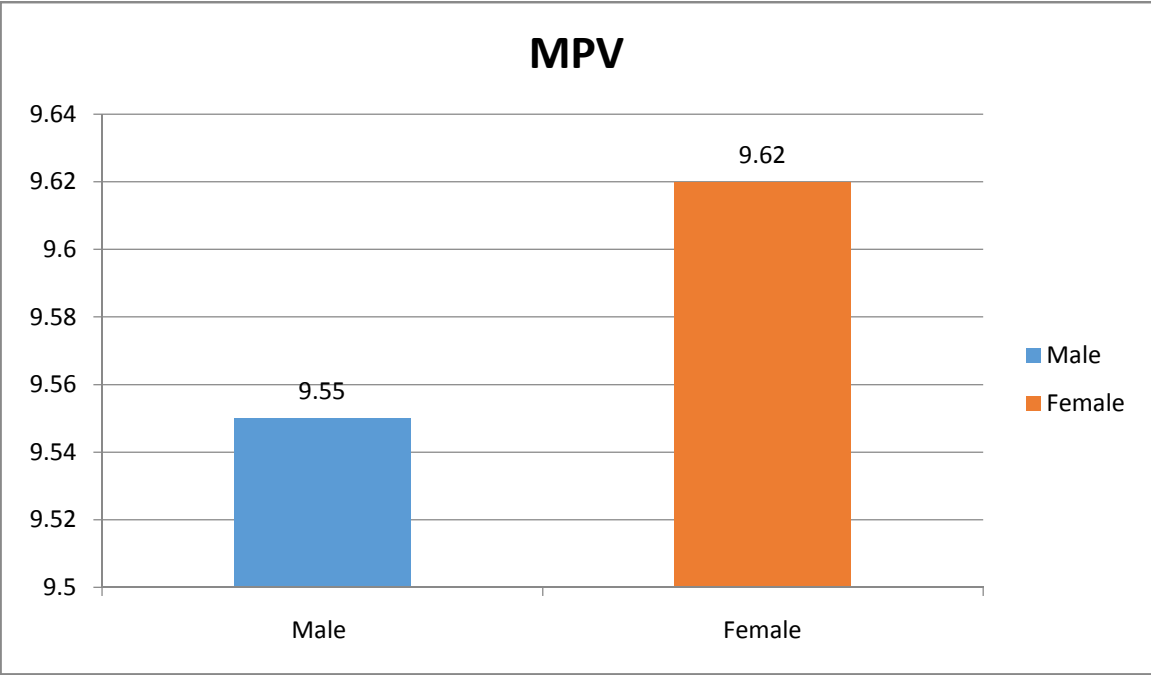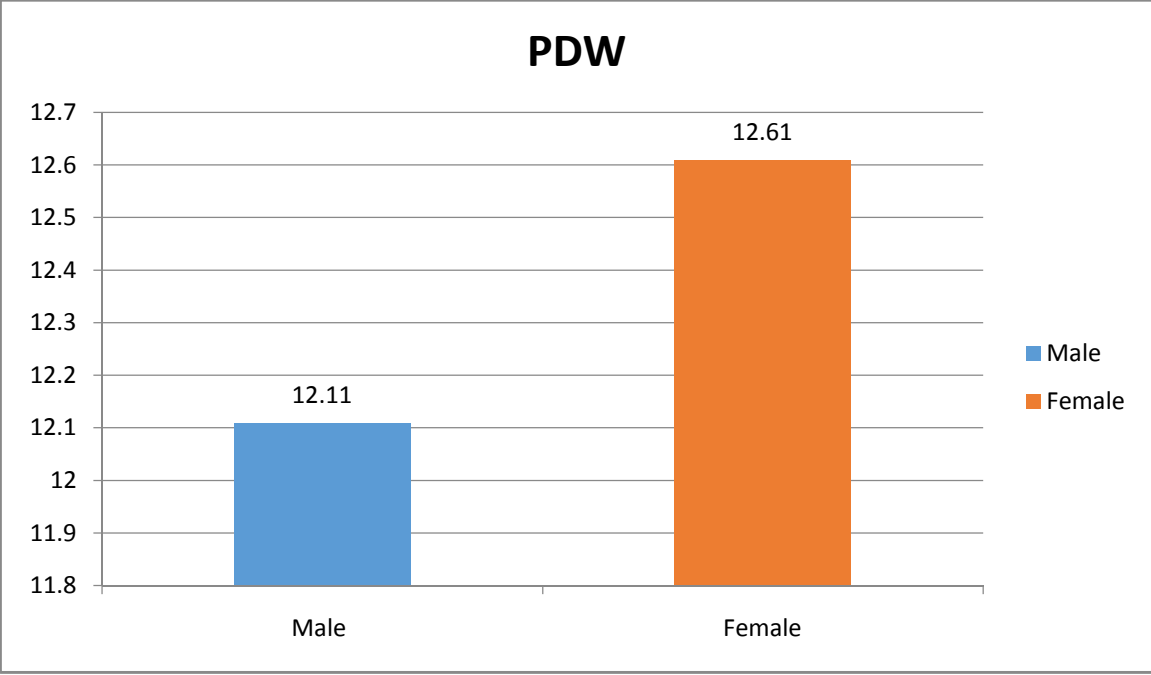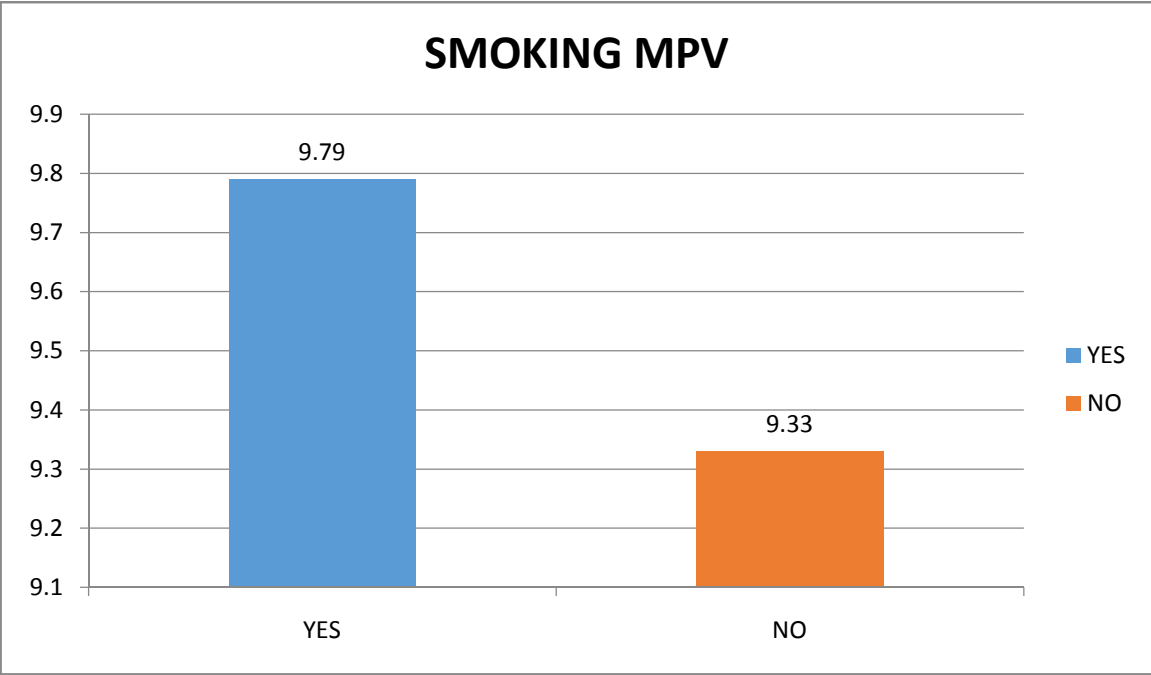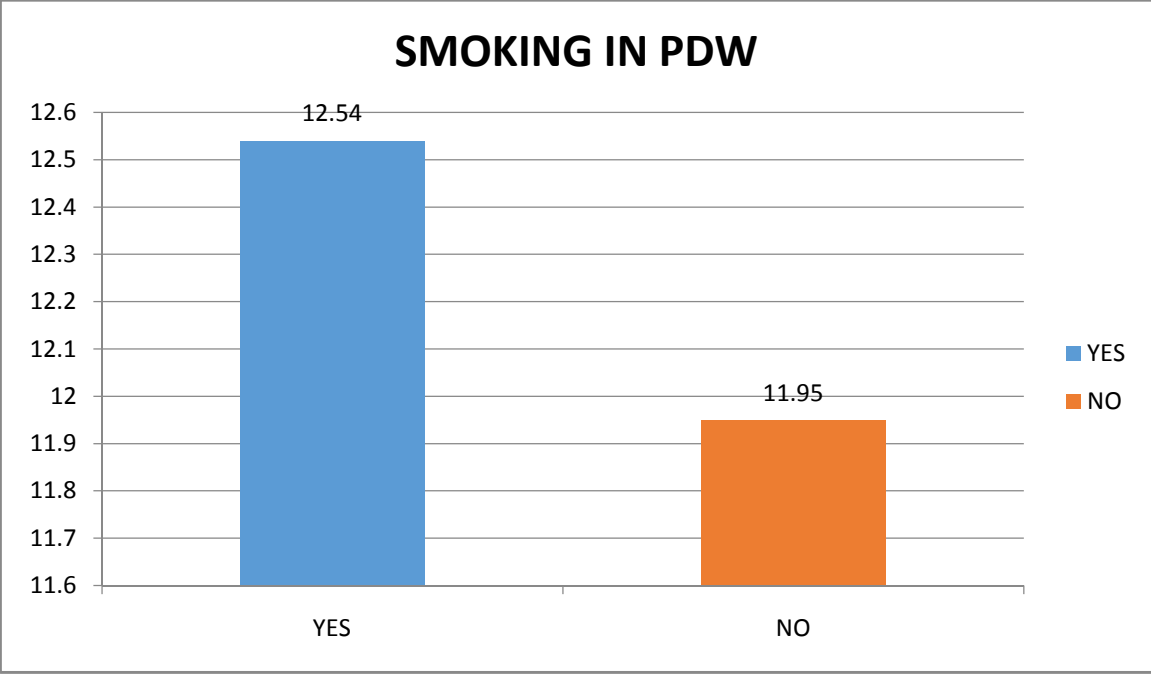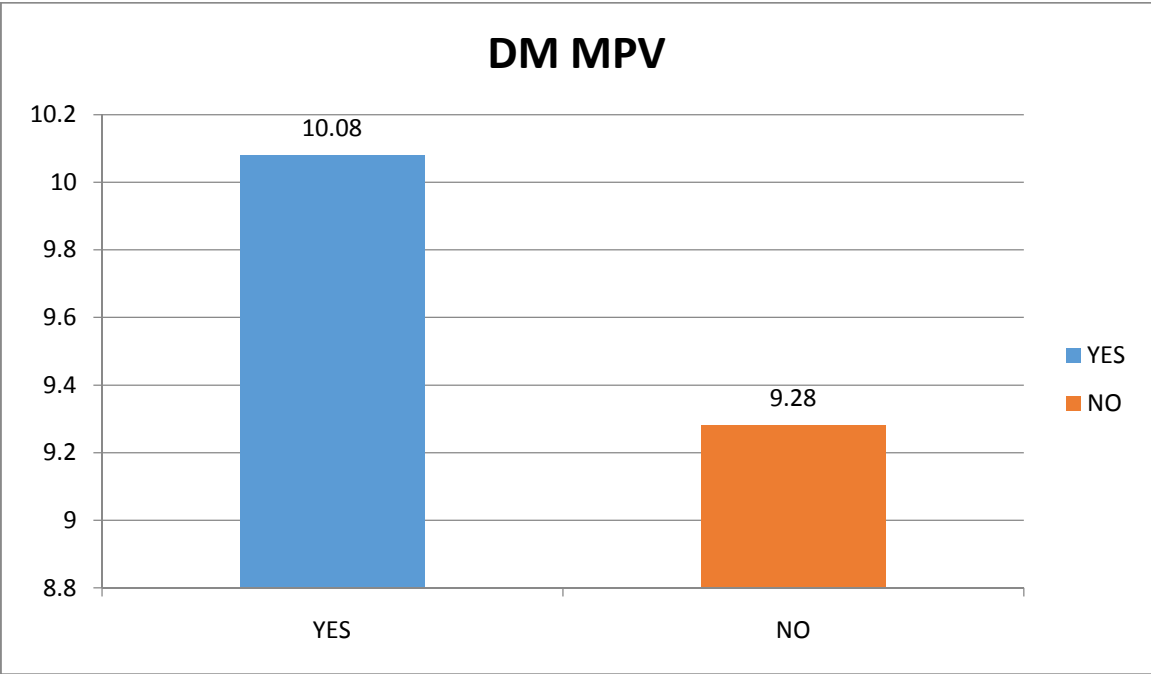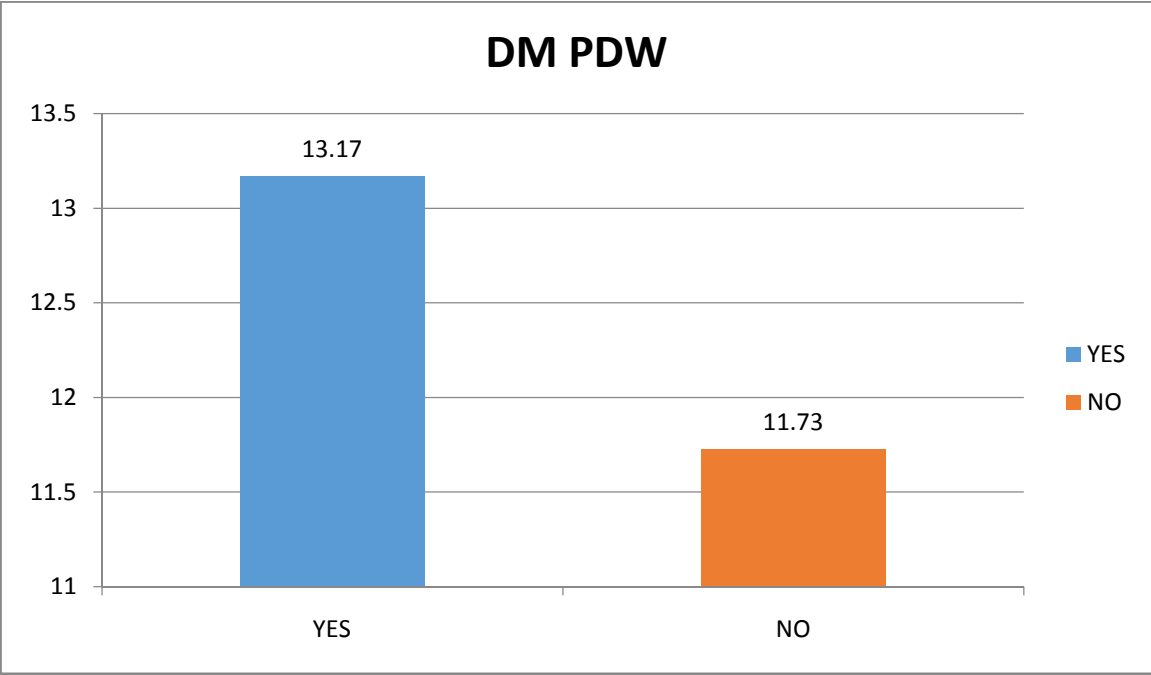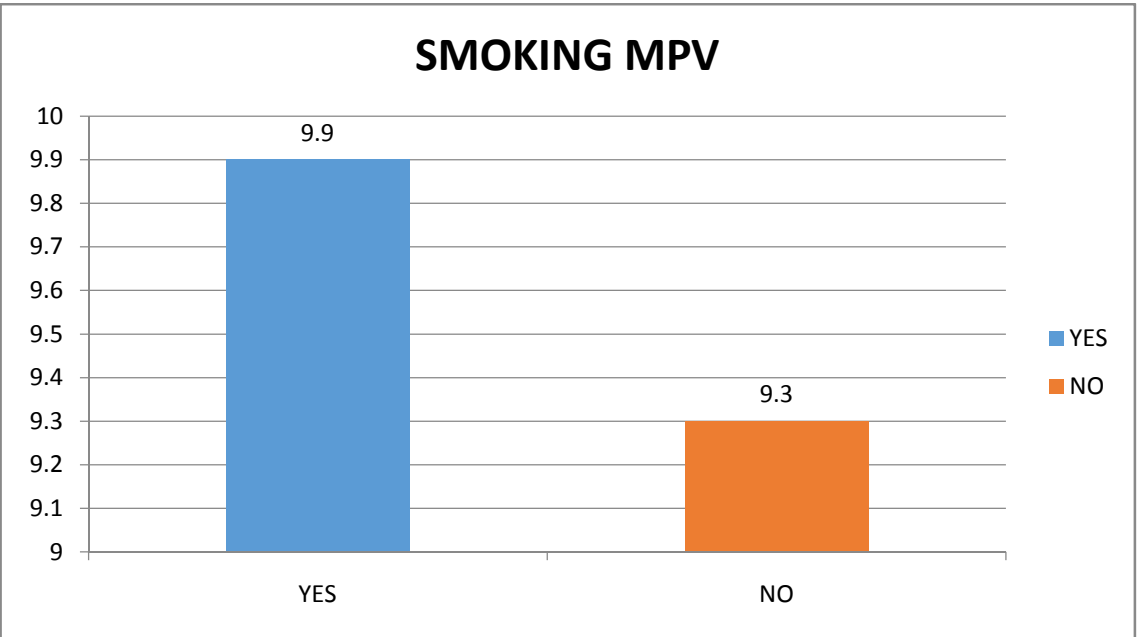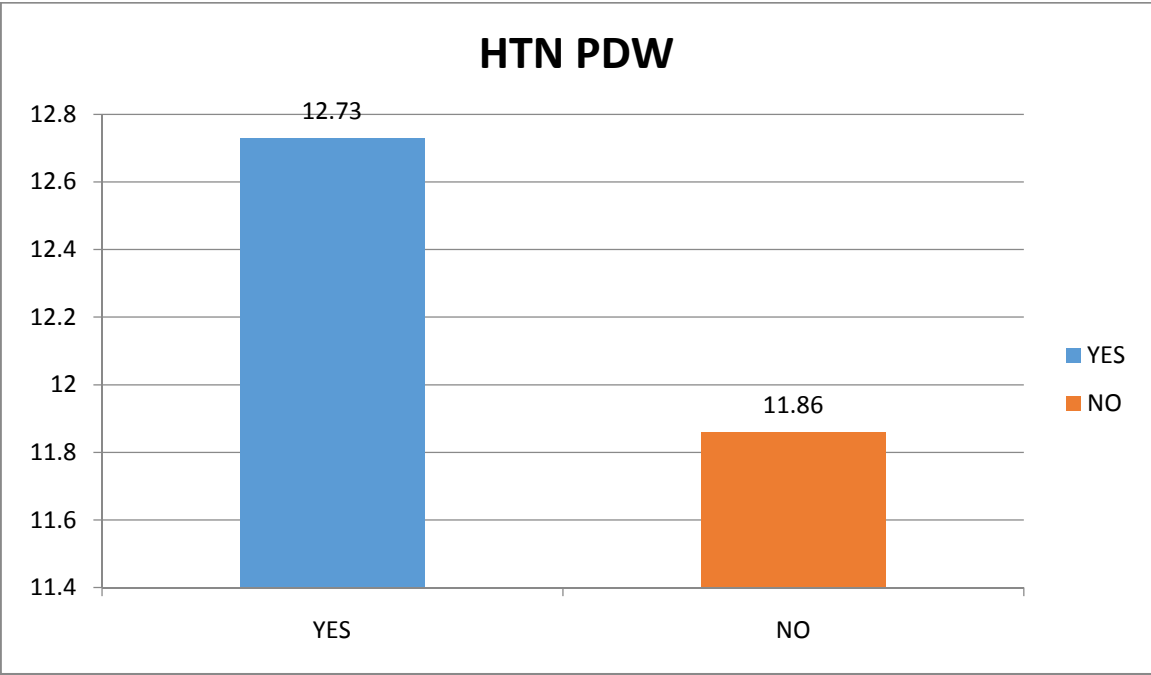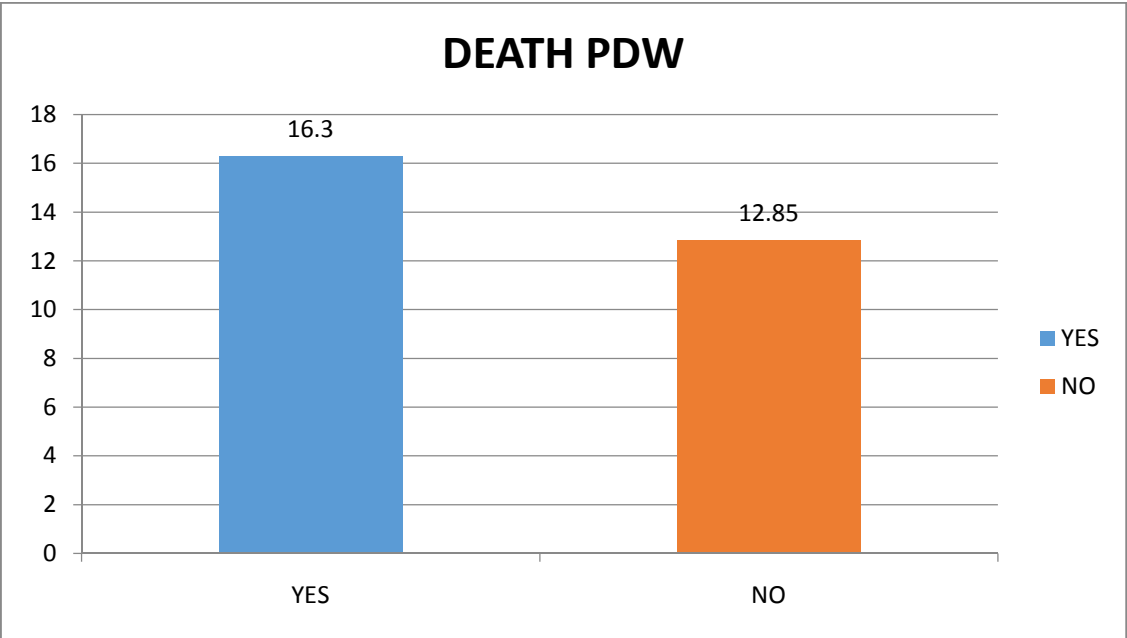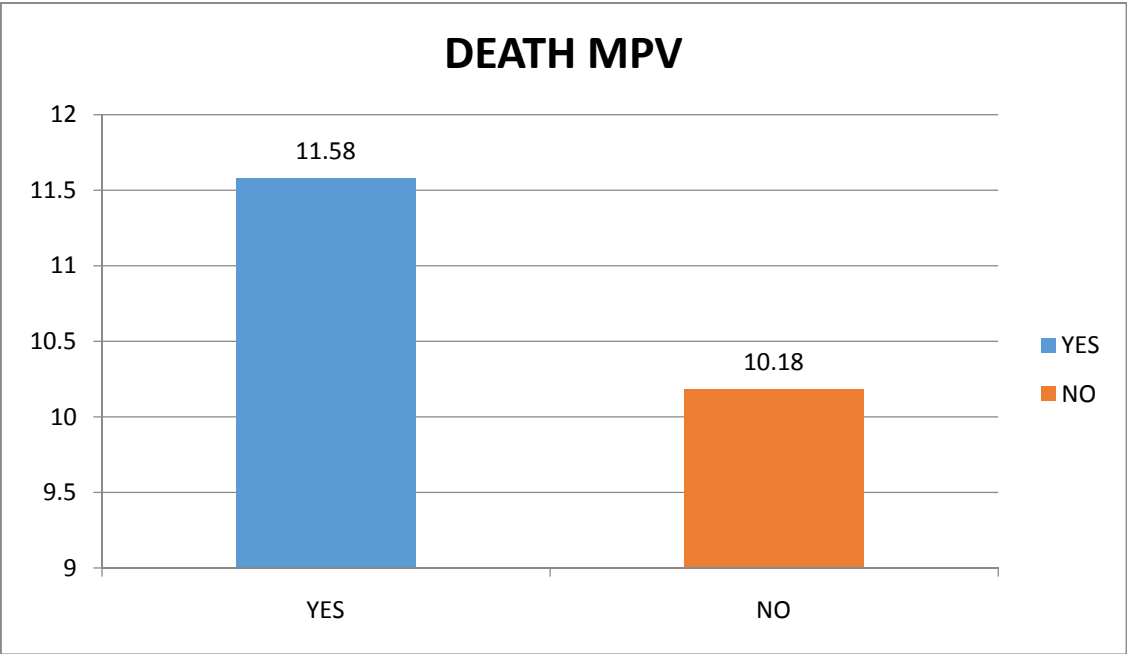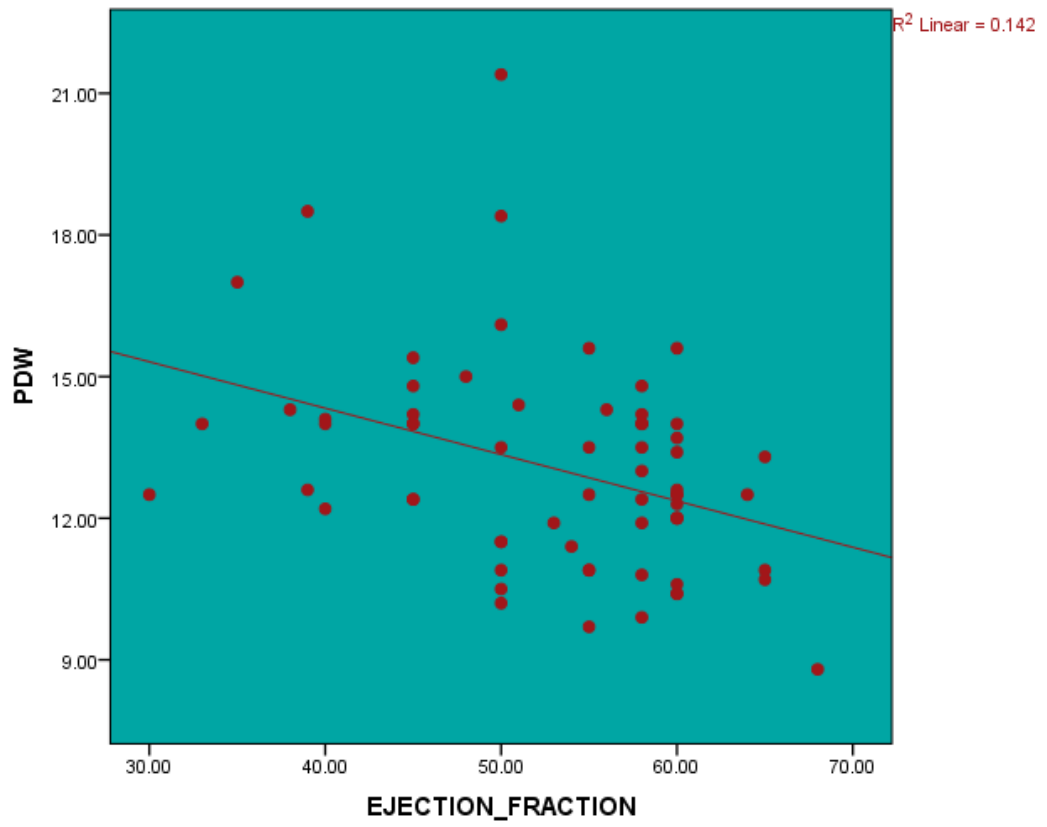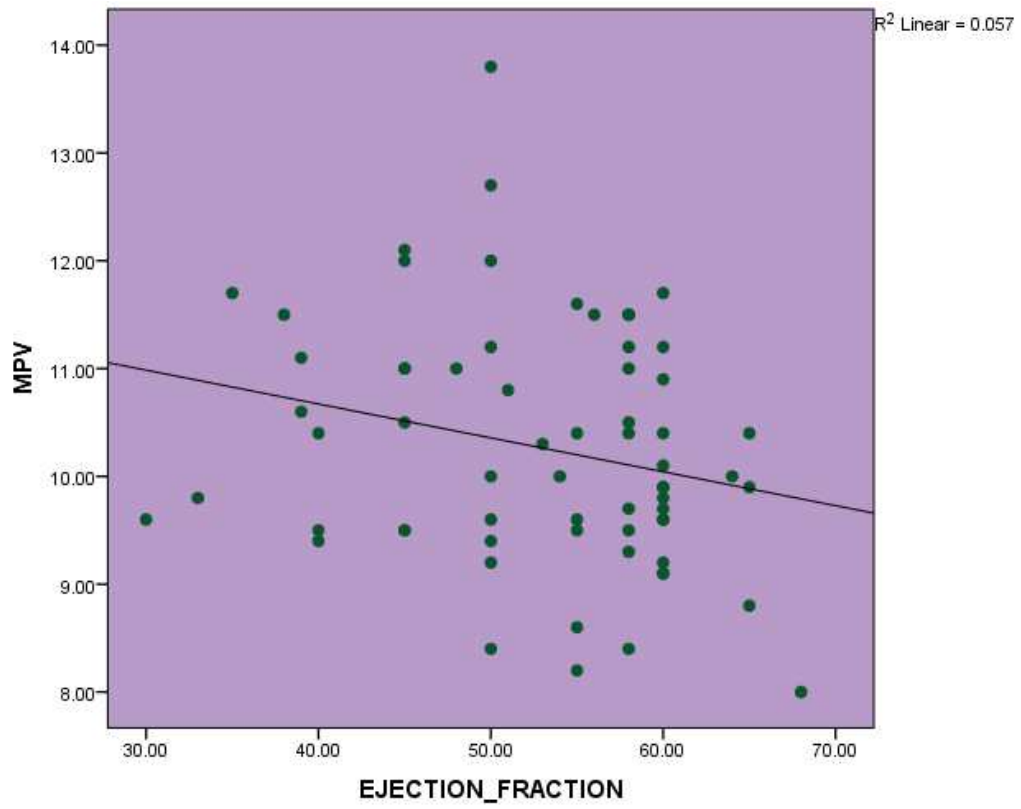
	CASE MI	CONTROL	P VALUE
AGE	52.98±15.00	49.03±10.36	0.167
MALE NO	45(69%)	25(71%)	0.819
CORONARY RISK FACTORS			
DM	36 (55%)	27(77%)	0.032*
HTN	30(46%)	2469%)	0.032
SMOKING	25(38%)	22(63%)	0.02*

DISCUSSION

Myocardial infarction is a major cause of morbidity and mortality in developed countries and is major problem in developing countries like India. The projection of large increase in Ischemic heart disease throughout worldwide, IHD is likely become most common of death worldwide by 2020.(Harrison's 2015)[43].Endogenous and exogenous risk factors like smoking, dyslipedemia, diabetes mellitus and systemic hypertension significantly increase the individual risk for myocardial infarction. However they only explain a part of the cases and there may be other relevant risk factors which need to be identified. Large platelets are more reactive, produce more thrombotic factors (Martin et al,1983)[44] and aggregate more easily (Haver et al,1981)[45]

Endler et al (2002)[46] stated that mean platelet volume (MPV) is an independent risk factor for myocardial infarction. Similar observation were made by Martin et al (1983),Cameron et al(1983)[47].But all these are studies conducted in the west with healthy western people serving as controls. so in this study an attempt was made to find out if any association existed between the platelet size and the occurrence of myocardial infarction among Indian population as reports are scanty.

In the study, the ages of the cases ranged from 20 to 80 years. The mean was 52.98 ±15 years. The maximum number of cases i.e.,25 cases fell in the age group 40-60 years. This comes to 38.5% of the total cases. With male predominant 69.2 % among total cases. This pattern corresponds to pattern reported in India, which is as follows. Coronary artery disease appears a decade earlier compared with the age incidence in developed countries and the peak period is attained between 51-60 years.(Park K.)

In the present study the relationship between age and MPV was Statistically significant as per Funiak et al (1994) found that MPV was significantly increased in patients of advanced age and it was statistically significant. But Bancroft et al (2000) stated that MPV decreases with age. But our study confirm with observation made by Funiak et al(1994).

Among 65 cases, 45 males (69.2%) and 20 females (30.8%).Before menopause, women have a lower age adjusted incidence and mortality for coronary heart disease than men. Gender specific incidence rates converge after menopause, suggesting a major role for oestrogen in delaying progression of atherosclerosis. Much of this effect results from beneficial action of oestrogen on lipid fractions. Estrogen reduces LDL-C by 10-15 %while increasing HDL-C.

In the present study MPV was independent of genders. This is in parallel with the observation made by Bancroft et al (2000).

Among 65 cases, 40 smokers (75.5%) and among 35 controls 13 smokers (24.5%).Although this shows an increased prevalence of smoking habit among cases than controls ,it was statistically significant. It has been calculated that in countries where smoking has been a wide spread habit, it is responsible for 25% of coronary heart disease death under 65 years of age(WHO).A uniquely human habit, smoking has been identified as a major coronary risk factor.

Although the MPV was higher among smokers compared to controls, the relationship was statistically significant. Tschope et al (1989)[48] and (Kario et al (1982) in separate studies found smokers to have an increased MPV. But Kishk et al (1985)[49] observed no relation between smoking and MPV. Our finding runs in parallel with that of Tschope et al(1985).

Among the myocardial infarction patients 35 had systemic hypertension (76.1%) .In study conducted by Gupta et al (2001) on myocardial infarction, the prevalence of systemic hypertension in myocardial infarction patients was found to be 32.6%.

In the study shows statistically significant relationship was observed between systemic hypertension and MPV, PDW. Osuna et al (1998) observed a higher MPV in those with systemic hypertension. In contrast, Bath et al (1996) observed no relationship between MPV and systemic hypertension.

Among myocardial infarction patients diabetes 29(78.4%).This is similar to the prevalence of diabetes mellitus in myocardial infarction patients in the study by Gupta et al (2001),which was 21%.

The MPV, PDW in patients with diabetes mellitus significantly larger from those without diabetes. Sharpe et al (1993)[50] stated that MPV and PDW was significantly increased in diabetic subjects compared with non diabetics. They stated that since the platelet size is a determinant of platelet function, with larger platelets being more reactive per unit volume, the platelets might play a part in the micro and macro vascular complications of diabetes mellitus. Osuna et al (1998) also observed a higher MPV with diabetes mellitus. The fact that in our study, observation runs parallel to that in Osuna et al (1998).

In the present study mean platelet count of the cases was 4.98±0.23lakhs/mm³ and that of the controls was 1.29±0.42 Lakhs/mm³.Although the mean platelet count among the cases was more

compared to the controls it was statistically significant. Senaran et al (2001)[51] stated that patients with acute myocardial infarction had increased platelet counts. Their observation was that in patients with myocardial infarction there were increased thrombopoeitin levels.

Increased thrombopoeitin levels may increase both platelet counts and platelet size, resulting in haemoststically more active platelets, which may contribute to the development and progression of coronary artery disease. However the observation by Senaran et al(2001) was in contrast to that of Cameron et al(1983) as they noticed that patients with acute myocardial infarction had a reduced platelet count compared to the controls. They suggested that reduced platelet count may be due to consumption of platelet at the thrombus site. But in our study, there was significant difference in the platelet counts between the case and the control groups.

There was significant relationship between platelet count and MPV, PDW in our study. There are few studies regarding this aspect. Senaran et al(2001) observed both an increased platelet count and MPV in patients with myocardial infarction as stated above. But Cameron et al (1983) observed a reduced platelet count and higher MPV in patient with myocardial infarction. They stated that the reduced platelet count may be due to the consumption of platelet at the thrombus site as stated above.

The observation in our study runs in parallel with the finding of Senaran et al.

The MPV of the cases ranged from 8.4 to 12 femtolitres with a mean of 10.2 ± 1.1femtolitres.The MPV of the controls ranged from 7 to 10.60 femtolitres with mean of 8.29±0.9 femtolitres. The difference between the two was statistically significant.

Previous studies on MPV and myocardial infarction have given different observations.

Halbmayer et al(1995) stated that MPV did not differ significantly between patients and controls in their study. In contrast to this Cameron et al (1983) found an increased mean platelet volume among those with acute myocardial infarction compared to controls.

Similar finding was observed by Martin et al(1983) in their study. Large platelets are denser, they produce more thromboxane A2 per unit volume of platelet cytoplasm. Large platelets aggregate more rapidly upon collagen challenge, release more serotonin and other granule contents and express more receptors per unit area(pizzuli et al,1998).So an increased MPV, as an indicator of larger, more reactive platelets, may represent a risk factor for myocardial infarction(Endler et al,2002)

One question is that, does the increase in platelet volume occur before the event of myocardial infarction or does it occur as a response to the platelet consumption in myocardial infarction. Martin et al (1983) who did a study on mean platelet volume in acute myocardial infarction measured platelet volume within 12 hours of admission to hospital and then later at 6 weeks. They stated that mean platelet volume was increased before myocardial infarction occurred for the following reasons. They said that increase in volume seen within the first 12 hours of admission suggested that the increase was present before infarction, as the life span of the platelet is about 8 days. More than 90% of the platelet population whose distribution was measured after myocardial infarction were circulating before the vascular occlusion occurred. They also observed that the increase in MPV persisted six weeks after discharge from hospital, when infarct would have been largely healed. This also supported the view that platelet volume was chronically large in the infarct group.

Further proof that the increase in mean platelet volume occurs before acute myocardial infarction is got from the study conducted by Endler et al (2002).They evaluated mean platelet volume in patients presenting with acute myocardial infarction and in patients with a history of previous myocardial infarction which had occurred upto 40 years

before the study. They compared it with those without myocardial infarction. They found that the MPV was significantly raised in those with acute MI and in those with history of previous myocardial infarction when compared to those without myocardial infarction. They also observed that there was no significant difference in the MPV between those with acute myocardial infarction and those with history of previous myocardial infarction. This showed that the time interval between the occurrence of MI and the estimation of MPV did not influence the value of MPV. This further adds to the proof that raised MPV precedes the occurrence of MI.

O'Malley et al (1995) conducted a similar study of MPV estimation in stroke patients. They estimated MPV in stroke patients within 48 hours of admission and also at 6 months later and compared it with controls. They found that MPV was significantly raised in stroke patients compared to controls. They suggested that the changes in MPV might have preceded the vascular event and is unlikely to be due to platelet consumption at the infarct site. They said that since the average life span of the platelets is about 8 days, elevated MPV seen within the first 48 hours after stroke represented platelet released before infarction.

Further it was unlikely that platelet consumption due to localized thrombosis would affect peripheral venous estimation of platelet

variables. They further stated that since there was no difference in MPV between large cortical strokes and small lacunar infarcts, it was unlikely that platelet consumption at the thrombus site would affect the peripheral venous estimation of platelet variables. They also added that, the fact that the observed increase in MPV had remained unchanged in post stroke survivors was further evidence that changes in MPV were likely to have preceded the acute event.

Although the highest MPV of 13.8 femtolitres, PDW 16.1 ,platelet count4, 27,000 was observed in those with least ejection fraction i.e $\leq$ 45%, there was statistically significant. Hendra et al (1988) stated that all patients with severe cardiac failure had larger platelet volume than patients with mild or no failure. Osuna et al (1998) also observed that an increased platelet volume was related to higher risk of cardiac failure.

But our study showed only significant higher platelet volumes in those with left ventricular dysfunction.

There was no significant difference in MPV among the group that survived without complication and the rest. The 4 patients who died did not have statistically significant MPV but had higher PDW level ranged 18 femtolitres.

According to Hendra J et al (1988)[52],Osuna et al(1998) and Martin et al(1991) ,the increase in MPV appear to provide prognostic information during the period of study. They observed that the MPV correlate with more established factors determining prognosis after infarction such as the size of the infarct. Their observation were in contrast to the observation by Cameron et al(1983)

According to Assiri et al(2012) [53]stated that platelet volume parameters mainly MPV and PDW are ,available and inexpensive markers ,detected significantly raised in patients admitted with acute coronary syndrome. From these observation, they suggested to use platelet parameters used to screen patients presenting to emergency room with chest pain suspected to have acute coronary syndrome.

LIMITATIONS

A multi centric study with a larger sample size and longer follow up is essential to assess the predictive power of these prognostication tools in a more comprehensive manner.

AREAS OF FURTHER WORK

1. MPV studies in other thrombotic episodes

2. Follow up study of MPV after the onset of clinical events and finding out association between MPV and platelet count by apply the log probability formula.

3. Studies to find out the physiological mechanism which regulates MPV within megakaryocyte.

4. Invitro studies on factors contributing for thyromegaly in order to introduce interventional measures.

CONCLUSION

➢ MPV was significantly elevated in patients with acute myocardial infarction (10.2 ± 1.1femtolitres) when compared with controls8 (29±0.9 femtolitres).

➢ MPV in patients with acute myocardial infarction was found to be elevated with advancing age, smoking, diabetes mellitus and systemic hypertension.

➢ Aspirin was not found to have any significant association with MPV

➢ Observed correlation between elevated platelet count and MPV

➢ Observed correlation between MPV and short term outcome after myocardial infarction.

The study "The platelet count and mean platelet volume and platelet distribution width in myocardial infarction "was done to assess the relationship between mean platelet volume (MPV) and the occurrence of acute myocardial infarction and to find out its association with the risk factors and short term prognosis after myocardial infarction With rigid criteria 65 patients were selected carefully and were evaluated on social, clinical and laboratory aspects after institutional ethical clearance with an informed consent.35 subjects were taken as controls. The data were entered in Microsoft Excel spread sheet and analysed statistically.

There were 45 males and 20 females in patient group. There were 25 males and 10 females in control group. The mean age of the patient

group was 52.98±15 and that of the control group 49.03±10.36 (p value 0.167 –not statistically significant) .The prevalence of smoking systemic hypertension, and diabetes mellitus were noticed in both group with statistical significance. The mean platelet count of the cases was 4.98±0.23lakhs/mm³ and that of the controls was 1.29±0.42 Lakhs/mm³ (p value 0.050 statistically significant). The MPV of the cases ranged from 8.4 to 12 femtolitres with a mean of 10.2 ± 1.1femtolitres.The MPV of the controls ranged from 7 to 10.60 femtolitres with mean of 8.29±0.9 femtolitres. The difference between the two was statistically significant.(p value 0.001 significant).

There was significantly relationship between age, sex, smoking, diabetes mellitus and systemic hypertension platelet count, MPV, PDW.MPV was found to be independent of aspirin intake. There was correlation observed between MPV and short term prognosis like left ventricular dysfunction and death.

According to Assiri et al (2012) stated that platelet volume parameters mainly MPV and PDW are available and inexpensive markers, detected significantly raised in patients admitted with acute coronary syndrome. From these observation, they suggested to use platelet parameters used to screen patients presenting to emergency room with chest pain suspected to have acute coronary syndrome.

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PROFORMA
Platelet count and Mean platelet volume and platelet distribution Width in Acute coronary syndrome

Name   :

Age/Sex   :

Socioeconomic status:

Marital status:

Occupation:

 HISTORY OF PRESENTING ILLNESS

1.chest pain

2.breathlessness

3.cough

4.palpitation

5.sweating

6.syncope

 PAST HISTORY

1.Hypertension

2.Dyslipidemia

3.Acute or chronic renal disease

4.Chronic lung disease

5.Diabetes Mellitus
6.Anemia

7.Drug or alcohol abuse

PERSONEL HISTORY

Smoking

Alcohol

VITALS

Pulse rate:

Respiratory rate:

Blood pressure:

Duration of stay :

General examination

Systemic examination

CVS: RS: P/A:
      CNS:

INVESTIGATIONS :

BLOOD PLATELET COUNT

MEAN PLATELET VOLUME

PLATELET DISTRIBUTION WIDTH

ECHO   COMPLICATION   DEATH

ABBREVIATIONS AND ACRONOMYS

ADP   -   Adenosine diphosphate

BP   -   Blood pressure

CAD   -   Coronary Artery Disease

MI   -   Myocardial infarction

MPV   -   Mean platelet Volume

NO   -   Nitric Oxide

PGI2   -   Prostacyclin

vWF   -   von Willebrand factor.

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

CERTIFICATE OF APPROVAL

To
Dr.M.Anitha
Post Graduate in MD General Medicine
Madras Medical College
Chennai 600 003

Dear Dr.M.Anitha,

The Institutional Ethics Committee has considered your request and approved your study titled **"A STUDY OF PLATELET COUNT AND THEIR INDICES – MEAN PLATELET VOLUME AND PLATELET DISTRIBUTION WIDTH AND THEIR PROGNOSTIC SIGNIFICANCE IN ACUTE CORONARY SYNDROME IN TERTIARY CARE HOSPITAL"** - **NO.03032016.**
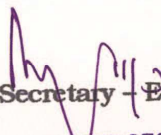
The following members of Ethics Committee were present in the meeting hold on **01.03.2016** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr.R.Vimala,MD.,Dean,MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4.Prof.B.Vasanthi,MD.,Inst.of Pharmacology,MMC,Ch-3	: Member
5.Prof.P.Raghumani,MS, Dept.of Surgery,RGGGH,Ch-3	: Member
6.Dr.Baby Vasumathi, Director, Inst. of O&G,Ch-8	: Member
7.Prof.M.Saraswathi,MD.,Director, Inst.of Path,MMC,Ch-3	: Member
8.Prof.Srinivasagalu,Director,Inst.of Int.Med.,MMC,Ch-3	: Member
9.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person
10.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
11.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

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

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

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

Originality | GradeMark | PeerMark

A study on platelet count and their indices
BY 201411002 MD GENMED ANITHA.M

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A STUDY ON PLATELET COUNT AND THEIR INDICES-MEAN
PLATELET VOLUME AND PLATELET DISTRIBUTION WIDTH
AND THEIR PROGNOSTIC SIGNIFICANCE IN ACUTE CORONARY
SYNDROME IN TERTIARY CARE HOSPITAL

Match Overview

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A STUDY ON PLATELET COUNT AND THEIR INDICES-MEAN
PLATELET VOLUME AND PLATELET DISTRIBUTION WIDTH
AND THEIR PROGNOSTIC SIGNIFICANCE IN ACUTE CORONARY
SYNDROME IN TERTIARY CARE HOSPITAL

INFORMATION SHEET

We are conducting a study on **"A STUDY ON PLATELET COUNT AND THEIR INDICES –MEAN PLATELET VOLUME AND PLATELET DISTRIBUTION WIDTH AND THEIR PROGNOSTIC SIGNIFICANCE IN ACUTE CORONARY SYNDROME IN TERTIARY CARE HOSPITAL"** among patients attending Rajiv Gandhi Government General Hospital, Chennai and for that your specimen may be valuable to us.

The purpose of this study is to assess the**"A STUDY ON PLATELET COUNT AND THEIR INDICES –MEAN PLATELET VOLUME AND PLATELET DISTRIBUTION WIDTH AND THEIR PROGNOSTIC SIGNIFICANCE IN ACUTE CORONARY SYNDROME IN TERTIARY CARE HOSPITAL"** and We are selecting certain cases and if you are found eligible physical examination done. 5ml blood will be collected and 2ml of urine will be collected. You will also undergo echocardiogram, electrocardiogram, chest x-ray. These tests and special studies do not affect your final report or management.

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of Investigator   signature of participant

Date:

Place:

<u>ஆராய்ச்சி தகவல் தாள்</u>

சென்னை இராஜிவ் காந்தி அரசு பொது மருத்துவமனையில் "இரத்த தட்டுகளின் எண்ணிக்கை மற்றும் மூன்றாம் நிலை மருத்துவமனையில் தீவிர மகுட நோய் தங்கள் குறியீடுகளில் என்றே பிளேட்லெட் தொகுதி மற்றும் பிளேட்லெட் விநியோகம் அகலம்" பற்றிய ஒரு ஆராய்ச்சி நடைபெற்று வருகிறது.

இருதய நோயினால் பிளேட்லெட் தொகுதி மற்றும் பிளேட்லெட் விநியோகம் அகலம் பற்றி இரத்த பரிசோதனை மூலம் அறிவதே இந்த ஆராய்ச்சியின் நோக்கமாகும்.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். முடிவுகளை அல்லது கருத்துக்களை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிடமாட்டோம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த சிறப்புப் பரிசோதனைகளின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவில் தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம்   பங்கேற்பாளர் கையொப்பம்

தேதி:

PATIENTS CONSENT FORM

Study title   :   **A STUDY ON PLATELET COUNT AND THEIR INDICES –MEAN PLATELET VOLUME AND PLATELET DISTRIBUTION WIDTH AND THEIR PROGNOSTIC SIGNIFICANCE IN ACUTE CORONARY SYNDROME IN TERTIARY CARE HOSPITAL**

Study Centre   :   Rajiv Gandhi Government General Hospital, Chennai.

Name   :

Age/Sex   :

Identification   :
Number

Patient may check (☑) these boxes

The details of the study have been provided to me in writing and explained to me in my own language

❏

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected.

❏

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required

❏

<center>ஆராய்ச்சி ஒப்புதல் கடிதம்</center>

ஆராய்ச்சி தலைப்பு:

"இரத்த தட்டுகளின் எண்ணிக்கை மற்றும் மூன்றாம் நிலை மருத்துவமனையில் தீவிர மகுட நோய் தங்கள் குறியீடுகளில் என்றே பிளேட்லெட் தொகுதி மற்றும் பிளேட்லெட் விநியோகம் அகலம்"பற்றிய ஆராய்ச்சி.

பெயர்:   தேதி:

வயது:   உள்நோயாளி எண்:

பால்:   ஆராய்ச்சி சேர்க்கை எண்:

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது. எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு நான் எனது சம்மதத்தை தெரிவிக்கிறேன்.

இருதய நோயினால் பிளேட்லெட் தொகுதி மற்றும் பிளேட்லெட் விநியோகம் அகலம் பற்றி பரிசோதனைகளைப் பற்றியும் ஆராய்ச்சியாளர் கூற முழுவதும் விளங்கப்பெற்றேன்.

மேற்கொண்ட பரிசோதனையின் போது ஏற்படக்கூடிய பின்விளைவுகளையும் முழுவதும் உணர்ந்து இந்த பரிசோதனைக்கு மனமார சம்மதிக்கிறேன்.

<div align="right">கையொப்பம்</div>

under the law. I agree not to restrict the use of any data or results that arise from this study.

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or wellbeing or any unexpected or unusual symptoms.

❏

I hereby consent to participate in this study.

❏

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological and biochemical tests.

❏

Signature/thumb impression

Patient's Name and Address:

Signature of Investigator
Study Investigator's Name:
Dr. M.ANITHA

SERIAL_NO	GROUP	HOSPITAL_N	AGE	SEX	SMOKING	DIABETES	YPERTENSIO	ON_ASPIRIN	STEMI	NSTEMI	NSTABLE_ANGIN	LATELET_COUN	PDW	MPV	JECTION_FRACTIO	OMPLICATIO	DEATH	AGE_GROUP	filter_$
1.00	1	95,795.00	50.00	2.00	2.00	2.00	2.00	1.00	1.00	2.00	2.00	279,000.00	14.20	11.00	45.00	1.00	2.00	2.00	1
2.00	1	92,255.00	48.00	1.00	1.00	2.00	1.00	1.00	1.00	2.00	2.00	94,000.00	14.00	11.20	60.00	2.00	2.00	2.00	1
3.00	1	89,260.00	52.00	1.00	1.00	2.00	1.00	1.00	1.00	2.00	2.00	427,000.00	10.90	8.20	55.00	2.00	2.00	2.00	1
4.00	1	89,279.00	55.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	2.00	300,000.00	10.50	9.20	50.00	1.00	2.00	2.00	1
5.00	1	92,279.00	40.00	1.00	2.00	1.00	2.00	2.00	1.00	2.00	2.00	255,000.00	14.00	10.50	45.00	1.00	2.00	1.00	1
6.00	1	92,201.00	35.00	1.00	2.00	2.00	2.00	2.00	1.00	2.00	2.00	206,000.00	15.60	11.60	55.00	2.00	2.00	1.00	1
7.00	1	94,857.00	52.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	2.00	8,800,000.00	14.80	12.10	45.00	1.00	2.00	2.00	1
8.00	1	97,063.00	61.00	2.00	1.00	1.00	1.00	1.00	1.00	2.00	2.00	245,000.00	14.10	9.50	40.00	1.00	2.00	3.00	1
9.00	1	98,011.00	70.00	1.00	1.00	1.00	1.00	1.00	1.00	2.00	2.00	150,000.00	14.30	11.50	38.00	1.00	1.00	3.00	1
10.00	1	98,189.00	70.00	2.00	2.00	2.00	2.00	1.00	1.00	2.00	2.00	317,000.00	17.00	11.70	35.00	1.00	1.00	3.00	1
11.00	1	98,901.00	45.00	1.00	1.00	2.00	1.00	1.00	1.00	2.00	2.00	228,000.00	18.40	12.70	50.00	1.00	2.00	2.00	1
12.00	1	94,562.00	83.00	2.00	2.00	1.00	1.00	1.00	1.00	2.00	2.00	299,000.00	18.50	11.10	39.00	1.00	1.00	4.00	1
13.00	1	95,966.00	65.00	1.00	1.00	2.00	1.00	1.00	1.00	2.00	2.00	141,000.00	12.40	9.50	45.00	2.00	2.00	3.00	1
14.00	1	98,857.00	53.00	1.00	1.00	1.00	2.00	1.00	1.00	2.00	2.00	222,000.00	16.10	12.00	50.00	2.00	2.00	2.00	1
15.00	1	98,995.00	65.00	1.00	1.00	2.00	1.00	1.00	1.00	2.00	2.00	163,000.00	14.40	10.80	51.00	2.00	2.00	3.00	1
16.00	1	96,693.00	70.00	1.00	1.00	1.00	2.00	1.00	2.00	2.00	2.00	150,000.00	14.30	11.50	56.00	2.00	2.00	3.00	1
17.00	1	97,191.00	50.00	2.00	1.00	2.00	2.00	2.00	1.00	2.00	2.00	173,000.00	21.40	13.80	50.00	1.00	2.00	2.00	1
18.00	1	97,192.00	44.00	2.00	2.00	2.00	1.00	1.00	1.00	2.00	2.00	327,000.00	12.50	9.60	30.00	2.00	2.00	2.00	1
19.00	1	94,302.00	57.00	2.00	2.00	2.00	2.00	2.00	1.00	2.00	2.00	404,000.00	12.40	9.50	45.00	2.00	2.00	2.00	1
20.00	1	95,062.00	60.00	2.00	2.00	1.00	2.00	1.00	1.00	2.00	2.00	275,000.00	11.50	10.00	50.00	2.00	2.00	2.00	1
21.00	1	93,985.00	40.00	2.00	2.00	1.00	2.00	2.00	1.00	1.00	2.00	214,000.00	13.50	11.20	50.00	2.00	2.00	1.00	1
22.00	1	94,867.00	55.00	2.00	2.00	2.00	2.00	2.00	2.00	1.00	2.00	180,000.00	14.00	11.20	58.00	2.00	2.00	2.00	1
23.00	1	98,456.00	34.00	2.00	2.00	2.00	2.00	2.00	2.00	1.00	2.00	211,000.00	12.00	9.70	60.00	2.00	2.00	1.00	1
24.00	1	97,456.00	70.00	2.00	1.00	2.00	1.00	2.00	1.00	1.00	2.00	191,000.00	15.60	11.70	60.00	1.00	2.00	3.00	1
25.00	1	95,432.00	65.00	2.00	1.00	1.00	1.00	1.00	2.00	1.00	2.00	343,000.00	13.50	10.40	55.00	2.00	2.00	3.00	1
26.00	1	94,663.00	42.00	2.00	2.00	2.00	2.00	2.00	2.00	1.00	2.00	267,000.00	11.50	9.40	50.00	2.00	2.00	2.00	1
27.00	1	93,356.00	32.00	1.00	1.00	2.00	1.00	1.00	2.00	1.00	2.00	170,000.00	11.40	10.00	54.00	2.00	2.00	1.00	1
28.00	1	98,917.00	37.00	1.00	1.00	2.00	2.00	1.00	2.00	1.00	2.00	98,000.00	14.00	11.00	58.00	2.00	2.00	1.00	1
29.00	1	97,645.00	78.00	1.00	1.00	1.00	1.00	1.00	2.00	1.00	2.00	183,000.00	12.40	10.50	58.00	2.00	2.00	3.00	1
30.00	1	94,487.00	70.00	2.00	2.00	2.00	2.00	2.00	2.00	1.00	2.00	289,000.00	12.50	10.00	64.00	2.00	2.00	3.00	1
31.00	1	95,966.00	73.00	1.00	1.00	2.00	1.00	1.00	2.00	1.00	2.00	270,000.00	12.60	10.60	39.00	1.00	2.00	3.00	1
32.00	1	97,825.00	34.00	1.00	1.00	1.00	2.00	2.00	2.00	1.00	2.00	255,000.00	10.20	8.40	50.00	2.00	2.00	1.00	1
33.00	1	97,461.00	87.00	1.00	1.00	2.00	1.00	1.00	1.00	2.00	2.00	260,000.00	10.90	9.60	55.00	2.00	2.00	4.00	1

C1	C2	C3	C4	C5	C6	C7	C8	C9	C10	C11	C12	C13	C14	C15	C16	C17	C18	C19	C20
1	2.00	2.00	2.00	55.00	9.50	12.50	254,000.00	2.00	1.00	2.00	1.00	2.00	2.00	1.00	1.00	54.00	95,492.00	1	34.00
1	3.00	2.00	2.00	58.00	9.50	10.80	235,000.00	2.00	1.00	2.00	1.00	2.00	2.00	2.00	1.00	74.00	99,031.00	1	35.00
1	2.00	2.00	2.00	55.00	8.60	9.70	188,000.00	2.00	1.00	2.00	1.00	1.00	1.00	2.00	1.00	52.00	98,851.00	1	36.00
1	1.00	2.00	2.00	60.00	9.10	12.30	120,000.00	2.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	35.00	99,099.00	1	37.00
1	3.00	2.00	2.00	58.00	11.50	14.20	66,000.00	2.00	2.00	1.00	2.00	2.00	2.00	1.00	1.00	64.00	96,713.00	1	38.00
1	3.00	2.00	2.00	60.00	9.60	12.50	120,000.00	2.00	1.00	2.00	1.00	2.00	2.00	1.00	1.00	65.00	99,211.00	1	39.00
1	2.00	2.00	2.00	58.00	11.50	13.00	296,000.00	2.00	2.00	1.00	2.00	2.00	2.00	1.00	1.00	42.00	96,610.00	1	40.00
1	2.00	2.00	2.00	45.00	11.00	14.00	225,000.00	1.00	2.00	2.00	2.00	1.00	1.00	1.00	1.00	50.00	96,951.00	1	41.00
1	3.00	2.00	2.00	58.00	8.40	9.90	260,000.00	1.00	2.00	2.00	2.00	2.00	2.00	2.00	2.00	61.00	98,546.00	1	42.00
1	3.00	2.00	2.00	60.00	9.10	10.40	345,000.00	1.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	65.00	96,031.00	1	43.00
1	2.00	2.00	2.00	50.00	9.60	10.90	325,000.00	1.00	2.00	2.00	2.00	2.00	2.00	1.00	1.00	55.00	96,924.00	1	44.00
1	2.00	2.00	2.00	48.00	11.00	15.00	480,000.00	1.00	2.00	2.00	2.00	2.00	2.00	1.00	1.00	45.00	98,462.00	1	45.00
1	3.00	2.00	2.00	33.00	9.80	14.00	198,000.00	1.00	2.00	2.00	2.00	2.00	2.00	1.00	1.00	62.00	93,476.00	1	46.00
1	2.00	2.00	2.00	40.00	9.40	12.20	355,000.00	1.00	2.00	2.00	2.00	2.00	2.00	2.00	1.00	49.00	97,834.00	1	47.00
1	3.00	2.00	2.00	40.00	10.40	14.00	141,400.00	1.00	2.00	2.00	2.00	2.00	2.00	2.00	1.00	65.00	93,562.00	1	48.00
1	1.00	2.00	2.00	53.00	10.30	11.90	304,000.00	1.00	2.00	2.00	2.00	1.00	1.00	2.00	1.00	39.00	94,567.00	1	49.00
1	3.00	2.00	2.00	60.00	9.60	12.50	327,000.00	1.00	2.00	2.00	2.00	2.00	2.00	2.00	2.00	61.00	97,168.00	1	50.00
1	1.00	2.00	2.00	60.00	10.90	13.70	198,000.00	1.00	2.00	2.00	2.00	2.00	2.00	2.00	2.00	38.00	97,271.00	1	51.00
1	3.00	2.00	2.00	58.00	9.30	11.90	111,400.00	1.00	2.00	2.00	1.00	1.00	1.00	2.00	2.00	66.00	97,763.00	1	52.00
1	1.00	2.00	2.00	60.00	10.10	12.60	297,000.00	1.00	2.00	2.00	2.00	2.00	2.00	1.00	1.00	20.00	92,245.00	1	53.00
1	1.00	2.00	2.00	65.00	9.90	10.90	199,000.00	1.00	2.00	2.00	2.00	2.00	2.00	1.00	1.00	25.00	93,465.00	1	54.00
1	1.00	2.00	2.00	68.00	8.00	8.80	448,000.00	1.00	2.00	2.00	2.00	2.00	2.00	1.00	1.00	28.00	94,567.00	1	55.00
1	1.00	2.00	2.00	60.00	9.20	10.60	243,000.00	1.00	2.00	2.00	2.00	1.00	1.00	1.00	1.00	32.00	95,147.00	1	56.00
1	2.00	2.00	2.00	60.00	9.80	10.40	166,000.00	1.00	2.00	2.00	2.00	2.00	2.00	2.00	1.00	47.00	97,812.00	1	57.00
1	2.00	2.00	2.00	60.00	9.90	12.00	305,000.00	1.00	2.00	2.00	2.00	2.00	2.00	2.00	1.00	45.00	93,265.00	1	58.00
1	1.00	2.00	2.00	65.00	10.40	13.30	100,000.00	1.00	2.00	2.00	1.00	1.00	1.00	1.00	1.00	28.00	92,792.00	1	59.00
1	3.00	2.00	2.00	65.00	8.80	10.70	113,000.00	1.00	2.00	2.00	2.00	2.00	2.00	2.00	2.00	77.00	95,289.00	1	60.00
1	3.00	2.00	2.00	60.00	10.40	13.40	278,000.00	2.00	2.00	2.00	2.00	2.00	2.00	2.00	2.00	63.00	94,567.00	1	61.00
1	2.00	2.00	2.00	58.00	9.70	14.80	167,000.00	2.00	2.00	1.00	1.00	1.00	1.00	1.00	1.00	56.00	90,345.00	1	62.00
1	2.00	1.00	1.00	45.00	12.00	15.40	230,000.00	2.00	2.00	1.00	1.00	1.00	1.00	2.00	1.00	52.00	92,765.00	1	63.00
1	2.00	2.00	2.00	60.00	9.90	12.00	124,000.00	2.00	1.00	2.00	2.00	2.00	2.00	2.00	2.00	39.00	99,167.00	1	64.00
1	2.00	2.00	2.00	58.00	10.40	13.50	8,800,000.00	2.00	2.00	1.00	2.00	1.00	2.00	1.00	1.00	48.00	90,348.00	1	65.00
0	2.00	#NULL!	#NULL!	#NULL!	8.20	11.00	110,000.00	#NULL!	#NULL!	#NULL!	#NULL!	#NULL!	2.00	1.00	1.00	52.00	94,792.00	2	1.00
0	1.00	#NULL!	#NULL!	#NULL!	8.80	10.90	111,000.00	#NULL!	#NULL!	#NULL!	#NULL!	#NULL!	1.00	2.00	1.00	40.00	91,144.00	2	2.00

0	1.00	#NULL!	#NULL!	#NULL!	7.80	9.60	134,000.00	#NULL!	#NULL!	#NULL!	2.00	2.00	2.00	1.00	38.00	88,450.00	2	3.00
0	2.00	#NULL!	#NULL!	#NULL!	7.00	10.80	137,000.00	#NULL!	#NULL!	#NULL!	1.00	1.00	1.00	1.00	55.00	86,274.00	2	4.00
0	2.00	#NULL!	#NULL!	#NULL!	8.00	12.00	114,000.00	#NULL!	#NULL!	#NULL!	#NULL!	1.00	1.00	1.00	47.00	93,377.00	2	5.00
0	1.00	#NULL!	#NULL!	#NULL!	8.30	11.40	120,000.00	#NULL!	#NULL!	#NULL!	2.00	2.00	2.00	2.00	35.00	91,102.00	2	6.00
0	3.00	#NULL!	#NULL!	#NULL!	9.30	11.20	126,000.00	#NULL!	#NULL!	#NULL!	2.00	1.00	2.00	1.00	66.00	95,574.00	2	7.00
0	2.00	#NULL!	#NULL!	#NULL!	8.50	12.40	128,000.00	#NULL!	#NULL!	#NULL!	2.00	2.00	1.00	1.00	45.00	94,422.00	2	8.00
0	1.00	#NULL!	#NULL!	#NULL!	7.80	11.30	115,000.00	#NULL!	#NULL!	#NULL!	2.00	2.00	2.00	1.00	38.00	96,782.00	2	9.00
0	2.00	#NULL!	#NULL!	#NULL!	7.40	12.60	133,000.00	#NULL!	#NULL!	#NULL!	1.00	2.00	1.00	2.00	55.00	93,876.00	2	10.00
0	3.00	#NULL!	#NULL!	#NULL!	7.00	9.40	197,000.00	#NULL!	#NULL!	#NULL!	1.00	2.00	2.00	1.00	67.00	92,174.00	2	11.00
0	2.00	#NULL!	#NULL!	#NULL!	8.70	8.50	119,000.00	#NULL!	#NULL!	#NULL!	1.00	1.00	1.00	2.00	53.00	90,236.00	2	12.00
0	3.00	#NULL!	#NULL!	#NULL!	7.70	10.50	115,000.00	#NULL!	#NULL!	#NULL!	2.00	1.00	2.00	1.00	61.00	94,781.00	2	13.00
0	2.00	#NULL!	#NULL!	#NULL!	7.40	9.40	114,000.00	#NULL!	#NULL!	#NULL!	2.00	2.00	1.00	2.00	44.00	92,678.00	2	14.00
0	1.00	#NULL!	#NULL!	#NULL!	7.20	8.80	104,000.00	#NULL!	#NULL!	#NULL!	2.00	2.00	2.00	1.00	32.00	99,236.00	2	15.00
0	2.00	#NULL!	#NULL!	#NULL!	9.50	10.70	119,000.00	#NULL!	#NULL!	#NULL!	1.00	2.00	1.00	1.00	58.00	91,873.00	2	16.00
0	1.00	#NULL!	#NULL!	#NULL!	9.30	12.00	110,000.00	#NULL!	#NULL!	#NULL!	2.00	2.00	2.00	1.00	28.00	93,821.00	2	17.00
0	2.00	#NULL!	#NULL!	#NULL!	8.30	13.70	107,000.00	#NULL!	#NULL!	#NULL!	1.00	2.00	1.00	1.00	47.00	92,745.00	2	18.00
0	2.00	#NULL!	#NULL!	#NULL!	9.30	12.20	113,000.00	#NULL!	#NULL!	#NULL!	1.00	2.00	1.00	2.00	52.00	91,632.00	2	19.00
0	2.00	#NULL!	#NULL!	#NULL!	7.00	8.50	149,000.00	#NULL!	#NULL!	#NULL!	2.00	2.00	2.00	1.00	60.00	93,641.00	2	20.00
0	2.00	#NULL!	#NULL!	#NULL!	7.30	9.40	137,000.00	#NULL!	#NULL!	#NULL!	1.00	2.00	1.00	1.00	45.00	93,452.00	2	21.00
0	1.00	#NULL!	#NULL!	#NULL!	7.80	8.80	133,000.00	#NULL!	#NULL!	#NULL!	2.00	2.00	1.00	1.00	39.00	95,673.00	2	22.00
0	2.00	#NULL!	#NULL!	#NULL!	9.00	8.20	122,000.00	#NULL!	#NULL!	#NULL!	1.00	1.00	2.00	2.00	53.00	98,256.00	2	23.00
0	2.00	#NULL!	#NULL!	#NULL!	8.90	12.00	157,000.00	#NULL!	#NULL!	#NULL!	1.00	2.00	2.00	2.00	46.00	90,234.00	2	24.00
0	2.00	#NULL!	#NULL!	#NULL!	10.60	11.60	190,000.00	#NULL!	#NULL!	#NULL!	1.00	1.00	1.00	1.00	51.00	91,456.00	2	25.00
0	2.00	#NULL!	#NULL!	#NULL!	10.00	10.50	106,000.00	#NULL!	#NULL!	#NULL!	1.00	2.00	2.00	2.00	56.00	92,783.00	2	26.00
0	3.00	#NULL!	#NULL!	#NULL!	7.00	11.40	115,000.00	#NULL!	#NULL!	#NULL!	1.00	2.00	2.00	2.00	73.00	90,567.00	2	27.00
0	1.00	#NULL!	#NULL!	#NULL!	9.40	12.00	104,000.00	#NULL!	#NULL!	#NULL!	1.00	1.00	1.00	1.00	37.00	93,567.00	2	28.00
0	2.00	#NULL!	#NULL!	#NULL!	8.50	11.00	104,000.00	#NULL!	#NULL!	#NULL!	1.00	1.00	1.00	1.00	45.00	90,327.00	2	29.00
0	2.00	#NULL!	#NULL!	#NULL!	8.90	12.00	149,000.00	#NULL!	#NULL!	#NULL!	2.00	2.00	2.00	2.00	47.00	96,729.00	2	30.00
0	2.00	#NULL!	#NULL!	#NULL!	9.00	11.40	119,000.00	#NULL!	#NULL!	#NULL!	1.00	2.00	1.00	1.00	55.00	98,456.00	2	31.00
0	2.00	#NULL!	#NULL!	#NULL!	7.30	8.40	150,000.00	#NULL!	#NULL!	#NULL!	1.00	2.00	2.00	2.00	60.00	93,786.00	2	32.00
0	1.00	#NULL!	#NULL!	#NULL!	7.70	11.70	159,000.00	#NULL!	#NULL!	#NULL!	1.00	2.00	2.00	1.00	38.00	90,267.00	2	33.00
0	2.00	#NULL!	#NULL!	#NULL!	8.40	11.00	134,000.00	#NULL!	#NULL!	#NULL!	1.00	2.00	1.00	1.00	43.00	91,067.00	2	34.00
0	2.00	#NULL!	#NULL!	#NULL!	7.90	10.40	167,000.00	#NULL!	#NULL!	#NULL!	2.00	2.00	1.00	1.00	55.00	92,430.00	2	35.00

ABBREVIATIONS IN MASTER CHART

SEX   1-Male,   2-Female

SYS.H.T   -   systemic Hypertension

DIA.MELL   -   Diabetes mellitus

ON.ASP   -   On aspirin

PLAT.COUNT   -   platelet count

MPV   -   Mean platelet count

PDW   -   platelet distribution Width

EJEC.FRA   -   Ejection Fraction

For these parameters 1   -   YES,2-NO