GENETIC STUDY OF CHEMOKINE RECEPTOR GENE (CCR5) POLYMORPHISM IN ACUTE CORONARY SYNDROME IN VIJAYAPURA POPULATIONBY

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LIST OF ABBREVIATIONS

ACS : ACUTE CORONARY SYNDROME

CCL2 : CHEMOKINE LIGAND 2

CCL3 : CHEMOKINE LIGAND 3

CCL4 : CHEMOKINE LIGAND 4

CCL5 : CHEMOKINE LIGAND 5

CCR2 : CHEMOKINE RECEPTOR 2

CCR5 : CHEMOKINE RECEPTOR 5

CS : CARDIOGENIC SHOCK

ECG : ELECTROCARDIOGRAPHY

HF : HEART FAILURE

LA : LEFT ATRIUM

LAD : LEFT ANTERIOR DESCENDING ARTERY

LCX : LEFT CIRCUMFLEX ARTERY

LV : LEFT VENTRICLE

LVEF : LEFT VENTRICULAR EJECTION FRACTION

MI : MYOCARDIAL INFARCTION

MACE : MAJOR ADVERSE CARDIAC EVENTS

NSTEMI : NON-ST ELEVATION MYOCARDIAL INFARCTION

PE : PULMONARY EDEMA

RCA : RIGHT CORONARY ARTERY

RWMA : REGIONAL WALL MOTION ABNORMALITY

STEMI : ST ELEVATION MYOCARDIAL INFARCTION

VPC : VENTRICULAR PREMATURE COMPLEXES

VT : VENTRICULAR TACHYCARDIA

UA : UNSTABLE ANGINA

ABSTRACT

BACKGROUND:

Acute coronary syndrome is one of the leading causes of morbidity and death in underdeveloped nations. Chemokine's and its receptor play crucial role in initiation and progression of atherosclerosis. Chemokine receptor 5 (CCR5) is an important mediator of leucocyte recruitment and leukopedesis. The study of CCR5 polymorphism and its role as genetic risk factor in acute coronary syndrome provides significant evidence for the therapeutic use of drug Maraviroc (anti-CCR5) in coronary artery disease patients.

AIMS AND OBJECTIVE:

To study genetic polymorphism of chemokine receptor (CCR5) genes associated with patient of acute coronary syndrome in Vijayapura population.

MATERIALS AND METHODS:

A prospective cross-sectional study was conducted in Shri B M Patil Medical College Hospital and Research Centre,Vijayapurain patients admitted for acute coronary syndrome. Clinical history and examination, electrocardiographic, laboratory profile and blood samples taken for analysis of CCR5 gene polymorphism as a part of work up. After collecting the blood samples, it was processed for DNA extraction, designing primer, PCR and gene sequencing was performed to look for CCR5 polymorphism. Patients were grouped according to presence of CCR5 polymorphism as group A (n=6), and group B (N=74) with absence of polymorphism. Patient's clinical profile, blood investigations and 2D-ECHO between the two groups were studied and analysed.

RESULTS: Total of 100 patients were admitted with acute coronary syndrome. Six patients with diabetes mellitus were excluded from the study based on exclusion criteria. Out of 94

patient's, 13 patient gene sequencing could not be conducted due to financial problem and remaining 81 patient's gene sequencing was analysed for CCR5 polymorphism and classified as group A and group B.The most common risk factors in group A, were smoking and tobacco chewing.On sequencing 6 patients had CCR5 gene polymorphism out of 81 with an incidence of 7.5%($p<0.001$). Out of 6 positive patients in group Ahad 3 males and 3 females ,1 patient of age 45 year and remaining 5 above 60 years.

CONCLUSION: Our study on the role of CCR5 polymorphism in acute coronary syndrome shows positive association between polymorphism and disease. The study shows that our population is genetically susceptible for acute coronary syndrome and CCR5 polymorphism could be considered as one of the etiologies for acute coronary syndrome.

By screening for CCR5 polymorphism in high-risk individuals, we can provide a better and effective early intervention to the individuals and thereby reduce the social burden, morbidity and mortality of disease.

KEYWORDS: Acute coronary syndrome, chemokine receptor 5(CCR5), Diabetes mellitus, Polymorphism.

INTRODUCTION

I. INTRODUCTION

Acute coronary syndrome is a multifactorial disease with complex pathogenesis, mainly a result of the interplay of genetic and environmental risk factors. The regulation of thrombosis, inflammation, and cholesterol, and lipid metabolism are the main factors, but there is a lack of study for the identification of novel genetic markers. As per the Global Burden of Disease study, estimated that 24.8% of all deaths in India are imputable to cardiovascular disease. According to this study, the age-standardized CVD death rate in India is 272 per 100000 people which is greater than the global death rate of 235 per 100000 people.[1]

Many predisposing risk factors have been proven for ACS, which are non-modifiable risk factors like age, sex, ethnicity, family history, genetic factors, and modifiable factors such as hypertension, diabetes mellitus, smoking/tobacco use, obesity, and diet.[2]

Leukocytes create soluble proteins called chemokine's, which act by binding to the G-protein-coupled receptor known as the chemokine receptor. The gene for CCR5, which is mostly found in endothelium and immune cells and is thought to be the unique surface marker for Th1 cells, is found on chromosome 3P21.3.[3] Atherosclerosis is a chronic inflammatory condition that worsens with time and is characterised by lipid build-up in the intima of blood vessel walls, endothelial dysfunction, and vascular inflammation.4 Leukocytes from the central circulation are drawn to the site of injury by damaged endothelial cells. Foam cells made of lipid are created when leukocytes chemotactically enter the walls of endothelial cells. These reactions are advantageous because they may serve as a defence against cancer and infection, but they can cause arterial plaque to form when leukocytes and endothelium interact.[5]

Hence chemokine's play an important role in the pathogenesis of atherosclerosis which is a risk factor for coronary heart disease. By detecting CCR5 delta 32 polymorphisms, we can establish the role of CCR5 in acute coronary syndrome.[6, 7]

Regarding cardiovascular risk, CCR5 delta 32 base pair deletion has either been linked to the development of atherosclerosis and the start of myocardial infarction, or no link between them has been discovered. As a result, there is contradictory information about the role of the CCR5 delta35 deletion variation in the development of coronary atherosclerosis.[8]

If the polymorphism is not detected early and treated, it can lead to sudden cardiac arrest, which is one of the top avoidable causes of death.

AIMS AND OBJECTIVES

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To study genetic polymorphism of chemokine receptor (CCR5) genes associated with patient of acute coronary syndrome in Vijayapura population.

REVIEW OF LITERATURE

III. REVIEW OF LITERATURE

Acute coronary syndrome is a prominent cause of death and morbidity in developing nations such as India. By evaluating the expression of many genes that are consistently connected with the occurrence of acute coronary syndrome and incorporating these genes as a risk factor for cardiovascular disease. The potential of gene study to identify a percentage of illness risk would hopefully allow us to intervene sooner and treat better, but ultimately to prevent acute coronary syndrome and its complications.

P González et al. investigated genetic variation at the chemokine receptors CCR5/CCR2 in myocardial infarction in 2001. The study found that individuals with the ccr5 allele were less likely to have an early MI. CCR5 and CCR5-ligands are expressed by cells in the arteriosclerotic plaque, which reduces inflammation and slows the course of the arteriosclerotic lesion in ccr5-carriers. They proposed that pharmaceutical CCR5 blockage might be the future of MI treatment. 9

Eleonora Simeoni et al investigated the Association of RANTES G-403A gene polymorphism with higher risk of coronary arteriosclerosis in 2003. The study found a substantial number of polymorphisms in patients when compared to controls. RANTES A-403 may enhance genetic vulnerability to CAD, and RANTES antagonists have been studied effectively in heart transplantation and HIV models of cardiovascular disease prevention. 8

S.Sharda et al examined CCR5 deletion polymorphism in North Indian patients with coronary artery disease in 2006 and discovered that CCR5 delta 32 heterozygotes genotype frequency was three times higher in patients with CAD than in normal persons. This demonstrates a favourable relationship between CAD and CCR5 polymorphism. 5

Jennifer K Pai et colleagues. investigated polymorphisms in the CC-chemokine receptor-2 (CCR2) and -5 (CCR5) genes and the risk of coronary heart disease in US women in 2006. The study concluded that the distribution of alleles was comparable across patients and controls. CCR2-CCR5 haplotypes were not related with CHD risk in this cohort, indicating a significant negative relationship between CCR5 variations and early age of CHD onset. 10

Ali R. Afzal et al. investigated the common CCR5-del32 frameshift mutation and its association with blood levels of inflammatory markers and cardiovascular disease risk in the Bruneck community in 2008. The mutation was linked to considerably lower C-reactive protein levels, decreased carotid intima-media thickness in the common carotid artery, and a lower risk of cardiovascular disease. These findings indicate that CCR5 mutations protect against atherosclerosis and cardiovascular disease in humans. 11

In 2010, Craig L. Hyde, et al studied Genetic Association of the CCR5 Region with Lipid Levels in At-Risk Cardiovascular Patients. Their results demonstrate an association between the CCR5Δ32 deletion and increased plasma high-density lipoprotein cholesterol and decreased plasma triglycerides, protective for cardiovasculardisease.[12]

In 2011, Neha Singh et al studied polymorphism in chemokine receptor genes and risk of acute myocardial infarction in the north Indian population and concluded that CCR5 delta 32 polymorphisms were significantly four times higher in acute myocardial infarction.[6]

In2012, Amani Kallel et al studied polymorphisms in the CC-chemokine receptor-2 (CCR2) and -5 (CCR5) genes and risk of myocardial infarction among Tunisian male patients. Haplotype analysis showed that MI patients had significantly less 64Val-Del haplotype and 64Ile-Ins haplotype. A protective effect of the CCR5-Δ32 polymorphism against MI in the Tunisian population was found.[13]

In 2014, Janet J. Maguire et al studied CCR5 chemokine receptor mediates vasoconstriction and stimulates intimal hyperplasia in human vessels in vitro. Data support a potential role for CCR5 in vasoconstriction and neointimal formation in vitro, vascular remodelling and augmented vascular tone in human coronary artery and vein graft disease. Hence providing therapeutic potential use of maraviroc for cardiovascular disease.[14]

Ke-Hsin Ting et al. investigated the relationship of genetic polymorphisms in the chemokine CCL5 and its receptor CCR5 with coronary artery disease in Taiwan in 2015. Finally, they discovered that the CCL5-403 polymorphism may enhance genetic vulnerability to CAD. 15

In 2015, Zhongwen Zhang et al studied association between chemokine receptor 5 (CCR5) delta32 gene variant and atherosclerosis: a meta-analysis of 13 studies. Analysis concluded that CCR5-delta32 (Δ32) genetic variants was not associated with increased risk of atherosclerotic disease, but CCR5 Δ32-positive genotype increases the risk of atherosclerotic disease in Asian population. This shows ethnicity as potent risk factor for atherosclerosis.[16]

In 2016, Jessica R. Golbus et al studied Common and Rare Genetic Variation in CCR2, CCR5, or CX3CR1 and Risk of Atherosclerotic Coronary Heart Disease and Glucometabolic Traits. Concluded that no chemokine receptor variant was associated with CAD, MI, or glucometabolic traits in large European ancestry cohorts but South Asian cohort, identified single nucleotide polymorphism associations with MI and type II diabetes mellitus.[17]

In 2018, Angelica Martins Batista et al studied Genetic Polymorphism at CCL5 Is Associated with Protection in Chagas' Heart Disease: Antagonistic Participation of CCR1+ and CCR5+ Cells in Chronic Chagas Cardiomyopathy.CCR5-deficient infected mice presented reduced TNF concentrations and injury in heart tissue and selective blockade of CCR1 (Met-RANTES therapy) in infected Ccr5−/− mice showed protective role for CCR1 in CCC. This provides CCL5-CCR1 axis as a therapeutic target for immunostimulation.[18]

ACUTE MYOCARDIAL INFARCTION

INTRODUCTION

Acute coronary syndrome (ACS) includes ST-elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI), and unstable angina. Acute coronary syndrome accounts for one-third of all mortality in people over the age of 35.

The World Health Organization (WHO) estimates that the global number of CAD deaths would rise from 7.2 million in 2002 to 11.2 million by 2020. According to the SCORE system or Framingham Heart Study database, males have a 49% chance of developing symptomatic CAD after the age of 40, while women have a 32% chance. 19

ACS is a manifestation of CHD and is usually a result of plaque disruption in coronary arteries (atherosclerosis). The common risk factors for the disease are smoking, hypertension, diabetes, hyperlipidaemia, male sex, physical inactivity, family obesity, and poor nutritional practices. Cocaine abuse can also lead to vasospasm.[20]A family history of early myocardial infarction (55years) is also a high-risk factor.

Acute myocardial infarction is broadly termed as cardiomyocyte death secondary to prolonged ischemia resulting from a sudden imbalance between oxygen supply and demand. Though multiple risk factors play a pivotal role in the progression of the disease, but ultimate pathogenesis leads to atheroma formation and vascular occlusion.[21]

DEFINITION

According to the Fourth Universal Definition of Myocardial Infarction (2018), the term acute myocardial infarction is defined as acute myocardial injury with clinical evidence of acute myocardial ischemia and with detection of a rise and/or fall of cTn values with at least one value above the 99th percentile upper reference limit and at least one of the following:

- Symptoms of myocardial ischemia.
- New ischemic ECG changes.
- Development of pathological Q waves.
- Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischemic aetiology.
- Identification of a coronary thrombus by angiography or autopsy (not for type 2 or 3 MIs).

Post-mortem demonstration of acute athero-thrombosis in the artery supplying the infarcted myocardium meets the criteria for type 1 MI. Evidence of an imbalance between myocardial oxygen supply and demand unrelated to acute athero-thrombosis meets the criteria for type 2 MI. Cardiac death in patients with symptoms suggestive of myocardial ischemia and presumed new ischemic ECG changes before cTn values become available or abnormally meet the criteria for type 3 MI. [22]

CLASSIFICATION

A. ANATOMICAL CLASSIFICATION:

- Transmural infarction: involves all three layers of the heart, namely the endocardium, myocardium and epicardium.

- Subendocardial infarction: involvement of small area in the subendocardial wall of the left ventricle, ventricular septum or papillary muscles.

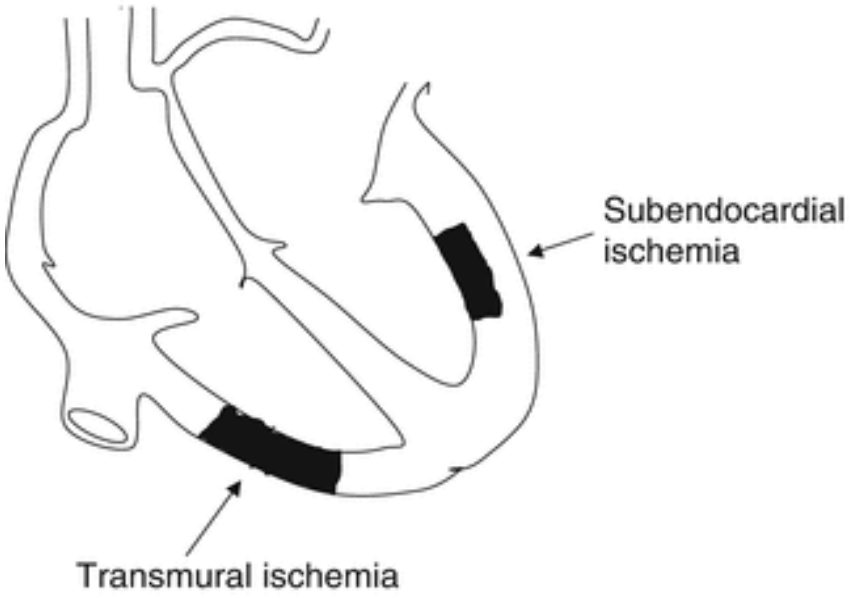


Figure 1: Anatomical classification of myocardial infarction.

B. CLINICAL CLASSIFICATION[22]:

i) TYPE 1: Spontaneous MI due to coronary thrombosis

ii) TYPE 2: Supply/demand mismatch by a secondary process other than coronary artery disease.

iii) TYPE 3: Suspected MI-related death

iv) TYPE 4a: Percutaneous coronary intervention-related death

TYPE 4b: Stent thrombosis

TYPE 4c: Restenosis associated with percutaneous coronary intervention.

v) TYPE 5: Coronary artery bypass grafting- related MI.

C. ELECTROCARDIOGRAPHIC CLASSIFICATION:

i) ST-segment elevation myocardial infarction.

ii) Non-ST-segment elevation myocardial infarction.

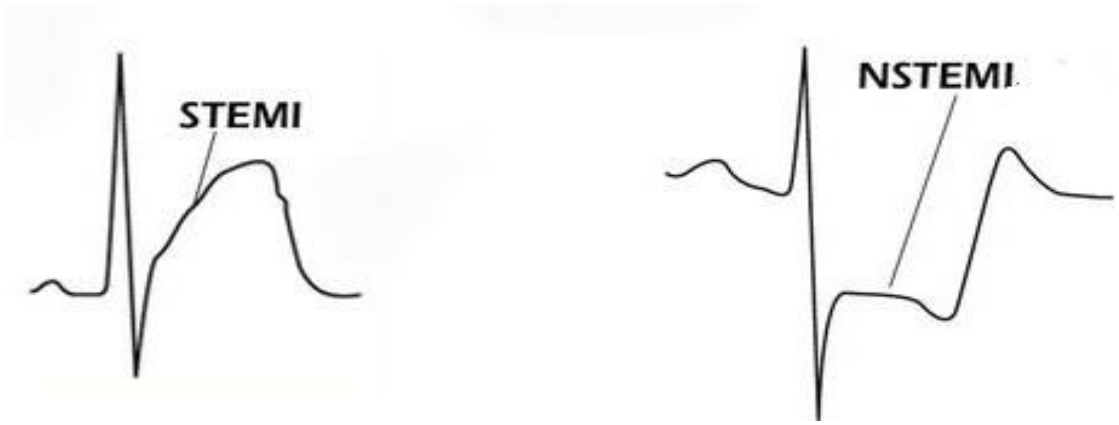


Figure 2: Electrocardiographic classification of myocardial infarction.

Diagnosis of Acute Coronary Syndrome:

The first step in evaluating ACS is an ECG, which helps differentiate between STEMI NSTEMI and unstable angina. American Heart Association guidelines states that any patient with complaints suspicious of ACS should get an ECG within 10 minutes of arrival. Cardiac enzymes, especially troponin, CPK MB is critical in assessing the STEMI and NSTEMI versus unstable angina. A chest x-ray helps diagnose causes, other than MI presenting with chest pain like pneumonia and pneumothorax. Aortic dissection and pulmonary emboli should be kept in differential and investigated when the situation warrants.[23]

ECG CRITERIA FOR DIAGNOSIS OF MYOCARDIAL INFARCTION[22]:

A. IN THE ABSENCE OF LEFT VENTRICULAR HYPERTROPHY AND BUNDLE BRANCH BLOCK:

i) ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION:

New ST-elevation at the J-point in 2 contiguous leads with the cut-point: ≥1 mm in all leads other than leads V2–V3 where the following cut-points apply: ≥2 mm in men ≥40 years; ≥2.5 mm in men <40 years, or ≥1.5 mm in women regardless of age .

When the magnitudes of J-point elevation in leads V2 and V3 are registered from a prior electrocardiogram, new J-point elevation ≥1 mm (as compared with the earlier electrocardiogram) should be considered an ischemic response.

ii) NON-ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION:

New horizontal or down-sloping ST-depression ≥0.5 mm in 2 contiguous leads and/orT inversion >1 mm in 2 contiguous leads with prominent R wave or R/S ratio >1 .

iii) CHANGES ASSOCIATED WITH PRIOR MYOCARDIAL INFARCTION:

Any Q wave in leads V2–V3 >0.02 s or QS complex in leads V2–V3. Q wave ≥0.03 s and ≥1 mm deep or QS complex in leads I, II, aVL, aVF or V4–V6 in any 2 leads of a contiguous lead grouping (I, aVL; V1–V6; II, III, aVF). R wave >0.04 s in V1–V2 and R/S >1 with a concordant positive T wave in the absence of conduction defect [22].

B. IN THE PRESENCE OF LEFT BUNDLE BRANCH BLOCK (LBBB) OR VENTRICULAR PACED RHYTHM, DIAGNOSIS IS BASED ON MODIFIED SGARBOSSA CRITERIA:

- ≥ 1 lead with ≥1 mm of concordant ST elevation
- ≥ 1 lead of V1-V3 with ≥ 1 mm of concordant ST depression
- ≥ 1 lead anywhere with ≥ 1 mm ST elevation and proportionally excessive discordant ST elevation, as defined by ≥ 25% of the depth of the preceding S-wave[24].

ST elevation is measured at the J-point and should be present in at least two contiguous leads. Assess right-sided leads (V3R and V4R) in inferior myocardial infarction and assess posterior leads (V7-V9) in suspected posterior myocardial infarction (ST depressions in V1-V3).

LOCALIZATION OF MYOCARDIAL INFARCTION ON ELECTROCARDIOGRAPHY:

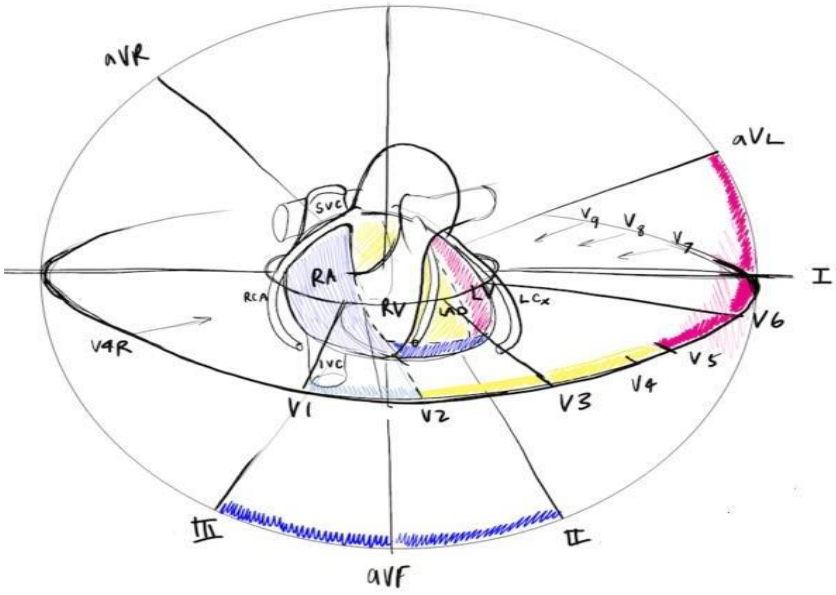


Figure 3: Localization of myocardial infarction on electrocardiograph.

SITE	ARTERY	ECG LEADS
Anterior	LAD	V3, V4
Anterolateral	LAD	V5, V6
Anteroseptal	LAD	V1, V2
Inferior	RCA	II, III, aVF
Posterior	RCA, LCX	V7-V9

Table1: Electrocardiographic localization of myocardial infarction and coronary artery territories.

CLINICAL PRESENTATION:

History and Physical Examination Findings:

UA discomfort is more acute, occurs at rest, and is commonly described as outright pain. Angina is generally described by patients as deep, poorly localised chest or arm pain that is exacerbated by exertion or mental stress and relieved by rest, nitroglycerin, or both. The soreness or pressure, which is most often felt in the substernal area (or, on rare occasions, the epigastric area), typically travels to the neck, chin, left shoulder, and left arm. In addition to chest discomfort, some patients may feel "anginal equivalents," which include dyspnea (the most common), nausea and vomiting, diaphoresis, and unexplained tiredness.[25]Atypical presentations are more likely in women and the elderly .

PATHOPHYSIOLOGY OF ACS:

(A) Initiation of Atherosclerosis: Role of the Endothelium:

Throughout a person's lifetime, atherosclerosis persistently evolves into an acute ischemic event. It is the continuous activity of plaque development that mostly affects the intima of large and medium-sized arteries. This process is influenced by a number of risk factors, such as smoking, diabetes, hypertension, hypercholesterolemia, and obesity.[26]These risk factors harm the blood vessel's endothelium and cause endothelial dysfunction, which is a critical first step in the development of atherosclerosis. Increased expression of adhesion molecules (such as selectins, vascular cell adhesion molecules, and intercellular adhesion molecules), decreased bioavailability of nitric oxide, excessive production of endothelin 1, which impairs vascular haemostasis, and increased thrombogenicity of blood are all signs of an endothelium that is dysfunctional .[27]

(B) Progression of Atherosclerotic Plaque: Role of Inflammation:

Inflammatory cells, in particular monocytes, move into the sub-endothelium after the endothelium has been injured by adhering to endothelial adhesion molecules. Once there, they undergo differentiation to become macrophages. Low-density lipoprotein (LDL) that has oxidised and penetrated the artery wall is digested by macrophages, resulting in foam cells and the development of fatty streaks. In order to continue the process, the activated macrophages release chemo-attractants and cytokines (such as monocyte chemoattractant protein 1, tumour necrosis factor, and interleukins), which draw more macrophages and vascular smooth muscle cells (which produce extracellular matrix components) to the plaque site. Additionally, macrophages produce matrix metalloproteinases, which break down the extracellular matrix and cause plaque disintegration.[28]The degree of plaque susceptibility and rupture depends critically on the proportion of smooth muscle cells to macrophages. ACS is more frequently caused by plaque rupture and total blockage of the artery, despite the fact that it is clinically silent. Atherosclerotic lesions develop at a varied and unexpected rate.[29]

(C) Stability of Plaques and Tendency for Rupture:

Atherosclerotic plaques have varying degrees of stability. Large lipid cores, thin fibrous caps, a high density of macrophages and T lymphocytes, a relative paucity of smooth muscle cells, locally increased expression of matrix metalloproteinases that degrade collagen, eccentric outward remodelling, increases in plaque neovascularity, and intraplaque haemorrhage are all characteristics of so-called high-risk or vulnerable plaques.[30] Even within the same individual, human atherosclerotic plaques have a surprisingly diverse nature. The "vulnerability" of plaques is significantly influenced by inflammation, which is associated with an increase in the activity of macrophages at the plaque site that are capable of destroying extracellular matrix, secreting proteolytic enzymes like plasminogen activators,

and thinned plaque caps. These features make the plaque more prone to rupture.[31]The frequency of plaque ruptures has been observed to positively correlate with elevated levels of C-reactive protein (CRP), which may indicate the activity of these macrophages .[32]

(D) Plaque Disruption, Thrombosis, and ACS

Inflammatory cells, thrombogenicity, and endothelial dysfunction interact synergistically throughout the pathogenesis of ACS.[33]Angiographically, noncritical coronary lesions (<50% stenosis in the artery diameter) may be associated with sudden progression to severe or complete occlusion and subsequently is responsible for up to two-thirds of ACS cases.[34] Controlling the level of thrombus formation and determining whether a specific plaque rupture will result in ACS require consideration of factors including the lipid and tissue factor content of the plaque, the severity of the plaque rupture, the degree of inflammation at the site, the blood flow in the area, and the patient's antithrombotic and prothrombotic balance. PlA2 polymorphism of glycoprotein IIIa and the occurrence of acute coronary thrombosis is significantly higher prevalence in subjects with at least one PlA2 allele in myocardial infarction or unstableangina.[35]Studies using intravascular ultrasonography have shown that at least 80% of patients with ACS exhibit multiple plaques rupture distinct from the culprit lesion .[36]

According to autopsy studies, plaque rupture is responsible for around 75% of fatal MIs, with superficial endothelium degradation accounting for the remaining 25%.[37]Thesubendothelial matrix, which is rich in tissue factor, a potent procoagulant, is exposed to the circulating blood following either plaque rupture or endothelial erosion. This exposure causes platelet adhesion, which is followed by platelet activation and aggregation, and the formation of a thrombus. There are two types of thrombi that can develop: one with a fibrin-rich clot (also known as a red clot) that results from an activated coagulation cascade and decreased flow in

the artery, and the other with a platelet-rich clot (also known as a white clot) that forms in areas of high shear stress and completely occludes the artery. Total blockage results from the red clot commonly superimposing over white clots. The major role of thrombosis in the pathophysiology of ACS is backed by a number of evidence-based studies.[38]

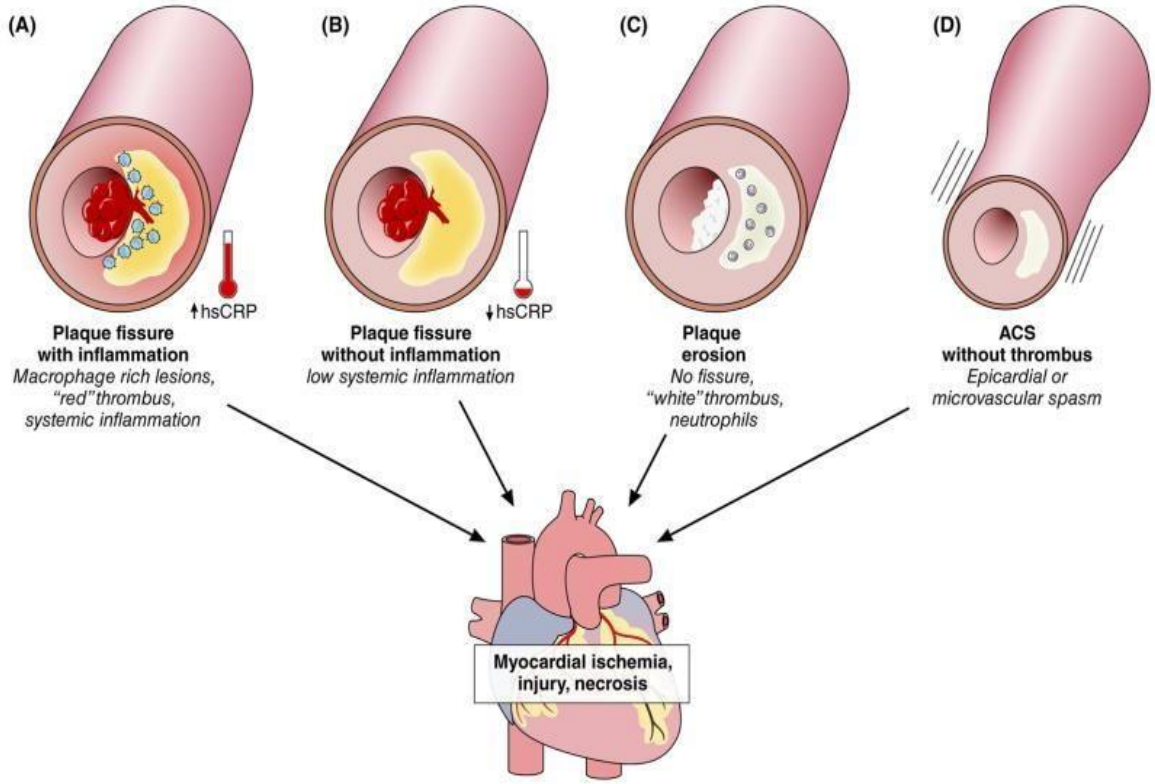


Figure 4: Different pathology in blood vessels leading to myocardial injury.

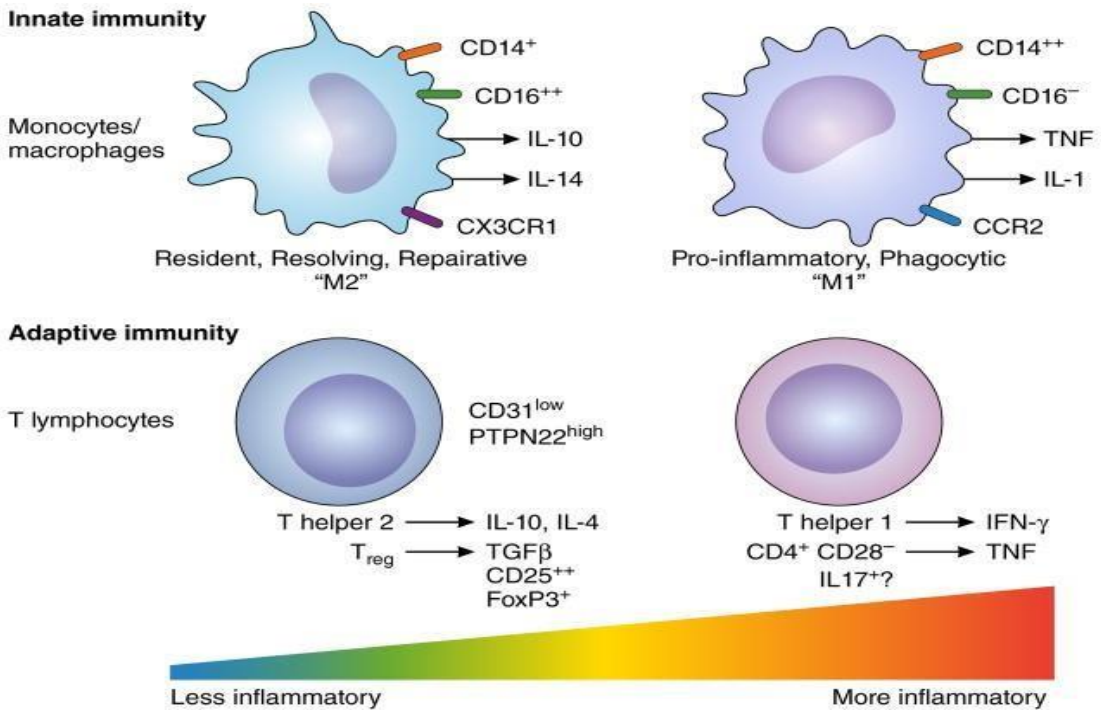


Figure 5: Imbalance in adaptive immune pathways and degree of inflammation.

Treatment / Management:

The initial therapies for all ACS include aspirin (325 mg), heparin bolus, or intravenous (IV) heparin infusion if there are no contraindications. For antiplatelet therapy, ticagrelor or clopidogrel are recommended. Ticagrelor is not given to patients who are receiving thrombolysis. Dual antiplatelet treatment is preferred over single antiplatelet therapy due to the CYP2C19 mutation.[39]When required, supporting measures are administered, such as pain relief with morphine or fentanyl and oxygen in the case of hypoxia. Nitroglycerin can be administered intravenously or sublingually to patients to ease their discomfort. Nitro-glycerine must be supplied exceedingly cautiously since it might cause severe hypotension in inferior wall ischemia.Arrhythmias must be regularly watched for in the heart. The next step in therapy depends on whether an ACS is a STEMI, NSTEMI, or unstable angina. The American Heart Association recommends an immediate catheterization and percutaneous intervention (PCI) with a door-to-procedure duration of fewer than 90 minutes for STEMI

(AHA). Tenecteplase or another thrombolytic is indicated if there is no PCI available and the patient cannot be brought to the catheterization lab in within 120 minutes. According to AHA recommendations, the door-to-needle (TNK/other thrombolytics) time must be less than 30 minutes.In addition to the initial aspirin and heparin treatment for NSTEMI/Unstable Angina, controlling symptoms. If the patient continues to feel discomfort, an urgent catheterization is indicated. The scheduling of catheterization and other assessment procedures, such as a cardiac perfusion scan, can be determined on a case-by-case basis based on comorbidities if symptoms are well controlled. For ACS, admission and an immediate cardiac assessment are always necessary. Computerized tomography angiography may also be utilised for extra workup, depending on availability and the cardiologist's preference .

Beta-blockers, statins, and ACE inhibitors should all be begun as soon as practical in instances of ACS if there are no contraindications. Cases that are not amenable to PCI are either taken for CABG (coronary artery bypass graft) or managed medically, depending on the comorbidities and the patient's desire.

Differential Diagnosis:

- Acute pericarditis
- Aortic stenosis
- Asthma
- Dilated cardiomyopathy
- Esophagitis
- Myocardial infarction
- Myocarditis

COMPLICATIONS OF ACUTE MYOCARDIAL INFARCTION

Most of the deaths in these patients are the direct result of pathophysiologic changes which occur as a result of the AMI. Many more patients suffer from complications of AMI. These patients require prompt and early recognition of these condition and aggressive management in order to prevent unnecessary morbidity and mortality. Complications of AMI can be broadly classified into:

A. Heart failure and Cardiogenic shock.

B. Ischemic Complication

i. Reinfarction

C. Mechanical Complications

i. Left ventricular aneurysm

ii. Myocardial rupture

iii. Rupture of the ventricular septum

iv. Pseudoaneurysm

D. Conduction Abnormalities

Post-infarction conduction abnormalities like tachyarrhythmias and bradyarrhythmia's leading to sudden cardiac deaths.

E. Embolic Complications

Stroke

F. Inflammatory complications

Dressler's syndrome and Post myocardial infarction pericarditis, occurs after one week up to several weeks of myocardial infarction, presenting as fever and chest pain.

CHEMOKINES

INTRODUCTION

Chemokine, also called as chemoattractant /chemotactic cytokines which, belongs to a group of small proteins with cysteine residues. Chemokine is produced by cells of the immune system like macrophages, Neutrophils, Mast cells, Eosinophils, dendritic, epithelial cells.[40]

Chemokines after recognizing their receptor, binds to the N terminus part of chemokine and activate the receptor. Ex: In CXCL12 & CCL5 (RANTES), the first N terminal residue is critical for the activation of the receptor and its function. Deletion of this portion of chemokine leads to complete inactivation of the receptor and behaves like an antagonist to that receptor.[40]

The sequence in the N-loop region affects the receptor's selectivity. A mutation in this area can improve the receptor's affinity and activity.

SITE – receptor is situated in the lipid layer of the cell surface and consists of seven transmembrane domains (7TM) belonging to G protein-coupled receptor.

STRUCTURE OF CHEMOKINES[41]

1. Primary.
2. Secondary.
3. Tertiary.
4. Quaternary.

1. Primary structure.

Chemokine's are identified by their primary amino acid sequence and arrangement of four cysteine residues along with mature protein. First, cysteine forms a disulphide bond with the third cysteine whereas 2nd and 4th cysteine forms disulphide bond between them. Chemokine's are sub classified in to 4 groups based on their primary sequence.

A. **Alpha chemokine/cxc** – one amino acid is present between first 2 cysteine's. Ex- IL-8 (CXCL8), PF4 (CXCL4).

B. **Beta chemokines/cc** – first 2 cysteine groups are adjacent to each other. Ex- RANTES (CCL5), MIP-1alpha (CCL3).

C. **Gamma chemokines/cx3c** – one protein (3 amino acids) is present between first two cysteines. Ex- Fractalkine (CX3CL1).

D. **Delta / C chemokine** – one cysteine residue is present out of the first 2 residues. Ex - Lymphotactin.

Even though chemokine were named based their specific function, in 2000 a systematic nomenclature was introduced which includes a subfamily designation by letter L (ligand) and a number according to when the genes was first isolated. Ex CXCL12, CXCL8.

2. Secondary structure

Before the first cysteine, a chemokine has an extended N terminal peptide. All chemokines that are produced have a N terminal that is eliminated by proteolysis when they are directed to the endoplasmic reticulum to increase the ability of the receptor to be activated. About ten residues make up the first two cysteine loops, which are typically followed by one strand of

the 310-helix structure. N loop, which is located between second cysteine and 310 helix, plays a crucial role. The c-terminal alpha helix comes after the 310 helix.

Dynamic investigations using nuclear magnetic resonance (NMR) revealed that the N loop had greater flexibility than other sections. This characteristic is crucial to the mechanism of chemokine receptor activation and binding.

3. Tertiary structure

X-ray crystallography and NMR are used to establish the 3D structures of certain chemokine's. After the N loop, 310 helixes are arranged antiparallel to one another to create a beta pleated sheet. Beta strands are connected to the following strand by flexible type 1 or 3 turns (30s), which are made up of 3 to 4 residues. The type 3 turn (50s) loop connects the third beta strand to the terminal alpha helix. Numerous chemokine's are activated by the 30s loop. Disulphide bonds have a key function in the chemokine structure's stability.

4. Quaternary structure

Chemokine's have a range of oligomeric configurations that are crucial to how they function. Even though the majority of chemokine's are monomers, the various quaternary structures for CXC, CC, and CX3C provide insight into the particular identification of receptors within a subfamily.[40]

When viewed alongside biological molecules like heparin, chemokine's oligomerise, or alter structure, and when examined by NMR or crystallography, they take on a different form.

Consider the compound CX3CL1 (fractalkine), which is monomeric in solution investigations but dimeric in NMR/crystallography crystal studies.The changes in quaternary structure / oligomeric form of chemokine's not only help in receptor binding but also for other functions.

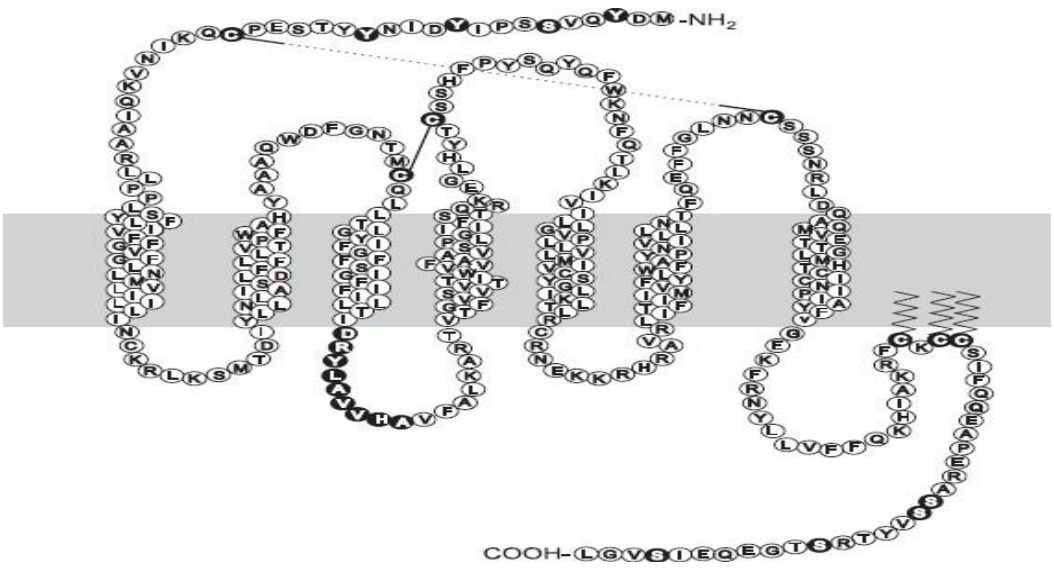


Figure 6: Two-dimensional topology of the human CCR5 sequence. Membrane topology of CCR5 with the extracellular space at the top and the intracellular space at the bottom.[41]

Types of chemokine receptor.[40]

1.Conventional CKR.

2.Atypical CKR.

The naming of the receptor is based on the type of chemokine that binds to it CXC, C, CC OR CX3C, which is followed by the letter R (receptor), and a number depicts its order of discovery.

Chemokine's on binding to receptor, N terminus enters heptahelical bundle of CKR and induces a conformational change that is translated in to intracellular signals leading to its activation.

1. **Conventional CKR:** There are 18 CCKRs named as per chemokine's binding to its receptor. Even though specific chemokine's bind to specific receptors, still receptor specificity

is a complex mechanism to understand. Many chemokine's bind to multiple CCKR, and receptors have many ligands.

2. **Atypical CKR:** Unlike the conventional CKR, atypical CKR do not couple to many chemokine receptors. There are only 4 ACKR known till present. ACKR 1,2,3,4.

Chemokine receptor gene (CCR5) role in cardiovascular disease

It is not surprising that several chemokine's and chemokine receptors have been related to atherosclerosis given the important roles that inflammation and immune cells play in the disease's aetiology. CCL2, CCL5, CX3CL1, and their receptors CCR2, CCR5, and CX3CR1 have received a lot of research since they appear to have significant yet different roles. Although it is possible that numerous chemokine family members have a role in atherogenesis, CCR5 stands out due to the existence of an authorised antagonist called MARAVIROC. The naturally occurring CCR5delta32 variant makes it possible to assess how the human CCR5 gene's knockdown affects various disorders, which is unusual for a G-protein coupled receptor.

Genetic epidemiology has used this opportunity to investigate links between CCR5 and human cardiovascular illness, albeit the results of this research have not yet yielded a clear-cut picture. The CCR5delta32 allele has been associated with decreased risk of myocardial infarction, decreased early onset of coronary heart disease in women, and decreased susceptibility to coronary artery disease.[42] The CCR5delta32 polymorphism, on the other hand, has not been linked to coronary artery disease or myocardial infarction in other groups, according to other studies. 5 These findings might be a result of fluctuations in the populations under study, such as changes in the frequency of the CCR5delta32 gene. However, these results serve as a catalyst for further research into CCR5's potential contribution to human atherosclerosis. It's also noteworthy that Hyde et al. (2010) discovered a relationship between

the CCR5delta32 polymorphism and higher plasma levels of high-density lipoprotein (HDL) cholesterol and lower plasma levels of triglycerides, both of which are advantageous lipid effects that would be anticipated to lower the risk of cardiovascular disease.[12]

CCR5 in pathogenesis of atherosclerosis:

While CCL2 is required for monocyte adhesion and vascular smooth muscle cell proliferation, and fractalkine acting on CX3CR1 appears to maintain chronic monocyte adhesion and survival within the plaque, CCL5 acting on CCR5 is thought to be required for monocyte recruitment during atherosclerosis development.[43]The in vivo observation that suppressing CCL2, CX3CR1, and CCR5 had synergistic effects in reducing atherosclerosis and that targeting all three systems was required for nearly 100% eradication of illness in an atherosclerotic mouse model confirm these independent functions. The study demonstrated that the aforementioned 3 receptors are responsible for the increased macrophage recruitment in atherosclerotic plaque. Atherosclerosis is facilitated by CCR2 and CXCR3 signalling in the abdominal and aortic roots, respectively. The recruitment of monocytes to the plaques is regulated by CCR5 signalling.[44] Atherosclerosis is highly influenced by monocytes, which have the primary chemokine receptor patterns CCR2+CX3CR1+Ly-6Chi and CCR2-CX3CR1++Ly-6Clo. According to Tacke et al. (2007), plaque infiltration in ApoE/ mice requires CCR2 and CX3CR1 in order for the 'classical' CCR2+Ly-6Chi monocyte subset, the predominant monocyte subset entering forming plaques, to occur. While the entry of "non-classical," or Ly-6Clo, is independent of CCR2 and CX3CR1, it is dependent on CCR5 signalling for the recruitment of T cells into existing plaques and the entry of monocytes into lesions .[45,46]

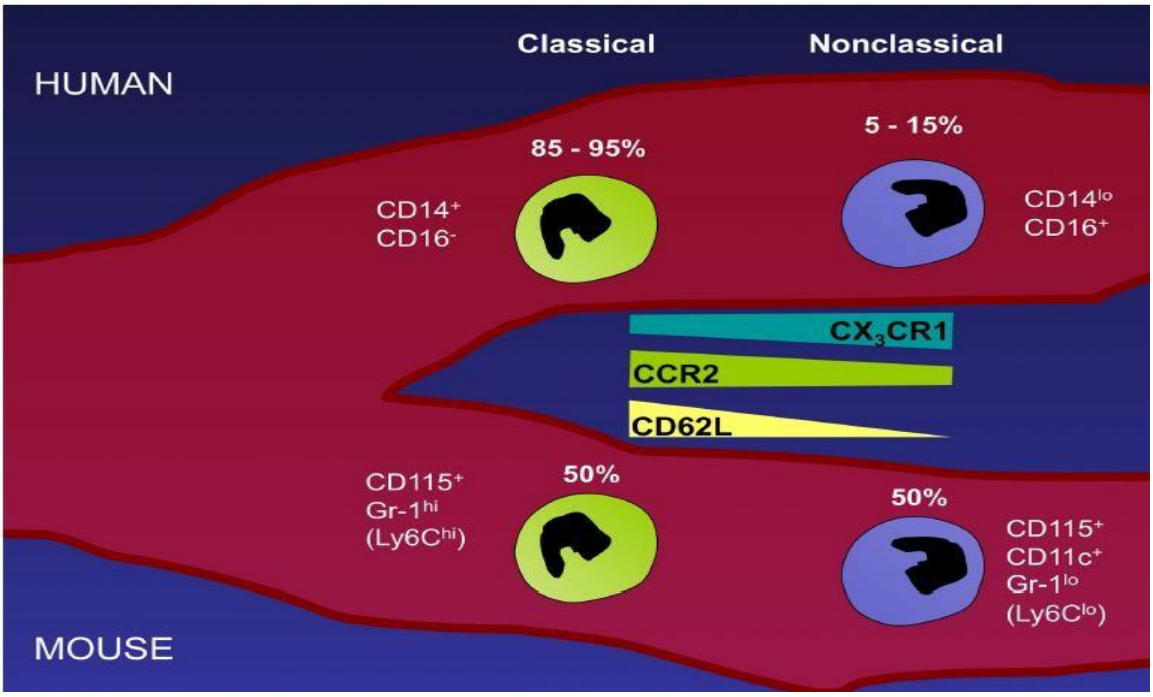


Figure 7: The major markers and frequency (relative %) of the two major monocyte subsets in human (top) and mouse (bottom) blood.

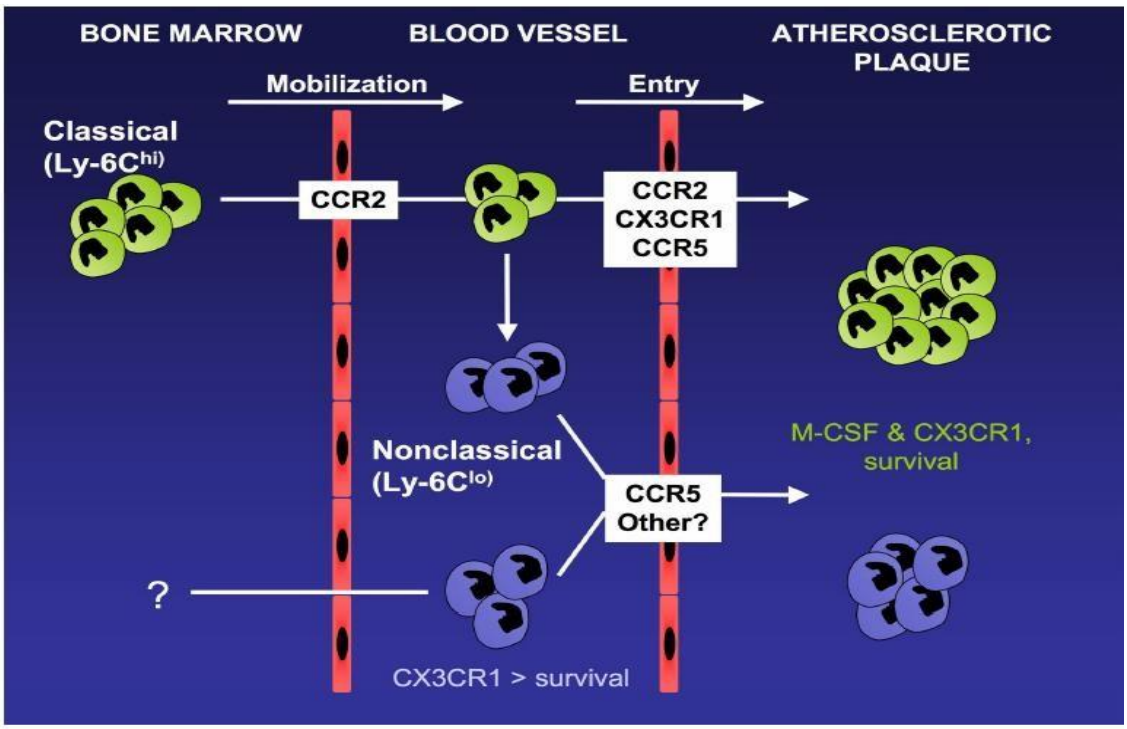


Figure 8: Life cycle of monocyte subsets and their recruitment to atherosclerotic plaques, with an emphasis on the role of chemokine receptors in these processes

High density lipoproteins (HDL) protect vasculature by efflux of cholesterol from cells especially macrophages in arterial wall. It also has antithrombotic, antioxidative and anti-inflammatory property. The chemokine's CCL2 and CCL5 are involved in smooth muscle proliferation (SMC) and neointimal hyperplasia. Recently, it was discovered that reconstituted HDL lowered CCL5 expression in human monocytes and human coronary artery endothelial cells, indicating that CCR5 activity contributes to HDL's atheroprotective effects.[47]

Less is known about the role of CCL3 and CCL4 acting on CCR5 in atherogenesis, however these chemokine's are expected to have a role in atheroma formation and the recruitment of inflammatory cells to plaques. Animal model data, on the other hand, imply that CCL5 is more significant than other CCR5 ligands in the development of atherosclerotic plaques. However, statin therapy lowers the increased synthesis of CCL3 and CCL4 in peripheral blood mononuclear cells reported in people with coronary artery disease.[48] When the CCL3 or CCR5 genes are deleted, MMP-9, a critical enzyme released by macrophages that is present in atherosclerotic plaques and contributes to atherogenesis, is expressed less on macrophages.

Table 2: List of reported CCR5 ligands and their nomenclature.[49]

Nomenclature	**Previous name(s)**
CCL2	MCP-1.
CCL3	MIP-1α.
CCL3L1	MIP-1αP.
CCL4	MIP-1β; LAG-1.
CCL4L1	CCL4L

CCL5	RANTES
CCL7 (antagonist)	MCP-3
CCL8	MCP-2
CCL11	Eotaxin
CCL13	MCP-4
CCL14	HCC-1
CCL16	^{0}HCC-4

MATERIALS AND METHODS

IV. MATERIALS AND METHODS

1.1. SOURCE OF DATA

This study was carried out in the department of General Medicine, BLDE (Deemed to be University) Shri B M Patil Medical College, Hospital and Research Centre, Vijayapura. The study was conducted from January 2020 to June 2022 on 100 patients admitted to our

hospital with acute coronary syndrome. This study was conducted after obtaining approval from the institutional ethical committee. Patients were explained about the procedure in detail and consent was obtained for the same.

1.2. Study Design: Prospective cross-sectional study.

1.3. Study Period: One and half years from January 2020 to June 2022.

1.4. Sample size calculation

Sample size

With anticipated Proportion of CCR5 among Acute MI 5.2 %[6], the study would require a sample size of 81 patients with a 95**%** level of confidence and 10% absolute precision**.**

Formula used

- $\mathbf{n=\frac{z^2\ p*q}{d^2}}$

Where Z= Z statistic at α level of significance

d^2= Absolute error

P= Proportion rate

q= 100-p

1.5. PATIENT SELECTION

A. INCLUSION CRITERIA:

i. Patients admitted with ST segment elevation myocardial infarction.

ii. Patients admitted with NON - ST segment elevation myocardial infarction.

iii. Unstable angina

B. EXCLUSION CRITERIA

i) Patients with Diabetes mellitus.

1.6. INVESTIGATIONS.

Investigations required in this study are standardized procedures. Baseline investigations like, Complete blood count, Blood glucose,Renal function test, Lipid, Serum electrolytes and Urine Examination were done. In addition, cardiac specific investigations like Troponin I, CPK MB, Electrocardiogram, Chest X ray, 2D Echocardiography study were done. Peripheral blood sample of 1ml was collected from patients for analysing CCR5 polymorphism.

METHODOLOGY:

2.1. INITIAL ASSESSEMENT

The study was conducted on patients who were admitted in BLDE (DU), Shri B M Patil Medical College Hospital and Research Centre, Vijayapura with prolonged chest discomfort typical of myocardial ischemia, underwent standardized assessment with clinical history and examination, electrocardiogram at admission, cardiac enzymes – Troponin I, CPK-MB, and other necessary laboratory investigations along with 1ml of peripheral blood sample of the patient for analysis of CCR5 polymorphism.

2.2 DETECTION OF CCR5 POLYMORPHISM

The blood samples collectedfrom the patients of acute coronary syndrome are processed step by step as explained below and gene sequencing is performed. Based on the results of gene sequencing patients were grouped according to the presence of CCR5 polymorphism as group A and without as group B.

Clinical Sample (Blood) Collection: written informed consent was obtained from the patient, who were enrolled in the study. After taking consent, 1 ml peripheral blood samples were collected in the EDTA-coated vacutainers (BD367863) and stored at 4°C until further use.

Primer designing: The web-based freely available program "Primer3" which is widely accepted was used, (http://frodo.wi.mit.edu/ primer3/ input. Html) for designing PCR primers. All the designed primers for our target genes or region are tabulated in table No. 1 along with the annealing temperature and amplicon size. Primers were got synthesized by a commercial oligo synthesizer (MWG Biotech, India).

Table 4. Details of the primer sequences and annealing temperatures used for the amplification of CCR5 gene.

Name of the primer	Sequence	Amplicon Size(BasePairs)	Annealing Temperature
CCR5F	Forward:5CTCCCAGGAATCATCTTTACC3'	287bp	59.5°C
CCR5R	Reverse: 5'-TCATTTCGACACCGAAGCAG-3'		

Polymerase Chain Reaction (PCR): PCR amplification was carried out and primer annealing temperature was set depending on the annealing temperature of the primer (Table-1) for 10sec 72^0C for 15sec (primer extension) and a final extension at 72^0C for 5 min. The PCR cycling conditions were as follows Initial Denaturation is for 98^0C for10 sec, Denaturation is 980C for 10 sec, annealing is primer dependent for 10 sec, Elongation 72^0C for 5min & hold at 40^0C.

Agarose Gel Electrophoresis of PCR Products:

Gel electrophoresis is one of the molecular biology techniques used to separate DNA and RNA depending on the length of fragments. Nucleic acid molecules are separated based on an electric field to move the negatively charged molecules through an agarose matrix. Shorter

molecules move faster and migrate farther than longer ones because shorter molecules migrate more easily through the pores of the gel. This phenomenon is called sieving.

DNA Sequencing (Capillary Based)

PCR products were subjected for capillary based Big-Dye terminator sequencing. Prior to sequencing, the PCR products were subjected to cycle sequencing and plate processing.[50]

Cycle Sequencing

As per the Sanger Sequencing protocol, Big-Dye labeling and chain termination were carried out by the cycle sequencing method. To label each base, the PCR amplicon was subjected to a cycle sequencing reaction with a single primer. Big-Dye TM terminator v3.1was used for cycle sequencing (Applied Biosystems, USA) following the manufacturer's guidelines.

Table 5. Standardised master mix conditions for sequencing

SL.No.	Constituents	Quantity
1	Molecular Biology grade water	6.3 µL
2	Big Dye Buffer (5X)	1.3 µL
3	Big Dye	1.0 µL
4	Template (PCR product)	1.0 µL
5	Forward Primer	0.2 µL
6	Reverse Primer	0.2 µL
Total		**10 µL**

Table 6. The cycle sequencing conditions

Process	Temperature (°C)	Time
Initial. Denaturation	98	10sec

Denaturation	98	10sec
Annealing	Primer Dependent	10sec
Elongation	72	5min
Hold	4	

Note: The annealing temperature is primer dependant and varies for each primer.

Sequencing Run

Sample information sheets which contain analysis protocols along with the sample details were prepared and imported into the data collection software. Prepared samples were analyzed on ABI 3730 genetic analyser (Applied Biosystems, USA) to generate DNA sequences or electropherograms. After completion of the sequencing reaction, the quality of generated sequence was checked by using Sequencing Analysis v5.4software (Applied Biosystems, USA).

Sequence Alignment

The generated sequences were aligned to their respective reference sequences with the use of Variant reporter software (ABI v1.1). It performs sequence comparisons for novel mutations, known variants, insertions, and deletions. The results of the variant reporter were tabulated in PDF format as the default program of the software. Here, we used this technique to check the isolated genomic DNA from whole blood. In all the 81 acute coronary syndrome samples as shown in figure 9 confirmed the presence of genomic DNA and the same samples were taken for quantification based on Nanodrop.

Figure 9: Agarose gel image of genomic DNA of Acute coronary syndrome samples.

Quantification of Genomic DNA

We used Tecon multimode reader for the quantification of genomic DNA.For double stranded DNA, an Optical Density (OD) of 1 at 260 nm correlates to a DNA concentration of 50 ng/µl, so that DNA concentration can be easily calculated from OD measurements" as shown in Table no. 7

Table 7: Quantification of Acute coronary syndrome Samples.

Sl. No. of DNA samples	OD at 260/280	Concentration in ηg/µl
1	1.23	64
2	1.54	75
3	1.25	84
4	1.74	78
5	1.64	126
6	1.56	54
7	1.74	72
8	1.25	63
9	1.71	78
10	1.56	91
11	1.45	84
12	1.49	56
13	1.58	73
14	2.02	55
15	2.15	92
16	1.51	84
17	1.88	89
18	2.09	69
19	1.93	56
20	2.04	90
21	2.2	81.5
22	2.05	95.5
23	1.86	83.9
24	2.05	67

25	2.00	71
26	2.04	115
27	2.00	117
28	1.66	94
29	1.69	82
30	1.86	54
31	1.75	65
32	1.40	44
33	1.90	70
34	1.57	136
35	1.98	64
36	1.84	82
37	1.92	73
38	1.65	68
39	1.79	111
40	1.85	64
41	1.81	66
42	1.75	53
43	2.02	65
44	2.15	82
45	1.51	94
46	1.88	49
47	2.09	39
48	1.93	46
49	2.04	100
50	2.6	51.5
51	2.35	85.5
52	1.96	73.9
53	3.05	57
54	2.01	81
55	2.24	125

56	2.09	137
57	1.76	104
58	1.96	92
59	1.58	93
60	1.81	53
61	1.72	66
62	1.63	42
63	1.69	68
64	1.75	126
65	1.71	66
66	1.65	73
67	2.02	63
68	2.25	76
69	1.41	101
70	1.58	64
71	2.10	56
72	1.73	53
73	1.63	55
74	1.65	72
75	1.30	65
76	1.70	56
77	1.86	54
78	1.75	65
79	1.40	44
80	1.90	70
81	1.57	136

Polymerase Chain Reaction (PCR)

We used CCR5 gene specific primers as given in table 1 and carried out the PCR reactions. After PCR, the products were subjected to Gel electrophoresis, and results were documented for acute coronary syndrome samples.Primers (CCR 5) specific amplification

results are shown in figure 4. After the PCR amplification, the amplicons were run through 1% agarose gel electrophoresis and the DNA bands were observed in gel documentation (Figure 10).

The PCR product of 287bp

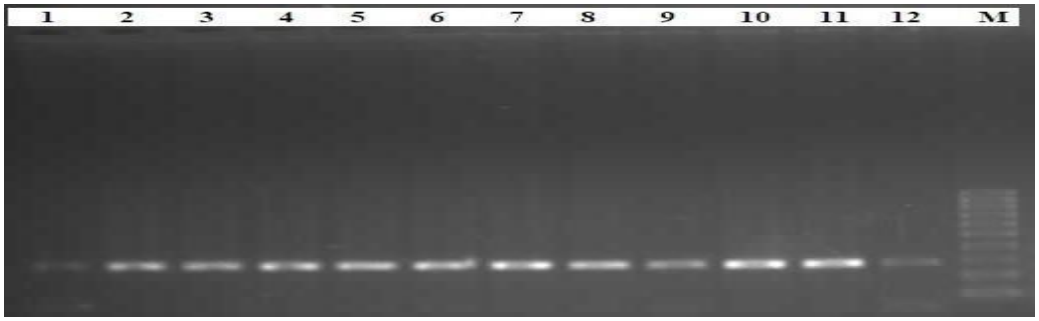


Figure 10: Agarose gel electrophoresis image of amplified products of gene. Lane No; 1-12 acute coronary syndrome samples, M: 100bp marker.

2.4 STATISTICAL ANALYSIS

- The data obtained is entered in a Microsoft Excel sheet, and statistical analysis will be performed using a statistical package for the social sciences (Version 20).
- Results are presented as Mean (Median) ±SD, counts and percentages, and diagrams.
- Categorical variables are compared using the Chi-square test.
- $p<0.05$ is considered statistically significant. All statistical tests are performed with two-tailed.
- Formula used

$$n = \frac{(z_\alpha + z_\beta)^2 2\, p^* q}{MD^2}$$

Where Z= Z statistic at a level of significance

MD= Anticipated difference between two proportions

P=Common Proportion

q= 100-p

- The data obtained is entered in a Microsoft Excel sheet, and statistical analysis will be performed using a statistical package for the social sciences (Version 20).
- Results are presented as Mean ± SD, counts and percentages, and diagrams.
- For normally distributed continuous variables between two groups is compared using independent t-test for not normally distributed variables Mann Whitney U test is used. Categorical variables between the two groups are compared using the Chi-square test.
- $p<0.05$ is considered statistically significant. All statistical tests were performed with two-tailed.

RESULTS

V. RESULTS

Total of 100 patients were admitted with acute coronary syndrome. Six patients were excluded from the study based on exclusion criteria. Out of 94 patients,13 patient gene sequencing could not be conducted due to financial problem and remaining 81 patient's gene sequencing was analysed for CCR5 polymorphism. Hence total of 81 patients were included in the study.

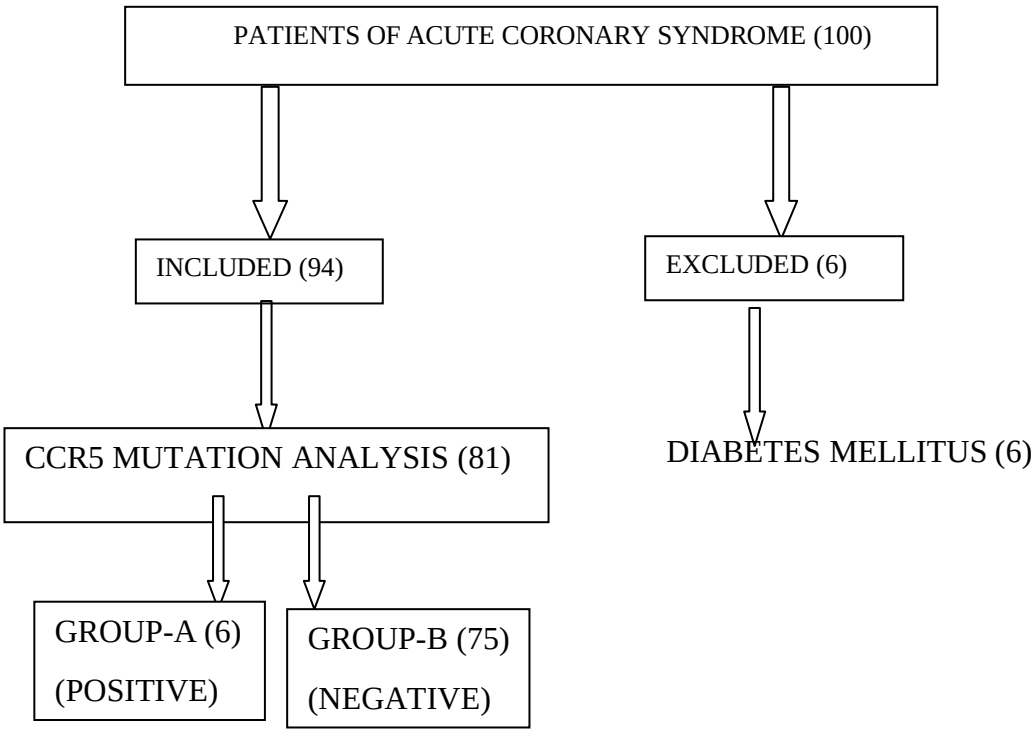


Figure 11: Flowchart showing included and excluded cases in the study

Note: - $p < 0.05$ – statistically significant

$P < 0.001$ – highly significant

Out of 81 patients with Acute coronary syndrome, 6 patients with presence of CCR5 mutation are in group A, and 75 patients with CCR5 mutation are in group B as shown in Table 8, Figure 11.

Table 8: Grouping of patients with CCR5 mutation.

CLASSIFICATION	NUMBER OF PATIENTS
GROUP A	6
GROUP B	75

1.1. AGE DISTRIBUTION

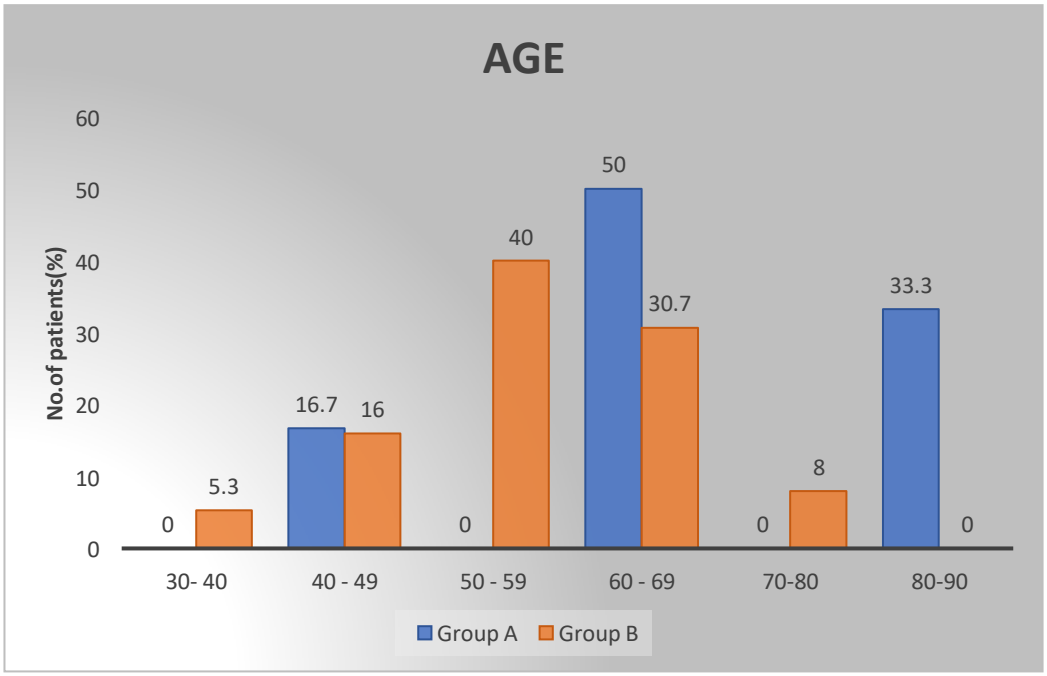
The 81 patients were grouped with an age frequency of 10 years. In group A patients between the age 40-49 years were 1 (16.7%), patients between the age 60-69 years were 3(50%), and patients aged more than 80 years were 2(33.3%). In group B patients aged between 30-40 years were 4(5.3%), patients between the age 40-49 years. were 12(16.0%), patients between the age 50-59 years were 30(40%), patients between the age 60-69 years were 23(30.7%), patients between the age 70-80 years were 6(8%). The most common age group in group A was 60-69years and group B was 50-59 years, with significant p value of 0.001 as described in Table 9, Graph 1.

Table 9: distribution of patients according to age

Age (Years)	Group A (n=6)		Group B (n=75)		P value
	NO. OF PATIENTS	Percentage	NO. OF PATIENTS	Percentage	
30- 40	0	0	4	5.3	0.001*
40 - 49	1	16.7	12	16.0	
50 - 59	0	0	30	40.0	
60 - 69	3	50.0	23	30.7	
70-80	0	0	6	8.0	
80-90	2	33.3	0	0	
Total	6	100.0	75	100.0	

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

Graph 1: DISTRIBUTION OF PATIENTS ACCORDING TO AGE



1.2. SEX DISTRIBUTION

Out of 81 patients in the study, 53 patients (65.4%) were male and 28 patients (34.5%) were female. In this study male patients were more than females as shown in table10. In group A 3 (50%) patients were male and 3 (50%) females; while 50(63.3%) patients were male and 25(33.3%) were female in group B as shown in Table 11, Graph 2.

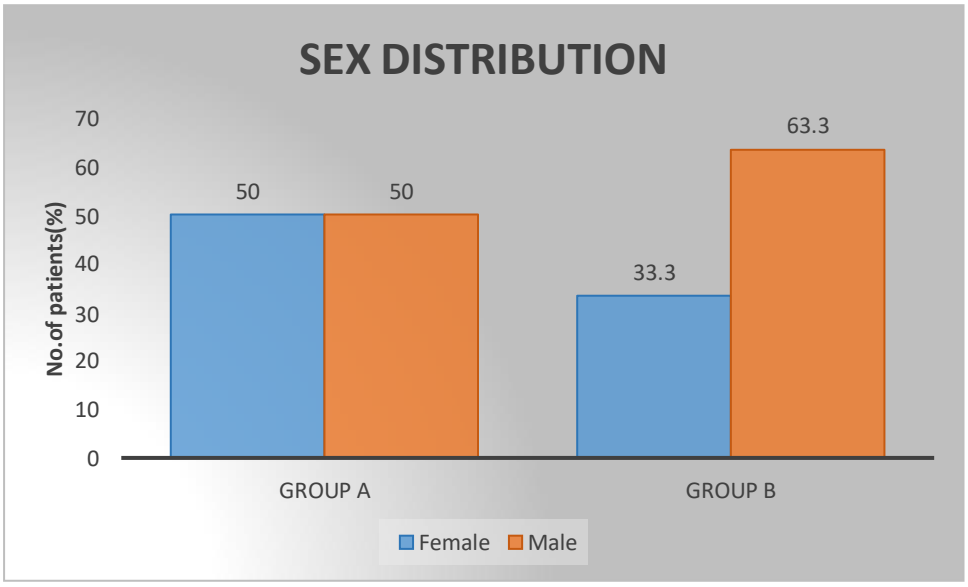
Table 10: Distribution of Sex among all cases

Sex	N	%
Male	53	65.4
Female	28	34.5
Total	81	100

Table 11: Distribution of Sex between study groups

SEX	Group A		Group B		P value
	NO. OF PATIENTS	Percentage	NO. OF PATIENTS	Percentage	
Female	3	50.0	25	33.3	0.4088
Male	3	50.0	50	63.3	
Total	6	100.0	75	100.0	

Graph 2: Distribution of Sex between study groups



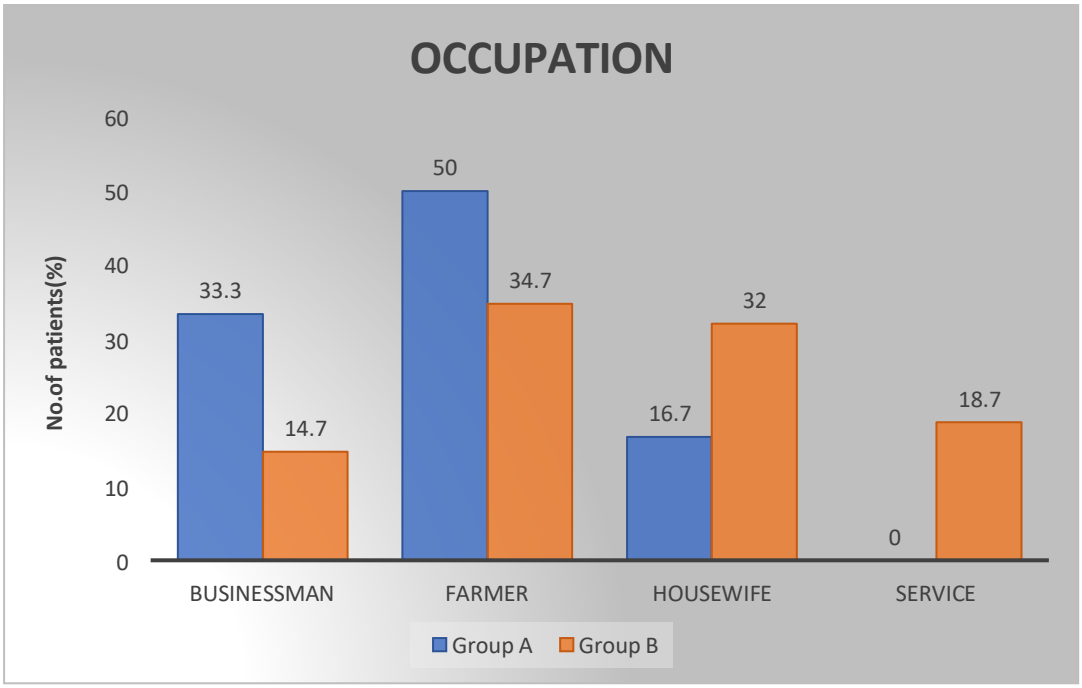
1.3. DISTRIBUTION OF PATIENTS ACCORDING TO OCCUPATION

In group A there were 3 (50%) farmers followed by businessman 2 (33.3%) and housewife- 1 (16.7%). while in group B, farmers-26(34.7%), housewife- 24 (32%), businessman- 11 (14.7%) and service employee- 14 (18.7%). The most common occupation associated with CCR5 mutation in this study was Farming followed by housewife, service employee and businessman, as depicted in Table 12, Graph 3.

Table 12: Distribution of Occupation between study groups

OCCUPATION	Group A		Group B		P value
	NO. OF PATIENTS	Percentage	NO. OF PATIENTS	Percentage	
BUSINESSMAN	2	33.3	11	14.7	0.3744
FARMER	3	50.0	26	34.7	
HOUSEWIFE	1	16.7	24	32.0	
SERVICE	0	0	14	18.7	
Total	6	100.0	75	100.0	

Graph 3: Distribution of Occupation between study groups



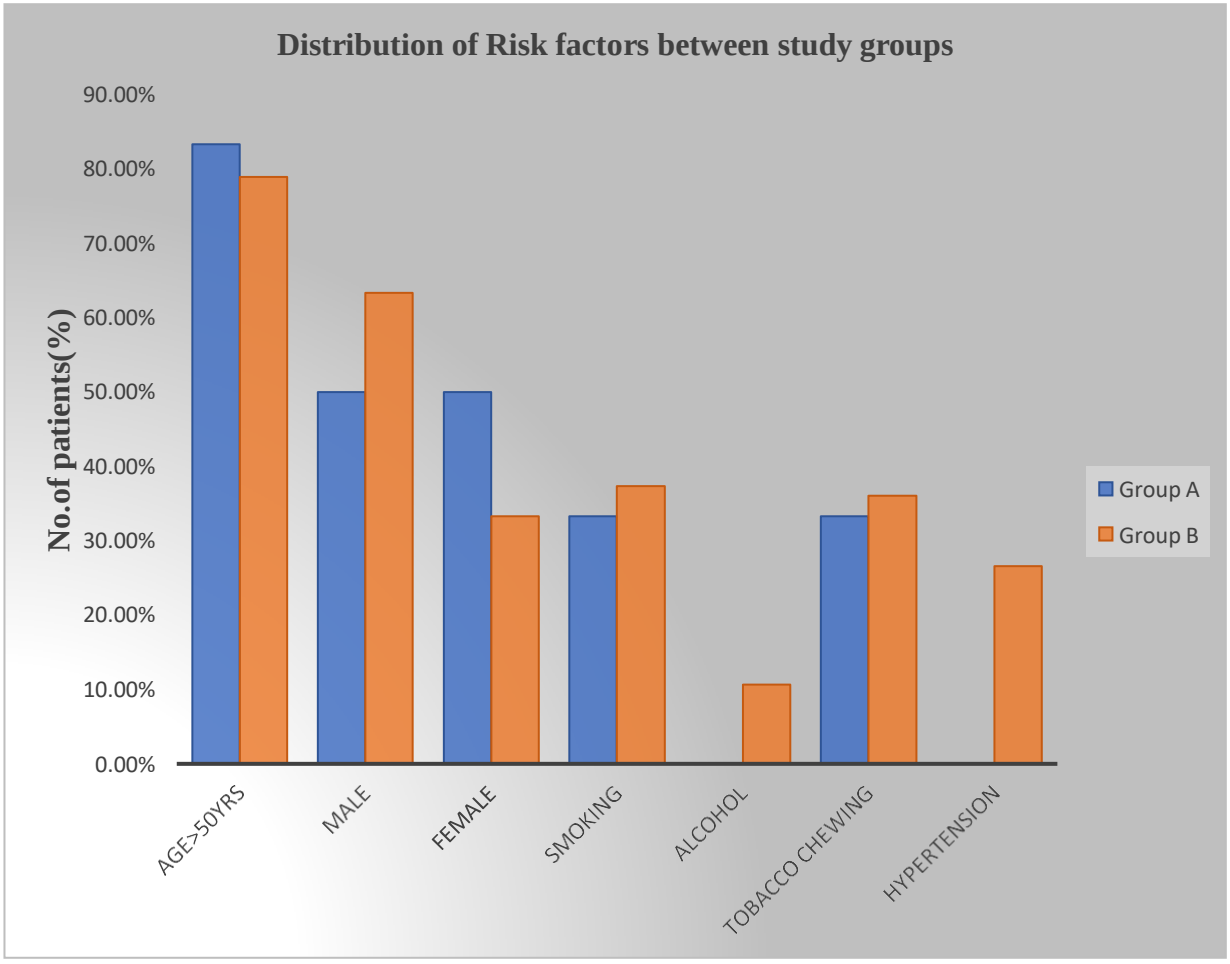
1.4. DISTRIBUTION OF PATIENTS ACCORDING TO RISK FACTORS:

Among risk factors, out of 81 patients in the study, 5 patients (83.3%) in group A compared to 59 patients (78.9%) in group B were aged more than 50 years. Male sex was seen in 3 patients (50%) compared to 50 patients (63.3%) in group B. Smoking habit was seen in 30 patients of which 2 patients (33.3%) are in group A and 28 patients (37.3%) in group B. Tobacco chewing was seen in 31 patients, of which 2 patients (33.3%) from group A and 29 patients (36%) in group B. Alcohol consumption was present in 8(10.6%) patients of only group B as shown in Table 13, Graph 4.

Table 13: Distribution of Risk factors between study groups

Risk factors		**Group A**		**Group B**		**p value**
		N	**%**	**N**	**%**	
Non-modifiable	Age>50yrs	5	83.3%	59	78.9%	0.906
	Sex					
	Male	3	50.0%	50	63.3%	0.4088
	Female	3	50.0%	25	33.3%	
Modifiable	Smoking	2	33.3%	28	37.3%	0.8452
	Alcohol	0	0%	8	10.6%	0.259
	Tobacco Chewing	2	33.3%	29	36.0%	0.7959
	Hypertension	0	0%	20	26.6%	0.1786

Graph 4: Distribution of Risk factors between study groups



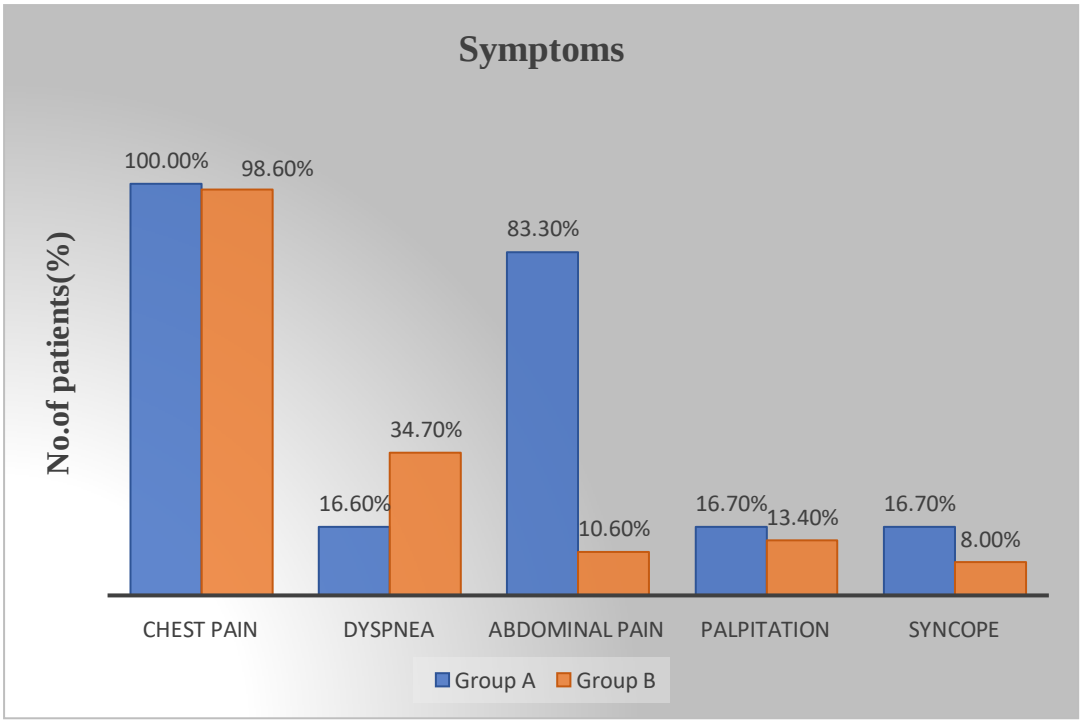
1.5. DISTRIBUTION OF PATIENTS ACCORDING TO SYMPTOMS:

In this study, as shown in Table 14, Graph 5, in both group A and group B the most common symptom was chest pain (100% vs 98.6%), followed by dyspnoea (16.6% vs 34.7%), abdominal pain (83.3% VS 10.6%), palpitations (16.7% VS 13.4%) and syncope (16.7% vs 8%).

Table 14: Distribution of Symptoms between study groups

Symptoms	Group A		Group B		P value
	NO. OF PATIENTS	%	NO. OF PATIENTS	%	
Chest pain	6	100.0%	74	98.6%	0.7759
Dyspnoea	1	16.6%	26	34.7%	0.3681
Abdominal Pain	5	83.3%	8	10.6%	0.0875
Palpitation	1	16.7%	10	13.4%	0.8186
Syncope	1	16.7%	6	8.0%	0.4672

Graph 5: Distribution of Symptoms between study groups



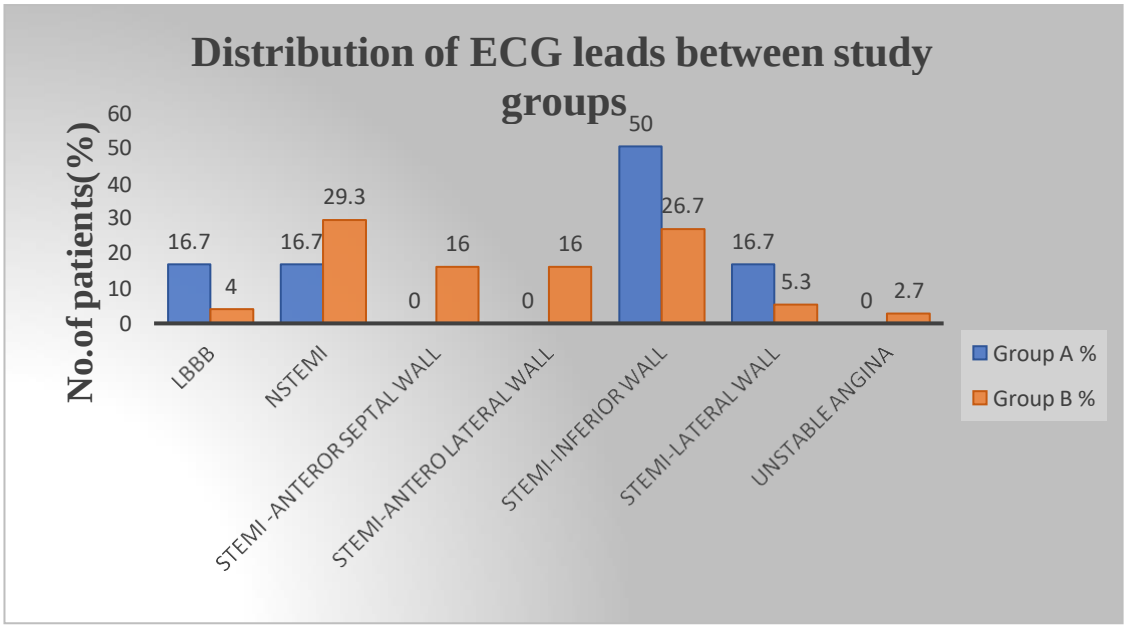
1.6. DISTRIBUTION OF PATIENTS ACCORDING TO ECG FINDINGS

Out of 81 patients, group A had inferior leads (II, III, aVF) ST elevation in 3 patients and 1 patient with LBBB, NSTEMI and lateral wall STEMI. In group B most common ECG finding was NSTEMI- 22(29.3%) followed inferior leads (II, III, aVF) ST elevation- 20(26.7%), antero-lateral (V3-V6, I, aVL) leads ST segment elevation-12(16%), unstable angina-2(2.7%) as depicted in Table 15, Graph 6.

Table 15: Distribution of ECG findings between study groups

ECG	Group A		Group B		p value
	N	%	N	%	
LBBB	1	16.7	3	4.0	0.5409
NSTEMI	1	16.7	22	29.3	
STEMI V1-V4	0	0	12	16.0	
STEMI V3-V6, I, aVL	0	0	12	16.0	
STEMI- II, III, aVF	3	50.0	20	26.7	
STEMI- I, aVL	1	16.7	4	5.3	
UNSTABLE ANGINA	0	0	2	2.7	
TOTAL	6	100	75	100	

Graph 6: Distribution of ECG leads between study groups



1.7. DISTRIBUTION OF PATIENTS ACCORDING TO ECHOCARDIOGRAPHIC VARIABLES:

In this study of 81 patients, echocardiographic parameters were analysed. Out of 6 patients in group A, 2 patients (33.3%) had antero-lateral wall hypokinesia, 1 patient (16.7%) had antero-septal wall hypokinesia, and 3 patients (50%) had inferior wall hypokinesia. While out of 75 patients in group B, 16 patients (21.3%) had antero-lateral wall hypokinesia, 14 patients (18.7%) had antero-septal wall hypokinesia, 9 patients (12%) had anterior wall hypokinesia, NO RWMA-2 patients (2.6%) and 32 patients (41.3%) had inferior wall hypokinesia. In this study most commonly, there was hypokinesia of inferior wall in both group A and group B with significant p value of 0.0290 as shown in Table 16, Graph 7.

In our study of 81patients were divided into group A and group B, distribution of left ventricular ejection fraction was studied as shown in table 17, Graph 8. In group A (CCR5 mutation Present cases) with 6 cases distribution of left ventricular ejection fraction according to regional wall motion abnormality showed LVEF of < 40% in 3 patients (50%) and > 40% in 3 patients (50%). While in group B (CCR5 mutation absent cases) with 75 cases distribution of left ventricular ejection fraction according to regional wall motion abnormality showed LVEF of < 40% in 42 patients (56%) and >40% in 33 patients (44%).

Table 16: Distribution of regional wall motion abnormality between study groups

Regional wall motion abnormality	Group A		Group B		p value
	N	%	N	%	
GLOBAL HYPOKINESIA	0	0	2	2.7	0.0290
HYPOKINESIA OF ANTERIOR WALL	0	0	9	12.0	
HYPOKINESIA OF ANTERO LATERAL	2	33.3	16	21.3	
HYPOKINESIA OF ANTERO-SEPTAL WALL	1	16.7	14	18.7	
HYPOKINESIA OF INFERIOR WALL	3	50	32	42.6	
NO RWMA	0	0	2	2.6	
Total	6	100	75	100	

Graph 7: Distribution of regional wall motion abnormality between study groups

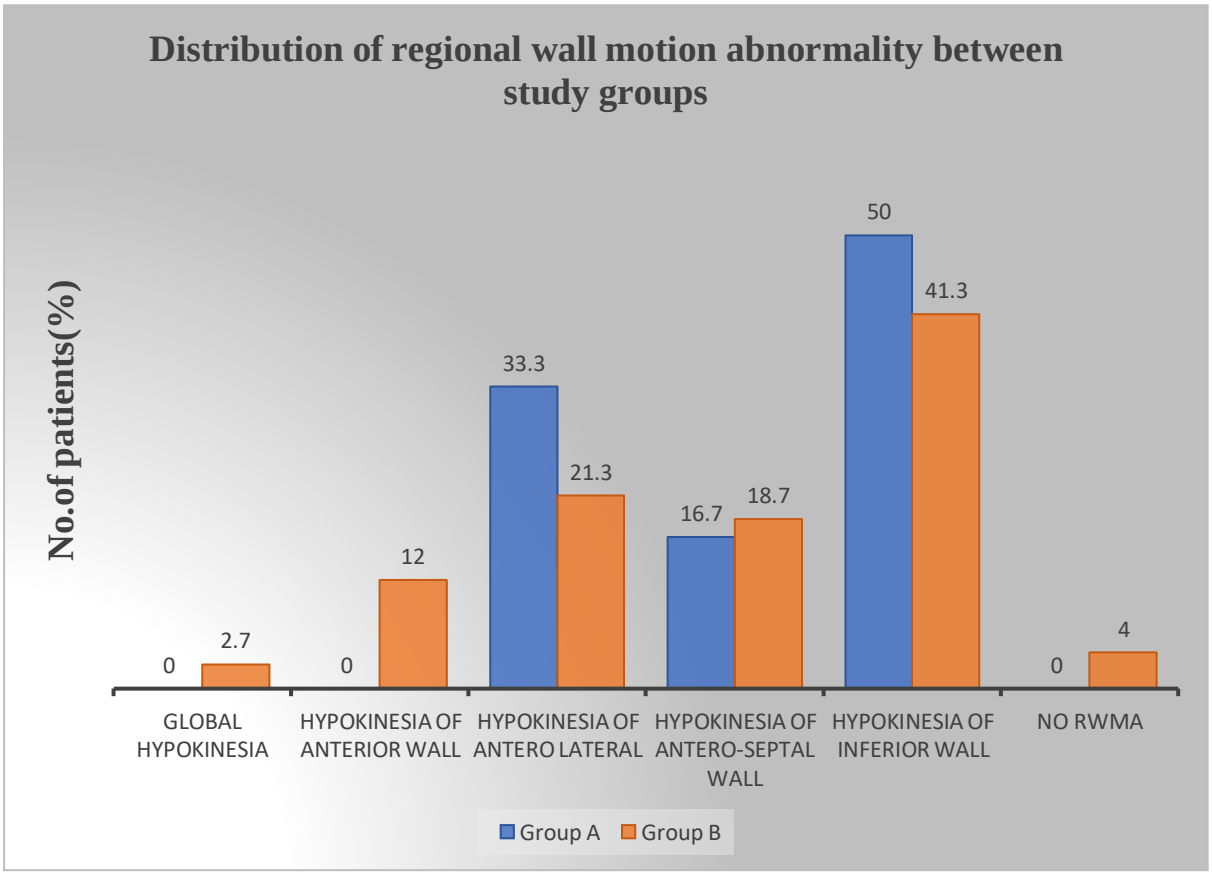


Table 17: Distribution of left ventricular ejection fraction between study groups

LVEF	Group A		Group B		P VALUE
	N	%	N	%	
<40%	3	50	42	56	0.8344
>40%	3	50	33	44	
TOTAL	6	100.00%	75	100.00%	

Graph 8: Distribution of left ventricular ejection fraction between study groups

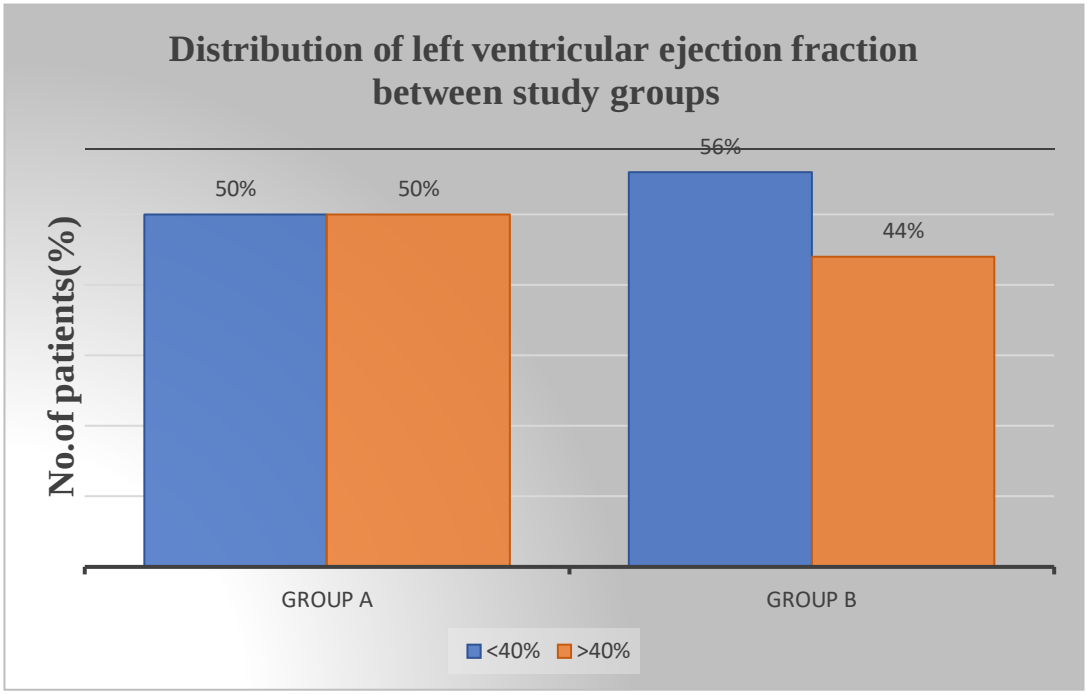


Table 18: Background Parameters between study groups

Parameters	Group A			Group B			p value
	Mean	Median	SD	Mean	Median	SD	
AGE (years)	65.83	64.00	13.92	55.75	57.00	9.146	**0.001***
Pulse Rate (beats per minute)	85.00	85.00	11.64	86.67	86.00	15.611	0.942
Respiratory Rate (cycles per minute)	18.83	18.00	1.60	18.60	18.0	2.422	0.817
Temperature (degree Celsius)	37.17	37.00	0.75	37.32	37.00	0.498	0.620
Haemoglobin (gm%)	12.00	12.00	1.60	13.12	13.00	2.205	0.177
Total Count (cells/cu.mm)	11186.6	10575.00	1184.39	11121.81	10400.0	3502.18	0.465
ESR (mm/hr)	23.33	19.50	17.17	16.08	15.00	9.548	0.425
RBS (mg/dl)	115.33	114.00	10.57	121.60	112.00	30.206	0.864
Blood Urea (mg/dl)	47.00	28.00	46.69	28.48	27.00	9.399	0.470
Sr. Creatinine (mg/dl)	1.67	1.00	1.21	0.97	1.00	0.231	**0.002***
Sr. Sodium (mmol/l)	137.50	137.50	3.50	136.32	136.00	3.824	0.435
Sr. Potassium (mmol/l)	4.33	4.50	0.81	4.17	4.00	0.476	0.366
Total cholesterol (mg/dl)	144.17	146.50	37.26	161.00	160.00	36.191	0.312
Triglycerides (mg/dl)	131.67	129.50	44.59	148.36	140.00	64.351	0.658
High-Density Lipoprotein(mg/dl)	34.00	34.50	12.94	35.56	34.00	7.963	0.928
Low-Density Lipoprotein(mg/dl)	76.67	61.50	27.59	84.80	82.00	26.642	0.170

Table 19: DISTRIBUTION OF CCR5 POLYMORPHISM IN STUDY SAMPLE

CCR5 mutation	Group A		Group B		P value
	NO. OF PATIENTS	Percentage	NO. OF PATIENTS	Percentage	
A	0	0	75	100.0	0.0001*
P	6	100.0	0	0	
Total	6	100.0	75	100.0	

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

Polymorphism analysis of CCR5 gene was done in the Genetic research lab, Department of Anatomy. Study was done on 81 patients admitted and diagnosed with Acute coronary syndrome. Out of 81 sample analysed for ccr5 polymorphism 6 (7.4%) were positive and 75 (92%) were negative. Out of 6 positive samples 5 of them had frameshift mutation. Base position in genomic DNA of all 6 positive mutation ranged from 8250 – 8340 in a narrow range.

Table 20: DETAILS OF CCR5 POLYMORPHISM ANALYSIS

SL No.	Sample no.	Base position in Genomic DNA Ref. NG_012637.1	Mutation type	Nucleotide change	AA change Ref. CCDS 2739.1	cDNA Ref. ENST00000445772.1	MUTATION (NOVEL/REPORTED)	Variant (v)
1	1 CCR5	g.8320T>G	Transversion	T-G	p.Y187D	C.559T>G	reported	Frameshift v
2	12 CCR5	g.8293T>A g.8429G>A	Transversion Transition	T-A G-A	p.C178S p.R223Q	C.532T>A C.668G>A	reported	Missense v Frameshift v
3	61 CCR5	g.8334G>T	Transversion	G-T	p.K191I	C.573G>T	reported	Frameshift v
4	65 CCR5	g.8332A>G	Transition	A-G	p.K191E	C.571A>G	reported	Frameshift v
5	66 CCR5	g.8334G>T	Transversion	G-T	p.K191I	C.573G>T	reported	Frameshift v
6	77 CCR5	g.8334G>C	Transversion	G-C	p.K191N	C.573G>C	reported	Frameshift v

DISCUSSION

VI. DISCUSSION

This study is a prospective cross-sectional study conducted from January 2020 to June 2022. The aim was to study CCR5 polymorphism in patients of acute coronary syndrome. 81 patients included in this study were analysed for clinical history, blood investigations, ECG, 2D-ECHO and CCR5 polymorphism.

1.1. AGE

In this study the most common age group was 50-70 years with a significant p value of 0.001. Similarly, in a study done by Rosengren et al, in 2006 on 10253 patients, concluded presenting age group was 65-74 years[51].

Another study done by Kobayashi A *et al* on 190 patients hospitalized with acute coronary syndrome between years January 2007- December 2013, they observed that common age group was 60-70yrs.[52]In a study done by Aygul N *et al*, mean age group was found to be 60-70 yrs.[53]

The reason could be lack of education about disease and risk factors, evidence-based treatment, lack of compliance of medications.

1.2. SEX

In this study there was male predominance as 60.7% of patients were males and female patients were 39.3%, which was similar to study done by Nedkoff L J et al. in year 1996 to 2007 on 29421 patients where male patients were 19601(66.6%) and female patients were 9820 (33.4%).[54]

In a study done by Jonathan D Newman et al. between February 1, 2009 and June 30, 2010 out of 476 subjects, male patients were 68.7% and female were 31.3%[55]. In another study

done by Sharma R et al. in 2014 on 1562 South Indian patients, Majority were male 1242 (79.5%) and rest were females320 (20.5%) which was significantly higher than this study[56].

1.3. OCCUPATION

In this study the most common occupation associated with CCR5 polymorphism in both group A and group B was farmers, 3 (50%) and 26 (34.7%) respectively. In group A there were 3 (50%) farmers followed by businessman 2 (33.3%) and housewife- 1 (16.7%). while in group B, farmers-26(34.7%), housewife- 24 (32%), businessman- 11 (14.7%) and service employee- 14 (18.7%). Most of these patients belong to low and middle socioeconomic status. The reason could be lack of education about disease, risk factors, inability to afford for treatment, lack of compliance to medication, inability to modify risk factors and lack of regular follow up.

1.4. RISK FACTORS:

Non- modifiable risk factors like age and gender are been discussed above. In this study, modifiable risk factors like Smoking habit were seen in 30 patients of which 2 patients (33.3%) are in group A and 28 patients (37.3%) in group B. Tobacco chewing was seen in 31 patients, of which 2 patients (33.3%) from group A and 29 patients (36%) in group B. Alcohol consumption was present in 8(10.6%) patients of only group B and none ingroup A. There is significant variation in various risk factors and their association with acute coronary syndrome in different studies. In a study done by Vinay Rao et al. in 2017, in 100 patients with acute coronary syndrome, it was observed that hypertension was present in 52% of patients, smoking was present in 61% of patients, alcohol consumption in 29% of patients[57]. This study has high incidence of risk factors like smoking and tobacco chewing for acute coronary syndrome compared to this study. In a study done by Unal et al. between 1981 to 2000, they concluded that, life expectancy of patients with ACS can be increased four times

than that is increased by modern cardiological treatment by modest reduction in major risk factors like smoking, hypertension, diabetes mellitus[58]. Therefore, there is need for policies to control tobacco use, promote healthy diet and educate patients regarding adverse effects of tobacco use, which help in improving life expectancy of patients with ACS.

1.5. SYMPTOMS

In this study, in both group A and group B the most common symptom was chest pain (100% vs 98.6%), followed by dyspnea (16.6% vs 34.7%), abdominal pain (83.3% VS 10.6%), palpitations (16.7% VS 13.4%) and syncope (16.7% vs 8%).

Similarly in a study done by Pravin K Goel et al. from January, 2008–December, 2008 on 609 patients admitted with ACS, they found that the most common symptom in patients with acute coronary syndrome was chest pain (n=510, 84%), followed by dyspnoea (n=53, 8.7%) and epigastric pain (n=16, 2.6%) which is similar to our study.[59]

In other study done by J G Conto et al. on 434877 patients admitted with acute myocardial infarction from June 1994 to March 1998 in the National Registry of Myocardial Infarction-2, which includes 1674 hospitals in the United States, they found that chest pain was present in 67% (n=291367) of patients which is less than that observed in this study.[60]

1.6. CHEMOKINE RECEPTOR 5 POLYMORPHISM

In this study out of 81 samples analysed for CCR5 polymorphism 6 patients were positive for polymorphism with majority of them showing frameshift mutation. Out of 6 positive cases there was equal distribution of polymorphism among male and female. In 2006, S. Sharda et al study on Chemokine receptor 5(CCR5) deletion polymorphism in North Indian patients with coronary artery disease showed 3 times higher frequency of polymorphism in CAD patient compared to normal individual.[5]In 2011, Neha Singh et al study showed similar results with four times higher frequency of polymorphism in acute myocardial infarction patient.[6]

In 2010, Craig L. Hyde, et al study showed CCR5Δ32 deletion and increased plasma high-density lipoprotein cholesterol and decreased plasma triglycerides, protective for cardiovascular disease. In 2008, Ali R. Afzal, et al study conducted in the Bruneck population, polymorphism was associated with significantly lower carotid intima-media thickness in the common carotid artery, and reduced incidence of cardiovascular disease. Similarly other studies conducted out of India in Spain, Czech-republic, Germany and Hungary show less frequency of polymorphism in CAD patients concluding protective role in their ethnicity and population. The two Indian studies done in North Indian population explained above, show significant positive association of CCR5 polymorphism and coronary artery disease with no protective role. Our study is the first to be conducted in South Indian population showing evidence of CCR5 polymorphism in acute coronary syndrome patients.

VII. CONCLUSION

Acute coronary syndrome is no more a disease of elderly population, nowadays incidence is increased substantially in younger individuals even with no associated comorbidities. Genetic study in each disease is gaining more popularity and importance to study the disease in detail.

Our study on the role of CCR5 polymorphism in acute coronary syndrome shows positive association between polymorphism and disease. The study shows that our population is genetically susceptible for acute coronary syndrome and CCR5 polymorphism could be considered as one of the etiologies for acute coronary syndrome.

By screening for CCR5 polymorphism in high-risk individuals, we can provide a better and effective early intervention to the individuals and thereby reduce the social burden, morbidity and mortality of disease.

SUMMARY

SUMMARY

Eighty-one patients with Acute coronary syndrome admitted at BLDE (Deemed to be University), Shri B M Patil Medical College Hospital and Research Centre, Vijayapura between from January 2020 to June 2022 were studied.

This study was conducted to know CCR5 polymorphism incidence and distribution of polymorphism in patients of acute coronary syndrome in Vijayapura population.

1. Total of 100 patients were studied out of which 6 were excluded based on exclusion criteria. Rest 81 patients were classified into group A, with presence of CCR5 polymorphism (6 patients) and group B, with absence of polymorphism (74 patients).
2. In this study male patients (65.4%) were more than females (34.5%), whereas in group A 3 male (50%) and 3 females (50%) compared to 50 male (63.3%) and 25 females (33.3%) in group B.
3. The most common age group in both group A and group B was between 60-69 years. 3 (50%) patients in group A, and 23 (30.7%) in group B were more than 60 years age.
4. The most common risk factors in group A, were smoking (33.3%), and tobacco chewing (33.3%).
5. The most common occupation in both group A and group B was farming. In group A farmers were followed by business, housewife and service employee.
6. The most common symptom in both group A and group B was chest pain followed by dyspnoea, abdominal pain, palpitations and syncope.
7. In this study most commonly, there was hypokinesia of inferior wall in group A (50%) and group B (42.6%).
8. In group A around 50% patients had ejection fraction less than 40% compared to 56% in group B.

9. Out of 81 patients CCR5 gene sequencing, 6 patients had polymorphism with an incidence of 7.5 % ($p<0.001$). The study shows our population is genetically susceptible for acute coronary syndrome and CCR5 polymorphism could be considered as one of the etiologies for acute coronary syndrome.

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IX. BIBLIOGRAPHY

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ANNEXURES

ANNEXURE I

INSTITUTIONAL ETHICAL CLEARENCE CERTIFICATE.

B.L.D.E. (DEEMED TO BE UNIVERSITY) IEC/100-9/2021 Date-22/01/2021

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Institutional ethical committee of this college met on 11-01-2021 at 11am to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: Genetic study of chemokine receptor gene (CCR5) polymorphism in acute coronary syndrome in vijayapura population

Name of PG student: Dr Prashanth M R, Department of Medicine

Name of Guide/Co-investigator: Dr Sharanabasawappa R Badiger
Prof & HOD of Medicine

DR S.V.PATIL
CHAIRMAN
Institutional Ethical Committee
B L D E (Deemed to be University)
Shri B.M. Patil Medical College,
VIJAYAPUR-586103 (Karnataka)

Following documents were placed before Ethical Committee for Scrutinization:

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.

4

ANNEXURE – II

CONSENT FORM

B.L.D.E. (DEEMED TO BE UNIVERSITY) SHRI B.M. PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTER, VIJAYAPURA-586103

INFORMED CONSENT FOR PARTICIPATION IN DISSERTATION / RESEARCH.

I, the undersigned,_____S/O D/O W/O_____, aged__years, ordinarily resident of_____do hereby state/declare that Dr PRASHANTH M.R of BLDE (DU), Shri. B. M. Patil Medical College Hospital and Research Centre has examined me thoroughly on_____at_____(place) and it has been explained to me in my own language that I am suffering from_____disease (condition) and this disease/condition mimic following diseases. Further Doctor Dr PRASHANTH M.R informed me that he/she is conducting dissertation/research titled "GENETICSTUDY OF CHMOKINE RECEPTOR GENE (CCR5) POLYMORPHISM IN ACUTE CORONARY SYNDROME IN VIJAYAPURA POPULATION" under the guidance of Dr. Badiger Sharanabasawappa requesting my participation in the study. Apart from routine treatment procedure, the pre-operative, operative, post-operative and follow-up observations will be utilized for the study as reference data. Doctor has also informed me that during conduct of this procedure like adverse results may be encountered. Among the above complications most of them are treatable but are not anticipated hence there is chance of aggravation of my condition and in rare circumstances it may prove fatal in spite of anticipated diagnosis and best treatment made available. Further Doctor has informed me that my participation in this study help in evaluation of the results of the study which is useful reference to treatment of other similar

cases in near future, and also, I may be benefited in getting relieved of suffering or cure of the disease I am suffering.

The Doctor has also informed me that information given by me, observations made photographs video graphs taken upon me by the investigator will be kept secret and not assessed by the person other than me or my legal hirer except for academic purposes.

The Doctor did inform me that though my participation is purely voluntary, based on information given by me, I can ask any clarification during the course of treatment / study related to diagnosis, procedure of treatment, result of treatment or prognosis. At the same time, I have been informed that I can withdraw from my participation in this study at any time if I want or the investigator can terminate me from the study at any time from the study but not the procedure of treatment and follow-up unless I request to be discharged.

After understanding the nature of dissertation or research, diagnosis made,

mode of treatment, I the undersigned Shri/Smt_____under my full conscious state of mind agree to participate in the said research/dissertation.

Signature of patient:

Signature of doctor:

Witness:

Date:

Place

ANNEXURE – III: SCHEME OF CASE TAKING PROFORMA

BLDE (DEEMED TO BE UNIVERSITY)

SHRI B.M. PATIL MEDICAL COLLEGEHOSPITAL AND RESEARCH CENTRE,

VIJAYAPUR.

"**GENETIC STUDY OF CHEMOKINE RECEPTOR GENE (CCR5) POLYMORPHISM IN ACUTE CORONARY SYNDROME IN VIJAYAPURA POPULATION**"

Name: CASE NO:

Age: IP NO:

Sex: DOA:

Religion: DOD:

Occupation:

Residence:

Presenting complaints:

History of present illness:

Past History:

Family History:

Personal History:

Diet/appetite

Sleep

Bladder and bowel habits:

Smoking/Tobacco chewing/Alcohol

General Physical Examination:

Vitals

PR:

BP:

RR:

Temp:

Hair:

Eyes:

Pupils:

Nose:

Ears:

Oral Cavity:

Upper Limbs:

Chest:

Abdomen:

Genitalia:

Lower Limbs:

Skin:

SYSTEMIC EXAMINATION

Cardiovascular System

Arterial system:

Pulse

Rate

Rhythm

Volume

Character

Condition of the vessel wall

Radio radial

Radio femoral delay

Other peripheral pulses

Venous system:

Engorged veins in the neck

Blood Pressure

Precordial examination:

Inspection:

Palpation:

Auscultation:

Respiratory System:

Per abdomen:

Central Nervous System:

INVESTIGATIONS

HAEMATOLOGY –

Haemoglobin	gm %
Total WBC counts	Cells/mm^3
Differential counts -	
Neutrophils	%

Lymphocytes	%
Eosinophils	%
Monocytes	%
Basophils	%
ESR	mm after 1 hour

BIOCHEMISTRY–

Random blood sugar	mg/dl
Blood urea	mg/dl
Serum creatinine	mg/dl
Serum sodium	mEq/L
Serum potassium	mEq/L

URINE EXAMINATION -

Albumin	
Sugar	
Microscopy	

LIPID PROFILE

Total cholesterol	mg/dl
Triglycerides	mg/dl
HDL	mg/dl
LDL	mg/dl

TROPONIN I:

CPK MB:

2D-ECHO DOPPLER:

ECG-

	ECG
Standardization	
Rate	
Rhythm	
P wave	
PR interval	
QRS complex	
QRS configuration	
QRS duration	
QRS Axis	
ST-Segment	

T wave	
QT interval	
QT_C interval	

ECG:

Chemokine receptor (CCR5) Polymorphism :

ANNEXURE IV: MASTER CHART

KEY TO MASTER CHART:

A: ABSENT

AF: ATRIAL FIBRILLATION

B: BUSINESSMAN

CCR5: CHEMOKINE RECEPTOR 5

DE: DEATH

D: DEPRESSION

E: ELEVATION

EM: EMPLOYEE

FA: FARMER

F: FEMALE

H: HOUSEWIFE

L: LABOUR

LBBB: LEFT BUNDLE BRANCH BLOCK

LVEF: LEFT VENTRICULAR EJECTION FRACTION

M: MALE

P-PRESENT

SR: SINUS RHYTHM

VT-VENTRICULAR TACHYCARDIA

SL N.	PATIENT NAME	AGE	SEX	OCCUPATION	IP N.	D.O.A	D.O.D	SYMPTOMS						DIABETES	HYPERTENSION	FAMILY HISTORY	HABITS			VITALS			HEMOGLOBIN	TOTAL COUNT	ESR	RBS	BLOOD UREA	Sr. CREATININE	Sr. SODIUM	Sr. POTASSIUM				TROPONIN I	CPK MB	ECG		2D-ECHO			
1	lalitha sadugol	60	F	HOUSEWIFE	43985	09-01-2021	15-01-2021	P	p	a	a	a	a	p	NA	a	a	p	90	180/110	37.5	20	13.6	12450	10	123	31	0.7	143	4.4	130	90	40	74	54.8	POSITIVE	LBBB	SR	GLOBAL HYPOKINESIA	25%	E	
2	MAHABUBBE CHIMALAGI	45	F	HOUSEWIFE	152027	22-03-2021	28-03-2021	P	A	A	P	A	A	A	NA	A	A	A	90	110/70	38	18	11	10400	15	110	16	0.5	135	3.8	200	240	40	100	14859	POSITIVE	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	45%	I	NEGATIVE
3	MAHANANDA EMM	55	F	HOUSEWIFE	3842	31-03-2021	04-04-2021	P	A	A	A	A	A	A	NA	A	A	p	84	120/80	36.7	18	11	15500	10	135	27	1	141	4.1	150	136	30	96	800	POSITIVE	NSTEMI	SR	HYPOKINESIA OF ANTERIOR WALL	45%	I	NEGATIVE
4	ANANDKUMAR LONARMATH	58	M	DESK JOB	4487	03-04-2021	08-04-2021	P	A	A	P	A	A	P	NA	P	A	A	98	170/100	38	20	16.9	10000	11	138	20	0.9	138	5	160	140	36	90	252	POSITIVE	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	45%	I	NEGATIVE
5	RENUKA	51	F	HOUSEWIFE	31203	21-04-2021	28-04-2021	P	A	A	A	A	A	A	NA	A	A	A	84	130/90	37	18	12	7000	15	118	30	0.9	132	3.7	158	110	34	88	1100	POSITIVE	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	61%	I	NEGATIVE
6	SHANKAR	70	M	FARMER	34178	23-04-2021	29-04-2021	P	P	A	P	A	A	A	NA	P	A	P	70	120/80	37	18	12	9000	15	120	25	0.9	132	4	164	200	30	80	1800	24	STEMI-INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	45%	I	NEGATIVE
7	SUKHDEV GALAVE	60	M	FARMER	34104	23-04-2021	28-04-2021	P	P	A	P	A	A	A	NA	P	A	P	70	118/70	38	16	14.4	9600	14	118	38	1.5	139	4.6	190	150	34	130	POSTIVE	POSITIVE	STEMI-INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	45%	I	NEGATIVE
8	LAXMIBAI PATIL	55	F	HOUSEWIFE	35468	24-04-2021	30-04-2021	P	A	A	A	A	A	A	NA	A	A	A	76	140/90	37	16	12.4	7200	11	162	25	1.1	138	3.9	174	180	30	78	1790	POSITIVE	NSTEMI	SR	HYPOKINESIA OF ANTERO LATERAL WALL	45%	I	NEGATIVE
9	SHIVALINGAPPA	48	M	TEACHER	80879	10-05-2021	15-05-2021	P	P	A	A	A	A	A	NA	P	A	A	90	100/60	38	18	15	11640	20	110	27	1.3	132	3.7	190	146	38	88	POSITIVE	POSITIVE	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	25%	I	NEGATIVE
10	CHINAWWA SAVADATTI	58	F	HOUSEWIFE	58448	15-05-2021	21-05-2021	P	P	A	P	A	A	A	NA	A	A	A	110	110/70	37	24	8.7	12500	13	100	52	0.9	134	4	131	215	27	61	16248	POSITIVE	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	35%	I	NEGATIVE
11	MHD.RAFIQ WALIKAR	54	M	BUSINESSMAN	58255	15-05-2021	23-05-2021	P	P	A	A	A	P	A	NA	A	A	P	86	100/70	37	24	15.1	10200	21	206	19	1	138	3.7	140	200	28	70	2520	POSITIVE	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	30%	E	
12	BHAGIRATHI GULED	45	F	HOUSEWIFE	60650	18-05-2021	25-05-2021	P	A	A	P	A	A	P	NA	A	A	A	90	90/70	37	18	15.7	8310	20	130	25	0.7	132	4	220	240	42	88	358	POSITIVE	LBBB	SR	GLOBAL HYPOKINESIA	25%	E	NEGATIVE
14	SITARAM RAJPUT	60	M	SHOP KEEPER	104252	04-06-2021	08-06-2021	P	A	A	A	A	A	P	NA	A	A	A	90	130/80	37.5	18	13.2	20580	5	110	40	0.6	142	4.6	190	145	30	130	POSITIVE	68	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	55%	I	NEGATIVE
15	SANGAPPA	49	M	FARMER	78120	12-06-2021	17-06-2021	P	A	A	A	A	A	P	NA	A	A	P	88	130/70	37	18	11	15000	13	110	60	1	135	4.8	170	130	30	90	351	POSITIVE	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERIOR WALL	45%	I	NEGATIVE
16	DASTAGIRSAB NADAF	60	M	BUSINESSMAN	80882	16-06-2021	22-06-2021	P	A	P	A	A	A	P	NA	P	A	P	94	110/70	37	20	15.7	12150	10	116	16	0.9	140	4.2	179	154	49	99	24000	POSITIVE	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERO SEPTAL WALL	35%	I	NEGATIVE
17	SHANTABAI BALI	80	F	HOUSEWIFE	84609	21-06-2021	26-06-2021	P	A	A	A	A	A	A	NA	A	A	A	80	110/70	37	18	11.9	12000	14	124	14	1.2	139	4.2	190	147	33	128	7679	POSITIVE	NSTEMI	SR	HYPOKINESIA OF ANTERO LATERAL WALL	40%	I	POSITIVE
18	BASAPPA LONI	70	M	FARMER	85323	22-06-2021	01-07-2021	P	P	A	A	A	A	A	NA	P	P	P	96	90/60	38	24	10	2500	30	89	31	1.2	137	4.3	88	59	30	60	8944	POSTIVE	STEMI-INFERIOR WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	30%	I	NEGATIVE
19	RAJSHEKHAR SINDAGI	65	M	FARMER	86249	23-06-2021	01-07-2021	P	P	A	A	A	A	A	NA	P	A	P	100	100/60	37	18	14.5	3600	5	125	29	0.9	139	3.8	136	188	29	69	223	POSITIVE	STEMI-INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	40%	I	NEGATIVE
20	YASHUBAI	60	F	HOUSEWIFE	86353	24-06-2021	28-06-2021	P	P	A	P	A	A	P	NA	A	A	P	90	90/60	38	24	13.8	10140	15	120	22	0.7	136	4.3	303	184	48	200	2587	86	STEMI-ANTERO LATERAL WALL	SR	HYPOKINESIA OF ANTERIOR WALL	30%	I	NEGATIVE
21	SAHEBAGOUDA IRAPPA	39	M	CLERK	98314	06-07-2021	16-07-2021	P	A	A	A	A	A	A	NA	A	A	A	90	130/80	37	18	15.7	10056	20	121	24	0.9	138	3.5	144	92	38	88	193	40	STEMI-ANTERO LATERAL WALL	SR	HYPOKINESIA OF ANTERO SEPTAL WALL	25%	I	NEGATIVE

22	GOURAMMA NUCHCHI	75	F	HOUSEWIFE	103994	09-07-2021	15-07-2021	P	P	A	A	A	A	A	NA	A	A	A	98	120/70	37	60	13	13000	13	140	34	0.6	137	4.6	177	57	43	123	7350	20	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERIOR WALL	40%	I	NEGATIVE
23	sushilabai chavan	55	f	HOUSEWIFE	76919	11-07-2021	16-07-2021	P	a	a	a	a	a	a	na	a	a	a	86	140/90	37	18	10	6400	15	126	16	0.8	137	3.5	166	333	28	71	109.2	38	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	40%	I	NEGATIVE
24	ashok hada pad	65	M	FARMER	105626	12-07-2021	23-07-2021	P	p	a	a	a	a	a	na	a	a	p	60	100/60	37	18	12	10000	15	96	35	1.2	136	4.2	59	58	16	40	positive	POSITIVE	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	45%	I	
25	NIRMALA PATTAR	38	F	HOUSEWIFE	78301	13-07-2021	24-07-2021	P	P	A	P	A	A	A	NA	A	A	A	88	110/70	37.8	15	14.5	7740	26	100	26	0.7	139	4.2	190	56	38	140	300	POSITIVE	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	50%	I	NEGATIVE
26	kantabai belage	70	F	HOUSEWIFE	90731	13-07-2021	25-07-2021	P	p	a	a	a	a	a	na	a	a	a	90	90/60	37.5	24	11.3	13570	23	126	38	1	135	4.3	269	164	52	184	>40,000	POSITIVE	STEMI-ANTERO LATERAL WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	25%	I	
27	BASAVANAGOUDA NAGAPPA	68	M	FARMER	108350	13-07-2021	18-07-2021	P	P	A	A	A	A	A	NA	P	P	P	64	110/80	36	16	11	9000	14	124	24	1	135	4.2	154	80	26	112	198	45	STEMI-ANTERO LATERAL WALL	SR	HYPOKINESIA OF ANTERIOR WALL	40%	I	
28	CHANDRAVVA	58	F	HOUSEWIFE	59447	15-07-2021	24-07-2021	P	A	A	A	A	A	A	NA	A	A	A	110	110/70	37.5	18	8.7	12500	16	145	52	0.9	120	4	110	68	50	72	16248	POSITIVE	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF ANTERO SEPTAL WALL	35%	I	NEGATIVE
29	SUREKHA	45	F	HOUSEWIFE	103883	16-07-2021	21-07-2021	P	A	A	A	A	A	A	NA	A	A	A	90	120/80	37	18	8.2	14300	35	134	28	0.6	139	4	140	180	32	60	200	POSITIVE	NSTEMI	SR	HYPOKINESIA OF ANTERO SEPTAL WALL	35%	I	NEGATIVE
30	SANGANGOUDA	65	M	BUS DRIVER	79932	17-07-2021	19-07-2021	P	P	A	A	A	A	A	NA	A	A	A	92	120/70	36	20	14.6	10450	29	104	23	0.9	129	4.6	240	124	46	180	103	POSITIVE	NSTEMI	SR	HYPOKINESIA OF ANTERO SEPTAL WALL	35%	I	NEGATIVE
31	SURESH SHRISHAIL	41	M	CLERK	113104	17-07-2021	21-07-2021	P	P	A	A	A	A	A	NA	P	A	A	80	100/70	37	18	14.2	8040	3	100	11	0.6	128	4.7	235	161	53	150	24909	132	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	45%	I	NEGATIVE
32	IRAYYA hiremath	58	m	FARMER	114796	20-07-2021	02-08-2021	P	a	a	a	a	a	p	na	p	a	p	98	160/90	38	16	16.2	14860	15	100	32	1.6	140	3.7	140	116	40	77	533	positive	EVOLVED MI- INFERIOR WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	35%	I	NEGATIVE
33	LAXMIBAI SIDAREDDY	50	F	HOUSEWIFE	119274	23-07-2021	02-08-2021	P	P	A	A	A	P	A	NA	A	A	A	116	90/60	38	22	14.7	18720	25	209	23	1.2	134	4.2	190	150	28	138	9843.5	POSITIVE	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERIOR WALL	25%	E	
34	MAHADEVI GOUDAPPANAVAR	66	F	HOUSEWIFE	127433	30-07-2021	04-08-2021	P	P	A	A	A	A	A	NA	A	A	A	84	150/90	37	20	10.4	11600	5	108	20	1	139	3.2	140	200	28	74	1090	POSITIVE	NSTEMI	SR	HYPOKINESIA OF ANTERIOR WALL	45%	I	NEGATIVE
35	MALLAPPA MIRAGI	52	M	FARMER	128687	31-07-2021	10-08-2021	P	P	A	A	P	A	A	NA	P	A	A	66	120/80	37	20	16.8	11600	15	109	18	1.2	134	4.3	180	140	30	92	>10000	POSITIVE	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERO SEPTAL WALL	35%	I	NEGATIVE
36	MALLIKARJUN KALLAPA	63	M	FARMER	63015	31-07-2021	05-08-3021	P	P	A	A	A	A	A	NA	P	A	P	64	110/70	37	16	9.7	4460	15	99	40	1	137	3.6	184	130	34	90	250	POSITIVE	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	50%	I	NEGATIVE
37	channayya mathad	70	M	FARMER	130610	02-08-2021	10-08-2021	P	P	A	A	P	A	P	NA	P	A	A	86	110/70	38	22	16.5	14420	25	116	17	1	136	4.3	210	140	30	98	2159	58	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	30%	I	NEGATIVE
38	SADASHIV SHIDHARAY	47	M	FARMER	130539	02-08-2021	07-08-2021	P	A	A	A	A	A	A	NA	A	A	P	84	120/70	37	16	15	8000	11	120	16	1.1	136	3.6	150	148	26	74	NEGATIVE	NEGATIVE	UNSTABLE ANGINA	SR	NO RWMA	63%	E	NEGATIVE
39	chanabasayya nandikol	42	M	BUSINESSMAN	130547	02-08-2021	07-08-2021	P	p	a	a	a	a	a	na	p	p	a	100	130/90	37	16	14.5	12506	30	112	32	0.9	140	4.3	130	80	30	66	positive	POSITIVE	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERIOR WALL	45%	I	
40	RAJASHEKAR ambigar	59	48	BANK EMPLOYEE	131809	04-08-2021	10-08-2021	P	p	a	a	a	A	p	na	a	a	a	90	180/120	37	20	13.8	15890	13	108	27	1.2	134	4.9	114	150	36	90	619	POSITIVE	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	40%	I	NEGATIVE
42	MAHADEV jambagi	57	m	CLERK	141806	13-08-2021	19-08-2021	P	a	a	a	a	a	a	na	a	a	a	66	130/80	37	16	11.9	8720	40	120	25	0.7	136	3.9	192	264	31	108	19217	POSITIVE	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	60%	I	NEGATIVE
43	NINGAPPA bheemanna	66	m	FARMER	150654	19-08-2021	25-08-2021	P	a	p	p	a	a	a	na	a	a	a	140	110/70	38	20	14.5	18000	25	78	29	0.8	136	4.1	130	92	32	80	357	POSITIVE	NSTEMI	SR	HYPOKINESIA OF ANTERIOR WALL	35%	I	NEGATIVE
44	SHANTABAI	80	F	HOUSEWIFE	95670	21-08-2021	30-08-2021	P	P	A	A	A	A	A	NA	A	A	P	80	110/70	36.5	18	12	15400	25	116	25	0.7	138	3.8	120	100	38	70	7679	POSTIVE	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERIOR WALL	45%	I	
45	PRALAD	55	M	CLERK	296570	25-08-2021	30-08-2021	P	A	A	A	P	A	A	NA	P	P	A	76	110/70	37.5	22	12.6	6570	5	110	25	0.7	134	4.6	150	110	38	60	4520	46	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERIOR WALL	45%	I	NEGATIVE
46	AKSHAY KUMAR	31	M	CLERK	196477	25-08-2021	01-09-2021	P	A	A	A	A	A	A	NA	P	P	A	100	110/80	37.5	22	11.6	10400	5	116	56	1.2	140	4.6	130	90	34	80	500	POSITIVE	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	45%	I	NEGATIVE
47	BHIMRAY padaganur	61	m	BUSINESSMAN	22209	26-08-2021	01-09-2021	P	a	a	a	a	p	a	na	a	a	p	78	126/90	37	14	14.9	10250	5	186	32	0.8	137	4.4	118	110	43	82	1131.8	POSITIVE	NSTEMI	SR	NO RWMA	60%	E	
48	RUDRAPPA	60	M	FARMER	199226	26-08-2021	02-09-2021	P	A	A	A	A	A	A	NA	P	P	A	120	110/80	37.5	22	12	15460	15	120	46	1.1	140	3.8	160	120	30	70	844	POSITIVE	STEMI-ANTERO LATERAL WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	35%	I	NEGATIVE
49	ROZUSAB	56	M	MECHANIC	169736	09-09-2021	14-09-2021	P	A	A	A	A	A	A	NA	P	A	P	78	120/80	37.5	18	14	7170	25	90	16	0.7	140	4.6	185	298	53	73	POSITIVE	376	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERO SEPTAL WALL	35%	I	NEGATIVE
50	AMBARAYYA	60	M	FARMER	173505	10-09-2021	22-09-2021	P	P	A	P	A	A	A	NA	P	A	P	90	80/50	37	22	12.7	10250	36	113	140	4.5	141	5.2	108	157	14	63	475	POSITIVE	STEMI-ANTERO LATERAL WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	30%	I	POSITIVE
51	MANSINGH naik	82	m	FARMER	201733	04-10-2021	11-10-2021	P	a	a	a	a	a	a	na	p	a	p	90	110/70	36.5	18	12.5	10500	10	130	26	1.2	136	3.1	174	200	28	60	344	POSITIVE	LBBB	SR	HYPOKINESIA OF ANTERO SEPTAL WALL	45%	E	POSITIVE
52	ASHOK	50	M	BUSINESSMAN	274185	30-11-2021	05-12-2021	P	P	A	A	A	A	A	NA	P	A	A	90	110/70	38	22	12.3	19700	10	120	34	0.7	138	5	170	90	32	84	2450	POSITIVE	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF ANTERIOR WALL	30%	I	NEGATIVE
53	VISHWANATH	57	M	FARMER	277897	02-12-2021	10-12-2021	P	p	A	A	A	A	P	NA	A	A	A	64	110/70	37	19	13.2	9000	16	108	32	0.9	140	3.8	174	110	36	84	450	POSITIVE	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	35%	I	NEGATIVE

54	KuLAPPA banikol	43	m	SHOP KEEPER	265066	02-12-2021	12-12-2021	P	a	a	a	a	a	a	na	p	a	p	90	120/70	37	16	15.2	12300	14	99	28	0.6	130	4.4	190	150	26	94	1000	POSITIVE	NSTEMI	SR	HYPOKINESIA OF ANTERO LATERAL WALL	50%	I	negative
55	dundappa katnalli	50	M	BUSINESSMAN	15458	02-12-2021	13-12-2021	P	a	a	a	a	a	a	na	a	a	p	108	140/90	37	17	11.7	12000	12	112	34	1.1	143	3.7	144	82	32	76	2230	POSITIVE	NSTEMI	SR	HYPOKINESIA OF ANTERIOR WALL	35%	I	
56	MANOHAR nistane	59	m	EMPLOYEE	286143	07-12-2021	13-12-2021	P	a	a	a	a	a	a	na	a	a	a	72	122/76	37	16	13.8	8900	12	109	23	0.9	136	4.2	140	92	30	86	POSITIVE	POSITIVE	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	45%	I	NEGATIVE
57	NEELAGANGa somanna	55	f	HOUSEWIFE	282414	07-12-2021	13-12-2021	P	p	a	a	a	a	a	na	a	a	a	80	1110/70	37	18	14.6	10400	11	98	24	0.6	137	4	158	140	36	84	2328	POSITIVE	STEMI-ANTERO LATERAL WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	55%	I	NEGATIVE
58	SADIK RAJASAHEB	54	M	MECHANIC	287286	08-12-2021	14-12-2021	P	P	A	A	A	A	A	NA	P	A	P	82	140/90	37	18	12.4	7500	5	110	18	1.1	141	3.6	130	110	40	74	NEGATIVE	NEGATIVE	UNSTABLE ANGINA	SR	NO RWMA	60%	I	NEGATIVE
59	hanamantrai jatteppa bakali	65	m	FARMER	287974	08-12-2021	13-12-2021	P	a	a	a	a	a	a	na	a	a	p	70	94/60	37	20	13.2	11600	12	98	34	1.2	132	4.2	180	150	34	90	5801	POSITIVE	STEMI-ANTERO LATERAL WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	40%	I	NEGATIVE
60	KALLAPPA komar	48	m	FARMER	274705	09-12-2021	15-12-2021	P	A	A	A	A	A	A	NA	P	A	A	70	110/70	37	14	11.7	9400	15	117	28	1	140	3.7	130	200	30	60	positive	62	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	45%	I	NEGATIVE
61	SHANKERAPPA SIDAPPA	45	M	FARMER	289513	10-12-2021	16-12-2021	P	P	A	A	A	A	A	NA	P	A	P	84	110/70	37	20	16.4	8460	23	110	30	0.8	139	4.2	140	90	30	82	4261	POSITIVE	STEMI-ANTERO LATERAL WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	40%	I	NEGATIVE
62	NINGANGONDAPPA DHARMASHEETY	70	M	FARMER	294610	13-12-2021	19-12-2021	P	P	A	A	A	A	A	NA	A	A	P	86	110/70	37	17	12	14000	10	110	27	0.9	137	5	190	160	28	90	400	POSITIVE	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	45%	I	NEGATIVE
63	HANUMANTAPPA	40	m	SHOP KEEPER	299172	16-12-2021	21-12-2021	P	a	a	a	a	a	a	na	a	a	a	56	110/80	37	12	14.6	16740	14	156	27	0.9	137	4.3	190	140	36	74	2018.4	POSITIVE	STEMI-ANTERO LATERAL WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	35%	I	NEGATIVE
64	SHABANA	35	F	HOUSEWIFE	284081	17-12-2021	24-12-2021	P	P	A	A	A	A	A	NA	A	A	A	100	110/70	37	16	13	8600	12	117	21	0.8	136	3.8	174	150	28	94	4392	POSITIVE	LBBB	SR	HYPOKINESIA OF ANTERO SEPTAL WALL	35%	E	NEGATIVE
65	GANGABAI Mashyal	65	f	HOUSEWIFE	304608	20-12-2021	25-12-2021	P	a	a	a	a	a	a	na	a	a	a	76	132/70	37	16	12	12000	12	90	22	0.7	134	3.6	150	100	32	78	2550	POSITIVE	NSTEMI	SR	HYPOKINESIA OF ANTERO LATERAL WALL	55%	I	NEGATIVE
66	RAFIQ MHD MUTTAWALI	60	M	SHOP KEEPER	304033	20-12-2021	26-12-2021	P	P	A	A	A	A	A	NA	P	A	A	80	120/70	37	16	13.6	11000	15	112	25	0.9	137	3.4	160	200	28	70	2120	POSITIVE	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF INFERIOR WALL	35%	I	NEGATIVE
67	SHRIRANGA kadam	60	m	FARMER	303688	20-12-2021	27-12-2021	P	a	a	a	p	a	a	na	p	a	p	70	120/80	37	17	15.4	7540	23	114	23	1	138	4.1	170	200	30	70	515	POSITIVE	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERO SEPTAL WALL	35%	I	NEGATIVE
68	basavaraj sahebgouda	60	M	FARMER	309659	24-12-2021	30-12-2021	P	a	a	a	a	a	a	na	a	a	p	76	140/90	37	18	13.6	10170	25	123	14	0.6	134	4.2	160	120	38	90	5414.4	POSITIVE	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERIOR WALL	40%	I	
69	SONABHAI shivappa	70	f	HOUSEWIFE	310632	25-12-2021	30-12-2021	P	a	p	p	a	a	p	na	a	a	a	84	190/100	37	20	9.7	5970	15	110	20	0.8	138	3.7	164	140	34	70	1000	POSITIVE	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	45%	I	NEGATIVE
70	gundawwa hebbal	65	F	HOUSEWIFE	311446	26-12-2021	31-12-2021	P	a	a	a	a	a	a	na	a	a	a	80	150/90	37	16	11.5	6700	15	98	23	0.5	141	2.7	135	98	37	78	712.2	23	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERIOR WALL	55%	I	
71	nagaraj kase	40	m	EMPLOYEE	9441	10-01-2022	15-01-2022	P	p	a	a	a	a	p	na	a	a	a	82	140/90	37.2	18	14.4	6530	15	123	32	0.8	143	4.3	257	267	53	151	58	POSITIVE	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	40%	I	
72	abdul rehman mokashi	75	m	BUSINESSMAN	14503	11-01-2022	15-01-2022	P	a	a	a	a	a	a	na	a	a	p	60	120/60	37.2	18	11.6	9570	24	114	34	0.9	140	4.9	170	150	40	90	>24909.0	POSITIVE	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERIOR WALL	35%	I	
73	RUKUMABAI Ramchandra	50	f	HOUSEWIFE	38301	29-01-2022	04-02-2022	P	a	p	a	a	a	p	na	a	a	a	82	150/80	37	20	13.3	9700	15	105	32	0.5	136	4.3	150	180	26	90	409	POSITIVE	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	50%	I	NEGATIVE
74	SIDDALINGAPPA adalli	61	m	FARMER	39880	31-01-2022	05-02-2022	P	a	p	a	p	P	a	na	p	p	p	64	120/70	37	18	15	11000	26	209	35	0.9	136	3.5	162	94	38	96	650	POSITIVE	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	45%	E	
75	ahmmadasha makandar	71	M	BUSINESSMAN	41281	01-02-2022	10-02-2022	P	a	a	a	a	a	a	na	a	a	p	82	100/60	37	22	14.2	7830	25	102	22	0.8	140	3.8	112	74	30	50	45	POSITIVE	RBBB	SR	RMWA	35%	E	
76	VIRUPAKSHAYYA SOMASHEKHARAYY	51	M	EMPLOYEE	43477	03-02-2022	11-02-2022	P	A	A	A	A	A	A	NA	A	A	A	80	110/70	37	18	12	9400	23	109	21	1	135	3.7	190	147	33	28	321	POSITIVE	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	40%	I	NEGATIVE
77	CHANDRAWWA SIDARAYYA	68	F	HOUSEWIFE	37003	04-02-2022	10-02-2022	P	A	A	A	P	A	A	NA	A	A	A	68	110/70	37	19	10.3	10480	25	110	28	0.8	136	5	100	90	40	60	800	POSITIVE	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	40%	I	POSITIVE
78	INDIRA guddanavar	53	f	HOUSEWIFE	49274	07-02-2022	13-02-2022	P	a	a	a	a	a	a	na	a	a	a	90	120/70	37	16	14.8	17120	15	118	15	0.7	138	3	158	140	36	80	35.1	POSITIVE	NSTEMI	SR	HYPOKINESIA OF ANTERO SEPTAL WALL	40%	I	NEGATIVE
79	HAjILAL mujawar	80	m	SHOP KEEPER	49346	07-02-2022	14-02-2022	P	a	a	a	a	A	a	na	a	a	p	70	130/70	37	16	12.4	9660	21	109	36	0.8	136	5.1	194	200	26	88	412	POSITIVE	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	40%	I	NEGATIVE
80	phAKIRAPPA maleshappa	56	m	FARMER	16626	07-02-2022	18-02-2022	P	a	a	a	a	a	a	na	p	a	p	70	110/70	37	18	13.5	9600	11	109	32	0.7	132	4.2	156	200	34	92	414	POSITIVE	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	40%	I	NEGATIVE
81	SIDDAWWA	70	F	HOUSEWIFE	49341	07-02-2022	12-02-2022	P	A	A	A	A	A	A	NA	A	A	A	60	120/70	37	22	10	12000	36	80	36	0.8	145	3.5	97	65	35	49	3422	POSITIVE	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	50%	I	NEGATIVE
82	VIKAS	45	M	CLERK	201665	08-02-2022	14-02-2022	P	A	A	A	A	A	A	NA	A	A	A	102	140/90	37.5	18	14.9	13240	5	100	46	1.5	141	4.5	128	84	36	60	POSITIVE	POSITIVE	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	45%	I	POSITIVE
83	JUMAPPA siddappa	65	m	FARMER	52366	09-02-2022	19-02-2022	P	p	a	a	a	a	p	na	p	a	p	88	140/90	37	22	12	14000	23	110	30	0.8	130	4	150	140	34	74	560	POSITIVE	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	30%	I	NEGATIVE
84	muttamma myakeri	57	f	HOUSEWIFE	56329	12-02-2022	16-02-2022	P	p	a	a	a	p	p	na	a	a	p	110	180/110	37.5	22	11	18600	75	401	20	0.7	139	4	164	112	53	88	933	POSITIVE	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	60%	E	
85	LALITHA	60	F	HOUSEWIFE	247507	04-03-2022	09-03-2022	P	A	A	A	A	A	P	NA	A	A	A	100	150/90	37.2	20	10.8	12600	40	101	30	1	134	4.1	130	90	40	74	POSITIVE	POSITIVE	STEMI-ANTERO LATERAL WALL	SR	HYPOKINESIA OF ANTERO SEPTAL WALL	35%	I	NEGATIVE
86	DYANAVVA	60	F	FARMER	47341	08-03-2022	14-03-2022	P	A	P	A	A	A	P	NA	A	A	P	76	110/70	37.5	20	12.2	10360	40	138	21	0.6	140	5	110	70	40	64	POSITIVE	POSITIVE	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	35%	I	NEGATIVE
87	LATA	50	F	HOUSEWIFE	106425	12-03-2022	23-03-2022	P	A	P	A	A	A	A	NA	A	A	A	106	90/60	37	22	10.4	8500	5	140	26	0.4	135	3.8	100	74	40	50	POSITIVE	POSITIVE	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	30%	I	NEGATIVE

88	IRANGOUDA	60	M	FARMER	80457	04-04-2022	15-04-2022	P	A	A	A	A	A	A	NA	A	A	A	90	100/70	37.5	18	16.3	11300	10	112	25	1	140	3.5	120	84	34	60	POSITIVE	105	STEMI-ANTERO LATERAL WALL	SR	HYPOKINESIA OF ANTERIOR WALL	35%	I	NEGATIVE
89	SANGAPPA	50	M	FARMER	78506	06-04-2022	12-04-2022	P	A	A	A	A	A	A	NA	A	A	P	76	110/70	37.5	16	13	10400	5	150	25	0.9	137	4.2	100	150	50	70	POSITIVE	60	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	50%	I	NEGATIVE
90	NEELAPPA	65	M	BUSINESSMAN	271179	07-04-2022	12-04-2022	P	A	A	A	A	A	P	NA	A	A	P	80	160/90	37.5	19	11	7950	5	112	36	0.7	140	4.2	224	425	38	101	POSITIVE	35	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	35%	I	NEGATIVE
91	KALAVATHI	57	F	HOUSEWIFE	289772	10-04-2022	16-04-2022	P	A	A	A	A	A	P	NA	A	A	A	98	110/80	37.4	20	13.2	9060	5	110	30	0.6	134	4.8	110	90	36	52	POSITIVE	POSITIVE	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF INFERIOR WALL	45%	I	NEGATIVE
92	IRANNA	59	M	FARMER	215458	14-04-2022	25-04-2022	P	A	P	A	P	A	A	NA	A	P	A	106	100/60	38.2	20	12.8	6160	5	112	40	0.7	142	3.6	175	75	70	90	POSITIVE	54	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERO SEPTAL WALL	45%	I	NEGATIVE
93	PARVATHI	60	F	HOUSEWIFE	196788	22-04-2022	27-04-2022	P	A	A	A	A	A	A	NA	A	A	A	80	140/90	37.5	18	10.3	10650	50	115	28	0.6	132	4.6	165	112	53	89	POSITIVE	33	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	50%	I	POSITIVE
94	SAHNKAR TOLL	50	M	CLERK	59656	24-04-2022	29-04-2022	P	P	A	A	A	A	A	NA	P	P	A	90	140/90	37.5	18	15.8	13110	10	98	36	0.8	136	4.8	146	103	38	88	POSITIVE	40	STEMI-ANTERO LATERAL WALL	SR	HYPOKINESIA OF ANTERO SEPTAL WALL	60%	I	NEGATIVE
95	MUDERYAPPA	60	M	FARMER	12920	01-05-2022	11-05-2022	P	A	A	A	A	A	A	NA	P	A	P	140	90/60	37.6	20	11.4	16600	5	140	40	0.6	132	4.6	117	96	53	45	POSITIVE	50	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	30%	I	NEGATIVE
96	BASALINGAPPA	46	M	SHOP KEEPER	18920	04-05-2022	10-05-2022	P	A	A	A	A	A	P	NA	A	P	P	80	150/90	37.5	16	14.9	9720	10	148	32	0.8	136	4.2	190	240	38	101	1330	30	STEMI-ANTERO LATERAL WALL	SR	HYPOKINESIA OF ANTERO SEPTAL WALL	45%	I	NEGATIVE
97	basavaraj shevati	63	m	FARMER	151217	06-05-2022	11-05-2022	P	A	A	A	A	A	p	NA	A	A	P	70	130/80	37	17	15.4	14300	11	110	27	1.1	138	4.4	140	150	40	68	890	POSITIVE	NSTEMI	SR	HYPOKINESIA OF INFERIOR WALL	50%	I	
98	sushilabai biradar	55	F	HOUSEWIFE	195601	08-06-2022	15-06-2022	P	A	A	A	A	A	A	NA	A	A	A	100	100/60	37	18	6.3	17000	25	130	27	0.7	137	4.3	166	333	28	71	103	POSITIVE	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	40%	I	NEGATIVE
99	CHANNAYYA	50	M	FARMER	213895	23-06-2022	30-06-2022	P	P	A	A	A	A	A	NA	A	A	A	92	170/110	37	20	14.6	21290	25	114	39	1.1	136	4.1	200	140	36	94	8800	POSITIVE	STEMI- INFERIOR WALL	SR	HYPOKINESIA OF INFERIOR WALL	30%	I	NEGATIVE
100	NINGAPPA YALLAPPA	50	M	FARMER	213895	23-06-2022	29-06-2022	P	A	A	A	A	p	A	NA	P	A	P	90	120/80	37	18	14.9	10000	25	262	39	1.1	136	4.1	180	200	26	70	300	POSITIVE	STEMI-ANTERO SEPTAL WALL	SR	HYPOKINESIA OF ANTERO LATERAL WALL	40%	E	