GENETIC STUDY OF NUCLEAR FACTOR KAPPA B1 GENE (NFKB1) POLYMORPHISM IN ACUTE CORONARY SYNDROME

By

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Dissertation submitted to BLDE (Deemed to be University),

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

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

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Dr. AMRUTA S MHASKE

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LIST OF ABBREVIATIONS

ACS	: ACUTE CORONARY SYNDROME
AMI	: ACUTE MYOCARDIAL INFARCTION
CAD	: CORONARY ARTERY DISEASE
CHD	: CORONARY HEART DISEASE
CS	: CARDIOGENIC SHOCK
cTn	: CARDIAC TROPONIN
ECG	: ELECTROCARDIOGRAPHY
HF	: HEART FAILURE
HIV-1	: HUMAN IMMUNODEFICIENCY VIRUS 1
HIV 2	: HUMAN IMMUNODEFIENCY VIRUS 2
IL 6.	: INTERLUKIN 6
INF-γ	: INTERFERON GAMMA
IKK	: INHIBITOR OF KAPPA B KINASE
LPS	: LIPOPOLYSACCHARIDE
MACE	: MAJOR ADVERSE CARDIAC EVENTS
MAPK	: MITOGEN-ACTIVATED PROTEIN KINASE
MMP	: MATRIX METALOPROTINASE
NFKB1	: NUCLEAR FACTOR KAPPA B 1 GENE
NSTEMI	: NON-ST ELEVATION MYOCARDIAL INFARCTION

NO : NITRIC OXIDE

OCT : OPTICAL COHERENCE TOMOGRAPHY

PE : PULMONARY EDEMA

PCI : PERCUTANEOUS CORONARY INTERVENTION

RWMA : REGIONAL WALL MOTION ABNORMALITY

ROS : REACTIVE OXYGEN SPECIES

STEMI : ST ELEVATION MYOCARDIAL INFARCTION

SMC : SMOOTH MUSCLE CELLS

TNFa : TUMOR NECROSIS FACTOR ALPHA

TGFh : TRANSFORMING GROWTH FACTOR H

UA : UNSTABLE ANGINA

VT : VENTRICULAR TACHYCARDIA

ABSTRACT

INTRODUCTION: Most prevalent cardiovascular condition in adults,Acute Coronary Syndrome (ACS), continue to be main source of death with morbidity worldwide. In ACS multiple environmental and genetic factors plays role.Nuclear factor kappa-B gene (NFKB), transcription factor, locates at chromosome 4q24, regulating expression of proinflammatory genes linked to atherosclerosis. This study is carried out to determine relation between NFKB1 gene polymorphism in patients of ACS.

AIM: To study genetic polymorphism of Nuclear factor kappa B 1 gene in patients with acute coronary syndrome.

MATERIALS AND METHODS: This was Prospective cross-sectional study done in BLDE (DU), Vijayapura, Karnataka, India, in 100 patients with ACS, 8 patients were excluded and 92 patients were included who underwent clinical examination, biochemical profiles, electrocardiography,blood samples were collected and analysed for NFKB1 gene polymorphism using PCR technique. Obtained data was entered into Microsoft excel sheet for analysis following which data was analysed statistically. Results were presented as Mean (Median) ±SD, counts and percentages, and diagrams.

RESULTS: This study male patients were 55 and female patients 37, commonest age group of patients were between 61-70 years, presented with chest pain ,dyspnea, risk factors included diabetes,hypertension, smoking was least common risk factor. Commonest ECG findingwas NSTEMI. This study found no mutation in exon 10 and 2 of NFKB1 gene in patients of acute coronary syndrome.

CONCLUSION: This study found no pathogenic mutation in exon 10 and 2 of NFKB1 gene in patients of acute coronary syndrome implying more research to be carried out with other pathogenic genomes and regulatory factors.

KEYWORDS: Acute coronary syndrome, nuclear factor kappa B1 gene, polymorphism, exon 10, exon 2, mutation.

INTRODUCTION

INTRODUCTION

Acute coronary syndrome is a syndrome of coronary arteries which involves various genetic and environmental factors. Inflammation significantly influences the development and advancement of atherosclerosis, suggesting that inflammatory cytokines contribute to the atherosclerotic process.[1]

One sign of coronary artery disease that has a significant hereditary component is acute myocardial infarction (AMI). Although myocardial infarction (MI) is more frequent in old age, it also becoming much more common in younger people, including those without known comorbidities. Nuclear factor kappa-B gene(NFKB), transcription factor, locates at chromosome 4q24, with 27 exons that regulates the expression of proinflammatory genes linked to atherosclerosis and regarded pathogenic through its implication in vascular inflammation, proliferation of smooth muscle cells in vessels, formation of foam cell[2]

Its response to inflammatory stimulus like Lipopolysaccharide (LPS), Reactive oxygen species (ROS), and Tumor Necrosis Factor alpha (TNFa) , NFKB transfers in nucleus gets activated, and initiates transcription of inflammation-related genes, including cytokines and chemokines, aiding in pathogenesis of chronic inflammatory diseases.[3]

NFKB, redox-sensitive transcription factor regulates battery of inflammation related genes, plays role in development of many pathological condition and is implicated crucial in initiation, progression of atherosclerosis, inflammatory bowel disease, asthma, glomerulonephritis, autoimmune arthritis, carcinogenesis.[4]

NFKB gene contains five types of proteins namely RelA (p65), RelB, c-Rel, NFKB1 (p50/p105) and NFKB2 (p52/p100). Out of five proteins heterodimers and homodimers induced, regulated via various signals, including viral, bacterial infections, growth factors, cytokines and various stressors. Resting cells, NFKB resides in cytoplasm, forming complex with I-κB keeping NFKB in inactive state, blocking translocation inside nucleus, binding of Deoxyribonucleic acid (DNA) and regulating gene expression.

Classic/Canonical and Alternative/Non-canonical are two signaling pathways for the activation of NFKB. Former gets activated via ligands of Toll like receptor (TLR), such as LPS , interleukin and TNF, or engaging with T- and B-cell receptors. Genes involved in cell proliferation, inflammation, angiogenesis, tumour metastasis are activated via this pathway. Alternative/non-canonical pathway gets activated via lymphotoxin, receptor activator of NFKB CD40 ligand, ligand and B-cell activating factor belonging to family of Tumor Necrosis Factor , playing important role in inducting genes related to development and maintenance of secondary lymphoid organs.[5-6]

Taking into consideration varying results and physiological importance of NFKB gene in atherosclerotic and inflammatory process, we conducted the present study to assess role of NFKB1 gene polymorphism in acute coronary syndrome.

AIMS AND OBJECTIVES

AIM AND OBJECTIVE OF THE STUDY

To study Genetic polymorphism of Nuclear Factor Kappa B 1 gene associated in patients with Acute Coronary Syndrome.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Unstable angina (UA), ST Elevation Myocardial Infarction (STEMI)and Non ST Elevation Myocardial infarction (NSTEMI)all are included in group of diseases known as Acute Coronary Syndrome (ACS). This particular form of CHD (Coronary Heart Disease) accounts for one-third of mortality in adults above 35year.

Aetiology

ACS is a sign of CHD and is typically caused by atherosclerosis, or plaque disruption in the coronary arteries. Male sex, physical inactivity, family obesity, smoking, high blood pressure, diabetes, hyperlipidemia, and bad eating habits are common risk factors for the illness. Vasospasm can also result from cocaine usage. Another high-risk factor is family history of MI before 55year.

Epidemiology

In the US, 15.5 million people suffer from CHD. Approximately every 41 seconds, a heart attack occurs is stated by American Heart Association . Most common cause of death in US is cardiovascular disease.[7]

Pathophysiology

The fundamental pathophysiology of ACS is a reduced blood supply to a section in heart muscle, which usually brought on by plaque rupture with thrombus development. Vasospasm, with or without underlying atherosclerosis, can occasionally cause ACS. A portion of the heart's musculature experiences reduced blood flow as a result, which first causes ischaemia and then causes myocardial infarction.

History and Physical Examination

Hallmark sign of ACS is substernal chest pain that radiates to jaw and/or left arm and is frequently described as a crushing or pressure-like sensation. This characteristic appearance is not always observed, and the primary complaints are frequently weakness, diaphoresis, nausea, epigastric pain, lightheadedness, isolated jaw or left arm discomfort, and difficulty breathing. Older age, female gender, and diabetes patients are all linked to ACS exhibiting nebulous symptoms. In these situations, a high level of mistrust is justified.

Diaphoresis and overall discomfort are frequently observed during the physical examination. Often, heart sounds are normal. There are occasionally

murmurs and gallops. The lung exam is normal, but crackles that indicate Congestive Heart Failure (CHF) may occasionally be heard. There may be bilateral leg oedema, which would indicate CHF. Unless there are co-pathologies, the remaining systems are usually within normal bounds. The presence of palpable abdominal soreness should prompt the healthcare professional to examine further conditions such as gastritis and pancreatitis. Aortic dissection should be considered when uneven pulses are present. A pulmonary emboli work-up should be warranted if there is unilateral leg swelling. In order to rule out any potentially fatal differences, a comprehensive physical examination is crucial.[8]

Evaluation

An Electrocardiogram (ECG), which aids in distinguishing between unstable angina, STEMI and NSTEMI is the initial evaluation step. According to American Heart Association guidelines, an Electrocardiogram should be performed within ten minutes of arrival for any patient exhibiting symptoms that could indicate ACS. Soon after STEMI is verified at percutaneous coronary intervention (PCI) facility, the cath lab should be turned on. When evaluating NSTEMI against myocardial ischaemia without tissue damage, cardiac enzymes like troponin and CK-MB/CK ratio-are crucial. A Chest x-ray can be used determine conditions like pneumonia and pneumothorax that appear with chest pain but are not related to MI. The same is true for blood tests that can assist distinguish between intraabdominal pathology and chest pain, such as the Liver Function Test, Complete Blood Count and Lipase. When the circumstances call for it, pulmonary emboli and aortic dissection should be kept in differential and looked into.

Treatment/Management

Anti-platelet like Aspirin, heparin boluses, and intravenousheparin infusions are first treatments for all ACS, provided there are no contraindications. It is also advised to use ticagrelor or clopidogrel as an antiplatelet medication. The decision is based on the preferences of local Cardiologist. Patients undergoing thrombolysis do not receive ticagrelor. When necessary, supportive measures are given, such as oxygen in the event of hypoxia and morphine/fentanyl for pain management. You can also use nitroglycerin infusion or sublingually to relieve discomfort. Nitroglycerine should be administered very sparingly, if at all, in situations of inferior wall ischaemia since it might produce severe hypotension. It is necessary to

continuously check the heart for arrhythmias. Whether ACS is an unstable angina or a STEMI/NSTEMI determines the course of treatment. When a STEMI patient has a door-to-procedure onset of time is less than 90 minutes, the American Heart Association (AHA) advises an emergent catheterisation and PCI. If PCI is not accessible and patient impossible to move to catheterisation lab within 120 minutes, a thrombolytic tenecteplase or another thrombolytic) is advised. The door-to-needle time for tenecteplase and other thrombolytics must be less than 30 minutes, according to AHA guidelines.

NSTEMI/Unstable Angina: Heparin and aspirin are attempted as part of the initial treatment to control symptoms. Catheterisation is advised immediately if the patient's pain persists. Effective symptom management allows for the scheduling of catheterisation and other diagnostic methods, such as myocardial perfusion studies, to be determined on a case-to-case basis based on comorbidities. Admitting cases and an emergency cardiology examination are always necessary for ACS. Further workup may also involve computerised tomography angiography, contingent on availability and cardiologist preference.

Unless there are contraindications, ACE inhibitors, statins, beta blockers should be initiated at earliest in every ACS cases. Depending on comorbidities and patient preference, those not susceptible to PCI are either handled medically or taken for coronary artery bypass grafts, or CABGs.[9]

Differential Diagnosis

- Anxiety Disorders
- Esophagitis
- Hypertensive emergencies
- Myocardial infarction
- Myocarditis
- Aortic Stenosis
- Dilated Cardiomyopathy

Emerging cause of death for adults above 35 is still coronary heart disease and acute coronary syndrome, which are still very common. When evaluating

individuals who may have ACS, healthcare professionals worldwide must use extreme caution and mistrust.

Pathophysiology of Acute Coronary Syndrome

Plaque Disruption*:* Luminal rupture of a "vulnerable" plaque is the primary and familiar event that results in an acute manifestation of coronary atherosclerosis. Among its characteristics are large lipid core combined with macrophages and foam cells. Thin fibrotic cap containing extracellular matrix components covers that atheroma. The coagulative cascade, thrombus development, and ischaemia are triggered when the protective cap ruptures acutely, releasing prothrombotic materials and chemicals from the plaque. Understanding the pathogenesis of atheroma thin cap rupture has been the subject of research for decades. Interleukin (IL)-1 and interferon-γ are two examples of inflammatory mediators that inhibit the synthesis extracellular matrix components causing macrophages, other cells produce proteases which break down extracellular molecules.[10,11]Prothrombotic components, fibrin, and plasminogen activator inhibitor-1 are produced in response to the same stimuli that break down the thin cap of the atheroma, which promotes the formation of clots.[12] Von Willebrand factor, collagen, and tissue factor are among exposed core materials that activate the circulating platelets. This intensifies the coagulative process, causing a fast thrombus to form and an abrupt onset of cardiac ischaemia.[13,14]

Plaque Erosion: The idea that ACS is caused by the surface erosion of atherosclerotic plaque is a relatively recent one. According to more recent findings, approximately 40% of ACS patients exhibit plaque erosion in the culprit lesions, whereas historical data suggests that 20% of ACS patients have this condition.[15,16]

Advances in technology, especially the Optical Coherence Tomography, have made it easier to detect this illness in vivo. Compared to those admitted for ACS because of plaque rupture, patients with plaque erosion are typically younger. According to data from a large OCT systematic analysis, patients with plaque erosion had an average lifespan of 53.8 years, while those with plaque rupture had an average lifespan of 65.1 years.[17]Between these two states, there is an imbalance in distribution of conventional cardiovascular risk factors. Patient with plaque erosion typically have greater haemoglobin concentrations, lower level of c reactive

protein and Low-Density Lipoprotein (LDL) cholesterol, and a reduced prevalence of diabetes mellitus and hypertension. Additionally, compared to individuals with plaque rupture, those with plaque erosion have less severe and complex CAD.[18]

Given the significant variations seen from a clinical perspective, it is critical to comprehend the molecular pathways in order to customize patient care and investigate novel therapeutic targets. The local shear tension associated with the plaque represents the first important concern. Studies on humans have demonstrated that areas of significant endothelial shear stress are where thromboses form. Fluid dynamic impact causes endothelial cell desquamation, basement membrane breakdown, and death. It has been proposed that innate immunity plays a role.[19] Type IV collagen , primary building block of basement membrane, is broken down byMatrix Metalloproteinase-14 (MMP) and MMP-2, which are expressed in response to inflammation and mildly oxidizedLDL.[20,21]

Calcified Nodules*:* Calcified nodules being one of less frequent causes in ACS. As the aetiology of ACS, their rates vary from 4% to 7%. People with chronic kidney illness and the elderly are more likely to have these severely calcified lesions. According to a histology analysis, the trigger might be the calcium film breaking into the lumen. From a pathological standpoint, they are distinguished by the existence of a damaged fibrous cap with an overlying thrombus and a fracture in the calcified sheet mixed with fibrin.[22] Lesions with calcified nodules have been found to exhibit negative remodeling more often than those with erosion ruptures.

Fourth Universal Definition of Myocardial Infarction[23]

In addition to aberrant biomarkers, criteria are needed to establish a diagnosis of MI. Non ischemic myocardial damage can result from a variety of cardiac disorders, including myocarditis, or it can be linked to non cardiac disorders, such as renal failure. Clinicians must therefore determine if patients with elevated cardiac troponin (cTn) values have experienced non ischemic myocardial damage or one of MI subtypes. Myocardial damage should be diagnosed in absence of data supporting existence of myocardial ischaemia. This diagnosis may be modified if further testing reveals MI criteria. These factors are reflected in current Fourth Universal Definition of MI as described in year 2018 consensus document, which follows clinical definition of Myocardial Infarction.

Myocardial Infarction Type 1

A type 1 MI is one that is brought on by atherothrombotic CAD and is typically brought on by disruption (rupture or erosion) of the atherosclerotic plaque.

Criteria for Type 1 MI

Minimum one value should be 99th percent of upper reference value and minimum one of following indicate rise or reduction in cTn values:

- Acute myocardial ischaemia symptoms;
- Fresh ischemic ECG abnormalities;
- Abnormal Q wave development;
- Imaging proof of fresh regional wall motion abnormalities or new loss of viable myocardium in pattern resembling an ischemic aetiology;
- Detection of thrombus in coronary using autopsy or angiography, includes intracoronary imaging.

Regardless of cTn values, the type 1 MI criteria are met if there is postmortem evidence of an atherosclerotic plaque in the blood supply the infarct myocardium or area of necrosis in the heart.

Myocardial Infarction Type 2

The pathophysiological mechanism known as type 2 MI occurs when the supply and demand of oxygen are out of balance, leading to ischemic myocardial damage. By definition, type 2 MI does not exhibit acute atherothrombotic plaque disruption.

Criteria for Type 2 MI

At least one of the following must be present in order to detect a spike or decline in cTn values with at least one value above 99th percentage of upper reference value, disproportionate between myocardial oxygen consumption and demand:

- Acute coronary ischaemia symptoms include
- New ECG abnormalities of ischaemia
- Abnormal Q wave
- New abnormal regional wall motion

Myocardial Infarction(MI) Type 3

Diagnosis of MI is based on identification of cardiac biomarkers in the blood. However, patients may die before blood can be drawn for the determination of cardiac biomarkers, or they may pass away shortly after the clinical manifestation, before an increase in biochemical marker has taken place. Patients may also present with the typical presentation of myocardial ischemia/infarction, including ventricular fibrillation or suspected new ischemic ECG changes.

Criteria for Type 3 MI

Patients who were died before the blood samples are taken for biochemical marker and before raising in cardiac biomarkers results , or before MI is suggested by autopsy and who had clinical features pointing towards myocardial ischaemia, are presumed new ischemic ECG abnormalities or ventricular fibrillation.

Percutaneous Coronary Intervention- related Myocardial Ischaemia (MI)-4a MI

Criteria should be less than 48 Hours Following Procedure

MI associated with coronary intervention is determined by elevating cTn readings in patients with normal baseline values by more than five times 99th percentage of upper reference value. The postoperative cTn must increase by more than 20% in patients with elevated preoperative cTn whose cTn level is constant (≤20% fluctuation) or declining. Still, at least five times 99th percentage of upper reference value the must be the absolute postprocedural value. Furthermore, one components is necessary:

New Q waves changes

New ECG alterations;

New anomalies in regional wall motion in 2D echo or new myocardial death in a pattern suggesting of an ischemic.

angiographic results that are in line with a procedural flow-limiting complication, such as distal embolization, collateral flow disruption, blockage of major epicardial artery or side branch occlusion/thrombus, or coronary dissection.

The solitary appearance of new Q waves abnormalities meets requirements for type 4a myocardial ischaemia if cTn value is high and increasing but <5 times 99th percentage of upper reference value.

Type 4b Myocardial Infarction (PCI STENT RELATED)

Elevation of cTn levels five times 99th percentage of upper reference value is an arbitrary definition of coronary intervention-related MI. The post-procedure cTn must increase by more than 20% in patients with increased pre-procedure cTn whose cTn level is steady (≤20% fluctuation) or declining. Still, at least five times the 99th percentile URL must be the absolute post-procedural value. Furthermore, one of the following components is necessary::

a. Novel ischemic ECG alterations;
b. New pathological Q waves are developed;
c. Angiogram findings that are compatible with procedural flow-limiting event include coronary dissection, blockage in major epicardial artery or side branch occlusion/thrombus, interruption of collateral flow, distant embolization.

Gene polymorphisms and risk of ACS

Gene polymorphism is existence of sequence variation in certain genomic location. The activity of a gene's product can be drastically altered by even little changes to the gene's nucleotide sequence. Under some conditions, such a change may either cause or prevent certain disease processes. The impact of genetic variation in the genes encoding the major histocompatibility complex on vulnerability to autoimmune disorders is one such instance. According to Liu et al.'s research on the connection between HLA-DQA1 polymorphism and susceptibility to IDC, 0201 variant of HLA-DQA1 protects against IDC, but 0501 allele increases susceptibility to it.[24] As a result, assessing gene variants implicated in a disease's development may be helpful for preventive as well as prognostics.

Gene polymorphism regulating Lipid metabolism and ACS risk.

It is commonly known that the severity of lipid diseases and atherosclerosis is influenced by polymorphism in the genes that control lipid metabolism (apolipoproteins, receptors, and enzymes). In the 1990s, the most frequently assessed gene polymorphism were those pertaining to the apolipoprotein E (apoE) gene. The population contains the E2, E3, and E4 alleles of apoE. The homozygote E3E3 is the most prevalent genotype. The significantly higher affinity of E2 and lower affinity of apoE4 for lipoprotein receptor are caused by the slight variation in amino acid sequence. E4E4 homozygotes (3% of the population) have polygenic hypercholesterolaemia, whereas E2E2 homozygotes (1% of the population) have

primary hyperlipoproteinemia type 3. It has been established that apoE polymorphism is associated with coronary artery stenosis and abnormalities in lipid metabolism. Additionally, there has been evidence linking the variance of the apoE gene to the risk of ACS. While E3 allele guards against ACS, the presence of E4 allele is linked to an increased risk of ACS.[25-27]

Genetic polymorphisms of inflammatory factors gene and ACS risk.

Interleukin 1h (IL-1h), interleukin-6 (IL-6), two well-known proinflammatory, prothrombotic cytokines that are implicated in pathogenesis of Acute Coronary Syndrome. Research has shown the idea of genetically driven differences in severity of inflammatory reactions in ACS patients may be supported, at least in part, by gene polymorphisms of promoter regions of IL-1h, IL-6. A significantly elevated risk of ACS has been linked to the presence of allele G of the 174G/C IL-6 polymorphism. In a similar vein, allele C of the 511C/T IL-1h polymorphism was linked to a higher risk of cardiac events in older males, but it also appears to be a predictor of a higher risk of cardiovascular and cerebrovascular events in younger people.[28] No particular results were found in studies on gene variations of cytokines involved in atherogenesis, ACS like TNFa and Transforming Growth Factor h (TGFh). In addition to humoral mediators, the inflammatory process is significantly influenced by cellular contacts and membrane receptors.

Genetic polymorphisms of endothelial factor and component of matrix and ACS risk.

Blood arteries were long thought to be a straightforward blood-supply system that was controlled by both local and systemic vasoactive agents. Recent endothelial research, however, has altered this perspective. One of the body's most active tissues, endothelium is involved in practically every aspect of cardiovascular physiology and pathophysiology. It participates in coagulation, fibrinolysis, immunological and inflammatory processes, and angiogenesis in addition to secreting vasoactive substances. The endothelium may respond differently to various activities depending on the sequence variation in the genes encoding endothelial mediators. Genetic polymorphisms encoding rein-angiotensin system results in susceptibility to arterial hypertension. In addition to its established association with poor vasodilation, the DD genotype of ACE has been shown to raise the risk of ACS.[29]However, there is

no correlation between the severity of coronary artery disease and occurrence of allele D of population. This shows that a specific kind of ACE participates in ACS due to poor regulation of vascular tension rather than the encouragement of atherogenesis.

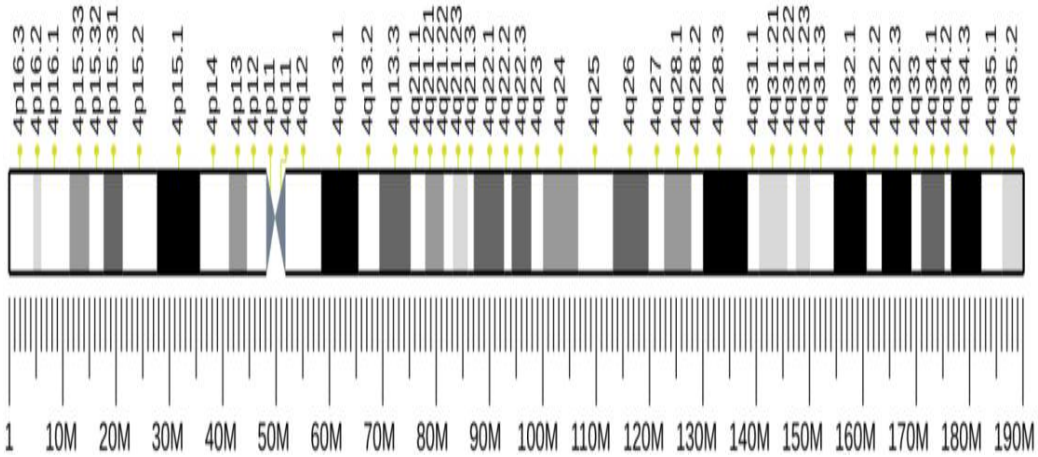


Figure1. Cytogenetic Location of NFKB1 Gene[29]

Activation of NFKB Gene

ROS, hypoxia, cytokines, Mitogen-Activated Protein Kinase (MAPK) activators, protein C kinase activators, bacterial, like LPS, dsRNA, or human T-cell leukaemia virus type 1 Tax protein, can all activate nuclear factor kappa-B. Following Figure 2 shows a schematic representation of NFKB activation. Members in NFKB family—p50, p52, p65 (RelA), c-Rel, and RelB-forms homo- and heterodimers, with p50 or p52/RelA heterodimer being the most prevalent active form. In cells at rest, NFKB dimers found in cytoplasm in inactive state, attached to inhibitory proteins called IκB. The NFKB dimer's activity is regulated by at least six IκB proteins. Two N-terminal serine residues on the two NFKB

stimulus-regulatory proteins, IκBα and IκBβ, are phosphorylated in response to various stimuli. Following phosphorylation, the IκBs undergo proteolytic degradation and ubiquitination. After being activated by this process, NFKB moves into nucleus, attaches itself to the promoter or enhancer regions of particular genes to start transcription.[30]

Phosphorylation of IκBs by 700–900 kDa multimeric complex known as IκB Kinase (IKK) complex is essential step in activating NFKB. NFKB essential modulator also known NEMO IKKγ, IKK-associated protein, or FIP-3, and two

catalytic subunits (IKK1/IKKα and IKK2/IKKβ) make up IKK complex. NEMO facilitates important

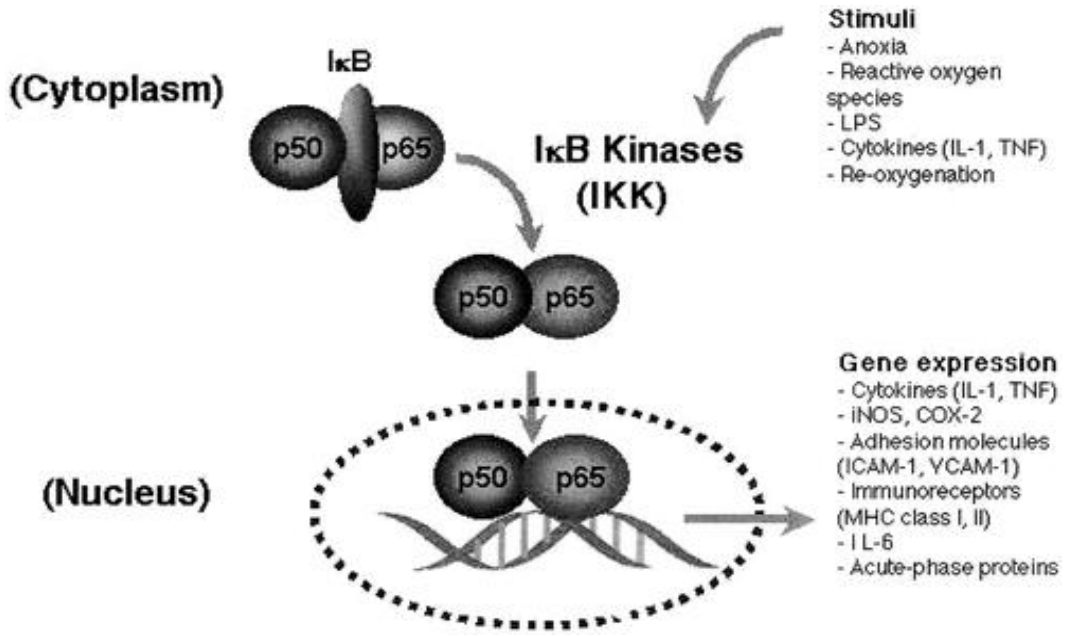


Figure 2. Depicts illustration of NFKB gene activation.[30-33]

protein-protein interactions such as upstream activators of kinase complex, but not being a kinase in and of itself. Upstream kinases, such as NFKB-Inducing Kinase (NIK) and MEKK1 of the MAP3K family, phosphorylate either IKKα or IKKβ to activate the IKK complex. Then, by phosphorylating serine residues, the IκB is drawn into the IKK complex. Although IKKα is essential for keratinocyte differentiation, cytokine-dependent activation of NFKB does not require it. On the other hand, IKKβ is necessary for pro-inflammatory stimuli to activate NFKB.[30-33]

Role of NFKB in atherosclerosis and unstable coronary syndromes

NFKB contributes in development of atherosclerosis, by converting pathogenic stimulus into gene expression. Atherosclerotic lesions have been found to include NFKB-regulated inflammatory mediators, including leukocyte adhesion molecules, cytokines, and inducible nitric oxide (NO) synthase. In human atherosclerotic plaques, activated NFKB seen in endothelial, smooth muscle cells, macrophages, but not healthy arteries. When pathogenically significant substances, such as ROS implicated in low-density LDL oxidation and elements bacteria like Chlamydia

pneumonia, stimulate lesion cells, NFKB may be activated. The finding that mice lacking NFKB signalling show less fatty-streak development when given a fatty diet lends credence to this theory.

It is generally thought that local events, which can have an infectious, immunologic, or general inflammatory aetiology, induce an atherosclerotic plaque to rupture, resulting in unstable coronary syndromes.[34,35] Several NFKB-regulated genes, including tissue factor, coagulation initiator, interlukin-1(IL-1), TNF-α, interferon-gamma(INF-γ), and IL-6, NO synthase, are expressed more frequently in plaques from individuals with unstable coronary syndromes. Acute-phase reactant and indicators of leukocyte activation are all elevated in these patients, indicating a systemic inflammatory response.

NFKB1 Gene Rs28362491 Polymorphism-Association with Acute Coronary Syndrome

The NFKB1 gene's rs28362491 (-94ATTGins/del) polymorphism has been linked in the past to a number of inflammatory illnesses, including systemic lupus erythematosus, ulcerative colitis, and Grave's disease.[36-38]

Boccardi V in 2011, did a study on Caucasian population in Southern Italy, in 253 cases of which 86 patients had CAD and 167 were healthy control group. Genotyping of 253 cases was done for 3′UTR A/G (rs696) allelic variant of NFKBIA gene and -94 ins/del ATTG (rs28362491) NFKB1 polymorphism. In 3′UTR A/G polymorphism frequency of AA, AG and GG genotypes was 16.8%, 46.8% and 36.4%, respectively. While in -94 ins/del ATTG polymorphism, frequency of II, ID and DD genotypes was 41.5%, 45.5% and 13%, respectively.Patients affected with MI were from older age group, had deranged lipid profile and high plasma fibrinogen levels compared to controls. This study found varying results were D allele frequency were higher in control group compared to MI cases and D carriers had significant low fibrinogen and CRP level compared to D non carriers which implies that -94 ins/del ATTG polymorphism causes lower MI susceptibility.[39]

Study in 2013 conducted by Avshesh Mishra et al, in two cohort group consisting 600 CAD cases and 230 controls in which 197 males and 33 females. Patients with reduced LVEF<45% were categorized to have Left ventricular dysfunction. The

NFKB1-94 ATTG ins/del (rs28362491), IL6-174 G/C (rs1800795) and TNF-a-308 G/A (rs1800629) polymorphisms were geno typed by PCR/ARMS-PCR methods. The study found that NFKB1-94 ATTG ins/del polymorphism was significantly associated with MI, reduced LVEF with LV remodelling i.e. LV mass and LV dimensions were deranged. [40]

Hong Mei Lai et al, in 2015 conducted case control study among 960 CAD Uygur patients (Stable angina pectoris [SAP]-680, ACS-280) and 1060 controls.Using TaqMan SNP Genotyping assay, all participants were typed for NFKB1 and NFKBIA gene polymorphisms (SNP rs 28362491, SNP rs696). 360 SAP And 360 Controls were randomly assigned for measuring IL-6 levels. In subgroup of SAP, NFKB1 del/del genotype was significantly associated with increased risk of SAP for males and females (for males, OR = 1.538, $P = 0.018$; for females, OR = 1.650, $P = 0.037$). In ACS subgroup, male group with NFKB1 del/del genotype had 1.620-fold increased risk of developing ACS and no female association of NFKB1-94ins/del ATTG polymorphism was observed. Further mean interlukin 6 values were higher in SAP subgroups then controls indicating its role in reducing NFKB1 promoter activity caused by deletion of ATTG repeat in the promoter region of NFKB1 gene and the consequent effects of increased p65/p50 heterodimer on IL-6 gene transcription.[41]

Shreedhar naik et al, in 2018 carried out study in 100 ACS cases and 100 controls in South India. Patients presenting with STEMI, NSTEMI, UA and Chronic stable angina above 18 years were included in this study. The exon 2 of NFKBIL1gene, located in active site of NFKBIL1gene was PCR amplified and was sequenced to compare with control sequence. Results of NFKBIL1gene sequence analysis showed 4patients having novel missense mutations, which resulted in structural and functional changes in transcription factor increasing risk of MI in study group.[42]

Eliecer Coto et al, did a case control study in 2019, in 609 CAD patients and 423 healthy subjects. Only male patients with age <55 years were included in the study based on first episode of CAD and then undergoing coronary angiogram which showed involvment of atleast one atherosclerotic coronary artery with luminal narrowing of >70%. Three variants in NFKB1 , NFKBIA , and NFKBIZ genes were genotyped, out of which NFKBIZ was seen associated with early onset CAD < 55

years by regulating IL-6 production NFKB1 promoter indel has been linked to the risk for CAD.

However, no significant association was detected between CAD andNFKB1 , NFKBIA variants.[43]

Study done by Jun-Yi-Luo et al in 2019, aimed to explore relationship between NFKB1 gene rs28362491 and MI by polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) in 359 MI patients and 1085 controls. Gensini score was used to calculate the degree of coronary artery stenosis in MI patients. The study found frequencies of D allele and DD genotype were high in MI cases compared to controls. Comparing to II and ID genotype carriers, gensini score was 32-43% higher in MI patients with DD genotype. Moreover, DD genotype was found to be associated with high levels of interlukin 6 and diseased coronary arteries.[44]

MATERIALS AND METHODS

MATERIALS AND METHODS

SOURCE OF DATA

This study was done in Shri B M Patil Medical college, Hospital and Research Centre, Department of General Medicine, BLDE (DU),Vijayapura from May 2023 to December 2024 on 100 patients admitted with ACS of which 92 patients were included and 8 patients were excluded based on inclusion and exclusion criteria. Study was conducted after approval from institutional ethical committee (IEC No-909/2023-24). Patients in study were explained about procedure in detail following that consent was taken for same.

Study Design: prospective cross-sectional study.

Sample size calculation

Considering the confidence limit of these studies to be 95% with 5% level of significance and margin of error 0.05. The sample size computed using the following formula Sample size (n) = (Z2 *p*(1-p)) /d2

Z is z score=1.96, margin of error=0.05, n=population size

P is the population proportion=0.06

The estimated sample size of this study is 87.[44]

PATIENT SELECTION

A. INCLUSION CRITERIA

Patients of acute coronary syndrome above age of 18 years

B. EXCLUSION CRITERIA

i. Patients with underlying valvular heart disease.
ii. Patients with congenital heart disease.
iii. Patients with ischemic cardiomyopathy.

METHODOLOGY:

INITIAL ASSESSEMENT

Patients who presented with prolonged chest discomfort typical of myocardial ischemia, underwent standardized assessment with detailed history, clinical examination, investigations like electrocardiogram, cardiac enzyme –Troponin I and coronary angiogram (if required) on admission were taken along with 1ml of blood sample of the patient for analysis of NFKB1 gene polymorphism.

DETECTION OF NFKB1 POLYMORPHISM

The blood samples collected from the patients of acute coronary syndrome are processed as explained below in figure 3 and gene sequencing is performed. Based on the results of gene sequencing patients were grouped according to the presence or absence of NFKB1 gene polymorphism.

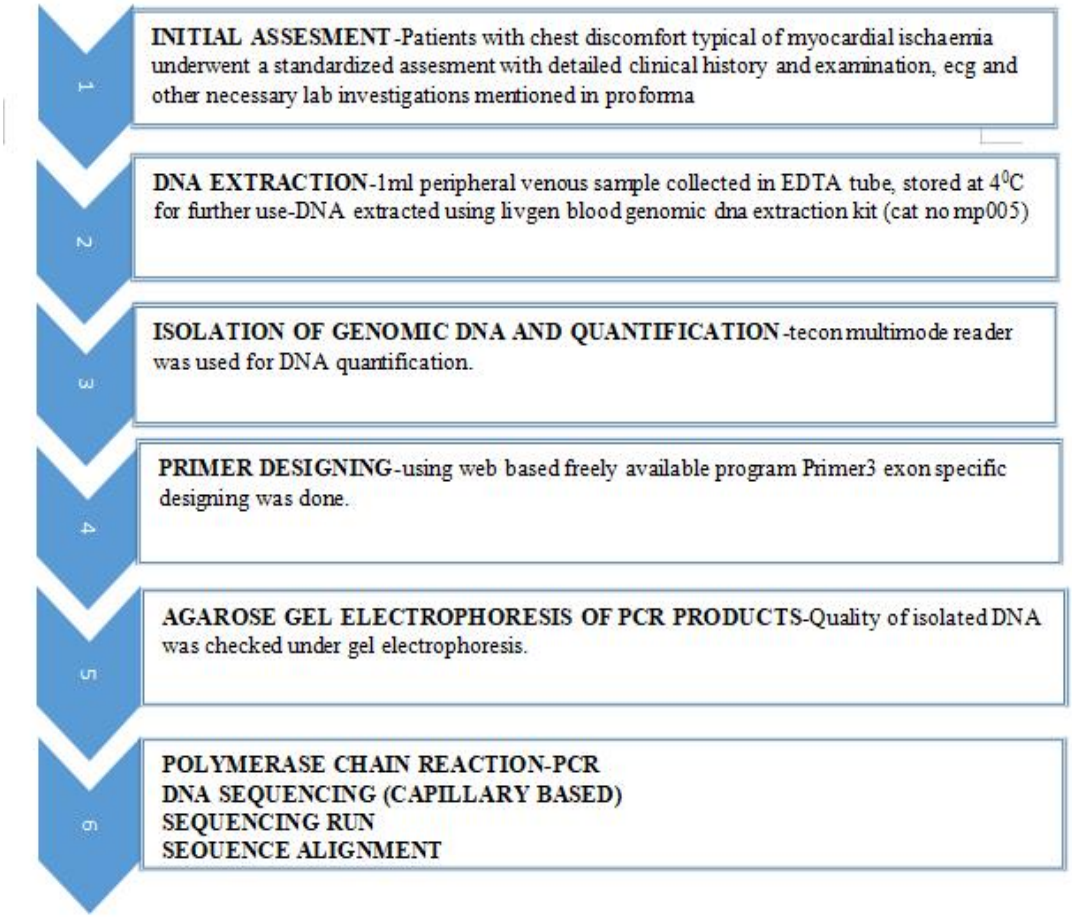


Figure 3. depicts process of gene sequencing from collected blood samples

DNA EXTRACTION: 1 ml of peripheral venous blood samples were collected and stored in the EDTA coated vacutainers and stored at 4^0 C for further use.Genomic DNA extraction from ACS samples was extracted using livgen blood genomic DNA extraction kit. (cat no mp005).

ISOLATION OF GENOMIC DNA AND DNA QUANTIFICATION: Genomic DNA was isolated from the extracted DNA samples and processsed for DNA quantification.DNA quantification was performed using Tecon multimode reader.

PRIMER DESIGNING: Widely accepted web based freely available program "Primer3" was used, (http://frodo.wi.mit.edu/ primer3/ input. Html) for designing PCR primers.Designed primers for our target genes or region arePrimer name NF10 EXON with forward primer AAT GAA AGT TGG GGC GCA TT and reverse primer TGA TTG TAC CAC TGC ACT GC with product size 388bp. Primer name NF2 EXON with forward primer CCC AAG AGT TCC ATG GCC TC and reverse primer CAT CTG GGC CCT GGC GGG CA with product size 277bp.

AGAROSE GEL ELECTROPHORESIS OF PCR PRODUCTS:
Gel electrophoresis separates DNA and RNA depending on the length of fragments-An electric field is used to separate the positive and negatively charged molecules of nucleic acid and they are transported through an agarose matrix. Shorter molecules may pass through the gel's pores more readily so they travel farther. The quality of the isolated DNA was checked under gel electrophoresis.

100 ml of 1% agarose gel was prepared (1gm of Agarose + 100 ml of 1X TAE buffer).The genomic DNA fragment separation by size from patients of acute coronary syndrome, lane number 1-38 samples is shown in figure 4.

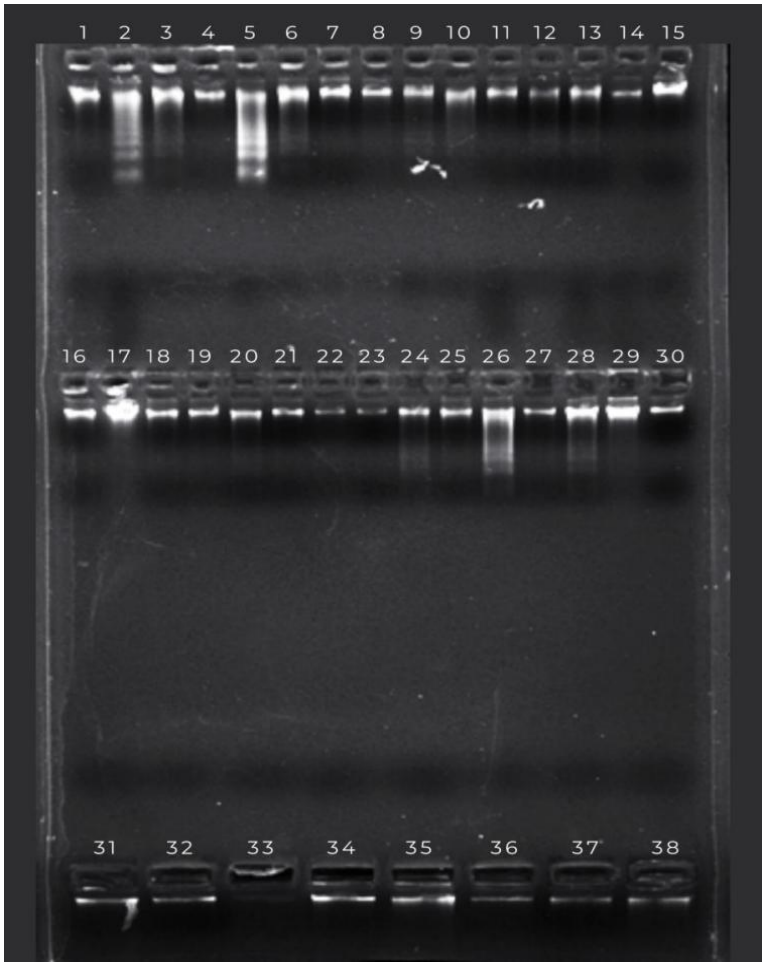


Figure 4. Agarose gelelectrophoresis of acute coronarysyndrome patients.

POLYMERASE CHAIN REACTION (PCR): PCR amplification was carried out. The following were the conditions for PCR cycling: First step is denaturation at 95 degrees Centigrade for five minutes, followed by primer-dependent annealing at temperature 56 degrees centigrade for ten seconds, elongation at 72 degrees centigrade for one minute, final extension at 72 degrees centigrade for five minutes and hold at 40 degree centigrade.

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. 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.

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 1: Standardized master mix conditions for sequencing.

Process	Temperature (°C)	Time
Initial. Denaturation	98	10sec
Denaturation	98	10sec
Annealing	60	10sec
Elongation	72	5min
Renaturation	72	5 mins
Hold	4	

Table 2: The cycle sequencing conditions

Note: The annealing temperature is primer dependent , varies for each primer

SEQUENCING RUN

Sample information sheets containing analysis protocols and the sample details were prepared and imported into the data collection software. These samples were analyzed on ABI 3730 genetic analyser (Applied Biosystems, USA) to generate DNA sequences or electropherograms.

SEQUENCE ALIGNMENT

Using Variant reporter software (ABI v1.1), produced sequences were aligned to corresponding reference sequences then screening of sequences was done to look for familiar variants like deletions, insertions and new mutations Here, we used this technique to check the isolated genomic DNA from whole blood. In all the 92 acute coronary syndrome samples the presence of genomic DNA was confirmed and the same samples were taken for quantification based on Nanodrop.

STATISTICAL ANALYSIS

The data obtained is entered in a Microsoft Excel sheet, and statistical analyses are performed using a statistical package for the social sciences (SPSS) (Version 20). Obtained results are presented as Mean, Standard deviation, counts and percentages, and diagrams.

RESULTS

RESULTS

Total of one hundred patients presenting with typical anginal chest pain, dyspnea were screened in the study. Out of which ninety two patients were included based on inclusion criteria and eight patients were excluded based on exclusion criteria in which five were valvular heart disease and three were ischemic cardiomyopathy. Included ninety two cases underwent NFKB1 gene analysis to look for genetic polymorphism in exon 10 and 2 of NFKB1 gene of which all ninety two cases showed no mutation in both targeted exons. Following figure 5. depicts the distribution of study population.

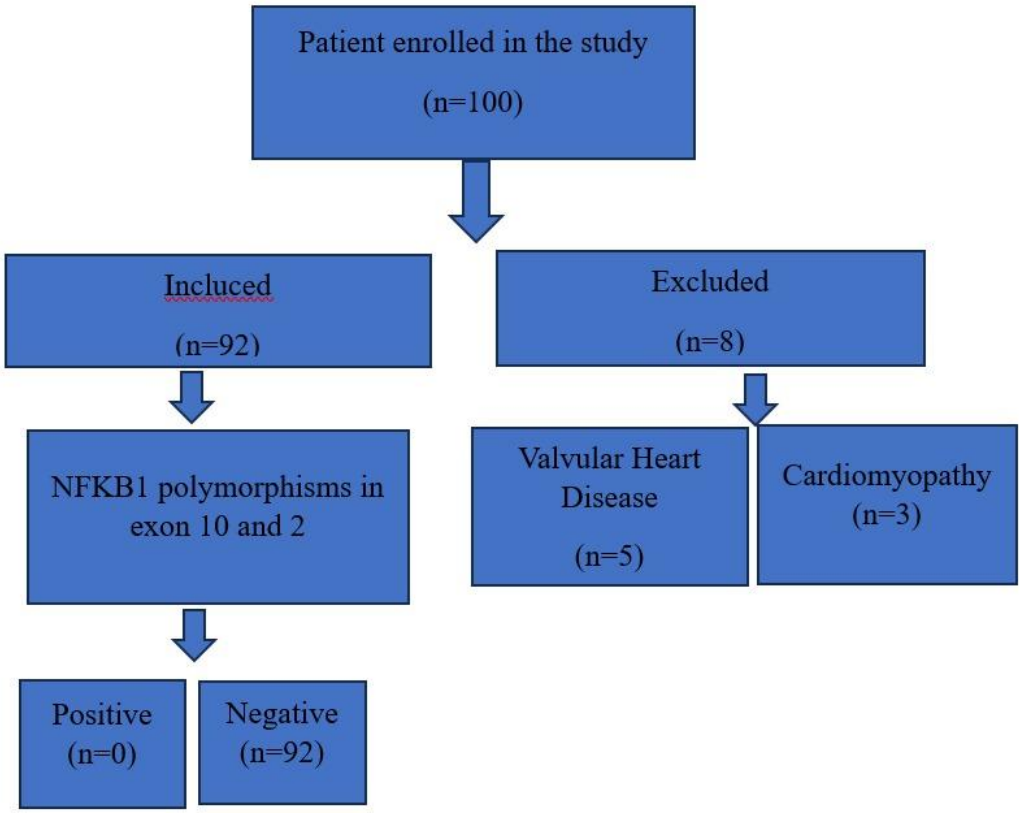


Figure 5. Flowchart depicts distribution of study population in the study.

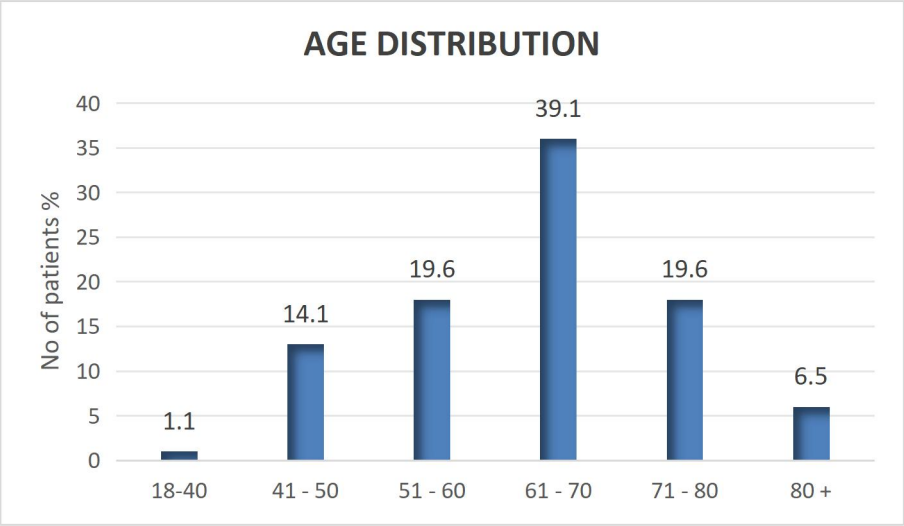
DISTRIBUTION OF PATIENTS ACCORDING TOAGE

All ninety two patients were grouped according to age in years. Following age distribution table 3 and graph 1 shows that one patient was between age 18-40 years age (1.1%), thirteen patients in age group 41-50 yrs (14.1%), eighteen patients included in age group of 51-60 yrs (19.6%),thirty six patients in age

group 61-70yrs, eighteen in age group 71-80yrs (19.6%), six patients in age group above 80yrs (6.5%).

Age (Years)	No.of patients (n=92)	Percentage %
18-40	1	1.1
41 - 50	13	14.1
51 - 60	18	19.6
61 - 70	36	39.1
71 - 80	18	19.6
80 +	6	6.5
Total	92	100.0

TABLE 3. DISTRIBUTION OF PATIENTS ACCORDING TOAGE



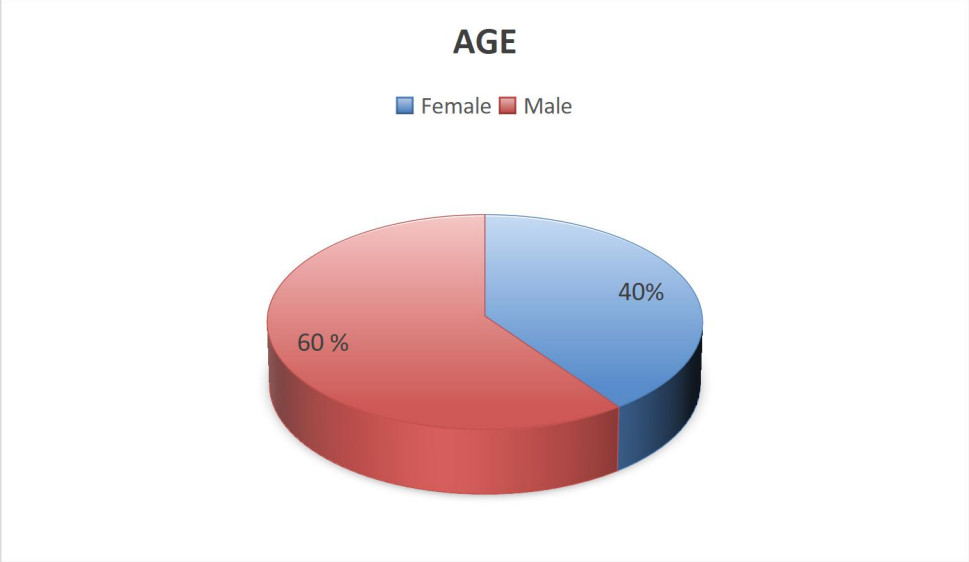
GRAPH 1: DISTRIBUTION OF PATIENTS ACCORDING TO AGE

SEX DISTRIBUTION IN THE STUDY POPULATION

Following table 4 and pie chart 2 depicts, age distribution among study group shows that 55 patients (60%) were men and 37 patients (40%) were women. Hence, male patients were more than female patients

GENDER	No. of patients (n=92)	Percentage %
Male	55	60
Female	37	40
Total	92	100

TABLE 4: SEX DISTRIBUTION IN THE STUDY POPULATION



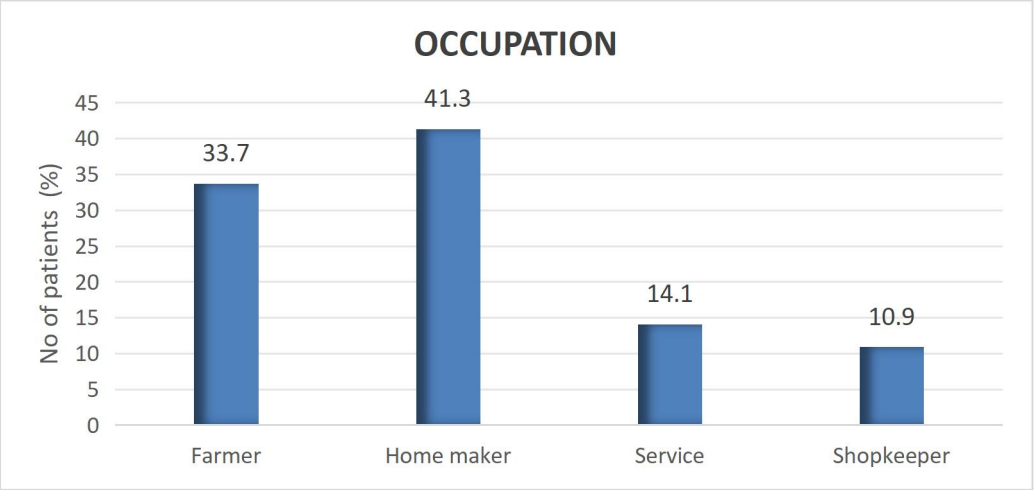
GRAPH 2: SEXDISTRIBUTION IN THE STUDY POPULATION

DISTRIBUTION OF PATIENTS ACCORDING TO OCCUPATION

The table 5 and graph 3 below shows occupational distribution in 92 patients with 31 patients (33.7%)were farmer, 38 patients (41.3%) were homemaker, 13 patients (14.1%) were servicemen, 10 patients (10.9%) were shopkeeper. In this study population homemaker were highest followed by farmer then servicemen and then shopkeepers.

OCCUPATION	No of patients (n=92)	Percentage %
Farmer	31	33.7
Home maker	38	41.3
Service	13	14.1
Shopkeeper	10	10.9
Total	92	100.0

TABLE 5: DISTRIBUTION OF OCCUPATION IN STUDY POLULATION



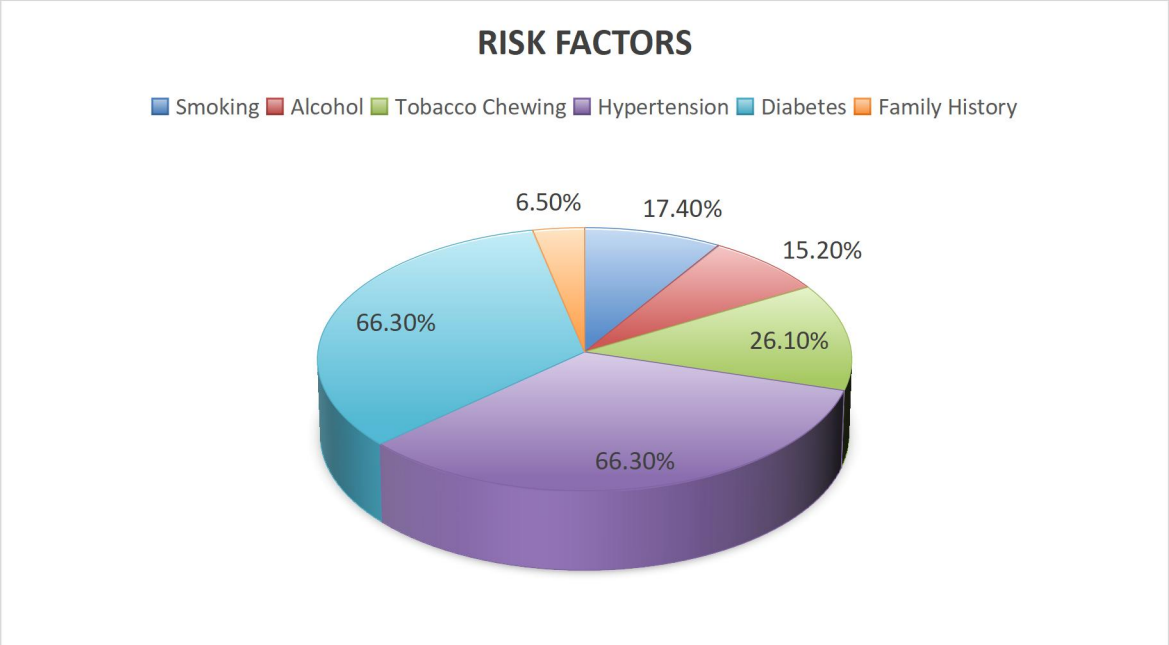
GRAPH 3: DISTRIBUTION OF OCCUPATION IN STUDY POPULATION

DISTRIBUTION OF PATIENTS ACCORDING TO RISK FACTORS

The following table 6 and graph 4 shows that, 61 patients (66.3%) were diabetic, hypertensives were 61 patients (66.3%).Patients with positive family history were 6 (6.5%).Smoking comprised 16 patients (17.4%) while in alcohol consumption 14 patients (15.2%) were present, tobacco chewing comprised 68 patients (73.9%).

RISK FACTORS	No of Patients (n=92)	Percentage %
Smoking	16	17.4
Alcohol	14	15.2
Tobacco Chewing	24	26.1
Hypertension	61	66.3
Diabetes	61	66.3
Family History	6	6.5

TABLE 6: DISTRIBUTION OF RISK FACTORS IN STUDYPOPULATION



GRAPH 4: DISTRIBUTION OF RISK FACTORS IN STUDY POPULATION

DISTRIBUTION OF PATIENTS ACCORDING TO SYMPTOMS

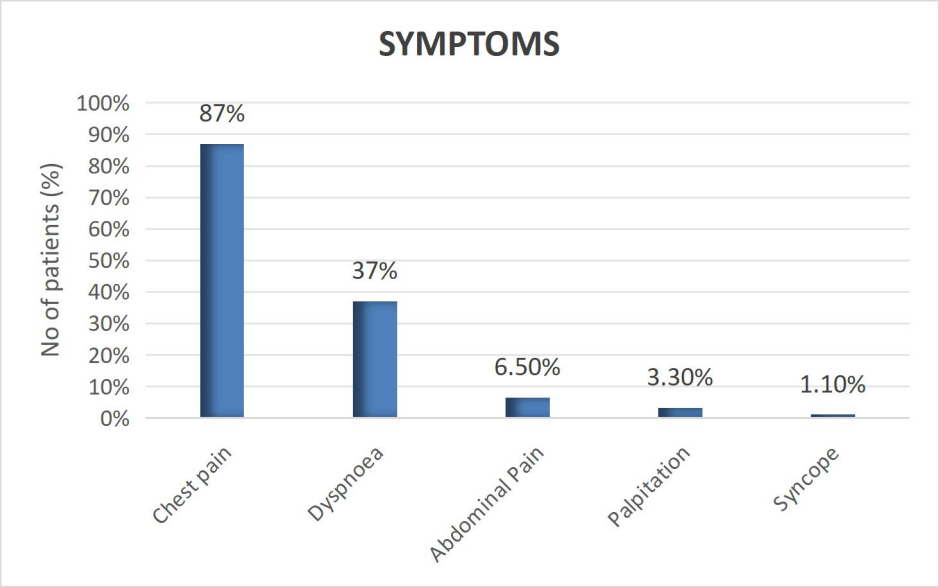
Following table 7 and graph 5 depicts various symptoms in this study population in which chest pain was seen in 80 patients (87%) followed by dyspnea seen in 34

patients (37%), abdominal pain seen in 6 patients (6.5%), palpitations seen in 3 patients(3.3%) while syncope in 1 patient (1.1%).

Out of the above symptoms, the most common was chest pain followed by dyspnea then abdominal pain and least common was palpitations and syncope.

SYMPTOMS	No. of patient (n=92)	Percentage %
Chest pain	80	87
Dyspnoea	34	37
Abdominal Pain	6	6.5
Palpitation	3	3.3
Syncope	1	1.1

TABLE 7. DISTRIBUTION OF SYMPTOMS IN STUDY POPULATION



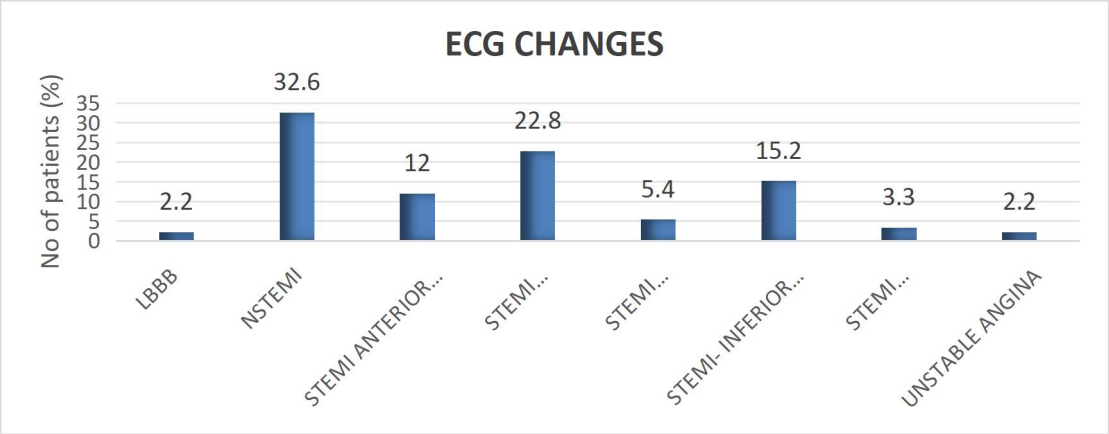
GRAPH 5: DISTRIBUTION OF SYMPTOMS IN STUDY POPULATION

DISTRIBUTION OF PATIENTS ACCORDING TO ECG FINDINGS

Following table 8 and graph 6 shows that on ECG out of 92 patients, 21 patients had ST Elevation MI (STEMI) Anteroseptal wall (V1,V2,V3,V4) , 30 patients had Non ST Elevation MI (NSTEMI), 14 patients had STEMI Inferior wall ((II,III, avf), 11 patients had STEMI Anterior wall (V3,V4), 5 patients showed STEMI anterolateral wall (V1-V6), 3 patients showed STEMI inferolateral wall leads (II,III, avf, v5,v6), 2 patients had LBBB, 2 patients had unstable angina.

ECG	No of patients (n=92)	Percentage %
LBBB	2	2.2
NSTEMI	30	32.6
STEMI ANTERIOR WALL	11	12
STEMI ANTEROSEPTAL WALL	21	22.8
STEMI ANTERIOLATERAL WALL	5	5.4
STEMI- INFERIOR WALL	14	15.2
STEMI INFEROLATERAL WALL	3	3.3
UNSTABLE ANGINA	2	2.2
TOTAL	92	100

TABLE 8: DISTRIBUTION OF ECG FINDINGS IN STUDY POPULATION



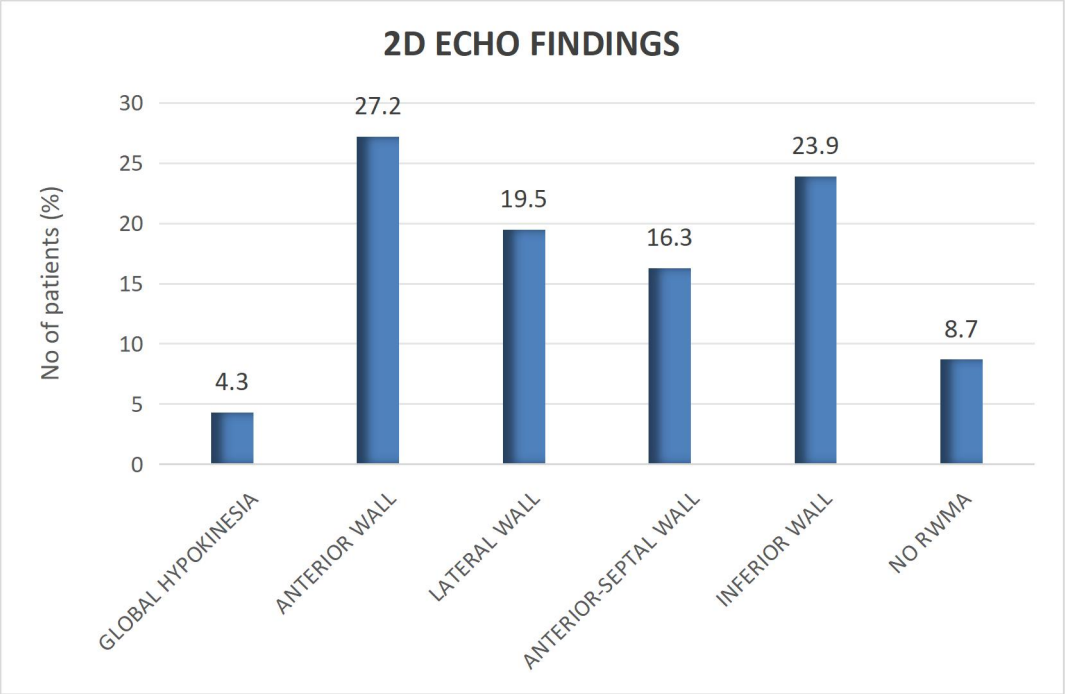
GRAPH 6: DISTRIBUTION OF ECG FINDINGS IN STUDY POPULATION

DISTRIBUTION OF PATIENTS ACCORDING TO ECHOCARDIOGRAPHIC VARIABLES:

Table 9 and graph 7 below shows echo cardiographic changes among 92 patients of which 25 patients (27.2%) showed hypokinesia of anterior wall, 18 patients (19.5%) showed hypokinesia of lateral wall, 22 patients (23.9%) showed hypokinesia of inferior wall, 15 patients (16.3%) showed hypokinesia of anterior and septal wall, while 4 patients (4.3%) showed global wall hypokinesia, 8 patients (8.7%)were found to have no motion wall abnormality.

REGIONAL WALL MOTION ABNORMALITY	**No ofpatients n=92**	**Percentage%**
GLOBAL HYPOKINESIA	4	4.3
ANTERIOR WALL	25	27.2
LATERAL WALL	18	19.5
ANTERIOR-SEPTAL WALL	15	16.3
INFERIOR WALL	22	23.9
NO RWMA	8	8.7
Total	92	100

TABLE 9: DISTRIBUTION OF REGIONAL WALL MOTION ABNORMALITY IN STUDY POPULATION



GRAPH 7: DISTRIBUTION OF REGIONAL WALL MOTION ABNORMALITY IN STUDY POPULATION

DISTRIBUTION OF NFKB1 GENE POLYMORPHISM IN STUDY POPULATION:

QUANTIFICATION OF GENOMIC DNA-DNA quantification was performed using Tecon multimode reader. 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 10.

Sl.No. of DNA samples	OD at 260/280	Concentration in ηg/µl		Sl.No. of DNA samples	OD at 260/280	Concentration in ηg/µl
1	1.86	54		47	1.83	47.2
2	1.75	65		48	1.59	53.2
3	1.40	44		49	1.62	68.2
4	1.90	70		50	1.54	78.1
5	1.57	136		51	1.63	79.2
6	1.98	64		52	1.58	89.1
7	1.84	82		53	1.92	95.2
8	1.92	73		54	1.85	45.3
9	1.65	68		55	1.74	65.1
10	1.79	111		56	1.65	69.4
11	1.85	64		57	1.52	74.2
12	1.81	66		58	1.51	53.2
13	1.75	53		59	1.57	58.1
14	1.59	65		60	1.59	79.2
15	1.66	82		61	1.64	78.1
16	1.51	94		62	1.83	88.2
17	1.88	49		63	1.82	89.1
18	1.92	39		64	1.83	75.1
19	1.93	46		65	1.74	95.1
20	1.74	100		66	1.73	115.2
21	1.65	51.5		67	1.54	96.7
22	1.89	85.5		68	1.76	58.4
23	1.96	73.9		69	1.74	55.6
24	3.05	57		70	1.63	79.2
25	2.01	81		71	1.64	81.2
26	2.24	125		72	1.56	75.2
27	2.09	137		73	1.91	1.5.3
28	1.76	104		74	1.93	112.0
29	1.96	92		75	1.74	145.2

30	1.58	93		76	1.85	49.2
31	1.86	54		77	1.81	56.8
32	1.75	65.4		78	1.49	78.5
33	1.40	44.5		79	1.55	64.2
34	1.90	70.3		80	1.66	86.2
35	1.57	95.3		81	1.74	69.2
36	1.98	64		82	1.72	54.9
37	1.84	82		83	1.71	58.1
38	1.92	73		84	1.73	75.1
39	1.65	68		85	1.78	67.2
40	1.79	111		86	1.79	57.1
41	1.85	64		87	1.80	45.2
42	1.81	66		88	1.70	47.2
43	1.68	54.2		89	1.76	63.2
44	1.85	63.5		90	1.84	89.2
45	1.74	56.2		91	1.83	87.1
46	1.56	48.2		92	1.83	47.2

TABLE 10: shows quantification of genomic DNA.

AGAROSE GEL ELECTROPHORESIS OF PCR PRODUCT

Sequencing is done as per Sanger sequencing protocol, Big Dye labeling and chain termination is done for both exons 10 and 2 of NFKB1 gene in acute coronary syndrome patients. The PCR products of exon 10 with base pair 388 and lane number 1-38 is shown in figure 6 and exon 2 with base pair 277 and lane number 1-37 and lane number 38-80 is shown in figure 7 and 8.

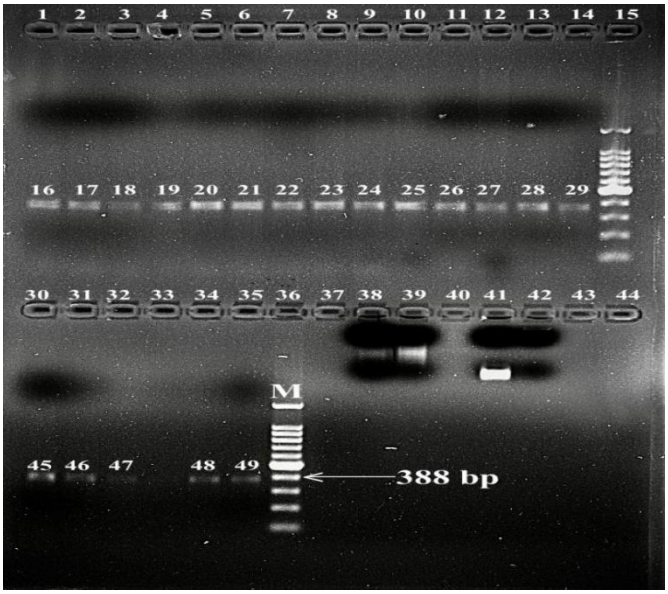


Figure 6. Agarose gel image of PCR products of exon 10 inNFKB1 gene

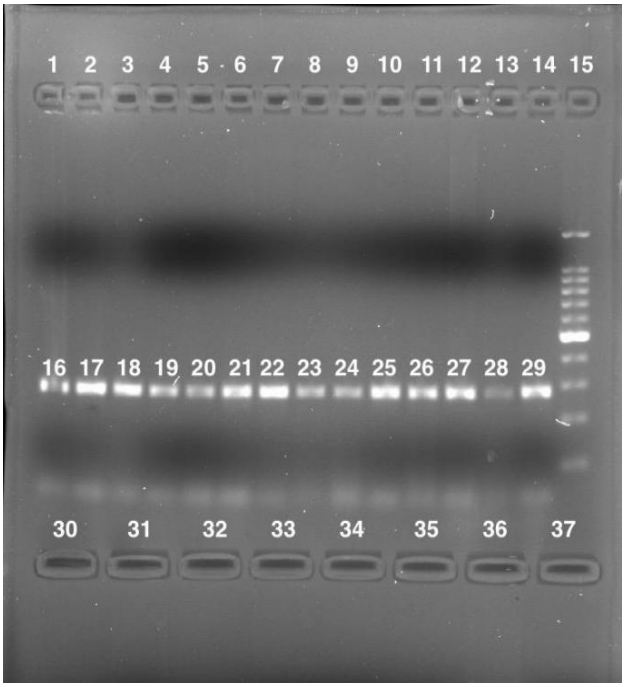


Figure 7. Agarose gel image of PCR products of exon 2 inNFKB1 gene.

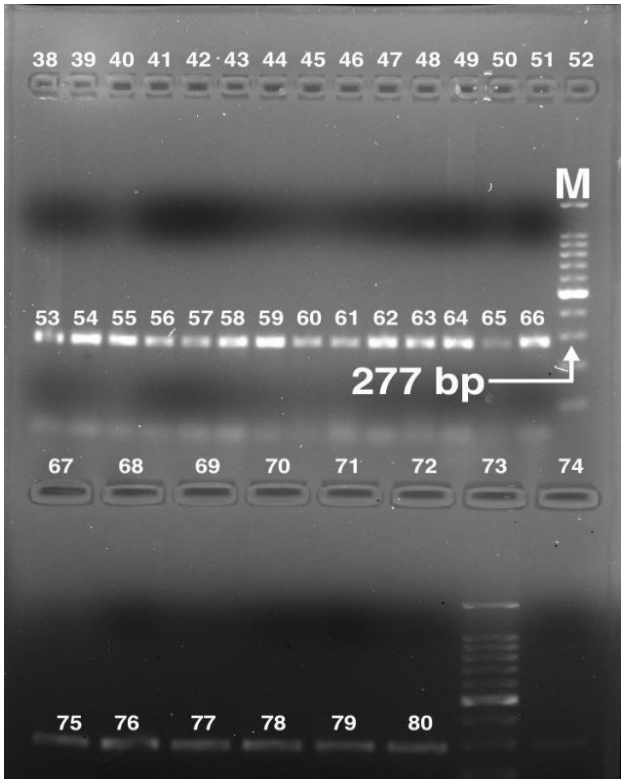
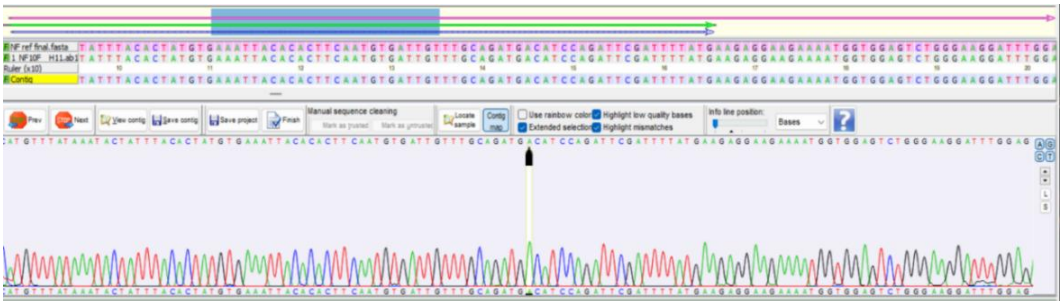


Figure 8.Agarose gel image of PCR products of exon 2 inNFKB1 gene.

An electropherogram was obtained for showing results of gene sequence analysis of exon 10 and 2 in NFKB1 gene. Following electropherograms shows DNA Sanger sequencing, black arrow indicates specific targeted region cytosine C which showed no pathogenic mutation in targeted exon 10 and 2 of 92 ACS samples as shown in figure 9.

Targeted region: 10th exon

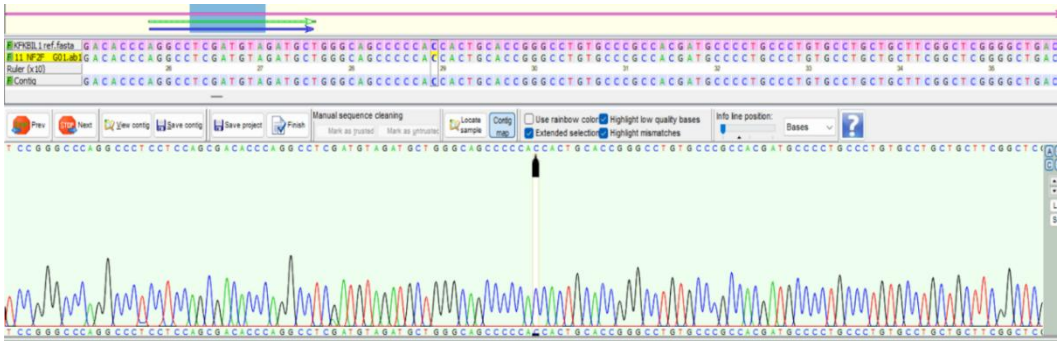


S1:No mutation detected in sequence analysis of exon 10 of NFKB1 gene.

S10: No mutation detected in sequence analysis of exon 10 of NFKB1 gene.

Targeted region: 2nd exon

S7. No mutation detected in sequence analysis inexon 2 of NFKB1 gene



S11. No mutation detected in sequence analysis inexon 2 of NFKB1 gene

Figure 9. Electropherogram

DISTRIBUTION OF NFKB1 GENE POLYMORPHISM IN STUDY POPULATION

Following table 11 shows results of genetic testing for mutations in NFKB1 gene in exon specific target 10 and 2 shows all ninety two patients of ACS had no genetic mutation detected.

NFKB1 gene mutation in exon 10 and exon 2	No. of Patients (n=92)	Percentage (%)
PRESENT	0	0
ABSENT	92	100
TOTAL	92	100

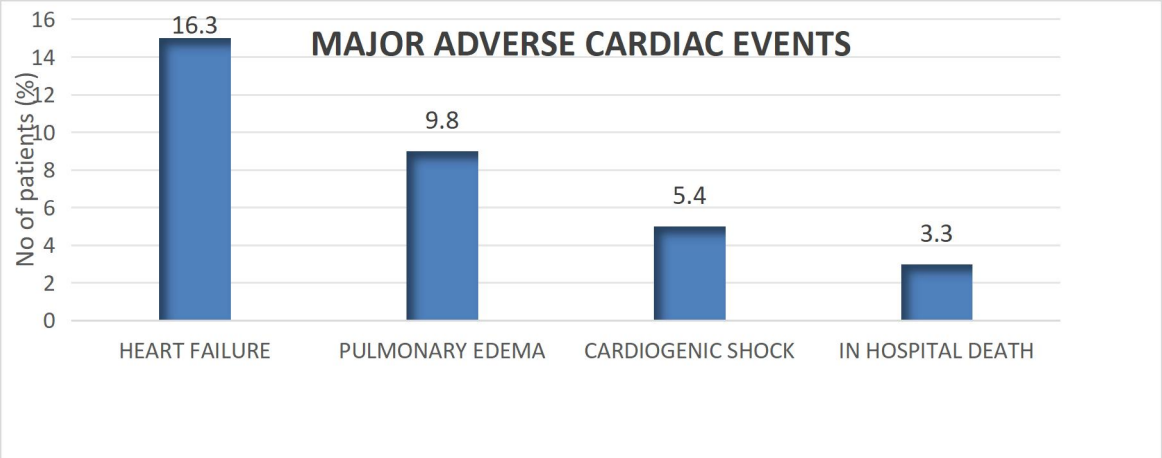
Table 11:DISTRIBUTION OF NFKB1 GENE POLYMORPHISM IN STUDY POPULATION

DISTRIBUTION OF PATIENTS ACCORDING TO MAJOR ADVERSE CARDIAC EVENTS

The following table 12 and graph 8 shows the Major Adverse Cardiac Events (MACE) in 92 patients during their period of in-hospital stay.Heart failure with ejection fraction less than 40% was seen in 15 patients (16.3%), pulmonary edema was seen in 9 patients (9.8%), cardiogenic shock was seen in 5 patients (5.4%), 3 (3.3%) in hospital death were observed.

MAJOR ADVERSE CARDIAC EVENTS	No of patients (n=92)	Percentage %
HEART FAILURE	15	16.3
PULMONARY EDEMA	9	9.8
CARDIOGENIC SHOCK	5	5.4
IN HOSPITAL DEATH	3	3.3

TABLE 12 : DISTRIBUTION OF PATIENTS ACCORDING TO MAJOR ADVERSE CARDIAC EVENTS



GRAPH 8:DISTRIBUTION OF PATIENTS ACCORDING TO MAJOR ADVERSE CARDIAC EVENTS

DISCUSSION

DISCUSSION

This is a prospective cross-sectional study where aim of the study was to look for NKB1 gene polymorphism in patients admitted with acute coronary syndrome. This study was conducted in 92 patients who fulfilled the inclusion criteria and were analyzed based on clinical history, blood investigations, ECG, 2D-ECHO and NKB1gene polymorphism.

AGE

In this study the most common age group with coronary artery disease was between 61-70 years which is similar to study done by Nadar Salari et al, in 2,982,6717 individuals in year 2023, concluded that prevalence of MI in population < 60 and > 60 years old were 3.8% and 9.5%, respectively. This is concurrent with our study [45]

In another study done by Sheetal Bodkhe et al, done in 2013-2014 in Maharashtra involving 1190 patients found that prevalence of coronary artery disease was common in age above 60 years.[46]

This study observes that maximum patients of ACS were of 60 years and above which suggests that old age is crucial risk factor for development of MI and is due to old age associated atherosclerosis, along with age associated comorbid conditions like diabetes and hypertension.

SEX

This study shows male preponderance with 55 patients (59.8%) been male, 37 patients (40.2%) been female which is similar to study done by Soumya Ranjan Mahapatra et al, in 2024 on 1000 patients of ACS which showed majority were male patients 700 (70%) while female patients were 300 (30%), showing significant male predominance.[47]

This implies that male group of patients are affected more then female with ACS as ill habits like smoking, tobacco chewing and alcohol consumption is found more in men.

OCCUPATION

This study contained participants of variety of occupation out of which maximum patients were homemaker 38 patients (48.3%), followed by farmer 31 patients (33.7%), then were servicemen consisting 13 patients (14.1%) and 10 patients (10.9%) were shopkeepers.

In a study done by, Alicja bortkiewicz et al, in 2010, in 1053 subjects, including 361 (34.3%) women and 692 (65.7%) men, concluded that in male group, farmers were 61% , 35% were servicemen, 3% were shopkeeper, while in female group 42 % were office workers, farmers were 25%, shopkeeper 15%, housewife were 2%,[48] hence maximum population in male group were from lower socioeconomic status, while females observed patients from high economic status.

In comparison to above mentioned study this study showed majority of patients were from low socioeconomic status.

Hence, this implies that knowledge of ACS among poor, illiterate population is very less and requires more dedication to educate such people.

SYMPTOMS

Various symptoms were observed in patient in this study in which chest pain in 80 patients (87%) was first most common symptom, second common symptom observed among 34 patients was dyspnea (37%) followed by abdominal pain in 6 patients (6.5%) then was palpitations in 3 patients (3.3%) and syncope 1 patient (1.1%).

Similar symptoms were observed in a study conducted by lena bjorck et al, in 1,72,981 patients registered in SWEDEHEART registry between 1996 and 2010, majority of patients presented with chest pain (84.4%) followed by dyspnea (29.6%).[49]

In another study conducted by David brieger et al, in 2004 among 20,881 patients, dominant presenting symptoms were syncope, nausea or vomitting, dyspnea, whereas 1,763 patients (8.4%) presented without chest pain,[50] which shows different initial presentation in ACS patients in comparison to this study.

As ACS can manifest with atypical symptoms rather than classic symptoms of ischaemia, there is a need to aware general population of such atypical symptoms

and care should be taken to identify such symptoms at earliest and intervene accordingly.

RISK FACTORS

Risk factors like smoking, tobacco chewing, alcohol consumption, hypertension and diabetes have been studied in 92 patients in this study. Based on risk factors this study concluded that Diabetes in 66 patients (66.3%) and Hypertension in 66 patients (66.3%) were majorly associated risk factors. Following this tobacco chewing among study group were more comprising 24 patients (26.1%). While smokers were16 patients (16%) and alcohol consumption was seen in 14 patients(14%). Family history for acute coronary syndrome was found in 6 patients (7.6%) of which 2 patients were young.

In a study done by Ewa M Maroszynska Dmoch et al, in 239 patients in kielce (poland) from 2001-2008 found most common risk factors associated with patients of ACS were hypertension, obesity, smoking and positive family history.[51]

In another study by Anushka Agarwal et al, in 13,071 South Asian participants across four south asian population were studied including Bangladesh, India, Nepal, and Pakistan from 2010-2022 concluded that smokeless tobacco, diabetes mellitus, hypertension, positive family history in young coronary artery disease patients were associated risk factors in ACS.[52]

Hence there is a need to spread awareness about hazardous effects of tobacco chewing and smoking among population. While measures to maintain healthy diet, regular physical activity and weight loss in obese should be promoted among population suffering from diabetes and hypertension to hault the incidences of ACS.

ELECTROCARDIOGRAM

In this study out of 92 patients maximum number of ACS patients that is 30 patients (32.6%) showed NSTEMI on ECG and11 patients (12.8%) showed STEMI anterior wall and STEMI inferior wall in 14 patients (15.2%).

In a study done by Abdallah Sanaani et al, in 131 patients in 2012, ECG findings showed maximum 102patients (77.9%) were of NSTEMI while 18 patients (13.7%) showed inferior STEMI, 11 patients (8.4%) showed anterior STEMI.[53]

Number of NSTEMI were maximum in both study group though they were significantly higher in study done by Abdallah Sanaani et al, followed by STEMI inferior wall and STEMI anterior wall while this study showed involvement of STEMI anterior wall more than STEMI inferior wall.

2D ECHOCARDIOGRAM

In this study, 25 patients (27.2%) showed anterior wall hypokinesia, 22 patients (23.9%) showed inferior wall hypokinesia, while 15 patients (19.5%) showed antero-septal wall hypokinesia, 4 patients (4.3%) showed global hypokinesia, 8 patients showed normal 2D ECHO findings.

In another study conducted by Ashish goel et al, in 2016, in 50 ACS patients in Gaziabad, observed that 64% patients showed anterior wall MI while 24% patients showed inferior wall MI and 12% showed antero-septal wall MI.[54]

In a study done by Manjit singh et al, in 2024, in North India in 55 MI cases, found that 5 patients (9.0%) on 2D ECHO showed global hypokinesia, 20 patients (36.5%)showednew regional wall motion abnormality and 30 patients (54.5%) showed normal 2D ECHO findings.[55]

Hence, anterior wall MI is common finding indicates involvement of Left anterior descending artery involvement which is common culprit to cause atherosclerosis.

NUCLEAR FACTOR KAPPA B 1 GENE POLYMORPHISM

NFKB1 gene (4q24) with 27 exons plays pathogenic role by regulating pro inflammatory genes linked to atherosclerosis and aids in plaque formation. This study was conducted to find genetic polymorphisms in exons 10 and 2 of NFKB1 gene, while no pathogenic mutation was found in both targeted exons. A study done by Shreedhar Naik et al, from 2015-2016 in 100 MI cases and 100 healthy controls in South India, found four novel missense mutations in exon 2 of NFKBIL1 gene in MI patients. They found that NFKBIL1 gene lead to structural and functional changes in transcription factor which in turn increased risk of MI.[56] While this study which specifically targeted exon 2 and 10 of NFKB1 gene found no single mutation in either exons in study group, implying that these critical regions are highly

conserved in this population. Hence, it describes complex, multifactorial nature of ACS pathophysiology and warrants future study targeting other genomic mutations.

In other study carried by Eliecer Coto et al, in 2019 in 1032 study population, targeting two common NFKB1 (-94 delATTG) and NFKBIA (rs8904) polymorphisms did not found association with CAD.[57]

In a study done by Boccardi V et al, in 2011 in 253 cases, -94 ins/del ATTG polymorphism, frequency of II, ID and DD genotypes was 41.5%, 45.5% and 13%, respectively. Frequency of D allele were higher in control group compared to MI cases and compared to D non carriers, D carriers had significant low fibrinogen and CRP level implying protective role of -94 ins/del ATTG polymorphism in causing MI.[58] In a study done by Zahra Darabi et al, in 2021-2022, on 462 patients in iran found no correlation between ATTG polymorphism of NFKB1 gene and cardiometabolic risk factors in patients of acute coronary syndrome.[59]

In contrast to this study, a study conducted by Jun-Yi-Luo from 2010-2014 in 359 MI cases and 1085 control, the relation between ins/del variation of NFKB1 gene rs28362491 and susceptibility of MI in a Chinese Han population was studied which showed genotype D allele and DD were more pronounced in MI cases compared to control group.[60]

MAJOR ADVERSE CARDIAC EVENTS

In 92 patients of ACS on average hospital stay of 2±22 days, Major adverse cardiac events (MACE) were observed among which heart failure was seen in 15 patients (16.3%) with ejection fraction less than 40%, following this was pulmonary edema in 9 patients (9.8%) , followed by cardiogenic shock in 5 patients (5.4%), there were 3 in hospital deaths (3.3%) observed. In a study done by Navdeep Singh Sidhu et al, in year 2020, in 621 patients observed MACE in 119 patients (19.2%) in which cardiac arrest was most common followed by heart failure (8.1%), shock occurred in 14 patients (2.3%), stroke occurred in 5 patients (0.3%) and 20 patients (3.2%) died during in hospital stay.[61] As seen patients in above mentioned study had cardiac arrest as first common MACE followed by heart failure. Whereas this study observed heart failure as most common MACE followed by pulmonary edema, percentage of in hospital deaths were similar in both studies.

CONCLUSION

CONCLUSION

This study investigated potential genetic variation in exons 10 and 2 of the NFKB1 gene in ninety two patients of acute coronary syndrome (ACS) and found no single mutation. This genetic heterogeneity suggests involvement of diverse genetic factors contributing to the disease pathogenesis and hence need for broader genomic analyses.

SUMMARY

SUMMARY

100 Patients presenting with symptoms of Acute Myocardial Infarction were screened in Shri B M Patil Medical college and hospital, Vijayapura from May 2023 to December 2024, in which 92 cases were included as per inclusion criteria and 8 cases were excluded as per exclusion criteria.

- The aim of this study was to detect Polymorphism of Nuclear factor kappa b1 gene in patients with Acute coronary syndrome.
- The most common age group of patients in this study were between age group of 61-70 years.
- This study showed male preponderance with 55 patients (59.8%) been male, 37 patients (40.2%) been female.
- Most common occupational group were from low socio-economic status which were homemaker (n=38) 41.3% then were farmers 33.7%.
- Diabetes (n=66) 66.3% and hypertension (n=66) 66.3% were commonly observed risk factors, followed by tobacco chewing (n=24) 26.1%.
- Chest pain (n=80) 87% most common symptom among patients, second common symptom was dyspnea (n=34)37% followed by abdominal pain (n=6) 6.5%, palpitations (n=3) 3.3%, syncope (n=1) 1.1% was least common symptom observed.
- On ECG maximum showed NSTEMI (n=30) 32.6%, then STEMI Antero septal wall (n=21) 22.8% , STEMI inferior wall (n=14) 15.2%.
- Most common 2D ECHO finding was anterior wall hypokinesia (n=25) 27.2%, followed by inferior wall hypokinesia (n=22) 23.9%, lateral wall hypokinesia (n=18), global hyopkinesiain (n=4) 4.3% patients.
- This study found no pathogenic mutation in targeted specific exon 10 and 2 of NFKB1 gene in ACS patients.
- Major adverse cardiac events seen in patients during hospital stay were heart failure in 15patients(16.3%), pulmonary edema in 9 patients (9.8%), cardiogenic shock in 5 patients (5.4%) and 3 in hospital death.

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BIBLIOGRAPHY

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ANNEXURE

ANNEXURE I

INSTITUTIONAL ETHICAL CLEARENCE CERTIFICATE

BLDE
(DEEMED TO BE UNIVERSITY)
Declared as Deemed to be University u/s 3 of UGC Act, 1956
Accredited with 'A' Grade by NAAC (Cycle-2)
The Constituent College
SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 909/2023-24 10/4/2023

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this University met on **Saturday, 18th March, 2023 at 11.30 a.m. in the CAL Laboratory, Dept. of Pharmacology,** scrutinizes the Synopsis/ Research Projects of Post Graduate Student / Under Graduate Student /Faculty members of this University /Ph.D. Student College from ethical clearance point of view. After scrutiny, the following original/ corrected and revised version synopsis of the thesis/ research projects has been accorded ethical clearance.

TITLE: "GENETIC STUDY OF NUCLEAR FACTOR KAPPA B 1 GENE POLYMORPHISM IN ACUTE CORONARY SUNDROME".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR.AMRUTA SUBHASH MHASKE

NAME OF THE GUIDE: DR.BADIGER SHARANABASAWAPPA, PROFESSOR AND HOD, DEPT. OF MEDICINE.

Dr. Santoshkumar Jeevangi
Chairperson
IEC, BLDE (DU),
VIJAYAPURA
Chairman,
Institutional Ethical Committee,
BLDE (Deemed to be University),
Vijayapura

Dr.Akram A. Naikwadi
Member Secretary
IEC, BLDE (DU),
VIJAYAPURA
MEMBER SECRETARY
Institutional Ethics Committee
BLDE (Deemed to be University)
Vijayapura-586103, Karnataka

Following documents were placed before Ethical Committee for Scrutinization.

- Copy of Synopsis/Research Projects
- Copy of inform consent form
- Any other relevant document

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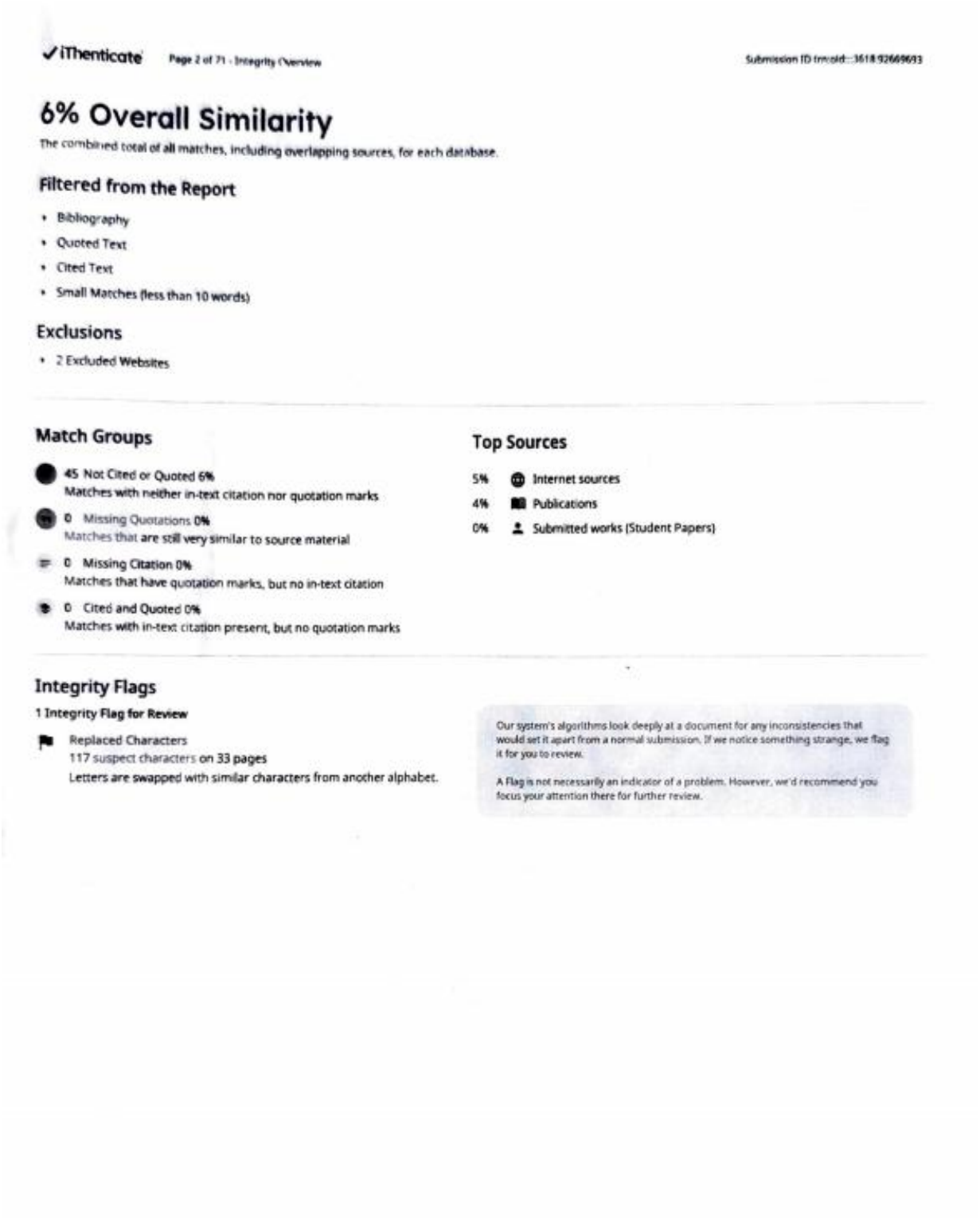

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ANNEXURE – II

CONSENT FORM

ಪ್ರಬಂಧ/ಸಂಶೋಧನೆಯಲ್ಲಿ **ಭಾಗವಹಿಸಲು ತಿಳುವಳಿಕೆಯುಳ್ಳ ಸಮ್ಮತಿ**

ನಾನು, ಕೆಳಗೆ ಸಹಿ ಮಾಡಿರುವ, _____, S/O D/O W/O _____, _____ವರ್ಷದ, _____ ನ ಸಾಮಾನ್ಯ ನಿವಾಸಿ, ಶ್ರೀ ಬಿ ಎಂ ಪಾಟೀಲ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜು ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರದ ಡಾ. ಅಮೃತಾ ಎಸ್ ಎಂಎಚ್‌ಕೆ ಅವರು ನನ್ನನ್ನು _____ ರಂದು ಸಂಪೂರ್ಣವಾಗಿ ಪರೀಕ್ಷಿಸಿರುವುದಾಗಿ ಈ ಮೂಲಕ ತಿಳಿಸುತ್ತೇನೆ/ಘೋಷಿಸಿ ಮಾಡುತ್ತೇನೆ. _____ (ಸ್ಥಳ), ಮತ್ತು ನಾನು _____ ಕಾಯಿಲೆಯಿಂದ ಬಳಲುತ್ತಿದ್ದೇನೆ ಎಂದು ನನ್ನ ಸ್ವಂತ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, ಮತ್ತು ಈ ರೋಗ/ಸ್ಥಿತಿಯು ಈ ಕೆಳಗಿನ ರೋಗಗಳನ್ನು ಅನುಕರಿಸುತ್ತದೆ. ಇದಲ್ಲದೆ, ವೈದ್ಯ ರಿಶರ್. ಅಮೃತಾ ಎಸ್ ಎಮ್‌ಹಾಸ್ಕೆ ಅವರು ವಿಜಯಪುರ ಜನಸಂಖ್ಯೆಯಲ್ಲಿ ತೀವ್ರವಾದ ವಿರಿದಮಿಯ ಸಿಂಡ್ರೋಮ್ ಹೊಂದಿರುವ ರೋಗಿಗಳಲ್ಲಿ ನ್ಯೂಕ್ಲಿಯರ್ ಫ್ಯಾಕ್ಟರ್ ಕಪ್ಪಾ ಬಿ 1 ಜೀನ್‌ನ ಜೆನೆಟಿಕ್ ಸ್ವರೂಪ ಎಂಬ ಪ್ರಬಂಧ/ಸಂಶೋಧನೆ ನಡೆಸುತ್ತಿದ್ದಾರೆ ಎಂದು ನನಗೆ ತಿಳಿಸಿದರು. ಡಾ.ಬಡಿಗೇರ್ ಶರಣಬಸವಪ್ಪ ಅವರ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವಂತೆ ವಿನಂತಿಸುತ್ತೇನೆ. ದಿನನಿತ್ಯದ ಚಿಕಿತ್ಸಾ ವಿಧಾನಗಳ ಹೊರತಾಗಿ, ಪೂರ್ವ-ಶಸ್ತ್ರಚಿಕಿತ್ಸಾ, ಶಸ್ತ್ರಚಿಕಿತ್ಸೆಯ ನಂತರದ ಮತ್ತು ಅನುಸರಣಾ ಅವಲೋಕನಗಳನ್ನು ಅಧ್ಯಯನಕ್ಕಾಗಿ ಉಲ್ಲೇಖ ದೇಣಿಗೆಯಾಗಿ ಬಳಸಿಕೊಳ್ಳಲಾಗುತ್ತದೆ. ಈ ಕಾರ್ಯವಿಧಾನದ ಸಮಯದಲ್ಲಿ, ಅಂತಹ ಪ್ರತಿಕೂಲ ಫಲಿತಾಂಶಗಳನ್ನು ಎದುರಿಸಬಹುದು ಎಂದು ವೈದ್ಯರು ನನಗೆ ತಿಳಿಸಿದ್ದಾರೆ. ಮೇಲಿನ ತೊಡಕುಗಳ ವೈಕಿ, ಅವುಗಳಲ್ಲಿ ಹೆಚ್ಚಿನವು ಚಿಕಿತ್ಸೆ ನೀಡಬಹುದಾದವು ಆದರೆ ನಿರೀಕ್ಷಿತವಲ್ಲ ಆದ್ದರಿಂದ ನನ್ನ ಸ್ಥಿತಿಯು ಉಲ್ಬಣಗೊಳ್ಳುವ ಸಾಧ್ಯತೆಯಿದೆ, ಮತ್ತು ಅವರೂಪದ ಸಂದರ್ಭಗಳಲ್ಲಿ, ನಿರೀಕ್ಷಿತ ರೋಗನಿರ್ಣಯ ಮತ್ತು ಉತ್ತಮ ಚಿಕಿತ್ಸೆ ಲಭ್ಯವಾಗಿದ್ದರೂ ಸಹ ಇದು ಮಾರಕವೆಂದು ಸಾಬೀತುಪಡಿಸಬಹುದು. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಯು ಅಧ್ಯಯನದ ಫಲಿತಾಂಶಗಳ ಮೌಲ್ಯಮಾಪನಕ್ಕೆ ಸಹಾಯ ಮಾಡುತ್ತದೆ ಎಂದು ವೈದ್ಯರು ನನಗೆ ತಿಳಿಸಿದ್ದಾರೆ, ಇದು ಮುಂದಿನ ದಿನಗಳಲ್ಲಿ ಇದೇ ರೀತಿಯ ಇತರ ಪ್ರಕರಣಗಳ ಚಿಕಿತ್ಸೆಗೆ ಉಪಯುಕ್ತ ಉಲ್ಲೇಖವಾಗಿದೆ ಮತ್ತು ನಾನು ಪರಿಹಾರವನ್ನು ಪಡೆಯುವಲ್ಲಿ ಪ್ರಯೋಜನ ಪಡೆಯಬಹುದು ನಾನು ಬಳಲುತ್ತಿರುವ ಕಾಯಿಲೆಯ ಸಂಕಟ ಅಥವಾ ಚಿಕಿತ್ಸೆ.

ನಾನು ನೀಡಿದ ಮಾಹಿತಿ, ಮಾಡಿದ ಅವಲೋಕನಗಳು, ತನಿಖಾಧಿಕಾರಿಗಳು ನನ್ನ ಮೇಲೆ ತೆಗೆದ ಛಾಯಾಚಿತ್ರಗಳ ವೀಡಿಯೊ ಗ್ರಾಫ್‌ಗಳನ್ನು ಗೌಪ್ಯವಾಗಿರಲಾಗುತ್ತದೆ ಮತ್ತು ಶೈಕ್ಷಣಿಕ ಉದ್ದೇಶಗಳಿಗಾಗಿ ಹೊರತುಪಡಿಸಿ ನನ್ನ ಕಾನೂನು ವಾರಿಗೆದಾರ ಅಥವಾ ನನ್ನನ್ನು ಹೊರತುಪಡಿಸಿ ಬೇರೆ ವ್ಯಕ್ತಿಯಿಂದ ಮೌಲ್ಯಮಾಪನ ಮಾಡಲಾಗುವುದಿಲ್ಲ ಎಂದು ವೈದ್ಯರು ನನಗೆ ತಿಳಿಸಿದ್ದಾರೆ. ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಯು ಸಂಪೂರ್ಣವಾಗಿ ಸ್ವಯಂಪ್ರೇರಿತವಾಗಿದ್ದರೂ, ನಾನು ನೀಡಿದ ಮಾಹಿತಿಯ ಆಧಾರದ ಮೇಲೆ, ರೋಗನಿರ್ಣಯಕ್ಕೆ ಸಂಬಂಧಿಸಿದ ಚಿಕಿತ್ಸೆಯ/ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ನಾನು ಯಾವುದೇ ಸ್ಪಷ್ಟೀಕರಣವನ್ನು ಕೇಳಬಹುದು, ಚಿಕಿತ್ಸೆಯ ವಿಧಾನ, ಚಿಕಿತ್ಸೆಯ ಫಲಿತಾಂಶ ಅಥವಾ ಮುನ್ನರಿವು ಕೇಳಬಹುದು ಎಂದು ವೈದ್ಯರು ನನಗೆ ತಿಳಿಸಿದರು. ಅದೇ ಸಮಯದಲ್ಲಿ, ನಾನು ಬಯಸಿದಲ್ಲಿ ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನನ್ನ ಭಾಗವಹಿಸುವಿಕೆಯಿಂದ ನಾನು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಹಿಂದೆ ಸರಿಯಬಹುದು ಅಥವಾ ತನಿಖಾಧಿಕಾರಿಯು ನನ್ನನ್ನು ಅಧ್ಯಯನದಿಂದ ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ವಜಾಗೊಳಿಸಬಹುದು ಆದರೆ ಚಿಕಿತ್ಸೆಯ ವಿಧಾನ ಮತ್ತು ಅನುಸರಿಸುವುದಿಲ್ಲ ಎಂದು ನನಗೆ ತಿಳಿಸಲಾಗಿದೆ- ನಾನು ಡಿಸ್ಚಾರ್ಜ್ ಮಾಡಲು ವಿನಂತಿಸದ ಹೊರತು. ಪ್ರಬಂಧ ಅಥವಾ ಸಂಶೋಧನೆಯ ಸ್ವರೂಪ, ಮಾದಲಾದ ರೋಗನಿರ್ಣಯ, ಚಿಕಿತ್ಸಾ ವಿಧಾನಗಳನ್ನು ಅರ್ಥಮಾಡಿಕೊಂಡ ನಂತರ, ನಾನು ನನ್ನ ಸಂಪೂರ್ಣ ಪ್ರಜ್ಞಾಪೂರ್ವಕ ಮನಸ್ಸಿನ ಅಡಿಯಲ್ಲಿ ಕೆಳಗೆ ಸಹಿ ಮಾಡಿದ ಶ್ರೀ/ಶ್ರೀಮತಿ _____ ಹೇಳಿದ ಸಂಶೋಧನೆ/ಪ್ರಬಂಧದಲ್ಲಿ ಭಾಗವಹಿಸಲು ಒಪ್ಪುತ್ತೇನೆ.

ರೋಗಿಯ ಸಹಿ:
ವೈದ್ಯರ ಸಹಿ:
ಸಾಕ್ಷಿ:
ದಿನಾಂಕ:
ಸ್ಥಳ:

BLDE (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 AMRUTA S MHASKE of Shri B M Patil Medical College Hospital and Research Centre have 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. AMRUTA S MHASKE informed me that he/she is conducting dissertation/research titled A STUDY OF NUCLEAR FACTOR KAPPA B1 GENE POLYMORPHISM IN ACUTE CORONARY SYNDROME IN B L D E (DEEMED TO BE UNIVERSITY) SHRI B M PATIL MEDICAL COLLEGE HOSPITAL AND RESEARCH CENTRE VIJAYAPURA, KARNATAKA. Under the guidance of Dr. BADIGER SHARANABASAWAPPA requesting my participation in the study. Apart from routine treatment procedures, the pre-operative, operative, postoperative, and follow-up observations will be utilized for the study as reference data. The Doctor has also informed me that during the 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 a chance of aggravation of my condition, and in rare circumstances, it may prove fatal despite the anticipated diagnosis and best treatment made available. Further Doctor has informed me that my participation in this study help in the evaluation of the results of the study, which is a useful reference to the treatment of other similar cases in the 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 my legal hirer or me except for academic purposes.

The Doctor did inform me that though my participation is purely voluntary, based on the information given by me, I can ask for any clarification during the course of treatment/study related to diagnosis, the 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 the patient:

Signature of Doctor:

Witness:

Date:

Place:

ANNEXURE – III: SCHEME OF CASE TAKING PROFORMA

B L D E (DEEMED TO BE UNIVERSITY) SHRI BM PATIL MEDICAL COLLEGE

HOSPITAL AND RESEARCH CENTRE, VIJAYAPUR.

SCHEME OF CASE TAKING

Name:	Case No:
Age:	IP No:
Sex:	Date of Admission:
Occupation:	Date of Discharge:
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

- Pulse Rate :
- Blood Pressure :
- Respiratory Rate:
- BMI:
- Temperature:
- Hair:
- Eyes:
- Pupils:
- Nose:
- Ears:
- Oral Cavity:
- Upper Limbs:
- Chest:

- Abdomen:
- Genitalia:
- Lower Limbs:
- Skin:

SYSTEMICEXAMINATION

CARDIOVASCULAR SYSTEM

Arterial system:

- Pulse
- Rate
- Rhythm Volume
- Character
- Condition of the vessel wall
- Radio radial delay
- Radio femoral delay
- Other peripheral pulses **Venous system: Engorged veins in the neck Jugular venous pulse:**

Blood Pressure

Precordial examination:

Inspection:

Palpation:

percussion:

Auscultation:

RESPIRATORY SYSTEM:

Inspection:

Palpation:

Percussion

Auscultation:

PER ABDOMEN:

Inspection:

Palpation:

Percussion:

Auscultation:

CENTRAL NERVOUS SYSTEM:

Higher mental function:

Cranial nerves examination:

Motor system examination:

Sensory system examination:

Cerebellar signs:**INVESTIGATIONS**

HAEMATOLOGY –

Hemoglobin	gm %
Total WBC counts	Cells/mm^3
Differential counts -	
Neutrophils	%
Lymphocytes	%
Eosinophils	%
Monocytes	%
Basophils	%
ESR	mm after 1 hour
Platelet count	10^9/L

BIOCHEMISTRY–

Blood Sugar	mg/dl
Blood Urea	mg/dl
Serum Creatinine	mg/dl
Serum Sodium	mEq/L
Serum Potassium	mEq/L
LDL	mg/dl
HDL	mg/dl
Triglycerides	mg/dl
VLDL	mg/dl
Total Cholesterol	mg/dl
Troponin I	ng/ml

URINE EXAMINATION -

Albumin	
Sugar	
Microscopy	

ELECTROCARDIOGRAPHY

Standardization	
Rate	
Rhythm	
P wave	
PR interval	
QRS configuration	

QRS duration	
QRS Axis	
ST-Segment	
T wave	
QT interval	
QTc	

ECG DIAGNOSIS:

ECHOCARDIOGRAPHY:

CORONARY ANGIOGRAPHY (If required)

MAJOR ADVERSE CARDIAC EVENTS:

MASTER CHART

MASTER CHART

SL.N.	NAME	AGE	SEX	OCCUPATION	PHONE NO	ADDRESS	IP N.	D.O.A	D.O.D	DURATION OF STAY (DAY)	CHEST PAIN	DYSPNEA	PALPITATION	SYNCOPE	ABDOMINAL PAIN	ATYPICAL MANIFESTATION	DIABETES	HYPERTENSION	FAMILY HISTORY	SMOKING	ALCOHOL	TOBACCO CHEWING	PR	SYSTOLIC BP(mmhg)	DIASTOLIC BP (MMHG)	TEMPERATURE	RR	TROP I	HEMOGLOBIN G/DL	TOTAL COUNT	ESR	FBS/PPBS/RBS
1	KASAPPA VEERUPAKSHAPPA MASALI	60	M	FARMER	9881635360	UKKALI, VIJAPUR	80914	11-03-2024	14-03-2024	###	P	A	P	A	A	Abse	P	A	A	P	A	A	116	90	60	37	20	4812.5	6.7	22	50	rbs-125
2	LAXMI MUTTAGI	65	F	HOME MAKER		NIDAGUNDI	74222	05-03-2024	15-03-2024	10	p	P	A	A	A	Abse	P	P	A	A	A	A	84	130	80	37	18	490.6	7.1	7.01	15mm/hr	rbs-138 mg/dl
3	AMEENABEE SHIKKALAGAR	72	F	HOME MAKER	9341660111	AP DARGA ROAD	063398	09-03-2024	11-03-2024	2	A	P	A	A	A	Abse	P	P	A	A	A	P	120	100	60	37	18	34.8	7.9	13.1	15mm/hr	rbs-186mg/dl
4	NAGAPPA KALLAPPA	54	M	SERVICE	9632070689	AP SIKARAK	2E+05	12-06-2024	04-07-2024	22	P	P	A	A	P	Ab	P	A	P	A	A	A	120	110	80	37	26	1308.6	15.2	10.8	17mm/hr	rbs-330mg/dl
5	KALAMMA BADIGER	83	F	HOME MAKER	9880781735	KALIKA NAGAR, BIJAPUR	3E+05	28-07-2024	03-08-2024	6	A	P	A	A	P	Abse	P	A	A	A	A	P	160	160	100	38	32	700.5	11.7	24.1	14mm/hr	rbs-209mg/dl
6	VITTAL BHARAMANNA HIREKURUBUR	62	M	SERVICE	9686162477	AP LOGAV TQ TIKOTA ,	3E+05	29-07-2024	02-08-2024	4	p	P	A	A	P	Abse	P	A	A	P	A	A	66	140	90	37	18	25605	11.4	22.5	23mm/hr	rbs-122mg/dl
7	SHARANAMMA BIRADAR	48	F	HOME MAKER	9742535506	AP BALAGAN	3E+05	02-08-2024	07-08-2024	5	P	A	A	A	A	P	A	P	A	A	A	P	100	130	80	37	18	11	11.4	12.4	8mm/hr	rbs-96mg/dl
8	KASTURIBAI KALABURAGI	72	F	HOME MAKER	7259683161	VIJAPUR	3E+05	01-08-2024	06-08-2024	5	P	A	A	A	A	P	P	P	P	A	A	A	76	150	90	37	18	7294	12.6	8	20mm/hr	rbs-95mg/dl
9	ARJUN SURYAVANSHI	42	M	SHOPKEEPER	8971512785	BEHIND RTO OFFICE, RAJAJI	3E+05	05-08-2024	09-08-2024	4	P	A	A	A	A	Absent	A	P	A	P	A	P	78	130	80	37	18	44.94	8.5	10.6	20mm/hr	rbs-209mg/dl
10	gurubai kavatagi	80	F	HOME MAKER	7411602953	AP LKALABI LAGI,	3E+05	05-08-2024	6/8/24 DEATH	1	A	P	A	A	A	Abse	A	P	A	A	A	A	120	150	90	37	28	170.8	11	9.91	28mm/hr	rbs-91mg/dl
11	JALABAYI LAMANI	71	F	HOME MAKER	9901795269	NARASAG ALGI LT	3E+05	07-08-2024	08-08-2024	1	P	A	A	A	A	Ab	A	A	A	A	A	A	81	130	80	37	18	1193.5	12.7	6.2	14mm/hr	rbs-98mg/dl
12	SHARANGOUDA BIRADAR	52	M	FARMER	9742535506	AP BALAGAN NUR TQ, TALIKOTI,	3E+05	07-08-2024	12-08-2024	5	P	A	A	A	A	Absent	P	P	A	A	A	A	76	120	70	38	18	2143.5	12	11.9	10mm/hr	FBS-256mg/dl
13	MALAKAPPA WADDAR	54	M	SERVICE	9663621801	A/P TREJARI COLONY,	15143	07-08-2024	10-08-2024	3	P	A	A	A	A	Abse	P	P	A	P	P	P	86	130	80	37	20	4328.1	9.4	8.82	30mm/hr	rbs-457mg/dl
14	MEENAXI SHABADI	75	F	HOME MAKER	7411044234	AP SHASHRI NAGAR,	3E+05	08-08-2024	13-08-2024	5	P	A	A	A	A	Abse	P	P	A	A	A	A	100	160	90	36	22	110.1	10.7	4.8	53mm/hr	rbs-122mg/dl
15	NEELAPPA HAJERI	75	M	FARMER	9741363117	AT TONA SYYAL TQ,	3E+05	10-08-2024	13-08-2024	3	P	A	A	A	A	Abse	P	A	A	A	A	A	32	70	40	37	18	17.8	15	8.05	10mm/hr	rbs-120mg/dl
16	GOPU CHAVAN	70	M	SERVICE	9740885955	A/P ITTANGIHAL 03,TQ	3E+05	10-08-2024	19-08-2024	9	P	P	A	A	A	Abse	P	P	A	A	A	P	106	200	110	37	20	28.3	10	13.3	10mm/hr	rbs-106mg/dl
17	GURUBAI SUDAM	55	F	HOME MAKER	9686749275	TADAVALAGA, INDI	3E+05	07-08-2024	12-08-2024	5	A	P	A	A	A	Abse	A	P	A	A	A	A	68	130	80	37	20	11	11.6	11.2	15mm/hr	rbs-122mg/dl
18	SHARANAPPA AYAPPA KOPPAD	55	M	FARMER	7259942002	AP SHIVANAGI TQ, BIJAPUR	3E+05	12-08-2024	19-08-2024	6	A	A	A	A	A	Absent	A	A	A	P	P	A	80	220	120	37	18	7.6	11.6	8.97	15mm/hr	rbs-152mg/dl
19	KASHINATH BALCHABAL	61	M	FARMER	8971212290	KALLAKAVATAGI, AP	3E+05	12-08-2024	19-08-2024	7	P	A	A	A	A	Abse	P	P	A	A	A	A	82	136	80	38	18	42	11.6	5.35	15mm/hr	rbs-150mg/dl
20	BHIMANNA SHANTAPPA KUMBAR	68	M	FARMER	8722148224	KUMBARONI INDI, BIJAPUR	3E+05	12-08-2024	19-08-2024	7	P	A	A	A	A	Abse	A	P	A	P	A	P	78	126	80	37	16	7	9.1	9.2	10mm/hr	rbs-150mg/dl
21	SIDDAPPA KENCHAPPA CHALAMI	65	M	FARMER	9741183777	AP YALAJERI TQ,	3E+05	12-08-2024	20-08-2024	8	P	P	A	A	A	Abse	P	A	A	P	A	P	78	160	80	38	26	2039	9.8	8.77	10mm/hr	rbs-193mg/dl

SL.N.	BLOOD UREA	Sr. Creatine	SERUM SODIUM	Sr.POTASSIUM	TOTAL CHOLESTROL	TRIGLYCERIDES	HDL(mg/dl)	LDL	ECG-RATE	RHYTHM	P WAVE	PR INTERVAL	QRS CONFIGURATION	QRS DURATION	QRS AXIS	ST-SEGMENT	T wave	QT	QTc	ECG DIAGNOSIS	positive t wave in avr	Fragmented qrs	2D ECHO REGIONAL MOTION WALL ABNORMALITY	LVEF	I/E	NFKB1 GENE MUTATION	CAG	HEART FAILURE	PULMONARY EDEMA	ARRYTHMIAS	CARDIOGENIC SHOCK	IN HOSPITAL DEATH
1	50	0.7	144	3.8	162	350	16	120	100BPM	Tachycardia	0.08	0.12	POOR R WAVE PROGRESSION	0.04s	N	ST DEPRESSION V2-V4,1 AVL	INVERTED V3-V6	480s	520s	NSTEMI ANTEROSEPTAL AND	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	##	I	A	NOT DONE	P	A	A	A	A
2	38	0.8	141	4.4	224	125	20	155	92BPM	Regular	0.08S	0.08S	Q WAVES V1-V6, NOTCHED Q WAVE V4,V5-RBB PATTERN	0.08S	N	RBBB	INVERTED V4-V6	520s	572s	RBBB	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	30	I	A	NOT DONE	P	A	A	A	A
3	32	1.4	145	3.6	260	350	35	180	65BPM	Regular	0.04S	0.20S	RS COMPLEX V1-V5	0.08s	N	ST E 2,3,AVF	INVERTED V1-V6,2,3,AVF	400s	460s	STEMI INFERIOR WALL	A	A	HYPOKINESIA OF INFERIOR AND LATERAL WALL	35	I	A	TVD	P	A	A	A	A
4	42	1.2	134	5.3	246	132	36	183	150BPM	Tachycardia	0.12S	0.20S	DEEP Q WAVE V2,V3,3,2,AVF	0.04s	LAD	ST ELEVATION	INVERTED 2,3,AVF	320s	453s	STEMI INFERIOR	A	A	INFERIOR WALL HYPOKINESIA	40	I	A	NOT DONE	P	A	A	A	A
5	23	0.9	131	3.1	172	196	30	103	150BPM	Tachycardia	0.08S	0.08S	DEEP S WAVES IN V1-V2 TALL RAVES V5,V6-LVH	0.08S	N	ST DEPRESSION 3,AVF,V1-V4	NOT WAVE CHANGES	280s	450s	NSTEMI	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	35	I	A	NOT DONE	P	A	A	A	A
6	26	0.8	128	3.5	180	155	35	122	75bpm	Regular	0.08S	0.12s	QRS COMPLEX 2,3,AVF	0.04s	N	ST ELEVATIONV2-V6,2,3,AVF	INVERTED V2-V6	440s	492s	STEMI ANTERIOR WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	50	I	A	SVD	A	A	A	A	A
7	28	1	136	4.4	160	175	33	125	60BPM	Regular	0.08S	0.12s	RS COMPLEX V1-V5	0.04s	N	ST ELEVATION	INVERTED V2,AVL,V3	480s	480s	STEMI ANTERIOR	A	A	NO MOTION WALL ABNORMALITY	60	I	A	SVD	A	A	A	A	A
8	32	1.1	140	4.4	242	145	34	160	75bpm	Regular	0.08S	0.12s	RS COMPLEX V1-V5 NOTCHED R WAVE IN V1	0.04s	N	ST DEPRESSION V2-V4	NOT WAVE CHANGES	480s	530s	NSTEMI	A	A	HYPOKINESIA OF INFERIOR AND LATERAL WALL	45	I	A	NOT DONE	P	A	A	A	A
9	29	0.9	138	3.9	186	155	28	132	75bpm	Regular	0.12S	0.16S	DEEP Q WAVES V1-V5	0.04s	N	ST E V2-V5,2,3,AVF	INVERTED V2-V5	440s	490s	STEMI INFERIOR WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	50	I	A	DVD	A	A	A	A	A
10	34	1.6	141	5.3	175	146	35	112	100BPM	Tachycardia	0.04S	0.20S	rSR PATTERN 2,3,AVF, POOR R WAVE	0.04s	LAD	ST E V2-V6	INVERTED V2-V6	280s	360s	STEMI ANTEROSEPTAL	A	A	GLOBAL HYPOKINESIA	25	I	A	DVD	P	P	A	A	P
11	19	0.7	143	5.3	288	100	36	198	100BPM	Tachycardia	0.08S	0.12s	Q WAVES 2,3,AVF,V5-V6	0.04s	N	ST E 2,3,AVF	INVERTED 2,3,AVF	440s	560s	STEMI INFERIOR	A	A	INFERIOR WALL HYPOKINESIA	55	I	A	SVD	A	A	A	A	A
12	26	0.8	138	3.4	207	147	26	140	75bpm	Regular	0.08S	0.20S	Q WAVES 3,AVF	0.04s	LAD	ST DEPRESSION V2-V4,2,3,AVF	INVERTED 2,3,AVF	480s	530s	NSTEMI	A	A	HPOKINESIA OF ANTEIOR AND ANTEROLATERAL WALL	45	I	A	TVD	A	A	A	A	A
13	29	0.6	136	4.3	181	139	32	111	100BPM	Tachycardia	0.08S	0.16S	DEEP Q WAVE 1,AVR,AVL, POOR R WVE PROGRESSION	0.12S	LAD	ST DEPRESSION V2-V4,1 AVL	NOT WAVE CHANGES	520s	670s	NSTEMI	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	35	I	A	NOT DONE	P	A	A	A	A
14	53	1.6	127	3.9	306	366	26	241	150BPM	Tachycardia	0.04S	0.08S	DEEP Q WAVE AAVR,V1, V2	0.04s	N	ST DEP -V3 -V6,2,3,AVF	INVERTED 2,3,AVF,V3 -V6	320s	410s	NSTEMI	A	A	NO MOTION WALL ABNORMALITY	60	E	A	NOT DONE	A	A	A	A	A
15	29	1.2	139	4.2	200	263	33	161	35BPM	Bradycardia	0.08S	0.24S	deep q wave v1-V2	0.04s	N	ST DEP V2,V3	INVERTED 2,3,AVF	360s	460s	NSTEMI	A	A	HYPOKINESIA OF INFERIOR AND POSTERIOR WALL	45	I	A	NOT DONE	A	P	A	A	A
16	46	1.7	134	5.1	262	117	21	201	75bpm	Regular	0.12S	0.20S	DEEP Q WAVES V1-V3	0.04s	N	ST E V2-V6,2 AVF	INVERTED V2-V6	440s	492s	STEMI ANTEROSEPTAL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	40	I	A	NOT DONE	P	A	A	A	A
17	23	0.8	140	4.7	320	344	35	263	100BPM	Regular	0.08S	0.12s	NOTCHES Q WAVE IN 3, qRQ PATTERN AVF	0.04s	LAD	ST DEPRESSION 2,3,AVF	INVERTED 2,3,AVF	400s	450s	NSTEMI	A	P IN 3	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	60		A	SVD	A	A	A	A	A
18	30	0.7	141	4.7	144	265	29	168	75bpm	Regular	0.08S	0.12s	Q WAVES 2,3,AVF	0.04s	N	ST DEPRESSION 2,3,AVF	INVERTED 2,3,AVF	440s	447s	NSTEMI	A	A	HYPOKINESIA OF INFERIOR AND INFERIO LATERAL WALL	45	I	A	NOT DONE	A	A	A	A	A
19	31	0.9	139	4.6	150	160	18	110	100BPM	Tachycardia	0.08S	0.12s	QRS COMPLEXES V2-V6	0.04s	N	ISOELECTRIC STSEGMENT	INVERTED V2-V4, AVL	400s	510s	NSTEMI	A	A	NO MOTION WALL ABNORMALITY	55	I	A	DVD	A	A	A	A	A
20	16	0.8	139	4	169	112	23	106	75bpm	Regular	0.04S	0.12s	RS COMPLEXES V2-V6	0.04s	LAD	ST DEPRESSION IN V2-V6	INVERTED V2-V6	440s	470s	NSTEMI	A	A	HPOKINESIA OF ANT ANTEROLATERAL	55	I	A	SVD	A	A	A	A	A
21	27	0.7	139	4.4	155	125	20	102	100BPM	Tachycardia	0.08S	0.12s	DEEP Q WAVES V1-V2	0.04s	N	ST DEPRESSION -V3 TO V6	INVERTED V2-V6	400s	560s	NSTEMI	positive	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	55	I	A	TVD	A	A	A	A	A

22	HANAMANTH PAWAR	40	M	SHOPKEE PER	1111222244	BIJAPUR	3E+05	12-08-2024	14-08-2024	2	P	A	A	A	A	Ab se	P	A	P	P	A	P	96	120	70	37	16	1.4	10	6.86	5mm/hr	rbs-144mg/dl	17	0.8	138	4.4	223	100	35	136	100BP M	Tachycardia	0.08S	0.16S	POOR R WAVE PROGRESSION Q WAVES IN 3,AVF	0.04 s	LAD	ST DEPRESSION 3,AVF	NO T WAVE CHANGES	400 s	510s	NSTEMI	A	A	INFERIOR WALL HYPOKINESIA	60	I	A	SVD	A	A	A	A	A
23	SANJEEV GUDDEVADI	53	M	SHOPKEE PER	7899544256	AP KANAKA DAS	3E+05	13-08-2024	17-08-2024	4	P	A	A	A	A	Ab se	P	P	A	A	A	A	100	150	90	37	18	500.2	13	6.99	20mm/hr	rbs-242mg/dl	16	0.8	138	3.7	186	12	27	131	100BP M	Tachycardia	0.08S	0.12s	NOTCHED QRS COMPLEX 2,3,AVL,AVF,V1-V4	0.04 s	N	ST ELEVATION 3,AVF,2	INVERTED V2-V4	360 s	460s	STEMI INFERIOR WALL	A	A	INFERIOR WALL HYPOKINESIA	55	I	A	TVD	A	A	P	A	A
24	REVANSIDAPPaA NG	69	M	SERVICE	7019729120	C/O GURULIN GAPPA NG 378	3E+05	13-08-2024	20-08-2024	7	P	A	A	A	A	Ab se nt	P	A	A	P	A	A	80	120	70	37	22	58.2	12.4	5.76	10mm/hr	rbs-138mg/dl	19	1.1	138	4.2	238	268	31	150	75bpm	Regular	0.08S	0.16S	Q WAVES V1-V6,AVL	0.12 S	N	ST ELEVATION V2,V3,, DEPRESSION	INVERTED V1-V4	400 s	447s	STEMI ANTEROSE PTAL WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	45	I	A	DVD	A	A	A	A	A
25	SAHEBI HUSEAINBASHA MASALI	75	F	HOME MAKER	8217666426	AP WATER TANK,	3E+05	14-08-2024	16-08-2024	2	P	P	A	A	A	Ab se	P	P	P	A	A	A	112	140	90	37	16	18.5	9.8	8.94	15mm/hr	rbs-128mg/dl	25	0.8	142	4.8	250	178	30	110	150BP M	Tachycardia	0.04S	0.12s	Q WAVES V1-V4	0.04 s	N	ST E V2-V5	INVERTED V2-V6	360 s	562s	STEMI ANTEROSE PTAL	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	35	I	A	NOT DONE	P	A	A	A	A
26	BASWANAND KUMBAR	64	M	FARMER	8660371887	AT POST HALAGU NAKI, TQ	3E+05	14-08-2024	21-08-2024	7	P	A	A	A	A	Ab se	P	P	A	P	A	A	56	140	70	37	18	556.7	12.2	12.1	10mm/hr	rbs-128mg/dl	28	0.7	140	4.1	170	261	39	198	150BP M	Tachycardia	0.08S	0.04S	DEEP Q WAVES V1-V3	0.08 S	N	ST ELEVATION V2-V5	INVERTED V1-V6	440 s	490s	STEMI ANTEROSE PTAL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	60	I	A	TVD	A	A	A	A	A
27	SWALIHA BEGUM MUNIRKHANPATHA N	49	F	HOME MAKER	9036225641	STATION ROAD, HASHIMP	3E+05	15-08-2024	21-08-2024	6	P	P	A	A	A	Ab se	A	P	A	A	A	P	68	126	80	37	24	26.3	13.5	10.2	15mm/hr	rbs-100mg/dl	33	0.7	145	4.7	255	178	27	166	75bpm	Regular	0.12S	0.20S	Rs complexes V1-V4	0.12 S	N	ST DEPRESSION 2 ,V1,AVR	NO T WAVE CHANGES	440 s	490s	NSTEMI	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	50	I	A	DVD	A	A	A	A	A
28	MAKANABAI PAWAR	70	F	HOME MAKER	7057275359	AT POST UTANAL, BIJAPUR	3E+05	16-08-2024	20-08-2024	4	A	P	A	A	A	Ab se	P	P	A	A	A	A	114	140	80	38	22	496.4	10	5.31	20mm/hr	rbs-158mg/dl	57	1.9	149	4.6	219	366	33	136	100BP M	Tachycardia	0.04S	0.16S	NOTCHED QRS 2,3,AVF, QSq pattern v1-v3	0.08 s	LAD	ST E V1-V3	inverted v2-v3	440 s	560s	LBBB	A	P	NO MOTION WALL ABNORMALITY	50	I	A	NOT DONE	A	A	A	A	A
29	RAVI MANE	41	M	SERVICE	9036568122	AP HANCHA NAL,	3E+05	16-08-2024	19-08-2024	3	P	P	A	A	A	Ab se	P	A	A	P	P	A	60	100	60	37	22	969.2	11.9	13.5	5mm/hr	rbs-126mg/dl	24	1	141	4.4	244	286	20	177	75BP M	Regular	0.12S	0.12s	Q WAVE IN V1	0.12 S	N	ST E V2-V4,AVF	INVERTED 2,3,AVF,V1-V6	480 s	530s	STEMI INFERIOR WALL	A	A	HYPOKINESIA OF INFERIOR AND POSTERIOR WALL	35	I	A	TVD	P	A	A	A	A
30	VAUDEO NADGOUDA	62	M	SERVICE	8618407896	JM ROAD NEAR BADIKAM	3E+05	16-08-2024	22-08-2024	6	P	A	A	A	A	Ab se	A	P	A	A	A	A	82	150	90	37	22	2.3	7.5	9.8	20mm/hr	rbs-342mg/dl	50	1.2	138	4.5	190	196	36	119	100BP M	Tachycardia	0.12S	0.16s	DEEP Q WAVES V1-V3	0.12 S	LAD	ST E V2-V4,2,3,AVF	INVERTED V2-V4	440 s	560s	STEMI ANTEROSE PTAL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	38	I	A	NOT DONE	P	A	A	A	A
31	HAMEEDABI MAKANDAR	67	F	HOME MAKER	9972698384	MANAGU LI, BASAVAN	3E+05	16-08-2024	24-08-2024	8	P	P	A	A	A	Ab se	P	P	A	A	A	P	90	110	70	37	22	2513.2	14	12.9	10mm/hr	rbs-116mg/dl	41	0.7	146	3.6	236	188	27	178	82BP M	Regular	0.12s	0.16s	Q WAVES V1-V4	0.12 S	N	ST ELEVATION V1-V4	HYPERAC UTE T WAVE V1-	360 s	420s	STEMI ANTEROSE PTAL	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	35	I	A	NOT DONE	A	P	P	P	P
32	CHANAWWA SOLAPUR	70	F	HOME MAKER	8861866240	HALAGA NI, TQ BABALES	3E+05	16-08-2024	23-08-2024	7	P	A	A	A	A	Ab se	A	A	A	A	A	P	82	180	110	37	22	23	13.2	5.21	10mm/hr	rbs-100mg/dl	30	1	139	4.8	200	172	33	159	100BP M	Regular	0.12s	0.16s	Rs complexes V1-V4	0.04 s	N	ST DEPRESSION 3,AVF,2	NO T WAVE CHANGES	480 s	620s	NSTEMI	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	60	I	A	DVD	A	A	A	A	A
33	MAHADEVI KHAKANDAKI	70	F	HOME MAKER	9901114294	BABALES HWAR TQ, BIJAPUR	3E+05	16-08-2024	21-08-2024	5	P	A	A	A	A	Ab se	A	A	A	A	A	P	80	94	70	37	18	50	8.9	6.23	15mm/hr	rbs-108mg/dl	30	0.7	145	3.4	120	218	35	130	100BP M	Regular	0.08s	0.16s	Rs complexes V1-V4	0.04 s	N	ST E V1-V4	INVERTED 3,AVF	440 s	560s	STEMI ANTEROSE PTAL	A	A	HPOKINESIA OF ANTEROLATERAL WALL	40	I	A	SVD	A	A	A	A	A
34	SIDRAY TOTAD	52	M	SERVICE		MALIONI, NAGANN UR, JHAMKHA	3E+05	16-08-2024	20-08-2024	4	P	A	A	A	A	Ab se nt	A	P	A	P	P	P	86	110	70	37	22	122	14	5.2	10mm/hr	rbs-186mg/dl	28	1.4	136	4.3	194	301	39	126	72bpm	Regular	0.12s	0.12s	rs complexes v4-v6	0.12 S	N	ST DEPRESSION 2,3,AVF,V4-V6	INVERTED V4-V6,3,AVF	380 s	420s	NSTEMI	A	A	HYPOKINESIA OF ANTEROINFERIOR WALL	60	I	A	SVD	A	A	A	A	A
35	IRAPPA S GALGALI	50	M	FARMER	9380617835	C/O SANGAPP A	3E+05	17-08-2024	21-08-2024	4	P	A	A	A	A	Ab se	A	P	A	A	A	A	96	150	100	38	20	25605	13	15.2	10mm/hr	rbs-106mg/dl	25	0.6	140	3.6	172	96	30	110	100BP M	Tachycardia	0.12S	0.12S	POOR R WAVE PROGRESSION	0.12 S	LAD	ST ELEVATION 2,3,AVF	INVERTED 2,3,AVF,V4-V6	400 s	510s	STEMI INFERIOR WALL	A	A	INFERIOR WALL HYPOKINESIA	45	I	A	NOT DONE	A	A	A	A	A
36	SUBHASH BAJANTRI	65	M	FARMER	9686395794	JUMANAL, BIJAPUR	3E+05	14-08-2024	28-08-2024	14	P	A	A	A	A	Ab se	P	P	A	P	A	A	90	70 SYS TO		37	22	11.7	13.3	24.2	15mm/hr	rbs-108mg/dl	43	1.1	135	3.7	198	172	36	141	100BP M	Tachycardia	0.12S	0.20S	Q WAVES V1-V4	0.08 S	N	ST E V2-V5 WITH ST DEP 2,3,AVF	INVERTED V2,AVF	440 s	560s	STEMI ANTEROSE PTAL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	25	I	A	NOT DONE	P	P	A	P	P
37	Bandagi Saab Mokashi	69	M	FARMER	9902591942	GUBBEW ADI	3E+05	19-08-2024	25-08-2024	6	P	A	A	A	A	Ab se	P	A	A	A	A	A	80	110	70	37	18	1540	12	11.1	10mm/hr	rbs-149mg/dl	20	0.8	132	3.2	235	190	30	162	75bpm	Regular	0.08S	0.16S	N	0.04 s	LAD	ST DEP V2,V3	NO T WAVE CHANGES	480 s	530s	STEMI ANTERIOR WALL	A	A	HPOKINESIA OF ANT ANTEROLATERAL	50	I	A	DVD	A	A	A	A	A
38	AMBAWWA SAGAI	65	F	HOME MAKER	9972454965	JALAKI INDI	3E+05	20-08-2024	29-08-2024	9	P	A	A	A	A	Ab se	P	P	A	A	A	A	78	130	80	37	18	556.7	12.2	12	30mm/hr	rbs-209mg/dl	23	1.2	139	4.2	200	170	26	116	100BP M	Tachycardia	0.08S	0.12s	Q WAVES 2,3,AVF	0.04 s	LAD	ST E V2-V4,AVF,2,3	INVERTED 2,3,	480	620	STEMI INFERIOR WALL	A	A	HYPOKINESIA OF INFERIOR AND LATERAL WALL	50	I	A	DVD	A	A	A	A	A
39	HANAMANTH KAMBLE	72	M	SERVICE	9036919366	JAMKAND I	1502	22-08-2024	02-09-2024	11	P	P	A	A	A	Ab se	A	A	A	P	A	A	90	14	90	37	18	100	10.9	19.6	29	RBS-236	61	1.2	138	4.2	180	150	35	115	75bpm	Regular	0.08S	0.16	Q WAVE IN V1,V6	0.04 s	LAD	ST E V2-V5	INVERTED V1,V6	520 s	580s	STEMI ANTERIOR WALL	A	A	HPOKINESIA OF ANT ANTEROLATERAL	30	I	A	TVD	P	A	A	A	A
40	LAXMAN HARIWAL	63	M	FARMER	7899544256	AP KANAKA DAS	21112	21-08-2024	28-08-2024	7	P	P	A	A	A	Ab se	A	P	A	P	A	P	60	170	100	37	20	36	16.3	#####	10mm/hr	rbs-268mg/dl	26	1.3	136	3.8	298	120	31	206	60BP M	Regular	0.12S	0.16S	S WAVE IN V1+R IN V8>38S-LVH	0.12 S	LAD	ST DEP LAVL,V5,V6	INVERTED V1-V6	400 s	410s	NSTEMI	A	A	HYPOKINESIA OF INFERIOR AND POSTERIOR WALL	45	I	A	SVD	A	A	A	A	A
41	VITTHAL DEVKHATE	70	M	FARMER		KYATANK ERI, INDI, BIJAPUR	23252	21-08-2024	28-08-2024	7	P	P	A	A	A	Ab se nt	P	A	A	P	P	P	58	130	70	38	18	1023	13	12.2	10mm/hr	rbs-161mg/dl	34	1	142	3.5	181	292	30	115	60BP M	Regular	0.08S	0.16s	Q WAVES IN 2,3,AVF	0.04 S	LAD	ST E 2,3,AVF	INVERTED 2,3,	400 s	400s	STEMI INFERIOR WALL	A	A	HYPOKINESIA OF INFERIOR AND INFERIO LATERAL WALL	40	I	A	NOT DONE	A	A	A	A	A
42	DJPATEL	83	M	SERVICE		BAGEWA DI, BIJAPUR	2E+05	21-08-2024	28-08-2024	7	P	A	A	A	A	Ab se	P	P	A	A	A	A	40	130	80	37	20	17	10	12	10mm/hr	rbs222mg/dl	20	0.8	140	3.2	150	192	30	102	43BP M	Bradycardia	0.12s	0.24s	rs in2,3,avf	0.12 s	LAD	ST E V1-V3	INVERTED V1	480 s	400s	STEMI ANTEROSE PTAL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	60	I	A	NOT DONE	A	A	A	A	A
43	SHRISHAIL BIRADAR	65	M	SHOPKEE PER		BAGEWA DI,	14233	21-08-2024	28-08-2024	7	P	P	A	A	A	Ab	P	P	A	A	P	P	97	100	70	37	20	88	12.8	13.3	5mm/hr	rbs-123mg/dl	17	0.7	131	4.3	186	244	35	122	100BP M	Tachycardia	0.12s	0.16s	q wave V1,3,AVR	0.12 S	LAD	ST ELEVATION	INVERTED V2,V3	360 s	480s	STEMI INFERIOR	A	A	INFERIOR WALL HYPOKINESIA	25	I	A	NOT DONE	P	A	A	A	A
44	DUNDAPPA GHANTI	60	M	SERVICE	8884888837	DARGA ROAD	0166	25-08-2024	01-09-2024	6	P	A	A	A	A	Ab	P	P	A	A	A	A	86	160	90	37	20	150	15.1	20.3	10mm/hr	RBS-137mg/dl	17	0.7	146	5	150	120	22	106	75bpm	Regular	0.08S	0.20S	Q WAVES 2,3,AVF	0.04 s	LAD	ST E 2,3,AVF	INVERTED 2,3,AVF	400 s	470s	STEMI INFERIOR	A	A	INFERIOR WALL HYPOKINESIA	40	I	A	SVD	P	A	A	A	A
45	MUNNIRA JATH	50	F	HOME MAKER		VIJAPUR	00020	26-08-2024	30-08-2024	4	P	A	A	A	A	Ab	A	A	A	A	A	A	52	80	60	37	18	2000	11.6	14	10mm/hr	rbs-120mg/dl	18	0.6	142	4.2	270	460	24	196	50BP M	Bradycardia	0.08S	0.12s	N	0.04 s	N	ST E 2,3,AVF	HYPERAC UTE T	520 s	470s	STEMI INFERIOR	A	A	INFERIOR WALL HYPOKINESIA	45	I	A	NOT DONE	A	A	A	A	A
46	BASANNA TORAVI	58	M	FARMER	9353675745	DOMANA L	00055	26-08-2024	01-09-2024	5	A	P	P	A	A	P	P	P	A	P	P	P	90	110	70	37	18	1154.4	12.5	8.5	10mm/hr	RBS-133mg/dl	35	1.4	134	3.8	175	136	26	101	100BP M	Tachycardia	0.08S	0.12s	Q WAVES 2,3,AVF,V5-V6	0.04 s	N	ST E 2,3,AVF,V2-V6	INVERTED 2,3,V4,V5	400 s	510s	STEMI ANTEROSE PTAL	A	A	HPOKINESIA OF ANT ANTEROLATERAL	35	I	A	TVD	P	A	A	A	A
47	TUKARAM RATHOD	54	M	SHOPKEE PER	9921255089	HANCHIN AL	00023	27-08-2024	03-09-2024	7	P	A	A	A	A	Ab se	P	A	A	P	A	A	70	140	80	37	20	415.08	12.4	8.3	15mm/hr	RBS-180mg/dl	27	0.9	136	4.2	250	135	32	188	75bpm	Regular	0.08S	0.16S	NOTCHED QRS 2,3,AVF,V2-V6	0.08 S	RAD	ST E 2,3,AVF,V2-V6	NO T WAVE CHANGES	400 s	470s	RBBB	A	P	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	35	I	A	NOT DONE	P	A	A	A	A

48	MAHADEVI KOLHAR	69	F	HOME MAKER	9980725920	MUDHOL	00025	28-08-2024	03-09-2024	6	P	A	A	A	A	Ab	A	P	A	A	A	P	78	150	90	37	18	179	11.6	5.8	15mm/hr	rbs-150mg/dl	30	0.7	140	3.3	180	250	35	115	75bpm	Regular	0.08S	0.12s	N	0.04 s	LAD	ST DEP V2,V3,V4,V5,V	INVERTED V2-V6	440 s	490s	NSTEMI	A	A	INFERIOR WALL HYPOKINESIA	45	I	A	NOT DONE	A	A	A	A	A
49	BANDAWA PATHAN SHETTY	76	F	HOME MAKER	9844704728	HONNUTAGI	4E+05	28-08-2024	03-09-2024	6	P	A	A	A	A	Ab	P	P	A	A	A	A	82	170	80	37	18	312	9.1	12.6	15mm/hr	RBS-192mg/dl	18	0.5	140	3.2	200	175	30	136	100BPM	Tachycardia	0.08S	0.16S	Q WAVES 2,3,AVF	0.04 s	N	ST E 2,3,AVF	INVERTED V2-V3	480	620s	STEMI INFERIOR	A	A	INFERIOR WALL HYPOKINESIA	50	I	A	NOT DONE	A	A	A	A	A
50	KAMALA BAI SONAD	63	F	HOME MAKER	9900168867	VIJAPUR	00179	28-08-2024	03-09-2024	6	P	A	A	A	A	Abse	P	P	A	A	A	A	98	120	80	37	18	24918	9.8	17.4	10mm/hr	rbs-122mg/dl	28	0.9	140	5.1	360	155	20	298	150BPM	Tachycardia	0.04S	0.12s	N	0.04 s	N	ST D V2-V6	NO T WAVE CHANGES	360 s	560s	NSTEMI	A	A	HPOKINESIA OF ANTEROLATERAL WALL	###	I	A	NOT DONE	A	A	A	A	A
51	BANUBI MULLA	66	F	HOME MAKER	9380359857	BABALAD	00069	29-08-2024	06-09-2024	8	P	P	A	A	A	Abse	P	P	A	A	A	P	82	130	80	37	18	122.1	10	7.3	10mm/hr	RBS-250mg/dl	20	0.8	135	3.4	180	165	35	110	100BPM	Tachycardia	0.08S	0.12s	DEEP Q WAVES V1-V3	0.04 s	N	ST E V1-V3	INVERTED V1-V4	440	560s	STEMI ANTERIOR WALL	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	45	I	A	NOT DONE	A	A	A	A	A
52	UMAKANTSONAD	73	M	HOME MAKER	8217056242	DEVARA HIPPARAGI	00042	30-08-2024	08-09-2024	9	P	P	A	A	A	Abse	P	A	A	A	A	A	72	120	80	37	18	0.1	13	8.41	10	FBS-189mg/dl	30	1.2	139	4	200	175	30	112	100BPM	Tachycardia	0.04S	0.12s	deep q wave v1-v5	0.04 s	N	ST E V2-V5	INVERTED V1-V5	400	510s	STEMI ANTEROSEPTAL	A	A	HPOKINESIA OF ANTEROLATERAL WALL	35	I	A	DVD	P	A	A	A	A
53	KASTURIBAI NAVI	55	F	HOME MAKER	8296365189	SALOTAGI	2857	12-09-2024	18-09-2024	6	P	P	A	A	A	Abse	A	A	A	A	A	A	86	90	60	37	18	1250	12.4	7.01	20mm/hr	RBS-286mg/dl	27	1.1	130	4.3	150	146	18	98	75bpm	Regular	0.08S	0.20S	deep q wave v1-V4	0.04 s	LAD	ST E V2-V5	INVERTED 2,3,AVF,V2-V6	400 s	440s	STEMI ANTERIOR WALL	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	30	I	A	TVD	P	P	A	A	A
54	SAMBAIMANDARD	73	F	HOME MAKER	8296670315	VIJAPUR	3017	13-09-2024	23-09-2024	10	P	A	A	A	A	Abse	A	P	A	A	A	P	66	110	70	37	18	101.1	9.8	9.52	15mm/hr	rbs-100mg/dl	43	1.1	136	3.8	176	156	23	110	75bpm	Regular	0.08S	0.16S	DEEP Q WAVES V1-V3	0.04 s	N	ST E V1-V5	ASYMETRIC T WAVE INVERVIS	360 s	400s	STEMI ANTEROSEPTAL	A	A	HYPOKINESIA OF INFERIOR AND LATERAL WALL	40	E	A	TVD	P	P	A	A	A
55	ZANIDANASEEN BURUJWALE	44	F	HOME MAKER	6361725907	ALLAPUR	4212	20-09-2024	27-09-2024	7	A	P	A	A	A	P	P	P	P	A	A	A	116	130	90	37	22	97.2	12.2	8.82	10mm/hr	RBS-323mg/dl	22	0.4	134	3.8	273	215	25	225	100BPM	Tachycardia	0.08S	0.12s	DEEP BROAD Q WAVES V1-V4	0.08 S	N	ST CHANGES V1-V4	INVERTED 2,3,AVF	400	510s	STEMI ANTEROSEPTAL	A	A	HYPOKINESIA OF SEPTUM	40	I	A	NOT DONE	P	A	A	A	A
56	ALLAMA KADADAGI	60	F	HOME MAKER	6361569127	MOTIGURAJ	4463	22-09-2024	29-09-2024	7	P	A	A	A	A	Abse	A	P	A	A	A	A	82	100	70	37	18	1221	13.5	22.2	15mm/hr	rbs-106mg/dl	29	0.7	142	4.2	207	118	33	122	75bpm	Regular	0.08S	0.12s	DEEP BROAD Q WAVES V1-V4	0.04 s	N	STEMI V1-V6	INVERTED V1-V6,2,3,AVF	480	530	STEMI ANTEROSEPTAL	A	A	HPOKINESIA OF ANTEROLATERAL WALL	45	I	A	NOT DONE	A	A	A	A	A
57	GAYATRI KULKARNI	60	F	HOME MAKER	9480259555	NEAR RAYAR	4472	22-09-2024	26-09-2024	6	P	P	A	A	P	P	P	P	P	A	A	A	117	120	90	37	20	785.2	10	15.4	10mm/hr	RBS-156mg/dl	44	1.3	138	4.5	250	164	33	184	100BPM	Regular	0.08S	0.12s	N	0.04 s	N	ST DEP V4-V6,2,3,AVF	INVERTED V4-	400 s	510s	NSTEMI	A	A	INFERIOR WALL HYPOKINESIA	60	I	A	NOT DONE	A	A	A	A	A
58	BHIMU KATTIMANI	35	M	FARMER	7709647757	KARAJAGI	4971	24-09-2024	28-09-2024	4	P	A	A	A	A	Abse	P	A	A	A	P	A	96	110	70	37	18	60	13.2	15.5	10mm/hr	RBS-101mg/dl	16	0.8	136	3.6	185	155	35	126	75bpm	Regular	0.08S	0.12s	DEEP Q WAVES V1-V4	0.04 s	N	ST E V2-V6	INVERTED V2-V6,2,3,AVF	400 s	440s	STEMI ANTEROSEPTAL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	30	I	A	SVD	P	A	A	A	A
59	REVANNSIDAPPA JOGUR	64	M	FARMER	8618846714	MAHADEV TEMPLE ROAD	3438	25-09-2024	01-10-2024	7	P	A	A	A	A	Abse	P	A	A	A	A	P	70	100	60	37	18	28	14.8	8.46	10mm/hr	RBS-151mg/dl	42	0.9	141	3.5	140	135	26	96	60BPM	Regular	0.08S	0.20S	DEEP BROAD Q WAVES V1-V4	0.04 s	LAD	ST E V1-V4	INVERTED V2-V6	480 s	480s	STEMI ANTEROLATERAL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	35	I	A	TVD	P	A	A	A	A
60	SHIVAPPA KALLIMANI	40	M	FARMER	7899948874	VIJAPUR	59934	26-09-2024	28-09-2024	2	P	A	A	A	A	P	P	P	A	P	A	A	86	130	90	37	18	10783	15.5	11.6	10mm/hr	rbs-150mg/dl	12	0.6	135	3.9	175	155	35	86	75bpm	Regular	0.08S	0.20S	N	0.04 s	N	ST E V1-V6	T WAVE CHANGES NOTED	520 s	580s	NSTEMI	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	40	I	A	SVD	P	A	A	A	A
61	MALLIKARJUN TEGGIHALLI	46	M	SHOPKEEPER	9880662046	ATHARGA	58504	26-09-2024	30-09-2024	4	P	P	A	A	A	Abse	P	P	A	P	P	P	92	140	80	37	18	200	15.1	9.63	10mm/hr	RBS-246mg/dl	25	0.8	134	4	160	142	32	102	100BPM	Regular	0.08S	0.24S	POOR R WAVE PROGRESSION,DEEP QE V1-V6	0.04 s	N	ST E V1-V6	INVERTED V1-V6	520	670s	STEMI ANTEROLATERAL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	40	I	A	TVD	P	A	A	A	A
62	LAXMAN HEGGOND	49	M	FARMER	9901495274	JAMKHANDI	5476	27-09-2024	01-10-2024	5	P	A	A	A	A	Abse	P	P	A	A	A	P	56	140	80	38	20	2300	14.1	10.4	15mm/hr	RBS-231mg/dl	17	0.9	139	3.9	386	222	28	268	60BPM	BRADYCARDIA	0.08S	0.12s	DEEP Q WAVE 3,AVR	0.04 s	N	ST E V2-V6	INVERTED V1-V6,AVL	480	480s	STEMI ANTEROLATERAL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	60	I	A	DVD	A	A	A	A	A
63	MURLIDHAR DEGINAL	69	M	FARMER	9380893381	INDI	5500	27-09-2024	02-10-2024	5	P	A	A	A	A	Ab	P	P	A	A	A	A	70	140	80	37	18	368	12.4	6.25	10mm/hr	RBS-200mg/dl	29	0.8	139	4.4	150	133	24	100	75bpm	Regular	0.04S	0.12s	RSR PATTERN IN 3	0.04 s	N	ST DEP 2,3,AVF	INVERTED AVR,AVL	480	530s	NSTEMI	A	A	INFERIOR WALL HYPOKINESIA	50	I	A	TVD	A	A	A	A	A
64	BHIMANGOUDA PATIL	87	M	FARMER	6363277251	SAINIK SCHOOL	5524	28-09-2024	07-10-2024	9	P	A	A	A	A	Abse	A	A	A	P	A	A	100	170	100	38	24	5590	14.3	16.7	20mm/hr	RBS-169mg/dl	31	0.8	138	3.9	175	155	30	120	100BPM	Tachycardia	0.08S	0.12s	POOR R WAVE PROGRESSION DEEP Q WAVE V1-V3	0.04 s	LAD	ST DEP V4-V6,	INVERTED V1,V2	480 s	620s	NSTEMI	A	A	HYPOKINESIA LATERAL WALL	30	I	A	NOT DONE	P	P	A	A	A
65	SUMITA SHAHA	70	F	HOME MAKER	9972504571	CHADCHAN	5539	28-09-2024	05-10-2024	7	A	A	A	A	A	P	A	A	A	A	A	A	112	13	80	37	16	89.9	11.9	13.9	20mm/hr	RBS-149mg/dl	36	0.5	133	3.3	140	182	18	86	100BPM	Tachycardia	0.08S	0.24S	DEEP Q WAVE V1-V3	0.04 s	N	ST E V4-V6,2,3,AVF	INVERTED V1-V6,2,3,AVF	520 s	670s	STEMI INFEROLATERAL	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	45	I	A	NOT DONE	A	A	A	A	A
66	ASHOK KUMBAR	81	M	SHOPKEEPER	9972691407	GACHINAKATTI	5664	28-09-2024	02-10-2024	4	P	A	A	A	A	Ab	A	P	A	P	A	A	90	180	90	37	16	6.9	12.7	6.19	10mm/hr	RBS-233mg/dl	14	0.6	141	4.5	180	162	35	110	75bpm	Regular	0.08S	0.20S	Q WAVE 3	0.04 S	N	ST CHANGES V1-V4	NO T WAVE	440 s	490s	UA	A	A	ANTERIOR WALL MI	60	I	A	TVD	A	A	A	A	A
67	BOURAMMA HUNASHYAL	60	F	HOME MAKER	6361342248	BASAVAN BAGEWA	6142	01-10-2024	05-10-2024	4	P	P	A	A	A	Ab	P	P	A	A	A	A	94	140	90	38	24	1301.4	7.5	13.3	15mm/hr	RBS-165mg/dl	84	2	137	6.2	251	134	30	160	75bpm	Regular	0.08S	0.20S	VPCS NOTED V1-V6,2,3	0.04 s	N	ST DEP V4-V6,	INVERTED V1-V6	400 s	440s	NSTEMI	A	A	NO MOTION WALL ABNORMALITY	60	I	A	NOT DONE	A	A	A	A	A
68	BASAPPA KANOLLI	70	M	FARMER	8050972740	AT POST MANNUR TALUK	6413	03-10-2024	07-10-2024	4	P	A	A	A	A	Abse	P	P	A	A	A	P	76	130	90	37	18	23068	13.3	8.41	10mm/hr	fbs-90mg/dl	23	0.6	135	4.5	262	371	33	215	75bpm	Regular	0.08S	0.12S	deep q wave v1-v6	0.04 S	N	ST ELEVATION 3,AVF,2	INVERTED 2,V4-V6	520 s	580s	STEMI INFERIOR WALL	A	A	HPOKINESIA OF ANT ANTEROLATERAL	45	I	A	NOT DONE	A	A	A	A	A
69	SIDDAPA GUBBI	60	M	FARMER	9380141409	AT MANAGULI TQ.	6478	03-10-2024	07-10-2024	4	P	P	A	A	P	Abse	A	A	A	A	P	P	82	120	80	37	18	166.4	11.9	5.09	10mm/hr	fbs-166mg/dl	26	0.8	132	5.1	267	178	30	202	75BPM	Regular	0.08s	0.12s	Rs complexes in v4-V6	0.04 s	N	ST DEP V4V5,V6	INVERTED 2,3,AVF,V4-V6	480 s	520s	NSTEMI	A	A	HPOKINESIA OF ANT ANTEROLATERAL	45	I	A	NOT DONE	A	A	A	A	A
70	TAJUDDIN DEGINAL	45	M	SHOPKEEPER	7022680374	SHAHAPUR AGASI	6832	05-10-2024	11-10-2024	6	P	A	A	P	A	Abse	P	A	A	P	A	P	62	90	50	37	22	8583.3	12.5	19.8	48MM/HR	RBS-256mg/dl	###	2	139	3.3	152	222	32	101	75bpm	Regular	0.08S	0.12s	TALL T WAVES V3-V5	0.04 s	N	ST ELEVATION 2,3,AVF,V4-V6	HYPERACUTE YT WAVE V1-	400 s	440s	STEMI INFEROLATERAL	A	P	GLOBAL HYPOKINESIA	25	I	A	NOT DONE	P	A	A	P	A
71	MAHADEVI PUJARI	65	F	HOME MAKER	6361578255	KOLLURAGI, INDI	7893	12-10-2024	20-10-2024	8	P	A	P	A	A	Ab	P	P	A	A	A	A	80	140	80	37	18	1325	14	9.4	10mm/hr	RBS-134mg/dl	15	0.6	137	2.7	198	162	27	138	100BPM	Tachycardia	0.04S	0.12s	N	0.04 s	N	ST DEP V3-V6	INVERTED V3-V6	400 s	510s	NSTEMI	A	A	NO MOTION WALL ABNORMALITY	60	I	A	DVD	A	A	A	A	A
72	BHIMSINGH RATHOD	55	M	FARMER		VIJAPUR	9778	14-10-2024	20-10-2024	6	P	P	A	A	A	Abse	P	A	A	A	A	A	78	110	70	37	18	1800	15.3	11.8	10mm/hr	RBS-250mg/dl	32	1.5	129	4.6	198	236	35	130	75bpm	Regular	0.08S	0.12s	DEEP Q WAVES V1-V3	0.04 s	N	ST E V1-V3	INVERTED V1-V3	480 s	440s	STEMI ANTERIOR WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	35	I	A	SVD	P	A	A	A	A
73	ANIL KAMBLE	53	M	SHOPKEEPER	9945271193	NEHRU NAGAR	8151	14-10-2024	20-10-2024	6	P	P	A	A	A	Abse	P	P	A	A	A	A	96	13	80	37	18	122	11	7.3	10mm/hr	RBS-341mg/dl	19	0.8	134	3.8	204	186	26	158	100BPM	Regular	0.08S	0.12s	Q WAVE AVR	0.04 s	N	ST CHANGES V1-V5	SYMMETRIC T WAVE INVERTED	480 s	520s	STEMI ANTERIOR WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	55	I	A	SVD	A	A	A	A	A
74	RAMU NAMANE	45	M	SERVICE	9449654946	BASAVANA BAGEWA	8562	16-10-2024	19-10-2024	3	P	A	A	A	A	Abse	P	A	A	P	A	A	58	150	70	37	18	1524	11.2	8.2	10mm/hr	rbs-152mg/dl	18	0.6	134	4.2	246	202	38	166	50BPM	Bradycardia	0.08S	0.12s	N	0.04 s	N	ST E V3-V6	INVERTED DEEP V2-V6	480	430	STEMI ANTEROLATERAL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	40	I	A	NOT DONE	P	A	A	A	A

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75	HALIMA MULLA	64	F	HOME MAKER		GANGAB AUDI NEAR	8470	16-10-2024	23-10-2024	7	P	A	A	A	A	Ab se	A	P	A	A	A	A	118	150	80	37	18	2352	13.2	5.19	10mm/hr	RBS-82mg/dl	20	0.8	136	4.4	196	282	34	118	150BPM	Tachycardia	0.08S	0.12s	DEEP Q WAVES V3-V4	0.04 s	N	STE V1-V5	INVERTED V3-V5	360	560	STEMI ANTEROSEPTAL	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	30	I	A	NOT DONE	P	A	A	A	A
76	GOVINDRAO VAJANTRI	65	M	SERVICE	8105859456	SIR DESHPANDE	8926	18-10-2024	23-10-2024	5	A	P	A	A	P	P	P	P	A	A	P	P	120	140	90	38	18	337	11.5	12.7	30mm/hr	RBS-247mg/dl	56	2.3	129	6.5	254	199	20	198	100BPM	Tachycardia	0.08S	0.12s	BROAD ORS WITH NOTCH V1-V6	0.08 S	LAD	STE V1-V4	INVERTED V1-V4	440 s	560s	LBBB	A	P	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	30	I	A	TVD	P	P	P	A	A
77	MALLAMA GULED	49	F	HOME MAKER	8217095267	SOLATAGI, INDI	8764	18-10-2024	23-10-2024	5	P	A	A	A	A	Ab se	P	P	A	A	A	P	100	120	80	38	20	2063	8.9	24.7	10mm/hr	RBS--88mg/dl	33	0.8	131	4.4	196	186	35	122	75bpm	Regular	0.04S	0.12s	DEEP Q WAVES 2,AVF,AVR	0.04 s	N	STE 2,3,AVF,V2-V6	INVERTED 2,3,AVF	440 s	490s	STEMI INFEROLATERAL	A	A	HPOKINESIA OF ANTEROLATERAL WALL	35	I	A	NOT DONE	P	A	A	A	A
78	SHANKARAPPA NAVI	85	M	FARMER	9008809424	BAGALKOTE	9082	19-10-2024	25-10-2024	6	P	A	A	A	A	Ab	P	P	A	A	A	P	75	130	80	37	18	1175	13	12	25	RBS-300mg/dl	62	1.1	135	4.5	270	250	30	152	75bpm	Regular	0.08S	0.12s	N	0.04s	N	EP V2-V5 NO T WAVE		400 s	440s	NSTEMI	A	A	NO MOTION WALL ABNORMALITY	60	I	A	TVD	A	A	A	A	A
79	TARABAI DASHAVANT	62	F	HOME MAKER		CHADCHAN	9130	20-10-2024	26-10-2024	6	P	A	A	A	A	Ab se	A	A	A	A	A	P	60	160	90	37	18	1221	14	9.8	10mm/hr	RBS-246mg/dl	22	1	132	3.4	180	172	28	130	75bpm	Regular	0.08S	0.12s	N	0.04 s	N	STE V1-V6	INVERTED DEEP V2-V6	480 s	530s	STEMI ANTEROSEPTAL	P	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	35	I	A	DVD	P	A	A	A	A
80	KASTURIBAI DONUR	70	F	HOME MAKER	7204308177	JAIL DARGA	9129	20-10-2024	25-10-2024	5	P	P	A	A	A	Ab se	A	A	A	A	A	A	114	120	80	38	18	5162	13	6.4	22MM/HR	RBS-176mg/dl	18	0.9	138	3.5	260	280	28	198	150BPM	Tachycardia	0.08S	0.12s	DEEP Q WAVES V3-V4,AVF	0.04 s	LAD	STE V1-V5	INVERTED V1-V5	400 s	630 ms	STEMI ANTEROSEPTAL	A	P	HPOKINESIA OF ANTEROLATERAL WALL	30	I	A	TVD	P	A	A	A	A
81	SIDDAPPA KUDAGI	61	M	SHOPKEEPER	8660232979	A/P UPPALAD	9367	21-10-2024	25-10-2024	4	P	A	A	A	A	Ab	P	P	A	P	P	P	80	150	60	38	16	2400	13.6	7.7	10mm/hr	rbs-120mg/dl	25	0.8	137	4.4	200	192	30	156	60BPM	Regular	0.08S	0.12s	N	0.04 s	N	ST CHANGES NOTED	NO T WAVE	480 s	480 ms	NSTEMI	A	A	NO MOTION WALL ABNORMALITY	60	I	A	TVD	A	A	A	A	A
82	SIDDAPA ILAGI	55	M	FARMER	9008304526	A/P MANANKALGI, INDI	9577	22-10-2024	26-10-2024	4	P	A	A	A	A	Ab se	A	P	A	P	A	A	80	120	70	37	18	5240	11.8	16.6	25MM/HR	RBS-82mg/dl	25	0.8	138	4	223	206	22	132	75bpm	Regular	0.08S	0.12s	DEEP Q WAVES V1-V3,NOTCHED QRS V3	0.04 s	N	STE V2-V3	INVERTED V1-V4,3,AVR	440 s	490s	STEMI ANTERIOR WALL	A	P	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	35	I	A	SVD	P	A	A	A	A
83	YENKAYYA METI	52	F	HOME MAKER		VIJAPUR	8427	22-10-2024	24-10-2024	2	P	A	A	A	A	Ab se	A	P	A	A	A	A	86	120	70	37	16	1352	13.3	9.4	36MM/HR	RBS-241mg/dl	16	0.8	136	4.1	198	365	22	142	100BPM	Regular	0.08S	0.12s	DEEP Q WAVES V1-V4	0.04 s	N	STE V2-V4	INVERTED V2-V5	480 s	620 ms	STEMI ANETROSEPTAL	A	P	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	30	I	A	SVD	P	A	A	A	A
84	MAHADEVI KORI	60	F	HOME MAKER		RAMAPUR, TIKOTA	16830	05-12-2024	10-12-2024	5	P	A	A	A	A	Ab se	P	P	A	A	A	P	90	100	60	37	18	30000	12	9.52	15mm/hr	RBS-195mg/dl	20	0.8	140	4	200	275	25	130	100BPM	Regular	0.08S	0.16S	DEEP Q WAVES 2,AVF,V2-V6,NOTCHED V3,V4,2	0.04 s	LAD	STE V1-V6	INVERTED V1-V6	320 s	410s	STEMI ANTEROSEPTAL	A	P	GLOBAL HYPOKINESIA	35	I	A	SVD	P	A	A	A	A
85	SHANTAVVA BADIGER	65	F	HOME MAKER	9845632104	WADAWA DAGI,BAS	17405	09-12-2024	14-12-2024	5	A	A	A	A	A	P	P	P	A	A	A	P	122	140	80	38	18	351	12.2	15.8	15mm/hr	RBS-199mg/dl	30	0.6	139	4.7	188	156	30	111	150BPM	Tachycardia	0.08S	0.12s	N	0.04 s	N	ST DEP V1-V6	INVERTED AND DEEP	320 s	500S	NSTEMI	A	A	GLOBAL HYPOKINESIA	40	I	A	TVD	P	A	A	A	A
86	GANGAPPA HIMAKAR	47	M	FARMER		POST MALGUR,JAMKHAN	17403	09-12-2024	16-12-2024	7	P	A	A	A	A	Ab se	P	P	A	P	A	P	92	110	70	37	18	173	16.9	21.5	10mm/hr	RBS-400mg/dl	33	1.2	133	4.2	265	215	34	145	150BPM	Tachycardia	0.08S	0.12s	DEEP Q WAVES V2-V6,RBBB PATTERN-V1	0.04 s	RAD	STE V2-V6	INVERTED V2-V6	280 s	440s	RBBB	P	A	HPOKINESIA OF ANT ANTEROLATERAL	40	I	A	SVD	P	A	A	A	A
87	NINGAYYA MATAPATI	69	M	FARMER	961022431	SASANUR, TALIKOTI	17407	09-12-2024	14-12-2024	5	P	A	A	A	A	Ab se	P	P	A	P	P	A	118	180	90	37	20	2000	15.1	18.9	20mm/hr	RBS-190mg/dl	19	1	134	2.9	182	168	33	110	150BPM	Tachycardia	0.08S	0.12s	DEEP Q WAVES V1-V4	0.04 s	N	STE V1-V4,AVL	DEEP INVERTED, ASYMMET	320 s	500S	STEMI ANTEROLATERAL	A	A	HPOKINESIA OF ANT ANTEROLATERAL	40	I	A	DVD	P	A	A	A	A
88	HANAMANTH TOPANINAGOL	64	M	FARMER		VIJAPUR	17529	10-12-2024	14-12-2024	4	P	A	A	A	A	Ab se	P	P	A	P	A	A	68	130	80	37	18	150	13.7	10.3	10mm/hr	RBS-134mg/dl	22	0.8	135	4	205	193	30	100	75bpm	Regular	0.12S	0.16S	NOTCHED Q WAVE IN 3	0.04 s	N	ST DEP V2-V5	NO T WAVE CHANGES	440 s	490s	NSTEMI	A	P	HPOKINESIA OF ANT ANTEROLATERAL	55	I	A	DVD	A	A	A	A	A
89	GURUPADAPPA KADABAGAONVI	54	M	FARMER	8150027732	HEBBAL,NIDAGUNDI	17631	10-12-2024	15-12-2024	5	P	A	A	A	A	Ab se	P	P	A	P	A	A	90	90	60	37	20	27.14	14.6	17.2	15mm/hr	RBS-121mg/dl	20	0.9	137	4.4	274	143	34	182	100BPM	Regular	0.08S	0.12s	rSR PATTERN 2,3,AVF,DEEQ WAVE V1-V4	0.08 S	RAD	STE V1-V6	INVERTED V1-V6	320	410	RBBB	A	P	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	25	I	A	SVD	P	A	A	P	A
90	JAIBUNISSA FOUJI	62	F	HOME MAKER		GANGAB AVADI	17660	11-12-2024	13-12-2024	3	A	P	A	A	A	Ab se	P	P	A	A	A	P	110	130	90	37	26	351	10.9	11	35	RBS-159mg/dl	23	1.2	128	3.5	178	155	30	116	75bpm	Regular	0.04S	0.12s	Q WAVE AVL	0.12 S	N	ST CHANGES 1,2,AVL	NO T WAVE CHANGES	420	420s	UA	A	P	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	40	I	A	TVD	P	P	A	A	A
91	RAMESH HIREGAN	50	M	FARMER	7795044467	A/P BABNAGAR,TIKOTA	19038	19-12-2024	25-12-2024	6	P	P	A	A	A	Ab se	A	P	A	A	A	P	70	110	70	37	18	68.1	15.9	6.5	16	rbs-116mg/dl	26	1	143	3.9	270	145	38	150	58BPM	Regular	0.04S	0.16S	Q WAVES 1,2	0.12 S	N	ST CHANGES	BIPHASIC T WAVE V2,V3	360 s	350 ms	STEMI ANTERIOR WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	60	I	A	NOT DONE	A	A	A	A	A
92	F I KARAJAGI	61	M	FARMER	8.797E+09	TIKOTA	3E+05	02-02-2024	10-02-2024	###	p	P	A	A	A	Ab se	A	A	A	p	p	P	84	140	90	37	18	12.8	13145	26	258	47	1	127	5.1	248	183	38	36	122	70BPM	Regular	.04S	0.12S		0.20 S	N	st elevation	STE V2,3,4,5,6	364 s	460 ms	STEMI ANTERIOR WALL	A	A	HYPOKINESIA OF DISTAL ANTERIOR WALL AND APEX	45	I	A	NOT DONE	A	A	A	A	A