

**GENETIC STUDY OF PEROXISOME
PROLIFERATOR-ACTIVATED RECEPTOR
GAMMA GENE POLYMORPHISM IN ACUTE
CORONARY SYNDROME**

By

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Dissertation submitted to BLDE (Deemed to be University), Vijayapura.



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

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

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ACKNOWLEDGEMENT

On completion of this contribution of scientific document, it gives me deep pleasure to acknowledge the guidance provided by my distinguished mentors.

I have got no words to express my deep sense of gratitude and regards to my guide **Dr. BADIGER SHARANABASAWAPPA_{M.D.}**, Professor, Department of General Medicine, under whose inspiring guidance & supervision, I am studying and continuing to learn the art of medicine. His deep knowledge, devotion to work and zeal to always discover newer scientific research makes him a source of inspiration not only to me but for others too. It is because of his generous help, expert and vigilant supervision, that has guided & helped me to bring out this work in the present form. My sincere thanks to **Dr. Aravind Patil**, Principal, and **Dr. BADIGER SHARANABASAWAPPA_{M.D.}** Professor, Department of General Medicine, BLDE (DU), Shri B M Patil Medical College, Hospital and Research Centre, Vijayapura for permitting me to utilize the resources in completion of my work. My heart filled thanks to **Dr. G.S. KADAKOL**, Research Scientist, Genetics Laboratory, Department of Anatomy, BLDE (Deemed to be University), Shri B M Patil Medical College, Hospital and Research Centre, Vijayapura who is the backbone of our research and helping us in each and every step of our work.

I wish to acknowledge my all teachers and take this opportunity to express my deep sense of gratitude and sincere thanks to Professors, **Dr. R. C. Bidri**, **Dr. M. S. Mulimani**, **Dr. S. S. Devarmani**, **Dr. S. N. Buntoor**, **Dr. V. G. Warad**, **Dr. R. M. Honnutagi**, **Dr. S. M. Biradar**, **Dr. P. G. Mantur** for their supervision and timely advice. I sincerely express my gratitude to Dr. Vijaya M. Sorganvi Associate Professor in Statistics Dept. of Community Medicine, BLDE (DU), Shri B M Patil Medical College, Hospital and Research Centre, Vijayapura

I am also thankful for the support extended by **Dr. Avinash Jugati**, and **Dr. Rahul Biradar**, and all the senior residents for their valuable help and guidance throughout my course. I would be failing in my duty, if I would not acknowledge my thanks to all the patients who were kind enough to help for this study.

I would also like to thank my parents who instilled an early love of learning, and who continue to serve as role models for life **SAIFUL ISLAM** and **JUSHNARA**

BEGUM, and my younger sisters **MEHJABIN and SAMIM**, without their constant encouragement & moral support, my studies would have been a distant dream. I would like to thank the **Almighty GOD**, Creator of all things, who is the source of all knowledge and healing power, may this work serve as an instrument of His will. At last, but not the least I am thankful to all my teachers and friends who have been always helping and encouraging me all these years. I have no valuable words to express my thanks, but my heart is still full of the favors received from every person.

Dr. JAHANGIR ALAM

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LIST OF ABBREVIATIONS

ACS	: ACUTE CORONARY SYNDROME
CAD	: CORONARY ARTERY DISEASE
CABG	: CORONARY ARTERY BYPASS GRAFTING
CK	: CREATININE KINASE
CKD	: CHRONIC KIDNEY DISEASE
CS	: CARDIOGENIC SHOCK
cTN	: CARDIAC TROPONIN
dHPLC	: DENATURING HIGH-PERFORMANCE LIQUID CHROMATOGRAPHY
ECG	: ELECTROCARDIOGRAPHY
HF	: HEART FAILURE
HsTn	: HIGH-SENSITIVITY TROPONIN
HDL-C	: HIGH-DENSITY LIPOPROTEIN CHOLESTEROL
IS	: ISCHAEMIC STROKE
LDL-C	: LOW-DENSITY LIPOPROTEIN CHOLESTEROL
MACE	: MAJOR ADVERSE CARDIAC EVENTS
MI	: MYOCARDIAL INFARCTION
NSTEMI	: NON-ST SEGMENT ELEVATION MYOCARDIAL INFARCTION
PCI	: PERCUTANEOUS CORONARY INTERVENTION
PE	: PULMONARY EDEMA
PPAR γ	: PEROXISOME PROLIFERATOR-ACTIVATED RECEPTOR-GAMMA
PPRE	: PEROXISOME PROLIFERATOR RESPONSE ELEMENTS
RWMA	: REGIONAL WALL MOTION ABNORMALITY
RFLP	: RESTRICTION FRAGMENT LENGTH POLYMORPHISM
STEMI	: ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION
TC	: TOTAL CHOLESTEROL
TG	: TRIGLYCERIDES
UA	: UNSTABLE ANGINA
VCAM	: VASCULAR CELL ADHESION MOLECULE
VT	: VENTRICULAR TACHYCARDIA

ABSTRACT

ABSTRACT:

Introduction: The myocardial ischemia, manifestation includes unstable angina, non-ST segment elevation and ST- segment elevation myocardial infarction, are together referred to as acute coronary syndrome(ACS). Numerous genetic and environmental variables influence atherosclerosis, which contributes to the development of coronary heart disease. Peroxisome Proliferator-Activated Receptors control the metabolism of fats and carbohydrates. Alpha, beta/delta, and gamma are the three proteins that make up the PPAR family. A nuclear transcription factor called peroxisome proliferator-activated receptor-gamma (PPAR gamma) is involved in energy regulation as well as glucose and lipid balance. The PPAR gamma gene, which is found on chromosome 3p25, uses alternative splicing and promoter use to produce four distinct PPAR γ mRNAs. It is found that PPAR gamma C161T genes polymorphism were linked to an increasing risk of ACS.

Aim: The purpose of this study is to investigate the role of the gene PPAR Gamma C161T in the pathophysiology of acute coronary syndrome.

Materials and Methods: This study was a prospective cross-sectional study carried out in BLDE (Deemed to be University), out of 100 patients screened for acute coronary syndrome, eight patients were excluded based on the exclusion criteria and 92 patients were included in the study. These patients then underwent detailed evaluation based on clinical examination, biochemical profiles, electrocardiographic and echocardiographic changes, their venous blood samples were then taken and analysed for PPAR Gamma gene polymorphism using PCR technique. These patients were divided into two groups: Nine patients in Group A i.e. PPAR Gamma Gene mutation positive and 83 patients in Group B i.e. Gamma Gene mutation absent. All the obtained data was entered into Microsoft excel sheet for analysis

data was analysed statistically, all continuous were compared using independent t-test, non-continuous variables and categorical variables were compared using chi square test.

Results: In this study males patients were more (59.8%), the most common age group of patients were between 50 to 70 years, most of these patients presented with chest pain (88%), followed by dyspnoea (33.70%), risk factors included smoking (38.3%), tobacco chewing (27.7%), diabetes and hypertension. Out of these 9 patients showed positive mutation (Group A) in this Group A commonest ECG finding was STEMI: inferior wall and 16 of this patient showed major adverse cardiac event.

Conclusion: This study we have demonstrated a significant relationship between PPAR Gamma gene polymorphism and acute coronary syndrome. One of the Observation was presence of the disease in all age groups above 40 years and also in few with no risk factors, hence there is need for more vigilant screening for the disease and use of Genetic profiling in all patients along with other routine biochemical and radiological tests.

Keywords: Acute coronary syndrome, PPAR Gamma gene polymorphism, PPAR γ

INTRODUCTION

INTRODUCTION

Acute Coronary Syndrome (ACS) is a condition which includes spectrum of diseases that are caused by myocardial ischemia such as unstable angina, non-ST segment elevation myocardial infarction and ST segment elevation myocardial infarction. When an atheromatous plaque is disrupted, there is a disparity between supply and demand for oxygen in the cardiac tissue which results ACS[1].

The most frequent mechanism causing ACS is the rupture of atherosclerotic plaque, which leads to partial or total blockage of an epicardial coronary blood flow. When plaque is broken down, subendothelial collagen is revealed, which activates platelets and starts the coagulation cascade, which causes thrombus to develop[2]. Ischaemic chest pain is caused by a decrease in blood flow brought on by coronary blockage and/or distal embolisation of thrombus into coronary artery microcirculation. Both full and partial occlusion of the thrombus are possible. Completely occluded patients typically exhibit ST-segment elevation myocardial infarction (STEMI). Transmural infarction may occur if the blockage is not cleared out quickly[3].

The atherosclerotic plaque mostly ruptures and cause ACS due to specific anatomical features. These include a large lipid core with many inflammatory cells, a thin fibrous cap, a high production of matrix metalloproteinases, and a comparatively low number of smooth muscle cells[2,3]. Also referred to as vulnerable plaque, such plaques can evade angiographic detection, as they may not be anatomically obstructive, and may remain silent until they trigger thrombosis[4,5]. Multiple patient factors are associated with rupture of plaque and resulting in ACS and sudden death.

Acute chest pain is one of the most common diagnostic challenges in emergency medicine [6]. Acute coronary syndrome (ACS) individuals have high risk of negative outcomes and who can improve from inpatient treatment are the main focus of diagnosis. The ECG is a vital tool for assessing any patient with suspected

ACS since it offers a rapid, affordable, and easy method of identifying individuals with ST segment alterations who are likely to benefit from admission[7]. But some people who have chest discomfort with a non-diagnostic or normal ECG might potentially be at considerable chance of a negative result. Biochemical cardiac markers, particularly troponins, can identify which patients with a normal or non-diagnostic ECG are at higher risk[8].

Numerous genetic and environmental variables influence atherosclerosis, which contributes to the development of coronary heart disease. PPARs, or peroxisome proliferator-activated receptors, control the metabolism of fats and carbohydrates. Alpha, beta/delta, and gamma are the three proteins that make up the PPAR family. Leukocyte migration into endothelial cells, lipid haemostasis, monocyte and macrophage inflammatory responses, and smooth muscle cell production of inflammatory cytokines are all regulated by the PPAR family[9].

The peroxisome proliferator-activator receptor gamma nuclear receptor plays a crucial part in intermediate metabolism. A nuclear transcription factor called peroxisome proliferator-activated receptor-gamma (PPAR) is involved in energy regulation as well as glucose and lipid balance[10]. The PPAR γ gene, which is found on chromosome 3p25, uses alternative splicing and promoter use to produce four distinct PPAR γ mRNAs. It is believed that having four distinct promoters allows for more precise control over gene expression. The same protein is encoded by the mRNAs for PPAR γ 1, PPAR γ 3, and PPAR γ 4 [11–13], whereas PPAR γ 2 mRNA product has an additional 30 amino acids (exon B) at the N terminus [14].

The Pro12Ala replacement, aC> G alteration in exon B, and 161C > T (Hys477Hys) in exon 6 are two frequent polymorphisms in the PPAR γ coding area that have been extensively studied. Other authors found the Pro12Ala polymorphism associated with type 2 diabetes [15–17], insulin resistance, obesity and cardiovascular diseases, while the T allele of the 161C > T polymorphism has been shown to be associated with reduced severity of coronary artery disease (CAD), measured as number of narrowed major coronary arteries. "Apart from the genetic component,

absence excessive calorie intake and an absence of physical activity are two factors contributing to the obesity.

So, the purpose of this study is to investigate the role of the gene PPAR Gamma C161T in acute coronary syndrome.

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AIMS AND OBJECTIVES

AIM AND OBJECTIVE OF THE STUDY

A study of Peroxisome Proliferator-Activated Receptor gamma gene polymorphism in patient of acute coronary syndrome.

REVIEW OF LITRETURE

REVIEW OF LITERATURE

Globally, coronary heart disease (CHD) was responsible for about **eight**million fatalities in 2013. Although CHD-related mortality has sharply decreased in recent decades, not all demographic groups have benefited equally from this general trend. The mortality rates from CHD declined considerably in elderly patients, but less so in younger adults, especially young women[18,19].The rise in **Non-ST-Elevation Myocardial Infarction (NSTEMI)**and the decrease in ST-segment elevation myocardial infarction (STEMI), which now makes up around one-third of all ACS cases, are two aspects impacting the reduction in CHD mortality. This change over the past decade may be due to the continued widespread use of high-sensitivity troponin (**HsTn**) assays, which are not yet approved in the United States, and the change in the risk factor profile of patients with ACS, which includes a decrease in smoking and poorly controlled hypertension, younger age, and widespread use of statins, as well as an increase in diabetes mellitus, metabolic syndrome, and chronic kidney disease (CKD)[20].More than **one**million hospital admissions in the US are caused by acute coronary syndrome each year, and it continues to be a leading cause of morbidity and mortality globally.

Pathophysiology

The atherosclerotic plaque may dislodge or break, which results in partial or complete blockage of the blood supply of heart by an epicardial coronary artery, is the most common mechanism causing atherosclerotic coronary artery disease (ACS). Disruption of the plaque reveals subendothelial collagen, which triggers platelet activation and the coagulation cascade, ultimately resulting in the development of thrombus. Ischaemic chest pain is caused by a decrease in blood flow brought on by coronary blockage and/or distal embolisation of thrombus into coronary microcirculation. Both full and partial occlusion of the thrombus are possible. Completely occluded patients typically exhibit ST-segment elevation myocardial infarction (STEMI). Transmural infarction may occur if the blockage is not cleared out quickly.This explains why patients with STEMI should have early

reperfusion using either pharmacological or catheter-based techniques. Although ST-segment elevation is typically absent in patients with partially blocked coronary arteries, other ischemia-related abnormalities (such as ST-segment depression and T wave inversions) may be present). These individuals are classified as having unstable angina (UA) or non-STEMI (NSTEMI) based on whether they have symptoms of myocardial injury (an increase in troponin). The plaque caused by atherosclerosis is more likely to rupture and result in ACS because of certain anatomical characteristics [3]. These consist of a thin fibrous cap, a big lipid core with many inflammatory cells, a significant production of matrix metalloproteinases, and a comparatively low number of smooth muscle cells [2,3]. These plaques, also known as susceptible plaque, can avoid angiographic detection since they might not be physically obstructive and might not be noticeable until they cause thrombosis. [4,5] Furthermore, a number of patient characteristics may raise the risk of plaque rupture leading to ACS and unexpected death. Local shear stress, platelet hyperreactivity, systemic inflammation, and prothrombotic conditions—transient hypercoagulability brought on by smoking, dehydration, infection, cocaine, cancer, etc.—all contribute to this process [3,21]. In the absence of atherosclerotic coronary artery disease, coronary artery dissection, emboli, or spasm can also cause myocardial ischaemia and/or infarction [22]. Lastly, myocardial necrosis may arise with coronary artery bypass surgery (CABG) or percutaneous coronary intervention (PCI) manipulation of the coronary arteries.

Clinical Features

The majority of ACS patients report profound, poorly localised chest pain that may radiate to the left arm, jaw, or neck. Usually lasting more than twenty minutes, the discomfort may not go away with rest or nitroglycerin. Although this is not always a good indicator of ACS, the discomfort associated with ACS is typically worse for people who have previously experienced periods of stable angina. In addition to chest pain, patients may also exhibit "angina equivalent" symptoms, such as the most common dyspnoea, nausea and vomiting, diaphoresis, and inexplicable exhaustion. Clinical examination of coronary heart disease patient are usually as

diaphoresis, cool and damp skin, the presence of an third heart sounds or fourth heart sound, in the apex area systolic murmur may be heard (caused by mitral regurgitation due to papillary muscle dysfunction), and finding pulmonary rales due to pulmonary oedema suggest ischaemia which is impending to cardiogenic shock, even though the majority of ACS patients may have an unremarkable physical examination. These patients should be taken care in the cardiac intensive care unit and/or have early coronary angiography since, despite their apparent stability, they can deteriorate rather quickly.

Cardiac Biomarkers

Elevation of cardiac biomarkers indicates MI in patients with clinical symptoms compatible with ACS. The sensitive and specific indicators of myocardial damage are cardiac troponins T and I, which have essentially taken the role of other biomarkers. A test is considered abnormal if the cardiac troponin level is higher than the 99th percentile upper limit of the normal range for the given assay. An initial negative test should induce a second test 6 to 9 hours later because troponin may not become elevated until up to 6 hours after the commencement of myocardial necrosis. It is crucial that patients with suspected STEMI receive reperfusion therapy as soon as troponin elevation is confirmed. Two negative tests spaced 6 to 9 hours apart often rule out NSTEMI in patients with suspected ACS but not UA, which by is ACS without myocardial necrosis. Troponin levels may stay elevated for up to two weeks after the initial damage, making it challenging to diagnose reinfarction with troponin assays, despite the fact that they have significantly improved the capacity to diagnose acute infarction. In that case, creatinine kinase (CK), particularly the MB fraction, may be utilised because to its shorter half-life (3-5 days). The sensitivity and specificity of detecting ACS have been substantially enhanced by the recent introduction of very sensitive troponin assays into clinical practice, particularly early in the course of disease when conventional troponin assays may stay negative. A single-sensitive troponin I assay performed at the time of admission significantly increased diagnosis accuracy (area under the curve 0.96) in a recent trial of 1818 patients with suspected ACS when compared to a standard

troponin assay[23]. This might make it possible to identify MI in patients with suspected ACS earlier. It is crucial to stress that, irrespective of the mechanism of injury, an increase in cardiac troponins indicates myocardial injury. Myocardial ischaemia from ACS (which affects the majority of patients) or other causes unrelated to ischaemia (such as decompensated heart failure, myopericarditis, pulmonary embolism, trauma, etc.) could be the mechanism. Troponin rise should therefore be interpreted within the relevant clinical context.

PEROXISOME PROLIFERATOR-ACTIVATED RECEPTOR GAMMA GENE POLYMORPHISM

Peroxisome proliferator-activator receptor-gamma (PPAR- γ) is a nuclear receptor that is essential for intermediate metabolism. Interactions between PPAR- γ and the peroxisome proliferator response elements (PPRE) occur in the regulatory domains of retinoid X receptor in the nucleus and a number of genes that control cellular metabolism create a heterodimer. The majority of PPAR- γ expression occurs in adipose tissue, although it is also present in vascular smooth muscle cells, pancreatic beta cells, vascular endothelium, macrophages [24], and foam cells of atherosclerotic lesions[25]. However, it also seems to have an anti-inflammatory and immune-suppressive action [26,27], which may promote an antiatherogenic impact[28,29]. This makes a possible candidate gene for coronary artery disease (CAD).

The transcription factor known as peroxisome proliferator-activated receptor (PPAR)- γ is a member of the same nuclear receptor family as thyroid hormone and steroid receptors[30]. Thiazolidinediones, a new class of insulin-sensitizing antidiabetic drugs, certain fatty acids, and prostanoids all activate it [31-33]. When activated, it binds to particular PPAR-responsive DNA regions and heterodimerises with the retinoid X receptor to stimulate the transcription of several target genes[12,25]. While most tissues express the isoform PPAR-1, PPAR-2 is unique to adipose tissue and is essential for controlling adipogenic differentiation there[34]. The distinct isoforms of the PPAR-gene, which is found on chromosome 3 [35], are caused by alternative mRNA splicing. It

includes a rare mutation in the form of addition of function (Pro115Gln) which is associated with obesity but not insulin resistance [36], loss of function mutation (Val290Met and Pro467Leu) reported in patients with severe insulin resistance but normal body weight [37]. There are several known genetic variations in the PPAR-gene. These include the extremely common Pro12Ala polymorphism in PPAR-2, the silent CAC478CAT mutation [38,39]. A CCA-to-GCA missense mutation at codon 12 of exon B of the PPAR gene causes the latter. The NH2-terminal residue that characterizes the adipocyte-specific PPAR-2 isoform is encoded by this exon. First discovered in 1997 [40], the Pro12Ala polymorphism in PPAR-2, the subject of this research, has unusual allele frequencies of 12% in Caucasians, 10% in 8% of Samoans, 4% of Japanese, 3% of African-Americans, 2% of Nauruans, 1% of Chinese, and Native Americans [41,42]. In the most prevalent ethnic group, Caucasians, this corresponds to a carrier prevalence of the polymorphism of about 25%.

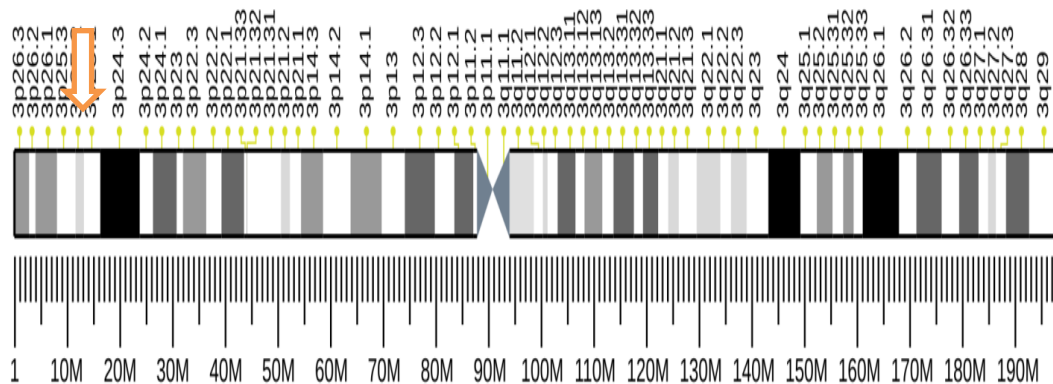


Fig 1: Cytogenetic Location of Peroxisome Proliferator-Activated Receptor Gamma

In a Japanese population, carotid artery intima media thickness is associated with the Pro12Ala polymorphism in 2, and it was significantly smaller in the Ala12allele group.[43]Ala for Pro substitution in the 2 gene decreased the incidence of acute myocardial infarction, demonstrating that gene polymorphism is also linked to a lower risk of myocardial infarction[44]. But among Chinese people, the Pro12Ala polymorphism is not connected to type 2 diabetes. Population, and in our previous

study (unpublished data), it was not associated with CAD. It was recently found that, irrespective of lipid abnormalities and obesity, a genetic variant (C161T) in exon 6 of was associated with a decreased risk of CAD[45].

At the transcriptional level, the gene controls the production of proinflammatory transcription factors such AP-1, STAT, and NF- κ B. AP-1, STAT, and NF- κ B regulate the transcription of reporter constructs including promoters for proinflammatory genes, including matrix metalloproteinases (MMPs) and tumour necrosis factor α (TNF- α). activation counteracts these transcriptions. It has been demonstrated that activated inhibits the production of TNF- α and MMPs in human monocytes and mouse macrophages[46,47]. Atherosclerosis development, atherosclerotic plaque rupture, and the onset of acute coronary syndrome (ACS) are all significantly impacted by proinflammatory cytokines. MMPs use the fibrous cap to break down the matrix components in susceptible plaques. One member of the MMP family, matrix metalloproteinase 9, is a crucial cytokine that encourages the rupture of susceptible plaques and is abundantly expressed in atherosclerotic plaques.[48,49] Tumor necrosis factor α is essential to the onset of atherosclerosis and contributes to lipid metabolism, inflammation, and insulin resistance[50,51]. Given that proinflammatory cytokines and polymorphism are linked to the development of CAD and that the gene mediates proinflammatory cytokines at the transcriptional level, the goal of the current study was to determine whether gene C161T substitution is linked to a lower risk of CAD and a lower expression of proinflammatory cytokines. To this end, we measured the plasma levels of MMP-9 and TNF- α as well as the C161T substitution in our well-characterized hospital-based patients whose coronary arterial status was angiographically documented.

Evangelistiet al in 2009 used dHPLC (denaturing high-performance liquid chromatography), heteroduplex analysis, direct sequencing, or restriction fragment length polymorphism (RFLP) analysis to test for mutations in 202 Italian patients with ACS and 295 healthy Italian people. PPAR γ genetic variations may influence

the susceptibility to atherosclerotic disorders, as evidenced by the preventive function of the 93695C > T polymorphism in the PPAR γ promoter in ACS[52].

Yilmaz-aydogan et al in 2011 examined the potential relationship between PPAR- γ 2 gene polymorphisms and blood lipid levels and coronary heart disease (CHD) incidence in a Turkish population that was prospectively characterized for the presence or absence of Type 2 diabetes. In CHD patients with diabetes, the -C161T CC homozygote genotype was associated higher rates of CHD than the T allele carriers (CT+TT) (OR:1.9510, 95%CI: 1.115–3.415, P = 0.0190), but the -P12A polymorphism was not associated to a highrisk of CHD (P >0.05). Serum HDL-C levels were found as low in controls with the P12A heterozygote than in those with the P12P homozygote (P = 0.002). In the diabetic CHD patients, the CT heterozygote genotype have high serum triglycerides than the CC homozygote genotype. They proposed that the C161T polymorphism's homozygote CC genotype may be linked to a higher risk of CHD, particularly in diabetic patients. They found that in CHD patients with diabetes, the C161T CT heterozygote genotype had a negative impact on the serum lipid profile; this effect was lessened when the P12P homozygote genotype was present[53].

Wu et al in 2012sought to assess the link more precisely and carried out a thorough meta-analysis. MEDLINE, Embase, CNKI, Wanfang, and CBM were used to screen publications authored in either Chinese or English. Eleven studies with 2,853 controls and 3,020 cases had their data retrieved. The conflicting results of the different studies could be combined using a random-effects model that addressed publication bias and between-study heterogeneity. Egger's linear regression test and the funnel plot approach did not reveal any overt publication bias (t = -0.11, P = 0.913). When combined, our findings showed that the C161T polymorphism may have a moderately protective impact against the development of CAD in Chinese people, but not in Caucasians[54].

Chehaibi et al in 2014 examined for the first time the connection between patients with type 2 diabetes mellitus (T2DM) and the C161T polymorphism and their risk

of ischaemic stroke (IS). Participants in this study were 196 IS patients (117 with diabetes and 79 without) and 192 controls. The PCR-RFLP technique was used to genotype C161T. It was discovered that controls have a larger 161T allele than IS patients (with or without T2DM) as compared to the C allele. Furthermore, CC homozygote carriers had substantially greater levels of triglycerides (TG) and ApoB than T allele carriers. These findings suggest that by modifying adipose metabolism, particularly TG and ApoB in IS patients, C161T of may lower the risk of IS[55].

Yufeng et al in 2016 sought to evaluate the relationship between PPAR polymorphisms and the risk of CHD in a methodical manner. We were particularly interested in the C161T polymorphism since it had distinct impacts on the risks of CHD and ACS. To assess the relationship between CHD risk and this polymorphism, a case-control research involving 446 participants was carried out. All PPAR polymorphisms were evaluated by meta-analyses. Overall odds ratios (ORs) were estimated using either a fixed-effects model or a random-effects model. PPAR-alpha intron 7G/C and L162V, PPAR-delta +294T/C, and PPAR-gamma C161T polymorphisms may influence CHD susceptibility, according to the data, and C161T polymorphism may have distinct impacts on CHD and ACS[56].

Oladi et al in 2015 examined the relationship between 787 people's PPAR- γ C1431T polymorphism with CAD and dyslipidaemia. Compared to CC-carriers, patients with the CT or CT+TT genotype had a higher risk of developing CAD (adjusted odds ratio: 2.03; 95% CI: 1.01-4.09; $p = 0.046$). In contrast, the group with a positive angiography had a higher incidence of the CT genotype in the general population. Additionally, in the first population sample of patients with a positive angiography, CT+TT genotypes were linked to a modified fasted lipid profile in contrast to the group with a negative angiogram. Serum C-reactive protein, fasting blood glucose, and triglycerides were all markedly elevated in angiogram-positive patients with the T allele. They discovered that in people with angiographically diagnosed CAD, the PPAR- γ C1431T polymorphism was substantially linked to their fasting serum lipid profile. Additional research is necessary to examine the relationship between this polymorphism and coronary

artery disease, as mounting evidence points to the involvement of PPAR- γ polymorphisms in CAD[57].

Wei et al in 2016 aimed to determine if PPAR γ C161T was connected to lipid levels and ischemic stroke brought on by large-artery atherosclerosis (LAA) in a Guangdong province Han Chinese community. The restriction of the polymerase chain reaction 149 LAA ischemic stroke patients and 125 healthy controls had their genotype PPAR γ C161T examined using the fragment length polymorphism (RFLP) technique. To identify risk factors for LAA ischaemic stroke, a logistic regression analysis was performed. Relationships with the PPAR γ C161T genotype, total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) were also investigated. PPAR γ C161T CT/TT was not a risk factor for LAA ischaemic stroke on its own, however it was linked to lower blood TC and LDL-C levels and LAA ischaemic stroke in this Han group[58].

Arat et al in 2020 sought to examine the relationship between proliferator-activated receptor-gamma (PPAR- γ) proline to alanine substitution (Pro12Ala), resistin-420 cytosine/guanine (C/G), and leptin glutamine to arginine substitution (Gln223Arg) polymorphisms in obese ACS patients in the Turkish community. This study comprised 42 healthy controls and 50 obese individuals who were also diagnosed with ACS. The techniques of agarose gel electrophoresis and restriction fragment length polymorphism in the PCR were used to examine these polymorphisms. The results of this study show that while the Resistin-420 C/G mutation GC genotype and the PPAR- γ Pro12Ala mutation Pro/Pro genotype may be protective factors against ACS in obese people, the PPAR- γ Pro12Ala polymorphism Pro/Ala genotype is a risk factor for ACS in obese people[59].

Cheng et al in 2021 assessed the relationship between IS risk and polymorphisms in the PPAR- γ gene. The IS PPAR- γ increase rs1801282 C/G and rs3856806 C/T polymorphisms was evaluated by using the pool association of odd ratio (ORs) and its 95.0 % confidence interval (CI). In addition, sensitivity analyses, publication

bias, cumulative analyses, and the heterogeneity test were performed. Their research concluded that the PPAR- γ rs1801282 C/G polymorphism most likely contributes significantly to the occurrence of IS. Further research should be done in the future to confirm the findings[60].

Kemanci et al in 2022 assessed clinically the association between acute coronary syndrome and polymorphisms in the alpha and gamma genes of the peroxisome proliferator-activated receptor. The groups were compared in terms of PPAR gamma C161T polymorphisms. The patient group had a greater CT heterozygous rate (74%) than the control group (7%). In contrast to the control group (0.03), the sick group had a higher prevalence of the T allele (0.37). Comparing the PPAR alpha L162V polymorphism, the sick group included 19 VV homozygous individuals while the control group had none. ACS were found to have a statistically greater V allele ($p < 0.01$). The results showed that increased polymorphisms in the PPAR alpha L162V and PPAR gamma C161T genes were linked to an increasing risk of ACS[61].

MATERIALS AND METHODS

MATERIALS AND METHODS

SOURCE OF DATA

This study was carried out in the Department of General Medicine, BLDE (Deemed to be University) Shri B M Patil Medical College, Hospital and Research Centre, Vijayapura. The study was conducted from May 2023 to December 2024 on 100 patients admitted to our hospital with acute coronary syndrome. This study was conducted after obtaining approval from the institutional ethical committee. Patients were explained about the procedure in detail and consent was obtained for the same.

- Study Design: Prospective cross-sectional study.
- Study Period: One and half years from May 2023 to December 2024
- Sample size: 92

Using JMP SAS 16 software for calculating the Sample size, Assuming the expected population standard deviation to be of TG (mmol/L) is 0.59, this study requires a sample size of 92 in order to predict a mean with 95% confidence interval and a precision of 0.13, done by using the t-distribution [61].

PATIENT SELECTION

INCLUSION CRITERIA:

- All patients more than 18 years admitted with Acute Coronary Syndrome

EXCLUSION CRITERIA

- Patients with Valvular heart disease and cardiomyopathy
- Patients with congenital heart disease

METHODOLOGY:

INITIAL ASSESSMENT

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 PPAR Gamma gene polymorphism.

DETECTION OF PPAR GAMMA 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 PPAR Gamma gene polymorphism.

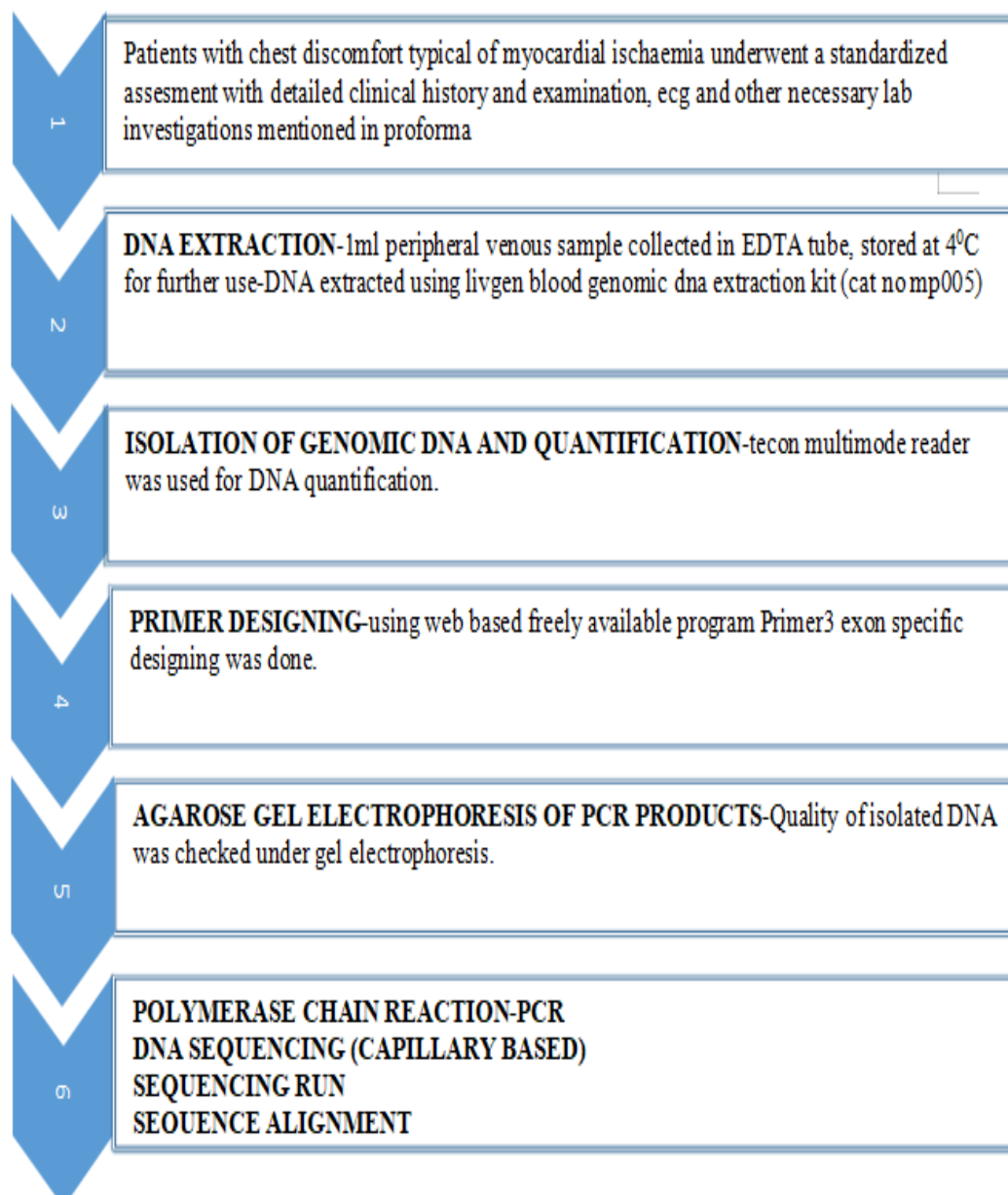


Figure 2. Depicts process of gene sequencing from collected blood samples

DNA EXTRACTION: 1 ml of peripheral venous blood samples were collected and stored in the EDTA coated vacutainers and stored at 4⁰ 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 processed for DNA quantification.

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.

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 are

Table No. 1:Primer for PPAR GAMMA GENE

Primer name	Forward primer	Reverse primer	Product size	Tm
PPAG 1 EXON	CAA GAC AAC CTG CTA CAAGC	TCC TTG TAG ATC TCC TGC AG	197bp	52 ⁰

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).

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 72degrees centigrade for one minute, final extension at 72 degrees centigrade for five minutes and hold at 40 degree centigrade.

Table No. 2: The cycle sequencing conditions

Process	Temperature (°C)	Time
Initial. Denaturation	98	5 mins
Denaturation	98	30sec
Annealing	Primer Dependent	30sec
Elongation	72	1min
Renaturation	72	5 mins
Hold	4	

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™ terminator v3.1 was used for cycle sequencing (Applied Biosystems, USA) following the manufacturer's guidelines.

Agarose Gel Electrophoresis of PCR Products:

Gel electrophoresis is one of the molecular biology techniques used to separate DNA and RNA depending on the length of fragments. The amplified PCR products were first resolved on a 1% agarose gel in 1X TAE buffer to verify amplification.

DNA bands were visualized under UV illumination using a gel documentation system (Figure 3)

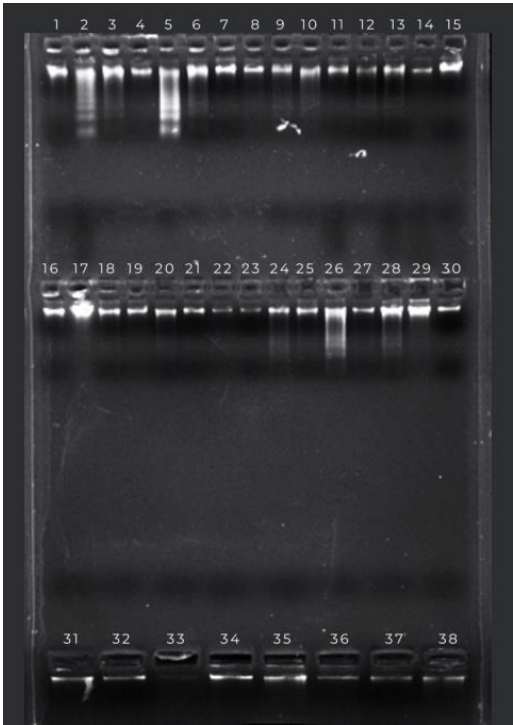


Figure 3: AGAROSE GEL IMAGE OF GENOMIC DNA OF ACUTE CORONARY SYNDROME SAMPLES.

Table No. 3: Standardized master mix conditions for sequencing

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

Sequencing Run

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

Sequence Alignment

The generated sequences were aligned to their respective reference sequences with the use of Variant reporter software (ABI v1.1). It performs sequence comparisons for novel mutations, known variants, insertions, and deletions. The results of the variant reporter were tabulated in PDF format as the default program of the software. Here, we used this technique to check the isolated genomic DNA from whole blood. In all the 92 acute coronary syndrome samples as shown in figure 9 confirmed the presence of genomic DNA and the same samples were taken for quantification based on Nanodrop.

RESULTS

RESULTS

Total of one hundred patients with acute coronary syndrome were taken into the study. Eight patients were excluded based on exclusion criteria. Five patients have valvular heart disease and three have cardiomyopathy. Ninety two patients were studied for PPAR gamma gene polymorphism.

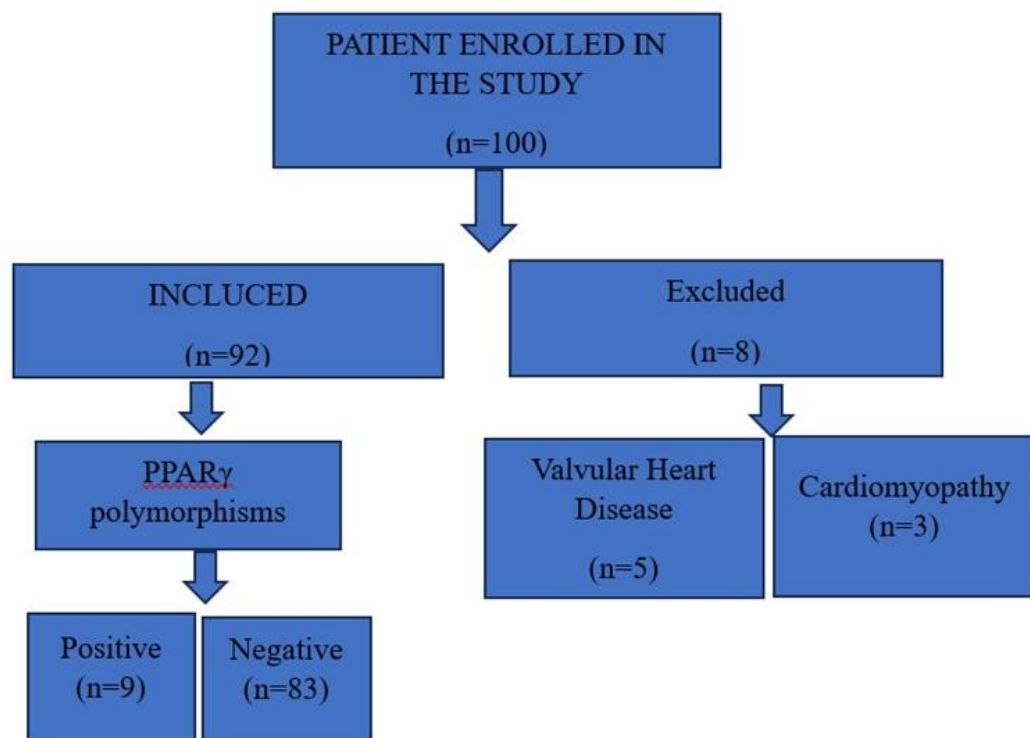


Figure 4: Flowchart showing included and excluded cases in this study

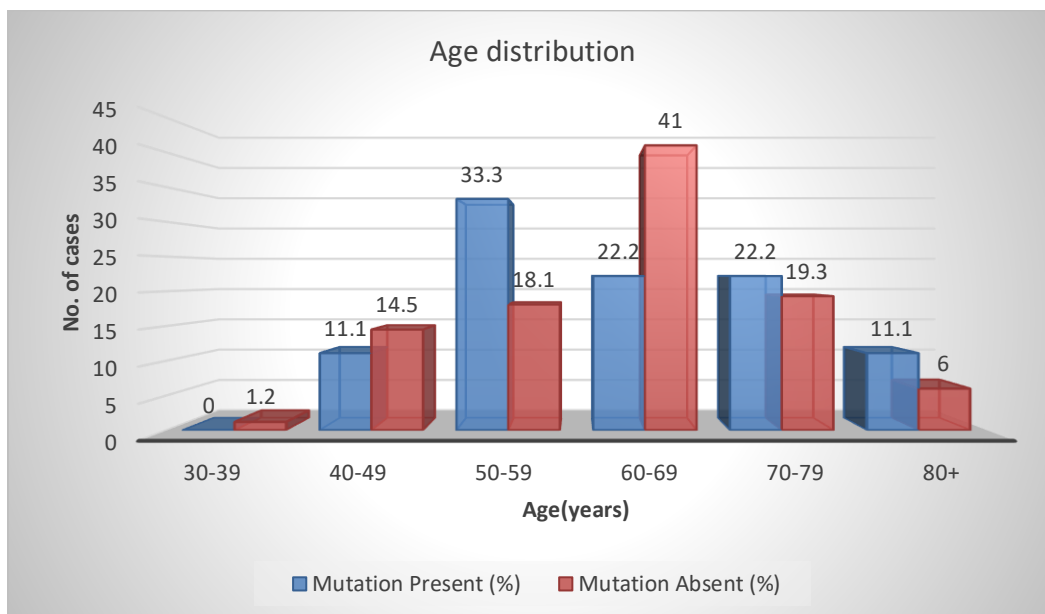
AGE DISTRIBUTION

The 92 patients were grouped with an age frequency. In group PPAR gamma gene mutation present patients between the age 41-50 years were 1 (11.1%), patients between the age 51-60 years were 3(33.33%), patients between the age 61-70 years were 2(22.2%), patient between the age 71-80 years were 2(22.2%) and above the age 80 years is 1(11.1%). In group PPAR GAMMA mutation absent patients aged between <40 years was 1(1.1%%), patients between the age 41-50 years were

12(14.5%), patients between the age 51-60 years were 15 (18.10%), patients between the age 61-70 years were 34(41.0%), patients between the age 71-80 years were 16(19.3%) and patients above 80 years were 5(6.0%). The group A was present in almost all patients age group. The most common age group for ACS in our study is 61-70 years, as described in Table 5, Graph 1.

Table No. 4: Distribution of Patients According to Age

Age (Years)	PPAR GAMMA MUTATION			Chisquare test	Significant value
	Group A (n=9)	Group B (n=83)	Total (n=92)		
18-40	0	1	1	2.226	P=0.817
	0.0%	1.2%	1.1%		
40 – 49	1	12	13		
	11.1%	14.5%	14.1%		
50 – 59	3	15	18		
	33.3%	18.1%	19.6%		
60 – 69	2	34	36		
	22.2%	41.0%	39.1%		
70 – 79	2	16	18		
	22.2%	19.3%	19.6%		
80+	1	5	6		
	11.1%	6.0%	6.5%		
Total	9	83	92		
	100.0%	100.0%	100.0%		

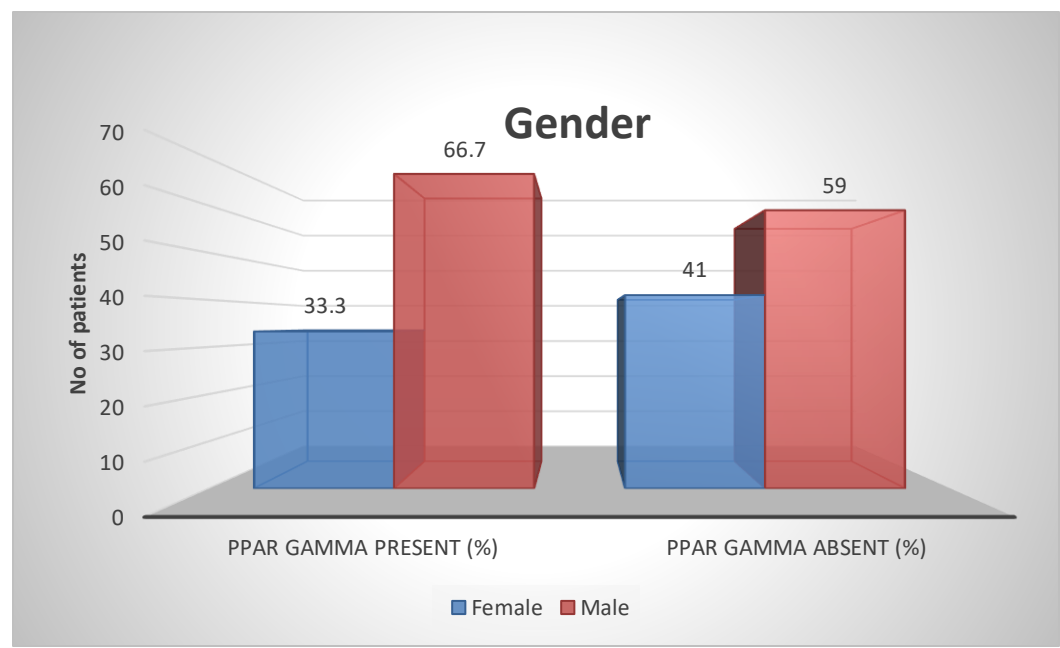
Graph 1: Distribution of Patients According to Age**SEX DISTRIBUTION**

Out of 92 patients in the study, 55 patients (59.8%) were male and 37 patients (40.2%) were female. In this study male patients were more than females as shown in table10. In group A 6 (66.7%) patients were male and 3 (33.3%) females; while 49(59.0%) patients were male and 34 (41%) were female in group B as shown in Table 06, Graph 2.

Table No. 5: Distribution of Sex Among All Cases

Gender	PPAR GAMMA MUTATION			Chi-square test	Significant value
	Group A	Group B	Total		
Female	3	34	37	.197	P=0.657
	33.3%	41.0%	40.2%		
Male	6	49	55		
	66.7%	59.0%	59.8%		
Total	9	83	92		
	100.0%	100.0%	100.0%		

Graph 02: Distribution of Sex Among All Cases

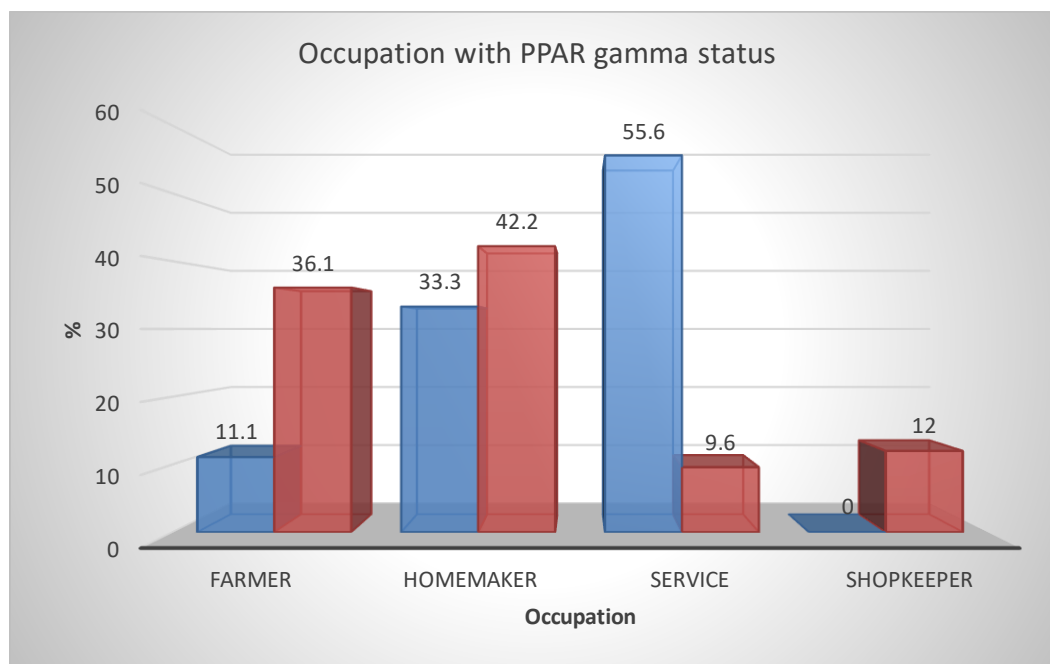


DISTRIBUTION OF PATIENTS ACCORDING TO OCCUPATION

In group A there were 5 (55.60%) service employee followed by housemaker 3 (33.3%) and Farmer 1 (11.1%). while in, group B farmers- 30(36.1%), housewife- 35 (42.2%), shopkeeper- 10 (12.0%) and service employee- 8 (9.6%). The most common occupation associated with Group A in this study was service employee, followed by housewife, farmer and shopkeeper as depicted in Table 7, Graph 3.

Table No. 6: Distribution of Occupation between study groups

Occupation								
Occupation	Group A	(%)	Group B	(%)	Total	(%)	CHI SQUARE	P-VALUE
Farmer	1	11.10	30	36.10	31	33.70	14.863	0.002
Homemaker	3	33.30	35	42.20	38	41.30		
Service	5	55.60	8	9.60	13	14.10		
Shopkeeper	0	0.00	10	12.00	10	10.90		
Total	9	100.00	83	100.00	92	100.00		

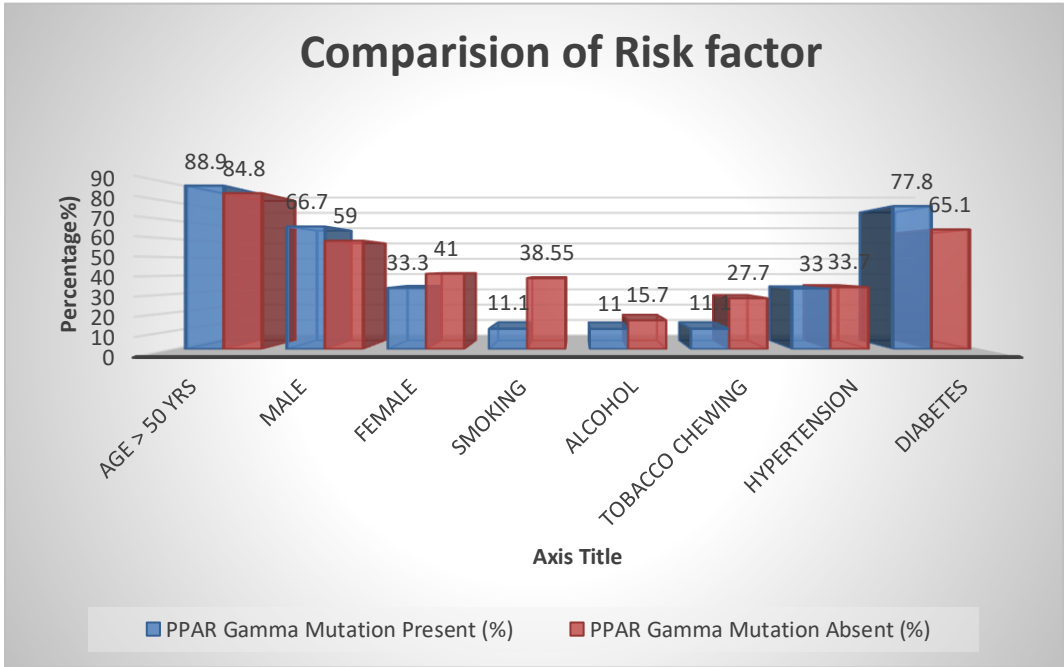
Graph 3: Distribution of Occupation between study groups**DISTRIBUTION OF PATIENTS ACCORDING TO RISK FACTORS:**

Among risk factors, out of 92 patients in the study, 8 patients (88.90%) in group A compared to 70 patients (84.80%) in group B were aged more than 50 years. Male sex was seen in 6 patients (66.70%) compared to 49 patients (59.00%) in group B. Smoking habit was seen in 33 patients of which 1 patient (11.10%) are in group A and 32 patients (38.5%) in Group B. Tobacco chewing was seen in 24 patients, of which 1 patient (11.10%) from group A and 23 patients (27.70%) in group B. Alcohol consumption was present in 1(11.10%) patient from group A and 13(15.70%) of only group B.

Table No. 7: Risk Factors

Risk factors		Group A		Group B		p value
		n=9	(%)	n=83	(%)	
Non-modifiable	Age>50yrs	8	88.90	70	84.80	0.817
	Sex					
	Male	6	66.70	49	59.00	0.9057
	Female	3	33.30	34	41.00	
Modifiable	Smoking	1	11.10	32	38.55	0.8452
	Alcohol	1	11	13	15.70	0.909
	Tobacco Chewing	1	11.10	23	27.70	0.7959
	Hypertension	3	33	28	33.70	0.1786

Graph 4 : Risk Factors



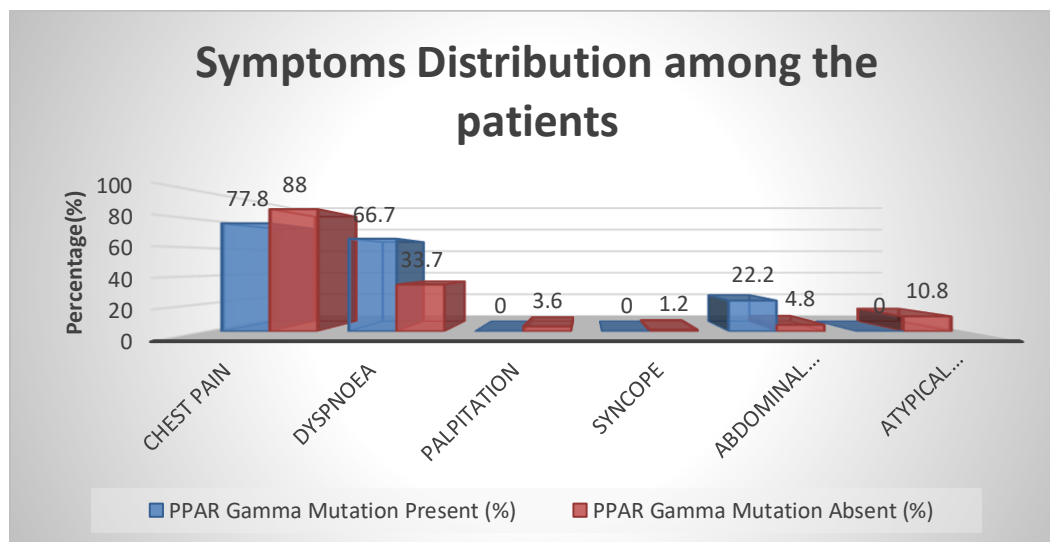
DISTRIBUTION OF PATIENTS ACCORDING TO SYMPTOMS:

In this study, as shown in Table 9, Graph 5, in both group A and group B the most common symptom was chest pain (77% vs 88.0%), followed by dyspnea (66.7% vs 33.70%), abdominal pain (22.2% VS 4.8%) where as other symptoms with patients presented are syncope, abdominal pain, and atypical symptoms of acute coronary syndrome.

Table No. 8: Symptom Distribution Among Patients

Symptom Distribution Among Patients					
Symptom	Group A (n=9)	%	Group B (n=83)	%	P-value
Chest Pain	7	77.80%	73	88.00%	0.237
Dyspnoea	6	66.70%	28	33.70%	0.052
Palpitation	0	0.00%	3	3.60%	0.562
Syncope	0	0.00%	1	1.20%	0.741
Abdominal Pain	2	22.20%	4	4.80%	0.045
Atypical Manifestation	0	0.00%	9	10.80%	0.716

Graph 5: Symptom Distribution Among Patients



DISTRIBUTION OF PATIENTS ACCORDING TO ECG FINDINGS

In our study ECG diagnosis distribution among patients with and without the PPAR Gamma Mutation have shown the following differences:

The patients with ACS STEMI in Inferior Wall is Significantly shows Higher in Mutation Present Group about **66.7%** of patients with the mutation had STEMI Inferior Wall, compared to only **8.4%** in the mutation-absent group.

The group PPAR Gamma Mutation absent STEMI Anteroseptal Wall is more common that is 22.9% whereas only 11.1% of mutation-present patients have STEMI Anteroseptal Wall myocardial infarction.

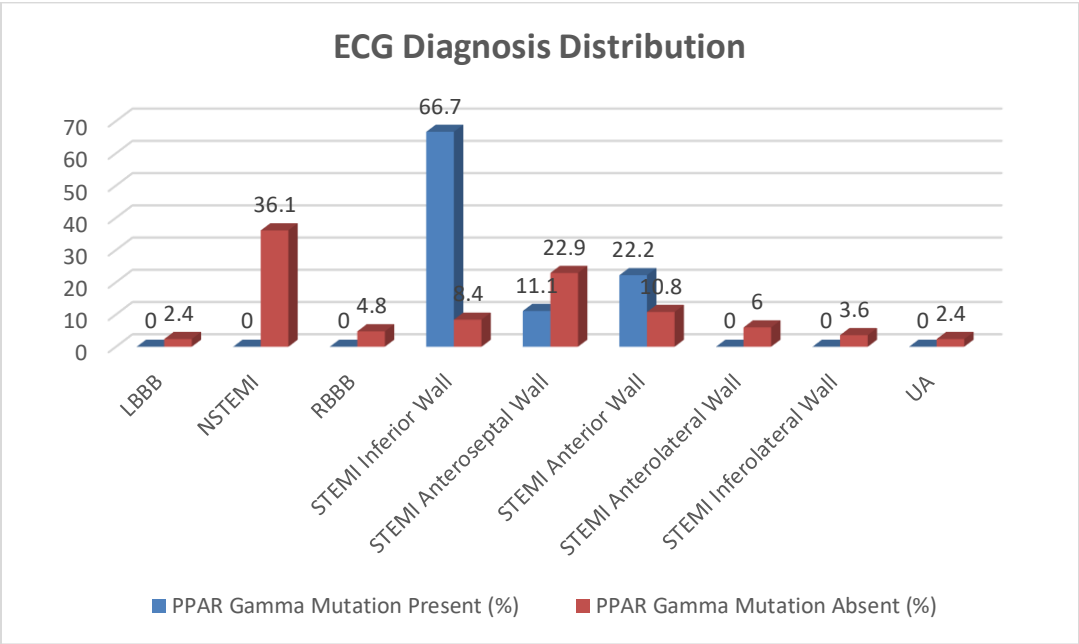
No Cases of NSTEMI or LBBB in Mutation-Present Patients.

Conditions such as NSTEMI (36.1%) and LBBB (2.4%) were only observed in mutation-absent patients

Table No 09: ECG Diagnosis Distribution

ECG Diagnosis Distribution						
ECG Diagnosis	Group A (n=9)	(%)	GroupB (n=83)	(%)	Total (n=92)	(%)
LBBB	0	0.00	2	2.40	2	2.20
NSTEMI	0	0.00	30	36.10	30	32.60
RBBB	0	0.00	4	4.80	4	4.30
STEMI Inferior Wall	6	66.70	7	8.40	13	14.10
STEMI Anteroseptal Wall	1	11.10	19	22.90	20	21.70
STEMI Anterior Wall	2	22.20	9	10.80	11	12.00
STEMI Anterolateral Wall	0	0.00	5	6.00	5	5.40
STEMI Inferolateral Wall	0	0.00	3	3.60	3	3.30
UA	0	0.00	2	2.40	2	2.20
Total	9	100.00	83	100.00	92	100.00

Graph 6 : ECG Diagnosis Distribution

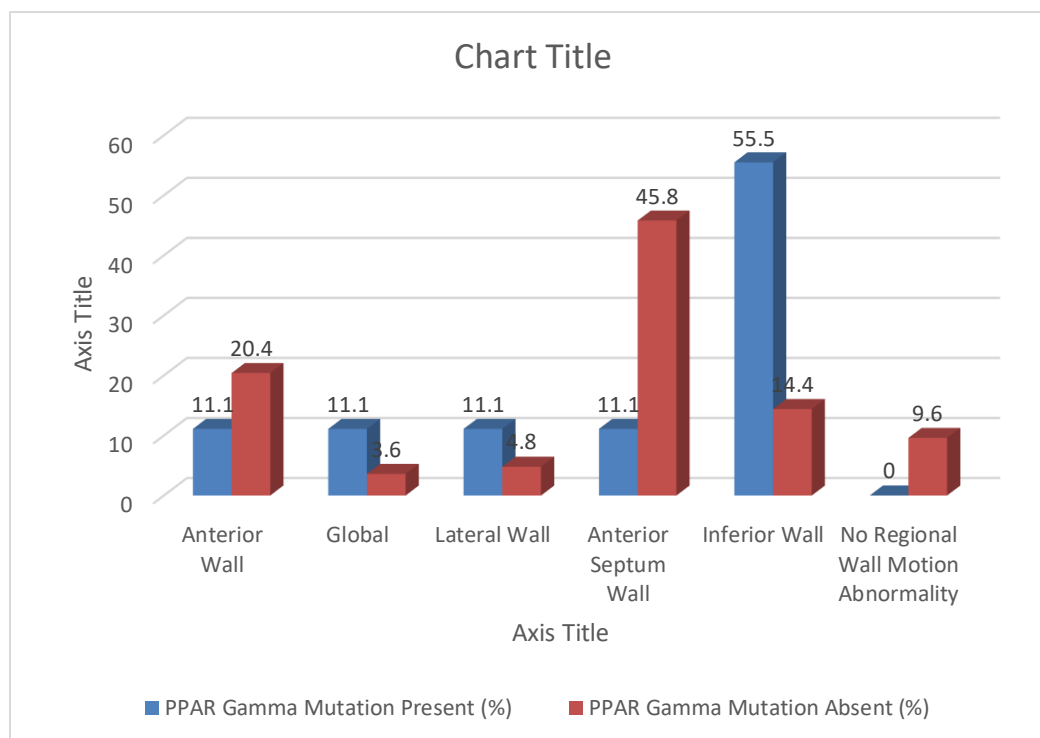


DISTRIBUTION OF PATIENTS ACCORDING TO ECHOCARDIOGRAPHIC VARIABLES:

In our study of 92 patients, echocardiographic parameters were analysed. Out of 9 patients in group A, 5 patients (55.5%) had inferior wall, 1 patient (11.1%) had antero-septal wall, 1 patient (11.1%) had lateral wall, 1 patient (11.1%) had anterior wall, and 1 patient (11.1%) had global wall hypokinesia. While out of 83 patients in group B with Anterior Wall 17 patient (20.4%) Global Hypokinesia: 3 patients (3.6%), Hypokinesia of lateral Wall: 4 patients (4.80%) , Hypokinesia of Inferior Wall: 12 patients (14.40%) , Hypokinesia of Anterio-Septal: 38 patients (45.8%) and No Regional Wall Motion Abnormality: 8 patients (9.6%). In this study most commonly, there was Hypokinesia of the Anterior Wall and Septum, affecting 39 patients (41.0%) as shown in Table 11, Graph 7.

Table No 10:2D Echocardiogram Regional Wall Motion Abnormality

2D Echo Regional Motion Wall Abnormality	Group A (n=9)	%	Group B (n=83)	%	Total (n=92)	%
Anterior Wall	1	11.10	17	20.40	18	31.50
Global	1	11.10	3	3.60	4	4.30
Lateral Wall	1	11.10	4	4.80	5	5.40
Anterio Septum wall	1	11.10	38	45.80	39	42.10
Inferior Wall	5	55.50	12	14.40	17	18.50
No Regional Wall motion Abnormality	0	0.00	8	9.60	8	8.70
Total	9	100.00	83	100.00	92	100.00

Graph 7 :2D Echocardiogram Regional Wall Motion Abnormality

QUANTIFICATION OF GENOMIC DNA

We used Tecon multimode reader for the quantification of genomic DNA. For double stranded DNA, an Optical Density (OD) of 1 at 260 nm correlates to a DNA concentration of 50 ng/μl, so that DNA concentration can be easily calculated from OD measurements” as shown in table no. 11

Table No.11: Quantification of DNA Samples of Acute Coronary Syndrome

Sl.No. of DNA samples	OD at 260/280	Concentration in ng/μl	Sl.No. of DNA samples	OD at 260/280	Concentration in ng/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
22	1.89	85.5
23	1.96	73.9
24	3.05	57
25	2.01	81
26	2.24	125
27	2.09	137
28	1.76	104
29	1.96	92
30	1.58	93
31	1.86	54
32	1.75	65.4
33	1.40	44.5
34	1.90	70.3
35	1.57	95.3
36	1.98	64
37	1.84	82
38	1.92	73
39	1.65	68
40	1.79	111
41	1.85	64
42	1.81	66
43	1.68	54.2
44	1.85	63.5
45	1.74	56.2
46	1.56	48.2

67	1.54	96.7
68	1.76	58.4
69	1.74	55.6
70	1.63	79.2
71	1.64	81.2
72	1.56	75.2
73	1.91	1.5.3
74	1.93	112.0
75	1.74	145.2
76	1.85	49.2
77	1.81	56.8
78	1.49	78.5
79	1.55	64.2
80	1.66	86.2
81	1.74	69.2
82	1.72	54.9
83	1.71	58.1
84	1.73	75.1
85	1.78	67.2
86	1.79	57.1
87	1.80	45.2
88	1.70	47.2
89	1.76	63.2
90	1.84	89.2
91	1.83	87.1
92	1.83	47.2

Table No. 12: DISTRIBUTION OF PPARGAMMA GENE POLYMORPHISM IN STUDY SAMPLE:

PPAR GAMMA GENE MUTATION	No. of Patients	Percentage (%)
Group A	9	9.8
Group B	83	92.2
Total	92	100.00

The genetic analyses for PPAR GAMMA gene Polymorphism was done in genetic research laboratory, Out of 92 patients analyzed only 9 patients showed association for PPAR gammagene mutation. In our study of 92 patients with acute coronary syndrome, 9 patients, 3 males and 2 females showed positive mutation for this PPAR Gamma gene. They showed specific in PPAR gamma gene polymorphism (rs3856806, c.1341 C>T, p.H447H) in 9 out of 92 patients (9.8%). This synonymous variant was found in heterozygous condition and it is classified as benign or likely benign.

Mutation analysis: PPAR gamma Gene The mutation analysis revealed that 9 patients (9.8%) had a specific PPAR gamma gene polymorphism, rs3856806 (c.1341 C>T, p.H447H), which is a synonymous mutation. All nine cases were found to be heterozygous for this polymorphism. This variant is categorized as benign or likely benign, suggesting it does not contribute significantly to disease phenotype. The details are shown in table 13.

Table No. 13: MUTATION ANALYSIS: PPAR GAMMA GENE

SL No	gDNA position	cDNA position	aa position	Status	Variant type	Condition	Phenotype/ Disease
1	g. 146,988 C>T	c. 1341 C>T	p. H 447 H	rs3856806	Synonymous variant	Heterozygous	Benign and likely benign

Agarose Gel Electrophoresis of PCR Products:

Gel electrophoresis is one of the molecular biology techniques used to separate DNA and RNA depending on the length of fragments. 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™ terminator v3.1 was used for cycle sequencing (Applied Biosystems, USA) following the manufacturer's guidelines. The presence of a band in the C allele-specific primer lane indicated the presence of the C allele, whereas a band in the T primer lane confirmed the T allele (Figure 5).

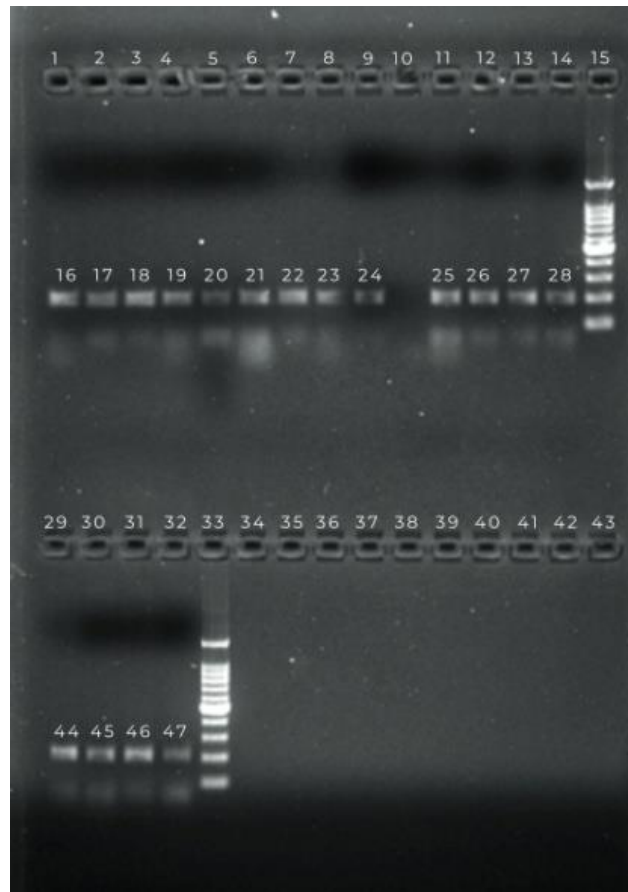


Figure 5 :AGAROSE GEL ELECTROPHORESIS IMAGE OF AMPLIFIED PRODUCTS OF GENE.

An electropherogram was obtained for the mutation-positive patients. The sequencing analysis confirmed the presence of a C>T substitution consistent with the synonymousvariant as depicted in Figure 6.

Figure 6: THEELECTROPHOROGRAM SHOWS THAT HETEROZYGOUS MUTATION C>T WITH SYNONYMOUS VARIANT



Figure 6a

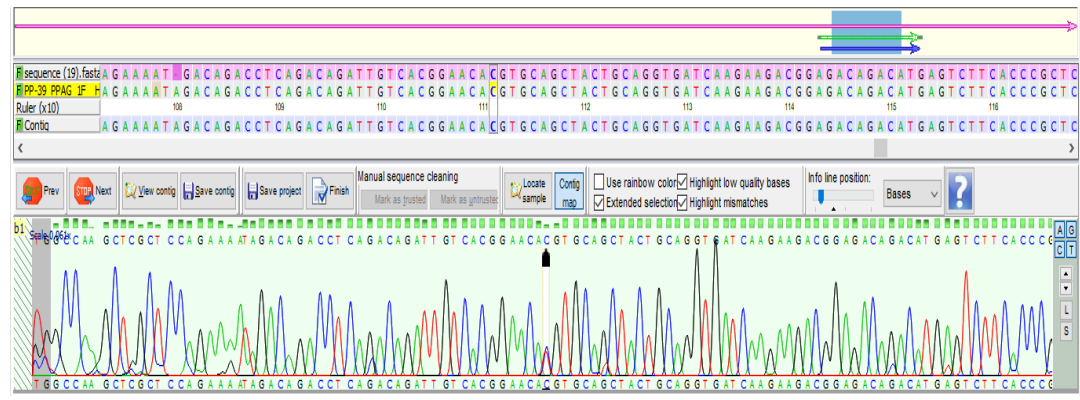


Figure 6b

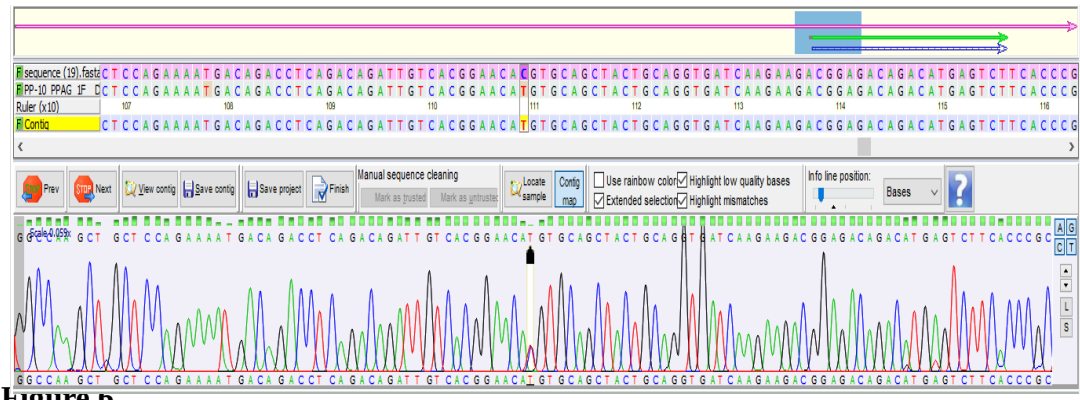


Figure 6

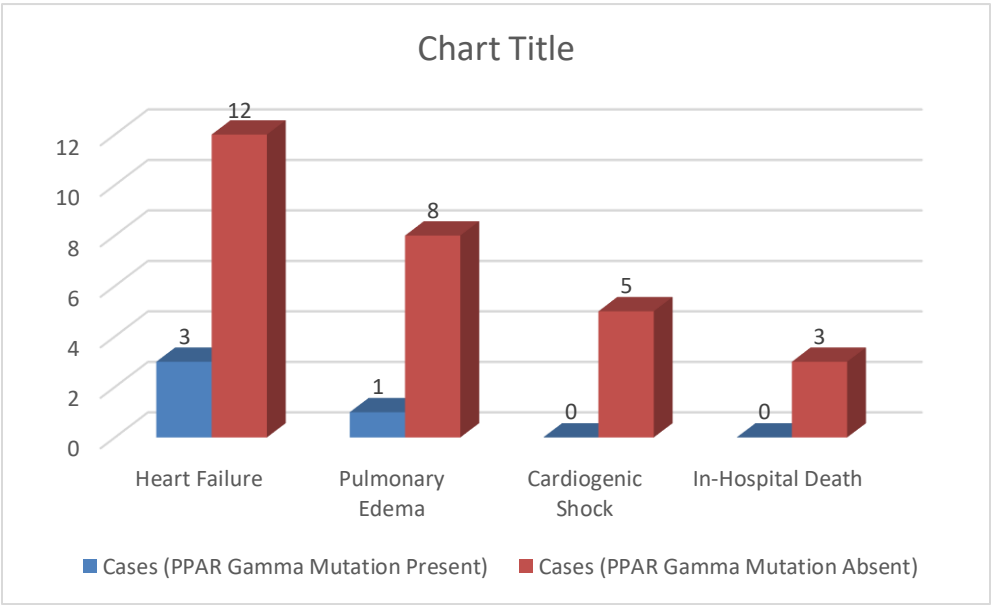
DISTRIBUTION OF PATIENTS ACCORDING TO MAJOR ADVERSE CARDIAC EVENTS:

This table lists out all the Major Adverse Cardiac Events (MACE) in 92 patients of both groups during their period of their in-hospital stay. Most common adverse event noted among both groups is heart failure that’s 15 patients followed by pulmonary enema 9 patients, 5 patients with Cardiogenic shock and 3 patients in hospital death

Table No 14: Major Adverse Cardiac Events

Condition	Group A (n=9)	Group B(n=83)	Total Cases	Percentage (%)	Chi-square	p-value
Heart Failure	3	12	15	16.30	1.93	0.587
Pulmonary Edema	1	8	9	9.78		
Cardiogenic Shock	0	5	5	5.43		
In-Hospital Death	0	3	3	3.26		

Graph 8: Major Adverse Cardiac Events



DISCUSSION

DISCUSSION

This is a prospective cross-sectional study where aim of the study was to look for PPAR gamma 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 PPAR gamma gene polymorphism.

AGE

In this study the age group of 50-70 years is the most commonly affected with coronary artery disease.

In India the Kerala ACS Registry 2007 studied a total 25748 patients with acute coronary syndrome between 2007 to 2009 suggested that most common group age was around 60 years old, which concurs with our observation of 39.1% of cases in the 60–69 age bracket [62].

Similarly, the INTERHEART study done by Yusuf et al, 2004 a total of 29,972 participants, with 15,152 cases and 14,820 controls concluded that South Asian patients have CAD at a younger age (mean age around 53 years) compared to Western populations. Whereas mean age group around 60 years in both group but 35% cases are below the age of 60 years [63].

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

SEX

In this study there were more male predominance as 59.8% of patients were males and female patients were 40.2%

The study done by van Oosterhout et al. 2019 reviewed systematically and analyse 27 studies consisting 1,413,881 patients found that 60% male and 40% female are affected which align with the present study [64].

Another study by Neha J et al between 2007 to 2008, 1565 patients was analysed and found that the 79% were male and 21 % were female which is higher than our study [65].

OCCUPATION

In our study the most common occupation associated with Group A was service employee 5 (55.6%) followed by homemaker which are female patients 3 (33.3%) and farmer 1 (11.1%).

Most of the patients **in the present study** belong to low and middle socioeconomic status. The reason could be lack of education about disease, risk factors, inability to afford for treatment, lack of compliance to medication, inability to modify risk factors and lack of regular follow up.

RISK FACTORS:

In **this** study of 92 patients, it is seen that that the cardiovascular traditional risk factors are present irrespective of genetic mutation. In this study, the majority of patients in both groups were older than 50 years (88.90% in Group A present vs. 84.80% Group B).

Benjamin et al. study in 2019 studied and observe that advanced age is the major risk factor CAD, due to age related vascular changes such as endothelial dysfunction and increased arterial stiffness [66].

Smoking and tobacco chewing was seen in 31 patients, of which 2 patients (33.3%) from Group A and 29 patients (36%) in Group B. Alcohol consumption was present in 8(10.6%) patients of only Group B and none in Group A. Dominique Himbert et al in March 2002 had analyzed data from 19325 patients and found that 27.3% patients were current smokers and, 29.5% were former smoker with significant p value of <0.001 [67].

Another study by Vikas Kadiyala et. al in India between November 2017 and October 2020, in 220 patients of which 102 were smokers and 118 were

nonsmokers found that smoking was associated with acute coronary syndrome by endothelial dysfunction and acting as prothrombotic state [68].

Yang Yang et al studied in March 2015, 214 340 participants and 7756 with acute coronary syndrome cases concluded that a nonlinear association was observed between CAD and alcohol consumption [69].

In this study diabetes status with the presence or absence of the PPAR gamma mutation of diabetes among subjects. Specifically, among those in one diabetes category, approximately 77.8% had the mutation absent, compared to 22.2% among those with mutation present. The overall numbers are relatively small (with a total of 9 cases in Group A and 83 Group B), so additional studies with larger sample sizes would be needed to confirm whether the mutation is significantly associated with an increased prevalence of diabetes.

Jae-Seung Yun et al. had analyzed 57 studies with 4 million individuals with diabetes and found that 32.2 % coronary artery disease patients with Type 2 diabetes mellitus [70].

The results for hypertension, show very similar proportions between the two mutation groups. Specifically, 33.0% of individuals in Group A and 33.7% of Group B were reported to have hypertension. These shows non-significant p-value ($p = 0.1786$), suggests that the presence of the mutation does not markedly influence the prevalence of hypertension in this study.

Vasiliki Christou et al. in 2014 at Nicosia General Hospital Cardiology Clinics studied 375 individuals with CAD and found that 59% of patient had history of hypertension [71].

Therefore, there is need for policies to control tobacco use, promote healthy diet and educate patients regarding adverse effects of tobacco use, which help in improving life expectancy of patients with ACS.

SYMPTOMS

Patients in this study from both the groups presented with various symptoms, amongst these, chest pain was the most common symptom seen in both the group. In Group A 77.80% and 88 % patients in Group B, second most common symptom was dyspnea seen in 66 % patients in Group A and 33.70 % patients in Group B this was followed by atypical symptoms (10.80%), abdominal pain (4.80%), palpitation (3.60%). While abdominal pain was reported in (22.20%) of Group A.

A study done by J G Conto et al studied under the title "Prevalence, Clinical Characteristics, and Mortality Among Patients With Myocardial Infarction Presenting Without Chest Pain" analyzed data from 434,877 patients with confirmed myocardial infarction (MI) enrolled in the National Registry of Myocardial Infarction 2 (NRMI-2) between 1994 and 1998, they found that chest pain was present in 67% (n=291367) of patients, which align with our study and 33% patient without chest pain [72].

A study conducted by David Brieger et al between July 1999 to June 2002, observational study in 14 countries and included 20881 patients with acute coronary syndromes. They noted that (8.4%) patients presented with atypical symptoms of ACS which also aligns with our study, where (10.8%) presented with atypical symptoms [73].

PEROXISOME PROLIFERATOR-ACTIVATED GAMMA GENE POLYMORPHISM

In this study of 92 patients with acute coronary syndrome, 9 patients, 3 males and 2 females showed positive mutation for this PPAR Gamma gene. They showed specific in PPAR gamma gene polymorphism (rs3856806, c.1341 C>T, p.H447H) in 9 out of 92 patients (9.8%). This synonymous variant was found in heterozygous condition and it is classified as benign or likely benign. The synonymous nature of the identified variant (p.H447H) means that while there is a nucleotide change (C>T), it does not alter the amino acid sequence of the protein. This is consistent with its classification as benign/likely benign. However, it's worth noting that even

synonymous variants can sometimes affect mRNA stability, protein folding kinetics, or splicing, potentially contributing to disease susceptibility.

In 2022, Aykut Kemanci et al. studied the Correlation between Peroxisome Proliferator-Activated Receptor Alpha and Gamma Polymorphisms and Acute Coronary Syndrome in 200 people, including 100 cases and 100 control and concluded that elevated PPAR alpha L162V and PPAR gamma C161T gene polymorphisms increases the risk of ACS [61].

In 2016, Yufeng Quin et al. studied the association between Peroxisome Proliferator-Activated Receptor alpha, delta and gamma Polymorphisms and association coronary heart disease or acute coronary syndrome in 446 subjects in Han. The findings revealed that PPAR-alpha intron 7G/C and L162V, PPAR-delta=+294T/C, and PPAR-gamma C161T polymorphisms, which directly associated with CHD in their cohort [56].

In this study we focus on different PPAR gamma SNP (rs3856806), our study also suggest genetic variations in PPAR gamma may contribute to the risk factor of ACS through its effects on lipid metabolism and inflammation.

In 2016 Wei et al. reported that individuals carrying the PPAR γ C161T CT/TT genotypes were observed to have lower blood lipid levels and a reduced risk of ischemic stroke due to large-artery atherosclerosis in a Han population [58].

Several meta-analyses and population-based studies have evaluated the broader role of PPAR gamma gene polymorphisms and found mixed results. Some studies suggest that even synonymous variations like rs3856806 might be linked with subtle regulatory effects influencing long-term metabolic outcomes.

In other studies, have explored how these polymorphisms interact with environmental risk factors (such as diet, body mass index, and smoking) to modify cardiovascular risk. These studies collectively imply that while individual polymorphisms (particularly synonymous ones) may not exert strong effects

independently, their cumulative or interaction effects might be clinically relevant over the long term.

Further research with larger sample sizes and functional studies would be valuable to better understand the clinical significance of this polymorphism

MAJOR ADVERSE CARDIAC EVENTS

92 patients of both groups with ACS were observed for Major adverse cardiac events (MACE) from the day of admission till discharge, out of this the most common Major adverse cardiac events observed was 16% n= 15 Heart failure and their ejection fraction was noted to be less than 40%, pulmonary edema which was seen in 12.8 % patients, also there were 3 in hospital deaths.

None of these MACE showed statistically significant P values; hence their occurrence could not be correlated with the PPAR Gamma gene polymorphism or any other clinical or biochemical parameters of the patients.

CONCLUSION

CONCLUSION

Acute coronary syndrome, is an acute form of coronary artery disease (CAD), it stands as the leading cause of deaths globally, the disease pathogenesis is versatile and complicated posing a challenge in diagnosis as well as timely decision-making regarding effective intervention; hence there is a need for usage of multi-step diagnostic tools which includes biological markers and genetic markers, is essential. Inflammation is the main culprit which plays a critical role in pathogenesis of acute coronary syndrome causing plaque formation and destabilization of plaques resulting in plaque rupture.

In this study we have demonstrated a relationship between PPAR gamma gene polymorphism and acute coronary syndrome and one of the Observation was presence of the disease in young age groups and also in few with no conventional risk factors, hence there is need for more vigilant screening for the disease and use of genetic profiling in all patients along with other routine biochemical and radiological tests.

Screening family members of patients with PPAR gamma gene positive mutation might help in early recognition of risk factors or even might be able to pick up the disease in the initial stages which will help in timely appropriate intervention, creating awareness about the disease will lead to overall reduction in disease burden by reducing the morbidity and mortality.

SUMMARY

SUMMARY

This study was conducted on 92 Patients of Acute coronary syndrome who were admitted and treated in BLDE (DU) Shri B M Patil Medical College Hospital and Research Centre, Vijayapura during the period of May 2023 to December 2024.

- The aim of this study was to detect Polymorphism of PPAR Gamma gene in Patients with Acute coronary syndrome.
- Out of 100 patients after applying the inclusion and exclusion criteria 92 patients were included in the study, based on the presence or absence of PPAR gamma gene mutation these patients were divided into Group A (9 patients) and Group B (83 patients) respectively.
- The most common age group of patients were between 50 to 70 years.
- In both groups Male patients were more than females, Group A 66.7 % (6 out of 9), Group B absent 59.8 % (49 out of 83).
- The most common occupation in Group A were service employee (55.6%), followed by housewives (33.30%) and farmer (11.10%) and in Group B the most common occupation was housemaker (42.20%), followed by Farmers (36.10%), service (14.10%) and shopkeeper (12.00%).
- The most common presenting symptom was Chest pain among both groups (77.8%) and (88%) respectively, next common was dyspnea 66.70% in Group A, 33.70 in Group B, least common was abdominal pain 4.80 % in Group B.
- ECG features of all patients in both groups were studied and most common ECG features in Group A was Inferior wall ST Elevation followed by NSTEMI, in Group B NSTEMI was most common 29.5%.
- ECHO findings were Inferior wall hypokinesia 66%, followed by anterior wall, anteroseptal wall cases.

- In our study of 92 patients with acute coronary syndrome, 9 patients, 3 males and 2 females showed positive mutation for this PPAR Gamma gene. They showed specific in PPAR gamma gene polymorphism (rs3856806, c.1341 C>T, p.H447H) in 9 out of 92 patients (9.8%).
- In addition Major Adverse Cardiac events were noted: Heart failure, Pulmonary edema, cardiogenic shock, and in hospital deaths.

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ANNEXURES

ANNEXURE I
INSTITUTIONAL ETHICAL CLEARANCE 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/ 876/2023-24

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 PEROXISOME PROLIFERATOR-ACTIVATED RECEPTOR GAMMA GENE POLYMORPHISM IN ACUTE CORONARY SYNDROME".

NAME OF THE STUDENT/PRINCIPAL INVESTIGATOR: DR. JAHANGIR ALAM

NAME OF THE GUIDE: DR.BADIGER SHARANABASAWAPPA, PROFESSOR & 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

Smt. Bangaramma Sajjan Campus, B. M. Patil Road (Sholapur Road), Vijayapura - 586103, Karnataka, India.

BLDE (DU): Phone: +918352-262770, Fax: +918352-263303, Website: www.bldedu.ac.in, E-mail: office@bldedu.ac.in
College: Phone: +918352-262770, Fax: +918352-263019, E-mail: bmpmc.principal@bldedu.ac.in

The combined total of all matches, including overlapping sources, for each database.

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- 31 Not Cited or Quoted 5%
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For Small

[Handwritten signature] 25/3/21



ANNEXURE – II

CONSENT FORM

**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 JAHANGIR ALAM 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. JAHANGIR ALAM informed me that he/she is conducting dissertation/research titled **“GENETIC STUDY OF PEROXISOME PROLIFERATOR-ACTIVATED RECEPTOR GAMMA GENE POLYMORPHISM IN ACUTE CORONARY SYNDROME”** IN BLDE (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:
- Temperature:
- Hair:
- Eyes:

- Pupils:
- Nose:
- Ears:
- Oral Cavity:
- Upper Limbs:
- Chest:
- Abdomen:
- Genitalia:
- Lower Limbs:
- Skin:

SYSTEMIC EXAMINATION

CARDIOVASCULAR SYSTEM

Arterial system:

- Pulse
 - Rate
 - Rhythm □ Volume
 - Character
 - Condition of the vessel wall
 - Radio radial 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	

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
CPK MB	ng/ml

URINE EXAMINATION -

Albumin	
Sugar	
Microscopy	

TROPONIN I :

PEROXISOME PROLIFERATOR- ACTIVATED RECEPTOR GAMMA GENE POLYMORPHISM

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:

ANNEXURE IV: MASTER CHART

	S.L.NL	1	2	3	4	5	6	7	8	9	10	11	12
		KASAPPA VEERUPAKSHA PRA MASHU	LAKMI MUTTAGI	AMENABEE SHEKOLAGAR	NAGAPPA KALLAPPA SHASHIPUR	KALAMMA BADGER	VITTAL BHARANANNA SHEKUTURUR	SHARANANMA BIDGAR	KASTURIBAI KALABIRAGI	AREJUN SURYAVANSHI	gambir karavagi	JALABAYI LAMANI	SHARANGODA BAGODE
		60	65	72	54	83	62	48	72	42	80	71	52
		M	F	F	M	F	M	F	F	M	F	F	M
		SEX	SEX	SEX	SEX	SEX	SEX	SEX	SEX	SEX	SEX	SEX	SEX
		OCCUPATION	HOME MAKER	HOME MAKER	SERVICE	HOME MAKER	SERVICE	HOME MAKER	HOME MAKER	SHOPKEEPER	HOME MAKER	HOME MAKER	FARMER
		PHONE NO	988565360	934660111	963078669	980781735	968562977	974235586	725948361	897512785	741602953	990795639	974235586
		ADDRESS	UKKALL VIJAPUR	NIGASUNDR AP PAKKA ROAD KAMPURAMATH SUDHAKHANE VILAPUR	AP PAKKA ROAD KAMPURAMATH SUDHAKHANE VILAPUR	KALUKA NAGAR, BIJAPUR	AP PAKKA ROAD TALBOTTL, BIJAPUR	AP PAKKA ROAD TALBOTTL, BIJAPUR	VIJAPUR	BEHIND RTO OFFICE, BAGGA NAGAR NEAR NAGADEGATTI TEMPLE, BIJAPUR	AP PAKKA ROAD, TALBOTTL, BIJAPUR	AP PAKKA ROAD, TALBOTTL, BIJAPUR	AP PAKKA ROAD, TALBOTTL, BIJAPUR
		IP.NL	88914	74227	24863	252278	269961	276598	274236	286652	286445	282765	285568
		D.O.A	11/01/2024	05/09/2024	12/06/2024	14/07/2024	29/07/2024	02/08/2024	01/08/2024	05/08/2024	05/08/2024	07/08/2024	07/08/2024
		D.O.D	14/01/2024	15/01/2024	04/07/2024	03/08/2024	02/08/2024	07/08/2024	06/08/2024	09/08/2024	06/08/2024	08/08/2024	22/08/2024
		DURATION OF STAY (DAY)	10	10	22	6	4	5	5	4	1	1	5
		CHEST PAIN	P	P	P	P	P	P	P	P	P	P	P
		DYSPEA	A	P	A	A	A	A	A	A	A	A	A
		PAUPTATION	A	A	A	A	A	A	A	A	A	A	A
		SYNCOPE	A	A	A	A	A	A	A	A	A	A	A
		ABDOMINAL PAIN	A	A	A	A	A	A	P	A	A	A	A
		ATYPICAL MANIFESTATION	A	A	A	A	A	A	A	A	A	A	A
		DIABETES	A	A	A	A	A	P	A	A	A	A	A
		HYPERTENSION	P	A	P	A	P	P	P	A	P	P	P
		FAMILY HISTORY	A	A	P	A	P	A	P	A	A	A	A
		SMOKING	P	A	A	A	A	A	A	A	A	A	A
		ALCOHOL	A	A	A	A	A	A	A	A	A	A	A
		TOBACCO CHewing	A	A	A	P	A	P	A	P	A	A	A
		PR	116	84	120	160	66	100	76	78	120	81	76
		SBP	116	130	100	160	140	130	150	130	150	130	120
		DBP	90	80	60	100	90	90	90	80	90	80	70
		SBP/DBP	116/90	130/80	100/60	160/100	140/90	130/90	150/90	130/80	150/90	130/80	120/70
		TEMPERATURE	37	37	37	38	37	37	37	37	37	37	38
		HR	20	18	18	18	18	11	18	18	28	18	18
		TRFPI	481.3	490.6	34.8	700.5	256.05	11	7294	44.94	170.8	1104	2144
		HEMOGLOBIN G/DL	7	7	13.1	24.1	22.5	12.4	8	10.6	0.91	6.2	11.9
		TOTAL COUNT	22	7.01	13.1	24.1	22.5	12.4	8	10.6	0.91	6.2	11.9
		ESR	50	15mm/hr	17mm/hr	14mm/hr	23mm/hr	8mm/hr	20mm/hr	20mm/hr	28mm/hr	14mm/hr	10mm/hr
		FBP/PPBS	rbis-12.5	rbis-138mg/dl	rbis-186mg/dl	rbis-209mg/dl	rbis-122mg/dl	rbis-96mg/dl	rbis-95mg/dl	rbis-209mg/dl	rbis-91mg/dl	rbis-98mg/dl	rbis-256mg/dl
		BLOOD UREA	50	38	32	42	26	28	32	29	34	19	26
		Sr Creatinine	0.7	0.8	1.4	1.2	0.8	1	1.3	0.9	1.6	0.7	0.8
		SERUM SODIUM	144	143	145	134	128	136	140	138	143	143	138
		Sr.POTASSIUM	3.8	4.4	3.6	5.3	3.5	4.4	4.4	3.9	5.3	5.3	3.4
		TOTAL CHOLESTROL	162	224	246	172	180	160	242	186	175	288	207
		TRIGLYCERIDES	350	125	132	196	155	175	145	155	146	100	147
		HDL(mg/dl)	16	20	36	30	35	33	34	28	35	36	26
		LDL	120	155	183	103	122	125	160	132	112	198	140
		ECG RATE	100BP M	92BP M	150BP M	150BP M	75hpm	60BP M	75hpm	75hpm	100BP M	100BP M	75hpm
		RHYTHM	Tachycardia	Regular	Tachycardia	Tachycardia	Regular	Regular	Regular	Regular	Tachycardia	Tachycardia	Regular
		P WAVE	0.08	0.08	0.12	0.08	0.08	0.08	0.08	0.12	0.04	0.08	0.08
		PRINTERVAL	0.12	0.08	0.20	0.08	0.12	0.12	0.12	0.16	0.20	0.12	0.20
		QRS CONFIGURATION	POOR R WAVE	Q WAVES V1-V6, NOTCHED Q WAVE V4-V6R PATTERN	DEEP Q WAVE V2-V5, J WAVE	DEEP S WAVES IN V1-V2 TALL R WAVES V5-V6, W CHANGES	QRS COMPLEX V1-V6	RS COMPLEX V1-V5	RS COMPLEX V1-V5	DEEP Q WAVES V1-V5	QRS PATTERN 2,3,AVF, POOR R WAVE PROGRESSION	Q WAVES 3,AVF 2,AVF V5-V6	Q WAVES 3,AVF 2,AVF V5-V6
		QRS DURATION	0.04s	0.08s	0.04s	0.08s	0.04s	0.04s	0.04s	0.04s	0.04s	0.04s	0.04s
		QRS AXIS	N	N	LAD	N	N	N	N	N	LAD	N	LAD
		ST SEGMENT	ST DEPRESSION V2-V4, J WAVE	RBBB	ST ELEVATION IN 2,3,AVF	ST DEPRESSION 3,AVF V2-V4	ST ELEVATION V2-V6, 2,3,AVF	ST ELEVATION V2-V4, J WAVE	ST DEPRESSION V2-V4	ST ELEVATION V2-V4, J WAVE	ST ELEVATION V2-V4	ST ELEVATION V2-V4	ST DEPRESSION V2-V4, J WAVE
		T wave	INVERTED V3-V6	INVERTED V4-V6	INVERTED 2,3,AVF	NOT WAVE CHANGES	INVERTED V2-V6	INVERTED V2-V6	NOT WAVE CHANGES	INVERTED V2-V6	INVERTED V2-V6	INVERTED 2,3,AVF	INVERTED 2,3,AVF
		QT	480s	520s	520s	280s	440s	480s	480s	440s	280s	440s	480s
		QTc	520s	570s	450s	480s	480s	480s	500s	480s	360s	580s	530s
		ECG DIAGNOSIS	STEMI ANTERIOSEPTAL AND INFERIOR WALL	RBBB	STEMI INFERIOR WALL	STEMI ANTERIOR WALL	STEMI ANTERIOR WALL	STEMI ANTERIOR WALL	STEMI INFERIOR WALL	STEMI INFERIOR WALL	STEMI ANTERIOSEPTAL WALL	STEMI INFERIOR WALL	STEMI INFERIOR WALL
		product leave in air	A	A	A	A	A	A	A	A	A	A	A
		Fragmented	A	A	A	A	A	A	A	A	A	A	A
		2D ECHO REGIONAL MOTION AND SEPTAL ABNORMALITY	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	NO MOTION	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	GLOBAL HYPOKINESIA	INFERIOR WALL HYPOKINESIA	INFERIOR WALL HYPOKINESIA
		LVEF	4000%	30%	35%	35%	50%	60%	45%	50%	25%	55%	45%
		LVE	A	A	A	A	A	A	A	A	A	A	A
		MYOGENE MUTATION	NOT DONE	NOT DONE	NOT DONE	NOT DONE	NOT DONE	NOT DONE	NOT DONE	NOT DONE	NOT DONE	NOT DONE	NOT DONE
		HEART FAILURE	A	A	A	A	A	A	A	A	A	A	A
		PULMONARY EDEMA	A	A	A	A	A	A	A	A	A	A	A
		ARRHYTHMIA	A	A	A	A	A	A	A	A	A	A	A
		CARDIOGENIC SHOCK	A	A	A	A	A	A	A	A	A	A	A
		IN HOSPITAL DEATH	A	A	A	A	A	A	A	A	A	A	A

[illegible]

Sr. No.	Patient Name	Age	Sex	Occupation	Phone No.	Address	IP No.	D.O.A	D.O.D	Duration of Stay (Day)	Chest Pain	Dyspnea	Palpitation	Syncope	Abscolum PAIN	Atypical Manifestation	Diabetes	Hypertension	Family History	Smoking	Alcohol	Tobacco Chewing	PR	Systolic Bp(mg)	Diastolic BP (mmHg)	Temperature	RHR	TCOP1	Hemoglobin G/DL	Total Count	ESR	FBN/PPMs/RBS	BLOOD UREA	S-Creatine	Serum Sodium	S-potassium	Total Cholesterol	Triglycerides	HDL(mg/dl)	LDL	EGR-RATE	Rhythm	P-WAVE	PR INTERVAL	QRS CONFIGURATION	QRS DURATION	QRS AXIS	ST SEGMENT	
24	REKHADEVI NING	69	M	SERVICE	705729150	G-0 GURUGRAMA NG SIRSIYANU VIZAR COMPOUND JALNAGAR, BANGALUR	290730	11/08/2024	20/08/2024	7		A	A	A	A	A	A	P	A	A	A	A	80	120	70	37	22	58.2	12	5.76	10mm/hr	rbs- 130mg/ dl	19	3.1	138	4.2		238	268	31	150	75bpm	Regular	0.08 s	0.16 s	Q WAVEs V1-V6-AVL	0.125	N	ST ELEVATION V2,VL DEPRESSION V2,V6F
25	SABEER HUSEINASHA MASALI	75	F	HOME MAKER	821966426	AP WATER PARK, BURJOR	293524	14/08/2024	16/08/2024	2		P	A	A	A	A	A	P	A	A	A	A	112	140	90	37	16	18.5	10	8.94	15mm/hr	rbs- 120mg/ dl	25	0.8	142	4.8		250	178	30	110	150BP M	Tachycardia	0.04 s	0.12 s	Q WAVEs V1-V4	0.04s	N	ST ECV V5 INVERTED V2V6
26	BAWNAARD KUMBAR	64	M	FARMER	866071887	AT POST HALADGANA TALUKA, DARGA, BURJOR	253478	14/08/2024	21/08/2024	7		A	A	A	A	A	A	A	P	A	A	A	56	140	70	37	18	556.7	12	12.1	10mm/hr	rbs- 120mg/ dl	28	0.7	140	4.1		170	261	39	198	150BP M	Tachycardia	0.08 s	0.04 s	DEEP Q WAVEs V1-V6	0.085	N	ST ELEVATION V2 V5 INVERTED V1-V6
27	SHUBHA BEGUN MUNIRCHANDRET HAN	49	F	HOME MAKER	908225641	STATION ROAD, BURJOR	294030	15/08/2024	22/08/2024	6		P	A	A	A	A	A	A	P	A	A	A	68	126	80	37	24	26.3	14	10.2	15mm/hr	rbs- 100mg/ dl	33	0.7	145	4.2		255	178	27	166	75bp m	Regular	0.12 s	0.20 s	Re-completes V1-V4	0.125	N	ST DEPRESSION I , J,T LAJLE
28	MAMANKAI PANWAR	70	F	HOME MAKER	705727559	AT POST OTTAWAL, BURJOR	294345	16/08/2024	20/08/2024	4		P	A	A	A	A	A	A	P	A	A	A	114	140	80	38	22	496.4	10	5.31	20mm/hr	rbs- 100mg/ dl	57	1.9	149	4.6		219	366	33	136	100BP M	Tachycardia	0.04 s	0.16 s	MATCHED QRS 1,LAJVS, Osy pattern c4-c5	0.08s	LAD	ST E CVI V3 INVERTED c4-c9
29	RAVI MAHE	41	M	SERVICE	908368122	AP HACHAMALLI, BURJOR	294855	16/08/2024	19/08/2024	3		P	A	A	A	A	A	A	P	A	A	A	60	100	60	37	22	969.2	12	13.5	5mm/hr	rbs- 120mg/ dl	24	1	141	4.4		244	286	20	177	75BP M	Regular	0.12 s	0.12 s	Q WAVE IN V1	0.125	N	ST ECV V4,VAF
30	VALEED INDOODIA	62	M	SERVICE	861040786	JHA ROAD NEAR BARAHMAN, BURJOR	294669	16/08/2024	22/08/2024	6		P	A	A	A	A	A	P	A	A	A	A	82	150	90	37	22	2.3	8	9.8	20mm/hr	rbs- 342mg/ dl	50	1.2	138	4.5		190	196	36	119	100BP M	Tachycardia	0.12 s	0.16 s	DEEP Q WAVEs V1-V9	0.125	LAD	ST ECV V4,L3,VAF INVERTED V2-VH
31	HAMEEDABI MAMKANDAR	67	F	HOME MAKER	997208884	MANAGULI, BANAVANGENWAL, BURJOR	293846	16/08/2024	20/08/2024	8		P	A	A	A	A	A	A	P	A	A	A	90	110	70	37	22	251.3	14	12.9	10mm/hr	rbs- 100mg/ dl	41	0.7	146	3.6		236	188	27	178	82BP M	Regular	0.12 s	0.16 s	Q WAVEs V1-V4	0.125	N	ST ELEVATION V1-VH
32	CHARANWA SOLAPUR	70	F	HOME MAKER	886386240	HALGAN TALUKA, BURJOR	293770	16/08/2024	23/08/2024	7		P	A	A	A	A	P	P	A	A	A	A	82	180	110	37	22	23	13	5.21	10mm/hr	rbs- 100mg/ dl	30	1	139	4.8		200	172	33	159	100BP M	Regular	0.12 s	0.16 s	Re-completes V2-VH	0.04s	N	ST DEPRESSION 3,VAFV2 INVERTED V4-V6
33	MAHADEVI KHAMKANDAI	70	F	HOME MAKER	990114294	848,LESHWAR TALUKA, BURJOR	293949	16/08/2024	21/08/2024	5		P	A	A	A	A	A	P	P	A	A	A	80	94	70	37	18	50	9	6.23	15mm/hr	rbs- 100mg/ dl	30	0.7	145	3.4		120	218	35	130	100BP M	Regular	0.08 s	0.16 s	Re-completes V4-V6	0.04s	N	ST ECV V4 INVERTED V4-V6
34	SIRANT TOTAD	52	M	SERVICE		MALONI, CHUDHALLI, PHANAGANWAL, BANGALUR	293735	16/08/2024	20/08/2024	4		P	A	A	A	A	P	P	A	A	P	P	86	110	70	37	22	122	14	5.2	10mm/hr	rbs- 140mg/ dl	28	1.4	136	4.3		194	301	39	126	72bpm	Regular	0.12 s	0.12 s	Re-completes V4-V6	0.125	N	ST DEPRESSION 2,VAFV4,V6 INVERTED V4-V6,VAFV6
35	IBRAPPA S GALGAU	50	M	FARMER	938627835	G-0 SANGAPPA CHUDHALLI, MANGALUR	29974	27/08/2024	21/08/2024	4		P	A	A	A	A	A	P	P	A	A	A	96	150	100	38	20	2560.5	13	15.2	10mm/hr	rbs- 100mg/ dl	25	0.6	140	3.6		172	96	30	110	100BP M	Tachycardia	0.12 s	0.12 s	POOR R WAVE PROGRESSION	0.125	LAD	ST ELEVATION 2,VAF INVERTED 2,VAFV4,V6
36	REKHADEVI NING	69	M	SERVICE	705729150	G-0 GURUGRAMA NG SIRSIYANU VIZAR COMPOUND JALNAGAR, BANGALUR	290730	11/08/2024	20/08/2024	7		A	A	A	A	A	A	P	A	A	A	A	80	120	70	37	22	58.2	12	5.76	10mm/hr	rbs- 130mg/ dl	19	3.1	138	4.2		238	268	31	150	75bpm	Regular	0.08 s	0.16 s	Q WAVEs V1-V6-AVL	0.125	N	ST ELEVATION V2,VL DEPRESSION V2,V6F
37	SABEER HUSEINASHA MASALI	75	F	HOME MAKER	821966426	AP WATER PARK, BURJOR	293524	14/08/2024	16/08/2024	2		P	A	A	A	A	A	P	A	A	A	A	112	140	90	37	16	18.5	10	8.94	15mm/hr	rbs- 120mg/ dl	25	0.8	142	4.8		250	178	30	110	150BP M	Tachycardia	0.04 s	0.12 s	Q WAVEs V1-V4	0.04s	N	ST ECV V5 INVERTED V2V6
38	BAWNAARD KUMBAR	64	M	FARMER	866071887	AT POST HALADGANA TALUKA, DARGA, BURJOR	253478	14/08/2024	21/08/2024	7		A	A	A	A	A	A	A	P	A	A	A	56	140	70	37	18	556.7	12	12.1	10mm/hr	rbs- 120mg/ dl	28	0.7	140	4.1		170	261	39	198	150BP M	Tachycardia	0.08 s	0.04 s	DEEP Q WAVEs V1-V6	0.085	N	ST ELEVATION V2 V5 INVERTED V1-V6
39	SHUBHA BEGUN MUNIRCHANDRET HAN	49	F	HOME MAKER	908225641	STATION ROAD, BURJOR	294030	15/08/2024	22/08/2024	6		P	A	A	A	A	A	A	P	A	A	A	68	126	80	37	24	26.3	14	10.2	15mm/hr	rbs- 100mg/ dl	33	0.7	145	4.2		255	178	27	166	75bp m	Regular	0.12 s	0.20 s	Re-completes V1-V4	0.125	N	ST DEPRESSION I , J,T LAJLE
40	MAMANKAI PANWAR	70	F	HOME MAKER	705727559	AT POST OTTAWAL, BURJOR	294345	16/08/2024	20/08/2024	4		P	A	A	A	A	A	A	P	A	A	A	114	140	80	38	22	496.4	10	5.31	20mm/hr	rbs- 100mg/ dl	57	1.9	149	4.6		219	366	33	136	100BP M	Tachycardia	0.04 s	0.16 s	MATCHED QRS 1,LAJVS, Osy pattern c4-c5	0.08s	LAD	ST E CVI V3 INVERTED c4-c9
41	RAVI MAHE	41	M	SERVICE	908368122	AP HACHAMALLI, BURJOR	294855	16/08/2024	19/08/2024	3		P	A	A	A	A	A	A	P	A	A	A	60	100	60	37	22	969.2	12	13.5	5mm/hr	rbs- 120mg/ dl	24	1	141	4.4		244	286	20	177	75BP M	Regular	0.12 s	0.12 s	Q WAVE IN V1	0.125	N	ST ECV V4,VAF
42	VALEED INDOODIA	62	M	SERVICE	861040786	JHA ROAD NEAR BARAHMAN, BURJOR	294669	16/08/2024	22/08/2024	6		P	A	A	A	A	A	P	A	A	A	A	82	150	90	37	22	2.3	8	9.8	20mm/hr	rbs- 342mg/ dl	50	1.2	138	4.5		190	196	36	119	100BP M	Tachycardia	0.12 s	0.16 s	DEEP Q WAVEs V1-V9	0.125	LAD	ST ECV V4,L3,VAF INVERTED V2-VH
43	HAMEEDABI MAMKANDAR	67	F	HOME MAKER	997208884	MANAGULI, BANAVANGENWAL, BURJOR	293846	16/08/2024	20/08/2024	8		P	A	A	A	A	A	A	P	A	A	A	90	110	70	37	22	251.3	14	12.9	10mm/hr	rbs- 100mg/ dl	41	0.7	146	3.6		236	188	27	178	82BP M	Regular	0.12 s	0.16 s	Q WAVEs V1-V4	0.125	N	ST ELEVATION V1-VH
44	CHARANWA SOLAPUR	70	F	HOME MAKER	886386240	HALGAN TALUKA, BURJOR	293770	16/08/2024	23/08/2024	7		P	A	A	A	A	P	P	A	A	A	A	82	180	110	37	22	23	13	5.21	10mm/hr	rbs- 100mg/ dl	30	1	139	4.8		200	172	33	159	100BP M	Regular	0.12 s	0.16 s	Re-completes V2-VH	0.04s	N	ST DEPRESSION 3,VAFV2 INVERTED V4-V6
45	MAHADEVI KHAMKANDAI	70	F	HOME MAKER	990114294	848,LESHWAR TALUKA, BURJOR	293949	16/08/2024	21/08/2024	5		P	A	A	A	A	A	P	P	A	A	A	80	94	70	37	18	50	9	6.23	15mm/hr	rbs- 100mg/ dl	30	0.7	145	3.4		120	218	35	130	100BP M	Regular	0.08 s	0.16 s	Re-completes V4-V6	0.04s	N	ST ECV V4 INVERTED V4-V6
46	SIRANT TOTAD	52	M	SERVICE		MALONI, CHUDHALLI, PHANAGANWAL, BANGALUR	293735	16/08/2024	20/08/2024	4		P	A	A	A	A	P	P	A	A	P	P	86	110	70	37	22	122	14	5.2	10mm/hr	rbs- 140mg/ dl	28	1.4	136	4.3		194	301	39	126	72bpm	Regular	0.12 s	0.12 s	Re-completes V4-V6	0.125	N	ST DEPRESSION 2,VAFV4,V6 INVERTED V4-V6,VAFV6
47	IBRAPPA S GALGAU	50	M	FARMER	938627835	G-0 SANGAPPA CHUDHALLI, MANGALUR	29974	27/08/2024	21/08/2024	4		P	A	A	A	A	A	P	P	A	A	A	96	150	100	38	20	2560.5	13	15.2	10mm/hr	rbs- 100mg/ dl	25	0.6	140	3.6		172	96	30	110	100BP M	Tachycardia	0.12 s	0.12 s	POOR R WAVE PROGRESSION	0.125	LAD	ST ELEVATION 2,VAF INVERTED 2,VAFV4,V6
48	REKHADEVI NING	69	M	SERVICE	705729150	G-0 GURUGRAMA NG SIRSIYANU VIZAR COMPOUND JALNAGAR, BANGALUR	290730	11/08/2024	20/08/2024	7		A	A	A	A	A	A	P	A	A	A	A	80	120	70	37	22	58.2	12	5.76	10mm/hr	rbs- 130mg/ dl	19	3.1	138	4.2		238	268	31	150	75bpm	Regular	0.08 s	0.16 s	Q WAVEs V1-V6-AVL	0.125	N	ST ELEVATION V2,VL DEPRESSION V2,V6F
49	SABEER HUSEINASHA MASALI	75	F	HOME MAKER	821966426	AP WATER PARK, BURJOR	293524	14/08/2024	16/08/2024	2		P	A	A	A	A	A	P	A	A	A	A	112	140	90	37	16	18.5	10	8.94	15mm/hr	rbs- 120mg/ dl	25	0.8	142	4.8		250	178	30	110	150BP M	Tachycardia	0.04 s	0.12 s	Q WAVEs V1-V4	0.04s	N	ST ECV V5 INVERTED V2V6
50	BAWNAARD KUMBAR	64	M	FARMER	866071887	AT POST HALADGANA TALUKA, DARGA, BURJOR	253478	14/08/2024	21/08/2024	7		A	A	A	A	A	A	A	P	A	A	A	56	140	70	37	18	556.7	12	12.1	10mm/hr	rbs- 120mg/ dl	28	0.7	140	4.1		170	261	39	198	150BP M	Tachycardia	0.08 s	0.04 s	DEEP Q WAVEs V1-V6	0.085	N	ST ELEVATION V2 V5 INVERTED V1-V6
51	SHUBHA BEGUN MUNIRCHANDRET HAN	49	F	HOME MAKER	908225641	STATION ROAD, BURJOR	294030	15/08/2024	22/08/2024	6		P	A	A	A	A	A	A	P	A	A	A	68	126	80	37	24	26.3	14	10.2	15mm/hr	rbs- 100mg/ dl	33	0.7	145	4.2		255	178	27	166	75bp m	Regular	0.12 s	0.20 s	Re-completes V1-V4	0.125	N	ST DEPRESSION I , J,T LAJLE
52	MAMANKAI PANWAR	70	F	HOME MAKER	705727559	AT POST OTTAWAL, BURJOR	294345	16/08/2024	20/08/2024	4		P	A	A	A	A	A	A	P	A	A	A</																											

SL. N.	NAME	AGE	SEX	OCCUPATION	PHONE NO	ADDRESS	IP N.	D.O.A	D.O.D	DURATION OF STAY (DAY)	CHEST PAIN	DYSPIEA	PALPITATION	SYNCOPE	ABDOMINAL PAIN	ATYPICAL MANIFESTATION	DIABETES	HYPERTENSION	FAMILY HISTORY	SMOKING	ALCOHOL	TOBACCO CHEWING	PR	SYSTOLIC B(p/mmHg)	DIASTOLIC UP (mmHg)	TEMPERATURE	RR	TROP1	HEMOGLOBIN G/DL	TOTAL COUNT	ESR	FBG/PPG/UBS	BLOOD UREA	Sc. Creatinine	SERUM SODIUM	Sc.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	LIVER	I/E	MEBI. GENE MUTATION CAG	HEART FAILURE	PULMONARY EDEMA	ARRHYTHMIA	CARDIOGENIC SHOCK	
36	SUBHASH RAJANITHI	65	M	FARMER	986895794	JUMMAL, BIJAPUR	291939	14/06/2024	28/08/2024	14	P	A	A	A	A	A	P	P	A	A	A	A	90	70 95 TO LIC	37	22	11.7	13	24.2	15mm /hr	rbS-108mg/dl		43	1.1	135	3.7	198	172	36	141	100BP M	Tachycardia	0.12 S	0.20 S	Q WAVES V1-V4	0.08S	N	ST E V4S WITH ST DEP 2,3,AVF	INVERTED V2,AVF	440s	560s				25.1	A		NOT DONE	P	P	A	P	
37	Bondag Saab Mokalshi	69	M	FARMER	9902931942	GUBBEWADI	298444	19/09/2024	25/09/2024	6	P	A	A	A	A	A	P	P	A	A	A	A	80	110	70	37	18	1540	12	10mm /hr	rbS-149mg/dl		20	0.8	132	3.2	235	190	30	162	75bp m	Regular	0.08 S	0.16 S	N	0.04s	LAD	ST DEP V2,V3	NOT WAVE CHANGES	480s	530s				50.1	A		DVD	A	A	A	A	
38	AMBABAWA SAGAL	65	F	HOME MAKER	9972545865	JALAKITHI	300611	20/09/2024	24/09/2024	9	P	P	A	A	A	A	P	P	P	A	A	A	78	130	80	37	18	556.7	12	30mm /hr	rbS-209mg/dl		23	1.2	139	4.2	200	170	26	116	100BP M	Tachycardia	0.08 S	0.12 S	Q WAVES 2,3,AVF	0.04s	LAD	ST E V2-V3,AVF,2,3	INVERTED 2,3	480	620				50.1	A		DVD	A	A	A	A	
39	HANAMANTHI KAMBLE	72	M	SERVICE	903619366	JAMKANDI	1502	22/09/2024	02/09/2024	11	P	P	A	A	A	A	P	P	A	A	A	A	90	14	90	37	18	100	11	29	RB5-236		61	1.2	138	4.2	180	150	35	115	75bp m	Regular	0.08 S	0.16	Q WAVE IN V1,V6	0.04s	LAD	ST E V2-V5	INVERTED V1,V6	520s	580s				30.1	A		DVD	P	A	A	A	
40	LAKSHMI HANWAL	63	M	FARMER	789542456	AP KANAKODAS BAGAVANNE, BIJAPUR	21112	21/06/2024	28/08/2024	7	P	P	A	A	A	A	P	P	A	A	A	A	60	170	100	37	20	36	16	10mm /hr	rbS-268mg/dl		26	1.3	136	3.8	298	120	31	206	60BP M	Regular	0.12 S	0.16 S	S WAVE IN V1,VR IN V5,SV5,1,VR	0.12S	LAD	ST DEP LA,VL,V5,V6	INVERTED V1-V6	400s	410s				45.1	A		SVD	A	A	A	A	
41	VITHAL DEVSHATE	70	M	FARMER		KYATAMBEH, INDI, BIJAPUR	2352	21/06/2024	28/08/2024	7	P	P	A	A	A	A	P	P	A	A	A	A	58	130	70	38	18	1023	13	10mm /hr	rbS-161mg/dl		34	1	142	3.5	181	292	30	115	60BP M	Regular	0.08 S	0.16 S	Q WAVES IN 2,3,AVF	0.04S	LAD	ST E 2,3,AVF	INVERTED 2,3	400s	400s				40.1	A		NOT DONE	A	A	A	A	
42	DJ PATEL	83	M	SERVICE		BAGEWADI, BIJAPUR	212343	21/09/2024	28/09/2024	7	P	A	A	A	A	A	A	P	P	A	A	A	40	130	80	37	20	17	10	10mm /hr	rbS-222 mg/dl		20	0.8	140	3.2	150	192	30	102	43BP M	Bradycardia	0.12 S	0.24 S	rs in 2,3,AVF	0.12s	LAD	STE V1-V3	INVERTED V1	480s	400s				60.1	A		NOT DONE	A	A	A	A	
43	SHRISHAIL BILADAR	65	M	SHOPKEEPER		BAGEWADI, BIJAPUR	14233	21/06/2024	28/08/2024	7	P	P	A	A	A	A	A	P	P	A	A	A	97	100	70	37	20	88	13	5mm/hr	rbS-123mg/dl		17	0.7	131	4.3	186	244	35	122	100BP M	Tachycardia	0.12 S	0.16 S	Q WAVE V1,3,AVR	0.12S	LAD	ST ELEVATION 3,AVF,2	INVERTED V2,V3	360s	480s				25.1	A		NOT DONE	A	A	A	A	
44	DURGAPPA GHANTI	60	M	SERVICE	888488837	DARGA ROAD	0166	25/06/2024	01/09/2024	6	P	A	A	A	A	A	A	P	P	A	A	A	86	160	90	37	20	150	15	10mm /hr	RB5-137mg/dl		17	0.7	146	5	150	120	22	106	75bp m	Regular	0.08 S	0.20 S	Q WAVES 2,3,AVF	0.04s	LAD	ST E 2,3,AVF	INVERTED	400s	470s				40.1	A		SVD	P	A	A	A	
45	MUNNIAJIATH DATODD	50	F	HOME MAKER	991225089	VUJAPUR	00020	26/09/2024	30/09/2024	4	P	A	A	A	A	A	A	P	P	A	A	A	52	80	60	37	18	2000	12	10mm /hr	rbS-120mg/dl		18	0.6	142	4.2	270	460	24	196	50BP M	Bradycardia	0.08 S	0.12 S	N	0.04s	N	ST E 2,3,AVF	HYPERACUTE T WAVE 2,3,AVF	520s	470s				40.1	A		NOT DONE	A	A	A	A	
46	BASANTIA TORANI	58	M	FARMER	9553672145	DOMANHAL	00055	26/09/2024	01/09/2024	5	P	A	A	P	A	A	P	P	P	A	A	P	90	110	70	37	18	1154	13	10mm /hr	RB5-133mg/dl		35	1.4	134	3.8	175	136	26	101	100BP M	Tachycardia	0.08 S	0.12 S	Q WAVES 2,3,AV5-V6	0.04s	N	ST E 2,3,AV5-V6	INVERTED 2,3,AV5	400s	510s				35.1	A		NOT DONE	TVD	A	A	A	
47	TUKARAM KOLHAR	54	M	SHOPKEEPER	991225089	HANCHINAL	00023	27/09/2024	03/09/2024	7	P	A	A	A	A	A	A	P	P	A	A	A	70	140	80	37	20	415.1	12	15mm /hr	RB5-180mg/dl		27	0.9	136	4.2	250	135	32	188	75bp m	Regular	0.08 S	0.16 S	N	0.08S	RAD	ST E 1,2,AVF,V2,V6	NOT WAVE CHANGES	400s	470s				35.1	A		NOT DONE	P	A	A	A	
48	MAHADEV KOLHAR	69	F	HOME MAKER	998072500	MUDHOL	00025	28/09/2024	03/09/2024	6	P	A	A	A	A	A	P	P	A	A	A	A	78	150	90	37	18	179	12	15mm /hr	rbS-150mg/dl		30	0.7	140	3.3	180	250	35	115	75bp m	Regular	0.08 S	0.12 S	N	0.04s	LAD	ST DEP V2,V3,VA,V5,V6	INVERTED V2-V6	440s	490s				45.1	A		NOT DONE	A	A	A	A	
49	BANDAWA PATILNAN SHETTY	76	F	HOME MAKER	984474728	HONNITAGI	407405	28/09/2024	03/09/2024	6	P	A	A	A	A	A	A	P	P	A	A	A	82	170	80	37	18	312	9	15mm /hr	RB5-139mg/dl		18	0.5	140	3.2	200	175	30	136	100BP M	Tachycardia	0.08 S	0.16 S	Q WAVES 2,3,AVF	0.04s	N	ST E 2,3,AVF	INVERTED V2-V3	480	620s				50.1	A		NOT DONE					

SL. N.	NAME	AGE	SEX	OCCUPATION	PHONE NO	ADDRESS	IP N.	D.O.A	D.O.D	DURATION OF STAY (DAY)	CHEST PAIN	DYSNOEA	PAUPTATION	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	FBG/FBS/MBIS	BLOOD UREA	St. Creatine	SERUM SODIUM	Sr.POTASSIUM	TOTAL CHOLESTROL	TRIGLYCERIDES	HDL(mg/dl)	LDL	ECG-RATE	RHYTHM	PWAVE	PR INTERVAL	QRS CONFIGURATION	QRS DURATION	QRS AXIS	ST-SEGMENT	T wave	QT	QTc	ECG DIAGNOSIS	postinet wave in aVF	Fragmented qrs	2D ECHO REGIONAL MOTION WALL	ANTEROLATERAL WALL	LVEF	LVE	NERVE GENE MUTATION	CAG	HEART FAILURE	PULMONARY OEDEMA	ABRTHMAS	CARDIOGENIC SHOCK	IN HOSPITAL DEATH
50	KAMALA BAI SONAD	63	F	HOME MAKER	990018867	VIJAPUR	00779	26/04/2024	03/05/2024	6	P	A	A	A	A	A	P	P	A	A	A	A	98	120	80	37	18	24918	10	17.4	10mm /hr	rbS-122mg/dl	28	0.8	135	5.1	360	155	20	298	150BPM	Tachycardia	0.04s	0.12s	N	0.04s	N	STD V2-V6	NOT WAVE CHANGES	360s	560s	NTSTEMI	A	A	HYPOKINESIA OF ANTERIOR AND INTERLATERAL WALL	5500	A	NOT DONE	A	A	A	A	A		
51	BANUBI MULLA	66	F	HOME MAKER	938039857	BABALAD	00069	29/04/2024	06/05/2024	8	P	A	A	A	A	A	P	P	A	A	A	A	82	130	80	37	18	122.1	10	7.3	10mm /hr	RBS-25.0mg/dl	20	0.8	135	3.4	180	165	35	110	100BPM	Tachycardia	0.08s	0.12s	DEEP Q WAVES V1-V3	0.04s	N	STE V1-V3	INVERTED V1-V4	440	560s	STEMI ANTERIOR	A	A	HYPOKINESIA OF ANTERIOR AND INTERLATERAL WALL	45	A	NOT DONE	A	A	A	A	A		
52	UMAANT SONAD	73	M	HOME MAKER	821705424	DEVNAR HPPABADGI	00042	30/04/2024	08/05/2024	9	P	A	A	A	A	A	P	P	A	A	A	A	72	120	80	37	18	0.1	13	8.41	10	FB-189mg/dl	FB-323mg/dl	30	1.2	139	4	200	175	30	112	100BPM	Tachycardia	0.04s	0.12s	deep q wave V1-V4	0.04s	N	STE E V2-V5	INVERTED V1-V5	400	510s	STEMI ANTERIOSEPTAL	A	A	HYPOKINESIA OF ANTERIOR AND INTERLATERAL WALL	35	A	NOT DONE	A	A	A	A	A	
53	JASTURIBAI NAVI	55	F	HOME MAKER	829503189	SALOTAGI	2857	11/05/2024	10/05/2024	6	P	A	A	A	A	A	P	P	A	A	A	A	86	90	60	37	18	125.0	12	7.01	20mm /hr	RBS-296mg/dl	27	1.1	130	4.3	150	146	18	98	75bpm	Regular	0.08s	0.20s	deep q wave v1-V4	0.04s	LAD	STE E V2-V5	INVERTED 2,3,AV2-V6	400s	480s	STEMI ANTERIOR	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	30	A	NOT DONE	A	A	A	A	A		
54	SAMBAL MANDAD	73	F	HOME MAKER	829670315	VIJAPUR	3027	13/05/2024	23/05/2024	10	P	A	A	A	A	A	P	P	A	A	A	A	66	110	70	37	18	101.1	10	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	0.04s	N	STE V1-V3	ASYMETRIC WAVE INVERSION V1-V3,2,3,AVF	360s	480s	STEMI ANTERIOSEPTAL	A	A	HYPOKINESIA OF INTERIOR AND LATERAL WALL	40	E	NOT DONE	A	A	A	A	A		
55	ZANDIMAKSEN BURIMWALE	44	F	HOME MAKER	634725907	ALLAPUR	4212	20/05/2024	27/05/2024	7	A	A	A	A	A	P	P	P	A	A	A	A	116	130	90	37	22	97.2	12	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	0.08s	N	ST CHANGES V1-V4	INVERTED 2,3,AVF	400	510s	STEMI ANTERIOSEPTAL	A	A	HYPOKINESIA OF SEPTUM	40	A	NOT DONE	A	A	A	A	A		
56	ALIANA KADADGI	60	F	HOME MAKER	636350317	MOTIGESBAI	4463	22/05/2024	29/05/2024	7	P	A	A	A	A	A	P	P	A	A	A	A	82	100	70	37	18	122.1	14	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	0.04s	N	STEMI V1-V6	INVERTED V2-V6,2,3,AVF	480	530	STEMI ANTERIOSEPTAL	A	A	HYPOKINESIA OF ANTEROLATERAL WALL	45	A	NOT DONE	A	A	A	A	A		
57	GAYATRI KULKARNI	60	F	HOME MAKER	948029555	NEAR MAYAR MATH	4472	22/05/2024	26/05/2024	6	P	A	A	A	A	P	P	P	A	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	0.04s	N	STE DEEP V4-V6,2,3,AVF	INVERTED V4-V6,2,3,AVF	400s	510s	NTSTEMI	A	A	INFERIOR WALL	60	A	NOT DONE	A	A	A	A	A		
58	BHIMU KATTIMANI	35	M	FARMER	770847757	KARAJAGI	4071	24/05/2024	28/05/2024	4	P	A	A	A	A	A	P	P	A	A	A	A	96	110	70	37	18	60	13	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	0.04s	N	STE V2-V6	INVERTED V2-V6,2,3,AVF	400s	440s	STEMI ANTERIOSEPTAL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	30	A	NOT DONE	A	A	A	A	A		
59	REVANSKIDAPPA JODUR	64	M	FARMER	8618846714	MAHARAJE TEMPLE ROAD	3438	25/05/2024	01/06/2024	7	P	A	A	A	A	A	P	P	A	A	A	A	70	100	60	37	18	28	15	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	0.04s	LAD	STE E V1-V4	INVERTED V2-V6	480s	480s	STEMI ANTEROLATERAL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	35	A	NOT DONE	A	A	A	A	A		
60	SHIVAPPA KALIMANI	40	M	FARMER	789948474	VIJAPUR	59944	26/05/2024	28/05/2024	2	P	A	A	A	A	P	P	P	A	A	A	A	86	130	90	37	18	10783	16	11.6	10mm /hr	rbS-150mg/dl	12	0.6	135	3.9	175	155	35	86	75bpm	Regular	0.08s	0.20s	N	0.04s	N	STE E V1-V6	T WAVE CHANGES NOTED	520s	560s	NTSTEMI	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	40	A	NOT DONE	A	A	A	A	A		
61	MALIKARJUN TEGGHALI	46	M	SHOPKEEPER	980606046	ATHARGA	58504	26/05/2024	30/05/2024	4	P	A	A	A	A	A	P	P	A	A	A	A	92	140	80	37	18	200	15	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	0.04s	N	STE E V1-V6	INVERTED V1-V6	520	670s	STEMI ANTEROLATERAL WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	40	A	NOT DONE	A	A	A	A	A		

S.N.	NAME	AGE	SEX	OCCUPATION	PHONE NO	ADDRESS	IP N.	D.O.A	D.O.D	DURATION OF STAY (DAY)	CHEST PAIN	DYSPIEA	PALPITATION	SYNCOPE	ABDOMINAL PAIN	ATYPICAL MANIFESTATION	DIABETES	HYPERTENSION	FAMILY HISTORY	SMOKING	ALCOHOL	TOBACCO CHewing	PR	SYSTEMIC BP(mmHg)	DIASTOLIC BP (MMHG)	TEMPERATURE	RR	TROPIN	HEMOGLOBIN G/DL	TOTAL COUNT	ESR	FBG/FBS/PBS	BLOOD UREA	Sr. Creatine	SERUM SODIUM	PO-PTASSIUM	TOTAL CHOLESTROL	TRIGLYCERIDES	HDL(mg/dl)	LDL	EKG-SATE	RHYTHM	P-WAVE	P-R INTERVAL	QRS CONFIGURATION	QRS DURATION	QRS AXIS	ST SEGMENT	T-wave	QT	QTc	ECG DIAGNOSIS
62	LAMVAN HEGDON	49	M	FARMER	9908493274	JAMKHANDI	5476	27/09/2024	01/10/2024	5	A	A	A	A	A	A	P	A	A	A	A	A	56	140	80	38	20	2300	14	10.4	15mm /hr	RBS-231mg/dl	17	0.9	139	3.9	386	222	28	268	60BP M	BRADYCARDIA	0.08 s	0.12 s	DEEP Q WAVE LAVR	0.04s	N	STE V2-V6	INVERTED V1-V6-AVL	480	480s	STEMI ANTERIO LATERAL WALL
63	MURIEDHAR DEGNAL	69	M	FARMER	938089381	INDI	5500	27/09/2024	02/10/2024	5	A	A	A	A	A	A	P	A	A	A	A	A	70	140	80	37	18	368	12	6.25	10mm /hr	RBS-200mg/dl	29	0.8	139	4.4	150	133	24	100	75bp m	Regular	0.04 s	0.12 s	RBR PATTERN IN 3	0.04s	N	ST DEP 2,3AVF	INVERTED AVF-AVL	480	500s	STEMI ANTERIO LATERAL WALL
64	BHIMANGOLA PATIL	87	M	FARMER	638277551	SANNE SCHOOL	5524	28/09/2024	07/10/2024	9	A	A	A	A	A	A	P	A	A	A	A	A	100	170	100	38	24	5590	14	16.7	20mm /hr	RBS-169mg/dl	31	0.8	138	3.9	175	155	30	120	100BP M	Tachycardia	0.08 s	0.12 s	POOR R WAVE PROGRESSION DEEP Q WAVE V1-V3	0.04s	N	ST DEP V4-V6,	INVERTED V1V2	480s	620s	STEMI ANTERIO LATERAL WALL
65	SUMITA SHAHA	70	F	HOME MAKER	997250571	CHACHAH	5559	28/09/2024	05/10/2024	7	A	A	A	A	A	P	P	A	A	A	A	A	112	13	80	37	16	89.9	12	13.9	20mm /hr	RBS-149mg/dl	36	0.5	133	3.3	140	182	18	86	100BP M	Tachycardia	0.08 s	0.24 s	DEEP Q WAVE V1-V3	0.04s	N	STE V4-V6,2,3AVF	INVERTED V1-V6,2,3AVF	520s	670s	STEMI INFERO LATERAL WALL
66	ASHOK KUMBAR	81	M	SHOPKEEPER	997691407	GAKHINA KAST TI	5664	28/09/2024	02/10/2024	4	A	A	A	A	A	A	P	A	A	A	A	A	90	180	90	37	16	6.9	13	6.19	10mm /hr	RBS-231mg/dl	14	0.6	141	4.5	180	162	35	110	75bp m	Regular	0.08 s	0.20 s	Q WAVE 1	0.04s	N	ST DEP V4-V6,	INVERTED V1-V6	400s	400s	STEMI ANTERIOR WALL MI
67	ROHAMMA BHUNASYAL	60	F	HOME MAKER	634192248	BAKAVAN BAGEWADI	6442	01/10/2024	05/10/2024	4	A	P	A	A	A	A	P	A	A	A	A	A	94	140	90	38	24	1301	8	13.3	15mm /hr	RBS-165mg/dl	84	2	137	6.2	251	134	30	160	75bp m	Regular	0.08 s	0.20 s	VPCS NOTED V1,V6,2,3	0.04s	N	ST DEP V4-V6,	INVERTED V1-V6	400s	400s	STEMI ANTERIOR WALL MI
68	BASAPPA KANOLI	70	M	FARMER	8650972740	TALUK DEVIAN BIAPURE	6413	03/10/2024	07/10/2024	4	A	A	A	A	A	A	P	A	A	A	A	A	76	130	90	37	18	23068	13	8.41	10mm /hr	fbs-90mg/dl	23	0.6	135	4.5	262	371	33	215	75bp m	Regular	0.08 s	0.12 s	deep q wave v1-v6	0.04s	N	ST ELEVATION 3,AVF,2	INVERTED 2,3AVF-V6	520s	500s	STEMI INFEORE LATERAL WALL
69	SIDDIPA GOBI	60	M	FARMER	938014609	AT MANAGUHI TAL. BAGEMADOL, BIAPURE	6478	03/10/2024	07/10/2024	4	A	P	A	A	A	P	P	A	A	A	A	A	82	120	80	37	18	166.4	12	5.09	10mm /hr	fbs-166mg/dl	26	0.8	132	5.1	267	178	30	202	75BP M	Regular	0.08 s	0.12 s	Rx complexes in v4-V6	0.04s	N	ST DEP V4-V6, V4/V5-V6	INVERTED 2,3,AVF-V4-V6	480s	520s	STEMI ANTERIO LATERAL WALL
70	TALOUDIN DEGNAL PHUBAI	45	M	SHOPKEEPER	7023548074	SHAHARTIR AGASI INDI	6832	05/10/2024	11/10/2024	6	A	P	A	A	A	P	A	A	A	A	A	A	62	90	50	37	22	8583	13	19.8	48MM /HR	RBS-256mg/dl	134	2	139	3.3	152	222	32	101	75bp m	Regular	0.08 s	0.12 s	TALL T WAVES V3-V5	0.04s	N	ST ELEVATION 2,3AVF-V4-V6	HYPERACUTE T WAVE V1-V6	400s	400s	STEMI INFERO LATERAL WALL
71	MAHADEV RATHOD	65	F	HOME MAKER	636378255	KOLHABOL INDI	7893	12/10/2024	20/10/2024	8	A	P	A	A	A	A	P	A	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	100BP M	Tachycardia	0.04 s	0.12 s	N	STE V3-V6	INVERTED V3-V6	400s	510s	STEMI ANTERIOR WALL		
72	BHINSINGH RATHOD	55	M	FARMER		VIAPUR	9778	14/10/2024	20/10/2024	6	A	P	A	A	A	A	P	A	A	A	A	A	78	110	70	37	18	1800	15	11.8	10mm /hr	RBS-250mg/dl	32	1.5	129	4.6	198	236	35	130	75bp m	Regular	0.08 s	0.12 s	DEEP Q WAVES V1-V3	0.04s	N	STE V3-V3	INVERTED V1-V3	480s	400s	STEMI ANTERIOR WALL
73	ANIL KAMBLE	53	M	SHOPKEEPER	996271130	NEHRU NAGAR	8551	16/10/2024	20/10/2024	6	A	P	A	A	A	P	A	A	A	A	A	A	96	113	80	37	18	122	11	7.3	10mm /hr	RBS-341mg/dl	19	0.8	134	3.8	204	186	26	158	100BP M	Regular	0.08 s	0.12 s	Q WAVE AVR	0.04s	N	ST CHANGES V1-V5	SYMMETRICT WAVE INVERTED V1-V6	480s	520s	STEMI ANTERIOR WALL
74	RAMU NAMANE	45	M	SERVICE	940656406	BARAVANA BAKENDI	8562	16/10/2024	19/10/2024	3	A	A	A	A	A	A	P	A	A	A	A	A	58	150	70	37	18	1524	11	8.2	10mm /hr	fbs-158mg/dl	18	0.6	134	4.2	246	202	38	166	50BP M	Bradycardia	0.08 s	0.12 s	N	STE V3-V6	INVERTED DEEP V2-V6	480	430	STEMI ANTERIO LATERAL WALL		

SL.N.	NAME	AGE	SEX	OCCUPATION	PHONE NO	ADDRESS	IP N.	D.O.A	D.O.D	DURATION OF STAY (DAY)	CHEST PAIN	DYSPEA	PAUPTATION	SYNCOPE	ABDOMINAL PAIN	ATYPICAL MANIFESTATION	DIABETES	HYPERTENSION	FAMILY HISTORY	SMOKING	ALCOHOL	TOBACCO CHEWING	PR	SYSTOLIC BP(mmHg)	DIASTOLIC BP (mmHg)	TEMPERATURE	RR	TRCP1	HEMOGLOBIN G/DL	TOTAL COUNT	ESR	FBS(7PMS)/RBS	BLOOD UREA	Sr. Creatine	Sr.POTASSIUM	TOTAL CHOLESTROL	TRIGLYCERIDES	HDL(mg/dl)	LDL	EKG-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 AND SEPTAL WALL	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	LVEF	LVE	NERVE GENE MUTATION	CAG	HEART FAILURE	PULMONARY EDEMA	ARRHYTHMIA	CARDIOGENIC SHOCK	IN HOSPITAL DEATH																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																			
75	HAUMA MULLA	64	F	HOME MAKER		GANGABADI NEAR GANDHI SCHOOL	8470	16/10/2024	23/10/2024	7	P	A	A	A	A	A	P	A	A	A	A	A	118	150	80	37	18	2352	13	5.19	10mm /hr	RBS- 82mg/dl		20	0.8	136	4.4	196	282	34	118	150BPM	Tachycardia	0.08 s	0.12 s	DEEP Q WAVES V3-V4	0.04s	N	ST E V3-V5	INVERTED V3-V5	360	560	STEMI ANTERIOSEPTAL WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTAL WALL	30 I	A																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																																									

SL. N.	NAME	AGE	SEX	OCCUPATION	PHONE NO	ADDRESS	IP N.	D.O.A	D.O.D	DURATION OF STAY (DAY)	CHEST PAIN	DYSPIREA	PAUPTATION	SYNCOPE	ABDOMINAL PAIN	ATYPICAL MANIFESTATION	DIABETES	HYPERTENSION	FAMILY HISTORY	SMOKING	ALCOHOL	TOBACCO CHEWING	PR	SYSTOLIC BIPHRAGTIC DIASTOLIC BP (MMHG)	TEMPERATURE	HR	TROP T	HEMOGLOBIN G/DL	TOTAL COUNT	ESR	FBG/PPBS/MBUS	BLOOD UREA	S. Creatinine	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	postdict wave in ar	Fragmented qrs	2D ECHO REGIONAL ABNORMALITY	HEART FAILURE	PULMONARY EDEMA	ARRHYTHMIAS	CARDIOGENIC SHOCK	HOSPITAL DEATH
87	NINGAYYA NATARAJI	69	M	FARMER	96302431	SAGANUR, TALUKOTI	17407	09/12/2024	14/12/2024	5	P	A	A	A	A	A	P	A	A	P	P	A	118	180	90	37	20	2000	15	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	0.04s	N	ST E V1-V4, AVL	DEEP INVERTED ASYMMETRIC V1-V4	326ms	326ms	STEM ANTERO LATERAL WALL	A	A	HYPOKINESIA OF ANTERO LATERAL WALL	HEART FAILURE	PULMONARY EDEMA	ARRHYTHMIAS	CARDIOGENIC SHOCK	HOSPITAL DEATH
88	HANAMANTH TOPANNA GOVIL	64	M	FARMER		VIJAYUR	17529	10/12/2024	14/12/2024	4	P	A	A	A	A	A	P	A	A	P	P	68	130	80	37	18	150	14	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	0.04s	N	ST DEP V2-V5	NO T WAVE CHANGES	440ms	450ms	INFERMI ANTERO LATERAL WALL	A	P	HYPOKINESIA OF ANTERO LATERAL WALL	HEART FAILURE	PULMONARY EDEMA	ARRHYTHMIAS	CARDIOGENIC SHOCK	HOSPITAL DEATH	
89	GURUDASAPPA KADABAGANNI	54	M	FARMER	815007732	HEBIAL, NIDAMUNDI	17631	10/12/2024	15/12/2024	5	P	A	A	A	A	A	P	A	A	P	P	90	90	60	37	20	27.14	15	15mm/hr	RBS-121mg/dl	20	0.9	137	4.4	274	143	34	182	100BPM	Regular	0.08s	0.12s	INVERTED V1-V6	0.08s	RAD	ST E V1-V6	INVERTED V1-V6	320	410	RBBB	A	P	HYPOKINESIA OF ANTERO LATERAL WALL AND SEPTAL WALL	HEART FAILURE	PULMONARY EDEMA	ARRHYTHMIAS	CARDIOGENIC SHOCK	HOSPITAL DEATH	
90	JABINISSA FOJJI	62	F	HOME		GANGABAVA DI	17660	11/12/2024	11/12/2024	3	P	P	A	A	A	A	P	A	A	P	P	110	130	90	37	26	351	11	11	RBS-159mg/dl	23	1.2	128	3.5	178	155	30	116	75bpm	Regular	0.04s	0.12s	ST CHANGES I, II, aVL	0.125s	N	ST CHANGES I, II, aVL	NOT WAVE CHANGES	420	420ms	LUA	A	P	HYPOKINESIA OF ANTERO LATERAL WALL AND SEPTAL WALL	HEART FAILURE	PULMONARY EDEMA	ARRHYTHMIAS	CARDIOGENIC SHOCK	HOSPITAL DEATH	
91	RAMESH HIRIESAN	50	M	FARMER	779504467	A/P BANGAGAL, TIKOTTA	19398	10/12/2024	25/12/2024	6	P	P	A	A	A	A	P	A	A	P	P	70	110	70	37	18	68.1	16	16	rbs-116mg/dl	26	1	143	3.9	270	145	38	150	58BPM	Regular	0.04s	0.16s	Q WAVES I, II, aVL	0.125s	N	ST CHANGES I, II, aVL	NOT WAVE CHANGES	360ms	360ms	STEMI ANTERO LATERAL WALL	A	A	HYPOKINESIA OF ANTERO LATERAL WALL AND SEPTAL WALL	HEART FAILURE	PULMONARY EDEMA	ARRHYTHMIAS	CARDIOGENIC SHOCK	HOSPITAL DEATH	
92	ELVARAGU	61	M	FARMER	8796577898	TIKOTTA	94722	02/02/2024	30/02/2024	08/01/2000	P	P	A	A	A	A	P	A	A	P	P	84	140	90	37	18	12.8	88	26	258	47	1.3	127	5.1	248	183	38	122	70BPM	Regular	0.04s	0.12s	ST CHANGES I, II, aVL	0.205s	N	ST CHANGES I, II, aVL	NOT WAVE CHANGES	364ms	364ms	STEMI ANTERO LATERAL WALL	A	A	HYPOKINESIA OF ANTERO LATERAL WALL AND SEPTAL WALL	HEART FAILURE	PULMONARY EDEMA	ARRHYTHMIAS	CARDIOGENIC SHOCK	HOSPITAL DEATH	