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.

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IN

GENERAL MEDICINE

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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

PPARγ : 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 PPARγmRNAs. 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 GroupAi.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 studymales 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 walland 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, PPARγ

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 PPARγ gene, which is found on chromosome 3p25, uses alternative splicing and promoter use to produce four distinct PPARγ 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 PPARγ1, PPARγ3, and PPARγ4 [11–13], whereas PPARγ2 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 PPARγ 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 eightmillion 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 onemillion 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 dislodged 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 deathLocal 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. Clinicalexamination of coronary heart disease patient areusually as diaphoresis, cool

anddamp 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 findingpulmonary 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, 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 (Pro115GIn) 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%.

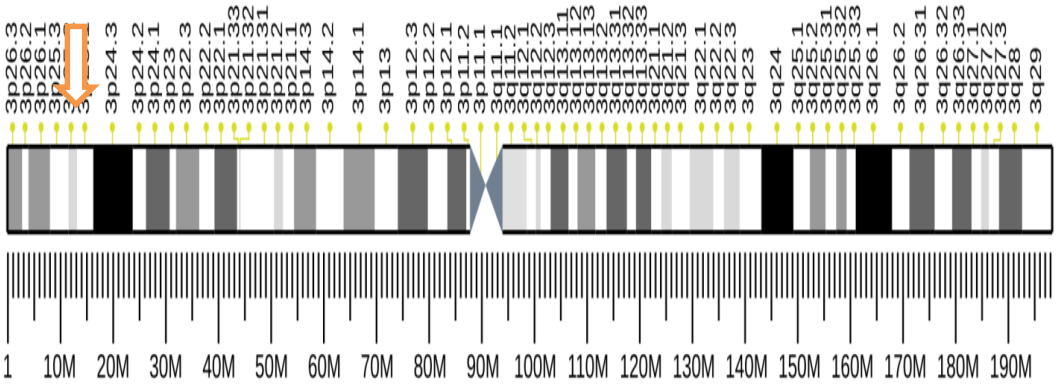


Fig 1: Cytogenetic Location of Peroxisome Proliferator-Activated Receptor Gamma

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-κB. AP-1, STAT, and NF-κB 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 useddHPLC (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. PPARγ genetic variations may influence the susceptibility to atherosclerotic disorders, as evidenced by the preventive function of the 93695C > T polymorphism in the PPARγ promoter in ACS[52].

Yilmaz-aydogan et al in 2011 examined the potential relationship between PPAR-γ2 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 >00.05). 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 2016aimed to determine if PPARΓ 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 PPARΓ 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 PPARΓ C161T genotype, total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C) were also investigated. PPARΓ 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 2020sought 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 2021assessed 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 2022assessed 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 18years 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, underwentstandardizedassessment with detailed history, clinical examination, investigations like electrocardiogram, cardiac enzyme –TroponinI and coronary angiogram (if required) on admission were taken along with 1ml of blood sample of the patient for analysis ofPPAR Gamma gene polymorphism.

DETECTION OF PPAR GAMMAPOLYMORPHISM

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.

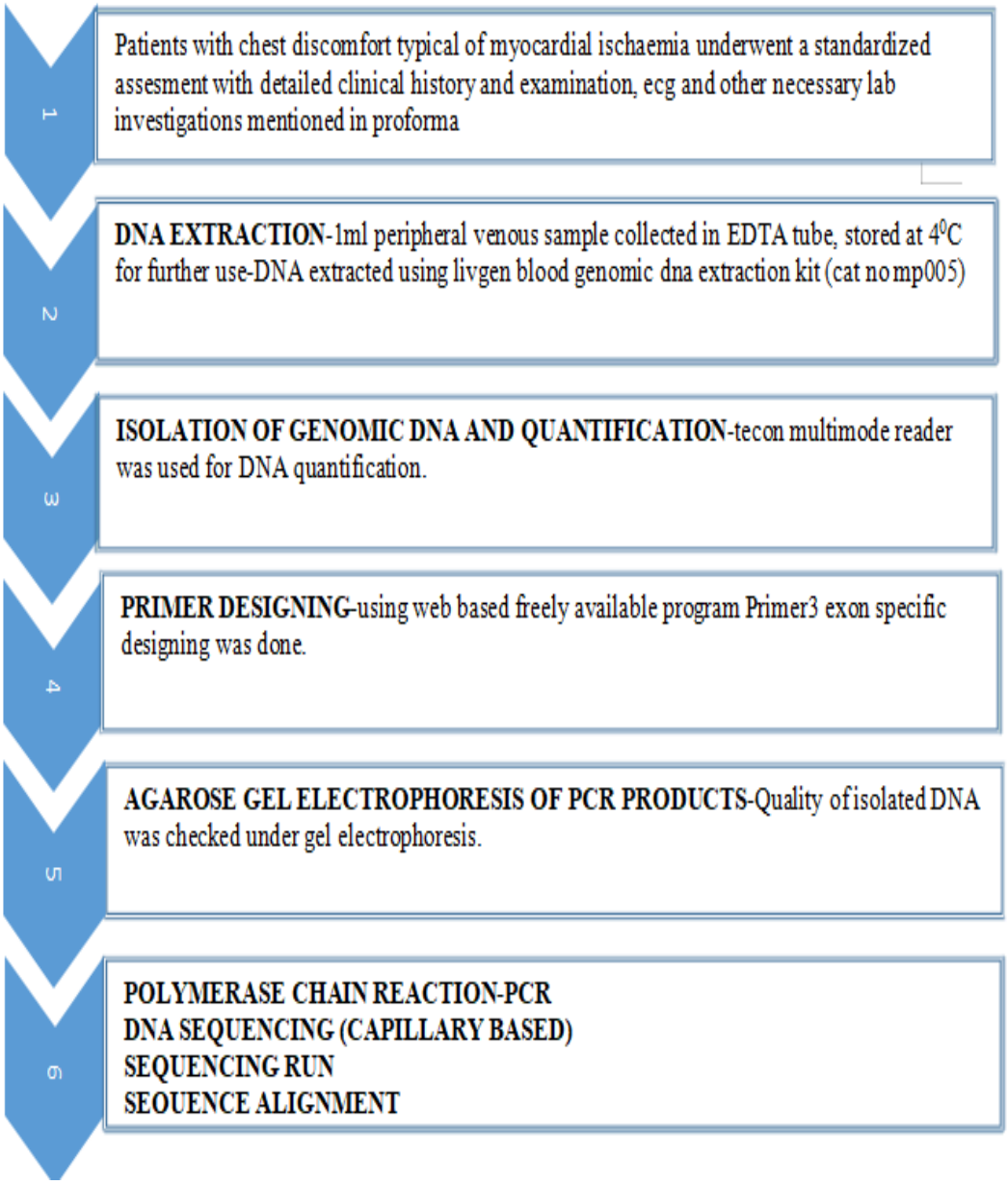


Figure 2. Depicts process of gene sequencing from collected blood samples

DNA EXTRACTION: 1 ml of peripheral venous blood samples were collected and stored in the EDTA coated vacutainers and stored at 4^0 C for further use.

Genomic DNA extraction from ACS samples was extracted using livgen blood genomic DNA extraction kit. (cat no mp005).

ISOLATION OF GENOMIC DNA AND DNA QUANTIFICATION: Genomic DNA was isolated from the extracted DNA samples and 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 CAA GC	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 forone 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 TM terminator v3.1was 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)

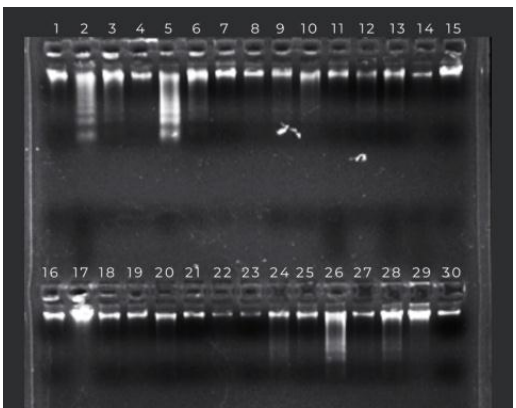


Figure 3: AGAROSE GEL IMAGE OF GENOMIC DNA OF ACUTE CORONARY SYNDROME SAMPLES.

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 v5.4software (Applied Biosystems, USA).

Sequence Alignment

The generated sequences were aligned to their respective reference sequences with the use of Variant reporter software (ABI v1.1). It performs sequence comparisons for novel mutations, known variants, insertions, and deletions. The results of the variant reporter were tabulated in PDF format as the default program of the software. Here, we used this technique to check the isolated genomic DNA from whole blood. In all the 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 patientshave valvular heart disease and three have cardiomyopathy.Ninety twopatients were studied for PPAR gamma gene polymorphism.

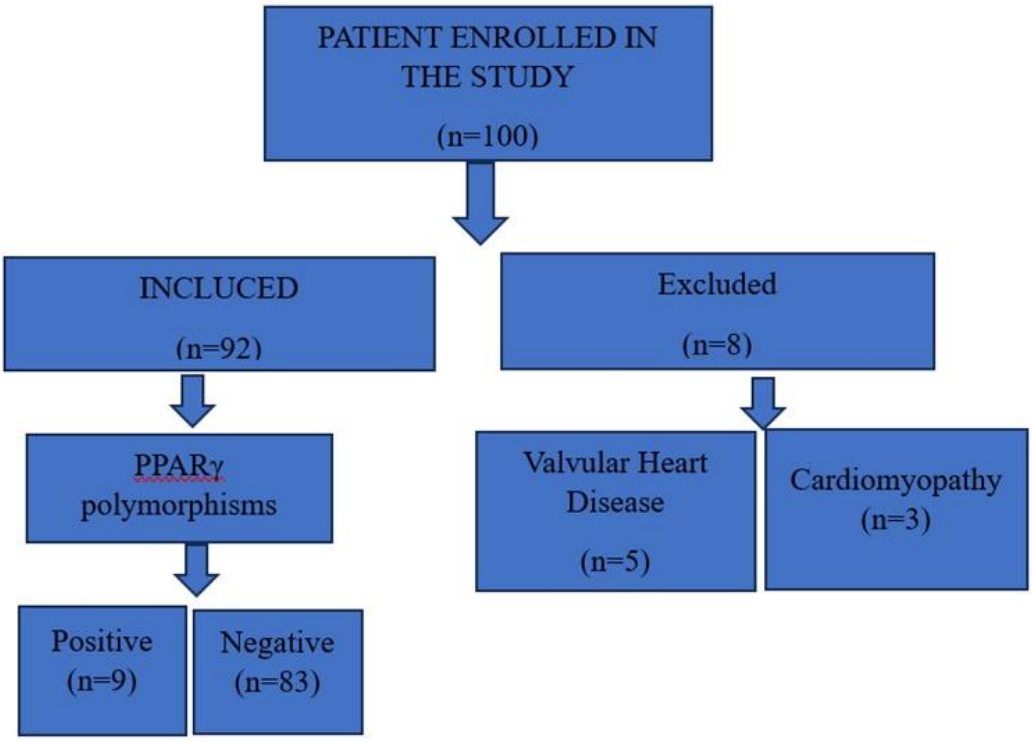


Figure 4: Flowchart showing included and excluded cases in this study

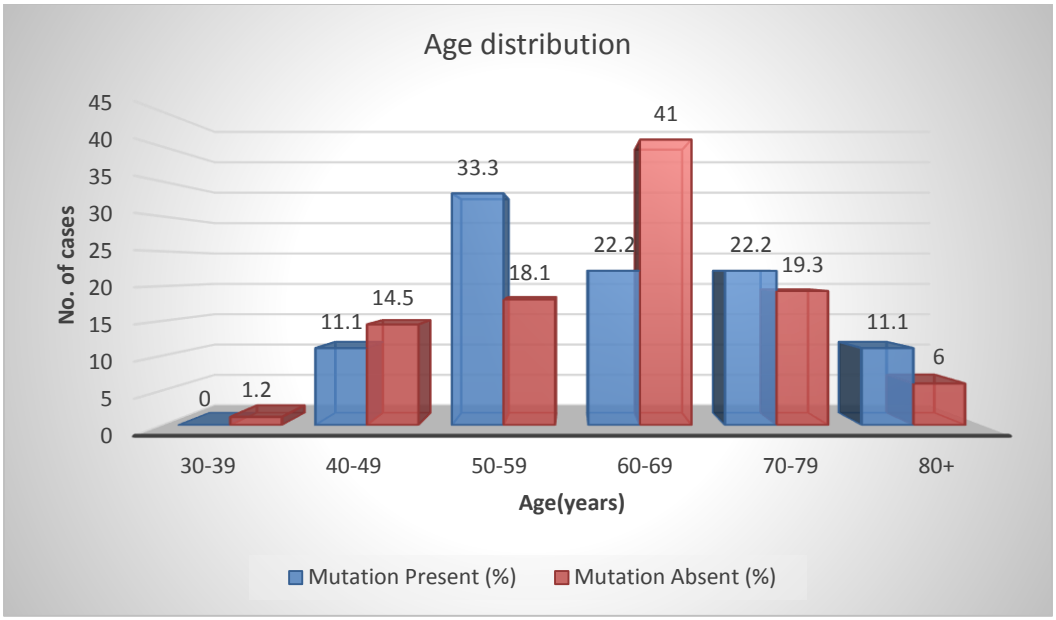
AGE DISTRIBUTION

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 groupPPAR GAMMA mutation absent patients aged between <40 years was 1(1.1%%), patientsbetween the age 41-50 yearswere 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



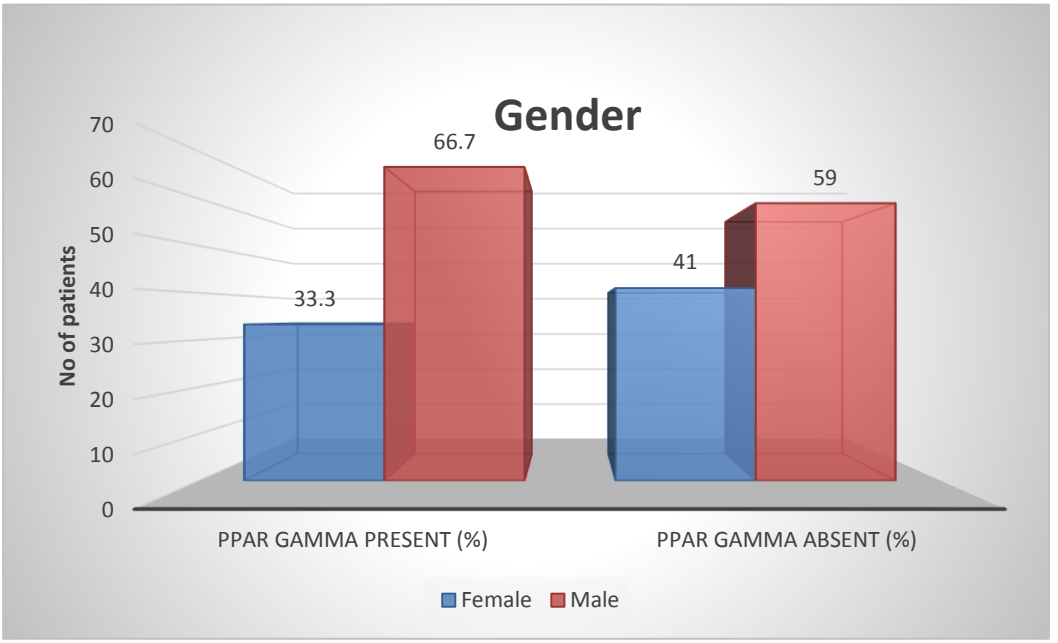
SEX DISTRIBUTION

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



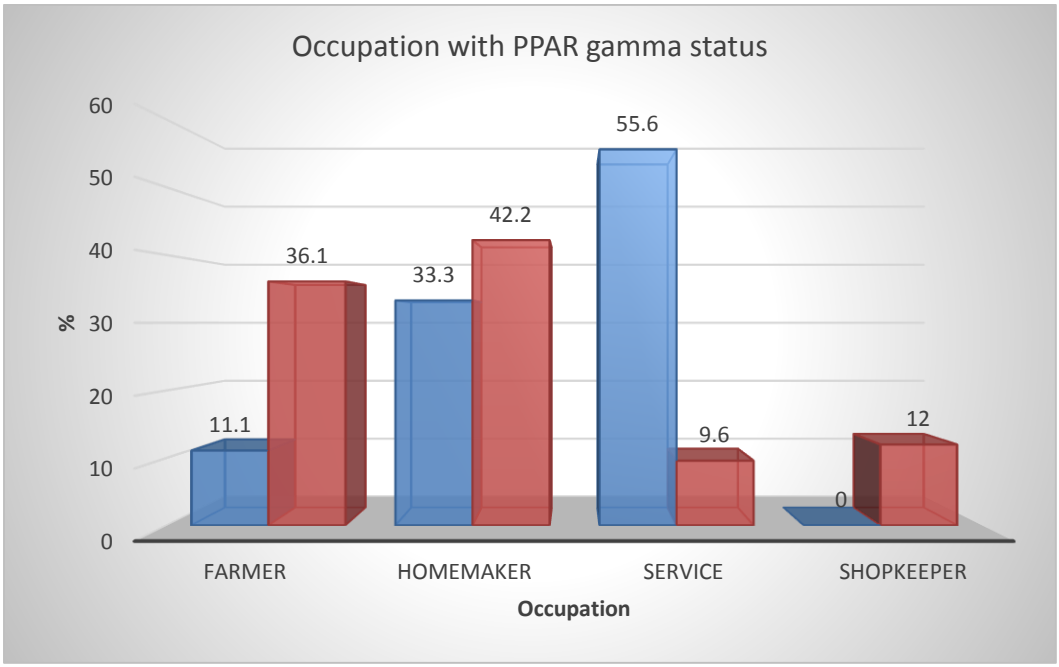
DISTRIBUTION OF PATIENTS ACCORDING TO OCCUPATION

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 Ain 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



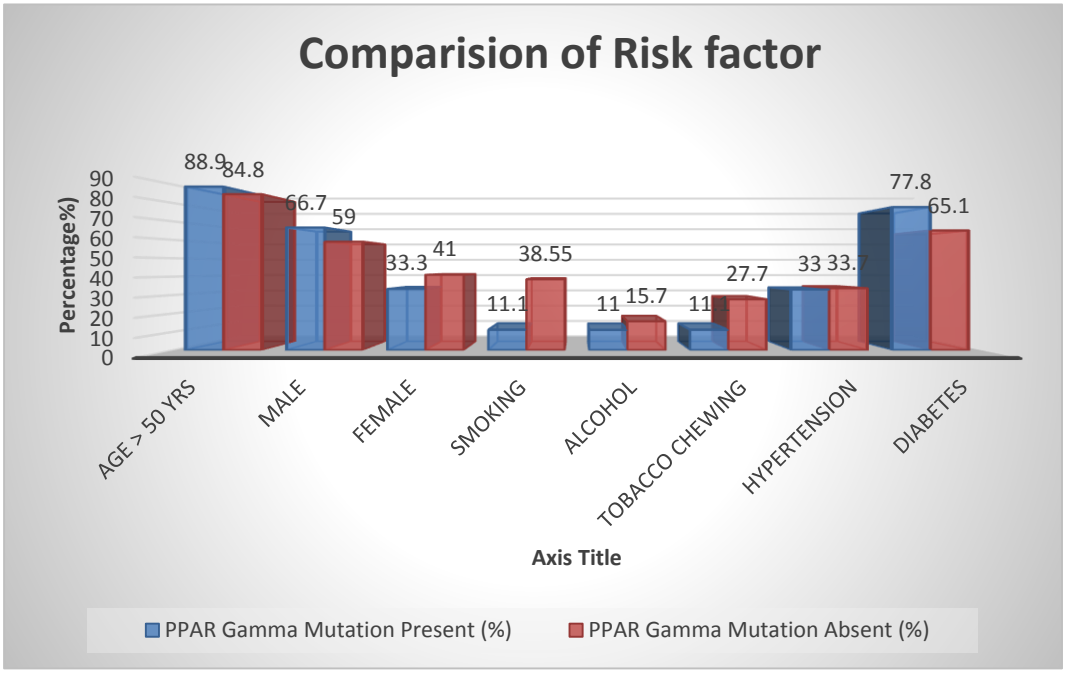
DISTRIBUTION OF PATIENTS ACCORDING TO RISK FACTORS:

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



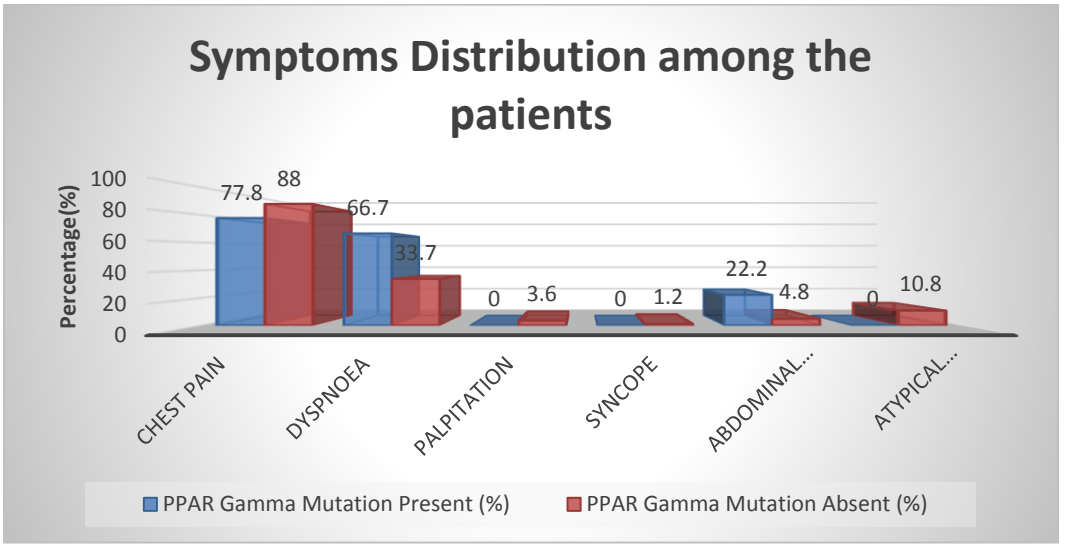
DISTRIBUTION OF PATIENTS ACCORDING TO SYMPTOMS:

In this study, as shown in Table 9, Graph 5, in both group A and group Bthe 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.

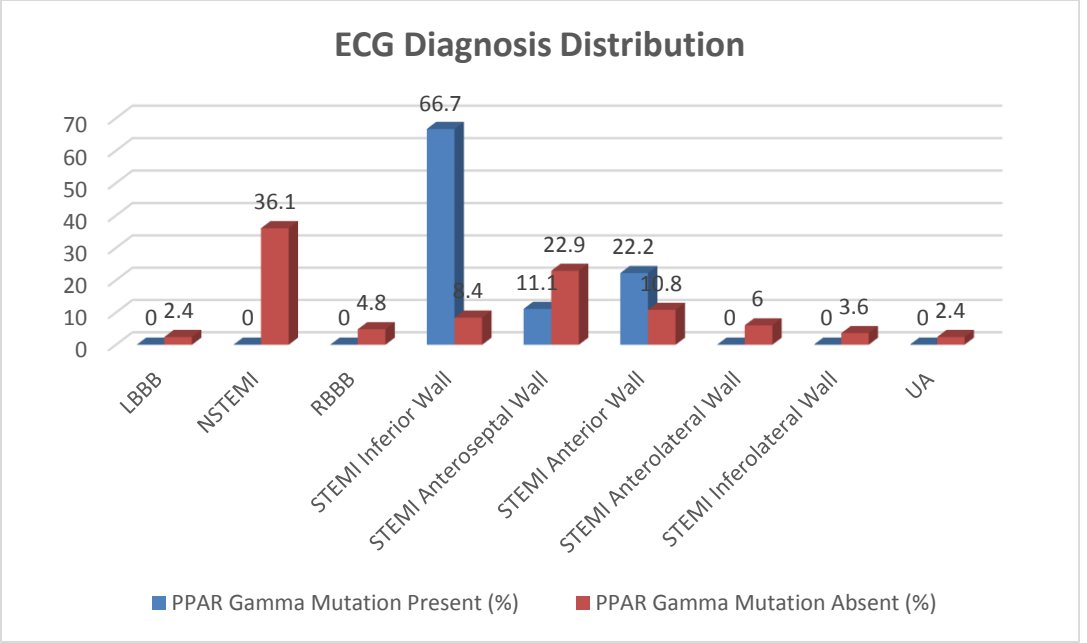
Conditions such as NSTEMI (36.1%) and LBBB (2.4%) were only observed in mutation-absent patients

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



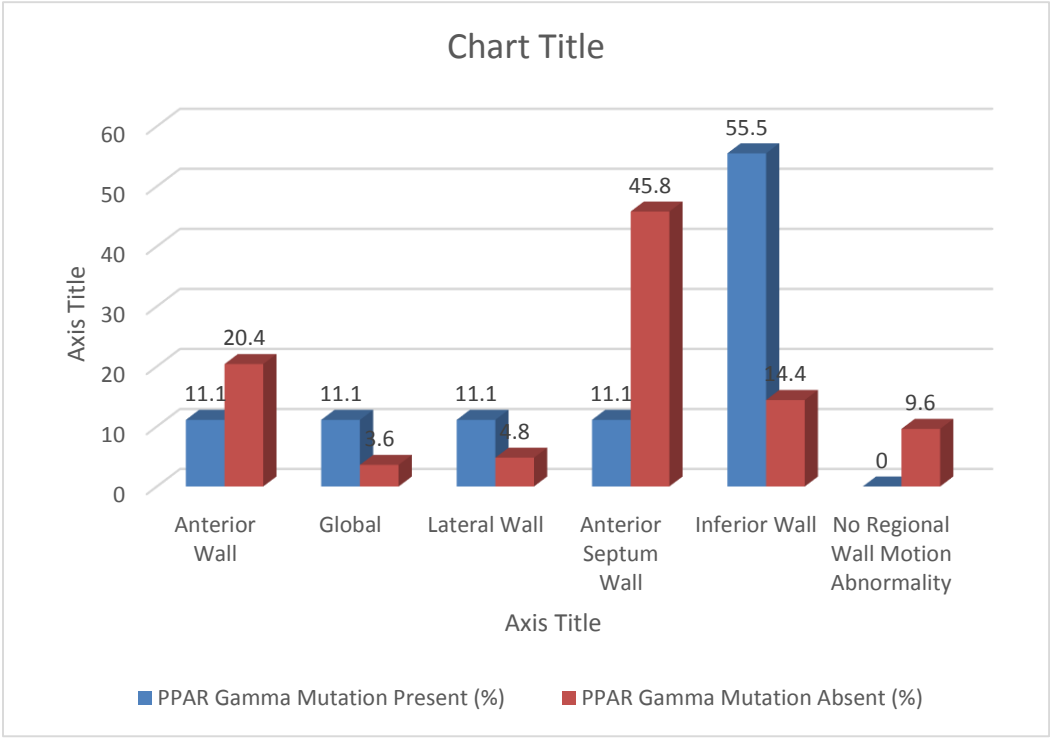
DISTRIBUTION OF PATIENTS ACCORDING TO ECHOCARDIOGRAPHIC VARIABLES:

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 ηg/µl		Sl.No. of DNA samples	OD at 260/280	Concentration in ηg/µl
1	1.86	54		47	1.83	47.2
2	1.75	65		48	1.59	53.2
3	1.40	44		49	1.62	68.2
4	1.90	70		50	1.54	78.1
5	1.57	136		51	1.63	79.2
6	1.98	64		52	1.58	89.1
7	1.84	82		53	1.92	95.2
8	1.92	73		54	1.85	45.3
9	1.65	68		55	1.74	65.1
10	1.79	111		56	1.65	69.4
11	1.85	64		57	1.52	74.2
12	1.81	66		58	1.51	53.2
13	1.75	53		59	1.57	58.1
14	1.59	65		60	1.59	79.2
15	1.66	82		61	1.64	78.1
16	1.51	94		62	1.83	88.2
17	1.88	49		63	1.82	89.1
18	1.92	39		64	1.83	75.1
19	1.93	46		65	1.74	95.1

20	1.74	100
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

66	1.73	115.2
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 TM terminator v3.1was 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).

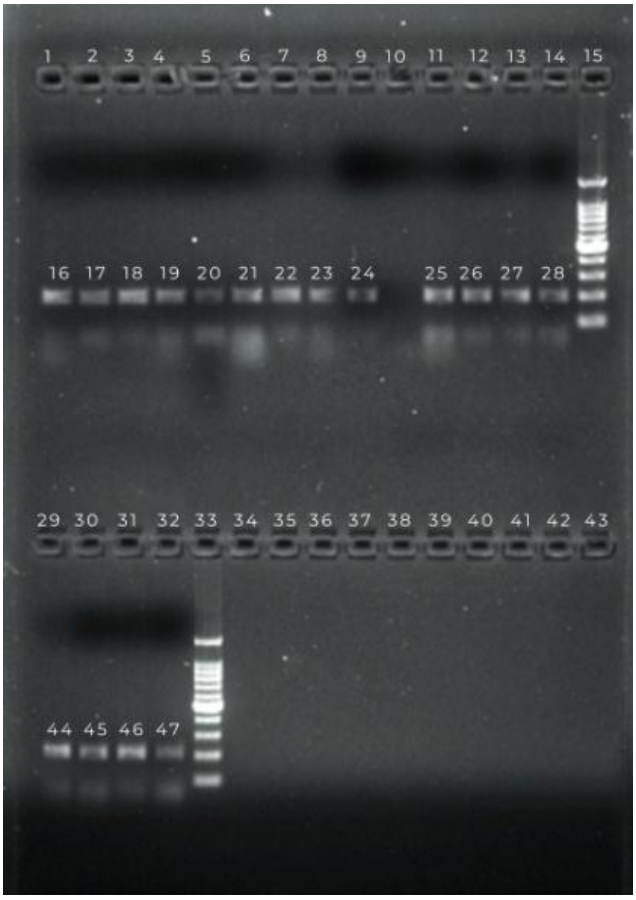


Figure 5 :AGAROSE GEL ELECTROPHORESIS IMAGE OF AMPLIFIED PRODUCTS OF GENE.

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

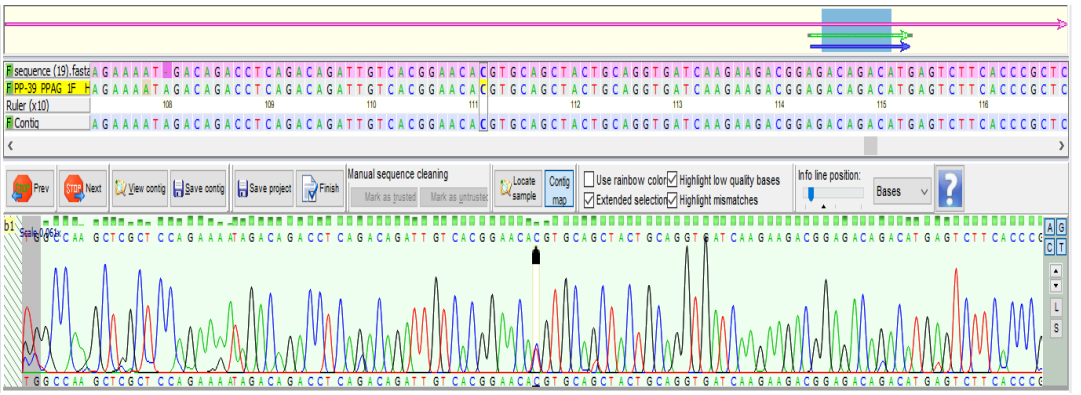


Figure 6b

Figure 6

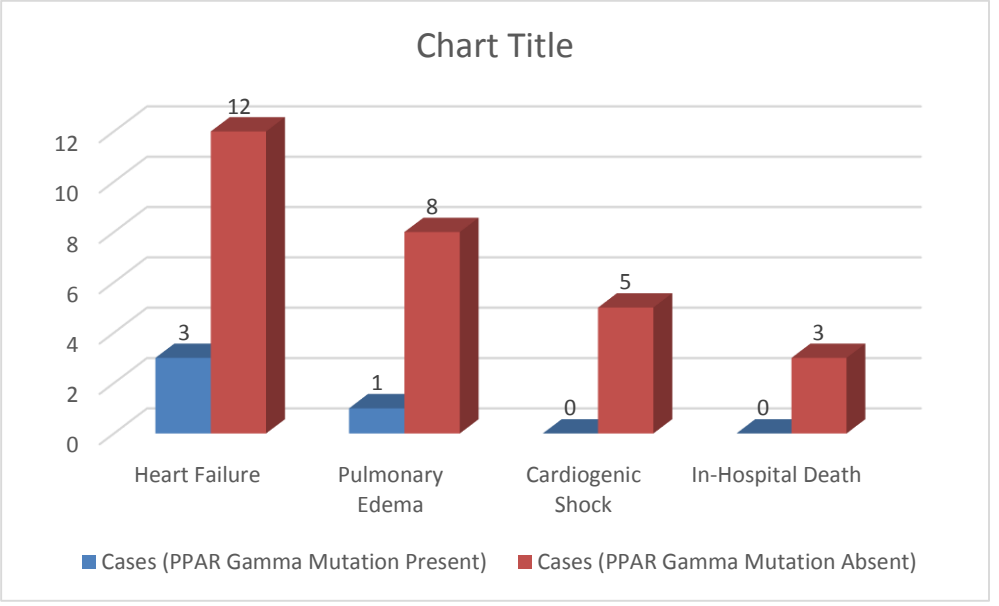
DISTRIBUTION OF PATIENTS ACCORDING TO MAJOR ADVERSE CARDIAC EVENTS:

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

Similarly, the INTERHEART study done by Yosuf 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 1,413,881patients found that 60% male and 40% female are aftected 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 tradional 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 The study "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 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 POLYMORHISM

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 PPARΓ 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 frication 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, thedisease pathogenesis is versatile and complicated posing achallenge 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 Babsent 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 Awas 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 additionMajor Adverse Cardiac events were noted: Heart failure, Pulmonary edema, cardiogenic shock, and in hospital deaths.

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ANNEXURES

ANNEXURE I

INSTITUTIONAL ETHICAL CLEARENCE CERTIFICATE.

BLDE

(DEEMED TO BE UNIVERSITY)

Declared as Deemed to be University u/s 3 of UGC Act, 1956

Accredited with 'A' Grade by NAAC (Cycle-2)

The Constituent College

SHRI B. M. PATIL MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, VIJAYAPURA

BLDE (DU)/IEC/ 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**

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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 ALAMinformed 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:

ಪ್ರಬಂಧ/ಸಂಶೋಧನೆಯಲ್ಲಿ ಭಾಗವಹಿಸಲು ತಿಳುವಳಿಕೆಯುಳ್ಳ ಸಮ್ಮತಿ

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

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

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

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

SL.N.	NAME	AGE	SEX	OCCUPATION	PHONE NO	ADDRESS	IP N.	D.O.A	D.O.D	DURATION OF STAY (DAY)	CHEST PAIN	DYSPNEA	PALPITATION	SYNCOPE	ABDOMINAL PAIN	ATYPICAL MANIFESTATION	DIABETES	HYPERTENSION	FAMILY HISTORY	SMOKING	ALCOHOL	TOBACCO CHEWING
1	KASAPPA VEERUPAKSHAPPA MASALI	60	M	FARMER	9881635360	UKKALI, VIJAPUR	80914	11/03/2024	14/03/2024	06/01/1900	P	A	P	A	A	A	A	P	A	P	A	A
2	LAXMI MUTTAGI	65	F	HOME MAKER		NIDAGUNDI	74222	05/03/2024	15/03/2024	10	p	P	A	A	A	A	A	P	A	A	A	A
3	AMEENABEE SHIKKALAGAR	72	F	HOME MAKER	9341660111	AP DARGA ROAD KARPURMATH COLONY, VIJAPUR	063398	09/03/2024	11/03/2024	2	A	P	A	A	A	A	A	P	A	A	A	P
4	NAGAPPA KALLAPPA SHAHAPUR	54	M	SERVICE	9632070689	AP SIKARAKHANE, VIJAPUR	204903	12/06/2024	04/07/2024	22	P	P	A	A	P	A	A	P	P	A	A	A
5	KALAMMA BADIGER	83	F	HOME MAKER	9880781735	KALIKA NAGAR, BIJAPUR	269278	28/07/2024	03/08/2024	6	A	P	A	A	P	A	A	P	A	A	A	P
6	VITTAL BHARAMANNA HIREKURUBUR	62	M	SERVICE	9686162477	AP LOGAVI TQ TIKOTA , BIJAPUR	269961	29/07/2024	02/08/2024	4	p	P	A	A	P	A	A	P	A	P	A	A
7	SHARANAMMA BIRADAR	48	F	HOME MAKER	9742535506	AP BALAGANUR, TALIKOTI, BIJAPUR	276798	02/08/2024	07/08/2024	5	P	A	A	A	A	P	A	P	A	A	A	P
8	KASTURIBAI KALABURAGI	72	F	HOME MAKER	7259683161	VIJAPUR	274236	01/08/2024	06/08/2024	5	P	A	A	A	A	P	A	P	P	A	A	A
9	ARJUN SURYAVANSHI	42	M	SHOPKEEPER	8971512785	BEHIND RTO OFFICE, RAJAJI NAGAR NEAR NADADEVATI TEMPLE, BIJAPUR	280662	05/08/2024	09/08/2024	4	P	A	A	A	A	A	A	P	A	P	A	P
10	gurubai kavatagi	80	F	HOME MAKER	7411602953	AP UKALABILAGI, JAMKHANDI	280445	05/08/2024	6/8/24 DEATH	1	A	P	A	A	A	A	A	P	A	A	A	A
11	JALABAYI LAMANI	71	F	HOME MAKER	9901795269	NARASAGALGI LT BAGEWADI, BIJAPUR	282769	07/08/2024	08/08/2024	1	P	A	A	A	A	A	A	P	A	A	A	A
12	SHARANGOUDA BIRADAR	52	M	FARMER	9742535506	AP BALAGANNUR TQ, TALIKOTI, BIJAPUR	283568	07/08/2024	12/08/2024	5	P	A	A	A	A	A	A	P	A	A	A	A

SL.N.	PR	SYSTOLIC BP(mmhg)	DIASTOLIC BP (MMHG)	TEMPERATURE	RR	TROP I	HEMOGLOBIN G/DL	TOTAL COUNT	ESR	FBS/PPBS/RBS	BLOOD UREA	Sr. Creatine	SERUM SODIUM	Sr.POTASSIUM	TOTAL CHOLESTROL	TRIGLYCERIDES	HDL(mg/dl)	LDL
1	116	90	60	37	20	4813	7	22	50	rbs-125	50	0.7	144	3.8	162	350	16	120
2	84	130	80	37	18	490.6	7	7.01	15mm /hr	rbs-138 mg/dl	38	0.8	141	4.4	224	125	20	155
3	120	100	60	37	18	34.8	8	13.1	15mm /hr	rbs-186mg/dl	32	1.4	145	3.6	260	350	35	180
4	120	110	80	37	26	1309	15	10.8	17mm /hr	rbs-330mg/dl	42	1.2	134	5.3	246	132	36	183
5	160	160	100	38	32	700.5	12	24.1	14mm /hr	rbs-209mg/dl	23	0.9	131	3.1	172	196	30	103
6	66	140	90	37	18	25605	11	22.5	23mm /hr	rbs-122mg/dl	26	0.8	128	3.5	180	155	35	122
7	100	130	80	37	18	11	11	12.4	8mm/hr	rbs-96mg/dl	28	1	136	4.4	160	175	33	125
8	76	150	90	37	18	7294	13	8	20mm /hr	rbs-95mg/dl	32	1.1	140	4.4	242	145	34	160
9	78	130	80	37	18	44.94	9	10.6	20mm /hr	rbs-209mg/dl	29	0.9	138	3.9	186	155	28	132
10	120	150	90	37	28	170.8	11	9.91	28mm /hr	rbs-91mg/dl	34	1.6	141	5.3	175	146	35	112
11	81	130	80	37	18	1194	13	6.2	14mm /hr	rbs-98mg/dl	19	0.7	143	5.3	288	100	36	198
12	76	120	70	38	18	2144	12	11.9	10mm /hr	FBS-256mg/dl	26	0.8	138	3.4	207	147	26	140

SL.N.	ECG-RATE	RHYTHM	P WAVE	PR INTERVAL	QRS CONFIGURATION	QRS DURATION	QRS AXIS	ST-SEGMENT	T wave	QT	QTc	ECG DIAGNOSIS	positive t wave in avr	Fragmented qrs	2D ECHO REGIONAL MOTION WALL ABNORMALITY	LVEF	I/E	NFKB1 GENE MUTATION	CAG	HEART FAILURE	PULMONARY EDEMA	ARRYTHMIAS	CARDIOGENIC SHOCK	IN HOSPITAL DEATH
1	100BPM	Tachycardia	0.08	0.12	POOR R WAVE PROGRESSION	0.04s	N	ST DEPRESSION V2-V4,1,AVL	INVERTED V3-V6	480s	520s	NSTEMI ANTEROSEPTAL AND INFERIOR WALL	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	4000%	I	A	NOT DONE	P	A	A	A	A
2	92BPM	Regular	0.08 S	0.08 S	Q WAVES V1-V6, NOTCHED Q WAVE V4,V5-RBB PATTERN	0.085	N	RBBB	INVERTED V4-V6	520s	572s	RBBB	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	30	I	A	NOT DONE	P	A	A	A	A
3	65BPM	Regular	0.04 S	0.20 S	RS COMPLEX V1-V5	0.08s	N	ST E 2,3,AVF	INVERTED V1-V6,2,3,AVF	400s	460s	STEMI INFERIOR WALL	A	A	HYPOKINESIA OF INFERIOR AND LATERAL WALL	35	I	A	TVD	P	A	A	A	A
4	150BPM	Tachycardia	0.12 S	0.20 S	DEEP Q WAVE V2,V3,3,2,AVF	0.04s	LAD	ST ELEVATION IN 2,3,AVF	INVERTED 2,3,AVF	320s	453s	STEMI INFERIOR WALL	A	A	INFERIOR WALL HYPOKINESIA	40	I	A	NOT DONE	P	A	A	A	A
5	150BPM	Tachycardia	0.08 S	0.08 S	DEEP S WAVES IN V1-V2 TALL RAVES V5,V6-LVH CHANGES	0.085	N	ST DEPRESSION 3,AVF,V1-V4	NO T WAVE CHANGES	280s	450s	NSTEMI	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	35	I	A	NOT DONE	P	A	A	A	A
6	75bpm	Regular	0.08 S	0.12 s	QRS COMPLEX 2,3,AVF	0.04s	N	ST ELEVATIONV2-V6,2,3,AVF	INVERTED V2-V6	440s	492s	STEMI ANTERIOR WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	50	I	A	SVD	A	A	A	A	A
7	60BPM	Regular	0.08 S	0.12 s	RS COMPLEX V1-V5	0.04s	N	ST ELEVATION V2,V3,AVL	INVERTED V2,AVL,V3	480s	480s	STEMI ANTERIOR WALL	A	A	NO MOTION WALL ABNORMALITY	60	I	A	SVD	A	A	A	A	A
8	75bpm	Regular	0.08 S	0.12 s	RS COMPLEX V1-V5 NOTCHED R WAVE IN V1	0.04s	N	ST DEPRESSION V2-V4	NO T WAVE CHANGES	480s	530s	NSTEMI	A	A	HYPOKINESIA OF INFERIOR AND LATERAL WALL	45	I	A	NOT DONE	P	A	A	A	A
9	75bpm	Regular	0.12 S	0.16 S	DEEP Q WAVES V1-V5	0.04s	N	ST E V2-V5,2,3,AVF	INVERTED V2-V5	440s	490s	STEMI INFERIOR WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	50	I	A	DVD	A	A	A	A	A
10	100BPM	Tachycardia	0.04 S	0.20 S	rSR PATTERN 2,3,AVF, POOR R WAVE PROGRESSION	0.04s	LAD	ST E V2-V6	INVERTED V2-V6	280s	360s	STEMI ANTEROSEPTAL WALL	A	A	GLOBAL HYPOKINESIA	25	I	A	DVD	P	P	A	A	P
11	100BPM	Tachycardia	0.08 S	0.12 s	Q WAVES 2,3,AVF,V5-V6	0.04s	N	ST E 2,3,AVF	INVERTED 2,3,AVF	440s	560s	STEMI INFERIOR WALL	A	A	INFERIOR WALL HYPOKINESIA	55	I	A	SVD	A	A	A	A	A
12	75bpm	Regular	0.08 S	0.20 S	Q WAVES 3,AVF	0.04s	LAD	ST DEPRESSION V2-V4,2,3,AVF	INVERTED 2,3,AVF	480s	530s	NSTEMI	A	A	HPOKINESIA OF ANTEIOR AND ANTEROLATERAL WALL	45	I	A	TVD	A	A	A	A	A

23	22	21	20	19	18	17	16	15	14	13	SL N.
SANJEEV GUDDEVADI	HANAMANTH PAWAR	SIDDAPPA KENCHAPPA CHALAMI	BHIMANNA SHANTAPPA KUMBAR	KASHINATH BALCHABAL	SHARANAPPA AYAPPA KOPPAD	GURUBAI SUDAM	GOPU CHAVAN	NEELAPPA HAJERI	MEENAXI SHABADI	MALAKAPPA WADDAR	NAME
53	40	65	68	61	55	55	70	75	75	54	AGE
M	M	M	M	M	M	F	M	M	F	M	SEX
SHOPKEEPER	SHOPKEEPER	FARMER	FARMER	FARMER	FARMER	HOME MAKER	SERVICE	FARMER	HOME MAKER	SERVICE	OCCUPATION
7899544256	1111222244	9741183777	8722148224	8971212290	7259942002	9686749275	9740885955	9741363117	7411044234	9663621801	PHONE NO
AP KANAKADAS BADAVANE, BIJAPUR	BIJAPUR	AP YALAJERI TQ, MUDDEBIHAL, BIJAPUR	KUMBAR ONI INDI, BIJAPUR	KALLAKAVATAGI, AP BABANAGAR, TQ TIKOTA, BIJAPUR	AP SHIVANAGI TQ, BIJAPUR	TADAVALAGA, INDI	A/P ITTANGIHAL 03,TQ BIJAPUR	AT TONA SYYAL TQ, BIJAPUR	AP SHASHRI NAGAR, BEHIND GODAVARI OTAL	A/P TREJARI COLONY, BIJAPUR	ADDRESS
290487	288820	288854	290163	290105	290006	283630	288016	287064	284976	15143	IP N.
13/08/2024	12/08/2024	12/08/2024	12/08/2024	12/08/2024	12/08/2024	07/08/2024	10/08/2024	10/08/2024	08/08/2024	07/08/2024	D.O.A
17/08/2024	14/08/2024	20/08/2024	19/08/2024	19/08/2024	19/08/2024	12/08/2024	19/08/2024	13/08/2024	13/08/2024	10/08/2024	D.O.D
4	2	8	7	7	6	5	9	3	5	3	DURATION OF STAY (DAY)
P	P	P	P	P	A	A	P	P	P	P	CHEST PAIN
A	A	P	A	A	A	P	P	A	A	A	DYSPNEA
A	A	A	A	A	A	A	A	A	A	A	PALPITATION
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	A	A	A	A	A	ATYPICAL MANIFESTATION
A	A	A	A	A	A	A	A	A	A	A	DIABETES
P	P	P	P	P	P	P	P	P	P	P	HYPERTENSION
A	P	A	A	A	A	A	A	A	A	P	FAMILY HISTORY
A	P	P	P	A	P	A	A	A	A	P	SMOKING
A	A	A	A	A	P	A	A	A	A	P	ALCOHOL
A	P	P	P	A	A	A	P	A	A	P	TOBACCO CHEWING
100	96	78	78	80	80	68	106	32	100	86	PR
150	120	160	126	136	220	130	200	70	160	130	SYSTOLIC BP(mmhg)
90	70	80	80	80	120	80	110	40	90	80	DIASTOLIC BP (MMHG)
37	37	38	37	38	37	37	37	37	36	37	TEMPERATURE
18	16	26	16	18	18	20	20	18	22	20	RR
500.2	1.4	2039	7	42	7.6	11	28.3	17.8	110.1	4328	TROP I
13	10	10	9	12	12	12	10	15	11	9	HEMOGLOBIN G/DL
6.99	6.86	8.77	9.2	5.35	8.97	11.2	13.3	8.05	4.8	8.82	TOTAL COUNT
20mm /hr	5mm/ hr	10mm /hr	10mm /hr	15mm /hr	15mm /hr	15mm /hr	10mm /hr	10mm /hr	53mm /hr	30mm /hr	ESR
rbs-242mg/ dl	rbs-149mg/ dl	rbs-193mg/ dl	rbs-150mg/ dl	rbs-150mg/ dl	rbs-152mg/ dl	rbs-122mg/ dl	rbs-106mg/ dl	rbs-120mg/ dl	rbs-122mg/ dl	rbs-457mg/ dl	FBS/PPBS/RBS
16	17	27	16	31	30	23	46	29	53	29	BLOOD UREA
0.8	0.8	0.7	0.8	0.9	0.7	0.8	1.7	1.2	1.6	0.6	Sr. Creatine
138	138	139	139	139	143	140	134	139	127	136	SERUM SODIUM
3.7	4.4	4.4	4	4.6	4.7	4.7	5.1	4.2	3.9	4.3	Sr.POTASSIUM
186	223	155	169	150	144	320	262	200	306	181	TOTAL CHOLESTROL
12	100	125	112	160	265	344	117	263	366	139	TRIGLYCERIDES
27	35	20	23	18	29	35	21	33	26	32	HDL(mg/dl)
131	136	102	106	110	168	263	201	161	241	111	LDL
100BPM	100BPM	100BPM	75bpm	100BPM	75bpm	100BPM	75bpm	35BPM	150BPM	100BPM	ECG-RATE
Tachycardia	Tachycardia	Tachycardia	Regular	Tachycardia	Regular	Regular	Regular	Bradycardia	Tachycardia	Tachycardia	RHYTHM
0.08 s	0.08 s	0.08 s	0.04 s	0.08 s	0.08 s	0.08 s	0.12 s	0.08 s	0.04 s	0.08 s	P WAVE
0.12 s	0.16 s	0.12 s	0.12 s	0.12 s	0.12 s	0.12 s	0.20 s	0.24 s	0.08 s	0.16 s	PR INTERVAL
NOTCHED QRS COMPLEX 2,3,AVL,AVF,V1-V4	POOR R WAVE PROGRESSION Q WAVES IN 3,AVF	DEEP Q WAVES V1-V2	RS COMPLEXES V2-V6	QRS COMPLEXES V2-V6	Q WAVES 2,3,AVF	NOTCHES Q WAVE IN 3, qRQ PATTERN AVF	DEEP Q WAVES V1-V3	deep q wave v1-V2	DEEP Q WAVE AAVR ,V1, V2	DEEP Q WAVE 1,AVR,AVL, POOR R WVE PROGRESSION	QRS CONFIGURATION
0.04s	0.04s	0.04s	0.04s	0.04s	0.04s	0.04s	0.04s	0.04s	0.04s	0.12s	QRS DURATION
N	LAD	N	LAD	N	N	LAD	N	N	N	LAD	QRS AXIS
ST ELEVATION 3,AVF,2	ST DEPRESSION 3,AVF	ST DEPRESSION-V3 TO V6	ST DEPRESSION IN V2-V6	ISOELECTRIC ST SEGMENT	ST DEPRESSION 2,3,AVF	ST DEPRESSION 2,3,AVF	ST E V2-V6,2.AVF	ST DEP V2,V3	ST DEP-V3-V6,2,3,AVF	ST DEPRESSION V2-V4,1.AVL	ST-SEGMENT
INVERTED V2-V4	NO T WAVE CHANGES	INVERTED V2-V6	INVERTED V2-V6	INVERTED V2-V4, AVL	INVERTED 2,3,AVF	INVERTED 2,3,AVF	INVERTED V2-V6	INVERTED 2,3,AVF	INVERTED 2,3,AVF,V3-V6	NO T WAVE CHANGES	T wave
360s	400s	400s	440s	400s	440s	400s	440s	360s	320s	520s	QT
460s	510s	560s	470s	510s	447s	450s	492s	460s	410s	670s	QTc
STEMI INFERIOR WALL	NSTEMI	NSTEMI	NSTEMI	NSTEMI	NSTEMI	NSTEMI	STEMI ANTEROSEPTAL WALL	NSTEMI	NSTEMI	NSTEMI	ECG DIAGNOSIS
A	A	positive t wave in avr	A	A	A	A	A	A	A	A	positive t wave in avr
A	A	A	A	A	A	PINI	A	A	A	A	Fragmented qrs
INFERIOR WALL HYPOKINESIA	INFERIOR WALL HYPOKINESIA	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	HPOKINESIA OF ANT ANTEROLATERAL WALL	NO MOTION WALL ABNORMALITY	HYPOKINESIA OF INFERIOR AND INFERIO LATERAL WALL	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	HYPOKINESIA OF INFERIOR AND POSTERIOR WALL	NO MOTION WALL ABNORMALITY	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	2D ECHO REGIONAL MOTION WALL ABNORMALITY
55	60	55	55	55	45	60	40	45	60	35	LVEF
I	I	I	I	I	I	E	I	I	E	I	I/E
A	A	A	A	A	A	A	A	A	A	A	NFKB1 GENE MUTATION
TVD	SVD	TVD	SVD	DVD	NOT DONE	SVD	NOT DONE	NOT DONE	NOT DONE	NOT DONE	CAG
A	A	A	A	A	A	A	P	A	A	P	HEART FAILURE
A	A	A	A	A	A	A	A	P	A	A	PULMONARY EDEMA
P	A	A	A	A	A	A	A	A	A	A	ARRYTHMIAS
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

35	34	33	32	31	30	29	28	27	26	25	24	SL N.
IRAPPA S GALGALI	SIDRAY TOTAD	MAHADEVI KHAKANDAKI	CHANAWWA SOLAPUR	HAMEEDABI MAKANDAR	VAUDEO NADGOUDA	RAVI MANE	MAKANABAI PAWAR	SWALIHA BEGUM MUNIRKHANPATHAN	BASWANAND KUMBAR	SAHEBI HUSEAINBASHA MASALI	REVANSIDAPPaA NG	NAME
50	52	70	70	67	62	41	70	49	64	75	69	AGE
M	M	F	F	F	M	M	F	F	M	F	M	SEX
FARMER	SERVICE	HOME MAKER	HOME MAKER	HOME MAKER	SERVICE	SERVICE	HOME MAKER	HOME MAKER	FARMER	HOME MAKER	SERVICE	OCCUPATION
9380617835		9901114294	8861866240	9972698384	8618407896	9036568122	7057275359	9036225641	8660371887	8217666426	7019729120	PHONE NO
C/O SANGAPPA GALAGALI, MAMADAPUR	MALIONI, NAGANNUR, JHAMKHANDI, BIJAPUR	BABALESHWAR TQ, BIJAPUR	HALAGANI, TQ BABALESHWAR, BIJAPUR	MANAGULI, BASAVANBAGEWADI, BIJAPUR	J M ROAD NEAR BADIKAMAN, BIJAPUR	AP HANCHANAL, BIJAPUR	AT POST UTANAL, BIJAPUR	STATION ROAD, HASHIMPUR DARGA, BIJAPUR	AT POST HALAGUNAKI, TQ INDI, BIJAPUR	AP WATER TANK, BIJAPUR	C/O GURULINGAPPA NG 378 WARD NO 22 SR COLONY JAL NAGAR, BIJAPUR	ADDRESS
295874	295755	295843	295770	295846	294569	294855	294345	294310	253478	293524	290730	IP N.
17/08/2024	16/08/2024	16/08/2024	16/08/2024	16/08/2024	16/08/2024	16/08/2024	16/08/2024	15/08/2024	14/08/2024	14/08/2024	13/08/2024	D.O.A
21/08/2024	20/08/2024	21/08/2024	23/08/2024	24/08/2024	22/08/2024	19/08/2024	20/08/2024	21/08/2024	21/08/2024	16/08/2024	20/08/2024	D.O.D
4	4	5	7	8	6	3	4	6	7	2	7	DURATION OF STAY (DAY)
P	P	P	P	P	P	P	A	P	P	P	P	CHEST PAIN
A	A	A	A	P	A	P	P	P	A	P	A	DYSPNEA
A	A	A	A	A	A	A	A	A	A	A	A	PALPITATION
A	A	A	A	A	A	A	A	A	A	A	A	SYNCOPE
A	A	A	A	A	A	A	A	A	A	A	A	ABDOMINAL PAIN
A	A	A	A	A	A	A	A	A	A	A	A	ATYPICAL MANIFESTATION
P	P	P	P	A	A	A	A	A	A	A	A	DIABETES
P	P	P	P	P	P	P	P	P	P	P	P	HYPERTENSION
A	A	A	A	A	A	A	A	A	A	P	A	FAMILY HISTORY
A	P	A	A	A	A	P	A	A	P	A	P	SMOKING
A	P	A	A	A	A	P	A	A	A	A	A	ALCOHOL
A	P	P	P	P	A	A	A	P	A	A	A	TOBACCO CHEWING
96	86	80	82	90	82	60	114	68	56	112	80	PR
150	110	94	180	110	150	100	140	120	140	140	120	SYSTOLIC BP(mmhg)
100	70	70	110	70	90	60	80	80	70	90	70	DIASTOLIC BP (MMHG)
98	97	97	97	97	97	97	98	97	97	97	97	TEMPERATURE
20	22	18	22	22	22	22	22	24	18	16	22	RR
25605	1.22	50	2.3	253.3	2.3	909.2	496.4	26.3	556.7	18.5	58.2	TROP I
13	14	9	13	14	8	12	10	14	12	10	12	HEMOGLOBIN G/DL
15.2	5.2	6.23	5.23	12.9	9.8	13.5	5.35	10.2	12.1	8.94	5.76	TOTAL COUNT
10mm /hr	10mm /hr	15mm /hr	10mm /hr	10mm /hr	20mm /hr	5mm/ hr	20mm /hr	35mm /hr	10mm /hr	15mm /hr	10mm /hr	ESR
rbs-106mg/ dl	rbs-186mg/ dl	rbs-158mg/ dl	rbs-100mg/ dl	rbs-116mg/ dl	rbs-342mg/ dl	rbs-126mg/ dl	rbs-158mg/ dl	rbs-160mg/ dl	rbs-128mg/ dl	rbs-128mg/ dl	rbs-138mg/ dl	FBS/PPBS/RBS
25	28	30	30	43	50	24	57	33	28	25	19	BLOOD UREA
0.6	1.4	0.7	1	0.7	1.2	1	1.9	0.7	0.7	0.8	1.1	Sr. Creatine
140	136	145	139	146	138	141	149	145	140	142	138	SERUM SODIUM
3.6	4.3	3.4	4.8	3.6	4.5	4.4	4.6	4.7	4.1	4.8	4.2	Sr.POTASSIUM
172	194	120	200	236	190	244	219	255	170	250	238	TOTAL CHOLESTROL
96	301	218	172	188	196	286	366	178	261	178	268	TRIGLYCERIDES
30	39	35	33	27	36	20	33	27	39	30	31	HDL(mg/dl)
110	126	130	159	178	119	177	136	166	198	110	150	LDL
100BPM	72bpm	100BPM	100BPM	82BPM	100BPM	75BPM	100BPM	75bpm	150BPM	150BPM	75bpm	ECG-RATE
Tachycardia	Regular	Regular	Regular	Regular	Tachycardia	Regular	Tachycardia	Regular	Tachycardia	Tachycardia	Regular	RHYTHM
0.12 s	0.12 s	0.08 s	0.12 s	0.12 s	0.12 s	0.12 s	0.04 s	0.12 s	0.08 s	0.04 s	0.08 s	P WAVE
0.12 s	0.12 s	0.16 s	0.16 s	0.16 s	0.16 s	0.12 s	0.16 s	0.20 s	0.04 s	0.12 s	0.16 s	PR INTERVAL
POOR R WAVE PROGRESSION	rs complexes v4-v6	Rs complexes V1-V4	Rs complexes V1-V4	Q WAVES V1-V4	DEEP Q WAVES V1-V3	Q WAVE IN V1	NOTCHED QRS 2,3,AVF, QSq pattern v1-v3	Rs complexes V1-V4	DEEP Q WAVES V1-V3	Q WAVES V1-V4	Q WAVES V1-V6,AVL	QRS CONFIGURATION
0.125	0.125	0.04s	0.04s	0.125	0.125	0.125	0.08s	0.125	0.085	0.04s	0.125	QRS DURATION
LAD	N	N	N	N	LAD	N	LAD	N	N	N	N	QRS AXIS
ST ELEVATION 2,3,AVF	ST DEPRESSION 2,3,AVF,V4-V6	ST E V1-V4	ST DEPRESSION 3,AVF,2	ST ELEVATION V1-V4	ST E V2-V4,2,3,AVF	ST E V2-V4,AVF	ST E V1-V3	ST DEPRESSION 2 ,V1,AVR	ST ELEVATION V2-V5	ST E V2-V5	ST ELEVATION V2,V3,, DEPRESSION 2,3,AVF	ST-SEGMENT
INVERTED 2,3,AVF,V4-V6	INVERTED V4-V6,3,AVF	INVERTED3,AVF	NO T WAVE CHANGES	HYPERACUTE T WAVE V1-V5	INVERTED V2-V4	INVERTED 2,3,AVF,V1-V6	inverted v2-v3	NO T WAVE CHANGES	INVERTED V1-V6	INVERTED V2-V6	INVERTED V1-V4	T wave
400s	380s	440s	480s	360s	440s	480s	440s	440s	440s	360s	400s	QT
510s	420s	560s	620s	420s	560s	530s	560s	490s	490s	562s	447s	QTc
STEMI INFERIOR WALL	NSTEMI	STEMI ANTEROSEPTAL WALL	NSTEMI	STEMI ANTEROSEPTAL WALL	STEMI ANTEROSEPTAL WALL	STEMI INFERIOR WALL	LBBB	NSTEMI	STEMI ANTEROSEPTAL WALL	STEMI ANTEROSEPTAL WALL	STEMI ANTEROSEPTAL WALL	ECG DIAGNOSIS
A	A	A	A	A	A	A	A	A	A	A	A	positive t wave in avr
A	A	A	A	A	A	A	P	A	A	A	A	Fragmented qrs
INFERIOR WALL HYPOKINESIA	HYPOKINESIA OF ANTEROINFERIOR WALL	HPOKINESIA OF ANTEROLATERAL WALL	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	HYPOKINESIA OF INFERIOR AND POSTERIOR WALL	NO MOTION WALL ABNORMALITY	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	2D ECHO REGIONAL MOTION WALL ABNORMALITY
45|	60|	40|	60|	35|	38|	35|	50|	50|	60|	35|	45|	LVEF
												I/E
A	A	A	A	A	A	A	A	A	A	A	A	NFKB1 GENE MUTATION
NOT DONE	SVD	SVD	DVD	NOT DONE	NOT DONE	TVD	NOT DONE	DVD	TVD	NOT DONE	DVD	CAG
A	A	A	A	A	P	P	A	A	A	P	A	HEART FAILURE
A	A	A	A	P	A	A	A	A	A	A	A	PULMONARY EDEMA
A	A	A	A	P	A	A	A	A	A	A	A	ARRYTHMIAS
A	A	A	A	P	A	A	A	A	A	A	A	CARDIOGENIC SHOCK
A	A	A	A	P	A	A	A	A	A	A	A	IN HOSPITAL DEATH

SL N.	36	37	38	39	40	41	42	43	44	45	46	47	48	49
NAME	SUBHASH BAJANTRI	Bandagi Saab Mokashi	AMBAWWA SAGAI	HANAMANTH KAMBLE	LAXMAN HARIWAL	VITTHAL DEVKHATE	DJ PATEL	SHRISHAIL BIRADAR	DUNDAPPA GHANTI	MUNNIRA JATH	BASANNA TORAVI	TUKARAM RATHOD	MAHADEVI KOLHAR	BANDAWA PATHAN SHETTY
AGE	65	69	65	72	63	70	83	65	60	50	58	54	69	76
SEX	M	M	F	M	M	M	M	M	M	F	M	M	F	F
OCCUPATION	FARMER	FARMER	HOME MAKER	SERVICE	FARMER	FARMER	SERVICE	SHOPKEEPER	SERVICE	HOME MAKER	FARMER	SHOPKEEPER	HOME MAKER	HOME MAKER
PHONE NO	9686395794	9902591942	9972454965	9036919366	7899544256				8884888837		9353675745	9921255089	9980725920	9844704728
ADDRESS	JUMANAL, BIJAPUR	GUBBEWADI	JALAKI INDI	JAMKANDI	AP KANAKADAS BADAVANNE, BIJAPUR	KYATANKERI, INDI, BIJAPUR	BAGEWADI, BIJAPUR	BAGEWADI, BIJAPUR	DARGA ROAD	VIJAPUR	DOMANAL	HANCHINAL	MUDHOL	HONNUTAGI
IP N.	291939	298444	300611	1502	21112	23252	212243	14233	0166	00020	00055	00023	00025	407405
D.O.A	14/08/2024	19/08/2024	20/08/2024	22/08/2024	21/08/2024	21/08/2024	21/08/2024	21/08/2024	25/08/2024	26/08/2024	26/08/2024	27/08/2024	28/08/2024	28/08/2024
D.O.D	28/08/2024	25/08/2024	29/08/2024	02/09/2024	28/08/2024	28/08/2024	28/08/2024	28/08/2024	01/09/2024	30/08/2024	01/09/2024	03/09/2024	03/09/2024	03/09/2024
DURATION OF STAY (DAY)	14	6	9	11	7	7	7	7	6	4	5	7	6	6
CHEST PAIN	P	P	P	P	P	P	P	P	P	P	A	P	P	P
DYSPNEA	A	A	A	P	P	P	A	P	A	A	P	A	A	A
PALPITATION	A	A	A	A	A	A	A	A	A	A	P	A	A	A
SYNCOPE	A	A	A	A	A	A	A	A	A	A	A	A	A	A
ABDOMINAL PAIN	A	A	A	A	A	A	A	A	A	A	A	A	A	A
ATYPICAL MANIFESTATION	A	A	A	A	A	A	A	A	A	A	P	A	A	A
DIABETES	P	P	P	P	P	P	P	P	P	P	P	P	P	P
HYPERTENSION	P	P	P	P	P	P	P	P	P	P	P	P	P	P
FAMILY HISTORY	A	A	A	A	A	A	A	A	A	A	A	A	A	A
SMOKING	P	A	A	P	P	P	A	A	A	A	P	A	A	A
ALCOHOL	A	A	A	A	P	P	A	P	A	A	P	A	A	A
TOBACCO CHEWING	A	A	A	P	P	P	A	P	A	A	P	A	P	A
PR	90	80	78	90	60	58	40	97	86	52	90	70	78	82
SYSTOLIC BP(mmhg)	70 SYS TO LIC	110	130	14	170	130	130	100	160	80	110	140	150	170
DIASTOLIC BP (MMHG)		70	80	90	100	70	80	70	90	60	70	80	90	80
TEMPERATURE	37	37	37	37	37	38	37	37	37	37	37	37	37	37
RR	22	18	18	18	20	18	20	20	20	18	18	20	18	18
TROP I	11.7	1540	556.7	100	36	1023	17	88	150	2000	1154	415.1	179	312
HEMOGLOBIN G/DL	13	12	12	11	16	13	10	13	15	12	13	12	12	9
TOTAL COUNT	24.2	11.1	12	19.6	####	12.2	12	13.3	20.3	14	8.5	8.3	5.8	12.6
ESR	15mm /hr	10mm /hr	30mm /hr	29	10mm /hr	10mm /hr	10mm /hr	5mm/ hr	10mm /hr	10mm /hr	10mm /hr	15mm /hr	15mm /hr	15mm /hr
FBS/PPBS/RBS	rbs-108mg/ dl	rbs-149mg/ dl	rbs-209mg/ dl	RBS-236	rbs-268mg/ dl	rbs-161mg/ dl	rbs>222 mg/dl	rbs-123mg/ dl	RBS-137mg/ dl	rbs-120mg/ dl	RBS-133mg/ dl	RBS-180mg/ dl	RBS-192mg/ dl	
BLOOD UREA	43	20	23	61	26	34	20	17	17	18	35	27	30	18
Sr. Creatine	1.1	0.8	1.2	1.2	1.3	1	0.8	0.7	0.7	0.6	1.4	0.9	0.7	0.5
SERUM SODIUM	135	132	139	138	136	142	140	131	146	142	134	136	140	140
Sr.POTASSIUM	3.7	3.2	4.2	4.2	3.8	3.5	3.2	4.3	5	4.2	3.8	4.2	3.3	3.2
TOTAL CHOLESTROL	198	235	200	180	298	181	150	186	150	270	175	250	180	200
TRIGLYCERIDES	172	190	170	150	120	292	192	244	120	460	196	135	250	175
HDL(mg/dl)	36	30	26	35	31	30	30	35	22	24	26	32	35	30
LDL	141	162	116	115	206	115	102	122	106	196	101	188	115	136
ECG-RATE	100BPM	75bpm	100BPM	75bpm	60BPM	60BPM	43BPM	100BPM	75bpm	50BPM	100BPM	75bpm	75bpm	100BPM
RHYTHM	Tachycardia	Regular	Tachycardia	Regular	Regular	Regular	Bradycardia	Tachycardia	Regular	Bradycardia	Tachycardia	Regular	Regular	Tachycardia
P WAVE	0.12 s	0.08 s	0.08 s	0.08 s	0.12 s	0.08 s	0.12 s	0.12 s	0.08 s	0.08 s	0.08 s	0.08 s	0.08 s	0.08 s
PR INTERVAL	0.20 s	0.16 s	0.12 s	0.16 s	0.16 s	0.16 s	0.24 s	0.16 s	0.20 s	0.12 s	0.12 s	0.16 s	0.12 s	0.16 s
QRS CONFIGURATION	Q WAVES V1-V4	N	Q WAVES 2,3,AVF	Q WAVE IN V1,V6	S WAVE IN V1+R IN V8>38S-LVH	Q WAVES IN 2,3,AVF	rs in2,3,avf	q wave V1,3,AVR	Q WAVES 2,3,AVF	N	Q WAVES 2,3,AVF,V5-V6	NOTCHED QRS 2,3,AVF,V2-V6	N	Q WAVES 2,3,AVF
QRS DURATION	0.085s	0.04s	0.04s	0.04s	0.12S	0.04S	0.12s	0.12S	0.04s	0.04s	0.04s	0.08S	0.04s	0.04s
QRS AXIS	N	LAD	LAD	LAD	LAD	LAD	LAD	LAD	LAD	N	N	RAD	LAD	N
ST-SEGMENT	ST E V2-V5 WITH ST DEP 2,3,AVF	ST DEP V2,V3	ST E V2-V4,AVF,2,3	ST E V2-V5	ST DEP I,AVL,V5,V6	ST E 2,3,AVF	ST E V1-V3	ST ELEVATION 3,AVF,2	ST E 2,3,AVF	ST E 2,3,AVF	ST E 2,3,AVF,V2-V6	ST E 2,3,AVF,V2-V6	ST DEP V2,V3,V4,V5,V6	ST E 2,3,AVF
T wave	INVERTED V2-V3	NO T WAVE CHANGES	INVERTED V2,3,	INVERTED V1,V6	INVERTED V1-V6	INVERTED 2,3,	INVERTED V1	INVERTED V2,V3	INVERTED 2,3,AVF	HYPERACUTE T WAVE 2,3,AVF	INVERTED 2,3,V4,V5	NO T WAVE CHANGES	INVERTED V2-V6	INVERTED V2-V3
QT	440s	480s	480	520s	400s	400s	480s	360s	400s	520s	400s	400s	440s	480
QTc	560s	530s	620	580s	410s	400s	400s	480s	470s	470s	510s	470s	490s	620s
ECG DIAGNOSIS	STEMI ANTEROSEPTAL WALL	STEMI ANTERIOR WALL	STEMI INFERIOR WALL	STEMI ANTERIOR WALL	NSTEMI	STEMI INFERIOR WALL	STEMI ANTEROSEPTAL WALL	STEMI INFERIOR WALL	STEMI INFERIOR WALL	STEMI INFERIOR WALL	STEMI ANTEROSEPTAL WALL	RBBB	NSTEMI	STEMI INFERIOR WALL
positive t wave in avr	A	A	A	A	A	A	A	A	A	A	A	A	A	A
Fragmented qrs	A	A	A	A	A	A	A	A	A	A	A	P	A	A
2D ECHO REGIONAL MOTION WALL ABNORMALITY	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	HYPOKINESIA OF ANT ANTEROLATERAL	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	HPOKINESIA OF ANT ANTEROLATERAL WALL	HYPOKINESIA OF INFERIOR AND POSTERIOR WALL	HYPOKINESIA OF INFERIOR AND INFERIO LATERAL WALL	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	HYPOKINESIA OF INFERIOR WALL	INFERIOR WALL HYPOKINESIA	INFERIOR WALL HYPOKINESIA	HPOKINESIA OF ANT ANTEROLATERAL WALL	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	INFERIOR WALL HYPOKINESIA	INFERIOR WALL HYPOKINESIA
LVEF	25	50	50	30	45	40	60	25	40	45	35	35	45	50
I/E	I	I	I	I	I	I	I	I	I	I	I	I	I	I
NFKB1 GENE MUTATION	A	A	A	A	A	A	A	A	A	A	A	A	A	A
CAG	NOT DONE	DVD	DVD	TVD	SVD	NOT DONE	NOT DONE	NOT DONE	SVD	NOT DONE	TVD	NOT DONE	NOT DONE	NOT DONE
HEART FAILURE	P	A	A	P	A	A	A	P	P	A	P	P	A	A
PULMONARY EDEMA	P	A	A	A	A	A	A	A	A	A	A	A	A	A
ARRYTHMIAS	A	A	A	A	A	A	A	A	A	A	A	A	A	A
CARDIOGENIC SHOCK	P	A	A	A	A	A	A	A	A	A	A	A	A	A
IN HOSPITAL DEATH	P	A	A	A	A	A	A	A	A	A	A	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	DYSPNEA	PALPITATION	SYNCOPE	ABDOMINAL PAIN	ATYPICAL MANIFESTATION	DIABETES	HYPERTENSION	FAMILY HISTORY	SMOKING	ALCOHOL	TOBACCO CHEWING
50	KAMALA BAI SONAD	63	F	HOME MAKER	9900168867	VIJAPUR	00179	28/08/2024	03/09/2024	6	P	A	A	A	A	A	P	P	A	A	A	A
51	BANUBI MULLA	66	F	HOME MAKER	9380359857	BABALAD	00069	29/08/2024	06/09/2024	8	P	P	A	A	A	A	P	P	A	A	A	P
52	UMAKANT SONAD	73	M	HOME MAKER	8217056242	DEVARA HIPPARAGI	00042	30/08/2024	08/09/2024	9	P	P	A	A	A	A	P	P	A	A	A	A
53	KASTURIBAI NAVI	55	F	HOME MAKER	8296365189	SALOTAGI	2857	12/09/2024	18/09/2024	6	P	P	A	A	A	A	P	P	A	A	A	A
54	SAMBAI MANDARD	73	F	HOME MAKER	8296670315	VIJAPUR	3017	13/09/2024	23/09/2024	10	P	A	A	A	A	A	P	P	A	A	A	P
55	ZANIDANASEEN BURUJWALE	44	F	HOME MAKER	6361725907	ALLAPUR	4212	20/09/2024	27/09/2024	7	A	P	A	A	A	P	P	P	P	A	A	A
56	ALLAMA KADADAGI	60	F	HOME MAKER	6361569127	MOTIGURAJ	4463	22/09/2024	29/09/2024	7	P	A	A	A	A	A	P	P	A	A	A	A
57	GAYATRI KULKARNI	60	F	HOME MAKER	9480259555	NEAR RAYAR MATH	4472	22/09/2024	26/09/2024	6	P	P	A	A	P	P	P	P	P	A	A	A
58	BHIMU KATTIMANI	35	M	FARMER	7709647757	KARAJAGI	4971	24/09/2024	28/09/2024	4	P	A	A	A	A	A	P	P	A	A	P	A
59	REVANNSIDAPPA JOGUR	64	M	FARMER	8618846714	MAHADEV TEMPLE ROAD	3438	25/09/2024	01/10/2024	7	P	A	A	A	A	A	P	P	A	A	A	P
60	SHIVAPPA KALLIMANI	40	M	FARMER	7899948874	VIJAPUR	59934	26/09/2024	28/09/2024	2	P	A	A	A	A	P	P	P	A	P	A	A
61	MALLIKARJUN TEGGIHALLI	46	M	SHOPKEEPER	9880662046	ATHARGA	58504	26/09/2024	30/09/2024	4	P	P	A	A	A	A	P	P	A	P	P	P

SL N.	PR	SYSTOLIC BP(mmhg)	DIASTOLIC BP (MMHG)	TEMPERATURE	RR	TROP I	HEMOGLOBIN G/DL	TOTAL COUNT	ESR	FBS/PPBS/RBS	BLOOD UREA	Sr. Creatine	SERUM SODIUM	Sr.POTASSIUM	TOTAL CHOLESTROL	TRIGLYCERIDES	HDL(mg/dl)	LDL	ECG-RATE	RHYTHM	P WAVE	PR INTERVAL
50	98	120	80	37	18	24918	10	17.4	10mm /hr	rbs-122mg/dl	28	0.9	140	5.1	360	155	20	298	150BPM	Tachycardia	0.04s	0.12s
51	82	130	80	37	18	122.1	10	7.3	10mm /hr	RBS-250mg/dl	20	0.8	135	3.4	180	165	35	110	100BPM	Tachycardia	0.08s	0.12s
52	72	120	80	37	18	0.1	13	8.41	10	FBS-189mg/dl	30	1.2	139	4	200	175	30	112	100BPM	Tachycardia	0.04s	0.12s
53	86	90	60	37	18	1250	12	7.01	20mm /hr	RBS-286mg/dl	27	1.1	130	4.3	150	146	18	98	75bpm	Regular	0.08s	0.20s
54	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
55	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
56	82	100	70	37	18	1221	14	22.2	15mm /hr	rbs-106mg/dl	29	0.7	142	4.2	207	118	33	122	75bpm	Regular	0.08s	0.12s
57	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
58	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
59	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
60	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
61	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

SL N.	QRS CONFIGURATION	QRS DURATION	QRS AXIS	ST-SEGMENT	T wave	QT	QTc	ECG DIAGNOSIS	positive t wave in avr	Fragmented qrs	2D ECHO REGIONAL MOTION WALL ABNORMALITY	LVEF	I/E	NFKB1 GENE MUTATION	CAG	HEART FAILURE	PULMONARY EDEMA	ARRYTHMIAS	CARDIOGENIC SHOCK	IN HOSPITAL DEATH
50	N	0.04s	N	ST D V2-V6	NO T WAVE CHANGES	360s	560s	NSTEMI	A	A	HPOKINESIA OF ANTEROLATERAL WALL	5500	I	A	NOT DONE	A	A	A	A	A
51	DEEP Q WAVES V1-V3	0.04s	N	ST E V1-V3	INVERTED V1-V4	440	560s	STEMI ANTERIOR WALL	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	45	I	A	NOT DONE	A	A	A	A	A
52	deep q wave v1-v5	0.04s	N	ST E V2-V5	INVERTED V1-V5	400	510s	STEMI ANTEROSEPTAL WALL	A	A	HPOKINESIA OF ANTEROLATERAL WALL	35	I	A	DVD	P	A	A	A	A
53	deep q wave v1-V4	0.04s	LAD	ST E V2-V5	INVERTED 2,3,AVF,V2-V6	400s	440s	STEMI ANTERIOR WALL	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	30	I	A	TVD	P	P	A	A	A
54	DEEP Q WAVES V1-V3	0.04s	N	ST E V1-V5	ASYMETRIC T WAVE INVERVISON V1-V3,2,3,AVF	360s	400s	STEMI ANTEROSEPTAL WALL	A	A	HYPOKINESIA OF INFERIOR AND LATERAL WALL	40	E	A	TVD	P	P	A	A	A
55	DEEP BROAD Q WAVES V1-V4	0.08S	N	ST CHANGES V1-V4	INVERTED 2,3,AVF	400	510s	STEMI ANTEROSEPTAL WALL	A	A	HYPOKINESIA OF SEPTUM	40	I	A	NOT DONE	P	A	A	A	A
56	DEEP BROAD Q WAVES V1-V4	0.04s	N	STEMI V1-V6	INVERTED V1-V6,2,3,AVF	480	530	STEMI ANTEROSEPTAL WALL	A	A	HPOKINESIA OF ANTEROLATERAL WALL	45	I	A	NOT DONE	A	A	A	A	A
57	N	0.04s	N	ST DEP V4-V6,2,3,AVF	INVERTED V4-V6,2,3,AVF	400s	510s	NSTEMI	A	A	INFERIOR WALL HYPOKINESIA	60	I	A	NOT DONE	A	A	A	A	A
58	DEEP Q WAVES V1-V4	0.04s	N	ST E V2-V6	INVERTED V2-V6,2,3,AVF	400s	440s	STEMI ANTEROSEPTAL WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	30	I	A	SVD	P	A	A	A	A
59	DEEP BROAD Q WAVES V1-V4	0.04s	LAD	ST E V1-V4	INVERTED V2-V6	480s	480s	STEMI ANTEROLATERAL WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	35	I	A	TVD	P	A	A	A	A
60	N	0.04s	N	ST E V1-V6	T WAVE CHANGES NOTED	520s	580s	NSTEMI	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	40	I	A	SVD	P	A	A	A	A
61	POOR R WAVE PROGRESSION,DEEP QE V1-V6	0.04s	N	ST E V1-V6	INVERTED V1-V6	520	670s	STEMI ANTEROLATERAL WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	40	I	A	TVD	P	A	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	DYSPNEA	PALPITATION	SYNCOPE	ABDOMINAL PAIN	ATYPICAL MANIFESTATION	DIABETES	HYPERTENSION	FAMILY HISTORY	SMOKING	ALCOHOL	TOBACCO CHEWING
62	LAXMAN HEGGOND	49	M	FARMER	9901495274	JAMKHANDI	5476	27/09/2024	01/10/2024	5	P	A	A	A	A	A	P	A	A	A	A	P
63	MURLIDHAR DEGINAL	69	M	FARMER	9380893381	INDI	5500	27/09/2024	02/10/2024	5	P	A	A	A	A	A	P	A	A	A	A	A
64	BHIMANGOUDA PATIL	87	M	FARMER	6363277251	SAINIK SCHOOL	5524	28/09/2024	07/10/2024	9	P	A	A	A	A	A	P	A	A	P	A	A
65	SUMITA SHAHA	70	F	HOME MAKER	9972504571	CHADCHAN	5539	28/09/2024	05/10/2024	7	A	A	A	A	A	P	P	A	A	A	A	A
66	ASHOK KUMBAR	81	M	SHOPKEEPER	9972691407	GACHINAKATTI	5664	28/09/2024	02/10/2024	4	P	A	A	A	A	A	P	A	A	P	A	A
67	BOURAMMA HUNASHYAL	60	F	HOME MAKER	6361342248	BASAVAN BAGEWADI	6142	01/10/2024	05/10/2024	4	P	P	A	A	A	A	P	A	A	A	A	A
68	BASAPPA KANOLLI	70	M	FARMER	8050972740	AT POST MANNUR TALUK DEVAR HIPPARAGI, BIJAPUR	6413	03/10/2024	07/10/2024	4	P	A	A	A	A	A	P	A	A	A	A	P
69	SIDDAPA GUBBI	60	M	FARMER	9380141409	AT MANAGULI TQ, BAGEWADI, BIJAPUR	6478	03/10/2024	07/10/2024	4	P	P	A	A	P	A	P	A	A	A	P	P
70	TAJUDDIN DEGINAL	45	M	SHOPKEEPER	7022680374	SHAHAPUR AGASI	6832	05/10/2024	11/10/2024	6	P	A	A	P	A	A	P	A	A	P	A	P
71	MAHADEVI PUJARI	65	F	HOME MAKER	6361578255	KOLLURAGI, INDI	7893	12/10/2024	20/10/2024	8	P	A	P	A	A	A	P	A	A	A	A	A
72	BHIMSINGH RATHOD	55	M	FARMER		VIJAPUR	9778	14/10/2024	20/10/2024	6	P	P	A	A	A	A	P	A	A	A	A	A
73	ANIL KAMBLE	53	M	SHOPKEEPER	9945271193	NEHRU NAGAR	8151	14/10/2024	20/10/2024	6	P	P	A	A	A	A	P	A	A	A	A	A
74	RAMU NAMANE	45	M	SERVICE	9449654946	BASAVANA BAGEWADI	8562	16/10/2024	19/10/2024	3	P	A	A	A	A	A	P	A	A	P	A	A

SL N.	PR	SYSTOLIC BP(mmhg)	DIASTOLIC BP (MMHG)	TEMPERATURE	RR	TROP I	HEMOGLOBIN G/DL	TOTAL COUNT	ESR	FBS/PPBS/RBS	BLOOD UREA	Sr. Creatine	SERUM SODIUM	Sr.POTASSIUM	TOTAL CHOLESTROL	TRIGLYCERIDES	HDL(mg/dl)	LDL	ECG-RATE	RHYTHM	P WAVE	PR INTERVAL
62	56	140	80	38	20	2300	14	10.4	15mm/hr	RBS-231mg/dl	17	0.9	139	3.9	386	222	28	268	60BPM	BRADYCARDIA	0.08s	0.12s
63	70	140	80	37	18	368	12	6.25	10mm/hr	RBS-200mg/dl	29	0.8	139	4.4	150	133	24	100	75bpm	Regular	0.04s	0.12s
64	100	170	100	38	24	5590	14	16.7	20mm/hr	RBS-169mg/dl	31	0.8	138	3.9	175	155	30	120	100BPM	Tachycardia	0.08s	0.12s
65	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	100BPM	Tachycardia	0.08s	0.24s
66	90	180	90	37	16	6.9	13	6.19	10mm/hr	RBS-233mg/dl	14	0.6	141	4.5	180	162	35	110	75bpm	Regular	0.08s	0.20s
67	94	140	90	38	24	1301	8	13.3	15mm/hr	RBS-165mg/dl	84	2	137	6.2	251	134	30	160	75bpm	Regular	0.08s	0.20s
68	76	130	90	37	18	23068	13	8.41	10mm/hr	fbs-90mg/dl	23	0.6	135	4.5	262	371	33	215	75bpm	Regular	0.08s	0.12s
69	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	75BPM	Regular	0.08s	0.12s
70	62	90	50	37	22	8583	13	19.8	48MM/HR	RBS-256mg/dl	134	2	139	3.3	152	222	32	101	75bpm	Regular	0.08s	0.12s
71	80	140	80	37	18	1325	14	9.4	10mm/hr	RBS-134mg/dl	15	0.6	137	2.7	198	162	27	138	100BPM	Tachycardia	0.04s	0.12s
72	78	110	70	37	18	1800	15	11.8	10mm/hr	RBS-250mg/dl	32	1.5	129	4.6	198	236	35	130	75bpm	Regular	0.08s	0.12s
73	96	13	80	37	18	122	11	7.3	10mm/hr	RBS-341mg/dl	19	0.8	134	3.8	204	186	26	158	100BPM	Regular	0.08s	0.12s
74	58	150	70	37	18	1524	11	8.2	10mm/hr	rbs-152mg/dl	18	0.6	134	4.2	246	202	38	166	50BPM	Bradycardia	0.08s	0.12s

SL N.	QRS CONFIGURATION	QRS DURATION	QRS AXIS	ST-SEGMENT	T wave	QT	QTc	ECG DIAGNOSIS	positive t wave in avr	Fragmented qrs	2D ECHO REGIONAL MOTION WALL ABNORMALITY	LVEF	I/E	NFKB1 GENE MUTATION	CAG	HEART FAILURE	PULMONARY EDEMA	ARRYTHMIAS	CARDIOGENIC SHOCK	IN HOSPITAL DEATH
62	DEEP Q WAVE 3,AVR	0.04s	N	ST E V2-V6	INVERTED V1-V6,AVL	480	480s	STEMI ANTEROLATERAL WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	60	I	A	DVD	A	A	A	A	A
63	RSR PATTERN IN 3	0.04s	N	ST DEP 2,3,AVF	INVERTED AVR,AVL	480	530s	NSTEMI	A	A	INFERIOR WALL HYPOKINESIA	50	I	A	TVD	A	A	A	A	A
64	POOR R WAVE PROGRESSION DEEP Q WAVE V1-V3	0.04s	LAD	ST DEP V4-V6,	INVERTED V1,V2	480s	620s	NSTEMI	A	A	HYPOKINESIA LATERAL WALL	30	I	A	NOT DONE	P	P	A	A	A
65	DEEP Q WAVE V1-V3	0.04s	N	ST E V4-V6,2,3,AVF	INVERTED V1-V6,2,3,AVF	520s	670s	STEMI INFEROLATERAL WALL	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	45	I	A	NOT DONE	A	A	A	A	A
66	Q WAVE 3	0.04S	N	ST CHANGES V1-V4	NO T WAVE CHANGES	440s	490s	UA	A	A	ANTERIOR WALL MI	60	I	A	TVD	A	A	A	A	A
67	VPCS NOTED V1-V6,2,3	0.04s	N	ST DEP V4-V6,	INVERTED V1-V6	400s	440s	NSTEMI	A	A	NO MOTION WALL ABNORMALITY	60	I	A	NOT DONE	A	A	A	A	A
68	deep q wave v1-v6	0.04S	N	ST ELEVATION 3,AVF,2	INVERTED 2,V4-V6	520s	580s	STEMI INFERIOR WALL	A	A	HPOKINESIA OF ANT ANTEROLATERAL WALL	45	I	A	NOT DONE	A	A	A	A	A
69	Rs complexes in v4-V6	0.04s	N	ST DEP V4V5,V6	INVERTED 2,3,AVF,V4-V6	480s	520s	NSTEMI	A	A	HPOKINESIA OF ANT ANTEROLATERAL WALL	45	I	A	NOT DONE	A	A	A	A	A
70	TALL T WAVES V3-V5	0.04s	N	ST ELEVATION 2,3,AVF,V4-V6	HYPERACUTE YT WAVE V1-V6	400s	440s	STEMI INFEROLATERAL WALL	A	P	GLOBAL HYPOKINESIA	25	I	A	NOT DONE	P	A	A	P	A
71	N	0.04s	N	ST DEP V3-V6	INVERTED V3-V6	400s	510s	NSTEMI	A	A	NO MOTION WALL ABNORMALITY	60	I	A	DVD	A	A	A	A	A
72	DEEP Q WAVES V1-V3	0.04s	N	ST E V1-V3	INVERTED V1-V3	480s	440s	STEMI ANTERIOR WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	35	I	A	SVD	P	A	A	A	A
73	Q WAVE AVR	0.04s	N	ST CHANGES V1-V5	SYMMETRIC T WAVE INVERTED V1-V6	480s	520s	STEMI ANTERIOR WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	55	I	A	SVD	A	A	A	A	A
74	N	0.04s	N	ST E V3-V6	INVERTED DEEP V2-V6	480	430	STEMI ANTEROLATERAL WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	40	I	A	NOT DONE	P	A	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	DYSPNEA	PALPITATION	SYNCOPE	ABDOMINAL PAIN	ATYPICAL MANIFESTATION	DIABETES	HYPERTENSION	FAMILY HISTORY	SMOKING	ALCOHOL	TOBACCO CHEWING	PR	SYSTOLIC BP(mmhg)	DIASTOLIC BP (MMHG)	TEMPERATURE	RR	TROP I	HEMOGLOBIN G/DL	TOTAL COUNT	ESR	FBS/PPBS/RBS	BLOOD UREA	Sr. Creatine	SERUM SODIUM	Sr.POTASSIUM
75	HALIMA MULLA	64	F	HOME MAKER		GANGABAUDI 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
76	GOVINDRAO VAJANTRI	65	M	SERVICE	8105859456	SIR DESHPANDE COLONY STATION ROAD, BIJAPUR	8926	18/10/2024	23/10/2024	5	A	P	A	A	P	P	P	A	A	A	P	P	120	140	90	38	18	337	12	12.7	30mm /hr	RBS-247mg/dl	56	2.3	129	6.5
77	MALLAMA GULED	49	F	HOME MAKER	8217095267	SOLATAGI, INDI	8764	18/10/2024	23/10/2024	5	P	A	A	A	A	A	P	A	A	A	A	P	100	120	80	38	20	2063	9	24.7	10mm /hr	RBS--88mg/dl	33	0.8	131	4.4
78	SHANKARAPPA NAVI	85	M	FARMER	9008809424	BAGALKOTE	9082	19/10/2024	25/10/2024	6	P	A	A	A	A	A	P	A	A	A	A	P	75	130	80	37	18	1175	13	12	25	RBS-300mg/dl	62	1.1	135	4.5
79	TARABAI DASHAVANT	62	F	HOME MAKER		CHADCHAN	9130	20/10/2024	26/10/2024	6	P	A	A	A	A	A	P	A	A	A	A	P	60	160	90	37	18	1221	14	9.8	10mm /hr	RBS-246mg/dl	22	1	132	3.4
80	KASTURIBAI DONUR	70	F	HOME MAKER	7204308177	JAIL DARGA	9129	20/10/2024	25/10/2024	5	P	P	A	A	A	A	P	A	A	A	A	A	114	120	80	38	18	5162	13	6.4	22MM /HR	RBS-176mg/dl	18	0.9	138	3.5
81	SIDDAPPA KUDAGI	61	M	SHOPKEEPE	866023297 9	A/P UPPALADINNI BABLESHWAR	9367	########	########	4	P	A	A	A	A	A	P	A	A	P	P	P	80	150	60	38	16	2400	14	7.7	10mm /hr	rbs-120mg/dl	25	0.8	137	4.4
82	SIDDAPA ILAGI	55	M	FARMER	9008304526	A/P MANANKALGI, INDI	9577	22/10/2024	26/10/2024	4	P	A	A	A	A	A	P	A	A	P	A	A	80	120	70	37	18	5240	12	16.6	25MM /HR	RBS-82mg/dl	25	0.8	138	4
83	YENKAYYA METI	52	F	HOME MAKER		VIJAPUR	8427	22/10/2024	24/10/2024	2	P	A	A	A	A	A	P	A	A	A	A	A	86	120	70	37	16	1352	13	9.4	36MM /HR	RBS-241mg/dl	16	0.8	136	4.1
84	MAHADEVI KORI	60	F	HOME MAKER		RAMAPUR, TIKOTA	16830	05/12/2024	10/12/2024	5	P	A	A	A	A	A	P	A	A	A	A	P	90	100	60	37	18	30000	12	9.52	15mm /hr	RBS-195mg/dl	20	0.8	140	4
85	SHANTAVVA BADIGER	65	F	HOME MAKER	9845632104	WADAWADAGI ,BASAVANA BAGEWADI	17405	09/12/2024	14/12/2024	5	A	A	A	A	A	P	P	A	A	A	A	P	122	140	80	38	18	351	12	15.8	15mm /hr	RBS-199mg/dl	30	0.6	139	4.7
86	GANGAPPA HIMAKAR	47	M	FARMER		POST MALGUR,JAMKHANDI	17403	09/12/2024	16/12/2024	7	P	A	A	A	A	A	P	A	A	P	A	P	92	110	70	37	18	173	17	21.5	10mm /hr	RBS-400mg/dl	33	1.2	133	4.2

SL N.	TOTAL CHOLESTROL	TRIGLYCERIDES	HDL(mg/dl)	LDL	ECG-RATE	RHYTHM	P WAVE	PR INTERVAL	QRS CONFIGURATION	QRS DURATION	QRS AXIS	ST-SEGMENT	T wave	QT	QTc	ECG DIAGNOSIS	positive t wave in avr	Fragmented qrs	2D ECHO REGIONAL MOTION WALL ABNORMALITY	LVEF	I/E	NFKB1 GENE MUTATION	CAG	HEART FAILURE	PULMONARY EDEMA	ARRYTHMIAS	CARDIOGENIC SHOCK	IN HOSPITAL DEATH
75	196	282	34	118	150BPM	Tachycardia	0.08 S	0.12 s	DEEP Q WAVES V3-V4	0.04s	N	ST E V1-V5	INVERTED V3-V5	360	560	STEMI ANTEROSEPTAL WALL	A	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	30	I	A	NOT DONE	P	A	A	A	A
76	254	199	20	198	100BPM	Tachycardia	0.08 S	0.12 s	BROAD ORS WITH NOTCH V1-V6	0.08S	LAD	ST E V1-V4	INVERTED V1-V4	440s	560s	LBBB	A	P	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	30	I	A	TVD	P	P	P	A	A
77	196	186	35	122	75bpm	Regular	0.04 S	0.12 s	DEEP Q WAVES 2,AVF,AVR	0.04s	N	ST E 2,3,AVF,V2-V6	INVERTED 2,3,AVF	440s	490s	STEMI INFEROLATERAL WALL	A	A	HPOKINESIA OF ANTEROLATERAL WALL	35	I	A	NOT DONE	P	A	A	A	A
78	270	250	30	152	75bpm	Regular	0.08S	0.12s	N	0.04s	N	ST DEP V2-V5 NO T WAVE		400s	440s	NSTEMI	A	A	NO MOTION WALL ABNORMALITY	60	I	A	TVD	A	A	A	A	A
79	180	172	28	130	75bpm	Regular	0.08 S	0.12 s	N	0.04s	N	ST E V1-V6	INVERTED DEEP V2-V6	480s	530s	STEMI ANTEROSEPTAL WALL	P	A	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	35	I	A	DVD	P	A	A	A	A
80	260	280	28	198	150BPM	Tachycardia	0.08 S	0.12 s	DEEP Q WAVES V3-V4,AVF	0.04s	LAD	ST E V1-V5	INVERTED V1-V5	400s	630ms	STEMI ANTEROSEPTAL WALL	A	P	HPOKINESIA OF ANTEROLATERAL WALL	30	I	A	TVD	P	A	A	A	A
81	200	192	30	156	60BPM	Regular	0.08 S	0.12 s	N	0.04s	N	ST CHANGES NOTED	NO T WAVE CHANGES	480s	480ms	NSTEMI	A	A	NO MOTION WALL ABNORMALITY	60	I	A	TVD	A	A	A	A	A
82	223	206	22	132	75bpm	Regular	0.08 S	0.12 s	DEEP Q WAVES V1-V3,NOTCHED	0.04s	N	STE V2-V3	INVERTED V1-V4,3,AVR	440s	490s	STEMI ANTERIOR WALL	A	P	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	35	I	A	SVD	P	A	A	A	A
83	198	365	22	142	100BPM	Regular	0.08 S	0.12 s	DEEP Q WAVES V1-V4	0.04s	N	ST E V2-V4	INVERTED V2-V5	480s	620ms	STEMI ANETROSEPTAL	A	P	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	30	I	A	SVD	P	A	A	A	A
84	200	275	25	130	100BPM	Regular	0.08 S	0.16 S	DEEP Q WAVES 2,AVF,V2-V6,NOTCHED V3,V4,2	0.04s	LAD	ST E V1-V6	INVERTED V1-V6	320s	410s	STEMI ANTEROSEPTAL WALL	A	P	GLOBAL HYPOKINESIA	35	I	A	SVD	P	A	A	A	A
85	188	156	30	111	150BPM	Tachycardia	0.08 S	0.12 s	N	0.04s	N	ST DEP V1-V6	INVERTED AND DEEP V3,V5,3	320s	500S	NSTEMI	A	A	GLOBAL HYPOKINESIA	40	I	A	TVD	P	A	A	A	A
86	265	215	34	145	150BPM	Tachycardia	0.08 S	0.12 s	DEEP Q WAVES V2-V6,RBBB PATTERN-V1	0.04s	RAD	ST E V2-V6	INVERTED V2-V6	280s	440s	RBBB	P	A	HPOKINESIA OF ANT ANTEROLATERAL WALL	40	I	A	SVD	P	A	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	DYSPNEA	PALPITATION	SYNCOPE	ABDOMINAL PAIN	ATYPICAL MANIFESTATION	DIABETES	HYPERTENSION	FAMILY HISTORY	SMOKING	ALCOHOL	TOBACCO CHEWING	PR	SYSTOLIC BP(mmhg)	DIASTOLIC BP (MMHG)	TEMPERATURE	RR	TROP I	HEMOGLOBIN G/DL	TOTAL COUNT	ESR	FBS/PPBS/RBS	BLOOD UREA	Sr. Creatine	SERUM SODIUM	Sr.POTASSIUM	TOTAL CHOLESTROL	TRIGLYCERIDES	HDL(mg/dl)	LDL	ECG-RATE	RHYTHM	P WAVE	PR INTERVAL	QRS CONFIGURATION	QRS DURATION	QRS AXIS	ST-SEGMENT	T wave	QT	QTc	ECG DIAGNOSIS	positive t wave in avr	Fragmented qrs	2D ECHO REGIONAL MOTION WALL ABNORMALITY	LVEF	I/E	NFKB1 GENE MUTATION	CAG	HEART FAILURE	PULMONARY EDEMA	ARRYTHMIAS	CARDIOGENIC SHOCK	IN HOSPITAL DEATH
87	NINGAYYA MATAPATI	69	M	FARMER	961022431	SASANUR, TALIKOTI	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	18.9	20mm /hr	RBS-190mg/ dl	19	1	134	2.9	182	168	33	110	150BP M	Tachycardia	0.08 S	0.12 s	DEEP Q WAVES V1-V4	0.04s	N	ST E V1-V4,AVL	DEEP INVERTED,ASYMMETRIC V1-V4	320s	500S	STEMI ANTEROLATERAL WALL	A	A	HPOKINESIA OF ANT ANTEROLATERAL WALL	40	I	A	DVD	P	A	A	A	A
88	HANAMANTH TOPANINAGO L	64	M	FARMER		VIJAPUR	17529	10/12/2024	14/12/2024	4	P	A	A	A	A	A	P	A	A	P	A	A	68	130	80	37	18	150	14	10.3	10mm /hr	RBS-134mg/ dl	22	0.8	135	4	205	193	30	100	75bp m	Regular	0.12 S	0.16 S	NOTCHED Q WAVE IN 3	0.04s	N	ST DEP V2-V5	NO T WAVE CHANGES	440s	490s	NSTEMI	A	P	HPOKINESIA OF ANT ANTEROLATERAL WALL	55	I	A	DVD	A	A	A	A	A
89	GURUPADAPPA KADABAGAONVI	54	M	FARMER	8150027732	HEBBAL, NIDAGUNDI	17631	10/12/2024	15/12/2024	5	P	A	A	A	A	A	P	A	A	P	A	A	90	90	60	37	20	27.14	15	17.2	15mm /hr	RBS-121mg/ dl	20	0.9	137	4.4	274	143	34	182	100BP M	Regular	0.08 S	0.12 s	rSR PATTERN 2,3,AVF,DEEQ WAVE V1-V4	0.08S	RAD	ST E V1-V6	INVERTED V1-V6	320	410	RBBB	A	P	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	25	I	A	SVD	P	A	A	P	A
90	JAIBUNISSA FOUJI	62	F	HOME		GANGABAVA DI	17660	11/12/2024	13/12/2024	3	A	P	A	A	A	A	P	A	A	A	A	P	110	130	90	37	26	351	11	11	35	RBS-159mg/ dl	23	1.2	128	3.5	178	155	30	116	75bp m	Regular	0.04 S	0.12 s	Q WAVE AVL	0.12S	N	ST CHANGES 1,2,AVL	NO T WAVE CHANGES	420	420s	UA	A	P	HYPOKINESIA OF ANTERIOR AND SEPTAL WALL	40	I	A	TVD	P	P	A	A	A
91	RAMESH HIREGAN	50	M	FARMER	7795044467	A/P BABNAGAR, TIKOTA	19038	19/12/2024	25/12/2024	6	P	P	A	A	A	A	P	A	A	A	A	P	70	110	70	37	18	68.1	16	6.5	16	rbs-116mg/ dl	26	1	143	3.9	270	145	38	150	58BP M	Regular	0.04 S	0.16 S	Q WAVES 1,2	0.12S	N	ST CHANGES	BIPHASIC T WAVE V2,V3	360s	350ms	STEMI ANTERIOR WALL	A	A	HYPOKINESIA OF ANTERIOR WALL AND SEPTUM	60	I	A	NOT DONE	A	A	A	A	A
92	F I KARAJAGI	61	M	FARMER	8796577898	TIKOTA	347322	02/02/2024	10/02/2024	08/01/1900	p	P	A	A	A	A	P	A	A	p	p	P	84	140	90	37	18	12.8	##	26	258	47	1.3	127	5.1	248	183	38	36	122	70BP M	Regular	.04S	0.12 S		0.20S	N	st elevation	ST E V2,3,4,5,6	364s	460ms	STEMI ANTERIOR WALL	A	A	HYPOKINESIA OF DISTAL ANTERIOR WALL AND APEX	45	I	A	NOT DONE	A	A	A	A	A