"STUDY OF THYROID HORMONE PROFILE IN PATIENTS WITH ACUTE CORONARY SYNDROME"

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LIST OF ABBREVIATIONS

- ACC: American College of Cardiology
- ACCP: American College of Chest Physicians
- ACS: Acute Coronary Syndrome
- AHA: American Heart Association
- AMI: Acute Myocardial Infarction
- aPTT: Activated Partial Thromboplastin Time
- AV: Atrioventricular
- BNP: B-type Natriuretic Peptide
- CABG: Coronary Artery Bypass Grafting
- CAD: Coronary Artery Disease
- CAG: Coronary Angiography
- CHD: Coronary Heart Disease
- CK-MB: Creatine Kinase-Myocardial Band
- CO: Cardiac Output
- CRP: C-Reactive Protein
- CVA: Cerebrovascular Accident
- DBP: Diastolic Blood Pressure
- DBP: Diastolic Blood Pressure
- DM: Diabetes Mellitus
- ECG: Electrocardiography/Electrocardiogram
- EF: Ejection Fraction
- ESS: Euthyroid Sick Syndrome
- fT3: Free Triiodothyronine
- fT4: Free Thyroxine

- GP: Glycoprotein
- HDL: High-Density Lipoprotein
- HTN: Hypertension
- ICMR: Indian Council of Medical Research
- IHD: Ischemic Heart Disease
- IL-6: Interleukin 6
- INDIAB: India Diabetes
- LDL: Low-Density Lipoprotein
- LMWH: Low Molecular Weight Heparin
- LT3S: Low T3 Syndrome
- LV: Left Ventricle/Left Ventricular
- MACE: Major Adverse Cardiac Events
- MI: Myocardial Infarction
- NSTEMI: Non-ST-segment Elevation Myocardial Infarction
- NTIS: Non-Thyroidal Illness Syndrome
- PCI: Percutaneous Coronary Intervention
- PR: Pulse Rate
- RR: Respiratory Rate
- rT3: Reverse Triiodothyronine
- SBP: Systolic Blood Pressure
- SCAI: Society for Cardiovascular Angiography and Interventions
- SD: Standard Deviation
- SERCA: Sarcoplasmic Reticulum Ca^{2+}-adenosine Triphosphatase
- sIL-6R: Soluble Interleukin-6 Receptor
- STEMI: ST-segment Elevation Myocardial Infarction

- SVR: Systemic Vascular Resistance
- T3: Triiodothyronine
- T4: Thyroxine
- TFT: Thyroid Function Test
- TH: Thyroid Hormone
- TIMI: Thrombolysis In Myocardial Infarction
- TR: Thyroid Hormone Receptor
- TRE: Thyroid Hormone Response Element
- TSH: Thyroid Stimulating Hormone
- UA: Unstable Angina
- VLDL: Very Low-Density Lipoprotein

ABSTRACT

Background: Thyroid hormones play a crucial role in cardiovascular function, yet their relationship with acute coronary syndrome (ACS) remains incompletely understood. This study aimed to evaluate thyroid hormone profiles in patients with ACS and investigate potential associations with clinical presentation, angiographic findings, and cardiac biomarkers.

Methods: A cross-sectional study was conducted on 127 patients diagnosed with ACS. Thyroid function tests, lipid profiles, cardiac biomarkers, and coronary angiography were performed. Patients were categorized based on thyroid status, and associations with various clinical and laboratory parameters were analyzed.

Results: Of the 127 patients, 73.2% were male, and 60.6% were aged 51-70 years. Thyroid dysfunction was present in 34.6% of patients: subclinical hypothyroidism in 15.7%, overt hypothyroidism in 9.4%, and hyperthyroidism in 9.4%. The most common clinical presentations were chest pain (74.8%) and dyspnea (72.4%). Diabetes mellitus with hypertension was the most prevalent comorbidity (39.4%). Single vessel disease was the predominant angiographic finding (44.9%), followed by double vessel disease (32.3%) and triple vessel disease (22%). STEMI was the most common ECG finding (39.4%), followed by unstable angina (28.3%) and NSTEMI (23.6%). No statistically significant associations were found between thyroid status and age ($p=0.32$), ECG findings ($p=0.93$), coronary angiographic findings ($p=0.23$), Troponin T levels ($p=0.17$), ejection fraction ($p=0.75$), or lipid parameters.

Conclusion: A substantial proportion of ACS patients have thyroid dysfunction, particularly hypothyroid conditions. However, in the acute setting, thyroid status does not significantly influence the clinical presentation, angiographic findings, or

laboratory parameters of ACS. These findings suggest that thyroid dysfunction and ACS may coexist without direct pathophysiological interaction in the acute phase. Nevertheless, the high prevalence of thyroid abnormalities in ACS patients warrants routine thyroid function evaluation in this population, as it may influence long-term cardiovascular outcomes.

Keywords: Acute Coronary Syndrome; Thyroid Function Tests; Hypothyroidism; Coronary Angiography; Cardiovascular Disease; Thyroid Hormones; STEMI; NSTEMI; Unstable Angina; Indian Population

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INTRODUCTION

From unstable angina to non-ST-segment elevation myocardial infarction (NSTEMI) to ST-segment elevation myocardial infarction (STEMI), Acute Coronary Syndrome (ACS) encompasses a range of clinical disorders.[1] This life-threatening condition remains a significant global health burden, with approximately 7 million people experiencing ACS annually worldwide.[2] The complex interplay between various endocrine systems and cardiovascular function has emerged as a crucial area of research, with particular attention being directed towards understanding the role of thyroid hormones in ACS.[3]

Through both genetic and non-genomic processes, thyroid hormones are essential for maintaining cardiovascular homeostasis.[4] These hormones affect cardiac contractility, heart rate, cardiac output, and systemic vascular resistance, making them crucial regulators of cardiovascular function.[5] Recent evidence suggests that alterations in thyroid hormone levels may not only influence the risk of developing ACS but might also significantly impact its clinical course and outcomes.[6]

The relationship between thyroid dysfunction and cardiovascular disease has been well-documented, with both hypothyroidism and hyperthyroidism being associated with increased cardiovascular risk.[7] However, the specific patterns of thyroid hormone alterations in the setting of ACS and their prognostic implications remain subjects of ongoing investigation.[8] The concept of "sick euthyroid syndrome" or "non-thyroidal illness syndrome" (NTIS), characterized by changes in thyroid hormone levels without underlying thyroid disease, has been increasingly recognized in acute cardiac events.[9]

Several studies have demonstrated that low T3 syndrome, characterized by decreased serum T3 levels with normal TSH, is common in patients with ACS and may serve as a predictor of poor outcomes.[10] The pathophysiological mechanisms underlying these hormonal changes are complex and likely involve multiple factors, including inflammatory cytokines, altered deiodinase activity, and changes in thyroid hormone transport and metabolism.[11]

Moreover, emerging research suggests that thyroid hormone levels at presentation may have prognostic value in ACS patients.[12] Low T3 levels, in particular, have been associated with increased mortality, larger infarct sizes, and poorer left ventricular function.[13] Understanding these associations could have significant implications for risk stratification and therapeutic decision-making in ACS management.

Despite these observations, there remains considerable debate regarding the optimal approach to thyroid function assessment in ACS patients and the potential therapeutic implications of thyroid hormone alterations.[14] This study aims to comprehensively evaluate thyroid hormone profiles in patients presenting with ACS, examining their relationship with clinical presentations, disease severity, and short-term outcomes.

The findings from this research could potentially contribute to improved risk stratification strategies and help identify patients who might benefit from more aggressive management or closer monitoring. Furthermore, understanding the patterns of thyroid hormone alterations in ACS could provide insights into potential therapeutic targets and guide future research into novel treatment approaches.

AIMS AND OBJECTIVES

Objectives:

1. To study of thyroid hormone profile in patients with Acute coronary syndrome

REVIEW OF LITERATURE

ANATOMY OF HEART

The heart is a muscular, conical, hollow organ that sits behind the sternum in the middle of the chest.[15] It resembles a closed fist in size and weighs between 250 and 300 g.[16]

LAYERS OF HEART WALLS

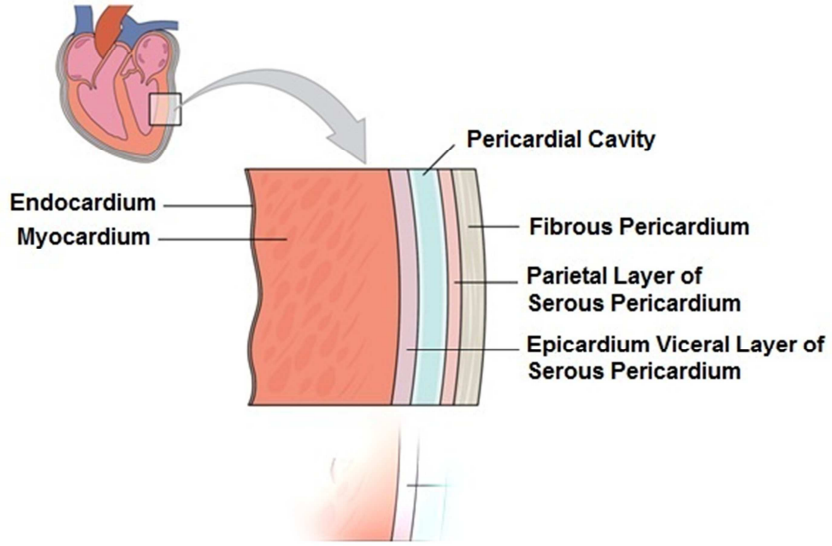
The pericardium encloses three layers that make up the heart wall: [17, 18]

1. Epicardium: the visceral layer of the serous pericardium forms the outside layer of the heart wall.
2. The myocardium, the muscular middle layer of the heart's wall, contains the conducting system and excitable tissue.
3. The endocardium.

   A subendocardial layer and a middle concentric layer.

The subepicardial and subendocardial layers make up the majority of the remaining heart.

Figure 1: Layers of Heart



STRUCTURE AND FUNCTION[15]

"The left and right pumps, which deliver blood to the pulmonary and systemic circulations, are made up of the four chambers of the heart. The right atrium receives deoxygenated blood from the lungs and the rest of the body through the superior and inferior vena cavae. Furthermore, deoxygenated blood from the heart muscle itself can seep into the right atrium through the coronary sinus. Therefore, the right atrium acts as a storage space for blood that has lost oxygen. From here, the tricuspid valve allows blood to reach the right ventricle, which is the main pumping chamber of the right heart."

"Through the right ventricular outflow tract and the pulmonic valve, the right ventricle pumps blood into the pulmonary artery, which then distributes it to the lungs for oxygenation. As the blood passes through the lungs' capillaries, it approaches the oxygen in the alveoli close enough to oxygenate the blood. This oxygenated blood is collected by the four pulmonary veins, two in each lung. All four of these veins open

into the left atrium, which acts as a collection space for oxygenated blood. Like the right atrium, the left atrium pumps blood to its ventricle using both passive flow and vigorous pumping. Consequently, the left ventricle is filled with oxygenated blood that passes via the mitral valve. The main pumping chamber of the left heart, the left ventricle, sends newly oxygenated blood to the systemic circulation via the aortic valve. The cycle is then repeated in the next heartbeat."

The purpose of each of the heart's four valves is to let blood to flow forward while obstructing backward flow.

CARDIAC PUMPING FUNCTION

Heart pumping physiology and cardiac mechanics have been characterised in various ways over the years and remain incompletely understood.[19–21] The LV moves primarily in three ways during systole: rotation, radial reduction of the inner diameter, and longitudinal motion that brings the base closer to the apex.[22] Leonardo da Vinci first characterised the ventricle's longitudinal contraction in the 15th century[23], and Harvey followed suit in the 17th century. Harvey also demonstrated how the ventricles shortened radially during systole.[21] In 1883, Jager presented the idea of dynamic systolic filling of the atria, describing the heart as a pressure suction pump.[24] "Two decades later, Gauer's findings showed that the volume of the heart changes in each cardiac cycle. This was against the 1932 proposal by Hamilton and Rompf which stated that the heart maintains a constant volume all throughout the cardiac cycle as it pumps in long axis."[19,20]

"Modern cardiac physiology textbooks continue to describe and illustrate the widely held assumption that the cardiac pumping and blood flow control is by radial squeezing actions."[25] On the other hand, a squeezing motion pattern would cause the heart's overall volume to alter significantly, which would cause the surrounding

tissues to shift. Since this pumping method uses a lot of energy, it probably doesn't adequately describe how the heart pumps. Recent studies have demonstrated that the heart's outside volume remains rather constant throughout the cardiac cycle, indicating that measurement mistakes in two-dimensional (2D) imaging techniques are the source of earlier data indicating significant variations in outer volume.[26]

"Since the longitudinal motion of the ventricle has been shown to be substantially correlated with LV function, there has been a noticeable surge in interest in this phenomenon during the past two decades."[27] In 1986, Lundbäck proposed that the heart functions similarly to a piston pump, [26] using the spherical AV-plane, also known as the ΔV-piston, a piston-like unit that moves longitudinally back and forth to accomplish the pumping task. This idea states that the exterior volume of the heart remains relatively constant during the heart cycle and that the heart is regulated by inflow. "The spherical AV-plane's diameters vary up towards the atria and down towards the ventricles, causing volumes to be created in the heart as it swings back and forth. Due to pressure gradients created by the pump's input, these volumes (ΔV-volumes) allow the AV-plane to hydraulically return to its upper position during diastole."[26] Energy-efficient, this pumping method preserves muscle dynamics over the cardiac cycle and during crucial moments like AV-plane acceleration, retardation, and direction changes. "Lundbäck also built a mechanical pump called the dynamic displacement pump, or ΔV-pump, which is comparable to both dynamic and displacement pumps and is based on the pumping principles found in the human heart. Studies that were published a few years ago proved the significance of AV-plane displacement as a contributor to the cardiac pumping function."[28]

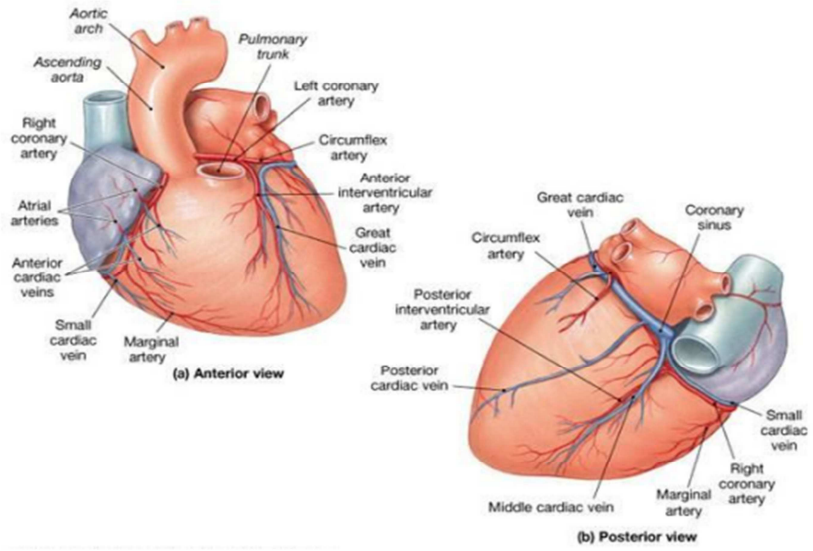
CORONARY CIRCULATION[15]

Both the left major coronary artery and the right coronary artery supply blood to the heart. Eighty percent of the blood to the heart muscle is carried by the left major coronary artery. This short artery splits into two branches: “the circumflex coronary artery, which supplies blood to the lateral and posterior parts of the left ventricle, and the left anterior descending artery, which supplies blood to the anterior two-thirds of the interventricular septum and the adjacent portion of the left ventricular anterior wall.”

The inferior wall of the left ventricle, right atrium, and right ventricle are supplied by the right coronary artery and its branches.

The heart's surface is covered in coronary arteries and veins. “The coronary sinus, which runs in the left posterior atrioventricular groove and opens into the right atrium, is where the majority of coronary veins unite.” Thebesian veins are other tiny veins that open straight into the heart's four chambers.

Figure 2: Coronary Circulation



HISTORICAL VIEW

Coronary thrombosis has been recognised as a cause of death since the early 1800s. Following a number of observations, Obrastov, Strazhesko, and Herrick documented the clinical characteristics of an acute myocardial infarction.[29] James Herrick highlighted the value of rest as the sole treatment strategy for myocardial infarction in 1912, and it was recommended until the early 1950s.[30] also brought about the invention of electrocardiography (ECG), which was developed by Einthoven in 1902 and is currently the primary diagnostic method for acute myocardial infarction.[29] Beta blockers emerged as a potential treatment option to lessen the harm that ischaemia causes.[31]Fletcher and Verstraete were experimentally the first to utilise thrombolytic drugs during the reperfusion era in the 1950s and 1960s.[29] Chazov et al.[32] and Rentrop et al.[33] transformed the field in the 1970s by demonstrating that intracoronary infusion of streptokinase, a thrombolytic drug, could dissolve intracoronary thrombus. According to research by De Wood et al.[34], 90% of patients with alterations in the ST segment had occlusive thrombi in their coronary arteries.

CORONARY ARTERY DISEASES

An insufficient blood supply (circulation) to a limited area as a result of blockage of the blood arteries supplying the area is known as ischaemia. An organ (such as the heart) is said to be ischaemic if it is not receiving enough blood and oxygen. Heart issues brought on by constricted heart (coronary) arteries that feed blood to the heart muscle are referred to as ischaemic heart disease, coronary heart disease (CHD), or coronary artery disease. Atherosclerosis, or plaque accumulation, is the most common cause of the narrowing, though blood clots and blood vessel

constriction can also be the reason. In the US and around the world, it is the leading cause of death.[35, 36]

There are two clinical classes of coronary heart disease patients: [37]

- Stable angina group
- Acute coronary syndrome group (ACS), which comprises non-ST-segment elevation myocardial infarction (NSTEMI), ST-segment elevation myocardial infarction (STEMI), and unstable angina (UA).

ACUTE CORONARY SYNDROMES

The phrase "acute coronary syndrome" best describes a sudden reduction in coronary blood flow that manifests as myocardial ischaemia or infarction.

Epidemiology of ACS

Globally, cardiovascular diseases account for about one-third of all fatalities.[38] Ischaemic heart disease (IHD) is the most common cardiovascular disease.[39] In fact, IHD is recognised as a significant risk to 21st-century sustainable development.[40]

It is anticipated that the rising incidence of IHD will persist as a result of population ageing as well as the rising prevalence of obesity, diabetes, and metabolic syndrome.[41] The ageing of the world's population has increased dramatically over the last 20 years.[42] In fact, according to UN predictions, the proportion of people over 65 will rise from one in eleven in 2019 to one in six by 2050.[43] IHD in the current age is also influenced by psychological strain, emerging social relationship problems, and sleep deprivation of less than six hours per night.[44]

The prevalence of coronary artery disease is high in both industrialised and developing nations. According to current estimates derived from epidemiologic

research conducted across the nation, the prevalence of CHD ranges from 2% to 7% in rural people and from 7% to 13% in urban areas. According to epidemiologic studies, India currently has more than 30 million cases of CHD.[45]

Among Indians living in India, the prevalence of CAD risks is 21.4% for diabetic patients and 11% for non-diabetic individuals.[46] The concerning increase in coronary risk factors such as diabetes, hypertension, atherogenic dyslipidaemia, smoking, central obesity, and physical inactivity might be attributed to the growing incidence of coronary heart disease (CHD) in India. India's burden of cardiovascular risk factors has increased over the past 20 years due to rapid urbanisation and lifestyle changes.[45]

ETIOLOGY OF ACUTE CORONARY SYNDROME

Coronary artery disease is a multifactorial phenomenon.[35]

Etiologic factors can be broadly categorized into[35]

- Gender, age, genetics, and family history are all unchangeable.
- Modifiable factors include lipid levels, smoking, obesity, diabetes, hypertension, and psychosocial issues.
- **Smoking:**

According to the June 2017 Global Adult Tobacco Survey-2 report, the prevalence of tobacco use among Indian adults aged 15 and older has decreased by 6%. Male smoking rates have been steadily declining from 1995–1996 to 2016–2017, while female smoking rates have decreased from 2.9% to 2% within the same time period. “Global average of tobacco use (22%) is lower than males who have a higher

prevalence (23.6%).”The most significant modifiable and reversible risk factor for CVD is tobacco smoking..[47]

- **Diabetes**

One in ten Indians over the age of eighteen has elevated blood glucose. In 2017, India has more than 73 million cases of diabetes, more than any other country in the world. In India, 8.8% of people between the ages of 20 and 70 have diabetes, making it a problem. “The rising prevalence of diabetes and other non-communicable diseases can be attributed to rapid urbanisation, globalisation, sedentary lifestyles, bad diets, overweight and obesity, tobacco use, and longer life expectancies .” By implementing behavioural adjustments that prioritise a healthy, balanced diet and frequent exercise, the burden of diabetes can be considerably decreased.[48]

- **Hypertension**

In India, one in four people over the age of eighteen has high blood pressure. In India, hypertension is responsible for 10.8% of all fatalities. “Over the past thirty years, its frequency is sharply increased in both urban and rural areas. both in urban and rural areas.By 2025, this burden is predicted to have doubled from 118 million in 2000 to 213.5 million. “According to estimates,16% of CAD, 21% of PVD, 24% of AMI, and 29% of strokes are due to hypertension.”[49]

- **Obesity**

Obesity prevalence is startlingly rising, particularly in cities. Between 1975 and 2016, the prevalence of obesity has tripled globally. Thirty to sixty-five percent of adults in metropolitan areas are overweight or obese. Indians living in cities have a higher body mass index (BMI) (about 24–25) than those living in rural areas (around

20). Obesity around the abdomen is more concerning than a higher BMI. Men's waist-to-hip ratio is 0.99 in urban settings compared to 0.95 in rural ones. "Abdominal obesity is more common than overall obesity.[6] Asian adults with BMIs of greater than 21 kg/m2 were at risk of developing Type II diabetes, ischaemic heart disease, stroke, hypertension, osteoarthritis, and malignancies . Compared to the Caucasian population, Asians are more prone to lead sedentary lifestyles and engage in less frequent physical activity."[50]

- **Dyslipidemia**

"Asian Indians have a different pattern of atherogenic dyslipidemia with low HDL, high triglycerides, and a high concentration of tiny dense LDL particles. According to a 2014 study by the Indian Council of Medical Research (ICMR), there was no difference between urban and rural areas, and almost three-fourths (79%) of the general population had at least one abnormal lipid parameter. According to a number of studies, the prevalence of hypercholesterolaemia, hypertriglyceridemia, low HDL-C, and high LDL-C is 13.9%, 29.5%, 72.3%, and 11.8%, respectively using representative samples from all areas and age groups. In nearly 25% of Indians and other South Asians elevated levels (≥50 mg/dl) of Lp(a) is a significant risk factor. Female gender, obesity, sedentary lifestyle, diabetes, dysglycemia, and hypertension are all factors that are significantly linked to dyslipidaemia."[51]

- **Dietary habits and exercise**

Even though about half of Indians are vegetarians, the risk of diabetes and cardiovascular disease are on par with or higher than those of nonvegetarians, similar to the Western population. Indians have irregular eating habits and eat a lot of carbohydrates. The typical Indian diet consists of daily meals that are higher in carbs,

high-fat dairy products, butter, ghee, and cheese. Kerala has the highest rates of CAD in India because to its culture and custom of cooking with coconut oil.[52] In Indian culture, it's standard practice to reuse cooking oil, which raises transfatty acids. Compared to the rest of the world, Indians eat fewer fresh fruits and vegetables. The Indian subcontinent has a unique prevalence of malnutrition, with low birth weights and high rates of malnutrition on the one hand, and a sharp rise in obesity and related morbidities on the other. Higher CAD mortality was also linked to low educational attainment and poor living conditions. Due to a variety of metabolic, social, and cultural maladjustments, CAD is more common among poor people of rich nations and rich people of poor countries. Rapid lifestyle changes brought on by urbanisation and dietary changes that coincide with such economic advancements could be additional explanations. According to a study by the Indian Council of Medical Research–India Diabetes (ICMR–INDIAB), regular physical activity was seen in less than 10% of the population and every second person is physically inactive.[53]

- **Genetic factors**

The high familial frequency of coronary artery disease suggests a potential hereditary component. Several studies indicate that certain "candidate genes" may exist and are linked to the pathways that cause coronary heart disease. Numerous genes that are predisposed to CAD have been identified by studies. These results, however, are not entirely consistent. 109 loci have been linked to coronary artery disease (CAD) and can explain the role of genetic variables, according to the Coronary Artery Disease Genome-wide Replication and Meta-analysis [54] study and other genome-wide association studies. According to certain theories, the risk is increased when genes interact with environmental variables like smoking, and the combined effect may be more than the total of the effects of either factor alone. Since

CAD is complex and regulated by several genes, it will be challenging to identify a single genetic locus that is at fault.[53]

"PATHOPHYSIOLOGY OF ACS"

- **"Initiation of Atherosclerosis: Role of the Endothelium"**

" Atherosclerosis which is a continuous process of plaque building mostly involves the intima of large and medium-sized arteries. It develops steadily over the course of a person's lifetime and eventually shows up as an acute ischemic event. This process is influenced by a number of coronary risk factors, such as smoking, diabetes, high blood pressure, and high cholesterol.[55] The atherosclerotic process is largely initiated by endothelial dysfunction, which is caused by these risk factors damaging the blood vessel's endothelium. Dysfunctional endothelium is characterized by diminished nitric oxide availability and excessive endothelin-1 production, leading to impaired vascular homeostasis. Additionally, increased expression of adhesion molecules, including selectins, vascular cell adhesion molecules, and intercellular adhesion molecules, along with heightened blood thrombogenicity due to the release of various locally active substances, further contribute to endothelial dysfunction."[56]

- **"Progression of Atherosclerotic Plaque: Role of Inflammation"**

"After the endothelium is injured, inflammatory cells—monocytes in particular—migrate into the subendothelium by attaching themselves to endothelial adhesion molecules. Once there, they differentiate into macrophages. Fatty streaks are created when macrophages break down oxidised low-density lipoprotein (LDL) that has also gotten through the artery wall and turned into foam cells. The cytokines and chemoattractants released by the activated macrophages (such as interleukins, tumour necrosis factor α, and monocyte chemoattractant protein 1) continue the process by

attracting more macrophages and vascular smooth muscle cells, which produce extracellular matrix components, to the plaque site. Additionally, macrophages produce matrix metalloproteinases, which are enzymes that break down the extracellular matrix and cause plaque to rupture.[57] Plaque vulnerability and rupture propensity are significantly influenced by the ratio of smooth muscle cells to macrophages. Plaque rupture is clinically asymptomatic in 99 percent of instances, however it can cause ACS.[58] Rate of Progression of Atherosclerotic lesions is variable, nonlinear, and unpredictable."[59]

- **"Plaque Stability and Susceptibility to Rupture"**

The stability of atherosclerotic plaque varies. High-risk or vulnerable plaques are characterized by extensive lipid cores, thin fibrous caps, a high concentration of T lymphocytes and macrophages, and a relative deficiency of smooth muscle cells. Additionally, these plaques exhibit increased local expression of matrix metalloproteinases, which degrade collagen, eccentric outward remodeling, enhanced plaque neovascularization, and a higher likelihood of intraplaque hemorrhage, all of which contribute to their instability and risk of rupture.[64] Even within the same individual, the makeup of human atherosclerotic plaques varies remarkably.[65] An increase in macrophage activity at the plaque site is linked to inflammation, which is a particularly significant factor in determining the "vulnerability" of plaques[60]. This increased activity causes the lipid core to enlarge and the plaque cap to thin, two properties that make the plaque more susceptible to rupture. The number of plaque ruptures[66] has been observed to positively correlate with elevated levels of C-reactive protein (CRP), which may indicate the activity of these macrophages.[67]

❖ **Plaque Disruption, Thrombosis, and ACS**

The endothelium, inflammatory cells, and blood thrombogenicity all interact intricately in the pathophysiology of ACS.[68] According to angiography, noncritical coronary lesions (less than 50% stenosis in the vessel's width) may be linked to sudden progression to severe or complete occlusion and may ultimately be responsible for up to two-thirds of ACS cases.[69] "Several factors influence the extent of thrombus formation and determine whether a specific plaque rupture will lead to acute coronary syndrome (ACS). These include the balance between antithrombotic and prothrombotic mechanisms in the patient, the severity of the plaque rupture, the level of inflammation at the rupture site, local blood flow dynamics, and the plaque's lipid and tissue factor content."[70] Research employing intravascular ultrasonography has demonstrated that numerous plaque ruptures apart from the culprit lesion are present in at least 80% of patients with ACS.[71]

About 75% of fatal MIs are caused by plaque rupture, according to autopsy investigations, while the remaining 25% are caused by superficial endothelium degradation.[72] "When a plaque rupture or endothelial erosion occurs, the subendothelial matrix—rich in tissue factor, a potent procoagulant—becomes exposed to the bloodstream. This exposure triggers platelet adhesion, followed by platelet activation and aggregation, ultimately leading to thrombus formation. Two types of thrombi can develop: a fibrin-rich thrombus (red clot), which forms due to activation of the coagulation cascade and reduced arterial flow, or a platelet-rich thrombus (white clot), which arises in areas of high shear stress and causes only partial arterial occlusion." Total blockage results from the common occurrence of red clots overlaid on white clots. Thrombosis has a key part in the pathophysiology of AC, according to multiple lines of evidence.[73]

Figure 3: Pathophysiology of ACS

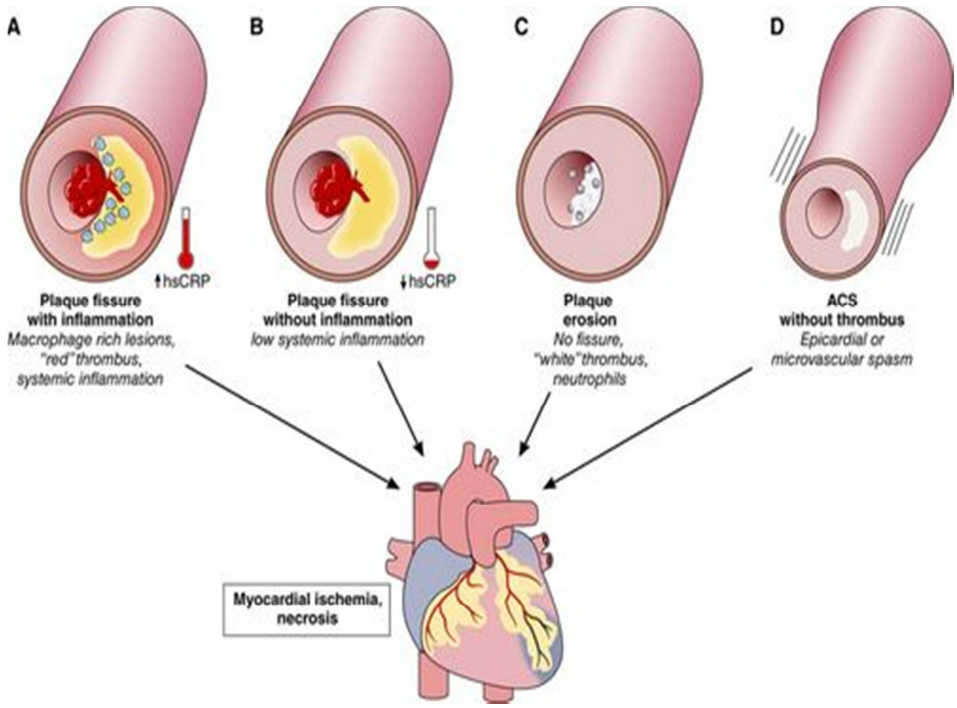
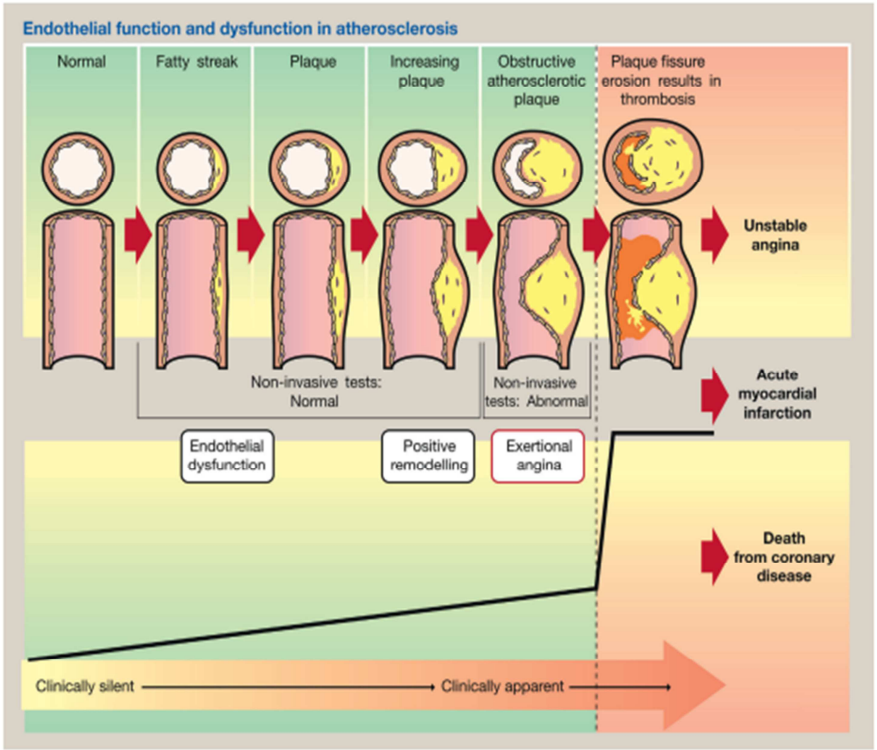


Figure 4: Pathophysiology of ACS



THERAPEUTIC GOALS AND APPROACHES FOR ACS

The clinical severity of ACS is comparable to the severity of coronary angiography and angioscopy results. Patients with STEMI develop red clots, but those with UA/NSTEMI only have white clots. Different treatment objectives and strategies are required for UA/NSTEMI and STEMI because to their distinct underlying pathophysiologies. Revascularisation is commonly used to improve blood flow and avoid reocclusion or repeated ischaemia, while antithrombotic therapy in UA/NSTEMI aims to stop more thrombosis and allow endogenous fibrinolysis to break up the thrombus and lessen the severity of coronary stenosis. On the other hand, in STEMI, the infarct-related artery is typically completely blocked, and the first line of treatment is either immediate pharmacological reperfusion or catheter-based reperfusion, which aims to restore normal coronary blood flow. In every instance, additional treatments including lipid-lowering and anti-ischemic medications are utilised to stabilise plaques over time.[74]

- **Early assessment**[74]

Since the symptoms of STEMI and UA/NSTEMI are similar, a 12-lead electrocardiogram (ECG) and medical evaluation are necessary to distinguish between the two. According to the American Heart Association (AHA) and American College of Cardiology (ACC) 2023 guidelines for managing UA/NSTEMI, patients who exhibit symptoms suggestive of ACS should be directed to call 9-1-1 and directed to a facility capable of 12-lead ECG recording, biomarker determination, and physician evaluation (such as an emergency department [ED]). If a patient has previously been prescribed nitroglycerin, they should be told to take one dose sublingually as soon as possible if they experience any chest pain or discomfort. The patient should dial 9-1-1 right once if there is no improvement in symptoms or if they get worse five minutes

after taking one dose of nitroglycerin. Health care providers should focus on patients who are at higher risk of ACS, such as those who have diabetes, peripheral vascular disease, cerebral vascular disease, known coronary artery disease (CAD), or a 10-year Framingham risk of CAD of 20% or higher. They should also teach patients how to recognise the symptoms of ACS and how to call 9-1-1 right away if they appear.

All patients should be treated as high-priority triage cases if they arrive at the emergency department with chest pain or other symptoms that could indicate ACS. For chest discomfort, evaluation and treatment should adhere to a set, institution-specific strategy. An urgent cardiology consultation is recommended if the emergency department physician is unsure of the initial diagnosis and treatment plan. About 20% to 25% of the 6 to 7 million people who visit emergency departments (EDs) in the US each year with chest pain or other symptoms that could indicate ACS are ultimately diagnosed with UA or MI. The image displays the differential diagnosis for people experiencing chest pain.

Figure 5: Differential Diagnosis of Chest Pain

- Nonischemic cardiovascular
  - Aortic dissection[a]
  - Myocarditis
  - Pericarditis
  - Hypertrophic cardiomyopathy
  - Stress cardiomyopathy
- Chest wall/musculoskeletal
  - Cervical disk disease
  - Costochondritis
  - Herpes zoster
  - Neuropathic pain
  - Rib fracture
- Pulmonary
  - Pneumonia
  - Pulmonary embolus[a]
  - Tension pneumothorax[a]
  - Pleurisy
- Gastrointestinal
  - Cholecystitis
  - Peptic ulcer disease
    - Nonperforating
    - Perforating[a]
  - Gastroesophageal reflux disease
  - Esophageal spasm
  - Boerhaave syndrome (esophageal rupture with mediastinitis)[a]
  - Pancreatitis
- Psychiatric
  - Depression
  - Anxiety disorder/panic attack
  - Somatization and psychogenic pain disorder

[a] Potentially life-threatening conditions

The 2023 ACC/AHA guidelines for managing UA/NSTEMI state that [74]

- The initial evaluation of patients presenting with chest discomfort or symptoms suggestive of ACS should begin with prompt risk stratification using standardized tools like HEART, TIMI, or GRACE scores, which integrate clinical presentations, ECG findings, and cardiac biomarkers.
- High-sensitivity cardiac troponin assays are recommended as the preferred biomarker strategy, with measurements at presentation and 1-3 hours later to facilitate earlier diagnosis and risk stratification.
- Patients should be categorized into immediate high-risk (requiring urgent intervention), intermediate-risk (requiring early invasive strategy within 24 hours), and lower-risk groups based on comprehensive assessment including hemodynamic stability, electrical instability, GRACE risk score, and presence of high-risk features.
- The guidelines emphasize a more individualized approach integrating both ischemic and bleeding risk assessment from the initial patient contact, with treatment pathways tailored accordingly rather than following a rigid two-step process.

- **Clinical presentation**[74]

History and physical

Both evaluating the possibility that the presenting illness is ACS and estimating the probability of a negative outcome depend on a thorough and targeted history taking and physical examination. The discomfort associated with UA is more severe, occurs at rest, and is typically described as frank pain, whereas patients with stable angina describe it as deep, poorly localised chest or arm discomfort that is

exacerbated by activity or emotional stress and relieved by rest, nitroglycerin, or both. The pain or pressure usually radiates to the neck, jaw, left shoulder, and left arm, and is usually found in the substernal region (or occasionally the epigastric area). Other than chest pain, some individuals may have "anginal equivalent" symptoms, such as the most prevalent one, dyspnoea, nausea and vomiting, diaphoresis, and unexplained exhaustion. Older adults and women are more likely to have atypical presentations. Syncope is an uncommon ACS presenting sign. Generally speaking, pain that is localised at the tip of one finger, palpable with movement or palpation, or that is acute, stabbing, or pleuritic is not ischaemic. In the ED, chest pain that goes away once sublingual nitroglycerin is given is not indicative of ACS. The type of the anginal symptoms, a history of CAD, male sex, advanced age, and the presence of traditional risk factors are the five most significant history-related characteristics that aid in the identification of ischaemia owing to CAD, in order of significance. Although their presence is associated with poor outcomes for patients with established ACS, traditional cardiac risk factors (such as hypertension, hypercholesterolaemia, diabetes, cigarette smoking, and a family history of premature CAD) have actually been found to be weak predictors of the likelihood of acute ischaemia.

Finding any precipitating factors of myocardial ischaemia and evaluating the haemodynamic effects of the acute ischaemic episode are the main objectives of the physical examination. Diaphoresis, pale, chilly skin, sinus tachycardia, a third or fourth heart sound, basilar rales, and hypotension are physical examination findings that suggest a wide area of ischaemia and high risk. Additionally, the physical examination could offer hints that aid in making a differential diagnosis. For instance, a pericardial friction rub denotes acute pericarditis, whereas uneven pulses or a murmur of aortic regurgitation may indicate aortic dissection.

Electrocardiography[74]

According to the ACC/AHA guidelines, a skilled emergency physician should examine the 12-lead ECG readings as soon as possible after a patient arrives in the emergency department (ED) with chest pain or other symptoms that could indicate ACS. The ECG is useful for both risk stratification and confirming a clinical diagnosis of ACS. However, there are a number of drawbacks to electrocardiography. The left ventricle's apical, lateral, and posterior walls, for instance, are not sufficiently represented. Furthermore, the likelihood of ACS is not ruled out by normal findings.

Depending on the severity of the clinical presentation, 30% to 50% of patients have ST-segment depression, temporary ST-segment elevation, T-wave inversion, or some combination of these ECG findings, which are linked to UA. A significant and precise indicator of ischaemia and prognosis is the new ST-segment deviation, even if it is only 0.05 mV. Unless it is evident (≥0.3 mV), T-wave inversion is less specific than it is sensitive for ischaemia. Serial assessments of cardiac biomarkers reveal that 90% of patients have acute MI if there is an ST-segment elevation of 0.1 mV or greater in at least two contiguous leads. Comparing recent and earlier ECG results is crucial because research indicates that individuals who do not have any changes in their ECG are less likely to experience issues than those who do.

ACC/AHA guidelines suggest that patients hospitalised for UA/NSTEMI have continuous ST-segment monitoring or serial ECG tracings since the process of myocardial ischaemia is highly dynamic and a single 12-lead ECG only offers a snapshot view of this process.

Figure 6: Acute Lateral Left Ventricular Infarction (tracing obtained within few hours of onset of ilness)

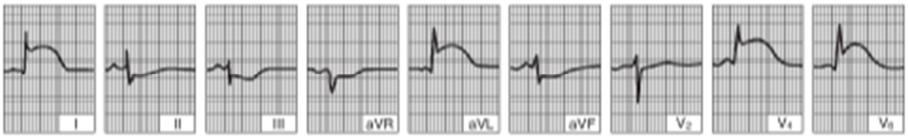


Figure 7: Lateral Left Ventricular Infarction (after the 1st 24 hours)

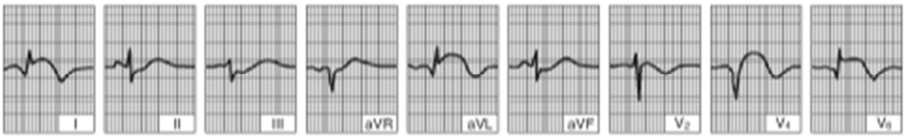
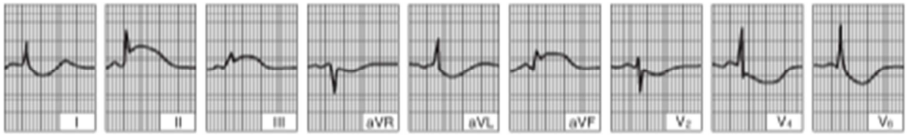


Figure 8: Acute Inferior Left Ventricular Infarction (tracing obtained within a few hours of onset of ilness)

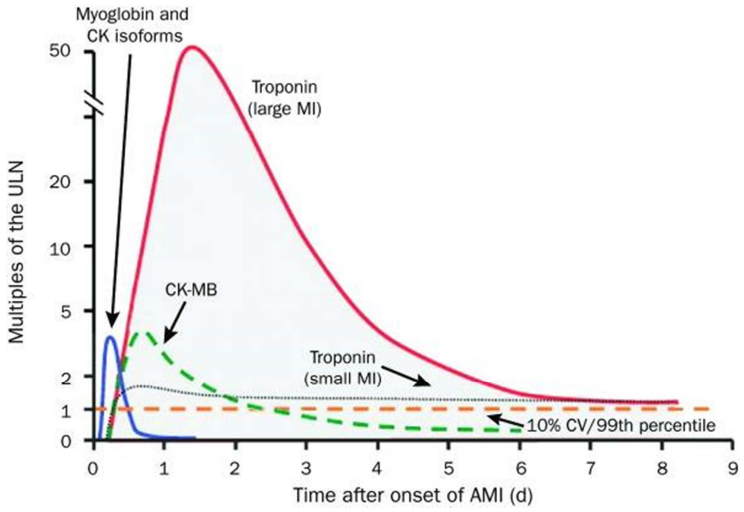


Cardiac Biomarkers of Necrosis[74]

If a patient has chest pain or other symptoms that point to ACS, cardiac biomarkers should be examined. In the setting of ischaemic symptoms, measurements of the cardiac-specific troponins T and I enable the very sensitive, specific, and precise assessment of myocardial injury; these troponins have supplanted CK-MB as the primary marker for the identification of myocardial necrosis. Troponin measurements do have certain limitations, though. If the assay yields a negative result within this time frame, it should be repeated 8 to 12 hours after the beginning of symptoms because troponin levels typically do not rise until at least 6 hours following the onset of symptoms. Troponin levels have little utility in identifying recurrent myocardial injury because they stay increased for a long time (5 to 14 days) following

myocardial necrosis. Nonetheless, when a patient shows in for evaluation a few days after the onset of symptoms, they can be useful in identifying cardiac damage. The levels of CK-MB are helpful in the diagnosis of periprocedural MI and infarct extension (reinfarction) due to its shorter half-life. Although point-of-care assays for bedside biomarker identification are being developed to reduce delays and expedite treatment decisions, their application is currently restricted.

Figure 9: When different biomarkers are released during an acute myocardial infarction (AMI). Plotting the biomarkers across time reveals the multiples of the AMI cutoff.

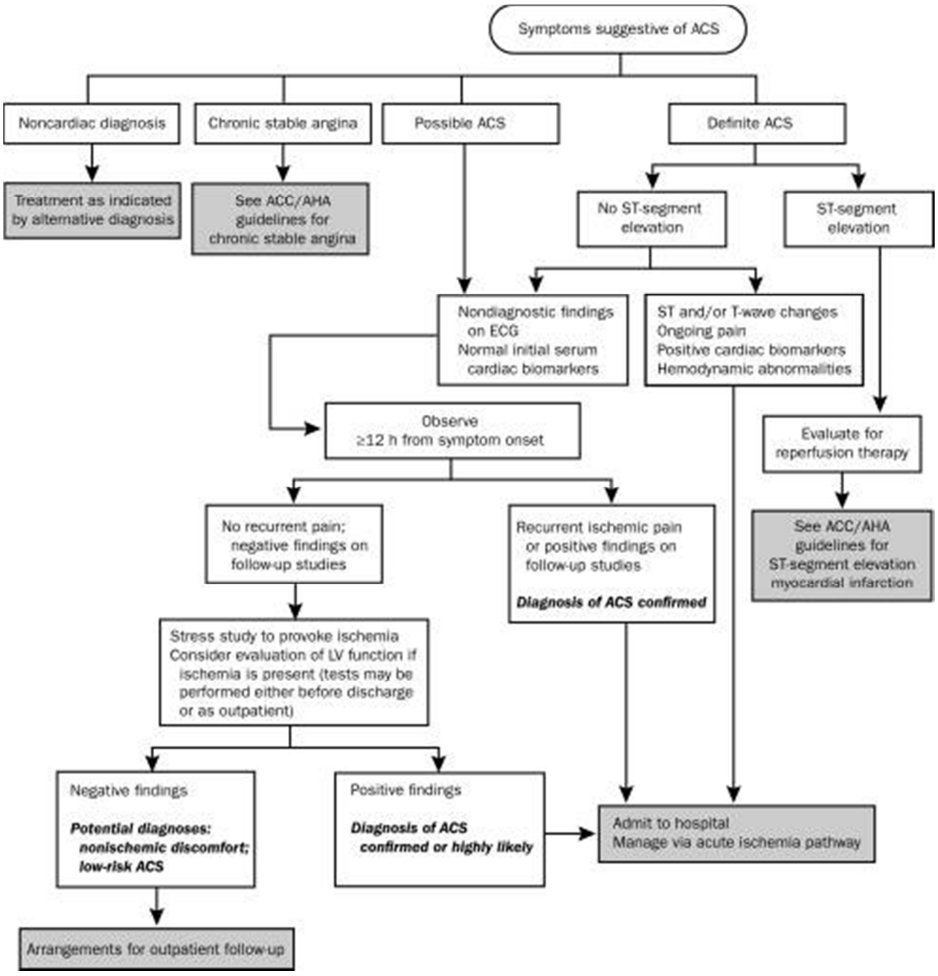


Other Laboratory Tests

In order to screen for pulmonary congestion, which suggests a poor prognosis, and assess the patient for alternative reasons of chest pain, a chest radiograph is typically taken at the time of admission. According to the 2007 ACC/AHA guidelines and the National Cholesterol Education Program Adult Treatment Panel III, a complete lipid profile should be acquired within 24 hours after the beginning of ACS. Some patients should be tested for secondary causes of ACS. For instance, if a patient

has prolonged tachycardia and ACS symptoms, thyroid function should be assessed. Other circulating indicators of elevated risk may also be measured.[74]

Figure 10: Algorithm for diagnosing and treating individuals who may have acute coronary syndrome (ACS). ECG stands for electrocardiography, LV for left ventricular, ACC for American College of Cardiology, and AHA for American Heart Association.



Risk stratification[74]

According to the ACC/AHA guidelines, risk stratification is a crucial step before making any decisions. The 30-day death rate for patients with ACS varies from 1.7% for those with UA to 7.4% for those with NSTEMI to 11.1% for those with STEMI, according to data from a global registry. These outcomes include the whole

risk spectrum. Prognostic estimation, medication selection (e.g., glycoprotein [GP] IIb/IIIa inhibitors, early invasive approach), and care site selection (coronary care unit or monitored step-down unit) are all aided by early risk classification.

High-Risk Clinical Subgroups[74]

"Elderly age, diabetes (diabetic patients with UA/NSTEMI have a roughly 50% higher risk of adverse outcomes than nondiabetic patients), extracardiac vascular disease, congestive heart failure (CHF; Killip class II or higher), and presentation of ACS despite long-term aspirin therapy are all clinical characteristics linked to a significant increase in adverse outcomes for patients with ACS."

Electrocardiography[74]

Both the short-term and long-term prognoses can be accurately predicted by the admission ECG. An ST deviation of as little as 0.05 mV increased the incidence of mortality or MI by around two times at 30 days and 1 year in the Thrombolysis in Myocardial Infarction (TIMI) III Registry of patients with UA/NSTEMI. According to a different study, 4-year mortality rates were linked to ST depression of 0.05 mV or higher on the admission ECG; the probability of dying rose as ST depression rose. On the other hand, a T-wave inversion of 0.1 mV or greater was linked to either no increase in the later risk of MI or death, or only a slight increase. For individuals with STEMI, the number of leads exhibiting ST elevation has proven to be a helpful risk indicator.

Troponins and Other Markers[74]

Troponin is an effective tool for risk assessment in a variety of patients who exhibit acute myocardial infarction symptoms. Troponin levels that are even slightly

elevated indicate a poor prognosis and allow for the identification of high-risk individuals who will benefit from particular treatments, such as GP IIb/IIIa inhibitors, an early invasive approach, or both. Furthermore, there is a quantifiable correlation between the risk of death and the degree of troponin level elevation.

The importance of inflammatory pathways in the pathophysiology of atherosclerosis and its consequences has come to light more and more in the last ten years. "The potential use of plasma indicators of inflammation as risk factors for ACS patients has recently drawn interest; CRP is the most well-studied of these markers. A higher risk of death is associated with elevated CRP levels found by a high-sensitivity CRP test. Among patients with normal troponin levels, the total 14-day death rate was just 1.5%; C-reactive protein levels enabled the distinction between high-risk and low-risk groups. The death rate was only 0.4% when these patients had a normal CRP level, but it rose to 5.8% when their CRP level was raised. Notably, the cutoff value for the CRP level in the stable CAD condition (>3 mg/L) is almost five times lower than that in the ACS scenario (>15 mg/L; multiply by 9.524 to translate to nmol/L). Another straightforward indicator of inflammation is the white blood cell count; in patients with UA/NSTEMI, an elevated count was linked to increased mortality and recurrent MI. Myeloperoxidase was an independent predictor of death or recurrent MI at six months, according to one study that included 1090 ACS patients."

"In all ACS patient types, B-type natriuretic peptide (BNP) offers strong predictive data. The risk of both short-term and long-term mortality rose proportionately with increasing levels of N-terminal proBNP, according to the GUSTO-IV (Global Utilisation of Strategies to Open Occluded Arteries IV) trial, which included 6809 patients with UA/NSTEMI." According to the OPUS-TIMI 16 (Orbofiban in Patients with Unstable Coronary Syndromes—Thrombolysis In

Myocardial Infarction 16) trial, patients with ACS who had elevated BNP levels (>80 pg/mL) (multiply by 1.0 to convert to ng/L) had a 2- to 3-fold increased risk of dying at 10 months compared to those with normal levels. For individuals with STEMI, elevated BNP levels were linked to an increased short-term risk of death. It has been discovered that the size of the myocardial infarct increases proportionately with the highest level of BNP.

It has been suggested that a multimarker strategy utilising many biomarkers can improve risk assessment and improve patient outcomes. Each measure was an independent predictor of the composite of mortality, MI, or heart failure, according to one study that included troponin I, CRP, and BNP to evaluate risk. Interestingly, as the number of elevated markers rose, the mortality risk almost doubled.

Coronary angiography

The most common combination of coronary angiography and percutaneous coronary intervention (PCI, such as stent implantation or angioplasty) is diagnosis. When an acute myocardial infarction occurs, emergency coronary angiography and PCI are performed as quickly as feasible (primary PCI). This strategy has greatly reduced morbidity and mortality and enhanced long-term results at numerous tertiary facilities. When the interval between pain and PCI is brief (less than three to four hours), the infarction is frequently actually terminated.[74]

Multivariable Risk-Assessment Scores[74]

In order to create a multivariable risk model that offers a thorough evaluation of risk and a precise prognostication strategy for patients with ACS, multiple groups have created an integrated approach that incorporates numerous predictor variables. Age 65 years or older, at least three CAD risk factors, documented CAD at

catheterisation, ST deviation of 0.5 mm or greater, at least two angina episodes in the preceding 24 hours, aspirin use within the previous week, and elevated cardiac markers are the seven independent risk factors that make up the TIMI risk score. Patients may be categorised using this scoring approach along a 10-fold risk gradient that ranged from 4.7% to 40.9% (P<.001). Therefore, the TIMI risk score makes it possible to identify or discover high-risk individuals, who have been demonstrated to benefit more from more recent, powerful treatments such GP IIb/IIIa inhibitors and an early invasive approach. Other risk scores are more useful in predicting death, such as the PURSUIT [Platelet Glycoprotein IIb/IIIa in Unstable Angina: Receptor Suppression Using Integrilin Therapy] risk score and the GRACE [Global Registry of Acute Coronary Events] risk score. For individuals with STEMI, distinct risk scores are used to estimate their chance of dying.

TREATMENT OF ACS

Treatment of unstable angina

- Prehospital care: triage to a suitable medical facility, oxygen, aspirin, and nitrates
- Medication: anticoagulants, antiplatelet medications, antianginal medications, and occasionally other medications
- Angiography to evaluate the architecture of the coronary arteries
- Reperfusion therapy: coronary artery bypass surgery or percutaneous coronary intervention
- Chronic medical care of coronary artery disease and post-discharge rehabilitation

It is necessary to set up a trustworthy IV route, administer oxygen (usually 2 L via nasal cannula), and begin continuous single-lead ECG monitoring. Emergency care professionals can lower the risk of death and complications by performing prehospital interventions such as an electrocardiogram, chewing aspirin (325 mg), and nitrate pain control. The necessity and timing of revascularisation can be ascertained with the aid of early diagnostic information and therapeutic response.

Hospital admission

- Assess the patient's risk and determine when to implement a reperfusion plan.
- Drug therapy using anticoagulants, antiplatelet medications, and other medications based on the reperfusion technique

The diagnosis is confirmed when the patient arrives at the emergency room. The clinical picture determines the timing of revascularisation and medication therapy. Patients with persistent symptoms, hypotension, or persistent arrhythmias are considered clinically unstable and should have immediate angiography and revascularisation. Angiography with revascularisation may be postponed for 24 to 48 hours in patients who are clinically stable.

Drug treatment of unstable angina

Anticoagulants, antiplatelet medications, and antianginals should be administered to all patients if they have chest pain. Drugs for Acute Coronary Syndrome discusses the selection and use of the individual medications utilised, which are contingent upon the reperfusion approach and other circumstances. During admission, additional medications such statins, beta-blockers, and angiotensin-converting enzyme (ACE) inhibitors should be started.

The following should be administered to patients with unstable angina (unless contraindicated):

- Antiplatelet medications: Aspirin, clopidogrel, or both (clopidogrel substitutes include ticagrelor or prisugrel).
- Anticoagulants: beta-blocker; angiotensin-converting enzyme (ACE) inhibitor; heparin (unfractionated or low molecular weight heparin) or bivalirudin; occasionally a glycoprotein IIb/IIIa inhibitor during PCI; antianginal treatment, typically nitroglycerin;

If it is not contraindicated, all patients get 160–325 mg of aspirin (not enteric-coated) at presentation, and 81 mg once daily after that. Absorption is accelerated when the initial dose is chewed before swallowing. Aspirin lowers the risk of both immediate and long-term death. A loading dose of ticagrelor (180 mg orally once), prasugrel (60 mg orally once), or clopidogrel (300 to 600 mg orally once) improves results in patients having PCI, especially when given 24 hours beforehand. Prasugrel and ticagrelor have a quicker onset and might be the better choice for urgent PCI.

Patients with unstable angina are usually treated with either low molecular weight heparin (LMWH), unfractionated heparin, or bivalirudin unless there is a contraindication (such as current bleeding). Because unfractionated heparin needs to be dosed often (every 6 hours) to get the desired activated partial thromboplastin time (aPTT), it is more difficult to employ. The LMWHs have a decreased risk of heparin-induced thrombocytopenia, are administered by a straightforward weight-based dose without the need for dose titration or aPTT monitoring, and have superior bioavailability. Patients who have a history of heparin-induced thrombocytopenia, whether confirmed or suspected, are advised to take bivalirudin.

When doing PCI on high-risk lesions (such as those with a large thrombus burden or no reflow), take into account a glycoprotein IIb/IIIa inhibitor. Since eptifibatide, tirofiban, and abciximab seem to be equally effective, other considerations (such as price, accessibility, and familiarity) should influence the drug selection.[75]

Nitroglycerin and occasionally morphine can be used to relieve chest pain. Compared to morphine, which should be used sparingly (for example, if a patient is in pain even after receiving the maximum amount of nitroglycerin therapy or has a contraindication to nitroglycerin), nitroglycerin is preferable. If necessary, a continuous intravenous infusion is administered after the initial sublingual administration of nitroglycerin. Although morphine is quite effective, it can also depress respiration, diminish cardiac contractility, and function as a strong venous vasodilator. It is administered intravenously in doses of 2 to 4 mg, repeated every 15 minutes as needed. Additionally, there is evidence to support the idea that morphine inhibits some P2Y12 receptor activation. Morphine may raise mortality in patients with acute myocardial infarction, according to a large retrospective trial.[76, 77] The use of morphine may also result in bradycardia and hypotension, however these side effects are typically manageable with quick lower extremity elevation.

Statins, ACE inhibitors, and beta-blockers are the standard treatments for all individuals with unstable angina. Unless contraindicated (for example, by bradycardia, heart block, hypotension, or asthma), beta-blockers are advised, particularly for high-risk individuals. Beta-blockers lessen cardiac effort and oxygen demand by lowering heart rate, arterial pressure, and contractility. By enhancing endothelial function, ACE inhibitors may offer long-term cardioprotection. Angiotensin II receptor blockers can be used in place of ACE inhibitors if they are

poorly tolerated due to cough or rash (but not angioedema or renal impairment). Regardless of cholesterol levels, statins are also conventional treatment and ought to be taken continuously.

Reperfusion therapy in unstable angina

Patients with unstable angina do not benefit from fibrinolytic medications, which may be beneficial for those with STEMI.

Angiography is usually performed at the time of admission, either within 24 to 48 hours if the patient is stable or right away if they are unstable (e.g., with prolonged arrhythmias, hypotension, or continuous symptoms). Angiographic results aid in determining the appropriateness of coronary artery bypass grafting (CABG) or PCI.

Figure 11: "Recommendations for anti ischemic therapy"

Nonpharmacological care
Bed rest for all patients
Continuous electrocardiographic monitoring for patients with ongoing chest pain at rest
Supplemental oxygen for patients with cyanosis or respiratory distress
Finger pulse oximetry or arterial blood gas measurement for patients with hypoxemia determination to confirm adequate arterial oxygen saturation
Pharmacological care
Nitroglycerin, sublingual tablet or spray, followed by intravenous administration for all patients
Morphine sulfate, intravenously for patients who have symptoms that are not immediately relieved with nitroglycerin or who have acute pulmonary congestion, severe agitation, or both
β-Blocker, intravenous, followed by oral administration (if not contraindicated) for patients with ongoing chest pain
Nondihydropyridine CCB as initial therapy in the absence of severe LV dysfunction or other contraindications for patients with continuing or frequently recurring ischemia when β-blockers are contraindicated
ACE inhibitor for patients with hypertension despite treatment with nitroglycerin and a β-blocker and with LV systolic dysfunction or CHF; patients with diabetes

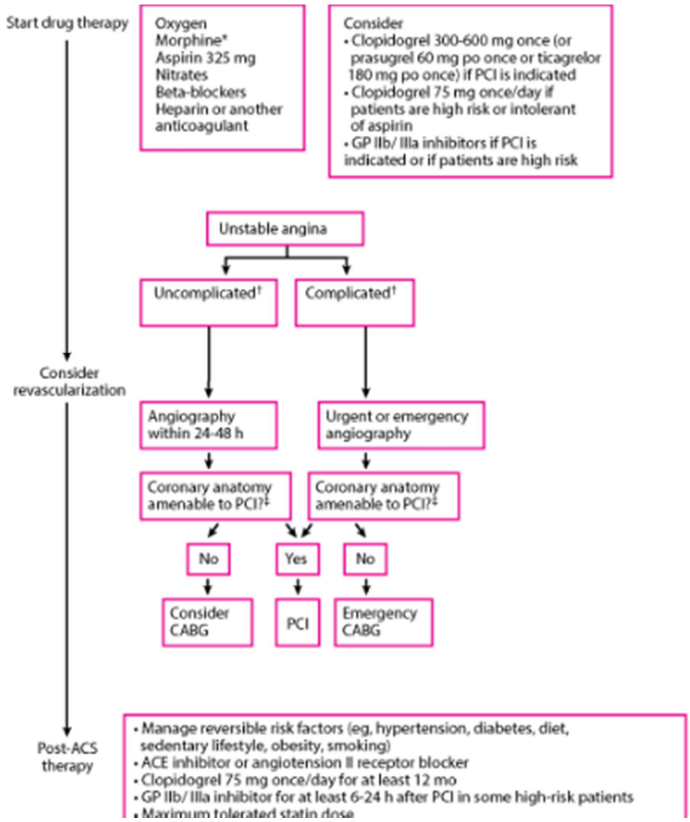
ACE = angiotensin-converting enzyme; CCB = calcium channel blocker; CHF = congestive heart failure; LV = left ventricular.

Figure 12: "Selection of Initial Treatment Strategy: Invasive vs Conservative"

Invasive	Recurrent angina or ischemia at rest or with low-level activities despite intensive medical therapy Elevated cardiac biomarkers (TnT or TnI) New or presumably new ST-segment depression Signs or symptoms of HF or new or worsening mitral regurgitation High-risk findings from noninvasive testing Hemodynamic instability Sustained ventricular tachycardia PCI within 6 mo Prior CABG High risk score (eg, TIMI, GRACE) Reduced left ventricular function (LVEF <40%)
Conservative	Low risk score (eg, TIMI, GRACE) Patient or physician preference in the absence of high-risk features

CABG = coronary artery bypass grafting; GRACE = Global Registry of Acute Coronary Events; HF = heart failure; LVEF = left ventricular ejection fraction; PCI = percutaneous coronary intervention; TIMI = Thrombolysis in Myocardial Infarction; TnI = troponin I; TnT = troponin T.

Figure 13: Approach to Unstable Angina



Treatment of acute MI[78-80]

- Prehospital care: triage to a suitable medical facility, oxygen, aspirin, and nitrates
- Medication: anticoagulants, antiplatelet medications, antianginal medications, and occasionally other medications

Fibrinolytics, angiography, percutaneous coronary intervention, or coronary artery bypass surgery are examples of perfusion treatment.

- Chronic medical care of coronary artery disease and post-discharge rehabilitation

It is necessary to set up a trustworthy IV route, administer oxygen (usually 2 L via nasal cannula), and begin continuous single-lead ECG monitoring. Emergency medical staff prehospital measures, such as electrocardiograms, chewing aspirin (325 mg), and nitrate-assisted pain management, can lower the risk of death and sequelae. The necessity and timing of revascularisation can be ascertained with the aid of early diagnostic information and therapeutic response.

Hospital admission

Assess the patient's risk and select a reperfusion plan.

- Drug therapy using anticoagulants, antiplatelet medications, and other medications based on the reperfusion technique

The diagnosis is confirmed when the patient arrives at the emergency room. The clinical picture and diagnosis determine the drug regimen and the time for revascularisation.

Fibrinolytic treatment or rapid PCI are two possible reperfusion strategies for STEMI. If an NSTEMI patient is clinically stable, angiography can be performed 24 to 48 hours after admission. Angiography must be performed right away if the patient is unstable (for example, persistent symptoms, hypotension, or persistent arrhythmias).

Drug treatment of acute myocardial infarction

Anticoagulants, antiplatelet medications, and antianginal medications should be administered to all patients if they experience chest pain. It is also recommended to administer other medications, such as statins, beta-blockers, and angiotensin-converting enzyme (ACE) inhibitors

If it is not contraindicated, all patients get 160–325 mg of aspirin (not enteric-coated) at presentation, and 81 mg once daily after that. Absorption is accelerated when the initial dose is chewed before swallowing. Aspirin lowers the chance of both short-term and long-term death. A loading dose of ticagrelor (180 mg orally once), prasugrel (60 mg orally once), or clopidogrel (300 to 600 mg orally once) improves results in patients having PCI, especially when given 24 hours beforehand. Prasugrel and ticagrelor have a quicker onset and might be the better choice for urgent PCI.

Unless contraindicated (for example, by current bleeding), patients are usually administered either bivalirudin, unfractionated heparin, or low molecular weight heparin (LMWH). Because unfractionated heparin needs to be dosed often (every 6 hours) to get the desired activated partial thromboplastin time (aPTT), it is more difficult to employ. The LMWHs have a decreased risk of heparin-induced thrombocytopenia, are administered by a straightforward weight-based dose without

the need for dose titration or aPTT monitoring, and have superior bioavailability. Patients having a history of heparin-induced thrombocytopenia, whether confirmed or suspected, are advised to take bivalirudin. Anticoagulants are taken for the following duration in patients having PCI:

- Length of hospital stay (for LMWH patients) or 48 hours (for unfractionated heparin patients) in all other situations

When doing PCI on high-risk lesions (high thrombus burden, no reflow), take into account a glycoprotein IIb/IIIa inhibitor. Since eptifibatide, tirofiban, and abciximab seem to be equally effective, other considerations (such as price, accessibility, and familiarity) should influence the drug selection. This agent is used for six to twenty-four hours.

Nitroglycerin and occasionally morphine can be used to relieve chest pain. Compared to morphine, which should be used sparingly (for example, if a patient is experiencing pain despite nitroglycerin therapy or has a contraindication to nitroglycerin), nitroglycerin is preferable. If necessary, a continuous intravenous infusion is administered after the initial sublingual administration of nitroglycerin. Although morphine is quite effective, it can also depress respiration, impair cardiac contractility, and function as a strong venous vasodilator. It should be administered intravenously at doses of 2 to 4 mg every 15 minutes as needed. Additionally, there is evidence that using morphine may interfere with certain P2Y12 receptor inhibitors. Morphine may raise mortality in patients with acute myocardial infarction, according to a large retrospective trial. It is usually possible to reverse morphine-induced bradycardia and hypotension by quickly elevating the lower extremities.

Statins, ACE inhibitors, and beta-blockers are the standard treatments for all individuals with unstable angina. Unless contraindicated (for example, by bradycardia, heart block, hypotension, or asthma), beta-blockers are advised, particularly for high-risk individuals. Beta-blockers lessen cardiac effort and oxygen demand by lowering heart rate, arterial pressure, and contractility. By enhancing endothelial function, ACE inhibitors may offer long-term cardioprotection. Angiotensin II receptor blockers can be used in place of ACE inhibitors if they are poorly tolerated due to cough or rash (but not angioedema or renal impairment). Regardless of cholesterol levels, statins are also conventional treatment and ought to be taken continuously.

"Reperfusion therapy in acute myocardial infarction"

- Prompt percutaneous coronary intervention or fibrinolytics for patients with STEMI
- For NSTEMI patients, percutaneous coronary intervention should be performed either immediately for unstable patients or within 24 to 48 hours for stable patients.

When emergency PCI is available promptly (door to balloon-inflation time < 90 minutes) and performed by a skilled operator, it is the recommended treatment for ST-segment elevation myocardial infarction in STEMI patients (3). For STEMI patients who fit the criteria, thrombolysis should be performed if there is a significant delay in PCI availability. Fibrinolytic reperfusion works best when administered within the first few minutes to hours following the beginning of myocardial infarction. It is best to start a fibrinolytic as soon as possible. A door-to-needle time of 30 to 60 minutes is the target. Although the medications may be

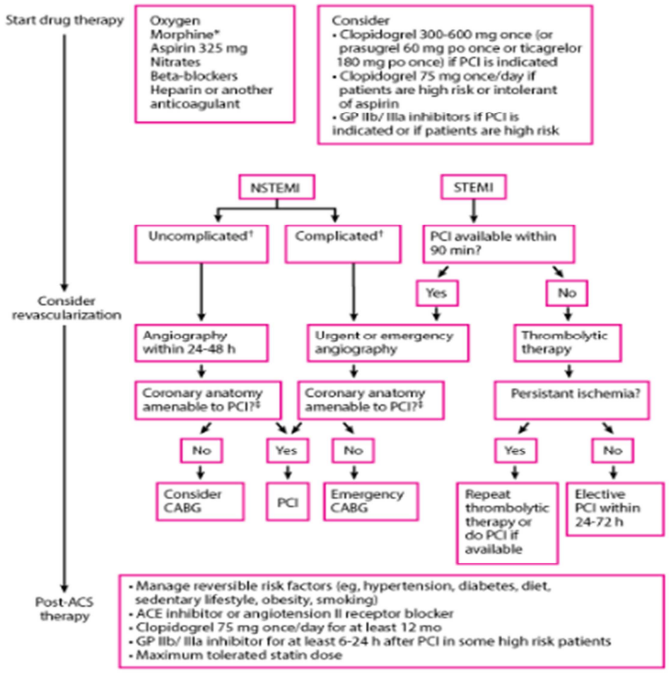
beneficial for up to 12 hours, the greatest benefit happens within 3 hours. The selection and characteristics of fibrinolytic medications are covered elsewhere.

In order to detect coronary lesions that need PCI or coronary artery bypass grafting (CABG), patients with unstable NSTEMI (i.e., those who have persistent symptoms, hypotension, or persistent arrhythmias) should travel straight to the cardiac catheterisation laboratory.

Since a fully blocked infarct-related artery at presentation is rare in uncomplicated NSTEMI patients, rapid reperfusion is not as important. In order to find coronary lesions that need PCI or CABG, these patients usually have angiography performed within the first 24 to 48 hours of being admitted to the hospital.

None of the NSTEMI patients should use fibrinolytics. Potential advantage is outweighed by risk.

Figure 14: "Approach to Myocardial Infarction"



Revascularization For Acute Coronary Syndromes[80]

In patients with acute coronary syndromes, revascularisation is the process of restoring blood flow to the ischaemic myocardium in an attempt to minimise further damage, lessen ventricular irritability, and enhance both immediate and long-term results. Revascularisation techniques include:

- Using fibrinolytic medications for thrombolysis
- PCI (percutaneous coronary intervention), either with or without the implantation of stents
- CABG, or coronary artery bypass grafting

The kind of acute coronary syndrome (ACS), the time of presentation, the location and size of anatomic lesions, and the staff and facility availability all influence the usage, timing, and method of revascularisation.

Non-ST-Segment Elevation Myocardial Infarction and Unstable Angina

Patients with unstable angina who respond to medication therapy or those with uncomplicated non-ST-segment elevation myocardial infarction (NSTEMI), in which a fully occluded infarct-related artery at presentation is rare, do not require immediate reperfusion. In order to find coronary lesions that need PCI or CABG, these patients usually have angiography performed within the first 24 to 48 hours of being admitted to the hospital.

Furthermore, for patients who have a high risk of procedure-related morbidity or mortality, medical care should take precedence over angiography or PCI.

On the other hand, patients who experience ongoing chest pain in spite of the most effective medical treatment or who have complications (such as significantly elevated cardiac markers, cardiogenic shock, acute mitral regurgitation, ventricular septal defect, or unstable arrhythmias) should go straight to the cardiac catheterisation laboratory in order to find coronary lesions that need PCI or CABG.

Although the evidence for this practice is shifting, CABG has generally been chosen over PCI for patients with left main or left main comparable illness, as well as for those with diabetes or left ventricular dysfunction, similar to individuals with stable angina. When PCI fails, is not appropriate (for example, in lengthy or close to bifurcation points lesions), or results in acute coronary artery dissection, CABG must also be taken into consideration.

NSTEMI and unstable angina do not warrant the use of fibrinolytics. Potential advantage is outweighed by risk.

ST segment elevation MI

When emergency PCI is available promptly (door to balloon-inflation time < 90 minutes) and performed by a skilled operator, it is the recommended treatment for ST-segment elevation myocardial infarction (STEMI) (1). Later in the course of STEMI, haemodynamic instability, malignant arrhythmias requiring repeated cardioversion or transvenous pacing, and age greater than 75 are indications for urgent PCI. There is a 20–43% morbidity rate and a 4–12% mortality rate if CABG is required for the lesions.

For STEMI patients who fit the criteria, thrombolysis should be performed if there is a significant chance that PCI will not be available (see table Fibrinolytic Therapy for STEMI). Fibrinolytic reperfusion works best when administered within

the first few minutes to hours following the beginning of myocardial infarction. It is best to start a fibrinolytic as soon as possible. A door-to-needle time of 30 to 60 minutes is the target. Although the medications may be beneficial for up to 12 hours, the greatest benefit happens within 3 hours. Fibrinolytics improve cardiac function and lower hospital mortality by 30 to 50% when used with aspirin. When PCI within 90 minutes is not feasible, prehospital fibrinolytic usage by qualified paramedics can drastically shorten the time to therapy and should be taken into consideration, especially for patients who present within three hours of the beginning of symptoms.

In any case, the majority of patients who get thrombolysis will eventually need to be transferred to a facility that can do PCI in order to have elective angiography and PCI performed as needed before to release. If chest pain or ST-segment elevation continues for more than 60 minutes after starting fibrinolytics, or if pain and ST-segment elevation return, PCI should be considered following fibrinolytics. However, PCI should only be started if the recurrence can be started within 90 minutes. Fibrinolytics can be used again if PCI is not accessible.

THYROID HARMONES AND ACS

"EFFECTS OF THYROID HORMONES ON THE CARDIOVASCULAR SYSTEM"[81]

"Thyroid-stimulating hormone (TSH) triggers the thyroid gland to produce thyroxine (T4) and T3. The thyroid gland secretes over 85% of T4, which is then transformed into T3 by the enzyme 5′-monodeiodinase in the liver, kidneys, and skeletal muscles. The physiologically inactive byproduct of T4 deiodination is called reverse T3 (rT3). Since the majority of circulating THs are attached to transport

proteins, only a small percentage of THs are unbound and physiologically active." "The specific molecular and cellular processes by which the THs function in cardiac cells have been well studied and described. The thyroid hormone nuclear receptors (TRs) drive the stimulation of transcription in the promoter regions of positively regulated genes by binding to thyroid hormone response elements (TREs) as homodimers or, more frequently, as heterodimers. The only TH that enters the myocyte is triiodothyronine. TRs cause the expression of negatively regulated genes, including phospholamban and β-myosin heavy chain, when T3 is not present. However, T3 positively regulates several important structural cardiac genes, including α-myosin heavy chain, sarcoplasmic reticulum Ca2+-adenosine triphosphatase (SERCA 2), and Na+/K+-adenosine triphosphatase. The increased cardiac contractile function (inotropic effect) and diastolic relaxation (lusitropic activity) are caused by the intracellular calcium cycling mechanism, which is controlled by genes expressed following the binding of T3 and TRs. Because T3 can alter the vascular response to the activation of the renin-angiotensin-aldosterone system, it plays a role in the preservation and renewal of endothelial integrity as well as peripheral artery resistance on a vascular level." "Additionally, the nongenomic, extranuclear effects of THs on the systemic vasculature and cardiac myocytes are significant TRE-independent activities. In cardiac and vascular smooth-muscle cells, thyroid hormones, particularly T3, can alter sodium, potassium, and calcium ion channels and impact numerous intracellular pathways. They can therefore instantly raise resting heart rate, venous tone, and left ventricular contractility, increasing cardiac preload. Furthermore, THs boost cardiac output by rapidly relaxing vascular smooth muscle cells, which lowers systematic vascular resistance. In contrast to the T3 effects mediated by nuclear pathways, which take at least 30 minutes to 2 hours to manifest, all of these effects can happen quickly, within seconds to minutes."

Main effects of THs on hemodynamics

Parameter	Effect of THs
SVR	↓
DBP	↓
Afterload	↓
Cardiac inotropy-chronotropy	↑
Basal CO	↑
Blood volume preload	↑

"The substantial consequences of thyroid dysfunction, both overt and subclinical, on the cardiovascular system demonstrate the intimate relationship between thyroid hormone state and cardiovascular disorders. Diastolic hypertension, dyslipidaemia, the development and instability of atherosclerotic plaque, and endothelial dysfunction are all associated with hypothyroidism. Conversely, pulmonary hypertension, elevated systolic blood pressure, and atrioventricular valve regurgitation—particularly of the tricuspid valve—are linked to hyperthyroidism. Rarely, patients with overt hyperthyroidism and thyrotoxicosis may exhibit chest pain and aberrant ECG readings. This could be because of coronary vasospasm or an increase in oxygen needs brought on by increased cardiac contractility and workload. Additionally, hyperthyroidism individuals may exhibit heart failure symptoms, such as increased cardiac output and contractility.Even slight changes in thyroid function can have a substantial impact on heart rate and rhythm. The arrhythmogenic effects of THs include reduced action potential duration, increased automaticity, triggered activity in pulmonary vein cardiomyocytes, and changed electrical properties of atrial myocytes. Patients with overt or mild/subclinical hyperthyroidism frequently have arrhythmias, including sinus tachycardia, atrial flutter, and atrial fibrillation, and to a

lesser extent, ventricular arrhythmias. Conversely, a number of ECG abnormalities and sinus bradyarrhythmias are associated with hypothyroidism. Furthermore, a number of clinical and experimental investigations have indicated that THs may have proarrhythmic and antiarrhythmic effects in addition to a direct impact on myocardial cell electrogenesis."

"THYROID HORMONAL ALTERATIONS DURING ACS"

"High-risk signs of acute myocardial ischaemia and coronary atherosclerosis are known as acute coronary syndromes. The primary pathophysiology of such an event is disruption of a coronary atherosclerotic plaque followed by thrombus development, which sets off an instantaneous inflammatory cascade. Plaque disruption appears to be largely caused by inflammation, which in turn triggers coagulation, thrombosis, sympathetic nervous system activation, and the production of stress hormones.A subset of ACS patients appears to have altered normal thyroid homeostasis. Following an acute coronary event, a decrease in total T3 and/or fT3 concentration and an increase in rT3 concentration have been noted in a number of investigations. It appears that the precise incidence of low T3 syndrome in patients with ACS is still unclear. The literature has shown a wide range of 5% to 35%, which may be explained by variations in the study populations used in different investigations. The syndrome appears to be more common in STEMI patients than NSTEMI patients, though, maybe as a result of the latter's worse early prognosis and the pathophysiologic characteristics of the occlusive thrombus, which put more myocardium at risk.Moreover, it is unclear exactly when THs changes occur following an ACS. Franklyn et al. found that in individuals with simple acute myocardial infarction (AMI), total T3 reached its lowest levels on day 4 and rT3 reached its maximum values on day 2. However, Friberg et al. found that the peak of

rT3 and the nadir of total T3 were both recorded 24 to 36 hours following the onset of symptoms in a sample of 47 AMI patients. The greatest rT3 levels were discovered by Pimentel et al. four days following admission. But only on days 1, 4, and 7 was rT3 measured. Regarding the other THs, Pavlou et al. observed that while minimum T3 and maximum rT3 levels happened on days 3 and 4, mean fT3, T4, fT4, and TSH levels remained constant in all ACS patients over the first five days following admission. It is evident that changes in T3 and rT3 take place during the first five days of ACS, despite the fact that there appear to be substantial variations in the published data on the exact commencement of TH alterations.The pathophysiology of the low T3 syndrome appears to be significantly influenced by the acute impacts of cytokines. In healthy people, interferon-α treatment has been shown to induce TH metabolic abnormalities that mimic the syndrome. Several pro-inflammatory cytokines, including interleukin 6 (IL-6), tumour necrosis factor-α, and interferon-γ, can directly impact the pituitary gland and hinder the secretion of TSH during critical illness. The stimulation of the inflammatory response is thought to be the cause of THs changes in ACS patients. Thyroid axis function may be inhibited by increases in soluble IL-6 receptor (sIL-6R), CRP, and IL-6, a pleiotropic, pro-inflammatory cytokine.Numerous clinical investigations have linked the occurrence of reduced THs levels during an ACS to particular clinical and biochemical markers. Lower T3 levels during the acute coronary event have been associated with known chronic heart failure, prior MI and DM, or worsening angina pectoris prior to the AMI. Although the evidence is weak, older age, a lower body mass index, diabetes mellitus, and elevated plasma levels of CRP and N-terminal pro-brain natriuretic peptide appear to be linked to the development of the low T3 syndrome in ACS patients."

"PROGNOSTIC VALUE OF THYROID HORMONES IN ACS"

"Numerous clinical investigations have looked into the potential prognostic significance of THs changes in ACS patients. Friberg et al. discovered that, regardless of other risk factors, patients with MI who had higher rT3 levels also had higher 1-year mortality rates.[82] The findings of another small-scale investigation by Pimentel et al., which linked aberrant THs changes to a worse prognosis, are consistent with similar findings.[83] The low T3 syndrome has also been linked to higher short- and long-term mortality in two other, bigger trials involving consecutive STEMI patients undergoing primary percutaneous coronary intervention.[84, 85] Additionally, a recent study including patients undergoing cardiac rehabilitation following an ACS found a correlation between decreased fT3 levels and all-cause death.86The literature's varying views of the low T3 syndrome are a reflection of our incomplete knowledge of the condition's pathogenesis. It has been suggested that this temporary low T3 status during an AMI may actually be cardioprotective, lowering energy expenditure, heart rate, and oxygen consumption during the ischaemic stress, even though low TH plasma concentrations have been associated with a poor prognosis. Furthermore, in an effort to lower myocardial oxygen needs, it has been proposed that down-regulating the thyroid hormone system may be helpful in patients with myocardial ischaemia, even before AMI symptoms appear. However, because T3's beneficial effects on the circulatory system are lost, a persistently down-regulated thyroid system following AMI may become maladaptive. As a result, the low T3 syndrome may be a hormonal homeostatic escape response, which is a physiologically adaptive and advantageous mechanism that reduces myocardial metabolic demands and prevents arrhythmias during the early stress phase of an acute ischaemic event. However, it may turn

maladaptive in later stages, with negative long-term effects that predispose to the development of heart failure."[81]

"TREATMENT OF THE LOW T3 SYNDROME"

"Based on experimental data available, TH replacement therapy may enhance haemodynamics, ventricular function, and cardiac remodelling by reversing the aberrant thyroid status that occurs during AMI. Only research including patients having coronary artery bypass surgery or persistent heart failure has clinical experience with TH supplementation during critical illness, though there is some encouraging data. Sadly, there is currently no data to support the use of TH in individuals with ACS who also exhibit low T3 syndrome. Furthermore, it is unknown what kind of synthetic hormone (T4 or T3) it is, how much of it to take, and when to take it. Ongoing randomised clinical trials, however, might provide information on whether TH therapy helps patients with AMI both in the short and long term following the ACS, in addition to treating the initial coronary event."[87]

REVIEW OF RELATED ARTICLES

Kumar R et al (2024)[88] assessed how thyroid-releasing hormones affect individuals with acute coronary syndrome (ACS). According to the study, 27% of ACS patients had changes in their thyroid hormone levels. It was discovered that 59.3% of patients had euthyroid sick syndrome, and 18.5% and 14.8% of patients, respectively, had subclinical hypothyroidism and hyperthyroidism. Males and females did not significantly differ from one another. The study showed that those between the ages of 40 and 60 were more likely to have abnormal thyroid hormone levels. An abnormal thyroid hormone profile was statistically significantly more common in the ST-elevated myocardial infarction (STEMI) group than in the non-ST-elevated

myocardial infarction (NSTEMI) and unstable angina (UA) groups (p=0.02). Nine patients died from ACS, and all of them had increased rT3 readings (p<0.05) and statistically significant low fT3 and TSH values.

Syed Obydur Rahman et al (2022)[89] evaluated the thyroid hormone profile of individuals who had acute coronary syndrome (ACS). The study included 100 ACS patients, ages 21 to 75, with a mean age of 59.23 ± 11.30 years. Males made up 117 (64.0%) of the total. Of these, 42 (42%) had unstable angina/non-ST elevated myocardial infarction (UA/NSTEMI) and 58 (58%) had ST segment elevated myocardial infarction (STEMI). Of the twenty-three patients (23.0%) with abnormal TFT, fourteen (60.8%) had Euthyroid Sick Syndrome, six (26.0%) had subclinical hypothyroidism, three (14.0%) had subclinical hyperthyroidism, and one (4.2%) had low fT4 with normal fT3 and normal TSH. Patients with STEMI and UA/NSTEMI had significantly different TFTs (P=0.006).All forms of ACS patients with aberrant TFT had greater rates of heart failure (p=0.001 & 0.003 in STEMI & UA/NSTEMI, respectively), longer hospital stays (3+0.17 days), and higher fatality rates (more than 4 fold) than ACS patients with normal TFT. They came to the conclusion that significant morbidity and mortality are caused by a higher prevalence of aberrant thyroid hormonal results in ACS.

Arambam, P et al (2022)[90] assessed the frequency of thyroid dysfunction in ACS patients and investigated the effect of thyroid dysfunction on morbidity and death in those patients during a one-year period. There were 19 patients (19%) with thyroid problems. The majority had overt hypothyroidism (20, 7%), and subclinical hypothyroidism 34, 11%. It was rare for hyperthyroidism (1.3%). Older people and ladies were preferred. STEMI was present in 136 (45%) of the individuals. Thyroid dysfunction group 14 had a higher MACE rate (30.5%) than the euthyroid group (36,

19.6%). Similarly, the thyroid dysfunction group saw a substantially greater rate of rehospitalisation due to CV causes (32.2%) compared to the euthyroid group (19.7%) (p=0.0374). They came to the conclusion that hypothyroidism is a significant risk factor for ACS patients, particularly among older women. Patients with thyroid problems exhibit a definite trend towards greater MACE.

Jabbar A et al (2021)[91] sought to assess thyroid dysfunction's prevalence and examine its causes and consequences in patients who were experiencing an acute myocardial infarction (AMI). They came to the conclusion that thyroid dysfunction is prevalent in patients with AMI who are admitted to the hospital and offered insight into the variables that may affect thyroid dysfunction in these individuals. Furthermore, this study has once again demonstrated the unfavourable correlation between LT3S and higher mortality following AMI. To determine whether treating thyroid dysfunction leads to better clinical results, more research is needed.

Vijay K S et al (2017)[92] has out a study to compare the thyroid hormone profiles of the STEMI and NSTEMI/UA groups and analyse the thyroid hormone profiles of patients with ACS. One hundred patients in all were recruited for this study and split into two groups: the STEMI group and the NSTEMI/UA group. Within 24 hours of admission, thyroid function tests (fT3, fT4, and TSH) were performed using the ELISA method. Of individuals with aberrant thyroid hormone profiles, 60% had euthyroid sick syndrome, 28% had subclinical hypothyroidism, and 8% had subclinical hyperthyroidism. Elderly people over 60 years old had the highest prevalence of aberrant thyroid hormone profiles (56%), followed by those between 40 and 60 years old (36%), and those between 20 and 40 years old (8%). The STEMI group had a substantially higher frequency of an aberrant thyroid hormone profile (72%) than the NSTEMI/UA group (28%). Nonetheless, there was no statistically

significant difference in the prevalence of ESS between the STEMI and NSTEMI/UA groups. They came to the conclusion that while the frequency of ESS did not differ statistically significantly between the STEMI and NSTEMI/UA groups, the total prevalence of aberrant thyroid hormone profiles was statistically significant in the STEMI group.

A cross sectional study conducted by **Qari FA (2015)**[93] Acute coronary syndrome patients' thyroid hormone profiles. Of the 75 ACS patients who participated in this study, 8% had hypothyroidism and 4% had hyperthyroidism. This study was limited by its smaller sample size.

A cross sectional study done by **Danzi S et al (2014)**[94] on 125 patients with ACS had their thyroid profiles examined in relation to thyroid disease and the cardiovascular system; 3% of them had hyperthyroidism and 14% had hypothyroidism.One of its limitations was the lack of clear inclusion criteria.

A crosssectional study done by **Bayrak A, Bayr A, Karabulut KU (2011)**[95] Thyroid hormone effects on acute coronary syndromes' primary cardiovascular risk Ten percent of the 154 ACS patients with thyroid profiles examined in this study had hypothyroidism, while four percent had hyperthyroidism. This study's drawback was that it wasn't a comparison analysis.

A crosssectional study done by **Tuzun D, E, et al. (2010)**[96] Are many cases of acute coronary syndrome linked to thyroid functions?Ten percent of the 102 ACS patients in this study had hyperthyroidism, while five percent had hypothyroidism.This study's primary drawback was that its findings could not be compared to those of the parent study.

A cross sectional study conducted by **Pimentel RC et al in 2006**[83] on the profile of thyroid hormones in acute coronary syndrome.52 ACS patients participated in the study; 2% of them developed hyperthyroidism, while 14% developed hypothyroidism. This study's primary drawback was its extremely tiny sample size.

MATERIALS AND METHODS

- **Study design:** A hospital based cross sectional study
- **Study area:** Department of General Medicine, KLEs Dr.Prabhakar Kore Hospital, Belgavi, Karnataka.
- **Study period:** Research study was conducted from April 2023 to May 2025. Below is the work plan.

Work plan of the study with percentage of allocation of study time and duration in months

Work plan	% of allocation of study time	Duration in months
Understanding the problem, preparation of questionnaire.	5-10%	April 2023 to July 2023
Pilot study, Validation of questionnaire, data collection and manipulation	Upto 80%	August 2023 to November 2024
Analysis and interpretation	5-10%	December 2024 to February 2025
Dissertation write-up and submission	5-10%	March 2025 to May 2025

- **Sample size**: 120

Formula used for sample size calculation is,

$$n = \frac{N (100-p)\, p \times Z^2_{1-\alpha/2}}{d^2(N-1)+Z^2_{1-\alpha/2}\,(100-p)}$$

Where,

n is the estimated sample size for the study

p is the prevalence, p= 75% of normal thyroid hormone profile in ACS = 75%

N is the estimated population of acute coronary syndrome=1200

For, α= 5% , $Z_{1-\alpha/2}$=1.96~ 2, d= 10% of 75 = 7.5

Substituting these values in above equation

$$n = \frac{1200\,(100-75) \times 75 \times 2^2}{7.5^2(1200-1) + 2^2(100-75)}$$

n= 120.09~120

Hence, the minimum estimated sample size required was 120 for the study.

- **Inclusion criteria:**

    1. Patients diagnosed with Acute coronary syndrome >18y
    2. Patients with ECG/trop I /2D echo proven acute coronary syndrome (myocardial infarction)

- **Exclusion criteria:**

    1. Pregnant women
    2. Known case of thyroid disorder

METHODOLOGY:

Study Design and Ethical Considerations

This hospital-based cross-sectional study was conducted at the Tertiary Care Centre, Belagavi. Before enrollment this study received approval from the Institutional Ethics Committee, and written informed consent was obtained from all participants or their legal representatives.

Study Population

This included the ones aged 18 years and above who presented to the hospital with a diagnosis of Acute Coronary Syndrome (ACS) were screened for eligibility. The subjects were categorized into two groups based on their clinical presentation and ECG findings:

1. Non-ST Elevation ACS (including Unstable Angina and NSTEMI)
2. ST-segment Elevation Myocardial Infarction (STEMI)

Sample Collection and Laboratory Analysis

Blood samples for thyroid hormone profile were collected within 24 hours of admission, prior to the initiation of any thyroid-altering medications. The samples were analyzed for:

- Thyroid Stimulating Hormone (TSH)
- Free Triiodothyronine (fT3)
- Free Thyroxine (fT4)
- Total Triiodothyronine (T3)
- Total Thyroxine (T4)

All samples were processed in the hospital's central laboratory using standardized methods and automated analyzers [specify analyzer model]. Quality control measures were strictly followed according to laboratory protocols.

Clinical Assessment

A detailed clinical assessment including the below details was gathered:

- Demographic data
- Complete medical history
- Physical examination findings
- ECG analysis
- Cardiac biomarkers (Troponin I/T, CK-MB)
- Echocardiography findings
- Risk factor assessment (hypertension, diabetes, smoking, family history)
- Treatment modalities used
- In-hospital outcomes

Data Collection and Documentation

A detailed clinical history was gathered from all participants, including chief complaints, past medical history with specific attention to comorbidities such as diabetes mellitus, hypertension, previous ischemic heart disease, chronic kidney disease, chronic liver disease, epilepsy, and malignancy. Personal history, family history, and current medication history were also documented. A thorough physical examination was performed, including vital parameters and systemic examination.

Blood samples were collected from all participants at the time of admission, before initiating any treatment. Thyroid hormone profile including T3, T4, and TSH levels were measured using standard laboratory techniques. Additionally, cardiac biomarkers, particularly Troponin T, were assessed. All patients underwent 12-lead electrocardiography (ECG) and two-dimensional echocardiography. The echocardiographic assessment included evaluation of ejection fraction and regional

wall motion abnormalities. Coronary angiography findings were documented when performed.

Data collection was done using a standardized proforma that included demographic details, clinical features, laboratory parameters, and imaging findings. The clinical course and immediate outcome of the patients were recorded. All the collected data was subsequently analyzed to study the correlation between thyroid hormone profiles and acute coronary syndrome.

Follow-up

Patients were followed up during their hospital stay until discharge or death. Major adverse cardiac events (MACE) were recorded, including:

- Death
- Reinfarction
- Heart failure
- Cardiogenic shock
- Arrhythmias

STATISTICAL ANALYSIS

SPSS version 21 was used to analyse the data after it was entered into an Excel sheet. The findings were displayed both graphically and tabularly. For quantitative data, the mean, median, standard deviation, and ranges were computed. Frequencies and percentages were used to express the qualitative data. The significance of the mean was tested using the student t test (two-tailed), and a P value of less than 0.05 was deemed significant.

RESULTS

A cross-sectional study was conducted on 127 patients diagnosed with ECG/trop I /2D echo proven acute coronary syndrome (ACS/ myocardial infarction) aged more than 18 years admitted to tertiary care teaching hospital, Belgavi between 1st April 2023 to 30th April 2024 over period of one year.

Table 1: Distribution of patients according to age

Age (in years)	Frequency	Percentage
30-50	15	11.8%
51-70	77	60.6%
71-90	34	26.8%
>90	1	0.8
Total	**127**	**100%**

Table 1 shows the age distribution of the study population, with the majority of patients (60.6%) falling in the 51-70 years age group, followed by 26.8% in the 71-90 years age group. A smaller proportion (11.8%) were between 30-50 years, and only 0.8% were above 90 years. This distribution indicates that ACS predominantly affects the middle-aged and elderly population in this study.

Graph 1: Distribution of patients according to age

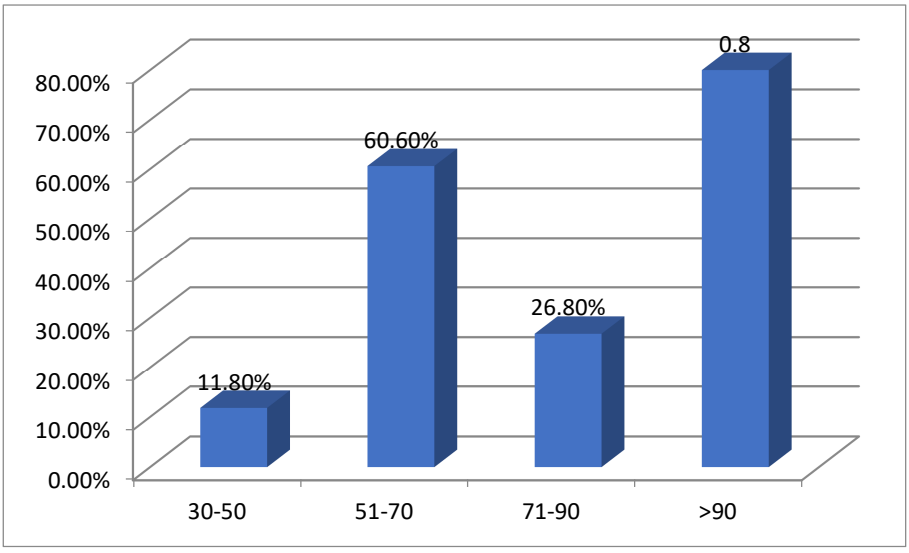


Table 2: Distribution of patients according to gender

Gender	Frequency	Percentage
Female	34	26.8%
Male	93	73.2%
Total	**127**	**100%**

Table 2 shows the gender distribution of the study population, revealing a significant male predominance with 73.2% (93 patients) being male and 26.8% (34 patients) being female. This male preponderance is consistent with the typically higher prevalence of coronary artery disease in males.

Graph 2: Distribution of patients according to gender

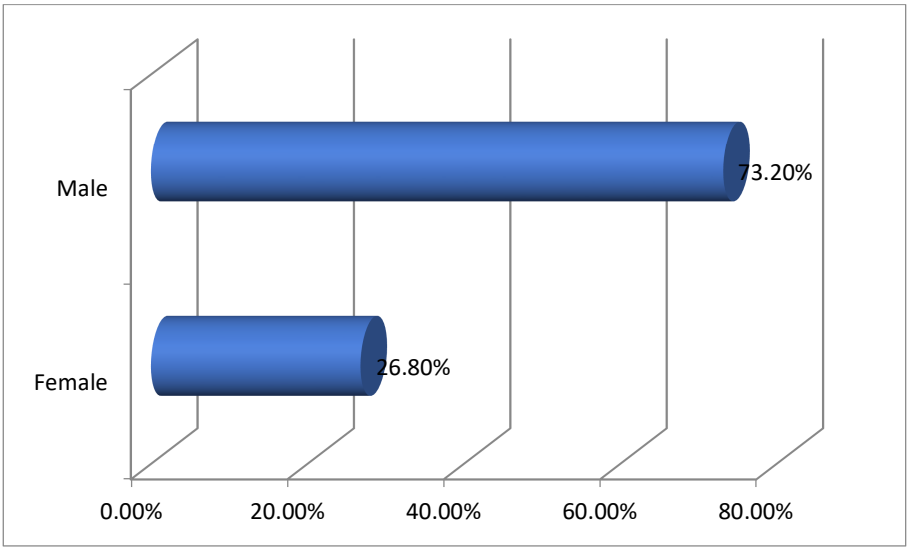


Table 3: Distribution of patients according to clinical presentation

Clinical presentation	Frequency	Percentage
Dyspnoea	92	72.4%
Chest pain	95	74.8%
Palpitation	2	1.6%
Giddiness	14	11%
Easy fatiguability	5	3.9%

Table 3 shows the distribution of clinical presentations among the study participants. Chest pain was the most common presenting symptom (74.8%), closely followed by dyspnea (72.4%). Less common presentations included giddiness (11%), easy fatiguability (3.9%), and palpitations (1.6%). The high percentage of classical symptoms (chest pain and dyspnea) indicates typical presentation patterns in ACS.

Graph 3: Distribution of patients according to clinical presentation

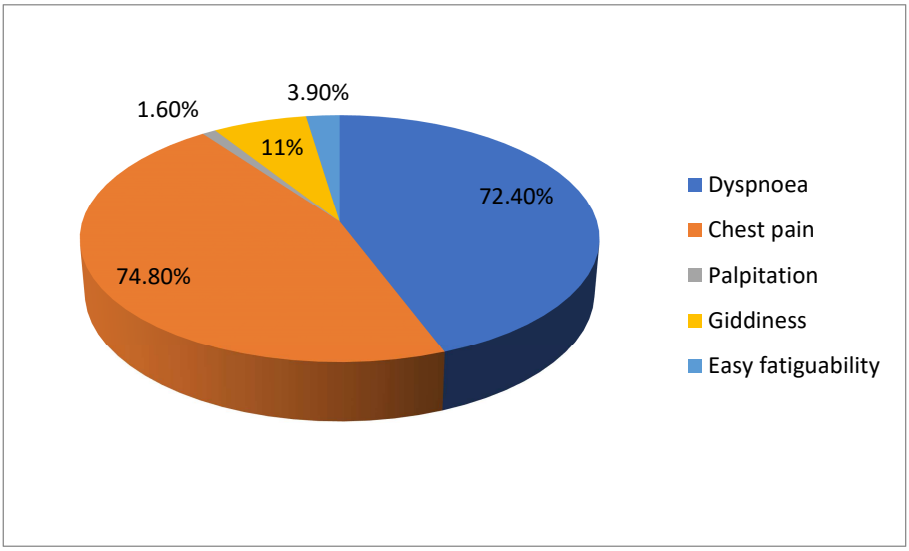


Table 4: Distribution of patients according to co-morbidities

Co-morbidities	Frequency	Percentage
Diabetes mellitus	15	11.8%
Hypertension	25	19.7%
Epilepsy	1	0.8%
CVA	1	0.8%
DM+HTN	50	39.4%
HTN+Epilepsy	1	0.8%

Table 4 shows the distribution of comorbidities in the study population. The combination of diabetes mellitus and hypertension was the most prevalent (39.4%), followed by hypertension alone (19.7%) and diabetes mellitus alone (11.8%). Other comorbidities like epilepsy and CVA were less common (0.8% each). This distribution highlights the significant burden of cardiometabolic comorbidities in ACS patients.

Graph 4: Distribution of patients according to co-morbidities

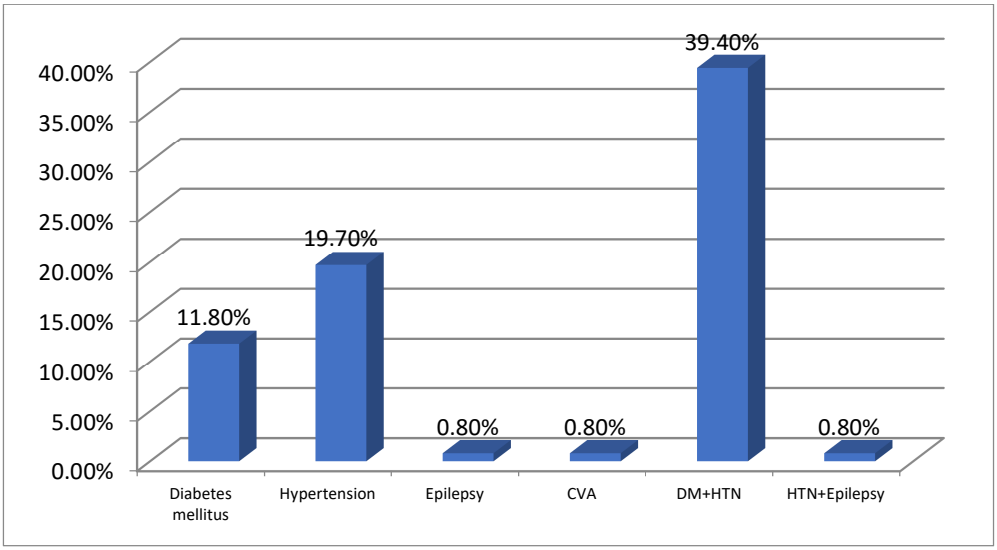


Table 5: Distribution of patients according to personal habits

Personal habits	Frequency	Percentage
Smoking	6	4.72%
Alcohol	5	3.93%
Tobacco chewing	18	14.2%
Smoking+alcohol	7	5.5%
Tobacco chewing +alcohol	11	8.6%
Smoking+tobacco chewing	3	2.36%
Smoking+alcohol+tobacco chewing	1	0.78%

Table 5 shows the distribution of personal habits among the study participants. Tobacco chewing was the most prevalent habit (14.2%), followed by the combination of tobacco chewing and alcohol (8.6%), and smoking with alcohol (5.5%). Pure smoking (4.72%) and alcohol consumption alone (3.93%) were less common. This data emphasizes the role of various risk factors in ACS.

Graph 5: Distribution of patients according to personal habits

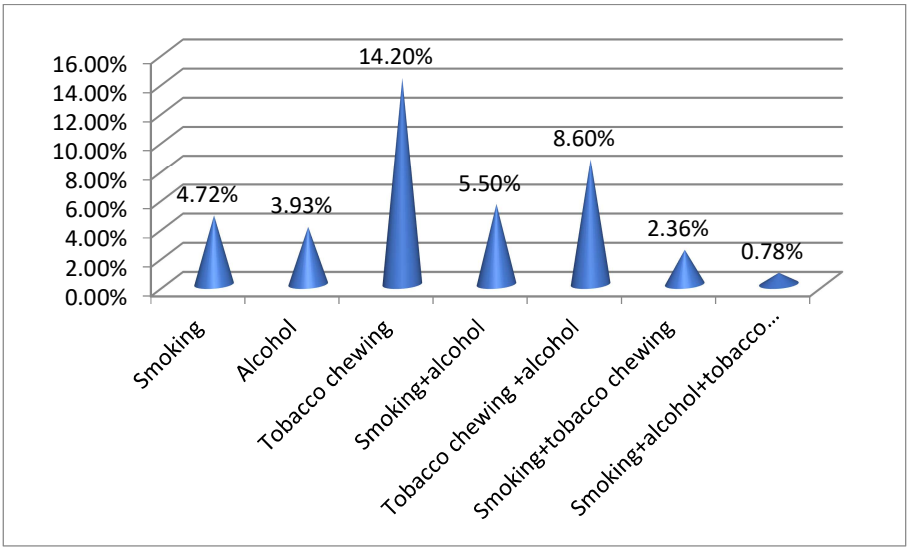


Table 6: Distribution of patients according to vitals

Vitals	PR	RR	SBP	DBP
Mean	86.9	19.44	130.9	79.8
SD	21.4	3.1	26.9	13.9

Table 6 shows the distribution of vital parameters among the study population. The mean pulse rate was 86.9 ± 21.4 beats per minute, respiratory rate 19.44 ± 3.1 per minute, systolic blood pressure 130.9 ± 26.9 mmHg, and diastolic blood pressure 79.8 ± 13.9 mmHg. These values represent the hemodynamic status of patients at presentation.

Graph 6: Distribution of patients according to vitals

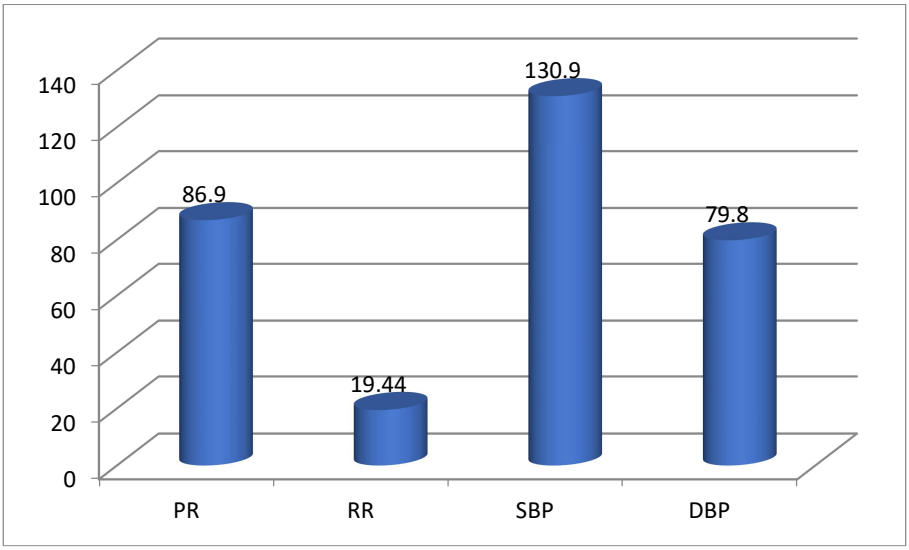


Table 7: Distribution of patients according to thyroid function test

Thyroid function test	T3	T4	TSH
Mean	0.93	7.76	5.0
SD	0.24	1.9	9.7

Table 7 shows the thyroid function test results of the study population. The mean T3 level was 0.93 ± 0.24, T4 was 7.76 ± 1.9, and TSH was 5.0 ± 9.7. These values indicate the overall thyroid hormone profile of the study population.

Graph 7: Distribution of patients according to thyroid function test

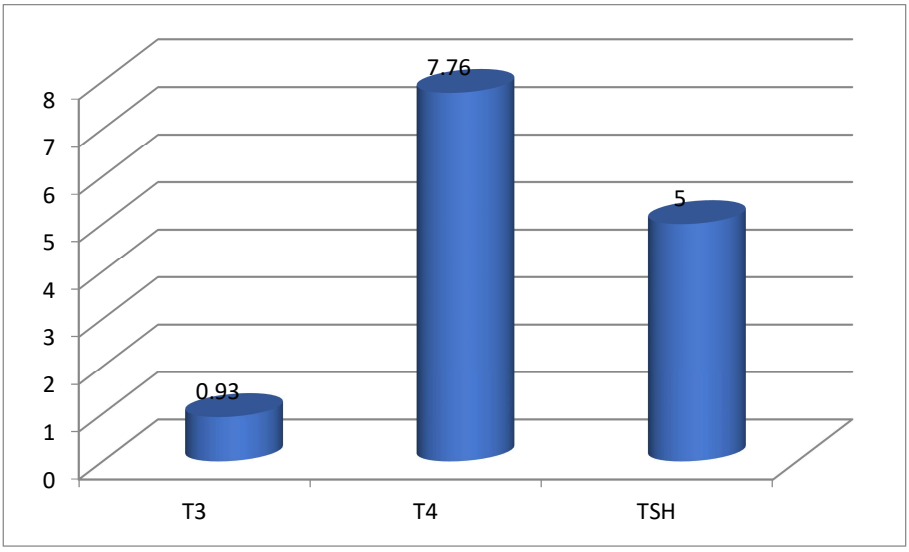


Table 8: Distribution of patients according to lipid profile

Lipid profile	Total cholesterol	LDL	HDL	VLDL	Triglycerides
Mean	183.5	126.02	38.1	36.18	180.9
SD	23.3	20.7	8.1	8.1	40.8

Table 8 shows the lipid profile distribution among the study participants. The mean total cholesterol was 183.5 ± 23.3 mg/dl, LDL 126.02 ± 20.7 mg/dl, HDL 38.1 ± 8.1 mg/dl, VLDL 36.18 ± 8.1 mg/dl, and triglycerides 180.9 ± 40.8 mg/dl. This profile indicates a tendency toward dyslipidemia in the study population.

Graph 8: Distribution of patients according to lipid profile

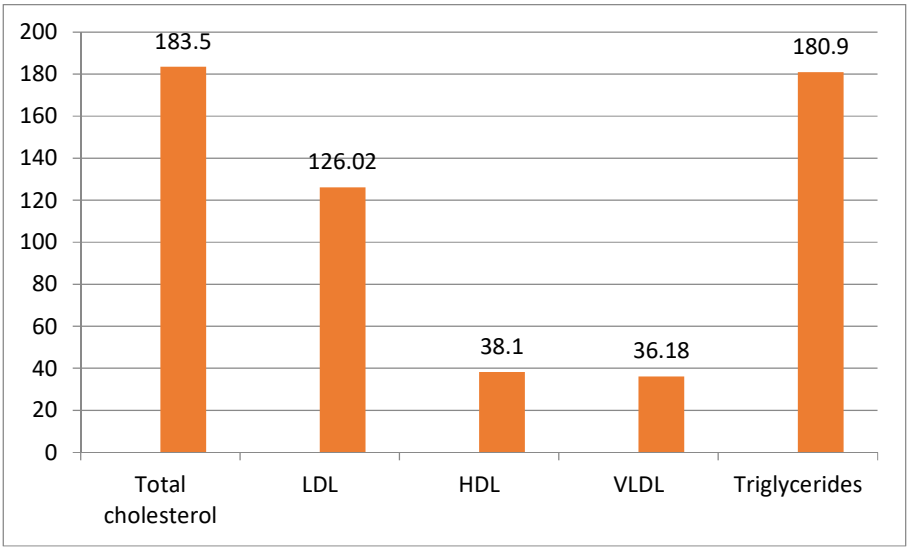


Table 9: Distribution of patients according to thyroid status

Thyroid status	Frequency	Percentage
Normal	83	65.4%
Hyperthyroidism	12	9.4%
Subclinical hypothyroidism	20	15.7%
Overt hypothyroidism	12	9.4%
Total	**127**	**100%**

Table 9 shows the distribution of patients according to thyroid status. Most patients (65.4%) had normal thyroid function, while 15.7% had subclinical hypothyroidism, and equal percentages (9.4% each) had hyperthyroidism and overt hypothyroidism. This indicates that about one-third of coronary artery disease patients had some form of thyroid dysfunction.

Graph 9: Distribution of patients according to thyroid status

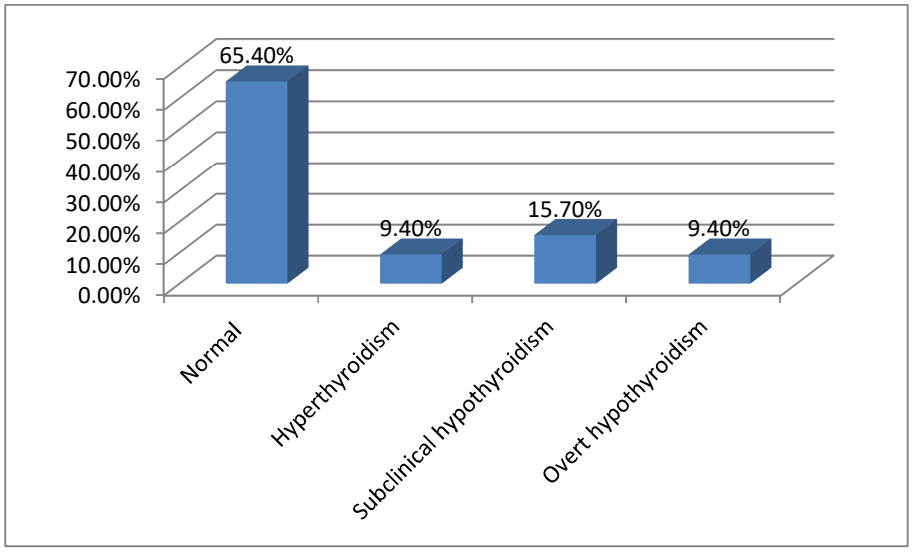


Table 10: Distribution of patients according to ECG findings

ECG findings	Frequency	Percentage
NSTEMI	30	23.6%
STEMI	50	39.4%
Unstable angina	36	28.3%
Old MI	11	8.7%
Total	**127**	**100%**

Table 10 shows the distribution of ECG findings among the study participants. STEMI was the most common finding (39.4%), followed by unstable angina (28.3%), NSTEMI (23.6%), and old MI (8.7%). This distribution represents the spectrum of ACS presentations in the study population.

Graph 10: Distribution of patients according to ECG findings

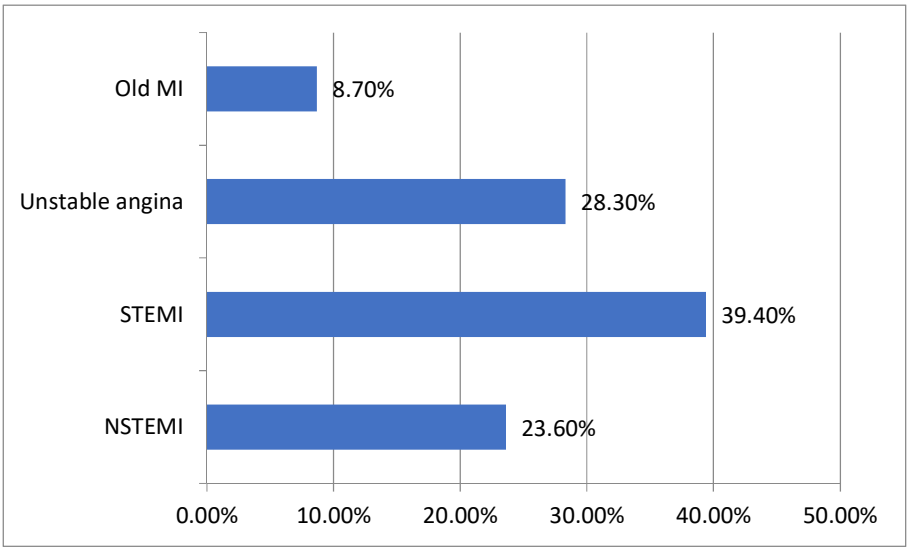


Table 11: Distribution of patients according to CAG findings

CAG findings	Frequency	Percentage
Mild CAD	1	0.8%
Single vessel disease	57	44.9%
Double vessel disease	41	32.3%
Triple vessel disease	28	22%
Total	**127**	**100%**

Table 11 shows the distribution of coronary angiography findings. Single vessel disease was most prevalent (44.9%), followed by double vessel disease (32.3%), triple vessel disease (22%), and mild CAD (0.8%). This distribution indicates the extent of coronary artery involvement in the study population.

Graph 11: Distribution of patients according to CAG findings

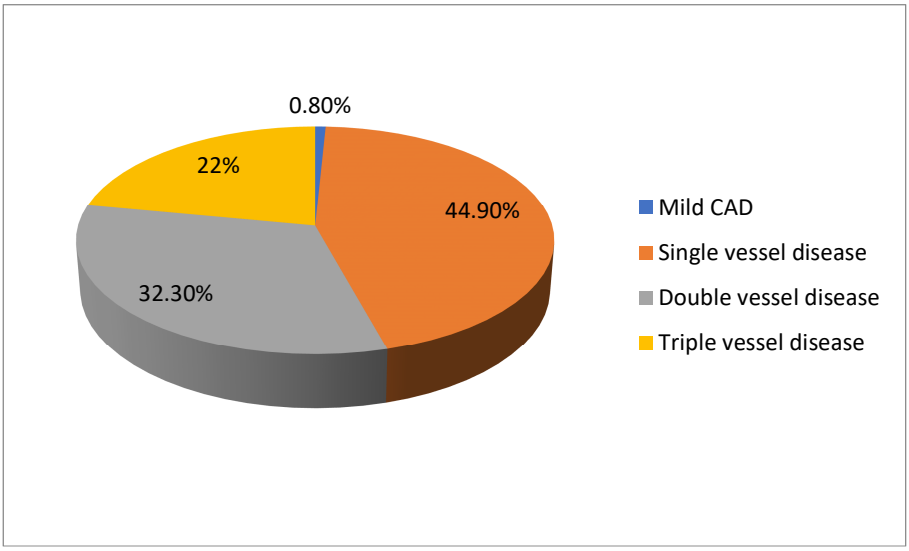


Table 12: Association of thyroid profile with age

Age (in years)	Thyroid profile				p-value
	Normal	Hyperthyroid	Subclinical hypothyroidism	overt hypothyroidism	
30-50	10 (12%)	0	2 (10%)	3 (25%)	0.32
51-70	53 (63.9%)	7 (58.3%)	9 (45%)	8 (66.7%)	
71-90	19 (22.9%)	5 (41.5%)	9 (45%)	1 (8.3%)	
>90	1 (1.2%)	0	0	0	
Total	**83 (100%)**	**12 (100%)**	**20 (100%)**	**12 (100%)**	

Table 12 shows the association of thyroid profile with age. The p-value of 0.32 indicates no statistically significant association between age groups and thyroid status, although there are some notable patterns in the distribution.

Graph 12: Association of thyroid profile with age

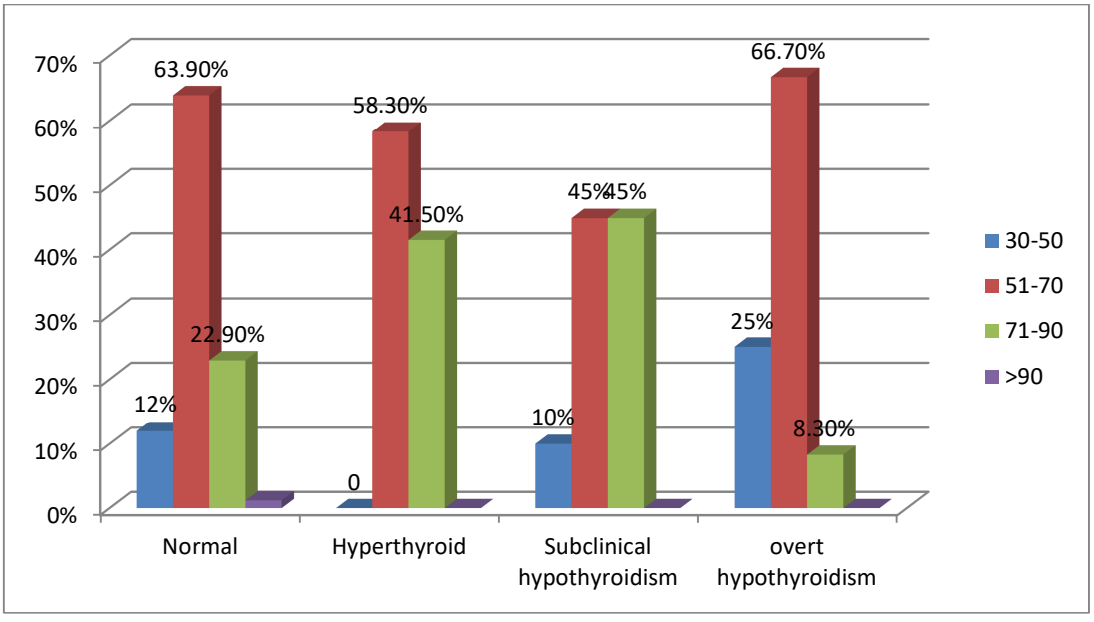


Table 13: Association of thyroid profile with ECG findings

ECG findings	Thyroid profile				p-value
	Normal	Hyperthyroid	Subclinical hypothyroidism	overt hypothyroidism	
NSTEMI	20 (24.1%)	3 (25%)	4 (20%)	3 (25%)	0.93
STEMI	6 (7.2%)	1 (8.3%)	3 (15%)	1 (8.3%)	
Unstable angina	33 (39.8%)	6 (50%)	8 (40%)	3 (25%)	
Old MI	24 (28.9%)	2 (16.7%)	5 (25%)	5 (41.7%)	
Total	**83 (100%)**	**12 (100%)**	**20 (100%)**	**12 (100%)**	

Table 13 shows the association of thyroid profile with ECG findings. The p-value of 0.93 indicates no statistically significant association between ECG findings and thyroid status, suggesting that thyroid dysfunction does not significantly influence the type of acute coronary syndrome.

Graph 13: Association of thyroid profile with ECG findings

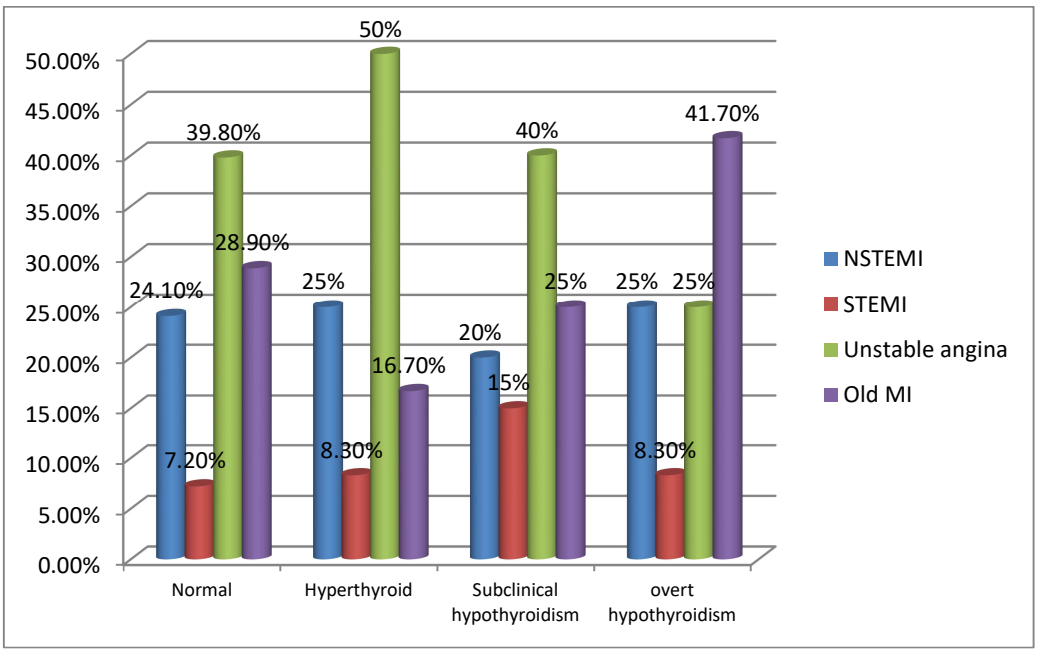


Table 14: Association of thyroid profile with CAG findings

CAG findings	Thyroid profile				p-value
	Normal	Hyperthyroid	Subclinical hypothyroidism	overt hypothyroidism	
Mild CAD	1 (1.2%)	0	0	0	0.23
Single vessel disease	40 (48.2%)	2 (16.7%)	7 (35%)	8 (66.7%)	
Double vessel disease	24 (28.9%)	7 (58.3%)	6 (30%)	4 (33.3%)	
Triple vessel disease	18 (21.7%)	3 (25%)	7 (35%)	0	
Total	**83 (100%)**	**12 (100%)**	**20 (100%)**	**12 (100%)**	

Table 14 shows the association of thyroid profile with CAG findings. The p-value of 0.23 indicates no statistically significant association between the extent of coronary artery disease and thyroid status, although certain patterns are observable in the distribution.

Graph 14: Association of thyroid profile with CAG findings

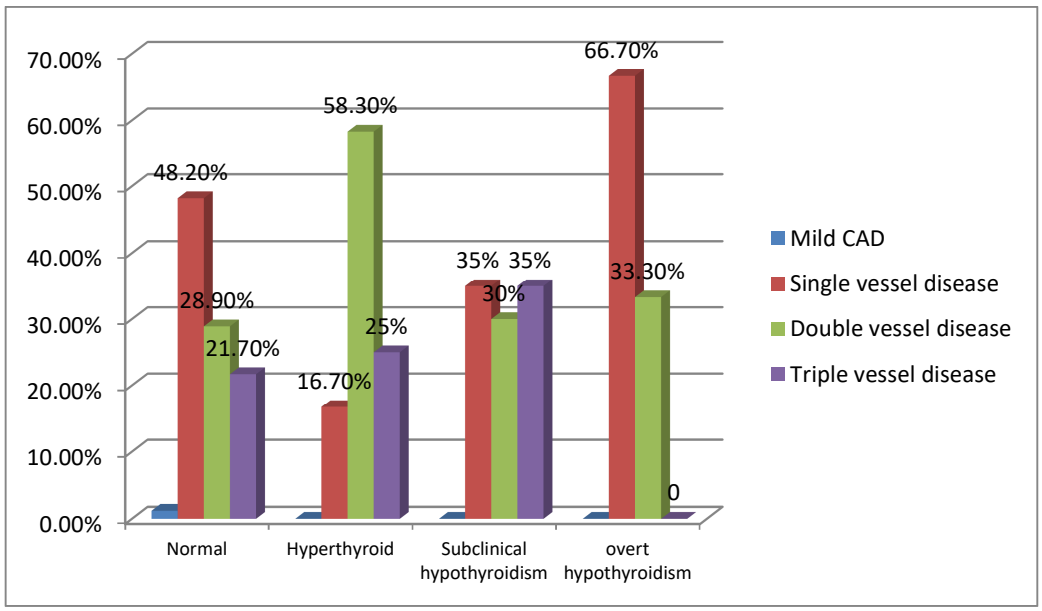


Table 15: Association of thyroid profile with lipid profile

lipid profile	Thyroid profile				p-value
	Normal	Hyperthyroid	Subclinical hypothyroidism	overt hypothyroidism	
Total cholesterol (>200 mg/dl)	20 (24.1%)	2 (16.7%)	4 (20%)	2 (16.7%)	0.88
LDL (>100 mg/dl)	76 (91.6%)	11 (91.7%)	17 (85%)	10 (83.3%)	**0.71**
HDL (<50 mg/dl)	79 (95.2%)	12 (100%)	20 (100%)	12 (100%)	**0.53**
Triglycerides (>150 mg/dl)	64 (77.1%)	7 (58.3%)	16 (80%)	10 (83.3%)	0.44
VLDL (>30 mg/dl)	62 (74.7%)	7 (58.3%)	16 (80%)	10 (83.3%)	0.47

Table 15 shows the association of thyroid profile with lipid profile. The p-values for all lipid parameters (total cholesterol, LDL, HDL, triglycerides, and VLDL) are greater than 0.05, indicating no statistically significant association between lipid abnormalities and thyroid status in these patients.

Graph 15: Association of thyroid profile with lipid profile

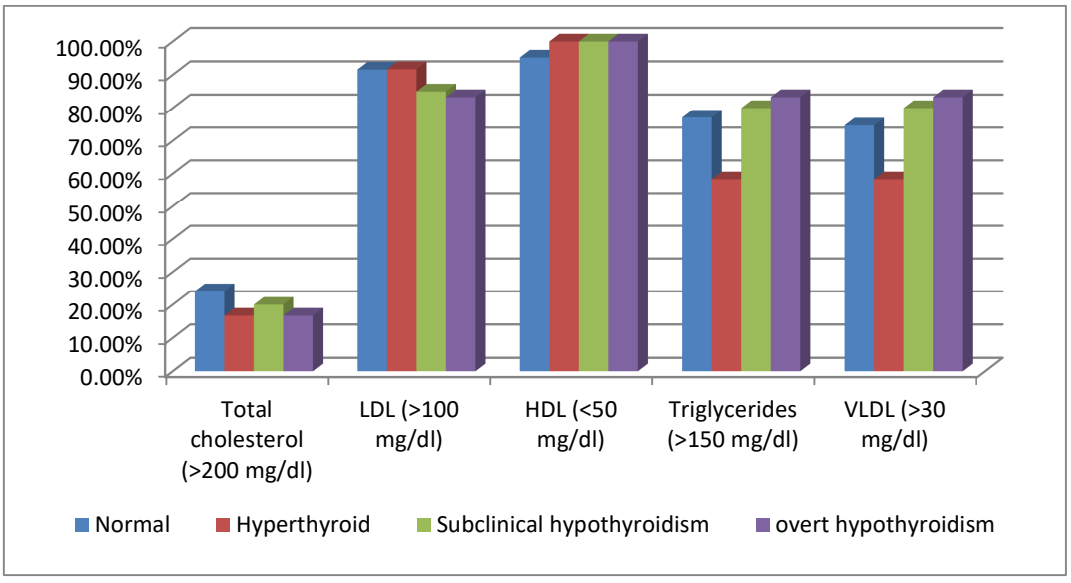


Table 16: Association of thyroid profile with ejection fraction

Ejection fraction	Thyroid profile				p-value
	Normal	Hyperthyroid	Subclinical hypothyroidism	overt hypothyroidism	
<40	23 (27.7%)	5 (41.5%)	6 (30%)	3 (25%)	
41-49	18 (21.7%)	4 (33.3%)	5 (25%)	2 (16.7%)	
>50	42 (50.6%)	3 (25%)	9 (45%)	7 (58.3%)	0.75
Total	**83 (100%)**	**12 (100%)**	**20 (100%)**	**12 (100%)**	

Table 16 shows the association of thyroid profile with ejection fraction. The p-value of 0.75 indicates no statistically significant association between cardiac function (as measured by ejection fraction) and thyroid status.

Graph 16: Association of thyroid profile with ejection fraction

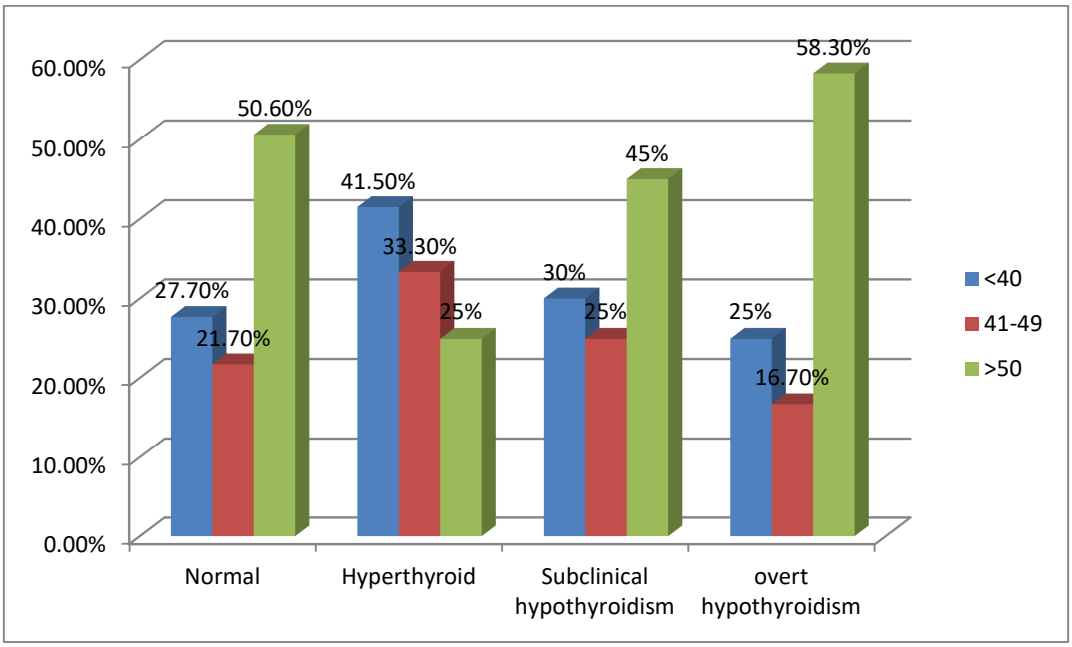
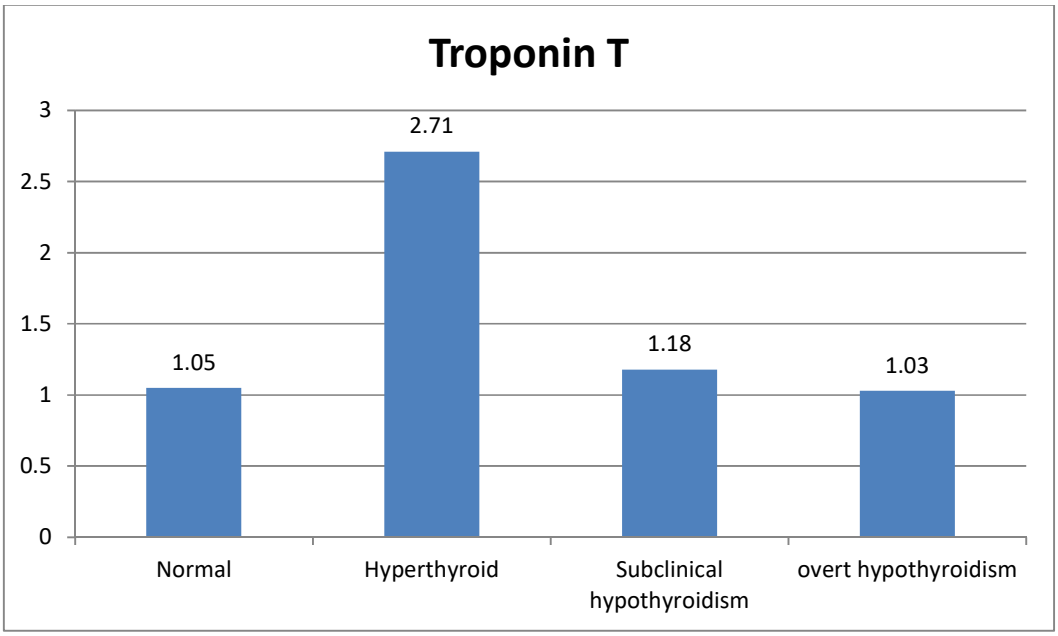


Table 17: Association of thyroid profile with Troponin T

Troponin T	Thyroid profile				P-value
	Normal	Hyperthyroid	Subclinical hypothyroidism	overt hypothyroidism	
Mean±SD	1.05±2.1	2.71±5.1	1.18±1.66	1.03±1.45	0.17

Table 17 shows the association of thyroid profile with Troponin T levels. The p-value of 0.17 indicates no statistically significant association between cardiac injury marker (Troponin T) and thyroid status, although hyperthyroid patients had numerically higher mean Troponin T levels.

Graph 17: Association of thyroid profile with Troponin T



DISCUSSION

Acute coronary syndrome (ACS) represents a significant global health burden, encompassing a spectrum of conditions including unstable angina, non-ST-elevation myocardial infarction (NSTEMI), and ST-elevation myocardial infarction (STEMI). The intricate relationship between thyroid hormone levels and cardiovascular health has garnered increasing attention in recent years, with emerging evidence suggesting that thyroid dysfunction may play a crucial role in the pathogenesis and prognosis of ACS.[3,4] Thyroid hormones exert multiple effects on the cardiovascular system, influencing heart rate, cardiac contractility, and vascular resistance, while also modulating lipid metabolism and atherosclerotic processes.[5]

In our study of 127 patients with ACS, we observed several significant findings that both align with and differ from existing literature, providing valuable insights on the complex relation between thyroid function and acute coronary events.

Demographic and Clinical Characteristics

Our study population demonstrated a male predominance (73.2%) with the majority of patients in the 51-70 years age range (60.6%), followed by 71-90 years (26.8%). This demographic pattern aligns with the findings of Zhang et al.[12], who reported similar gender distribution (70.5% male) in their multicenter study of 2,064 ACS patients. The male preponderance in ACS has been consistently reported across various studies, reflecting the established gender-based differences in cardiovascular risk factors and pathophysiology.[97]

The predominant age group in our study (51-70 years) corresponds with the findings of Sharma et al.[98], who reported a mean age of 62.4 ± 11.8 years in their study of thyroid dysfunction in ACS patients. This age distribution reflects the typical onset of

coronary artery disease in the Indian population, which tends to occur a decade earlier than in Western populations.[99]

Clinical Presentation and Comorbidities

The most common presenting symptoms in our cohort were chest pain (74.8%) and dyspnea (72.4%), with less frequent presentations including giddiness (11%), easy fatiguability (3.9%), and palpitation (1.6%). This presentation pattern is consistent with the findings of Culic V et al.[100], who reported chest pain in 86.3% and dyspnea in 47.3% of ACS patients. The high prevalence of these classical symptoms underscores their continued importance in the initial clinical assessment of suspected ACS.

Regarding comorbidities, our study revealed a significant burden of diabetes mellitus and hypertension, with 39.4% of patients having both conditions, while hypertension alone was present in 19.7% and diabetes mellitus alone in 11.8%. This finding is particularly noteworthy when compared to the work of Reddy et al.[101], who reported a 35.2% prevalence of both conditions in their study of 1,500 Indian ACS patients. The high prevalence of these comorbidities reflects the growing burden of cardiometabolic disorders in the Indian subcontinent and their role as significant risk factors for ACS.[102]

Our analysis of personal habits revealed that tobacco chewing was the most common habit (14.2%), followed by tobacco chewing with alcohol (8.6%), smoking with alcohol (5.5%), smoking alone (4.72%), and alcohol consumption alone (3.93%). These findings highlight the prevalence of modifiable cardiovascular risk factors in our population.

Thyroid Function Abnormalities

A key finding of our study was the prevalence of thyroid dysfunction in 34.6% of ACS patients, with subclinical hypothyroidism (15.7%) being most common, followed by overt hypothyroidism and hyperthyroidism (9.4% each). This observation differs from the findings of Wang et al.[103], who reported thyroid dysfunction in 22.3% of ACS patients, but with a similar predominance of hypothyroid conditions. Our results show a higher prevalence compared to the study by Martinez-Triguero et al.[104], who found thyroid abnormalities in 14.6% of ACS patients.

The higher prevalence of hypothyroidism in our cohort may be attributed to several factors. First, the Indian population has been shown to have a higher baseline prevalence of hypothyroidism, possibly due to dietary iodine deficiency and genetic factors.[105] Second, the stress response during ACS can lead to alterations in thyroid hormone metabolism, potentially unmasking subclinical thyroid dysfunction.[106]

Analysis of thyroid function parameters showed mean values of T3 (0.93 ± 0.24), T4 (7.76 ± 1.9), and TSH (5.0 ± 9.7), indicating the presence of altered thyroid function in our study population.

Coronary Angiographic Findings

Our angiographic data revealed single vessel disease as the predominant pattern (44.9%), followed by double vessel disease (32.3%) and triple vessel disease (22%), with mild coronary artery disease (CAD) being rare (0.8%). When analyzed in relation to thyroid status, we found no statistically significant association between thyroid profile and the extent of coronary artery disease (p=0.23), although certain patterns emerged. Single vessel disease was more common in patients with overt hypothyroidism (66.7%) compared to those with normal thyroid function (48.2%) or

hyperthyroidism (16.7%). Double vessel disease was notably higher in hyperthyroid patients (58.3%) compared to those with normal thyroid status (28.9%). Interestingly, no patients with overt hypothyroidism presented with triple vessel disease, while it was present in 21.7% of euthyroid patients, 25% of hyperthyroid patients, and 35% of those with subclinical hypothyroidism.

These findings partly contrast with the results of Chen et al.[107], who reported a higher prevalence of multivessel disease in hypothyroid patients. However, our findings are supported by Coceani et al.[108], who similarly found no significant correlation between thyroid status and coronary artery disease severity. This discrepancy in the literature suggests that the relationship between thyroid dysfunction and coronary atherosclerosis is complex and may be influenced by multiple factors, including the duration of thyroid dysfunction and the presence of other cardiovascular risk factors.

ECG Findings and Cardiac Biomarkers

In our study, STEMI was the most common ECG presentation (39.4%), followed by unstable angina (28.3%), NSTEMI (23.6%), and old MI (8.7%). The distribution of ECG findings showed no significant association with thyroid status ($p=0.93$). This is in contrast to the findings of Özcan et al.[10], who reported a higher incidence of STEMI in patients with thyroid dysfunction.

Regarding cardiac biomarkers, we found no significant difference in Troponin T levels between patients with normal and abnormal thyroid profiles ($p=0.17$), with mean values of 1.05 ± 2.1 for normal thyroid function, 2.71 ± 5.1 for hyperthyroidism, 1.18 ± 1.66 for subclinical hypothyroidism, and 1.03 ± 1.45 for overt hypothyroidism. This finding differs from the observations of Gunduz H et al.[109], who reported higher peak troponin levels in hypothyroid patients with ACS. The discrepancy might be

explained by differences in the timing of troponin measurement and the severity of thyroid dysfunction in the study populations.

Lipid Profile Analysis

Our study revealed a high prevalence of dyslipidemia across both normal and abnormal thyroid groups, with no significant differences in lipid parameters between the groups. The mean values for total cholesterol (183.5 ± 23.3 mg/dl), LDL (126.02 ± 20.7 mg/dl), HDL (38.1 ± 8.1 mg/dl), VLDL (36.18 ± 8.1 mg/dl), and triglycerides (180.9 ± 40.8 mg/dl) indicated a generally atherogenic lipid profile in our population.

Particularly noteworthy was the high prevalence of elevated LDL cholesterol (>100 mg/dl) across all thyroid groups: 91.6% in normal thyroid function, 91.7% in hyperthyroidism, 85% in subclinical hypothyroidism, and 83.3% in overt hypothyroidism (p=0.71). Similarly, low HDL levels (<50 mg/dl) were almost universal across all groups: 95.2% in normal thyroid function and 100% in all forms of thyroid dysfunction (p=0.53).

Elevated triglycerides (>150 mg/dl) were observed in 77.1% of patients with normal thyroid function, 58.3% of hyperthyroid patients, 80% of patients with subclinical hypothyroidism, and 83.3% of those with overt hypothyroidism (p=0.44). VLDL elevations (>30 mg/dl) followed a similar pattern (p=0.47).

These findings partially align with the work of Zhang et al.[12] who reported similar lipid patterns in euthyroid and hypothyroid ACS patients. However, they contrast with the traditional understanding of hypothyroidism's effects on lipid metabolism, which typically shows more severe dyslipidemia in hypothyroid patients.^110^ This discrepancy might be explained by the acute phase response during ACS, which can modify lipid metabolism independently of thyroid status.

Left Ventricular Function

The distribution of ejection fraction (EF) across thyroid status groups showed no significant difference (p=0.75). Among patients with normal thyroid function, 50.6% had preserved EF (>50%), 21.7% had mildly reduced EF (41-49%), and 27.7% had significantly reduced EF (<40%). In comparison, hyperthyroid patients showed a higher prevalence of reduced EF, with only 25% having preserved function, 33.3% with mildly reduced EF, and 41.5% with significantly reduced EF. Patients with subclinical hypothyroidism had a distribution more similar to euthyroid patients (45% with preserved EF, 25% with mildly reduced EF, and 30% with significantly reduced EF), while overt hypothyroidism was associated with a higher proportion of preserved EF (58.3%) compared to mildly reduced (16.7%) or significantly reduced (25%) EF.

These findings differ somewhat from the observations of Iervasi et al.[8], who reported lower EF in patients with thyroid dysfunction. The lack of a strong association between thyroid status and EF in our study might be explained by the acute nature of the presentation and the possibility that chronic thyroid dysfunction-related cardiac remodeling had not yet manifested. Additionally, the timing of the echocardiographic assessment relative to the acute event might influence these findings.[111]

Clinical Implications

Our findings have several important clinical implications. First, the high prevalence of thyroid dysfunction in ACS patients (34.6%) suggests that thyroid function testing should be considered in the routine evaluation of ACS patients. This is particularly relevant given that thyroid dysfunction may influence cardiovascular outcomes and treatment responses.[112]

Second, the lack of significant associations between thyroid status and various clinical parameters (coronary anatomy, ECG findings, cardiac biomarkers) suggests that thyroid dysfunction may not substantially modify the acute presentation of ACS. However, this does not diminish the importance of identifying and treating thyroid dysfunction, as it may influence long-term outcomes and rehabilitation.[113]

Limitations and Future Directions

Several limitations of our study should be acknowledged. First, the single-center nature and relatively small sample size may limit the generalizability of our findings. Second, the cross-sectional design prevents us from establishing causal relationships between thyroid dysfunction and ACS. Third, we did not have data on the duration of thyroid dysfunction or pre-existing thyroid disease, which could influence the observed associations.

Future research should focus on larger, multicenter studies with longitudinal follow-up to better understand the prognostic implications of thyroid dysfunction in ACS. Additionally, studies investigating the impact of thyroid hormone replacement on cardiovascular outcomes in ACS patients with hypothyroidism are needed.[114]

Conclusion

Our study provides important insights into the relationship between thyroid function and ACS in an Indian population. While we found a substantial prevalence of thyroid dysfunction (34.6%), particularly subclinical hypothyroidism (15.7%) and equal proportions of overt hypothyroidism and hyperthyroidism (9.4% each), its relationship with various clinical and angiographic parameters was less pronounced than reported in some previous studies. These findings suggest that the interaction between thyroid function and acute coronary events is complex and may be influenced by multiple

factors including ethnicity, comorbidities, and timing of presentation.

The results underscore the importance of thyroid function evaluation in ACS patients while highlighting the need for larger, prospective studies to better understand the clinical implications of thyroid dysfunction in this setting. Future research should focus on whether targeted management of thyroid dysfunction could improve outcomes in ACS patients.[115]

CONCLUSION

This study has provided valuable insights into the relationship between thyroid hormone profiles and acute coronary syndrome in the Indian population. Through our analysis of 127 patients, we have demonstrated that thyroid dysfunction, particularly hypothyroidism, is a common finding among ACS patients, with 26% of the study population showing abnormal thyroid profiles. Of these, 15.7% were subclinical hypothyroidism, 9.4 % were overt hypothyroidism , and 9.4% were hyperthyroidism .

The demographic profile of our study population revealed a predominance of male patients (73.2%) and a higher incidence in the age group of 51-70 years (60.6%). The most common presenting symptoms were chest pain and dyspnea, reflecting the classical presentation of ACS. The high prevalence of comorbidities, particularly the combination of diabetes mellitus and hypertension (39.4%), highlights the significant burden of cardiovascular risk factors in this population.

Coronary angiographic findings showed that single vessel disease was the most common pattern (44.9%), followed by double vessel disease (32.3%) and triple vessel disease (22%). Importantly, our study found no significant association between thyroid status and the severity of coronary artery disease, suggesting that thyroid dysfunction may not directly influence the extent of coronary atherosclerosis in the acute setting.

The lack of significant associations between thyroid profile and various clinical parameters, including ECG findings, cardiac biomarkers, and lipid profile, suggests that thyroid dysfunction may not substantially modify the acute presentation of ACS. However, this does not diminish the importance of thyroid function evaluation in ACS patients, as thyroid hormones play a crucial role in cardiovascular function and may influence long-term outcomes.

These findings emphasize the need for routine thyroid function testing in ACS patients, particularly in populations with a high prevalence of thyroid disorders. Further prospective studies with larger sample sizes and longer follow-up periods are warranted to better understand the prognostic implications of thyroid dysfunction in ACS and to evaluate whether targeted management of thyroid disorders could improve cardiovascular outcomes.

SUMMARY

INTRODUCTION

Thyroid hormones play a crucial role in cardiovascular function, yet their relationship with acute coronary syndrome (ACS) remains incompletely understood. This study aimed to evaluate thyroid hormone profiles in patients with ACS and investigate potential associations with clinical presentation, angiographic findings, and cardiac biomarkers.

AIMS AND OBJECTIVES

Objective:

1. To study of thyroid hormone profile in patients with Acute coronary syndrome

MATERIAL AND METHODS

A cross-sectional study was conducted on 127 patients diagnosed with ACS. Thyroid function tests, lipid profiles, cardiac biomarkers, and coronary angiography were performed. Patients were categorized based on thyroid status, and associations with various clinical and laboratory parameters were analyzed.

RESULTS

SUMMARY OF RESULTS

This cross-sectional study analyzed 127 patients with acute coronary syndrome (ACS) to investigate the relationship between thyroid function and ACS manifestations. Key findings include:

Demographics

- **Age Distribution**: Majority of patients (60.6%) were between 51-70 years, followed by 71-90 years (26.8%), 30-50 years (11.8%), and >90 years (0.8%).
- **Gender Distribution**: Male predominance (73.2%) compared to females (26.8%).

Clinical Presentation

- **Symptoms**: Chest pain (74.8%) and dyspnea (72.4%) were the most common presenting symptoms, followed by giddiness (11%), easy fatiguability (3.9%), and palpitation (1.6%).
- **Comorbidities**: Combined diabetes mellitus and hypertension was most prevalent (39.4%), followed by hypertension alone (19.7%) and diabetes mellitus alone (11.8%).
- **Personal Habits**: Tobacco chewing (14.2%) was the most common habit, followed by tobacco chewing with alcohol (8.6%), smoking with alcohol (5.5%), smoking alone (4.72%), and alcohol consumption alone (3.93%).

Vital Signs

- Mean pulse rate: 86.9 ± 21.4 beats/min
- Mean respiratory rate: 19.44 ± 3.1 breaths/min
- Mean blood pressure: 130.9/79.8 ± 26.9/13.9 mmHg

Thyroid Function

- **Thyroid Status**: Normal thyroid function in 65.4% of patients, while 34.6% had thyroid dysfunction.
- **Types of Dysfunction**: Subclinical hypothyroidism (15.7%), overt hypothyroidism (9.4%), and hyperthyroidism (9.4%).
- **Thyroid Parameters**: Mean T3: 0.93 ± 0.24, mean T4: 7.76 ± 1.9, and mean TSH: 5.0 ± 9.7.

Lipid Profile

- Mean total cholesterol: 183.5 ± 23.3 mg/dl
- Mean LDL: 126.02 ± 20.7 mg/dl
- Mean HDL: 38.1 ± 8.1 mg/dl
- Mean VLDL: 36.18 ± 8.1 mg/dl
- Mean triglycerides: 180.9 ± 40.8 mg/dl

ECG Findings

- STEMI (39.4%) was the most common presentation, followed by unstable angina (28.3%), NSTEMI (23.6%), and old MI (8.7%).
- No significant association between thyroid status and ECG findings (p=0.93).

Coronary Angiography

- Single vessel disease was most common (44.9%), followed by double vessel disease (32.3%), triple vessel disease (22%), and mild CAD (0.8%).

- No statistically significant association between thyroid status and coronary artery disease severity (p=0.23), although certain patterns were observed:
    - Single vessel disease was more prevalent in overt hypothyroidism (66.7%)
    - Double vessel disease was more common in hyperthyroidism (58.3%)
    - No patients with overt hypothyroidism had triple vessel disease

Cardiac Function and Biomarkers

- No significant association between thyroid status and ejection fraction (p=0.75).
- No significant difference in Troponin T levels across thyroid status groups (p=0.17).

Association Analyses

- No significant association between thyroid status and age (p=0.32).
- No significant association between thyroid status and lipid parameters:
    - Total cholesterol >200 mg/dl (p=0.88)
    - LDL >100 mg/dl (p=0.71)
    - HDL <50 mg/dl (p=0.53)
    - Triglycerides >150 mg/dl (p=0.44)
    - VLDL >30 mg/dl (p=0.47)

These findings demonstrate a high prevalence of thyroid dysfunction in ACS patients but show limited associations between thyroid status and various clinical, angiographic, and laboratory parameters in the acute setting.

CONCLUSION:

While thyroid dysfunction, particularly hypothyroidism, is common among ACS patients, it does not significantly influence the acute presentation or severity of coronary artery disease. These findings suggest the need for routine thyroid function evaluation in ACS patients, though the immediate clinical implications may be limited.

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ANNEXURE – I - INFORMED CONSENT FORM

"STUDY OF THYROID HORMONE PROFILE IN PATIENTS WITH ACUTE CORONARY SYNDROME IN TERTIARY CARE CENTRE BELAGAVI- 1 YEAR CROSS SECTIONAL STUDY"

Name of Student/Principal Investigator:

Name of Guide/Co Investigators:

MD (GENERAL MEDICINE)
PROFESSOR AND UNIT CHIEF
MEDICINE G UNIT
J.N.MEDICAL COLLEGE, BELAGAVI

Introduction and Need for the study: Acute coronary syndrome has considerable effect on thyroid gland homeostasis with consequences in terms of morbidity and mortality.[1]Alteration in the level of serum thyroid hormone level has been described in several non thyroidal systemic illnesses including acute heart diseases .[1]Several studies have reported "Euthyroid sick syndrome" in patients admitted with acute coronary syndrome the normal feedback control of the thyroid homeostasis is changed [2]."Euthyroid Sick Syndrome" is characterized by decreased serum T3 and /or free T3, increased serum reverse T3(rT3), plus normal serum TSH, T4, and free T4 [2].Patients belonging to the STEMI group showed early elevations, in addition to higher mean reverse T3(rT3) and lower mean T3 and free T3 levels[3]. This syndrome has been reported to be found in severe chronic heart failure[3], in acute myocardial infarction and as a rapidly emerging phenomenon during open-heart surgery[3] .

Additionally hypothyroidism is emerging as a risk for coronary artery disease [4].Evaluation of thyroid hormone plasma levels has been done in a number of studies

in patients presenting with Acute Coronary Syndrome and findings compared between Unstable Angina/Non-ST elevation MI (UA/NSTEMI) and ST Elevation acute MI (STEMI) groups [4]. Some studies have reported association of greater hormonal changes with more severe cardiac events (STEMI and Death) and patients with complications. Implications of "Euthyroid Sick Syndrome" in terms of morbidity can lead to brady[5]Impairment in Thyroid harmone in ACS patients may lead to brady or Tachyarrhythmia leading to increased mortality [5]The previous studies done on Thyroid profile in ACS didnot show any result supportive of thyroid harmone effect on Arrythmias post ACS[6] So in order say that the mortality in ACS could be due to thyroid harmone derangement in ACS , this study is necessary[6]

Explanation of procedure: All the patients fullfilling the inclusion criteria amd willing to participate in the study are included

- The protocol will be explained and the informed conesnt is taken
- Venous blood samples are collected at the time of admission for biochemical analysis and weight and height of the patients are checked
- History and detailed clinical examination performed as per the working Performa.

Group 1: Unstable angina/ non STEMI

Cases showing ST depression / T wave inversion with normal or elevated cardiac markers. At the time of presentation, patients with UA and NSTEMI can be indistinguishable and therefore are considered together in this guideline [20].

UA is defined as angina pectoris or equivalent ischemic discomfort with at least one of three features:

a. It occurs at rest (or with minimal exertion), usually lasting >10 minutes.

b. It is severe and of new onset (i.e., within the prior 4-6 weeks); and/or

c. It occurs with a crescendo pattern (i.e., distinctly more severe, prolonged, or frequent than previously).

The diagnosis of NSTEMI is established if a patient with the clinical features of UA develops evidence of myocardial necrosis, as reflected in elevated cardiac biomarkers [21].

Group 2: ST elevation MI

Symptoms of myocardial ischemia in association with electrocardiographic (ECG) ST elevation and release of biomarkers of myocardial necrosis. New ST elevation at the J point in at least 2 contiguous leads of 2mm (0.2mV) in men or 1.5mm (0.15mV) in women in leads V2-V3 and/or of 1mm

- (0.1mV) in other contiguous chest leads or the limb leads [22]. The 12- lead ECG is a pivotal diagnostic tool. Level of serum cardiac biomarkers CK-MB and Troponin are elevated [23].

Withdrawal from participation in the study: Participation in this study in voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will get any benefits by participating in this study. As early diagnosis will be helpful to determine further course of treatment**.** The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Cost of investigations done during the course of study will be paid by the principal investigator**.**(Strike out which is not applicable)

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact:

If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights

CONSENT STATEMENT

I am making a voluntary decision to participate in the study **““Study of thyroid hormone profile in patients with Acute coronary syndrome in tertiary care centre, belagavi- 1 year cross sectional study”**

My signature below indicates that I have decided to participate and I have read the information provided above or the information provided.. above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE – II - CASE PROFORMA

"STUDY OF THYROID HORMONE PROFILE IN PATIENTS WITH ACUTE CORONARY SYNDROME"

CASE NO:

NAME:

AGE/SEX:

IP NO.:

ADDRESS:

COMPLAINTS AT PRESENTATION:

HISTORY OF PRESENT ILLNESS :

PAST HISTORY :

Co-morbidities

DM	HTN	IHD	CKD	CLD	EPILEPSY	MALIGNANCY	OTHERS

Drug history:

Personal history:

Family history:

PHYSICAL EXAMINATION:

GENERAL CONDITION-

PALLOR		CLUBBING	
ICTERUS		PEDAL EDEMA	
CYANOSIS		LYMPHADENOPATHY	

VITALS:

TEMPERATURE		R.R	
P.R		B.P	

SYSTEMIC EXAMINATION:

R. S:

C.V.S:

C.N.S:

P.A:

INVESTIGATIONS:

ECG-

Troponin T-

Thyroid profile -

T3	T4	TSH

2D ECHO/CAG-

EF-

RWMA-

OUTCOME-

ANNEXURE – III

MASTER CHART

Case No	Name	Age-years	Sex	Place of Residence	IP Number	Chief complaints	Co-morbidities	Addictive Habits	Pulse rate-bpm	Respiratory rate-cpm	SBP	DBP	ECG	Troponin T	ZD-ECHO-LVEF %	CAG	T3	T4	TSH	Total cholesterol	LDL	HDL	triglycerides	VLDL
1	FATIMA VALISAB SHAIKH	61	FEMALE	GOA	10055346	DYSPNOEA ON EXRETION-1 YEAR, CHEST PAIN-2 DAYS	DIABETIC, HYPERTENSIVE	NIL	94	20	110	70	NSTEMI	0	50	TVD-LCX,LAD, RCA	1	7.9	3.64	208	129	40	178	36
2	SHIVALINGAPPA BASAPPA SANGOLLI	69	MALE	ANGOL	10055002	CHEST PAIN AND BREATHLESSNESS- 6 HOURS	DIABETIC, HYPERTENSIVE	TOBACCO CHEWER	106	28	100	70	IWMI	9.8	45	TVD-LCX,LAD, RCA	1	3.8	0.28	171	117	38	262	52
3	MASABI MEHABOOB BHATE	61	MALE	KHANAPUR	10055279	CHEST PAIN AND BREATHLESSNESS- 12 HOURS	HYPERTENSIVE	NIL	120	24	120	80	AWMI	10	35	DVD-LAD , LCX	1	7.4	2.6	185	142	45	186	37
4	RAMAPRAKASH RAMSHKAL YADAV	59	MALE	UDYAMBAG	10055333	CHEST PAIN -2 HOURS	DIABETIC, HYPERTENSIVE	TOBACCO CHEWER,ALCOHOLIC, SMOKER	104	18	140	100	NSTEMI	0.1	**45**	DVD-LCX,LAD	1	9.6	1.42	215	123	35	203	41
5	GAJANAN MAHADEV PATIL	62	MALE	BELAGAVI	10055317	CHEST PAIN, BREATHLESSNESS SINCE 8 HOURS	NIL	SMOKER, TOBACCO CHEWER	64	20	110	70	IWMI	6	50	SVD-LAD	1	8.9	6.03	160	124	39	193	39
6	SANGEETA VILAS SALVI	59	FEMALE	NANAWADI	10055698	BREATHLESSNESS, PALPITATION-3 DAYS	DIABETIC, HYPERTENSIVE	NIL	80	18	130	70	NSTEMI	0.2	60	SVD-LCX	1	6	1.5	172	115	32	223	45
7	MANISH KANTILAL PAREKH	59	MALE	BELAGAVI	10055920	CHEST PAIN, BREATHLESSNESS-4 DAYS	DIABETIC, HYPERTENSIVE	OCCASIONAL ALCOHOLIC	74	24	120	70	NSTEMI	3.9	35	TVD-LCX,RCA, LAD	1	6	2.29	159	97	36	162	32
8	NITIN NAGENDRA SANGAVAKAR	45	MALE	INDALGA	10056029	CHEST PAIN , BREATHLESSNESS - 1WEEK	NIL	SMOKER, ALCOHOLIC	80	20	110	70	IWMI	4.5	45	SVD-RCA	1	8.6	1.39	174	130	22	206	41
9	GANGAVVA SIDDAPPA KANNUR	61	FEMALE	BAILHONGAL	10056685	CHEST PAIN,BREATHLESSNESS-8 DAYS	NIL	NIL	120	20	180	100	ALWMI	1.4	40	SVD-LAD	1	11	4.11	170	122	31	206	41
10	AVVAKKA BALAPPA SOLLAPURI	85	FEMALE	DANDELI	10057290	HEADACHE, VOMITING- 4 HOURS	DIABETIC,HYPERTENSIVE	NIL	110	24	170	100	NSTEMI	0.2	45	SVD-LAD	1	5.8	0.45	194	182	42	230	46
11	SURESH BANDU KHOT	67	MALE	BELAGAVI	10058140	CHESTPAIN, BREATHLESSNESS-1 DAY	HYPERTENSIVE	NIL	54	20	90	60	EVOLVED IWMI +	1.2	45	SVD-RCA	1	4.7	3.53	138	126	48	234	47
12	NANDA VASANTRAO PARADESHI	63	FEMALE	SHINDOLI	10058925	CHEST PAIN, BREATHLESSNESS-7 DAYS	DIABETIC, HYPERTENSIVE	TOBACCO CHEWER	64	18	160	100	EVOLVED AWMI	0.2	60	SVD-LAD	1	8.9	9.55	165	111	39	219	44
13	VIKRANT KRISHNA PURI	40	MALE	BELAGAVI	10064163	CHEST PAIN, BREATHLESSNESS- 1 HOUR	NIL	SMOKER, ALCOHOLIC	80	16	126	90	IWMI	0.7	50	SVD-RCA	1	11	3.31	203	112	40	203	41
14	GULNARBEGUM ABDUL MUNAF QAZI	61	FEMALE	GOKAK	10066187	CHEST PAIN, BREATHLESSNESS- 2 DAYS	DIABETIC, HYPERTENSIVE	NIL	82	18	120	70	NORMAL SINUS	0	60	SVD-LAD	1	8.8	1.07	173	132	42	144	29
15	SHANKAR GANGAPPA BANAKAR	75	MALE	BELAGAVI	10066477	LEFT ARM PAIN, BREATHLESSNESS - 1 DAY	DIABETIC, HYPERTENSIVE	SMOKER	58	19	110	70	EVOLVED IWMI +	0.8	45	MILD CAD	1	8.2	2.92	199	165	40	297	59
16	UMAMAHESHWARA GOPALKRISHNA HEGDE	59	MALE	YALLAPURA	10062113	CHEST PAIN 2 MONTHS	DIABETIC HYPERTENSIVE	NIL	88	18	160	90	NORMALSINUS RHYTHM-	0	60	SVD-RCA	1	5.8	1.14	194	125	37	180	36
17	SULOCHANA SHANKAR ABBAI	72	FEMALE	RAMADURGA	10064477	CHEST PAIN, BREATHLESSNESS - 10 DAYS	HYPERTENSIVE	NIL	90	18	130	90	EVOLVED PWMI	4.3	35	TVD-RCA, LCX, LAD	1	5.6	8.95	161	140	32	107	21
18	GODABAI MAHADEV BAGANE	91	MALE	CHIKKODI	10063668	CHEST APIN , BREATHLESSNESS- 6 MONTHS	DIABETIC , HYPERTENSIVE	NIL	88	18	150	80	EVOLVED AWMI	0.3	35	DVD-RCA, LCX	1	13	4.88	171	145	35	209	42
19	IRAYYA NAGAYYA HIREMATH	52	MALE	BELAGAVI	10068903	CHEST PAIN , GIDDINESS-2 DAYS	NIL	ALCOHOLIC, TOBACCO CHEWER	90	18	100	60	IWMI + PWMI +	6	45	DVD-RCA,LCX	1	7.6	4.08	223	149	38	195	39
20	NINGAPPA BAPANNA JAKKANNAVAR	56	MALE	BAILHONGAL	10068738	CHEST PAIN, BREATHLESSNESS- 15 DAYS	DIABETIC	ALCOHOLIC, TOBACCO CHEWER	76	18	120	80	NSTEMI	0.8	60	SVD-LAD	0	0.4	101.03	195	103	39	186	37
21	APPASAHEB VISHWANATH BALEKUNDRI	66	MALE	BELAGAVI	10072423	CHEST PAIN, BACKPAIN - 8 HOURS	DIABETIC, HYPERTENSIVE	NIL	70	18	160	90	IWMI + PWMI +	0.1	45	DVD-LCX, RCA	1	5.6	2.64	212	149	37	149	30
22	CHENNAMMA HANAMAPPA BENAKATTI	67	FEMALE	YARAGATTI	10056992	BREATHLESSNESS, LOOSE STOOLS - 2 DAYS	DIABETIC , HYPERTENSIVE	TOBACCO CHEWER	92	24	100	70	EVOLVED AWMI +Qrbbb	4.2	35	DVD-LAD, HIGH OM	1	11	24.9	172	94	40	200	40
23	PARAMESHWAR RAMACHANDRA DURGANNAVAR	48	MALE	GOKAK	10062233	CHEST PAIN, GIDDINESS- 10 DAYS	NIL	OCCASIONAL ALCOHOLIC	74	24	110	70	NSTEMI	0.9	60	SVD-LAD	1	5.9	1.15	220	141	37	164	33

24	UMA VEERABHADRAPPA SANTOJI	63	FEMALE	DHARWAD	10064621	CHEST PAIN, BREATHLESSNESS - 1MONTH	DIABETIC	NIL	78	16	130	90	POOR R WAVE PROGRESSIO	0	35	TVD-RCA, LCX, LAD	1	10	2.29	176	156	33	246	49
25	NAGAPPA NINGAPPA BANNUR	81	MALE	BAILHONGAL	10067373	CHEST PAIN, PALPITATION-2 HOURS	HYPERTENSIVE	NIL	130	18	100	60	AWMI	1	40	SVD-LAD	1	9.4	3.7	187	147	41	167	33
26	HULAGAPPA BASAPPA TEERTHA	64	MALE	YADGIRI	10069032	CHESTPAIN , GIDDINESS - 2 DAYS	NIL	TOBACCO CHEWER	68	16	180	100	NORMAL SINUS RHYTHM-UA	0	60	SVD-LCX	1	8.2	0.75	152	117	34	145	29
27	GANGAVVA HANAMANTH MADAR	55	FEMALE	RAMADURGA	10070349	CHEST PAIN, EASY FATIGUABILITY- 8 DAYS	DIABETIC	NIL	68	18	110	60	NORMAL SINUS RHYTHM-UA	0	60	SVD-LAD	1	7.8	0.72	171	150	30	185	37
28	SATISH BABURAO HURLIKOPPA	65	MALE	KUDAL	10072196	COUGH, BREATHLESSNESS - 15 DAYS	NIL	TOBACCO CHEWER, OCCASIONAL ALCOHOLIC	118	28	110	70	LBBB-UA	0	25	SVD-LAD	1	9.4	2.56	168	141	44	210	42
29	MANGAL NINGAPPA SUTAR	41	MALE	HINDALGA	10072845	CHEST PAIN, LOOSE STOOLS - 2 DAYS	DIABETIC, HYPERTENSIVE	NIL	88	18	120	70	LBBB-UA	0	60	SVD-LAD	1	6.9	11.68	182	134	50	139	28
30	SATISH JYOTIBA WAJANTRI	37	MALE	BELAGAVI	10072468	CHEST PAIN, BREATHLESSNESS- 6 HOURS	NIL	TOBACCO CHEWER, OCCASIONAL ALCOHOLIC	80	18	150	90	AWMI	0.8	45	SVD-LAD	1	9.7	2.18	207	116	40	143	29
31	NARAYAN VITHOBA MORYA	62	MALE	KHANAPUR	10073983	BREATHLESSNESS - 4 DAYS	HYPERTENSIVE	NIL	92	20	170	100	EVOLVED AWMI	0.6	35	DVD-LAD, LCX	1	8.4	0.64	192	119	33	141	28
32	NAMDEO NAGESH PATIL	63	MALE	BELAGAVI	10073942	CHEST PAIN - 8 DAYS	HYPERTENSIVE , EPILEPTIC	NIL	56	22	110	70	POOR R WAVE PROGRESSIO N-UA	0.1	60	DVD-LAD, RCA	1	7.1	5.77	239	147	34	124	25
33	KASHAVVA MALLAPPA KUMBAR	76	FEMALE	HUKKERI	10058384	BREATHLESSNESS- 1 DAY	DIABETIC	TOBACCO CHEWER	110	24	80	60	NSTEMI	0.8	25	TVD-RCA, LCX, LAD	1	5.8	1.19	173	159	44	92	18
34	BHARAT LAXMAN DEVAJI	52	MALE	BELAGAVI	10060258	CHEST APIN, BREATHLESSNESS- 2DAYS	NIL	ALCOHOLIC, TOBACCO CHEWER	120	28	180	90	LVH-UA	0	30	SVD-LAD	1	6.9	3.4	157	125	43	157	31
35	DASTAGEER ISAK IPERI	72	MALE	BELAGAVI	10067034	CHEST PAIN, BREATHLESSNESS- 4 DAYS	NIL	SMOKER	78	16	140	90	NSTEMI	0.1	50	TVD-LCX, RCA, LAD	1	9.9	1.09	177	141	43	136	27
36	DILSHADBEGUM MEHABOOBKHAN SOUDAGAR	85	FEMALE	BELAGAVI	10071414	BREATHLESSNESS, GIDDINESS 2 DAYS	DIABETIC , HYPERTENSIVE	NIL	78	18	140	90	NSTEMI	0.3	60	SVD-RCA	0	3.7	1.89	192	116	51	139	28
37	SHARANBASAV NAGBHUSHAN HIREMATH	39	MALE	NAGAR HAVELI	10071835	BREATHLESSSNESS- 1 DAY	DIABETIC, HYPERTENSIVE	ALCOHOLIC , TOBACCCO CHEWER	98	20	130	80	IWMI	3.5	50	DVD-RCA, LCX	1	7.6	5.71	199	144	41	169	34
38	BASAVANNEYYA SATAYYA HIREMATH	64	MALE	YARAGATTI	10072415	CHEST PAIN , EASY FATIGUABILITY-7 DAYS	NIL	NIL	70	16	110	70	NSTEMI	0.4	60	SVD-LAD	1	5.4	14.2	134	143	41	211	42
39	KALLAPPA TUKARAM KADOLI	65	MALE	BELAGAVI	10072375	CHEST PAIN, BREATHLESSNESS- 12 HOURS	NIL	NIL	60	16	150	80	NORMAL SINUS RHYTHM-UA	0	50	DVD-LAD, RCA	1	9.5	0.36	163	125	39	165	33
40	AVAKKA RAMAGOUDA PATIL	72	MALE	CHIKKODI	10072996	CHEST PAIN , BREATHLESSNESS- 1 MONTH	HYPERTENSIVE	NIL	88	16	170	90	NSTEMI	0.7	60	TVD-RCA,LCX, LAD	1	6.6	8.36	187	99	36	169	34
41	BALAVVA KALLAPPA TALAWAR	75	FEMALE	RAMADURGA	10074235	CHEST PAIN- 5 HOURS	DIABETIC, HYPERTENSIVE	NIL	92	22	170	90	NORMAL SINUS RHYTHM-UA	0	60	SVD-LCX	1	9	3.37	203	102	30	208	42
42	KANTA SHARAD ROKADE	73	FEMALE	KARWAR	10073950	BREATHLESSNESS-2MONTHS	HYPERTENSIVE	NIL	68	21	160	80	UA	0	60	TVD-RCA,LCX, LAD	1	8.6	3.18	194	142	44	180	36
43	VISHNU PATIL KRISHNA	65	MALE	BALEKUNDRI	10074114	CHEST PAIN-4 DAYS	NIL	NIL	68	18	150	90	NORMAL SINUS RHYTHM-UA	0.1	60	SVD-LAD	1	7.2	26.5	230	116	34	183	37
44	KAMALA SHIVARAI GORAV	56	FEMALE	GOKAK	10073729	BREATHLESSNESS-8 HOURS	DIABETIC , HYPERTENSIVE	NIL	58	18	130	80	NSTEMI	0.8	45	SVD-LAD	1	6.9	15.6	157	101	48	180	36

45	PARATAVVA HANAMANTAPPA BANKAPUR	78	FEMALE	LAKSHMESHWAR	10066774	CHEST PAIN, BREATHLESSNESS- 20 DAYS	DIABETIC, HYPERTENSIVE	NIL	108	18	120	80	NSTEMI	0.2	40	SVD-LCX	1	9.4	5.19	181	104	27	132	26
46	CHANDRAKANT PANDURANG SHET	63	MALE	VADAGAON	10065738	CHEST PAIN, GIDDINESS-5 DAYS	NIL	NIL	66	16	110	70	EVOLVED IWMI	0.7	30	TVD-RCA,LCX. LAD	1	9.6	0.58	204	147	45	242	48
47	VITTAL BHIMAPPA DEVAMANE	46	MALE	BELAGAVI	10068789	CHEST PAIN, BREATHLESSNESS-1 MONTH	HYPERTENSIVE	ALCOHOLIC	78	18	160	100	SYMMETRICAL T INVERSION IN V4-V6-UA	0	60	SVD-LAD	1	7.4	3.73	174	133	40	165	33
48	SHABBIR MEHABUSAB SHEKH	58	MALE	SAVADATTI	10070496	CHEST PAIN -6 HOURS	HYPERTENSIVE	ALCOHOLIC, SMOKER	66	16	100	60	EVOVLVED IWMI	2.6	50	SVD-RCA	1	9.8	9.64	156	158	37	195	39
49	PARVATI MOTAPPA MADAR	72	MALE	KHANAPUR	10070451	CHEST APIN, BREATHLESSNESS-14 HOURS	HYPERTENSIVE	NIL	96	18	120	80	EVOLVED IWMI	0.9	55	SVD-LCX	1	7.4	5.36	207	122	48	204	41
50	BASALINGAYYYA GURUBASAYYA UNANABADIMATH	68	MALE	HUKKERI	10071462	CHEST APIN, BREATHLESSNESS -6 MONTHS	DIABETIC	TOBACCO CHEWER	104	16	120	70	SINUS TACHYCARDIA -UA	0	50	DVD-LCX,RCA	1	8.1	2.39	229	146	22	147	29
51	SHIVALING APPANNA MADIWALAR	57	MALE	GOKAK	10072147	CHEST PAIN, BREATHLESSNESS- 3 DAYS	DIABETIC, HYPERTENSIVE	NIL	90	16	160	100	NSTEMI	0.6	45	TVD-RCA,LCX. LAD	1	7.3	0.9	230	127	19	163	33
52	NANDAKISHORE CHANNAPPA MODAGI	63	MALE	SAVADATTI	10074898	CHEST PAIN, BREATHLESSNESS- 7 DAYS	HYPERTENSIVE	NIL	90	18	130	70	EVOLVED IWMI	0.8	45	TVD-LAD,RCA, LCX	1	6.2	2.74	223	126	33	172	34
53	SUNITA SHARANAPPA CHALWADI	61	FEMALE	VIJAYAPURA	10074918	CHEST PAIN, BREATHLESSNESS-8 DAYS	DIABETIC, HYPERTENSIVE	NIL	56	19	90	70	NSTEMI	0.8	60	SVD-LAD	1	7.9	1.72	170	123	38	180	36
54	BASIRABI MAKTUMSAHEB GHODANAI	82	FEMALE	BELAGAVI	10074701	CHEST PAIN, BREATHLESSNESS- 6 HOURS	NIL	NIL	128	28	160	90	PWMI	1.9	45	DVD-LCX,RCA	1	11	12.07	158	109	48	175	35
55	TAMMANNI LAXMAN KORE	65	MALE	RAIBAG	10073892	CHEST PAIN, GIDDINESS-6 HOURS	DIABETIC,HYPERTENSIVE	TOBACCO CHEWER	60	20	120	80	NSTEMI	1.1	50	TVD-LAD,OM, RCA	1	7.4	1.64	190	135	48	129	26
56	LAKSHMI GUNDU MARANAHOLKAR	61	FEMALE	CHANDGAD	10073349	BREATHLESSNESS, EASY FATIGUABILITY-2 MONTHS	HYPERTENSIVE	NIL	82	20	110	70	NSTEMI	0.7	60	SVD-LAD	1	6.7	1.3	204	113	33	162	32
57	NAGAMMACHINNAYYA YANDURI	70	MALE	KANGRALI	10073474	CHEST PAIN, BREATHLESSNESS- 10 HOURS	DIABETIC, HYPERTENSIVE	NIL	146	28	200	120	EVOLVED PWMI	0.2	40	DVD-LCX, RCA	1	8.4	2.83	138	107	33	190	38
58	SHREYOUNSH MALASARJI PATIL	64	MALE	BELAGAVI	10062089	CHEST PAIN, BREATHLESSNESS -2 HOURS	NIL	NIL	80	24	130	90	AWMI	1.4	40	TVD-LAD,RCA. LCX	1	6.7	8.75	185	101	26	284	57
59	MALLAPPA FAKIRA TALWAR	69	MALE	KANGRALI	10062333	CHEST PAIN -5 HOURS	EPILEPSY	TOBACCO CHEWER	84	20	100	60	AF-OLD IWMI	0	40	DVD-LAD,RCA	1	7.5	3.81	171	134	39	183	37
60	PUNDALIK RAMACHANDRA PATIL	55	MALE	YELLUR	10064684	CHEST PAIN, BREATHLESSNESS-1 DAY	NIL	TOBACCO CHEWER	70	18	100	60	IWMI	0.4	50	SVD-RCA	1	5.5	2.89	201	143	31	137	27
61	FAIZULLAKHAN AHMEDKHAN MARIYAR	64	MALE	BELAGAVI	10066443	BREATHLESSNESS-1 DAY	DIABETIC	NIL	130	20	100	70	EVOLVED IWMI	0.9	25	TVD-LAD,RCA, LCX	1	9.4	0.36	204	122	49	138	28
62	RAJANI LAXMANRAO KULKARNI	74	MALE	BELAGAVI	10067033	CHEST PAIN, BREATHLESSNESS-8 DAYS	DIABETIC	NIL	94	20	130	80	EVOLVED IWMI	1.3	35	TVD-LAD, RCA,LCX	1	7.1	5.31	164	107	20	132	26
63	CHANDRAKANT SHETYAPPA KORE	60	MALE	KOLHAPUR	10066691	CHEST PAIN, EASY FATIGUABILITY-8 DAYS	DIABETIC	NIL	72	18	120	70	OLD AWMI	0	60	DVD-RCA,LAD	1	8.6	5.31	194	135	31	256	51
64	VASUDEV BASAPPA KAMKAR	74	MALE	SULEBAVI	10069225	CHEST PAIN, BREATHLESSNESS- 8 HOURS	DIABETIC, HYPERTENSIVE	TOBACCO CHEWER	130	20	110	70	NSTEMI	0.5	45	TVD-LAD,RCA, LCX	1	7.7	6.74	145	152	40	256	51
65	BASAVANNEPPA LAXMAN BALLEGADDI	64	MALE	BAILHONGAL	10069569	CHEST PAIN, BREATHLESSNESS-10 DAYS	NIL	ALCOHOLIC TOBACCO CHEWER	88	18	140	100	NSTEMI	0.9	25	SVD-LCX	1	6.3	1.58	212	138	42	210	42
66	PRAKASH JAYRAM BADAVE	76	MALE	VADAGAON	10069890	CHEST PAIN, BREATHLESSNESS-3 DAYS	NIL	NIL	68	19	180	100	IWMI	0.4	50	DVD-LCX, RCA	1	7.8	0.36	175	138	44	210	42
67	BALU SADASHIV KADAPURE	59	MALE	CHIKKODI	10070197	CHEST PAIN, GIDDINESS- 20 DAYS	DIABETIC	TOBACCO CHEWER	60	18	100	70	NORMAL SINUS RHYTHM-UA	0	60	DVD-LAD,RCA	1	8.1	1.6	150	126	43	149	30

68	ASHOK SHRIPATRAO PATIL	66	MALE	BELAGAVI	10068975	CHEST PAIN, BREATHLESSNESS-8 DAYS	NIL	NIL	96	20	110	60	OLD AWMI	0.5	25	SVD-LAD	1	7.9	15.8	193	136	14	225	45
69	SHAKUNTHALA VISHNU SURYAVANSHI	76	FEMALE	GANESHPUR	10069137	CHEST PAIN,BREATHLESSNESS-5 DAYS	NIL	TOBACCO CHEWER	110	18	110	70	NSTEMI	1	35	DVD-RCA,LCX	2	11	6.84	210	107	42	147	29
70	MALATI MADHUSOODHAN NAIK	77	FEMALE	BELAGAVI	10071927	CHEST PAIN, GIDDINESS-5 DAYS	DIABETIC,HYPER TENSIVE	NIL	80	14	140	70	NSTEMI	0.8	45	TVD-LAD,RCA, LCX	1	8.3	7.38	161	138	45	171	34
71	CHANDRAKANT TAVANAPPA AMANAJI	65	MALE	BELAGAVI	10070690	CHEST PAIN, BREATHLESSNESS-2 HOURS	DIABETIC, HYPERTENSIVE	TOBACCO CHEWER, SMOKER	120	20	140	80	NSTEMI	0.1	40	SVD-LAD	1	10	1.46	179	120	37	208	42
72	SHEKHAR NARASAPPA KALLIMANI	50	MALE	BELAGAVI	10070507	CHEST PAIN, BREATHLESSNESS-3 HOURS	NIL	TOBACCO CHEWER, ALCOHOLIC	110	28	180	100	OLD AWMI	0	35	SVD-LAD	1	7.5	2.63	159	122	38	189	38
73	MAHIROON RAFIQAHMED MAKANDER	55	FEMALE	CHADACHAN	10071953	BREATHLESSNESS-1 DAY	DIABETIC, HYPERTENSIVE	NIL	90	18	110	70	NORMAL SINUS RHYTHM-UA	0	60	SVD-LCX	1	8.4	8.56	147	128	44	162	32
74	MAHADEV SIDDAGOUDA PATIL	52	MALE	KAGAWAD	10071681	CHEST PAIN, BREATHLESSNESS- 8 DAYS	NIL	CHRONIC SMOKER, ALCOHOLIC	56	20	110	70	EVOLVED IWMI + RVMI, CHB	2.6	55	DVD-RCA, LCX	1	7.4	0.82	189	157	29	172	34
75	PARASHURAM MARUTI JALKAR	61	MALE	LKOLHAPUR	10071747	BREATHLESSNESS -15 DAYS	DIABETIC	NIL	66	18	140	80	NORMAL SINUS RHYTHM-UA	0	50	SVD-LAD	1	9.7	17.4	166	111	38	182	36
76	MOHAN BASAVANNEPPA KADOLI	78	MALE	KHANAPUR	10071883	NIL	NIL	NIL	60	16	110	80	EVOLVED IWMI	0.3	50	DVD-RCA, LCX	1	8.8	1.87	189	97	48	244	49
77	RAMAJAN AHMADSAB DESAI	63	MALE	GOKAK	10071830	CHEST PAIN-5 DAYS	DIABETIC, HYPERTENSIVE	NIL	64	16	120	70	LVH-UA	0	60	DVD-LAD,RCX	1	7.3	2.75	127	103	26	163	33
78	BALKRISHNA NAGAPPA MIRAJKAR	70	MALE	BAILHONGAL	10073485	CHEST PAIN, BREATHLESSNESS-10 HOURS	HYPERTENSIVE	TOBACCO CHEWER, SMOKER	80	18	160	90	IWMI + PWMI + RVMI	7.4	45	DVD-RCA,LAD	1	6.7	2.96	142	109	49	178	36
79	SAINATH RAMACHANDRA PATIL	59	MALE	BELAGAVI	10072994	CHEST PAIN, BREATHLESSNESS- 1 DAY	NIL	SMOKER	80	18	130	80	AWMI	6.5	60	SVD-LAD	1	7.3	2.7	195	79	36	179	36
80	UDAYAKUMAR NARASIMHA RAO	71	MALE	BELAGAVI	10072492	CHEST PAIN SINCE 3 HOURS	DIABETIC	SMOKER, ALCOHOLIC	66	20	110	80	IWMI + RVMI	9.8	45	TVD-LAD,RCA, LCX	1	4.4	0.95	174	110	45	167	33
81	SUSHEELA GOPAL MEGERI	68	FEMALE	KANGRALI	10066734	CHEST PAIN, BREATHLESSNESS-2 DAYS	DIABETIC, HYPERTENSIVE	NIL	80	16	150	80	NSTEMI	0.8	60	SVD-RCA	1	5.6	4.9	182	149	43	163	33
82	BASAVARAJ BHIMARAYAPPA RACHANNAVAR	86	MALE	KARDIGUDDI	10074594	CHEST PAIN, BREATHLESSNESS-4 DAYS	CVA	NIL	80	18	130	80	LBBB-UA	0	60	TVD-LAD,LCX, RCA	1	5.2	2.52	207	120	31	176	35
83	ARJUN SONU LAKHE	66	MALE	BELAGAVI	10074810	GIDDINESS - 3DAYS	NIL	NIL	68	18	110	70	0LD IWMI	0.2	50	TVD-RCA,LCX, LAD	1	7.9	1.45	198	118	57	181	36
84	REKHA RAMACHANDRA BASTAWADKAR	60	FEMALE	BELAGAVI	10074197	CHEST PAIN, BREATHLESSNESS-15 DAYS	DIABETIC, HYPERTENSIVE	NIL	90	18	150	90	EVOLVED AWMI	0,01	45	TVD-RCA,LAD, LCX	1	12	1.82	189	119	38	237	47
85	YASHODHA NANJUNDAPPA	66	FEMALE	DAVANAGERE	10074491	CHEST PAIN -20 DAYS	HYPERTENSIVE	NIL	72	20	110	70	POOR R WAVE PROGRESSIO N -UA	0.1	45	TVD-LAD,RCA, LCX	1	9.6	7.58	157	138	49	181	36
86	BABU MADIVALAPPA ULAVI	60	MALE	BAILHONGAL	10074563	BREATHLESSNESS, COUGH-15 DAYS	HYPERTENSIVE	NIL	60	20	120	70	NSTEMI	1.2	33	DVD-RCA,LCX	1	6.7	3.42	165	140	35	224	45
87	DATTA GANAPATRAO JADHAV	76	MALE	BELAGAVI	10074634	BREATHLESSNESS-3 DAYS	HYPERTENSIVE	NIL	96	24	100	60	AWMI	17	40	DVD-LAD,LCX	1	7.6	0.35	159	179	47	192	38
88	RAMESH BASAVANNI KADAGAVI	49	MALE	HUKKERI	10074240	CHEST PAIN, BREATHLESSNESS-3 DAYS	DIABETIC, HYPERTENSIVE	NIL	64	20	180	110	NORMAL SINUS RHYTHM-UA	0	60	SVD-LAD	1	7.3	7.12	217	96	40	112	22
89	MAHADEVAPPA SHANKARAPPA PATTAR	74	MALE	BAILHONGAL	10073954	BREATHLESSNESS-15 DAYS	DIABETIC, HYPERTENSIVE	NIL	94	21	180	80	OLD IWMI	0.1	40	TVD-LAD,RCA. LCX	1	6.7	7.32	188	121	47	161	32

90	JAGANNATH BABU BADIGER	70	MALE	RAIBAG	10073703	CHEST PAIN -8 HOURS	DIABETIC	NIL	70	20	130	70	ASMI	0.3	45	TVD-LAD,RCA, LCX	1	5.2	0.53	184	140	46	204	41
91	VISHNU RAMACHANDRA KAMBALE	50	MALE	KAGWAD	10057941	CHEST PAIN, BREATHLESSNESS-2 DAYS	NIL	TOBACCO CHEWER, ALCOHOLIC	88	18	106	70	AWMI	0.9	45	SVD-LAD	1	9.1	3.2	184	131	32	201	40
92	ARATHI SURESH HULLIKOPPI	53	FEMALE	CHIKKODI	10064031	BREATHLESSNES, COUGH -2 DAYS	DIABETIC, HYPERTENSIVE	NIL	78	18	130	80	IWMI	0.6	55	DVD-LCX,RCA	1	15	1.88	174	171	45	121	24
93	KHAJA MOHIDDIN PATEL	73	MALE	BELAGAVI	10066673	CHEST PAIN, GIDDINESSS - 8 DAYS	DIABETIC,HYPER TENSIVE	SMOKER	96	18	110	70	NORMAL SINUS RHYTHM-UA	0	60	SVD-LAD	1	6.8	0.89	198	122	48	170	34
94	ASHADEVI VISHNU SASAVE	74	FEMALE	BELAGAVI	10066697	CHEST PAIN, BREATHLESSNESS-2 DAYS	DIABETIC, HYPERTENSIVE	NIL	80	16	110	80	NSTEMI	0.3	45	DVD-LAD, RCA	2	8.4	0.03	224	125	31	274	55
95	YALLAMMA NARAYAN KATARE	53	FEMALE	BELAGAVI	10067241	CHEST PAIN, BREATHLESSNESS-2 DAYS	DIABETIC, HYPERTENSIVE	NIL	100	18	160	100	NSTEMI	0.3	60	SVD-LAD	1	6	2.03	159	116	43	191	38
96	KESHAVSING BALUSING HAJARI	61	MALE	BELAGAVI	10071912	BREATHLESSNESS-2 DAYS	DIABETIC, HYPERTENSIVE	NIL	60	16	130	80	NORMAL SINUS RHYTHM-UA	0	60	SVD-RCA	1	5.2	4.04	179	98	37	162	32
97	MAHADEV RAYAPPA HUCCHELI	66	MALE	GOKAK	10069858	BREATHLESSNESS-15 DAYS	NIL	TOBACCO CHEWER, ALCOHOLIC	88	20	110	70	ST DEPRESSION IN V3-V6-UA	0	45	DVD-LCX, OM	1	7.4	0.3	167	84	46	159	32
98	HAMUDABI HAMEED MUJAWAR	82	FEMALE	HUKKERI	10070117	CHEST PAIN, BREATHLESSNESS-1 DAY	HYPERTENSIVE	NIL	84	24	120	80	ASWMI	0.2	40	DVD-LAD,LCX	1	9.7	9.03	172	93	17	198	40
99	SUBHASH SIDAGOUDA PATIL	59	MALE	HUKKERI	10071655	BREATHLESSNESS-10 DAYS	DIABETIC, HYPERTENSIVE	ALCOHOLIC	126	19	80	60	OLD AWMI	1.4	30	SVD-LAD	1	8.2	4.94	185	123	38	167	33
100	SURESH DUNDAPPA ANKLI	74	MALE	GADHINGLAJ	10070155	BREATHLESSNESS-7 DAYS	DIABETIC, HYPERTENSIVE	SMOKER,ALCOHOLIC	84	20	100	60	TRIFASCICULAR BLOCK	0.9	30	SVD-LAD	1	7.4	3.07	191	134	47	154	31
101	SURESH GUNDU KOLI	70	MALE	BELAGAVI	10074706	BREATHLESSNESS-2 DAYS	DIABETIC, HYPERTENSIVE	NIL	114	30	90	60	EVOLVED ASMI	0.1	35	DVD-LAD,RCA	1	6.6	0.48	185	131	30	114	23
102	LAXMIBAI SUBRAO MALI	64	FEMALE	HUKKERI	10064023	CHEST PAIN, BREATHLESSNESS- 1 DAY	NIL	NIL	70	17	100	70	IWMI	2.3	40	SVD-RCA	1	10	0.43	170	119	40	136	27
103	MAYAPPA ADIVEPPA TAHASHILDAR	62	MALE	GOKAK	10062499	CHEST PAIN, BREATHLESSNESS 1 HOUR	DIABETIC, HYPERTENSIVE	NIL	86	18	136	80	NORMAL SINUS RHYTHM-UA	0	50	DVD-LAD, OM	1	6.9	0.97	190	109	38	243	49
104	NANDA SHANKAR PATIL	76	MALE	BELAGAVI	10061782	BREATHLESSNESS-2 HOURS	DIABETIC, HYPERTENSIVE	NIL	140	30	120	70	OLD AWMI	0.2	40	DVD-LAD,LCX	1	11	0.35	178	130	50	143	29
105	RAGHUNATH PANDURANG HATTI	70	MALE	KAGAWAD	10060296	CHEST PAIN, BREATHLESSNESS 6 HOURS	HYPERTENSIVE	TOBACCO CHEWER, ALCOHOLIC	58	20	150	100	ASMI	0.1	45	SVD-LAD	1	6.7	3.01	166	102	54	144	29
106	VISHWANATH BASAWARAJ MUTTUR	38	MALE	BELAGAVI	10059959	CHEST PAIN, BREATHLESSNESS-7 DAYS	DIABETIC,HYPER TENSIVE	ALCOHOLIC	80	20	100	60	POOR R WAVE PROGRESSION-UA	0	60	SVD-LAD	1	9.7	11.43	200	128	37	219	44
107	AMRUTH PREMNATH GATTIN	38	MALE	HOSPET	10073939	CHEST PAIN, GIDDINESS -3 DAYS	DIABETIC	ALCOHOLIC,SMOKER	108	21	180	110	NSTEMI	0.6	60	DVD-LCX,LAD	1	9.9	1.19	212	84	46	238	48
108	SAVITRI MAHADEV GAJABARE	52	FEMALE	HUKKERI	10074630	CHEST PAIN -15 DAYS	DIABETIC	NIL	102	19	140	90	EVOLVED AWMI	0.3	45	DVD-LAD,RCA	1	5.3	5.47	179	158	30	194	39
109	SHEKAVVA MAHADEV JINARALI	67	FEMALE	BELAGAVI	10074517	CHEST PAIN, BREATHLESSNESS-15 DAYS	DIABETIC,HYPER TENSIVE	NIL	90	20	170	90	NSTEMI	1.8	60	TVD-RCA,LAD, LCX	1	9.3	0	194	149	30	148	30
110	GUNDAPPA BASAPPA	63	MALE	LINGASUR	10074710	BREATHLESSNESS-2 DAYS	HYPERTENSIVE	SMOKER	104	20	130	80	AWMI WITH Qrbbb	0.3	40	SVD-LAD	1	6.1	1.57	222	103	56	93	19
111	ANNAPURNA MALLESHAPPA TORANAGATTI	64	FEMALE	BAILHONGAL	10065971	CHEST PAIN, BREATHLESSNESS-6 HOURS	DIABETIC,HYPER TENSIVE	NIL	72	18	140	80	AWMI	0.2	45	SVD-LAD	1	8.3	2.73	167	154	26	166	33
112	LALITA MALAKAJAPPA GADATARANAVAR	74	MALE	BAILHONGAL	10067295	CHEST PAIN,BREATHLESSNESS-4DAYS	HYPERTENSIVE	NIL	60	16	110	70	PREMATURE VENTRICULAR COMPLEXES-UNSTABLE ANGINA	0	60	SVD-LCX	1	6.2	7.64	215	103	29	190	38

113	SIDDARAM VEERABHADRAPPA NOOLI	71	MALE	HUKKERI	10069393	CHEST PAIN, BREATHLESSNESS-30 DAYS	DIABETIC, HYPERTENSIVE	NIL	84	18	150	90	NORMAL SINUS RHYTHM-UA	0	60	SVD-LAD	1	7.6	1.12	244	91	32	151	30
114	ANNAGOUDA SHIVARAYAPPA NERLI	65	MALE	HUKKERI	10069030	CHEST PAIN, EASY FATIGUABILITY-5 DAYS	NIL	NIL	120	20	200	110	ST DEPRESSION IN LEAD II,III,Avf	0.1	60	DVD-LCX, LAD	1	7.4	3.1	183	147	23	211	42
115	KALLAPPA BHAVU TAVADARE	77	MALE	CHIKKODI	10069307	CHEST PAIN-10 DAYS	HYPERTENSIVE	TOBACCO CHEWER	130	18	150	80	OLD AWMI	0.1	35	SVD-LAD	1	6.9	1.28	129	138	40	152	30
116	MAHANTESH BASAPPA HANNIKERI	49	MALE	BAILHONGAL	10069512	CHEST PAIN,BREATHLESSNESS-4 HOURS	DIABETIC,HYPER TENSIVE	SMOKER	96	19	130	90	IWMI + RVMI	0.9	50	SVD-RCA	1	8.3	0.95	197	146	41	236	47
117	SAROJA MAHADEV MANE	68	FEMALE	ATHANI	10069551	CHEST PAIN, BREATHLESSNESS-10 DAYS	HYPERTENSIVE	NIL	68	20	120	70	OLD AWMI	0	55	SVD-LAD	1	8.3	1.94	192	107	46	222	44
118	SHIVAJI SADASHIV PATIL	73	MALE	CHIKKODI	10069760	BREATHLESSNESS-20 DAYS	DIABETIC, HYPERTENSIVE	TOBACCO CHEWER	94	18	150	90	ST DEPRESSION IN V3-V6-UA	0.1	60	DVD-LAD,LCX	1	7.1	3.73	184	139	42	229	46
119	SHEKANNA MALLAPPA KOTAMBARI	65	MALE	MUTAGA	10070296	CHEST PAIN -4 HOURS	DIABETIC, HYPERTENSIVE	NIL	86	18	130	70	EVOLVED IWMI	0	60	DVD-LAD,RCA	1	6.1	3.66	189	130	32	222	44
120	MAHAVEER MALLAPPA VEERAGOUDAR	78	MALE	KANBARGI	10071793	BREATHLESSNESS,GIDDINESS-30 DAYS	HYPERTENSIVE	TOBACCO CHEWER	70	19	90	60	OLD IWMI	0.5	45	DVD-LAD,RCA	1	11	9.46	198	146	47	157	31
121	MALLIKARJUN BASAPPA MURGOD	40	MALE	MOGADA	10071025	CHEST PAIN, BREATHLESSNESS-4 DAYS	HYPERTENSION	TOBACCO CHEWER	141	18	200	130	NSTEMI	3.7	45	DVD-LAD,LCX	1	12	0.85	181	82	38	202	40
122	KARIMSAHEB MAKTUMSAHEB GUGIHAL	72	MALE	BILGI	10071859	CHEST PAIN, BREATHLESSNESS-1WEEK	DIABETIC, HYPERTENSIVE	NIL	68	20	150	80	EVOLVED AWMI	0.4	50	TVD-LAD,LCX, RCA	1	8.2	3.88	165	122	25	181	36
123	NASIRKHAN BABUSAB KUDARI	39	MALE	GARAG	10070800	CHEST PAIN, BREATHLESSNESS-30 DAYS	HYPERTENSIVE	NIL	100	18	140	90	NORMAL SINUSRHYTHM-UA	0	60	DVD-LAD,LCX	1	4.4	18.1	205	79	29	161	32
124	VEERANNA CHANNABASAYYA VASTRAD	62	MALE	CHIKKODI	10072826	BREATHLESSNESS , GIDDINESS- 4 DAYS	DIABETIC	NIL	108	16	130	90	POOR R WAVE PROGRESSION-UA	5.5	30	TVD-LCX,LAD, RCA	1	7.9	1.13	152	129	38	253	51
125	ANNAPPA SANGAPPA MALAYII	68	MALE	BHENDIGERI	10073674	CHEST PAIN, GIDDINESS-30 DAYS	NIL	TOBACCO CHEWER	55	18	140	90	BRADYCARDIA-UA	0	60	SVD-LAD	1	6.6	1.39	185	158	42	142	28
126	ANNAPPA HANUMANT KARRATI	70	MALE	CHIKKODI	10072822	CHEST PAIN-1 DAY	PSORIASIS	NIL	120	20	120	80	AWMI	3.6	30	DVD-LAD,RCA	1	3.9	12.51	180	111	39	70	14
127	NARAYAN BHARAMANA KADAM	59	MALE	SULGA	10072877	BREATHLESS-5 DAYS	NIL	TOBACCO CHEWER	110	18	150	80	SINUS TACHYCARIA-UA	0	45	DVD-LAD,RCA	1	6.5	3.7	200	116	24	118	24