“PROGNOSTIC VALUE OF LEUKO-GLYCEMIC INDEX IN ACUTE MYOCARDIAL INFARCTION”.

By:

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Under The Guidance Of

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DR PARIMI SAMBASIVA RAO

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ABBREVIATIONS

Glossary	Abbreviations
ACS	Acute Coronary Syndrome
CHD	Coronary Heart Disease
STEMI	ST Elevation of Myocardial Infarction
MI	Myocardial Infarction
LGI	Leuko-glycemic Index
HF	Heart failure
NPS	Natriuretic Peptide System
CAD	Coranary artery disease
PCI	Percutaneous Coronary Intervention
NSTEMI	Non ST Elevation of Myocardial Infarction
RAAS	Renin-Angiotensin-Aldosterone System
NPS	Natriuretic Peptide System
AHA	American Heart Association
CABG	Coronary Artery Bypass Graft
LMWHs	Low-molecular-Weight Heparins
UFH	Unfractionated heparin
LDL	low-density lipoprotein
SBP	Systolic Blood Pressure
HR	Heart rate
FBS	Fasting Blood Glucose
VT/VF	ventricular tachycardia/ ventricular fibrillation
KK	killip –kimball

ABSTRACT

BACKGROUND:

India has the world's largest Acute Coronary Syndrome (ACS) burden. In India the rising incidence of ACS may be linked to lifestyle changes, western style of food habits, increasing the prevalence of diabetes mellitus and expected genetic factors. Number of circumstances associated with sudden, reduced reduced blood flow to the heart is termed as ACS. Heart attack (Myocardial Infarction [MI]) is one such condition — when cell death outcomes in damaged or destroyed heart tissue. Even though ACS does not cause cell death, decreased blood flow changes the way the heart purposes and is a symptom of a high risk of heart attack. ACS often causes severe pain or distress in the chest. It is a medical emergency that needs timely analysis and care. Leukocytosis and hyperglycemia are correlated with a worse short-term prediction in patients with ACS, but their new relationship, called Leuko-glycemic Index (LGI), has been narrowly evaluated. LGI is a simple, low cost tool that allows re-stratification of non-diabetic people with low Thrombolysis in Myocardial Infarction (TIMI) score, at higher risk of death or severe Heart Failure (HF).

OBJECTIVES:

1) *To measure leukocyte count (mm^3), and fasting blood glucose levels (mg/dL) in patients with acute MI.*
2) *To calculate LGI and correlate with adverse cardiovascular outcomes.*

MATERIALS AND METHODS*:*

Source of data: The present study will be carried out with patients attending the services of the department of General Medicine at RL Jalappa hospital, Kolar.

Sample Size: Based on area under the curve (Receiver Operating Characteristic curve) for leuko-glycemic index with discriminatory capacity of 0.66, power of 80% and confidence interval of 95% a sample size of 100 will be taken [calculated sample size:leuko-glycemic index >1000, 50 samples, and <1000, 50 samples].

***Study design**: A Comparative two group clinical study*

Statistical Methods: MEDCAL software package was used for statistical analysis. Student´s t-test will be used as test of significance. Continuous data will be represented as mean and standard deviation. Independent t-test will be used as test of significance to identify the mean difference. p value <0.05 will be considered as statistically significant.

RESULTS:

This Comparative two group clinical study was conducted in 104 patients of both sexes, *total 104 patients included in the study, 52 patients had included in the Group LGI<1000 and 52 patients had included in the Group LGI>1000.* Of total, 31 (29.8%) had <100 in fasting blood glucose level in both the groups, 36 (34.6%) had 100-126 in in both the groups and 37 (35.6%) had >126 in both the groups of fasting blood levels. *Of total, 36 (34.6%) had ST elevation in I, aVL, 45 (43.3%) had ST elevation in II, III, aVF, v1-v3 and 12 (11.5%) had St elevation in v1 to v4 and 11 (10.6%) had St elevation in v3- v6 in Group LGI<1000 and Group LGI>1000. P=0.581.* Of total, 46 (44.2%) had dyslipidemia, 37 (35.6%) had diabetics and 46 (44.2%) had hypertension in Group LGI<1000 and Group LGI>1000. Of total, 86 (82.7%) had no prior MI and 18 (17.3%) had prior MI in Group LGI<1000 and Group LGI>1000. P=0.120. Of total, 94 (90.4%) had no H/O PCI and 10 (9.6%) had H/O PCI in Group LGI<1000 and Group LGI>1000.

CONCLUSION:

Leukocytosis and hyperglycemia are associated with worse short-term prognosis in patients with ACS, but their new relationship, called leuko-glycemic index, has been scarcely evaluated.

The leuko-glycemic index is a simple, low cost tool allowing re-stratification of non-diabetic patients with low TIMI score, at higher risk of death or severe HF.

INTRODUCTION

INTRODUCTION

India has the world's largest Acute Coronary Syndrome (ACS) burden. In India the rising incidence of ACS may be linked to lifestyle changes, western style of food habits, increasing the prevalence of diabetes mellitus and expected genetic factors.[1] Number of circumstances associated with sudden, reduced reduced blood flow to the heart is termed as ACS. Heart attack (Myocardial Infarction [MI]) is one such condition — when cell death outcomes in damaged or destroyed heart tissue. Even though ACS does not cause cell death, decreased blood flow changes the way the heart purposes and is a symptom of a high risk of heart attack. ACS often causes severe pain or distress in the chest. It is a medical emergency that needs timely analysis and care.[2]

ACS is the type of Coronary Heart Disease (CHD) and is usually the effect of plaque disruption in coronary arteries (atherosclerosis). Common risk factors of the disease include smoking, hypertension, diabetes, hyperlipidemia, sex, physical inactivity, family obesity and reduced nutritional practices. Cocaine abuse can leads to vasospasm, too. A family history of early MI (55 years of age) is also a high-risk factor.[3]

Contrary to decline in the prevalence of numerous risk factors such as hypertension, hypercholesterolemia, smoking and diabetes is an increasing health burden in the Western world. Due to proatherosclerotic, proinflammatory and prothrombotic states associated with diabetes, diabetic patients with ACS is at high risk for subsequent cardiovascular events.[4]

Hyperglycemia is mutual and is associated with significantly greater mortality rates in patients hospitalized with ACS. Despite the fact that several studies have reported this association, a hyperglycemia relic underestimated as a risk factor, and is often not treated in ACS patients. This is mostly due to the limitations of previous studies and the remaining

critical gaps in our understanding of the relationship between hyperglycemia and lowly outcomes.[5]

The signs and symptoms of ACS usually start abruptly. They include:[2]

- Chest pain (angina) or discomfort
- Pain spreading from the chest to the arms, shoulders, upper abdomen, back, neck or jaw
- Nausea or vomiting
- Indigestion
- Shortness of breath
- Sudden, heavy sweating
- Lightheadedness, dizziness
- Unusual or unexplained fatigue
- Feeling restless

Diagnostic tests include:[2]

- Electrocardiogram (ECG)
- Blood tests
- Coronary angiogram
- Echocardiogram
- Myocardial perfusion imaging
- Computerized Tomography (CT) angiogram
- Stress test

Diagnostic valuation of acute chest pain has enhanced in the recent years with advances in the sensitivity and precision of cardiac troponin assays, new biomarkers, improved imaging modalities, and release of new clinical decision algorithms. This advancement has allowed physicians to diagnose or rule-out acute MI earlier after the early patient presentation, typically in emergency department settings, which may assist in prompt initiation of evidence-based treatments, investigation of alternative diagnoses for chest pain, or discharge, and permit better utilization of healthcare resources.[6]

Leukocytosis and hyperglycemia are correlated with a worse short-term prediction in patients with ACS, but their new relationship, called Leuko-glycemic Index (LGI), has been narrowly evaluated.[7]

LGI is a simple, low cost tool that allows re-stratification of non-diabetic people with low Thrombolysis in Myocardial Infarction (TIMI) score, at higher risk of death or severe Heart Failure (HF).[7]

AIMS & OBJECTIVES

OBJECTIVES:

1) To measure leukocyte count (mm^3), and fasting blood glucose levels (mg/dL) in patients with acute MI.
2) To calculate LGI and correlate with adverse cardiovascular outcomes.

REVIEW OF LITERATURE

REVIEW OF LITERATURE:

The coronary arteries emerge from the sinuses of Valsalva, past the origin of the aortic root. The Right Coronary Artery (RCA), start from the anterior aortic sinus, transports blood to the right atrium, right ventricle, sinoatrial node, Atrioventricular (AV) node, and select portions of the left ventricle. The Left Coronary Artery (LCA) begins from the left posterior aortic sinus and quickly bifurcates into the Left Circumflex Artery (LCX) and Left Anterior Descending (LAD) artery, which deliver blood to the left atrium and left ventricle. There is substantial overlap in these blood supplies due to the existence of collateral vessels and variant anatomy, but these intricacies are beyond the possibility of the current debate.[8]

Coronary arteries can be generally classified as epicardial and intramuscular vessels. The former are larger and more superficial, and act as blood flow conductors. The latter are small and course within the myocardium; their various branches and arterioles provide have higher resistance, but more fine-tuned directive of blood flow.[8]

In most tissues, the blood flow peaks through ventricular systole due to improved pressure in the aorta and its distal branches. Flow through the coronary vessels, however, looks to be paradoxical and peaks during ventricular diastole. This uncommon pattern is a result of the external compression of the coronary vessels by the myocardial tissue during systole. Notably, this compression may be severe sufficient to reverse coronary flow, predominantly in the intramuscular vessels of the thicker left ventricle. When the ventricles relax during the diastole, the coronary vessels are no longer compressed, and normal blood flow is resumed. Due to this design of blood flow, tachycardia and the resulting decrease in time spent in diastole may decrease the effectiveness of myocardial perfusion.[8]

At rest, about 60% to 70% of oxygen is take out from blood in the coronary arteries. This level of oxygen extraction is a evidence to the high metabolic activity of the myocardium. This also highlights the importance of increasing overall coronary flow during times of increased demand for myocardial oxygen.[8]

Myocardial oxygen demand can rise manifold depending on ventricular rate, contractility, and pressures. Due to the high baseline oxygen intake of the myocardium, increased oxygen extraction provides only a limited buffer capacity. Most of this claim must be fulfilled by increased coronary flow, the mechanisms of which are only partly understood. Current evidence indicates a multifactorial model of coronary regulation. Downstream oxygen intake metabolites, such as carbon dioxide, are considered to be the key determinant of coronary flow under physiological conditions at rest. In the meantime, localized hypoxia, along with the resulting release of vasodilatory substances, is likely to contribute coronary vasodilation during numerous physiologic and pathophysiologic stages of uneven oxygen supply and demand.[8]

Local hypoxemia and hypercarbia have been established to correlate with coronary vasodilation at the most basic level. Measurements of coronary venous pO2 and pCO2, however, show little, if any, change during states of physiologically amplified demand (i.e., exercise). This indicates that substitute factors must contribute to coronary regulation under normal conditions to avoid hypoxemia and hypercarbia. In fact, several studies have shown that the concentrations of both oxygen and carbon dioxide are insufficient in explaining the majority of the total extent of coronary vasodilation in response to increased oxygen demand. Although localized hypoxemia and hypercarbia are expected to play a role in coronary regulation during pathophysiologic conditions, it is not yet clear whether an intermediary molecule is involved in the process.[8]

ACS refers to any condition attributed to coronary artery obstruction that decreases blood flow to the heart, including unstable angina and MI.[9]

Although not included under the umbrella of ACS, stable angina is categorized within ischemic heart disease. Temporary discomfort is caused by a persistent flow-restricting lesion within the coronary artery, which occurs when the demand for blood supplied to myocardium is increased, for e.g., during physical exertion or emotional stress.[9]

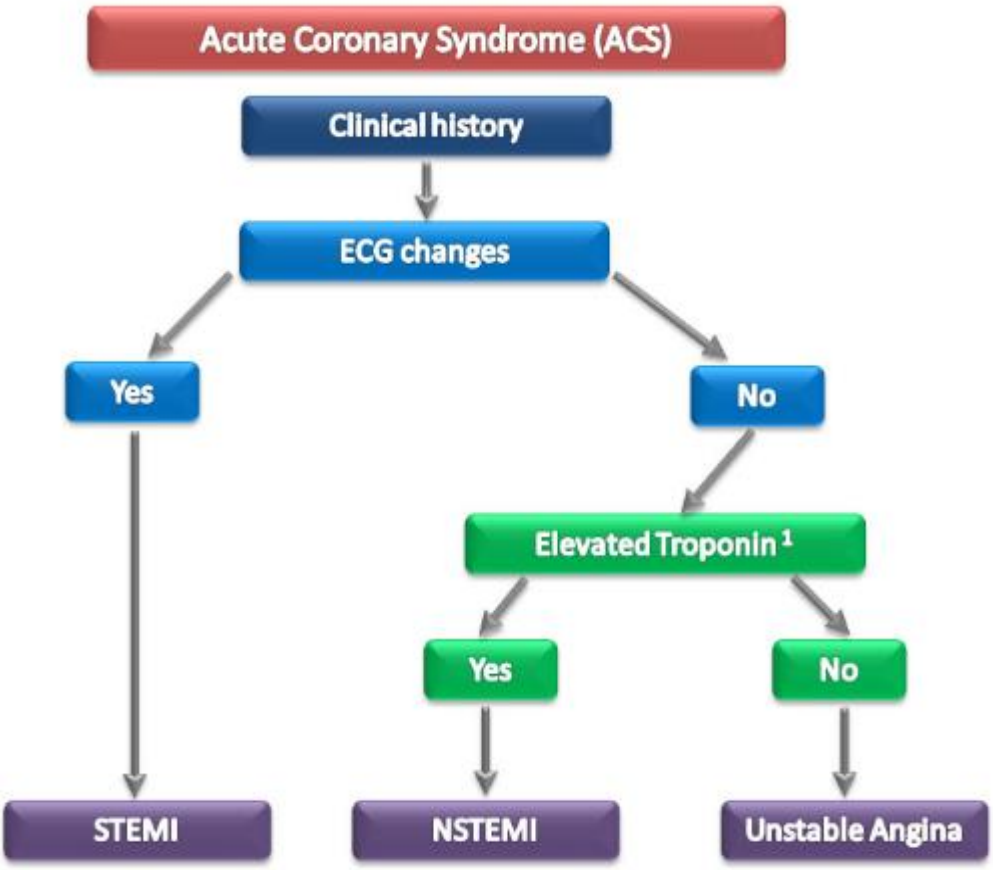


Figure 1: Defining ACS

ST Elevation of Myocardial Infarction (STEMI) refers to an elevation of ST segment on a patient's ECG who generally have cardiac biomarkers (i.e., elevated troponin level) suggesting necrosis of the heart muscle. The pathway for clinical management focuses on early reperfusion therapy either by thrombolytic therapy or revascularization with Percutaneous Coronary Intervention (PCI).[9]

Non ST Segment of Elevation Acute Coronary Syndrome (NSTEACS) refers to symptomatic individuals whose first ECG demonstrates no ST elevation. Risk stratification occurs until a diagnosis of the NSTEMI or unstable angina has been made. These patients are differentiated as low, intermediate or high risk in terms of adverse outcome.[9]

Non ST Elevation of Myocardial Infarction (NSTEMI) refers to those people who have not had ST elevation on their ECG, but, subsequent cardiac biomarkers are elevated. Up to the 50% of patients diagnosed as NSTEMI have an ECG that is normal or shows only minor changes.[9]

Unstable angina is an accelerated pattern of the angina with or without ECG changes. NSTEMI is characterized by an absence of elevated cardiac biomarkers.[9]

Symptoms:[2]

The signs and symptoms of ACS usually start abruptly. They include:

- Chest pain (angina) often described as aching, pressure, tightness or burning
- Pain spreading from the chest
- Nausea or vomiting
- Indigestion
- Shortness of breath
- Sudden, heavy sweating
- Lightheadedness, dizziness
- Unusual or unexplained fatigue

- Feeling restless

Chest pain is the most common symptom. Nevertheless, signs and symptoms may differ significantly depending on the individual age, sex and other medical conditions.

Causes:

ACS usually outcomes from the buildup of fatty deposits in and on the walls of coronary arteries, the blood vessels supplying oxygen and nutrients to the heart muscles. When a plaque breaks or splits, a blood clot forms. This clot blocks the flow of blood to the muscles of the heart. If the supply of oxygen to the cells is too low, cells of the heart muscles may die. The death of cells resulting in muscle tissue damage is a heart attack (MI). Even when there is no cell death, the decline in oxygen still results in heart muscles that dont work the way they should. This change might be temporary or permanent. If ACS doesn't results in cell death, it is called unstable angina.[2]

Risk factors:[2]

The risk factors for ACS are the same as those for other types of heart disease. ACS risk factors include:

- Aging
- High blood pressure
- High blood cholesterol
- Cigarette smoking
- Lack of physical activity
- Unhealthy diet
- Obesity or overweight
- Diabetes
- Family history of chest pain, heart disease or stroke

- History of high blood pressure, preeclampsia or diabetes during pregnancy

PATHOPHYSIOLOGY:

The underlying process in ACS is reduced blood flow to the part of heart muscles, which is typically due to plaque rupture and thrombus formation. At times, ACS might be due to vasospasm with or without underlying atherosclerosis. As a result, the blood flow to a part of heart musculature shrinks, resulting first in ischemia and infarction of that portion of the heart.[3]

Pathophysiology of ischemia:

Ischemia is caused by the reduced supply of blood and oxygen to myocardium and is usually caused by a restriction or occlusion of at least one coronary artery. The utmost common cause of ACS and sudden death is occlusion of a coronary artery due to disruption of atherosclerotic plaque with subsequent thrombus formation.[9]

In general, there are the two types of coronary vessel lesions:

Stenotic lesions are coupled with fibrous plaques containing collagen and calcium precipitates. These typically cause thickening and expansion of the vessel wall that clinically manifests as ischemia. Management is usually by local therapy and revascularization.[9]

Nonstenotic lesions are linked with plaque that has a core of lipid laden cells and a thin fibrous cap. These plaques often cause the most damage. They tend not to narrow the lumen of the vessel, but instead are more prone to sudden rupture leading to thrombus formation, which subsequently occludes the vessel. The clinical manifestation is infarction that is treated by drug therapy and lifestyle modification. A complete rupture of the fibrous cap of the plaque most commonly causes fatal MI. Myocardial cell death occurs within as little as 20 minutes of a result of prolonged ischemia. Absolute necrosis may occur within 2-4 hours.[9]

The course of plaque development and progression is extremely complex and involves the interplay of multiple pathological processes. Management targets rapid reperfusion to prevent avoidable cell death, and thereby maintaining myocardial function as best as possible.[9]

The choice of treatment is governed by multiple factors including the:[9]

- Underlying pathology
- Location of the blockage
- Number of vessels involved
- Residual cardiac function

Clinical features of ischemia:

Ischemic symptoms differ from individual to individual and may occur in a various ways. This may include but it is not limited to, discomfort in the chest, jaw, neck, back, scapular area, upper limbs or epigastric region. Symptoms may occur at a single location (e.g., chest only) or in various combinations (e.g., chest and upper limbs). They may also occur with exertion or at rest. The discomfort is usually nonmechanical in that it's not intensified or relieved with positional changes, and is often diffuse and not localized.[9]

MI is the endpoint of this ischemia, which causes heart tissue death secondary to lack of an adequate blood supply. In general, acute MI-related discomfort lasts at least 20 minutes and may be associated with breathlessness, syncope, diaphoresis, nausea and/or vomiting. Due to significant change in symptoms, MI may be misdiagnosed as a musculoskeletal, neurological, pulmonary, gastrointestinal, or psychological disorder. Occasionally, there might be no signs of MI due to the silent ischemia. Diabetics and those with previous MIs are at pointedly higher risk of silent ischemia.[9]

Pathophysiology of HF:

HF is the debilitating condition characterized by shortness of breath, fatigue and intolerance to exercise. HF is a medical condition that may arise from any structural or functional cardiac disorder that damages the ability of the ventricle to fill with or eject blood at rest or during physical activity. Variations to heart muscle tissue and function, neurohormonal changes, and vascular and skeletal muscle function changes are often referred to as the ―vicious cycle" of HF as illustrated in the figure below.[9]

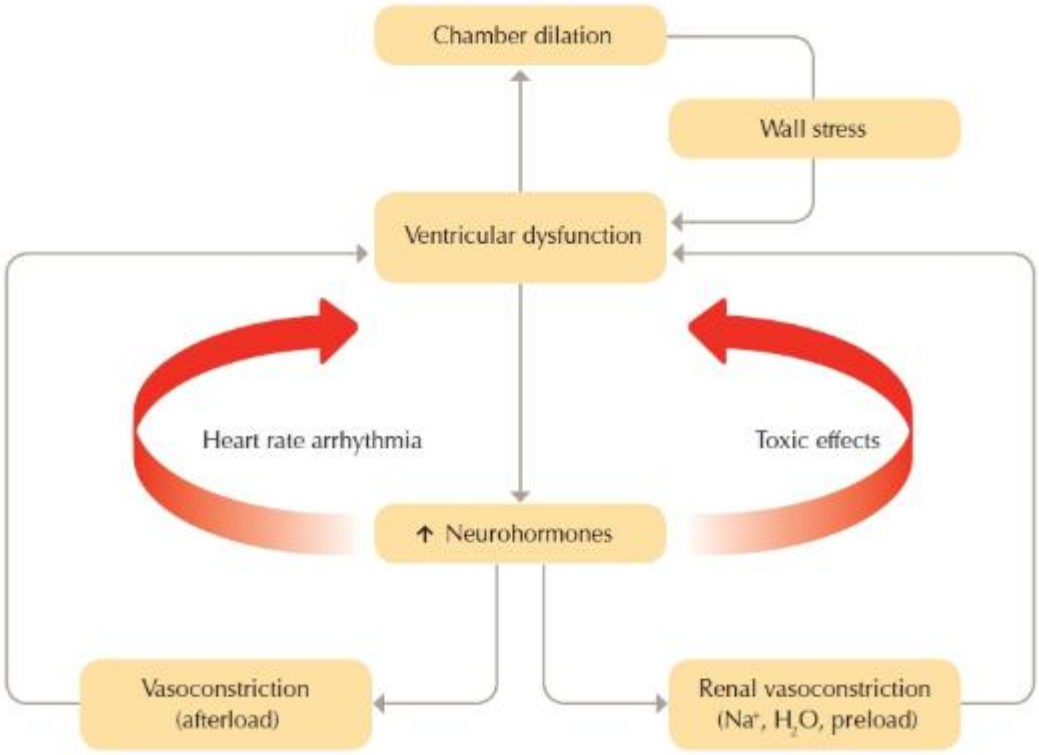


Figure 2: The "vicious cycle" of HF pathophysiology

The cycle starts with an insult to the myocardium. Neurohormonal compensatory activities are triggered when cardiac output declines.[9]

Heart Failure with Reduced Ejection Fraction (HFREF)

HF with decreased left ventricular systolic function resultant in poor cardiac output activates neurohormonal responses that might be dysfunctional or weak over the long term. Many HF medications alter neurohormonal responses to break the vicious cycle of HF.[9]

The neurohormonal systems involved in the cycle of HF include the Sympathetic Nervous System (SNS), Renin-Angiotensin-Aldosterone System (RAAS) and the Natriuretic Peptide System (NPS). SNS and RAAS work to increase circulating blood volume through vasoconstriction and sodium and water retention. Over the long term, SNS and RAAS over-activation may result in: Counterproductive neurohormonal adaptation as attempts to maintain arterial pressure advances deterioration in heart function.[9]

Raising levels of noradrenaline, angiotensin II and aldosterone, leading to chronic systemic and pulmonary vasoconstriction, cardiac hypertrophy and ischemia, edema, hyponatremia and susceptibility to cardiac arrhythmia.[9]

Systemic changes in renal, vascular and skeletal muscle function further deteriorate cardiac performance. Renal impairment triggered by a decline in cardiac performance and major alteration in regional blood flow leads to change in skeletal muscle function as seen by general muscle weakness.[9]

Deteriorating blood flow and reduced muscle function contribute to signs and symptoms such as salt and fluid retention, thirst and fatigue.[9]

Cardiac remodelling (changes in structure and shape) further decreases cardiac output.

The NPS plays a vital role in water and salt homeostasis by increasing sodium excretion in the urine. When the heart is under stress due to improved filling pressures, B-Natriuretic Peptide (BNP) and Atrial Natriuretic Peptide (ANP) are released. The NPS counter balances

vasoconstriction, caused by the SNS and RAAS, by promoting vasodilatation and diuresis and can be effective in the early phases of HF. Nevertheless, NPS activation cannot fully mitigate the adverse effects of over-activation of these systems over the long term.[9]

Heart Failure with Preserved Ejection Fraction (HFPEF)

The pathophysiology of HFPEF is less evident than in HFREF. In HFPEF, fluid overload and other symptoms do not significantly reduce left ventricular function. Current pathophysiological theories related to HFPEF include: Coronary microvascular inflammation, central arterial stiffening, abnormalities in contractile and chronotropic reserve, and abnormalities in skeletal muscle oxygen delivery.[9]

CLINICAL PRESENTATION

History and Physical:

The classic symptom of ACS is substernal chest pain, often categorized as squeezing or pressure-like feeling, radiating to the jaw and/or left arm. This classic presentation is not continually seen, and the complaint can be very vague and subtle, with main complaints often being difficulty breathing, lightheadedness, isolated jaw or left arm pain, nausea, epigastric pain, diaphoresis, and weakness. Female patients with diabetes and ageing are all associated with ACS presenting with vague symptoms. In such cases, a high level of suspicion is warranted. General distress and diaphoresis are often seen in the physical exam. Heart sounds are often normal. Sometimes, gallop and murmur can be heard. Lung exam is normal, while at times crackles may be heard pointing toward associated Congestive Heart Failure (CHF). Bilateral edema of the leg may be present indicating CHF. The rest of the systems are in general within the normal limits unless co-pathologies are present. The existence of the

abdominal tenderness to the palpation should make the provider think about other pathologies like pancreatitis and gastritis. The presence of irregular pulses warrants consideration of aortic dissection. The presence of the unilateral leg swelling should warrant work-up for pulmonary emboli. Hence, a careful physical exam is very important to rule out other life-threatening differentials.[3]

The primary purposes of physical examination are to check for any precipitating causes of myocardial ischemia and to evaluate the hemodynamic consequences of the acute ischemic event. Physical examination findings that suggest a large area of ischemia and high risk include diaphoresis; pale, cool skin; sinus tachycardia; a third or fourth heart sound; basilar rales and hypotension. Physical examination may also provide clues that may help in the determination of the differential diagnosis. For e.g., irregular pulses or a murmur of aortic regurgitation indicates possible aortic dissection, whereas a pericardial friction rub indicates acute pericarditis.[10]

ELECTROCARDIOGRAPHY

The ACC/AHA guidelines state that an experienced emergency physician should review the outcomes of 12-lead ECG within the 10 minutes of arrival in the ED of a patient with chest discomfort or other symptoms suggestive of ACS. The value of the ECG is 2-fold: To support a clinical diagnosis of ACS and to assist in risk stratification. Electrocardiography, however, has a number of limitations. For e.g., the posterior, lateral, and apical walls of left ventricle are not adequately represented. In addition, normal findings do not rule out the possibility of ACS.[10]

Findings on ECG associated with UA include ST-segment depression, transient ST-segment elevation, T-wave inversion, or some combination of these factors; depends on the severity of the clinical presentation, these findings are present in 30% to 50% of patients. The new ST-

segment deviation of only 0.05 mV is a significant and precise measure of ischemia and prognosis. T-wave inversion is sensitive for ischemia but is less specific, unless it is marked (≥0.3 mV). An ST-segment elevation of 0.1 mV or more, if present at least 2 contiguous leads, indicates acute MI in 90% of patients, as confirmed by serial measurements of cardiac biomarkers. It is vital to compare current and previous ECG results, as studies suggest that patients with no ECG changes are at a lower risk of complications than those with ECG changes.[10]

Because of the process of myocardial ischemia is quite complex and single 12-lead ECG offers only a snapshot view of the process, the ACC/AHA guidelines recommend that patients hospitalized for UA/NSTEMI undergo serial ECG tracings or continuous ST-segment monitoring.[10]

OTHER LABORATORY TESTS

A chest radiograph is generally performed at times of admission so that patients can be assessed for other causes of chest pain and screened for pulmonary congestion, which implies an adverse prognosis. A full lipid profile should be done within an 24 hours of the onset of ACS, as recommended by the National Cholesterol Education Program Adult Treatment Panel III and by the 2007 ACC/AHA guidelines. Selected patients must be tested for secondary causes of ACS, for e.g., thyroid function should be assessed when the patient presents with symptoms of ACS and has persistent tachycardia. Measurement of other circulating markers of increased risk may also be considered.[10]

DIAGNOSTIC PATHWAYS IN THE ED

The current Emergency Department (ED) pathways for evaluating and managing patients who may have ACS depend on 4 main diagnostic tools: Clinical history, ECG results, levels

of cardiac markers and the results of stress testing. On the basis of the initial information, patients are assigned to one of 4 categories: A noncardiac diagnosis, chronic stable angina, possible ACS, or definite ACS. The pathway proposed by the ACC/AHA guidelines is shown in Figure 3.[10]

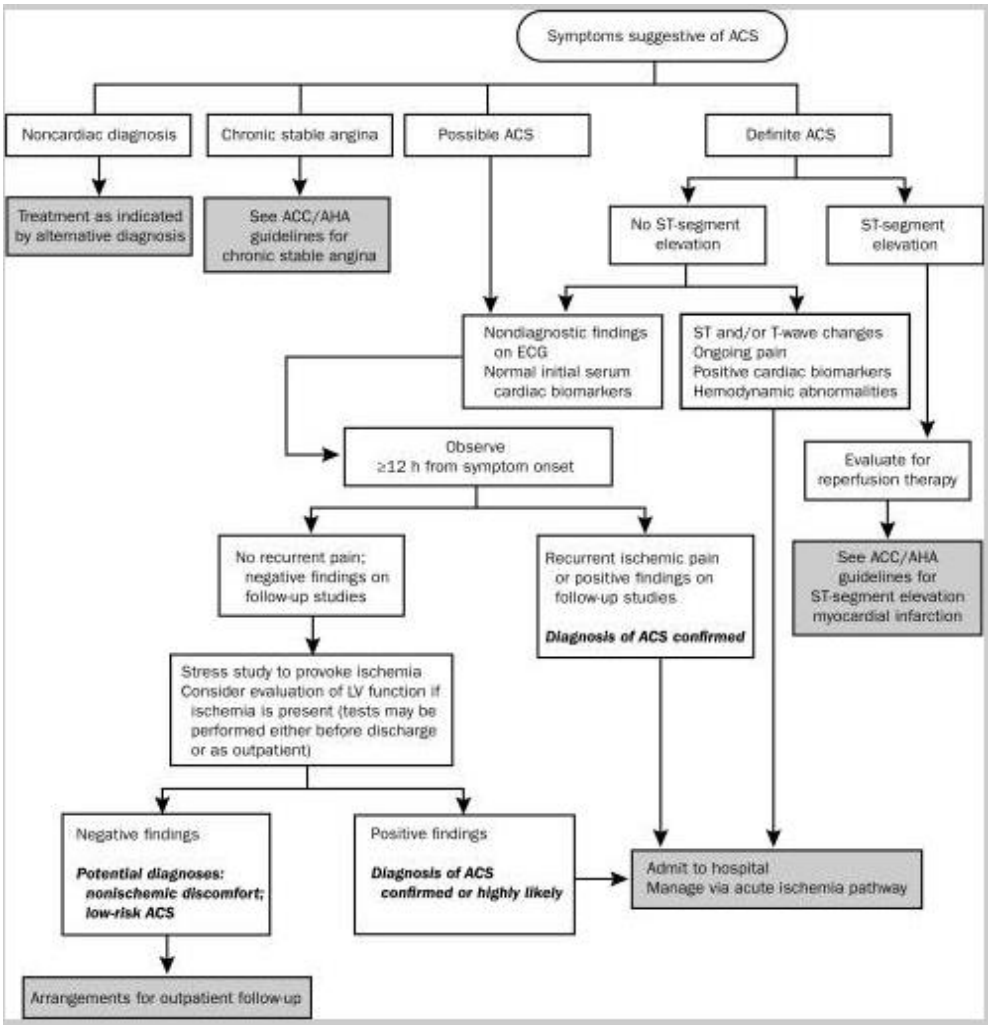


Figure 3: Algorithm for evaluation and management of patients with suspected ACS (ACS). ACC = American College of Cardiology; AHA = American Heart Association; ECG = electrocardiography; LV = left ventricular.

Patients with definite ACS are admitted to the hospital for further care. Admittance to a critical care unit is suggested if there is evidence of active, ongoing ischemia or injury or of

hemodynamic or electrical instability; otherwise, it is rational to place patients in a telemetry step-down unit. The patients with persistent ST-segment elevation must be assessed for emergency reperfusion therapy.

Patients with clearly atypical chest pain and evidence of a noncardiac diagnosis (e.g., gastrointestinal or musculoskeletal disorders) can be discharged home and instructed to follow up with their primary physician (chronic stable angina may also be diagnosed in this setting). The remaining patients, those with possible ACS, must be observed in a facility with cardiac monitoring capabilities (e.g., a chest pain unit, an ED, or a hospital telemetry ward), and ECG (or continuous 12-lead ECG monitoring) and cardiac biomarker measurements should be repeated at predetermined, specified time intervals. If new ST-segment changes or elevations in cardiac marker levels are noted, the diagnosis of ACS is considered highly likely, and patient is taken off the pathway and admitted to the hospital. If the patient remains pain-free and the results of ECG and cardiac marker tests are negative, an early stress test should be performed either prior to discharge or on an outpatient basis within 72 hours. The patients with negative diagnostic test outcomes may be discharged with specific instructions for activity, medications, and additional testing. The patients with evidence on stress testing of ischemia or Left Ventricular Dysfunction (LVD) should be admitted to the hospital and managed according to an acute ischemia pathway.[10]

Evaluation:

The main step of evaluation is an ECG, which aids distinguish between STEMI and NSTEMI unstable angina. American Heart Association guidelines maintain any patient with complaints suspicious of ACS should get an ECG within 10 minutes of arrival. Cath lab must be activated as soon as STEMI is confirmed in a Percutaneous Coronary Intervention (PCI)

center. Cardiac enzymes especially troponin, CK-MB/CK ratio is vital in evaluating the NSTEMI versus myocardial ischemia without tissue destruction. A chest x-ray is obliging in diagnosing causes other than MI presenting with chest pain similar to pneumonia and pneumothorax.[3] The LGI is a simple, low cost tool allowing re-stratification of no diabetic patients with low TIMI score, at higher risk of death or severe HF (KK 3–4). This index is probably associated with greater infarct size and severe complications.[7]

Treatment/Management:

The primary treatment for ACS consists of aspirin (300 mg) and heparin bolus and Intravenous (IV) heparin infusion if there are no contraindications to same. Antiplatelet ticagrelor or clopidogrel therapy is recommended. The choice depends on the preference of a local cardiologist. Ticagrelor is not administered to patients receiving thrombolysis.[3]

Supportive measures like pain control with morphine/fentanyl and oxygen in case of hypoxia are providing as necessary. Sublingual or infusion nitroglycerin can be utilized for pain relief as well. In cases of inferior wall ischemia, nitroglycerin could cause severe hypotension and, if at all, should be used with extreme caution. Continuous cardiac monitoring for arrhythmia is required.[3]

Further treatment of ACS would depends on whether it is STEMI/NSTEMI or unstable angina. The American Heart Association (AHA) endorses an emergent catheterization and Percutaneous Intervention (PCI) for STEMI with door to procedure start time of less than the 90 minutes. A thrombolytic (tenecteplase or other thrombolytic) is suggested if no PCI is available and the patient cant be moved to the catheterization lab in less than 120 minutes. AHA guideline dictates the door to needle (TNK/other thrombolytics) time to be less than the 30 minutes.[3]

NSTEMI/unstable angina-symptom control is tried laterally with the primary treatment with aspirin, and heparin. If the patient continues to have pain, then emergent catheterization is advised. If symptoms are controlled effectively, then a decision might be taken on the timing of catheterization and other valuation techniques including myocardial perfusion study from case to case basis depending on comorbidities. ACS always requires an admission and emergent cardiology evaluation. Computerized tomography angiography may also be used for further workup depending on availability and choice of the cardiologist.[3]

Beta-blockers, statins and ACE inhibitors should be started in all ACS cases as soon as possible unless the contraindications exist. Cases not amenable to PCI are taken for Coronary Artery Bypass Graft (CABG) or managed medically depending upon the comorbidities and patient's preference.[3]

UNSTABLE ANGINA/NSTEMI

The 2007 ACC/AHA guidelines that states the goal of immediate treatment of patients with UA/NSTEMI is to provide relief of ischemia and to prevent the recurrence of adverse ischemic events. Treatment with anti-ischemic, antiplatelet, and anticoagulant agents is essential to achieve this goal. Additionally to aggressive medical therapy, 2 treatment pathways have emerged for treating UA/NSTEMI patients: An early invasive strategy and an initial conservative strategy. Risk stratification helps to assess how aggressive we should be with respect to both medical therapy and treatment strategy.[10]

Early Invasive Strategy or Initial Conservative Strategy

An early invasive strategy involves regular cardiac catheterization; usually within 4 to 24 hours post admission, followed by revascularization with PCI or CABG, as appropriate, depending on the coronary anatomy. The conservative strategy, on the other hand, consists of

initial medical treatment accompanied by catheterization and revascularization only if ischemia recurs despite vigorous medical therapy, either when the patient is at rest or during a noninvasive stress test. The 2007 ACC/AHA guidelines have given the early invasive strategy a class I, level of sign A recommendation for patients with UA/NSTEMI who were at high risk. The guidelines recommend either a conservative or an invasive strategy for low-risk patients because the outcomes achieved by these approaches are comparable for these patients. Nonetheless, the guidelines provide the conservative strategy a class I recommendation for women with low-risk characteristics.[10]

To date, 10 randomized trials have evaluated these 2 general strategies. Although there were no substantial differences in results between the first 3 trials and the most recent, the remaining 6 trials have shown that an early invasive approach provides significant benefits.[10]

The Framingham and Fast Revascularization During Instability in the Coronary Artery Disease II (FRISC II) trial, which is involved 2,457 patients with UA/NSTEMI, found that the early invasive strategy achieved a significantly lesser rate of the primary endpoint of death or MI at 6 months (9.4%) than did the conservative strategy (12.1%; p=0.031). The Treatment for Angina with Aggrastat and Determine Cost of Therapy with an Invasive or Conservative Strategy—Thrombolysis in MI 18) trial randomly assigned 2200 patients, who were treated with aspirin, heparin, and tirofiban, to an early invasive or a conservative strategy. At 6 months, the rate of the primary endpoint of death, MI, or re-hospitalization for ACS was 19.4% for the conservative strategy group and 15.9% for the early invasive group (odds ratio, 0.78; p=0.025) Patients with higher troponin concentrations, ST-segment changes, and a high TIMI risk score (≥3) benefited most from the early invasive strategy. The most recent study, the Invasive Versus Conservative Treatment in the Unstable Coronary

Syndromes (ICTUS), randomly assigned 1,200 patients with ACS to an early invasive strategy or a conservative strategy and found no significant changes between the groups at 1-year and at the 3 years in the rate of the primary endpoint of death, MI, or re-hospitalization for angina.[10]

A meta-analysis of contemporary randomized trials of treatments for NSTEMI found that the early invasive strategy was related with a statistically significant 25% lower incidence of all-cause mortality than was the conservative strategy (p=0.001). Another meta-analysis of 8 randomized trials compared invasive and conservative strategies for women and men with non-ST-segment elevation ACS found that an early invasive strategy was equally beneficial for men and for women who were considered to have increased-risk disease on the source of increased levels of biomarkers of necrosis.[10]

A recent randomized trial, The timing of Intervention in Patients with Acute Coronary Syndrome (TIMACS), compared the outcomes attained by an early invasive strategy (intervention within 24 hours of presentation) and a delayed invasive strategy (intervention at any time >36 hours after presentation) for 3,031 high-risk patients with UA/NSTEMI. The early invasive strategy was not greater to the delayed invasive strategy in reducing the primary endpoint of death, MI, or stroke at 6 months (9.6% vs. 11.3%; Hazard Ratio [HR], 0.85; 95% Confidence Interval [CI], 0.68-1.06; p=0.15), except for high-risk patients with the GRACE risk score higher than 140 (13.9% vs. 21.0%; HR, 0.65; 95% CI, 0.48-0.89; p=0.006).[10]

ANTI-ISCHEMIC THERAPY

The ACC/AHA class I recommendations for anti-ischemic therapy include both nonpharmacological and pharmacological measures.[10]

Nitroglycerin

Nitroglycerin is a vasodilator which reducess the myocardial oxygen demands by reducing ventricular preload via venodilation; it improves myocardial oxygen delivery by dilating large coronary arteries and enhancing collateral flow to ischemic areas. Nitroglycerin must initially be administered sublingually or by buccal spray (0.3-0.6 mg) every 5 minutes for the total of 3 doses. If pain persists, the administration of IV nitroglycerin should be initiated (initial rate of 5-10 μg/min with increases of 10 μg/min every 3 to 5 minutes until symptoms are relieved or if systolic blood pressure drops below 100 mmHg). Topical or oral nitrates may be used if the episode of pain has resolved, and may replace IV nitroglycerin if the patient has been pain-free for 12 to 24 hours. Absolute contraindications to the use of nitroglycerin are hypotension or the usage of sildenafil within the previous 24 hours or of tadalafil within the previous 48 hours.[10]

Morphine and Other Analgesics

Morphine is indicated when the symptoms associated with ischemia are not resolved after 3 doses of nitroglycerin or when these symptoms recur during treatment. In such cases, 1–5 mg of morphine sulfate can be administered IV every 5–30 minutes as needed, with careful monitoring of blood pressure and respiratory rate. Morphine serves as a potent analgesic and anxiolytic agent; in addition, its hemodynamic effects may be useful in treating UA/NSTEMI. The 2007 ACC/AHA guidelines downgraded the reference for the use of morphine for uncontrolled ischemic discomfort from class I to class IIa because data from a large observational registry, although subject to uncontrolled selection bases, suggested that the adjusted likelihood of death was higher when morphine was used.[10]

The ACC/AHA guidelines that states the use of nonsteroidal anti-inflammatory drugs, both nonselective agents and cyclooxygenase-2 selective agents (except for aspirin), should be

terminated when a patient presents with an UA/NSTEMI because of the known cardiovascular risks associated with the agents, and also because the Enoxaparin and Thrombolysis Reperfusion for Acute Myocardial Infarction Treatment—Thrombolysis In Myocardial Infarction (EXTRACT-TIMI 25) trial found that these agents were linked with an greater risk of adverse cardiovascular events.[10]

β-Blockers

β-Blockers inhibit β-1 adrenergic receptors in the myocardium and decrease myocardial contractility and heart rate, thus reducing myocardial oxygen demand. The 2007 ACC/AHA guidelines state that, in the absence of contraindications, therapy with oral β-blockers should be started within first 24 hours after onset of ACS (class I recommendation). For all patients, the oral dose should be adjusted to achieve a target resting heart rate of 50 to 60 beats/min. It is reasonable to administer IV β-blockers to patients who are hypertensive at the time of presentation (class IIa recommendation).

The Clopidogrel and Metaprolol in Myocardial Infarction (COMMIT) trial found the risk of cardiogenic shock was higher for patients treated with IV β-blockers than for those who were not (especially for patients with tachycardia, hypotension, or in Killip class II or III CHF). Because of this finding, the 2007 ACC/AHA guidelines suggest caution in use of IV β-blockers. Contraindications to β-blockade include severe sinus bradycardia (heart rate <50 beats/min), marked first-degree atrioventricular block (ECG P-R interval >0.24 second) or any second-degree or third-degree atrioventricular block, persistent hypotension, pulmonary edema, history of bronchospasm, evidence of a low-output state (eg, oliguria), and increased risk of cardiogenic shock. Numerous placebo-controlled trials involving patients with UA/NSTEMI have established the benefit of β-blockers in decreasing the incidence of subsequent MI, recurrent ischemia, or both.[10]

Calcium Channel Blockers

Calcium channel blockers inhibit the contraction of both the myocardium (thereby decreasing myocardial oxygen demand) and vascular smooth muscle (thereby causing coronary vasodilatation and enhancing myocardial blood flow). The ACC/AHA guidelines recommend these agents for patients with chronic or recurrent symptoms following treatment with full-dose nitrates and β-blockers, for patients with contraindications to β-blockade, and for the patients with Prinzmetal type of angina. For such patients, calcium channel blockers that slow down the heart rate (e.g., diltiazem or verapamil) are prescribed. These agents are not be given to patients with severe LV dysfunction or pulmonary edema. Danish Verapamil Infarction Trial (DAVIT) is the largest randomized trial to date to have evaluated the effectiveness of a calcium channel blocker for patients with ACS. The results revealed a trend toward lower rates of death or MI when verapamil was administered to patients with suspected ACS. Similar reduces in the rates of MI and refractory angina with diltiazem have been identified. Nifedipine, which does not decrease the heart rate, has been shown to be harmful to patients with acute MI when it is administered without the simultaneous administration of a β-blocker. The newer dihydropyridine calcium antagonists amlodipine and felodipine have not been evaluated specifically for administration to patients with ACS, but studies involving normotensive patients with CAD or hypertensive patients with cardiovascular risk factors had shown that these agents provide significant benefits.[10]

Inhibitors of the Renin-Angiotensin-Aldosterone System

The 2007 ACC/AHA guidelines recommend that, in the absence of hypotension or other known contraindications, an Angiotensin-converting Enzyme (ACE) inhibitor (or an angiotensin II receptor blocker for patients who cannot tolerate ACE inhibitors) should be administered orally within the first 24 hours to patients with pulmonary congestion or an LV

ejection fraction of 40% or lower (class I recommendation) and should be considered for administration to patients without these features (class IIa recommendation). The recommendation for ACE inhibitor therapy is based on the results of numerous large studies showing that mortality rates were substantially decreased when ACE inhibitors were initiated within the 24 hours of MI. The angiotensin II receptor blocker valsartan was found to be active as captopril in patients at greater risk of cardiovascular events following MI; however, providing a combination of the 2 agents was found to be harmful. Long-term use of ACE inhibitors is recommended for many patients with high-risk chronic CAD.[10]

Eplerenone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study (EPHESUS) found that the selective aldosterone receptor blocker eplerenone decreased morbidity and mortality rates for patients with MI due to LV dysfunction and either CHF or diabetes mellitus. Long-term administration of eplerenone is recommended for those patients in the absence of severe renal dysfunction or hyperkalemia.

Other Anti-Ischemic Therapies

Ranolazine is a recently approved anti-ischemic agent that is recommended for use alone or in combination with nitrates, β-blockers, or amlodipine for the treatment of chronic refractory angina. The (Metabolic Efficiency with Ranolazine for Less Ischemia in Non-ST-Elevation Acute Coronary Syndromes-Thrombolysis in Myocardial Infarction 36 (MERLIN-TIMI 36) study showed that ranolazine had a benefit over placebo in reducing the incidence of recurrent ischemia (HR, 0.87; 95% CI, 0.76-0.99; p=0.03) when administered within 48 hours of the onset of UA/NSTEMI. Nevertheless, ranolazine had no effect on the composite endpoint of cardiovascular death, MI, or recurrent ischemia (HR, 0.92; 95% CI, 0.83-1.02; p=0.11)[10]

Antithrombotic treatment is the basis of treatment for patients with UA/NSTEMI. It has 2 components: (1) antiplatelet therapy, which decreases platelet activation and aggregation, integral steps in the formation of a thrombus after plaque disruption, and (2) anticoagulant therapy, which targets the clotting cascade to avoid the deposition of fibrin strands in the clot. The ACC/AHA guidelines recommend tailoring the specific antithrombotic agents to the treatment strategy selected. Figure 4 shows the algorithm for choosing agents for patients managed with an invasive strategy, and Figure 5 shows the algorithm for patients managed with a conservative strategy.[10]

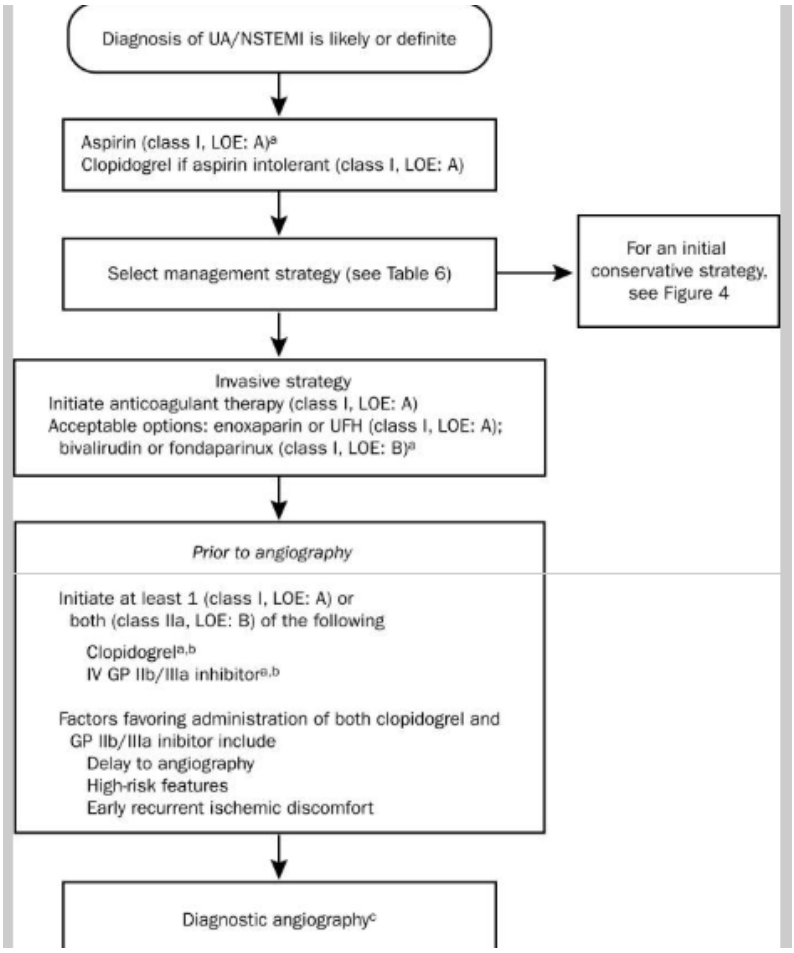


Figure 4: Algorithm for the patients with UA/NSTEMI managed by an initial invasive strategy. When multiple drugs are listed, they are in alphabetical order and not in order

of preference. GP = glycoprotein; IV = intravenous; LOE = level of evidence; NSTEMI = non—ST-segment elevation MI; UA = unstable angina; UFH = unfractionated heparin.

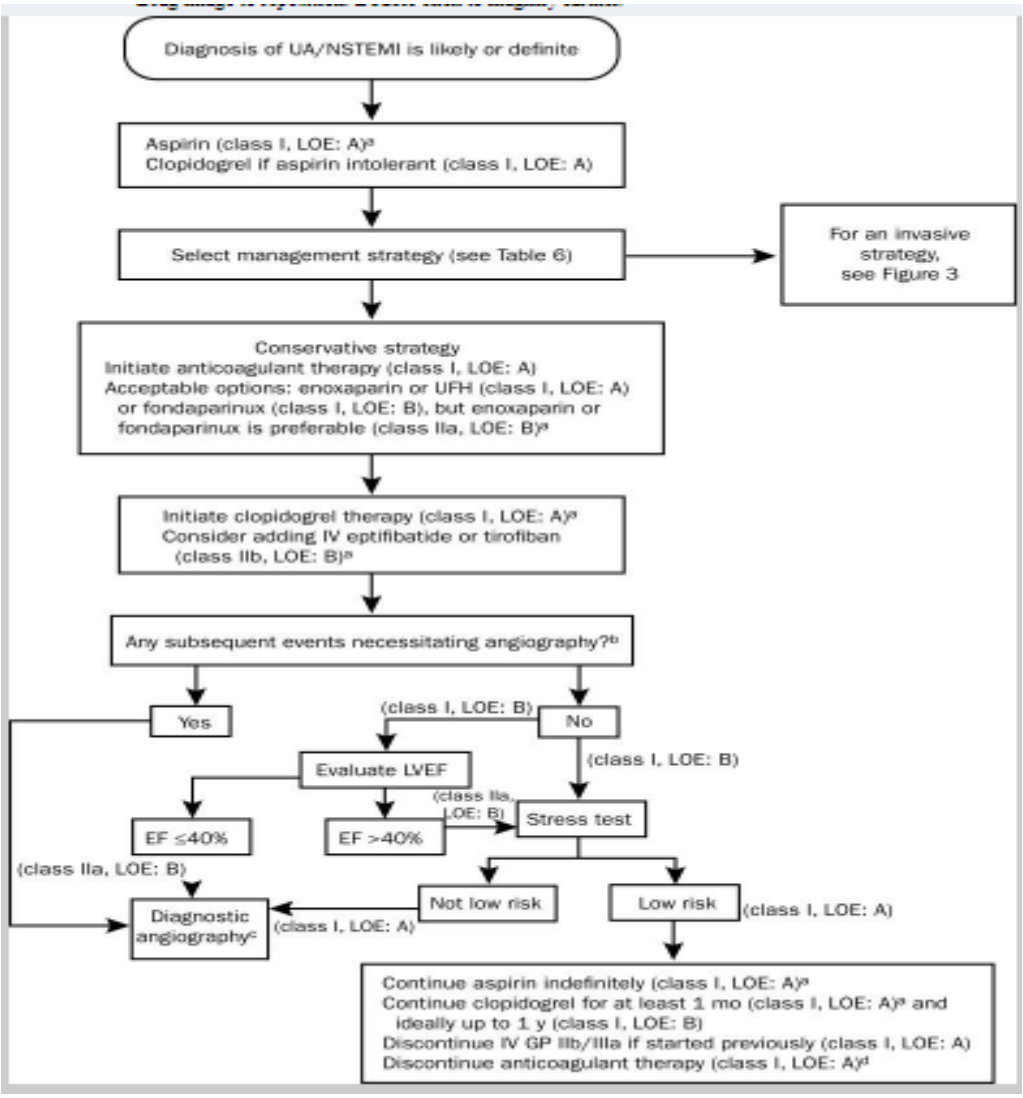


Figure 5: Algorithm for patients with UA/NSTEMI managed by an initial conservative strategy. When multiple drugs are listed, they are in alphabetical order and not in order of preference. EF = ejection fraction; GP = glycoprotein; LOE = level of evidence; LVEF = left ventricular ejection fraction; NSTEMI = non—ST-segment elevation MI; UA = unstable angina; UFH = unfractionated heparin.

ANTIPLATELET THERAPY

Aspirin. Aspirin blocks the synthesis of thromboxane A2 by irreversibly inhibiting cyclooxygenase 1, thus diminishing platelet aggregation. Four randomized studies have each showed that, compared with placebo, aspirin reduces the risk of death or MI by more than 50% for patients presenting with UA/NSTEMI. The ACC/AHA guidelines recommend an initial daily dose of 162 to 325 mg, followed by a daily dose of 75 to 162 mg for long-term secondary prevention. Absolute contraindications to aspirin therapy include documented aspirin allergy (e.g., asthma or anaphylaxis), active bleeding, or a known platelet disorder. Clopidogrel is a recommended alternative for patients with who are unable to tolerate aspirin.[10]

Clopidogrel:

Clopidogrel is a thienopyridine derivative that blocks the P2Y12 Adenosine Diphosphate (ADP) receptor on platelets. This action reduces platelet activation and aggregation, enhances bleeding time and decreases blood viscosity. Clopidogrel and aspirin is recommended for all patients with UA/NSTEMI.[10]

The Clopidogrel in Unstable Angina to Prevent Recurrent Events (CURE) study randomly assigned 12,562 patients to receive either aspirin alone (75-325 mg/day) or aspirin plus clopidogrel (300 mg loading dose, then 75 mg/day). The incidence of the primary endpoint of cardiovascular death, MI, or stroke was 20% lower for both low-risk and high-risk patients with UA/NSTEMI who were administered aspirin plus clopidogrel (11.4%) than for those who were administered aspirin alone (9.3%; $p<0.0001$). Benefit was seen as early as 24 hours post initiation of treatment (the Kaplan-Meier curves began diverging after just 2 hours) and continued throughout the 1-year treatment period of the study. Clopidogrel was associated

with significantly more cases of major bleeding but not with more cases of life-threatening bleeding. The prespecified subgroup analysis, PCI-CURE,

showed that treatment with clopidogrel before PCI was also associated with a substantial benefit: The decline in cardiac events was 31% at 30 days and at 1 year.[10]

On the basis of the results of the PCI-CURE study, the Clopidogrel for the Reduction of Events During Observation (CREDO) study and the Clopidogrel as an Adjunctive Reperfusion Therapy—Thrombolysis in Myocardial Infarction 28 (CLARITY-TIMI 28) trial, together with the result of a meta-analysis (which found that, in comparison with no pretreatment, clopidogrel pretreatment decreased the incidence of cardiovascular death, MI, or stroke from randomization through 30 days by 41%; p=0.001), the 2005 guidelines from the ACC, the AHA, and the Society for Coronary Angiography and Interventions contain a class I, levels of evidence A recommendation for clopidogrel pretreatment before PCI.[10]

The risk of significant bleeding increased when patients received clopidogrel within 5 days of CABG. Therefore, the ACC/AHA guidelines recommend stopping the administration of clopidogrel at least 5 days prior to surgery, if possible. The current practice in most hospitals is either to begin clopidogrel administration at the time of admission (this intervention has the benefit of reducing the prevalence of early ischemic events and pretreatment before PCI) or to postpone treatment until after coronary angiography has been performed, in which case the drug may be either administered while PCI is carried out or withheld until after CABG has been performed.[10]

Newer P2Y12 ADP Inhibitors

High rates of recurrent atherothrombotic events in spite of the administration of dual-antiplatelet therapy with aspirin and clopidogrel have given rise to a great deal of interest in finding more potent inhibitors of the P2Y12 ADP receptor.[10]

Prasugrel is an irreversible P2Y12 ADP receptor antagonist recently approved by the US Food and Drug Administration (US FDA). Many studies have shown that prasugrel achieves significantly higher (nearly double) levels of platelet inhibition than the daily dose of 75 mg or even 150 mg clopidogrel. The Trial to Assess Improvement in Therapeutic Outcomes by Optimizing Platelet Inhibition with Prasugrel—Thrombolysis in Myocardial Infarction 38 (TRITON-TIMI 38) trial administered prasugrel (a 60-mg loading dose and a 10-mg daily maintenance dose) or clopidogrel to 13,608 high-risk patients with ACS who were scheduled for PCI. The incidence of the primary endpoint of cardiovascular death, MI or stroke at 6 to 15 months was significantly lesser in the prasugrel group (9.9%) than in the clopidogrel group (12.1%; $p<0.001$). The incidence of stent thrombosis was 52% lesser with prasugrel (1.1%) than with clopidogrel (2.4%; $p<0.001$). The risk of TIMI major bleeding, including the risk of fatal bleeding, was higher for the patients receiving prasugrel (2.4%) than for those receiving clopidogrel (1.8%; $p=0.03$).[10]

Ticagrelor (AZD6140) is a reversible oral P2Y12 receptor antagonist with a half-life of about 12 hours. The recently completed study of Platelet Inhibition and Patient Outcomes (PLATO) randomized 18,624 patients with ACS to either ticagrelor (loading dose of 180 mg followed by 90 mg twice daily) or clopidogrel for up to 12 months. The primary endpoint of death from vascular causes, MI or stroke occurred in 9.8% of patients receiving ticagrelor vs. 11.7% of those receiving clopidogrel (HR, 0.84; 95% CI, 0.77-0.92; $p<0.001$). The rate of

death from any cause was also reduced with ticagrelor vs. clopidogrel (4.5% vs. 5.9%; p<0.001). Overall, the rates of major bleeding were comparable between the ticagrelor and clopidogrel groups (11.6% vs. 11.2%; p=0.43).[10]

GP IIb/IIIa Inhibitors

The platelet GP IIb/IIIa inhibitors are potent and specific inhibitors of platelet aggregation. They act by interrupting the final common pathway of fibrinogen-mediated cross-linkage of platelets. Several large studies involving patients with UA/NSTEMI have shown that the GP IIb/IIIa inhibitors are of significant benefit for patients at high risk, those undergoing PCI or both. There are currently three agents available for use: Abciximab, eptifibatide and tirofiban. Abciximab is indicated only if the angiography is not significantly delayed and the PCI is likely to be performed; otherwise, IV eptifibatide or tirofiban is the preferred option. The risk is associated with GP IIb/IIIa inhibitors is an increased rate of hemorrhage, typically at the site of vascular intervention. Therefore, patients should be monitored closely for bleeding, and complete blood cell counts should be determined on a regular basis.[10]

GP IIb/IIIa inhibition tends to have the greatest benefits for patients at higher risk of complications, e.g., those with increased troponin concentrations, diabetes, ST-segment changes, recurrent angina, previous aspirin use or a TIMI risk score of 4 or higher. The benefit of GP IIb/IIIa inhibition has been confirmed even for patients who have been pretreated with clopidogrel. The optimal timing for the start of GP IIb/IIIa inhibitors has been debated. The Early Glycoprotein IIb/IIIa Inhibition in Non-ST-Segment Elevation Acute Coronary Syndrome (EARLY ACS) trial involved 9,492 patients who were randomly assigned either to early GP IIb/IIIa inhibition or to the provisional use of GP IIb/IIIa inhibitors post angiography. The results exposed that early eptifibatide exerted no statistically

significant benefit in reducing the composite endpoint of adverse cardiovascular events but was associated with a statistically significant increase in bleeding rates.[10]

The 2007 ACC/AHA guidelines recommend that, for patients with UA/NSTEMI who will be treated initially according to an invasive strategy, either an intravenous GP IIb/IIIa inhibitor or clopidogrel should be added to aspirin and anticoagulant therapy (upstream) before diagnostic angiography is performed (class I recommendation). They also state that adding both agents is reasonable (class IIa recommendation).[10]

ANTICOAGULANT THERAPY

The 2007 ACC/AHA UA/NSTEMI guidelines recommend starting anticoagulant therapy for all patients (without contraindications) as soon as possible after presentation (class I recommendation). The guidelines recommend 4 agents as options: Unfractionated Heparin (UFH), enoxaparin, fondaparinux, and bivalirudin (approved only for patients managed according to an invasive strategy).[10]

Unfractionated Heparin

The findings of several randomized trials show that UFH is associated with lower rates of death or MI than aspirin alone. The anticoagulant effects of UFH are variable. The ACC/AHA guidelines recommend weight-adjusted dosing of UFH (60 U/kg bolus and 12 U/kg/hr infusion), frequent monitoring of activated partial thromboplastin time (every 6 hours until 2 consecutive values are within the target range, and every 24 hours thereafter), and titration of UFH according to a standardized nomogram with a target range of activated

partial thromboplastin time between 1.5 and 2.0 times that of control or approximately 50 to 70 seconds. Administration of UFH should continue for at least 48 hours post presentation with UA/NSTEMI.[10]

Complete blood cell counts must be assessed at least daily during treatment with UFH. Autoimmune heparin-induced thrombocytopenia in association with thrombosis is a rare but dangerous complication of UFH administration (incidence is <0.2%). If clinical findings suggest that this complication has occurred, all heparin therapy should be stopped immediately.[10]

Low-Molecular-Weight Heparin

Because the rates of recurrence of ischemic events remain high even when UFH is administered, Low-molecular-Weight Heparins (LMWHs) have been developed with a goal of providing superior anticoagulation. They are active against both factor Xa and factor IIa; therefore, they inhibit both action and thrombin generation. Their other advantages over UFH include a lower rate of thrombocytopenia, more bioavailability and less binding to plasma proteins, a factor that renders monitoring level of anticoagulation unnecessary.[10]

A variety of LMWHs (dalteparin, enoxaparin, and nadroparin) have been compared with UFH for the treatment of UA/NSTEMI, but only enoxaparin has been found to have a clear benefit. Early trials, such as Efficacy and Safety of Subcutaneous Enoxaparin in Non-Q-Wave Coronary Events (ESSENCE) and the TIMI 11B, showed that, compared with UFH, enoxaparin achieved a 20% reduction in the incidence of death, MI, recurrent ischemia or some combination of these factors. The greater Yield of the New Strategy of Enoxaparin, Revascularization and Glycoprotein IIb/IIIa Inhibitors (SYNERGY) study found that enoxaparin was not inferior to UFH in the setting of an early invasive strategy. Nevertheless,

enoxaparin had a clear advantage over UFH in the setting of a conservative strategy, as shown by the older studies and the more recent Aggrastat to Zocor (A-to-Z) study. The 2007 ACC/AHA guidelines include a class IIa recommendation stating that enoxaparin or fondaparinux (see Factor Xa Inhibitors) is preferable to UFH as anticoagulant therapy for UA/NSTEMI patients who will be treated conservatively, unless CABG is planned within 24 hours.

The benefit of enoxaparin is greater for patients at higher risk, such as those with ST-segment deviation, elevated troponin concentrations, and high TIMI risk scores. The incidences of major bleeding associated with LMWHs had been found to be similar to those associated with UFH, with 1 exception: the SYNERGY trial found a statistically significant increase in the incidence of major bleeding in reminder with enoxaparin administration.[10]

Direct Thrombin Inhibitors

Direct thrombin inhibitors have several potential benefits over indirect thrombin inhibitors (such as UFH or LMWH): They do not require a cofactor such as antithrombin for their action and can therefore directly inhibit clot-bound thrombin; do not interact with plasma proteins; and do not cause thrombocytopenia.[10]

The administration of bivalirudin to patients with UA/NSTEMI was recently studied in the Acute Catheterization and Urgent Intervention Triage Strategy (ACUITY) trial, which randomly assigned 13,819 patients with ACS managed with an early invasive strategy to one of 3 antithrombotic regimens: UFH (or enoxaparin) plus a GP IIb/IIIa inhibitor, bivalirudin plus a GP IIb/IIIa inhibitor, or bivalirudin alone. No differences in the rates of the primary

endpoint (composite of death, MI, unplanned revascularization for ischemia, and major bleeding at 30 days) were found between the group receiving UFH plus a GP IIb/IIIa inhibitor and the group receiving bivalirudin plus a GP IIb/IIIa inhibitor. Nonetheless, the 30-day net clinical outcomes were significantly better for the group receiving bivalirudin alone than for the group receiving UFH plus a GP IIb/IIIa inhibitor (rates of primary endpoint, 10.1% vs. 11.7%; p=0.015); this difference was due primarily to a significantly reduced rate of major bleeding. The ACC/AHA guidelines have given bivalirudin a class I recommendation for the treatment of patients with UA/NSTEMI designated for an early invasive strategy. The guidelines further state that it is reasonable to omit the administration of an intravenous GP IIb/IIIa antagonist if a thienopyridine is administered at the same time as bivalirudin (class IIa recommendation).[10]

The 2007 ACC/AHA guidelines recommend the use of other direct thrombin inhibitors, such as lepirudin (recombinant hirudin) and argatroban, only for patients with heparin-induced thrombocytopenia.[10]

Factor Xa Inhibitors

Fondaparinux is a synthetic pentasaccharide that is an indirect factor Xa inhibitor and requires antithrombin for its action. The Fifth Organization to Assess Strategies in Acute Ischemic Syndromes (OASIS-5) trial, which involved 20,078 patients with high-risk UA/NSTEMI, compared subcutaneous fondaparinux at a once-daily dose of 2.5 mg with standard-dose enoxaparin. Fondaparinux was found to be not inferior to enoxaparin in reducing the incidence of the primary outcomes of death, MI or refractory ischemia at 9 days. The rate of major bleeding, however, was almost 50% lower in the fondaparinux arm than in the enoxaparin arm, and analyses using the composite variable of the primary outcome and

major bleeding at 9 days showed an advantage of fondaparinux over enoxaparin (incidence, 7.3% vs 9.0%; HR, 0.81; P <.001). Fondaparinux was also coupled with a statistically significant reduction in 30-day and 6-month mortality rates. In the subset of patients undergoing PCI, the risk of catheter-related thrombi was more than 3 times higher in the fondaparinux arm than in the enoxaparin arm; supplemental UFH at the time of catheterization appeared to reduce the risk of this complication.[10]

The 2007 ACC/AHA guidelines include a class I recommendation for fondaparinux as treatment for patients with UA/NSTEMI who will be treated by either a conservative strategy or an early invasive strategy, unless CABG is planned within 24 hours. They further state that fondaparinux is preferred over other anticoagulants for patients who are selected for a conservative treatment strategy and who are at an increased risk of bleeding (class I recommendation).[10]

Oral Anticoagulation

Oral anticoagulation trials with warfarin post ACS have demonstrated the benefit of the combination of warfarin plus aspirin over aspirin alone, provided a sufficient degree of anticoagulation is achieved. However, a similar degree of benefit is seen with clopidogrel plus aspirin rather than with aspirin alone, without the drawback of monitoring the International Normalized Ratio (INR), as is required with warfarin therapy. Additionally, the use of clopidogrel is well established for patients with ACS who undergo PCI and stenting. Therefore, the clinical use of aspirin plus warfarin is limited. Rarely, an indication for warfarin, in addition to aspirin and clopidogrel, arises after UA/NSTEMI (e.g., for patients with atrial fibrillation, a mechanical prosthetic valve, or LV thrombus).[10]

Discharge Antithrombotic Therapy

The 2007 ACC/AHA guidelines provide clear recommendations for antithrombotic therapy at the time of discharge; these recommendations are based on the management strategy (Figure 6). The advantages and disadvantages of triple antithrombotic therapy with aspirin, clopidogrel and warfarin have not been clearly established. Such therapy should be selected only when clear indications are present and should be administered for the shortest possible time and at the lowest effective doses: aspirin, 81 mg; warfarin, titrated to the dosage necessary to sustain an INR of 2.0 to 2.5 (class IIb recommendation).[10]

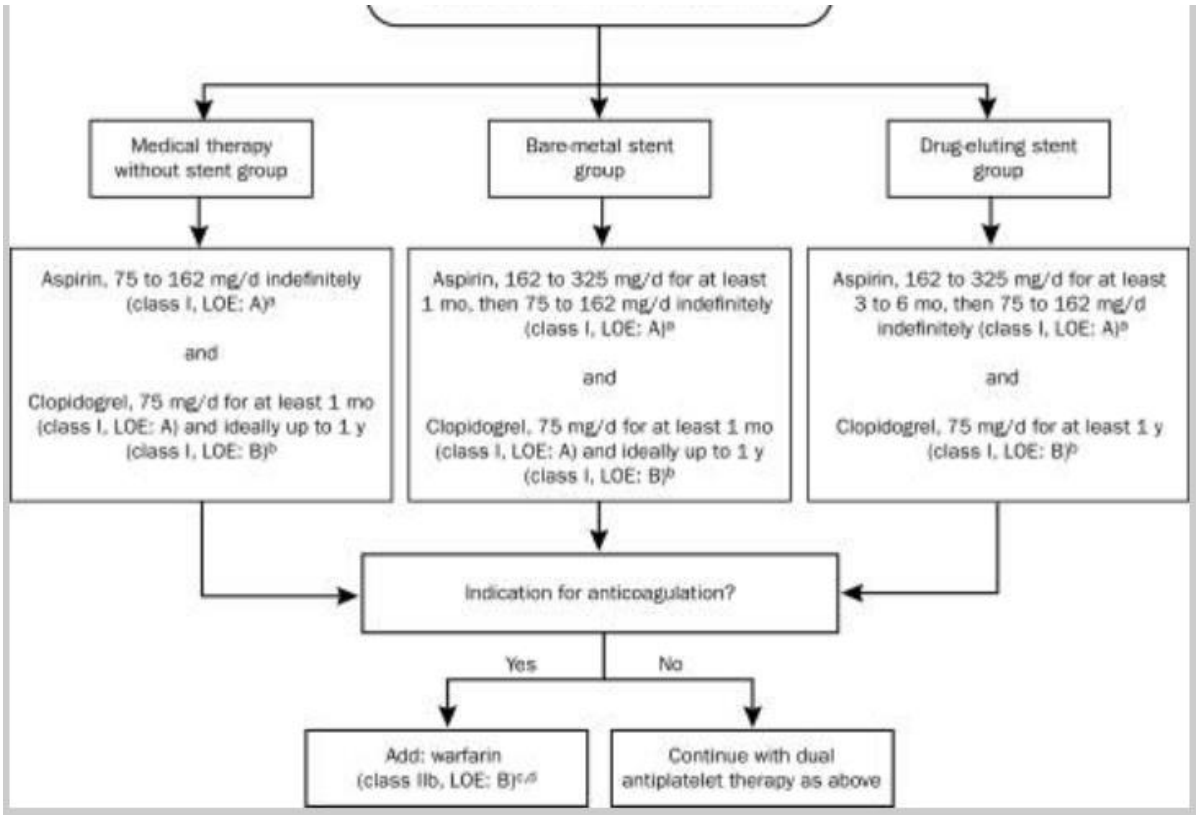


Figure 6: Long-term antithrombotic therapy at hospital discharge after unstable angina (UA)/non—ST-segment elevation MI (NSTEMI). LOE = level of evidence.

LIPID-LOWERING THERAPY

In the absence of contraindications, lipid-lowering statin therapy should be started in all patients with UA/NSTEMI, irrespective of baseline LDL cholesterol levels. If the LDL cholesterol concentration is 100 mg/dL (to convert to mmol/L, multiply by 0.0259) or higher, cholesterol-lowering therapy should be initiated or intensified with a view to achieving an LDL cholesterol concentration below 100 mg/dL. An update to both the Adult Treatment Panel III guidelines and the 2007 ACC/AHA

guidelines states that further titration to a dose necessary to sustain an LDL cholesterol concentration of 70 mg/dL or lower is reasonable (class IIa recommendation).[10]

The Long-Term Intervention with Pravastatin in Ischemic Disease (LIPID) trial showed that, compared with placebo, pravastatin achieved a 26% reduction in mortality rates (p=0.004) for patients with UA, as well as statistically significant reductions in the incidence of subsequent MI, coronary revascularization and stroke. The Pravastatin or Atorvastatin Evaluation and Infection Therapy (PROVE IT)-TIMI 22 trial found that, compared with moderate lipid lowering after ACS with standard-dose pravastatin (40 mg/d), intensive lipid lowering with high-dose atorvastatin (80 mg/d) achieved a 16% decrease in the primary composite end point of all-cause death, MI, UA requiring re-hospitalization or re-vascularization and stroke. The benefit was associated with statistically significant reductions in both LDL cholesterol and CRP concentrations.[10]

MATERIALS & METHODS

MATERIALS AND METHODS:

Source of data: The present study will be carried out with patients attending the services of the department of General Medicine at RL Jalappa hospital, Kolar.

Sample Size: Based on area under the curve (Receiver Operating Characteristic curve) for leuko-glycemic index with discriminatory capacity of 0.66, power of 80% and confidence interval of 95% a sample size of 100 will be taken [calculated sample size:leuko-glycemic index >1000, 50 samples, and <1000, 50 samples].

Study design: A Comparative two group clinical study

Statistical Methods: MEDCAL software package was used for statistical analysis. Student´s t-test will be used as test of significance. Continuous data will be represented as mean and standard deviation. Independent t-test will be used as test of significance to identify the mean difference. p value <0.05 will be considered as statistically significant.

RESULTS

RESULTS:

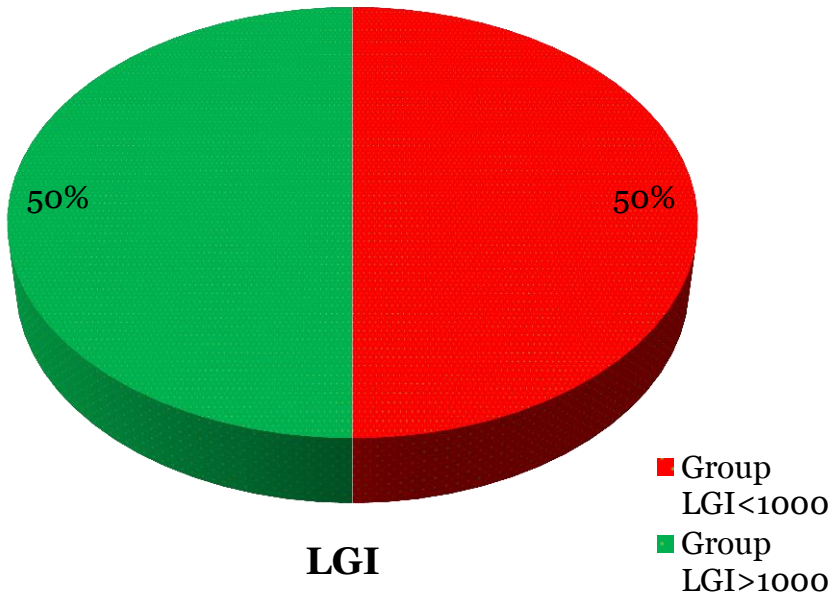
Leuko Glycemic Index:

Of the total 104 patients included in the study, 52 patients had included in the Group LGI<1000 and 52 patients had included in the Group LGI>1000.

Table 1: Leuko Glycemic Index of study subjects:

LGI	No. of patients	%
Group LGI<1000	52	50.0
Group LGI>1000	52	50.0
Total	104	100.0

Figure 7: Leuko Glycemic Index



Age distribution of study subjects:

The age of patients ranged from less than 50 to more than 80 years. The patients in the study were divided into 5 age groups, viz., less than 50 years, between 50 to 60 years, between 61 to 70 years, between 71 to 80 years and more than 80 years.

Among all, there were 1 (1.9%) and 3 (5.8%) in Group LGI<1000 and Group LGI>1000 had age less than 50; 11 (21.2%) and 14 (26.9%) in Group LGI<1000 and Group LGI>1000 had in between the age 50-60; 23 (44.2%) and 22 (42.3%) in Group LGI<1000 and Group LGI>1000 had age in between 61-70; 14 (26.9%) and 13 (25%) in Group LGI<1000 and Group LGI>1000 had age in between 71-80; and 3 (5.8%) in Group LGI<1000 had an age group of more than 80.

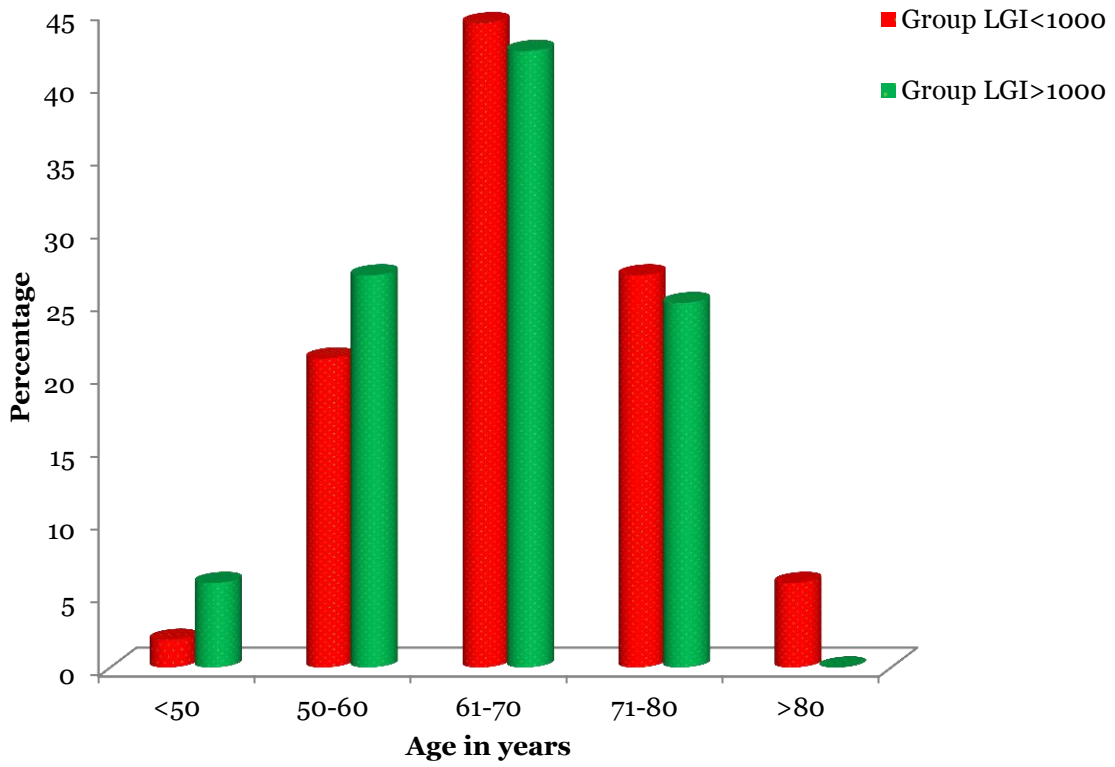
The mean of Group LGI<1000 and Group LGI>1000 were 68.04±8.92 and 64.90±8.72 .

Table 2: Age distribution of study subjects:

Age in years	LGI		Total
	Group LGI<1000	Group LGI>1000	
<50	1(1.9%)	3(5.8%)	4(3.8%)
50-60	11(21.2%)	14(26.9%)	25(24%)
61-70	23(44.2%)	22(42.3%)	45(43.3%)
71-80	14(26.9%)	13(25%)	27(26%)
>80	3(5.8%)	0(0%)	3(2.9%)
Total	52(100%)	52(100%)	104(100%)
Mean ± SD	68.04±8.92	64.90±8.72	66.47±8.92

P=0.352, Not significant, Fisher Exact test

Figure 8: Age distribution of study subjects:



Gender distribution:

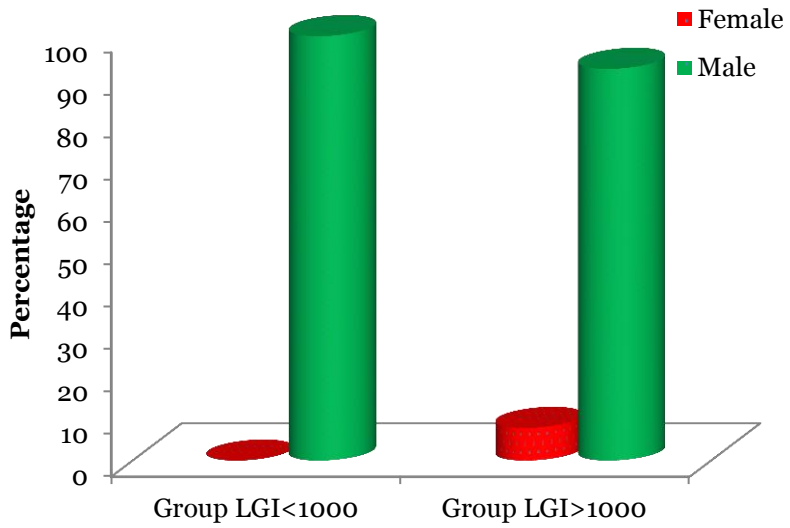
Of all the patients included in the study, 4 (7.7%) were female in the Group LGI>1000; 52 (100%) and 48 (92.3%) were males in the Group LGI<1000 and Group LGI>1000.

Table 3: Gender distribution of study subjects:

Gender	LGI		Total
	Group LGI<1000	Group LGI>1000	
Female	0(0%)	4(7.7%)	4(3.8%)
Male	52(100%)	48(92.3%)	100(96.2%)
Total	52(100%)	52(100%)	104(100%)

P=0.118, Not significant, Chi-Square test

Figure 9: Gender distribution of study subjects:



Systolic Blood Pressure distribution:

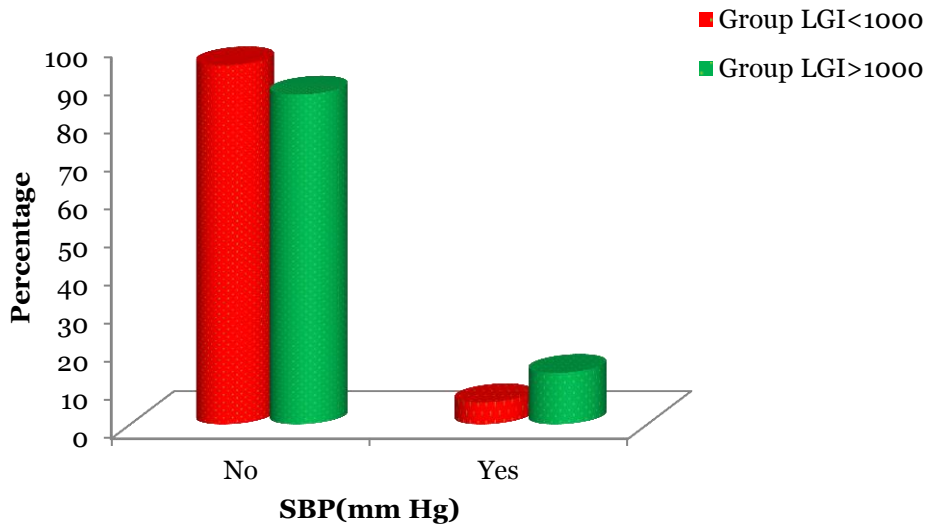
Of total, 49 (94.2) and 45 (86.5%) had no Systolic Blood Pressure in Group LGI<1000 and Group LGI>1000; 3 (5.8%) and 7 (13.5%) had Systolic Blood Pressure in Group LGI<1000 and Group LGI>1000. P=0.483.

Table 4: SBP (mm Hg) distribution in two groups of study subjects:

SBP (mm Hg)	LGI		Total
	Group LGI<1000	Group LGI>1000	
No	49(94.2%)	45(86.5%)	94(90.4%)
Yes	3(5.8%)	7(13.5%)	10(9.6%)
Total	52(100%)	52(100%)	104(100%)

P=0.483, Not significant, Chi-Square test

Figure 10: SBP (mm Hg) distribution in two groups of study subjects:



HR (Heart rate) distribution:

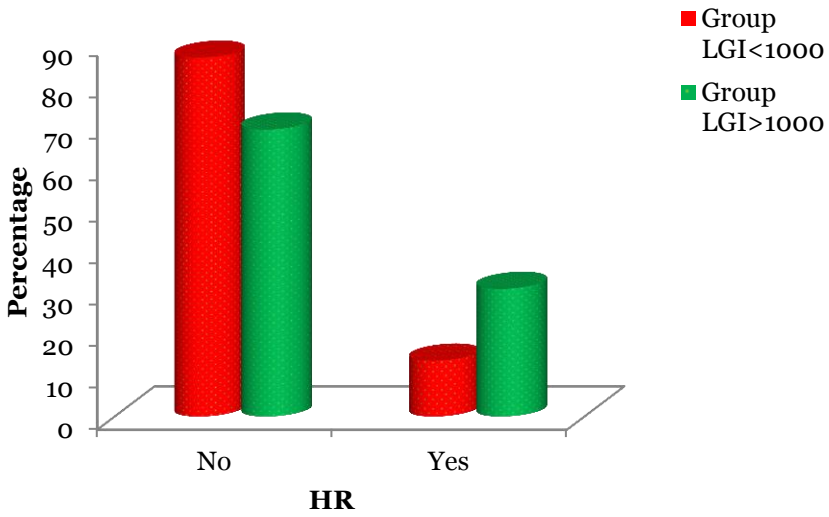
Of total, 81 (77.9%) had showed no heart rate and 23 (22.1%) had showed heart rate in both the groups. P= 0.057.

Table 5: HR distribution in two groups of study subjects:

HR	LGI		Total
	Group LGI<1000	Group LGI>1000	
No	45(86.5%)	36(69.2%)	81(77.9%)
Yes	7(13.5%)	16(30.8%)	23(22.1%)
Total	52(100%)	52(100%)	104(100%)

P=0.057+, significant, Chi-Square test

Figure 11: HR distribution in two groups of study subjects:



Fasting Blood Glucose (FBS) (mg/dl) distribution:

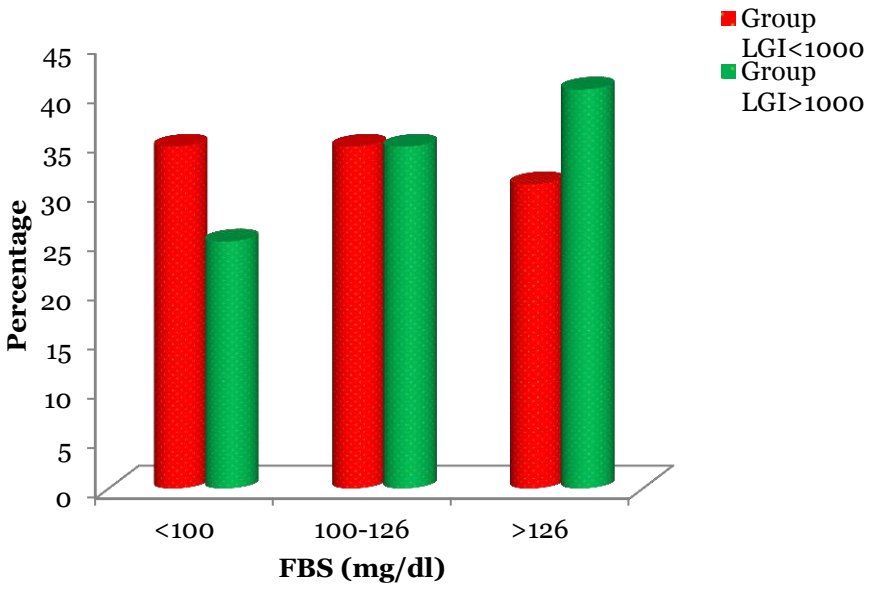
Of total, 31 (29.8%) had <100 in fasting blood glucose level in both the groups, 36 (34.6%) had 100-126 in in both the groups and 37 (35.6%) had >126 in both the groups of fasting blood levels.

Table 6: FBS (mg/dl) distribution in two groups of study subjects:

FBS (mg/dl)	LGI		Total
	Group LGI<1000	Group LGI>1000	
<100	18(34.6%)	13(25%)	31(29.8%)
100-126	18(34.6%)	18(34.6%)	36(34.6%)
>126	16(30.8%)	21(40.4%)	37(35.6%)
Total	52(100%)	52(100%)	104(100%)

P=0.102, Not significant, Chi-Square test

Figure 12: FBS (mg/dl) distribution in two groups of study subjects:



Leukocyte count distribution:

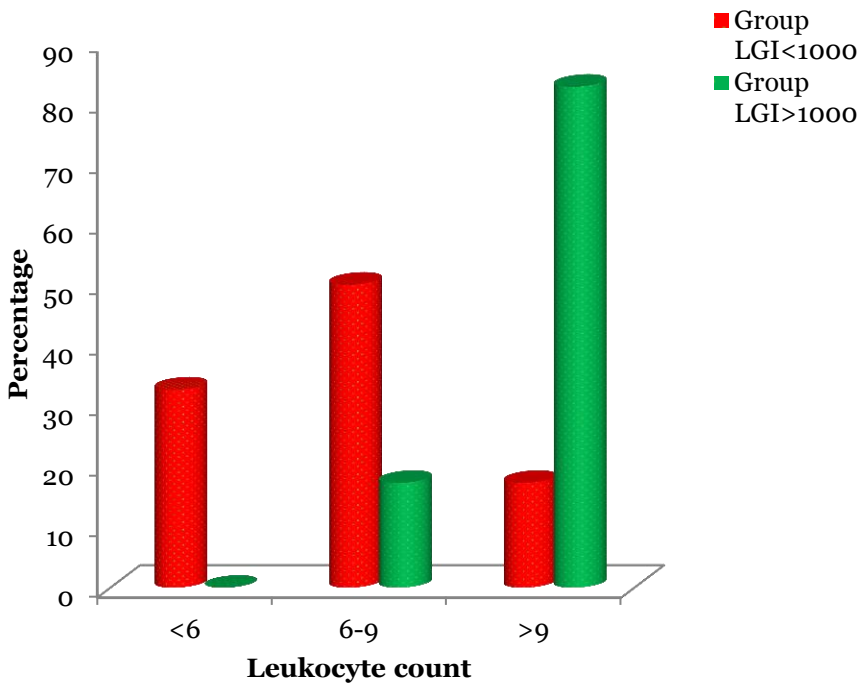
Of total, 17 (16.3%) had <6 in Group LGI<1000 and Group LGI>1000, 35 (33.7%) had 6-9 and 52 (50%) had >9 in Group LGI<1000 and Group LGI>1000. P<0.001

Table 7: Leukocyte count distribution in two groups of study subjects:

Leukocyte count	LGI		Total
	Group LGI<1000	Group LGI>1000	
<6	17(32.7%)	0(0%)	17(16.3%)
6-9	26(50%)	9(17.3%)	35(33.7%)
>9	9(17.3%)	43(82.7%)	52(50%)
Total	52(100%)	52(100%)	104(100%)

P<0.001**, significant, Chi-Square test

Figure 13: Leukocyte count distribution in two groups of study subjects:



Electrocardiogram (ECG) examination distribution:

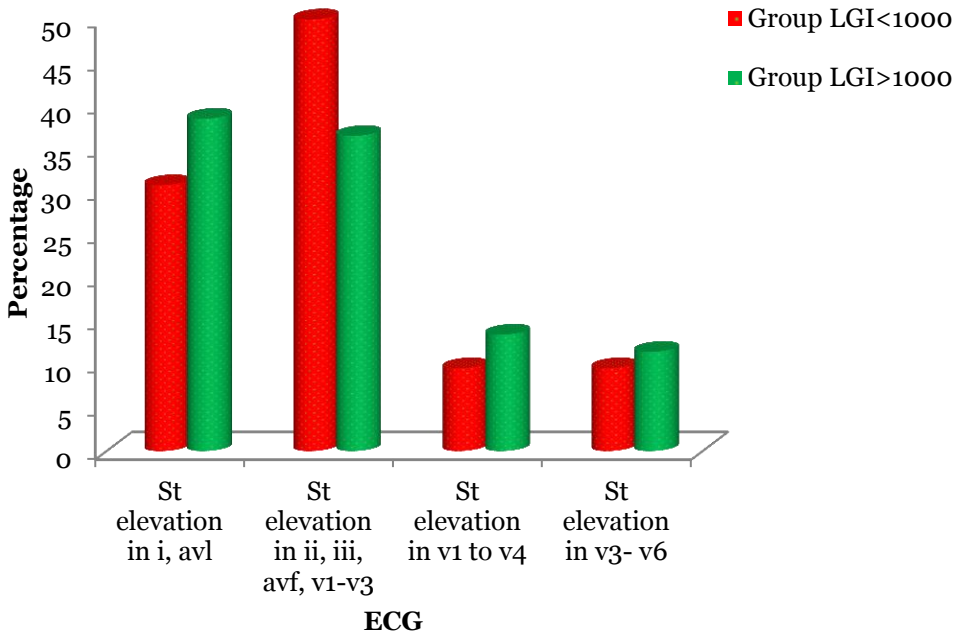
Of total, 36 (34.6%) had St elevation in I, aVL, 45 (43.3%) had St elevation in II, III, aVF, V1-V3 and 12 (11.5%) had St elevation in V1 to V4 and 11 (10.6%) had St elevation in V3-V6 in Group LGI<1000 and Group LGI>1000. P=0.581.

Table 8: ECG examination distribution in two groups of study subjects:

ECG	LGI		Total
	Group LGI<1000	Group LGI>1000	
St elevation in I, aVL	16(30.8%)	20(38.5%)	36(34.6%)
St elevation in II, III, aVF, V1-V3	26(50%)	19(36.5%)	45(43.3%)
St elevation inV1 to V4	5(9.6%)	7(13.5%)	12(11.5%)
St elevation in V3- V6	5(9.6%)	6(11.5%)	11(10.6%)
Total	52(100%)	52(100%)	104(100%)

P=0.581, Not significant, Chi-Square test

Figure 14: ECG examination distribution in two groups of study subjects:



TROP I (troponin I) - distribution:

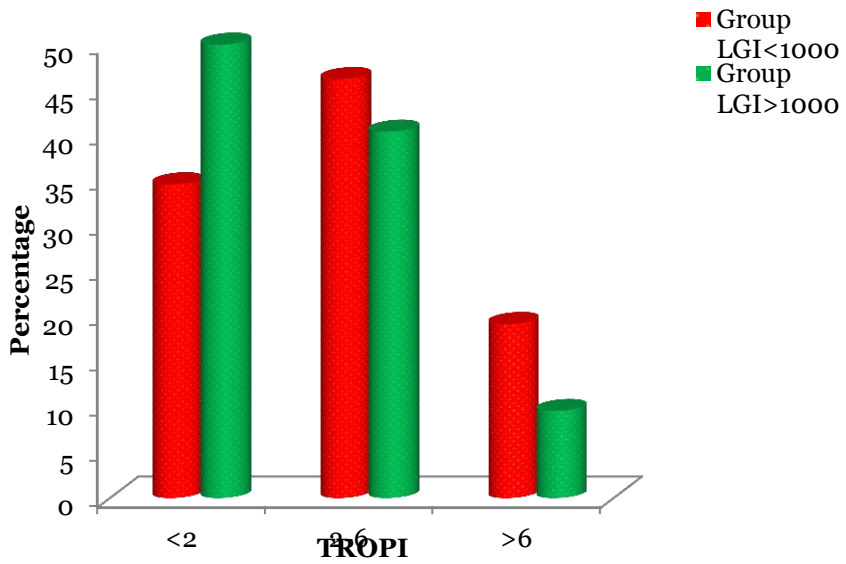
Of total, 44 (42.3%) had <2 troponin I, 45 (43.3%) had 2-6 troponin I and 15 (14.4%) had >6 troponin I in Group LGI<1000 and Group LGI>1000. P=0.116.

Table 9: TROPI- distribution in two groups of study subjects:

TROPI	LGI		Total
	Group LGI<1000	Group LGI>1000	
<2	18(34.6%)	26(50%)	44(42.3%)
2-6	24(46.2%)	21(40.4%)	45(43.3%)
>6	10(19.2%)	5(9.6%)	15(14.4%)
Total	52(100%)	52(100%)	104(100%)

P=0.116, Not significant, Chi-Square test

Figure 15: TROPI- distribution in two groups of patients studied:



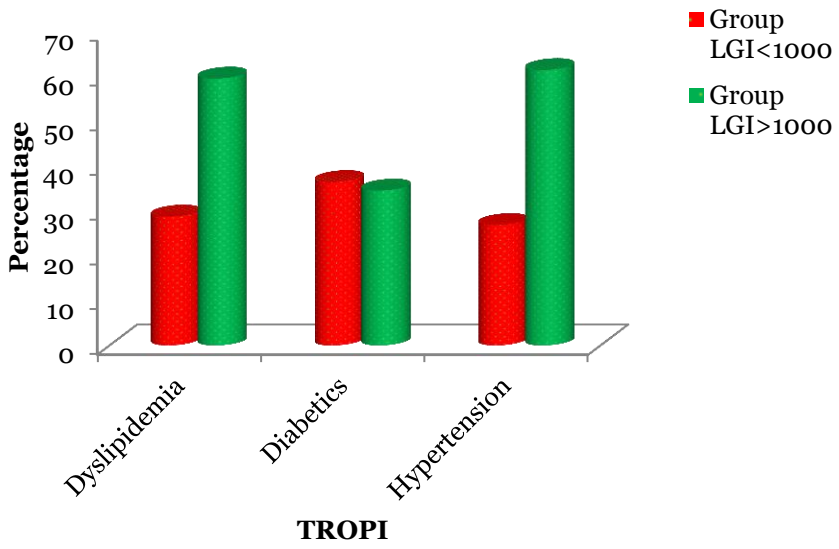
Incidence of Dyslipidemia, Diabetics and Hypertension distribution:

Of total, 46 (44.2%) had dyslipidemia, 37 (35.6%) had diabetics and 46 (44.2%) had hypertension in Group LGI<1000 and Group LGI>1000

Table 10: Incidence of Dyslipidemia, Diabetics and Hypertension distribution in two groups of study subjects

	LGI		Total (n=104)	P value
	Group LGI<1000 (n=52)	Group LGI>1000 (n=52)		
Dyslipidemia	15(28.8%)	31(59.6%)	46(44.2%)	0.002**
Diabetics	19(36.5%)	18(34.6%)	37(35.6%)	0.838
Hypertension	14(26.9%)	32(61.5%)	46(44.2%)	<0.001**

Figure 16: Incidence of Dyslipidemia, Diabetics and Hypertension distribution in two groups of study subjects



Prior MI (myocardial ischemia) distribution:

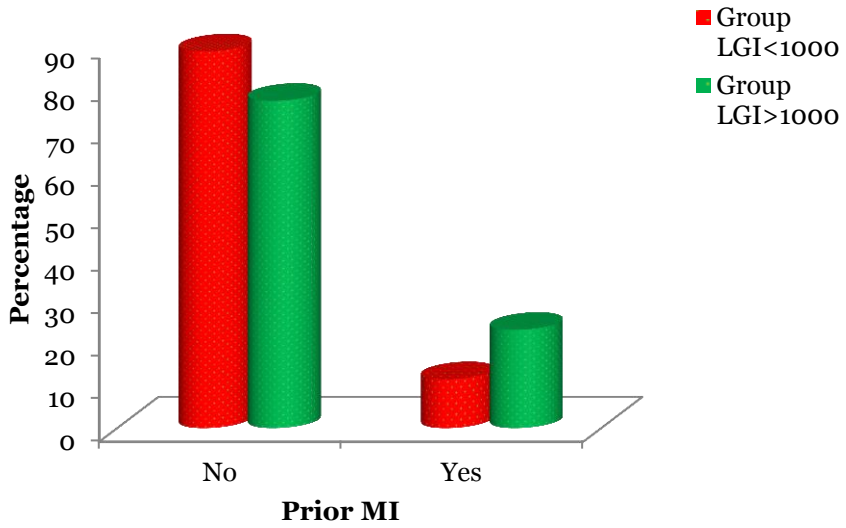
Of total, 86 (82.7%) had no prior MI and 18 (17.3%) had prior MI in Group LGI<1000 and Group LGI>1000. P=0.120.

Table 11: Prior MI distribution in two groups of patients studied

Prior MI	LGI		Total
	Group LGI<1000	Group LGI>1000	
No	46(88.5%)	40(76.9%)	86(82.7%)
Yes	6(11.5%)	12(23.1%)	18(17.3%)
Total	52(100%)	52(100%)	104(100%)

P=0.120, Not significant, Chi-Square test

Figure 17: Prior MI distribution in two groups of patients studied



H/O PCI and CABG distribution:

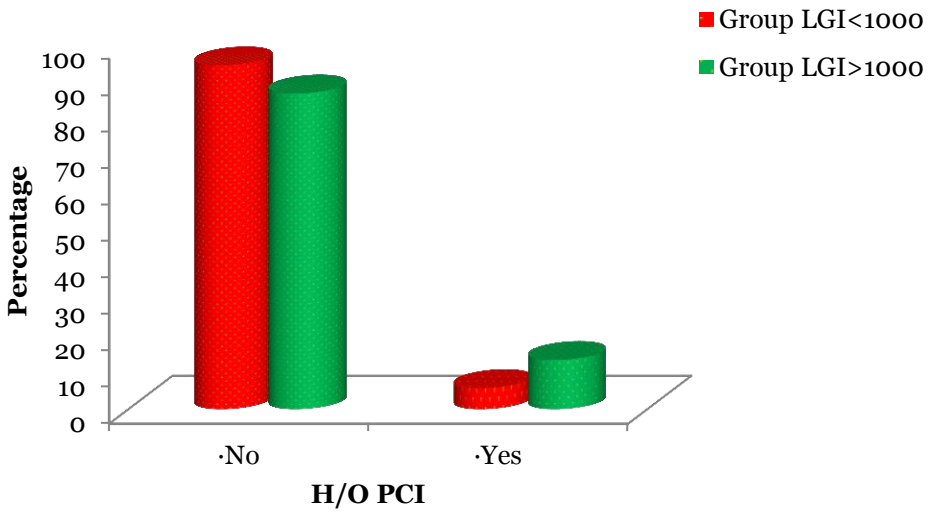
Of total, 94 (90.4%) had no H/O PCI and 10 (9.6%) had H/O PCI in Group LGI<1000 and Group LGI>1000.

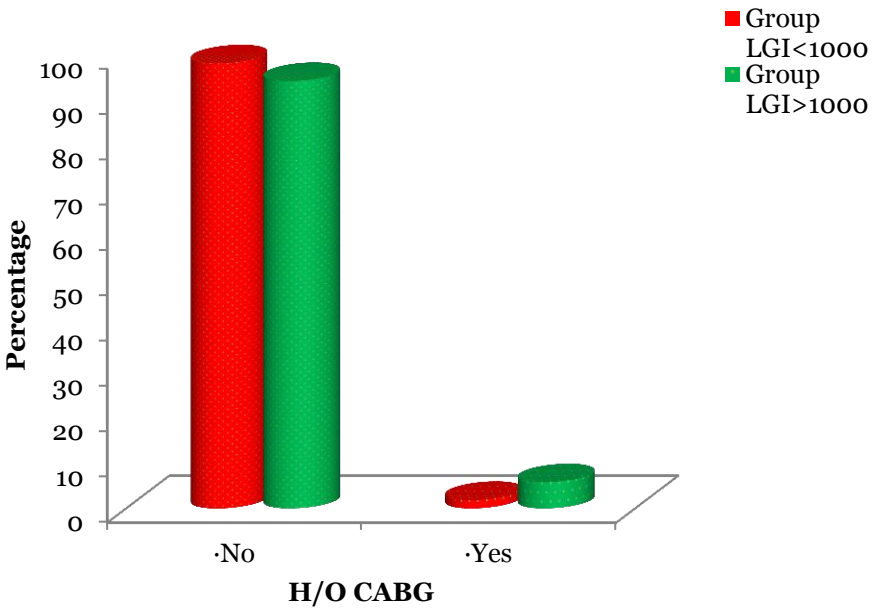
Of total, 100 (96.2%) had no H/O CABG and 4 (3.8%) had H/O CABG in Group LGI<1000 and Group LGI>1000.

Table 12: H/O PCI and CABG distribution in two groups of patients studied

	LGI		Total (n=104)	P value	
	Group LGI<1000 (n=52)	Group LGI>1000 (n=52)			
H/O PCI					
• No	49(94.2%)	45(86.5%)	94(90.4%)	0.183	
• Yes	3(5.8%)	7(13.5%)	10(9.6%)		
H/O CABG					
• No	51(98.1%)		49(94.2%)	100(96.2%)	0.308
• Yes	1(1.9%)		3(5.8%)	4(3.8%)	

Figure 18: H/O PCI and CABG distribution in two groups of patients studied




Group LGI<1000
Group LGI>1000
100
90
80
70
60
50
40
30
20
10
0
Percentage
·No
·Yes
H/O CABG

Fibrinolytics distribution:

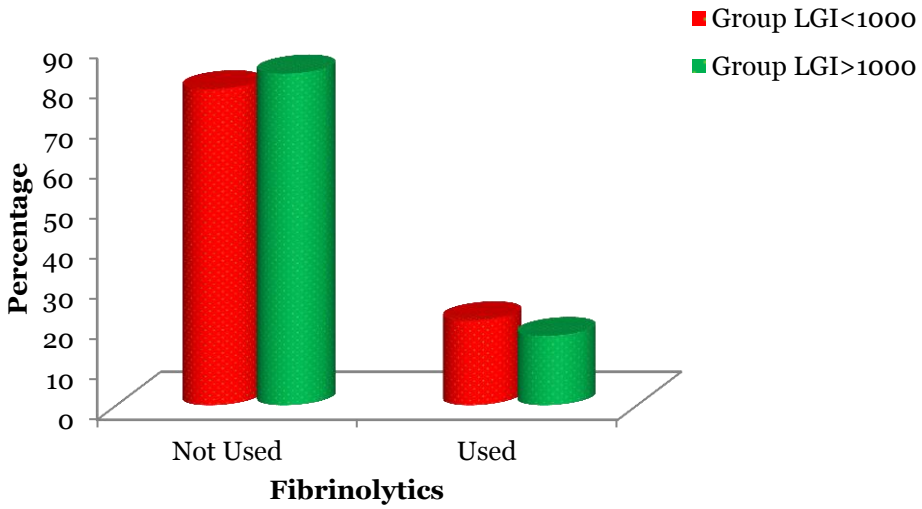
Of total, 84 (80.8%) had not used the Fibrinolytics, 20 (19.2%) had used Fibrinolytics in both the groups. P=0.619.

Table 13: Fibrinolytics distribution in two groups of study subjects

Fibrinolytics	LGI		Total
	Group LGI<1000	Group LGI>1000	
Not Used	41(78.8%)	43(82.7%)	84(80.8%)
Used	11(21.2%)	9(17.3%)	20(19.2%)
Total	52(100%)	52(100%)	104(100%)

P=0.619, Not significant, Chi-Square test

Figure 19: Fibrinolytics distribution in two groups of study subjects



PCI (Percutaneous Coronary Intervention) distribution:

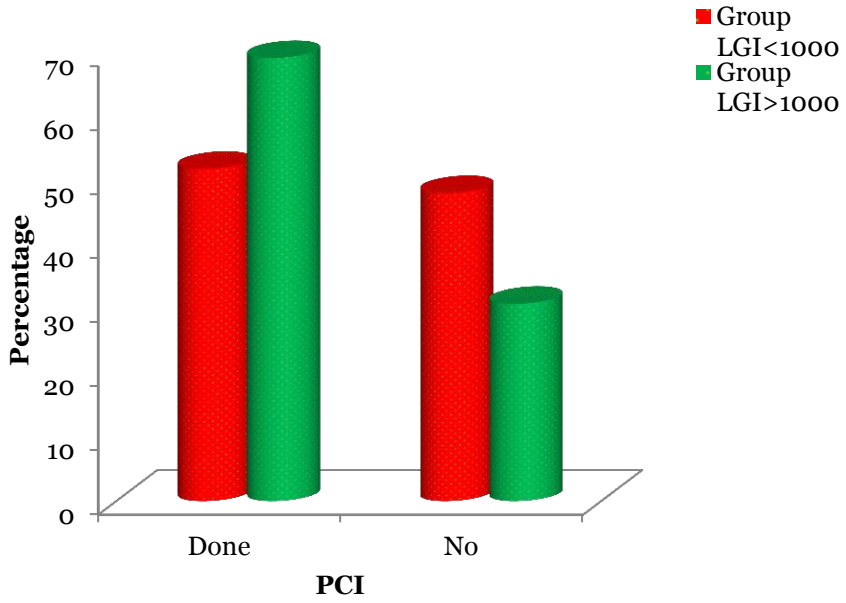
Of total, 63 (60.6%) had done with PCI and 41 (39.4%) had not done with PCI in Group LGI<1000 and Group LGI>1000.

Table 14: PCI distribution in two groups of study subjects:

PCI	LGI		Total
	Group LGI<1000	Group LGI>1000	
Done	27(51.9%)	36(69.2%)	63(60.6%)
No	25(48.1%)	16(30.8%)	41(39.4%)
Total	52(100%)	52(100%)	104(100%)

P=0.071+, significant, Chi-Square test

Figure 20: PCI distribution in two groups of study subjects:



AV Block 2/3 distribution:

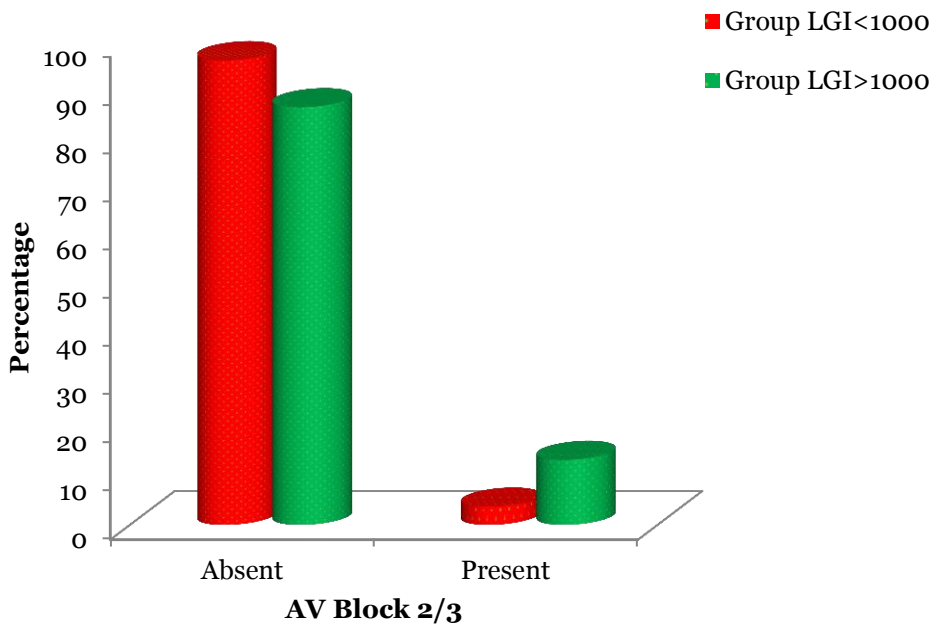
Of total, 95 (91.3%) had no/absent AV block 2/3 and 9 (8.7%) had AV Block 2/3 in Group LGI<1000 and Group LGI>1000. P=0.081.

Table 15: AV Block 2/3 distribution in two groups of study subjects:

AV Block 2/3	LGI		Total
	Group LGI<1000	Group LGI>1000	
Absent	50(96.2%)	45(86.5%)	95(91.3%)
Present	2(3.8%)	7(13.5%)	9(8.7%)
Total	52(100%)	52(100%)	104(100%)

P=0.081+, significant, Chi-Square test

Figure 21: AV Block 2/3 distribution in two groups of study subjects:



VT/VF (ventricular tachycardia/ ventricular fibrillation) distribution:

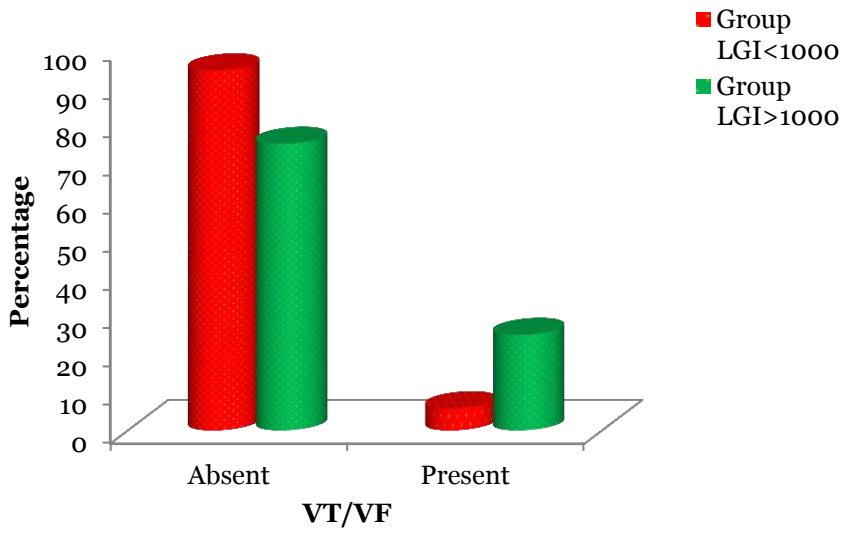
Of total, 88 (84.6%) had absence of VT/VF and 16 (15.4%) had VT/VF in Group LGI<1000 and Group LGI>1000. P=0.007.

Table 16: VT/VF distribution in two groups of study subjects:

VT/VF	LGI		Total
	Group LGI<1000	Group LGI>1000	
Absent	49(94.2%)	39(75%)	88(84.6%)
Present	3(5.8%)	13(25%)	16(15.4%)
Total	52(100%)	52(100%)	104(100%)

P=0.007**, significant, Chi-Square test

Figure 22: VT/VF distribution in two groups of study subjects:



KK34 (killip –kimball) distribution:

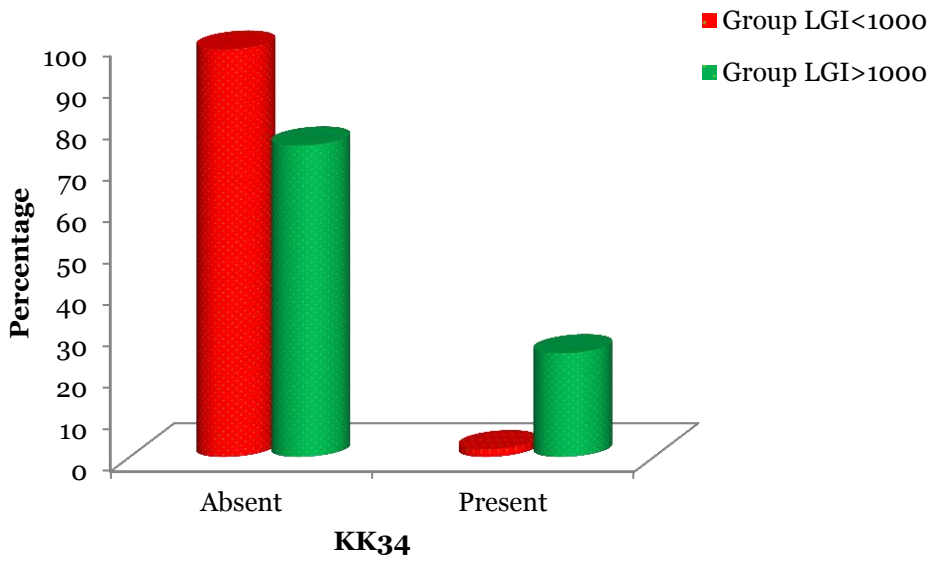
Of total, 90 (86.5%) had showed absence of **KK34 and** 14 (13.5%) had showed presence of **KK34 in** in Group LGI<1000 and Group LGI>1000. P=0.001.

Table 17: KK34 distribution in two groups of study subjects

KK34	LGI		Total
	Group LGI<1000	Group LGI>1000	
Absent	51(98.1%)	39(75%)	90(86.5%)
Present	1(1.9%)	13(25%)	14(13.5%)
Total	52(100%)	52(100%)	104(100%)

P=0.001**, significant, Chi-Square test

Figure 23: KK34 distribution in two groups of study subjects



Mortality distribution:

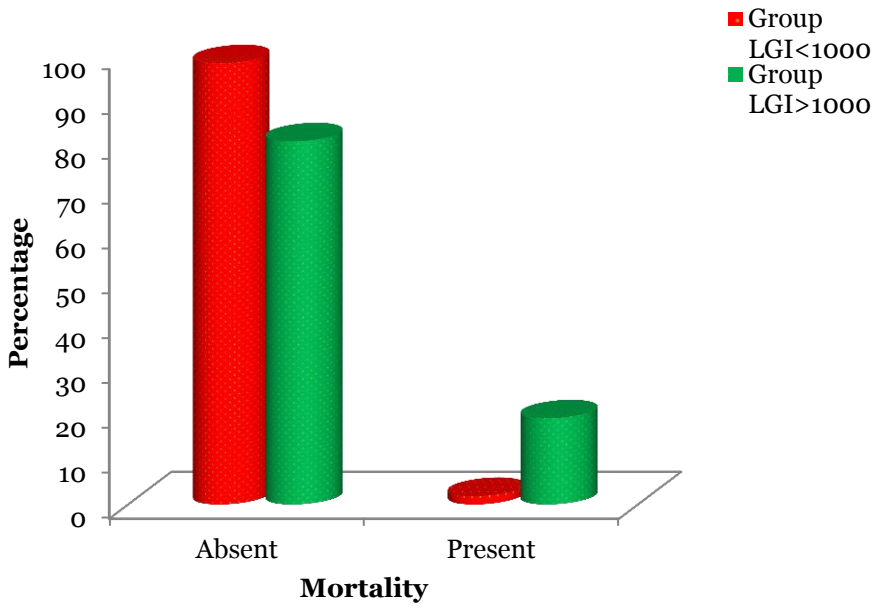
Of total, 93 (89.4%) had showed absence of mortality and 11 (10.6%) had showed presence of mortality distribution in Group LGI<1000 and Group LGI>1000. P=0.004.

Table 18: Mortality distribution in two groups of study subjects

Mortality	LGI		Total
	Group LGI<1000	Group LGI>1000	
Absent	51(98.1%)	42(80.8%)	93(89.4%)
Present	1(1.9%)	10(19.2%)	11(10.6%)
Total	52(100%)	52(100%)	104(100%)

P=0.004**, significant, Chi-Square test

Figure 24: Mortality distribution in two groups of study subjects



DISCUSSION

DISCUSSION

- Leon-Aliz E, et al., conducted a study on leuko-glycemic index as an in-hospital prognostic marker in patients with ST-segment elevation MI. Blood glucose and white blood cell count on admission showed prognostic significance in patients with MI; leuko-glycemic index, a recently proposed marker, still lacks enough knowledge about its value. Patients who had a poor outcome such as death, major cardiac complications and failed-thrombolysis, showed higher values of leuko-glycemic index ($p<0.01$), which was associated with several variables such as Killip class and heart rate on admission ($p=0.000$). They obtained a cut-off point of 1.158, patients with higher values had 3 times higher probability of death and complications (odds ratio=3,0; IC 95%: 1,2-7,3; $p=0.005$); so leuko-glycemic index was an independent predictor after multivariate analysis. The leuko-glycemic index was associated with an increased occurrence of hospital complications, death and failed-thrombolysis; its pathological value was an independent predictor of in-hospital death and complications in the studied sample.[11]

- Saldaña AM, et al., evaluated the association of the leuko-glycemic index and complications in patients with ACS. This was a descriptive, cross-sectional, retrospective study. It was performed in 34 patients diagnosed with ACS, acute MI with ST segment elevation, acute MI without ST segment elevation and unstable angina, who came to the Intensive Care Unit from May 1, 2016 to May 31, 2017. Clinical data were recorded during the first 72 hours of the event, as well as the laboratory results, which included glycemia and leukocyte count at admission. From these data, the leuko-glycemic index was calculated and its prognostic value was evaluated by the use of the $\chi 2$ test. The leuko-glycemic index was applied to the

population under study and four groups were formed according to the classification obtained: grade I, 0-800 points; grade II, 801-1600 points; grade III, 1601-2400 points; and grade IV,

more than 2400 points. The highest prevalence was found in group 2 (800-1601 points), with 38.24%, followed by group 4 (>2400 points), with 35.29%. The mortality at 72 hours was 23.53%. Arrhythmias occurred in 50% of the case. There was no statistically significant correlation ($p>0.05$) between the leuko-glycemic index and complications.[12]

- Ascaso JF., conducted a study on leuko-glycemic index in ST elevation acute MI, a simple and useful parameter in the predicting complications.[13]

- Diaz Benítez RE, et al., conducted a study on glycosylated hemoglobin and leuko-glycemic index as prognostic determinations in ACS. The ACS is one of the most frequent causes of morbidity and mortality globally, that is why it is important to find laboratory determinations of easy access, to help evaluating the prognosis of these patients. This was a cross-sectional descriptive study carried out in 142 diabetic and non-diabetic patients, with ACS, admitted to the Hospital Universitario Dr. Celestino Hernández Robau of Santa Clara, Cuba, from October 2012 to October 2013. The glycosylated hemoglobin, leuko-glycemic index, and complications after admission were evaluated during the study. A total of 40 diabetic and 102 non-diabetic patients were detected, with a mean age of 68.2 years and a prevalence of hypertension and dyslipidemia. As the leuko-glycemic index numbers increased, the frequency of complications in diabetics ($p=0.422$) and non-diabetics ($p=0.007$) also increased. The mean glycosylated hemoglobin of complicated diabetics (8.8%) was higher than that

of non-diabetics (7.5%) ($p<0.01$). The 1443 of leuko-glycemic index values and 6.9% of glycosylated hemoglobin were established as complication predictors. The joint evaluation of leuko-glycemic index and glycosylated hemoglobin was a predictor of high specificity and good sensitivity in both groups of study.[14]

SUMMARY

SUMMARY

- This Comparative two group clinical study was conducted in 104 patients of both sexes
- Of the total 104 patients included in the study, 52 patients had included in the Group LGI<1000 and 52 patients had included in the Group LGI>1000.
- The age of patients ranged from less than 50 to more than 80 years. The patients in the study were divided into 5 age groups, viz., less than 50 years, between 50 to 60 years, between 61 to 70 years, between 71 to 80 years and more than 80 years.
- Among all, there were 1 (1.9%) and 3 (5.8%) in Group LGI<1000 and Group LGI>1000 had age less than 50; 11 (21.2%) and 14 (26.9%) in Group LGI<1000 and Group LGI>1000 had in between the age 50-60; 23 (44.2%) and 22 (42.3%) in Group LGI<1000 and Group LGI>1000 had age in between 61-70; 14 (26.9%) and 13 (25%) in Group LGI<1000 and Group LGI>1000 had age in between 71-80; and 3 (5.8%) in Group LGI<1000 had an age group of more than 80.
- Of all the patients included in the study, 4 (7.7%) were female in the Group LGI>1000; 52 (100%) and 48 (92.3%) were males in the Group LGI<1000 and Group LGI>1000.
- Of total, 49 (94.2) and 45 (86.5%) had no Systolic Blood Pressure in Group LGI<1000 and Group LGI>1000; 3 (5.8%) and 7 (13.5%) had Systolic Blood Pressure in Group LGI<1000 and Group LGI>1000. P=0.483.
- Of total, 81 (77.9%) had showed no heart rate and 23 (22.1%) had showed heart rate in both the groups. P= 0.057.
- Of total, 31 (29.8%) had <100 in fasting blood glucose level in both the groups, 36 (34.6%) had 100-126 in in both the groups and 37 (35.6%) had >126 in both the groups of fasting blood levels.

- Of total, 36 (34.6%) had ST elevation in I, aVL, 45 (43.3%) had ST elevation in II, III, aVF, v1-v3 and 12 (11.5%) had St elevation in v1 to v4 and 11 (10.6%) had St elevation in v3- v6 in Group LGI<1000 and Group LGI>1000. P=0.581.
- Of total, 46 (44.2%) had dyslipidemia, 37 (35.6%) had diabetics and 46 (44.2%) had hypertension in Group LGI<1000 and Group LGI>1000
- Of total, 86 (82.7%) had no prior MI and 18 (17.3%) had prior MI in Group LGI<1000 and Group LGI>1000. P=0.120.
- Of total, 94 (90.4%) had no H/O PCI and 10 (9.6%) had H/O PCI in Group LGI<1000 and Group LGI>1000.
- Of total, 100 (96.2%) had no H/O CABG and 4 (3.8%) had H/O CABG in Group LGI<1000 and Group LGI>1000.
- Of total, 63 (60.6%) had done with PCI and 41 (39.4%) had not done with PCI in Group LGI<1000 and Group LGI>1000.
- Of total, 95 (91.3%) had no/absent AV block 2/3 and 9 (8.7%) had AV Block 2/3 in Group LGI<1000 and Group LGI>1000. P=0.081.
- Of total, 88 (84.6%) had absence of VT/VF and 16 (15.4%) had VT/VF in Group LGI<1000 and Group LGI>1000. P=0.007.
- Of total, 93 (89.4%) had showed absence of mortality and 11 (10.6%) had showed presence of mortality distribution in Group LGI<1000 and Group LGI>1000. P=0.004.

CONCLUSION

CONCLUSION:

Leukocytosis and hyperglycemia are associated with worse short-term prognosis in patients with ACS, but their new relationship, called leuko-glycemic index, has been scarcely evaluated.[7]

The leuko-glycemic index is a simple, low cost tool allowing re-stratification of non-diabetic patients with low TIMI score, at higher risk of death or severe HF.[7]

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ANNEXURES

PROFORMA

1. IP No: 2. Date:

3.Serial No:

4. Name:

5. Age:

6.Gender:

7. Occupation:

8. Date of admission:

9. Address

10. phone no:

11. Chief complaints:

12. Past history:

13. Drug/ Treatment therapy:

14. Personal history:

15. General physical examination(at admission):

PR: BP: Temp:

Resp rate: SpO_2:

Pallor: Icterus: Cyanosis:

Clubbing: Lymphadenopathy: Oedema:

16. Systemic examination:

CVS:
RS:

PA:
CNS:

17. Diagnosis:
18. Duration of hospital stay:

19. Type of devices used :

20. Procedures done:

21. INVESTIGATIONS:

- COMPLETE BLOOD COUNT:

- RENAL FUNCTION TEST:

- URINE ROUTINE :

- ESR: CRP: RBS:
- CXR:
- ECG:
- TROP I:

SIGNATURE

INFORMED CONSENT FORM

Name of the investigator: DR. P SAMBASIVA RAO

Name of the organisation: R L JALAPPA HOSPITAL AND RESEARCH CENTRE ATTACHED TO SRI DEVARAJ URS MEDICAL COLLEGE

Name of the participant: SI no:

I Mr./Mrs. have been explained in my own understandable language, that I will be included in a study which is –Prognostic Value of the Leuko-glycemic Index in Acute Myocardial Infarction." being conducted in RL JALAPPA HOSPITAL.

I have been invited to take part in this research study. The information in this document is meant to help me decide whether or not to take part. I have clarified my doubts regarding this study with the principal investigator.

I have been asked to participate in this study because I satisfy the eligibility criteria .

I request and authorise Dr. P SAMBASIVA RAO to perform the designated tests for my blood sample. My signature below constitutes my acknowledgement that the benefits, risks and limitations of this testing have been explained to my satisfaction by a qualified health professional.

Participation is totally voluntary and there would be no payment for sample collection. All test results are treated with medical confidentiality and will not be disclosed to any outsider except if it is required by the law.

I give my consent to allow my sample to be used for medical research, test validation or education as long as my privacy is maintained.

I understand that I remain free to withdraw from this study at any time and this will not change my future care.

I have read and received a copy of patient information sheet. I understand the information provided in this document and I have had the opportunity to ask questions I might have about the testing, the procedure, the associated risk and alternatives.

Subject name and signature/ thumb impression Date:

Parent's/ guardian's name/ thumb impression Date:

Signature of the person taking consent Date:

ಮಾಹಿತಿಯುಕ್ತಸಮ್ಮತಿಯನಮೂನೆ

ಸಂಶೋಧಕರಹೆಸರು: ಡಿಆರ್. ಪಿ ಸಂಬಶಿವಾ ರಾ

ಸಂಸ್ಥೆಯಹೆಸರು: ಶ್ರೀ ದವರಾಜ್ಯು ಆರ್ಯಸ್ಮೈಡಿಕಲ್ಕಾಲೇಜ್ಗೆಸೇರ್ಪಡೆಗೊಂಡಆರ್ಯಲ್ಜಲಪಪಾಆಸ್ಪತ್ರೆಮತ್ತುಸಂಶೋಧನಾಕೇಂದ್ರ
ಪಾಲ್ಗೊಳ್ಳುವವರಹೆಸರು: SI ಸಂಖ್ಯೆ:

ನಾನುಶ್ರೀ / ಶ್ರೀ. ನನ್ನಸ್ವಂತಅರ್ಥವಾಗುವಂತಹಭಾಷೆಯಲ್ಲಿವಿವರಿಸಲ್ಪಟ್ಟಿದೆ, ನಾನುಆರ್ಯಲ್ಜಲಾಪಾಆಸ್ಪತ್ರೆಯಲ್ಲಿನಡೆಸಿದ
"ತೀವ್ರಮಯೋಕಾರ್ಡಿಯಲ್ಇನ್ಫಾರ್ಕ್ಷನ್ನಲ್ಲಿಲ್ಯುಕೋ-ಗ್ಲೈಸೆಮಿಕ್ಸೂಚ್ಯಂಕದಪ್ರೊಗ್ನೋಸ್ಟಿಕ್ಮೌಲ್ಯ"
ವನ್ನುಒಳಗೊಂಡಿರುವಅಧ್ಯಯನದಲ್ಲಿಸೇರಿಸಲಾಗುವುದು.

ಈಸಂಶೋಧನಾಅಧ್ಯಯನದಲ್ಲಿಪಾಲ್ಗೊಳ್ಳಲುನನಗೆಆಹ್ವಾನಿಸಲಾಗಿದೆ.
ಈಡಾಕ್ಯುಮೆಂಟ್ನಲ್ಲಿನಮಾಹಿತಿಯುಪಾಲ್ಗೊಳ್ಳಲುಇಲ್ಲವೇಎಂಬುದನ್ನುನಿರ್ಧರಿಸಲುನನಗೆಸಹಾಯಮಾಡುವಉದ್ದೇಶವಾಗಿದೆ.
ಪ್ರಧಾನಸಂಶೋಧಕನೊಂದಿಗೆನಾನುಈಅಧ್ಯಯನಕ್ಕೆಸಂಬಂಧಿಸಿದಂತೆನನ್ನಅನುಮಾನಗಳನ್ನುಸ್ಪಷ್ಟಪಡಿಸಿದೆ.

ನಾನುಈಅಧ್ಯಯನದಲ್ಲಿಪಾಲ್ಗೊಳ್ಳುವಂತೆಕೇಳಿದೆಏಕೆಂದರೆನಾನುಅರ್ಹತಾಮಾನದಂಡಗಳನ್ನುಪೂರೈಸುತ್ತೇನೆ.

ನನ್ನರಕ್ತದಮಾದರಿಗಾಗಿಗೊತ್ತುಪಡಿಸಿದಪರೀಕ್ಷೆಗಳನ್ನುನಡೆಸಲುನಾನು ಡಾ.ಪಿ.ಸಂಬಾಶಿವಾ ರಾ
ವೊನನ್ನುವಿನಂತಿಸುತ್ತೇನೆಮತ್ತುಪ್ರಮಾಣೀಕರಿಸುತ್ತೇನೆ. ಕೆಳಗಿನನನ್ನಸಹಿನನ್ನಅರ್ಹತೆಯನ್ನುಹೊಂದಿದ್ದು, ಈಪರೀಕ್ಷೆಯಪ್ರಯೋಜನಗಳು,
ಅಪಾಯಗಳುಮತ್ತುಮಿತಿಗಳನ್ನುಅರ್ಹಆರೋಗ್ಯವೃತ್ತಿಪರರಿಂದನನ್ನತೃಪ್ತಿಗೆವಿವರಿಸಲಾಗಿದೆ.

ಭಾಗವಹಿಸುವಿಕೆಸಂಪೂರ್ಣವಾಗಿಸ್ವಯಂಪ್ರೇರಿತವಾಗಿರುತ್ತದೆಮತ್ತುಮಾದರಿಸಂಗ್ರಹಣೆಗೆಯಾವುದೇಪಾವತಿಯಿಲ್ಲ.
ಎಲ್ಲಾಪರೀಕ್ಷಾಫಲಿತಾಂಶಗಳನ್ನುವೈದ್ಯಕೀಯಗೌಪ್ಯತೆಯೊಂದಿಗೆಪರಿಗಣಿಸಲಾಗುತ್ತದೆಮತ್ತುಕಾನೂನಿನಅಗತ್ಯವಿದ್ದರೆಹೊರತುಪಡಿಸಿಯಾ
ವುದೇಹೊರಗಿನವರಿಗೆಬಹಿರಂಗಪಡಿಸುವುದಿಲ್ಲ.

ನನ್ನಗೌಪ್ಯತೆನಿರ್ವಹಿಸಲ್ಪಡುವವರೆಗೆವೈದ್ಯಕೀಯಪರೀಕ್ಷೆ,
ಪರೀಕ್ಷೆಮೌಲ್ಯಮಾಪನಅಥವಾಶಿಕ್ಷಣಕ್ಕಾಗಿನನ್ನಮಾದರಿಯನ್ನುಬಳಸಲುಅನುಮತಿಸಲುನನ್ನಒಪ್ಪಿಗೆಯನ್ನುನಾನುನೀಡುತ್ತೇನೆ.

ಈಅಧ್ಯಯನದಿಂದನಾನುಯಾವುದೇಸಮಯದಲ್ಲಿಹಿಂತೆಗೆದುಕೊಳ್ಳಲುಮುಕ್ತವಾಗಿರುತ್ತೇನೆಮತ್ತುಇದುನನ್ನಮುಂದಿನಕಾಳಜಿಯನ್ನುಬದಲಿ
ಸುವುದಿಲ್ಲಎಂದುನಾನುಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆ.

ರೋಗಿಯಮಾಹಿತಿಹಾಳೆಯನ್ನುನಾನುಓದಿದ್ದೇನೆಮತ್ತುಸ್ವೀಕರಿಸಿದ್ದೇನೆ.
ಈಡಾಕ್ಯುಮೆಂಟಿನಲ್ಲಿಒದಗಿಸಿದಮಾಹಿತಿಯನ್ನುನಾನುಅರ್ಥಮಾಡಿಕೊಂಡಿದ್ದೇನೆಮತ್ತುಪರೀಕ್ಷೆ, ಕಾರ್ಯವಿಧಾನ,
ಸಂಬಂಧಿಸಿದಅಪಾಯಮತ್ತುಪರ್ಯಾಯಗಳಬಗ್ಗೆನಾನುಹೊಂದಿರುವಪ್ರಶ್ನೆಗಳನ್ನುಕೇಳಲುನನಗೆಅವಕಾಶವಿದೆ.

ವಿಷಯಹೆಸರುಮತ್ತುಸಹಿ / ಹೆಬ್ಬೆರಳುಗುರುತು: ದಿನಾಂಕ:

ಪೋಷಕರ / ಪೋಷಕರಹೆಸರು / ಹೆಬ್ಬೆರಳುಗುರುತು: ದಿನಾಂಕ:

ಒಪ್ಪಿಗೆತೆಗೆದುಕೊಳ್ಳುವವ್ಯಕ್ತಿಯಸಹಿ: ದಿನಾಂಕ:

PATIENT INFORMATION SHEET

Study title: Prognostic Value of the Leuko-glycemic Index in Acute Myocardial Infarction.

Study location: R L Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar.

Details- Acute coronary syndrome is the greatest single cause of mortality and loss of disability-adjusted life years (DALYs) worldwide, accounting for roughly 7 million deaths and 129 million DALYs annually.

The purpose of the study is to identify acute coronary syndrome and risk of reinfarction,heart failure,death by leuko-glycemic index.

Numerous markersare available for evaluation and prognostication of Acute coronary syndrome, such as C-reactive protein (CRP), the complement system, myeloperoxidase,troponin and interleukin 6 (IL-6). However, cost factor and availability are the hurdles for implementation of these markers in day today practice, especially in developing countries.

As Leuko-glycemicindex is simple to calculate with available low cost investigations, leucocyte count and fasting blood glucose values, it could be useful in low complexity centers with no accessibility to standard investigation, troponin marker.

A history of AMI and risk factors such as age, hypertension, diabetes,high blood chelestrol,obesity,stress smoking, will be enquired, leukoglycemic index will be measured at admission in all patients ,All patients will undergo ECG, screening 2D ECHO ,CARDIAC MARKERS, CBC, RFT, SERUM ELECTROLYTES, RBS,FBS, LIPID PROFILE ,CXR,All patients will be followed up at the hospital.

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study we will collect information (as per proforma) from you or a person responsible for you or both. Relevant history will be taken. This information collected will be used only for dissertation and publication.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the Institutional Ethics Committee.

There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

For further information contact

Dr. P SAMBASIVA RAO (Post graduate)

Department of General Medicine

SDUMC , KOLAR

Contact NO : 9581434314

ತಾಳ್ಮೆ ಮಾಹಿತಿ ಶೀಟ್

ಅಧ್ಯಯನ ಶೀರ್ಷಿಕೆ: ಅಕ್ಯುಟ್ ಮಯೋಕಾರ್ಡಿಯಲ್ ಇನ್ಫಾರ್ಕ್ಷನ್ನಲ್ಲಿ ಲ್ಯುಕೊ-ಗ್ಲೈಸೆಮಿಕ್ ಸೂಚ್ಯಂಕದ ಪ್ರೊಗ್ನೋಸ್ಟಿಕ್ ಮೌಲ್ಯ.

ಅಧ್ಯಯನ ಸ್ಥಳ: ಆರ್ ಎಲ್ ಜಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ ಶ್ರೀ ಕೋರಾರ್ನ ತಮಾಕ ಶ್ರೀ ದೇವರಾಜ್ ಉರ್ಸ್ ಮೆಡಿಕಲ್ ಕಾಲೇಜಿನಲ್ಲಿ ಜೋಡಿಸಲಾಗಿದೆ.

ವಿವರಗಳು- ತೀವ್ರ ಪರಿಧಮನಿಯ ಸಿಂಡ್ರೋಮ್ ಮರಣದ ದೊಡ್ಡ ಏಕೈಕ ಕಾರಣವಾಗಿದೆ ಮತ್ತು ವಿಶ್ವಾದ್ಯಂತ ಅಸಾಮರ್ಥ್ಯ-ಸರಿಹೊಂದಿಸಲಾದ ಜೀವಿತಾವಧಿಯ (DALYs) ನಷ್ಟ, ಸುಮಾರು 7 ದಶಲಕ್ಷ ಸಾವುಗಳು ಮತ್ತು 129 ದಶಲಕ್ಷ ಡಾಲಿಗಳನ್ನು ವಾರ್ಷಿಕವಾಗಿ ಲೆಕ್ಕಹಾಕುತ್ತದೆ.

ತೀವ್ರ ಪರಿಧಮನಿಯ ಸಿಂಡ್ರೋಮ್ ಮತ್ತು ಮರುಫಾರ್ಕ್ಷನ್, ಹೃದಯ ವೈಫಲ್ಯ, ಲ್ಯುಕೋ-ಗ್ಲೈಸೆಮಿಕ್ ಸೂಚ್ಯಂಕ ರ ಮರಣದ ಅಪಾಯವನ್ನು ಗುರುತಿಸುವುದು

ಈ ಅಧ್ಯಯನದ ಉದ್ದೇಶವಾಗಿದೆ. ಸಿ-ರಿಯಾಕ್ಟೀವ್ ಪ್ರೋಟೀನ್ (ಸಿಆರ್ಪಿ), ಪೂರಕ ವ್ಯವಸ್ಥೆ, ಮೈಯೊರೊಪೊಕ್ಸಿಕ್ಸಿಡೆಸ್, ಟ್ರೊಪೋನಿನ್ ಮತ್ತು ಇಂಟರ್ಲ್ಯೂಕಿನ್ 6 (ಐಎಲ್ -6) ಮುಂತಾದ ತೀವ್ರ ಪರಿಧಮನಿಯ ಸಿಂಡ್ರೋಮ್ನ ಮೌಲ್ಯಮಾಪನ ಮತ್ತು ಪ್ರಚ್ಛೆಗೆ ಸಂಬಂಧಿಸಿದಂತೆ ಹಲವಾರು ಗುರುತುಗಳು ಲಭ್ಯವಿವೆ. ಆದಾಗ್ಯೂ, ವೆಚ್ಚದ ಅಂಶ ಮತ್ತು ಲಭ್ಯತೆಯು ಈ ಮಾರ್ಕರ್ಗಳನ್ನು ಅನುಷ್ಠಾನಗೊಳಿಸಲು ಅಡಚಣೆಗಳಾಗಿವೆ,

ಇಂದು ದಿನಂಪ್ರತಿ ಅಭ್ಯಾಸದಲ್ಲಿ, ವಿಶೇಷವಾಗಿ ಅಭಿವೃದ್ಧಿಶೀಲ ರಾಷ್ಟ್ರಗಳಲ್ಲಿ. ಲಭ್ಯವಿರುವ ಕಡಿಮೆ ವೆಚ್ಚದ ತನಿಖೆಗಳು, ಲ್ಯುಕೋಸೈಟ್ ಕೌಂಟ್ ಮತ್ತು ಉಪವಾಸ ರಕ್ತ ಗ್ಲುಕೋಸ್ ಮೌಲ್ಯಗಳೊಂದಿಗೆ ಲೆಕ್ಕಹಾಕಲು ಲ್ಯುಕೋ-ಗ್ಲೈಸೆಮಿಕ್ಇಕ್ಸ್ ಸರಳವಾಗಿದ್ದು, ಇದು ಪ್ರಮಾಣಿತ ತನಿಖೆ, ಟ್ರೋಪೋನಿನ್ಗೆ ಯಾವುದೇ ಪ್ರವೇಶಸಾಧ್ಯವಿಲ್ಲದೇ ಕಡಿಮೆ ಸಂಕೀರ್ಣತೆ ಕೇಂದ್ರಗಳಲ್ಲಿ ಉಪಯುಕ್ತವಾಗಿದೆ. ಎಎಮ್ಐ ಮತ್ತು ವಯಸ್ಸು, ಅಧಿಕ ರಕ್ತದೊತ್ತಡ, ಮಧುಮೇಹ, ಅಧಿಕ ರಕ್ತದೊತ್ತಡ, ಸ್ಥೂಲಕಾಯತೆ, ಒತ್ತಡದ ಧೂಮಪಾನ ಮುಂತಾದ ಅಪಾಯಕಾರಿ ಅಂಶಗಳು ವಿಚಾರಣೆಗೆ ಒಳಗಾಗುತ್ತವೆ, ಎಲ್ಲಾ ರೋಗಿಗಳಲ್ಲಿ ಪ್ರವೇಶಾನುಗುಣವಾಗಿ ಲ್ಯೂಕೋಗ್ಲೈಸೆಮಿಕ್

ಸೂಚಿಯನ್ನು ಅಳತೆ ಮಾಡಲಾಗುವುದು, ಎಲ್ಲಾ ರೋಗಿಗಳು ಇಸಿಜಿಗೆ ಒಳಗಾಗುತ್ತಾರೆ, 2D ಇಕೊ, ಕಾರ್ಡಿಯಾಕ್ ಮಾರ್ಕರ್ಸ್, ಸಿಬಿಸಿ, ಆರ್ಎಫ್ಟಿ, ಸೆರುಮ್ ವಿದ್ಯುನ್ಮಾನ, ಆರ್ಬಿಎಸ್, ಎಫ್ಬಿಎಸ್, ಲಿಪಿಡ್ ಪ್ರೊಫೈಲ್, ಸಿಎಕ್ಸ್ಆರ್, ಎಲ್ಲಾ ರೋಗಿಗಳನ್ನು ಆಸ್ಪತ್ರೆಯಲ್ಲಿ ಹಿಂಬಾಲಿಸಲಾಗುತ್ತದೆ.

ಕೆಳಗಿನ ಮಾಹಿತಿಯನ್ನು ಓದಿ ಮತ್ತು ನಿಮ್ಮ ಕುಟುಂಬ ಸದಸ್ಯರೊಂದಿಗೆ ಚರ್ಚಿಸಿ. ಅಧ್ಯಯನದ ಬಗ್ಗೆ ನೀವು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಬಹುದು. ನೀವು ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಒಪ್ಪಿಕೊಂಡರೆ ನಾವು ನಿಮ್ಮಿಂದ (ಮಾಹಿತಿ ಪ್ರಕಾರ) ಮಾಹಿತಿಯನ್ನು ಅಥವಾ ನಿಮ್ಮ ಅಥವಾ ಎರಡಕ್ಕೂ ಜವಾಬ್ದಾರರಾಗಿರುವ ವ್ಯಕ್ತಿಗಳನ್ನು ಸಂಗ್ರಹಿಸುತ್ತೇವೆ. ಸಂಬಂಧಿತ ಇತಿಹಾಸವನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುವುದು. ಸಂಗ್ರಹಿಸಿದ ಈ ಮಾಹಿತಿಯನ್ನು ಪ್ರೌಢಪ್ರಬಂಧ ಮತ್ತು ಪ್ರಕಟಣೆಗಾಗಿ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ.

ನಿಮ್ಮಿಂದ ಸಂಗ್ರಹಿಸಿದ ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ಗೌಪ್ಯವಾಗಿರಿಸಲಾಗುವುದು ಮತ್ತು ಯಾವುದೇ ಹೊರಗಿನವರಿಗೆ ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ನಿಮ್ಮ ಗುರುತನ್ನು ಬಹಿರಂಗಪಡಿಸಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನವು ಸಾಂಸ್ಥಿಕ ನೀತಿಶಾಸ್ತ್ರ ಸಮಿತಿಯಿಂದ ಪರಿಶೀಲಿಸಲ್ಪಟ್ಟಿದೆ ಮತ್ತು ನೀವು ಸಂಸ್ಥೆಯ ಎಥಿಕ್ಸ್ ಸಮಿತಿಯ ಸದಸ್ಯರನ್ನು ಸಂಪರ್ಕಿಸಲು ಮುಕ್ತವಾಗಿರುತ್ತೀರಿ

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ಈ ಅಧ್ಯಯನಕ್ಕೆ ಒಪ್ಪಿಗೆ ನೀಡಲು ಯಾವುದೇ ಕಡ್ಡಾಯವಿಲ್ಲ. ನೀವು ಭಾಗವಹಿಸಲು ಬಯಸದಿದ್ದರೆ ನೀವು ಪಡೆಯುವ ಕಾಳಜಿ ಬದಲಾಗುವುದಿಲ್ಲ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನೀವು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಕೊಳ್ಳುವುದಾದರೆ ಮಾತ್ರ ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆಗೆ ನೀವು ಸಹಿ / ನೀಡಬೇಕಾಗಿದೆ.

ಹೆಚ್ಚಿನ ಮಾಹಿತಿಗಾಗಿ

ಡಾ. ಪಿ. ಸಮ್ಬಶಿವಾ ರಾವೊ (ಪೋಸ್ಟ್ ಪದವಿ) ಜನರಲ್ ಮೆಡಿಸಿನ್ ಇಲಾಖೆ,

SDUMC,ಕೋಲಾರ್ ,ಸಂಪರ್ಕ ಸಂಖ್ಯೆ: 9581434314

MASTER CHART

Name	Age	Sex	UHID	SBP(<100mm hg)	HR(>100)	FBS	LEUKOCYTE COUNT	LGI	ECG	TROP I	DYSLIPIDEMIA	DIABETES	HYPERTENSION	PRIOR MI	H/O PCI	H/O CABG	FIBRINOLYTICS	PCI	2/3 DEGREE AV BLOCK	VT/VF	KK 3-4	DEATH
Anjanappa	47	M	600669	NO	NO	102	6.23	635	ST ELEVATION IN I, AVL	6.93	ABSENT	NO	NO	NO	NO	NO	NOT USED	NO	ABSENT	ABSENT	ABSENT	ABSENT
Dhyavenappa	53	M	615950	NO	NO	128	8.45	1081	ST ELEVATION IN II,III, AVF	3.13	present	NO	YES	NO	NO	NO	NO	DONE	present	ABSENT	ABSENT	ABSENT
Srinivas	58	M	599282	NO	YES	146	9.12	1328	ST ELEVATION IN V1 TO V4	2.34	PRESENT	YES	YES	YES	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	PRESENT
Narayanappa	75	M	600070	NO	NO	98	10.67	1038	ST ELEVATION IN I, AVL, V4-V6	0.96	PRESENT	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
Narayana swamy	74	M	599686	NO	YES	142	5.98	849	ST ELEVATION IN II,III, AVF	1.06	absent	YES	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
Hanumanthaiah	68	M	617525	NO	NO	187	4.62	863	ST ELEVATION IN II, III, AVF, V1-V3	4.59	ABSENT	YES	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
Krishna	59	M	522677	NO	NO	93	9.45	878	ST ELEVATION IN I, AVL	7.66	PRESENT	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
Raghuram reddy	72	M	618871	NO	YES	105	12.34	1308	ST ELEVATION IN V3- V6	0.65	PRESENT	NO	YES	NO	YES	NO	NO	DONE	ABSENT	ABSENT	ABSENT	PRESENT
Syed sab	70	M	619783	NO	NO	77	10.43	803	ST ELEVATION IN II, III, AVF, V1-V3	0.98	ABSENT	NO	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
Yashoda	60	M	629585	NO	NO	114	7.88	898	ST ELEVATION IN II,III,AVF	7.54	absent	NO	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
Farzeena	70	F	597535	NO	NO	128	9.77	1250	ST ELEVATION IN I, AVL	2.08	PRESENT	NO	YES	NO	NO	NO	NO	NO	ABSENT	ABSENT	PRESENT	ABSENT
Mallika begum	74	M	471286	NO	YES	120	13.56	1627	ST ELEVATION IN I, AVL	1.76	ABSENT	NO	YES	NO	YES	NO	NO	DONE	PRESENT	PRESENT	ABSENT	ABSENT
shahanaz	60	M	567654	yes	YES	205	16.76	3435	ST ELEVATION IN II,III, AVF	3.44	PRESENT	YES	YES	YES	YES	YES	USED	DONE	PRESENT	PRESENT	ABSENT	PRESENT
nazia	79	M	593249	yes	YES	197	14.32	2821	ST ELEVATION IN V1 TO V4	1.08	ABSENT	YES	YES	YES	NO	NO	USED	DONE	ABSENT	PRESENT	PRESENT	PRESENT
chinnarayappa	62	M	558921	NO	NO	102	13.9	1417	ST ELEVATION IN I, AVL, V4-V6	6.21	PRESENT	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
Abdul sab	72	M	589218	NO	NO	128	6.4	819	ST ELEVATION IN II,III, AVF	0.56	PRESENT	NO	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
Muunavar khan	64	M	595538	NO	NO	114	9.1	1037	ST ELEVATION IN II, III, AVF, V1-V3	0.49	PRESENT	NO	YES	NO	NO	NO	USED	DONE	ABSENT	ABSENT	PRESENT	ABSENT
konappa	75	M	594371	NO	NO	86	7.9	679	ST ELEVATION IN I, AVL	4.35	ABSENT	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
venkateshappa	55	M	585985	NO	NO	110	16.8	1848	ST ELEVATION IN V3- V6	3.78	absent	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	present	ABSENT
shankar	70	M	542440	NO	NO	118	7.3	861	ST ELEVATION IN II, III, AVF, V1-V3	2.98	ABSENT	NO	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
Mohamad jaffer	70	M	589214	yes	YES	122	19.6	2391	ST ELEVATION IN I, AVL	1.23	PRESENT	NO	YES	YES	NO	NO	USED	DONE	PRESENT	PRESENT	PRESENT	PRESENT
maimunnisa	71	M	589228	NO	YES	94	15.8	1485	ST ELEVATION IN V3- V6	1.65	PRESENT	NO	YES	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
rathnam	61	M	589226	NO	NO	85	5.9	501	ST ELEVATION IN II, III, AVF, V1-V3	7.54	ABSENT	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
manjunath	80	M	589547	NO	NO	143	5.3	757	ST ELEVATION IN II,III,AVF	5.56	ABSENT	YES	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
hanumappa	70	M	597434	NO	NO	107	18.5	1979	ST ELEVATION IN I, AVL	4.67	PRESENT	NO	YES	YES	NO	NO	USED	DONE	ABSENT	ABSENT	ABSENT	ABSENT
venkataswamy	65	M	486667	NO	NO	129	9,6	1238	ST ELEVATION IN I, AVL	8.56	PRESENT	NO	NO	NO	YES	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
ramappa	60	M	400976	NO	NO	91	7.8	709	ST ELEVATION IN II,III, AVF	3.69	absent	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
VENKATESHAPPA	60	M	545712	NO	NO	111	12.6	1398	ST ELEVATION IN V1 TO V4	2.45	PRESENT	NO	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
SONEPILLAPA	75	M	592493	NO	NO	112	5.76	645	ST ELEVATION IN I, AVL	1.23	ABSENT	NO	YES	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
SRI CHANNARAYAPPA	70	M	582671	yes	NO	126	7.4	932	ST ELEVATION IN II,III, AVF	1.98	present	YES	YES	YES	NO	NO	USED	DONE	ABSENT	PRESENT	ABSENT	ABSENT
SAMELLA KHAN	52	M	583136	NO	NO	167	4.3	718	ST ELEVATION IN V1 TO V4	0.55	absent	YES	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
RAHEM KHAN	80	M	582668	NO	NO	92	6.4	1062	ST ELEVATION IN I, AVL, V4-V6	0.67	present	NO	YES	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
SRI NARAYANAPPA	82	M	583251	NO	YES	104	7.2	748	ST ELEVATION IN II,III, AVF	0.98	absent	NO	NO	NO	NO	NO	NO	NO	PRESENT	ABSENT	ABSENT	ABSENT
LUKAPPA	65	M	584441	NO	NO	186	5.1	947	ST ELEVATION IN II, III, AVF, V1-V3	2.46	absent	YES	NO	NO	YES	NO	USED	DONE	ABSENT	ABSENT	ABSENT	ABSENT
NANJUDA GOWDA	75	M	384814	NO	NO	92	11.6	1065	ST ELEVATION IN I, AVL	3.74	absent	NO	YES	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
VENKATESHAPPA	85	M	583447	NO	NO	88	10.2	897	ST ELEVATION IN V3- V6	5.12	ABSENT	NO	YES	YES	NO	YES	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
NARAYANASWAMY	65	M	471211	NO	NO	102	11.9	1213	ST ELEVATION IN II, III, AVF, V1-V3	2.14	ABSENT	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	PRESENT
SANINEED KHAN	55	M	683136	NO	NO	108	4.8	518	ST ELEVATION IN I, AVL	6.23	absent	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
NARAYANA REDDY	75	M	584277	NO	YES	122	5.3	646	ST ELEVATION IN V3- V6	4.76	PRESENT	YES	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
VENKATAPPA	70	M	583954	NO	NO	96	6.7	643	ST ELEVATION IN II, III, AVF, V1-V3	8.56	absent	NO	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
PAPANNA	70	M	586380	NO	NO	106	7.2	762	ST ELEVATION IN II,III,AVF	3.45	PRESENT	NO	YES	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
HANUMANTHAPPA	55	M	585434	NO	NO	108	6.6	712	ST ELEVATION IN I, AVL	0.67	ABSENT	NO	YES	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
VENKATA SWAMY	76	M	556790	NO	NO	84	7.8	655	ST ELEVATION IN II, III, AVF, V1-V3	0.45	ABSENT	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
SHISPALA	55	M	525523	NO	YES	130	9.2	1196	ST ELEVATION IN II,III,AVF	0.92	PRESENT	YES	NO	NO	NO	NO	NO	DONE	ABSENT	present	ABSENT	ABSENT
Rajusab	70	M	586501	NO	YES	121	12.3	1488	ST ELEVATION IN I, AVL	1.23	PRESENT	NO	YES	NO	NO	NO	NO	DONE	ABSENT	ABSENT	PRESENT	ABSENT
VENKATESHAPPA	65	M	532661	NO	YES	96	16.2	1555	ST ELEVATION IN I, AVL	4.56	ABSENT	NO	YES	YES	NO	NO	USED	DONE	ABSENT	PRESENT	ABSENT	ABSENT
SHAIK ANNWAR	64	M	589032	NO	NO	78	14.2	1107	ST ELEVATION IN II,III, AVF	2.63	PRESENT	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
RAMAPPA	66	M	588728	NO	NO	106	13.4	1420	ST ELEVATION IN V1 TO V4	3.56	absent	YES	YES	NO	NO	NO	NO	DONE	ABSENT	ABSENT	PRESENT	ABSENT
SRI DAMOSAR	60	M	596088	yes	YES	162	9.3	1506	ST ELEVATION IN I, AVL	0.45	ABSENT	YES	YES	NO	NO	NO	NO	DONE	ABSENT	ABSENT	PRESENT	ABSENT
GOPAL KRISHNA	48	M	598189	NO	NO	176	8.4	1478	ST ELEVATION IN II,III, AVF	0.96	absent	YES	YES	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
JAYACHANDRA SHETTY	65	M	598074	yes	YES	132	15.6	2059	ST ELEVATION IN V1 TO V4	0.56	PRESENT	NO	YES	YES	NO	YES	USED	DONE	ABSENT	PRESENT	PRESENT	PRESENT
SHANKARAPPA	55	M	593859	yes	YES	247	9.8	2420	ST ELEVATION IN I, AVL, V4-V6	0.12	ABSENT	YES	YES	YES	YES	NO	USED	DONE	ABSENT	PRESENT	PRESENT	PRESENT
NAGARAJ	66	M	593249	NO	NO	174	10.5	1827	ST ELEVATION IN II,III, AVF	0.74	PRESENT	YES	NO	NO	NO	NO	NO	DONE	PRESENT	ABSENT	ABSENT	ABSENT
SHEIK SAYED	75	M	583101	NO	NO	92	17.2	1582	ST ELEVATION IN II, III, AVF, V1-V3	1.98	ABSENT	NO	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
SAMELLUA KHAN	53	M	583136	NO	NO	85	16.9	1438	ST ELEVATION IN I, AVL	4.7	PRESENT	NO	YES	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
SUBRAMANI REDDY	65	M	575265	NO	YES	122	15.3	1866	ST ELEVATION IN V3- V6	3.9	PRESENT	NO	YES	YES	NO	NO	NO	DONE	PRESENT	PRESENT	PRESENT	PRESENT
CHOEDAMMA	70	M	568056	NO	NO	110	14.3	1573	ST ELEVATION IN II, III, AVF, V1-V3	4.4	ABSENT	NO	YES	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
KRISHNAPPA	55	M	575689	NO	NO	62	9.4	582	ST ELEVATION IN I, AVL	2.6	absent	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
VENKATARAMAPPA	58	M	157836	yes	NO	78	10.6	826	ST ELEVATION IN V3- V6	2.45	absent	NO	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
BASAWANTHAPPA	72	M	157832	NO	NO	82	16.8	1377	ST ELEVATION IN II, III, AVF, V1-V3	1.98	absent	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
MUNIVENKATAPPA	70	M	157358	NO	YES	104	9.4	977	ST ELEVATION IN II,III,AVF	1.34	PRESENT	YES	NO	NO	YES	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
MUNIGOEDA	80	M	574482	NO	NO	96	10.1	969	ST ELEVATION IN I, AVL	3.12	ABSENT	NO	NO	YES	NO	NO	USED	DONE	ABSENT	ABSENT	ABSENT	ABSENT
VENKATESHAPPA	64	M	575709	NO	NO	120	9.45	1134	ST ELEVATION IN II, III, AVF, V1-V3	5.43	PRESENT	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
RAMAPPA	92	M	575280	NO	NO	112	6.2	694	ST ELEVATION IN II,III,AVF	6.28	absent	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT

NARAYANASWAMY	65	M	471211	NO	NO	123	5.6	688	ST ELEVATION IN I, AVL	1.56	absent	YES	YES	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
HIMANNA	55	M	577673	NO	NO	132	6.2	818	ST ELEVATION IN I, AVL	0.23	PRESENT	YES	YES	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
CHIKKAMUNIYAPPA	70	M	553982	NO	YES	143	5.7	813	ST ELEVATION IN II,III, AVF	5.6	ABSENT	YES	NO	NO	NO	NO	USED	NO	ABSENT	ABSENT	ABSENT	ABSENT
SRINIVAS MURTHY	56	M	576647	NO	NO	170	8.5	1145	ST ELEVATION IN V1 TO V4	0.48	absent	YES	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
VENKATESHAPPA	65	M	579475	NO	NO	193	3.7	704	ST ELEVATION IN I, AVL	0.69	absent	YES	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
MANJUNATH	78	M	414999	NO	NO	186	4.8	892	ST ELEVATION IN II,III, AVF	4.87	absent	YES	YES	NO	NO	NO	USED	DONE	ABSENT	ABSENT	ABSENT	ABSENT
VENKATESHappa	72	M	580739	NO	NO	142	6.6	937	ST ELEVATION IN V1 TO V4	3.64	ABSENT	YES	YES	YES	NO	NO	NO	DONE	PRESENT	ABSENT	ABSENT	ABSENT
CHUKKAPPA	68	M	580729	NO	NO	113	11.8	1333	ST ELEVATION IN I, AVL	1.56	PRESENT	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
SRINIVAS MURTHY	65	M	578822	NO	NO	118	7.5	885	ST ELEVATION IN II,III, AVF	2.54	absent	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
VENKATESHAPPA	70	M	580139	NO	NO	98	6.2	607	ST ELEVATION IN V1 TO V4	3.23	absent	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
PARNDAMAYYA	60	M	558157	NO	NO	89	7.1	631	ST ELEVATION IN I, AVL, V4-V6	5.24	PRESENT	NO	YES	NO	NO	NO	NO	NO	ABSENT	ABSENT	PRESENT	ABSENT
RAHIM KHAN	48	M	580302	NO	NO	79	16.2	1279	ST ELEVATION IN II,III, AVF	4.89	PRESENT	NO	YES	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
RARHNAM	45	F	437093	NO	NO	83	15.3	1267	ST ELEVATION IN II, III, AVF, V1-V3	6.36	PRESENT	NO	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
MUNIYAPPA	72	M	496021	NO	NO	183	7.8	1425	ST ELEVATION IN I, AVL	5.34	ABSENT	YES	YES	NO	NO	NO	NO	DONE	ABSENT	PRESENT	ABSENT	ABSENT
RAMAKRISHNAPPA	70	M	615384	NO	NO	176	8.3	1460	ST ELEVATION IN V3- V6	0.34	PRESENT	YES	YES	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
GURUSWAMY	65	M	483319	NO	NO	148	6.3	932	ST ELEVATION IN II, III, AVF, V1-V3	1.89	PRESENT	YES	NO	YES	NO	NO	USED	NO	ABSENT	PRESENT	ABSENT	ABSENT
SOMANNA	65	M	604485	NO	YES	132	13.7	1808	ST ELEVATION IN I, AVL	0.67	absent	YES	NO	YES	YES	NO	USED	DONE	PRESENT	PRESENT	PRESENT	ABSENT
MUNISWAMY	70	M	604449	NO	NO	103	12.6	1297	ST ELEVATION IN V3- V6	2.77	absent	NO	YES	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
HANUMAPPA	55	M	399533	NO	NO	206	6.8	1401	ST ELEVATION IN II, III, AVF, V1-V3	0.09	PRESENT	YES	NO	NO	NO	YES	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
VENKATAPATHI	66	M	614180	NO	YES	104	9.2	956	ST ELEVATION IN II,III,AVF	0.56	PRESENT	NO	NO	NO	YES	NO	USED	DONE	ABSENT	ABSENT	ABSENT	ABSENT
SHANTHAMMA	55	F	544760	NO	NO	130	8.8	1144	ST ELEVATION IN I, AVL	1.89	present	YES	YES	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
MUNIVENKATAPPA	80	M	509745	NO	NO	96	13.6	1305	ST ELEVATION IN II, III, AVF, V1-V3	8.645	ABSENT	NO	YES	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
GURAPPA	70	M	513528	yes	NO	146	9.8	1430	ST ELEVATION IN II,III,AVF	4.34	PRESENT	YES	NO	YES	NO	NO	NO	DONE	ABSENT	PRESENT	ABSENT	ABSENT
MUNYAMMA	55	F	612439	NO	YES	121	12.7	1536	ST ELEVATION IN I, AVL	5.59	PRESENT	NO	YES	NO	NO	NO	NO	DONE	ABSENT	ABSENT	PRESENT	ABSENT
KRISHNAPPA	78	M	525946	NO	NO	91	15.6	1419	ST ELEVATION IN II,III, AVF	1.45	ABSENT	NO	YES	YES	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
MUNISWARAYYA	80	M	403294	NO	NO	66	9.8	646	ST ELEVATION IN V1 TO V4	9.34	absent	NO	NO	NO	NO	NO	USED	NO	ABSENT	ABSENT	ABSENT	ABSENT
PYARSAB	66	M	401748	NO	NO	72	6.9	496	ST ELEVATION IN I, AVL	2.76	ABSENT	NO	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
KRISHNAYYAI	62	M	553838	NO	YES	85	7.2	612	ST ELEVATION IN II,III, AVF	7.34	PRESENT	NO	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
RAMACHANDRAIAH	58	M	591460	NO	NO	106	10.6	1123	ST ELEVATION IN V1 TO V4	1.56	ABSENT	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
CHIKKA HANUMATHAIAH	74	M	590620	NO	NO	105	7.9	829	ST ELEVATION IN I, AVL	0.59	absent	NO	NO	NO	NO	NO	USED	DONE	ABSENT	ABSENT	ABSENT	ABSENT
NANJUNDAPPA	73	M	428847	NO	NO	134	5.6	750	ST ELEVATION IN II,III, AVF	0.34	PRESENT	YES	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
KAREM PASHA	70	M	590873	yes	NO	156	6.2	967	ST ELEVATION IN V1 TO V4	2.45	absent	YES	YES	YES	NO	NO	USED	DONE	ABSENT	ABSENT	ABSENT	ABSENT
JANARDHANA CHARY	59	M	599684	NO	NO	168	3.7	621	ST ELEVATION IN I, AVL, V4-V6	5.321	PRESENT	YES	YES	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
SURESH	76	M	544619	NO	NO	106	6.9	732	ST ELEVATION IN II,III, AVF	3.89	ABSENT	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
MUNIYAPPA	62	M	608425	NO	NO	134	9.6	1286	ST ELEVATION IN II, III, AVF, V1-V3	0.87	ABSENT	YES	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
CHANDRANNA	64	M	605005	NO	NO	97	5.7	552	ST ELEVATION IN I, AVL	0.45	PRESENT	NO	NO	NO	NO	NO	NO	DONE	ABSENT	ABSENT	ABSENT	ABSENT
SEENAPPA	70	M	448920	NO	NO	109	8.4	915	ST ELEVATION IN V3- V6	2.56	PRESENT	NO	YES	NO	NO	NO	USED	DONE	ABSENT	PRESENT	ABSENT	ABSENT
GOPAL KRISHNA	62	M	605193	NO	NO	90	6.9	623	ST ELEVATION IN II, III, AVF, V1-V3	7.43	ABSENT	NO	NO	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	ABSENT
RAVINDRA	77	M	65586	NO	NO	230	7.8	1794	ST ELEVATION IN I, AVL	6.67	PRESENT	YES	YES	NO	YES	NO	NO	DONE	ABSENT	PRESENT	ABSENT	PRESENT
CHINNALLO	70	M	532734	NO	NO	136	5.6	765	ST ELEVATION IN V3- V6	2.678	ABSENT	YES	YES	NO	NO	NO	NO	NO	ABSENT	ABSENT	ABSENT	PRESENT