"CORRELATION BETWEEN SERUM MAGNESIUM AND ARRHYTHMIAS IN ACUTE MYOCARDIAL INFARCTION – ONE YEAR CROSS SECTIONAL STUDY IN A TERTIARY CARE HOSPITAL"

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ENDORSEMENT

This is to certify that the dissertation entitled **"CORRELATION BETWEEN SERUM MAGNESIUM AND ARRHYTHMIAS IN ACUTE MYOCARDIAL INFARCTION – ONE YEAR CROSS SECTIONAL STUDY IN A TERTIARY CARE HOSPITAL"** is a bonafide research work done by **CANDIDATE REG NO. BG0113002**.

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LIST OF ABBREVIATIONS USED

/Cumm	-	Per cubic millimeter
ACC	-	American College of Cardiology
ACE	-	Angiotensin-converting enzyme
AHA	-	American Heart Association
ALMI	-	Anteriolateral wall myocardial infarction
AMI	-	Acute myocardial infarction
ARB	-	Angiotensin receptor blocker
ASMI	-	Anterioseptal wall myocardial infarction
ATP	-	Intracellular Adenosine Triphosphate
AV	-	Atrioventricular
AWMI	-	Anterior wall myocardial infarction
BMD	-	Bone mineral density
BMI	-	Body mass index
bpm	-	Beats per minute
Ca++	-	Calcium
CABG	-	Coronary artery bypass grafting
CAD	-	Coronary artery disease
CHD	-	Coronary heart disease
CI	-	Confidence interval
CK-MB	-	Creatinine kinase MB
CPK-MB	-	Creatinine phosphokinase MB
DM	-	Diabetes mellitus
e.g.,	-	For example
ECG	-	Electrocardiogram

gm	-	Grams
h	-	Hours
HDL-C	-	High density lipoprotein cholesterol
i.e.	-	That is,
I.V	-	Intravenous
ICCU	-	Intensive cardiac care unit
ICDs	-	Implantable cardioverter-defibrillators
ICU	-	Intensive care unit
IWMI	-	Inferior wall myocardial infarction
K+	-	Potassium
LAD	-	Left anterior descending
LAFB	-	Left anterior fascicular block
LBBB	-	Left bundle branch block
LDL	-	Low-density lipoprotein
LMWH	-	Low-molecular-weight heparin
LV	-	Left ventricular
LWMI	-	Lateral wall myocardial infarction
MAGIC	-	Magnesium in Coronaries
Mg	-	Magnesium
mg	-	Milligrams
mg/dL	-	Milligrams per deciliter
Mg++	-	Magnesium
MgSO4	-	Magnesium sulphate
MI	-	Myocardial infarction
mm Hg	-	Millimeters of mercury

mmol	-	Millimole
mmol/l	-	Millimole per liter
n	-	Total number
Na-K-ATP	-	Sodium potassium adenosine triphosphate
NSTEMI	-	Non-ST elevation myocardial infarction
NYHA	-	New York Heart Association
p	-	Probability
PCI	-	Percutaneous coronary intervention
PVCs	-	Premature ventricular contractions
RBBB	-	Right bundle branch block
RV	-	Right ventricular
SD	-	Standard deviation
STEMI	-	ST elevation myocardial infarction
TH1	-	T-helper cell type 1
UK	-	United Kingdom
USA	-	United States of America
vs	-	Versus
VT	-	Ventricular tachycardia
WHO	-	World Health Organisation

ABSTRACT

Background and objectives

Life threatening arrhythmias are more frequent in patients with AMI with low serum magnesium levels. The present study was planned to evaluate the relationship between arrhythmias and serum magnesium levels in patients with acute myocardial infarction.

Materials and methods

This one year cross-sectional study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. A total of 100 patients who were admitted with acute myocardial infarction from January 2014 to December 2014 were studied. All the patients underwent estimation of serum magnesium levels and monitored for arrhythmias.

Results

More than three fourth of the study population was comprised of males (76%) and male to female ratio was noted as 3.16:1. Most of the patients were aged between 46 to 60 years (47%) and mean age was 57.11 ± 12.16 years. History of hypertension and diabetes mellitus present in 36% and 33% of the patients respectively. Majority of the patients (90%) presented with chest pain and 17% of the patients had crepitations. The commonest type of MI was AWMI (38%) followed by IWMI (28%). Serum magnesium levels were low (<1.80) in 30% of the patients. Arrhythmias were noted in 14 (14%) patients and 3 patients each (21.43% each) had first degree AV block, supraventricular tachycardia and ventricular tachycardia. Significantly higher number of patients with low serum

magnesium levels had arrhythmias (40% vs 2.86%; p<0.001) and also mean serum magnesium levels were significantly low in patients who had arrhythmias (1.52 ± 0.34 vs 2.10 ± 0.43; p<0.001). No association was found between types of arrhythmia with serum magnesium levels.

Conclusion

Patients presenting with AMI are likely to have hypomagnesemia and these patients have high risk of cardiac arrhythmias.

Key words

Acute myocardial infarction; Cardiac arrhythmias; Serum magnesium levels;

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INTRODUCTION

Myocardial infarction (MI) continue to be a significant cause of mortality and morbidity.[1] Myocardial infarction occurs when myocardial ischemia, a diminished blood supply to the heart, exceeds a critical threshold and overwhelms myocardial cellular repair mechanisms designed to maintain normal operating function and homeostasis. Ischemia at this critical threshold level for an extended period results in irreversible myocardial cell damage or death.[2]

Acute myocardial infarction (AMI) is myocardial necrosis in a clinical setting consistent with acute myocardial ischemia and detection of elevated values of cardiac biomarkers (CK-MB/troponin-I) above the 99th centile of the upper reference limit four hours after starting of symptom.[3]

Acute myocardial infarction remains a leading cause of death worldwide.[4] In the United States alone, approximately one million people suffer MI each year. In the UK, the annual incidence of MI (using 2006 CHD mortality data) was estimated to be about 146,000 of all aged individuals (men: ~87,000 and women: ~59,000), and the estimated prevalence in those aged >35 years is more than 1.4 million (men: ~970,000 and women: ~439,000).[5] The incidence of MI in India is 64.37/1000 people[6] in men aged 29-69 years.[7]

Myocardial infarction by pathology is myocardial cell death due to prolonged ischemia.[8] The most common cause of MI is coronary atherosclerotic plaque rupture or erosion, resulting in the exposure of thrombogenic contents to the blood which leads to thrombus formation and consequently MI.[5]

Several risk factors are associated with MI.[5] The risk factors include age, gender, low-birth weight, race/ethnicity, genetic factors (non-modifiable risk factors), hypertension, exposure to cigarette smoke, diabetes, atrial fibrillation and certain other cardiac conditions, dyslipidemia, carotid artery stenosis, sickle cell disease, postmenopausal hormone therapy, poor diet, less physical activity, obesity, and body fat distribution (well-documented and modifiable risk factors) and metabolic syndrome, alcohol abuse, drug abuse, oral contraceptive use, sleep-disordered breathing, migraine headache, hyperhomocysteinemia, elevated lipoprotein(a), elevated lipoprotein-associated phospholipase, hypercoagulability, inflammation, infection (less well-documented or potentially modifiable risk factors).[9]

Complication of AMI includes arrhythmic, mechanical, inflammatory sequel, about 90% develops some form of cardiac arrhythmia. In 25% patients, such rhythm abnormalities manifest within first 24 hours and risk of ventricular fibrillation is maximum in the 1st hour and declines thereafter.[10]

Major mechanisms of arrhythmias in the acute phase of coronary occlusion are reentry caused by inhomogeneity of the electrical characteristics of ischemic myocardium and cellular electrophysiological mechanism for reperfusion arrhythmia appears to include washout of various ions such as lactate, potassium, and toxic metabolic substances that have accumulated in the ischemic zone.[11]

It is found that in patients of MI, who became critical and who died suddenly, had low serum magnesium levels.[12,13] Similarly, life-threatening arrhythmias were found to be more frequent in patients with AMI with low serum magnesium levels.[14,15] It was also shown that the magnesium content of the infracted/ischemic

myocardium was much lower (about 40-50%) as compared to that of normal heart muscle. The magnesium depletion modifies coronary blood flow, blood clotting, and atherogenesis.[16] Magnesium lowers systemic vascular resistance, dilate coronary arteries, decrease platelet aggregation, improve myocardial metabolism, protect against catecholamine-induced myocardial necrosis, and stabilize cell membranes.[12]

Hypomagnesaemia is present in AMI as shift of magnesium from extracellular to intracellular compartments occur as it is taken up by adipocytes after catecholamine induced lipolysis and combined with soaps formed by free fatty acids. Extra cellular Mg++ declines markedly, especially over the first 24 to 48 hours after the onset of acute of myocardial infarction. Hypomagnesaemia in the initial phase of post AMI period is very critical, as ventricular tachyarrhythmia sudden cardiac death and re-infarction are the usual outcome.[17] Therefore patients with AMI and low magnesium levels are more prone to get arrhythmias which can be prevented by magnesium supplementation. It is stated that IV magnesium is a safe and effective method of reducing the frequency of arrhythmias and mortality following the acute myocardial infarction.[12]

Hence the present study was planned to evaluate the relationship between arrhythmias and serum magnesium levels in patients with AMI.

OBJECTIVES

The objective of this research was to study the arrhythmias in relation to serum magnesium in acute myocardial infarction.

REVIEW OF LITERATURE

Coronary artery disease (CAD) is the number one cause of death in the Western world and as such constitutes an immense public health problem. While CAD mortality declined in the last four decades in the USA as life expectancy increased, the use of age-adjusted rates to describe the CAD mortality obscures the fact that the decline largely represents the postponement of CAD deaths until older age. Thus, the burden of CAD is increasing in parallel with the increase in life expectancy.[18]

Coronary artery disease is a chronic disease with stable and unstable periods. During unstable periods patients may develop myocardial infarction (MI) with the involvement of activated inflammation in vascular wall. A myocardial infarction may be the first manifestation of CAD and in patients with established disease, it may occur repeatedly.[19]

Acute myocardial infarction

Acute myocardial infarction (MI) remains a leading cause of morbidity and mortality worldwide. Myocardial infarction occurs when myocardial ischemia, a diminished blood supply to the heart, exceeds a critical threshold and overwhelms myocardial cellular repair mechanisms designed to maintain normal operating function and homeostasis. Ischemia at this critical threshold level for an extended period results in irreversible myocardial cell damage or death.[2]

Critical myocardial ischemia can occur as a result of increased myocardial metabolic demand, decreased delivery of oxygen and nutrients to the myocardium

via the coronary circulation, or both. An interruption in the supply of myocardial oxygen and nutrients occurs when a thrombus is superimposed on an ulcerated or unstable atherosclerotic plaque and results in coronary occlusion.[20]

A high-grade (>75%) fixed coronary artery stenosis caused by atherosclerosis or a dynamic stenosis associated with coronary vasospasm can also limit the supply of oxygen and nutrients and precipitate an MI. Conditions associated with increased myocardial metabolic demand include extremes of physical exertion, severe hypertension (including forms of hypertrophic obstructive cardiomyopathy), and severe aortic valve stenosis. Other cardiac valvular pathologies and low cardiac output states associated with a decreased mean aortic pressure, which is the prime component of coronary perfusion pressure, can also precipitate MI.[2]

Myocardial infarction can be subcategorized on the basis of anatomic, morphologic, and diagnostic clinical information. From an anatomic or morphologic standpoint, the two types of MI are transmural and nontransmural.[2]

A transmural MI is characterized by ischemic necrosis of the full thickness of the affected muscle segment(s), extending from the endocardium through the myocardium to the epicardium.[2]

A nontransmural MI is defined as an area of ischemic necrosis that does not extend through the full thickness of myocardial wall segment(s). In a nontransmural MI, the area of ischemic necrosis is limited to the endocardium or to the endocardium and myocardium. It is the endocardial and subendocardial zones of the

myocardial wall segment that are the least perfused regions of the heart and the most vulnerable to conditions of ischemia.[2]

An older subclassification of MI, based on clinical diagnostic criteria, is determined by the presence or absence of Q waves on an electrocardiogram (ECG). However, the presence or absence of Q waves does not distinguish a transmural from a nontransmural MI as determined by pathology.[21]

A consensus statement was published to give a universal definition of the term myocardial infarction. The authors stated that MI should be used when there is evidence of myocardial necrosis in a clinical setting consistent with MI. Myocardial infarction was then classified by the clinical scenario into various subtypes.[3]

- Type 1 is a spontaneous MI related to ischemia from a primary coronary event (e.g., plaque rupture, thrombotic occlusion).
- Type 2 is secondary to ischemia from a supply-and-demand mismatch.
- Type 3 is an MI resulting in sudden cardiac death.
- Type 4a is an MI associated with percutaneous coronary intervention, and 4b is associated with in-stent thrombosis.
- Type 5 is an MI associated with coronary artery bypass surgery.[3]

A more common clinical diagnostic classification scheme is also based on electrocardiographic findings as a means of distinguishing between two types of MI, one that is marked by ST elevation (STEMI) and one that is not (NSTEMI).[2]

Management practice guidelines often distinguish between STEMI and non-STEMI, as do many of the studies on which recommendations are based. The

distinction between STEMI and NSTEMI also does not distinguish a transmural from a nontransmural MI. The presence of Q waves or ST-segment elevation is associated with higher early mortality and morbidity; however, the absence of these two findings does not confer better long-term mortality and morbidity.[22]

Incidence

The incidence of myocardial infarction (MI) in the world varies greatly. According to a Spanish study, the crude coronary heart disease (CHD) incidence rate was 300.6/100,000 person-years for men and 47.9/100,000 person-years for women.[7,24] The incidence of MI in India is 64.37/1000 people[6] in men aged 29-69 years.[7,24]

Risk factors

Predisposing risk factors for myocardial infarction are generally divided into two categories.[24]

- Nonmodifiable risk factors include age, sex, family history of premature coronary heart disease, male pattern baldness.[25]

- While modifiable risk factors include smoking or other tobacco use, diabetes mellitus (with or without insulin resistance), obesity, hypertension, Hypercholesterolemia, hypertriglyceridemia, including inherited lipoprotein disorders, dyslipidemia, obesity, sedentary lifestyle and/or lack of exercise, psychosocial stress, poor oral hygiene, type A personality.[26]

According to INTERHEART study,[26] risk factors for MI are divided into 2 categories i.e.;

- Emerging risk factors (homocysteine, glucose abnormalities, nutritional factors, abdominal obesity and psychosocial factors)

- Conventional risk factors (hypertension, diabetes, smoking and elevated cholesterol) between people of varying geographic and ethnic origin.

However, these known risk factors would explain only about 50% of cases of heart disease.[27]

Modifiable predisposing risk factors

Smoking

Smoking is considered as strong risk factor for myocardial infarction, premature atherosclerosis and sudden cardiac death. Smoking results in early STEMI especially in otherwise healthier patients. Smoking causes an average of 7 years earlier and more likely twice the chances of infarction than non smokers.[28]

Physical activity

Inactive people with multiple cardiac risk factors are more likely to develop MI. To get benefit, these individuals should start from modest exercise training.[29]

LDL and triglyceride levels

Elevated triglyceride levels and dense, small LDL particles act as predisposing risk factors for MI. Non fasting triglyceride levels appear to be a strong and independent predictor of future risk of MI, particularly when the total cholesterol level is also elevated. The reason behind it is that decreased HDL-C levels and increased triglyceride levels cause metabolic perturbations thus causing

adverse consequences. To identify high risk individuals, elevated triglyceride levels may become markers.[30]

Obesity/ Body mass index (BMI)

Increased BMI is directly related to incidence of MI. Infarction is greatly enhanced by extreme obesity because it is a recognized risk factor for MI.[24]

Diabetes mellitus (DM)

Significant differences in parameters measured were noted when all diabetic and non-diabetic patients were compared to the control group. It was found that in men with myocardial infarction there are significant differences between diabetic and nondiabetic patients with respect to certain risk factors such as age, hypertension and hypertriglyceridemia in diabetic patients while smoking and family history are predominant factors in non diabetic patients. However, newly diagnosed diabetic men have similar risk profiles to their known diabetic counterparts.[24]

Hypertension

Hypertension is strong and independent risk factor for MI. It is major risk factor of causing atherosclerosis in coronary blood vessels, result in heart attack or MI.[24]

Psychosocial Stress

Chronic life stress, social isolation and anxiety increase the risk of heart attack and stroke.[31]

Non -modifiable predisposing risk factors

Increasing Age

Older adults are more likely to die of heart disease. About 80% of heart disease deaths occur in people aged 65 or older.[24]

Gender

Men tend to have heart attacks earlier in life than women. Women's rate of heart attack increases after menopause but does not equal men's rate. Even so, heart disease is the leading cause of death for both men and women.[31]

Heredity/Family history

Increased risk if a first degree blood relative has had coronary heart disease or stroke before the age of 55 years for male relative and 65 years for female relatives.[31]

Less important factors

Fruits and vegetable consumption

According to epidemiological studies fruits and vegetables has negative effect on coronary heart disease mortality. But this is not always true. Studies show that this always occur due to antioxidant vitamins. However, it is also true that vegetables and fruits have high content of folate. Folate tends to lower plasma homocysteine levels. A slight elevation in plasma homocysteine level act as strong risk factor for arteriosclerosis of cerebral, peripheral or coronary arteries. Thus increases the incidence of MI.[32]

Sexual activity

Sexual activity predisposes the onset of myocardial infarction but chances to develop condition are relatively low. Moreover, this triggering can be reduced by regular exercise and proper physical activity. It is also found that relative risk is not increased in patients with prior history of cardiac disease. These findings are useful for decreasing the fear of sexual activity by counseling because this fear prevents complete rehabilitation from cardiovascular disease.[24]

Uncommon factors

Various other uncommon conditions can block a coronary artery and cause an MI. For example inflammation of the coronary arteries (rare); a stab wound to the heart; a blood clot forming elsewhere in the body (for example, in a heart chamber) and traveling to a coronary artery where it gets stuck; taking cocaine which can cause a coronary artery to go into spasm; complications from heart surgery and some other rare heart problems.[33]

Rosiglitazone vs. pioglitazone

Studies have suggested that the use of rosiglitazone may be associated with an increased risk of serious cardiovascular events compared with other treatments for type 2 diabetes. Compared with prescription of pioglitazone, prescription of rosiglitazone was associated with an increased risk of stroke, heart failure, and all-cause mortality and an increased risk of the composite of AMI, stroke, heart failure, or all-cause mortality in patients 65 years or older.[34]

Effect of calcium

Calcium supplements (without co administered vitamin D) are associated with an increased risk of myocardial infarction. As calcium supplements are widely used these modest increases in risk of cardiovascular disease might translate into a large burden of disease in the population. A reassessment of the role of calcium supplements in the management of osteoporosis is warranted.[35]

Psoriasis

Psoriasis may confer an independent risk of MI. Psoriasis is the most common T-helper cell type 1 (TH1) immunological disease. Evidence has linked TH1 diseases to myocardial infarction (MI). Psoriasis has been associated with cardiovascular diseases but risk is greatest in young patients with severe psoriasis. Low bone mineral density (BMD): Lower BMD was associated with an increase in MI risk for both men and women. Women had consistently lower hazard ratios compared to men in all models. Adjusting for smoking, hypertension, hypertriglyceridemia, and diabetes did not distinctively weaken these associations.[24]

Consumption of cocaine

Myocardial infarction is increasingly recognized as a complication of cocaine abuse. A significant number of persons suffering from myocardial infarction associated with cocaine abuse do not have significant coronary atherosclerosis, and the mechanism for infarction in these patients has remained obscure. Studies describe people with angiographically normal coronary arteries; cocaine abuse produces coronary artery spasm leading to coronary thrombosis and infarction.[24]

Clinical signs and symptoms

Acute MI can have unique manifestations in individual patients. The degree of symptoms ranges from none at all to sudden cardiac death. An asymptomatic MI is not necessarily less severe than a symptomatic event, but patients who experience asymptomatic MIs are more likely to be diabetic. Despite the diversity of manifesting symptoms of MI, there are some characteristic symptoms.[2]

- Chest pain described as a pressure sensation, fullness, or squeezing in the midportion of the thorax
- Radiation of chest pain into the jaw or teeth, shoulder, arm, and/or back
- Associated dyspnea or shortness of breath
- Associated epigastric discomfort with or without nausea and vomiting
- Associated diaphoresis or sweating
- Syncope or near syncope without other cause
- Impairment of cognitive function without other cause

An MI can occur at any time of the day, but most appear to be clustered around the early hours of the morning or are associated with demanding physical activity, or both. Approximately 50% of patients have some warning symptoms (angina pectoris or an anginal equivalent) before the infarct.[2]

Pathophysiology and natural history

Most myocardial infarctions are caused by a disruption in the vascular endothelium associated with an unstable atherosclerotic plaque that stimulates the formation of an intracoronary thrombus, which results in coronary artery blood flow

occlusion. If such an occlusion persists for more than 20 minutes, irreversible myocardial cell damage and cell death will occur.[2]

The development of atherosclerotic plaque occurs over a period of years to decades. The two primary characteristics of the clinically symptomatic atherosclerotic plaque are a fibromuscular cap and an underlying lipid-rich core. Plaque erosion can occur because of the actions of matrix metalloproteases and the release of other collagenases and proteases in the plaque, which result in thinning of the overlying fibromuscular cap. The action of proteases, in addition to hemodynamic forces applied to the arterial segment, can lead to a disruption of the endothelium and fissuring or rupture of the fibromuscular cap. The loss of structural stability of a plaque often occurs at the juncture of the fibromuscular cap and the vessel wall, a site otherwise known as the *shoulder region.* Disruption of the endothelial surface can cause the formation of thrombus via platelet-mediated activation of the coagulation cascade. If a thrombus is large enough to occlude coronary blood flow, an MI can result.[2]

The death of myocardial cells first occurs in the area of myocardium most distal to the arterial blood supply: the endocardium. As the duration of the occlusion increases, the area of myocardial cell death enlarges, extending from the endocardium to the myocardium and ultimately to the epicardium. The area of myocardial cell death then spreads laterally to areas of watershed or collateral perfusion. Generally, after a 6- to 8-hour period of coronary occlusion, most of the distal myocardium has died. The extent of myocardial cell death defines the magnitude of the MI. If blood flow can be restored to at-risk myocardium, more heart muscle can be saved from irreversible damage or death.[2]

The severity of an MI depends on three factors: the level of the occlusion in the coronary artery, the length of time of the occlusion, and the presence or absence of collateral circulation. Generally, the more proximal the coronary occlusion, the more extensive the amount of myocardium that will be at risk of necrosis. The larger the myocardial infarction, the greater the chance of death because of a mechanical complication or pump failure. The longer the period of vessel occlusion, the greater the chances of irreversible myocardial damage distal to the occlusion.[2]

STEMI is usually the result of complete coronary occlusion after plaque rupture. This arises most often from a plaque that previously caused less than 50% occlusion of the lumen. NSTEMI is usually associated with greater plaque burden without complete occlusion. This difference contributes to the increased early mortality seen in STEMI and the eventual equalization of mortality between STEMI and NSTEMI after 1 year.[2]

Diagnosis

Identifying a patient who is currently experiencing an MI can be straightforward, difficult, or somewhere in between. A straightforward diagnosis of MI can usually be made in patients who have a number of atherosclerotic risk factors along with the presence of symptoms consistent with a lack of blood flow to the heart. Patients who suspect that they are having an MI usually present to an emergency department. Once a patient's clinical picture raises a suspicion of MI, several confirmatory tests can be performed rapidly. These tests include electrocardiography, blood testing, and echocardiography.[2]

Diagnostic Procedures

The first diagnostic test is electrocardiography (ECG), which may demonstrate that a MI is in progress or has already occurred. Interpretation of an ECG is beyond the scope of this chapter; however, one feature of the ECG in a patient with an MI should be noted because it has a bearing on management. Practice guidelines on MI management consider patients whose ECG does or does not show ST-segment elevation separately. As noted earlier, the former is referred to as ST elevation MI and the latter as non-ST elevation MI. In addition to ST-segment elevation, 81% of electrocardiograms during STEMI demonstrate reciprocal ST-segment depression as well.[2]

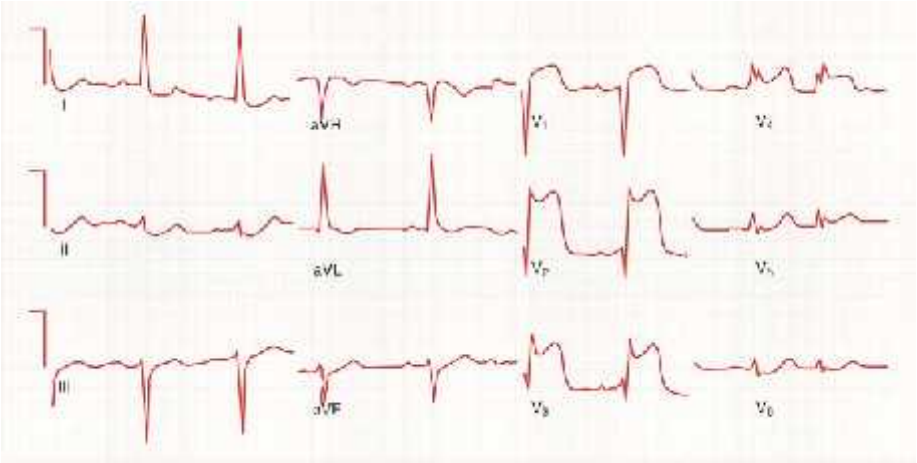


Figure 1. ECG showing ST elevation MI[2]

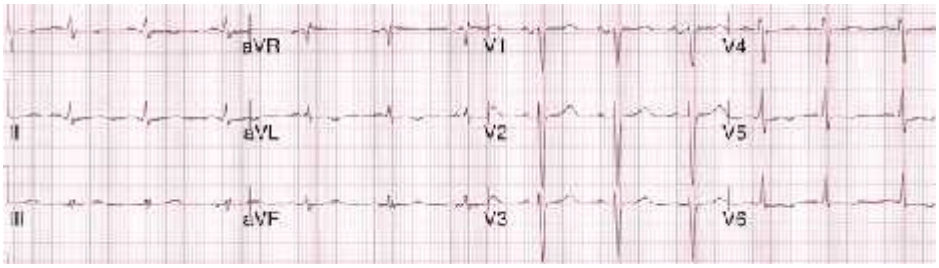


Figure 2. ECG showing non-ST elevation MI[2]

Laboratory Tests

Living myocardial cells contain enzymes and proteins (e.g., creatine kinase, troponin I and T, myoglobin) associated with specialized cellular functions. When a myocardial cell dies, cellular membranes lose integrity, and intracellular enzymes and proteins slowly leak into the blood stream. These enzymes and proteins can be detected by a blood sample analysis. These values vary depending on the assay used in each laboratory. Given the acuity of a STEMI and the need for urgent intervention, the laboratory tests are usually not available at the time of diagnosis. Thus, good history taking and an ECG are used to initiate therapy in the appropriate situations. The real value of biomarkers such as troponin lies in the diagnosis and prognosis of NSTEMI.[2]

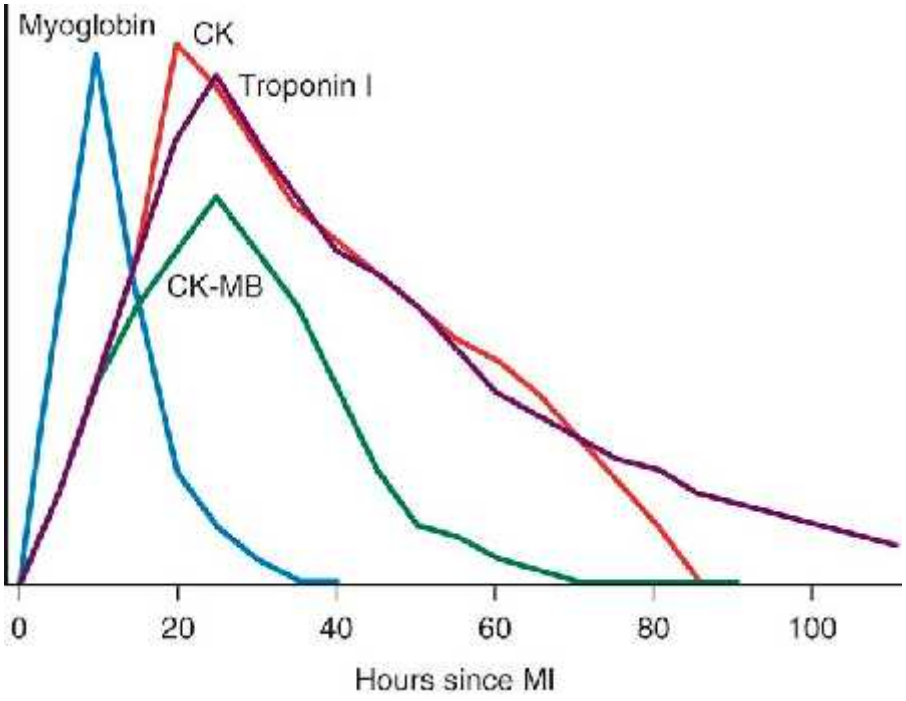


Figure 3. Biomarkers in the diagnosis of NSTEMI[2]

Imaging

An echocardiogram may be performed to compare areas of the left ventricle that are contracting normally with those that are not. One of the earliest protective actions of myocardial cells used during limited blood flow is to turn off the energy-requiring mechanism for contraction; this mechanism begins almost immediately after normal blood flow is interrupted. The echocardiogram may be helpful in identifying which portion of the heart is affected by an MI and which of the coronary arteries is most likely to be occluded. Unfortunately, the presence of wall motion abnormalities on the echocardiogram may be the result of an acute MI or previous (old) MI or other myopathic processes, limiting its overall diagnostic utility.[2]

Treatment

The goals of therapy in acute MI are the expedient restoration of normal coronary blood flow and the maximum salvage of functional myocardium. These goals can be met by a number of medical interventions and adjunctive therapies. The primary obstacles to achieving these goals are the patient's failure to recognize MI symptoms quickly and the delay in seeking medical attention. When patients present to a hospital, there are a variety of interventions to achieve treatment goals. “Time is muscle” guides the management decisions in acute STEMI, and an early invasive approach is the standard of care for acute NSTEMI.[22]

Medical Options

Antiplatelet Agents

The use of aspirin has been shown to reduce mortality from MI. Aspirin in a dose of 325 mg should be administered immediately on recognition of MI signs and symptoms.[22,36] The nidus of an occlusive coronary thrombus is the adhesion of a small collection of activated platelets at the site of intimal disruption in an unstable atherosclerotic plaque. Aspirin irreversibly interferes with function of cyclooxygenase and inhibits the formation of thromboxane A2. Within minutes, aspirin prevents additional platelet activation and interferes with platelet adhesion and cohesion. This effect benefits all patients with acute coronary syndromes, including those with amyocardial infarction. Aspirin alone has one of the greatest impacts on the reduction of MI mortality. Its beneficial effect is observed early in therapy and persists for years with continued use. The long-term benefit is sustained, even at doses as low as 75 mg/day.[2]

The Clopidogrel and Metoprolol in Myocardial Infarction Trial/Second Chinese Cardiac Study (COMMIT-CCS 2) trial evaluated the use of clopidogrel versus placebo in patients who were taking aspirin but not undergoing reperfusion therapy. It demonstrated a benefit in favor of clopidogrel when used with aspirin.[2]

The Clopidogrel as Adjunctive Reperfusion Therapy Thrombolysis in Myocardial Infarction 28 (CLARITY-TIMI 28) study compared clopidogrel versus placebo in patients receiving fibrinolytics within 12 hours of STEMI and showed a benefit in favor of clopidogrel as well.[37]

Supplemental Oxygen

Oxygen should be administered to patients with symptoms or signs of pulmonary edema or with pulse oximetry less than 90% saturation.[22] The rationale for using oxygen is the assurance that erythrocytes will be saturated to maximum carrying capacity. Because MI impairs the circulatory function of the heart, oxygen extraction by the heart and by other tissues may be diminished. In some cases, elevated pulmonary capillary pressure and pulmonary edema can decrease oxygen uptake as a result of impaired pulmonary alveolar-capillary diffusion. Supplemental oxygen increases the driving gradient for oxygen uptake.[20]

Arterial blood that is at its maximum oxygen-carrying capacity can potentially deliver oxygen to myocardium in jeopardy during an MI via collateral coronary circulation. The recommended duration of supplemental oxygen administration in a MI is 2 to 6 hours, longer if congestive heart failure occurs or arterial oxygen saturation is less than 90%. However, there are no published studies demonstrating that oxygen therapy reduces the mortality or morbidity of an MI.[2]

Nitrates

Intravenous nitrates should be administered to patients with MI and congestive heart failure, persistent ischemia, hypertension, or large anterior wall MI.[22,36] The primary benefit of nitrates is derived from its vasodilator effect. Nitrates are metabolized to nitric oxide in the vascular endothelium. Nitric oxide relaxes vascular smooth muscle and dilates the blood vessel lumen. Vasodilatation reduces cardiac preload and afterload and decreases the myocardial oxygen requirements needed for circulation at a fixed flow rate. Vasodilatation of the coronary arteries

improves blood flow through the partially obstructed vessels as well as through collateral vessels. Nitrates can reverse the vasoconstriction associated with thrombosis and coronary occlusion.[2]

When administered sublingually or intravenously, nitroglycerin has a rapid onset of action. Clinical trial data have supported the initial use of nitroglycerin for up to 48 hours in MI. There is little evidence that nitroglycerin provides substantive benefit as long-term post-MI therapy, except when severe pump dysfunction or residual ischemia is present.[22] Low BP, headache, and tachyphylaxis limit the use of nitroglycerin. Nitrate tolerance can be overcome by increasing the dose or by providing a daily nitrate-free interval of 8 to 12 hours. Nitrates must be avoided in patients who have taken a phosphodiesterase inhibitor within the previous 24 hours.[22]

Pain Control

Pain from MI is often intense and requires prompt and adequate analgesia. The agent of choice is morphine sulfate, given initially IV at 5 to 15 minute intervals at typical doses of 2 to 4 mg.[22] Reduction in myocardial ischemia also serves to reduce pain, so oxygen therapy, nitrates, and beta blockers remain the mainstay of therapy. Because morphine can mask ongoing ischemic symptoms, it should be reserved for patients being sent for coronary angiography. This was downgraded to a IIa recommendation in the latest STEMI guidelines.[2]

Beta Blockers

Beta blocker therapy is recommended within 12 hours of MI symptoms and is continued indefinitely.[22,36] Treatment with a beta blocker decreases the incidence

of ventricular arrhythmias, recurrent ischemia, reinfarction, and, if given early enough, infarct size and short-term mortality. Beta blockade decreases the rate and force of myocardial contraction and decreases overall myocardial oxygen demand. In the setting of reduced oxygen supply in MI, the reduction in oxygen demand provided by beta blockade can minimize myocardial injury and death.[2]

The use of a beta blocker has a number of recognized adverse effects. The most serious are heart failure, bradycardia, and bronchospasm. During the acute phase of an MI, beta blocker therapy may be initiated intravenously; later, patients can switch to oral therapy for long-term treatment. The COMMIT-CCS 2 trial raised safety concerns about the use of early intravenous beta blockers in high-risk patients.[10] In some patients who are considered high risk due to age or hemodynamic instability, it may be reasonable to hold off on early intravenous therapy.[2]

According to the 2007 guideline updates, anticoagulation should be added to standard medical therapy for most patients after myocardial infarction.[22]

Unfractionated Heparin

Unfractionated heparin is beneficial until the inciting thrombotic cause (ruptured plaque) has completely resolved or healed. Unfractionated heparin has been shown to be effective when administered intravenously or subcutaneously according to specific guidelines. The minimum duration of heparin therapy after MI is generally 48 hours, but it may be longer, depending on the individual clinical scenario. Heparin has the added benefit of preventing thrombus through a different mechanism than aspirin.[2]

Low-Molecular-Weight Heparin

Low-molecular-weight heparin (LMWH) can be administered to MI patients who are not treated with fibrinolytic therapy and who have no contraindications to heparin. The LMWH class of drugs includes several agents that have distinctly different anticoagulant effects. LMWHs are proved to be effective for treating acute coronary syndromes characterized by unstable angina and NSTEMI.[22] Their fixed doses are easy to administer, and laboratory testing to measure their therapeutic effect is usually not necessary.[2]

Warfarin

Warfarin is not routinely used after MI, but it does have a role in selected clinical settings. The latest guidelines recommend the use of warfarin for at least 3 months in patients with left ventricular aneurysm or thrombus, a left ventricular ejection fraction less than 30%, or chronic atrial fibrillation.[2]

Fibrinolytics

Restoration of coronary blood flow in MI patients can be accomplished pharmacologically with the use of a fibrinolytic agent. Fibrinolytic therapy is indicated for patients who present with a STEMI within 12 hours of symptom onset without a contraindication. Absolute contraindications to fibrinolytic therapy include history of intracranial hemorrhage, ischemic stroke or closed head injury within the past 3 months, presence of an intracranial malignancy, signs of an aortic dissection, or active bleeding. Fibrinolytic therapy is primarily used at facilities without access to an experienced interventionalist within 90 minutes of presentation.[36]

As a class, the plasminogen activators have been shown to restore normal coronary blood flow in 50% to 60% of STEMI patients. The successful use of fibrinolytic agents provides a definite survival benefit that is maintained for years. The most critical variable in achieving successful fibrinolysis is time from symptom onset to drug administration. A fibrinolytic is most effective within the first hour of symptom onset and when the door-to-needle time is 30 minutes or less.[36]

Angiotensin-Converting Enzyme Inhibitors and Angiotensin Receptor Blockers

Angiotensin-converting enzyme (ACE) inhibitors should be used in all patients with a STEMI without contraindications. ACE inhibitors are also recommended in patients with NSTEMI who have diabetes, heart failure, hypertension, or an ejection fraction less than 40%. In such patients, an ACE inhibitor should be administered within 24 hours of admission and continued indefinitely. Further evidence has shown that the benefit of ACE inhibitor therapy can likely be extended to all patients with an MI and should be started before discharge.[22,36] Contraindications to ACE inhibitor use include hypotension and declining renal function.[2]

ACE inhibitors decrease myocardial afterload through vasodilatation. One effective strategy for instituting an ACE inhibitor is to start with a low-dose, short-acting agent and titrate the dose upward toward a stable target maintenance dose at 24 to 48 hours after symptom onset. Once a stable maintenance dose has been achieved, the short-acting agent can be continued or converted to an equivalent-dose long-acting agent to simplify dosing and encourage patient compliance. For patients

intolerant of ACE inhibitors, angiotensin receptor blocker (ARB) therapy may be considered.[2]

Glycoprotein IIb/IIIa Antagonists

Glycoprotein IIb/IIIa receptors on platelets bind to fibrinogen in the final common pathway of platelet aggregation. Antagonists to glycoprotein IIb/IIIa receptors are potent inhibitors of platelet aggregation. The use of glycoprotein IIb/IIIa inhibitors during percutaneous coronary intervention (PCI) and in patients with MI and acute coronary syndromes has been shown to reduce the composite end point of death, reinfarction, and the need to revascularize the target lesion at follow-up. The current guidelines recommend the use of a IIb/IIIa inhibitor for patients in whom PCI is planned. For high-risk patients with NSTEMI who do not undergo PCI, a IIb/IIIa inhibitor may be used for 48 to 72 hours.[22]

Evidence is less well established for the direct thrombin inhibitor, bivalirudin. The 2007 American College of Cardiology (ACC) and the American Heart Association (AHA) guidelines recommend bivalirudin as an alternative to heparin therapy for patients who cannot receive heparin for a variety of reasons (e.g., heparin-induced thrombocytopenia).[22,36]

Statin Therapy

A statin should be started in all patients with a myocardial infarction without known intolerance or adverse reaction prior to hospital discharge. Preferably, a statin would be started as soon as a patient is stabilized after presentation. The Pravastatin or Atorvastatin Evaluation and Infection—Thrombolysis in Myocardial Infarction

22 (PROVE IT-TIMI 22) trial suggested a benefit of starting patients on high-dose therapy from the start (e.g., atorvastatin 80 mg/day).[38]

Aldosterone Antagonists

In the Epleronone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study (EPHESUS) trial, a mortality benefit was seen with eplerenone administration in all post-MI patients, provided multiple criteria were met. The criteria included concomitant use of an ACE inhibitor, ejection fraction less than 40%, symptomatic heart failure or diabetes, a creatinine clearance greater than 30 mL/min, and a potassium level less than 5 mEq/dL.[39] In patients that meet these criteria, the use of eplerenone has a Class I indication.

Other Treatment Options

Percutaneous Coronary Intervention

Patients with STEMI or MI with new left bundle branch block should have PCI within 90 minutes of arrival at the hospital if skilled cardiac catheterization services are available.[36] Patients with NSTEMI and high-risk features such as elevated cardiac enzymes, ST-segment depression, recurrent angina, hemodynamic instability, sustained ventricular tachycardia, diabetes, prior PCI, or bypass surgery are recommended to undergo early PCI (<48 hours). PCI consists of diagnostic angiography combined with angioplasty and, usually, stenting. It is well established that emergency PCI is more effective than fibrinolytic therapy in centers in which PCI can be performed by experienced personnel in a timely fashion.[40]

An operator is considered experienced with more than 75 interventional procedures per year. A well-equipped catheterization laboratory with experienced personnel performs more than 200 interventional procedures per year and has surgical backup available. Centers that are unable to provide such support should consider administering fibrinolytic therapy as their primary MI treatment.[2]

Restoration of coronary blood flow in a MI can be accomplished mechanically by PCI. PCI can successfully restore coronary blood flow in 90% to 95% of MI patients. Several studies have demonstrated that PCI has an advantage over fibrinolysis with respect to short-term mortality, bleeding rates, and reinfarction rates. However, the short-term mortality advantage is not durable, and PCI and fibrinolysis appear to yield similar survival rates over the long term. PCI provides a definite survival advantage over fibrinolysis for MI patients who are in cardiogenic shock. The use of stents with PCI for MI is superior to the use of PCI without stents, primarily because stenting reduces the need for subsequent target vessel revascularization.[41]

Surgical Revascularization

Emergent or urgent coronary artery bypass grafting (CABG) is warranted in the setting of failed PCI in patients with hemodynamic instability and coronary anatomy amenable to surgical grafting.[36]

Surgical revascularization is also indicated in the setting of mechanical complications of MI, such as ventricular septal defect, free wall rupture, or acute mitral regurgitation. Restoration of coronary blood flow with emergency CABG can limit myocardial injury and cell death if performed within 2 or 3 hours of symptom

onset. Emergency CABG carries a higher risk of perioperative morbidity (bleeding and MI extension) and mortality than elective CABG. Elective CABG improves survival in post-MI patients who have left main artery disease, three-vessel disease, or two-vessel disease not amenable to PCI.[2]

Implantable Cardiac Defibrillators

The results of a multicenter automatic defibrillator implantation trial have expanded the indications for automatic implantable cardioverter-defibrillators (ICDs) in post-MI patients. The trial demonstrated a 31% relative risk reduction in all-cause mortality with the prophylactic use of an ICD in post-MI patients with depressed ejection fractions.[42]

The current guidelines recommend waiting 40 days after an MI to evaluate the need for ICD implantation. ICD implantation is appropriate for patients in NYHA functional class II or III with an ejection fraction less than 35%. For patients in NYHA functional class I, the ejection fraction should be less than 30% before considering ICD placement. ICDs are not recommended while patients are in NYHA functional class IV.[43]

Treatment Outcomes

An individual patient's long-term outcome following an MI depends on numerous variables, some of which are not modifiable from a clinical standpoint. However, patients can modify other variables by complying with prescribed therapy and adopting lifestyle changes.[2]

Complications

Complications of MI are failure of reperfusion (ischemic), cardiac rupture, thrombosis and emboli, heart failure, psychological complications including depression and pericarditis. Severe left ventricular dysfunction or other mechanical causes most of the deaths following myocardial infarction.[24,44-46] The pumping ability of heart is reduced if a large area of heart muscle is damaged. This causes occurrence of other complications after myocardial infarction because less blood is pumped around body. These complications include heart failure, swollen ankles, tiredness and breathlessness. If electrical activity of heart is affected abnormal heart rhythms, sudden, fast or chaotic heart beats may occur. An immediate electrical shock treatment given by defibrillator is needed. If further buildup of atheroma continues or coronary arteries are badly affected, there is more likely to be the occurrence of myocardial infarction in future.[47]

Complications of acute myocardial infarction[48]

Complication Type	Manifestations
Ischemic	Angina, reinfarction, infarct extension
Mechanical	Heart failure, cardiogenic shock, mitral valve dysfunction, aneurysms, cardiac rupture
Arrhythmic	Atrial or ventricular arrhythmias, sinus or atrioventricular node dysfunction
Embolic	Central nervous system or peripheral embolization
Inflammatory	Pericarditis

Arrhythmic Complications of MI

About 90% of patients who have an acute myocardial infarction (AMI) develop some form of cardiac arrhythmia during or immediately after the event. In 25% of patients, such rhythm abnormalities manifest within the first 24 hours. In this group of patients, the risk of serious arrhythmias, such as ventricular fibrillation, is greatest in the first hour and declines thereafter. The incidence of arrhythmia is higher with an ST-elevation myocardial infarction (STEMI) and lower with a non–ST-elevation myocardial infarction (NSTEMI).[49]

The clinician must be aware of these arrhythmias, in addition to reperfusion strategies, and must treat those that require intervention to avoid exacerbation of ischemia and subsequent hemodynamic compromise. Most peri-infarct arrhythmias are benign and self-limited. However, those that result in hypotension, increase myocardial oxygen requirements, and/or predispose the patient to develop additional malignant ventricular arrhythmias should be aggressively monitored and treated.[50]

Pathophysiology of arrhythmic complications

AMI is characterized by generalized autonomic dysfunction that results in enhanced automaticity of the myocardium and conduction system. Electrolyte imbalances (eg, hypokalemia and hypomagnesemia) and hypoxia further contribute to the development of cardiac arrhythmia. The damaged myocardium acts as substrate for re-entrant circuits, due to changes in tissue refractoriness.[50]

Enhanced efferent sympathetic activity, increased concentrations of circulating catecholamines, and local release of catecholamines from nerve endings in the heart muscle itself have been proposed to play roles in the development of

peri-infarction arrhythmias. Furthermore, transmural infarction can interrupt afferent and efferent limbs of the sympathetic nervous system that innervates myocardium distal to the area of infarction. The net result of this autonomic imbalance is the promotion of arrhythmias.[50]

Classification of peri-infarction arrhythmias[50]

Peri-infarction arrhythmias can be broadly classified into the following categories:

- Supraventricular tachyarrhythmias, including sinus tachycardia, premature atrial contractions, paroxysmal supraventricular tachycardia, atrial flutter, and atrial fibrillation.
- Accelerated junctional rhythms
- Bradyarrhythmias, including sinus bradycardia and junctional bradycardia
- Atrioventricular (AV) blocks, including first-degree AV block, second-degree AV block, and third-degree AV block
- Intraventricular blocks, including left anterior fascicular block, right bundle branch block (RBBB), and left bundle branch block (LBBB)
- Ventricular arrhythmias, including premature ventricular contractions (PVCs), accelerated idioventricular rhythm, ventricular tachycardia, and ventricular fibrillation
- Reperfusion arrhythmias

Supraventricular Tachyarrhythmias

Sinus tachycardia is associated with enhanced sympathetic activity and can result in transient hypertension or hypotension. The elevated heart rate increases myocardial oxygen demand, and a decreased length of diastole compromises coronary flow, worsening myocardial ischemia.[50]

Premature atrial contractions

Premature atrial contractions often occur before the development of paroxysmal supraventricular tachycardia, atrial flutter, or atrial fibrillation. The usual cause of these extra impulses is atrial distention due to increased left ventricular (LV) diastolic pressure or inflammation associated with pericarditis.[50]

Paroxysmal supraventricular tachycardia

The incidence of a paroxysmal supraventricular tachycardia in the setting of an AMI is less than 10%. In the absence of definitive data in the patient with AMI, the consensus is that adenosine can be used when hypotension is not present.[50]

Atrial flutter

Atrial flutter occurs in less than 5% of patients with AMI. Atrial flutter is usually transient and results from sympathetic overstimulation of the atria.[50]

Atrial fibrillation

The rate of atrial fibrillation is 10-15% among patients who have AMIs. The onset of atrial fibrillation in the first hours of AMI is usually caused by LV failure, ischemic injury to the atria, or RV infarction. Pericarditis and all conditions leading

to elevated left atrial pressure can also lead to atrial fibrillation in association with an AMI. The presence of atrial fibrillation during an AMI is associated with an increased risk of mortality and stroke, particularly in patients who have anterior-wall MIs.[50]

Accelerated Junctional Rhythm

An accelerated junctional rhythm results from increased automaticity of the junctional tissue that leads to a heart rate of 70-130 bpm. This type of dysrhythmia is most common in patients who develop inferior myocardial infarctions. Treatment is directed at correcting the underlying ischemia.[50]

Bradyarrhythmias

Sinus bradycardia

Sinus bradycardia is a common arrhythmia in patients with inferior or posterior acute myocardial infarctions (AMIs). The highest incidence, 40%, is observed in the first 1-2 hours after AMI.[50]

The likely mechanism leading to bradycardia and hypotension is stimulation of cardiac vagal afferent receptors that result in efferent cholinergic stimulation of the heart. In the early phases of an AMI, resultant sinus bradycardia may actually be protective, reducing myocardial oxygen demand. Clinically significant bradycardia that decreases cardiac output and hypotension may result in ventricular arrhythmias and should, therefore, be treated aggressively. Isolated sinus bradycardia is not associated with an increase in the acute mortality risk, and therapy is typically unnecessary when the patient has no adverse signs or symptoms.[50]

Junctional bradycardia

Junctional bradycardia is a protective AV junctional escape rhythm at a rate of 35-60 bpm in patients who have an inferior MI. This arrhythmia is not usually associated with hemodynamic compromise, and treatment is typically not required.[50]

AV and Intraventricular Blocks

First-degree AV block

First-degree AV block is characterized by prolongation of the PR interval to longer than 0.20 seconds. This type of block occurs in approximately 15% of patients who have an acute myocardial infarction (AMI), most commonly an inferior infarction. Almost all patients who develop first-degree AV block have conduction disturbances above the His bundle. In these patients, the progression to complete heart block or ventricular asystole is rare.[50]

Second-degree AV block

Mobitz type I, or Wenckebach, AV block occurs in approximately 10% of patients who have an AMI and accounts for 90% of all patients who have an AMI and a second-degree AV block. A second-degree AV block is associated with a narrow QRS complex and is most commonly associated with an inferior MI. It does not affect the patient's overall prognosis.[50]

A Mobitz type II AV block accounts for 10% of all second-degree AV blocks (overall rate of < 1% in the setting of AMI). A Mobitz type II block is characterized by a wide QRS complex, and it is almost always associated with anterior infarction. This type of block often progresses suddenly to a complete heart

block. Mobitz type II AV blocks are associated with a poor prognosis, as the mortality rate associated with their progression to a complete heart block is approximately 80%. Therefore, this type of second-degree AV block should be immediately treated with transcutaneous pacing or atropine.[50]

Third-degree AV block

A third-degree AV block (ie, a complete heart block), occurs in 5-15% of patients who have an AMI and may occur with anterior or inferior infarctions. In patients with inferior infarctions, this type of block usually develops gradually, progressing from first-degree or a type I second-degree block. In most patients, the level of the block is supranodal or intranodal, and the escape rhythm is usually stable with a narrow QRS and rates exceeding 40 bpm. In 30% of patients, the block is below the His bundle, where it results in an escape rhythm with a rate slower than 40 bpm and a wide QRS complex.[50]

Intraventricular blocks

Conduction from the His bundle is transmitted through 3 fascicles: the anterior division of the left bundle, the posterior division of the left bundle, and the right bundle. An abnormality of electrical conduction in 1 or more of these fascicles is noted in about 15% of patients with AMI. Isolated left anterior fascicular block (LAFB) occurs in 3-5% of patients with AMI; progression to complete AV block is uncommon. Isolated left posterior fascicular block occurs in only 1-2% of patients who have an AMI. The blood supply of the posterior fascicle is larger than that of the anterior fascicle; therefore, a block here is associated with a relatively large infarct and high mortality rate.[50]

The right bundle branch receives its dominant blood supply from the left anterior descending (LAD) artery. Therefore, a new RBBB, which is seen in approximately 2% of patients with AMI, suggests a large infarct territory. However, progression to complete heart block is uncommon. In patients who develop an anterior MI and a new RBBB, the substantial risk for death is mostly from cardiogenic shock, which is presumably due to the large size of the myocardial infarct.[50]

The combination of RBBB with an LAFB is known as bifascicular block and commonly occurs with occlusion of the proximal LAD coronary artery. The risk of developing complete AV block is heightened, but complete block is still uncommon. Mortality is mostly related to the amount of muscle loss. Bifascicular block in the presence of first-degree AV block is called a trifascicular block. In 40% of patients, a trifascicular block progresses to a complete heart block.[50]

Ventricular Arrhythmias

Premature ventricular contractions

In the past, frequent premature ventricular contractions (PVCs) were considered to represent warning arrhythmias and indicators of impending malignant ventricular arrhythmias. However, presumed warning arrhythmias are frequently observed in patients who have an acute myocardial infarction (AMI) and who never develop ventricular fibrillation. On the converse, primary ventricular fibrillation often occurs without antecedent premature ventricular ectopy.[50]

Accelerated idioventricular rhythm

An accelerated idioventricular rhythm is seen in as many as 20% of patients who have an AMI. This pattern is defined as a ventricular rhythm characterized by a wide QRS complex with a regular escape rate faster than the atrial rate, but less than 100 bpm. AV dissociation is frequent. Slow, nonconducted P waves are seen; these are unrelated to the fast, wide QRS rhythm.[50]

Most episodes are short and terminate spontaneously. They occur with equal frequency in anterior and inferior infarctions. The mechanism might involve (1) the sinoatrial node or the AV node, which may sustain structural damage and depress nodal automaticity, and/or (2) an abnormal ectopic focus in the ventricle that takes over as the dominant pacemaker.[50]

The presence of accelerated idioventricular rhythm does not affect the patient's prognosis; no definitive evidence has shown that an untreated occurrence increases the incidence of ventricular fibrillation or death. This rhythm occurs somewhat more frequently in patients who develop early reperfusion than in others; however, it is neither sensitive nor specific as a marker of reperfusion.[50]

Temporary pacing is not indicated unless the rhythm is sustained and results in hypotension or ischemic symptoms. An accelerated idioventricular rhythm represents an appropriate escape rhythm. Suppression of this escape rhythm with an antiarrhythmic drug can result in clinically significant bradycardia or asystole. Therefore, an accelerated idioventricular rhythm should be left untreated.[50]

Nonsustained ventricular tachycardia

Nonsustained ventricular tachycardia is defined as 3 or more consecutive ventricular ectopic beats at a rate of greater than 100 bpm and lasting less than 30 seconds. In patients who experience multiple runs of nonsustained ventricular tachycardia, the risk for sudden hemodynamic collapse may be substantial.[50]

Nonetheless, nonsustained ventricular tachycardia in the immediate peri-infarction period does not appear to be associated with an increased mortality risk, and no evidence suggests that antiarrhythmic treatment offers a morbidity or mortality benefit. However, nonsustained ventricular tachycardia occurring more than 48 hours after infarction in patients with LV systolic dysfunction (LV ejection fraction < 0.40) poses an increased risk for sudden cardiac death; electrophysiologic testing and appropriate therapy are indicated in these patients.[50]

Sustained ventricular tachycardia

Sustained ventricular tachycardia is defined as 3 or more consecutive ventricular ectopic beats at a rate greater than 100 bpm and lasting longer than 30 seconds or causing hemodynamic compromise that requires intervention. Monomorphic ventricular tachycardia is most likely to be caused by a myocardial scar, whereas polymorphic ventricular tachycardia may be most responsive to measures directed against ischemia. Sustained polymorphic ventricular tachycardia after an AMI is associated with a hospital mortality rate of 20%.[50]

Ventricular fibrillation

The incidence of primary ventricular fibrillation is greatest in the first hour after the onset of infarct (4.5%) and declines rapidly thereafter. Approximately 60% of episodes occur within 4 hours, and 80% occur within 12 hours.[50]

Secondary or late ventricular fibrillation occurring more than 48 hours after an MI is usually associated with pump failure and cardiogenic shock. Factors associated with an increased risk of secondary ventricular fibrillation are a large infarct, an intraventricular conduction delay, and an anteroseptal AMI. Secondary ventricular fibrillation in conjunction with cardiogenic shock is associated with an in-hospital mortality rate of 40-60%.[50]

Reperfusion Arrhythmias

In the past, the sudden onset of rhythm disturbances after thrombolytic therapy in patients with AMI was believed to be a marker of successful coronary reperfusion. However, a high incidence of identical rhythm disturbances is observed in patients with AMI in whom coronary reperfusion is unsuccessful. Therefore, these so-called reperfusion arrhythmias are neither sensitive nor specific for reperfusion and should be treated as discussed under Accelerated Idioventricular Rhythm in the Arrhythmic Complications: Ventricular Arrhythmias section above.[50]

Role of serum magnesium

A fall in serum magnesium following acute myocardial infarction has been observed by many workers and magnesium deficiency is known to cause cardiac arrhythmias. It has been observed in many studies, that cases of myocardial

infarction who have lower serum magnesium level have more chances of developing tachyarrhythmias.[51]

Several epidemiologic studies[52,53,54] have identified magnesium deficiency as a risk factor for ischemic heart disease. Cardiovascular death rates in a given geographic region appear to correlate inversely with the water and soil magnesium content of the region. Moreover, three of the most potent in- dependent risk factors for coronary artery disease-namely, hypertension, hyperlipidemia, and diabetes mellitus-are all associated with magnesium deficiency.[54]

Catecholamine-induced myocardial necrosis during the first 48 h of AMI is augmented by the fall in serum magnesium levels that occurs in patients. AMI can produce a functional magnesium deficiency even in normomagnesmic patients. Myocardial infarction effects an increase in free fatty acids that combine with magnesium, trapping it in soaps and there- by decreasing available free extracellular magnesium levels.[55] It should be noted that patients with magnesium deficiency frequently have concomitant hypokalemia. Moreover, potassium deficiency cannot be fully repleted until the concomitant magnesium deficiency has been corrected with magnesium therapy.[56]

Therefore, catecholamine-induced hypokalemia during the initial 48 h of an AMI, which predisposes to early lethal ventricular arrhythmias, must be treated with both vigorous potassium and magnesium repletion. Indeed, magnesium has been demonstrated to reduce the risk of serious post- infarction ventricular and supraventricular arrhythmias and has also proven to be an efficacious antiarrhythmic

agent in torsade de pointes, digitalis toxicity, and ventricular tachycardia resulting from congestive heart failures.[57]

Magnesium, a divalent cation, is a physiologic calcium an- tagonist that inhibits calcium entry into vascular smooth muscle cells.8 Furthermore, magnesium promotes coronary artery vasodilation and peripheral systemic arterial vasodilation, thereby increasing coronary blood flow and reducing afterload. Magnesium may reduce ischemia and decrease sinus node and atrioventricular conduction. Because of its ability to inhibit myocardial cell sodium and calcium influx as well as potassium egress, magnesium may diminish infarct-related reperfusion injury and myocardial stunning, thereby limiting infarct size." However, it is essential to note that magnesium must be administered prior to thrombolytic or reperfusion therapy to exert its maximal effect. Indeed, magnesium and other calcium antagonists are ineffective when administered late after reperfusion.[52]

An older study has demonstrated that very early administration of magnesium in an animal infarct model can reduce infarct size if reperfusion of the artery occurs early.[58] Moreover, two additional animal studies[59,60] underscore the fact that magnesium sulfate decreases myocardial infarct size when administered before but not after coronary reperfusion.

It should be noted that the beneficial effects of magnesium in the latter two studies were most likely the result of a direct myocellular effect as evidenced by the absence of any difference in myocardial blood flow or hemodynamics between the magnesium-treated and control animals. Furthermore, by inhibiting catecholamine release, magnesium may prevent infarct extension. Magnesium possesses potent

antiplatelet properties and inhibits both in vitro and in vivo platelet aggregation. The formation of platelet thrombi in injured rabbit arteries in vivo is attenuated by magnesium administration.[52]

Moreover, magnesium sulfate significantly diminishes plate- let deposition and microthrombi formation at the site of endothelial damage in both injured canine coronary arteries and rat carotid arteries. Local application of magnesium salts decreases thrombosis in microvascular surgery. Human platelet aggregation, induced by a wide variety of agonists including collagen and thrombin, is inhibited by magnesium. The latter inhibitory effects of magnesium appear to be mediated by the inhibition of calcium influx as well as a reduction in the synthesis and release of proaggregatory eicosanoids involved in platelet aggregation, such as various cyclooxygenase (thromboxane A2) and lipoxygenase (1 2-hydroxyeico- satetraenoic acid, 12-HETE) products.[52]

Insulin potentiates the inhibitory effects of magnesium on platelet aggregation, a finding that may have ramifications for the treatment of diabetics with coronary artery disease. Magnesium stimulates the synthesis of endothelial cell prostacyclin, a very important arterial vasodilator and inhibitor of platelet aggregation in human. It is interesting to note that magnesium has been shown to augment the levels of high-density lipoprotein (HDL) cholesterol in patients with coronary artery disease. HDL, which is reduced in patients with AMI and unstable angina, stabilizes the half-life of prostacyclin and increases its synthesis.[52]

Antiplatelet properties of magnesium[52]

- Inhibits in vitro and in vivo platelet aggregation

- Decreases platelet deposition and rnicrothombi formation in animal
- Decreases thrombus formation in microvascular surgery
- Reduces synthesis and release of cyclooxygenase (thrornboxane A2) and lipoxygenase (12-HETE) products.
- Stimulates prostacyclin synthesis
- Increases high-density lipoprotein levels which stabilize prostacyclin models

Clinical trials of Magnesium Therapy for Acute Myocardial Infarction

Several clinical trials[61-69] of magnesium therapy for AMI have proffered conflicting results. Pooled data from seven randomized controlled trials of magnesium in 1,300 patients with AMI, analyzed by a meta-analysis, revealed a 55% reduction in the odds of death with magnesium compared with placebo. There was a reduced incidence of congestive heart failure and ventricular arrhythmias in the patients treated with magnesium compared with the patients treated with placebo.[61]

The Second Leicester Infarction Magnesium Intervention Trial (LIMIT-2) randomized 2,300 patients with AMI to receive either magnesium or placebo.[62,63] In LIMIT-2, magnesium therapy resulted in a 21% mortality reduction and a 25% reduction in early left ventricular failure at a mean follow-up of 2.7 years in the magnesium group compared with the placebo group. Patients in LIMIT-2 received an immediate loading dose of 8 mmol of magnesium sulfate followed by a 24 h infusion of 65 mmol magnesium sulfate or placebo and received thrombolytic therapy within 1 h of the immediate loading dose of magnesium sulfate or placebo.[62,63]

It should be noted that, except for LIMIT-2 in which approximately 33% of patients received thrombolytic therapy, the other early magnesium studies were undertaken when the treatment of AMI patients with thrombolytic therapy, aspirin, beta blockers, and angiotensin-converting enzyme inhibitors was not pervasive.[64]

The ISIS-4 trial[64] randomized 58,050 patients in a factorial design to one of three vasodilator therapies, captopril, isosorbide mononitrate, and magnesium. ISIS-4 reported that magnesium sulfate therapy consisting of an 8 mmol bolus with a subsequent 72 mmol infusion over 24 h did not affect 35-day mortality when compared with placebo.[64,65] However, the discrepant results between the early favorable magnesium trials such as LIMIT-2 and ISIS-4 may have several explanation.[63,67] The major difference is that, in LIMIT-2, magnesium was received by patients prior to thrombolytic therapy at a median of 3 h after the onset of chest pain. In contrast, in ISIS-4, magnesium was received by patients several hours after thrombolytic therapy at a median of 8 h after the onset of chest pain, thereby limiting the ability of magnesium to inhibit reperfusion injury and diminish myocardial stunning.[63,67] Moreover, in 30% of patients in ISIS-4 who did not receive thrombolytics, randomization to study drug did not take place until a median of 12 h; the exact time of administration of magnesium after randomization is unknown.[67]

Indeed, the ISIS-4 trial failed to test directly the hypothesis that magnesium therapy given prior to restoration of coronary blood flow would decrease reperfusion injury. In addition, the mortality of the control group in ISIS-4 was only about 7%.[67] A control group mortality this low makes it difficult to demonstrate a positive effect of an intervention such as magnesium therapy.[63]

When ISIS-4 is pooled with the earlier magnesium trials using the random effects model of meta-analysis, which incorporates terms accounting for heterogeneity (inter- and intratrial variability) among the various trials, magnesium is found to de- crease mortality in AMI.[68]

An Israeli clinical trial of magnesium therapy for AMI has provided further insightful results. Intra- venous magnesium sulfate was given to patients with AMI deemed ineligible to receive thrombolytic therapy. In this randomized, double-blind, placebo-controlled trial, 96 patients received intravenous magnesium over 48 h as opposed to the control group of 98 patients who received placebo. Of note, the average time to treatment in this latest trial was 7 h from the onset of chest pain, a full 5 h earlier than the median time to randomization in the nonthrombolytic subgroup in ISIS-4. Magnesium decreased the incidence of ventricular arrhythmias, congestive heart failure, and conduction distur- bances when compared with placebo. Left ventricular ejection fraction measured at 1 to 2 months after infarction was significantly greater in the magnesium group (52%) than in the placebo group (45%) ($p=0.01$). Moreover, magnesium sulfate significantly decreased in-hospital mortality (4 vs. 17%: $p<0.001$), particularly in the subgroup of elderly patients aged > 70 years.[69]

Thus, magnesium therapy for AMI may be efficacious in patients who are ineligible for thrombolytic therapy. Although magnesium therapy has the numerous afore- mentioned salutary properties in addition to its low cost and relative ease of administration, it is not devoid of adverse effects.[52]

Taken together, the conflicting results of prior trials with magnesium therapy for AMI demonstrate the need for further, better designed clinical trials to assess fully the potential impact of magnesium therapy. In particular, its early administration, either prior to or within less than 1 h of thrombolytic therapy or primary percutaneous transluminal coronary angioplasty, warrants further investigation. Moreover, magnesium therapy may prove to have a major role in the treatment of patients ineligible for thrombolytic therapy. One such trial that will further investigate magnesium therapy for AMI is the multicenter MAGIC (Magnesium in Coronaries) trial sponsored by the NIH. MAGIC will determine whether the early administration of magnesium to prevent reperfusion injury will decrease mortality in 10,000 high-risk patients with MI. A dosing regimen consisting of a bolus dose of magnesium followed by a continuous infusion for at least 24 h to maintain elevated serum levels of magnesium will be used.[52]

Magnesium therapy has numerous desirable properties for the treatment of myocardial infarction. It is a coronary and systemic vasodilator, calcium antagonist, antiarrhythmic agent, and antiplatelet drug that modulates autonomic function and limits reperfusion injury when given early in the setting of acute myocardial infarction. Clinical trials of magnesium therapy for acute myocardial infarction have yielded conflicting results. Despite the very large number of patients enrolled in ISIS-4, the late administration of magnesium therapy coupled with a low control group mortality rate may have biased the trial to a null effect of magnesium.[52] Also all these studies were old which were conducted from 1980 to 2000.[62-69]

Recently, serum magnesium was estimated in 35 patients admitted to the ICCU, of these 33 of patients (92.8%) had lowered serum magnesium levels, mean

serum magnesium levels being 1.63% (control mean 2.52%). Of these, 18 patients were admitted with in 6 hours of onset of chest pain, and all 100% had lowered of serum magnesium.[70]

Serum magnesium levels checked in 25 patients of acute myocardial infarction 25 patients control subjects. Mean serum magnesium levels was significantly low in cases of Acute myocardial infarction. Patients with very low magnesium levels, had more arrhythmias, and mortality.[71]

Magnesium deficiency leads to arrhythmias by reduced activity of the magnesium dependent Na-K-ATP ase with consecutive intracellular K-deficiency degreased resting membrane potential and increased digitalis toxicity. Magnesium has benefit in acute myocardial infarction and prevention of sudden cardiac death.[72]

Patients of myocardial infarction had significantly lower levels of serum magnesium when compared to control. The values noted were serum Magnesium (%) in myocardial infarction (22 case) – 1.27 ± 0.51 and in control (15 cases) – 2.41 ± 0.54. Findings suggested that magnesium deficiency can potentiate oxidative injury to post ischemic myocardium.[73]

Serum magnesium levels checked in 905 patients admitted in ICCU. Among 905, 342 were admitted with acute myocardial infarction and 563 with other diagnosis. The incidence of serious ventricular Ectopic Beats, atrial fibrillation and supraventricular tachycardia were Higher in the Hypomagnesemic patients.[74]

35 patients with acute myocardial infarction admitted in ICCU were studied. Serum magnesium was found to be significantly lower on the first day, especially in

those with arrhythmias and left ventricular failure. It gradually rose to normal value by the Twenty first day.[75]

El-Shafei MM et al.[76] study revealed prevalence rate of 30% in the acute myocardial infarction who developed arrhythmias. This study has highly significant difference ($p<0.001$) was found between serum magnesium level in the first 24 hours among the infarction group and controls and insignificant difference was found between groups after 24 hours. In same study a significant difference ($P<0.05$) was found between magnesium level in cases who developed arrhythmias and those who escaped this complications in infarction group.

Study of MBK Choudhury et al.[77] reveled incidence of 86.66% having hypomagnesmia in patients of acute myocardial infarction .This study shows magnesium deficiency may potentiate the tendency to early ventricular tachyarrhythmias in the setting of acute myocardial infarction.

In Fawcett WJ et al.[78] study it shows that frequency of cardiac arrhythmias occurring after myocardial infarction is higher in hypomagnesaemic patients and is reduced by magnesium administration.

In Mark J et al.[79] publication its quoted that cardiac arrhythmias and coronary artery vasospasm can be caused by Mg deficiency and Intravenous Mg reduces the risk of arrhythmia and death immediately after acute myocardial infarction.[7]

Indeed, the relative safety and low cost of magnesium, its numerous salutary properties, and its reduction of mortality in patients ineligible for thrombolytic

therapy buttress the contention that additional clinical trials that focus on early administration of magnesium are needed to define precisely the role of magnesium therapy for acute myocardial infarction.

METHODOLOGY

This one year cross-sectional study was undertaken at the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2014 to December 2014.

Study design and duration

The study design was a one year cross-sectional study.

Study period

This study was carried out from January 2014 to December 2014.

Source of Data

Patients admitted with acute myocardial infarction under the Department of Medicine and Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum were enrolled.

Sample size

A total of 100 patients admitted with acute myocardial infarction during the study were enrolled.

Sample size calculation

The sample size was determined by following formula.

$$n = Z_{\alpha}\, p\, q / d^2$$

Where,

Zα is the reliability coefficient at 95% confidence interval (1.96)

p is the sensitivity (76%) derived from previous study[77]

q is complement of p determined by 100 – p (100-76 = 24).

d is absolute error = 8.4%

Therefore,

$n = (1.96)^2 * (76) * (24) / (8.4)^2$

$n = 98.69 \approx 100$

Hence a total of 100 patients was considered for the study.

Selection criteria

Inclusion Criteria

- Patients with ECG findings and biochemical markers suggestive of acute myocardial infarction.
- Both ST segment elevation myocardial infarction (STEMI) and Non-ST segment elevation myocardial infarction (NSTEMI).
- Patients aged more than 18 years of age.

Exclusion Criteria

- Patients with previous history of documented arrhythmias.
- Patients having history of previous myocardial infarction.
- Patients with
    a. Chronic kidney diseases.
    b. Dyselectrolytemia.

c. Hyperthyroidism.

d. Chronic diaarhoea

e. Malabsorption syndrome

f. Hungry bone syndrome

g. Hyperaldosteronism

Ethical clearance

The study was approved by the Ethical and Research Committee of Jawaharlal Nehru Medical College, Belgaum.

Informed consent

The patients were screened for eligibility and those who fulfilled selected selection criteria were informed about the nature of study and included after obtaining a written informed consent (Annexure–I).

Data collection

Patients were interviewed and demographic data, history of other comorbid conditions and personal history was obtained. The patients underwent clinical examination and systemic examination. These findings were noted on a predesigned and pretested proforma (Annexure-II).

Investigations

The patients underwent following investigations

- 12-Lead electrocardiography
- Complete blood count

- Serum magnesium levels on the day 0
- Echocardiography
- Cardiac enzymes - CPK-MB and Troponin I
- Renal function tests (if required).
- Thyroid function tests (if required).
- Liver function tests (if required).

Procedure

Patients were evaluated for the presence of myocardial infarction based on history, clinical examination, ECG and elevated levels of serum CPK-MB isoenzymes and Trop I.

A 12-Lead electrocardiography was taken immediately after admission. Patients were connected to cardiac moniter for minimum of 24 hours and more if needed. Repeated ECGs were taken from time of admission up to 24 hours since onset of symptoms.

Sample for serum magnesium was collected immediately after the admission. Sample is also sent for complete blood count, liver function tests and renal function tests. Echocardiography was done if indicated.

Outcome variables

Estimation of serum magnesium levels

Blood samples were collected from the subjects with all aseptic precautions. 10 ml of venous blood was collected from median cubital vein by disposable plastic syringe. The needle was detached from the nozzle and blood was transferred

immediately in to a dry, clean, ionized, graduated, screw capped plastic test tube with a gentle push to avoid hemolysis. Serum was separated by centrifuging and serum samples are stored in the ultra-freeze at -20^0C. Routine investigations were restricted to patients who really needed them. Estimation of serum magnesium was done by absorption spectrophotometer and Serum CK-MB by kinectic immunoinhibition method using the available regent kit.[80]

Interpretation of serum magnesium levels

The serum magnesium levels were interpreted as below.[80]

- Normal – 1.80 to 2.50
- Low - < 1.80

Arrhythmias

The patients were monitored for arrhythmias.

Statistical methods

The data obtained was coded and entered into the Microsoft Excel Spreadsheet (Annexure III). The data was analysed using SPSS statistical software version 20.0. The categorical data was expressed in terms of ratios and percentages while continuous data was expressed as mean ± standard deviation (SD). The comparison of categorical data was done using chi-square test or Fisher's exact test and continuous data was compared using independent sample 't' test. At 95% CI, p value of ≤ 0.050 was considered as statistically significant.

RESULTS

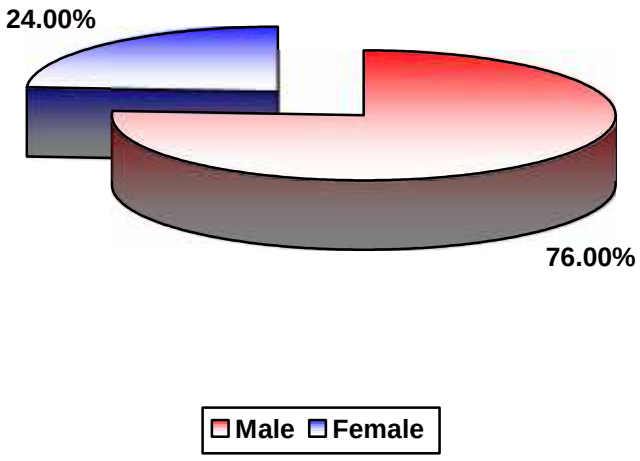
The present one year cross-sectional study was done in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum from January 2014 to December 2014. A total of 100 patients who were admitted with acute myocardial infarction during the study were studied.

The data was analysed and the final results were tabulated as below.

Table 1. Distribution of patients according to sex

Sex	Distribution (n=100)	
	Number	**Percentage**
Male	76	76.00
Female	24	24.00
Total	**100**	**100.00**

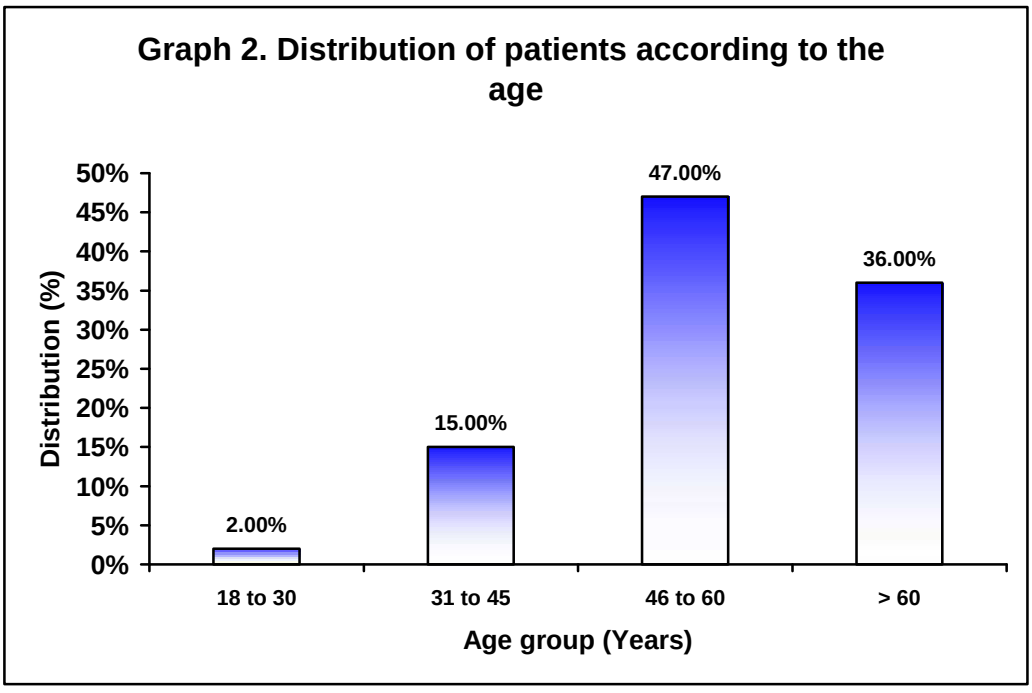
Graph 1. Distribution of patients according to sex



In the present study majority of the patients (76%) were males and male to female ratio was noted as 3.16:1.

Table 2. Distribution of patients according to the age

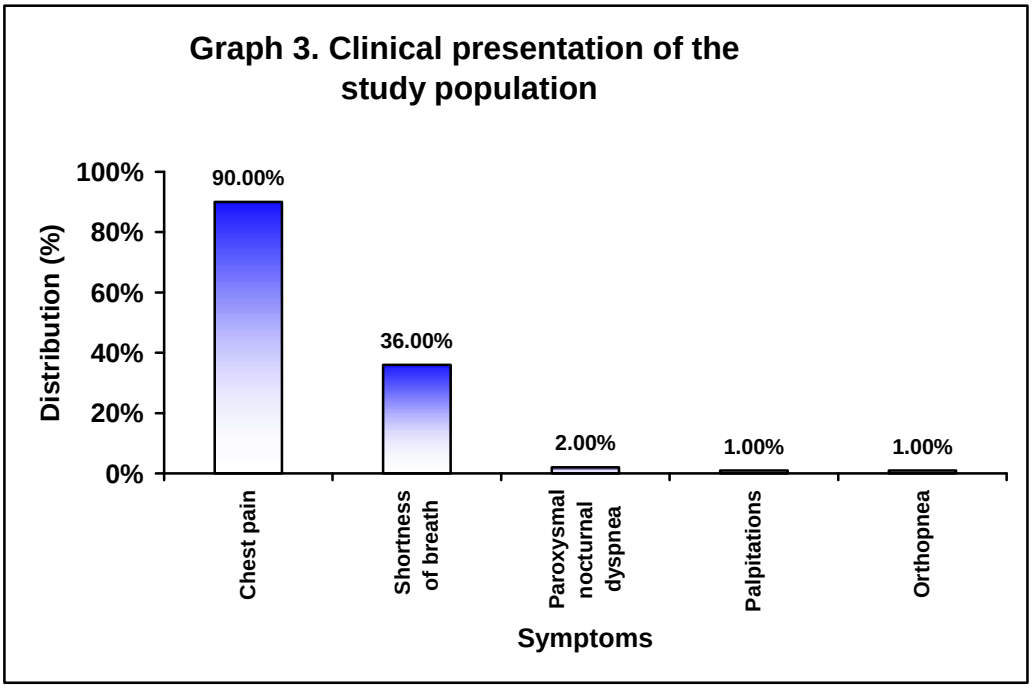
Age group (Years)	Distribution (n=100)	
	Number	**Percentage**
18 to 30	2	2.00
31 to 45	15	15.00
46 to 60	47	47.00
> 60	36	36.00
Total	**100**	**100.00**



In the present study most of the patients belonged to the age group between 46 to 60 years (47%) followed by > 60 years (36%).

Table 3. Clinical presentation of the study population

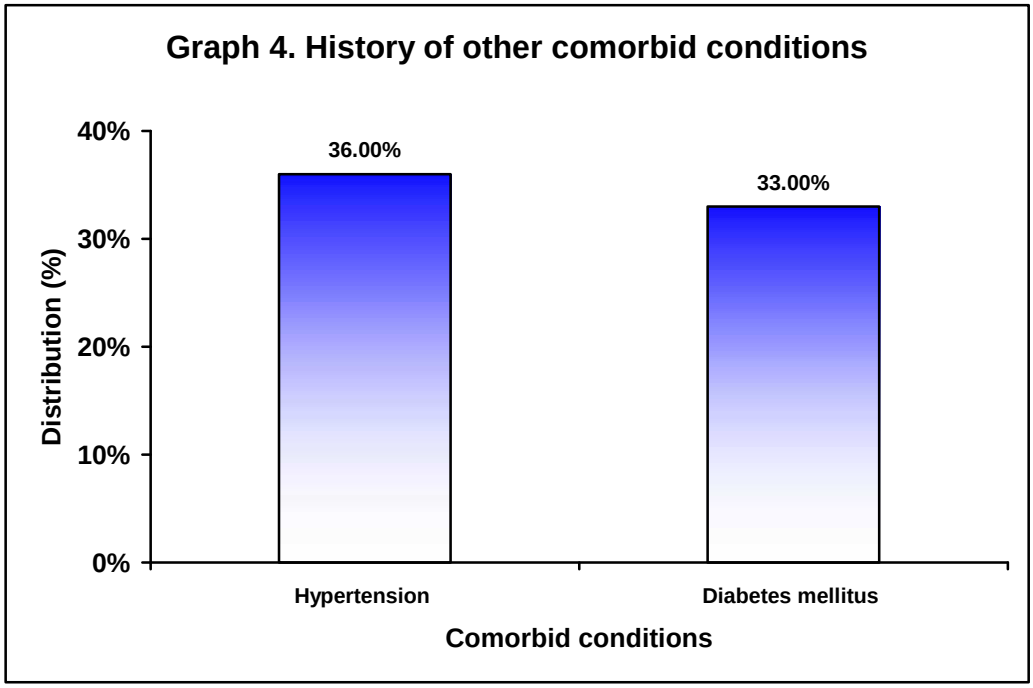
Symptoms	Distribution (n=100)	
	Number	**Percentage**
Chest pain	90	90.00
Shortness of breath	36	36.00
Paroxysmal nocturnal dyspnea	2	2.00
Palpitations	1	1.00
Orthopnea	1	1.00



In the present study majority of the patients (90%) presented with chest pain followed by shortness of breath (36%).

Table 4. History of other comorbid conditions

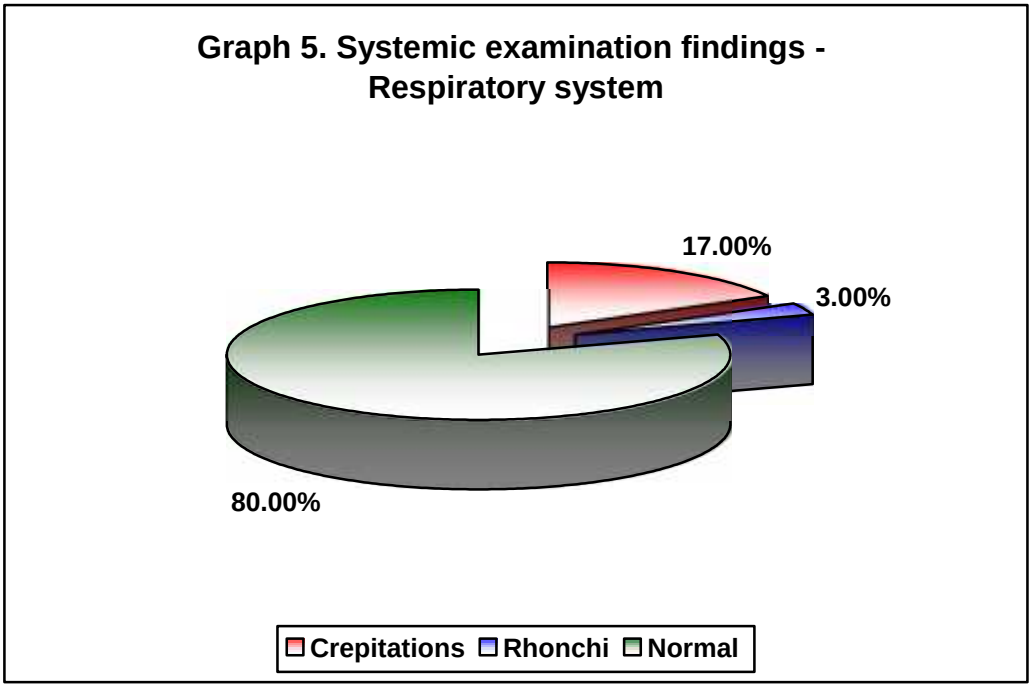
History	Distribution (n=100)	
	Number	Percentage
Hypertension	36	36.00
Diabetes mellitus	33	33.00



In this study history of hypertension and diabetes mellitus was noted in 36% and 33% of the patients.

Table 5. Systemic examination findings - Respiratory system

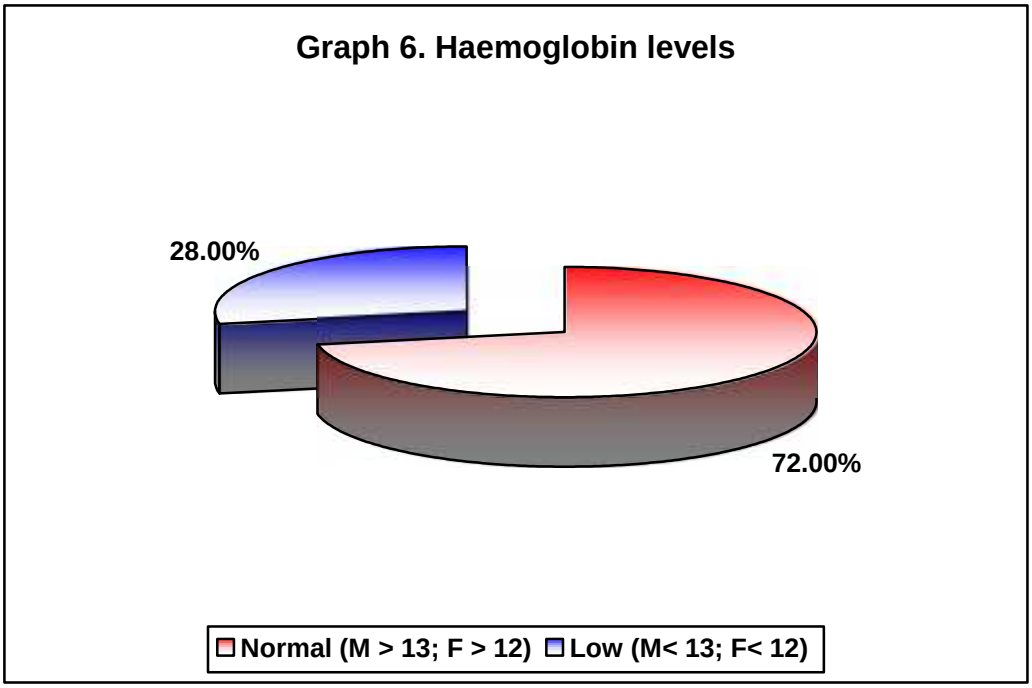
Findings	Distribution (n=100)	
	Number	**Percentage**
Crepitations	17	17.00
Rhonchi	3	3.00
Normal	80	80.00
Total	**100**	**100.00**



In the present study systemic examination findings revealed crepitations in 17% of the patients.

Table 6. Haemoglobin levels

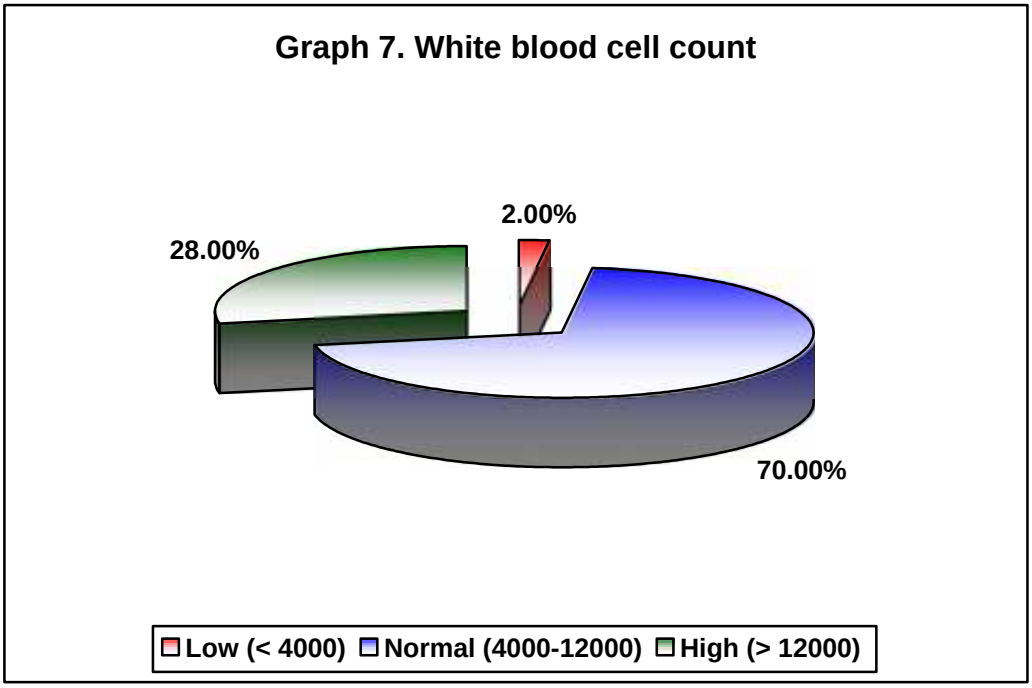
Haemoglobin (gm%)	Distribution (n=100)	
	Number	**Percentage**
Normal (M>13; F>12)	72	72.00
Low (M<13; F<12)	28	28.00
Total	**100**	**100.00**



In this study 28% of the patients presented with lower haemoglobin levels (M<13; F<12).

Table 7. White blood cell count

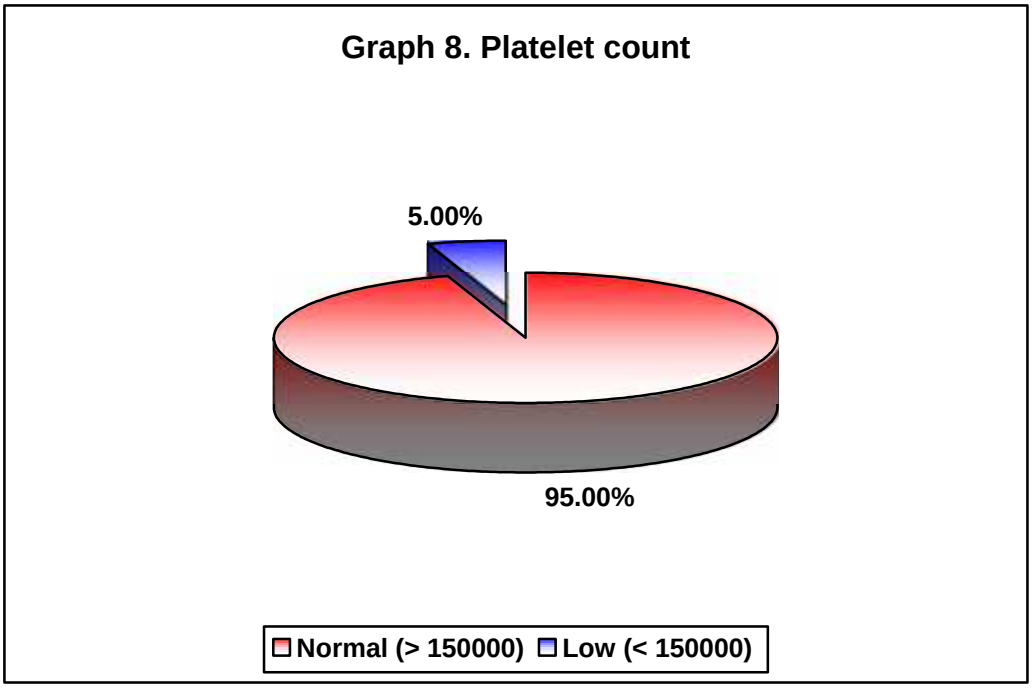
WBC (/Cumm)	Distribution (n=100)	
	Number	**Percentage**
Low (<4000)	2	2.00
Normal (4000-12000)	70	70.00
High (>12000)	28	28.00
Total	**100**	**100.00**



In the present study white blood cell count was high (>12000) in 28% of the patients.

Table 8. Platelet count

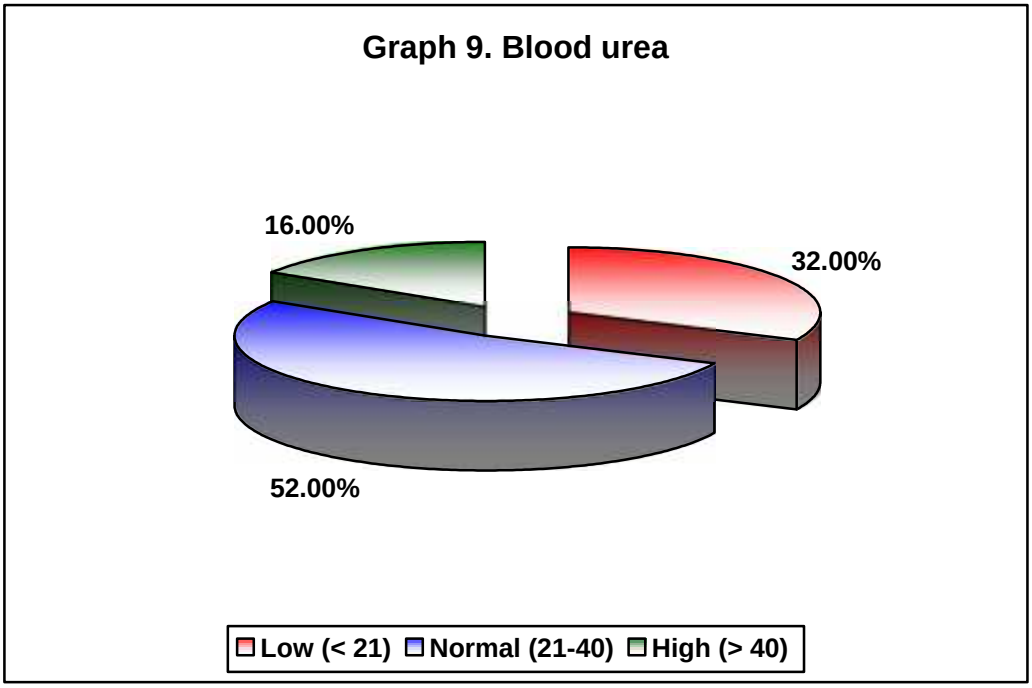
Platelet count (/Cumm)	Distribution (n=100)	
	Number	**Percentage**
Normal (>150000)	95	95.00
Low (<150000)	5	5.00
Total	**100**	**100.00**



In this study the platelet count was low (<150000 /Cumm) in 5% of the patients.

Table 9. Blood urea

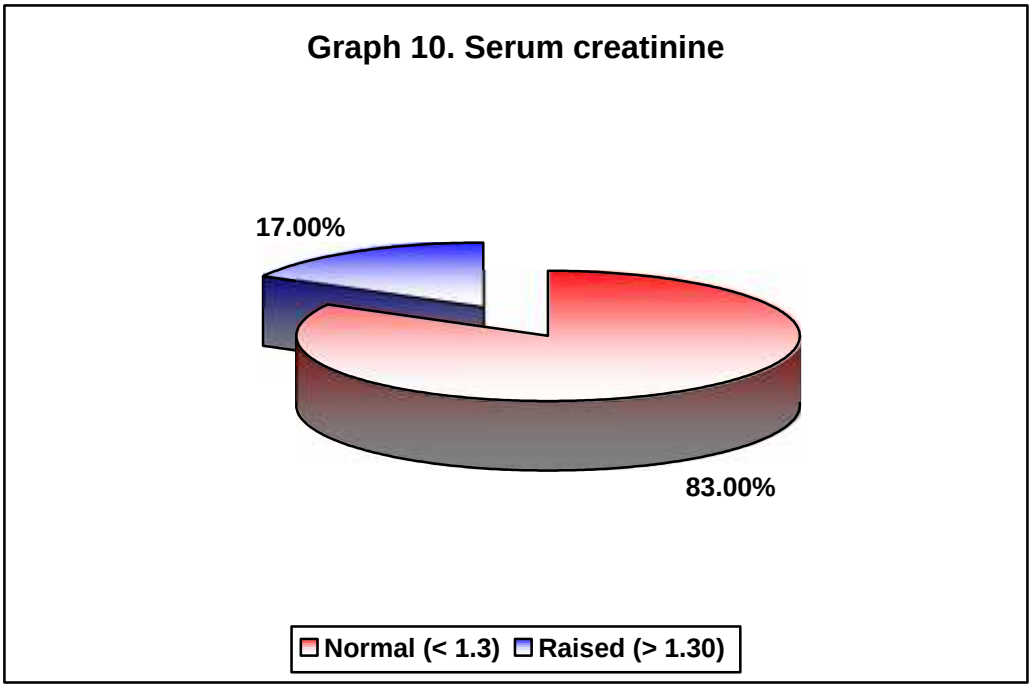
Blood urea (mg/dL)	Distribution (n=100)	
	Number	**Percentage**
Low (<21)	32	32.00
Normal (21-40)	52	52.00
High (>40)	16	16.00
Total	**100**	**100.00**



In the present study the blood urea nitrogen was raised in 16% of the patients.

Table 10. Serum creatinine

Serum creatinine (mg/dL)	Distribution (n=100)	
	Number	**Percentage**
Normal (<1.3)	83	83.00
Raised (>1.30)	17	17.00
Total	**100**	**100.00**

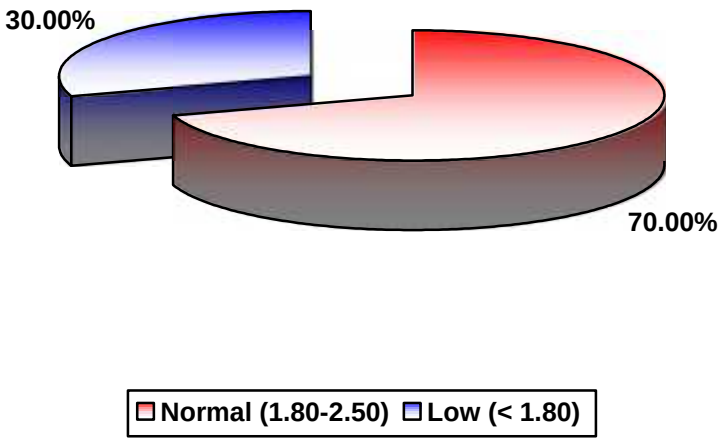


In this study raised serum creatinine (>1.30 mg/dL) levels were noted in 17% of the patients.

Table 11. Serum magnesium

Serum magnesium	Distribution (n=100)	
	Number	**Percentage**
Normal (1.80-2.50)	70	70.00
Low (<1.80)	30	30.00
Total	**100**	**100.00**

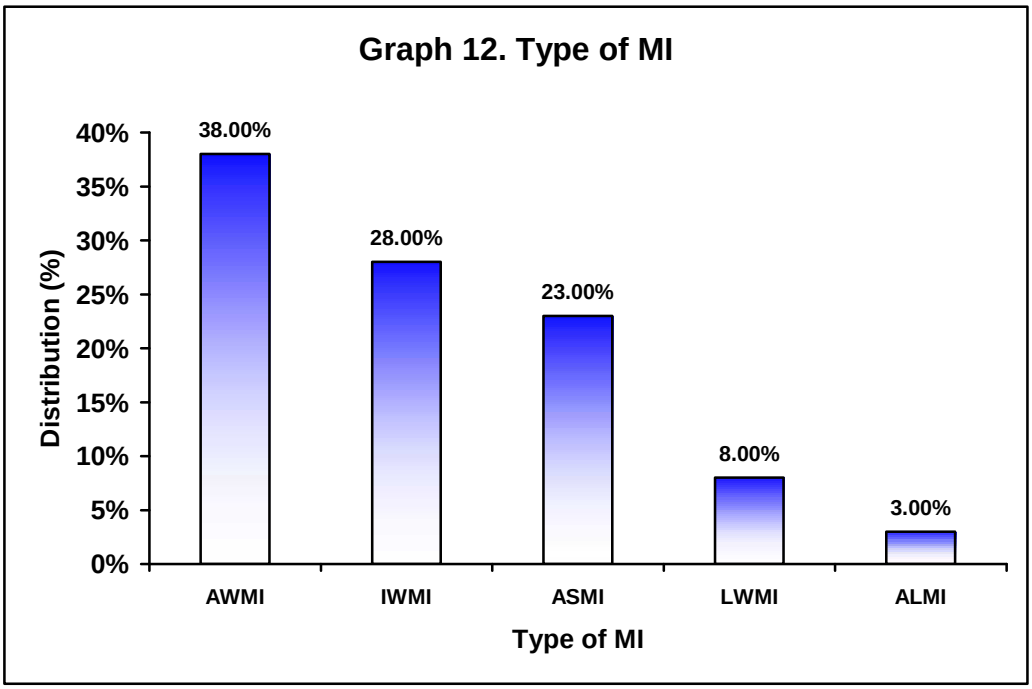
Graph 11. Serum magnesium



In this study serum magnesium levels were low (<1.80) in 30% of the patients.

Table 12. Type of MI

Type of MI	Distribution (n=100)	
	Number	Percentage
AWMI	38	38.00
IWMI	28	28.00
ASMI	23	23.00
LWMI	8	8.00
ALMI	3	3.00
Total	**100**	**100.00**

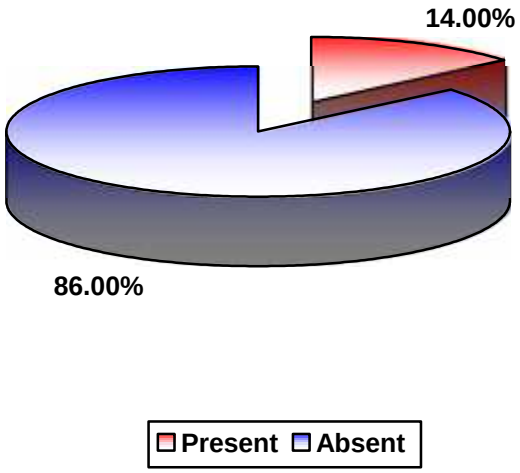


In the present study the commonest type of MI was AWMI (38%) followed by IWMI (28%), ASMI (23%), LWMI (8%) and ALMI (3%).

Table 13. Frequency of arrhythmia

Arrhythmia	Distribution (n=100)	
	Number	**Percentage**
Present	14	14.00
Absent	86	86.00
Total	**100**	**100.00**

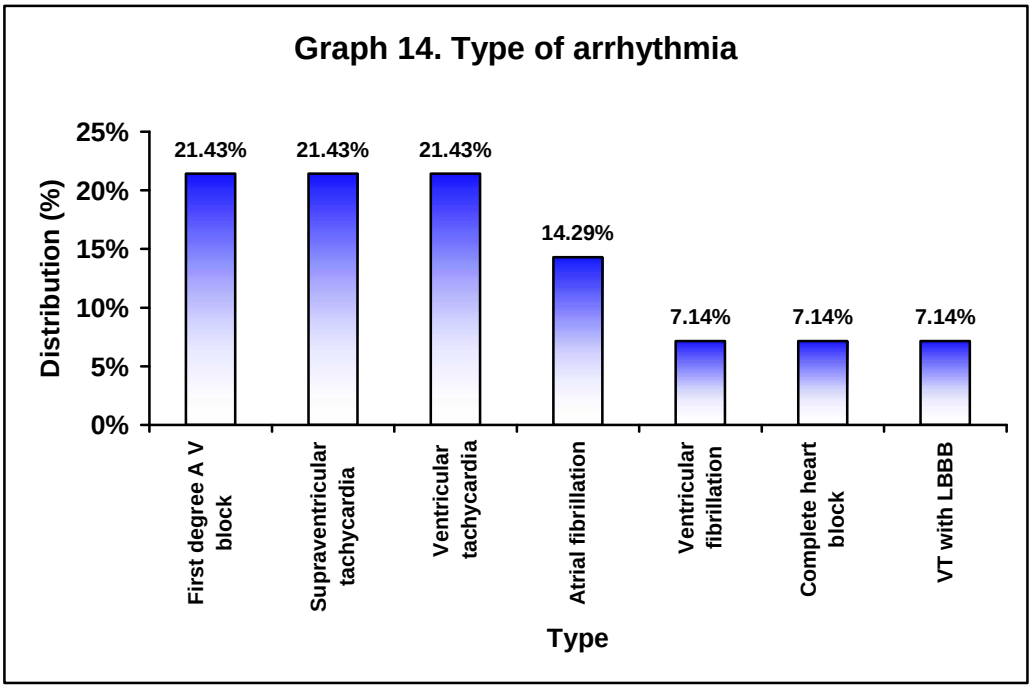
Graph 13. Frequency of arrhythmia



In this study arrhythmias were noted in 14% of the patients.

Table 14. Type of arrhythmia

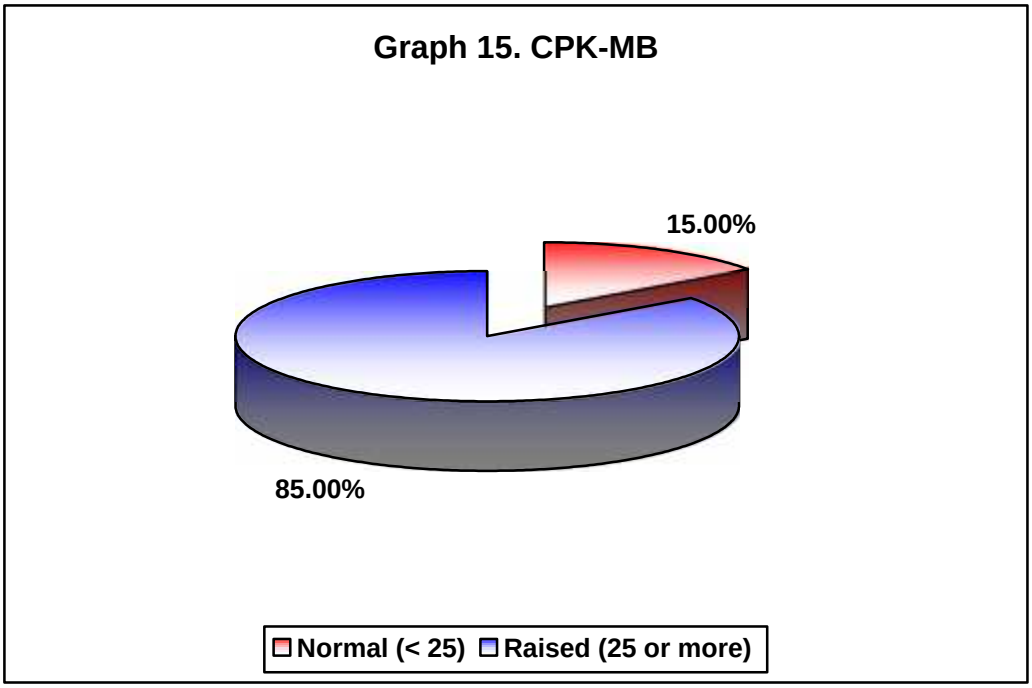
Type	Distribution (n=14)	
	Number	**Percentage**
First degree AV block	3	21.43
Supraventricular tachycardia	3	21.43
Ventricular tachycardia	3	21.43
Atrial fibrillation	2	14.29
Ventricular fibrillation	1	7.14
Complete heart block	1	7.14
VT with LBBB	1	7.14
Total	**14**	**100.00**



In this study, first degree AV block, supraventricular tachycardia and ventricular tachycardia were noted in 3 of the patients each (21.43% each).

Table 15. CPK-MB

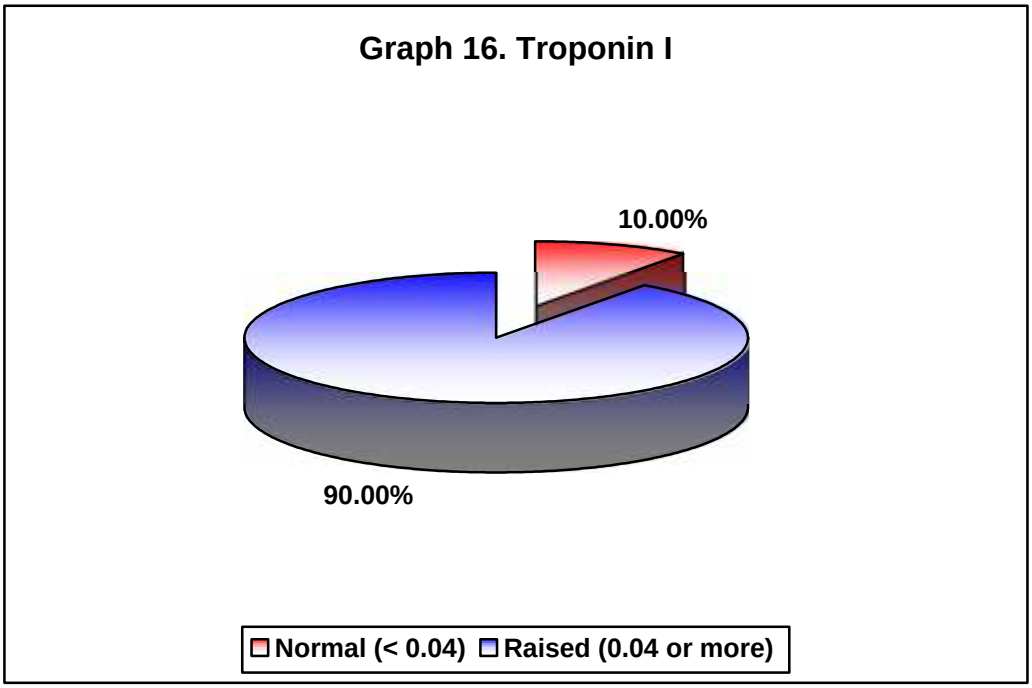
CPK-MB	Distribution (n=100)	
	Number	**Percentage**
Normal (<25)	15	15.00
Raised (25 or more)	85	85.00
Total	**100**	**100.00**



In the present study CPK-MB was raised in 85% of the patients.

Table 16. Troponin I

Troponin I	Distribution (n=100)	
	Number	**Percentage**
Normal (<0.04)	10	10.00
Raised (0.04 or more)	90	90.00
Total	**100**	**100.00**



In this study raised troponin I were noted in 90% of the patients.

Table 17. Association of arrhythmia with type of MI

Type of MI	Arrhythmia				Total	
	Absent		Present			
	No.	%	No.	%	No.	%
AWMI	32	84.21	6	15.79	38	100.00
ALMI	2	66.67	1	33.33	3	100.00
ASMI	20	86.96	3	13.04	23	100.00
IWMI	24	85.71	4	14.29	28	100.00
LWMI	8	100.00	0	0.00	8	100.00
Total	**86**	**86.00**	**14**	**14.00**	**100**	**100.00**

p = 0.641

In the present study no association was found between type of MI and arrhythmia (p=0.641).

Table 18. Association of arrhythmia with serum magnesium levels

Serum magnesium levels	Arrhythmia				Total	
	Absent		Present			
	No.	%	No.	%	No.	%
Normal (1.80-2.50)	68	97.14	2	2.86	70	100.00
Low (<1.80)	18	60.00	12	40.00	30	100.00
Total	**86**	**86.00**	**14**	**14.00**	**100**	**100.00**

p<0.001

In this study arrhythmias were noted in 40% of the patients with low serum magnesium levels while only 2.86% of the patients with normal serum magnesium levels had arrhythmias. This difference was statistically significant (p<0.001).

Table 19. Association of type of MI with serum magnesium levels

Type of MI	Serum magnesium levels				Total	
	Normal (1.80-2.50)		Low (<1.80)			
	No.	%	No.	%	No.	%
AWMI	26	68.42	12	31.58	38	100.00
ALMI	1	33.33	2	66.67	3	100.00
ASMI	14	60.87	9	39.13	23	100.00
IWMI	23	82.14	5	17.86	28	100.00
LWMI	6	75.00	2	25.00	8	100.00
Total	**70**	**70.00**	**30**	**30.00**	**100**	**100.00**

p = 0.268

In the present study the serum magnesium levels were comparable in patients with different types of MI (p=0.268).

Table 20. Comparison of mean serum magnesium levels with and without arrhythmia

Variable	Arrhythmia				p value
	Absent		Present		
	Mean	SD	Mean	SD	
Serum magnesium	2.07	0.39	1.53	0.35	<0.001

In the present study the serum magnesium levels were significantly high (2.07 ± 0.39) compared to patients who did develop complications of arrhythmia (1.53 ± 0.35) (p<0.001).

Table 21. Profile of study population

Variable	Mean		Median	Range	
	Mean	SD		Min	Max
Age (Years)	57.11	12.16	60	28	80
Temperature (^{0}F)	98.54	0.65	98.60	96	100
Pulse (/Minute)	87.54	17.06	88	34	130
Respiratory rate (/Minute)	17.13	3.45	16	12	34
Systolic BP (mm Hg)	122.96	25.09	118	70	210
Diastolic BP (mm Hg)	77.94	11.54	80	50	100
Haemoglobin (gm%)	13.42	2.50	13.85	1	22
White blood cell count (/Cumm)	13062.20	20535.67	10300	2200	211000
Platelet count (/Cumm)	316574.00	114439.28	275000	110000	569000
Blood urea (mg/dL)	27.73	11.92	25	12	85
Serum creatinine (mg/dL)	1.06	0.38	1.01	0.17	3.26
Serum magnesium (mg/dL)	1.99	0.42	2.10	0.90	2.50
CPK MB	61.65	43.86	50	12	250
Troponin I	2.77	5.85	1.20	0	50

The clinical and biochemical profile of the study population is as shown in table 21.

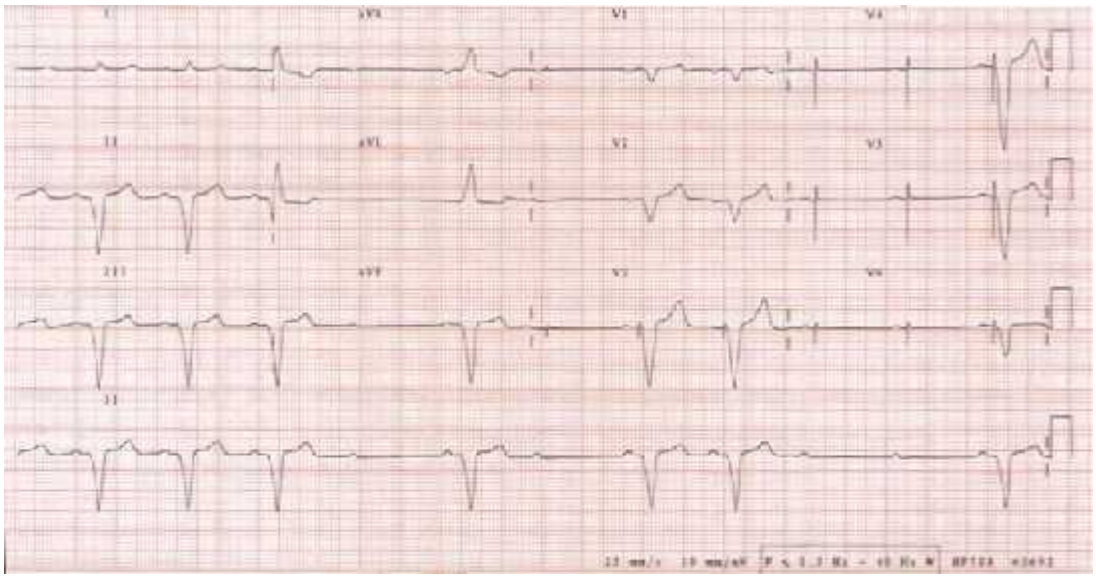


Figure 4. ECG showing complete heart block

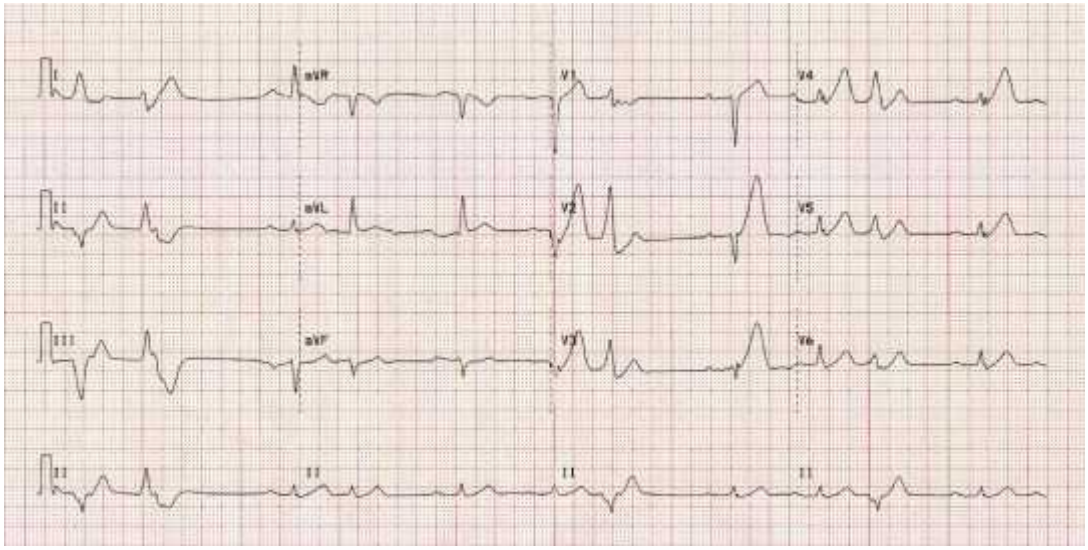


Figure 5. ECG showing first degree AV block in anterior wall MI

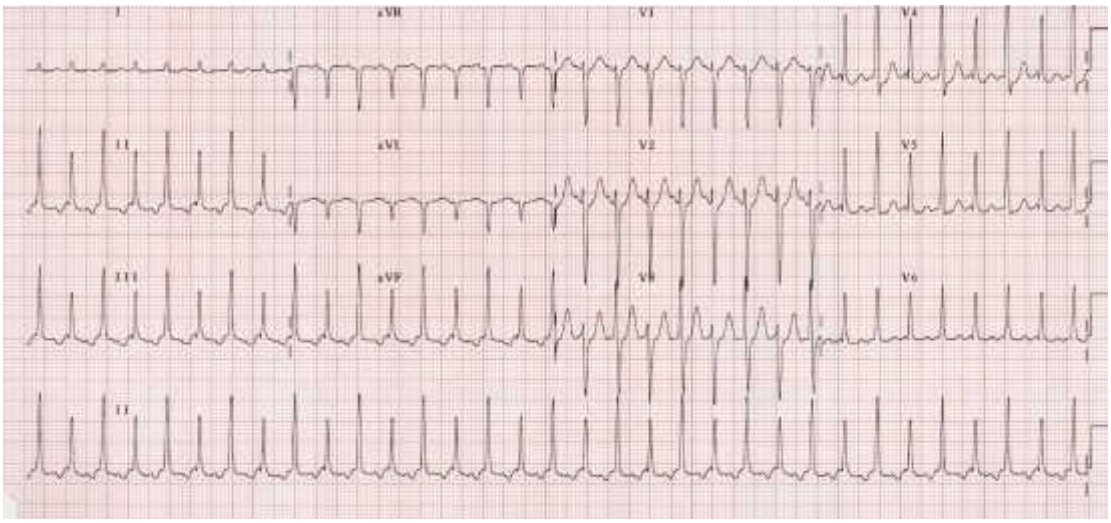


Figure 6. ECG showing supraventricular tachycardia

DISCUSSION

Acute coronary syndromes are reported to be a major cause of death in the world.[1,4] Despite the substantial progress in the management of coronary artery disease, it remains foremost and important cause of death all over the world. Many deaths are due to the development of arrhythmias during MI.[81] Arrhythmias are more common in AMI. Most of the patients with AMI develop some kind of arrhythmias. One of all the arrhythmias developed during initial 24 h with almost half of these occur during first hour after AMI.[82]

Cardiac arrhythmias and conduction abnormalities cause difficulties in AMI which have been associated with adverse prognosis in many reports. Almost any rhythm disturbance can be associated with acute myocardial infarction, including bradyarrhythmias, supraventricular tachyarrhythmias, ventricular arrhythmias and atrioventricular block.[82]

Recent reports suggest that patients with myocardial infarction who became critical and who died suddenly, had low serum magnesium levels.[13] Similarly, life-threatening arrhythmias were found to be more frequent in patients with acute myocardial infarction with low serum magnesium levels.[14,15] It was also shown that the magnesium content of the infracted/ischemic myocardium was much lower (about 40-50%) as compared to that of normal heart muscle. The magnesium depletion modifies coronary blood flow, blood clotting, and atherogenesis.[16] Magnesium lowers systemic vascular resistance, dilate coronary arteries, decrease platelet aggregation, improve myocardial metabolism, protect against catecholamine-induced myocardial necrosis, and stabilize cell membranes. It is also

cheap and easy to handle. Thus, it would appear to be an excellent contender for a place in the routine treatment of myocardial infarction, but it has not achieved this status yet. Therefore, the use of magnesium in myocardial infarction is a worthy topic of serious consideration.[62-69]

However, there are conflicting results of clinical trials examining the efficacy of magnesium administration for AMI and failed to establish conclusively whether magnesium therapy is useful for AMI or not. Hence this study was designed to find the frequency of arrhythmias in patients with AMI and to evaluate its relation with serum magnesium.[62-69]

This one year cross-sectional study was carried out in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. A total of 100 patients admitted with AMI from January 2014 to December 2014 were enrolled. Patients were evaluated for the serum magnesium levels and monitored for the development of arrhythmias.

In coronary artery disease previous studies have shown that the male gender is one of the classic risk factors for CAD.[83,84] The same was true in the present study. In the present study AMI was widely prevalent among males as 76% of the patients were males and male to female ratio was 3.16:1. These findings were strongly in agreement with a recent study from Bangalore by Subramanyam NT et al.,[85] who reported 76% of the males and 24% females. The findings of this study were also consistent with previous literature which showed that overall risk factors were more likely in males when compared to females.[83,84] The Copenhagen City Heart Study[86] with 21 year follow-up of 12000 men and women from reported incidence of CAD

in males as twice compared to female. Similarly, in a study by Hadjadj S, et al[87] the incidence of CAD in male (n=119) was twice that found in female (n=27). Recently, Jayachandra S, et al.[88] in Andhra Pradesh, India to determine the conventional risk factors of CAD in young and elderly aged patients also reported male preponderance. Comparison of data from the Reykjavik study[89] for male and female cohorts indicated higher incidence rates male subjects than females.[90]

Age is the most powerful independent risk factor for atherosclerosis. In the present study nearly half of the study population (47%) presented with age between 46 to 60 years and one third of the study population (36%) was aged > 60 years. The mean age was 57.11 ± 12.16 years with youngest patient being 28 years and oldest being 80 years. These findings suggest that, AMI was widely prevalent after fifth decade of life. Coronary artery disease in India is said to peak between 51- 60 years of age. Recently a study to evaluate serum magnesium levels in patients with AMI by Subramanyam NT et al.,[85] from Bangalore reported maximum number of patients were seen in the age group 51 -60 years (38%). The findings of the present study were consistent with another study by Mansoor AH, et al[91] who reported higher risk of acute coronary syndrome in average age group of 57.5 years among Indians. Similar observations were reported in The INTERHEART study[92] conducted in Southeast Asia, Japan, North America. The mean age observed in the present study that is, 57.11 ± 12.16 years was close to a recent study by Mehmoud KS et al[93] from Egypt (55.6 ± 8.8 years) and comparable with The CREATE-ECLA Randomized Controlled Trial.[94]

Chest pain has been reported as the cardinal feature in patients with AMI. According to the WHO guideline, presence of chest pain as one of the important

feature for the diagnosis of chest pain.[95] The present study was exception as majority of the patients (90%) had chest pain while shortness of breath was reported by 36% of the patients. This was similarly described in Huggins GS et al.[96] in 1996.

Hyperinsulinemia, insulin resistance, and the higher rate of prevalence of metabolic syndrome in people with type-2 diabetes were attributed to high coronary risk in south Asians.[97,98] Indians are genetically prone to develop type-2 diabetes mellitus due to insulin resistance. The hyperinsulinimia accelerates the atherosclerotic process in the coronary arteries. During the past decade, the number of people with diabetes in India increased from 32 million to 50 million, and the projected figure may reach 87 million by 2030.[99] In this study other associated comorbidities were history of hypertension and diabetes mellitus which were present in one third of the study population (36% and 33% of the patients respectively). These findings were comparable with a recent study by Mehmoud KS et al[93] from Egypt in patients with acute ST-segment elevation myocardial infarction where hypertension was noted in 46% and diabetes in 34% of the patients. A study by Murangmei L. et al.[10] from Manipur India reported Risk factors diabetes mellitus in 22% and hypertension in 26%.

In this study laboratory parameters revealed lower haemoglobin levels in 28% of the patients (<13 gm% in males and <12 gm% in females) and raised white blood cell count (>12000 /cumm) in 28% of the patients while platelet count was low (<150000 /Cumm) in 5% of the patients. Blood urea nitrogen and serum creatinine levels were raised in 16% and 17% of the study population.

In the present study more than one third that is, 38% of the patients had AWMI and IWMI, ASMI, LWMI and ALMI were noted in 28%, 23%, 8% and 3% respectively. Similar type of AMI pattern was reported by Patil BM[82] in their study to identify the type of arrhythmias and outcome in patients presenting with acute myocardial infarction. He reported most of the infarctions in anterior wall (55.24%) followed by inferior wall MI (40.78%). The AMI type observed in the present study was also similar to a recent study by Rathod S. et al.[100] where AWMI was noted in 52% and IWMI in 25%.

In this study nearly one third of the study population (30%) presented with lower serum magnesium levels (<1.80). The mean serum magnesium levels were 1.99 ± 0.42 mg/dL with lowest value being 0.90 and highest being 2.50 mg/dL. However, the serum magnesium levels were comparable in patients with different types of MI (p=0.268). These findings suggest that, a considerable subset of patients presenting with AMI are likely to have low serum magnesium levels irrespective of the type of AMI. Several other studies[12-14,62-69,85] have reported lower magnesium levels in patients with AMI.

Wannasilp N et al.[101] 2001 demonstrated that CAD patients may be associated with Mg deficiency and contribute to the pathogenesis of CAD.(11) They reported Mean value of serum Mg level in 100 CAD patients as 2.14±0.39 mg/dl (p= 0.052) with 100 healthy controls (mean value 2.24± 0.3 mg/dl) in the study. However, the prevalence of Mg deficiency did not differ significantly between the study and control groups however it tended to be higher in CAD patients.

Simmikharb et al.[102] 1999 demonstrated mean serum Mg levels in 22 acute MI cases to be 1.27±0.57 mg/dl ($p<0.001$) compared to 2.41±0.54 mg/dl mean value in 15 controls. They also concluded that Mg deficiency in MI patients can potentiate oxidative injury to post ischemic myocardium.

A study by Subramanyam NT. et al.[85] to evaluate the prognostic value of serum magnesium in acute myocardial infarction reported mean serum magnesium levels of 1.62±0.27 mg/dl with range from 1.2 to 3.3 mg/dl.

Khan GQ et al.[103] 2002 reported statistically significant ($p < 0.001$) fall of serum Mg in 50 cases of acute MI with mean serum Mg in controls being 2.2±0.24 mg/dl. Further, the serum Mg level was found comparatively lower in the patients getting cardiac arrhythmias. They concluded that, the fall in serum Mg in acute MI can be taken as sensitive diagnostic index, especially in early hours of postinfarction period when cardiac enzymes and ECG may not be significant.

Mohan G. et al.[51] demonstrated that mean serum Mg levels in 53 acute MI cases as 1.38±0.21 mg/dl compared to mean value of 2.51±0.16 mg% in 30 controls ($p<0.001$).

Though the finding of this study could not be compared with these studies[51,85,101-103] due to the methodological and sample variations, they strongly suggest that, a considerable subset of patients presenting with AMI are likely to have lower serum magnesium levels.

In this study arrhythmia was noted in 14 (14%) patients. Among these, three patients each had first degree AV block, supraventricular tachycardia and ventricular tachycardia (21.43% each) and atrial fibrillation was present in two patients (14.29%

each) while one case each (7.14% each) had ventricular fibrillation, complete heart block and VT with LBBB. The frequency of arrhythmias observed in the present study was low compared to other studies in India. A recent study by Rathod S. et al.[100] from Manipur, India reported arrhythmias in 76 cases while 24 cases did not develop arrhythmia. More recently another study by Toshniwal SP. et al.[104] from Maharashtra reported arrhythmias in 79.66% of the patients. In a study by Aufderheide TP,[105] 90% of patients with acute MI have some cardiac rhythm abnormality during the first 24 h following infarct onset. The lower incidence of arrhythmias in the present study can be attributed to the intensive interdisciplinary care of the patients and preventive strategies during their stay ICU following AMI.

In the present study of the 14 patients with arrhythmia, six (15.79%) were seen in patients with AWMI, four (14.29%) in patients with IWMI and three (13.04%) in ASMI while one cases (33.33%) was note in patients with ALMI. No association was found between type of MI and arrhythmia ($p=0.641$). The lack of association observed between AMI and arrhythmias in the present study can be explained by the smaller sub-set of patients with AMI and wide variation in the type of AMI.

In this study significantly higher number of patients with low serum magnesium levels developed arrhythmias. Of the 14 patients who developed arrhythmia, 12 (40%) patients had lower serum magnesium levels and only 2 (2.86%) with normal serum magnesium levels developed arrhythmias ($p<0.001$). Furthermore, the serum magnesium levels were significantly low in patients who developed arrhythmias (1.53 ± 0.35 vs 2.07 ± 0.39; $p<0.001$). These findings suggest that, patients presenting with AMI and lower serum magnesium levels are

significantly at high risk of developing cardiac arrhythmias. Despite limited number of patients with cardiac arrhythmia in the present study the there was strong association between lower serum magnesium levels and development of arrhythmias. Direct comparison of these findings was not possible due to the scarcity of recent data in the literature. However the older studies have reported similar association and relationship between development of arrhythmia and lower serum magnesium levels in patients with AMI.

Earlier in 1987, Rasmussen HS et al.[106] in a double-blind placebo-controlled study on 130 patients with AMI administered magnesium or placebo treatment intravenously immediately upon admission to hospital. The incidence of arrhythmias requiring treatment during the initial week of hospitalization was registered. Serum magnesium concentrations were increased from 0.7 mmol/l to 1.3 mmol/l as a result of the magnesium infusions. This pharmacologically induced hypermagnesemia resulted in a reduction in the incidence of arrhythrmas from 47% in the placebo group to 21% in the magnesium group ($p=0.003$). In the magnesium-treated patients, increments in serum concentrations of magnesium and potassium correlated positively ($r=0.47$, $p<0.001$). They concluded that, magnesium infusion in the postinfarct period reduces the incidence of supraventricular tachyarrhythmias.

Mohan G. et al.[51] demonstrated that lower serum Mg levels of 1.26±0.19 mg/dl on 1st day in 42 cases of acute MI with complications compared to 1.41 ±0.13 mg/dl in all 11 cases of acute MI without complications. By 10th day, the mean serum Mg level in acute MI with complications raised to 2.36 ±0.12 mg/dl compared to mean serum Mg values in acute MI without complications of 2.29±0.16 mg%. It

was observed that serum Mg were lowest in patients who died due to major arrhythmias and cardiogenic shock followed by pump failure.(14)

Iseri et al.[107] in their study treated multifocal atrial tachycardias successfully with parenteral magnesium and potassium. Magnesium administered together with potassium, stabilized the ionic balance of the cells and thus prevents spontaneous ectopics.

Ceremuzynski L. et al.,[108] proved that life threatening arrhythmias in AMI are prevented by I.V. magnesium sulfate. This was in agreement with the findings of Rasmussen et al., and Smith et al.,[109] Schechter et al.,[110] and this encourages implementation of magnesium treatment into clinical practice.

Ising et al.,[111] performed seven 24 hours electrocardiograph (ECG) recordings and blood samples were taken within three weeks in 42 patients. Ca++, K+, and Mg++ concentrations in serum, and K+ and Mg++ in the erythrocytes, were determined by atomic absorption spectroscopy. One half of the patients were infused with 81 mmol/day as MgSO4 for 3 days. In patients who exhibited intense electrolytic alterations 10-20 days after AMI, there was a significantly higher rate in the frequency of couplets and/or tachycardia in the 2-20 days period after AMI. In patients infused with MgSO4, the fluctuation in serum electrolytes and the rate of arrhythmias were significantly reduced.

Woods KL. et al.,[62] proved in “Leicester Intravenous magnesium intervention trials”, the efficacy of I.V. magnesium in reducing the mortality in patients of acute myocardial infarction. They conducted a double blind placebo controlled study in 2,316 patients who received either I.V. magnesium sulfate or

physiological saline. The primary outcome measure was the 28 days mortality which was ascertained in 99.3% of patients. The groups were well balanced for prognostic factors. By intention to treat, mortality from all causes was 7.8% in the magnesium group and 10.3% in the placebo group, a relative reduction of 2-4%. Teo KK. et al.[61] conducted a study of the seven randomized trials collectively and proved that there were 25 (3.8%) deaths among 657 patients allocated to receive magnesium and 53 (8.2%) deaths among 644 patients allocated control during the 4-week-period. This represents a 55% reduction in the odds of death ($P < 0.001$). A number of reports particularly from European countries have shown subjective and objective improvements in patients with ischemic heart disease treated with magnesium salts.

The Second Leicester Intravenous Magnesium Intervention Trial (LIMIT-2) included 2,316 patients, who were randomized to receive I.V. magnesium sulfate or matching placebo. Patients received placebo or magnesium for 5 min before initiation of thrombolytic therapy, followed by an infusion for the next 24 h. It concluded that there was 24% reduction in 28-day mortality, a 25% reduced incidence of left ventricular failure, and an improvement in long-term survival in terms of reduction of long-term mortality from ischemic heart disease (average follow-up period of 2.7 years).[62,63]

The findings of the present study were in agreement with these studies in the literature which hypothesize that, serum magnesium therapy in patients with AMI is beneficial in avoiding complications especially arrhythmias.

Magnesium probably functions as an inorganic calcium channel blocker and there are several plausible mechanisms for a beneficial effect in acute myocardial

infarction.[62] Research on animals and humans has shown that magnesium is a peripheral and coronary vasodilator. It can increase the threshold for depolarization of cardiac myocytes, thereby reducing the likelihood of cardiac arrhythmia caused by injury currents near ischemic or infarcted tissue. Magnesium decreases reperfusion injury by preventing or lessening mitochondrial calcium overload in ischemic myocardial cells during the first few minutes of reperfusion (namely, the restoration of blood flow to an organ or tissue) and preserving intracellular Adenosine Triphosphate (ATP) and creatine phosphate reserves, and inhibits platelet function, perhaps indirectly by release of prostacyclin. Thus, magnesium-infusion started early after the onset of myocardial ischemia might limit infarct size, prevent serious arrhythmias, and reduce mortality.[12]

Magnesium therapy has numerous desirable properties for the treatment of myocardial infarction. It is a coronary and systemic vasodilator, calcium antagonist, antiarrhythmic agent, and antiplatelet drug that modulates autonomic function and limits reperfusion injury when given early in the setting of acute myocardial infarction. Clinical trials of magnesium therapy for acute myocardial infarction have yielded conflicting results. Despite the very large number of patients enrolled in ISIS-4, the late administration of magnesium therapy coupled with a low control group mortality rate may have biased the trial to a null effect of magnesium. Indeed, the relative safety and low cost of magnesium, its numerous salutary properties, and its reduction of mortality in patients ineligible for thrombolytic therapy buttress the contention that additional clinical trials that focus on early administration of magnesium are needed to define precisely the role of magnesium therapy for acute myocardial infarction. Such trials will be initiated shortly.[52]

CONCLUSION

Based on the findings of this study it may be concluded that, patients with AMI are prone to hypomagnesemia immediately after the development of the infarction. Furthermore there is strong association between development of cardiac arrhythmias with hypomagnesemia in patients with AMI irrespective of the type of infarction. This prompts evaluation of serum magnesium levels in patients presenting with AMI and administer appropriate magnesium therapy which may reduce the incidence of arrhythmias.

SUMMARY

Life threatening arrhythmias are more frequent in patients with AMI with low serum magnesium levels. The present study was planned to evaluate the relationship between arrhythmias and serum magnesium levels in patients with acute myocardial infarction.

This one year cross-sectional study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. A total of 100 patients who were admitted with acute myocardial infarction from January 2014 to December 2014 were studied. All the patients underwent estimation of serum magnesium levels and monitored for arrhythmias.

More than three fourth of the study population was comprised of males (76%) and male to female ratio was noted as 3.16:1. Most of the patients were aged between 46 to 60 years (47%) and mean age was 57.11 ± 12.16 years. History of hypertension and diabetes mellitus present in 36% and 33% of the patients respectively. Majority of the patients (90%) presented with chest pain and 17% of the patients had crepitations. The commonest type of MI was AWMI (38%) followed by IWMI (28%). Serum magnesium levels were low (<1.80) in 30% of the patients. Arrhythmias were noted in 14 (14%) patients and 3 patients each (21.43% each) had first degree AV block, supraventricular tachycardia and ventricular tachycardia. Significantly higher number of patients with low serum magnesium levels had arrhythmias (40% vs 2.86%; $p<0.001$) and also mean serum magnesium levels were significantly low in patients who had arrhythmias (1.52 ± 0.34 vs 2.10 ±

0.43; $p<0.001$). No association was found between types of arrhythmia with serum magnesium levels.

Patients presenting with AMI are likely to have hypomagnesemia and these patients have high risk of cardiac arrhythmias. Hence, patients presenting with AMI should be evaluated for serum magnesium levels and those having hypomagnesemia should be offered magnesium supplementation so as to avoid the complications of arrhythmias.

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ANNEXURE I – CONSENT FORM

TITLE OF RESEARCH STUDY: "CORRELATION BETWEEN SERUM MAGNESIUM AND ARRHYTHMIAS IN ACUTE MYOCARDIAL INFARCTION – ONE YEAR CROSS SECTIONAL STUDY IN A TERTIARY CARE HOSPITAL"

Principal Investigator
Dr. *** *******,**
Post Graduate Student,
Department Of General Medicine,
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Guide
Dr. **** *******,**
Professor,
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Introduction and Purpose

In acute myocardial infarction patients, low magnesium levels are more prone to get arrhythmias, so by knowing magnesium level we can prevent arrhythmias in acute myocardial infarction by supplementing intravenous magnesium. Sudden cardiac death is very common by arrhythmias in myocardial infarction. In this study correlation is done between magnesium and arrhythmias in acute myocardial infarction.

Procedure

If you agree to be part of the research study, you will be asked the history of any chest pain, associated with sweating, radiating to left shoulder/neck and will be subjected to relevant examination and investigations like serum magnesium levels. You will also have to give blood and urine samples for the necessary investigations.

Risk and Benefits

The only risk and possible discomfort you might get is while taking blood from my arm for the investigations. It may cause swelling, pain, redness, bruising or infection (rarely happens) at the site from where the blood is drawn.

The benefit is that it helps in risk stratification of acute myocardial infarction and arrhythmias.

Alternatives

Taking part in this study is voluntary. You may choose not to take part in this study, or if I decide to take part I can later change my mind and withdraw from the study. Your decision will not change the present or future health care or other services that you receive. The study doctor or sponsor may stop your participation in this study at any time. If you choose not to take part in the study, you will receive the standard treatment for patients with your condition.

Privacy and Confidentiality

All information collected about you during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify you in this research record. Information from this study may be published but your identity will be confidential in any publication.

Institution / Sponsor's policy / compensation

In case of any injury related to the study, treatment will be made available at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. There is no compensation or payment for such medical treatment by law.

Financial incentives for participation

You will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results

`The results of the study would be forwarded to the KLE University, Belgaum as part of requirement towards the completion of MD degree, review and publishing.

Questions

During study or in future you may contact following persons,

1**. Dr. ***** ********,**
Investigator,
Post Graduate in General Medicine,
Jawaharlal Nehru Medical College,
Belgaum – 590 010

2. Dr. *** *******,**
Professor,
Dept of General Medicine,
Jawaharlal Nehru Medical
Belgaum – 590 010

3. Dr. **** ********,**
Chairman,
Jawaharlal Nehru Medical College,
Ethical Committee for Human Research,
Jawaharlal Nehru Medical College,
Belgaum – 590 010
Phone Number: ***** ******

CONSENT STATEMENT

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read, or it has been read to me, this entire consent form, and have had all my questions answered.

Name of the Participant: _____

Signature / Thumb print: _____

Name of the Witness: _____

Signature/ Thumb print: _____

Investigator Name: _____

Signature: _____

ANNEXURE II – PROFORMA

TITLE OF RESEARCH STUDY: "CORRELATION BETWEEN SERUM MAGNESIUM AND ARRHYTHMIAS IN ACUTE MYOCARDIAL INFARCTION – ONE YEAR CROSS SECTIONAL STUDY IN A TERTIARY CARE HOSPITAL"

Case no :

Name :

Age / Sex :

In Patient Number :

Address :

Occupation :

Complaints at presentation	Yes	No
Chest pain		
Palpitations		
Shortness of breath		
Paroxysmal nocturnal dyspnea		
Orthopnea		
Syncope		
Heamoptysis		

Past history

Treatment history

Physical examination

General condition

Pallor	:	yes / no
Icterus	:	yes / no
Lymphadenopathy	:	yes / no
Cyanosis	:	yes / no
Clubbing	:	yes / no
Edema	:	yes / no

Vitals:

Temperature	:
Pulse	:
Respiratory rate	:
Blood pressure	:

Systemic examination

Respiratory system	:
Cardiovascular system	:
Central nervous system	:

Investigations

Complete blood count	:
Serum magnesium level	:
12-lead Electrocardiography	:
Echocardiography	:
Cpk-mb, Troponin – I	:
Renal function test	:
Thyroid function tests (if required)	:
Liver function tests	:

ANNEXURE III – KEY TO MASTER CHART

-	-	Absent
/Cumm	-	Per cubic millimeter
+	-	Present
A/E	-	Air entry
AF	-	Atrial fibrillation
AIWMI	-	Anterio inferior wall myocardial infarction
ALMI	-	Anterolateral wall myocardial infarction
ASMI	-	Anteroseptal wall myocardial infarction
AWMI	-	Anterior wall myocardial infarction
B/L	-	Bilateral
BP	-	Blood Pressure
CK MB	-	Creatine kinase MB
Crep	-	Crepitations
ECG	-	Electrocardiogram
F	-	Female
gm	-	Grams
IWMI	-	Inferior wall myocardial infarction
LBBB	-	Left bundle branch block
LWMI	-	Lateral wall myocardial infarction
M	-	Male
mg/dL	-	Milligrams per deciliter
MI	-	Myocardial infarction
mm Hg	-	Millimeters of mercury

SVT	-	Suptraventricular tachycardia
VF	-	Ventricular fibrillation
VT	-	Ventricular tachycardia