A Dissertation on

"SERUM MAGNESIUM LEVELS IN ACUTE MYOCARDIAL INFARCTION "

Dissertation submitted to

THE TAMILNADU Dr.M.G.R. MEDICAL UNIVERSITY

CHENNAI – 600 032

With partial fulfillment of the regulations

For the award of the degree of

M.D. GENERAL MEDICINE

BRANCH – I

COIMBATORE MEDICAL COLLEGE

COIMBATORE

CERTIFICATE

This is to certify that the dissertation **"SERUM MAGNESIUM LEVELS IN ACUTE MYOCARDIAL INFARCTION"** is a bonafide research work done by **Dr. J. ANANDARAJ** Post graduate in M.D. General Medicine under my direct guidance and supervision to my satisfaction, in partial fulfillment of the requirements for the degree of M.D. General Medicine.

Date : **Professor & Unit chief M-6**

Date : **Professor & Head Of The Department**

Department of General Medicine

Date : **The Dean, Coimbatore medical college**

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serum magnesium levels in acute myocardial infarction

BY ANANDHU JAYARAMAN

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DECLARATION

I hereby declare that this dissertation entitled **"SERUM MAGNESIUM LEVELS IN ACUTE MYOCARDIAL INFARCTION"** is a bonafide and genuine research work carried out by me under the guidance of Prof. Dr. Isaac Christian Moses M.D., Department of Medicine ,Coimbatore medical college, Coimbatore .

Date :

Place : **Dr. J.ANANDARAJ**

ACKNOWLEDGEMENT

I realize that dissertation being a work of cooperation and assistance, it would be far from complete without due acknowledgement of the help gratefully received.

It is my distinct honour and privilege to have under the able supervision of my teacher, **Prof. Dr. Isaac Christian Moses M.D.,** Professor, Department of Medicine, Coimbatore medical college , Coimbatore. Indeed, I am fortunate to get the benefit of his vast experience, valuable guidance and advise at every step of my study. It is with humble gratitude, I pay respects and thanks to him for his keen interest in the study and guidance for the preparation of this dissertation.

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the Department of Medicine for their valuable suggestions.

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I am very much thankful to my family, friends and colleagues especially for their kind cooperation.

I feel indebted to my patients and hospital, laboratory staff for their kind cooperation in the study.

Date:
Place: **Dr. J. ANANDARAJ**

LIST OF ABBREVIATIONS USED

AF .....Atrial fibrillation

CAD .....Coronary artery disease

ATP .....Adenosine triphosphate

VT .....Ventricular tachycardia

VF .....Ventricular fibrillation

ECG.....Electrocardiogram

IHD .....Ischemic heart disease

CCF .....Congestive cardiac failure

LVF .....Left ventricular failure

JVP.....Jugular venous pressure

$MgSO_4$.....Magnesium sulphate

ACLS .....Advanced cardiac life support

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ABSTRACT

Background: Magnesium has been implicated in the pathogenesis of acute myocardial infarction and its complication like arrhythmia. Magnesium improves myocardial metabolism, inhibits calcium accumulation and myocardial cell death. It improves vascular tone, peripheral vascular resistance, after load and cardiac output, reduces cardiac arrhythmias and improves lipid metabolism. Magnesium also reduces vulnerability to oxygen derived free radicals, improves endothelial function and inhibits platelet function including platelet aggregation and adhesion.

Objective: To know the relationship between the serum magnesium levels and arrhythmias in patients with acute myocardial infarction.

Method: By using simple random method, 50 cases of acute myocardial infarction, admitted in Coimbatore Medical College and Hospital.

Results: There is a significant difference in the magnesium levels in patients with arrhythmias and without arrhythmias.

Conclusion: In acute myocardial infarction, patients with low magnesium levels are more prone to get arrhythmias. So magnesium treatment can be considered in patients of acute myocardial infarction with low magnesium levels.

Key words: Magnesium; Myocardial infarction; Arrhythmias.

INTRODUCTION

It has been known that inorganic salts are necessary for the normal growth and functioning of all biological forms. Pasteur (1860) proposed that yeast will grow only when inorganic compounds were present in the culture medium. In the human body, there is a maintenance of fluid balance, not only as a whole but also intercompartmentally between the three compartments such as intracellular, interstitial and intravascular fluid compartments. Various forces such as hemodynamic, electrolyte and other forces act together and contribute to maintain the normal fluid balance of the body. It is now evident that not only proteins, fats and carbohydrates, but also minerals play an essential role in the normal homeostasis of the body. Intensive investigation is now going on about the importance of trace elements not only of vitamins but also of minerals.

Magnesium has been known to have an influence in the causation of acute myocardial infarction and also its sequelae like arrhythmias. It plays a major role in the pathogenesis of other cardiovascular diseases as well. Magnesium ions are found to be essential for the maintenance of the normal functional integrity of the myocardium[1].

Several investigations have shown that the serum magnesium level is low in the first 48 hours following a acute myocardial infarction and later on rose gradually to attain the normal level in about three weeks time. Infarcted myocardium was found to have reduced magnesium concentration. The above said findings correlated directly with the associated complications of acute myocardial infarction, such as arrhythmias.

In patients with sudden death because of ischemic heart disease, magnesium concentration in the cardiac muscle was found to be decreased[2]. Hypomagnesium acts a provoking factor in the occurrence of ventricular fibrillation, which is usually the cause of sudden death in IHD. The coronary vasospasm which occurs as a result of hypomagnesemia has been considered as an important factor in the causation of sudden death in IHD.

Magnesium deficiency contributes to the progression of atheromatous plaques occuring as a result of hyperlipidemia.

Myocardial infarction is one of the common causes of death where its prognosis depends on various factors. This study is designed to know the contribution of magnesium levels in the serum of acute MI patients to the occurrence of arrhythmias.

AIMS AND OBJECTIVES

To know the correlation between serum magnesium levels and arrhythmias in patients with acute myocardial infarction who are presenting within 12 hours of onset of symptoms.

REVIEW OF LITERATURE

Historical Review

During the time of Lavoisier (1743-1794) only 26 elements were discovered. Followed shortly by the discovery of elements such as sodium, potassium, calcium and magnesium. It was only Liebig (1803-1873) who appreciated the significance of the minerals as normal constituents of plants and animal tissues. Eventhough the inorganic constituents of the body constitute only small fraction of the total amount, they should not be considered as insignificant. As a matter of fact, they are found to play a pivotal role in the maintenance of the normal homeostasis of the body.

Greenberg and associates in 1936 demonstrated myocardial degeneration associated with fibrosis and polyplastic infiltration in rats fed on diets low in magnesium since birth (Burch et al, 1977)[1]. From the sequence of structural abnormalities observed it was interpreted that interference with the enzymes dependent on magnesium which are involved in oxidative phosphorylation was responsible for the pathogenesis of the lesion observed.

The term 'magnesium' derived its name from the name of ancient Gracian town of 'Magnesia'(a district of Thessaly). Sir

Humphrey Davy in 1808, investigated the alkaline earth metal and proposed the white magnesia stone by its modern name 'magnesium'.

Until the middle of twentieth century, magnesium metabolism had not been given much importance, though extensive literature about the consequences of magnesium deficiency in the lower species was available. During this period, the progress of the work on magnesium metabolism in man was very slow because of the lack of uniformity and difficulty in estimating magnesium levels and also the magnesium status of the body.

Now the availability of more accurate and uniform means of serum magnesium estimation in the laboratory has escalated the work on magnesium metabolism in man to greater heights.

Only 1% of total body magnesium is present in the extra-cellular fluid and out of this, about 25% is present in the plasma, rest is seen in the red cells. About 50% of serum magnesium is found to be free, 32% remains bound to proteins and the rest 13% exists as magnesium phosphate, citrate and other unidentified complexes.

Since the intravascular space contributes to only a fractional

portion of the body involved in the homeostasis of magnesium, it is clear that the estimation of serum magnesium levels alone does not always indicate the actual total body magnesium stores.

As a matter of fact, magnesium deficiency can occur when the intracellular magnesium content is normal and intracellular magnesium deficiency may occur without any decrease in serum magnesium levels (Vermon et al, 1978)[3].

Nevertheless, compared to the complex studies on determination of the tissue levels, measurement of serum magnesium is the rapid, simple and most effective approach for the assessment of magnesium deficiency states. Measurement of the serum magnesium levels has been used widely for identifying various clinical syndromes that have responded well to the replacement therapy with magnesium salts.

CORONARY CIRCULATION[04]

Anatomy of Coronary circulation

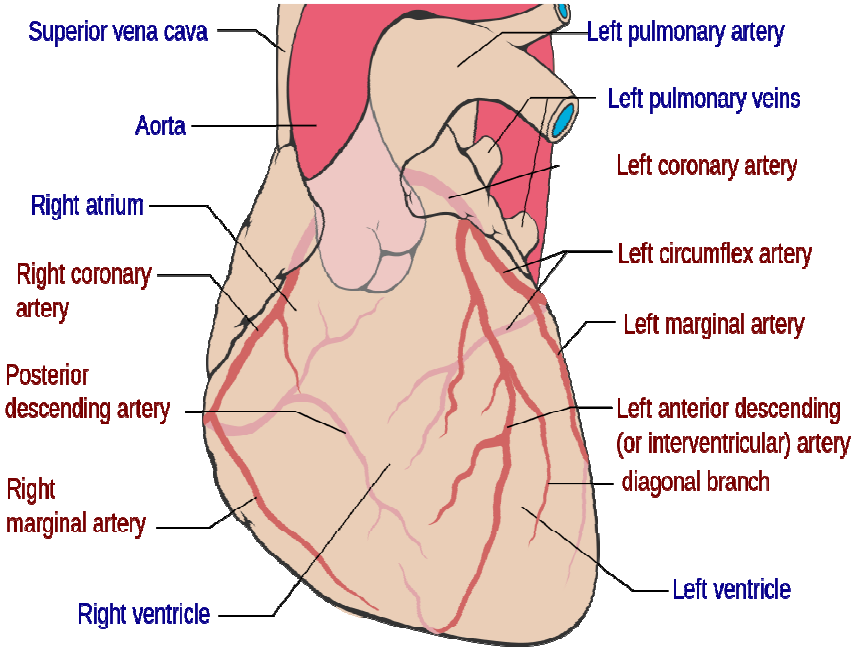
Coronary arterial system supplies blood to the heart. It is comprised of the right coronary artery and the left coronary artery.

The origin of right coronary artery is from the anterior aortic sinus. Soon after its origin, it courses between the right auricular appendage and the infundibular portion of the right ventricle. It traverses in the atrioventricular groove passing vertically downwards. Then the artery turns posteriorly at the lower border of the heart and courses posteriorly. It gives off several branches to both atria and ventricles as it passes down the atrio ventricular groove. At the inferior border of the heart, the marginal branch traverses to the left along the right ventricle. The inferior interventricular branch arises on the diaphragmatic surface and courses along the groove in between the ventrices to the cardiac apex. The terminal branches of right coronary artery anastomoses with the terminal arterioles of the left coronary artery at the lower aspect of left atrium.

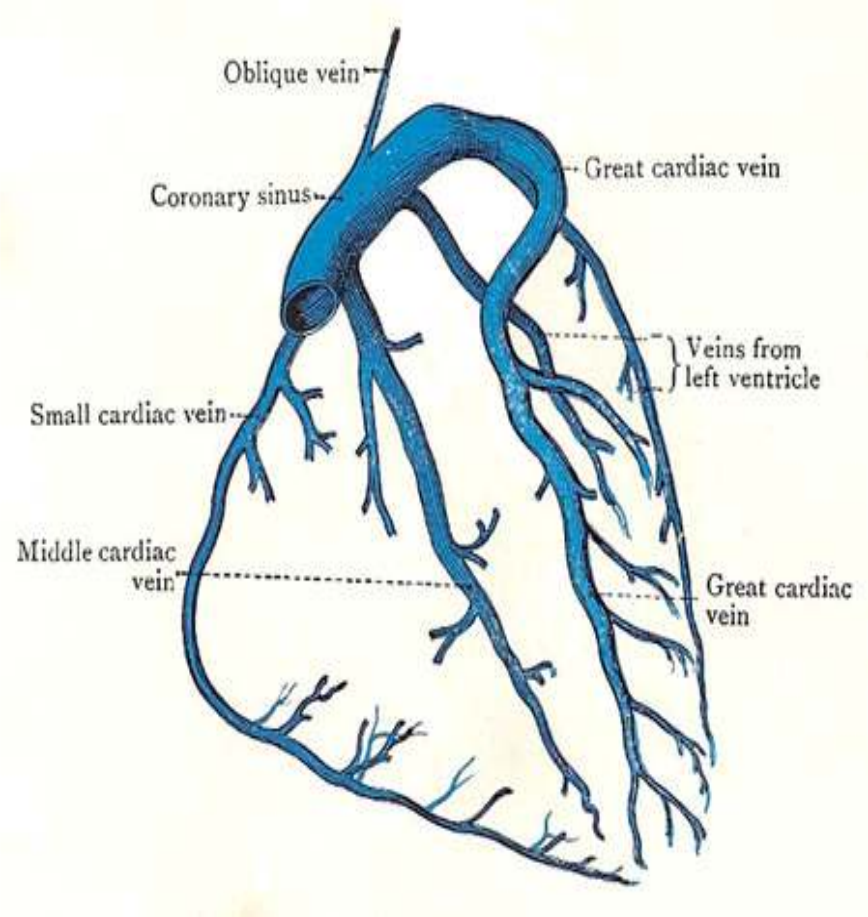
The left coronary artery soon after its origin divides into left anterior descending artery (LAD) and left circumflex artery (LCA).

The left anterior descending artery traverses the interventricular groove to anastomoses with the terminal branches of the inferior interventricular artery, which is a branch of right coronary artery, at the apex. The left circumflex artery gives branches to the posterior wall of the left ventricle and courses down to anastomose with the termination of the right coronary artery, below the coronary sinus. In 40% of the individuals it gives off a larger branch which runs over the posterior surface of the left atrium terminates in the auricular appendage of the right atrium at the sino-atrial node.

ARTERIAL SYSTEM OF THE HEART



VENOUS DRAINAGE OF THE HEART



Anastomoses of Coronary Arteries

There exists an anastomoses between the terminal portions of right and left coronary arteries in the atrrioventricular groove but these surface anastomoses are insignificant. There exists

intercoronary anastomoses at the arteriolar level, between the inter-ventricular arteries. If the intraventricular arteries meet at the apex, maximum anastomoses may be provided. If the intraventricular arteries meet slightly away from the apex above or below, the potential anastomotic area may be diminished. In 10% of the individuals, both the inferior as well as the anterior interventricular artery arises out of the left coronary artery itself, in these cases there is no anastomoses exists in this condition between the coronaries.

There exists a potential anastomoses between the coronary arteries and pericardial arteries which usually arises from the pericardiophrenic, the bronchial and the internal thoracic arteries. Very rarely, one of these may open up to replace a coronary artery

CORONARY ANASTOMOSES

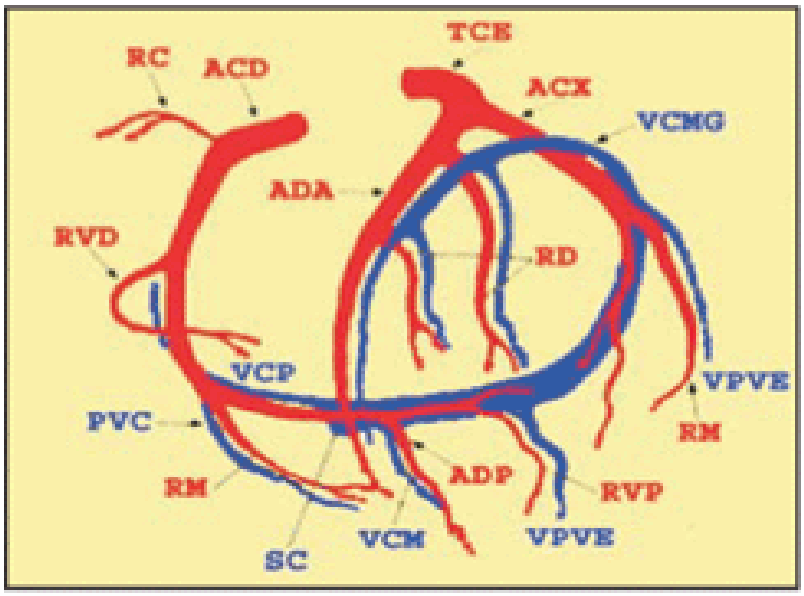


Figure 1. Coronary arteries and veins. Arterial system. The left coronary arterial tree consists of the left coronary trunk (TCE), anterior descending artery (ADA), circumflex artery (ACX), diagonal branches (RD) and marginal branches (RM). The right coronary arterial tree consists of the right coronary artery (ACD), conus branch (RC), right ventricular branch (RVD), marginal branch (RM), posterior descending artery (ADP) and posterior ventricular branch (RVP). Venous system. Consisting of coronary sinus (SC), great cardiac vein (v. cordis magna – VCMG), middle cardiac vein (v. cordis media – VCM), small cardiac vein (v. cordis parva – VCP), posterior veins of the lef ventricle (VPVE) and small cardiac veins (PVC).

Distribution of the Coronaries

Right ventricle is supplied by the right coronary artery with the exception of the upper margin of its anterior surface which is supplied by branches of anterior interventricular arteries.

Left ventricle is supplied by the left coronary artery with the exception of a narrow strip of the diaphragmatic surface which is supplied by the inferior interventricular artery. The two interventricular arteries equally supplies the interventricular septum.

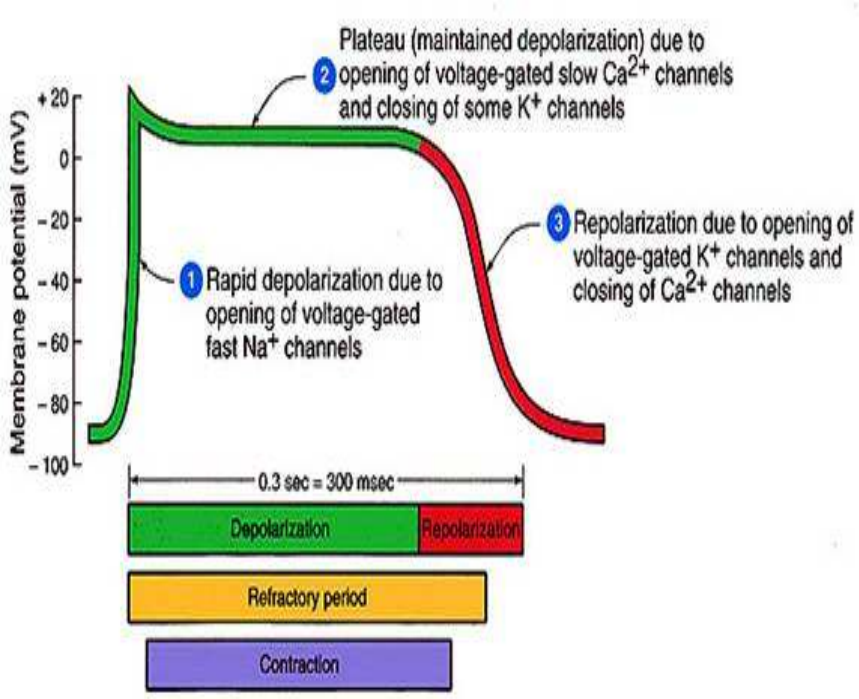
Right coronary artery supplies the anterior surface of the right atrium. Left coronary artery supplies the posterior surface and the auricular appendage of the left atrium.

SA Node: In 60% of cases**,** it is supplied by a branch of right coronary artery and in 40% of cases, it is from left coronary artery. The inferior interventricular artery supplies AV node and bundle of His, which arises in 90% of cases from the right coronary artery and in only 10% from the left coronary artery.

Dominant Arteries: Right coronary is dominant in 67% of the cases, in 15% of cases, left coronary artery is dominant and in 18% of cases, there is a balanced between these two.

CARDIAC PHYSIOLOGY

Cardiac action potential



Physiology of Coronary Circulation

Physiologically, the right and left coronary arteries behave like end arteries, eventhough innumerable inter coronary anastomoses are present in most of the normal hearts.

Normal Coronary Blood Flow

Coronary blood flow in human beings during resting state is approximately about 225 ml/ min or 0.7-0.8 ml/g of cardiac muscle or 4.5% of the total cardiac output. This may increase upto 4 to 5 fold during exercise.

Coronary Blood Flow changes during cardiac cycle

Because of cardiac muscle contraction, blood flow to the heart is decreased during systole and increased during diastole. Blood supply to the left ventricle is affected more than that of the right ventricle because of its increased thickness.

During cardiac muscle contraction, the subendocardial blood vessels are compressed more than the epicardial vessels because of increased myocardial pressures. The subendocardial vessels are usually larger compared to the nutrient arteries in the middle and external layers of the heart, which may cause a proportionate increase in the blood flow during cardiac diastole. Hence, subendocardial portions of the heart receive most of its blood supply during diastole.

Regulation of Coronary Blood Flow:

Coronary blood flow can be regulated as follows:

1. Local myocardial metabolism
2. Nervous control

i) **Local metabolism of myocardium**: As the force of contraction increases, the rate of coronary blood flow also increases. As the activity decreases, coronary blood flow also decreases. This is dependent on the following factors:

a) **Oxygen Demand**:

As oxygen extraction is near complete in resting state only, increase in oxygen demand has to be met with by increasing the blood flow. This is achieved probably by the following mechanisms:

1. **Vasodilator Theory**: Anoxia will liberate many vasodilator materials from myocardial cells which increase the blood flow:

i) Adenosine from the ATP

ii) Potassium ion

iii) Hydrogen ion

iv) Carbon dioxide

v) Bradykinin and possibly

vi) Prostaglandins

2. **Arterial Smooth Muscle Relaxation Theory**: Decrease in oxygen supply leads to anoxia of coronary arterial smooth muscle cells, which loses their tone thus getting the artery dilated. Factors that determine the oxygen consumption are:

   a) Greater the work, the greater the oxygen consumption, within the physiological limitations.

   b) Oxygen consumption is proportionate to peak myocardial muscle tension.

   i. Increased arterial pressure, increases the work load and hence tension.

   ii. Dilatation of the heart increases the tension development in myocardium to pump the blood according to Laplace law, which states that tension required to generate a given pressure increases in proportion to the diameter of the heart.

   c) Other factors which increases the oxygen consumption like stimulation of the heart by epinephrine and norepinephrine, thyroxine, digitalis, calcium ions, increased temperature of heart, will increase the oxygen consumption.

   d) Reactive hyperemia: Anoxia brings about increase flow

because of coronary dilatation after a brief period of coronary occlusion.

iii) **Nervous Control**

a) Indirect: Sympathetics increases the heart rate and contractility, through the local metabolic mechanisms, and hence increases the coronary flow. Parasympathetics decrease the heart rate and depresses the myocardium and hence brings about coronary constriction.

b) Direct Effect:

Parasympathetics: As the vagal supply to ventricles is negligible, except for slight dilatation which may occur, there is no effect of its stimulation.

Sympathomimetics: Epinephrine and norepinephrine through their receptors in coronary vessels usually bring about vasoconstriction or no change. When alpha effect dominates, severe constriction occurs which may bring about anginal attack.

EPIDEMIOLOGY OF CORONARY HEART DISEASE[85]

Coronary Artery Disease (CAD) is the major cause of morbidity and mortality in the age group of 45 years or more all over the world including India. Wide variations have been seen in the prevalence rate of CAD in various geographical zones. Death rates from CAD seems to be higher in Finland and US.

In the US, among those over 30 years of age it has been estimated that 213 per 100,000 individuals are said to have ischaemic heart disease.

Accurate data about the prevalence of CAD in India are not available. Various surveys are carried out in recent years, in various geographical locations and in small population groups which use different protocols. It has been estimated that the prevalence rate may be about 5% in urban population and a much lower prevalence has been seen in the rural setting.

The pattern of CAD in India has been reported to be:

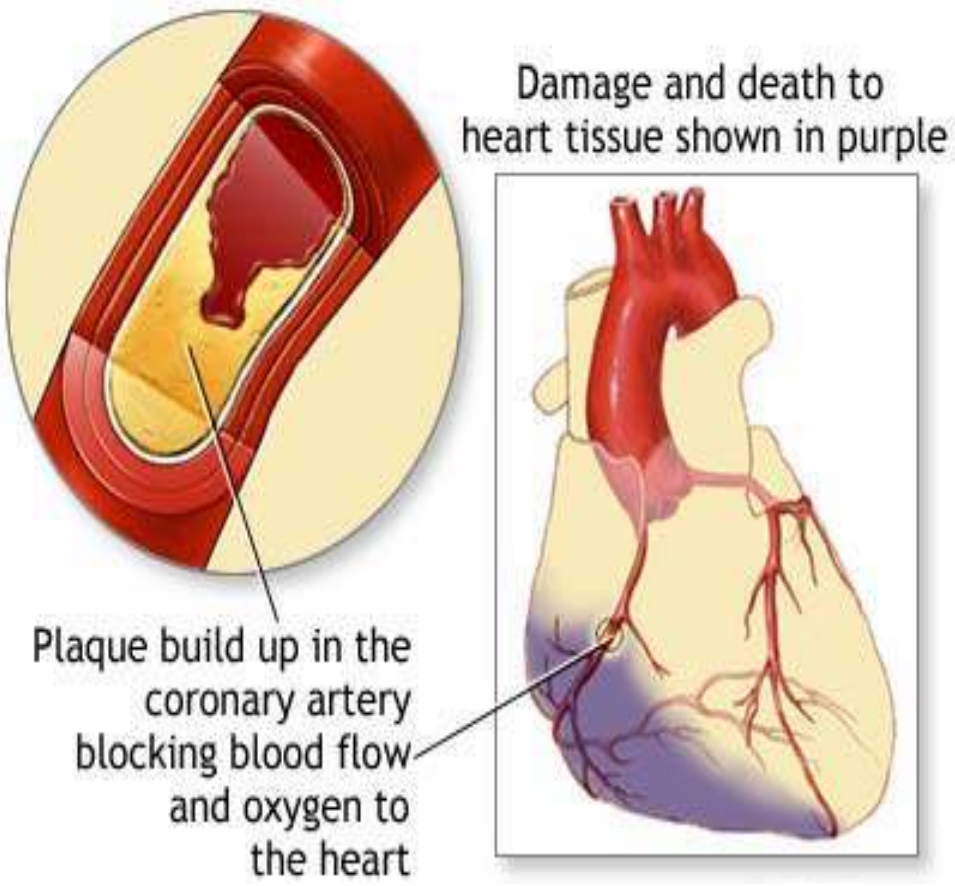
a) Males are affected more than females.
b) Hypertension and diabetes accounts for about 40% of all cases.
c) Heavy smoking is the precipitating factor in a large number

of cases.

d) High fat & energy rich diet, sedentary life style are other contributing factors.

It has been believed that the prevalence of CAD in India has been increasing over the last three decades and the younger persons are prone to the prone to develop CAD since 1970 because of the increasing risk factors. There has been approximately 30% reduction in mortality due to CAD.

MYOCARDIAL INFARCTION



PATHOPHYSIOLOGY OF ACUTE MYOCARDIAL INFARCTION[85]

Myocardial infarction occurs when there is an abrupt reduction in coronary blood flow usually occurring as a result of thrombotic occlusion of the coronary artery already been narrowed by the formation of atherosclerotic plaques which fissures, ruptures or ulcerates and under favourable conditions thrombogenesis may take place. A mural thrombus forms at the site of plaque rupture and leads to coronary artery occlusion. An initial platelet monolayer forms at the site of the plaque rupture, a series of agonists (collagen, ADP, epinephrine, serotonin)released may promote platelet activation. Following stimulation by agonists, thromboxane A_2 is produced and released. This may further activate platelets and hence aggravating thrombogenesis.

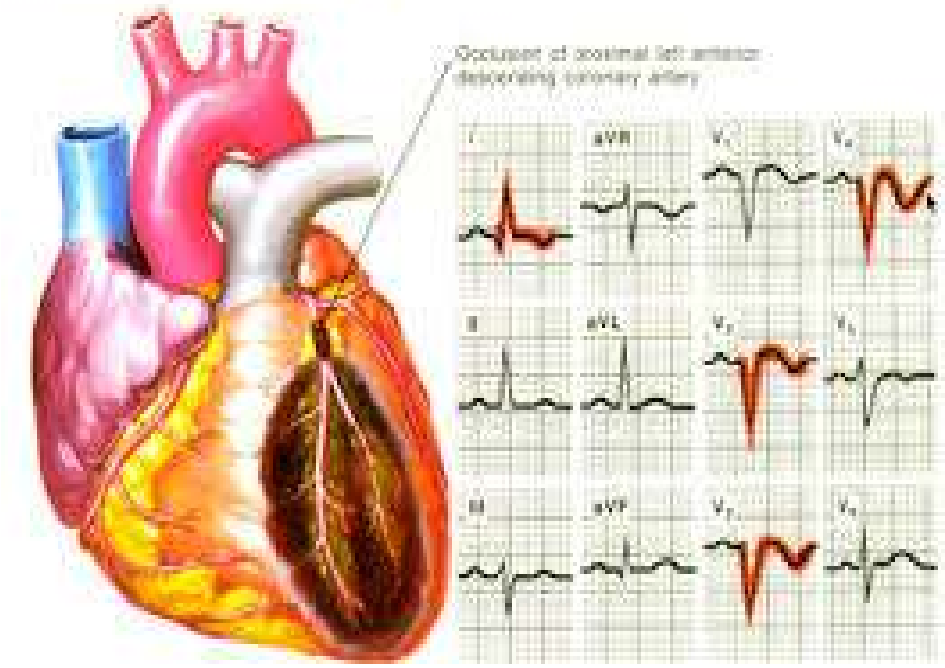
In addition to the production of thromboxane A_2, platelet activation by agonists produce a definitive conformational change in the glycoprotein IIb- IIIa receptors. Once it is converted to its functional state, glycoprotein IIb- IIIa receptor develops a high affinity for the arginine – glycine – aspartic acid sequence on the alpha chain of the fibrinogen and also for a dodecapeptide sequence on the gamma chain of the fibrinogen. Fibrinogen is a

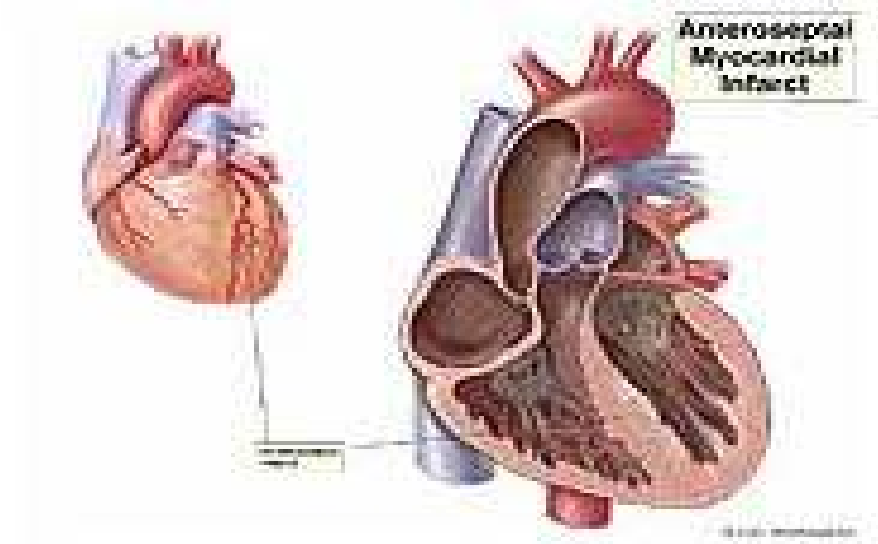
multivalent molecule and it has the ability to bind to two different platelets simultaneously, leading to cross linking of platelets and aggregation.

At the site of the plaque rupture, tissue factor is released from the damaged endothelial cells, which on exposure gets activated, followed by the activation of coagulation cascade occurs. Through extrinsic and intrinsic pathways, Factor VII and X are activated, ultimately leading to the conversion of prothrombin to thrombin, Thrombin, then converts fibrinogen to fibrin. Finally, crosslinkage of fibrin and formation of fibrin clot occurs. The coronary artery eventually gets occluded by a thrombus made up of platelet aggregates and fibrin strands.

Ultimately the extent of myocardial damage produced by coronary occlusion depends upon the blood vessel affected, the territory it supplied, whether or not the vessel becomes totally occluded, presence of native factors which can produce early spontaneous lysis of the occlusive thrombus, the extent of collateral vasculature formation and the oxygen demand of the myocardium whose blood supply has been abruptly limited.

Anterior MI


Occlusion at proximal left anterior descending coronary artery
I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6


Anteroseptal Myocardial Infarct

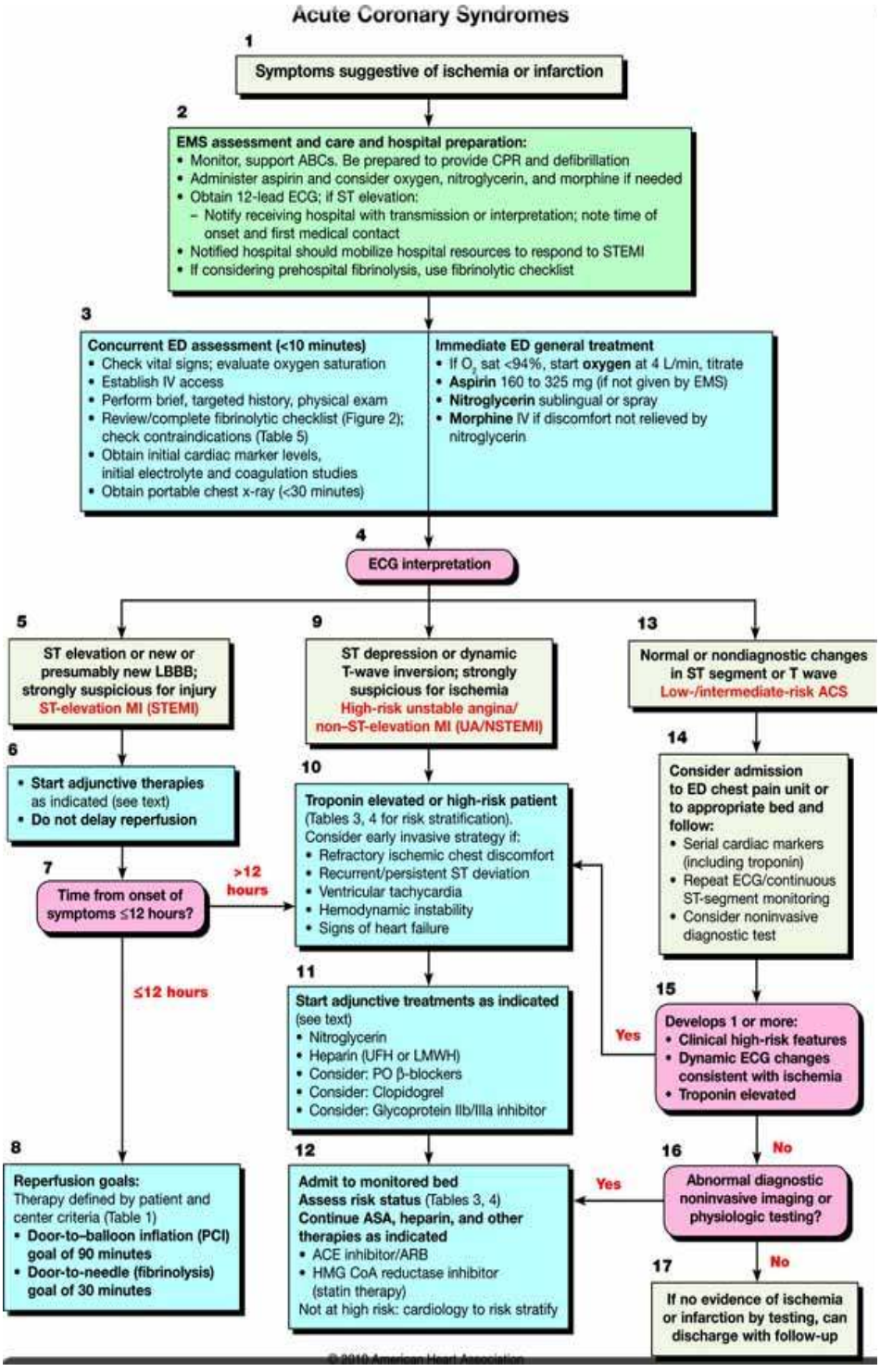


Chart 2 : ACLS 2010 GUIDELINES FOR THE MANAGEMENT OF ACUTE CORONARY SYNDROMES

CLINICAL FEATURES OF ACUTE MYOCARDIAL INFARCTION[85]

Acute myocardial infarction usually presents itself as a sudden catastrophic accident and its clinical picture may be variable and unpredictable.

AMI may present with one or combination of the following:

1. Chest pain
2. Shock
3. Pulmonary edema or other evidence of LV failure.
4. Congestive cardiac failure
5. Some cases may present with combination of any of the above.

1. Pain in the Chest:

In 80-85% of cases this is the most common presenting complaint, characterized by a deep visceral pain usually involving the central portion of the chest and epigastrium, felt as tightness, heaviness or constriction in the chest. In 25% of cases, radiation of pain to the arms especially left arm and ulnar aspect of the forearm. It is commonly associated with weakness, forehead sweating, nausea, giddiness and anxiety. It may be precipitated by exertion

and emotional outbursts and not relieved with rest and compels the patient to move about to find a comfortable position.

2. Breathlessness

The next common symptom is breathlessness which may be sudden in onset, usually grade 2 or 3, may be exertional. It is usually seen in diabetics, elderly and those having complications like cardiogenic shock and pulmonary edema in whom it presents as ‘ silent myocardial infarction’.

3. Sudden loss of consciousness, a sense of profound weakness or unexplained hypotension associated with giddiness, syncope, confusional state and/ or convulsions may be the presenting complaint.

4. Choking sensation felt in the neck may be the only presenting symptom.

Very few patients present with breathlessness of gradual onset, paroxysmal nocturnal dyspnea, abdominal pain with oliguria and bilateral pitting pedal edema, swelling of lower limbs, a picture characteristic of CCF.

In rare cases, endocardial thrombosis may occur where the infarct may go unrecognized resulting in systemic embolism.

Physical Signs : Patient may arrive at the emergency room with the hand placed over their precordium where there is maximum intensity of pain (Levine sign).

This may often be associated with perspiration and coolness of extremities, cyanosis may occur when the patient has severe pulmonary edema or shock.

Pulse

Bradycardia, normal sinus rhythm, tachycardia with or without irregularities, depends upon the presence or absence of arrhythmias and the type of arrhythmia.

Blood Pressure

Usually there may be an increase in BP initially because of pain, anxiety or the unfamiliarity of the environment, which becomes normal within 3 or 4 days. Decrease in the blood pressure may occur as a result of cardiogenic shock or due to 'Bezold-Jarisch reflex', which may be caused by increase vagal tone that occurs in inferior wall infarction.

Neck veins:

Collapse of neck veins may be seen when patient is in shock, cannon waves can be seen in case of complete heart block.

Precordium:

There is a difficulty in palpating the apical impulse. In certain patients with anterior wall infarction, an abnormal systolic pulsation may be observed in the periapical area during the first few days of illness, which usually resolves later, representing a transient, palpable systolic bulging of the infarcted ventricle. Muffling of heart sounds, atrial (S4), ventricular (S3) gallop sounds and paradoxical splitting of the 2^{nd} sound. A apical systolic murmur due to mitral regurgitation may be heard transiently secondary to papillary muscle dysfunction in case of acute infarction. If the infarction is transmural, pericardial friction rub may be heard.

Temperature fluctuaions in the range of 37 to 38ºC are common during the first 3 to 4 days due because of myocardial necrosis.

Respiratory System:

Tachypnea may be present and fine crepitations may be heard at the base initially, then all over the lung fields depending upon the amount of pulmonary congestion.

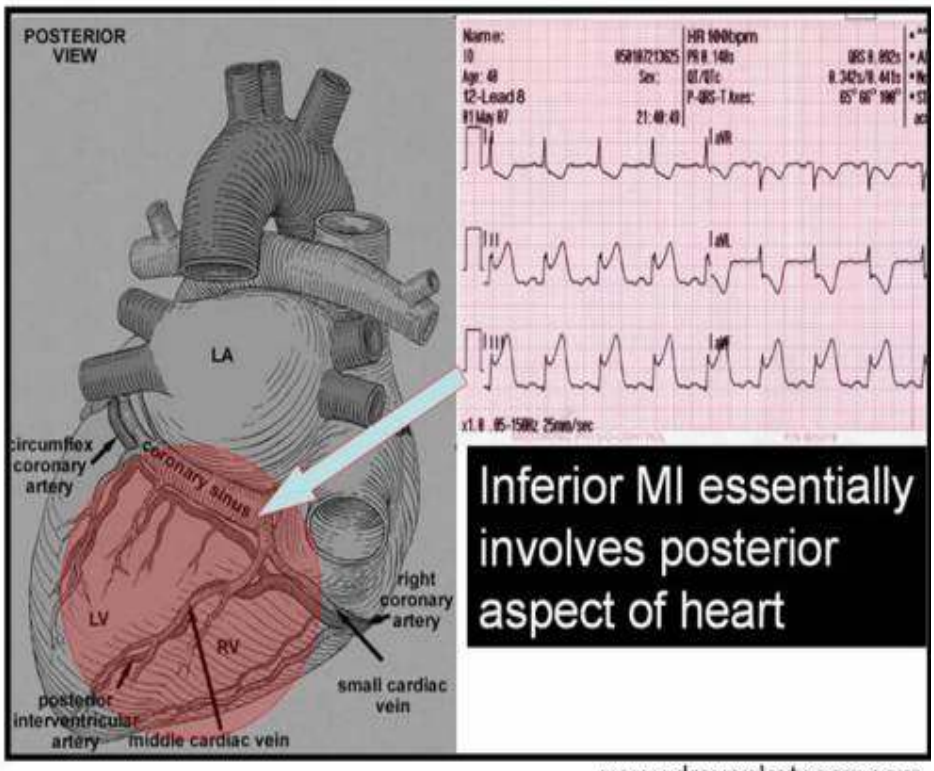
Gastrointestinal System:

Tender hepatomegaly may be present if there is congestive cardiac failure.

Central Nervous System:

Anxiety, restlessness, stupor, coma, focal neurological deficit may be present. When there is hypotension, fall in blood pressure and/ or thromboembolic phenomenon.

INFERIOR WALL MI



MAGNESIUM HOMEOSTASIS

Magnesium (Mg) is the fourth cation to be seen in abundance in the body. It is the second most intracellular cation seen abundantly, next to potassium[5]. In adults, the normal magnesium content of the body is about approximately 2000 milli equivalents (meq) or 24 grams or 1000 mmol. Magnesium has an uneven distribution, with highest concentration in tissues with the high metabolic activity such as brain, heart and kidney. Approximately 60% of the total body magnesium is present in the bone. Out of which, one third has been shown to be exchangeable. This fraction of exchangeable magnesium serves as a reservoir for the maintenance of normal extracellular magnesium concentration.

Extracellular magnesium constitutes only about 1% of total body magnesium content. The remaining of the body magnesium is intracellular. The normal serum magnesium concentration is about 1.8 – 2.9 mgs/dl[7]approximately. On an average, 70-75% of magnesium in plasma can be ultra filtered. The non-filterable portion remains bound to plasma proteins, especially albumin.

The intracellular magnesium concentration in various tissues vary widely, but is of the order of 1-3 mmols/ l[8]. In general, the metabolic activity of the cell is proportionate to its magnesium

content. Decrease in magnesium concentration in the serum, usually implies magnesium deficiency. However, the serum magnesium level may not reflect intracellular magnesium. Intracellular magnesium depletion may be seen despite a normal serum magnesium concentration[9]. Estimation of intracellular magnesium concentration is not usually done because of the difficulty in performing tissue and cellular assays. Hence, determination of the serum magnesium concentration is the method widely used in clinical practice to identify magnesium deficiency.

Myocardial Magnesium

Normal body content of magnesium is about 21 to 28 gms or about 3 mg/kg of fat free tissue. 60% of which is present in the bone. Magnesium is concentrated in significant amounts in cardiac muscle which is about 17.4- 19.8 meq/l. Magnesium concentration in ventricles are found to be higher than that in the atria. No significant difference has been found between magnesium concentration in the right and left ventricles or inter-ventricular septum (Burch et al, 1977)[1]. Magnesium has been found to be involved in various mechanisms essential for the contraction of heart muscle such as ATP hydrolysis by myofibrils, sineresis and super-precipitation of actinomycin gels, and binding and release of calcium ions by sacrotubule systems. It also stimulates oxidative

phosphorylation in mitochondria of the cardiac muscle, and influences sodium potassium ATPase of heart membranes, and activates adenylyl cyclase and also phosphorylase kinase in the heart. Magnesium also influences muscle tone and conducting system of the heart, though sensitivity to magnesium is lesser in myocardium compared to the nervous tissue (Wecker et al, 1968)[5].

Renal handling of Magnesium

Kidneys play a significant role in the regulation of magnesium homeostasis. Each day, about 8 meq of magnesium is excreted into the urine. In magnesium depleted states, there is avid retention of magnesium by the kidneys and only negligible quantities of magnesium is lost in the urine per 24 hours period. If the dietary magnesium intake is high or administered parenterally, the filtered load exceeds the normal plasma concentration resulting in rapid excretion of excess magnesium[10]. In humans, the renal handling of magnesium is mainly a filtration – reabsorption process. The proximal tubule and thick ascending limb of Henle are found to be the important sites of magnesium retention.

In the proximal convoluted tubule, there is a passive reabsorption of about 20 to 30 % filtered magnesium. Magnesium reabsorption follows change in salt and water reabsorption. About

65% of filtered magnesium is reabsorbed in the thick ascending limb of Henle by an active transport process.

Aldosterone promotes the renal excretion of magnesium, whereas parathormone inhibits its excretion. Parathyroid hormone regulates both calcium and magnesium excretion and metabolism. Parathormone reaction is reduced by increase in blood magnesium concentration and vice versa[5]. In patients with either primary hyperparathyrodism or hypoparathyrodism, the serum magnesium concentration was found to be normal indicating that PTH plays only a minimal role in the regulation of magnesium homeostasis[10].

Intestinal absorption of magnesium

The intestinal absorption of magnesium is inversely proportional to the intake of magnesium[11]. The recommended minimum requirement per day is 300-500 mg. The estimated magnesium intake per day ranges from 150-350 mg/day. In general, about 30-50% of ingested magnesium is absorbed[12]. Groundnuts, cereals pulses and meat are rich in magnesium.

Absorption of magnesium takes place throughout the GIT, the ileum and jejunum are the sites of maximum absorption. Magnesium is most efficiently absorbed in the alkaline

environment of small intestine in chloride form[13]. The higher fractional absorption at low dietary magnesium intake is because of the existence of an unsuitable passive transport system for magnesium absorption in addition to the hormone controlled magnesium transport.

Hormone controlled intestinal magnesium transport constitutes the major transport mechanism of magnesium. Vitamin-D and its breakdown products, 25 hydroxy and 1,25 dihydroxy compounds enhance magnesium absorption by the intestine[14]. Bioavailability of magnesium may also contribute to intestinal absorption of magnesium. The presence of certain substances such as free fatty acids, phytates, oxalates, phosphate and fiber in excess in the ingested food may impair absorption by binding to it [5].

Intracellular Magnesium

Magnesium is intracellularly compartmentalized and is bound to proteins and other negatively charged molecules. Magnesium is present in the nucleus, mitochondria and endoplasmic reticulum as well as cytoplasm in significant quantities[15]. 80% of magnesium in the cytoplasm is present as a complex with adenosine triphosphate (ATP)[00]. The free ionized magnesium (mg^{2+}) concentration is about 0.1 mmol/l to 1 mmol/l. It is about 0.55 to 5% of the total cellular magnesium. The

intracellular magnesium concentration seems to be maintained relatively constant.

Various studies about magnesium transport have suggested that the rate at which magnesium exchange occurs in organs such as heart, liver and kidneys far exceeds that in the skeletal muscle, red cells and brain. There is an increase in intracellular magnesium content in rapidly proliferating normal cells indicating a possible relationship between the metabolic activity of a cell and relative rates of transport of magnesium into and out of cells. Magnesium absorbed is excreted by the kidneys and the amount excreted in the stool is less than 1.4% of amount given.

The kidneys filter about 2.5gm of magnesium approximately per day and retains 95% in normal conditions, excreting approximately 100 mg/day of magnesium in the urine for maintaining homeostasis. In magnesium depleted states, kidneys retain magnesium and hence its excretion can be reduced to less than 12 mg/ day.

Aldosterone promotes the renal excretion of magnesium, whereas parathormone inhibits its excretion. Parathormone also regulates calcium and magnesium excretion and metabolism. An

increase in the serum magnesium concentration inhibits the action of parathormone and vice versa[5].

PHYSIOLOGICAL ROLE OF MAGNESIUM

Magnesium plays a principal role in various enzymatic processes in the body[5]. It is essential for the formation of various substrates and it has a direct role in the activation of enzymes such as phosphofructokinase, creatine kinase, adenylate cyclase and sodium-potassium ATPase involved in various metabolic processes. The effect of magnesium on certain metabolic processes such as oxidative phosphorylation, glycolysis, protein biosynthesis, nucleotide metabolism implies the significance of magnesium in cellular metabolism.

Magnesium activates the sodium-potassium ATPase, thereby maintaining low extracellular and high intracellular potassium levels against large concentration gradients. It has been shown that hypomagnesemia may cause impairment of the ability of the cells to maintain the potassium gradient which may lead to intracellular potassium depletion.

The compromise in the cell membrane cation pump results in loss of intracellular potassium associated with the accumulation of intracellular sodium. This is similar to the effect which occurs during therapy with digitalis and this is how hypomagnesemia

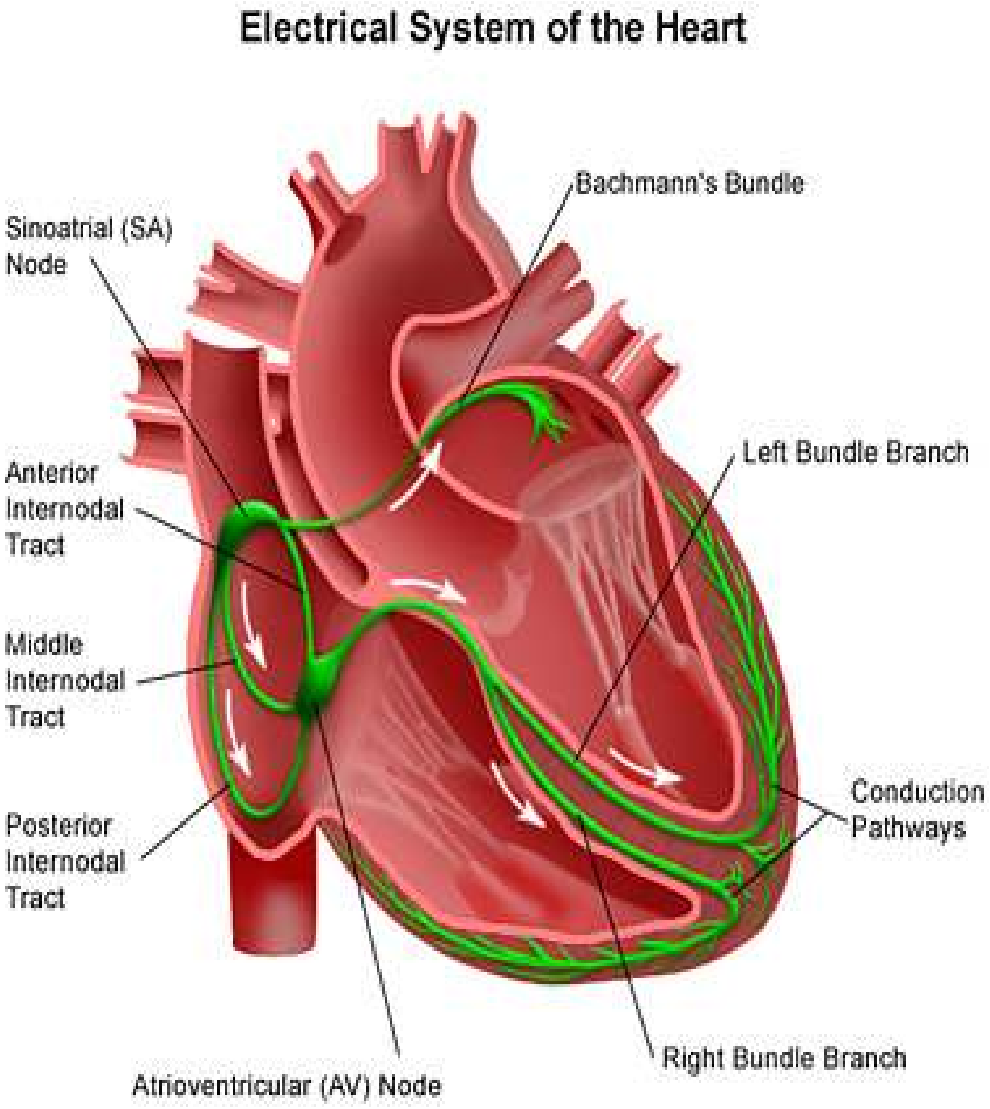
causes digitalis toxicity.

Influence of Magnesium on the tone of the blood vessels

Magnesium is known to be a naturally available calcium antagonist[41]. It decreases the output of calcium out of and into the calcium stores, and protecting the tissues against the excess amounts of cacium occurring during states of reduced blood supply to the heart. Magnesium decreases the resistance of both systemic and pulmonary blood vessels resulting in drop in blood pressure and an rise in cardiac index[42]. Increase in extracellular magnesium level decreases the arteriolar tone in various arteries[43] and augments the vasodilatory action of certain substances occuring in vivo and also administered pharmacological (isoproterenol and nitroprusside) vasodilators[43]. Because of its mild reducing effect on systolic blood pressure, magnesium may reduce the resistance of the systemic vessels unloading the ischemic ventricles. Kugiyama et al[44] showed that in patients with variant angina, angina pectoris stimulated by exercise may abolished by administration magnesium parenterally, perhaps due to suppression of coronary artery spasm thereby improving regional myocardial blood flow. Altura and Altura[45] in an experimental model using smooth musce of the blood vessels found that hypomagnesemia could be responsible for the increase in arterial pressure occuring in toxic

states of pregnancy. This effect may be due to potentiation of increased intracellular calcium activity. The response to intravenous magnesium therapy eclampsia may be due to its calcium inhibitory effect.

Influence of magnesium on rhythm of the heart



Hypomagnesemia may be found usually alongwith hypokalemia, sodium excess and increased excitation of the cells. Magnesium has the following effects such as prolongation of the actual and corrected recovery time of the sinus node, prolongation of the function of the atrioventricular node, relative and effective periods of refractoriness, increase in the length of the QRS complex during pacing of the ventricle and increase in the interval between atrium and His bundle resulting in atrioventricular nodal Wenckebach conduction[46]. In 1935, Zwillinger[31] first observed that magnesium has an inhibitory effect on the initiation of arrhythmias when it was used to convert paroxysmal tachycardia to sinus rhythm. Thereafter, it was successfully used in the treatment of ventricular tachycardias not responding to treatment, ventricular arrhythmias caused by overdosage with digitalis , also in torsades de pointes, ventricular dysrhythmia which may be prove fatal.

Magnesium may be considered to be useful in the treatment of dysrhythmias of supraventricular origin, (e.g) atrial tachycardia of multifocal origin. It also increases the responsiveness of tachycardia of atrial origin to the drug therapy with digoxin.

According to ACC/AHA guidelines, magnesium has been

considered as the drug therapy of third choice (amiodarone as the 1st choice and lidocaine as the 2nd choice) in the ACLS protocol for CPCR of patients with shockable peri arrest rhythms.

Infuence of Magnesium on metabolism of lipids

Magnesium has an important role in regulating the metabolism of lipids eventhough its mechanism is not known completely. Magnesium serves as an adjunct of two enzymes that play an important role in the metabolism of lipids; lecithin-cholesterol acyltransferase (LACT) and lipoprotein lipase. In an experimental study with rabbit model, animals were fed with a normal diet, or a diet rich in cholesterol plus supplemention of variable quantities of magnesium. The addition of supplemental magnesium has been found to achieve a dose-dependent drop in the cholesterol content in large vessels such as aorta[48] associated with reduction in aortic lesions. On the other hand, in rats which are fed with magnesium deficient diets, adverse lipid changes occured. In a rat model, diets completely devoid of magnesium resulted in increase in the levels of total cholesterol, LDL-cholesterol and triglycerides in plasma, with a corresponding decrease in plasma levels of high density lipoproteins-cholesterol (HDL-C)[49].

Rassmussen et al[51] administered magnesium 15 mmol/ day

for a period of 3 months and noticed that there was a 27% drop in triglycerides and (VLDL-C) levels in plasma associated with decrease in levels of apoprotein B and increase in HDL-C levels in plasma.

Davis et al[50] in their 4-month clinical trial reported a remarkable increase in the ratio of HDL-C , LDL-C and VLDL-C by the administration of magnesium 18 mmol per day

Niemela et al[52] reported that in men, but not in women, the intracellular levels of magnesium in platelets are inversely proportional to th serum levels of total cholesterol , LDL-C and apolipoprotein B in the serum. They also reported that decrease in intracellular levels of magnesium in platelets may cause alterations in the cell membrane of platelets. This, in turn may influence the participation of platelets in thrombus and atheroma formation.

Magnesium as an anticoagulant/ antiplatelet

Greville and Lehmann[53] in the year 1943, observed that addition of little quantity of magnesium to freshly prepared human plasma which is unclotted I nature resulted in prolongation of the time taken for clotting . In Germany, sulphate salt of magnesium has been used widely for skeletal muscle relaxation, and it has been noted after the examination of their blood postmortem that their

blood has not clotted after the administration of sulphate salt of magnesium. Anstall et al[54] in 1959 showed that magnesium can inhibit coagulation of human blood.

Many studies have reported that magnesium by its effects as a platelet inhibiting agent can reduce the progression of thrombi in the coronary arteries and also complete block of the coronary artery after recanalization which has occurred either by itself orstimulated by the process of fibrinolysis[55,56]. Numerous studies have analysed and reported the effect of magnesium in the inhibition of aggregation of platelets in volunteers who aredevoid of the disease[55,56]. Elevated levels of magnesium in plasma may suppress clotting of blood and initiation of thrombus in the body, decrease aggregation of platelets, decrease the production of thromboxane A_2, which acts as an agonist on platelets and suppress the inward current of calcium, which may be stimulated by thrombin.

Activation of platelets is the major step in involved in acute vascular thrombosis of the vasculature, which contributes to the causation of AMI and expected adverse effect after coronary balloon angioplasty and stenting. Various studies have reported that magnesium can suppress the stimulation of platelets by suppressing the factors which stimulate platelets like thromboxane A_2 or by

inducing the production of factors which inhibit platelets like prostacyclin[57,58]. IV infusion of magnesium in volunteers who were healthy, suppressed the aggregation of platelets which was induced by ADP by about 40%. The attachment of fibrinogen or expression glycoprotein IIb-IIIa complex GMP-140 on the surface was also inhibited by about 30%[58]. Hence, magnesium at therapeutic concentrations, intensively suppresses the function of the platelets.

Gawaz[59] et al demonstrated that there was an increase in the surface expression of P-selectin on the surface of platelets which may be expressed on its own or induced by ADP, also a rise in the adhesion of platelet and leucocyte in patients who were symptomatic with coronary artery disease compared to the controls who were healthy. However, there was a significant reduction in both expression of P-selectin on platelet surface and adhesion of platelet leukocyte after administration of intravenous magnesium.

Influence of magnesium on endothelial function:

In an animal model, Pearson et al[60] showed that magnesium deficiency caused selective impairment of the discharge of NO from endothelium of the coronary vassculature. NO is a nitrovasodilator produced in vivo and it may suppress adhesion and

aggregation of platelets . Hence, it has been considered that magnesium deficiency may cause constriction of the vasculature and eventually thrombosis of the coronary arteries.

Influence of Magnesium on the extent of infarction

Magnesium deficiency can cause vasoconstriction of the coronary and systemic vasculature and may increase systemic vascular resistance. Magnesium administration can suppress the worsening of ischemia if started as early as possible during the initial stages of occurrence of ischaemia. This, in turn can decrease the occurrence of arrhythmias due to elevated levels of catecholamines.[61]

In animal studies, it has been shown that low magnesium concentrations may stimulate the causation of myocardial necrosis induced by catecholamines[61]. Hypomagnesemia can also affect the healing process, healing of the blood vessels and infarcted tissues of the heart and can also cause incomplete angiogenesis[62]. These changes can contribute to potentially lead to inadequacy in the formation of collateral circulation and extension of the infarct. Magnesium decreases the susceptibility of the myocardium to free radicals released due to superoxidation, injury due to reperfusion and stunning of myocardium.

HYPOMAGNESEMIA

Magnesium has been called occasionally the "forgotten cation". The changing trends in the diagnosis of electrolyte disturbances has now brought the cation into the limelight. Hypomagnesemia is now well recognized due to increased clinical awareness, and the greater frequency of assessment of magnesium status. Approximately 10% of the admissions at major health care facilities are found to be hypomagnesemic. This may escalate to as much as 65% in severely ill patients. Hypomagnesemia commonly occurs due to loss of magnesium from either the gastrointestinal tract or the kidneys.

ETIOLOGY:

Due to decreased magnesium intake :

- Dependence on alcohol
- Parenteral administration of nutrition
- Starvation

Due to redistribution of magnesium

- Syndrome of refeeding

- diabetic ketoacidosis- treatment
- Acute pancreatitis
- alcohol withdrawal
- Hungry bone disease

Gastrointestinal magnesium loss :

- Nasogastric suction
- Diarrhea
- Vomiting
- Gastrointestinal fistulas
- Secondary to hypocalcemia

Renal magnesium loss

- Gitelman syndrome
- Familial hypomagnesemia with hypercalciuria and nephrocalcinosis (FHHNC)
- Classic Bartter syndrome (Type III Bartter syndrome)
- ADHH
- IRH with normocalcemia
- IDH with hypocalciuria
- Hypomagnesemia secondary to hypocalcemia

Drugs :

- Antimicrobials - Amphotericin B, aminoglycosides, pentamidine, capreomycin, viomycin, and foscarnet
- Diuretics - Loop diuretics, osmotic diuretics, and thiazides on prolonged use
- Cisplatin
- Tacrolimus and cyclosporine
- Omeprazole, pantoprazole

Other causes :

- Ethanol
- Hypercalcemia
- Chronic metabolic acidosis
- Primary hyperaldosteronism
- Acute tubular necrosis – recovery phase

Hypomagnesemia may be seen in about 28% of patients with acute hemorrhagic pancreatitis. The low serum magnesium concentration may predispose to pancreatitis.

Renal wasting of magnesium

Loss of magnesium in the urine is underlying mechanism of

hypomagnesemia in many patients. Reabsorption of magnesium from the proximal renal tubule is in parellel with the fluid flow through the renal tubule and retention of sodium[63]. Hence, intravenous fluid administration for prolonged period of time, especially with sodium containing fluid may result in hypomagnesemia. Similarly in diabetic mellitus, osmotic diuresis may result in loss of magnesium in the urine.

Hypercalcemia has been known to reduce absorption of magnesium in the proximal tubule and loop of Henle and probably is the mechanism of renal magnesium loss or the tendency towards hypomagnesemia in most of the conditions of hypercalcemia [64].

Gastrointestinal Disorder

The magnesium level of gastrointestinal tract is about 1 meq/l on an average. Hence, emesis and suctioning through nasogastric tube may contribute to magnesium loss. The magnesium lost through fistulous drainage and diarrheal fluid are very high (up to 15 meq/l) and hence, magnesium deficiency is much commonly seen in diarrheal diseases of acute or chronic origin , ulcerative colitis, Crohn's disease, fistulous lesions of the intestinal and biliary systems [65].

Syndromes of altered absorption occurring due to non-

tropical disease, injury due to exposure to radiotherapy given in diseases like carcinoma related to the head of the pancreas and cervical carcinoma can lead to magnesium depletion probably because of the damage to the mucosa of the intestines[66]. Fatty stools can cause formation of non-absorbable magnesium lipid salts resulting in magnesium malabsorption. Small bowel surgeries used for the treatment of certain bowel disorders may also result in hypomagnesemia.

The commonest cause of magnesium loss is liberal use of diuretics[67]. Diuretic drugs acting on the proximal renal tubules, such as mannitol and carbonic anhydrase inhibitors,can increase excretion of magnesium. Diuretics such as frusemide and ethacrynic acid act at the thick ascending Henle's loop may result in magnesium deficiency.

Aminoglycoside therapy using capreomycin, gentamycin and recently with tobramycin, amikacin has been shown to produce renal magnesium wasting. Use of certain antifungal agents may also lead to magnesium loss through the kidneys. Cisplastin, a chemo-therapeutic drug used in the therapy for neoplasms of epithelial tissues may lead to renal magnesium wasting .

Endocrine and Metabolic Disorders

Some endocrine and metabolic diseases are also associated with magnesium loss, which commonly occurs through renal magnesium loss. Diabetes mellitus is the commonest disorder of metabolism found with hypomagnesemia[68]. The magnesium levels in the serum is inversely proportional to the glucose levels in the serum and also the extent of glycosuria. The process of magnesium wasting in diabetes mellitus may probably be due to glycosuria (osmotic diuresis)[69]. And also, insulin may cause shifting of magnesium into the cell resulting in magnesium deficiency.

Hypomagnesemia can also be seen in various other endocrine abnormalities. Hypophosphatemia has been shown to produce loss of magnesium in urine resulting in hypomagnesemia. Hence, hypophosphatemia acts as a contributing factor in the causation of magnesium deficiency.

Other conditions such as hyperthyroidism, thyrotoxicosis may also cause urinary magnesium wasting resulting in hypomagnesemia. In primary hyperaldosteronism, hypomagnesemia may be due to plasma volume expansion followed by renal magnesium wasting.

Miscellaneous Causes

Magnesium wasting fron the entire body can occur via uncommon modes of excretion. Abnormal sweating may result in significant extent of magnesium losses.

Pancreatitis may also cause magnesium deficiency but the basis of loss of magnesium seems to be unknown. It has been postulated that abnormal deposition of magnesium and fat complexes in the soft tissues may be one of the causes. Magnesium redistribution into intracellular compartment or the bone is the most common reason for reduction in magnesium levels in the serum.

MAGNESIUM AND ACUTE MYOCARDIAL INFARCTION

Epidemiological studies have proposed that the incidence of myocardial infarction and of sudden death is high in areas of soft water intake[2]. Myocardial magnesium content has been found to be decreased in patients whose death was attributed to acute myocardial infarction[16]. However, it is not known whether the decrease in magnesium content predisposes to myocardial infarction or is result of it. Myocardial magnesium gets exchanged rapidly with plasma magnesium. A number of clinical trials have shown a drop in the concentration of magnesium in the serum within the first 24 to 48 hours after acute myocardial infarction[17].

Various studies have shown that there is a fall in magnesium concentration in the infarcted myocardium but the serum magnesium values in first 24 hours, following acute myocardial infarction has been variable. Some studies showed no significant change of serum magnesium. Hence, it has been proposed that there exists a reverse relationship between serum magnesium level in the serum and coagulability of the blood, serum cholesterol levels, following acute myocardial infarction.

Injury to the myocardium was confirmed by histological examination of cardiac tissue. A marked elevation in the loss of

magnesium in the urine was seen during the first two hours. The level decreased soon after that but was still maintained above the control level. In the infarcted myocardium, magnesium content decreased significantly.

Various authors have demonstrated a reduction in serum magnesium level following AMI. Abraham S et al[17] (1980) studied serum magnesium levels in forty two patients with acute MI, nine patients with coronary insufficiency and fourteen patients with non-cardiac chest pain. In patients with acute MI and those with acute coronary insufficiency, a reduction of magnesium levels in the serum compared was seen, whereas no difference has been noted in patients with non-cardiac chest pain. A marked reduction of magnesium levels in the serum was noted during the first five days and normal levels were recorded by the 12^{th} day.

Singh A et al[75] (1976) measured magnesium levels in the serum of twenty patients diagnosed of having acute MI on the first 7^{th} and 12^{th} day of admission. Significant reduction in serum magnesium level has been recorded in all the cases on the first day.

Babel S.Bhatnagar, HNS Bhatnagar[18] (1983) studied the prognostic significance of serum magnesium levels in patients with acute MI. Twenty five patients diagnosed to have acute MI were taken as the study population. Serum levels were reported to be

significantly reduced on the first day and it gradually increased and attained the normal value by the twenty first day.

Certain studies have reported that magnesium deficiency resulted in decrease in intracellular magnesium and potassium and increase in intracellular calcium and sodium[19]. In patients with coronary artery disease there was a decrease in the exchangeable magnesium and in patients diagnosed of having acute myocardial infarction, there was retention of abnormally high quantities of magnesium during magnesium tolerance test. Skeletal muscle magnesium content was found to be reduced, suggesting the presence of hypomagnesemia[20].

Magnesium deficiency predisposes to vascular spasms especially coronary artery spasm and also potentiates the contractile response to vasopressors such as angiotensin II and norepinephrine. Magnesium deficiency may worsen angina and may predispose to acute myocardial infarction. Magnesium therapy in acute MI has been shown to reduce infarction size, the incidence of cardiac arrhythmias and also the mortality rate[21,22,23].

ANTEROSEPTAL MI

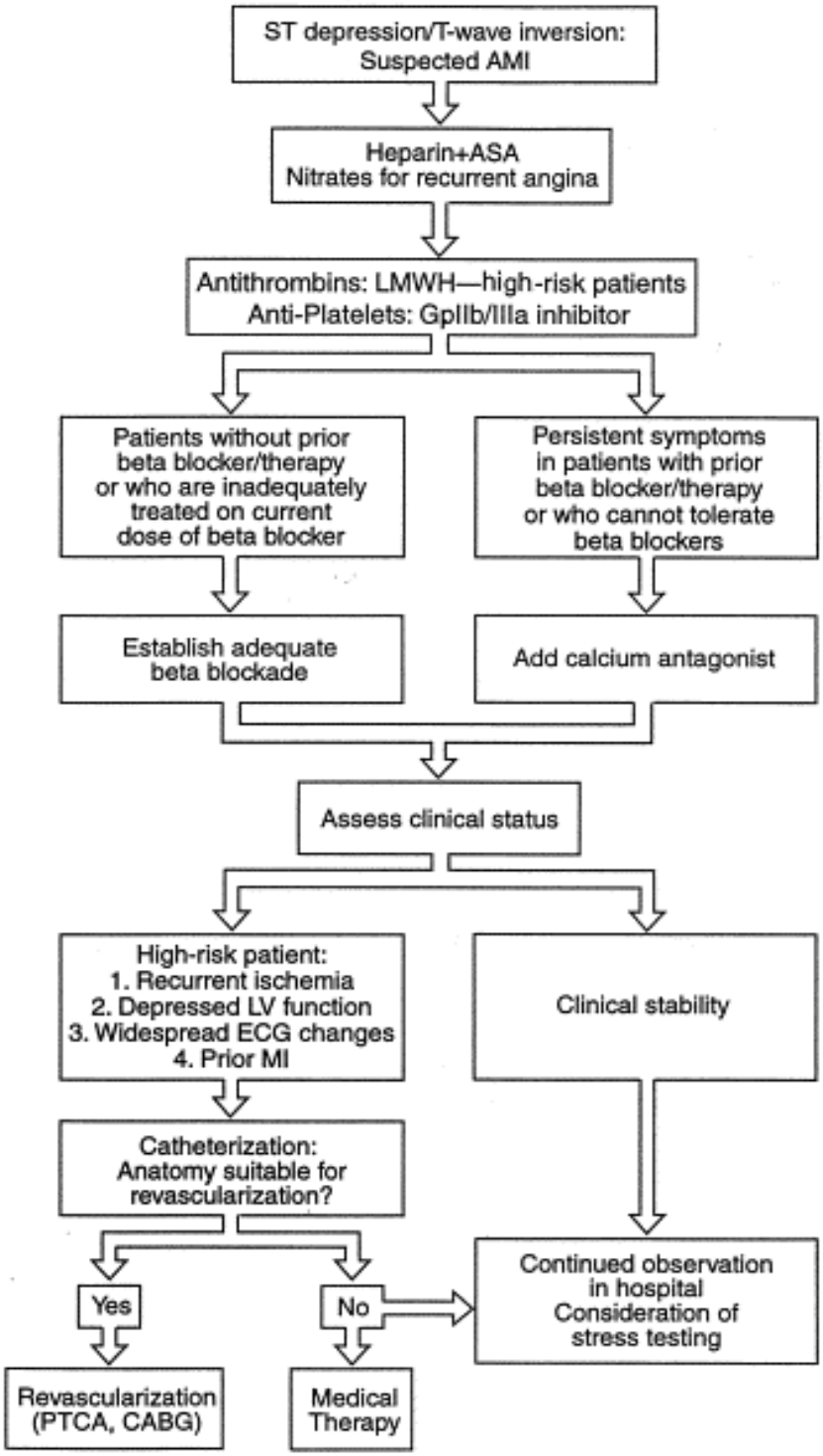
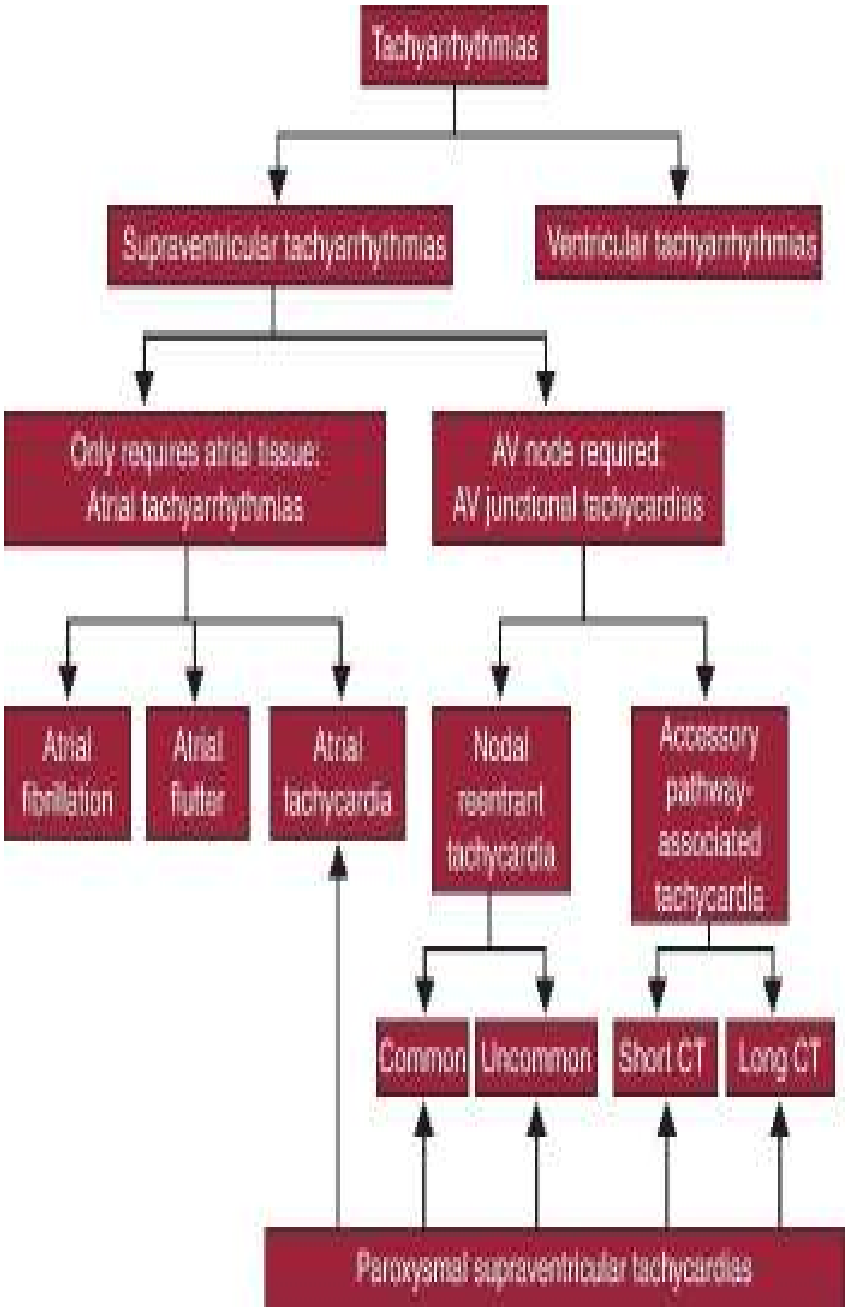


Chart 1 : ACC/AHA GUIDELINES FOR THE MANAGEMENT OF ACUTE MYOCARDIAL INFARCTION

MAGNESIUM AND ARRHYTHMIAS

CLASSIFICATION OF TACHYARRYTHMIAS



Mechanism of Tachyarrhythmias:

The mechanism by which magnesium decreases the incidence of cardiac dysrrhythmias remains unknown. Magnesium is essential for ATP activation, which is necessary for the maintenance of the sodium-potassium pump. It has a significant role in the maintenance of the resting membrane potential of electrocardiac cells, which is dependent on the intracellular potassium gradient. Magnesium deficiency may be associated with decrease in intracellular potassium, an increase in intracellular sodium concentration and an increase in excitability of the cardiac cells. It has been hypothesized that absence of reentry of potassium into the depolarized cell or a loss of potassium from already depolarized cells may result in abnormal conduction, phenomena of reentry and fibrillation [26,27] of the ventricles.

Another theory proposes magnesium to be a calcium blocker. The increase in intracellular sodium occurring due to magnesium depletion may be followed by a sodium-calcium exchange, resulting in an increase in intracellular calcium. Phasic influx of intracellular calcium may predispose to transient depolarization leading to repetitive dysrhythmias. Infusion of

magnesium has been shown to produce a clinical scenario similar to that produced by the infusion of a calcium channel blocking agent, characterized by peripheral vasodilatation, flushing, hypotension and decreased contractile strength of the heart[28].

Lysophosphatidyl choline (LPC) is an endogenous phospholipid usually released from the cell membranes during periods of ischemia and has significant local effects on cardiac tissue. LPC promotes membrane depolarization by reducing potassium conductance of the inward rectified current, thereby inducing cardiac arrhythmias. LPC also induces intracellular calcium accumulation in cardiac cells by the inhibition of sodium-potassium adenosine triphosphatase (ATPase) pump[29]. Increase in cytosolic calcium may prove detrimental to the cells by subsequent activation of calcium dependent phospholipases and proteases and by the generation of additional toxic fatty acids. Excess free intracellular calcium may potentiate the harmful effects of free radicals[30]. Magnesium serves as a critical cofactor of numerous myocardial ion pumps and also antagonizes calcium influx. Magnesium inhibits LPC induced increase in intracellular calcium. Hence, producing its antiarrhythmic effects by this mechanism.

Ventricular Tachyarrhythmias and Magnesium

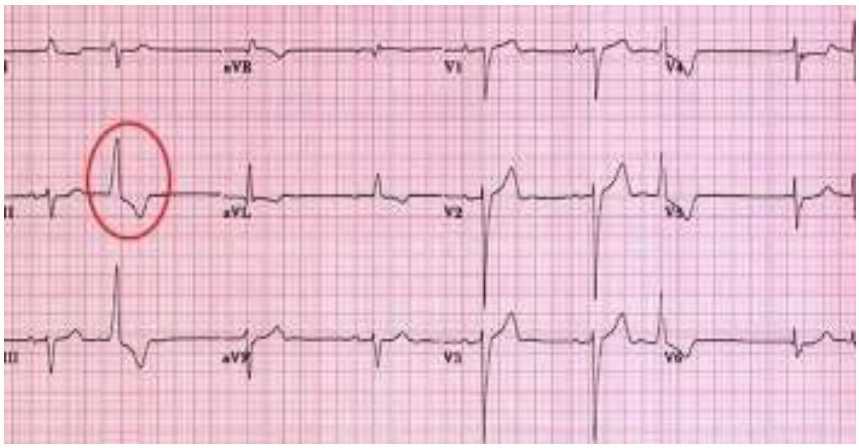
Zwillinger[31] in 1935, administered 15 ml bolus of a 20% solution of magnesium sulfate ($MgSO_4$) into the left ventricle of a patient with refractory ventricular fibrillation resistant to other therapy. It has been noted that the rhythm changed immediately to sinus rhythm.

Boyd and Schesf[32] in 1943 used 10-20 ml of 10% $MgSO_4$ for treating spontaneous dysrrhythmias..

Rasmussen et al[33] administered about 1.2 g of magnesium chloride ($MgCl_2$) to the patients with acute MI in the first 24 hours after AMI and about 300 mg in the second 24 hours. After comparing the results with placebo control group, it was concluded that those patients treated with $MgCl_2$ had significantly lesser incidence of dysrrhythmias (21% in control group versus 47%in study group) ($p<0.05$).

Digitalis toxicity induced ventricular dysrrhythmias are highly responsive to magnesium therapy. Magnesium deficiency is often seen during digitalis toxicity. Eventhough the serum magnesium levels are found to be normal, the intracellular magnesium is often decreased. Magnesium is antagonistic to the inhibitory effects of digitalis on sodium/ potassium ATPase. During therapy with digitalis, increase in intracellular calcium occurs resulting in increase in cellular excitability and inotropism [34]. In a study in monkeys, low magnesium levels were found to increase the risk and duration of digitalis toxicity.

VPCs

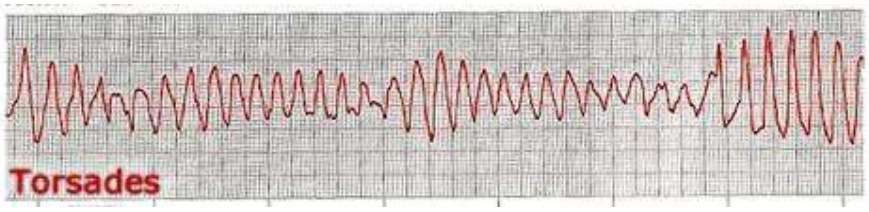


Holden et al[35] observed a marked reduction in Mg^{2+} during cardiovascular bypass surgery and also the first postoperative day. Dysrhythmias following cardiovascular surgery are reported to be

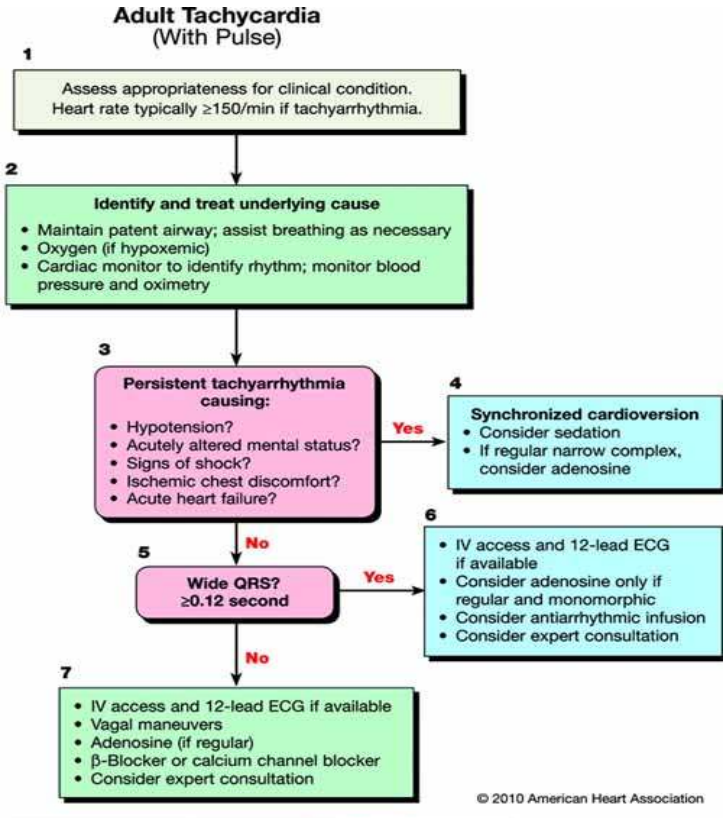
partially caused by hypomagnesemia occuring as a result of anticoagulants usage during surgery. Anticoagulants causes hypomagnesemia by binding with Mg^{2+}. Administration of Mg^{++} during the postoperative period has reduced the incidence of dysrrhythmias.

Torsades de Pointes

Torsades de Pointes (TdP) is a life-threatening ventricular dysrhythmia characterised repetitive polymorphic ventricular tachycardia commonly occurs in conditions where QT prolongation is seen. TdP is induced by type Ia antidysrhythmic drugs such as quinidine or disopyramide. Amiodarone, which produces QT prolongation may cause TdP. Hypokalemia and hypomagnesemia may also induce the occurance of TdP and in rare cases can be the cause[36].



In a longitudinal study of 12 patients with TdP , a single bolus dose of 2g of $MgSO_4$ reverted TdP within 1 to 5 minutes in nine patients[27]. In the other three patients, a second dose of $MgSO_4$ given after 5 to 15 minutes TdP completely. No side effects were associated with the treatment.



Doses/Details

Synchronized Cardioversion
Initial recommended doses:
- Narrow regular: 50-100 J
- Narrow irregular: 120-200 J biphasic or 200 J monophasic
- Wide regular: 100 J
- Wide irregular: defibrillation dose (NOT synchronized)

Adenosine IV Dose:
First dose: 6 mg rapid IV push; follow with NS flush.
Second dose: 12 mg if required.

Antiarrhythmic Infusions for Stable Wide-QRS Tachycardia

Procainamide IV Dose:
20-50 mg/min until arrhythmia suppressed, hypotension ensues, QRS duration increases >50%, or maximum dose 17 mg/kg given.
Maintenance infusion: 1-4 mg/min.
Avoid if prolonged QT or CHF.

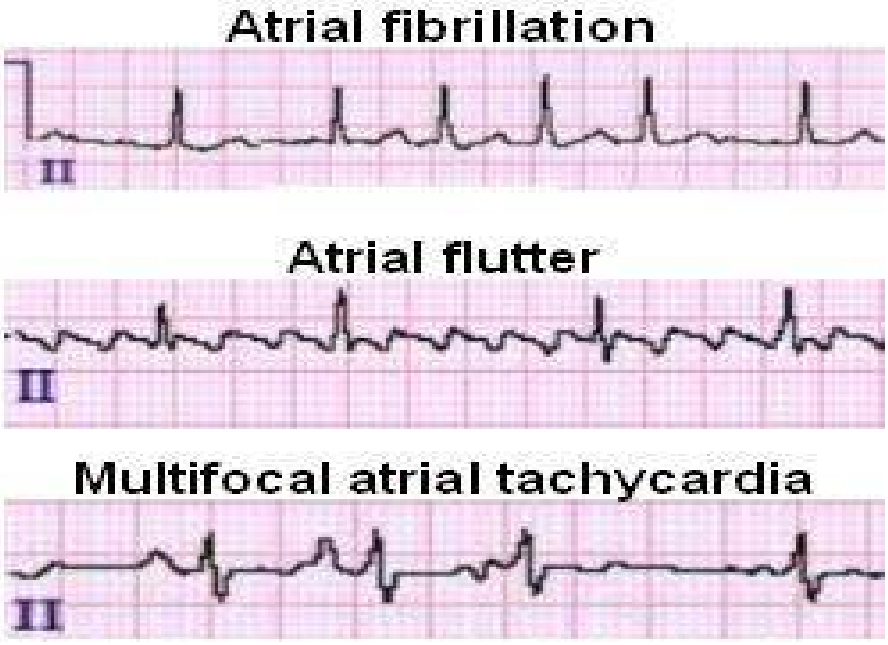
Amiodarone IV Dose:
First dose: 150 mg over 10 minutes.
Repeat as needed if VT recurs.
Follow by maintenance infusion of 1 mg/min for first 6 hours.

Sotalol IV Dose:
100 mg (1.5 mg/kg) over 5 minutes.
Avoid if prolonged QT.

Chart 3: ACC/AHA 2010 GUIDELINES FOR THE MANAGEMENT OF ADUT TACHYCARDIA

Atrial dysrrhythmias

Treatment of atrial fibrillation (AF) becomes difficult[38] in patients with magnesium deficiency. In a study of 45 patients with atrial fibrillation, 20% had serum magnesium levels approximately <1.5 mEq/L. Magnesium deficient patients needed twice the dose of IV digoxin to control AF. From the study it has been inferred that monitoring of serum magnesium level and magnesium replacement may prove to be useful in patients with symptomatic AF, especially in patients on digoxin therapy [39].



CLINICAL MANIFESTATIONS OF HYPOMAGNESEMIA

Intake of diets poor in magnesium in otherwise normal persons may lead to a syndrome characterized clinically by features like personality change, tremor, fasciculation , generalized spasticity and spontaneous carpo-pedal spasm. Administration of dietary magnesium results in a complete remission of its manifestations.

As magnesium deficiency usually occurs alongwith a primary pathology or a therapy. The effects of magnesium deficiency may be masked by the manifestations of the major illness. Hypomagnesemia either moderate or severe can manifest as follows:

Table-1

Manifestations of Moderate to Severe hypomagnesemia

Biochemical

Hypocalcemia

- Impaired PTH secretion
- Renal and skeletal resistance to PTH
- Resistance to vitamin D.

Hypokalemia[8]

- Renal potassium wasting
- Decreased intracellular potassium

Neuromuscular

- Seizures
- carpo-pedal spasms
- Muscular weakness, loss of musce bulk
- Nystagmus, vertigo, ataxia,.
- Psychiatric: Depressive and psychotic illness
- Athetoid movements
- choreiform movements

Cardiac Arrhythmias

ECG – prolongation of PR , QT interval and appearance of U-waves

Atrial tachycardia, atrial premature beats, atrial fibrillation

Ventricular premature beats

Ventricular tachycardia and fibrillation

Torsades de pointes (TdP)

Myocardial infarction

Biochemical Abnormalities

Hypokalemia

Hypokalemia is the most common feature of hypomagnesemia. During magnesium depletion, there is a loss of intracellular potassium resulting in subsequent depletion of intracellular potassium. This is followed by the failure of the kidney to retain potassium. Replacement of the potassium deficit with potassium therapy without simultaneous magnesium therapy may prove unsuccessful. The reason for the deranged metabolism of potassium may be due to dependence of Mg^{2+} on the Na^{+} K^{+} ATPase. During magnesium depletion , there is an increase in intracellular sodium and calcium associated with decrease in magnesium and potassium.

Magnesium seems to play a significant role in the regulation of potassium channels in cardiac cells.

Hypocalcemia

Hypomagnesemia is one of the most common causes of hypocalcemia[70]. In patients with hypocalcemia associated with hypomagnesemia, serum concentration of parathormone may be usually reduced.

Patients with hypomagnesemia are also resistant to the effects of vitamin-D. This has been attributed to the impairment in the metabolism of vitamin-D because of reduced concentration of 1, 25 dihydroxy vitamin-D in the serum. Hence, it should be taken into account that calcium or vitamin D therapy for the hypomagnesemia induced hypocalcemia may not be usually sufficient to correct hypocalemia. Magnesium therapy is indicated for regaining normal serum calcium concentration.

Neuromuscular effects

Neuromuscular hyper-excitability is the most common presenting complaint in a patient with hypomagnesemia. Latent tetany, characterized by positive Chvostek's and Trousseau's sign, or occurance of spontaneous carpo-pedal spasm may be found. Generalized tonic- clonic seizures may also occur.

Other signs often seen include vertigo, ataxia, nystagmus, athetoid and choreiform movements, tremor, muscular fasciculation,muscle wasting and weakness also may be seen[5].

The various mechanisms involved in neuromuscular manifestations include the following:

i) Magnesium is necessary for the stabilization of the nerve

axon. Decrease in the serum magnesium concentration reduces the threshold for stimulation of the axon and increases the velocity of nerve conduction.

ii) Magnesium induces the release of neuro-transmitters at the neuromuscular junction by inhibiting competitively, the entry of calcium into the pre-synaptic nerve terminal[71]. A fall in extracellular Mg^{2+} allows an influx of calcium in larger amounts into the pre-synaptic nerve terminal followed subsequently by the release of large amounts of neurotransmitters, leading to exaggerated neuromuscular activity.

Another mechanism by which hypomagnesemia and decreased intracellular magnesium concentration alters the neuromuscular excitability is attributed to the effects of magnesium on calcium handling by the muscle cell.

DIAGNOSIS OF MAGNESIUM DEFICIENCY

Magnesium deficiency can be assessed primarily by determining the serum magnesium concentration and intracellular magnesium content.

Serum Magnesium Concentration

Magnesium is primarily an intracellular cation. Only less than 1% of the total body magnesium content is present in the extracellular fluid. Hence, the serum magnesium concentration may not reflect the actual intracellular magnesium content. The measurement of the concentration of serum magnesium is the most commonly used test to assess magnesium status. The serum magnesium level less than 1.7 mg/100ml denotes the presence of hypomagnesemia.

Exogenous and endogenous catecholamines may cause a slight decrease in the serum magnesium concentration. Increased catecholamine leves may contribute to the occurance of hypomagnesemia in states of acute illness and stress[17]. Hypovolemia and rhabdomyolysis (cellular magnesium release) may increase the serum magnesium concentration and may falsely mask the intracellular magnesium deficit.

Intracellular Magnesium Content

The magnesium level of the peripheral leucocyte has been studied, it parallels with the magnesium content of skeletal and cardiac muscle. Estimation of the intracellular magnesium content appears to be a good marker of total body magnesium level than the magnesium concentration in the serum.

Magnesium Tolerance Test

It is an accurate method of assessing magnesium status of the body. Parenterally administered magnesium is retained at higher rate in both hypomagnesemic patients and patients who have normal magnesium levels at risk of developing hypomagnesemia..

The recommended protocol is as follows:

1. Baseline urine (spot and timed) should be collected for estimating magnesium creatinine ratio.
2. 0.2 meq (2.4 mg) elemental magnesium per kg body weight in 50 ml 5% dextrose water should be infused for 4 hours.
3. 24 hours urine collection to estimate magnesium level.
4. Percent of magnesium retained should be calculated

using the following formula.

$$\text{percentage magnesium retained} = 1 - \frac{\text{(Post infusion 24 hours,) – (preinfusion urine Mg2+/ Cr) x (post infusion Cr)}}{\text{Total Mg2+ administered}} \times 100\%$$

5. Criteria for diagnosis:

>1/2 retention at 1 day = definite deficiency

>1/4 retention at 1 day =probable deficiency

Treatment of Magnesium Deficiency

Those who have clinical manifestations suggesting hypomagnesemia and are at risk of becoming hypomagnesemic must be given magnesium therapy

Most patients with magnesium deficiency can be treated by g dietary advice itself. But , losses through GIT or kidney is present, supplements should be given. Before starting therapy, renal function should be assessed. Even in severely hypomagnesemic subjects, about 50% of the dose administered is excreted in the urine so in the presence of renal failure, therapeutic doses should be reduced markedly. At magnesium levels about 1 to 2 mequivalents/kg, patients will be symptomatic.

Patients with moderate to severe magnesium deficiency are

usually symptomatic and should be treated by parenterally administering magnesium.

Administration of Parenteral Magnesium

A single dose of IV Mg^{++} can be effective in the treatment of hypomagnesemia and a large amount of the dose administered will be excreted by the kidneys.

The maximum loading dose of Mg^{++} recommended was found to be 150 mg/min[72]. In emergency situations such as ventricular tachycardia or ventricular fibrillation, the adult ACLS guidelines recommends 1 g of magnesium diluted in 100 ml to be given over 1 to 2 minutes[73]. During magnesium therapy, continuous electrocardiogram monitoring should be done to avoid cardiac toxicity. Blood pressure monitoring should also be done during IV administration of Mg^{++} since hypotension may be caused if rapidly infused. The infusion should be given slowly if hypotension occurs. Rapid IV administration of Mg^{++} may cause cutaneous flushing, sweating, fall in blood pressure, somnolence, decreased deep tendon reflexes, hypocalcemia, tetany, respiratory impairment followed by respiratory failure and cardiac arrest can occur.

These side effects can be reduced by slowing the infusion rate. During drug therapy, serum magnesium level, neurological status, respiratory status, and renal function shoud also be monitored. Before starting treatment and also during treatment, assessment of patellar reflex should be done. If there is any impairment of the reflexes, the therapy should be stopped immediately.

If renal insufficiency is present, the magnesium dose is reduced by 25% to 50% to prevent magnesium overload. Magnesium therapy is closely guided according to the serum magnesium level to avoid toxicity. Most common complication associated with $MgSO_4$ administration is hypocalcemia. The sulfate in $MgSO_4$ binds with calcium, forming calcium sulfate and reducing ionized calcium. Zaloga and Charnow suggested IV magnesium chloride, instead of $MgSO_4$ to prevent precipitation of calcium sulphate. Calcium gluconate should be kept ready for the emergency management of hypocalcemia or tetany.

Administration of Oral Magnesium

For mild forms of hypomagnesemia, oral magnesium supplementation can be given, although its bioavailability may vary. Higher doses of oral magnesium salts may often cause

diarrhea. However, Mg^{++} in the Mg^{++} chloride form or in enteric-coated tablets is well tolerated usually. A course of three magnesium chloride tablets per day for 30 days may reduce the deficiency in most patients[74]. In patients on diuretics, the substitution or addition of a potassium/ magnesium-sparing diuretic may prove beneficial.

HYPERMAGNESEMIA

Hypermagnesemia usually occurs iatrogenically and is commonly observed in cases of acute and chronic renal failure. It is especially seen with administration of exogenous magnesium such as antacids, enemas or parenteral nutrition.

In early stages of chronic renal failure, serum magnesium level seems to be normal. As renal insufficiency worsens, Mg2+ loss increases in parallel with that of Na2+and other elements. Excess Mg2+ can be removed by dialysis, but the use of dialysate may itself cause hypermagnesemia because of its high magnesium concentration. Therapeutic administrationof magnesium parenterally conditions like toxemia of pregnancy, adrenal insufficiency and hypothyroidism may result in magnesium excess.

Enemas rich in magnesium content may cause hypermagnesemia even in subjects having functional kidneys. This has been attributed to the ECF depletion which might reduce the Mg2+ loss in urine.

Magnesium excess may manifest clinically if Mg2+ levels are more than 4 mequivalents/L.

Table 2: Dose related manifestations of hypermagnesemia

Magnesium Serum level (mg/dL)	Dose related effects
1.7–2.4	Normal serum levels
5–8	Nausea, headache, light headedness, cutaneous flushing.
9–12	Absent deep tendon reflexes, somnolence, hypotension.
12–15	Sinoatrial and atrioventricular block, muscle paralysis, hypoventilation
>15	Cardiac asystole, respiratory arrest, coma

Neurological manifestations occur when plasma magnesium level exceeds 10 meq/L. Depression of neuromuscular function occurs initially which is characterized by impaired / absent DTR. paresis of muscles which act voluntarily occur resulting in flaccid quadriparesis/ insufficiency in respiration . Level of consciousness may not be affected. If magnesium is present in excess amounts, altered sensorium can occur.

When serum Mg2+ more than (15 mequivalent/l), cardiac effects occur. Hypotension, prolonged PR-interval in ECG, intraventricular conduction defects, complete heart block or cardiac

arrest in systole may occur.

Treatment of magnesium excess are withholding of the magnesium infusion, dialysis by PD/HD . Calcium blocks the effects of Mg2+. Hence, IV administration of calcium ion 5-10 meq (200 mg) may be required to treat the manifestation of hypermagnesemia.

Magnesium can also be used in the treatment of COPD and bronchial asthma. Several studies have reported that there will be an improvement in peak expiratory flow rates after infusion of magnesium who are not responding to the conventional therapy. Magnesium is also used in the treatment of severe preeclampsia and eclampsia , as a tocolytic in preterm labour. Anticonvulsant effect may be due to the depression of neuromuscular transmission or as a result of direct depressant effect on smooth muscle (or) as a central nervous system depressant. Tocolytic effect of magnesium is due to antagonism of calcium mediated myometrial contraction. Now magnesium has been on trial for the treatment of vascular headaches.

MATERIALS AND METHODS

Data resource:

50 patients diagnosed of having acute myocardial infarction, admitted to IMCU, Coimbatore medical college and hospital, over a period of 1 year i.e., between August 2013 to August 2014 were selected by using simple random method.

Selection of patients:

Inclusion Criteria:

Patients who presented to the hospital within 12 hours of onset of symptoms were included in the study.

The following criteria has been used to diagnose acute myocardial infarction. The presence of any of the two criteria has been considered :

1. History of discomfort in the chest.
2. Changes in the ECG suggestive of acute myocardial infarction
3. Rise of cardiac enzymes.

Exclusion Criteria

1. Patients having hypokalemia.
2. Patients on diuretics

After the selection of patients through random method, after obtaining informed written consent from all the patients included in the study, relevant history and physical examination was done. Patients were subjected to undergo investigations like complete blood count, urine examination, blood sugar, blood urea, serum creatinine, fasting lipid profile, cardiac enzymes and ECG was done in all cases.

Estimation of serum magnesium level was done on day 1 and day 5.

Method of estimation serum magnesium:

The method used was colorimetric end point test with Xylidyl blue as the reagent.

Magnesium standard: 2.5 mg/dL.

Principle:

Magnesium reacts with xylidyl blue at alkaline pH resulting in the formation of a chelating red colored compound. The increase in the red colour (or) the decrease in blue color are proportionate to the concentration of magnesium in the serum..

Specimen

Analysis of non-hemolyzed serum or lithium heparin plasma

may be done since the concentration of magnesium inside the red cells is 10 times greater than that in the ECF. Separation of serum from the cell should be done as early as possible and hemolysis should be avoided.

Normal range for magnesium

Serum magnesium: 1.6 – 2.4 mg/dl.

RESULTS

The data collected was statistically analysed using various statistical tools such as ANOVA, Chi-square test, student 't' test.

Table-2: Age and Sex Distribution of the Study group

Age range (years)	Sex		Total
	Male	Female	
30 – 40	10	--	10
40 – 50	14	2	16
50 – 60	5	5	10
60 – 70	11	1	12
70 – 80	2	--	2

In this study group of 50 cases, 42 were males and 8 were female patients with a male-female ratio of 5.25:1. The maximum Incidence of acute myocardial infarction was seen in the 4^{th} and 5^{th} decades, followed by 6^{th} and 7^{th} decades. 28% patients were in the age group of 4^{th} and 5^{th} decade, 22% were in the age group of 60-70.

Figure-1: Age and Sex Distribution of the Study group

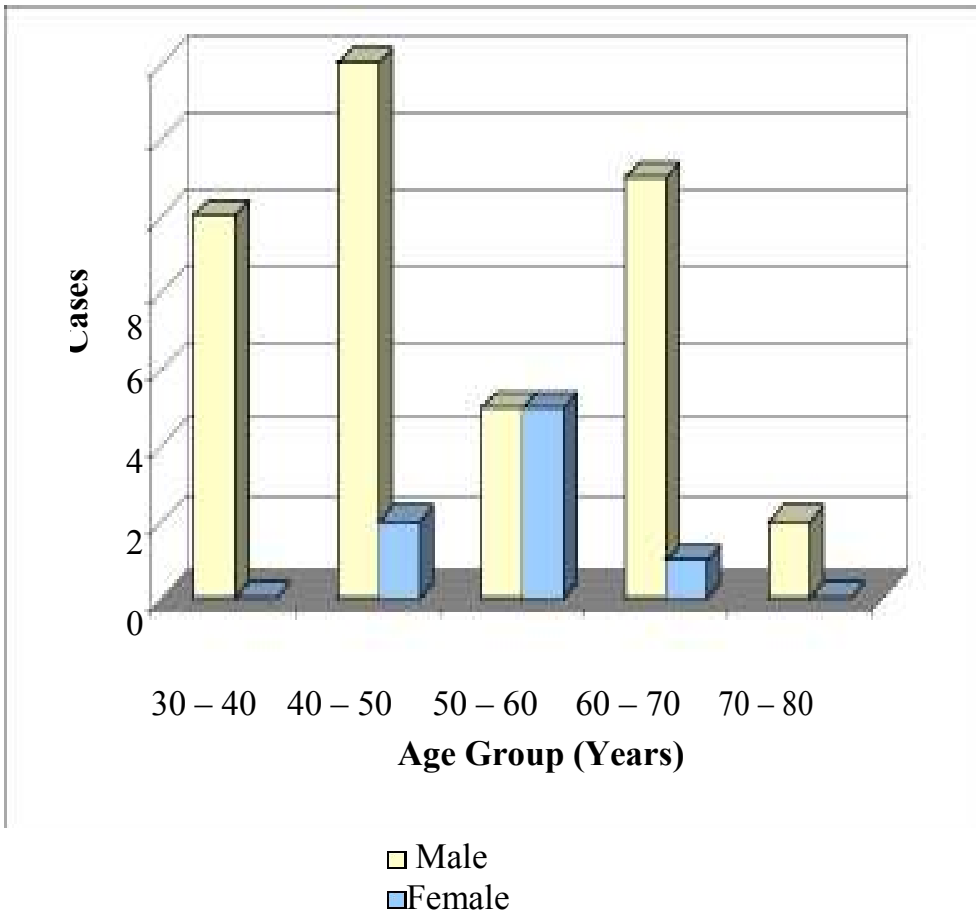
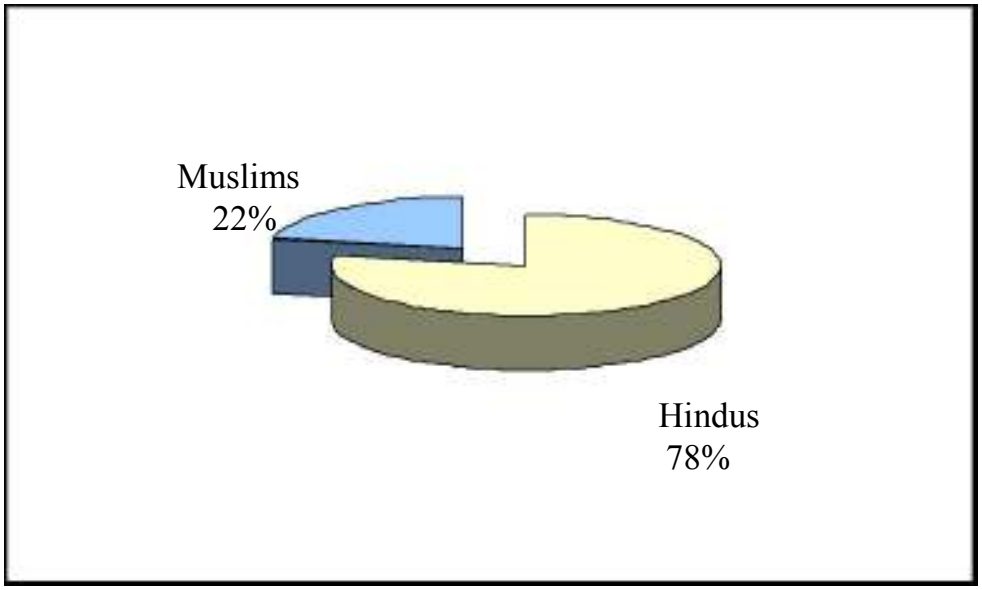


Table-3: Religion wise Distribution of cases

Religion	No. of cases	Percentage
Hindus	39	78.00
Muslims	11	22.00

In the study of 50 patients, 39 (78%) were Hindus and 11 (22%) were Muslims. The higher incidence of acute myocardial infarction is not factual one but reflects the difference in population.

Figure-2: Religion wise Distribution of cases



Diet:

In the study of 50 patients, 15 (30%) patients were vegetarian by diet and 35 (70%) of them consumed mixed diet. Non-vegetarian run higher risk of acute myocardial infarction owning to their higher content of cholesterol in their diet compared to the vegetarian.

Figure-3: Diet

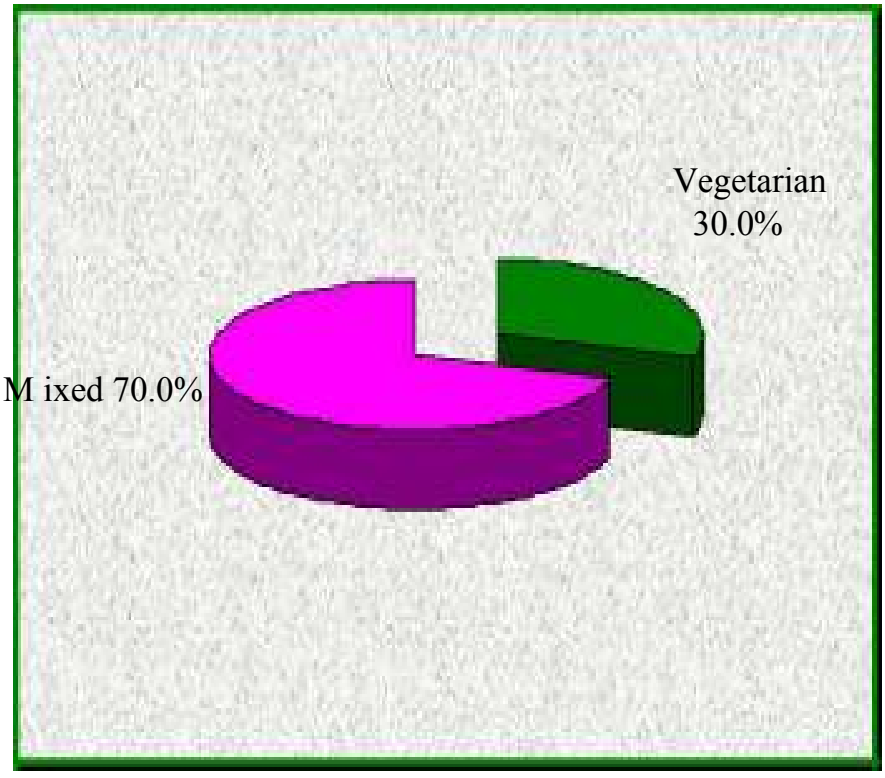


Table-4: Risk Factors

Risk factors	No. of cases	Percentage
Smoking	35	70.00
Family history of HTN, DM, IHD, CVA	10	20.00
Obesity	12	24.00
Hypertension	15	30.00
Diabetes mellitus	18	36.00
Dyslipidemia	6	12.00

Smoking

In the study, smoking is the most common risk factor found in the patients with acute myocardial infarction. Cigarette smoking accelerates coronary atherosclerosis in both sexes and at all ages and increases the risk of thrombosis, plaque instability and myocardial infarction. In addition, by increasing myocardial oxygen needs and reducing oxygen supply, it aggravates angina.

Obesity

In the present study, out of 50 patients, 12 (24%) were found to be obese based on National Cholesterol Education Programme.

Waist circumference was measured in all patients. Men whose waist circumference is more than 102 cm and females whose waist circumference is more than 88cms were considered to be obese.

In the present study of 50 patients, 15 (30%) patients were found to be hypertensive. Patients whose blood pressure is more than 130/85 are considered to be hypertensive.

In the present study of 50 patients,18(36%) patients were found to be diabetics and 12 (24%) patients were found to be dyslipidemic.

Table-5: Time of Presentation

Time at presentation	No. of cases	Percentage
0 – 3 hours	14	28.00
3 – 6 hours	23	46.00

In the present study, 23 (46%) cases presented to the hospital between 3-6 hours of onset of symptoms and 14 (28%) cases presented between 0-3hours.

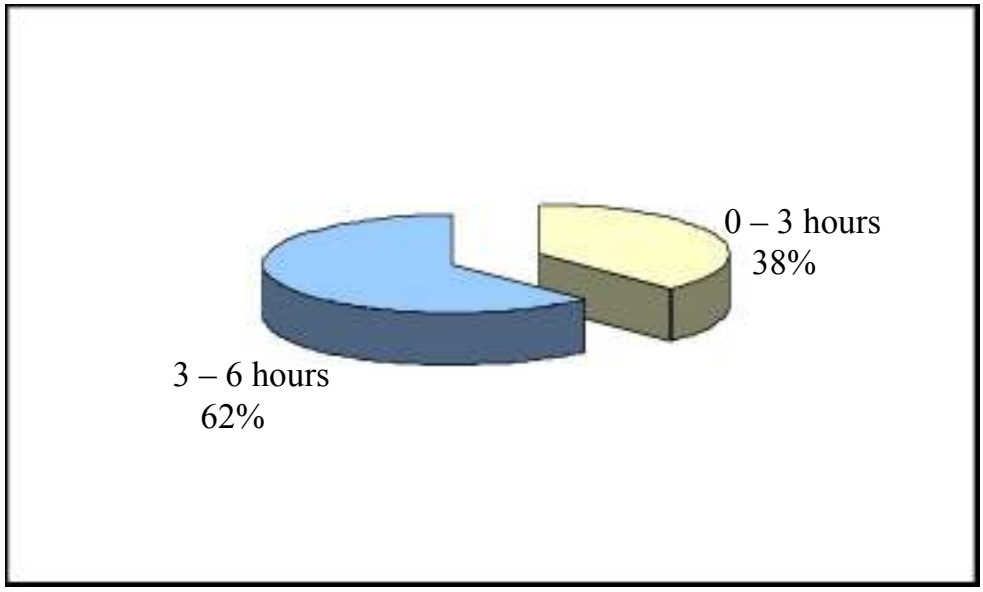
Presentation to the Hospital

Chest pain was the commonest symptom and was present in all of the patients in the present study (100%). In this study chest pain is associated with sweating 13 (26%) of patients. Chest pain is associated with breathlessness in 8 (16%) of the patients. Palpitation associated with chest pain was present in 1 patient (2%).

Variation in type of Myocardial Infarction

In the present study of 50 patients, 21 (42%) patients had anterior wall MI, 17 (34%) patients had inferior wall MI and 9 (18%) patients had anteroseptal MI and 3 (6%) patients had anterolateral MI.

Figure-4: Time of Presentation



Serum magnesium level in acute myocardial infarction in relation to arrhythmia

In this cross sectional study of 50 patients, the mean serum magnesium level on day-1 in all 50 patients was 1.86±0.39 and the mean serum magnesium level on day-5 was 2.26±0.5.

Mean serum magnesium level in the group with Arrhythmia on Day-1and Day-5

In the present study, out of 50 patients 25 patients had significant ventricular premature contractions or ventricular tachycardia or ventricular fibrillation during their 5-days course in the hospital.

Table-6: Serum magnesium levels in patients with arrhythmias

Serum magnesium levels (mg/dL)	Day-1	Percent	Day-5	Percent
<1.6	8	16.00	2	4.00
1.6 to 2.40	17	34.00	16	32.00
>2.4	--	--	2	4.00

Table-7: Serum magnesium levels in patients without arrhythmias

Serum magnesium levels (mg/dL)	Day-1	Percent	Day-5	Percent
<1.6	2	4.00	--	--
1.6 to 2.40	17	34.00	14	28.00
>2.4	6	12.00	9	18.00

Table-8: Mean serum magnesium level

	Day-1	Day-5
Mean serum magnesium level in 50 cases	1.86±0.39	2.26±0.50
Mean serum magnesium level in patients with arrhythmia(25patients)	1.65±0.26	1.98±0.25

Figure-5: Mean serum magnesium level

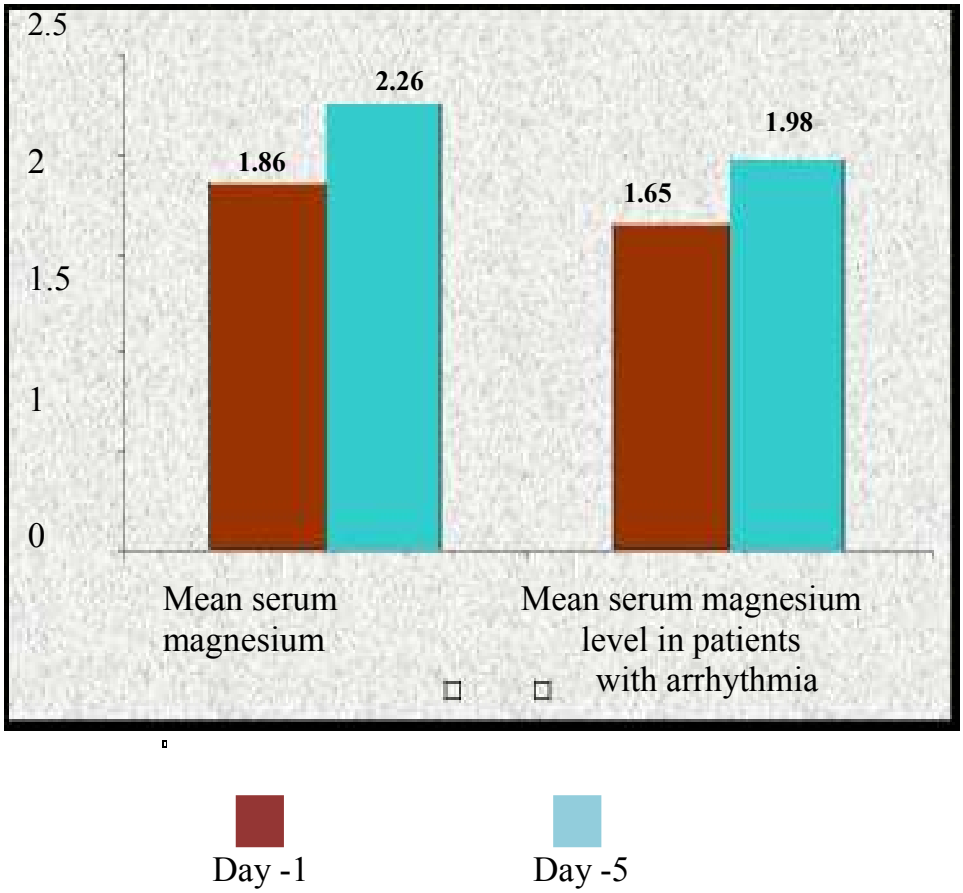


Table-9: Comparison of Serum Magnesium level in patients with Arrhythimas and without Arrhythmias (Day-1)

	No. of Cases	Serum magnesium Day-1	t- value	p-value
Mean serum magnesium level in patients with arrhythmia	25	1.65±0.26	4.63	<0.001
Mean serum magnesium level in patients without arrhythmia	25	2.08±0.41		

The above table shows that out of 50 patients, 25 patients had arrhythmias. The mean value of serum magnesium on day-1 those with arrhythmias is 1.65±0.26 those without arrhythmias is 2.05±0.4 ($p<0.001$). There is a significant difference in the magnesium level in patient with arrhythmias and without arrhythmias.

Figure-6: Comparison of Serum Magnesium level in patients with Arrhythimas and without Arrhythmias (Day-1)

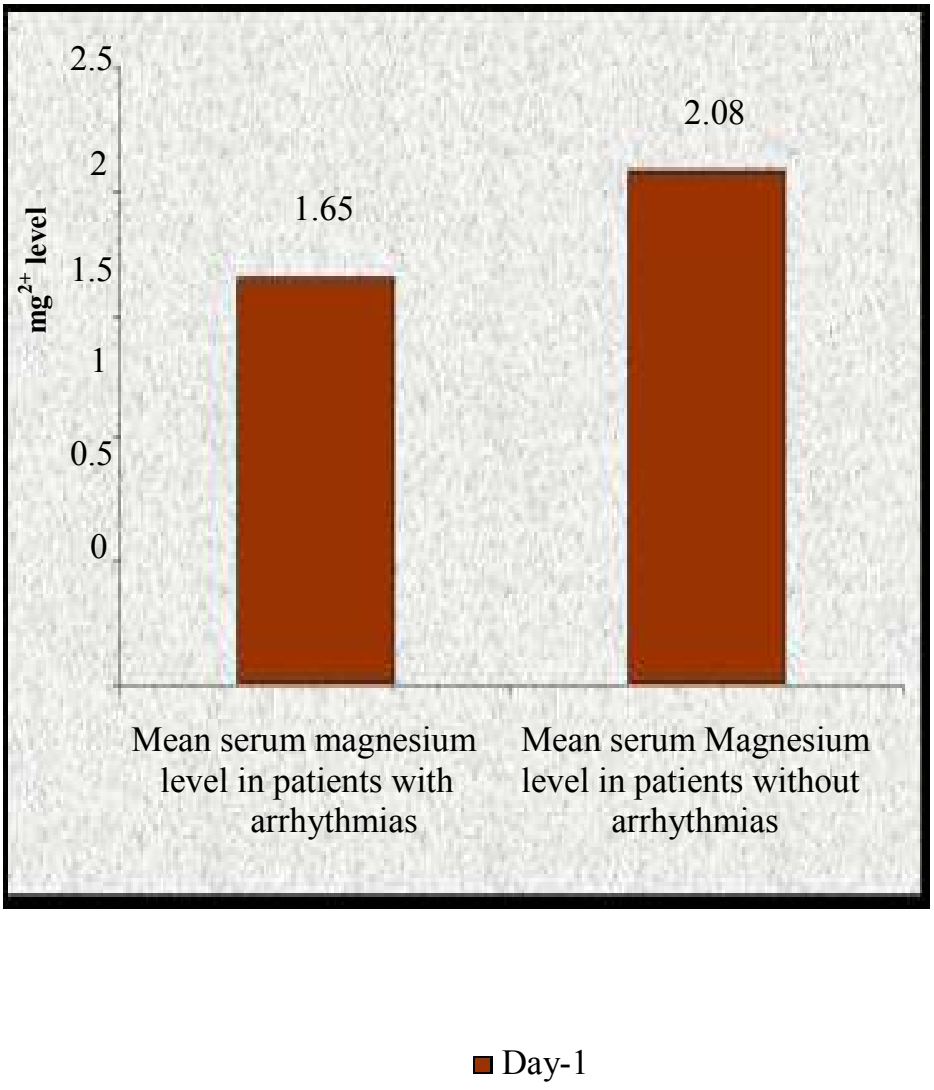
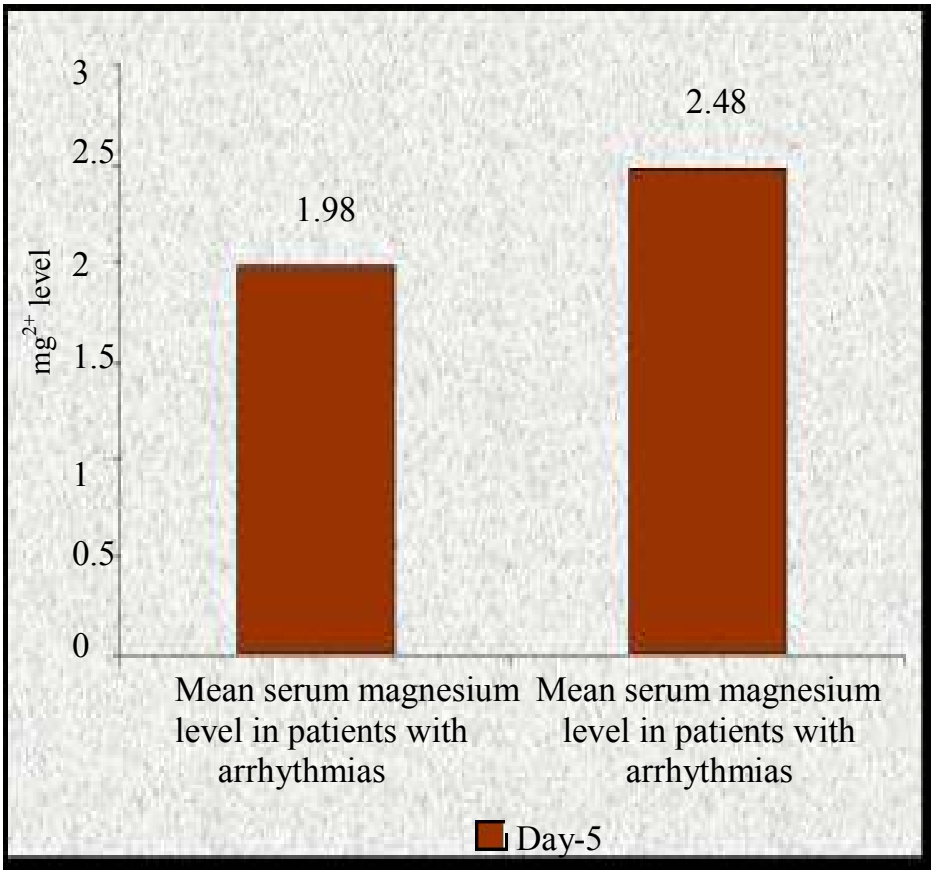


Table-10: Comparison of Serum Magnesium level in patients with Arrhythimas and without Arrhythmias (Day-5)

	No. of cases	Serum magnesium Day-5	t- value	p-value
Mean serum magnesium level in patients with Arrhythmia	20	1.98±0.25	4.17	<0.001
Mean serum magnesium level in patients without Arrhythmia	23	2.48±0.52		

The above table shows that serum magnesium level in patients with arrhythmia on Day-5 is 1.98±0.25 and in those without arrhythmia is 2.48±0.5. The difference between these two is found to be statistically significant with p- value ($p<0.001$).

Figure-7: Comparison of Serum Magnesium level in patients with Arrhythimas and without Arrhythmias (Day-5)



Mortality:

In the above study of 50 patients, 7 patients died during their 5 days hospital course. 5 patients were died of ventricular tachycardia or ventricular fibrillation, 2 patients were died of cardiogenic shock. Mortality percentage was 14%

DISCUSSION

Magnesium ion has recently been considered as a principle cardiovascular cation. It has many critically significant roles in the maintenance of normal homeostasis of the body. It plays a major role in cardiac homeostasis. Magnesium is essential ATP activation necessary for the maintenance of the sodium-potassium pump. Magnesium deficiency has been attributed to the causation of arrhythmias in acute myocardial infarction patients.

In the study group comprising of 50 patients, 42 were males and 8 were females with a male-female ratio of 5.25:1. The maximum incidence of acute myocardial infarction was seen in the 4^{th} and 5^{th} decades.

In the present study of 50 patients, the mean serum magnesium level on day-1 in all 50 patients was 1.86±0.39 and the mean serum magnesium level on day-5 was 2.26±0.5.

Abraham et al[13] studied the level of serum magnesium in 65 patients admitted and diagnosed to have acute myocardial infarction. Concentration of magnesium in serum was noticed to be reduced in patients who were diagnosed with AMI (mean 1.70 mg/dl, $p<0.001$) or acute coronary insufficiency (mean 1.61 mg/dl,

p<0.01), but was not seen in the control group or patients had chest pain of non cardiac origin (mean 1.91 mg/dl).

Singh A et al[75] investigated magnesium levels in the serum of twenty patients diagnosed of having acute myocardial infarction on the 1st, 7th and 12th day of admission. In most of the cases, there was a marked reduction in the magnesium level of the serum on the first day.

Dimtruk[76] conducted the study among 67 patients diagnosed with ischemic heart disease and made out that there was a remarkable decrease in the magnesium levels in the serum during the first 3 days from the time of presentation and found that the magnesium levels returned back to normal by 15-25 days from the time of presentation.

Sachdev et al[77] (1978) selected 30 patients, diagnosed with myocardial infarction and monitored the magnesium levels within 24 hours, 5th and 8th day and it was found to be 1.83±0.087 mgm%, 1.91±0.149 and 1.97±0.089 whereas in the control group, it was 2.44±0.162 mgm%. The values were reported to be statistically reduced on all the three days and increased thereafter.

In the present study, the serum magnesium level on day-1 was significant lower in patients with arrhythmias than those without arrhythmia (p<0.001). There was an increase in serum

magnesium from Day-1 to Day-5 in both those with arrhythmias and those without arrhythmias.

Ceremuzynski et al[7] selected 48 patients with acute myocardial infarction of duration over 24 hours and infused magnesium or placebo. The occurrence of ventricular tachycardia (3 or more subsequent premature ventricular beats with a rate more than 120/ min) was significantly decreased ($p<0.001$), but the occurrence of other ventricular arrhythmias was unaffected.

Raismusen et al[21] selected 273 patients with diagnosis of acute myocardial infarction and subjected them to IV administration of magnesium or placebo. A significant reduction in the occurrence of ventricular arrhythmia in the magnesium group was noticed when compared to placebo group ($p<0.05$).

Shecter et al[79] subjected 103 patients diagnosed of having acute myocardial infarction to magnesium infusion or placebo for 48 hours. A significant fall in mortality rate ($p<0.01$) was found. The occurrence of tachyarrhythmias in need of treatment (10/50) has been very low in the magnesium group when compared to the placebo group (24/53).

Smith et al[80] randomly administered 24 hours continuous

magnesium sulphate infusion or placebo in 400 patients with the suspicion AMI. Out of which, 200 patients were diagnosed of having acute myocardial infarction. There is no significant variation in the mortality rate or the occurrence of ventricular dysrhythmia in need of treatment among the magnesium and placebo groups.

Abraham et al[81] randomly allocated 94 patients diagnosed of having acute myocardial infarction and administered a daily bolus of magnesium of about 30 mmol or placebo for 3-days. No significant variation was noticed in the mortality rate or life threatening arrhythmias among patients on magnesium treatment and with placebo.

Felstedt et al[82] randomly subjected 298 patients with the suspicion of acute myocardial infarction to magnesium infusion or placebo for 24 hours. Myocardial infarction was confirmed in 162 patients. During 245 days period, no variation was noted in the occurrence of tachyarrhythmias. Increase in the occurrence of bradyarrhythmias was noted among patients infused with magnesium.

Singh et al[83] subjected 264 patients with suspicion of acute myocardial infarction to potassium, magnesium, 2% glucose or

10% glucose infusion. 228 patients were confirmed to have myocardial infarction. There was no difference in the mortality rate and ventricular tachycardia or fibrillation between the two groups.

Morton et al[84] assigned 76 patients to get either magnesium infusion 0.38 mmol/l per kg every 12th hourly or placebo for the first 36 hours of hospital stay. No difference was noticed in the occurance of ventricular tachycardia among the two study groups.

Dyckner T et al, among 905 admission, found 342 patients with acute myocardial infarction, 563 with other diagnoses. Both acute myocardial infarction and non AMI group had markedly reduced serum magnesium levels compared to the reference group. The occurrence of life threatening ventricular premature beats, ventricular tachycardia or ventricular fibrillation on admission was found to be high in patients with acute myocardial infarction with reduced serum magnesium levels.

SUMMARY

This study was carried out in 50 patients of acute myocardial infarction who are admitted to the ICCU, Coimbatore Medical College Hospital, Coimbatore.

1. The male to female ratio in the study group was 5.25:1 and the maximum incidence of acute myocardial infarction was seen in 4^{th} and 5^{th} decade.
2. In the study Hindus were 78% and Muslim were 22%.
3. In the study, the most common presentation symptom was chest pain and is associated with sweating in 26% of patients and breathlessness in 16% of patients and palpitation in 2%.
4. In the study, the most common risk factor found was smoking followed by hypertension and diabetes.
5. In the study group, mean serum magnesium level in 50 patients on day-1 is 1.86±0.39 and on day-5 is 2.26±0.5.
6. In the study group, mean serum magnesium level in 25 patients with arrhythmia is 1.65±0.26 on day-1 and 1.98±0.25 on day-5.
7. In the study group, mean serum magnesium level in 25 patients without arrhythmia is 2.05±0.41 on day-1 and2.48±0.52 on day-5.

8. The difference between the magnesium level in patients with arrhythmia and without arrhythmia is statistically significant on both day-1 and day-5.

CONCLUSION

Coronary artery disease is the major cause of morbidity and mortality throughout the world. Major cause of death in coronary artery disease may be due to complications like arrhythmias.

In the present study, patients with acute myocardial infarction with low magnesium levels are more prone to develop ventricular arrhythmias compared to those who are having normal magnesium levels. Magnesium replacement therapy in patients with acute myocardial infarction who are having low serum magnesium level may reduce the incidence of arrhythmias.

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PROFORMA

Name:	Sl. No.	Unit
Age	IP No.	DOA
Sex	DOD	
Occupation	Religion	
Final diagnosis		

Presenting Symptoms

1. Chest pain
2. Sweating
3. Dyspnoea
4. Orthopnea
5. PND
6. Palpitation
7. Others

History of present illness

1. Chest pain

   * Location

   * Nature — Severity: Mild/ moderate/ severe
   * Nature — Character: Pricking/ throbbing; Squeezing/ constricting; Burning; Agony/ other types

   * Onset – Hrs/ days back
   * Radiation
   * Continuous/ intermittent
   * Aggravating factor
   * Relieving factor — On taking rest/ Drugs

2. Sweating
3. Dyspnea
4. Orthopnea
5. PND
6. Palpitation

    * Nature Continuous/ racing of heart beats

    Other irregularities

    *After excitement/ exercise/ drugs

    *Periodicity

    *Duration

7. Cough

    *Dry/ productive

    *Frothy sputum/ hemoptysis

8. Gastrointestinal symptoms

    *Nausea

    *Vomiting

    *Dyspepsia/ heart burn/ indigestion

    *Hiccoughs

9. Neurological symptoms:

    *Convulsions

    *Giddiness/ Fainting

    -Time of onset after pain

    -Duration

    -Consciousness

10. Vascular symptoms

    Phlebitis

    Varicosity Claudicatin

    * Raynaud's phenomenon

11. Endocrine symptoms

* Polyuria/ polydypsia/ polyphagia
* Excessive heat/ cold intolerance
* Any change in voice
* Change of hair distribution

12. Miscellaneous

* Swelling of the limbs
* Abdominal distension
* Visual disturbance

III Past History

1. Angina
   * Type
   * Duration
   * Frequency
   * Any treatment taken
2. Myocardial infarction
   * No. of attacks
   * Details of admission and treatment
3. Hypertension
   * Duration
   * Treatment – not taken/ regular/ irregular
   * Controlled/ Uncontrolled
4. Stroke/ TIA
   * Treatment
5. Diabetes mellitus
   * Duration
   * Treatment – Not treated/ regular/ irregular
   * Controlled/ Uncontrolled

6. Renal diseases
7. Peptic ulcer
8. Rheumatic fever

ii) Diuretics

iii) Others

10. Drinking water source
11. If female

i) Menstrual history

ii) Obstetric history

iii) Oral contraceptives

VI} General Physical Examination

1. Built: Well built/ dwarf/ average

   Height _____ Weight _____
2. Nutrition: Normal/ Obese/ undernourished
3. State of consciousness
4. Cyanosis
5. Clubbing
6. Anemia
7. Edema
8. Xanthoma/ Xanthelasmas
9. Arcus senilis/ Juvenilis

VII] Systemic Examination

1. Pulse

   *Rate

   *Rhythm

   *Character

   *Volume

*Condition of vessel wall – normal/ thickened

*Other peripheral pulses

2. BP

Upper limb	Lower limb

3. JVP

4. Epigastric pulsation

a) Inspection

i) Shape of chest

ii) Apical impulse

iii) Other pulsations

b) Palpation

i) Apical impulse

ii) Palpable heart sounds

iii) Parasernal heave

iv) Thrills

v) Other pulsations

c) Percussion

i) Left border of the heart

ii) Right border of the heart

iii) Upper border of liver dullness

iv) Any abnormal areas of dullness (Lt/ Rt I ics)

d) Auscultation:

i) Heart sounds: Normal/ accentuated/ muffled/ slit

*1^{st} sound

*2^{nd} sound

*3^{rd} sound

*4^{th} sound

Mitral area

Tricuspid area

Pulmonary area

Aortic area:

a) Extra sound: Irregular rhythm/ triple rhythm/ galloop rhythm

Murmur

Pericardial rub

b) Respiratory system

c) Gastrointestinal system

d) Genitourinary system

VIII] Investigation

1. Urine Albumin

Sugar

Micro

2. ECG at the time of admission

3.

	S.magnesium	Sodium	Potassium	SGOT	SGPT
1st day	_____	_____	_____	_____	_____
5th day	_____	_____	_____	_____	_____

IX] Course in Hospital

	Clinical parameters	ECG	Management
1st day			
2nd day			
3rd day			
4th day			
5th day			
6th day			
7th day			

X] Final remarks and the summary of the case:

CONSENT FORM

Mr/Mrs/Ms……..…
….S/W/D of……(legal guardian) is being asked to be a participant in the research study titled " SERUM MAGNESIUM LEVELS IN ACUTE MYOCARDIAL INFARCTON " in Coimbatore Medical College. He /she satisfies eligibility as per the inclusion criteria. You(legal guardian) can ask any question you may have before agreeing to participate.

Research Being Done

SERUM MAGNESIUM LEVELS IN ACUTE MYOCARDIAL INFARCTION

Purpose of Research

SERUM MAGNESIUM LEVELS IN ACUTE MYOCARDIAL INFARCTION

Procedures involved

. It includes details like age, sex and about history of the problem and associated risk factors as well as clinical examination.

Investigations includes complete hemogram, renal function tests(blood urea, serum creatinine), Serum magnesium ,ECG .

Decline from Participation

You have the option to decline from participation in the study existing protocol for your condition.

Privacy and Confidentiality

Privacy of individuals will be respected and any information about you or provided by you during the study will be kept strictly confidential.

Authorization to publish Results

Results of the study may be published for scientific purposes and/or presented to scientific groups, however you will not be identified.

Statement of Consent

I volunteer and consent to participate in this study. I have read the consent or it has been read to me. The study has been fully explained to me, and I may ask questions at any time.

---------- ----------

Signature /Left thumb impression (volunteer) Date

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Signature of witness Date

ஒப்புதல் படிவம்

பெயர் :

பாலினம் :

வயது :

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் பொது மருத்துவத் துறையில் பட்ட மேற்படிப்பு பயிலும் மாணவர் மேற்கொள்ளும் **"கடுமையான மாரடைப்பின் போது இரத்தத்தின் மெக்னீசியம் அளவு"** குறித்த ஆய்வில் செய்முறை மற்றும் அனைத்து விவரங்களையும் கேட்டுக் கொண்டு எனது சந்தேகங்களை தெளிவுப்படுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடனும், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னுடைய அனைத்து விபரங்கள் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபனை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் இந்த ஆய்விலிருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இடம் : கையொப்பம் / ரேகை

நாள் :

KEY TO MASTER CHART

CVS.....Cardiovascular system

RS.....Respiratory system

mm of Hg .....Millimeter of mercury

BP.....Blood pressure

Mg^{2+} .....Magnesium

Na^{+} .....Sodium

K^{+} .....Potassium

mg/dL.....Milligram/ deciliter

meq/l .....Milliequivalent liter

VPC……Ventricular Premature contractions

VT .....Ventricular tachycardia

ECG.....Electrocardiogram

CCF .....Congestive cardiac failure

MASTER CHART

Sl. No.	IP No.	Age	Sex	Symptoms	Signs BP in mm HG	ECG dignosis	Days	Serum			Complications
								Mg2+(mg/dL)	Na(meq/L)	K+(meq/L)	
1	52369	40	M	Chest pain -6hrs	BP=130/90 CVS:S1 S2+	Antero Septal MI	1 5	1.7 2.0	138 136	4.4 4.0	recovered
2	52584	60	F	chest pain-3hrs dyspnoea-3hrs	BP=100/70 CVS:S1 S2+	Acute Anterior MI	1 5	1.7 2.2	138 134	5.0 4.8	occasional VPCs recovered
3	52869	60	M	chest pain-3hrs	BP=180/100 CVS :S1S2+	ant. wall MI	1 5	2.0 -	141 -	5.2 -	Patient died of VT
4	52964	45	M	chest pain sweating: 3hrs	BP=180/120CVS:S1 S2+	Ant.septal MI	1 5	1.1 2.2	138 136	4.4 4.0	Occasional VPC recovered
5	53008	56	M	chest pain: 1hrs	BP=70/50CVS:S1 S2+HEART SOUNDS MUFFLED	Inf.wall MI	1 5	1.55	138 -	4.4 -	cardiogenic shock expired on 3rd day
6	55956	35	M	chest pain:-2hrs	BP=110/70CVS:S1 S2+	accute Antiwall MI	1 5	2.6 3.3	140 142	4 4.2	Patient recovered
7	55972	40	M	chest pain-4hrs	BP=120/80 CVS:S1 S2+	Anti wall MI	1 5	1.2 1.5	140 145	4.2 3.8	occasional VPCs recovered
8	56688	30	M	chest pain-7hrs sweating-5hrs	BP=120/80 CVS:S1 S2+	Anti wall MI	1 5	1.9 2.2	136 140	3.4 3.6	occasional VPCs recovered
9	56883	38	M	chest pain-1hrs sweating-1hrs	BP=130/90CVS:S1 S2+	Inf.wall MI	1 5	1.61 1.78	140 145	4.2 3.8	occasional VPCs recovered
10	56541	40	M	chest pain-3hrs sweating-3hrs	BP=130/90CVS:S1 S2+	ant.hat wall MI	1 5	2.1 2.9	139 140	3.8 3.6	Recovered
11	56854	62	M	chest pain, Breath lessness.3hrs	BP=160/100CVS:S1S2+	Ant septal MI	1 5	1.85 1.93	140 145	3.8 3.6	occasional VPCs recovered
12	56512	45	M	chest pain-4hrs	BP=140/90CVS:S1 S2+	Inf.wall MI	1 5	2.0 2.2	140 142	5.0 4.5	Recovered

Sl. No.	IP No.	Age	Sex	Symptoms	Signs BP in mm HG	ECG dignosis	Days	Serum			Complications
								Mg2+(mg/dL)	Na (meq/L)	K+(meq/L)	
13	56598	65	M	chest pain-4hrs	BP=160/100CVS:S1S2+	Inf.wall MI	1	2.4	137	5	Recovered
							5	3.2	135	4.5	
14	56749	45	M	chest pain-4hrs	BP=160/80CVS:S1 S2+	Inf.wall MI	1	2.8	136	4.2	Recovered
							5	3.0	138	4.8	
15	56626	53	M	chest pain: 6hrs	BP=190/110CVS:S1 S2+	Ant.wall	1	2.0	137	5	Recovered
						MI	5	2.01	135	4.5	
16	51652	60	F	chest pain :8hrs	BP=190/110CVS:S1 S2+	Ant.wall	1	2.4	138	4.1	Recovered
						MI	5	3.2	140	3.2	
17	61648	69	M	chest pain :8hrs	BP=200/60CVS:S1 S2+	Ant.wall	1	2.2	134	4.7	Recovered
						MI	5	2.4	136	4.2	
	57554			chest pain:6hrs sweating:6hrs			1	1.70	136	4.2	occasional VPCs
							5	1.92	138	4.8	recovered
18		45	F		BP=70/40CVS :S1 S2	Inf wall MI					
19	57235	50	M	chest pain:6hrs	BP=130/70CVS:S1 S2+	Ant.wall	1	1.9	138	4.5	occasional VPCs
						MI	5	2.9	136	4.2	recovered
20	57277	70	M	chest pain: 4hrs sweating:4hrs	BP=70/40CVS:S1 S2+	Inf wall MI	1	1.2	137	4.0	Recovered, occasional VPC
							5	1.6	138	3.8	
21	57249	65	M	chest pain & breathlessness-8hrs	BP=130/90CVS:S1 S2+	Antero Septal MI	1	1.5	136	3.8	Died of VT
							5				
22	54675	82	M	chest pain -5hrs breathlessness:6hrS	BP=130/80CVS:S1 S2+	Ant.wall	1	1.4	136	5.8	occasional VPCs
						MI	5	2.0	138	4.2	recovered
23	52382	67	M	chest pain -3hrs	BP=150/100CVS:S1 S2+	Inf wall	1	1.9	135	4.9	Recovered
						MI	5	2.3	138	4.3	
24	57496	45	M	chest pain &breathness -1hrs	BP=110/80CVS:S1 S2+	Inf wall MI	1	1.58	136	3.8	Died of VT
							5				
25	52491	65	F	chest pain-6hrs	BP=100/70CVS:S1 S2+	non ST	1	2.3	136	3.9	occasional VPCs
						elevation MI	5	2.5	138	3.6	recovered

Sl. No.	IP No.	Age	Sex	Symptoms	Signs BP in mm HG	ECG dignosis	Days	Serum			Complications
								Mg^{2+}(mg/dL)	Na(meq/L)	K+(meq/L)	
26	57682	65	M	chest pain &sweating-8hrs	BP=80/60CVS:S1 S2+	INf wall MI	1 5	1.70 1.86	143 139	3.9 4.2	occasional VPCs recovered
27	57703	39	M	chest pain &sweating-3hrs	BP=140/100CVS:S1 S2+	Ant septal MI	1 5	1.85 1.96	140 138	5.0 5.2	occasional VPCs recovered
28	51942	70	M	chest pain-1hrsdyspnoea 1hrs	BP=180/100 CVS :S1S2+	Ant wall MI	1 5	2.3 2.5	139 138	4.3 4.0	Recovered
29	57696	43	M	chest pain sweating:-7hrs	BP=110/70CVS:S1 S2+	extansive Ant wall MI	1 5	1.68 1.72	139 142	5.1 4.9	occasional VPCs recovered
30	52711	30	M	chest pain.2hrs	BP=120/80 CVS:S1 S2+	Inf wall MI	1 5	1.9 2.4	140 142	3.8 3.8	Recovered
31	57733	58	M	chest pain & breathlessness-8hrs	BP=110/70CVS:S1 S2+	Ant septal MI	1 5	1.70 1.92	140 143	4.8 5.0	Occasional VPC, recovered
32	51686	65	F	chest pain:6hrs	BP=140/90CVS:S1 S2+	Ant wall MI	1 5	2.6 3.0	141 138	3.4 3.2	Recovered
33	57786	63	M	chest pain :4hrs	BP=100/60CVS:S1 S2+	Ant septal MI	1 5	1.21	139	4.5	Died of VT
34	57852	48	M	chest pain :8hrs	BP=100/70CVS:S1 S2+	truk post MI	1 5	1.75 1.76	140 142	3.8 4.0	RBBB recovered
35	60119	45	M	chest pain; 2hrs	BP=100/70CVS:S1 S2+	Ant wall MI	1 5	2.1 2.2	140 142	3.8 3.6	Recovered
36	58214	52	M	chest pain :10hrs	BP=90/60CVS:S1 S2+,S3+	Inf wall MI	1 5	1.68 1.82	141 139	5.1 4.8	occasional VPCs recovered
37	58374	46	M	chest pain -11hrs palpitation-11hrs	BP=90/60CVS:S1 S2+,S3+	Ant wall MI	1 5	1.75 1.92	135 138	4.7 5.2	LVF recovered
38	50130	50	M	chest pain-6hrs	BP=140/90CVS:S1 S2+	Inf wall MI	1 5	2.4 3.0	138 137	4.2 4.0	Recovered
39	59147	65	M	chest pain sweating &mi 2hrs	BP=150/100CVS:S1 S2+,S3+,RS BASAL CREPITATIONS+	Ant lat MI	1 5	1.1 -	137	4.5	Cardiogenic shock, expires on 2nd day

Sl. No.	IP No.	Age	Sex	Symptoms	Signs BP in mm HG	ECG dignosis	Days	Serum			Complications
								Mg^{2+}(mg/dL)	Na(meq/L)	K+(meq/L)	
40	59268	62	M	chest pain-5hrs	BP=140/90CVS:S1 S2+	Inf wall MI	1 5	2.07 2.04	132 140	3.5 5.0	recovered
41	58938	56	F	chest pain-4hrs	BP=130/80CVS:S1 S2+	Inf wall MI	1 5	1.7 1.92	137 142	5 3.5	occasional VPCs recovered
42	61984	85	M	chest pain breathlessness:-6hrs	BP=200/120CVS:S1 S2+	Inf wall MI	1 5	1.64	140	4.2	VT expired on 2nd day
43	54917	50	M	chest pain -3hrs	BP=120/80 CVS:S1 S2+	Ant wall MI	1 5	2.5 3.0	132 138	4.2 4.0	Recovered
44	59079	45	F	chest pain sweating:-7hrs	BP=130/80CVS:S1 S2+	Antero Septal MI	1 5	1.70 1.86	137 135	5.1 5.2	Recovered
45	59729	39	M	chest pain -3hrs	BP=140/70CVS:S1 S2+	Ant wall MI	1 5	1.81 1.82	142 140	4.0 3.9	Recovered
46	59943	49	M	chest pain sweating;-4hrs	BP=110/70CVS:S1 S2+	Antero Septal MI	1 5	1.70 1.89	135 140	3.8 3.3	Recovered, occasional VPC
47	70291	40	M	chest pain:6hrs	BP=110/70CVS:S1 S2+	Ant wall MI	1 5	2.8 3.2	138 136	4.0 3.8	Recovered
48	60423	35	M	chest pain sweating:6hrs	BP=110/90CVS:S1 S2+	Antero Septal MI	1 5	1.67 2.35	140 135	3.5 4.0	occasional VPCs recovered
49	60089	42	M	chest pain :4hrs	BP=190/110CVS:S1 S2+	INf wall MI	1 5	1.7 1.92	142 140	3.9 4.2	Recovered
50	54917	50	M	chest pain:8hrs	BP=120/80 CVS:S1 S2+	Ant wall MI	1 5	1.5 2.3	137 138	4.2 4.0	occasional VPCs recovered