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A Study on Serum Magnesium Levels in Acute Myocardial Infarction

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Abstract

Background: The proportion of myocardial infarction hospitalization has increased in recent years. Most common cause of death in MI is arrhythmia – VF. Magnesium has been associated with pathogenesis of myocardial infarction. The cardiological consequence of magnesium deficiency include multifocal necrosis with calcium accumulation in mitochondria in a pattern reminiscent of myocardial ischemia and catecholamine induced cardiomyopathy, atherogenesis and increased tendency to platelet aggregation, coronary vascular resistance, peripheral vascular resistance, repolarisation abnormalities and ventricular tachyarrhythmias

Objective: To know the relation between level of serum magnesium and arrhythmia in patients with acute myocardial infarction who are presenting within 12 hours of onset.

Method: By using prospective observational study 100 patients of acute myocardial infarction admitted to SRI Ramakrishna Hospital, Coimbatore over period of 12 months between Dec. 2020 to Dec. 2021patient selected in simple random method.

Results: With help of the study we found that there is significant association between magnesium levels and arrhythmia.

Conclusion: Patient with low magnesium levels in acute myocardial infarction are more prone to get arrhythmias. So careful monitoring of magnesium and correction of magnesium level to be done if found deficit.

Keywords: Myocardial Infarction, Magnesium, Arrythmia.

Introduction

Acute myocardial infarctions (AMIs) are a subset of the acute coronary syndrome (ACS), a grouping that also includes unstable angina (UA), AMI with or without ST

elevation, and other conditions. A typical rise and fall in the level of biochemical markers of myocardial necrosis along with at least one of the following—ischemic symptoms, EKG changes—confirms the diagnosis of AMI.[1]The proportion of acute myocardial infarction (AMI) hospitalizations involving young patients has grown over the past 20 years, most noticeably among women. This pattern coincides with an increase in cardio-vascular risk factors among young patients hospitalized with acute myocardial infarction, such as hypertension and diabetes mellitus.[2]

Myocardial perfusion is reduced to a level sufficient to result in cell necrosis, which leads to an AMI. This is most commonly caused by the formation of a thrombus in a coronary artery. An atherosclerotic plaque rupture or fissure is what triggers the inciting event, exposing the blood to thrombogenic lipids and activating platelet and clotting factors. The coronary plaques that are most likely to rupture have a thick fibrous cap and a rich lipid core. Other uncommon causes of a myocardial infarct include cocaine use, coronary artery dissection, hypotension, anemia, and coronary artery embolism from a valvular vegetation or intracardiac thrombi.[3]

Three categories can be used to categories risk factors for MI: Age, gender, and family history are non-modifiable risk factors. Modifiable risk factors include alcohol, smoking, restricted physical activity, uncontrolled hypertension, diabetes, and dyslipidemias. Emerging risk factors include C-reactive protein (CRP), fibrinogen, coronary artery calcification (CAC), homocysteine, lipoprotein(a), and small, dense (LDL). The major risk factors were used by the Framingham Heart Study to develop a coronary risk estimate that calculated a person's 10-year cardiovascular risk.[4]

The primary goal of AMI prevention is to lower the risk factors that can be changed. Goals include making lifestyle changes. Additionally, advised are drugs that lower cholesterol and control blood pressure.[5]

Due to the involvement of a neural reflex pathway via the thoracic and cervical nerves, patients with AMI typically present with chest pain. It is a severe, visceral pain that is frequently described as being heavy, tight, crushing, and occasionally stabbing or burning. It typically arises from the substernal region and may extend to the corresponding dermatomes (C7-T4) that supply afferent nerves to the same spinal cord segments as the heart. The epigastric, shoulders, arms, back (interscapular region), lower jaw, and neck are among them. Acute myocardial infarction is better predicted by radiation to both arms.[6]

Heart disease is the leading cause of death among adult males. Ischemic heart disease, which was anticipated to be the leading cause of death worldwide, killed more than six million people in recent years.[7] A study showed that men are more likely than women to suffer from acute myocardial infarction (48 percent of cases were in the 50 to 59 age range, and 76 percent of the cases were male). Heart disease risk factors for coronary artery disease are both numerous and non-modifiable. Many risk factors for the development of coronary, peripheral, and cerebrovascular disease have been identified through experimental animal studies, epidemiological studies, and clinical interventional trials. Risk factors have a multiplicative rather than an additive effect.[8] Magnesium's role in cardiovascular disease has drawn a lot of attention. Arrhythmias and hypomagnesemia have a well-established connection. Additionally, a number of researchers have identified a link between a lack of magnesium and coronary artery disease.[9]

Magnesium (atomic number 12, atomic mass 24.30 Da) is a member of the second group of the periodic table of elements and is categorized as an alkaline earth metal. Due to its high reactivity, magnesium frequently occurs as the free cation Mg2+ in aqueous solution or as the mineral component of a wide range of compounds, such as chlorides, carbonates, and hydroxides, rather than in a native metallic state. It has the same oxidation state as calcium, which is 2+.[10]Magnesium (Mg2+) plays a significant role in the human body. The second-most abundant intracellular cation after potassium is magnesium, which is the body's fourth-most abundant cation.[11] It functions as a cofactor for over 300 enzymes, controlling a variety of fundamental functions including muscle contraction, neuromuscular conduction, glycemic control, myocardial contraction, and blood pressure.[12]

According to observational studies, high circulating magnesium levels and magnesium intake is associated with a modest reduction in the risk of cardiovascular disease, including coronary heart disease.[32,33]Still, it is unclear what causes these associations. The other potentially cardioprotective nutrients could explain the inverse relationship between magnesium and cardiovascular disease in foods high in magnesium or the dietary habits of those who consume these foods. Green leafy vegetables, legumes, nuts, seeds, avocados, dark chocolate, whole grains, yogurt, and fish are some foods high in magnesium. Magnesium intake from a typical Western diet is thought to be frequently insufficient.[34]

Many such studies have found a link between Mg and the progression of coronary artery disease (CAD). According to data from the National Health and Nutrition Examination Survey Epidemiologic Follow-up Study (NHANES), serum Mg levels were inversely related to cardiovascular deaths and hospitalizations.[37,38] The link between hypomagnesemia and arrhythmias is well established. Numerous studies have found a link between magnesium deficiency and coronary artery disease.[8,9]

Magnesium improves myocardial metabolism and prevents calcium build-up and cell death. It improves vascular tone, peripheral vascular resistance, afterload, and cardiac output, as well as lowering cardiac arrhythmias and improving lipid metabolism. Magnesium also improves endothelial function and inhibits platelet function, including aggregation and adhesion.[39] Myocardial magnesium concentrations were found to be very low in patients who died suddenly from ischemic heart disease.[40] The use of magnesium to reduce infarct size has significant research and clinical implications.[41]

Hypomagnesemia is a major risk factor for post-acute MI complications. Several international studies have found that the serum Mg level in cases of AMI is not only low at admission but also continues to fall for days after exposure of AMI.[42,43]It is unclear, however, whether the low cardiac content precedes or results from the myocardial infarction. Acute myocardial infarction (AMI) causes hypomagnesemia because magnesium moves from extracellular to intracellular compartments.[44] Several clinical studies have found a decrease in serum magnesium concentrations during the first 24 to 48 hours after a myocardial infarction.[45] One Dhaka study found that AMI has significantly lower serum Mg and K levels than chronic IHD, and the drop in serum Mg immediately after AMI may be due to catecholamine-induced high FFA. It causes bindings and precipitation of Mg into the cells, resulting in a sudden decrease in total plasma Mg level.[46] By means of pumps,

carriers, and channels, magnesium modifies ion transport. It interferes with sodium/potassium ATPase (NA+/K+ ATPase) and serum calcium function.

Causes of hypomagnesemia can be categorized into genetic causes and acquired causes.[47] The acquired causes can be attributed to decreased oral intake or GI absorption, increased renal loss, or redistribution triggered by severe illness.[13] Several medications are also known to influence serum magnesium levels by different mechanisms.[35,48] Several dietary surveys have shown that people in North America and Europe consume less than recommended daily allowance (RDA) for magnesium as a result of food processing and the use of poor soil for agriculture.[13,18,36] Hypomagnesemia can also occur in times of prolonged fasting, total parenteral nutrition, or prolonged nasogastric suctioning.[49] Impaired gastro-intestinal absorption of magnesium can be caused by a number of factors including chronic diarrhoea, pancreatic insufficiency, celiac disease, chronic alcoholism, inflammatory bowel diseases, and short gut syndrome.[50]

Additionally, it has been discovered that hypomagnesaemia raises the possibility of complications and cerebrovascular events. When the endothelium was intact, Szabo et al. discovered that a slight reduction in extracellular magnesium from 1.2 to 0.8 mM caused a sustained relaxation; however, when the endothelium was disrupted, the slight magnesium reduction caused an increase in vascular tone. Magnesium modifies smooth muscle tone indirectly, rather than directly, by modulating an endothelium-derived relaxing factor, and that magnesium deficiency appears to promote endothelial dysfunction and, consequently, atherosclerosis.[55]

Symptoms of magnesium deficiency can be nonspecific and usually overlap with symptoms of other electrolyte imbalances. The severity of symptoms and signs depends on the degree of magnesium depletion and rate of magnesium decline. The symptoms usually occur when serum magnesium levels fall below 0.5 mmol/L (1.2 mg/dL).[50] The clinical manifestations of hypomagnesemia may affect every system including neuromuscular, cardiovascular, renal, and gastrointestinal systems.[56,57]

Recent studies show that patients with AMI have lower intracellular magnesium levels. Since magnesium is primarily an intracellular ion and less than 1% of the total body's magnesium is found in the intravascular compartment, serum measured values do not adequately reflect this deficiency.[60] Additionally, no discernible change in serum magnesium was found in some studies. The importance of magnesium in cardiac disease has been discussed in a number of reviews over the past ten years. However, most doctors do not fully understand the qualitative and quantitative contributions of magnesium.

There are two important issues that clinicians should deal with when treating young women with AMI. The strategy of urgent care, which includes percutaneous coronary intervention, comes first (PCI). According to Nakashima et al., among 130 women with AMI who were 50 years of age, atherosclerotic plaque was discovered in 55 patients (42%), and spontaneous coronary artery dissection was discovered in 45 patients (35%).[61] Both of these etiology require different approaches to treatment. When a patient has atherosclerotic plaque, PCI may be advised; however, when a patient has spontaneous coronary artery dissection, conservative therapy may be used first, followed by PCI. Therefore, coronary angiography and

intravascular ultrasound should be used to thoroughly assess the target lesion.

Metallic coronary stents should be implanted as infrequently as possible during PCI because they may interfere with future surgical procedures or pregnancy in the patient. An implanted bare-metal stent was used in this specific instance. But compared to bare-metal stents, second- or third-generation drug-eluting stents (DESs) have produced better results, including lower rates of stent thrombosis. A balloon-alone PCI strategy using a balloon with a drug coating may be the best course of action (DCB). Due to the fact that DCB leaves no metallic mesh, non-stent-based local drug delivery was investigated.[62]In addition to leaving no metallic mesh, DCBs have many advantages over DESs, including ensuring homogeneous drug distribution, encouraging positive vessel remodelling, and possibly necessitating a shorter course of dual antiplatelet therapy.

Magnesium intravenously has pharmacological effects that have been found to be helpful in arrhythmia. The aggregation of thrombosis may be reduced by magnesium and potassium therapy. Magnesium also relaxes the blood vessels and enhances muscle contraction. Magnesium therapy for patients with ischaemic heart disease significantly lowers the risk of AMI-related mortality.[63]

Altering dietary habits to include more magnesium-rich foods, putting magnesium in community water supplies, fortifying foods with magnesium, and oral supplementation are all ways to increase magnesium as a potential way to lower sudden cardiac death. The effectiveness of magnesium supplementation as a primary preventive measure for sudden cardiac death needs to be further investigated in prospective, large-scale studies.

Materials and Methods

Study Area: Sri Ramakrishna hospital

Study Population: Patients diagnosed of having acute myocardial infarction, admitted to Sri Ramakrishna hospital.

Study Design: Prospective observational study

Study Duration: 12 months (Dec2020- Dec2021)

Sample Size Calculation Formula

$$n = \frac{Z_{1-\alpha/2}^2 * p\,(1-p)}{d^2}$$

Description

n = required samples sizes

Z= confidence level of 95%(standard value of 1.96)

p = Expected frequency of the factor under study – 6.6%

d = margin of error of 5%(standard value of 0.05)

$$n = \frac{1.962x\ 0.\text{-}66\ (\ 1 - 0.066\ )}{0.52}$$

$$n = 95$$

Contingency

The sample is further increased by 5% to account for contingencies such as non – responsive or recording error

n + 5% =95 +5% = 99 samples

Round off: 100 samples

Sample Size: 100

Method of Collection of Data

After getting consent from the patient and fulfilling the above criteria these patients data are collected and documented

Inclusion Criteria

Patients who presented to the hospital within 12 hours of onset of symptoms were included in the study. The following criteria have been used to diagnose acute myocardial infarction. The presence of any of the two criteria has been considered:

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

Exclusion Criteria

Patients with Hypokalaemia

Results and Analysis

The data collected were subjected to Statistical Analysis using SPSS version 16. Descriptive Statistics, Frequency analysis, One-way ANOVA and Independent Sample‘t’ tests were performed for appropriate variables. The probability value, p was defined as 0.05 for all the significance tests. A ‘p’ value less than 0.05 is considered significant and a value less than 0.01 is considered as highly significant. The results of the Statistical analysis are presented in subsequent tables.

Table 1: Age wise Distribution of the Patients

Age in Years	Frequency	Percent	Cumulative Percent
<60	29	29.0	29.0
60-70	25	25.0	54.0
>70	46	46.0	100.0
Total	100	100.0	

Table 1: Presents the distribution of patients based on age. It can be inferred from the table that 29% of the patients are below 60 years of age, 25% are between 60 and 70 years and 46% are above 70 years of age.

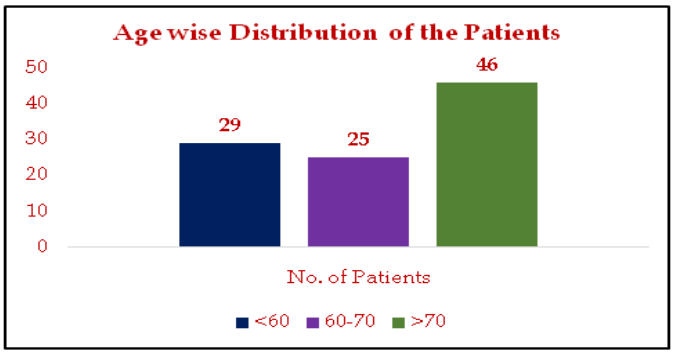

Fig. 1: Age wise distribution of the Patients

Table 2: Gender wise Distribution of the Patients

Gender	Frequency	Percent	Cumulative Percent
Male	67	67.0	67.0
Female	33	33.0	100.0
Total	100	100.0	

Table 2: Shows the distribution of patients based on gender. It can be depicted from the table that 67% of the patients are male and 33% are female.

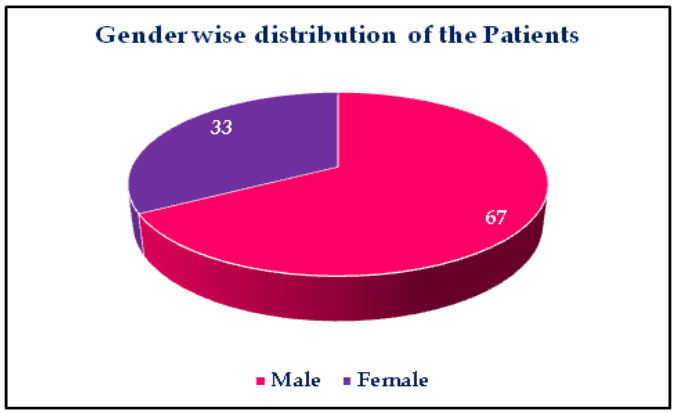

Fig. 2: Gender wise distribution of the Patients

Table 3: Symptom wise Distribution of the Patients

Symptoms	Frequency	Percent	Cumulative Percent
Chest Pain	100	100.0	100.0

Table 3: Shows that all the patients (100%) were admitted due to Chest pain.

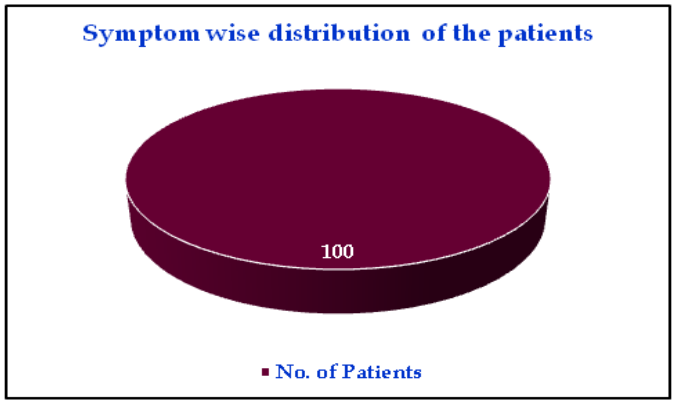

Fig. 3: Symptom wise distribution of the Patients

Table 4: Distribution of the Patients based on other symptoms

Other Symptoms	Frequency	Percent	Cumulative Percent
Sweating	42	42.0	42.0
Breathlessness	38	38.0	80.0
Palpitation	11	11.0	91.0
No other Symptom	9	9.0	100.0
Total	100	100.0	

Table 4: Displays the other symptoms experienced by the patients along with chest pain. It is clear from the table that 42% of the patients had sweating, 38% had Breathlessness, 11% had Palpitation and 9% did not have any other symptom than chest pain.

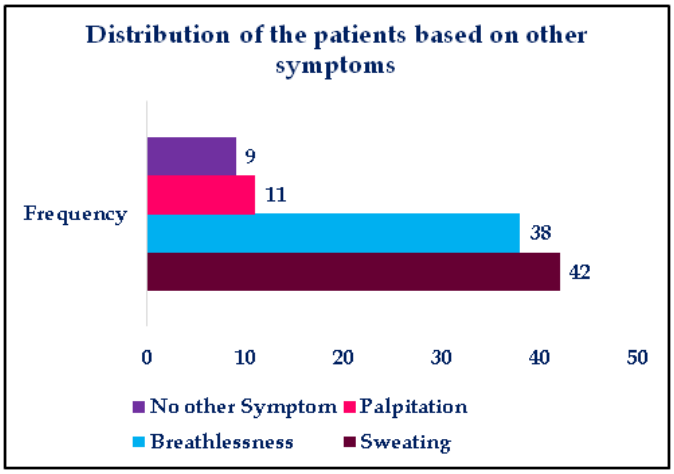

Fig. 4: Distribution of the Patients based on other symptoms

Table 5: Distribution of the Patients based on their diet

Type of Diet	Frequency	Percent	Cumulative Percent
Vegetarian	22	22.0	22.0
Mixed	78	78.0	100.0
Total	100	100.0	

Table 5: Portrays the distribution of patients based on diet followed by them. It can be inferred from the table that 78% of the patients follow mixed diet and 22% are vegetarians.

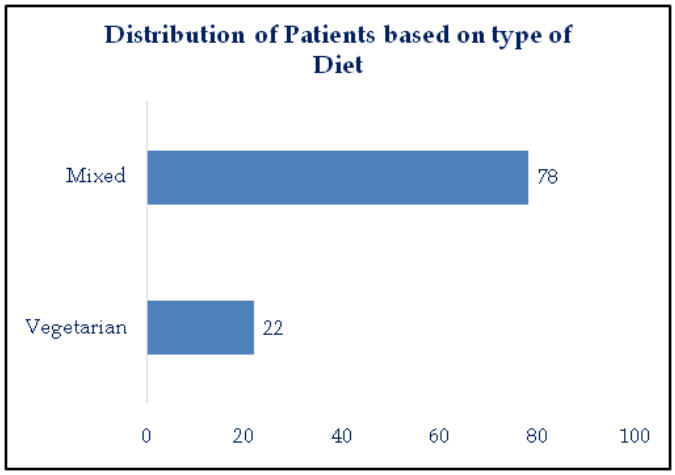

Fig. 5: Distribution of the Patients based on type of diet

Table 6: Distribution of the Patients based on smoking Habit

Smoking Habit	Frequency	Percent	Cumulative Percent
Yes	41	41.0	41.0
No	59	59.0	100.0
Total	100	100.0	

Table 6: Displays the distribution of patients based on their smoking habit. Majority of the patients (59%) are non-smokers and the remaining 41% are smokers. Smoking is a risk factor of Myocardial Infarction.

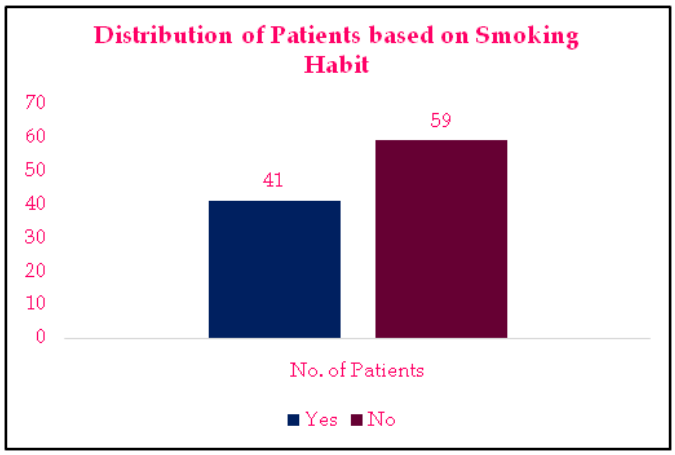

Fig. 6: Distribution of the Patients based on Smoking Habit

Table 7: Distribution of the Patients based on Family history of Diseases

Family History of Diseases	Frequency	Percent	Cumulative Percent
Yes	24	24.0	24.0
No	76	76.0	100.0
Total	100	100.0	

Table 7: Shows the distribution of the patients based on their family history for diseases such as HTN, IHD, CVA and DM. Majority of the patients (76%) do not have family history for such diseases and the remaining 24% show family history of diseases.

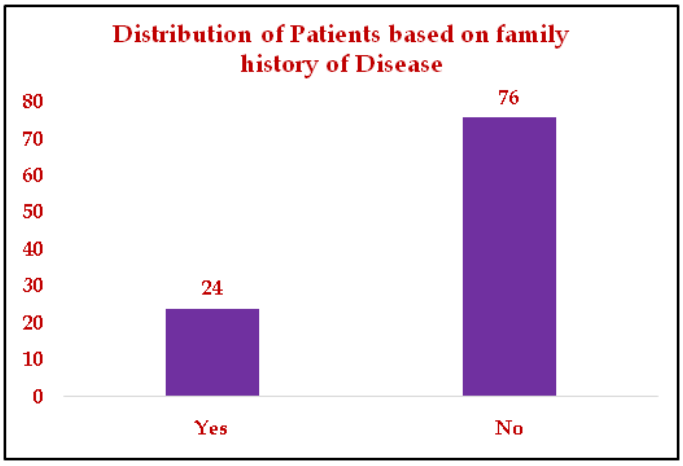

Fig. 7: Distribution of the Patients based on family history of Disease

Table 8: Distribution of the Patients based on Obesity

Obesity	Frequency	Percent	Cumulative Percent
Yes	32	32.0	32.0
No	68	68.0	100.0
Total	100	100.0	

Table 8: Displays the distribution of patients based on Obesity. Majority of the patients (68%) are not obese and the remaining 32% are obese.

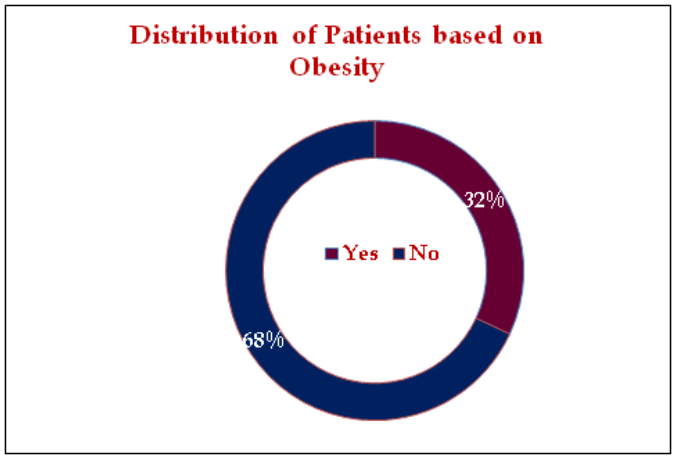

Fig. 8: Distribution of the Patients based on Obesity

Table 9: Distribution of the Patients based on presence of Diabetes Mellitus

Presence of Diabetes Mellitus	Frequency	Percent	Cumulative Percent
Yes	40	40.0	40.0
No	60	60.0	100.0
Total	100	100.0	

Table 9: Portrays the distribution of patients based on prevalence of Diabetes Mellitus. It can be inferred from the table that majority of the patients (60%) do not have Diabetes Mellitus and the remaining 40% have Diabetes mellitus.

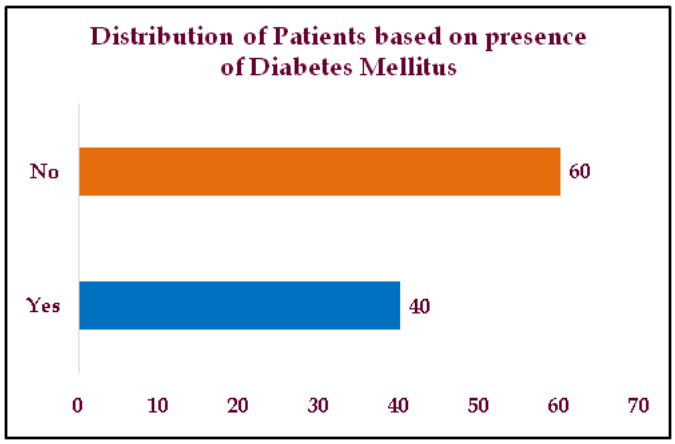

Fig. 9: Distribution of the Patients based on presence of Diabetes mellitus

Table 10: Distribution of the Patients based on presence of Dyslipidaemia

Presence of Dyslipidaemia	Frequency	Percent	Cumulative Percent
Yes	17	17.0	17.0
No	83	83.0	100.0
Total	100	100.0	

Table 10: Shows the distribution of patients based on prevalence of Dyslipidaemia. It is clear from the table that 83% of the patients do not have dyslipidaemia and 17% have dyslipidaemia.

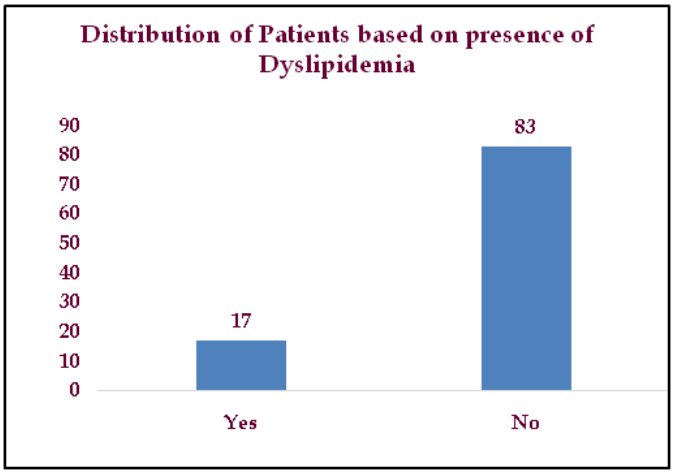

Fig. 10: Distribution of the Patients based on presence of Dyslipidaemia

Table11: Distribution of the Patients based on presence of Hypertension

Presence of Hypertension	Frequency	Percent	Cumulative Percent
Yes	42	42.0	42.0
No	58	58.0	100.0
Total	100	100.0	

Table 11: Shows the distribution of patients based on prevalence of Hypertension. It can be understood from the table that majority of the patients (58%) do not have hypertension and only 42% have Hypertension.

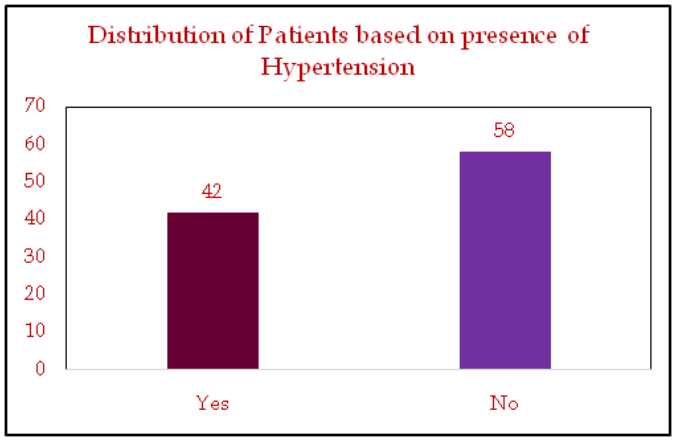

Fig. 11: Distribution of the Patients based on presence of Hypertension

Table 12: Distribution of the Patients based on the complication

Complication	Frequency	Percent	Cumulative Percent
SVT Recovered	7	7.0	7.0
No complication	54	54.0	61.0
VPCs Deceased	9	9.0	70.0
VT Deceased	14	14.0	84.0
VPCs Recovered	8	8.0	92.0
LVF Recovered	3	3.0	95.0
Cardiogenic Shock	5	5.0	100.0
Total	100	100.0	

Table 12: Shows the distribution of the patients based on the complication experienced by them. It is clear from the table that 54% had no complication, 8% have recovered from VPC, 3% have recovered from LVF, 5% have recovered from cardiogenic shock and 7% have recovered from SVT. However, 14% died of VT and 9% died of VPC.

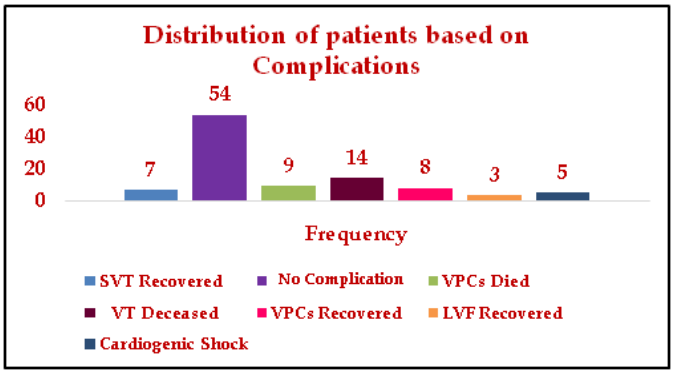

Fig.12: Distribution of the Patients based on Complications

Table 13: Magnesium levels on Day 1 and Day 5

Magnesium level (mg/dL)	N	Minimum	Maximum	Mean	Std. Deviation
Day 1	100	1.22	2.59	1.86	0.370
Day 5	78	1.72	2.89	2.29	0.210

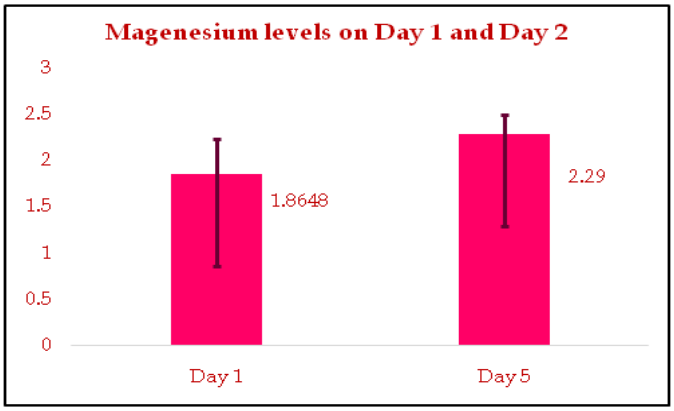

Fig. 13: Magnesium levels on Day 1 and Day 5

The mean Magnesium level in the 100 patients on Day 1 was 1.86 ± 0.370 mg/dl as depicted in table 4.13. Similarly, the magnesium level on Day 5 was observed only in 78 patients and the mean was 2.29±0.210 mg/dL.

Table 14: Magnesium levels in patients with different Complications on Day 1

Complication	N	Mean	Std. Deviation	ANOVA 'F' Value (Significance 'p' value)
SVT Recovered	7	1.81	0.23	27.304 p<0.01 Highly Significant
No complication	54	2.09	0.26	
VPCs Deceased	9	1.39	0.07	
VT Deceased	14	1.34	0.09	
VPCs Recovered	8	1.78	0.26	
LVF Recovered	3	1.96	0.15	
Cardiogenic Shock	5	1.86	0.26	
Total	100	1.86	0.37	

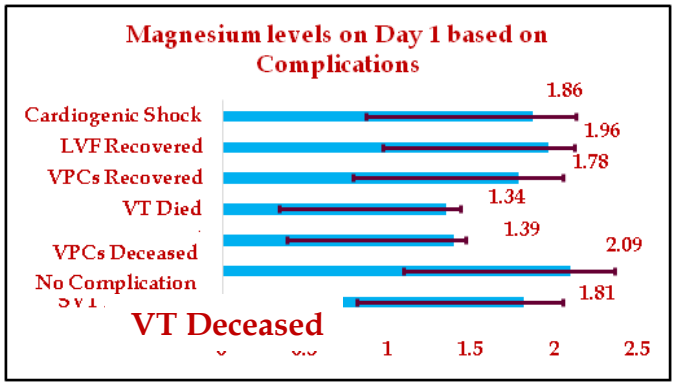

Fig.14: Magnesium levels in patients with different Complications on Day 1

Table 14: Shows the Magnesium level on Day 1 in patients based on the Complications seen in them. The mean Mg level in SVT recovered patients was 1.81±0.23 mg/dL, in patients with no complication was 2.09±0.26 mg/dL, VPC recovered patients were 1.78±0.26, LVF recovered patients were 1.96±0.15 mg/dL and Cardiogenic shock recovered patients were 1.86±0.26. Magnesium level was very low in the patients who died. The mean MG level was 1.39±0.07 mg/dL in VPC deceased patients on Day 1 and 1.34±0.09 mg/dL in patients who died of VT. There is a highly significant association between Magnesium level on day 1 and Complications involved as depicted by the highly significant 't' value of 27.304 (p<0.01).

Table 15: Magnesium levels in patients with different Complications on Day 5

Complication	N	Mean	Std. Deviation	ANOVA 'F' Value (Significance 'p' value)
SVT Recovered	7	2.15	0.18	90.628 p<0.01 Highly Significant
No complication	54	2.35	0.19	
VPCs Deceased	9	0.00	0.00	
VT Deceased	14	0.00	0.00	
VPCs Recovered	8	2.11	0.24	
LVF Recovered	3	2.24	0.08	
Cardiogenic Shock	5	2.17	0.12	
Total	100	1.82	0.93	

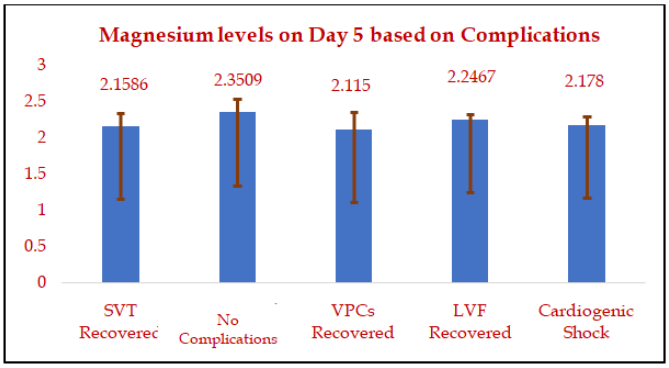

Fig.15: Magnesium levels in patients with different Complications on Day 5

Table 15: Shows the Magnesium level on Day 5 in patients based on the Complications seen in them. The mean Mg level in SVT recovered patients was 2.15±0.18 mg/dL, in patients with no complication was 2.35±0.19 mg/dL, VPC recovered patients were 2.11±0.24, LVF recovered patients were 2.24±0.08 mg/dL and Cardiogenic shock recovered patients were 2.17±0.12. In the patients who died, Magnesium level on Day 5 could not be recorded. There is a highly significant association between Magnesium level on day 5 and Complications involved as depicted by the highly significant 't' value of 90.628 (p<0.01).

Table 16: Magnesium levels on Day 1 based on age group

Age in years	N	Mean	Std. Deviation	ANOVA 'F' Value (Significance 'p' value)
<60	29	1.90	0.42052	1.709 p>0.05 Not Significant
60-70	25	1.74	0.33032	
>70	46	1.90	0.35050	

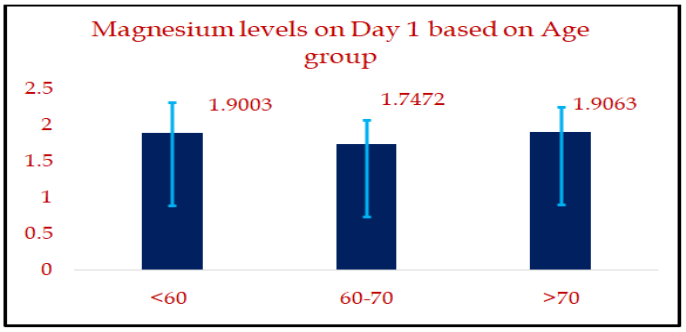

Fig.16: Magnesium levels on Day 1 based on age group

Table 16: Displays the Magnesium levels in patients on Day 1 based on age group. It is clear from the table that the mean Mg level in patients below 60 years was 1.90±0.42052 mg/dL, for patients between 60 and 70 years of age was 1.74±0.33032 mg/dL and for patients above 70 years it was 1.90±0.35050 mg/dL. The minimum Magnesium level is seen in patients between 60 and 70 years of age. However, there is no significant association between age and Magnesium level on Day 1 as depicted by the insignificant 'F' value of 1.709 (p>0.05).

Table 17: Magnesium levels on Day 5 based on age group

Age in years	N	Mean	Std. Deviation	ANOVA 'F' Value (Significance 'p' value)
<60	29	1.8903	0.90206	1.248
60-70	25	1.5712	1.00861	p>0.05
>70	46	1.9239	0.91486	Not Significant

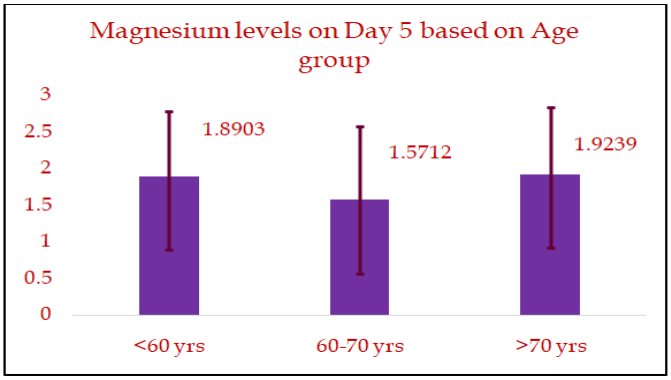

Fig.17: Magnesium levels on Day 5 based on age group

Table 17: Displays the Magnesium levels in patients on Day 5 based on age group. It is clear from the table that the mean Mg level in patients below 60 years was 1.89±0.90206 mg/dL, for patients between 60 and 70 years of age was 1.57±1.00861 mg/dL and for patients above 70 years it was 1.92±0.91486 mg/dL. The minimum Magnesium level is seen in patients between 60 and 70 years of age. However, there is no significant association between age and Magnesium level on Day 5 as depicted by the insignificant 'F' value of 1.248 (p>0.05).

Table 18: Magnesium levels on Day 1 based on Gender

Gender	N	Mean	Std. Deviation	't' Value (Significance 'p' value)
Male	67	1.87	0.38934	0.394
Female	33	1.84	0.33232	p>0.05
				Not Significant

Table 18: Presents the mean Magnesium level on Day 1 in the patients based on their gender. There is no significant association between gender and Magnesium level on Day 1 as depicted by the insignificant 't' value of 0.394 (p>0.05). The mean Magnesium level on Day 1 was 1.87±0.38934 mg/dL in Male and 1.84±0.33232 mg/dL in Female. The Serum Magnesium level was little lower in female than male on Day 1.

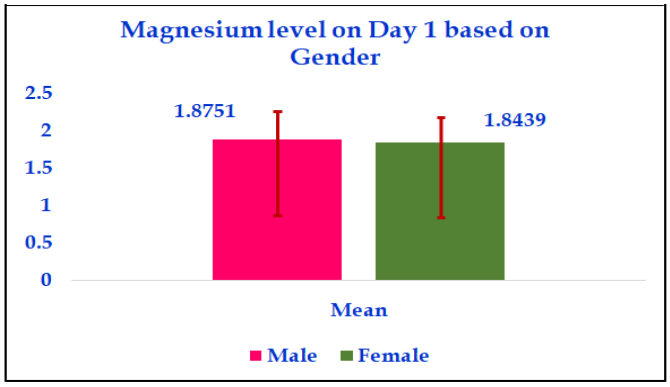

Fig.18: Magnesium levels on Day 1 based on Gender

Table 19: Magnesium levels on Day 5 based on Gender

Gender	N	Mean	Std. Deviation	't' Value (Significance 'p' value)
Male	67	1.85	0.93681	0.370
Female	33	1.77	0.95202	p>0.05
				Not Significant

Table 19: Presents the mean Magnesium level on Day 5 in the patients based on their gender. There is no significant association between gender and Magnesium level on Day 5 as depicted by the insignificant 't' value of 0.370 (p>0.05). The mean Magnesium level on Day 5 was 1.85±0.93681 mg/dL in Male and 1.77±0.95202 mg/dL in Female. The Serum Magnesium level was little lower in female than male on Day 5.

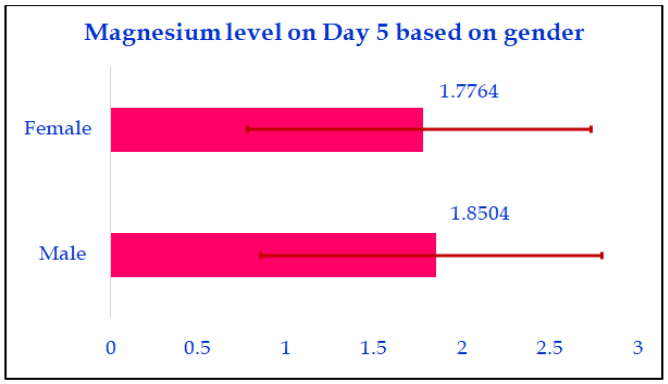

Fig. 19: Magnesium levels on Day 5 based on Gender

Table 20: Mean and standard deviation of Sodium and Potassium levels in patients

Parameters	N	Mean	Std. Deviation
Na+ level (mEq/L)	100	139.630	3.1258
K+ level (mEq/L)	100	4.41	0.43

Table 20: Shows the Sodium and Potassium level in Patients in the study.

The average Sodium level was 139.630±3.1258 mEq/L and average Potassium level was 4.41±0.43 mEq/L.

Table 21: Distribution of patients based on time of presentation to Hospital

Time of Presentation in hrs	Frequency	Percent	Cumulative Percent
0-3	31	31.0	31.0
3-6	32	32.0	63.0
>6	37	37.0	100.0
Total	100	100.0	

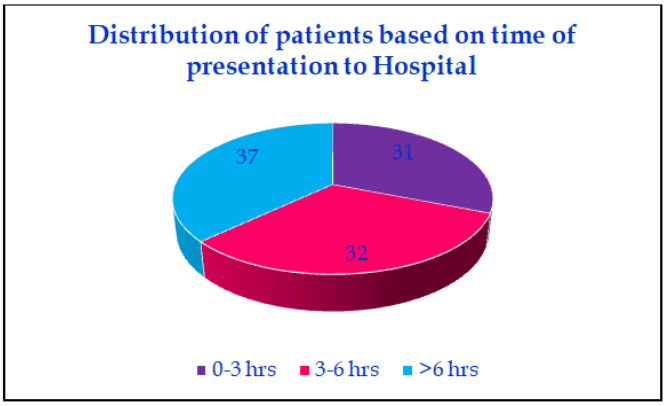

Fig. 20: Distribution of patients based on time of presentation to Hospital

Table 21: Portrays the distribution of patients based on their time of presentation to the Hospital. It can be understood from the table that 31% of the patients have been presented within 3 hours of onset of chest pain, 32% were presented between 3 and 6 hours of onset of chest pain and the remaining 37% were presented only after 6 hours of chest pain.

Table 22: Distribution of patients based on type of MI

Type of MI	Frequency	Percent	Cumulative Percent
AWMI	45	45.0	45.0
ASMI	28	28.0	73.0
IWMI	27	27.0	100.0
Total	100	100.0	

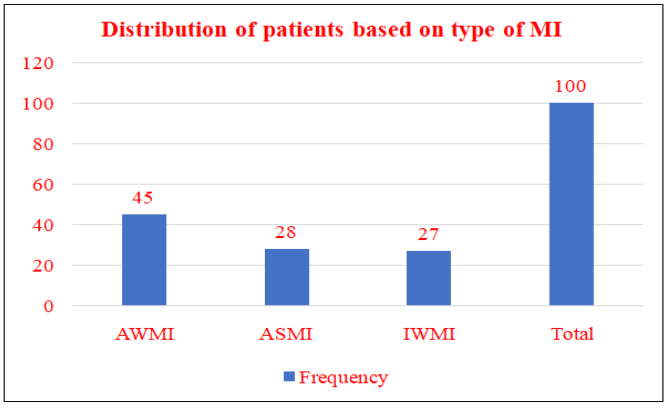

Fig. 21: Distribution of patients based on type of MI

Table 22: Displays the distribution of patients based on the type of Myocardial Infarction. It is clear from the table that 45% of the patients have Anterior Wall Myocardial Infarction, 28% have Anteroseptal Myocardial Infarction and 27% have Inferior Wall Myocardial Infarction.

Table 23: Magnesium level on Day 1 based on type of MI

Type of MI	Magnesium level on Day 1 (mg/dl)			ANOVA 'F' Value (Significance 'p' value)
	N	Mean	Std. Deviation	
AWMI	45	1.88	0.35626	0.729 p>0.05 Not Significant
ASMI	28	1.90	0.41495	
IWMI	27	1.79	0.34675	
Total	100			

Table 23: Presents the Magnesium level in patients on Day 1 based on the type of MI. There is no significant association between type of MI and Serum Magnesium level on Day 1 as depicted by the insignificant 'F' value of 0.729 (p>0.05). The Mean Magnesium level on Day 1 for patients with AWMI was 1.88±0.35626 mg/dL, for patients with ASMI was 1.90±0.41495 mg/dL and for patients with IWMI was 1.79±0.34675 mg/dL. The Serum Magnesium level on Day 1 was low in patients with IWMI in the study.

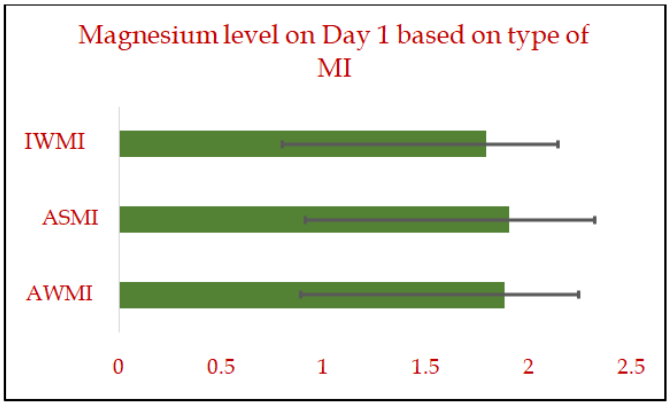

Fig. 22: Magnesium level on Day 1 based on type of MI

Table 24: Magnesium level on Day 5 based on type of MI

Type of MI	Magnesium level on Day 5 (mg/dl)			ANOVA 'F' Value (Significance 'p' value)
	N	Mean	Std. Deviation	
AWMI	45	1.83	0.94606	0.009 p>0.05 Not Significant
ASMI	28	1.83	0.99210	
IWMI	27	1.80	0.90029	
Total	100			

Table 24: Presents the Magnesium level in patients on Day 5 based on the type of MI. There is no significant association between type of MI and Serum Magnesium level on Day 5 as depicted by the insignificant 'F' value of 0.009 (p>0.05). The Mean Magnesium level on Day 5 for patients with AWMI was 1.83±0.94606 mg/dL, for patients with ASMI was 1.83±0.99210 mg/dL and for patients with IWMI was 1.80±0.90029 mg/dL. The Serum Magnesium level on Day 5 was low in patients with IWMI in the study.

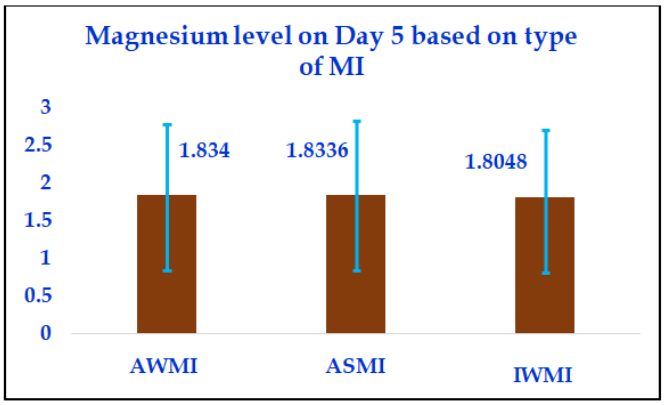

Fig. 23: Magnesium level on Day 5 based on type of MI

Table 25: Distribution of patients based on presence of Arrythmia

Presence of Arrythmia	Frequency	Percent	Cumulative Percent
Yes	46	46.0	46.0
No	54	54.0	100.0
Total	100	100.0	

Table 25: Displays the distribution of patients based on presence of Arrythmia in the study. It can be depicted from the Table that 46% of the patients had irregular heart beat and 54% did not have Arrythmia.

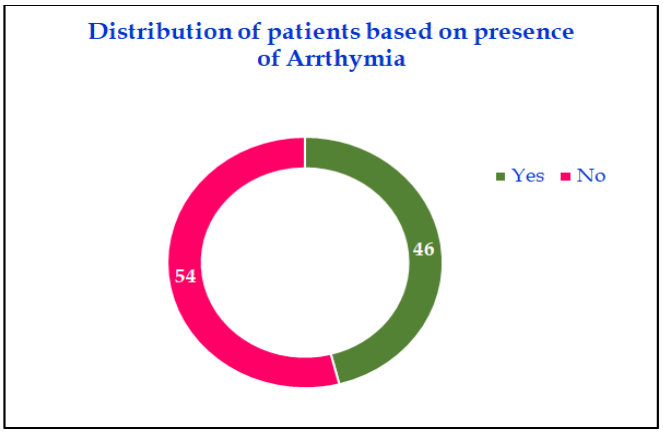

Fig.24: Distribution of patients based on presence of Arrythmia

Table 26: Magnesium level on Day 1 based on presence of Arrythmia

Presence of Arrthymia	Magnesium level on Day 1 (mg/dL)			't' Value (Significance 'p' value)
	N	Mean	Std. Deviation	
Yes	46	1.59	0.29665	-8.882 p<0.01 Highly Significant
No	54	2.09	0.26053	

Table 26: Shows the Magnesium level on Day 1 of the patients based on presence of Arrythmia. The mean Magnesium level on Day 1 in Patients with Arrythmia was 1.59±0.29665 mg/dL and for patients without Arrythmia it was 2.09±0.26053 mg/dL. The serum Magnesium level was low in the patients with Arrythmia in the study. Also, there is a highly significant difference in the Serum Magnesium level on Day 1 in patients with and without Arrythmia as depicted by the highly significant 't' value of -8.882 (p<0.01).

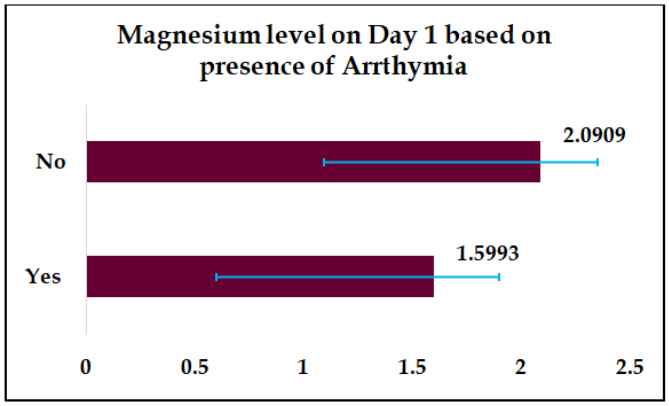

Fig. 25: Magnesium level on Day 1 based on presence of Arrythmia

Table 27: Magnesium level on Day 5 based on presence of Arrythmia

Presence of Arrythmia	Magnesium level on Day 1 (mg/dL)			't' Value (Significance 'p' value)
	N	Mean	Std. Deviation	
Yes	46	1.20	1.08233	-7.613 p<0.01 Highly Significant
No	54	2.35	0.19354	

Table 27: Shows the Magnesium level on Day 5 of the patients based on presence of Arrythmia. The mean Magnesium level on Day 5 in Patients with Arrythmia was 1.20±1.08233 mg/dL and for patients without Arrythmia it was 2.35±0.19354 mg/dL. The serum Magnesium level on Day 5 was low in the patients with Arrythmia in the study. Also, there is a highly significant difference in the Serum Magnesium level on Day 5 in patients with and without Arrythmia as depicted by the highly significant 't' value of -7.613 (p<0.01).

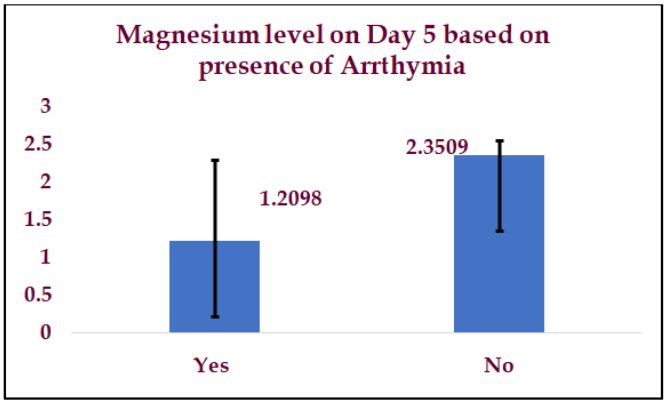

Fig. 26: Magnesium level on Day 5 based on presence of Arrythmia

Table 28: Distribution of patients based on mortality

Mortality	Frequency	Percent	Cumulative Percent
Yes	22	22.0	22.0
No	78	78.0	100.0
Total	100	100.0	

Table 28: Displays the distribution of patients based on mortality. It is clear from the Table that 22% have died and 78% have recovered in the study.

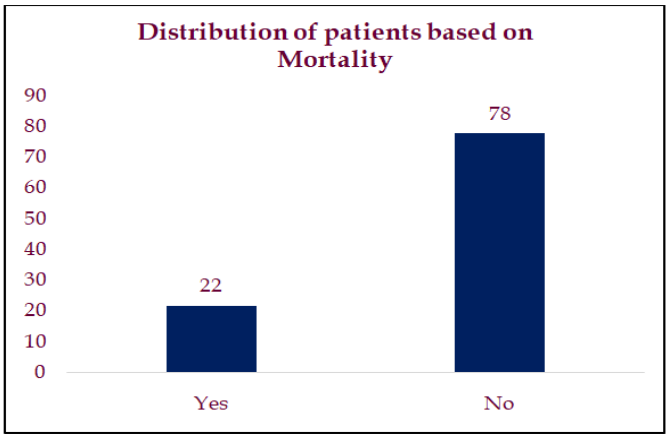

Fig.27: Distribution of patients based on Mortality

Table 29: Magnesium level on Day 1 based on Mortality

Mortality	Magnesium level on Day 1 (mg/dL)			't' Value (Significance 'p' value)
	N	Mean	Std. Deviation	
Yes	22	1.3577	0.08679	-10.615
No	78	2.0078	0.28261	p<0.01 Highly Significant

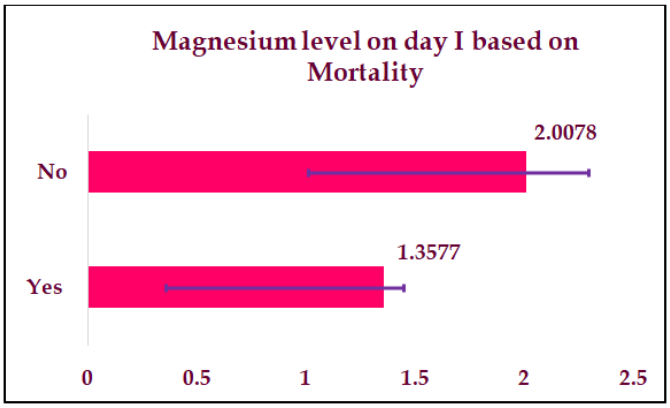

Fig.28: Magnesium level on Day 1 based on Mortality

Table 29: Shows the Magnesium level on Day 1 of the patients based on mortality. The mean Magnesium level on Day 1 in patients who have died was 1.35±0.08679 mg/dL and for patients who recovered was 2.0078±0.28261 mg/dL. The serum Magnesium level was low in the patients who died. Also, there is a highly significant difference in the Serum Magnesium level on Day 1 in patients who died and survived as depicted by the highly significant 't' value of -10.615 ($p<0.01$).

Table 30: Magnesium level on Day 5 based on Mortality

Mortality	N	Mean	Std. Deviation
Yes	22	0.0	0.0
No	78	2.2642	0.33269

Table 30: Shows the Magnesium level on Day 5 of the patients based on mortality. The mean Magnesium level on Day 5 in Patients who have died could not be recorded and for patients who recovered was 2.2642±0.33269 mg/dL.

Table 31: Distribution of patients based on Magnesium levels on Day 1 and Day 5

Magnesium level (mg/dL)	Day 1	Day 5
<1.6 mg/dL	28 (28%)	20 (20%)
1.6-2.5 mg/dL	61 (61%)	61.0 (61%)
>2.5 mg/dL	11 (11%)	19.0 (19%)
Total	100	100.0

Table 31: Shows the distribution of patients based on Magnesium levels on Day 1 and Day 5. 28% of the patients had Serum Magnesium level below 1.6 mg/dL, 61% had Serum Magnesium level between 1.6 and 2.5 mg/dL and 11% had above 2.5 mg/dL on Day 1. Similarly, on Day 5, 20% had below 1.6 mg/dL, 61% had between 1.6 and 2.5 mg/dL and 19% had above 2.5 mg/dL.

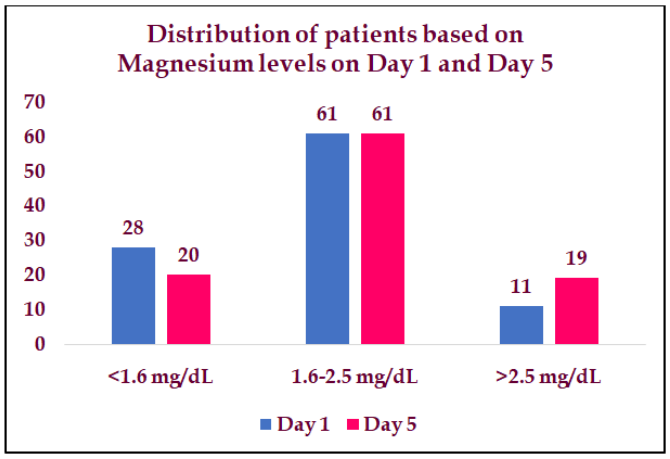

Fig.29: Distribution of patients based on Magnesium levels on Day 1 and Day 5.

Discussion

Age: In the present study, 29% of the patients are below 60 years of age, 25% are between 60 and 70 years and 46% are above 70 years of age. Majority of the patients were above 70 years of age which is in line with the study by Shafiq et al [146].

Gender: 67% of the patients are male and 33% are female in the present study. This correlates with the study by Shafiq et al [146] where the male was 61%.

Symptoms during Admission: All the patients (100%) in the study were admitted to the Hospital due to Chest pain. This result correlates with the study by Abdul et al [147] where 100% of the cases had Chest pain.

In addition, 42% of the patients had sweating, 38% had Breathlessness, 11% had Palpitation and 9% did not have any other symptom that chest pain. Similarly, in a study by Abdul et al [147]chest pain was associated with sweating in 30 (60%) of patients. Chest pain was associated with breathlessness in 32 (64%) the patients. Palpitation associated with chest pain was present in 25 patients (50%).

Diet: 78% of the patients follow mixed diet and 22% are vegetarians in the present study.

Risk Factors: Majority of the patients (59%) are non-smokers and the remaining 41% are smokers. Majority of the patients (76%) do not have family history for such diseases and the remaining 24% show family history of diseases such as HTN, IHD, CVA and DM. Majority of the patients (68%) are not obese and the remaining 32% are obese. Majority of the patients (60%) do not have Diabetes Mellitus and the remaining 40% have Diabetes mellitus. 83% of the patients do not have dyslipidaemia and 17% have dyslipidaemia. Majority of the patients (58%) do not have hypertension and only 42% have Hypertension. These findings correlates well with the study by Adbul et al [147].

Complication: 54%recovered, 8% have recovered from VPC, 3% have recovered from LVF, 5% have recovered from Cardiogenic shock and 7% have recovered from SVT. However, 14% died of VT and 9% died of VPC.

Magnesium Levels: The mean Magnesium level in the 100 patients on Day 1 was 1.86 ± 0.370 mg/dl. Similarly, the magnesium level was observed only in 78 patients and the mean was 2.29±0.210 mg/dL.

Magnesium Level and Complication: The mean Mg level in SVT recovered patients was 1.81±0.23 mg/dL, in the recovered patients was 2.09±0.26 mg/dL, VPC recovered patients were 1.78±0.26, LVF recovered patients were 1.96±0.15 mg/dL and Cardiogenic shock recovered patients were 1.86±0.26. Magnesium level was very low in the patients who died. The mean MG

level was 1.39±0.07 mg/dL in VPC deceased patients on Day 1 and 1.34±0.09 mg/dL in patients who died of VT. There is a highly significant association between Magnesium level and Complications involved as depicted by the highly significant 't' value of 27.304 ($p<0.01$).

Magnesium level and Age

The mean Mg level in patients below 60 years was 1.90±0.42052 mg/dL, for patients between 60 and 70 years of age was 1.74±0.33032 mg/dL and for patients above 70 years it was 1.90±0.35050 mg/dL. The minimum Magnesium level is seen in patients between 60 and 70 years of age. However, there is no significant association between age and Magnesium level on Day 1 as depicted by the insignificant 'F' value of 1.709 ($p>0.05$).

Magnesium level and Gender: There is no significant association between gender and Magnesium level on Day 1 as depicted by the insignificant 't' value of 0.394 ($p>0.05$). The mean Magnesium level on Day 1 was 1.87±0.38934 mg/dL in Male and 1.84±0.33232 mg/dL in Female. Similarly, There is no significant association between gender and Magnesium level on Day 5 as depicted by the insignificant 't' value of 0.370 ($p>0.05$). The mean Magnesium level on Day 5 was 1.85±0.93681 mg/dL in Male and 1.77±0.95202 mg/dL in Female. The Serum Magnesium level was little lower in female than male on Day 1 as well as Day 5. This is well correlated with the study by Shafiq et al [146]Where the female patients had low Mg level.

Sodium and Potassium Levels: The average Sodium level was 139.630±3.1258 mEq/L and average Potassium level was 4.41±0.43 mEq/L.

Time of Presentation: 31% of the patients have been presented within 3 hours of onset of chest pain, 32% were presented between 3 and 6 hours of onset of chest pain and the remaining 37% were presented only after 6 hours of chest pain. This is in line with the findings of Abdul et al [147] Where 54% of the cases were admitted within 6 hours.

Distribution of Magnesium level: 28% of the patients had Serum Magnesium level below 1.6 mg/dL, 61% had Serum Magnesium level between 1.6 and 2.5 mg/dL and 11% had above 2.5 mg/dL on Day 1. Similarly, on Day 5, 20% had below 1.6 mg/dL, 61% had between 1.6 and 2.5 mg/dL and 19% had above 2.5 mg/dL.

Conclusion

The present study was conducted with 100 patients and the results imply that Magnesium levels play major role in Mortality rate in acute MI. It is also a deciding factor in arrythmias. Low Magnesium levels are to be considered very serious as per the present study. It can be concluded that routine investigation of Serum Magnesium should be done in all cases presented with MI. In addition, continuous cardiac monitoring should be undertaken for every patient with acute MI immediately after admission into CCU to identify any cardiac arrhythmia early so that definite treatment and preventive measures can be taken promptly.

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