

## Original Article

# Correlation between serum magnesium and troponin I in acute myocardial infarction

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### Abstract

Magnesium is one of the seven essential macro minerals in our human body and the 4<sup>th</sup> most abundant cation in the body. The role of serum magnesium in cardiovascular disease has been acknowledged extensively. The association between hypomagnesemia and acute myocardial infarction has been well recognized. A cross-sectional study was conducted at Chettinad Hospital and Research Institute, and the samples were recruited from the clinical laboratory of the Biochemistry department. Fifty patients diagnosed with acute MI for whom serum magnesium and troponin T levels were taken into the study. Statistical analysis was done in SPSS version 29. The mean age of study subjects is 65±13.78. The scatter plot illustrates the association between all study subjects' cardiac troponin I and serum Magnesium values. The association between troponin I and magnesium is negative. The Pearson correlation coefficient "r" is -0.05. It shows that the inverse correlation between serum magnesium and troponin I, as shown in the current study, could mean that low serum magnesium may have a higher area of infarction.

**Keywords:** cardiovascular disease, myocardial infarction, mineral.

### Introduction

Cardiovascular disease is a wide-ranging term that includes diseases of the heart and blood vessels and the build-up of plaque, which can cause blockages inside central blood vessels in the body [1]. Cardiac disease is a chief source of disease and death on a globally large scale, with the lifetime threat being greater than 60% [2]. CVD is a collection of diseases of the vital organ and an artery. They contain coronary heart disorders, cerebrovascular disease, peripheral arterial illness, rheumatic heart disease, strokes and heart attacks [3]. Acute myocardial infarction, generally recognized as cardiac arrest, is one of the main reasons of death across the world. The first few hours of AMI are the most crucial because ignoring the diagnosis during these hours can be fatal. A heart attack can be caused by one or more clots in the coronary blood vessel. A blockage is developed due to an accumulation of plaque, a material typ-

ically made of lipids and cellular waste products [4]. Coronary disease is the most common reason for adult mortality. It is in charge of 30% of mortality globally [5]. Risk factors include high BP, diabetes, obesity, cholesterol, aging, and genetics.

Troponin I, CK CK-, MB and BNP are the myocardial markers for diagnosing AMI. Troponin I is tremendously definite for the cardiac muscle, making it an ideal marker. Troponin-I can be noticed in the circulation 6–8 hours post myocardial infarction and peaks by 12–24 hours. Then, troponin can be raised for up to 7–10 days. Troponins make finding recurrent MI difficult because they can endure increased for up to 10 days [6]. Troponin T has a dual presence within the cardiac myocyte, mostly bound to contractile elements and freely within the cytosol. It can remain elevated for 10 to 14 days post myocardial infarction [7].

Mg is one of the seven important macro minerals to humans. It is the fourth richest cation in our body



and the second most predominant intracellular cation. Magnesium deficit in human beings is constantly connected with other illnesses, most commonly as an inborn error of metabolism. Standard levels of serum Magnesium are 1.8–2.2 mg/dL. Magnesium is also required to maintain electrical potential in muscle membranes and nerves—mg deficit results in neuromuscular dysfunction. A few researches show that magnesium deficit in individuals might play a most significant role in cognitive impairment and in causing Alzheimer’s disease [8]. About 30–40% of the dietary magnesium is absorbed from the small intestine [9].

The part of serum magnesium in cardiac disease has been acknowledged extensively. The association between magnesium deficiency and irregular heartbeat is well recognized. Numerous researchers have similarly distinguished the relationship between magnesium deficit and CAD. Mg advances myocardial metabolism and prohibits the accumulation of calcium and myocardial apoptosis. It enhances vascular tone, exterior vascular resistance, decreases cardiac arrhythmias and helps build on lipid metabolism. The concentration of myocardial magnesium in subjects with unpredicted demise because of ischemic disease is very low. These conclusions are directly associated with the resulting difficulties of MI, such as irregular heartbeat. For the post-AMI complication, hypomagnesemia is considered to be a significant risk factor. It has been stated in numerous intercontinental researches that the serum magnesium level is not only lower in Acute Myocardial

Infarction (heart attack) but also remains to fall even for days after the start of Acute MI [10]. It has been found to be one of the important factors in the causation of AMI, resulting in complications like arrhythmias. Magnesium acts as a natural calcium antagonist and disturbs the contractility and conductivity of the myocardium. It also has direct electrophysiological effects by regulating vascular calcification, vascular tone, multiplication, and endothelial and vascular smooth muscle cell migration. Magnesium results in CAD due to its part in the coronary spasm, the pathogenesis of arteriosclerosis, AMI, increased platelet aggregation and the risk of arrhythmias. Magnesium has independent effects on blood pressure, lipid profile, and glycaemic parameters, thus affecting the bulk of risk factors for MI [11].

### Material and methods

This study was conducted at Chettinad Hospital and Research Institute, and the samples were recruited from the clinical laboratory of the Biochemistry department. This study was conducted only after getting approval from the institutional ethical committee. Fifty subjects were identified with acute MI for whom serum magnesium and cardiac troponin-I levels were taken into the study. Also, other parameters, including their glucose levels, LFT, RFT, and lipid profile, are taken (Table 1). All subjects diagnosed with cardiac

Table 1: Descriptive statistics for study participants.

Parameter	Descriptive statistics		
	N=50	Male (N=24)	Female (N=26)
	Mean±SD	Mean±SD	Mean±SD
Age	65±13.78	63.92±12.12	66.00±15.32
BMI kg/m <sup>2</sup>	25.81±2.11	24.68±1.82	26.85±1.83
Troponin-I pg/ml	1493.01±3574.81	1766.02±4108.78	1241.00±3062.38
BNP pg/ml	830.42±1226.44	829.62±1048.51	831.15±1391.6
CK-MB ng/ml	16.588±65.45	25.74±93.91	8.13±11.2
Magnesium mg/dL	2.05±0.41	2.01±.40	2.08±.43
Totalp protein g/dL	6.86±0.74	6.88±.62	6.85±.86
ALB g/dL	3.2±0.65	3.24±.52	3.15±.76
Globulin g/dL	4.15±3.33	4.65±4.78	3.69±.47
Bilirubin total mg/dL	1.22±2.92	1.28±2.82	1.17±3.06
Bilirubin direct mg/dL	0.76±2.61	0.78±2.67	0.74±2.60

Table 1: Continued.

Parameter	Descriptive statistics		
	N=50	Male (N=24)	Female (N=26)
	Mean±SD	Mean±SD	Mean±SD
AST mg/dL	80.32±153.08	71.91±124.32	88.07±177.71
ALT U/L	45.18±43.87	39.45±41.22	50.46±46.3
ALPH U/L	117.2±59.67	115.37±63.43	118.88±57.19
Gamma GT U/L	71.04±80.59	68.25±61.09	73.61±96.3
BUN mg/dL	36.69±26.45	43.79±29.22	29.30±21.7
Serum CR mg/dL	2.96±2.68	3.88±3.16	2.12±1.85
RBS mg/dL	166.08±69.99	176.04±88.58	156.88±46.90
TC mg/dL	146.5±51.00	140.87±50.63	151.69±51.79
TGL mg/dL	124.14±79.57	114.04±84.20	133.46±75.48
HDL mg/dL	41.34±23.01	43.04±24.46	39.76±21.96
LDL mg/dL	82.22±43.20	78.91±45.29	85.26±41.84
VLDL mg/dL	24.9±15.97	22.95±16.91	26.69±15.15

arrest and both genders of age group from 18 were included in the study. Cases diagnosed with hypoproteinaemia, malnutrition, malignancy, and patients who are on drugs interfering with thyroid function (amiodarone, lithium, propranolol) were excluded from this study. Troponin-I is estimated through the CLIA method using UniCel dxi-600 – access immunoassay systems Beckman colter. The normal values for

serum troponin-I are up to 19.8 pg/ml in males and 11.6 pg/ml in females. Serum magnesium is estimated through methyl thymol blue, and the normal reference range is 1.8–2.6 ml/dL.

The completed data was entered into an MS Excel worksheet. The collected data of study subjects were analyzed with IBM SPSS (Statistical Package Social Service) version 29.0.

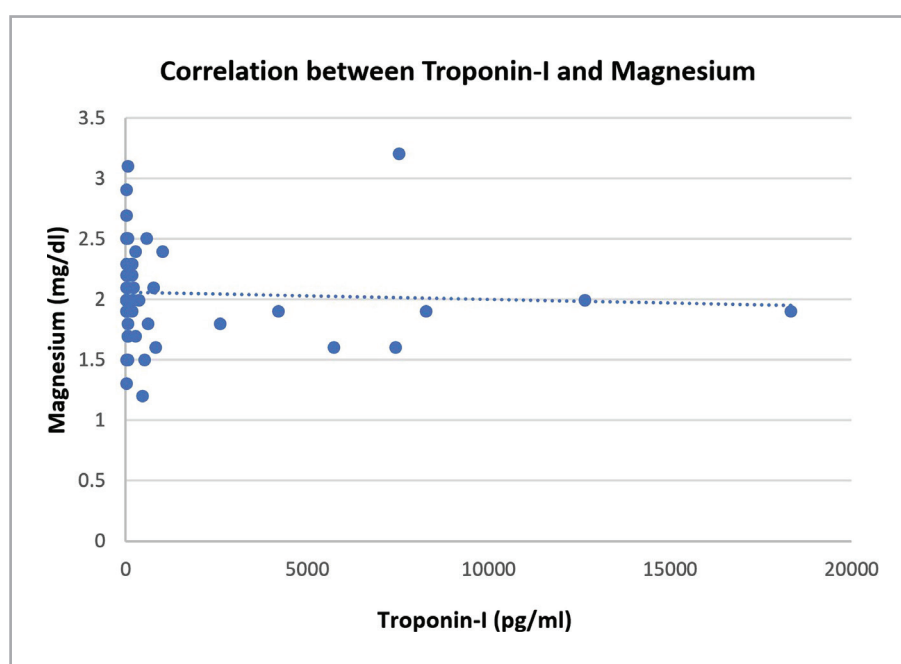


Figure 1: Correlation between serum magnesium and troponin-I.

## Results and discussion

In this study, the total sample size is 50. Among them, 26 (52%) are females, and 24 (48%) are males. The mean±SD age for males is 63.92±12.12, and for females is 66.00±15.32. Mean±SD age of the study population was 65±13.78.

The study conducted on the association between serum magnesium and troponin-I in acute myocardial infarction showed a negative correlation. It shows that lower magnesium levels may indicate extensive myocardial damage, which portends poor cardiovascular disease. As exposed in Figure 1, plasma magnesium levels are associated inversely with cardiac troponin I values. Troponin I is statistically significant, with a “p” value of 0.732 and a “r” value of -0.05, which indicates that magnesium is inversely correlated with troponin I (Figure 1).

A similar study by Ajay Kumar *et al.* illustrates the evaluation of serum magnesium and troponin-T in acute coronary syndrome. The study subjects include 41 subjects identified with severe coronary events. The scatter plot shows the correlation between serum magnesium and Troponin-T, which is weakly negative but is non-zero. The Pearson coefficient “r” is -0.022. The adverse association between plasma magnesium and cardiac troponin-T revealed by the research means that the lower human serum magnesium levels may have increased myocardial damage. This might be indirect because serum Mg is related to the complex severity of multiple chronic conditions such as dyslipidemia, impaired insulin sensitivity, deprived glycaemic controller and damaged CHO ingestion [12]. Therefore, from this study, the comparison between human plasma Magnesium and cardiac troponin-T in coronary disease results in a weak negative association, which denotes that magnesium deficiency can cause myocardial damage, which enhances CVD in older people.

The study by Vedamanickam R. *et al.* proved that lower blood serum magnesium was significantly found in Acute MI subjects. This specific study is conducted to evaluate the comparison of the levels of plasma Mg in subjects with heart attack (AMI) presenting in less than 1 day. A total number of 73% of subjects with severe coronary thrombosis were above 41 years of age, 53% had frontal wall myocardial infarction, 40% of them existed along with inferior wall asystole and 7% through anteroseptal myocardial ischemia. The average plasma Mg levels in Acute MI subjects is 1.23±0.98 mg/dL and 2.12±0.68 mg/dL in the healthy group. This indicates that the levels of human Serum Magnesium were sig-

Table 2: Average mean values of troponin-I.

Gender	Mean values of troponin-I
Male	1766.02
Female	1241.00

nificantly lower in acute myocardial patients. Hence, magnesium supplements may enhance the patient’s result [13]. From our study, Table 2, a graphical presentation of Troponin-I, shows the average mean values of males is 1766.02 and females is 1241.00, which indicates that males are more susceptible to developing the risk of cardiac diseases than females. Table 3 shows the troponin-I average values by age-wise classification. By this, it is evident that the age group of 51–60 years has a higher risk of developing cardiac disease than other age groups, and they are mostly susceptible to CVD. Age plays a dynamic role in developing the risk of CVD in older adults. The mean range of BMI for males is 24.68, and for females is 26.85, where a BMI less than 18.5 to 24.9 is considered healthy. In this study, the female subject is considered to be overweight.

Magnesium helps regulate heart rhythm, which aids the functioning of the nerves all over the body; it is essential for managing the movement of the vital organ muscles and the nerves, which help initiate the heart-beat. If serum magnesium levels are lower than normal, people are more likely to risk developing arrhythmias (irregular heartbeats). Magnesium helps lower the BP and decrease the risk of arrhythmia, which is also frequently difficult in those with congestive cardiac failure; a weak heart might benefit from consuming more from this micro-mineral. It helps keep coronary blood vessels from developing contractions, which mainly causes the intensive cardiac arrest known as angina pectoris. Magnesium deficiency is the most common reason for such coronary arterial spasms. Magnesium might play a major part in the reduction of elevated BP,

Table 3: Troponin-I values for age groups.

Age group	Troponin-I
31–40	2901.45
41–50	108.72
51–60	3340.37
61–70	1677.5
71–80	468.83
>80	63.54

an essential feature in reducing the danger of stroke and cardiac attack. The effect of magnesium on higher blood pressure is improved since it helps equalize serum potassium and sodium levels in the blood. Numerous research shows that serum magnesium and dietary magnesium have an adverse association with total CVD, which consists of elevated BP and hyperlipidemia.

## Conclusion

We conclude that the inverse correlation between serum magnesium and troponin I shown in the present study could mean that lower serum magnesium might have a higher area of infarction. Henceforth, further research is compulsory with a large sample size to arrive at a certain conclusion, and there is a need for conducting studies in different regions of the world to document the state of serum magnesium deficiency in severe myocardial infarction patients.

## Conflict of interest

The authors declare no conflict of interest.

## Ethics approval

The approval for this study was obtained from the Institutional Human Ethics Committee – Chettinad Academy of Research and Education (CARE IHEC II) (approval ID: IHEC-I/1400/22).

## Consent to participate

Written informed consent was obtained from all the participants.

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