



ROLE OF CARDIAC BIOMARKERS IN ACUTE MYOCARDIAL INFARCTION

Dr Sahana Y¹, Dr Rakshitha M N², Dr Pramod N J^{3*}

¹Associate Professor, Department of Biochemistry, Subbaiah Institute of Medical Sciences and Research Centre, Shivamogga, Karnataka

²Assistant Professor, Department of Biochemistry, Subbaiah Institute of Medical Sciences and Research Centre, Shivamogga, Karnataka

^{3*}Assistant Professor, Department of Biochemistry, Subbaiah Institute of Medical Sciences and Research Centre, Shivamogga, Karnataka

Corresponding author: Dr Pramod N J,

*Assistant Professor, Department of Biochemistry, Subbaiah Institute of Medical Sciences and Research Centre, Shivamogga, Karnataka

ABSTRACT

Background: Acute myocardial infarction (AMI) remains one of the leading causes of morbidity and mortality worldwide, affecting both developed and developing countries. Despite significant advances in diagnostic modalities and therapeutic strategies over recent decades, AMI continues to pose a major public health challenge, particularly in low- and middle-income nations undergoing rapid epidemiological transition. The diagnosis of AMI traditionally relies on a combination of clinical features, electrocardiographic (ECG) changes, and biochemical markers of myocardial injury such as creatine kinase (CK), creatine kinase-MB (CK-MB), lactate dehydrogenase, and cardiac troponins. However, conventional enzymes like CK and CK-MB lack absolute myocardial specificity and may be elevated in several non-cardiac conditions, thereby limiting their diagnostic accuracy. Cardiac troponin I (cTnI), owing to its high myocardial tissue specificity and sensitivity, has emerged as the gold standard biomarker for the early diagnosis of myocardial injury.

Materials and Methods: This hospital-based case-control study was conducted in the Department of Biochemistry, Subbaiah Institute of Medical Sciences and Research Centre, Shivamogga, Karnataka over a period of 1 year. Thirty apparently healthy individuals without risk factors for coronary artery disease served as controls, while 30 patients admitted to Hospital, with acute chest pain and ECG changes suggestive of AMI constituted the study group. Venous blood samples were collected, and serum was analyzed for cardiac biomarkers including troponin I, CK-MB, and total CK, along with lipid profile parameters—total cholesterol (TC), high-density lipoprotein (HDL), low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), and triglycerides (TG). Statistical analysis was performed to compare mean values between controls and cases, and sensitivity and specificity of biomarkers were calculated.

Results: The mean serum troponin I levels were significantly elevated in AMI patients compared to controls ($4.439 \pm 3.229 \mu\text{g/ml}$ vs $0.012 \pm 0.006 \mu\text{g/ml}$; $p < 0.001$). CK-MB and CK levels were also markedly increased in cases ($97.067 \pm 80.439 \text{ U/L}$ and $233.767 \pm 92.565 \text{ U/L}$, respectively) compared to controls ($8.794 \pm 6.041 \text{ U/L}$ and $59.207 \pm 18.831 \text{ U/L}$; $p < 0.001$). Troponin I demonstrated 100% sensitivity and 100% specificity for the diagnosis of AMI, outperforming CK-MB and CK. Lipid profile analysis revealed significantly higher levels of TC, LDL, VLDL, and

triglycerides and significantly lower HDL levels in AMI patients compared to healthy controls ($p < 0.001$).

Conclusion: The study highlights that cardiac troponin I is a superior, highly sensitive, and specific biomarker for the early diagnosis of acute myocardial infarction compared to CK and CK-MB. Additionally, significant dyslipidemia observed in AMI patients underscores the critical role of lipid abnormalities as major risk factors for ischemic heart disease. Early detection of myocardial injury using troponin I, along with aggressive management of lipid disorders, may contribute to improved clinical outcomes in patients with AMI.

Keywords: Acute Myocardial infarction, Cardiac biomarker, Cardiac troponin I.

INTRODUCTION

Acute myocardial infarction (AMI), the leading cause of deaths in both developed and developing countries¹. Despite impressive studies in diagnosis and management over the past three decades, AMI continues to be a major public health problem in the industrialized world and is becoming an increasingly important problem in developing countries². It is due to the impairment of heart function due to inadequate blood flow to the heart compared to its needs, caused by obstructive changes in the coronary circulation to the heart³. It is cause of 25-30 percent of deaths in most industrialized countries.

Globally, ischemic heart disease affects more than 200 million individuals, with AMI accounting for a substantial proportion of cardiovascular-related hospital admissions and deaths⁴. Current estimates indicate that AMI contributes to nearly 7–9 million deaths annually, representing a significant public health burden⁵.

In India, the prevalence of coronary artery disease (CAD), the underlying cause of AMI, has increased markedly over the past few decades⁶. Population-based studies report a CAD prevalence of approximately 8–10% in urban populations and 3–5% in rural populations. AMI accounts for a major share of emergency cardiovascular admissions in tertiary care hospitals⁷. Notably, Indians tend to develop AMI at a younger age, often 5–10 years earlier than individuals in Western countries, with a high prevalence observed in the fourth and fifth decades of life. AMI is more prevalent among males; however, the incidence in females increases significantly after menopause, narrowing the gender gap in older age groups⁸.

MI results from the reduction of coronary flow to such an extent that supply of oxygen to the myocardium does not meet the oxygen demand of myocardial tissue⁹. When this ischemia is prolonged and irreversible then the myocardial cell death and necrosis occurs which is defined as AMI¹⁰. The diagnosis of AMI is based on clinical symptoms, electrocardiographic findings (ECG), characteristic pattern of changes in some serum enzymes such as CKMB lactate dehydrogenase and its isoenzymes (LDH), aspartate Transaminase (AST), cardiac troponins etc., ECG is the most widely used method for the diagnosis of MI, but many times ECG shows inconclusive pattern¹¹. Estimation of serum biochemical markers of myocardial injury arises to confirm the diagnosis of myocardial injury¹².

WHO criteria have classically been used to diagnose MI; a patient is diagnosed with MI if two (probable) or three (definite) of the following criteria are satisfied. Clinical history of ischemic type chest pain lasting for more than 20 minutes. Rise and fall of serum cardiac biomarkers such as CK-MB fraction and Troponin¹³.

These parameters are also unsatisfactory in patients with multiorgan diseases or with skeletal muscle lesions, because in such cases a definite differentiation of skeletal muscle lesions and heart muscle necrosis is impossible because of the organ distribution of CK, LDH or their isoforms.

A new immunoassay for cTnT which eliminates most of the earlier shortcomings of laboratory infarction diagnostics was planned to be carried out to evaluate the utility value of Troponin T in early diagnosis of AMI. Elevated levels of CK-MB, Troponin T and Troponin I have been regarded as biochemical markers of myocyte necrosis¹⁴. CK, CK-MB still have a formal place in defining MI¹⁵. These enzymes normally exist in cellular compartment and leak out into the plasma during

myocardial injury due to disintegration of contractile elements and sarcoplasmic reticulum. Troponin T has high myocardial tissue specificity and offers an improved sensitivity and specificity for MI compound to traditional biochemical markers¹⁶.

MATERIALS AND METHODS

This hospital-based case-control study was conducted in the Department of Biochemistry, Subbaiah Institute of Medical Sciences and Research Centre, Shivamogga, Karnataka over a period of 1 year. The control who were perfectly healthy and not having risk of coronary artery disease, hypertension, diabetes mellitus. There was no history of smoking and the controls were not obese. Age group of the controls ranged between 30 to 55 years.

Inclusion Criteria :

Cardiac Chest Pain

Age Group in between 30 to 55 Years.

ECG changes suggestive of AMI

Exclusion Criteria:

Non Cardiac Chest pain

Patients with known coronary artery disease.

Patients with age less than 30 years and greater than 55 years.

Who are not willing for study.

COLLECTION OF SAMPLES :

Blood samples were collected and

The serum was separated within one hour after the collection of samples.

In the controls 10ml of blood was collected with a sterile syringe and needle in the morning into a sterile bottle and serum was separated within one hour after collection of the sample.

RESULTS

TABLE-1: VALUES OF TROPONIN-I FOR THE 30 SUBJECTS EACH FROM CONTROL GROUP AND THE STUDY GROUP

CASE No.	CONTROL GROUP	STUDY GROUP
1	0.002	1.54
2	0.008	10
3	0.011	1.981
4	0.001	1.080
5	0.0152	9.536
6	0.0106	2.16
7	0.005	1.007
8	0.002	2.041
9	0.0129	1.473
10	0.0159	5.43
11	0.013	2.8
12	0.0185	0.518
13	0.0024	2.008
14	0.019	4.90
15	0.007	3.218

16	0.0176	5.04
17	0.019	5.83
18	0.012	2.13
19	0.0158	8.26
20	0.001	11.8
21	0.0129	9.02
22	0.002	2.103
23	0.0195	5.81
24	0.0180	6.28
25	0.0184	5.09
26	0.0165	5.32
27	0.0174	10
28	0.015	4.29
29	0.0179	2.415
30	0.0142	1.091
MEAN (µg/ml) ± S.D	0.012 ± 0.006	4.439 ± 3.229

In the **control group**, values remain consistently low, ranging from **0.001 to 0.0195 µg/ml**, indicating minimal baseline levels of the measured parameter. The mean concentration in the control group was **0.012 ± 0.006 µg/ml**, reflecting both low absolute values and narrow variability, which is characteristic of a physiologically normal or non-diseased population. In contrast, the **study group** shows substantially elevated values, ranging from **0.518 to 11.8 µg/ml**. The mean value in the study group was **4.439 ± 3.229 µg/ml**, which is markedly higher than that observed in the control group.

TABLE 2: COMPARISON OF TROPONIN-I LEVELS BETWEEN THE HEALTHY CONTROLS AND THE STUDY GROUP

PARAMETER	CONTROLS (n=30) Mean + S.D	STUDY GROUP (n=30) Mean + S.D	COMPARISON p< 0.001
TROPONIN-I (µg/ml)	0.012 + 0.006	4.439 + 3.229	Highly Significant

The table compares mean serum Troponin-I levels in the control group were very low, with a mean value of **0.012 ± 0.006 µg/ml**, indicating normal physiological concentrations and absence of myocardial injury. In contrast, the study group demonstrated markedly elevated Troponin-I levels, with a mean of **4.439 ± 3.229 µg/ml**. Statistical comparison between the two groups revealed a **highly significant difference (p < 0.001)**, confirming that Troponin-I levels were significantly higher in the study group compared to controls.

TABLE 3: COMPARISON OF CK-MB LEVELS BETWEEN THE HEALTHY CONTROLS AND THE STUDY GROUP

PARAMETER	CONTROLS (n=30) Mean + S.D	STUDY GROUP (n=30) Mean + S.D	COMPARISON
CK-MB (U/L)	8.794 + 6.041	97.067 + 80.439	p< 0.001 Highly Significant

In the control group, CK-MB levels were within normal limits, with a mean value of 8.794 ± 6.041 U/L, indicating the absence of significant myocardial injury. In contrast, the study group showed markedly elevated CK-MB levels, with a mean of 97.067 ± 80.439 U/L. Statistical analysis demonstrated a **highly significant difference between the two groups ($p < 0.001$)**, confirming that the elevation of CK-MB in the study group is not due to random variation. This statistically

TABLE 4: COMPARISON OF CK LEVELS BETWEEN THE HEALTHY CONTROLS AND THE STUDY GROUP

PARAMETER	CONTROLS (n=30) Mean + S.D	STUDY GROUP (n=30) Mean + S.D	COMPARISON
CK (U/L)	59.207 + 18.831	233.767 + 92.565	p< 0.001 Highly Significant

The mean serum creatine kinase (CK) level in the study group was 233.77 ± 92.57 U/L, which was markedly higher than that observed in the control group (59.21 ± 18.83 U/L). Statistical analysis revealed that this difference was highly significant ($p < 0.001$).

TABLE 5: COMPARISON OF TOTAL CHOLESTEROL LEVELS BETWEEN THE HEALTHY CONTROLS AND THE STUDY GROUP

PARAMETER	CONTROLS (n=30) Mean + S.D	STUDY GROUP (n=30) Mean + S.D	COMPARISON
TC mg/dl	171.033 + 11.245	254.700 + 37.388	p< 0.001 Highly Significant

In the control group, total cholesterol levels were within normal limits, with a mean value of 171.033 ± 11.245 mg/dl, indicating a healthy lipid profile. In contrast, the study group demonstrated markedly elevated total cholesterol levels, with a mean of 254.700 ± 37.388 mg/dl. Statistical comparison between the two groups showed a **highly significant difference ($p < 0.001$)**, confirming that the elevated TC levels in the study group are unlikely to be due to chance.

TABLE 6: COMPARISON OF HDL LEVELS BETWEEN THE HEALTHY CONTROLS AND THE STUDY GROUP

PARAMETER	CONTROLS (n=30) Mean + S.D	STUDY GROUP (n=30) Mean + S.D	COMPARISON
HDL mg/dl	62.970 + 4.930	33.014 + 7.482	p< 0.001 Highly Significant

In the control group, HDL levels were relatively high, with a mean value of 62.970 ± 4.930 mg/dl, reflecting a favorable lipid profile and normal cardiovascular risk status. In contrast, the study group exhibited significantly reduced HDL levels, with a mean of 33.014 ± 7.482 mg/dl. Statistical

analysis demonstrated a **highly significant difference between the two groups ($p < 0.001$)**, confirming that the reduction in HDL levels in the study group is not attributable to random variation.

TABLE 7: COMPARISON OF LDL LEVELS BETWEEN THE HEALTHY CONTROLS AND THE STUDY GROUP

PARAMETER	CONTROLS (n=30) Mean + S.D	STUDY GROUP (n=30) Mean + S.D	COMPARISON
LDL mg/dl	82.990 + 10.560	165.978 + 32.869	p< 0.001 Highly Significant

In the control group, LDL levels were within normal physiological limits, with a mean value of **82.990 ± 10.560 mg/dl**, indicating a favorable lipid profile and lower cardiovascular risk. In contrast, the study group exhibited markedly elevated LDL levels, with a mean of **165.978 ± 32.869 mg/dl**. Statistical analysis revealed a **highly significant difference between the two groups ($p < 0.001$)**, confirming that the elevation in LDL levels among study subjects is unlikely to be due to chance.

TABLE 8: COMPARISON OF VLDL LEVELS BETWEEN THE HEALTHY CONTROLS AND THE STUDY GROUP

PARAMETER	CONTROLS (n=30) Mean + S.D	STUDY GROUP (n=30) Mean + S.D	COMPARISON
VLDL mg/dl	25.073 + 3.193	55.707 + 19.318	p< 0.001 Highly Significant

In the control group, VLDL levels were within normal limits, with a mean value of **25.073 ± 3.193 mg/dl**, indicating normal triglyceride transport and lipid metabolism. In contrast, the study group exhibited markedly elevated VLDL levels, with a mean of **55.707 ± 19.318 mg/dl**. Statistical comparison revealed a **highly significant difference between the two groups ($p < 0.001$)**, confirming that the elevation in VLDL levels in the study group is unlikely to be due to chance.

TABLE – 9: SENSITIVITY AND SPECIFICITY OF VARIOUS PARAMETERS

PARAMETERS	SENSITIVITY	SPECIFICITY
Troponin-I	100%	100%
CK-MB	93.3%	100%
CK	96.7%	100%
TOTAL CHOLESTROL	93.3%	100%
HDL	100%	100%
LDL	100%	100%
VLDL	93.3%	100%
TRIGLYCERIDES	93.3%	100%

Troponin-I demonstrated **100% sensitivity and 100% specificity**, indicating that it correctly identified all affected individuals while showing no false-positive results among controls. **CK-MB** showed a high sensitivity of **93.3%** and perfect specificity (**100%**), suggesting strong diagnostic accuracy with minimal false-negative results and no false positives. Similarly, **total CK** exhibited a sensitivity of **96.7%** and specificity of **100%**, confirming its usefulness as a supportive marker of myocardial damage. Among lipid parameters, **total cholesterol**, **VLDL**, and **triglycerides** each demonstrated a sensitivity of **93.3%** with **100% specificity**, indicating their strong ability to

identify affected individuals while maintaining excellent discrimination from healthy controls. Notably, **HDL and LDL showed 100% sensitivity and 100% specificity**, reflecting complete diagnostic accuracy in differentiating study subjects from controls.

DISCUSSION

AMI refers to two subtypes of ACS namely non-ST- elevated MI and ST-elevated MI which are the most frequently manifestation of coronary artery disease. Cardiac markers are released due to ischemia into circulation after the event... they are cTnI, CK, CKMB, LDH, AST etc

Comparison of TnI, CK, CKMB originating exclusively from the myocardium clearly differs from the skeletal muscle Tn-I. As a result of its high tissue specificity, cTnI is a cardio specific, highly sensitive marker for Myocardial damage. The peak value is detected from 3 hours up to 5 days and remains elevated upto 14 days where as CKMB and CK elevation typically begins 4-6 hrs after onset of infarction but is not elevated in all till 20 hrs of onset of occlusion. CK-MB unlike Troponin cannot be used for late diagnosis of MI. The method used to measure the activity of CK-MB also measures the CK-MB present in the serum. Hence the test is not very specific for CK-ME¹⁷.

CK-MB rises in number of conditions like aortic stenosis, hypertrophied myocardium, malignancies, inflammatory myopathies, collagen vascular diseases, Scleroderma, Hypothyroidism etc Thus decreasing the specificity of the test¹⁸ cTnI are most sensitive and specific markers available for the diagnosis of Myocardial injury. Because there are highly desirable characteristics for diagnostic markers, Troponin has been universally accepted as the gold standard to diagnose Myocardial tissue injury¹⁹. Current guidelines recommend, that values above the 99th percentile of normal reference population should be used to diagnose myocardial necrosis and in the appropriate clinical scenario of MI, though CK-MB and cTnI assays are used to detect same phenomenon cTnI assays have the advantage of being more sensitive thus being capable of detecting smaller amount of myocardial necrosis and are more specific this providing nearly certainty of a myocardial origin of the increased values For these reasons replacing CK-MB with cTnI^{20,21}.

The present study shows that cTroponin I is 100% sensitive and 100% specific where as CK-MB is 93.3% sensitive and 100% specific. These findings are in correlation with Thygesen K. et al making cTnI more sensitive and specific marker of myocardial injury although the sample size is small.

In the present study the Mean and SD are 254.700 ± 37.388 for TC, 33.014 ± 7.482 for HDL, 165.978 ± 32.869 for LDL, 55.707 ± 19.318 for VLDL and 278.533 ± 96.596 for TGL which are in close agreement with the normal laboratory values quoted by Kruppl.

The above findings indicate that TC, LDL, VLDL and Triglycerides are increased in cases when compared to the control values which are statistically significant. Risk of IHD varies over a five fold range in relation to serum cholesterol found in an average population. Framingham study 1976 proved that in the person where cholesterol level is above 220 mg/dl the incidence of IHD is more, Kannel W. B. et al.²²

Recent interest has centered around the possibility that HDL may be a protective factor. Where, HDL has been measured in prospective studies, low levels do seem to be predictive of subsequent IHD and in community where IHD is uncommon HDL levels are high, In the present study also the abnormality of HDL cholesterol was noted in cases²³.

Increase in Total triglycerides and levels of VLDL are usually associated in prospective studies with an increased IHD rates. Findings of present study showed abnormality of TGL and VLDL in cases which are significant²⁴.

CONCLUSION

In the present study 30 controls of age group 30-55 years with no risk factors and 30 cases of acute chest pain, ECG showing changes suggestive of AMI with same age group admitted in Hospital were chosen. The blood samples were collected from cases immediately after admission and cTnI, CK-MB, CK and Lipid profile was estimated. cTnI levels of cases were elevated in all the samples

which are statistically significant where as CK-MB and CK showed 93.3% and 96.7% sensitivity of cTnI is more when compared to CK-MB and CK. Serum Total Cholesterol, TG, VLDL and LDL are significantly elevated in the cases. HDL cholesterol was reduced which was statistically significant. Based on the present study the following conclusions are drawn. Serum cTnI seems to be a better and more sensitive parameter for detection and diagnosis of Acute Myocardial infarction when compared to CK-MB and CK. Elevated levels of TC, LDL, VLDL and TG have increased risk of IHD whereas increased HDL has protective effect.

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