Section: Miscellaneous

Original Research Article

REVISITING TRADITIONAL CARDIAC ENZYME IN THE ERA OF HIGH SENSITIVITY TROPONIN: DO AST AND LDH STILL HOLD DIAGNOSTIC VALUE?

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ABSTRACT

Background: Myocardial infarction (MI), most commonly due to obstruction of a coronary artery remains one of the foremost causes of morbidity and mortality worldwide, posing a significant public health challenge. The present study was undertaken to compare the serum levels of CK-MB, LDH, CRP, and AST between troponin-positive and troponin-negative subjects.

Materials and Methods: A prospective case–control study was conducted in the Department of Biochemistry at a tertiary care teaching hospital. A total of 202 participants were enrolled and divided into two groups. Patients of either sex aged >35 years with a confirmed diagnosis of non-diabetic myocardial infarction based on electrocardiographic (ECG) findings and positive cardiac biomarkers (Troponin T and/or CK-MB) were included in the study.

Results: The mean value of CK MB in the Troponin T positive group was 115.26±31.05 and in the Troponin T negative group was 18.72±4.35 (p value <0.001). AST was 78.4±7.23 IU/L in Troponin positive patients while 46.8±5.83 IU/L in Troponin negative subjects (p value <0.001).

Conclusion: Results of our study suggest that all the CK MB, CRP and LDH are effective biomarkers only in the presence of highly sensitive Troponin T in diagnosis and prognosis of MI. Further, individually these markers are not decisive indicator particularly for CAD. Further, AST individually or along with Troponin T is not reliable marker for MI as it highly sensitive to hepatocyte injury. Similarly, Higher level of LDH independently is not dependable marker for CAD.

Keywords: MI, Troponin T, CK-MB, AST.

INTRODUCTION

Myocardial infarction (MI), most commonly due to obstruction of a coronary artery remains one of the foremost causes of morbidity and mortality worldwide, posing a significant public health challenge.[1] Timely identification and rapid initiation of appropriate treatment are critical for limiting myocardial injury, preserving cardiac function, and improving patient survival.[2]

Although cardiac troponin testing is highly sensitive and specific for detecting myocardial injury, the traditional lipid profile remains a useful screening tool for identifying individuals at elevated risk of future cardiovascular events.[3]

CK-MB positivity is a sensitive biochemical marker of acute myocardial injury and plays an important role in the diagnosis of myocardial infarction.[4] CK-MB and troponin T positivity are more sensitive and specific indicators of acute myocardial injury and are essential for the diagnosis of myocardial infarction.[5]

The lactate dehydrogenase (LDH) is one of the markers of late diagnosis of myocardial infarction,

However, Sensitivity Troponin T is significantly high in comparison of LDH.[6]

C-reactive protein (CRP) is a key acute-phase reactant produced in response to interleukin-6, which activates CRP gene transcription in the liver during inflammation or infection.[7] The inflammatory process is a fundamental contributor to the pathogenesis of atherosclerosis and is also triggered by myocardial damage. Elevated concentrations of inflammatory markers are predictive of an increased risk of future cardiovascular events among healthy populations as well as patients with stable and unstable coronary artery disease (CAD).[8]

Aspartate transaminase (AST) is one of the earliest biochemical markers reported to increase following acute myocardial infarction due to the release of the enzyme from injured cardiomyocytes.[9,10]

The present study was undertaken to compare the serum levels of CK-MB, LDH, CRP, and AST between troponin-positive and troponin-negative subjects. Furthermore, it aimed to investigate the diagnostic and prognostic significance of these biomarkers, with special emphasis on AST.

MATERIALS AND METHODS

A prospective case–control study was conducted in the Department of Biochemistry at a tertiary care teaching hospital. A total of 202 participants were enrolled and divided into two groups.

Group I (Cases) comprised 112 non-diabetic patients diagnosed with acute myocardial infarction (MI) who were admitted to the Emergency Department and Intensive Care Units of Santosh Medical College Hospital & Research Centre, Ghaziabad, and Combined District Hospital, Sanjay Nagar, Ghaziabad.

Group II (Controls) included 90 apparently healthy individuals with no history of diabetes mellitus or myocardial infarction.

Patients of either sex aged >35 years with a confirmed diagnosis of non-diabetic myocardial infarction based on electrocardiographic (ECG) findings and positive cardiac biomarkers (Troponin T and/or CK-MB) were included in the study. Patients with diabetes mellitus, recent surgery or trauma within the preceding two months, renal insufficiency (serum creatinine >1.5 mg/dL), current or previous cerebrovascular accident, any acute or chronic systemic illness, or those receiving hormone replacement therapy were excluded.

Qualitative estimation of cardiac Troponin T was performed using a rapid immunoassay kit manufactured by Roche Diagnostics International Ltd., Rotkreuz, Switzerland. [11] Serum creatine kinase-MB (CK-MB) activity was measured using commercial kits supplied by Transasia Bio-Medicals Ltd., India, employing the monoreagent method. [12] Serum lactate dehydrogenase (LDH) levels were also estimated using commercial kits from Transasia Bio-Medicals Ltd., India, following the monoreagent procedure. [13]

C-reactive protein (CRP) was measured by the antigen–antibody end-point reaction method using commercial reagent kits manufactured by Erba Lachemas.r.o., Brno, Czech Republic.[14]

The lipid profile included total cholesterol (TC), triglycerides (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). Total cholesterol and triglycerides were estimated by enzymatic colorimetric methods, while HDL-C was measured using a direct enzymatic assay. LDL-C was calculated using the Friedewald equation [LDL-C = TC − HDL-C − (TG/5)] for samples with triglyceride concentrations below 400 mg/dL. All biochemical analyses were performed on a fully automated biochemistry analyzer following standard laboratory protocols. [11]

AST (aspartate aminotransferase) was measured by using Kinetic assays utilizing coupled enzyme systems using commercial kits supplied by Transasia Bio-Medicals Ltd., India. [15]

Categorical variables were expressed as frequencies and percentages, whereas continuous variables were presented as mean ± standard deviation (SD) and median, as appropriate. Differences between the two groups were analysed using the Independent Student's *t*-test for normally distributed data or the Mann–Whitney *U* test for non-normally distributed data. A *p*-value of <0.05 was considered statistically significant. Data were entered into Microsoft Excel and analysed using the Statistical Package for the Social Sciences (SPSS) version 21.0.

RESULTS

Table 1 shows that a total of 112 Troponin T positive cases (36 Females and 76 Males) and 90 Troponin T negative (30 Females and 60 Males) as controls were studied. The mean age in the cases were 62.05 ± 6.17 years and in the controls were 62.26 ± 8.25 years. There was no statistical difference between the two groups (p-value = 0.65).

Table 1: Mean of age distribution

Age	Trop T Positive(n=112)	Trop T Negative(n=90)	P value
Mean ± Stdev	62.05 ± 6.17	62.26 ± 8.25	0.65
Median(Range)	62.6(40-78)	62.5(39-79)	
Inter quartile Range	55 – 70	54 – 68	

Table 2: Comparison of lipid profile of Group I Trop T Positive patients with Group II Trop T Negative subjects

	Group I (Trop T Positive)	Group II (Trop T Negative)	p value	Changes in percentage (%) in Trop T positive
Total Cholesterol mg/dl	**223 ± 37.23**	**212.33 ± 28.17**	**<0.001**	**4.90%**
Triglycerides mg/dl	**123.63 ± 28.53**	**119.45 ± 26.22**	**<0.001**	**4.15%**
HDL mg/dl	**37.54 ± 7.09**	**40.86 ± 6.14**	**<0.001**	**-8.12%**
LDL mg/dl	**155.57± 30.95`**	**146.67 ± 25.35**	**<0.001**	**5.72 %**

It is evident from table 2 that there was a significantly high values of cholesterol (**223 ± 37.23** mg/dl vs **212.33 ± 28.17** mg/dl, p= <0.001), triglycerides (**123.63 ± 28.53**vs **119.45 ± 26.22** mg/dl, p= <0.001), low density lipids (10.04 mg/dl, p= <0.001) in **Group I Trop T Positive** patients in comparison of **Group II Trop T Negative** patients. Further, high density lipids showed significantly low (-2.32 mg/dl) in **Group I Trop T Positive** patients in comparison of **Group II Trop T Negative** patients. (p= <0.001)

The mean value of CK MB in the Troponin T positive group was 115.26±31.05 and in the Troponin T negative group was 18.72±4.35. The difference was statistically significant (P-value < 0.0001) as shown in Figure 1.

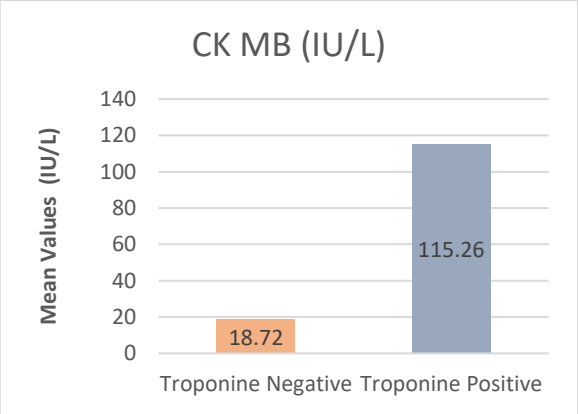

Figure 1: Mean distribution of CK MB

Table 3: Mean distribution of LDH

LDH (IU/L)	Trop T Positive(n=112)	Trop T Negative(n=90)	P value
Mean ± Stdev	573.63 ± 112.9	218.33 ± 34.54	<0.0001
Median(Range)	569.9 (350-966)	222.5(153-319)	
Inter quartile Range	456 – 618	186 – 263	

Table 3 showed that the mean value of LDH in the Troponin T positive group was significantly high 573.63 ± 112.9 compare to 218.33 ± 34.54 in the Troponin T negative group. The difference was statistically significant (P-value < 0.0001).

Table 4: Mean distribution of CRP

CRP (mg/L)	Trop T Positive(n=112)	Trop T Negative(n=90)	P value
Mean ± Stdev	18.44 ± 5.6	7.35 ± 2.3	<0.0001
Median(Range)	17.65(4.5-29.6)	6.87(1.4-10.84)	
Inter quartile Range	12.56 - 20.48	4.12 - 8.73	

The mean value of CRP in the Troponin T positive group was 18.44 ± 5.6and in the Troponin T negative group was 7.35 ± 2.3. The difference was statistically significant (P-value < 0.0001) as shown in Table 4.

Figure 2 depicted that there was a significant difference between the AST of troponin positive patients and control subjects (p value <0.001). AST was 78.4±7.23 IU/L in Troponin positive patients while 46.8±5.83 IU/L in Troponin negative subjects.

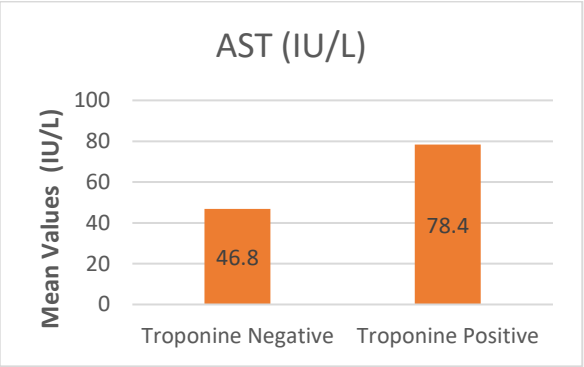

Figure 2: Mean distribution of AST

DISCUSSION

The analysis of cardiac biomarkers has become the frontline diagnostic tools for AMI, and has greatly enabled the doctors in the rapid diagnosis and

treatment planning, thereby reducing the mortality rate by a great extent. =====

Results of the present study showed that there was an insignificant difference between the mean age of control group I troponin negative and troponin positive group II patient (p-value = 0.65)

On the other hand, there was a significant difference between TC (10.67±9.06, p value <0.001), TG (4.18±2.31, p value <0.001) and LDL (8.9±5.6, p value <0.001) of group I troponin positive in comparison of group II control subjects.

While, there was significantly high HDL (3.32± -0.95, p value <0.001) in group II control subjects compare to group I MI patients.

These findings are very similar to the findings of the earlier studies of Kumar A et al,[16] and De Silva LD et al,[17] as they recorded higher level of TC, TG and LDL, while lower levels of HDL in MI patients.

These results of the current study highlight the importance of lipid profile in screening of population high risk of developing MI.[16,17] Studies have suggested that higher lipid profile has been associated with increased risk of cardiovascular diseases.[18,19]

Further, current study showed significantly high level of CK MB in troponin positive patient compare to control subjects. These findings are consistent with the results of previous studies of Meyer Tet al,[20] and Gruberg L et al,[21] as they observed higher level of CK MB in patients of MI. Positive troponin and elevated CK MB are associated with higher risk CAD and may serve as valuable prognostic tool.[20,21]

CK MB has long been recognized as an important biomarker for the diagnosis of myocardial infarction because of its relatively high concentration in cardiac muscle tissue. Despite the widespread adoption of cardiac troponins as the preferred biomarkers for myocardial injury, CK-MB continues to have clinical utility in specific situations, including the diagnosis of reinfarction and in healthcare settings where troponin testing is unavailable or inaccessible.[22]

In addition, our study recorded Significantly high level of LDH in MI patients in comparison of control subjects. These findings are very like earlier studies of Licka M et al,[23] and Vittal DA et al,[24] as they also recorded higher level of LDH in troponin positive patients.

Elevated LDH levels, along with positive troponin T and increased CK-MB concentrations, are associated with myocardial infarction (MI). However, these cytosolic enzymes are also present in non-cardiac muscle tissues and may be elevated due to conditions unrelated to myocardial injury. Such conditions include renal failure, myopathies, trauma, vigorous physical exercise, and chronic alcoholism.[23,24]

Our study recoded higher level of CRP in MI patients compare to control which is very alike to previous study Capone Det al,[25] and Lenderink T et al 26 as they also recorded higher level of CRP level in MI patients. CRP is an independent marker of inflammation along with positive Troponin T may be helpful in diagnosis as well as prognosis of MI.[25,26]

Furthermore, current study showed significantly high level of AST in Troponin positive patients in contrast of Troponin negative subjects. These results are continuing with the findings of earlier studies of Shetty DD et al,[27] and Djakpo DK et al.[28]

Serum AST levels were significantly higher in troponin-positive patients compared with troponin-negative subjects, reflecting greater myocardial injury. However, because AST is not specific to cardiac tissue, its diagnostic utility is inferior to cardiac troponins, although it may provide supportive prognostic information when interpreted alongside cardiac-specific biomarkers.[29]

Serum lactate dehydrogenase (LDH) is a known pathologic marker for a diversity of diseases, including myocardial ischemia. Strenuous and enduring physical activity can transiently induce a greater total serum LDH level, still within its normal range. To date, however, it has not been determined whether normal-range LDH might be inversely associated with coronary artery disease (CAD) in the low-cardiovascular-risk, physically active, adult population.[14]

AST was the first cardiac biomarker to be used.[9] AST is commonly used in liver function testing and is found in the liver, heart, skeletal muscles, brain and kidneys. Due to its lack of specificity to the cardiac tissue, it is no longer used for the diagnosis of AMI.[9,24,25]

Although its use has declined with the introduction of highly sensitive cardiac troponins; elevated AST levels continue to reflect the extent of myocardial injury. Therefore, AST may still serve as a useful adjunctive biomarker, particularly in resource-limited settings or when interpreted alongside established cardiac markers such as troponin and CK-MB.[30]

In addition, these cytosolic enzymes are distributed in both cardiac and non-cardiac muscle tissues. As a result, elevated enzyme levels may occur in the absence of myocardial cell damage and can be associated with various disease conditions such as chronic renal failure, myopathies.[23]

CONCLUSION

Results of our study suggest that all the CK MB, CRP and LDH are effective biomarkers only in the presence of highly sensitive Troponin T in diagnosis and prognosis of MI. Further, individually these markers are not decisive indicator particularly for CAD. Further, AST individually or along with Troponin T is not reliable marker for MI as it highly sensitive to hepatocyte injury. Similarly, Higher level of LDH independently is not dependable marker for CAD.

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