RESEARCH ARTICLE

Importance of Cardiac Marker Enzymes (Aspartate Transaminase and Troponin I) in Acute Myocardial Infarction: A Case-Control Study from South India

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ABSTRACT

Background: Cardiac marker enzymes are measured to evaluate the heart function. The diagnosis of acute myocardial infarction can be achieved by electrocardiogram (ECG) changes and elevation of cardiac marker enzymes like creatine kinase, lactate dehydrogenase (LDH), aspartate transaminase (AST) and troponin I.

Objective: To estimates the levels of AST and troponin I among patients of acute myocardial infarction, and to compare with those among health controls.

Materials and Methods: This study was carried out among 50 cases of acute myocardial infarction and 50 age and sex matched healthy individuals. Serum samples of cases, collected after 5 hours and within 24 hours from the onset of chest pain and of controls were analyzed for AST by modified IFCC method and for troponin I by chemiluminescence – sandwich method.

Results: The mean levels of AST and troponin I in cases and controls revealed that mean levels of AST and cardiac troponin I in cases (296.02±SD 135.69 IU/L and 57.34±SD 12.83 ng/ml, erspectively) are significantly higher than among controls (25.50±SD 6.22 IU/L and 0.31±SD 0.15 ng/ml, respectively). The differences between cases and controls are statistically significant ($p<0.0001$).

Conclusion: The diagnostic efficiency of AST and troponin I were superior because they are specific to myocardial injury.

Key Words: Cardiac Enzymes; Aspartate Transaminase; Troponin I; Myocardial Infarction

INTRODUCTION

Cardiac marker enzymes are measured to evaluate the heart function. The diagnosis of acute myocardial infarction can be achieved by electrocardiogram (ECG) changes and elevation of cardiac marker enzymes like creatine kinase, lactate dehydrogenase (LDH), aspartate transaminase (AST) and troponin I. The parameters like creatine kinase and LDH are unsatisfactory because of their isoforms and organ distribution.[1] So Serum enzyme AST and protein troponin I are the best indicators for cardiac muscle injury because of their early rise, i.e. 3-5 hours from the commencement of cardiac muscle injury and they have no isoforms. Hence the present study estimates the levels of AST and troponin I among patients of acute myocardial infarction, and compares with those among health controls.

METHODS

This study was carried out among 50 cases of acute myocardial infarction admitted in the hospital of the medical college of the primary authors. Fifty age and sex matched healthy individuals were served as controls. The serum samples were collected from all the patients after 5 hours and within 24 hours from the onset of chest pain. The samples from cases and controls were collected in vials containing no anti coagulant or preservative and care was taken to get non haemolysed samples. The samples were analyzed for AST by modified IFCC method using Dade Dimensions-Siemens Auto Analyzer with reference range of 8-37 IU/L and troponin I by chemiluminescence – sandwich method using Centaur CP-Siemens Auto Analyzer with reference range of ≤ 0.76 IU/L. All patients/or their family members and controls were informed and their consent was obtained.

RESULTS

The mean levels of AST and troponin I in cases and controls were shown in Table 1. The mean levels of AST in cases and controls are 296.02 ± SD 135.69 IU/L and 25.50 ± SD 6.22 IU/L, respectively. The difference between cases and controls is statistically significant ($p<0.0001$). The mean level of cardiac troponin I among cases (57.34 ± 12.83 ng/ml) is significantly higher than that of controls (0.31 ± SD 0.15 ng/ml) ($p<0.0001$).

Table-1: Levels of Aspartate Transaminase and Troponin I in Acute Myocardial Infarction Patients and Corresponding Age and Sex Matched Controls

Cardiac Marker Enzyme	Cases (Mean ± SD)	Controls (Mean ± SD)	Reference Values	p value
Aspartate Transaminase (IU/L)	296.02 ± 135.69	25.50 ± 6.21	8 – 37	<0.0001
Troponin I (ng/ml)	57.34 ± 12.82	0.31 ± 0.15	Upto 0.78	<0.00001

SD: standard deviation

DISCUSSION

Acute myocardial infarction is diagnosed traditionally based on the characteristic clinical history, ECG abnormalities and increased serum concentrations of cardiac marker enzymes like creatine kinase, LDH, AST and troponin I. As the differential diagnostic value is limited and ECG changes have various degrees of sensitivity and specificity[2], the measurement of serum enzymes plays an important role in diagnosis of acute myocardial infarction. The use of LDH in diagnosis of acute myocardial infarction is discouraged due to its non-specificity as its increased levels are found in progressive muscular dystrophy, myoglobinuria, leukemia, pernicious anemia, megaloblastic and hemolytic anemia, renal disease and in generalized carcinoma.[3] Creatine kinase, which usually rise within 6 hours of acute myocardial infarction is also unsuitable as a marker of myocardial injury because of its presence in large amounts in skeletal muscle and increased levels found in muscular dystrophy, hypothyroidism, hypothermia, alcoholism, cerebrovascular accidents and a variety of myopathies.[3] AST, an enzyme which is present in both mitochondria and cytoplasm released into blood when certain organs or tissues, particularly liver and heart are injured. The determination of AST was used to diagnose acute myocardial infarction. The amount of AST is directly related to the number of cells affected by disease or injury, but level of elevation depends on length of time of myocardial injury. Serum AST levels become elevated by 5-8 hours of cardiac cell injury, peaks at 24-48 hours and returns to normal level in 4-6 days. Serum AST levels are elevated markedly about 10-100 times the upper adult reference limit in acute myocardial infarction but a moderate increase in circulatory failure (shock), acute hepatitis and mild rise in severe hemolytic anaemia, post surgery/trauma and skeletal muscle diseases. Other myocardial injuries such as angina or pericarditis do not increase AST levels.[4] So AST determination has formally been used in diagnosis of acute myocardial infarction. The present study noted a highly significant change in AST level in myocardial infarction patients when compared

with controls with 4-10 times increase with reference limit. The present study findings are similar to that of Sobel and Shell, which reveals that true positive AST elevation occurred in 97% acute myocardial infarction.[4] It was proven by autopsy of 119 cases and of those, 115 had shown elevated AST levels. Intra cellular proteins are also potentially useful markers of acute myocardial infarction. Troponin I is a myocardial specific myofilament component, a monomer with molecular mass of 24 KDa.[5] It is an inhibitory polypeptide subunit of myofribillar regulating troponin complex. In cardiomyocytes, troponin I is compartmentalized into minor cytosolic (3%) and major myofribillar fraction (97%). And both these fractions are released during severe myocardial ischemia. Troponin I has found to increase in acute myocardial infarction after 3- 6 hours of the onset of chest pain, peak in about 12 hours and remain elevated for 3-10 days because of continuous release of myofilament components from the injured cardiac muscle.[6-8] Hence, minor myocardial injury is easily detectable by measuring cardiac troponin I but not by other conventional cardiac markers like creatine kinase and LDH.[9] Troponin I is myocardial specific and it is released as early as creatine kinase and remains elevated as long as LDH and has potential to serve as a sole marker for acute myocardial infarction. The present study noted a highly significant change in cardiac troponin I level with an increased levels in these patients. It showed almost 70 times rise compared to the reference limit and these observations are similar to the studies of Nigam[3], Adams et al.[10] and Chiu et al.[11].

In our study of 50 cases with acute myocardial infarction after 5 hrs and within 24 hours of myocardial injury, AST and troponin I levels were estimated where their levels are elevated when compared to 50 age and sex matched apparently healthy controls. It is inferred that the diagnostic efficiency of AST and troponin I were superior because they are specific to myocardial injury.

CONCLUSION

The diagnostic efficiency of AST and troponin I were superior because they are specific to myocardial injury.

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