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Original article

Studies on Cardiac Troponin I in patients with Cardiogenic Shock

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Abstract

Despite recent advances in the care of patients with acute coronary disease and the benefits associated with the early use of reperfusion strategies, cardiogenic shock as a complication of acute myocardial infarction continues to be associated with a dismal prognosis. There is a strong relationship between serum cardiac troponin I with cardiogenic shock as a complication of acute myocardial infarction. A case control study was designed to see the association of serum cardiac troponin I with cardiogenic shock. The study was done from July 2008 to June 2009. Sixty subjects were selected as study population which were taken from department of Cardiology, Mymensingh Medical College hospital, Mymensingh. Among them 30 were diagnosed case of cardiogenic shock and 30 were age and sex matched control. It revealed that the mean cardiac troponin- I levels in case group were 15.998±28.31 ng/ml and control group were 0.065 ± 0.08 ng/ml respectively. The study suggest that serum cardiac troponin- I level is significantly associated with cardiogenic shock.

Key words: Cardiac Troponin I, Cardiogenic shock, Myocardial infarction.

Introduction:

When the pumping function of the heart is impaired to the point that blood flow to the tissues is no longer adequate to meet resting metabolic demands, the condition that results is called cardiogenic shock. It is most commonly due to extensive infarction of the left ventricle, but it can also be caused by other diseases severely compromise ventricular function[1]. The incidence of the cardiogenic shock in patients with myocardial infarction is about 10%, and it has a mortality of 60-90%[2].

Troponin is a component of thin filaments (along with actin and tropomyosin), and is the protein to which calcium binds to accomplish this regulation. Troponin has three subunits, TnC, TnI, and TnT. When calcium is bound to specific sites on TnC, tropomyosin rolls out of the way of the actin filament active sites, so that myosin (a molecular motor organized in muscle thick filaments) can attach to the thin filament and produce force and/or movement. In the absence of calcium, tropomyosin interferes with this action of myosin, and therefore muscles remain relaxed[3].

Troponin is released into the bloodstream 4 to 6 hours after AMI, peaks after approximately 18 to 24 hours, and can stay elevated for up to 14 days. Assessment of cTnI or cTnT by automated assay is today the most sensitive and specific method for diagnosing AMI. However, cTn is not released only in response to ischemic insults but by any condition that is associated with and/or causes cardiac injury, eg, decompensated heart failure, pulmonary embolism, end-stage renal disease, and stroke[4].

Cardiogenic shock is highly lethal, but the likelihood of developing shock after acute MI clearly can be reduced by modern multimodal treatment, beginning in the emergency department[5]. Increased level of serum cardiac troponin I may

be an important factor associated with cardiogenic shock.. So, the present study was carried out to see the association of serum cardiac troponinI with cardiogenic shock.

Material and Method:

The study was carried out in the Department of physiology, Mymensingh Medical College, Mymensingh (MMC) during the period of July'2008 to June 2009. The patients were taken from the department of cardiology MMC, Mymensingh. Cardiogenic shock patients were considered as case and the control were age & sex matched healthy volunteers. Cases were the patients who clinically suffered from Cardiogenic shock admitted in the department of cardiology, Mymensingh Medical College hospital, Mymensingh during the study period. In this study sample size were taken as 60. Thirty Cardiogenic shock patients were taken as cases and 30 healthy volunteers were taken as controls. Data were analyzed by computer with the help of SPSS version 12 software package. All data were recorded systematically in a preformed data collection sheet. Mean values of the findings were compared between two groups. Pearson's 'χ 2' test was performed to justify the relation between cardiac troponin I with cardiogenic shock in study & control group. The correlation co-efficient between age, and Cardiac Troponin-I also studied for both control and study group. For all the statistical analysis 2 - tailed 'p' values < 0.05 were considered as significant.

Results :

The study showed that the mean Serum Cardiac Troponin-I was 0.065 ng/ml in control group. Where as the mean Serum Cardiac Troponin-I value was 15.998 ng/ml in the study group. There was highly significant difference of mean Serum Cardiac Troponin-I level between the case and control group ($p < 0.033$) (Table-I). The study showed the correlation coefficient between age and Serum Cardiac Troponin-I of control group was -0.003 and 0.45 in study group. This implies if age increases than MI as well as cardiogenic shock increases. (Table-II).

Table-1: Serum Cardiac Tropin-1 level of study group and control group

Group	Mean	SD	χ□2
Study group	15.998	28.312	16.738
Control group	0.065	0.079	

SD = Standard Deviation
Level of significance was 0.05.

Figure-1:
Serum Cardiac Troponin –I values in Study and Control Group

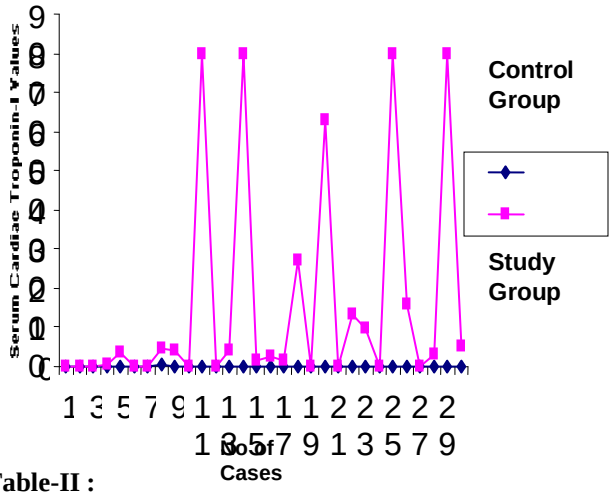

Table-II :
Comparative study of correlation coefficient between control and study group

Variables	Control	Study
Age & Serum cardiac troponin-I	r = -0.003	r = 0.45

r = correlation coefficient

Discussion:

The cardiac-specific isoform of troponin I (Cardiac Troponin I) has been known as a marker of heart damage for more than 10 years. At present Cardiac Troponin I is considered to be one of the most specific and sensitive markers of myocardial cell death. However, clinical chemists and physicians diagnosing and treating acute myocardial infarction (AMI) patients are puzzled by the up to 10-fold differences in cut off values and even larger differences in measured concentration obtained by different assays[6].

The criteria for diagnosing acute coronary syndrome and myocardial infarction (AMI) changed in the year 2000 with the endorsement of the American College of Cardiology/European Society of Cardiology guidelines, which designated cardiac troponin (cTn) as the biochemical marker of choice[7].

Cardiac troponin T (cTnT) and I (Cardiac Troponin I) are highly specific and sensitive biomarkers of myocardial cell damage and are now accepted as the 'gold standard' diagnostic test for acute coronary syndrome and supersede the classical muscle enzyme biomarkers[8].

In the present study, the mean serum cardiac troponin I level was 0.065 ng/ml in control group. Where as the mean Serum

Cardiac Troponin-I value was 15.998 ng/ml in the study group. There was highly significant difference of mean Serum Cardiac Troponin-I level between the case and control group ($p < 0.033$) Gensini et al. (1998)[9] in their study found Eight patients had Q-wave PMI. All PMI patients had elevated peak cTnI values (all >9.2 ng/mL), whereas the 34 nonPMI patients had peak values <9.0 ng/mL; therefore, sensitivity and specificity (with a 9.0 ng/mL cut-off value) are 100%. MB-CK measurement peak values did not demonstrate such a high specificity and sensitivity[10].

The study showed the correlation coefficient between age and Serum Cardiac Troponin-I of control group was -0.003 and 0.45 in study group. This implies if age increases MI as well as cardiogenic shock increases. Thus, the result of present study is consistent with the previous studies and showed a correlation between inccrease cardiac troponin I levels with cardiogenic shock.

Conclusion:

Serum cardiac troponin I level is an important determinants of myocardial infarction. Analytical results of this study reveals that the patients of cardiogenic shock have been found to have close association with increased level of serum cardiac troponin I. So in-order to prevent cardiac morbidity and mortality estimation of serum cardiac troponin I is essential.

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