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RESEARCH ARTICLE

SERUM LEVELS OF TROPONIN T AS SENSITIVE MARKERS IN EARLY DETECTION OF (ACUTE MYOCARDIAL INFARCTION) AMI

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ABSTRACT

The troponins are regulatory proteins found in skeletal and cardiac muscle. Most of the early markers identified were enzymes, and as a result, the term "cardiac enzymes" is sometimes used. However, not all of the markers currently used are enzymes. Cardiac biomarkers should be measured in all patients who present with chest discomfort consistent with acute coronary syndrome. Elevations of cardiac enzyme levels should be interpreted in the context of clinical and ECG findings. In this study, cTnT levels of 50 AMI patients were compared with the 25 normal blood donors. We observed 20 times increase in cTnT levels in AMI patients as compared to normal donors. Cardiac troponins T and I are the preferred markers for myocardial injury as they have the highest sensitivities and specificities for the diagnosis of acute myocardial infarction. Peak circulating enzyme levels tend to occur earlier and are often higher following successful thrombolytic therapy.

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INTRODUCTION

Cardiac markers are biomarkers measured to evaluate heart function. Most of the early markers identified were enzymes, and as a result, the term "cardiac enzymes" is sometimes used. However, not all of the markers currently used are enzymes. For example, in formal usage, troponin would not be listed as a cardiac enzyme. (Rao *et al*., 1999) An early diagnosis is essential for therapeutic decision and risk stratification in patients with suspected acute myocardial infarction (AMI). (Kitamura *et al*., 2013) Cardiac troponins T and I are the preferred markers for myocardial injury as they have the highest sensitivities and specificities for the diagnosis of acute myocardial infarction. (Daubert and Jeremias, 2010) Cardiac troponin I and T have displaced myoglobin and creatine kinase-MB as the preferred markers of myocardial injury. (Christenson and Christenson, 2013) Troponin is a protein released from myocytes when irreversible myocardial damage occurs. It is highly specific to cardiac tissue and accurately diagnoses myocardial infarction with a history of ischaemic pain or ECG changes reflecting ischaemia. Cardiac troponin level is dependent on infarct size, thus providing an indicator for the prognosis following an infarction. (Chan and Ng, 2010)

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New high-sensitivity cardiac troponin assays have been developed that can measure troponin values at much lower levels. With the use of these high-sensitivity assays, more patients with unstable angina will be classified as having non-ST-elevation myocardial infarction. These assays may therefore define a high-risk patient population and may lead to more appropriate therapy and improved outcomes in these patients. (McCord, 2013)

Aim

To study the levels of Cardiac Troponin T in patients of Acute Myocardial Infarction. To compare the cTnT levels between AMI patients and normal blood donors.

MATERIALS AND METHODS

The study carried out on the patients admitted to the ICCU diagnosed as myocardial infarction in MGM medical college, Aurangabad. The blood sample was collected at zero hours (i.e. at the time of admission of the patient) after an informed and written consent from all the patients. The blood samples were collected in vials containing no anticoagulant or preservative and were analysed for Cardiac Troponin T. The study consist of 50 patients with AMI out of which 12 were females and 38

were males. Twenty five subjects were selected from normal blood donors as controls. The serum Cardiac Troponin T concentration was measured using the reagent kit manufactured by Boehringer Mannheim based on enzyme linked immunoassay. Values were expressed in mean± SD. Data was analysed using student paired t test. A p value <0.05 was considered as statistically significant.

Table 1. Mean±SD values of Cardiac Troponin T in AMI patients and normal blood donors

Parameter	AMI patients(ng/ml)	Normal Blood Donors(ng/ml)	p value
Cardiac Troponin T	2.48±1.24	0.03±0.01	0.001

Table 2. Mean ±SD values of cTnT in males and females

Parameter	Males(ng/ml)	Females (ng/ml)
Cardiac Troponin T	2.01±0.98	1.79±0.73

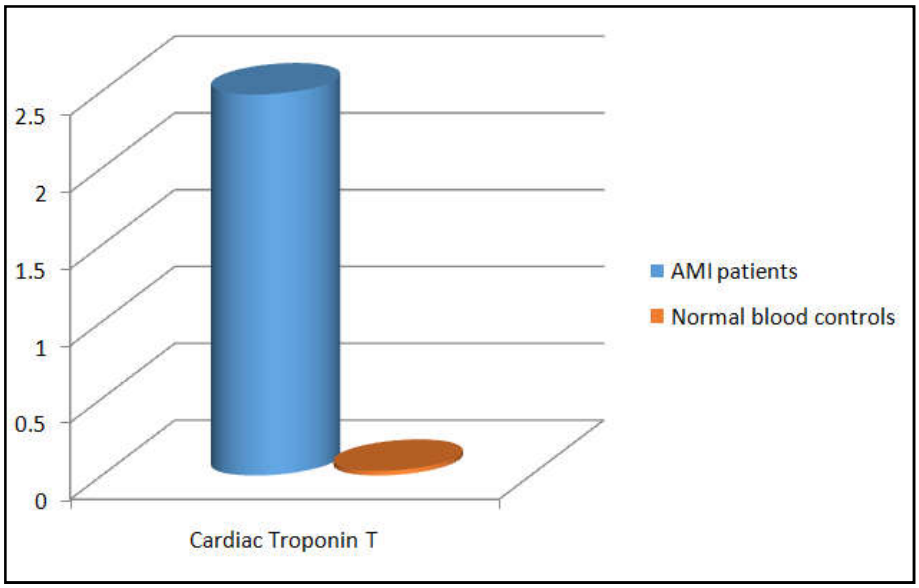

Figure 1. Mean values of Cardiac Troponin T in AMI patients and normal blood donors

RESULTS AND OBSERVATION

Cardiac Troponin T (cTnT) was elevated at the time of admission of the patient itself. This increase of cTnT in AMI patients was 20 times higher than the normal blood donors and was statistically significant. (Table and Figure 1) The controls included 25 normal blood donors. The study shows that cTnT estimation could serve in the early diagnosis of AMI. The increase of cardiac troponin T in AMI patient was 20 times higher than the normal blood donors in AMI patients at the time of admission. Table 2 shows that mean cTnT in more in males as compared to females. Cardiac troponin T in serum appears to be a more sensitive indicator of myocardial cell injury than CKMB activity and its detection in the circulation may be a useful prognostic indicator in patients with unstable angina as well. When the blood of normal blood donors was analysed the troponin T values were well within the normal range in both the above categories showing that cardiac troponin T is highly specific for heart tissue. Although CKMB and cardiac troponin T are released soon after the myocardial injury, the release of cardiac troponin T is much earlier than CKMB thereby invalidating the important role of cardiac troponin T in diagnosing AMI. Cardiac troponin T has been shown to be highly sensitive for cardiac injury and not elevated in any other trauma, heavy exercise or skeletal muscle injury. Cardiac troponin T is ordinarily undetectable in healthy individuals, and so its measurement can serve as a powerful tool in the diagnosis of AMI.

DISCUSSION

The diagnosis of acute myocardial infarction (AMI) has traditionally been based on the characteristic clinical history, electrocardiographic abnormalities and increased serum concentrations of cardiac marker enzymes. As the differential diagnostic value of chest pain is limited (Rude *et al*., 1983) and the electrocardiographic changes have various degrees of sensitivity and specificity (Drexel *et al*., 1983), the measurements of serum enzymes as a reflection of damage to myocardial muscle cells still play an important role in the diagnosis of AMI. Measurements of creatine kinase (CK), aspartate aminotransferase, and lactate dehydrogenase are well-established methods for this. Considerable effort has been made in recent years to improve the specificity and sensitivity of methods for diagnosing AMI. Myoglobin is an early and sensitive marker of cardiac cell damage but lacks specificity (Eisenberg and Kielley, 1974). The use of LDH isoenzyme 1 improves specificity in diagnosing AMI; however, this isoenzyme is not restricted to cardiac muscle tissue, and increase in its serum concentration has been observed in

noncardiac conditions also (Katz, 1977). Clinicians wouldtherefore benefit from the introduction of a new cardiac specific marker of damage to myocardial muscle cells. The contractile and regulatory proteins of the myocardium provide such a useful diagnostic tool. Troponin T (TnT) is a part of the troponin complex in striated muscles, where it binds the troponin complex to tropomyosin. Although both cardiac and skeletal muscles contain TnT, the amino acid sequence of the protein in the two types of muscles differs (Mair *et al*., 1991; Basil and William, 1988), making it possible to raise antisera against cardiac-specific TnT. The high specificity and sensitivity of cardiac TnT in diagnosing and monitoring AMI had been demonstrated by Johannes Mair *et al*. (1991). Troponin T is a part of the regulatory system of the contractile complex of skeletal and heart muscle. It is expressed in two different isoforms, namely skeletal muscle troponin T and cardiac troponin T. After loss of the integrity of the cell membrane, it is released into the circulation similar to myoglobin or creatine kinase (Rude *et al*., 1983). Because cardiac troponin T is ordinarily undetectable in healthy individuals its measurement is a powerful tool in the diagnosis of acute myocardial infarction. Moreover, about onethird of patients with unstable angina pectoris show increased cardiac troponin T in serum . These patients may be a subgroup with a high risk for complications i.e. acute myocardial infarction (Katz, 1977). After myocardial cell necrosis an increased concentration of cTnT is observable in blood for more than a week. Thus cardiac Troponin T measurement is particularly useful in clinical circumstances in which traditional enzyme determiations fail to diagnose myocardial cell damage efficiently. (Basil and William, 1988; Adams *et al*., 1993; Gerhardt *et al*., 1992)

Conclusion

Troponin is released during MI from cytosolic pool of the myocytes. Its subsequent release is prolonged with degradation of actin and myosin filaments. Isoforms of the protein, T and I, are specific to myocardium. Differential diagnosis of troponin elevation includes acute infarction, severe pulmonary embolism causing acute right heart overload, heart failure, myocarditis. Troponins can also calculate infarct size but the peak must be measured in the 3rd day. After myocyte injury, troponin is released in 2-4 hours and persists for up to 7 days.

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