INTRODUCTION
Cardiac injury is referred to as some form of cardiac insult, which may encompass myocardial cell injury and/or cell death. The causes of cardiac injury are multifarious, including ischemia, direct trauma to the heart, drug-induced myocardial toxicity, viral myocarditis, end-stage renal failure, congestive heart failure and pulmonary embolism [1,2]. Early diagnosis of cardiac injury is crucial in planning the treatment modalities and other therapeutic interventions, which are of primary importance in reducing morbidity and mortality. Earlier, diagnosis relied on clinical history, symptoms and ECG changes. Although ECG is more objective than clinical history and symptoms, it lacks sensitivity and specificity [3]. The emergence of cardiac biomarkers gained a high degree of significance in the early diagnosis of cardiac injury. During cardiac injury, the disruption of normal cardiac myocyte membrane integrity results in the release of intracellular constituents including detectable levels of a variety of biologically active cytosolic and structural proteins such as cardiac troponin (cTn), creatine kinase-myocardial band (CK-MB), myoglobin, heart-type fatty acid binding protein (H-FABP), heart specific C-reactive protein (CRP), B-type natriuretic peptide (BNP), lactate dehydrogenase (LD), etc., into the extracellular space [4]. Early and specific diagnosis can be made possible by the determination of these cardiac markers. Limitations of existing cardiac biomarkers has led to the search for markers uniquely expressed by the myocardium.

From a clinical point of view, an ideal cardiac biomarker that detects myocardial injury should satisfy the following properties:

- It should be present in myocardium in high concentration and absent in other tissues thereby ensuring high cardiac specificity.
- It should be released rapidly in blood stream after myocardial injury, so as to achieve optimal sensitivity in early phase after the onset of myocardial injury.
- It should remain abnormal for several days thereby offering wide diagnostic window time.

The release of cardiomyocyte components like cardiac specific biomarkers into the blood stream in higher than normal quantities indicates cardiac injury. The
The advent of cardiac troponins (cTn), unarguably the most sensitive and specific marker encompass all the requirements for accurate staging and better risk stratification of patients with acute coronary syndrome when compared to other existing cardiac biomarkers. Troponins are regulatory proteins found in skeletal and cardiac muscle. The troponin complex modulates calcium-mediated actin and myosin interaction in striated muscle. The cardiac troponin complex is made up of 3 subunits that have been identified as troponin I (inhibitory), troponin C (calcium binding), and troponin T (tropomyosin binding) proteins. The genes that code for the skeletal and cardiac isoforms of troponin C (TnC) are identical; thus, no structural difference exists between them [5]. However, the skeletal and cardiac subforms for troponin I (TnI) and troponin T (TnT) are distinct and structurally different and immunoassays have been designed to differentiate between them.

cTnI indeed scores over cTnT in the specificity aspect because cTnI is released only during myocardial cell damage and not released in normal or injured or regenerating skeletal muscle [6]. The cardiac isoform of Troponin I (cTnI) has a molecular weight of 24 kDa and it has an extra 30 amino acid sequence at the N terminal portion of molecule making it absolutely specific to cardiac muscle. Some of the unique characteristics of cTnI are a wide diagnostic window time with early appearance of 4 to 8 hours after symptom onset and prolonged presence (7 to 10 days) in circulation and allows detection of even minor myocardial injury because cTnI levels is almost absent in normal healthy individuals [7]. It is reported that cTnI and cTnT are useful at ruling out acute myocardial infarction (AMI) when the value is negative at 10 or more hours from the onset of chest pain and a positive cTnT value is only moderately useful whereas cTnI value appears to be highly useful at ruling in AMI at 6 hours from the onset of chest pain and therefore cTnT appears to be better at ruling in MI. Thus cTnI determination promises higher diagnostic efficacy than cTnT [8]. The present study was carried out to evaluate the role of cardiac troponin I for the diagnosis of cardiac injury and to assess its utility as a confirmatory test for myocardial injury in comparison to other available cardiac markers.

**MATERIALS AND METHODS**

This study was conducted at a private hospital in Chennai. Clinical data of 150 patients with suspected MI reporting within 24 hours of onset of symptoms was analysed. The study period was between January 2012 to April 2012. Details regarding age, sex and symptoms pertaining to chest pain along with detailed clinical history and ECG were recorded. Blood samples were collected from the patients and the qualitative analysis of cardiac troponin I was determined by Rapha’s Veda Lab Troponin I qualitative assay in serum in vitro (Kit method). This is a rapid qualitative one step assay for the detection of cTnI in serum and it employs a unique combination of monoclonal dye conjugate and polyclonal solid phase antibodies to identify Troponin I in the test sample with a high degree of sensitivity of 1ng/ml. The results were analysed and correlated with age and clinical symptoms.

**RESULTS AND DISCUSSION**

The qualitative analysis of cTnI determination in the patients showed 28% positive cases during the study period. Correlation between age and troponin positive cases is outlined in table 1. Highest positive cases (50%) was observed in the age group of 71-80 years, followed by 33.33% positive cases among the patients in the age group of 51-60 years and 25% positive cases among patients in the age group of 61-70 years, indicating an age-related correlation between positive cases for myocardial infarction and troponin I determination. In the other age groups studied 31-40, 41-50 and 81-90 years, cTnI was found to be negative.

<table>
<thead>
<tr>
<th>S. NO.</th>
<th>AGE GROUP (YEARS)</th>
<th>PATIENTS WITH TROPONIN +ve (in %)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>31-40</td>
<td>0%</td>
</tr>
<tr>
<td>2.</td>
<td>41-50</td>
<td>0%</td>
</tr>
<tr>
<td>3.</td>
<td>51-60</td>
<td>33.33%</td>
</tr>
<tr>
<td>4.</td>
<td>61-70</td>
<td>25%</td>
</tr>
<tr>
<td>5.</td>
<td>71-80</td>
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<tr>
<td>6.</td>
<td>81-90</td>
<td>0%</td>
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</tbody>
</table>

Cardiac troponins are contractile proteins of the myofibril. The cardiac isoforms are very specific for cardiac injury and are not present in serum of healthy people. Since, they are normally not present in serum unless cardiac cell necrosis has occurred, they are more cardiac specific than CK-MB and this enhanced specificity allows for more accurate diagnosis of cardiac injury [7]. Cardiac troponins which are sensitive markers of myocardial necrosis is an important component of risk stratification and are useful in the prediction of therapeutic efficacy for pharmacological and percutaneous interventions [9]. Recent guidelines (2007) from the American College of Cardiology Foundation (ACCF)/ European Society of Cardiology (ESC) state that cardiac troponins are the preferred markers for detecting myocardial cell injury. Ohman et al. [10] reported that, using troponin I there was a 95-97% chances of detection of patients who were ultimately shown to have a myocardial infarction. Hence the present study was done to assess the utility of troponin I in high risk group with reference to age reporting with symptoms of suspected myocardial infarction. Since it is only a qualitative assay of troponin I and as no follow up was done to trace the outcome of these patients, it is not possible to highlight actual sensitivity and specificity of this test in the confirmatory diagnosis of myocardial infarction. Quantitative determination of troponin I would serve as a more appropriate indicator of actual myocardial injury as reported in various studies [4,10]. Cardiac troponin elevation in blood is integral to the diagnosis.
of myocardial infarction, the joint ESC/ACCF/AHA/WHF Task force for the universal definition of myocardial infarction had advocated diagnosis of acute myocardial infarction based on a rising and/or falling pattern of cardiac troponins in the appropriate clinical situations [11].

Concerning the clinical significance of elevated troponin levels in the diagnosis of myocardial damage, there are both cardiac and non-cardiac conditions in which their levels may also be increased and this needs to be carefully evaluated before a confirmatory diagnosis could be made. A raised cardiac troponin concentration is not just confined to myocardial injury from coronary plaque rupture or occlusion, but is also increased in many other conditions associated with cardiac arrhythmias, large pulmonary emboli, cardiomyopathy, sepsis and chemotherapy [4]. Metra et al. [12] found that any elevated cTn over the course of acute heart failure hospitalization conferred substantially increased risk. Raised serum concentrations of cardiac troponins represent myocardial damage; however, this does not necessarily equate to myocardial infarction. It remains for the clinician to distinguish whether a raised cardiac troponin concentration is the result of coronary plaque rupture/occlusion or whether it has any other cause. The specific diagnosis of the cause of myocardial damage can only be made after detailed clinical assessment, which should include a clinical history and serial ECG recordings. A raised cardiac troponin alone will result in a clinical diagnosis, although one cannot detract from the fact that cardiac troponin measurements have been an invaluable step forward in the identification of high risk patients with acute coronary syndromes [13]. In recent years, serum troponins have been increasingly used in the diagnosis of acute coronary syndromes as studies have shown their greater clinical sensitivity over creatine kinase-MB (CK-MB). In patients with non-Q-wave myocardial infarction (MI) or unstable angina, serum troponins can provide risk stratification for short-term and long-term cardiac events and mortality. This has been attributed mainly to the ability of serum troponins to detect microinfarcts, areas of necrosis too small to produce electrocardiographic changes, etc [14]. This study is a preliminary attempt to evaluate the significance of cardiac troponin I in patients with suspected myocardial infarction presenting with clinical symptoms like chest pain, etc. However, only qualitative determination of cTnl was performed. In order to substantiate and confirm cardiac injury/damage, quantitative estimation of cTnl is required along with consideration of other clinical parameters. As suggested by various studies, cTnl assay individually is a powerful, independent mortality risk factor in patients with acute myocardial infarction and hence would be of value in confirming its diagnosis as well as a prognostic tool in heart failure [10,15].

The use and interpretation of cardiac biomarkers such as troponin I depends on the specific clinical setting and what information is being sought. The type and stage of development of the cardiac disease pose difficulty in the development of an appropriate and specific biomarker and their interpretation. There is no doubt that knowledge about cardiac biomarkers will continue to evolve with technology and in the future would provide valuable information in screening, diagnosis, monitoring and prognosis of cardiac disease.

REFERENCES


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