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Original Research Article

Cardiac Troponin-T levels in heart blood as a marker to diagnose postmortem myocardial infarction

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Abstract

Cardiac deaths account for 50% of all deaths in developed and 25% in the developing world. One-sixth of world's population lives in India and heart disease accounts for 24% of all deaths. Sudden death accounts for two-thirds of all autopsies in Forensic Medicine. Actual detection of histological sequence of the infected myocardium will develop only after significant time of, between onsets of myocardial infarction (MI) in death. Cardiac Troponin-T is not normally present in serum unless cardiac necrosis has occurred therefore cardiac Troponin levels act as a specific and sensitive indication of myocardial infarction. The present study was conducted on cases coming for medico legal autopsy to the Forensic Medicine Department at Gandhi Medical College/Hospital, Hyderabad, Andhra Pradesh, India for a period of 1 year from January 2014 to December 2014. Total 12 cases with 6 controls were analyzed. Cardiac Troponin-T Level was markedly elevated >2.000 ng/ml in all except one case of suspected MI. Sensitivity was found to be 91.66% and specificity 66.66%.

Key words

Heart disease, Cardiac Troponin-T, Myocardial infarction.

Introduction

In 21st century, cardiac deaths accounted for 50% of all deaths in the developed and one fourth in the developing world. It is accepted universally that myocardial infarctions are fore most killers and destroyers of mankind today [1]. By 2020, heart disease will lead to 25 million deaths all over the world annually [2]. One-sixth

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of world's population lives in India and heart disease accounts for 24% of all deaths [3]. Indian people have been found to have the unfortunate distinction of having the highest prevalence of coronary artery disease among all ethnic groups in the world, the projected rise in the mortality rates (>100%) in the next 25 years [4].

Sudden death accounts for approximately two-thirds of autopsies in forensic medicine [5]. Natural death within 1 hour after the beginning of acute symptoms is defined as sudden cardiac death [6]. Due to ventricular fibrillation caused by myocardial irritability induced by ischemia or infarction. Acute myocardial infarction (AMI) is a serious and potentially lethal manifestation of coronary artery disease, affecting more than 7 million people worldwide each year and proved to be a cause of sudden death [7].

In postmortem examination of dead bodies subjected to autopsy with probable sudden cardiac death, a diagnosis of myocardial infarction is usually made based on the finding of severe atherosclerosis occlusive coronary artery disease. Actual detection of histological sequelae of the infracted myocardium will develop, only after significant time lag between onset of myocardial infarction and death [8]. Detectable loss of lactate dehydrogenase will be seen 5 hours after infarction using enzyme histo-chemistry [9].

Recently several studies have shown keen interest on the application of biomarkers to diagnose AMI. The presence of cardiac biomarkers in blood with increase sensitivity to detect cardiac cell necrosis enables to diagnose AMI in 1/3rd of patients who might not had accomplished the criteria for diagnosis of myocardial infarction [10].

Troponin is a protein complex, situated on the actin filament and regulates calcium mediated interaction of actin and myosin filaments during muscular contraction. It has three subunits cTnI (inhibitory), cTnT (Trophomyosin Binding) and TnC (calcium Binding). Cardiac Troponin T is the double filament protein and predominantly this protein is bound within myocytes, and less than 10% is dissolved in the cytosol [11].

Cardiac Troponin T is not normally present in the serum unless cardiac cell necrosis has occurred. Thus it is more cardiac specific. Recent studies have shown that cardiac Troponin levels act as a specific and sensitive indicator of myocardial infarction [12].

The prognostic value of cardiac Troponin T is most important as it is not dependent on age, sex, ECG changes as well as levels of age old biochemical markers such as Creatine Kinase (CKMB). Cardiac Troponin T (cTnT) is elevated in all patients with acute myocardial infarction diagnosed by World Health Organization (WHO) criteria. Increased levels of cardiac Troponin T appears in the blood within 3-24 hours after AMI depending on factors such as infarct size and they can be estimated for up to 2 weeks in the living [13].

The cardiac Troponin-T assay is based on monoclonal antibody system using a poly-(streptavidin)-biotin capture system with a ruthenium complex cTnT in the blood sample or plasma combines with both the biotinylated anti-Cardiac Troponin T and anti-Cardiac Troponin T and antibody [14].

The European Society of Cardiology (ESC)/ American College of Cardiology (ACC) guidelines recommend using 'the 99th percentile of a healthy population as a cut-off for AMI using an assay with an acceptable precision. An acceptable precision has been defined as a co-

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ISSN: 2394-0034 (O)

efficient of variation < 10%. For cardiac Troponin T, the 99th percentile value of a healthy population is 0.1ng/ml; however, the 10% coefficient of variation requirement for the usual assay was met at a higher level (i.e. 0.3 ng/ml) [15]. In clinical practice, the cTnT assay has been approved for the diagnosis of AMI with high sensitivity and specificity [16].

Aim and objectives

- To estimate postmortem cardiac Troponin T levels in the heart blood to diagnose acute myocardial infarction in autopsy.
- To correlate cardiac Troponin T levels with histopathological diagnosis of acute myocardial infarction in autopsy.

Material and methods

Present study was prospective study conducted at Forensic Medicine Department, Gandhi Medical College/Hospital, Andhra Pradesh, India from January 2014 to December 2014.

Inclusion criteria

A sudden death, adult dead body with cause of death unexplained by external examination (body was found dead at home, outdoors or in the hospital emergency room). Autopsy was done within 48 hours of death.

Exclusion criteria

- All dead bodies subjected for autopsy after 48 hours of death.
- Dead bodies found to have septicemia, renal disease, pulmonary embolism, myopericardtis, and congestive heart failure.
- Dead bodies found to undergone cardiac surgery or cardio pulmonary resuscitation or cardiac concussion.

The internal autopsy was done for 12 sudden death bodies and 6 controls consisting of deaths due to poisoning, stroke, Tuberculosis etc.

Hearts were exposed and blood was withdrawn from the pericardial cavity, right and left chamber of the heart with a 21G sterile syringe with needle and immediately transferred to a sterile sampling anticoagulant added test tube and sent for analysis at Department laboratory for the measurement of cardiac Troponin T levels with cobase 232 “SANDWICH” Analyzer.

Test principle

Sandwich principle and total time duration of assay was 9 minutes.

Cobase 232 analyzers

- **1st incubation:** 50 µL of sample, a biotinylated monoclonal anti-cardiac Troponin T-specific antibody, and a monoclonal anti-cardiac Troponin T-specific antibody labeled with a ruthenium complex react to form a sandwich complex.
- **2nd incubation:** After adhesion of streptavidin-coated micro particles, the complex becomes bound to the sold phase via interaction of biotin and streptavidin.

Cobase 232avec scanner

- During a 9 minute incubation period, the antigen in the sample (50 µL), a biotinylated monoclonal anti-cardiac Troponin T-specific antibody, monoclonal anti-cardiac Troponin T-specific labeled with a ruthenium complex and streptavidin-coated micro particles reacted to form a sandwich complex, which was bound to the solid phase. The reaction mixture was seen in strip method. Cardiac Troponin T levels were above 2.000 ng/ml.

ISSN: 2394-0026 (P)
ISSN: 2394-0034 (O)

The autopsy was followed by microscopic study. Once the heart was removed it was washed with running water and weighed. Gross examination of the entire heart was done to look for the presence of scar due to old infarct then after serial sections of coronary artery were made at a distance of 3 mm to look for presence of plaques or thrombus. Serial transverse sections involving full thickness of heart was made at a distance of 1 cm each from the apex to atrioventricular (AV) groove. Slices were examined for old fibrotic scar and softening. All the samples were sent for histopathological examination. Histopathological diagnosis of acute myocardial infarction was made from following criteria, which included wavy myofibers, coagulative necrosis, ischemic contraction, band necrosis, and polymorphous infiltration into the interstium.

Results

The study and control sample contained 11 and 5 men respectively and one woman in each group. The average age of the control sample was 50 (48-56) years and the average age of the cardiac death sample was 52 (44-60) years. In the control sample, two patients died due to acute organophosphorus poisoning. Three cases died due to cerebro-vascular accident and or metabolic encephalopathy, and one was due to pulmonary tuberculosis. Cardiac Troponin T level was markedly elevated (>2.000 ng/ml) in all except one case of suspected MI in the study sample and also markedly elevated in the control sample except two cases (probably due to autolysis).

Histopathological examination (HPE) of the hematoxylin and eosin stained, heart slices, showed that four cases of the study group had evidence of early myocardial infarction with features of myonecrosis, waviness of fibers, nucleomegaly, pyknotic nuclei and polymorphous infiltration. Three cases of acute MI deaths demonstrated, myocardium with multiple foci of infarction showing hyalinized collagen fibers. In areas of old infarction, HPE revealed evidence of dense fibrous tissue replacing normal myocardial tissue. Four of the control cases showed mild eosinophilia of the myocardial fibers with mild infiltration of polymorphs.

Microscopic features of MI

The microscopic appearances of human myocardial infarcts were complex as per **Table - 1**.

Cardiac Troponin T concentration levels in heart blood and pericardial fluid by highly sensitive Cardiac Troponin T quantitative assay were as per **Table – 2** and **Table - 3.**

Histopathological examination results were as per **Table – 4** and **Table - 5.**

Sensitivity and specificity of cardiac Troponin T in study and control groups

$$\text{Sensitivity} = \frac{\text{Total MI cases with positive cTnT test/ HPE results}}{\text{Total of all MI cases tested}}$$

Sensitivity = 11÷12 x 100 = **91.66%**

$$\text{Specificity} = \frac{\text{Total non MI/healthy cases with negative cTnT test/HPE results}}{\text{Total of all healthy cases tested}}$$

Specificity = 4÷6x100 = **66.66%**

Discussion

World Health Organization (WHO) defines sudden death as those that occurs within 24 hours of onset of terminal illness. 80% of sudden cardiac deaths are due to coronary atherosclerosis. Establishment of cause of death

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ISSN: 2394-0034 (O)

in cases of sudden cardiac death is a challenging task to the Forensic Pathologist. Identification of early myocardial infarction during autopsy pose difficulty, as apparent gross change of infarction takes 24 to 48 hours following occlusion of major artery in humans [1].

Even though minimal microscopic evidence is recognized as early as 6 hours, in the absence of gross changes the involved area may be missed when random blocks are taken for histo pathological examination. Very few studies are conducted on human heart during autopsy for detection of early myocardial infarction [2]. The rationale of using the measurement of a protein in blood to detect injury to cells is straightforward and requires consideration of a few major factors as mentioned below [3].

Criteria for a blood marker of cell death

I. Sensitivity
   - Abundance in cell
   - Location in cell

II. Sample timing
   - Mode of entry into blood
   - Half-life of elimination

III. Specificity
   - Distribution in different cells/organs

The myocyte is the major cell in the myocardium, and the heart's action is to pump blood. Because myocytes essentially cannot be regenerated, if heart cells die, then cardiac function has a high probability of being impaired. When the cell dies, the proteins inside the cell will be released, with proteins in the cytoplasm leaving the cell more rapidly than ones in the membranes or fixed cell elements. The most sensitive markers should be those in highest abundance in the cell, the proteins involved in contraction and producing the energy to support it should be good candidates for biomarkers of cardiac injury which could be detected in blood. Also, one has to consider the means by which the markers can reach the blood. Since occlusion of blood flow is the primary cause of myocardial infarction, most of the proteins reach the blood via the lymphatics where they are prone for degradation leading if the protein has a cardiac specific form. While utilizing abundant cardiac proteins involved in contraction or energy production seems obvious, it is not exactly the way the field evolved. Today, we can identify candidate cell injury markers via gene expression or proteomics in a reasonably straightforward manner. However, these are recent advances which required sequencing entire genomes and the development of technologies which were not available until recently.

The aim of this short study was to ascertain the relationship of deaths due to acute MI between cardiac Troponin T levels and correlate them with the HPE findings. Limitations of this study included factors like cohort size, autolysis, variation in time since death, and cold storage duration. In spite of these shortcomings, the data was analyzed that may help our understanding of cardiac Troponin T [6].

At the outset, it is inevitable that under normal conditions almost all bodies subjected for postmortem examination will demonstrate some degree of autolysis and hemolysis which will cause a rise of cardiac Troponin T levels above their premortem levels as free hemoglobin interferes with assays for measuring Troponin, CK, and CK-MB [7, 8].

In this study, from only two cases of heart blood, cTnT level from the control sample was within normal range for living patients, while the remaining samples showed high levels, may be due to autolysis that would be false positive of AMI in a living patient similar to other studies [16, 17].

ISSN: 2394-0026 (P)
ISSN: 2394-0034 (O)

Others have reported levels similar to living persons and have dismissed the role of hemolysis and autolysis. Some have propounded that cardiac Troponin T levels may be elevated in many deaths because of nonspecific cardiac injury due to lack of oxygen during the agonal period [18, 19]. Pericardial fluid bathes the myocardium and some studies have shown increased levels of cardiac Troponin T in pericardial fluid when compared to peripheral blood. Although this was a small study, the cardiac Troponin T levels in heart blood of right ventricle, left ventricle and pericardial fluid were statistically significant. Cardiac arrhythmias during the ischemic period may play a key role in sudden cardiac death, wherein, MI could not be demonstrated [20, 21].

This study showed that there was a strong relationship between postmortem cardiac Troponin T (cTnT) reactivity with death caused by myocardial infarction correlating significantly with histopathology findings. In this study almost all values were above the normal range for living patients.

Clinical uses of cardiac Troponin-T

- Diagnosis of AMI (Acute Myocardial Infarction)
- Reperfusion detection
- Infarct size estimation
- Stratification of risk
- Detecting peri operative myocardial infarction

Troponins have proven value as prognostic markers. In patients who have non-ST segment elevation acute coronary syndrome and who were studied in the Thrombolysis in Myocardial Infarction (TIMI) IIIB Trial, graded increase in Troponin I at baseline were significantly and independently correlated with increasing mortality.

Cardiac specific Troponins

Troponin is a protein complex consisting of the three subunits namely Troponin T (TnT), Troponin I (TnI) and Troponin C (TnC) located on the thin filament of striated muscles. Cardiac specific Troponin T (cTnT) and cardiac specific Troponin-I (cTnI) have amino acid sequences different from those of the skeletal muscle forms of these proteins. Since cTnT and cTnI are not normally detectable in the blood of healthy individuals but may increase after ST segment elevation MI (STEMI) to levels >20 times higher than the upper reference limit (the noise level of the assay), the measurement of cTnT or cTnI is of considerable diagnostic usefulness and they are now the preferred biochemical marker for MI Levels start rising within 3-6 hours and peaks by 24 hours and peaks by 24 hours. They remain elevated for 7 to 10 days after STEMI.

Cardiac Troponins are superior to CKMB, as follows.

- Troponins are markedly cardiac specific especially Cardiac Troponin I (100%).
- Its marked increase and extended duration of half life in the blood helps to detect delayed cases of AMI.
- Due to the cardiac specificity and their very low concentration in serum of normal individuals, cTns have greater sensitivity for minor degrees of myocardial injury.
- In unstable angina cases, they are effective prognostic indicators than CKMB.
- A single level of cTnT in peripheral blood after AMI can be used for estimation of infarct size whereas CKMB requires repeat samples.

Cardiac Troponin T versus cardiac Troponin I

- Recently, highly sensitive third generation cTnT assays (hscTnT) are highly cardiac specific with the overall

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ISSN: 2394-0034 (O)

diagnostic sensitivity, specificity and efficiency better than cTnI. Standardization of cTnI assays is difficult as it has many manufacturers with varied results, whereas cTnT assay I manufactured by only one manufacturer (Roche).

Other biochemical markers

Glycogen Phosphorylase (GPBB)

Of late, GPBB is one of the best markers for early detection of AMI however it is also elevated in UA when glycogen is broken.

History of biochemical marker usage in myocardial injury was as per **Table – 6.**

In the year 2000, the ESC/ACC guidelines recognized the major role of biochemical markers and made elevations in their levels the key to diagnose AMI.

At that point of time, they had also recognized and acknowledged that cTnT and cTnI had surpassed Creatinine Kinase-MB as the analytes of choice for diagnosis.

Historical review of methods for detection of early myocardial infarction in autopsy cases

Different methods have been discovered for identification of early myocardial infarction during autopsy. Every method has its own advantages and limitations. Many animal experiments were conducted for detection of early infarction. In the year 1960, histo chemical staining using Tetrazolium dyes was introduced as a method to identify early MI. Other methods include Hematoxylin-basic fuchsine-picric acid staining, Barbeito-Lopez Trichrome staining, measuring K^+/Na^+ ion ratio, Mg^+/Ca^+ ion ratio, fluorescent microscopy, measurement of sarcomere length and determination of density of blood.

Conclusion

Forensic pathologists seldom request Troponin assay/ test in the investigation of sudden cardiac death because of firm diagnosis of cardiac death as evident on gross autopsy and histological findings coupled with significant symptoms. However not all cardiac deaths show these finding e.g. Micro infarcts of the myocardium may produce cardiac arrhythmias and subsequent death. The search of a reliable marker to support diagnosis of cardiac injury was made by this study.

We also should be aware of the laboratory's normal range, the elevated levels seen in postmortem control samples, and the anatomic site from which the sample is drawn. Although, blood from central locations as the cardiac ventricles and pericardial fluid showed markedly increased levels, they best equate anti-mortem physiologic levels. Moreover, it is necessary to correlate the laboratory reports with the place of occurrence, history of the case, and HPE findings. One issue with this study and studies up to date is that cardiac Troponin T levels are tested in obvious patients with acute MI and compared with patients who died of non cardiac causes. Inevitably, all of these studies concluded that increased cardiac Troponin T levels correlate with the cause of death and supported the gross and HPE findings. Therefore more large scale studies are needed before definite conclusions can be drawn from these assays. It is hoped that this study can be a foundation for future larger studies.

The sensitive markers should be that highest abundance in the cell. The major function of the heart is contraction, the proteins involved in contraction in producing the energy are important biomarkers of cardiac injury which could be detected in blood and cardio specific proteins like Cardiac Troponin T levels are more sensitive and specific biomarkers.

ISSN: 2394-0026 (P)
ISSN: 2394-0034 (O)

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Table – 1: Microscopic features of MI.

Time	Light microscopic features
0 to 30 minutes	Nil changes
30 minutes to 4 hours	Usually no change; variable waviness of fibers at the border.
4 to 12 hours	Early coagulation necrosis; edema; hemorrhage,
12 to 24 hours	Continuing coagulative necrosis; pyknosis of nuclei; myocytes hyper eosinophilla; marginal contraction band necrosis; early neutrophil infiltration.
1 to 3 days	Coagulation necrosis with loss of nuclei and striations; brisk neutrophil infiltration.
3 to 7 days	Beginning of disintegration of dead myofibers with dying neutrophils; early phagocytosis of dead cells by macrophages at the border.
7 to 10 days	Well developed phagocytosis of dead cells; early formation of granulation tissue at margins
10 to 14 days	Well established granulation tissue with new blood vessels and collagen deposition
2 to 8 weeks	Increased collagen deposition with decreased cellularity
>2 months	Dense collagen scar

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ISSN: 2394-0034 (O)

Table - 2: Cardiac Troponin T concentration levels in study group (n=12).

Sr. No.	P.M. No.	Cardiac Troponin T Levels (ng/ml) (> 0.2 ng/ml +ve result)			Postmortem Interval	Inference
		Left ventricle	Right ventricle	Pericardial Fluid		
1.	886/13	>2.000	>2.000	>2.000	8 hours	Markedly increased
2.	887/13	>2.000	>2.000	>2.000	10 hours	Markedly increased
3.	987/13	>2.000	>2.000	>2.000	19 hours	Markedly increased
4.	1014/13	>2.000	>2.000	>2.000	16 hours	Markedly increased
5.	1129/13	>2.000	>2.000	>2.000	16 hours	Markedly increased
6.	1423/13	>2.000	>2.000	>2.000	12 hours	Markedly increased
7.	1426/13	>2.000	>2.000	>2.000	17 hours	Markedly increased
8.	1436/13	>2.000	>2.000	>2.000	5 hours	Markedly increased
9.	2170/13	>2.000	>2.000	>2.000	12 hours	Markedly increased
10.	2239/13	0.063	0.051	0.093	6 hours	Normal
11.	2303/13	>2.000	>2.000	>2.000	18 hours	Markedly increased
12.	2308/13	>2.000	>2.000	>2.000	24 hours	Markedly increased

ISSN: 2394-0026 (P)
ISSN: 2394-0034 (O)

Table - 3: Cardiac Troponin T concentration levels in control group (n=6).

Sr. No.	P.M. No.	Cardiac Troponin T levels (ng/ml) (>2.000 ng/ml +ve result)			Postmortem Interval	Inference
		Left Ventricle	Right Ventricle	Pericardial Fluid		
1.	890/13	>2.000	>2.000	>2.000	30 hours	Markedly increased
2.	894/13	>2.000	>2.000	>2.000	28 hours	Markedly increased
3.	982/13	>2.000	>2.000	>2.000	40 hours	Markedly increased
4.	1010/13	0.05	0.06	0.03	6 hours	Normal
5.	1121/13	0.05	0.08	0.06	8 hours	Normal
6.	1420/13	>2.000	>2.000	>2.000	36 hours	Markedly increased

Table - 4: Histopathological examination results in Study group (n=12).

Sr. No.	Observations	No. of cases
1	Evidence of early myocardial infarction	4
2	Evidence of early myocardial infarction and old infarction	3
3	Evidence of old infarction	2
4	Normal myocardial histology no areas of infarction	3

Table - 5: Histopathological examination results in control group (n=6).

Sr. No.	Observations	No. of cases
1	Evidence of early myocardial infarction	Nil
2	Evidence of old infarction	2
3	Normal myocardial histology. No areas of infarction	4

Table – 6: History of biochemical marker usage in myocardial injury.

Time period	Marker
Late 1950s	Aspartate amino Transferase (AST, SGOT)
1960s	Creatine Kinase isoenzyme (CK-MB activity)
1970s	Lactate dehydrogenase isoenzymes (ratio of LDH1 to LDH2)
Late 1980s	Creatine Kinase-MB mass concentration
Mid 1990s	Troponin I, Troponin T