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Original Research Article

Study of troponin-t elevation in critically ill patients-incidence and outcome

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ABSTRACT

Background: Besides cardiac disease, critical illness patients are often subjected to myocardial injury. Hence, the goal of present study was to analyze the incidence of elevated cardiac troponin levels in critically ill patients and its outcome which in turn can act as a prognostic marker.
Methods: The settings were the ICU's of a tertiary care hospital in south India. A sample of 54 was identified using purposive sampling technique. Serial monitoring of troponin T was done on day 1, 3 and 5. A performa was used to collect the baseline data of who met the inclusion and exclusion criteria. The collected data was analyzed by using descriptive and inferential statistics.
Results: Among 54 patients 17 (31.5%) of them expired. It was found that patients with mean APACHE score were significantly greater among mortality group (P<0.001). When analyzing the outcome in relation to the troponin T levels, it can be concluded that troponin levels were higher among patients who died but it was not statistically significant. (p=0.56, 0.84 and 0.67 on day 1, 3 and 5 respectively). Among the 54 patients it is inferred from the findings that most of the critically ill patients do have high troponin T levels.
Conclusions: The study illustrated the incidence of high troponin T levels in critically ill patients which is an informative prognostic indicator. The relation couldn't be proved due to less sample size and other imposing factors in critically ill such as broad diagnostic categories.

Keywords: Cardiac troponin, Outcome, Diagnostic utility, Prognosis

INTRODUCTION

Elevated levels of cardiac troponin not due to acute coronary syndromes have been reported in various populations of patients in the intensive care unit (ICU): heterogeneous medical and surgical patients and critically ill patients with systemic hypotension and with conditions such as trauma, sepsis, pulmonary embolism, stroke, renal failure, and chronic obstructive pulmonary disease.[1-8] Thus, troponin elevation among patients with sepsis and the systemic inflammatory response syndrome with or without shock in the intensive care unit setting is common and largely affects patients without significant coronary artery disease. Elevated troponin levels are clinically important because they may act as an adverse prognostic marker.[9] In the ICU, endotracheal intubation, coma due to underlying illness, and use of sedatives and narcotics all limit the ability of patients to report symptoms associated

with ischemia. Nevertheless, recognition of myocardial infarction in critically ill patients most likely is important because the development of myocardial infarction may contribute to increased morbidity and mortality.[10] Outside the ICU setting, patients with a diagnosis of myocardial infarction benefit from thrombolytic therapy, coronary revascularization and use of anticoagulants, antiplatelet agents, β-blockers, statins, and angiotensin-converting enzyme inhibitors. However, myocardial infarction due to non-thrombotic mechanisms may not respond favourably to antithrombotic agents, and the impact of these therapies on outcomes in ICU patients with myocardial infarction is unknown. Furthermore, the risk-benefit ratio of these agents in the ICU may differ considerably from when they are used in patients with myocardial infarction outside the ICU.[11]

A fundamental understanding of the prognostic significance of elevated levels of cardiac troponin and their relationship to myocardial infarction in critically ill patients is therefore an important first step toward devising and testing appropriate management strategies. This study has been designed to document the incidence of elevated serum levels of cardiac troponin and the outcome of critically ill patients having elevated troponin T levels.

Objective of this study was to analyse the incidence and outcome of troponin T positivity in patients admitted in critical care unit and to assess the diagnostic utility and limitations of evaluating troponin T in critically ill patients.

METHODS

This was an explorative, non-randomized, descriptive clinical study undertaken at a tertiary care hospital in south India over a period of 2 years from October 2012 to October 2014.

Inclusion criteria

- Patients admitted into the medical intensive care unit expected to be in the critical care unit for more than 48 hours
- All patients with a provisional diagnosis of acute coronary syndrome, unstable angina
- All patients with a provisional diagnosis of chronic coronary artery disease.

Exclusion criteria

- Patients with a history of trauma in the last 1 month
- Patients with a history of surgery in the last one month
- Patients with pregnancy and in palliative care
- Patients who were treated for MI in the last 1 month
- Patients not willing to be included in the study.

A detailed proforma was filled up for each patient, which included age, sex, IP number, detailed present history and co-morbid illness. A detailed clinical examination was done. All consecutive patients admitted to ICU from October 2012 to June 2014 underwent systematic screening of cardiac Troponin T and 12-lead ECG. Serial troponin T estimation was done on day 1, day 3 and day 5. ECGs with ischemic changes were considered abnormal. Patient data that was already available in the medical chart were collected, but no additional screening troponin measurements or ECG was performed if consent was not obtained.

Blood samples were obtained and centrifuged immediately at 4°C and stored at -80°C. Cardiac troponin T was measured with the fourth-generation cardiac troponin T electrochemiluminescence immunoassay (Elecsys, Roche Diagnostics, Indianapolis, Ind). An admission (within 6 hours) cardiac troponin T of 0.01 ng/mL which exceeds the 99th percentile of a reference population in accord with the European Society of Cardiology/American College of Cardiology/American Heart Association guidelines was considered elevated.[1-3]

On ICU admission, patient demographics and baseline data were collected. New clinical events and complications (development of arrhythmias, pulmonary edema, nonfatal cardiac arrest, and cardiogenic shock), need for advanced life support (mechanical ventilation, inotropes and hemodialysis), and ischemic cardiac symptoms were collected. Information on whether a diagnosis of MI during the patient's ICU stay was noted. All patients were followed up all throughout their ICU stay and duration of stay in ICU and hospital were noted down. Data were recorded on paper case report forms and entered into Microsoft Excel program for analysis. Recruitment of subjects and collection of data was spread over a period of two years. The study included 54 critically ill patients admitted to ICU fulfilling the inclusion and exclusion criteria.

Statistical methods

- Data were recorded on paper case report forms and entered into Microsoft Excel program for analysis
- Descriptive statistics were reported using mean and SD for the normally distributed continuous variables, else median and inter-quartile ranges
- Categorical variables were reported using number and percentages
- Chi-square test or Fisher’s exact test was used to test the association between the categorical variables
- Chi-square test is any statistical hypothesis test in which the sampling distribution of the test statistic is a chi-squared distribution when the null hypothesis is true
- Independent T test or Mann Whitney U test was used to compare the mean /medians between alive and dead groups, as appropriat

- The independent samples t-test is used when two separate sets of independent and identically distributed samples are obtained, one from each of the two populations being compared P value less than 5% was considered statistically significant.

RESULTS

In the study group, 56% (30) were males and 44% (24) were females (Table 1).

Table 1: Sex distribution of study subjects (n=54).

Gender	Frequency	Percent
Male	30	55.6
Female	24	44.4

Table 2: Troponin T levels in relation to sex distribution.

Gender	High No. (%)	Normal No. (%)	P value
Male	23 (52.3)	7 (70)	0.30
Female	21 (47.7)	3 (30)	

It was found in the study that there was no association between elevated troponin T levels and sex distribution (Table 2).

Table 3: Distribution of comorbidities in the study population (n=54).

Co-morbidities	Frequency	Percent
Bronchial asthma	4	7.4
Diabetes	26	48.1
Hypertension	26	48.1
IHD	8	14.8
COPD	12	22.2
CVA	7	13.0

In this study, the co-morbidities noted were Diabetes, Hypertension in 26 patients (48.1%) followed by COPD in 12 patients (22.2%), IHD in 8 patients (14.8%), CVA in 7 patients (13%) and Bronchial Asthma in 4 patients (7.4%) (Table 3).

In this study, no specific comorbidity was found to cause significant high Troponin T levels (Table 4).

Among the study individuals 55.6% (30) had normal ECG and 44.4% (24) had abnormal ECGs (Table 5).

In the study population, the mean APACHE score was 22.24, maximum of 34 and minimum of 14 with a standard deviation of 5.51 (Table 6).

Here it was found that patients with mean APACHE score were significantly greater among mortality group (P<0.001) (Table 7).

Table 4: Correlation of troponin T levels in relation to comorbidities.

	High No. (%)	Normal No. (%)	Trop-T day 1 P value
Diabetes			
Yes	21 (47.7)	5 (50.0)	0.89
No	23 (52.3)	5 (50.0)	
Hypertension			
No	22 (50)	6 (60)	0.78
Yes	22 (50)	4 (40)	
IHD			
No	36 (81.8)	10 (100)	0.14
Yes	8 (18.2)	0	
COPD			
No	34 (77.3)	8 (80)	0.85
Yes	10 (22.7)	2 (20)	
CVA			
No	37 (84.1)	10 (100)	0.17
Yes	7 (15.9)	0	

Reported as number and percentages; p-value using chi-square test.

Table 5: Frequency of ECG findings in the study (n=54).

ECG	Frequency	Percent
Normal	30	55.6
Abnormal	24	44.4

In the study group patients who had mortality had more days of hospital stay, ICU stay and ventilator requirement as compared to those who improved (p<0.001) (Table 8).

Table 6: APACHE score among study population (n=54).

	Min.	Max.	Mean	Std. deviation
APACHE score	14.00	34.00	22.24	5.51

Table 7: APACHE score in relation to outcome (n=54).

	Alive (N=37)	Dead (N=17)	P-value
APACHE score	20.0±4.2 19	27.1 ±5.1 29	<0.001

Reported as Mean ± Standard Deviation, Median.

Of the study group 31.5% had mortality irrespective of troponin T levels (Table 9).

Among the study individuals the incidence of high troponin T levels was 81.5% (44) (Table 10).

Although, it was observed that troponin levels were higher among patients who died compared to alive group, it was statistically not significant (Table 11).

Table 8: Outcome of patients with respect to hospital, ICU stay and ventilator status in study individuals (n=54).

	Alive (N=37)	Dead (N=17)	P-value
Number of hospital stay	9.5±7.3 8.0	11.6±5.4 10	0.04
ICU stay	5.5±4.9 4	9.5±5.6 8	<0.001
Days in ventilator	2.2 ±4.7 0	7.5±5.4 7	<0.001

Reported as Mean ± Standard Deviation, Median

Table 9: Distribution of outcome in study group (n=54).

Outcome	Frequency	Percent
Alive	37	68.5%
Dead	17	31.5%

Table 10: Incidence of elevated troponin T levels in study population (n=54).

Troponin T (Day 1)	Frequency	Percent
High	44	81.5%
Normal	10	18.5%

There was no significant association between troponin levels and mortality status in day 1 (p=0.91) (Table 12).

Table 11: Outcome of patients' relation to troponin T levels on day 1, 3, 5.

	Alive (N=37)	Dead (N=17)	P-value
Trop-T day 1	0.76±2.4 0.04 (0.02, 0.16)	0.35±0.61 0.07 (0.02, 0.40)	0.56
Trop-T day 3	0.50±1.5 0.05 (0.02, 0.13)	0.33±0.63 0.10 (0.01, 0.26)	0.84
Trop-T day 5	0.29±0.76 0.04 (0.02, 0.09)	0.44 ±0.91 0.09 (0.001, 0.44)	0.67

Reported as mean ± SD, median (25^{th}, 75^{th} percentile).

Table 12: Outcome of patients in relation to high and normal troponin T levels.

	Alive No (%)	Dead No (%)	P value
Trop-T day 1			
High	30 (81.1)	14 (82.4)	0.91
Normal	7 (18.9)	3 (17.6)	
Trop-T day 3			
High	31 (83.8)	13 (76.5)	0.52
Normal	6 (16.2)	4 (23.5)	
Trop-T day 5			
High	29 (78.4)	11 (64.7)	0.28
Normal	8 (21.6)	6 (35.3)	

In the study population the mean troponin T levels on Day 1, 3, 5 were 0.6, 0.4, and 0.3 respectively (Table 13).

Table 13: Mean values of troponin T levels among the study population.

	Min.	Max.	Mean	SD	95%CI	
TroponinT day 1 value	0.01	10.00	0.6289	2.01	0.080	1.177
Troponin T day 3 value	0.00	6.72	0.4460	1.254	0.104	0.788
Troponin T day 5 value	0.00	3.54	0.3370	0.807	0.117	0.557

Among this study population, incidence of high troponin T levels were found in patients with IHD, Carcinoma, CKD, CVA, Dyselectrolytemia, Hypertension, Infectious disease (Table 14).

DISCUSSION

Troponin T (cTnT) elevation is common in patients in the Intensive Care Unit (ICU) and is associated with morbidity and mortality. Irrespective of the cause, detectable troponin among stable patients with end-stage renal disease appears to be a powerful predictor of increased intermediate- term mortality.[12] Of note, multi-vessel coronary artery disease was more prevalent across progressively higher quartiles of troponin T, suggesting an ischemic mechanism for the troponin increase in this patient population.[13] In heart failure patients, elevated troponin is associated with the risk of death and re-hospitalization regardless of underlying cause and the three-year survival is about 29%.[14,15] Critically ill patients who require stay in an intensive care unit are a heterogeneous group of patients who are ill for a variety of heterogeneous diseases. Thus, estimation of cardiac troponins and their significance in these patients are heterogeneous.[16]

The aim of the study was to determine the epidemiology of raised cTnT levels and to predict the prognosis of the critically ill patients to suggest it as a prognostic marker. Cardiac troponin elevation is common and is observed in 40 to 50% of critically ill medical and surgical patients. Elevated levels appear to identify patients at increased risk for death in the intensive care unit or hospital setting. This finding, and its relation to myocardial infarction and

acute coronary syndromes, requires prospective study to better understand the implications for diagnosis and management.

Table 14: Mean troponin T values among the diagnosis in critically ill.

	Mean	No.
Anaemia	0.1725	2
ARDS	0.0765	2
Carcinoma	0.3610	1
Chronic liver disease	0.4910	2
CKD	0.0380	1
CVA	1.2970	2
Dyselectrolytemia	0.1597	3
Hypertension	0.2400	1
IHD	2.3988	9
Infectious disease	0.9150	6
Neuro infection	0.0293	3
Respiratory infection	0.1023	11
Sepsis	0.0303	8
SIRS	0.1380	1
Valvular heart disease	0.0470	2
Total	0.6289	54

The study was carried out in a tertiary care hospital over a period of 2 years. The study was designated as descriptive clinical study which included 54 patients. In this study, we analyzed the incidence and outcome of troponin T positivity in patients admitted in critical care unit with various medical disorders including acute coronary syndrome and other medical conditions.

In the study as we consider the troponin T levels in relation to the sex distribution, it was shown that among 44 high troponin T level patients 23 (52.3%) were males and only 21 (47.7%) were females. When we consider the 10 normal troponin T level patients 7 of them were males (70%) and only 3 of them were females (30%). It was found in the study that there was no association between elevated troponin T levels and sex distribution (p=0.30).

As we look into the co-morbidities in the 54 study samples, 26 patients (48.1%) had Diabetes and Hypertension and the least comorbidity found was bronchial asthma i.e., only for 4 patients (7.4%). In this study, no specific comorbidity was found to cause significant high troponin T levels.

Among the 54 study individuals, 55.6% (30) had normal ECG and 44.4% (24) had abnormal ECGs. In the study population, the mean APACHE score was 22.24, with a maximum of 34 and minimum of 14 with a standard deviation of 5.51. Here it was found that patients with mean APACHE score were significantly greater among mortality group (P<0.001). The mean troponin T values was 0.629 (SD=2.01), 0.447 (SD=1.254) and 0.337 (SD=0.807) on day 1, 3 and 5 respectively.

When analyzing the outcome of patients in relation to the troponin T levels computed on Day 1, 3, 5 it was clearly evident that among 37 living patients the value (median) of troponin T was 0.04, 0.05 and 0.04 on day 1, 3, 5 respectively. But when we compare this data with the dead, the troponin T levels were 0.07, 0.1, and 0.09 on day 1, 3 and 5 respectively. It can be concluded that troponin levels were higher among patients who died compared to live group.[17]

Even though it was found that troponin T levels were significantly high among the mortality group, it was not statistically significant: p=0.56, 0.84 and 0.67 on day 1, 3 and 5 respectively. It's proved here that in the outcome of patients in relation to high and normal troponin T levels, among patients with high troponin T levels, 82.4% it accounted to mortality. In patients with normal troponin T level patients, the live patients were more i.e., 18.9% as compared to the dead 17.6%.

Among the 54 critically ill patients, most of them were diagnosed with IHD and respiratory infection, and these patients had high troponin T levels. Interestingly in the 54 study samples the least critically ill patients were from the category of CKD, carcinoma and hypertension respectively (1 each), for these categories also most of them were having high troponin T levels. So it is inferred from the findings that most of the critically ill patients do have high troponin T levels.

In the study 'A prospective study of the impact of serial troponin measurements on the diagnosis of myocardial infarction and hospital and six-month mortality in patients admitted to ICU with non-cardiac diagnosis' data from 144 patients were analyzed (42% female; mean age 61.9 (SD 16.9)). A total of 121 patients (84%) had at least one cTnT level ≥15 ng/L which correlates with the present study where 81.5%had high cTnT levels. A total of 20 patients (14%) had a definite MI, 27% had a possible MI, 43% had a cTnT rise without contemporaneous ECG changes, and 16% had no cTnT rise. ICU, hospital and 180-day mortality was significantly higher in patients with a definite or possible MI. Only 20% of definite MIs were recognized by the clinical team. There was no significant difference in mortality between recognized and non-recognized events.[17]

A study done to assess the correlation among clinically recognized cardiac dysfunction and high troponin level found that cardiac patients with known coronary artery disease, who have been admitted for a medical reason and became critically ill, will have significantly higher in-hospital mortality if they have a high cardiac troponin level.[18]

At the time of cTnT rise, 100 patients (70%) were septic and 58% were on vasopressors. Patients who were septic when cTnT was elevated had an ICU mortality of 28% compared to 9% in patients without sepsis. ICU mortality

of patients who were on vasopressors at the time of cTnT elevation was 37% compared to 1.7% in patients not on vasopressors. This study confirms that raised troponin levels are very common among critically ill patients admitted to ICU for non-cardiac reasons and are associated with an increased mortality up to 180 days. The large majority of ICU patients (84%) had one or more elevated troponin values and 41% met the criteria for MI.[19]

A similar study "High Level of troponin I Decreases the One-year Survival Rate of non-cardiac Critically Ill Patients" was aimed to evaluate the effect of high level cardiac troponin I (cTnI) on one-year survival rate of non-cardiac critically ill patients. A total of 153 non-cardiac critically ill patients were enrolled in the research. Serum cTnI and CK-MB (creatine kinase-MB) levels were measured simultaneously on admission. Clinical and laboratory parameters were recorded and compared between these two groups. In addition, the one-year survival rate was evaluated among patients with high cTnI level and normal cTnI level. CK-MB levels, LVEF (left ventricular ejection fraction) and the length of hospitalization didn't show any significant differences between the two groups ($P>0.05$). APACHE II scores, usage of mechanic ventilation and MODS (multiple organ dysfunction syndrome) incidence of high cTnI group were significantly increased compared with normal cTnI group ($P<0.05$). The one-year survival rate of high cTnI group was lower than normal cTnI group (log-rank test, $\pm 2=4.29$, $P<0.05$). Non-cardiac critically ill patients with high cTnI levels presented low one-year survival rate. cTnI level can be used as a maker to predict short time survival in non-cardiac critically ill patients.[19]

In other prospective studies, 15% to 70% of patients in a general ICU had at least a single measurement indicating an elevation in the serum level of cardiac troponin. The prevalence of elevation in cardiac troponin T in specific ICU populations has been studied in patients who had sepsis, cardiac surgery, or non-cardiac diagnosis. Prevalence estimates for patients with non-cardiac diagnosis ranged from 16% to 55%. In a study of 34 consecutive patients who were treated with mechanical ventilation or who underwent thoracic or vascular surgery, 11 (32%) had elevated levels of cardiac troponin T, and 4 (36%) of the 11 had ECG changes compatible with myocardial infarction.[19]

In the study 'Elevated troponin T concentrations in critically ill patients' it was shown that elevations in cardiac troponin T levels have also been associated with increased ICU and hospital stays in surgical ICU patients. Although supported by fewer studies, elevated levels of cardiac troponin T are similarly a predictor of mortality in ICU patients with early sepsis and after cardiac surgery and are associated with prolonged ICU stay after cardiac surgery. Results of randomized trials indicate that antithrombotic therapy improves outcomes in patients with myocardial infarction.[19]

Limitations of this study include its size and subjective assessment. ECGs were performed and interpreted at the discretion of one clinician. The diagnostic categories were broad, but enhanced specificity may be beneficial in order to identify patients at greatest risk of adverse event.

None of our patients underwent stress echocardiography or autopsy to detect if they had flow limiting coronary artery disease.

Larger numbers of patients would also facilitate risk stratification, and would raise more hypotheses about specific medication effects. A larger number of patients would also likely confirm a gradient effect on mortality with increasing troponin T levels.

CONCLUSION

In summary, elevated levels of troponin T is common in critically ill patients. The role of elevations in cardiac troponin T as a prognostic marker in the ICU is still uncertain. It is challenging to establish the diagnosis of myocardial infarction in critically ill patients because of limitations in determining whether the patients have typical symptoms of ischemia and because of uncertainty about the interpretation of abnormal levels of cardiac troponin T. By this study we would suggest that critically ill patients with high troponin levels should closely monitored for grave complications. Additional research is required on the optimal evaluation and management of critically ill patients who have elevated levels of cardiac troponin T.

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