Section: General Medicine

Original Research Article

A STUDY OF TROPONIN I LEVELS FOR ASSESSING SEPSIS INDUCED MYOCARDIAL DYSFUNCTION IN PATIENTS WITH SEPTIC SHOCK

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Abstract

Background: Troponin I levels are elevated in patients with sepsis induced myocardial dysfunction in patients with septic shock and recent data suggestive of association between troponin I elevation and SIMD. Therefore measurement of troponin I levels help in outcome measurement in septic shock patients. **Materials and Methods:** The study comprised 270 patients over the age of 18 who were admitted to the KIMS hospital in hubballi with a diagnosis of Sepsis and Septic shock based on the consensus conference committee of the American college of chest physicians and critical care medicine criteria. Acute coronary syndrome, clinical evidence of congestive heart failure, cardiac pulmonary resuscitation/defibrillation prior to admission, and a history of previous cardiac disease, CKD, stroke, Subarachnoid hemorrhage were all excluded from the study. On the day of admission, ECG, ECHO, Cardiac enzymes, Cardiac Troponin I, and other needed investigations were performed. The levels of cTnI were measured in patients with sepsis and septic shock. **Result:** The study comprised 270 patients with septic shock. Troponin I was positive in 29 individuals (72.5%) and normal in 11 patients (27.5%). (The significance level is 0.001438). In the troponin I positive group, ECHO was positive in . (0.342558). was a weak positive correlation between MAP (mmHg) and Troponin I Levels, and this correlation was not statistically significant (rho = 0.09, p = 0.134). 44.4% of the participants had ECG: Sinus Tachycardia. 18.5% of the participants had ECG: Sinus Tachycardia + ST Depression. 18.5% of the participants had ECG: ST Depression. 14.8% of the participants had ECG: Sinus Rhythm. 3.7% of the participants had ECG: Sinus Bradycardia. There was a significant difference between the 5 groups in terms of Troponin I Levels ($\chi 2$ = 33.786, p = <0.001), with the median Troponin I Levels being highest in the ECG: Sinus Tachycardia + ST Depression group. **Conclusion:** The strong association of cTnI in patients with sepsis and septic shock without ACS, according to this study, shows inflammatory damage to the myocardium in addition to ischemia damage. It indicates a high rate of morbidity and mortality.

INTRODUCTION

Sepsis is defined as dysregulated host response to infection that leads to organ dysfunction. It involves both pro-inflammatory and anti-inflammatory mediators.[1] Troponin I is a cardiac biomarker which is elevated in Acute Myocardial Infarction.[2] It is an inhibitory sub unit of troponin. It gives risk stratification and bad prognosis. It was also seen to be elevated in critically ill patients like sepsis, septic shock,[3] and in pulmonary embolism, exacerbation of copd, poisoning, snake bite, rhabdomyolysis, chronic kidney disease, stroke, Subarachnoid hemorrhage, infiltrative disorders, chemotherapeutic agents.[4]

We investigated the role of troponin I in patients with sepsis and septic shock in the current investigation. In the setting of systemic inflammatory response syndrome (SIRS), sepsis, or septic shock, it is seen in 31% to 80% of patients. Increased troponin I levels suggest increased morbidity and death.[5]

It also implies that it isn't only Myocardial infarction which is associated with irreversible ischemia

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damage.[6] However, it can also be raised in inflammatory situations where myocardial injury that is reversible.

However, several explanations have been offered to explain the rise in troponin I levels.[7-11] The cardiovascular system is one of the most commonly impacted systems by sepsis and septic shock. Waisbren was the first to describe sepsis-related cardiovascular impairment in1951.

With bounding pulses, flushing, and a hyperdynamic state, recognised it. Fever, oliguria, and hypotension are all symptoms of this condition. also described a smaller group of patients who were clammy, pallor, and hypotensive, with low volume pulseswho appeared to be more seriously ill.[12-14]

In retrospect, the latter group may have made a better decision. Volume has been resuscitated, and timely and enough volume therapy has been administered. One of the most effective supportive interventions in sepsis has been demonstrated.[15-18]

The occurrence of cardiovascular dysfunction in sepsis is linked to a higher risk of death,[19] a much higher mortality rate of 70% to 90% compared to 20% in septic shockpatients who do not have a cardiovascular impairment As a result, sepsis causing myocardial dysfunction has been the subject of extensive inquiry.[20] In this investigation, we used a prospective design.

Troponin I levels were studied, and a link between cTnI and sepsis and septic shock diagnosed according to the criteria.

MATERIALS AND METHODS

270 patients with sepsis and septic shock were admitted to Karnataka institute of medical sciences, Hubballi. The research was carried out over an 18-month period.270 patients were admitted to KIMS Hospital ICU with a diagnosis of sepsis and septic shock. Patients with a history of heart disease or ECG changes were excluded. The patient's relatives provided informed consent. In all patients, a detailed history was taken regarding the presenting complaints, predisposing factors, and accompanying illness. A comprehensive clinical examination was performed.

On the day of admission, all patients had CBC, Platelet count, Serum electrolytes, RBS, ABG, RFT, LFT, PT, APTT, INR, Urine routine, ECG, ECHO, Cardiac Enzymes, and Troponin I tests performed. Troponin I was measured using the ELISA method. All patients received a chest X-ray, USG-Abdomen and Pelvis, blood culture, urine culture, swab culture, and sputum culture.

Criteria for Inclusion

Age >18yrs Sepsis and Septic shock diagnosed using the criteria of the American College of Chest Physicians and Critical Care Medicine's consensus conference commitee.

Criteria for Exclusion

Prior to admission, patients with Acute Coronary Syndrome, clinical evidence of congestive cardiac failure, and cardiac pulmonary resuscitation/defibrillation.

Patients with a history of previous cardiac disease and ECG changes.

Statistical Analysis

Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. Chi- square test was used as test of significance for qualitative data. Continuous data was represented as mean and SD. Independent t test was used as test of significance. ANOVA (Analysis of Variance) was the test of significance to identify the mean difference between more than two groups for quantitative and qualitative data respectively. Paired t test is the test of significance for paired data such as before and after surgery for quantitative and qualitative data respectively. Pearson correlation or Spearman's correlation was done to find the correlation between two quantitative variables and qualitative variables respectively. Categorical data is presented as percentages, while continuous data is presented as mean and median. To compare categorical and continuous variables, the Pearson chi-test was used.

RESULTS

The mean Age (Years) was 53.83 ± 14.05.

24 (8.9%) of the participants had Age: 18-30 Years.

23 (8.5%) of the participants had Age: 31-40 Years.

50 (18.5%) of the participants had Age: 41-50 Years.

69 (25.6%) of the participants had Age: 51-60 Years.

77 (28.5%) of the participants had Age: 61-70 Years.

25 (9.3%) of the participants had Age: 71-80 Years.

2 (0.7%) of the participants had Age: 81-90 Years.

181 (67.0%) of the participants had Gender: Male. 89 (33.0%) of the participants had Gender: Female.

Table 1: Summary of Age/Gender

Age/Gender	Mean ± SD || Median (IQR) || Min-Max || Frequency (%)
Age (Years)	53.83 ± 14.05 || 55.00 (46.00-64.75) || 18.00 - 86.00
Age	
18-30 Years	24 (8.9%)
31-40 Years	23 (8.5%)
41-50 Years	50 (18.5%)
51-60 Years	69 (25.6%)
61-70 Years	77 (28.5%)
71-80 Years	25 (9.3%)
81-90 Years	2 (0.7%)
Gender	

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Male	181 (67.0%)
Female	89 (33.0%)

Table 2: Summary of Investigations

Investigations	Mean ± SD	Median (IQR)	Min - Max
TLC (x10³/mm³)	19.47 ± 9.03	18.80 (14.20-25.50)	1.1 - 40.6
Platelet Count (Lacs/cu.mm)	1.15 ± 0.45	1.20 (0.70-1.60)	0.1 - 1.9
Total Bilirubin (mg/dL)	3.23 ± 2.37	1.90 (1.40-5.30)	0.6 - 8.5
Direct Bilirubin (mg/dL)	0.90 ± 0.82	0.60 (0.30-1.10)	0.1 - 3.3
Blood Urea (mg/dL)	38.60 ± 37.90	16.00 (11.00-45.00)	10.0 - 120.0
S. Creatinine (mg/dL)	1.74 ± 1.28	1.10 (0.60-3.10)	0.3 - 4.6
Serum Lactate	3.07 ± 0.65	3.10 (2.60-3.50)	0.0 - 4.6
Troponin I Levels	0.11 ± 0.15	0.01 (0.01-0.16)	0.0 - 0.5

The mean TLC (x10³/mm³) was 19.47 ± 9.03.
The mean Platelet Count (Lacs/cu.mm) was 1.15 ± 0.45.
The mean Total Bilirubin (mg/dL) was 3.23 ± 2.37.
The mean Direct Bilirubin (mg/dL) was 0.90 ± 0.82.
The mean Blood Urea (mg/dL) was 38.60 ± 37.90.
The mean S. Creatinine (mg/dL) was 1.74 ± 1.28.
The mean Serum Lactate was 3.07 ± 0.65.
The mean Troponin I Levels was 0.11 ± 0.15.

Table 3: Distribution of the Participants in Terms of SOFA Score (n = 270)

SOFA Score	
Mean (SD)	9.35 (3.18)
Median (IQR)	10 (7-12)
Range	1 - 17

The variable SOFA Score was not normally distributed (Shapiro-Wilk Test: p = <0.001).
The mean (SD) of SOFA Score was 9.35 (3.18). The median (IQR) of SOFA Score was 10.00 (7-12). The SOFA Score ranged from 1 - 17.

Table 4: Summary of Imaging

Imaging	Mean ± SD || Median (IQR) || Min-Max || Frequency (%)
USG Abdomen	
Normal	189 (70.0%)
Abnormal	81 (30.0%)
Chest Xray	
Normal	95 (35.2%)
Abnormal	175 (64.8%)
ECG Abnormality (Present)	110 (40.7%)
ECG	
Sinus Tachycardia	120 (44.4%)
Sinus Tachycardia + ST Depression	50 (18.5%)
ST Depression	50 (18.5%)
Sinus Rhythm	40 (14.8%)
Sinus Bradycardia	10 (3.7%)
2D-ECHO Abnormality (Present)	110 (40.7%)
2D-ECHO: LV Dysfunction (Yes)	80 (29.6%)
2D-ECHO: RV Dysfunction (Yes)	46 (17.0%)
2D-ECHO: RWMA (Yes)	46 (17.0%)

189 (70.0%) of the participants had USG Abdomen: Normal. 81 (30.0%) of the participants had USG Abdomen: Abnormal.
95 (35.2%) of the participants had Chest Xray: Normal. 175 (64.8%) of the participants had Chest Xray: Abnormal.
110 (40.7%) of the participants had ECG Abnormality: Present. 160 (59.3%) of the participants had ECG Abnormality: Absent.
120 (44.4%) of the participants had ECG: Sinus Tachycardia. 50 (18.5%) of the participants had ECG: Sinus Tachycardia + ST Depression. 50 (18.5%) of the participants had ECG: ST Depression. 40 (14.8%) of the participants had ECG: Sinus Rhythm. 10 (3.7%) of the participants had ECG: Sinus Bradycardia.
110 (40.7%) of the participants had 2D-ECHO Abnormality: Present. 160 (59.3%) of the participants had 2D-ECHO Abnormality: Absent.
80 (29.6%) of the participants had 2D-ECHO: LV Dysfunction: Yes. 190 (70.4%) of the participants had 2D-ECHO: LV Dysfunction: No.

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46 (17.0%) of the participants had 2D-ECHO: RV Dysfunction: Yes. 224 (83.0%) of the participants had 2D-ECHO: RV Dysfunction: No.
46 (17.0%) of the participants had 2D-ECHO: RWMA: Yes. 224 (83.0%) of the participants had 2D-ECHO: RWMA: No.

Table 5: Summary of Diagnosis and Outcome

Diagnosis and Outcome	Mean ± SD || Median (IQR) || Min-Max || Frequency (%)
Diagnosis	
BPN	97 (35.9%)
Lobar Pneumonia	50 (18.5%)
UTI	50 (18.5%)
SBP	20 (7.4%)
GE	18 (6.7%)
ARDS	17 (6.3%)
TBM	8 (3.0%)
ARDS+AKI	3 (1.1%)
BPN+AKI	3 (1.1%)
Disseminated Tb	2 (0.7%)
AGE+AKI	1 (0.4%)
GE+AKI	1 (0.4%)
Outcome	
Improved	160 (59.3%)
Death	110 (40.7%)

97 (35.9%) of the participants had Diagnosis: BPN. 50 (18.5%) of the participants had Diagnosis: Lobar Pneumonia. 50 (18.5%) of the participants had Diagnosis: UTI. 20 (7.4%) of the participants had Diagnosis: SBP. 18 (6.7%) of the participants had Diagnosis: GE. 17 (6.3%) of the participants had Diagnosis: ARDS. 8 (3.0%) of the participants had Diagnosis: TBM. 3 (1.1%) of the participants had Diagnosis: ARDS+AKI. 3 (1.1%) of the participants had Diagnosis: BPN+AKI. 2 (0.7%) of the participants had Diagnosis: Disseminated Tb. 1 (0.4%) of the participants had Diagnosis: AGE+AKI. 1 (0.4%) of the participants had Diagnosis: GE+AKI.
160 (59.3%) of the participants had Outcome: Improved. 110 (40.7%) of the participants had Outcome: Death.

Table 6: Distribution of the Participants in Terms of Diagnosis (n = 270)

Diagnosis	Frequency	Percentage	95% CI
BPN	97	35.9%	30.3% - 42.0%
Lobar Pneumonia	50	18.5%	14.2% - 23.8%
UTI	50	18.5%	14.2% - 23.8%
SBP	20	7.4%	4.7% - 11.4%
GE	18	6.7%	4.1% - 10.5%
ARDS	17	6.3%	3.8% - 10.1%
TBM	8	3.0%	1.4% - 6.0%
ARDS+AKI	3	1.1%	0.3% - 3.5%
BPN+AKI	3	1.1%	0.3% - 3.5%
Disseminated Tb	2	0.7%	0.1% - 2.9%
AGE+AKI	1	0.4%	0.0% - 2.4%
GE+AKI	1	0.4%	0.0% - 2.4%

35.9% of the participants had Diagnosis: BPN. 18.5% of the participants had Diagnosis: Lobar Pneumonia. 18.5% of the participants had Diagnosis: UTI. 7.4% of the participants had Diagnosis: SBP. 6.7% of the participants had Diagnosis: GE. 6.3% of the participants had Diagnosis: ARDS. 3.0% of the participants had Diagnosis: TBM. 1.1% of the participants had Diagnosis: ARDS+AKI. 1.1% of the participants had Diagnosis: BPN+AKI. 0.7% of the participants had Diagnosis: Disseminated Tb. 0.4% of the participants had Diagnosis: AGE+AKI. 0.4% of the participants had Diagnosis: GE+AKI.

Table 7: Distribution of the Participants in Terms of Outcome (n = 270)

Outcome	Frequency	Percentage	95% CI
Improved	160	59.3%	53.1% - 65.1%
Death	110	40.7%	34.9% - 46.9%

59.3% of the participants had Outcome: Improved. 40.7% of the participants had Outcome: Death.

DISCUSSION

The present study comprised of 270 patients presenting with history, characteristic clinical features and laboratory features of septic shock. These patients were admitted in intensive care unit. Eligible patients, based on inclusion and exclusion criteria, were evaluated taking into consideration history, clinical examination and investigations. The

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data collected were analysed and compared with various other similar studies.

The purpose of the study was to to estimate the troponin I levels and correlate with association between troponin I levels, echocardiographic findings and sepsis induced myocardial dysfunction, and also to predict the outcome of SIMD based on troponin I levels and echocardiography,

The mean age group in my study is 53.83 years which is nearly correlated with lamia et al. Present study shows more number of males than females which correlates with the Lamia et al and kim et al.[21,22]

For evaluation of sepsis, SOFA scoring were used, which in the present study. In the study, SOFA acore was comparable with other studies – kim et al and lamia et al.[21,22]

The median serum levels of TROPONIN I in the present study was 11 ng/L whereas the median of TROPONIN I in studies by Kim et al was 15.36ng/L, lamia et al was 36ng/L respectively.[21,22] The values observed in the present study is comparable to the studies by Kim et al reflects the similar intensity of sepsis in our patients.[21] Whereas in lamia et al,[22] study the median TROPONIN I was comparatively higher than the other studies thereby indicating an increased severity of SIMD.

Echo findings in present study is seen in 110(40.7%) of which and in Kim et al,[21] study out of 660 TROPONIN I positive 258 cases (65%) showed SIMD (LV systolic dysfunction (n = 163, 63.2%), LV diastolic dysfunction (n = 104, 40.3%), RV dysfunction (n = 97, 37.6%), and WMA (n = 186, 72.1%).

The mean (SD) of Troponin I Levels in the 2D-ECHO Abnormality: Present group was 0.15 (0.15). The mean (SD) of Troponin I Levels in the 2D-ECHO Abnormality: Absent group was 0.08 (0.14) the hs-TnI level was statistically higher in the patients with LV systolic dysfunction (1.609 vs. 0.540 ng/mL; p< 0.001), RV dysfunction (2.478 vs. 0.546 ng/mL; p < 0.001), and WMA (1.950 vs. 0.455 ng/mL; p < 0.001).

In David R. Altmann et.al study Pneumonia was seen in 14 (37%) patients, Acute GE in 3 (8%) pts, Urosepsis in 1(3%) pts whereas in present study pneumonia was seen in 147(54.4%) patients, Acute GE in 18(6.7%) patients, Urosepsis in 50 (18.5%) pts.[23,24]

In this study, we have studied association of troponin I levels in sepsis and septic shock. Troponin I, a cardiac biomarker which is elevated in Acute Myocardial Infarction is also associated to be elevated in critically ill patients like sepsis and septic shock which was observed in 110 cases 40.7 % cases. In our study which was statistically significant (p value< 0.001).

40.7% of patients with septic shock had elevated troponin I levels which indicates ongoing severe myocardial depression caused by various mechanisms of sepsis, which indicates increased morbidity and close monitoring of these patients is required.

96.5% of patients with gram negative and 63.6% gram positive bacteria were isolated in troponin I positive which was statistically significant (p value 0.05) which indicates that there could be endotoxin associated myocardial depression as it is one of the significant exogenous myocardial depressant substance released by gram negative bacteria proposed to cause myocardial depression, lower SVR, lower mean EF by activating inflammatory mediators and inflammatory cascade and similar inflammatory damage by other mechanisms by gram positive bacteria.. Isolation of fungi and virus (H1N1) also contribute to elevation of troponin I by activating inflammatory mediators was also statistically significant.

It shows that cTnI is not only linked to myocardial infarction, which is a sign of permanent ischemia damage, but it can also be raised in circumstances that induce inflammatory, toxic, reversible heart damage.

When the underlying myocardial injury is undergoing, the ECG and ECHO may be normal in the early stages of sepsis and septic shock.

Troponin I has a higher sensitivity for small myocardial injury, and its isoform is encoded by specialised genes exclusive to the myocardium, making it a more reliable indication for detecting myocardial damage, which is present in the majority of patients in sepsis and septic shock.

CONCLUSION

In addition to ischemia damage caused by MI, increased troponin I levels in sepsis and septic shock indicate inflammatory and toxic damage to the heart. As a result, it can be utilised as a marker for silent my ocardial damage found in sepsis and septic shock, which may not be detected early on by ECG and ECHO in early sepsis.

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