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Original Research Article

Study of association and prognostic correlation of cardiac troponin I estimation in acute decompensated heart failure patients

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ABSTRACT

Background: Heart failure is a major public health problem since last few decades affecting significant number of people worldwide. Acute decompensated heart failure is a major cause of hospitalization in elderly people with a high mortality rate. Heterogeneity and non-specificity of symptoms makes diagnosis of heart failure by clinical presentation alone more challenging. Aim of current study was to investigate troponin biomarkers in diagnosis, prognosis and management of acute decompensated heart failure.
Methods: Present study was a prospective observational study conducted on 100 patients at Department of Cardiology, Superspeciality hospital, NSCB medical college Jabalpur and Department of cardiology Superspeciality hospital, SS medical college Rewa from October 2019 to August 2020. Patients were investigated for clinical, echocardiographic parameters and NYHA classification. Cardiac functions were analyzed by color doppler echocardiography.
Results: According to study findings, 65.2% of TnI positive patients were males whereas 34.8% were females. Mean age of TnI positive group was observed to be higher. Majority of troponin positive patients were in NYHA class IV. Recurrent hospitalization was observed more in TnI positive group. Logistic regression analysis depicted systolic blood pressure reduced significantly ($p<0.001$) on follow up study in TnI positive patients, FBS was significantly more in TnI positive patients (131.4+42.9 mg/dl) ($p=0.049$). LVID was significantly more in TnI positive patients ($p=0.022$). Reduction in EF was statistically significant ($p=0.03$) at the three months follow up study.
Conclusions: A positive prognostic correlation was established between ADHF and troponin positivity, large prospective randomized trials are necessary to recommend quantitative troponin I determination in all patients of acute decompensated heart failure for prognosis and guiding therapy.

Keywords: Acute decompensated heart failure, Biomarkers, Troponins, Prognosis

INTRODUCTION

Heart failure (HF) is defined as cardiac dysfunction resulting in an inability to provide sufficient blood flow to meet the needs of the body for oxygenated blood during regular activity or to do so with elevated pressure.[1] Heart failure (HF) is a complex syndrome which is usually a chronic and progressive condition, accompanied with enormous societal burden in terms of morbidity, cost and mortality.[2] Acute decompensated HF (ADHF) is defined as the acute or gradual onset of clinical progression of HF requiring urgent medical care.[3] Cardiovascular and noncardiovascular conditions as well as patient-related and iatrogenic factors may result in rapid development or deterioration of signs and symptoms of heart failure, leading to an acute heart failure episode that usually requires hospitalization.[4]

HF contributes as a major factor in public health problem; affecting a total of 26 million people worldwide.[5]

Significant increased rates were observed in the incidence of HF over the last three decades. The prevalence of HF usually rises with age and follows an exponential pattern.[5,6] Heart failure affects are more dominant to aging population, mostly over the age of 65 years.[7,8] ADHF is a major cause of hospitalization in elderly people with in-hospital mortality rate ranging from 4% to 7%.[8] The burden of cardiovascular disease is increasing in India too with an estimated prevalence of 2-3/1000 population. Mortality rates due to ADHF have been reported as high as 16.7% in Indian hospitals, compared with rates of 4-7% in Western country hospitals.[9-12]

The diagnosis of heart failure is basically dependent on clinical symptoms and signs rather than any standalone test.[2,3] Patients with HF exhibit signs and symptoms that are often nonspecific making diagnosis by clinical presentation alone more challenging. Some signs and symptoms, such as dyspnea, orthopnea and paroxysmal nocturnal dyspnea, are due to congestion while some are due to lack of adequate cardiac output, including fatigue, weakness and exercise intolerance.[2-4] This heterogeneity of presentation often results in delays in definitive diagnosis and treatment of HF due to poor prognosis.[3,4]

Biomarker testing in HF is prevalent since several decades, first analysis of CRP (C-reactive protein) as a biomarker assay from serum of HF patients was reported by Braunwald et al.[13,14] With significant investigations the role of biomarkers is increasingly recognized in heart failure (HF) management. Biomarkers are predicted to supplement traditional clinical and laboratory testing to improve understanding of the complex conditions like HF.[13-15] Biomarkers can also be used to assess conditions such as fibrosis, inflammation, myocardial injury, and remodeling in a HF patient. Specifically, biomarkers in HF can be used to make initial diagnosis, to aid in prognosis and to determine patient's response to therapeutic intervention.[16,17] Clinical biomarker testing in HF has three important goals of identifying possible causes of heart failure, confirming the presence of the heart failure syndrome and estimating the severity of heart failure along with risk of progression.[13,16] For a HF biomarker to be useful, it should possess qualities like; thorough method by which a biomarker can be judged, robust assays procedure to measure biomarker, specific pathophysiologic pathway involved in the HF condition represented by the biomarker, delivery of more elaborative information other than what is already available by routine physical examination and laboratory evaluation and value added clinical judgment for understanding diagnosis, prognosis, or management of HF.[16]

A number of HF-related biomarkers like natriuretic peptides, C-reactive proteins, adrenomedullin, copeptin, neutrophil gelatinase-associated lipocalin, endothalin, arginine vasopressin, quiescinQ6, chromagranin-A, galectin-3, osteoprotegerin-A, adiponectin, aldosterone, growth differentiation factor-15, troponins etc. have been investigated in HF condition, to diagnose heart failure as well as to define adverse prognostic effect due to presence of any of the listed biomarkers. Current study is focused on determining the role of troponins as biomarkers for HF.[16-19]

Biomarkers like troponin serve two potential roles, they provide insight into the pathophysiology of the disease and aid in clinical decision making by clarifying diagnosis, prognosis or response to therapies.[19,20] Troponins are proteins involved in the regulation of cardiac muscle contraction. Different isoforms of troponin (C, I and T) exist within cardiac and skeletal muscle and regulate the calcium mediated contractile process of striated muscle.[21] Troponin T (TnT) and troponin I (TnI) forms of troponin are cardiac muscle specific and unlike troponin C, they are not expressed in skeletal muscle. T and I forms of troponin are not detected in the blood of healthy people but they leach out of the cell when membrane integrity is compromised, in conditions like hypoxia, ischemia, sepsis.[22] Therefore increases in circulating cardiac troponins are highly specific for ongoing myocardial damage and have been utilized for the past two decades as markers for defining myocardial infarction. One possible explanation for elevated cardiac troponins in chronic heart failure condition is due to reversible or irreversible myocardial demand and supply mismatch.[21,22] TnT and TnI typically increases more than 20 times above the reference range in conditions of myocardial dysfunction, this feature of cardiac specific troponin assays provide an improved signal to noise ratio, enabling the detection of even minor degrees of myocardial necrosis.[22] Troponins levels are observed to be increased in patients with heart failure, where they can be used to predict mortality and ventricular rhythm abnormalities. Release of cardiac troponins may also signify increased cardiomyocyte turnover in the setting of progressive myocardial dysfunction.[23] Elevated troponin levels in patients with either acutely decompensated heart failure or compensated heart failure in the outpatient setting are associated with acute mortality risk. Thus, troponin is a promising biomarker in acute HF, being associated to disease severity, worse clinical outcomes, and increased mortality.[22-24]

Cardiac troponin levels may be elevated in both acute and chronic heart failure, however, elevated concentrations of circulating cardiac troponins do not necessarily prove an ischaemic aetiology, troponins can also aid risk stratification in acute pulmonary embolism as a differential diagnosis of acute HF. Other settings with less clear mechanism of myocardial injury such as septic shock, pulmonary embolism, myocarditis, drug induced cardiotoxicities and renal dysfunction also exhibit elevated troponin levels.[23-25]

Different genes encode TnT and TnI in cardiac and skeletal muscle, thus permitting the production of specific antibodies for the cardiac troponin forms (TnT and TnI) that enable their quantitative assay.[25] After an episode of myocardial ischemia detectable troponin levels may not

appear for up to 12 hours, so an initially undetectable levels does not equate to no myocyte death and repeat up to 12 hours from symptom onset are essential for correct and safe diagnosis.[25] Levels may remain detectable for 14 days and this offer diagnostic help for events that occurred several days before presentation but make the diagnosis of reinfarction difficult unless frequent estimates are performed. Thus, troponin assay may be used as additive or confirmatory test to that of the natriuretic peptide biomarker assay.[23-25]

Aim and objectives

Aim of current research study was to investigate the prognostic value of high sensitivity troponins in patients with acute decompensated heart failure. Specific objectives of the presented research were to correlate the prognosis of ADHF (acute decompensated heart failure) patients with elevated TnI levels and to evaluate the adverse outcome of TnI positive patients in 3 months follow up period.

METHODS

Study type, population and duration

The study was a prospective observational study conducted from October 2019 to August 2020.

Sample size

Total 100 patients, with acute decompensated heart failure admitted to indoor department of cardiology, Department of cardiology, NSCB medical college, Jabalpur and Department of cardiology, SS medical college, Rewa were studied. 50 patients from each centre were included in the study who satisfied the inclusion criteria.

Inclusion criteria

Inclusion criterion for the patients to be enrolled in current study was; all male and female patients with acute decompensated heart failure whose age was≥18 years and who gave their consent.

Exclusion criteria

Exclusion criteria for the patients to be enrolled in current study were; patients with myocardial infarction in the last 4 weeks, with serum creatinine levels>2 mg/dl and patients with age≤18 years.

Procedure

Total 100 patients of acute decompensated heart failure were investigated through clinical examination and laboratory evaluation. Patients were given adequate treatment for their condition. Within 24 hours of admission quantitative serum analysis of troponin I was done and the patients were categorized as troponin I positive (>50 ng/l) or troponin I negative (<50ng/l). Patients were evaluated for diabetes and impaired renal function by testing admission fasting blood sugar and creatinine respectively. Other laboratory investigations like hemoglobin, blood urea, serum sodium and potassium were performed. Cardiac functions were analyzed by two-dimensional, m-mode and colour doppler echocardiography using ASE (American society of echocardiography) diagnostic criterion of echocardiography. Measurement of LV dimensions and function was determined by the use of average of 3 cycles. All patients of acute heart failure received standard treatment of heart failure with diuretics, ACE inhibitors, betablockers, and spironolactone in optimum dosage. Digoxin was added if required. Some patients who presented shock and hypotension were treated with ionotropic drugs like dopamine, dobutamine and noradrenaline. Patients were advised to attend the cardiology outpatient department for follow up evaluation after three months. Patients unable to visit for follow up were excluded from the study. During follow up the patients were again investigated for clinical examination and echocardiographic parameters for comparison, NYHA (New York heart association) class of the patients during follow up was determined and compared with the recorded class during admission.

Statistical analysis

Central tendency and variability in data were determined through mean and standard deviation respectively. Chi square or binominal test were used for comparison of data. Comparison of groups was done through t test and ANOVA test. Quantification of association between two variables was done through Spearman correlation test and to predict value from other measured variables, simple or multiple regression analysis was done. Pooled data was analyzed using SPSS statistical software.

RESULTS

According to findings of current study 65.2% of patients in TnI positive group are males whereas 34.8% patients were females. In troponin negative group, 55.9% were males and 44.1% were females (Table 1).

Table 1: Sex distribution in TnI positive and negative patients.

Sex	TnI<50 (n=34)	TnI≥50 (n=66)
Male, frequency (%)	19 (55.9)	43 (65.2)
Female, frequency (%)	15 (44.1)	23 (34.8)

Average age of patients in current study was 67.17±14.9 years. Minimum, maximum and mean age for TnI positive patients were observed to be 29 years, 90 years and 68.07±14.61 years respectively and for TnI negative patients it was observed as 19 years, 94 years and 65.35±15.59 years respectively. Mean age in TnI positive

group was observed to be higher than in negative group (Table 2).

Table 2: Age distribution in TnI positive and negative patients.

Age (years)	TnI<50 (n=34)	TnI≥50 (n=66)
Mean±SD	65.35±15.59	68.07±14.61
Range	19-94	29-90

Total 88% of troponin negative patients and 86% of troponin positive group were in NYHA class IV. On discharge, 26.4% of troponin negative patients and 73.8% of troponin positive group were in NYHA class IV. Upon follow up, 23.5% of TnI negative and 77.5% of TnI positive patients were observed in NYHA class IV (Table 3).

Table 3: Distribution of TnI positive and negative patients as per NYHA classification.

NYHA class	TnI<50 (n=34), frequency (%)	TnI≥50 (n=66), frequency (%)
Admission		
2	0	0
3	4 (11.8)	9 (13.6)
4	30 (88.2)	57 (86.4)
Discharge		
2	4 (11.8)	0
3	21(61.8)	16 (26.2)
4	9 (26.4)	45 (73.8)
Follow up		
2	11 (32.4)	2 (4.1)
3	15 (44.1)	9 (18.4)
4	8 (23.5)	38 (77.5)

Table 4: Number of hospitalization and mortality rate amongst TnI positive and negative patients.

Number of hospitalization	TnI<50 (n=34), frequency (%)	TnI≥50 (n=66), frequency (%)
1	26 (76.5)	14 (21.2)
2	6 (17.7)	36 (54.5)
3	1 (2.9)	14 (21.2)
4	1 (2.9)	2 (3.1)
Mortality		
Death in hospital	3 (8.8)	23 (34.8)
Death outside hospital	0	6 (9.1)

Recurrent hospitalization (≥2 times) was a common problem observed in current study. 54.5% patients in the TnI positive group were hospitalized two times in comparison only 17.7% of patients in the negative group. More than two times hospitalization was observed more in TnI positive group than negative group (Table 4). In hospital mortality was higher in the TnI positive group compared to negative group. Death outside hospital was nil in TnI negative patients but was observed to be 9.1% in TnI positive group (Table 4).

Ejection fraction (EF) which is a measure of systolic function was observed to be 35.6±4.3 at admission in TnI negative group and 30.2±6.7 for positive group. On discharge, EF was found to be 28.1±7.5 for TnI positive group and 35.3±5.2 in the negative group. At 3 months follow up EF was observed as 28±7.6 in the troponin positive group, in comparison to 36.8±7.3 in the negative group (Table 5). Mean systolic blood pressure was observed to be higher in troponin negative patients on admission (109.1±14.1 mmHg) than troponin positive patients (91±10.5 mmHg). At 3 months follow up SBP was much lower in TnI positive patients (91±10.3 mmHg) than in TnI negative patients (108.8±10.5 mmHg) (Table 5). In TnI positive cases mean fasting blood sugar level on admission was found to be 131.4±42.9 mg/dl which was higher than that of troponin negative patients, 98.9±33.2 mg/dl (Table 5).

Left ventricular internal dimensions (LVID) on admission in TnI positive patients were observed to be 63.4±8.5 mm and in negative patients it was 56.8±6.2 mm. On follow up study, LVIDD increased to 65±8.4 mm in troponin positive patients but decreased to 55.5±8.0 mm in troponin negative patients (Table 5). Systolic internal diameter on echocardiography was found to be 46.6±5.8 mm in troponin I negative patients and 53.7±8.9 mm in troponin positive patients on admission. At discharge, it was 57.9±15.8 mm in TnI positive group and 49.3±20.2 mm in troponin negative patients (Table 5). Duration of stay in hospital was observed to be 11.9±5.3 days in TnI positive patients whereas it was found to be 5.9±3.6 in negative patients of troponin. Number of hospitalization was 2.1±0.74 times in troponin positive patients and 1.3±0.68 times for troponin negative patients.

The logistic regression analysis of data collected depicted following findings; systolic blood pressure was reduced significantly ($p<0.001$) on follow up study in TnI positive patients. FBS was significantly increased on admission in TnI positive patients (131.4+42.9 mg/dl) in comparison to TnI negative patients (98.9+33.2 mg/dl) ($p=0.049$). LVID (diastolic) was significantly increased at admission, discharge and follow up study in TnI positive patients. The increase was observed to be statistically significant at three month follow up examination ($p=0.022$). LVID (systolic) was raised in both the groups but the increase was more in TnI positive patients and statistically significant at the time of discharge ($p=0.002$). Reduction in EF (%) was statistically significant ($p=0.03$, CI=0.75 to 1.94 and odds ratio 0.84) at the three months follow up study. Duration of stay in hospital was found to be significantly more in TnI positive patients than in troponin negative patients ($p<0.001$) (Table 6).

Table 5: Distribution of ejection fraction, systolic blood pressure, fasting blood sugar levels, LVID (systolic and dystolic), duration of stay and number of hospitalization with respect to TnI.

Variable	TnI <50 (n=34)				TnI ≥50 (n=66)			
	Mean	SD	Min.	Max.	Mean	SD	Min.	Max.
Ejection fraction (%)								
Admission	35.6	4.3	24	40	30.2	6.7	20	40
Discharge	35.3	5.2	25	43	28.1	7.5	15	43
Follow-up	36.8	7.3	20	46	28.0	7.6	17	47
Systolic blood pressure (mmHg)								
Admission	109.1	14.1	80	132	96	10.5	72	120
Discharge	105.5	10.9	88	132	91.2	10.3	74	132
Follow-up	108.8	10.5	90	126	91.0	10.3	72	120
Fasting blood sugar (mg/dl)								
Admission	98.9	33.2	38	242	131.4	42.9	76	327
LVID (mm) (diastolic)								
Admission	56.8	6.2	47	76	63.4	8.5	47	79
Discharge	55.4	6.9	47	74	64.9	8.9	48	78
Follow-up	55.5	8.0	39	72	65	8.4	46	78
LVID (mm) (systolic)								
Admission	46.6	5.8	39	66	53.7	8.9	39	69
Discharge	45.7	6.6	37	60	55.5	9.2	39	71
Follow-up	49.3	20.2	36	156	57.9	15.8	38	148
Duration of stay in hospital (days)	5.9	3.6	2	21	11.9	5.3	1	24
Number of hospitalization	1.3	0.68	1	4	2.1	0.74	1	4

Table 6: Logistic regression analysis results of ejection fraction, systolic blood pressure, fasting blood sugar levels, LVID (systolic and dystolic), duration of stay and creatinine with respect to TnI positivity.

Variable	Odds ratio	Std. error	95% CI	P value
Ejection fraction (%)				
Admission	0.98	0.07	0.85-1.14	0.862
Systolic blood pressure (mmHg)				
Admission	0.99	0.03	0.94-1.05	0.838
Discharge	1.01	0.04	0.93-1.10	0.788
Follow-up	0.86	0.04	0.79-0.93	<0.001
Fasting blood sugar (mg/dl)				
Admission	1.02	0.01	1.00-1.04	0.049
LVID (mm) (diastolic)				
Admission	0.89	0.07	0.76-1.05	0.172
Discharge	0.99	0.13	0.77-1.28	0.972
Follow-up	1.23	0.11	1.03-1.46	0.022
LVID (mm) (systolic)				
Admission	0.87	0.06	0.75-1.01	0.064
Discharge	1.27	0.09	1.09-1.48	0.002
Follow-up	1.01	0.01	0.97-1.03	0.953
Duration of stay in hospital (days)	1.38	0.09	1.20-1.59	<0.001
Creatinine				
Admission	0.33	0.09	0.15 - 0.51	<0.001

DISCUSSION

Current study depicts significant number of HF patients (N=66) were positive for TnI. In HF trial 64% patients were positive for TnI at baseline. In a study reported by Shakuja et al 46% of patients had measureable troponin I,>0.01 ng/ml. In reports of Fig et al nearly all patients with acutely decompensated heart failure had a highly sensitive troponin I or T value above 99 percentile.[26] Miller et al and Perna et al also reported increased incidence of detectable troponins in their studies.[27,28] Current study findings that

show 66% HF patients as positive TnI corroborate the findings of earlier reported literature.

In-hospital mortality was higher (23.88%) for troponin positive patients (n=23) in comparison to troponin negative patients (3.12%) (n=3). Out of hospital death rates were also higher in TnI positive patients (n=6) (9.1%) when compared with troponin negative patients (n=0). These results corroborate with previous studies of Latini et al and Kocial et al that showed higher incidence of troponin positivity in ADHF patients and prognosis.[29,30] Carlo et al in their studies found a clear cut association of increased incidence of death in troponin detectable and/or troponin positive patients of acutely decompensated heart failure patients.[31] In ADHERE registry; mortality was reported to be 8% in troponin positive patients versus 2.7% in negative patients for troponin. In published study report, at 24 months of followup of ADHF patients, 16.5% mortality was observed in patients with non-measurable troponin versus 43.3% in patients who had measureable TnT.[30,31] In Warris et al study of acute heart failure, 51.1% patients had TnI and 29.7% had TnT levels above the cut off and mortality at 6 months was reported to be 18.7%.[32] Figal et al found 83% of patients had an TnT level above the 99 percentile value of 0.013 ng/ml. A total of 29 patients died (27.1%) and the patients who died had significantly higher concentration of TnT (0.028 to 0.124 ng/ml).[33] It was observed in through the current study findings that recurrent hospitalization was a problem, which was very much significant in troponin positive patients in comparison to troponin negative patients this was in concordance with the earlier reports.[30-33]

Multiple investigators like Carlo et al and Hudson et al found significant association between detectable troponins and HF rehospitalisations.[31,34] Duration of stay in hospital was more in troponin I positive patients as observed in current study ($p<0.001$). Latini et al also demonstrated that patients with heart failure who were positive for troponins required greater use of hospital resources including longer stay in hospitals and intensive care units.[30] According to current study findings ejection fraction which is an indication of cardiac systolic function is low in both troponin I positive and negative patients on admission but the deterioration of systolic function was noted to be significant during the followup period in troponin positive patients ($p=0.003$). NYHA functional class which denotes clinical condition of the patient was mostly III or IV during admission in both troponin I positive or negative patients of ADHF, but with time symptoms and signs deteriorated more in the troponin positive patients and accordingly NYHA class changed on a higher direction (like III to IV) as per the present study findings.[32-34]

Thus, current study demonstrated that the patients who were positive for troponin I had worse prognosis than troponin I negative patients and with time there was deterioration of symptoms or functional class in these patients. These findings are corroborative with earlier reports and indicate that there is progressive cariomyocyte loss from myonecrosis and apoptosis.

But current study could not test for TnI in the follow up or as follow up period was only three months in current study, it could not be definitely commented whether with therapy troponin I concentration decreases with time or whether troponin has definite relation in determining prognosis and guiding therapy in acutely decompensated heart failure patients. More prospective randomized trials are necessary for concrete recommendation.

Limitations

Limitations of current study were; the small sample size of the study group is not adequate to make recommendations for performing TnI test for every patient of ADHF. TnI concentrations could not be measured in the follow up to compare whether the rising or decreasing troponin levels has some correlation with prognosis in ADHF patients, moreover the follow up period in current study was only three months, more significant correlations could have been drawn with the investigation for a longer follow up period. Correlation of cardiac troponin with other biomarkers like NTproBNP could not be established for prognostication due to limitation of resource and logistics.

CONCLUSION

The present study conducted to determine the prognostic correlation of cardiac troponin I estimation in acute decompensated heart failure patients admitted in a tertiary care hospital depicts a higher troponin I concentration in patients of ADHF. The patients with troponin I positivity show higher mortality (both in hospital and outside) and recurrent hospitalization in comparison with troponin I negative patients. Duration of stay in hospital was observed to be higher for TnI positive patients when compared with troponin negative patients. NYHA class and ejection fraction changed in a negative direction in patients of ADHF who were troponin positive. Though the present study findings, establishes a positive indication in making prognostic correlation of ADHF patients with troponin positivity, large prospective randomized trials are necessary before coming to definite conclusion in making recommendation for determining quantitative troponin I in all patients of acute decompensated heart failure for prognosis and guiding therapy.

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