International Journal of Research in Medical Sciences
Prabhakaran SP et al. Int J Res Med Sci. 2017 Oct;5(10):4363-4368
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20174559

Original Research Article

Prognostic significance of troponin T in acute myocardial infarction

Sunil Prasobh Prabhakaran[1], Abhilash Kannan[2]*

[1]Department of Medicine, Government Medical College, Thiruvananthapuram, Kerala, India
[2]Department of Medicine, Sree Gokulam Medical College, Venjarammoodu, Thiruvananthapuram, Kerala, India

Received: 16 July 2017
Accepted: 28 August 2017

***Correspondence:**
Dr. Abhilash Kannan,
E-mail: drabhilashkannan@yahoo.com

ABSTRACT

Background: Cardiac markers traditionally have been used only to establish the diagnosis in patients with acute coronary syndromes. In those with suspected acute STEMI, markers have been deemed to have little value, although smaller studies have suggested that troponin T may be valuable for risk stratification. Study aim was to study the prognostic significance of admission Troponin T in acute STEMI and also the relation between Troponin positivity and ST segment resolution after thrombolysis and also relationship with ejection fraction by echocardiogram.
Methods: This was a descriptive study conducted in 50 patients admitted with acute STEMI within eight hours in the department of medicine in a tertiary care centre in South Kerala. A blood sample was sent for assessing troponin T. All Patients underwent thorough clinical examination and investigations including echocardiogram was done and were managed with thrombolysis. They were closely followed up for in hospital and 30 days mortality and complications. ST segment resolution after thrombolysis with streptokinase was also assessed.
Results: In present study 48% of the patients were troponin T positive. Total six patients died of which all were Troponin T positive. There was a significant increase in the complications in troponin T positive group (46% vs 16%). 44% of the patients had an anterior wall myocardial infarction of which 46% had complications. ST segment resolution after thrombolysis was below 30% in 66.7% of the troponin T positive patients. Ejection fraction was below 50% in 80% of troponin T positive patients.
Conclusions: There was a statistically significant correlation between admission troponin T levels and in hospital complications and also mortality rates at 30 days. Troponin T positivity at admission was significantly associated with lower rates of reperfusion after thrombolysis with streptokinase and also lower rate of ejection fraction on echocardiogram. Troponin T positive anterior wall myocardial infarction was associated with more complications than non-anterior wall myocardial infarction.

Keywords: Ejection fraction, STEMI, Streptokinase, Troponin T, Thrombolysis

INTRODUCTION

The classic role of biochemical markers of myocyte damage in patients admitted with acute coronary syndromes has been retrospective confirmation of myocardial infarction. ST -segment elevation, if present, occurs very shortly after coronary artery occlusion, is highly specific for myocardial infarction and identifies the only group that unequivocally benefits from the use of thrombolytic agents.[1,2] Routine early measurement of biochemical markers has been deemed unnecessary in this patient group. The newer markers of myocardial damage like troponin T (TnT) is highly cardiac specific, and in the setting of myocardial injury is released into the circulation slightly earlier than creatinine kinase (CK).[3,4] It also has prognostic significance, because if present, it

identifies a subgroup of patients who are high risk for early cardiac events.[5,6] The purpose of the present study was to examine the prognostic value of admission TnT concentration measurement in patients with myocardial infarction defined according to WHO criteria.

There are evidences suggesting that an elevated cardiac troponin T (cTnT) at admission is also associated with an increased cardiac risk in patients with acute ST-segment elevation myocardial infarction (STEMI).[7-9] The reason why an elevated cTnT at admission bears prognostic information is unclear but may be related to more extensive infarction in patients presenting later after onset of symptoms, to a higher failure rate of recanalization and to less efficient microvascular perfusion.[10,11] Lower rates of unimpeded epicardial flow in the infarct-related artery following streptokinase therapy have been reported in patients with an elevated cTnT at admission.[9] There is evidence suggesting that lower rates of successful recanalization, and persistently more severely impaired microvascular reperfusion, despite successful restoration of normal epicardial flow in cTnT- positive patients undergoing primary percutaneous coronary intervention (PCI).[10]

The aim of present study was to assess correlation between admission cTnT levels in patients admitted with acute myocardial infarction within eight hours and in hospital complications and 30 days clinical outcome including death, reinfarction, left ventricular failure (LVF), cardiogenic shock, post-infarction angina (PIA) and arrhythmias. We also assessed the correlation between admission troponin T (TnT) and ST segment resolution in ECG after thrombolysis with Streptokinase.

METHODS

Current study was a hospital based descriptive study conducted in patients admitted within 8 hours of the onset of chest pain who were diagnosed to have acute STEMI. The study was conducted in the intensive care unit (ICU) under department of medicine, in a tertiary care hospital in South Kerala over a period of one year. Study exclusion criteria were: 1) recent acute myocardial infarction / unstable angina within two weeks, 2) renal failure, 3) skeletal muscle injury, 4) patients with history of prolonged CPR, 5) patients on temporary pacemaker. Only those patients admitted with history of chest pain within eight hours were included for the study. Detailed history regarding nature of chest pain, risk factors were taken and patients were examined and investigated. ECG was taken to confirm STEMI. An echocardiogram was done to evaluate ejection fraction. At the time of admission to ICU a serum sample was sent for estimating troponin T level. All the patients were given thrombolysis with streptokinase after ruling out any contraindications.

ST segment elevation was judged to be present if >1 mm elevation was present in two contiguous leads. The sum of the ST segment elevation was recorded at base line and at 90 minutes and 180 minutes after thrombolysis with Streptokinase.

Percentage decrease in the sum of ST segment score (reperfusion criteria) between the baseline and 90mts is considered as mild (<30%), moderate (30-50%) and good (>70%). Full clinical details of all patients were recorded. Particular attention was paid to history of coronary artery disease in the past, nature of chest pain, risk factors for MI, admission clinical findings, in hospital clinical course. A 30 day follow up of all the patients were also done. Complications which were particularly looked for was post-infarction angina, reinfarction, left ventricular failure, cardiogenic shock, arrhythmias and death. Survival status and cause of death were established for all patients. Blood samples were sent at admission for estimating troponin T levels. Troponin T was assayed using electro chemiluminescence immunoassay (ECLIA). Troponin T was considered positive if values >0.1 ng/ml.

The base line data were analyzed using the percentage methods. The comparisons and associations among different groups were explored using suitable tests like cross tabulation and Chi square test. Pearson's correlation analysis was performed to find out the relationship between different parameters especially with the Troponin T levels. The troponin T levels and complications were subjected to Regression analysis to find out the relationship between them.

RESULTS

Of the 50 patients enrolled in the study there were 42 males (84%) and eight females (16%). The age of the patients varied from 29 years to 80 years. The number of patients were more in the age groups between 40-49 years and >70 years. There were 15 (30%) patients in the age group between 40 and 49 years (Figure 1).

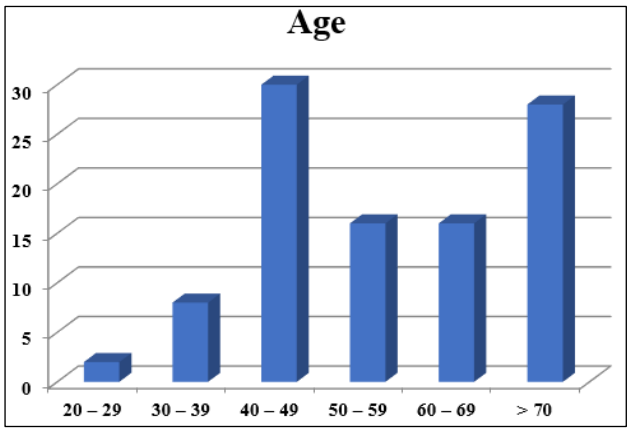
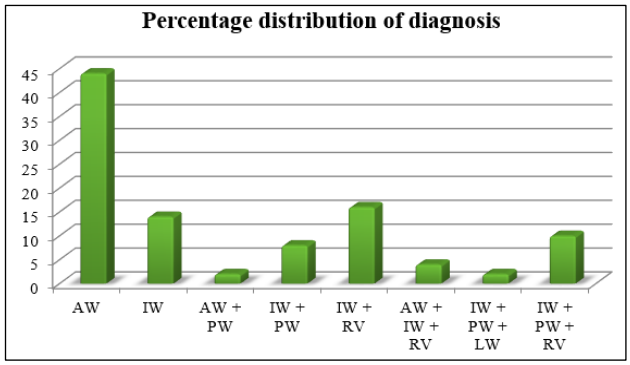


Figure 1: Age distribution.

The number of patients with diabetes, hypertension and dyslipidemia were 11 (22%), 16 (32%), 32 (64%) respectively. About three patients (6%) had history of coronary artery disease and a family history of coronary

artery disease. About 33 (66%) patients gave history of smoking.



AW- anterior wall, IW- inferior wall, PW- posterior wall, LW- lateral wall, RV- right ventricular.

Figure 2: Types of myocardial infarction.

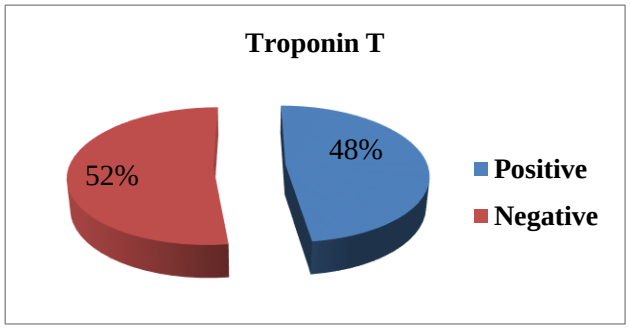


Figure 3: Distribution based on troponin positivity.

Of the 50 patients 22 (44%) patients had anterior wall myocardial infarction alone and seven (14%) had inferior wall myocardial infarction alone. The rest 21 (42%) had combinations of anterior, inferior, posterior and right ventricular myocardial infarction (Figure 2). Troponin T was positive in 24 (48%) and negative was 26 (52%) (Figure 3).

Table 1: Complications based on troponin T positive versus negative.

	Negative	Positive
PIA	2	8
LVF	1	
Cardiogenic shock	1	
Arrhythmias	3	1
Death	1	1
PIA+LVF		4
PIA+arrhythmias		2
PIA+death		1
Arrhythmias+death		1
PIA+LVF+death		1
PIA+LVF+cardiogenic shock+ death		1
All except death		1
All		2
Total	8	23

$X^2 = 16.415$; $p < 0.05$.

Table 2: Distribution of complications based on troponin levels.

	Troponin T			
	< 0.01	0.1 to 0.99	1 to 1.99	≥ 2
PIA	2 (20%)	7 (70%)	1 (10%)	
LVF	1 (100%)			
Cardiogenic shock	3 (75%)			
Arrhythmias	1 (50%)	1 (25%)		
Death	1 (50%)	1 (50%)		
PIA + LVF		4 (100%)		
PIA + arrhythmias		2 (100%)		
PIA + death		1 (100%)		
Arrhythmias + death		1 (100%)		
PIA + LVF + death		1 (100%)		
PIA + LVF + cardiogenic shock + death				1 (100%)
All except death				1 (100%)
All			1 (50%)	1 (50%)
Total	8 (25.81%)	18 (58.06%)	2 (6.45%)	3 (9.67%)

($X^2 = 49.32$; $P < 0.05$).

The various complications of myocardial infarction like post infarction angina, acute left ventricular failure, cardiogenic shock, arrhythmias and death were studied in both Troponin T positive and negative groups (Table 1). Complications were more in TnT positive group and showed statistically significance. Relation between TnT levels and complications were evaluated (Table 2). Pearson's correlation analysis and regression analysis between complications and quantitative troponin levels were statistically significant ($r = 0.591$; $p < 0.01$).

Relation between troponin positivity and reperfusion at 90 minutes after thrombolysis with streptokinase were studied (Table 3) and was found to be statistically significant. TnT positive patients had significantly lower reperfusion at 90 minutes after thrombolysis. Relation between TnT positivity and Ejection fraction on echo was studied (Table 4). Troponin positive cases were significantly associated with lower ejection fraction.

Table 3: Troponin positivity and reperfusion at 90 minutes.

Percentage reperfusion at 90mts	Troponin		Total	X^2	P value
	Negative	Positive			
< 30	4	8	12	4.46	< 0.05
	33.3%	66.7%	100		
30-70	9	12	21	4.13	< 0.05
	42.9%	57.1%	100		
> 70	13	4	17	4.76	< 0.05
	76.5%	23.5%	100		
Total	26	24	50	6.45	< 0.05
	52%	48%	100		

Table 4: Troponin T positivity and ejection fraction.

Ejection fraction	Troponin		Total	X^2	P value
	Negative	Positive			
< 50	2 (20)	8 (80)	10 (100)	8.791	< 0.05
50-70	19 (54.3)	16 (45.7)	35 (100)		
> 70	5 (100)		5 (100)		
Total	26 (52)	24 (48)	50 (100)		

Table 5: Relation between troponin, type of AMI and complications.

Troponin T		Diagnosis							Total	X^2	P value
		Aw	Iw	Aw + pw	Iw + pW	Iw + RV	Aw + iw + RV	Iw + pw + RV			
- VE	Count % within complications	2	2		2			2	8	14.667	> 0.05
		25	25		25			25	100		
+ VE	Count % within complications	14	2	1		3	1	2	23	68.726	< 0.05
		60.9	8.7	4.3		13	4.3	8.7	100		

Table 6: Relation between troponin T positivity and death.

Troponin T		Death		Total
		Nil	Yes	
Negative	Count % within complications	25	1	26
		96.1	3.9	100
Positive	Count % within complications	18	6	24
		75	25	100

Relationship between TnT, type of myocardial infarction and complications were studied (Table 5). Anterior wall myocardial infarction with TnT positive was associated with more complications. We also studied the relationship between TnT positivity and death (Table 6).

DISCUSSION

Cardiac markers traditionally have been used only to establish the diagnosis in patients with acute coronary syndromes. In those with suspected AMI and ST-segment elevation, markers have been deemed to have little value, although smaller studies have suggested that troponin T may be valuable for risk stratification. This study clearly defined that a positive quantitative troponin T on admission is an independent marker of higher 30-days mortality and other complications. Furthermore, the base-line cardiac troponin T level provides incremental prognostic information even when there is ST segment elevation. Therefore, this study not only confirms the

observations of small trials in selected patients with unstable angina but also brings to the notice the importance of a single blood test obtained early for risk stratification of patients with myocardial infarction.

The present study was conducted in 50 patients admitted in ICU with AMI. Those patients admitted within 8 hours of onset of ischemic chest pain were selected for the study. Of the 50 patients 42 (84%) patients were male. Of the age distribution 15 patients (30%) were in 40-49 age group and 14 patients (28%) in >70 years group. Median age group of our patients was 54 years. This was compared with Ohman et al, Kurowski et al (62.8±11).[7,12] Thus, median age group of patients was seen to be a decade earlier. As far as risk factors were considered patients who had diabetes mellitus was 22% comparable with Ohman et al 1996 (21%) and Gusto III trail (15.6%).[7] Patients with hypertension was 32%. This was low when compared with Ohman et al (50%).[7] Patients with dyslipidemia was 36% comparable with Ohman et al (1996) (41%). 66% patients were smokers comparable with Reddy et al (52%)and Yadav et al (58%).[13,14] Among the 50 patients, 44% had AWMI and rest 56% had non AWMI comparable with Kurowski et al (41% AWMI) and Ohman et al Gusto III trial (47.8%).[8,12] In this study 24 patients (48%) were TnT positive and 26 patients (52%) were TnT negative. This was comparable to Ohman et al (33%) and Kurowski et al (48%).

Of the seven patients died, six were troponin T positive which shows an increased mortality in TnT positive cases (25% versus 3.9%). This result was statistically highly significant. This result was comparable to results of various other clinical trials. Stubbs et al showed that admission TnT positive was associated with a higher risk of subsequent cardiac events and death on follow up (at 1 year 15% versus 5% and at 3 years 28% versus 7.5%).[9] Kurowski et al showed TnT positive was associated with higher mortality rates at 30 days (14.6% versus 3.5%) and 12 months (17.3% versus 4.6%).[12] Ohman et al for Gusto III investigators showed that positive TnT level was the variable most strongly related to 30 days mortality (13% versus 4.7%).[7] Ohman et al for Gusto III investigators showed that patients with elevated TnT has significantly higher mortality at 30 days (15.7% versus 6.2% for negative patients).[8]

In this study admission TnT positive patients had increased in hospital complications and 30-days clinical outcomes including death, left ventricular failure, cardiogenic shock, re-infarction, post-infarction angina, arrhythmias. There was a significant increase in complications in troponin T positive cases (46% versus 16%).

Relation between admission TnT and percentage reperfusion after thrombolysis with streptokinase were studied. It was found that percentage reperfusion was better in troponin T negative group compared with TnT positive groups (52% versus 48%). This was found to be statistically significant. When various grades of reperfusion were analysed, 24% was in poor reperfusion group, of which 66% was TnT positive. Of the 34% of the good reperfusion 76.5% was TnT negative. A similar study by Kazmi et al evaluated the relationship between admission TnT response to streptokinase in acute myocardial infarction.[15] TnT positivity was associated with poor response to streptokinase than TnT negative patients (37% versus 49%). TnT positive non-responders has higher mortality on 18 months follow up. Stubbs et al reported on less effective reperfusion (50 versus 72%) in admission TnT positive patients with STEMI after thrombolysis with streptokinase.[9]

Troponin T positive patients were associated with decreased ejection fraction on echocardiogram (48% versus 52%) This decrease in ejection fraction in TnT positive cases was found to be statistically significant. A study by Rao et al showed that TnT > 2.8 mcg/L predicted a LVEF <40%.[16]

Patients with various risk factors like smoking diabetes, hypertension, dyslipidemia were assessed in both TnT positive and negative groups. In all these cases when TnT positive cases were associated with these risk factors complications were more. This needs to be studied in a larger population.

TnT positivity was correlated with type of acute myocardial infarction and associated complications. Out of eight TnT negative patients with complications, two had AWMI. In 23 TnT positive patients with complications, 14 had AWMI. Thus, an increase in complications were found in TnT positive AWMI group. (60.9% versus 29.1%). This was statistically significant. A similar study by Kurowski et al showed that TnT could discriminate patients at high and low risk for cardiac death at 30 days and 12 months follow up among anterior wall (19.2% versus 7.9%) and non-anterior wall myocardial infarction.

The exact mechanism by which an elevated cTnT at admission is linked to mortality is unclear. Longer time intervals from onset of symptoms to reperfusion therapy represent an attractive explanation for the poorer prognosis in cTnT positive patients. However, even after elimination of a potential confounding effect of longer time intervals, cTnT remained an excellent predictor of long-term risk, indicating that additional mechanisms may be involved. The poorer prognostic value of duration of ischemia as assessed by ischemic pain might be explained by different thresholds of pain perception, episodes of spontaneous reflow and occlusion, and the alleviation of ischemia by collateral flow and ischemic preconditioning.

CONCLUSION

Admission troponin T positivity was significantly associated with increased in hospital complications

including death, LVF, cardiogenic shock, PIA, arrhythmias and mortality at 30 days. Quantitative admission TnT levels were directly proportional to the in-hospital complications and mortality rates at 30 days. TnT positivity at admission was significantly associated with lower rates of reperfusion after thrombolysis with streptokinase. TnT positivity at admission was significantly associated with lower ejection fraction on echocardiogram. TnT positive anterior wall myocardial infarction was associated with more complications than TnT positive non-anterior wall myocardial infarction.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Yusuf S, Pearson M, Sterry H, Parish S, Ramsdale D, Rossi P, et al. The entry ECG in the early diagnosis and prognostic stratification of patients with suspected acute myocardial infarction. Eur Heart J. 1984;5:640-6.
2. GISSI Investigators. Italian group for streptokinase study in myocardial infarction (GISSI). Effectiveness of intravenous thrombolytic treatment in acute myocardial infarction. Lancet. 1986;1:397-402.
3. Katus HA, Remppis A, Neumann FJ, Scheffold T, Diederich KW, Vinar G, et al. Diagnostic efficiency of troponin T measurements in acute myocardial infarction. Circulation. 1991;83:902-12.
4. Collinson PO, Stubbs P. The prognostic value of serum troponin T in unstable angina. N Engl J Med. 1992;327:1760-1.
5. Burlina A, Zaninotto M, Secchiero S, Rubin D, Accorsi F. Troponin T as a marker of ischemic myocardial injury. Clin Biochem. 1994;27:113-21.
6. Wu A, Valdes R, Apple F, Gornet T, Ingersoll-Stroubus I, Mayfield-Stokes S, et al. Cardiac troponin T immunoassay for diagnosis of acute myocardial infarction and detection of minor myocardial injury. Clin Chem. 1994;40:900-7.
7. Ohman EM, Armstrong PW, Christenson RH, Granger CB, Katus HA, Hamm CW. Cardiac troponin T levels for risk stratification in acute myocardial ischemia: GUSTOII an Investigators. N Engll J Med. 1996;335:1333-41.
8. Ohman EM, Armstrong PW, White HD, Granger CB, Wilcox RG, Weaver WD. Risk stratification with a point-of-care cardiac troponin T test in an acute myocardial infarction: the GUSTO-III Investigators. Am J Cardiol. 1999;84:1281-6.
9. Stubbs P. Collinson P. Moseley D, Greenwood T, Noble M. Risk stratification with appoint-of-care cardiac troponin T concentrations in myocardial infarction. Circulation. 1996;94:1291-7.
10. Giannitsis E, Muller-Bardorff M, Lehrke S, Wiegand U, Tolg R, Weidtmann B, et al. Admission troponin T level predicts clinical outcomes, TIMI flow, andmyocardial tissue perfusion after primary percutaneous intervention for acute ST-segment elevation myocardial infarction. Circulation. 2001;104:630-5.
11. Matetzky S, Sharir T, Domingo M, Noc M, Chyu KY, Kaul S, et al. Elevated troponin T level on admission is associated with adverse outcome of primary angioplasty in acute myocardial infarction. Circulation. 2000;102:1611-6.
12. Kurowski V, Hartmann F, Killermann DP, Giannitsis E, Wiegand UK, Frey N, et al. Prognostic significance of admission cardiac troponin T in patients treated successfully with direct percutaneous interventions for acute ST elevation myocardial infarction. Crit Care Med. 2002;30(10):2229-35.
13. Reddy KS, Shah P, Shrivastava U, Prabhakaran D, Joshi M, Puri SK, et al. Coronary heart disease risk factors in an industrial population of North India. Can J Cardiol. 1997;13(l B):3.
14. Yadav NA, Raghu K, Patnaik AN, Krishna LSR, Gouthami V. Emerging risk factors in acute myocardial infarction: a tertiary care centre study. Indian Heart J. 2003;55:(5):19.
15. Kazmi KA, Iqbal MP, Rahbar A, Mehboobali N. Admission troponin T is a prognostic marker and its relationship to streptokinase treatment in patients with acute myocardial infarction. Trop Doct . 2003;33(1):18-22.
16. Rao AC, Collinson PO, Rose AJ, John C, Canepa-Anson R, Joseph SP. Prospective evaluation of role of routine cardiac troponin T measurement to identify left ventricular ejection fraction less than 40% after first myocardial infarction. Heart. 2003;89:559-60.

Cite this article as: Prabhakaran SP, Kannan A. Prognostic significance of troponin T in acute myocardial infarction. Int J Res Med Sci 2017;5:4363-8.