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CORRELATION OF TROP T WITH LEFT VENTRICULAR DYSFUNCTION AFTER ACUTE MYOCARDIAL INFARCTION

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ABSTRACT

Coronary artery disease (CAD) is a modern epidemic. Amongst this myocardial infarction (MI) is the leading cause of death and disability. Cardiac markers, troponin complex, cTnI and cTnT increases after ST segment elevation myocardial infarction (STEMI), related to infarct size. Left Ventricular Ejection Fraction (LVEF) is the predictor of moribidity and mortality. This study is for correlation of troponin T and Left Ventricular dysfunction after MI in southern Rajasthan. **S**tudy conducted in MBGH, Udaipur, Rajasthan included 50 patients of Acute myocardial Infarction (AMI). cTnT was estimated in all patients between 12-48 hours after the onset of chest pain using ECLIA method. Echocardiographic calculation of LVEF and E/A ratio was done from 4 days to 4weeks after AMI. Male were affected more than females and age group of AMI was between 45-55years. Most common clinical presentation of AMI was chest pain and physical finding was hypertension. Most common risk factor for AMI was dyslipidemia and smoking, with clustering of other risk factors. Serum cTnT level was significantly higher in inferolateral MI, posterior wall MI and inferoposterior MI as compared to inferior MI as area necrosed is more in these sites, so it can be concluded that cTnT level is directly related to size of the infarcted area. Strong negative correlation was seen between cTnT level and LVEF so cTnT shows excellent promise as a useful marker of infarct size and for all assessment of LVEF. Serum troponin T concentration measured 6-48hours after onset of chest pain for first myocardial infarction is a reliable, simple, quick, cost effective and non-invasive method for identifying patients with LVEF < 50% for whom there is a poor prognosis.

Key words: Coronary artery disease, Left Ventricular Ejection Fraction, ST segment elevation myocardial infarction, Acute myocardial Infarction.

INTRODUCTION

Coronary artery disease is a modern epidemic [1]. It is now the leading cause of death worldwide and it is expected that rate of CAD will only accelerate in the next decade [2]. The world health organization (WHO) estimates that by 2020 the global number of deaths from CAD will have risen from 7.1 million in 2002 to 11.1million [3]. clinical spectrum is wide and ranges from angina, acute myocardial infarction(AMI), CHF, and sudden death, of all these syndromes AMI is the most dramatic & dreadful. Amongst this AMI is the leading cause of death and disability. Certain proteins called serum Cardiac markers, are released into the blood in large quantities from necrotic muscles after AMI [4]. The rate of liberation of specific proteins differs depending on their intracellular location and molecular weight and local blood and lymphatic flow. These are creatinine phosphokinase-MB isoenzyme, cardiac specific troponin T & troponin I, myoglobin. TROPONINS are now preferred biochemical markers of MI. cTnI and cTnT increases after STEMI to levels >20times higher than upper reference limit. Troponins release related to infarct size. Therefore inversely correlates with left ventricular ejection function(LVEF). LVEF is the predictor of moribidity and mortality following AMI [4]. left ventricular diastolic dysfunction has been related to development of heart failure and mortality after AMI [5]. This study is for "correlation of

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troponin T and left ventricular dysfunction after AMI in southern population of Rajasthan.

AIMS AND OBJECTIVES

To study the correlation between serum troponin T concentration and left ventricular ejection fraction after acute myocardial infarction. To study correlation between serum troponin T concentration and left ventricular diastolic dysfunction after acute myocardial infarction.

MATERIAL AND METHODS

This prospective study was conducted on patients admitted to ICCU/Cardiology ward of MBGH attached to RNT Medical College, Udaipur for a period of 6 months (July 2014 to December 2014). A total of 50 subjects were selected for the study. According to consensus document prescribed jointly by the European Society of Cardiology and American Society of Cardiology main features of revised definition of MI are [6].

Criteria For Acute, Evolving Or Recent MI

Either one of the following criteria satisfies the diagnosis for an acute, evolving or recent MI. Typical rise and gradual fall (troponin) or more rapid rise and fall (CK-MB) of biochemical markers of myocardial necrosis with atleast one of the following :

a) Ischemic symptoms
b) development of pathological Q waves on ECG reading.
c) ECG changes indicative of ischemia (ST segment elevation or depression)
d) Coronary artery intervention (e.g. Coronary angioplasty)
2) Pathological findings of an acute MI

Criteria For Established MI

Either of the following criteria satisfies the diagnosis for established MI. Development of new pathological Q waves or serial ECG reading. The patient may or may not remember previous symptoms. Biochemical markers of myocardial necrosis may have normalized, depending of the length of time that has passed since the infarct developed. Pathological finding of healed or healing MI.

Inclusion Criteria

Patients were selected on the basis of criteria 1 for acute, evolving or recent MI i.e, Typical rise and gradual fall (troponin) or more rapid rise and fall (CK-MB) of biochemical markers of myocardial necrosis with atleast one of the following :

a) Ischemic symptoms
b) Development of pathological Q waves on ECG reading.
c) ECG changes indicative of ischemia (ST segment elevation or depression)
d) Coronary artery intervention (e.g. Coronary angioplasty)

Exclusion criteria

1. Patients with significant renal impairment (serum creatinine >2.5 mg/dl)
2. Patient of Rheumatoid Arthritis
3. Past history of MI or heart failure.

Methods

All patients were subjected to detailed clinical examination. All routine and special investigations were done as per protocol. Troponin-T concentration will be measured 12-48 hrs after the onset of chest pain. Echocardiography will be performed at 4 days to 4 weeks after MI. Quantative estimation of cTnT was done by "ECLIA" method.

Standardization

According to the new definition, MI is diagnosed when blood levels of cardiac troponin are above 99th percentile of reference limit (of a healthy population) in the clinical setting of ischemia. It has been recommended that troponin concentration that meets 10% of coefficient of variation should used as a medical diagnostic guide. This is 0.03pg/L for diagnosis of AMI.

Echocardiography was obtained by using echocardiographic machine in subjects lying in the left lateral decubitus position or supine position. The echocardiographic technique and calculations of difference and cardiac dimensions will be performed according to the recommendations of the American Society of Echocardiography. The ejection fraction was obtained using the modified biplane Simpson's method from apical two chamber and four chamber view. Measurements were made from 3 consecutive beats and the average of three beats was used for analysis. LVEF less than 50% was taken as a systemic dysfunction. Doppler recording of diastolic mitral flow was obtained by using apical four chamber view.

Statistical Analysis

All data was expressed as arithmetic mean ± SD. Data were analysed using correlation, regression and student 't' test. Statistical significance was setup at $p < 0.05$

OBSERVATION

Out of 50 patients 37 (74%) were male and 13 (26%) were female. Maximum no of cases 15 (30%)were present in age group of 51-60 years followed by 26% (61-70 years) , 16% (more than 70 years) and 14% each in age group of 41-50 years and less than 40 years. Maximum no of male cases (24%) were present in age group of 51-60 years followed by 20% (61-70 years), 12% (less than 40 years), 10% (41-50 years) and 8% (more than 70 years) . maximum no of female patients (8%) were present in age group of more than 70 years followed by 6% each in age group of 51-60 years and 61-70 years, 4% (41-50 years) and 2% (less than 40 years).

Table 1 shows the various presentation of AMI. Most common presentation was chest pain 92% , followed by vomiting 32%, sweating 26% , breathlessness 20% and

palpitations 18%. Many patients had combination of symptoms at the time of presentation.

Table 2 shows the most common risk factor was dyslipidemia i.e. 31 (62%), in both male and female patient 22 (44%) and 9 (18%) respectively, followed by smoking 40% ,diabetes mellitus 28%, hypertension 26%, and family history of MD (20%). There was no risk factors in 10% of the patients.

Table 3. shows that clustering of 2 risk factors was present in maximum no. of patients 19 (38%) followed by single risk factor 14 (28%), 3 risk factors 8 (16%) and four risk factors 4(8%).

Table. 4 shows that anterior wall MI 26 (52%) was most common site of infarction followed by inferior wall MI 12(24%), inferior and RV MI 5 (10%), inferolateral MI 4(8%) , inferoposterior MI 2(4%) and posterior wall MI 1 (2%). Table 5 showing physical findings at the time of presentation, most common was hypertension 11(22%), followed by tachycardia 7 (14%), crackles and raised JVP 6 (12%) each, bradycardia 5 (10%), hypotension and S3 and S4 4 (8%) each and 1 (2%) each had systolic murmur at apex, CHB and VPC's.

Table 6 shows that there is no relation between lipid profile and AMI.

Table 7 shows that cTnT level was highest in cases of inferolateral MI (9.37 ± 7.48) followed by posterior wall MI (6.40) , inferoposterior wall MI (6.21 ± 3.49), anterior wall MI (5.96 ± 4.66), inferior and RV MI (4.28 ± 3.1) and lowest in cases of inferior wall MI (4.08 ± 3.04).

Table 8. shows that cTnT level was significantly higher in patients with LVEF <50%.

Table 1. Clinical Presentation Of AMI

Symptoms	No. of patients	%
Chest pain	46	92
Sweating	13	26
Vomiting	16	32
Breathlessness	10	20
Palpitations	9	18

Table 2. Incidence of risk factors

Risk Factor	Sex				Total	
	Male		Female			
	No.	%	No.	%	No .	%
Dyslipidemia	22	44	9	18	31	62
Hypertension	8	16	5	10	13	26
Diabetes Mellitus	10	20	4	8	14	28
Smoking	20	40	00	00	20	40
Family H/O IHD	7	14	3	6	10	20
No risk factor	4	8	1	2	5	10

Table 3. Clustering of risk factors

Number of risk factors	No. of patients	%
No risk factors	5	10
1 risk factor	14	28
2 risk factors	19	38
3 risk factors	8	16
4 risk factors	4	8
Total	50	100

Table 4. Distribution of patients according to the site of infarction.

Site	No. of patients	%
Anterior wall	26	52
Inferior wall	12	24
Posterior wall	1	2
Inferoposterior	2	4
Inferior and RV	5	10
Inferolateral	4	8
Total	50	100

Table 5. Physical findings

Physical findings	Sex				Total	
	AWMI		IWMI			
	No.	%	No.	%	No.	%
tachycardia	7	14	-	-	7	14
Bradycardia	-	-	5	10	5	10
Hypertension	7	14	4	8	11	22
Hypotension	2	4	2	4	4	8
Crackles	5	10	1	2	6	12
Raised JVP	5	10	1	2	6	12
S3/S4	4	8	-	-	4	8
Systolic murmur at apex	1	2	-	-	1	2
CHB	-	-	1	2	1	2
VPC's	1	2	-	-	1	2

Table 6. Lipid Profile Of Patients

Lipid profile mg/dl	Mean	S.D	S.E	't' value
T.cholesterol	172.34	31.02	4.39	6.30
S.triglyceride	136.14	44.48	6.29	2.20
LDL	105.24	30.945	4.38	5.65
HDL	39.24	4.18	0.59	1.29

Table 7. Serum cTnT levels in relation to site of infarction

Site	No. of patients	Troponin T (ng/ml) (mean ± SD)
Anterior wall	26	5.96±4.66
Inferior wall	12	4.08±3.04
Posterior wall	1	6.40
Inferoposterior	2	6.21±3.49
Inferior and RV	5	4.48±3.1
Inferolateral	4	9.37±7.48

Table 8. Troponin T level (mean ± SD) in relation to ejection fraction.

Ejection fraction	N	Tropinin T (ng/ml) (mean ± SD)
Less than 50%	38	6.35±4.79
More than 50%	12	3.43±1.72

Table 9. Regression coeffiecient

Y		Coefficients	Standard error	Significance
EF	a	51.473	2.904	0.00
	b	-1.735	0.406	0.001

Critical value of troponin T for EF <50% = 1.07ng/ml, 't' value of troponin T and EF = -0.439, P value = 0.001

Table 10. Troponin T levels (mean ± SD) in relation to E/A ratio

EF	N	Troponin T (ng/ml) (mean ± SD)
<1	12	6.36±7.03
>1	38	5.42±3.32

Table 11. Regression co-efficent

Y		Co-efficients	Standard error	Significance
EF	a	1.643	0.157	0.000
	b	-0.0208	0.022	0.348

Critical value of troponin T for E/A <1 = 30.913ng/ml; 't' value of troponin T and E/A = - 0.135; 'p' value = 0.348; This shows that there is no signinficant co-relation between E/A and Troponin T.

DISCUSSION

After acute myocardial infarction, patients prognosis is closely related to the extent of irreversibly damaged myocardium. In routine clinical practice, infarct size is estimated non-invasively by ECG, Imaging techniques (such as radionuclide imaging and ECHO) and serological tests.

There is increasing awareness of the limitations of standard biochemical markers of cardiac damage in patients with AMI. A desire to improve sensitivity, specificity and prognostic value has lead to the search for the markers uniquely expressed by the myocardium. The cardiac troponin T has been found to have excellent sensitivity and specificity and is superior to CK-MB as indicator of myocardial necrosis[7]. cTnT is uniquely located in the myocardium and its release closely relates to infarct size[8] therefore inversely co-relates with LVEF. We performed this study to find out the level of troponin-T after AMI and its co-relation with LVEF and LVDD. This prospective study was conducted MBGH attached to RNT medical college Udaipur , Rajasthan. A total of 50 patients of AMI admitted to ICCU were included.

Out of 50 patients of AMI, 37(74%) were male and 13 (26%) were female. Maximum no of patients were in 51-60 yrs of age group. These results were similar to the study performed by Sinha [9]. in which it was found that in india, CAD appears a decade earlier compared with the age incidence in developed countries and the peak period is attained between 51-60 years of age. In this study male were affected more than females. Mintz [10] analyzed 572 patients of AMI and ratio of male : female was 2.2:1. Comparable results were found in our study (2.85:1). Maximum no of female 7 (53.8%) were above 60 years of age. These observations were consistent with the notion that oestogen is protective and there is a dramatic rise in incidence of AMI in woman after 55 years of age [11].

Clinical presentation of AMI was quite variable. All the patients were symptomatic at the time of presentation. Most common presentation was chest pain 46(92%) followed by vomiting 16 (32%) sweating 13 (26%). Breathlessness 10 (20%) palpitations 9 (18%). Gupta [12]. performed a 5 years study of clinical profile of AMI and this study also finds most common presentation was chest pain 88%.

Most common risk factor for AMI was dyslipidemia 62% followed by smoking 40% diabetes mellitus 28% hypertension 26% and family history of IHD 20% there were no risk factors for 10% of patients. These results were little different from the study of Kulu [13] in which one third of patients of AMI had no risk factors.

Patients had clustering of risk factors in our study. Maximum no. patients had clustering of two risk factors (38%) , 16% of patients had 3 risk factors. These results substantiate the outcome of study conducted by Barette [14]. which demonstrated that many of the CAD risk factors cluster together and act synergistically.

The physical findings at the time of presentation were variable. Tachycardia was present in 14% of the cases all of them had AWMI. Bradycardia was present in 10% of the cases all of them had inferior wall MI. Out of 11 (22%) hypertension patients 7 (14%) had anterior wall MI and 4(8%) had IWMI. Hypotension was present in 4 (8%) of cases, 2 (4%) each has AWMI and IWMI, in 6 (12%) of cases crackles were present on auscultation in basal lung fields, 5 (10%) had AWMI and 1(2%) had IWMI. JVP was raised in 6(12%) patients, 5(10%) AWMI and 1(2%) had IWMI. S3/S4 sound was present in 4(18%) patients all of them had AWMI, systolic murmur at apex was heard in 1 patient of AWMI. VPC's was present in 1 patient who had AWMI. CHB was present in 1 case of IWMI. So results of our study shows that AWMI are often symptomatic.

In our study we took all the patients of antero-septal, anterolateral and extensive anterior wall MI as AWMI, while patients of IWMI, posterior wall MI, Inferoposterior MI, Innferolateral MI and Inferior and RV MI were categorized separately. AWMI 26 (52%) was most common site of infarction followed by inferior wall MI 12 (24%) , inferior and RVMI 5 (10%) , inferolateral MI 4(8%), inferoposterior MI 2(4%) and 1(2%) patient had posterior wall MI.

The serum cTnT level in cases of AWMI (5.96 ± 4.66ng/ml), inferoposterior MI (6.21± 3.49 ng/ml) , posterior wall MI (6.40 ng/ml) and inferolateral MI (9.37 ± 7.48 ng/ml) was significantly higher than serum cTnT levels in cases of inferior wall MI (4.08 ± 3.04 ng/ml). Omura [15] evaluated 34 patients of AMI doing serum cTnT measurement and measuring infarct size by resting 201 thallium myocardial single photon emission computed tomography (SPECT). It was found that extent score (ES) and severity score (SS) which were estimated for resting 201 thallium SPECT image, showed excellent linear co-relations with cTnT levels (ES:$r = 0.77$, $p < 0.001$, SSr = 0.66, $p< 0.001$). Although no such measurement was done in our study yet it can be concluded that the serum cTnT level co-relates with the infarct size.

There was a strong negative co-relation between cTnT level and LVEF. The Pearson's co-relation co-efficient between cTnT and LVEF was $r = -0.439$. The cTnT value was higher (6.35±4.79ng/ml) in patients with LVEF <50% was compared to patients with LVEF >50% (3.43±1.72ng/ml). The difference was statistically significant ($p < 0.001$). Critical value of cTnT for LVEF <50% was 1.07ng/ml. These results were similar to the results obtained in the study by Kanna et. Al and Omura [15] which showed significant negative co-relation between serum cTnT level and LVEF with ($r = -0.48$, $p <0.001$) and ($r = -0.68$, $p<0.001$) respectively. So therefore these findings, cTnT shows excellent promise as a useful marker of infarct size and the assessment of LVEF.

There was no co-relation between cTnT and E/A ratio. The Pearson's co-relation co-efficient between cTnT and E/A was $r = -0.135$. The difference was not statistically

significant (P = 0.348). No study co-relating cTnT and E/A was found using MEDLINE database.

CONCLUSION

One of the limitation of the study was that 2D Echo calculation of LVEF was done which is always smaller than those determined by LV angiography and chances of technical errors there. Secondly E/A values were not corrected for age. Hence it can be concluded that:- Serum troponin T concentration measured 6-48hours after onset of chest pain for first myocardial infarction is a reliable, simple, quick, cost effective and non-invasive method for identifying patients with LVEF < 50% for whom there is a poor prognosis.

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Nil

CONFLICT OF INTEREST

No interest

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