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FREQUENCY OF LEFT VENTRICULAR DYSFUNCTION IN PATIENTS WITH STEMI HAVING MARKEDLY RAISED TROPONIN T

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All the authors contributed significantly to the research that resulted in the submitted manuscript.

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ABSTRACT

Objective: To determine the frequency of left ventricular dysfunction in patients with STEMI having raised troponin T.

Methodology: 86 patients of acute MI with markedly raised Troponin T, admitted to the between February to July 2011 were included in the study. Patient's details, relevant investigations and results were recorded in the proforma.

Results: Majority of the Acute STEMI patients with LVD lie in [51-60 years] age group. Moreover minimum age was noted as 31 years while maximum 70. It was also found that mean age was [54.41±9.90]. 73(84.9%)were males and 13 (15.1%) were females. It was found that among total of 86 patients there were 82 patients who had LVD while 2 patients did not show LVD. Moreover it was found that 12(14%) had mild, 42(48.8%) had moderate and 28(32.6%) has severe LVD. With relation to age among total 8 (9.3%) were of age group of 31-40. LVD was mild in 1(8.3%) moderate in 3 (7.1%) and severe in 4(14.3%). While 28 (32.6%) of patients of age group of 41-50 years, LVD was mild in 1(8.3%) moderate in14(33.3%) and severe in 13(46.4%).Similarly 29 (33.7%) of patients of age group of 51-60, years LVD was mild in 3(25%) moderate in 16(38.1%) and severe in 7(25%) while 21 (24.4%) of patients of age group of 61-70, years LVD was mild in 7(58.4%) moderate in 9(21.5%) and severe in 11(25%).

Conclusion: Left ventricular dysfunction is frequently seen in STEMI patients with markedly raised Troponin –T, which serve as simple, inexpensive, quick and non-invasive method of identifying patients with a left ventricular dysfunction.

Key Words: Acute Myocardial Infarction, Troponin T, Left Ventricular Dysfunction.

INTRODUCTION

Myocardial Infarction (MI) is the part of Acute Coronary Syndrome (ACS) that includes unstable angina, non ST elevation MI (NSTEMI) and ST elevation MI (STEMI). MI is defined as "Detection of rise and/or fall of cardiac biomarkers (preferably troponin,) with at least one value above 99th percentile of upper reference limit, together at least one of following: ischemic symptoms, ECG changes indicative of new (New ST-T changes or new LBBB), development of pathological Q waves, Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality."[1]. Coronary disease is the leading killer of individuals worldwide and a leading cause of healthcare expenditure. On a global scale, ischemic heart disease kills over 6 million individuals each year and is projected by the World Health Organization to be the greatest single-disease cause of death worldwide by an increasing margin into 2030.[2]

The clinical spectrum of ischemic heart disease is diverse, ranging from silent ischemia to acute myocardial infarction (MI).[3] By definition an acute myocardial infarction (AMI) is an area of myocardial necrosis due to severe reduction or blockage of the nutrient flow.[4] Partial or complete epicardial coronary artery occlusion from plaques vulnerable to rupture or erosion is the commonest cause of myocardial infarction, accounting for around 70% of fatal events.[5] Ischemic Heart Disease (IHD) is responsible for about three fourth of all cases of cardiac failure (pump failure), that is a clinical syndrome in which an abnormality of cardiac structure or function is responsible for the inability of the heart to eject or fill with blood at a rate commensurate with the requirements of the metabolizing tissues.[6] AMI has both immediate and delayed effects on the ventricle: a healthy ventricle may become severely dysfunctional almost instantaneously due to massive myocardial ischemia and subsequent necrosis. In a few patients, acute ventricular septal defect or papillary muscle dysfunction or rupture may lead to heart failure.[7]

In the US National Registry of Myocardial Infarction (NRMI) database, approximately 20% of those with AMI had heart failure at the time of hospital admission and approximately 9% developed heart failure thereafter.[7,8] In a French study, 38% of patients exhibited heart failure during the first five days after MI.[7,9]

The underlying cause for most patients developing heart failure after MI is a moderate amount of myocardial necrosis with consequent ventricular remodeling.[10] Ventricular remodeling consists of left ventricular wall thinning in the infarct area, ventricular chamber dilatation, and compensatory hypertrophy via lengthening of the non-infarcted portion of the myocardium. Remodeling initially maintains stroke volume and pump function of the left ventricle but over time these changes become maladaptive, leading to increased wall stress and oxygen demand, interstitial fibrosis, decreased contractility and a vicious downward spiral to heart failure.[11] 30-day risk of death increased in proportion to the number of cardiac biomarkers elevated at baseline with a near doubling of the mortality risk for each additional biomarker that was elevated, similar relationships existed for the endpoints of MI, CHF, and the composite of both.[12]

The cardiac Troponin I (cTnI) has been found to have excellent sensitivity and specificity and is superior to creatine kinase–MB (CK-MB) as indicator of myocardial necrosis. Troponin is a globular protein of muscle that binds to tropomyosin and has a marked affinity for calcium ions, and is thus a central regulatory protein of muscle contraction.[13]After acute myocardial infarction (AMI), a patient's prognosis is closely related to the extent of irreversibly damaged myocardium[13,14,15] Cardiac troponins I (cTnI) is uniquely located in the myocardium[13] and its release closely relates to infarct size[13,14]and therefore inversely correlates with left ventricular ejection fraction, as there is inverse relation between infarct size and left ventricular ejection fraction.[16]There is a correlation between detectable cTnT and progressive decline in ejection fraction over time, in patients with detectable levels of cTnT, 44% had a decrease in LVEF on follow-up echocardiography, compared with only I8 % of patients with undetectable cTnT levels ($P<0.01$).[17]

Left ventricular function is the best individual predictor of mortality after acute myocardial infarction[18, 19].It was demonstrated that an angiographically determined left ventricular ejection fraction (LVEF) < 40 % could be identified by a single cardiac troponin T (cTnT) measurement at the diagnostically efficient time point of 12–24 hours from admission and later a larger prospective study performed and compared the measurement of cTnT and peak CK with early estimation of LVEF by echocardiography.[21]

METHODOLOGY

It is a Cross Sectional Study done at , Cardiology department, which is tertiary care ,teaching hospital of Interventional Cardiology. The duration of the study was 6 months. The sample was collected by Purposive non probability Technique. All patients of either gender and of age 18 years or above presenting with first episode of Acute STEMI having markedly raised Troponin T, measured between 12 and 48 hours after onset of chest pain. While patients with Past history of heart failure, hypertension, Ischemic Heart Disease, Valvular lesions, Cardiac intervention, Diabetes mellitus, Renal failure, Liver failure, Anemia , Thyrotoxicosis were excluded. Proper permission is taken from Institutional Ethical Committee to conduct this study . Eight six patients of acute STEMI, presenting in emergency department, fulfilling the inclusion criteria were

selected. Informed consent was taken from the subjects describing the procedure of study, ensuring confidentiality and explaining that there is no risk involved to the patients by taking part in this study .Serum Troponin T level was measured by consultant pathologist in the hospital pathology laboratory by using Cardiac reader Rosh(Cobas H-32) appratus. Echocardiography is done on the third day of acute MI to assess the Left Ventricular Dysfunction and patients were categorized in to four data sets ,as no Left Ventricular Dysfunction, mild ,moderate and severe Left Ventricular Dysfunction. Echocardiography is done by the consultant cardiologist who is FCPS Cardiology and has 5 years of postgraduate experience of clinical echocardiography as a consultant .It is done by using GE-vivid 7 machines. Confounding variables like age and gender were controlled by making stratified cross matching tables. Data is entered and analyzed using SPSS version 10.0 (a statistical software). Numerical variable is age and it is calculated by mean and standard deviation. Confounding variables like age and gender were controlled by making stratified cross matching tables. Frequency and percentages calculated for categorical variables like sex, raised troponin and Left Ventricular Dysfunction.

RESULTS

The results showed that most of the patients lie in [51-60] age group. Thus majority of the AMI patients with LVD patients have 51-60 years of ages. Moreover minimum age was noted as 31 years while maximum 70. It was also found that mean age was 54.41 years with standard deviation of 9.90 i.e. [54.41±9.90]. Among the total 86 patients there were 82 (95.4%) patients who had LVD while 4(4.6%) patients did not show LVD . Among the 82(95.4%) 14% shows mild LVD, 48.8% showed moderate and 32.6% showed severe LVD. Among 8 (9.3%) of patients of age group of 31-40 years, LVD was mild in 1(8.3%) moderate in 3 (7.1%) and severe in 4(14.3%). 28 (32.6%) of patients of age group of 41-50 years, LVD was mild in 1(8.3%) moderate in14(33.3%) and severe in 13(46.4%).29 (33.7%) of patients of age group of 51-60 years, LVD was mild in 3(25%) moderate in 16(38.1%) and severe in 7(25%). 21 (24.4%) of patients of age group of 61-70 years, LVD was mild in 7(58.4%) moderate in 9(21.5%) and severe in 11(25%). Results also indicated that among total 73 male patients LVD was absent in 3(75%) mild in 10(83.3%) moderate in 37(88.1%) and severe in 23(82.1%).Compared

Table 1: Age Distribution of Patients with Myocardial Infarction (n)

Age (in years)	No. of Patients	Percentage (%)
31 — 40	8	9.3
41— 50	28	32.6
51— 60	29	33.7
61— 70	21	24.4
Total	86	100.0

Mean age±SD=54.41±9.90 years.

Table 2: Left Ventricular Dysfunction in Patients with Myocardial Infarction (n)

LVD	No. of Patients	Percentage (%)
Present	82	95.4
Absent	4	4.6
Total	86	100.0

Key: *LVD=Left Ventricular Dysfunction*

Table 3: Severity of Disease in Patients with Myocardial Infarction (n)

Severity	No. of Patients	Percentage (%)
Mild	12	14.0
Moderate	42	48.8
Severe	28	32.6
No dysfunction	4	4.6

Table 4: Age Distribution of Patients with Myocardial Infarction in Relation to Severity of Left Ventricular Dysfunction

Age (in years)	No. of Patients			
	Mild	Moderate	Severe	No dysfunction
31— 40	1(8.3%)	3(7.1%)	4(14.3%)	-
41— 50	1(8.3%)	14(33.3%)	13(46.4%)	-
51— 60	3(25%)	16(38.1%)	7(25%)	3(75%)
61— 70	7(58.4%)	9(21.5%)	4(14.3%)	1(25%)
Total	12	42	28	4

Table 5: Gender Distribution of Patients with Myocardial Infarction in Relation to Severity of Left Ventricular Dysfunction

Sex	No. of Patients			
	Mild	Moderate	Severe	No dysfunction
Male	10(83.3%)	37(88.1%)	23(82.1%)	3(75%)
Female	2(16.7%)	5(11.9%)	5(17.9%)	1(25%)
Total	12	42	28	4

to it among total 13 female patients LVD was absent in 1(25%) mild in 2(16.7%) moderate in 5(11.9%) and severe in 5(17.9%).

DISCUSSION

Ischemic Heart Disease (IHD) is responsible for about three fourth of all cases of cardiac failure (pump failure), [6] and left ventricular dysfunction is the single most common cause of heart failure after myocardial infarction [7] LV function and dilation after MI have been extensively studied and have been related to heart failure and cardiac mortality.[22] After acute myocardial infarction (AMI), a patient's prognosis is closely related to the extent of irreversibly damaged myocardium. Recently, serum troponin T has emerged as a specific indicator of myocardial damage in acute myocardial infarction. [19] cTnI is uniquely located in the myocardium and its release closely relates to infarct size; therefore, inversely correlates with left ventricular ejection fraction (LVEF).[13,19]

This marker offers a simple, inexpensive, quick, non-invasive method of identifying patients with a left ventricular ejection fraction of < 40%. Estimation of troponin T can also be used to identify those patients who may benefit from other treatments—for example, ACE inhibitors and ARBs.

Results of our study indicated that among total of 86 patients there were 82 (95.4%) patients who had LVD while 4(4.6%) patients did not show LVD. The figure of 95.4 % is comparable with figure of 75%- 100% in the past studies.[13, 19-25] Our study also assessed the severity of LVD in patients with markedly raised troponin T suffering from acute STEMI. It showed that 14(14%) had mild, 42(48.8%) had moderate and 28(32.6%) had severe LVD. Again it is comparable with 15.4 to 67.6%.LVEF related with raised troponin T in past sudies[19,26-28] In our study most of the patients had LVEF in the range of 30 to 40% comparable with the mean LVEF of 37.6% in one study[29], <50% in another study [13]and < 40% in most of other studies. [19-21, 25-28]

The number of patients studied in past were in the range of 20-201 in most of the studies.[13,19-21,24, 26,28-30] only two studies included large proportion of patients like 378 in one[25] and a much higher number of 2457 in another study.[27] Although even with such a higher number of patients in few past studies results of our study are comparable with them.

Some of the studies in past studied association of LVD with Troponin I, after Myocardial infarction[13,20-25,28,] while other studied with Troponin T[19,26,27,30] We studied frequency of LVD in patients with raised Troponin T. The troponins T, and I both are subunits of the thin filament-associated troponin-tropomyosin complex, which is involved in regulating striated muscle contraction.[3,31,32,] The release kinetics of troponins T and I are similar; both are detectable in the serum within 4 to 12 h after the onset of myocardial necrosis and depending on the duration of ischemia and reperfusion status, peak values occur 12 to 48 h from symptom onset.[3] That is why it should not be the cause of any difference of our study from the previous studies.

Although the results of our study are comparable with the past studies but there were some differences in the our study method and previous studies. We studied frequency of LVD

in patients with markedly raised Troponin T in patients suffering from acute myocardial infarction and LVD was assessed by echocardiography, while in some previous studies different imaging techniques like SPECT[24,26] and MRI[30] were used.

The other difference was that we studied frequency of LVD purely related with troponin T concentration after MI regardless of the patients undergoing any specific treatment like reperfusion or PCI while few past studies gave attention to the relation of troponin with LVD with regard to the patients undergoing PCI[20,25,28] and reperfusion therapy. [20,25,30] Although there is evidence that unlike creatine kinase and myoglobin, Troponin T concentration is unaffected by thrombolysis after the first 12 hours, following which it shows a stable plateau for about 48 hours.[19]

The other difference of note is that few studies in past also looked at the time relation of Troponin concentration and LVD after MI.[26,28,30] while we included patients with markedly raised troponin T measured between 12-48 hours after onset of chest pain and followed them up only during the duration of there current admission. It was not followed up later to see the relation between the troponin T concentration and any improvement or progression of the LVD with the passage of time .

There were few limitations to our study as because of our setup it is very difficult to follow the patients for long duration like few months. Some patients do not turn up for the follow up and some feel as if they are treated forever. It can be achieved by improving patient education and self care.

This result of our study showed significant relation of markedly raised Troponin T and LVD in patients suffering from acute STEMI. This marker can offer a simple, inexpensive, quick, non-invasive method of identifying patients with a left ventricular dysfunction who may benefit from other treatments—for example, ACE inhibitors.Further studies focusing on follow up of these patients can give a more better insight into the long-term relation of Troponin T concentration with the LVD in patients of acute MI and to improve treatment plans further.

It is advisable that building on disease management models that have been successful in patients with chronic heart failure , clinicians should ensure that patients who have had a myocardial infarction receive the right medications, comprehensive self-care education to decrease the risk for new cardiac events, and an appointment for follow-up after discharge .It is crucial to organize hospital care structures to ensure that all cases of LVD after myocardial infarction are detected , managed and educated appropriately.

CONCLUSION

Regardless of age and gender Left ventricular dysfunction is frequently seen in patients of acute STEMI with markedly raised Troponin –T. Thus this marker can offer a simple, inexpensive, quick, non-invasive method of identifying patients with a left ventricular dysfunction.

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