Impact of Left Ventricular Hypertrophy on Peak Serum Troponin T Levels in Patients With Acute Myocardial Infarction

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Previous studies have reported that peak serum troponin I levels were disproportionately elevated in patients with acute anterior ST-segment elevation myocardial infarction (STEMI) and left ventricular (LV) hypertrophy (LVH) compared with those with normal LV mass. The purpose of this retrospective study was to assess the relation of peak serum troponin T levels in patients with normal LV mass and in subjects with mild, moderate, and severe LVH in patients with acute STEMI or non-ST segment elevation myocardial infarction (NSTEMI) when stratified on variables that might be expected to affect serum troponin T levels. The study population consisted of 262 patients; 91 with STEMI and 161 with NSTEMI. Serum troponin levels and 2-dimensional echocardiograms were obtained within the first 24 hours of hospitalization for STEMI or NSTEMI. There was no significant difference in serum troponin T levels in LV mass and/or LVH groups ($p = 0.3210$). There was no significant difference in serum troponin T levels in LV mass and/or LVH groups when these data were stratified on third variables including serum creatinine >1.2 mg/dl ($p = 0.3681$), LV ejection fraction <60% ($p = 0.0978$), STEMI ($p = 0.2576$), NSTEMI ($p = 0.4994$), and location of severe coronary stenosis ($p = 0.1981$). The results of this study suggest that there is no association between peak serum troponin T levels and LV mass and/or LVH groups when such groups are stratified on a third variable that may influence peak serum troponin T levels. **(Am J Cardiol 2019;123:1745−1750)**

Cardiac troponins are sensitive and specific markers of cardiomyocyte injury and death.[1−7] Cardiac troponins (I and T) have become the diagnostic standard for confirmation of acute myocardial infarction (AMI) when used in conjunction with clinical manifestations and electrocardiographic abnormalities.[1−7] Peak serum troponin concentrations correlate strongly with infarct size and left ventricular (LV) systolic dysfunction.[1−7] High serum cardiac troponin levels have been associated with a poor prognosis in patients with AMI.[1−7] Previous studies suggested that peak cardiac troponin I levels overestimated infarct size in an ovine model of LV hypertrophy (LVH) and in humans with LVH.[8−11] These findings were attributed to increased troponin I content in individual hypertrophied cardiomyocytes.[9] This study assessed the relation of cardiac troponin T to LV mass in patients with AMI by comparing peak serum troponin T levels in patients with normal LV mass with those in patients with various degrees of severity of LVH.

Methods

This was a retrospective study. The medical records of consecutive patients with ST segment elevation myocardial infarction (STEMI) or non-ST segment elevation myocardial infarction (NSTEMI) who were referred for coronary angiography between January 1, 2015 and December 31, 2016 were reviewed. Standard 12 lead electrocardiograms were obtained on all patients during the index hospitalization. To be included in the study, patients were required to have undergone transthoracic 2-dimensional echocardiography during their hospitalization for AMI. Patients were excluded if serial serum troponin T levels were not obtained or if transthoracic echocardiography was not performed during the index hospitalization. Patients with hypertrophic obstructive cardiomyopathy or evidence of acute myocarditis were also excluded.

Each patient underwent left coronary angiography using the Judkins technique or through radial artery access. Coronary artery stenoses were classified as those located in the major coronary arteries or their branches. Significant coronary stenosis was defined as >70% stenosis in 1 or more of the major coronary arteries or their branches (>50% for left main stenosis). The degree of stenosis was assessed visually by 2 investigators (JP and SK). Initial disagreements were resolved by consensus. A minimum of 3 serum troponin T levels were obtained during the first 24 hours of hospitalization. A rise or decline in serum troponin T concentrations was considered to be suggestive of AMI. The normal serum troponin T levels in our laboratory is <0.01 ng/ml. Each patient received at least 1 12 lead electrocardiogram during the first 24 hours of hospitalization. Twelve lead electrocardiograms were performed using Mortara ELI 250 or 280 electrocardiographs with a gain setting of 10 mm/mV and a paper speed of 25 mm/sec. Two-dimensional echocardiograms were performed using a General Electric Vivid 9 echocardiograph

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in accordance with American Society of Echocardiography guidelines.[11] Left ventricular mass was calculated using the method of Devereux et al.[11,12] LV mass was classified as being normal or increased. Increased LV mass is heretofore referred as LVH. LV mass classifications were as follows: normal LV mass (96 to 200 g), mild LVH (201 to 227 g), moderate LVH (228 to 254 g), and severe LVH (>254 g).[11] LV ejection fraction (LVEF %) was measured by 2-dimensional echocardiography in accordance with American Society of Echocardiology guidelines. Renal dysfunction was defined as serum creatinine >1.2 mg/dl.

Statistical analysis was performed using SAS version 9 software (SAS Institute Inc., Cary, North Carolina). Categorical variables were summarized using frequency count (numbers of patients) and the percentage of total patients or patients in individual groups. Continuous variables were summarized using mean values ±1 SD. When a standard Gaussian distribution could not be assigned from the data, nonparametric statistics were used to compare mean values across strata. The Cochran-Mantel-Haenszel test (which takes the ordered nature of categories into account) was used to determine if significant differences existed in groups when stratifying on a third variable. The Kruskal-Wallis test (which does not take the ordered nature of categories into account) served as a 1-way analysis of variance to determine if mean values of continuous variables differed in groups. The Dwass, Steel, Fligner-Critchlow method was used for pairwise 2-sided comparisons between groups if the Cochran- Mantel-Haenszel or the Kruskal-Wallis tests identified significant differences in groups. Spearman rank correlations were calculated to determine the relation of troponin T values to specific continuous variables (LV mass, serum creatinine, and LVEF). Receiver operating characteristic curves were generated from mean serum troponin T levels collected on admission, 6 hours after admission, and 12 to 18 hours after admission for patients with and without LVH and for each of the 4 LV mass and/or LVH groups. Student's 2-tailed *t* Test for unpaired data was used to compare total areas under the curve (AUC) for patients with and without LVH. A 1-way analysis of variance was applied to determine if significant differences existed across strata for total AUC for the 4 LV mass and/or LVH groups. A p value <0.05 was required for statistical significance.

This study was conducted in accordance with precepts the Declaration of Helsinki and was approved by the University of Missouri Institutional Review Board.

Results

A total of 836 patients underwent coronary angiography for STEMI or NSTEMI between January 1, 2015 and December 31, 2016. Of those, 262 patients fulfilled entry criteria with regard to troponin T collection and performance of transthoracic echocardiography. STEMI occurred in 91 patients and NSTEMI occurred in 171 patients. Patient characteristics in subjects with normal LV mass and in those with LVH are shown in Table 1.

Mean, values, and SDs for peak serum troponin T levels in patients with normal LV mass and in those with mild,

Table 1
Patient characteristics

Patient characteristics	All patients n = 262	Left ventricular hypertrophy			
		None n = 138	Mild n = 28	Moderate n = 37	Severe n = 59
Men	171 (65%)	77 (56%)	21 (75%)	23 (62%)	50 (85%)
Women	91 (35%)	61 (44%)	7 (25%)	14 (38%)	9 (15%)
Mean age (years)	63 ± 13	63 ± 14	61 ± 14	63 ± 12	65 ± 12
STEMI	91 (65%)	53 (38%)	10 (36%)	18 (49%)	10 (17%)
NSTEMI	171 (65%)	85 (62%)	18 (64%)	19 (51%)	49 (83%)
PCI ≤24 hours	128 (49%)	71 (51%)	17 (61%)	17 (46%)	23 (39%)
LVEF ≥60%	170 (65%)	95 (69%)	19 (68%)	22 (59%)	34 (58%)
LVEF <60%	92 (35%)	43 (31%)	9 (32%)	15 (41%)	25 (42%)
Serum creatinine >1.2 mg/dl	78 (30%)	34 (25%)	6 (21%)	9 (24%)	29 (49%)
Serum creatinine ≤1.2 mg/dl	184 (70%)	104 (75%)	22 (79%)	28 (76%)	30 (51%)
Coronary artery stenosis >70%					
Left anterior descending	68 (26%)	34 (25%)	9 (32%)	10 (27%)	15 (25%)
Left circumflex	42 (16%)	20 (14%)	6 (21%)	6 (16%)	10 (17%)
Right	53 (20%)	33 (24%)	6 (21%)	8 (22%)	6 (10%)
Ramus intermedius	4 (2%)	2 (1%)	1 (4%)	1 (3%)	0 (0%)
>1 coronary artery	40 (15%)	18 (13%)	4 (14%)	4 (11%)	14 (24%)
Coronary artery stenosis <70%	54 (21%)	32 (23%)	3 (11%)	6 (16%)	13 (22%)
History of hypertension	169 (65%)	78 (57%)	19 (68%)	25 (68%)	47 (80%)

LV = left ventricular; LVEF = left ventricular ejection fraction; LVH = left ventricular hypertrophy; NSTEMI = non-ST segment elevation myocardial infarction; PCI ≤24 hours = percutaneous coronary intervention performed during first the 24 hours of hospitalization; STEMI = ST segment elevation myocardial infarction.

Categorical data are expressed as number of patients and percentage in each group (in parentheses). Age expressed as mean value ± 1 SDs.

moderate, and severe LVH are shown in Table 2. Using both the Cochran-Mantel-Haenszel and Kruskal-Wallis tests, we found no significant differences in mean serum troponin T values in the 4 groups. Pairwise analysis showed no significant differences in peak serum troponin T level in any of the paired comparisons. The Spearman rank correlation coefficient (r_s) and related p value which assessed the relation of ungrouped LV mass to peak serum troponin T level were as follows: $r_s = 0.0168$, $p = 0.787$. There was no significant difference in the frequency of percutaneous coronary intervention in LV mass and/or LVH groups ($p = 0.6901$, Table 1).

Table 2 shows the mean values and SDs for serum creatinine and peak serum troponin in the normal LV mass group and in the mild, moderate, and severe LVH groups. The Cochran-Mantel-Haenszel test, using renal dysfunction (serum creatinine >1.2 mg/dl) as a stratifying variable, showed no significant association between peak serum troponin level and LV mass and/or LVH groups. The absence of an association occurred although that the Kruskal-Wallis test showed significant differences in mean serum creatinine values in LV mass and/or LVH groups ($p = 0.0012$). Pairwise analysis of LV mass and/or LVH groups showed that the comparison of mean values of serum creatinine in the normal LV mass and severe LVH groups were significantly different from each other. Spearman rank correlation coefficients and were assessed using ungrouped data. There was no significant association between serum creatinine and peak serum troponin T levels ($r_s = 0.0521$, $p = 0.4011$). There was significant correlation between serum creatinine and LV mass ($r_s = 0.2347$, $p < 0.0004$).

LVEF was <60% in 92 patients and ≥60% in 170 patients. Mean values and SDs in the normal LV mass group and in the mild, moderate, and severe LVH groups are shown in Table 2. The Cochran-Mantel-Haenszel test using LVEF <60% as a stratifying variable showed no significant association between peak serum troponin T levels and LV mass and/or LVH groups ($p = 0.0978$). The absence of an association occurred although that the Kruskal-Wallis test suggested that significant differences in mean LVEF values may exist in LV mass and/or LVH groups ($p = 0.0256$). However, pairwise comparisons using the Dwass, Steel, Critchlow-Fligner method showed no significant differences between specific LV mass and/or LVH groups. There were however, significant negative correlations between LVEF and both ungrouped peak serum troponin T levels and ungrouped LV mass. The Spearman correlation coefficients and associated p values were as follows: ($r_s = -0.2034$, $p = 0.009$) for peak serum troponin T; $r_s = -0.1284$, $p = 0.0378$ for LV mass.

Of the 262 patients entered into the study, 91 had an STEMI and 171 had a NSTEMI. Mean values and SDs in the normal LV mass and mild, moderate, and severe LVH groups are shown in Table 2. The Cochran-Mantel-Haenszel test, using STEMI and NSTEMI individually as stratifying variables, showed that peak serum troponin T levels were significantly greater with STEMI than with NSTEMI in patients with normal LV mass and in each of the LVH groups ($p < 0.0001$). The Cochran-Mantel-Haenszel test was also used to determine if there was a relation between peak serum troponin T levels and LV mass and/or LVH groups in either STEMI or NSTEMI patients. There was no significant relation between troponin T levels and LV mass and/or LVH groups for either STEMI or NSTEMI. Spearman rank correlations did not differ from zero for either STEMI ($r_s = 0.0716$, $p = 0.4997$) or NSTEMI ($r_s = 0.05363$, $p = 0.4699$).

Table 3 shows the relation of peak serum troponin T levels to the location of severe coronary stenoses which, in the angiographer's judgment were the culprit lesion(s) causing AMI. Also shown are peak serum troponin T levels in patients with >1 severely stenotic coronary artery and in those with coronary stenosis <70%. The Kruskal-Wallis test showed that there was a significant difference in peak troponin level in LV mass and/or LVH groups. Pairwise comparison using the Dwass, Steel, Critchlow-Fligner method indicated that peak serum troponin T levels were significantly lower in patients with <70% stenosis compared with those with ≥70% stenosis in the left anterior descending coronary artery ($p < 0.0001$), left circumflex coronary artery ($p < 0.0001$), right coronary artery ($p < 0.0001$), or >1 severely stenotic coronary artery ($p < 0.0001$). As there were only 4 patients with >70% stenosis in the ramus intermedius

Table 2
Mean values for peak serum troponin T, serum creatinine and LVEF in normal LV Mass and LVH group

Variable	Left ventricular hypertrophy				
	None n = 138	Mild n = 28	Moderate n = 38	Severe n = 59	p
Peak serum troponin T (ng/ml)					
All patients	2.5 ± 3.9	5.6 ± 10.0	2.9 ± 3.0	1.8 ± 0.7	0.3210* 0.0843†
STEMI	4.4 ± 4.7	9.9 ± 11.5	4.6 ± 3.6	4.4 ± 3.5	0.2576*
NSTEMI	1.3 ± 2.8	3.3 ± 8.6	1.3 ± 1.7	1.3 ± 2.1	0.4994*
Serum creatinine (mg/dl)	1.1 ± 0.9	1.2 ± 1.3	1.2 ± 1.0	2.1 ± 2.7‡	0.3681*
LVEF (%)	48 ± 13	44 ± 12	50 ± 12	44 ± 14	0.0978*

LV = left ventricular; LVEF = left ventricular ejection fraction; NSTEMI = non-ST segment elevation myocardial infarction; STEMI = ST segment elevation myocardial infarction.

* Cochran-Mantel-Haenszel test.

† Kruskal-Wallis test.

‡ $p = 0.0008$ compared with serum creatinine in normal LV mass group.

Table 3
Peak serum troponin T levels based on coronary artery location(s) and severity of stenosis

Severely stenotic coronary artery	N	Mean peak serum troponin T level (ng/ml)
Left anterior descending	68	4.1 ± 5.8*
Left circumflex coronary artery	42	3.1 ± 4.9*
Right	53	2.6 ± 3.0*
>1 coronary artery	41	2.9 ± 6.5†
Coronary artery stenosis <70% (referent)	54	0.5 ± 0.8

* $p < 0.0001$ compared with referent.
† $p = 0.005$ compared with referent. Peak serum troponin T levels are expressed as mean values ± 1 standard deviation.

branch, these data were excluded. Using the Cochran-Mantel-Haenszel test to stratify for coronary stenosis <70%, we found no significant association between peak serum troponin T level and LV mass and/or LVH groups ($p = 0.1981$) based on the location of severely stenotic lesions.

Table 4 shows mean peak serum troponin values in the LV mass and/or LVH groups when stratified against individual coronary artery groups based on location and severity of stenosis. No significant differences were noted in peak serum troponin levels in the 4 LV mass and/or LVH groups for any coronary artery category.

Respective mean total AUC (±1 standard error of the mean) for patients with normal LV mass and those with LVH were 246 ± 19 and 218 ± 20 ng/ml ($p = 0.907$). Mean total AUC (±1 standard error of the mean) for the 4 LV mass and/or LVH groups were as follows: normal LV mass (246 ± 19 ng/ml), mild LVH (70 ± 16 ng/ml), moderate LVH (72 ± 8 ng/ml), and severe LVH (74 ± 7 ng/ml). After adjusting for sample size no significance and differences existed across strata ($p = 0.255$).

Discussion

LVH is among the most common causes of cardiac troponin elevation not due to AMI.[13–17] Speculation exists that elevated serum troponin levels in such patients may be due in part by increased troponin content in hypertrophied cardiomyocytes.[16,17] It has been postulated that mechanical stress due to beta adrenergic stimulation and conditions such as tachycardia may alter membrane permeability, thus facilitating troponin leak from cardiomyocytes.[13–17] The combination of increased troponin content in and troponin leak from hypertrophied cardiomyocytes has led to concern that LVH may contribute to overestimation of infarct size in patients with AMI.[8–10]

Despite this concern, relatively little information exists concerning the relation of LV mass to cardiac troponin levels in patients with AMI due to coronary atherosclerosis. In 2012, Fernandez-Jiminez et al reported the results of a retrospective study of troponin I and creatine kinase (CK) release in 504 patients with STEMI (anterior wall in 47%) with and without LVH assessed by transthoracic echocardiography.[8] Biomarkers were assessed in patients without LVH and in patients with mild and moderate and/or severe LVH.[8] Mean serum troponin levels were significantly greater in patients with mild LVH ($p = 0.015$) and in subjects with moderate and/or severe LVH ($p = 0.014$) than in those without LVH.[8] In contrast, there were no significant differences in CK levels in LV mass groups. Fernandez-Jimenez et al subsequently studied 140 patients with their first acute anterior STEMI to determine the impact of LVH on CK and troponin release during AMI. Total CK and cardiac troponin I concentrations were monitored during the 72-hour period following AMI. Cardiac magnetic resonance imaging (MRI) was performed 7 days and 6 months after infarction. In this study, LVH was associated with significantly a higher peak area under the curve for troponin I concentrations, but was not associated with differences in total CK concentration. In a companion translational study, these investigators generated acute STEMI in 10 pigs with induced LVH and in 8 sham-operated pigs. Biomarker analysis and its relation to LVH were similar to that observed in the clinical study. Immunofluorescence analysis showed significantly higher troponin I content in hypertrophied cardiomyocytes than in nonhypertrophied cardiomyocytes. In a study of 100 patients with first anterior STEMI and an occluded left anterior descending coronary artery. Daaboul et al reported that in a multivariate analysis, LV mass (determined using cardiac MRI) remained independently associated with biomarker measures of infarct size.

Table 4
Peak serum troponin levels in patients with and without left ventricular hypertrophy based on coronary artery location and degree of stenosis

Coronary artery and degree of stenosis	Left ventricular hypertrophy					
	N	None	Mild	Moderate	Severe	p
Left anterior descending ≥70%	68	3.31 ± 4.34	8.40 ± 2.00	4.32 ± 4.31	3.49 ± 3.25	0.2375
Left circumflex ≥70%	42	4.33 ± 6.42	2.58 ± 2.41	1.75 ± 1.07	1.92 ± 3.17	0.4864
Right ≥70%	53	2.60 ± 2.99	2.68 ± 4.25	4.00 ± 2.94	1.79 ± 0.81	0.8120
>1 coronary artery ≥70%	41	2.00 ± 3.20	12.52 ± 18.27	2.45 ± 2.00	1.58 ± 2.21	0.1836
Coronary artery stenosis <70%	54	0.64 ± 0.99	0.22 ± 0.16	0.23 ± 0.38	0.47 ± 0.63	0.2133

Data are expressed as mean values ± 1 standard deviation.

Our study assessed the relation of serum troponin T levels to LV mass in patients with normal LV mass and subjects with mild, moderate, and severe LVH who presented with acute STEMI or NSTEMI. The major finding in this study was that there was no association between LV mass and serum troponin T levels. These results differ from previous studies which showed that increased LV mass was associated with higher serum troponin I levels in patients with anterior STEMI. Our study differs from previous studies assessing the relation of LV mass to serum troponin levels in several ways that may help to explain this disparity. The present study was designed to assess the relation of LV mass to serum troponin T levels in AMI. Previous studies were designed primarily to assess the relation of LV mass to LV infarct size as determined by cardiac troponin and CK-MB levels, and by cardiac MRI. Our study measured serum troponin T levels, whereas the other studies measured serum troponin I levels. In the present study, the relation of LV mass groups to serum troponin levels was stratified against variables that could potentially influence serum levels of troponin T including impaired renal function, LVEF, type of AMI (STEMI or NSTEMI), presence or absence of severe coronary artery disease, and location of the severely-stenotic coronary artery or arteries.[18–22] Previous studies confined patient selection to acute anterior STEMI involving the left anterior descending coronary artery in most and perhaps all cases. In the previous clinical studies assessing the influence of LV mass on serum troponin levels, the variables utilized in multiple regression and multivariate analyses included some that might influence cardiac troponin levels, but others that were not likely to do so. Our study was not designed to assess infarct size. However, the lack of a significant difference between mean total AUC for patients with and without LVH suggests that in this study, LVH did not produce overestimation of infarct size based on serum troponin T levels. This observation is further supported by the lack of significant differences in adjusted mean total AUC in the 4 LV mass and/or LVH groups.

Major limitations of this study included the small sample size in subgroups and variability in the timing of serum troponin T collections during the first 24 hours.

In conclusion, this study suggests that there is no association between peak serum troponin T levels normal LV mass or mild, moderate, or severe LVH when serum troponin T levels and LV mass and/or LVH categories are stratified on a third variable that may influence peak serum troponin T levels.

Disclosures

The authors have no conflicts of interest to disclose.

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