Section: Cardiology

Original Research Article

MECHANICAL LEFT ATRIAL DYSFUNCTION IN ST-ELEVATION MYOCARDIAL INFARCTION PATIENTS AND THEIR ANGIOGRAPHIC CORRELATION

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Abstract

Background: The left atrium (LA) plays a critical role in cardiac performance through its reservoir, conduit, and booster pump functions. After STEMI, LA dysfunction may arise from reduced left ventricular (LV) systolic function, elevated LA pressure, or ischemic involvement through the left circumflex artery. **Materials and Methods:** This study assessed LA function in acute STEMI patients and compared it with healthy controls. Conducted at SMS Medical College, it included 25 STEMI patients (outside the revascularization window) and 25 age- and risk factor-matched controls. ECG, 2D echocardiography, and coronary angiography (in 21 cases) were performed. LA phasic volumes (LAmax, LApre-A, and LAmin) were measured, and functional indices were calculated: LA ejection fraction (LAEF%), Expansion Index, Passive EF, Active EF, LAFI, and LA ejection force. **Result:** Results revealed significantly reduced LAEF% in STEMI cases compared to controls (40.78 ± 13.39 vs. 51.19 ± 10.89, P < 0.01). LAEF% correlated positively with LVEF (r = 0.551, P = 0.004) and LAFI, while negatively correlating with volume indices (LAVImax, LAVIpre-A, LAVImin). Among STEMI subtypes, inferior wall myocardial infarction (IWMI) patients had a higher LAEF% (46.14 ± 8.34) compared to anterior wall myocardial infarction (AWMI) patients (34.22 ± 19.02), though this was not statistically significant (P = 0.24). LVEF was significantly higher in IWMI patients (47.07 ± 1.68) compared to AWMI patients (36.73 ± 7.14, P = 0.001). LA reservoir, conduit, and booster functions were generally better in IWMI compared to AWMI. LAFI was significantly reduced in STEMI patients, showing a strong correlation with LAEF%. LAVImin appeared more reflective of LA dysfunction than LAVImax. **Conclusion:** LA functions are affected in patient with STEMI though the type of MI or artery involved bears no relation with LA function. All components of LA function are affected in STEMI patient but reservoir function is most important amongst them.

INTRODUCTION

The left atrium (LA) provides several functions that influence the overall cardiac performance.

During left ventricular (LV) systole, the LA serves as a reservoir storing pulmonary venous blood that passively empties into the LV after the mitral valve opening- RESERVOIR FUNCTION. This reservoir function permits continuous flow of blood from pulmonary circulation to left heart and thus protect against raised PCWP and pulmonary congestion

Throughout the diastasis, the LA acts as a transit chamber permitting passive flow from the pulmonary veins into the LV- CONDUIT FUNCTION. The blood flow during the early filling phase is constituted by two components first being the stored blood in left atrium during ventricular systole and the other component is the passive transit of the blood from pulmonary veins to left atrium to ventricle.

Finally, the LA is a contractile chamber that actively contributes to the final LV end-diastolic pressure (LVEDP) and volume- BOOSTER PUMP FUNCTION. Loss of this function has been linked to the fall in cardiac output and development of heart failure in patients with AF.[1]

Studies done previously have theorized that as the diastolic dysfunction ensues early filling phase contribution decreases and systolic pump function of left atrium starts compensating for the same but beyond a certain point the booster pump function

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gives in and this marks the beginning of heart failure.[2]

Left Atrium gets affected either directly or indirectly post STEMI. A few studies of LA dysfunction in IWMI patients have been done previously.

LA abnormalities post STEMI can be explained in three ways: reduced LV systolic function causing reduced pull exerted on the atrioventricular plane during LV systole which leads to decreased LA expansion, increased LA pressure as shown by the raised E/E′ or as intrinsic LA ischaemic abnormalities in those with involvement of LA branches.[3]

Arteries supplying the LA are among the earliest branches of the LCA, usually from the LCX, and originate along the AV groove.[4]

Left Atrial dysfunction can be studied as electrical (picked up on ECG) and mechanical dysfunction (studied on echocardiography, CMR or catheterization study). Atrial tachyarrythmias (most commonly atrial fibrillation) occur in about 8% of acute inferior myocardial infarction.[2-5]

Several parameters can be studied on echocardiography for assessing mechanical dysfunction of Left Atrium. These include volumetric measurements or Doppler measurements.

Maximum LAV has emerged as an important biomarker for adverse cardiac events in a variety of cardiovascular conditions and is an established surrogate for the severity and chronicity of LVDD.[6] Whereas maximum LA volume represents the long-term diastolic memory of the heart, minimum LA volume represents also the short-term diastolic memory of the heart.[7]

Minimum LA volume is also influenced by the extent of atrial contraction, a compensatory mechanism influenced by acute haemodynamic changes in the setting of acute MI.

Recent findings suggest that LA minimum volume index (LAVImin) is more strongly related to LV filling disorders and is a better prognostic marker of future cardiovascular events than LAVImax.[8]

Studies have shown that the LA emptying fraction (LAEF), measured as (LVImax-LAVImin)/ LAVImax, is even more strongly related to cardiovascular events than its component LA phasic volumes in a community-based cohort.[9]

A novel measure of atrial function, the LA functional index (LAFI), is the product of LA emptying fraction and LV outflow tract velocity time integral, divided by the LAVi.[10] It is a marker which incorporates left atrial function and left ventricular systolic and diastolic function. LAFI has been extensively studied in various studies to be of prognostic importance of developing heart failure, AF development and cardiovascular events.[10] Left Atrial Ejection Force (LAEF) is calculated as the product of the mass and acceleration of blood from the LA during atrial systole. LAEF was calculated using the formula:

$$\text{Left atrial function Index (LAFI)} = \frac{\text{LA emptying fraction} \times \text{LVOT VTI (cm)}}{\text{LAESV indexed to BSA (cc/m}^2\text{)}}$$

Left atrial ejection force has been shown to increase in hypertensive patients and studies have also linked falling LAEF as a marker of developing future CHF.[11] Atrial ejection force provides a physiologic assessment of atrial systolic function and is a potentially useful index for assessing atrial contribution to diastolic performance. In patients who successfully underwent cardioversion from atrial fibrillation, atrial ejection force improved over several weeks only in the subgroup in which sinus rhythm was maintained.

LAEF in Kdynes = 1/3 × MVA × square of trans-mitral A wave velocity.

MATERIALS AND METHODS

Study Population: The study was performed in SMS medical college and allied hospitals. 25 cases were recruited from cardiology emergency with presentation of acute STEMI falling outside window period for revascularization. Patients excluded included those with previous history of ACS/RHD/ AF or those requiring inotropes/ pacemaker or CPR. The control population included 25 age0 and risk factor matched (DM/HTN/SMOKING) apparently healthy individual recruited from cardiology OPD of the same institute with no prior cardiac disease. The ethical committee of the institute approved the study. Written informed consents of all the participants were taken. All cases and controls were evaluated with a brief history including demographic data and risk factor evaluation followed by resting 12 lead ECG and detailed 2D ECHO evaluation with emphasis on LA function. 21 cases underwent coronary angiography, and 4 patients did not consent to invasive treatment and were advised conservative management.

ECG Evaluation: All cases and controls underwent standardized 12 lead ECG with additional right side and posterior lead ECG for patients with IWMI. The type of STEMI was diagnosed. P wave duration and amplitude were calculated. Rhythm was analysed to look for any atrial arrythmias.

Echocardiographic Evaluation: Experienced echocardiographers performed detailed 2d ECHO evaluation of all cases and control using a commercially available system (GE). ECG gated images were acquired in supine or left lateral recumbent positions.

All measurements were performed by experienced echocardiographers. Conventional PLAX, short axis (at aortic basal, mid and apical levels) and apical (4 chambers, 2 chamber, 5 chamber) views were recorded. Chamber dimensions were assessed in plax m-mode. Valvular assessment was done according to standard techniques in appropriate views. LVEF was assessed by eyeballing in short axis views.

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LA phasic volumes were assessed by area biplane at 3 points in cardiac cycle- At the end of QRS (LAV MAX), at the start of p wave (LAV pre A), at the start of QRS (LAV MIN). Area in 2 chamber and 4 chamber view were noted and atrial length was noted in 4 chamber view. Methods (Left atrial volume= Area 4 chamber*Area 2 chamber*0.85/atrial length). All the volumes were indexed to body surface area by Du-Bois formula.

Table 1: Echocardiographic assessment of volumetric parameters of left atium.

LA function	LA Volume fraction	Calculation
Global function; reservoir	LA EF (or total EF)	[(LAmax- LAmin)/LAmax]
Reservoir function	Expansion index	[(LAmax-LAmin)/LAmin]
Conduit function	Passive EF	[(LAmax-LApre-A)/LAmax]
Booster pump	Active EF	[(LApre-A-LAmin)/LApre-A]

Mitral inflow velocities were studied with pulse wave placed at the tip o mitral valves. Mitral early rapid filling E, atrial systolic filling A, velocity time integral of a wave A- VTI, velocity time intergral of LVOT in 5 chambers. Tissue doppler was used to calculate e' at the medial annulus. Mitral valve area was traced in parasternal short axis at basal level. E/e' was calculated using the above values.

LA functional index (LAFI) was calculated as the product of LA emptying fraction and LV outflow tract velocity time integral, divided by the LAVi Max

LAEF was Calculated using the Formula: LAEF in Kdynes = 1/3 × MVA × square of trans-mitral A wave velocity Where MVA in the mitral valve area assessed by 2-D planimetry.

Coronary Angiographic Evaluation: All cases who consented to invasive evaluation underwent coronary angiography with standard operating protocols. Lesion quantification was done based on visual inspection by experienced operators. Coronary dominance was ascertained and culprit vessel causing acute STEMI was identified.

Statistical Evaluation: The collected data was entered into Microsoft Excel and then analysed and statistically evaluated using SPSS-25 version.

Normality of each variable was assessed by using the Kolmogorov- Simirnov test. Quantitative data was expressed by mean, standard deviation and difference between means of two group were tested by Unpaired t test or Mann Whitney U test while Qualitative data were expressed in percentage and difference between percentage of two group were tested by chi square test or Fisher exact test. Spearman correlation coefficient was used to see correlation between two quantitative variables 'P' value less than 0.05 would be considered statistically significant.

RESULTS

The baseline characteristics are presented in Table 2. The mean age amongst cases (62.36±10.17) and control (59.92±12.81) were comparable. The study population was predominantly males with no statistical difference of sex ratio in the two groups (case 22:3 and controls 20:5). Diabetics (5 cases and 4 controls) and hypertensives (7 cases and 8 controls) and smoking addiction (14 cases and 8 controls) were comparable in the two groups. Average heart did not show any statistical difference among cases (80.56±10.60) and controls (79.44±6.17). SBP as well as DBP was significantly low among cases compared to controls.

The comparison of various parameters between cases (n=25) and controls (n=25) [Table 2] revealed significant differences in several measurements. The E wave velocity was significantly lower in cases compared to controls (61.48 ± 19.33 vs. 71.92 ± 14.21, P = 0.03), while the A wave velocity showed no significant difference (67.76 ± 12.02 vs. 73.96 ± 11.47, P = 0.06). The e' velocity was notably reduced in cases (5.52 ± 1.61 vs. 6.60 ± 2.04, P = 0.01), but there was no significant difference in the e/a ratio (0.91 ± 0.27 vs. 0.99 ± 0.22, P = 0.29) or e/e' ratio (12.20 ± 7.75 vs. 10.35 ± 3.13, P = 0.93). Among the STEMI patients diastolic dysfunction was present in 21 patients (grade I -14, grade II-6 grade III- 1) compared to 18 subjects in control group (grade 1-14, grade II- 4). The mitral valve area (MVA) and A wave velocity-time integral (VTI) were comparable between groups (P = 0.58 and P = 0.49, respectively).

Left atrial parameters showed significant variations: LAEF% was significantly lower in cases (40.78 ± 13.39 vs. 51.19 ± 10.89, P < 0.01), as were reservoir function (76.56 ± 36.70 vs. 115.12 ± 48.85, P < 0.01) and conduit function (17.69 ± 10.53 vs. 23.65 ± 9.28, P = 0.05). Booster function also showed a trend toward reduction in cases (27.70 ± 14.32 vs. 35.83 ± 12.78, P = 0.05), while left atrial ejection force did not differ significantly (6930.08 ± 2544.12 vs. 8362.65 ± 2724.23, P = 0.07). The left atrial volume index (LAVI) at maximum, pre-A, and minimum points showed no significant differences, though LAVImin was slightly higher in cases (14.15 ± 6.93 vs. 11.17 ± 2.99, P = 0.12). Additionally, the left atrial functional index (LAFI) was significantly reduced in cases (28.56 ± 14.74 vs. 48.30 ± 33.86, P < 0.01). Lastly, LVOT VTI was markedly lower in cases (13.98 ± 3.27 vs. 20.75 ± 11.97, P < 0.001).

The correlation analysis of left atrial ejection fraction percentage (LAEF%) with various parameters in cases revealed significant associations (Table 4). LAEF% showed a negative correlation with age (r = -0.495, P = 0.012), indicating that LAEF% decreases with increasing age. It positively correlated with left ventricular ejection fraction (LVEF; r = 0.551, P = 0.004), suggesting better left atrial function with improved ventricular performance. LAEF% did not

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show a significant correlation with the e/e' ratio (r = -0.106, P = 0.613).

Among left atrial volume indices, LAEF% negatively correlated with LAVImax (r = -0.508, P = 0.009), LAVI pre-A (r = -0.651, P < 0.001), and LAVImin (r = -0.852, P < 0.001), reflecting impaired left atrial function with increased atrial volumes. Reservoir function exhibited a perfect positive correlation with LAEF% (r = 1.000), while conduit function (r = 0.414, P = 0.040) and booster function (r = 0.750, P < 0.001) also showed significant positive correlations. Additionally, left atrial ejection force correlated positively with LAEF% (r = 0.415, P = 0.039). Lastly, the left atrial functional index (LAFI) showed a strong positive correlation with LAEF% (r = 0.768, P < 0.001).

The comparison of parameters between patients with anterior wall myocardial infarction (AWMI) and inferior wall myocardial infarction (IWMI) showed several differences, though many were not statistically significant. Heart rate (HR) was slightly higher in AWMI patients (83.45 ± 14.17) compared to IWMI patients (78.29 ± 6.36, P = 0.52), while systolic blood pressure (SBP) was lower in IWMI (106.29 ± 7.13 vs. 118.73 ± 23.17, P = 0.20). Diastolic blood pressure (DBP) was similar between groups (23.68 ± 8.43 for AWMI vs. 22.53 ± 5.78 for IWMI, P = 0.10), as was age (61.45 ± 10.52 vs. 63.07 ± 10.23, P = 0.60).

The comparison of left atrial ejection fraction percentage (LAEF%) across different subgroups (Table 3) highlighted variations in specific groups. Among non-diabetic and diabetic participants, LAEF% was higher in diabetics (49.17 ± 7.02) compared to non-diabetics (38.68 ± 13.89), though the difference was not statistically significant (P = 0.13). Similarly, no significant difference was observed in LAEF% between participants without hypertension (41.06 ± 12.48) and those with hypertension (40.07 ± 16.56, P = 0.87). However, smoking status revealed a significant impact, with smokers showing a lower LAEF% (36.36 ± 14.21) compared to non-smokers (46.41 ± 10.27, P = 0.04).

No significant difference was observed in e/e' ratio (P = 0.84), maximum left atrial volume index (LAVImax, P = 0.91), pre-A left atrial volume index (LAVI pre-A, P = 0.54), or minimum left atrial volume index (LAVImin, P = 0.38). Reservoir function tended to be higher in IWMI (85.14 ± 24.39 vs. 65.65 ± 47.18, P = 0.10), and conduit function also trended higher in IWMI (21.08 ± 11.20 vs. 13.37 ± 8.16, P = 0.09). Booster function and left atrial ejection force were slightly higher in IWMI, though not significantly different (29.84 ± 9.10 vs. 24.98 ± 19.23, P = 0.32, and 7672.46 ± 2490.24 vs. 5985.23 ± 2391.41, P = 0.12, respectively). Similarly, the left atrial functional index (LAFI) was comparable between groups (30.27 ± 10.34 for IWMI vs. 26.38 ± 19.31 for AWMI, P = 0.47). However, left ventricular ejection fraction (LVEF) was significantly higher in IWMI patients (47.07 ± 1.68) compared to AWMI patients (36.73 ± 7.14, P = 0.001).

The comparison of left atrial ejection fraction percentage (LAEF%) across different coronary artery (Table 6 & 7) blockages and culprit arteries showed varying patterns. For left anterior descending artery (LAD) blockages, LAEF% was lower in cases with >70% blockage (38.70 ± 15.18) compared to those with <70% blockage (47.11 ± 10.92), though the difference was not statistically significant (P = 0.18). In left circumflex artery (LCX) blockages, LAEF% was similar between groups with <70% and >70% blockages (40.07 ± 16.36 vs. 41.18 ± 13.67, P = 0.94). Similarly, for right coronary artery (RCA) blockages, no significant difference was observed between groups with <70% and >70% blockages (38.14 ± 13.30 vs. 41.73 ± 15.26, P = 0.31).

When comparing LAEF% across different culprit arteries, the mean LAEF% was highest in cases with RCA as the culprit artery (46.14 ± 8.34), followed by LCX (42.72 ± 0.62) and LAD (34.22 ± 19.02). However, the differences in LAEF% between culprit arteries were not statistically significant (P = 0.24).

Table 2: Baseline characteristics in study subjects.

	Cases (n=25)	Controls (n=25)	P value
Mean age	62.36±10.17	59.92±12.81	0.46
Male: Female	22:3	20:5	0.44
DM	5	4	1.0
HTN	7	8	0.75
Smoking	14	8	0.08
HR	80.56±10.60	79.44±6.17	0.65
SBP	111.76±17.25	126.88±14.77	<0.01
DBP	73.04±10.92	79.12±7.87	0.02

Table 3: Comparison of different parameters between cases and controls

	Cases (n=25)	Controls (n=25)	P value
E	61.48±19.33	71.92±14.21	0.03
A	67.76±12.02	73.96±11.47	0.06
e'	5.52±1.61	6.60±2.04	0.01
e/a	0.91±0.27	0.99±0.22	0.29
e/e'	12.20±7.75	10.35±3.13	0.93
MVA	4.45±0.65	4.56±0.74	0.58
A wave VTI	6.38±1.20	6.61±1.20	0.49
LAVI max	23.03±6.93	23.25±5.87	0.79

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LAVI pre A	19.01±6.47	17.47±3.73	0.65
LAVI min	14.15±6.93	11.17±2.99	0.12
LAEF%	40.78±13.39	51.19±10.89	<0.01
Reservoir function	76.56±36.70	115.12±48.85	<0.01
Conduit function	17.69±10.53	23.65±9.28	0.05
Booster function	27.70±14.32	35.83±12.78	0.05
Left atrial ejection force	6930.08±2544.12	8362.65±2724.23	0.07
LAFI	28.56±14.74	48.30±33.86	<0.01
LVOT VTI	13.98±3.27	20.75±11.97	<0.001

Table 4: Comparison of LAEF% between different subgroups

	Non-Diabetic (n=20)	**Diabetic (n=5)**	**P value**
LAEF%	38.68±13.89	49.17±7.02	0.13
	Non-HTN (n=18)	HTN (n=7)	P value
LAEF%	41.06±12.48	40.07±16.56	0.87
	Nonsmoker (n=11)	Smoker (n=14)	P value
LAEF%	46.41±10.27	36.36±14.21	0.04

Table 5: Correlation of LAEF with different parameters in cases

		LAEF%
AGE	r value	-.495
	p value	.012
	N	25
LVEF	r value	.551
	p value	.004
	N	25
e/e'	r value	-.106
	p value	.613
	N	25
LAVI max	r value	-.508
	p value	.009
	N	25
LAVI pre A	r value	-.651
	p value	.000
	N	25
LAVI min	r value	-.852
	p value	.000
	N	25
RESERVOIR FUNCTION	r value	1.000
	p value	.
	N	25
CONDUIT FUNCTION	r value	.414
	p value	.040
	N	25
BOOSTER FUNCTION	r value	.750
	p value	.000
	N	25
LEFT ATRIAL EJECTION FORCE	r value	.415
	p value	.039
	N	25
LAFI	r value	.768
	p value	.000
	N	25

Table 6: Comparison of different parameters between type of MI

	AWMI	**IWMI**	**P value**
HR	83.45±14.17	78.29±6.36	0.52
SBP	118.73±23.17	106.29±7.13	0.20
DBP	23.68±8.43	22.53±5.78	0.10
Age	61.45±10.52	63.07±10.23	0.60
e/e’	13.91±11.29	1.86±2.93	0.84
LAVI max	23.68±8.43	22.53±5.78	0.91
LAVI pre A	20.48±7.64	17.85±5.39	0.54
LAVI min	16.28±9.26	12.47±3.98	0.38
Reservoir function	65.65±47.18	85.14±24.39	0.10
Conduit function	13.37±8.16	21.08±11.20	0.09
Booster function	24.98±19.23	29.84±9.10	0.32
Left atrial ejection force	5985.23±2391.41	7672.46±2490.24	0.12
LAFI	26.38±19.31	30.27±10.34	0.47
LVEF	36.73±7.14	47.07±1.68	0.001

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Table 7: Comparison of LAEF% with angiographic vessel involvement

	<70%	>70%	P value
LAD	47.11±10.92	38.70±15.18	0.18
LCX	40.07±16.36	41.18±13.67	0.94
RCA	38.14±13.30	41.73±15.26	0.31

Table 8: Comparison of LAEF% between different culprit artery

	N	Mean	Std. Deviation	P value
RCA	10	46.1425	8.343	0.24
LCX	2	42.7244	0.623	
LAD	9	34.2221	19.023	

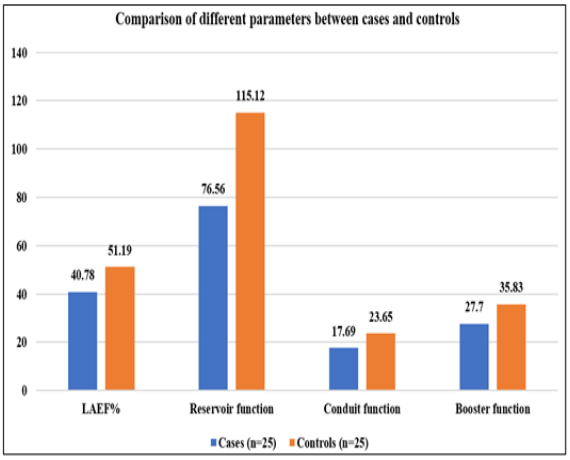

Figure 1: Comparison of different parameters between cases and controls

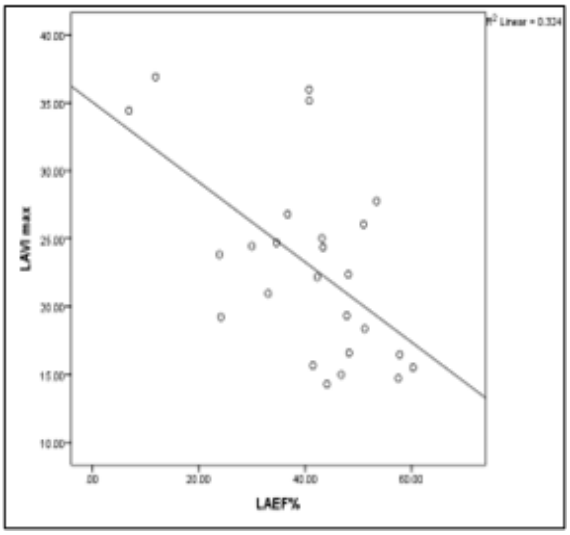

Figure 2: Relationship of Left atrial emptying fraction (LAEF%) with LAV Imax

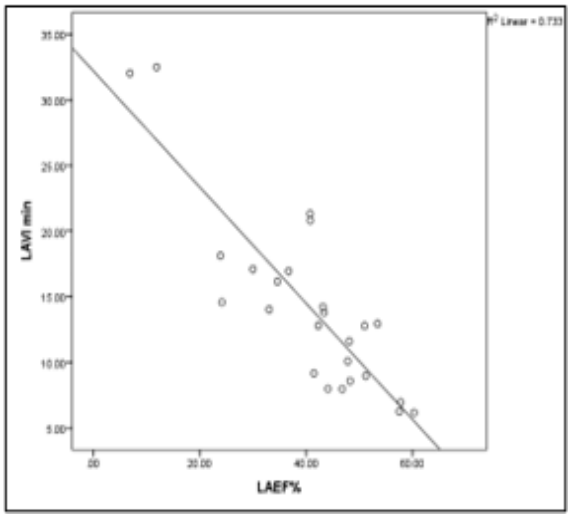

Figure 3: Relationship of Left atrial emptying fraction (LAEF%) with LAVImin

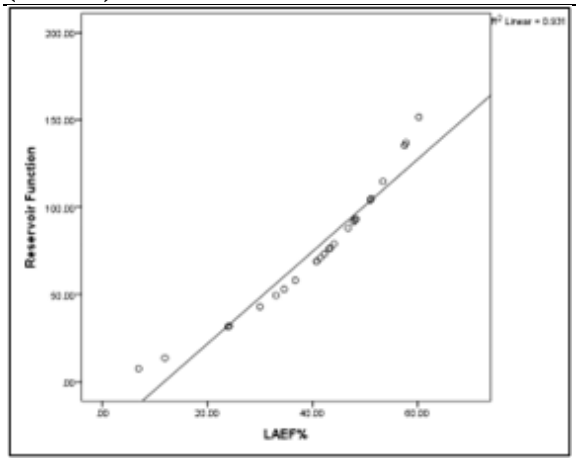

Figure 4: Relationship of Left atrial emptying fraction (LAEF%) with reservoir function

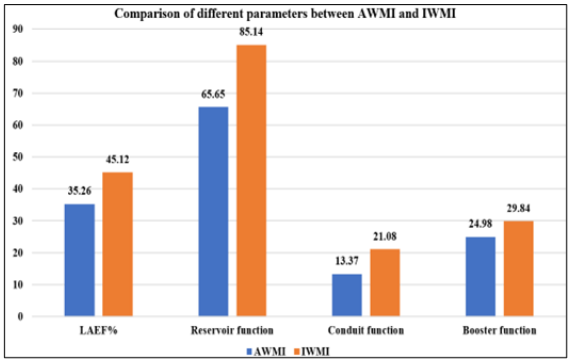

Figure 5: Comparison of different parameters between AWMI and IWMI

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DISCUSSION

All the characteristics were well matched with respect to age, sex distribution, Diabetic status, hypertension & smoking. Cases had lower SBP and DBP compared to controls secondary to acute LV dysfunction.

LA function is affected with age as evidenced by our findings. LAEF shows a significant fall in the cases when compared to controls. This rejects our null hypothesis and establishes the fact that LA functions are affected in STEMI patients compared to controls. LA volumes in different phases are higher in the cases compared to controls though statistically insignificant which can be explained by the fact that it takes long standing increase in LVEDP or long standing systolic or diastolic dysfunction to cause an increase in left atrial volumes. LAVI min is affected more than LAVI max which accounts for the affection of pump function of Left atrium in acute MI. All the functions of left atrium are affected in STEMI compared to healthy individuals. The most significantly affected function is the reservoir function i.e. the ability of the left atrium to expand.

This underscores the importance of mechanics of LA expansion which is not merely a passive phenomenon but aided significantly by the pull generated by the left ventricle during ventricular systole. During ventricular systole the pull of the ventricle on mitral annulus causes the left atrium to stretch and become cylindrical, thus contributing to LA expansion.

Conduit function is a passive phenomenon and is affected primarily because of the impaired LV relaxation. Diastolic dysfunction is the first abnormality to set in during acute MI even before systolic dysfunction.

Booster pump function is affected by multiple mechanisms but primarily because of the impaired mechanical pump function secondary to ischemia of left atrium. The other possible contributing factor could be a left atrial failure in line with the frank starling law as during acute MI the new onset diastolic and systolic dysfunction puts extra burden on left atrium thus giving away beyond a limit.

So what takes the additional burden of blood passage if all functions of left atrium decrease during acute MI. This brings us to the key concept of transit volume. Blood flow from atria to ventricle during diastolic rapid filling is contributed by two components- first is the stored blood of left atrium during ventricular systole (LA volume reduces and the blood is emptied into LV); second is the transit volume that is direct passage from pulmonary veins to LA to LV (does not involve LA expansion).

When the LA expansion ability is reduced, less blood is stored in left atrium during ventricular systole, and more blood stays in the pulmonary veins, finally draining as transit volume during early rapid filling of ventricular diastole. So, transit volume compensated for the loss of conduit and reservoir function of left atrium. But a rise in transit volume suggests more blood in pulmonary veins resulting in raised PCWP and in clinically recognizable terms left heart failure.

This underscores the importance of left atrial function in cardiac hemodynamics. LVEF has moderate correlation with LAEF as LA reservoir function is significantly affected by LV systolic function mostly by affecting the reservoir function of left atrium.

Indexed LA volumes in all phases showed significant increase with LAEF with strongest association seen with LAVI min. So, we can fairly conclude that of all the phases minimum atrial volume has better ability to predict LA function than LAVI Max (which is conventionally used). This could be explained by the fact that LA min volume incorporates two parameters- long standing increase in LA volumes

Our study revealed lower peak E and A wave velocities as well as lower A wave VTI compared to controls. Theoretically E wave has a non-linear relationship with LV diastolic dysfunction (first decrease then increase). Similarly, A wave during grade 1 diastolic dysfunction increases and then progressively falls as dysfunction worsens. So, deriving any conclusion from this observation would be inappropriate E/A was comparable in the groups falling between 0.8-1.5 as both grade 0 and grade II will have E/A in this range (more grade 2 in STEMI compared to grade 0 in controls).

Medial e' is known to decrease with worsening LV diastolic dysfunction and so significantly lower in STEMI patients compared to controls

E/e' has an established correlation with LVEDP. STEMI patients have non-significant higher values compared to controls but both groups have values < 14 which can be explained by the fact that our study included hemodynamically stable patients not in failure after adequate volume control with diuretics.

The left atrial function index (LAFI) combines the reservoir function of the left atrium (LA), the adjusted LA volume and the stroke volume and is thus not only representing a marker of atrial function but also reflecting LV systolic and diastolic function. Left atrial ejection force is decreased among cases compared to control though statistically insignificant. This is possible likely because it depends heavily on A wave VTI and as discussed the atrial pump function increases with increasing LV systolic and diastolic dysfunction only to give away beyond a certain point. Left atrial function is a complex outcome of multiple contributing mechanism. Though IWMI is associated with higher chances of ischemic LA disturbances, on the other hand AWMI with more severe LV systolic and diastolic dysfunction affects LA function to a greater extent by the other mechanism. Hence the tradeoff resulted in similar LA dysfunction regardless of the type of MI

The study found no association of the vessel involved with left atrial function. The possible explanation could be that most cases had multivessel (TVD-7, DVD-8, SVD-6). Similarly, LCX known to be vessel supplying the left atrium was involved in only 2 cases.

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Limitations: Sample size though adequately powered for the primary objective was small for secondary objectives. Sex distribution was not even. We selected cases which were outside window period and hemodynamically stable which does not truly represent ACUTE STEMI subset as a whole. Angiographic data did not take into consideration the site of critical stenosis (corroborating with blood supply of left atrium). Angiography of controls was not done. Sub clinical CAD cannot be ruled out with surety. Window period ranged from 12 hrs to 7 days. LV and consequently LA hemodynamics change rapidly in the early days post MI.

CONCLUSION

LA functions are affected in patient with STEMI though the type of MI or artery involved bears no relation with LA function. All components of LA function are affected in STEMI patient but reservoir function is most important amongst them. LAFI, a newer index of LA function, is significantly affected in STEMI patients and shows good correlation with LA function. LAVI min is a better marker of LA dysfunction than LAVI max.

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