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Frequency of LVF in First ST Elevation Myocardial Infarction (STEMI) and Comparison of Mean Left Atrial Volume in Patients With and Without Left Ventricular Failure (LVF) in First STEMI

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ABSTRACT

Introduction: The epidemic of heart failure (HF) is an important public health issue facing the health care system.[1] Acute myocardial infarction (AMI) is one of the major causes of HF.[2] Left ventricular failure (LVF) is a serious complication of AMI that can lead to sudden cardiac death.[3] Advanced Diastolic dysfunction is associated with poor prognosis of patients with AMI despite having preserved systolic function.[4] Increase Left atrial volume (LAV) which is a marker of diastolic dysfunction, is an independent predictor of death or HF hospitalization following high-risk MI.[5] **Objective:** To determine the frequency of LVF in First ST Elevation Myocardial Infarction (STEMI) and Comparison of mean LAV in Patients With and Without LVF in first STEMI. **Study Design**: Cross sectional survey. **Study Setting:** Department of Cardiology, Sheikh Zayed Hospital, Lahore. **Duration Of Study:** From 29th April 2013 to 29th October 2013. **Patients & Method:** 150 patients fulfilling the inclusion criteria were enrolled from coronary care unit of Shaikh Zayed Hospital, Lahore. All patients underwent a complete echocardiographic examination with a 3.5 MHz transducer, and were recorded on CDs. Left ventricular volume was measured by manually tracing the LV cavity using the biplane modified Simpson's algorithm when >80% of the endocardial border could be detected in both the apical four- and two-chamber views, and by a single plane when 80% of the endocardial border could be detected only in the apical four-chamber view. All patients were assessed for LVF by signs and symptoms and those who developed LVF were graded according to Killip class. Demographic data (age, name, sex, address were noted) and data was recorded on proforma attached. **Results:** Mean left atrial volume was almost same in the patients with and without left ventricular failure i.e. 35.24±1.14 and 35.16±1.19 ml. **Conclusion:** There is no significant difference in mean left atrial volume of patients who initially present with or without heart failure and a first STEMI. However this study was done on small group of patients. For better evaluation of relationship between left atrial volume and left ventricular failure in STEMI, a study with large number of patients is required.

Key words: First STEMI, LA volume, LVF.

INTRODUCTION

Cardiovascular disease is the leading cause of death in today's era. Approximately 500,000-700,000 deaths related to the coronary arteries occur in each year[1]. Acute Myocardial infarction (AMI) may lead to impairment of systolic and diastolic function and to increased predisposition to long-term complications. Left ventricular failure (LVF) occurs in about 17% of patients with acute myocardial infarction who are non diabetic and this figure is doubled in diabetics.[2]

LVF is a manifestation of acute alterations in left ventricular function. The incidence of LVF has been reported to increase with age, almost doubling after 60 years of age. Systolic dysfunction leads to LVF which is a recognized and major cause of death following AMI.[3]

Diastolic heart failure occurs when signs and symptoms of heart failure are present but left ventricular systolic function is preserved (i.e., ejection fraction greater than 50 percent). The incidence of diastolic heart failure increases with age; therefore, 50 percent of older patients with heart failure may have isolated diastolic dysfunction.

With early diagnosis and proper management the prognosis of diastolic dysfunction is more favorable than that of systolic dysfunction.[4] In AMI, diastolic dysfunction is an important parameter to define because advanced diastolic dysfunction leads to increase in hospital mortality.[3]

After an AMI, myocardial ischemia, cell necrosis, micro vascular dysfunction, and regional wall motion abnormalities will influence the rate of active relaxation. In addition, interstitial edema, fibrocellular infiltration, and scar formation will directly affect LV chamber stiffness. Thus, abnormalities in LV filling are common in this setting.

Mitral inflow velocity is the common method to determine the diastolic dysfunction. By mitral inflow velocities, grade III or IV diastolic dysfunction is associated with poor in hospital survival in patients with AMI.[5] But mitral inflow velocities are suddenly changed in acute insult of myocardium such as in AMI.

Hence, mitral inflow velocities at the time of the AMI do not accurately define the chronic LA remodeling against increased LV filling pressures as in diastolic heart failure because these variables reflect the beat-to-beat interaction of LV filling pressures and ventricular compliance, making them sensitive to rapid alternations in ventricular preload and after load.

LAV which is an index of left atrial size[6] is directly exposed to LV filling pressures during diastole of Left ventricle. The LA acts as a conduit between the pulmonary vascular bed and the LV, receiving blood from the pulmonary veins and conveying it to the LV through passive and active filling.

In addition, the atrium acts as an efficient volume sensor, releasing natriuretic peptides and other neurohormones to the circulation as a consequence of increased atrial wall stress.[7] After opening of the mitral valve, the LA and LV diastolic pressures will rapidly equalize, and emptying of the LA will be determined largely by LV filling dynamics.

Left atrium is a thin walled structure and its dimensions are affected in consequence of increased LV filling pressures as in case of diastolic dysfunction.LA size is considered to be an expression of the diastolic burden, and increased LA volume usually reflects elevated ventricular filling pressure. As an adaptation to the decreased ventricular compliance following MI, LA pressure rises, increasing LA wall tension and stretching the atrial myocardium.

LAV also reflects the duration and severity of increased LA pressure.[8] In the setting of an acute MI, patients with higher chronic LV filling pressure and worse previous diastolic dysfunction have lower hemodynamic ‘cardiac reserve’ that helps to withstand an acute decrease in myocardial contractility.[8]

Early after MI, LA size has been shown to provide prognostic information incremental to clinical data and standard echocardiographic predictors of outcome.[9] In the acute phase post-MI LA size was a better prognostic predictor of outcome than transmitral Doppler indices.

Indeed, Doppler indices may be quite sensitive to acute changes in the loading conditions secondary to HF and/or to drugs.[8] LAV is likely to be less affected by acute hemodynamic changes and may represent a more stable indicator of the duration and severity of diastolic function and filling pressure over time.[10] LAV provides prognostic information in AMI.

If LA volume is normal, outcome is good, even if systolic function is depressed. This suggests that more favorable hemodynamics before infarction may enable these patients to withstand an acute decrease in myocardial contractility.[11] LAV obtained early, within the first 48 h of admission, is a powerful independent predictor of survival after acute MI.

Moreover, new findings include the fact that LAV becomes a significant independent predictor of mortality within the first year following acute MI and are even more powerful for predicting five-year survival, and that LA volume obtained early upon

admission is a more powerful independent long-term mortality predictor than LV volume.[12]

Baseline LA size is an independent predictor of death or hospitalization for HF in patients with high-risk MI.[13]

Objectives

To determine the frequency of patients with LVF in first STEMI and to compare the mean LAV in patients with and without LVF in first STEMI.

Operational Definitions

- **STEMI:** (Defined according to consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the Redefinition of Myocardial Infarction).
- **LAV:** Measured by biplane area and length method from standard 4-chamber and 2-chamber views at end diastole.
- **LVF:** Patients will be assessed clinically by signs and symptoms and graded in Killip class I to IV(clinical signs and symptoms manifested by tachycardia, and tachypnea or dyspnea, fine crackles in the lung persisting after vigorous cough, elevated jugular venous pressure often accompanied by a third heart sound and hemodynamic compromise in class IV)

MATERIALS AND METHODS

This Cross sectional study was conducted at Department of Cardiology, Shaikh Zayed Hospital, Lahore from April 2013 to October 2013.

Sample Size

Sample size of 150 patients was calculated with 95% confidence level, 8% margin of error with taking expected percentage of LVF i.e.33.3% in patients presenting with first STEMI.

Sampling Technique

Non-Probability Consecutive Sampling

Inclusion Criteria

All patients of any gender with first STEMI with or without LVF.

Exclusion Criteria

Patients with congenital heart disease, valvular abnormality, pericardial disease, chronic atrial fibrillation/flutter and cardiomyopathy. (assessed on history, echocardiography and ECG findings)

Data Collection Procedure

A total of 150 patients fulfilling inclusion criteria were enrolled in the study from coronary care unit of SZH Lahore. All patients were assessed for LVF by sign and symptoms and those who developed, were graded according to signs and symptoms in relevant Killip class. A single cardiologist calculated LAV by standard technique at rest of each patient.

Data Analysis Procedure

Collected data was entered and analyzed using SPSS v22.0. The demographic variables included identification data and demographic characteristics. Quantitative data was described by mean, median, standard deviation and bar charts while qualitative data was described using frequency and pie charts. Paired sample t-test was used to check the significant of difference in left atrial volume. A p-value less than 0.05 was considered to be significant.

RESULTS

There were total 150 patients of which 80 (53%) were male while 70 (47%) were female with a mean age of 55.28±10.01 years. Majority of patients were within the age range of 40 to 70 years (Table 1).

When patients were evaluated for history of different factors it was revealed that diabetes was found in 105 (70%) of patients while 45 (30%) were non diabetic (defined according to WHO guidelines 2003) (Table 2).

Hypertension (defined according to JNC 7) was noted in 115 (76.7%) of patients (Table 3), while 35 (23.3%) were normotensive.68 (45%) of patients were smokers while 82 (54.7%) were non smokers (Table 4).

Most of the patients were negative for Hyperlipidemia (according to AHA guidelines) as it

was noted in just 38 (25%) of cases. Chronic kidney disease (defined according to guidelines) was noted in 43 (28%) of cases (Table 5).

Table 1: Distribution according to gender.

Gender	Frequency	Percent
Male	80	53.30
Female	70	46.70
Total	150	100.00

Table 2: Distribution according to diabetes mellitus.

Diabetes mellitus	Frequency	Percent
Yes	105	70.00
No	45	30.00
Total	150	100.00

Table 3: Distribution according to hypertension.

Hypertension	Frequency	Percent
Yes	115	76.70
No	35	23.30
Total	150	100.00

Table 4: Distribution according to being a smoker.

Smoker	Frequency	Percent
Yes	68	45.30
No	82	54.70
Total	150	100.00

Table 5: Distribution according to hyperlipidemia.

Hyperlipidemia	Frequency	Percent
Yes	38	25.30
No	112	74.70
Total	150	100.00

Left ventricular failure (assessed by signs and symptoms) was found in 56(37%) of cases while it was absent in 94(63%) of patients. In total 150 patients, 93(62%) were in Killip class I, 39(26%) were in Killip class II, 9(6%) were in Killip class III and 9(6%) were in Killip class IV. Among patients of LVF, 69 percent were in Killip class II, 16 percent were in Killip class III and similar figure was observed in Killip class IV (cardiogenic shock).

When diastolic dysfunction was analyzed by mitral inflow velocities, it was observed that among 56 patients of LVF, 9(16%) had grade I diastolic dysfunction, 33(58%) grade II, 13(23%) grade III and 2 (2%) had grade IV diastolic dysfunction.

Among 94 patients without LVF, 23(24%) had grade I diastolic dysfunction and 71(76%) had grade II diastolic dysfunction.

In total 150 patients, 32(21%) had grade I diastolic dysfunction, among which 28% had LVF and 72% were without LVF.103 patients (69%) had grade II diastolic dysfunction, among which 31% had LVF and 68% were without LVF.13 patients (13%) had grade III and 2 patients (2%) had grade IV diastolic dysfunction.

All patients with grade III and grade IV diastolic dysfunction had LVF. Interestingly among total 150 patients nobody had normal diastolic dysfunction.

Mean left atrial volume was almost the same in the patients with and without left ventricular failure i.e. 35.24±1.14 and 35.16±1.19. No significant difference in LAV among different grades of diastolic dysfunction was observed. However, an increasing trend in mean LAV was observed from grade II to grade IV diastolic dysfunction in patients with LVF in our study.

Table 6: Distribution according to chronic kidney disease.

Chronic Kidney Disease	Frequency	Percent
Yes	43	28.70
No	107	71.30
Total	150	100.00

Table 7: Distribution according to left ventricular failure.

Left Ventricular Failure	Frequency	Percent
Yes	56	37.30
No	94	62.70
Total	150	100.00

Table 8: Distribution according to Killip Class.

Killip Class	Frequency	Percent
1	93	62.00
2	39	26.00
3	9	6.00
4	9	6.00
Total	150	100.00

Table 9: Frequency of Patients in Different Grades of Diastolic Dysfunction with LVF.

Mitral Inflow Velocity	Frequency	Percent
Grade I	9	16
Grade II	32	58
Grade III	13	23
Grade IV	2	3
Total Patients	56	100

Table-10: Frequency of Patients in Different Grades of Diastolic Dysfunction without LVF.

Mitral Inflow Velocity	Frequency	Percent
Grade I	23	24
Grade II	71	76
Grade III	-	-
Grade IV	-	-
Total Patients	94	100

Table 11: Mean Left atrial Volume in Patients With and Without LVF.

Left Ventricular failure	Mean	N	Std. Deviation
Yes	35.2429	56	1.14731
No	35.1606	94	1.19625
Total	35.1913	150	1.17502

Table 12: Mean LAV in Different Grades of Diastolic Dysfunction in Patients With and Without LVF.

Mitral inflow velocity	Mean	N	Std. Deviation
Grade 1	35.5688	32	1.53716
Grade 2	34.9942	103	1.06106
Grade 3	35.7000	13	.60553
Grade 4	36.0000	2	0.00000
Total	35.1913	150	1.17502

Table 13: Mean LAV in different grades of diastolic dysfunction in patients without LVF.

Mitral inflow velocity	Mean	N	Std. Deviation
Grade 1	35.6087	23	1.63009
Grade 2	35.0155	71	0.98932
Total	35.1606	94	1.19625

Table 14: Mean LAV in different grades of diastolic dysfunction in patients with LVF.

Mitral inflow velocity	Mean	N	Std. Deviation
Grade 1	35.6182	11	1.28516
Grade 2	35.0258	31	1.12752
Grade 3	35.7500	12	0.61126
Grade 4	36.0000	2	0.00000
Total	35.3321	56	1.09181

DISCUSSION

This study was planned to determine the frequency of LVF in patients with AMI and to find the association of mean left atrial volume and LVF in first STEMI as a number of studies conducted internationally showed variable results while local data was lacking. Most of the previous studies which were conducted, had prospective analysis of association of LAV and long term morbidity and mortality.[14-16] No local or international study was available regarding the association of mean left atrial volume with LVF in first STEMI or lack thereof.

It has been demonstrated in a number of studies that LAV provides independent and additional prognostic value for long-term mortality and cardiovascular deaths in patients with AMI.[14-16]

In one previous study, when LAV was measured in initial 48 hours of presentation with STEMI, it was observed that patients with LAVI >32 ml/m2, had subsequently more admissions with heart failure (Killip score >2), compared to patients who had LAVI <32ml/m^2.[17] Neither the LV filling pattern nor the E/A ratio was significantly different between the groups with LAVI <32 ml/BSA and LAV >32ml/BSA .[17]

During a follow-up period of 5 years,

mortality rate was significantly higher in patients with LAVI >32 ml/m^2 as compared to patients with LAV <32ml/m^2, (34.5% vs. 14.2%, p<0.001). This difference was particularly significant after the first year of the acute MI .But there was no significant difference between the two groups in mortality rate during the first 30 days.[17]

The VALIANT Echo Study showed that post-MI early left atrial remodeling(during the first month) as well as late remodeling(at 20 months) was a significant predictor of all-cause mortality, HF hospitalization, or the composite outcome of all-cause mortality or hospitalization for HF.[18]But LA remodeling was not significantly related to age and baseline Killip class.[18]

In another study patients with Left atrial volume index >32 ml/m^2, had a higher incidence of congestive heart failure on admission (24% vs. 12%, p<0.01).[19]

In our study, we failed to determine any association between mean LAV and LVF in AMI at initial presentation. Patients who presented with LVF and those without LVF didn't have any significant difference in mean LAV, measured within initial 48 hours.

We cannot compare our results with most of the above mentioned studies because in these studies, mean LAV was not compared in patients with and without LVF in AMI. Baseline LAV was used as a reference value and then left atrium volume was assessed as prognostic marker prospectively in these studies (i.e. after a period of one month, one year, 20 months and five year after AMI)[14,19] except in one study in which LAV index was assessed as prognostic marker at the time of presentation.[20] However mean LAV was never compared in AMI in patients with and without LVF in any previous study.

The reason why no significant difference was observed in our study between mean LAV of the two groups, may be because left atrial remodeling needs time after AMI as in one study there was no difference in mortality and morbidity at 30 days in patients with LAV>32/ml but a difference was observed at 20 months.[14]

In another study among patients in whom Doppler tissue imaging was performed in patients of AMI with LAV of less than and more than 32ml, no significant correlation was found between diastolic dysfunction and LA volume index.[20]

In our study no significant association between mitral inflow velocities and left atrial volume was found in AMI which is consistent with the older studies.[20]

Killp class of heart failure is significantly associated with increase left atrial volume on admission. Patients who were admitted with LAV >32ml had worse Killip class (>II) on admission.[19] In our study, no significant difference in LAV among patients of different Killip classes was observed which is not consistent with previous studies. However, an increasing trend in LAV was observed from Killip II to Killip IV in patients with LVF in our study. This may have become significant if the studied group was larger in number.

One interesting finding in our study is that no AMI had normal LV filling patterns. This finding is compatible with one previous study in which frequency of diastolic dysfunction after AMI was 96%.[21] But in another study, frequency of diastolic dysfunction after AMI was 58%.[22]

Limitations of study:

Regarding limitations of study:

1) Our study consisted of small number of patients.
2) We did not know the pre infarct size of the left atrium.
3) There was no follow up of the patients with LVF as in most of the previous studies; LAV was assessed as a prognostic marker prospectively because left atrial remodeling after AMI is associated with long term morbidity and mortality.[12,14]

These limitations may be avoided in further trials to strengthen the results of the current study.

CONCLUSION

From our study it is concluded that mean LAV has no impact on LVF in AMI. Larger studies are warranted to enhance our understanding of impact of mean LAV on left ventricular function in AMI. The utility of LA volume and function for

monitoring cardiovascular risk and for guiding therapy is an evolving science and may prove to have a very important public health impact.

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