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ORIGINAL ARTICLE

Characteristics Of Patients with Left Ventricular Free Wall Rupture Related Death in Acute Myocardial Infarction Setting: A Retrospective Analysis of Patients from A Single Tertiary Cardiac Centre.

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Abstract

Introduction: Left Ventricular free wall rupture (LVFWR) is a lethal and rare complication of acute myocardial infarction (AMI). LVFWR accounts for 8–17% of mortality in STEMI patients. Characteristics, predisposing factors which are vital in preventing this complication are yet to be studied well.
Objective: To study the characteristics and predisposing factors of patients with LVFWR related death in AMI setting.
Methods: All AMI patients, whose death is documented to be due to primarily LVFWR, were included in the study. Patient's demographic data, risk/precipitating factors, clinical characteristics, in-hospital course, revascularization strategies, and angiographic data were collected from our institutional database.
Results: A total of 69 patients with LVFWR related deaths were identified. Mean age of the study subjects was 65.32 years. 36 patients were females, 33 were males. 40% of subjects were in the age group of 61-70 years. Hypertension is the most common associated risk factor seen in 55% patients. Mean duration of presentation since the onset of symptoms is around 23 hours. Thirty patients (43%) presented in a state of cardiogenic shock. Beta blockers could be initiated in just above 20% of patients. Majority of patients (71%) received thrombolysis. Majority had single vessel disease (61% of those who underwent angiogram). LAD was the most common Culprit vessel seen in 8 out of 13 angiograms (61.5%).
Conclusion: Classical risk factors of LVFWR like advanced age, female gender, delayed presentation, presence of hypertension; first acute MI and cardiogenic shock at the time of presentation were well appreciated. Majority of LVFWR patients had single vessel disease predominantly left anterior descending artery disease. Majority had undergone thrombolysis.

Keywords: Left ventricular free wall rupture, acute myocardial infarction, STEMI, cardiogenic shock

Introduction

Left Ventricular free wall rupture (LVFWR) is a lethal and rare complication of acute myocardial infarction (AMI). Its Incidence is 1.3–2.5% in STEMI patients.1- 3 LVFWR accounts for 8–17% of mortality in STEMI patients.4 It can occur in one of two types: either acute lethal form or a subacute form where a blood clot in rare circumstances seals the defect and results in the formation of a ventricular pseudoaneurysm. Subacute variant occurs in one third of LVFWR patients.

Approximately 50% of patients with LVFWR are diagnosed within 5 days of the AMI, with 90% diagnosed within 2 weeks. Risk factors for the development of free-wall rupture following AMI

include age, female gender, hypertension, first MI, and poor coronary artery collateralization. The advent of primary percutaneous interventions (PCI), when compared to the pre-thrombolytic or the thrombolytic eras, has considerably reduced the rates of LVFWR.5

Urgent transthoracic echocardiography (TTE) is the gold standard for definitive diagnosis of LVFWR. Cardiac Magnetic Resonance (CMR) / Multi-Detector Computed Tomography (MDCT) are feasible options for well characterization of Psuedoaneurysm in subacute cases. Urgent surgical repair is still the rule for treatment of LVFWR using pericardial patch closure or less frequently infarctectomy with patch placement and ventricular wall reconstruction. Recently sutureless techniques are being employed more frequently.

LVFWR carries a poor prognosis. However, due to its rare and unpredictable incidence, there is a large lacuna in our understanding of the disease. Not many studies are available in the literature regarding LVFWR. Characteristics, predisposing factors which are vital in preventing this complication are yet to be studied well, as previous Indian studies have included a very small sample size. This study, with a relatively larger sample size would be helpful in understanding the characteristic of the disease better in Indian setup.

Materials and methods

This is a retrospective observational study. All acute MI (AMI) patients, whose death is documented to be due to primarily LVFWR, were included in the study. Patient's demographic data, risk/precipitating factors, clinical characteristics, in-hospital course, revascularization strategies, and angiographic data (wherever applicable), were collected from our institutional database between August 2018 and May 2020. Identification of LVFWR was primarily done by 2D Echocardiographic parameters. As an institution protocol, LVFWR is identified on 2D echocardiography by an experienced board certified cardiologist. Useful findings in Echocardiography involve a pericardial collection with signs of cardiac tamponade, including collapse of the right atrium and ventricle in diastole, a dilated inferior vena cava, and marked respiratory variation in mitral and tricuspid valve inflow.

Statistical analysis

Descriptive statistical analysis has been carried out in the present study, expressed as percentage and mean.

Results

From the database of patients presenting with AMI between August 2018 and May 2020, 69 patients with LVFWR related deaths were identified. Table I shows the gender distribution of the subjects. Mean age of the study subjects was 65.32 years. 36 patients were females, 33 were males.

Table 1: Gender distribution of Study subjects

Gender distribution	N (%)	Mean age (y)
Males	33 (47.8)	63.3
Females	36 (52.1)	67.3
Overall	69	65.3

Table 2: Age distribution of study subjects

Age group in years	N(%)
>80	2 (2.8)
71-80	17 (24.6)
61-70	27 (39.1)
</=60	23 (33.33)

Majority of the study subjects i.e. 40% were in the age groupof 61-70 years as shown in Table II. Very elderly i.e. > 80 y constituted less than 3% of patients.

Table 3: Distribution of study subjects based on Risk factors

Associated risk factors	N (%)
Past history of IHD	8 (11.5)
Diabetes mellitus	20 (28.9)
Hypertension	38 (55)
Smoking	32 (46.3)
Dyslipidemia	8 (11.5)

As shown in Table III, Hypertension is the most common associated risk factor seen in 55% patients, followed by smoking which is seen in 46% of patients. Diabetes Mellitus is a less frequently seen risk factor in only 20 out of 69 patients (28.9%). Only eight patients (11%) had past history of IHD.

Table 4: Details of clinical presentation

Clinical presentation	Frequency (N) %
Mean Duration between symptom onset and presentation to the hospital	23.38 hours
Mean TIMI score	5.83
Type of MI:	
AMI site	
-Anterior	44(63.7%)
-Inferior	20 (28.9%)
-Lateral	2 (2.8%)
-Right	0
-Posterior	9 (13%)
Timing of ruptue:	
Early(< 48 h)	40(57.9%)
Late (>48 h)	29 (42%)
Killip class	
I	25 (36.2%)
II	8 (11.5%)
III	6 (8.6%)
IV	30(43.4%)

Table IV shows the details of clinical presentation. Mean duration of presentation since the onset of symptoms is around 23 hours. Forty four patients presented less than 24 h (63.76%) and 25 patients presented after 24 h duration (36.23%).Thirty patients (43%) presented in a state of cardiogenic shock.

Eight patient out of 69 (11.5%) had complete heart block. One patient had 2:1 AV block in ECG.

Table 5: Investigations

Parameter	Mean value
Laboratory Parameters :	
Creatinine	1.12
Troponin T	1.36
Echo Parameters :	
EF	36.09
LVIDd	4.85

Table V shows relevant investigation findings. Mean EF at the time of presentation was just over 36%.

Table 6: Treatment received

Treatment	N(%)
Medication:	
Beta blockers	15 (21.7)
ACE inhibitors/ ARBs	37 (53.6)
Statins	
	69 (100)
Revascularization	
Thrombolysis	49 (71)
PTCA	
-Facilitated	3 (4.3)
-Rescue	5 (7.2)
-Primary	4 (5.7)

Table VI shows the treatment received by the patients. Beta blockers could be initiated in just above 20% of patients. Majority of patients (71%) received thrombolysis with streptokinase. Out of which five patients had failed thrombolysis and underwent rescue PTCA. Other 3 patients underwent elective PTCA.

Table 7: Coronary Angiographic data

Coronary Angiogram Findings	N(%)
CAG performed	13
LM	1 (7.6)
SVD	8 (61.5)
DVD	3 (23)
TVD	2 (28.9)
Culprit vessel PTCA	Total- 12
LAD	
LCX	8 (66.6)
RCA	3 (25)
	1 (8.3)

Table VII shows the coronary angiogram data. Only 13 out of 69 underwent Coronary angiogram. Twelve patients underwent PTCA. One had Left main with Double vessel disease and was awaiting CABG. Primary PTCA was possible in only 4 out of 69 patients. Majority had single vessel disease (61% of those who underwent CAG). LAD was the most common Culprit vessel seen in 8 out of 13 angiograms (61.5%).

Discussion

This study is one of the largest among Indian studies on LVFWR in terms of number of subjects involved. There is a paucity of data from the Indian population regarding LVFWR. In this study we looked into the characteristics of AMI patients who died due to in-hospital LVFWR.

LVFWR following AMI usually occurs in elderly patients between 65 and 70 years of age.6-8 In our study, the mean age of subjects was about 65 years. Age group of 61-70 y constituted about 40% of subjects. Our study also showed a marginal female predominance which is consistent with previous data. 9-11

LVFWR is more often reported to occur after the first (transmural) myocardial infarction.8,12 In our study only eight out of 69 patient had past history of IHD. Diabetes mellitus and peripheral vascular disease are less incidental in these patients, as these conditions are associated with the development of collateral circulation, which may protect from cardiac rupture.12 In our study Diabetes mellitus was seen in less than 30% of subjects.

Previous studies showed a higher incidence of arterial hypertension in LVFWRpatients.7,13,14 One of the triggering factors for LVFWR is the presence of persistent arterial hypertension during the first 24 hours. Our study showed prevalence of hypertension in 38% of subjects.

Present study showed an obvious finding of a delay in hospital admission in the subjects. Mean duration of presentation to the hospital after symptom onset is around 23hours. This correlated with previous data, which showed a delay in hospital admission as one of the major predictor of LVFWR.8,15 In an Indian study by Swaroop et al median duration of presentation to the hospital after the onset of symptoms was 24 h.16

In our study a high incidence of patients presenting in cardiogenic shock was observed (43%). A study by Swaroop et al16 showed similar high rate of cardiogenic shock upto 28.6% upon presentation. In the present analysis, 63.7% of patients had anterior wall STEMI. This correlated with the previous data which found anterior location of infarction has a higher incidence of LVFWR.

In some studies, administration of thrombolytics has been related to an increase in LVFWR ,17-19 while in others, it was observed less frequently.20,21 Other than in a few trials,6 multivariable analysis failed to identify the use of lytics as an independent risk factor for LVFWR. In our study, proportion of patients who received thrombolysis was71% and primary PTCA was received by only 5.7% of patients. Late or failed thrombolysis is usually linked to increased rates of LVFWR.22,23 In our study, 5 patients had failed thrombolysis and all of them underwent successful rescue PTCA of culprit artery. PCI independently reduces the risk of LVFWR in comparison with thrombolysis.24 Paradoxically, despite conferring an overall mortality-rate benefit, fibrinolytic agents have been implicated in the accelerated occurrence of LVFWR within the first 24 to 48 h after AMI. 25 Cardiac rupture is prevented by early thrombolysis therapybut is promoted by late treatment.

Previous data suggest that approximately 50% of patients with free-wall rupture are diagnosed within 5 days of the AMI, with 90% diagnosed within 2 weeks. In our study, 57.9% had LVFWR less than 48 h of AMI.

In the study by Swaroop et al.16 majority of the LVFWR patients presented with a single-vessel disease. Our study reinforced this, as 61.5% of subjects had SVD. A study by Markowicz et al reported that the most common culprit coronary lesion associated with LV rupture as the left anterior descending artery in 42% of patients, the circumflex coronary artery in 40% of patients, and the right coronary artery in 18% of patients.26 In our study, out of 12 patients who underwent PTCA, 66.6% had LAD, 25% LCX and 8.3 % RCA as the culprit coronary artery.

Angiographic reperfusion success is the most significant protective factor from FWR.1 In our study 16 (23.1%) patients did not undergo any reperfusion as they were out of window period for lysis and had cardiac rupture before CAG was planned.

The treatment for LVFWR is mainly supportive in the immediate setting. None of the patient underwent surgery, as they did not survive till the surgery was undertaken. In our study usage of β-blockers among subjects was meager 21.7 % due to coexisting contraindications. Use of β-blockers and ACEI/ARB have all contributed to lower incidence and death rate of FWR as previously reported.27,28

Limitations

1. This is a single-center- based, retrospective study
2. No patients who underwent surgery could be included in the study.
3. Only acute forms of LVFWR were selected
4. Number of patients who underwent primary PTCA was very less as compared to thrombolysis group.
5.

Conclusion

This study reinforces the contributions of Classical predictors of LVFWR after myocardial infarction (AMI) in Indian setup, viz: advanced age, female gender, delayed presentation, presence of hypertension, first acute MI and cardiogenic shock at the time of presentation. Majority of LVFWR had single vessel disease predominantly LAD. β-blocker usage was considerably less in LVFWR patients. As majority of LVFWR patients had undergone thrombolysis, it is conceptualized that reduction of LVFWR may be achieved with PTCA in place of thrombolysis. This study underscores the fact that PTCA should be the modality of choice in the treatment of STEMI for prevention of complications like LVFWR.

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