ORIGINAL ARTICLE

Frequency of Left Ventricular Systolic Dysfunction in Patients with Unstable Angina

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ABSTRACT

Objective: To determine the frequency of left ventricular systolic dysfunction (LVSD) in patients with unstable angina (UA).

Study design: Cross-sectional study.

Place & Duration: North ward of NICVD Karachi for six months from January to June 2014.

Material & Methods: A total of 113 cases were collected from all medical wards having history of first time unstable angina. After informed consent echocardiogram was done, the severity of left ventricular systolic dysfunction was determined on basis of ejection fraction. All the information was entered in a proforma designed for the study.

Results: Among 113 cases 65 (57.5%) were males. 52 (46%) patients had duration of symptoms of < 24 hours, 46 (40.7%) patients had symptoms on exertion, 48 (42.5%) patients had left ventricular dysfunction and 65 (57.5%) patients had normal left ventricular function (p=0.091). 23 (20.4%) patients had mild LV dysfunction, 14 (12.4%) had moderate LV dysfunction and 11 (9.7%) [8 males & 3 females] had severe LV dysfunction (p=0.380)

Conclusion: Less than half of study population had left ventricular systolic dysfunction. Preinfarction diagnosis of LVSD will help in the management of cases who will develop myocardial infarction.

Key Words. Unstable angina, Left ventricular Systolic Dysfunction, Acute coronary syndrome

INTRODUCTION

Pain is defined as a private, internal event that cannot be observed directly. Pain assessment is usually based on a person's self-report and it seems that pain is not uni-dimensional Left ventricular (LV) systolic dysfunction is a common complication of acute coronary syndrome (ACS). In case of ACS, LV systolic dysfunction may occur during the acute period or later.Unstable Angina (UA) and Non ST-segment elevation myocardial infarction (NSTEMI) are common heterogeneous disorders that involve widely different risks but have similar clinical presentation[1]. Overall, data suggest that the annual incidence of UA is higher than that of myocardial infarction (MI)[2]. It accounts for much of the morbidity and mortality of cardiovascular disease worldwide over the past two decades[3]. Left ventricular systolic dysfunction (LVSD) is a common and serious complication that leads to greatly increase in the risks of 2-3 folds for heart failure (HF) and death[4].Prevalence of LVSD after ACS is about 30 % to 40 %. However, in one study, incidence of LVSD after UA is 12 %[5].

Early risk stratification plays a pivotal role in the optimal management of unstable angina. Few recent randomized trials have shown a

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significant reduction in adverse cardiac events with an early invasive strategy in this group of patients[6]. The presence of LVSD has been associated with treatment disparities and worse outcomes in patients with STEMI, but little information is available for other ACS subsets, particularly unstable angina[7]. The rationale is that by determining the frequency of left ventricular systolic dysfunction (LVSD) we may be able to see the magnitude of patient's problem with unstable angina and thereby optimize the management, particularly coronary intervention and newer pharmacologic strategies that when used together, appear to have an important synergistic effect in reducing morbidity and mortality.

OPERATIONAL DEFINITION

Unstable angina (UA)

Patients were considered to have UA if they had following criteria:

a) Presence of one or more symptoms with or without exertion like chest, upper extremity, jaw, or epigastric discomfort, lasting at least 20 min or more.

b) New horizontal or down-sloping ST depression 0.05 mV in two contiguous leads.

c) Negative cardiac bio markers (Troponin-I).

LV systolic function:

LV systolic function was categorized according to the LV ejection fraction (EF).

- EF ≥55%: normal LV systolic function;
- EF 45%54%, 30% to 44 %,< 30%, mild, moderate, severe LV systolic dysfunction, respectively based on 2 dimensional method by using Transthoracic echocardiography.

Ejection fraction:

The fraction of blood contained in the ventricle at the end of diastole that is expelled during its contraction, i.e., the stroke volume divided by end-diastolic volume.

EF = (SV / EDV) X 100 where SV = stroke volume, EDV = end-diastolic volume.

Material & Methods:

This cross-sectional study was conducted in the North ward of NICVD Karachi for six months during January to June 2014. 113 patients were collected from all medical wards at National Institute of Cardiovascular Disease Karachi

Sample technique: Non-probability purposive.

Inclusion criteria:

. Male and female patients of UA meeting with above criteria for UA experiencing for the first time as stated in operational definition admitted in NICVD were included.

- Age >20 years.

Exclusion Criteria: following patients were excluded from the study:

- with known CAD.
- with known LV dysfunction.
- with Post CABG angina or MI.
- with Post PCI.
- with valvular heart disease.
- with pericardial disease.
- with age <20 years.
- with cardiomyopathies.
- refusal for study

Data Collection procedure: The patients were taken from medical wards of NICVD. Those patients who reported with chest pain and qualifying the inclusion criteria informed consent was taken, their first degree relatives or attendants for inclusion in the study. Finally echocardiogram was done by the post fellow of adult cardiology who have two years experience after his/her fellowship to determine severity of left ventricular systolic dysfunction on basis of EF so if it is > 55% was labeled as normal and if it was 45% to 54%, 30% to 44%, < 30%, mild, moderate, severe LV systolic dysfunction, respectively. The information was entered in the proforma attached as annexure.

Data Analysis: Data was analyzed by SPSS version 12. The frequency and percentages are calculated for Left ventricular systolic dysfunction (LVSD), gender and severity of LVSD (Mild, Moderate & Severe).

Mean± SD was computed for quantitative data like age, ejection fraction. Stratification was done with respect to age and gender, duration of symptoms with or without exertion to see the effect of these on outcomes.

RESULTS:

One hundred and thirteen patients fulfilling the inclusion criteria were included in this study. There were 65 (57.5%) males and 48 (42.5%) females. Mean ± standard deviation (SD) age of study population was 56.2 ± 12.38 years (Table I). Mean age of male patients was 55.3 ± 10.2 years and mean age of female patients was 57.3 ± 14.8 years (p=0.419) (Table I).

52 (46%) [31 males & 21 females] patients had duration of symptoms of < 24 hours, 32 (28.3%) [20 males & 12 females] patients of < 60 minutes and 29 (25.7%) [14 males & 15 females] patients had duration of symptoms of < 30 days (p=0.489).

Out of 113 patients 46 (40.7%) [31 males & 15 females] had symptoms on exertion and 67 (59.3%) [34 males & 33 females] had no any symptom on exertion (p=0.079).

Out of 113, 48 (42.5%) [32 males & 16 females] patients had left ventricular dysfunction and 65 (57.5%) [33 males & 32 females] patients had normal left ventricular function (p=0.091) (Table 2 & Figure. 1).

Table-1. Age Analysis among the Gender (n=113)

Gender	N	Mean	Std. Deviation
Male	65	55.3	10.2
Female	48	57.3	14.8

P=0.419

Table-2. Analysis of Echocardiographic Findings (n=113)

Gender	Echocardiographic findings		Total
	Left ventricular function normal	Left ventricular dysfunction	
Male	33	32	65
	50.8%	49.2%	100.0%
Female	32	16	48
	66.7%	33.3%	100.0%

P=0.091

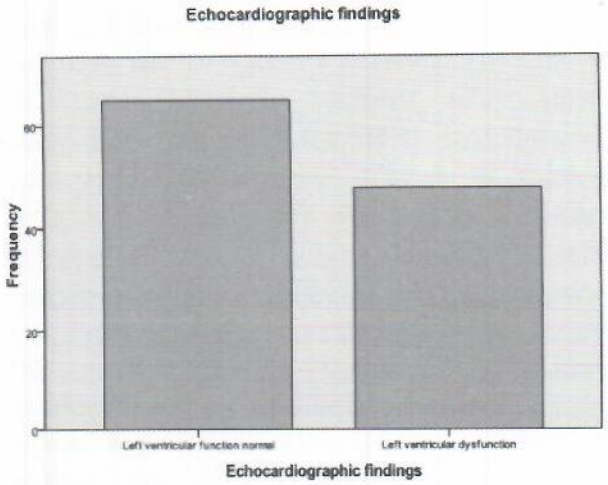

Fig-1. Analysis of Echocardiographic Findings (n=113)

DISCUSSION:

Most frequently LV systolic dysfunction is caused by ACS. The rate of the progression of LV systolic dysfunction and its clinical manifestation 'heart failure' is determined by the degree and duration of myocardial ischemia, stunning myocardium, the damaged area of the myocardium, the localization of the damage, the degree of infarct-related coronary artery lesion, and changes in the myocardium prior to ACS[8]. In the presence of myocardial changes, the course of LV systolic dysfunction is related to the myocardial remodeling process and its consequences, dilatation of cardiac chambers, mitral regurgitation, and diastolic dysfunction. LV systolic dysfunction is significantly influenced by changes in the myocardium (hypertensive or diabetic cardio-myopathy) caused by risk factors arterial hypertension and diabetes mellitus before ACS. Increased activity of neurohumoral factors (renin-angiotensin system, genome expression, and cell mediators endothelin and growth factor), as well as age, gender, and lifestyle are equally important for the development of LV systolic dysfunction and its sequelae[9].

It is obvious that the prognostication of the clinical course of LV systolic dysfunction, related to the aforementioned factors (some precipitating and some suppressing its progression), is complicated, as is the relationship of LV systolic dysfunction with heart failure. Martinez-Salles et al. Indicate that LV systolic dysfunction

following previous MI is a significant prognostic factor for the evaluation of heart failure and mortality[10]. However, in patients with ACS in whom LVsystolic dysfunction was detected during the acute period, the prognosis and the relationship of the condition with the development of heart failure (HF) cannot be determined based solely on the decreased LV EF. LV systolic dysfunction may develop within the first hours or within the first several days from the occurrence of ACS, may pass or persist, may be asymptomatic, & may manifest itself through acute HF or later on through chronic HF[11]. It has been indicated that systolic dysfunction (LV EF <40%) is detected in 40% of patients with MI and later on in 1.3-8.6% cases per year[12].

Although LV systolic dysfunction resulting in HF is a common complication of ACS, data on the prognostication of its later course are scarce. Most scientific publications focus on LV remodeling and HF at the same time evaluating LV systolic dysfunction[13-18].

LV systolic dysfunction during the unstable angina was detected in less than half (42.5%) of our studied patients. The lower incidence of LV systolic dysfunction, compared to that indicated by other authors, may be due to the difference in the studied contingents.

We found that the risk of the persistence and development of LV systolic dysfunction following ACS was not uniform. The instability of LV systolic dysfunction that develops during the acute period of ACS is another reason for the prognostication of LV systolic dysfunction that may develop later on.

The recovery in LVEF after one year in patients may have been conditioned by the impairment of LV function during the first days of ACS that occurred as a result of damage to certain segments of the myocardium, whose function subsequently improved after the normalization of blood flow in coronary arteries. In part of patients with remaining large-scale myocardial damage, failed or delayed normalization of blood circulation in the CA resulted in the myocardial function remaining low or decreasing further due to LV remodeling processes.

Gaudron and Gianuzi et al. indicated that progressing late LV remodeling that results in LV systolic dysfunction develops in one-fifth of patients who had MI[19]. According to Zhang et al., late remodeling of the myocardium occurs if MI involves more than 15% of the myocardium in case of anterior MI and more than 20% of the myocardium in case of inferior MI[20].

The most significant independent determinants of the acute period of ACS for the prognostication of late LV systolic dysfunction were used in a study: decreased LV systolic function (EF <40%), anterior Q wave MI, Killip class III-IV, frequent ventricular extrasystoles, pseudo-normal/restrictive LV dysfunction, and LV WMI>1.5. The determinants of CA stenoses and mitral regurgitation II-III that influenced LV systolic dysfunction were strongly correlated with the aforementioned determinants, but their informative value was lower and did not increase the accuracy of the model.

The determinants of the acute period of ACS are indicated by numerous researchers as determinants also having a prognostic value for the prognostication of the unfavorable course of the disease (LV remodeling, the development of chronic HF, and death) and used in the development of models for the prognostication of such events[21].

The determinants of the acute period of ACS, reflecting an unfavourable course of the disease, were evaluated in points, and a mathematical model was designed allowing for the prognostication of the risk for late LV systolic dysfunction. Our model for the prognostication of late LV systolic dysfunction during the acute period of ACS is simple and is based on standard clinical and echocardiographic findings; the scoring system prognosticates individual risk for late LV systolic dysfunction, the model has good sensitivity and specificity, and correct prognosis is made in the majority of cases. The selection of high-risk patients (in whom LV systolic dysfunction may either persist or develop during the later period) during the acute period of ACS may help in planning the treatment and close observation of such patients. This would decrease the risk of chronic HF and death.

CONCLUSION & RECOMMENDATIONS:

Preinfarction diagnosis of LVSD will help in the management of patients who will develop myocardial infarction.

We recommend that an extensive study should be done so that we can determine the exact frequency of patients with symptomatic and asymptomatic LVSD especially in relation to duration of disease, gender, compliance with treatment and follow up in patients with myocardial ischemia.

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