International Journal of Research in Medical Sciences
Verma VK et al. Int J Res Med Sci. 2014 Aug;2(3):1101-1106
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: 10.5455/2320-6012.ijrms20140892

Research Article

Echocardiographic assessment of the impact of cardiovascular risk factors on left ventricular systolic function in patients with acute myocardial infarction

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Received: 9 July 2014
Accepted: 20 July 2014

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ABSTRACT

Background: Objectives of current study were to determine the magnitude of left ventricular systolic dysfunction in patients with acute myocardial infarction in the rural sub-population of Uttar Pradesh in India and to evaluate the impact of cardiovascular risk factors on the risk of impairment of left ventricular systolic function.
Methods: One hundred and fifty seven consecutive patients with first acute myocardial infarction were enrolled into the study. Most patients were male (73.2%) and the mean age of presentation was 52.7 years. Two dimensional echocardiography was utilized to assess conventional parameters such as Left Ventricular End-Diastolic Diameter (LVEDD), Left Ventricular End-Systolic Diameter (LVESD), LV End-Diastolic Volume (LVEDV), LV End-Systolic Volume (LVESV) and Left Ventricular Ejection Fraction (LVEF). The LV volumes (end-systolic and end-diastolic) and LVEF were calculated from the conventional apical two-and four-chamber images using the biplane Simpson's technique. LV systolic function was considered depressed when LVEF was less than 45%. The chi-square test was used in the statistical analysis to compare proportions and a logistic regression model was used to assess the independent effect of the each variable.
Results: The study projects a high proportion (42.7% of the patient population) of left ventricular systolic dysfunction in patients with Acute Myocardial Infarction (AMI). No association was found between gender or age and LV systolic dysfunction. The proportion of patients with diabetes mellitus was higher in the sub-group of patients with impaired LV systolic function (45.2% vs. 30.2%, P = 0.01); the proportion of patients with history of current or past smoking was also higher in the sub-group of patients with impaired LV systolic function (48.9% vs. 34.2%, P = 0.03). On the other hand, hypertension and dyslipidemia were not associated with impaired LVEF. After adjustment of other variables, diabetes and smoking were associated with a significantly higher risk of LV systolic dysfunction (diabetes: OR = 3.73; 95% CI = 1.25-11.16; smoking: OR = 3.8; 95% CI = 1.37-11.05).
Conclusion: Since the proportion of patients with LV systolic dysfunction in patients with AMI remains relatively high, LV systolic function variables such as LVEF and LVESV should be echocardiographically evaluated in all patients with AMI. Since the post-infarction LV systolic function remains the single most important determinant of survival, treatment of AMI patients should be aimed at limitation of infarct size and prevention of ventricular dilation. Moreover, cardiovascular risk factors such as diabetes mellitus and smoking have a significant impact on the likelihood of impairment of LV systolic function in patients with AMI and hence could influence long-term prognosis.

Keywords: Acute myocardial infarction, Left ventricle, Ejection-fraction, Diabetes mellitus, Hypertension, Dyslipidemia, smoking, Cardiovascular risk factors.

INTRODUCTION

The prognosis in survivors of patients with AMI depends on multiple factors, which relate both to the acute event, such as infarct size, location, transmurality etc. as well as the characteristics preceding the infarction such as age, sex, cardiovascular risk factors such as diabetes mellitus, hypertension, dyslipidemia etc. and history of prior infarction.[1]

However, the single most important determinant of survival is post-infarction left ventricular systolic function.[2]

Various therapeutic modalities such as thrombolytic therapy and Primary Percutaneous Coronary Interventions (PPCI) aim at limitation of infarct size, thereby preserve LV systolic function and improve survival.

Other clinical variables such as ischemic preconditioning are associated with risk for adverse events following AMI. The occurrence of episodes of angina prior of AMI, a marker of ischemic preconditioning, reduces the risk of left ventricular remodeling and dilation.[3]

On the other hand, hyperglycemia on admission is associated with likelihood of significantly greater LV systolic dysfunction after AMI even after successful reperfusion therapy.[4]

Since post-infarction LV systolic function remains the key prognostic factor influencing both immediate and long-term mortality even in the era of primary PCI, determination of LV systolic function is vital for risk-stratification in patients with AMI to help formulate appropriate therapeutic strategy and measures to prevent further progression of LV systolic impairment and overt heart failure.[5]

Though, there are several studies conducted to address this issue in the urban patient sub-population both in India and abroad, to the best of our knowledge, no study is conducted so far to determine the impact of AMI and cardiovascular risk factors on impairment of LV systolic function on the rural sub-population of Uttar Pradesh in India.

Objective

The objective of our study was to determine the magnitude of left ventricular systolic dysfunction in patients with acute myocardial infarction in the rural sub-population of Uttar Pradesh in India and to determine the association between traditional cardiovascular risk factors (gender, age, smoking, hypertension, diabetes and dyslipidemia) and the likelihood of left ventricular systolic dysfunction following AMI.

METHODS

Subjects

This cross-sectional prospective study enrolled a cohort of 157 consecutive patients with Acute Myocardial Infarction (AMI) admitted to the emergency department and coronary care unit of Uttar Pradesh rural institute of medical sciences and research, Saifai, Etawah, Uttar Pradesh, India between July 2011 to March 2014. All of the patients included in the study fulfilled the inclusion criteria of "patients with AMI residing in the rural Uttar Pradesh."

Myocardial infarction was defined according to the standard criteria of typical rise and/or fall of biochemical markers of myocardial necrosis and with at least one of the following: a) ischemic symptoms; b) development of pathological Q waves in the serial ECG tracings; c) electrocardiographic changes indicative of ischemia (ST segment elevation or depression); d) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.[6] The term "acute" meant duration of symptoms of MI not more than seven days. Exclusion criteria were as follows: prior myocardial infarction; arrhythmias; inadequate 2D echocardiographic images for analysis; patients with AMI who were critically sick such as patients with cardiogenic shock and multi organ failure; significant associated valvular heart disease; concurrent presence of other life threatening co morbidities and patient's refusal to join the study.

Written informed consent was obtained from all patients; and the study protocol was approved by the ethics committee of the institute.

Clinical and laboratory measurements

a) History:

After clinical stabilization, the patients provided details of their demographic and social characteristics; history proper of disease; history of current or previous medications; relevant family history as well as past history and in depth enquiry into cardiovascular risk factor profile. Hypertension was defined as the presence of elevated systolic (>140 mmHg) and/or diastolic (>90 mmHg) blood pressure or the current use of antihypertensive drugs. A patient was considered smoker if he or she was smoking at the current moment or reported cessation less than six months in past. The criteria for the diagnosis of diabetes mellitus were Fasting Plasma Glucose (FPG) ≥126 mg/dl; or 2 hour post-prandial/OGTT plasma glucose ≥200 mg/dl; or classic symptoms of hyperglycemia and random plasma glucose ≥200 mg/dl or glycosylated hemoglobin (HbA_{1C}) ≥6.5%; or patient is on anti-diabetic therapy.[7]

As per NCEP/ATP3 guidelines, dyslipidemia was defined as serum total cholesterol levels of 200 mg/dl or more;

LDL-C more than 100 mg/dl; triglycerides equal to or more than 150 mg/dl or use of statin or other medications.[8]

b) Thorough general physical and cardiovascular examinations were carried out in all patients.

c) Laboratory measurements:

1. Routine investigations including FPG, 2HrPPG, KFT, LFT, electrolytes, urinalysis etc. were carried out in all patients.

2. The FPG and lipid profile samples were obtained after at least 12 hours of over- night fasting using the "Olympus All-400" auto-analyzer. The value of LDL-C was calculated using Freidwald's formula except when the serum TG concentration was more than 400mg/dl.

3. Glycosylated hemoglobin (HbA_{1C}) was measured by High Performance Liquid Chromatography (HPLC).

d) Diagnosis of Acute Myocardial Infarction (AMI):

Serum biochemical markers of myocardial damage such as creatine kinase MB, analyzed by enzyme immunoassay and cardiac specific troponins such as troponin-T (qualitative) and troponin-I (quantitative) and serial ECG tracings were obtained in all patients to facilitate diagnosis of AMI.

e) Transthoracic two-dimensional echocardiography:

All AMI patients were subjected to 2D ECHO within seven days of symptom of AMI. Patients were imaged in the left lateral decubitus position using commercially available equipment (Hitachi-Aloka). Standard images were obtained using a 3.5 MHz transducer, at a depth of 15 centimeter in the parasternal (long-and short-axis images) and apical (two-chamber and four-chamber images) views. Standard 2D ECHO and color-Doppler data, triggered to the QRS complex were saved to a cine-loop format. Measurements were averaged from at least three consecutive beats.

2D Echocardiography was used to assess conventional parameters such as LV End-Systolic Diameter (LVESD); LV End-Diastolic Diameter (LVEDD); LV End-Systolic Volume (LVESV); LV End-Diastolic Volume (LVEDV); LV Ejection-Fraction (LVEF); Wall Motion Score Index (WMSI); the mitral inflow peak early velocity (E)/mitral annular peak early velocity (E′) or E/E′ ratio.

The LV volumes (LVESV and LVEDV) and LVEF were calculated from the conventional apical two four chamber images using the biplane Simpson's technique. Impaired left ventricular systolic function was considered to exist when LVEF was estimated at less than 45%.

Statistical analysis of the data

For statistical analysis of the data, the chi-square test was used to compare proportions and the Student's t test to compare mean ages of the patients with depressed and preserved LV systolic functions. A logistic regression model, initially including all the variables studied, was subsequently adjusted to assess the independent effect of each variable. A confidence interval of 95% was used. A p value of less than 0.05 was considered to be statistically significant.

RESULTS

The study projects a high proportion of the patient population (42.7%) with impaired LV systolic function. All patients were managed conservatively. Approximately 65% of the patient population received reperfusion therapy in form of thrombolysis while approximately 35% of the patients received no perfusion therapy due to presence of contraindications to thrombolysis or due to delayed presentation.

Table 1: The clinical, demographic and biochemical characteristics of patients with or without depressed left ventricular systolic functions.

Variable	Normal LV syst. function group	Depressed LV syst. function group	P value
n (%)	90 (57.3%)	67 (42.7%)	ns
Male gender (n) (%)	66 (42.03%)	49 (31.21%)	ns
Age (years)	50.2 ± 10.4	52.7 ± 11.3	ns
IHD risk factors: n (%)			
HTN	32 (35.56%)	34 (50.74%)	ns
DM	34 (37.78%)	37 (55.22%)	0.01
Smoking	48 (53.33%)	47 (70.15%)	0.01
Dyslipidemia	27 (30%)	25 (37.31%)	ns
Infarct location			
Anterior	28 (31.11%)	27 (40.3%)	0.001
Other	62 (68.88%)	40 (59.7%)	ns
Q-wave MI	28 (31.11%)	27 (40.3%)	0.001
Biochemical parameters			
Trop-I (µg/ml)	11.4 (2.9; 31.6)	17.6 (3.4; 35.4)	0.03
WBC (cells/l)	11.4 ± 3.4	12.2 ± 3.6	ns
CRP (mg/l)	10.58 (2.2; 25.1)	11.2 (2.1; 25.8)	ns
Reperfusion strategy			
Thrombolysis: yes	58 (64.44%)	45 (67.16%)	0.05
Thrombolysis: no	32 (35.56%)	22 (32.84%)	ns
Primary PCI	None	None	-

IHD=ischemic heart disease; HTN=hypertension; DM= diabetes mellitus; ns=non-significant; CRP= c-reactive protein; PCI=per-cutaneous coronary interventions; Trop-I=troponin-I; MI=myocardial infarction. A P value of less than 0.05 was considered to be statistically significant

The demographic, clinical and biochemical characteristics of the patient population studied are presented in Table 1.

Univariate analysis

No association was found between risk for left ventricular systolic dysfunction and gender or age. With regard to cardiovascular risk factors studied, univariate analysis showed that the proportion of patients with diabetes mellitus was higher in the sub-group of patients with depressed LV systolic functions (45.2% vs. 30.2%, P = 0.01); the proportion of patients with current or past smoking was also higher in the sub-group of patients with depressed LV systolic function (48.9% vs. 34.2%, P = 0.03).

On the other hand, hypertension and dyslipidemia were not associated with impairment of LV systolic function. Patients with anterior wall STEMI were associated with a significant higher risk for depressed LV systolic function compared to patients with STEMI of other locations. Among the conventional 2D echocardiographic parameters studied, LVESV was most significantly associated with sub-group of patients with impaired LV systolic functions (Table 2).

Table 2: Conventional 2-dimensional echocardiographic parameters in patients with and without depressed left ventricular systolic functions.

Variable	Normal LV systolic function	Depressed LV systolic function	P value
n	90 (57.3%)	67 (42.7%)	-
LVEF (%)	55.2 ± 7.6	38.2 ± 4.2	<0.05
LVEDD (mm)	47.4 ± 4.5	50.2 ± 6.1	<0.05
LVESD (mm)	32.5 ± 4.7	38.2 ± 4.8	<0.01
LVEDV (ml)	86.4 ± 19.3	104.2 ± 28.5	<0.05
LVESV (ml)	45.2 ± 13.1	52.2 ±1 9.8	0.001
LA (mm)	39.8 ± 4.7	40.3 ± 5.6	ns
E/E′	13.2 ± 2.6	15.4 ± 3.8	ns
WMSI	1.65 ± 0.32	1.49 ± 0.31	ns

LVEF=left ventricular ejection fraction; LVEDD=left ventricular end-diastolic dimension; LVESD=left ventricular end-systolic diameter; LVEDV=left ventricular end-diastolic volume; LVESV=left ventricular end-systolic volume; LA=left atrium; WMSI=wall motion systolic index

Multivariate analysis

The results of multivariate analysis are presented in Table 3a and table 3b. After adjustment of other variables, diabetes mellitus and smoking were significantly associated with the higher risk of impairment of LV systolic function (Diabetes: OR 3.73, 95% CI 1.25-11.16; Smoking: OR 3.9, 95% CI 1.37-11.07).

Table 3a: Cardiovascular risk factors and depressed ejection fraction: multivariate analysis.

Variable	OR	95% CI
Females	0.97	0.32-2.91
Age 65 years	0.92	0.29-2.76
Diabetes mellitus	4.72	1.43-15.72
Hypertension	0.38	0.11-1.01
Dyslipidemia	0.34	0.13-0.92
Smoking	3.78	1.06-14.21
Anterior wall STEMI	5.65	2.17-14.9

OR=Odd′s ratio; CI=confidence interval; STEMI=ST segment elevation myocardial infarction

Table 3b: Factors independently associated with depressed ejection fraction.

Variable	OR	95% CI
Diabetes mellitus	4.12	1.42-12.42
Smoking	6.17	1.53-16.1
Anterior wall STEMI	3.8	1.03-9.8

OR=Odd′s ratio; CI=confidence interval

DISCUSSION

The present study projects a high proportion of patients with depressed LV systolic function who experienced index AMI. Though, the findings of this study are in accordance with other studies conducted elsewhere;[6,7] it is not possible to apply these findings directly to the rural sub-population of Uttar Pradesh because our study being conducted in a tertiary care referral centre; data could be weak representative of the true incidence of impaired LV systolic function amongst the patients with AMI in this population.

Excluding patients with previous myocardial infarction or significantly associated valvular heart disease from the study meant that the depressed LVEF observed could be attributed to the index coronary event, by eliminating patients with possible pre-existing left ventricular dysfunction of ischemic origin or related to valvular heart disease.[8,9] However, the lack of long-term follow-up of the patients studied prevents complete clarification of the relationship between the results obtained for in-hospital left ventricular systolic function and prognosis after discharge. Further studies are needed to clarify this issue.

The presence of previous cardiovascular risk factors has a significant impact on the likelihood of left ventricular systolic dysfunction following index acute coronary event (AMI).[10,11] A history of diabetes mellitus has been consistently linked with greater morbidity and mortality; both in-hospital and post-discharge. Diabetes mellitus contributes to impairment of endothelium dependent vasodilatation, vascular smooth muscle dysfunction, increased arterial stiffness and rapidly advancing

atherosclerosis. Polydistrectual atherosclerotic macroangiopathy in diabetic subjects usually leads to more diffuse involvement of multiple coronary arteries and other vascular beds as compared to non-diabetic subjects; thereby contributing to likelihood of significantly greater impairment of LV systolic function in diabetic patients in comparison to non-diabetic patients following AMI.[12,13]

Other than advanced age, smoking is the single most important risk factor for coronary artery disease and has a major impact on myocardial infarction and all-cause mortality. Beyond acute unfavorable effects on blood pressure and sympathetic tone, and a reduction in myocardial oxygen supply; smoking affects atherothrombosis through several other mechanisms. In addition to accelerating atherosclerosis progression, long-term smoking may enhance the oxidation of LDL-C and impair endothelium dependent coronary artery vasodilatation.[14] In addition, smoking has adverse hemostatic and inflammatory effects, including increased levels of fibrinogen, homocysteine, Plasminogen Activator Inhibitor-1 (PAI-1), CRP and soluble Inter-Cellular Adhesion Molecule-1 (ICAM-1). Chronic smoking, results in left ventricular morphological alterations consistent with LV remodeling, characterized by an increase in both end-systolic and end-diastolic dimensions; thereby resulting into alteration in cardiac geometry, volume, mass and myocardial constituents in response to alteration in loading conditions.[15] In an experimental study, rats exposed to chronic cigarette smoke exhibit left ventricular remodeling process which was ultimately accompanied by a significant decrease in the left ventricular systolic function assessed by the use of fractional-shortening and ejection-fraction.[16]

Although elderly and female patients have a worse prognosis after AMI, no association was found between age or gender and risk of left ventricular systolic dysfunction; these results are similar to those reported by Ottervanger et al.[17] This suggests that the increased mortality observed in elderly and females could be related to other factors and not to impaired LV systolic function per se.

CONCLUSION

Since the proportion of patients with LV systolic dysfunction in patients with AMI remains relatively high, LV systolic function variables such as LVEF and LVESV should be echocardiographically evaluated in all patients with AMI. Since the post-infarction LV systolic function remains the single most important determinant of survival, treatment of AMI patients should be aimed at limitation of infarct size and prevention of ventricular dilation. Moreover, cardiovascular risk factors such as diabetes mellitus and smoking have a significant impact on the likelihood of impairment of LV systolic function in patients with AMI and hence could influence long-term prognosis.

Abbreviations

AMI (acute myocardial infarction); LV (left ventricle); LVEF (left ventricular ejection-fraction); LVESV (left ventricular end-systolic volume); LVEDV (left ventricular end- diastolic volume); LVEDD (left ventricular end- diastolic diameter); LVESD (left ventricular end-systolic diameter).

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the institutional ethics committee

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DOI: 10.5455/2320-6012.ijrms20140892
Cite this article as: Verma VK, Rawat R, Sagar V, Kumar G. Echocardiographic assessment of the impact of cardiovascular risk factors on left ventricular systolic function in patients with acute myocardial infarction. Int J Res Med Sci 2014;2:1101-6.