Assessment of left ventricular long axis contraction in patients with ischemic mitral regurgitation after acute myocardial infarction

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KEYWORDS
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Abstract
Introduction: The development of ischemic mitral regurgitation (IMR) after myocardial infarction (MI) may impose hemodynamic load during a period of active left ventricular remodeling and promote heart failure (HF). The aim of our study was to evaluate left ventricular (LV) long axis contraction assessed by both mitral annular plane systolic excursion (MAPSE) and peak systolic velocity (Sa) in patients with ischemic MR after acute MI.

Methods: Thirty-eight patients with a first attack of acute MI were classified into two groups. Group I comprised 18 patients with MI and ischemic MR, and group II comprised 20 patients with MI without IMR. Twenty age-matched subjects were considered as the control group (group III). Measurement of MAPSE from M-mode tracing of the mitral annulus in apical 4- and 2-chamber view, and pulsed wave tissue Doppler imaging (PW-TDI) of the 4 sides of the mitral annulus for assessment of the Sa velocity were done.

Results: A significant decrease of MAPSE was observed in 4 sides in patients with acute MI with IMR compared to MI without IMR and control group (P < 0.05). Peak systolic velocity (Sa) in septal, anterior, and inferior sides of mitral annulus was significantly decreased in MI patients compared to control group (P < 0.05). A significant correlation between MAPSE on anterior side of mitral annulus and LV ejection fraction (P < 0.001) in patients with ischemic MR after acute MI was found.
Introduction

The association between ischemic mitral regurgitation (IMR) either secondary or functional and poor prognosis in relation to morbidity and mortality after myocardial infarction (MI), chronic heart failure, percutaneous or surgical revascularization is well known [1].

Expansion of infarcted tissue begins acutely after MI. A more gradual remodeling process, however also involves the non-infracted areas [2]. Myocardial infarction causes MR by altering ventricular geometry and function [3]. Ischemic MR doubles the risk of death after MI [4].

It has been well recognized that LV systolic function is a major predictor of outcome after acute MI [5]. Assessment of LV ejection fraction (LVEF) after MI is difficult because of poor endocardial border definition and is often time consuming and poorly reproducible [6]. Wall motion score index (WMSI) is an alternative to LVEF which also reflects regional systolic function. However, the assessment of WMSI is semi-quantitative and experience dependent [7].

Mitral annular velocities can readily be recorded by pulsed wave TDI after MI. The reduced peak systolic velocity seems to be an expression of regionally reduced systolic function and correlates well with LVEF [8]. Using the ratio of early trans-mitral flow velocity to the early mitral annular velocity (E/e), a close approximation of LV filling pressure can be obtained in wide spectrum of patients [9]. The E/e is superior to other echocardiographic indices in this respect [10]. After acute MI, an elevated E/e ratio predicts higher mortality and an increased risk of adverse remodeling [11].

Mitral annular plane systolic excursion (MAPSE) has been considered a reliable method for the assessment of LV longitudinal function and correlates with global systolic function of the LV [12].

Aim of the work

The aim of our study was to evaluate left ventricular (LV) long axis contraction assessed by both MAPSE and peak Sa in patients with ischemic MR after acute MI.

Patients and methods

Study population

The study test group comprised 38 patients with a first acute MI admitted to coronary care unit in Mansoura Specialized Hospital, Mansoura University, in the period between March 2012 and February 2013.

Patients included in this study were subsequently classified into two main groups according to the presence of MR clinically and by echocardiography.

Group I: comprised 18 patients with acute MI and ischemic MR, mean age 64.2 years (11 males and 7 females).

Group II: comprised 20 patients with acute MI without ischemic MR, mean age 60.9 years (14 males and 6 females).

The diagnosis of AMI was based on the detection of a rise and/or fall of cardiac biomarker values and with at least one of the following: Symptoms of ischemia, new or presumed new significant ST-segment changes, development of pathologic Q waves in the ECG, imaging evidence of new loss of viable myocardium or new regional wall motion abnormality, and identification of an intracoronary thrombus by angiography [13]. All the patients were treated by thrombolysis (streptokinase 1,500,000 unit); none received primary percutaneous coronary intervention, which was not in routine use at our hospital.

Twenty age-matched subjects with no evidence of coronary artery disease were considered as the control group (group III).

An informed consent was taken from all the patients and the protocol was approved by our institute research plan.

Exclusion criteria

Patients with previous history of MI, chronic heart failure, valvular heart disease, atrial fibrillation, conduction abnormalities, and severe (more than grade II) MR were excluded from the study.

History and clinical examination

Patients and controls were subjected to:

(a) Thorough history taking with special stress on age, sex, ischemic chest pain, dyspnea, orthopnea, and risk factors for ischemic heart disease.

(b) Clinical examination including general examination for pulse, blood pressure, edema lower limb, congested neck veins. Local cardiac and chest examination for murmur of ischemic MR, gallop and bilateral basal rales.

Laboratory investigation

Routine laboratory investigation and cardiac enzymes were done for all patients.

Electrocardiography

Standard 12-lead ECG was analyzed for site and extent of MI.

Echocardiography

Patients and controls were examined at rest in supine left lateral decubitus positions using GE (vivid 3 pro) Norway using 2.5 MHz phased array transducer following the recommendation of the American Society of Echocardiography.

M-mode and two dimensional echocardiography was done for measurements of both LV and left atrial (LA) dimensions...
and the presence of wall motion abnormality. LV ejection fraction was calculated using the biplane method according to the modified Simpsons rule [7].

Ischemic mitral regurgitation is a valvular incompetence associated with myocardial ischemia or infarction in the absence of primary leaflet or chordal pathology [14]. Mitral regurgitation was categorized using mapping of regurgitant jet expansion in the LA in apical 4 and 2-chamber views at end systole from 3 separate cardiac cycles [15]:

- Mild, when regurgitant jet area > 5% and < 20% of the LA area.
- Moderate when regurgitant jet area > 20% and < 40% of the LA area.
- Severe, when regurgitant jet area > 40% of the LA area.

**Echocardiographic procedure of long axis function**

To assess long axis function, pulsed-wave TDI and M-mode were performed. The longitudinal motion was obtained from apical four and two chamber view with the cursor positioned from apex to the insertion of the atrioventricular plane. At least three cardiac cycles were analyzed and averaged.

Mitral annular plane systolic excursion (MAPSE) was measured from four sites of the atrioventricular plane corresponding to the septal, lateral, anterior, and posterior walls using the apical four- and two-chamber views by M-mode echocardiography. The M-mode cursor was aligned parallel to the LV walls. The systolic excursion of mitral annulus was measured from the lowest point at end-diastole to aortic valve closure. (maximum systolic displacement; Fig. 1) [16,17].

Pulsed wave TDI of different 4 sides of the mitral annulus in apical 4- and 2-chamber views was obtained for measurement of the peak systolic (Sa) and diastolic (Ea and Aa) velocities. E/Ea ratio was obtained to reflect noninvasive estimate of LV filling pressure [18].

**Statistical analysis**

All data were analyzed using a SPSS software package (version 17.0, SPSS Inc., Chicago, Illinois, USA). Continuous variables were expressed as mean ± SD, and categorical variables were expressed as percentage. Comparisons between the different groups were performed using the unpaired Student’s t test (for normally distributed variables) and Mann–Whitney U test (for non-normally distributed variables). Pearson correlation coefficients were used to explore relationships between the MAPSE and EF, Sa, E/Ea. P value of < 0.05 was considered statistically significant.

**Results**

Table 1 represents clinical characteristics of study groups. The mean age of patients with acute MI and IMR was 64.2 years, 61.1% were males, 50% were in Killip class III, 72.2% had hypertension, 38.2% had diabetes mellitus, 22.2% had anterior MI, and S3 gallop was present in 16 patients. Patients with acute MI without MR had a mean age of 60.9 years, 70% were males, 20% were in Killip class III, 55% had hypertension, 35% had diabetes mellitus, 30% had anterior MI, and S3 gallop was present in 2 patients.

Table 2 shows a significant decrease of MAPSE in 4 sides in patients with acute MI with IMR compared to group II (MI without MR) and control group (P < 0.05). There was a significant decrease of MAPSE in lateral side of mitral annulus in group II (MI without MR) compared to control group (P 0.007), while there was no significant difference of MAPSE in septal, anterior, and inferior sides of mitral annulus in MI without MR compared to control group. A significant decrease

![Fig. 1 Lateral mitral annular plane systolic excursion.](image-url)
of LV EF was observed either in group I or group II compared to the control group. A significant increase of LA diameter was observed in group I (acute MI with IMR) when compared with group II or III ($P < 0.05$).

Table 3 revealed a significant decrease of peak systolic velocity (Sa) in septal, anterior, and inferior sides of mitral annulus when comparing group I (MI with IMR) and group II (MI without MR) with control group ($P < 0.05$), while there was no difference on the lateral side of the mitral annulus ($P > 0.05$). A significant increase of $E$/sep $Ea$ ratio was observed in groups I and II compared to the control group ($P < 0.001$) and this ratio increased in group I (MI with IMR) when compared with group II (MI without MR) but not statistically significant ($P = 0.318$).
Table 4 shows a significant correlation between MAPSE on anterior side of mitral annulus and LV EF ($P = 0.001$) in patients with acute MI with or without MR (Fig. 2).

**Discussion**

The development of ischemic mitral regurgitation (IMR) after MI may impose hemodynamic load during a period of active left ventricular remodeling and promote heart failure (HF) [19]. The intriguing observation that even mild MR was associated with an increase in the risk for HF is not easily explained. It has been postulated that mild ischemic MR is a marker of geometric abnormalities that lead to progressive remodeling and increased load on the non-infarcted myocardium [20].

![Fig. 2](image-url)  
**Fig. 2** Correlation of MAPSE on the anterior side with LVEF.

The assessment of LV function in patients with myocardial infarction complicated by MR is not completely understood. Although MR is more common in patients with LV dysfunction, the relation between MR grade and LVEF is not linear as unloading of the ventricle tends to increase LVEF [21].

The assessment of the velocity and the amplitude of LV long-axis motion by echocardiography are more sensitive than simple determination of LVEF [22]. The basal portion of the ventricle is connected with longitudinal subendocardial muscle fibers that are sensitive to ischemia and the increase in wall stress [23].

In the present study, MAPSE was significantly decreased in patients with acute MI and IMR compared to those without MR and control group. This reflects more impairment of LV longitudinal function in patients with MI and IMR, and provides complementary information to LV ejection fraction (EF), which represents the global result of both longitudinal and circumferential contraction. Long axis shortening (from longitudinal fibers) occurs before short axis shortening during the isovolumic contraction phase, whereas the ejection phase is mainly due to shortening of circumferential fibers in the short axis [24]. Therefore, when subendocardial ischemia occurs, impaired long axis shortening is evident before changes in the short axis because of the orientation of the myocardial fibers, and this explained the borderline significant difference between the two groups of acute MI as regards the LVEF in this study.

The main cause of functional MR is augmented leaflet tethering by apical displacement of the papillary muscle (PM) because of LV remodeling [1]. The subendocardial fibers are aligned longitudinally and are connected with the mitral annulus; therefore, the measurement of MAPSE reflects impairment of LV longitudinal shortening and relaxation [23]. So the presence of MR at baseline is associated with worse LV function, greater LV enlargement and chamber distortion, increased mitral annular area and worse mitral annular contraction.
On the other hand, patients with acute MI without MR had no difference in MAPSE in septal, anterior, and inferior sides with only a significant decrease in the lateral side. This is attributed to the fact that MAPSE are higher in the lateral side in normal individuals than other MAPSE [16].

Left ventricular long axis function assessed by pulsed wave TDI peak systolic velocity was not different between both MI groups. This raises the superiority of MAPSE over tissue Doppler peak systolic velocity for evaluation of LV longitudinal function in patients with acute MI. This finding is further reinforced by the presence of positive correlation of the MAPSE of the anterior side with LV EF. MAPSE represents the amount of displacement of the mitral annular plane toward the apex and thus assesses the global change in size of the LV cavity (in the long-axis direction). Thus, it can be interpreted as the volume change during ejection with a close association between the long-axis shortening and ejection fraction (EF) [25]. Longitudinal function is altered before radial/circumferential indices change, [26] therefore MAPSE could be used as a simple and sensitive echocardiographic index to assess myocardial abnormalities involving predominantly longitudinal changes, especially in early phase. Tsang et al. [27] propose that speckle-tracking-derived mitral annular displacement is a clinically useful tool for rapid, accurate, and robust estimates of LVEF irrespective of LV endocardial definition. Alam observed an association between MAPSE and LV systolic function in patients with first-time acute myocardial infarction. They showed a decreased MAPSE in MI patients compared with controls and a more pronounced reduction at the sites of infarction [28].

We did not find a significant correlation between MAPSE and either Sa, E/Ea, which may be attributed to the small sample volume. Willenheimer et al. found that patients with advanced LV diastolic filling abnormalities had lower MAPSE than patients with preserved diastolic filling despite similar fractional shortening, and suggested MAPSE could be as well reflect LV diastolic function [29].

In our study there was no significant difference in E/Ea between both MI groups. This indicates that the rise in diastolic pressures is similar in the early phase of MI between both groups. There are conflicting data on the accuracy of E/Ea as a surrogate of LV filling pressure in patients with functional MR. Some investigators reported that E/Ea ratio accurately predicts LV filling pressure in patients with functional MR [30,31], whereas others found a poor correlation [32]. Mitral annular plane systolic excursion is a simple and sensitive echocardiographic parameter for assessing longitudinal LV function in acute MI patients with IMR. Although good imaging quality is required for most of the modern echocardiographic techniques for reasonable interpretation of LV systolic function, MAPSE are measurable in the majority of patients quiet independent of imaging quality.

Study limitations

The present study has several limitations. First, Ischemic MR is a dynamic lesion, and its severity may vary over time because of ongoing ventricular remodeling. The severity of MR in the present study was assessed only in the early post infarction period. Location of MI could affect mitral annular velocity. Determinants of survival in patients with secondary MR include quantitative measures of MR, such as regurgitant volume and the effective regurgitant orifice, which were not contemplated in this study.

Conclusion

MAPSE, reflecting longitudinal myocardial shortening, is a useful and superior parameter over peak Sa for assessment of longitudinal LV function in patients with ischemic MR after MI.

Conflict of interest

None declared.

References


