In Patients with Post-Infarction Left Ventricular Dysfunction, How Does Impaired Basal Rotation Affect Chronic Ischemic Mitral Regurgitation?

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Background: The aim of this study was to explore the contribution of left ventricular (LV) basal rotation to the mechanism of chronic ischemic mitral regurgitation (MR).

Methods: Fifty-seven patients (52 men; mean age, 68.3 ± 11.8 years) with postinfarction LV dysfunction (defined as an ejection fraction ≤45%) were prospectively enrolled. Each invariably had functional MR. To assess MR degree, the effective regurgitant orifice area (EROA) was quantified by echocardiography using the proximal isovelocity surface area method. Furthermore, mitral valve deformation (valve tenting and annular function) and LV global (systolic and diastolic volumes, function, and sphericity) and local remodeling (displacement of papillary muscles, regional strain, and rotation by speckle-tracking) were assessed. The patients were subsequently subdivided into two groups according to the absence (group A) or presence (group B) on transthoracic echocardiography of infarct area in the inferior and/or posterior basal segments.

Results: A larger EROA was found in group B than in group A ($P = .034$) and in subjects with asymmetric rather than symmetric tethering in either group ($P = .036$ and $P = .040$ for groups A and B, respectively). Basal radial ($P = .009$), circumferential ($P = .042$), and longitudinal ($P = .005$) strain and rotation ($P = .021$) were lower in group B than in group A. There was also a significant inverse correlation between EROA and basal rotation in group B ($r = -0.75$, $P < .001$). Furthermore, using multivariate linear regression analysis, we found that the independent determinants of EROA were end-diastolic volume ($P < .001$) and tenting area ($P = .004$) in group A and asymmetric tethering ($P = .029$) and basal rotation ($P < .001$) in group B.

Conclusions: Impaired basal rotational mechanics occurring after an inferior-posterior myocardial infarction is associated with increased MR. (J Am Soc Echocardiogr 2013;■:■-■.)

Keywords: Chronic ischemic mitral regurgitation, Myocardial infarction, Echocardiography, Basal rotation

Chronic ischemic mitral regurgitation (MR) is a common finding in patients with postinfarction left ventricular (LV) dysfunction and is independently associated with reduced life expectancy.[1,2] Several coexisting pathophysiologic mechanisms are independently involved in MR pathogenesis, including LV systolic dysfunction in itself, LV local and global remodeling, LV dyssynchrony, annular shape, and mechanical alterations.[3-5] However, the imbalance between the closing and tethering forces acting on the mitral valve (MV) leaflets, particularly asymmetric tethering, has been considered to be the main determinant of MR severity.[6] Furthermore, for several years, inferior myocardial infarction (MI) has been recognized as the most frequent cause of ischemic MR because of the geometric distortion in the papillary muscle (PM)–bearing segments.[7,8] In addition, the integrity of LV basal function also plays a role in decreasing ischemic MR.[7,8] Furthermore, the restricted sphincter motion of the mitral annulus (MA) after an inferior or posterior MI is a cofactor that can modulate the degree of ischemic MR.[9] Therefore, the site of post-MI LV remodeling might be a more important determinant of MR degree in LV dysfunction than the extent of post-MI LV remodeling. Recent advances in cardiac imaging techniques, such as speckle-tracking echocardiography, have provided new insights into LV mechanics after MI.[10-14] However, the involvement of impaired basal rotation in the pathophysiology of MR has not been fully evaluated. In the present study, we sought (1) to investigate whether basal rotational mechanical failure is associated with increased MR and (2) to provide a hypothesis for the pathophysiologic mechanism by which basal rotation affects MR.

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METHODS

Study Population

From December 2010 to December 2011, we prospectively studied 80 consecutive patients referred for conventional echocardiography for previous MI.

Abbreviations
BSA = Body surface area
CS = Circumferential strain
EF = Ejection fraction
EROA = Effective regurgitant orifice area
LS = Longitudinal strain
LV = Left ventricular
MA = Mitral annulus
MAA = Mitral annular area
MI = Myocardial infarction
MR = Mitral regurgitation
MV = Mitral valve
PISA = Proximal isovelocity surface area
PM = Papillary muscle
RS = Radial strain
WM = Wall motion

Of the 80 patients, 57 (52 men; mean age, 68.3 ± 11.8 years) with systolic LV dysfunction, defined as an ejection fraction (EF) ≤ 45% and any degree of functional MR, were selected for the present study.

Twenty-three patients were excluded for one or more of the following reasons: (1) clinical and echocardiographic evidence of other cardiac and/or heart valve disease (five patients), (2) previous coronary artery bypass graft surgery (three patients), (3) morphologic abnormalities of the MV apparatus (two patients), (4) acute recurrence of MI (within the past 2 weeks; one patient), (5) atrial fibrillation (four patients), (6) left bundle branch block (three patients), (7) technically inadequate color flow Doppler images for the proximal isovelocity surface area (PISA) method (i.e., markedly eccentric regurgitant jets and/or multiple regurgitant orifices for which the feasibility of the PISA method would have been poor; two patients), and (8) technically inadequate two-dimensional echocardiographic images for speckle-tracking echocardiographic analysis (three patients).

All patients underwent preliminary cardiologic examinations with comprehensive clinical data collection, including cardiovascular risk factors, documented history of prior transmural ST-segment elevation MI,[15] and current medication use. Moreover, particular care was taken to ensure that all of the patients met the recommended diagnostic criteria for MR, which can be summarized as follows: MR occurring >16 days after MI with one or more LV segmental wall motion (WM) abnormalities, significant coronary disease in a territory supplying the WM abnormalities, and structurally normal MV leaflets and chordae tendinae.[3]

Two-Dimensional Echocardiography

LV Remodeling and Function. Each subject underwent standard transthoracic echocardiogram using Vivid 7 echocardiography equipment (GE Vingmed Ultrasound AS, Horten, Norway). The parameters of global LV remodeling, including LV end-diastolic volume (EDV) and end-systolic volume and biplane Simpson's EF, were measured.[16] Body surface area (BSA) was calculated using the Du Bois formula, and LV volumes were indexed accordingly. The sphericity index, which represents LV shape, was defined as the short-axis/long-axis dimension ratio in the end-systolic apical four-chamber view.[17] A qualitative assessment of regional WM was made using the 16-segment model of the American Society of Echocardiography,[16] and the WM score index was then calculated. Because previous clinical studies have validated the accuracy of the E/e′ ratio for predicting LV filling pressure in ischemic MR and prognosis after MI,[18-20] we used this parameter as an estimate of LV diastolic function.[21] For this purpose, e′ was derived from the average of the velocities of the septal and lateral MA using spectral Doppler tissue imaging.[22] The mitral deceleration time and E/A ratio were also recorded for each patient as markers of diastolic function.[21]

Identification of Infarct Area and Definition of the Subgroups. To detect the contribution of LV basal rotation in determining MR degree, the study population was divided into two groups according to the site and extent of the infarct area. The latter was defined as a myocardial region with segmental WM abnormality (hypokinesia, akinesia, or dyskinesia) associated with increased brightness and/or reduced wall thickness on transthoracic echocardiography.[23] Patients with infarct areas only in the anterior LV segments (apex, anterior wall, and/or anterior septum) were included in group A ($n = 26$), whereas those with infarct areas in the anterior and inferoposterior segments (apex, anterior wall, and/or anterior septum plus the mid to basal inferior, posterior wall, or inferoseptum) were included in group B ($n = 31$). No patient with either an isolated anterior or inferior-posterior infarct and an LV EF ≤ 45% was observed.

MR Quantification and MV Deformation Analysis. To estimate the degree of MR, the effective regurgitant orifice area (EROA) was calculated according to the formula from the PISA.[24-26]

To assess MV geometric deformation, the coaptation depth, defined as the distance between the point of leaflet coaptation and the MV annular plane, and the tenting area, calculated as the area subtending the annulus and the MV leaflets, were measured in the parasternal long-axis view in the mesosystolic phase of the cardiac cycle.[3,24] The pattern of tethering was also evaluated, and an asymmetric pattern was diagnosed when a predominant posterior tethering of both leaflets with the anterior leaflet overriding superior to the posterior one was present and also showed a "hockey stick configuration," whereas the symmetric pattern was defined by a predominant apical tethering of both leaflets.[3] To evaluate the sphincteric function of the posterior MA, a long-axis apical view was used, whereas the corresponding orthogonal commissure-commissure plane measurement was made using the apical four-chamber view at the junction of the leaflet and left atrium, as recently recommended.[27] The end-diastolic and end-systolic diameters of the MA were calculated,[28,29] and the respective MA areas (MAAs) were obtained using the following equation: $MAA = (\pi r_1 r_2)/4$, where r_1 and r_2 are the diameters of the ellipse.[2,29] Finally, MA contractility was derived as the percentage area reduction according to the following formula: mitral annular contractility = (end-diastolic MAA − end-systolic MAA)/(end-diastolic MAA) × 100%.[30]

The outward displacement of PMs was calculated as the lengths between the anterolateral PM and posteromedial PM tips and the contralateral anterior MA in midsystole in the apical four-chamber and two-chamber views; the anterior MA was used as a reference point.[31]

Global and Regional LV Strain and Rotational Mechanics. For offline analysis of strain and rotation, LV short-axis views, acquired at the basal, mid, and apical levels, and standard LV apical four-chamber, three-chamber, and two-chamber views were recorded with a mean frame rate of ≥70 frames/sec. The two-dimensional strain and rotation data were analyzed by frame-to-frame tracking of the grayscale patterns using dedicated software (EchoPAC version 7.0.0; GE Vingmed Ultrasound AS); Automated Function Imaging (GE Vingmed Ultrasound AS) was used to evaluate longitudinal strain (LS). Both the strain and rotation peaks were measured during the end-systolic frame of the cardiac cycle. LV rotation was calculated from the apical and basal short-axis images, with counterclockwise rotation marked as a positive value and clockwise rotation as a negative value.[11]