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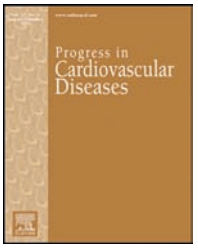

Assessment of the severity of native mitral valve regurgitation

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ABSTRACT

Mitral regurgitation (MR) is a major cause of cardiovascular morbidity and mortality. MR is classified as primary (organic) if it is due to an intrinsic valve abnormality, or secondary (functional) if the etiology is because of remodeling of left ventricular geometry and/or valve annulus. Transthoracic echocardiography (TTE) is the initial modality for MR evaluation. Parameters used for the assessment of MR include valve structure, cardiac remodeling, and color and spectral Doppler. Quantitative measurements include effective regurgitant orifice area, regurgitant volume, and regurgitant fraction. Knowledge of advantages and limitations of echo-Doppler parameters is essential for accurate results. An integrative approach is recommended in overall grading of MR as mild, moderate, or severe since singular parameters may be affected by several factors. When the mechanism and/or grade of MR is unclear from the TTE or is discrepant with the clinical scenario, further evaluation with transesophageal echocardiography or cardiac magnetic resonance imaging is recommended, the latter emerging as a powerful MR quantitation tool.

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Statement of conflict of interest: see page 332.

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Abbreviations and Acronyms

CMR = cardiovascular magnetic resonance

CWD = continuous wave Doppler

D_{MVA} = diameter at mitral valve annulus

EROA = effective regurgitant orifice area

LA = left atrium

LV = left ventricle

MR = mitral regurgitation

MV = mitral valve

MVP = mitral valve prolapse

PISA = proximal isovelocity surface area

PWD = pulsed wave Doppler

RF = regurgitant fraction

Rflow = regurgitant flow

RVol = regurgitant volume

SV_{MVA} = stroke volume at mitral valve annulus

TEE = transesophageal echocardiography

TTE = transthoracic echocardiography

VC = vena contracta

VCA = vena contracta area

VCW = vena contracta width

VHD = valvular heart disease

Introduction

Mitral regurgitation (MR) continues to be an important cause of morbidity and mortality worldwide.[1–3] Careful history and physical examination remain at the core of the overall evaluation of MR; however, more diagnostic imaging methods are needed to assess the etiology, mechanism, and severity of MR. In addition, imaging modalities provide a valuable insight into the associated remodeling of cardiac chambers—the left ventricle (LV) and atrium (LA)—in response to the volume overload state, important for optimal timing of intervention. Significant advances in mitral valve (MV) imaging and MR assessment have occurred over the past decade, especially in three dimensional (3D) echocardiography and cardiovascular magnetic resonance (CMR)[4–7] along with evidence linking echocardiographic quantitation of MR severity to clinical outcomes.[1] Two recent guidelines have addressed valvular heart disease (VHD): one on assessment of native valvular regurgitation by the American Society of Echocardiography and the Society of Cardiac Magnetic Resonance (ASE/SCMR),[8] and the other, an update on VHD by the American Heart Association and the American College of Cardiology (ACC/AHA).[3] These guidelines will form the backbone of this review which aims to address the evaluation of MR in the adult using the non-invasive modalities of echocardiography and CMR.

MV apparatus

The MV apparatus is a complex structure composed of many components that interact together in a highly coordinated fashion. The mitral annulus is a saddle-shaped fibromuscular structure to which the anterior and posterior leaflets attach. Because of its histologic composition with less dense and organized fibroelastic cords, the posterior annulus is more prone to pathologic dilation than the anterior annulus.[9,10] As per the widely used Carpentier scheme,[11] the anterior and posterior leaflets are divided into three segments. Although this division relies on a true anatomic segmentation (3 scallops separated by incisures/clefts) at the level of the posterior leaflet, there is no such anatomic feature at the level of the anterior leaflet; the A1/A2/A3 being defined as part of the anterior valve facing P1/P2/P3. The leaflets bear a smooth atrial surface that is free of attachments and a LV surface to which the anterolateral and posteromedial papillary muscles are connected via an intricate set of chordae tendinae.

Primary and secondary MR

MR can be broadly divided into two main categories – primary or organic, and secondary or functional – based on the abnormality leading to the regurgitation. This distinction between primary and secondary MR is essential as the diagnostic and therapeutic approaches, and clinical outcomes are quite different.[12,13]

In primary MR (Fig. 1, upper panels), an intrinsic abnormality of the MV apparatus is the underlying etiology of the regurgitation. In contrast to low/medium income countries where rheumatic disease remains highly prevalent, the most common cause of primary MR in high income countries is myxomatous degeneration, most often MV prolapse (MVP).[11] Barlow's disease is a less frequent but more extensive form of myxomatous degeneration in which multiple segments of the leaflets are thickened and redundant. A flail leaflet because of a torn chord in the setting of MVP frequently leads to severe MR; in this situation, the leaflet tip and body are both in the LA – as opposed to isolated prolapse in which the involved leaflet tip is still attached to the chordae and points towards the LV.

In secondary MR (Fig. 1, lower panels) the leaflets are intrinsically normal. Leaflet malcoaptation and regurgitation result from alteration of one or more of the components of the MV apparatus, largely due to LV and or LA remodeling. In LV global dilation or regional deformation after an infero-posterior infarct, there is displacement of the papillary muscles, tethering of the chordae, tenting of the mitral valve leaflets, and possible