Ischemic Mitral Regurgitation

Mechanisms, Intraoperative Echocardiographic Evaluation, and Surgical Considerations

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KEYWORDS

- Ischemic mitral regurgitation • Ventricular remodeling
- Intraoperative transesophageal echocardiography • Mitral valve repair

KEY POINTS

- Ischemic mitral regurgitation (IMR) is a subcategory of functional rather than organic, mitral valve (MV) disease.
- Whether reversible or permanent, left ventricular remodeling creates IMR that is complex and multifactorial.
- A comprehensive TEE examination in patients with IMR may have important implications for perioperative clinical decision making. Several TEE measures predictive of MV repair failure have been identified.
- Current practice among most surgeons is to typically repair the MV in patients with IMR. MV replacement is usually reserved for situations in which the valve cannot be reasonably repaired, or repair is unlikely to be tolerated clinically.

DEFINITION AND PRESENTATION

Ischemic mitral regurgitation (IMR) is a subcategory of functional rather than organic, mitral valve (MV) disease. By definition, IMR occurs directly as a result of coronary artery disease (CAD), and is therefore not an intrinsic pathologic process. As opposed to structural mitral regurgitation (MR), the valve leaflets in patients with IMR have normal architecture and are therefore neither myxomatous, rheumatic, endocarditic, congenitally malformed, nor otherwise diseased. The valve instead is adversely affected by abnormal, subvalvular support structures. Carpentier[1] described 3 general types of mechanisms of MR associated with abnormal leaflet motion (**Fig. 1**). Type I

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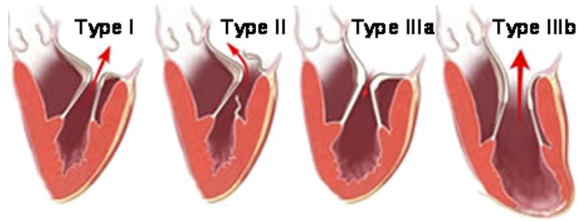

Fig. 1. Carpentier classification scheme for defining MR mechanisms based on leaflet motion abnormalities. Type I: normal leaflet motion (annular dilation; leaflet perforation); type II: increased leaflet motion (prolapse; flail); type III a: leaflet restriction during systole and diastole; type IIIb: leaflet restriction only during systole. (*Modified from* Carpentier A, Adams D, Filsoufi F. Carpentier's reconstructive valve surgery. From valve analysis to valve reconstruction. Philadelphia: Saunders Elsevier; 2010.)

describes pure annular dilation or leaflet perforation with otherwise normal leaflet architecture, usually resulting from left ventricular (LV) dilation and ventricular remodeling. Type II denotes excessive leaflet motion from leaflet prolapse or flail resulting from laxity or rupture of the chordae tendineae, or less commonly from acute papillary muscle rupture after myocardial infarction (MI). Type III describes 2 forms of valve leaflet restriction, occurring during both diastole and systole most commonly seen with rheumatic heart disease (type IIIa), or primarily only during systole (type IIIb).

Ischemic MR is divided into 2 broad categories: acute or chronic. The mechanism of each is different. In acute IMR, regurgitation is secondary to type II dysfunction with excessive leaflet motion caused by acute, postinfarction papillary muscle rupture or severe dysfunction. Although this condition represents a smaller percentage of IMR presentations, it is immediately life-threatening and requires more emergent decision making. Typically, the treatment of acute, ischemic MR is MV replacement (MVR).

Chronic IMR occurs as a consequence of ventricular dilatation secondary to ischemic ventricular remodeling, which results in papillary muscle displacement and a subsequent failure of leaflet coaptation. Regurgitation occurs secondary to annular dilation (type I) and leaflet restriction during systole (type IIIb). Chronic IMR requires regional or global ventricular dysfunction with chronic remodeling and subsequent apical displacement (eg, apical tethering or tenting), which prevents normal leaflet coaptation during systole. The cycle of long-standing CAD leading to negative ventricular remodeling with annular dilation and MR exacerbates cardiac ischemia, leads to worsened MR, and causes greater ventricular deterioration and heart failure.[2] Most patients with chronic IMR present with varying degrees of both coronary ischemia and congestive heart failure, with volume overload with poor exercise tolerance.

MECHANISMS

Specific mechanisms for the development of IMR may be reversible with revascularization and restoration of oxygen delivery or the MR may be fixed with irreversible LV remodeling. Whether reversible or permanent, remodeling creates MR that is complex and multifactorial (**Fig. 2**).[1] LV remodeling secondary to acute and chronic ischemia remains the fundamental, general mechanism for IMR and depends on apical tethering and an excessive tenting volume, which cause coaptation failure of the mitral leaflets (**Fig. 3**). Although both regional and general LV remodeling have been implicated as probable causes for IMR, the specific site of remodeling may be most relevant. Posterior MIs, especially when involving at least 30% of the LV, are more likely associated

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