Papillary Muscle Infarction, Mitral Regurgitation, and Long-Term Prognosis

Jeroen J. Bax, MD, PhD; Victoria Delgado, MD, PhD

In patients with acute myocardial infarction, the presence of mitral regurgitation (MR) is clinically important. The reported prevalence of MR after myocardial infarction varies. Bursi et al have pointed out that the prevalence varies between 1.6% and 19% in angiographic studies, but may be higher in echocardiographic studies. Indeed, among 773 patients undergoing echocardiography <30 days after infarction, MR was present in 50% of patients, of which MR was mild in 38% patients and moderate to severe in 12%. Thus, the variability in the reported prevalence of MR seems to be related to the technique used to assess its presence and to quantify its severity. It may also be related to the timing of MR assessment after infarction. In a recent report from the Valsartan in Acute Myocardial Infarction (VALIANT) study, the prevalence of MR was quantitatively assessed at baseline, 1 month, and 20 months in 341 patients after infarction. In this study, MR worsened by 1 degree in 78 patients (23%) and by 2 degrees in 10 patients (3%) after 20 months of follow-up. Conversely, 47 patients (14%) improved in MR by ≥1 degree, whereas in the remaining 206 patients (60%), the severity of MR remained unchanged. In particular, at 20 months follow-up, 15% of patients had moderate to severe MR.

It has been reported in various studies that patients with MR after infarction have a worse prognosis. In 1190 patients with acute infarction, the hazard ratios for development of heart failure, as compared with patients without MR, were 2.8 for patients with mild MR and 3.6 for patients with moderate to severe MR. In the same population, the hazard ratios for death were 1.2% (mild MR) and 2.0% (moderate to severe MR). The precise mechanism for development of MR after acute infarction, the hazard ratios for development of heart failure, as compared with patients without MR, were 2.8 for patients with mild MR and 3.6 for patients with moderate to severe MR. In the same population, the hazard ratios for death were 1.2% (mild MR) and 2.0% (moderate to severe MR). The latter may result in regional myocardial dysfunction where the PMs are inserted, along with mitral annular dilatation, systolic retraction, and displacement of the PMs, resulting in reduced leaflet coaptation.

The precise role of PM involvement in the development of MR after infarction is not clear. Several single-center studies have used contrast-enhanced magnetic resonance imaging (MRI) to detect infarction of the PMs. In this issue of Circulation: Cardiovascular Imaging, Eitel et al report findings derived from a multicenter study, the AIDA STEMI trial (the Abciximab Intracoronary versus intravenously Drug Application in STEMI), in which contrast-enhanced MRI was performed <10 days after acute infarction. Contrast-enhanced MRI is the technique of choice for detecting scar tissue and fibrosis formation after myocardial infarction, and particularly the high resolution of this technique permits careful delineation of partial or complete involvement of the PMs in the infarcted area. The different studies that have used contrast-enhanced MRI to assess PM infarction are summarized in the Table. Several observations can be derived from these studies.

First, the prevalence of PM infarction varies among the different studies, ranging from 25% to 53%. This variability may be related to underlying patient characteristics and subsequent treatment, with more recent studies reporting lower prevalence of PM infarction because of improvements in reperfusion therapy. The differences in prevalence may also be related to the MRI technique used. For example, Peters et al compared 2-dimensional with 3-dimensional late gadolinium enhancement, demonstrating the superiority of the 3-dimensional approach for detecting PM infarction. In addition, Yang et al compared multicontrast late enhancement MRI with conventional late gadolinium enhancement using the inversion recovery fast gradient echo technique in 23 patients with infarction. The authors noted superior visual contrast scores between infarcted and noninfarcted tissue/blood pool with the multicontrast late enhancement technique, and that detection of PM infarction was feasible in all 23 patients with this approach, as compared with only 9 patients with the conventional approach.

Second, there also seems to be some variability in the extent of PM involvement after infarction. The data suggest that most often only 1 PM is infarcted. However, both PMs may be involved in the infarced area in up to one third of patients (Table). Chinitz et al evaluated 153 patients 27±8 days after acute myocardial infarction, with 46 (30%) patients showing PM infarction. In this study, the posteromedial PM was infarcted in 33 (72%) patients, whereas the anterolateral PM was infarcted in 18 (39%) patients (of note, in 5 patients both PMs were involved). Similarly, in the current study by Eitel et al, the posteromedial PM was more frequently affected.

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From the Department of Cardiology, Leiden University Medical Center, the Netherlands.
Correspondence to Jeroen J. Bax, MD, PhD, Department of Cardiology, Leiden University Medical Center, Albinusdreef 2, 2333 ZA Leiden, the Netherlands. E-mail j.j.bax@lumc.nl
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than the anterolateral PM (69% versus 31%). The increased vulnerability of the posteromedial PM is likely related to its blood supply, which is usually dependent on 1 artery (either the right coronary artery [RCA] or the left circumflex coronary artery [LCX]), whereas the anterolateral PM is supplied by 2 arteries (the left anterior descending coronary artery [LAD] and the LCX). This was further supported by Chinitz et al who demonstrated that in patients with posteromedial PM infarction the LCX was the infarct-related artery in 33% and the RCA in 67%, whereas the LAD was never involved. Conversely, in patients with anterolateral PM infarction, the LAD was the infarct-related artery in 72% of patients, the LCX in 22%, and the RCA in 6%.

Third, there seems to be some variability in the clinical correlates associated with PM infarction. Chinitz et al demonstrated that patients with PM infarction had larger infarct size on MRI (16.0±10.9% of the LV versus 12.3±8.9% in patients without PM involvement; P<0.05). In these patients, myocardial scar most often involved the lateral and inferior walls. In addition, the infarct-related artery was more often the RCA or LCX. Similar findings were reported in the current study by Eitel et al: patients with PM infarction had delayed reperfusion, more often multivessel disease, and a lower frequency of LAD as infarct-related artery and anterior infarction. On MRI, these patients had larger infarct size, scar more often observed in lateral and inferior walls, less myocardial salvage, increased microvascular obstruction, more intramyocardial hemorrhage, and larger LV volumes with impaired LV function.

Finally, the various studies that used contrast-enhanced MRI to detect PM infarction have also (to some extent) evaluated the relation with MR. Okayama et al evaluated 60 patients with contrast-enhanced MRI at 25±47 months after infarction; 53% of patients had 1 or both PMs involved. From the same MRI examination, the mitral valve (presence of regurgitation, systolic retraction of the leaflets, annular size) and the left ventricle (function and sphericity) were evaluated. Patients with involvement of both PMs demonstrated larger LV volumes, lower LV ejection fraction, and worse LV sphericity index. These patients had more severe MR associated with mitral annular dilatation, reduced leaflet coaptation, and systolic retraction of the leaflets (expressed as a larger mitral valve tenting area). Chinitz et al performed the MRI <27±8 days after infarction and used echocardiography to assess the severity of MR. Among the total study population, moderate to severe MR was present in 14%; these patients had worse LV function, LV dilatation, mitral annular dilatation, increased tenting height, and reduced leaflet coaptation. On contrast-enhanced MRI, the patients with MR had more frequent lateral wall infarction (which remained an independent predictor on the multivariable analysis). However, PM infarction was not associated with MR in multivariable analysis. Eitel et al reported similar findings, but is the first to report on the prognostic value of PM infarction. The authors demonstrated that patients with PM infarction had higher death rate (7.7% as compared with 1.9% in patients without PM infarction; P<0.001), reinfarction, and heart failure at 12 months follow-up. Interestingly, MR did not predict outcome.

From the studies on contrast-enhanced MRI, it became clear that PM infarction is not directly related to MR. Indeed, animal studies have already demonstrated that isolated PM infarction is not associated with significant MR. Conversely, patients with PM infarction have larger infarctions, often with lateral wall involvement. However, PM infarction is related to LV remodeling, increased mitral annular dilatation, systolic retraction of the mitral leaflets, and reduced coaptation. Possibly, development of MR is mediated through infarct location (lateral wall) and LV remodeling with secondary dysfunction of the mitral valve apparatus, rather than through PM infarction itself.

The strong prognostic value of PM infarction is intriguing and could be related to ventricular arrhythmias, but may also be related to accompanying LV dysfunction and development of ischemic MR. The study by Eitel et al along with other studies with contrast-enhanced MRI to assess PM infarction have been performed early after infarction. Further insight into the relation between PM infarction, LV scar formation, LV remodeling, and development of MR could be obtained by a comprehensive study using contrast-enhanced MRI.
MRI early after infarction with sequential assessments of the LV and the MR. These findings could then be further related to long-term outcome.

Disclosures

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