

# 'Angina' of the papillary muscle: an overlooked but reversible etiology of mitral regurgitation

FADY Y. MARMOUSH, MD; MAZEN O. AL-QADI, MD; WASEEM Y. BARHAM, MD; AHMAD M. ABDIN, MD; AHMAD H. DARAGHMEH, MD; JOE F. YAMMINE, MD

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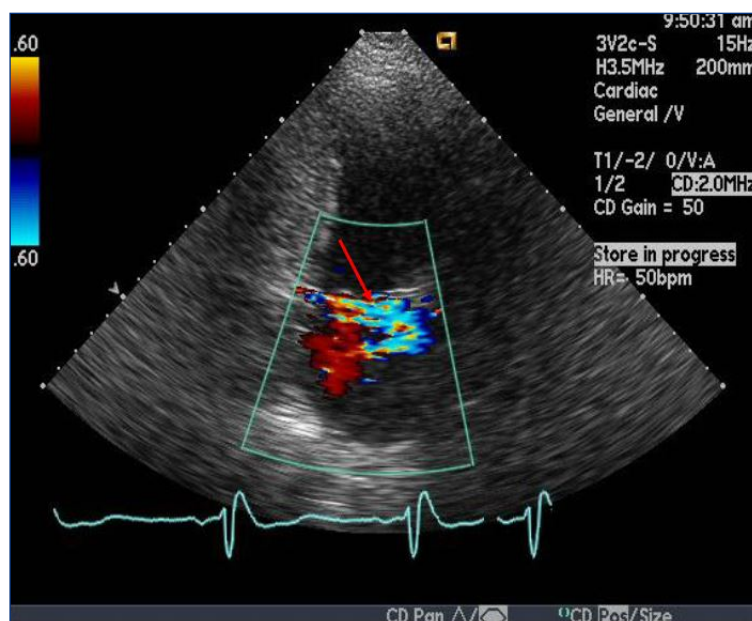
## INTRODUCTION

In the absence of acute myocardial infarction (MI), ischemic papillary muscle dysfunction (PMD) is a rare complication of coronary artery disease. Dynamic PMD results in intermittent mitral regurgitation (MR). We present a successfully-treated case of recurrent pulmonary edema due to dynamic PMD secondary to chronic coronary heart disease involving the mitral apparatus. A pathophysiologic similarity could be drawn with intermittent angina pectoris ("angina equivalent").

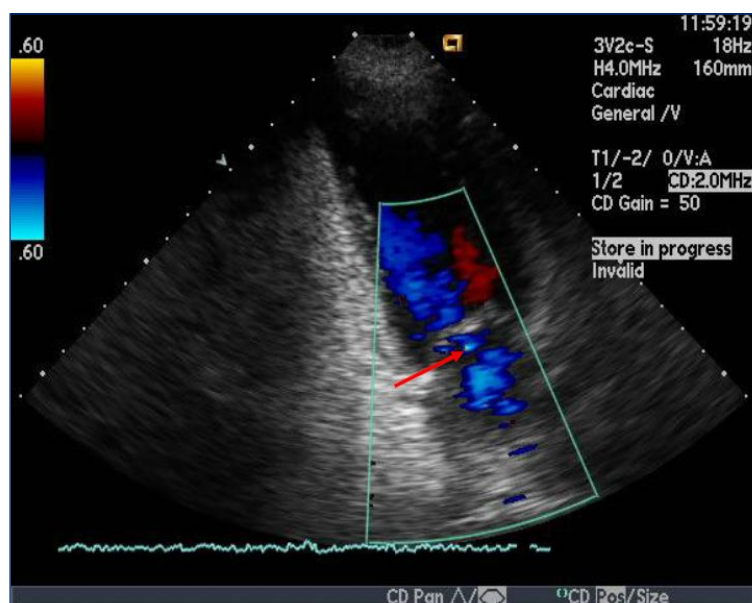
## CASE PRESENTATION

A 46-year-old woman was hospitalized for the third time within two months, with the same clinical picture of severe acute dyspnea shortly after ingesting a high load of salt and water (eating popcorn and drinking soda). Her medical history was significant for hypertension, dyslipidemia, active tobaccoism and chronic mild to moderate mitral regurgitation (her ejection fraction was known to be preserved). Physical exam revealed severe respiratory distress, bilateral crackles in her lungs, elevated jugular venous distension and an apical systolic murmur; no leg edema was noted. An acute myocardial infarction was ruled out by serial electrocardiograms and cardiac enzymes, which remained unremarkable throughout her stay. Echocardiographic evaluation this time showed severe MR with an anteriorly directed jet (Figure 1). She was treated with diuretics and her condition improved. Coronary angiography showed 80–90% middle circumflex artery stenosis, which was successfully-treated with angioplasty and stenting. Four weeks later, a follow-up echocardiogram demonstrated complete resolution of her MR (Figure 2). She had no recurrence of symptoms over the next two years.

**Figure 1.** Trans thoracic echocardiogram (2 chamber view) showing advanced, eccentric, anteriorly directed mitral regurgitation (prior to PCI)



**Figure 2.** Trans thoracic echocardiogram (2 chamber view) showing trace mitral regurgitation (after PCI)



## DISCUSSION

The mitral valve apparatus consists of two leaflets, the annulus, chordae tendineae, anterolateral and posteromedial papillary muscles. These structures work in full synchrony within a high-pressure environment. Voci P. et al<sup>1</sup> demonstrated that the anterolateral papillary muscle has more often a dual blood supply from the left anterior descending artery through the first diagonal artery and from the left circumflex artery through the first obtuse marginal artery while the posteromedial papillary muscle has a single blood supply in 63% of the cases through either the right coronary artery in 80% or the third obtuse marginal artery in 20% branching off the left circumflex artery. These findings match our case, where the culprit lesion was in the mid circumflex artery, giving rise to ischemia distally at the level of third obtuse marginal branch. The mitral valve is attached to the left atrial and ventricular walls; therefore, the valvular function is altered if one or both walls are diseased or dilated.

Mitral regurgitation is frequently associated with coronary heart disease. While it is commonly referred to as “ischemic MR,” this is rather a misnomer as the etiology is usually not related to ischemia of the mitral apparatus per se; the pathophysiology is rather explained by conditions such as annular dilatation, adverse left ventricular remodeling with posterior papillary muscle displacement, or geometrical derangement of different components of the mitral apparatus. Papillary muscle displacement is secondary to either regional or global ventricular remodeling, giving rise to tethering effects as the papillary muscle is non-extensible and therefore tenting (which is asymmetric in posterior papillary muscle displacement), which eventually results in malcoaptation of the leaflets. Acute MR is seen in acute inferior wall MI or endocarditis. Because of the usually permanent damage seen in these entities, it tends to be irreversible. This is supported by the study of MacHaalany J. et al<sup>2</sup> which showed that moderate to severe ischemic MR following ST elevation MI does not improve after percutaneous coronary intervention (PCI) and that the severity of ischemic MR was determined by the duration of ischemia during the acute event reflecting the irreversible nature of MR following MI. In terms of the outcome, our case demonstrated a single vessel disease related MR with complete resolution of MR after PCI. On the other hand, Yousefzai R. et al<sup>3</sup> found in a prospective study that PCI improved severe ischemic MR in one third of the patients and that left atrial size was the only predictor of improvement after PCI. Moreover, outcomes of coronary artery bypass surgery and stenting of multi-vessel disease in ischemic MR were similar<sup>4</sup>. Reversible ischemia of the posterior papillary muscle from high grade stable lesions involving the supplying vessel (the left circumflex artery in our case), and causing acute intermittent and reversible MR is rarely reported.<sup>5</sup>

Exercise increases the regurgitant volume across the mitral valve in subjects with established left ventricular

dysfunction admitted with acute pulmonary edema.<sup>6</sup> In our case, a similarity with “stable angina” could be established where dyspnea and the MR were the angina equivalents, while the volume stress from salt and volume overload was the decompensating factor.

## CONCLUSION

Dynamic PMD from high-grade stable coronary ischemia of the mitral apparatus territory could be an overlooked etiology of acute and recurrent mitral regurgitation. This under-reported entity should be differentiated from other more common mechanisms of MR. It can be reversed by coronary revascularization.

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## Authors

Fady Y. Marmoush, MD, Memorial Hospital of Rhode Island, Alpert Medical School of Brown University  
 Mazen O. Al-Qadi, MD, Memorial Hospital of Rhode Island, Alpert Medical School of Brown University  
 Waseem Y. Barham, MD, Memorial Hospital of Rhode Island, Alpert Medical School of Brown University  
 Ahmad M. Abdin, MD, Memorial Hospital of Rhode Island, Alpert Medical School of Brown University  
 Ahmad H. Daraghme, MD, Memorial Hospital of Rhode Island, Alpert Medical School of Brown University  
 Joe F. Yammine, MD, Memorial Hospital of Rhode Island, Alpert Medical School of Brown University

## Correspondence

Fady Y. Marmoush, MD  
 Memorial Hospital of Rhode Island  
 Internal Medicine Residency Program  
 111 Brewster Street  
 Pawtucket, RI 02860  
 401-4578480  
[fady\\_marmoush@brown.edu](mailto:fady_marmoush@brown.edu)