CARDIOLOGY Rounds®

As presented in the rounds of the Division of Cardiology, St. Michael's Hospital, University of Toronto

Papillary muscle rupture post-myocardial infarction

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Among the mechanical complications of myocardial infarction (MI), papillary muscle rupture (PMR) poses a worthy challenge to the clinician. When unrecognized, it is lethal in nearly all patients; however, when it is promptly recognized and surgically corrected, survival is possible in the majority of cases.

Epidemiology

Progressive pump failure is the cause of the majority of in-hospital mortality in acute MI patients. Papillary muscle rupture occurs in about 1% of MIs and accounts for 1%-5% of the overall mortality in acute MI.[1-3] A case series of myocardial ruptures post-MI demonstrated that free wall rupture is the most common site of rupture and that septal rupture and PMR are less common.[4] Combined ruptures, such as septal rupture with free wall rupture, also occured in this series.

Patients with PMR often present with cardiogenic shock post-MI. In the SHould we emergently revascularize Occluded Coronaries in cardiogenic shocK (SHOCK) registry, the most common etiology of cardiogenic shock post-MI was pump failure (78%), followed by severe mitral regurgitation (6.9%).[5] Septal rupture (3.9%) and free wall rupture (1.4%) were much less common causes of cardiogenic post-MI shock in the SHOCK registry. The mortality of patients with cardiogenic shock post-MI due to severe mitral regurgitation was 55%, which is similar to the overall mortality of 60% for all patients in the SHOCK registry.

Post-infarction mitral regurgitation

Mitral regurgitation (MR) post-MI is common and prognostically important.[6,7] Approximately 19% of patients with an MI have significant MR according to data from the Survival and Ventricular Enlargement (SAVE) study.[7] Furthermore, in a case-controlled study of 303 patients post-MI, MR independently predicted a worst prognosis.[6] In this study, the 5-year mortality in patients with ischemic MR was 62% compared to 39% in post-MI patients without MR. The severity of MR, as defined echocardiographically by effective regurgitant orifice and regurgitant volume, predicted a worse prognosis.

PMR is a distinct entity among the overall group of post-MI patients with MR. Patients with PMR are more likely to have 1- or 2-vessel coronary artery disease, unlike the overall group of

Editor's note: This April issue, as well as the upcoming May and June/July issues of *Cardiology Rounds,* are late in reaching you. This delay was caused by the SARS emergency measures imposed in most of the hospitals in Toronto during March and April. During that time, almost all academic rounds were cancelled. We apologize for the delay in the series.

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post-MI patients with moderate to severe MR who are most likely to have 3-vessel disease.[8,9] Because there are many more cases of post-MI MR (non-papillary rupture) than there are PMR cases, the first challenge is to establish the basis of MR.

Primary angioplasty and mitral regurgitation

In a meta-analysis by Kinn et al, primary angioplasty was associated with an 86% reduction in the risk of mechanical complications post-MI when compared to fibrinolytics.[3] Primary angioplasty reduced acute MR by 82% over the use of fibrinolytics. More effective reperfusion therapies help reduce and limit infarct size, making it less likely for the papillary muscles to be affected by the infarction. Moreover, fibrinolytics may cause hemorrhagic conversion of necrotic tissue, further weakening the area of infarction.

Pathophysiology

There are two papillary muscles: the anterolateral and posteromedial papillary muscles. The posteromedial papillary muscle is 6-12 times more likely to rupture because it is solely supplied by the posterior descending artery. The anterolateral papillary muscle is relatively protected because it is supplied by both the left anterior descending and left circumflex arteries. Since the papillary muscles are subendocardial structures, it is important to recognize that small endocardial infarcts can cause papillary muscle ruptures. The rupture can either be complete or partial, and each type makes up half of the overall cases.[10-12] It is also important to recognize that a partial tear can convert to a complete tear and patients may deteriorate dramatically. Classically, the MI that causes a PMR is one that is small- to moderate-sized. The remainder of the left ventricle retains sufficient systolic function to exert shear forces on the papillary muscle that lead to its disruption.

Current literature

The existing literature on PMR is sparse and consists of 1 clinical case series and 2 autopsy series performed during the pre-thrombolysis era. The clinical case series was composed of 17 patients with papillary muscle rupture post-MI treated at the Mayo Clinic between 1939-81.[10] The mean interval from onset of acute MI to PMR was 6 days. Subendocardial infarctions made up 52% of the infarcts, whereas 48% of the cases were transmural infarcts. These data help dispel the myth that PMR is caused only by large Q-wave infarctions.

The first autopsy series reported on 25 cases of PMR that occurred between 1977 and 1987.[11] Twelve percent of the cases were subendocardial infarcts versus 88% that were transmural infarcts. This contrasts with the clinical study and likely demonstrates the bias of a pathological study. Interestingly, 56% of these cases involved the posteromedial papillary muscle versus 16% that involved the anterolateral papillary muscle and 32% that involved both papillary muscles. Furthermore, 68% of cases had necrosis of the right ventricle. Unfortunately, no clinical data were available for this study, thereby limiting its usefulness.

The second autopsy study reported on 22 patients with PMR from 1973-86.[13] Posteromedial PMR made up 73% of the cases as expected by the anatomy of the blood supply. The mean length of time between MI and death was 5 days. If the PMR was complete, death occurred at a mean of 1.8 days versus 10 days with a partial PMR. The rapidity of death underscores the importance of rapid diagnosis and surgical intervention.

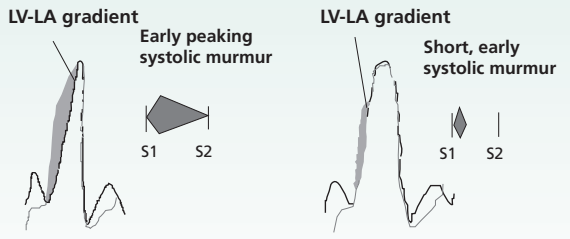
An unpublished case series of 12 consecutive cases of PMR from St. Michael's Hospital during 1997-2003 probably represents the most contemporary data. In this series, the mean age of patients was 68 years and 42% were male. Almost all patients were in cardiogenic shock and 91% were in Killip class IV post-MI. Similar to other data, 58% of the patients had a subendocardial infarction versus 42% who had a transmural infarct, while 75% involved the posteromedial papillary muscle. In-hospital mortality was only 25%; this is remarkably lower than historical case series. Seventy-five percent of the patients had surgery and for this subgroup, the in-hospital mortality was only 12%.

To summarize, subendocardial infarctions account for approximately half of all PMRs and the posteromedial papillary muscle is most commonly involved. Due to their precipitous decline, there is a much shorter period of time for intervention in patients with a complete PMR as compared to those with a partial PMR.

Clinical presentation

Patients with a PMR classically present with hypotension and tachycardia due to inadequate cardiac output and with florid pulmonary edema due to elevated left atrial pressure. The murmur of an acute MR may be loud and holosystolic, but is often soft and short, as shown in Figure 1. In fact, it may be absent, especially in cases of complete papillary muscle rupture where the left atrium (LA) and left ventricle (LV) become a common chamber and there is no resultant gradient to create a murmur (refer to Figure 1). The autopsy study by Vlodaver et al found that only 11% of patients with a complete PMR had a murmur, whereas 70% of patients with a partial PMR had a murmur.[4] In the overall series, only 42% of patients with

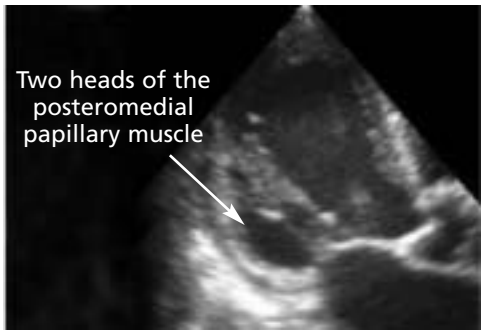
Figure 1: Murmurs of papillary muscle rupture



If the LA and LV become a nearly common chamber due to acute severe MR, the murmur may shorten or disappear

LA = Left atrium; LV = Left ventricle; MR = mitral regurgitation

Figure 2: Transthoracic echocardiogram (3-chamber view) demonstrating a normal posterior papillary muscle)



PMR had a murmur. Accompanying clinical signs may include a soft or absent S1 (related to poor mitral closure), an S3, or a loud P2, as well as pulmonary edema.

Echocardiography

The differential diagnosis of a post-MI murmur includes mitral regurgitation (PMR and non-PMR related), ventricular septal defect, free wall rupture, and tricuspid regurgitation. In these patients, echocardiography is critical to help differentiate the etiology of the new murmur. On transthoracic echocardiography in PMR, characteristic findings are severe MR, a flail or prolapsing mitral leaflet, partial papillary muscle disruption, a regional wall motion abnormality with overall, good LV function, and pulmonary hypertension (Figures 2, 3, and 4). The velocity of the MR jet may be lower than expected (<4 m/s) due to lower LV systolic pressure and higher left atrial pressure. In an echocardiographic study of PMR at the Mayo Clinic, Kishon et al examined 21 cases of PMR between 1980-91.[8] On transthoracic echocardiography, the PMR was directly identified in only 45% of cases and a flail mitral leaflet was seen in only 73% of cases. These data suggest that transthoracic echocardiography is useful, but in a significant proportion of patients, transesophageal echocardiography (TEE) is necessary to confirm the diagnosis of a PMR. On TEE, the classic diagnostic finding for a complete PMR is visualization of the ruptured papillary muscle prolapsing into the left atrium (Figure 5). In a study of 21 patients undergoing TEE for PMR, the papillary muscle was not visualized prolapsing into the left atrium in 35% of patients, presumably because the papillary muscle head was held on by strands or knotted in the cords.[13] Importantly, 90% of patients in the series had a large-amplitude erratic mass visualized in

Figure 3: Transthoracic echocardiogram (2-chamber view) demonstrating papillary muscle rupture and resultant severe mitral regurgitation (colour Doppler)

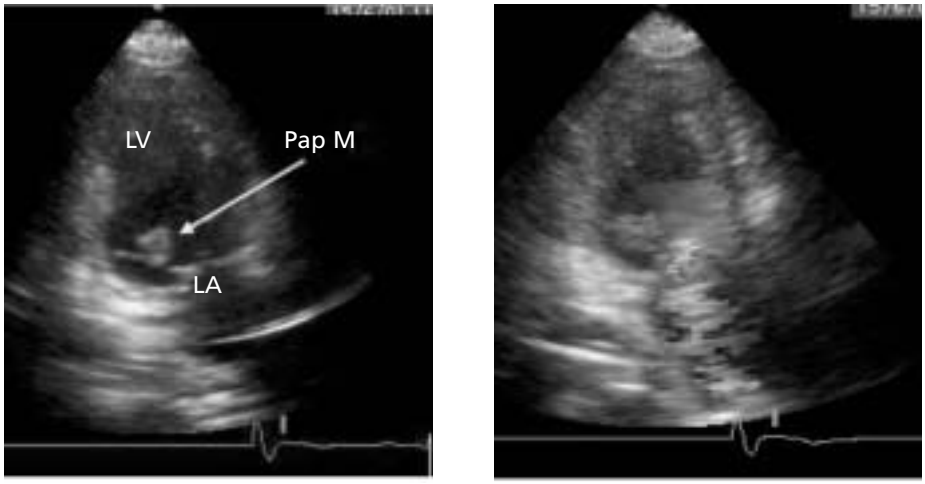


Figure 4: Pulmonary vein flow demonstrating systolic reversal suggestive of severe mitral regurgitation

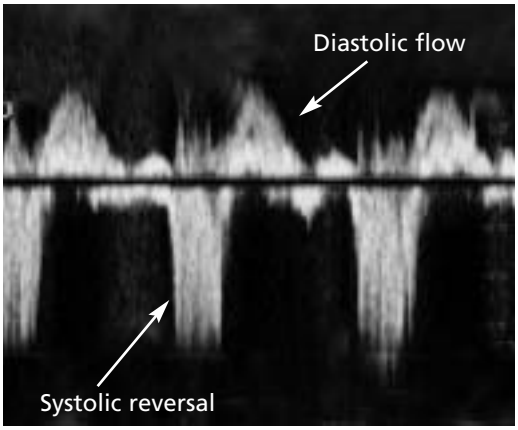
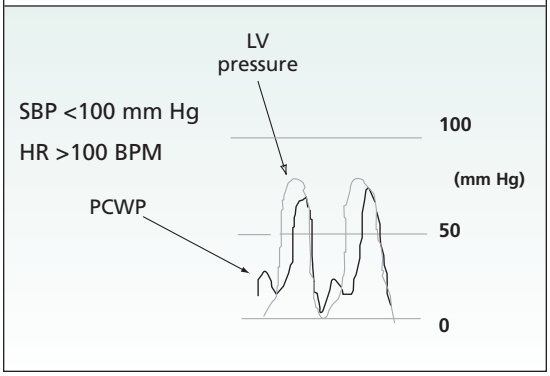


Figure 6: Typical left and right heart hemodynamics of complete papillary muscle rupture



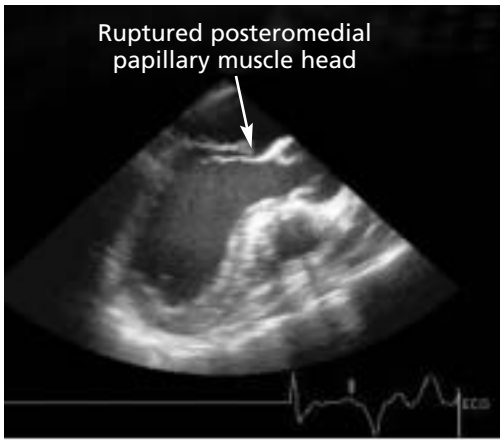
PCWP = Pulmonary capillary wedge pressure; BPM = Beats per minute; SBP = Systolic blood pressure; HR = Heart rate

the LV and by combining these two criteria, all patients with PMR in this small series were detected.

Hemodynamics

Classically in patients with PMR post-MI, one expects to see large V waves and a low cardiac index, as shown in Figure 6. In the clinical series of PMR by Nishimura et al, the mean pulmonary capillary wedge pressure (PCWP) was 21 mm Hg, mean peak V wave was 55 mm Hg, and mean cardiac index was 2.4 L/min/m^2.[10] The differential diagnosis for large V waves includes ventricular septal defect, left ventricular failure, and severe MR with intact papillary muscles. It is important to realize, however, that V waves may be absent. In a study by Haskell et al of patients with known MR, only 60% of those with known severe MR had large V waves on PCWP tracing.[14]

Figure 5: Transesophageal echocardiogram demonstrating ruptured papillary muscle head in left atrium



Furthermore, in another study, Goldman et al described two cases of PMR post-MI that did not have V waves on PCWP tracing and, in fact, in one case, the diagnosis was missed until post-coronary artery bypass surgery.[15] In summary, large V waves on the PCWP tracing neither rule-in nor rule-out the diagnosis of PMR; therefore, echocardiography must be performed if the diagnosis is suspected.

Management

Prompt diagnosis and surgical referral are likely the most critical factors in managing patients with PMR. Medical therapy is only a bridge to definitive surgical therapy. In patients who are normotensive, vasodilators such as nitroprusside or hydralazine are the medical therapies of choice to reduce the regurgitant fraction and improve cardiac output. This was demonstrated in a study by Chatterjee et al in patients with severe MR, where vasodilators were shown to reduce the regurgitant fraction and improve both cardiac output and forward stroke volume.[16] In hypotensive patients, intra-aortic balloon counterpulsation (IABP) is the recommended therapy to reduce afterload and improve cardiac output. This was also demonstrated in a study by Gold et al in 6 patients with cardiogenic shock and severe MR post-MI, where IABP counterpulsation reduced PCWP and improved the cardiac index and mean arterial pressure.[17]

Medical therapy provides a bridge to definitive surgical therapy because without surgery, mortality at 1 week is reported to be 80%.[1] Surgical therapy is either in the form of mitral valve repair or replacement. Repair involves sewing the ruptured papillary

muscle to an adjacent healthy one. Patients with small infarctions that spare an adjacent papillary muscle are better candidates for mitral repair. Mitral valve repair is theoretically preferable because a conventional mitral valve replacement alters LV geometry and may affect function by disrupting the anatomic continuity between the mitral annulus and the papillary muscles and impairing contraction of the vasoconstrictor muscles by implantation of a rigid prosthesis in the mitral annulus.[18]

In a study examining the feasibility of repair, 6 consecutive patients at a single centre underwent mitral repair for PMR post-MI.[19] In this study, the mean age of patients was 60 years and 50% were in cardiogenic shock. Survival was 100% at a mean follow-up of 8.6 months. While these results are encouraging, they are only from a single small study and need to be repeated on a larger scale. In a larger, retrospective, surgical study of 141 patients with MR secondary to ischemic heart disease at the Los Angeles Heart Institute between 1970-83, 50% had infarcted or ruptured papillary muscles.[20] Patients in this study had a mean age of 63 years and 93% were in NYHA class III or IV heart failure. Unfortunately, only 40% received the operation within 30 days of infarct and so this group had a clear survivorship bias. Patients who underwent repair had better long-term survival than those who underwent replacement. Patients undergoing mitral repair had only an 8% re-operation rate over a 10-year follow-up, suggesting the durability of repair. However, the likelihood of bias in these small studies is high; patients undergoing repair may be healthier with smaller infarcts and so these results need to be interpreted with caution. Some authors argue that mitral valve replacement is preferred in these sick patients because of shorter cross-clamp times; however, in these small series, cross-clamp times were similar in both groups. In summary, these case series suggest that mitral repair, when it is possible, is durable and has a good long-term outcome.

Outcomes

It is often argued that the mortality associated with surgery in PMR patients is too high. However, although the associated mortality is high, surgery does salvage 60% of shock cases and 75% of non-shock cases.[20] The natural history of PMR without surgery is 50% mortality at 24 hours and 80% mortality at 1 week.[1]

The long-term outcome post-operatively is good for patients with cardiogenic shock and papillary muscle rupture if they survive the operation.This was confirmed in a study by David et al, who looked at patients undergoing mitral valve replacement for PMR post-MI. In the subset with cardiogenic shock, survival stabilized after 6 months at 73% for a follow-up of 4 years.[18] Factors that predicted surgical risk were severity of heart failure, cardiogenic shock, duration of shock, age, LV function (few survived surgery if LVEF < 35%), and right ventricular infarction.

Summary

A high index of suspicion is crucial when diagnosing post-MI PMR, a condition that is potentially lethal. The clinical presentation is generally acute pulmonary edema or cardiogenic shock with, or without, a new murmur post-MI. It is important to remember that a PMR can occur with a subendocardial infarction, as well as a transmural infarction. Echocardiography is the test of choice with transesophageal echocardiography to confirm the diagnosis. Vasodilators and intra-aortic balloon pump counterpulsation are important to temporarily stabilize the patient. It is critical to obtain an urgent surgical consultation for consideration of mitral valve repair or replacement.

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