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Journal of Acute Disease

journal homepage: www.jadweb.org

Review article http://dx.doi.org/10.1016/j.joad.2015.11.001

Clinical presentation, diagnosis and management of acute mitral regurgitation following acute myocardial infarction

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ARTICLE INFO

Article history:
Received 14 Aug 2015
Received in revised form 17 Sep 2015
Accepted 10 Nov 2015
Available online 8 Jan 2016

Keywords:
Acute mitral regurgitation
Acute myocardial infarction
Papillary muscle rupture
Cardiogenic shock

ABSTRACT

Acute mitral regurgitation (MR) is a frequent complication of acute myocardial infarction, with a variable presentation depending on the severity of MR and the integrity of the subvalvular apparatus. While most cases are asymptomatic or have mild dyspnea, rupture of chordae tendinea or papillary muscles are catastrophic complications that may rapidly lead to cardiogenic shock and death. Despite the presence of pulmonary edema and/or cardiogrenic shock, the murmur of acute MR is usually subtle due to rapid equalization of left atrial and left ventricular pressure gradient, and therefore misleading. Echocardiography is the definite diagnostic modality, allowing quantification of the severity of MR and the structural abnormalities within the subvalvular apparatus. Severe MR accompanied by rupture of chordae or papillary muscles should be managed with temporary stabilization with medical treatment or with mechanical circulatory support, with subsequent surgical intervention to repair or replace the valve.

1. Introduction

Several pathogenic mechanisms and dynamics may lead to ischemic mitral regurgitation (MR) after an acute myocardial infarction (MI). These mechanisms include papillary muscle ischemia and changes in left ventricular (LV) geometry[1–4]. Papillary muscle dysfunction, as well as partial/complete rupture of a papillary muscle or mitral chordae may cause hemodynamic instability due to acute mitral regurgitation, with subsequent pulmonary edema or cardiogenic shock, eventually resulting with poor short-term outcomes. All mechanical complications of an acute MI are life-threatening (rupture of ventricular free wall, papillary muscle or ventricular septum) and require early recognition and prompt initiation of aggressive treatment[5].

2. Epidemiology

The advent of primary percutaneous coronary intervention, which is one of the most important therapeutic interventions for acute ST elevation MI in the present era, reduced the general incidence of mechanical complications (< 1%)[2,5–8]. The incidence for papillary muscle rupture has been reported between 0.25% or 0.26% in contemporary registries. These figures were much higher in older studies, with a reported incidence between 1% and 5%, and in average 2.3% of patients with an acute myocardial infarction experienced a mechanical complication[2,6,9–11]. Nevertheless, mortality due to mechanical complications following an acute MI remains unacceptably high (around above 50%)[5], and cardiogenic shock is still as an important cause for early mortality despite improvements in therapeutic strategies, complicating 3%–7% of cases with acute MI[12–16]. In several trials, it has been observed that a higher proportion of cardiogenic shocks were caused by acute MR in women. This was attributed to several factors related to gender-specific predisposition, such as differences in patterns of mitral/papillary vascular supply, collateralization and structural differences within the connective tissue, but the exact cause remains unknown[9,17,18].

3. Pathophysiology

Mitral valve is composed of mitral leaflets and the subvalvular apparatus (chordae tendineae and papillary muscles), which are parts of the LV and are altered by functional and structural changes in LV. The relationship is bidirectional, as the alterations within the subvalvular apparatus itself could induce

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Peer review under responsibility of Hainan Medical College.

changes in LV geometry and systolic performance[19]. Two papillary muscles (anterolateral and posteromedial) arising from the left ventricle originate from embriologic trabeculae carneae, and in order to ensure valvular competence during the ventricular systole, they are attached beneath the ipsilateral commissures of the mitral valve[6,20]. Each papillary muscle is connected to both leaflets via chordae tendinea[6,11,20]. Ischemic MR due to papillary dysfunction/rupture is associated with two mechanisms, namely, interruption of the coronary blood supply and alterations within the LV geometry. Subendocardial branches of coronary arteries supplying papillary muscles cross through the full thickness of myocardium. These arteries have a very long intramyocardial course, making them vulnerable to ischemia[6]. Anterolateral papillary muscle (ALPM) receives blood supply from two major coronary branches, left anterior descending and left circumflex artery (LCx). However, blood supply to the posteromedial papillary muscle (PMPM) is limited to a single coronary artery, the posterior descending artery originating either from right coronary artery in right dominant systems or from the LCx in left dominant systems. Rupture or infarction of the posteromedial muscle occur more frequently as it receives blood supply from a single source[2,6,11]. According to various registries, the rupture of PMPM is six to twelve times more frequent than the rupture ALPM[6,20–22]. In most instances, the rupture of PMPM is partial and does not involve full thickness of the muscle. In contrast, complete rupture of ALPM occurs more frequently than complete rupture of PMPM. This apparent contradiction is explained by different anatomical properties of each papillary muscle, as ALPM often has single body or head, while the posteromedial papillary muscle usually includes two bodies or heads[6,19,23]. In addition, the majority of MR cases caused by rupture or dysfunction of papillary muscle are due to relatively small infarctions. Since the jeopardized myocardial mass is rather small, LV function is usually well-maintained in these patients (median LV EF is 35%–37% according to various registries)[6,9,21,24,25].

4. Signs and symptoms

Signs and symptoms are quite variable, ranging from mild dyspnea to cardiopulmonary arrest (Table 1). Following an acute MI complicated with acute MR, hemodynamic stability depends on the integrity of the subvalvular apparatus, presence of valvular dysfunction and the extent and location of myocardial damage[1,16]. The time to papillary muscle rupture following an acute MI was cited as 1–14 days in older studies, with the days 2–7 constituting the most vulnerable period[2,5,11,26,27]. In SHOCK registry, however, the average time to papillary muscle rupture was 13 h[1,9]. A complete rupture generally results with cardiogenic shock and death, while the partial rupture of a papillary muscle or associated chordae usually presents with respiratory distress or pulmonary edema rather

Table 1
Summary for the clinical presentation and findings in patients with acute mitral regurgitation complicating an acute myocardial infarction.

Clinical presentation	Characteristics	Findings
Symptoms	- Exertional dyspnea - Orthopnea - Palpitations - Fatigue - Cough - Chest discomfort or angina - Syncope - Symptoms of inadequate organ perfusion (cold clammy extremities, sweating, alterations in consciousness). - Cardiac arrest	Symptoms are unreliable for the estimation of the underlying pathology due to high variability. Complete or incomplete papillary muscle rupture is frequently accompanied by cardiogenic shock or cardiac arrest.
Physical examination	- New pansystolic murmur best auscultated from apex and radiates to left parasternal bordar or axilla. - Jugular venous distension. - Elevated respiratory rate, inspiratory fine crackles, contraction of accessory respiratory muscles.	No direct correlation between the severity of murmur and the severity of MR. No displacement of cardiac apex.
Electrocardiography	- Tachycardia with or without sinus rhythm. - ST segment elevation in inferior (usual) or anterior (rarer) leads. - Non-specific findings in some occasions.	Not specific and reflects underlying MI pattern only. ST segment elevation may be absent despite total occlusion of left circumflex artery.
Chest X-ray	- Bilateral interstitial or alveolar infiltrates.	Cardiomegaly is absent
Echocardiography	- Severity and mechanism of MR. - Left ventricular function. - Motion of left ventricular segments. - Differential diagnosis for other possible complications.	Transesophageal echocardiography has superior accuracy and allows better visualization of the valve. TEE tends to overestimate the severity of MR preoperatively and underestimate perioperatively.
Right heart catheterization	- Elevated pulmonary arterial pressure and pulmonary capillary wedge pressure. - Giant V or CV waves.	CV waves could be observed with other complications, reducing the sensitivity.

than cardiogenic shock[1,2]. Patients with MR could present with a myriad of symptoms, ranging from cough or vague chest discomfort to severe shortness of breath or significant angina[11]. Findings from a cardiovascular examination include tachycardia, along with a new pansystolic murmur loudest at the cardiac apex and radiating to the axillae, which may also have a diastolic component. When a PMPM rupture is present, the murmur may radiate to the left sternal border, which could be similar to the murmur of ventricular septal rupture (VSR). There is no direct correlation between the intensity of the murmur and the severity of MR[1,2,11]. In patients with poor cardiac output, the murmur may be soft or even absent due to the reduced pressure gradient between the LV cavity and the overloaded LA during systole[1]. Jugular venous distention may be present in some cases. Pulmonary examination may reveal an elevated respiratory rate, contraction of accessory respiratory muscles, and bilateral fine crackles at the basal portion of the lungs or crackles covering both lung fields[11,28–30]. Localized pulmonary edema at the right upper lobe may also be encountered as the regurgitant flow and directed at the right superior pulmonary vein[1,2].

5. Electrocardiography

ECG usually demonstrates ST segment elevation in the inferior and posterior leads, while ST elevation in anterior leads is rarer[11], and only non-specific ST and T wave alterations are present in some instances (Table 1). ECG should be routinely recorded on admission and repeated as needed. This is dictated by the clinical course, and repeat ECG's should be obtained after clinical stabilization or further deterioration[2,31]. Whilst ST elevation of anterior location is the predominant presentation in patients with LV failure and shock, patients presenting with pulmonary edema frequently have an ST elevation on posterior leads[18]. In spite of the fact that more than half of the MR cohort in SHOCK Trial did not have ST elevation and new Q waves, in many of these patients it was recognized that there was complete vessel occlusion and a significant proportion of these had completed occlusion in LCx artery. This is explained with the scarce use of posterior leads when the SHOCK Trial Registry was commenced[9]. Alveolar pulmonary congestion should be confirmed with chest radiography in patients with acute MI, which shows diffuse bilateral interstitial or alveolar infiltrates without associated cardiomegaly[11,25,28–30].

6. Echocardiography

Echocardiography should be carried out immediately after hospital admission in order to confirm the diagnosis of acute MR when suspected clinically. Beyond confirming the diagnosis, echocardiography also allows differential diagnosis for other mechanical complications, such as VSR. Transthoracic echocardiography (TTE) with color flow Doppler is the initial imaging modality, having a sensitivity of 65%–85% for the diagnosis of papillary muscle rupture with concomitant MR[2,11,32]. Additionally, LV wall motion abnormalities, global left ventricular function, severity of leaflet prolapse, presence of flail chords or the integrity of papillary muscles could be assessed with TTE. Transesophageal echocardiography (TEE) has a superior sensitivity and diagnostic accuracy compared with TTE since the mitral apparatus is located posteriorly and TEE probe is much closer to the valve, allowing better visualization (Table 1). This is most pronounced in the presence of eccentric regurgitant jets coursing towards the posterior wall of the LA. Therefore, TEE has a diagnostic yield ranging between 95% and 100%[11,28], for assessing the anatomy of the mitral valve and identifying the pathophysiology underlying MR[2,11]. Both TTE and TEE may be used in combination as different diagnostic modalities if necessary. Akinesia and hypokinesia involving the inferior and posterior walls in patients with MR can be detected by both echocardiographic modalities[25]. TTE generally tends to overestimate the LV function and underestimate the degree of acute MR, which could be attributed to hyperdynamic left ventricle in the presence of an acute MR, poor acoustic windows, resting tachycardia and rapid equalization of pressure between left ventricle and left atrium. In contrast, TEE is prone to overestimate mitral regurgitation in the preoperative period as the position of the probe is relatively closer to the regurgitant jet, while it underestimates the degree of MR in perioperative period due to effects of anesthesia[1,2]. Echocardiography should be repeated after a new anginal episode, dyspnea or clinically deterioration in the coronary care unit.

Pulmonary capillary wedge pressure tracings from pulmonary artery may show large V waves in the presence of an acute MR. However, this finding is not pathognomonic since it may be seen in others conditions, such as VSR and massive LV failure[1,2,33].

7. Management

Treatment of acute MR is varied with respect to underlying mechanism or type of mitral regurgitation (Table 2). Thus, acute and chronic ischemic MR require different management strategies as the pathophysiologic mechanisms underlying these conditions are not same[25]. Mild to moderate chronic MR following acute MI is seen in 15%–45% of patients[2,34]. These are usually asymptomatic, well tolerated and addressed with surgical interventions if needed in the follow up. Acute MR necessitates emergency treatment if accompanied by pulmonary edema or cardiogenic shock. In addition, emergency surgery is indispensable in the presence of an acute MR resulting from the rupture of a papillary muscle[2,25]. The patient should be treated immediately with medical therapy to relieve symptoms and ensure clinical stability in the initial term. The goal of pharmacological therapy is to obtain a reduced regurgitant fraction and increased forward stroke volume via reducing left ventricular filing pressure and afterload[2,25]. Mechanical support with an intra-aortic balloon counterpulsation, which decreases afterload and increases aortic diastolic pressure, can be used to achieve aforementioned objectives in selected patients. Temporary mechanical cardiac support with a LV assist device (such as Impella Recover device) or VA-ECMO can be used to reduce in the cardiac afterload (for the former) and to improve coronary circulation, end-organ perfusion and oxygenation in patients with ongoing cardiogenic shock and severe myocardial ischemia[2,5]. Medical therapy includes morphine sulfate, nitroglycerin or sodium nitroprusside (continuous infusion), diuretics and ACE inhibitors; in addition to standard antiaggregant and anticoagulant therapies. In patients with hypotension, positive inotropic agents such as dobutamine and dopamine should be considered[31].

Table 2
Management strategies in patients with severe acute mitral regurgitation following acute myocardial infarction.

Strategies	Treatment
Medications	- Vasodilators (nitroglycerine, sodium nitroprusside, morphine sulfate) - ACE inhibitors - Diuretics - Inotropic agents with or without additional vasodilator effects (dobutamine, dopamine)
Mechanical ventilation	- Non-invasive (preferred if the patient is stable) - Invasive
Intraaortic baloon counterpulsation	
Temporary left ventricular assist devices	- Impella - Tandem heart - Levitronix - Berlin EXCOR or similar pulsatile pumps
Extracorporeal membrane oxygenation (venoarterial)	
Therapeutic hypothermia	
Definite surgical correction	

Also, invasive or non-invasive mechanical ventilation should be used as necessary. While IMV is considered in the setting of severe pulmonary edema, cardiogenic shock and cardiopulmonary arrest that results with deterioration of consciousness due to acute and persistent respiratory insufficiency (in the arterial blood gas analysis $pO_2 < 60$ mmHg, $pCO_2 > 60$ mmHg and respiratory rate > 40 respiration/min), noninvasive mechanic ventilation is adequate and effective in most episodes of stand-alone acute pulmonary edema or pulmonary congestion[2,28,31]. IMV is associated with long and short term mortality and is an independent predictor of mortality[35]. Therapeutic hypothermia may be applied to patients that do not regain consciousness following cardiopulmonary resuscitation, with a target core temperature of 33–34 °C achieved by infusing 4 °C Ringer solution and surface cooling using an external cooling system such as the Arctic Sun 2000 gel pads system (Bard, Covington, Georgia, USA) in the CCU, as suggested by guidelines[28,36,37].

Emergent cardiac catheterization should be performed to identify coronary lesions and to bridge to revascularization[2,38,39]. Urgent or emergent surgical intervention, synchronous with the initial medical treatment should be considered as soon as possible due to the catastrophic potential for the rupture of a papillary muscle. Although the overall mortality for CS in the SHOCK trial (60%) was lower than the figures given in previous reports (80%–90%), it still remains extremely and unacceptably high[18,40,41]. The reduced mortality rate was explained with increased LV assist with IABP device and improved revascularization techniques and strategies. In the SHOCK Trial Registry, hospital mortality rate was around 40% for patients in whom mitral valve surgery was performed[9,18]. Mitral valve surgery encompasses multiple strategies including mitral valve repair and mitral valve replacement. If there is a partial papillary muscle rupture with limited necrosis and the tissues were found suitable for repair at the time of surgery, valve repair may be preferred over replacement. However, ruptured papillary muscle is usually fragile following an acute myocardial infarction and the preferable procedure is the valve replacement that provides long term durability. Fortunately, complete rupture of the papillary muscle is an extremely rare complication[11]. Addition of coronary revascularization to mitral valve surgery is associated with improved short and long term outcomes. The median time to surgery (combined valve replacement and revascularization) was seven days in one trial[2,11,38,42]. Although emergent or urgent surgery with IABP support in early period after MI was advocated due to the risk of sudden hemodynamic collapse, postponing the intervention up to a couple of weeks was associated with lower surgical mortality (17% for acute MR) in several studies, if patients did not fulfill criteria for shock or did not have initial hemodynamic instability. This period is for the development of fibrotic tissue[11,43–50]. When emergent valve surgery is needed, additional surgical revascularization did not confer any mortality benefit in the early period (27.3% *vs.* 26.4%; $P > 0.05$), but was associated with a significant improvement in long term survival up to 15 years after the index event (additional CABG group: 64% *vs.* no additional CABG group: 23%; $P < 0.001$)[2]. Emergency surgery, however, is fundamental in patients with cardiogenic shock as the survival is better when the time period between the onset of symptoms and surgical intervention is shorter[43].

8. Conclusions

High mortality remains as the main problem despite improvements in surgical and revascularization strategies. To avoid catastrophic consequences, diagnosis should be confirmed rapidly and definitely, with subsequent referral to surgery as soon as possible. Emergent medical therapy should be initiated immediately after confirmation of the diagnosis since patients may rapidly deteriorate and die while waiting for surgery. Clinicians should be aware of mechanical complications following AMI, and attention should be given to patients presenting with non-ST elevation MI, patients having an inferior or posterior STEMI, and particularly for women with acute coronary events owing to the fact that majority of the patients with severe MR with or without shock were female (52% *vs.* 37%, $P = 0.004$) according to the SHOCK Trial results[9].

Conflict of interest statement

The authors report no conflict of interest.

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