Acute mitral regurgitation
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ABSTRACT
The diagnosis of acute mitral regurgitation (MR) is often missed or delayed because the clinical presentation is substantially different from that in patients with chronic MR. Management of acute MR depends on the specific aetiology of valve dysfunction and there is a lack of consensus on the optimal therapeutic approach in many patients. In particular, management of secondary MR due to acute ischaemia is challenging because of unique mechanisms of valve incompetence compared with chronic ischaemic MR. Another clinical challenge is management of acute MR due to transient systolic anterior motion of the mitral valve in the acute phase of Takotsubo cardiomyopathy, which commonly resolves within a few weeks. Additionally, iatrogenic MR induced by intraventricular devices is a recently recognised aetiology of acute MR. Acute primary MR typically requires early surgical intervention, for example, with a flap leaflet or endocarditis, because of acute cardiovascular decompensation with an abrupt increase in left atrial pressure. In an emergency situation and high surgical risk, a percutaneous mitral valve edge-to-edge repair is an alternative therapeutic option. Firm diagnosis of the severity and aetiology of acute MR is necessary for proper decision making, including timing and types of surgical intervention.

OVERVIEW
Acute mitral regurgitation (MR) is a medical and surgical emergency. Patients usually present with severe decompensated heart failure due to the sudden pressure and volume load imposed on a previously normal left atrium. In addition, there is an acute reduction in forward cardiac output because a major proportion of the blood pumped by the non-dilated left ventricles goes backwards into the left atrium instead of forward into the aorta. However, acute MR is often misdiagnosed as acute pulmonary disease in the emergency department because of severe dyspnoea, an abnormal chest radiograph and the absence of an audible cardiac murmur. Acute MR typically is ‘silent’ in severely deteriorated hemodynamic conditions because of the equalisation of left ventricular and left atrial pressure in mid-systole leading to a soft or absent murmur. However, when the differential diagnosis includes acute MR, the diagnosis can be made by transthoracic or transoesophageal echocardiography.

CAUSES
Identifying the cause of acute regurgitation is key to proper management of emergency haemodynamic deterioration (figures 1 and 2). The major causes of primary acute MR include spontaneous ruptured chordae tendineae, myxomatous degeneration of the leaflet and chordae, and infective endocarditis. Infective endocarditis can affect previously normal valves, in addition to valves with pre-existing anatomical abnormalities, and cause destruction of the leaflet tissue, often with leaflet perforation or inadequate apposition and coaptation in systole. Rapid valve destruction and haemodynamic deterioration can occur with bacteremia or sepsis; any delay in diagnosis further worsens the systemic condition.

Ischaemic papillary muscle rupture is another major aetiology of acute MR, which is often fatal. Papillary muscle rupture occurs more frequently in inferior myocardial infarction (MI) than in anterior MI, and can occur with a relatively small infarction and only a modest extent of coronary artery disease. Acute ischaemic MR can complicate acute MI even without papillary muscle rupture due to systolic leaflet tethering related to the sudden onset of regional or global left ventricular dysfunction. Although acute ischaemic MR usually is only mild to moderate in severity, in some patients it progresses to severe MR resulting in pulmonary oedema or cardiogenic shock in the acute phase of MI.

Penetrating or non-penetrating chest trauma, systemic inflammatory diseases such as systemic lupus erythematosus, or other systemic diseases are less common causes of acute MR. Acute rheumatic fever is a rare cause of acute MR in developed countries but remains a concern in endemic areas with severe leaflet destruction in the acute phase of the disease. Careful history taking and consideration of these causes is necessary for diagnosis of these uncommon aetiologies.

Another uncommon cause of acute MR is transient systolic anterior motion (SAM) of the mitral leaflet complicating Takotsubo cardiomyopathy. In the acute phase of Takotsubo cardiomyopathy, hyperdynamic motion of the basal left ventricle results in apparent SAM with consequent severe MR. Simultaneous occurrence of left ventricular dysfunction and severe MR causes severe congestive heart failure or cardiogenic shock.

Iatrogenic balloon-induced trauma during percutaneous mitral valvotomy for rheumatic mitral stenosis is a well known cause of acute MR, although the likelihood of this complication is low with careful patient selection. More recently developed therapeutic approaches, such as transcatheter prosthetic aortic valves and intracardiac left ventricular assist devices, have also been reported to cause acute MR in some cases, presenting with acute haemodynamic changes during the procedure.

CLINICAL PRESENTATION
Patients with acute MR usually present with acute onset of shortness of breath due to severe congestive heart failure. Sudden onset of severe MR in
Figure 1  Schematic presentation comparing acute and chronic mitral regurgitation (MR). In acute MR, the left atrium and left ventricle are normal in size or mildly dilated, and left ventricular contraction is commonly hyperkinetic. In chronic MR, the left atrium is enlarged to adapt to volume and pressure overload and the left ventricle gradually compensates by increase in volume along the course of the disease. LV, left ventricle; RV, right ventricle; LA, left atrium.

Figure 2  Schematic examples of the cause of acute mitral regurgitation. LV, left ventricle; PM, papillary muscle; SAM, systolic anterior motion.

Table 1  Typical clinical presentations and haemodynamics: acute versus chronic mitral regurgitation (MR)

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<th>Acute MR</th>
<th>Chronic MR</th>
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<tr>
<td>Onset</td>
<td>Sudden</td>
<td>Gradual</td>
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<tr>
<td>Symptoms</td>
<td>Severe dyspnoea</td>
<td>Gradually progressed dyspnoea after asymptomatic period</td>
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<td></td>
<td>Hypotension</td>
<td>Palpitation due to atrial fibrillation</td>
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<td>Cardiogenic shock</td>
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<td>Chest pain in cases of acute coronary syndrome</td>
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<tr>
<td></td>
<td>Pulmonary oedema</td>
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<td></td>
<td>Severe</td>
<td>Usually asymptomatic at rest</td>
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individuals with a previously normal heart is associated with no or little enlargement of the left atrium. The acute increase in left atrial volume in systole, without time for adaptive chamber enlargement, results in a rapid increase in left atrial pressure and development of pulmonary congestion. Similarly, the normal sized left ventricle cannot increase total stroke volume acutely so the forward stroke volume is reduced by the volume of blood ejected backwards into the left atrium. This sudden reduction in forward stroke volume leads to hypotension and cardiogenic shock (Table 1).

Physical examination reveals findings that are largely similar to those seen in acute pulmonary syndrome, including pulmonary congestion and severe pulmonary hypertension as a result of the acute severe reversal of flow into an unadapted left atrium. Auscultation is often elusive, typically with a soft early systolic murmur that may diminish or disappear in late systole as the reverse left ventricular to left atrial systolic pressure gradient declines to near zero due to the markedly increased left atrial pressure. In fact, a systolic murmur may be entirely absent even with severe regurgitation in patients with left ventricular systolic dysfunction or low systemic blood pressure. Approximately 30% of patients with acute MI with significant MR have no audible

Figure 2  Schematic examples of the cause of acute mitral regurgitation. LV, left ventricle; PM, papillary muscle; SAM, systolic anterior motion.
Even when the murmur is audible, the soft short murmur may not be recognised in an emergency condition in a patient with severe respiratory distress.

**HAEMODYNAMICS**

Left atrial compliance and left ventricular dilatation are important determinants of the haemodynamics in patients with severe MR. In chronic MR, the left atrium and left ventricle gradually compensate by an increase in volume over time. The enlarged compliant left atrium maintains low systolic pressure despite the regurgitation flow and the enlarged left ventricle increases total stroke volume to maintain normal forward stroke volume.

In contrast with acute MR, the left ventricle may initially compensate to some extent with hyperdynamic motion to increase stroke volume. However, the left atrium does not have time to adapt to sudden pressure and volume overload. This results in marked elevation of the mean left atrial pressure, and increase in phasic left atrial pressure, with a large ‘v-wave’ in late systole. Despite hyperdynamic left ventricular function, the normal sized left ventricle in patients with acute MR cannot provide enough forward stroke volume to compensate for the reverse stroke volume into the left atrium21 (figure 1).

**IMAGING**

In patients with acute MR, ECG findings are non-specific and chest radiographic findings are consistent with acute pulmonary oedema.22 Occasionally, pulmonary oedema is unilateral if the MR jet is directed eccentrically into either the right or the left pulmonary vein.23 24

**Echocardiography**

Transthoracic echocardiography can be performed at the bedside, even in the emergency room, and should be the first-line exam in the assessment of acute dyspnoea. The normal left atrial and left ventricular size with hyperdynamic left ventricular contraction on two-dimensional imaging may be misleading unless acute MR is superimposed on chronic MR or abnormal mitral leaflet motion is recognised. Colour Doppler echocardiography reliability identifies the presence of MR and provides a clue to the aetiology based on the size of the regurgitant orifice (flow convergence) and the regurgitant jet direction. Accurate quantification of regurgitant volume and effective regurgitant orifice area is often difficult in acute severe MR because of severe acute congestive heart failure with tachycardia but may not be necessary in an acutely ill patient with more than mild MR. The vena contracta width measurement and density of the continuous wave Doppler signal are the simplest techniques to quickly determine if significant MR is present. The continuous wave Doppler regurgitant flow velocity curve often shows the characteristic triangular (instead of rounded) shape due to a rapid decline in late systolic velocity reflecting the abrupt increase in left atrial pressure, consistent with a ‘v-wave’ (figure 3). Systolic reverse flow in the pulmonary veins may not be reliable with a rapid heart rate or atrial fibrillation. Calculation of regurgitant orifice areas and volume by the proximal isovelocity surface area...
Papillary muscle rupture with severe mitral regurgitation complicated to anterolateral acute myocardial infarction. Chest X-ray showed severe pulmonary congestion. ECG showed ST-T elevation in I, aVL, V5-6. Culprit coronary lesion was diagnosed as the diagonal branch (red arrow). Two-dimensional and three-dimensional transoesophageal echocardiography (TOE) show bileaflet prolapse and severe regurgitation with ruptured papillary muscle head (yellow arrows). Surgical findings revealed the completely ruptured papillary muscle head with twisted chordae tendineae. LV indicates left ventricle; LA, left atrium; MV, mitral valve; PM, papillary muscle.

approach can be challenging in the acute setting and may not be necessary.

Echocardiography also often identifies the cause of acute MR. Regional wall motion abnormalities of the left ventricle indicate underlying coronary artery diseases. A ruptured papillary muscle head (observed as a triangular mass) swings back and forth between the left atrium and the left ventricle with the entire leaflet prolapsing into the left atrium in systole (figure 4). Papillary muscle ischaemia may cause incomplete or impending papillary muscle rupture, which presents with relatively modest heart failure, not as dramatic as complete papillary muscle rupture. Newly developed leaflet prolapse during the acute phase of MI can be a sign of impending papillary muscle rupture which needs careful attention.

Ischaemic MR due to leaflet tethering will also be evident with wall motion abnormalities in the region of the culprit coronary artery and characteristic leaflet ‘tenting’ due to papillary muscle displacement. Apical shift of leaflet coaptation results in
a reduced coaptation area and regurgitation with a wide effective orifice along the curved coaptation line and a curtain-like regurgitant jet. Serial follow-up echocardiography is necessary in patients with acute ischaemic MR, as the degree of regurgitation can dramatically change along with left ventricular reverse remodelling or with progression of left ventricular remodelling during the course of the disease after primary revascularisation (Figure 5).

During placement of transcatheter devices across the aortic valve into the left ventricle, such as placement of a transcatheter aortic valve replacement (TAVR) or insertion of an axial flow left ventricular assist device, careful monitoring by transoesophageal echocardiography is critical to avoid irreversible mitral valve or subvalvular destruction. A pigtail catheter or curved guidewire in the left ventricle may inadvertently hook the chordae, resulting in substantial MR (Figure 6). Continuous monitoring by transoesophageal echocardiography is no longer performed at most centres during the TAVR procedure. However, frequent echocardiographic evaluation of the position of the transcatheter left ventricular assist device is recommended both for optimal positioning for flow and to ensure they do not affect the mitral apparatus. When device-related acute MR is identified, the echocardiographer should immediately share the critical situation with the operators and carefully guide them to adjust the device position to avoid impairment of mitral valve function.

Takotsubo cardiomyopathy may be complicated by transient SAM of the mitral valve resulting in severe MR. Typical findings of apical ballooning and hyperdynamic LV basal wall induce abnormal mitral valve motion in the acute phase with the colour Doppler echocardiography showing turbulent flow due to outflow tract obstruction and the posteriorly directed MR jet (Figure 7). In most cases, these findings spontaneously resolve within a few weeks.

**MANAGEMENT**

In acute MR, the most appropriate therapeutic strategy depends on the aetiology of valve dysfunction, degree of cardiac dysfunction, comorbid conditions and severity of haemodynamic compromise in each individual. Surgical correction is usually needed for acute primary MR, such as ruptured chordae, papillary muscle rupture or infective endocarditis.

A cohort study of 89,085 patients from the Society of Thoracic Surgery database investigated the impact of mitral disease aetiologies (hypertrophic obstructive cardiomyopathy, trauma, tumour or congenital), (6) degenerative primary MR, (7) chronic ischaemic MR, and (8) pure annular dilatation. Acute ischaemic MR was diagnosed when MI was recorded within 21 days preoperatively. Acute ischaemic MR with mitral valve replacement had the highest operative mortality (15.1%), whereas mitral valve repair in chronic degenerative prolapse had the lowest unadjusted operative mortality (1.2%). In patients with mitral stenosis undergoing transcatheter balloon valvotomy, acute MR is often fatal and surgical correction is required as early as possible.

Mitrval valve repair can be an option for many patients with ruptured chordae, while valve replacement is generally required in papillary muscle rupture or infective endocarditis with severe destruction of the valve leaflets and subvalvular apparatus. In most cases, 24 patients with acute ischaemic papillary muscle rupture suggest that operative risk and longer-term outcomes are acceptable and that incomplete papillary muscle rupture may be treated with mitral valve repair. The challenges in treating these seriously ill patients are illustrated by a case report of spontaneous acute papillary muscle rupture during pregnancy treated with emergency mitral valve repair via a minimally invasive right thoracotomy following caesarean delivery.

In patients with acute secondary MR or when the patient is too ill for immediate surgical intervention, pharmacological afterload reduction or mechanical circulatory support may be life-saving as bridges to recovery or to later surgical therapy. Mechanical support options include an intra-aortic balloon pump, venoarterial extracorporeal membrane oxygenation or a ventricular assist device. In some particular aetiologies without organic valve disorder such as ischaemic/functional MR or Takotsubo cardiomyopathy, medical treatment would be the choice to stabilise the heart failure condition, expecting the spontaneous recovery of the left ventricular function and then reduction of the acute functional regurgitation. In Takotsubo cardiomyopathy, initial diagnosis of SAM by echocardiography is important, as vasodilators, inotropic agents or intra-aortic balloon counterpulsation can worsen the patient’s clinical status.

**Revascularisation for acute ischaemic MR**

Acute ischaemic MR is associated with worse short-term and long-term prognoses even if the degree is only mild or moderate. Emergency coronary revascularisation can reduce the magnitude of regurgitation both in the early stage and the late stage after acute MI and may be associated with a beneficial effect on short-term and long-term mortality. Some studies suggest that revascularisation alone can reduce the degree of ischaemic MR in patients with acute MI and that earlier reperfusion time is associated with greater reduction in MR severity. Ischaemic MR can persist or worsen along with adverse left ventricular remodelling, despite successful primary coronary intervention.

A three-dimensional study of the mitral valve apparatus suggests that sudden-onset left ventricular dysfunction in acute MI causes loss of leaflet coaptation even with a relatively mild degree of valve tethering compared with the greater degree of leaflet tethering seen with chronic ischaemic MR. Another study indicated that acute MR related to MI is not due to papillary muscle displacement, as expected with chronic ischaemic MR, but is related to separation and excess angulation of the papillary muscle instead, likely due to new regional wall motion abnormality without global left ventricular remodelling.

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**Figure 7** Takotsubo cardiomyopathy and systolic anterior motion of the mitral valve (SAM). Left ventricular apical ballooning is seen as a typical finding of Takotsubo cardiomyopathy. SAM occurs due to hyperdynamic basal wall and causes severe MR. LV indicates left ventricle; RV, right ventricle.
Percutaneous transcatheter intervention for acute MR

Transcatheter mitral valve edge-to-edge repair and transcatheter mitral valve replacement are newly developed, less-invasive approaches for management of selected patients with chronic MR. There have been some reports of the use of transcatheter mitral valve repair to manage acute severe MR and cardiogenic shock, including in a few patients with papillary muscle rupture. Although surgical intervention should be the first-line treatment for acute severe MR, transcatheter repair has been suggested as an alternative or ‘rescue’ procedure for patients deemed too high risk for surgical intervention. Further studies evaluating the role of transcatheter approaches for management of acute MR are needed.

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REFERENCES


