Special situations in acute heart failure:

Acute Mitral regurgete

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Acute severe mitral regurgitation is a serious medical condition. Whilst clear guidelines exist regarding the management of chronic MR, acute severe MR is usually treated on an individual basis.

Etiology

- Native valve
- Prosthetic valve
- Primary
- Secondary (functional)
Native valve.. Main causes

- **Flail leaflet** due to myxomatous disease (mitral valve prolapse), infective endocarditis, or trauma.
- **Chordae tendineae rupture** due to trauma, spontaneous rupture, infective endocarditis, or acute rheumatic fever.
- **Papillary muscle rupture** or displacement due to acute myocardial infarction or severe ischemia or trauma.
- Don’t forget → **Iatrogenic causes esp. after BMV**
- N.B. Reported rare causes e.g. Takotsubo

Prosthetic valves

- **Tissue valve leaflet rupture** due to degeneration, calcification, or endocarditis.
- **Impaired closure of mechanical valve** (due to valve thrombosis, infection, or pannus formation).
- **Paravalvular regurgitation** due to infection or suture rupture (often related to a calcified or scarred annulus).
- **Strut fracture and disk escape** (?? with older generation mechanical valves).
Mortality/Morbidity

- The prognosis of patients with mitral regurgitation depends on the underlying etiologies and the state of the left ventricular function.
- Acute pulmonary edema and cardiogenic shock often complicate the course of acute regurgitation. The operative mortality in these cases approaches 80%. A patient with ruptured chordae tendineae and minimal symptoms has a much better prognosis.
- When ischemic heart disease is the mechanism for regurgitation, the extent of anatomic disease and left ventricular performance are prognostic determinants. Complicating events include sudden death and myocardial infarction.

Pathophysiology: AMR

- Flail leaflet
- Chordae tendineae rupture
- Papillary muscle rupture
- Abnormal reversal of blood flow from LV → LA.
- Sudden large Vol load imposed on LA & LV of normal size & compliance

↑ LA volume / LAP → Pulmonary congestion

↓ Forward SV

Compensatory ↑ HR

Neurohumoral Response:

↑ Vascular resistance

Shock
If the patient tolerates the acute phase, the chronic compensated phase begins.

- The chronic compensated phase results in eccentric left ventricular hypertrophy. The combination of increased preload and hypertrophy produces increased end-diastolic volumes, which, over time, result in left ventricular muscle dysfunction.

- This muscle dysfunction impairs the emptying of the ventricle during systole. Therefore, regurgitant volume and left atrial pressures increase, leading to pulmonary congestion.
CLINICAL MANIFESTATIONS

- Usually a **cardiac emergency** with the sudden onset and rapid progression of pulmonary edema, hypotension, and signs and symptoms of cardiogenic shock
- May not be so dramatic if acute MR is superimposed upon chronic MR or the patient is younger and physically fit
- ??Unilateral Pulmonary edema

Cardiac auscultation

- **S3** may be heard
- Pressure gradient between the left atrium and ventricle diminishes or disappears by the end of systole (combination of a low systemic blood pressure and elevated left atrial pressure), the systolic murmur is often soft, low pitched and decrescendo, ending before A2 → **Approximately 50% of patients with moderate to severe MR have no audible murmur**
- Best heard along the left sternal border and base of the heart, generally without a thrill, and may radiate to the back. It can be confused with an acute ventricular septal defect. In PML involvement, the murmur could be heard at base of heart.
- Sometimes murmur has seagull character (rupture chordae) or musical (flail leaflet)

"In acute mitral regurgitation, the examination usually is consistent with acute pulmonary edema and left ventricular failure"
Chest radiograph

- Normal size cardiac silhouette, with severe left-sided congestive heart failure and pulmonary edema.
- An enlarged left ventricle and atrium may be present if chronic MR has been present prior to the acute event.

So......

- History is not helpful
- Cardiac examination has low sensitivity
- Chest Xray may be misleading
Patients who received an echocardiogram early (in the emergency room) have a correct admitting diagnosis and proper management.

Delayed recognition and initial misdiagnosis often results in morbid outcomes.

**Echocardiography (TTE / TEE)**

- Diagnosis, mechanism, and etiology
- Left atrial size may be normal
- Left ventricular size is normal
- Systolic function is normal or hyperdynamic
- Findings are related to the etiology of acute MR:
  - Evidence of a flail mitral leaflet when the etiology is papillary muscle or chordal rupture.
  - Vegetations on the leaflets may be seen in patients with endocarditis.
- The severity of regurgitation is evaluated with Doppler studies.
### Table 4: Echocardiographic criteria for the definition of severe valve regurgitation: an integrative approach (adapted from Lancellotti et al. 

<table>
<thead>
<tr>
<th>Qualitative</th>
<th>Aortic regurgitation</th>
<th>Mitral regurgitation</th>
<th>Tricuspid regurgitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valve</td>
<td>Abnormal/flat/aortic defect</td>
<td>Aortic/aortic/atrial annulus defect</td>
<td>Abnormal/flat/aortic defect</td>
</tr>
<tr>
<td>Colour flow jet</td>
<td>Large in central jet, variable in eccentric jet</td>
<td>Very large central jet or eccentric jet, abutting, shearing, and reaching the perimeter wall of the LA</td>
<td>Very large central jet or eccentric jet, abutting wall of LA</td>
</tr>
<tr>
<td>CW signal of regurgitant jet</td>
<td>Dense</td>
<td>Dense/dense</td>
<td>Dense/dense with early peaking (peak &lt; 3 ms in mitral TR)</td>
</tr>
<tr>
<td>Other</td>
<td>Rheoblastic flow reversal in descending aorta (S/DV &gt; 30 cm/s)</td>
<td>Large flow convergence zone*</td>
<td>–</td>
</tr>
</tbody>
</table>

**Quantitative**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Aortic</th>
<th>Mitral</th>
<th>Tricuspid</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valve width (mm)</td>
<td>&gt;6</td>
<td>&gt;7 (for transplantation)</td>
<td>&gt;8</td>
</tr>
<tr>
<td>Upstroke venous flow*</td>
<td>–</td>
<td>Systolic pulmonary venous flow reversal</td>
<td>Systolic hepatic venous flow reversal</td>
</tr>
<tr>
<td>Flow</td>
<td>–</td>
<td>E-wave dominant ± 0.5 m/s</td>
<td>E-wave dominant ± 0.5 m/s</td>
</tr>
<tr>
<td>Other</td>
<td>Pressure half-time &lt; 500 ms</td>
<td>TV-in/TV-systolic &gt; 1.6</td>
<td>PSA radius &gt; 10 mm</td>
</tr>
</tbody>
</table>

**Thresholds**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Primary</th>
<th>Secondary</th>
</tr>
</thead>
<tbody>
<tr>
<td>EROA (cm²)</td>
<td>&gt;30</td>
<td>&gt;20</td>
</tr>
<tr>
<td>Regurgitant volume (mL/day)</td>
<td>&gt;60</td>
<td>&gt;30</td>
</tr>
<tr>
<td><em>Additional of aortic chamber size</em></td>
<td>LV</td>
<td>LVEF</td>
</tr>
</tbody>
</table>

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* CW = continuous wave; S/DV = early-diastolic velocity; EROA = effective regurgitant orifice area; LA = left atrium/left, LV = left ventricle/left ventricle, PSA = proximal isovelocity surface area; LA = left atrium; LV = left ventricle/ventricle; RV = right ventricle; TR = tricuspid regurgitation; TV = tricuspid velocity integral.

* For patients between age 4 and 20 years old.

* Unless otherwise specified, mitral flow time is determined using pulsed wave Doppler.

* If the absence of other causes of elevated RA pressure.

* Pressure half-time is shortened with increasing LV diastolic pressure, vasodilator therapy, and in patients with a dilated compliant aorta, or lengthened in chronic atrial fibrillation.

* Based on Nyquist limit shift of 29 cm/s.

* Different thresholds are used in secondary mitral regurgitation where an EROA > 30 cm² and regurgitant volume > 100 mL identify a subset of patients at increased risk of cardiac events.
Figure 3. Transesophageal three-dimensional real-time echocardiography, showing the ruptured papillary muscle head protruding during systole into the left atrium (large arrow) as visualized from the left atrium. Note that the ruptured head is hanging from a band of chordae tendineae. Next to the head is the flail P2 scallop, which entirely lacks supporting subvalvular tissue (small arrow).
Cardiac catheterization???

- Ventriculography {not recommended}→ immediate, complete, and usually persistent opacification of the left atrium on the first beat after ventricular injection. Opacification of the pulmonary veins is also frequently seen due to the high atrial pressures during systole
- Coronary angiography.. Preoperative and for ischemic mitral regurge
Management

The most important step in management is to ... **DIAGNOSE**

Emergency Department Care

- Acute mitral regurgitation is a specific case of acute heart failure in which immediate intervention in the ED can make a difference.
- The treatment of pulmonary edema should include oxygen, diuretics, nitrates, and early intubation if respiratory failure results.
- If the etiology is myocardial infarction, infusion of thrombolytics/or primary PCI may reestablish the blood flow to the papillary muscle, possibly restoring function.
- The mainstay of medical treatment in most other cases of mitral regurgitation is **afterload reduction**.
- Afterload reduction decreases the impedance to left ventricular ejection and, as a result, decreases the regurgitant volume.
- These individuals can benefit from afterload reduction with nitroprusside, even in the setting of a normal blood pressure.
- Acute functional mitral regurgitation in the presence of acute cardiomyopathy or decompensated heart failure can respond to aggressive heart-failure therapy.
Nitroprusside should not be given as monotherapy in patients who are hypotensive at presentation → inotropic agent such as dobutamine, intraaortic balloon pump (IABP) is often inserted {can be continued into the early postoperative period until hemodynamics stabilize}

**Surgery** (Mortality rates as high as 50%)

- **Chordal rupture:**
  - often with mitral valve repair → lower operative mortality, improved preservation of left ventricular function, better long-term survival, and risks of a prosthetic valve and anticoagulation are avoided

- **Endocarditis:**
  - Emergency surgery for acute mitral regurgitation due to endocarditis if refractory heart failure or an intracardiac fistula is present

*If possible, mitral valve repair is preferred to mitral valve replacement as it is associated with a risk of infection of prosthetic materials*
Ischemic MR

- Percutaneous revascularization may lead to resolution of the MR → medical therapy and an IABP may be used during the acute episode with weaning of these modalities as myocardial function improves.
- Papillary muscle rupture occurs in 1% to 3% of MIs and is associated with a mortality rate of 80% with medical therapy alone. Historically, operative mortality rates were as high as 67%, and patients were frequently denied surgery for this reason. With the addition of CABG to mitral valve replacement or repair, the operative mortality rate is now less than 10%.
- Partial papillary muscle rupture → may stabilize the patient and delay surgery for 6-8 weeks after myocardial infarction to avoid operating on the necrotic myocardial tissue. Valve repair is preferred, but myocardial necrosis may necessitate valve replacement.

In patients with acute functional mitral regurgitation, therapy is directed at restoring blood flow to the territory at risk.

- Acute mitral regurgitation in patients presenting with shock is a sign of a poor prognosis: an observed mortality rate of 55% improved to only 39% in patients who were selected for emergency surgery.
- The Shock trial determined that early revascularization improved outcomes at 6 months in patients with cardiogenic shock and acute MI. Upon further analysis, the prognostic importance of mitral regurgitation to the short- and long-term survival of such patients was inversely related to the degree of regurgitation.
- This suggests that aggressive treatment with early revascularization may improve survival in patients with severe mitral regurgitation after acute MI. Therefore, if revascularization is needed, the presence of severe mitral regurgitation should prompt surgical intervention.
Surgical versus medical management of patients with acute ischemic mitral regurgitation: a systematic review

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Abstract

Aims: Acute ischemic mitral regurgitation (MR) is seen in patients with myocardial infarction and is associated with increased morbidity and mortality. The optimal treatment strategy of this condition however, is not well established. The aim of this manuscript is to conduct a systematic review of the medical literature to assess the relative benefits and harms of mitral valve surgery with medical therapy versus medical management alone for patients with acute ischemic MR of at least moderate severity.

Methods: We performed a literature search in MEDLINE, Embase.com, and Cochrane Central Register of Controlled Trials. We restricted the search to randomized clinical trials comparing surgical to medical management of acute ischemic MR. Exclusion criteria included non-randomized trials, trials enrolling patients with non-ischemic MR, and trials excluding acute ischemic MR. The primary outcomes were short-term and long-term mortality. Two reviewers (WA, WM) screened titles and abstracts of identified citations independently and in duplicate using calibration exercises and standardized screening forms.

Results: The search strategy identified 887 citations (137 were duplicates and removed). Of the 750 titles, 719 were excluded (519 were non-relevant and 190 were review articles and case reports). Of the 41 remaining abstracts, 17 were retrospective cohorts and four excluded acute MR, leaving no eligible study for analysis. An ongoing study that is being conducted at Southern Illinois University entitled by “Medical Versus Surgical Management of Patients With Moderate Mitral Regurgitation Following Percutaneous Coronary Intervention for Myocardial Infarction: A Pilot Prospective Randomized Trial” was identified; however, it was just withdrawn after failing to enroll patients during 4-years.

Conclusion: This is an empty systematic review that identified no published randomized trials for the management of acute MR complicating acute MI. The only ongoing randomized study that was identified was just withdrawn after failing to enroll patients. There is an urgent need for conducting proper randomized trials in order to guide informed decision making in the treatment of acute ischemic MR.

PROSPERO registration number: CRD42013005943

Keywords: Mitral regurgitation, Acute myocardial infarction, Mitral valve surgery, Mortality

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Fig. 1 Proposed algorithm for management of patients with acute ischemic mitral regurgitation. MR, mitral regurgitation; MVR, mitral valve replacement/repair; RCT, randomized clinical trial.
In summary,

- Although acute, severe valvular regurgitation can be a true surgical emergency, accurate diagnosis and subsequent treatment decisions require clinical acumen, appropriate imaging, and sound judgment.
- An accurate and timely diagnosis is essential for successful outcomes and requires appropriate expertise and a sufficiently high degree of suspicion in a variety of settings.
- Whereas cardiovascular collapse is the most obvious and common presentation of acute mitral regurgitation, findings may be subtle, and the clinical presentation can often be nonspecific. Consequently, other acute conditions such as sepsis, pneumonia, or nonvalvular heart failure may be mistaken for acute valvular regurgitation.
The Egyptian museum (tahrir, Cairo)

The new Grand Egyptian museum (under construction)