

MITRAL REGURGITATION

ETIOLOGIES

ACUTE	CHRONIC
Annulus disorder <ul style="list-style-type: none"> Endocarditis (abscess) Trauma (surgery) Paravalvular leak by suture interruption (surgery) 	Inflammatory <ul style="list-style-type: none"> Rheumatic heart disease Lupus Scleroderma
Leaflet disorder <ul style="list-style-type: none"> Endocarditis (perforation) Trauma (BMV) Tumor (atrial myxoma) Myxomatous degeneration LED – Libman-Sacks lesion 	Degenerative <ul style="list-style-type: none"> Myxomatous degeneration (Barlow, MVP) Marfan Ehlers-Danlos Pseudoxanthoma elasticum Calcification of mitral annulus
Rupture of chordae tendineae <ul style="list-style-type: none"> Spontaneous Myxomatous degeneration (prolapse, Marfan, Ehlers-Danlos) Endocarditis Acute rheumatic fever Trauma 	Infective <ul style="list-style-type: none"> Endocarditis
Papillary muscle disorder <ul style="list-style-type: none"> ACS Acute LV dysfunction Infiltrative disease (sarcoidosis, amyloidosis) Trauma 	Structural <ul style="list-style-type: none"> Rupture chordae tendineae (spontaneous or secondary to ACS, trauma, MV prolapse, endocarditis) Rupture or dysfunction of papillary muscle Dilation of mitral valve annulus and LV cavity Hypertrophic cardiomyopathy Paravalvular prosthetic leak
Primary mitral valve prosthetic disorder <ul style="list-style-type: none"> Cusp perforation (endocarditis) Cusp degeneration Mechanical failure Immobilized disc or ball 	Congenital <ul style="list-style-type: none"> Mitral valve clefts or fenestrations Parachute mitral valve with endocardial cushions defects, endocardial fibroelastosis, Transposition of great arteries Anomalous origin of left coronary artery

Carpentier's classification

- Type I:** normal leaflet motion – jet is central
- Type II:** increased leaflet motion (leaflet prolapse) – jet is directing towards **opposite** side
- Type IIIa:** restricted leaflet motion during diastole and systole – jet is central or same side
- Type IIIb:** restricted leaflet motion predominantly during systole (ischemic) – jet is same side

Primary MR: degenerative (Barlow, IE, connective disease)

Secondary MR: functional (LV dysfunction...)

Coronary artery disease

30% patients with CAD have some degree of MR

- Tethering of posterior leaflet because of regional LV dysfunction
- Worse MR because associated to LV remodeling and systolic dysfunction

- Also ischemic damage to papillary muscles, dilation of mitral valve ring and/or loss of systolic annular contraction contributing to MR
- Severe MR associated to poor prognosis
- 20% MR after ACS – more adverse outcomes

PATHOPHYSIOLOGY

- 50% regurgitant volume is ejected in LA before aortic valve opening
- Orifice size and pressure gradient are labile – gradient depends on SVR and mitral annulus
- Severe MR – **LV volume overload** (↑ preload and afterload)
- LV and LA compliance increases = low LV filling pressure - ↑ LVEDV = **chronic compensated stage of severe MR**
 - ↑mitral annulus + regurgitant orifice = ↓ contractility LV
 - Eccentric hypertrophy (↑ mass)
- LA can dilate and cause afib
- Eventually LV function deteriorates, EF falls, ESV and EDV ↑, LV compliance ↓, LV filling pressure ↑ → pulmonary congestion with mild effort and ↓ CO
- ↓ **SV in late in chronic severe MR**
 - ↓EF reflect impaired myocardial function (contractility) and patients do poorly after surgical correction of MR → EF < 35% = high risk patients without post-op benefits
 - **LVESV or diameter**: predictor of function and survival after surgery (> 40mm)

Acute MR

- Sudden onset severe reflux into normal sized LA
- Signs and symptoms depend on size and compliance of pre-existing LA

CLINICAL SYMPTOMS

- Fatigue (**low CO**), left heart failure symptoms, palpitations (AF), and pulmonary congestion
- Right heart failure present in acute MR

PHYSICAL EXAM

JVP, carotid, precordial motion directly related to severity of leak not cause

CAROTID

- Mild – normal
- Moderate-severe – brisk, often ↓ pulse volume (if presence of heart failure)
 - b/c N or decrease forward SV ejected more rapidly than normal during early systole
- Severe – quick rising, poorly sustained, low amplitude

JVP

- Large V waves
- AF – loss a, V more prominent

PRECARDIAL

LV impulse

- Hyperdynamic
- Displaced
- Dilated
- +/- Palpable S3 – early diastole, pt hold breath end-exp (decubitus)

Parasternal impulse

- Pulm HTN – sustained RV lift, holosystolic
 - More common in combined MR/MS

- Also loud P2, RV S4, TR/PR
- Systolic expansion of **enlarged LA** = late systolic thrust

S1

- ↓S1 amplitude (masked by murmur)
- Loud if holosystolic prolapse of MV

S2

- Severe – wide split and early A2 (shortening of LV ejection)
- P2 louder if pHTN $P2 > A2$

S3

- **Common** – means large regurgitation volume
- Also present if LV dysfct with dilation

S4

- LV S4 never seen in rheumatic MR b/c LA dilated and can't generate force
- S4 common in **acute or papillary muscle dysfunction due to cardiomyopathy**
- RV S4 if PHTN (louder with insp, max intensity at LLSB)

MURMUR

SYSTOLIC

- Starts with S1 and extends to S2 – **beyond A2** (pressure gradient between LV-LA after Ao valve closure)
 - 50% of entire regurg vol reflux to LA before AV open
- **Holosystolic**
 - **Late systolic**: mild MR – PVM or papillary muscle dysfunction
 - **Early systolic**: Acute MR – diamond shaped decreasing in late systole (↑ pressure non compliant LA)
 - **Holosystolic**: Severe MR – long murmur + diastolic rumble
- High pitch
- Loudest at **apex**
- Radiate
 - Axilla or left scapula (murmur directed **posteriorly** – **anterior leaflet involvement**)
 - Towards sternum and base (murmur directed **anterior** – **prolapse of posterior leaflet**)
- Ruptured chordae – murmur usually harsh, loud (>gr 3)
- **Not amplified post PVC**
- Regurgitant murmur – fremitus
- ↓ upward position, Valsalva
- ↑ handgrip, squatting
- Little correlation between intensity of systolic murmur and severity of MR
- Silent MR: LV dilatation, ACS, paraprosthetic valvular regurgitation, emphysema, obesity, chest deformity, prosthetic valve

DIASTOLIC

- short mid-diastolic flow murmur, brief, low- to med-pitched apically best heard with bell when pt decubitus, light pressure - ↑ flow during filling in diastole

DIFFERENTIAL DIAGNOSIS

TR

- hard to separate if RV enlarged
- TR usually increase with **inspiration** (Carvallo), MR softer

VSD

- maximum location at LLSB

HOCM

- also has long SEM
- may not have typical radiation
- attn to change with posture, maneuver, drugs

ddx MR and **AS** – carotid, length of murmur, murmur post PVC (murmur increases with AS)

ECHOCARDIOGRAPHY

Primary MR

- Assess LV size and function, RV function, left atrial size, PA pressure, mechanism and severity of primary MR
- CMR is indicated in chronic primary MR to assess LV and RV volumes, function or MR severity

Secondary MR

- Assess the etiology of chronic secondary MR and extent and location of wall motion abnormalities and LV function, severity of MR and pulmonary hypertension.

★ **Suggested reference:** Zoghbi W. et al. Recommendations for the evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. 2003 J Am Society Echocardiography;16:777-802.

Poor prognosis when ERO ≥ 20 mm²

TEE

- Not recommended for routine evaluation and follow-up
- Performed when TTE images are **inadequate**
- Useful in **IE** – better visualization of underlying infected structures
- More precise quantification of regurgitation severity and better evaluation of likelihood of success of MV repair

Follow-up

- Severe primary MR (C1): annual or biannual
- Moderate MR: 1-2 years
- Mild MR: 1-2 years

★ **Suggested reference:** Nishimura R. A. et al. 2014 ACC/AHA guidelines for the management of patients with valvular heart disease. J Am Coll Cardiol 2014;63:e57-185 – **Tables 15-16**

CATHETERIZATION

- Ventriculography and hemodynamic measurements when noninvasive testing are inconclusive
 - o Severity of MR
 - o LV function
 - o Need for surgery
- Discrepancy between noninvasive testing and clinical symptoms

★ **Suggested reference:** Nishimura R. A. et al. 2014 ACC/AHA guidelines for the management of patients with valvular heart disease. J Am Coll Cardiol 2014;63:e57-185.

EXERCISE TESTING

Can be used to establish the presence of symptoms in patients with chronic primary MR and exercise tolerance.

TREATMENT

★ **Suggested reference:** Nishimura R. A. et al. 2014 ACC/AHA guidelines for the management of patients with valvular heart disease. J Am Coll Cardiol 2014;63:e57-185 – **Table 17, 18, figure 4**

MEDICAL

Acute MR

- Vasodilators
- IABP
- Prompt mitral surgery

INTERVENTIONS

Primary MR

MV repair >> MV surgery if possible (especially when **posterior leaflet** involved)

- LVEF greater than 30% and symptoms
- LVEF between 30-60% and asymptomatic, with LVESD of 40 mm or more

Surgical repair

- Pliable valves
- Degenerative MR due to MVP
- Annular dilatation
- Papillary muscle dysfunction due to ischemia or rupture
- Chordal rupture
- Perforation of mitral leaflet cause by IE

Older valves with calcium, deformed, rigid, severe subvalvular chordal thickening and loss of leaflet substances = MVR necessary

↓ **EF 10% if valve apparatus not preserved!**

Minimal invasive procedures vs. conventional sternotomy showed similar performance results when performed by experienced surgeons.

Annuloplasty and repair of posterior leaflet shows better outcomes than MVR – mortality < 1%, 95% freedom of reoperation, 80% with MR < 3/4 at 15-20 years post-op.

Annuloplasty and repair is more complex with 2 leaflets but shows better outcomes than MVR – 80% freedom of reoperation, 60% with MR < 3/4 at 15-20 years post-op. Durability of the procedure is uncertain.

MitraClip

- Clip is safe (Everest I) but less effective than surgical repair (Everest II)
- Residual MR by creating 2 regurgitant orifice – similar to Alfieri procedure
- **Reduce symptoms by reducing MR, reverse LV remodeling**
- For patients with chronic severe MR with symptoms NYHA 3-4 despite medical therapy for HF and are not candidate for surgery
 - o Degenerative MR with malcoaptation A2-P2, functional MR
 - o Coaptation depth < 11 mm
 - o Coaptation length > 2 mm
- **Class IIb** indication in guidelines

★ **Suggested reference:** Mauri L. et al. 4-Year results of a randomized controlled trial of percutaneous repair versus surgery for mitral regurgitation. J Am Coll Cardiol 2013;62:317-328.

Secondary chronic MR

- Reasonable if severe secondary MR undergoing surgery for CABG
- If patient has persistent symptoms despite optimal medical therapy
- Recent data not conclusive....

★ **Suggested reference:** Asgar A. et al. Secondary mitral regurgitation in heart failure. *J Am Coll Cardiol* 2015;65:1231-48.

Content of this summary from these references:

- Otto C & Bonow R. Valvular Heart Disease. (2012) In Bonow R. *et al.* Braunwald's Heart Disease, 9th edition, pp. 1468-1539. Philadelphia, PA: Elsevier.
- Nishimura R. A. et al. 2014 ACC/AHA guidelines for the management of patients with valvular heart disease. *J Am Coll Cardiol* 2014;63:e57-185.
- Zoghbi W. et al. Recommendations for the evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. 2003 *J Am Society Echocardiography*;16:777-802.