DEFINITION

Mitral stenosis is condition of pathological narrowing of mitral valve. Normal square area of mitral valve is 4-6 square cm. When it is reduced to half of it, significant hemodynamic changes take place (Fig. 2.1).

ETIOLOGY

Commonest - Rheumatic heart disease
Rare causes
1. Congenital
2. Lutembacher’s syndrome (ASD with congenital or acquired mitral stenosis).
3. Infective endocarditis with large vegetations causing obstruction.
4. Endomyocardial fibrosis.
5. Hurler’s syndrome.
7. Methysurgide.

Pathology

1. Commissural fusion is the hallmark.
2. Thickening of leaflets and fibrosis.
3. Stenosis develops after a latest period of about 10 years.

Fig. 2.1: Mitral valve stenosis
HEMODYNAMICS OF MITRAL STENOSIS (FIG. 2.2)

I. *Rise in left atrial pressure*: Mitral valve narrowing—backflow—rise in LA pressure.

II. *Pulmonary venous congestion*: Raised LA pressure is transmitted back to pulmonary veins (because no valves at their junction with LA) thus increasing pressure in pulmonary venules and capillaries-fluid transudate into interstitial space, alveoli and causes pulmonary edema and engorgement of lymphatics. Congested bronchial veins cause airway obstruction.

III. *Rise in pulmonary artery pressure*: Rise in pulmonary venous pressure causes constriction of corresponding pulmonary arterioles eventually causing pulmonary arterial HT.

IV. *Redistribution of blood flow to upper lobes*: High left atrial pressure and high hydrostatic pressure affects lower lobe vessels most. So pulmonary arterial vasoconstriction occurs most in lower lobes and most of blood flow as a compensation is diverted to upper lobe.

V. *Rt ventricular failure*: Pulmonary arterial hypertension causes strain to emptying of Rt.V and eventually Rt heart failure.

*Left Ventricular Function Remains Normal*

**Fig. 2.2: Hemodynamics of mitral stenosis**

CLINICAL PICTURE

Symptoms

Develop 10-20 years after initial rheumatic insult.

- Dyspnea on exertion.
- Orthopnea, paroxysmal nocturnal dyspnea and symptoms of acute pulmonary edema.
Fatigue due to low cardiac output.
Palpitation due to AF
Hemoptysis due to various mechanisms.

Signs

I. Peripheral
   - Mitral facies with malar flush, and small volume pulse, peripheral cyanosis.

II. Cardiovascular:
   1. Tapping apical impulse.
   2. Diastolic thrill may be present at the apex.
   3. Parasternal heave due to RVH.
   4. Auscultation (Fig. 2.3)
      - Loud I sound at apex.
      - Opening snap.
      - Mid diastolic murmur at apex.
      - Presystolic accentuation.

Mechanism and description of clinical features

Loud Ist sound: High left atrial pressure holds the mitral valve wide open until the end of diastole and ventricular systole slams it shut.

Opening snap: Produced by sudden bellowing down of mitral cusps into left ventricle at the end of isometric relaxation period.
Loud I sound and opening snap indicate that the valve is pliable and not fibrosed or calcified.

Opening snap is a short, sharp, snapping and high pitched sound – best heard at apex but also heard over left lower parasternal area and may extend to cardiac base.

Fig. 2.3: Auscultatory features of mitral stenosis
**Mitral Stenosis**

**Diastolic murmur**
Low pitched, described as rumble.
Begins with opening snap and has varying duration.
Usually occupies mid diastole.
Best heard with bell stethoscope in left lateral position with breath held in expiration.
Usually grade 2 or 3 but may be loud or faint;
Usually accentuates before I heart sound.

**Pre-systolic accentuation**
Produced by LA contraction; may be absent in AF.
Not always absent in AF; persists in short diastole (due to high velocity).

*How to demonstrate diastolic murmur in sub clinical MS?*
After exercising the patient put him in left lateral and auscultate.

**Pulmonary area**
When pulmonary hypertension develops?
II heart sound splits,
P₂ becomes loud,
Rarely Graham Steell murmur of pulmonary insufficiency occurs- caused by dilatation of pulmonary artery.

*All auscultatory features of pulmonary hypertension are listed below:*
- Loud P₂, II heart sound split narrowly.
- Pulmonary ejection click.
- Ejection systolic murmur increasing with inspiration.
- Graham Steell murmur early diastolic murmur at pulmonary area.
- Murmur of tricuspid incompetence.
- Rt atrial Gallop.


**Variability** in auscultatory features- that may occur:
Soft I sound- rigid /acuffic valve.
In severe MS, MDM may be absent/soft.
PSM absent when dominant mitral regurgitation is pressure
Silent MS-Tight MS with large RV displacing LV posteriorly and so masking the murmur.

**Diagnostic Features**
Loud I sound at apex
Opening snap
Low pitched rumbling mid diastolic murmur with presystolic-accentuation: best heard, with patient in left lateral position with breath held in expiration.

**Differential Diagnosis for Clinical Picture of Mitral Stenosis**

1. LA myxoma,
2. Ball valve thrombus,
3. Cor triatriatum.
Left atrial myxoma - murmur varies with posture.

**Differential Diagnosis—For Mid Diastolic Murmur**

1. Mitral stenosis.
2. Tricuspid stenosis (murmur accentuating with inspiration)
3. Flow murmurs (caused by increased blood flow across the mitral or tricuspid valve during diastole in the following)
   - Mitral regurgitation
   - Tricuspid regurgitation
   - ASD
   - VSD
   - PDA.
4. *Carey coomb’s murmur*: In acute rheumatic mitral valvulitis - functional murmur.
5. *Austin Flint murmur*: Occurs in free aortic incompetence; functional murmur mid diastolic murmur at apex (see Fig. 6.3).

**Other Conditions Causing Loud I Sound**

1. Tachycardia.
2. Hyperdynamic states (valve remains open at end diastole and is forcibly closed by hyper contractile LV).

**Other Conditions Causing Opening Snap**

Tricuspid stenosis, myxoma.

**Assessments in Mitral Stenosis**

1. Degree of stenosis (severity)
2. The mobility of mitral valve
3. Pulmonary hypertension
4. Presence of other lesions and dominance.

A. *Degree of stenosis* is assessed by:
   1. $S_2 - OS$ interval which is inversely proportional to severity
      
      Usual range of $S2 - OS$ interval is 0.08 – 0.12 sec; 0.06 and less in severe.
2. Duration of murmur: It is directly proportional to severity: longer the duration severe the stenosis.
3. Degree of LA enlargement in X-ray and ECG and by Echo.
4. Presence of pulmonary hypertension in pure MS.

B. **Mobility of valve** is denoted by:
   1. Presence of loud I sound
   2. Opening snap
   3. Absence of valve calcification.

In combined mitral lesion factors which denote *dominant MS* are:
- Presence of opening snap
- Loud I sound
- Presystolic accentuation
- Absence of III ht sound
  (Which mitral lesion causes giant LA? Mitral regurgitation).

**Complications of mitral stenosis**

I. Complications of pulmonary venous congestion
II. Complications of enlarged left atrium
III. Complications at valve level.

**Complications of Pulmonary Venous Congestion**
- Paroxysmal nocturnal dyspnea
- Acute pulmonary edema
- Hemoptysis
- Pulmonary arterial hypertension
- Right heart failure
  Precipitants of pulmonary edema: AF, pregnancy, exercise, emotion, chest infection, anesthesia.

**Complications of Enlarged Left Atrium**
- Atrial fibrillation –deteriorates the condition suddenly.
- LA thrombus formation with thromboembolic signs
  - Cerebral
  - Renal
  - Coronary
  - Peripheral
  - Leriche’s syndrome
- Pressure on esophagus causing dysphagia
- Pressure on L. recurrent laryngeal nerve causing hoarseness of voice
  – Ortner’s syndrome
- Lifting up of L main bronchus – causing emphysema.
Complications at Valve Level

- Infective endocarditis – rare.

Causes of hemoptysis: Rupture of bronchial vein, pulmonary edema, pulmonary infarct, bronchitis, PHT.

INVESTIGATIONS

X-ray Chest

Shows features of mitralisation of heart:
1. Straightening of left border of the heart (hypoplastic aorta, prominent MPA, enlarged left atrial appendage, all these 3, come in line with LV border and so straightening).
2. Features of pulmonary hypertension: Dialated main pulmonary artery, narrowing of peripheral pulmonary arteries.
3. Features of pulmonary venous congestion: Thin short horizontal lines just above costophrenic angle representing interlobar septae thickened by edema and fibrosis-Kerley b lines near costophrenic angle; perpendicular to pleural surface.
4. Pulmonary edema: Perihilar congestion, haziness spreading from hilum to periphery – “bat’s wing appearance” and nodular opacities.
5. LA enlargement seen as opacity within RA opacity.
6. Redistribution of blood from the base to the apex causing enlarged upper lobe bronchial veins.
7. Left upper lobe bronchus elevated causing compensatory emphysema.
8. Hemosiderosis consequent feature of repeated pulmonary congestion.

Barium Swallow

Enlarged LA causes esophageal indentation in right oblique view. X-ray and barium swallow contraindicated in pregnancy.

ECG

P pulmonale/P mitral; RVH with Rt axis deviation, atrial fibrillation; biphasic P wave.
P mitrale: P wave duration >120 ms; prominent notched P
P pulmonale: Peaked P waves, amplitude >2.50 m V.

ECHO

Confirms the diagnosis:
- Assesses the severity
- Shows presence or absence of thrombus/calcification
- Shows other associated lesions if any.
Also indicates mitral valve area, mitral valve morphology.

Echocardiographic mitral morphology score is derived from following features:
1. Leaflet rigidity,
2. Leaflet thickening,
3. Valvular calcification, and
4. Subvalvular disease each graded as 1+ to 4+; score >8 fare badly.

Doppler

Shows (1) pressure gradient across mitral valve and (2) pulmonary artery pressure.

TEE

Transesophageal echocardiogram: Shows LA thrombus, endocarditis.

Catheterization

1. Shows pressure gradient between LA and LV; measures PA, LA, LV diastolic pressures – Catheterization is indicated when clinical and echo/Doppler data are discordant.
2. Helps to assess MR severity in those posted for balloon valvotomy especially when clinical and other data are discordant.

Multislice CT: A new modality to assess mitral valve area.

TREATMENT

Medical:
Treatment of cardiac failure
Treatment of arrhythmia – atrial fibrillation
Treatment of pulmonary edema
Prophylaxis/treatment of infective endocarditis
Secondary prophylaxis for rheumatic fever.

Avoid:
Anemia, tachyarrhythmia and vigorous exercise which can precipitate cardiac failure.

INTERVENTION

1. Timing—mild/moderate symptoms
2. MVarea <1 cm²/m²
3. Delay worsens prognosis.
4. If patient is symptomatic despite medical treatment.
Mitral Balloon Valvuloplasty

Indications for:
- Significant symptoms class II-IV
- Isolated mitral stenosis
- No or trivial mitral regurgitation
- No calcification or rigidity of the valve-valve must be mobile
- Left atrium-free of thrombus
- Pulmonary hypertension 50-60 mm of Hg
- Valve area of,<1sq cm.

Technique of Balloon Valvuloplasty

Valvotomy is done by inflating a balloon mounted on a cardiac catheter, via rt femoral vein balloon mounted catheter is passed trans septally; Advanced into LA over a curly guide wire, now the distal portion of balloon is inflated slightly and balloon is advanced up to apex of LV. Then balloon is gradually withdrawn, until positioned across mitral valve. At this position the balloon is inflated procedurally, splitting the commissural adhesions.
- Valvuloplasty only requires light sedation, like for cardiac catheterization.
- After 7-10 years of valvuloplasty, valve usually restenoses and mitral valve replacement is indicated.

Complications of Balloon Valvuloplasty

Mitral regurgitation
Left to Rt shunt
Thromboembolism
Pericardial tamponade
Myocardial infarction.

Surgery for Mitral Stenosis

Mitral Valvotomy

Surgical separation of fused commissures
- Adhesions that cause the mitral orifice to be narrowed, are lysed mechanically (by hand or by dilator). Valvotomy can be closed or open.
- Valvotomy is done if facilities or expertise for balloon valvuloplasty is not available.

Closed Valvotomy

Prerequisite: Flexible, non-calcified valves with no LA thrombus. Performed through a left thoracotomy without a bypass.
Contraindication are same as for balloon valvuloplasty and additional contraindications are chest deformity, severe lung disease and elderly frail patients.
Closed valvotomy is rarely performed now a days in developed countries.

Open Valvotomy
Prerequisite: Flexible, non-calcified valves.
Performed on a cardiopulmonary bypass through a mid sternotomy.
Indications: Failure of balloon valvuloplasty/closed commissurotomy.
Associated mild MR, LA thrombus.

After valvuloplasty or valvotomy-
Antibiotic prophylaxis for endocarditis is a must and yearly follow up for restenosis is done.

MITRAL VALVE REPLACEMENT

Indications
1. Restenosis
2. Severe advanced MS Valve area ≤ 1sq cm (But patient must have good LV function).
3. Class II, III, IV symptoms.
4. Severe MS unfit for valvotomy or valvuloplasty: calcified, rigid valve
5. Severe associated MR, thickened fused chordae
6. Pulmonary artery systolic pressure of > 60-80 mm of Hg.
7. Recurrent thrombo embolic manifestations.

Types of Prosthetic Valves (Fig. 2.4)
1. Bioprosthetic valve
2. Mechanical prosthetic valve.

Fig. 2.4: Prosthetic valves
Mechanical

*Caged Ball valve*: e.g. Starr-Edward valve:
- Advantage—Good durability
- Disadvantage—Bulky and space occupying.

**Complications**
- Periprosthetic mitral regurgitation
- Hemolytic anemia
- Jaundice
- Thromboembolism
- Life long anticoagulation needed
- Infective endocarditis
- Paravalvular ring abscess.

*Tilting disc valve*: e.g. St. Jude valve, Björk-Shiley valve (not being used now)

*Bioprosthetic valve*:
- Autograft: from patient’s pericardium
- Advantage—anticoagulant’s not so much necessary: disadvantage—not durable: just 10 years or less
- Porcine tissue—thromboembolism less.

Following valve replacement long term anticoagulant therapy is indicated.
- Patient with prosthetic valve replacement – put on warfarin for rest of their life.
- But xenograft (porcine) or homograft (cadaver) do not require anticoagulation, except in presence of large LA and AF.

*Note:* Patients after valvuloplasty/valvotomy require prophylaxis for inf. endocarditis.

**Complications of Valve Replacement**
- Restenosis
- Thromboembolic phenomenon
- Hemolytic anemia
- Mechanical or material defects of valve itself.
  - e.g., failure of closure or opening/detachment partial/full.

**SUMMARY**

i. In most case of MS: *Balloon valvotomy* is successful.
ii. If valve anatomy is unfavourable for valvotomy: *valve replacement*

*Timing* of valve replacement:
- a. NYHA functional class 3 or 4.
- b. Pulmonary hypertension (pulmonary arterial systolic pressure <50 mm Hg)
Mitral Stenosis

Bioprosthetic valve can degenerate within a decade and necessitate-reoperation with attendant increased surgical risk.

iii. In young women who wish to bear children:
  • If in these women balloon valvuloplasty is not feasible, valve replacement is advised but it is complicated: Mechanical valve needs anticoagulation; warfarin causes
    – High incidence of fetal malformation; heparin has been shown to have
    – Serious complication.

Management of Mitral Stenosis

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Indications</th>
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<tbody>
<tr>
<td>1. Percutaneous balloon mitral vavotomy</td>
<td>Symptomatic NYHA class II, III, IV</td>
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<tr>
<td></td>
<td>Mitral valve area &lt;1 cm²</td>
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<tr>
<td></td>
<td>With no MR, no LA thrombus, no calcification</td>
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<td>2. Mitral valve repair for MS. Closed or open mitral valvotomy</td>
<td>NYHA Class III-IV</td>
</tr>
<tr>
<td></td>
<td>Mitral valve area &lt;1 cm²</td>
</tr>
<tr>
<td></td>
<td>If balloon valvotomy is not available</td>
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<tr>
<td></td>
<td>Valve morphology favourable for repair with no MR, no calcification, no LA thrombus.</td>
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<tr>
<td>3. Mitral valve replacement for MS</td>
<td>NYHA Class III-IV</td>
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<tr>
<td></td>
<td>Moderate/severe MS with valve area &lt;1 cm² who are unfit for valvotomy or repair</td>
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<td>-Those with LA clot or mitral calcification or rigid mitral valve combined MS and MR</td>
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<tr>
<td></td>
<td>Mitral valve restenosis.</td>
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</tbody>
</table>

Note:
In addition to factors mentioned above, various parameters like EF, LVEDD, PHT, AF, Valve morphology and others have to be considered in details in deciding about surgery.