RHEUMATIC HEART DISEASE
ORDER OF VALVULAR INVOLVEMENT IN RHD

Commonest acquired heart disease in children
Chronic sequelae of rheumatic fever
1) Valve involvement
Order of valve involvement in RHD is
   – MV ➔ AV ➔ TV ➔ PV
     – Why because the stress for the valves are only when they are closed

2) Pericardial involvement
MITRAL STENOSIS

- Primarily a result of rheumatic fever
- Scarring & fusion of valve apparatus
- Pure or predominant MS occurs in approximately 40% of all patients with rheumatic heart disease
- Two-thirds of all patients with MS are female
- Reduction of valvular size to 25% or less
- Rarely it may occur in 6 months to 2 years (juvenile mitral stenosis)
MITRAL STENOSIS: PATHOPHYSIOLOGY

- Normal valve area: 4-6 cm²
- Mild mitral stenosis:
  - MVA 1.5-2.5 cm²
  - Minimal symptoms
- Mod mitral stenosis
  - MVA 1.0-1.5 cm² usually does not produce symptoms at rest
- Severe mitral stenosis
  - MVA < 1.0 cm²
RHEUMATIC MS

Mitral 'Stenosis'

Normal
PATHOLOGY

- Fibrosis of the mitral ring with contracture of valve leaflets, chordae and papillary muscles and commissural fusion
**MITRAL STENOSIS : PATHOPHYSIOLOGY**

<table>
<thead>
<tr>
<th>Right Heart Failure:</th>
<th>Pulmonary HTN</th>
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<tbody>
<tr>
<td>Hepatic Congestion</td>
<td>Pulmonary Congestion</td>
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<tr>
<td>JVD</td>
<td>LA Enlargement</td>
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<tr>
<td>Tricuspid Regurgitation</td>
<td>Atrial Fib</td>
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<tr>
<td>RA Enlargement</td>
<td>LA Thrombi</td>
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- RV Pressure Overload
- RVH
- RV Failure

- LA Pressure

- LV Filling
MITRAL STENOSIS : SYMPTOMS

• Palpitations
• Cough
• Left sided failure
  – Orthopnea
  – PND
• Pulmonary infection
• Right sided failure
  – Hepatic Congestion
  – Edema

• Atrial fibrillation
• Systemic embolism
• Worsened by conditions that ★ cardiac output.
  – Exertion, fever, anemia, tachycardia, Atrial fibrillations, pregnancy, thyrotoxicosis
• Hemoptysis
RECOGNIZING MITRAL STENOSIS

**Palpation:**
- Small volume pulse
- Tapping apex-palpable S1
- +/- palpable opening snap (OS)
- RV lift
- Palpable S2

**ECG:**
- LAE, A Fib, RVH, RAD
Auscultation:

- Loud S1- as loud as S2 in aortic area
- A2 to OS interval inversely proportional to severity
- Diastolic rumble: length proportional to severity
- In severe MS with low flow- S1,
- OS & rumble may be inaudible
PHYSICAL EXAMINATION

- S1_____________S2__OS________________S1

- First heart sound (S1) is accentuated and snapping
- Opening snap (OS) after aortic valve closure
- Low pitch diastolic rumble at the apex
- Pre-systolic accentuation (esp. if in sinus rhythm)
MITRAL STENOSIS: NATURAL HISTORY

- Progressive, life long disease,
- Usually slow & stable in the early years.
- Progressive acceleration in the later years
- 20-40 year latency from rheumatic fever to symptom onset.
- Additional 10 years before disabling symptoms
INVESTIGATION

ECG :-

• LAE
• RVH
• Premature contractions
• Atrial flutter and/or fibrillation
  – ↑ freq. in pts with mod-severe MS for several years
  – A fib develops in ≈ 30% to 40% of pts with symptoms
• X ray chest
• Echocardiogram
MITRAL STENOSIS: THERAPY

• Medical
  – Diuretics for LHF/RHF
  – Digitalis/Beta blockers/CCB: Rate control in A Fib
  – Anticoagulation: In A Fib
  – Endocarditis prophylaxis
• Balloon valvuloplasty
  – Effective long term improvement
MITRAL STENOSIS: THERAPY

- Surgical
  - Mitral commissurotomy - ideal for pliable valve
  - Mitral Valve Replacement - calcified valve
    - Mechanical
    - Bio prosthetic
MITRAL REGURGITATION
PATHOLOGY

• Loss of valvular structure and shortening of chordae tendinae
MR PATHOPHYSIOLOGY

- Chronic LV volume overload $\rightarrow$ compensatory LVE initially maintaining cardiac output

- Decompensation (increased LV wall tension) $\rightarrow$ CHF

- LVE $\rightarrow$ annulus dilation $\rightarrow$ increased MR

- Backflow $\rightarrow$ LAE, Afib, Pulmonary HTN
MR SYMPTOMS

• Similar to MS
• Dyspnea, Orthopnea, PND
• Fatigue
• Pulmonary HTN, right sided failure
• Hemoptysis
• Systemic embolization in A Fib
RECOGNIZING CHRONIC MR

- Pulse:
  - brisk, low volume
- Apex:
  - hyperdynamic
  - laterally displaced
  - palpable S3, +/- thrill
  - late parasternal lift 2° to LA filling
- S 1 soft or normal
- S 2 wide split (early A2) unless LBBB
- Murmur-Fixed MR:
  - pan systolic
  - Loudest, apex to axilla
  - no post extra-systolic accentuation
- Murmur-Dynamic MR(MVP)
  - mid systolic
  - +/- click
  - $\uparrow$ upright
- S 3 / flow rumble if severe
RECOGNIZING ACUTE SEVERE MR

• Acute severe dyspnea, CHF & hypotension
• LV size normal
• LV may/may not be hyper dynamic
• Loud S1
• Systolic murmur may/may not be pansystolic
• Inflow/rumble
• S3 present-may be only abnormality

• RV lift
• Chordal or papillary muscle rupture/tear
  – Infarction with papillary muscle ischemia or tear
  – Infectious endocarditis with leaflet perforation or disruption or chordal tear
  – Flail MV segment
RECOGNIZING ACUTE SEVERE MR

- **ECG:**
  - LA enlargement
  - A fib
  - LVH (50% pts. With severe MR)
  - RVH (15%)
  - Combined hypertrophy (5%)

- **CXR:**
  - ↑ LV
  - ↑↑ LA
  - ↑ pulmonary vascularity
  - CHF
MR ECHOCARDIOGRAM
MR STAGES

LV size and function defined by echo

• Stage 1-compensated:
  – End-diastolic dimension less 63mm, ESD less 42mm
  – EF more than 60

• Stage 2-transitional
  – EDD 65-68mm, ESD 44-45mm, EF 53-57

• Stage 3-decompensated
  – EDD more than 70mm, ESD more than 45mm, EF less than 50
MITRAL VALVE SURGERY

• Only effective treatment is valve repair/replacement
• Optimal timing determined:
  – Presence/absence of symptoms
  – Functional state of ventricle
  – Feasibility of valve repair
  – Presence of A fib/PHTN
  – Preference/expectations of patient
MV REPAIR VS REPLACEMENT

- Lower operative mortality
- Better late outcome
- Curative
- Avoids anticoagulation unless atrial fibrillation
- Open A fib ablation
• Valve replacement:
  – Mortality 2-7%
  – Anti-coagulation
  – Decreased LVEF
• Tissue prosthetic valve degeneration
• Mechanical prosthetic valve dysfunction/thrombosis

• Valve repair
  – Mortality 2-3%
  – No anticoagulation (unless Afib)
  – Preservation of LVEF
• Valve repair always preferable
  – Feasible in 70-90% of patients
PATHOPHYSIOLOGY

• Sclerosis of aortic valve and retraction of cusps
• Left ventricular enlargement
• Mild cases are asymptomatic
SYMPTOMS

• Orthopnea
• Wide pulse pressure
• Exertional angina
• Heaving apex
• Blowing early diastolic murmur in aortic area - best heard in expiration
• Austin Flint murmur – apical presystolic murmur due to large flow across mitral valve
INVESTIGATIONS

• X-ray
• ECG
• Echocardiogram
• Cardiac catheterization
TREATMENT

• Rheumatic prophylaxis
• Decongestive-vasodilators, ACE inhibitors (digoxin increases the regurgitation)
• Infective endocarditis prophylaxis
• Valve replacement if left ventricular function is progressively reducing
AORTIC REGURGITATION
SYMPTOMS

- Orthopnea
- Wide pulse pressure
- Exertional angina
- Heaving apex
- Blowing early diastolic murmur in aortic area - best heard in expiration
- Austin Flint murmur – apical presystolic murmur due to large flow across mitral valve
INVESTIGATIONS

- X-ray
- ECG
- Echocardiogram
- Cardiac catheterization
TREATMENT

• Rheumatic prophylaxis
• Decongestive- vasodilators, ACE inhibitors (digoxin increases the regurgitation)
• Infective endocarditis prophylaxis
• Valve replacement if left ventricular function is progressively reducing
VALVULOPLASTY
PERICARDITIS
PATHOPHYSIOLOGY

• Noninflammatory involvement of pericardium
• Accumulation of fluid in the cavity more than 10 to 15 ml
PERICARDITIS
CLINICAL FEATURES

- Precordial pain
- Cough
- Dyspnea
- Pericardial rub
INVESTIGATION

• X ray chest – water bottle appearance of heart
• ECG – low voltage of QRS complexes, elevation of ST segment and T wave inversion
• Echocardiography
• Pericardial tap
TREATMENT

- Bed rest
- Rheumatic prophylaxis
- Steroids
- Aspiration of pericardial effusion
THANK YOU