Disclosures

None
Objectives

• Review the following stenotic lesions:
  – Aortic stenosis
  – Mitral stenosis

• Review the following regurgitant lesions:
  – Aortic regurgitation
  – Mitral regurgitation
  – Tricuspid regurgitation
Aortic Stenosis

• Causes
• Clinical Presentation
• Diagnosis
• Disease Course
• Treatment
Causes of Aortic Stenosis

• Calcification of a trileaflet valve
• Calcification of a bicuspid valve
  – Represents 1% of the population
  – 50% of all AVRs are performed for bicuspids
• Rheumatic valve disease
Aortic Sclerosis- A precursor to AS

• Often seen on echocardiogram or computed tomography
  – Focal thickening and increased echogenicity in the leaflets seen on imaging
  – No significant obstruction to left ventricular outflow
Clinical Presentation of AS

• Symptoms:
  – Often asymptomatic on presentation, noted by physical exam or findings on echocardiography
  – Classically:
    • Angina, heart failure, or syncope
    – Decreased exercise capacity
    – Exertional dyspnea
    – Chest pressure
    – Lightheadedness
Clinical Presentation of AS

• Physical Exam
  – Systolic ejection type murmur, loudest at base with radiation to carotids or LV apex
  – Delayed and diminished carotid upstroke
Diagnosis

- Echocardiography is the gold standard
- Left heart catheterization used if echocardiography nondiagnostic
- Computed tomography
Disease Course of AS

### Classification of Aortic Stenosis Severity

<table>
<thead>
<tr>
<th></th>
<th>Aortic Velocity (m/s)</th>
<th>Mean Transaortic Gradient (mm Hg)</th>
<th>Aortic Valve Area (cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>&lt;3.0</td>
<td>&lt;20</td>
<td>&gt;1.5</td>
</tr>
<tr>
<td>Moderate</td>
<td>3.0 - 4.0</td>
<td>20 - 40</td>
<td>1.0 - 1.5</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt;4.0</td>
<td>&gt;40</td>
<td>&lt;1.0</td>
</tr>
<tr>
<td>Very severe</td>
<td>&gt;5.0</td>
<td>&gt;60</td>
<td>&lt;0.7</td>
</tr>
</tbody>
</table>
Treatment of AS

• Medical therapy
  – No therapy slows progression
  – Concurrent risk factors should be addressed
    • Treatment of hyperlipidemia
    • Smoking cessation
    • Hypertension=double load for ventricle
    • With severe AS, avoid strenuous activity and competitive sports

• Aortic valve replacement
  – AVR indicated at symptoms onset with severe AS or with LV dysfunction
Mitral stenosis

- Etiology
- Diagnosis
- Treatment

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Mitral stenosis - Etiology

- Most commonly as a consequence of rheumatic fever
  - Group A hemolytic Streptococcus
  - Autoimmune process due to infection
  - Inflammation of heart valves
- Congenital MS
- Inflammatory diseases (SLE, RA)
- Carcinoid
- Infiltrative diseases (amyloidosis)
- Drug induced (methysergide)
- Radiation induced
Mitral stenosis - Diagnosis

• History
  – Symptoms of increased left atrial pressure and/or low cardiac output
    • Dyspnea, pulmonary edema, and hemoptysis
    • Hoarseness (enlarged atrium = compression of recurrent laryngeal nerve)
    • Fatigue
Mitral stenosis- Diagnosis

- Physical exam
  - Rales
  - Increased intensity of S1
  - Opening snap (OS) of mitral valve after S2
  - Low pitched holodiastolic decrescendo rumbling after the OS which increases in intensity at end diastole
  - Graham-Steele murmur also may be heard-high-pitched blowing murmur at the base (typically secondary to concomitant aortic regurgitation)
  - Long standing MS may cause secondary right heart failure as well (peripheral edema, hepatomegaly, JVD)
EKG and CXR in MS

• EKG: left atrial enlargement, right axis deviation, RV hypertrophy

• CXR:
  – Early: enlarged left heart silhouette, prominence of the main pulmonary artery, displacement of the esophagus
  – Later: Enlarged structures proximal to the mitral valve (PA, RV, LA, RA)
Mitral stenosis - Diagnosis

• Mainstay:
  – Echocardiography to assess
    • Mitral valve area
    • Consequences of mitral stenosis
    • Suitability for balloon valvuloplasty or surgery

• If echocardiography inconclusive
  – Discrepancy between noninvasive assessment and clinical findings
## Classification of Severity of Mitral Valve Stenosis

<table>
<thead>
<tr>
<th>Mitral Stenosis</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean gradient (mm Hg)</td>
<td>&lt;5</td>
<td>5-10</td>
<td>&gt;10</td>
</tr>
<tr>
<td>Pulmonary artery systolic pressure (mm Hg)</td>
<td>&lt;30</td>
<td>30-50</td>
<td>&gt;50</td>
</tr>
<tr>
<td>Valve area (cm$^2$)</td>
<td>&gt;1.5</td>
<td>1.0-1.5</td>
<td>&lt;1.0</td>
</tr>
</tbody>
</table>

Table 1
Classification of Severity of Mitral Valve Stenosis
Medical Therapy-

- Infective endocarditis prophylaxis no longer recommended unless high risk patient
- Prophylaxis for rheumatic fever
  - Primary prevention: benzathine PCN treatment for those with strep pharyngitis
  - Secondary prevention: Pts with prior history of rheumatic fever use of antibiotics as prophylaxis against repeat infections
Medical therapy in MS

• Patients in sinus rhythm
  – Diuretics, beta blockers (lengthen filling time)

• Patients in atrial fibrillation
  – Can cause sudden deterioration and acute pulmonary edema
    • Rate control with beta blocker or calcium channel blocker
    • If unstable cardioversion
  – Anticoagulation needed in those with MS and:
    • Afib
    • Prior embolic event
    • Left atrial thrombus
Invasive treatment of MS

• Percutaneous mitral valvuloplasty
  – Indicated for
    • Symptomatic patients
    • Asymptomatic pts with pulmonary hypertension, moderate or severe MS, and favorable morphology
  – Best for pliable, noncalcified valves with minimal fusion of the subvalvular apparatus and without significant mitral regurgitation
Surgical Therapy of MS

- Recent emphasis on valve sparing procedures
  - Commissurotomy
- Mitral valve replacement
Aortic regurgitation

- Etiology
- Pathophysiology
- Clinical presentation
- Clinical Assessment
- Management
# Etiology of Aortic regurgitation

<table>
<thead>
<tr>
<th>Primary Valve Disorder</th>
<th>Disorder of the Aortic Root</th>
</tr>
</thead>
<tbody>
<tr>
<td>Degenerative calcific stenosis</td>
<td>Degenerative (age-related) dilatation</td>
</tr>
<tr>
<td>Infective endocarditis</td>
<td>Degeneration of the extracellular matrix</td>
</tr>
<tr>
<td>Trauma</td>
<td>• Associated with Marfan’s or Loeys-Dietz syndromes</td>
</tr>
<tr>
<td>Congenital defects</td>
<td>• Associated with bicuspid aortic valves</td>
</tr>
<tr>
<td>• Unicuspid, bicuspid, quadricuspid aortic valve</td>
<td>Aortic dissection</td>
</tr>
<tr>
<td>• Rheumatic</td>
<td>Systemic hypertension</td>
</tr>
<tr>
<td>• Ventricular septal defect</td>
<td>Osteogenesis imperfecta</td>
</tr>
<tr>
<td>• Subaortic membrane</td>
<td>Syphilitic aortitis</td>
</tr>
<tr>
<td>Rheumatic heart disease</td>
<td>Ankylosing spondylitis</td>
</tr>
<tr>
<td>Myxomatous infiltration</td>
<td>Giant cell arteritis</td>
</tr>
<tr>
<td>Systemic disorders that affect the aortic valve</td>
<td>Behcet syndrome</td>
</tr>
<tr>
<td>• Lupus erythematosus</td>
<td>Psoriatic arthritis</td>
</tr>
<tr>
<td>• Giant cell arteritis</td>
<td>Relapsing polychondritis</td>
</tr>
<tr>
<td>• Takayasu’s arteritis</td>
<td>Reiter syndrome</td>
</tr>
<tr>
<td>• Ankylosing spondylitis</td>
<td></td>
</tr>
<tr>
<td>• Jaccoud’s arthropathy</td>
<td></td>
</tr>
<tr>
<td>• Whipple’s disease</td>
<td></td>
</tr>
<tr>
<td>• Crohn’s disease</td>
<td></td>
</tr>
<tr>
<td>Appetite suppressant drugs</td>
<td></td>
</tr>
</tbody>
</table>

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Pathophysiology of Chronic, Severe Aortic Regurgitation

1. Severe AR
2. LV dilates
3. Afterload mismatch/Drop in EF
4. Irreversible LV dysfunction

Asymptomatic → Heart Failure
Clinical Presentation of AR

• Chronic AR
  – Symptoms of heart failure
    • Shortness of breath at rest or with exertion
    • Palpitations (sinus tach with minimal exertion, PACs, or PVCs)
    • Syncope
    • Angina
Clinical Presentation of AR

• Chronic AR
  – Physical examination
    • Hyperdynamic apical impulse, displaced laterally and inferiorly
    • Diastolic murmur
      – Along LSB if related to primary valve issue
      – Along RSB if related to aortic root problem
      – Murmur increases with squatting or isometric exercise
      – Murmur decreases with decreases in afterload (decreased BP)
      – As becomes more severe, becomes holodiastolic
      – Associated Austin Flint murmur due to physiologic mitral stenosis (AR jet hits mitral valve)
Clinical Presentation of AR

• Acute Aortic regurgitation
  – LV has not had time to dilate
  – Symptoms primarily due to decreased stroke volume
    • Attempt to maintain cardiac output by increases heart rate
    • Tachypnea
    • Pulmonary edema
  – Physical exam reveals short diastolic murmur
Clinical Assessment of AR

- CXR: enlarged cardiac silhouette
- Echocardiography: mainstay of diagnosis
- Cardiac catheterization used if echocardiography inconclusive
Management of AR

• Pharmacologic treatment
  – Diastolic hypertension is treated with vasodilators
    • Nifedipine and ace inhibitors preferred
    • Avoid beta blockers

• Surgical treatment
  – Acute AR
    • Emergency surgery
      – Afterload reduction while awaiting surgery
      – Beta blockers contraindicated (compensatory tachycardia protective of cardiac output)
  – Chronic AR
    • Class I indications in:
      – Symptomatic severe AR
      – Asymptomatic severe AR with LV systolic dysfunction (<= 50%)
      – Chronic severe AR undergoing CABG or aortic surgery or valve surgery
Mitral regurgitation

- Etiologies
- Pathophysiology
- Diagnostic testing
- Treatment
- Surgery and Outcomes
Etiologies of Mitral Regurgitation

- Coronary artery disease (ischemic MR)
- Myxomatous degeneration
  - Mitral valve prolapse
- Nonischemic dilated cardiomyopathy
- Infective endocarditis
- Rheumatic heart disease
- Mitral annular calcification
- Connective tissue diseases
  - Rheumatoid arthritis
  - Systemic lupus erythematosus
  - Antiphospholipid antibody syndrome
- Radiation
- Drugs
  - Ergotamines
Pathophysiology of MR

• Chronic
  – Left ventricular volume overload → ventricular remodeling and eccentric hypertrophy → dilation of the LV without an increase in thickness to maintain cardiac output → over time this may lead to reduced contractility and systolic dysfunction
  – On exam: displaced apical impulse and holosystolic or late systolic murmur
  – Manifestations primarily depend on elevation of left atrial pressure
    • Pulmonary congestion
    • Pulmonary hypertension
    • Atrial fibrillation
Pathophysiology of MR

• Acute
  – Chordal or papillary muscle rupture from MI or infective endocarditis
    • Acute increase in left atrial and ventricular volume
    • Pulmonary venous pressure increases (pulmonary edema), stroke volume reduced
  • Compensatory increase in heart rate to maintain stroke volume
  • Physical Exam
    – Respiratory distress, tachycardia
    – S3 may be heard reflecting high LAP and rapid increase in LV diastolic pressure
Diagnostic testing in Mitral regurgitation

- Echocardiography defines
  - Severity
  - Etiology (abnormalities in the mitral valve apparatus, LV abnormalities)
  - Sequalae (PH, LAE, LV dilatation, LS systolic dysfunction)
Monitoring of MR

• ACC/AHA Class I recommendation
  • Severe MR (asymptomatic) = echocardiography every 6 months
  • Moderate MR (asymptomatic) should have echocardiography every 12 months
  • Asymptomatic, mild MR or mitral valve prolapse does not need routine echocardiography unless clinical status changes
Treatment of MR

• Acute, severe MR:
  – Afterload reduction to increase LV forward stroke volume and cardiac output (nitroprusside, intra-aortic balloon pump) as temporizing measures
  – Urgent surgery is the only definitive treatment

• Chronic, severe MR:
  – No real pharmacologic therapy used in primary valvulopathy causing MR, however if MR is secondary to LV dysfunction or CAD (functional MR) treatment is directed at treating primary pathology (beta blockers, ace inhibitors, or aldosterone antagonists)
  – Chronic, severe MR with NYHA Class II, III, IV symptoms → Surgery
  – Asymptomatic pts: LV systolic dysfunction and/or LV dilation
  – Asymptomatic, severe MR with new onset atrial fibrillation or evidence of pulmonary hypertension
Surgical treatment of MR

- Mitral repair is preferable to mitral replacement
  - Depends on anatomy, presence of calcification, operator and center experience
  - Decreased long-term complications
Tricuspid Regurgitation

- Causes
- Diagnostic testing
- Treatment
Causes of Tricuspid Regurgitation

• Functional
  – Dilated cardiomyopathy
  – Right ventricular dysplasia/cardiomyopathy
  – Pulmonary hypertension
  – Left to right intracardiac shunts (ASD or anomalous pulmonary venous return)

• Structural
  – Congenital (Ebstein’s anomaly, Tricuspid valve prolapse or cleft, TV hypoplasia)
  – Acquired (carcinoid, rheumatic, endocarditis, iatrogenic (radiation, pacemaker lead, drugs))
Diagnostic testing in TR

- Physical exam
  - Holosystolic murmur at the left sternal border which increases during inspiration (more venous return)
  - Severe TR may not have a significant murmur
  - JVD
  - Hepatomegaly
  - Lower extremity edema
  - Ascites
Diagnostic testing in TR

- Echocardiography
  - Gold standard to assess severity
  - Defines the cause
    - Primary vs functional
  - Also assesses hemodynamic consequence (RA or RV dilatation)
Treatment of TR

• Gentle diureses
  – Over diuresis can decrease LV stroke volume and cardiac output
  – In patients with pulmonary hypertension, treatment of PH can improve TR

• Surgical replacement (annuloplasty or tricuspid replacement)
  – Usually in conjunction with other valve surgery
  – IIa indication for symptoms and severe TR
References

- ACCSAP 8