Aortic valve disease

- Aortic stenosis.
- Aortic regurgitation.

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Aortic stenosis

- The normal aortic area is 3-4 cm², in severe valvular AS (< 1 cm²).
- Normally, little or no pressure difference across the aortic valve.
- In significant aortic stenosis, LV pressure may exceed that of the aorta by >50 mmHg.
- AS causes progressive obstruction of LV outflow tract resulting in pressure hypertrophy of LV.
- Untreated, As is assoc with significant morbidity & mortality.
Types Aortic stenosis

valvular  Subvalvular  Supravalvular

Congenital condition  (uncommon)

fibromuscular membrane
in the LVOT below AV
Etiology of Valvular AS

- Congenital
- Rheumatic (often coexist with AR & MVD)
- Bicuspid (1-2%, predominate in males, in 9% of 1rst degree, may be stenotic or regurgitant)
- Age-related calcific degeneration
Symptom of AS

- **Angina** (↓ myocardial perfusion, associated CAD (occurs in 40-80% of pts with angina & 25% in pts with out angina))
- **Syncope**
- **Heart failure**
- **Sudden death** (hypotension, arrhythmia due to ischaemia, LVH or impaired LVF)
Physical finding in AS

- Arterial examination:-
  Diminished & delayed carotid upstroke (pulsus parvus et tardus)

- Palpation :-
  None displaced, diffuse, sustained apical impulse, double apical impulse (palpable a wave or S4 caused by noncompliant LV).
  Systolic thrill in the 2ND Rt ICS.
Auscultatory finding in AS

Often present due to LVH & poor LV compliance

Intensity decreases as AS progresses

Murmur is systolic, harsh best at Rt upper sternal border radiates to carotids peaks in early to mid systole until late in the course when it peaks later & is more intense

S4 S1

Intensity decreases as LVF worsens

S2 becomes soft as A2 decreases in intensity. May paradoxically split as severity increases may become soft & single late in the course.

A2 P2

S2 S3

S3 may be present late in the course
Diagnostic testing of AS

- ECG (LA abnormality, LVH)
- CXR (boot shape, cardiomegally, calcification)
- Echocardiography (TTE, TEE)
- Cardiac catheterization.
Therapy of AS

**Medical**
- Antibiotic prophylaxis
- Asymptomatic: (1ry prevention of CAD maintenance of SR, blood pressure control,
- Symptomatic: treatment of heart failure

**Percutaneous aortic balloon valvuloplasty (PABV)**
- Pediatric congenital, noncalcific, Rh AS

**Surgical AVR** (main stay in severe AS)
Indication of PABV in adults with AS

- Bridge to surgical aortic valve replacement in haemodynamically unstable patient.
- Palliative therapy for nonoperative candidate
  - (high surgical risk due to multiple comorbidities or pt refuses surgery)
- Pts with critical AS who require urgent noncardiac surgery
Recommendation of AVR

- Symptomatic Pts with severe aortic AS
- Pts with severe AS undergoing CABG.
- Pts with severe AS undergoing aortic or other valve surgery.

Possibly indicated:
- Pts with moderate AS who require CABG or aortic or other valve surgery.

- A symptomatic Pts with severe AS &:
  - impaired LV F (EF < 50%)
  - symptoms, hypotension or ventricular arrhythmias during exercise.
Aortic regurgitation

- Can develop from
  - Primary disease of the valve leaflets
  - Abnormality of aortic root or ascending aorta.

- Can be acute or chronic
Failure of the aortic valve to close tightly causes back flow of blood into the left ventricle.
<table>
<thead>
<tr>
<th>Major causes of chronic AR</th>
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<tr>
<td><strong>Leaflet abnormalities</strong></td>
<td><strong>Aortic root or Ascending Aorta Abnormalities</strong></td>
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<tr>
<td>Rheumatic fever</td>
<td>Age related aortic dilatation</td>
</tr>
<tr>
<td>Infective endocarditis</td>
<td>Annuloaortic ectasia</td>
</tr>
<tr>
<td>Trauma</td>
<td>Cystic medial necrosis of aorta (isolated or marfan syndrome)</td>
</tr>
<tr>
<td>Bicuspid aortic valve</td>
<td>Systemic hypertension</td>
</tr>
<tr>
<td>Myxomatous degeneration</td>
<td>Aortitis (syphilis, giant cell arteritis)</td>
</tr>
<tr>
<td>Congenital aortic regurgitation</td>
<td>Reiter’s syndrome</td>
</tr>
<tr>
<td>SLE</td>
<td>Ankylosing spondylitis</td>
</tr>
<tr>
<td>Rheumatoid arthritis</td>
<td>Behçet syndrome</td>
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<tr>
<td>Ankylosing spondylitis</td>
<td>Psoriatic arthritis</td>
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<tr>
<td>Takayasu’s arteritis</td>
<td>Osteogenesis imperfecta</td>
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<tr>
<td>Crohn’s disease</td>
<td>Relapsing polychondritis</td>
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<tr>
<td>Drug induce valvulopathy</td>
<td>Ehlers–Danlos syndrome</td>
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# Major cause of acute AR

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<th>Aortic rot or ascending aorta abnormalities</th>
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<tr>
<td>Traumatic rupture</td>
<td>Acute aortic dissection</td>
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<tr>
<td>Acute infective endocarditis</td>
<td>Perivalvular leak or dehiscence of prosthetic valves</td>
</tr>
<tr>
<td>Acute prosthetic valve dysfunction</td>
<td></td>
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<tr>
<td>Poor aortic balloon valvuloplasty</td>
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</table>
## Symptoms of AR

<table>
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<th>Chronic AR</th>
<th>Acute AR</th>
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<td>Usually asymptomatic for along time.</td>
<td>Sudden haemodynamic deterioration hypotension, tachycardia, pallor, cyanosis, Diaphoresis, cool extremities, pulmonary congestion</td>
</tr>
<tr>
<td>After LV dysfunction – symptoms of pulmonary congestion - dyspnea, PND</td>
<td>Weakness, altered mental status, SOB syncope</td>
</tr>
<tr>
<td>Chest discomfort (due to LV enlargement exaggerated after PVCs &amp; in supine position)</td>
<td>If severe chest pain is present aortic dissection should be suspected.</td>
</tr>
<tr>
<td>Angina (uncommon, due to latent CAD, reduced diastolic coronary perfusion, nocturnal bradycardia, LVH, subendocardial ischaemia)</td>
<td></td>
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</table>
Physical findings in chronic AR

- Characteristic peripheral pulses, cardiac auscultatory findings.
- Pts with chronic AR should be examined for:-
  - Peripheral manifestation of infective endocarditis
  - Signs of Marfan syndrome.
  - Evidence of chronic aortic dissection
  - Signs for collagen vascular disorder.
### Physical signs associated with hyperdynamic pulse in chronic AR

<table>
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<tr>
<th>Physical sign</th>
<th>Description</th>
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<tbody>
<tr>
<td>Water hammer or corrigan’s pulse</td>
<td>Rapid upstroke followed by quick collapse</td>
</tr>
<tr>
<td>De Musset’s sign</td>
<td>Head bob with each heart beat</td>
</tr>
<tr>
<td>Traube’s sign</td>
<td>Pistol shot sounds heard over the femoral arteries in both systole &amp; diastole</td>
</tr>
<tr>
<td>Müller’s sign</td>
<td>Systolic pulsation of the uvula</td>
</tr>
<tr>
<td>Duroziez’s sign</td>
<td>Systolic murmur over the femoral artery when compressed proximally &amp; diastolic murmur when compressed distally or systolic-diastolic murmur with increasing compression over femoral artery</td>
</tr>
<tr>
<td>Quincke’s sign</td>
<td>Capillary pulsations visible in the lunula of the nail bed</td>
</tr>
<tr>
<td>Hill’s sign</td>
<td>Popliteal cuff systolic pressure &gt; brachial cuff systolic pressure by &gt;60mmHg</td>
</tr>
<tr>
<td>Becker’s sign</td>
<td>Arterial pulsations visible in the retinal arteries &amp; pupils</td>
</tr>
</tbody>
</table>
Auscultatory finding in AR

A2 may be soft due to poor coaptation
P2 may be obscured by the murmur

May be narrowly split or paradoxically split due to prolonged systolic ejection time

Murmur is diastolic, blowing, Decrescendo best heard at the Lt upper S B severity correlates more with duration than intensity

Note: - Austin flint murmur mid-late diastolic rumble (vibration of AML) may be audible

Intensity decreases as LVF worsens

Often present due to LVH & poor LV compliance
**Physical examination maneuver in AR**

<table>
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<tr>
<th><strong>Increase murmur</strong></th>
<th><strong>Decrease murmur</strong></th>
</tr>
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<tbody>
<tr>
<td>Isometric exercise (e.g., hand grip)</td>
<td>Standing from squatting position</td>
</tr>
<tr>
<td>squatting</td>
<td>Strain of Valsalva maneuver</td>
</tr>
<tr>
<td>Inotrope infusion</td>
<td>Inhalation of amyl nitrite</td>
</tr>
</tbody>
</table>
Physical findings in acute AR

- Signs of hyperdynamic circulation characterize chronic AR often absent.
- Pulse pressure normal or slightly wide.
- Heart size normal (undisplaced apex).
- S1 may be diminished due to preclosure of MV P2 may be loud, S3 (if cardiac decompensation).
- Murmur is shorter & lower in pitch, if severe may not be audible.
- Systolic flow murmur may be audible.
- Austin flint, if present, is shorter.
Diagnostic testing of AR

- **ECG**: LVH, LAD, Lt atrial abnormality, in acute AR non-specific ST-T changes.
- **CXR**: cardiomegaly (acute AR may be normal) pulmonary congestion.
- **Echo**: TTE (2D M mode, Doppler color flow), TEE.
- **Cardiac catheterization**: coronary angiography to exclude CAD.
Medical Therapy of AR

**Chronic AR :-**

- Antibiotic prophylaxis.
- Drugs: (vasodilator (hydralazine, ACEI, CCB)) to slow progression of LV dysfunction & LV dilatation, B blocker in in aortic root dilatation.

**Acute AR :-**

- IV vasodilators & inotropic agents in pt with cardiogenic shock.
- Antibiotic if assoc with infective endocarditis.
- Management of acute AR Usually surgical according to the cause.
# Indications of aortic valve replacement in AR

## Class 1
- NYHA class 111 or 1V or CHA class 11 to 1V symptoms (with or without CAD) with normal LVF (EF ≥50%)

## Class 11a
- NYHA class 111 & preserved LV systolic function (EF ≥50%) with progressive LV dilatation or declining EF at rest or declining exercise tolerance.

## Other indications
- A symptomatic or symptomatic pts with mild to moderate LV dysfunction at rest (EF 25 -49%)
- Pts undergoing CABG or surgery in the aorta or other heart valves.
## Surgical therapy of AR

### Indications of aortic valve replacement in AR (continued)

<table>
<thead>
<tr>
<th>Class 11b</th>
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<tr>
<td>Pts with severe LV dysfunction (EF &lt; 25%)</td>
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<tr>
<td>Asymptomatic pts with normal LVSF at rest &amp; progressive LV dilatation which is moderately severe (EDD 70-75, ESD 50-55% mm)</td>
</tr>
<tr>
<td>Asymptomatic pts with normal LVSF at rest but with decline in EF during (stress echo or exercise radionuclide angiography)</td>
</tr>
<tr>
<td>Asymptomatic pts with normal LVSF at rest &amp; LV dilatation which is not severe (EDD &lt; 70 mm, DSD &lt; 50 mm)</td>
</tr>
</tbody>
</table>
Pt with chronic AR should be observed closely for the development of LV systolic dysfunction.

Follow up evaluation typically is conducted with serial echocardiography.

If signs of LV systolic dysfunction develop, surgical therapy should be considered.
THANKS