BICUSPID AORTIC VALVE

EPIDEMIOLOGY AND PATHOPHYSIOLOGY

1-2% population
Men >>> women (70-80% cases)
Familial clusters – autosomal dominant inheritance with incomplete penetration (20-30%)
2 cusps:
- Fusion of R and L cusps in 70-80%
- Fusion of R and NC cusps in 20-30% (more dilatation of ascendant aorta)
- Fusion of L and NC = rare
Diagnosis based on systolic opening of the valve with only 2 commissures in TTE
Unicuspid valves: 1 leaflet with 1 aortic commissure

CLINICAL MANIFESTATIONS

Normal valve function until later in life – related to valvular dysfunction
Risk factors of cardiac events: ≥ 30 y-o, moderate to severe AR, and moderate or severe AS
- 20% of bicuspid will develop severe AR requiring AVR between 10-40 y-o
- Increase risk of endocarditis
- Severe stenosis later in life > 50 y-o
  o Pathology identical to trileaflet valves BUT
  o Earlier in life because of turbulent flow through abnormal architecture that cause accelerated valvular changes leading to AS

DIAGNOSIS & FOLLOW-UP

No specific gene testing.
Recommendation: ETT - 1st degree family members if associated aortopathy, family history of VHD or aortopathy.
Angio CT and MRI – aortic diameters 1-2 mm larger because it includes the aortic wall


TREATMENT

- No medical treatment proven to reduce the aortic dilatation progression
- Treat if hypertension

Surgical intervention

Content of this summary from these references: