VALVULAR HEART DISEASE

RHEUMATIC AND NON-RHEUMATIC

PROF. E N OGOLA

LECTURE OBJECTIVES

At the end of the lecture the student should be able to:

- Describe the aetiology of VHD esp RHD
- Describe the pathology of valvular damage esp in RHD
- Describe the pathophysiological consequences of valvular disease
- > Elicit the clinical features of VHD
- Carry out investigations in VHD
- Outline the management of a patient with VHD

PREAMBLE

- Predominantly rheumatic in this environment
- Most important cause of cardiac disease in teenagers, young adults
- Epidemiology reflects Rh. Fever: underprivilege, overcrowding, young age group
- Management unavailable, expensive, complex, risky
- Hence the significance of prevention primary, secondary
- Other causes:
 - -Congenital
 - -Degenerative
 - -Ischaemic, inflammatory etc

PREAMBLE 2 RHD

- Consequence of rheumatic endocarditis
- Only long term sequel of RF
- Fibrosis, distortion of valvular apparatus
- Stenosis, requrgitation or both
- Most often affects left heart valves
- Mitral > Aortic
- Tricuspid often functional
- Almost never pulmonic
- Involvement often multiple
- Rheumatic fever prophylaxis

Specific valvular lesions

- FUNCTIONAL ANATOMY:
 - -Leaflets, commissures, chordea tendinae
 - **-MVA Normal 4-6 cm2**
 - 1-2cm2 significant stenosis
 - < 1cm² critical stenosis
- AETIOLOGY:
 - Almost invariably rheumatic
 - Congenital, Calcification, Inflammatory
- PATHOLOGY:
 - Commissural fusion
 - Fibrosis, distortion of chordae, leaflets

MITRAL STENOSIS PATHOPHYSIOLOGY

Impaired opening of MV

- Inadequate LA emptying:
 - Increased LA pressures
 - Pulmonary venous congestion
 - Pulmonary Hypertension > RV hypertrophy and failure
 - Increased LA size
 - LA thrombosis
 - Atrial fibrillation
- Inadequate LV filling
 - Low cardiac output

- CLINICAL FEATURES SYMPTOMS:
 - Effort intolerance: dyspnoea, easy fatiguablity
 - Palpitations, may be at rest
 - Cough, haemoptysis
 - Orthopnea, PND
 - Abdominal discomfort, swelling

- CLINICAL FEATURES SIGNS:
 - Mitral facies =
 - Low volume pulse, rapid, ± irregular
 - Apex NOT displaced, tapping (palpable S1)
 - Palpable P2
 - Left parasternal heave (RVH)
 - Auscultation:
 - Loud S1, P_{2.}
 - Opening snap , mid diastolic murmur, presystolic accentuation at the apex
 - Features of TR
 - Pulmonic EDM due to PR (Graham Steele) =
 - Features of heart failure

COMPLICATIONS:

- Heart Failure
- Atrial Fibrillation
- Thromboembolism
- Infective Endocarditis

- INVESTIGATIONS:
 - CXR features of LA enlargement: double shadow, filling of pulmonary bay, widened carina
 - Pulmonary congestion
 - Normal cardio-thoracic ratio (CTR)
 - MV calcification
 - ECG LAE, RVH, RAD
 - Atrial Fibrillation
 - ECHO Morphology
 - Doppler valve area, gradients, pulmonary pressures
 - Secondary changes LAE, RVH

- MANAGEMENT PHARMACOLOGICAL
 - Limited
 - Diuretics
 - HR slowing Beta blockers, digoxin
 - No role for usual HF management
 - Management of Complications

- MANAGEMENT DEFINITIVE
 - Surgical:
 - Valvotomy closed, open
 - Valve replacement/repair
 - Interventional:
 - Percutaneous balloon dilatation

- FUNCTIONAL ANATOMY:
 - Leaflets, annulus, chordea, papillary muscles

□ AETIOLOGY:

- Predominantly rheumatic locally
- Degenerative (MVP), Ischaemic, ventricular dilatation, inflammatory, endocarditis

□ PATHOLOGY:

- Fibrosis, distortion leaflets, chordae (RHD)
- Rupture chordae, papillary muscles (Ischaemia, Inf. End.)
- Annular dilatation ventricular dilatation
- Degenerative leaflets (MVP)

- PATHOPHYSIOLOGY:
 - Increased SV to accommodate regurgitant volume
 - Volume overload
 - Eccentric hypertrophy (dilatation)
 - Enlarged LA

- CLINICAL FEATURES SYMPTOMS:
 - -Insidious and slowly progressing
 - May be asymptomatic even in severe
 MR
 - Onset often depends on complications e.g. PHT, A.fib or precipitating factors.
 - Features of CCF

- CLINICAL FEATURES SIGNS:
 - Pulse maybe large volume
 - Apex, displaced, heaving
 - Muffled S1, S3+
 - Apical pan systolic murmur, radiating to axilla

- COMPLICATIONS:
 - CCF
 - Infective endocarditis
 - A.Fib

- INVESTIGATIONS:
 - CXR- cardiomegaly
 - pulmonary congestion
 - ECG- LAE, LVH, A.Fib
 - ECHO- Morphology
 - Quantification
 - LV size and function

- MANAGEMENT:
 - Pharmacological :
 - Management of CCF
 - Complications
 - Definitive;
 - Surgery valve replacement, repair

- FUNCTIONAL ANATOMY Cusps, commissures, (infra/supra valvular)
- AETIOLOGY
 - In young adults predominantly RHD, usually with MVD
 - Children congenital
 - Older adults degenerative

- PATHOPHYSIOLOGY:
 - Obstruction to LV emptying
 - Pressure overload
 - Marked concentric hypertrophy
 - Increased oxygen demand(LV mass)
 - Elevated LV diastolic pressures (LV stiffness)

- CLINICAL FEATURES SYMPTOMS:
 - Long latent period
 - Once symptoms supervene, rapid progression
 - Classical triad exertional:
 - Dyspnoea
 - Angina
 - Syncope

- CLINICAL FEATURES SIGNS:
 - Pulse small volume, slow rising
 - Apex may not be displaced but heaving
 - Ejection click
 - Ejection systolic murmur > carotids
 - **S4**

- COMPLICATIONS:
 - LV failure diastolic, systolic
 - Arrhythmias Atrial (A.fib), Ventricular (sudden death)
 - Infective endocarditis

- INVESTIGATIONS:
 - CXR- Maybe normal,
 - Normal CTR
 - Calcification
 - Post stenotic dilatation
 - ECG- Marked LVH with ST depression, T wave inversion
 - ECHO- Valve morphology
 - Doppler valve area, gradient
 - LVH, LV function

- MANAGEMENT:
- Medical very limited
 - Cautious diuresis
 - "CCF" management only in systolic dysfunction
 - Complications
- Definitive Surgical (AVR)
 - Balloon dilatation
 - Trans catheter aortic valve implantation (TAVI)

- FUNCTIONAL ANATOMY
 - Valve cusps Main mechanism in rheumatic
 - Root dilatation degenerative, inflammatory

- AETIOLOGY:
 - Congenital
 - Acquired:
 - -Rheumatic
 - -Syphilis
 - -Dissecting aneurysm
 - -Inflammatory disorders
 - -Degenerative

PATHPHYSIOLOGY:

- Hyperdynamic circulation
- Regurgitation > volume overload > eccentric LVH (dilatation)
- Much severer than MR hence very large hearts (Cor Bovis)

- CLINICAL FEATURE SYMPTOMS:
 - Long latency
 - Features of hyperdynamic state:
 - -Pounding in chest, head, palpitations
 - Features of heart failure late

- CLINICAL FEATURES SIGNS:
 - Displaced ,hyperdynamic apex
 - Hyperdynamic signs; =
 - Large volume, collapsing pulse
 - Corrigan's sign
 - Wide pulse pressure
 - Pistol shots(Traube's), Duroziez's
 - Quincke's, de Musset's signs

- SIGNS CONT:
 - Soft S1, A2
 - Early diastolic murmur LSE 3rd, 4th ICS
 - Ejection systolic murmur aortic
 - Apical mid diastolic murmur(Austin Flint)

- INVESTIGATIONS:
 - ECG LVH, marked ST segment, T wave changes
 - CXR Massive cardiomegaly
 - ECHO Morphology
 - Quantification
 - LV size, function

MANAGEMENT

- Medical Management of CCF
- Definitive Surgical (aortic valve replacement)

- Predominantly rheumatic
- Usually occurs with MS, masks presentation
- Pathophysiology:
 - RV-RA gradient : elevated RA pressure
 - > systemic venous congestion
 - Impaired RV filling > low cardiac output

- Symptoms:
 - Low output:- fatigue
 - Systemic congestion:- abd swelling and discomfort, leg swelling, fluterring in the neck
 - Absence of chest symptoms (even with MS)

- Signs:
 - Prominent "a" waves on JVP
 - Low volume pulse
 - **Negatives** No PHT, RVH and clear lung fields even with MS
 - LSE MDM- increased on inspiration

- INVESTIGATIONS:
 - CXR Marked "cardiomegaly'- RA enlargement, with clear lung fields
 - ECG RAH, ? Biatrial hypertrophy with NO RVH
 - ECHO Confirm stenosis, gradient
 - Coexistent MS

- MANAGEMENT:
 - Medical Sodium restriction, diuresis
 - Surgical Valvotomy(open/closed),
 valve replacement

- Aetiology Often "functional" due to RV annular dilatation
 - ✓ PHT with RVH
 - ✓ Other causes of RV dilatation
 - Others Rheumatic, cong, inflammatory, carcinoid syndrome
- □ Pathophysiology RV volume overload
 - Primary pathology especially PHT

CLINICAL

- Usually well tolerated in absence of PHT
- Features of RVF
 - ✓ Low output Fatigue, Cachexia
 - ✓ Systemic congestion Leg, Abdominal swelling, anorexia
- Signs
 - Elevated JVP with prominent V waves
 - ✓ Left parasternal heave (RVH)
 - ✓ Loud P2
 - ✓ LLSE pan systolic murmur
 - ✓ Pulsatile hepatomegaly
 - ✓ Ascites

- INVESTIGATIONS:
 - ECG RVH, primary pathology
 - ECHO Morphology
 - Quantification
 - Pulmonary pressures
 - RVH, primary pathology
 - CXR Cardiomegaly due to RVH

- MANAGEMENT:
 - Surgical annuloplasty, valve replacement
 - Primary condition
 - Diuresis

PULMONIC VALVE

- STENOSIS Almost always congenital
- REGURGITATION Secondary to pulmonary hypertension
 - Presentation, management is of primary disease

Questions??