Valvular Heart Disease

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Introduction

• remarkable changes in the evaluation and management of patients with valvular heart disease
• Advances in surgical approaches and interventional cardiology procedures have improve patient’s outcomes
Valvular heart disease

Mitral
Aortic
Tricuspid
Pulmonary

Stenosis
Regurgitation
Diagnosis

• Etiology and pathology
• Pathophysiology
• Clinical manifestation
• Physical Findings
• Laboratory Examination
• Differential Diagnosis
• Treatment
Key Concepts

• Echocardiography remains the gold standard for diagnosis and follow up patients with valvular heart disease.

• Stenotic valvular lesions can be monitored clinically until symptoms appear.

• Regurgitant valvular lesions require careful echocardiographic monitoring for left ventricular function and may require surgery even if no symptoms are present.
• Medical therapy aims at control of symptoms.
• Surgery is the treatment for most symptomatic lesions or for lesions causing left ventricular dysfunction even in the absence of symptoms.
MITRAL STENOSIS (MS)
ETIOLOGY AND PATHOLOGY

• Rheumatic fever
• Two-thirds are female
• 25% of all patients have pure MS
• 40% have combined MS and mitral regurgitation (MR)
• The incidence of MS is declining
• A major problem in tropical climates and developing countries
• Rheumatic fever results in four forms of fusion leading to stenosis:
  ➢ Commissural
  ➢ Cuspal
  ➢ Chordal
  ➢ Combined
• mitral valve cusps fuse at the their edges
• fusion of the chordae tendineae results in thickening and shortening of these structures
• Calcification of the valve immobilizes the leaflets and narrows the orifice
• lead to narrowing of the valve (fish-mouth)
• dilated left atrium (LA)
• Thrombus frequently arise from LA in patients with atrial fibrillation (AF)
• Other causes: congenital, malignant carcinoid, SLE, Amyloid, etc.
PATHOPHYSIOLOGY

• mitral valve orifice is 4 to 6 cm²
  ➢ mild: 1.5-2cm²
  ➢ moderate: 1-1.5cm²
  ➢ Severe (critical): <1cm²

• transvalvular pressure gradient, pulmonary venous and arterial wedge pressures elevated - exertional dyspnea
• Tachycardia augments the transvalvular gradient and LA pressure
• the CO is abnormal at rest and may fail to rise or may even decline during activity in patients with severe MS
• Pulmonary hypertension
CLINICAL MANIFESTATIONS

• As MS progresses
  ➢ No symptoms
  ➢ Dyspnea, cough
  ➢ Orthopnea
  ➢ Paroxysmal nocturnal dyspnea

• Hemoptysis
  ➢ rupture of pulmonary-bronchial venous connections
• **Systemic Embolism**
  - more frequent in patients with AF

• **Other Symptoms**
  - Hoarness (Ortner syndrome)
  - hepatomegaly, edema, ascites, hydrothorax (right-sided heart failure)
Physical Findings

• Inspection and Palpation
  - mitral facies
  - prominent jugular venous pulse
  - RV tap-left sternal border
  - diastolic thrill-apx
  - Hepatomegaly, ankle edema, ascites, and pleural effusion in patients with MS and RV failure
• Auscultation
  - S1, P2 is accentuated
  - The opening snap (OS) follows A2
  - Low-pitched, rumbling, diastolic murmur at the apex in the left lateral recumbent position
  - The Graham Steell murmur of pulmonary regurgitation (PR) results from dilatation of the pulmonary valve ring
**Laboratory Examination**

- **ECG**
  - P wave duration in lead II > 0.12 s
  - P wave axis between +45 and -30 degrees
  - Suggests LA enlargement
  - Atrial fibrillation
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<tr>
<th></th>
<th>Normal</th>
<th>Right</th>
<th>Left</th>
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<tr>
<td>II</td>
<td>RA, LA</td>
<td>RA, LA</td>
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<td>V₁</td>
<td>RA, LA</td>
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- RV overload - tall P wave
- LA enlargement - notched, broad P wave
Biphasic P wave in V1
• Echocardiography
  - most sensitive and specific noninvasive method for diagnosing MS
  - mitral orifice size, the presence and severity of accompanying MR, the extent of restriction of valve leaflets
  - cardiac chamber size, the LV function
• **X-ray (Roentgenogram)**
  - straightening of the left border of the cardiac silhouette
  - prominence of the main pulmonary arteries
  - enlarged LA
  - Kerley B lines
• Cardiac Catheterization and Angiocardiography
  - not usually necessary
  - positive noninvasive stress tests for myocardial ischemia, coronary angiography is advisable to detect patients with critical coronary obstructions in males >45 years of age, females > 55 years of age, or younger with risk factors
Differential Diagnosis

- MR
- AR (Austin Flint murmur)
- Atrial septal defect
- Left atrial myxoma
Treatment

- Penicillin prophylaxis to prevent rheumatic fever and infective endocarditis
- Restriction of sodium intake and maintenance doses of oral diuretics
- Digoxin, Beta blockers
- Warfarin for systemic and/or pulmonary embolization and AF
• Recent, mild MS with AF
• reversion to sinus rhythm pharmacologically or by electrical shock
Mitral Valvotomy

• percutaneous balloon mitral valvotomy (PBMV)
• surgical valvotomy
• for symptomatic patients with isolated MS whose orifice is < 1.0 cm2/m2 body surface area, or < 1.7 cm2 in normal-sized adults
PBMV
• Mitral valve replacement (MVR) is necessary in patients with MS and significant associated MR
MITRAL REGURGITATION
ETIOLOGY

• abnormal mitral leaflets, chordae tendineae, papillary muscles, and mitral annulus

• mitral valve prolapse (MVP), rheumatic heart disease, infective endocarditis, annular calcification, cardiomyopathy, and ischemic heart disease
• increased LV and LA volume
• elevated LA and PA pressure
• reduced forward CO
• ejection fraction (EF) rises
• with longstanding MR, LV contractility becomes reduced
SYMPTOMS

- Fatigue
- Exertional dyspnea
- Orthopnea
- Acute severe MR--- acute pulmonary edema
PHYSICAL FINDINGS

• S1 absent
• A holosystolic murmur of grade III/VI intensity is the most characteristic auscultatory finding, most prominent at the apex and radiates to the axilla
• MVP of posterior mitral leaflet, the regurgitant jet strikes the LA wall, the systolic murmur is transmitted to the base of the heart

• "sea gull" murmur--ruptured chordae
LABORATORY EXAMINATION

• EKG
  ➢ LA, LV, RV enlargement
  ➢ AF

• ECHO: 2D, color doppler
  ➢ most accurate noninvasive technique for diagnosis of MR
TREATMENT

• Medical Treatment
  - reducing sodium intake
  - use of diuretics
  - vasodilators and digitalis
  - (ACE) inhibitors
  - Intravenous nitroprusside or nitroglycerin reduce afterload for patients with acute and/or severe MR
• Surgical Treatment

- asymptomatic or limited only during strenuous exertion, LV functions are normal—no surgery
- severe MR in asymptomatic patients, or LV dysfunction is progressive, with LV EF <60%, and/or end-systolic cavity dimension >45 mm—surgery
- MVR, mitral valvuloplasty/annuloplasty
M R
MITRAL VALVE PROLAPSE (MVP)

- systolic click-murmur syndrome
- Barlow's syndrome
- floppy-valve syndrome
- billowing mitral leaflet syndrome
• excessive or redundant mitral leaflet associated with myxomatous degeneration
• The posterior leaflet is usually more affected than the anterior
• the mitral valve annulus dilated
• rupture or redundant chordae tendineae
• cause mitral regurgitation
CLINICAL FEATURES

• more common in females, 14-30 years of age
• a broad spectrum of severities
  - mild prolapse, only a systolic click and murmur
  - chordal rupture, severe MR
• arrhythmias, syncope, chest pain, infective endocarditis, TIA, sudden death
Auscultation

- the mid- or late systolic click after the S1 generated by the sudden tensing of elongated chordae or by the prolapsing mitral leaflet when it reaches its maximum excursion.
- may be followed by a high-pitched, late systolic murmur at the apex
LABORATORY EXAMINATION

• **EKG**
  - biphasic or inverted T waves in leads II, III, and aVF, and premature contractions

• **ECHO**
  - systolic displacement (in the parasternal view) of the mitral valve leaflets by at least 2 mm into the LA superior to the plane of the mitral annulus
TREATMENT

• prevention of infective endocarditis
• Beta blockers, antiarrhythmic agents
• Aspirin for TIA patients
• For severe MR, mitral valve repair/replacement
AORTIC STENOSIS
• one-fourth of all patients with chronic VHD
• 80% of adult patients with symptomatic valvular AS are male
ETIOLOGY

• degenerative calcification of the aortic cusps

• congenital or rheumatic inflammation
PATHOPHYSIOLOGY

• The obstruction to LV outflow produces a systolic pressure gradient between the LV and aorta

• A peak systolic pressure gradient >50 mmHg, or an effective aortic orifice < 1.0 cm² or <0.6 cm²/m² body surface area, severe obstruction
• elevated LV end-diastolic pressure
• hypertrophied LV wall
• diminished compliance of LV wall
• LA, PA, and RV pressures rise
• myocardial ischemia
SYMPTOMS

• exertional dyspnea
• angina pectoris
• syncope
• LV failure in the advanced stages of the disease
Auscultation

- an early systolic ejection sound
- an ejection (mid) systolic murmur, low-pitched, rough and rasping in character, and loudest at the base of the heart, in the second right intercostal space, transmitted upward along the carotid arteries, grade III/VI
LABORATORY EXAMINATION

- EKG
  - might be normal
  - LV hypertrophy
  - ST-segment depression and T-wave inversion
• Echocardiography
  ➢ LV hypertrophy
  ➢ valvular calcification
  ➢ transaortic valvular gradient
  ➢ MS, AR

• Catheterization
  ➢ CAD suspected
• NATURAL HISTORY
  - angina pectoris, 3 years;
  - syncope, 3 years;
  - dyspnea, 2 years;
  - congestive heart failure, 1.5 to 2 years
Medical Treatment

- strenuous physical activity should be avoided in patients with severe AS (<0.5 cm²/m²)
- avoid volume depletion
- statins may be helpful to slow progression
• Surgical Treatment
  ➢ severe AS (valve area <1.0 cm² or 0.6 cm²/m² body surface area) who are symptomatic
  ➢ LV dysfunction
  ➢ expanding poststenotic aortic root, even asymptomatic.

• Percutaneous Balloon Aortic Valvuloplasty
  ➢ in children and young adults with congenital, noncalcific AS
ETIOLOGY

• Primary Valve Disease
  - three-fourths are males
  - two-thirds is rheumatic in origin
  - degenerative calcification, congenital or rheumatic
  - thickening, deformity, and shortening, prolapse of the aortic valve cusps

• Primary Aortic Root Disease
  - aortic root/annulus dilatation
  - Marfan syndrome, hypertension, dissection, syphilis, spondylitis
PATHOPHYSIOLOGY

• LVEDV↑↑
• dilatation and eccentric hypertrophy of the LV
• elevation of the LA, PA wedge, PA, and RV pressures
• reduced LVEF
• myocardial ischemia
SYMPTOMS

• Acute, severe AR--IE, trauma
  ➢ pulmonary edema, cardiogenic shock may develop rapidly
• Chronic, severe AR--a long latent period
  ➢ palpitation, exertional dyspnea, angina
PHYSICAL FINDINGS

- Arterial Pulse
  - "water-hammer" (Corrigan's pulse)
  - capillary pulsations (Quincke's pulse)
  - "pistol-shot" ("pistol-shot")
  - to-and-fro murmur (Durozize's sign)
  - arterial pulse pressure is widened
• Palpation
  - LV impulse is heaving and displaced laterally and inferiorly
  - A diastolic thrill is often palpable along the left sternal border
• Auscultation

- A2 is usually absent
- A high-pitched, blowing, decrescendo diastolic murmur, heard best in the third intercostal space along the left sternal border
- with the patient sitting up, leaning forward, and with the breath held
• Austin Flint murmur
  - soft, low-pitched, rumbling middiastolic
  - diastolic displacement of the anterior leaflet of the mitral valve by the AR stream
  - Not hemodynamically significant mitral obstruction
LABORATORY EXAMINATION

- EKG
  - LV hypertrophy
  - ST-segment depression and T-wave inversion leads I, aVL, V5, and V6 ("LV strain")
• Echocardiogram
  - wall motion are normal or even supernormal
  - rapid, high-frequency fluttering of the anterior mitral leaflet
  - thickening and failure of coaptation of the leaflets
  - dilatation of the aortic annulus
  - Color flow Doppler echocardiographic imaging is very sensitive in the detection of AR
• **X-RAY**

  - LV enlargement, the apex is displaced downward and to the left
  - the ascending aorta and aortic knob may be dilated
TREATMENT

• Medical Treatment

- digitalis, salt restriction, diuretics, and vasodilators, especially ACE inhibitors
- Cardiac arrhythmias and infections must be treated promptly and vigorously.
• Surgical Treatment

- operation should be carried out in asymptomatic patients, when a left ventricular ejection fraction (LVEF) <55% or a LV end-systolic volume >55 mL/m2.

- AVR, AV repair, narrowing the annulus
TRICUSPID STENOSIS

- uncommon
- generally rheumatic in origin
- more common in females than in males
- It does not occur as an isolated lesion and is usually associated with MS, TR
A diastolic pressure gradient between the RA and RV is elevated.

A mean diastolic pressure gradient of 4 mmHg is sufficient to result in systemic venous congestion.

Ascites and edema.
SYMPTOMS

• TS can mask the hemodynamic and clinical features of the MS
• Amelioration of MS symptoms should raise the possibility that TS may be developing
• Dyspnea, hepatomegaly, ascites, and edema
PHYSICAL FINDINGS

• marked hepatic congestion, distended jugular veins, jaundice, splenomegaly

• the tricuspid murmur is generally heard best along the left lower sternal margin, augmented during inspiration
LABORATORY EXAMINATION

• EKG
  ➢ RA enlargement include tall, peaked P waves in lead II, upright P waves in lead V1.

• ECHO
  ➢ thickened tricuspid valve; elevated transvalvular gradient
TREATMENT

• intensive salt restriction and diuretic therapy are required during the preoperative period

• Repair, or replacement
TRICUSPID REGURGITATION (TR)

• Most commonly, TR is functional and secondary to marked dilatation of the tricuspid annulus.

• It is commonly seen in the late stages of heart failure with severe pulmonary hypertension, as well as in ischemic heart disease, cardiomyopathy, and cor pulmonale.
• Infarction of RV papillary muscles, tricuspid valve prolapse, carcinoid heart disease, endomyocardial fibrosis, infective endocarditis, and trauma
• Congenital heart disease: defects of the atrioventricular canal, Ebstein's malformation
• Symptoms of right heart failure
• A blowing holosystolic murmur along the lower left sternal margin, which may be intensified during inspiration
• EKG: RV enlargement, AF
• ECHO: RV dilatation, prolapsing of tricuspid leaflets, severity of TR
TREATMENT

• Isolated TR, in the absence of pulmonary hypertension, may not require operation.
• Treatment of the primary disease, MS
• Tricuspid annuloplasty, tricuspid valve repair/replacement
PULMONIC VALVE DISEASE

- The most common acquired abnormality is regurgitation secondary to dilatation of the pulmonic valve ring as a consequence of severe pulmonary hypertension.
- Graham Steell murmur, a high-pitched, decrescendo, diastolic blowing murmur along the left sternal border
- It is usually of little hemodynamic significance
SUMMARY

• Etiology
  ➢ rheumatic
  ➢ myxomatous degeneration
  ➢ aging
  ➢ congenital abnormality
  ➢ trauma
  ➢ infective endocarditis
  ➢ infiltrative diseases
  ➢ dilation of valve annulus
PATHOPHYSIOLOGY

• Preload increases
  ➢ MR, AR
• Afterload increases
  ➢ AS, PS
• Enlargement of cardiac chamber size
• Heart failure
• Arrhythmia
SYMPTOMS

• None
• Nonspecific
• Left heart failure
• Right heart failure
• Arrhythmia
PHYSICAL FINDINGS

- Cardiac size
- S1, S2
- Murmur, characteristic
- Heart failure
LABORATORY EXAMINATION

- X-ray
- EKG
- **Echo**: gold standard for diagnosis
- Cath
TREATMENT

• Medical Treatment
  ➢ Sodium restriction
  ➢ diuretics and digitalis
  ➢ Vasodilators
  ➢ ACE inhibitor, β-blockers
  ➢ anticoagulants
  ➢ Endocarditis prophylaxis
  ➢ Careful follow-up
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<tr>
<th>Lesion</th>
<th>Symptom Control</th>
<th>Secondary Prevention and Natural History</th>
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<tr>
<td>Mitral stenosis</td>
<td>Diuretics for heart failure; Digoxin, β blockers, and rate-limiting calcium antagonists for rate control in atrial fibrillation</td>
<td>Penicillin prophylaxis against recurrent episodes of rheumatic fever; Anticoagulants to prevent systemic thromboembolism</td>
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<tr>
<td>Mitral regurgitation</td>
<td>Diuretics and vasodilators (usually ACE inhibitors) for heart failure</td>
<td>No proven treatment</td>
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<tr>
<td>Aortic stenosis</td>
<td>Diuretics for heart failure; nitrates and β blockers for angina</td>
<td>No proven treatment but lipid lowering therapy may slow progression of calcific aortic stenosis</td>
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<tr>
<td>Aortic regurgitation</td>
<td>Diuretics and vasodilators (usually ACE inhibitors) for heart failure</td>
<td>Vasodilators (nifedipine or ACE inhibitors) to protect the left ventricular myocardium and delay the need for surgery</td>
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*Source: NA Boon, P Bloomfield: The medical management of valvular heart disease, Heart 87: 395, 2002, with permission*
• Surgical treatment:
  - Interventional therapy
  - Surgical operation
    - Valve repair
    - Valve replacement