CARDIOLOGY Short notes

Essential Guide for Doctors & Medical Students



1ST EDITION

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educate yourself to empower yourself

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Ischemic Heart Disease

- Unstable Angina
- NSTEMI
- STEMI
- Chronic Stable Angina



Myocardial Infarction



Angioplasty



Coronary Artery Bypass Grafting





Unstable Angina

Definition

A type of acute coronary syndrome (ACS) where there is new or worsening chest pain due to inadequate blood supply to the heart muscle (myocardial ischemia) without myocardial infarction.

Risk Factors

- Age
- Hypertension
- Hyperlipidemia
- Smoking
- Diabetes mellitus
- Family history of coronary artery disease

Clinical Presentation

- New onset chest pain or discomfort
- Pain at rest or minimal exertion
- Increasing frequency, duration, or intensity of angina
- Pain may radiate to the arm, neck, jaw, or back

Etiology



Thrombosis



- Vasospasm
- Atherosclerotic plaque rupture
- Coronary artery dissection

Diagnosis

- 1. History and Physical Examination:
 - Detailed symptom evaluation
 - Risk factor assessment
- 2. Electrocardiogram (ECG):
 - ST-segment depression or Twave inversion
 - No persistent ST-segment elevation
- 3. Biomarkers:
 - Cardiac troponins (typically negative in UA)
 - ∘ CK-MB
- 4. Imaging:
 - o Coronary angiography
 - o Echocardiography
- 5. Stress Testing:
 - Exercise or pharmacologic stress test (poststabilization)



Management

Medical Therapy

Antiplatelet Agents

- Aspirin
- P2Y12 inhibitors (clopidogrel, ticagrelor)

Anticoagulants:

Unfractionated heparin or low molecular weight heparin

Anti-Ischemic Therapy

- Nitrates
- Beta-blockers
- Calcium channel blockers (if beta-blockers contraindicated)

Statins

High-intensity statin therapy

Invasive Management:

Percutaneous Coronary Intervention (PCI):

 Indicated for high-risk patients or those with refractory symptoms

Coronary Artery Bypass Grafting (CABG):

 Considered in patients with significant multi-vessel disease



Complications

- Progression to myocardial infarction
- Arrhythmias
- Heart failure
- Cardiogenic shock

Prognosis:

- Variable, depending on the extent of coronary artery disease and response to treatment
- Requires long-term management of risk factors and adherence to medical therapy

Follow-Up:

- Regular follow-up with cardiologist
- Lifestyle modification: smoking cessation, diet, exercise
- Monitoring and managing comorbidities (e.g., hypertension, diabetes)

- Unstable angina is a medical emergency requiring prompt evaluation and treatment.
- Differentiation from myocardial infarction is crucial for appropriate management.
- Early intervention can prevent progression to myocardial infarction and improve outcomes.



Non-ST-segment Elevation Myocardial Infarction (NSTEMI)

Definition

A type of myocardial infarction (heart attack) characterized by the absence of ST-segment elevation on an electrocardiogram (ECG).

Pathophysiology

- Caused by partial occlusion of a coronary artery leading to ischemia.
- Results in subendocardial infarction (partial thickness of the heart muscle).
- Plaque rupture with superimposed thrombus formation is a common cause.

Clinical Presentation

- Chest pain: Often described as pressure, squeezing, or tightness, usually lasting more than 20 minutes.
- Pain may radiate to the arm, neck, or jaw.
- Associated symptoms: Dyspnea, diaphoresis, nausea, and fatigue.

Diagnosis

- Electrocardiogram (ECG):
 - ST-segment depression or transient ST-segment elevation.
 - T-wave inversions or flattening may be seen.

Cardiac Biomarkers:

- Elevated levels of troponins (TnI or TnT).
- May also see raised CK-MB.

• Risk Stratification Scores:

- TIMI (Thrombolysis in Myocardial Infarction) score.
- GRACE (Global Registry of Acute Coronary Events) score.





Management

Initial Management

- Antiplatelet therapy: Aspirin and a P2Y12 inhibitor (e.g., clopidogrel, ticagrelor).
- Anticoagulation: Unfractionated heparin or low-molecular-weight heparin.
- Anti-ischemic therapy: Betablockers, nitrates, and statins.

Revascularization

- Considered based on risk stratification.
- Percutaneous coronary intervention (PCI) is preferred for high-risk patients.
- Coronary artery bypass grafting (CABG) - in certain cases.

Adjunctive Therapy

- ACE inhibitors or ARBs for patients with heart failure, hypertension, or diabetes.
- Aldosterone antagonists for patients with heart failure or diabetes and an ejection fraction < 40%.

Complications

- Arrhythmias (e.g., atrial fibrillation, ventricular tachycardia).
- Heart failure.
- Recurrent ischemia or reinfarction.
- Mechanical complications (e.g., mitral regurgitation, ventricular septal defect).

Prognosis

- Dependent on early recognition and treatment.
- Use of risk stratification tools helps guide therapy and predict outcomes.
- Long-term management focuses on lifestyle modification and secondary prevention (e.g., smoking cessation, control of diabetes, hypertension, hyperlipidemia).

Follow-Up and Secondary Prevention

- Lifestyle changes: Smoking cessation, diet modification, regular exercise.
- Medications: Continued use of antiplatelets, statins, betablockers, and ACE inhibitors/ARBs.
- Monitoring for recurrence of symptoms or new complications.

- NSTEMI is a serious but treatable condition.
- Prompt diagnosis and risk stratification are crucial.
- Management includes medical therapy and possibly revascularization.
- Secondary prevention is vital to improve long-term outcomes.

ST-Segment Elevation Myocardial Infarction (STEMI)

Definition

A type of myocardial infarction (MI) characterized by the elevation of the ST segment on an electrocardiogram (ECG), indicating a full-thickness infarction of the myocardium.

Pathophysiology

- Occurs due to a complete and sustained occlusion of a coronary artery, often due to rupture of an atherosclerotic plaque and subsequent thrombus formation.
- Leads to ischemia and necrosis of the myocardium supplied by the affected artery.

Clinical Presentation



- Chest Pain: Severe, acute, and persistent, often described as crushing or squeezing, radiating to the left arm, neck, or jaw.
- Associated Symptoms
 Dyspnea, diaphoresis, nausea, vomiting, and syncope.

• Atypical Presentation Especially in women, elderly, and diabetic patients.



Diagnosis

ECG Findings

- ST-segment elevation in two or more contiguous leads.
- New or presumed new left bundle branch block (LBBB).
- Reciprocal ST-segment depression in opposite leads.

Cardiac Biomarkers

- Elevated troponins (Troponin I or T) confirm myocardial injury.
- CK-MB can also be used but is less specific than troponins.

Management:

Immediate Management

- Aspirin: 162-325 mg chewed.
- P2Y12 Inhibitors: Clopidogrel, prasugrel, or ticagrelor.
- Anticoagulants: Unfractionated heparin or low-molecular-weight heparin.
- Nitroglycerin: For pain relief unless contraindicated.
- Morphine: For pain not relieved by nitroglycerin.

Reperfusion Therapy

- Primary Percutaneous Coronary
 Intervention (PCI): Preferred within
 90 minutes of first medical contact.
- Fibrinolysis: Indicated if PCI is not available within 120 minutes; best within 30 minutes of hospital arrival.

Long-term Management

- Dual Antiplatelet Therapy (DAPT): Aspirin plus a P2Y12 inhibitor for at least 12 months.
- Beta-blockers: Reduce myocardial oxygen demand.
- ACE Inhibitors/ARBs: Especially in patients with left ventricular dysfunction or heart failure.
- Statins: High-intensity statin therapy regardless of initial cholesterol levels.
- Lifestyle Modifications: Smoking cessation, dietary changes, regular physical activity.

Complications

Early Complications

- Arrhythmias (ventricular fibrillation/tachycardia, atrial fibrillation).
- Cardiogenic shock.
- Heart failure.
- Mechanical complications
 (ventricular septal rupture, papillary muscle rupture, free wall rupture).

Late Complications

- Heart failure.
- Recurrent MI.
- Dressler's syndrome (post-MI pericarditis).



Prognosis

- Early reperfusion significantly improves outcomes.
- Mortality is highest within the first 30 days but decreases with timely and effective treatment.

Follow-up

- Regular follow-up visits to monitor for recurrent ischemia, heart failure, and adherence to secondary prevention strategies.
- Cardiac rehabilitation programs are beneficial for recovery and reducing recurrence.



Chronic Stable Angina

Definition

Chronic stable angina is characterized by chest pain or discomfort due to myocardial ischemia, typically triggered by physical exertion or emotional stress and relieved by rest or nitroglycerin.

Pathophysiology

- Imbalance between myocardial oxygen supply and demand.
- Commonly caused by atherosclerosis of the coronary arteries.
- Reduced blood flow leads to ischemia and angina symptoms.

Clinical Features

- Retrosternal chest pain or discomfort, often described as pressure, tightness, or heaviness.
- Pain may radiate to the arms, neck, jaw, shoulder, or back.

- Predictable and reproducible with certain levels of exertion or stress.
- Relieved by rest or sublingual nitroglycerin within minutes.

Diagnosis

- History and physical examination.
- Electrocardiogram (ECG): May show ischemic changes during an episode.
- Stress testing: Exercise ECG, Stress echocardiography, or Nuclear perfusion imaging.
- Coronary angiography: Gold standard for assessing coronary artery disease.





Management

Lifestyle Modifications

- Smoking cessation.
- Regular physical activity.
- Healthy diet (low in saturated fats, high in fruits and vegetables).
- Weight management.
- Stress reduction.

Pharmacological Therapy

- Anti-anginal medications
 - Nitrates: Short-acting (nitroglycerin) for acute relief; long-acting (isosorbide mononitrate) for prevention.
 - Beta-blockers: Reduce heart rate and myocardial oxygen demand.
 - Calcium channel blockers: Used if beta-blockers are contraindicated or not tolerated.
 - **Ranolazine**: Used for refractory angina.
- Antiplatelet therapy
 - Aspirin: Reduces the risk of myocardial infarction.
 - **Clopidogrel**: For those intolerant to aspirin or at high risk.
- Lipid-lowering agents
 - **Statins**: To achieve target LDL cholesterol levels.
- ACE inhibitors/ARBs: In patients with hypertension, diabetes, or left ventricular dysfunction.

Revascularization

- Percutaneous Coronary Intervention
 (PCI): Indicated if symptoms are not controlled by medical therapy.
- Coronary Artery Bypass Grafting (CABG): For patients with left main disease, triple vessel disease, or failed PCI.

Prognosis



- Generally good with appropriate treatment and lifestyle modifications.
- Regular follow-up to monitor symptom progression and treatment efficacy.



Monitoring

- Regular follow-up appointments.
- Periodic stress testing to assess the effectiveness of treatment.
- Monitoring and management of risk factors such as hypertension, diabetes, and hyperlipidemia.

- Early recognition and treatment are crucial to prevent complications.
- A comprehensive approach including lifestyle changes, medication, and possibly revascularization is essential.
- Education on recognizing symptoms and when to seek medical help is important for patients.

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Valvular Heart Disease

- Aortic Stenosis (AS)
- Aortic Regurgitation
- (AR) Mitral Stenosis
- (MS) Mitral Regurgitation (MR)
- Tricuspid Stenosis (TS)
- Tricuspid Regurgitation (TR)
- Pulmonary Stenosis (PS)
- Pulmonary Regurgitation (PR)

Aortic Stenosis (AS)

Definition

Aortic stenosis is the narrowing of the aortic valve opening, leading to obstructed blood flow from the left ventricle to the aorta.

Etiology

Congenital:

- Bicuspid aortic valve.
- Acquired:
 - Degenerative calcific aortic stenosis (common in elderly).
 - Rheumatic heart disease.

Pathophysiology

- Increased afterload causes left ventricular hypertrophy (LVH).
- Reduced cardiac output, especially during exertion.
- Elevated left ventricular pressure.

Clinical Features

Symptoms

- Angina.
- Syncope, especially on exertion.
- Heart failure (dyspnea, orthopnea, PND).

<u>Signs</u>

- Ejection systolic murmur (crescendo-decrescendo) heard best at the right upper sternal border, radiating to the carotids.
- Slow-rising and weak pulse (pulsus parvus et tardus).
- Left ventricular heave or thrill.

Investigations

1. Echocardiography:

- Key diagnostic tool.
- Assesses valve area, gradient, and LV function.

2. ECG:

 LVH, left atrial enlargement.

3. Chest X-ray:

 May show post-stenotic aortic dilatation, calcified aortic valve, and LVH.

4. Cardiac catheterization:

 For definitive assessment, especially pre-surgery.

Surgical

- Aortic valve replacement
 (AVR) for symptomatic
 severe AS or asymptomatic
 with LV dysfunction.
- Transcatheter aortic valve implantation (TAVI) for patients at high surgical risk.

Prognosis

- Poor without intervention once symptomatic.
- Regular follow-up essential for asymptomatic patients with moderate or severe AS.

Key Points

- Early identification and timely intervention are crucial.
- Regular monitoring with echocardiography for progression.
- Choice of intervention (surgical vs. TAVI) depends on patient's surgical risk and comorbidities.

Management

<u>Medical</u>

- Symptomatic relief (e.g., diuretics for heart failure).
- Risk factor management (e.g., hypertension, hyperlipidemia).

Aortic Regurgitation (AR)

Definition

• A condition where the aortic valve fails to close properly, causing blood to flow back into the left ventricle from the aorta during diastole.

Etiology

Acute Causes

- o Infective endocarditis
- Aortic dissection
- o Trauma

Chronic Causes

- o Rheumatic heart disease
- o Congenital bicuspid aortic valve
- Aortic root dilation (e.g., Marfan syndrome, syphilis)
- Degenerative valve disease

Pathophysiology

- Increased left ventricular volume load leads to:
- Left ventricular dilation and hypertrophy
- Increased stroke volume
- Elevated systolic blood pressure
- Decreased diastolic blood pressure
- Progressive left ventricular dysfunction

Clinical Features

Symptoms

- Dyspnea (exertional, orthopnea, paroxysmal nocturnal dyspnea)
- Fatigue
- Palpitations
- Angina (less common)
- Heart failure symptoms in advanced stages

<u>Signs</u>

- Wide pulse pressure (high systolic, low diastolic)
- Bounding "water hammer" pulse
- De Musset's sign (head bobbing with each heartbeat)
- Corrigan's pulse (rapidly rising and collapsing pulse)
- Duroziez's sign (systolic and diastolic murmurs heard over the femoral artery)
- Traube's sign (pistol-shot sounds over femoral arteries)

Auscultation

- Diastolic decrescendo murmur, best heard at the left sternal border
- Austin Flint murmur: low-pitched, rumbling diastolic murmur at the apex due to mitral valve functional stenosis

Diagnostic Investigations

Echocardiography

- Quantifies severity of regurgitation
- Assesses left ventricular size and function

Cardiac MRI

- Detailed assessment of aortic root and valve anatomy
- Chest X-ray
 - Cardiomegaly
 - Aortic root dilation
- ECG
 - Left ventricular hypertrophy

Management

<u>Medical</u>

- Vasodilators (e.g., ACE inhibitors, nifedipine) to reduce afterload
- Diuretics for symptom control in heart failure
- Beta-blockers (use cautiously, more common in Marfan syndrome)

Surgical

Aortic valve replacement

 (indicated in symptomatic
 patients or those with significant
 left ventricular
 dysfunction/dilation)

 Valve repair or aortic root surgery in selected cases

Prognosis

- Dependent on the etiology and severity
- Chronic AR has a better prognosis with timely surgical intervention
- Untreated severe AR can lead to left ventricular failure and increased mortality

Follow-Up

- Regular monitoring with
 echocardiography
- Surveillance for symptoms and left ventricular function

- Early recognition and appropriate intervention are critical.
- Symptomatic patients and those with evidence of left ventricular dysfunction should be referred for surgical evaluation.
- Medical management can help alleviate symptoms and delay progression in selected patients.

Mitral Stenosis

Definition

 Mitral stenosis (MS) is the narrowing of the mitral valve opening, impeding blood flow from the left atrium to the left ventricle.

Etiology

- Rheumatic fever: Most common cause.
- Congenital mitral stenosis.
- Other causes: Calcification in elderly patients, infective endocarditis, and certain connective tissue diseases.

Pathophysiology

- Narrowed mitral valve leads to increased left atrial pressure.
- Results in pulmonary congestion and hypertension.
- Over time, right ventricular hypertrophy and failure may occur.
- Reduced cardiac output due to limited left ventricular filling.

Clinical Features

Symptoms

- Dyspnea on exertion.
- Orthopnea and paroxysmal nocturnal dyspnea.
- Palpitations (often due to atrial fibrillation).
- Hemoptysis (due to pulmonary congestion and hypertension).
- Fatigue and reduced exercise tolerance.

<u>Signs</u>

- **Mitral facies**: Flushed cheeks with a bluish tinge.
- Jugular venous distention.
- Peripheral edema.
- Auscultation:
 - Diastolic murmur at the apex with an opening snap.
 - Loud S1 and possible S2 splitting.
 - Mid-diastolic rumble best
 heard with the bell of the
 stethoscope in left lateral
 decubitus position.

Diagnosis

- Echocardiography: Gold standard.
- Measures mitral valve area, pressure gradients, and assesses severity.
- ECG:
- May show left atrial enlargement, atrial fibrillation.
- Chest X-ray:
- Left atrial enlargement, pulmonary congestion.
- Cardiac catheterization:
- Measures pressure gradients across the mitral valve if non-invasive tests are inconclusive.

Severity Classification (by mitral valve area)

- Mild: >1.5 cm².
- Moderate: 1.0–1.5 cm².
- Severe: <1.0 cm².

Management

<u>Medical</u>

- Diuretics for symptom relief.
- Beta-blockers or calcium channel blockers to control heart rate.
- Anticoagulation if atrial fibrillation is present (risk of thromboembolism).

Interventional

 Percutaneous balloon mitral valvotomy (PBMV): Preferred for suitable candidates.

<u>Surgical</u>

 Mitral valve repair or replacement if PBMV is not feasible or unsuccessful.

Complications

- Atrial fibrillation and thromboembolism.
- Pulmonary hypertension.
- Right-sided heart failure.
- Infective endocarditis.

Follow-Up

- Regular monitoring with echocardiography.
- Manage risk factors and comorbid conditions.
- Monitor for and manage atrial fibrillation and heart failure symptoms.

Prognosis

- Depends on severity, timely intervention, and management of complications.
- Excellent outcomes with appropriate treatment and follow-up.

Mitral Regurgitation (MR)

Definition

Mitral Regurgitation is a condition where the mitral valve fails to close properly during systole, causing blood to flow backward into the left atrium.

- Left ventricular volume overload
- Reduced cardiac output
- Pulmonary hypertension in chronic cases

Etiology

Primary (Organic) MR

- Degenerative: Mitral valve prolapse, myxomatous degeneration
- o Rheumatic heart disease
- o Infective endocarditis

Secondary (Functional) MR

- Left ventricular dysfunction:
 Ischemic heart disease,
 dilated cardiomyopathy
- Left atrial enlargement: Atrial fibrillation

Pathophysiology

 Increased left atrial pressure and volume

Clinical Features

Symptoms

- Asymptomatic in early stages
- Dyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnea
- o Fatigue and weakness
- Palpitations (due to atrial fibrillation)

<u>Signs</u>

- Holosystolic (pansystolic) murmur at the apex, radiating to the axilla
- S3 gallop (sign of left ventricular dysfunction)
- Displaced apex beat

Diagnosis

- **Physical Examination:** Auscultation of murmur
- Echocardiography: Confirmatory test; assesses severity, valve morphology, and left ventricular function
- Electrocardiogram (ECG): May show left atrial enlargement, atrial fibrillation
- Chest X-ray: Cardiomegaly, pulmonary congestion
- Cardiac MRI: Detailed anatomy and function assessment
- Cardiac Catheterization: Hemodynamic assessment, especially if coronary artery disease is suspected

- Mitral Valve Replacement: For severe MR not amenable to repair
- Percutaneous Mitral Valve Repair (e.g., MitraClip): For high-risk surgical patients

Prognosis

- Dependent on etiology, severity, and timely management
- Untreated severe MR can lead to heart failure and reduced survival

Follow-Up

- Regular monitoring with echocardiography
- Clinical evaluation for symptoms and signs of heart failure

Management

Medical Therapy:

- Diuretics for symptom relief (pulmonary congestion)
- ACE inhibitors or ARBs for afterload reduction
- Beta-blockers and anticoagulation for atrial fibrillation

Surgical/Interventional Treatment:

• **Mitral Valve Repair:** Preferred over replacement when feasible

- Early diagnosis and intervention are crucial.
- Surgical repair is often preferable to valve replacement.
- Regular follow-up is necessary to monitor disease progression and manage complications.

Tricuspid Stenosis (TS)

Definition

Narrowing of the tricuspid valve orifice, impeding blood flow from the right atrium to the right ventricle.

Etiology

- Rheumatic heart disease: Most common cause, often associated with mitral valve disease.
- **Congenital causes**: Rare, includes Ebstein anomaly.
- **Carcinoid syndrome**: Can lead to TS due to plaque-like deposits.
- Infective endocarditis: Can cause valve damage and stenosis.
- Other rare causes: Systemic lupus erythematosus, Whipple's disease, tumors.

Pathophysiology

- Obstruction of blood flow from the right atrium to the right ventricle.
- Increased right atrial pressure and hypertrophy.
- Reduced right ventricular filling.
- Systemic venous congestion.

Clinical Features

Symptoms

- Fatigue and weakness due to low cardiac output.
- Abdominal discomfort and hepatomegaly from venous congestion.
- Peripheral edema and ascites.
- Jugular venous distension with a prominent "a" wave.

<u>Signs</u>

- Diastolic murmur best heard at the lower left sternal border, increasing with inspiration.
- Hepatomegaly and pulsatile liver.
- Peripheral edema and ascites.
- Cyanosis in severe cases due to reduced cardiac output.

Diagnosis

- Echocardiography
 - Diagnostic tool of choice.
 - Shows thickened tricuspid valve, restricted motion, and increased gradient across the valve.
- Doppler studies
 - Assess the severity of stenosis.
- ECG
 - May show right atrial enlargement.
- Chest X-ray
 - Right atrial enlargement and possible signs of pulmonary congestion.
- Cardiac catheterization
 - Confirms diagnosis and assesses severity.

Management

Medical

- Diuretics to manage fluid overload.
- Sodium restriction.
- Treat underlying causes (e.g., rheumatic disease, infective endocarditis).

Surgical

- Percutaneous Balloon Valvotomy: Considered in select cases.
- Valve Replacement: Indicated in severe cases, particularly when associated with other valve diseases

<u>Follow-Up</u>

- Regular monitoring with echocardiography.
- Manage comorbid conditions and complications.

Prognosis

- Depends on the underlying cause and presence of associated valvular disease.
- Rheumatic TS often coexists with other valvular diseases, influencing overall prognosis.
- Early intervention and management of underlying conditions improve outcomes.

- Rare condition, often secondary to rheumatic heart disease.
- Presents with systemic venous congestion and low cardiac output symptoms.
- Echocardiography is crucial for diagnosis and assessment.
- Management includes both medical therapy for symptom relief and surgical intervention for severe cases.

Tricuspid Regurgitation (TR)

Definition

Tricuspid regurgitation is the backflow of blood from the right ventricle into the right atrium during systole due to improper closure of the tricuspid valve.

Pathophysiology

- Increased right atrial pressure
- Right atrial and ventricular enlargement
- Systemic venous congestion
- Decreased cardiac output

Etiology

Primary TR

- Congenital abnormalities (Ebstein anomaly)
- Infective endocarditis
- 。 Rheumatic heart disease
- Carcinoid syndrome

Secondary TR

- Right ventricular dilatation due to left heart failure
- Pulmonary hypertension
- Right ventricular infarction
- Atrial fibrillation

Clinical Features

Symptoms

- Fatigue
- 。 Weakness
- Peripheral edema
- Ascites
- Hepatomegaly

<u>Signs</u>

- Jugular venous distention
- $_{\circ}~$ Pulsatile liver
- Holosystolic murmur at the left lower sternal border
- Hepatojugular reflux
- 。Cyanosis (in severe cases)

Diagnostic Investigations

Echocardiography

- Primary diagnostic tool
- Assess valve anatomy, regurgitation severity, and right ventricular function

Electrocardiography (ECG)

 May show right atrial enlargement and right ventricular hypertrophy

Chest X-ray

- Cardiomegaly
- Right atrial enlargement
- Pleural effusion

Cardiac MRI/CT

For detailed anatomical assessment

Right Heart Catheterization

 Measure pressures in the right atrium and ventricle

Management

<u>Medical</u>

- Diuretics for symptom relief (reduce congestion)
- Management of underlying causes (e.g., pulmonary hypertension)

Surgical

- Annuloplasty (repair)
- Tricuspid valve replacement (in severe cases)
- Indications include severe symptomatic TR and TR with concomitant left-sided valve surgery

Prognosis

- Depends on the underlying cause and severity
- Early intervention can improve outcomes
- Severe untreated TR can lead to right heart failure and reduced survival

Follow-up

- Regular echocardiographic
 monitoring for progression
- Manage contributing conditions (e.g., control of heart failure and pulmonary hypertension)
- Monitor for complications such as arrhythmias and worsening heart failure

- TR is often secondary to left heart disease or pulmonary hypertension.
- Clinical suspicion arises from signs of right heart failure and jugular venous distention.
- Echocardiography is the cornerstone of diagnosis.
- Management ranges from medical therapy for mild cases to surgical intervention for severe cases.

Pulmonary Stenosis (PS)

Definition

Pulmonary Stenosis (PS) is the narrowing of the pulmonary valve or the infundibulum (right ventricular outflow tract), leading to obstruction of blood flow from the right ventricle to the pulmonary artery.

Etiology

- **Congenital**: Most common cause, often associated with other congenital heart defects (e.g., Tetralogy of Fallot).
- Acquired: Rare, can result from rheumatic heart disease, carcinoid syndrome, or infective endocarditis.

Pathophysiology

- Increased Right Ventricular Pressure: The right ventricle must work harder to pump blood through the narrowed valve.
- **Right Ventricular Hypertrophy** Chronic pressure overload leads to thickening of the right

ventricular muscle.

• Post-stenotic Pulmonary Artery Dilatation: Turbulent flow causes the pulmonary artery to dilate just beyond the stenosis.

Clinical Features

Symptoms

May be asymptomatic; when present, they include dyspnea on exertion, fatigue, chest pain, and syncope.

<u>Signs</u>

- Systolic ejection murmur at the left upper sternal border
- Ejection click that decreases with inspiration
- Right ventricular heave
- Wide split of the second heart sound (S2) if severe

Diagnosis

- Electrocardiogram (ECG): Right ventricular hypertrophy and right axis deviation.
- **Chest X-ray**: May show poststenotic dilatation of the pulmonary artery and right ventricular enlargement.
- Echocardiography: Main diagnostic tool; assesses valve morphology, gradient across the valve, and right ventricular function.
- Cardiac Catheterization: Measures pressure gradient across the pulmonary valve and evaluates for other congenital anomalies.

 Valve Replacement: Rarely needed but may be required in severe cases or if other interventions fail.

Prognosis

- Generally good with appropriate treatment.
- Lifelong follow-up is necessary to monitor for complications and recurrence.

Complications

- Right heart failure
- Infective endocarditis
- Arrhythmias

Management

- Mild PS: Often requires no treatment; regular follow-up.
- Moderate to Severe PS:
- Balloon Valvuloplasty: First-line treatment for isolated valvular PS.
- Surgical Valvotomy: Indicated if balloon valvuloplasty is not successful or in cases of complex congenital heart defects.

Special Considerations

- Pregnancy: Women with mild to moderate PS usually tolerate pregnancy well; those with severe PS should be evaluated and managed appropriately before conception.
- Endocarditis Prophylaxis: Recommended for patients with prosthetic valves or a history of endocarditis.

Pulmonary Regurgitation

Definition

Pulmonary regurgitation is the backward flow of blood from the pulmonary artery into the right ventricle during diastole due to the insufficiency of the pulmonary valve.

Etiology

Primary Causes

- Congenital valve abnormalities
 (e.g., tetralogy of Fallot, bicuspid
 pulmonary valve)
- Rheumatic heart disease
- o Infective endocarditis
- Carcinoid syndrome

Secondary Causes

- Pulmonary hypertension
- Right ventricular dilatation
- Post-surgical repair of congenital heart disease (e.g., following repair of tetralogy of Fallot)

Pathophysiology

- Incompetent pulmonary valve allows blood to flow back into the right ventricle during diastole.
- Results in right ventricular volume overload.

• Chronic regurgitation can lead to right ventricular dilatation and dysfunction.

Clinical Features

- Often asymptomatic in mild cases.
- Symptoms may include:
 - Fatigue
 - Dyspnea on exertion
 - Right-sided heart failure (ascites, peripheral edema)
 - Palpitations

Physical Examination

<u>Heart sounds</u>

- Decrescendo diastolic murmur (Graham Steell murmur) best heard at the left upper sternal border.
- Increased intensity of P2 in cases of pulmonary hypertension.

Signs of Right Heart Failure

- Jugular venous distension
- Hepatomegaly
- Peripheral edema

Investigations

- Echocardiography
- Main diagnostic tool.
- Reveals valve morphology, regurgitant flow, right ventricular size and function.

Cardiac MRI

- Provides detailed assessment of right ventricular volume and function.
- Electrocardiogram (ECG)
- May show right ventricular hypertrophy.

Chest X-ray

- May show an enlarged right heart border and increased pulmonary vasculature.
- Cardiac Catheterization
- Used for precise hemodynamic measurements and to assess pulmonary pressures.

Management

Medical Treatment

- Symptomatic management of heart failure.
- $_{\circ}$ Diuretics for fluid overload.
- Management of underlying conditions (e.g., pulmonary hypertension).

Surgical Treatment

- Valve repair or replacement in severe cases with symptomatic right ventricular dysfunction.
- Consideration for intervention in asymptomatic severe PR with evidence of right ventricular dilatation or dysfunction.

Follow-up

- Regular monitoring with echocardiography to assess right ventricular size and function.
- Lifelong follow-up for patients with congenital heart disease.

Prognosis

- Generally good in mild cases.
- Prognosis depends on the underlying cause and the severity of right ventricular dysfunction.

- PR often asymptomatic initially.
- Main diagnostic tool is echocardiography.
- Management focuses on treating the underlying cause and monitoring right ventricular function.
- Severe PR with right ventricular dysfunction may require surgical intervention.

3 Heart Failure

- Systolic Heart Failure
- Diastolic Heart Failure
- Right-Sided Heart Failure
- Left-Sided Heart Failure
- Congestive Heart Failure

Systolic Heart Failure

Definition

Inability of the heart to pump blood effectively due to impaired contraction of the left ventricle.

Etiology

- Ischemic heart disease
- Hypertension
- Dilated cardiomyopathy
- Valvular heart disease

Clinical Features

- Dyspnea on exertion
- Fatigue
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Edema

Pathophysiology

- Decreased contractility leads to reduced ejection fraction (<40%).
- Compensatory mechanisms:
 - Activation of reninangiotensin-aldosterone system
 - Sympathetic nervous system activation
 - Myocardial hypertrophy

Investigations

- Echocardiography: Reduced ejection fraction
- Chest X-ray: Cardiomegaly, pulmonary congestion
- BNP levels: Elevated

Management

Pharmacotherapy

- ACE inhibitors/ARBs
- Beta-blockers
- Diuretics
- Aldosterone antagonists

Device therapy

- Cardiac resynchronization therapy (CRT)
- Implantable cardioverterdefibrillator (ICD)

Lifestyle modifications

- Sodium restriction
- Fluid restriction
- Regular exercise

Prognosis

- Variable depending on underlying cause and response to treatment.
- High morbidity and mortality rates, especially in advanced stages.
- Close monitoring and aggressive management improve outcomes.

Diastolic Heart Failure

Definition

A condition characterized by impaired relaxation or increased stiffness of the ventricles during diastole, leading to decreased ventricular filling and subsequent heart failure symptoms.

Clinical Presentation

- Dyspnea on exertion.
- $_{\circ}\,$ Fatigue.
- $_{\circ}$ Orthopnea.
- Paroxysmal nocturnal dyspnea.
- Preserved ejection fraction.

Epidemiology

- Prevalence increasing with aging population.
- More common in women and those with hypertension.

Pathophysiology

- Impaired ventricular relaxation leads to decreased diastolic filling.
- Elevated filling pressures result in pulmonary congestion and symptoms of heart failure.

Diagnostic Evaluation

- Echocardiography: assess diastolic function, left atrial size, and exclude other causes.
- B-type natriuretic peptide(BNP) levels: may be elevated.
- Invasive hemodynamic monitoring in selected cases.

Management

- Control of underlying conditions (hypertension, diabetes).
- Diuretics for symptom relief from volume overload.
- Beta-blockers and calcium channel blockers for rate control and symptom management.
- Lifestyle modifications: salt restriction, weight loss, exercise.
- Consideration of anti-fibrotic agents in selected cases.

Prognosis

- Often underestimated due to preserved ejection fraction.
- Morbidity and mortality rates similar to systolic heart failure.
- Requires aggressive management to improve outcomes.

Complications

- Pulmonary congestion leading to respiratory compromise.
- Increased risk of atrial fibrillation.
- Reduced exercise tolerance and quality of life.

Future Directions

- Research ongoing into targeted therapies for diastolic dysfunction.
- Improved understanding of underlying mechanisms may lead to more effective treatments.
- Emphasis on early detection and intervention to prevent progression to overt heart failure.

Right-Sided Heart Failure

Definition

Inadequate function of the right ventricle to pump blood to the lungs, often due to pulmonary hypertension or left-sided heart failure. Impaired right ventricular function causes blood to back up into systemic circulation, leading to peripheral edema and congestion.

Causes

- Pulmonary hypertension
- Left-sided heart failure (backward failure)
- Chronic lung diseases (e.g., COPD)
- Right ventricular myocardial infarction
- Pulmonary embolism
- Pulmonary valve stenosis

Clinical Features

- Peripheral edema (legs, ankles, sacrum)
- Hepatomegaly
- Ascites
- Jugular venous distention (JVD)
- 。 Hepatojugular reflux (HJR)
- Weight gain due to fluid retention

Pathophysiology

 Increased right ventricular afterload leads to hypertrophy and eventual failure.
Investigations

- Echocardiography: Assess right ventricular function and evaluate for underlying causes.
- Chest X-ray: Enlarged right atrium and ventricle, pulmonary congestion.
- Electrocardiogram (ECG):
 Right axis deviation, right
 ventricular hypertrophy.
- Right heart catheterization:
 Gold standard for diagnosing pulmonary hypertension.

Prognosis

- Prognosis depends on the underlying cause and response to treatment.
- Without intervention, RHF can lead to progressive deterioration and death.

Remember, timely diagnosis and management of right-sided heart failure are crucial to improve patient outcomes and quality of life.

Management

- Treat underlying cause (e.g., pulmonary hypertension, leftsided heart failure).
- Diuretics: Reduce fluid overload and edema.
- Vasodilators: Improve pulmonary circulation and reduce afterload.
- Oxygen therapy: Improve oxygenation and reduce pulmonary vasoconstriction.
- In severe cases, mechanical support or heart transplantation may be necessary.

Left-Sided Heart Failure

Definition

Inadequate pumping of blood by the left side of the heart, leading to congestion in the lungs and systemic circulation.

Etiology

- Coronary artery disease
- Hypertension
- Myocardial infarction
- Valvular heart disease (e.g., aortic stenosis, mitral regurgitation)
- Cardiomyopathies

Pathophysiology

- Impaired left ventricular function leads to decreased cardiac output.
- Increased preload and pressure in the left atrium and pulmonary veins result in pulmonary congestion.
- Fluid leaks into alveoli, causing pulmonary edema.

Clinical Features

- Dyspnea (especially on exertion or at night)
- $_{\circ}$ Orthopnea
- Paroxysmal nocturnal dyspnea
- Fatigue
- Crackles on lung auscultation
- 。 S3 gallop rhythm

Diagnostic Evaluation

- Echocardiography: Assess left ventricular function, valvular abnormalities.
- Chest X-ray: Cardiomegaly, pulmonary congestion.
- BNP (B-type natriuretic peptide) levels: Elevated in heart failure.

Management

- Pharmacotherapy: ACE inhibitors, beta-blockers, diuretics, aldosterone antagonists.
- Lifestyle modifications: Sodium restriction, fluid restriction, weight management.
- Device therapy: Cardiac resynchronization therapy (CRT), implantable cardioverter-defibrillator (ICD).
- Surgical interventions:
 Valve repair or
 replacement, coronary
 artery bypass grafting
 (CABG).

Prognosis

- Left-sided heart failure is associated with significant morbidity and mortality.
- Prognosis depends on the underlying cause, severity of symptoms, and response to treatment.

Complications

- Pulmonary edema
- Arrhythmias
- Renal dysfunction
- Hepatic congestion
- Cardiogenic shock

Prevention

- Control of hypertension and diabetes
- Lifestyle modifications

 (e.g., smoking cessation, regular exercise)
- Early detection and management of cardiac conditions
- Patient education on medication adherence and symptom recognition.

Congestive Heart Failure (CHF)

Definition

 CHF is a chronic condition where the heart is unable to pump blood effectively, leading to fluid buildup in the lungs and other parts of the body.

Etiology

- Common causes include coronary artery disease, hypertension, valvular heart disease, and cardiomyopathy.
- Other factors: obesity, diabetes, smoking, and family history.

Pathophysiology

- Heart's pumping efficiency decreases, leading to inadequate circulation.
- Compensatory mechanisms (e.g., neurohormonal activation) initially maintain cardiac output but become detrimental over time.

Clinical Manifestations

- Dyspnea (especially on exertion), fatigue, orthopnea, paroxysmal nocturnal dyspnea.
- Peripheral edema, ascites, hepatomegaly.
- Pulmonary crackles, decreased exercise tolerance.

Diagnostic Evaluation

- Echocardiography: Assess ventricular function, valvular abnormalities.
- BNP (B-type natriuretic peptide): Elevated levels suggestive of heart failure.
- Chest X-ray: Cardiomegaly, pulmonary congestion.

Treatment

- Lifestyle modifications:
 Sodium restriction, fluid
 restriction, weight management,
 smoking cessation.
- Medications: Diuretics, ACE inhibitors/ARBs, beta-blockers, aldosterone antagonists.
- Device therapy: CRT (Cardiac Resynchronization Therapy),
 ICD (Implantable Cardioverter Defibrillator).
- Surgical options: Coronary artery bypass grafting (CABG), valve repair/replacement, heart transplantation.

Complications

- Arrhythmias: Atrial fibrillation, ventricular tachycardia.
- Thromboembolism: Risk of stroke due to stasis and atrial fibrillation.
- Renal dysfunction: Reduced renal perfusion due to decreased cardiac output.

Prognosis

- CHF has a variable prognosis depending on underlying etiology, severity, and response to treatment.
- Regular follow-up and adherence to treatment are crucial for improving outcomes.

Prevention

- Control of risk factors: Hypertension, diabetes, dyslipidemia.
- Regular exercise, healthy diet, smoking cessation.
- Timely management of cardiovascular diseases to prevent progression to heart failure.

4

Arrhythmias

Supraventricular Arrhythmias

- Atrial Fibrillation (AF)
- Atrial Flutter
- Paroxysmal Supraventricular Tachycardia (PSVT)

Ventricular Arrhythmias

- Ventricular Tachycardia (VT)
- Ventricular Fibrillation (VF)
- Torsades de Pointes

Bradyarrhythmias

- Sinus Bradycardia
- Heart Block (First-degree, Second-degree, Thirddegree)

Conduction Disorders

- Wolff-Parkinson-White (WPW) Syndrome
- Bundle Branch Blocks

Atrial Fibrillation

Definition

- A common arrhythmia characterized by rapid, irregular atrial contractions.
- Atrial electrical activity disorganized, leading to ineffective atrial contraction.

Epidemiology

- Prevalence increases with age.
- Higher incidence in patients with underlying heart disease.

Etiology

- Hypertension
- Coronary artery disease
- Valvular heart disease
- Thyroid disorders
- Alcohol consumption
- Sleep apnea
- Stimulant use (e.g., caffeine, nicotine)

Clinical Features

- Palpitations
- Fatigue
- Dyspnea
- Chest discomfort
- Dizziness or syncope
- Reduced exercise tolerance

Diagnosis

- Electrocardiogram (ECG) showing absence of P waves, irregular R-R intervals.
- Holter monitor for intermittent monitoring.
- Echocardiography to assess underlying structural heart disease.

Classification

- Paroxysmal AF: Selfterminating episodes lasting < 7 days.
- Persistent AF: Sustained beyond 7 days, requiring intervention for termination.
- Long-standing persistent AF:
 Continuous AF lasting > 1 year.
- Permanent AF: Decided upon by patient and physician to not pursue rhythm control.

Management

- Rate control: Beta-blockers, calcium channel blockers, digoxin.
- Rhythm control: Antiarrhythmic drugs, electrical cardioversion.
- Anticoagulation: Reduce risk of thromboembolic events (e.g., stroke).
- Catheter ablation: For symptomatic patients refractory to medications.
- Lifestyle modifications: Alcohol cessation, weight loss, sleep apnea management.

Complications

- Stroke: Higher risk due to stasis of blood in the atria.
- Heart failure exacerbation.
- Reduced exercise tolerance.
- Impaired quality of life.

Prognosis

- Depends on underlying comorbidities and rate of complications.
- Early detection and appropriate management improve outcomes.

Follow-up

- Regular monitoring for symptom control, rhythm assessment, and anticoagulation management.
- Patient education on symptom recognition and importance of adherence to medications.

Atrial Flutter

Definition

Rapid, regular atrial rhythm characterized by a sawtooth pattern on ECG.

Etiology

Often associated with structural heart disease (e.g., CAD, valve disorders), pulmonary embolism, hyperthyroidism.

Pathophysiology

Reentrant circuit within atria leads to rapid depolarization, causing atrial contraction at rates of 250-350 bpm.

Clinical Presentation

Palpitations, dizziness, dyspnea, fatigue, chest discomfort.

ECG Findings

Sawtooth "F waves" (atrial flutter waves) with atrial rate typically 250-350 bpm and 2:1, 3:1, or variable AV conduction.

Management

- Acute: Cardioversion if hemodynamically unstable.
- Rate control: Beta-blockers, calcium channel blockers, digoxin.
- Rhythm control: Antiarrhythmic drugs (e.g., amiodarone, flecainide), catheter ablation.
- Anticoagulation: Assess stroke risk using CHA2DS2-VASc score; anticoagulate accordingly.

Complications

- Stroke (thromboembolism)
- Heart failure exacerbation,
- Tachycardia-induced cardiomyopathy.

Prognosis

Generally favorable with appropriate management, but recurrence risk exists.

Paroxysmal Supraventricular Tachycardia (PSVT)

Definition

PSVT is a rapid heart rate originating above the ventricles, often characterized by sudden onset and termination.

Etiology

- Reentry circuit involving atrioventricular (AV) node, accessory pathway, or both.
- Triggers include stress, caffeine, alcohol, and tobacco.

Clinical Features

- Palpitations, chest discomfort, lightheadedness, and syncope.
- Rapid regular heartbeat usually between 150-250 bpm.
- $_{\circ}~$ Often begins and ends suddenly.

Diagnosis

- Electrocardiogram (ECG) during an episode.
- Rule out other causes of tachycardia (e.g., atrial fibrillation, atrial flutter).

Management

- Vagal maneuvers (Valsalva, carotid sinus massage).
- Adenosine for acute termination.
- Beta-blockers, calcium channel blockers for rate control.
- Catheter ablation for recurrent or symptomatic cases.

Complications

- Hemodynamic compromise.
- Recurrent episodes may lead to cardiomyopathy.

Prognosis

- Generally benign but can impact quality of life.
- Excellent prognosis with appropriate management.

Prevention

- Avoid triggers.
- Long-term management with medications or catheter ablation when indicated.

Ventricular Tachycardia (VT)

Definition

Rapid heart rhythm originating in the ventricles with a rate exceeding 100 beats per minute.

Etiology

- Structural heart disease (e.g., myocardial infarction, cardiomyopathy)
- Electrolyte abnormalities (e.g., hypokalemia, hypomagnesemia)
- Drug toxicity (e.g., antiarrhythmics, digitalis)
- Inherited arrhythmia syndromes (e.g., long QT syndrome, Brugada syndrome)

Clinical Features

- Palpitations
- Dizziness or syncope
- Hypotension
- Cardiac arrest

Diagnosis

- Electrocardiogram (ECG)
 showing wide QRS complexes (>
 120 ms) with a rate > 100 bpm.
- 12-lead ECG for morphology assessment.

Management

Stable VT

- Medications: Amiodarone, procainamide, sotalol.
- Electrical cardioversion if medications fail.

Unstable VT

 Immediate synchronized cardioversion.

Recurrent VT

- Consideration of antiarrhythmic therapy.
- Implantable cardioverterdefibrillator (ICD) for high-risk patients.

Prognosis

- Depends on underlying etiology and comorbidities.
- VT in the setting of acute myocardial infarction carries a worse prognosis.

Complications

- Hemodynamic compromise leading to shock or cardiac arrest.
- Recurrent episodes may lead to ventricular fibrillation and sudden cardiac death.

Prevention

- Identification and treatment of underlying structural heart disease.
- Avoidance of triggers (e.g., electrolyte imbalances, QTprolonging medications).

Follow-up

- Regular monitoring of symptoms and ECG findings.
- Adjustment of medications as needed.

Education

- Patient education on recognizing symptoms and seeking prompt medical attention.
- Counseling on lifestyle modifications and adherence to medications.

Ventricular Fibrillation (VF)

Definition

Rapid, irregular, and ineffective heart rhythm originating from the ventricles.

Etiology

Often precipitated by acute myocardial infarction (AMI), electrolyte imbalances, drug toxicity, or cardiac trauma.

Pathophysiology

Chaotic electrical activity leads to uncoordinated ventricular contractions, compromising cardiac output.

Clinical Features

Sudden loss of consciousness, absence of pulse, and absence of effective circulation.

Diagnosis

ECG reveals irregular undulations without discernible QRS complexes.

Management

- Immediate Defibrillation:
 Priority to restore normal rhythm and perfusion.
- CPR: Initiate chest compressions promptly to maintain blood flow.
- Medications: Epinephrine and antiarrhythmic drugs like amiodarone may be administered.
- Advanced Life Support
 (ALS): Rapid transport to a facility capable of advanced cardiac care.

Prognosis

Without prompt intervention, VF leads to cardiac arrest and death within minutes.

Prevention

Emphasize public education on CPR, early defibrillation, and prompt medical response.

Torsades de Pointes (TdP)

Definition

A specific form of polymorphic ventricular tachycardia characterized by a twisting of the QRS complexes around the isoelectric baseline.

Causes

- Prolonged QT interval (acquired or congenital)
- Electrolyte imbalances (especially hypokalemia and hypomagnesemia)
- Medications (e.g., antiarrhythmics, antibiotics, antipsychotics)
- Structural heart disease
- Bradycardia

Clinical Presentation

- Palpitations
- Syncope
- Seizures
- Cardiac arrest

Diagnosis

- ECG showing characteristic twisting pattern
- Prolonged QT interval

Management

- Correct underlying electrolyte abnormalities
- Discontinue offending medications
- IV magnesium sulfate
- Isoproterenol or temporary pacing for bradycardia
- Immediate defibrillation if unstable

Long-Term Management

- Avoid QT-prolonging drugs
- Monitor electrolytes regularly
- Consider implantable cardioverter-defibrillator (ICD) for high-risk patients

Prognosis

 Mortality risk is higher in patients with underlying structural heart disease or recurrent episodes.

Prevention

- Education about QTprolonging medications
- Regular follow-up for monitoring and management of risk factors.

Sinus Bradycardia

Definition

Heart rhythm characterized by a slow sinus rate, typically <60 beats per minute.

Etiology

- Physiological: Seen in athletes, during sleep, and in healthy individuals.
- Pathological: May result from intrinsic cardiac disorders (e.g., sick sinus syndrome), increased vagal tone, medications (e.g., beta-blockers, calcium channel blockers), hypothyroidism, electrolyte imbalances (e.g., hyperkalemia), or increased intracranial pressure.

Clinical Features

- Often asymptomatic.
- Symptoms may include fatigue, dizziness, syncope, or angina in severe cases.

Diagnosis

Electrocardiogram (ECG) showing regular P waves originating from the sinus node, with a heart rate <60 bpm.

Management

- Treat the underlying cause.
- Consider discontinuation or adjustment of medications contributing to bradycardia.
- In symptomatic patients, particularly those with hemodynamic compromise, temporary pacing or permanent pacemaker implantation may be necessary.

Prognosis

Generally good, especially if the underlying cause is reversible and promptly addressed.

Heart Block

Definition

Heart block, also known as atrioventricular (AV) block, is a condition where the conduction of electrical impulses through the AV node is impaired, resulting in delayed or blocked signals between the atria and ventricles.

Classification

- 1. First-Degree AV Block
- Description: Prolonged PR interval (>200 ms) without missed beats.
- **ECG Findings:** PR interval consistently longer than 200 ms.
- Clinical Significance: Often asymptomatic; may indicate underlying heart disease or medication effect.
- 2. Second-Degree AV Block

Type I (Wenckebach/Mobitz I)

- Description: Progressive lengthening of PR interval until a beat is dropped.
- ECG Findings: Progressive PR lengthening followed by a nonconducted P wave (dropped QRS).
- Clinical Significance: Usually benign; may cause occasional symptoms like dizziness.

Type II (Mobitz II)

• **Description:** Intermittent nonconduction of P waves without PR interval prolongation.

- ECG Findings: Constant PR interval with random dropped beats.
- Clinical Significance: More serious; can progress to complete block; often requires pacemaker.
- 3. Third-Degree (Complete) AV Block
 - Description: Complete absence of AV conduction; atria and ventricles beat independently.
 - ECG Findings: P waves and QRS complexes present but no relationship between them.
 - Clinical Significance: High risk of asystole and sudden death; usually requires pacemaker.

Etiology

- Intrinsic Causes:
 - Aging (degenerative changes)
 - o Ischemic heart disease
 - Cardiomyopathies
 - o Congenital heart defects
- Extrinsic Causes:
 - Medications (beta-blockers, calcium channel blockers, digoxin)
 - Electrolyte imbalances (hyperkalemia)
 - Inflammatory diseases (myocarditis, Lyme disease)

Clinical Presentation

- First-Degree: Usually asymptomatic.
- Second-Degree Type I: Generally asymptomatic or mild symptoms (dizziness, fatigue).
- Second-Degree Type II and Third-Degree: More severe symptoms (syncope, severe fatigue, heart failure symptoms).

Diagnosis

- History and Physical Examination: Check for symptoms, medication use, and underlying conditions.
- Electrocardiogram (ECG): Key diagnostic tool to identify the type and degree of block.
- Holter Monitoring: For intermittent blocks.
- Electrophysiological Studies: In complex cases to localize the block.

Management

- First-Degree and Second-Degree Type I:
 - Often no treatment needed.
 - Monitor for progression.
- Second-Degree Type II and Third-Degree:
 - Pacemaker implantation is the primary treatment.
 - Address underlying causes (e.g., discontinue causative medications).
 - Temporary pacing may be needed in emergency situations.

Prognosis

- First-Degree and Second-Degree Type I: Generally good with monitoring.
- Second-Degree Type II and Third-Degree: Variable, depends on timely intervention with pacemaker and underlying health.

Key Points

- Early recognition and differentiation of heart block types are crucial.
- Pacemaker therapy significantly improves outcomes in higherdegree AV blocks.
- Regular follow-up is necessary to monitor for progression and manage symptoms.

Wolff-Parkinson-White (WPW) Syndrome

Definition

WPW Syndrome is a congenital heart disorder characterized by an abnormal accessory electrical pathway between the atria and ventricles, leading to episodes of rapid heart rate (tachycardia).

Pathophysiology

The syndrome arises due to the presence of an accessory pathway called the Bundle of Kent, which allows electrical impulses to bypass the normal pathway (AV node) between the atria and ventricles, causing premature ventricular excitation.

Clinical Features

- Palpitations
- Rapid or irregular heartbeat (tachycardia)
- Dizziness or lightheadedness
- Syncope (fainting), especially during episodes of tachycardia

Diagnosis

- Electrocardiogram (ECG): Characteristic findings include a shortened PR interval (<0.12 seconds), widened QRS complex (>0.12 seconds) with a delta wave (slurred upstroke), and a short QT interval.
- Exercise stress test: Helpful in inducing symptoms and evaluating heart rate response.
- Holter monitor: Useful for capturing intermittent arrhythmias.

Management

Acute Management

Vagal maneuvers (e.g., Valsalva maneuver), adenosine administration, or synchronized cardioversion for symptomatic tachycardia.

Long-Term Management:

- Depends on symptoms and risk stratification.
- **Asymptomatic:** No specific therapy may be needed, but regular follow-ups are essential.
- Symptomatic: Medications (e.g., betablockers, calcium channel blockers), radiofrequency catheter ablation to eliminate the accessory pathway, or surgical intervention (rarely).

Complications

- Atrial fibrillation/flutter
- Sudden cardiac death (rare but can occur due to rapid ventricular response during atrial fibrillation/flutter)
- Heart failure (in severe cases or with persistent tachycardia)

Prognosis

With appropriate management, the prognosis is generally favorable. Radiofrequency catheter ablation is curative in most cases, with low recurrence rates. However, regular monitoring is necessary due to the risk of arrhythmia recurrence.

Bundle Branch Blocks (BBB)

Definition

Interruption or delay in the conduction of electrical impulses through the bundle branches of the heart.

Types

- Right Bundle Branch Block (RBBB): Impaired conduction through the right bundle branch.
- Left Bundle Branch Block (LBBB): Impaired conduction through the left bundle branch.

Electrocardiogram (ECG) Findings

- **RBBB**: Wide QRS complex (>0.12 seconds), predominantly in V1 and V2.
- LBBB: Wide QRS complex (>0.12 seconds), predominantly in V5 and V6.

Clinical Significance

- Often incidental findings.
- Can indicate underlying heart disease, particularly in older patients.
- May occur in conditions like myocardial infarction, cardiomyopathy, and electrolyte imbalances.

Symptoms

- $_{\circ}~$ Often asymptomatic.
- Symptoms, if present, can include palpitations, dizziness, or syncope.

Management

- Treat underlying conditions if present.
- Close monitoring for progression or development of associated cardiac issues.

Prognosis

- Generally benign if isolated.
- May indicate increased risk of arrhythmias or heart failure in certain contexts.

Differential Diagnosis

 Other causes of widened QRS complex on ECG should be considered, such as ventricular tachycardia, electrolyte abnormalities, or ventricular paced rhythms.

Follow-Up

- Regular monitoring of cardiac function and ECG changes.
- Educate patients about symptoms and when to seek medical attention.

Conclusion

- Bundle Branch Blocks are common ECG findings.
- Understanding their significance and implications for patient care is crucial in clinical practice.

5 Cardiomyopathies

- Dilated Cardiomyopathy (DCM)
- Hypertrophic Cardiomyopathy (HCM)
- Restrictive Cardiomyopathy (RCM)
- Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)
- Takotsubo Cardiomyopathy (Stress Cardiomyopathy)
- Peripartum Cardiomyopathy

Dilated Cardiomyopathy (DCM)

Definition

A condition characterized by the dilation and impaired contraction of the left or both ventricles.

Epidemiology

- **Prevalence:** Affects approximately 1 in 2500 individuals.
- Age: Can occur at any age but most commonly diagnosed in adults between 20-60 years.
- **Gender:** More prevalent in males than females.

Etiology

- Idiopathic: Unknown cause in many cases.
- **Genetic:** 20-35% cases are familial; mutations in sarcomeric proteins, cytoskeletal proteins.
- Infectious: Viral myocarditis (e.g., Coxsackievirus, Adenovirus).
- **Toxic:** Alcohol abuse, cocaine, chemotherapeutic agents (e.g., Doxorubicin).
- **Metabolic:** Thyroid disorders, nutritional deficiencies (e.g., thiamine, selenium).

- Autoimmune: Sarcoidosis, systemic lupus erythematosus (SLE).
- **Peripartum:** Occurs during the last month of pregnancy or within five months postpartum.

Pathophysiology

- Ventricular Dilation: Leads to impaired systolic function.
- Increased Ventricular Volumes: Causes a rise in end-diastolic pressure.
- Mitral/Tricuspid Regurgitation: Due to annular dilation.
- Heart Failure: Both systolic and diastolic dysfunctions contribute to heart failure symptoms.

Clinical Features

- **Symptoms:** Dyspnea, orthopnea, paroxysmal nocturnal dyspnea, fatigue, edema.
- Signs: Jugular venous distension, displaced apical impulse, S3 gallop, pulmonary rales, hepatomegaly.

Diagnosis

- Echocardiography: Key imaging modality showing ventricular dilation, reduced ejection fraction.
- ECG: Non-specific findings, may show LV hypertrophy, atrial fibrillation.
- **Chest X-ray:** Cardiomegaly, pulmonary congestion.
- **MRI:** For detailed myocardial structure assessment.
- **Biopsy:** Endomyocardial biopsy in selected cases to rule out specific causes like myocarditis.

Treatment

Medical Management

- **ACE Inhibitors/ARBs:** Reduce afterload, improve survival.
- **Beta-Blockers:** Improve symptoms, reduce mortality.
- **Diuretics:** For fluid overload management.
- Aldosterone Antagonists: For patients with severe symptoms.
- **Digoxin:** In selected patients to improve symptoms.
- Anticoagulation: In patients with atrial fibrillation or history of thromboembolism.

Device Therapy

 Implantable Cardioverter-Defibrillator (ICD): For primary/secondary prevention of sudden cardiac death. Cardiac Resynchronization Therapy (CRT): In patients with ventricular dyssynchrony.

Surgical/Interventional:

- LVAD (Left Ventricular Assist Device): For advanced heart failure.
- **Heart Transplantation:** Considered in end-stage disease unresponsive to medical therapy.

Prognosis

- Variable: Dependent on etiology, severity at diagnosis, response to treatment.
- Mortality: Higher in patients with advanced heart failure symptoms.

Follow-Up

- **Regular Monitoring:** Clinical assessment, echocardiography, and lab tests.
- Lifestyle Modifications: Low-sodium diet, fluid restriction, exercise recommendations tailored to the individual.

Prevention

- Genetic Counseling: For familial cases.
- Managing Risk Factors: Control of hypertension, diabetes, avoidance of alcohol and illicit drugs.

Hypertrophic Cardiomyopathy (HCM)

Definition

A genetic cardiovascular disease characterized by left ventricular hypertrophy (LVH) not explained by secondary causes, leading to various cardiac issues.

Epidemiology

- **Prevalence**: Approximately 1 in 500 adults.
- **Genetics**: Autosomal dominant inheritance; mutations in sarcomeric protein genes.

Pathophysiology

- Cardiac Hypertrophy: Asymmetric septal hypertrophy most common, but can involve any part of the left ventricle.
- Myofibrillar Disarray: Disorganized muscle fibers.
- **Diastolic Dysfunction**: Stiff ventricular walls impair filling.
- **Obstruction**: Dynamic left ventricular outflow tract obstruction (LVOTO) in some patients.

• Mitral Valve Abnormalities: Systolic anterior motion (SAM) of the mitral valve.

Clinical Features

- **Symptoms**: Dyspnea, chest pain, palpitations, syncope, sudden cardiac death (SCD).
- **Signs**: Harsh systolic murmur (increased with Valsalva maneuver), S4 gallop, bifid carotid pulse.

Diagnosis

- Echocardiography: Primary diagnostic tool, shows LVH, SAM, and LVOTO.
- **MRI**: Detailed myocardial structure, fibrosis detection.
- ECG: Non-specific changes, LVH, deep Q waves, T wave inversions.
- Genetic Testing: Identifies specific mutations in sarcomeric proteins.

Management

Lifestyle Modifications: Avoidance of intense competitive sports.

Medications:

- Beta-Blockers: First-line for symptom relief.
- Calcium Channel Blockers: Verapamil for non-obstructive HCM.
- Disopyramide: For refractory symptoms.

Surgical Options:

- Septal Myectomy: For severe LVOTO.
- Alcohol Septal Ablation:
 Alternative to myectomy.

Implantable Cardioverter Defibrillator (ICD):

For primary or secondary prevention of SCD.

Prognosis

• Variable: Many live normal lifespans; some at risk for SCD, particularly young athletes.

Monitoring and Follow-up

- Regular Clinical
 Evaluation: Symptom
 assessment, physical exam,

 ECG, and
 echocardiography.
- Family Screening: Genetic counseling and screening for relatives.

Key Points

- Early Detection: Crucial for preventing complications and SCD.
- Multidisciplinary
 Approach: Cardiologists, geneticists, and sometimes surgeons.

• Patient Education: Importance of lifestyle changes and adherence to treatment.

Restrictive Cardiomyopathy (RCM)

Definition

Restrictive Cardiomyopathy is a form of heart muscle disease characterized by diastolic dysfunction due to stiff and noncompliant ventricles, leading to impaired ventricular filling with preserved systolic function.

Etiology

- Primary (Idiopathic)
- Secondary Causes:
 - Infiltrative Diseases:
 Amyloidosis, Sarcoidosis
 - Storage Diseases: Hemochromatosis, Glycogen storage diseases
 - Fibrotic Disorders:
 Scleroderma, Endomyocardial fibrosis
 - Radiation or Chemotherapyinduced Fibrosis

Pathophysiology

 Increased ventricular stiffness and reduced compliance

- Elevated ventricular filling pressures
- Reduced stroke volume and cardiac output
- Normal or near-normal systolic function

Clinical Features

Symptoms

- $_{\circ}$ Dyspnea on exertion
- Fatigue
- Peripheral edema
- Ascites
- Syncope (rare)

<u>Signs</u>

- Elevated jugular venous pressure
- Hepatomegaly
- Ascites
- Peripheral edema
- Kussmaul's sign (increased JVP on inspiration)
- Third heart sound (S3)

Diagnosis

- Echocardiography
 - o Thickened ventricular walls
 - Dilated atria
 - Normal or slightly reduced ventricular size
 - Diastolic dysfunction (restrictive filling pattern)

Cardiac MRI

 Tissue characterization (e.g., late gadolinium enhancement in amyloidosis)

Endomyocardial Biopsy

- Confirms diagnosis in cases of infiltrative diseases
- Electrocardiogram (ECG)
 - Non-specific findings
 - Low voltage QRS in amyloidosis
 - $_{\circ}~$ Atrial fibrillation common

Management

Symptomatic Treatment

- Diuretics for volume overload
- $_{\circ}$ Rate control for atrial fibrillation
- Anticoagulation if atrial fibrillation is present

Disease-Specific Treatments

 Targeted therapy for underlying causes (e.g., chemotherapy for amyloidosis)

Advanced Therapies

 Consider heart transplantation in severe, refractory cases

Prognosis

- Varies widely based on underlying cause
- Generally poor in cases of advanced infiltrative diseases like amyloidosis

Key Points

- Differentiation from other cardiomyopathies and constrictive pericarditis is crucial.
- Comprehensive evaluation including imaging and tissue biopsy is often required.
- Management focuses on symptomatic relief and treating underlying etiology.
- Prognosis depends significantly on the specific cause and severity of disease.

Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

Definition

ARVC is a genetic disorder characterized by the replacement of myocardial tissue with fibrofatty tissue, primarily affecting the right ventricle.

Epidemiology

- Prevalence: Approximately 1 in 1,000 to 5,000 individuals.
- Gender: Slight male predominance.
- Age of Onset: Typically manifests in adolescents and young adults.

Etiology

- Genetic mutations in desmosomal proteins (e.g., plakophilin-2, desmoplakin, plakoglobin).
- Inheritance: Mostly autosomal dominant with variable penetrance.

Pathophysiology

- Progressive loss of myocytes and replacement by fibrofatty tissue.
- Predominantly affects the right ventricle but can involve the left ventricle in advanced stages.
- Leads to arrhythmias, heart failure, and sudden cardiac death (SCD).

Clinical Presentation

- Palpitations, syncope, atypical chest pain.
- Ventricular arrhythmias (e.g., ventricular tachycardia).
- Heart failure symptoms in advanced stages.
- Sudden cardiac death, particularly during exertion.

Diagnostic Criteria (Task Force Criteria)

Major Criteria

- Structural abnormalities on imaging (e.g., RV akinesia, dyskinesia).
- Tissue characterization by biopsy (fibrofatty replacement).
- Repolarization abnormalities (T-wave inversions in right precordial leads).
- Depolarization/conduction abnormalities (epsilon waves on ECG).
- Arrhythmias (sustained or nonsustained VT with LBBB morphology).
- Family history of ARVC or confirmed pathogenic mutation.

Minor Criteria

 Structural, histological, ECG, arrhythmic findings not meeting major criteria.

Diagnostic Tools

- ECG: T-wave inversions, epsilon waves.
- Echocardiography: RV dilatation and dysfunction.
- Cardiac MRI: Fibrofatty infiltration, wall motion abnormalities.
- Endomyocardial biopsy: Histological confirmation.
- Genetic testing: Identification of pathogenic mutations.

Management

Lifestyle Modifications

 Avoid competitive sports and strenuous exercise.

Pharmacological Therapy

 Antiarrhythmic drugs (e.g., beta-blockers, amiodarone).

Device Therapy

 Implantable cardioverterdefibrillator (ICD) for secondary prevention of SCD.

Catheter Ablation

For refractory ventricular tachycardia.

Heart Transplantation

In cases of refractory heart failure.

Prognosis

- Variable; depends on the extent of disease and risk of arrhythmias.
- Regular follow-up and monitoring are essential for managing disease progression and complications.

Key Points

- ARVC is a genetic disorder primarily affecting the right ventricle with significant risk of arrhythmias and sudden cardiac death.
- Diagnosis is based on a combination of clinical, imaging, histological, and genetic criteria.
- Management includes lifestyle modifications, pharmacotherapy, and potentially ICD implantation.
- Regular follow-up is crucial for optimizing patient outcomes.

Takotsubo Cardiomyopathy (Stress Cardiomyopathy)

Definition

A transient, reversible cardiomyopathy characterized by acute, temporary dysfunction of the left ventricle.

Epidemiology

- Predominantly affects postmenopausal women (80-90% of cases).
- Often triggered by acute physical or emotional stress.

Etiology

- Exact cause is unclear.
- Suspected mechanisms include catecholamine excess, microvascular dysfunction, and coronary artery spasm.

Pathophysiology

- Results in a distinctive ballooning of the left ventricular apex with a hyperkinetic base ("apical ballooning").
- Mimics acute coronary syndrome but without significant coronary artery obstruction.

Clinical Features

- Chest pain, dyspnea, syncope.
- Often precipitated by an acute stress event (emotional or physical).

Diagnosis

- **1. Electrocardiogram (ECG)**: STsegment elevation or T-wave inversion.
- 2. Cardiac Biomarkers: Elevated troponin, but usually not as high as in myocardial infarction.
- **3. Echocardiogram**: Demonstrates apical ballooning and left ventricular dysfunction.
- **4. Coronary Angiography**: Normal or non-obstructive coronary arteries.
- 5. Cardiac MRI: Can differentiate from myocarditis and infarction; shows edema and absence of delayed gadolinium enhancement.

Diagnostic Criteria

- Transient hypokinesis, akinesis, or dyskinesis of the left ventricular mid-segments with or without apical involvement.
- Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture.
- 3. New ECG abnormalities or modest elevation in cardiac troponin.
- Absence of pheochromocytoma or myocarditis.

Management

- Supportive care is the mainstay of treatment.
- Beta-blockers and ACE inhibitors for ventricular dysfunction.
- Anticoagulation if left ventricular thrombus is present.
- Avoidance of precipitating stressors.

Prognosis

- Generally good with complete recovery in most cases.
- Recurrence is possible but uncommon.
- Mortality is low but can be higher in the presence of complications such as heart failure or arrhythmias.

Complications

- Heart failure
- Cardiogenic shock
- Left ventricular outflow tract
 obstruction
- Mitral regurgitation
- Ventricular arrhythmias
- Thromboembolism

Follow-Up

- Regular follow-up to monitor ventricular function.
- Psychological support may be beneficial to address stress management.

Peripartum Cardiomyopathy (PPCM)

Definition

Peripartum Cardiomyopathy (PPCM) is a form of heart failure that occurs during the last month of pregnancy or within five months postpartum without any other identifiable cause of heart failure.

Etiology and Risk Factors

- **Unknown cause**; multifactorial hypotheses include:
 - Genetic predisposition
 - Autoimmune responses
 - Viral infections
 - Nutritional deficiencies
- Risk factors:
 - Advanced maternal age
 - Multiparity (having given birth more than once)
 - Multiple gestations (twins, triplets, etc.)
 - o Hypertension or preeclampsia
 - African descent

Pathophysiology

- **Cardiac remodeling**: Dilation of the left ventricle with decreased systolic function
- Inflammation and oxidative stress: Potential involvement in myocardial damage

Clinical Presentation

- Symptoms
 - Fatigue
 - Dyspnea (shortness of breath) on exertion or at rest
 - Orthopnea (difficulty breathing when lying flat)
 - Paroxysmal nocturnal dyspnea (sudden nighttime breathing difficulty)
 - Peripheral edema (swelling of legs, ankles)
- Signs
 - Elevated jugular venous pressure (JVP)
 - S3 gallop (third heart sound)
 - Pulmonary rales (crackles)
 - Cardiomegaly (enlarged heart) on chest X-ray

Diagnosis

- Echocardiography: Key diagnostic tool
 - Left ventricular ejection fraction (LVEF)
 < 45%
 - o Dilated left ventricle
- Biomarkers: Elevated BNP or NTproBNP levels
- Exclusion of other causes: Comprehensive evaluation to rule out other cardiomyopathies or causes of heart failure

Management

- Medical therapy
 - Standard heart failure treatment (with caution regarding pregnancy and breastfeeding safety):
 - ACE inhibitors/ARBs (contraindicated during pregnancy, used postpartum)
 - Beta-blockers (e.g., metoprolol, carvedilol)
 - Diuretics (to manage fluid overload)
 - Anticoagulation if indicated (e.g., in cases of significant left ventricular dysfunction)

Lifestyle modifications

- Sodium restriction
- Fluid restriction
- Avoidance of subsequent pregnancies (recommended in cases of persistent left ventricular dysfunction)

Monitoring

- Regular follow-up with echocardiography
- Monitoring of symptoms and functional status

Prognosis

- Variable outcomes:
 - **Recovery**: Some patients fully recover left ventricular function
 - Persistent dysfunction: Others may have ongoing heart failure or progressive disease

Factors influencing prognosis:

- Severity of initial left ventricular dysfunction
- Timeliness and effectiveness of treatment
- Absence of significant comorbidities

Complications

- Heart failure: Chronic condition with potential for acute exacerbations
- Thromboembolism: Due to stasis of blood in a dilated heart
- Arrhythmias: Increased risk of both atrial and ventricular arrhythmias

Key Points

- High index of suspicion required in peripartum women with heart failure symptoms.
- Early diagnosis and management crucial to improve outcomes.
- Multidisciplinary approach involving cardiologists, obstetricians, and primary care providers essential.

Congenital Heart Diseases

Cyanotic Heart Diseases

- Tetralogy of Fallot
- Transposition of the Great Arteries
- Truncus Arteriosus
- Total Anomalous Pulmonary Venous Connection (TAPVC)

Acyanotic Heart Diseases

- Ventricular Septal Defect (VSD)
- Atrial Septal Defect (ASD)
- Patent Ductus Arteriosus (PDA)
- Coarctation of the Aorta

Tetralogy of Fallot (TOF)

Definition

Congenital heart defect characterized by four anatomical abnormalities:

- 1. Ventricular Septal Defect (VSD)
- 2. Pulmonary Stenosis
- 3. Right Ventricular Hypertrophy
- 4. Overriding Aorta

Pathophysiology

- VSD allows mixing of oxygenated and deoxygenated blood.
- Pulmonary stenosis causes right ventricular outflow obstruction, leading to right-to-left shunt.
- Overriding aorta receives blood from both ventricles, increasing systemic circulation of deoxygenated blood.
- Right ventricular hypertrophy results from increased workload due to pulmonary stenosis.

Clinical Features

- Cyanosis (especially during crying or feeding in infants)
- "Tet spells" (hypercyanotic episodes)
- Digital clubbing
- Dyspnea on exertion
- Squatting posture (in children) to relieve symptoms

Diagnosis

- Echocardiography: Main diagnostic tool, visualizes the four defects.
- Chest X-ray: "Boot-shaped" heart due to right ventricular hypertrophy.
- Electrocardiogram (ECG): Right axis deviation and right ventricular hypertrophy.
- Cardiac catheterization: Assesses the severity of pulmonary stenosis and other defects.

Management

Medical

Prostaglandin E1 to maintain ductus arteriosus patency in newborns; beta-blockers for hypercyanotic spells.

Surgical

Complete repair typically performed in infancy, involving VSD closure and relief of right ventricular outflow tract obstruction.

Palliative

Blalock-Taussig shunt to increase pulmonary blood flow if complete repair is not immediately possible.

Prognosis

- With surgical repair, most patients achieve significant improvement.
- Long-term follow-up required for residual defects, arrhythmias, and right ventricular function.

Complications

- Residual VSD
- Pulmonary regurgitation
- Arrhythmias
- Heart failure
- Infective endocarditis

Special Considerations

- Genetic counseling may be indicated as TOF can be associated with genetic syndromes (e.g., Down syndrome, DiGeorge syndrome).
- Pregnancy in women with repaired TOF requires close monitoring due to increased cardiovascular demands.

Summary

Tetralogy of Fallot is a critical congenital heart defect with characteristic anatomical abnormalities. Early diagnosis and timely surgical intervention significantly improve outcomes, though lifelong monitoring is essential due to potential complications and long-term sequelae.

Transposition of the Great Arteries (TGA)

Definition

• Transposition of the Great Arteries (TGA): A congenital heart defect where the two main arteries leaving the heart are reversed (transposed), resulting in parallel rather than series circulation.

Epidemiology

• **Incidence**: Approximately 1 in 3,500 to 5,000 live births.

Types

d-TGA (Dextro-TGA): The aorta arises from the right ventricle and the pulmonary artery from the left ventricle (more common).

I-TGA (Levo-TGA): The ventricles and corresponding great arteries are both transposed.

Pathophysiology

- Circulatory Disconnection: Systemic and pulmonary circulations run in parallel, leading to cyanosis.
- Mixing Sites: Patent foramen ovale (PFO), atrial septal defect (ASD),

ventricular septal defect (VSD), or patent ductus arteriosus (PDA) are critical for oxygenated blood mixing.

Clinical Features

- **Symptoms**: Cyanosis (bluish skin), tachypnea, poor feeding, failure to thrive, and hypoxia.
- **Signs**: Central cyanosis, loud second heart sound (single S2), murmur if VSD present.

Diagnosis

- **Prenatal Ultrasound**: Can detect TGA before birth.
- Echocardiography: Definitive diagnostic tool, visualizes the abnormal origin of the great arteries.
- Cardiac MRI/CT: Detailed anatomy, especially in complex cases.
- Chest X-ray: "Egg on a string" appearance due to narrow mediastinum.
- ECG: Right ventricular hypertrophy.
Management

Initial Stabilization

- Prostaglandin E1 Infusion: To maintain PDA and enhance mixing.
- Balloon Atrial Septostomy
 (BAS): Rashkind procedure to create or enlarge ASD for better oxygenation.

Definitive Surgical Repair

- Arterial Switch Operation

 (ASO): Corrective surgery
 within the first few weeks of life;
 repositions great arteries and
 re-implants coronary arteries.
- Atrial Switch Procedures
 (Senning/Mustard): Historical
 procedures, now largely
 replaced by ASO.

Prognosis

- Surgical Outcomes: Excellent with ASO, low mortality if performed early.
- Long-term Issues: Possible coronary artery complications, arrhythmias, or heart failure; requires lifelong follow-up.

Monitoring and Follow-up

- **Postoperative Care**: Regular cardiology visits, echocardiograms, ECGs.
- Growth and Development: Monitoring for normal development and addressing any developmental delays.
- Endocarditis Prophylaxis: As needed for certain procedures.

Key Points

- **Early Diagnosis**: Crucial for timely intervention and prevention of severe hypoxia.
- Family Education: Importance of recognizing symptoms and adhering to follow-up schedules.
- Lifelong Care: Continued surveillance for potential late complications post-surgery.

Truncus Arteriosus

Definition

Truncus arteriosus is a congenital heart defect characterized by the failure of septation of the embryonic truncus arteriosus into the aorta and pulmonary artery, resulting in a single arterial vessel arising from the heart.

Anatomy

- Single arterial trunk arises from both ventricles.
- The trunk gives off coronary arteries and branches to the systemic, pulmonary, and coronary circulations.
- A ventricular septal defect (VSD) is always present.

Classification

- **Type I:** Single arterial trunk gives rise to pulmonary arteries and systemic circulation branches.
- Type II (Rarer): Single arterial trunk gives rise to pulmonary arteries but receives a separate origin of the coronary arteries.

Clinical Features

- Cyanosis: Due to mixing of oxygenated and deoxygenated blood.
- Heart Murmur: Continuous murmur due to the large VSD.
- Congestive Heart Failure (CHF): Symptoms may present in infancy due to increased pulmonary blood flow.

Diagnosis

- Echocardiography Confirms the diagnosis, assesses the anatomy, and evaluates associated anomalies.
- Cardiac Catheterization May be performed for further evaluation.

Management

Surgical Repair

Typically done in infancy.

- Close VSD with patch repair.
- Separate pulmonary arteries from the common arterial trunk.
- o Reconstruct the aorta.

Prostaglandin Infusion

To maintain ductal patency in neonates until surgical repair.

<u>Medical Management of</u> <u>Heart Failure</u>

Diuretics, afterload reduction.

Prognosis

- Surgical outcomes have significantly improved with modern techniques.
- Long-term outcomes depend on the extent of associated anomalies and postoperative complications.

Key Points

- Truncus arteriosus requires surgical correction in infancy.
- Close monitoring for associated anomalies and postoperative complications is essential.
- Collaboration between pediatric cardiologists, surgeons, and neonatologists is crucial for optimal management.

Total Anomalous Pulmonary Venous Connection (TAPVC)

Definition

Congenital heart defect where all pulmonary veins fail to connect to the left atrium, instead draining into systemic veins or right atrium.

Types

- Supracardiac (50-60%)
- Cardiac (15-20%)
- Infracardiac (20-30%)
- Mixed (5-10%)

Pathophysiology

- Mixed oxygenated and deoxygenated blood due to abnormal venous connection.
- Pulmonary hypertension due to increased pulmonary blood flow.
- Right-sided heart failure in severe cases.

Clinical Presentation

- Cyanosis (depends on degree of pulmonary venous obstruction).
- Tachypnea, respiratory distress.
- Signs of heart failure (e.g., hepatomegaly, peripheral edema).
- Murmur may be absent or nonspecific.

Diagnostic Evaluation

- Echocardiography: Initial imaging modality.
- Cardiac catheterization: Confirms diagnosis, assesses pulmonary vascular resistance.

- Chest X-ray: Cardiomegaly, pulmonary congestion.
- Electrocardiogram: Right atrial enlargement, RV hypertrophy.

Management

- **Stabilization:** Oxygen, diuretics for heart failure.
- Surgical Correction: Definitive treatment.
 - Reconnection of pulmonary veins to left atrium.
 - o Repair of associated defects.
- **Prognosis:** Depends on type of TAPVC, degree of pulmonary hypertension, and associated anomalies.

Key Points

- TAPVC is a congenital heart defect where pulmonary veins fail to connect normally to the left atrium.
- Clinical presentation includes cyanosis, respiratory distress, and signs of heart failure.
- Diagnosis is confirmed by echocardiography and cardiac catheterization.
- Surgical correction is the definitive treatment, aiming to restore normal pulmonary venous drainage.
- Prognosis varies depending on the type and severity of TAPVC, as well as associated anomalies. Early detection and intervention are crucial for optimal outcomes.

Ventricular Septal Defect (VSD)

Definition

Congenital heart defect characterized by an abnormal opening in the ventricular septum, allowing blood to flow between the left and right ventricles.

Epidemiology

- Most common congenital heart defect.
- Often diagnosed in infancy or childhood.

Types

- **1. Perimembranous (Most Common)**: Adjacent to the membranous septum.
- 2. Muscular: Involves the muscular septum.
- 3. Supracristal: Near the aortic valve.
- **4. Subpulmonic (Infundibular)**: Beneath the pulmonary valve.

Pathophysiology

- Left-to-right shunt occurs due to the pressure difference between the left and right ventricles.
- Increased pulmonary blood flow leads to pulmonary hypertension and right ventricular hypertrophy.

Clinical Features

- May be asymptomatic.
- Symptoms include poor feeding, failure to thrive, recurrent respiratory infections, and signs of congestive heart failure.
- Loud, harsh holosystolic murmur heard best at the lower left sternal border.

Diagnosis

- **1.Physical Examination**: Murmur, signs of heart failure.
- **2.Electrocardiogram (ECG)**: May show signs of left ventricular hypertrophy or right axis deviation.

- **3.Chest X-ray**: Enlarged heart, pulmonary congestion.
- **4.Echocardiography**: Confirms the diagnosis, determines size and location of the defect, assesses for associated anomalies.

Management

- Medical Management: Diuretics, afterload reducers (ACE inhibitors), digoxin for heart failure management.
- Surgical Repair: Indicated for large defects or failure of medical therapy.
- **Transcatheter Closure**: An alternative to surgery for selected patients with suitable anatomy.
- **Prophylactic Antibiotics**: To prevent infective endocarditis before surgical repair.

Prognosis

- Prognosis depends on the size of the defect and the presence of associated complications.
- Surgical closure typically results in excellent long-term outcomes.

Complications

- Pulmonary hypertension
- Eisenmenger syndrome (reversed shunt leading to cyanosis)
- Infective endocarditis
- Heart failure

Follow-Up

- Regular follow-up to monitor growth, development, and cardiac function.
- Assessment for potential complications.

Atrial Septal Defect (ASD)

Definition

Atrial Septal Defect (ASD) is a congenital heart defect characterized by an abnormal opening in the atrial septum, allowing blood to flow directly between the atria.

Classification

- Secundum ASD: Most common type, located in the fossa ovalis of the atrial septum.
- **Primum ASD**: Located near the atrioventricular valves, associated with cleft mitral valve and left axis deviation on ECG.
- Sinus venosus ASD: Near the superior or inferior vena cava, associated with anomalous pulmonary venous return.
- **Coronary sinus ASD**: Rare, associated with persistent left superior vena cava.

Pathophysiology

- Left-to-right shunt: Blood flows from the left atrium to the right atrium, causing volume overload of the right heart chambers.
- **Pulmonary overcirculation**: Increased blood flow to the lungs can lead to pulmonary hypertension and right ventricular hypertrophy over time.

Clinical Presentation

- Asymptomatic: Often diagnosed incidentally during evaluation for other conditions.
- **Symptomatic**: Variable presentation depending on the size of the defect and age of the patient.
 - o Fatigue
 - o Dyspnea on exertion
 - o Palpitations
 - Recurrent respiratory infections (especially in children)
 - Stroke (in older adults)

Diagnosis

- Echocardiography: Gold standard for diagnosis, identifies the size, location, and hemodynamic significance of ASD.
- Electrocardiogram (ECG): Typically normal in small ASDs, may show right atrial enlargement and incomplete right bundle branch block in larger defects.
- **Chest X-ray**: Enlarged right heart chambers, increased pulmonary vascular markings in significant shunts.

Management

- **Observation**: Small ASDs without significant shunting may require only regular follow-up.
- **Surgical Repair**: Indicated for symptomatic patients, large defects, or those with evidence of right heart enlargement or pulmonary hypertension.
 - **Transcatheter closure**: Minimally invasive option for selected patients with suitable anatomy.
- Antibiotic Prophylaxis: Recommended for patients with unrepaired ASD prior to certain dental or surgical procedures to prevent infective endocarditis.
- Follow-up: Regular monitoring of symptoms, echocardiography, and cardiac function.

Complications

- **Pulmonary hypertension**: Progressive shunting can lead to irreversible pulmonary vascular changes.
- Arrhythmias: Atrial fibrillation, atrial flutter, and supraventricular tachycardia.
- **Paradoxical emboli**: Thrombi originating in the venous circulation can pass through the ASD to the systemic circulation, causing stroke or systemic embolization.

Patent Ductus Arteriosus (PDA)

Definition

Persistence of the ductus arteriosus after birth.

Pathophysiology

Failure of the ductus arteriosus to close results in left-to-right shunting of blood, leading to pulmonary overcirculation.

Clinical Features

- Continuous machinery-like murmur heard best at the left upper sternal border.
- $_{\circ}~$ Wide pulse pressure.
- Signs of left heart volume overload (e.g., bounding peripheral pulses, widened pulse pressure).
- May lead to failure to thrive, recurrent respiratory infections, and heart failure if untreated.

Diagnosis

- Echocardiography is the gold standard.
- Chest X-ray may show pulmonary overcirculation.

Management

- Medical: Indomethacin or ibuprofen to close the ductus in preterm infants.
- Surgical: Ligation or transcatheter closure in symptomatic cases.

Complications

- Pulmonary hypertension.
- Infective endarteritis.

Prognosis

Excellent with timely closure; untreated PDA can lead to significant morbidity and mortality.

Coarctation of the Aorta

Definition

Narrowing of the aorta, typically near the ductus arteriosus insertion point.

Epidemiology

More common in males, often associated with other congenital heart defects.

Pathophysiology

Increased left ventricular pressure due to obstruction, leading to hypertension proximal to the coarctation and hypotension distally.

Clinical Features

- Hypertension in upper extremities
- Weak or absent femoral pulses
- Blood pressure discrepancy between upper and lower extremities

Diagnostic Approach

- Echocardiography: Initial screening tool
- Cardiac catheterization: To assess severity and plan intervention
- MRI or CT angiography: Detailed evaluation of the aorta

Management

- Surgical repair: Often necessary, especially in infants
- Balloon angioplasty: Alternative in older children and adults
- Long-term follow-up: To monitor for hypertension, recurrent coarctation, and associated complications

Complications

- 。 Hypertension
- Aortic aneurysm/dissection
- Premature coronary artery disease

Prognosis

Good with timely intervention, but lifelong monitoring is necessary.



- Acute Pericarditis
- Pericardial Effusion
- Cardiac Tamponade
- Constrictive Pericarditis

Acute Pericarditis

Definition

Inflammation of the pericardium, the thin sac-like membrane surrounding the heart.

Etiology

- Viral infections (most common cause)
- o Autoimmune conditions
- Post-myocardial infarction
- o Idiopathic

Clinical Features

- $_{\circ}$ Sharp, pleuritic chest pain
- Often relieved by sitting up and leaning forward
- Pericardial friction rub (heard on auscultation)
- $_{\circ}$ Fever
- $_{\circ}$ Dyspnea

Diagnostic Workup

- Electrocardiogram (ECG): Diffuse STsegment elevation
- Echocardiography: May reveal pericardial effusion
- Elevated inflammatory markers (e.g., CRP, ESR)

Management

- Symptomatic relief with NSAIDs (e.g., ibuprofen, indomethacin)
- Colchicine for recurrent cases or in combination with NSAIDs
- Corticosteroids for refractory cases or autoimmune etiology
- Treat underlying cause if identified

Complications

- Pericardial effusion
- Cardiac tamponade
- Constrictive pericarditis (chronic)

Prognosis

Generally favorable with appropriate management; recurrence possible in some cases.

Key Points for Patient Education

- Importance of follow-up to monitor for complications
- Avoidance of strenuous activities during acute phase
- Compliance with prescribed medications
- Awareness of symptoms warranting immediate medical attention (e.g., worsening chest pain, shortness of breath)

Pericardial Effusion

Definition

Accumulation of fluid in the pericardial sac surrounding the heart.

Can range from asymptomatic to lifethreatening, depending on the volume and rate of accumulation.

Causes

Infectious: Viral, bacterial, fungal pericarditis.

Inflammatory: Autoimmune diseases like rheumatoid arthritis, lupus.

Neoplastic: Metastatic cancer, primary pericardial tumors.

Traumatic: Blunt or penetrating chest trauma.

latrogenic: Complication of cardiac surgery or invasive procedures.

Idiopathic: No identifiable cause.

Clinical Features

- Chest pain: Typically sharp and pleuritic, can radiate to the back or neck.
- Dyspnea: Due to compression of cardiac chambers or impaired diastolic filling.
- Pericardial friction rub: Scratching or grating sound heard on auscultation.
- Beck's triad (in severe cases): Hypotension, muffled heart sounds, elevated jugular venous pressure.
- Signs of tamponade: Pulsus paradoxus, tachycardia, distant heart sounds.

Investigations

• ECG: Nonspecific changes (e.g., low-voltage QRS complexes, electrical alternans).

- diagnosis, assesses fluid volume and hemodynamic impact.
- Chest X-ray: May show an enlarged cardiac silhouette or water bottle-shaped heart.
- CT or MRI: Useful for assessing the extent and etiology of effusion.

Management

- **Conservative:** Observation with serial echocardiograms for small, asymptomatic effusions.
- **Pericardiocentesis:** Removal of fluid for diagnostic or therapeutic purposes.
- **Pericardial window:** Surgical creation of a permanent opening in the pericardium.
- **Treatment of underlying cause:** Antibiotics for infections, anti-inflammatories for autoimmune diseases, etc.

Complications

- Cardiac tamponade: Life-threatening compression of cardiac chambers by the fluid.
- Constrictive pericarditis: Fibrotic thickening of the pericardium, impairing cardiac function.
- Recurrence: Effusion may reaccumulate, especially if the underlying cause is not treated.

Prognosis

- Depends on the underlying cause, size of effusion, and promptness of intervention.
- Small effusions with benign etiologies generally have a favorable prognosis.
- Tamponade and constrictive pericarditis carry higher morbidity and mortality rates.
- Echocardiography: Gold standard for

Cardiac Tamponade

Definition

Compression of the heart due to accumulation of fluid in the pericardial sac, leading to impaired cardiac function.

Diagnosis

- ECG: Low voltage QRS complexes
- Echocardiography: Gold standard

Etiology

- 。Trauma
- Pericarditis
- Malignancy
- Aortic dissection

Treatment

- Pericardiocentesis
- Surgical pericardial window
- Inotropic support
- Volume resuscitation

Clinical Features

- Beck's triad: Hypotension, distended neck veins, muffled heart sounds
- Pulsus paradoxus
- $_{\circ}$ Dyspnea
- Tachycardia

Complications

- Cardiogenic shock
- Death if untreated

Constrictive Pericarditis

Definition

Constrictive pericarditis is a condition characterized by scarring and thickening of the pericardium, leading to impaired diastolic filling of the heart.

Etiology

- Idiopathic
- Prior pericarditis (infectious or inflammatory)
- Radiation therapy
- Cardiac surgery
- Tuberculosis

Clinical Features

- Exertional dyspnea
- Fatigue
- Peripheral edema
- Ascites
- Hepatomegaly
- Elevated jugular venous pressure with prominent x and y descents

Diagnostic Tools

- Echocardiography: May reveal pericardial thickening and septal bounce.
- Cardiac MRI: Useful for assessing pericardial thickness and constriction.
- Cardiac catheterization: Shows equalization of diastolic pressures in all cardiac chambers.

Treatment

- Diuretics: To manage fluid overload.
- Pericardiectomy: Surgical removal of the pericardium is the definitive treatment, especially in symptomatic patients.
- Anti-inflammatory therapy: In cases of underlying inflammatory etiology.

Prognosis

- Favorable with timely diagnosis and surgical intervention.
- Untreated, it can lead to heart failure and significant morbidity and mortality.



- Primary Cardiac Tumors
- Metastatic Cardiac Tumors

Primary Cardiac Tumors

Definition

Tumors originating within the heart, either benign or malignant.

Incidence

Rare; <0.1% of all cardiac neoplasms.

Types

- Benign: Myxoma (most common), Fibroma, Lipoma.
- Malignant: Sarcoma (e.g., Angiosarcoma, Rhabdomyosarcoma), Lymphoma, Pericardial Mesothelioma.

Clinical Features

- Myxoma: Most common benign tumor. May present with obstructive symptoms (dyspnea, syncope) or embolic events.
- Sarcoma: Aggressive, often metastasizes. Presents with heart failure symptoms, arrhythmias, or constitutional symptoms.

Diagnostic Evaluation

- Imaging: Echocardiography (TTE, TEE) for initial evaluation.
- Histopathology: Gold standard for diagnosis; obtained via biopsy or surgical resection.

Treatment

- Surgical Resection: Mainstay for symptomatic or potentially malignant tumors.
- **Chemotherapy/Radiation:** Adjuvant therapy for malignant tumors.
- **Heart Transplant:** Considered in select cases.

Prognosis

- **Benign Tumors:** Favorable prognosis after complete resection.
- Malignant Tumors: Poor prognosis due to metastatic potential and limited treatment options.

Considerations

- Metastasis: Especially common with sarcomas; commonly spreads to lungs.
- Multidisciplinary Approach: Involvement of cardiothoracic surgeons, oncologists, and pathologists for optimal management.

Follow-Up

- Regular surveillance imaging and clinical evaluation post-resection.
- Long-term monitoring for recurrence, especially with malignant tumors.

Metastatic Cardiac Tumors

Definition

Tumors originating from primary sites outside the heart that spread (metastasize) to the heart.

Prevalence

Relatively rare; often found incidentally during autopsies or diagnostic imaging.

Primary Sources

Common primary sites include lung, breast, melanoma, lymphoma, and esophagus.

Pathophysiology

Tumor cells metastasize via hematogenous or lymphatic routes to the heart, lodging primarily in the pericardium or myocardium.

Clinical Presentation

Often asymptomatic; symptoms, if present, depend on tumor size, location, and extent of infiltration. Symptoms may include dyspnea, chest pain, arrhythmias, or signs of heart failure.

Diagnostic Evaluation

Echocardiography, CT, MRI, or PET scans can reveal cardiac masses. Biopsy may be necessary for definitive diagnosis.

Management

Treatment focuses on relieving symptoms and managing complications. Surgical resection may be considered for solitary, accessible tumors. Chemotherapy, radiotherapy, or targeted therapies may be used in select cases.

Prognosis

Generally poor, with median survival ranging from months to a few years, depending on the primary tumor type, extent of metastasis, and response to treatment.

Complications

Potential complications include cardiac tamponade, arrhythmias, myocardial ischemia, and heart failure.

Follow-up

Regular imaging and clinical assessments are needed to monitor for disease progression or recurrence.

Multidisciplinary Approach

Management often involves collaboration among cardiologists, oncologists, radiologists, and surgeons for optimal patient care.



Vascular Diseases

- Aortic Aneurysm
- Aortic Dissection
- Peripheral Artery Disease
- Kawasaki Disease

Aortic Aneurysm

Definition

Abnormal dilation of the aorta, often asymptomatic but potentially life-threatening.

Types

- **Thoracic Aortic Aneurysm (TAA)**: Involves the part of the aorta that runs through the chest.
- Abdominal Aortic Aneurysm (AAA):
 Affects the portion of the aorta that passes through the abdomen.

Etiology

- **Atherosclerosis**: Major cause, particularly for AAA.
- Genetic factors: Family history increases risk.
- **Connective Tissue Disorders**: e.g., Marfan syndrome, Ehlers-Danlos syndrome.
- **Trauma**: Can lead to aortic dissection or aneurysm formation.

Clinical Presentation

- Often asymptomatic until rupture or complications arise.
- Symptoms may include chest or abdominal pain, back pain, dyspnea, hoarseness, dysphagia, or syncope.

Diagnostic Workup

- **Imaging:** CT, MRI, or ultrasound for diagnosis and monitoring.
- Screening: Recommended for high-risk individuals, e.g., older adults with smoking history or family history.

Management

- **Medical:** Blood pressure control to prevent enlargement.
- Surgical: Repair indicated for aneurysms at risk of rupture based on size, growth rate, and symptoms.
- Endovascular Aneurysm Repair (EVAR): Minimally invasive alternative to open surgery for selected cases.

Complications

- Rupture: Catastrophic event with high mortality.
- **Aortic Dissection**: Tear in the aortic wall, leading to separation of layers.

Prognosis

- Early detection and management crucial for preventing rupture and improving outcomes.
- Mortality rates high for ruptured aneurysms, emphasizing the importance of surveillance and intervention.

Prevention

- Smoking cessation, blood pressure control, and regular monitoring for high-risk individuals.
- Screening programs in place for at-risk populations to detect aneurysms early.

Aortic Dissection

Definition

Tear in the inner layer of the aorta, allowing blood to flow between the layers and potentially causing life-threatening complications.

Types

Classified into Stanford Type A (involving the ascending aorta) and Type B (involving the descending aorta).

Risk Factors

- o Hypertension
- Connective tissue disorders (e.g., Marfan syndrome)
- Bicuspid aortic valve
- Aortic aneurysm
- o Cocaine use

Clinical Presentation

- Sudden, severe chest pain (often described as tearing or ripping)
- Pain may radiate to the back or abdomen
- Syncope
- Neurological deficits (in cases of involvement of the carotid arteries)
- Blood pressure discrepancy between arms

Diagnosis

- CT angiography (gold standard)
- Transesophageal echocardiography (for hemodynamically unstable patients)
- o MRI

Management

- Emergent surgical intervention for Type A dissections
- Medical management for Type B dissections:
 - Blood pressure control with betablockers and vasodilators
 - Pain management
 - Serial imaging to monitor for progression

Complications

- Aortic rupture leading to exsanguination
- Cardiac tamponade
- Aortic valve insufficiency
- o Stroke or other neurological deficits
- Mesenteric ischemia or renal failure due to branch vessel involvement

Prognosis

Mortality rate remains high despite advances in management, particularly for Type A dissections. Early diagnosis and intervention are crucial for improved outcomes.

Peripheral Artery Disease (PAD)

Definition

Atherosclerotic narrowing of arteries supplying the limbs, leading to reduced blood flow.

Epidemiology

Common in older adults, smokers, and those with diabetes or hypertension.

Clinical Presentation

- Intermittent claudication: Pain in calf or thigh during physical activity, relieved by rest.
- Ischemic rest pain: Pain at rest due to severe arterial insufficiency.
- Non-healing wounds or ulcers, especially in lower extremities.

Diagnostic Evaluation

- Ankle-Brachial Index (ABI): Ratio of ankle to brachial systolic blood pressure.
- Doppler ultrasound: To assess blood flow and detect stenosis or occlusion.
- Angiography: To visualize arterial blockages.

Management

- Lifestyle modifications: Smoking cessation, exercise, and healthy diet.
- Pharmacotherapy: Antiplatelet agents, statins, and cilostazol.
- Revascularization: Angioplasty with or without stenting, or surgical bypass.

Complications

- Critical limb ischemia: Severe PAD with rest pain, ulcers, or gangrene.
- Risk of cardiovascular events:
 PAD is a marker of systemic atherosclerosis.

Prognosis

Associated with increased mortality and morbidity, particularly from cardiovascular events.

Prevention

Control of risk factors such as smoking, diabetes, and hypertension can mitigate progression and complications.

Kawasaki Disease

Definition

A rare but serious condition primarily affecting children, characterized by inflammation of the blood vessels throughout the body.

Epidemiology

- $\circ~$ Peak incidence in children under 5 years old.
- $\circ~$ More common in boys than girls.
- $\circ~$ Higher prevalence in Asian populations.

Etiology

- Exact cause unknown, possibly viral or bacterial.
- Immunological response implicated.

Clinical Features

- Fever lasting more than 5 days (often high and unresponsive to antibiotics).
- o Bilateral non-exudative conjunctivitis.
- Mucous membrane changes (red lips, strawberry tongue).
- Rash (polymorphous, non-vesicular).
- $\circ~$ Swelling and erythema of the hands and feet.
- \circ Cervical lymphadenopathy.

Complications

- $\circ~$ Coronary artery abnormalities (most serious).
- o Myocarditis.
- Pericarditis.
- o Arrhythmias.
- o Heart failure.

Diagnostic Criteria

- Clinical criteria supplemented by laboratory and imaging findings.
- o No definitive diagnostic test.
- Exclude other possible causes of similar symptoms.

Treatment

- Intravenous immunoglobulin (IVIG) and aspirin therapy.
- High-dose aspirin initially for antiinflammatory effect, followed by low-dose aspirin for antiplatelet effect.
- Treatment ideally within 10 days of symptom onset to reduce risk of coronary artery complications.

Prognosis

- Prompt treatment reduces risk of coronary artery complications.
- Most children recover fully, but some may experience long-term cardiovascular effects.
- Risk of recurrence is low.

Follow-Up

- Regular monitoring of cardiac function and coronary artery status.
- Long-term management of any cardiac sequelae.

Prevention

- No specific vaccine or preventive measures.
- Awareness among healthcare providers for prompt recognition and treatment.

9 Other Heart Diseases

- Infective Endocarditis
- Rheumatic Heart Disease

Infective Endocarditis

Definition

Infective Endocarditis (IE) is an infection of the endocardial surface of the heart, including the valves and adjacent structures.

Etiology

Mainly caused by bacterial infections, most commonly Staphylococcus aureus and Streptococcus viridans. Other pathogens include fungi and less commonly, viruses.

Risk Factors

- $_{\odot}$ Pre-existing heart value abnormalities
- o History of previous endocarditis
- o Intravenous drug use
- o Prosthetic heart valves
- o Immunocompromised states

Clinical Features

- $_{\circ}$ Fever, chills, and malaise
- New or changed heart murmur
- Petechiae, Osler's nodes, Janeway lesions
- Splinter hemorrhages
- Embolic phenomena (e.g., stroke, pulmonary embolism)

Diagnostic Evaluation

- Blood cultures (at least 3 sets)
- Echocardiography (transthoracic or transesophageal) to visualize vegetations on valves or other endocardial structures

Treatment

- Empiric antibiotic therapy targeting likely pathogens
- Surgical intervention may be necessary for complications such as valve destruction, abscess formation, or persistent infection despite antibiotic therapy

Prognosis

- Mortality rates vary depending on factors such as the causative organism, the presence of complications, and the promptness of treatment
- Early diagnosis and appropriate management are crucial for improving outcomes

Prevention

- Antibiotic prophylaxis for high-risk procedures in susceptible individuals (e.g., dental procedures in patients with prosthetic valves or certain congenital heart defects)
- Good oral hygiene to reduce the risk of bacteremia from dental procedures

Complications

- $_{\odot}$ Valve destruction leading to heart failure
- Embolic events causing stroke or organ infarction
- Mycotic aneurysms
- Septic emboli to other organs/systems

Rheumatic Heart Disease

Definition

Chronic condition resulting from rheumatic fever, leading to damage of heart valves and myocardium.

Etiology

Caused by untreated or inadequately treated group A Streptococcus infection.

Pathophysiology

Autoimmune response to streptococcal antigens leads to inflammation, scarring, and fibrosis of heart valves and myocardium.

Clinical Features

- Polyarthritis: Affects large joints, migratory pattern.
- Carditis: Inflammation of heart muscle and valves, causing murmurs, heart failure symptoms.
- Chorea: Involuntary movements, typically in children.
- Subcutaneous nodules: Painful, small, firm nodules over bony prominences.
- Erythema marginatum: Pink rings with central clearing on trunk and limbs.

Diagnosis

- History and clinical examination.
- ECG: May show conduction abnormalities.
- Echocardiography: To assess valve structure and function.
- Lab tests: Elevated inflammatory markers, antistreptolysin-O titers.

Management

- Antibiotics: Acute treatment with penicillin to eradicate streptococcal infection.
- Anti-inflammatory therapy: NSAIDs, corticosteroids to manage inflammation.
- Long-term prophylaxis: Penicillin or other antibiotics to prevent recurrent infections.
- Valve repair or replacement: For severe valve damage.
- **Symptomatic treatment:** Diuretics, betablockers, ACE inhibitors for heart failure symptoms.

Complications

- Valve stenosis or regurgitation.
- Infective endocarditis.
- Heart failure.
- Arrhythmias.
- Systemic embolism.
- Prevention
- Prompt treatment of streptococcal infections.
- Secondary antibiotic prophylaxis for at-risk individuals.
- Public health measures to improve access to healthcare and antibiotics in at-risk populations.

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