BLOCK O OSPE ist
19TH DECEMBER 2024

STATION 1

Chest X ray of a 6 year old who has has history of low grade fever and weight loss for last three months .

POST TUBERCULOUS PLEURAL EFFUSION

Q-1 what are the x ray findings?

Post-tuberculosis (TB) pleural effusion can lead to several radiological findings, which can be seen on chest X-ray (CXR), ultrasound, and CT scans. These findings depend on the stage of resolution and any complications, such as fibrosis or trapped lung.

Radiological Findings:

1. Chest X-ray (CXR):

Residual Pleural Thickening – Most common finding after TB pleural effusion, seen as a linear opacity along the pleural surface.

Blunted Costophrenic Angle – Due to pleural thickening or fibrosis.

Pleural Calcification – Seen in chronic cases.

Volume Loss – Shift of mediastinum or trachea toward the affected side due to fibrosis.

Trapped Lung Appearance – Hyperlucent lung field with a sharp pleural margin.

Q-2 what investigations will you do ?
Q-3what is the treatment?
Case: 6-Year-Old with History of Low-Grade Fever and Weight Loss for 3 Months – Post-Tuberculous Pleural Effusion

A child with chronic low-grade fever, weight loss, and post-tuberculous pleural effusion likely has residual changes from TB. Below are the relevant findings and management approach.

Q1: Chest X-ray (CXR) Findings in Post-Tuberculous Pleural Effusion

a) Pleural Findings:

Pleural Thickening – Smooth, uniform thickening, commonly at the costophrenic angle or lateral chest wall.

Blunted Costophrenic Angle – Due to fibrosis or residual effusion.

Pleural Calcifications – May be seen in chronic cases.

b) Lung and Mediastinal Findings:

Volume Loss & Mediastinal Shift – Trachea and mediastinum shift toward the affected side due to fibrosis.

Elevated Hemidiaphragm – Due to pleural fibrosis.

Trapped Lung Appearance – Hyperlucent lung with a sharp pleural border due to fibrous encasement.

Post-TB Parenchymal Changes – Fibrotic streaks, consolidation, or cavitations in upper lobes.

Q2: Investigations for Post-Tuberculous Pleural Effusion

Since the child has ongoing symptoms (fever, weight loss), additional workup is needed to rule out active TB or complications.

1. Imaging:

Chest X-ray (CXR) – To assess pleural thickening, lung volume loss, and mediastinal shift.

Chest Ultrasound – To check for residual pleural fluid, fibrin strands, or loculations.

Chest CT Scan – If needed, to assess pleural fibrosis, lung involvement, or persistent fluid collections.

2. Laboratory Tests:

Complete Blood Count (CBC) – Look for anemia, leukocytosis, or lymphocytosis.

Erythrocyte Sedimentation Rate (ESR) & C-Reactive Protein (CRP) – Indicators of inflammation.

Tuberculin Skin Test (TST) / Mantoux Test – To assess TB exposure.

Interferon-Gamma Release Assay (IGRA, e.g., QuantiFERON-TB Gold) – If available, helps confirm latent TB.

3. Pleural Fluid Analysis (if effusion is still present):

Diagnostic Thoracentesis – If pleural fluid is seen on imaging.

Appearance: Clear, straw-colored (in chronic cases) or cloudy (if secondary infection).

Protein >3.5 g/dL (Exudative Effusion, TB-specific).

Lymphocyte Predominance (>80%).

ADA (Adenosine Deaminase) >40 U/L – Suggestive of TB.

GeneXpert / PCR for TB – Highly specific for TB.

4. Sputum or Gastric Aspirate for TB Detection:

AFB (Acid-Fast Bacilli) Stain & Culture – To confirm TB if child can produce sputum.

GeneXpert MTB/RIF – Detects TB DNA and rifampicin resistance.

Gastric Lavage (for younger children) – If sputum is not available.

Q3: Treatment of Post-Tuberculous Pleural Effusion

1. Anti-Tuberculosis Therapy (ATT) – If Active or Latent TB is Suspected

Standard TB Treatment (2HRZE / 4HR):

Intensive Phase (2 months):

H – Isoniazid

R – Rifampicin

Z – Pyrazinamide

E – Ethambutol

Continuation Phase (4 months):

H – Isoniazid

R – Rifampicin

Total Duration: 6 months (may extend if complications like extensive fibrosis or empyema occur).

2. Adjunctive Therapy:

Corticosteroids (Prednisolone 1 mg/kg/day for 4 weeks, then taper) – If severe pleural thickening or persistent symptoms.

Nutritional Support – High-protein diet, vitamins (especially vitamin D).

Chest Physiotherapy & Breathing Exercises – To prevent lung restriction.

3. Management of Pleural Fibrosis or Trapped Lung:

If severe pleural fibrosis is causing lung restriction:

Decortication Surgery – In cases of trapped lung with significant respiratory compromise.

Pulmonary Rehabilitation – Breathing exercises to improve lung function.

Key Takeaways:

X-ray: Blunted costophrenic angle, pleural thickening, mediastinal shift toward the affected side.

Investigations: Chest X-ray, ultrasound, CT, pleural fluid analysis (if effusion is present), and TB tests (TST, GeneXpert, AFB).

Treatment: Standard anti-TB therapy (HRZE/HR for 6 months), steroids (if needed), and supportive care.

STATION 2

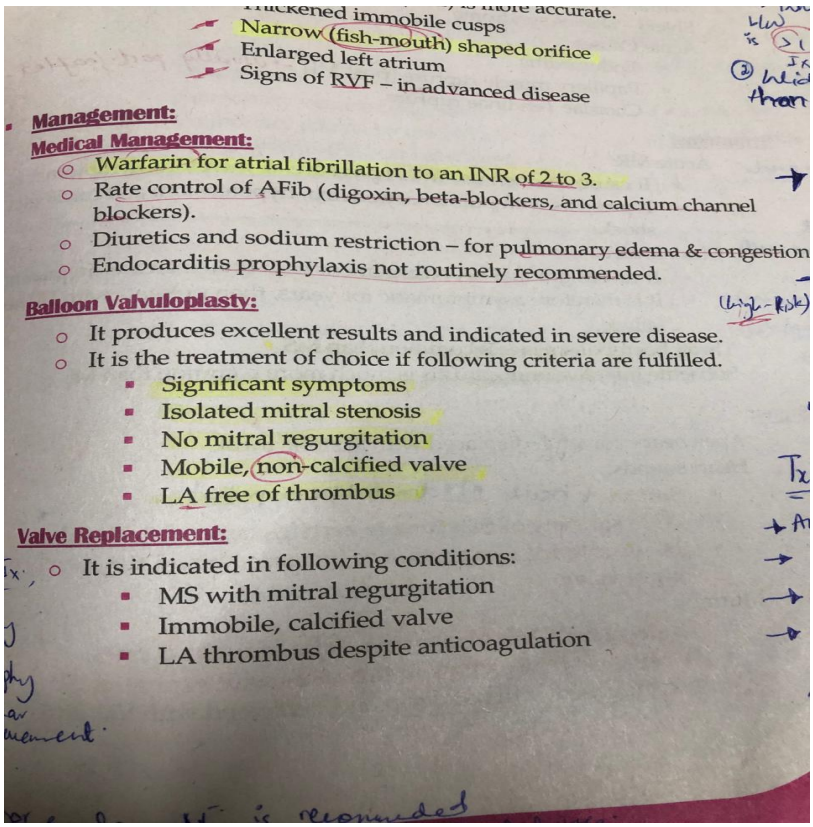
A dummy was present ,consider it a 1 year old child do chest examination only

STATION 3

INTERACTIVE STATION

Pulmonary artery wedge pressure = 30 mm hg
Left ventricular pressure = 5 mm hg

1) Diagnosis MITRAL STENOSIS
2) Treatment



STATION 4

SPLINTER HEMORRHAGE

1) Write two signs shown in picture
2) Write two causes

Splinter Hemorrhages – Causes

Splinter hemorrhages are small, red to brown linear streaks under the nails, caused by microvascular damage in the nail bed. They can be associated with local trauma or systemic diseases.

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1. Infectious Causes:

Infective Endocarditis – Most common systemic cause, due to septic emboli from heart valves.

Sepsis – Disseminated microvascular injury.

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2. Vascular and Hematologic Causes:

Small Vessel Vasculitis – E.g., Systemic Lupus Erythematosus (SLE), Polyarteritis Nodosa (PAN), or Rheumatoid Vasculitis.

Disseminated Intravascular Coagulation (DIC) – Microthrombi formation.

Antiphospholipid Syndrome (APS) – Hypercoagulability leads to embolic events.

Severe Anemia / Thrombocytopenia – Leads to fragile capillaries.

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3. Cardiovascular Causes:

Subacute Bacterial Endocarditis (SBE) – Due to septic emboli from heart valves.

Atrial Fibrillation – Can lead to embolic phenomena.

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4. Autoimmune & Inflammatory Causes:

SLE, Rheumatoid Arthritis (RA), Dermatomyositis – Associated with vasculitis and capillary fragility.

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5. Metabolic & Systemic Diseases:

Chronic Renal Failure / Dialysis – Due to uremic microvascular changes.

Diabetes Mellitus – Increased risk of small vessel disease.

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6. Trauma & Drug-Induced Causes:

Repeated Nail Trauma – Common in manual labor, sports, or nail biting.

Medications:

Anticoagulants (Warfarin, Heparin, DOACs) – Increased bleeding tendency.

Chemotherapy Agents (e.g., Taxanes) – Can cause microvascular injury.

Immunosuppressants (e.g., Cyclosporine, Steroids) – Affect capillary fragility.

Key Clinical Approach:

Single Nail Involvement → Likely trauma.

Multiple Nails, No Trauma → Consider systemic causes like endocarditis, vasculitis, or hematologic disorders.

Associated Fever & Cardiac Murmur → Strong suspicion for infective endocarditis.

Recurrent Episodes → Consider vasculitis, APS, or a hypercoagulable state.

STATION 5

Patient presented with sob and chest pain since last one hour . ECG shows ST elevation in lead 2,3 and aVF .

1) What is ur diagnosis?
2) How will u manage this patient ?

Management of Inferior Wall Myocardial Infarction (IWMI)
Inferior wall myocardial infarction (IWMI) involves the inferior portion of the heart, usually supplied by the right coronary artery (RCA) or sometimes the left circumflex artery (LCx). Management of IWMI differs slightly from anterior MI, especially regarding fluid management, heart block, and right ventricular involvement.

1. Immediate Management (Pre-Hospital & Emergency Department)

A. Initial Assessment & Stabilization
ABC approach (Airway, Breathing, Circulation)
ECG: Look for ST-elevation in leads II, III, aVF
Check right-sided ECG (V4R) to assess for right ventricular infarction
Assess for associated posterior MI (ST-depression in V1-V3, obtain posterior leads V7-V9)
B. First-Line Treatment (MONA-B)
Morphine: Only if pain is severe (avoid in hypotension)
Oxygen: Only if $SpO_2 < 90\%$
Nitroglycerin: Use with caution in RV infarction (can cause severe hypotension)
Aspirin: 325 mg chewable, plus P2Y12 inhibitor (Clopidogrel 300-600 mg or Ticagrelor 180 mg)
Beta-blockers: Avoid if hypotension, bradycardia, or heart block
2. Reperfusion Strategy

Primary PCI: Preferred if available within 90 minutes
Thrombolysis (e.g., Tenecteplase, Alteplase) if PCI is unavailable within 120

minutes and no contraindications
3. Special Considerations in IWMI

A. Right Ventricular Infarction (RVI)
Seen in 30-50% of IWMI
Signs: Hypotension, clear lungs, raised JVP
Management:
IV Fluids (to maintain preload)
Avoid nitrates, diuretics, and excessive opioids
Inotropes (e.g., Dobutamine, Dopamine) if persistent hypotension
B. Bradyarrhythmias & AV Blocks
Common in IWMI (due to RCA supplying AV node)
Sinus bradycardia & 1st-degree AV block: Usually benign, no intervention needed
2nd-degree Type I (Wenckebach): Observe; atropine if symptomatic
2nd-degree Type II or 3rd-degree AV block: Urgent pacing if symptomatic
4. Post-Reperfusion Management

Dual antiplatelet therapy (DAPT): Aspirin + P2Y12 inhibitor for at least 1 year
High-intensity statin (Atorvastatin 40-80 mg)
Beta-blockers (if no contraindications)
ACE inhibitors (if LV dysfunction or diabetes)
Lifestyle modifications & risk factor control
5. Complications to Watch For

RV infarction → Hypotension, cardiogenic shock
Papillary muscle rupture → Acute mitral regurgitation, pulmonary edema
Ventricular septal rupture → New systolic murmur, cardiogenic shock
Pericarditis → Pleuritic chest pain, pericardial rub
Arrhythmias → VT/VF, heart blocks
Summary
Early reperfusion (PCI or thrombolysis) is key
IV fluids instead of nitrates in RV infarction
Monitor and treat bradyarrhythmias
Long-term management includes DAPT, beta-blockers, statins, and lifestyle changes

STATION 6

ECG of ACUTE PERICARDITIS
1) Write diagnosis
2) What will be the management ?
3) Write investigations?

ECG in Acute Pericarditis

Acute pericarditis classically presents with a 4-stage ECG evolution:

1. Stage 1 (Hours to Days):

Diffuse ST elevation (concave, “saddle-shaped”) in multiple leads (except aVR, V1).

PR depression (best seen in lead II, V5, V6).

No reciprocal ST depression (unlike myocardial infarction).

2. Stage 2 (Days to 1 Week):

Normalization of ST segments.

PR depression may persist.

3. Stage 3 (1 to 3 Weeks):

T-wave inversion (not always present).

4. Stage 4 (Weeks to Months):

ECG returns to normal.

Diagnosis:

✅ Acute Pericarditis based on:

Chest pain (pleuritic, sharp, worse on lying down, relieved by sitting up).

Pericardial friction rub (best heard at the left lower sternal border).

ECG findings (diffuse ST elevation, PR depression).

Supporting findings: Pericardial effusion on echocardiography.

Management of Acute Pericarditis:

1. First-Line Treatment:

NSAIDs (e.g., Ibuprofen 600–800 mg TID for 1–2 weeks) – Reduces inflammation.

Colchicine (0.5–1 mg/day for 3 months) – Lowers recurrence risk.

Gastric Protection (e.g., PPI with NSAIDs) – To prevent gastritis.

2. If Symptoms Persist or Severe Cases:

Corticosteroids (Prednisolone 0.5 mg/kg/day, tapered over weeks) – Only if NSAIDs fail or contraindicated.

Pericardiocentesis – If large pericardial effusion or tamponade.

3. Treat Underlying Cause:

If Tuberculous Pericarditis → Anti-TB Therapy.

If Post-MI (Dressler's Syndrome) → Avoid NSAIDs early, use aspirin.

If Autoimmune Pericarditis → Consider steroids & immunosuppressants.

Investigations for Acute Pericarditis:

1. Blood Tests:

CBC – Leukocytosis (infection, inflammation).

ESR & CRP – Elevated in inflammation.

Troponin I/T – Normal or mildly elevated (rule out myocarditis).

Blood Cultures – If infectious cause suspected (e.g., sepsis, endocarditis).

2. Cardiac Imaging:

Echocardiography – Checks for pericardial effusion or tamponade.

Chest X-ray – May show an enlarged cardiac silhouette (if effusion is large).

Cardiac MRI/CT – If persistent or complicated cases.

3. Other Specific Tests (If Needed):

ANA, Rheumatoid Factor – If autoimmune etiology suspected.

TB Testing (Mantoux, IGRA, GeneXpert, Pericardial Fluid Analysis) – If TB pericarditis is suspected.

Viral Serology (Coxsackie, HIV, etc.) – If viral pericarditis is suspected.

STATION 7

X-ray of PLEURAL EFFUSION

1) Diagnosis
2) Chest radiograph findings

Pleural Effusion: Radiographic Findings & Treatment

I. Radiographic Findings
A. Chest X-ray (CXR) Findings
Upright CXR:
Blunting of costophrenic angle (earliest sign, seen with >200 mL of fluid)
Meniscus sign (concave upward fluid level)
Loss of diaphragmatic and cardiac borders (silhouette sign)
Mediastinal shift away from the effusion if large
Decubitus CXR:
Helps differentiate free-flowing vs. loculated effusion
Effusion layers along the dependent hemithorax
If fluid does not shift, suspect loculated effusion (empyema, malignancy)
Supine CXR:
Common in ICU patients; fluid spreads along the posterior chest
Diffuse haziness or veil-like opacification
May mimic lung consolidation
B. Ultrasound Findings
Anechoic or hypoechoic fluid collection
Septations → Suggests loculated effusion (empyema or malignancy)

Dynamic movement of lung with respiration (lung flapping sign)
C. CT Chest Findings
Best modality to detect small effusions (<10 mL)
Loculated vs. free-flowing effusion
Thickened pleura or pleural nodules → Suggests malignancy
Enhancing pleura ("split pleura sign") → Suggests empyema
II. Treatment of Pleural Effusion

A. Management Based on Underlying Cause
1. Transudative Effusion (Systemic Cause)

Due to increased hydrostatic pressure or low oncotic pressure
Common causes: Heart failure (most common), cirrhosis, nephrotic syndrome
Treatment:
Diuretics & sodium restriction for heart failure
Albumin infusion in hypoalbuminemia
Therapeutic thoracentesis if symptomatic
2. Exudative Effusion (Local Disease)

Due to inflammation, infection, malignancy, trauma
Causes: Pneumonia (parapneumonic effusion, empyema), TB, malignancy, PE
Treatment:
Parapneumonic effusion: Antibiotics, thoracentesis
Empyema (pus or pH < 7.2): Chest tube drainage ± fibrinolytics
Malignant effusion: Serial thoracentesis, pleurodesis (talc, doxycycline), or indwelling pleural catheter
TB effusion: Anti-TB therapy
B. Thoracentesis Indications
Symptomatic relief (dyspnea, hypoxia)
To determine cause (exudate vs. transudate – Light's criteria)
Recurrent effusions (palliative or definitive management)

STATION 8

Interactive station on MITRAL REGURGITATION
Medical Management of Mitral Regurgitation (MR)

Mitral regurgitation (MR) is the backflow of blood from the left ventricle (LV) into the left atrium (LA) due to improper mitral valve closure. The medical approach depends on whether it is acute or chronic, and whether it is primary (valve problem) or secondary (ventricular dysfunction).

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1. Medical Management of Acute Mitral Regurgitation

(A medical emergency, usually due to papillary muscle rupture, chordae tendineae rupture, or infective endocarditis.)

Goals: Reduce Afterload & Stabilize the Patient

IV Vasodilators (Nitroprusside, Nicardipine) – Reduce afterload and improve forward flow.

Diuretics (Furosemide) – Decrease pulmonary congestion.

Inotropes (Dobutamine, Milrinone) – If the patient has low cardiac output.

Intra-Aortic Balloon Pump (IABP) – Temporarily reduces afterload before surgery.

Definitive Treatment: Urgent Valve Surgery (Repair or Replacement).

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2. Medical Management of Chronic Mitral Regurgitation

(Most cases are degenerative, ischemic, or due to dilated cardiomyopathy.)

A) Asymptomatic or Mild MR:

No specific treatment needed if LV function is preserved.

Monitor with Echocardiography (every 6–12 months if moderate, every 2–3 years if mild).

Blood Pressure Control: ACE inhibitors, ARBs (for afterload reduction).

B) Symptomatic or Moderate-to-Severe MR (If Surgery Is Not Yet Indicated)

Afterload Reduction:

ACE Inhibitors / ARBs (Lisinopril, Losartan) – Reduce LV volume overload.

Hydralazine + Nitrates – Alternative if ACE inhibitors are not tolerated.

Rate Control & Rhythm Management (If Atrial Fibrillation Exists):

Beta-Blockers (Carvedilol, Metoprolol) – Reduce heart rate and improve LV filling.

Anticoagulation (Warfarin, DOACs) – If AF is present to prevent thromboembolism.

Diuretics (Furosemide, Spironolactone):

Helps relieve pulmonary congestion & volume overload.

SGLT2 Inhibitors (Dapagliflozin, Empagliflozin):

If MR is secondary to heart failure with reduced ejection fraction (HFrEF).

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3. When is Surgery Indicated?

Severe MR with Symptoms (NYHA Class II-IV).

LV Dysfunction (EF < 60% or LVESD > 40 mm).

Severe MR with Pulmonary Hypertension or Atrial Fibrillation.

Primary MR: Prefer valve repair over replacement when feasible.

Secondary MR: Consider mitral valve repair or transcatheter edge-to-edge repair (TEER, e.g., MitraClip).

STATION 9

LOGBOOK MEDICINE

STATION 10

Examine the precordium

STATION 11

DO RESPIRATORY EXAMINATION FROM THE BACK.

STATION 12

INTERACTIVE STATION ABOUT PNEUMOTHORAX

STATION 13

CALCULATE JVP

STATION 14

XRAY OF CCF
CT ratio was increased and there was pleural effusion

1) What are two x ray findings
2) What are two investigations

Congestive Cardiac Failure (CCF) – X-ray Findings, Investigations, and Management

I. Chest X-ray (CXR) Findings in Congestive Cardiac Failure

A chest X-ray is an essential tool in diagnosing and assessing heart failure. Key findings include:

A. Pulmonary Congestion & Edema
Cardiomegaly → Cardiothoracic ratio (CTR) >50%
Pulmonary venous congestion (Cephalization of vessels) → Upper lobe veins become more prominent due to increased pressure
Kerley B lines → Short, horizontal peripheral lines at lung bases (indicating interstitial edema)
Bat-wing pattern (Alveolar edema) → Bilateral perihilar opacities, sparing the periphery
Pleural effusion → Blunting of costophrenic angles (often bilateral but may be asymmetric)
Peribronchial cuffing → Thickened bronchial walls due to fluid accumulation
Prominent hilum → Due to pulmonary venous congestion
B. Stages of Pulmonary Congestion on CXR (Pulmonary Edema Progression)
Stage 1 (Mild congestion) → Cephalization of pulmonary veins
Stage 2 (Interstitial edema) → Kerley B lines, peribronchial cuffing
Stage 3 (Alveolar edema - Pulmonary edema) → Bat-wing opacities, air bronchograms, pleural effusion
II. Investigations for Congestive Cardiac Failure

A. Bedside Investigations
Electrocardiogram (ECG) → May show LVH, ischemic changes, AF, or bundle branch blocks
Pulse oximetry & ABG → Assess for hypoxia & acidosis
B. Blood Tests
BNP (Brain Natriuretic Peptide) or NT-proBNP
BNP > 100 pg/mL or NT-proBNP > 300 pg/mL suggests heart failure
Higher values indicate severe failure

Complete blood count (CBC) → Rule out anemia or infection
Renal function tests (Urea, Creatinine, Electrolytes) → Assess for cardiorenal syndrome
Liver function tests (LFTs) → Evaluate congestive hepatopathy
Thyroid function tests (TFTs) → Rule out thyroid-related heart failure
Lipid profile, HbA1c → Assess cardiovascular risk factors
C. Imaging & Functional Tests
Echocardiography (ECHO) – Gold Standard
Ejection fraction (EF) assessment:
HFrEF (EF < 40%) → Systolic failure
HFpEF (EF ≥ 50%) → Diastolic failure
Chamber size, wall motion abnormalities, valve disease, pulmonary pressure
Chest X-ray (CXR) → Assess pulmonary congestion (findings above)
Cardiac MRI → Further structural and functional assessment
Coronary angiography → If ischemic heart disease (IHD) is suspected
III. Management of Congestive Cardiac Failure

A. Acute Decompensated Heart Failure (ADHF) – Emergency Management
Mnemonic: LMNOP

Loop diuretics (e.g., Furosemide IV) → Reduces pulmonary congestion
Morphine (if severe distress) → Decreases preload & anxiety
Nitrates (e.g., Nitroglycerin) → Reduces afterload/preload (avoid in hypotension!)
Oxygen (if hypoxic) or Non-invasive ventilation (CPAP/BiPAP)
Position (Sit patient upright) → Reduces preload
If hypotensive or in shock:

Inotropes (Dobutamine, Milrinone)
Vasopressors (Noradrenaline, Dopamine) if needed
B. Chronic Heart Failure Management
1. Lifestyle Modifications

Low-sodium diet (<2g/day)
Fluid restriction (1.5-2L/day)
Weight monitoring (daily weight changes can indicate fluid retention)
Regular exercise (if stable)
Smoking and alcohol cessation
2. Medications (For HFrEF, EF < 40%)

First-line drugs:
ACE inhibitors (e.g., Ramipril, Enalapril) OR ARBs (if intolerant)
Beta-blockers (e.g., Bisoprolol, Carvedilol, Metoprolol)
Mineralocorticoid receptor antagonists (MRA) – Spironolactone/Eplerenone
Additional therapies (if persistent symptoms)
SGLT2 inhibitors (Dapagliflozin, Empagliflozin)
ARNI (Sacubitril-Valsartan) instead of ACEI/ARB
Ivabradine (if HR > 70 despite beta-blockers)

Symptomatic Management
Diuretics (Loop ± Thiazide) for fluid overload
Digoxin (only in AF or persistent symptoms despite therapy)
3. Advanced Heart Failure Options

Cardiac Resynchronization Therapy (CRT) → For LBBB & low EF
Implantable Cardioverter Defibrillator (ICD) → If EF <35% to prevent sudden cardiac death
Heart transplant or LVAD → For end-stage heart failure
Summary
CXR findings → Cardiomegaly, pulmonary congestion, Kerley B lines, pleural effusion
Investigations → BNP, Echo, ECG, CXR, renal/liver function
Management → Acute: LMNOP + inotropes if needed; Chronic: ACEI/ARB, Beta-blockers, Diuretics, MRA, SGLT2i

STATION 15

SVT two stations

Supraventricular Tachycardia (SVT): ECG Findings & Management

1. ECG Findings in SVT

✅ Rate: 150–250 bpm (regular rhythm).
✅ Narrow QRS complexes (<120 ms) (unless aberrancy present).
✅ P waves may be hidden (buried in QRS) or seen as pseudo "S" wave in inferior leads or pseudo "R'" in V1.
✅ Regular RR intervals (helps differentiate from atrial fibrillation).
✅ No significant ST-segment elevation (to rule out STEMI).

Types of SVT Based on ECG:

1. Atrioventricular Nodal Reentrant Tachycardia (AVNRT) – Most common, no visible P waves.

2. Atrioventricular Reentrant Tachycardia (AVRT, e.g., WPW syndrome) – May have delta waves in sinus rhythm.

3. Atrial Tachycardia – Visible abnormal P waves, often warm-up and cool-down phases.

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2. Management of SVT

A) Hemodynamically Unstable SVT (Hypotension, Shock, Chest Pain, Syncope)

✅ Immediate Synchronized Cardioversion (50–100 J).

B) Hemodynamically Stable SVT

Step 1: Vagal Maneuvers (First-Line)

Valsalva maneuver – Bear down as if having a bowel movement.

Carotid sinus massage – Contraindicated in carotid stenosis.

Cold water immersion (Diving reflex).

Step 2: Adenosine (If Vagal Maneuvers Fail)

✅ Adenosine 6 mg IV push (Rapid bolus + flush)

If ineffective, repeat with 12 mg IV (can be given twice).

Temporary asystole may occur, followed by normal rhythm.

Step 3: AV Nodal Blocking Agents (If Adenosine Fails or Contraindicated)

Beta-Blockers (Metoprolol 5 mg IV, Esmolol IV infusion).

Calcium Channel Blockers (Verapamil 5 mg IV, Diltiazem 15–20 mg IV).

Step 4: Antiarrhythmic Drugs (If Refractory SVT or Structural Heart Disease Present)

Amiodarone (150 mg IV over 10 min, then infusion).

Procainamide (for WPW-related SVT, avoid AV nodal blockers).

C) Long-Term Management (Prevention of Recurrent SVT)

✅ Lifestyle Modification: Avoid caffeine, alcohol, and stress triggers.
✅ Medications:

Beta-blockers (Metoprolol, Propranolol) – First-line for prevention.

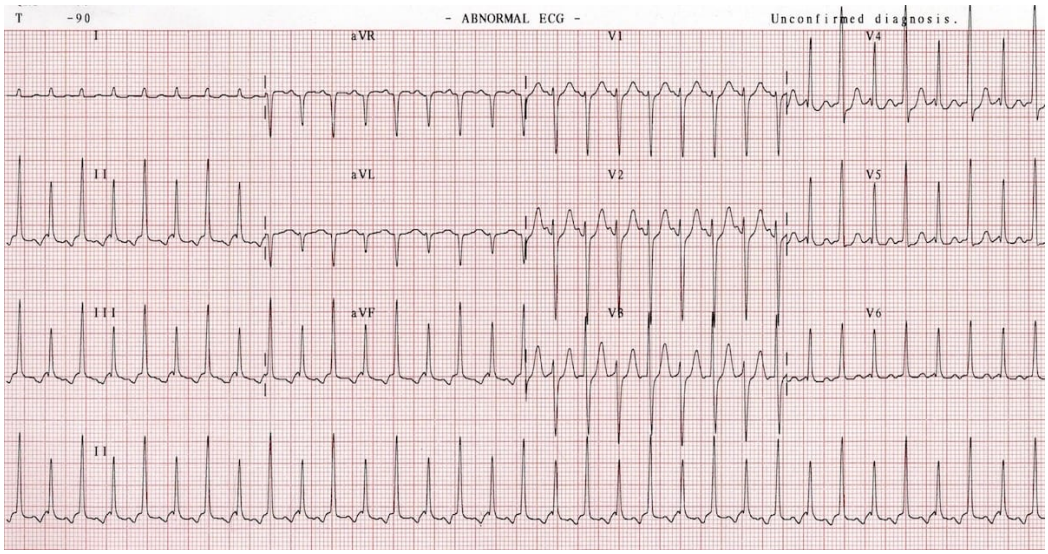
Calcium Channel Blockers (Diltiazem, Verapamil) – Alternative.
✅ Catheter Ablation (Definitive Treatment for Recurrent SVT).

Key Takeaways:

Stable SVT → Vagal maneuvers → Adenosine → BB/CCB → Antiarrhythmics.

Unstable SVT → Immediate synchronized cardioversion.

Recurrent SVT → Consider catheter ablation.



STATION 16
CROUP
Croup (Laryngotracheobronchitis) – Investigations & Management

I. Overview

Croup is a viral upper airway infection that causes subglottic inflammation, leading to barking cough, stridor, and respiratory distress. It is most common in children aged 6 months to 3 years and is usually caused by parainfluenza virus.

II. Investigations for Croup

Croup is primarily a clinical diagnosis, but investigations may be done in severe cases or to rule out other conditions.

A. Clinical Diagnosis
Classic symptoms:
Barking cough
Inspiratory stridor
Hoarseness
Respiratory distress (variable severity)
Westley Croup Severity Score (Used for severity grading):
Mild (≤2): Barking cough, no stridor at rest
Moderate (3-7): Stridor at rest, mild retractions, no agitation
Severe (≥8): Severe stridor, marked retractions, agitation, lethargy, cyanosis
B. Radiological Investigations (Only if Unclear Diagnosis)
Neck X-ray (AP view) – "Steeple Sign"
Subglottic narrowing of the trachea (resembles a church steeple)
Helps differentiate from bacterial tracheitis or epiglottitis
Chest X-ray → To rule out foreign body aspiration or pneumonia
C. Laboratory Tests (Not Routine)
Viral PCR (Nasopharyngeal swab) → Identifies causative virus (if needed for

epidemiology)
CBC & CRP → Rarely needed; may help differentiate from bacterial infections
III. Management of Croup

A. Mild Croup (No Stridor at Rest, No Distress)
Supportive care:
Keep child calm (agitation worsens airway obstruction)
Hydration, antipyretics (if fever), humidified air
Single dose of oral Dexamethasone (0.15–0.6 mg/kg) → Reduces airway swelling
B. Moderate to Severe Croup (Stridor at Rest, Respiratory Distress)
1. First-Line Treatment

Dexamethasone (0.6 mg/kg PO/IM/IV, max 10 mg) → Reduces inflammation
Nebulized Epinephrine (5 mL of 1:1000 or 0.5 mL/kg of 1:1000 in 2–3 mL saline)
Rapid improvement in airway swelling
Monitor for rebound worsening → Observe for 4–6 hours after administration
2. Supportive Measures

Oxygen (if SpO_2 < 92%)
Nebulized saline or humidified air (not strongly proven but sometimes used)
Avoid agitation (minimize handling, allow comfort measures)
3. Hospitalization Indications

Severe respiratory distress (persistent stridor, retractions, tachypnea)
Poor response to epinephrine or steroids
Hypoxia or altered mental status
4. Intubation Criteria (Rare, Severe Cases)

Impending respiratory failure: Exhaustion, cyanosis, altered mental status
Severe airway obstruction despite treatment
IV. Differential Diagnoses to Consider

Epiglottitis (Emergency!) → Drooling, toxic appearance, thumb sign on X-ray
Bacterial tracheitis → High fever, toxic appearance, poor response to epinephrine
Foreign body aspiration → Sudden onset, localized wheezing
Allergic reaction/anaphylaxis → Urticaria, rapid swelling
Summary
Diagnosis: Clinical, steeple sign on X-ray (if needed)
Mild cases: Oral dexamethasone + supportive care
Moderate-severe: Nebulized epinephrine + dexamethasone
Hospitalization: If persistent distress, hypoxia, or poor response

STATION 17
BRONCHIACTESIS

Bronchiectasis: Overview, Diagnosis, and Management

1. Definition:

Bronchiectasis is a chronic irreversible dilation of the bronchi due to airway inflammation, destruction, and mucus accumulation, leading to recurrent infections and airflow obstruction.

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2. Causes of Bronchiectasis:

✅ Infectious Causes:

Post-Tuberculosis (most common cause in developing countries).

Pneumonia (Bacterial, Viral, Fungal).

Cystic Fibrosis (CF) – Common in children.

Non-Tuberculous Mycobacteria (NTM) – e.g., Mycobacterium avium complex (MAC).

Primary Ciliary Dyskinesia (PCD) – e.g., Kartagener's Syndrome.

✅ Non-Infectious Causes:

Autoimmune Diseases (RA, SLE, Sjögren's Syndrome).

Aspiration (GERD, Recurrent Vomiting).

Allergic Bronchopulmonary Aspergillosis (ABPA).

Obstruction (Foreign body, Tumors, Lymphadenopathy).

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3. Clinical Features

✅ Symptoms:

Chronic productive cough with large amounts of purulent sputum.

Recurrent lung infections.

Hemoptysis (due to inflamed bronchial vessels).

Dyspnea & Wheezing.

Fatigue & Weight loss (if severe).

✅ Signs (on Physical Exam):

Crackles (coarse inspiratory) over affected areas.

Wheezing (if airflow obstruction).

Digital Clubbing (if chronic hypoxia).

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4. Diagnosis of Bronchiectasis

A. Imaging (Key Diagnostic Tool)

1. High-Resolution CT (HRCT) Scan (Gold Standard):

Signet Ring Sign: Dilated bronchi larger than adjacent pulmonary artery.

Tram-Track Sign: Parallel thickened airway walls.

Cystic Changes, Mucus Plugging.

2. Chest X-ray (Less Sensitive):

Dilated bronchi with thick walls (if advanced).

Increased lung markings & volume loss.

B. Laboratory & Functional Tests

Sputum Culture: Identify Pseudomonas aeruginosa, Haemophilus influenzae, Staphylococcus aureus, or Mycobacteria.

Spirometry (PFTs): Shows obstructive pattern (↓FEV1/FVC, ↑RV).

CF Testing (Sweat Chloride, Genetic Test) – If suspected in young patients.

Immunoglobulin Levels (IgG, IgA, IgM) – To check for immunodeficiency.

Allergy Tests (Aspergillus IgE, Skin Prick Test) – If ABPA suspected.

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5. Management of Bronchiectasis

✅ 1. Airway Clearance & Mucus Drainage

Chest Physiotherapy & Postural Drainage (daily).

Hypertonic Saline Nebulization (mucus thinning).

Bronchodilators (Salbutamol, Tiotropium) – If wheezing or airflow obstruction.

✅ 2. Antibiotic Therapy (For Infections & Exacerbations)

Mild Exacerbations: Amoxicillin-Clavulanate, Azithromycin, Doxycycline (7–14 days).

Severe or Pseudomonas Colonization: Ciprofloxacin (oral) or IV Piperacillin-Tazobactam, Meropenem.

Chronic Pseudomonas Infections: Inhaled Tobramycin or Aztreonam.

Macrolide Therapy (Azithromycin, Clarithromycin): Long-term anti-inflammatory effect.

✅ 3. Anti-Inflammatory & Supportive Therapy

Corticosteroids (Inhaled or Oral) – For ABPA or severe inflammation.

Oxygen Therapy – If chronic hypoxemia.

Pulmonary Rehabilitation & Nutrition Optimization.

✅ 4. Surgical & Advanced Treatment Options

Surgical Resection – If localized disease with frequent infections.

Lung Transplant – End-stage bronchiectasis.

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6. Prevention & Lifestyle Modifications

Vaccination (Influenza, Pneumococcal).

Smoking Cessation.

Early Treatment of Respiratory Infections.

Avoid Aspiration (GERD Control).

Key Takeaways:

HRCT is the gold standard for diagnosis (Signet Ring & Tram-Track Signs).

Airway clearance therapy is essential (Chest Physiotherapy, Nebulized Saline).

Antibiotics for exacerbations; inhaled macrolides for chronic infections.

Consider surgery or lung transplant in advanced cases.

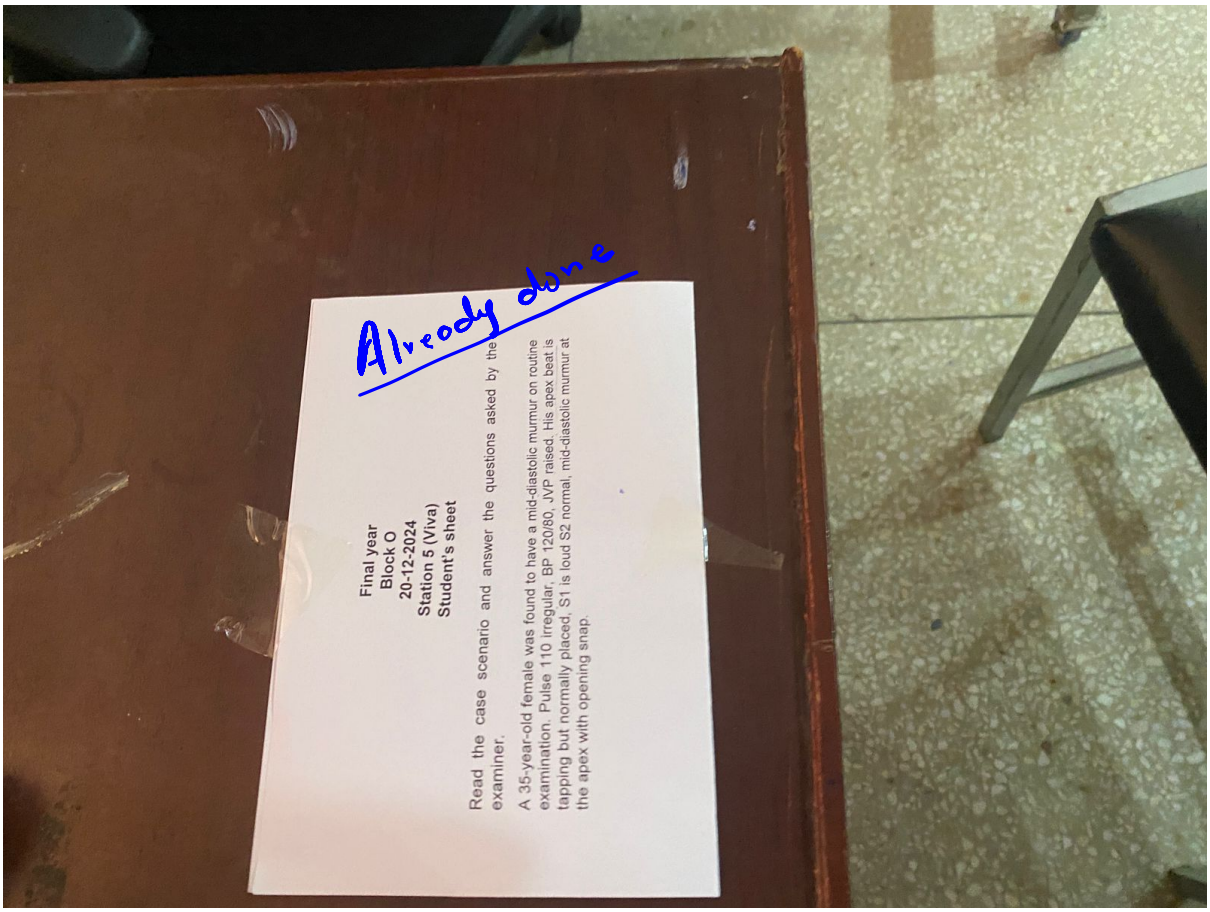
DAY 2
20TH DECEMBER 2024

STATION 1

STATION 2

INTERACTIVE STATION ABOUT MITRAL STENOSIS

1) DIAGNOSIS
2) MANAGEMENT PLAN



STATION 3
INTERACTIVE STATION ABOUT UNSTABLE ANGINA
1) DIAGNOSIS
2) MANAGEMENT PLANS

Unstable Angina (UA) Management Plan
Unstable angina is a medical emergency requiring rapid evaluation and management to prevent myocardial infarction (MI) or death. The approach includes initial stabilization, risk stratification, and definitive treatment.

1. Immediate Management (Emergency Department/Pre-hospital)

A. General Measures (MONA-B)
Morphine: Only if pain persists despite nitrates.
Oxygen: If hypoxic (SpO_2 <90%).
Nitrates: Sublingual GTN 0.3–0.6 mg every 5 min as needed, then IV if persistent pain.
Aspirin: 162–325 mg (chewable), unless contraindicated.
Beta-blocker (e.g., Metoprolol 25–50 mg PO): If no contraindications (e.g., hypotension, bradycardia, heart failure).
2. Risk Stratification (TIMI/GRACE Score)

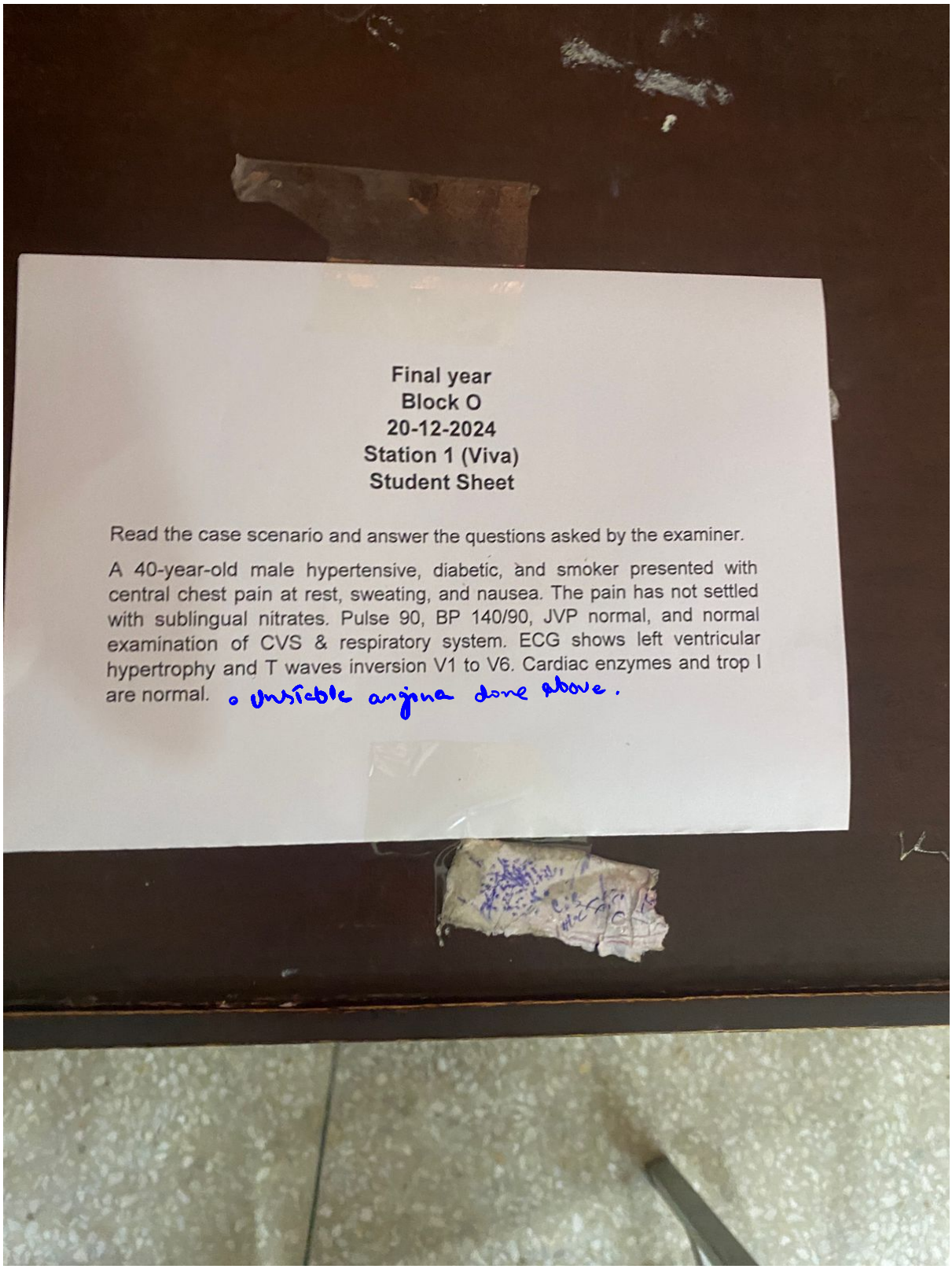
Determines the likelihood of adverse events (MI, death).

Higher scores → Early invasive strategy preferred.
3. Pharmacologic Management

A. Antiplatelet Therapy
Aspirin (loading 162–325 mg, then 81–162 mg daily lifelong).
$P2Y_{12}$ inhibitor (e.g., Clopidogrel 300–600 mg loading, then 75 mg/day OR Ticagrelor 180 mg loading, then 90 mg BID).
Glycoprotein IIb/IIIa inhibitors (e.g., Eptifibatide or Tirofiban) in high-risk patients undergoing PCI.
B. Anticoagulation
Unfractionated Heparin (UFH) (bolus 60–70 U/kg IV, then infusion 12–15 U/kg/hr) OR
Low Molecular Weight Heparin (LMWH, e.g., Enoxaparin 1 mg/kg SC BID)
Fondaparinux 2.5 mg SC daily (if high bleeding risk).
C. Additional Medications
Beta-Blockers (e.g., Metoprolol, Atenolol) unless contraindicated.
ACE Inhibitors/ARBs: Especially in patients with diabetes, heart failure, or hypertension.
Statins (High-Intensity, e.g., Atorvastatin 40–80 mg daily).
Aldosterone Antagonists (e.g., Spironolactone) in heart failure with reduced EF.
4. Early Invasive vs. Conservative Strategy

A. Invasive Strategy (PCI/CABG) – Preferred for:
Refractory angina despite medical therapy.
High-risk features (e.g., dynamic ECG changes, high TIMI/GRACE score).
Positive stress test findings.
B. Conservative Strategy
Stable patients with low-risk features.
Continue aggressive medical therapy.
5. Long-Term Secondary Prevention

Lifestyle changes: Smoking cessation, diet, exercise.
Medication adherence: Continue DAPT (aspirin + $P2Y_{12}$ inhibitor), beta-blockers, ACE inhibitors, and statins.
Regular follow-ups: Monitor for recurrent symptoms, compliance, and side effects.



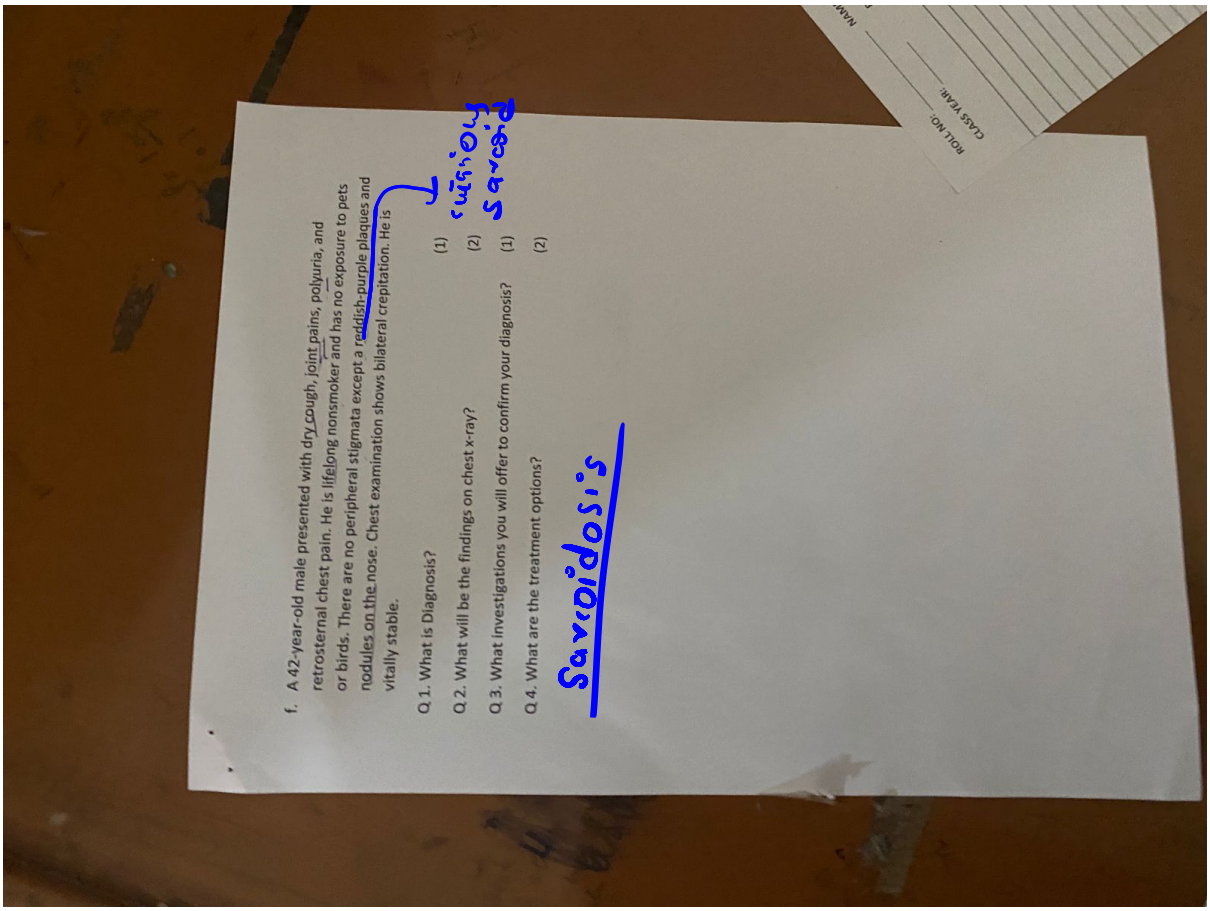
STATION 4
SARCOIDOSIS

Case Summary:

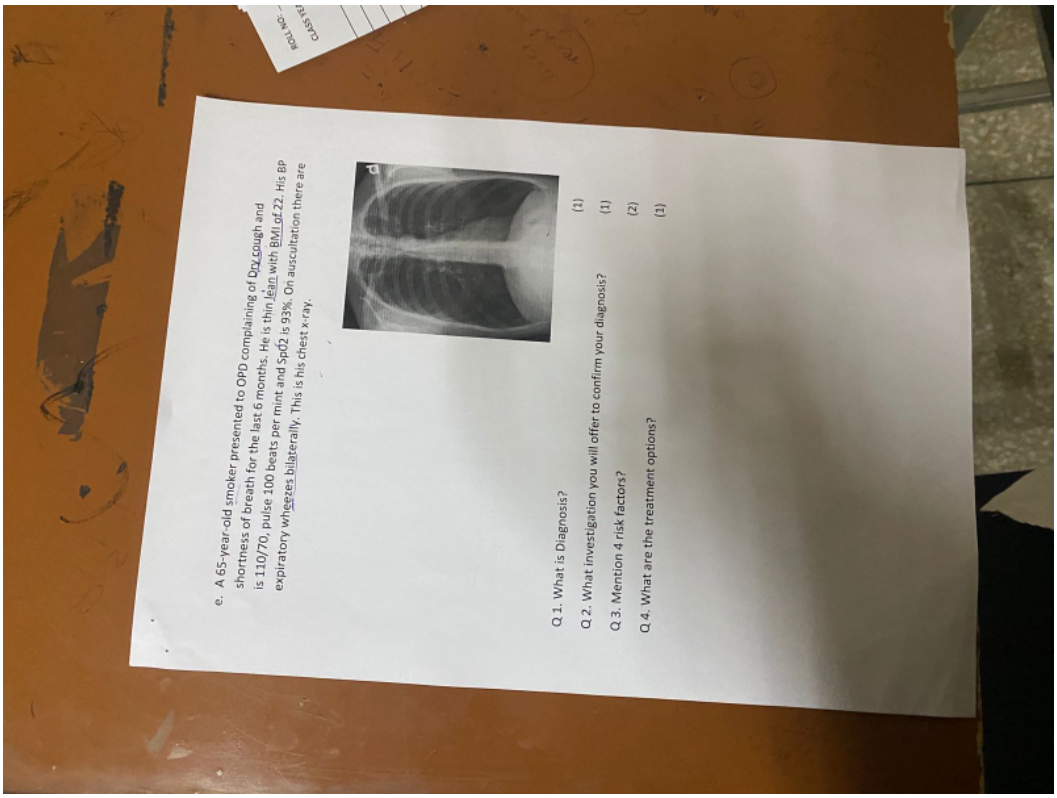
42-year-old male with dry cough, joint pains, polyuria, and retrosternal chest pain.
Non-smoker, no exposure to pets or birds.
Skin lesions: Reddish-purple plaques and nodules on the nose.
Chest examination: Bilateral crepitations.
Vitals: Stable.
Q1: What is the Diagnosis?
Likely Diagnosis: Sarcoidosis

Multisystem involvement: Pulmonary (cough, crepitations), musculoskeletal (joint pains), renal (polyuria), and dermatological (nodules, plaques).
Löfgren syndrome (acute sarcoidosis) may present with joint pain.
Erythema nodosum (tender, red nodules) is a common skin manifestation.
Noncaseating granulomas are characteristic.
Q2: What will be the Findings on Chest X-ray?
Bilateral hilar lymphadenopathy (BHL) – hallmark finding.
Reticulonodular infiltrates in lung fields (especially upper lobes in chronic disease).
Pulmonary fibrosis in advanced cases.
Q3: What Investigations Will You Offer to Confirm Your Diagnosis?
Blood tests:
Serum ACE (Angiotensin-Converting Enzyme) – elevated in sarcoidosis.
Calcium levels – hypercalcemia/hypercalciuria (due to increased vitamin D activity).
Renal function tests – assess nephrocalcinosis risk.
Imaging:
Chest X-ray – Bilateral hilar lymphadenopathy.
High-resolution CT (HRCT) chest – Parenchymal lung involvement (nodules, fibrosis).
Pulmonary function tests (PFTs):
Restrictive pattern (↓ FVC, normal/↑ FEV1/FVC ratio).
↓ DLCO (diffusion capacity).
Tissue biopsy (confirmatory test):
Noncaseating granulomas (bronchoscopy with transbronchial lung biopsy or skin biopsy from nasal lesions).
Bronchoalveolar lavage (BAL):
Increased CD4+/CD8+ T-cell ratio (>3.5).
Ophthalmologic evaluation (sarcoid uveitis).
ECG (if cardiac involvement suspected).
Q4: What are the Treatment Options?
Observation (if asymptomatic or mild disease)
Many cases resolve spontaneously.
Corticosteroids (First-line)
Prednisolone 20–40 mg/day for moderate/severe disease.
Taper over months to avoid recurrence.
Immunosuppressants (if steroids fail or for steroid-sparing)

Methotrexate, Azathioprine, or Mycophenolate.
Hydroxychloroquine – For skin and joint involvement.
Anti-TNF agents (Infliximab, Adalimumab)
For refractory cases.
Symptomatic treatment
NSAIDs for joint pain.
Calcium and vitamin D monitoring to prevent hypercalcemia.



STATION 5



Case Summary:
65-year-old smoker
Symptoms: Dry cough, progressive shortness of breath for 6 months
Physical findings: Thin/lean body, BMI = 22, BP = 110/70, HR = 100 bpm, SpO_2 = 93%
Auscultation: Bilateral expiratory wheezes
Chest X-ray: Shows hyperinflated lungs (suggestive of emphysema/COPD)
Q1: What is the Diagnosis?
Likely Diagnosis: Chronic Obstructive Pulmonary Disease (COPD)

Chronic progressive dyspnea, smoking history, wheezing, and hyperinflated lungs suggest COPD.
Two main phenotypes:
Emphysema-dominant (Pink Puffer) – Thin, breathless, pursed-lip breathing.
Chronic Bronchitis-dominant (Blue Bloater) – Chronic cough, mucus production, cyanosis.
Q2: What Investigations Will You Offer to Confirm the Diagnosis?
Pulmonary Function Test (PFT/Spirometry) – Gold Standard
$FEV_1/FVC < 70\%$ (Post-bronchodilator, confirms airflow limitation).
Severity staging based on FEV_1 (% predicted) (e.g., GOLD classification).
Chest X-ray (Already Provided)
Findings: Hyperinflation, flattened diaphragm, increased AP diameter, bullae.
Arterial Blood Gas (ABG) Analysis
To check for respiratory acidosis (↑ CO_2 in severe cases).
CT Chest (If needed)

Better visualization of emphysematous changes (bullae, fibrosis).
Alpha-1 Antitrypsin (AAT) Deficiency Screening
If young onset COPD (<45 years) or no smoking history.
Complete Blood Count (CBC) & ECG
Polycythemia (↑ RBC due to chronic hypoxia).
ECG for cor pulmonale (right heart strain).
Q3: Mention 4 Risk Factors for COPD
Smoking – Primary risk factor (>80% of cases).
Environmental Exposure – Air pollution, occupational dust/chemicals.
Genetic Factor – Alpha-1 Antitrypsin Deficiency.
Recurrent Respiratory Infections – In early life, predisposes to lung damage.
Q4: What are the Treatment Options?
A. Non-Pharmacological Management

Smoking Cessation – Most effective intervention.
Pulmonary Rehabilitation – Breathing exercises, physical activity.
Vaccination – Annual influenza + pneumococcal vaccine to prevent exacerbations.
B. Pharmacological Therapy (Based on Severity)

Short-Acting Bronchodilators (SABA/SAMA) – Mild COPD
Salbutamol (SABA) or Ipratropium (SAMA) for symptom relief.
Long-Acting Bronchodilators (LABA/LAMA) – Moderate to Severe
Tiotropium (LAMA) or Salmeterol/Formoterol (LABA).
Inhaled Corticosteroids (ICS) – Severe COPD with Frequent Exacerbations
ICS + LABA (e.g., Budesonide/Formoterol).
Oral Theophylline – Used in refractory cases.
C. Oxygen Therapy

Long-Term Oxygen Therapy (LTOT) – If chronic hypoxemia (PaO_2 <55 mmHg or SpO_2 <88%).
D. Advanced Therapy (Severe Cases)

Lung Volume Reduction Surgery (LVRS) – For emphysematous COPD.
Lung Transplantation – For end-stage disease.

STATION 6

EXAMINE THE RESPIRATORY SYSTEM OF A ONE YEAR OLD
DUMMY EXAMINATION

STATION 7

EXAMINE THE PRECORDIUM OF A 2 YEAR OLD THEN DO ASSOSICATED EXAMINATIONS OS CVS

STATION 8

CALCULATE JVP
AFTER THIS SIR ASKS QUESTIONS LIKE TELL SCENERIOS IN WHICH JVP IS RAISED
DO THE ENTIRE PROCEDURE

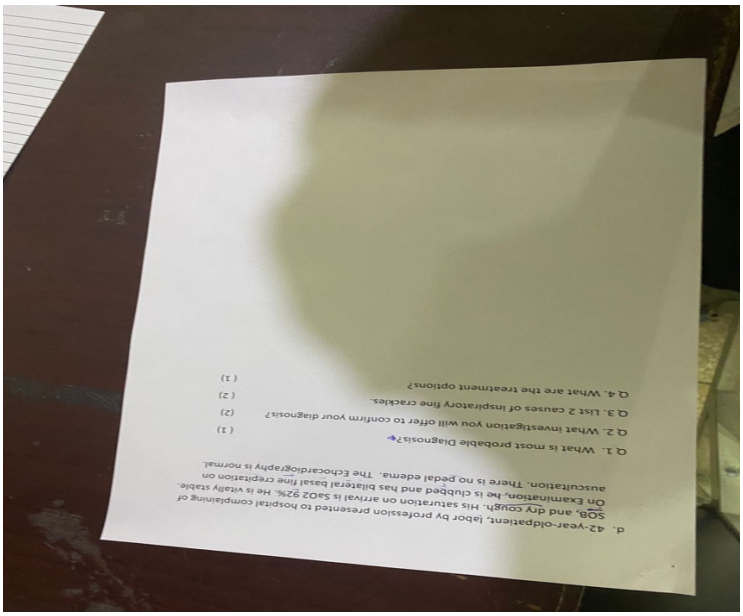
STATION 9

ON DUMMY SO THE ENTIRE CHEST EXAMINATION FROM FRONT AND BACK USKE BAAD THE TEACHER ASKS VIVA QUESTIONS RELEVANT TO MURMURS AND VALVULAR PROBS

STATIONS 10

EXAMINE THE CHEST FROM FRONT ON A REAL PATIENT
YE WALA TEACHER RUDE THE THODA

STATION 11



Q1: What is the most probable diagnosis?

Answer: Pulmonary Fibrosis

Pulmonary fibrosis is a chronic, progressive lung disease characterized by scarring (fibrosis) of the lung tissue, leading to dyspnea, dry cough, and reduced oxygen exchange.

Q2: What investigation(s) will you order to confirm your diagnosis?

Key Investigations for Pulmonary Fibrosis:

1. High-Resolution CT (HRCT) Chest – Gold standard

Shows honeycombing, reticular opacities, and ground-glass opacities

2. Pulmonary Function Tests (PFTs)

Restrictive pattern: ↓ FVC, ↓ TLC, normal or increased FEV1/FVC

3. 6-Minute Walk Test

Evaluates oxygen desaturation during exertion

4. Serologic Tests (if autoimmune cause is suspected)

ANA, RF, Anti-CCP, etc.

5. Bronchoscopy with BAL (Bronchoalveolar Lavage) or Lung Biopsy

To rule out infection, malignancy, or hypersensitivity pneumonitis

Q3: List 2 causes of inspiratory fine crackles.

1. Pulmonary Fibrosis – Due to stiff, fibrotic lung tissue

2. Congestive Heart Failure (CHF) – Due to fluid accumulation in the lungs (pulmonary edema)

Q4: What are the treatment options?

Treatment for Pulmonary Fibrosis Depends on the Cause:

1. Idiopathic Pulmonary Fibrosis (IPF):

Antifibrotic drugs: Pirfenidone, Nintedanib (slow disease progression)

Oxygen therapy (for hypoxemia)

Pulmonary rehabilitation (exercise & breathing techniques)

Lung transplant (for end-stage disease)

2. Autoimmune-Related Fibrosis (e.g., Scleroderma-associated ILD):

Immunosuppressants: Prednisolone, Mycophenolate, Azathioprine

3. Hypersensitivity Pneumonitis:

Avoid antigen exposure (e.g., bird proteins, mold)

Steroids (if severe cases)

4. Supportive Treatment:

Vaccination: Influenza, Pneumococcal

Smoking cessation

Summary of Answers:

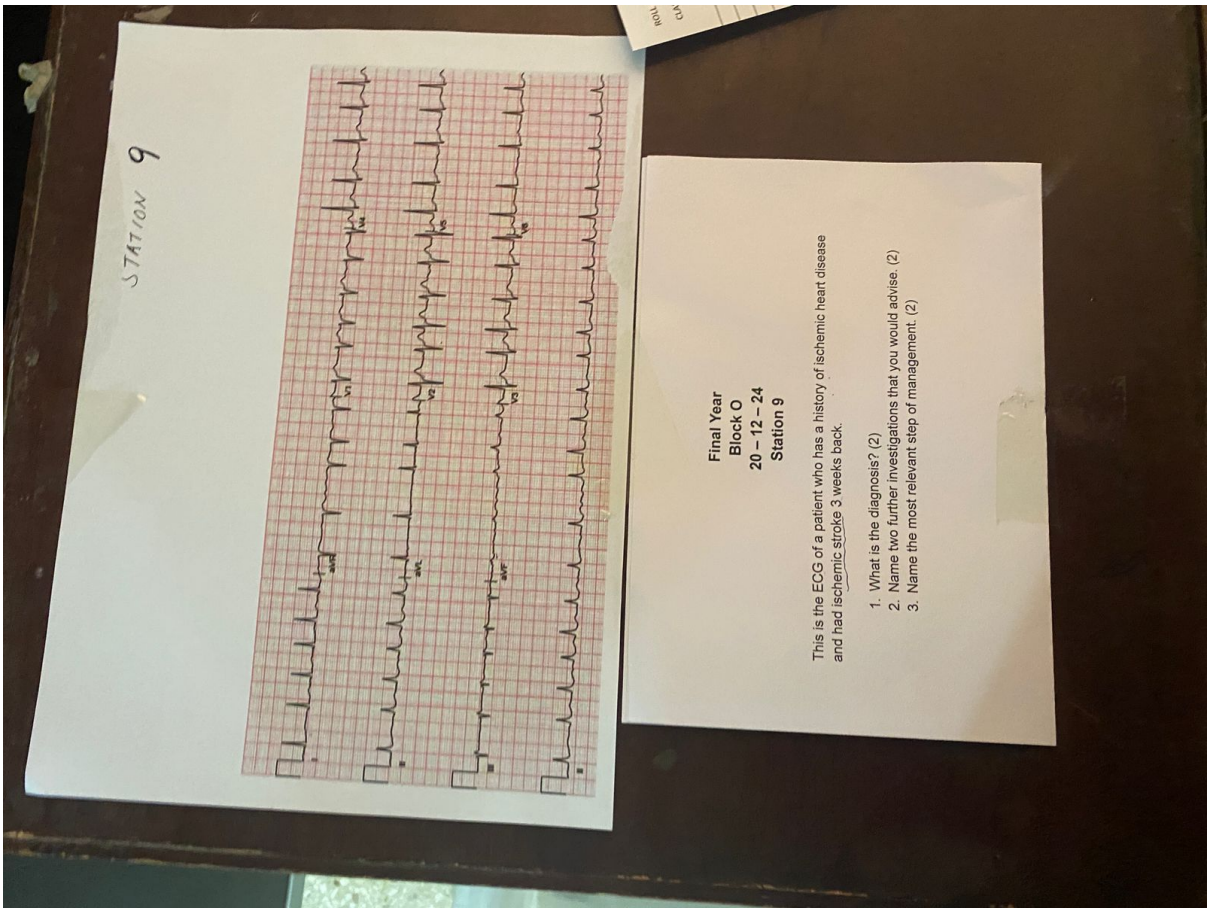
1. Most probable diagnosis: Pulmonary Fibrosis

2. Investigations: HRCT, PFTs, 6MWT, serology, bronchoscopy/biopsy

3. Causes of fine inspiratory crackles: Pulmonary fibrosis, CHF

4. Treatment options: Antifibrotics, oxygen therapy, pulmonary rehab, immunosuppressants (if autoimmune), lung transplant

STATION 12



Atrial Fibrillation (AF) Due to Ischemic Heart Disease – Investigations & Management

I. Investigations for AF Due to Ischemic Heart Disease
A. Clinical History and Physical Exam

Symptoms: Palpitations, fatigue, dyspnea, chest discomfort (angina-like symptoms)
Physical Exam: May show signs of heart failure (e.g., elevated JVP, edema)

or ischemia (e.g., signs of angina).
B. Electrocardiogram (ECG)

AF: Characterized by irregularly irregular rhythm, absence of P waves, and variable ventricular response.
Ischemic changes: Look for ST-segment changes, T-wave inversion, or Q waves, which suggest active or old myocardial ischemia/infarction.
C. Echocardiography (TTE or TEE)

Left ventricular function: Assess for systolic dysfunction due to ischemia (ischemic cardiomyopathy) or diastolic dysfunction from heart failure.
Valvular disease: To evaluate if ischemic heart disease (IHD) has affected heart valves.
Left atrial size: A larger left atrium is common in AF and contributes to the development of arrhythmias.
D. Blood Tests

Cardiac biomarkers (e.g., Troponin, CK-MB): To rule out acute myocardial infarction (MI).
BNP or NT-proBNP: To assess for heart failure.
Electrolytes (especially K+ and Mg2+): Low potassium or magnesium can precipitate AF.
E. Coronary Angiography

Gold standard for diagnosing ischemic heart disease: In patients with AF and suspected coronary artery disease (CAD), coronary angiography can identify significant coronary artery blockages.
Stress testing (e.g., stress echocardiography or stress ECG): If coronary angiography is not immediately available or if non-invasive assessment is preferred, this can identify areas of myocardial ischemia.
F. Cardiac MRI (if needed)

To assess cardiac structure, ventricular function, and scar tissue from previous myocardial infarctions (scarred tissue is a potential substrate for AF).
II. Management of AF in the Context of Ischemic Heart Disease
A. Rate Control

First-line treatment in most patients with AF, particularly in those with ischemic heart disease, to avoid tachycardia-induced ischemia.
Beta-blockers (e.g., Metoprolol, Carvedilol, Atenolol)
Preferred for rate control in IHD because they reduce myocardial oxygen demand and prevent tachycardia.
Calcium channel blockers (e.g., Diltiazem, Verapamil)
Used in rate control, but typically avoided in patients with heart failure with reduced ejection fraction (HFrEF).
Digoxin

Less effective for rate control during exercise, but can be considered in sedentary patients with heart failure and ischemic heart disease.

B. Rhythm Control

Rhythm control may be considered in patients with symptomatic AF or poor rate control despite adequate management. However, in patients with IHD, rate control is usually preferred.

Antiarrhythmic drugs (Class I or III):

Amiodarone (Class III): Often used in patients with ischemic heart disease due to its effectiveness in both rhythm control and heart failure.

Flecainide or Propafenone (Class IC): These can be used in patients without significant structural heart disease or when ischemic heart disease is not too advanced.

Electrical cardioversion (Direct Current Cardioversion, DCCV):

Consider in patients with symptomatic AF and hemodynamic instability. However, DCCV should be avoided in patients with significant coronary artery disease or in those who have persistent risk of thromboembolism, unless they are adequately anticoagulated.

C. Anticoagulation (Stroke Prevention)

Anticoagulation is essential for stroke prevention in AF patients, especially if the patient has ischemic heart disease (CAD), which predisposes to left atrial thrombus formation.

CHA2DS2-VASc score: Assess stroke risk:

Score ≥2 in men or ≥3 in women: Oral anticoagulation is recommended.

Anticoagulation options include:

Direct oral anticoagulants (DOACs) (e.g., Apixaban, Rivaroxaban, Dabigatran) for ease of use.

Warfarin (if DOACs are not suitable, with regular INR monitoring).

Consider antiplatelet therapy (e.g., Aspirin or Clopidogrel) in low-risk patients or if contraindication to anticoagulants, but this is generally not recommended over anticoagulation for stroke prevention.

D. Management of Underlying Ischemic Heart Disease

Revascularization:

Percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG) should be considered for patients with significant coronary artery disease and symptomatic ischemia.

Revascularization may also help improve left ventricular function and reduce AF burden.

Optimal Medical Therapy for IHD:

Aspirin (if not contraindicated), statins, and antianginals (e.g., nitroglycerin or beta-blockers) to manage angina and prevent further ischemic events.

Management of risk factors:

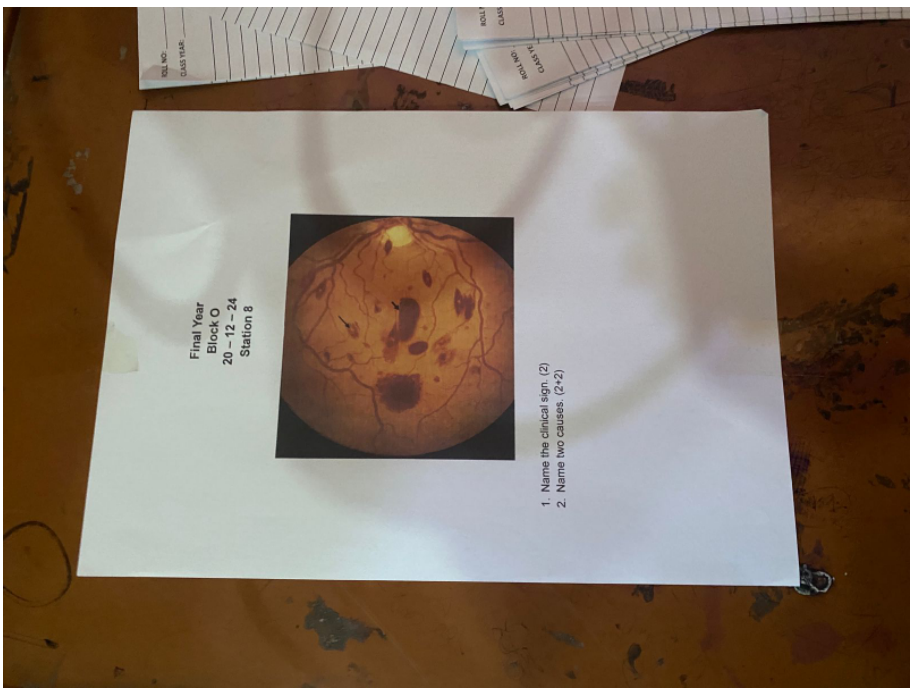
Hypertension, diabetes, and hyperlipidemia should be aggressively treated in patients with both AF and ischemic heart disease to improve overall cardiovascular health.

E. Management of Heart Failure (if present)

If heart failure is present due to ischemic heart disease, it should be managed with the standard heart failure therapy:
ACE inhibitors or ARBs, beta-blockers, aldosterone antagonists, and diuretics (for fluid control).
F. Catheter Ablation

Catheter ablation for AF may be considered in cases of persistent AF or recurrent AF despite pharmacological treatment. This is especially true if AF is symptomatic and significantly affecting the patient's quality of life.
III. Summary of Key Steps in Management
Rate control: Use beta-blockers, calcium channel blockers, or digoxin depending on clinical context.
Anticoagulation: Based on CHA2DS2-VASc score, use DOACs or warfarin for stroke prevention.
Management of ischemic heart disease: Consider PCI or CABG for revascularization, alongside antianginal therapy (e.g., beta-blockers).
Rhythm control: Consider antiarrhythmic drugs or cardioversion if symptomatic.
Heart failure management (if applicable): Standard therapy with ACE inhibitors, beta-blockers, aldosterone antagonists.
Catheter ablation: Consider for refractory AF or if rhythm control fails.

STATION 13



Roth spots are retinal findings characterized by white-centered hemorrhages and are typically seen on fundoscopy. They can be

associated with a variety of conditions, often linked to systemic diseases. The white center of a Roth spot represents a focal area of retinal hemorrhage surrounded by a pale region, and this appearance is often due to localized microvascular changes.

Here are the main causes of Roth spots:

1. Infective Endocarditis
Most common association: Roth spots are strongly linked with bacterial endocarditis (especially left-sided valve involvement). They result from embolic phenomena (e.g., septic emboli) causing microinfarctions in the retina.
Other signs of infective endocarditis include Osler nodes (painful lesions on fingers/toes), Janeway lesions (non-tender spots on palms/soles), and positive blood cultures.
2. Leukemia
Acute or chronic leukemia, particularly acute myeloid leukemia (AML), can cause retinal hemorrhages and Roth spots due to disseminated intravascular coagulation (DIC) or leukemic infiltration of retinal vessels.
3. Diabetic Retinopathy
Advanced diabetic retinopathy can cause a variety of retinal changes, including Roth spots, due to microvascular damage and retinal ischemia.
4. Hypertensive Retinopathy
Severe hypertension can lead to changes in the retinal vasculature, including Roth spots, typically associated with retinal hemorrhages and optic disc swelling.
5. Anemia
Severe anemia (particularly megaloblastic anemia and hypoproliferative anemia) can lead to retinal hemorrhages and Roth spots, often as a result of retinal hypoxia and increased blood flow to the retina.
6. Systemic Infections
Meningococcemia or sepsis (due to various bacterial or viral infections) can cause retinal hemorrhages, including Roth spots, due to septic emboli and DIC.
7. Endocrine Disorders
Systemic lupus erythematosus (SLE) and other autoimmune diseases may cause Roth spots due to the involvement of small blood vessels in the retina (vasculitis).
8. Cytomegalovirus (CMV) Retinitis
Particularly in immunocompromised individuals (e.g., HIV/AIDS patients), CMV retinitis can result in retinal hemorrhages and Roth spots, though CMV retinitis is typically characterized by retinal necrosis.
9. Subacute Bacterial Endocarditis (SBE)
Associated with infective endocarditis, specifically SBE, in which immune complexes or embolic events can lead to retinal changes such as Roth spots.
10. Coagulopathies and Thrombocytopenia

Conditions causing thrombocytopenia or disordered coagulation, such as idiopathic thrombocytopenic purpura (ITP), DIC, or hemophilia, can lead to retinal hemorrhages and Roth spots.

11. Toxins or Drugs

Cocaine use or methamphetamine can lead to retinal hemorrhages due to their effects on blood pressure and vascular tone.

12. Cardiac Embolism

Embolic events, such as those from atrial fibrillation, patent foramen ovale (PFO), or mechanical heart valves, may lead to septic or thrombotic emboli in the retina, causing Roth spots.

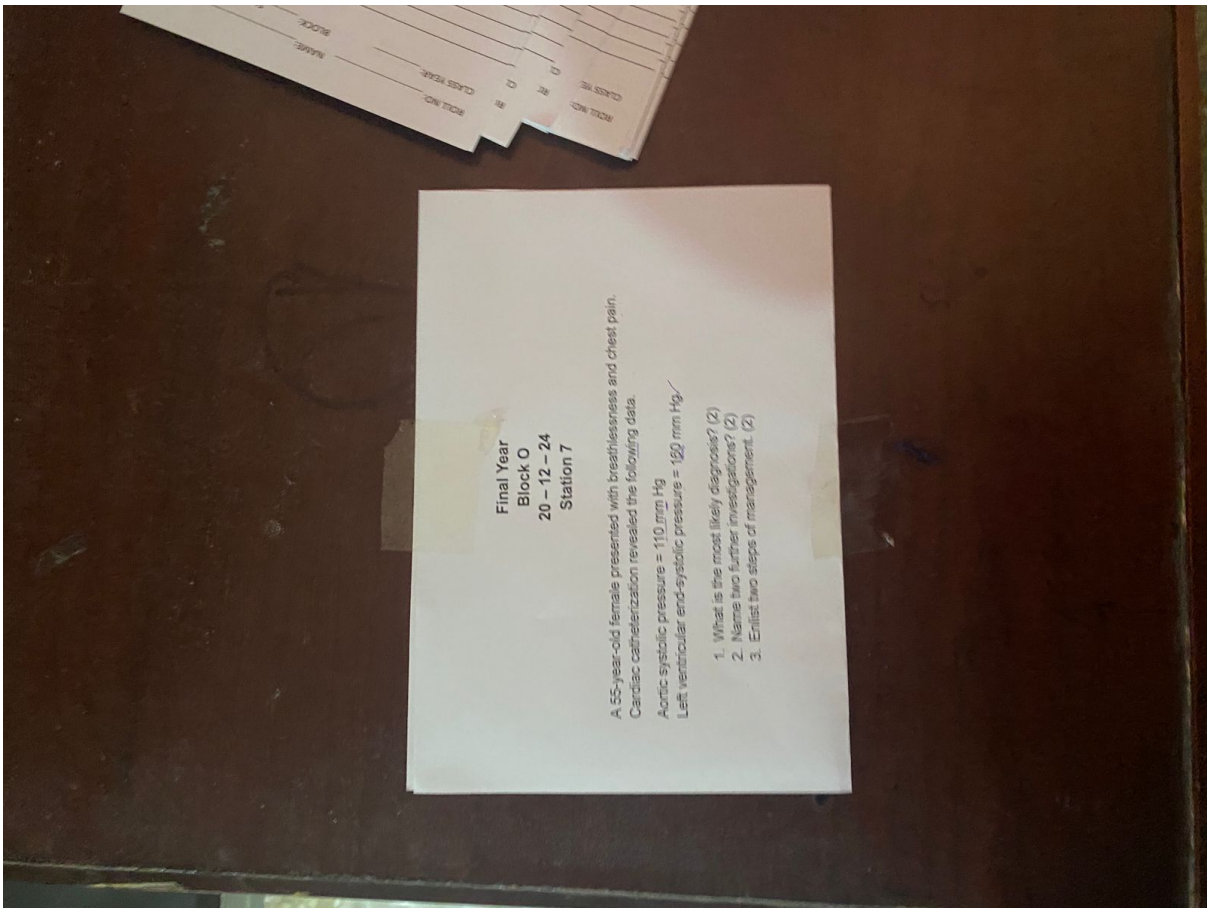
Summary of Conditions Associated with Roth Spots:

Infective endocarditis
Leukemia
Diabetic retinopathy
Hypertensive retinopathy
Severe anemia
Systemic infections (e.g., meningococcemia)
Endocrine disorders (e.g., lupus)
CMV retinitis (immunocompromised)
Coagulopathies and thrombocytopenia
Toxins or drugs (e.g., cocaine)
Cardiac embolism

Management

Identify and treat the underlying condition (e.g., antibiotics for endocarditis, chemotherapy for leukemia, anticoagulation for thromboembolic events, etc.).
Monitor retinal health closely, especially in patients with significant systemic disease.

STATION 14



Case Study:
A 65-year-old female presented with breathlessness and chest pain. Cardiac catheterization revealed the following data:
- Aortic systolic pressure: 110 mm Hg
- Left ventricular end-systolic pressure: 120 mm Hg

Questions:
1. What is the most likely diagnosis? (2)
2. Name two further investigations? (2)
3. Enlist two steps of management. (2)

Let's break it down:

1. Most Likely Diagnosis:
The discrepancy between the aortic systolic pressure (lower) and left ventricular end-systolic pressure (higher) could indicate an obstruction, often associated with conditions like aortic stenosis. The most likely diagnosis here is **aortic stenosis.**

2. Further Investigations:
To further investigate this condition, the following tests are recommended:
- **Echocardiogram:** To visualize the heart's structure and function, including the aortic valve.

- **Electrocardiogram (ECG):** To check for signs of left ventricular hypertrophy or other electrical abnormalities.

3. Steps of Management:
Management for aortic stenosis may include:
- **Surgical Intervention:** Aortic valve replacement is a common surgical procedure for severe cases.
- **Medications:** To manage symptoms and associated conditions (e.g., antihypertensives if there is concurrent high blood pressure).

STATION 15

INTERACTIVE STATION ABOUT PULMONARY EMBOLISM THERE WAS A SCENERIO IN WHICH IT WAS MENTIONED THE LADY HAD ANTI PHOPHOLIPID SYNDROME SUDDENLY DEVELOPED SOB AND HEMOPTYSIS

1) DIAGNOSIS
2) INVESTIGATIONS
3) MANAGEMENT

Pulmonary Embolism (PE) – Investigations and Management
I. Investigations for Pulmonary Embolism

Clinical Assessment
History: Risk factors for PE, such as recent surgery, immobilization, cancer, long flight, pregnancy, oral contraceptive use, or history of deep vein thrombosis (DVT).
Symptoms: Common signs include sudden-onset shortness of breath, chest pain, tachypnea, and hemoptysis. Patients may also report leg swelling (if DVT present).
Physical Examination
Tachypnea (increased respiratory rate), tachycardia, hypoxia, cyanosis.
Signs of DVT: Swelling, redness, or tenderness in the calf or thigh.
Lung auscultation: May reveal crackles or pleural rub.
Initial Investigations
D-dimer: Elevated levels suggest increased clot formation and fibrinolysis. It is sensitive but non-specific. It is used to rule out PE when the clinical probability is low. A normal D-dimer excludes PE in low-risk patients.
Arterial blood gases (ABG): Hypoxia and respiratory alkalosis (due to hyperventilation) are common in PE, but they are non-specific.
Imaging Studies
CT Pulmonary Angiography (CTPA): The gold standard for diagnosing PE. It provides a direct visualization of emboli in the pulmonary arteries.
Ventilation-Perfusion (V/Q) scan: Used when CTPA is contraindicated (e.g., in patients with contrast allergy or renal impairment). A high probability V/Q scan can diagnose PE in low-risk patients. A normal V/Q scan essentially rules out PE.

Chest X-ray: Typically normal but may show indirect signs of PE (e.g., atelectasis, pleural effusion). This test is not diagnostic but is helpful to rule out other causes of symptoms (e.g., pneumonia).
Ultrasound of the legs: If DVT is suspected, compression ultrasound is used to identify deep vein thrombosis as the source of emboli.
Echocardiography
Transthoracic echocardiogram (TTE): May show signs of right heart strain (e.g., right ventricular dilation, right atrial enlargement, or tricuspid regurgitation) and can suggest the presence of PE, especially in massive PE.
Transesophageal echocardiogram (TEE): Less commonly used but may be helpful in certain cases.
Electrocardiogram (ECG)
Sinus tachycardia is common in PE.
Other findings: S1Q3T3 pattern (deep S wave in lead I, Q wave and inverted T wave in lead III) may be seen in massive PE but is not diagnostic.
Pulmonary Angiography (rarely performed)
This is a gold standard test but is now rarely used due to the availability of CT pulmonary angiography.
II. Management of Pulmonary Embolism

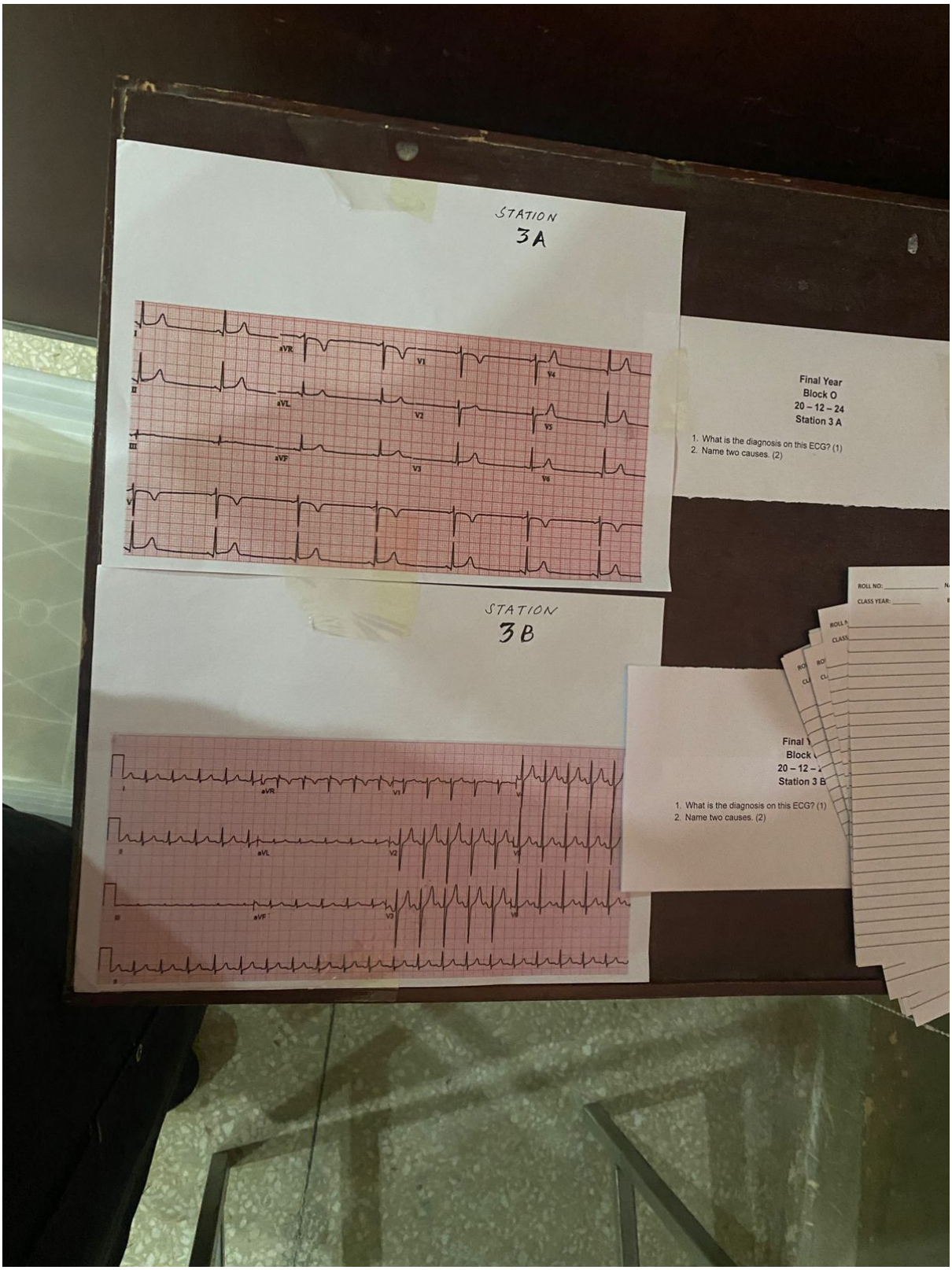
The management of PE depends on the severity of the embolism (massive, submassive, or low-risk) and the patient's clinical condition.

Initial Management
Oxygen therapy: To correct hypoxia (often required for most patients).
Hemodynamic support: If the patient is in shock, fluids and vasopressors may be required.
Anticoagulation: The cornerstone of treatment for all types of PE.
Anticoagulation Therapy
Heparin (unfractionated heparin or low-molecular-weight heparin) is started immediately for acute PE.
Unfractionated heparin (UFH) is typically used in patients requiring invasive procedures or those at high risk for bleeding.
Low-molecular-weight heparin (LMWH) (e.g., Enoxaparin) is preferred in most cases because it is easier to administer, and requires less frequent monitoring.
Oral anticoagulants (e.g., apixaban, rivaroxaban, dabigatran) are used for long-term anticoagulation after the initial heparin bridge.
Warfarin (if DOACs are contraindicated) is started after heparin therapy, with target INR of 2-3.
Thrombolysis
Fibrinolytic therapy (e.g., alteplase) is considered for massive PE with hemodynamic instability or cardiogenic shock.
Thrombectomy may be considered in patients with massive PE who are not candidates for thrombolysis or if thrombolysis fails.
Surgical Embolectomy
Indicated for patients with massive PE and persistent hemodynamic instability despite thrombolysis or fibrinolysis. This is often a last-resort option due to the

risks involved.
Inferior Vena Cava (IVC) Filter
An IVC filter may be considered in patients with contraindications to anticoagulation (e.g., active bleeding) or those with recurrent PE despite adequate anticoagulation.
Retrievable filters can be removed after the acute risk period has passed.
Supportive Care
Pain management: NSAIDs or opioids for managing chest pain associated with PE.
Monitoring: Close monitoring of vital signs, oxygen saturation, and laboratory tests (e.g., D-dimer, renal function).
III. Long-Term Management and Follow-Up

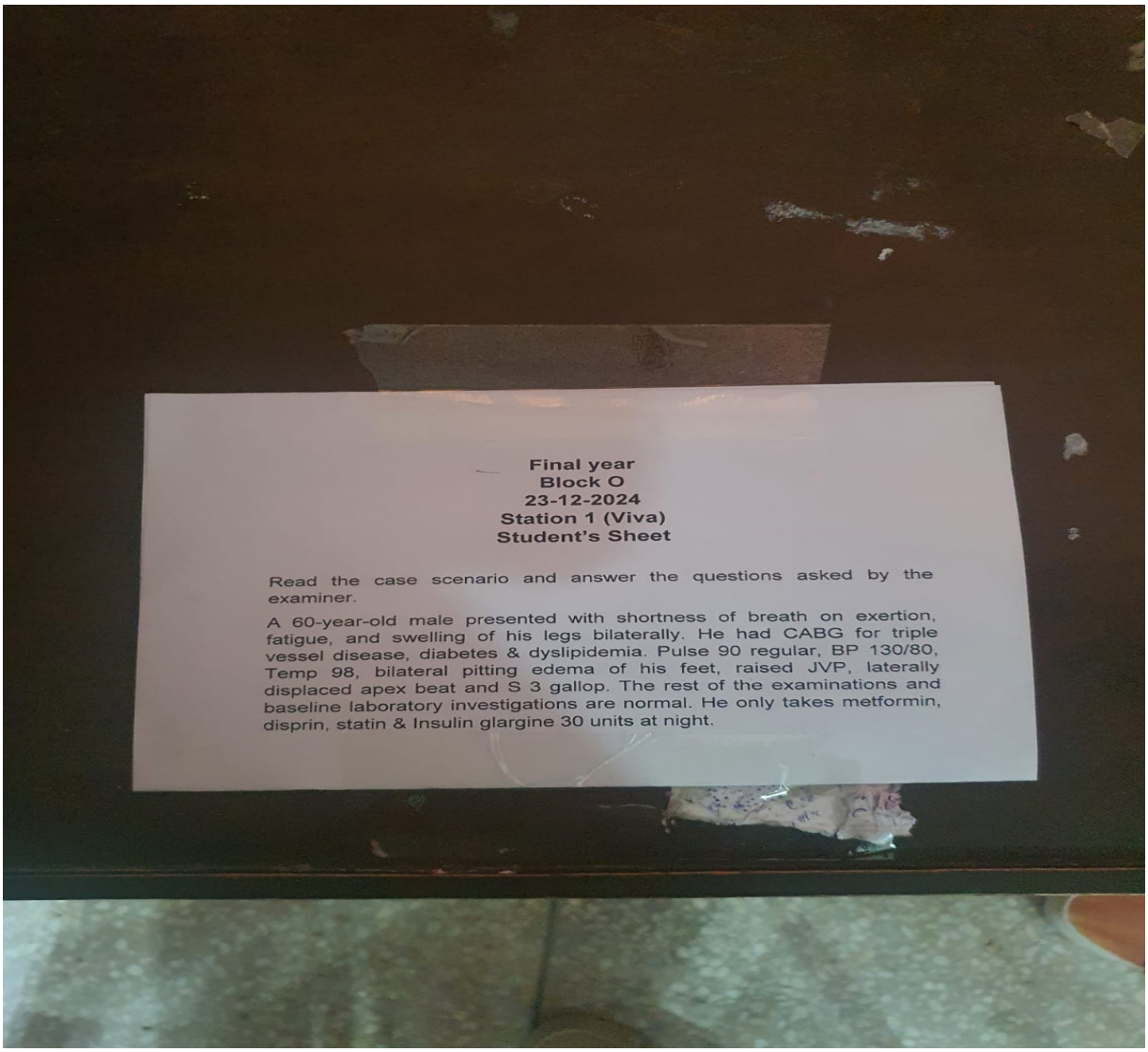
Duration of Anticoagulation
For provoked PE (e.g., after surgery or trauma), 3-6 months of anticoagulation is usually sufficient.
For unprovoked PE, long-term anticoagulation may be required, typically for 6-12 months, or even indefinitely if the risk of recurrence is high.
Risk stratification (e.g., through the PE recurrence risk, cancer, history of DVT/PE) will guide the decision for long-term anticoagulation.
Post-PE Management
Regular follow-up with the primary care provider or pulmonologist to monitor for recurrence of symptoms or complications.
Consider referral to a pulmonary rehabilitation program if there is ongoing respiratory distress or limitations due to PE.
IV. Summary of Management Steps
Initial management: Oxygen therapy, anticoagulation with LMWH or UFH.
Thrombolysis or surgery: For massive PE with hemodynamic instability.
Long-term anticoagulation: With DOACs or warfarin, duration based on risk.
IVC filter: In patients with contraindications to anticoagulation or recurrent PE.
Supportive care: Pain management, monitoring, and rehabilitation if needed.

STATION 16



DAY 3
23RD DECEMBER 2024

STATION 1



Final year
Block O
23-12-2024
Station 1 (Viva)
Student's Sheet

Read the case scenario and answer the questions asked by the examiner.

A 60-year-old male presented with shortness of breath on exertion, fatigue, and swelling of his legs bilaterally. He had CABG for triple vessel disease, diabetes & dyslipidemia. Pulse 90 regular, BP 130/80, Temp 98, bilateral pitting edema of his feet, raised JVP, laterally displaced apex beat and S 3 gallop. The rest of the examinations and baseline laboratory investigations are normal. He only takes metformin, disprin, statin & Insulin glargine 30 units at night.

1) DIAGNOSIS:

Heart Failure (likely systolic heart failure): The patient's symptoms (shortness of breath on exertion, fatigue, leg swelling), along with the physical findings (raised JVP, laterally

displaced apex beat, S3 gallop, bilateral pitting edema), strongly suggest heart failure. His history of CABG and diabetes are significant risk factors. The S3 gallop is a classic sign of systolic heart failure.

2) INVESTIGATIONS:

ECG (Electrocardiogram): To assess heart rhythm and look for any new changes since his CABG.
Chest X-ray: To evaluate heart size and check for pulmonary congestion (fluid in the lungs).
Echocardiogram: This is crucial to assess the heart's structure and function, particularly the ejection fraction (how well the left ventricle pumps blood). This will confirm the type of heart failure (systolic vs. diastolic).
Blood Tests:
BNP (B-type natriuretic peptide) or NT-proBNP: These are markers of heart failure.
Complete Blood Count (CBC): To check for anemia, which can exacerbate heart failure symptoms.
Comprehensive Metabolic Panel (CMP): To assess kidney and liver function, as these can be affected by heart failure and its treatment.
Thyroid Function Tests: To rule out thyroid problems that can mimic heart failure.
Possibly a repeat Coronary Angiogram: Since he has a history of CABG, this might be considered if there's concern for graft failure or progression of coronary artery disease.

3) ADVICE:

Medical Management:

Diuretics: To reduce fluid overload and alleviate symptoms like leg swelling and shortness of breath.
ACE Inhibitors or Angiotensin Receptor Blockers (ARBs): To improve heart function and reduce blood pressure.
Beta-blockers: To slow heart rate and improve heart function.
Mineralocorticoid Receptor Antagonists (MRAs): Such as spironolactone or eplerenone, to further reduce fluid retention and improve outcomes.
Optimization of diabetes and dyslipidemia management: It's

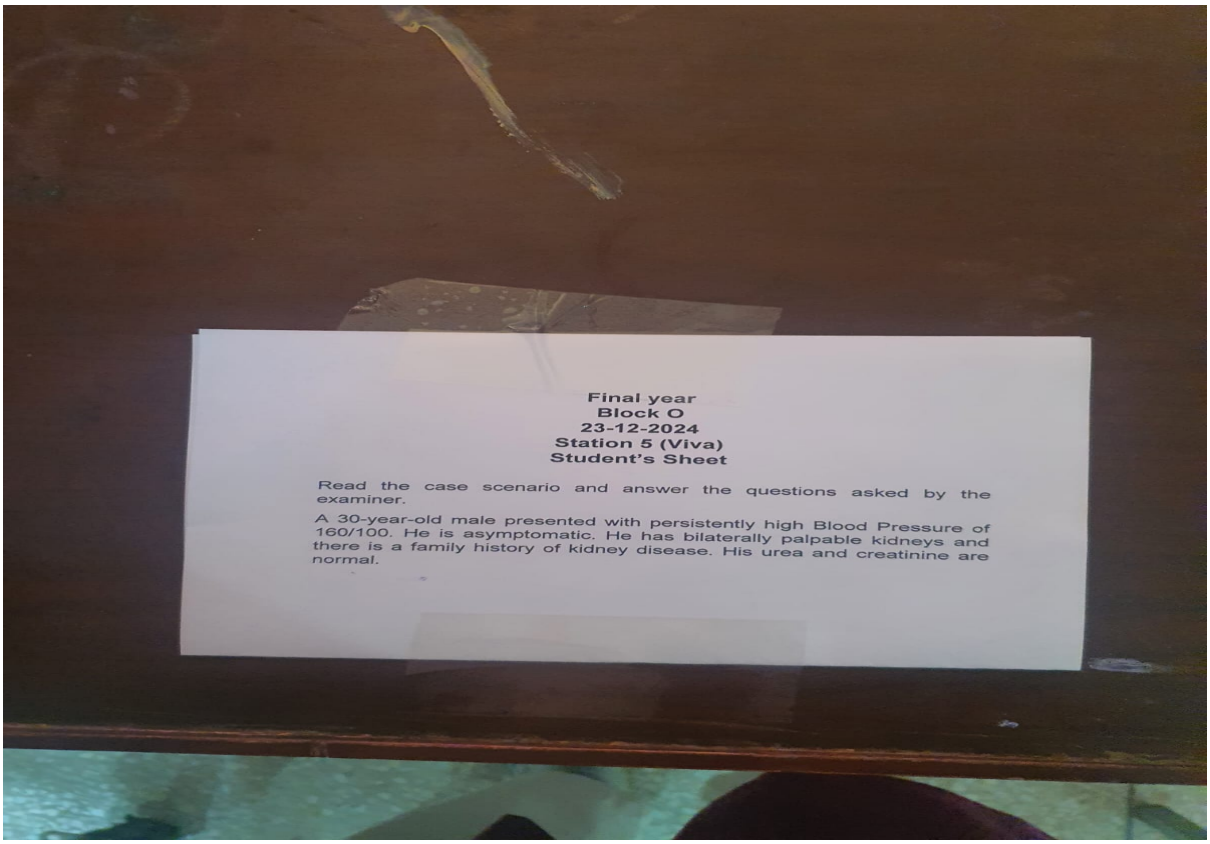
crucial to ensure his diabetes and cholesterol are well-controlled.
Lifestyle Modifications:

Sodium Restriction: Limiting salt intake can help reduce fluid retention.
Fluid Restriction: In some cases, limiting fluid intake may be necessary.
Weight Management: If the patient is overweight or obese, weight loss can improve symptoms.
Cardiac Rehabilitation: A supervised exercise program can improve cardiovascular fitness.
Smoking Cessation: If applicable.
Regular Monitoring: Close follow-up with a cardiologist is essential to monitor symptoms, adjust medications, and assess response to treatment.
Patient Education:

Understanding Heart Failure: The patient should be educated about the condition, its causes, and its management.
Medication Adherence: Emphasizing the importance of taking medications as prescribed.
Symptom Monitoring: Teaching the patient to recognize and report worsening symptoms.
When to Seek Medical Attention: Providing clear instructions on when to seek urgent medical care.
1) DIOAGNOSIS
2) INVESTIGATION
3) ADVICE

STATION 2

1) DIAGNOSIS
2) TREATMENT AND DIETARY ADVICE TO PATIENT



Treatment & Dietary Advice for Adult Polycystic Kidney Disease (ADPKD)

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1. Treatment for ADPKD

A. Slowing Disease Progression:

Tolvaptan (Vasopressin V2 receptor antagonist)

Slows cyst growth and kidney function decline.

Requires monitoring for liver toxicity (LFTs).

B. Blood Pressure Control (Target <130/80 mmHg):

ACE Inhibitors (e.g., Enalapril) or ARBs (e.g., Losartan)

Preferred for protecting kidney function.

C. Pain Management:

Acetaminophen (Paracetamol) preferred over NSAIDs (NSAIDs can worsen kidney function).

Cyst drainage (if pain is due to large cysts).

D. Infection Management:

Cyst Infection: Fluoroquinolones (e.g., Ciprofloxacin) or TMP-SMX (Bactrim).

UTIs: Treat promptly with antibiotics.

E. Managing Kidney Function & Dialysis:

Monitor kidney function (serum creatinine, eGFR).

If ESRD develops → Dialysis or Kidney Transplant.

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2. Dietary Advice for ADPKD

✅ Recommended Diet:

1. Low Sodium (<2.3g/day) → Helps control blood pressure.

2. Increase Water Intake → 3L/day (reduces vasopressin levels, slowing cyst growth).

3. Moderate Protein Intake → Avoid high-protein diets (can stress kidneys).

4. Low Phosphorus & Potassium (if kidney function declines):

Avoid dairy, processed foods (high in phosphorus).

Limit bananas, oranges, potatoes (high in potassium).

5. Healthy Fats & Fiber-Rich Diet

Olive oil, nuts, whole grains, fruits, and vegetables.

6. Limit Alcohol & Caffeine → May promote cyst growth.

❌ Foods to Avoid:

Processed & fast foods (high in sodium).

Sugary drinks (increase metabolic stress).

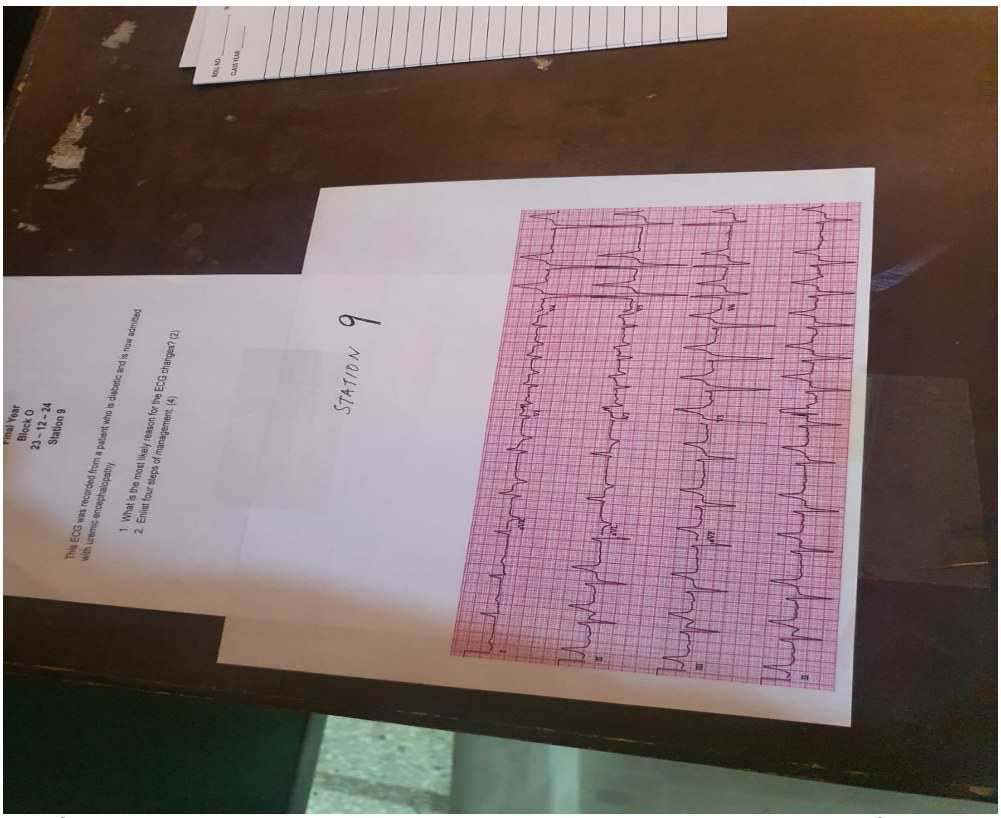
Excessive red meat (protein overload).

Lifestyle Modifications:

✅ Regular exercise (but avoid contact sports to prevent kidney injury).

✅ Quit smoking & reduce alcohol (protects kidney function).
✅ Monitor blood pressure & kidney function regularly.

STATION 3



Let's analyze this ECG in the context of the patient's clinical situation: diabetes and uremic encephalopathy (brain dysfunction due to kidney failure).

Initial Impressions:

Rhythm: Difficult to determine precisely due to the image quality, but it appears to be irregular. There are likely P waves present, but they are not consistently related to the QRS complexes.
Rate: Appears to be slow, likely in the bradycardic range.
Axis: Difficult to assess with certainty due to the rhythm irregularity.
Intervals:
PR Interval: Variable, likely due to the rhythm irregularity.
QRS Duration: Appears normal, but difficult to measure precisely.

QT Interval: Difficult to assess due to the rhythm irregularity.
Detailed Analysis:

P Waves: Present, but not consistently followed by QRS complexes. This suggests the possibility of atrioventricular (AV) block.
QRS Complexes: Appear relatively narrow.
ST Segments and T Waves:
ST Segments: Difficult to assess due to the rhythm irregularity.
T Waves: Appear broad and peaked, particularly in the precordial leads. This is a significant finding.
Key Finding:

Broad, Peaked T Waves: This is a classic sign of hyperkalemia (high potassium levels). In the context of uremic encephalopathy, hyperkalemia is a very likely cause.
Answers to the Questions:

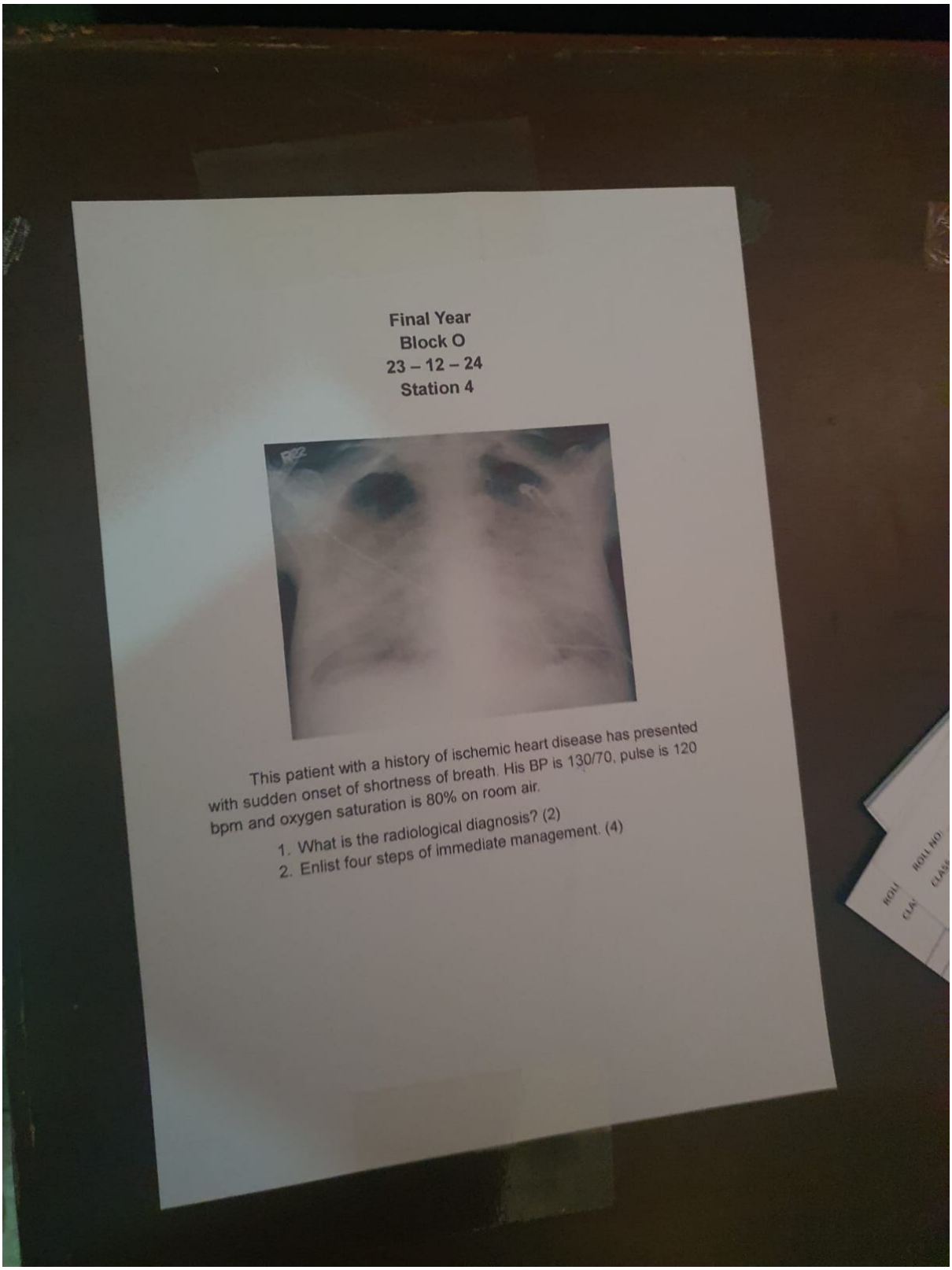
What is the most likely reason for the ECG changes? (2)

Hyperkalemia (high potassium levels) is the most likely reason for the broad, peaked T waves. The irregular rhythm could be related to the hyperkalemia or other electrolyte imbalances.
Enlist four steps of management. (4)

Stabilize the Cardiac Membrane: Administer calcium gluconate or calcium chloride to counteract the effects of potassium on the heart.
Shift Potassium Intracellularly:
Insulin and Glucose: Administer regular insulin with glucose to drive potassium into cells.
Sodium Bicarbonate: Can be used if the patient is acidotic.
Beta-2 Agonists (e.g., Albuterol): Can also shift potassium into cells.
Remove Potassium from the Body:
Diuretics (if the patient has adequate kidney function): Loop diuretics can help excrete potassium.
Sodium Polystyrene Sulfonate (Kayexalate): Exchanges sodium for potassium in the gut.
Hemodialysis: The most effective method for rapid potassium

removal, especially in patients with renal failure.
Address the Underlying Cause: Manage the patient's uremic encephalopathy and kidney failure. This may involve dialysis, medications to control blood pressure and diabetes, and other supportive measures.

STATION 4



*Case Study:**
- **Patient:** History of ischemic heart disease
- **Symptoms:** Sudden onset of shortness of breath
- **Vitals:** BP: 130/70, Pulse: 120 bpm, Oxygen saturation:

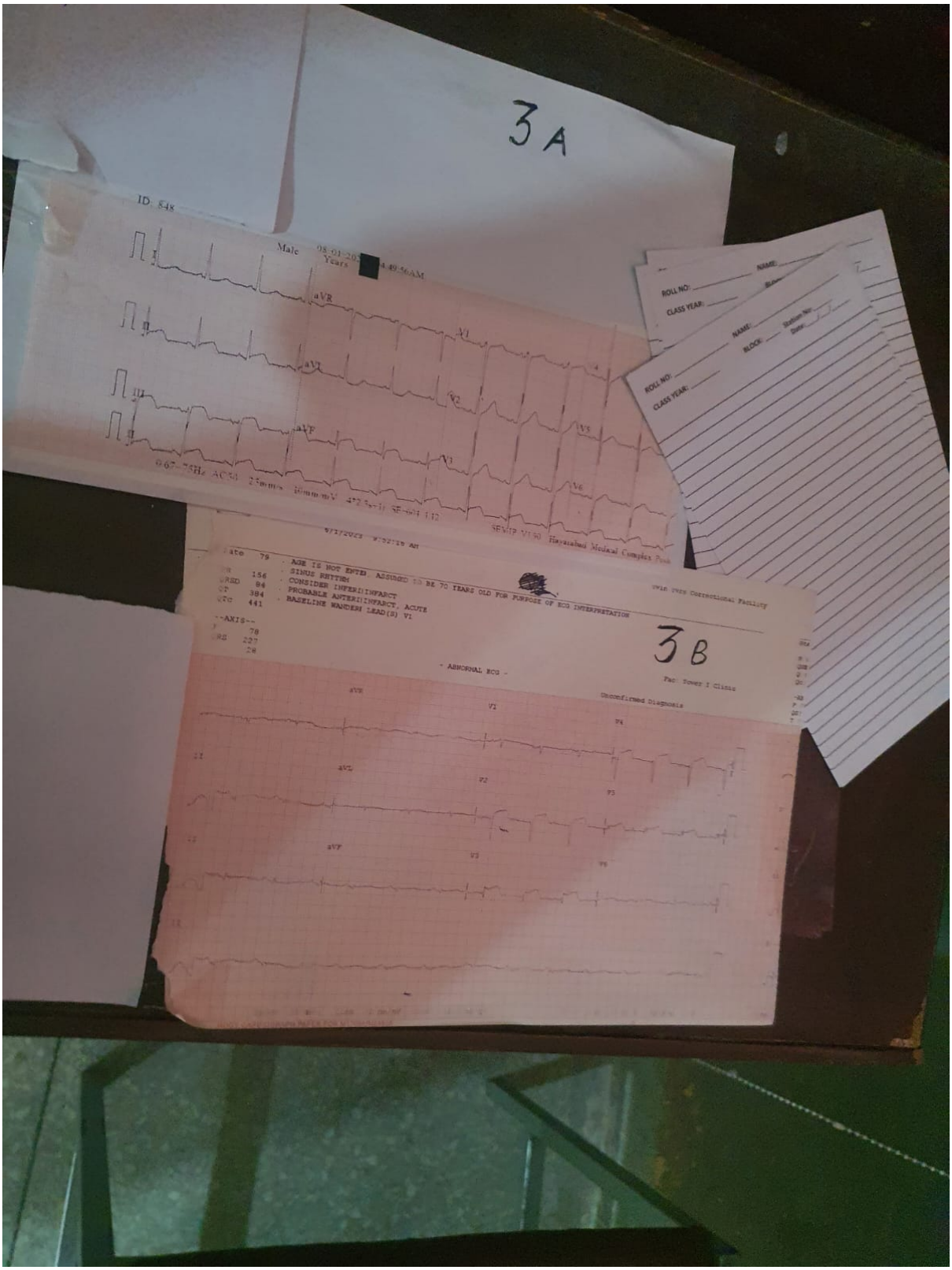
80% on room air

Questions:

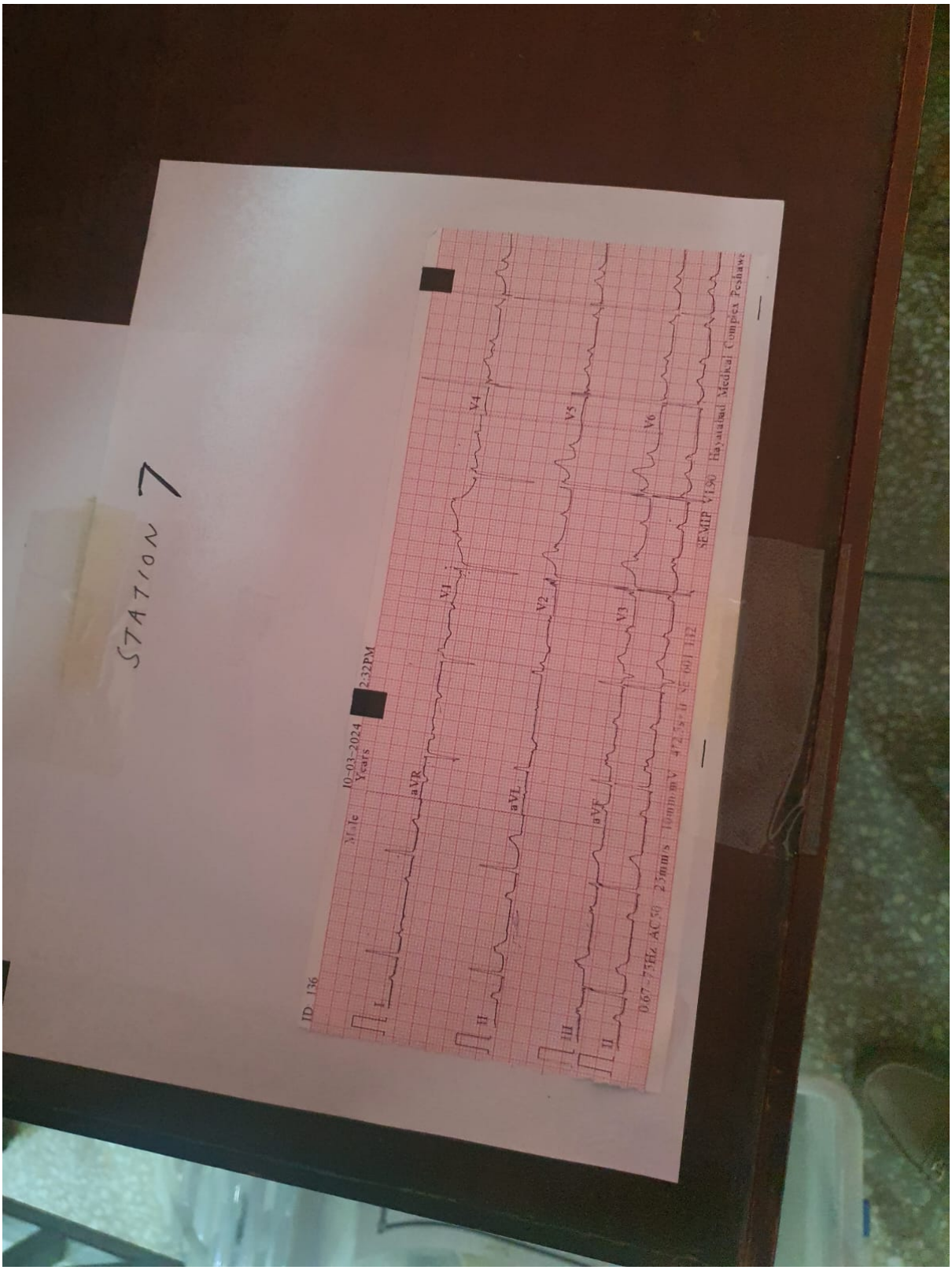
1. **What is the radiological diagnosis?**
   - The radiological diagnosis is likely **pulmonary edema**. This condition often presents as a result of heart failure, especially in patients with a history of ischemic heart disease. The chest X-ray would typically show signs of fluid accumulation in the lungs, such as bilateral infiltrates or an increased vascular marking.

2. **Enlist four steps of immediate management.**
   - **Oxygen Therapy:** Administering oxygen to improve oxygen saturation levels.
   - **Diuretics:** Administering diuretics like furosemide to reduce fluid overload.
   - **Positioning:** Elevating the head of the bed to alleviate breathlessness.
   - **IV Access and Monitoring:** Establishing IV access for medication administration and continuous monitoring of vitals.

STATION 5



STATION 6



STATION 7

A 23 year old girl with chronic productive cough her sister also

had the same disease and died

1) Cystic fibrosis leading to bronchiectasis
2) Defect ?
3) First line investigation
4) Management

In this case, the clinical features suggest **cystic fibrosis** (CF), which can lead to **bronchiectasis** due to chronic infections and inflammation in the airways.

Defect:
Cystic fibrosis is caused by mutations in the **CFTR (Cystic Fibrosis Transmembrane Conductance Regulator) gene**, located on chromosome 7. The most common mutation is **F508del**, which affects chloride and sodium ion transport across epithelial cells, leading to thick, viscous secretions in the lungs, pancreas, and other organs.

First-line Investigation:
1. **Sweat Chloride Test** – This is the gold standard diagnostic test for cystic fibrosis. Elevated chloride levels in sweat (>60 mmol/L) are highly indicative of CF.
2. **Genetic Testing** – To confirm mutations in the CFTR gene.
3. **Spirometry** – To assess lung function and detect any obstructive patterns due to bronchiectasis.

Management:
1. **Airway clearance therapies** (e.g., chest physiotherapy, positive expiratory pressure devices) to help remove mucus.
2. **Bronchodilators** (e.g., salbutamol) to improve airflow.
3. **Inhaled antibiotics** (e.g., tobramycin, aztreonam) for chronic Pseudomonas aeruginosa infection.
4. **Mucolytics** (e.g., dornase alfa) to thin mucus and improve airway clearance.
5. **Pancreatic enzyme supplements** for pancreatic insufficiency.
6. **Nutritional support** including fat-soluble vitamins (A, D, E, K).
7. **CFTR modulators** (e.g., ivacaftor, lumacaftor) in patients with specific mutations.
8. **Vaccination** (influenza and pneumococcal vaccines) to prevent respiratory infections.

9. **Lung transplantation** may be considered in end-stage lung disease.

Regular monitoring and multidisciplinary care are essential for managing CF patients.

STATION 8

EXAMINATION OF JVP
HOW TO DIFFERENTIATE JVP AND CAROTID PULSE ?
IN WHAT CONDITIONS IS JVP RAISED?
Carotid Pulse vs. JVP:

Carotid Pulse:

Location: Felt in the neck, medial to the sternocleidomastoid muscle.
Characteristics:
A strong, palpable pulsation.
Reflects arterial pressure.
A single outward movement (systolic).
Not significantly affected by respiration.
Not obliterated by light pressure.
Clinical Significance: Assesses arterial pulse characteristics (rate, rhythm, amplitude).
Jugular Venous Pressure (JVP):

Location: Observed in the neck, specifically the internal jugular vein.
Characteristics:
A softer, more undulating movement.
Reflects right atrial pressure.
Has a complex waveform with multiple components (a, c, and v waves, and x and y descents).
Varies with respiration (decreases during inspiration).
Obliterated by light pressure.
Height of JVP changes with posture.
Clinical Significance: Provides information about right atrial pressure and volume status.
Key Differences Summarized:

Nature of Pulsation: Arterial (carotid) vs. venous (JVP).

Response to Respiration: Minimal (carotid) vs. significant (JVP).
Response to Pressure: Not obliterated (carotid) vs. obliterated (JVP).
Waveform: Single outward movement (carotid) vs. complex waveform (JVP).
Conditions in Which JVP Is Raised:

An elevated JVP indicates increased right atrial pressure, which can be caused by several conditions:

Right-sided heart failure: The most common cause, where the right ventricle cannot pump effectively.
Tricuspid regurgitation: Backflow of blood from the right ventricle to the right atrium.
Tricuspid stenosis: Narrowing of the tricuspid valve, obstructing blood flow.
Pulmonary hypertension: Increased pressure in the pulmonary arteries, leading to right ventricular overload.
Cardiac tamponade: Compression of the heart by fluid in the pericardial space.
Constrictive pericarditis: Thickening and stiffening of the pericardium, restricting heart filling.
Superior vena cava obstruction: Blockage of blood flow in the superior vena cava.
Volume overload: Excessive fluid in the circulatory system.
Important Note:

Accurate JVP assessment requires proper technique and clinical expertise.
It's always crucial to consider the JVP findings in conjunction with other clinical findings and investigations.

STATION 9

RESPIRATORY EXAMINATION OF A ONE YEAR OLD ON DUMMY

STATION 10

40 YEAR OLD WITH PRODUCTIVE COUGH AND FOUL SMELLING SPUTUM AND BILATERAL WHEEZING

1) DIAGNOSIS?
2) INITIAL TEST
3) MANAGEMENT

This presentation suggests **bronchiectasis**, particularly given the productive cough, foul-smelling sputum, and bilateral wheezing, which are hallmark features. Bronchiectasis is characterized by the permanent dilation of bronchi due to chronic infection, inflammation, and airway destruction.

Diagnosis:
The likely diagnosis is **bronchiectasis**.

Initial Test:
1. **Chest X-ray** – To look for signs of bronchial wall thickening or abnormal lung architecture. This can give clues, but it may not be conclusive.
2. **High-Resolution CT (HRCT) Scan of the Chest** – This is the **gold standard** for diagnosing bronchiectasis. It will show dilated, thick-walled bronchi, which confirm the diagnosis.

Additional tests may include:
- **Sputum Culture** – To identify pathogens, especially if Pseudomonas or other resistant organisms are suspected.
- **Pulmonary Function Tests (PFTs)** – To assess the degree of airflow obstruction.
- **Blood tests** – To evaluate for underlying causes, such as immune deficiencies.

Management:
1. **Airway Clearance Techniques** (e.g., chest physiotherapy, postural drainage) to help clear mucus.
2. **Bronchodilators** (e.g., salbutamol or ipratropium) to relieve wheezing and improve airflow.
3. **Antibiotics** – Tailored to sputum culture results. Empirical treatment may start with broad-spectrum antibiotics (e.g., amoxicillin-clavulanate or levofloxacin). If chronic Pseudomonas infection is present, inhaled antibiotics (e.g., tobramycin or colistin) may be used.
4. **Mucolytics** (e.g., nebulized hypertonic saline) to thin the mucus and make it easier to expectorate.
5. **Anti-inflammatory therapy** – Inhaled corticosteroids may be

used in some cases, particularly if there is an asthma or chronic obstructive pulmonary disease (COPD) overlap.
6. **Management of underlying conditions** – If there is an underlying cause such as immune deficiency or cystic fibrosis, this must be addressed.
7. **Vaccination** – Influenza and pneumococcal vaccines to prevent infections.

Long-term management:
- Regular follow-up with pulmonary rehabilitation.
- Surgery may be considered in localized cases or severe disease.

STATION 11

1) DIAGNOSIS
2) TYPES OF PLEURAL EFFUSION
3) MANAGEMENT
4) SIGNS OF PLEURAL EFFUSION

Types of Pleural Effusions:

Pleural effusions are categorized based on the protein content of the fluid:

Transudative Effusions:

These occur due to systemic factors that alter the hydrostatic or oncotic pressures in the pleural capillaries.
The pleural membranes themselves are not directly involved.
The fluid is typically low in protein.
Common causes include:
Heart failure
Liver cirrhosis
Nephrotic syndrome
Hypoalbuminemia
Exudative Effusions:

These occur due to diseases that directly involve the pleural membranes, leading to increased capillary permeability or impaired lymphatic drainage.
The fluid is typically high in protein.

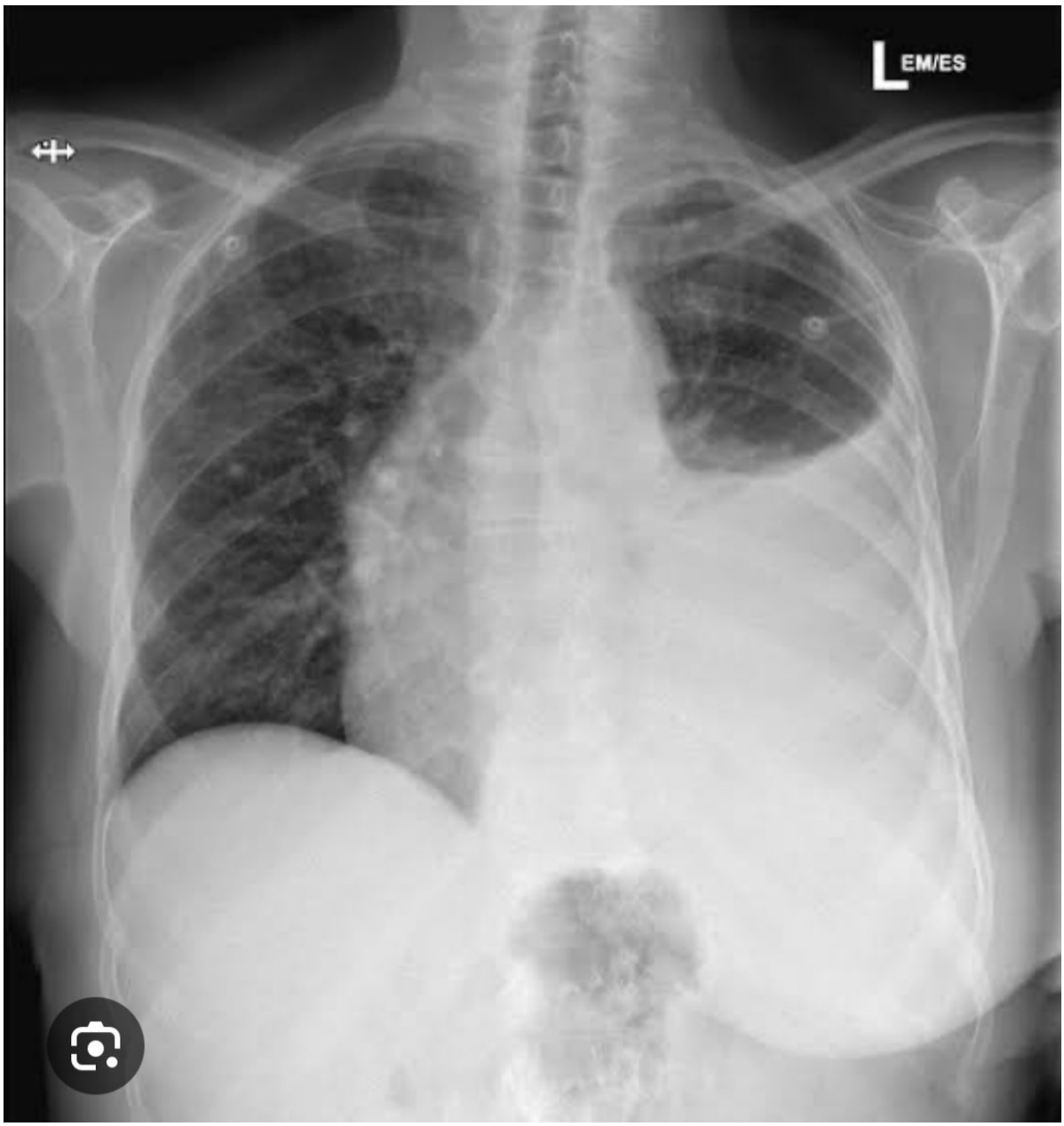
Common causes include:
Infections (pneumonia, tuberculosis)
Malignancy (lung cancer, mesothelioma)
Pulmonary embolism
Connective tissue diseases (rheumatoid arthritis, lupus)
Pancreatitis
Chylothorax (lymphatic fluid in the pleural space)
Management of Pleural Effusions:

Management depends on the type, size, and cause of the effusion:

Treat the Underlying Cause: This is paramount. For example, treating heart failure with diuretics or antibiotics for pneumonia.
Therapeutic Thoracentesis:
A needle is inserted into the pleural space to drain fluid.
This is done for large effusions causing respiratory distress or to obtain fluid for diagnostic analysis.
Pleurodesis:
A sclerosing agent (e.g., talc) is instilled into the pleural space to cause inflammation and adhesion of the pleural layers, preventing fluid accumulation.
This is used for recurrent malignant effusions or pneumothorax.
Pleural Catheter Drainage:
A small catheter is inserted into the pleural space for continuous drainage.
This is useful for large or recurrent effusions.
Surgery (VATS or Open):
In some cases, surgery may be needed to remove thickened pleura or treat underlying conditions.
Signs of Pleural Effusion on X-Ray:

Blunting of the costophrenic angle: This is the earliest sign, where the normally sharp angle between the ribs and diaphragm becomes obscured.
Increased opacity of the hemithorax: The affected side of the chest appears whiter than the other side.
Mediastinal shift: In large effusions, the mediastinum (the space between the lungs) may be pushed to the opposite side.
Meniscus sign: A curved upper border of the effusion as it rises along the chest wall.

Subpulmonic effusion: When the fluid collects under the lung, it may cause apparent elevation of the diaphragm.
Loculated effusion: This means that the effusion is trapped in a specific area and may appear as a mass-like density.



STATION 12

50 YEAR OLD MAN HAVING PRODUCTIVE COUGH AND BILATERAL WHEEZE SMOKER FROM 15 YEARS SATURATION IS 93 BP AND

PULSE NORMAL ,BY BRONCHODILATOR FEV1 IS IMPROVEMENT IS LESS THAN 15 %

1) DIAGNOSIS
2) HOW ASTHMA IS DIFFERENTIATED FROM COPD
3) INDICATIONS FOR INVASIVE MECHANICAL VENTILATION IN THIS PATIENT
4) MANAGEMENT

1) DIAGNOSIS:

The most likely diagnosis is Chronic Obstructive Pulmonary Disease (COPD).

Why COPD? The patient's age, smoking history, productive cough, and limited response to bronchodilators are classic features of COPD. While asthma can also cause wheezing, the limited reversibility with bronchodilators strongly points towards COPD.

Feature	Asthma	COPD
Onset	Often childhood or young adulthood	Typically middle age or older
Smoking History	Not always present	Usually present
Symptoms	Intermittent, variable, often worse at night/early morning	Persistent, progressive, worse with exertion
Sputum	May be present, often mucoid	Often present, may be purulent
Allergy/Atopy	Often present	Less common
Response to Steroids	Good response	Less responsive
Allergy/Atopy	Often present	Less common
Response to Steroids	Good response	Less responsive

Bronchodilator Response	Significant improvement in FEV1 (>15%)	Limited improvement in FEV1 (<15%)
Inflammation	Eosinophilic	Neutrophilic
Airway Remodeling	Reversible	Irreversible
Other Factors	Allergies, family history of asthma	Occupational exposures
Spirometry	Normal FEV1 between exacerbations	Reduced FEV1, not fully reversible
Diurnal Variation	Greater than 20% in peak expiratory flow	Less than 20% in PEFR

3) INDICATIONS FOR INVASIVE MECHANICAL VENTILATION IN THIS PATIENT:

Given the patient's current presentation (saturation 93%, normal BP/pulse), he does not currently require invasive mechanical ventilation. However, indications for invasive ventilation in a COPD patient would include:

Severe respiratory acidosis (pH < 7.25) with hypercapnia ($PaCO_2$ > 60 mmHg): This indicates respiratory failure.
Severe hypoxemia (PaO_2 < 55 mmHg) despite supplemental oxygen: This suggests inadequate gas exchange.
Respiratory muscle fatigue: Evidenced by rapid, shallow breathing, use of accessory muscles, and paradoxical abdominal movements.
Altered mental status: Due to hypercapnia or hypoxemia.
Hemodynamic instability: Such as hypotension or cardiac arrhythmias.
Inability to protect the airway: Due to decreased consciousness or severe cough.

4) MANAGEMENT:

Oxygen Therapy: Titrate oxygen to maintain saturation between 88-92%.
Bronchodilators:
Short-acting beta-agonists (SABA) like albuterol: For symptom relief.
Short-acting muscarinic antagonists (SAMA) like ipratropium: Can be used in combination with SABA.
Long-acting beta-agonists (LABA) like salmeterol or formoterol: For maintenance therapy.
Long-acting muscarinic antagonists (LAMA) like tiotropium: For maintenance therapy.
Inhaled Corticosteroids (ICS): In combination with LABA or LAMA for patients with frequent exacerbations.
Systemic Corticosteroids: For acute exacerbations, short course (5-7 days) of oral prednisone.
Antibiotics: If there is evidence of a bacterial infection (e.g., increased sputum purulence, fever).
Pulmonary Rehabilitation: To improve exercise tolerance and quality of life.

Smoking Cessation: Essential to slow disease progression.
Vaccinations: Influenza and pneumococcal vaccines to prevent infections.
Non-invasive Ventilation (NIV): May be considered for acute exacerbations before resorting to invasive ventilation.
STATION 13

EXAMINE PRECORDIUM KEEPING IN VIEW CONGENITAL HEART DISEASES

STATION 14

EXAMINE THE PRECORDIUM IN ADULT

STATION 15

EXAMINE THE RESPIRATORY SYSTEM IN ADULTS

STATION 16

GRANDPARENTS HAD CHRONIC COUGH AND BABY NOW HAVE CONSOLIDATION (TB SCENERIO)

1) DIAGNOSIS
2) INVESTIGATION
3) VACCINATION

Diagnosis

The most likely diagnosis in this scenario is Pulmonary Tuberculosis (TB) in the infant, likely due to household exposure from grandparents with chronic cough (suggesting TB transmission).

Investigations

1. Chest X-ray – To identify consolidation, cavitation, or miliary TB.

2. Tuberculin Skin Test (TST/Mantoux Test) – Positive if >10 mm induration.

3. Gastric Aspirate or Induced Sputum for AFB Stain & Culture – Since infants cannot produce sputum easily.

4. GeneXpert MTB/RIF (PCR for TB) – Rapid detection of TB and rifampicin resistance.

5. Complete Blood Count (CBC) – May show anemia, leukocytosis, or lymphocytosis.

6. Erythrocyte Sedimentation Rate (ESR) & C-reactive Protein (CRP) – Elevated in active TB.

7. HIV Testing – Always check for co-infection, especially in endemic areas.

Vaccination

Bacillus Calmette-Guérin (BCG) Vaccine

Given at birth to prevent severe forms of TB (miliary TB, TB meningitis).

If unvaccinated, BCG can be given after ruling out active TB.

Next Steps

Start Anti-TB Therapy (HRZE – Isoniazid, Rifampicin, Pyrazinamide, Ethambutol) if TB is confirmed.

Screen and treat close contacts (including grandparents) for TB.

Nutritional support & Vitamin D supplementation.