NEET SS 2024 Diploma Tuberculosis and Chest Disease Question Paper 2 with Solutions

Time Allowed :3 Hours	Maximum Marks :100	Total Questions :10

General Instructions

Read the following instructions very carefully and strictly follow them:

1. The test is of 3 hours duration.
2. The question paper consists of 10 questions. The maximum marks are 100.
3. Each Question is of 10 marks.

Q1. a) Pulmonary Langerhans cell histiocytosis (PLCH). [5]

Solution:

Step 1: Introduction to Pulmonary Langerhans Cell Histiocytosis (PLCH).
Pulmonary Langerhans cell histiocytosis (PLCH) is a rare interstitial lung disease characterized by the accumulation of Langerhans cells (a type of dendritic cell) in the lungs. It primarily affects young adults, particularly smokers, and is considered a form of histiocytosis that leads to the destruction of lung tissue. It is often associated with cystic changes, fibrosis, and inflammation in the lungs.

Etiology:
- PLCH is strongly associated with cigarette smoking, with most cases occurring in smokers or former smokers. The pathogenesis involves clonal proliferation of Langerhans cells, which leads to the formation of granulomas and cystic lung changes.
- The disease is more common in young adults (20-40 years), with a male predominance.

Step 2: Clinical Features of PLCH.
- Symptoms: Patients may present with a variety of symptoms, including:
- Cough, often non-productive or productive of minimal sputum.
- Dyspnea on exertion or at rest.
- Chest pain (less common).
- Fatigue and weight loss in more advanced cases.
- Signs: On examination, patients may have:
- Normal findings in early stages of disease.
- Crackles or wheeze in more advanced disease, typically related to interstitial lung disease or airflow obstruction.

Step 3: Diagnosis of PLCH.
- Chest X-ray: The classic finding is bilateral, upper lobe cysts, often described as "bunches of grapes". These cysts can range in size and may be accompanied by nodules.
- CT scan: High-resolution CT (HRCT) shows bilateral cystic changes with a predilection for

the upper lobes. The cysts may be surrounded by ground-glass opacities, nodules, and bronchial wall thickening.
- Biopsy: Bronchoscopy with transbronchial biopsy may show Langerhans cells in granulomas, with eosinophils and CD1a+ cells, confirming the diagnosis.
- Pulmonary Function Tests (PFTs): PFTs can show restrictive or obstructive patterns depending on the stage of the disease.

Step 4: Management of PLCH.
- Smoking cessation is the most important step in halting disease progression.
- Corticosteroids or other immunosuppressive therapies (e.g., methotrexate, azathioprine) may be considered in patients with progressive disease.
- In severe cases with respiratory failure, lung transplantation may be considered.
- Supportive care includes oxygen therapy, pulmonary rehabilitation, and symptomatic management for respiratory symptoms.

Quick Tip

The most crucial step in managing PLCH is smoking cessation. Early detection and intervention can prevent disease progression and improve outcomes.

Q1. b) Lymphangioleiomyomatosis (LAM). [5]

Solution:

Step 1: Introduction to Lymphangioleiomyomatosis (LAM).
Lymphangioleiomyomatosis (LAM) is a rare, progressive disease that primarily affects women of reproductive age, particularly those with tuberous sclerosis complex (TSC). It is characterized by the abnormal growth of smooth muscle-like cells in the lungs, lymphatics, and abdominal organs. This leads to lymphatic obstruction, cystic lung changes, and pulmonary insufficiency.

Etiology and Pathogenesis:
- LAM is associated with mutations in the TSC1 or TSC2 genes, which are involved in regulating the mTOR signaling pathway, leading to the uncontrolled growth of smooth muscle cells.
- Tuberous sclerosis complex (TSC), which is present in up to 50% of patients with LAM, increases the risk of developing this disease. However, LAM can also occur sporadically without TSC.

Step 2: Clinical Features of LAM.
- Symptoms: The most common symptoms include:
- Dyspnea on exertion or at rest.
- Cough, which may be dry or productive.
- Chest pain and hemoptysis (less common).
- Fatigue, weight loss, and frequent pneumothoraces (collapsed lungs) are also seen in advanced disease.

- Signs: On examination, patients may have:
- Normal findings in the early stages of the disease.
- In more advanced stages, there may be tachypnea, hypoxia, or signs of pneumothorax.

Step 3: Diagnosis of LAM.
- Chest X-ray: May show multiple cysts, often most prominent in the mid to lower lung fields.
- CT scan: High-resolution CT (HRCT) demonstrates diffuse, bilateral cystic changes in the lungs, often with normal lung parenchyma between cysts. This is the hallmark finding in LAM.
- Pulmonary Function Tests (PFTs): LAM often presents with a restrictive pattern of lung dysfunction, and progressive obstructive patterns may develop as the disease progresses.
- Biopsy: Diagnosis is confirmed through lung biopsy showing smooth muscle-like cells (LAM cells) and TSC gene mutations.
- MRI/CT of the abdomen: May reveal renal angiomyolipomas, which are present in TSC and sometimes in LAM.

Step 4: Management of LAM.
- Oxygen therapy is used to manage hypoxia in advanced cases.
- Sirolimus (an mTOR inhibitor) has shown promise in reducing disease progression, improving lung function, and preventing pneumothoraces.
- Lung transplantation is considered in severe, progressive cases of LAM with end-stage lung disease.
- Pneumothorax is managed with chest tubes or surgery in recurrent cases.
- Hormonal therapy may be considered in some cases to reduce estrogen-driven progression of the disease.

Quick Tip

Sirolimus, an mTOR inhibitor, is effective in slowing disease progression in LAM patients, particularly in those with progressive lung disease.

Q2. a) Diagnosis and treatment of pneumocystis pneumonia. [5]

Solution:

Step 1: Introduction to Pneumocystis Pneumonia (PCP).
Pneumocystis pneumonia (PCP) is a severe lung infection caused by the fungus Pneumocystis jirovecii. It primarily affects immunocompromised individuals, particularly those with HIV/AIDS or those receiving immunosuppressive therapy (e.g., chemotherapy, corticosteroids, or organ transplantation). PCP can lead to significant morbidity and mortality if not diagnosed and treated promptly.

Step 2: Diagnosis of Pneumocystis Pneumonia.

(1) Clinical Features:
- Patients with PCP typically present with progressive dyspnea, non-productive cough, and fever.
- Fatigue and weight loss are also common, especially in HIV-positive individuals with a low CD4 count.
- Chest pain and hemoptysis may occur, though less frequently.

(2) Radiological Findings:
- Chest X-ray and CT scan typically show bilateral diffuse infiltrates, often described as "ground-glass opacities".
- In severe cases, pleural effusions or cystic changes may be observed.

(3) Microbiological Diagnosis:
- Sputum analysis can be performed, but it often yields poor sensitivity.
- Bronchoalveolar lavage (BAL) or induced sputum is the preferred method for collecting samples. These samples can be analyzed using:
- Giemsa stain, silver stain, or direct fluorescence antibody testing to identify the organism.
- PCR (Polymerase Chain Reaction) can also be used for more sensitive detection.

(4) Blood Tests:
- Arterial blood gases (ABG) may show hypoxemia due to impaired gas exchange.
- Lactate dehydrogenase (LDH) is often elevated in PCP, though it is not specific.
- In HIV-infected patients, a low CD4 count (¡200 cells/mm^3) is a major risk factor for PCP.

Step 3: Treatment of Pneumocystis Pneumonia.

(1) First-Line Therapy: - Trimethoprim-sulfamethoxazole (TMP-SMX) is the first-line treatment for PCP and should be initiated as soon as the diagnosis is suspected. The standard regimen is:
- 15-20 mg/kg/day of TMP and 75-100 mg/kg/day of SMX, divided into 3-4 doses per day for 21 days. - TMP-SMX is effective and widely available, but can cause side effects such as rash, fever, and elevated liver enzymes.
- If the patient is unable to tolerate TMP-SMX, alternative therapies include:
- Pentamidine (IV or aerosolized).
- Atovaquone or Clindamycin + Primaquine.
(2) Adjuvant Therapy: - Corticosteroids are recommended in moderate to severe cases of PCP, particularly for patients with hypoxemia (PaO_2 ¡ 70 mmHg) or high LDH levels.
- Prednisone (40 mg twice daily for 5 days, then tapering) helps reduce inflammation and immune-mediated damage.

(3) Oxygen Therapy:
- Supplemental oxygen is administered to maintain oxygen saturation (SpO_2 ¿ 92%) and to prevent respiratory failure.
- In severe cases, mechanical ventilation or non-invasive positive pressure ventilation (NIPPV) may be required.

Quick Tip

Early diagnosis and initiation of therapy with TMP-SMX is crucial in managing PCP. Always consider the need for corticosteroids in severe cases to reduce inflammation.

Q2. b) Treatment of pulmonary non-tuberculous mycobacterial (NTM) disease. [5]

Solution:

Step 1: Introduction to Pulmonary Non-Tuberculous Mycobacterial (NTM) Disease.
Non-tuberculous mycobacteria (NTM) are a group of mycobacterial organisms that are found in the environment and can cause pulmonary infections, especially in individuals with pre-existing lung disease (e.g., chronic obstructive pulmonary disease (COPD), cystic fibrosis, or bronchiectasis) or those who are immunocompromised. NTM infections are often difficult to diagnose and treat, requiring a combination of antibiotics and sometimes surgical intervention.

Common NTM species that cause pulmonary disease include:
- Mycobacterium avium complex (MAC).
- Mycobacterium abscessus.
- Mycobacterium kansasii.
- Mycobacterium fortuitum.

Step 2: Diagnosis of NTM Disease.
- Sputum culture is essential for diagnosing NTM, and multiple sputum samples should be obtained to identify the species.
- Chest X-ray and CT scan often reveal nodular or cavitary lung lesions, which can be confused with tuberculosis or fungal infections.
- Bronchoscopy with lavage may be necessary if sputum cultures are negative but suspicion remains high.
- Blood tests may be performed to assess immune function and to monitor the response to treatment.
- Molecular methods (PCR, DNA probes) can be used to identify specific NTM species more rapidly than culture.

Step 3: Treatment of Pulmonary NTM Disease.

(1) First-Line Therapy:
- The treatment of pulmonary NTM involves a combination of antibiotics. The choice of antibiotics depends on the species of NTM identified. Commonly used antibiotics include:
- MAC infections: The regimen typically includes clarithromycin or azithromycin, rifampin or rifabutin, and ethambutol.
- M. abscessus: Treatment includes amikacin, imipenem, and clarithromycin.
- M. kansasii: A combination of isoniazid, rifampin, and ethambutol is often used.

(2) Treatment Duration:
- Long-term therapy is usually required, typically 12 months or longer, until cultures are negative for NTM and the patient is clinically stable.
- It is important to assess for drug resistance, especially in the case of M. abscessus, which can be resistant to many antibiotics.

(3) Adjunctive Therapy:
- Surgical resection may be necessary in cases where the infection is localized to a specific lung region and does not respond to antibiotics.
- Nutritional support and respiratory physiotherapy are important components of supportive care.

(4) Monitoring:
- Regular monitoring of sputum cultures, chest imaging, and drug side effects is essential throughout the course of treatment.
- Adjust therapy based on the microbiological results and clinical response.

Quick Tip

Long-term, multidrug therapy is essential for treating NTM infections. Early diagnosis, species identification, and monitoring of response to treatment are key to successful outcomes.

Q3. a) Management of empyema. [5]

Solution:

Step 1: Introduction to Empyema.
Empyema is the collection of pus in the pleural cavity that usually results from infection in the lung, commonly following pneumonia, thoracic surgery, or trauma. It is characterized by the accumulation of inflammatory exudate, which can cause lung compression, respiratory failure, and sepsis if not managed appropriately. The condition typically evolves through three stages: the exudative stage, the fibropurulent stage, and the organized stage.

Step 2: Clinical Features of Empyema.
- Symptoms: The most common symptoms of empyema include:
- Fever, cough, and pleuritic chest pain.
- Dyspnea and tachypnea.
- Malaise and weight loss in advanced cases. - Signs: On examination, signs may include:
- Decreased breath sounds on the affected side.
- Dullness to percussion.
- Increased tactile fremitus.

Step 3: Diagnosis of Empyema.
(1) Chest X-ray: Typically shows a blunting of the costophrenic angle and a pleural effusion. In later stages, loculated fluid may be observed.
(2) CT scan: Can identify the presence of loculated fluid collections and assess the extent of the disease.
(3) Thoracentesis: Involves aspirating pleural fluid to assess the presence of pus. Pleural fluid analysis typically shows:
- High white blood cell count, with predominantly neutrophils.
- Low glucose levels (due to bacterial consumption).
- High LDH (lactate dehydrogenase) and protein levels.
(4) Microbiological Culture: Fluid culture can identify the causative pathogen. Gram stain and culture are essential to identify whether the infection is bacterial (e.g., Streptococcus pneumoniae, Staphylococcus aureus) or tuberculous.

Step 4: Management of Empyema.
(1) Antibiotics: The choice of antibiotics should be based on the etiological agent identified from cultures or empirical therapy until results are available. Empiric treatment typically involves:
- Broad-spectrum antibiotics such as ceftriaxone or piperacillin-tazobactam for community-acquired infections.
- For methicillin-resistant Staphylococcus aureus (MRSA), consider vancomycin or linezolid.
- Tuberculosis should be suspected in high-risk patients, and anti-tuberculous therapy (e.g., rifampin, isoniazid, ethambutol, pyrazinamide) should be started if indicated.
(2) Drainage: Effective drainage is key to the management of empyema. Options include:
- Thoracentesis for diagnostic purposes.
- Chest tube insertion for continuous drainage of pus in the fibropurulent stage.
- Video-assisted thoracoscopic surgery (VATS) or thoracotomy may be required in organized empyema to remove loculated fluid and adhesions. (3) Surgical Intervention: If the pleural effusion becomes organized and does not respond to drainage, surgical intervention like VATS or decortication may be required to remove fibrous tissue and restore lung expansion.
(4) Supportive Care: This includes oxygen therapy, nutritional support, and pain management.

Quick Tip

Early diagnosis and aggressive management with antibiotics and drainage are crucial to prevent progression to chronic empyema and to avoid the need for surgical intervention.

Q3. b) Management of tuberculosis in patient with renal disease. [5]

Solution:

Step 1: Introduction to Tuberculosis (TB) in Renal Disease.
Tuberculosis (TB) is a contagious infection caused by Mycobacterium tuberculosis that primarily affects the lungs but can involve other organs, including the kidneys. Renal disease in

the context of TB can result in renal TB, renal failure, or complicate the management of TB in patients with chronic kidney disease (CKD) or those on dialysis. Patients with renal disease may be at higher risk of developing drug-induced nephrotoxicity from anti-TB medications. Managing TB in these patients requires careful selection and monitoring of anti-TB drugs.

Step 2: Diagnosis of Renal TB.
- Clinical Features: Patients with renal TB may present with flank pain, hematuria, pyuria, or abdominal masses. - Urinary Analysis: Typically shows sterile pyuria, hematuria, and acidic urine. - Imaging: Ultrasonography or CT scan may reveal hydronephrosis, renal calcification, or perinephric abscesses. - Urine Culture: Diagnosis is confirmed by urine culture or PCR for M. tuberculosis. Mycobacterial urine cultures may take weeks to return results, so empiric therapy is often initiated.

Step 3: Management of Tuberculosis in Renal Disease.

(1) Anti-TB Therapy: - The standard treatment for renal TB follows the same principles as for pulmonary TB, consisting of a combination of first-line anti-TB drugs:
- Isoniazid, rifampin, pyrazinamide, and ethambutol for the initial phase of treatment.
- After 2 months of the intensive phase, rifampin and isoniazid are continued for 4-7 months in the continuation phase. - Adjustments for Renal Disease: In patients with chronic kidney disease (CKD), the dosages of some anti-TB drugs may need to be adjusted due to altered pharmacokinetics and the risk of drug toxicity. For example:
- Isoniazid and rifampin do not require dose adjustments, but pyrazinamide should be used with caution in renal failure, and its dosage may need to be reduced or discontinued if renal function worsens.
- Ethambutol requires dose reduction in patients with renal insufficiency to avoid optic neuropathy.

(2) Monitoring for Toxicity:
- Renal Function Monitoring: Regular monitoring of serum creatinine and urine output is crucial to detect renal toxicity due to anti-TB drugs, especially pyrazinamide and ethambutol.
- Hepatic Monitoring: Liver enzymes should also be monitored, as hepatotoxicity is a known side effect of rifampin and isoniazid.

(3) Dialysis Considerations: - Hemodialysis: In patients on hemodialysis, rifampin and isoniazid are typically dialyzed out, so additional doses may be required.
- Peritoneal Dialysis: Anti-TB medications may require adjustment in patients on peritoneal dialysis to avoid toxicity.

(4) Adjunctive Measures: - For patients with advanced renal disease, renal transplant may be considered once TB is well-controlled and the patient is in remission, though active TB infection is a contraindication for transplantation.

Quick Tip

Adjust anti-TB medications in patients with renal disease to prevent nephrotoxicity. Regular monitoring of renal and hepatic function is essential throughout treatment.

Q4. a) Diagnosis of the bronchiectasis. [5]

Solution:

Step 1: Introduction to Bronchiectasis.
Bronchiectasis is a chronic respiratory condition characterized by the irreversible dilation and destruction of the bronchi, leading to a vicious cycle of infection, inflammation, and airway damage. It can result from various underlying causes, including infections, immune deficiencies, or genetic disorders such as cystic fibrosis. It is commonly associated with chronic cough, purulent sputum, and recurrent respiratory infections.

Step 2: Clinical Features of Bronchiectasis.
- Chronic cough with purulent sputum is the hallmark of bronchiectasis. The sputum may be green or yellow and often has a foul odor due to bacterial colonization.
- Hemoptysis (coughing up blood) may occur, especially in advanced stages of the disease.
- Patients often experience dyspnea, especially with exertion, and may have a history of recurrent respiratory infections.
- Wheezing and crackles on chest auscultation may be heard in more severe cases.

Step 3: Diagnosis of Bronchiectasis.

(1) Clinical History:
- A thorough history should be obtained, including any prior respiratory infections, history of recurrent pneumonia, and any underlying conditions such as cystic fibrosis, immune deficiency, or autoimmune disorders.
- Family history of respiratory diseases or cystic fibrosis should be explored.

(2) Imaging Studies:
- Chest X-ray: A Chest X-ray may show increased lung markings, ring shadows, or tram-track opacities, suggestive of bronchial wall thickening. However, high-resolution CT (HRCT) of the chest is the gold standard for diagnosis.
- HRCT scan reveals characteristic findings such as:
- Bronchial dilation (larger than the adjacent pulmonary arteries).
- Bronchial wall thickening.
- Air trapping and mucus plugging in the affected areas.
- In severe cases, cystic changes or "signet ring" sign can be observed, where the dilated bronchus appears to encircle the accompanying blood vessel.

(3) Pulmonary Function Tests (PFTs):
- Spirometry often shows a mixed obstructive and restrictive pattern with reduced FEV1 (forced

expiratory volume in 1 second) and FVC (forced vital capacity).
- Reduced FEV1/FVC ratio indicates airway obstruction, while decreased TLC (total lung capacity) and RV (residual volume) suggest a restrictive component.

(4) Microbiological Tests:
- Sputum culture is essential to identify the causative organisms, particularly Pseudomonas aeruginosa, Haemophilus influenzae, or Staphylococcus aureus.
- Blood tests may reveal elevated C-reactive protein (CRP), white blood cell count, and immunoglobulin levels to check for associated immune deficiencies.

(5) Additional Investigations:
- Sweat chloride test is recommended if cystic fibrosis is suspected, especially in younger patients.
- Bronchoscopy may be performed to assess the extent of disease and to clear any obstructed airways.

Quick Tip

HRCT is the gold standard for diagnosing bronchiectasis. Sputum culture helps identify the causative pathogens, and early identification of underlying causes is crucial for targeted therapy.

Q4. b) Management for inoperable patients of mycetoma lung. [5]

Solution:

Step 1: Introduction to Mycetoma Lung.
Mycetoma is a chronic, granulomatous infection typically caused by fungi (eumycetoma) or bacteria (actinomycetoma). The infection usually affects the skin and soft tissues but can spread to deeper structures like the bones and lungs. Mycetoma lung refers to the involvement of the lungs in this infection, typically presenting with chronic cough, hemoptysis, and respiratory distress. It is a rare condition, often occurring in patients with untreated or poorly managed mycetoma.
Step 2: Clinical Features of Mycetoma Lung.
- Patients may present with symptoms like persistent cough, expectoration of sputum, hemoptysis, and weight loss.
- Chest pain and dyspnea may occur in more advanced cases due to pleural involvement or increased inflammation in the lungs.
- Fungal mycetoma typically presents with swelling and discharge of pus from sinus tracts in the skin, and actinomycetoma presents similarly with abscesses and sinus formation.

Step 3: Diagnosis of Mycetoma Lung.
- Chest X-ray and CT scan may show cavitary lesions, masses, and pleural effusions, with characteristics similar to other chronic pulmonary infections.

- Microbiological confirmation can be done by sputum culture, skin biopsy, or tissue biopsy for fungal or bacterial identification. PCR and histopathology are also useful for confirming the diagnosis.

Step 4: Management of Inoperable Mycetoma Lung.

(1) Antifungal Therapy:
- For eumycetoma (fungal mycetoma), treatment typically involves long-term antifungal therapy with agents such as:
- Itraconazole or terbinafine as first-line therapy.
- Amphotericin B may be considered in severe cases, especially if the infection is disseminated or resistant to other treatments.
- Treatment duration may range from 6 months to 2 years, depending on the severity of the disease and response to therapy.

(2) Antibiotic Therapy:
- For actinomycetoma, surgical drainage is generally not an option, so long-term antibiotics are necessary. Options include:
- Sulfamethoxazole-trimethoprim (SMX-TMP) and doxycycline for Nocardia or Streptomyces infections.
- Amikacin and gentamicin for resistant strains.
- Treatment typically continues for 6-12 months, based on clinical response.

(3) Supportive Care:
- Patients may require oxygen therapy in cases of respiratory distress or hypoxemia.
- Analgesics and anti-inflammatory drugs may be used to control symptoms and improve the patient's quality of life.

(4) Surgical Interventions (if possible):
- Although surgical resection is often not feasible in cases of mycetoma lung, in patients with localized disease and if they are good surgical candidates, partial lung resection or lobectomy may be considered to control the infection.

(5) Follow-up and Monitoring:
- Regular follow-up is essential to monitor for disease progression, response to therapy, and development of complications such as secondary infections or pulmonary fibrosis.

Quick Tip

Long-term antifungal or antibiotic therapy is crucial in managing mycetoma lung. Monitoring for adverse effects of drugs and managing complications are key to improving outcomes in these patients.

Q5. a) Radiological features of lung hydatid disease. [5]

Solution:

Step 1: Introduction to Lung Hydatid Disease.
Hydatid disease, also known as echinococcosis, is a parasitic infection caused by the tapeworm Echinococcus granulosus. The infection most commonly affects the liver and lungs, with the lungs being the second most frequent organ affected. The disease results from the formation of cystic lesions that grow within the affected organ, leading to various complications such as rupture, secondary infection, and anaphylaxis.
Step 2: Clinical Features of Lung Hydatid Disease.
- Symptoms: In the early stages, lung hydatid disease may be asymptomatic. As the cysts grow, patients may experience:
- Cough, which may be productive or non-productive.
- Chest pain (especially if the cyst ruptures or causes pleural irritation).
- Dyspnea or wheezing.
- In advanced cases, hemoptysis (coughing up blood) may occur, especially if there is cyst rupture.
- Signs: On physical examination, signs may include decreased breath sounds over the affected area and signs of pleural effusion if the cyst causes fluid accumulation.
Step 3: Radiological Features of Lung Hydatid Disease.

(1) Chest X-ray:
- Lesions on chest X-ray are usually well-defined, round, or oval, and may show cystic shadows with air-fluid levels in the case of cyst rupture or infection. The right lung is more commonly affected than the left.
- As the disease progresses, multiple cysts may develop, and the lung fields may show opaque masses. Calcification of the cyst wall may occur in chronic cases, presenting as a ring-shaped opacity.
- Pleural effusion may be noted on X-ray, especially if the cyst ruptures and leaks fluid into the pleural space.

(2) CT Scan:
- CT scan is the gold standard for diagnosing lung hydatid disease. It provides detailed images of cysts and their effects on lung tissue. Key features include:
- Single or multiple cysts with well-defined borders.
- Cyst within the lung parenchyma with a thin, smooth wall.
- The cyst may show an air-fluid level if there is rupture or secondary infection.
- In more advanced cases, complicated cysts may show thickened walls, calcifications, or cyst rupture.
- Hydatid cysts may also cause compression atelectasis or bronchial displacement.

(3) MRI:
- MRI may be used in some cases to evaluate the cysts, particularly when there are concerns about cyst rupture or involvement of adjacent structures like the diaphragm or mediastinum. It provides good soft tissue contrast and can help delineate the extent of the disease.

(4) Ultrasound:
- Ultrasonography is typically used for abdominal hydatid disease but can also detect pleural

effusions or cystic lesions in the chest, especially if there is any fluid involvement. It can assist in determining whether the cyst is simple or complicated.

Quick Tip

CT scans are the most effective tool for diagnosing lung hydatid disease, providing detailed imaging of cysts, their location, and associated complications.

Q5. b) Management for severe exacerbations of COPD. [5]

Solution:

Step 1: Introduction to Severe Exacerbations of COPD.
A severe exacerbation of chronic obstructive pulmonary disease (COPD) is defined as a worsening of respiratory symptoms, including increased dyspnea, cough, and sputum production, that requires medical intervention. Severe exacerbations can lead to significant morbidity and mortality, particularly in patients with advanced COPD. Management focuses on improving oxygenation, reducing inflammation, and preventing further deterioration.

Step 2: Clinical Features of Severe Exacerbations of COPD.
- Symptoms: Patients typically present with:
- Severe dyspnea that is worsening or unresponsive to home treatments.
- Increased sputum production, which may be purulent.
- Coughing and wheezing.
- Cyanosis and fatigue due to inadequate oxygenation.
- Signs: On examination, patients may have:
- Tachypnea and tachycardia.
- Use of accessory muscles during respiration.
- Hypoxemia, as demonstrated by pulse oximetry or arterial blood gases (ABG).

Step 3: Management of Severe Exacerbations of COPD.

(1) Oxygen Therapy:
- Supplemental oxygen should be administered to maintain SpO_2 ¿ 88% and PaO_2 ¿ 60 mmHg. Careful monitoring is essential, especially in patients with hypercapnia (elevated CO_2 levels), as excessive oxygenation may worsen respiratory acidosis.
- Use low-flow oxygen systems (nasal cannula or simple face mask) or non-invasive ventilation (NIV) to avoid excessive CO_2 retention.

(2) Bronchodilators:
- Short-acting bronchodilators such as albuterol (beta-agonists) and ipratropium (anticholinergics) are given via nebulizer or metered-dose inhaler (MDI) to relieve bronchospasm and improve airflow. These should be given every 1-2 hours during the exacerbation.

(3) Corticosteroids:
- Systemic corticosteroids such as prednisone (40 mg daily) are essential in reducing airway inflammation. Treatment typically lasts for 5-7 days. These medications help reduce the severity of the exacerbation and improve lung function.

(4) Antibiotics:
- Antibiotics are indicated in the presence of purulent sputum and worsening symptoms, especially if there is a history of bacterial infections. Common agents include:
- Amoxicillin-clavulanate, azithromycin, or levofloxacin for empiric therapy.
- The choice of antibiotic may be adjusted based on sputum culture results.

(5) Non-invasive Ventilation (NIV):
- Non-invasive positive pressure ventilation (NIV), such as BiPAP or CPAP, is used in patients with respiratory failure (e.g., hypercapnic respiratory failure). NIV helps improve ventilation by assisting with CO_2 elimination and reducing the work of breathing. It is beneficial in preventing the need for endotracheal intubation and mechanical ventilation.

(6) Intubation and Mechanical Ventilation:
- If NIV is unsuccessful or the patient is in severe respiratory failure, endotracheal intubation and mechanical ventilation may be required.

Quick Tip

Early and aggressive treatment of severe COPD exacerbations with oxygen therapy, bronchodilators, corticosteroids, and antibiotics is essential to avoid complications and improve patient outcomes.

Q6. a) Tropical pulmonary eosinophilia. [5]

Solution:

Step 1: Introduction to Tropical Pulmonary Eosinophilia (TPE).
Tropical pulmonary eosinophilia (TPE) is a parasitic lung disease caused by Wuchereria bancrofti or Brugia malayi, which are filarial worms transmitted through the bite of infected mosquitoes. TPE is characterized by eosinophilic inflammation in the lungs and is typically seen in individuals living in tropical regions where lymphatic filariasis is endemic. It primarily affects adults, but children can also be affected.
Step 2: Clinical Features of Tropical Pulmonary Eosinophilia.
- Cough (often persistent and dry), wheezing, and dyspnea (shortness of breath) are common symptoms.
- Fever is often present, and patients may report night sweats or fatigue.
- Chest tightness and pain may occur due to lung inflammation.
- Eosinophilia in the peripheral blood is characteristic, with marked increase in eosinophils (often ¿3000 cells/mm^3).

- Pulmonary infiltrates may be seen on imaging, and these are typically patchy or bilateral with a subpleural distribution.

Step 3: Diagnosis of Tropical Pulmonary Eosinophilia.
(1) Clinical suspicion: A history of residence in an endemic area and symptoms like persistent cough, dyspnea, and eosinophilia in the blood may raise suspicion.
(2) Peripheral blood eosinophilia: A hallmark feature of TPE is a marked increase in eosinophils. This can be confirmed by routine blood tests.
(3) Immunodiagnostic tests: Detection of IgG4 antibodies specific to filarial antigens or circulating filarial antigens in the blood confirms infection with filarial worms.
(4) Chest X-ray: May show bilateral infiltrates with peripheral distribution, indicative of eosinophilic inflammation.
(5) Lung biopsy: Rarely required, but histopathological examination may reveal eosinophilic granulomas and filarial microfilariae.
(6) Ultrasound or PCR: These may help in identifying microfilariae or confirming filarial infection.

Step 4: Treatment of Tropical Pulmonary Eosinophilia.

(1) Antifilarial Therapy: - The mainstay of treatment for TPE is the use of diethylcarbamazine (DEC), which is effective against the filarial worms. DEC is given for 12-21 days, typically in doses of 6 mg/kg/day.
- If DEC is not available or tolerated, ivermectin or albendazole may also be used as alternative treatments.
- Corticosteroids may be used to manage severe eosinophilic inflammation and lung injury (e.g., prednisolone 20-40 mg daily for a few weeks).

(2) Symptomatic Treatment: - Bronchodilators like beta-agonists (e.g., salbutamol) and antihistamines can help relieve wheezing and cough.
- Oxygen therapy may be necessary in severe cases with significant respiratory compromise.
- Antipyretics like paracetamol can help manage fever and general malaise.

Quick Tip

Early diagnosis and treatment with DEC are essential for managing TPE. Corticosteroids may be added in severe cases to control inflammation.

Q6. b) Treatment of pulmonary cystic echinococcosis. [5]

Solution:

Step 1: Introduction to Pulmonary Cystic Echinococcosis.
Pulmonary cystic echinococcosis (CE) is a parasitic disease caused by the larval stage of the Echinococcus granulosus tapeworm, typically transmitted by contact with infected animals

(e.g., dogs, sheep). The disease results in the formation of hydatid cysts in various organs, including the lungs, liver, and spleen. Pulmonary involvement is the second most common after hepatic involvement, and it can cause cough, hemoptysis, and dyspnea. In some cases, rupture of the cyst can lead to anaphylaxis and severe complications.

Step 2: Clinical Features of Pulmonary Cystic Echinococcosis.

- Symptoms depend on the size and location of the cyst but typically include:
- Cough, which may be productive or associated with hemoptysis.
- Chest pain (pleuritic or non-pleuritic).
- Dyspnea, especially if the cyst is large or causing compression of lung tissue.
- In more advanced cases, ruptured cysts may lead to anaphylactic shock, pneumothorax, or pleural effusion.

Step 3: Diagnosis of Pulmonary Cystic Echinococcosis.

(1) Clinical Suspicion: The diagnosis is suspected in patients with a history of exposure to animals, particularly in endemic areas, and symptoms of chronic cough, hemoptysis, and dyspnea.
(2) Imaging:
- Chest X-ray: Shows well-defined, round cystic lesions in the lung, often with calcified walls in chronic cases.
- CT scan or MRI provides more detailed imaging of the hydatid cysts, showing characteristic cystic structures with a fluid-fluid level, indicative of cyst contents. It can also identify any complications, such as rupture or secondary infection.
(3) Serological Tests:
- ELISA (enzyme-linked immunosorbent assay) or indirect hemagglutination test (IHA) can detect Echinococcus-specific antibodies, supporting the diagnosis.
(4) Aspiration and Cyst Examination:
- Aspiration of the cyst fluid can be performed for diagnosis, but careful technique is required to avoid cyst rupture and spillage of infectious material.
- PCR can be used to identify Echinococcus DNA in cyst fluid.

Step 4: Treatment of Pulmonary Cystic Echinococcosis.

(1) Surgical Treatment: - Surgical resection remains the mainstay of treatment, particularly for uncomplicated cysts. The goal is to remove the cyst while avoiding rupture and spillage of cyst contents.
- Cystectomy (removal of the cyst wall) is commonly performed, and in some cases, lung resection (wedge resection or lobectomy) may be necessary if the cyst is large or involves critical lung structures.
- Preoperative precautions include aspiration of cysts with a sterile technique to prevent anaphylactic reactions or cyst rupture.

(2) Medical Treatment: - Albendazole or mebendazole, both anti-helminthic drugs, are used as adjunctive therapy to shrink cysts and prevent recurrence. These are particularly important for inoperable cysts or when surgery is not feasible.
- Albendazole is typically given at a dose of 10-15 mg/kg/day for 28 days, and the treatment may be repeated.
- Praziquantel may also be used in some cases in combination with albendazole to treat cysts

that are resistant to standard therapy.

(3) Percutaneous Treatment: - In inoperable patients, percutaneous aspiration, injection, and re-aspiration (PAIR) technique may be used, where the cyst is aspirated, followed by the injection of a sclerosing agent (e.g., hypertonic saline or absolute ethanol) to kill the cyst.
- This technique is less invasive but carries a higher risk of complications, including cyst rupture and secondary infection.

(4) Postoperative and Long-term Care:
- Post-surgery, antibiotic prophylaxis and anti-parasitic therapy should be continued. Follow-up imaging is crucial to monitor for recurrence.
- Long-term monitoring includes annual chest X-rays or CT scans to detect any new cysts or recurrences.

Quick Tip

For pulmonary cystic echinococcosis, surgical resection is the preferred treatment, with anti-parasitic therapy used as adjunctive treatment or for inoperable cases. Careful management is required to avoid complications like cyst rupture.

Q7. a) Molecular targeted therapy in advanced non-small-cell lung cancer. [5]

Solution:

Step 1: Introduction to Molecular Targeted Therapy in Advanced Non-Small-Cell Lung Cancer (NSCLC).
Non-small-cell lung cancer (NSCLC) is the most common type of lung cancer, accounting for about 85% of all cases. Molecular targeted therapies are a newer class of treatments that target specific molecular changes within cancer cells, which are responsible for cancer growth and progression. These therapies are typically used in advanced stages of NSCLC, particularly when the cancer is metastatic or when chemotherapy is no longer effective.
Step 2: Types of Molecular Targeted Therapies in NSCLC.

(1) Epidermal Growth Factor Receptor (EGFR) Inhibitors:
- EGFR is a receptor that is often overexpressed in NSCLC cells. In patients with EGFR mutations (most commonly exon 19 deletions or L858R point mutations), EGFR tyrosine kinase inhibitors (TKIs) are effective. These include:
- Erlotinib, Gefitinib, and Afatinib.
- These agents work by inhibiting the activity of EGFR, thereby blocking downstream signaling that promotes cancer cell growth.

(2) Anaplastic Lymphoma Kinase (ALK) Inhibitors:
- In ALK-positive NSCLC, typically found in younger, non-smokers, ALK inhibitors such as Crizotinib, Ceritinib, and Alectinib are used.

- These drugs target the ALK gene rearrangements, preventing the fusion protein from promoting tumor growth.

(3) Programmed Death-1 (PD-1) and Programmed Death Ligand-1 (PD-L1) Inhibitors:
- Immune checkpoint inhibitors like Pembrolizumab and Nivolumab target the PD-1/PD-L1 pathway, which cancer cells use to evade the immune system. These agents work by reactivating T-cells to attack cancer cells.
- Pembrolizumab is used in cases where PD-L1 expression is high, and it has shown significant benefit in advanced-stage NSCLC.

(4) Vascular Endothelial Growth Factor (VEGF) Inhibitors:
- Bevacizumab is a monoclonal antibody that inhibits VEGF, preventing the formation of new blood vessels (angiogenesis) that tumors need to grow. It is used in combination with chemotherapy for certain NSCLC cases.

(5) BRAF Inhibitors:
- In BRAF V600E-mutant NSCLC, BRAF inhibitors like Dabrafenib and Trametinib are used. These agents target the mutated BRAF gene, which is involved in cancer cell growth.

Step 3: Benefits and Challenges of Molecular Targeted Therapy in NSCLC.
- Benefits: - Targeted therapies tend to be more effective with fewer side effects compared to traditional chemotherapy.
- These therapies can offer significant survival benefits and improve quality of life for patients with advanced or metastatic disease.
- Challenges: - Development of resistance to these therapies over time, often due to secondary mutations.
- These therapies are often expensive and may require genetic testing to identify the specific mutations.

Quick Tip

Molecular targeted therapies, such as EGFR inhibitors and ALK inhibitors, offer personalized treatment for advanced NSCLC based on specific genetic mutations, leading to better outcomes with fewer side effects.

Q7. b) Treatment of sarcoidosis in patients who progress on or cannot use methotrexate. [5]

Solution:

Step 1: Introduction to Sarcoidosis and Methotrexate.
Sarcoidosis is a systemic inflammatory disease characterized by the formation of granulomas (clusters of immune cells) in various organs, most commonly in the lungs, lymph nodes, and skin. The cause of sarcoidosis is unknown, but it is thought to involve an immune response triggered by an environmental or infectious agent. Methotrexate (MTX) is commonly used to

treat sarcoidosis, especially when patients have progressive or symptomatic disease. However, in cases where patients cannot tolerate MTX or do not respond to it, alternative treatments need to be considered.

Step 2: First-Line and Alternative Treatments for Sarcoidosis.

(1) Corticosteroids (Prednisone):
- Corticosteroids, such as prednisone, remain the first-line treatment for active sarcoidosis. They reduce inflammation and granuloma formation.
- A typical regimen for mild-to-moderate sarcoidosis is prednisone 20-40 mg/day for 4-6 weeks, followed by gradual tapering.
- High-dose corticosteroids may be required for severe or acute presentations, such as in cardiac, neurological, or ocular involvement.

(2) Immunosuppressive Agents:
- Methotrexate is commonly used when corticosteroids are not effective or cause intolerable side effects. If a patient cannot use methotrexate, other immunosuppressive agents include:
- Azathioprine or mycophenolate mofetil for patients with chronic or multisystem involvement.
- Leflunomide or cyclophosphamide may be used in refractory sarcoidosis or cases with significant organ damage.

(3) Anti-TNF Therapy:
- TNF inhibitors like Infliximab or Adalimumab are increasingly used in refractory sarcoidosis cases, particularly when there is pulmonary or cutaneous involvement. These biologics are effective in controlling granulomatous inflammation and improving symptoms.

(4) Hydroxychloroquine:
- For cutaneous sarcoidosis or sarcoid arthritis, hydroxychloroquine can be used, especially in cases that are unresponsive to corticosteroids or when patients are unable to tolerate other therapies.

(5) Other Therapies:
- For neurological or cardiac sarcoidosis, immunosuppressive agents (such as azathioprine or cyclophosphamide) may be used in conjunction with anti-TNF therapy to manage inflammation.
- Biologics are generally reserved for more severe cases that fail conventional therapies.

Step 3: Monitoring and Side Effects of Therapy.
- Monitoring: Patients on long-term corticosteroids or immunosuppressive agents should be monitored for infection, liver function, renal function, and bone health.
- Side effects: Corticosteroids can cause weight gain, hyperglycemia, osteoporosis, and hypertension, while methotrexate can cause hepatotoxicity, bone marrow suppression, and pulmonary toxicity. Regular blood tests are necessary to monitor for these adverse effects.

Quick Tip

For patients who progress on or cannot use methotrexate, corticosteroids, azathioprine, and anti-TNF therapy are key alternatives. Regular monitoring is crucial to prevent adverse effects.

Q8. a) Sleep disorders related to abnormalities in REM sleep. [5]

Solution:

Step 1: Introduction to REM Sleep and Sleep Disorders.
Rapid Eye Movement (REM) sleep is a phase of the sleep cycle that is characterized by rapid eye movements, muscle atonia (muscle paralysis), vivid dreaming, and increased brain activity similar to wakefulness. REM sleep plays a critical role in memory consolidation, emotional regulation, and overall health. Abnormalities in REM sleep can lead to several sleep disorders. The most notable disorders related to abnormalities in REM sleep include:
Step 2: Sleep Disorders Related to REM Sleep Abnormalities.

(1) REM Sleep Behavior Disorder (RBD):
- Definition: RBD is characterized by vivid dreaming with violent movements during sleep, where patients may act out their dreams (e.g., kicking, punching, talking). Unlike in normal REM sleep, patients with RBD do not experience muscle atonia and may exhibit complex motor behaviors.
- Etiology: It is often associated with neurodegenerative diseases, such as Parkinson's disease, Lewy body dementia, and multiple system atrophy. It may also occur in isolation without underlying disease.
- Management: Treatment options include clonazepam, which helps suppress abnormal muscle activity, and melatonin, which regulates circadian rhythms and reduces symptom severity.

(2) Narcolepsy with Cataplexy:
- Definition: Narcolepsy is a neurological disorder characterized by excessive daytime sleepiness and sudden, uncontrollable episodes of sleep. Cataplexy, a symptom of narcolepsy, involves sudden muscle weakness triggered by strong emotions (e.g., laughter, anger). Narcolepsy is linked to REM sleep abnormalities, specifically intrusions of REM sleep into wakefulness.
- Etiology: Narcolepsy is often associated with autoimmune destruction of orexin-producing neurons in the hypothalamus, which helps regulate sleep-wake cycles.
- Management: Treatment includes the use of stimulants (e.g., modafinil, amphetamines) to reduce daytime sleepiness, sodium oxybate to improve sleep quality and reduce cataplexy, and antidepressants (e.g., SSRIs, SNRIs) to manage cataplexy.

(3) Sleep Paralysis:
- Definition: Sleep paralysis is a phenomenon where individuals are unable to move or speak upon awakening or falling asleep. This typically occurs during the transition between REM sleep and wakefulness, when the body is still in a state of muscle atonia (paralysis) that normally prevents acting out dreams.

- Etiology: It is commonly seen in individuals with sleep deprivation, irregular sleep schedules, stress, or sleep disorders like narcolepsy.
- Management: Ensuring regular sleep schedules, adequate sleep duration, and reducing stress may help alleviate symptoms. In severe cases, medications like SSRIs or tricyclic antidepressants can be prescribed.

(4) REM Sleep Fragmentation:
- Definition: Fragmentation of REM sleep refers to interruptions in the normal progression and duration of REM sleep cycles, often resulting in poor sleep quality and daytime fatigue. This condition can be caused by sleep apnea, insomnia, or depression.
- Etiology: Conditions like sleep apnea or insomnia can lead to frequent awakenings during the night, disrupting normal REM sleep patterns.
- Management: Treating underlying conditions, such as using CPAP for sleep apnea or cognitive behavioral therapy (CBT) for insomnia, may help reduce REM sleep fragmentation and improve overall sleep quality.

Quick Tip

REM sleep abnormalities, such as RBD, narcolepsy, and sleep paralysis, can significantly affect sleep quality. Early diagnosis and treatment are essential, and therapies like clonazepam and melatonin can help manage symptoms.

Q8. b) Positive airway pressure (PAP) therapy for adults with obstructive sleep apnea (OSA). [5]

Solution:

Step 1: Introduction to Obstructive Sleep Apnea (OSA).
Obstructive sleep apnea (OSA) is a common sleep disorder characterized by repeated episodes of upper airway obstruction during sleep, leading to breathing pauses and hypoxemia. These apneic episodes are often associated with snoring, excessive daytime sleepiness, and poor-quality sleep. OSA is linked to significant health risks, including cardiovascular disease, hypertension, diabetes, and stroke.

Step 2: Mechanism of Positive Airway Pressure (PAP) Therapy.
Positive airway pressure (PAP) therapy is the primary treatment for moderate to severe OSA. PAP works by providing a continuous flow of air through a mask to keep the upper airway open during sleep, preventing apneas and hypopneas. The main types of PAP therapy include:

(1) Continuous Positive Airway Pressure (CPAP):
- CPAP is the most commonly used PAP device for OSA. It delivers a continuous stream of air at a set pressure to maintain airway patency. CPAP is effective in reducing the number of apneas and improving oxygen saturation during sleep.
- Indication: CPAP is recommended for patients with moderate to severe OSA, particularly

those who have frequent apneic episodes and daytime symptoms such as excessive sleepiness and fatigue.

(2) Bilevel Positive Airway Pressure (BiPAP):
- BiPAP delivers two levels of pressure: a higher pressure during inhalation (IPAP) and a lower pressure during exhalation (EPAP). BiPAP is typically used for patients with central sleep apnea, comorbidities, or those who have difficulty tolerating CPAP.
- Indication: BiPAP is indicated for patients with more complex sleep-disordered breathing, such as heart failure or neuromuscular disorders.

(3) Auto-adjusting Positive Airway Pressure (APAP):
- APAP adjusts the pressure automatically throughout the night based on detected changes in airflow and airway resistance. It is particularly useful for patients with variable pressure needs, such as those with mild to moderate OSA.
- Indication: APAP is beneficial for patients who experience fluctuating airway pressures during sleep and need personalized adjustments in their therapy.

Step 3: Benefits of PAP Therapy for OSA.
- Reduction in Apnea-Hypopnea Index (AHI): PAP therapy effectively reduces the number of apneic episodes per hour of sleep, improving overall sleep quality.
- Improved Oxygen Saturation: PAP therapy maintains normal oxygen levels during sleep, reducing the risk of hypoxemia and its associated complications, such as cardiac arrhythmias and hypertension.
- Reduction in Daytime Sleepiness: By improving sleep quality, PAP therapy helps reduce excessive daytime sleepiness, improving cognitive function, mood, and quality of life.
- Cardiovascular Benefits: Regular use of CPAP has been shown to reduce blood pressure and the risk of stroke, heart failure, and myocardial infarction in patients with OSA.

Step 4: Complications and Challenges of PAP Therapy.

(1) Patient Adherence:
- The most significant challenge in PAP therapy is patient adherence. Many patients find wearing the mask uncomfortable, leading to poor compliance. Education, proper mask fitting, and gradual acclimatization can help improve adherence.
- Humidification of the air may be needed to prevent dryness or irritation of the nasal passages.

(2) Side Effects:
- Common side effects of PAP therapy include nasal congestion, dry mouth, skin irritation, and aerophagia (swallowing air).
- In some cases, claustrophobia or discomfort from the mask can lead to therapy discontinuation.

(3) Long-Term Monitoring:
- Ongoing follow-up is necessary to ensure that the therapy is effective. This may include regular checkups, assessment of symptom improvement, and adjustments to air pressure settings.

Quick Tip

PAP therapy, particularly CPAP, is highly effective in managing OSA. However, patient education and proper mask fitting are crucial for improving adherence and minimizing side effects.

Q9. a) Surgical emphysema. [5]

Solution:

Step 1: Introduction to Surgical Emphysema.
Surgical emphysema refers to the presence of air or gas within the subcutaneous tissue of the body, often occurring after trauma or surgical procedures. It can also result from air leakage from the lungs or respiratory tract, which allows air to track into the soft tissues. Surgical emphysema is typically associated with procedures such as thoracic surgery, tracheostomy, or lung biopsy, but it can also occur after blunt or penetrating trauma to the chest.
Step 2: Clinical Features of Surgical Emphysema.
- Palpation: The hallmark sign of surgical emphysema is the feeling of crepitus (a crackling sensation) under the skin, which occurs when air is trapped in the subcutaneous tissue.
- Visual Findings: There may be visible swelling or distention of the skin, particularly in the neck, chest, or face. The affected area may appear puffy or bubbly.
- Respiratory Symptoms: In severe cases, surgical emphysema may be associated with dyspnea (difficulty breathing) or hypoxia if the air tracks into the mediastinum or around the lungs, leading to compression of vital structures like the trachea or major blood vessels.

Step 3: Diagnosis of Surgical Emphysema.
(1) Clinical Examination: The diagnosis of surgical emphysema is usually made based on physical examination, where crepitus can be palpated in the subcutaneous tissues.
(2) Imaging:
- Chest X-ray can reveal pneumomediastinum (air in the mediastinum) and pneumothorax (air in the pleural cavity), which may accompany surgical emphysema.
- CT scan is more sensitive and can identify the extent of air tracking into the chest wall, mediastinum, or neck. It can also help detect any underlying lung injuries or pneumothorax.

Step 4: Treatment of Surgical Emphysema.
- Conservative Management: In many cases, surgical emphysema resolves spontaneously, especially if the air leakage is small or localized. Supportive measures include:
- Observation to monitor for progression or worsening.
- Oxygen therapy may be provided if the patient shows signs of hypoxia or if air in the mediastinum or around the lungs is compressing critical structures.
- Intervention: If the emphysema is severe or causing compression of respiratory structures, interventions include:
- Needle aspiration or chest tube insertion to evacuate air in the pleural cavity or mediastinum.
- Surgical repair may be required if the air leakage is persistent or if there is damage to the lung or chest wall.

Quick Tip

Surgical emphysema often resolves spontaneously, but in severe cases, it can lead to complications like pneumothorax or mediastinal compression. Monitoring and early intervention are key.

Q9. b) Lung carcinoid tumors. [5]

Solution:

Step 1: Introduction to Lung Carcinoid Tumors.
Lung carcinoid tumors are a type of neuroendocrine tumor that arises from the neuroendocrine cells of the lung. These tumors are typically slow-growing and are considered a low-grade form of cancer. They account for 1-2% of all lung cancers and are more commonly found in young adults (usually under 50 years of age), with a slight female predominance. Carcinoid tumors can be classified into two main types:
- Typical carcinoids (80% of cases), which are generally non-aggressive and have a better prognosis.
- Atypical carcinoids (20% of cases), which have more aggressive behavior and a higher tendency to metastasize.

Step 2: Clinical Features of Lung Carcinoid Tumors.
- Symptoms can vary depending on the size and location of the tumor. Common symptoms include:
- Cough, often with bloody sputum (hemoptysis).
- Wheezing and dyspnea due to airway obstruction.
- Chest pain (less common).
- Carcinoid syndrome is a rare but specific manifestation of carcinoid tumors that occurs when the tumor secretes serotonin and other vasoactive substances, leading to symptoms such as:
- Flushing, diarrhea, and wheezing.
- Tachycardia, hypotension, and skin changes.

Step 3: Diagnosis of Lung Carcinoid Tumors.
(1) Imaging Studies:
- Chest X-ray may show a solitary nodule or mass, although lesions may also be peripheral or difficult to detect.
- CT scan provides more detailed images, showing the size, location, and possible bronchial obstruction due to the tumor.
- In carcinoid syndrome, octreotide scintigraphy or PET scans may help detect neuroendocrine tumors.
(2) Bronchoscopy:
- A bronchoscopy can confirm the diagnosis by visualizing endobronchial lesions, which are often polypoid or well-defined. Biopsy can be performed to obtain histological confirmation.

(3) Histopathology:
- Carcinoid tumors are characterized by uniform cells with neuroendocrine features (e.g., salt-and-pepper chromatin). Immunohistochemical staining for chromogranin A, synaptophysin, and CD56 is typically positive in carcinoid tumors.

Step 4: Treatment of Lung Carcinoid Tumors.

(1) Surgical Resection:
- Surgical resection is the treatment of choice for localized tumors, and complete excision provides the best chance for cure.
- Lobectomy or wedge resection may be performed depending on the location and size of the tumor.
- In cases of lymph node involvement, a lymphadenectomy may be necessary.
- Atypical carcinoids may require more extensive surgery due to their aggressive nature.

(2) Chemotherapy and Radiation Therapy:
- Chemotherapy is generally reserved for advanced or metastatic carcinoid tumors, particularly for atypical carcinoids. Common chemotherapeutic agents include etoposide and platinum-based agents.
- Radiation therapy is used in cases where surgical resection is not possible or when local recurrence occurs.

(3) Management of Carcinoid Syndrome:
- Octreotide or lanreotide, somatostatin analogs, are used to control the symptoms of carcinoid syndrome, particularly flushing, diarrhea, and wheezing. These medications inhibit the release of serotonin and other vasoactive substances.
- Interferon-alpha may also be used in the treatment of carcinoid syndrome to reduce symptoms and tumor growth.

Quick Tip

Surgical resection is the most effective treatment for lung carcinoid tumors. Chemotherapy and radiation are options for metastatic or unresectable disease, while somatostatin analogs are helpful in controlling carcinoid syndrome.

Q10. a) Nicotine replacement therapies. [5]

Solution:

Step 1: Introduction to Nicotine Replacement Therapy (NRT).
Nicotine replacement therapy (NRT) is a therapeutic strategy used to help individuals quit smoking by reducing withdrawal symptoms and cravings associated with nicotine addiction. NRT provides a controlled dose of nicotine without the harmful chemicals found in tobacco smoke, which helps the smoker gradually reduce their dependence on nicotine.

Step 2: Types of Nicotine Replacement Therapies.

(1) Nicotine Patches:
- Nicotine patches are applied to the skin and deliver a steady, low dose of nicotine throughout the day. They are generally worn for 16-24 hours and are available in different dosages, with patients typically starting with a higher dose and gradually reducing the dosage over time.
- Advantages: Convenient, once-daily application, provides steady nicotine levels.
- Disadvantages: May cause skin irritation or sleep disturbances if worn at night.

(2) Nicotine Gum:
- Nicotine gum is chewed to release nicotine, helping to relieve cravings and withdrawal symptoms. The gum is available in 2 mg and 4 mg doses, and users can chew a piece when they feel the urge to smoke.
- Advantages: Provides immediate relief from cravings and can be used as needed.
- Disadvantages: Some people may experience jaw discomfort or gum irritation. It can also be difficult for some individuals to use it properly, leading to reduced effectiveness.

(3) Nicotine Lozenges:
- Nicotine lozenges dissolve slowly in the mouth, providing a steady release of nicotine. They come in various strengths (typically 2 mg or 4 mg).
- Advantages: Convenient for those who do not like chewing gum.
- Disadvantages: May cause mouth or throat irritation.

(4) Nicotine Inhalers:
- Nicotine inhalers are devices that allow the user to inhale nicotine vapor. They mimic the hand-to-mouth ritual of smoking, which can help address psychological cravings.
- Advantages: Helps simulate the act of smoking, which can be particularly useful for smokers who miss the behavioral aspects of smoking.
- Disadvantages: Requires use of the device frequently and may cause throat irritation.

(5) Nicotine Nasal Spray:
- Nicotine nasal spray delivers nicotine through the nasal mucosa, providing rapid nicotine absorption. It is available in a metered-dose spray and is used several times a day as needed.
- Advantages: Quick absorption, relieves cravings almost immediately.
- Disadvantages: May cause nasal irritation, sneezing, or watery eyes.

Step 3: Benefits and Challenges of Nicotine Replacement Therapy.
- Benefits: - NRT significantly improves the chances of quitting smoking when used alongside behavioral therapy and support.
- It reduces nicotine withdrawal symptoms and cravings, making it easier for individuals to quit smoking successfully.
- It is safer than smoking and helps reduce the risk of heart disease and lung cancer over time.
- Challenges: - NRT does not address the behavioral and psychological aspects of smoking addiction, so it is often most effective when combined with counseling or other smoking cessation programs.
- Some individuals may misuse NRT products, continuing to use them for longer than necessary, leading to prolonged nicotine dependence.

Step 4: Monitoring and Cessation Strategies.
- Gradual Reduction: The goal of NRT is to gradually reduce dependence on nicotine. The dosage should be tapered down over time to eventually reach nicotine-free status.
- Behavioral Support: Combining NRT with counseling or support groups can improve the success rate of quitting smoking.

Quick Tip

Nicotine replacement therapies are effective in managing withdrawal symptoms and cravings, but combining NRT with behavioral therapy can improve the chances of successfully quitting smoking.

Q10. b) Treatment of sarcoidosis in patients who progress on or cannot use methotrexate. [5]

Solution:

Step 1: Introduction to Sarcoidosis and Methotrexate.
Sarcoidosis is a systemic granulomatous disease that commonly affects the lungs, lymph nodes, and skin. It is characterized by the formation of non-caseating granulomas in affected organs. Methotrexate (MTX) is frequently used for the treatment of progressive or symptomatic sarcoidosis, especially when corticosteroids are ineffective or lead to significant side effects. However, in some patients, MTX may not be effective, or they may experience adverse effects that prevent its use.
Step 2: First-Line Treatment for Sarcoidosis.
- Corticosteroids are typically the first-line treatment for sarcoidosis. Prednisone is commonly used at a dose of 20-40 mg/day, with a gradual tapering based on clinical response. However, corticosteroids may cause significant side effects when used long-term, such as osteoporosis, hyperglycemia, and weight gain.
Step 3: Alternative Treatments for Sarcoidosis.

(1) Azathioprine (AZA):
- Azathioprine is a purine analog that acts as an immunosuppressant. It is commonly used as an alternative to MTX in patients who cannot tolerate MTX or in those who have unsatisfactory responses.
- The typical dose is 2 mg/kg/day. It is particularly useful in patients with multisystem involvement and those with chronic disease.
- Side effects: Myelosuppression, hepatotoxicity, and gastrointestinal upset. Regular blood monitoring is required.

(2) Mycophenolate Mofetil (MMF):
- Mycophenolate mofetil is an immunosuppressive agent that is used to treat refractory sarcoidosis. It is particularly helpful in cases of cutaneous or neurologic sarcoidosis.
- The typical dose is 1-2 g/day, but it should be adjusted for renal function.

- Side effects: Gastrointestinal distress, myelosuppression, and increased risk of infection.

(3) Anti-TNF Therapy:
- Tumor necrosis factor-alpha (TNF-α) inhibitors such as Infliximab and Adalimumab have been shown to be effective in patients with refractory sarcoidosis. These biologic agents work by inhibiting the TNF-α pathway, reducing inflammation and granuloma formation.
- Indication: They are particularly useful for patients with pulmonary or cutaneous sarcoidosis and those with extra-pulmonary involvement.
- Side effects: Infusion reactions, increased risk of infection, and reactivation of tuberculosis.

(4) Hydroxychloroquine:
- Hydroxychloroquine is particularly effective in treating cutaneous sarcoidosis and sarcoid arthritis, especially when other medications like MTX or AZA are not suitable.
- It is well-tolerated, but regular eye examinations are required, as it can cause retinopathy in long-term use.
- The usual dose is 200-400 mg/day, depending on the severity of symptoms.

(5) Interferon-alpha:
- Interferon-alpha is sometimes used in cases of refractory sarcoidosis, particularly in neurologic or ocular sarcoidosis. However, it is not used routinely due to side effects such as flu-like symptoms and hematological abnormalities.

Step 4: Monitoring and Long-Term Management.
- Monitoring: Regular follow-up is necessary to assess response to therapy, monitor for side effects, and ensure disease stability. This may include chest X-rays, pulmonary function tests (PFTs), and blood work to check for medication toxicity.
- Lifestyle Modifications: Patients should be encouraged to stop smoking and avoid any potential respiratory irritants to prevent disease exacerbations.

Quick Tip

In patients who progress on or cannot use methotrexate for sarcoidosis, azathioprine, mycophenolate mofetil, or anti-TNF therapy are key alternatives. Regular monitoring is essential to prevent side effects and ensure optimal treatment.
