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Essence of Practice of Medicine Volume 4
Diseases of Respiratory System

JV'n Dr. Ravi Jain

JAYOTI VIDYAPEETH WOMEN'S UNIVERSITY, JAIPUR

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Essence of Practice of Medicine

Volume 4

Diseases of Respiratory System

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Forward

First of I would like to give my gratitude towards our Honourable Chairperson Mam Jv'nMithilesh Garg ji and our Honourable Advisor sir JV'nMrVedant Garg sir for providing me an opportunity to write this book and publish in university press for the need of our students at JayotiVidyapeeth Women's University Jaipur. This book is dedicated to the Students of BHMS, BAMS, BNYS.The book will be published in 5 volumes. This part covers diseases of Respiratory System in a short and easier way. Diseases of Respiratory system are very common thes days and are affecting lot of individuals causing restriction in their day-to-day activities making them dependent on others. Proper care has been taken to include pictures of the various diseases and have been placed with the diseases to get clear and easy understanding of the disease. The topics are explained in short and only points are given for a quick review. In further volumes more chapters will be included for the benefit of the students. The matter is collected from very authentic book sources for the students and no attempt has been made to claim the ownership of the matter in order to avoid any sort of controversy. This provides a readymade instrument for quick review for many competitive exams and for the quick review during the theory main exams. Although an attempt has been done to keep the accuracy. Although if any issue is found feel free to contact the author for changes in the subsequent editions.

Jv'n Dr Ravi Jain

Author

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Chapter 1

Bronchial Asthma

- Asthma is a syndrome characterized by airflow obstruction that varies markedly, both spontaneously and with treatment.
- The inflammation in the airways makes them more responsive to a wide range of triggers, leading to excessive narrowing with consequent reduced airflow and symptomatic wheezing and dyspnea.
- Narrowing of the airways is usually reversible but in some patients with chronic asthma there may be an element of irreversible airflow obstruction.

Prevalence

- One of the most common chronic disease.
- It affects approximately 300 million people worldwide.
- There is a rising prevalence, which is associated with increased urbanization.
- The prevalence of atopy and other allergic diseases has also increased.
- Most patients with asthma in affluent countries are atopic, with allergic sensitization to the house dust mite *Dermatophagoidespteronyssinus*and other environmental allergens, such as animal fur and pollens.
- **Incidence :**
- Age **:**can present at any age, with a peak age of 3 years.
- Sex :In childhood, twice as many males as females are asthmatic, but by adulthood the sex ratio has equalized.
- Children may become asymptomatic during adolescence but that asthma returns in some during adult life.

Risk Factors & Triggers

- Asthma is a heterogeneous disease with interplay between genetic and environmental factors.
- **Endogenous Factors**
- Genetic predisposition
- Atopy
- Airway hyper responsiveness
- Gender
- Ethnicity
- Obesity
- Early viral infections

- **Environmental Factors**
- Indoor allergens
- Outdoor allergens
- Occupational sensitizers
- Passive smoking
- Respiratory infections
- Diet
- Acetaminophen (paracetamol)

Triggers

- Allergens

- Upper respiratory tract viral infections
- Exercise and hyperventilation
- Cold air
- Sulphur dioxide and irritant gases
- Drugs (β blockers, aspirin)
- Stress
- Irritants (household sprays, paint,fumes)
- **Atopy**: Atopy is a predisposition toward developing certain allergic hypersensitivityreactions.
- Patients with asthma commonly suffer from other atopic diseases, particularly allergic rhinitis, and atopic dermatitis (eczema).
- Most common allergens are derived from house dust mites, cat and dog fur, cockroaches (in inner cities), grass and tree pollens, and rodents.
- Production of specific IgE antibody.
- **Genetic Predisposition :**associations with polymorphisms of genes on chromosome 5q, including the T helper 2 (TH2) cells interleukin (IL)-4, IL-5, IL-9, and IL-13, which are associated with atopy.
- **Infections :**viral infections (especially rhinovirus) are common triggers of asthma exacerbations.
- **Diet :**diets low in antioxidants such as vitamin C and vitamin A, magnesium, selenium, and omega-3 polyunsaturated fats are associated with an increased risk of asthma.
- **Air Pollution :**Air pollutants, such as sulfur dioxide, ozone, and diesels particulates, may trigger asthma symptoms.
- **Allergens :**responsible for allergic sensitization.

- **Occupational Exposure :**Over 300 sensitizing agents have been identified. Chemicals such as toluene diisocyanate and trimellitic anhydride.
- **Obesity :**occurs more frequently in obese people (body mass index >30 kg/m2)
- **Other Factors :**lower maternal age, duration of breast-feeding, prematurity and low birthweight, and inactivity.
- **Intrinsic Asthma :**
- Negative skin tests to common inhalant allergens and normal serum concentrations of IgE.
- Usually show later onset of disease (adult-onset asthma)
- They usually have more severe, persistent asthma.

Asthma Triggers

- Several stimuli trigger airway narrowing, wheezing, and dyspnea in asthmatic patients.
- **Allergens** : Inhaled allergens activate mast cells with bound IgE directly leading to the immediate release of bronchoconstrictor mediators.
- **Virus infections :**Upper respiratory tract virus infections such as rhinovirus, respiratory syncytial virus, and coronavirus are the most common triggers.
- **Pharmacologic agents :** Several drugs may trigger asthma. Betaadrenergic blockers acutely worsen asthma. All β blockers need to be avoided.
- **Exercise :**Exercise is a common trigger of asthma. The mechanism is linked to hyperventilation. Exercise-induced asthma (EIA) worse in cold, dry climates than in hot, humid conditions.
- **Physical factors :** Cold air and hyperventilation may trigger asthma. Laughter, hot weather, weather changes, strong smells or perfumes.
- **Food and diet :** shellfish and nuts, Metabisulfite(food preservative)

- **Air pollution :** sulfur dioxide, ozone, and nitrogen oxides
- **Occupational factors :**If removed from exposure within the first 6 months of symptoms, there is usually complete recovery. More persistent symptoms lead to irreversible airway changes.
- **Hormones :** Asthma symptoms are worse before menses. It is related to a fall in progesterone.
- **Gastroesophageal reflux :** it might trigger reflex bronchoconstriction.
- **Stress :** bronchoconstriction through cholinergic reflex pathways.

Asthma

- Asthma shows chronic airway inflammation and increased airway hyper-responsiveness.
- Typical symptoms include wheeze, cough, chest tightness and dyspnoea
- It is accompanied by the presence of airflow obstruction that is variable over short periods of time
- Symptoms are reversible with treatment.

Epidemiology *:*

- The prevalence of asthma increased steadily over the latter part of the last century.
- It is estimated that asthma affects 300 million people world-wide with an additional 100 million persons by 2025.
- The socio-economic impact is enormous.
- Explored the role of microbial exposure, diet, vitamins, breastfeeding, air pollution and obesity.

Pathophysiology

- Asthma is associated with a specific chronic inflammation of the mucosa of the lower airways.

- **Airway hyper-reactivity** (AHR)—the tendency for airways to contract too easily and too much in response to triggers.
- **Pathology**
- Revealed through examining the lungs of patients who have died of asthma and from bronchial biopsies.
- The airway mucosa is infiltrated with activated eosinophils and T lymphocytes, and there is activation of mucosal mast cells.
- The degree of inflammation may even be found in atopic patients without asthma symptoms.
- In persistent asthma, a chronic and complex inflammatory response ensues, which is characterised by an influx of numerous inflammatory cells, the transformation and participation of airway structural cells, and the secretion of an array of cytokines, chemokines and growth factors.
- Smooth muscle hypertrophy and hyperplasia, thickening of the basement membrane, mucous plugging and epithelial damage.

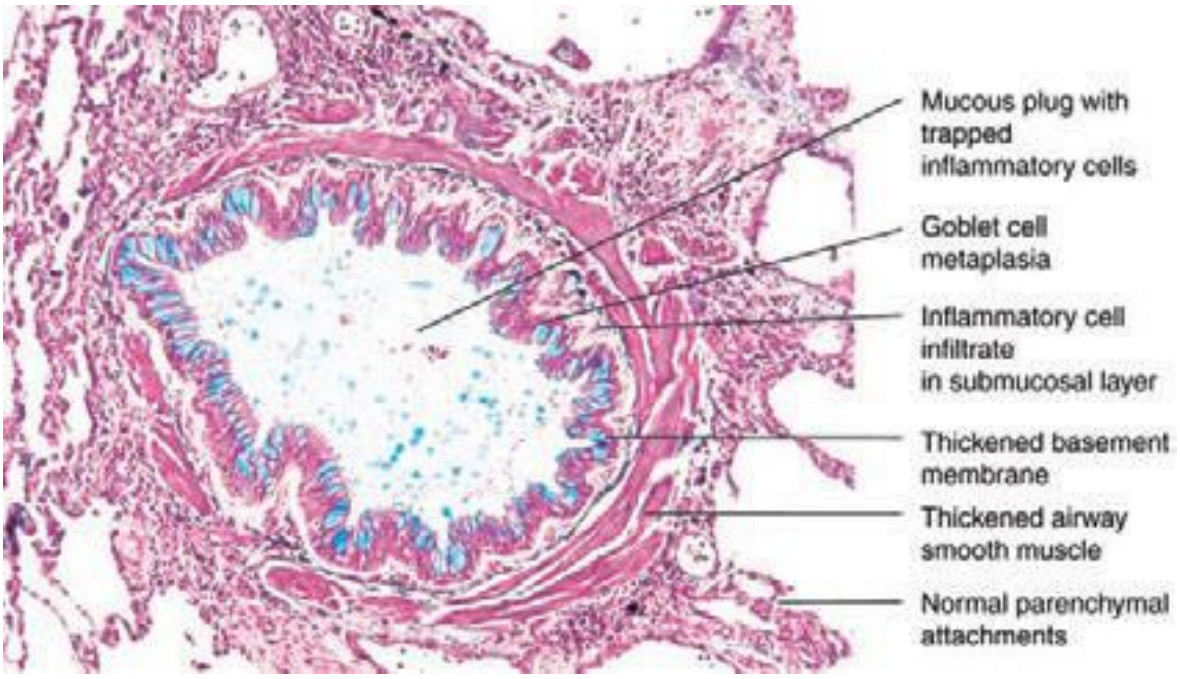

Fig: Showing Pathological Changes in Bronchial Asthma

- There are structural changes in the airways called remodeling.
- Thickening of the basement membrane due to subepithelial collagen deposition.
- The airway wall is thickened and edematous, particularly in fatal asthma.
- Occlusion of the airway lumen by a mucous plug.
- There is also vasodilation and increased numbers of blood vessels (angiogenesis).
- The airways are narrowed, erythematous, and edematous.
- These pathologic changes are found in all airways.
- There is inflammation in the respiratory mucosa from the trachea to terminal bronchioles, but with a predominance in the bronchi.
- Inhalation of an allergen into the airway is followed by a two-phase bronchoconstrictor response with both an early and a late-phase response.
- **Many inflammatory cells** are involved in asthma :
- Mast cells
- Macrophages and dendritic cells
- Eosinophils
- Neutrophils
- T lymphocytes etc
- **Airway Remodeling :**Several changes in the structure of the airway are characteristically found in asthma, and these may lead to irreversible narrowing of the airways.
- **Physiology** : Limitation of airflow is due mainly to bronchoconstriction, but airway edema, vascular congestion, and luminal occlusion with exudate.
- **Airway Hyperresponsiveness :**AHR is the characteristic physiologic abnormality of asthma and describes the excessive bronchoconstrictor response to multiple inhaled triggers that would have no effect on normal airways.

Clinical Features

- Wheezing
- Dyspnea
- Coughing
- These are variable, both spontaneously and with therapy.
- Symptoms may be worse at night, and patients typically awake in the early morning hours.
- Increased mucus production, tenacious mucus that is difficult to expectorate.
- **Prodromal symptoms** : precede an attack,
- Itching under the chin,
- Discomfort between the scapulae
- Inexplicable fear
- **Physical Signs**
- Inspiratory and expiratory, rhonchi throughout the chest, and with hyperinflation.
- Nonproductive cough.
- Asthma is mistaken for a cold or chest infection that is failing to resolve.
- Precipitants include exercise, cold weather, exposure to airborne allergens or pollutants, and viral upper respiratory tract infections.
- Patients with mild intermittent asthma are usually asymptomatic between exacerbations.
- Patients with persistent asthma report on-going breathlessness and wheeze.
- In some circumstances, asthma is triggered by medications eg aspirin, non-steroidal anti-inflammatory drugs(NSAIDs)
- These induce bronchospasm, and other may also induce wheeze.

- The presents in middle age with asthma, rhinosinusitis and nasal polyps.

Diagnosis

- Usually apparent from the symptoms of variable and intermittent airways obstruction.
- Lung Function Tests :Simple spirometry
- Hematologic Tests :Total serum IgE and specific IgE to inhaled allergens (radioallergosorbent test RAST) may be measured.
- Skin Tests :Skin prick tests to common inhalant allergens.

Differential Diagnosis

- Upper airway obstruction by a tumor or laryngeal edema.
- Left ventricular failure may mimic the wheezing of asthma. - basilar crackles present in LVF.
- Chronic obstructive pulmonary disease (COPD)

Management

- Bronchodilators
- Beta blockers
- Anticholinergics
- Controller Therapies
- Inhaled Corticosteroids
- Systemic Corticosteroids – IV hydrocortisone or methylprednisolone

Severe Acute Asthma

- Increasing chest tightness, wheezing, and dyspnea that are often not relieved by their usual reliever inhaler.
- Breathless that they are unable to complete sentences and may become cyanotic.

- **Signs** : increased ventilation, hyperinflation, and tachycardia.
- Marked fall in spirometric values and PEF.
- Arterial blood gases on air show hypoxemia, and PCO2 is usually low due to hyperventilation.

Management

- Face mask to achieve oxygen saturation of >90%.
- High doses of SABA given either by nebulizer. (short-acting beta-2 agonist).
- Sedatives should never be given because they may depress ventilation .

Chapter 2

Chronic Obstructive Pulmonary Disease (COPD)

- A disease state characterized by airflow limitation that is not fully reversible.
- It includes:
- **Emphysema** :an anatomically defined condition characterized by destruction and enlargement of the lung alveoli.
- **Chronic bronchitis**:a clinically defined condition with chronic cough and phlegm.
- **Small airways disease** :a condition in which small bronchioles are narrowed.
- COPD is present only if chronic airflow obstruction occurs.
- COPD is the third leading cause of death and affects >10 million persons in the United States.
- Estimates suggest that COPD will rise from the sixth to the third most common cause of death worldwide by 2020.

Asthma V/S COPD

- **Dutch hypothesis**. This suggests that asthma, chronic bronchitis, and emphysema are variations of the same basic disease, which is modulated by environmental and genetic factors to produce these pathologically distinct entities.
- **British hypothesis** contends that asthma and COPD are fundamentally different diseases: Asthma is viewed as largely anallergic phenomenon, whereas COPD results from smoking-related inflammation and damage.

Risk Factors

- **Cigarette smoking** : accelerated decline in FEV1 in a dose-response relationship to the intensity of cigarette smoking.

- **Airway Responsiveness and COPD :**many patients with COPD also share this feature of airway hyperresponsiveness. Increased airway responsiveness is a significant predictor of subsequent decline in pulmonary function.
- **Respiratory Infections :**respiratory infections are important causes of exacerbations of COPD
- **Occupational exposures :**exposure to dust and fumes at work. Several specific occupational exposures, including coal mining, gold mining, and cotton textile dust, have been suggested as risk factors for chronic airflow obstruction.
- **Ambient air pollution :**ambient air pollution is a much less important risk factor for COPD than cigarette smoking.
- **Passive smoking exposure :**Exposure of children to maternal smoking results in significantly reduced lung growth.
- **Genetic considerations :**Severe α1 antitrypsin deficiency is a proven genetic risk factor for COPD.

Clinical Presentation

- **History of**
- Cough
- Sputum production
- Exertional dyspnea : increased effort to breathe, heaviness, air hunger, or gasping.
- As COPD advances, there is worsening dyspnea on exertion with increasing intrusion on the ability to perform vocational or avocational activities.
- In the most advanced stages, patients are breathless doing simple activities of daily living.

Physical Findings :

- Early stages, patients usually have an entirely normal physical examination.

- Current smokers may have signs of active smoking, including an odor of smoke or nicotine staining of fingernails.
- In more severe disease, the physical examination is notable for a prolonged expiratory phase and may include expiratory wheezing.
- The signs of hyperinflation include a barrel chest and enlarged lung volumes with poor diaphragmatic excursion as assessed by percussion.
- In severe airflow obstruction exhibit use of accessory muscles of respiration
- Sitting in the characteristic "tripod" position to facilitate the actions of the sternocleidomastoid, scalene, and intercostal muscles.
- Patients may develop cyanosis, visible in the lips and nail beds.

Fig: Tripod Position

- Predominant emphysema, termed "**pink puffers**" are thin and noncyanotic at rest and have prominent use of accessory muscles.
- Patients with chronic bronchitis are more likely to be heavy and cyanotic "**blue bloaters**"
- Advanced disease is accompanied by cachexia, with significant weight loss, bitemporal wasting, and diffuse loss of subcutaneous adipose tissue.

Chapter 3

Emphysema

- It is defined as a pathological increase increase in the size of airspaces distal to the terminal bronchioles, with destruction of alveolar walls.
- Etiology :
- Alpha 1 antitrypsin deficiency.
- Cigarette smoking
- Repeated respiratory infections
- Occupational causes : requiring forceful expiration

Pathology: Seen during autopsy :

- Findings includes :
- Lungs in inflated position
- Elastic tissue is damaged
- Diaphragm is depressed
- Alveoli are overdistended
- Septa rupture and neighbouring coalesce to form cyst.
- Pulmonary vasular bed is diminished leading to pumonary hypertension.
- Reduction of alveolar surface area leads to impairment of gas exchange.
- Right ventricular hypertrophy and cor pulmonale develops.

Clinical Features

- Dyspnoea : With grades :
- 0 No breathlessness except with strenuous exercise
- 1 Breathlessness when hurrying on the level or walking up a slight hill

- 2 Walks slower than contemporaries on level ground because of breathlessness or has to stop for breath when walking at own pace.
- 3 Stops for breath after walking about 100 m or after a few minutes on level ground
- 4 Too breathless to leave the house, or breathless when dressing or undressing.

Physical Examiniation

- **Barrel shaped chest** : rounded, bulging chest that resembles the shape of a barrel.
- Hyper resonance on percussion.
- Obliteration of cardiac and liver dullness
- Diminished breath sounds
- Prolonged expiration
- Right ventricular hypertrophy

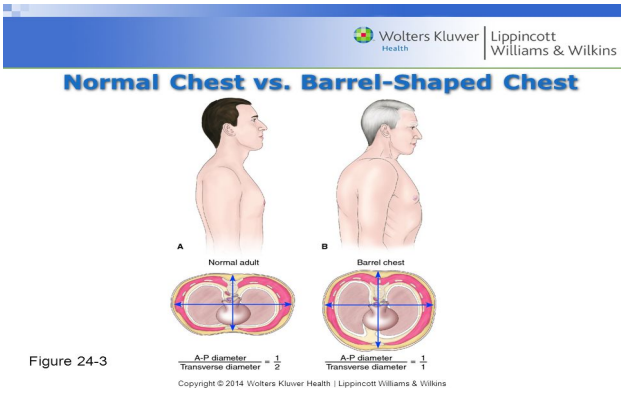

Fig: Showing changes in Emphysema

- Emphysema is classified into distinct pathologic types, the most important being centriacinar and panacinar.
- **Centriacinaremphysema:**the type most frequently associated with cigarette smoking.

- Characterized by enlarged air spaces found (initially) in association with respiratory bronchioles.
- most prominent in the upper lobes and superior segments of lower lobes and is often quite focal.

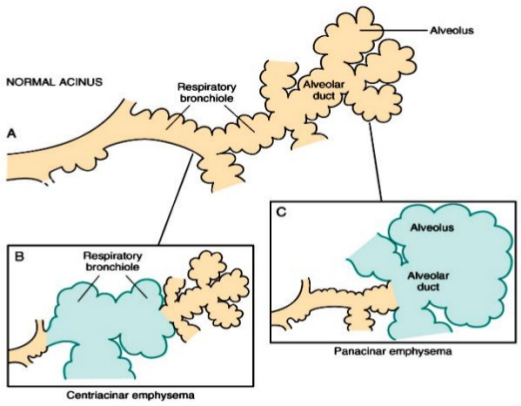

Fig: Showing changes in lung parenchyma in cases of Emphysema

- **Panacinar emphysema**
- It refersto abnormally large air spaces evenly distributed within and across acinar units.
- Usually observed in patients with α1AT deficiency.
- It has a predilection for the lower lobes.

Special Forms of Emphysema

- **Compensatory emphysema** : Normal lung tissue undergoes hypertrophy to compensate for extensive damage.
- **Atrophic emphysema** : The condition results from senile atrophy of inter alveolar septa. The lung volume is not increased.
- **Bullous emphysema** : Air spaces exceeding 1 cm in diameter develop either genetically or acquired.With time the bullae enlarge to become giant bullous emphysema.

Chapter 4

Chronic Bronchitis

- **Disease characterized by**:
- Hyper-secretion of mucous sufficient to cause cough and sputum for most of the days for at least three months in a year for two or more consecutive years in absence of any other respiratory or cardiovascular disease.
- Larger air passage are effected. Later small airways are affected producing obstructive features.
- Infection causes aggravation of symptoms and sputum becomes purulent.
- As airway obstruction sets in emphysema develops together known as Chronic bronchitis emphysema syndrome. (CEBS)

Pathology

- Bronchial mucosa shows hypertrophy and increase of mucous glands and goblet cells with overproduction of mucous.
- Distal airways shows narrowing of lumen.
- Mucosa becomes ulcerated and when ulcers heal, fibrosis occurs resulting in distortion of lumen with stenosis and dilatation.
- Distortion of airways leads to permanent obstruction.
- Secondary infection occurs in the later stage.
- Ciliary movement is impaired.

Clinical Features

- Depends on severity and duration.
- Cough recurring year after year specially in winter months later becomes constant.
- Expectoration is mucoid and sputum is tenaceous, specially on waking In morning.
- Tightness in chest

Diagnosis

- History of chronic cough.
- Mucopurulent sputum
- X ray normal in initial stage
- Lung function test shows reduction in vital capacity, increase in closing volume and features of airway obstruction.
- Pulmonary function testing shows airflow obstruction with a reduction in FEV1 and FEV1/FVC

Spirometry values are markedly reduced.

Global Initiative for Lung Disease GOLD

- GOLD Criteria for Severity of Airflow Obstruction in COPD
- GOLD Stage Severity Spirometry
- I Mild FEV1/FVC <0.7 and FEV1 ≥80% predicted
- II Moderate FEV1/FVC <0.7 and FEV1 ≥50% but <80%predicted
- III Severe FEV1/FVC <0.7 and FEV1 ≥30% but <50%predicted
- IV Very severe FEV1/FVC <0.7 and FEV1 <30% predicted

Complications

- Frequent respiratory infections
- Respiratory failure
- Right sided heart failure.

Management

- **General management**
- Smoking cessation
- Improvement of general health
- Regular exercise
- Deep breathing exercises
- Adequate sleep
- Treatment of obesity.

Specific treatment

- **STABLE PHASE COPD**
- Only three interventions:
- Smoking cessation,
- Oxygen therapy in chronically hypoxemic patients
- Lung volume reduction surgery in selected patients with emphysema.
- Use of inhaled glucocorticoids may alter mortality rate (but not lung function).
- Current therapies are directed at improving symptoms and decreasing the frequency and severity of exacerbations.

- This should be followed by an assessment of response to therapy, and a decision should be made whether or not to continue treatment.

Pharmacotherapy

- Smoking cessation
- Bronchodilators
- Anticholinergic agents : Ipratropium bromide improves symptoms and produces acute improvement in FEV1.
- Beta agonists : provide symptomatic benefit.
- Inhaled glucocorticoids :apparent benefit from the regular use of inhaled glucocorticoids on the rate of decline of lung function.
- Oral Glucocorticoids : not recommended, significant side effects, including osteoporosis, weight gain, cataracts, glucose intolerance, and increased risk of infection.

Non Pharmacologic Therapies

- General Medical Care : Patients with COPD should receive the influenza vaccine annually. Polyvalent pneumococcal vaccine.
- Pulmonary Rehabilitation : treatment program that incorporates education and cardiovascular conditioning.
- Lung Volume Reduction Surgery (LVRS) : offers both a mortality benefit and a symptomatic benefit in certain patients with emphysema
- Lung Transplantation

Patient Assessment :

- The history should include quantification of the degree of dyspnea by asking about breathlessness during activities of daily living and typical activities for the patient.

- The patient should be asked about fever; change in character of sputum; any ill contacts; and associated symptoms such as nausea, vomiting, diarrhea, myalgias, and chills.
- Inquiring about the frequency and severity of prior exacerbations can provide important information.

Acute Exacerbations

- Bronchodilators : typically with an inhaled β agonist, often with the addition of an anticholinergic agent.
- Antibiotics : for potential respiratory pathogens.
- Glucocorticoids : to reduce the length of stay, hasten recovery, and reduce the chance of subsequent exacerbation or relapse.
- Oxygen : O2 is b supplied to keep arterial saturations ≥90%.
- Mechanical Ventilatory Support :The initiation of noninvasive positivepressure ventilation (NIPPV)

Chapter 5

Bronchiectasis

- Irreversible airway dilation that involves the lung in either a focal or a diffuse manner.
- Permanent dilatation and distortion of the bronchi.
- Classically has been categorized as cylindrical or tubular (the most common form), varicose, or cystic.

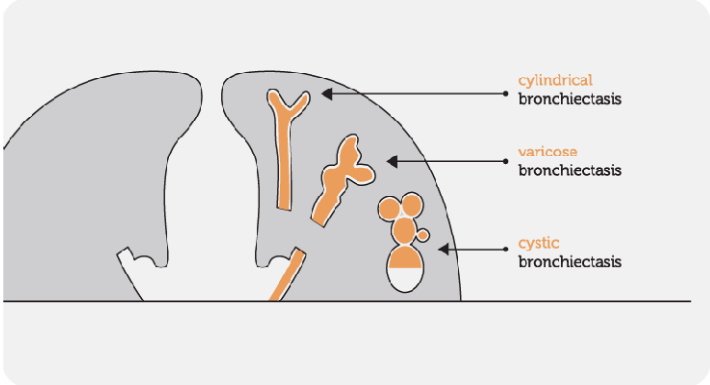

Fig Showing changes in Bronchiectasis

Etiology

- Can arise from infectious or noninfectious causes.It can be:
- Focal bronchiectasis
- Diffused bronchiectasis.
- Focal bronchiectasis : refers to bronchiectatic changes in a localized area of the lung.
- Diffuse bronchiectasis is characterized by widespread bronchiectatic changes throughout the lung.
- Focal bronchiectasis :from obstruction by,

- Extrinsic factors e.g., due to compression by adjacent lymphadenopathy or parenchymal tumor mass.
- Intrinsic factors e.g., due to an airway tumor or aspirated foreign body, a scarred/stenotic airway, or bronchial atresia from congenital underdevelopment of the airway.
- Diffuse bronchiectasis : from an underlying systemic or infectious disease process.
- Involvement of the upper lung fields is most common in cystic fibrosis (CF) and is also observed in postradiation fibrosis.
- Predominant involvement of the lower lung fields usually has its source in chronic recurrent aspiration, endstage fibrotic lung disease, or recurrent immunodeficiency-associated infections.
- The midlung fields : resulting from infection by nontuberculous mycobacteria (NTM), most commonly The Mycobacterium avium-intracellulare complex (MAC).
- Congenital causes of bronchiectasis with predominant midlung field involvement include the dyskinetic/ immotile cilia syndrome.
- In many cases, the etiology of bronchiectasis is not determined called idiopathic disease.

Epidemiology

- The epidemiology varies greatly with the underlying etiology.
- The incidence of bronchiectasis increases with age.
- Incidence is higher among women than among men.
- High incidence of malnutrition in certain areas may predispose to immune dysfunction and development of bronchiectasis.
- 25–50% of patients with bronchiectasis have idiopathic disease.

Pathogenesis and Pathology

- Vicious cycle hypothesis : susceptibility to infection and poor mucociliary clearance result in microbial colonization of the bronchial tree.
- *Pseudomonas aeruginosa*, exhibit a particular propensity for colonizing damaged airways and evading host defense mechanisms.
- Classic studies demonstrated small-airway wall inflammation and larger airway wall destruction as well as dilation, with loss of elastin, smooth muscle, and cartilage.
- The ongoing inflammatory process in the smaller airways results in airflow obstruction.
- Bronchiectasis and emphysema have been observed in patients with α1 antitrypsin deficiency.
- Mechanisms for noninfectious bronchiectasis include immune-mediated reactions that damage the bronchial wall (e.g., those associated with systemic autoimmune conditions such as Sjogren's syndrome and rheumatoid arthritis).
- *Traction bronchiectasis* refers to dilated airways arising from parenchymal distortion as a result of lung fibrosis (e.g., postradiation fibrosis or idiopathic pulmonary fibrosis).

Clinical Manifestations

- Persistent productive cough with ongoing production of thick, tenacious sputum.
- Physical findings often include crackles and wheezing on lung auscultation.
- Some patients exhibit clubbing of the digits.
- Mild to moderate airflow obstruction is often detected on pulmonary function tests.
- Acute exacerbations are characterized by increased volume and purulence of sputum production.
- Typical signs and symptoms of lung infection, such as fever and new infiltrates, may not be present.

Approach to Patient

- Elicitation of clinical history, chest imaging, and a workup to determine the underlying etiology.
- Bronchoscopy to exclude airway obstruction by an underlying mass or foreign body in focal bronchiectasis.
- Pulmonary function test.
- Bacteriological and mycological examination of sputum
- Assessment of ciliary function.

Diagnosis

- Persistent chronic cough and sputum production accompanied by consistent radiographic features.
- Chest radiographs lack sensitivity, the presence of “tram tracks” indicating dilated airways is consistent with bronchiectasis.
- Chest computed tomography (CT) is more specific . CT findings include airway dilation (detected as parallel “**tram tracks**” or as the “**signet-ring sign**”. (a cross sectional area of the airway with a diameter at least 1.5 times that of the adjacent vessel),
- Lack of bronchial tapering, bronchial wall thickening, or cysts emanating from the bronchial wall.

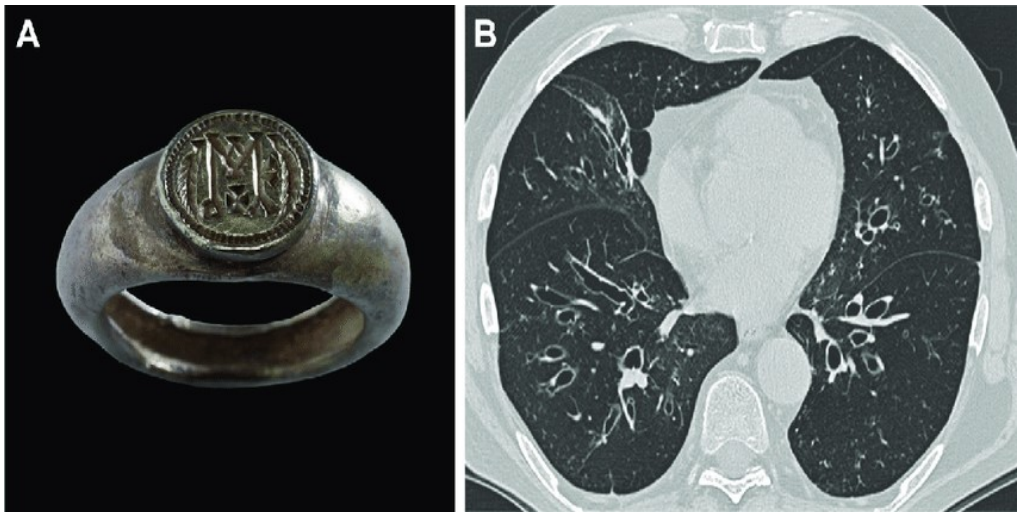

Fig Showing Signet ring appearance in Bronchiectasis

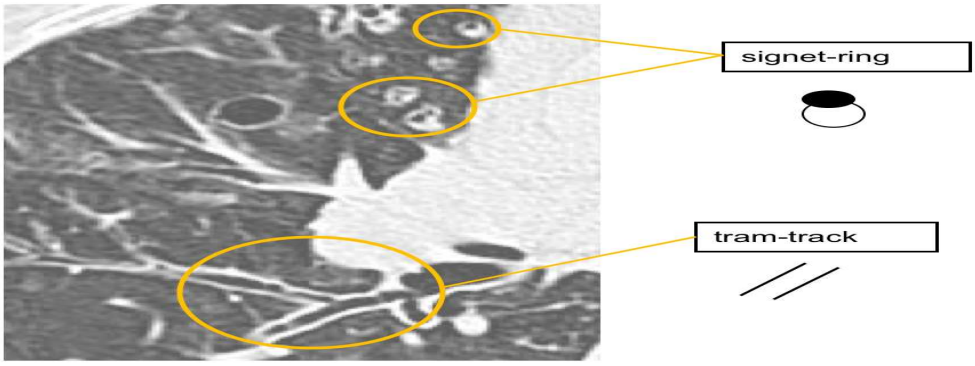

Fig: X Ray Showing Signet Ring and Tram Tracks in case of Bronchiectasis

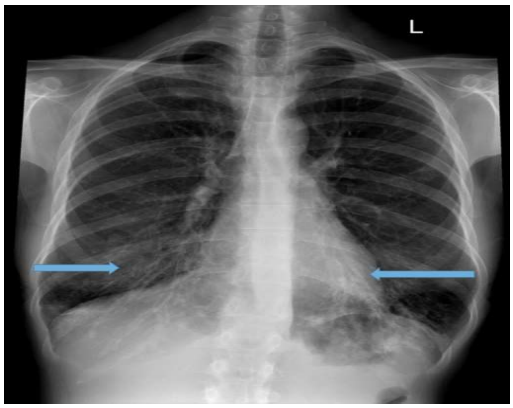

Fig: Showing X Ray Changes in Bronchiectasis

Management

- Control of active infection
- Improvements in secretion clearance
- Bronchial hygiene
- To decrease the microbial load within the airways and minimize the risk of repeated infections.
- **Antibiotic Treatment:** targetingthe causative or presumptive pathogen (*Haemophilus influenzae* and *P. aeruginosa).* For *Pseudomonas*, oral ciprofloxacin or ceftazidime by intravenous injection or infusion may be required.
- **Bronchial Hygiene**: to enhance secretion clearance,hydration and mucolytic administration, regular daily physiotherapy to assist the drainage of excess bronchial secretions.
- **Surgical treatment:** Excision of bronchiectatic areas is only indicated in a small proportion of cases.

Complications

- In severe cases of infectious bronchiectasis and recurrent infections, microbial resistance to antibiotics may develop.
- Recurrent infections can result in injury to superficial mucosal vessels with bleeding and, in severe cases, life-threatening hemoptysis.

Prognosis

- The prognosis varies widely with the underlying etiology and may also be influenced by the frequency of exacerbations and the specific pathogens involved.
- In The disease is progressive when associated with ciliary dysfunction and cystic fibrosis, and eventually causes respiratory failure.
- The prognosis can be relatively good if physiotherapy is performed regularly and antibiotics are used aggressively.

Prevention

- Reversal of an underlying immunodeficient state by administration of gamma globulin.
- Vaccination of patients with chronic respiratory conditions by influenza and pneumococcal vaccines.

Chapter 6

Pneumonia

- Pneumonia is an inflammatory condition of the lung usually caused by infection with bacteria or viruses affecting primarily the small air sacs known as alveoli. Typically symptoms include some combination of productive or dry cough, chest pain, fever, and trouble breathing.
- Can be of following types :
- Community acquired
- Hospital acquired
- Suppuration and aspirational pneumonia
- Pneumonia in immunocompromised

Community-acquired pneumonia (CAP)

- 5–11/1000 adults suffer from CAP each year.
- The incidence varies with age, being much higher in the very young and very old, in whom the mortality rates are also much higher.
- Spread by droplet infection
- It accounts for almost $1/5^{th}$ of the childhood deaths worldwide with approximately 2 million children under 5 die each year.
- The classical pathological response evolves through the phases of congestion, red and grey hepatisation and finally resolution with little or no scarring.

Factors that predispose to pneumonia

- Cigarette smoking
- Upper respiratory tract infections
- Alcohol

- Corticosteroid therapy
- Old age
- Recent influenza infection
- Pre-existing lung disease
- HIV
- Indoor air pollution
- Caused by : Influenza A and B, chickenpox (respiratory syncytial virus, parainfluenza, measles and adenoviruses)

Clinical Features

- Presents as an acute illness in which systemic features such as
- Fever,
- Rigors,
- Shivering
- Vomiting predominate.
- The appetite is lost and headache.
- Breathlessness and cough, which at first is characteristically short, painful and dry, but later accompanied by the expectoration of mucopurulent sputum.
- Pleuritic chest pain may be a presenting feature and may be referred to the shoulder or anterior abdominal wall.
- Clinical signs reflect the nature of the inflammatory response.
- Majority of the cases of CAP are due to infection with Streptococcus pneumonia
- Recent infection may predispose to Staphylococcus aureus.

Investigations

- Objectives are to exclude other conditions that mimic pneumonia assess the severity, and identify the development of complications.
- A chest X-ray usually provides confirmation of the diagnosis.

Microbiological Investigations

- Sputum: direct smear by Gram and Ziehl– Neelsen stains.
- Culture and antimicrobial sensitivity testing
- Blood culture: frequently positive in pneumococcal pneumonia
- Serology: acute and convalescent titres for *Mycoplasma, Chlamydia, Legionella,* and viral infections.
- Pneumococcal antigen detection in serum or urine
- PCR: *Mycoplasma* can be detected from swab of oropharynx
- Throat/nasopharyngeal swabs: helpful in children or during influenza epidemic.

Differential Diagnosis

- Pulmonary infarction
- Pulmonary/pleural TB
- Pulmonary oedema
- Pulmonary eosinophilia
- Malignancy: bronchoalveolar cell carcinoma.

Management

- Oxygen
- Fluid Balance
- Antibiotics: Amoxicillin, Erythromycin etc

- Treatment of pleural pain
- Physiotherapy

Prevention

- Smoking cessation
- Influenza and pneumococcal vaccination.
- Tackling malnourishment and indoor air pollution.
- Encouraging immunisation against measles, pertussis and *Haemophilus influenza* type b in children.

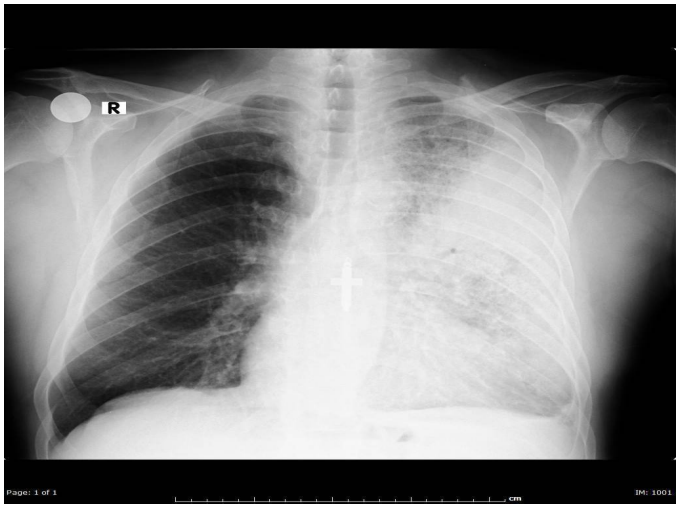

Fig: Showing Changes in X Ray in case of Pneumonia

Hospital-acquired pneumonia

- A new episode of pneumonia occurring at least 2 days after admission to hospital.
- At risk :
- Older people,
- Patients in intensive care units,

- especially when mechanically ventilated, in which case the term ventilator- associated pneumonia (VAP)
- Health care- associated pneumonia (HCAP) : development of pneumonia in a person who has spent at least 2 days in hospital within the last 90 days, attended a haemodialysis unit, received intravenous antibiotics, or been resident in a nursing home or other long-term care facility.
- Aetiology : Gram-negative bacteria (e.g. Escherichia, Pseudomonas and Klebsiella species), Staph. aureus (including meticillin-resistant Staph. aureus (MRSA)) and anaerobes.

Predisposing Factors

- **Reduced host defences against bacteria,** e.g. corticosteroid treatment, diabetes, malignancy)
- **Aspiration of nasopharyngeal or gastric secretions,** Immobility or reduced conscious level, Nasogastric intubation.
- **Bacteria introduced into lower respiratory tract,** Endotracheal intubation/tracheostomy, Infected ventilators/nebulisers/bronchoscopes.
- **Bacteraemia,** Abdominal sepsis, I.v. cannula infection.

Clinical Features

- Any hospitalised or ventilated patient who develops purulent sputum (or endotracheal secretions).
- Unexplained increase in oxygen requirement.
- A core temperature > 38.3°C,
- **Investigations**
- A leucocytosis or leucopenia.

Investigations are similar to those outlined for CAP

Management

- Empirical antibiotic therapy should be based on local knowledge of pathogens and drug resistance patterns, and variables such as length of hospital stay, recent antibiotics and comorbidity.
- Antibiotics cefotaxime, gentamicin.
- MRSA is treated with intravenous vancomycin.
- Physiotherapy in those who are immobile or old.
- Adequate oxygen therapy,
- Fluid support
- Monitoring is essential.

Prevention

- Despite appropriate management, the mortality from HAP is approximately 30%, emphasising the importance of prevention.
- Good hygiene, including both hand washing an equipment.
- Steps to minimise the chances of aspiration and limit the use of stress ulcer.
- Prophylaxis with proton pump inhibitors.
- Oral antiseptic (chlorhexidine 2%).

Suppurative pneumonia, Aspiration pneumonia

- Aetiology and clinical features overlap in both the conditions.
- Suppurative pneumonia is characterised by destruction of the lung parenchyma by the inflammatory process.
- **Caustiveagents** : Strep. pneumoniae, Staph. aureus, Strep. pyogenes, H. influenzae.

Clinical Features

- Productive cough with large amounts of sputum sometimes fetid and blood-stained.

- Pleural pain
- High remittent pyrexia
- Digital clubbing (10–14 days)
- Chest examination usually reveals signs of consolidation.
- Pleural rub
- Rapid deterioration in general health with marked weightloss.

Investigations

- Radiological features homogeneous lobar or segmental opacity consistent with consolidation or collapse.
- Abscesses are characterised by cavitation and fluid level.
- Pre-existing emphysematous bulla becomes infected and appears as a cavity containing an air-fluid level.
- Culture blood and sputum.

Management

- Oral treatment with amoxicillin, co-amoxiclav, oral metronidazole
- Surgery if no improvement occurs despite optimal medical therapy.
- Removal or treatment of any obstructing endobronchial lesion.

Pneumonia in the immunocompromised patient

- Patients immunocompromised by drugs or disease.
- **Causative factors** : Gram-negative bacteria, especially *Pseudomonas aeruginosa*, viral agents, fungi, mycobacteria.

Causes of Immunosuppression

- **Defective phagocytic function :**Acute leukaemia, Cytotoxic drugs.

- **Defects in cellmediatedimmunity :**Immunosuppressive drugs Cytotoxic chemotherapy.
- **Defects in antibody production :**Multiple myeloma, Chronic lymphocytic leukaemia.

Clinical Features

- Fever, cough and breathlessness. Cough and breathlessness can be present several days or weeks
- Invasive pulmonary aspergillosis causes fever, pleural pain or haemoptysis or pleural rub.
- Invasion of pulmonary vessels causes thrombosis and infarction,
- Systemic spread may occur to the brain, heart, kidneys and other organs.

Diagnosis

- Invasive investigations such as bronchoscopy transbronchial biopsy or surgical lung biopsy are often impractical.
- Induced sputum for obtaining microbiological samples.

Management

- Broad-spectrum antibiotic therapy.
- Antifungal or antiviral therapies may be added.
- Mechanical ventilation increases the risk of nosocomial pneumonia.

Chapter 7

Tuberculosis

- Tuberculosis is caused by infection with *Mycobacterium tuberculosis.*
- *M. tuberculosis* is a rod-shaped, non-spore-forming, thin aerobic, acid-fast bacilli.
- *M. bovis*(reservoir cattle)
- *M. africanum*(reservoir human).
- Around one-third of the world's population has latent TB.
- This disease most often affects the lungs, although other organs are involved in up to one-third of cases.

Pathology & Pathogenesis

- *M. bovis*:arises from drinking non-sterilised milk from infected cows.
- *M. tuberculosis* is spread by the inhalation of aerosolised droplet nuclei from other infected patients by coughing, sneezing, or speaking.
- The organisms lodge in the alveoli and initiate the recruitment of macrophages and lymphocytes.
- Macrophages undergo transformation into epithelioid and Langhans cells which aggregate with the lymphocytes to form the classical tuberculous granuloma (The normal lung tissue is lost and replaced by a mass of fibrous tissue with granulomatous inflammation characterised by the presence of large numbers of macrophages and multinucleate giant cells)
- Numerous granulomas aggregate to form a primary lesion or **Ghonfocus**(a pale yellow, caseous nodule, usually a few mm to 1–2 cm in diameter), which is characteristically situated in the periphery of the lung.
- The combination of a primary lesion and regional lymph nodes is referred to as the '**primary complex of Ranke**'.
- This lesion eventually calcifies and is clearly seen on a chest X-ray.

- Secondary foci in other organs including lymphnodes, serous membranes, meninges, bones, liver, kidneys and lungs.
- Infection has occurred may be the appearance of a cell-mediated, delayed-type hypersensitivityreaction to tuberculin, demonstrated by tuberculin skin testing.

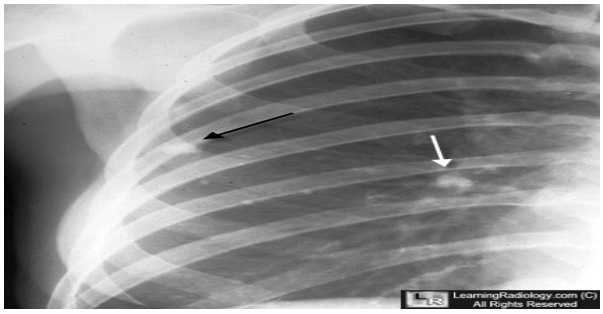

Fig: Showing X Ray Changes in Tuberculosis

Clinical features:Primary Pulmonary disease:

- Infection of a previously uninfected (tuberculin-negative) individual.
- Self-limiting febrile illness
- Influenza-like illness
- Skin test conversion
- Primary complex
- Lymphadenopathy
- Consolidation
- Obstructive emphysema
- Pleural effusion
- Erythema nodosum
- Miliary TB : Blood-borne dissemination.
- 2–3 weeks of fever,

- Night sweats,
- Anorexia,
- Weight loss
- Dry cough.
- Chest X-ray : The classical appearances of fine 1–2 mm lesions (‘millet seed’).
- Anaemia and leucopenia reflect bone marrow involvement.

Post-primary pulmonary TB

- Exogenous (new infection) or endogenous (reactivation of a dormant primary lesion) infection in a person who has been sensitised by earlier exposure.
- Most common, occurs in the apex of an upper lobe where the oxygen tension favours survival of the strictly aerobic organism.
- Insidious onset, developing slowly over several weeks.
- Systemic symptoms include fever, night sweats, malaise, and loss of appetite and weight.
- Chronic cough, often with haemoptysis
- Pyrexia of unknown origin
- Unresolved pneumonia
- Exudative pleural effusion
- Asymptomatic (diagnosis on chest X-ray)
- Weight loss, general debility
- Spontaneous pneumothorax
- Radiological changes
- Ill defined opacification in one or both of the upper lobes, and as progression occurs, consolidation, collapse and cavitation to varying degrees.

Clinical features: extrapulmonary disease

- Lymphadenitis
- Gastrointestinal disease
- Pericardial disease
- Central nervous system disease
- Bone and joint disease
- Genitourinary disease

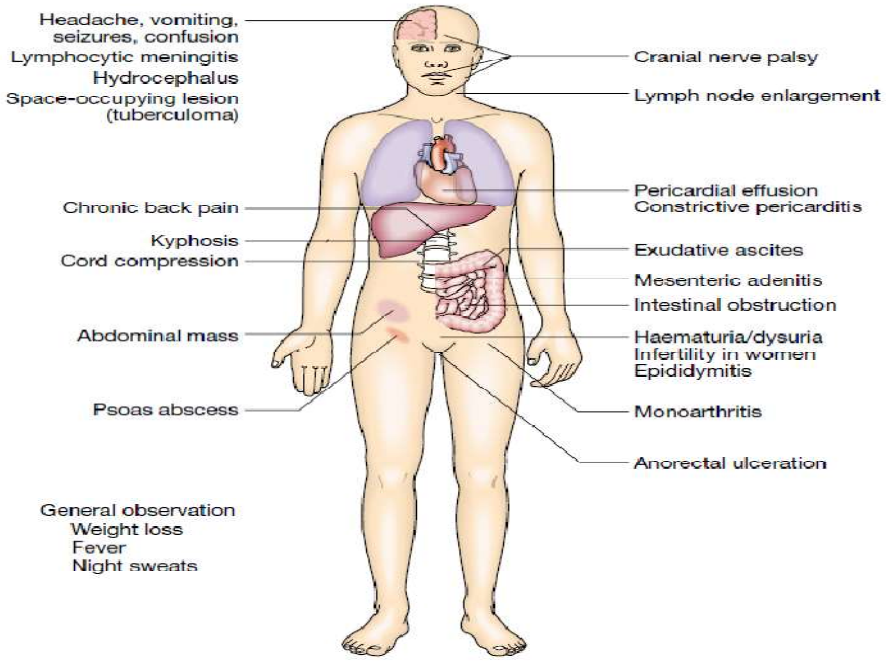

Fig: Showing changes in Extrapulmonary Tuberculosis

Diagnosis

- Unexplained cough for more than 2–3 weeks, particularly in an area where TB is highly prevalent.
- Direct microscopy of sputum

Management

First Line of Treatment

- Initial Phase (2months)
- Rifampicin
- Isoniazid
- Ethambutol
- Pyrazinamide
- Continuation Phase (4 Months)
- Isoniazid
- Rifampicin
- **If Pyrazinamide is not given or can not be tolerated**
- **Initial Phase (2 Months)**
- Ehambutol
- Isoniazid
- Rifampicin
- **Continuation Phase (7Months)**
- Isoniazid
- Rifampicin

Second line Antituberculous Drugs

- Steptomycin
- Kanamycin
- Amikacin
- Capreomycin

- Ofloxacin
- Levofloxacin
- Moxifloxacin
- Ethionamide
- Prothionamide

Second line Antituberculous Drugs

- Cycloserine
- PAS
- These 2 are bacteriostatic

Chapter 8

Cystic Fibrosis

- A monogenic disorder. It is an autosomal recessive exocrinopathy affecting multiple epithelial tissues.
- It result of mutations affecting a gene on the long arm of chromosome 7.
- The gene product responsible for CF the cystic fibrosis transmembrane conductance regulator [CFTR].
- It influences salt and water movement across epithelial cell membranes.
- Incidence : 1 in 2500 live births
- It is charcterizedby :
- Chronic bacterial infection of airways leading to bronchiectasis,
- Exocrine pancreatic insufficiency
- Intestinal dysfunction ,
- Abnornal sweat gland function
- Urogenital dysfunction

Pathophysiology

- The genetic defect causes increased sodium and chloride content in sweat and increased resorption of sodium and water from respiratory epithelium.
- Relative dehydration of the airway epithelium predispose to chronic bacterial infection and ciliary dysfunction, leading to bronchiectasis.
- The gene defect also causes disorders in the gut epithelium, pancreas, liver and reproductive tract.
- CF produces organ specific effects on electrolyte and water transport.
- GIT: Defect in Na+ absorption and Cl- secretion causes dehydration of surface epithelium.

- It causes dehydration of intramural contents and intestinal obstruction..
- In hepatobiliary system defective Cl- and water secretion causes thickened biliary secretions, focal biliary cirrhosis and bile duct perforation.
- Inability of CF gall bladder epithelium to secrete salt and water can lead to both both cholecystitis and cholelithiasis.
- Sweat gland : Sweat secretion is normal but not able to absorb Nacl from sweat duct.

Clinical Features

- Respiratory tract: Lungs are normal at birth, but bronchiolar inflammation and infections usually lead to bronchiectasis in childhood.
- Common infections : Staphylococcus aureus, Pseudomonas aeruginosa.
- Recurrent exacerbations of bronchiectasis, cause progressive lung damage resulting ultimately in death from respiratory failure.
- Upper respiratory tract disease : Chronic sinusitis, incidence of nasal polyp.
- In lower respiratory tract : Cough which becomes persistant, and produces viscous, purulent, and often greenish sputum.
- Periods of clinical stability interrupted by pulmonary exacerbations, often triggered by viral infections.
- Symptoms includes increased cough, weight loss, low grade fever, increased sputum volume, and decreased pulmonary function.
- Over the course of time the exacerbations frequency increases, recovery is lost and lung functions becomes incomplete leading to respiratory failure.
- Complications leads to bronchiectasis, Pneumothorax, Haemoptysis, Nasal polyps, Respiratory failure, Cor pulmonale.
- **Gastrointestinal :**Meconium ileus**,** Malabsorption and steatorrhoea
- Distal intestinal obstruction syndrome (DIOS)

- Biliary cirrhosis and portal hypertension
- Gallstones
- **Exocrine pancreatic insufficiency** : occurs in >90% pt with CF.
- Insufficiency leads to deficient pancreatic enzyme secretion hence protein and fat malabsorption.
- Frequent bulky and foul smelling stools.
- Malabsorption of fat soluble vitamins.
- In pancreas can involve Beta cells leading to diabetes mellitus.
- **Genitourinary**: azoospermia in males due to obliteration of vas deferens due to defective liquid secretion.
- 20% women are infertile.
- **Others**: Diabetes, Delayed puberty, Male infertility, Stress incontinence, psychosocial problems, osteoporosis, arthropathy

Diagnosis

- Commonly made from the clinical picture :
- Bowel obstruction,
- Failure to thrive,
- Steatorrhoea
- Chest symptoms in a young child
- Diagnosis is supported by :
- Sweat electrolyte testing.
- Genotyping (DNA analysis).
- Decreased lung function test FVC, FEV1.

- X ray Hyperinflation of lung.

Management

Respiratory complaints

Non Respiratory complaints

Respiratory complaints management

- Main objective of therapy is to promote clearance of secretions and control of lung infection.
- Pulmonary secretions clearing techniques :exercise,chest percussion, etc
- 95% deaths occurs due to complications of lung infection.
- Staph. Aureus infection : oral antibiotics, intravenous treatment
- Psedomonasinfection :nebulised antibiotic therapy.
- Inhaled Beta antagonists to control airway contraction.
- Regular chest physiotherapy.

Non-respiratory manifestations management

- Oral pancreatic enzyme supplements
- Vitamins E and K.
- Insulin for diabetes.
- Gene therapy : Manufactured normal CF gene can be packaged within a viral or liposome vector and delivered to the respiratory epithelium to correct the genetic defect.
- Management of psychosocial problems like counseling for health insurance, career options, family planning etc.

Chapter 9

Homoeopathic Management

- **AmbraGrisea :**Asthmatic breathing with eructation of gas, Nervous. spasmodic cough, with hoarseness and eructation, on waking in morning; worse in presence of people. Gets out of breath when coughing. Hollow, spasmodic, barking cough, coming from deep in chest. Choking when hawking up phlegm.

- **Ammonium carbonicum**: Cough every morning about three o'clock, with dyspnoea, palpitation, burning in chest; worse ascending. Chest feels tired.

- **Argentum nitricum**: High notes cause cough. Chronic hoarseness. Suffocative cough. Dyspnoea, Painful spots in chest.

- **Arsenicumalbum** : Unable to lie down; fears suffocation. Air-passages constricted. **Asthma worse midnight**. Burning in chest. Suffocative catarrh. Cough worse after midnight; worse lying on back. Expectoration scanty, frothy. Darting pain through upper third of right lung. Wheezing respiration.

- **Arsenicumiodatum:** Slight hacking cough, with dry and stopped-up nostrils. Cough dry, with little difficult expectoration.

- **Antimoniumars:** an excellent remedy for COPD with emphysema. There is excessive dyspnea and cough with much mucus secretion , worse on eating or lying down.

- **Antimoniumtart** : indicated in emphysema of the aged, coughing and gasping consequently . There is great rattling of mucus in the lungs and rapid , short, difficult breathing.

- **Aspidosperma -** is one of the best remedies for COPD and it is considered a tonic for lungs. It removes temporary obstruction to the oxidation of blood by stimulating respiratory centres. Another guiding symptom is want of breath during exertion .

- **Bryoniaalba :**excellent remedy prescribed when there is frequent desire to take long breath, must expand lungs.There is dry, barking cough , worse at night. Respiration is quick , difficult with pain in the chest.< motion, > absolute rest.

- **Blattaorientalis**: A remedy for asthma. Especially when associated with bronchitis. Indicated after arsenic when this is insufficient. Much pus-like mucus.

- **Bromium** : Dry cough, with hoarseness and burning pain behind sternum. Spasmodic cough, with rattling of mucus in the larynx; suffocative. Hoarseness. Difficult and painful breathing. Violent cramping of chest. Chest pains run upward. Every inspiration provokes cough. Spasmodic constriction. Asthma; difficulty in getting air into lung. (Chlorum, in expelling.) Better at sea, of seafaring men when they come on land, great dyspnoea.

- **BalsamumPeruvianum**: Bronchitis, and phthisis, with muco-purulent, thick, creamy expectoration. Loud rales in chest. Very loose cough. Hectic fever and night-sweats, with irritating, short cough and scanty expectoration.

- **Copavia** : Cough, with profuse, gray, purulent expectoration. Tickling in larynx, trachea, and bronchi. Bronchial catarrh, with profuse greenish, offensive discharge.

- **Coca** : prescribed for COPD where there is want of breath or shortness of breath. It is especially useful for aged sports men and alcoholics. There is hoarseness or loss of voice and dyspnea.

- **Curare** - where there is threatened cessation of respiration on falling asleep . Short breath, short dry cough, very distressing dyspnea.

- **Chlorum :**Sudden dyspnoea from spasm of the vocal cords, with staring protruding eyes, blue face, cold sweat, pulse small. Inspiration free, with obstructed expiration.

- **Cuprum metallicum :**Cough as a gurgling sound, better by drinking cold water. Suffocative attacks, worse 3 a.m. Spasm and constriction of chest; spasmodic asthma, alternating with spasmodic vomiting.

- **CalcareaIod**: It is in the treatment of scrofulous affections, especially enlarged glands, tonsils, etc Chronic cough; Pain in chest, difficulty breathing after syphilis . Hectic fever; green purulent expectoration

- **Drosera:** dry cough, worse at night and from the warmth of the bed, triggered by speaking or laughing, with a feeling of tickling in the throat.

- **Eriodyctioncalifornicum (Yerba Santa) :**Wheezing; asthma, with coryza and mucous secretions. Dull pain in right lung. Burning in fauces. Asthma relieved by expectoration.

- **Heparsulph :**relieves dry, hoarse cough worsened by cold air and cold drinks, occurring at the beginning and at the end of the night.

- **Ipecacuanha** :Dyspnoea; constant constriction in chest. Asthma. Yearly attacks of difficult shortness of breathing. wheezing cough. Cough incessant and violent, with every breath. Chest seems full of phlegm, but does not yield to coughing. Bubbling rales. Suffocative cough.

- **Iodum:** Great debility, the slightest effort induces perspiration.Great weakness about chest. Palpitation from least exertion. Pleuritic effusion. Tickling all over chest. Iod cough is worse indoors, in warm, wet weather, and when lying on back.

- **Kali Sulph**: Coarse rales. *Rattling of mucus in chest* .Cough; worse in evening and in hot atmosphere. Croupy hoarseness.Cough with yellow expectoration. Rise of temperature at night. Intermittent fever.

- **Kalium carbonicum**: Dry, hard cough about 3 a.m., with stitching pains. Expectoration scanty and tenacious, but increasing in morning and after eating; aggravated right lower chest and lying on painful side.

- **Lobelia inflata :**Dyspnoea from constriction of chest; worse, any exertion. Sensation of pressure or weight in chest; better by rapid walking. Asthma; attacks, with weakness, felt in pit of stomach and preceded by prickling all over.

- **Mephitis Putorius :**Suffocative feeling, asthmatic paroxysms, spasmodic cough; cough so violent, seems as if each spell would terminate life. Child must be raised up,gets blue in face, cannot exhale, Mucous rales through upper part of chest. Patient wants to bathe in ice-cold water.

- **Naphthaline**: indicated where there is dyspnea and sighing respiration. It is useful for emphysema of the aged with asthma. There is long and continued paroxysms of coughing and tenacious expectoration.

- **PhellandriumAquaticum** :Dyspnoea, and continuous cough, early in morning. Cough, with profuse and fetid expectoration; compels him to sit up.
- **Pulsatilla** : Dry cough in evening and at night; must sit up in bed to get relief; and loose cough in the morning, with copious mucous expectoration. Expectoration bland, thick, bitter, greenish. Short breath, anxiety, and palpitation when lying on left side.
- **Phosphorus :**hoarseness and a tickly cough, cough can be aggravated by talking, laughing, and exposure to cold air, thirst for cold drinks.
- **Sambucus nigra** : Paroxysmal, suffocative cough, coming on about midnight, with crying and dyspnoea. Child awakes suddenly, nearly suffocating, sits up, turns blue. Cannot expire.
- **Spongiatosta**: asthmatic cough, worse cold air, with profuse expectoration and suffocation; worse, lying with head low and in hot room.
- **Sulphur**:Oppression and burning sensation in chest. Difficult respiration; wants windows open, Dyspnoea in middle of night, relieved by sitting up. Much rattling of mucus.
- **Senega :**an effective remedy with increased respiration. There is excessive dyspnea and sharp contractive pains in the muscles of chest. Persistent cough.
- **Strychninum:** Respiration increased. Excessive dyspnea. Sharp contractive pains in the muscles of chest. Persistent cough.
- **SanguinarinumNitricum**: Short, hacking cough, with expectoration of thick, yellow, sweetish mucus.
- **Stannummetallicum** : mucus expelled by forcible cough. Violent, dry cough in evening until midnight. Cough excited by laughing, singing, talking; worse lying on right side. During day, with copious green, sweetish, expectoration.
- **Tuberculinum**: Of undoubted value in the treatment of incipient tuberculosis. Especially adapted to the light-complexioned, narrow-chested subjects.

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