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Diseases of the respiratory system: A basic review

Dr. Siva Rami Reddy E
Faculty of Homoeopathy, Tantia University, Sri Ganganagar, Rajasthan, India

Abstract
Respiratory diseases (basic review) are to understand very easily for graduate, post graduate and post-doctoral ayush, dental, medical etc., students. I am explaining main and important diseases in respiratory system in day to day practical life for medical students and professionals. Diseases are chronic obstruction pulmonary disease, bronchiectasis, cystic fibrosis, pneumonia, tuberculosis, bronchial asthma, pleural effusion, and empyema.

Keywords: respiratory diseases, causes, clinical features, investigation, management

Introduction
We have lot of diseases to explain in respiratory system. But only main/few diseases are reviewing for under graduate, post graduate and post-doctoral ayush, dental, medical, nursing etc., entrance and main examination purpose.

Chronic obstruction pulmonary disease (COPD)
Chronic obstructive pulmonary disease (COPD) is the name for a group of lung conditions that cause breathing difficulties. It includes:

- Emphysema – damage to the air sacs in the lungs.
- Chronic bronchitis – long term inflammation of the airways.

Chronic obstructive pulmonary disease is a common condition that mainly affects middle aged or older adults who smoke. Many people do not realise they have it. The breathing problems tend to get gradually worse over time and can limit your normal activities, although treatment can help keep the condition under control.

Definition
Chronic obstructive pulmonary disease is the respiratory disorder characterized by airflow obstruction mainly resulting from chronic bronchitis, emphysema. This does not change markedly over several months. The impairment of lung function is largely fixed but may be partially reversible by bronchodilator therapy. Historically, the term chronic bronchitis was used to define any patient who coughed up sputum on most days of at least three consecutive months or more for more than two successive years. Emphysema referred to the pathological process of a permanent destructive enlargement of the airspaces distal to the terminal bronchioles. According to WHO the death rate from Chronic obstructive pulmonary disease currently exceeds 5000 per year in north, west, east, south India and this condition accounts for over 10% of all hospital medical admission in the India [1, 2].

Causes
Chronic obstructive pulmonary disease is cigarette smoking and a direct correlation exists between the number of cigarettes smoked in pack year (one pack year =20 cigarettes smoked daily for one year) and the likelihood of developing the disease. Smoking is thought to have its effect by inducing persisting airway inflammation and causing a direct imbalance in antioxidant capacity and antiproteinase/proteinase load in the lungs. Individual susceptibility (homoeopathy) to smoking is, however, very wide, with only 15 t0 20% of smokers likely to develop clinically significant Chronic obstructive pulmonary disease. A small additional contribution to the severity of chronic obstructive pulmonary disease has been reported in patients exposed to dusty or air polluted environments. Also causes of low birth weight, bronchial hyper responsiveness and the formation of chronic obstructive pulmonary disease. Alpha antitrypsin deficiency can cause emphysema in non-smokers but this risk is increased strongly in enzyme deficient patient who smoke. Stopping smoking slows the average rate of decline in forced expiration volume from 50 – 70 ml/year to 30 ml/year (equal to non-smokers).

Pathology
Chronic obstructive pulmonary disease patients develop airway wall inflammation hyper trophy of the mucus secreting glands and increase in the number of goblet cells in the bronchi and bronchioles with a consequent decrease in ciliated cells. Its leads to less efficient transport of the increased mucus in the airways and loss of alveolar attachments around such airway makes them more liable to collapse during expiration. Emphysema is usually centriacinar, involving respiratory bronchioles, alveolar ducts and centrally located alveoli. Paraseptal emphysema develops with the latter responsible for blebs on the lung surface and giant bullae. Pulmonary vascular remodelling caused by persistent hypoxaemia results in pulmonary hypertension and right ventricular hypertrophy and dilation.

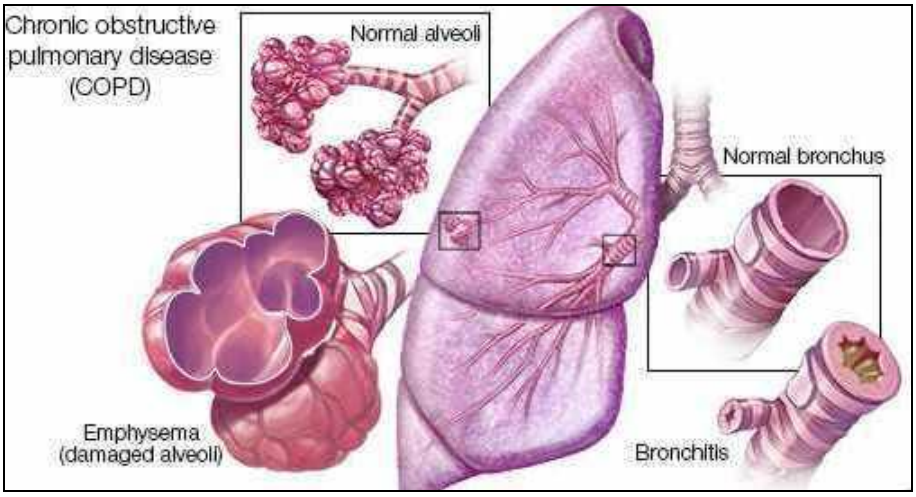

Fig 1: Difference between normal and COPD bronchus, alveoli

Clinical features

Patient have a shortness of breath (SOB) and coughing as a normal part of aging, but these could be signs of chronic obstructive pulmonary disease. Chronic obstructive pulmonary disease can progress for years without noticeable shortness of breath. Symptoms of chronic obstructive pulmonary disease can be different for each person, but common symptoms are:

- Increased shortness of breath.
- Frequent coughing (with and without mucus).
- Increased breathlessness.
- Wheezing.
- Tightness in the chest.

Patients will suffer recurrent respiratory infections, extertional breathlessness, regular morning cough, severe wheeze. Sputum may be scanty, mucoid, tenacious and occasionally streaked with blood during infective exacerbations. Frankly purulent sputum is indicative of bacterial infection. Shortness of breath is aggravated by infection, excessive cigarette smoking and adverse atmospheric condition. In auscultation method can find creptitions (crackles) which usually, but not always, disappear after coughing may be clear audible over lower zones.

Classification of Chronic obstructive pulmonary disease: It is divided in to mild, moderate and severe.

In mild Chronic obstructive pulmonary disease is spirometry Forced expiration volume is 60 – 70% predicted and symptoms are smokers cough or exertional breathlessness.

In moderate Chronic obstructive pulmonary disease is Forced expiration volume is 40 – 50% predicted and exertional breathlessness, cough, sputum and wheeze presented.

In severe Forced expiration volume is 60 – 70% predicted is Forced expiration volume is less than 40 % predicted and exertional breathlessness, cough, sputum, wheeze and swollen legs presented.

Investigation

Investigation of chronic obstructive pulmonary disease is spirometry, pulse x ray of chest (anterior posterior view and lateral view), pulse oxymetry to know the oxygen percentage and haematology (polychythaemia may develop but should not be assumed to be secondary without measurement of P_aO_2), CT scan can be used to quantify the extent and distribution of emphysema.

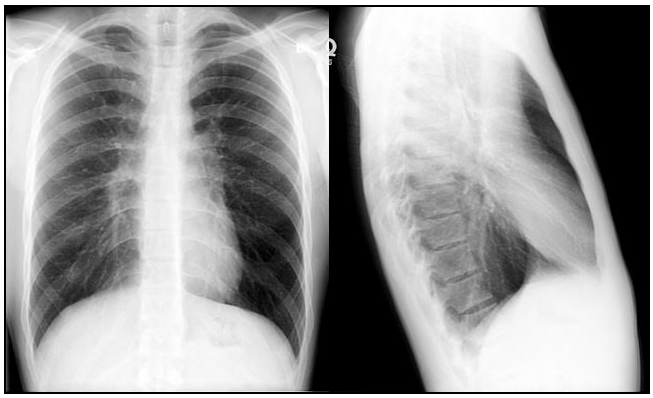

Fig 2: Chest radiograph of chronic obstructive pulmonary disease

Management

Chronic obstructive pulmonary disease can be managed by antibiotics, stop smoking completely/ permanently, bronchodilator therapy, surgical intervention, homoeopathic medication.

Bronchiectasis

It is defined as a ‘abnormal dilation and chronic enlargement of the bronchi/bronchus’. The passageways from the trachea to the alveoli that are the air exchanging parts of the lungs. It may be acquired or less commonly, congenital. Bronchiectasis may occur in a single portion of the lung (localized) or throughout the lungs (diffuse) and is the major lung abnormality of cystic fibrosis. It may have several different contributing factors, such as abnormal cilia, and its course may vary greatly from causing no symptoms to causing death [3, 4].

Etiology of bronchiectasis

- Congenital cause of bronchiectasis are ciliary dysfunction syndromes like primary ciliary dyskinesia (immotilie cilia syndrome), kartageners syndrome, young syndrome, cystic fibrosis and primary hypogammaglobulinaemia.
- Acquired in children causes are pneumonia (complication, whooping cough or measles), primary tuberculosis, foreign body.
- Acquired in adults causes are suppurative pneumonia,

pulmonary tuberculosis, allergic bronchopulmonary aspergillosis and bronchial tumors.

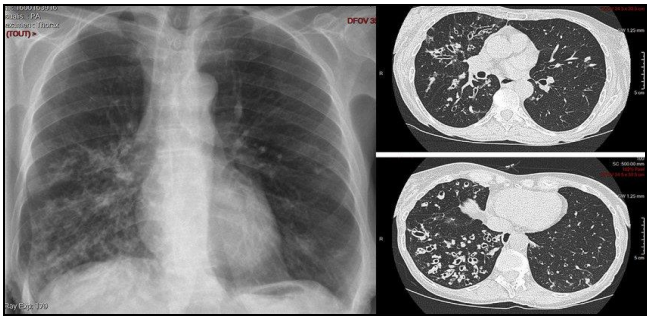

Fig 3: Chest Radiograph and CT images of Bronchiectasis

Pathology

Bronchiectasis cavities may be lined by granulation tissue, squamous epithelium or normal ciliated epithelium. There may also be inflammatory changes in the deeper layers of the bronchial wall and hypertrophy of the bronchial arteries. Chronic inflammatory and fibrotic changes are usually found in the surrounding lung tissue.

Clinical features

Bronchiectasis may occur any part of the lungs, but the more efficient drainage by gravity of the upper lobes usually produces less serious symptoms and complication than the lower lobes. Chronic productive of cough usually aggravated by morning and often brought on by changes of posture. Sputum often copious and persistently purulent in advanced disease (due to accumulation of pus in dilated bronchi/bronchus). Fever, malaise and increased cough, sputum volume when spread of infection causes pneumonia, which is frequently associated with pleurisy. Recurrent pleurisy in the same site often occurs in this condition (due to inflammatory changes in lung and pleura surrounding dilated bronchi). Purulent sputum, haemoptysis. It is called dry bronchiectasis. Associated symptoms are weight loss, anorexia, lassitude, excessive sweating at night time and digital clubbing.

Investigation

In advanced disease the cystic bronchiectasis spaces may be visible. Abnormalities produced by associated pulmonary infection and collapse are evident. A diagnosis of bronchiectasis can only be made with certainty by CT scan. Cilliary function test when patients suspected of having a ciliary dysfunction syndrome.

Management

Bronchiectasis can be managed with postural drainage, antibiotic therapy, surgical treatment and homoeopathic treatment.

Cystic fibrosis

Cystic fibrosis (CF) is the most common, life shortening genetic disease in Caucasians. It affects the transport of salt and water across cells and affects different organs, but lung disease is responsible for the majority of symptoms, burden of care, and lost years of life. The gene that causes the disease has now been identified and sequenced [5, 6].

Epidemiology

Cystic fibrosis affects at least 30,000 people in the United States; between 900 and 1,000 new cases are diagnosed every year (1). One in 29 people of Caucasian ancestry is an unaffected carrier of the CF gene mutation. In the United States, cystic fibrosis occurs at a rate of 1 in 3,400 births. While it occurs in persons of all racial and ethnic backgrounds, it is most common in Caucasians of Northern European ancestry. Historically, half of affected individuals were diagnosed by five months of age, though the average age at diagnosis was five years, and some individuals were not diagnosed until adulthood. In 2010, however, all states began requiring that newborns undergo screening for cystic fibrosis. This should be helpful because early diagnosis and treatment reduce symptoms, improve health, and reduce costs associated with disease complications.

Cystic fibrosis is caused by mutations in the cystic fibrosis transmembrane regulator (CFTR) gene. A recessive genetic disorder, it is inherited when two carrier parents (who have one normal gene and one gene with a mutation) each contributes the abnormal CFTR gene to their child. Thus, the likelihood that two carrier parents will have an affected child is 1:4 for each pregnancy.

The abnormality in the CFTR gene causes a defective CFTR protein to be produced, resulting in abnormal transport of salt (sodium and chloride) and water across cells that line the respiratory, digestive, and genital tracts. This results in a reduction of water in the fluid lining the airways. Diminished water causes the respiratory secretions to become thicker and clog small airways. The stagnant sputum becomes infected as bacteria that are inhaled or brought into the lungs through the mouth become lodged there. Persistent stagnation allows persistent infection and chronic inflammation to develop. Inflammatory cells trapped in the sputum add to its tenacity.

Clinical features

The bronchi dilate and their walls weaken, setting up a condition called bronchiectasis that results in further airflow obstruction. The vicious cycle of airway obstruction, inflammation, and persistent infection leads to a progressive decline in lung function and eventually causes respiratory failure and death. Clogged mucus secretions in the digestive tract can lead to malnutrition and vitamin deficiencies. The genital tract abnormality can lead to infertility in men and women.

Environmental exposures worsen cystic fibrosis lung disease. Children who are exposed to tobacco smoke have lower lung function and more pulmonary exacerbations than those who live in smoke-free environments. High levels of air pollution are associated with an increased rate of adverse pulmonary events.

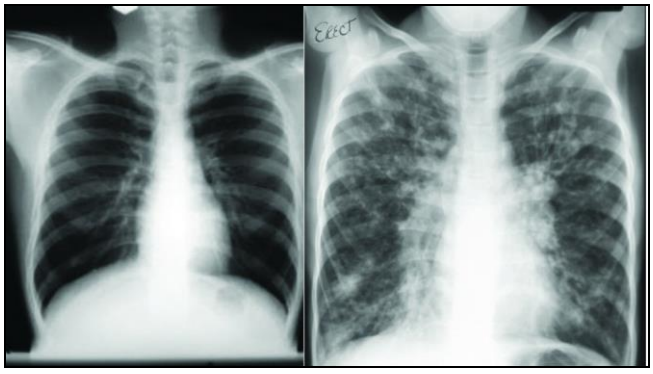

Fig 4: Chest radiograph of cystic fibrosis

Management

All the patients with cystic fibrosis who produce sputum should have regular chest physiotherapy, which should be performed more frequently during exacerbations. Cystic fibrosis can be managed by potential for somatic gene therapy and homoeopathic constitutional remedies.

Pneumonia

Pneumonia is a "lung infection characterized by severe cough and fever mainly resulting from bacteria, virus or fungi". It involving the lung alveoli (air sacs) and can be caused by microbes, including bacteria, viruses, or fungi. It is the leading infectious cause of hospitalization and death in the India and exacts an enormous cost in economic and human terms. Healthy individuals can develop pneumonia, but susceptibility is greatly increased by a variety of personal characteristics. Community acquired pneumonia occurs outside of the hospital (Streptococcus pneumonia), hospital acquired pneumonia occurs contracted by a patient in a hospital at least 48–72 hours after being admitted. It is bacterial infection rather than viral infection. It includes post-operative pneumonia and most common pathogens are gram negative bacilli (staphyloccus) [7].

Pneumonia was described 2,500 years ago by Hippocrates, the father of medicine. Pneumonia occurs commonly in individuals living in their home communities (community acquired pneumonia) as well as in individuals who are hospitalized for other reasons (hospital acquired pneumonia). The lobular Pneumonia is a radiological and pathological term refereeing to homogeneous consolidation (red hepatisation) of one or more lung lobes, often with associated pleural inflammation. Bronchopneumonia referees to more patchy alveolar consolidation associated with bronchial and bronchiolar inflammation often affecting both lower lobes [8].

Stages of pneumonia

Four stages of lobar pneumonia have been described. In the first stage, which occurs within 24 hours of infection, the lung is characterized microscopically by vascular congestion and alveolar edema. Many bacteria and few neutrophils are present. The stage of red hepatization (2-3 d), so called because of its similarity to the consistency of liver, is characterized by the presence of many erythrocytes, neutrophils, desquamated epithelial cells, and fibrin within the alveoli. In the stage of gray hepatization (2-3 d), the lung is gray brown to yellow because of fibrinopurulent exudate, disintegration of RBCs, and hemosiderin. The final stage of resolution is characterized by resorption and restoration of the pulmonary architecture. Fibrinous inflammation may lead to resolution or to organization and pleural adhesions.

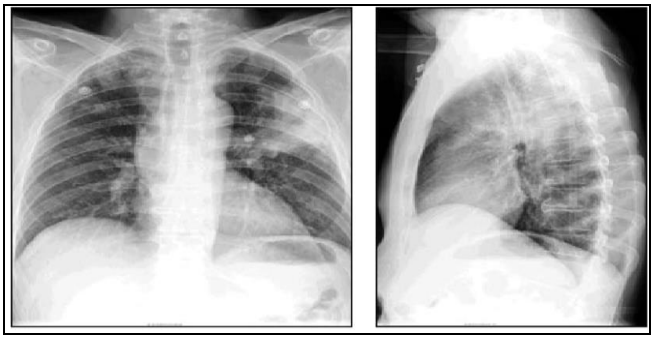

Fig 5: Radiograph of Pneumonia

Clinical features

Patients present with complains of severe cough, high grade fever, malaise, often associated with pleuritic chest pain, which is occasionally referred to the shoulder or anterior abdominal wall. The cough is characterized by short, painful and at first dry, but later becomes productive and may become rust coloured or even frankly blood stained. The sudden onset of rigors due to high grade fever, in children vomiting or a febrile convulsion. Appetite is usually lost and headache is a frequent accompanying symptoms. In advance pneumonia confusion can be an early and dominant problem.

Physical signs tachycardia, hypoxaemia, hypotension, confusion and tachypnoea. Pleurisy often results in diminution of respiratory movements and a pleural rub on the affected side. At a variable time after onset, generally within two days, signs of consolidation appear. The percussion note and high pitched bronchial breath sounds. If a pleural effusion develops, physical signs of fluid in the pleural space are usually found, but bronchial breath sounds can persist and the presence of an empyema may be suspected only from the recurrence or persistence of pyrexia.

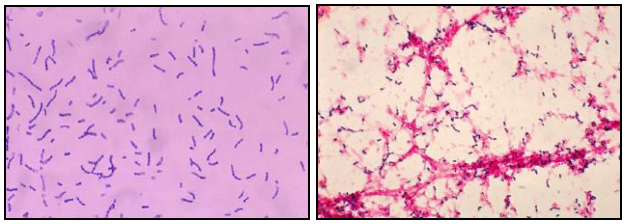

Fig 6: Gram positive diplococci characteristic of strep. Pneumonia

Investigations

In lobar pneumonia, the chest radiograph shows a homogeneous opacity localized to the affected lobe or segment; this usually appears within 12 -18 hours of the onset of the illness. Radiological examination is also particularly helpful if a complication such as pleural effusion, intrapulmonary abscess formation or empyema is suspected.

Differential diagnosis

- Pulmonary infraction like bacterial pneumonia.
- Pulmonary/pleural tuberculosis like
- Pulmonary oedema, especially if unilateral and localized.
- Inflammatory conditions below the diaphragm condition such as cholecystitis, peptic ulcer, acute pancreatitis, hepatic amoebiasis.
- Pulmonary eosinophilia, wegeners granulomatosis.

Management

Pneumonia can be manage by oxygen should be administered to all hypoxaemic patients, antibiotic treatment, mild analgesics for pleural pain, physiotherapy, homoeopathic medicines.

Tuberculosis

Tuberculosis remains one of the major global health threats leading to morbidity and mortality. One in three persons across the world representing 2–3 billion individuals are known to be infected with Mycobacterium Tuberculosis (M. Tuberculosis) of which 5–15% are likely to develop active.

In 2014, an estimated 9.6 million people fell ill due to tuberculosis, around 1.5 million people died from the disease including 1.1 million HIV negative persons and 400,000 HIV patients. While tuberculosis is present in every country majority of tuberculosis sufferers live in low income and middle income countries especially in regions such as Sub Saharan Africa and South East Asia. Over the past decade, significant progress has been made towards tuberculosis control with most of the tuberculosis targets set as part of the Millennium Development Goals having been achieved. In all, effective diagnosis and treatment of tuberculosis has been estimated to have saved over 40 million lives between 2015 and 2019. The End tuberculosis strategy serves as the key guide for countries to reduce tuberculosis deaths by 90% by 2030 as well as achieve an 80% reduction in tuberculosis incidence rate compared with 2015. In this respiratory disorder paper, I provide a general overview of tuberculosis by highlighting the basics [9].

Pathology

The initial primary tuberculosis usually formed in the lung, but occasionally in the tonsil or alimentary tract, especially the ileocaecal region. Primary infection differs from subsequent infections in that the primary focus in lung, tonsil or bowel is almost invariably accompanied by caseous lesions in the regional lymph nodes, such as the mediatinal, cervical or mesenteric groups respectively. Primary infection and the associated lymph node lesions heal and calcify [10].

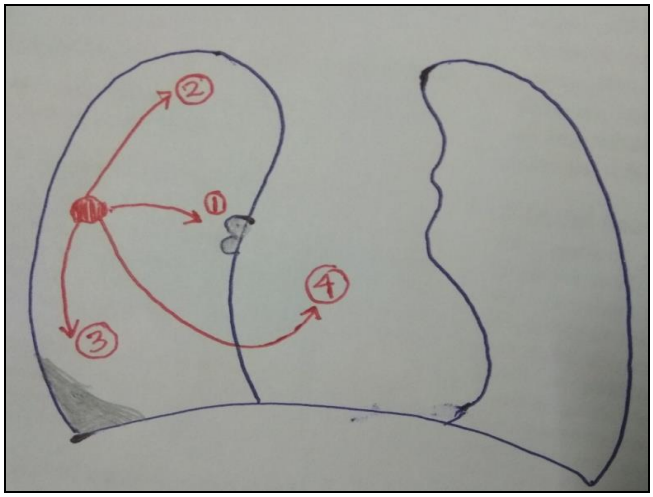

Fig 7: Primary pulmonary tuberculosis

(1) Spread from primary focus to hilar and mediastinal lymph glands to form the primary complex (2) Direct extension of the primary focus – Progressive pulmonary tuberculosis (3) spread to the pleura pleural effusion (4) Blood borne spread: Pulmonary, skeletal, renal, genitourinary; massive spread- military tuberculosis and meningitis.

In a few, healing, particularly in lymph nodes, is incomplete and viable tubercle bacilli may enter the blood stream. Lesions more common in the lungs, bones, joints and kidneys and lesions may develop months or even years after primary infection. Sometimes primary infections do not heal. Infection may also be carried by lymphatics from tuberculosis mediastinal lymph nodes to the pleura or pericardium, with the production of tuberculosis pleurisy or pericarditis. Rarely a caseous tuberculosis focus ruptures in to a vein and produces acute dissemination throughout the body, a condition known as *acute military tuberculosis*. Meningitis often complicates this condition. *Post primary pulmonary tuberculosis* characteristic pathological feature of which is the tuberculous cavity, formed when the caseated and liquefied centre of a tuberculous pulmonary lesion is discharged in to a bronchus. Blood bonrne dissemination to other organs is uncommon in post primary pulmonary tuberculosis.

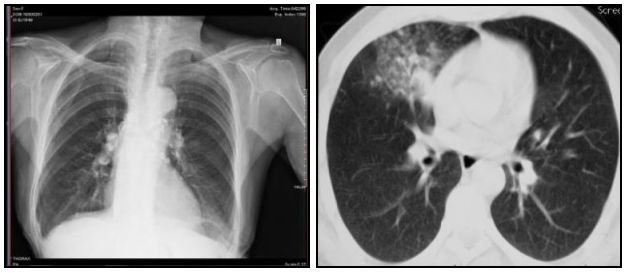

Fig 8: Radiograph & C T of Pulmonary tuberculosis

Clinical features

Persistent cough, haemoptysis, pleural pain not associated with an acute illness, spontaneous pneumothorax, lethargy, weight loss.

Investigation

Microscopy

Sputum smear microscopy still remains the basis for diagnosis of tuberculosis in developing countries. Sputum smears can be screened using fluorochrome stains such as an auramine stain where mycobacteria appear as fluorescent rods against a dark background using an ultraviolet light microscope. Other specimens should be stained using the Ziehl-Neelson method — mycobacteria are seen as pink rods against a blue or green background.

Culture

Culture still remains the gold standard for diagnosis of tuberculosis, and it also permits the diagnosis of drug resistance, including the emerging mutations. Traditional egg based (Lowenstein Jensen) and agar based (Middlebrook 7H10/11) methods are widely used. Patients with pulmonary disease should have three sputum samples sent for microscopy and culture. If sputum is not expectorated, an induced sputum, broncheoalveolar lavage or gastric aspirate can be examined. Gastric aspirates are particularly useful in diagnosing children. Other specimens taken depend on the sites affected, but may include cerebrospinal fluid (CSF), blood, peritoneal and pericardial fluid, early morning urine, lymph node aspirates or tissue samples. CSF should be tested for cell count, protein and glucose because tuberculous meningitis is associated with an elevated lymphocyte count, high protein and low glucose.

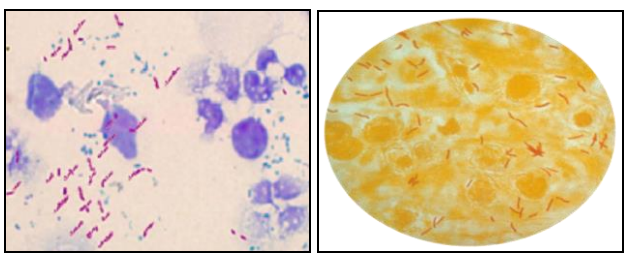

Fig 9: Examination of sputum

Tuberculin skin testing

Popularly known as Mantoux test involves injecting the purified protein derivative (PPD) of mycobacterial tuberculosis intradermally in the forearm and the resulting reaction is read after 48–72 hours. A positive skin test is indicated by a skin reaction at the point of the injection. A blood test has recently been developed which measures interferon g released from T cells in response to stimulation with mycobacterial antigens. Studies using ESAT-6 and CFP-10, two antigens absent from the BCG vaccine strain, have shown promising results for the diagnosis of active and latent infection.

Management

BCG is a strain of bovine tuberculin of low virulence which is used for intradermal vaccination (0.1 ml of reconstituted freeze dried vaccine), conferring protection for up to 7 years. Vaccination reduces the incidence of pulmonary tuberculosis in young adults by 80% and minimizes the risk of serious disseminated disease – military tuberculosis and tuberculous meningitis.

Chemoprophylaxis: healed tuberculous scars may still contain viable bacteria and if cellular immunity is suppressed for any reason there may be recrudescence of disease.

Chemotherapy is the mainstay of modern treatment of tuberculosis, although the occasional patient still requires surgery for drainage of an empyema, caseatingtuberculous lymph nodes or more rarely, resection of bronchiectatic areas of the lung, or emergency surgery to prevent spinal paraplegia, antituberculosis drugs (ATD) and homoeopathy medicines used in tuberculosis.

Bronchial Asthma

Bronchial asthma is heterogeneous pulmonary disorder characterized by recurrent episodes of cough, breathlessness and wheezing, which may resolve spontaneously or after the use of bronchodilator medication. The global prevalence of asthma is anticipated to be approximately 4.5 percentages. There are about 334 million patients with asthma affecting all age groups, across the world. The prevalence of asthma has increased over time and an additional 100 million people worldwide are expected to develop asthma by the year 2025. The prevalence of asthma has increased over time and an additional 100 million people worldwide are expected to develop asthma by the year 2025. Although asthma is a major health problem in the world, there are some important issues, particularly its management. Asthma is seen in all ages of life, from earliest infancy up to old age. It has observed that males were more prone to asthma than the female [11, 12].

Etiology

Heredity: It is estimated that about 30 percent of patients will give us a family history of allergy and asthma or either of these.

Allergies: Allergies can be allotted in large number of patients for causing or precipitating asthmatic assaults. Usually, a massive exposure to allergens is followed by an acute asthmatic attack. In immediate onset of asthma, the symptoms of asthma (acute occur within a few minutes of exposure.

Environmental cause: winds, rains, sudden changes in the climate aggravate allergic manifestations. Physical agents like colds, hearts etc., do start an allergic phenomenon and hence could be called as pseudo allergens.

Emotional: Emotional disturbances do play a vital role in the llife of an asthmatic.

Infections: Repeated upper respiratory infections are the main precipitating factors in m any cases.

Type of asthma

Extrinsic is hereditary disposition. It starts early in life and serum levels of IgE are elevated. It occurs due to pollens of trees, grass and weeds.

Intrinsic asthma: it is also known as idiopathic asthma. Serum IgE levels are normal. It usually starts late in life and perennial symptoms are common.

Catarrhal asthma/millers asthma is seen in association with acute or chronic catarrhal disorders, the mucous membrane of the throat and bronchial tubes being in a n altered state, in severe cases badly inflamed.

Hay asthma is a type of asthmatic breathing that occurs in association with acute bronchial irritation and catarrh. It is summer catarrhal affection that passes under the name of rose cold or hay fever.

Millers asthma is frequently applied to spasmodic group or laryngesmus stridulous and by some authors is still mentioned as a type of asthma occurring children.

Atopic asthma is definite antigenic etiology. There is a definite history of allergy and through the hyper sensitivity tests one finds that the patient shows a positive result.

Non-atopic asthma is no allergic factor in this kind of asthma. Intrinsic asthmatics when tested for IgE levels would not have a rise in IgE levels.

Pathophysiology

Current theoris include early exposure toaero allergens, early viral infections, diet or paradoxically, fever childhood infections resulting from improved public health standards.

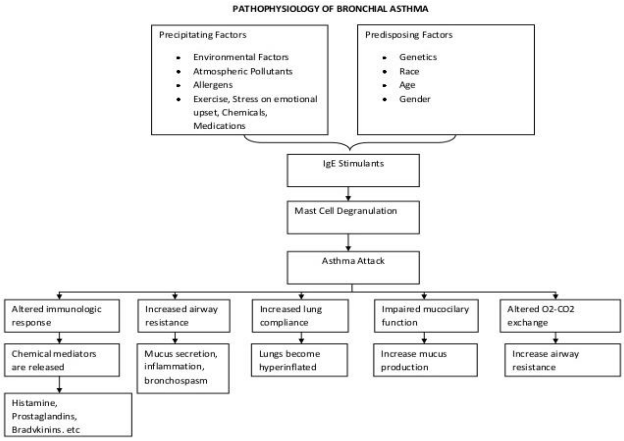

Fig 10: Pathophysiology of asthma flow chart

Clinical features

In episodic asthma paroxysms of wheeze and dyspnoea occur at any hour of the day or night are of sudden onset and may be preceded by a feeling of tightness in the chest. Expiration is exhausting, while inspiration is short and gasping. Patient adopts to an upright position, fixing the shoulder girdle to assist the accessory muscles of expiration. In severe attacks there is tachycardia, pulsus paradoxus and central cyanosis. The symptoms usually sets in suddenly and generally at night, occurring, as a rule, without the least

premonition, although it may be attendee in children by a stage of cold, cough and ordinary catarrhal symptom prior to the development of asthmatic breathing. The eyes are prominent and starring and the patient is compelled to lie or sit with his mouth partly open in order to get sufficient breath for his needs. The temperature usually not elevated, and may even be subnormal. The surface is usually cold and clammy and sometimes cyanosis is so pronounced that a fatal issue is feared.

Investigation

Completed blood picture, x- ray, sputum examination, ECG, pulmonary function test, arterial blood gases, measurement of serum IgE, detection of IgE antibody etc.,

Management:

In acute severe asthma has to give oxygen, high doses of inhaled beta 2 adrenoreceptor agonists, systemic corticosteroids. In chronic asthma can be managed with homoeopathic medicines.

Pleural effusion

Pleural effusion is used when serous fluid accumulates in the pleural space. The passive transudation of fluid in to the pleural cavity occurs in cardiac failure and in conditions causing hypoproteinaemia such as nephritic syndrome, liver failure and severe malnutrition. Pleural effusion may be unilateral or bilateral. Bilateral effusion often occur in cardiac failure, but are also seen in much less common disorders such as the connective tissue diseases and hypoproteinaemia [13, 14].

Etiology

There are many causes of pleural effusions. The following is a list of some of the major causes: pneumonia, tuberculosis, pulmonary infract, malignant disease, subdiaphragmatic disorder (subphrenic abscess, pancreatitis etc.,),crdiac failure, hypoproteinaemia (nephritic syndrome, liver failure, malnurtrition), connective tissue disease, acute rheumatic fever, post myocardial infarction syndrome, meigs syndrome (ovarian tumour plus effusion), myxoedema, uraemia, asbestos related benign pleural effusion, yello nail syndrome.

Symptoms

Chest pain, difficulty breathing, painful breathing (pleurisy), cough is either dry or productive. Deep breathing increase pain, fever, chills, loss of appetite,

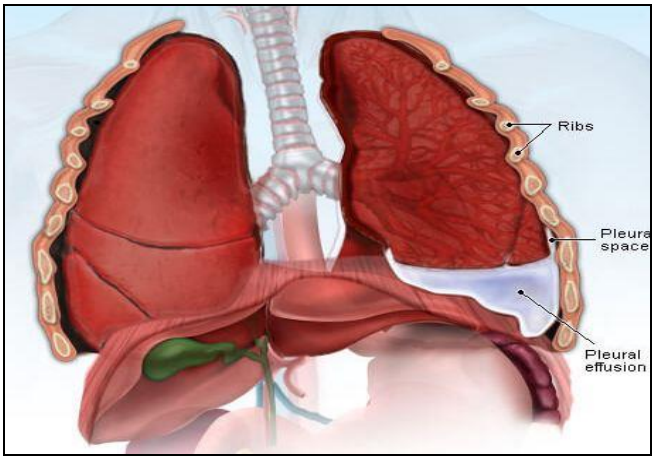

Fig 11: Pleural effusion

Investigations

Radiological examination shows a dense uniform opacity in the lower and lateral parts of the hemithorax, shading off above and medially in to translucent lung (see figure 12). Occasionally the fluid is localized below the lower lobe (subpulmonary effusion), the appearances simulating an elevated hemidiaphragm. A localized opacity may be seen when the effusion is localated – for example in an inter lobar fissure. Ultrasonography helps to localize an effusion prior to aspiration and pleural biopsy. Pleural aspiration can absolute proof that an effusion is present can be obtained only by the aspiration fluid. Pleural biopsy is always indicated whenever a diagnostic aspiration of pleural fluid is performed because the chances of obtaining a diagnosis from pleural biopsy material are much greater than by examination of the pleural liquid alone.

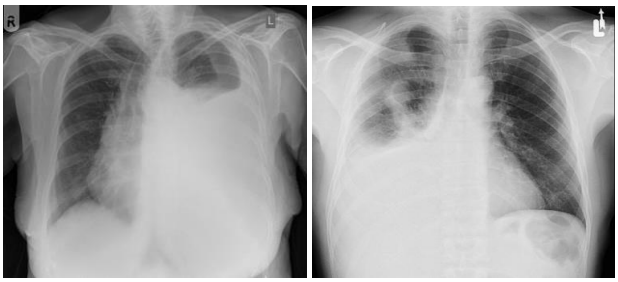

Fig 11: Radiograph of Pleural effusion

Management

Aspiration of pleural fluid may be necessary to relieve breathlessness. It is inadvisable to remove more than one liter on the first occasion because re expansion pulmonary oedema occasionally follows the aspiration of larger amounts.

Empyema

The term empyema defines “pus in the pleural space”, gram-positive, or culture from the pleural fluid. Empyema is usually associated with pneumonia but may also develop after thoracic surgery or thoracic trauma. In the United States, there are approximately 32,000 cases per year. Empyema is associated with elevated morbidity and mortality, around 20% to 30% of patients affected will either die or required further surgery in the first year after developing empyema. Early intervention is crucial in the management of empyema [15, 16].

Etiology

Around 20% of patients with pneumonia will develop a parapneumonic effusion that may lead to empyema. Seventy percent of patients with empyema have parapneumonic effusion, the other 30% of cases are related to trauma, post thoracic surgery, esophageal ruptures, or cervical infections, and a small number are not related to previous pneumonia or intervention, this is known as primary empyema. Also, comorbidities of the patients need to be taken into consideration. For community-acquired empyema, gram positive bacteria are more common, especially S*treptococcus* species. In this setting, the presence of gram-negative bacteria has been associated with increased comorbidities of patients with alcohol abuse, gastroesophageal reflux disease (GERD), and diabetes. In hospital acquired empyema, *Staphylococcus aureus* are common.

Pathophysiology

Development of empyema can be described in a sequence of events. During an inflammatory process such as pneumonia, there is an increase in fluid production in the pleural cavity known as the exudates stage. As the disease progresses microorganisms, usually bacteria, can colonize the fluid and generated an empyema. This fluid is characterized by elevated lactate dehydrogenase, proteins, neutrophils, and dead cells. Macroscopically is a thick opaque fluid found in the fibrinopurulent stage. After the resolution of the infection and as a consequence of the inflammation, there is a process of fibrosis that can lead to restriction of the lung parenchyma. Appropriate and early intervention is vital to decrease complications and mortality.

Clinical features

The presentation may be similar to pneumonia, and cough, sputum production, fever, and pleuritic type chest pain may be present. Patients with empyema may have symptoms for a more extended period. Research has shown that patients presented after a median of 15 days after the onset of symptoms. On physical exam there may be dullness to percussion on the affected area, egophonia, increase palpable fremitus, and fine crackles.

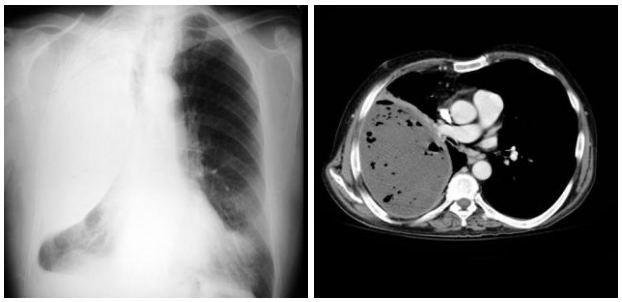

Fig 12: Radiological & CT of Empyema

Investigation

To evaluate for the presence of any pleural effusion, the first test that should be ordered is a chest x-ray. It is a widely available and simple test, but it is not 100% sensitive. A certain amount of fluid needs to be present to be detected, usually 75 ml in a lateral view, and approximately 175 ml in an anterior view. On an x-ray, some of the characteristics of a pleural effusion are blunted due to costodiaphragmatic angles and lungs filled with radiolucent fluid. If an effusion is suspected with the chest x-ray, the next step is an ultrasound. Ultrasound is increasingly common because of its benefits, namely because it is widely available, it can be done at a patient's bedside, it is more sensitive at identifying pleural effusions than an x-ray, it allows differentiation between parenchyma and pleural fluid, and it also has a therapeutic use. Ultrasound can be useful in guiding a chest tube placement during thoracentesis. CT scan of the chest must be done in patients with empyema. It may be an alternative option after a chest -ray or ultrasound. CT scan ideally is done with intravenous (IV) contrast to enhance the pleura. CT scan can also be diagnostic and therapeutic, thoracentesis and tube thoracotomy can be performed under this modality. Some of the characteristics on CT scan are thickening of the pleura (present in approximately 80% to 100% patients), pleural enhancement, split pleural sign, bubbles in the absence of tube drainage, and septations. With a CT scan practitioners can better assess the lung parenchyma and the position of a chest tube.

Management

Treatment of empyema usually involves medical and surgical treatment. In community-acquired empyema, use antibiotics. Antibiotic should be given for 2 to 6 weeks, depending on patient response, source control, and organism. Tube thoracostomy is the most common type of drainage, bore tube versus smaller tubes have not shown any difference regarding mortality and prognosis, but bigger tubes are associated with more pain. For this reason, small tubes are frequently placed. The position of the tube should be confirming with an x-ray or CT scan. Lack of clinical improvement in the first 24 hours is usually related to tube malposition or blockage. Blockage of the chest tube can be prevented with frequent flushing, but the necessary amount and frequency of this process is unclear. Any indication of a persistent fluid or other locations should be addressed with more aggressive therapy including a larger tube, more tubes, or surgery. The chest tube can usually be removed when the daily production of pleural fluid is proximal 350 ml/day or less.

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