**St Peter’s Institute of Pharmaceutical Sciences**

**Course: Bachelor of Pharmacy**

**Subject: PATHOPHYSIOLOGY (THEORY)**

**Subject Code:** BP 204T

**CHRONIC OBSTRUCTIVE PULMONARY DISEASE**

COPD is an obstruction to the airflow at any level from trachea to the smallest airways resulting in functional disability of the lungs.

In COPD, the airway obstruction is due to following

* The decrease in the airways and air sacs elasticity.
* Inflammation of airway walls.
* The more production of mucus in airways causes clog.

**Pathophysiology**

Toxic gases cause inflammatory response of the lungs

Inflammation occurs in airways, parenchyma, and pulmonary vasculature

Narrowing of the airway takes place

Destruction of parenchyma leads to emphysema

Lung parenchyma destruction leads imbalance of proteinases/antiproteinases.

Pulmonary vascularchanges: Thickening of vessels- Collagen deposit- destruction of capillary bed

Mucus hypersecretion(cilia dysfunction, airflow limitation,corpulmonale(RVF)

Chronic cough and sputum production

**The following entities are included in COPD**:

I. Chronic bronchitis

II. Emphysema

III. Bronchial asthma

IV. Bronchiectasis

V. Small airways disease (bronchiolitis)

**CHRONIC BRONCHITIS**

Chronic bronchitis is a condition causes persistent cough with expectoration for minimum 3 months to one or two more consecutive years. The main cause of cough is due to over secretion of mucus.

**ETIOPATHOGENESIS**

Cigarette smoking, atmospheric pollution and other contributory factors are occupation, infection, familial and genetic factors.

**Smoking**

Chronic smoker more than 10 times per day.

i) It impairs ciliary movement.

ii) Alveolar macrophages function is inhibited.

iii) It further leads to hypertrophy and hyperplasia of mucus-secreting glands.

iv) It causes obstruction of small airways.

v) It mainly stimulates the vagus nerve and leads to bronchoconstriction.

**Atmospheric pollution**

Some of the atmospheric pollutants which increase the risk of developing chronic bronchitis are sulphur dioxide, nitrogen dioxide, particulate dust and toxic fumes.Sudden airway constriction in response to inhaled irritants.

Irritants irrritate the airway

Excess mucus production

Inflammation

Increase in number mucus secreting glands and goblet cells to

Ciliary function is reduced

More mucus production

The bronchial walls become thickens, lumen gets narrowed and airway gets blocked with mucus

Alveoli becomes damaged and fibrosed

Alter function of alveolar macrophages

Infection

**CLINICAL FEATURES**

1. Persistent cough

2. Recurrent respiratory infections are common.

3. Dyspnoea.

4. Patients are called ‘blue bloaters’ due to cyanosis andoedema.

5. Features of right heart failure (corpulmonale) are common.

6. Chest X-ray shows the evidence of enlarged heart.

7. Shortness of breath, cough,Diarrhea, vomiting, stuffy or runny nose,fever, body aches, fatigue (tiredness), sore throat.

8. Chronic symptoms: chest discomfort, wheezing, and coughing.

Diagnosis: ABG analysis, CBC, lung function tests, chest x ray, Mucus -to see whether you have a bacterial infection.

**EMPHYSEMA**

Pulmonary emphysema is the condition where the combination of dilatation of air spaces distal to the terminal bronchioles and the destruction of the walls of dilated air spaces.

**Pathophysiology:**

* Alveoli are destroyed
* The alveolar surface area contact with capillaries decreases causing dead spaces leads to hypoxia.
* Carbon dioxide increases in arterial blood causes respiratory acidosis

**Classification of ‘true emphysema’ and ‘overinflation**

**A. TRUE EMPHYSEMA**

1. Centriacinar emphysema

2. Panacinar(panlobular) emphysema

3. Paraseptal emphysema

4. Irregular(para-cicatricial) emphysema

5. Mixed(unclassified) emphysema

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5. Mixed(unclassified) emphysema

**DIAGNOSIS**: History, PFT, Spirometry-to find out airflow obstruction, ABG analysis, CT scan of the lung.Screening of alpha antitrypsin deficiency, X-ray radiography may aid in the diagnosis.

**BRONCHIECTASIS**

Bronchiectasis is a condition in which dilatation of the bronchi and bronchioles more than 2mm diameter, and further develops to secondary inflammatory weakening of the bronchial walls.

**Clinical manifestation:** Persistent cough with expectoration of copious amounts of foul-smelling, purulent sputum.

**ETIOPATHOGENESIS**

Endobronchial Infection leads to obstruction by foreign bodies.

Neoplastic growth or enlarged lymph nodes causes resorption of air distal and further leads to obstruction.

These 2 mechanisms

1. **Hereditary and congenital factors**:

i) Congenital bronchiectasis

ii) Cystic fibrosis

iii) Hereditary immune deficiency diseases

iv) Immotile cilia syndrome

v) Positive family history

**2. Obstruction Post-obstructive bronchiectasis**: Causes endobronchial tumours, foreign bodies, and compression.

**CLINICAL FEATURES:** chronic cough with foulsmelling sputum production, haemoptysis, recurrent pneumonia, and sinusitis.

**SMALL AIRWAYS DISEASE**

Bronchiolitis and bronchiolitis obliterans are the condition of inflammation affecting the small airways.

**Etiologic factors**: viral infection (frequently adenovirus and respiratory syncytial virus), bacterial infection, fungal infection, inhalation of toxic gases and aspiration of gastric contents.

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