CHRONIC BRONCHITIS

INTRODUCTION

- It is diagnosed on clinical basis:
  - Defined as presence of a persistent productive **cough for at least 3** consecutive **months** in at least **2** consecutive **years**.
- The cough is caused by over secretion of mucus.
- Quite frequently, chronic bronchitis is associated with emphysema and termed as chronic obstructive pulmonary disease(COPD).

ETIOPATHOGENESIS

- Common among cigarette smokers and urban dwellers in smog-ridden cities.

- Distinctive feature of chronic bronchitis is,
  - Mucus **hypersecretion** beginning in large airways
- Most important etiologic factors for majority of cases of chronic bronchitis are: cigarette smoking and atmospheric pollution.
- Other contributory factors are occupation, infection, familial and genetic factors.

PATHOGENESIS

a. **Most important cause is cigarette smoking**

Prolonged cigarette smoking impairs ciliary movement.

It inhibits the function of alveolar macrophages.

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

It causes considerable obstruction of small airways & stimulates the vagus causing bronchoconstriction.

b. Environmental irritants induce:

- Hypertrophy of mucous glands in trachea and bronchi
- Increase in mucin-secreting goblet cells in smaller airways
- Cause inflammation
- Some of atmospheric pollutants that increase the risk of developing chronic bronchitis are sulfur dioxide, nitrogen dioxide, particulate dust and toxic fumes.

c. Occupation

Workers engaged in certain occupations like in cotton mills (byssinosis), plastic factories etc are exposed to various organic or inorganic dusts which contribute to disabling chronic bronchitis in such individuals.

d. Infection Bacterial, viral and mycoplasmal infections do bronchitis. Cigarette smoke, however, predisposes to infection responsible for acute exacerbation in chronic bronchitis.

e. Familial and genetic factors

There are some poorly defined familial tendency and genetic predisposition to develop chronic bronchitis.

However, it is more likely in nonsmoker family members who are exposed to air-pollution and hence have increased blood levels of carbon monoxide.

PATHOGENESIS

- Airflow obstruction in chronic bronchitis results from:
  - small airway disease, induced by mucous plugging, inflammation, and bronchiolar wall fibrosis,
  - coexistent emphysema
- Small airway disease *(**chronic bronchiolitis**)*
  - early, mild airflow obstruction
- With significant airflow obstruction:
  - Almost always is complicated by emphysema in later stage

- Effects of environmental irritants:
  - release of cytokines IL-13 from T cells
  - Mucin production
- Tobacco causes:
  - Production of neutrophil elastase
- Microbial infection:
  - Often present but has secondary role

MORPHOLOGY

Gross:

- Mucosal lining of the larger airways:
  - the bronchial wall is thickened, Hyperemic and oedematous
  - Covered by mucinous or mucopurulent secretions
- Smaller airways:
  - filled with secretions

MICROSCOPY

- Diagnostic feature in larger airways:
  - **Enlargement of the mucus secreting glands**
- Magnitude of increase in size is assessed by:
  - **Increased Reid Index**: Ratio of thickness of submucosal gland layer to that of bronchial wall (**Reid index**— normally 0.4).
  - The increase in thickness can be quantitatively assessed by micrometer lens or by morphometry
- Inflammatoy cells: Neutrophils, macrophages
- The bronchial epithelium may show squamous metaplasia and dysplasia.

MICROSCOPY

- **Chronic bronchiolitis:**
  - Goblet cell metaplasia, mucous plugging, inflammation, and fibrosis
- In severe cases:
  - Complete obliteration of lumen by fibrosis seen **(bronchiolitis obliterans).**
- Emphysematous changes often coexist.

CLINICAL FEATURES

- Variable features
- In some patients, cough and sputum production persist indefinitely without ventilatory dysfunction, initially beginning in a heavy smoker with ‘morning catarrh’ or ‘throat clearing’ which worsens in winter.
- Recurrent respiratory infections.
- Features of right heart failure (corpulmonale) are common.
- Chest X-ray shows enlarged heart with prominent vessels.

- Dyspnoea is generally not prominent at rest but is more on exertion.
- Progressive disease:
  - pulmonary hypertension, sometimes leading to cardiac failure

- Absence of increased respiratory drive the patient retains carbon dioxide, becoming hypoxic and often cyanotic/ edematous so called **“blue bloaters”.**