Lecture (3)

Chronic bronchitis and pulmonary embolism

Chronic bronchitis is common among cigarette smokers and urban dwellers in smog-ridden cities; some studies indicate that 20% to 25% of men in the 40- to 65-year-old age group have the disease. The diagnosis of chronic bronchitis is made on clinical grounds: it is defined by the *presence of a persistent productive cough for at least 3 consecutive months in at least 2 consecutive years.* In early stages of the disease, the productive cough raises mucoid sputum, but airflow is not obstructed. Some patients with chronic bronchitis may demonstrate hyperresponsive airways with intermittent bronchospasm and wheezing. A subset of bronchitic patients, especially heavy smokers, develop chronic outflow obstruction, usually with associated emphysema.

PATHOGENESIS:

The distinctive feature of chronic bronchitis is **hypersecretion of mucus,** beginning in the large airways. Although the single most important cause is cigarette smoking, other air pollutants, such as sulfur dioxide and nitrogen dioxide, may contribute. These environmental irritants induce **hypertrophy of mucous glands** in the **trachea and main bronchi**, leading to a marked increase in mucin-secreting goblet cells in the surface epithelium of smaller bronchi and bronchioles.

In addition, these irritants cause inflammation with infiltration of CD8+ lymphocytes, macrophages, and neutrophils. In contrast with asthma, **there are no eosinophils in chronic bronchitis**.

MORPHOLOGY

The mucosal lining of the larger airways usually is **hyperemic (**is the increase of blood flow to different tissues in the body) **and swollen** by edema fluid. It often is covered by a layer of mucinous **secretions.** The smaller bronchi and bronchioles also may be filled with similar secretions.

On histologic examination, the diagnostic feature of chronic bronchitis in the trachea and larger bronchi is **enlargement of the mucus-secreting glands** (Fig. 3–1).

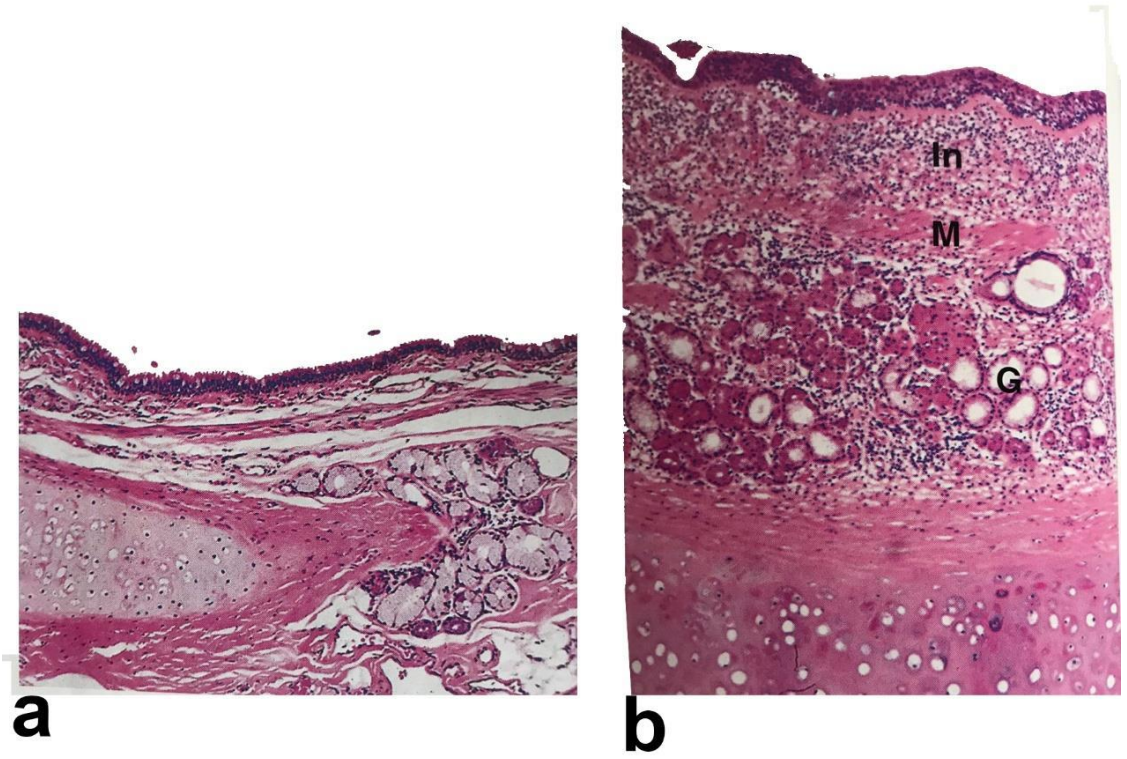

Figure 3–1. (a).Normal bronchial wall, (b). Bronchial wall in chronic bronchitis

Three factors contribute to the increased thickness of the bronchial wall:

1- Infiltration of the submucosa by chronic inflammatory cells (**In**).

2- Marked hypertrophy of mucosal smooth muscle (**M**).

3- Marked hyperplasia of the mucous glands (**M**). with production mucus.

The magnitude of the increase in size is assessed by the ratio of the thickness of the submucosal gland layer to that of the bronchial wall (the **Reid index** normally 0.4). Inflammatory cells, largely mononuclear but sometimes admixed with neutrophils, are frequently present in variable density in the bronchial mucosa.

"The Reid Index is a mathematical relationship that exists in a human bronchus section observed under the microscope. It is defined as ratio between the thickness of the submucosal mucus secreting glands and the thickness between the epithelium and cartilage that covers the bronchi."

Chronic bronchiolitis (small airway disease),

Characterized by goblet cell metaplasia, mucous plugging, inflammation, and fibrosis, is also present. In the most severe cases, there may be complete obliteration of the lumen as a consequence of fibrosis (bronchiolitis obliterans). It is the submucosal fibrosis that leads to luminal narrowing and airway obstruction. Changes of emphysema often co-exist.

Pulmonary Embolism, Hemorrhage, and Infarction:

When a blood clot gets caught in one of the arteries that go from the heart to the lungs, it's called a pulmonary embolism (PE). The clot blocks the normal flow of blood.

This blockage can cause serious problems, like damage to your lungs and low oxygen levels in your blood. The lack of oxygen can harm other organs in your body, too. If the clot is big or the artery is clogged by many smaller clots, a pulmonary embolism can be fatal.

Pulmonary embolisms usually travel to the lungs from a deep vein in the legs (deep vein thrombosis DVT). These clots develop when the blood can't flow freely through the legs because r body is still for a long time, during a long flight or drive. It might also happen if patient on bed rest after surgery or illness.

Clinical Features

The clinical consequences of pulmonary thromboembolism are summarized as follows:

- Most pulmonary emboli (60% to 80%) are clinically silent because they are small; the embolic mass is rapidly removed by fibrinolytic activity, and the bronchial circulation sustains the viability of the affected lung parenchyma until this is accomplished.
- In 5% of cases, sudden death, acute right-sided heart failure (acute cor pulmonale), or cardiovascular collapse (shock) may occur typically when more than 60% of the total pulmonary vasculature is obstructed by a large embolus or multiple simultaneous small emboli.

- Obstruction of relatively small to medium pulmonary branches (10% to 15% of cases) that behave as end arteries causes pulmonary infarction when some element of circulatory insufficiency is present. Typically, persons who sustain such infarction manifest dyspnea.
- In a small but significant subset of patients (accounting for less than 3% of cases), recurrent multiple emboli lead to pulmonary hypertension, chronic right-sided heart strain (chronic cor pulmonale), and, in time, pulmonary vascular sclerosis with progressively worsening dyspnea.