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Emphysema and Chronic Bronchitis

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The diagnosis of chronic obstructive pulmonary disease (COPD) is predicated upon the recognition of its two major forms: emphysema and chronic bronchitis. These distinct entities represent different manifestations of COPD, although they frequently coexist in the same individual. Physiologic impairment occurs as a consequence of the impediment of airflow and air trapping as measured by pulmonary function testing. Other forms of airflow obstruction are discussed in the chapters on bronchial obstruction (Chapter 5), asthma (Chapter 15), and pathology of small airways (Chapter 25).

Chronic obstructive pulmonary disease is a major cause of morbidity and mortality throughout the world. It has been estimated that 5% to 15% of adults in industrialized nations suffer from COPD, and COPD is the fourth leading cause of death in the United States, exceeded only by cardiovascular diseases, cancer, and cerebrovascular diseases. By 2020, COPD may become the third leading cause of death worldwide, commensurate with the rising rates in lung cancer. Chronic obstructive pulmonary disease is almost entirely preventable. The major cause by far is cigarette smoking, but air pollution (including passive smoking) and occupational exposures are also contributing factors.

The early recognition of these disorders is important, not only in terms of prevention of further progression, but because advances in treatment developed over the past two decades can reduce the incidence of exacerbations of COPD. The mainstay of treatment is long-term bronchodilators and inhaled corticosteroids.

Emphysema is a morphologic condition best observed in lung specimens that have been fixed, either by inflation fixation followed by freeze-drying or by intrabronchial instillation of formalin under pressure. When this methodology is employed, the prevalence of emphysema is much greater than the clinical prevalence of COPD would imply. Approximately 50% of smokers have detectable emphysema in inflation-fixed lungs, with an average extent of 25% of surface area as determined by point counting. Emphysema is also detected in approximately 16% of nonsmokers, with a mean extent of only 7%. Regression analysis of the extent of emphysema and age of death indicates that emphysema begins at about the age of 18 years, which coincides with the average age of initiating cigarette smoking (Fig. 24.1).

Chronic bronchitis, in contrast with emphysema, has a clinical definition (see below). The morphologic correlate of this clinical syndrome is mucous gland hyperplasia and an increase in the proportion of goblet cells in the surface mucosa. The pathologic findings, clinical correlates, etiology, and pathogenesis of chronic bronchitis and emphysema are discussed in more detail in the following sections.

Chronic Bronchitis

Definition

Chronic bronchitis is defined clinically as a productive cough for at least 3 months out of the year for 2 consecutive years, with no other apparent explanation. This clinical definition excludes patients with cystic fibrosis and other causes of bronchiectasis, which show similar histologic features in the large airways (see Chapter 5).

Pathology

Gross examination of bronchi in patients with chronic bronchitis may show mucous plugging and prominence of bronchial mucosal pits. The latter represent the orifices of bronchial mucous glands, which are dilated from mucous distention. In addition, cross-sectional views may show thickening of the bronchial walls with narrowing of the lumen (Fig. 24.2).

The histologic correlate of chronic bronchitis is the presence of mucous gland hyperplasia. This manifests as an increased percentage of the bronchial wall occupied by
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FIGURE 24.1. Regression of correlation between age at death and percentage of lung involvement by centrilobular emphysema, determined by point counting, in 173 autopsies of smokers who had the disease. Slope represents disease progression at rate of 7% per 10 years. Correlation coefficient is 0.27, and probability (p) that this trend might have occurred by chance is <.001. The equation for the regression line is as follows: Emphysema extent = 0.69 x Age – 13.2. Values in parentheses represent number of cases seen in each 10-year age group. Horizontal bars represent average extent of emphysema per group.

submucosal mucous glands (Fig. 24.3). Classically, this increase has been defined by the Reid index, which is the ratio of the width of the mucous glands to the distance between the basal lamina of the mucosa and the inner


perichondrium (see Fig. 2.7B in Chapter 2). Since this width may vary from site to site in the bronchial wall, an average value is obtained from the measurement at several locations. The index is normally less than 0.4. Values of 0.5 or greater are indicative of mucous gland hyperplasia. There is a direct correlation between the value of the Reid index and the volume of daily sputum production by the patient. In some cases, oxyphil metaplasia of the mucous glands may be observed. In the authors’ experience, this is usually seen in the bronchial resection margin of cigarette smokers who have undergone resection for lung cancer (see Fig. 2.8B in Chapter 2).

Other findings in chronic bronchitis include an increase in proportion of goblet cells in the surface mucosa (Fig. 24.4) and distention of mucous gland ducts (Figs. 24.5 and 24.6). Surface mucosal goblet cells normally average about one per 20 ciliated cells in the trachea and are progressively fewer in lobar and segmental bronchi. In chronic bronchitis, goblet cells may actually outnumber the ciliated cells. In some cases, squamous metaplasia or diffuse reserve cell hyperplasia may also be observed. A chronic inflammatory infiltrate composed of lymphocytes and plasma cells is a variable feature and may be entirely absent. For this reason, it has been suggested that the term chronic large airways disease is preferable to chronic bronchitis, which implies an inflammatory component.

The morphology of chronic bronchitis is similar whether it is due to cigarette smoking, air pollution, or occupational exposures. In patients with chronic bronchitis secondary to silica exposure, fibrosis may be observed in the wall of the large airways (Fig. 24.7). In some cases, this may result in obstructive abnormalities.