

12

AIR SPACE DISEASE

Objectives:

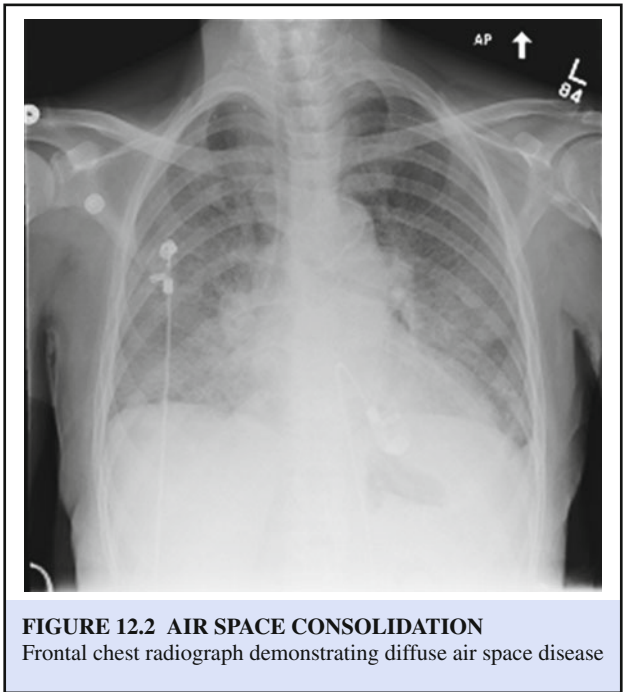
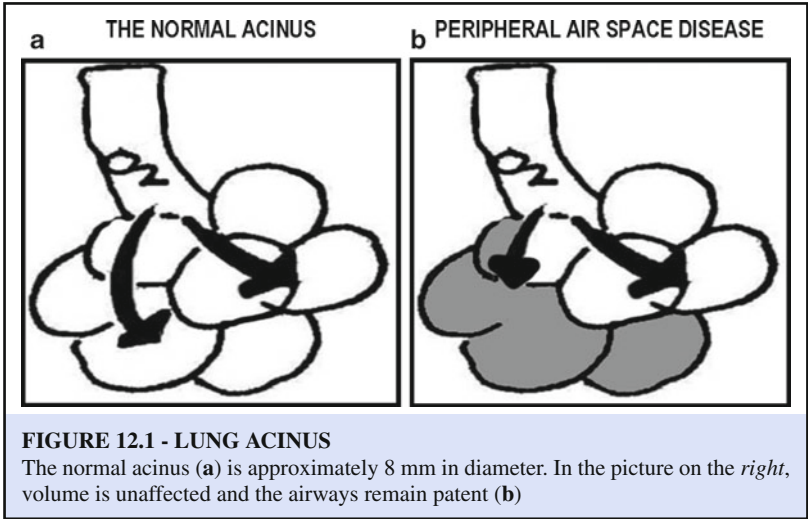
1. Define “pulmonary acinus.”
2. List the radiographic findings characteristic of air space disease.
3. List the major differential diagnostic categories for acute air space disease.
4. List two causes of chronic air space disease.

The purpose of this unit is to demonstrate the appearance of air space disease in the lungs.

The pulmonary acinus is the basic structural unit of the lung involved in gas exchange. It consists of a terminal bronchiole and the alveolar ducts, sacs, and alveoli distal to it. Recall that a terminal bronchiole is the most peripheral airway that is purely conductive in function with no gas exchange capability.

The acinus is visible when opacified on the plain chest radiograph as a slightly irregular nodular shadow approximately 8 mm in size (Fig. 12.1a). Disease within the air space manifest on the radiograph as soft tissue density. Disease may involve numerous acini or spread from one acinar unit to another via the pores of Kohn and canals of Lambert (Fig. 12.1b). The opacified acini become confluent, producing a fluffy, homogeneous radiographic pattern characteristic of air space disease as noted on Fig. 12.2.

Since disease which primarily affects the air space tends to spare the larger conductive airways, these airways become visible as tubular, branching, air-filled structures surrounded by the fluid-filled acini. These air-filled structures are normally surrounded by air-filled lung and are hence not distinguished normally on the chest radiograph. These air-filled bronchi surrounded by opacified air space are called air bronchograms. Air bronchograms are the radiographic hallmark of air space disease. Figure 12.3 is an excellent example of air bronchograms in a patient with pneumonia. The distribution of air space disease may be useful in determining its etiology.



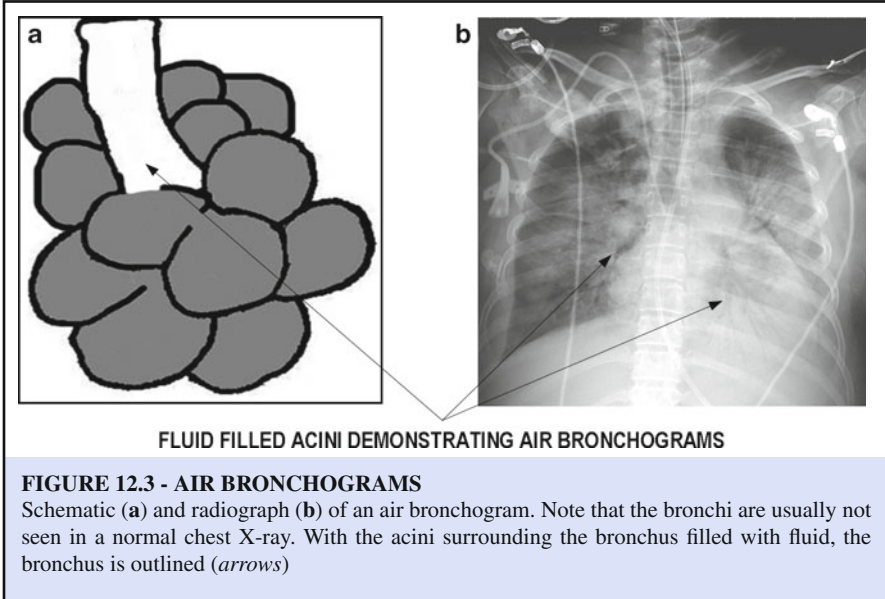


Figure 12.4 demonstrates a right lower lung pneumonia on the frontal radiograph. The right dome of diaphragm and right heart border are preserved. A lateral view is necessary to localize this to the right middle or lower lobes.

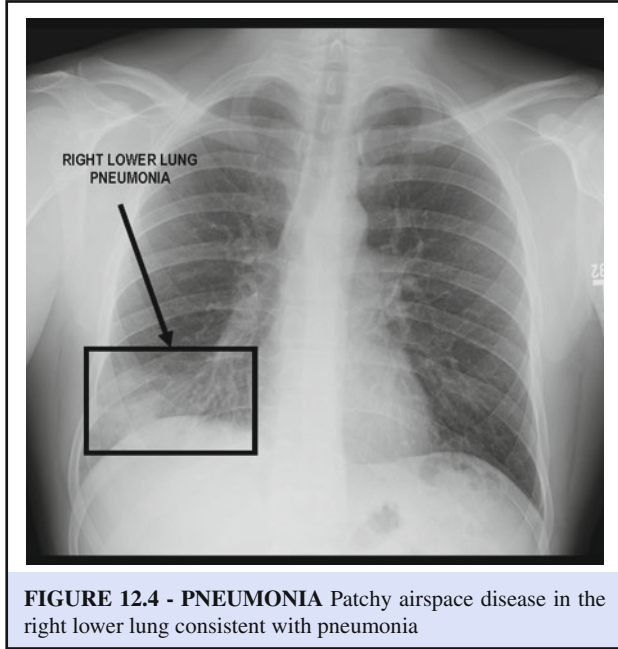
Consider the differential for acute airspace disease:

1. Pulmonary Edema: transudate fills the alveoli.
2. Infection: pneumonic exudate fills the alveoli.
3. Hemorrhage: blood fills the alveoli.

Within each of these major categories, however, multiple pathologies may be included.

1. Pulmonary edema – cardiogenic (CHF) or noncardiogenic (ARDS).
2. Infection – numerous organisms may cause pneumonia.
3. Hemorrhage – may be produced by many causes including pulmonary contusion, pulmonary infarcts, Goodpasture’s syndrome and diseases which produce vasculitis such as collagen vascular diseases.

This list is certainly not inclusive of all possibilities. Clinical information must be coordinated with old studies to narrow the diagnostic possibilities. In other words, fever would suggest pneumonia, while hemoptysis would suggest pulmonary hemorrhage. The distribution of the abnormality may also help. Cardiogenic edema tends to be diffuse, predominantly perihilar and bilateral and also associated with other findings (enlarged heart, pleural effusions). Pneumonia is classically more focal.



Pathologic processes involving the air space (alveoli) can be further subdivided into acute and chronic in nature. The time course of appearance and regression of airspace disease is useful. Edema can come and go quickly (onset may be within minutes, regression can occur within hours). Pneumonia and hemorrhage move more slowly, especially in regression. The slowest moving processes which present with airspace patterns are neoplasms such as bronchoalveolar carcinoma or pulmonary lymphoma which may present as chronic airspace disease.