Imaging of Small Airway Diseases

Abigail V. Berniker, MD, Travis S. Henry, MD*

KEYWORDS

• Small airways disease • Bronchiolitis • Centrilobular nodules • Tree-in-bud • Air trapping
• High-resolution computed tomography • Constrictive bronchiolitis

KEY POINTS

- Small airways disease, or bronchiolitis, refers to bronchiolar inflammation and fibrosis that is caused by numerous entities. Small airways disease is broadly classified into either cellular or constrictive subtypes.
- High-resolution computed tomography (HRCT) plays an important role in diagnosing small airways disease and is used in conjunction with clinical data and pathologic findings to solidify a diagnosis. Normal small airways are too small to be visible on HRCT.
- Cellular bronchiolitis is an inflammatory process that usually presents as centrilobular nodules, which is a direct finding of the disease. Centrilobular nodules may vary in size and attenuation.
- Constrictive bronchiolitis is a fibrotic process that usually manifests as mosaic attenuation from air trapping, an indirect finding of airway obstruction. Constrictive bronchiolitis often appears similar, despite etiology, so clinical history is crucial.
- Many mimics of small airways disease exist. Centrilobular nodules may be a result of arteriolar disease or aerogenous spread of adenocarcinoma. Mosaic attenuation may be secondary to small vessels disease or ground-glass opacity.

INTRODUCTION

Small airways disease, or bronchiolitis, is a broad term encompassing numerous diseases that cause bronchiolar inflammation or fibrosis. Bronchioles are small airways located at the center of the secondary pulmonary lobule (SPL), which are too small to see on imaging when they are normal, and are therefore usually visible only when abnormal.[1] High-resolution computed tomography (HRCT) plays an important role in detecting small airways disease and is used in conjunction with clinical data and pathologic findings to solidify a diagnosis. Direct signs of bronchiolitis include centrilobular nodules or opacities, typically reflecting a cellular or inflammatory form of bronchiolitis. Air trapping is an indirect sign and usually represents a constrictive, or obliterative bronchiolitis where the small airways are obstructed. This article reviews the following topics: normal bronchiolar anatomy, HRCT protocols for evaluating small airways disease, a histologic classification of bronchiolitis, the imaging features of small airways disease (divided into cellular and constrictive subcategories), and important radiologic mimics.

NORMAL ANATOMY

The SPL refers to the smallest functional anatomic unit of the lungs that is recognizable on imaging. A basic knowledge of the SPL anatomy is critical to recognizing and understanding small airways disease. SPLs measure 10 mm to 25 mm in size and are bounded by the interlobular septa, which

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Department of Radiology and Biomedical Imaging, University of California, San Francisco, 505 Parnassus Avenue, San Francisco, CA 94143, USA
* Corresponding author.
E-mail address: Travis.Henry@ucsf.edu

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contain peripheral connective tissue, veins, and lymphatics. At the heart of the SPL are the small airways, arterioles, and additional lymphatics.[2]

The small airways, or bronchioles, are less than 2 mm internal diameter and lack cartilage. Membranous bronchioles are purely air conducting, whereas respiratory bronchioles supply alveoli and provide gas exchange. These airways provide little resistance in the normal lung, but their total cross-sectional area is so large that even mild abnormalities of the small airways can have a profound effect on lung function.[2,3]

Normal bronchioles are below the size threshold for HRCT. As such, visible bronchiolar abnormalities are a clue to underlying small airways inflammation and/or fibrosis. Bronchiolar inflammation typically manifests as centrilobular nodules on HRCT. These nodules may vary in size (as small as 1–2 mm, or large enough to occupy nearly an entire SPL) and attenuation (ranging from faint ground-glass to soft tissue attenuation) but the most important characteristic is that centrilobular nodules should spare the pleural and fissural surfaces, thus distinguishing them from random or perilymphatic nodules (**Fig. 1**).[2]

Similarly, bronchiolar fibrosis often manifests as mosaic attenuation because of air trapping. This air trapping often has sharply delineated borders where abnormal SPLs interface with normal SPLs. Bronchial wall thickening and bronchiectasis may be present.

Both imaging patterns seen in small airways disease (nodules and mosaic attenuation) are commonly encountered on CT. However, there are many mimics of small airways disease that are also frequently encountered, which are also discussed in this article.

IMAGING TECHNIQUE

HRCT is the primary imaging tool for evaluation of small airways disease. HRCT uses thin sections

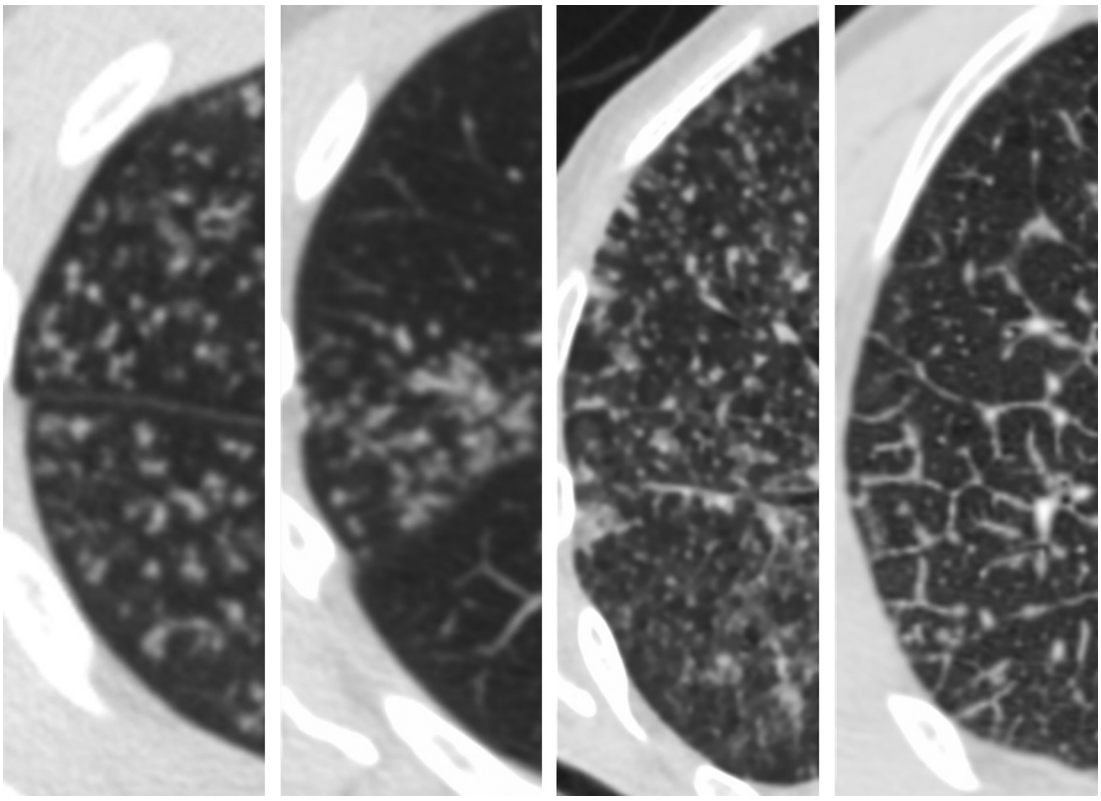

Fig. 1. CT images from four different patients illustrating various distributions of pulmonary nodules. Centrilobular nodules (*far left image*) are distinguishable because they spare the pleural and fissural surfaces (note the 1- to 2-mm gap between the nodules and these surfaces). Many of these nodules have a tree-in-bud pattern, a subset of centrilobular nodules, in this case representing tuberculosis. Perilymphatic (*second from left*, sarcoidosis) and random (*second from right*, miliary tuberculosis) nodules both touch pleural surfaces, thus distinguishing them from centrilobular nodules. *Far right* image shows nodular beading of the interlobular septae from esophageal cancer, nicely outlining the secondary pulmonary lobules.