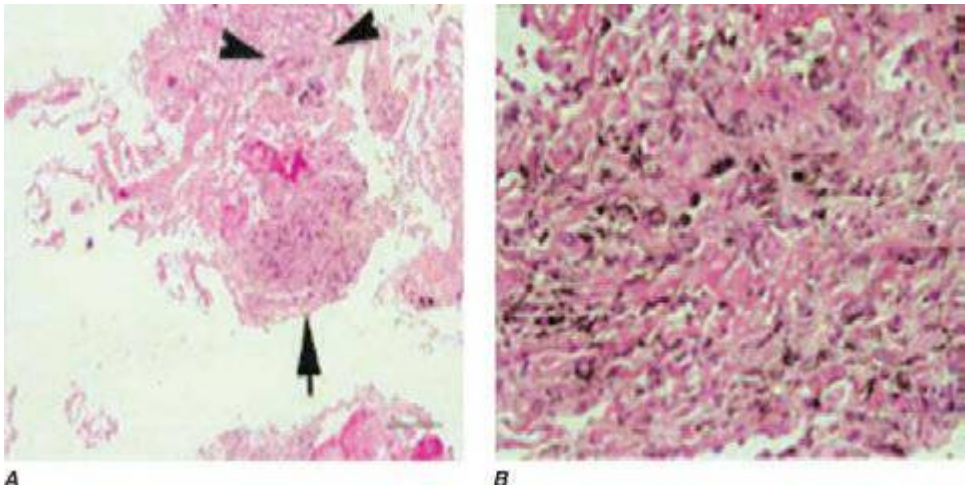

FIGURE 311-4 Histopathologic features of biomass smoke–induced interstitial lung disease. *A.* Anthracitic pigment is seen accumulating along alveolar septae (*arrowheads*) and within a pigmented dust macule (*single arrow*). ***B.*** A high-power photomicrograph contains a mixture of fibroblasts and carbon-laden macrophages.

of fine particulate matter, a component of biomass smoke, have been reported to be 2–30 times higher than the National Ambient Air Quality Standards set by the U.S. EPA.

Epidemiologic studies have consistently shown associations between exposure to biomass smoke and both chronic bronchitis and COPD, with odds ratios ranging between 3 and 10 and increasing with longer exposures. In addition to the common occupational exposure to biomass smoke of women in developing countries, men from such countries may be occupationally exposed. Because of increased migration to the United States from developing countries, clinicians need to be aware of the chronic respiratory effects of exposure to biomass smoke, which can include interstitial lung disease (Fig. 311-4). Evidence is beginning to emerge that improved stoves with chimneys can reduce biomass smoke–induced respiratory illness in both children and women.

312 Bronchiectasis

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Bronchiectasis refers to an irreversible airway dilation that involves the lung in either a focal or a diffuse manner and that classically has been categorized as cylindrical or tubular (the most common form), varicose, or cystic.

ETIOLOGY

Bronchiectasis can arise from infectious or noninfectious causes (Table 312-1). Clues to the underlying etiology are often provided by the pattern of lung involvement. *Focal bronchiectasis* refers to bronchiectatic changes in a localized area of the lung and can be a consequence of obstruction of the airway—either extrinsic (e.g., due to compression by adjacent lymphadenopathy or parenchymal tumor mass) or intrinsic (e.g., due to an airway tumor or aspirated foreign body, a scarred/stenotic airway, or bronchial atresia from congenital underdevelopment of the airway). *Diffuse bronchiectasis* is characterized by widespread bronchiectatic changes throughout the lung and often arises from an underlying systemic or infectious disease process.

More pronounced involvement of the upper lung fields is most common in cystic fibrosis (CF) and is also observed in postradiation fibrosis, corresponding to the lung region encompassed by the radiation port. Bronchiectasis with predominant involvement of the lower lung fields usually has its source in chronic recurrent aspiration (e.g., due to esophageal motility disorders like those in scleroderma), end-stage fibrotic lung disease (e.g., traction bronchiectasis from idiopathic pulmonary fibrosis), or recurrent immunodeficiency-associated infections (e.g., hypogammaglobulinemia). Bronchiectasis resulting from infection by nontuberculous mycobacteria (NTM), most commonly the *Mycobacterium avium-intracellulare* complex (MAC), often preferentially affects the midlung fields. Congenital causes of bronchiectasis

TABLE 312-1 MAJOR ETIOLOGIES OF BRONCHIECTASIS AND PROPOSED WORKUP

Pattern of Lung Involvement	Etiology by Category (Examples)	Workup
Focal	Obstruction (aspirated foreign body, tumor mass)	Chest imaging (chest x-ray and/or chest computed tomography); bronchoscopy
Diffuse	Infection (bacterial, nontuberculous mycobacterial)	Sputum Gram's stain/culture; stains/cultures for acid-fast bacilli and fungi. If no pathogen is identified, consider bronchoscopy with bronchoalveolar lavage.
	Immunodeficiency (hypogammaglobulinemia, HIV infection, bronchiolitis obliterans after lung transplantation)	Complete blood count with differential; immunoglobulin measurement; HIV testing
	Genetic causes (cystic fibrosis, Kartagener's syndrome, α_1 antitrypsin deficiency)	Measurement of chloride levels in sweat (for cystic fibrosis), α_1 antitrypsin levels; nasal or respiratory tract brush/biopsy (for dyskinetic/immotile cilia syndrome); genetic testing
	Autoimmune or rheumatologic causes (rheumatoid arthritis, Sjögren's syndrome, inflammatory bowel disease); immune-mediated disease (allergic bronchopulmonary aspergillosis)	Clinical examination with careful joint exam, serologic testing (e.g., for rheumatoid factor). Consider workup for allergic bronchopulmonary aspergillosis, especially in patients with refractory asthma.[a]
	Recurrent aspiration	Test of swallowing function and general neuromuscular strength
	Miscellaneous (yellow nail syndrome, traction bronchiectasis from postradiation fibrosis or idiopathic pulmonary fibrosis)	Guided by clinical condition
	Idiopathic	Exclusion of other causes

[a]Skin testing for *Aspergillus* reactivity; measurement of serum precipitins for *Aspergillus*, serum IgE levels, serum eosinophils, etc.