Recent changes in the demographics of HIV infection, along with advances in therapy and prophylaxis, have influenced the relative frequency and patterns of pulmonary infections associated with AIDS [1–3]. Bacterial respiratory infections, including infectious airways disease and pneumonia, currently account for most pulmonary infections diagnosed in HIV-infected individuals in the United States [4, 5].

The significance of bacterial pneumonia in HIV infection is underscored by the inclusion of two or more episodes of bacterial pneumonia within a 1-year period as an AIDS-defining illness for an HIV-infected patient, regardless of the CD4 cell count [6]. Considering the frequency and significance of bacterial respiratory infections in HIV-infected patients, radiologists should be familiar with radiologic manifestations of these infections. For example, HIV-infected patients with bacterial pneumonia most commonly present with chest radiographic findings of focal consolidation and clinical symptoms of productive cough and fever of less than 1 week’s duration [3]. We review the spectrum of nontuberculous bacterial respiratory infections that occur in HIV-infected individuals, with a special emphasis on imaging features.

**Bacterial Pneumonia**

**HIV Infection and Risk of Bacterial Pneumonia**

Although HIV infection is most closely associated with altered cell-mediated immunity (which is manifested by a decrease in CD4 count), a number of additional immune deficits may occur in association with HIV infection [7–9], including a poor antibody response due to B cell dysfunction and defects in chemotaxis, phagocytosis, and intracellular killing by monocytes, macrophages, and neutrophils [8, 9]. In addition, HIV-infected individuals may experience impairment of local defenses, manifested by a depression of specific IgA at the mucosal surfaces [7, 9]. These immune abnormalities all contribute to an increased risk of bacterial infection among HIV-infected persons, particularly by encapsulated bacteria, such as *Streptococcus pneumoniae* and *Haemophilus influenzae*.

The overall rate of bacterial pneumonia in HIV-infected persons is approximately six times greater than that in the general population [4]. The incidence of pneumococcal pneumonia is five to 18 times greater than that in the general population, and the development of pneumococcal septicemia is 100 times greater [10]. It has been estimated that greater than one third of all persons with AIDS will develop at least one episode of severe bacterial pneumonia over the course of their HIV infection [8]. Considering these data, Afessa et al. [11] found bacterial pneumonia to be the most frequent pulmonary complication (42%) in a recent autopsy series of 233 HIV-infected individuals.

IV drug abusers constitute the HIV risk factor group with the highest prevalence of bacterial infection. In this population, bacterial pneumonia rates are more than double those of other HIV risk factor groups, regardless of the CD4 lymphocyte count [4]. A notable demographic trend of HIV infection in the United States in the past decade has been an increased frequency of HIV infection among IV drug abusers and a decreased prevalence of infection among male homosexuals [1]. This demographic trend has contributed to the increased prevalence of bacterial respiratory infections in AIDS patients.

Although bacterial pneumonia often occurs in the early stages of HIV infection, the risk of bacterial infection increases steadily with declining CD4 lymphocyte counts [4]. For example, HIV-infected individuals with CD4 counts of less than 200 cells/mm³ have a fivefold increased prevalence of bacterial pneumonia.
compared with infected persons with CD4 counts greater than 500 cells/mm$^3$. Cigarette smoking has also been associated with an increased risk of bacterial pneumonia among HIV-infected patients with CD4 lymphocyte counts of less than 200 cells/mm$^3$ [4]. Smoking cessation has thus been recommended as a means of decreasing the rate of bacterial respiratory infections in this subgroup [4].

Recent trends in the prophylaxis and treatment of HIV-infected individuals have influenced the relative frequency of various pulmonary infections. For example, the widespread use of prophylaxis for Pneumocystis carinii pneumonia has dramatically decreased the incidence of this infection [3]. The use of trimethoprim-sulfamethoxazole as a P. carinii pneumonia prophylactic agent also provides a lesser degree of protection from bacterial infection [4].

The introduction of highly active antiretroviral therapy, a combination therapy comprising a three-drug regimen of HIV protease inhibitors and nucleoside analog reverse transcriptase inhibitors, has also resulted in a profound decrease in the rate of opportunistic infections in HIV-infected individuals [12]. Because the rate of bacterial pneumonia increases with declining immune status in HIV infection, one would expect that the overall rate of bacterial pneumonia would decline with enhanced immune function associated with highly active antiretroviral therapy [12]. Sullivan et al. [12] studied the rate of pulmonary infections in 1898 HIV-infected patients and found a significant overall decline in bacterial pneumonia associated with the use of highly active antiretroviral therapy. Another study by Wolff and O’Donnell [13] found that bacterial pneumonia was relatively more common in the era after the introduction of highly active antiretroviral therapy compared with the era before this introduction. However, Wolff and O’Donnell emphasized that their findings reflected a relative increase in bacterial pneumonia compared with opportunistic infections rather than a true increased risk for bacterial pneumonia. In other words, highly active antiretroviral therapy is associated with a greater decreased risk for opportunistic infections rather than for bacterial pneumonia, thus resulting in an increase in the relative proportion of pulmonary infections that are bacterial in origin.

**Spectrum of Organisms**

Similar to that in the general population, bacterial pneumonia in HIV-infected individuals is usually community-acquired. *S. pneumoniae* is the most common infecting organism [4, 14]. *H. influenzae*, *Staphylococcus aureus*, *Escherichia coli*, and *Pseudomonas aeruginosa* account for most of the remainder of cases [14–16]. *P. aeruginosa* has been increasingly recognized as an important source of bacterial pneumonia in HIV-infected individuals, particularly in those with neutropenia, steroid use, multiple antibiotic therapy, myelosuppressive therapy, and indwelling catheters [16–18]. Atypical agents such as *Legionella pneumophila* and *Mycoplasma pneumoniae* are rarely diagnosed in HIV-infected patients with community-acquired pneumonia [19].

AIDS patients with advanced immune suppression are also vulnerable to a host of unusual infectious agents, including *Nocardia asteroides*, *Rhodococcus equi*, and *Bartonella henselae* and *qintana*. *N. asteroides* is a soil-borne aerobic actinomycete, acquired by inhalation. It usually occurs in southern and rural regions of the United States and, compared with urban areas, possibly reflects differential exposure of the populations in these areas to soil-borne pathogens [15]. The zoonosis *R. equi* causes pneumonia in horses and other farm animals. Human infection occurs exclusively in immunocompromised hosts, including AIDS patients with CD4 cell counts of less than 200 cells/mm$^3$ [20]. *B. henselae* and *B. qintana* are additional unusual bacteria that are responsible for the clinical syndromes of bacillary angiomatosis and peliosis in HIV-infected patients. Exposure to cats and cat fleas is the main risk for infection with *B. henselae*, which causes cat-scratch fever. Exposure to lice is the main risk for infection with *B. qintana*, which causes trench fever [15, 21].

**Clinical and Radiographic Features of Common Bacterial Pathogens**

HIV-infected patients with bacterial pneumonia usually have the same signs and symptoms as those in the general population. Typically, they present with a relatively rapid onset of clinical symptoms such as productive cough, fever, shaking chills, pleuritic chest pain, and dyspnea. Symptoms are usually present for less than 1 week before the patient seeks medical attention [14, 22–26].

Recently, Gold et al. [27] studied HIV patients with abnormal chest radiographic findings and a lack of respiratory symptoms. None of the patients in this series had bacterial pneumonia as a cause of the radiographic findings; in contrast, mycobacterial infection was a common cause of infection. Thus, bacterial pneumonia is rarely the cause of radiographic findings in the absence of symptoms.

Despite atypical manifestations and overlapping features among many pulmonary complications of HIV infection, the chest radiograph is reasonably accurate in depicting common complications such as bacterial pneumonia, particularly when radiographic findings are correlated with clinical and laboratory data [3]. The most common radiographic pattern in bacterial pneumonia is focal consolidation (Fig. 1), which typically presents in either a segmental or lobar distribution [22–24, 27–31]. In two studies of HIV-infected individuals with bacterial pneumonia by Boiselle et al. [29] and Magenet et al. [22], focal consolidation was observed in approximately 45–60% of patients. Similarly, a CT study by Sider et al. [30] found that focal segmental alveolar consolidation was usually associated with bacterial pneumonia.

The combination of focal consolidation seen on chest radiography and clinical symptoms of less than 7 days’ duration has a high specificity for the diagnosis of bacterial pneumonia [24]. In a study that assessed the clinical, laboratory, and radiographic predictors of various respiratory infections in HIV-infected hospitalized patients, Selwyn et al. [24] found that the combination of focal consolidation on chest radiography and a history of fever for fewer than 7 days was associated with a sensitivity of 48% and a specificity of 94% for the diagnosis of bacterial pneumonia.

In almost half of the cases of bacterial pneumonia, a radiographic pattern other than focal consolidation is observed [22, 29]. Thus, bacterial pneumonia has been found to be more difficult to diagnose radiographically than either *P. carinii* or pulmonary tuberculosis [29]. Similarly, bacterial pneumonia has been shown to frequently mimic other infections radiographically [29]. For example, a bilateral pattern of alveolar or interstitial opacities may be observed in bacterial pneumonia, which can mimic *P. carinii* [22, 29].

Bacterial infections may also present as solitary or multiple lung nodules [29, 32–35]. For example, in a recent study regarding the cause of pulmonary nodules in HIV-infected patients, bacterial pneumonia was the most common cause, followed by tuberculosis [35]. Cavitary pulmonary lesions (Fig. 2) are another recognized radiologic finding often associated with bacterial pneumonia in HIV-infected patients. In a study that investigated the cause of cavitary lung disease on chest CT scans of HIV-infected patients, Aviram et al. [36] found a bacterial
cause in 85% of cases. More than one pathogen was identified in most patients in this series. The most frequently identified pathogens were *P. aeruginosa* (Fig. 2) and *S. aureus* (Fig. 3). *S. aureus* is frequently associated with septic emboli among IV drug abusers and typically presents radiographically as multiple cavitary nodules (Fig. 3). This subset of patients is also prone to develop empyemas [37]. Less common bacterial causes of cavitary nodules or cavitary consolidation include *N. asteriodes* and *R. equi* infections. Mycobacterial and fungal infections are important differential diagnostic considerations for these findings.

Although conventional radiographs are the mainstay of imaging of bacterial respiratory infections in HIV-infected persons, CT may be useful in selected cases. For example, nodules, cavities, and pleural fluid collections (Fig. 4) are often better delineated on CT than on conventional radiographs. CT may thus be useful for further characterizing atypical radiographic findings, for diagnosing complications of infection such as abscess or empyema, and for guiding biopsy or drainage procedures in selected cases.

Parapneumonic pleural effusions are seen in a significant minority of cases of bacterial pneumonia and are usually small [29]. Thoracic empyema is more common in IV drug

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**Fig. 1.**—Round pneumonia in 33-year-old HIV-positive woman. (Reprinted with permission from [29])

A, Frontal chest radiograph reveals focal round consolidation overlying right hilum. B, Lateral chest radiograph localizes consolidation to right middle lobe. Bacterial pneumonia may occasionally display round appearance. In such cases, follow-up radiographs are important to document resolution and to exclude neoplastic mass.

**Fig. 2.**—Cavitary *Pseudomonas aeruginosa* in 47-year-old HIV-positive man with advanced immune suppression. CT scan (lung window setting) at level of right upper lobe bronchus shows cavitary pneumonia in right upper lobe. Note diffuse bilateral ground-glass opacities and loculated pleural fluid collection lateral to area of cavitation.

**Fig. 3.**—Cavitary nodules due to septic emboli in 32-year-old woman with risk factor of IV drug abuse. CT scan (lung window setting) at level of superior segment bronchi shows multiple peripheral lung nodules, some of which show cavitation. Note bilateral pleural effusions.
abusers but has an overall low prevalence [38]. Borge et al. [38] postulated that the impact of HIV on the cellular immune system may impair the development of empyema.

Although intrathoracic lymph node enlargement is not usually evident on chest radiographs, mildly enlarged nodes are frequently seen on CT scans of patients with bacterial pneumonia [29, 39]. However, mediastinal and hilar lymphadenopathy are more frequently associated with mycobacterial infection [30, 36, 39].

HIV-infected individuals with uncomplicated bacterial pneumonia due to typical pathogens usually show a clinical and radiographic response to antibiotic therapy, with a time course similar to that of normal hosts undergoing treatment for community-acquired pneumonia [14]. In contrast to that of normal hosts, however, bacterial pneumonia in AIDS patients tends to progress more rapidly (Fig. 5) and is more often complicated by cavitation and abscess formation [37]. In general, resolution of radiographic abnormalities from treated bacterial pneumonia tends to be more rapid when compared with that in other lung diseases associated with HIV infection [14].

Bacteremia is more common in patients with HIV infection than in the general population [10, 14]. Shah et al. [40] reported that the presence of bacteremia does not influence the radiographic pattern of pneumococcal pneumonia in HIV-infected patients. HIV-infected patients with bacteremia have a mortality rate similar to patients with seronegative bacteremia [41].

Clinical and Radiographic Features Associated with Unusual Bacterial Pathogens

HIV-infected individuals with advanced immune suppression are also at risk for a variety of unusual organisms, including R. equi, N. asteroides, and B. henselae and B. quintana. Patients with R. equi infection usually present with an indolent course of cough, fever, and dyspnea. Radiographically, R. equi pneumonia usually presents with one or more focal areas of consolidation, predominantly in the upper lobes, with frequent cavitation [42] (Fig. 6). Additional features may include empyema and lymphadenopathy [31]. The clinical presentation of N. asteroides infection is similar to that of R. equi [7]. The most common radiographic presentation is a lobar or multilobar distribution of alveolar consolidation, with an upper lobe predominance and frequent cavitation [7].

Bacillary angiomatosis, an infection caused by B. henselae and B. quintana, is characterized by a neovascular proliferation involving multiple sites in the body including the skin, liver, spleen, lymph nodes, and lungs [43, 44]. Exposure to cats, cat fleas, and lice are the main risk factors for this infection. Affected patients typically present with angiomatous skin lesions that mimic Kaposi’s sarcoma. Clinical symptoms include fever, night sweats, cough, and occasional hemoptysis. Bacillary angiomatosis has several radiographic manifestations in the chest, including endobronchial lesions, parenchymal nodules (Fig. 7), pleural effusions, and chest wall masses [43, 44]. Mediastinal lymphadenopathy is common and is characterized by intense enhancement after administration of IV contrast material. One should strongly consider this treatable infection in patients with suspected Kaposi’s sarcoma who lack the typical risk factor (homosexual contact) for this neoplasm [15, 43, 44].

Pyogenic Airways Disease

HIV-infected patients are also at increased risk for developing infectious airways disease such as bacterial tracheobronchitis and bronchiolitis [45–48]. Acute bacterial bronchitis was the predominant lower respiratory infection in cohort members of the Pulmonary Complications of HIV Infection Study Group [45] who entered the study with CD4 levels above 200 cells/mm$^3$. The most common bacterial organisms responsible for infectious airways disease include H. influenzae, P. aeruginosa, Streptococcus viridans, and S. pneumoniae [5, 46, 47].

Pyogenic airways infections lead to inflammatory changes to the walls of the bronchi and bronchioles, resulting in airway wall thickening and dilation [46] (Fig. 8). These changes may be irreversible if not treated early with antimicrobial agents [46]. Bronchiectasis has
CT and Radiography of AIDS Patients

Fig. 5.—Rapidly progressive pneumococcal pneumonia in 50-year-old HIV-positive man. (Reprinted with permission from [3])
A, Portable frontal chest radiograph reveals focal areas of consolidation in lingula and left lower lobe.
B, CT scan (lung window settings) of lower chest obtained as part of abdominal CT scan 1 day after A shows progressive consolidation in lingula and left lower lobe.
C, Portable frontal chest radiograph obtained 1 day after B shows rapid progression of pneumonia, which now involves left lung diffusely. Patient was treated with intubation for respiratory failure but responded to appropriate antibiotic therapy and fully recovered.

Fig. 6.—Cavitary pneumonia due to Rhodococcus equi infection in 34-year-old HIV-positive man. CT scan (lung window setting) obtained at level of carina shows peripheral focus of cavitary consolidation in left upper lobe. Note subtle foci of ground-glass attenuation in adjacent lung parenchyma. (Courtesy of Costello P, Boston, MA)

Fig. 7.—Lung nodules due to bacillary angiomatosis in 38-year-old HIV-positive man who presented with skin lesions and had HIV risk factor of IV drug abuse. CT scan (lung window setting) obtained at level of mainstem bronchi shows numerous scattered lung nodules (arrows), which are randomly distributed. Several nodules abut pleural surfaces (contrast appearance with centrilobular nodules in Figure 8 that spare pleural surfaces). Random distribution of nodules can be seen in hematogenous spread of infection or neoplasm. Note bilateral pleural effusions. (Courtesy of White C, Baltimore, MD)
been noted to develop in a relatively short time after an episode of pulmonary pyogenic infection in HIV-infected patients [48]. This shortened time frame suggests that AIDS patients may experience an accelerated form of bronchiectasis. In a few HIV-infected individuals, bronchiectasis may occur in the absence of a history of prior infections and may subsequently predispose affected patients to future recurrent infections [49]. HIV-infected patients with pyogenc bronchitis or bronchiolitis typically present with dyspnea, fever, and productive cough [26]. Early in the course of bacterial infectious airways disease, conventional chest radiographs may fail to show any obvious abnormality [46]. In some cases of infectious bronchitis, bronchial wall thickening (tram tracks) may be observed radiographically [46]. Extensive bronchiolitis may create an apparent interstitial pattern of reticulonodular opacities that represents impacted bronchioles [2]. This pattern is typically symmetrically distributed, with lower lobe predominance. Such findings may mimic P. carinii [2].

Chest CT, particularly high-resolution CT, is more sensitive and specific than radiography for detecting inflammatory changes of the bronchi and bronchioles [46]. Thus, in a symptomatic patient with dyspnea, fever, and a productive cough, a high-resolution CT scan may show findings of small airways disease despite the absence of radiographic findings [2].

The characteristic findings of infectious bronchiolitis on high-resolution CT consist of centrilobular opacities arranged in a tree-in-bud pattern, manifested by small, Y- and V-shaped opacities in the lung periphery [2, 3] (Fig. 8), which represent bronchioles that are impacted with inflammatory secretions. Focal regions of air trapping may also be evident on expiratory CT scans [50]. Although bacteria are the most common cause of small airways disease in AIDS patients, the differential diagnosis for this pattern includes mycobacterial, viral, and fungal infections.

In summary, bacterial respiratory infections, including bacterial airways disease and pneumonia, account for most pulmonary infections in HIV-infected patients. The most common radiographic pattern of bacterial pneumonia is the presence of one or more areas of focal consolidation in a segmental or lobar distribution. Bacterial pneumonia is also a common cause of lung nodules and cavities. Pyogenic infectious airways diseases is challenging to diagnose using conventional radiographs. In contrast, CT features are characteristic and include bronchial dilation, wall thickening, and nodular and branching centrilobular opacities.

Fig. 8.—Pyogenic airways disease in 39-year-old HIV-positive man with recurrent respiratory infections. High-resolution CT scan of lung bases shows multilobar bronchial dilation, bronchial wall thickening, and bronchiolitis (arrow). Note clustering of small nodular and branching opacities that spare pleural surfaces. Such centrilobular distribution is highly suggestive of infectious cause. Note minimal foci of peripheral consolidation in right middle lobe and lingula. (Reprinted with permission from [5])

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CT and Radiography of AIDS Patients


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