# Lung Abscess in Patients with AIDS

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**We identified 31 patients with human immunodeficiency virus (HIV) infection and lung abscess. All patients had advanced HIV disease, and the mean CD4 cell count was 17/mm$^3$ (range, 2–50/mm$^3$). Twenty-two patients (71%) had previous opportunistic infections, and 24 (77%) had previous pulmonary infections. Symptoms at the time of presentation included fever (90% of patients), cough (87%), dyspnea (35%), pleuritic chest pain (26%), and hemoptysis (10%). The microbiological etiology was established for 28 patients, and the pathogens recovered were bacteria (65%), *Pneumocystis carinii* (6%), fungi (3%), and mixed microorganisms (16%). The pathogens included *Pseudomonas aeruginosa* (11), *Streptococcus pneumoniae* (6), *P. carinii* (5), *Klebsiella pneumoniae* (5), *Staphylococcus aureus* (4), *Aspergillus* species (3), viridans streptococcus (2), *Haemophilus influenzae* (1), *Streptococcus milleri* (1), *Proteus mirabilis* (1), and *Cryptococcus neoformans* (1). *Mycobacterium tuberculosis* was not isolated; two patients for whom a microbiological etiology was not established responded to antituberculous therapy. Patients were treated for 2–12 weeks; 25% of the patients received >4 weeks of therapy. The outcome was poor: 36% of the patients had recurrences, and 19% died. In patients with AIDS, lung abscess is associated with advanced HIV infection, is due to a broad spectrum of pathogens, responds poorly to antibiotics, and has a poor prognosis.**

The spectrum of opportunistic infections diagnosed for patients with HIV infection continues to change [1]. Patients are surviving longer and, therefore, remain at extended risk for various infections. Furthermore, broader use of prophylaxis has changed the presentation of several common opportunistic infections [2, 3]. Unusual diseases and uncommon presentations of common diseases are increasingly being reported.

At our institution, we have witnessed an increase in the number of cases of lung abscess in patients infected with HIV. The etiology, predisposition, and outcome of this disease in HIV-negative patients are well described. The disease occurs in middle-aged patients, who often have a history of alcohol abuse or gingivitis, and is usually secondary to aspiration of anaerobes [4, 5]. However, to our knowledge, no study has systematically examined the etiology, clinical characteristics, and outcome of lung abscess in HIV-positive patients. Isolated case reports have described lung abscess associated with *Pneumocystis carinii* [6–8], *Mycobacterium avium/Mycobacterium intracellulare* complex [9], *Rhodococcus equi* [10, 11], *Salmonella* species [12], *Staphylococcus aureus* [13], *Legionella* species [14], *Alcaligenes xylosoxidans* [15], *Aspergillus* species [16], and *Pseudomonas aeruginosa* [17]. Infection due to *Mycobacterium tuberculosis,* which often causes upper-lobe cavity disease in HIV-negative patients, seldom presents as cavitary lesions in HIV-infected individuals [18–21].

To determine the clinical characteristics of lung abscess in patients with AIDS, we reviewed our experience with 31 cases of lung abscess treated over a 5-year interval (1989–1994) at The New York Hospital/Cornell University Medical College.

## Methods

The New York Hospital/Cornell University Medical College is a 880-bed tertiary medical center in New York City. Patients with HIV infection and lung abscess who were treated from 1989 to 1994 were identified by a systematic search of physician outpatient records, radiology reports, International Classification of Diseases–9 discharge diagnoses, and daily admission censuses from the AIDS inpatient care unit. Medical records were reviewed, and data (including patient demographics, medical history, clinical characteristics, hospital course, and outcome) were collected onto a standardized data form.

A case of lung abscess was defined on the basis of both radiographic criteria (evidence of a lung cavity on a chest radiograph or CT) and clinical criteria (evidence of active infection requiring antibiotic treatment of a pulmonary infection). Patients with recently resolved pulmonary infection and radiographic evidence of a lung cavity were excluded from the study if they had completed a course of antibiotic therapy for the pulmonary infection within the past month. The radiographic abnormality in these patients was considered scarring.

Forty-three patients with radiographic evidence of a lung cavity were identified. Of these patients, eight were excluded from further analysis because they had recently completed a course of treatment for a pulmonary infection and the radiographic abnormalities were thought to represent scarring rather than active disease (six patients with *P. carinii* pneumonia and

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two patients with bacterial pneumonia). Four additional patients were excluded from the study because a lung cavity was noted on a chest radiograph or CT in the absence of symptoms. These patients did not undergo any evaluation or receive treatment of the lung process.

The microbiological cause of lung abscess was determined by several diagnostic tests, including culture of sputum, bronchoalveolar lavage (BAL) fluid, fine-needle aspirates, or transbronchial biopsy specimens. However, because the workup was not standardized for each patient, it was difficult to evaluate the sensitivity of the different diagnostic modalities. For those patients for whom diagnoses were made by more than one method, the least-invasive method was considered diagnostic for result purposes (i.e., if sputum and BAL fluid cultures yielded the same results, then sputum analysis was considered diagnostic). Patients were considered to have no known diagnosis when all diagnostic tests were unrevealing. All specimens were routinely sent for aerobic, bacterial, fungal, and mycobacterial cultures. Anaerobic cultures were done on all five fine-needle aspirates and on five of 21 BAL fluid specimens.

Four outcomes were defined: resolution, the disappearance of both symptoms and radiographic evidence of active infection; recurrence, relapse of clinical symptoms and worsening of radiographic abnormalities at least 1 month after completion of a course of antibiotic therapy for lung abscess; death due to lung abscess, death due to an uncontrolled pulmonary infection without other identifiable causes at the time of death; and death due to other causes, death within 3 months of the diagnosis of lung abscess.

Statistical analysis was done by $\chi^2$ analysis. $P$ values of $<.05$ were considered statistically significant.

## Results

Thirty-one patients with HIV infection and lung abscess were identified. The mean age of the patients was 38 years (range, 16–59 years); 22 (71%) were male, 13 (42%) were Caucasian, 9 (29%) were African American, and 9 (29%) were Hispanic (table 1). Risk factors for HIV infection included intravenous drug use (36% of patients), homosexual contact (32%), heterosexual contact (26%), and transfusion of infected blood products (6%). Twenty-nine percent of the patients had a history of tobacco use, and 10% had a history of alcohol use. Twenty-four patients (77%) were receiving prophylaxis for *P. carinii* pneumonia, including trimethoprim-sulfamethoxazole (29% of patients), aerosolized pentamidine (26%), dapsone (19%), and atovaquone (3%).

All patients had advanced HIV disease; the mean CD4 cell count was 17/mm$^3$ (range, 2–50/mm$^3$). Twenty-two patients (71%) had previous opportunistic infections, and for four patients (13%), HIV infection was first diagnosed as a result of lung abscess. Twenty-four patients (77%) had a history of pulmonary infections, including bacterial pneumonia (52% of patients), *P. carinii* pneumonia (49%), and *M. tuberculosis* infection (6%). Other prior opportunistic infections included *M. avium/M. intracellulare* complex bacteremia (5 patients), cytomegalovirus retinitis (3), esophagitis (3), toxoplasmosis (2), Kaposi's sarcoma (1), progressive multifocal leukodystrophy (1), and microsporidosis (1). Two patients (6%) had evidence of altered consciousness at the time of presentation. No patient had evidence of active periodontal disease.

**Table 1.** Characteristics of 31 patients with AIDS and lung abscess.

| Characteristic | Value* |
|---|---|
| Mean age (range) in y | 38 (16–59) |
| Mean CD4 cell count (range) in mm$^3$ | 17 (2–50) |
| Mean WBC count (range) in mm$^3$ | 5.4 (1.8–9.3) |
| Gender | |
| Male | 22 (71) |
| Female | 9 (29) |
| Race | |
| Caucasian | 13 (42) |
| African American | 9 (29) |
| Hispanic | 9 (29) |
| Risk factor for HIV infection | |
| Intravenous drug use | 11 (36) |
| Homosexual contact | 10 (32) |
| Heterosexual contact | 8 (26) |
| Transfusion of blood products | 2 (6) |
| History of alcohol use | |
| Yes | 3 (10) |
| No | 25 (80) |
| Unknown | 3 (10) |
| History of tobacco use | |
| Yes | 9 (29) |
| No | 19 (61) |
| Unknown | 3 (10) |
| PCP prophylaxis | |
| Trimethoprim-sulfamethoxazole | 9 (29) |
| Aerosolized pentamidine | 8 (26) |
| Dapsone | 6 (19) |
| Atovaquone | 1 (3) |
| None | 5 (16) |
| Unknown | 2 (7) |
| Previous pulmonary infection | 24 (77) |
| Previous bacterial pneumonia | 16 (52) |
| Previous PCP | 15 (48) |
| Previous opportunistic infection | 22 (71) |

NOTE. PCP = *Pneumocystis carinii* pneumonia.

* Unless stated otherwise, data are number (%) of patients with indicated characteristic.

## Clinical Features

Symptoms at the time of presentation included fever (90% of patients), cough (87%), shortness of breath (35%), pleuritic chest pain (26%), and hemoptysis (10%) (table 2). For two asymptomatic patients, lung abscess was noted on routine chest radiographs at the time of admission. The duration of symptoms ranged from 3 days to 2 months (mean, 14 days). The duration

**Table 2.** Clinical symptoms of 31 patients with AIDS and lung abscess at the time of presentation.

| Symptom | No. (%) of patients |
|---|---|
| Fever | 28 (90) |
| Cough | 27 (87) |
| Shortness of breath | 11 (36) |
| Pleuritic chest pain | 8 (26) |
| Hemoptysis | 3 (10) |
| Asymptomatic | 2 (6) |

of symptoms did not correlate with the type of pathogen isolated or the outcome.

The location of the abscess was in decreasing order of frequency the right upper lobe (39%), the left upper lobe (32%), the right lower lobe (16%), the right middle lobe (10%), and the left lower lobe (3%). Two patients had an abscess in more than one lobe, and one patient had multiple abscesses. Of 16 patients who underwent chest CT, nine (56%) had radiographic evidence of bronchiectasis.

The microbiological etiology was established for patients by sputum examination (26% of patients), BAL fluid analysis (26%), fine-needle aspirate examination (16%), BAL fluid analysis and transbronchial biopsy (10%), sputum and/or blood culture (10%), and blood culture (3%). No diagnosis was made for three patients despite extensive evaluation, including transbronchial biopsy. An average of 3.4 respiratory specimens (including both sputum and bronchoscopy specimens) from every patient were sent for mycobacterial culture.

As shown in table 3, the pathogens recovered were bacteria (65%), *P. carinii* (6%), fungi (3%), and mixed microorganisms (16%). The pathogens included *P. aeruginosa* (11), *Streptococcus pneumoniae* (6), *P. carinii* (5), *Klebsiella pneumoniae* (5), *S. aureus* (4), *Aspergillus* species (3), viridans streptococcus (2), *Haemophilus influenzae* (1), *Streptococcus milleri* (1), *Proteus mirabilis* (1), and *Cryptococcus neoformans* (1). *M. tuberculosis* was not isolated. More than one type of organism was recovered from five patients' abscesses, including two patients with mixed infections due to bacteria and fungi, two patients with mixed infections due to bacteria and protozoa, and one patient with a mixed infection due to bacteria, fungi, and protozoa. More than one bacterium was also recovered from four other patients; however, it was not revealed if these organisms were single or multiple pathogens.

*P. aeruginosa* was the most common organism isolated from the abscesses. Five patients had evidence of invasive disease: the organism was isolated from two patients' abscesses at postmortem examination, two patients had positive blood and sputum cultures, and the organism was isolated from one patient's fine-needle aspirate. Three of the four patients for whom *P. aeruginosa* was identified in sputum specimens had gram-stain evidence of gram-negative bacilli and polymorphonuclear leukocytes.

Compared with patients with infections due to pathogens other than *P. aeruginosa,* patients with *P. aeruginosa* infections were more likely to have a history of bacterial infection ($P = .002$), to be receiving prophylaxis with trimethoprim-sulfamethoxazole for *P. carinii* pneumonia ($P = .028$), and to be concurrently taking antibiotic therapy ($P = .04$). In addition, all patients with *P. aeruginosa* infections had a history of opportunistic infections, compared with only 50% of patients without pseudomonas infections ($P = .005$).

The microbiological cause of each abscess could not be predicted on the basis of clinical grounds. However, bacterial organisms were more likely to be recovered from abscesses in patients with previous bacterial pneumonia ($P = .016$), and all patients with a history of intravenous drug abuse had bacterial abscesses. Previous *P. carinii* pneumonia or other pulmonary infections, the type of prophylaxis for *P. carinii* pneumonia, bronchiectasis, and concurrent medications were not associated with a specific microbiological cause of lung abscess.

### Treatment and Outcome

Patients were treated for 2–12 weeks; 25% of the patients received >4 weeks of therapy. Six patients (19%) received empirical antituberculosis therapy; two of these patients had a clinical response.

Eleven patients (36%) had complete resolution of lung abscess. The average follow-up after diagnosis was 16 months. Two patients were lost to follow-up 3 and 6 months after presentation, respectively.

**Table 3.** Microbiological etiology of lung abscess in 31 patients with AIDS.

| Microbiological etiology | No. (%) of patients |
|---|---|
| Type of pathogen | 20 (65) |
| Bacteria | |
| *Pneumocystis carinii* | 2 (6) |
| Fungi | 1 (3) |
| Mixed* | 5 (16) |
| None | 3 (10) |
| Organism identified† | 11 (36) |
| *Pseudomonas aeruginosa* | |
| *Streptococcus pneumoniae* | 6 (19) |
| *P. carinii* | 5 (16) |
| *Klebsiella pneumoniae* | 5 (16) |
| *Staphylococcus aureus* | 4 (13) |
| *Aspergillus* species | 3 (10) |
| Viridans streptococcus | 2 (6) |
| *Haemophilus influenzae* | 1 (3) |
| *Streptococcus milleri* | 1 (3) |
| *Proteus mirabilis* | 1 (3) |
| *Cryptococcus neoformans* | 1 (3) |

* Two patients with mixed infections due to bacteria and fungi, two patients with mixed infections due to bacteria and protozoa, and one patient with a mixed infection due to bacteria, fungi, and protozoa.

† A total of 40 microorganisms were recovered.

Eleven patients (36%) had recurrence of lung abscess. Recurrences developed 2–16 months (mean, 8.6 months) after completion of the initial antibiotic course. Organisms recovered from recurrent lung abscesses included *P. aeruginosa* (4), *S. pneumoniae* (2), *S. aureus* (2), *H. influenzae* (1), *Serratia marcescens* (1), *Acinetobacter* species (1), *K. pneumoniae* (1), and *P. carinii* (1). Four patients had recurrence with the same organism, and four patients had more than one recurrence. In addition, four patients died of recurrent lung abscess.

Six patients (19%) died of lung abscess with active pulmonary infection. The average interval from the time of presentation to death was 3.5 weeks; three patients died within 2 weeks of presentation. Three patients (10%) died of unrelated causes within 3 months of the diagnosis and therefore were excluded from outcome analysis because the follow-up interval was too short (table 4).

Outcome was not related to the microbiological etiology of the abscess, a history of opportunistic infections, the number of organisms isolated, the number of abscesses, or the duration of treatment. Female patients had a worse outcome: 78% either died of lung abscess or had recurrent disease compared with 45% of male patients ($P = .04$). All three patients for whom a microbiological etiology was not established had resolution of lung abscess; two responded to empirical antituberculosis therapy, and one responded to treatment with broad-spectrum antibiotics.

## Discussion

We identified 31 patients with AIDS and lung abscess who were seen at The New York Hospital/Cornell University Medical College since 1989. All our patients had advanced HIV infection and CD4 cell counts of $<50/mm^3$, and most had a history of pulmonary infections. The microbiological etiology of lung abscesses was diverse, yet aerobic bacterial infections were most common. Multiple pathogens were isolated from almost one-third of the patients' lung abscesses. The prognosis was poor, with resolution occurring in only one-third of the cases.

The risk factors associated with the development of lung abscess in HIV-negative patients are well described. In the classic series by Bartlett et al. [4], 85% of the patients had altered mental status, and 46% had a history of alcohol use. Others investigators have defined periodontitis and gingivitis as risk factors [5, 22]. None of our HIV-positive patients had periodontitis or gingivitis; only 6% had recent altered consciousness, and 10% had a history of alcohol use. Potential associations identified in our series included advanced HIV disease, a history of pulmonary infections, and possibly bronchiectasis, which may be more common in patients with AIDS (especially compared with other immunocompromised patients) [23].

**Table 4.** Clinical outcome for 31 patients with AIDS and lung abscess.

| Outcome | No. (%) of patients |
|---|---|
| Recurrence of lung abscess | 11 (36) |
| Resolution of lung abscess | 11 (36) |
| Death due to lung abscess | 6 (19) |
| Died of unrelated causes within 3 mo of lung abscess | 3 (9) |

Because this study was retrospective, the diagnostic evaluation for each patient was not standardized. Therefore, it is difficult to draw any meaningful conclusions regarding the sensitivities of each diagnostic modality. Therefore, we are unable to make recommendations concerning the most appropriate workup for lung abscess in HIV-positive patients.

The microbiological etiology of lung abscess in HIV-positive patients was different from that described in HIV-negative patients. While pathogens recovered from HIV-negative patients most often are mixed anaerobic bacteria, anaerobic bacteria were not isolated from our patients, despite appropriate cultures. In addition, viridans streptococcus, which is commonly recovered from HIV-negative patients, was isolated from only two patients in our series. Furthermore, although bacteria were most commonly isolated, fungi and protozoa were recovered from 25% of the patients. This finding demonstrates that the microbiological etiology of lung abscess in HIV-positive patients is distinct from that previously described in HIV-negative patients.

*P. aeruginosa* was the most common organism isolated and was more often recovered from patients with prior AIDS-defining illnesses or late-stage disease than from other patients in this series. Furthermore, the rate of isolation of *Pseudomonas* organisms from lung abscesses was higher among patients who were concurrently receiving antibiotic therapy and prophylaxis with trimethoprim-sulfamethoxazole.

Recently, the role of *P. aeruginosa* infection in patients with AIDS has been increasingly appreciated [17, 24–26]. In their series of cases of *P. aeruginosa* bacteremia, Mendelson et al. [24] found cavitary lung disease in six of 12 patients with pneumonia. In another study of 15 patients with *P. aeruginosa* pneumonia [17], 50% had cavities at the time of admission, and 19% subsequently had cavitary disease. Similar to the patients in these series, none of our patients with pseudomonas infection were neutropenic or had intravascular devices.

The outcome for HIV-positive patients with lung abscess in our series was poor: 36% had a recurrence, and 19% died of lung abscess. In contrast, since the advent of broad-spectrum antibiotics, the outcome for HIV-negative patients with lung abscess has been excellent. In the series by Bartlett et al. [4], 24 of 26 patients responded to antimicrobial therapy, and no patient required surgical drainage; the two fatalities were unrelated to lung abscess. In another series [22], none of the 63 patients had a recurrence, despite the presence of residual cystic or bronchiectatic changes.

In HIV-negative patients, the clinical characteristics associated with death or relapse include longer duration of symptoms, the presence of bronchiectasis, concurrent medical discase, and cavity size [5, 27]. In our series, these features were not associated with a poor outcome. Instead, female gender and the presence of *P. aeruginosa* infection were associated with a poor outcome.

Surprisingly, a longer duration of antibiotic therapy was not associated with a better outcome. In our series, eight of 14 patients receiving 2–4 weeks of therapy and three of five patients receiving >4 weeks of treatment relapsed or died. In the series by Bartlett et al. [4], the outcome was very good; patients were treated from 3 to 20 weeks until regression of a cavity was noted on chest radiographs. We still believe that patients with AIDS and lung abscess should receive longer courses of treatment, although this belief is not borne out by our study (most likely because of the small sample size).

There were several limitations to our study. First, no standardized approach to diagnosis was used. Therefore, diagnosis for some patients was made by sputum examination, while others underwent more-invasive evaluations at which time additional pathogens were recovered. Second, not all the organisms isolated may have been actual pathogens. Five patients had mixed infections, and although coverage was provided for each pathogen, we cannot exclude the possibility that some organisms were colonizers. Third, because we did not have any control group, it is difficult to draw conclusions regarding risk factors for the development of lung abscess. Fourth, because not all patients' specimens were sent for anaerobic cultures, we may have underestimated the role of anaerobes as pathogens of lung abscesses in HIV-positive patients. Finally, although we did not find any significant association between duration of treatment and outcome, our relatively small sample size may have made it difficult to demonstrate any significant difference.

In summary, we have described the clinical features of and outcome for patients with AIDS and lung abscess. Lung abscess is seen exclusively in patients with advanced AIDS, is due to a broad spectrum of pathogens different from that causing lung abscesses in HIV-negative patients, responds poorly to antibiotics, and has a very poor prognosis. Future studies are needed to determine the optimal method of diagnosis and the best duration of treatment.

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