Endobronchial Mycobacterium avium-intracellulare Infection in a Patient with AIDS*


The pulmonary manifestations of AIDS are well described in the medical literature; however, MAI infection presenting as an endobronchial lesion has not, to our knowledge, been reported in a patient with AIDS. We report a unique case of an AIDS patient who developed endobronchial polypoid lesions secondary to MAI infection. Complications resulting from these lesions included hemoptysis and later bronchiectasis. (Chest 1989; 96:119-200)

The clinical manifestations of AIDS are many and diverse. The reported spectrum of pulmonary diseases has included opportunistic infections, Kaposi's sarcoma, non-specific lymphoid interstitial pneumonitis, adult respiratory distress syndrome, and even bronchospastic airways disease. Among the opportunistic infections, mycobacterial disease has emerged as an increasingly common manifestation of AIDS.

Infection with Mycobacterium tuberculosis in patients with AIDS is frequently severe and often presents with unusual manifestations such as fulminant extrapulmonary or disseminated infection. Another unusual finding that has been reported recently in a patient with AIDS is endobronchial tuberculosis. Endobronchial tuberculosis has been well described in patients without AIDS, but has become less common in the modern chemotherapeutic era.

Mycobacterium avium-intracellulare also produces pulmonary and extrapulmonary disease. Pulmonary involvement may range from asymptomatic colonization of the airway to invasive parenchymal or cavitary disease. To our knowledge, there have been no reports of endobronchial disease due to MAI patients with AIDS described in the English literature. We report a unique case of endobronchial MAI infection in a patient with AIDS.

**Case Report**

A 27-year-old man was admitted to the hospital with a ten-day history of worsening cough and mild dyspnea. His past history was significant for pneumonia due to Hemophilus influenzae and sinusitis, and presumed cryptomegaloviral pneumonia one month previously, which was diagnosed by culture of fluid from BAL obtained during fiberoptic bronchoscopy. Of note, the appearance of the endobronchial tree was entirely normal at that time. Current medications included oral nystatin, acyclovir, and AZT.

Upon admission, the findings on physical examination were significant for thrush, anterior cervical and submental lymphadenopathy, bibasilar crackles on pulmonary auscultation, and herpetic proctitis. Pertinent laboratory results included antibody to HIV virus by ELISA and Western blot assays, a WBC of 3,600/cu mm, and total T-lymphocyte count of 225/cu mm (normal, 870/cu mm to 2,415/cu mm), T-helper population of 17/cu mm (normal, 436/cu mm to 1,394/cu mm), and T-helper to T-suppressor ratio of 0.15. A chest roentgenogram revealed fine interstitial infiltrates, more prominent in the region of the RUL, without evidence of hilar or mediastinal adenopathy.

A recent PPD skin test was nonreactive. A Ziehl-Neelsen stain of expectorated sputum revealed acid-fast bacilli. Subsequently, cultures of BAL fluid from the previous bronchoscopy one month earlier grew MAI.

On the fifth day of hospitalization, the patient developed massive hemoptysis (approximately 300 ml of fresh blood over 12 hours). Coagulation studies and a platelet count were normal. Immediate bronchoscopic examination revealed fresh thrombus in the posterior segment of the RUL bronchus. No endobronchial lesions were seen, and endobronchial brushings revealed acid-fast bacilli. The hemoptysis resolved spontaneously. A computed tomographic scan of the chest showed no mass or cavitary lesion.

One week later, the patient presented to the emergency room again with copious hemoptysis. Bronchoscopy at this time revealed thrombus in the posterior segment in the RUL, and no endobronchial lesions were seen. Bronchial arterial angiography demonstrated abnormal vascularity, vascular blushing, and late extravasation in the posterior segment of the RUL. Selective embolization of this vessel brought resolution of the hemoptysis.

Follow-up bronchoscopy one month later revealed several endobronchial sessile based polypoid lesions, approximately 0.5 cm in diameter, located in the posterior segment of the RUL, as well as in the segmental bronchi of the RML, RLL, and LLL (Fig 1). Biopsy of these lesions showed necrotizing and nonnecrotizing granulomas, and culture of the tissue specimens grew MAI organisms.

Five months after these lesions were discovered, recurrent fever, hemoptysis, and a chest roentgenogram typical of bronchiectasis (Fig 2) prompted reexamination with bronchoscopy. Polypoid lesions were again seen in segmental bronchi of the RML, RLL, and LLL, with total occlusion of segmental bronchi in the RLL and LLL. Piecemeal resection with biopsy forceps resulted in removal of all four lesions. This allowed drainage of purulent secretions from distal segments of the airway.

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*From the Department of Pulmonary Disease (Drs. Mehle, Adamo, Mehta, and Wiedemann), and the Department of Infectious Disease (Drs. Keys and Longworth), Cleveland Clinic Foundation, Cleveland.

Reprint requests: Dr. Mehta, 9600 Euclid Avenue, Cleveland 44106

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**Figure 1.** Endobronchial photographs of segmental bronchi in RUL (A), RML (B), RLL (C), and LLL (D), revealing polypoid lesions representing endobronchial MAI infection.
DISCUSSION

Pulmonary and disseminated MAI infections are common in patients with AIDS. Recent reports have described documented MAI infection in 10 to 20 percent of the patients with AIDS during life and a prevalence of MAI infection at autopsy of 50 percent. Infection with MAI is usually a disseminated disease in AIDS, with a high incidence of positive cultures of blood and bone marrow, as well as frequent involvement of the reticuloendothelial system at the time of autopsy. Clinical and radiographic findings are often difficult to ascribe to MAI alone because of the high frequency of concomitant pulmonary disease.

Previously described biopsies of patients with AIDS who have MAI infections have demonstrated absence of granulomas or rare poorly formed granulomas with "foamy" macrophages and numerous acid-fast bacilli. This is in contrast to immunocompetent hosts with MAI disease in whom granuloma formation is prominent and the histopathologic features are indistinguishable from the lesions of ordinary tuberculosis. To our knowledge, there have been no published reports of polyoid endobronchial MAI disease in the English literature. Intraoral lesions have been described in a patient with AIDS who had disseminated MAI. In the era before AIDS, Merckx et al. reported the findings in a patient with multiple granulomatosus lesions in the pharyngolaryngeal region due to photochromogenic mycobacteria (likely not MAI). In addition, these investigators noted "endobronchitis" in four patients with nonchromogenic mycobacterial pulmonary disease, but these findings were not further characterized.

In this case report, we describe a young man with AIDS who was found to have endobronchial masses due to MAI. He had initially presented with hemoptysis and was subsequently found to have endobronchial lesions on follow-up bronchoscopy. These lesions contained necrotizing granulomas in two of four biopsies and nonnecrotizing granulomas in one biopsy. All of the granuloma specimens failed to disclose acid-fast organisms, fungi, or Pneumocystis on special stains. This finding is similar to typical nonendobronchial granulomas in MAI disease in immunocompetent hosts. Tissue cultures from our patient subsequently grew MAI. No other pathogens were isolated.

The finding of well-formed granulomas in our patient contrasts sharply with the poorly formed and AFB-laden granulomas reported in most MAI-infected patients with AIDS. This may reflect a lesser degree of immunocompromise in our patient or perhaps a beneficial effect of AZT therapy in partially restoring immune function. This is admittedly a highly speculative hypothesis.

In summary, we report a unique case of endobronchial MAI disease in a patient with AIDS. The histopathology more closely resembled that of nonimmunocompromised patients with MAI disease. Hemoptysis and bronchiectasis were complications associated with and likely attributable to this patient's endobronchial MAI infection.

ADDENDUM

Since the acceptance of this manuscript, a report has been published describing two patients with AIDS who had endobronchial lesions due to MAI infection. Notably, both patients were receiving AZT.

REFERENCES

13. Jagadha V, Andavolu RH, Huang CT. Granulomatous inflamma-
Sarcoidosis Diagnosed in a Patient with Known HIV Infection*

CDR Laurence E. Coots, MC, USNR‡ and
CDR Angelina A. Lazarus, MC, USN, F.C.C.P.¶

A 29-year-old black man with HIV infection had an abnormal chest x-ray film with bilateral hilar adenopathy. Sarcoidosis was suspected, but a thorough and comprehensive evaluation was completed to differentiate the multiple infectious and noninfectious causes of these findings. Biopsy of a hilar node and pulmonary tissue revealed sarcoidosis. (Chest 1989; 96:201-02)

Coexistence of HIV infection and active sarcoidosis appears to be quite rare. To date, there are only two case reports of HIV infection and sarcoidosis occurring in the same patient.1,2 and in both cases, sarcoidosis was diagnosed at least ten years before the HIV infection was documented. We report the findings in a patient who was found to have stage 1 sarcoidosis during the evaluation of HIV infection.

CASE REPORT

The patient is a 29-year-old black man who at the age of 26 years had had a normal chest x-ray film as part of a physical examination for enlistment in the armed forces. Two years later, during routine screening required by the Department of Defense, he was found to be positive for HIV antibody by ELISA and Western blot. During initial HIV evaluation, he denied all known risk factors for HIV infection. There was no occupational exposure to beryllium or other agents known to cause a granulomatous process. The patient was asymptomatic. The findings from physical examination were unremarkable except for generalized adenopathy. A chest x-ray film revealed bilateral hilar adenopathy (Fig 1). The PPD skin test was negative. Laboratory data included a WBC of 5,000/cu mm. T-cell subsets determined by flow cytometry revealed a total T-lymphocyte cell count of 1,569/cu mm; the T4 cell count was 666/cu mm; the T8 cell count was 963/cu mm, and the T4/T8 ratio was 0.75. The patient was classified by the CDC classification system† as HIV infection group 3.

At the required reevaluation one year later, the patient remained asymptomatic, and the generalized adenopathy was unchanged. A chest x-ray film again showed bilateral hilar adenopathy unchanged from the previous year's study. The PPD test remained negative. Laboratory data showed the WBC to be 5,300/cu mm, with a total T-cell count of 1,378/cu mm. The T4 cell count was 384/cu mm, the T8 cell count was 929/cu mm, and the T4/T8 ratio was 0.41.

A gallium scan revealed uptake in the hilar regions bilaterally. At thoracotomy, the right hilar nodes and the right lung were biopsied. Histopathologic studies revealed noncaseating granulomas in the node and pulmonary samples (Fig 2). Special stains failed to reveal fungi, acid-fast bacilli, or viral inclusion bodies. Cultures for fungi and mycobacteria showed no growth after eight weeks. Sarcoidosis was diagnosed on the basis of the histopathologic findings and the lack of evidence of other infections, inflammatory, or neoplastic conditions.

DISCUSSION

Serologic testing for HIV antibodies has been available since 1984. Flint et al3 tested 17 patients with sarcoidosis for HIV antibody using both ELISA and Western blot techniques. These investigators3 found all 17 to be negative, eliminating the concern over possible false-positive results. Of the two previously reported cases of concurrent HIV infection and sarcoidosis, one patient had serologic testing for HIV antibodies performed. That patient was positive by both the ELISA and Western blot methods.1 The other patient died in 1980 before testing was available.2 Our case report confirms that a patient with sarcoidosis can be accurately tested for HIV infection by the ELISA and Western blot tests.

Sarcoidosis has been reported in a variety of immunode-