Bronchoalveolar Lavage in Sarcoidosis and HIV Infection

To the Editor:

We read with great interest the case presented by Coots and Lazarus concerning a young black man who was found to be HIV positive on routine military screening and developed bilateral hilar lymphadenopathy compared to a chest film taken two years previously. Gallium uptake in the hilar lymph nodes performed one year later on reevaluation led to a diagnostic thoracotomy, with noncaseating granuloma found in the lung and lymph node biopsy. This case raises clinical questions about discerning of sarcoidosis from advanced HIV infection. We have diagnosed a similar case and wish to report the utility of bronchoalveolar lavage and flow cytometry in patients with both these diseases.

A 37-year-old white man was referred for evaluation of positive HIV serology and sarcoidosis. Medical history revealed a diagnosis of stage I sarcoidosis made in 1977 by mediastinal lymph node biopsy, high risk behavior for HIV infection, and a 30 pack-year smoking history. The patient was asymptomatic at the time of initial presentation and had received no therapy. A follow-up chest roentgenogram (CXR) in 1985 showed bilateral hilar and right paratracheal lymphadenopathy without parenchymal disease. HIV-positive serology was detected on routine military screening in June, 1987. The patient's physical examination was normal except for bilateral axillary lymphadenopathy. Laboratory evaluation revealed cutaneous anergy and a stable CRX film. Pulmonary function tests (PFTs) performed in June, 1987 revealed a normal forced vital capacity of 4.62 L (89 percent of predicted), a one-second forced expiratory volume of 3.89 L (97 percent of predicted), and a diffusion capacity for carbon monoxide 80 percent of predicted. The patient presented in February, 1988 with increasing dyspnea on exertion and fatigue. Physical examination, PFT, and CXR (Fig 1) film were unchanged. He was referred for evaluation of advanced HIV infection progression of sarcoidosis. Laboratory evaluation revealed a peripheral white blood cell count of 3.8 x 10^9/L (total lymphocyte count 1,100/cu mm), and a CD4 lymphocyte count of 120/cu mm with a CD4/CD8 ratio of 49 percent. Bronchoscopy with bronchoalveolar (BAL) was performed. BAL fluid showed 28 percent lymphocytes with a CD4/CD8 ratio of 34 percent. Special stains and cultures for pathogens were negative. The patient was not treated and received only close follow-up. A repeat bronchoscopy eighteen months later with BAL showed 32 percent lymphocytes with a CD4/CD8 ratio of 28 percent. The patient has remained free of symptoms to date.

Clinical features of this case are typical for both advanced HIV infection and sarcoidosis. Advanced HIV infection is suggested with a reversed CD4/CD8 ratio in both the lung and peripheral circulation. Bronchoalveolar lavage has shown utility in the staging and diagnosis of both disease. Flow cytometric analysis of lymphocyte subsets has been helpful in characterizing lymphocytic alveolitis with high CD4/CD8 ratios (ie, sarcoidosis and berylliosis) or low CD4/CD8 (ie, hypersensitivity pneumonitis). The differentiation of sarcoid alveolitis and HIV infection has therapeutic implications concerning the use of corticosteroid therapy and can be made with BAL and flow cytometry.

Warren L. Whitlock, M.D., F.C.C.P.; William S. Lovery, M.D., and Robert A. Dietrich, M.D., F.C.C.P., Pulmonary Disease Service, Department of Medicine, Letterman Army Medical Center, San Francisco

References

1 Coots LE. Sarcoidosis diagnosed in a patient with known HIV infection. Chest 1989; 96:201-02

To the Editor:

We read with interest the recent report by Coots et al entitled, "Sarcoidosis diagnosed in a patient with known HIV infection" (Ches 1989; 96:201-202) concerning the diagnosis of sarcoidosis in a patient with previously known HIV infection.

A 25-year-old bisexual black man was evaluated with a history of one week of pleuritic chest pain, non-productive cough, chills, and night sweats. Subsequent radiographic examination showed a 30 percent right pneumothorax, and serology for HIV antibody test was positive. The patient subsequently presented with bilateral hilar adenopathy, and the differential diagnosis included the possibility of lymphoma, granulomatous disease, histoplasmosis, tuberculosis, and other inflammatory conditions. On mediastinoscopy, however, the lymph node biopsy showed noncaseating granulomatous lymphadenitis without evidence of opportunistic infection and no evidence of malignancy. All special stains for the lymph nodes for mycobacteria and fungi were negative.

The patient also had evidence of a skin rash with epidermal changes of ichthyosis, and on biopsy this skin lesion revealed numerous non-caseating granulomas in the superficial and deep dermis.

We would like to call attention to this additional case as previously reported in the Virginia Medical Journal (1989; 3:122-24).

We would like to reiterate that a positive HIV antibody titer need not imply the presence of an opportunistic lung infection nor the existence of an immunodeficiency state and that tissue diagnosis should be pursued in patients who present in this setting.

Barry S. Dicicco, M.D., F.C.C.P.