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 90 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 mm (total lymphocyte count 1,100/L mm), and a CD4 lymphocyte count of 120/L mm with a CD4/CD8 ratio of 0.4 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.

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REFERENCES

To the Editor:

We read with interest the recent report by Coots et al entitled, "Sarcoidosis diagnosed in a patient with known HIV infection" (Chest 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; 3:122-34).

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.
Their pneumonitis.

Their almost report effusions, presence sarcoidosis. Their report supports this hypothesis in the case of nonspecific pneumonitis.

Dr. Dicicco's letter lead us to his case report, which confirms our report that sarcoidosis can develop in the presence of active HIV infection. In their article, Ingram et al postulate that the results of T4/T8 subsets in BAL may differentiate active sarcoidosis from nonspecific pneumonitis in HIV-infected patients. They did not report BAL findings in their case. The patient in our case study would not allow us to perform BAL, and as yet there have been no reports of BAL from a patient with HIV infection and active sarcoidosis.

In conclusion, we agree that the T4/T8 ratio in BAL might help to differentiate active sarcoidosis infiltrates from non-specific pneumonitis in patients with both sarcoidosis and HIV infection. We wait for the report of BAL results in such a patient. If the T4/T8 ratio is high, then a method of differentiation may be found. This information will lead to the proper therapy: steroids in conventional doses, or combined with thymostimulin, for the patient with active sarcoidosis; and observation for the patient with nonspecific pneumonitis.

Reference

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Reference


Mesothelial Cells and Tuberculous Pleuritis

To the Editor:

We read with great interest the report by Kam-Yung Lau (Chest 1989; 96:2:438-39) of two cases of pleural tuberculosis with significant numbers of mesothelial cells. We wish to make several comments.

As the author points out, at present it is believed that this finding almost excludes a diagnosis of tuberculous pleural effusion. However, various data in the literature and our own experience do not support this concept. Actually, we have recently found numerous mesothelial cells in a patient with tuberculous pleural effusion. Moreover, there are several series—some extensive—in which the presence of significant numbers of mesothelial cells was not a rarity. For instance, in a retrospective study of 88 tuberculous pleural effusions, the authors found more than 5 percent mesothelial cells in 15 patients (15 percent). Other studies have shown similar results.1,2

In pleural tuberculosis (as in other chronic pleural effusions), the extensive inflammation of both pleural layers prevents the exfoliation of mesothelial cells into the pleural space. However, in an early phase when the inflammatory process is acute and not extensive, it is possible to find numerous mesothelial cells. In fact, in patients with tuberculous pleuritis the disappearance of mesothelial cells on serial thoracentesis has been observed as the white cell count changes from neutrophil to lymphocyte predominance.1

Therefore we think, in accordance with Kam-Yung Lau, that pleural tuberculosis remains a diagnostic possibility in the presence of more than 5 percent mesothelial cells. If the thoracentesis is performed in an early phase of the disease, this finding is not exceptional.

Sebastián de los Santos-Sastre, M.D.; Emilia Barrot-Cortés, M.D., and Francisco L. Barragán-Marquez, M.D., Department of Internal Medicine, Infanta Elena Hospital, Huelva, Spain

References


To the Editor:

I am grateful to Santos-Sastre et al for their interest in my article. Their experience supports the notion that mesothelial cells in tuberculous pleural effusion may not be as rare as previous studies have suggested.

Dr. Santos-Sastre proposed that, in the early phase of pleural tuberculosis, the inflammatory process is not extensive and exfoliation of mesothelial cells into the pleural fluid is still possible. However, this does not seem to be applicable to the cases that I described. In my article, numerous mesothelial cells were found in both the early and late stages of pleural tuberculosis. In fact, in Case 1 of my report, serial thoracentesis revealed that mesothelial cells had increased from 3 to 6 percent. The cause for this unusual finding of numerous mesothelial cells in not clear. Further studies would be warranted to clarify the issue of mesothelial cells in tuberculous pleural effusions.

Kam-Yung Lau, M.D., F.C.C.P., Riverside Medical Clinic, Riverside, CA

Variables in Histamine Inhalation Tests

To the Editor:

Cockcroft et al (Chest 1989; 96:505-08) have elegantly and precisely documented and quantified evaporative water loss during standard histamine inhalation tests performed with Wright nebulizers.

The evaporative water loss was associated with a 11°C drop in measured temperature over ten minutes of nebulizer operation. After 2 min, the drop was 7°C. The authors stated they have no knowledge of such a measurement prior to their work.