A case of pulmonary sarcoidosis diagnosed in a human immunodeficiency virus (HIV)-infected man is reported. The transbronchial lung biopsy specimen revealed noncaseating granuloma. A comparison of the lymphocyte subsets in both peripheral blood and bronchoalveolar lavage fluid revealed a pattern more typical of HIV infection than of classic sarcoidosis. A course of prednisone led to improvement in symptoms, roentgenographic findings, lung volumes, and diffusion capacity. (Chest 1992; 102:1899-1901)

Sarcoidosis is a disease of unknown cause that has an estimated prevalence in the United States of 5 per 100,000 whites and 40 per 100,000 nonwhites. The human immunodeficiency virus (HIV) type 1 is estimated to infect 1 million individuals in the United States. There have been only four cases of coexistent sarcoidosis and HIV infection reported in the English literature. To date and to our knowledge, no simultaneous assessment of both peripheral and lung lymphocytes has been made. We add a case of coincident sarcoidosis and HIV infection, including an evaluation of cell counts and lymphocyte subsets both in peripheral blood and in the bronchoalveolar lavage (BAL) fluid.

**CASE REPORT**

A 35-year-old Hispanic man presented to the Metropolitan Hospital Center, New York, complaining of a nonproductive cough and dyspnea. The patient had a history of intravenous drug abuse for approximately ten years and had tested positive for HIV antibody three years previously in 1987. There was a history of a pneumococcal pneumonia in 1986 and orbital cellulitis in 1990 but no opportunistic infections had occurred. In December 1986 a chest roentgenogram was normal.

The patient had attended the outpatient infectious disease clinic since 1987, where he had complained intermittently of dyspnea on exertion and a nonproductive cough. A chest roentgenogram in June 1988 revealed hilar prominence. In March 1990, a gallium-67 lung scan showed diffuse uptake bilaterally. In October 1990, a chest roentgenogram showed bilateral interstitial and alveolar infiltrates (Fig 1). Computed tomography of the chest confirmed these findings and revealed bilateral hilar and mediastinal lymphadenopathy.

The patient consented to bronchoscopy in October 1990. At that time physical examination revealed a cachectic man who was febrile with normal vital signs. Small cervical and axillary lymph nodes were present. Rales were noted over the left lung field.

The peripheral white blood cell (WBC) count was 2,400 cu mm. The CD4 cell count was 310/cu mm, and the CD8 count was 578/cu mm. The CD4/CD8 ratio was 0.53 (Table 1). The SMA-20 revealed normal calcium. Total protein was 10.2 g/dl, and albumin was 2.9 g/dl. Spirometry showed a mild restrictive ventilatory pattern with a severely reduced diffusion capacity at 28 percent of predicted (Table 2).

A bronchoscopic evaluation revealed normal endobronchial anatomy and mucosa. Histologic study of transbronchial lung biopsy specimens showed noncaseating granuloma (Fig 2). Special stains and cultures for Pneumocystis, fungus, and mycobacteria were negative. The BAL had a total of 780 WBCs per cubic millimeter, of which 70 percent were lymphocytes. The total BAL T-lymphocyte count was 524/cu mm or 96 percent of total lymphocytes. The CD4/

| **Table 1—T-Lymphocyte Subsets in Peripheral Blood and Bronchoalveolar Lavage Fluid** |
|-----------------|-----------------|-----------------|
| **Peripheral Blood, cells/cu mm (%)** | **BAL, cells/cu mm (%)** | **Reference Range for Peripheral Blood, cells/cu mm (%)** |
| Total WBC count | 2,400 (100) | 780 (100) | 3,900-11,400 |
| Total lymphocyte count | 1,070 (45) | 546 (70) | 1,049-3,581 (10-47) |
| Total T lymphocytes (CD3) | 945 (89) | 524 (96) | 575-2,147 (50-84) |
| CD4 subset | 310 (29) | 44 (8) | 337-1,571 (31-59) |
| CD8 subset | 578 (54) | 470 (86) | 235-753 (13-33) |
| CD4/CD8 ratio | 0.53 | 0.09 | 1.2-3.8 |
Coexistent sarcoidosis with HIV infection (Newman et al) 1900

**FIGURE 2.** Transbronchial biopsy specimen showing a noncaseating epithelioid granuloma in a peribronchial location (hematoxylin-eosin, original magnification × 125).

CD8 ratio was 0.09 (Table 1). A diagnosis of sarcoidosis in an HIV-infected man was made.

Based on the BAL lymphocytosis and the patient's complaints of cough and severe dyspnea, prednisone therapy was started at 40 mg/day. Coincidentally, prophylaxis for tuberculosis was initiated with isoniazid. At follow-up four months later, cough had disappeared and dyspnea was markedly reduced. Pulmonary function testing showed normalization of lung volumes. Diffusion capacity had improved from 28 to 45 percent of predicted (Table 2). A chest roentgenogram showed clearing of infiltrates (Fig 3).

**DISCUSSION**

While the cause of sarcoidosis is as yet unknown, it has been reported to occur on occasion in certain immune

**Table 2—Pulmonary Function Test Results before and after Steroid Treatment**

<table>
<thead>
<tr>
<th></th>
<th>Observed (%) Predicted</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>10/26/90</td>
</tr>
<tr>
<td>FVC, L</td>
<td>3.71 (69)</td>
</tr>
<tr>
<td>FEV1, L</td>
<td>3.08 (69)</td>
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<tr>
<td>FEV1/FVC</td>
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</tr>
<tr>
<td>TLC, L</td>
<td>5.12 (72)</td>
</tr>
<tr>
<td>FRC, L</td>
<td>3.10 (88)</td>
</tr>
<tr>
<td>DECO, single breath</td>
<td>9.42 (28)</td>
</tr>
</tbody>
</table>

depicted states. Nevertheless, a causal relationship has not been established. Both sarcoidosis and HIV infection are well known to affect the numbers and relative proportions of inflammatory cells, particularly macrophages and lymphocytes. Furthermore, different effects can be seen in the lung and vascular compartments.17

In normal nonsmoking individuals, a BAL yields approximately 100,000 cells/ml. Macrophages and lymphocytes are the predominant cell types, representing approximately 90 percent and 10 percent of the total count, respectively. Neutrophils, eosinophils, and basophils constitute less than 1 percent of the total. Among the lymphocyte population, T cells constitute approximately 65 to 80 percent of the cells and B cells make up the remainder. The ratio of the CD4 helper cells to the CD8 suppressor cells is approximately 1.6 and is similar to their ratio in blood.7,9

In sarcoidosis, there is typically a severalfold increase in the total number of cells recovered in a BAL sample. Increases are predominantly in the macrophage and lymphocyte components, with the proportion of lymphocytes averaging about 30 percent of total BAL cells. Furthermore, the T-lymphocyte number increases relative to B cells and can account for up to 90 percent of total lymphocytes. Within the T-cell series, there is a relative increase of CD4 over CD8 cells and their ratio can increase to 10:1.17 The degree of lymphocyte alveolitis has correlated with lung tissue cellularity and has been proposed as a marker of disease severity.7

By contrast to the cellular alterations found in the lung compartment, patients with sarcoid demonstrate a T-cell lymphopenia in peripheral blood and a CD4/CD8 ratio that is usually less than 1. Some authors consider this as evidence of some degree of recruitment of CD4 cells from the circulating blood pool into the lung.1

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**FIGURE 3.** Chest roentgenogram showing clearing of infiltrates after prednisone therapy.

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Studies of BAL fluid in HIV-infected patients who have no clinical evidence of lung disease show that 70 percent of these individuals have a lymphocytosis due to an increase in CD8 cells. There is a decreased CD4/CD8 ratio similar to or even lower than that found in their peripheral blood. The absolute number of BAL lymphocytes may be increased tenfold, but there is usually peripheral lymphopenia. In contrast to sarcoidosis, patients with HIV demonstrate a positive correlation between the percentages of CD4 cells in blood and in lung. 

Although sarcoidosis occurring in HIV-positive patients has been reported, to date and to our knowledge, no simultaneous assessment of both peripheral blood and BAL lymphocyte subsets has been made. Ingram et al described a 25-year-old HIV-positive black man who subsequently developed hilar adenopathy and was found to have noncaseating granuloma in a hilar lymph node biopsy specimen. The angiotensin-converting enzyme level was elevated and the peripheral T-helper/.suppressor ratio was 0.59. In a case reported by Coots and Lazarus, HIV infection and pulmonary sarcoidosis were diagnosed simultaneously. Again, the peripheral blood CD4/CD8 ratio was low at 0.41.4

AIDS has also been reported to follow the diagnosis of sarcoidosis. Kalter and Lopez-Berestein described a 35-year-old bisexual black man who had sarcoidosis for ten years prior to the onset of AIDS. Peripheral T-helper cell numbers were reduced. A second case of sarcoid predating the onset of HIV infection by ten years was reported by Wurm et al.

When HIV seropositivity and pulmonary sarcoidosis occur in the same individual, an opportunity exists to evaluate how the two disease entities interact in view of their opposite effects on the BAL CD4/CD8 ratio. In sarcoidosis, high CD4 cell counts have been considered to represent an enhanced and abnormally modulated immune response associated with lung injury. By contrast, the low CD4 counts in patients with HIV predispose to opportunistic infection. In the case described in this report, the low CD4/CD8 ratio in the BAL is more characteristic of HIV infection than of sarcoidosis. Nevertheless, steroid treatment resulted in clinical, physiologic, and roentgenographic improvement, as might occur in non HIV-positive pulmonary sarcoidosis. It seems unlikely that our patient had an HIV-related infiltrate responsive to steroids alone since such a condition has not been generally reported.

This case demonstrates that sarcoid granuloma can occur in immune-deficient HIV-infected individuals and that an elevated CD4 count and CD4/CD8 ratio are not necessary prerequisites for lung injury in sarcoidosis. Thus, other cells and mediators may be involved in sarcoid lung injury in the absence of CD4 lymphocytosis. The complex relationship of cells and mediators leading to an enhanced inflammatory response in coexistent sarcoidosis and HIV infection merits further study.

ACKNOWLEDGMENT: The authors wish to express their gratitude to Maggie Collins for secretarial support.

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**Mediastinal Bronchogenic Cyst* A Cause of Upper Airway Obstruction**

Michael Lippmann, M.D., F.C.C.P., Robert Solit, M.D., Steen K. Goldberg, M.D., F.C.C.P., and Denise Najjar, M.D.

Although bronchogenic cysts may involve the mediastinum, they have rarely been responsible for significant upper airway obstruction. We describe a young man who had a rapidly expanding cervical mass due to the migration of a mediastinal bronchogenic cyst. Flow-volume loops confirmed the presence of a variable intrathoracic obstruction. The patient rapidly developed respiratory failure requiring urgent intubation and surgical resection.

(Chest 1992; 102:1901-03)

A young, previously active man presented with severe life-threatening upper airway obstruction due to an enlarging posterior mediastinal mass. At surgery, the mass was found to be a large bronchogenic cyst. The pathophysiology is discussed adding mediastinal bronchogenic cysts to the list of disorders associated with a variable intrathoracic obstruction.

**CASE REPORT**

A 26-year-old black male athlete, with no pulmonary history,