Diagnosis of Pulmonary Strongyloidiasis by Bronchoalveolar Lavage*

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Bronchoalveolar lavage was performed on a patient with disseminated strongyloidiasis and $4.5 \times 10^9$ cells/65 ml of lavage fluid were recovered. Eighty-five percent of cells were polymorphonuclear leukocytes; 15 percent were pulmonary alveolar macrophages. Rhabditiform larvae ($1 \times 10^9$) were recovered in 65 ml of lavage fluid. This is the first report of bronchoalveolar lavage used in diagnosing disseminated strongyloidiasis. (Chest 1988; 94:643-44)

Strongyloides stercoralis is an intestinal nematode endemic to certain parts of the southeastern United States. In persons with defects in cell-mediated immunity, a hyperinfection syndrome can occur. Pulmonary manifestations of this syndrome include symptoms of chronic bronchitis, asthma, hemoptysis, eosinophilia, and pulmonary infiltrates.1-4 In a patient population with a high prevalence of chronic lung disease, many of these symptoms are nonspecific and the diagnosis is often missed. We have previously described the utility of the routine Gram stain of sputum in the diagnosis of pulmonary Strongyloides stercoralis.1-4 We describe a patient with diffuse pulmonary infiltrates and fever in whom bronchoalveolar lavage was performed to diagnose suspected opportunistic infection. The diagnosis of pulmonary strongyloidiasis was made by visualizing larvae in large numbers in the lavage fluid.

**CASE REPORT**

A 61-year-old man with a long history of chronic obstructive pulmonary disease, maintained on therapy with 10 mg of prednisone daily, was admitted to the Veterans Administration Medical Center (Johnson City, TN) with complaints of progressive shortness of breath, fever, and purulent sputum production. A month prior to admission he was found to have a left hilar mass on chest x-ray examination. Subsequent bronchoscopy and biopsy revealed squamous cell carcinoma of the left lower lobe; computerized tomography of the chest indicated mediastinal involvement. Outpatient radiation therapy was begun and, by the time of readmission, he had received 3,000 rads.

Physical examination at the time of admission revealed a cachetic white man in moderate respiratory distress using accessory muscles of respiration. Pertinent physical findings included bilateral rhonchi and crepitations at both lung bases. The patient showed ecchymosis and thin, easily bruised skin consistent with long corticosteroid use. White blood cell count was 4,200/cu mm with 31 percent polymorphonuclear leukocytes, 45 percent bands, 5 percent lymphocytes, 7 percent mononuclear cells, and no eosinophils. Arterial blood gas study revealed pH 7.48, Po$_4$ 49, Pco$_2$ 30.8, and Hco$_3$ 22 while breathing room air. Chest x-ray examination revealed bilateral diffuse lower lobe alveolar infiltrates. Initial Gram stain revealed many polymorphonuclear leukocytes and gram-negative rods, and eventually grew Pseudomonas aeruginosa. Blood and urine cultures revealed no growth. The patient was empirically treated with amikacin (500 mg intravenously every 12 hrs) and ticarcillin (3 g/mg intravenously every 4 h). The patient also received therapy with intravenous aminophylline, albuterol updrafts, and dexamethasone (4 mg orally daily). The patient appeared to gradually improve, but 11 days after admission he developed worsening hypoxemia, diffuse bilateral alveolar filling infiltrates on chest x-ray film, gastrointestinal bleeding, and respiratory failure requiring intubation and mechanical ventilation.

Bronchoscopy revealed the absence of any obstruction in the major bronchi, and changes in the mucosa of the left main bronchus consistent with radiation effect. Bronchoalveolar lavage was performed using standard techniques.5 A return of approximately 65 ml of lavage fluid was obtained. From other segments of the lung, bronchial brush specimens were obtained using both a sheathed protected catheter as well as unprotected brush specimens. Fluid was centrifuged at 500 g for four minutes; the pellet was resuspended in 0.8 ml of saline solution and cells counted.

Cytocentrifuge preparations were used to determine the differential count using Wright's stain and nonspecific esterase stain. Cell count in lavage fluid revealed $4.5 \times 10^9$ cells in the 65 ml of lavage fluid with 85 percent polymorphonuclear leukocytes and 15 percent pulmonary alveolar macrophages. Numerous rhabditiform larvae were seen on wet preparations and on Gram stain of the lavage fluid (Fig 1). Using a hemocytometer counting chamber, it was possible to estimate the number of larvae present at $1 \times 10^9$ per 65 ml lavage fluid. Special stains and cultures of the lavage fluid and protected brush specimens revealed no other opportunistic pathogen. Stool examination revealed Strongyloides stercoralis larvae. Thiabendazole therapy (1.5 g orally twice a day) was begun and continued until the patient's demise 20 days after admission. Sputum Gram stain continued to show Strongyloides stercoralis larvae despite therapy with thiabendazole. Death was from irreversible cardiorespiratory failure. At autopsy, the patient was found to have disseminated Strongyloides infection, metastatic squamous cell carcinoma, bilateral hemorrhagic pneumonia, severe tracheitis and bronchitis with bronchiectasis of right middle and lower lobes.

**DISCUSSION**

The antemortem diagnosis of pulmonary Strongyloides has been made in many different ways. As far back as 1911, Gage6 noted the presence of larvae in a wet preparation of sputum. Since that time, diagnosis has been made by examination of the sputum, bronchial brushings, pleural fluid, and bronchial biopsy.3-7,12

To our knowledge, our case is the first report of the use of

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**FIGURE 1.** Rhabditiform larvae of Strongyloides stercoralis in bronchoalveolar lavage fluid. Trichrome stain.
bronchoalveolar lavage in the diagnosis of pulmonary strongyloidiasis. The finding of a predominant neutrophil response in the lung of our patient is interesting as traditionally the response to parasitic infection is thought to involve lymphocytes, eosinophils, and macrophages. The paucity of eosinophils could have been related to the prior use of corticosteroids. The extremely high larval burden (10^8 larvae in 65 ml of lavage fluid) provides an appreciation of the gravity of this disease.

Our previous experience with seven patients with pulmonary strongyloidiasis has demonstrated that patients with chronic lung disease who are receiving corticosteroid therapy are at highest risk.3,4 Five of the seven patients with pulmonary strongyloidiasis died; death was not from Strongyloides hyperinfection, but from bacterial infection (bacteremia in two, nosocomial pneumonia in all) superimposed on the severe underlying diseases. Six of the seven patients received treatment with antacids and all but the two patients who survived received cimetidine. This raises the possibility that gastric acid may in some fashion be protective.3 Further support for this hypothesis comes from a prospective screening study of stools of hospitalized and domiciliary patients at our institution which revealed the presence of Strongyloides in stool samples of 6.1 percent of hospitalized patients and 2.6 percent of domiciliary patients.34 Infected patients were more likely to have eosinophilia and neutrophilic stools and to have been treated with corticosteroids, antacids and cimetidine. Screening of stool specimens from patients with these risk factors in an area endemic for Strongyloides seems worthwhile. Standard thiabendazole therapy in stool-positive patients has a relapse rate of 15 percent.14

Pulmonary strongyloidiasis is a significant hazard in the patient with chronic lung disease on corticosteroid therapy; it must be added to the list of opportunistic pathogens that can be diagnosed by bronchoalveolar lavage.

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Pulmonary Leiomyomatosis
Showing Endobronchial Extension and Giant Cyst Formation*

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A 49-year-old woman who had previously undergone hysterectomy was found to have multiple pulmonary masses and cysts and experienced respiratory failure. Bronchoscopic study revealed an endobronchial mass obstructing the left main bronchus and protruding into the trachea. She was treated with an endoscopic laser to eliminate the airway obstruction. After recovery from the respiratory failure, two-stage thoracotomies were performed at six-month intervals. The endobronchial mass originated from a tumor in the left basal segment. The histologic findings on the cysts and nodules of both lungs were compatible with fibroleiomyoma. The final diagnosis was pulmonary leiomyomatosis with myoma uteri. (Chest 1993; 94:644-46)

Pulmonary leiomyomatosis is a very rare disorder. The present case is an unusual type of pulmonary leiomyomatosis, which showed endobronchial growth and giant cyst formation.

Case Report

A 49-year-old woman with respiratory failure was referred for evaluation and treatment of an endobronchial tumor. Abnormal bilateral cystic shadows had been detected by routine chest roentgenograms 20 years before. The size of the cysts had increased gradually. Some solid round shadows had changed into cystic lesions. She had received no treatment because she was asymptomatic. She had undergone a simple hysterectomy for myoma of the uterus 20 years before admission. Chest x-ray films on admission disclosed pneumothorax and atelectasis in the left hemi-

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