travenous dexamethasone, and sedation while on mechanical ventilation. Twenty-four hours after the original resection, his right mainstem bronchus was enlarged to a diameter of 4 to 6 mm along the length of the tumor and smoothed by the use of the Nd-YAG laser through the flexible bronchoscope. The patient was immediately extubated, breathing spontaneously, without evidence of pulmonary hyperinflation or collapse (Fig 3).

DISCUSSION

Nd-YAG laser resection of endobronchial tumors has been shown to have utility in the care of patients with both malignant and nonmalignant obstruction of the mainstem bronchi.4 Complications include bleeding, intra-airway fire, anesthetic complications, airway perforation and pneumonia, all of which are potentially fatal. This patient demonstrates other serious potential complications of laser bronchoscopy: pulmonary hyperinflation distal to an area of recently opened obstruction, as well as postprocedure wheezing due either to bronchospasm or partial obstruction of a newly opened airway. Postprocedure bronchospasm can be due to reflex, inhaled smoke, or blood aspirated during the resection. The elevation of intrathoracic pressures during his forced exhalation may have caused enough dynamic airway collapse and obstruction of the mildly tortuous, recently opened right mainstem bronchi to produce a one-way valve effect leading to right lung hyperinflation. Despite the severe obstruction noted in the left mainstem bronchus, the bulky extrinsic tumor may have effectively stented the airway open and allowed the left lung to empty of air and progressively collapse. Positive pressure ventilation with PEEP overcame the obstruction of the right mainstem bronchus. Treatment for bronchospasm and subsequent removal of irregularly shaped tissues in the right mainstem bronchi relieved the one-way valve effect and allowed extubation.

This patient demonstrates that partial return of function in an area of the tracheobronchial tree after laser resection of endobronchial tumor may worsen pulmonary mechanics due to air trapping and hyperinflation. Resections should be planned to return the airway to as nearly normal caliber as possible. Irregularities of the bronchial wall should be minimized and the resected airway should be as widely patent as possible. Positive pressure ventilation and aggressive treatment of bronchospasm in the postresection period may be used to relieve the one-way valve effect in the event of hyperinflation, prior to more definitive therapy.

REFERENCES


Recovery from Cryptococcosis and the Adult Respiratory Distress Syndrome in the Acquired Immunodeficiency Syndrome*

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We describe a patient who presented with cryptococcosis and the adult respiratory distress syndrome (ARDS) as the initial manifestation of the acquired immunodeficiency syndrome. This patient represents the first reported recovery from ARDS secondary to widespread cryptococcosis. He is currently doing well as an outpatient on maintenance therapy with amphotericin B and azidothymidine.

*Cryptococcus neoformans* infects 6-13 percent of patients with the acquired immunodeficiency syndrome (AIDS).1,4 In greater than half of the AIDS patients infected by *Cryptococcus*, it is the initial opportunistic pathogen.1,3 Because of the propensity for *Cryptococcus* to disseminate in the immunocompromised host, diverse clinical presentations can result.4,5 We now describe a homosexual male who presented with the adult respiratory distress syndrome (ARDS) and cryptococcosis as the first manifestation of AIDS.

CASE REPORT

A 26-year-old black homosexual male, known to have positive antibody titers to human immunodeficiency virus (HIV), presented with the complaint of progressive dyspnea for one week. He had previously felt well except for intermittent fevers, which were presumed secondary to AIDS-related complex (ARC). He denied cough, hemoptysis or chest pain and there was no prior history of opportunistic infection. Bilateral hilar adenopathy had been noted previously on a chest radiograph.

On admission, the patient was in moderate respiratory distress, breathing 28 times per minute. Physical examination revealed a temperature of 39.4°C, oral thrush and minimal anterior cervical adenopathy. Chest auscultation was significant for diffuse end-inspiratory crackles. The neurologic examination was unremarkable. There were no skin lesions.

The white blood cell count was 4,800/cu mm with 65 percent neutrophils, 15 percent band forms and 17 percent lymphocytes. Lymphocyte subset studies revealed an OKT3, of 2 percent (nl 40-60 percent) and an OKT, of 54 percent (nl 20-40 percent). The alkaline phosphatase level was 459 IU/L (nl 95-300); SGOT, 209 IU/L (nl 15-55); SGPT, 107 IU/L (nl 2-46); and the LDH, 545 IU/L (nl 80-180). Arterial blood gas levels on room air showed PaO2, 54 mm Hg; PaCO2, 30 mm Hg; pHa, 7.47. The initial chest radiograph revealed bilateral hilar adenopathy and bibasilar interstitial and alveolar infiltrates (Fig 1).

The patient was empirically treated with erythromycin, cefotaxime and trimethoprim-sulfamethoxazole. During the 12 hours fol-

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Cryptococcosis, ARDS and AIDS (Murray et al)
lowing admission, his respiratory status progressively deteriorated and he was intubated and placed on mechanical ventilation. Auscultation of the chest again revealed only end-inspiratory crackles; results of the cardiac examination were normal. The \( \text{PaO}_2 \) level was 78 mm Hg inspiring 80 percent oxygen with 5 cmH\(_2\)O of positive end-expiratory pressure (PEEP). A repeat chest radiograph now showed diffuse alveolar infiltrates (Fig 2). Right heart catheterization demonstrated pulmonary artery pressure of 25/14 mm Hg and a pulmonary artery occlusion pressure of 13 mm Hg. The cardiac output was 12.9 L/min.

Bronchoalveolar lavage specimens obtained by fiberoptic bronchoscopy revealed organisms morphologically consistent with \textit{Cryptococcus neoformans}. His antibiotic therapy was changed to intravenous amphotericin B (60 mg/day) and oral ketoconazole (400 mg/day). A lumbar puncture was performed and cerebrospinal fluid (CSF) examination revealed one white blood cell/cu mm and normal levels of glucose and protein. Findings on India ink preparation were negative. Subsequently, both blood and bronchial lavage fluid grew \textit{Cryptococcus neoformans}; the CSF remained culture negative. The cryptococcal antigen titer was reactive in the serum at 1:65,536 and at 1:4 in the CSF.

Eighteen hours after admission, the systolic blood pressure fell to 80 mm Hg. Blood pressure was supported with dopamine and oxygenation was maintained with increasing levels of PEEP and reversal of the ventilatory inspiratory:expiratory ratio.

During the next six days, the patient defervesced and there was gradual improvement in his oxygenation. On the ninth hospital day he was successfully extubated. Three weeks later the patient was discharged. Over the past five months, he has received maintenance amphotericin B therapy (40 mg IV three times weekly) and azidothymidine (AZT, 100 mg every four hours). He has done well and has no pulmonary complaint. His current chest radiograph shows only hilar adenopathy and the liver function test results have normalized.

**DISCUSSION**

Although fungal disease is recognized as an infectious cause of ARDS, in only three previous cases has Cryptococcus been reported to cause this syndrome. In 1962, Kent and Layton\(^6\) described two patients who had disseminated cryptococcosis after corticosteroid therapy for chronic liver disease or acute lymphocytic leukemia. At postmortem examination, both patients were found to have proteinaceous alveolar exudate in the lungs. In 1985, Perla and colleagues\(^6\) described a 32-year-old Haitian man, presumed to have AIDS, who developed ARDS and was found to have extensive cryptococcal disease on open lung biopsy. That patient subsequently died secondary to complications from progressive central nervous system infection. Our patient, to the best of our knowledge, is therefore the first reported recovery from ARDS secondary to disseminated Cryptococcus.

Little correlation appears to exist between the initial clinical presentation of cryptococcal infection and subsequent outcome. Kovacs et al\(^7\) examined the course of 27 infected AIDS patients and found no clinical or laboratory parameter that reliably predicted survival. Zuger et al\(^7\) studied 24 additional patients and found that only recurrent CNS infection or a cryptococcal cerebrospinal fluid antigen titer of 1:10,000 or greater reliably predicted death. These investigators also observed that prognosticators of poor outcome in previous studies such as high CSF opening pressure, low CSF glucose level or leukocyte count, or the presence of body fluid cultures positive for Cryptococcus were not helpful. The recovery of our patient from cryptococccemia and ARDS, a syndrome with 50 percent mortality regardless of etiology, again emphasizes that clinical presentation is a poor predictor of eventual outcome.

Currently, there are no clear guidelines for the use of mechanical ventilation in AIDS patients. Since AIDS is ultimately fatal, and very few patients survive ventilatory support, any decision regarding intubation and mechanical ventilation must include frank discussion with the patient about their prognosis.\(^8,9\) However, our case demonstrates that aggressive intervention may favorably influence survival and the quality of life in selected patients.

We have described a patient who presented with disseminated cryptococcosis and the adult respiratory distress syndrome as the initial manifestation of AIDS. The patient survived ARDS and continues to do well as an outpatient maintained on therapy with amphotericin B and AZT. Because of its propensity to cause disseminated disease in immunocompromised patients, Cryptococcus should be
considered as a possible infectious etiology in AIDS patients who develop ARDS. Additional information needs to be collected to determine the overall impact of intensive care unit interventions, maintenance amphotericin B therapy, and AZT on the course of disseminated cryptococcosis in AIDS patients.

REFERENCES

Castleman's Disease*

An Uncommon Computed Tomographic Feature

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A case of localized intrathoracic Castleman tumor demonstrated, on CT scan, calcifications in a circumferential distribution. The prevalence of calcifications in Castleman's disease and the differential diagnosis of the above unusual CT findings are discussed.

Castleman's disease is characterized by localized lymph node enlargement, most commonly located in the

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FIGURE 1. Frontal chest x-ray film demonstrating right mediastinal mass lesion.

The chest. The typical histopathologic findings are follicular hyperplasia with either proliferation of hyalized blood vessels (hyaline-vascular type) or abundance of plasma cells (plasma cell type).

The lesion is usually discovered while evaluating nonspecific complaints (low grade fever, night sweats, or recurrent URTIs).

The radiologic and computed tomographic findings of Castleman's disease (angiofollicular lymph node hyperplasia) in the chest have been described. 1*

We report a case of localized Castleman's disease presenting as a mediastinal mass with an uncommon pattern of calcification, best demonstrated by the CT scan.

CASE REPORT

A 28-year-old man was admitted for investigation of nonspecific complaints: chest pain, malaise, and dizziness. The patient had been involved in a serious car accident 18 years prior to the current hospitalization and sustained a craniocerebral injury manifested as hemiparesis and dysarthria.

History revealed no constitutional complaint other than night sweats and occasional headaches. On physical examination, a rib cage deformity of the left hemithorax, dysarthria, and left hemiparesis were noted, all results of the road accident 18 years before. Complete medical work-up was normal, with the exception of prolonged PT and ECG showing inverted T waves in leads 1, aVL, V1, V2.

FIGURE 2. CT scan at the level of the main pulmonary arteries shows right mediastinal and hilar mass containing calcified densities in a peripheral distribution (arrows).