Pulmonary Malacoplakia and Rhodococcus equi Infection in a Patient with AIDS*

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We describe a case of pulmonary malacoplakia and Rhodococcus equi cavitary pneumonia in a patient with AIDS. The relationship between Rhodococcus equi, a rare bacterial human pathogen, and malacoplakia, an unusual type of chronic granulomatous inflammation, is discussed.

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A number of infectious complications and immunologic aberrations have been reported in patients with AIDS. Malacoplakia, a distinct chronic granulomatous inflammation reflecting impaired host defenses, has not yet been reported. Rhodococcus equi is the proposed classification for the aerobic actinomycete previously classified as Corynebacterium equi. Rhodococcus equi is commonly found in soil around livestock areas and typically causes suppurative pneumonia or visceral abscesses in foals. Its pathogenicity in humans is rather uncommon, with only 22 cases of human disease reported. Most human cases of infection with R. equi have occurred in patients with defective cellular immunity and have manifested predominantly as pneumonia. Eight of the reported cases involved subjects with HIV infection, six of whom had cavitary pneumonia, usually in an upper lobe. Herein we report a case of pulmonary malacoplakia in a man with R. equi pneumonia and AIDS.

**CASE REPORT

A 25-year-old homosexual male horse trainer was relatively well until August 1987, when he developed a persistent, pruritic vesicular rash on an erythematous base over the dorsa of his hands and upper extremities. Eschars and depigmentation developed after the vesicles ruptured. Skin biopsy and biochemical studies confirmed a diagnosis of PCT. In February 1988, the patient was treated for Hemophilus influenzae pneumonia. Antibodies to HIV were present. After treatment, the patient remained afebrile, and his chest x-ray film remained abnormal.

In May 1988, the patient presented complaining of severe dyspnea, cough productive of foul-smelling, brown sputum, right-sided pleuritic chest pain, odynophagia, and a 9.1-kg (20-lb) weight loss. Physical examination revealed fever, tachypnea, the previously described rash, cachexia, axillary lymphadenopathy, and oropharyngeal thrush. Laboratory studies included the following: pH 7.60; PaCO2, 17 mm Hg; PaO2, 43 mm Hg; chest x-ray film with a right upper lobe consolidated mass and distal infiltrate (Fig 1); WBC 5.7 × 109/L (0.80 neutrophils, 0.11 lymphocytes; 0.09 monocytes).

FIGURE 1. Rhodococcus equi pneumonia presenting as right upper lobe consolidated mass with distal infiltrate in patient with AIDS.

Abnormal hepatic function tests (alanine and aspartate aminotransferases, lactate dehydrogenase, and alkaline phosphatase); and presence of antibodies to HIV and to the core and surface antigens of hepatitis B virus. The patient fulfilled criteria for presumptive diagnosis of esophageal candidiasis and, therefore, AIDS.

Transbronchial biopsy and BAL of the right upper lobe disclosed intra-alveolar collections of fibrin and granulocytes and negative methenamine silver and AFB stains of all specimens. Initial cultures grew only Proteus species. Based on sensitivity patterns, oral ciprofloxacin (750 mg/12 h) was given. Within 24 hours the patient became asymptomatic and afebrile. The chest x-ray film showed cavitation developing in the right upper lobe.

The patient was discharged, and he discontinued his therapy with ciprofloxacin. In three weeks, he returned complaining of fever

FIGURE 2. Malacoplakia. Section reveals sheets of histiocytes with abundant granular cytoplasm infiltrating pulmonary parenchyma. Note: dark, round, targetoid, classic MG body in center of field (hematoxylin-eosin, original magnification × 400).
and dyspnea. A chest x-ray film showed increased right upper lobe cavitation. Again, TBB with BAL was performed. Smears and cultures for AFB were negative. Fungal cultures of sputum and BAL showing Candida albicans were thought to represent oral contamination. Methenamine silver stains were negative. Transbronchial biopsy showed alveolar hemorrhage and fibrin deposition. Gram stains of BAL showed 1 to 10 WBCs per high-power field, without organisms. Routine chocolate and 5 percent sheep blood agar cultures of the BAL and specimens of sputum grew mucoid nonhemolytic colonies that were subsequently found to be R equi. Therapy was changed to intravenous vancomycin (1 g/12 h) and oral rifampin (300 mg/12 h) based on sensitivity studies. Because the patient's condition did not improve and the serial chest x-ray films showed increasing cavitation of the right upper lobe and a new right-sided pleural effusion, he underwent a right upper lobectomy. At surgery the right upper lobe was markedly adherent to the parietal pleura, hilar structures, and right middle lobe, necessitating removal of the middle lobe in addition.

On gross examination the apical segment of the right upper lobes revealed a 13.5-cm firm portion covered by pleura and a rim of pulmonary parenchyma. A cross-section through the firm portion revealed a 7.5-cm inner necrotic abscess cavity filled with friable tan to light-brown material partially adherent to an incomplete abscess cavity lining.

Microscopic sections of the abscess wall revealed a focally persistent bronchial mucosal lining. The remainder of the bronchial wall was obliterated by confluent sheets of histiocytes appearing slightly larger than usual alveolar macrophages and containing abundant, delicately reticulated and granular cytoplasm (von Hansemann cells). Occasional cells showed classic MG bodies (Fig 2) pathognomonic for malacoplasia. After surgery, the patient's condition slowly improved, and he was discharged on therapy with rifampin, erythromycin, and ciprofloxacin.

**DISCUSSION**

This case is unique in that it is the first reported case of malacoplasia in a patient with AIDS who, additionally, has the rare features of R equi pneumonia and PCT.

Malacoplasia is a rare but distinct type of chronic granulomatous inflammation usually affecting the lower urinary tracts of women. The presence of MG bodies and the characteristic von Hansemann histiocyte distinguish malacoplasia from other types of inflammation. The rounded MG body has a targetoid appearance that represents a variegated and complex phagolysosome core mineralized by deposition of electron-dense spicules and surrounded by one or more peripheral rings of more homogeneous aggregated granular, filamentous, and membranous material. The cytoplasm of the von Hansemann histiocyte may contain basophilic nuclear fragments of ingested degenerate neutrophils, but its enhanced granularity is due to an abundance of phagolysosomes or lysosomal bodies.

Defective host immunity and concomitant bacterial infection of tissue are both thought to be pathogenetic factors in the development of malacoplasia. The bactericidal activity of histiocytes may be reduced in some cases of malacoplasia, and an impairment of the histiocyte's ability to fully digest and eliminate products of intracellular killing is probably reflected by the presence of multiple complex lysosomal bodies. It has been postulated that excess accumulation of these bodies may interfere with other cellular functions and aggravate the process further. Rhodococcus may challenge the impaired ability of the histiocytes in patients with AIDS and predispose to the development of malacoplasia.

Similarities between malacoplasia and Whipple's disease are noteworthy. Despite clinical and cytologic differences, both disorders are associated with impaired processing of microorganisms within histiocytes. Whipple's disease or Whipple-like disease occurs occasionally in people with AIDS. Most of the cases of Whipple-like disease in AIDS were caused by infection with MAI.

Interestingly, one case of Whipple-like disease in an HIV-infected patient was thought to be secondary to R equi infection. *Rhodococcus equi* possesses some of the properties characteristic of corynebacteria, one of the types of microorganisms cultured from patients with Whipple's disease. One can speculate that organisms that require active histiocytic function for their degradation and removal may lead, in the host with defective cellular immunity, to disorders such as malacoplasia, Whipple's disease, or the Whipple-like disorders associated with MAI infection.

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**REFERENCES**