such a penetrating trauma has been well described in immunocompetent hosts, most notably in a 43-year-old man who developed septic arthritis due to MAC 35 years after a similar injury [5]. Our patient did not have disseminated disease or a history of trauma to the site of infection, thus clearly defining this infection as primary septic arthritis.

The presence of a vigorous local immune response with the formulation of granulomas despite the marked immunodeficiency has been noted previously [2]. It has been speculated that this response may represent recruitment of CD3$$^+$$, CD4$$^+$$, and Thy1$$^+$$ cells, which are relatively preserved in patients with advanced HIV infection [2]. An alternate explanation in our patient’s case is that the recently introduced antiretroviral regime had induced enough viral suppression to allow a degree of immune reconstitution. This hypothesis is supported by the increase in CD4 cell count and the decrease in the viral load seen in November 1996.

This case illustrates that in the growing spectrum of MAC disease in the AIDS patient, septic arthritis can present in the context of disseminated disease, as a de novo infection or after local injury with inoculation. Since a response to chemotherapy was documented in all reported cases of arthritis due to MAC [1–5], MAC should be considered early in the differential diagnosis of subacute arthritis in patients with AIDS even in the absence of systemic mycobacterial disease.

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**References**


**Microsporidium Species in Pulmonary Cavitary Lesions of AIDS Patients Infected with *Rhodococcus equi***

In the past few years, both *Rhodococcus equi* and Microsporidium species have been added to the growing list of AIDS-associated opportunistic organisms [1, 2]. Both organisms have been found to cause multi-organ infections [1, 2], but *R. equi* has been reported with greater frequency and has a greater tendency to disseminate than does *Microsporidium* species. Although *Microsporidium* species were initially believed to be a cause of AIDS-related diarrhea, they were subsequently shown to be pathogenic in the conjunctivae, liver, peritoneal cavity, and lungs.

Only a few cases of respiratory infection due to *Microsporidium* species have been documented, and in these cases microsporidia were found only at the epithelial level of conducting airways. Alveolar spaces as well as interstitium were found to be unaffected by *Microsporidium* species, and no cavitary lesions have thus far been reported in association with respiratory infections due to this organism [3, 4]. We describe herein two *R. equi*–infected AIDS patients with respiratory symptoms in whom *Microsporidium* species were the only agents identified in newly developed pulmonary cavitary lesions.

A routine chest roentgenogram was obtained in December 1994 for a 37-year-old HIV-infected male with a history of *Pneumocystis carinii* pneumonia and disseminated cryptococcosis; the results of this roentgenogram were negative. One month later he presented to the hospital with fever, cough, and bloody sputum; his CD4$$^+$$ cell count was 4/$$\mu$$L. A chest roentgenogram revealed a newly formed cavitary lesion in the upper part of the right lower lobe. Following unsuccessful diagnostic attempts with noninvasive procedures, bronchoscopy with bronchoalveolar lavage (BAL) and transbronchial biopsy (TBB) was performed. *Microsporidium* species were detected in both BAL and biopsy specimens.

In our case, *Microsporidium* species were first identified in hematoxylin-eosin stained specimens; many organisms (1–3 $$\mu$$m in diameter) were seen within macrophages and epithelial cells at the
level of bronchioles, alveolar ducts, and alveolar spaces, in the presence of mononuclear inflammatory infiltration. *Microsporidium* species were also detected by electron microscopy in a biopsy specimen (figure 1). In the following weeks, *R. equi* was isolated in blood agar cultures of both a bone marrow aspirate and sputum. Despite the administration of a three-drug antimicrobial regimen (rifampin, ciprofloxacin, and azithromycin were given on the basis of susceptibility test results) and of albendazole (400 mg b.i.d., as was recently suggested for microsporidiosis) [5], the lesion remained unchanged; the patient died 6 months later after progressive wasting occurred.

A 36-year-old man whose condition had been diagnosed as AIDS (he had disseminated cryptococcosis in August 1995) presented to the hospital in November 1995 with fever and a productive bloody cough. His CD4+ T lymphocyte count was 22/μL. A chest roentgenogram showed a single cavitary lesion in the perihilar region of the right lower lobe that was surrounded byparenchymal consolidation (this finding was confirmed by a CT scan); the lesion had not appeared 3 months earlier.

*R. equi* (5,000,000 cfu/mL) was isolated from sputum, and transient amelioration of the symptoms occurred after a three-drug antimicrobial regimen was administered. A repeated chest roentgenogram did not reveal any new changes and was followed by bronchoscopy with BAL and TBB. *R. equi* was again isolated (from a BAL specimen), and *Microsporidium* species were detected in a biopsy specimen by both light and electron microscopy. Albendazole therapy was started, and a mild decrease in the respiratory symptoms and partial clearance of the perilesional infiltration were noted; however, the cavitary lesion persisted and was unchanged. Ten months after it was first detected, the cavitary lesion was unchanged.

Pulmonary cavitary lesions represent an increasingly frequent diagnostic challenge in patients infected with HIV [6]. In the two extremely immunosuppressed AIDS patients described herein, *Microsporidium* species were the only organisms directly detected at the site of disease (e.g., in newly developed pulmonary cavities that were sampled by means of TBB). To our knowledge, no cases of cavitary pulmonary disease have thus far been associated with *Microsporidium* species [6], which are still mostly considered to be an intestinal pathogen [2, 5].

The pathological examination also revealed that our two cases were unique because of the presence of numerous microsporidia in the alveolar spaces. In prior descriptions of respiratory infections due to *Microsporidium* species, the organisms were found with progressively reduced frequency as airway caliber decreased, thus suggesting that the pathogenicity of *Microsporidium* species in the respiratory tract was limited to conducting airways [4].

On the contrary, our findings suggest that *Microsporidium* species may also play a pathogenic role at the alveolar level and that this organism might also be responsible for cavitations in the lung. However, the patients described herein also had evidence of disseminated *R. equi* infection (an organism known to cause pulmonary cavities), with culture of airway and bone marrow specimens yielding this organism.

According to the diagnostic criteria for *R. equi* cavitary pneumonitis, there would have been no doubt about the diagnosis if microsporidia were not seen at the disease site. On the basis of our current knowledge about the pathogenicity of *R. equi* and *Microsporidium* species, we hypothesize that the primary causative agent of the cavitary lesion was *R. equi* and that *Microsporidium* species gained access to the lesion only after the parenchymal architecture was altered. However, further clinical observations and investigations are needed to clarify whether *Microsporidium* species may display pathogenic properties in the lung or whether they are commensal organisms.

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