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**Rhodococcus equi**

**Infection after Alemtuzumab Therapy for T-cell Prolymphocytic Leukemia**

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**Rhodococcus equi**, mainly known from veterinary medicine as a pathogen in domestic animals, can also cause infections in immunocompromised humans, especially in those with defects in cellular immunity. Alemtuzumab, an anti-CD52 monoclonal antibody, causes lymphocytopenia by eliminating CD52-positive cells. We report a patient in whom *Rhodococcus equi* infection developed after alemtuzumab therapy.

*Rhodococcus equi* is a soil-borne, asporogenous, nonmotile, obligate aerobe; it is also a facultative, intracellular, gram-positive microorganism that can survive inside macrophages, the characteristic considered the basis for its pathogenicity (1). In foals and other domestic animals, it is an important respiratory and intestinal pathogen (2). Human infection with *R. equi* is rare but can occur in immunocompromised patients, especially those who have HIV infection and a CD4+ cell count <100 × 10^6/L (3). The clinical manifestations are diverse, although 80% of patients have some pulmonary involvement (3). In recent decades, an increased incidence of *R. equi* infections in humans has been reported. This increase may be due to the rising number of immunocompromised patients as a result of increasing numbers of organ transplantations and intensified antitumor chemotherapy. We describe a patient with T-prolymphocytic leukemia (T-PLL) in whom a febrile disease with lung abscess due to *R. equi* developed 10 weeks after the complete remission of leukemia was induced by chemotherapy combined with alemtuzumab.

**Case Report**

A 68-year-old man with T-PLL (leukocyte count 174.5 × 10^9/L, 96% lymphoid cells) was treated with chemotherapy consisting of cyclophosphamide, doxorubicin, vincristine, and prednisolone every 2 weeks (CHOP14), in combination with alemtuzumab 30 mg subcutaneously on days 1, 5, and 9 of each cycle. This combined therapy was well tolerated. Complete cytologic and immunohistochemical remission was confirmed by blood and bone marrow examination 2 weeks after the latest chemotherapy treatment. Ten weeks later, the patient experienced flu-like symptoms and had a fever of 38.9°C. One week earlier, the antimicrobial prophylaxis, which consisted of valacyclovir, 500 mg 2 times/day, and trimethoprim-sulfamethoxazole, 960 mg 3 times/week, had been stopped, although the alemtuzumab-induced lymphocytopenia was still present (leukocytes 7.2 × 10^9/L, 84% neutrophils, 0.6% lymphocytes). Outpatient evaluation showed 2 lung abscesses. From 3 consecutive blood cultures and from the bronchoalveolar lavage fluid, a gram-positive bacillus with mucoid growth was isolated and identified as *R. equi* (API Coryne, bioMérieux, Marcy l’Etoile, France). The isolated strain was resistant to β-lactam antimicrobial drugs and trimethoprim-sulfamethoxazole and susceptible to aminoglycosides, tetracyclines, fluoroquinolones, glycopeptides, erythromycin, and rifampin. Treatment with moxifloxacin and rifampin was begun. After 3 weeks of treatment, fever developed in the patient again. Blood cultures grew *R. equi*. The patient was admitted to the hospital for intravenous treatment with imipenem/cilastatin, 500 mg/500 mg 3 times/day, and vancomycin, 1.5 g once a day. A computed tomographic scan of the chest showed progression of the pulmonary abscesses and mediastinal lymphadenopathy. Clarithromycin, 500 mg 2 times/day, was added, and the vancomycin was increased to 2 g once a day, which resulted in clinical improvement. Purple, subcutaneous, oval lesions, 2–3 cm in diameter and not painful to palpation, were seen on the upper portion of both legs. Pathologic examination of these lesions after biopsy showed suspected localization of T-PLL. *R. equi* could not be demonstrated in these skin lesions by either pathologic or microbiologic examination. After 2 weeks of receiving intravenous antimicrobial drugs, the patient was discharged with oral rifampicin, 600 mg once a day; ciprofloxacin, 750 mg twice/day; and azithromycin, 500 mg once a day.

He was readmitted to our hospital 9 weeks later because he had become dyspneic and febrile. Evaluation showed pleural effusion on the right side. Progression of the T-PLL was also diagnosed. After 1 week’s incubation of the pleural fluid, mucoid nonpigmented colonies were growing, consisting of gram-positive coccoid rods, which were catalase positive. *Rhodococcus* infection was suspected and confirmed by 16S rDNA sequencing without further conventional identification. The isolate showed intermediate susceptibility to ciprofloxacin (MIC 0.75 mg/L), moxifloxacin (MIC 0.5 mg/L), and erythromycin (MIC 1.5 mg/L). Drainage of the pleural effusion was performed once a day. after 4 weeks, the patient showed signs of clinical improvement.

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In summary, longstanding alemtuzumab-induced lymphocytopenia is the most likely cause of the uncontrollable opportunistic *R. equi* infection in the described patient. This case illustrates the therapeutic challenges of this kind of infection in severely immunocompromised patients.

Dr Meeuse is completing a residency in internal medicine at the University Medical Center Groningen. He is also a PhD candidate in the field of palliative medicine, focusing on measurement and treatment of pain of malignant origin.

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