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## *Rhodococcus equi* systemic infection in an HIV-infected child

*Clin Microbiol Infect* 1998; 4: 353–354

*Rhodococcus equi* is a well-known pathogen in veterinary medicine. It is also a rare but emerging opportunistic pathogen in immunocompromised patients. Since the first description of a human infection in 1967 [1], about 50 cases have been reported. The incidence of human *R. equi* infection is increasing, largely owing to the human immunodeficiency virus (HIV) pandemic [2–5]. We present a case of *R. equi* bacteremia in an HIV-1-infected child, in whom the opportunistic infection revealed HIV-1 infection.

A 5-year-old girl was referred to St Mary Pediatric Hospital in Iasi, Romania, with fatigue, loss of appetite, weight loss, dry cough and fever (38–40°C) in August 1994. The fever had started 3 weeks previously and persisted despite ambulatory treatment with penicillin and ampicillin.

On examination she had micropolyadenopathy with painless, mobile, asymmetric nodes, tonsillar hypertrophy and hepatomegaly. Fine crackles were heard on both sides of her chest. She had tachycardia (120 beats/min) and a systolic murmur in all the auscultation points. The peripheral white blood cell count was $11.4 \times 10^9$/L, with 74% neutrophils. No evidence of pneumonia was observed on the chest radiograph.

A Gram-positive coccobacillus was isolated from two of three blood samples, after 4 and 7 days in culture. The colonies were small, irregular, round, smooth, semi-transparent, mucoid and salmon-pink. The bacterium was pleomorphic, ranging from distinctly coccoid to rod-shaped. It was an obligate aerobe, catalase positive, oxidase negative and urease positive. *R. equi* infection was confirmed by full identification at the Pasteur Institute in Paris (Dr M. Kiredjian).

The disk diffusion method showed that the two isolates were sensitive to penicillin, ampicillin, ceftriaxone (MIC 0.03 mg/L), ceftazidime (MIC 0.05 mg/L), cefotaxime (MIC 0.03 mg/L), erythromycin, gentamicin and co-trimoxazole, intermediately susceptible to rifampicin, and resistant to tetracycline.

Specific parenteral therapy was started with ceftriaxone, and gentamicin was added 4 days later. A clinical and biological response was observed after 24 h, fever started to abate on day 2 and ceased on day 5, and the C reactive protein level was normal by day 7. This treatment was maintained for 2 weeks and was followed by ampicillin therapy for 35 days. As *R. equi* infection occurs mainly in immunocompromised hosts, the patient was tested for HIV-1 infection; serotyping by subtype-specific enzyme immunoassay (SSEIA) showed she was infected by HIV-1 subtype F [6]. Flow cytometry showed a CD4/CD8 cell ratio of 0.65 (CD4 cell count: $380 \times 10^6$/L). She made a full recovery and did not relapse on treatment withdrawal.

This case has several interesting features:

(1) To our knowledge this is the first description of *R. equi* infection in an HIV-1-infected child. She had had no known contact with animals and no specific site of infection; in particular, there was no clinical or radiologic evidence of pulmonary involvement, in contrast to the majority of reported cases [1–3,5,7].
(2) *R. equi* infection was diagnosed prior to the discovery of HIV-1 infection, and was the first clinical manifestation of HIV-1 disease.
(3) The response to antibiotic therapy was favorable, after a short course (2 weeks) of ceftriaxone and gentamicin, followed by 35 days of ampicillin. This confirms a previous report showing the efficacy of ceftriaxone in the treatment of *R. equi* infection [7]. In contrast, there have been several reports of treatment failure in immunocompromised patients

despite prolonged therapy with drugs shown to be active in vitro [8]. We observed no recurrence of clinical symptoms on the discontinuation of therapy with ceftriaxone and gentamicin.

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Accepted 4 February 1998

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## Symptomatic occupational transmission of hepatitis C virus (HCV)

*Clin Microbiol Infect* 1998; 4: 354

Previous reports suggest that there is a low risk of occupational transmission of hepatitis C virus (HCV) among healthcare workers [1], but it has been shown to occur [2]. We present our experience of following up needlestick accidents involving biological fluids from patients who were HCV-antibody positive. Since 1990, 87 such episodes have been reported to the Preventive Medicine Unit of this hospital. These involved 14 physicians (16%), 46 nurses (53%) and 27 ancillary personnel (31%). Thirty-nine of these patients were also HIV-antibody positive, and a further one was both anti-HIV and HBsAg positive. Antibodies to HIV and HCV and HBsAg were detected with commercial tests (years 1990–93, Abbott; years 1994–96, Boehringer Mannheim).

Serum samples were taken from each staff member at 0 (100%), 3 (100%), 6 (94%) and 12 (77%) months after the episode. Two were already anti-HCV positive at the time of the accident. One nurse, involved in an accident with a patient positive only for anti-HCV, developed acute clinical hepatitis (ALT 2750 IU) 6 weeks later, with seroconversion by ELISA and Western blot (core+NS3, Murex Diagnostics). HCV RNA was also detected in both patient and nurse. The accident consisted of a double needlestick injury to the fourth finger of the right hand. The nurse was enrolled in an acute HCV interferon treatment protocol at a reference hospital and the infection was cleared. There was no seroconversion to either HBV or HIV in any of the staff members involved.

Our study confirms a low rate of occupational transmission of HCV (1.15%, 95% CI: 0.06–7.13%) but we report another documented symptomatic case and, in the absence of specific preventative measures, we reiterate the need for universal precautions.

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Accepted 4 February 1998

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