zoonotic; cats are primary reservoirs, and humans and dogs are accidental hosts (1). However, B. clarridgeiae was recently detected in rodent fleas in China (9) and B. koehlerae was isolated from feral pigs from the southeastern United States (10), suggesting that these pathogens also have multiple reservoir species.

Clarifying whether Bartonella infections in raccoons are caused by spillover from feral cats needs further study. Additional samples from raccoons and other species in urbanized and undeveloped habitats with different host species composition (e.g., cat-free environment) might enable further Bartonella spp. characterization in wildlife. We suspect urban raccoons and feral cats play a major role in Bartonella spp. transmission.

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Rifampin-Resistant Mycobacterium bovis BCG–Induced Disease in HIV-Infected Infant, Vietnam

To the Editor: Guidelines for the diagnosis and management of Mycobacterium bovis BCG disease in HIV-infected children are lacking. BCG strains are intrinsically resistant to pyrazinamide and in some cases have low-level resistance to isoniazid (6). However, data on acquired drug resistance in M. bovis BCG are limited. We describe a case of BCG disease caused by a rifampin-resistant strain of M. bovis BCG in an HIV-infected infant in Vietnam.

The daughter of a known HIV-infected woman, who did not fully adhere to antiretroviral therapy (ART) during pregnancy, received the M. bovis intradermal BCG (Pasteur strain) vaccine at birth. HIV infection was diagnosed in the infant by PCR when she was 8 weeks of age. At 9 months of age, she was admitted to the Pediatric Infectious Diseases Department of the Pham Ngoc Thach Hospital (Ho Chi Minh City, Vietnam) because of a voluminous ipsilateral axillary mass at the site of the vaccination, fever, weight loss, and hepatosplenomegaly. The percentage of CD4+ T cells was 27% (1,620 cells/mm3). Regional BCG disease was clinically diagnosed without microbiological investigation, and a broad antimycobacterial therapy targeting M. tuberculosis complex species was started with 5 mg/kg isoniazid, 10 mg/kg rifampin, and 25 mg/kg pyrazinamide. After 6 weeks of antimycobacterial therapy, ART was initiated with lamivudine, stavudine, and abacavir. After 6 months of antimycobacterial treatment, the infant was hospitalized again for recurrent inflammation and fistulization of the axillary lymph nodes associated with fever.
Fluid from the axillary mass was collected by fine-needle aspiration for bacteriologic investigations. Direct microscopic examination showed acid-fast bacilli, and the mycobacterial infection was confirmed by culture. By using conventional biochemical methods, the mycobacterial isolate was assigned to the M. bovis species. Pyrazinamide was discontinued, and antimycobacterial therapy was continued for 4 supplementary months with rifampin (15 mg/kg) and isoniazid (10 mg/kg). After 2 months, drug susceptibility testing results confirmed pyrazinamide intrinsic resistance and isoniazid and ethambutol susceptibility and showed rifampin resistance. The late inflammatory reaction after introduction of ART was evocative of immune reconstitution inflammatory syndrome. Nevertheless, drug resistance may have contributed. Despite the rifampin resistance, the patient showed clinical improvement, and the rifampin/isoniazid treatment was continued for 2 more months. The child’s BCG disease was cured on completion of 10 months of antituberculous treatment.

Retrospective molecular investigations using the GenoType MTBC Kit (Hain Lifescience, Nehren, Germany) enabled identification of the isolate stored at −80°C as M. bovis BCG strain. A mutation in the rpoB gene (codon 531, Ser531Tyr) associated with rifampin resistance and isoniazid and ethambutol susceptibility and showed rifampin resistance. The late inflammatory reaction after introduction of ART was evocative of immune reconstitution inflammatory syndrome. Nevertheless, drug resistance may have contributed. Despite the rifampin resistance, the patient showed clinical improvement, and the rifampin/isoniazid treatment was continued for 2 more months. The child’s BCG disease was cured on completion of 10 months of antituberculous treatment.

Because disseminated BCG disease in HIV-infected children presents a high risk for illness and/or death, these patients should receive optimal tuberculosis treatment (2) based on 4-drug (rifampin, isoniazid, ethambutol, and pyrazinamide) regimen doses for at least 9 months until M. tuberculosis is ruled out (3). Untreated local BCG immune reconstitution inflammatory syndrome may not necessarily progress to dissemination; therefore, treatment would not appear necessary (8).

Some studies suggest that the survival of HIV-infected children with BCG disease could be attributed to early initiation of ART in combination with other treatments (1,3). In the South Africa case, the child died, and the authors suggested that this outcome was related to the severity of the clinical features, the severe HIV-related immune suppression, and the absence of ART (4). In the case in Vietnam, despite the emergence of drug resistance, the early initiation of ART in a child with a localized disease, the persistent efficacy of isoniazid, and the spontaneous fibulization of the abscess probably contributed to the good outcome for the infant.

In conclusion, this case highlights the challenges in management of BCG disease in children. It also emphasizes the possible risk for emergence of acquired drug resistance in M. bovis BCG strains, complicating the medical management of such cases.

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Bulleidia extructa Periprosthetic Hip Joint Infection, United States

To the Editor: Bulleidia extructa is an obligately anaerobic, nonmotile, non–spore-forming gram-positive bacillus first described in 2000 by Downes et al. (1), after having isolated a bacterium from the oral cavity of persons with periodontitis and dentoalveolar abscesses that did not correspond to any known species. After phenotypic and genetic characterization, the investigators proposed a new genus, Bulleidia, and the species B. extructa. Since then, additional reports have associated the organism with oral infections, specifically periodontal disease (2–5). While B. extructa’s association with human periodontal disease is well documented, the bacterium has so far not been implicated in other pathogenic processes. We report here a case of a total hip arthroplasty infection caused by B. extructa in an immunocompetent patient.

In November 2010, an 82-year-old man with a non-cemented right total hip arthroplasty that was performed 26 years previously was evaluated for right hip pain. He had been in his usual state of health without any complaints until a month earlier, when he lost his footing and hyperabducted his right hip. He had been in an immuno-competent patient. After 3 weeks of intravenous ceftriaxone treatment was prescribed, and the patient was instructed to revisit a dentist for a full dental examination. Before seeking treatment for this episode, he reported that he was seeing a dentist on a regular basis and denied any recent dental surgery or infections.

The patient underwent total hip arthroplasty resection. Intraoperatively, purulence was noted upon entering the hip joint. Histopathologic examination of removed tissue revealed acute inflammation. Five hip tissue specimens were obtained for culture; 3 specimens yielded B. extructa. Six weeks of intravenous ceftriaxone treatment was prescribed, and the patient was instructed to revisit a dentist for a full dental examination. Before seeking treatment for this episode, he reported that he was seeing a dentist on a regular basis and denied any recent dental surgery or infections.

The patient was seen in a follow-up visit 2 months after reimplantation surgery; at that time, he reported minimal pain and had begun to bear weight on the affected side. There was no evidence for infection recurrence. Periprosthetic joint infections are a major complication after joint replacement. The number of procedures for total hip and knee replacements has

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