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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Jusun Hwang and Nicole L. Gottdenker

Author affiliation: University of Georgia, Athens, Georgia, USA

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Address for correspondence: Nicole Gottdenker, Department of Veterinary Pathology, College of Veterinary Medicine, University of Georgia, Athens, GA 30602, USA; email: gottdenk@uga.edu

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/mm^3). 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 was detected by using the GenoType MTBDRplus Kit (Hain Lifescience) and partial sequencing of the *rpoB* gene (*5*,*7*). No mutation in the *katG* and *inhA* genes, frequently associated with isoniazid resistance, was detected.

To our knowledge, this case is the second report of rifampin-resistant *M. bovis* BCG disease in HIV-infected children. The first report involved a child in South Africa who was vaccinated with the Danish BCG strain (*4*); this strain shows low-level resistance to isoniazid and therefore has a high risk of evolving to multidrug resistance in instances of suboptimal isoniazid levels. The *M. bovis* BCG Pasteur strain (American Type Culture Collection 35734) used for vaccination in Vietnam is isoniazid and rifampin susceptible and pyrazinamide resistant (*9*). Despite appropriate antimycobacterial treatment, the relatively low doses of isoniazid (5 mg/kg), poor adherence, or inadequate absorption of drugs because of HIV-related gastrointestinal disease may have resulted in subtherapeutic in vivo drug concentrations and thus in selection of a drug-resistant *M. bovis* BCG strain. This case should alert clinicians of the possible emergence of rifampin resistant *M. bovis* BCG strains.

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 fistulization 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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Duc Nguyen Hong, Mai Nguyet Thu Huyen, Nguyen Thi Ngoc Lan, Nguyen Huy Duong, Vi Vi Nguyen Ngo, Duong Tran Ngoc, Khanh Truong Huu, Tuyen Nguyen, Viet Do Chau, Oliver Marcy, Philippe Van de Perre, Anne-Laure Bañuls, and Sylvain Godreuil

Author affiliations: Pham Ngoc Thach Hospital, Ho Chi Minh City, Vietnam (D. Nguyen Hong, M.N.T. Huyen, N.T.N. Lan, N.H. Duong, V.V. Nguyen Ngo, D.T. Ngoc, T Nguyen); Pediatric Hospital Nhi Dong 1, Ho Chi Minh City (K.T. Huu); Pediatric Hospital Nhi Dong 2, Ho Chi Minh City (V.D. Chau); Institut Pasteur, Phnom Penh, Cambodia (O. Marcy); INSERM U 1058, Montpellier, France (P. Van de Perre, S. Godreuil); Université Montpellier 1, Montpellier (P. Van de Perre, S. Godreuil); CHU Montpellier, Montpellier (P. Van de Perre, S. Godreuil); and MIVEGEC, Universités Montpellier 1 et 2, Montpellier (A.-L. Bañuls)

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Address for correspondence: Sylvain Godreuil, Centre Hospitalo–Universitaire, Hôpital Arnaude Villeneuve—Laboratoire de Bactériologie, 371 Avenue du Doyen Gaston Giraud, Montpellier cedex 5 34295, France; email: godreuil@yahoo.fr

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 hip joints, involuntarily performing a split, while washing a boat cover with a power washer. Since then, he reported right hip pain that somewhat limited his mobility.

Physical examination revealed an antalgic gait, mild swelling of the right lower extremity, and impaired hip mobility related to pain on the right side, specifically with extension, flexion, abduction, and adduction. Results of the patient's blood work were notable for normocytic anemia (hemoglobin 10.6 g/dL), thrombocytosis (459 × 10^9/L), elevated erythrocyte sedimentation rate (101 mm/h), and elevated C-reactive protein (88.7 mg/L). Leukocyte count was within normal limits (9.6 × 10^9 cells/L). An ultrasound examination of the right hip joint showed extensive synovitis and a large, 4.3 × 5.0 × 5.1–cm vascular mass extending anteriorly from the joint space. Aspiration of the joint space yielded 1 mL of blood-stained fluid with 111,595 cells/μL (95% neutrophils, 5% monocytes/macrophages). Anaerobic bacterial culture grew a gram-positive bacillus identified as *B. extructa* by partial 16S rRNA sequencing. DNA was prepared for PCR amplification by using PrepMan Ultra (Applied Biosystems, Foster City, CA, USA) and amplified and bidirectionally sequenced by using primers 5′-TGGAGAGTTTGATCCTGGCTCAG-3′ and 5′-TACCGCGGCTGCTGGCAC-3′. The generated 484-bp sequence differed by 2 bp from 483 bp of available sequence from *B. extructa* GenBank accession no. AF220064. The isolate was susceptible to penicillin, clindamycin, and metronidazole by using E-test.

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