at symptom onset, but results for patient serum samples cultured under the same conditions as the homogenized parasites remained negative. Significant titers of IgG against *B. quintana* and *B. henselae* or IgG seroconversion in paired serum samples were observed for all patients except the grandfather (Table).

Oral clarithromycin and doxycycline were administered to the children and adults, respectively, for 10 days. The apartment was repeatedly treated with insecticide, and the hole in the roof was repaired, leading to eradication of the mites. The few dead and dry mites that were available for additional parasitologic analysis were mounted in Swan mounting medium (information about the medium is available from the authors), but no characteristics allowing differentiation between species of the genus *Dermanyssus* were recognized during examination by light microscopy. Failed attempts were made to trap pigeons that had lived on the roof of the apartment or in the same city; however, samples from trapped synanthropic pigeons from the north (n = 20) and central (n = 33) part of the country were negative for *Bartonella* spp. by the culture and amplification methods described above. Recurrent fever reported by adult patients resolved in 3 months, and all patients made a full clinical recovery. Laboratory findings for the patients were followed for 6 months after symptom onset (Table).

The fact that the suspected vector was a hematophagous mite (*Dermanyssus* sp.), a parasite of synanthropic pigeons and a suspected vector of other bacterial pathogens (*8*,*9*), and that the 16S rRNA *Bartonella* spp. gene was detected in mites (*Steatonyssus* sp. from the superfamily *Dermanyssoidea*) (*10*) remains a challenge for additional study. Pigeons probably played the role of accidental host in this outbreak, but the source of the infection remains unclear.

Acknowledgments

We thank V. Rupeš for parasitologic analysis, A. Valkoun for serologic analysis of specific antibodies to *Rickettsia* and *Coxiella* spp., D. Kafková for collection of patient data, and E. Kodytková for manuscript review.

Oto Melter, Mardjan Arvand, Jiří Votýpka, and Dagmar Hulínská

Author affiliations: Charles University, Prague, Czech Republic (O. Melter); Zentrum für Gesundheitsschutz, Dillenburg, Germany (M. Arvand); and National Institute of Public Health, Prague (J. Votýpka, D. Hulínská)

DOI: http://dx.doi.org/10.3201/eid1801.110186

References

1. Drancourt M, Mainardi JL, Brouqui P, Vandenesch F, Carta A, Lehnert F, et al. *Bartonella (Rochalimaea) quintana* endocarditis in three homeless men. N Engl J Med. 1995;332:419–23. doi:10.1056/NEJM199502163320702
2. Comer JA, Paddock CD, Childs JE. Urban zoonoses caused by *Bartonella, Coxiella, Ehrlichia, and Rickettsia* species. Vector Borne Zoonotic Dis. 2001;1:91–118. doi:10.1089/153036601316977714
3. Breitschwerdt EB, Hegarty BC, Hancock SI. Sequential evaluation of dogs naturally infected with *Ehrlichia canis, Ehrlichia chaffeensis, Ehrlichia equi, Ehrlichia ewingii*, or *Bartonella vinsonii.* J Clin Microbiol. 1998;36:2645–51.
4. Anderson B, Sims K, Regnery R, Robinson L, Schmidt MJ, Goral S, et al. Detection of *Rochalimaea henselae* DNA in specimens from cat scratch disease patients by PCR. J Clin Microbiol. 1994;32:942–8.
5. Arvand M, Schäd SG. Isolation of *Bartonella henselae* DNA from the peripheral blood of a patient with cat scratch disease up to 4 months after the cat scratch injury. J Clin Microbiol. 2006;44:2288–90. doi:10.1128/JCM.00239-06
6. Massung RF, Slater KG. Comparison of PCR assays for detection of the agent of human granulocytic ehrlichiosis, *Anaplasma phagocytophilum.* J Clin Microbiol. 2003;41:717–22. doi:10.1128/JCM.41.2.717-722.2003
7. Hulinska D, Votypka J, Plch J, Vlcek E, Valešová M, Bojar M, et al. Molecular and microscopical evidence of *Ehrlichia* spp. and *Borrelia burgdorferi* sensu lato in patients, animals and ticks in the Czech Republic. New Microbiol. 2002;25:437–48.
8. Valiente Moro C, De Luna CJ, Tod A, Guy JH, Sparagano OAE, Zenner L. The poultry red mite (*Dermanyssus gallinae*): a potential vector of pathogenic agents. Exp Appl Acarol. 2009;48:93–104. doi:10.1007/s10493-009-9248-0
9. Valiente Moro C, Thioulouse J, Chauve C, Normand P, Zenner L. Bacterial taxa associated with the hematophagous mite *Dermanyssus gallinae* detected by 16S rRNA PCR amplification and TTGE fingerprinting. Res Microbiol. 2009;160:63–70. doi:10.1016/j.resmic.2008.10.006
10. Reeves WK, Dowling APG, Dasch GA. Rickettsial agents from parasitic *Dermanyssoidea* (Acari: Mesostigmata). Exp Appl Acarol. 2006;38:181–8. doi:10.1007/s10493-006-0007-1

Address for correspondence: Oto Melter, Department of Medical Microbiology, 2nd Medical Faculty, Charles University, V Úvalu 84, 150 06 Prague 5—Motol, Prague, Czech Republic; email: oto.melter@lfmotol.cuni.cz

Urban Transmission of Human African Trypanosomiasis, Gabon

To the Editor: We describe a confirmed case of human African trypanosomiasis (HAT) in an expatriate returning to France from Gabon after a probable tsetse fly bite in the urban setting of Libreville. This case indicates a possible urban transmission of HAT in Gabon and stresses the need for entomologic studies in Libreville.

HAT is endemic to sub-Saharan Africa. *Trypanosoma brucei rhodesiense* (eastern Africa) and *T.b. gambiense* (western Africa) parasites are transmitted to humans by tsetse flies of the *Glossina morsitans* group (*T.b. rhodesiense*) and of the *G. palpalis* group (*T.b. gambiense*), which are found only in Africa. *T.b. gambiense* represents >90% of all reported cases

of HAT worldwide. HAT has always been a travel-associated disease. It is a rare cause of fever, cutaneous lesions, and neurologic signs in travelers returning from disease-endemic areas and involves *T.b. rhodesiense* in 70% of the cases, resulting mostly from an exposure during safari in game parks (*1,2*).

A 58-year-old previously healthy Portuguese man who worked in Gabon for 13 years for a French company was admitted to the tropical and infectious diseases ward because of a 2-month history of intermittent fever, fatigue, and a 10-kg weight loss. The patient recalled a painful unidentified insect bite on his right thigh 2 months before in his garden in Libreville (Lalala quarter). A 8-cm, indurated, erythematous, and painful plaque (chancre) progressively developed (Figure) in the following weeks after the assumed insect bite. When admitted to the hospital, the patient had a temperature of 39°C, anorexia, insomnia, pruritus of the left arm, and paresthesia of the hands and feet. Two additional large annular erythematous macules, centrally pale (trypanids), were found on his back (Figure). A subclavicular 0.5-cm lymph node was observed. There was no hepatosplenomegaly.

His laboratory results showed moderate anemia (hemoglobin 11.8 g/dL) and thrombopenia (134,000 platelets/mm^3) and elevated levels of C-reactive protein (30.6 mg/L) and gammaglobulins (23.9 g/L). A thick-blood smear showed no malaria parasites but a few trypomastigotes of *Trypanosoma* spp. PCR of blood identified *T.b. gambiense.* A cerebrospinal fluid sample showed moderate elevation of total proteins (0.43 g/L) and albumin (291 mg/L), 11 leukocytes, and no IgM elevation. Direct examination and PCR showed no trypanosome in the cerebrospinal fluid. Specific antibodies were found in the blood by indirect immunofluorescence (titer 200). Biopsies of 2 skin lesions (thigh, back) showed a lymphoplasmocytic vasculitis consistent with cutaneous locations of HAT; no parasite was observed in situ. The patient was treated successfully with a 7-day course of pentamidine. The case was reported to World Health Organization Control of Neglected Tropical Diseases Department.

A total of 328 HAT cases were reported to the World Health Organization in Gabon during 2000–2009; most infections were acquired in the mangrove swamp Atlantic coast focus in Noya (Estuaire Province) and some in the focus of Bendje (Ogooué-Maritime Province) (*3*). Four of 6 cases of *T.b. gambiense* imported to Europe during 2005–2009 were in expatriates with a travel history to Gabon (*1*). In the 4 case-patients infected in Gabon, an exposure in rural forest areas was assessed (*4–6*; D. Malvy, pers. comm.). In the fifth case reported here, the tsetse bite likely occurred in the urban setting of Libreville.

The patient did not report occupational exposure to tsetse bites outside Libreville during the previous year. He occasionally went in Pointe Denis during weekends but did not remember having been bitten by a tsetse fly. Although the patient did not identify the insect in his garden, the chronology of his clinical history and the presence of a typical chancre at the place of the insect bite that occurred before symptoms provide strong arguments in favor of this hypothesis. The bite occurred during the morning hours, in the patient's home garden in the Lalala area of Libreville (0.357568N, 9.475365E) near the Ogombié River. This area is located 125 km and 75 km from the Bendje and Noya HAT foci, respectively.

Two studies provided evidence for urban transmission of HAT in Kinshasa (Democratic Republic of Congo) and in Bonon (Côte d'Ivoire) (*7,8*). Concurrently, some tsetse species, such as *G. palpalis*, adapt to high human densities and are found in the largest urban centers of western Africa (*9*). Entomologic studies in Libreville should prompt further investigation into a possible urban transmission of HAT in Gabon, as we suspect in the case reported.

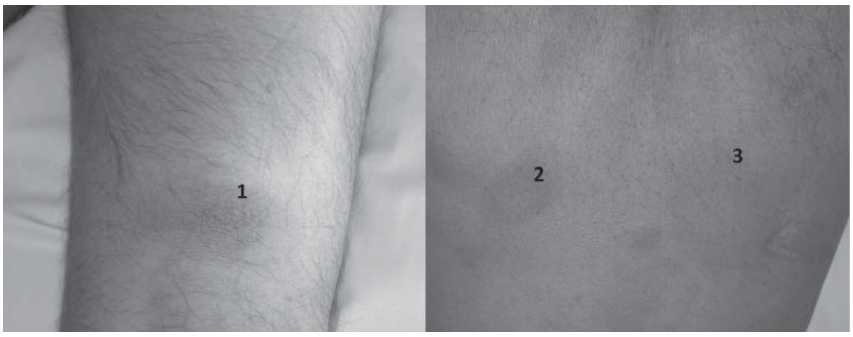

Figure. Thigh chancre (1) and back trypanids (2 and 3) in a patient with human African trypanosomiasis infection, Gabon. A color version of this figure is available online (wwwnc.cdc.gov/EID/article/18/1/11-1384-F1.htm).

Fabrice Simon, Marie Mura, Frédéric Pagès, Gabriel Morand, Philippe Truc, Francis Louis, and Philippe Gautret

Author affiliations: Hôpital d'Instruction des Armées Laveran, Marseille, France (F. Simon, M. Mura, G. Morand, P. Gautret); Institut de Médecine Tropicale du Service de Santé des Armées, Marseille (F. Pagès); Institut de Recherche pour le Développement, Montpellier, France (P. Truc); and Organisation de Coordination pour la Lutte contre les Endémies en Afrique Centrale, Yaoundé, Cameroon (F. Louis)

DOI: http://dx.doi.org/10.3201/eid1801.111384

References

1. Gautret P, Clerinx J, Caumes E, Simon F, Jensenius M, Loutan L, et al. Imported human African trypanosomiasis in Europe, 2005–2009. Euro Surveill. 2009; 14:pii:19327.
2. Migchelsen SJ, Büscher P, Hoepelman AI, Schallig HD, Adams ER. Human African trypanosomiasis: a review of non-endemic cases in the past 20 years. Int J Infect Dis. 2011 Jun 15; [Epub ahead of print].
3. Simarro PP, Cecchi G, Paone M, Franco JR, Diarra A, Ruiz JA, et al. The Atlas of human African trypanosomiasis: a contribution to global mapping of neglected tropical diseases. Int J Health Geogr. 2010;9:9–57. doi:10.1186/1476-072X-9-57
4. Bisoffi Z, Beltrame A, Monteiro G, Arzese A, Marocco S, Rorato G, et al. African trypanosomiasis gambiense, Italy. Emerg Infect Dis. 2005;11:1745–7.
5. Ezzedine K, Darie H, Le Bras M, Malvy D. Skin features accompanying imported human African trypanosomiasis: hemolymphatic *Trypanosoma gambiense* infection among two French expatriates with dermatologic manifestations. J Travel Med. 2007;14:192–6.. doi:10.1111/j.1708-8305.2007.00114.x
6. Hope-Rapp E, Moussa Coulibaly O, Klement E, Danis M, Bricaire F, Caumes E. Double trypanosomal chancre revealing West African trypanosomiasis in a Frenchman living in Gabon. Ann Dermatol Venereol. 2009;136:341–5. Epub 2009 Feb 26. doi:10.1016/j.annder.2008.09.023
7. Simo G, Mansinsa Diabakana P, Kande Betu Ku Mesu V, Manzambi EZ, Ollivier G, Asonganyi T, et al. Human African trypanosomiasis transmission, Kinshasa, Democratic Republic of Congo. Emerg Infect Dis. 2006;12:1968–70. doi:10.3201/eid1212.060516
8. Courtin F, Dupont S, Zeze DG, Jamonneau V, Sané B, Coulibaly B, et al. Human African trypanosomiasis: urban transmission in the focus of Bonon (Côte d'Ivoire). Trop Med Int Health. 2005;10:340–6. doi:10.1111/j.1365-3156.2005.01398.x
9. Courtin F, Sidibé I, Rouamba J, Jamonneau V, Gouro A, Solano P. Population growth and global warming: impacts on tsetse and trypanosomoses in West Africa. Parasite. 2009;16:3–10.

Address for correspondence: Fabrice Simon, Department of Infectious Diseases and Tropical Medicine, Laveran Military Teaching Hospital, Bd Alphonse Laveran, BP 60149, 13384 Marseille CEDEX 13, France; email: simon-f@wanadoo.fr

Rickettsia felis Infections, New Zealand

To the Editor: Members of the genus *Rickettsia* have garnered much attention worldwide in recent years with the emergence of newly recognized rickettsioses. In New Zealand, only *Rickettsia typhi* and *R. felis,* belonging to the typhus and spotted fever groups, respectively, have so far been found (*1*). *R. typhi*, primarily transmitted by the oriental rat flea (*Xenopsylla cheopis*), has a worldwide distribution and causes murine typhus in humans (*2*). At the end of 2009, a total of 47 cases of murine typhus had been recorded in New Zealand. In contrast, although the cat flea (*Ctenocephalides felis*) can carry *R. felis* in New Zealand (*3*), no human infections have been reported. However, because *R. felis* shares a similar clinical profile to murine typhus, infection can be mistaken for a suspected case of *R. typhi* (*4*).

Clinical suspicion of rickettsial infection is widely confirmed by serologic tests with the indirect immunofluorescence assay (IFA) being the standard test. However, antibodies against *R. felis* in human sera are known to cross-react with *R. typhi* in IFA (*5*). Western blot (WB) and cross-adsorption assays, in combination with IFA, can differentiate between several rickettsioses (*5*,*6*). We report on the trial in New Zealand of WB and cross-adsorption assays for differentiating retrospectively between past *R. typhi* and *R. felis* infections and evidence of *R. felis* infection in persons living in the country.

Serum samples were obtained from 24 volunteers from the Institute of Environmental Science and Research Limited, Porirua, New Zealand. Samples were tested using *R. typhi* IFA slides (Australian Rickettsial Reference Laboratory [ARRL], Geelong, Victoria, Australia). After incubation (37°C for 30 min), slides were washed 3 times, incubated with fluorescein-conjugated antihuman IgG, IgM, and IgA (ARRL), and washed again before examination. All samples were then tested by using an IgG IFA kit (Focus Diagnostics, Cypress, CA, USA) against typhus group (TG) *R. typhi* and spotted fever group (SFG) *R. rickettsii*.

TG-positive and SFG-negative serum samples may represent *R. typhi* infections, and SFG-positive and TG-negative serum samples may represent *R. felis* infections. Because *R. typhi* can cross-react with SFG rickettsiae (*7*), and *R. felis* with *R. typhi* (*5*), results that are TG positive and SFG positive may be caused by either rickettsiae. Positive reactivity may also represent overseas-acquired rickettsioses. Thus, WB and cross-adsorption assays using *R. typhi* (Wilmington) and *R. felis* (URRWXCal2) antigens (Unité des Rickettsies, Marseilles, France) were used to confirm any *R. typhi* or *R. felis* infections (*6*).

Antigens (2 mg/mL) were solubilized (100°C for 10 min) in 2× Laemmli buffer (*6*) and subjected to electrophoresis (20 μg/well; 20 mA, 2.5 h) through polyacrylamide gels (12.5% resolving; 4% stacking) (BioRad, Hercules, CA, USA). Resolved antigens were electroblotted (100 V for 1 h) onto 0.45-μm polyvinylidene difluoride membranes, which were blocked by using 5% milk–Tris-buffered saline with 0.1% Tween 20. Each antigen lane was divided into 2 strips before incubation (room temperature for 1 h) with serum (diluted 1:200). After three 10-min washes with Tris-buffered saline with 0.1% Tween 20, strips were incubated (room temperature, 1 h) with horseradish peroxidase–conjugated antihuman IgG (1:150,000; SouthernBiotech, Birmingham, AL, USA) and washed again. Enhanced chemiluminescent detection of bound horseradish peroxidase (ECL Plus; GE Healthcare, Buckinghamshire,