3. Malfait P, Lopalco PL, Salmaso S, Germinario C, Salamina G, Quarto M, et al. An outbreak of hepatitis A in Puglia, Italy, 1996. Euro Surveill. 1996;1:33–5.

4. Lopalco PL, Malfait P, Salmaso S, Germinario C, Quarto M, Barbuti S, et al. A persisting outbreak of hepatitis A in Puglia, Italy, 1996: epidemiological follow-up. Euro Surveill. 1997;2:31–2.

5. Lopalco PL, Malfait P, Menniti-Ippolito F, Prato R, Germinario C, Chironna M, et al. Determinants of acquiring hepatitis A virus disease in a large Italian region in endemic and epidemic periods. J Viral Hepat. 2005;12:315–21.

6. Andre FE. Universal mass vaccination against hepatitis A. Curr Top Microbiol Immunol. 2006;304:95–114.

7. Wasley A, Samandari T, Bell BP. Incidence of hepatitis A in the United States in the era of vaccination. JAMA. 2005;294: 194–201.

8. Dagan R, Leventhal A, Anis E, Slater P, Ashur Y, Shouval D. Incidence of hepatitis A in Israel following universal immunization of toddlers. JAMA. 2005;294: 202–10.

9. Dominguez A, Salleras L, Carmona G, Batalla J. Effectiveness of a mass hepatitis A vaccination program in preadolescents. Vaccine. 2003;21:698–701.

Address for correspondence: Pietro Luigi Lopalco, Universita' di Bari, Policlinico, Piazza Giulio Cesare, 70124 Bari, Italy; email: p.lopalco@igiene.uniba.it

Human *Rickettsia sibirica mongolitimonae* Infection, Spain

To the Editor: *Rickettsia sibirica mongolitimonae* has been recently reported as a subspecies of *R. sibirica* (*1*). The first evidence of *R. sibirica mongolitimonae* pathogenicity in humans was documented in France in 1996 (*2*). Since then, 11 more cases in France, Algeria, South Africa, Greece, and Portugal have been reported (*3*–*6*). Because the main clinical manifestations include lymphangitis, the acronym LAR (lymphangitis-associated rickettsiosis) has been proposed (*3*). We report a case from Spain that confirms the broad distribution of this agent in southern Europe.

A 41-year-old man was admitted on June 19, 2007, to the Hospital de Cruces (Baracaldo, Spain) with fever (39°C), malaise for a week, sweating, lumbar and knee pain, disseminated myalgias, and headache. He reported that 20 days before admission he had removed an engorged tick from his right leg while working as a topographer in the Balmaseda Mountains, 30 km from Bilbao. He had also removed several ticks from his body 4 days before the onset of symptoms. Physical examination did not demonstrate relevant findings. There was no inoculation eschar at the tick-bite sites. Rash, lymphadenopathies, and lymphangitis were not observed.

Chest radiograph did not show consolidation or other abnormality. Initial laboratory examination, on June 21, 2007, showed a leukocyte count $5.2 \times 10^3/\mu L$, hemoglobin 14.1 g/dL, platelet count 190,000/μL, erythrocyte sedimentation speed 9 mm/h, urea 38 mg/dL, creatinine 0.9 mg/dL, aspartate aminotransferase 229 IU/L, alanine aminotransferase 170 IU/L, alkaline phosphatase 158 IU/L, gamma-glutamyl-transpeptidase 111 IU/L, total bilirubin 1.3 mg/dL, and C-reactive protein 4.3 mg/dL. Because the patient had been bitten by a tick, acute-phase serum and EDTA-treated blood samples were sent to the Special Pathogens Laboratory (Área de Enfermedades Infecciosas – Hospital San Pedro from La Rioja), where a presumptive diagnosis of rickettsiosis was made. On June 22, 2007, treatment with doxycycline was begun (100 mg/day for 12 days), and his condition rapidly improved.

The early-phase serum yielded low immunoglobulin (Ig) G titer (<64) against *Rickettsia conorii* and *Anaplasma phagocytophilum* antigens, and results of ELISA and Western blotting for Lyme borreliosis were negative. A convalescent-phase serum sample collected 7 weeks later did not contain IgG antibodies against spotted fever group *Rickettsia* species when *R. conorii* antigen was used.

DNA was extracted from the early whole-blood specimen by using QIAamp DNA Blood minikit (QIAGEN, Hilden, Germany) according to the manufacturer's instructions. This DNA extract was used as template in nested PCR assays targeting the spotted fever group rickettsial *ompB* (420 bp) and *gltA* (337 bp) genes (*7*). Quality control included both positive (with *R. conorii* Malish #7 grown in Vero cells) and negative controls that were extracted and PCR amplified in parallel with the specimens. Negative controls consisted of sterile water instead of template DNA. Amplification products of the expected size were obtained. The sequences of these amplicons allowed the identification of *R. sibirica mongolitimonae* with 99.5% and 100% similarity for *ompB* and *gltA*, respectively (GenBank accession nos. DQ097083 and DQ097081).

To our knowledge, *Rickettsia* species have never been detected in ticks or human specimens in Spain. The host ticks of this rickettsia are likely *Hyalomma* species, which are more prevalent in southern Spain. In our region in northern Spain, *Hyalomma marginatum* represented 8% of ticks that fed on humans during 2001–2005, although an increase in this number was recorded last year (data not shown).

In our patient, *Rickettsia*'s pathogenic role was demonstrated by PCR, a technique that has previously enabled us to identify other arthropod-borne *Rickettsia* species (*8,9*). This case suggests that *R. sibirica mongolitimonae* infection should be considered in the differential diagnosis of rickettsiosis and tick-bite febrile patients in Spain and confirms the distribution of this rickettsia in southern Europe. According to the literature (*3*), some patients in whom *R. sibirica mongoli-*

timonae infection is diagnosed have >1 eschar, which raises the suspicion that some cases of Mediterranean spotted fever with multiple eschars reported in Spain could be caused by this rickettsial species. More studies about the vectors of this bacteria are needed because studies of *Hyalomma* and *Rhipicephalus* ticks (the suspected hosts) conducted in our area have not demonstrated the presence of this *Rickettsia* species.

Koldo Aguirrebengoa,*
Aránzazu Portillo,†
Sonia Santibáñez,†
Juan J. Marín,†
Miguel Montejo,*
and José A. Oteo†

*Hospital de Cruces, Baracaldo, Spain; and †Hospital San Pedro–Centro de Investigación Biomédica de La Rioja (CIBIR), Logroño, Spain

References

1. Fournier PE, Zhu Y, Yu X, Raoult D. Proposal to create subspecies of *Rickettsia sibirica* and an emended description of *Rickettsia sibirica.* Ann N Y Acad Sci. 2006;1078:597–606.
2. Raoult D, Brouqui P, Roux V. A new spotted-fever-group rickettsiosis. Lancet. 1996;348:412.
3. Fournier PE, Gouriet F, Brouqui P, Lucht F, Raout D. Lymphangitis-associated rickettsiosis, a new rickettsiosis caused by *Rickettsia sibirica mongolotimonae*: seven new cases and review of the literature. Clin Infect Dis. 2005;40:1435–44.
4. Pretorius AM, Birtles RJ. *Rickettsia mongolotimonae* infection in South Africa. Emerg Infect Dis. 2004;10:125–6.
5. Psaroulaki A, Germanakis A, Gikas A, Scoulica E, Tselentis Y. Simultaneous detection of "*Rickettsia mongolotimonae*" in a patient and in a tick in Greece. J Clin Microbiol. 2005;43:3558–9.
6. de Sousa R, Barata C, Vitorino L, Santos-Silva M, Garrapato C, Torgal J, et al. *Rickettsia sibirica* isolation from a patient and detection in ticks, Portugal. Emerg Infect Dis. 2006;12:1103–8.
7. Choi YJ, Jang WJ, Kim JH, Ryu JS, Lee SH, Park KH, et al. Spotted fever group and typhus group rickettsioses in humans, South Korea. Emerg Infect Dis. 2005;11:237–44.
8. Oteo JA, Portillo A, Santibáñez S, Pérez-Martínez L, Blanco JR, Jiménez S, et al. Prevalence of spotted fever group *Rickettsia* species detected in ticks in La Rioja, Spain. Ann N Y Acad Sci. 2006;1078:320–3.
9. Jado I, Oteo JA, Aldámiz M, Gil H, Escudero R, Ibarra V, et al. *Rickettsia monacensis* and human disease, Spain. Emerg Infect Dis. 2007;13:1405–7.

Address for correspondence: José A. Oteo, Área de Enfermedades Infecciosas, Hospital San Pedro, C/ Piqueras, 98-7ª NE, 26006 – Logroño (La Rioja), Spain; email: jaoteo@riojasalud.es

Lymphangitis in a Portuguese Patient Infected with *Rickettsia sibirica*

To the Editor: We report a case of *Rickettsia sibirica* mongolitimoniae strain infection associated with lymphangitis (*1*). A 44-year-old man was admitted to São Bernardo Hospital in Setubal, Portugal, on August 21, 2006. Twelve days previously while on vacation at Troia Peninsula, he noted malaise, insomnia, and dry buccal mucosa. The next day he observed a small erythematous pruritic lesion on the lower right forearm that 2 days later developed into an eschar. He also had fever and sought medical care. After treatment with topical bacitracin, floxacillin, and acetaminophen for 2 days, fever (38.7°C) continued with lymphangitis extending from the right wrist to the elbow. The medication was changed to nimesulide. Three days later a rash developed on the trunk and arms, and lymphangitis extended to the axilla. Fever and chills continued, leading to hospital admission. No history of tick exposure was reported. Physical examination showed blood pressure 128/73 mm Hg, pulse 96/min, and a rubbery, nontender right supraclavicular lymph node ≈1 cm in diameter. Several 5- to 10-mm maculopapular erythematous lesions were observed on the patient's palms. He had inflammation on the right forearm suggestive of lymphangitis and an eschar with surrounding edema and erythema on the dorsal lower right forearm (Figure). Admission evaluation showed platelets 117,000/µL, total bilirubin 0.42 mg/dL, albumin 3.42 g/dL, creatinine 1.1 mg/dL, alanine aminotransferase 244 U/L, aspartate aminotransferase 54 U/L, alkaline phosphatase 1061 U/L, creatine phosphokinase 87 U/dL, lactate dehydrogenase 784 U/L, C-reactive protein 7.1 mg/dL, radiographic pulmonary diffuse reticular pattern, arterial pO_2 68 mm Hg, O_2 saturation 94%, pCO_2 22 mm Hg, and arterial blood pH 7.35. The differential diagnoses included rickettsiosis, pneumonia, and cellulitis. Treatment with vancomycin, ceftriaxone, and 100 mg of doxycycline twice a day was begun. On the day after hospitalization, a heparinized blood sample and 2 skin biopsy samples were collected. Vancomycin and ceftriaxone were discontinued at 48 hours when rickettsial infection was confirmed by PCR on skin biopsy; 48 hours later, the patient was afebrile.

Immunofluorescence assay for antibodies that used *R. sibirica* mongolitimonae strain as antigen demonstrated seroconversion with no antibodies in the serum sample collected on August 21 and immunoglobulin G (IgG) and IgM antibodies at a titer of 256 in serum collected on August 30. DNA was extracted from 1 skin biopsy sample by using a DNeasy Tissue Kit (QIAGEN, Hilden, Germany). The products of nested PCR showed 100% similarity with *gltA* (353/353) and *ompA* (350/350) nucleotide sequences of *R. sibirica* mongolitimonae strain (GenBank accession nos. DQ423368.1 and DQ423367.1) (*1*).

Cutaneous biopsy indicated epidermal and dermal necrosis with extensive lymphocyte- and macro-