leaflet associated with the vegetation was destroyed. The vegetation culture, pericardial fluid culture, tissue culture from the resected mitral valve, and 3 blood cultures yielded no bacterial growth.

The patient required ventilator support for 7 days. Follow-up computed tomography on day 8 showed a stable appearance of cerebral infarcts. Coumadin was started to prevent thrombus development at the prosthetic valve. He went home after completing a 4-week treatment course of ceftriaxone. At that time, there was still noticeable left-sided weakness of the extremities. He could only communicate by using eye movements with no verbal response.

During the 20th century influenza pandemics, secondary bacterial pneumonia was a notable cause of death. The current pandemic (H1N1) 2009 outbreak is evolving rapidly, and it is unknown if pandemic (H1N1) 2009 may lead to an increase in rare complications of pneumococcal infection, such as endocarditis. Thus, Austrian syndrome should be considered in any patient with pandemic (H1N1) 2009 complicated by pneumococcal infection and a new heart murmur.

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to the Editor: Tick-borne rickettsioses are zoonoses caused by spotted fever group (SFG) Rickettsia spp. (1), which have been reported as a frequent cause of fever in international travelers (2). In Egypt, Mediterranean spotted fever caused by Rickettsia conorii transmitted by the brown dog tick, Rhipicephalus sanguineus, is known to be present, although cases are rarely documented. Moreover, an emerging pathogen, R. aeschlimannii, has been detected in Hyalomma dromedarii ticks, collected from camels, and in H. impeltatum and H. marginatum rufipes, collected from cows (3). We report a case of Rickettsia sibirica mongolitimonae infection in a French traveler who returned from Egypt.

In September 2009, a previously healthy 52-year-old man living in France was admitted to the infectious diseases unit of a hospital in Nantes, France, with a 10-day history of fever, asthenia, headache, and arthromyalgia. Three days earlier, he had returned from a 2-week trip to Egypt. He had fever (38°C), painful axillary lymphadenopathies, and an inoculation eschar surrounded by an inflammatory halo on the left scapular area (online Appendix Figure, www.cdc.gov/EID/content/16/9/1495-appF.htm), but he did not have a rash. During his travel, he had been unsuccessfully treated for headache, arthromyalgia, and diarrhea by amoxicillin-clavulanate (3 g/d), nonsteroidal antiinflammatory drugs, and gentamicin cream on the eschar for 3 d. No tick bite was reported by the patient. We suspected an SFG rickettsiosis. The patient received 200 mg doxycycline in a single dose and rapidly improved.

The immunofluorescence assay for antibodies reactive against SFG antigens showed increased levels of immunoglobulin M (titer 16) and G (titer 128). Results of Western blot with cross-adsorption assays supported the hypothesis that the infection was caused by R. sibirica mongolitimonae (1). To identify the involved rickettsiae, PCR amplifications and sequencing gltA, ompA, and ompB fragment genes of Rickettsia spp. and multispacer typing (MST), based on the sequence of variable intergenic spacers, were performed by using DNA samples obtained from an eschar biopsy and a lesion swab (4,5). A negative control (sterile water and DNA from a sterile biopsy specimen) and a positive control (DNA from R. montanensis) were included in each test. Amplicon sequencing confirmed the presence of R. sibirica mongolitimonae DNA in patient samples. The sequence homology to R. sibirica mongolitimonae DNA was ompA, 99.4%; gltA, 99.7%; and ompB, 100% (GenBank accession nos. DQ097082, DQ097081, and AF123715, respectively). The MST sequences were 100% homologous to the genotype of R. sibirica mongolitimonae MST type U (idem HA-91). We injected shell vial cultures with eschar biopsy specimens (4). Fifteen days later, positive Gimenez staining and immunofluorescence confirmed

Rickettsia sibirica mongolitimonae in Traveler from Egypt

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the presence of Rickettsia sp. in cell culture, and *R. sibirica mongolitimonae* was identified by PCR and sequencing as described above (online Appendix Figure).

*R. sibirica mongolitimonae* was first isolated in Beijing in 1991 from *H. asiaticum* ticks (formerly named *R. sibirica* HA-91), and the first human infection was reported in 1996 (4). Since that time, *R. sibirica mongolitimonae* infections have been diagnosed in 15 additional patients: 12 from Europe (France, Portugal, Greece, the People’s Republic of China, Senegal, and in *Rh. pusillus* ticks in Portugal (6–8). Although *Hyalomma* spp. ticks seem to be associated with *R. sibirica mongolitimonae*, more experimental data are needed to determine the tick vectors and reservoirs of this rickettsia.

Clinicians in Egypt and those who may see patients returning from this country should be aware that several species of rickettsiae are found in this region. Thus, they should consider a range of SFG rickettsial diseases in the differential diagnosis of patients with febrile illnesses.

**Letters**

**Learning Objectives**

Upon completion of this issue, readers should be able to:

1. Describe the clinical features associated with *Rickettsia sibirica mongolitimonae* infection.
2. Explain the differential diagnosis of patients with rickettsial infections.
3. Discuss the importance of tickborne rickettsiosis in global health.

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**Increase in Neisseria meningitidis**

**Serogroup W135, Niger, 2010**

**To the Editor:** Meningococcal epidemics in the African meningitis belt are generally caused by *Neisseria meningitidis* serogroup A strains, but they also can be caused by serogroup W135 or X strains. The largest reported outbreak caused by serogroup W135 occurred in Burkina Faso in 2002 with ≈13,000 suspected cases (1). Sporadic cases of meningitis caused by serogroup W135 have, however, been