



Figure. Computed tomography images of the brain of an adult patient with pandemic (H1N1) 2009 virus infection and neurologic signs. A noncontrast study showed hypodense lesions in both occipital lobes (A) and in both upper parietal lobes (B).

These included the lack of CSF albuminocytologic dissociation, the fact that the clinical signs occurred during the outbreak of pandemic (H1N1) 2009 virus infection rather than after it, and the fact that antibodies were not found in gangliosides. CSF albuminocytologic dissociation and serum ganglioside antibodies may be found in 85%–90% of Guillain-Barré syndrome patients (2).

Alternatively, the patient might have had central nervous system complication from pandemic (H1N1) 2009 virus infection. Acute disseminated encephalomyelitis is a condition that might occur within 30 days after an infectious process (3). It can lead to quadriplegia and diffuse white matter lesions. The clinical feature that makes acute disseminated encephalomyelitis less likely in this patient was the CSF findings in the reference range. In summary, however, we believe that pandemic (H1N1) 2009 virus infection can cause neurologic complications affecting both the peripheral and central nervous systems in adult patients.

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Rickettsia felis,
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To the Editor: A spay–neuter (sterilization) program for feral cats from Basseterre, the capital of the Caribbean Island St. Kitts, found that most (45/58; 66%) cats had antibodies to spotted fever group rickettsiae (SFGR). The antibodies were detected with *Rickettsia rickettsii* antigen in a standard microimmunofluorescence assay (1). Titers for 13 (20%) cats were ≥ 320 .

Most SFGR are transmitted by ticks, but because of their grooming habits, cats seldom have many ticks (2), and we did not find any ticks on the cats we saw through the program. We did, however, commonly find cat fleas, *Ctenocephalides felis*, which are the main vector of *R. felis*, a recently described member of the SFGR. *R. felis* seems to be apathogenic in cats (3) but is the agent of flea-borne spotted fever in humans (4). Although *R. felis* has been reported from North and South America, Europe, Africa, the Middle-East, and Oceania (4), its presence in the Caribbean islands has not been established. To provide this information we tested DNA extracted with the QIAamp DNA Mini-Kit (QIAGEN, Valencia, CA, USA) from *C. felis* fleas preserved in 70% ethanol.

Of 57 (19%) *C. felis* fleas from St. Kitts, 11 were positive for *R. felis* DNA when tested by PCR using primers targeting SFGR *ompA* (5) or Taq-Man assay using primers targeting *gltA* and a probe specific for the organism (6,7). For a negative control we used distilled water; for a positive control we used DNA from *R. montanensis* cultures or recombinant control plasmids constructed by amplifying target fragments from *R. typhi* strain Wilmington and *R. felis* strain LSU (7). The sequences of the *ompA* and *gltA* amplicons obtained had 100% nucleotide sequence similarity with homologous fragments of the type reference isolate *R. felis* URRxCal2. We used the Na-

tional Center for Biotechnology Information basic local alignment sequence tool, BLAST (www.ncbi.nlm.nih.gov/blast/Blast.cgi).

To determine whether *R. felis* occurs on another Caribbean island, we tested 32 *C. felis* fleas from Dominica and found 1 (3%) to be positive by PCR when primers targeting *ompA* were used. The sequence obtained was also identical to that of *R. felis* URRxCal2.

Our study provides further evidence that cats can be sentinels for the presence of rickettsiae (1). However, although rickettsemia can develop in cats experimentally infected with *R. felis* (3), no compelling evidence shows that cats help maintain the organism or transmit it to humans (8,9). Rather, it appears that *C. felis* fleas, which are also commonly found on dogs and to a lesser extent other mammals, are the major reservoir hosts and vectors of infection, although the exact mechanisms are unknown (10). Our study also expands the known distribution of *R. felis* and should alert healthcare workers who see residents of or vacationers from the Caribbean islands of the possibility of flea-borne spotted fever in their patients.

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Rickettsia africae, Western Africa

To the Editor: *Rickettsia africae*, the causative agent of African tick-bite fever, is transmitted by *Amblyomma hebraeum* and *A. variegatum* ticks (1,2). These ticks are common in western, central, and southern Africa. Adults rarely feed on humans, although nymphs attach more frequently and larvae are sometimes serious pests (abundant and aggressive) (3).

African tick-bite fever is a neglected disease that has been mainly detected in tourists who were bitten by a tick while traveling in disease-endemic areas (2). A recent worldwide report showed rickettsial infection incidence to be 5.6% in a group of travelers in whom acute febrile infection developed after they returned from sub-Saharan Africa. African tick-bite fever is the second most frequently identified cause for systemic febrile illness among travelers, following malaria (4). Seroprevalence for spotted fever group rickettsiae is high in the Sahel regions of Africa (5), although there may be different emergent and classic rickettsioses in Africa.

R. africae has been detected by PCR in many African countries, including Niger, Mali, Burundi, and Sudan (6), and in most countries of equatorial and southern Africa (Figure). Most strains and cases have been found in South Africa (2). *R. africae* and African tick-bite fever have not previously been reported in Senegal, and few positive human serum samples have been documented in western Africa. *A. variegatum*, the main vector of *R. africae*, was introduced by cattle into Guadeloupe, West Indies, from Senegal in the early 1800s. Spotted fever caused by *R. africae* has become endemic there in the past 30 years (7). In addition to *R. africae*, *A. variegatum* ticks may transmit other human and animal pathogens, including Crimean-Congo hemorrhagic fever virus, Dugbe virus, Thogoto virus,