Rift Valley Fever in Kedougou, Southeastern Senegal, 2012

To the Editor: Rift Valley fever (RVF) is an acute, febrile, viral disease caused by Rift Valley fever virus (RVFV), a phlebovirus of the family Bunyaviridae that is endemic to sub-Saharan Africa. RVF mortality and abortion rates among young domesticated ruminants and pregnant females are high.

In humans, clinical manifestations range from mild to severe syndromes, which can include neurologic, hemorrhagic, and hepatic features and retinitis, and which sometimes result in death (1). Diagnosis of RVF is challenging for clinicians because clinical manifestations are not specific (2). Heavy rainfall and flooding create conditions for emergence of RVF vectors (Aedes and Culex spp. mosquitoes), and dispersion of this disease into new areas is linked to migration of infected livestock, wildlife, or mosquitoes.

Since 1987, when the Diama dam was built, RVF outbreaks in Mauritania have been reported regularly (3). In Kedougou, southeastern Senegal, RVFV was isolated 4 times from Ae. dalzieli mosquitoes and once from a person with a mild case of RVF (4). We report results of a field investigation and laboratory findings for a human case of RVF detected by surveillance of acute febrile illnesses in Kedougou.

On October 16, 2012, a 27-year-old man (school teacher) who lived and worked in Baya village in the Kedougou region of Senegal (12°27′50″N, 12°28′6″W) visited the nearest health posts in Ibel and Thiokoye villages during October 2012 were also tested during surveillance of acute febrile illnesses.

All 334 samples were negative for RVFV RNA and IgM and IgG against RVFV except for samples from 3 patients, including the case-patient, which were positive for RVFV-specific IgG and malaria parasites. The 2 other patients were a 32-year-old tradesman and a 20-year-old housewife sampled during surveillance of acute febrile illnesses in Kedougou and Bandafassi, which is 30 km from Baya (online Technical Appendix Figure, wwwnc.cdc.gov/EID/article/20/3/13-1174-Techapp1.pdf). No RVFV RNA was detected...
from 519 mosquito pools sampled in the Kedougou region during October 2012, although these pools included 7 species previously found associated with RVFV and which represented 26.6% of the pools.

The patient reported no travel outside Kedougou in the 2-year period before his illness. Because no evidence of recent RVFV circulation among humans and mosquitoes was found, we believe that the patient was infected by contact with an animal imported from Mauritania. This hypothesis is based on reports by farmers from neighboring villages (Baya, Ibel, Thiokoye, and Dondol) of the presence of ruminants imported from Mauritania in the market in Thiokoye village and of deaths and abortions among sheep and goats in their villages during October–November 2012. However, no animals were sampled during the investigation.

There is an abundance of competent vectors for RVFV in Kedougou (4). In addition, there are massive human migrations resulting from gold mining and regular importation of animals from RVF-endemic regions of western Africa. Thus, an integrated human and animal surveillance system should be implemented or reinforced to avoid large-scale RVF outbreaks in Kedougou.

Acknowledgments

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References


Figure. Phylogenetic tree of a 581-bp sequence of the nonstructural protein gene on the small RNA segment of Rift Valley fever viruses. Boldface indicates strain isolated in this study. Bootstrap values are indicated along branches. Scale bar indicates nucleotide substitutions per site.


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Concomitant Multidrug-Resistant Pulmonary Tuberculosis and Susceptible Tuberculous Meningitis

To the Editor: In 2012, a 34-year-old HIV-seronegative man was hospitalized after several months of cough, fever, night sweats, 10-kg weight loss, and, in the past month, severe headache. The patient was born in Romania and had lived in France for 2 years. He had a history of pulmonary tuberculosis (TB) for which treatment was started in Romania in 2006 and 2008, but he did not complete treatment. The treatment he received in Romania was unknown.

At hospital admission, the patient had a fever of 39°C, stiff neck, and swollen cervical and axillary lymph nodes. A chest radiograph showed multiple cavities and nodular opacities in both superior lobes. Sputum auramine staining indicated that acid-fast bacilli was positive, which supported the diagnosis of pulmonary TB. Examination of cerebrospinal fluid (CSF) revealed hypoglycorrhachia (0.95 mmol/L, concentration ratio CSF/blood: 0.2 [reference range 0.5–0.75]), hyperproteinorrhachia (1.3 g/L [reference range 0.2–0.4 g/L]), erythrocyte count 2,000 μL (reference value <10 μL), and leukocyte count 150 μL (reference value <10 μL). Auramine staining showed no acid-fast bacilli in CSF. Standard antituberculous therapy with rifampin (RIF), isoniazid (INH), pyrazinamide, and ethambutol was started.

Genomic amplification–based assay (Xpert MTB/RIF; Cepheid, Maurens-Scopont, France), performed on sputum, confirmed the presence of the Mycobacterium tuberculosis genome and detected resistance to RIF (Table). The line probe assay Genotype MTBDRplus (Hain Lifescience, Bandol, France) performed on sputum showed a positive signal for all wild-type sequences and for rpoB (S531L associated with RIF resistance) and katG (S315T associated with INH resistance) mutations, suggesting the presence of mixed susceptible and resistant M. tuberculosis. Second-line treatment was started: moxifloxacin, amikacin, ethionamide, para-aminosalicylic acid, cycloserine and linezolid. The presence of mixed M. tuberculosis organisms in lungs was confirmed with culture methods and by phenotypic drug susceptibility testing (DST) that showed 1% resistant mutant to RIF and INH (proportion method) (1). The isolate was considered resistant to RIF and INH and was thus categorized as multidrug resistant (MDR). DST also showed that the sputum isolate had an elevated proportion of ofloxacin-resistant mutants (2 mg/L, 0.02% resistant mutants). Although no mutation in gyrA or gyrB was detected on colonies grown in the absence of fluoroquinolone, a gyrB N538D mutation was identified on colonies grown on ofloxacin medium. On the basis of the DST results, the treatment was changed to ethambutol, pyrazinamide, amikacin, moxifloxacin, para-aminosalicylic acid, and linezolid. CSF culture was eventually positive for M. tuberculosis on Lowenstein-Jensen medium after 30 days. DST performed on the CSF isolate showed a drug-sensitive phenotype; thus, RIF was reintroduced in addition to the other antimicrobial drugs. After 3 months, although the patient had improved, he left the hospital against medical advice without providing follow-up contact information.

Mycobacterial interspersed repetitive unit–variable number tandem repeat (MIRU-VNTR) analysis was conducted on culture of the sputum and CSF sample and on colonies grown on INH, RIF, and ofloxacin–containing medium, as described (2) (Table). This analysis showed different genotypes for the lung MDR and the CSF-susceptible isolates. None of the MIRU-VNTR patterns

Table. Localization, drug-susceptibility results, and MIRU-VNTR genotypes of Mycobacterium tuberculosis strains recovered from a man with both pulmonary tuberculosis and tuberculous meningitis, France, 2012*

<table>
<thead>
<tr>
<th>Tissue</th>
<th>MIRU-VNTR genotype 1</th>
<th>MIRU-VNTR genotype 2</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>22221322223422515333622</td>
<td>22424312223422514333622</td>
</tr>
<tr>
<td>Lung</td>
<td>Isoniazid and rifampin resistant, fluoroquinolone</td>
<td>Pan-susceptible except to fluoroquinolones, no</td>
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<tr>
<td></td>
<td>susceptible, rpoB S531L, katG S315T, no mutations in</td>
<td>mutations in rpoB, katG, and gyrA; gyrB N538D</td>
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<tr>
<td></td>
<td>gyrA and gyrB</td>
<td>Pan-susceptible, no mutations in rpoB, katG, gyrA, and</td>
</tr>
<tr>
<td>Cerebrospinal fluid</td>
<td></td>
<td>gyrB</td>
</tr>
</tbody>
</table>

*MIRU-VNTR, mycobacterial interspersed repetitive unit–variable number tandem repeat.
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Technical Appendix

Technical Appendix Figure. Geographic distribution of Rift Valley fever cases, southeastern Senegal, 2012.