

gust 2003 that compared HIV prevalence in 426 patients with Buruli ulcer and 613 controls in southern Benin. HIV prevalence among patients with Buruli ulcer was higher (2.6%, 11/426) than among controls (0.3%, 2/613) (odds ratio 8.1) (8). However, none of these reported HIV-positive patients with Buruli ulcer were treated with rifampin/streptomycin and antiretroviral therapy (8).

A study of 224 patients with Buruli ulcer in Benin that evaluated the WHO-recommended regimen of 8 weeks of treatment with rifampin/streptomycin showed promising results (9). Chemotherapy alone was successful in achieving a cure rate of 47% of patients and was effective against ulcers <5 cm in diameter (9). However, HIV testing was not performed in this study. In Spain, an HIV-positive patient with aggressive, multifocal Buruli ulcer and osteomyelitis was cured by surgery, broad-spectrum antimicrobial drugs (not rifampin/streptomycin), and antiretroviral drugs (10). Relapse was not reported in this study at 6-months follow-up.

For control of Buruli ulcer in HIV-positive patients, patients should be treated with rifampin/streptomycin and antiretroviral therapy to stimulate their immunity. Our report emphasizes the urgent need to evaluate treatment of HIV-positive patients infected with Buruli ulcer with rifampin/streptomycin and antiretroviral drugs.

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**Kapay Kibadi,
Robert Colebunders,
Jean-Jacques
Muyembe-Tamfum,
Wayne M. Meyers,
and Françoise Portaels**

Author affiliations: Institute of Tropical Medicine, Antwerp, Belgium (K. Kibadi, R. Colebunders, F. Portaels); Institut National de Recherche Biomédicale, Kinshasa, Democratic Republic of Congo (K. Kibadi, J.-J. Muyembe-Tamfum); University of Kinshasa, Kinshasa (K. Kibadi, J.-J. Muyembe-Tamfum); University of Antwerp, Antwerp (R. Colebunders); and Armed Forces Institute of Pathology, Washington, DC, USA (W.M. Meyers)

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Address for correspondence: Françoise Portaels, Mycobacteriology Unit, Institute of Tropical Medicine, Nationalestraat 155, B-2000 Antwerp, Belgium; email: portaels@itg.be

Hantavirus Pulmonary Syndrome, French Guiana

To the Editor: Hantaviruses are rodent-borne negative-sense RNA viruses belonging to the *Bunyaviridae* family, genus *Hantavirus*. Since the first report of a hantavirus in 1993 in the United States (1), different viruses belonging to this genus have been reported in the Americas (2–5). These New World viruses are responsible for a disease called hantavirus pulmonary syndrome (HPS), a respiratory illness caused by the inhalation of dust contaminated by rodent feces or urine containing the virus (6–8).

Until recently, no information was available concerning the presence of hantaviruses in French Guiana, a French overseas department (administrative unit) in South America. Nevertheless, the description of atypical pneumonia cases not related to any known etiologic agent and the identification of hantavirus reservoirs in neighboring countries led us to con-

duct a serologic study in a selected population of patients with compatible symptoms. The prevalence of immunoglobulin (Ig) G antibodies to hantavirus in this population was 1.42% (9). Subsequently, we systematically screened patients who had suggestive pathologies for hantavirus serology, which led us to the characterization of a divergent hantavirus.

On August 4, 2008, a 38-year-old man sought medical attention at the emergency department of Cayenne Hospital. He had persistent symptoms of fever ($\geq 38.5^{\circ}\text{C}$), myalgia, diarrhea with melena, cough for 8 days, recurrent vomiting for 4 days, and dyspnea for 2 days. At consultation, tachypnea (respiratory rate 28/min) and oxygen desaturation (SaO_2 83%) were observed. Chest radiograph showed bilateral diffuse interstitial infiltrates causing respiratory distress; mechanical ventilation was required. The patient was admitted to the intensive care unit for treatment of acute

respiratory distress syndrome. Results of laboratory investigations performed when the patient was admitted showed thrombocytopenia (50,000 cells/mm³), leucocytosis (22,500 cells/mm³) associated with a high neutrophil count (20,300 cells/mm³), moderate hepatonephritis (alanine aminotransferase 17 IU/L, aspartate aminotransferase 31 IU/L, gamma-glutamyl transferase 44 IU/L; alkaline phosphatase 44 IU/L; creatinine 192 $\mu\text{mol}/\text{L}$ and urea 9.3 mmol/L); and an elevated C-reactive protein concentration (>192 mg/L). Laboratory tests for infectious agents ruled out malaria, dengue, leptospirosis, Chagas disease, Q fever, cytomegalovirus, and HIV, and blood cultures were negative for bacterial growth. The patient remained under respiratory assistance for 25 days in the intensive care unit and was discharged from hospital 47 days after admission with a complete clinical recovery.

With no etiologic agent identified, 2 factors led to the suspicion of han-

tavirus infection: clinical symptoms compatible with HPS and the patient's exposure to potential reservoirs. Indeed, a month before the onset of symptoms, he had moved to a rural municipality located near agricultural lands and forest.

Retrospective serologic investigations were performed with the 3 available serum samples obtained during the hospitalization. These samples were tested by IgM capture with inactivated Sin Nombre virus antigens and by indirect ELISA with recombinant antigens to detect IgG antibodies to Sin Nombre virus (10). IgM to Sin Nombre virus were present in the samples collected 8 and 9 days, respectively, after onset of the disease, confirming hantavirus infection. Furthermore, IgG to Sin Nombre virus were only detected in the convalescent-phase serum samples obtained on day 41 of the disease. These serologic results suggested a recent infection with hantavirus.

Molecular investigations were performed to characterize and identify the virus. Viral RNA was extracted from the 2 acute serum samples. Reverse transcription-PCR was performed with consensus primers targeting the S segment of the hantavirus genome as described in Johnson et al. (4). Amplification products of the expected size (434 bp of the nucleoprotein N-encoding region) were obtained from both samples. Cloning and sequencing of these products allowed obtaining a consensus sequence, which was deposited with GenBank (GQ179973). Database searches using BLAST (www.ncbi.nlm.nih.gov/blast) demonstrated that this sequence, although novel, is most similar to Rio Mamore hantavirus strain OM-556 (GenBank accession no. U52136), showing 83% nucleotide identity (393 bp analyzed, excluding the primers). In addition, comparison with representative hantavirus sequences from New World isolates showed that the amplified fragment exhibited from

Table. Comparison of nucleotide and deduced amino acid sequences between Maripa virus and representative New World hantaviruses*

Maripa virus	% Nucleotide sequence identity	% Amino acid sequence identity
RIOM_B	83.0	96.9
RIOM_P	81.9	96.2
ANAJ	81.2	96.2
LAN	81.2	96.2
RIME	77.9	94.7
PARN	78.4	93.9
ARAQ	80.2	93.1
AND	78.6	93.1
ORN	78.4	93.1
MCL	76.8	93.1
CHO	76.1	93.1
PRG	76.6	93.1
LEC	80.2	92.4
BMJ	79.4	92.4
CAD	77.1	92.4
ARAC	77.6	91.6
C_Plata	77.6	90.8
SN	73.5	90.1

*RIOM_B, Rio Mamore virus strain OM-556 (U52136); RIOM_P, Rio Mamore virus strain HTN-007 (AF133254); ANAJ, Anajatuba virus isolate Of58 (DQ451829); LAN, Laguna Negra virus strain 510B (AF005727); RIME, Rio Mearim virus isolate Hs85 (DQ451828); PARN, Paranao virus (EF576661); ARAQ, Araraquara virus (AF307325); AND, Andes virus strain AH-1 (AF324902); ORN, Oran virus strain 22996, (AF482715); MCL, Maciel virus strain 13796 (AF482716); CHO, Choclo virus (DQ 285046); PRGm Pergamino virus strain 14403 (AF482717); LEC, Lechiguana virus strain 22819 (AF482714); BMJ, Bermejo virus strain Oc22531 (AF482713); CAD, Cano Delgado virus isolate VHV-574 (AF000140); ARAC, Araucaria virus strain HPR/04-102 (AY740633); CAS, Castelo dos Sonhos virus (AF307324); C_Plata, Central Plata virus strain 714LC (EU564715); SN, Sin Nombre virus strain NM H10 (L25784).

73.5% to 81.9% nucleotide sequence identity and from 90.1% to 96.9% amino acid sequence identity (Table). This level of sequence divergence, as well as the geographic specificity of this hantavirus in French Guiana led us to provisionally name it Maripa virus.

Results of a serologic survey to identify cases of respiratory disease with no evident etiology led us to identify an HPS case-patient in French Guiana who had been infected with a new divergent hantavirus strain. Human hantavirus epidemics are associated with fluctuations of rodent populations caused by climatic, ecologic and environmental changes or with changes in human activities associated with nature or agriculture. Therefore, in this region where 90% of the land is tropical rain forest but in which there is increasing economic development, continuous surveillance for the virus in the human population would be beneficial. Surveys of potential reservoirs may help reduce the risk of viral emergence.

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**Séverine Matheus,
Félix Djossou, David Moua,
Anne Marie Bourbigot,
Didier Hommel,
Vincent Lacoste,
Philippe Dussart,
and Anne Lavergne**

Author affiliations: Institut Pasteur de la Guyane, Cayenne, French Guiana (S. Matheus, D. Moua, V. Lacoste, P. Dussart, A. Lavergne); and Centre Hospitalier André Rosemon, Cayenne (F. Djossou, A.M. Bourbigot, D. Hommel)

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Address for correspondence: Séverine Matheus, Laboratoire de virologie, Centre National de Référence des Arboviruses, Institut Pasteur de la Guyane, 23 avenue Pasteur, BP 6010 – 97306 Cayenne CEDEX, French Guiana; email: smatheus@pasteur-cayenne.fr

Fatal Human Case of West Nile Virus Disease, Mexico, 2009

To the Editor: West Nile virus (WNV; family *Flaviviridae*, genus *Flavivirus*) was first recognized in the Western Hemisphere in 1999 during an outbreak of human, equine, and avian encephalitis in New York (1). The virus has since spread across the United States and Canada, where it has caused ≈30,000 human infections and ≈1,000 deaths. Serologic evidence has demonstrated that WNV is present throughout Mexico, Central America, South America, and the Caribbean region (2–8). However, WNV illness in humans and vertebrate animals in these regions has been only sparsely reported. For instance, 7 human cases of WNV infection have occurred in Mexico (excluding the case described here), 3 of which were severe. All patients survived. To our knowledge, no fatal human cases of WNV infection have occurred in Central America, South America, or the Caribbean region.

We describe a fatal case of WNV infection in a human in Central America. The patient, a man 40 years of age, lived in Monterrey, Nuevo León State, in northern Mexico. He had not traveled outside of the metropolitan area in the 6 months before illness onset. On June 11, 2009, influenza-like signs and symptoms (i.e., fever, malaise, fatigue, arthralgia, headache, and dizziness) developed in the patient. On June 26, the signs and symptoms had not resolved, and the man was admitted to University Hospital “Dr. José E. Gonzalez” at the Universidad Autónoma de Nuevo León (UANL). At the time of admission, cerebrospinal fluid (CSF) was collected, and laboratory analysis indicated a markedly elevated leukocyte count (182 cells/mm³; reference range 0–5 cells/mm³) and slightly elevated protein and glucose levels.