

kidneys, although brain and lung cultures were negative.

Another group of guinea pigs that had also been injected with *Leptospira* was humanely killed as soon as symptoms appeared. Necropsy showed primary lung injury. Lungs were pale with hemorrhages widely spread over the surface. Lesions were similar to those observed in one of the patients. Neither jaundice nor renal damage was found. *Leptospira* was isolated from kidneys, lungs, and brain. Jaundice has been reported in severe forms of human disease. Thrombocytopenia has been associated with renal failure and death in human patients.

Respiratory involvement in leptospirosis could be classified as a) mild to moderate (20% to 70% of patients), with pulmonary infiltrates commonly associated with jaundice and minimal alteration of renal function; b) severe, with jaundice, nephropathy, hemorrhages (severe Weil's syndrome) (4), and occasional death due to renal failure, myocarditis, or massive hemorrhages with cardiovascular collapse; and c) pulmonary hemorrhage which is frequently fatal, without jaundice, nephropathy, or other hemorrhages.

In the past two decades, an increasing number of cases of leptospiral pulmonary hemorrhages have been reported, especially from South-east Asia (5). In a review of leptospirosis in Brazil, death was associated with renal failure in 76.2% of fatal cases, while 3.5% were related to pulmonary hemorrhages (6). In the epidemic outbreak in Nicaragua in 1995, this form was considered the cause of death in the 40 fatal cases reported (7).

The two cases reported here were associated with pulmonary hemorrhage. This clinical form has not been previously reported in the Buenos Aires metropolitan area. Environmental and social factors, the prevalence of infection in reservoirs, and the virulence of the isolated strains must be considered in primary or critical-care

units in the diagnosis of new cases, whether or not associated with an outbreak.

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First Documentation of Human Crimean-Congo Hemorrhagic Fever, Kenya

To the Editor: On October 21, 2000, a previously healthy 25-year-old male farmer was admitted to a mission hospital in western Kenya with an acute hemorrhagic illness. Four days before admission, the patient had rapid onset of fever, headache, nausea, vomiting, severe muscle pains, and diarrhea, which became bloody. On admission his temperature was 36.4°C, pulse was 60/minute, respiratory rate was 20/minute, and blood pressure was 90/40 mm Hg. In addition to the signs and symptoms listed above, the only other abnormal finding on admission was neck stiffness. The differential diagnoses included bacterial dysentery and meningitis. Results of a blood smear for malaria parasites and Widal test for typhoid were negative, and cerebrospinal fluid and urine examinations were normal.

The patient was treated with doxycycline, cotrimoxazole, metronidazole, and intravenous fluids. On the day after admission, the patient's vomitus became blood stained and blood was passed rectally. The patient was isolated and strict barrier nursing implemented on the suspicion of viral hemorrhagic fever (VHF). Progressive hypotension developed, resistant to resuscitation efforts with intravenous fluids and corticosteroids, and later massive bleeding from the nose, mouth, and upper and lower gastrointestinal tract occurred. The patient died on the second day of admission, 6 days after onset of illness. A serum sample was sent to the Arbovirus and Viral Hemorrhagic Fever Reference Laboratory in Nairobi for diagnostic screening.

Serologic tests in Nairobi were negative for yellow fever, dengue, West Nile, Chikungunya, and Rift Valley fever (immunoglobulin [Ig] M-

capture enzyme-linked immunosorbent assay) and reverse transcriptase-polymerase chain reaction (RT-PCR) tests for flaviviruses, alphaviruses, and Bunyamwera serogroup bunyaviruses were also negative. RT-PCR for *Crimean-Congo hemorrhagic fever virus* (C-CHFV) was positive. Tests for anti-C-CHFV-specific IgM antibody by indirect immunofluorescence were negative. Virus isolation attempts were then terminated because the cultivation of C-CHFV (the presumptive cause) requires biosafety level 4 facilities. The specimen was submitted to the Special Pathogens Unit in Johannesburg for confirmation of the result. The sample was positive by RT-PCR for C-CHFV and was IgM and IgG antibody negative. No isolation of the virus could be made from the serum sample, possibly because it was received by the Johannesburg laboratory 8 days after initial collection and following freeze-thaw conditions. The specimen was insufficient to attempt C-CHFV antigen detection assays. Sequencing of the RT-PCR amplicon confirmed C-CHFV.

C-CHFV is a tick-borne virus of the genus *Nairovirus*, family *Bunyaviridae*, and is widely distributed throughout eastern Europe and the Crimea, to the Middle East and western China, Pakistan, and Africa. Natural hosts for this virus are varied (including wild and domestic animals and birds) and may reflect the feeding preferences of the host tick (1). While C-CHFV infections are rare in humans, the virus is notorious for nosocomial outbreaks of VHF, typically following admission of an index case to a health-care facility where VHF was not suspected, with mortality rates up to 40%.

Previous evidence for C-CHFV in Kenya is limited and based on serology (human and bovine) and two isolations of C-CHFV from non-human sources (1,2). This report represents the first documented case of acute human C-CHFV infection in Kenya. The hospital concerned belongs to a VHF surveillance network serving to

increase awareness and preparedness within Kenyan health-care facilities. In this case suspicion of VHF was raised, and the patient was immediately isolated, noninvasive procedures were instigated, and barrier nursing was implemented to prevent nosocomial transmission. No family or hospital staff member who had close contact with the patient became ill. Although VHFs are rare, this report stresses the need for health facilities in Kenya and East/Central Africa to include VHFs in their differential diagnosis of unexplained fever with hemorrhagic tendencies, as well as the utility of the surveillance network. The causative agents of Ebola hemorrhagic fever, Marburg hemorrhagic fever, C-CHFV, Rift Valley fever, and yellow fever are all endemic in East and Central Africa, and sporadic cases, as well as outbreaks, are likely to continue to occur in this region (3–5).

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¹Dr. Kazooba-Voskamp, the attending physician in this case, has requested that the hospital's identity remain anonymous.

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Preparing at the Local Level for Events Involving Weapons of Mass Destruction

To the Editor: The use of hijacked airplanes in the attacks on the World Trade Center and the Pentagon on September 11, 2001, clearly illustrated the immediate and massive destruction that can result from a well-orchestrated, long-planned, and purposeful terrorist act. Weapons of mass destruction (WMD) events (i.e., biological, nuclear, or chemical attacks) present different challenges than other incidents involving mass casualties (e.g., chemical spills, transportation mishaps, or natural disasters). Persons involved in a biological weapons attack, for example, may take days to develop symptoms and seek medical care (1); a large geographic area may be affected, or persons may travel long distances and unwittingly infect others, including hospital personnel (2). Furthermore, traditional hazardous materials and emergency medical procedures may be inadequate to respond to a WMD event (3–5). As events of September 11 and its aftermath make clear, medical public health systems were not optimally prepared. An effective response to a WMD event focuses on two key areas: joint efforts between the medical community and public health agencies and better trained and coordinated first responders (i.e., law enforcement, public safety, hospital personnel, and public health officials) (1–3).

In early 2001, telephone interviews with West Virginia county health directors (CHDs) or their equivalent were conducted to ascer-