Medical News

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Hantavirus Outbreak Suggests Person-to-Person Transmission

In 1995, a novel Hantavirus (Andes virus) was identified in samples from patients in southern Argentina. The patients had a Hantavirus pulmonary syndrome (HPS), a disease first described 2 years earlier in the United States, in association with the Sin Nombre virus (a New World Hantavirus).

Old World Hantavirus (Puumala, Seoul, Hantaan, and Dobrava) has been a recognized cause of hemorrhagic fever with renal syndrome for at least 40 years. It is responsible for more than 100,000 cases each year in Eurasia.

Hantaviruses are rodent-borne, viral, zoonotic agents believed to be transmitted to humans primarily by inhalation of aerosols of infected rodent excretions. Person-to-person transmission of Hantaviruses has not been reported despite extensive epidemiological studies.

Between September 22 and December 5, 1996, 18 cases of HPS occurred in residents of, or visitors to, the towns of El Bolson, Bariloche, and Esquel in southern Argentina. Two additional persons who had contact with El Bolson patients, but had not visited the area, contacted HPS during this period.

Five of the patients were physicians; three were directly responsible for the clinical care of an HPS patient. Epidemiological links between all but four of the cases and evidence of low rodent population density in the area strongly suggest person-to-person transmission of HPS during this outbreak.

The investigators concluded that the transmission of Hantavirus from patient C to her doctor (patient D) provided the best epidemiological evidence to support the hypothesis of person-to-person transmission of Hantavirus.

The investigators reported that 21 and 20 days, respectively, after the 41-year-old index case in El Bolson became symptomatic, his 70-year-old mother and one of his doctors (patient A) contracted HPS. The doctor's spouse, also a doctor (patient C), became ill with HPS 27 days after her husband's first symptoms (19 days after his death). She traveled to Buenos Aires for medical care. While in a hospital in Buenos Aires, an admitting doctor (patient D) spent 1 hour taking a clinical history and examining her. The doctor (patient D) applied pressure to a venipuncture site on the patient's arm with multiple layers of gauze; no obvious blood contact occurred. The only other contact between the doctor (patient D) and patient C occurred 2 days later, when the doctor briefly visited the hospital's intensive-care unit to attend to another patient. Twenty-four days after attending to patient C, the doctor became ill with HPS. The doctor, patient D, had not traveled outside Buenos Aires and reported no contact with rodents during the 2 months preceding the illness.

The unique pattern of person-to-person transmission in this outbreak has not been a feature of Hantavirus epidemiology. In a study performed in New Mexico in 1993, no disease or Hantavirus antibodies were found in serum specimens taken from 396 healthcare workers, including 266 who had been exposed to patients with HPS or their body fluids 2 to 4 weeks earlier.

The high case fatality rate (50%) precluded a detailed description of the exact nature of contact between cases. Consequently, the likely mode of transmission, whether through direct contact, droplets, infectious aerosols, or contaminated fomites, is not known. Genetic sequencing of viruses from patients and rodents is being determined to define more clearly the patterns of transmission.

In the absence of any clear-cut evidence for nosocomial transmission of Sin Nombre or a related virus in the United States, the authors note that it is premature to suggest changing the existing guidelines for care of HPS patients.

FROM: Wells RM, Estani SS, Yadon ZE, et al. An unusual Hantavirus outbreak in southern Argentina: person-to-person transmission? *Emerg Infect Dis* 1997;3(2):171-174.

Continued Transmission of *Legionella* Despite Control Efforts

The CDC recently reported the results of an investigation of sustained transmission of nosocomial Legionnaires' disease (LD) in two hospitals located in Arizona and Ohio, implicating the hot-water distribution system in both hospitals. In 1996, hospital A in Arizona had eight cases of nosocomial LD among cardiac and bone marrow transplant patients. After an intensified investigation, 25 cases (16 definite and 9 possible) of nosocomial LD were linked to hospitalization during 1987 to 1996. No single risk factor was identified; however, information on exposure to showers, other aerosol sources, or ingested water for some patients was unavailable. During August 1996, cultures of Lp-6, Lp-11, *Legionella anisa,* and a *Legionella*-like organism designated as D-1620 were obtained from the hot-water distribution system. Lp-1, Lp-4, Lp-6, and Lp-11 were grown from swabs and water samples from the water softeners. Water from the wellhead of a private well that supplied some of the hospital water contained Lp-1. Lp-6 was grown from samples obtained from taps and showers in patients' rooms and a carpet cleaning unit used on the transplant ward. Air samples within patient showers identified Lp-6 in respirable (1-5 μm) droplets.

Thermal decontamination of the hot-water distribution system was conducted in hospital A in July 1996, but legionellae later were isolated from potable water, and three cases occurred after thermal decontamination. In response, the hot-water distribution system was hyperchlorinated, and the water temperature was maintained at 120°F; however, following these measures, legionellae Lp-6 again was grown from potable water. As a result, additional

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measures that were implemented included installing chlorine injection devices, removing areas of low flow (deadlegs) in the potable water plumbing, disconnecting the water softeners, and repeating the hyperchlorination procedures. No new cases have been identified since September 1996.

In a separate investigation in hospital B in Ohio, nosocomial LD occurred in two patients during January to June 1996. Since 1989, as part of surveillance for nosocomial LD in this hospital, urine samples from all patients with nosocomial pneumonia are tested for Lp-1 antigen. Following the identification of these two cases, an investigation identified a total of 38 cases (9 definite and 29 possible) of nosocomial LD. Although information about exposure to showers, aerosol sources, or ingested water was incomplete, in a case control study, cases were more likely than controls to have documented exposure to common aerosol-producing devices (showers or medication nebulizers) during the 2 weeks before onset. Medication nebulizers in this hospital sometimes were rinsed with tap water between doses to reduce clogging.

Lp-1 was isolated from samples obtained from multiple sites in the hot-water distribution system during 1994 to 1996. All Lp-1 isolated from potable hot-water samples in testing done from 1994 to 1996 and in 1984 were identical to the three clinical isolates from 1992, 1994, and 1995. Periodic cultures of the hot-water distribution system were used to guide decontamination efforts.

Thermal heating (to 160°F at the tap for 5 minutes) and chlorine decontamination (maintaining a chlorine level of 1 to 2 mg/L at the tap for at least 5 minutes) had been only temporarily effective in reducing the number of positive sites. A copper-silver ionization system installed in 1985 neither reduced the number of positive samples nor terminated transmission. Additional interventions were initiated, including discontinuing the use of tap water to rinse medical nebulizer equipment, repeating the hyperchlorination procedures as needed in response to positive potable water cultures, increasing the hot-water temperature at the point of use to at least 120°F, and identifying deadlegs in the potable water plumbing. Following these interventions, no new cases were identified until February 1997; Lp-1 isolates from this patient were identical to all previous isolates. A previously undocumented cross-connection between the hot-water tank from an adjacent outpatient building and the critical-care unit was discovered. After this tank was cleaned and the supply system hyperchlorinated, no new cases have been identified since March 1997.

These reports indicate the capacity for legionellae to colonize hospital plumbing systems for long periods and represent an ongoing risk for infection. Colonization rates are higher in large hospitals with older, large hot-water tanks in which water is held at lower temperatures. Growth of legionellae to high concentrations occurs most often at water temperatures of 77°F to 108°F. Concern about the risk for scalding injuries has prompted some jurisdictions to regulate temperatures in hospital hot-water systems at levels conducive to legionellae growth. Additions and alterations to hospital plumbing systems in response to changing hospital facility needs may create areas of stagnation and sediment build-up, factors also shown to enhance legionellae colonization. These stagnant areas may be resistant to chlorination, thermal disinfection, and ionization. Because eradication of legionellae from water distribution systems generally is not possible, a maintenance program to minimize regrowth of legionellae also should be implemented. Raising the water temperature to 120°F to 125°F at the fixture (higher temperatures may increase risk of scalding) and infusion of chlorine to maintain consistent levels of 1 to 2 mg/L have been employed to achieve long-term decontamination in many hospitals. Although metal ionization systems may be effective, it is not clear whether they offer any advantage over conventional methods.

Respiratory therapy equipment should be rinsed only with sterile (not distilled) or tap water.

FROM: Centers for Disease Control and Prevention. Sustained transmission of nosocomial legionnaires' disease—Arizona and Ohio. *MMWR* 1997;46(19):416-421.

An Outbreak of *Enterobacter hormaechei*

Wenger and co-investigators from CDC's Hospital Infections Program and the Hospital of the University of Pennsylvania recently reported an outbreak of *Enterobacter hormaechei* infections among premature infants in the intensive-care nursery (ICN) of a tertiary-care hospital.

Ten premature infants were found to be culture positive for *E hormaechei* (six were infected and four colonized) between November 29, 1992, and March 17, 1993.

All isolates were resistant to ampicillin and gentamicin. Isolates from the six infected infants were susceptible to aztreonam, ceftazidime, ceftriaxone, ciprofloxacin, imipenem, and trimethoprim-sulfamethoxazole. All isolates were resistant to cephalothin, piperacillin, and tobramycin.

A cohort study showed the 10 case patients had a lower median estimated gestational age and birth weight than did other ICN infants. Environmental cultures of isolettes and a doorknob in the ICN were positive for the organism. The environmental and case-patient *E hormaechei* isolates had identical pulsed-field gel electrophoresis band patterns. During the investigation, a number of breaks in infection control practices were observed. These included failure to use gloves, failure to change gloves or gowns between patients, and failure to wash hands or use proper handwashing techniques. Although handwashing facilities were adequate, soap and towels were not always available.

Control measures included the use of contact isolation for culture-positive infants in a common cohort room, additional inservice education of staff, and increased attention to cleaning environmental surfaces (eg, isolette tops).

The outbreak ended after patients were isolated from other infants, adherence to infection control was increased, cleaning of environmental surfaces was enhanced, and empirical antimicrobial covered was changed.