Letters to the Editor

Molecular Epidemiology and Legionnaire's Disease

To the Editor:

Legionella pneumophila is a common cause of nosocomial and community respiratory infection. Most legionella pneumonias are due to ***L pneumophila*** serogroup 1, the most common serogroup recovered in environmental investigations.[1,2]

Legionellosis has been a problem in our hospital, a 500-bed teaching institution, since it was inaugurated in 1983. In 1989, our situation was critical, with 40 cases (2.8/1,000 discharges). Restriction endonuclease analysis (REA) of genomic DNA using *Hind*III and conventional electrophoresis[3] demonstrated the existence of one "clone" of ***L pneumophila*** in the hospital water that was stable over time and identical to all clinical isolates. The chromosomal DNA pattern of the hospital isolate was very different from environmental and clinical isolates from outside our hospital.[4]

In response, we initiated energetic environmental interventions that included periodic microbiologic surveillance of the water system (bathroom faucets, shower heads, hot water tanks, and cooling towers) and monthly superheating (elevation of the temperature in the hot water system for 48 hours every month). The frequency of nosocomial legionella pneumonia declined to 20, 12, and 5 cases in 1990, 1991, and 1992, respectively (1.1, 0.7, and 0.35 cases/1,000 discharges).

The epidemiologic importance of a known, stable REA pattern to discriminate between nosocomial and community-acquired legionella pneumonia is illustrated by the following case. A 68-year-old man was admitted to the hospital to start chemotherapy for pulmonary oat cell carcinoma and then discharged for outpatient management. One week later, he was readmitted because of fever and obtundation. He had a history of chronic bronchitis and smoked two packs of cigarettes daily for 40 years. His temperature was 39.5°C, his pulse was 110 beats/minute, and his blood pressure was 90/50 mm Hg. Physical examination revealed dullness in the left hemithorax. The patient's white cell count was 8,000, with 82% neutrophils, 15% lymphocytes, and 3% eosinophils. His hematocrit was 31%, the urea 25.4 mmol/L, the sodium was 113 mEq/L, and the potassium was 3.8 mmol/L. The partial pressure of oxygen was 62 mm Hg, the partial pressure of carbon dioxide 35 mm Hg, and the pH level was 7.54. A chest radiograph was unchanged. Ceftazidime and aztreonam were started, but the patient deteriorated and died on day 3. The postmortem examination revealed consolidated pneumonia, and the culture was positive for ***L pneumophila*** serogroup 1. REA of genomic DNA using *Hind*III showed identity between the clinical isolate and the hospital isolate pattern (Figure).

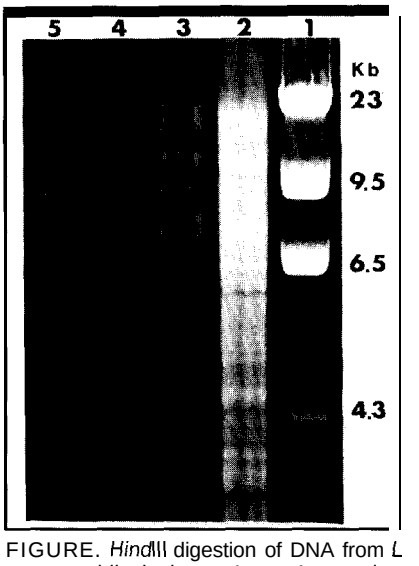


FIGURE. *Hind*III digestion of DNA from *L pneumophila* isolates. Lane 1 contains lambda marker: lane 2, endemic hospital isolate; lane 3, isolate from the patient; lanes 4 and 5, isolates from outside the hospital. Lanes 2 and 3 are identical, suggesting that the patient's pneumonia was nosocomially acquired.

This patient's pneumonia appeared presumptively to be linked to the hospital. However, the absence of recent nosocomial cases and the negative environmental screening during the prior 6 months didn't support this hypothesis. The issue was important, as confirmed nosocomial acquisition might obligate us to revise our ***Legionella*** control measures, involv-

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ing considerable institutional effort and major personnel resources. The demonstration by REA of identity between the patient isolate and the hospital isolate pattern strengthened the epidemiological association and supported the adoption of new control measures, including enhanced environmental surveillance, control of water temperatures in outlets during the superheating periods, and continuous hyperchlorination of the hot water system to maintain free chlorine levels >2 ppm.

This case demonstrates the usefulness of molecular typing methods[4-7] in resolving epidemiological problems and the importance of careful documentation of previous nosocomial isolates.

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Nosocomial Infection Control in Latin America

To the Editor:

In 1984, my mentor, Samuel Ponce de León, MD, wrote an editorial for this journal entitled, "Nosocomial Infection Control in Latin America: We Have to Start Now."[1] The article briefly summarized the prevailing situation in most public hospitals in Latin America: deficient sanitary systems, inadequate hospital-bed distribution, hospital personnel ignorance on infection control measures, and limited economic resources that resulted in a scarcity of medical equipment and supplies.

Almost ten years later, Mexico has shown notable progress in two areas. First, the number of medical articles on nosocomial infections has multiplied considerably. To cite an example, ***the Boletin Medico del Hospital Infantil de Mexico,*** the leading journal on pediatrics, published five articles on the subject during the last two years,[2-6] compared with only one article from 1989 through 1990.[7] Second, there has been increasing concern by both national and state health authorities with the problem. In 1986, surveillance of nosocomial infections was declared mandatory by law,[8] and in recent years, pressure has been exerted on hospitals throughout the country to comply with norms on monthly meetings for infection control committees and periodic notification of nosocomial infection rates to the National Ministry of Health.

Although information on the epidemiology of nosocomial infections and interest by health authorities are two essential conditions for achieving effective infection control, it is clear that these two alone are insufficient.

Dr. Ponce de León's editorial predicted that "We are not only facing a problem today but one that will be increasing in upcoming years." In the last decade, budget cuts for public hospitals in Mexico have been severe in every area, including staff, equipment, and hospital maintenance. During this period, only 0.37% of the GNP was invested in the public hospitals pertaining to the Secretaria de Salud, which covers approximately 40% of the Mexican population.[9]

The result is a frustrating situation in which one may know the particular risk factors for a certain infection, but not have the resources to eliminate or reduce them.

In the case of my hospital in Mérida, Yucatán, how can we expect to reduce our infection rate when we use large feeding tubes as intravenous catheters, when only open urinary drainage systems are available, when we disinfect surgical instruments and endotracheal tubes with benzalkonium chloride, and have a constant shortage of soap and paper towels? How can we strive for infection control when faced with gross understaffing, with a nurse-to-patient ratio that reaches 1:40 on weekends and night shifts? In the middle of endemic rates surpassing 35% and constant epidemics, I ask, "Is it possible to start effective infection control now?" The answer, I'm afraid, is no, with many more infections -- and deaths -- awaiting us.

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