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Fatal *Legionella maceachernii* Pneumonia in Canada

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A case of pneumonia and acute tubular necrosis was caused by an initially unknown species of *Legionella*. The organism was later identified as *Legionella maceachernii* by a combination of cultural, biochemical, and serological methods along with a gas-liquid chromatographic profile.

Legionella maceachernii is a recently described organism originally isolated from a potable-water system (2). It has also been reported as the cause of a fatal pneumonia in a patient with multiple myeloma (10). In this report, we describe a case of fatal *L. maceachernii* pneumonia occurring in an immunocompromised patient, which we believe to be the first such reported case in Canada.

The patient, a 54-year-old male, was admitted in September 1990 to a peripheral hospital with fever, chills, rigors, and right lower lobe pneumonia. He had been taking danazol and prednisone for autoimmune hemolytic anemia diagnosed in July 1990. He was initially treated with intravenous ampicillin, and later cefotaxime, tobramycin, and ticarcillin were added because of deterioration in his pulmonary status. The tobramycin was discontinued after two doses. His renal function on admission was normal, but 4 days later his urine output decreased dramatically and he developed acute renal failure with oliguria. He was given fluid challenges and diuretics, tobramycin was discontinued, and oral trimethoprim-sulfamethoxazole (Bactrim) and intravenous hydrocortisone sodium succinate (Solu-Cortef) were added to his treatment. He continued to deteriorate and was transferred to Regina General Hospital on the 10th day of treatment. On admission, he was found to have oliguria with a serum creatinine level of 900 μmol/liter, consolidation of the right lung, and marked tachypnea with hypoxia. His chest X ray showed large areas of consolidation involving the right lung with pleural effusion. He had a hemoglobin level of 104 g/liter and a leukocyte count of 16.4×10^9/liter with 95.4% polymorphs and a marked shift to the left. Sputum cultures (including a culture for *Legionella* species) and blood cultures were collected on admission and were later reported as negative. The initial impression was that he had an atypical pneumonia, and the possibility of legionella or mycoplasma infection was strongly considered. He was intubated and placed on intravenous erythromycin (1 g every 6 h) and intravenous cefotaxime. In view of the undiagnosed rapidly progressive pneumonia and abrupt onset of renal failure, the possibility of Goodpasture's syndrome was raised. The patient therefore underwent an open renal biopsy the next day, but he had a cardiac arrest soon after the procedure and could not be resuscitated.

At autopsy, the patient was found to have a confluent pneumonia affecting the major part of the right lung and acute renal tubular necrosis with no evidence of glomerulonephritis. He also had a large spleen associated with a hypercellular bone marrow, with the features being consistent with a hemolytic anemia. Cultures of the lung yielded a gram-negative bacillus, which grew aerobically on buffered charcoal-yeast extract (BCYE) agar but not on blood agar or chocolate agar. The organism resembled *Legionella* species but did not stain with direct immunofluorescence conjugates to *Legionella pneumophila* (Zeus Scientific Inc., Raritan, N.J.). The isolate was referred to the National Laboratory for Bacteriology, Laboratory Centre for Disease Control (LCDC) in Ottawa, Ontario, Canada, for further identification.

Characterization of the organism. The cultural characteristics of the organism were as follows. *Legionella*-like colonies were observed when the isolate was incubated in a candle jar on BCYE agar containing cysteine or cysteine with an antibiotic supplement consisting of polymyxin B, anisomycin, and vancomycin added, at 25, 37, and 42°C. However, such colonies were not observed at 55°C or on BCYE agar without cysteine. Growth with only small amounts of brown pigmentation was observed on buffered agar containing tyrosine (3). Autofluorescence was not observed. The catalase test was positive, and the oxidase test was weakly positive. β-Lactamase, hippurate hydrolysis, carbohydrate fermentation, urease, and nitrate reduction were negative. Contrary to observations noted previously for *L. maceachernii* (2), gelatinase production was not observed, even with prolonged incubation and repeated testing. Gas-liquid chromatographic profiles, obtained after extracting cellular fatty acids from a 72-h growth on BCYE agar as described previously (1), were consistent with those described for the type strain (7, 8). This profile included (averages of duplicate runs are given) a-15:0 (35%), 15:1B (5%), 15:0 (3%), i-16:1H (1%), i-16:0 (7%), 16:1cis9 (13%), 16:0 (5%), a-17:1C (5%), a-17:0 (22%), 17:0 (1%), and 20:0 (1%). The organism was reactive at only a 2+ intensity with a direct fluorescent antibody (DFA) monovalent antiserum used to detect *L. micdadei* prepared at LCDC; it gave a 1+ reaction against the *L. micdadei* Centers for Disease Control (Atlanta, Ga.) (CDC) monovalent antiserum for DFA and gave a negative reaction to *L. micdadei* monovalent antiserum (Prolab, Richmond Hill, Ontario, Canada). All other *Legionella* antisera for DFA in the LCDC collection were negative. This provided the first indirect evidence of the identity of this organism, as weak cross-reactivity between CDC-produced *L. micdadei* monovalent antiserum for DFA and the type strain of *L. maceachernii* had been previously observed (2, 9). LCDC-produced rabbit antiserum directed

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against *L. micdadei* used in the slide agglutination test was observed to give a 4+ reaction with *L. micdadei* TATLOCK but was negative against the unidentified organism. CDC-produced polyvalent pool V for the slide agglutination test, which contains antisera for detecting *L. micdadei*, *L. maceachernii*, *L. wadsworthii*, and *L. israelensis* antigens, gave a 4+ reaction against both *L. micdadei* TATLOCK and the unknown organism. All other CDC polyvalent pools for the slide agglutination test (I, II, III, IV, VI, VII, VIII, IX, and X) were negative. The CDC subsequently verified the identity of the strain as *L. maceachernii* by using monospecific antiserum for SAT.

This case report corroborates the report by Wilkinson et al. (10) that *L. maceachernii*, which was originally isolated from the environment, can be a cause of fatal pneumonia in patients who are immunocompromised. The patient we have described must be regarded as having been immunocompromised, as he was on corticosteroid therapy for an autoimmune hemolytic anemia.

Acute renal failure is a recognized complication of *L. pneumophila* infection (5) but is rare in cases of legionellosis not caused by *L. pneumophila* (4).

Identification of the specific causative agent in this case was based on biochemical, cultural, and gas-liquid chromatography results, as well as the observation of cross-reactivity of the organism with reagents used to detect *L. micdadei*, in the absence of monospecific reagents to identify this bacterium to the species level. The strain was confirmed as *L. maceachernii* by the CDC. A recent study has suggested that this organism be transferred to the genus *Tatlockia* on the basis of extensive genetic and chemotaxonomic analyses (6).

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REFERENCES

1. **Bernard, K. A., M. Bellefeuille, and E. P. Ewan.** 1991. Cellular fatty acid composition as an adjunct to the identification of asporogenous, aerobic gram-positive rods. J. Clin. Microbiol. **29:**83–89.
2. **Brenner, D. J., A. G. Steigerwalt, G. W. Gorman, H. W. Wilkinson, W. F. Bibb, M. Hackel, R. L. Tyndall, J. Campbell, J. C. Feeley, W. L. Thacker, P. Skaliy, W. T. Martin, B. J. Brake, B. S. Fields, H. V. McEachern, and L. K. Corcoran.** 1985. Ten new species of *Legionella*. Int. J. Syst. Bacteriol. **35:**50–59.
3. **Edelstein, P. H.** 1983. Legionnaire's disease laboratory manual, p. 64–66. Medical and Research Services, U.S. Veterans Administration, Washington, D.C.
4. **Fang, G.-D., V. L. Yu, and R. M. Vickers.** 1989. Disease due to Legionellaceae (other than Legionella pneumophila). Medicine (Baltimore) **68:**116–132.
5. **Fenves, A. Z.** 1985. Legionnaire's disease associated with acute renal failure: a report of two cases and review of the literature. Clin. Nephrol. **23:**96–100.
6. **Fox, K. F., A. Brown, A. Fox, and G. Schnitzer.** 1991. Tatlockia, a genetically and chemically distinct group of bacteria. Proposal to transfer *Legionella maceachernii* (Brenner et al.) to the genus Tatlockia, as *Tatlockia maceachernii* comb. nov. Syst. Appl. Microbiol. **14:**52–56.
7. **Lambert, M. A., and C. W. Moss.** 1989. Cellular fatty acid compositions and isoprenoid quinone contents of 23 *Legionella* species. J. Clin. Microbiol. **27:**465–473.
8. **Marmet, D., N. Borstein, and J. Fleurette.** 1988. Identification des Legionella par analyse des acides gras en chromatographie phase gazeuse (CPG) et des ubiquinones en chromatographie liquide haute performance (CHLP). Ann. Biol. Clin. **46:**371–375.
9. **Thacker, W. L., B. B. Plikaytis, and H. W. Wilkinson.** 1985. Identification of 22 *Legionella* species and 33 serogroups with the slide agglutination test. J. Clin. Microbiol. **21:**779–782.
10. **Wilkinson, H. W., W. L. Thacker, D. J. Brenner, and K. J. Ryan.** 1985. Fatal *Legionella maceachernii* pneumonia. J. Clin. Microbiol. **22:**1055.