**Legionella oakridgensis**: Laboratory Diagnosis of a Human Infection

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We report the laboratory diagnosis of a case of pneumonia caused by *Legionella oakridgensis*. *L. oakridgensis*, originally isolated from industrial cooling towers, has not previously been associated with human disease.

Of the 11 species and 12 serogroups of *Legionella* described, only *Legionella oakridgensis* and *L. sainthelensi* have not yet been associated with human infections (1, 6). We describe a case in which *L. oakridgensis* has been implicated as the cause of a human infection.

A 38-year-old woman was admitted in April 1984 to a local hospital for progressive systemic sclerosis (diagnosed in January 1984), renal failure, and cardiomyopathy. The patient was on prednisone (40 mg daily) from March 1984 on in an attempt to control her progressive renal insufficiency and myopathy. After 4 weeks in the hospital, a dry cough, tachypnea, and hypoxemia without fever developed. Chest films showed consolidation of the anterior segment of the right upper lobe and of the superior segment of the right lower lobe, along with bilateral pleural effusions. Aspiration pneumonia was considered in the differential diagnosis, and treatment was begun with clindamycin (600 mg every 6 h intravenously [i.v.]), erythromycin (250 mg every 6 h i.v.), and tobramycin (80 mg once i.v. and subsequently in dialysis fluids).

A right lung biopsy was performed. Routine bacteriological examination of the biopsy specimen yielded a scant growth (a few colonies each) of *Streptococcus faecalis*, *Serratia marcescens*, and *Morganella morganii*.

Direct fluorescent-antibody testing (2) for *Legionella* spp. was performed with the following 19 conjugates prepared in our laboratory as described previously (2): *L. pneumophila* serogroups 1 through 8, *L. bozemanii* serogroups 1 and 2, *L. longbeachae* serogroups 1 and 2, *L. micdadei*, *L. dumoffii*, *L. gormanii*, *L. wadsworthii*, *L. jordanis*, *L. oakridgensis*, and *L. feelei*. The species and serogroup specificities of each conjugate were verified before use. Culturing was done with charcoal–yeast extract–ACES buffer (Sigma Chemical Co.)–α-ketoglutarate medium (3), charcoal–yeast extract–ACES buffer–α-ketoglutarate medium supplemented with anisomycin, polymyxin B, and ceftamadole (3), blood agar medium, and MacConkey agar medium. Paired serum samples collected at 1-week intervals were tested by the indirect fluorescent-antibody test (5), except that formalized antigens of cultures grown on BCYEa medium were used; the antigens used for indirect fluorescent-antibody testing were similar to those used for direct fluorescent-antibody testing. The serum specimens were further tested by tube agglutination (4) with *L. pneumophila* serogroup 6 and *L. oakridgensis* antigens only. Further patient serum samples were not available.

After the diagnosis of legionellosis was made, erythromycin (500 mg every 6 h i.v.) and cefotaxime (500 mg every 6 h i.v.) were administered, the other antimicrobial agents were discontinued, and prednisone was tapered gradually to 25 mg daily. Over the next month there was a slow, but steady, clinical and radiological improvement, although the patient remained seriously ill from her underlying disease.

Five days after the 4-week course of erythromycin (1 week i.v. and 3 weeks orally) was finished, fever, shock, and respiratory failure developed abruptly, along with a new right-lung infiltrate. *S. marcescens* was cultured from blood and sputum, and the patient died 3 days later of adult respiratory distress syndrome. Permission for autopsy was denied.

The direct fluorescent-antibody test results showed brightly fluorescent (+), morphologically typical organisms against conjugates of *L. oakridgensis* and *L. pneumophila* serogroup 6 at >50 and 25 to 30 cells per smear, respectively. No fluorescence was observed either with the other 17 *Legionella* conjugates used or with the 3 bacterial species isolated from the lung biopsy specimen as a scant growth. Serum samples collected 5 days apart demonstrated at least a fourfold seroconversion in the indirect fluorescent-antibody test (from <1:32 for the first serum sample to 1:128 for the second) against *L. oakridgensis* only; the reaction for all other 18 antigens used, including *L. pneumophila* serogroup 6, was negative at a screening dilution of 1:32. The tube agglutination technique (4), which in our laboratory has always proven to be very specific, yielded negative results (<1:8) for the first serum sample and at least a fourfold rise to 1:32 for the second with *L. oakridgensis* antigen only. No growth of *Legionella* spp. was observed on any of the culture media. This may be the result of antibiotic treatment a few days before the biopsy was performed.

*L. oakridgensis* was originally isolated from industrial cooling towers and has not yet been incriminated in human infections, although its pathogenicity for guinea pigs suggests that it may be an unrecognized human pathogen (6). Demonstration of the organism in lung tissue and seroconversion in serum samples suggested that *L. oakridgensis* causes pneumonia in humans. The presence of *L. pneumophila* serogroup 6 indicated a double *Legionella* infection. It is unclear whether *L. oakridgensis* can act independently as a sole etiological agent or only in a synergistic relationship with other pathogens.

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