A Case of Corynebacterium pseudodiphtheriticum Nosocomial Pneumonia

To the Editor: Corynebacterium pseudodiphtheriticum has seldom been isolated from patients with upper respiratory tract infections and pneumonia. Most reported infections are community acquired and occur in patients with underlying disease and immunosuppression (1). We report a case characterized by hospital-acquired pneumonia in a debilitated patient. Review of the literature indicates that C. pseudodiphtheriticum should be regarded an emerging pathogen.

On April 1, 1998, a 68-year-old woman was admitted to the intensive care unit for acute respiratory distress. She had a 14-month history of amyotrophic lateral sclerosis. Three weeks before admission, she had been hospitalized for Staphylococcus aureus pneumonia and had recovered after treatment with amoxicillin and clavulanic acid plus ciprofloxacin. At the time of admission, the patient had a temperature of 38°C. Systolic blood pressure was 120 mm Hg. Ventilation was spontaneous, with respirations 24 per minute; pulmonary sibilants were noted. Respiratory acidosis was also identified, with a pH of 7.35, SaO₂ 92%, PaO₂ 60 mm Hg, and PaCO₂ 60 mm Hg. Laboratory data included 18,000 leukocytes per ml (90% polymorphonuclear cells) and a serum fibrin level of 7 g/L. A chest X-ray showed pneumopathy of the lower segment of the right lung, which was compatible with the diagnosis of inhalation pneumopathy. On day 2 of admission, the patient’s temperature was 39°C, and she had paresis of the vocal cords. After C. pseudodiphtheriticum infection was diagnosed, treatment with intravenous cloxacillin (1 g 3 times/day) and amoxicillin plus clavulanic acid (1 g 3 times/day) was started. On day 3 after admission, the patient’s breathing worsened, a radiograph showed bilateral pneumopathy, and she was intubated for mechanical ventilation. Two days later, her breathing improved, and a second bronchic aspiration was sterile. The patient eventually died of unrelated complications.

Direct microscopy examination of a Gram-stained bronchial aspiration sample showed numerous polymorphonuclear cells and gram-positive bacilli in parallel rows, which did not show pleomorphism. After 48 hours of incubation at 37°C, 10⁶ colony-forming units/ml of a coryneform bacillus further identified as C. pseudodiphtheriticum grew in pure culture on blood agar gelose (BioMérieux, La Balme les Grottes, France) under a 5% CO₂ atmosphere and did not produce hemolysis. The test for catalase was positive, and the following biochemical characteristics were obtained by using a commercial identification strip (API-Coryne, BioMérieux): absence of carbohydrate fermentation, urea hydrolysis, and nitrate reduction compatible with C. pseudodiphtheriticum. Minimum inhibitory concentrations (disk diffusion method) were 2 mg/L for amoxicillin, 2 mg/L for cefalotin, 0.09 mg/L for doxycycline, 0.03 mg/L for gentamicin, <4 mg/L for vancomycin, 16 mg/L for erythromycin, and 20 mg/L for trimethoprim-sulfamethoxazole. Identification was confirmed by analysis of the cell-wall fatty acid profile by the Sherlock system, by the trypticase soy broth agar database 3.9 (MIDI Inc., Newark, DE), and by 16S rRNA sequence analysis under previously described conditions (2). The 16S rRNA gene sequence was compared with all eubacterial 16S rRNA sequences available in the GenBank database by using the multisequence comparison Advanced Blast NCBI. The sequence had a 99% similarity to that of C. pseudodiphtheriticum (1039/1047 base pairs).

Eighty-nine cases of infection possibly caused by C. pseudodiphtheriticum have been identified in the last 57 years. Of these, 57 (62.9%) were upper respiratory infections, which included rhinosinusitis, tracheitis, tracheobronchitis, and bronchitis; 19 (21.3%) were pneumonia (3-7). Ten (11.2%) cases of endocarditis were reported (8); there was also one case each of urinary tract malakoplakia after renal transplantation (9), lung abscess (10), diskitis (11), and lymphadenitis (12).

Unlike C. diphtheriae, C. pseudodiphtheriticum is a commensal bacterium that does not produce toxins and needs predisposing factors to become a pathogen causing pneumonia. Of patients with hospital-acquired C. pseudodiphtheriticum upper respiratory tract infections and pneumonia (7 of 26 upper respiratory tract infections and 2 of 14 cases of pneumonia reported in the early 1990s), all had underlying pathologic features. Predisposing factors were as follows: 33.7% had lung and tracheobronchial diseases, including chronic obstructive pulmonary disease, angina
(5), chronic emphysema, asthma, and bronchitis (6); 32.5% had congestive heart failure (5). Of those with immunodepression, 5% had AIDS, 7.2% had undergone chemotherapy or prolonged steroid use; and 18.2% had other pathologic features, disseminated intravascular coagulation (6), chronic renal failure, diabetes mellitus (5), and connective tissue disease (5,6).

The first source of pneumonia is usually inhalation, as was the case for our patient, who had paresis of the vocal cords. She was not immunosuppressed but was debilitated by amyotrophic lateral sclerosis. The second factor is often an endotracheal intubation, as reported in a previously healthy 29-year-old trauma victim who contracted pneumonia due to C. pseudodiphtheriticum after 7 days of intubation (7). An increase in cases reported from 1932 to 1998 indicates the emergence of infections due to C. pseudodiphtheriticum. Thirty-four cases were reported from 1932 to 1989 (57 years), and 55 cases were reported from 1990 to 1998 (8 years). Reasons for the emergence of C. pseudodiphtheriticum infections may include confusion between C. diphtheriae and C. pseudodiphtheriticum infections. For example, two cases of C. pseudodiphtheriticum exudative upper respiratory tract infections with a pseudomembrane were first diagnosed as respiratory diphtheria. In the first case, C. pseudodiphtheriticum was isolated from a 32-year-old Uzbek man who had a severe sore throat and dysphagia lasting 2 days (3). In a second case, a 4-year-old girl had exudative pharyngitis with a pseudomembrane, which was possibly caused by C. pseudodiphtheriticum (4). The availability of commercial strips for the identification of C. pseudodiphtheriticum and 16S rRNA sequencing eliminates such confusion.

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References

Family Outbreak of Rickettsia conorii Infection

To the Editor: Over a 15-day period, three young siblings were separately taken to an emergency room in Israel, with symptoms suggesting a contagious viral illness (fever, maculopapular rash, hepatosplenomegaly, lymphadenopathy, neutropenia, and thrombocytopenia). None of the children had been in direct contact with animals. Specific immunoglobulin (IgM) immunofluorescence assay (IFA) 7 to 8 days after admission of each child confirmed the diagnosis of Rickettsia conorii infection.

Spotted fever is the generic name given to a variety of tickborne rickettsial diseases distributed worldwide. In Mediterranean countries, including Israel, spotted fever is caused by members of the R. conorii complex. Spotted fever has been endemic in Israel for more than 40 years, with several hundred cases reported annually. In 1997, two fatal cases were reported