(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 32year-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

- Brown AE. Other Corynebacteria and Rhodococcus. In: Mandell GL, Bennett JE, Dolin R, editors. Principles and practice of infectious diseases. 4th ed. New York: Churchill Livingstone; 1995. p. 1874.
- Weisburg WC, Barns SM, Pelletier DA, Lane DJ. 16S ribosomal DNA amplification for phylogenetic study. J Bacteriol 1991;173:697-703.

- 3. Santos MR, Ghandi S, Vogler M, Hanna BA, Holzman RS. Suspected diphtheria in a Uzbek national; isolation of *C. pseudodiphtheriticum* resulted in a false positive presumptive diagnosis. Clin Infect Dis 1996;22:735.
- Izurieta HS, Strebel PM, Youngblood T, Hollis DG, Popovic T. Exudative pharyngitidis possibly due to C. pseudodiphtheriticum, a new challenge in the differential diagnosis of diphtheria. Emerg Infect Dis 1997;3:65-8.
- Manzella JP, Kellog JA, Parsey KS. Corynebacterium pseudodiphtheriticum: a respiratory tract pathogen in adults. Clin Infect Dis 1995;20:37-40.
- Ahmed K, Kawakami K. Corynebacterium pseudodiphtheriticum: a respiratory tract pathogen. Clin Infect Dis 1995;20:41-6.
- Miller RA, Rompalo A, Coyle MB. Corynebacterium pseudodiphtheriticum pneumonia in an immunologically intact host. Diagn Microbiol Infect Dis 1986;4:165-71.
- 8. Wilson ME, Shapiro DS. Native valve endocarditis due to *Corynebacterium pseudodiphtheriticum*. Clin Infect Dis 1992;15:1059-60.
- Nathan AW, Turner DR, Aubrey C, Cameron JS, Williams DJ, Ogg CS, et al. Corynebacterium hofmannii infection after renal transplantation. Clin Nephrol 1982;17:315-8.
- Andavolu RH, Venkita J, Lue Y, McLean T. Lung abscess involving Corynebacterium pseudodiphtheriticum in a patient with AIDS-related complex. New York State Journal of Medicine 1986:86:594-6.
- 11. Wright ED, Richards AJ, Edje AJ. Discitis caused by Corynebacterium pseudodiphtheriticum following ear, nose and throat surgery. Br J Rheumatol 1995;34:585-6.
- LaRocco M, Robinson C, Robinson A. Corynebacterium pseudodiphtheriticum associated with suppurative lymphadenitis. European Journal of Clinical Microbiology 1987;6:79.

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

(1). Spotted fever is caused by a variant member of *R. conorii*, which is transmitted by the dog tick *Rhipicephalus sanguineous* (2,3). The disease has a broad spectrum of clinical signs, from asymptomatic to fatal (4,5). Symptoms and signs include fever, headache, vomiting, myalgia, conjunctivitis, and a typical maculopapular or purpuric rash. The tache noir at the site of the tick bite, which is found in patients in Europe, is seldom, if ever, seen in Israel.

The first patient, a 6-year-old boy, was taken to the pediatric emergency room with high fever and a diffuse rash, approximately 1 week after visiting a cousin who had similar complaints. Physical examination showed temperature of 40°C, chills, diffuse maculopapular rash all over the body, including the hands and feet, hepatosplenomegaly, and lymphadenopathy. Blood tests showed neutropenia, thrombocytopenia, and hyponatremia. Because Rickettsia was included in the differential diagnosis, immunofluorescent assay (IFA) for Rickettsia was performed and intravenous doxycycline (2 mg/kg/day) was initiated. One week later, the boy's 8-month-old sister was brought to the emergency room with similar complaints, and 2 days afterwards his 2-year-old sister began to have the same symptoms. A detailed history revealed that all children had played on a lawn frequented by dogs.

All three siblings had fever, chills, and diffuse maculopapular rash all over the body, including the hands and feet. An IgM IFA test for *R. conorii* from the first child was negative on the day of admission and became positive 8 days later. On the day of the boy's hospital discharge, his 8-month-old sister was taken to the emergency room. Her serology test was negative on admission but became positive 7 days later. The third (2-year-old) sibling's first blood test was negative, and the family did not agree to a second blood test. All three children responded well to doxycycline (2 mg/kg/day, with a double dose the first day) for 5 to 7 days. Most symptoms subsided within 48 hours.

Spotted fever is usually a sporadic illness and is not spread from person to person. Clusters of cases have been reported. Yagupsky reported spotted fever in Israel in a few children living near each other in an agricultural settlement (6). A report from the Delaware Division of Public Health described a group of children who had been camping together where contact with ticks

was likely (7). This case illustrates that spotted fever may be acquired even without direct contact with animals, through exposure to ticks in places frequented by infected animals. Our report suggests that Rickettsial illness should be considered in the differential diagnosis of fever with rash in disease-endemic areas, even if the timing of similar complaints in several family members suggests a contagious viral illness.

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References

- Spotted fever. Jerusalem, Israel: Ministry of Health, Department of Epidemiology; 1997.
- Goldwasser RA, Steiman Y, Klinberg W, Swartz TA, YJingberg MA. The isolation of strains of *Rickettsiae* of the spotted fever group in Israel and their differentiation from other members of the group by immunofluorescence methods. Scand J Infect Dis 1974;6:53-62.
- 3. Manor E, Ighbarieh J, Sarov B, Kassis 1, Regnery R. Human and tick spotted fever group rickettsia isolated from Israel: a genotypic analysis. J Clin Microbiol 1992;30:2653-6.
- Wolach B, Franci S, Bogger-Goren S, Drucker M, Goldwasser RA, Sadan N, et al. Clinical and laboratory findings of spotted fever in Israeli children. Pediatr Infect Dis J 1989;8:152-5.
- 5. Yagupsky P, Wolach B. Fatal Israeli spotted fever in children. Clin Infect Dis 1993;17:850-3.
- Agupsky P, Sarov B, Sarov I. A cluster of cases of spotted fever in a kibbutz in southern Israel. Scand J Infect Dis 1989;21:155-60.
- Rotz L, Callejas L, McKechnie D, Wolfe D, Gaw E, Hathcock L, et al. An epidemiologic and entomologic investigation of a cluster of Rocky Mountain spotted fever cases in Delaware. Del Med J 1998;70:285-91.

Iron and the Role of *Chlamydia* pneumoniae in Heart Disease

To the Editor: Chronic infection of the coronary arteries by *Chlamydia pneumoniae* has been proposed as a heart disease risk factor (1). One reason for this proposal is the organism's association with one or more other risk factors for heart disease (2). However, an independent pathogenic role for *C. pneumoniae* in heart disease is unlikely if its presence is only a marker for another risk factor. In the Helsinki Heart Study (3), markers of chronic *C. pneumoniae* infection were a significant risk factor for a cardiac event, independent of most traditional