Peritonsillar Abscess Caused by *Nocardia asteroides*

JOHN C. ADAIR, INA J. AMBER, AND JEFFREY M. JOHNSTON**

Division of Infectious Diseases, Department of Internal Medicine, University of Utah School of Medicine, Salt Lake City, Utah 84132

Received 8 June 1987/Accepted 28 July 1987

A 22-year-old man with recurrent pharyngitis developed a peritonsillar abscess from which aspirated material yielded a pure culture of *Nocardia asteroides*. It is likely that the organism was introduced iatrogenically during a prior tonsillar incision. Although unusual, *Nocardia* species should be considered and microbiological specimens should be handled appropriately in pharyngeal abscesses that respond poorly to conventional therapy.

*Nocardia* species are aerobic, filamentous, gram-positive bacteria that are found in nature as soil saprophytes. They are uncommonly reported human pathogens and cause disease predominately in immunocompromised hosts (7). The true incidence of nocardial infections is unknown and may be underestimated in the medical literature, because nocardiosis can masquerade as a variety of more common granulomatous, neoplastic, or infectious processes (6). The clinical presentation of nocardiosis includes localized cutaneous disease and primary pulmonary infection, often with dissemination to other organ systems.

This report documents a unique case of peritonsillar abscess caused by *Nocardia asteroides* in an otherwise healthy subject. Our case suggests iatrogenic inoculation of the organism into an unusual site and illustrates the importance of considering *Nocardia* species in the differential diagnosis of pharyngeal abscesses that respond poorly to conventional therapy.

In October 1986, a 22-year-old male with a 5-year history of recurrent pharyngitis of unknown etiology presented to an outside clinic complaining of a sore throat. On exam, bilateral tonsillar swelling was observed. The physician incised both tonsils and expressed purulent drainage from the right side. Normal pharyngeal flora was cultured from this material, and the cultures were discarded after 48 h. The patient received a 5-day course of intravenous cephalothin followed by a 10-day course of oral amoxicillin. He refused surgical intervention at that time and did not return for follow-up evaluation.

The patient developed a sore throat associated with fever and severe odynophagia in February 1987. He presented to another clinic, where physicians again observed bilateral prominence of the tonsillar fossae. The lesions were incised but no purulent drainage was noted, and cultures were not obtained. He was treated empirically with lincomycin and cefaclor. When symptoms persisted after 5 days of therapy, the patient was referred to the ear, nose, and throat clinic at our hospital and subsequently was admitted. He specifically denied recent cough or dyspnea. Physical examination revealed a low-grade fever (38.2°C), bilateral distention and inflammation of the supratonsillar folds with overlying pale exudate, and tender anterior cervical lymphadenopathy. The total peripheral leukocyte count was 16,900/mm³, with a normal differential. The patient’s chest roentgenogram was normal. The remainder of his physical exam and laboratory evaluation was unremarkable.

The tonsillar abscesses were aspirated on the day of admission, and the patient was treated with intravenous penicillin G. A Gram stain of the aspirate demonstrated polymorphonuclear leukocytes, but no organisms were observed. When symptoms persisted despite 2 days of antibiotic therapy, the patient underwent bilateral tonsillectomy. Shortly after surgery on hospital day 3, the microbiology laboratory reported the isolation of 4+ *Nocardia* species in pure culture from the initial aspirates. Penicillin was discontinued, and therapy with intravenous trimethoprim-sulfamethoxazole (160/800 mg every 6 h) was initiated. The patient defervesced in 2 days and, after a 1-week course of intravenous antibiotic therapy, was placed on a 3-month course of oral trimethoprim-sulfamethoxazole. Before discharge, the patient tested negative for human immunodeficiency virus antibody. The *Nocardia* species was eventually identified as *N. asteroides*.

Our patient may have had pharyngeal nocardiosis before evaluation at our institution. *Nocardia* species grow slowly, requiring that cultures be maintained for longer periods (4 days to 4 weeks) than routine bacteriologic specimens (2). Thus, previous cultures from our patient may not have been monitored for a period of time adequate to detect *Nocardia* species. In addition, the infection apparently recurred after therapy appropriate for organisms commonly encountered in peritonsillar abscess (mixed anaerobic bacteria, *Streptococcus pyogenes, Staphylococcus aureus*), suggesting the presence of an organism resistant to this therapy. Alternatively, earlier episodes of pharyngitis or tonsillitis may have resulted from a separate undetermined viral or bacterial pathogen, and *Nocardia* species may have been introduced by the incision and drainage procedure undertaken before admission to our hospital.

Although *Nocardia* species are most commonly acquired through the respiratory tract, with subsequent dissemination to extrapulmonary sites (3), we hypothesize that our case resulted from inoculation of organisms into the peritonsillar soft tissues. Reasons to suspect direct contact as the mode of transmission include the limited anatomic distribution of the patient’s abscesses, the recent tissue injury (drainage procedure), and the absence of evidence for systemic nocardiosis. We believe that nocardial colonization of our patient’s oropharynx preceded puncture of the mucosal surface, although this was never proven microbiologically. Nocardial colonization of the skin (4) and upper respiratory tract (5)
has been documented in immunologically competent patients.

Iatrogenic nocardial abscess involving the upper respiratory tract has occurred following dental manipulations and transtracheal aspiration. Goldman and Light noted a high incidence of nocardial anterior cervical abscess complicating transtracheal aspiration (A. L. Goldman and L. Light, Correspondence, Am. Rev. Respir. Dis. 111:707–708, 1975). Of note, all of these subjects had active pulmonary nocardiosis and immunodeficiency at the time of the procedure, whereas our patient had no apparent pulmonary infection or immunologic impairment. In an experimental murine model, the virulence of Nocardia species is critically dependent upon the route of inoculation (1). Perhaps inoculation of Nocardia species through the respiratory mucosa into the subcutaneous tissue, as in people infected after dental injury and transtracheal aspiration and, possibly, in our patient, creates a favorable environment for the development of nocardial abscess.

Although ulcerative nocardial pharyngitis in the absence of tissue trauma was described previously (7), our case is, to our knowledge, the first case of peritonsillar abscess caused by N. asteroides.

LITERATURE CITED