Acid-Fast Actinomyces in a Child with Pulmonary Actinomycosis

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Open-lung biopsy in a child with chronic pneumonia revealed branched gram-positive acid-fast organisms, later identified as Actinomyces israelii. Like Nocardia asteroides, A. israelii can be acid fast by the commonly used Putt stain. The pneumonia was cured with 12 weeks of penicillin treatment.

Pulmonary actinomycosis is a rare disease in childhood. The causative organism, Actinomyces israelii, belongs to the same family as Nocardia asteroides. Both are branching gram-positive bacteria capable of causing a chronic and progressive pneumonia. According to most textbooks, A. israelii, unlike N. asteroides, is not acid fast. We report a 14-year-old boy with pulmonary actinomycosis in whom the diagnosis was initially mistaken for nocardiosis because a lung biopsy revealed branched gram-positive, acid-fast organisms.

Case report. The patient is a 14-year-old Mexican-American male with Cornelia de Lange syndrome diagnosed at 21 months of age. He was well until July 1978, when fever and cough developed 2 weeks after extraction of a carious tooth. A diagnosis of pneumonia was made, and he was treated with oral penicillin, followed by erythromycin. On September 12 he was admitted to another hospital because of continued fever, cough, lethargy, and weight loss. He was treated with intravenous ampicillin and methicillin but continued to have fevers up to 41°C.

On September 26 he was transferred to Children’s Hospital Medical Center in Oakland for diagnostic bronchoscopy. On admission, his respiratory rate was 28/min, his heart rate was 104/min, blood pressure was 110/80 mmHg, and his temperature was 38°C. He was a short male with features of Cornelia de Lange syndrome. He had multiple carious, malaligned teeth. His gag reflex was intact. He had a grade 2/6 systolic flow murmur. His chest was barrel-shaped with no retraction. Rales were present diffusely in the basal area. He had no clubbing or cyanosis. The leukocyte count was 14,800 with 84% polymorphonuclear leukocytes, the hemoglobin was 9.1 g/dl, and the sedimentation rate was 58 mm/h. Quantitative immunoglobulins showed elevation of immunoglobulin G, sweat chloride was normal, and coccidioides and histoplasma complement fixation titers were negative. Sputum Gram stain and culture showed oral flora, and several acid-fast smears were negative. His chest radiograph showed right pleural thickening and bilateral alveolar and interstitial infiltrates (Fig. 1).

Bronchoscopy, right thoracentesis, and right lung aspirate were performed on 29 September. Aerobic and anaerobic cultures of all these specimens were negative. In the hospital he had intermittent fevers, lethargy, and anorexia. On 6 October his respiratory rate increased to 60/min and his heart rate increased to 150/min. An arterial blood gas showed a partial pressure of oxygen of 59 mmHg, a partial pressure of carbon dioxide of 23 mmHg, and a pH of 7.50. His chest radiograph showed increased infiltrate and a right pleural effusion. He was treated with a fractional inspired oxygen concentration of 0.40. An open-lung biopsy showed multiple areas of consolidation and abscess formation. Gram stain of this specimen showed gram-positive branched organisms which were acid-fast by a Putt stain. No sulfur granules were seen. Nocardiosis was suspected initially, and the patient was treated with trimethoprim-sulfamethoxazole. After 7 days, anaerobic cultures grew gram-positive branching rods and gram-negative rods, and penicillin was substituted.

The organisms were subsequently identified as A. israelii, Bacteroides melaninogenicus, and Fusobacterium nucleatum. Penicillin G, 107 U per day, was continued for 8 weeks. The patient remained afebrile with gradual improvement. Oral penicillin was continued for another 4 weeks. Five months after stopping treatment, the patient remains well. Repeat chest radiograph shows only minimal pleural thickening without pulmonary infiltrates.

Discussion. Very few cases of pulmonary actinomycosis in children have been reported in recent literature, and it has been suggested that
this disease may be on the decline due to improved dental hygiene in the general population (9). Although cervicofacial infections are the most common form of actinomycosis, thoracic involvement accounts for 15% of infections. In their classic review of thoracic actinomycosis in 1957, Bates and Cruickshank reported that 7% of their 85 cases occurred at less than 10 years of age, and 20% occurred between 10 and 20 years (1). Their youngest patient was 28 days old.

As part of the normal oral flora, A. israelii is found in increasing numbers when dental caries are present (2). Pneumonia results from aspiration of oropharyngeal secretions containing a heavy inoculum of A. israelii, often after dental procedures.

Diagnosis depends on a high index of suspicion and identification of the organism from lung biopsy, pulmonary needle aspiration, or pleural fluid. Gram stain of clinical material may show only gram-positive rod or coccobacillary forms due to fragmentation. These can be mistaken for contaminating diphtheroids. Anaerobic cultures must be kept for at least 7 days. Colonies on agar plates may be only 0.5 to 1 mm in diameter after 7 days of incubation, and, in broth, growth may not be evident for 5 to 7 days, when nodular whitish granules can be seen in the bottom half of the tube (10). Isolation from sputum may merely reflect normal colonization.

Histopathological examination of the biopsy material may be of more immediate help to the clinician, since the anaerobic Actinomyces species, as well as the aerobic Nocardia species with which they may be confused, are slow to grow, and their precise identification can be lengthy. Presence of sulfur granules in lung tissue confirms the diagnosis of actinomycosis. The granules are composed of colonies of A. israelii in a matrix of calcium phosphate; granule size varies from microscopic to several millimeters in diameter. However, even on multiple sections these granules may be difficult to find; in one large series they were present in only 25% of the cases (3).

In the absence of sulfur granules, and pending the results of cultures, the acid-fast characteristics of the Actinomyces and Nocardia species are often used to distinguish the two groups of organisms. Most textbooks, including a well-known pediatrics text (5), state that whereas N.
asteroides is often acid fast, A. israelii is not. As our case illustrates, and as demonstrated earlier by Robboy and Vickery (8), using Putt stains of five of five isolates of A. israelii from various tissue specimens, and Hotchi and Schwarz (7), using tonsillar actinomycotic granules, this is not always true. The Putt stain, using a weaker acid solution to decolorize the principal dye carbofuchsin, is more sensitive than the Ziehl-Neelsen stain for demonstrating acid-fastness in N. asteroides.

This stain, however, should not be relied upon to distinguish between Nocardia and Actinomyces. Other modifications of the Ziehl-Neelsen stain, the Fite-Faraco method, or the Kinyoun stain may be useful in this regard. Although these stains are more difficult to perform, A. israelii does not appear acid fast by these methods (7, 8).

Distinguishing between A. israelii and N. asteroides is clinically important because the two bacteria have different antibiotic sensitivities. Untreated, pulmonary actinomycosis is ultimately fatal (4), whereas early treatment will prevent the late complications of extensive disease and result in cure rates of over 90% (1). Intravenous penicillin G is the treatment of choice for pulmonary actinomycosis, followed by long-term oral penicillin. Erythromycin, clindamycin, and tetracycline are effective alternatives in patients with serious penicillin hypersensitivity (6). Prolonged treatment, often for several months, is required to prevent relapse, unlike the more usual anaerobic aspiration pneumonias, where shorter antibiotic regimes are effective.

LITERATURE CITED