NOTES

Blood-Borne Pulmonary Infection with Nocardia asteroides in a Heroin Addict

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Nocardia asteroides frequently causes primary lung infection with possible hematogenous dissemination to various organs, especially in patients with altered immune responses. The preponderance of pulmonary infections suggests an airborne route of contamination. We report a case of pulmonary nocardiosis in a previously healthy intravenous heroin abuser. The clinical and epidemiologic data strongly suggest a paraphrenal blood-borne infection in this patient, a mode of contamination which has not been previously reported.

Nocardia are aerobic, filamentous, branching, gram-positive, partially acid-fast organisms usually found in soil or composting vegetation (2, 14). Nocardia asteroides is the most common species associated with human systemic nocardiosis although other pathogens, i.e., N. brasiliensis and N. caviae, can also cause infection (2, 6, 11, 18). Nocardiosis occurs predominantly in patients with renal transplant or other conditions that compromise host defenses (2, 14). The predominance of pulmonary infections suggests an airborne route of contamination (2, 9, 14). This report is, to our knowledge, the first case of blood-borne pulmonary infection with N. asteroides occurring in a previously healthy intravenous drug abuser. Of particular interest is the probable mode of contamination by accidental injection of material containing nocardia into the venous circulation, resulting in septic pulmonary embolism and pleural effusion.

A 17-year-old man was admitted to the Henri Mondor Hospital on 28 May 1984 for a 1-week history of right pleuritic chest pain and a 2-day history of sweats and fever. He indicated that he had been addicted to heroin for one year, taking two shots daily until the day of admission. He denied homosexual practice and had no history of previous illness. On examination, he appeared in good condition. His temperature was 38.5°C. His blood pressure was 120/70 mm Hg, and his respiratory rate was 24 per min. He had needle track marks on both forearms, but no other cutaneous lesions or evidence of thrombophlebitis. Examination of the thorax disclosed dullness and audible rales at the base of the right lung. The heart sounds were normal, without murmur or gallop. Abdominal examination was unremarkable. The liver and spleen were not felt. No lymphadenopathy was found, and the neurologic findings were normal. An initial chest roentgenogram revealed a small right pleural effusion and a right lower lobe infiltrate. Laboratory tests were as follows: hemoglobin 15 g/dl; leucocyte count, 10,800/mm³ with 65% polymorphonuclear cells and 28% lymphocytes; erythrocyte sedimentation rate, 50 mm/h; platelet count, 353,000/mm³; prothrombin time was normal; serum creatinine, urea nitrogen level, and alanine transaminase and serum alkaline phosphatase activities were within normal ranges. Serum protein electrophoresis disclosed an albumin level of 4.1 g/dl; immunoglobulin A, G, and M values were normal. Within 24 h of admission, the patient’s temperature spontaneously returned to normal but the sharp pleuritic chest pain persisted. An echocardiogram disclosed no evidence of valvular disease or vegetation. A right-sided thoracentesis was performed which yielded 30 ml of purulent fluid containing 10,800 leukocytes per mm³, with 58% neutrophils, 34% lymphocytes, and 8% monocytes. The protein level was 6.4 g/dl. A Gram stain examination of the pleural fluid disclosed no microorganism. No staphylococcus was available for examination. During the next few days in the hospital, the patient remained afebrile. Two other attempts at thoracentesis were unsuccessful. A second echocardiogram, performed at day 5, was unchanged. Clindamycin was given orally (1,200 mg/day). By the tenth hospital day, the pleuritic chest pain persisted. A repeated chest X-ray film appeared unchanged.

Cultures of the pleural fluid were obtained on Mueller-Hinton agar (Institut Pasteur Production [IPP], France), chocolate agar slants (Biomérieux, France) and Schaedler broth (Biomérieux). All incubations were at 37°C, and one of the chocolate agar slants was incubated in 5% CO₂-enriched atmosphere. Cultures were examined daily for 5 days, then at days 8 and 10. Nine blood cultures were drawn on aerobic and anaerobic media (IPP) between the first and sixth hospital days. No special medium for fungi was used. The cultures were examined daily. After 15 days of incubation at 37°C, the cultures were subcultured on chocolate agar in CO₂-enriched atmosphere and on 5% horse blood Columbia agar (IPP) in an anaerobic jar (BBL Microbiology Systems, Cockeysville, Md.). They remained sterile. After 8 days of incubation, the Mueller-Hinton agar and Schaedler broth inoculated with pleural fluid yielded growth of gram-positive, filamentous, branching bacteria which were identified as Nocardia species on the basis of aerobic growth on various media (Sabouraud agar, Lowenstein-Jensen me-

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dium), cultural characteristics, and microscopic morphology. The organism was not acid-fast. The isolate was identified as *N. asteroides* (3) since it could degrade urea and esculin, but not tyrosine, casein, or hypoxanthine. It produced acid from glucose and rhamnose, but not from lactose, mannitol, sorbitol, inositol, or galactose. The *N. asteroides* isolate was resistant to ampicillin, cefalothin, clindamycin, gentamicin, tobramycin, and trimethoprim and was susceptible to amikacin, minocycline, and sulfanomides, as tested by the disk-agar diffusion method. The MICs of cefotaxime, minocycline, and sulfamethoxazole were determined by the agar dilution method, as described by Bach et al. (1), with a few modifications. Briefly, a large inoculum of the test strain was placed in 10 ml of Mueller-Hinton broth containing sterile glass beads. The tube was incubated at 37°C in a shaker bath for 48 h. The inoculum was then adjusted to 10⁷ CFU/ml and streaked on antibiotic-containing Mueller-Hinton agar plates. The MICs were read after 4 days of incubation at 37°C and were 2, 4, and 32 µg/ml for cefotaxime, minocycline, and sulfamethoxazole, respectively.

When culture results became available, sulfamethoxazole-trimethoprim was substituted for clindamycin at daily doses of 2,400 and 480 mg, respectively. The pleuritic pain disappeared within 48 h, and a repeated chest X-ray film showed a persistent but smaller pleural effusion; the right lower lobe infiltrate was unchanged. The patient was discharged on July 13 on a 4-week course of antibiotics, but he declined follow-up. Possible sources of contamination by *N. asteroides* were sought. Although neither the patient’s injection equipment nor a sample of his heroin was available for culture, he related that 1 month before admission he had stored his syringes and needles in a flower-bed in anticipation of a police raid.

Pulmonary nocardiosis is an opportunistic infection which occurs most frequently in debilitated patients or in patients with otherwise abnormal host defenses (2, 14). Our patient was a heterosexual intravenous-drug abuser, which is a known risk factor for the acquired immunodeficiency syndrome (5, 10). However, he had no clinical marks of acquired cellular immunodeficiency or lymphopenia and no previous opportunistic infections (7, 10).

The exposure of our patient’s heroin paraphernalia to the soil is strongly suggestive of a direct intravenous inoculation of nocardia by means of the contaminated injection equipment. Drug contaminants and nonsterile injection techniques have been emphasized as a common source of blood-borne infections in intravenous-drug abusers. This could explain in part the variety of organisms, especially gram-negative bacilli (4, 15, 17) and fungi (8, 13), found responsible for septic pulmonary emboli, septicemia, or endocarditis in this group, *N. asteroides* has been cultured from one heroin sample in a microbiologic study of addicts’ equipment obtained by Tuazon et al. (19), and this organism probably contaminates paraphernalia. However, no case of septic pulmonary embolism due to *N. asteroides* has previously been reported in drug addicts. It is probable that, in our patient, massive or repeated contaminated intravenous injections caused septic embolization of the lungs.

Septic pulmonary emboli in intravenous-drug abusers are extremely suggestive of right-sided endocarditis (12, 17), although they may occur in its absence (4). *N. asteroides* endocarditis could not be ruled out in our patient on the basis of the negative blood cultures because they are usually negative in disseminated disease (9, 16); blood cultures on appropriate fungal media, which seem superior to routine media for recovery of nocardia (14, 16) were not performed. However, endocarditis seems unlikely for the following reasons: (i) the rapid, spontaneous abating of the fever within 24 h of admission; (ii) the stability of the clinical condition over a 6-day period without the use of antibiotics, followed thereafter by treatment with clindamycin, which is not effective against nocardia infection; and (iv) the absence of vegetation or valvular involvement on two echocardiographic examinations.

Our observations suggest that *N. asteroides* should be added to the list of opportunistic pathogens that may cause systemic infection via self-administration of contaminated material by intravenous-drug abusers.

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**LITERATURE CITED**


