Pulmonary *Nocardia transvalensis* infection: A case report and review


ABSTRACT

Infections due to *Nocardia transvalensis* are rare; only five cases of isolated primary pulmonary disease due to this species have been previously reported. Our patient is a 53-year-old diabetic who developed primary pulmonary *N. transvalensis* pneumonia and who was successfully treated with trimethoprim-sulfamethoxazole and amikacin. A summary of the published cases with *N. transvalensis* infection is included. The report emphasizes the increasing importance of *N. transvalensis* as an etiologic agent of pulmonary disease in immunosuppressed patients.

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**Keywords:** *Nocardia transvalensis* infection, pulmonary nocardiosis, emerging pathogens, nocardiosis in diabetics.

Human pulmonary infections due to *Nocardia* species are infrequently recognized although their actual incidence may be much higher. Recently, *Nocardia transvalensis*, although extremely rare, has emerged as an important pathogen causing potentially fatal pulmonary and disseminated disease in immunocompromised patients. Based on Medline search, only three cases with disseminated infection and five with primary pulmonary infection have been previously reported. We document here a case of pulmonary *N. transvalensis* infection in a male diabetic from Kuwait.

**Case report.** A 53-year-old male diabetic (type II), and a chronic smoker (2 packs/day for 20 years) was admitted with history of fever and productive cough without hemoptysis. His presenting symptoms also included weakness, sweating and pleuritic chest pain on the right side. In addition, he had known diabetic complications such as peripheral neuropathy, retinopathy and unhealed diabetic foot. Clinical examination revealed him to be febrile, tachycardic (pulse 120/min) and normotensive. He had evidence of mild pleural effusion with pneumonic consolidation on the right side. His cardiovascular system and abdomen were normal. Laboratory investigations revealed high blood sugar (20.8 mmol/L), mild anemia and no leucocytosis (white blood cell count 9,900 cells/mm3), and normal liver and kidney functions. His chest radiograph confirmed right-sided haziness in the midzone with mild pleural effusion. Two days after admission, the blood culture grew *Streptococcus pneumoniae* sensitive to penicillin and he was started on crystalline penicillin. The patient did not respond to this treatment and his respiratory symptoms persisted. Further, he developed right pleuritic chest pain with definite areas of consolidation in the mid and lower zones. His chest roentgenogram confirmed these findings. On the sixth day of admission, he underwent bronchoscopy and a bronchoalveolar lavage (BAL) specimen was collected for microbiological investigations. Gram-stained smears demonstrated thin, filamentous, branched, beaded gram positive organisms. On culture, the specimen yielded dry, chalky white, slow growing colonies on blood agar after 4 days of incubation at 37°C. The smears from the culture showed characteristic microscopic morphology of *Nocardia* and exhibited partial acid fast character when stained with modified Kinyoun's method using 1% sulphuric acid. The organism was identified as *Nocardia transvalensis* on the basis of positive hydrolysis reaction with xanthine, hypoxanthine and tyrosine. Adenine and casein were not decomposed. The identity of the isolate was confirmed by Dr. Patrick Boiron, Mycology Unit, Pasteur Institute, Paris, France. Antimicrobial susceptibility test was carried out by modified Kirby Bauer disk diffusion method using Mueller-Hinton agar. The isolate was susceptible to amikacin, tobramycin, gentamicin, kanamycin, minocycline, sulfamethoxazole and trimethoprim-sulfamethoxazole (TMP-SMX) but was resistant to ampicillin, amoxicillin-calvunate, imipenem, cefotaxime, ceftiraxone, ciprofloxacin and erythromycin. The patient was started on intravenous TMP-SMX. On day 14,
amikacin was added to regimen. He improved steadily during the next 14 days and became asymptomatic. The lung lesion disappeared and he became afebrile. He was discharged on oral TMP-SMX which was continued for the next six months on regular follow-up.

**Discussion.** *Nocardia transvalensis* was first described by Piiper and Pullinger in 1927 as the cause of mycetoma pedis in an African patient. Subsequent infections due to this species were reported rather infrequently. Gordon et al. studied isolates from three patients with mycetomas originating from Cameroon, Mexico and St. Louis and identified them as *N. transvalensis*. In 1982, Gugnani et al. documented an isolated case of *N. transvalensis* mycetoma of thumb in a 30-year-old female Nigerian and investigated its virulence for laboratory mice. Recently, McNeil et al. reviewed 16 cases of *N. transvalensis* infection/colonization reported between 1982-1992 and accepted the etiologic role of this species in only 10 of them. Three additional cases of *N. transvalensis* infection, two with subcutaneous disease and one with disseminated disease have since been published from Israel, United States and Pakistan. The first case of primary pulmonary nocardiosis due to *N. transvalensis* was seen in a Barbadian girl who developed upper lobe consolidation. Four more cases of isolated pulmonary nocardiosis, three from Queensland one from USA have since been reported. Salient features of these cases are summarized in Table 1. Considering that cases of *N. transvalensis* infection have occurred in countries located in different geographic regions it is reasonable to infer that this species, like other pathogenic nocardiae, has worldwide distribution. No environmental source of *N. transvalensis* infection is identified. As has been found for other *Nocardia* species, soil is the probable reservoir for this unusual actinomycete. Likely modes of its transmission are traumatic implantation which may result in mycetoma and inhalation which in a predisposed immunocompromised patient may result in pneumonia or disseminated disease. Our patient had diabetes mellitus and was a chronic smoker which could have predisposed him for this infection. In addition, he was a manual labourer and was exposed to dust. Chronic granulomatous disease, chronic alcoholism, primary lung disease and immunosuppressed state have been recognised as other predisposing factors for *N. transvalensis* infection (Table 1). Pulmonary infection may progress causing locally invasive or disseminated disease. Nodular pulmonary shadows and pleural effusion have all been reported in *N. transvalensis* infection. Our patient had the latter two manifestations.

Antimicrobial treatment of nocardiosis is problematic and TMP-SMX is the therapy of choice because these two drugs are believed to be synergistic in action and have a good penetration to cerebrospinal fluid. Even though, a high rate of resistance to aminoglycosides has been reported for this species, our isolates were sensitive to aminoglycosides but resistant to all cephalosporins hence amikacin was added to the therapeutic regimen which led to the successful outcome. About 18% isolates of *N. transvalensis* has been reported to be resistant to TMP-SMX combination. Nonetheless, previous clinical experience with *N. asteroides* suggests that TMP-SMX should probably be regarded as first line agent for treatment of infection with *N. transvalensis*. Antimicrobial susceptibility testing for *Nocardia* could be helpful in optimal drug selection particularly when primary drug resistance is noted. However, as yet there is no reliable data correlating in vitro observations with in vivo therapeutic benefit. Out-patient represents only the
second report of \textit{N. transvalensis} infection from the Middle-East. The present report underscores the increasing recognition of \textit{N. transvalensis} as an etiologic agent of pulmonary disease in immunocompromised patients.

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\textbf{References}