Case Report

Fatal pulmonary infection by trimethoprim-sulfamethoxazole resistant Nocardia otitidiscaviarum: report of two cases and review

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Abstract

Introduction: Nocardia otitidiscaviarum is a rare cause of human infections, mostly causing cutaneous and lymphocutaneous infections of mild severity. We report two cases of fatal pulmonary infection caused by Nocardia otitidiscaviarum in elderly patients.

Methodology: Case 1: A 70-year old woman presented with fever and cough with expectoration for a month. On physical examination, she had tachypnea and inspiratory crepitations in bilateral basal regions. Case 2: A 74-year old man presented with productive cough with foul smelling expectoration, fever and shortness of breath for one week. On examination, he had tachypnea, bilateral wheezing and inspiratory crepitations. In both cases, sputum was sent to microbiology laboratory. On direct microscopy Gram-positive, finely branching filaments were observed which were acid fast with 1% sulphuric acid. Chalky white opaque wrinkled colonies with musty basement type odour were seen on blood agar. Both patients were treated empirically with trimethoprim-sulfamethoxazole for Nocardia infection after notification of microscopy findings however both expired on Day 2 and Day 5 of admission, respectively. Both isolates were susceptible to amikacin, linezolid, ciprofloxacin and gentamicin. They were resistant to trimethoprim-sulfamethoxazole, ampicillin, amoxicillin-clavulanic acid, erythromycin, and imipenem. Based on biochemical identification and antimicrobial susceptibility pattern, the organism was identified as Nocardia otitidiscaviarum. The identification was confirmed using MALDI-TOF (Vitek MS, Biomerieux, France).

Conclusion: Our report highlights the importance of early identification of Nocardia to species level to improve treatment outcomes especially in critically ill patients. Mass spectrometry can become an integral part of diagnostic algorithms for nocardiosis.

Key words: Nocardia otitidiscaviarum; pulmonary infection; fatal.


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Introduction

Nocardia spp. are Gram-positive aerobic, branching bacilli, which are variably acid fast [1]. They are commonly found in soil, decomposing vegetation, water and air [1]. Pulmonary nocardiosis is the most common clinical presentation of infection as inhalation is the primary route of bacterial exposure [2]. The most common lung isolates of Nocardia have historically belonged to the formerly so-called Nocardia asteroides complex [3] subsequently identified mostly as N. cyriacigeorgica [4]. In recent reports, commonly isolated species include N. asteroides, N. cyriacigeorgica, N. abscessus, N. nova, N. brasiliensis and N. farcinica while other species are rarely reported [5-7].

Nocardia spp have been reported as a rare cause of infections in India although exact incidence of disease is difficult to estimate due to lack of awareness and diagnostic facilities. Although a large study from north-west India reported 36 cases of pulmonary nocardiosis over the period of 5 years, species level identification was not performed [8]. Smaller case series of 6 – 8 cases from different regions of India have reported N. asteroides and N. brasiliensis as the most commonly isolated causes of pulmonary nocardia infections [9-11].

Nocardia otitidiscaviarum has been less frequently reported as a cause of human infections, accounting for only 3-5% of all nocardia infections [3,7]. Recent studies from India have reported sporadic infections with Nocardia otitidiscaviarum [10,11]. Solid organ transplant and immunosuppressive therapy were the common risk factors for infection. Most patients were young to middle aged. This report describes two cases of fatal pulmonary infection caused by Nocardia otitidiscaviarum in elderly patients.
Case Presentation

Case 1

A 70-year-old lady presented to the emergency referred from another hospital. She had cough with expectoration for the past one month. The expectoration was foul smelling and brownish in colour. She also complained of mild fever present on and off, shortness of breath and chest pain on both sides mostly while coughing for the same duration. The patient had been prescribed some antibiotics by a local physician but there was no improvement in her condition; instead symptoms had worsened over the past month.

She had no past history of tuberculosis, diabetes mellitus, hypertension, bronchial asthma, chronic lung disease, coronary artery disease. There was no history of smoking, alcohol or tobacco intake. She did not have any history of occupational exposure to hay. The patient had no history of steroid intake and was not immunocompromised.

On examination, the patient was conscious, oriented, afebrile but looked sick. Mild pedal edema was present which was pitting in nature. There were no signs of pallor, icterus, cyanosis or clubbing. She had tachypnea with a respiratory rate of 25 breaths per minute. There was decreased air entry in right interscapular region and occasional crepitations in bilateral basal region (more marked on the left side as compared to the right). Tenderness was present in the right hypochondrium. No abnormality was detected on examination of cardiovascular and central nervous systems. The patient was admitted to the medicine ward and prescribed injection amoxicillin-clavulanate 2g thrice daily and tab azithromycin 500 mg once daily. The patient was then investigated. On chest X-ray, right lung showed consolidation and lower lobe collapse (Figure 1). Complete blood count revealed that her haemoglobin was 13.5 g/L, total leucocyte count was 16,300 cells/mm³ with 93.6% neutrophils, 5.1% lymphocytes, 1.3% monocytes, 1.4% basophils and 0.6% eosinophils. On arterial blood gas analysis, PaO₂ was 55.4 mmHg, PaCO₂ was 43.6 mmHg and blood pH was 7.45. Her liver function and renal function tests were within normal limits.

In view of worsening general condition, the patient was transferred to the pulmonary medicine ICU on day 2 of admission. The antimicrobial treatment was continued. Oxygen therapy was provided through mask (3-4L/hour). Patient was also nebulized with ipratropium bromide, salbutamol and budesonide. Sputum was sent to the microbiology laboratory for investigation. On gram staining, Gram-positive, finely branching beaded filaments were observed (Figure 2). Modified acid fast staining using 1% H₂SO₄ for decolourization detected acid fast beaded branching filaments (Figure 2). Acid fast staining with 20% H₂SO₄ did not show any acid fast bacilli. The physician was
notified regarding suspected pulmonary Nocardiosis and tab trimethoprim-sulfamethoxazole (TMP-SMX) 160/800 mg twice daily was added to the treatment. Unfortunately, the patient died the same night after receiving a single dose of TMP-SMX. As divided doses of 5 to 10 mg/kg per day of the trimethoprim component (or 25 to 50 mg/kg per day of sulfamethoxazole) are recommended to produce sulfonamide serum concentrations between 100 and 150 μg/mL [2], the prescribed dose may have been inadequate for treatment.

Sputum was inoculated on 5% sheep blood agar and MacConkey agar and incubated at 37 ± 2°C. On blood agar after overnight incubation chalky white dry opaque wrinkled colonies 1-2 mm in size with clear margin and musty basement type odor were observed (Figure 2). No growth was seen on MacConkey agar. On Gram staining, gram positive branching filaments were seen and modified Ziehl Neelsen (ZN) staining confirmed these were acid fast bacilli.

Case 2

A 74-year-old man presented to the outpatient department with productive cough, fever and shortness of breath for past one week. Sputum was foul smelling and dark yellow in colour. The above symptoms had been present on and off since last 18 months, but became severe since past 1 week. The patient had taken some medication over the past year for the symptoms with no apparent improvement. He could not recall the exact medication he had taken and no prescriptions were available with him. He had history of pulmonary tuberculosis over ten years ago and had been declared cured after taking antitubercular drugs for six months. There was no history of diabetes mellitus, hypertension, bronchial asthma, chronic lung disease or coronary artery disease. He gave history of smoking and alcohol consumption off and on. There was no history of intake of steroid intake or immunosuppressed state.

On examination, the patient was conscious but disoriented. He was febrile and sick looking. He had tachypnea with a respiratory rate of 40 breaths per minute. On auscultation, bilateral wheezing and inspiratory crepitations were present. The patient was admitted in the department of pulmonary medicine and investigated. On chest X-ray, fibronecrotic changes in both lungs were detected. Complete blood count showed a total leucocyte count of 16,500 cells/mm³ with 94.6% neutrophils, 4.3% lymphocytes, 1.1% monocytes, 1.2% basophils and 0.2% eosinophils. On arterial blood gas analysis, PaO₂ was 56.2 mmHg, PaCO₂ was 42.4 mmHg and blood pH was 7.5. His liver and renal function tests were within normal limits.

The patient was prescribed i. v. meropenem 2 g thrice daily and i. v. colistin 2.5mg/kg/day in two divided doses as antibacterial therapy. He was also prescribed injection enoxaparin, oxygen by mask (3-4 litres/hour) and nebulized with ipratropium bromide, salbutamol and budesonide. On sputum microscopy by Gram staining, fine branching, gram positive filaments were observed and modified Ziehl Neelsen (ZN) staining confirmed these were acid fast bacilli.

### Table 1. Scheme for identification of *Nocardia otitidiscaviarum* from clinical specimen.

<table>
<thead>
<tr>
<th>Test</th>
<th>Finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gram staining</td>
<td>Gram-positive, finely branching beaded filaments</td>
</tr>
<tr>
<td>Ziehl-Neelsen staining with 1% H₂SO₄</td>
<td>Acid-fast beaded branching filaments</td>
</tr>
<tr>
<td>Culture characteristics on sheep blood agar</td>
<td>Chalky white dry opaque wrinkled colonies 1-2 mm in size with clear margin and musty basement type odor</td>
</tr>
<tr>
<td>Paraffin bait technique</td>
<td>White growth at the junction of the paraffin and the yeast nitrogen base medium</td>
</tr>
<tr>
<td>Biochemical reactions</td>
<td>Reduction of nitrate; hydrolysis of xanthine, hypoxanthine and urea, and the inability to hydrolyse casein and tyrosine</td>
</tr>
<tr>
<td>Antibiotic susceptibility testing [1]</td>
<td>Susceptible to kanamycin, gentamicin, amikacin, trimethoprim-sulfamethoxazole, and ciprofloxacin; resistant to ceftriaxone, ampicillin, amoxicillin-clavulanic acid, carbenicillin, and imipenem (often resistant to all β-lactam antibiotics)</td>
</tr>
<tr>
<td>Mass spectrometry, 16s rRNA sequencing</td>
<td>For confirmation of species</td>
</tr>
</tbody>
</table>
Identification of species

Nocardia have the capability of utilizing paraffin as the sole source of carbon and therefore paraffin baiting technique was used to isolate suspected Nocardia spp. from the sample (Figure 2). A white growth was seen at the junction of the paraffin and the yeast nitrogen base medium and confirmed that the organism was Nocardia spp. The isolates reduced nitrate, hydrolysed urea and did not hydrolyse tyrosine. The scheme for identification of Nocardia otitidiscaviarum has been detailed in Table 1.

Antibiotic susceptibility testing was performed by Kirby Bauer disk diffusion method. Inoculum was prepared as previously described [12]. Isolates were subcultured on sheep blood agar to ensure purity. They were subsequently subcultured in Mueller-Hinton Broth (MHB) and incubated at 35 ± 2°C for 2-4 days in ambient air. The MHB tubes were periodically vortexed at high speed for 2 to 3 min to achieve a uniform suspension of the organisms, and vortexing was repeated prior to optical density determinations. After obtaining a turbidity equivalent to 0.5-1.0 McFarland, the suspension from MHB was uniformly swabbed onto a Mueller-Hinton agar plate and the following drugs were applied for susceptibility testing. The antibiotics used are described in Table 2. – amikacin, amoxicillin-clavulanic acid, ciprofloxacin, erythromycin, gentamicin, imipenem, linezolid and TMP-SMX [12]. Plates were incubated at 35 ± 2°C for 3 days and read after every 24 hours. For each antibiotic disk, the final diameter of the inhibition zone was recorded and compared with breakpoints described in Table 2 [13,14]. Both isolates were sensitive to amikacin, linezolid, ciprofloxacin and gentamicin. They were resistant to ampicillin, amoxicillin-clavulanic acid, erythromycin, TMP-SMX and imipenem. Based on biochemical identification and antimicrobial susceptibility pattern, the organism was identified as Nocardia otitidiscaviarum [1]. The identification was confirmed using Vitek MS (Biomerieux, Marcy-l'Étoile, France), a matrix assisted laser desorption ionization-time of flight mass spectrometry (MALDI-TOF) system.

Discussion

Nocardiosis is an acute or chronic infection caused by members of genus Nocardia. Human infection with Nocardia spp. can result due to inhalation of mycelial fragments in case of pulmonary nocardiosis or contact with the bacteria via a cut or abraded skin (cutaneous nocardiosis-cellulitis, ulcers), and the infection can then disseminate to the brain, kidneys, joints, heart and bones [15]. Pulmonary disease is the most common manifestation of nocardia infection [1]. Extrapulmonary manifestations occur in 50% of cases with pulmonary disease and include spread to pleura, pericardium, mediastinum and superior vena cava [16].

N. otitidiscaviarum was first recognized from a Sumatran cavy or guinea pig with ear disease by Snijders in 1924 [17]. Reports of human infections first appeared in 1960s and first case of systemic infection was reported in 1974 [3]. In large studies, it was noted that N. otitidiscaviarum is isolated rarely, constituting about 3-5% of all Nocardia infections [3,7]. Reduced pathogenicity, lower prevalence in soil and incomplete identification up to species level have been postulated as various reasons for this low incidence [18]. In the present report, the species was determined based on biochemical identification and antimicrobial susceptibility pattern identification [1] and confirmed by mass spectroscopy (MALDI-TOF).

N. otitidiscaviarum has been reported to cause pulmonary, cutaneous, lymphocutaneous and disseminated infections involving brain, bones and liver in both immunocompromised and immunocompetent patients [18].

Table 2. Antibiotic disks used for antibiotic susceptibility testing and interpretation thresholds.

<table>
<thead>
<tr>
<th>Antibiotic</th>
<th>Dose/disk (µg)</th>
<th>Interpretation threshold (mm)*</th>
<th>S</th>
<th>R</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ampicillin</td>
<td>10</td>
<td>≥ 21</td>
<td>&lt; 16</td>
<td></td>
</tr>
<tr>
<td>Amoxicillin/clavulanic acid</td>
<td>20/10</td>
<td>≥ 23</td>
<td>&lt; 16</td>
<td></td>
</tr>
<tr>
<td>Imipenem</td>
<td>10</td>
<td>≥ 24</td>
<td>&lt; 17</td>
<td></td>
</tr>
<tr>
<td>Gentamicin</td>
<td>15</td>
<td>≥ 18</td>
<td>&lt; 16</td>
<td></td>
</tr>
<tr>
<td>Amikacin</td>
<td>30</td>
<td>≥ 17</td>
<td>&lt; 15</td>
<td></td>
</tr>
<tr>
<td>Erythromycin</td>
<td>15IU</td>
<td>≥ 22</td>
<td>&lt; 17</td>
<td></td>
</tr>
<tr>
<td>Linezolid</td>
<td>30</td>
<td>≥ 28</td>
<td>&lt; 24</td>
<td></td>
</tr>
<tr>
<td>Trimethoprim/sulphamethoxazole</td>
<td>1.25/23.75</td>
<td>≥ 16</td>
<td>&lt; 10</td>
<td></td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>5</td>
<td>≥ 25</td>
<td>&lt; 22</td>
<td></td>
</tr>
</tbody>
</table>

*This table is adapted from guidelines of the Antibiogram Committee of the French Society of Microbiology (CA-SFM) devoted to Gram-positive bacteria [13].
<table>
<thead>
<tr>
<th>Reference</th>
<th>Year</th>
<th>Age/sex</th>
<th>Associated risk factors</th>
<th>Immune status/ immunosuppressive therapy</th>
<th>Drug susceptibility</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Princess et al.</td>
<td>2018</td>
<td>51/M</td>
<td>Bronchial asthma</td>
<td>On corticosteroids</td>
<td>Resistant to TMP-SMX, amoxicillin-clavulanate; Susceptible to amikacin, ciprofloxacin, linezolid, imipenem, and ceftriaxone</td>
<td>Azithromycin + Doxycycline $\rightarrow$ Imipenem + TMP-SMX</td>
<td>Died</td>
</tr>
<tr>
<td>Savitha et al.</td>
<td>2017</td>
<td>65/M</td>
<td>None</td>
<td>Immunocompetent</td>
<td>Resistant to TMP-SMX; Susceptible to TMP-SMX, ceftriaxone, imipenem, linezolid</td>
<td>TMP-SMX</td>
<td>Recovered</td>
</tr>
<tr>
<td>Candel et al.</td>
<td>2017</td>
<td>79/M</td>
<td>COPD</td>
<td>On corticosteroids</td>
<td>Resistant to TMP-SMX; Susceptible to aminoglycosides, beta-lactams and carbapenems</td>
<td>Levoflaxacin + Vancomycin + Tobramycin</td>
<td>Died</td>
</tr>
<tr>
<td>Liu et al.</td>
<td>2017</td>
<td>58/M</td>
<td>Cotton farmer, Hepatitis B Carrier</td>
<td>Immunocompetent</td>
<td>Not performed</td>
<td>TMP-SMX $\rightarrow$ Imipenem + Amikacin + TMP-SMX</td>
<td>Died</td>
</tr>
<tr>
<td>Sadamatsu et al.</td>
<td>2017</td>
<td>72/F</td>
<td>Influenza A, Bronchial Asthma</td>
<td>On Inhaled corticosteroids</td>
<td>Resistant to ceftriaxone; Susceptible to TMP-SMX, minocycline, levofloxacin, imipenem</td>
<td>TMP-SMX + Minocycline</td>
<td>Recovered</td>
</tr>
<tr>
<td>Deepa et al.</td>
<td>2016</td>
<td>14/F</td>
<td>Rheumatic heart diseases</td>
<td>Immunocompromised</td>
<td>Resistant to amoxicillin-clavulanate, cefotaxime, ceftriaxone; Susceptible to gentamicin, ciprofloxacin, amikacin,TMP-SMX, tetracycline, imipenem</td>
<td>Ceftriaxone</td>
<td>Died</td>
</tr>
<tr>
<td>Mahgoub et al.</td>
<td>2016</td>
<td>41/F</td>
<td>Bronchial asthma</td>
<td>Immunocompetent</td>
<td>Resistance to azithromycin, erythromycin, ceftazidime, penicillin, rifampin, and TMP-SMX; Susceptible to amikacin, cefditoren, ciprofloxacin, meropenem and streptomycin</td>
<td>TMP-SMX + Amikacin + Imipenem $\rightarrow$ Ceftriaxone + amikacin + ciprofloxacin</td>
<td>Recovered</td>
</tr>
<tr>
<td>Huang et al.</td>
<td>2015</td>
<td>42/M</td>
<td>Alcoholic liver disease</td>
<td>On corticosteroids</td>
<td>Not performed</td>
<td>Piperacillin-tazobactam $\rightarrow$ vancomycin $\rightarrow$ meropenem + TMP-SMX + amikacin</td>
<td>Recovered</td>
</tr>
<tr>
<td>Ramamoorthi et al.</td>
<td>2011</td>
<td>36/F</td>
<td>Farmer</td>
<td>Immunocompetent</td>
<td>Not performed</td>
<td>TMP-SMX</td>
<td>Recovered</td>
</tr>
<tr>
<td>Betran et al.</td>
<td>2010</td>
<td>57/M</td>
<td>Diabetes mellitus, thrombocytopenia</td>
<td>On corticosteroids</td>
<td>Resistant to imipenem, amoxicillin-clavulanate, cefotaxime, tobramycin, ciprofloxacin; Susceptible susceptible to amikacin, gentamycin and TMP-SMX</td>
<td>TMP-SMX</td>
<td>Recovered</td>
</tr>
<tr>
<td>Peleaz et al.</td>
<td>2009</td>
<td>85/F</td>
<td>Hypertension, coronary disease, COPD</td>
<td>On corticosteroids</td>
<td>Resistant to penicillin, ampicillin, vancomycin, erythromycin; Susceptible to TMP-SMX, imipenem, linezolid, gentamicin, amikacin, ciprofloxacin</td>
<td>TMP-SMX $\rightarrow$ Linezolid</td>
<td>Died</td>
</tr>
<tr>
<td>Chawla et al. – 4 cases</td>
<td>2009</td>
<td>1-23/M 2 – 17/M 3 – 62/F 4 – 36/M</td>
<td>Case 1 and 2 -Post renal transplant Case 3and 4 – Pulmonary TB</td>
<td>On Immunosuppressive therapy</td>
<td>All were susceptible to TMP-SMX</td>
<td>TMP-SMX + cefepime + amikacin</td>
<td>Recovered</td>
</tr>
</tbody>
</table>
The risk factors described for infections include smoking, sickle cell anemia, diabetes mellitus, chronic obstructive pulmonary disease, renal insufficiency, mixed connective tissue disorder, cirrhosis, human immunodeficiency virus (HIV) infection, malignancies, long-term corticosteroid therapy, and bone marrow or solid organ transplant [18]. The mean age of the patients has been reported as 56.61 ± 18.58 years [18].

The PubMed and Google scholar database was searched with ‘Nocardia otitidiscaviarum’, ‘lung infection’, ‘pulmonary’ and ‘pneumonia’ and 23 cases have been reported [19-37], which are presented in Table 3. Sixteen cases were male and seven were female patients. The median age at the time of presentation was 58 years (range 14-85 years). Nine of the twenty-three reported cases had associated chronic pulmonary diseases including bronchial asthma, chronic obstructive pulmonary disease (COPD), tuberculosis at the time of infection [19,20,23,25,29,30,31,37]. Thirteen of these twenty-three cases were reported in immunocompromised patients. Corticosteroid therapy was identified as a risk factor for *N. otitidiscaviarum* in seven of the thirteen immunocompromised cases [19,20,23,26,28,29,31]. In the other six reports, post-renat transplant [10], Cushing’s disease [33], HIV- AIDS (acquired immunodeficiency syndrome) [34,37] and rheumatic heart disease [29] were identified as the cause of immunodeficiency. The remaining cases were reported in immunocompetent patients. Four of these otherwise healthy patients were engaged in farming where inhalation of dust and hay were postulated as a source of infection [22,27,30,35]. Only in a single case reported by Savitha et al. no associated risk factors like immunocompromised state, underlying lung disease or farming was observed [20].

Our patients were elderly, both over 70 years of age and a review of literature suggests that elderly patients were most commonly affected with *N. otitidiscaviarum* (Table 3). However, they did not suffer from any immunosuppressive disorders, underlying lung disease and gave no history of steroid intake. Both patients had normal renal and liver functions. Most cases of *N. otitidiscaviarum* recover after long term treatment (6-12 months) but there have been reports of mortality especially in case of severe and disseminated disease.
Only five of the fifteen pulmonary cases reviewed were fatal including four patients who were immunocompromised. Both our patients had chronic illnesses where diagnosis of nocardiosis had been missed on previous examinations. On presentation to our hospital, the infection was severe and rapidly fatal within the first week of hospitalization. Only one case of fatal N. otitidiscaviarum in an immunocompetent patient from China has been reported so far [22].

In our patients, TMP-SMX was prescribed as empirical treatment for nocardiosis pending susceptibility testing. Both isolates later tested resistant to TMP-SMX and the patients expired before the change in treatment could be effected. Sulphonamides have been regarded as the drug of choice for N. otitidiscaviarum infections for more than 50 years and treatment is effective in most cases [1]. However, definitive treatment should be guided by antimicrobial susceptibility testing, especially for N. otitidiscaviarum as most studies have reported isolates which are resistant to beta-lactam drugs like ampicillin, amoxicillin-clavulanic acid, and imipenem and inconsistent susceptibility to sulphonamides is being increasingly reported (Table 3) [2,18,22]. We found resistance to TMP-SMX in seven of the seventeen (41.2%) cases where antibiotic susceptibility had been performed. Therefore, it is imperative that species level identification is performed to guide the treatment protocol. Mass spectrometry or MALDI-TOF can provide rapid identification of the isolate by MALDI-TOF. RS conducted the literature review and drafted the manuscript. MKS and JCS clinically managed the patients and revised the manuscript. RG was involved in supervision of the laboratory tests and crucially revised the manuscript. RD and RS confirmed the identification of isolate by MALDI-TOF. RS collected the data from the patient’s medical records. RG was involved in supervision of the laboratory tests and crucially revised the manuscript. RD and RS confirmed the identification of isolate by MALDI-TOF. RS conducted the literature review and drafted the manuscript. MKS and JCS clinically managed the patients and revised the manuscript. All authors have read and approved the manuscript.


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