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Nocardia yamanashiensis causing skin abscess in an immunocompetent child

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Abstract
Nocardiosis is caused by an aerobic actinomycete, most commonly introduced through the respiratory tract. Primary cutaneous nocardiosis infections are rare and are caused by traumatic introduction of the organism. It manifests usually as an opportunistic infection. We report a case of cutaneous nocardiosis in an immunocompetent child, presented as an abscess. Nocardia yamanashiensis was isolated from drained pus and child responded well to antibiotics.

Introduction

Nocardiosis is a chronic and severe pyogranulomatous disease caused by environmentally ubiquitous actinomycete of the *Nocardia* genus.[1] It was first isolated by veterinarian Edmond Nocard in 1888 from a case of bovine lymphadenitis. The genus *Nocardia* is widely distributed in soil, organic matter, fresh and salt water. They are aerobic, filamentous gram-positive, atypical acid-fast bacteria that can cause localised or systemic infections mostly in immunocompromised patients.[2]

Primary cutaneous nocardiosis, however, is a rare disease characterised by nodules, subcutaneous abscess formation, ulcerations, pyoderma or cellulitis. In contrast to systemic nocardiosis, mostly immunocompetent patients get affected. The most frequently isolated species is *N. brasilensis*.[3] *Nocardia yamanashiensis* was first isolated from a skin abscess of a 30-year-old female Japanese patient in 1987, but only in 2004 its taxonomic position was established.[4]

Treatment of primary cutaneous nocardiosis is often challenging and depends mainly on the susceptibility pattern and severity of disease. Medical therapy with prolonged courses of antimicrobials is associated with substantial clinical improvement. The most common antimicrobials used for this condition are sulfa drugs, aminoglycosides, beta-lactams and tetracyclines. Combined drug therapy is always preferred to avoid drug resistance and to achieve microbiologic cure.[4]

Case Report

A five month old male child presented in paediatric surgery department with complaint of swelling on the left arm for 2 days. There was no

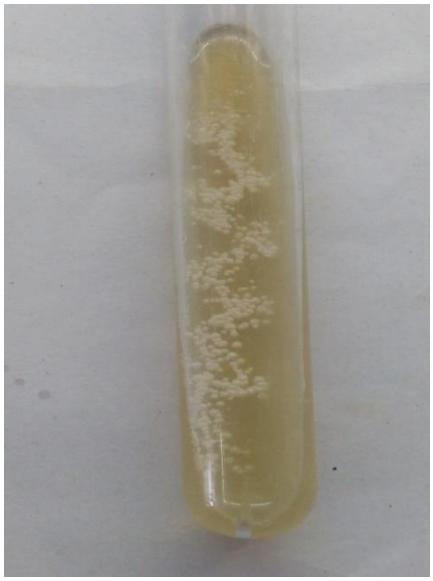
associated history of fever, discharge or any local trauma. No history of any similar swelling elsewhere in the body. No previous history of recurrent pneumonia, failure to thrive, sepsis or any other illness suggestive of immunisuppression in the past. On examination a globular, cystic swelling of size 3×2 cm with smooth surface was observed. The abscess was drained and the pus was sent for culture and sensitivity. The patient was started empirically on parenteral Cefotaxime and Amikacin.

On gram staining, pus cells were present along with gram positive filamentous bacilli. On modified Zeil-Neelssen staining with 1% sulphuric acid, the organism was partially acid fast. The sample was inoculated on routine culture media, Sabouraud's dextrose agar and Löwenstein–Jensen medium.

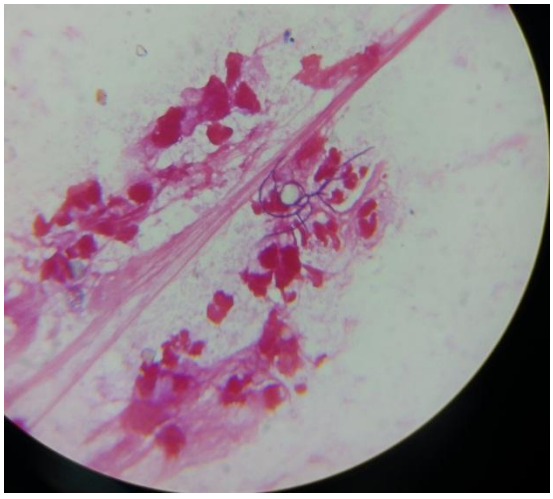
After 48 hours of incubation, on blood agar moderate growth of 1-2 mm sized chalky white rough non haemolytic colonies, firmly adherent to the underlying medium were observed. Gram staining of the colony smears revealed gram positive filamentous bacilli which were partially acid fast on modified Zeil-Neelssen staining with 1% sulphuric acid. Among biochemical reactions, the organism was catalase positive, oxidase negative, reduced nitrate to nitrite and urea and aesculin hydrolysed. Later similar colonies were also obtained on Sabouraud's dextrose agar and Löwenstein–Jensen medium.

Later to confirm, the organism was sent for MALDI-TOF test and identified as *Nocardia yamanashiensis.* Based on culture results, antibiotics were changed to oral Co-Trimoxazole (200/40 mg, 5ml twice daily). Child improved on antibiotic and sent home on oral Co-Trimoxazole, to continue for 6 months.

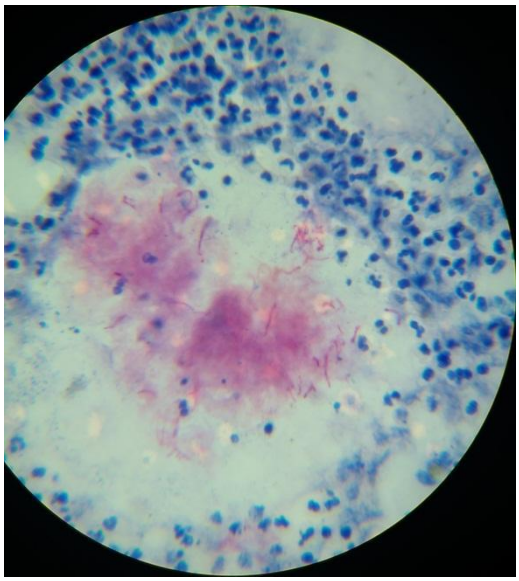
Colonies on LJ medium



Colonies on SDA



Gram positive filamentous bacilli on gram staining



Modified Zeil-Neelssen staining

Discussion

Nocardia yamanashiensis rarely cause infection in humans and only few cases are reported.[5] But the overall incidence of human nocardiosis increased in the last two decades across several countries, particularly in patients affected by immunosuppressive diseases or therapies.[1] However, the cutaneous presentations are not always associated to a previous health condition, indicating possible transmission through cutaneous trauma.[1] The disease usually affects young adult males, indicating the occupational risk of human infection by *Nocardia* species.[6] In this case the patient is a five month old child with no history of immunosuppression or significant illness in the past. Parents did not give any history of cutaneous trauma too.

Managing *Nocardia* infection is often complicated by drug intolerance (e.g. cutaneous eruption following use of sulphonamides), treatment failure, recovery of primary drug-resistant strains, or development of resistance during therapy.[4] Trimethoprim/ sulfamethoxazole (Co-trimoxazole) has been the drug most often employed.[7] The recommended dosage is 5 to 10 mg/kg trimethoprim and 25 to 50 mg/kg sulfamethoxazole in two to four divided doses, depending on the extent of disease.[8] Combination of imipenem and amikacin is recommended for severe cases and central nervous system infections.[7] Other antimicrobials with clinical benefit are extended spectrum cephalosporins, fluoroquinolones (especially moxifloxacin), clindamycin, erythromycin, ampicillin, aminoglycosides (particularly amikacin), tetracycline (including minocycline) and linezolid.[9] Regarding the duration of treatment, 6-12 month is recommended for many cases corresponding to the underlying disease and infectious lesions.[7] Patients with primary cutaneous nocardiosis respond very well to medical therapy. Co-trimoxazole is the mainstay of therapy. Minimal surgical intervention, in the form of draining an acute abscess, is sometimes needed. Clinical response to therapy with co-trimoxazole is evident within a few weeks, but complete clearance may take up to three months. The drug has to be continued for a further three months to prevent recurrence.[10]

Primary cutaneous nocardiosis remains a diagnostic challenge. The majority of cases go unsuspected and undiagnosed because of the non-specific clinical pictures and the difficulties involved in isolation of the organism. Rapid and reliable molecular techniques, though available, are beyond the reach of investigators in many countries. A high degree of clinical suspicion is needed for the diagnosis of the condition along with stringent efforts of the microbiologist to isolate the organism.[10]

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