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Case Report

Nocardia farcinica complicating Cogan's syndrome

Dimos Merinopoulos*, Haroon Khan, Sara Ginwalla, Suzanne Lane and Richard Watts

The Ipswich Hospital NHS Trust, Ipswich, UK

*Correspondence address. The Ipswich Hospital NHS Trust, Heath Road, Ipswich IP4 5PD, UK.
E-mail: dmerinopoulos@yahoo.com

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Nocardiosis is an uncommon bacterial gram-positive infection caused by aerobic actinomycetes in the genus *Nocardia*. Nocardiosis is typically regarded as an opportunistic infection with approximately two-thirds of infected patients being immunocompromised. In this case report we describe a 45-year-old female who presented with a right thigh abscess. She had been taking high-dose prednisolone and ciclosporin for Cogan's syndrome. She presented with erythema and severe pain over her right thigh. Ultrasound showed a collection and *Nocardia farcinica* was isolated. This case report stresses the importance of considering atypical infections in immunocompromised patients, even with minor symptoms, to avoid delay in diagnosis and treatment.

INTRODUCTION

Nocardia species are parasitic bacteria, which grow and reproduce on organic material. Their main habitat is carbon-rich sources such as soil, vegetable matter and aquatic environment [1]. The main mode of infection in humans is aerogenic. The lung is most commonly affected, but *Nocardia* can disseminate haematogenously to the central nervous system (CNS) and also to bone, retina, heart, joints, kidneys and soft tissues. Cutaneous infection is the least common site of infection and presents mainly with abscesses [2, 3]. Cell-mediated immunity is critical in containing Nocardia infection and this makes immunocompromised patients with cell-mediated abnormalities, more susceptible to Nocardia infections [4, 5]. The most common causes are glucocorticoid therapy, malignancy, immunosuppressive therapy, organ transplantation and HIV infection. Although there are more than 80 species in the genus *Nocardia*, only 33 species cause disease in humans [4, 6]. *Nocardia farcinica* is among the most virulent species, since infection is more likely to result in disseminated disease. Nocardia can disseminate from a pulmonary or cutaneous focus to virtually any organ.

CASE REPORT

A 45-year-old Caucasian lady presented following a 3-week history of progressively worsening pain, swelling and erythema in her right thigh. She initially reported the symptoms in clinic when they were minimal with no external signs and an ultrasound was requested. She was taking ciclosporin (2 mg/kg/day) and prednisolone 15 mg/day for Cogan's syndrome. In 2009 she developed recurrent episodes of scleritis and audiovestibular disturbance, leading to a diagnosis of Cogan's syndrome in 2010. She had received prednisolone (60 mg/day), azathioprine, mycophenolate mofetil and cyclophosphamide for her Cogan's syndrome. Other past medical history included autoimmune hepatitis and monoclonal gammopathy of unknown significance. She had had a urine infection, treated with Trimethoprim, a few days before admission and also a recent dental work. She denied any history of trauma or insect bite to the thigh. On examination her right thigh was swollen, erythematous, tender and warm to touch. She was afebrile and systemically well. Her initial blood revealed a WCC 4.8×10^9/l, neutrophils 3.6×10^9/l, CRP 8 mg/l, ESR 61 mm/h. Ultrasonography of her right thigh revealed a 6-cm hypoechoic collection within the right vastus lateralis muscle (Figure 1). The collection was aspirated and she was started on empirical treatment with IV benzylpenicillin 1.2 g tds and IV flucloxacillin 2 g QDS. Ciclosporin was withheld and prednisolone increased to 20 mg/day. Cultures grew branched and beaded Gram-positive rods, suggestive of *Nocardia* species. The antibiotic regimen was therefore changed to IV meropenem 1 g tds and amikacin 7.5 mg/kg bd. Meropenem and amikacin were stopped after 2 days, as the

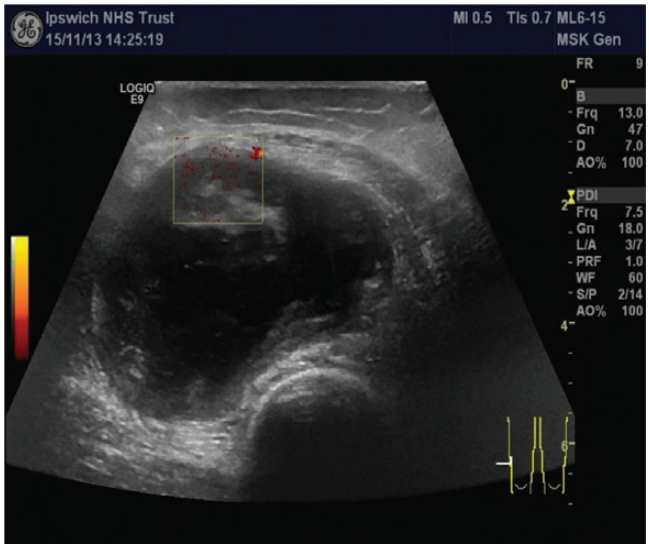

Figure 1: Six-centimetre hypoechoic collection (abscess) within the right vastus lateralis muscle.

patient described symptoms of cough, chest tightness and wheeze. She was commenced on co-trimoxazole 960 mg po tds. However, 4 days later *Nocardia farcinica* sensitive to co-amoxiclav, co-trimoxazole, minocycline, doxycycline and clarithromycin was confirmed and the antibiotic was changed to IV co-amoxiclav 1.2 tds. During her admission the patient remained generally well. A computed tomography (CT) abdomen and pelvis revealed ring-enhancing lesions within the left gluteus muscle. These lesions were attributed to a fall that the patient had in the ward; there were no signs of abscess. A CT head, performed after the patient complained of headache and nausea, was also normal. Both CT scans were undertaken to rule out disseminated Nocardial infection. After 7 days of IV co-amoxiclav the patient was discharged on minocycline 100 mg po bd for 6 months. She developed abnormal liver function and antibiotic therapy was changed to moxifloxacin (400 mg/day). She remained well for the past 6 months taking oral prednisolone.

DISCUSSION

Nocardiosis is an uncommon infection, affecting mainly patients with cell-mediated immunodeficiency [4, 5]. In the USA, there are ~500–1000 new cases each year [7].

The majority of patients with nocardiosis are immunocompromised. In a review of 1050 cases, it was found that 64% of patients with nocardial infections were immunocompromised [5]. The most important risk factors are glucocorticoid therapy, malignancy, organ and hematopoietic stem cell transplantation and HIV infection.

Thirty-three species of *Nocardia* have been shown to cause disease in humans. Members of the *N. asteroides* complex (*N. asteroides sensu stricto*, *N. farcinica* and *N. nova*) are most common worldwide [8]. *Nocardia farcinica*, the identified organism in our case, appears to be among the most virulent species of the *N. asteroids* complex as it is more likely to result in disseminated disease and usually shows high resistance to antibiotics [9]. *Nocardia farcinica* is the second most common species after *N. nova* [9]. The usual antibiotics of choice for *Nocardia* have been co-trimoxazole and amikacin; meropenem can also be used [9].

In our case, meropenem and amikacin, initially used for *Nocardia*, were stopped after the patient developed intolerance to them (unclear which one) and were switched to co-trimoxazole. The patient remained on co-trimoxazole until *Nocardia farcinica* sensitive to co-amoxiclav was identified. Due to the potential side effects of co-trimoxazole and the fact that our patient was systemically well, co-trimoxazole was switched to IV co-amoxiclav. The patient was discharged on minocycline for 6 months. Minocycline was preferred to co-amoxiclav, as the patient would require higher doses than the po 625 mg.

The best data on the sites of nocardial infection come from a 1994 literature review of 1050 cases of nocardiosis [5]. Systemic (>2 sites involved), 32%; pulmonary (only), 39%; CNS (only), 9%; cutaneous or lymphocutaneous, 8%; single site extrapulmonary (e.g. eyes, bone), 12%. Systemic disease due to nocardiosis, especially sepsis, is associated with a high mortality rate of 44–85% [10].

Our patient presented with subcutaneous nocardiosis, progressively worsening pain and erythema over her right thigh. Cutaneous nocardiosis is clinically indistinguishable from lesions produced by common pyogenic bacteria such as *Staphylococcus aureus* and Group A streptococcus. It may present as ulcerations, pyoderma, cellulitis, nodules and subcutaneous abscesses [3]. This case report describes a rather subacute presentation of a *Nocardia* abscess without systemic involvement. The clinical challenge in nocardiosis is that there are no pathognomonic signs or symptoms for it. Therefore, the diagnosis lies on clinical suspicion, which must be high in immunocompromised patients. This case report stresses the importance of considering atypical infections in immunocompromised patients, even with minor symptoms, to avoid delay in diagnosis and treatment.

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