Nocardia farcinica bacteraemia presenting as a prostate abscess

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ARTICLE INFO

Article history:
Received 12 March 2016
Received in revised form 2 June 2016
Accepted 7 June 2016

Keywords:
Prostate abscess
Nocardiosis
Nocardia farcinica
Immunosuppression
Corticosteroids

ABSTRACT

Nocardia is characterised as a Gram positive filamentous rod and is found worldwide in soil, decaying vegetable matter and aquatic environments. Localised pulmonary infection is the most common clinical presentation. However, Nocardia can present in a wide variety of clinical manifestations, especially in the immunocompromised individual. We present the first case of a prostate abscess caused by Nocardia farcinica in a man with a history of severe psoriasis and psoriatic arthritis. He had been on long term immunosuppression for this with prednisolone and etanercept. His Nocardia was likely contracted through direct skin inoculation while gardening with haematological dissemination to the prostate. He responded well to long term sulfamethoxazole and trimethoprim.

Introduction

A prostate abscess caused by Nocardia is rare. We present the first case of a prostate abscess caused by Nocardia farcinica in a man who had been on long term steroids and etanercept for severe psoriasis and psoriatic arthritis.

Case presentation

A 68 year old man presented in March 2015 with a one week history of intermittent dysuria and suprapubic pain. He had a two day history of urinary hesitancy, fever, vomiting, diarrhoea and dyspnoea. Three weeks previously he cut his left middle finger whilst gardening. This became infected and required a one week course of amoxicillin.

He has been heavily immunosuppressed for 30 years for severe psoriasis and psoriatic arthritis. He has been on prednisolone 10 mg daily and etanercept 50 mg weekly for the past 5 years. His previous immunosuppressive agents include methotrexate and leflunomide. His other medical history includes asthma, hypertension, steroid-induced diabetes and atrial fibrillation. There was no history of any prostate surgery or instrumentation.

On admission he looked unwell. His observations were heart rate 167, blood pressure 163/75 mmHg, respiratory rate 22, oxygen saturations 97% on room air and a temperature of 38.2 °C. On examination he had right base crepitations and suprapubic tenderness. Urine analysis was positive for blood. His urine microscopy showed 30 white blood cells but no growth. His chest x-ray showed no consolidation. The C reactive protein was 390 mg/L with a white cell count of 24.9 x 10 to 9/L. He was empirically treated with ceftriaxone, gentamicin and flucloxacillin in the emergency department for sepsis of unknown source. His acute medical unit admission diagnosis was likely urosepsis and prostatitis.

On day 6 his blood cultures grew N. farcinica (taken pre-antibiotics on day 1). The identification was performed by 16s rRNA gene sequencing. Trimethoprim and sulfamethoxazole (TMP-SMX) were additionally commenced. Two further blood cultures taken on day 7 and 10 had no growth. A CT chest and brain found no evidence of Nocardia seeding although a 5 mm soft tissue nodule was noted in the right lower lobe.

Two days into his admission he developed urinary retention requiring catheterisation. A rectal exam revealed a tender prostate. A computed tomography (CT) scan identified "a 3.6 x 3 x 3.4 cm left prostate abscess and a possible sub-centimetre abscess involving the right prostate gland (Fig. 1)". His antibiotics were then changed to intravenous ciprofloxacin 500 mg twice a day.

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He responded to treatment and his inflammatory markers normalised. He was discharged home after 15 days on oral ciprofloxacin and TMP-SMX. The etanercept was held indefinitely and prednisolone weaned back to his normal dose. He has currently been on antibiotics for 8 months and his repeat CT showed his prostate abscess had resolved.

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http://dx.doi.org/10.1016/j.idcr.2016.06.001
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Discussion

Prostate infections are most commonly caused by Gram-negative organisms associated with urinary tract infections or Staphylococcus aureus from haematological dissemination from a primary focus. There are no literature case reports of a prostate abscess caused by Nocardia. There are 7 case reports of epididymo-orchitis and/or testicular abscesses caused by Nocardia and one disseminated Nocardia infection associated with a urinary tract infection. In all cases, except for one, the patients were immunosuppressed with steroids [1–8].

Nocardia is characterised as a Gram positive filamentous rod and is found worldwide in soil, decaying vegetable matter and aquatic environments [9]. It is a slow growing organism and can take up to 7 days to be identified on blood cultures. The genus of Nocardia is rapidly expanding and now contains more than 50 known species. Identification of clinical isolates beyond the genus level is important since Nocardia species differ in the clinical spectrum of the disease they cause and their susceptibility to antibiotics. In particular, N. farcinica tends to be more virulent resulting in disseminated disease and increased resistance to treatment [10–12].

Sixty percent of all reported cases of Nocardia are associated with underlying immunosuppression [10]. The overall incidence of Nocardia in different types of immunosuppressed populations has been estimated to range between 0.4% and 3.6% [10]. In the literature, solid organ transplants are the most common underlying associated condition followed by HIV infection [12]. Minero et al. found that treatment with corticosteroids was the most important risk factor; 62% (N = 23) had received corticosteroids for a median of 3 months with a median dose of 25 mg before developing Nocardia [10].

The main route of acquisition is through direct inhalation of airborne spores or by direct inoculation through the skin. Nocardia involves the lung in 60–70% of cases and skin in 10%. However, it can affect virtually any organ through haematological dissemination, most commonly the central nervous system [10,11]. Nocardia isolation from blood cultures is rare (even with disseminated disease) and has been associated with central venous catheters and prosthetic heart valves, neither of which our patient had [12]. Nocardia bacteraemia carries a high mortality rate of 50–85% [12,13].

Cutaneous Nocardia is characterised by one of two manifestations: primary cutaneous infection via inoculation or disseminated infection with skin involvement. Primary cutaneous infection more commonly affects immunocompetent individuals; the most common isolated species being N. brasiliensis [14]. In our case we presumed he acquired Nocardia through skin inoculation whilst gardening with haematological dissemination to his prostate. Although the most common radiological finding of pulmonary Nocardia is a lung nodule we did not get a biopsy to confirm a tissue diagnosis as the patient was responding to treatment [15].

General treatment recommendations for Nocardia are hindered by lack of prospective controlled trials. TMP-SMX is recommended as an initial empiric regimen since most studies show high rates of susceptibility of Nocardia to this combination [12]. The duration is prolonged to minimise risk of disease relapse; the mean duration of treatment is 6–12 months but should be tailored to the underlying immunosuppression and clinical progression of the patient [15].

We accept that a limitation of this case was that we were unable to obtain a tissue diagnosis. Unfortunately, both the interventional radiology and urology teams were reluctant to perform a transrectal drainage given the high risk of a fistula and further seeding.

The second limitation is that we did not culture the organism from a second set of blood culture and this is because he was empirically started on broad-spectrum antibiotics on admission. Nocardia species in blood culture is rarely considered a contaminant especially in patients presenting with sepsis in the background of profound immunosuppression [16].

Conclusion

Nocardia has a rising incidence due to increasing numbers of immunosuppressed patients and improved methods of detection [12]. Early detection and treatment of patients is clinically important due to its high mortality. This case report serves to highlight the importance of early, pre-antibiotic blood cultures and the necessity for clinicians to consider unusual presentations of opportunistic infections in immunocompromised patients.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Conflict of interests

None.

Support and financial disclosures

None.

Statement of contribution

Dr Hana Scorey: I wrote the original manuscript.
Dr Santhosh Daniel: primary treating infectious disease consultant. He provided clinical guidance and editing of the manuscript.
Both authors have approved the final manuscript.

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