**Nocardia farcinica** as the causative agent in a primary psoas abscess in a previously healthy cattle inspector

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A 42-year-old, previously healthy cattle inspector presented with a 7-day history of fever, a painful left knee, malaise and muscular pain. He did not suffer from an underlying disease, nor was he immunocompromised. After 12 days of hospitalization, a unilocular abscess in the left psoas muscle was diagnosed. *Nocardia farcinica* was isolated from the aspirate. No connection with his work could be demonstrated. The patient was successfully treated with trimethoprim–sulfamethoxazole for 11 months.

**Keywords** *Nocardia farcinica*, infection, psoas abscess

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**INTRODUCTION**

Nocardiosis is a localized or disseminated infection caused by an aerobic actinomycete [1]. The genus is named after Edmond Nocard, who in 1888 isolated an aerobic actinomycete from a case of bovine farcy, later called *Nocardia farcinica*. The first human case of nocardiosis (caused by *Nocardia asteroides*) was reported by Eppinger in 1890. There are at least 12 taxonomically different species within the genus *Nocardia*; *N. asteroides* is the most important etiologic agent in human nocardiosis [1,2]. Since 1975, *N. farcinica* has been recognized as a human pathogen. Cases of human infection with *N. farcinica* are increasingly being diagnosed because of a growing population of immunocompromised hosts and improved methods for detection and identification of *Nocardia* spp. in the clinical laboratory [1,3]. Nocardiosis is chiefly diagnosed in patients with predisposing conditions, although 15% of the patients do not have underlying disease [1,2]. We describe a previously healthy patient with a primary psoas abscess caused by *N. farcinica*.

**CASE REPORT**

A 42-year-old, previously healthy Dutch man presented with a 7-day history of continuing fever (>39 °C), a painful left knee, malaise, muscular pain and anorexia. During an attack of fever, he experienced more pain in his knee. On admission to hospital, he had been using pain medications for 1 week. He did not receive any antibiotics prior to admission. There was no history of visiting a foreign country, or any traumatic event. The patient was working in the veterinary industry as a cattle inspector.

On examination, the patient’s temperature was 38.2 °C. Further general examination did not show any abnormalities. A focus for his fever was not found, and there were no signs of pulmonary or gastrointestinal disease. Physical examination of both lower extremities was normal, including that of the left knee. Laboratory evaluation showed an elevated C-reactive protein (CRP) level of 192 mg/L (<10), leukocytes 25.6 × 10⁹/L (4.0–11.0), steps 1.3 × 10⁹/L (<0.6), polymorphonuclear leukocytes 20.7 × 10⁹/L (2.1–6.7), neutrophils 13.7 × 10⁹/L (2.0–7.5), lymphocytes 0.8 × 10⁹/L (0.7–3.2), monocytes 2.8 × 10⁹/L (0.3–0.9), and erythrocyte sedimentation rate 75 mm/h (<8). The liver enzymes appeared normal: aspartate aminotransferase 19 U/L (<45), alanine aminotransferase 12 U/L (<45), alkaline phosphatase 100 U/L (<120), and gamma glutamyl transferase 39 U/L (<50).

Chest and left knee radiographs showed no abnormalities. Scintigraphic examination of the
skeleton, performed on day 10 after admission, showed only a very slight elevated uptake of the radiomaterial in the skeleton of the left knee. There were no signs of osteomyelitis or of any localized infectious process.

Three sets of blood cultures were drawn on day 1. *Streptococcus mitis* was isolated from one of these samples on the following day. Penicillin (3 × 10^6 IE qid intravenously) was started subsequently. Follow-up blood cultures (two sets on day 5, one set on day 6, and one set on day 12) all remained sterile. A cardiology work-up (trans thoracic echocardiography on day 5 and TEE on day 12) did not reveal any signs of endocarditis. No dental abnormalities, as potential portals of entry for *S. mitis*, were found.

Despite adequate therapy for infection by streptococci (the minimum inhibitory concentration (MIC) of penicillin for the isolated *S. mitis* was 0.032 mg/L), the fever persisted (>39°C). Penicillin was replaced after 3 days by cefuroxime and gentamicin. On day 12, the fever had not subsided, and CRP and leukocyte counts had increased to 201 mg/L and 30 × 10^9/L, respectively.

Ultrasound of the abdomen, which was performed to detect a focus for the fever, showed a mass in the left inguinal region, suggestive of a psoas abscess. On the same day, computed tomography (CT) was performed, and demonstrated a unilocular psoas abscess of dimensions 6 × 9 × 11 cm in the left psoas muscle (Figure 1).

No signs of tuberculous spondylodiscitis or vertebral osteomyelitis were found. Percutaneous drainage of the psoas abscess under CT guidance was performed on the same day. A direct smear from the evacuated pus showed Gram-positive, filamentous rods. Ziehl–Neelsen staining did not show the bacteria to be clearly acid-fast. Modified acid-fast staining was not performed at that time. The aspirate was cultured on standard media for aerobic and anaerobic bacteria from the abdomen, as well as for yeasts and mycobacteria, by standard microbiological methods [4]. Because actinomycosis was suspected, the antimicrobial therapy was changed to amoxicillin (2 g qid intravenously). A Mantoux test (with Purified Protein Derivative) test for tuberculosis was negative. Four days after drainage (day 17 after admission), small orange–yellow colonies appeared in the culture from the pus specimen. No acid-fast bacteria were seen after Ziehl–Neelsen staining. However, Kinyoun staining of the isolated bacteria was positive, as was modified acid-fast staining. The diagnosis was then changed to nocardiosis, and co-trimoxazole (trimethoprim–sulfamethoxazole) at a high dose (10 mg/kg per day of trimethoprim and 50 mg/kg per day of sulfamethoxazole) was started intravenously. The bacteria were finally biochemically determined to be *N. farcinica* [4]. The determination was confirmed by fatty acid analysis and 16S rDNA partial sequencing analysis. The MICs of several antimicrobial agents for the isolated strain, determined with the Etest on IsoSensitest agar with 5% lyzed horse blood, were as follows:

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**Figure 1** CT scan performed 12 days after admission. A large abscess in the left psoas muscle is seen, just before drainage.
trimethoprim–sulfamethoxazole 2 mg/L; amikacin 0.50 mg/L; imipenem 0.25 mg/L; ceftriaxone 3 mg/L; and amoxicillin–clavulanate 0.50 mg/L. Under treatment (drainage and trimethoprim–sulfamethoxazole), the fever subsided totally, and laboratory evaluation showed a decreasing CRP (77 mg/L), as well as decreasing leukocyte count (17.3 × 10^9/L) on day 27.

Immunoscreening was performed in the peripheral blood. No abnormalities were found in B-cell markers (CD19 11% (7–23)), or T-cell markers (CD3 82% (60–85), CD4 41% (29–59), or CD8 35% (19–48)). The CD3−/CD16+CD56+ ratio was 7% (6–29), and the CD4/CD8 ratio was 1.2 (0.6–2.8). An HIV test was not performed.

Repeated CT of the thorax and abdomen did not reveal any other focus of infection. CT or MRI of the brain, to exclude active foci of infection in the brain, was not performed. The knee pain at admission was considered to be arthritic or reactive. After 11 days of drainage, the drain fell out spontaneously. At that time, the patient’s clinical condition had greatly improved. CT showed an evident decrease in size of the abscess (Figure 2), and infection parameters decreased to normal levels.

The patient was discharged from the hospital on day 29, in good clinical condition, on oral trimethoprim–sulfamethoxazole. Ten months after discharge from hospital, he had recovered completely. The infection parameters, white blood cell counts and liver function were all completely within the normal ranges, and the antibiotic treatment was discontinued.

**DISCUSSION**

Primary psoas abscess is an uncommon lesion, usually (80–90%) caused by *Staphylococcus aureus* [5]. Occasional cases due to *Proteus mirabilis, Escherichia coli, streptococci, Pasteurella* spp. and *Mycobacterium xenopi* have been reported [6–9]. *N. farcinica* as the causative agent for a primary psoas abscess, as reported in this paper, is an uncommon finding.

Despite the occurrence of nocardiosis in animals, there is no evidence of respiratory spread from infected animals to humans [1]. Inter-human transmission was documented in a nosocomial outbreak in the USA in 1998 [10]. *N. farcinica* is the classical pathogen of bovine nocardiosis. We found no signs of animal bites or scratches in our patient. Our patient was a cattle inspector, and it is not known if the animals he was working with were suffering from nocardiosis. No cases have been reported in The Netherlands recently, but active screening of veterinary cadavers for nocardiosis is not being performed in The Netherlands.

Although, in the present case, polymicrobial pyomiositis of the psoas muscle with *N. farcinica* and *S. mitis* could not be ruled out, the isolation of *S. mitis* from blood was regarded as not being clinically relevant. The normal TEE, the absence of oropharyngeal foci and the rapid improvement

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**Figure 2** CT scan 11 days after drainage of the psoas abscess. The size of the abscess is diminished dramatically.
of the clinical condition after drainage of the abscess and administration of trimethoprim–sulfamethoxazole made the presence of endocarditis unlikely. Contamination of the culture or a short farnethoxazole made the presence of endocarditis abscess and administration of trimethoprim sulfamethoxazole made the presence of endocarditis

The genus *Nocardi a* consists of Gram-positive, variably acid-fast, strictly aerobic bacteria [11]. In our case, the acid-fast staining on pus was negative. Modified acid-fast staining was not performed at that time. Perhaps the diagnosis could have been established earlier if this test had been performed on the original material. Routine blood cultures are infrequently positive. *Nocardi a* colonies may grow after 48 h of incubation of direct smears from a clinical specimen (i.e. pus from an abscess), but in specimens with a mixed flora (i.e. urine, sputum) the small nocardial colonies are easily obscured by those of the more rapidly growing bacteria [1,3]. Therefore, a sufficiently long incubation period is essential.

The majority (85%) of nocardia infections are seen in immunocompromised patients using immunosuppressive agents, but they may also appear in a previously healthy patient [2]. *N. farcinica* causes a localized or disseminated infection, the latter (28–30%) being especially observed in immunocompromised hosts [2].

There are no optimal antimicrobial regimens that have been established by controlled clinical trials. Clinical experience has shown that successful therapy of nocardiosis involves antimicrobial drug(s) in combination with appropriate drainage or debridement. The introduction of sulfonamides has resulted in substantial improvements in outcome; the mortality of patients with nocardiosis has decreased from 75% to 37% [12]. Trimethoprim–sulfamethoxazole is at present the drug of choice for treatment of nocardiosis, although it has not been established whether treatment with trimethoprim–sulfamethoxazole is superior to treatment with sulfonamides alone [3]. Trimethoprim–sulfamethoxazole is also considered to be the first choice for treatment of infection with *N. farcinica*. *N. farcinica* is characteristically multiply resistant to antimicrobial agents, including third-generation cephalosporins and tobramycin [1,12]. Of concern is the high rate of trimethoprim–sulfamethoxazole resistance in vitro, especially in Europe [2]. For this reason, amikacin combined with imipenem has been proposed by some authors for the treatment of disseminated and central nervous system infections. The optimal dosage and duration of antimicrobial treatment have not been unequivocally determined, but dosage is usually high and (because of high relapse rates) continued for many months. Immunocompetent patients with pulmonary or systemic nocardiosis should be treated for 6–12 months. Immunocompromised patients or patients with brain abscesses should be treated for at least 12 months. Two to four months of treatment is sufficient in primary cutaneous nocardiosis without bone involvement [1].

REFERENCES


