Case Report

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Pulmonary and cutaneous nocardiosis-a case report

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Nubia Bernardes Carvalho
Department of Radiology of Hospital Felício Rocho, Brazil

Correspondence: Nubia Bernardes Carvalho, Department of Radiology of Hospital Felício Rocho, Pós-Graduação Lato Sensu of Fundação Lucas Machado Belo Horizonte, Minas Gerais, Brazil, Email nubia_bc@hotmail.com

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Introduction

Nocardiosis is an acute, subacute, or chronic infectious disease that occurs in cutaneous, pulmonary, and disseminated forms, caused by various aerobic soil saprophytes of the genus Nocardia. *N. asteroides* is the most common human pathogen and *N. brasiliens* is most commonly causes skin infection, particularly in tropical climates. Infection is via inhalation or by direct inoculation of the skin. Person-to-person spread is rare. Nocardiosis occurs worldwide in all age groups, but incidence is higher in older adults, especially men, and immunocompromised patients. The estimated incidence of nocardiosis in the United States is 500-1000 cases per year. Among solid organ transplant recipients, Nocardia infection has a frequency of 0.6% to 3% and has been well described in kidney, heart, and liver recipients. Without treatment, pulmonary nocardiosis and disseminated nocardiosis are usually fatal. Among patients who are treated with appropriate antibiotics, the mortality rate is highest (>50%) in immunocompromised patients with disseminated infections and is about 10% in immunocompetent patients with lesions restricted to the lungs.

Case report

A 63-year-old male kidney transplanted was admitted to hospital with myalgia and weight loss. Neither cough nor chest pain was reported. He had cutaneous lesions in different evolutionary phases (nodules, vesicles, pustules and crusts (Figure 1–3), an abscess on the right arm (Figure 4) and candidiasis on the palate. A chest tomography Figure 5 showed bilateral confluent nodules and pleural thickening. Growth of Nocardia sp was isolated from the secretion of cutaneous lesions. Treatment was started with meropenem and then modified to trimethoprim/ sulfamethoxazole as recommended. However, the patient progressed with worsening renal function, forcing the switch to meropenem. After two weeks, renal function normalized and then treatment with a lower dose of trimethoprim/sulfamethoxazole returned and was mantained for at least six months.

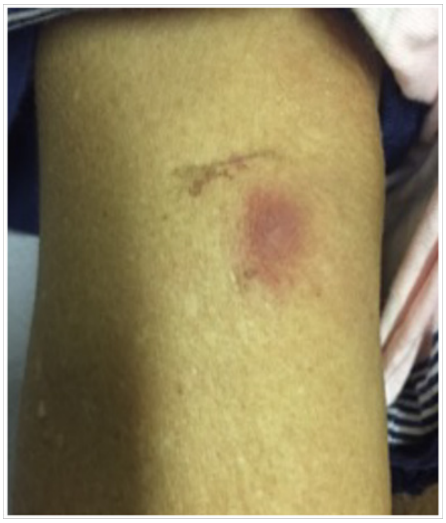

Figure 1 Nodule.

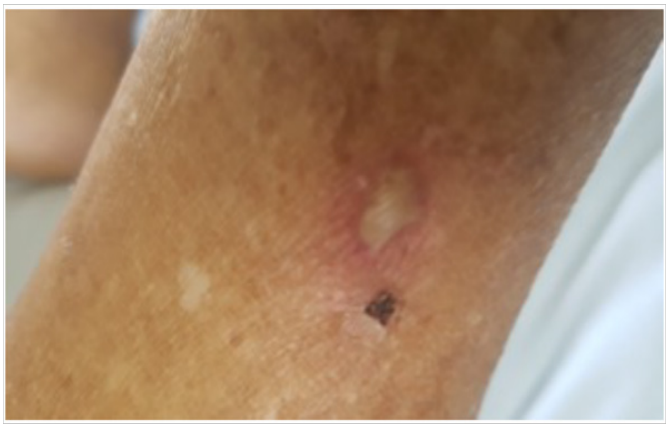

Figure 2 Pustules.

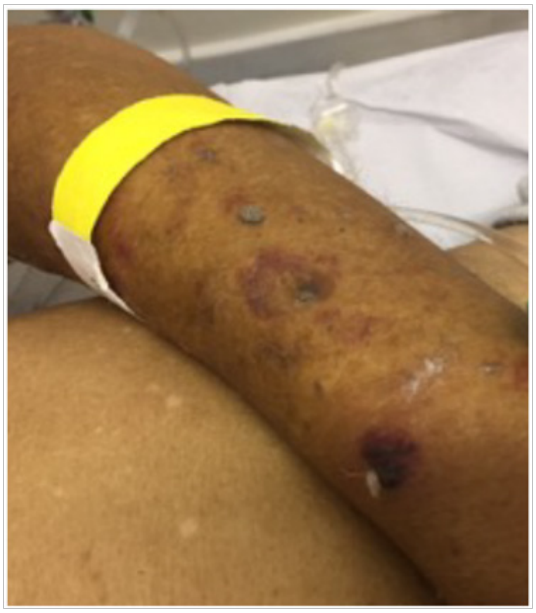

Figure 3 Crusts.

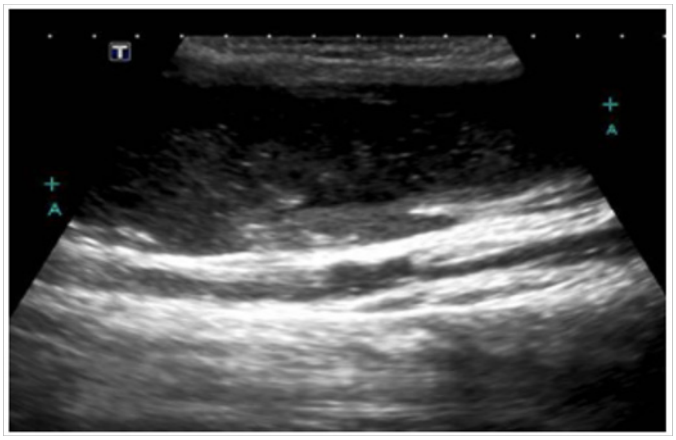

Figure 4 Abscess on the right arm on the ultrasound.

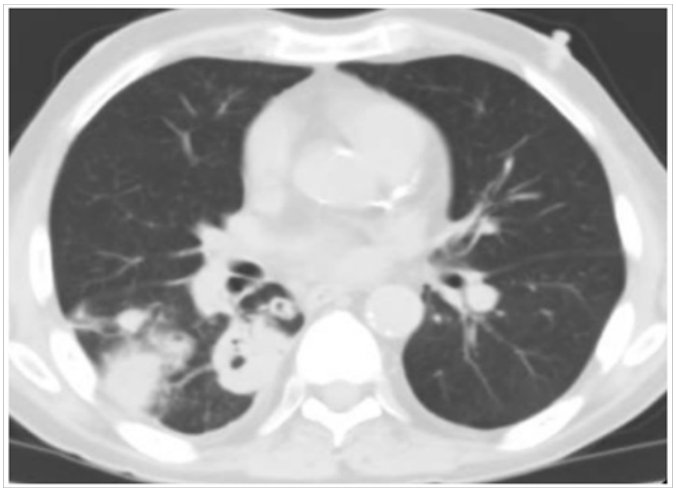

Figure 5 Chest tomography with bilateral confluent nodules.

Discussion

Pulmonary nocardiosis is the most common clinical presentation of infection because inhalation is the primary route of bacterial exposure. The onset of symptoms may be subacute to more chronic and can include productive or nonproductive cough, shortness of breath, chest pain, hemoptysis, fever, night sweats, weight loss, and progressive fatigue. Pleural effusion may also occur. Primary cutaneous nocardiosis can result from traumatic injury to the skin that involves contamination with soil and manifests as cutaneous infection (cellulitis or abscess), lymphocutaneous infection (sporotrichoid nocardiosis), or subcutaneous infection (actinomycetoma). Unlike other forms of nocardiosis, primary cutaneous disease usually develops in immunocompetent hosts.

The lymphocutaneous syndrome consists of a primary pyoderma lesion and lymphatic nodules resembling sporotrichosis and an actinomycetoma begins as a nodule, suppurates, spreads along fascial planes, and drains through chronic fistulas. And disseminated nocardiosis may involve any organ; lesions in the brain or meninges are most common. Patients may have one or more brain abscesses and present with headache, nausea, vomiting, seizures, or alteration in consciousness. Diagnosis is by identification of Nocardia sp in tissue or in culture of samples from localized lesions identified by physical examination, x-ray, or other imaging studies. Clumps of beaded, branching filaments of gram-positive bacteria are often seen.

The treatment is with trimethoprim/sulfamethoxazole (TMP/SMX) 10mg/kg/day (of the trimethoprim component). In immunocompromised patients and patients with disseminated disease, TMP/SMX should be used with amikacin, imipenem, or meropenem pending species identification and susceptibility testing results because adverse reactions to high-dose TMP-SMX therapy are frequent and include myelosuppression, hepatoxicity, and renal insufficiency. When sulfonamide hypersensitivity or refractory infection is present, amikacin, a tetracycline (particularly minocycline), imipenem/cilastatin, meropenem, ceftriaxone, cefotaxime, extended-spectrum fluoroquinolones, dapsone, or cycloserine can be used. In vitro susceptibility data should guide the choice of alternative drugs. Sometimes abscesses or wound infections need to be surgically drained. Combination therapy should continue until clinical patient improvement occurs and Nocardia species identification and antimicrobial drug susceptibility information can be confirmed. Duration of treatment is generally prolonged to minimize risk of disease relapse. Immunocompetent patients with pulmonary or multifocal (non-CNS) nocardiosis may be successfully treated with 6 to 12 months of antimicrobial therapy. Immunosuppressed patients and those with CNS disease should receive at least 12 months of antimicrobial therapy with the appropriate clinical monitoring.

Immunosuppressive therapy predisposes patients to Nocardia infection. In patients who are on these drugs because they have undergone transplantation, immunosuppression cannot be stopped, but the dose should be decreased as much as possible. In patients receiving immunosuppressive therapy for other reasons, it is ideal to discontinue the immunosuppressive agent if alternatives are available. Trimethoprim-sulfamethoxazole provides effective prophylaxis to prevent Pneumocystis pneumonia and also can decrease the risk of nocardial infections. Daily TMP-SMX prophylaxis most reliably prevents nocardiosis and may also account for the decreased prevalence of nocardiosis in patients with advanced human immunodeficiency virus infection.

Conclusion

Increases in the number of patients receiving immunosuppressive therapies for solid organ or hematopoietic stem cell transplants, hematologic and solid tissue cancers, and autoinflammatory conditions, ensure that Nocardia will remain a formidable pathogen. Although this organism is capable of producing serious and metastatic disease in the appropriate host, early recognition and initiation of appropriate treatment can lead to successful outcomes. Cure rates for patients with skin infection are usually >95%. Ninety percent of pleuropulmonary infections can be cured with appropriate therapy.

Acknowledgements

None.

Conflict of interest

The author declares that there is no conflict of interest.

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