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Disseminated Nocardiosis in a Young Female Immunosuppressed for Systemic Lupus Erythematosus with Successful Treatment – A Case Report

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Abstract

Nocardiosis is a rare disease caused by aerobic actinomycetes of the genus *Nocardia*. Infections caused by nocardia species are infrequent, but challenging to clinicians to handle it. Though it is common in immunocompromised individuals, few cases were reported in immunocompetent individuals as well. We report a case of disseminated nocardiosis caused by *Nocardia cyriacigeorgica* in a patient with systemic lupus erythematosus secondary to non-tapering of oral corticosteroids. This case emphasizes high index of clinical suspicion of nocardiosis in immunocompromised host and the importance of tapering oral corticosteroids during maintenance phase while treating rheumatological diseases.

Keywords: Nocardiosis, systemic lupus erythematosus, Immunocompromised host

Introduction

Nocardiosis is an acute, subacute, or chronic suppurative infection caused by *Nocardia* that occurs in cutaneous, pulmonary, and disseminated forms. Primary cutaneous nocardiosis manifests as cutaneous infection (cellulitis or abscess), lymphocutaneous infection (sporotrichoid nocardiosis), or subcutaneous infection (actinomycetoma). Pleuropulmonary nocardiosis manifests as an acute, subacute, or chronic pneumonitis, usually in immunocompromised hosts, although isolated cases have been reported in immunocompetent hosts. Disseminated nocardiosis may involve any organ; lesions in the brain or meninges are most commonly involved.

Case Report

We report a 23 year old female who was a known case of Systemic lupus erythematosus on treatment with oral steroids, presenting with the complaints of swelling in the left side of thorax for 7 days duration. It was gradually increasing in size and was associated with throbbing pain. It was associated with high grade intermittent fever with chills and rigors and dry cough. She had past history of lupus pneumonitis, lupus pancreatitis and SLE related lung injury and all of which was successfully treated. On examination, she had a large swelling of 15*17 cm size in left inframammary region extending up to left infrascapular region.

CT Chest and Abdomen showed multifocal areas of pleural based consolidation in left upper and lower lobes, large hypodense collection measuring 6*4.2 cm in left subdiaphragmatic region indenting on segment 3 of liver and similar large hypodense lesions in left lateral

abdominal wall and anterior ends of last 4 ribs. The above collection extended into the peritoneal space with thickening of peritoneum. CT Abdomen with contrast revealed peripherally enhancing multiseptated collection - abscess on either side of left lower lobe.

Figure 1:

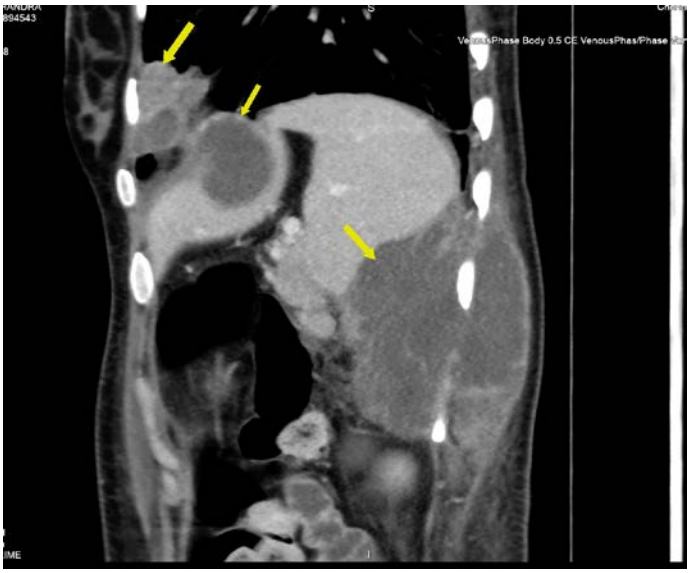

Figure 1: CECT Abdomen showing collection in left lateral abdominal wall and another collection noted indenting the left lobe of liver.

Laparoscopic approach of abscess drainage was considered. Omental adhesion with left perisplenic diaphragm was divided and purulent collection was drained. Another purulent collection in parietal wall was identified and drained. Thorough wash was given into abscess cavity and peritoneal cavity. Drain was placed into abscess cavity. Microbiology Lab was alerted at the possibility of opportunistic infections including Nocardia, due to the clinical presentation. Pus culture grew Nocardia cyriacigeorgica which was resistant to imipenem and cotrimoxazole. Hence she was treated with ceftriaxone and amikacin based on sensitivity. She had another swelling in left loin with abscess cavity tracking from thoracic spine extending along intercostal spaces for which incision and drainage was done. She responded well to the treatment and was discharged in stable condition. Retrospectively upon further probing, patient said that she did not taper the dose of oral steroids as advised by the Rheumatologist, which possibly resulted in opportunistic infection for her in the form of nocardiosis.

Discussion

The genus Nocardia was named after Edward Nocard who in 1888 described the isolation of an aerobic actinomycete from cattle with bovine farcy. Epinger reported the first human case of nocardiosis in 1890. They are aerobic, filamentous, gram positive, weakly acid fast organisms that live as soil saphrophytes. The taxonomy has been challenging and likely remains in evolution. [1, 2]

The species called *Nocardia asteroides* was previously reported to be the most common cause of human disease. Among more than 85 identified species of *Nocardia,* approximately 25 species are associated with human infections and include *Nocardia asteroides* complex (more than 50% human cases), *N. brasiliensis, N. abscessus, N. cyriacigeorgica, N. farcinica, N. nova, N. transvalensis complex, N. nova*complex, *N. pseudobrasiliensis,* and the recently reported *Nocardia veteran and N. cerradoensis* [3-4]. *Nocardia cyriacigeorgica,* is prominently associated with pulmonary infection and poor outcomes.[5)]

In addition to neutrophils and macrophages, cell-mediated and humoral immunities play roles in protecting the host against *Nocardia* invasion, explaining the wide range of immunocompromised patients who are at increased risk for contracting nocardiosis.

Pulmonary disease is the predominant clinical finding in most patients with nocardiosis. [6,7)] . Cough with sputum production and fever are the dominant symptoms. At least 40% of patients with disseminated nocardiosis have

pulmonary infection; therefore, the clinical presentation may be dominated by the pulmonary symptoms. Clinical manifestations include inflammatory endobronchial masses or localized or diffuse pneumonias, which may be accompanied by cavitation, abscess formation, pleural effusion, or empyema. Local findings associated with metastatic abscesses may be present at almost any site but are typical in the lower extremities. Symptoms in patients with nocardiosis are indistinguishable from those in patients with similar pulmonary infections of other microbial etiology, such as TB. The diagnosis of nocardiosis is established with culture of the causative organism from the infection site(s). Because nocardiae grow slower than common bacteria, the microbiology laboratory should always be notified when nocardiosis is clinically suspected.

Sulfonamides have long been the first-line antimicrobial therapy for nocardiosis. Trimethoprim-sulfamethoxazole (TMP-SMZ) is considered the therapy of choice by most authorities. Divided doses of 5-10 mg/kg/d of the trimethoprim component should be administered to produce sulfonamide levels of 100-150 mcg/mL. Additional or alternative parenteral therapies include carbapenems (imipenem or meropenem, but not ertapenem), third-generation cephalosporins (cefotaxime or ceftriaxone), and amikacin, alone or in combination [(1)].

Nocardia cyriacigeorgica was first reported as Nocardia cyriacigeorgici from a patient with chronic bronchitis in 2001. [10] In the recent times, with the advent of newer molecular diagnostic techniques such as 16S rRNA gene or matrix-assisted laser desorption/ionization time of fight (MALDI TOF), invasive infections by Nocardia Cyriacigeorgica are being increasingly reported. Review of literature showed that among 765 Nocardia isolates submitted to the United States Centers for Disease Control and Prevention between 1995 and 2004, 13% were due to Nocardia cyriacigeorgica. [11] There are case reports of septicemia, disseminated infection as well as infective endocarditis due to Nocardia cyriacigeorgica.[12]

Nocardiosis has a variable prognosis, depending on the site of infection, extent of infection, and underlying host factors [8]. It has been noted that a delay in diagnosis among immunosuppressed patients infected with *Nocardia* may be responsible for treatment failure and poor prognosis [9].

Conclusion

Nocardiosis is often overlooked since the presentation is insidious and nonspecific and often lacks the clinical findings and laboratory evidence of bacterial infection. This case is being reported in the view of its rarity of presentation and the importance of tapering steroid therapy during treatment of maintenance phase of any rheumatological condition. Despite medical advice as in our case, the patient did not taper the dose, which puts us in a situation of stringent monitoring of these fragile patients and throw lights on the importance of regular periodic follow-up. As clinical presentation it can mimic any other infection like tuberculosis, clinicians should have a high index of suspicion for nocardiosis in immunocompromised host. Hence early diagnosis and treatment can result in better outcome.

Conflicts of interest

There are no conflicts of interest

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