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Pulmonary Nocardiosis Presenting as Bilateral Round Pneumonia in a Patient Having Idiopathic Thrombocytopenic Purpura

A Case Report & Review of the Subject

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ABSTRACT

Nocardiosis is an uncommon Gram positive bacterial infection having predilection for lung and central nervous system. It is typically regarded as an opportunistic infection, but approximately one-third of infected patients are immunocompetent. This patient while using corticosteroids for immune thrombocytopenic purpura developed fever and respiratory symptoms consistent with lower respiratory tract infection. Her chest radiograph revealed bilateral atypical perihilar rounded shadows which on bacteriological evaluation of bronchial washings turned out to be nocardia pneumonia that responded well to appropriate treatment.

INTRODUCTION

Nocardia species are found worldwide in soil, decaying vegetable matter, and aquatic environments, and can become airborne particularly on dust particles. Inhalation of the organism is considered to be the most frequent mode of entry, which is supported by the observation that the majority of infections involve the lungs. Radiologically, the presentation can be with lobar consolidation, reticulonodular infiltrates, interstitial infiltrates, including some of the atypical presentations like single or multiple nodules, lung masses, subpleural plaques, and pleural effusions. Lab isolation of nocardia requires Gram staining and standard blood culture media requiring prolonged incubation. Treatment consist of an induction phase with two to three antibiotics according to drug susceptibility pattern and an oral regimen mostly treated with cotrimoxazole for a prolonged duration from six months to one year.

CASE REPORT

A 50 years old widow lady resident of Lahore was hospitalized through emergency department with one month history of low grade continuous fever along with cough productive of mucopurulent sputum and dyspnea on mild to moderate exertion having 10 days duration. These symptoms were also associated with easy fatigability and loss of appetite accompanying 10 kg weight loss. Three months ago, she underwent surgery due to right para-umblical hernia in a private centre that was complicated by cardiogenic shock and she was referred to Punjab institute of cardiology where further work up including angiography revealed ischemic heart disease (triple vessel disease) requiring surgery that was postponed due to discovery of her low platelet counts. Work up for dengue fever was negative and her bone marrow biopsy was suggestive of idiopathic thrombocytopenic purpura (ITP). During her hospitalization, she received blood and platelet transfusions and received rituximab infusion once and prednisolone 60 mg/day for 2 weeks tapering to 30 mg/day by her private physician. She developed diabetes mellitus after 3 weeks as well as the above mentioned presenting complaints after 2 months of corticosteroids treatment. She was a non smoker, kept no pet bird or animals at home and had no history of a job with any chemical exposure including insignificant family, social and menstrual

history.

On physical examination, she was fully oriented having puffy face and appeared lethargic and pale. Her pulse was 98/min, BP 120/70 mmHg, oral temperature 99°F and respirations were regular and 22/minute with SpO_2 95% on room air. There was no cyanosis, peripheral lymphadenopathy or thyromegaly and her JVP was not raised. Respiratory system evaluation was consistent with bronchial breathing, coarse crackles, increased vocal resonance & impaired percussion note on right middle & lower and left middle parts of the chest with bilateral occasional wheezes. Skin examination showed no purpura bruises or rashes. Her cardiovascular, central nervous, gastrointestinal and musculoskeletal systems revealed no abnormality on physical examination. Chest radiograph showed bilateral perihilar rounded shadows with the right sided slightly larger shadow having a partial air fluid level and the left one showing smaller cavities (Fig. 1).

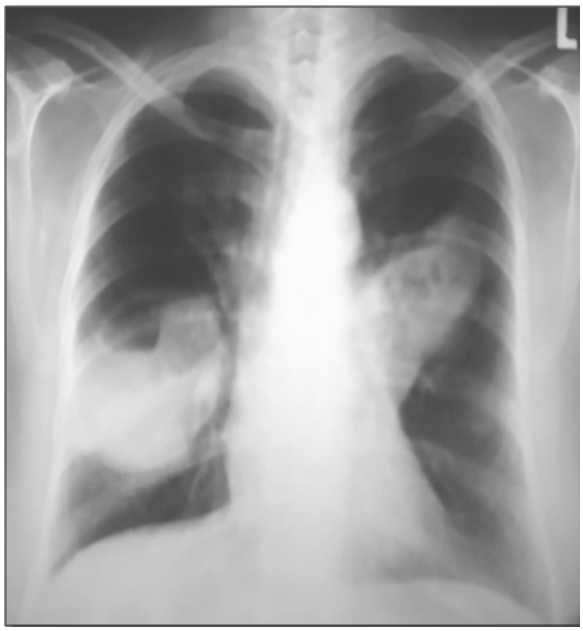

Fig. 1: Bilateral perihilar round air space (pneumonic) shadows.

She was hospitalized for further work up. Lab results showed: complete blood count (CBC): Hb 10.5 g/l, WBC: 7.29, platelets 88000/cmm, DLC: N 92%, M 2%, L 4%, E 1%, ESR 80 mm, BUN 17 mg/dl, Creatinine 0.8 mg/dl, ALT 41 U/L, AST 56 U/L, alkaline phosphatase 78 U/L, bilirubin 0.32 mg/dl, Na^+ 138 mmol/l, K^+ 3.0 mmol/l and random blood glucose 252 mg/dl. Blood gas analysis on room air showed pH 7.38 PCO_2 38 mmHg, PO_2 84 mmHg and HCO_3 28. Prior to this hospitalization, she took two courses of broad spectrum antibiotics including cefipime and ciprofloxacin without any relief in symptoms. Her blood culture was sent and she underwent HRCT chest that showed bilateral rounded areas of consolidation involving left upper and right lower zones (Fig. 2).

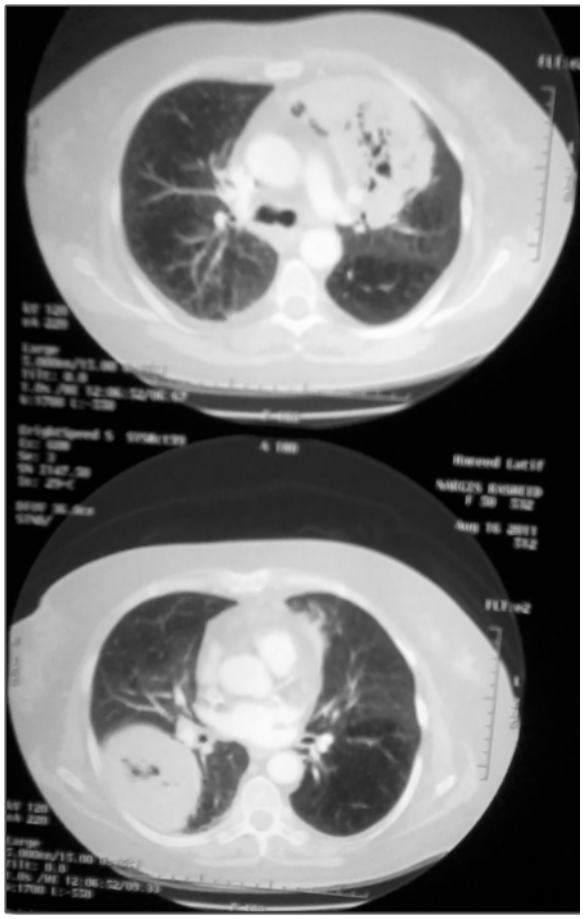

Fig. 2: CT chest (contrast) showing left upper lobe & right lower lobar consolidation with cavitation.

Her bronchoscopy showed generalized hyperemia of airways and purulent secretions bilaterally which were sent for microbiology. Staining of bronchial washing showed numerous

Gram positive branching rods suggestive of nocardia that on quantitative culture showed >10,000 CFU/ml of nocardia species. Fungal & mycobacterial staining (ZN) were negative and blood culture revealed no growth after 72 hours. She was finally diagnosed to have bilateral nocardia pneumonia as a consequence of immunosuppression and was given a combination of meropenem, gentamycin and co- trimoxazole according to the antibiogram for 2 weeks along with her other medications including prednisolone 10 mg daily, omeprazole 20 mg, losartan 50 mg daily and insulin for diabetic control. She had improvement in her clinical symptoms and mobility and her follow up chest radiograph showed bilateral reduction in size of the consolidation (Fig. 3).

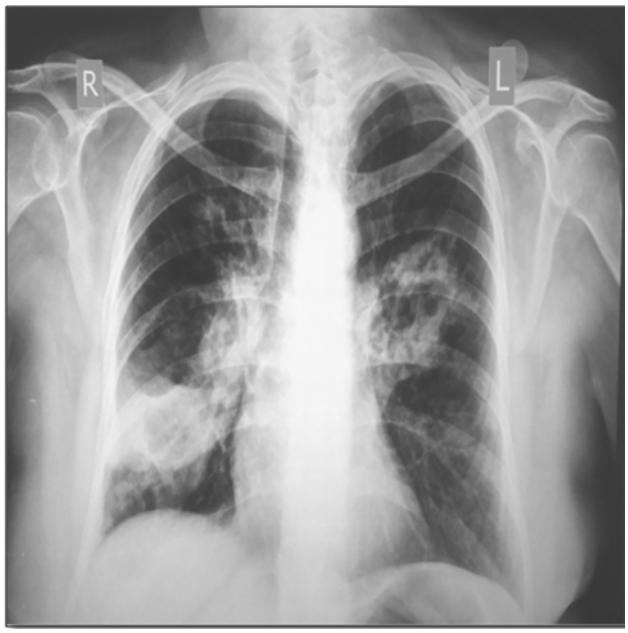

Fig. 3: Partial resolution of bilateral perihilar round air space (pneumonic) shadows.

She was discharged after 2 weeks of parenteral treatment and was advised to continue oral cotrimoxazole (2 double strength tablets thrice daily, trimethoprim 10 mg/kg) with monitoring of CBC, renal chemistry and electrolytes 4 weekly. Her further regular follow up visits at 1 and 3 months showed sustained improvement in her clinical and radiological parameters and she tolerated the regimen well. The treatment was stopped after 6 months when her chest radiograph was completely clear (Fig. 4).

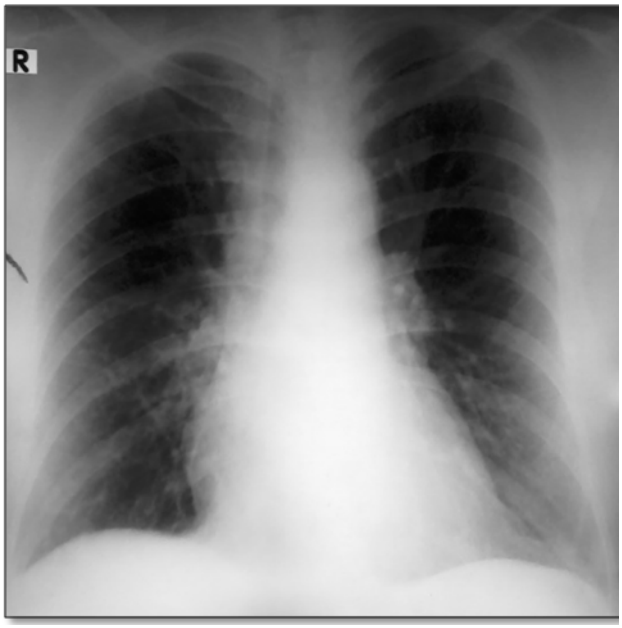

Fig. 4: CXR-PA at end of treatment showing complete resolution of radiologic shadows.

DISCUSSION

The risk of nocardial infection is increased in immunocompromised patients, particularly those with defects in cell-mediated immunity.[1] The etiologies of immunocompromise have included HIV infection, solid organ or hematopoietic stem cell transplantation, malignancy, diabetes mellitus and glucocorticoid therapy.[2] Nocardia species are not normally found in the respiratory tract as flora and its isolation from a respiratory specimen is almost always indicative of infection. Most pulmonary infections are primary but nocardia can spread to the lung from other sites, such as the skin.[3] The clinical presentation of pulmonary nocardiosis may be acute, subacute, or chronic and is not distinguished by any specific signs or symptoms. Fever, night sweats, fatigue, anorexia, weight loss, dyspnea, cough, hemoptysis, and pleuritic chest pain can occur together or in isolation.[4] In addition, complications such as empyema, mediastinitis, pericarditis, and superior vena cava syndrome can occur following contiguous spread of nocardial

infection from a pulmonary, pleural, or cutaneous focus.[5] The mean time from the development of symptoms to diagnosis has, in different studies, ranged from 42 days to 12 months.[2, 6] The difficulty in establishing a diagnosis of nocardiosis may be related to the inadequacy of specimens obtained by noninvasive means and usually invasive procedures are required to establish the diagnosis of nocardiosis.[6] In the appropriate clinical setting, a presumptive diagnosis of nocardiosis can be made if partially acid-fast filamentous branching rods are visualized in clinical specimens.[7] Most routine aerobic bacterial, fungal, and mycobacterial culture media can support nocardia growth but, selective media, such as buffered charcoal yeast extract and modified Thayer-Martin agar may be beneficial.[8] In routine aerobic cultures, nocardia species have variable colonial morphology, from chalky white to pigment-producing orange, yellow, or brown colonies, and usually require 5 to 21 days for growth.[9, 10] Since most routine fluid or tissue cultures are discarded at 48 to 72 hours, laboratory personnel must be notified when nocardiosis is suspected in order to ensure an adequate incubation period.[10,11] The radiographic appearances of nocardia infection are diverse varying from a small nodule to bilateral infiltrates with cavitation.[12] Clinical isolates of nocardia species are variably resistant to antibiotics. As a result, it is recommended giving empiric coverage with two or three agents in patients with severe infection.[9] Patients with severe infection that does not involve the CNS be treated initially with trimethoprim-sulfamethoxazole/TMP-SMX (15 mg/kg IV of the trimethoprim component per day in two to four divided doses) plus amikacin (7.5 mg/kg IV every 12 hours). An alternative approach that some clinicians favor for the initial treatment of severe disease is imipenem (500 mg IV every 6 hours) plus amikacin.[13] Recommended antibiotics that can be part of an oral regimen following induction 2-3 weeks intravenous therapy include either a sulfonamide (eg, TMP-SMX 10 mg/kg of the trimethoprim component per day in 2-3 divided doses) and/or minocycline (100 mg twice daily) and/or amoxicillin-clavulanate (875 mg twice daily.[1,14] The optimal duration of antimicrobial treatment for severe disease has not been determined, but most authorities recommend to treat serious pulmonary infection for 6 to 12 months or longer. All immunocompromised patients (except those with isolated cutaneous infection) as well as patients with CNS involvement should be treated for at least one year. [15]

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