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Case Report

Multiple nodules accompanied by pulmonary nocardiosis in a patient with systemic lupus erythematosus

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ABSTRACT

Nocardia a common saprophyte of soil is an opportunistic pathogen that may cause invasive and disseminated infections in patients with compromised immune function. Pulmonary nocardiosis is one of the common invasive infections which may pursue a perilous course in patients with underlying chronic lung disease and patients on long term steroids. Any evidence of the presence of this organism even in potentially contaminated sample like sputum deserves due cognizance because of rare occurrence of *Nocardia* as laboratory contaminants. Identification and institution of appropriate therapy is absolutely critical to the positive clinical outcome of this otherwise fatal infection.

Keywords: Immunosuppressive therapy, *Nocardia*, Pulmonary nocardiosis

INTRODUCTION

Genus *Nocardia* includes gram positive aerobic bacteria that grow as branching filamentous forms.[1] Acid fastness is due to the presence of mycolic acids in the cell wall. *Nocardia* are saprophytic and can be found and recovered from soil, decomposing organic matter, fresh and salinated water etc.[2] Taxonomy of *Nocardia* have undergone several revisions and currently more than 85 species using 16s RNA typing have been identified with at least 30 species associated with human infections. More than 50% of human infections are attributable to *N. asteroides* complex. Other species of importance includes *N. brasiliensis*, *N. otitidiscaviarum*, *N. farcinia*, and *N. transvalensis* complex.[3] *N. cyriacigeorgica* is also being increasingly isolated. *N. asteroides* complex remains the most important cause of pulmonary infections.

Nocardia being an opportunist pathogen causing majority of infections in patients with compromised immune function though it can attack immune competent patients also.[4,5]

We report here a case of pulmonary nocardiosis in a female patient of systemic lupus erythematosus who was on long term oral steroids.

CASE REPORT

The patient is a 37-year-old woman who had been diagnosed with systemic lupus erythematosus (SLE) along with lupus nephritis 6 years earlier and she was on immunosuppressant drugs from 3 months.

This patient presented to our hospital with symptoms of left sided chest pain, cough with expectoration, breathlessness and fever for one month. Physical examination upon admission revealed tachypnoea (36 breaths/min), temperature of 38.2ºC, blood pressure of 170/80 mm Hg, and heart rate of 130 bpm. Auscultation revealed diminished breath sounds at the base of the left

lung. Results of arterial blood gas analysis showed a pH of 7.47, PaCO2 of 32 mm Hg, PaO_2 of 38 mm Hg, HCO_3 of 23.3 mEq/L.

The hemogram revealed marked leukocytosis (18,000/mL, 92% PMN cells), hemoglobin 5.7 g/dL and a platelet count 1.8 lac/mm^3. Biochemical parameters signaled evidence of chronic renal disease (creatinine 2.3 mg/dL, urea 95 mg/dL), and urinary protein of 2.5 gm/day. Serum sodium and potassium were 137 mmol/L and 2.2 mmol/L respectively. Serum LDH was 895 U/L. Other parameters were normal.

The chest radiograph revealed multiple nodules in left lung field and left pleural effusion. These findings were confirmed by computed tomography which showed fibrotic changes in left upper lobe, multiple subpleural and perilymphatic nodules in left upper lobe and along major fissure respectively, left sided pleural effusion with underlying collapsed lung segment and mild pericardial effusion. An initial attempt at ventilation with continuous positive airway pressure support failed to change the clinical picture or arterial blood gas results, indicative of severe respiratory insufficiency. Therapeutic pleural tap was done and pleural fluid was sent for ADA and cytology. Level of ADA in pleural fluid was 5.69 and in cytology 300-350 W.B.C/mm^3 seen, out of which 80% were PMN and 20% were lymphocytes. Pleural fluid Sugar and protein were 71 mg/dl, 1.8 gm/dl respectively. Gene expert for pleural fluid was negative.

Empirical treatment with cefixime and azithromycin was started, and the corticosteroid and mycophenolate mofetil (MMF) prescribed for the underlying disease, were continued.

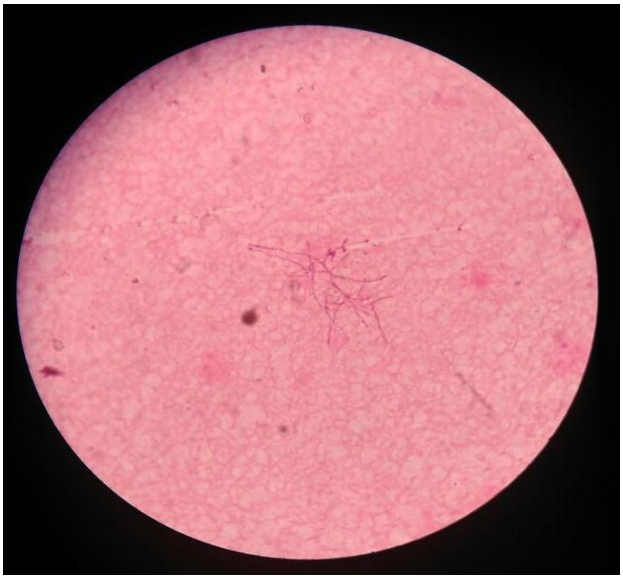

Figure 1: Gram stain showing gram positive.

Direct examination of the pleural fluid showed gram-positive filamentous bacilli on gram stain and branching acid-fast bacilli on modified Ziehl Neelson stain suggestive of *Nocardia* species. On Lowenstein Jensen media, orange coloured colonies were evident at 7 days, which were chalky in appearance.

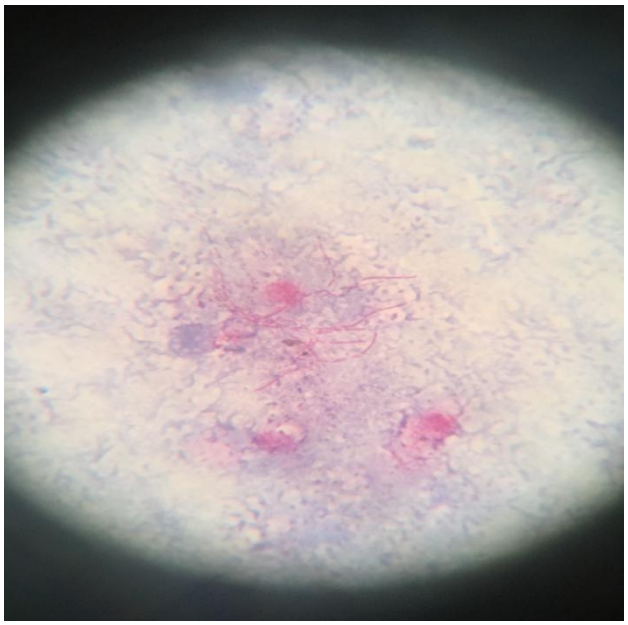

Figure 2: Ziehl Neelsen stain showing acid fast filamentous bacilli branching bacilli.

Figure 3: Colonies of *Nocardia* asteroides on Lowenstein Jensen media.

Species identification was done by using a battery of tests, including hydrolysis of adenine, casein, tyrosine, xanthine, hypoxanthine, esculin, production of arylsulftase, nitrate reductase, urease and utilization of acetamide, citrate. On the basis of biochemicals, isolate was identified as *Nocardia* asteroids.

After the diagnosis of nocardiosis was made, she was started on 15 mg/kg of daily cotrimoxazole in three divided doses and imipenem 750 mg I/V once daily. She had a dramatic response to antibiotics and was discharged with maintenance dose of cotrimoxazole with instruction to come for follow up after three months.

DISCUSSION

The present case draws attention to the formidable pathogenic potential of *Nocardia* in ever enlarging pool of patients with compromised immune function or those on immunosuppressive therapy.[6-8] Pulmonary nocardiosis is the most common clinical presentation presumably because inhalation remains the primary route of exposure. The patients usually presents with features of productive or non-productive cough chest pain, dysnoea, hemoptysis etc. Radiological findings range from local to multifocal disease with nodular or cavitary lesions. About one third of patients may develop pleural effusion.

This patient was an old diagnosed case of lupus compatible with possibility of *Nocardia* etiology but this patient was aggressively investigated for evidence of tubercular and fungal infection because clinically it may be difficult to differentiate *Nocardia* from fungal or mycobacterial disease. Diagnosis of nocardiosis was accidental finding in Gram stain of the aspirated pleural tap sample which was corroborated in modified acid fast staining and confirmed in culture. This case is a glaring reflection of lack of awareness about the potential role of pathogens like *Nocardia* in patients on immunosuppressive drugs.[9]

Nocardia usually takes about three to five days to grow and may not even grow in contaminated samples like sputum.[10] These shortcomings and difficulties inherent to the culture systems reinforce and uphold the role of basic investigations like gram and acid fast stain in providing presumptive signals of the presence of the organism and suggesting further targeted work up of the sample.

Nocardia species are non-fastidious and can grow on media as simple as nutrient agar, blood agar or Saboraud's agar but media with selective characters like LJ medium, provide the advantage of selecting out *Nocardia* species even from a potentially contaminated samples.

Identification of *Nocardia* species with the conventional biochemical system has limited applications. Molecular techniques are not only rapid but also aid in providing signals to the presence of novel species. Since the availability of molecular techniques is restricted to a couple of special facility labs only, the application of this molecular techniques is restricted to a couple of special facility labs only, the application of this modality has little favor and advantage for general microbiology laboratories.

The management of *Nocardia* infections is mainly empirical because the results of antimicrobial sensitivity are inconsistent and lack reproducibility. Sulfonamides, ceftriaxone, amikacin, linezolid, imipenam, minocycline etc. are some of the common drugs chosen for treatment.[9,10] This patient was started on 15 mg/kg of cotrimoxazole and imipenem and the patient responded extremely well to this strategy and at the time of discharge, there was complete mitigation of respiratory features indicating this pathogen has the capability to cause serious troubling inflictions and that early recognition and institution of appropriate management strategies can have wonderful outcomes.

CONCLUSION

There is certainly under diagnosis and reporting of Pulmonary nocardiosis cases possibly because clinicians are not acutely vigilant to the possibility of these infections and most of the microbiology laboratories lack necessary competence to pick up this organism unless specific requests for this organism are referred. Hence patients with compromised immune function, some underlying lung pathology and on prolonged steroids need to be on diligent surveillance of respiratory physicians for the possibility of *Nocardia* infection.

Sulphonamides are a natural choice of drugs but most of the patients report late with serious infection and the escalation of treatment to amikacin plus ceftriaxone or imipenem becomes absolutely essential.[9]

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