CASE REPORT

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Disseminated Nocardiosis Masquerading as Metastatic Malignancy

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ABSTRACT

Nocardiosis is an uncommon Gram-Positive bacterial infection caused by aerobic Actinomycetes of the genus Nocardia. It can be localized or systemic and is regarded as an opportunistic infection that is commonly seen in immunocompromised patients. We report a case of disseminated Nocardiosis in a patient on immunosuppressant therapy in whom the clinical presentation was highly suggestive of a metastatic disease.

Keywords: Disseminated Nocardiosis, Metastatic disease, Actinomycetes

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INTRODUCTION

Nocardiosis is a disease caused by Gram positive, filamentous, strictly aerobic bacteria from genus Nocardia.[1] Nocardiosis can be localized or widely disseminated infection with varying progression from Acute, subacute or chronic disease. It spreads through pulmonary or cutaneous route. Chronic disseminated pulmonary infections often mimic presentation of Tuberculosis or malignancy.[9,10]

Involvement of two non contagious sites constitutes disseminated Nocardiosis. Nocardia possesses the ability to invade virtually any organ. CNS Involvement, particularly Nocardial brain abscess can occur through hematogenous seeding.It can present as an intracranial space occupying lesion, lacking symptoms typifying an infection.This atypical presentation can lead physicians to suspect a malignancy at initial presentation, only to be surprised on tissue biopsy to find filamentous bacteria sprawling the specimen.

CASE REPORT

70 years female, resident of Dahisar, known case of Diabetes mellitus was suffering from anemia since June 2018. She presented to private hospital as Congestive cardiac failure with Hemoglobin of 4.3 gm, Pro BNP level increased to 1075, LDH-1333. 2 D ECHO was suggestive of Moderate Mitral regurgitation, mild pulmonary hypertension, LV ejection fraction 50%. She was managed conservatively and referred to Hematologist. Bone marrow aspirate was mildly hypercellular with erythroid hyperplasia, no blasts, no ring sideroblasts. So she was diagnosed as Coombs Negative hemolytic anemia, was started on inj. Methylprednisolone 1 gm for 3 days, later switched to oral steroids, Tab. Prednisolone 40 mg & later tapered by 10 mg /month. Since 2018, she was on steroids on and off. In between due to worsening anemia, She was started on Mycophenolate Mofetil 1.5 gm/ day. Later on tab Dapsone 100mg once a day was added. In Jan 2019, she again developed severe anemia, Hemoglobin 3.4 gm. She received inj. Methylprednisolone 1 gm for 3 days, later switched to Tablet Prednisolone 60 mg/day, Tablet Azathioprine 100 mg /day was added.This time her DCT/ICT came Positive and she was labeled as Autoimmune hemolytic anemia. She was on Tablet Prednisolone 60 mg/day and Tab.Azathioprine 100 mg /day till April 2019.

She came to our hospital on 22/4/2019. She presented to our hospital with generalized body swelling, dyspnea on exertion sine 15 days, fever low grade on and off since 2

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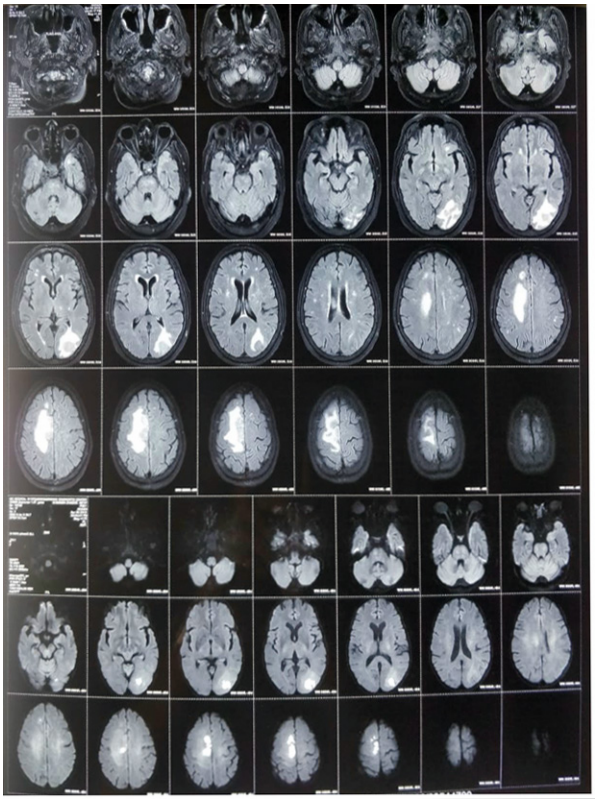

Figure 1. MRI brain-Lesions in right parafalcine region,right occipital region showing peri-lesional edema and diffusion restriction on DWI

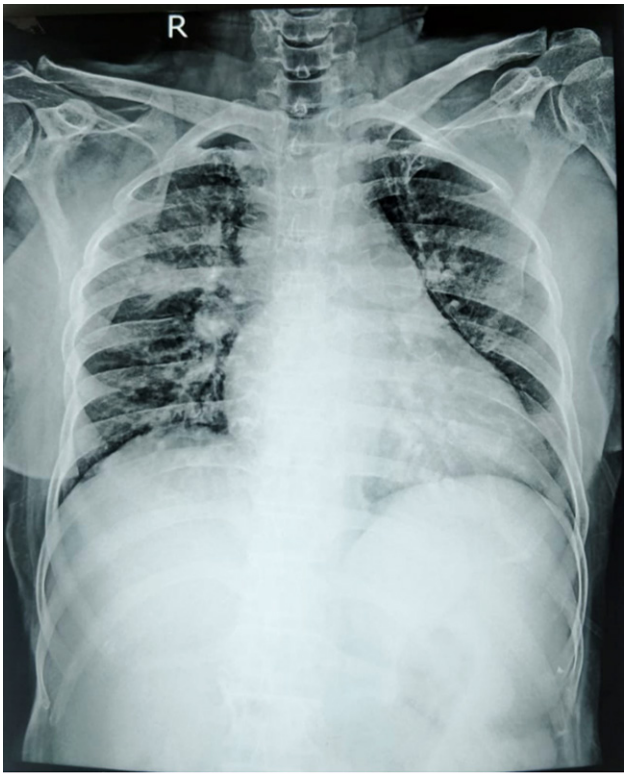

Figure 2. X-ray chest- Consolidation in right middle lobe of lung with lymphadenopathy

months. She is complaining of difficulty in walking with left upper limb and left lower limb weakness since 2 months. She didn't have altered sensorium, seizure, difficulty in speech. She noticed a small lump in breast and para-umbilical region. She was vitally stable. On examination she had left upper limb and lower limb weakness with power 0/5. She was conscious oriented. No cranial nerve involvement, no bladder bowel involvement. She had a small 1x1cm soft lump in right breast and 2x2cm lump in right para-umbilical region. No cervical, inguinal or axillary lymphadenopathy. No other lump palpable, no organomegaly. She was very pale, suffering from severe anemia. At admission her reports were- HB-4.8gm, platelets- 3.62 lakhs/mm3, WBC count-8000, total Bilirubin 2.2 mg, Indirect Bilirubin 1.7mg, Sr. Creatinine 1.1 mg, Sr.LDH- 889. Her direct and indirect Comb's tests came Positive in high titers(2+), two 2 units of blood were transfused. She was given pulse Methyl Prednisolone 1gm for 3 days & later started on tab Prednisolone 40 mg and Mycophenolate Mofetil 1gm per day. She had deranged sugars on admission- fasting 214mg and post prandial blood sugar 357mg. She was given subcutaneous insulin according to sugar levels. In view of left hemiparesis, her MRI brain and spine was done which was suggestive of lesions in right Parafalcine region, right Occipital region showing peri-lesional edema and diffusion restriction on DWI, T2 isointense lesion in prevertebral region at D12-L1level, degenerative changes in spine at L4-5 and L5-S1 level, ? Malignancy and Metastasis vs Infective etiology **[Figures 1,3,4].**

In view of fever and lump in abdomen,CT Chest , Abdomen, Pelvis was done which showed moderate sized ill defined area of consolidatory collapse with central necrotic changes in right middle lobe of lung with adjacent lung parenchyma revealing multiple small sized nodular lesions with ground glass haziness, which was also evident on Chest X ray **(Figure 2).** Small sizes lymph nodes in prevascular, precarinal and left hilar region. Small sized well defined nodular lesions in the breast on both sides, measuring 2.5.*2 cm on the right side and 1.5*1.2 cm on left side with mild post contrast enhancement. Small nodule in the subcutaneous plane 1.8*1.7 cm in the para-umbilical region with mild post contrast enhancement? Metastatic ? infective. Possibility of primary in right lung **[Figure 5].**

With the possibility of infective etiology such as multiple abscesses, patient was started on inj. Ceftriaxone 2 gm/day, inj Vancomycin 2gm/day, tab Prednisolone 50 mg (1mg/kg) was continued. We stopped Azathioprine in view of infective etiology. CT guided aspiration of abdominal wall nodule was attempted which came out to be pus, it was aspirated

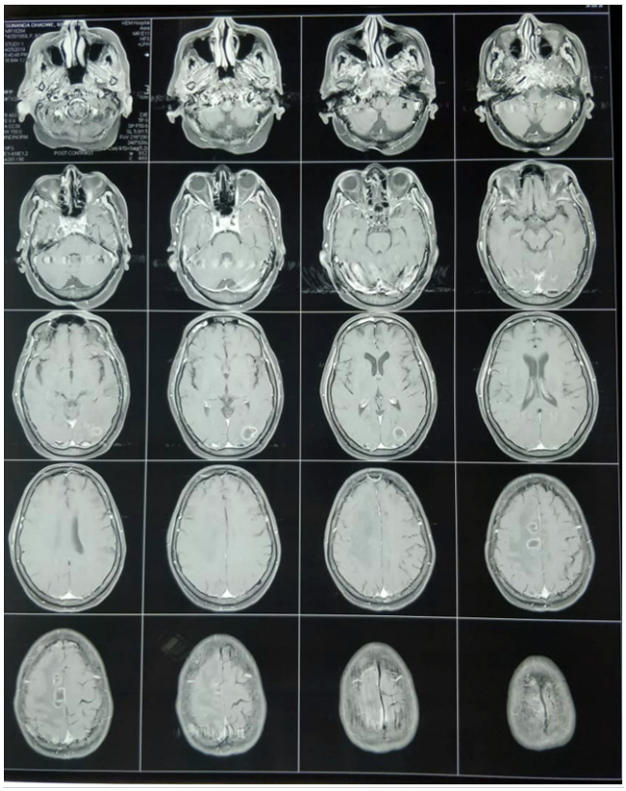

Figure 3. MRI Brain-Lesions in right parafalcine region,right occipital region showing peri-lesional edema and diffusion restriction on DWI

and sent for culture. Aerobic Culture grew Gram Positive bacilli. Modified ZNCF stain showed acid fast branching filaments suggestive of Nocardia. On Gram stain -Gram positive branching filaments suggestive of Nocardia seen susceptible to Trimethoprim / sulfamethaxole, Amikacin, Linezolid, Imipenem. So finally diagnosis of disseminated Nocardiosis involving lung, brain, spine, subcutaneous tissue with multiple abscesses was done and she was started on inj Imipenem 500mg tds,inj Amikacin, Trimethoprim / Sulfamethaxole.

DISCUSSION

Nocardiosis is an opportunistic infection caused by a gram positive bacteria belonging to the genus Nocardia. As an aerobic saprophytic Actinomycetes thriving in soil and water, Nocardia Species are known to cause disseminated suppurative disease in humans and animals upon infection.[1-5] Nocardiosis routinely affects individuals with compromised immune system, particularly defects in cell mediated immunity, like infection with HIV, steroid therapy, malignancy, transplant recipients on immunosuppressive treatments. Approximately one-third of infected patients are immunocompetent.[4]

Nocardiosis is notoriously known for its ability to disseminate extensively and relapse in spite of adequate therapy necessitating prolong treatment regimens. Its indolent yet invasive nature commonly leads physicians to suspect malignancy.

The classification of Nocardiosis is based upon the location and extent of disease and includes pulmonary, central

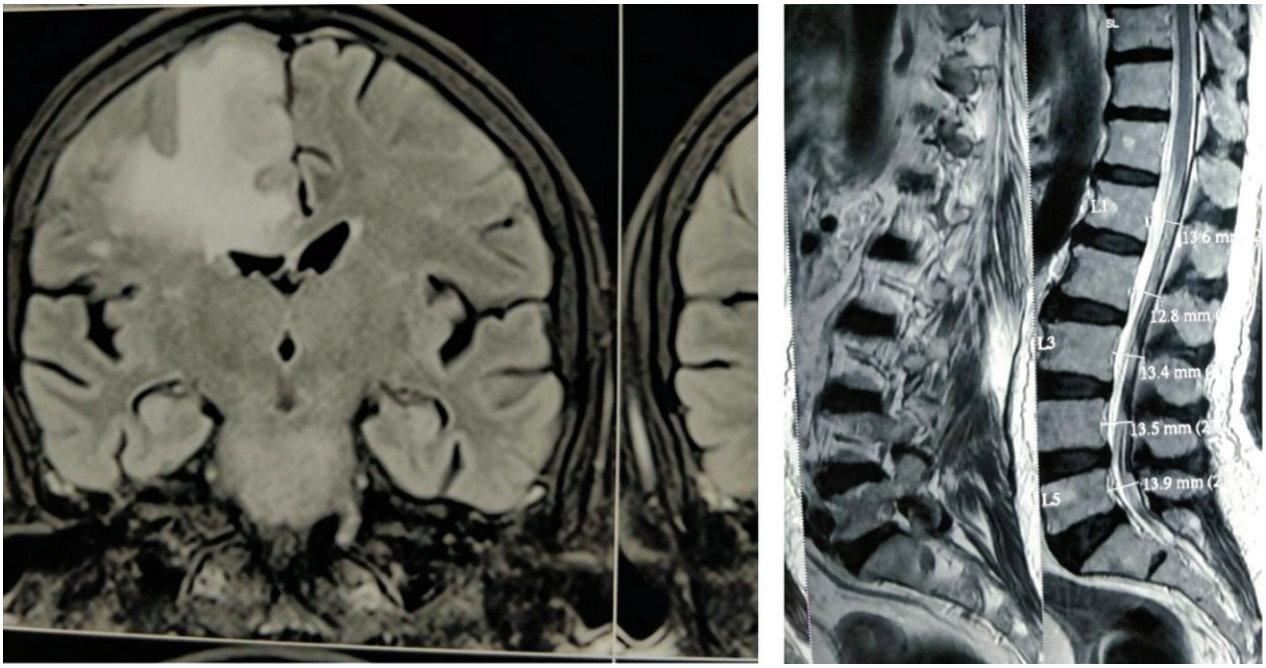

Figure 4. MRI Spine-T2 isointense lesion in prevertebral region at D12-L1level, degenerative changes in spine at L4-5 and L5-S1 level

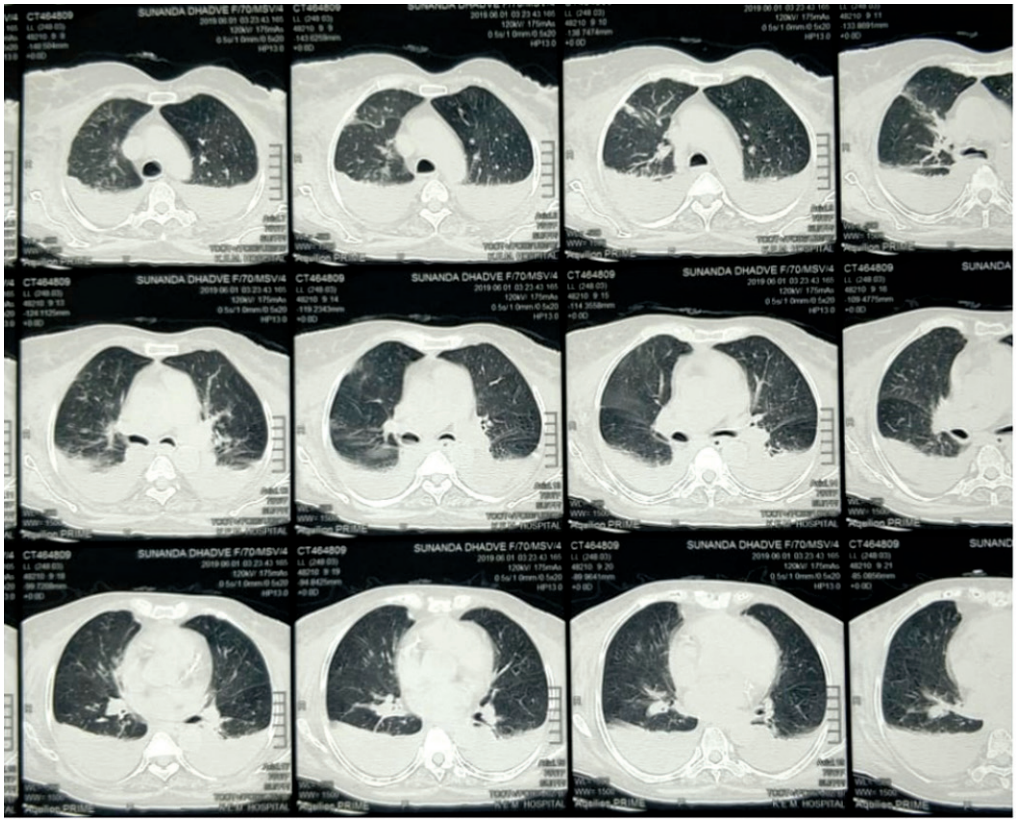

Figure 5. CECT chest-Area of consolidatory collapse with central necrotic changes in right middle lobe with multiple nodules & ground glass haziness with lymphadenopathy and Nodular lesions in breast on both sides

nervous system, cutaneous, and disseminated disease.[3,6] Although there are no pathognomonic signs or symptoms of Nocardiosis, it should be suspected in any patient who presents with brain, soft tissue, or cutaneous lesions and a concurrent or recent pulmonary process.

The lungs are the primary site of Nocardial infection in more than two-thirds of cases, Nocardia species are not normally found in the respiratory tract; as a result, isolation of Nocardia from the sputum is almost always indicative of infection.

Nocardiosis can present in acute ,subacute or chronic form with symptoms ranging from non specific fatigue, night sweats, Fever, weight loss, anorexia to cough, dyspnea, hemoptysis or pleuritic chest pain pointing toward pulmonary pathology.[3,6,8] Pulmonary Nocardiosis is usually primary, but secondary spread to lungs from skin infection has been documented.[7]

In people suffering from chronic lung diseases like Sarcoidosis or chronic obstructive lung disease Nocardiosis can present as an exacerbation of underlying disease,[9,10] often leading to delay in diagnosis. Glucocorticoid treatment given for these lung diseases tends to worsen Nocardial infection.

Most common organ affected by Nocardiosis is the lungs. Even in cases of extra pulmonary Nocardiosis up to 50 percent of the times lungs are the the primary source of infection. Brain is the most common site of dissemination. Only 20 percent of extra pulmonary *Nocardi*a infections occur in the absence of pulmonary disease.[6] Empyema, Mediastinitis, Pericarditis, and Superior vena cava syndrome are known to complicate pulmonary infection by means contagious spread.[11-13]

A multitude of imaging findings have been demonstrated in pulmonary nocardiosis, including single or multiple nodules, lung masses (with or without cavitation), reticulonodular infiltrates, interstitial infiltrates, lobar consolidation, subpleural plaques, and pleural effusions.[5,14] As a result, Nocardiosis has frequently been misdiagnosed initially as tuberculosis (since upper lobe involvement is common and Nocardia species are weakly acid fast), invasive fungal disease, and malignancy.[3]

The hallmark of CNS Nocardiosis is formation of a parenchymal abscess that can occur in any region of the brain.[5,15,16] Patients may present with fever, headache, meningismus, seizures, and/or focal neurologic deficits.[5] CNS Nocardiosis can present with symptoms suggesting a mass lesion without any symptoms typically associated with infection.[17] In such patients, Nocardial brain abscess may be erroneously diagnosed as a primary or metastatic neoplasm prior to biopsy. Although there are no pathognomonic signs or symptoms of Nocardiosis, it should be suspected in any patient who presents with brain, soft tissue, or cutaneous lesions and a concurrent or recent pulmonary process.

CONCLUSION

Our case was a disseminated Nocardiosis in a patient on immunosuppressant therapy in whom the clinical presentation was highly suggestive of a metastatic disease. A

definitive diagnosis of Nocardiosis requires the isolation and identification of the organism from a clinical specimen. Delay in establishing the correct diagnosis is common due to the nonspecific and diverse clinical presentation of Nocardiosis and the inherent difficulty in cultivating *Nocardia*.

END NOTE

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