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Case Presentation on Nocardiosis

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Abstract
Nocardiosis is a rare infection that is often difficult to treat and may be life-threatening. There is no consensus on its management.Nocardiosis, a primarily opportunistic infection which may occur in immunocompetent persons, most commonly involves the lungs and frequently disseminates to other sites including the central nervous system. Trimethoprim-sulfamethoxazole is the preferred agent for initial therapy, because Nocardia is very often susceptible to this agent, and because it has been the keystone of nocardiosis treatment for years. Linezolid, to which Nocardia is almost always susceptible, may be an alternative. When combination therapy is required, the repertoire of companion drugs includes third generation cephalosporins, amikacin and imipenem. Therapeutic modifications should consider clinical response to initial therapy and AST results. Treatment duration of 6 months is appropriate for most situations, but longer durations are preferred for disseminated nocardiosis and shorter durations are reasonable in low-risk situations. Secondary prophylaxis may be considered in selected individuals with permanent immunosuppression.

Key Words: *Immunocompetent, Pleural effusion, Pneumonia*

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I. Introduction

Nocardiosis is a disease caused by bacteria found in soil and water. It can affect the lungs, brain, and skin. It is most common in people with weakened immune systems who have difficulty fighting off infections (for example, people with cancer or those taking certain medications such as steroids). Antibiotic treatment might be given for several months to get rid of the infection.

People with very weak immune systems are at risk for getting nocardiosis. Several diseases and conditions can cause the immune system to be weak. These include Diabetes, Cancer, HIV/AIDS, Pulmonary alveolar proteinosis (an illness that causes the air sacs of the lungs to become plugged), Connective tissue disorder (a disease that affects the tissue that connects and supports different parts of the body), Alcoholism, having a bone marrow or solid organ transplant, taking high doses of drugs called corticosteroids[1, 2].

The symptoms of nocardiosis vary depending on which part of your body is affected. Nocardiosis most commonly occurs in the lungs. If your lungs are infected, you can experience Fever, Weight loss, Night sweats, Cough, Chest pain, Pneumonia[1].

When lung infections occur, the infection can spread to the brain. If your central nervous system (brain and spinal cord) is infected, you can experience Headache, Weakness, Confusion, Seizures.

Skin infections can occur when soil containing *Nocardia* species gets into open wounds or cuts. Farming or gardening without gloves and protective clothing increases the risk of cuts, thorn pricks, or other minor injuries. If your skin is infected, you can develop: Skin ulcers (shallow wound on the surface of the skin). Nodules, sometimes draining, with the infection spreading along lymph nodes[3,4].

Nocardiosis is a disease caused by a type of bacteria that is found in the environment, typically in standing water, decaying plants, and soil. These bacterial species belong to the genus *Nocardia* giving the disease its name. *Nocardia* and other related bacteria are opportunistic pathogens. These are bacteria that infect humans and animals when the conditions are right. They can cause severe infections in people with weakened immune systems who have difficulty fighting off infections (for example, people with cancer or those taking certain medications such as steroids)[5].

II. Case Presentation:

A 53 year's old male patient, farmer by occupation, Ex-smoker, non-alcoholic has a medical history of Hansen's disease, hypertension, type 2 diabetes mellitus, coronary artery disease, S/P PTCA stent (2019) and

came with the chief complaints of cough with expectoration since 15days, shortness of breath from past one week and progressed from grade 2 to 4 mMRC, right chest pain, pleuritic in nature, non-radiating type.
Patient has completed his treatment for Hansen's disease in 2014 and developed lepra reactions later for which steroids(prednisolone) was started. He has been on and off steroids ever since depending on lepra reactions.
Patient was admitted in the emergency department as he was presented excess shortness of breath (SOB) and spo2 75%. Patient was primarily treated with Lasix, hydrocortisone, 15 litres of oxygen support, nebulization and within 15 minutes patient was stable. Initially patient was treated as heart failure before evaluation.
Blood reports, 2D ECHO, X RAY, investigations were done. 2D ECHO showed ejection fraction (EF) 40% and moderate LV dysfunction, blood reports were moderately abnormal. Blood urea 95 mg/dl, HBA1C- 7.4 and patient was negative for HIV and hepatitis. Later patient was impressed as "Acute decompensated cardiac failure" and treated with O2 inhalation (5L/min), injection LASIX 40mg, ATORVASTATIN 40mg, ECOSPIRIN 75mg, METXL 25mg, PAN 40mg, ZOFER 4mg.
X-RAY was done and showed as 'Pleural effusion'. Based on the X-RAY, patient was impressed as 'Acute decompensated cardiac failure with community acquired pneumonia'.

On 2nd day of treatment 'THORACENTESIS' procedure was done 650ml of pleural fluid was removed and sent it for further fluid analysis (CSF, ADA, LDH, Fluid cytology). LDH-1418 IU/L, ADA- 32.8 U/L and pleural fluid appearance was straw coloured and investigated for TB fluorescence staining which showed negative, and Gene Xpert also showed negative. Patient was sent for CT scan which showed 'Necrotising pneumonia' in upper and middle lobe, consolidated lung field shows heterogenous enhancement. Likely infective etiology, exudative loculated collection, cardiomegaly with reactive pericardial effusion, pericardial thickening, mediastinal fat strandings.

After confirmation, patient was shifted to pulmonary ward from emergency department. After shifting,steroidswere tapered from 40mg to 20mg and MET XL 25mg was stopped and remaining treatment was continued as above.

In pulmonary ward, patient was started on injection PIPTAZ 4.5gm QID, DOXYCYCLINE 100mg BD. On 4th day of treatment patient was suddenly presented with excess SOB and sweating after patient understood that steroid was tapered to 20mg as he was psychologically dependent on steroids then patient was immediately shifted to ICU and treated with hydrocortisone and nebulization with oxygen support. Even though his vitals were stable, but patient was presenting excess SOB, so the doctors confirmed it was because of panic attack. As the patient was psychologically dependent on steroids, he was continuously asking for steroids starting dose(40mg). patients stay in ICU was for 3days and shifted again to the pulmonary ward after being stable. But patient was showing increased heart rate then again doctors administered MET XL 25mg. Even after 10 days of treatment with antibiotics, there was no improvement shown in X RAY reports. Patient was also investigated multiple times for TB staining which showed negative.

So, they planned for 'Pigtail' procedure. 250ml of fluid was removed with 'Pigtail catheterisation. After 2days, there was no fluid coming out. So, the patient was sent to doppler test. It showed multiple septation. So, the fluid removed or coming out was impossible. USG chest was also done which showed loculated effusion in right pleural cavity. So, they planned for 'STREPTOKINESE'therapy for breaking of locules. On 2nd day of streptokinase therapy, left foot abscess was seen and started bleeding. So, immediately streptokinase therapy was stopped.

On each administration of injection PIPTAZ, patient reports showed decreased potassium levels (HYPOKALEMIA). For left foot abscess, patient was started with AUGUMENTIN 1.2gm, CLINDAMYCIN 600mg. PIPTAZ and DOXY stopped. Steroids was also tapered from 20mg to 10mg then 5mg. 5mg was given alternative days and then completely stopped. On the first 2 days of stoppage, patient got fever spikes and low levels of blood pressure and sugar. Heart rate showed normal on receiving MET XL 25mg.

Sputum test was also done, and the culture reports showed scanty growth of gram-negative bacilli and gram-positive cocci. ESR- 150mm/hr, D-dimer- 5989ng/ml. HS. TROP showed negative always. Albumin levels gradually decreased. So, the patient was started on tablet SHELCAL.

Pleural fluid cytology was also done in which smears were negative for malignancy and finally diagnosed with inflammatory pathology with "NOCARDIAL INFESTATION". Then patient was started with injection MEROPENEM 1gm and Tablet SEPTRAN DS (Sulfamethoxazole and trimethoprim). AUGMENTIN was stopped. Patient symptomatically improved. Cough was reduced but patient showed SOB while mobility. X-RAY report showed improvement on SEPTRAN DS and MEROPENEM. Patient got fungal infection in mouth because of continuation nebulization. Patient showed nausea and burning sensation in GI tract. Endoscopy was done which showed double channel pylori and RUT test also showed positive. Triple therapy(clarithromycin+amoxicillin+pantaprazole) was started for peptic ulcer disease. After 3 weeks of treatment meropenem was stopped and septran ds was prescribed to continue for 6 months.

CRP increased on day-to-day progress then again clindamycin was started. BP and sugar levels showed low and sometimes patient fainted due to low BP and sugar levels. CBP drastically decreased haemoglobin and

RBC count then one pint blood was transfused. Slowly patient showed hand tremors and investigated for MRI brain which showed no abnormalities. Clindamycin was stopped as the patient was symptomatically improved and haemodynamically stable. Patient was planned for discharge with medications of tab SEPTRAN DS, FEROPENEM, LASILACTONE, CARDACE, ATORVA, ECOSPIRIN and AMIKACIN for 5days. Review after 1 week.

After long stay of hospital patient was discharged to home. On first 2 days patient was stable. On 3rd day patient behaviour was changed. Patient was showing hallucinations, extreme tremors. So, the patient was started with tab PREGABALIN with physician suggestion. Patient was stable on the follow up visit.

III. Discussion:

Patient has the known history of CAD to control blood pressure MET XL 25mg was prescribed But on Day 1 MET XL 25mg was stopped in Day 4even through the patient was stable it was noticed that the patient is still having the rise in heart rate and blood pressure in order to control it on DAY 4 Doctor has again prescribed MET XL 25 mg. Metoprolol is a selective cardiac beta-1-adrenergic receptor inhibitor that blocks beat-1 receptors and it shows action by decreasing the cardiac output by negative inotropic and chronotropic effects. As the result of readministering Met XL patient heart rate was stabled. As the subject is depending on the steroids because of the adversity of it doctor has suggested to tamper the dosing on from Day 2 and its was identified that after tampering the dose patient had symptoms such as fever spikes, hypoglycaemia and hypotension it was identified that tampering steroids dose is one of major reason behind the occurrence of the events, however administering the medications such as paracetamol along with monitoring insulin levels has helped to overcoming the effects of steroids tampering.Upon investigation doctor came to know that patient is completely dependent on steroids since so many years and as per many research studies it was confirmed that long term exposure of steroids can cause the suppression of the hypothalamic-pituitary-adrenal (HPA) axis. HPA axis means interaction between the hypothalamus, pituitary gland and adrenal glands, this coordination is contributing a significant role when the body reacts to stress, this response is immediate and result in secretion of epinephrine and norepinephrine, which generally result in increased heart rate and perspiration.So, sudden tampering of steroids would have caused the adrenal insufficiency since the adrenal gland would not has the sufficient time to produce the hormones to mee the body's requirement. Long term usage of steroids can also cause the immune suppression.

IV. Conclusion:

Nocardia is an opportunistic pathogen and patients with the immunosuppression will have chance of getting infected by the nocardia. Optimal antimicrobial therapy for the nocardiosis is hindered because of lack of prospective controlled trials. However, there are some clinical and laboratory standards institute has published recommendations for antimicrobial susceptibility testing for nocardia and other aerobic actinomycetes. Nocardia which is separated from the clinically significant infections should undergo antimicrobial susceptibility testing to assist in treatment decisions. Trimethoprim-sulfamethoxazole is the active against the most of the nocardia species. Amoxicillin-clavulanic acid is active against many strains of nocardia. Doctor has prescribed all medications which was considered as effective against the nocardiosis.

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