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RESEARCH ARTICLE

PULMONARY NOCARDIOSIS CONCOMITANT INFECTION WITH PULMONARY TUBERCULOSIS IN IMMUNOCOMPROMISED PATIENT.

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Nocardiosis, Cavitation, Tuberculosis,
 Immunocompromised, Endobronchial.

Abstract

Till date we have seen a very few literature reporting pulmonary nocardiosis concomitant infection with mycobacterium tuberculosis. We present a 74 year old case in which a patient with long segment myelitis, who was on long term steroids on thorough work up for PUO was found to have mycobacterium tuberculosis co-infected with nocardiosis. Physiological doses of oral steroids recommended for long segment myelitis might be the predisposing factor for opportunistic infection of nocardiosis in patient with MTB.

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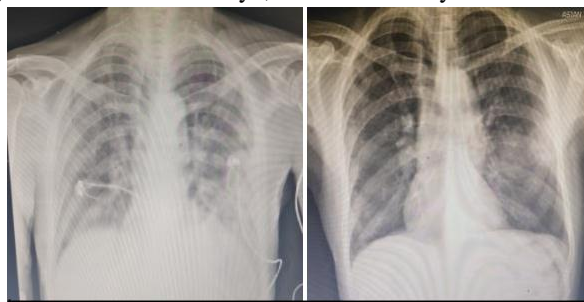
Introduction:-

History of Present Illness: A 74-year-old man with history of cough with expectoration, breathlessness (MMRC II) and high grade fever with chills since 7 days. Patient was already a k/c/o long term myelitis, SACD (c3-c7) and was on oral steroid treatment since last 6 months.

On Examination: conscious oriented, Chest : B/L coarse crepts. Initial vital signs were within normal limits.

Laboratory and Radiology: Pertinent laboratory findings revealed leukocytosis with a left shift. Liver and renal testing was normal. HbA1c 7.1, a diagnosis of steroid induced hyperglycemia was made. Sputum cultures, acid-fast bacilli (AFB) and blood cultures were obtained all were sterile. Chest X-ray (CXR) (Figure 1) s/o patchy infiltrates in left lower zone. CT scan (Figure 2) revealed a large thick walled cavitary lesion with air fluid level in left mid zone. A large avid thin walled cavity in paramediastinal upper lobe of left lung. Few, thin walled irregular cavities in posterior segment of left lower lobe lung. Multiple, irregular soft tissue nodular opacities predominantly in the left lung, multiple mediastinal lymphadenopathy.

Figure1:-CXR AP on Day1, CXR PA 15 days after treatment



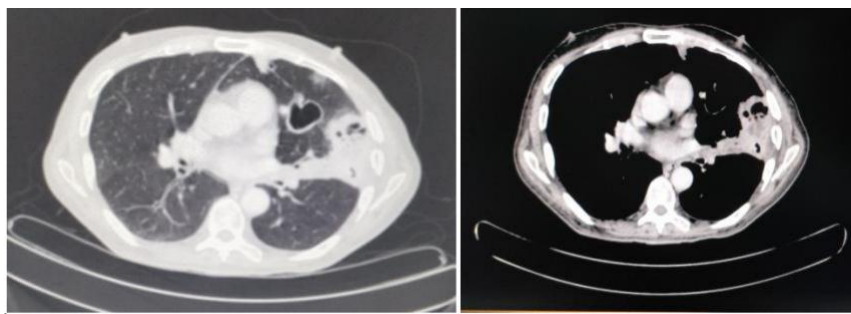


Figure 2:-left upper lobe consolidation with internal necrotic lesion with multiple cavitation.

Bronchoscopy was performed BAL done , showed modified AFB, nocardia present, AFB smear 3+, gene xpert MTB detected(Rif. Sensitive). PCR was positive for MTB.

Hospital Course: After admission, the patient was started on broad spectrum antimicrobials with clarithromycin and ceftriaxone + salbactam along with other supportive measures. Later bronchoscopy was performed following its result antitubercular (HRZE) regime with trimethoprim-sulfamethoxazole for newly diagnosed pulmonary nocardiosis was started. The patient showed considerable improvement in his respiratory symptoms and was transitioned to oral trimethoprim-sulfamethoxazole for outpatient treatment of nocardiosis with continuation of ATT for MTB.

Discussion:-

Nocardiosis is a rare infection caused by the *Nocardia asteroides* bacterium a weakly acid fast gram positive filamentous bacteria and is a possible cause of pulmonary and systemic infection in immunocompromised host, rarely isolated from immune competent patients(1). The first human case was reported in 1890 by Eppinger.

Nocardia omnipresent in the environment: soil, organic matter, and water. Human infection usually occurs from minor trauma and direct inoculation of the skin or soft tissues or by inhalation. Outbreaks in oncology and transplant wards and surgical wounds have occurred from fomites, hospital construction with resultant contaminated dust, and health care worker hands. The annual incidence of ubiquitous environmental saprophyte nocardiosis is 500-1000 cases per year in US especially in tropical and subtropical regions. However, due to the increase in the number of immunosuppressed patients during recent decades, the incidence of nocardiosis is also increasing. Nocardiosis has been reported worldwide in all ages and races, and it is 2 to 3 times more common in men than in women. Pulmonary disease (pneumonia, endobronchial inflammatory mass, lung abscess, cavitary disease and progressive fibrotic diseases) is the most frequent clinical presentation (approximately 50% of cases), and most of the infective organisms are from the former *Nocardia asteroides* complex. Approximately 1/3 of patients with pulmonary nocardiosis will develop disseminated disease.[2] The inability to be killed by white cells takes on additional significance in the immunoincompetant who have WBC dysfunction that tips the battle between host and pathogen in favor of the *Nocardia*.

Common CT findings of nocardiosis include ground glass opacities, lung nodules, cavitation, pleural effusion and masses[3,4,5]. The usual treatment of choice includes sulphonamides and more recently TMP-SMX (cotrimoxazole). With certain complications, surgical drainage may be required. The prognosis can vary with those with disseminated nocardiosis having mortality rates up to 60% and 41% in Pulmonary nocardiosis, but if it reaches CNS then it is 100%. Subtherapeutic levels of above mentioned antibiotics if given, can result in flare-ups of the diseases on treatments. Linezolid, Cotrimoxazole, imipenem and minocycline were found to be very effective, in vitro, against most *Nocardia* species for duration of 6 months to a year based on the disseminated status.

Nocardia accounts for less than 3-4% of pulmonary infections in HIV infected patients[6]. Why this case seems to be of importance? as we usually expect PN in immunocompromised host but forget to give importance to this infection in other patients in COPD on steroids [7] or any steroid dependent patient who later develop TB and often do not consider to send the sputum for nocardiosis stain also.

So, it is advisable to rule out nocardiosis in MTB patient especially who are immunocompromised, COPD steroid dependent [7] and transplant patient [8] with a high degree of clinical suspicion is imperative to promptly treat infection with both organisms for better outcome.

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