CASE REPORT

Bilateral Tubercular Lung Abscess in a Diabetic Female

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Abstract:

Liquefactive necrosis of the lung tissue caused by microbial infection, lung abscess is characterised by formation of cavities containing necrotic debris. In the vast majority of cases of lung abscess, polymicrobial bacteria can be found with predominance of anaerobes. Mycobacterium has been described as a very rare causative agent of community acquired lung abscess. We are presenting a case of middle aged diabetic female, who had bilateral lung abscesses, aetiology of which was established to be tubercular. Astonishing it may sound; based upon extensive web and library search, it's the first case report on tubercular lung abscess in a diabetic from India, and perhaps from the world itself.

Keywords: Lung Abscess, Bilateral Lung Abscess, Tubercular Lung Abscess, Tubercular Lung Abscess in a Diabetic Female

Introduction:

Lung abscess is still considered a dreaded disease with high mortality in India. Patients of lung abscess most commonly presents as "non-resolving pneumonia." It is seen as a cavity with air fluid level on chest X-ray and computed topography. The aetiology is believed to be anaerobe-predominant-polymicrobial [1]. Understandably, the empiric treatment is directed towards that particular direction. Mycobacterium tuberculosis is believed to a rare etiological agent of community acquired lung abscess and the current empirical therapy doesn't include Anti Tubercular Therapy (ATT) as its component. This case report is about a diabetic woman whose lung abscess was found to be tubercular and she responded brilliantly with ATT, after a failed treatment with current conventional regime.

Case Report:

A 42 year old female was admitted in the emergency room of Govt. Medical College And Guru Nanak Dev Hospital, Amritsar, with complains of fever with chills with night sweats since 2 months, cough with foul smelling sputum production from 1.5 months, gradually increasing shortness of breath along with decreased appetite and loss of weight since 1.5 months. Normotensive, she was diabetic since last 7 years-well controlled on regular oral hypoglycaemics. Her glycaemic control had recently worsened with random blood sugar at presentation being 406 mg/dl. The patient turned to us after treatment from various quacks and local practitioners. During that span, besides various home remedies, she had consumed amoxicillin+clavulinic acid, azithromycin, erythromycin, cough suppressants, opoid pain killers, and steroids. She was a baptised Sikh with no history of smoking, drinking or any other substance abuse. Her history for seizure disorder, tuberculosis, leprosy, bronchial asthma, coronary artery disease was negative.

Upon examination, she was conscious, dyspnoeic with respiratory rate of 32/min, blood pressure 112/76 mm Hg, fever 103^0F and heart rate was 120

beats/min. She had grade 3 clubbing and mild pallor. There was no icterus, edema, cyanosis, lymphadenopathy, joint pain or rashes over the body. Her oxygen saturation was 76% on room air. On auscultation, breath sounds were diminished at the apexes and mammary regions, coarse inspiratory crackles were audible over these areas. Cavernous breath sounds were audible in the right interscapular region. Mediastinum was central. Apart from tachycardia, the cardiac auscultation was normal. Her orodental hygiene was fair, there was moderate fatty distension of abdomen - everything else in gastrointestinal examination was normal. The central nervous system examination was benign. Her saturation pressure of oxygen was 76% at room air. The haemoglobin was 9.2 gms, total leucocyte count was 7800/cmm with differential being neutrophils 65% and lymphocytes 35%. Her ESR was 91mm at the end of first hour. She tested nonreactive for HIV ELISA, Australia antigen and hepatitis C virus antigen. Renal function and liver function tests were grossly normal. Her montoux was negative and she tested negative for acid fast bacilli in sputum examination. The blood cultures, urine cultures and sputum cultures were sterile.

X-ray chest was requested and it showed a big cavity with air fluid level on the right lung field and another cavity of similar character but smaller in size on the left side at an almost similar level (Fig. 1). The Contrast Enhanced Computed Tomography (CECT) chest of the patient showed bilateral cavitatory lesions in the upper lobes with air fluid level suggestive of lung abscess (Fig. 2).

We requested the dental department for assistance and they made a note of 1 periodontal compromised tooth which was advised for extraction once the patient gets stabilised.

After getting her culture and sensitivity for antibiotics, the patient was put on intravenous clindamycin, metronidazole, subcutaneous short acting insulin, mucolytics and antipyretics. Special instructions were issued to the nursing team regarding her posture, postural drainage and percussion therapy. Work up for anti-nuclear antibodies, angiotensin converting enzyme, rheumatoid arthritis factor were negative during the further course of management.

No improvement was noted after 8 days of treatment. The symptoms rather worsened. X-ray taken on the eighth day of treatment showed increased size of the cavity (Fig. 3). With keeping the strong index of suspicion for tubercular lung abscess, the patient was resend for sputum examination for AFB. It came out be negative again in both the samples. Then she was sent for bronchoscopy followed by Bronchoalveolar Lavage (BAL). CT guided biopsy was planned as the next step. Her cytopthological report of brush smears and BAL tested negative for malignant cells. No acid fast bacilli were seen in micro-biological analysis of post bronchoscopy sputum but the microbiological examination of BAL showed AFB in both direct and concentrated smears. The cultures of BAL specimen also yielded positive results with growth of mycobacterium tuberculosis. The patient was immediately started on ATT under the standard RNTCP's, DOTS regimen for Category-I (2H3R3Z3E3+4H3R3). The response was dramatic. In the next 48 hours, she became afebrile. She continued showing great improvement as her toxaemia disappeared, became completely afebrile, glycaemic status became excellent, cough got better and her dyspnoea improved over the next 48 hours. Cavity size on chest X-ray after 2 weeks of ATT appeared

decreased to a great extent (Fig. 4). She was discharged from the ward after 1.5 months with immense improvement of her symptoms and the weight gain of around 5 kilos (Fig. 5).

The patient was monitored on monthly basis with the watsapp photos of bi-weekly chest X-rays taken at her native village primary health centre (Fig. 6) as well as monthly personal visits in outdoor of our institute. The patient has fully recovered and has been declared cured after her 6 months complete course of ATT (Fig.7).

Fig. 1: X-ray of the Patient at the Time of Admission showing Bilteral Lung Abscess Cavities

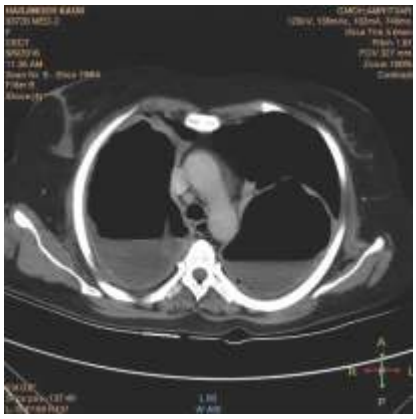

Fig. 2: CECT of the Patient at the Same Time

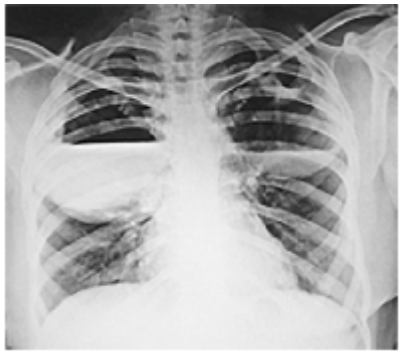

Fig. 3: X-Ray Showing Increase in Cavity Size despite Empirical Therapy

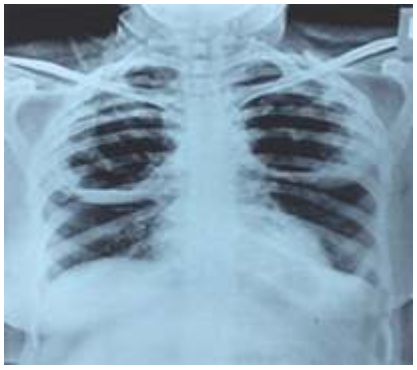

Fig. 4: X-Ray Showing Reduction in Cavity Size after 2 Weeks of ATT

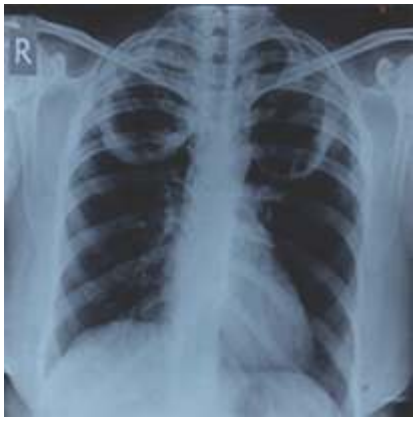

Fig. 5: X-Ray Showing Further Reduction in Cavity Size after 1 Month of Treatment with ATT

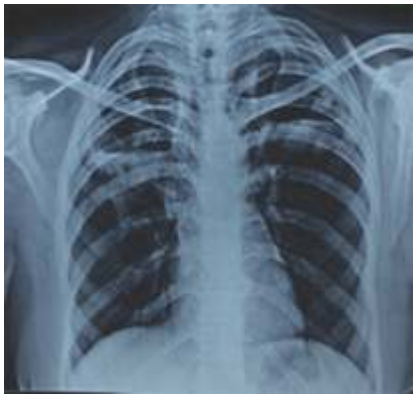

Fig. 6: X-Ray Showing Great Reduction of Cavity Size after 2 Months of ATT Treatment

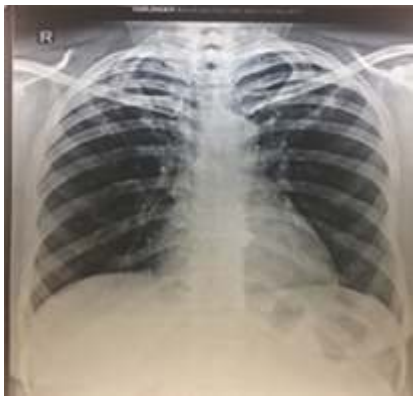

Fig. 7: X-Ray of the Patient Showing Complete Resolution and Disappearence of Both Lung Abscess Cavities at the End of Treatment with ATT

Discussion:

Lung abscess is a type of liquefactive necrosis of the lung tissue characterised by formation of cavities containing necrotic debris or fluid. Patients with lung abscesses commonly present to hospital with "non resolving pneumonia." Both host and infectious factors play a contributory role in the development of the disease. Amongst the host factors, the most common risk factors are aspiration and a systemic or local immuno-compromised status, such as chronic lung disease, malignancy, and diabetes mellitus [1].

Community-acquired lung abscesses has polymicrobial bacterial etiology in over 90% cases [2]. From anaerobic bacteria in lung abscess predominant isolates being gram-negative *Bacteroides fragilis*, *Fusobacterium capsulatum* and necrophorum, gram-positive anaerobic Peptostreptococcus and microearophillic streptococci. From aerobic bacteria predominant isolates in lung abscess being *Staphylococcus aureus*, *Streptococcus pyogenes* and *Streptococcus pneumonia*, *Klebsiella pneumonia*, *Pseudomonas aeruginosa*, *Haemophilus influenza* (type B), Acinetobacter spp, *Escherichia coli*, and Legionela [3,4].

Rarely, etiologic pathogens for lung abscess include Mycobacterium spp, Aspergillus, Cryptococcus, Histoplasma, Blastomyces, Coccidoides, Entamoeba histolytica, Paragominus westermani Actinomyces and Nocardia asteroids [5-6].

For a long time, its being widely believed that anaerobic bacteria are the most dominant type of bacteria in lung abscess. Amongst them, Streptococcus spp (*Streptococcus pneumonia* serotype 3, *Streptococcus anginosus* complex) were considered to be the most common types [7]. However, Wang *et al.* found *Klebsiella pneumonia* as the most isolated type of bacteria in lung abscess in their research at Taiwan [8]. From India, a case has been reported of a lung abscess due to *Mycobacterium tuberculosis* in an infant [9].

Diagnosis is by chest radiography showing a lung cavity with an air-fluid level. Ultrasound of the thorax and Computed Tomography (CT scan) of the chest are performed in suspicious cases or to further refine the diagnosis. CT is more sensitive than radiography because, although very rarely, chest X-ray can miss the small lesions or lesions in the hidden areas [10].

Diabetics, by virtue of their blunted self defence due to multiple reasons, are more predisposed to developing infections than the non- diabetics with normal immunity. So lung abscesses are more common in them as compared to general population. However, there is no study that can suggest that the aetiology is different in diabetics than non-diabetics. The aetiology of lung abscess in them is believed to the same polymicrobial with anaerobic predominance. But in India, the situation might be different as the country has the greatest burden of TB. This case might just be a protopype!

Conclusion:

Although infections by anaerobes or mixed polymicrobial infections are considered the usual suspects of causing lung abscesses, tubercular aetiology should be considered as one of the important differentials during the management of lung abscess in countries with its greater burden. Index of suspicion should rise further when the patient doesn't show improvement with the conventional empirical therapy which is directed predominantly against anaerobes.

References

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