

PULMONARY MYCETOMA DUE TO CANDIDA ALBICANS MASQUERADING BRONCHOGENIC CARCINOMA IN A PATIENT WITH TYPE -2 DIABETES MELLITUS

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ABSTRACT

Fungal lesions of lung presenting as a mass are called mycetomas. Mycetoma is a conglomeration of cellular debris and mucous with finely woven fungal hyphae present in a cavity in pulmonary parenchyma or a bronchus. The most common infections causing pulmonary mycetoma are aspergillus. Candida species causing pulmonary mycetoma is rare. A 70 year old patient of Diabetes and chronic obstructive pulmonary diseases (COPD), presented with acute on chronic respiratory symptoms. On contrast enhanced CT scan of thorax he was diagnosed as having a heterogeneously enhancing mass lesion in left lower lobe and minimal pleural effusion with a provisional diagnosis of neoplasm. Thorough examination of oral cavity revealed thrush. CT guided biopsy confirmed pulmonary candidiasis. Patient responded well to medical treatment. Only a few such cases have been reported in literature.

Key words: Candidiasis, Pulmonary Mycetoma, Bronchogenic carcinoma

INTRODUCTION

Candida albicans is a ubiquitous fungus which can be detected in approximately 32–55% of healthy individuals. Candida cause diseases in human when the body defense is compromised. Pulmonary disease caused by candida species has been reported rarely. We here by present a patient with pulmonary candidiasis who was initially diagnosed as bronchial neoplasm (1).

CASE REPORT

A 70 years old gentleman, known diabetic for 15 years and known case of COPD presented in emergency room with complaints of dyspnea on exertion, productive cough with mucoid sputum, loss of appetite, loss of weight for last 6 months. He had been a smoker for the last 40 years. This time he had fever, increased cough with scanty sputum and breathlessness at rest with loose stools for 2 days. He had poor oral intake and was dehydrated. On general examination he was of average built, pulse - 110/minute, blood pressure-110/80 mmHg, respiratory rate- 22/minute, SpO₂ was 96% in the room air, Random blood sugar was 230 mg/ dl. Glycosylated Hb was 8.5%. There was deposition of white cheesy material on oropharyngeal examination. Systemic examination was unremarkable except bilateral basal crepitations and rhonchi on chest auscultation. Investigations showed - Hemoglobin - 12.6 gm/dl, Total leucocyte count

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Fig.1: This is CECT thorax before starting antifungal treatment(07-aug-2012)

19,300/cmm with 94% polymorphs. Biochemical parameters were normal except hypoalbuminemia. Serological investigations for dengue, malaria and viral hepatitis were negative. Blood, urine and sputum cultures were also sterile. His x ray chest revealed heterogenous, nodular opacity in left lower lobe (2).

CECT THORAX

Contrast enhanced computed tomography of thorax showed heterogeneously enhancing mass lesion in left lower lobe abutting descending aorta with areas of collapse and patchy consolidation in left posterior apical segment suggestive of space occupying lesion probably neoplastic with minimal left sided pleural effusion (3).

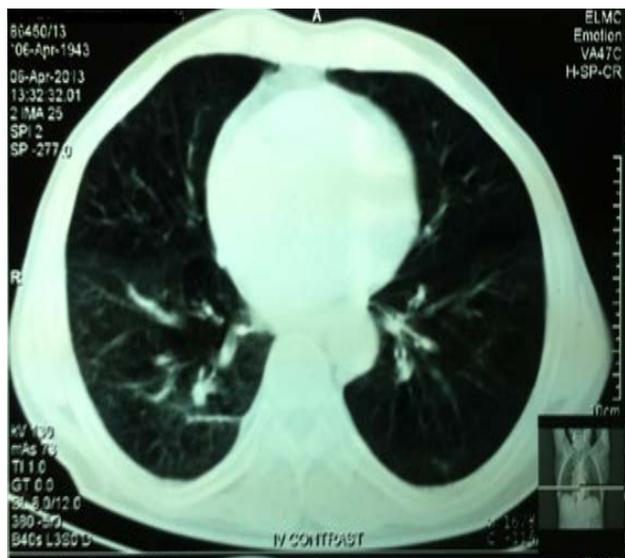


Fig.2:This is CECT thorax after antifungal treatment(06-apr-2013)

Bronchoscopic alveolar lavage (BAL) was done which showed isolated *Candida* and was negative for bacteria, acid-fast bacilli and malignant cells. We suspected the mass to be malignant and went for CT-guided fine needle aspiration cytology (FNAC) and true cut biopsy. CT-guided FNAC showed plenty of fungal hyphae in a necro-inflammatory background with absence of granuloma or malignancy. Giemsa-stained smear showed broad, irregular, hyphae with right-angled branching consistent with the diagnosis of fungal infection. Gomori's methenamine silver staining of CT-guided true cut biopsy also demonstrated the typical hyphae suggestive of mycosis. Hematoxylin and eosin staining of the true cut biopsy material showed wide areas of necrosis and inflammatory cell infiltration. Fungal culture of the specimen inoculated on Saboraud's Dextrose Agar medium yielded white colonies within 3 days and organism was identified as *Candida albicans*. Bronchoalveolar lavage showed absence of malignant cells. The diagnosis of pulmonary candidiasis was confirmed. As patient refused surgery, we started medical treatment. Patient was treated with intravenous fluids, nebulized bronchodilators, insulin, intravenous antibiotics cefoperazone 2 gm twice daily and metronidazole 500 mg trice daily with oral fluconazole 400mg daily and

clotrimazole mouth paint. He was put on maintenance treatment with insulin and oral fluconazole. Inhaled bronchodilators were prescribed. With appropriate dietary and lifestyle advice patient was discharged. He didn't turn up until after 9 months when he came for a follow-up visit. According to him he was still taking the same treatment and was much better than earlier. He gained approximately 5Kg weight and his blood sugar was controlled. A repeat CT scan of thorax eventually showed disappearance of the lesion (4).

DISCUSSION

In our patient oropharyngeal candidiasis was present that might have led to pulmonary lesion. Current Infectious Diseases Society of America (IDSA) guidelines for the empirical treatment of suspected candidemia or invasive *Candida* infection recommend that fluconazole may be used in clinically stable patients. *Candida* is a small, thin-walled, ovoid yeast that measures 4–6 micrometer in diameter and grows readily on simple medium. Species are identified by biochemical testing or on special agar (Saboraud's Dextrose Agar medium). *Candida* probably enters the bloodstream from mucosal surfaces after growing to large numbers as a consequence of bacterial suppression by antibacterial drugs, sometimes the organism may enter from the skin. Patients with severe burns, low-birth-weight neonates, IV drugs, HIV-infected with low CD4+ T cell counts and with diabetes mellitus are susceptible for *Candida* (5).

Patients with diabetes and HIV-infected patients with low CD4+ T cell counts are susceptible to mucocutaneous infection, which may eventually develop into the disseminated form (6).

Candidemia and invasive candidiasis are the most common causes of fungal infections, linked to a number of risk factors such as immunosuppressive therapy, intravenous catheters, and critical illness. Its saprophytic nature in human respiratory tract obscures its diagnosis (7).

Diagnosis of pulmonary candidiasis is based on demonstration of the fungus in the lung tissue. Pulmonary candidiasis can be because of two reasons, hematogenously disseminated candidiasis, along with involvement of multiple additional organs, rarely, after aspiration of oropharyngeal material, primary pneumonia due to *Candida* may develop (8). Our patient was a chronic smoker and immunocompromised due to uncontrolled diabetes mellitus. He never had been on inhalation therapy. *Candida albicans* was grown from bronchial specimen, cultures were negative for pyogenic organisms as well as AFB. Patient with type II

diabetes mellitus have more infections and course of infections is more complicated.

One possible explanation for this, is a defect in immune response. It has been shown that a TH2-axis shift, which decreases TH1-dependent immunity. In addition, decrease in cytokine response after stimulations or low-complement factor 4 may contribute for the compromise of humoral innate immunity (8).

CONCLUSION

Radiological suspicion of a mass lesion being neoplastic in origin must always be confirmed by a tissue diagnosis (5-6). A high index of suspicion for a fungal cause should be kept in mind while treating immuno-compromised individuals. Adequate antifungal treatment is to be instituted early in such patients with complete resolution in most cases(7-8).

CONFLICT OF INTEREST

The authors declare that they have no competing interests.

REFERENCES

1. Bachh,AAHaq,I Gupta R,VarudkarHG, Ram MB. Pulmonary candidiasis presenting as mycetoma :Lung India, 2008: Oct-Dec; 25(4): 165–167.
2. Shelly MA, Poe RH, Kapner LB. Pulmonary mycetoma due to *Candida albicans*: case report and review. *Clin Infect Dis*, 1996;22(1):133–5
3. Prats E, Sans J, Valldeperas J, Ferrer JE, Manresa F. Pulmonary mycetoma-like lesion caused by *Candida tropicalis*. *Respir Med*, 1995;89:303–4
4. Watanakunakorn C. Acute pulmonary mycetoma due to *Candida albicans* with complete resolution. *J Infect Dis*, 1983;148:1131
5. Yokoyama T., Sasaki J., Matsumoto K., Koga C., Ito Y., Kaku Y et al necrotic lung ball caused by co-infection with *Candida* and *Streptococcus pneumoniae* *Infect Drug Resist*, 2011: 4: 221–224. Published online 2011 Dec 15
6. Abel AT, Parwer S, Sanyal CS. Pulmonary mycetoma probably due to *Candida albicans* with complete resolution. *Respir Med*, 1998;92:1079–91
7. Sobel JD, Vazquez J. Candidemia and systemic candidiasis. *Semin Respir Infect*, 1990;5:123–37.
8. Miyazaki T, Kohno S, Mitsutake K, Maesaki S, Tanaka K, Ishikawa N et al Plasma (1-->3)-beta-D-glucan and fungal antigenemia in patients with candidemia, aspergillosis, and cryptococcosis. *J Clin Microbiol*, 1995;33:3115–8.

