Case Report

Pulmonary histoplasmosis: The masquerader of lung malignancy and tuberculosis

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ABSTRACT

Pulmonary histoplasmosis is a rare fungal infection caused by inhalation of spores of Histoplasma capsulatum, spread by bird/bat dropping. Pulmonary manifestations are the hallmark of the disease.it is rare in India, but few cases are seen in UP and West Bengal. Clinically, patient presents with wide spectrum of symptoms including respiratory failure. Pulmonary histoplasmosis mimics tuberculosis, sarcoidosis and malignancy Diagnosis is usually confirmed by histopathology and demonstration of organism in fungal cultures. In clinico-radiological scenario where investigations fail to confirm the diagnosis or patient doesn't respond well to the therapy, one should consider differential diagnosis of pulmonary histoplasmosis.

KEY WORDS: Biopsy, BAL, heterogenous mass, histopathology, histoplasmosis, lung cancer, pleural effusion, tuberculosis

INTRODUCTION

Pulmonary histoplasmosis is a rare fungal infection caused by the inhalation of spores of "*Histoplasma capsulatum*," spread occurs through bird/bat dropping. This dimorphic yeast is a member of the ascomycetes family. Pulmonary manifestations are the hallmark of histoplasmosis. The disease is endemic in America, especially in the Ohio and Mississippi region, whereas, in India, it is quite rare, but few cases have been reported from the Gangetic plains[1] of Uttar Pradesh and West Bengal. In clinical practice, the patient presents with a spectrum from an asymptomatic infection to a more dreadful diffuse alveolar disease which causes respiratory failure and ultimately death.

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CASE REPORT

We report a case of 55-year, non-smoker, male presented in 2018 to Rohilkhand Medical College and Hospital, Bareilly, Uttar Pradesh with complaints of progressively increasing dry cough and breathlessness for the past 3 months. There was no complaints related to chest pain, fever, hemoptysis, night sweats, or loss of weight. Patients temperature was 98.5°C, pulse rate was 86 beats/min, blood pressure was 130/80 mm Hg, on examination, there was no pallor, icterus, or no superficial lymphadenopathy.

On systemic examination, there were physical signs of the left side pleural effusion with ipsilateral collapse were elicited. Chest X-ray confirmed signs of the left-sided collapse with signs of left-sided mild pleural effusion. Pleural fluid analysis revealed ADA of 52 U/L with lymphocytic predominance. Pleural fluid and sputum sample were negative for AFB on microscopy and CBNAAT examination. His blood investigation revealed hemoglobin level of 10.4 g% (normal range 11.5–15 g%), white blood cell count of 6000 cells/cu mm (normal range 4000–11000 cells/cu mm), red blood cell count 3.8 million/cu mm (normal

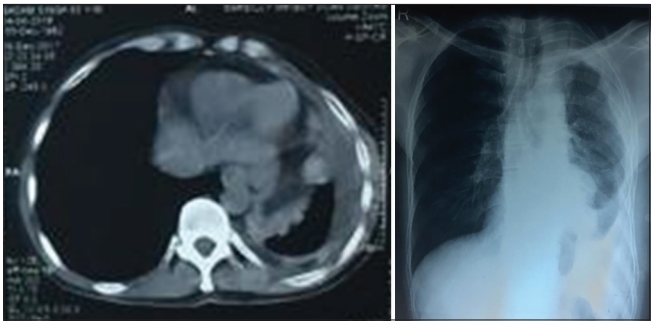

Figure 1: CT scan: Large lobulated heterogenous mass in lingula. Chest Xray: Left sided pleural Effusion with Collapse

range 3.55.5 million/cu mm), platelet count of 1.8 lac/cu mm (normal range 1.5–4.5 lac/cu mm), an elevated erythrocyte sedimentation rate, and 38 mm/l (normal range <20 mm/l). In view of raised ADA levels in pleural fluid, the patient was started on ATT, on follow–up, there was no clinicoradiological response; therefore, the patient was further investigated.

We did a bronchoscopy, which was within normal limits and BAL sample was negative for mycobacterium tuberculosis. We also did a CECT thorax, which showed large lobulated heterogeneous mass (7.5 × 3.9 cm) in the lingular segment of the left upper lobe along the mediastinal aspect suggestive of neoplastic disease. FNAC from the mass revealed suspicious cells, which required to further investigate. Then, a CT-guided biopsy was taken, on histopathological special stains (PAS and Grocott's methenamine silver [GMS]) showed highlighted fungal elements; on immunohistochemistry, Napsin and CK7 were highlighting the alveolar epithelium, CK20, and COX2 which were negative, thus confirming the fungal (histoplasmosis) nature of the disease. The patient was put on antifungal agent (Itraconazole 200 mg twice daily), gradually improved with clinical as well as radiological improvement.

DISCUSSION

Infection with *H. capsulatum* has a varied spectrum of presentations, acute pulmonary histoplasmosis, chronic pulmonary histoplasmosis, and disseminated progressive disease (mainly in immunocompromised patients). Acute pulmonary histoplasmosis may be asymptomatic or with mild flu-like symptoms to high-grade fever, non-productive cough, headache, and chest pain with mediastinal lymphadenopathy or mass such as lesion on radiology.[2] Chronic pulmonary histoplasmosis generally presents in a non-cavitary (reticulo-nodular shadows) or a cavitary form radiologically. Finally, the fatal kind of presentation is seen in immunocompromised patients as progressive disseminated histoplasmosis.

Pulmonary histoplasmosis mimics commonly occurring tuberculosis, sarcoidosis, and malignancy; hence, it is rarely diagnosed as it is rare in India and rarely considered in differentials. In our case, the patient was a EPTB suspect (tuberculosis is highly endemic in India) with high ADA levels with lymphocytic predominant pleural fluid, CECT done for further evaluation suggested malignancy by demonstration of mass. Histopathological examination confirmed the diagnosis of pulmonary histoplasmosis.

Availability of urine antigen test and antigen detection in sputum/BAL sample has made diagnosis easier, but high degree of suspicion is required. The diagnosis always requires confirmation by histopathology (GMS) and gold standard is demonstration of the organism on fungal cultures.

Few serological tests for detection of antigen and antibodies are available for the diagnosis of histoplasmosis but are generally missed due to lack of suspicion. Mild disease is mostly self-limiting, treatment is warranted in patients with aggressive disease, that is, diffuse infection, progressive disease, or with mediastinal lymphadenopathy with obstructive symptoms.

Acute pulmonary involvement with symptoms beyond 4 weeks is treated with, a 3-month course of itraconazole. Chronic pulmonary histoplasmosis with non-cavitary disease and cavitary disease is treated for 6-months and 12 months, respectively. Aggressive disease requires liposomal amphotericin-B (induction therapy for 4 weeks) followed by 12 months itraconazole therapy.[3] Antifungal therapy may prevent reactivation of histoplasmosis although it is controversial and further studies are recommended.

CONCLUSION

In clinicoradiological scenario suggesting tuberculosis, sarcoidosis, or malignancy, where investigations fails to confirm the diagnosis or patient does not respond well to the therapy that one should consider the differential diagnosis of pulmonary histoplasmosis.[4]

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