Pulmonary histoplasmosis mimicking carcinoma lung

Sir,

Human histoplasmosis is an endemic mycosis caused by a dimorphic fungus. Human histoplasmosis is an endemic mycosis caused by a dimorphic fungus-limiting pulmonary illness. However, patients with advanced AIDS usually develop progressive disseminated histoplasmosis.[1] In a minority of cases the manifestations can mimic primary or metastatic malignancies leading to delay in appropriate treatment or unwarranted therapeutic interventions. Here we report a case of histoplasmosis which was radiologically mimicked as lung malignancy with pleural deposits.

A 70-year-old South Indian lady who was apparently normal 3 months before presented with neck pain and generalized fatigue for 2-month duration. She was conservatively managed at local hospital, but her symptoms were deteriorating with increased tiredness and occasional cough. It was associated with loss of weight and loss of appetite. There was no history of fever, dyspnea, night sweats, dysphagia and hemoptysis. She is a farmer by occupation who was actively farming until 10 years back. But she had no history of smoking or working in the mining industry. She had no history of tuberculosis or other lung disease in her family. She was afebrile and her vitals were stable. General examination revealed grade 2 clubbing and there was no pallor, icterus or generalized lymphadenopathy. There was no hepatosplenomegaly. Breath sounds were decreased in the right lower zone. The rest of the respiratory examination was unremarkable. Routine blood investigations revealed hemoglobin of 12.1 gm%, total count of 10,000/cmm (neutrophils: 66.9%, lymphocytes: 21.3%, eosinophil 4.2%, monocytes: 7.2%, basophil: 0.4%) and Erythrocyte sedimentation rate of 90.Viral markers including HIV, HBsAg, and HCV were negative. Chest X-ray revealed widening of mediastinum, upper zone collapse and bilateral lower rib erosion. Contrast-enhanced CT chest was performed which revealed

6 × 3 cm ill-defined mass lesion in the anterior mediastinum having broad area of contact with pulmonary artery and aortic arch [Figure 1a]. Multiple well-defined pleural based lesions are seen along the right hemithorax and largest measuring 7 × 5 cm [Figure 1b]. Few small nodular lesions in the bilateral lower zones and bronchiectasis seen in the right middle zone. Fine needle aspiration cytology smears done from the pleural-based nodules showed macrophages, that contained numerous small-sized intracellular organisms having a single nucleus surrounded by a clear halo [Figure 2]. Multinucleate giant cells and epithelioid histiocytes were also noted. Gomorial methenamine silver stain shows fungal organisms as rounded black colored structures in the cytoplasm confirming the diagnosis of histoplasmosis [Figure 3].

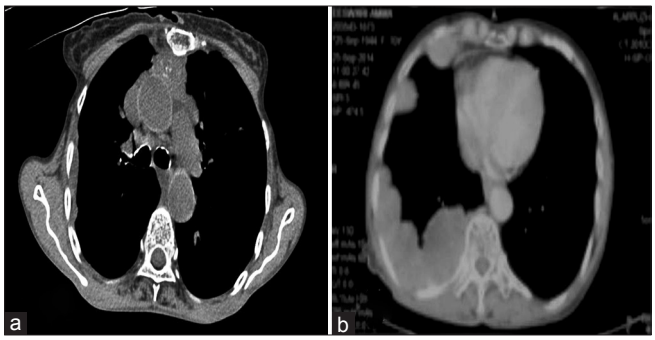

Figure 1: (a and b) Computed tomography scan of thorax showing mass lesion in the anterior mediastinum and multiple well-defined pleural-based lesions along the right hemithorax

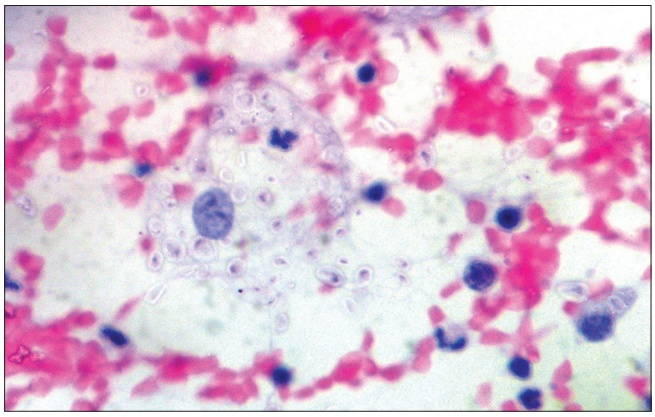

Figure 2: Fine needle aspiration cytology smears from the pleural-based nodules shows macrophages, that contained numerous small-sized intracellular organisms having a single nucleus surrounded by a clear halo

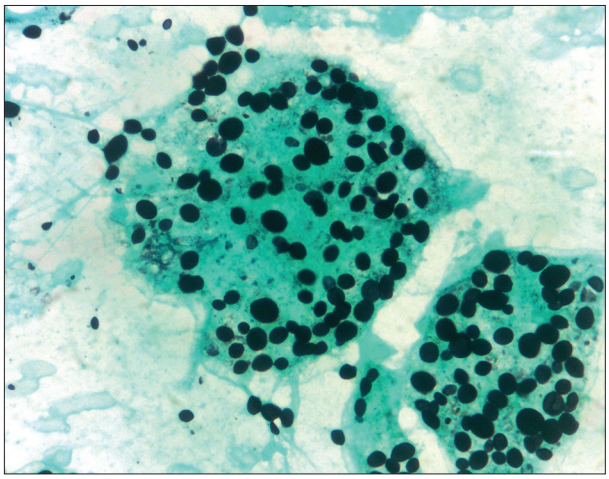

Figure 3: GMS staining shows budding cells with black coloured structures in the cytoplasm

Histoplasmosis is a fungal infection caused by *Histoplasma capsulatum*. Histoplasmosis was first described by an American physician, Samuel Darling, who was working in the Canal Zone in Panama.[2] Histoplasmosis usually develops through inhalation of fungal spores and hematogenous spread to reticuloendothelial system develops within a week or month. The majority of infected persons have an asymptomatic, self-limiting illness. In immunocompromised patients *H. capsulatum* may present as disseminated disease. Patients with CD4 counts of <150 cells/μl are at most risk. The spectrum of disseminated infection includes acute, severe, life-threatening sepsis and chronic, slowly progressive infection. Histoplasmosis can involve every organ system during the course of dissemination. Clinical pneumonia occurs in those with exposure to a large number of infecting spores. Resolution of the pneumonia often leaves calcified pulmonary nodules, calcified mediastinal lymph nodes. Chronic disease, which mimics tuberculosis. Delayed manifestations arise months or years after the primary infection.[3]

The diagnosis of histoplasmosis requires a high index of suspicion. The differential diagnosis of histoplasmosis include malignancy, tuberculosis or sarcoidosis. Culture remains the gold standard for the diagnosis of histoplasmosis, but it requires a lengthy incubation period. The optimal treatment for histoplasmosis varies according to the patient's clinical syndrome. Most infections are self-limited and require no therapy. However, patients who are immunocompromised usually require antifungal therapy. Acute severe pulmonary histoplasmosis is managed with liposomal amphotericin followed by itraconazole for 12 weeks and chronic cavitory pulmonary histoplasmosis is managed with itraconazole for 12 months.[4]

Our case revealed that diagnosis of histoplasmosis is a major challenge even in the present era and required multidisciplinary team with major resource facilities. Mortality associated with severe histoplasmosis without treatment is 80% but can be reduced to <25% with anti-fungal therapy.[5]

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