HISTOPLASMOSIS IN ACQUIRED IMMUNODEFICIENCY SYNDROME

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Introduction

Opportunistic infections are common and major causes of morbidity in patients with Acquired Immunodeficiency Syndrome (AIDS). Histoplasmosis is a common opportunistic infection in patients with Human Immunodeficiency Virus (HIV) infection who reside in areas where *Histoplasma capsulatum* is endemic [1]. Defence against *Histoplasma capsulatum* is largely by cell mediated response. The infection can therefore be prolonged in individuals with defective T cell mediated response, including those with AIDS. In them it becomes a progressive, potentially fatal infection [2]. Histoplasmosis is generally a late manifestation of HIV infection. Diagnosis of Histoplasmosis is best made by visualization of yeast in tissue or by culture [3].

Case Report

A 28-year-old sailor became symptomatic in May 1998 with fever and weight loss of one month duration. He also had cough, loose motions, malaise and oral ulcers. The individual was unmarried and belonged to Assam. He gave history of unprotected peno-vaginal sexual contact with commercial sex workers in Mumbai from 1991 to 1993. In March 1993 he was treated for acute viral hepatitis (HbsAg+ve) and investigation done at this time showed him to be positive for HIV by ELISA and Western Blot tests. At the time of admission he was emaciated with weight being 41 kg (height 164 cms). He had fever, pallor, generalised lymphadenopathy and shallow ulcers on the palate. There was hepatosplenomegaly and a 6 cm diameter firm, round mass around umbilicus suggestive of abdominal lymph node mass. Chest was clear. Fine needle aspiration cytology (FNAC) of lymph node from neck showed a few epithelioid cell granulomas and isolated monocytes in background of loose lymphoid cells and necrotic debris. Yeast form of *Histoplasma capsulatum* was observed both intracytoplasmic in monocytic cells and also lying extracellularly in small clusters (Fig-1). Bone marrow aspirate showed Histoplasma lying intracellularly within mononuclear cells. PAS and methanamine silver stain were positive. Fungal culture was sterile. Chest radiograph showed hilar lymphadenopathy and multiple infiltrative lesions both middle zones and right upper zone. Liver function test and cerebro spinal fluid examination were normal.

Ultrasonography study of abdomen showed hepatosplenomegaly and enlarged aortocaval and mesenteric lymph nodes. CT scan of chest showed well defined nodular infiltrates in both lung fields relatively sparing bases. Many of the infiltrates showed calcifications. Mantoux test was non-reactive. Haemoglobin was 6.0 gm/dl and peripheral smear showed microcytic hypochromic anaemia. His CD4 lymphocyte count was 82/cmm. Sputum smear examination for mycobacterium tuberculosis was negative. He was treated with Inj Amphotericin B 40 mg IV daily for six weeks and showed symptomatic improvement. Fever subsided and there was weight gain of four kilograms. There was partial resolution of abdominal lymph node mass. He was not on any anti-retro viral drugs during his entire period of illness.

Discussion

It is important to take Histoplasmosis into account in the differential diagnosis of prolonged fever and weight loss in patients with HIV infection from endemic zones. Histoplasmosis is a life threatening disease in patients with advanced HIV infection. Histoplasmosis is usually disseminated in AIDS patients. The endemic areas of Histoplasmosis include part of the USA, West Indies, Central and South America, Africa, India and the Far-East [4]. Our patient belonged to Assam where Histoplasmosis has been reported. In AIDS patients with Histoplasmosis symp-

Fig. 1 : FNAC of cervical lymph node showing phagocytes stuffed with minute yeast forms of histoplasma capsulatum

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toms may be non-specific with weight-loss and fever, although some clues such as hepatosplenomegaly or multiple skin lesions may be seen [5].

Our patient had presumably acquired HIV infection between 1992 and 1993 through unprotected heterosexual contact with commercial sex workers in Mumbai. Clinical presentation in 1998 of Histoplasmosis was with fever, cough, loose motions, oral ulcers and weight loss. He had anaemia, generalised lymphadenopathy, hepatosplenomegaly and oral ulcers and radiological evidence of pulmonary infiltrative lesions with calcification. *Histoplasma capsulatum* was demonstrated in aspirate from lymphnode and bone marrow examination. The patient was severely immunocompromised with a CD4 count of 82/cmm. There was no evidence of any other opportunistic infection. The patient responded to injection amphotericin B with disappearance of fever, weight gain and a sense of well being.

In a report of 46 residents of San-Francisco of AIDS with Histoplasmosis, presentation was with prolonged fever, chills, sweats, cough or dyspnoea, gastro intestinal complaints, malaise or weight loss. Physical examination and imaging studies were notable for hepato splenomegaly, lymphadenopathy and abnormal pulmonary findings in more than half of the patients. Laboratory studies revealed a high rate of cytopenia, elevated serum lactate dehydrogenase, abnormal liver functions tests, respiratory alkalosis with hypoxaemia

and median CD4 lymphocyte count of 36/cmm [6].

Treatment with amphotericin B or itraconazole is effective if the patient is not seriously ill at the time of diagnosis but the mortality approaches 50% or more in those with multi organ failure. In disseminated Histoplasmosis in setting of AIDS it is necessary to use long term suppressive therapy with itroconazole or intermittent amphotericin B after induction of remission, otherwise relapses will normally occur. Prophylaxis with itroconazole may be appropriate in areas with an incidence of Histoplasmosis.

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