Different Faces of HIV in a Single Patient

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Abstract

Opportunistic infections (OIs) are a major cause of morbidity and mortality in patients with human immunodeficiency virus (HIV) infection. The most common opportunistic infection is tuberculosis, followed by candidiasis, infections causing diarrhoea, and pneumocystis carinii pneumonia (PCP). We hereby report the case of a 34-year-old male with clinical stage-IV acquired immunodeficiency syndrome (AIDS) who had multiple OIs including visceral leishmaniasis (VLs) an uncommon co-infection in the Indian scenario. This patient also had features of multiple pulmonary infections-bacterial, mycobacterial and fungal, a rare clinical problem in HIV.

Keywords: HIV, Opportunistic infections, Single patient.

Introduction

HIV infection is a global pandemic. The 2006 estimates suggest national adult HIV prevalence in India is approximately 0.36 percent, amounting to between 2 and 3.1 million people. The risk behaviors and vulnerabilities of specific populations and their networks determine the dynamics of HIV epidemics. HIV seems to be affecting the economically productive, sexually active group; and majority of patients are migrant males, thus having a tremendous impact on the livelihood of the affected family.[1] Opportunistic infections (OIs) are the major cause of morbidity and mortality in patients with HIV infection. The progression toward AIDS in HIV-1-infected individuals appears to be directly related to CD4 cell count decline and HIV-1 viral loads in the plasma and lymphoid tissues.[2] The spectrum of OIs depends upon the CD4 cell count and a highly significant inverse correlation has been observed. We hereby report the case of a 34-year-old male who was initially diagnosed as a case of VL and found to be HIV positive, had multiple OIs associated with bicytopenia and ATT induced hepatitis.

Case Report

A 36-year-old male, resident of Delhi, presented with the complaints of anorexia, weight loss, heaviness in the left upper abdomen, and intermittent cough with altered colored, foul smelling sputum associated with low-grade fever with typical evening rise of temperature for the last 2-3 months. He now developed hemoptysis since 2 days and was brought to our hospital. There was no history of petechiae or purpurae, vomiting, loose motions, chest pain, dyspnea, or tuberculosis in the past and the patient also denied any history of contact with tuberculosis.

On examination, he was ill-looking. Signs of dehydration, sternal tenderness, and lymphadenopathy were absent. He was normotensive, and had severe pallor. His respiratory system revealed bronchial breath sounds in left infra-mammary region and massive splenomegaly on per abdominal examination. Eye examination showed bilateral cataract. The rest of the systemic examinations were unremarkable.

Blood investigations revealed hemoglobin of 6.9 g/dL, total leucocyte count 3300/mm^3, differential leucocyte count P72L23E3M2, platelet count 48000/mm^3. Serum electrolytes (sodium/ potassium) were 142/4.0 mEq/L. His liver and kidney functions were deranged. His urea was 93 mg with creatinine of 2.3 mg/dL. His LFT revealed a total serum bilirubin of 1.0 mg/dL, serum alanine aminotransferase 61 U/L, serum aspartate aminotransferase 158 U/L, and serum alkaline phosphatase 672 U/L. Peripheral smears showed dimorphic anemia. Urine routine showed no active sediment.

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Urine culture was sterile but bactec showed growth of candida parapsilosis. Initial sputum culture was positive for pseudomonas aeruginosa, *Escherichia coli*, and non-candida albicans. Subsequently, sputum culture showed growth of *Aspergillus fumigatus*.

Indirect fluorescent antibody test for *kala azar* was positive (1:400) and bone marrow smear examination revealed leishmania donovani (LD) bodies.

ELISA for HIV was positive, and CD4 cell count was 77/μL.

Chest X-ray showed cavitatory lesion left lung. Ultrasound abdomen showed splenomegaly with multiple abdominal lymphadenopathy. NCCT chest and abdomen showed loculated hydropneumothorax in upper anterior left pleural cavity, bronchiectasis, cavitation with air fluid level in left lower lobe of lung, centrilobular nodules in bilateral lung and splenomegaly with abdominal lymphadenopathy. HRCT chest revealed bilateral tree in bud opacities with encysted pneumothorax in left pleural cavity with areas of consolidation and cavitation in left lung field. Both imagings were suggestive of pulmonary kochs. But sputum smear examinations were negative for AFB stain. Repeated arterial blood gas analysis had shown hypoxemia.

There was no evidence of cytomegalovirus retinitis on fundus examination. Sputum smear examinations were negative for pneumocystis carinii.

A final diagnosis of clinical stage-IV AIDS associated with multiple OIs: pulmonary tuberculosis with supra-added bacterial infection, candida albicans and non albicans, *A. fumigatus* and visceral leishmaniasis was kept. He was started on trimethoprim/ sulphamethoxazole (TMP/ SMX) and azithral propylaxis along with fluconazole which was subsequently changed to liposomal amphotericin B, intravenous antibiotic according to culture sensitivity and modified antitubercular therapy (ATT) as he developed drug-induced liver injury. ART was started after two weeks of ATT along with supportive care. Patient responded to the above therapy and was discharged; however he was lost to follow up.

Discussion

People with advanced HIV infection are vulnerable to infections or malignancies that are called “opportunistic” because they take advantage of the opportunity offered by a weakened immune system. Various treatments and prophylaxis-some simple and low-cost, others highly complex and expensive-exist to counter the most common opportunistic diseases. The sequence of pulmonary infections (bacterial, viral, fungal and/ or mycobacterial) occurring in HIV-infected individuals parallels the depletion of CD4+ lymphocytes.[3]

TB may negatively impact the natural history of HIV infection. Several studies have indicated that TB co-infection increases the risk of HIV progression and death, particularly in persons with untreated HIV disease. The clinical presentation of pulmonary TB can vary widely in both immunocompetent and immunocompromised hosts. In general, the presentation in HIV-infected patients is similar to that seen in HIV-uninfected patients, although the signs and symptoms (such as fevers, weight loss, and malaise) may be attributed to HIV itself and the possibility of TB overlooked. In HIV-infected patients, clinical manifestations of pulmonary TB reflect different levels of immunosuppression. Earlier in the course of HIV disease, TB is more likely to present as classic reactivation-type disease, whereas patients with advanced immunosuppression are more likely to present with findings consistent with primary TB.

The prevalence of extrapulmonary TB and disseminated TB are both increased in HIV-infected patients. Low CD4 cell counts are associated with an increased frequency of extrapulmonary TB, positive mycobacterial blood cultures, and atypical chest radiographic findings, reflecting an inability of the impaired immune response to contain infection. Infection may be present in bone, brain, meninges, gastrointestinal tract, lymph nodes, or any viscera.[4]

One of the major threats to control of visceral leishmaniasis (VL) is its interaction with HIV infection. VL has emerged as an important opportunistic infection associated with HIV. In areas endemic for VL, many people have asymptomatic infection. A concomitant HIV infection increases the risk of developing active VL by between 100 and 2320 times. VL/HIV co-infection has important clinical, diagnostic and epidemiological implications. The two diseases are mutually reinforcing: HIV-infected people are particularly vulnerable to VL, while VL accelerates HIV replication and progression to AIDS. The risk of treatment failure for VL is high, regardless of the drug used, and all co-infected patients will relapse-and eventually die-unless they are given antiretroviral therapy (ART). Indirect methods of diagnosis such as serological tests for VL frequently fail; direct methods such as aspirations (bone marrow, lymph node or splenic) are reliable but are invasive, require skilled

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microscopy, and have less value in treated and relapsing patients. Further, co-infected patients can serve as human reservoirs, harboring numerous parasites in their blood and becoming a source of infection for the insect vector. The use of highly effective systemic therapy for leishmaniasis is important. Liposomal amphotericin B is FDA-approved for treatment of visceral leishmaniasis. Regimen for immunosuppressed patients consists of 4 mg per kg daily on days 1-5, 10, 17, 24, 31, and 38 (total dose of 40 mg/kg). The best way for travelers to prevent infection is to protect themselves from sandfly bites. Personal protective measures include minimizing nocturnal outdoor activities, wearing protective clothing, and applying insect repellent to exposed skin.[5]

Candidiasis is a common opportunistic infection in HIV-infected patients. The spectrum of Candida infection is diverse, starting from asymptomatic colonization to pathogenic forms. The low absolute CD4+ T-lymphocyte count has traditionally been cited as the greatest risk factor for the development of oropharyngeal candidiasis and current guidelines suggest increased risk once CD4+ T lymphocyte counts fall below 200 cells/µL. Gradual emergence of non-*albicans Candida* species as a cause of refractory mucosal and invasive Candidiasis, particularly in patients with advanced immunosuppression and problem of resistance to azoles and other antifungal agents in the Candida species is a point of concern.[6]

HIV-related aspergillosis may present with a wide variety of clinical syndromes. Lower respiratory tract infections due to *Aspergillus spp*. may present with one of five clinical syndromes including: i) invasive pulmonary aspergillosis; ii) obstructing bronchial aspergillosis; iii) ulcerative or pseudomembranous tracheobronchitis; iv) aspergilloma; v) and rarely empyema. Diagnostic procedures to be considered in patients with compatible presentations include: CT scan, preferably high-resolution computed tomography; bronchoscopy with bronchoalveolar lavage; and possibly percutaneous transthoracic needle aspiration. The definitive diagnosis of Aspergillus infection requires both microscopic invasion seen in tissue and isolation of the organism by culture. Amphotericin B at a dosage of 1 mg/kg daily has been the treatment of choice for most forms of IA, regardless of the cause of the underlying immunosuppression.[7]

Conclusion

In the present scenario, as India is contributing significantly to the global burden of HIV, high index of clinical suspicion is required for early diagnosis and prompt treatment of such cases to prevent uneventful outcome.

Conflict of Interest: Nil

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