Cryptococcal mesenteric lymphadenitis in an immunocompromised host

Sir,
Cryptococcal infection is one of the most common life-threatening diseases seen more commonly in patients with acquired immunodeficiency syndrome (AIDS) and in transplant patients on immunosuppressive agents or other forms of immunosuppression. The infection starts in the respiratory tract but secondarily involves the central nervous system, lungs, skin, lymph nodes, bone marrow, gastrointestinal tract, retina, liver, spleen, and other parts of the body.[1] Most cryptococcal infections present as meningoencephalitis, followed by pulmonary and skin infection. However, even in disseminated cryptococcosis, lymph nodes are usually spared. In addition, selective involvement of lymph nodes, so called “lymphonodular cryptococcosis,” is a rare entity and was reported more frequently in children.[2] Although there have been several reported cases of cryptococcal lymphadenitis,[3] lymph-node involvement by cryptococcus is not a common manifestation in AIDS patients as compared to other infections.[4]

A 17-year-old female presented with history of acute pain abdomen, anorexia, and weight loss for the past 5 months. The patient was being treated for tuberculosis since 3 months and was not responding to therapy. On physical examination, no lymph nodes were palpable. Ultrasonography of the abdomen was done, which showed enlarged mesenteric lymph nodes. Percutaneous ultra sono graphy (USG)-guided fine needle aspiration (FNA) of mesenteric lymph node was performed to rule out tuberculosis and lymphoma. Cultures and PCR for mycobacterium were requested. Cytological examination of FNA smears revealed abundant histiocytes, epithelioid granulomas, and multinucleated histiocytic giant cells in a necrotic background. Both histiocytes as well as giant cells revealed abundant intracytoplasmic as well as extracellular small spores which were 2–15 μ in diameter, ovoid to spherical with a clear halo around them [Figure 1]. No abnormal lymphoid infiltrate was seen. Fungal stains with periodic acid-schiff (PAS) and diastase showed numerous fungal yeast forms, which were both intra and extracellular. These yeasts were variably sized and showed frequent, unequal sized narrow-based budding [Figure 2]. The capsule was positive for mucicarmine. Thus the morphology was consistent with the diagnosis of Cryptococcus neoformans. ZN stain for tubercle bacilli as well as culture and polymerase chain reaction (PCR) for mycobacterium were negative. Serological tests for HIV virus were positive. In this paper, the patient was admitted because of acute pain in abdomen, the rare presentation of mesenteric cryptococcosis. Therefore, a prompt diagnosis is mandatory in such situations for early initiation of treatment.

The laboratory diagnosis of cryptococcal infection includes the use of special stains, such as India ink, PAS-AB and mucicarmine stains, and serologic detection of cryptococcal antigen by latex agglutination and culture, which confirms the subtype of the organism. To this list, fine needle aspiration serology (FNAC) should be added as another
Letters to Editor

Coinfection of hepatitis B and hepatitis C in human immunodeficiency virus infected patients in a tertiary care hospital in North West India

Sir,

Due to shared routes of transmission, human immunodeficiency virus (HIV) patients have a high probability of getting coinfected with hepatitis B virus (HBV) and hepatitis C virus (HCV). Among the 33.3 million HIV-infected patients worldwide,[1] 2–4 million are estimated to have chronic HBV infection, while 4–5 million are coinfected with HCV. They differ in their prevalence by geographic region and the efficiency by which certain types of exposures transmit them.[2] Improved survival due to success of highly active antiretroviral therapy (HAART) in HIV patients has enabled infections such as chronic viral hepatitis to become a major source of comorbidity.

A study was carried out in the Department of Microbiology, SMS Medical College, Jaipur, from January 2008 to December 2009, where clinically suspected patients were tested for HIV antibodies after pretest counseling and informed consent as per World Health Organization (WHO) guidelines. A total of 600 treatment naïve HIV-infected patients were included in the study. Clinical staging of the disease was done according to Centers for Disease Control and Prevention (CDC) guidelines.[3] CD4 cell count estimation was done by flow cytometry. All patients were tested for hepatitis B surface antigen (HBsAg) and anti-HCV antibodies by enzyme-linked immunosorbent assay. Sera of HBsAg positive patients were further screened for presence of anti-HBe antibodies and HBe antigen.

Serum samples from 600 HIV negative healthy blood/organ donors (controls) were screened for HBsAg and anti-HCV antibodies and their prevalence was found to be 1.3 and 0.16%, respectively. The mode of transmission was heterosexual in 552/600 (92%) followed by transfusion of blood products 3/600 (0.5%) and in the rest it was unidentified. Majority of coinfected patients were between 31 and 40 years of age.

Of the 600 HIV seropositive patients, 63 (10.5%) were positive for HBsAg. In this group, 42/63 (66.66%) of patients belonged to group C and 30/42 (71.4%) patients of these were HBeAg positive. CD4 counts were significantly lower in the HIV/HBV coinfected group as compared to HIV alone. HBV has considerable potential to activate HIV replication directly. In addition, chronic and persistent activation of the immune system by an ongoing immune response (e.g., an infection with a hepatotropic virus) increases the expression of HIV and may therefore accelerate immunodeficiency and the course of HIV infection.[4] HCV was detected in 6/600 (1%) of the HIV-positive patients. It appears that HIV-associated immunosuppression stimulates

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