

A Rare but Sinister Case of Hodgkin Lymphoma Transforming into Large B-cell Lymphoma in HIV Infected Patient

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ABSTRACT

A dual diagnosis of cancer and HIV places a greater psychosocial burden on the patient. From a medical perspective, it complicates treatment for both diseases. Potential drug interactions, compounded side effects, and chemotherapy's tendency to negatively impact CD4 count or HIV viral load, can all complicate treatment. It is imperative to advocate a keen and subtle vigilance while managing such patients to enhance the overall care for the patients. Here, we report such a case in a 36-year-old man who presented with HL as first manifestation of HIV infection that later transformed into DLBCL despite successful virologic suppression with ART.

Keywords: Hodgkin Lymphoma, AIDS- Defining Cancer, Diffuse Large B Cell Lymphoma

Introduction

Patients with human immunodeficiency virus (HIV) infection are more prone to developing a number of viral-induced malignancies. Immunosuppression is considered the most relevant factor in promoting oncogenesis in HIV population. Although the prognosis of HIV-associated malignancies remain poor, introduction of highly active antiretroviral therapy (HAART) combined with chemotherapy allows a substantial number of these patients to be cured.

Here, we describe a peculiar case of a patient who presented with Hodgkin Lymphoma (HL), a non-AIDS- defining cancer as first clinical evidence of HIV infection that later was transformed into Diffuse large B cell lymphoma (DLBCL), an AIDS- defining cancer despite receiving HAART and a successful viral suppression.

A search of the literature yielded no similar published cases, with this being the first report of this kind.

Case Report

A 36-year-old heterosexual Indian male was admitted for the evaluation of shortness of breath, loss of weight and appetite for 3 months. A physical examination at the time was unremarkable except for enlarged bilateral axillary and cervical lymph nodes. His vital signs were: blood pressure, 110/80 mmHg; pulse rate, 98/min; body temperature; 38.0 C and respiratory rate, 20/min. There was no localized tenderness of the abdomen on physical examination.

Laboratory work up showed a haemoglobin level of 7.7 g/dl, WBC count 1400/ μ L, platelet count 38000/ μ L, ESR was more than 100 mm/hr.

Serological investigation documented an HIV infection, with initial HIV RNA level of 28,000 copies/mL and 110 CD4+ lymphocyte s/ μ L.

Biopsy specimens of the left axillary node and bone marrow demonstrated Hodgkin Reed Sternberg cells (HRS cell) consistent with Hodgkin's lymphoma (Figure 1). On Immunohistochemistry, cells are positive for CD15, CD 30, PAX5 (weak) and negative for CD20 and EMA. Abdominal CT scan and PET/CT performed at the time were unremarkable. Thus, he was diagnosed with HIV associated stage IV classical Hodgkin lymphoma. Patient was started on anti neoplastic therapy with Adriamycin, bleomycin sulphate, vinblastine sulphate, and dacarbazine (ABVD) and concomitant anti retroviral therapy with efavirenz/emtricitabine/tenofovir disoproxil. The patient then underwent 3 cycles of ABVD therapy. But 20 days after the third cycle of chemo, patient presented with progressive abdominal pain and high grade fever. Physical examination at the time revealed grossly enlarged liver and spleen. Blood investigations showed worsening pancytopenia, deranged liver function tests, raised LDH levels (1227 IU/ml), CD4+ lymphocyte 21/ μ L. No HIV RNA was detected (<20 copies/mL). Abdominal CT scan was performed that demonstrated multiple liver lesions (Figure 2) and a core needle biopsy of one of the liver masses demonstrated cells that are positive for CD20, BCL-6, PAX-5, consistent with DLBCL (Germinal centre type)

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(Figure 3). Lymph node biopsy was performed again that revealed persistence of HL in the lymph node. Further staging confirmed stage IV DLBCL with high International Prognostic Index score. Taking into consideration the new clinical findings, a final diagnosis of HIV associated HL transforming into DLBCL was delineated. Specific chemotherapy with R-EPOCH (Rituxan, etoposide, prednisone, vincristine, cyclophosphamide and doxorubicin) was begun; of which she has received four cycles so far followed by near complete resolution of her large B-cell lymphoma. We are contemplating administration of a total of six cycles of R-EPOCH.

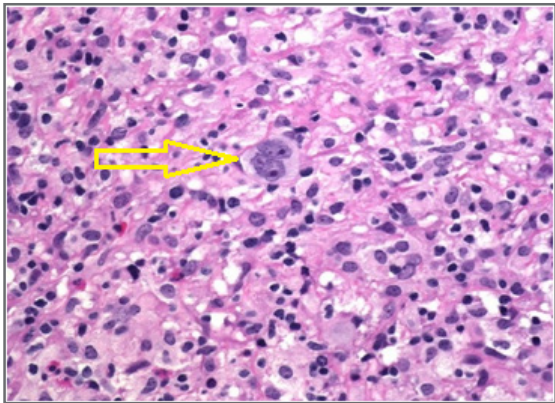


Figure 1: Histopathological features of lymph node biopsy. The architecture is effaced by polymorphous population comprised of lymphocytes, plasma cells and histiocytes. Many Reed Sternberg cells (shown by arrow) are seen scattered and loosely forming clusters. (Haematoxylin and Eosin stain)



Figure 2: Contrast enhanced CT scan of abdomen shows low-attenuated lesions in liver

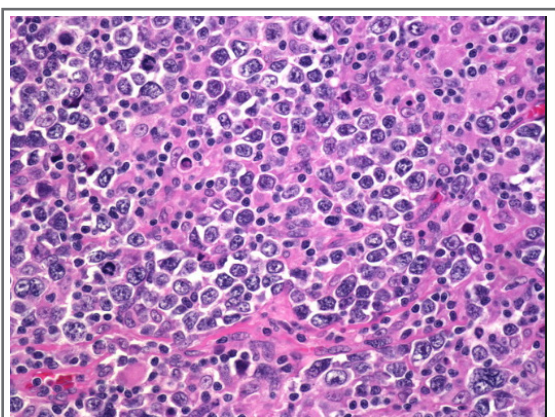


Figure 3: Histopathological features of liver biopsy. There is diffuse infiltration with sheets of atypical mononuclear cells with a high N:C ratio; both in the parenchyma and portal tract. (Haematoxylin and Eosin stain)

Discussion

The introduction of HAART changed the trend in malignancies in people living with HIV/AIDS in many countries including India [1-3]. The incidence of cancers is higher when patients have lower CD4 cell counts, initially present with cancer or revisit after loss to follow-up [4]. Our patient presented with a HL as first manifestation of HIV infection; initial CD4+ lymphocyte count at diagnosis was 110 cells/μL. At the time of DLBCL diagnosis, CD4+ lymphocyte count was 21 cells/μL although no HIV RNA was detected. These findings suggest that multiple malignancies can occur in PWHA despite the administration of HAART and successful virologic suppression. Our case highlights the importance of monitoring and accurate diagnosis of new symptoms in HIV patients even in the situation of complete viral suppression with ART, that would help in extending patient survival. In addition, the incidence of transformation in Nodular lymphocyte predominant HL (a subtype of HL) is reported to be much higher (30%) than in classical HL (1%) [5]. Our case report demonstrates a unique case of such a transformation and underscores that all physicians treating patients with HL, especially patients with classical HL, should be aware of the possibility of transformation into DLBCL that would aid in timely diagnosis and would prevent debilitating disease course.

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