



HIV/AIDS and Cancer: The Hidden Epidemic

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Commentary

People infected with Human Immunodeficiency Virus (HIV) are at high risk to develop different types of neoplasms even the widespread of Highly Active Antiretroviral Therapy (HAART). Some of these tumors are included as AIDS-defining malignancies, because high rate of presentation and clinical and histopathological manifestations that generally differs with the general population. AIDS-defining malignancies include Kaposi's Sarcoma (KS), Non-Hodgkin Lymphomas (NHL), the invasive carcinoma of the cervix and its equivalent in men who have sex with men (MSM) named as Anal Squamous Cell Carcinoma (ASCC). All these neoplasms are related in its pathogenesis with viral chronic infections. Human Herpes-virus type-8 is a cofactor associated with all the clinical forms of KS (classic, sporadic, iatrogenic or post-transplant and epidemic or AIDS-associated KS). Finally, invasive cervix carcinoma and ASCC are strongly associated with chronic Human Papillomavirus (HPV) infection [1].

The clinical course of AIDS-related KS range from a minimal cutaneous or mucosal involvement to extensive and severe disseminated disease with lymph nodes, lungs and gastrointestinal tract involvement. In some areas, the prevalence of KS has declined since the advent of HAART [2].

HIV-related NHL is strongly associated with the Epstein Barr virus (EBV) infection, in different percentage according with the histopathological subtype. Since 1996, the introduction and widespread of HAART improve the prognosis and survival of HIV infected patients with both, KS and NHL [3,4]. Approximately 25% to 30% of all HIV/AIDS subjects develop lymphomas during the natural history of the disease. NHL being more frequent than Hodgkin disease in a rate of 4:1 [5]. NHL-related with AIDS presents some clinical characteristics that differ with the general population. The most important clinical characteristic of AIDS-associated NHL is the frequent extranodal involvement as a primary manifestation of the disease [6]. HAART should be indicated as soon as NHL is diagnosed, before chemotherapy is initiated. Additionally, a few number of cases have been reported including patients with clinical remission with HAART alone [7,8].

Several epidemiological studies have shown that patients infected with HIV have a high risk of develop other cancers [9,10]. HIV/AIDS patients has an increased risk of non-AIDS defining solid tumors, especially lung cancer, that is the most common non-AIDS defining cancer in people with HIV infection. These malignancies named as non-AIDS-defining tumors include Hodgkin Lymphoma (HL), lung carcinoma and liver cancer. Approximately, 50% of the estimated non-AIDS-defining cancers involve the lung, liver or are cases of HL [11].

Lung cancer is the most frequent solid tumor in this kind of neoplasms. The risk to develop lung cancer seems to be higher in HIV-infected patients compared with the general population of the same age. Men have significantly most risk than women with a male to female ratio of 9/10:1 [12,13]. Similar to other non-AIDS-defining cancers there is no clear relationship between the grade of immunosuppression and the risk of lung cancer [14]. Also, HIV patients with lung carcinoma are younger compared with the general population with a predominance between the 3th to 5th decades of the life.

The distribution of histopathological subtypes of lung cancer in HIV-infected patients differs to the usual prevalence in the non-HIV infected people. Adenocarcinoma appears to be the most frequent subtype followed by the squamous cell carcinoma [15].

HL is one of the most common non-AIDS-defining malignancies in patients infected with HIV. The incidence of HL is increased approximately 10-fold compared with the general population. Paradoxically, the incidence has increased in the HAART era [11,16-18]. The median of CD4-t-cell count is generally increased at the time of HL diagnosis compared with the same parameter in NHL [19,20]. This finding is associated with the diagnosis of HL in patients under HAART,

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with undetectable plasma viral load and CD4 more than 200 cell/ μ L [21]. Similar to other non-AIDS-defining cancers there are some differences in the histopathological subtypes between HIV-HL and HL in the general population [17]. Mixed Cellularity (MC) subtype is the most commonly observed in the HIV patients in contrast with HL in general population in which nodular sclerosis is the predominant subtype. While some number of cases of the classical HL associated with AIDS has been diagnosed in the last years, MC predominance continues to be higher [22]. EBV is strongly related with the pathogenesis of HL in AIDS patients and Reed-Sternberg cells mainly express EBV-encoded genes (EBNA and LMP). Clinical presentation and diagnosis of HL-HIV not differ that the general population. Approximately 65% to 80% of cases presents with advanced stages of the neoplastic disease and “B” symptoms that can confuse with several opportunistic infections that can compromise HIV-infected patients, especially, tuberculosis, other disseminated mycobacteriosis and histoplasmosis. The bone marrow is more frequent involved in HIV-infected patients and may be the only site of the disease. For this reason, HL should be considered in all HIV patients with fever and pancytopenia [23].

Finally, it is important to consider that KS continue being the most frequent HIV-associated neoplasm in patients who do not receive HAART, or those that showing a poor adherence or patients who do not know their serological status related to the retrovirus. In the other hand, B-NHL and non-AIDS defining malignancies as a result of the extended use of HAART, can appears as the most common malignancies in individuals with HIV infection [9,24]. The combination of HAART and chemotherapy is the gold standard treatment for all AIDS-associated malignancies.

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