

Human Immunodeficiency Virus Infection and Chronic Myeloid Leukemia: Is there an Association?

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It is well known that there is an increased risk of malignancy in individuals who are Human immunodeficiency virus (HIV) seropositive [1,2]. This is particularly true with regard to AIDS (Acquired Immune Deficiency Syndrome) defining malignancies such as Kaposi's sarcoma, high grade B-cell non-Hodgkin lymphoma and invasive carcinoma of the cervix, and non-AIDS defining/AIDS-associated malignancies such as Hodgkin lymphoma, anal carcinoma and squamous cell carcinoma of the conjunctivae [1-4].

Chronic myeloid leukemia (CML) and HIV occurring in association has rarely been described [5]. Indeed, there are less than 50 reported cases in the literature, with more than half of these patients being described from sub-Saharan Africa [5,6].

HIV as well as AZT (azidothymidine/zidovudine) an antiretroviral agent are known to cause of myelodysplasia. However, there are no reports of the myelodysplasia evolving to CML [7-9]. Other risk factors including radiation and immunosuppression do not play a role with regard to this association. It is therefore plausible to assume that the association between CML and HIV is likely to be coincidental rather than causal.

Recently, we described the largest reported series of 18 patients with HIV-CML from Chris Hani Baragwanath Academic Hospital (CHBAH), Soweto, and Johannesburg, South Africa [5]. This accounts for 7.5% of all the CML patients seen at the Clinical Hematology unit, Department of Medicine at this single centre between January 1991 and June 2011. There were 10 males and 8 females with a male to female ratio of 1.25:1. The patients presented at a younger median age of 37 years (14-47). In 72% of the patients, the diagnosis of the HIV was new, made simultaneously at the time of the diagnosis of CML. In general, the patients presented with more advanced disease compared to their seronegative counterparts, based a high risk Sokal score in 89% of the patients [5,10] and a higher number of patients with accelerated and blast phase disease – 44%. There is a higher risk of infections such as Tuberculosis in this group of patients. The mean CD4 count was 868/ul (46-2163). This higher than expected CD4 count is likely a reflection of the high white cell count rather than a surrogate of the immune status of the patient. The latter 6 patients in this series were treated with TKI's (tyrosine kinase inhibitors) and combination Antiretroviral Therapy (cART) used in all patients irrespective of the CD4 count, with a more favourable outcome than the former 12 patients who did not have access to the TKI's, in particular Imatinib [5].

There has been a significant and major advance in the treatment of CML and HIV in the past decade. However, both CML and HIV may cause myelosuppression and immunosuppression. Despite this, and noting the potential drug interactions, the TKI's and cART have been used safely and effectively, resulting in renewed optimism in the management of HIV-CML and improved long term survival [11-13].

There is likely to be underreporting of HIV-CML, particularly from areas with a high HIV seroprevalence, such as sub-Saharan

Africa. Since the publication of our series, we have had a further 10 patients with HIV-CML diagnosed at CHBAH (i.e. from July 2011 to May 2014).

The association between CML and HIV is likely to be coincidental. Clinicians need to be made aware of the association of HIV-CML, the more aggressive and advanced presentation of the patients, yet currently, the better clinical, genetic and molecular outcome with concomitant TKI's and cART, albeit the response being less favourable than in HIV seronegative CML.

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