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The prevalence of HIV-1 DNA in AIDS-related lymphoma and Kaposi Sarcoma throughout the AIDS epidemic

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Background

Chronic inflammation is linked to tumorigenesis for many cancer types and likely contributes to tumor development in the HIV-infected patient population. AIDS-related lymphoma (ARL) and Kaposi Sarcoma (KS), two AIDS-defining cancers, are associated with the tumor viruses EBV and KSHV, respectively. However, EBV is only detectable in ~40% of ARLs and KSHV alone is not sufficient for KS development. Recent studies have shown that HIV is localized to tumor associated macrophages (TAM), not malignant B cells, in a portion of EBV-negative ARLs suggesting, that HIV infected TAM may play a role in tumorigenesis. The goal of this research was to determine the prevalence of HIV+ ARL and KS throughout the AIDS epidemic and examine tumor associated HIV for unique genetic signatures.

Material and methods

Whole genomic amplified DNA from ARL and KS biopsies was used for quantitative HIV gag gene amplification. The 3' *env-LTR* segment of HIV-1 genomes from tumor and non-tumor tissues from two patients that died of ARL were sequenced and Bayesian phylogenies were inferred using BEAST. All specimens were provided by the AIDS and Cancer Specimen Resource.

Results

Of the 119 ARL and 91 KS biopsies, 45% and 40% contained detectable HIV-1 DNA, respectively. There was a significant decrease in the prevalence of HIV-1 DNA positive ARL and KS cases in the post-HAART era (after 1996; ARL=39%, KS=16.7) as compared to pre-HAART (ARL=54%, KS=45%). Our data suggest the overall amount of HIV DNA is less in tumor biopsies from the post-HAART era. A subset of ARL contained extremely high levels of HIV-1 DNA (~1 copy/cell). In addition, visceral KS had a higher prevalence of HIV-1 DNA (51.9%) as compared to skin KS (30.7%). HIV sequence evolution analysis of metastatic ARLs revealed that HIV was compartmentalized within sites of lymphoma and was distinct from HIV present in non-lymphoma sites.

Conclusions

The prevalence of HIV-1 DNA positive ARLs declined in the post-HAART era, but not to the same extent as KS, consistent with the incidence of both tumor types in the post-HAART era. Higher prevalence of HIV-1 DNA in visceral sites of KS and lymphoma specific-HIV sequences in sites of metastatic lymphoma suggests that HIV, especially HIV infected macrophages, may play a role in the pathogenesis of KS and ARL disease progression. Additionally, HIV-infected macrophages are a source of chronic inflammation that may further enhance tumorigenesis. Our data suggest a tumor specific form of HIV may be evolving within individuals who develop ARL.

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