

Poster presentation

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CD57+, a global marker of immunosenescence, is elevated in an atypical cohort of patients with Kaposi sarcoma and well-controlled HIV

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Background

Traditionally, KS has been associated with advanced age or with significant immunosuppression. We recently reported an atypical cohort of antiretroviral-treated HIV infected patients who developed cutaneous KS despite having undetectable plasma HIV RNA levels and high CD4+ T cell counts. The KS lesions seen in these patients were indolent and reminiscent of classical KS seen in the HIV-negative elderly. The mechanism of KS in the elderly remains undefined, but many have postulated a potential role of immunosenescence, which is generally defined as the gradual loss of immunologic function during advanced age. Given emerging evidence suggesting that prolonged periods of untreated HIV infection results in accelerated and perhaps irreversible "aging" of the immune system, we hypothesized that immunosenescence may account for the development of KS in otherwise young well-treated HIV infected individuals.

Methods

Peripheral blood was collected from two patient groups: (1) antiretroviral-treated HIV infected patients with undetectable viral loads who developed KS (n = 10) and (2) treated patients with undetectable viral loads who did not develop KS (n = 86). All KS cases had CD4 counts >300. Flow cytometry was performed on cryopreserved cells to determine the proportion of CD4+ and CD8+ T cells that

were activated or senescent. CD38 and HLADR were used to measure activation and CD57 was used as a measure of immunosenescence as this cell surface marker has been associated with shorter telomere length, a history of cell division, and decreased proliferative ability.

Results

Consistent with our initial hypothesis, in patients who had developed KS a higher percentage of CD4+ T cells expressed CD57 than in those individuals who did not develop KS (median of 10.8% vs. 6.4%, $P = 0.03$). Similarly, those with KS had higher percentage of CD8+T cells that expressed CD57 compared with those without KS (42% vs. 32%, $P = 0.02$). There was no difference in T cell activation markers between the KS and non-KS groups. The difference in CD57 expression between those with and without KS was not explained by age, gender or HCV co-infection.

Conclusion

In this study we find that a well recognized global immunosenescence marker (CD57) was elevated among patients with well-controlled HIV who developed KS as compared to comparable patients who do not develop KS. Collectively, these observations suggest that (1) HIV infection drives premature immunologic aging, (2) HAART does not fully reverse this process and (3) HIV associated

immunosenesence may result in increased risk of the type of KS commonly observed in certain elderly populations. This association of immunosenescence and chronic well-controlled HIV in the setting of an AIDS-defining malignancy carries implications for adequacy of current immunologic monitoring of HIV progression. It also raises questions with regard to regulation of chronic inflammation and replication in the long-term management of HIV infection, and for the potential of immune-related morbidity in the aging population of HIV-positive patients.

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