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Poster presentation

P10-11. NK cells do not accumulate at sites of HIV-replication but show increased activation

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Background

Mounting evidence suggests that NK cells play a critical role in the control of HIV infection. Early HIV replication is thought to occur preferentially in lymphoid organs, including lymph nodes. Thus we hypothesized that early control of HIV replication may be associated with the preferential recruitment of specific populations of NK cells to lymph nodes where they are involved in effective control of viral replication.

Methods

We therefore characterized the size and phenotype of NK cells in the lymph nodes and blood of 10 HIV infected individuals in the first year of HIV-1 infection compared to 4 healthy controls.

Results

The frequency and subset distribution of NK cells in lymph nodes of HIV-infected individuals was not altered compared to uninfected controls. However, we observed a significant decline in the proportion of 2B4 and CD161+ NK cells, and elevated levels of CD62L and TRAIL on NK cells of HIV infected individuals compared to healthy controls ($p < 0.05$, for all comparisons). Interestingly, KIR+ NK cells were absent from the lymph nodes of both HIV-infected and uninfected individuals. Peripheral KIR+ NK cells in HIV-infected individuals expressed reduced fractalkine receptor (CX3CR1) and CXCR1, potentially contributing to impaired recruitment of KIR+ NK cells to the lymphnodes, despite active viral replication.

Conclusion

These data demonstrate for the first time that NK cells do not accumulate in the lymph node during early HIV-1 infection, however that the NK cells in lymph nodes are more activated and may contribute to control of viral replication through non-cytolytic mechanisms such as TRAIL. Furthermore, the lack of recruitment of cytolytic NK cells expressing KIR during early HIV infection may allow the virus to replicate unabated by cytolytic innate immune pressure, thereby utilizing the lymph node as a protected niche in with to replicate free from immune pressure mediated by these innate immune effectors.