Poster presentation

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P16-22. In impact of nadir CD4 counts on skewed distributions of functional subsets in peripheral CD4+ T cells in patients chronically infected with HIV-1

K Sakai*1, H Gatanaga2, S Oka2 and M Takiguchi1

Address: ¹Viral Immunology, Center for AIDS Research, Kumamoto University, Kumamoto-shi, Japan and ²AIDS Clinical Center, International Medical Center of Japan, Shinjuku-Ku, Japan

* Corresponding author

from AIDS Vaccine 2009 Paris, France. 19–22 October 2009

Published: 22 October 2009 Retrovirology 2009, **6**(Suppl 3):P251 doi:10.1186/1742-4690-6-S3-P251

This abstract is available from: http://www.retrovirology.com/content/6/S3/P251

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Background

Infection with human immunodeficiency virus type 1 (HIV-1) induces dysfunction of CD4+T cells, and CD4+Tcell responses do not fully develop in many infected individuals. Previous studies suggested correlations between disease outcome and CD4+T-cell dysfunction caused by chronic immune activation and abnormal maturation. To understand the mechanism of virally induced T-cell dysfunction, we examined phenotypic changes occurred in the peripheral CD4+T cells of patients chronically infected with HIV-1.

Methods

We defined CD4+T-cell subsets based on the expression of surface molecules associated with specific functional subsets. We used four CD4-subset markers, CD45RA, CCR7, CD27, and CD28, to characterize naive, central memory, effector memory, and effector cells in 25 chronic patients. Among the patients, 23 individuals were on anti-retroviral therapy at the time of sample collection (mean CD4 count = 511). The frequency of each subset was determined by multi-color flow cytometry.

Results

Peripheral CD4⁺T cells from HIV+ patients had a skewed population distribution with a higher proportion of T_{CM} and decreased T_{EM} . Significant difference was also observed among patients. Particularly, patients with nadir CD4 count <100 cells per microliter had substantially

smaller naïve and increased effector subsets than patients who maintained at least 100 CD4⁺T cells per microliter, even after their CD4 counts returned to a normal or nearly normal range with years of anti-retroviral therapy.

Conclusion

HIV-1 infection disturbed the proper maintenance of the functional subsets in the peripheral CD4+ T-cell population in chronic patients. The recovery of the population distribution was highly dependent on the nadir CD4 counts. The skewed distribution of the functional subsets persisted for years in patients with low nadir CD4 counts, which could lead to the dysfunction of peripheral CD4+T cells observed in many HIV+ patients.