Background: Plasma cytokine and chemokine levels in HIV infected individuals are known to have direct association in disease progression. However, this association is not well elucidated between different groups of HIV-infected individuals. Immune control mechanisms at cellular level are often attributed to disease non-progression in long-term non-progressors (LTNPs). Hence, to investigate the possible association of plasma cytokine and chemokine levels with disease progression, in this preliminary study we have evaluated the relative proportion of cytokine and chemokine levels with disease progression, in this preliminary study we have evaluated the relative proportion of cytokine and chemokine levels in plasma among LTNPs and progressors. We also correlated the levels with the markers of disease progression such as CD4 T-cell count and plasma viral load (PVL).

Methods & Materials: Concentration of cytokines - IL-2, IFN-γ, TNF-α, IL-6 and IL-22 and chemokines - IP-10, MIP-1β, MCP-1 and RANTES in plasma were estimated using ELISA in LTNPs (n=15) and progressors (n=13). Statistical analyses were performed using Mann Whitney U test and Spearman rank correlation test. A p value of <0.05 was considered significant.

Results: The median age of LTNPs were 38 years with median CD4 T-cell count of 870 cells/μL and median PVL of 154 copies/mL, while the median age of Progressors were 30 years, with median CD4 of 384 cells/μL and median PVL of 1,39,308 copies/mL, Opportunistic infections such as tuberculosis (TB), and gastroenteral infections were reported in 9 of 13 progressors (69%). IFN-γ (p=0.0009) and IP-10 (p=0.018) levels were significantly elevated in progressors than in LTNPs, whereas MCP-1 (p<0.005) and RANTES (p=0.03) were significantly increased in LTNPs. No significant correlation was found between disease progression markers and the levels of cytokines and chemokines.

Conclusion: Unlike observed in cellular expression, plasma levels of CXCR3 ligand, IFN-γ were lower in LTNPs, which in turn might have influenced the lower IP-10 levels in LTNPs. Also, higher MCP-1 and RANTES levels in LTNPs might have resulted in higher monocyte infiltration and CCR5 receptor binding. Minimal events of opportunistic infections, lesser migration of TH1 and TH2 cells due to lower cytokine activity at peripheral circulation and higher inhibition of HIV binding to co-receptors due to elevated chemokine levels might explain the possible reasons behind disease non-progression in LTNPs.

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