



POSTER PRESENTATION

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Highly efficient neutralization of human immunodeficiency viruses by plasma from antiretroviral drug treated patients is mediated by IgG fractions

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Background

Little is known about the neutralizing activity in patients on antiretroviral therapy (ART), as most recent studies have focused on drug naïve individuals. ART may lead to a significant increase in B cell numbers and normalization of B cell subpopulations, providing a possible explanation for improved B cell responses after ART.

Methods

Thirty-four HIV-1 seropositive patients on ART (25 males and 9 females) within the age range of 20-55 years were recruited in this study. The patients had a median CD4 count and viral load of 283 cells and 178 RNA copies respectively, and were on treatment for a few days up to two years. Heat inactivated plasma samples were tested for neutralization against a panel of 14 subtype-A, B and C tier 1 and tier 2 viruses in TZM-bl assay.

Results

Of the 34 plasma samples, remarkably all the plasma samples were able to neutralize at least one virus while 32 (94%) samples were found to neutralize $\geq 50\%$ viruses tested. Clustering analysis revealed that AIIMS253 (a clade-C virus) was the most sensitive while RHPA4259.7 (a clade-B isolate) was most resistant to antibody neutralization. The Immunoglobulin-G fractions from two representative samples AIIMS221 and AIIMS265 were shown to mediate neutralization exclusively. The IgG fractions retained binding to subtype-A, B and C recombinant gp120 proteins. We did not find any association of mean reciprocal ID50 neutralization titers with the

plasma levels of ART drugs and clinical and immunological variables like CD4 count ($p=0.35$), viral load ($p=0.37$) and plasma total IgG ($p=0.46$). However we observed a positive association of neutralization with duration of ART ($p=0.02$) with a similar trend in two follow up patient samples.

Conclusion

Plasma antibodies from patients on ART display high neutralizing activity most likely due to an improved B cell function induced by ART despite low antigenic stimulation.

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