Background

Cardiovascular disease (CVD) is a leading cause of death in people with HIV. Chronic HIV-1 infection is characterized by systemic immune activation and inflammation and may contribute to increased cardiovascular disease (CVD) risk among HIV-infected individuals on stable antiretroviral therapy (ART). We assessed associations of monocyte subtypes, CD16 T cell activation and plasma cytokines, with change in carotid intima-media thickness (cIMT) over 2 years.

Methods

Longitudinal data from Hawaii Aging with HIV - Cardiovascular Disease (HAHV-CVD) Study, a study examining the role of oxidative stress and inflammation in HIV-associated CVD. We studied 50 subjects: 84% male, median age 49 (Q1, Q3; 46, 56) years, median CD4 count 461 (317, 578) cells/µL, with HIV RNA <50 copies/mL in 84%. Change in cIMT was correlated with log values of baseline absolute CD8+ T cell activation (% frequency of CD38+HLA-DR+ expression) at baseline. Plasma inflammatory biomarkers (sE-selectin, sVCAM-1, sICAM-1, MMP-9, tPA-γ, CRP, IL-6, IL-8, IL-10, TNF-α, MCP-1, VEGF, IFN-γ) were performed measured in plasma using a Luminescence multiplex platform. Results in this study were stratified by the percentage of non-classical monocytes (R2=0.2%). No correlation was noted in 84%. Change in cIMT correlated positively with log values of baseline absolute CD8+ T cell activation and cIMT change. Additionally, if study subjects were stratified by the percentage of non-classical monocytes into high and low with a median value (5.4%), then the baseline cIMT value itself tended to predict the change in cIMT at the high (R²=0.6%), but not low level of non-classical monocytes (R²=0.2%). No correlation was noted with CD16 T cell activation and change in cIMT.

Results

Conclusions

Non-classical monocytes play a role in the progression of CVD in HIV-infected individuals on suppressive ART, independent of traditional cardiovascular and HIV immune-inflammatory factors. Levels of non-classical monocytes level parallel increases in the pro-inflammatory cytokines MCP-1. Non-classical monocytes may serve as diagnostic index of CVD risk and a potential therapeutic target to ameliorate CVD risk in this vulnerable population.

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