HIV infection does not contribute to increased cardiovascular risk as assessed by Framingham risk score

Ramsay, I1; Pryce-Roberts, A1; Williams, S1; Bolton, E1; Hannig, E1; Perry, N1; Fisher, M2 and Rajkumar, C3

1Brighton and Sussex University Hospitals Trust, Brighton, UK. 2Brighton and Sussex University Hospitals Trust, Lawson Unit, Brighton, UK. 3Brighton and Sussex University Hospitals Trust, Department of Geriatric and Stroke Medicine, Brighton, UK.

HIV-1-infected patients are thought to be at higher risk of cardiovascular events. Measures of arterial stiffness are independently associated with cardiovascular risk [1]. The aim of our study was to determine if higher Framingham risk is associated with higher carotid femoral pulse wave velocity (cfPWV) in HIV-infected volunteers (HIV cohort) and to establish whether there is a difference in cfPWV between the HIV cohort and age- and gender-matched controls. We recruited 47 males (HIV cohort) on antiretroviral treatment, from a UK HIV clinic between October 2010 and March 2012 (31 low Framingham risk <10% and 16 high risk >20%). This group was matched with 46 healthy subjects from a contemporaneous study performed by our group. The inclusion criteria were: age 35–75 years with Framingham risk ≥20% or ≤10%, on antiretroviral treatment with undetectable viral load, no previous coronary heart disease, stroke or insulin therapy. Subjects underwent cfPWV measurement using Complior (Artech, France). Student’s t-test was used to evaluate differences between high- and low-risk groups and also between cases and controls. The mean age of the HIV cohort was 49.43 ± 9.35 years (mean ± SD) and in the control group 52.20 ± 8.80 years (p = 0.15). Mean duration of HIV infection was 13.83 ± 7.25 years, mean CD4 count was 728.81 ± 312.62 x 10^3/L and all viral loads were undetectable. In the HIV cohort, cfPWV was 8.39 ± 1.09 m/s in the low-risk group and 10.43 ± 2.93 m/s in the high-risk group (p = 0.02). Multivariate analysis with cfPWV as dependent variable, and age, systolic blood pressure, cholesterol, smoking history, duration of HIV infection and antiretroviral therapy, zenith viral loads and nadir CD4 counts as independent variables was performed in the high- and low-risk groups. This showed age alone to be a significant predictive factor (p = 0.002). With Framingham risk as dependent variable and using the above factors as independent variables, no HIV-related factors were significant predictors. The overall mean cfPWV for the HIV cohort (n = 47) was 9.09 ± 2.13 m/s compared to 11.95 ± 2.37 m/s in the control group (n = 46) (p < 0.01). HIV infection does not contribute to increased cardiovascular risk as assessed by Framingham risk score or carotid-femoral pulse wave velocity. This may be due to good control of traditional cardiovascular risk factors and a healthy lifestyle in this cohort.

Reference