

# 5th International Workshop on Adverse Drug Reactions and Lipodystrophy in HIV



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## CAROTID INTIMA-MEDIA THICKNESS IS MODERATELY INCREASED OVER TIME IN HIV-1-INFECTED PATIENTS

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**OBJECTIVE:** Dyslipidaemia and lipodystrophy syndrome, associated with other classical cardiovascular risk factors, expose HIV-infected patients to atherosclerosis cardiovascular disease. We aimed in a 12-month follow-up study to describe changes in carotid intima-media thickness (IMT), a surrogate marker of atherosclerosis, and its determinants.

**METHODS:** In a multicentre prospective cohort study of HIV-infected patients predominantly treated by highly active antiretroviral therapy (HAART), 346 patients were investigated with two IMT measurements by B-mode ultrasonography at M0 and M12.

**RESULTS:** We showed a significant but moderated increase of the mean common carotid artery (CCA)-IMT from 0.57 to 0.59 mm ( $P < 10^{-4}$ ) that remains in the normal range value observed in the general population. We observed a significant association between cross-sectional CCA-IMT measures at M0 and M12 and the conventional cardiovascular risk factors, such as older age ( $P < 10^{-4}$ ), male gender ( $P = 0.02$ ), tobacco consumption ( $P = 0.05$ ) and CD4 cell count ( $P = 0.01$ ). Only CD4 cell count at M0 was statistically associated with the variation of IMT (M12-M0) ( $P = 4.10^{-3}$ ). According to quartile of CD4 cell count distribution at M0, IMT progression was 1.98  $\mu\text{m}$  for 3–253

CD4 cells count/mm<sup>3</sup> at M0; 10.17  $\mu$ m for 253–402; 42.67  $\mu$ m for 402–590; and 27.73  $\mu$ m for 590–2270.

**DISCUSSION:** We hypothesize that chronic HIV infection and HAART use could promote atherosclerosis through several mechanisms; first, the higher prevalence of the atherogenic metabolic disorders and second, the immunological restoration and/or the stimulated CD4 cell increase able to produce CD40L, which is widely implicated in the progression of atherosclerosis. In this view, unstable atherosclerosis plaque should be investigated in order to better understand the pathophysiological mechanism of atherosclerosis and/or acute thrombotic events in HIV-infected patients.

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