Journal of the International AIDS Society

Poster presentation

Cardiovascular risk estimation in Spanish HIV-infected patients: a multicenter cohort study

E Ferrer[1], C Minguez[2], A Mariño[3], P Geijo[4], F Brun[5], J Sanz[6], M Velasco[7], C Cortés[8], A Castro[9], A Ortí[10], L Force[11], P Barrufet[11], C Villalonga[12] and D Podzamczer*[1]

Address: [1]Hospital Universitari de Bellvitge, Barcelona, Spain, [2]Hospital General de Castellon, Castellon, Spain, [3]Hospital Arquitecto Marcide, Ferrol, Spain, [4]Hospital Virgen de la Luz, Cuenca, Spain, [5]Hospital Jerez de la Frontera, Jerez de la Frontera, Spain, [6]Hospital de la Princesa, Madrid, Spain, [7]Hospital Fundación Alcorcón, Madrid, Spain, [8]Hospital de L'Hospitalet, Barcelona, Spain, [9]Hospital Juan Canalejo, La Coruña, Spain, [10]Hospital Verge de la Cinta, Tortosa, Spain, [11]Hospital de Mataró, Mataró, Spain and [12]Hospital Son Dureta, Palma de Mallorca, Spain

* Corresponding author

from Ninth International Congress on Drug Therapy in HIV Infection
Glasgow, UK. 9–13 November 2008

Published: 10 November 2008

Journal of the International AIDS Society 2008, **11**(Suppl 1):P92 doi:10.1186/1758-2652-11-S1-P92

This abstract is available from: http://www.jiasociety.org/content/11/S1/P92

Purpose of the study

To assess estimated cardiovascular risk in a population of adult HIV-infected patients.

Methods

This is an ongoing, prospective, longitudinal, multicenter cohort study of estimated cardiovascular risk (eCVR). After obtaining informed consent, demographic characteristics, metabolic parameters, and clinical status, including CVR factors, of patients (pts) enrolled from February to August 2006 at 12 Spanish sites were recorded in a central database using an Access data set. eCVR was evaluated by the Framingham equation. For analysis purposes pts were grouped into: 1) low eCVR (< 10% at 10 years); or 2) moderate/high eCVR (> 10%). Descriptive statistics were expressed as median (interquartile range) for quantitative variables and as percentages for qualitative variables. Logistic regression was used to identify the factors that might account for a higher eCVR. Baseline data are presented.

Summary of results

807 pts were included: 73% men, median age 41 (18–83) years, 42% drug users, 26% AIDS, CD4 465/uL, 51% undetectable viral load, 74% taking HAART (51% PI, 49% NNRTI regimens), 64% smokers, 10% hypertension, 5% diabetes, 3.5% coronary or cardiovascular disease (CVD). 81% pts had < 10 eCVR (Grp. 1) and 19% ≥10 eCVR (Grp. 2). Grp. 2 pts were more frequently men, older, non-drug users, smokers, diabetics, hypertensive, had CVD, lipid disturbances, greater waist circumference, greater BMI, undetectable viral load, and used more stavudine and indinavir. On univariate analysis, male sex (OR 8.51), coronary disease (5.77), CVD (3.87), diabetes (3.87), hypertension (3.66), smoking (1.96), age (1.16), lipid alterations (total cholesterol, HDL, LDL, Tryglicerides, no-HDL and TC/HDL, 1.02 to 1.52), waist circumference (1.04), and BMI (1.04) were associated with increased eCVR, while detectable viral load was associated with a low eCVR (0.47). First or current antiretroviral drugs were not associated with increased eCVR. On multivariate analysis, male sex (OR 19.96), age (25.08), smoking (24.12), hypertension (5.41), and total cholesterol (1.04) remained significantly associated with increased eCVR while detectable viral load (0.38) and HDL (0.94) remained significantly associated with a low eCVR.

Conclusion

Traditional cardiovascular risk factors are associated with increased eCVR in our HIV population, and seem to have a stronger role than HAART and HIV infection. Lower eCVR in pts with detectable viral loads is probably related

to younger age, lower waist circumference and systolic blood pressure, and a better lipid profile. Pts with higher eCVR may benefit from lipid-friendly ARV therapy, but interventions on life habits are crucial.