

Hepatitis Co-infection: Cardiovascular and Malignancy Association

Session Day and Time: Tuesday, 1-4 pm

Room: Hall B

1082

Hepatitis Virus Co-infections and Risk of Diabetes Mellitus and Myocardial Infarction in HIV-infected Persons: The D:A:D Study

Rainer Weber^{*1}, C Sabin², P Reiss³, S De Wit⁴, S Worm⁵, M Law⁶, F Dabis⁷, A d'Arminio Monforte⁸, E Fontas⁹, J Lundgren⁵, and The D:A:D Study Group

¹Univ Hosp Zurich, Switzerland; ²Royal Free and Univ Coll London, UK; ³ATHENA, Academic Med Ctr, Amsterdam, The Netherlands; ⁴Ctr Hosp Univ St Pierre, Brussels, Belgium; ⁵Copenhagen HIV Prgm, Univ of Copenhagen, Denmark; ⁶Australian HIV Observational Database, Natl Ctr in HIV Epidemiology and Clin Res, Sydney; ⁷Univ Victor Segalen, Bordeaux, France; ⁸ICONA, Hosp San Paolo, Univ of Milan, Italy; and ⁹Nice Cohort, Ctr Hosp Univ Nice, Hosp de l'Archet, France

Background: Studies among HIV⁻ individuals have reported a link between hepatitis C infection and the development of both diabetes mellitus (DM) and myocardial infarction (MI). However, the limited data available in HIV^{+ve} populations are conflicting.

Methods: Using prospective data from the D:A:D cohort, we considered whether hepatitis B virus (HBV) or C virus (HCV) co-infection were associated with new onset DM or MI. Individuals were classified as HCV seronegative, seropositive, or unknown and as HBV seronegative/vaccinated or having inactive, active (HBs, HBe, HBV DNA positive), or unknown HBV infection. Poisson regression assessed the influence of HCV or HBV infection on the development of DM or MI after adjustment for potential confounders (age, sex, risk group, ethnicity, previous AIDS, smoking, family history of cardiovascular disease [CVD], previous CVD, cohort, calendar year, body mass index, and exposure to ART); analyses of MI also controlled for DM development.

Results: Of 32,395 participants in D:A:D who were free of DM at study entry, 783 developed DM over 152,054 person-years (event rate: 5.15 /1000 person-years, 95% confidence interval, 4.79 to 5.51). Event rates in those who were HCV^{-ve} and HCV^{+ve} were 4.95 (4.50 to 5.40) and 5.55 (4.73 to 6.36), respectively, and for those who were HBV^{-ve} or had inactive or active infection were 5.28 (4.84 to 5.71), 4.83 (3.69 to 5.97), and 4.12 (2.81 to 5.43), respectively. After controlling for potential confounders, HCV seropositivity was associated with an increased risk of DM (relative rate compared to HCV^{-ve}: 1.32 (1.04 to 1.69), but no similar association was apparent for HBV infection whether inactive (0.85; 0.65 to 1.12) or active (0.74; 0.53 to 1.02). Further adjustment for changes in lipids had little impact on the results. Over 157,912 person-years, 517 patients developed MI (3.27; 2.99 to 3.56/1000 person-years). Event rates in those who were HCV^{-ve} and HCV^{+ve} were 3.32 (2.96 to 3.69) and 2.73 (2.17 to 3.29), respectively, and for those who were HBV^{-ve} or had inactive or active infection were 3.16 (2.83 to 3.49), 4.17 (3.13 to 5.21), and 2.84 (1.77 to 3.91), respectively. After adjustment, there was no relationship between HCV seropositivity (0.86; 0.62 to 1.19), inactive HBV (1.07; 0.79 to 1.43), or active HBV infection (0.78; 0.52 to 1.15), and the development of MI.

Conclusions: We found a significant association between HCV co-infection and the development of new onset DM. There was no association with HBV nor between either HCV or HBV co-infection and the development of MI.

