Abstract 56

Featuring HIV/HCV coinfected women in the Icona Cohort: epidemiological and clinical aspects according to gender.

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Background: HIV/HCV women represent a significant challenge in terms of management compared to HIV/HCV men. In HCV monoinfection, a possible higher risk of drug toxicity, higher impact of comorbidities and partially different predictors of fibrosis progression have been reported according to gender; data in HIV/HCV are lacking.
Objectives: To describe demographic, epidemiological and clinical characteristics of HIV/HCV women in the cohort. To identify predictors of HIV/HCV status, of liver damage, and HCV-RNA replication according to gender.
Methods: We included patients (pts) in the ICONA cohort with at least 1 HCVAb test available. Characteristics at time of the first HCVAb determination (baseline) were compared in positive (HCV+) and negative (HCV-) pts using X2/Wilcoxon tests, according to gender. Overall, and gender specific factors associated with HCV+ were identified by logistic regression. Further analyses, with gender as variable of interest, were also performed on HCV+ pts with end points: liver damage at baseline (FIB4>3.25) and evidence of HCV replication (HCVRNA+). Prevalence of different HCV genotypes in males and females was also assessed.
Results: 10318 pts were included; 29.2% tested HCV+. The prevalence of HCV among females and males was 29.4% and 30.0% respectively. Characteristics at first HCV+ according to HCV serostatus and gender are shown in table 1. Female gender did not result to be an independent higher risk of HCV+ (AOR 1.2, 95%CI 0.9-1.4, p=.07). Among females, Caucasian race (AOR 4.7, 95%CI 2.1-10.7, p<0.001), IDU (39.4, 95%CI 27.1-57.4, p<0.001) earlier HIV diagnosis 0.9, 95%CI 0.9-1.0, p<0.001, for 1 year more), calendar year of enrollment (0.9, 95%CI 0.8-0.9, p<0.001, for each year later) were predictive of HCV+; similar factors resulted predictive of HCV+ in males, except for Caucasian race. Among HCV+, % of females with fib4>3.25 at enrollment was significantly lower as compared to males (10.2% vs. 15.3%, p=.0002).
However after adjusting for potential confounders (g1/4; year of enrollment; year of HIV diagnosis; age, HbSAg, IDU, CD4 and HIV-RNA) the protective role of female gender on liver fibrosis was not confirmed (female AOR 0.8, 95%CI 0.4-1.3). Similarly, gender was not predictive of HCVRNA+ (female AOR 0.9, 95%CI 0.6-1.3). HCV genotype was available for 37.5% of pts; in 15.8% G1 was found, with no differences according to gender.
Conclusions: Similar risk factors accounted for HCV infection among males and females, mainly related to IDU in earlier years among Caucasians. Among females, mostly in a premenopausal age, a similar degree of fibrosis compared to their male counterpart was found. Thus in HIV/HCV coinfection, gender seems not to represent a protective factor for advanced liver disease and it could suggest that immunosuppression can equalize HCV-specific outcomes in women and men.