15th Conference on Retroviruses and Opportunistic Infection 2008

Factors associated with survival and first hepatic decompensation in a large prospective cohort of HIV-HCV co-infected patients with liver cirrhosis.

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Introduction: There are few data about factors associated to survival and hepatic decompensation in HIV/HCV coinfected patients with liver cirrhosis.

Methods: in 331 HIV-HCV coinfected patients with cirrhosis we compared the association of survival (defined as time to death, hepatocarcinoma or liver transplant) with: age, sex, time since cirrhosis/HIV diagnosis, alcohol use, CD4 (nadir, baseline and <100 at baseline), HIV viremia, suppressed HIV replication, history of anti-HCV treatment, HCV genotype, sustained viral response (SVR) to anti-HCV treatment, concomitant chronic hepatitis B, history of cirrhosis decompensation, Child-Pugh score (CPS) and HAART (at baseline, continuous/interrupted during follow-up). For the 251 patients without prior hepatic decompensation we analyzed variables associated with the occurrence of first decompensation. Univariate/Multivariate Cox proportional hazard models were used.

Results: Male 78%. Median age: 44. Median follow-up: 18 months. 87% on HAART at baseline (50% received un-interrupted HAART during follow-up). Median CD4 384. 74% with undetectable HIV-RNA. Median time since HIV/cirrhosis diagnosis: 16/3 years. 6% had concomitant chronic hepatitis B. 27% HCV genotype 2/3. 30% had history or excessive alcohol intake. 58% had received/were receiving anti-HCV treatment (27% with SVR). During follow-up 62 endpoints occurred. Factors significantly associated to decreased survival (univariate): male gender, alcohol abuse, CD4<100 (baseline), unsuppressed HIV replication (baseline), not having received anti-HCV treatment, no SVR to anti-HCV treatment, history of decompensation, CPS-B and C, not receiving HAART at baseline and non-continuous HAART during follow-up. In the multivariate analysis 5 independent factors were associated with decreased survival: CD4 nadir (p=0.014; HR 1,003); CPS-B (p=0.0001; HR 10.1); CPS-C (p=0.0001; HR 19); unsupressed HIV replication at baseline (p=0.045; HR 1.9) and not continuous HAART during follow-up (p=0.001; HR 11.3). For patients with compensated cirrhosis at baseline the only variable associated with development of decompensation was a CPS-B (HR 7.4, p = 0.0001, 95%CI 2.5-22.2).

Conclusions: CPS-B and C are significantly associated with decreased survival in HIV-HCV coinfected patients with cirrhosis. Maintaining HIV viral suppression and continuous HAART use are associated with prolonged survival. CPS-B is significantly associated with the short-term risk of first hepatic decompensation