**Hepatitis C Virus (HCV) Coinfection Does Not Influence the CD4 Cell Recovery in HIV-1 Infected Patients with Maximum Virologic Suppression within the EuroSIDA Cohort**

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**INTRODUCTION**

Conflicting data exist whether HCV coinfection affects the CD4 T-cell recovery in HIV patients starting antiretroviral treatment. Of the major cohort studies some have found thatcoinfected patients had a blunted CD4 cell count increase compared to HIV-monoinfected patients (1,2), while others found no effect of HCV on CD4 recovery (1,3). In addition, the influence of HCV genotype on the response to antiretroviral treatment is not well studied. Previous studies have been limited by using HCV viremia positivity as a criterion for HCV infection, whereby patients with spontaneous HCV-RNA clearance are grouped with those with chronic infection. Furthermore, HCV-RNA viral load (VL) suppression was defined as HCV-RNA <40 copies/mL (1,4) or <400 copies/mL (1,3) which means that low level viral replication-influencing the CD4 cell recovery could not be ruled out.

**OBJECTIVES**

To investigate the influence of chronic HCV coinfection and HCV genotype on the CD4 cell recovery in HIV’s infected patients with maximum virologic suppression (HIV viral load <50 copies/mL) within the EuroSIDA cohort.

**METHODS**

 Patients

5282 patients from the EuroSIDA observational cohort who fulfilled the following inclusion criteria

Inclusion criteria and definitions

All patients tested for anti-HCV antibodies
- If HCV-RNA positive, a quantitative HCV-RNA (limit of detection 615 IU/ml) should be available
- If HCV-RNA positive, patients were required to be HCV genotyped
- At least two consecutive HIV viral loads <50 copies/mL after starting combination antiretroviral therapy (cART)

cART was defined as a minimum of three drugs, of which at least two were nucleos(t)ides. For each pair of HIV VL <50 copies/mL, the following criteria were required (figure 1):

1) A CD4 count measured within 45 days of each HIV VL measurement
2) The CD4 count to be different for each of the viral loads in the viral load pair (VLP)
3) No HIV treatment change between HIV VL measurements in a VLP

Statistical methods

Change in CD4, occurring between each pair of consecutive VL <50 copies/mL was calculated and standardized for the time between viral load measurements to give the rate of change in CD4 cells/µL per year. Each patient could contribute data for more than one viral load pair (VLP).

Generalised linear models, using a normal distribution and an identity link function, adjusted for repeated measures, were used to describe the rate in CD4 count changes (5).

Three comparisons were performed:

1. HCV-seropositive vs. HCV-monoinfected patients
2. Comparison between genotypes 1-4, in HCV-RNA positive patients
3. Among HCV-seropositive patients, comparison of those viremic vs. aviremic (HCV-RNA <659 IU/mL).

Baseline was arbitrarily defined as the latest of the first two consecutive VL <50 mL after starting cART and the date of first lab for anti-HCV antibodies.

**RESULTS**

Out of 14,182 patients in the EuroSIDA cohort 5,626 were included representing 54,718 pairs of HIV VL measurements with VL <50 copies/mL, and 10,422 person years of follow up (figure 2).

Table 1 describes the baseline characteristics of the patients. 809 (21%) patients were HCV-seropositive and among them 621 (77%) had detectable HCV-RNA.

Figure 3 shows the crude, unadjusted, annual change in CD4 count with maximum virologic suppression. For HCV-seropositive and seronegative patients it was 4.21 cells/µL (95% confidence interval [CI] 3.93–4.49) and 4.70 cells/µL (95% CI 4.50–4.90), respectively (p=0.86). Similarly, no significant difference in annual CD4 change was found when comparing between HCV genotypes (p=0.51) or HCV viremic vs. nonviremic in HCV-seropositive patients (p=0.80).

Figure 4 shows the crude, unadjusted, annual change in CD4 count with maximum virologic suppression. For HCV-seropositive and seronegative patients it was 4.21 cells/µL (95% confidence interval [CI] 3.93–4.49) and 4.70 cells/µL (95% CI 4.50–4.90), respectively (p=0.86). Similarly, no significant difference in annual CD4 change was found when comparing between HCV genotypes (p=0.51) or HCV viremic vs. nonviremic (p=0.80) (figure 4).

Adjusting additionally for HIV treatment and adherence, time to initial virologic suppression <50 copies/mL, we found no difference in annual CD4 change when comparing according to HCV serostatus (p=0.48), between genotypes (p=0.51) or when comparing HCV viremic vs. nonviremic (p=0.29).

**CONCLUSIONS**

In this prospective EuroSIDA cohort study we have shown compelling evidence that HCV coinfection does not impair the CD4 response to cART, even if the cART-modification will lower the risk of hepatotoxicity induced by antiretroviral drugs and progression of liver disease.

References:


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