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Objectives: Recently, HCV RNA viral load was described to have a role in fields other than antiviral treatment and associated with liver fibrosis and the development of chronic kidney disease. We performed a longitudinal comparative analysis of the 3 group of HIV-HCV patients identified by low HCV viral load (<600000 IU/ml).

Methods: Adult patients with chronic HIV and HCV infections were retrospectively selected if HCV therapy naïve or previous anti-HCV treatment failed, with regular viro-immunological follow-up of HIV infection. HIV viremia was classified as undetectable if no plasma value exceeded 50 copies/ml: only a single viral blip for year was tolerated. Starting from 2009 (time T1), patients were followed during 2 intervals of time: from 2009 to 2011 (Initial Follow-Up, IFU) and from 2011 to 2012 (Final Follow-Up, FFU). A comparative analysis of the 3 groups of patients was performed and they were classified as subjects with low level viremia at T1 and T2 (group 2Low), subjects with low level viremia only at T1 or only at T2 (group 1Low), subjects who never experienced low level viremia (group 0Low). Statistical analysis: Fisher's exact test, independent samples t-test and t test for paired data were employed where appropriate.

Results: A total of 82 patients were eligible for the study. Main characteristics of the three groups of patients are summarized in Table 1. A protease inhibitor based regimen was ongoing in 50 patients (71.4% of the treated subjects) in 2012. No difference reported for age, gender, advanced liver fibrosis, nonresponse to antiviral therapy, HIV virological control, percentage of subjects on ART. Genotype 1 was prevalent in 0Low group and 1Low group (p=0.01 respect to 2Low). CD4 value at T2 was higher in 0Low patients respect to 2L subjects (marginally statistically, p=0.06) and to 1Low individuals (p=0.02) and, moreover, the gain in CD4 positive cells/mmc was significant only in 0Low group (from 624 to 733 cells/mmc p=0.001).

Table 1 Demographic, clinical and virological features of the 3 groups of HIV-HCV co-infected patients identified on the basis of the detection of a low HCV viral load at T1 and T2 (Group 2L), either at T1 or at T2 (Group 1L), neither at T1 and at T2 (Group 0L).

ART: Anti Retroviral Therapy, Clean IFU: HIV viremia undetectable during initial follow-up (0-24 months from T1), regardless of being on HAART; Clean FFU: HIV viremia undetectable during final follow-up (24-36 months from T1), regardless of being on HAART

	Group 2L (11 pz, 13.4%)	Group 1L (19 pz, 23.2 %)	Groups 0L (52 pts, 63.4%)
Age, mean and SD	50 (±7)	48 (±7)	48(±6)
Male, n (%)	9 (81.8)	15 (78.9)	38 (73.1)
Genotype 1, n (%)	3 (27.3)	7 (36.8)	38 (73.1)
Previous HCV therapy, n (%)	1 (9.1)	4 (21)	12 (23.1)
Advanced liver fibrosis, n (%)	3 (27.3)	4 (21)	12 (23.1)
CD4 cell count at T1 (cells/mmc), mean and SD	465 ±274	506±267	624±279
CD4 cell count at T2 (cells/mmc) mean and SD,	502 ±417	526 ±265	733 ±346
HCV RNA at T1 (IU/ml), mean and SD	180697 (±153204)	1284363 (1708144)	5681360 (5928957)
HCV RNA at T2 (IU/ml) mean and SD,	181376 (±170577)	1018457 (1118477)	4454513 (3506212)
Ongoing HAART at T2, n (%)	8 (72.7)	17 (89.5)	48 (92.3)
Clean IFU, n (%)	4 (36.4)	9 (47.4)	26 (50)
Clean FFU, n (%)	5 (45.5)	12 (63.1)	40 (76.9)

Conclusion: Only a minority of HIV-HCV patients had a confirmed low HCV RNA viral load and they did not differ from other groups. The gain in CD4 positive count from T1 to T2 was significant only in the 0Low group: besides the lower virological control of HIV viremia in 2Low group and 1Low group, the recently described correlation between an increase of CD4 cell apoptosis and reduction of HCV RNA viral load could play a role