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Background: Liver disease is a frequent cause of complications amongst HIV-infected individuals. The availability of non-invasive tools to determine liver fibrosis (e.g. Fibroscan™, FS) allows to prospectively follow large number of patients.

Methods: A prospective cohort of HIV-infected patients examined by FS was established in 2004. In subjects with 2 FS examinations separated by, at least, 18 months, factors related with liver fibrosis regression (LFR) were analyzed in 2008. LFR was defined as a decrease of $\geq 30\%$ in liver stiffness in patients with significant fibrosis (METAVIR F3-F4, >9.2 KPa) at 1st FS between 1st and 2nd FS. Metabolic and HIV-related parameters were prospectively recorded at the time of 1st FS and every 3 months until 2nd FS. The presence and treatment of hepatitis B and C viruses, antiretroviral treatment characteristics and alcohol abuse were assessed as well.

Results: A total of 632 HIV-infected patients (median age 45 years, 78% males, 63% IDUs, median BMI 23 kg/m², median CD4 count 494 cells/ μ L, HCV-RNA (+)72%, HBsAg (+)10%, alcohol abuse 9%) had two paired examinations separated by a median of 27 (19-33) months. At 1st FS Metavir F3-F4 was detected in 193 patients (30%), and at the 2nd FS 65 patients (10%) had criteria for LFR. As compared with patients without LFR, the LFR group had a lower percentage of males (67.7 vs 83.6%, $p=0.01$) and a higher proportion of patients treated for hepatitis C (81.3 vs 44.2%, $p<0.001$). Patients with LFR had a greater decrease in glycaemia between the 2 FS (-3.2 vs 1.9 mg/dL, $p=0.04$) and lower HIV-RNA levels at FS2 (1.8 vs 2.1 log₁₀ copies/mL). Factors related with LFR (OR, 95% CI) in multivariate analysis were hepatitis C treatment (3.8 [1.2-12.1], $p=0.02$) and better glycemic control (1.05 [1.03-1.09], $p=0.03$).

Conclusions: Treatment of hepatitis C and better glycemic control are associated with an improvement in liver fibrosis in HIV-infected patients. No differences were found in terms of exposure to antiretroviral agents.

Table 1. Study population baseline characteristics

	Liver fibrosis regression (n=65; 33.6%)	No liver fibrosis regression (n=128; 66.4%)	p-value
Age	47.2 \pm 5.9	45.8 \pm 5	0.08
Male sex (%)	67.7	83.6	0.01
Parenteral transmission (%)	69.4	84.3	0.02
Alcohol abuse (%)	2.6	8.9	0.27
HIV-RNA (log ₁₀ copies/mL)	4.18 \pm 4.86	3.8 \pm 4.44	0.34
CD4 cell count (cells/ μ L)	500 \pm 304	491 \pm 266	0.85
Time between FS (months)	28.1 \pm 8.2	27.5 \pm 8.6	0.71
HCV infection (%)	80.3	91.5	0.03
HCV treatment (%)	81.3	44.2	<0.001
HCV sustained viral response (%)	60	25.6	0.001
HBV infection (%)	8.9	13.5	0.4
Glycose (mg/dL)	101.8 \pm 13.7	108.1 \pm 30.1	0.11
Triglycerides (mg/dL)	148.2 \pm 141.6	150 \pm 124	0.93
LDL-cholesterol (mg/dL)	106.2 \pm 31.9	93.2 \pm 29	0.007
Hepatic steatosis on abdominal ultrasound (%)	9.2	6.3	0.45
Antiretroviral-naïve	4.6	13.3	0.06

Table 2. HIV-related parameters

	Liver fibrosis regression (n=65; 33.6%)	No liver fibrosis regression (n=128; 66.4%)	p-value
HIV-RNA at 2 nd FS (log ₁₀ copies/mL)	1.8 \pm 0.4	2.1 \pm 1	0.001
CD4 cell count (cells/ μ L)	494.3 \pm 276	466.4 \pm 282	0.5
NNRTI-only base (%)	4.6	12.6	0.12
PI-only base (%)	10.8	14.1	0.65

Table 3. Metabolic parameters

	Liver fibrosis regression (n=65; 33.6%)	No liver fibrosis regression (n=128; 66.4%)	p-value
LDL-cholesterol variation (mg/dL)	6.7 \pm 31.5	2.5 \pm 27	0.36
Triglycerid variation (mg/dL)	-31.57 \pm 135.48	7.5 \pm 146	0.08
Glycose variation (mg/dL)	-3.25 \pm 10.8	1.9 \pm 23.6	0.04
Median BMI between FS (kg/m ²)	23.5 \pm 3.5	23.5 \pm 3.9	0.98

Table 4. Multivariate analysis

	OR (95% CI)	p-value
Treatment for HCV	3.8 (1.2-12.1)	0.02
Glycose variation	0.95 (0.91-0.99)	0.03
Age	0.98 (0.87-1.11)	0.76
Male sex	0.56 (0.17-1.87)	0.35
Alcohol abuse	1.25 (0.98-16.17)	0.86
log ₁₀ RNA-HIV at 2 nd FS	0.27 (0.05-1.56)	0.12

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