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Pegylated interferon α2a plus ribavirin versus pegylated interferon α2b plus ribavirin for the treatment of chronic hepatitis C in HIV-infected patients

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***Objectives*: The two currently available types of pegylated interferon (peg-IFN) used to treat hepatitis C have different pharmacokinetic properties. It is unclear how these differences affect response to therapy. We compared the effectiveness and safety of peg-IFN-α2a and peg-IFN-α2b, both with ribavirin, against chronic hepatitis C virus (HCV) infection in HIV-infected patients.**

***Methods*: From the GESIDA HIV/HCV cohort, we analysed patients treated with peg-IFN-α2a ($n = 315$) or peg-IFN-α2b ($n = 242$). The primary endpoint was a sustained virological response (SVR).**

***Results*: Both groups were well matched in baseline characteristics except for a higher frequency of injection drug users in the peg-IFN-α2b group than in the peg-IFN-α2a group (85% versus 76%; $P = 0.01$) and a higher frequency of bridging fibrosis and cirrhosis (F3–F4) in the peg-IFN-α2b group than in the peg-IFN-α2a group (42% versus 33%; $P = 0.04$). End-of-treatment response was significantly lower among patients treated with peg-IFN-α2b [40% versus 52%; odds ratio (OR), 1.63; 95% confidence interval (95% CI), 1.16–2.29; $P < 0.01$]. However, no significant differences were found in SVR between patients treated with peg-IFN-α2b and those treated with peg-IFN-α2a (31% versus 33%; OR, 1.09; 95% CI, 0.75–1.59; $P = 0.655$). Therapy was interrupted due to adverse events in 33 (14%) patients treated with peg-IFN-α2b and 47 (15%) patients treated with peg-IFN-α2a.**

***Conclusions*: No differences in effectiveness and safety were found between peg-IFN-α2b and peg-IFN-α2a for the treatment of chronic HCV infection in HIV-infected patients.**

Keywords: comparative study, IFN, HCV, effectiveness, safety, infections

Introduction

Hepatitis C virus (HCV) infection is one of the leading problems in HIV-infected patients and affects approximately one-third of this population.[1] HIV infection modifies the natural history of chronic hepatitis C with faster progression of fibrosis and a higher risk of cirrhosis and end-stage liver disease in HIV/HCV co-infected patients than in HCV mono-infected patients.[2–6]

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JAC antiviral

Since the introduction of potent antiretroviral therapy (ART), end-stage liver disease has become a frequent cause of hospital admission and death in populations co-infected with HIV and HCV.[7,8] For this reason, all HIV-infected individuals should be screened for HCV infection; individuals with positive HCV-RNA should be considered as candidates for anti-HCV treatment, providing HIV infection is well controlled and there are no contraindications to therapy with interferon (IFN) or ribavirin. Several clinical trials have proved that the most effective therapy for chronic HCV infection in HIV-infected patients is pegylated IFN (peg-IFN) plus ribavirin.[9–12]

Currently, there are two approved types of peg-IFN: peg-IFN-α2a with a molecular mass of 40 kDa and peg-IFN-α2b with a molecular mass of 12 kDa. In comparison with peg-IFN-α2a, peg-IFN-α2b has a larger volume of distribution and more effective renal clearance. Peg-IFN-α2a is administered as a flat dose, whereas dosing of peg-IFN-α2b is based on body weight.

Until recently, it was unclear how these differences affected response to therapy. In the last year, three clinical trials comparing the two approved types of peg-IFN have been reported.[13–15] In the largest of these trials, the 'Individualized Dosing Efficacy versus Flat Dosing to Assess Optimal Pegylated Interferon Therapy' (IDEAL) study,[14] similar sustained virological response (SVR) rates were found among treatment-naive, genotype 1 HCV mono-infected patients treated with ribavirin plus peg-IFN-α2a or peg-IFN-α2b.

We compared the effectiveness and safety of peg-IFN-α2a and peg-IFN-α2b, both in combination with ribavirin, against chronic HCV infection in HIV-infected patients.

Methods

Design and patient selection

The patients from this observational ambispective study were selected from the '*Grupo de Estudio del SIDA*' (GESIDA) of the '*Sociedad Española de Enfermedades Infecciosas y Microbiología Clínica*' cohort of patients treated with IFN and ribavirin (GESIDA HIV/HCV cohort) that was established in 2003 in order to follow HIV/HCV co-infected patients who started IFN and ribavirin therapy after January 2000 at 11 institutions in Spain. The primary objective of the GESIDA HIV/HCV cohort was to determine the effect of achieving an SVR after therapy with IFN and ribavirin on the long-term clinical outcomes of co-infected patients (including liver-related complications) and liver-related mortality. The study cohort received the approval of all of the Ethics Committees of the participating centres for analysis with a view to scientific publication based on anonymized routine clinical data without requiring written informed consent. All the information was entered directly into a common database at each institution by means of an *ad hoc* online application that satisfied local requirements of data confidentiality. This database included all demographic, clinical, virological (HIV and HCV) and laboratory data. Completion of treatment was followed by an active follow-up every 6 months that analysed clinical and laboratory parameters including survival, presence of any liver decompensation, presence of HIV-related diseases, ART, CD4 cell count, HIV viral load, HCV-RNA, liver biopsy and anti-HCV therapy. Anti-HCV therapy in Spain is provided by hospital pharmacies and is covered by the National Health System. The decision to administer anti-HCV therapy to co-infected patients was taken by infectious diseases physicians at each institution according to national and international guidelines. The eligibility criteria for anti-HCV therapy included absence of prior hepatic decompensation, CD4+ T cell count >200 cells/mm^3, stable ART or no need for ART, absence of active opportunistic infections and no active drug addiction. Patients were counselled against the use of alcohol. Anti-HCV therapy was stopped in all patients with detectable HCV-RNA at week 24 of treatment. Since 2002, some institutions have been applying the so-called '2-log stopping rule', i.e. discontinuation of therapy in patients with detectable HCV-RNA at week 12 of treatment with a reduction of <2 log IU/mL in HCV-RNA.

The objective of this substudy of the GESIDA HIV/HCV cohort was to compare the effectiveness and safety of peg-IFN-α2a and peg-IFN-α2b, both in combination with ribavirin, against chronic HCV infection in HIV-infected patients. The inclusion criteria for the study were initiation of peg-IFN and ribavirin therapy between January 2000 and December 2005 and no prior HCV therapy. For each patient, we extracted the following data from the central database: age, sex, height and weight at the initiation of therapy with peg-IFN plus ribavirin, HIV transmission category, prior AIDS-defining conditions, baseline and nadir CD4 cell counts and baseline HIV viral load. We also recorded information about highly active ART (HAART)—including type, date of initiation and whether it was maintained or changed during therapy. Information related to HCV infection included genotype, HCV-RNA levels and estimated year of HCV infection, which was estimated for injection drug users by assuming that it started the first year that needles were unsafely shared. Duration of HCV infection was considered to be unknown for subjects infected through sexual contact. We also recorded the results of liver biopsies; fibrosis was scored following the criteria established by the METAVIR Cooperative Study Group[16] as follows: F0, no fibrosis; F1, portal fibrosis; F2, periportal fibrosis or rare portal–portal septa; F3, fibrous septa with architectural distortion and no obvious cirrhosis (bridging fibrosis); and F4, definite cirrhosis. Patients were also asked about their alcohol intake. We considered the consumption of at least 50 g of alcohol per day for at least 12 months as a high intake.

Assessment

For each patient, we assessed the end-of-treatment response (ETR), defined as an undetectable serum HCV-RNA level at the end of therapy. The SVR was defined as an undetectable serum HCV-RNA level 24 weeks after discontinuation of therapy. Patients not fulfilling SVR criteria, including those who relapsed after achieving an ETR, were classified as non-SVR. Safety was assessed by laboratory tests and evaluation of adverse events during therapy.

Statistics

Differences between groups were analysed using the χ^2 test, Fisher's exact test, Student's *t*-test or Mann–Whitney *U*-test, as appropriate. Analyses were carried out on an intention-to-treat basis. Logistic regression models were used to explore baseline factors predicting an SVR. In the model, we included the baseline factors that were associated with SVR by univariate regression analysis as well as the type of peg-IFN used, and two variables of particular interest in this population, namely, the presence of F3–F4 in liver biopsy and an alcohol intake $\geq$50 g/day. The Statistical Package for the Social Sciences (SPSS) was used to analyse the study data (version 15.0; SPSS Inc., Chicago, IL, USA).

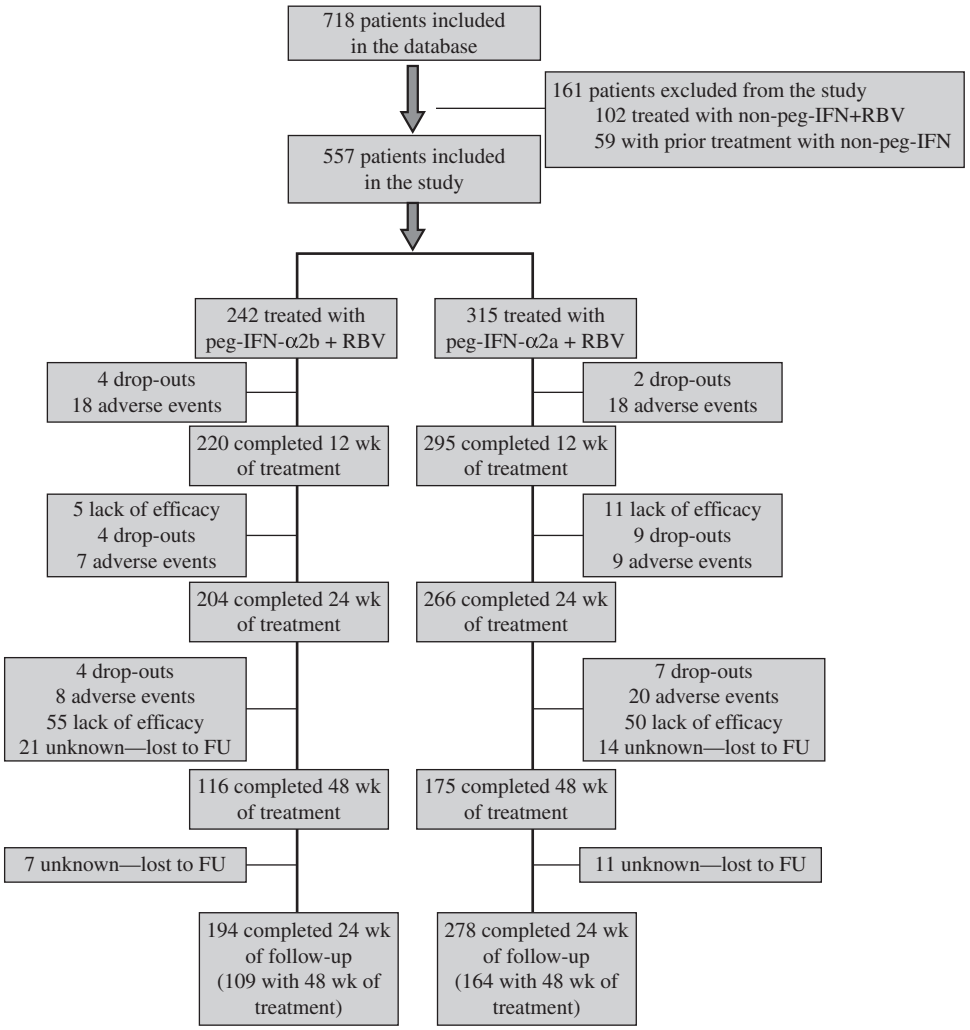

Figure 1. Flow chart reporting the reasons for non-inclusion and the outcome of the included patients. RBV, ribavirin; wk, weeks; FU, follow-up.

Results

Patient characteristics

Between January 2000 and December 2005, 718 patients were included in the GESIDA HIV/HCV cohort database. For the purpose of this study, we analysed 557 IFN-naive patients who were treated with peg-IFN-α2a plus ribavirin ($n=315$) or peg-IFN-α2b plus ribavirin ($n=242$). The remaining 161 patients were not included in the study because they were treated with conventional IFN and ribavirin (102 patients) or because they had previously received IFN without ribavirin (59 patients) (Figure 1). The baseline characteristics of the patients are shown in Table 1. Patient characteristics were similar in both groups except for a higher frequency of injection drug users in the peg-IFN-α2b group (85% versus 76%; $P=0.01$) and a higher frequency of bridging fibrosis and cirrhosis (F3–F4) in the peg-IFN-α2b group (42% versus 33%; $P=0.04$).

Treatment details

Patients in the peg-IFN-α2b group received subcutaneous injections of 1.5 μg/kg peg-IFN-α2b (Peg Intron®, Schering-Plough, Alcobendas, Spain) once weekly and patients in the peg-IFN-α2a group received 180 μg of peg-IFN-α2a (Pegasys®, Roche, Madrid, Spain) once weekly. All patients also received oral ribavirin twice a day. The median dose [and interquartile range (IQR)] of ribavirin was 13.3 mg/kg (12.3; 14.7) in the peg-IFN-α2b group and 14.0 mg/kg (11.8; 15.7) in the peg-IFN-α2a group ($P=0.09$). The median (IQR) duration of anti-HCV therapy was 8 months (6–11) in the peg-IFN-α2b group and 11 months (6–11) in the peg-IFN-α2a group ($P=0.130$).

Response to treatment

The sample size of our study had an 80% power to detect differences in SVR between groups of ≥11% with an alpha of 5%. We found that ETR was significantly lower among patients treated with peg-IFN-α2b [40% versus 52%; odds ratio (OR), 1.63; 95% confidence interval (95% CI), 1.16–2.29; $P<0.01$]. However, no significant differences were found in SVR between patients treated with peg-IFN-α2b and those treated with peg-IFN-α2a (31% versus 33%; OR, 1.09; 95% CI, 0.75–1.59; $P=0.655$). The relapse rate was significantly lower among

Table 1. Baseline characteristics of the patients

Characteristics	Peg-IFN-α2b plus ribavirin ($n=242$)	Peg-IFN-α2a plus ribavirin ($n=315$)	P value
Sex—*n* (%)[a]			0.71
male	181 (75)	229 (73)	0.78
female	61 (25)	83 (26)	0.78
Age—years, median (quartiles)	39 (35.9; 42.7)	40 (36.7; 43.1)	0.13
Weight—kg, median (quartiles)	67 (60; 75)	68 (59; 75)	0.37
Mode of infection—*n* (%)			0.05
injection drug use	206 (85)	239 (76)	0.01
sexual exposure	24 (10)	36 (11)	0.66
transfusion	6 (2)	21 (7)	0.04
unknown or other	6 (2)	19 (6)	0.68
CDC disease state—*n* (%)[b]			0.97
A	118 (49)	158 (50)	0.22
B	67 (28)	87 (28)	0.37
C	52 (21)	66 (21)	0.96
CD4+ cells baseline—cells/mm^3, median (quartiles)	492 (363; 740)	563 (411; 749)	0.91
CD4+ cells nadir—cells/mm^3, median (quartiles)	208 (110; 331)	204 (100; 324)	0.22
HIV-RNA <50 copies/mL—*n* (%)	135 (56)	184 (58)	0.59
Duration of HCV infection—years, median (quartiles)	17 (12; 21)	18 (13; 22)	0.27
Serum ALT—IU/dL, median (quartiles)	98 (62; 151)	93 (63; 138)	0.51
HCV genotype—*n* (%)			0.32
1	127 (52)	156 (50)	0.40
2	9 (4)	8 (3)	0.55
3	76 (31)	99 (31)	0.96
4	19 (8)	37 (12)	0.19
unknown	11 (5)	15 (5)	0.28
HCV-RNA ≥500000 IU/mL—*n* (%)	151/222 (68)	188/273 (69)	0.92
Liver biopsy—*n* (%)	204 (84)	253 (80)	0.27
Fibrosis F3–F4—*n* (%)	86 (42)	83 (33)	0.04
HBsAg-positive—*n* (%)	6 (2)	7 (2)	0.86
High alcohol intake[c]—*n* (%)	10 (4)	9 (3)	0.90
Methadone use—*n* (%)	24 (10)	43 (14)	0.14
Antiretroviral therapy—*n* (%)			
none	43 (18)	52 (17)	0.78
any	199 (82)	263 (83)	0.78
3 NRTI	32 (13)	34 (11)	0.46
2 NRTI+PI	45 (19)	98 (31)	0.01
2 NRTI+NNRTI	101 (42)	110 (35)	0.12
2 NRTI+NNRTI+PI	8 (3)	20 (6)	0.15
other/unknown	13 (5)	1 (<1)	<0.01

ALT, alanine aminotransferase; CDC, Centers for Disease Control and Prevention; HBsAg, hepatitis B surface antigen; HIV, human immunodeficiency virus; HCV, hepatitis C virus; NNRTI, non-nucleoside reverse transcriptase inhibitor; NRTI, nucleoside reverse transcriptase inhibitor; PI, protease inhibitor.
[a]Sex unknown for three patients in the peg-IFN-α2a group.
[b]CDC disease state unknown for five patients in the peg-IFN-α2b group and four patients in the peg-IFN-α2a group.
[c]Consumption of at least 50 g of alcohol per day for at least 12 months.

patients treated with peg-IFN-α2b than among those treated with peg-IFN-α2a (21% versus 37%; $P=0.011$) (Table 2).

No significant differences were found in SVR between patients treated with peg-IFN-α2b and those treated with

Table 2. Virological response at the end of treatment and follow-up by intention-to-treat analysis

	Peg-IFN-α2b plus ribavirin	Peg-IFN-α2a plus ribavirin	*P* value
Overall			
ETR	96/242 (40%)	163/315 (52%)	0.006
SVR	76/242 (31%)	103/315 (33%)	0.816
relapse	21%	37%	0.011
SVR by genotype and HCV-RNA			
genotypes 1–4	21/146 (14%)	37/193 (19%)	0.311
<500 000 IU/mL	9/37 (24%)	14/51 (27%)	0.933
≥500 000 IU/mL	10/96 (10%)	20/125 (16%)	0.316
genotypes 2–3	39/85 (46%)	48/107 (45%)	0.996
<500 000 IU/mL	14/32 (44%)	16/33 (48%)	0.893
≥500 000 IU/mL	23/50 (46%)	25/60 (42%)	0.792

ETR, end-of-treatment response; HCV, hepatitis C virus; SVR, sustained virological response.

peg-IFN-α2a when the patients were grouped according to HCV genotype. Patients infected with HCV genotypes 1 and 4 had lower rates of SVR than those infected with HCV genotypes 2 and 3 (Table 2). Among the patients infected with HCV genotype 1 or 4, those with a high pre-treatment HCV-RNA level (≥500 000 IU/mL) had a trend towards lower rates of SVR than those with lower pre-treatment HCV-RNA levels. The pre-treatment HCV-RNA level did not affect the rate of SVR among patients infected with genotype 2 or 3 (Table 2).

Factors associated with SVR

The baseline factors that were associated with SVR by univariate regression analysis were CDC clinical category, nadir CD4+ cell count, HCV genotype and HCV-RNA level. The final model identified two variables that were independently associated with increased odds of an SVR: HCV genotypes 2–3 (OR, 3.77; 95% CI, 2.23–6.36; $P<0.001$) and a CDC disease category other than C (OR, 2.45; 95% CI, 1.16–5.21; $P=0.019$) (Table 3). Receiving HAART was not a predictive factor of

Table 3. Factors associated with an SVR by multiple logistic regression analysis

	Univariate analysis		Multivariate analysis	
Variable	OR (95% CI)	*P* value	OR (95% CI)	*P* value
Peg-IFN-α2a plus ribavirin	1.09 (0.75–1.59)	0.655	1.35 (0.81–2.26)	0.250
CDC disease state (A/B versus C)	2.36 (1.39–4.01)	0.002	2.45 (1.16–5.21)	0.019
CD4+ cells nadir	1 (1–1)	0.049	1 (1–1)	0.099
Intake >50 g alcohol daily	1.56 (0.51–4.79)	0.437	1.87 (0.39–8.96)	0.432
Liver fibrosis F3–F4	1.49 (0.96–2.32)	0.075	1.19 (0.63–2.22)	0.595
HCV genotypes 2–3	4.01 (2.70–5.99)	<0.001	3.77 (2.23–6.36)	<0.001
HCV-RNA ≥500 000 IU/mL	1.76 (1.17–2.66)	0.007	1.27 (0.74–2.17)	0.390

CDC, Centers for Disease Control and Prevention; HCV, hepatitis C virus.

Table 4. Reason for interruption of therapy with peg-IFN plus ribavirin

	n (%)	
Reason	peg-IFN-α2b plus ribavirin (*n*=242)	peg-IFN-α2a plus ribavirin (*n*=315)
Treatment completion	116 (48)	175 (56)
Lack of efficacy	60 (25)	61 (19)
Withdrawn due to adverse events	33 (14)	47 (15)
Unknown—lost to follow-up	21 (9)	14 (4)
Drop-out	12 (5)	18 (6)

SVR in our study. In fact, the OR (95% CI) of SVR for patients with HAART compared with those without HAART adjusted by genotype and CDC category was 1.252 (0.792–1.980).

Safety

Reasons for interruption of therapy were not different between the groups (Table 4). Similarly, no significant differences were found between the peg-IFN-α2b and peg-IFN-α2a groups in terms of the frequency of severe adverse events (22% versus 17%, $P=0.16$), or in the reduction in peg-IFN dosage (14% versus 15%, $P=0.79$) or ribavirin dosage (16% versus 15%, $P=0.92$) due to adverse events. The number of adverse events (investigator criteria) related to antiretroviral agents during the treatment of hepatitis C was 28 (35%) in the peg-IFN-α2b group and 32 (26%) in the peg-IFN-α2a group ($P=0.23$).

Eleven patients had an episode of liver decompensation during the treatment of hepatitis C: 4 (2%) in the peg-IFN-α2b group and 7 (2%) in the peg-IFN-α2a group ($P=0.86$). Only two of the patients with liver decompensation were treated with didanosine, both in combination with stavudine.

New episodes of AIDS-defining conditions during the treatment of hepatitis C were identified in one (<1%) patient in the peg-IFN-α2b group and three (1%) patients in the peg-IFN-α2a group ($P=0.81$). The AIDS-defining conditions identified were oesophageal candidiasis ($n=3$) and progressive multifocal leucoencephalopathy ($n=1$). During the treatment of hepatitis C, four patients (2%) died in the peg-IFN-α2b group and two (<1%) in the peg-IFN-α2a group ($P=0.46$). The causes of death in the peg-IFN-α2b and peg-IFN-α2a groups were liver-related in two cases each. There were no treatment-related deaths.

Discussion

This ambispective comparative study of peg-IFN-α2b and peg-IFN-α2a, both in combination with ribavirin, against chronic HCV infection in HIV-infected patients included 557 subjects who were well matched in baseline characteristics. Peg-IFN-α2a was associated with a higher ETR but also with higher relapse rates, resulting in similar SVR rates between the two peg-IFN formulations.

At the time of writing, three clinical trials comparing the safety and efficacy of the two approved types of peg-IFN in HCV mono-infected patients have been reported. In the IDEAL study, more than 3000 treatment-naive genotype 1 patients with chronic hepatitis C were randomized to three treatment arms: peg-IFN-α2b 1.5 μg/kg/week; peg-IFN-α2b 1.0 μg/kg/week plus ribavirin (800–1400 mg/day based on weight); and peg-IFN-α2a 180 μg/week plus ribavirin (1000–1200 mg/day based on weight). SVR rates were comparable between treatment groups regardless of the peg-IFN-α2b dose or formulation of peg-IFN used (peg-IFN-α2b 1.5 μg/kg/week, 40%; peg-IFN-α2b 1.0 μg/kg/week, 38%; and peg-IFN-α2a, 41%). Interestingly, peg-IFN-α2a was associated with a higher ETR but higher relapse rates.[14] However, different results were found in the MIST study,[15] a single-centre trial that randomized 431 HCV mono-infected patients to receive peg-IFN-α2a or peg-IFN-α2b, both in combination with ribavirin. In the peg-IFN-α2a group, the daily ribavirin dose for genotype 1 and 4 patients was 1000–1200 mg based on weight, while patients with genotype 2 or 3 received a fixed dose of ribavirin of 800 mg. In the peg-IFN-α2b group, ribavirin doses were 800–1400 mg based on weight for all genotypes. There were no significant differences in safety. However, the SVR rates were significantly higher in the peg-IFN-α2a group than in the peg-IFN-α2b group (66% versus 54%, $P=0.02$). Recently, a small randomized, multicentre, open-label clinical trial including 182 HIV/HCV co-infected patients that compared the efficacy and safety of peg-IFN-α2a ($n=96$) with that of peg-IFN-α2b ($n=86$), both in combination with ribavirin (800–1200 mg/day adjusted to body weight), has been reported.[13] No statistically significant differences were found in tolerance and efficacy; overall SVR was 42% for peg-IFN-α2b compared with 46% for PEG-IFN-a2a. This trial had a power of 80% to detect differences above 20%.

The rates of SVR found in our study for all genotypes with peg-IFN-α2a (33%) and for peg-IFN-α2b (31%) are within the limits of SVR found in randomized clinical trials of peg-IFN and ribavirin in HCV/HIV co-infected patients (27% to 40%).[9–11] These figures are also consistent with the results of a recent research synthesis of seven studies that found a pooled estimate of responses to peg-IFN and ribavirin in HCV/HIV co-infected patients of 33%.[17] The percentages of SVR in patients infected with HCV genotypes 1–4 who were treated with peg-IFN-α2a and peg-IFN-α2b (19% and 14%) are in the range of those previously reported in patients co-infected with HIV and HCV (14% to 29%).[9–11] Similarly, the rates of SVR in patients infected with HCV genotypes 2–3 who were treated with peg-IFN-α2a and peg-IFN-α2b (45% and 46%) are within the ranges previously reported in this population (44% to 73%).[9–11]

In this study, we identified two variables that were independently associated with increased odds of an SVR: HCV genotypes 2–3 and a CDC disease category other than C. The HCV genotype has consistently been found to be associated with SVR in clinical trials of peg-IFN in HIV/HCV co-infected patients.[9–12] Other factors associated with SVR in these trials were HCV-RNA level,[9] detectable HIV viral load at baseline and prior injection drug use,[10] as well as age, use of protease inhibitors and alanine aminotransferase level.[11]

The frequency of severe adverse events and the frequency of premature discontinuation of treatment due to adverse events were not different between patients treated with peg-IFN-α2b and those treated with peg-IFN-α2a, and were similar to those previously reported in clinical trials.[9–12] Likewise, we did not find significant differences between the groups in the frequency of reduction of peg-IFN dosage or ribavirin dosage due to adverse events or in the frequency of adverse events related to antiretroviral agents.

Eleven patients experienced liver decompensation during the treatment of hepatitis C (~2% in both groups): most of them had an advanced fibrosis score in the liver biopsy. This figure is consistent with that found in the APRICOT trial, in which 14 out of 859 patients (1.6%) experienced liver decompensation during IFN-based therapy; all of them had cirrhosis.[18] The risk factors associated with hepatic decompensation in this trial were increased bilirubin, decreased haemoglobin, increased alkaline phosphatase or decreased platelets, and treatment with didanosine.[18] In our cohort, only two patients were treated with didanosine, both in combination with stavudine.

During treatment with peg-IFN and ribavirin, <1% of our patients had new episodes of AIDS-defining conditions. Four (2%) patients in the peg-IFN-α2b group and two (<1%) in the peg-IFN-α2a group died during treatment: four of the deaths

were liver-related. This low frequency of clinical progression and death is consistent with the findings of clinical trials analysing peg-IFN plus ribavirin in HIV-infected patients with chronic HCV infection.[9–12]

This study has the limitations that are typical of retrospective observational cohort studies. In particular, it was not possible to analyse on-treatment viral kinetics (i.e. rapid virological response and early virological response), adherence to peg-IFN and ribavirin or decrease in levels of haemoglobin, neutrophils and platelets. However, the large number of patients in each group and the fact that the patients were fairly well matched in baseline characteristics support the conclusions.

The results of our study, which was carried out in a large cohort and in a clinical practice setting, suggest that there are no differences in effectiveness or safety between peg-IFN-α2a and peg-IFN-α2b for the treatment of chronic HCV infection in HIV-infected patients.

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The GESIDA HIV/HCV cohort

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Transparency declarations

None to declare.

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