INTRODUCTION

Due to shared routes of transmission, HIV co-infection is common among patients with chronic HCV infection. In Italy, it is estimated that 40% of HIV-infected patients are also infected with HCV. It is well known that HIV accelerates the course of HCV-related chronic liver disease. Although HIV has revolutionized the treatment of HCV, including its treatment in patients with HIV co-infection, few data are available on liver disease progression following viral eradication due to DAA treatment in HIV/HCV coinfected patients in real-life settings.

AIM

We aimed to assess the epidemiological, clinical, and treatment aspects in a real-life cohort of patients with HIV/HCV coinfection compared to HCV mono-infected patients after successful DAA treatment. We evaluated differences in clinical evolution in terms of liver-related complications in patients with cirrhosis after SVR, according to HIV coinfection.

METHODS

Patients consecutively enrolled in the PETER cohort between April 2014 and June 2019, who have started DAA treatment and with at least 12 weeks follow-up after the end of DAA treatment (median follow-up 38.9 months, range 4.1-60.8), were analysed. Emergence of a liver complication (de novo HCC occurrence, hepatic decompensation, Child-pugh (C-P) class deterioration) was evaluated in patients with pre-treatment diagnosis of liver cirrhosis. Variables independently associated to development of a liver complication after achieving SVR12 were evaluated by Cox proportional hazard models. Analyses were carried out using the STATA/SE 15.1 statistical package.

RESULTS

We included 244 HIV/HCV coinfected patients (74.6% men, median age 52, range 32-77) and 2870 HCV infected patients (54.1% men, median age 61, range 20-86). A total of 128 (52.2%) coinfected patients and 1445 (50.3%) mono-infected patients were started on IFN-based therapy in the F4/cirrhosis stage. Table 1 describes main baseline characteristics of the cirrhotic patients. There were no significant differences between cirrhotic monoinfected and coinfected patients for baseline AST, platelet count, serum albumin, bilirubin and international normalized ratio (INR) value.

Compared to HCV infected patients, HCV/HIV coinfected patients had significantly lower BMI (64.8% of coinfected patients are in the normal BMI group, while mono-infected patients are equally distributed between the normal (41.3%) and the overweight (44.5%) group (p<0.001). A significant different distribution of HCV genotypes in mono-infected compared to coinfected patients was observed. Among the 128 coinfected patients (n=759, 52.5%) were infected by HCV genotype 1b, whereas genotype 1a and 3 were dominant in coinfected patients (n=39, 30.5% and n=41, 32%, respectively). Compared to infected patients with significant lower age at mono-infected patients (p<0.001) and higher liver disease severer in terms of Child Pugh (C-P) class score (p<0.001). No differences were observed in the prevalence of HCC, history of decompenated cirrhosis and previous liver transplant, between mono-infected and coinfected patients.

Table 1. Baseline characteristics of cirrhotic patients

<table>
<thead>
<tr>
<th>Variable</th>
<th>Median</th>
<th>Range</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>62</td>
<td>29-86</td>
<td>0.01</td>
</tr>
<tr>
<td>BMI</td>
<td>25.3</td>
<td>17.5-37.5</td>
<td>0.001</td>
</tr>
<tr>
<td>Platelets (ref. &gt;100.000 U/µL)</td>
<td>246</td>
<td>70-1300</td>
<td>0.001</td>
</tr>
<tr>
<td>MELD</td>
<td>12</td>
<td>3-42</td>
<td>0.001</td>
</tr>
<tr>
<td>Child-Pugh</td>
<td>7</td>
<td>2-15</td>
<td>0.001</td>
</tr>
<tr>
<td>INR</td>
<td>1.13</td>
<td>0.9-1.51</td>
<td>0.001</td>
</tr>
</tbody>
</table>

DISCLOSURES

Nothing to disclose

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CONCLUSION

These real life data confirm the high effectiveness of DAA treatment in achieving SVR in advanced liver disease patients, independently by HIV coinfection. However, the effectiveness of DAA treatment in patients with advanced liver cirrhosis is not as high as its efficacy. HIV coinfection was not associated with a higher probability of developing liver complications in HCV-infected patients with advanced liver disease; an advanced pre-treatment liver disease (low platelet levels as surrogate of portal hypertension, low albumin levels, high INR and/or HCC), remained the main independent predictive factor for liver disease progression (C-P class deterioration, new events of liver decompensation and/or HCC development) following viral eradication.

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REFERENCES