

INTRODUCTION

High SVR rates are reported in patients treated with DAAs in the real life. However, other than HCV, several factors as NAFLD/NASH, HBV and HIV infection, alcohol use, present in patients with chronic HCV infection are also involved in the progression of liver damage. Potential liver disease progression in patients who present other than HCV risk factor following HCV eradication need to be better evaluated (1-4).

AIM

We aimed to evaluate the prevalence of cofactors involved in liver disease progression in HCV-treated patients who achieved the SVR12 following a DAA therapy in the PITER cohort (5).

METHOD

Data of HCV infected patients, consecutively enrolled in PITER (from January 2015 to September 2017), who were treated and achieved the SVR12, were evaluated. In patients for whom at least 6 months follow-up post-SVR12 was available, the Liver Function Tests and Child Pugh score changes according to the presence of alcohol use, non-virus- non-alcohol fatty liver, diabetes, hypertension, cardiovascular disease, Body Mass Index higher than 25, HBs Ag positivity, HIV positivity, were evaluated.

RESULTS

Of 3485 patients who achieved the SVR12, mean age 61 (SD 11 years), 1985 (54%) were men and 1965 (56%) had liver cirrhosis. Factors independently associated with liver cirrhosis by Logistic Regression Analysis in patients who achieved the SVR12 in PITER cohort are reported in Table 1.

Parameters	Adjusted OR	95% Confidence Limits	
Age	1.03	1.02	1.04
Male sex	1.19	1.09	1.29
BMI>25	1.29	1.02	1.63
Actual alcohol use	1.21	1.10	1.33
HCV Genotype 3	1.22	1.07	1.39
HIV positivity	1.08	0.91	1.29
HBV positivity	1.02	0.81	1.70
Previous IFN Therapy	1.22	1.13	1.31
Diabetes	1.53	1.37	1.71

Table 1

Age, male sex, BMI>25, actual alcohol use, HCV genotype 3, previous IFN treatment and diabetes were independent factors associated to cirrhosis by logistic regression analysis.

Of the overall patients evaluated (3485) following the SVR12:

- 1164 (33%) reported actual alcohol use
- 693 (20%) had non-virus-non-alcohol-related fatty liver
- 567 (16%) were diabetics
- 1781 (51%) had BMI>25 of whom 60% had hypertension and 30% had BMI≥30,
- 1060 patients had hypertension of whom 80% were on anti-hypertensive therapy
- 212 patients had ongoing cardiovascular disease (reported as chronic coronary artery disease)
- 43 (1%) were HBsAg positive
- 185 (5%) were HIV infected

Prevalence of none, 1 or more than 1 of the potential risk factors for liver disease progression (or progression from NAFLD to NASH) are reported in Figure 1.

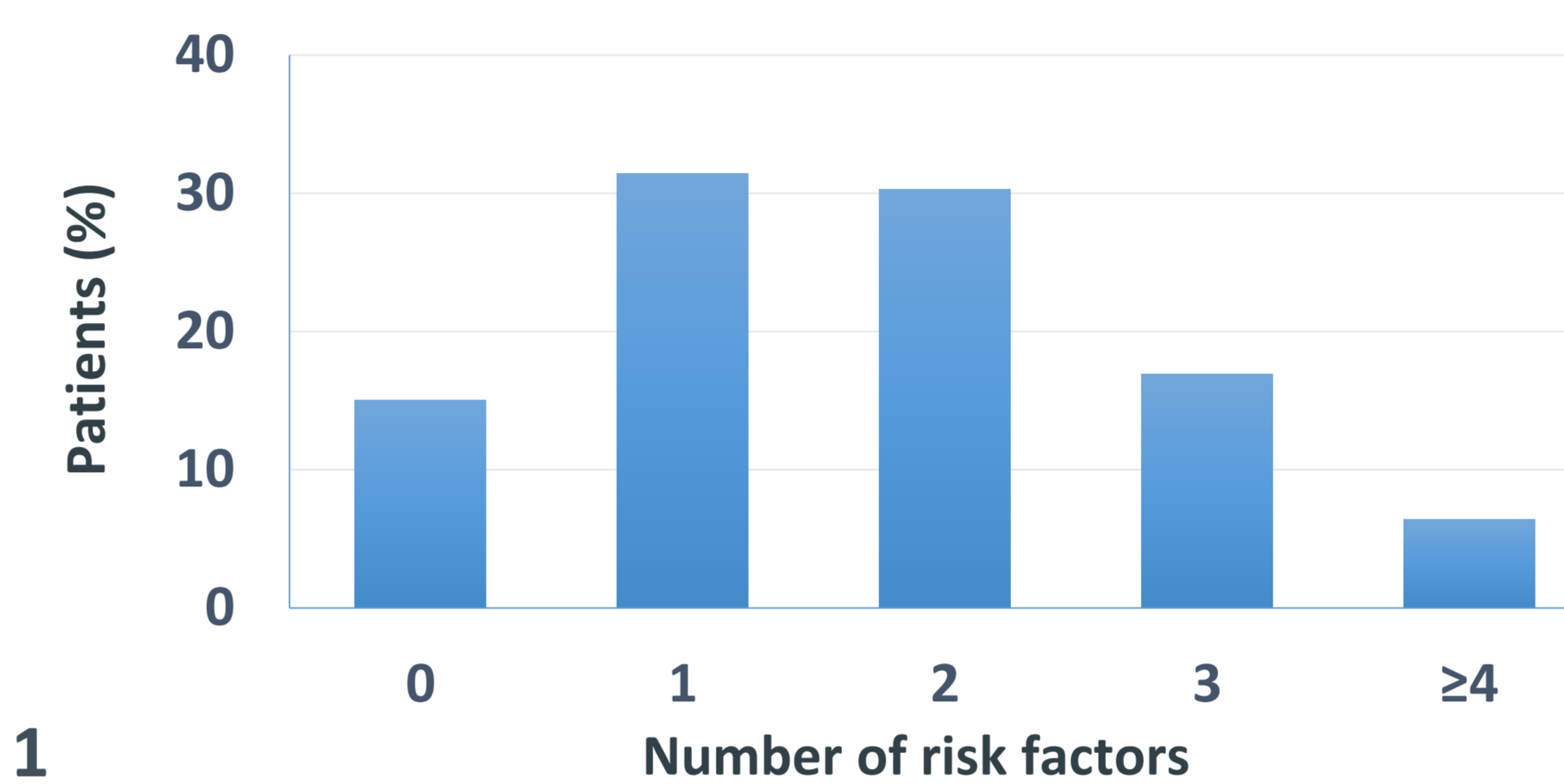


Figure 1

The prevalence of cofactors of liver disease progression in patients with liver cirrhosis according to the Child Pugh Class are reported in Table 2.

	CHILD				Total
	A	%	B or C	%	
	N. patients	%	N. patients	%	
Total	1680	85	285	15	1965
Steatosis	379	22.5	37	13.0	416
Actual alcohol use	565	33.6	105	36.8	670
HBsAg+	25	1.5	3	1.1	28
HIV+	52	3.1	39	13.7	91
Diabetes	355	21.1	67	23.5	422
Hypertension	577	34.3	79	27.7	656
BMI > 25	931	55.4	161	56.5	1092
Cardiovascular	108	6.4	13	4.6	121
NAFLD (Steatosis+Diabetes+Hypertension+BMI>25)	50	3.0	8	2.8	337
Number of risk factors					
0	208	12.4	33	11.6	241
1	537	32.0	90	31.6	627
2	531	31.6	97	34.0	628
3	313	18.6	48	16.8	361
4	73	4.3	16	5.6	89
5	18	1.1	1	0.4	19

Table 2

Diabetes, non-alcoholic liver steatosis and BMI>25 were present in 2% of patients with Fibrosis F0-F3 and in 3% of patients with cirrhosis. Of 1450 patients (942 patients with cirrhosis) for whom follow-up were available at least 6 months following the SVR12, no differences regarding liver function tests were observed according to the comorbidity pattern.

During a median follow-up of 10 months, improvement in Child Pugh score were observed in 72% of 324 patients with Child Pugh score higher than A6, in 25% of whom more than 2 points of Child Pugh score, without differences (p>0.5) according to the comorbidity pattern of concurrent risk factors for liver disease progression.

CONCLUSIONS

Concurrent risk factors for liver disease progression are present in a significant proportion of patients who successfully eradicated HCV infection. Although no further liver disease progression was associated to the presence of such cofactors in a short term evaluation, their role in the overall morbidity and mortality is a health issue that need to be addressed. In the lack of longer prospective studies, a modelling of liver disease progression after HCV eradication, using these real life data, is ongoing (6).

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