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Management of Hepatitis C Virus Infection in Patients with Cirrhosis

Aziza Ajlan and Hussien Elsiesy

Abstract

In this chapter, we review the history of HCV infection in patients with liver cirrhosis. Selection of appropriate regimens for HCV-infected patients with cirrhosis, consistent with approved indications, practice guidelines, and emerging data is presented. Finally, this chapter explains individualization of therapy to maximize SVR rates in HCV-infected patients with cirrhosis and to critically appraise the role of newer agents and regimens in the management of HCV-infected patients with cirrhosis.

Keywords: HCV, liver cirrhosis, treatment

1. Introduction

Hepatitis C virus (HCV) is the leading cause of liver cirrhosis and hepatocellular carcinoma (HCC) [1]. It remains the main indication for liver transplantation in North America and Europe [2]. The indication for liver transplantation has changed in the past two decades where NASH surpasses HBV to become the second most common cause of liver transplantation but HCV remains unchanged.

Chronic hepatitis C infection in patients with cirrhosis escalates the chances of developing severe liver-related complications, including hepatic decompensation, hepatocellular cancer and subsequently, death. It is been a matter of large debate whether to treat cirrhotic patients and what could be the potential benefit as cirrhosis is irreversible. However, multiple studies have shown that successful treatment of hepatitis C in patients with compensated cirrhosis will decrease subsequent cirrhosis-related complications.
HCV causes increased mortality compared to any other infection; therefore, both the American Association for the Study of the Liver (AASLD) and European Association for the Study of the Liver (EASL) guidelines recommend that treatment be indicated for all HCV-infected patients. However, due to outrageously high cost of the new directly acting antivirals (DAA), treating every HCV-infected patient is not practical even in countries with strong economy.

Given the high cost of the medications for HCV, both AASLD and EASL guidelines prioritize the treatment for specific population with liver cirrhosis among the top list. The goal of HCV treatment in patients with liver cirrhosis depends on the stage of disease. For Child’s class A compensated liver cirrhosis, the goal of treatment is to prevent progression or to reverse cirrhosis [3] and to decrease the prevalence of HCC [4–6].

The goal in decompensated liver cirrhosis is to reverse decompensation, delisting from the liver transplant waiting list or preventing the disease recurrence after liver transplantation [7–9]. More importantly, achieving sustained virological response (SVR) was associated with reduced all-cause mortality in patients with advanced fibrosis related to HCV [6].

Several studies have shown reversal of cirrhosis, delisting from liver transplant waiting list, improvement of liver function and decrease the risk of HCC in patients who achieved SVR. Decrease in model for end-stage liver disease (MELD) due to biochemical improvement without resolution of ascites may delay the liver transplantation by lowering the patient’s rank on the liver transplant waiting list.

There are also studies showing prevention of disease recurrence after liver transplantation on those who achieved SVR before liver transplantation.

We predict NASH to be the leading cause of liver transplant in the next decade, not only because of the growing obesity epidemic and increasing rate of diabetes, but because of the predicted long-term effect of HCV treatment.

The HCV treatment has evolved since the introduction of Interferon monotherapy in early 1990 until having several options of highly effective interferon-free DAA.

The first randomized multicentre trial comparing interferon alfa-2b versus no treatment in compensated HCV cirrhosis did not show benefit, whoever, it was small in number, have high drop-out rate and did not evaluate the patients who achieved sustained virological response (SVR) well but established safety [10]. In the same year, a study showed that patients with chronic hepatitis C who have an SVR to IFN therapy, there is a dramatic effect on normalization of ALT levels, improvement of histological activity and slowing of fibrosis progression [11].

From 2000 to 2011, the combination of PEG-IFN/RBV became the standard of care for HCV treatment, the overall SVR is 40–50% in genotypes 1 and 4 and 70–80% in genotypes 2 and 3; however, the SVR rate was significantly lower in patients with liver cirrhosis, about 22% for genotypes 1 and 4, and 55% for genotypes 2 and 3 [12–14].

Treating patients with decompensated HCV cirrhosis was challenging, it is associated with poor tolerance, higher side effect profile, and lower SVR rate. Everson and co-workers reported the results of a low-accelerating dose regimen of IFN or PEG-IFN with RBV in 124 patients
with decompensated cirrhosis. The SVR was 24%, it was significantly lower in patients with genotype 1 (13%) than in those with non-1 genotype (50%); \(P < 0.0001\). SVR was highly predictive of maintaining viral clearance after liver transplant [8].

Forns et al. evaluated the treatment with IFN a-2b/RBV in 30 patients awaiting Orthotopic liver transplant (OLT) [9]. A virological response was observed in nine patients (30%). After LT, six of them (20%) remained negative after liver transplantation.

The study by Carrion et al. evaluated PEG-IFN/RBV therapy in 51 patients with HCV and cirrhosis awaiting LT matched with 51 untreated controls [15]. The aim of this study is to evaluate both the prevention of post-transplantation recurrent HCV and the risk of bacterial infections during therapy. Only 15 patients (29%) were HCV RNA-negative at transplantation and 10 (20%) achieved an SVR after transplantation.

There is major safety concern of PEG-IFN therapy in patients with decompensated cirrhosis. The haematological side effect includes neutropenia (50–60%), thrombocytopenia (30–50%), and anaemia (30–60%). There is an increased risk of infection (4–13%) or hepatic decompensation during therapy (11–20%) [8, 9]. Carrion et al. reported high incidence of episodes of bacterial infection, mostly spontaneous bacterial peritonitis in treated patients (25%) compared to controls (6%) \(P = 0.01\) [15]. Variables independently associated with the occurrence of bacterial infections were antiviral treatment and a Child-Pugh score of B–C. The adverse effect of this therapy increase as the child score increase, where child C patients has very high complication rate with extremely low response. We reported the safety and efficacy of PEG-IFN and ribavirin therapy in 90 patients with liver cirrhosis, 18% required dose reduction, 33% stopped treatment because of adverse effects, 9% had deterioration of liver function, 7% died and 13% of patients SVR. The rate of serious complications was 16.3% in child’s class A, 48% in B, and 100% in C \(P = 0.005\). Serum albumin was a significant predictor for worsening liver function \(P = 0.007\), none of the child C patients achieved SVR [16].

2. New direct acting antivirals (DAAs)

Accordingly, the AASLD-IDSA guidelines consider any patient with chronic hepatitis C infection who is diagnosed with compensated cirrhosis highest priority for hepatitis C treatment [4].

For HCV-infected patients with decompensated cirrhosis or hepatocellular cancer, treatment of HCV may provide benefit, but the treatment plans and goals may need modifying if the patient is planning to undergo liver transplantation.

2.1. Patients with compensated cirrhosis

For patients with compensated cirrhosis (Child-Turcotte-Pugh Class A), including those with hepatocellular carcinoma, the AASLD/IDSA/IAS-USA guidance [4] recommends using the
same general treatment approach as used for patients without cirrhosis, with several key exceptions primarily related to duration of therapy or inclusion of ribavirin.

2.1.1. Genotype 1

2.1.1.1. Ledipasvir/sofosbuvir

Ledipasvir (90 mg) and sofosbuvir (400 mg) are a fixed-dose combination (Harvoni®) of two direct-acting antiviral agents that were initially studied in the ION-1 trial. The trial that included 865 treatment-naïve patients, looked at the length of treatment (12 weeks versus 24 weeks) as well as the need for RBV [5]. SVR12 rates exceeded 97%, with no added benefit observed with longer treatment duration, the addition of RBV length of treatment, nor HCV genotype 1 subtype. In the study, 16% of the included patients had cirrhosis. The presence of cirrhosis did not affect SVR12 rates compared with those without cirrhosis (97%) versus (98%) [5].

2.1.1.2. Paritaprevir/ritonavir/ombitasvir + dasabuvir (PrOD)

The 3D combination was studied in the TURQUOISE-II and TURQUOISE-III trials. The trial included 261, HCV genotype 1a and CTP class A, the patients were both treatment-naïve and -experienced. The study compared 12 weeks or 24 weeks of PrOD regimen with the addition of RBV. SVR12 rates were higher in patients who received 24 weeks arm (89% vs. 95%) [6]. Factors that may have contributed to these differences could be the inclusion of patients who failed previous PEG-IFN/RBV therapy. Overall, treatment-naïve patient had slightly better response to therapy (92% vs. 95%). Interestingly, in patients with HCV genotype 1b patient, the SVR12 rates reached 98.5% in the 12-week arm [6]. Subsequently, the TURQUOISE-III trial questioned the role of RBV with the 3D regimen for 12 weeks in patients with HCV genotype 1b and compensated cirrhosis. Among the 60 patients included, more than 50% of the patients had negative predictors of response as follows: 55% treatment-experienced, 83% with IL28B non-CC genotype, 22% had platelet counts of greater than $90 \times 10^9 \text{L}^{-1}$, and 17% had albumin levels greater than 3.5 g/dL. SVR12 rates were 100%. Hence, this regimen was approved for HCV genotype 1b for 12 weeks irrespective of previous treatment history or the presence or of cirrhosis [7].

The PrOD regimen, however, carries FDA warning [8]. In October 2015, the US FDA announced that the PrOD and PrO are contraindicated in patients with Child-Turcotte-Pugh (CTP) class B or C cirrhosis. This was based on reports by the manufacturer of accelerated liver injury in patients who were receiving PrOD or PrO. The onset of liver harm and decompensating incidents were observed mainly during the first month of therapy and mainly involved a quick rise in total and direct bilirubin, as well as a concomitant increase in liver transaminases. Timely recognition and termination of PrOD or PrO resulted in resolution of injury, death was reported in two cases with compensated cirrhosis. If the decision is made to initiate treatment with PrOD or PrO, patients should be made aware of the risks associated with such therapy in addition to adequate monitoring.
2.1.1.3. **Simeprevir + sofosbuvir**

Simeprevir + sofosbuvir regimen were studied in the OPTIMIST-2 trial. The single armed, open-label trial looked at 12 weeks of simeprevir plus sofosbuvir in 103 cirrhotic patients [9]. SVR12 rates were 88% (44/50) of treatment-naïve and 79% (42/53) of treatment-experienced patients with the total SVR12 rate was 83% (86/103). Furthermore, both genotype 1a and the presence of Q80K mutation negatively affected SVR12 (genotype 1 and 1b 84% [26/31] and 92% [35/38], respectively. And 74% [25/34] with Q80K mutation. Currently, there is no data that proves that extending treatment, with or without the addition of RBV, will increase efficacy of these two groups. Hence, until further data proves otherwise, this regimen should be avoided in patients with genotype 1a or in the case Q80K mutation is present.

2.1.1.4. **Daclatasvir + sofosbuvir**

Cirrhotic patients tend to take advantage from extension of therapy with daclatasvir and sofosbuvir to 24 weeks, with or without RBV [10, 11]. The data from ALLY-1 trial investigated daclatasvir and sofosbuvir with RBV dosed at 600 mg, in 60 patients with advanced cirrhosis [12]. Only 76% of patients with HCV genotype 1a (n = 34) and 100% of patients with HCV genotype 1b (n = 11) achieved an SVR at 12 weeks (SVR12). It is unclear how many treatment failures were among treatment-naïve patient was 54% or those with CTP class A cirrhosis. SVR was significantly lower in CTP class C cirrhosis (54%) when compared with CTP classes A and B 92% and 94% (see **Table 1**).

<table>
<thead>
<tr>
<th>SVR12 rates in patients with Child Pugh A cirrhosis</th>
<th>GT1a</th>
<th>GT1b</th>
<th>GT2</th>
<th>GT3</th>
<th>GT4</th>
<th>GT5/6</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SOF+SIM 12 weeks</strong></td>
<td>83% (9)*</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td><strong>SOF+DAC 12 weeks</strong></td>
<td>76% (12)</td>
<td>100% (12)</td>
<td>NA</td>
<td>85.9% (27)</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td><strong>SOF+DAC 24 weeks</strong></td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td><strong>SOF+LED+RBV 12 weeks</strong></td>
<td>97.98% (5)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td><strong>SOF+LED 24 weeks</strong></td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td><strong>PrOD 12 weeks</strong></td>
<td>98.5% (6)</td>
<td>100% (6)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td><strong>PrO 12 weeks</strong></td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>96% (30)</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td><strong>SOF+VEL 12</strong></td>
<td>99%</td>
<td>95%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td><strong>GRZ+ELB 12</strong></td>
<td>97% (13, 14)</td>
<td>99%</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td><strong>GRZ+ELB 16</strong></td>
<td>100% (15)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
</tbody>
</table>

*With 88% (44/50) of treatment-naïve and 79% (42/53) of treatment-experienced patients.

*With ribavirin.

$100%$ SVR12 rates achieved with extending the duration to 16 weeks.


**Table 1.** SVR12 rates among HCV-infected patients with compensated cirrhosis.
2.1.1.5. Elbasvir/grazoprevir

For genotype 1a, recommendations for cirrhotic patients are based on 92 (22%) patients in the phase III C-EDGE trial that had Metavir F4 disease [13]. SVR 12 was 97% in the subgroup of cirrhotic patients. A similar 97% (28/29) SVR 12 rate had previously been demonstrated in genotype 1 cirrhotic treatment-naïve patients treated with 12 weeks of elbasvir/grazoprevir without ribavirin in the open-label phase II C-WORTHY trial [14]. The presence or absence of compensated cirrhosis does not appear to alter the efficacy of the elbasvir/grazoprevir regimen [13, 14].

The presence of NS5A resistance-associated variants (RAVs) at baseline was found to be associated with reduced efficacy in patients with genotype 1a, and was not apparent with genotype 1b [13]. In this phase III open-label trial of elbasvir/grazoprevir that enrolled treatment-experienced patients; among 58 genotype 1a patients who received 16 weeks of therapy with elbasvir/grazoprevir plus ribavirin, there were no virologic failures and the SVR12 rates were 100% [15–17].

2.1.1.6. Sofosbuvir/velpatasvir

The use of this combination in patients with decompensated cirrhosis was investigated in the ASTRAL-4 trial. The study was multicentre, open-label patients were randomly assigned in a 1:1:1 ratio to receive a fixed-dose combination tablet containing 400 mg of sofosbuvir and 100 mg of velpatasvir, administered orally once daily for 12 weeks; sofosbuvir-velpatasvir plus ribavirin once daily for 12 weeks; or sofosbuvir-velpatasvir once daily for 24 weeks. Ribavirin was administered orally with food twice daily, with the dose determined according to body weight (1000 mg daily in patients with a body weight of greater than 75 kg and 1200 mg daily in patients with a body weight ≥75 kg). The overall SVR12 rates in the three groups were 83, 94 and 86%, respectively. The study highlights a potential role of RBV in such population [18]. Nineteen percent of the patients included in the ASTRAL-1 study had cirrhosis and observed SVR12 rates of 99% when received sofosbuvir/velpatasvir for 12 weeks [19].

2.1.2. Genotype 2

Sofosbuvir (400 mg daily) was combined with weight-based RBV for treatment-naïve patients with HCV genotype 2 infection in three clinical trials, each of which enrolled patients with HCV genotype 2 or 3: FISSION, POSITRON and VALENCE with very high SVR12 rates [20–22]. However, patients with cirrhosis have lower response rates that were seen in treatment-naïve patients with cirrhosis compared to in those without cirrhosis [23]. One may consider extending treatment duration when cirrhosis is present despite the lack of data to support such extension, as longer treatment duration is known to improve SVR in treatment-experienced patients with cirrhosis [22, 24]. Due to the small numbers of patients with HCV genotype 2 infection and cirrhosis enrolled in the registration trials, several phase III b studies are ongoing to specifically determine the appropriate length of treatment for this subgroup of patients (see Table 1).
2.1.3. Genotype 3

2.1.3.1. Sofosbuvir/daclatasvir

ALLY-3 is a phase III study of the once-daily NS5A inhibitor daclatasvir plus sofosbuvir for 12 weeks; the study included 101 treatment-naïve patients and demonstrated an SVR12 rate of 90%. Cirrhotic patients (Metavir F4), 58% achieved SVR12 [25]. Hence extension of therapy may be considered in such cases. European compassionate use program has supported these recommendations in cohort studies, which reported and improvement in rates of up to 70% versus 86% when daclatasvir and sofosbuvir was used for 12 weeks and 24 weeks. RBV did not seem to have a big impact on SVR12 (85.9% without RBV compared to 81.3% with RBV). SVR12 rates were also higher in those with compensated Child-Pugh A cirrhosis (85–90% compared to 70.6% in child B/C). Previous data suggested that SVR 12 rates were higher in treatment-naïve patients (91–100%) compared to experienced (81–82%) [26].

2.1.4. Genotype 4

2.1.4.1. Ledipasvir/sofosbuvir

The SYNERGY trial was an open-label study evaluating 12 weeks of ledipasvir/sofosbuvir in 21 HCV genotype 4-infected patients, Among that 60% were treatment-naïve and 43% had advanced fibrosis (Metavir stage F3 or F4) [27]. All patients achieved an SVR12. Note that the study used an assay by ROCH with lower limit of quantitation (LLOQ) of 43 IU/ml, while the AASLD guidelines recommended to use an assay with LLOQ of 25 IU/ml. However, this had no impact on SVR12 results [28].

2.1.4.2. Paritaprevir/ritonavir/ombitasvir (PrO)

Pro regimen has interesting SVR12 rates according to the AGATE-I trial. The trial randomized 120 subjects with genotype 4 HCV and compensated cirrhosis to 12 weeks or 16 weeks of paritaprevir/ritonavir/ombitasvir (PrO) in addition to weight-based ribavirin. The SVR12 rates were 96% and 100% in the 12 week and 16 week arms, respectively [29]. On the other hand, the AGATE-II trial randomized 60 patients with compensated (1:1) to Pro for either 12 weeks or 24 weeks. SVR12 rates in the 12 weeks group were 97% versus 93% in the 24 week group [30].

2.1.4.3. Sofosbuvir/simiprevir

In a study by Moreno et al., the combination was studied in patients with advanced fibrosis/cirrhosis. All patients achieved end of treatment response but SVR12 data were not available [31]. In another study by Kayali et al., the combination was found to achieve SVR12 rates of 77% MELD scores remain unchanged. Interestingly, black gender and BMI were identified as independent negative predictors of response in univariate regression analysis (see Table 1) [32].
3. Patients with decompensated cirrhosis

3.1. Sofosbuvir/ledipasvir

The SOLAR-1 study was a multicentre, randomized controlled trial of 108 patients with HCV genotype 1 and 4 who had decompensated cirrhosis, of whom 59 were classified as CTP class B and 49 classified as CTP class C cirrhosis. Subjects were randomly assigned to receive daily fixed dose combination ledipasvir/sofosbuvir and RBV (initial dose of 600 mg, increased as tolerated) for 12 or 24 weeks. Extension of treatment in cirrhotic patients did not seem to affect SVR rates much. For CTP B patients, SVR rates were 87% versus 89% in subjects who received 12 versus 24 weeks, respectively. Likewise, the rates of SVR CTP class C subjects were 86 and 87%, respectively, with 12 and 24 weeks of antiviral therapy [33]. During the study, only one patient with CTP class C cirrhosis died.

The SOLAR-2 study was a multicentre randomized controlled trial of 108 subjects with decompensated cirrhosis secondary to HCV genotypes 1 and 4. Some of the patients were treatment-experienced, with CTP class B cirrhosis or CTP class C cirrhosis. The patients were randomly assigned to receive daily fixed-dose combination ledipasvir/sofosbuvir and RBV (initial dose of 600 mg, increased as tolerated) for 12 weeks or 24 weeks. Sustained virologic response (SVR) was achieved in 87% of those given the 12-week treatment course and 89% of those given the 24-week treatment course. On the 4th week of treatment, the total bilirubin and serum albumin levels improved compared with baseline in all patients. Despite the fact that some patients experienced worsening of hepatic function, baseline CTP and model for end-stage liver disease (MELD) scores improved in more than 50% of the treated patients. Five patients died during the study period but none of the death occurred was attributed to the study medication. Adverse events were more common in the 24-week arm (34%) than in the 12-week arm (15%). These results indicate that a 12-week course of ledipasvir/sofosbuvir and RBV (initial dose of 600 mg, increased as tolerated) is an appropriate regimen for patients with decompensated cirrhosis who are infected with HCV genotype 1 or 4. Such therapy may lead to objective improvements in hepatic function and reduce the likelihood of recurrent HCV infection after subsequent transplantation [33].

3.2. Sofosbuvir/daclatasvir

Patients with advanced cirrhosis (Child-Turcotte-Pugh [CTP] class B and C; \( n = 60 \)) were particularly investigated in the ALLY-1 study [34]. The study found the use of daclatasvir (60 mg daily) with sofosbuvir (400 mg) and low initial dose of RBV (600 mg) for 12 weeks to treatment-naive and -experienced patients with HCV genotype 1 infection. The overall SVR12 rate was 83% among those with advanced cirrhosis. The SVR12 rate was slightly lower in patients with genotype 1a compared with patients with genotype 1b (76 and 100%, respectively). Response rates were also affected by severity of disease among those with advanced cirrhosis (94% SVR12 rates in patients with CTP class B and 56% in patients with CTP class C). Patients with genotype 3 had also lower SVR12 rates 83%.
In another real-world study by Foster et al., involving 235 genotype 1 patients with decompensated cirrhosis, the SVR rates were comparable in the genotype 1 subjects (n = 235) receiving SOF/LDV/RBV or SOF/LDV (86% vs. 81%) and those receiving SOF/DCV/RBV or SOF/DCV therapy (82–60%). In this study, 91% of the patients received ribavirin with 20% requiring a RBV dose reduction and only 6% discontinued RBV. Improvement in MELD scores was observed in 42% of treated patients and worsening occurred in 11%. Moreover, 14 deaths occurred with relatively higher incidence of SAE (26%) but none were attributed to study medication.

3.3. Genotype 2 and 3

A multicentre, compassionate use study included 101 genotype 3 patients to be treated with daclatasvir (60 mg), sofosbuvir (400 mg) ± RBV for 24 weeks [35]. Of those, 81% had CTP class B cirrhosis, the MELD score was higher than 15 in 16%, and 7% were post-liver transplant. The reported SVR 12 data has demonstrated an SVR of 85–100%. Two patients died while 22 patients had an SAE and therapy was discontinued in five subjects. Summary of SVR in Child B and C (Table 2).

<table>
<thead>
<tr>
<th></th>
<th>GT1a</th>
<th>GT1b</th>
<th>GT2</th>
<th>GT3</th>
<th>GT4</th>
<th>GT5/6</th>
</tr>
</thead>
<tbody>
<tr>
<td>SOF +SIM 12 weeks</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>SOF +DAC 12 weeks</td>
<td>76%</td>
<td>100%</td>
<td>NA</td>
<td>83%</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>SOF +DAC 24 weeks</td>
<td>NA</td>
<td>NA</td>
<td>85%</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>SOF +LED +RBV 12 weeks</td>
<td>87%</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>SOF +VEL 12</td>
<td>88</td>
<td>89</td>
<td>100</td>
<td>50</td>
<td>100</td>
<td>NA</td>
</tr>
<tr>
<td>SOF +VEL +RBV 12</td>
<td>94</td>
<td>100</td>
<td>100</td>
<td>85</td>
<td>100</td>
<td>NA</td>
</tr>
<tr>
<td>SOF +VEL 24</td>
<td>93</td>
<td>88</td>
<td>75</td>
<td>50</td>
<td>100</td>
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</tr>
</tbody>
</table>

*SVR12 rate was 94% among patients with CTP class B cirrhosis but only 56% among patients with CTP class C cirrhosis.

Table 2. SVR12 rates in patients with Child Pugh B and/or C cirrhosis.

4. Summary

There is a remarkable advance in treatment of HCV in the recent few years allowing an excellent result in difficult to treat patients with liver cirrhosis with good safety profile.
Treating HCV in patients with liver cirrhosis is a high priority to prevent decompensation and prevent HCV recurrence after liver transplantation.

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