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MINIREVIEWS

Role of etiological therapy in achieving recompensation of decompensated liver cirrhosis

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Abstract

The traditional view of the decompensated stage as a point of no return in the natural history of liver cirrhosis (LC) is currently being questioned. This is due to the appearance of data indicating the possibility of restoring the structure and function of the liver, reducing the portal pressure with a positive effect on complications associated with portal hypertension and decreasing the risk of developing hepatocellular carcinoma after elimination of the etiological factor. To create a unified understanding the recompensation of decompensated LC, at the Baveno VII consensus workshop were developed criteria confirming it. At the moment, the efficacy of etiological therapy in achieving established criteria for recompensation has been evaluated only in patients with alcohol-related, as well as hepatitis B virus-related and hepatitis C virus-related decompensated LC. The purpose of the review is to provide up–to-date information on the role of etiological therapy in achieving recompensation of decompensated LC according to Baveno VII criteria. So far, only the first steps have been taken in studying this problem. To further understand it, research is needed to identify pathophysiological mechanisms, modifying factors, predictors, and potential noninvasive biomarkers of recompensation of decompensated LC.

Key Words: Liver cirrhosis; Decompensation; Portal hypertension; Complications; Etiological therapy; Recompensation

Core Tip: Numerous publications in recent years have shown the efficacy of etiological therapy in achieving recompensation of decompensated liver cirrhosis (LC). The criteria for the recompensation of alcohol-related, as well as hepatitis B virus-related and hepatitis C virus-related decompensated LC were developed at the Baveno VII consensus workshop. This review provides up-to-date information on the role of etiological therapy in achieving recompensation of decompensated LC according to these criteria.

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INTRODUCTION

Liver cirrhosis (LC) is an unfavorable event in the evolution of most chronic liver diseases. In the natural history of LC there are a compensated stage (without or with clinically significant portal hypertension (PH) and a decompensated stage (Figure 1). The development of clinically significant PH in compensated LC patients serves as a key prognostic factor, since it leads to an increased risk of the first decompensation, the most important manifestation of which is gastroesophageal variceal bleeding (GEVB)[1]. The diagnostic criteria for clinically significant PH are hepatic venous pressure gradient (HVPG) ≥ 10 mmHg, or liver stiffness values by transient elastography ≥25 kPa[2]. Further LC decompensation is a stratification variable of poor prognosis and is accompanied by a decrease in median survival to 2-4 years[3]. This is due to the development of such life-threatening complications as recurrent GEVB, recurrent ascites (requires ≥ 3 large volume paracentesis within 1 year), recurrent encephalopathy, spontaneous bacterial peritonitis and/or hepatorenal syndrome-acute kidney injury, jaundice[4].

The traditional view of the decompensated stage as a point of no return in the natural history of LC is currently being questioned. Moreover, in 2021, at the Baveno VII consensus workshop has defined the concept of recompensation of decompensated LC. It implies that after elimination of the etiological factor, there is at least a partial regression of structural and functional disorders in the liver, reduction of portal pressure with a positive effect on PH-related complications[5]. At the moment, the efficacy of etiological therapy in achieving developed criteria for recompensation has been evaluated only in patients with alcohol-related, as well as hepatitis B virus (HBV)-related and hepatitis C virus (HCV)-related decompensated LC, which is due to the lack of clear ideas about the etiology of other chronic liver diseases[6,7]. Nevertheless, current data indicate the prospects of this approach, for example, in patients with non-alcoholic steatohepatitis-related LC, when, under certain circumstances, regression of its characteristic histological signs may occur[8]. In a study by Hofer *et al*[9] recompensation of decompensated LC in patients with primary biliary cholangitis was achieved by ursodeoxycholic acid therapy, especially in those who showed an adequate biochemical response after a 1-year of treatment according to Paris-II criteria.

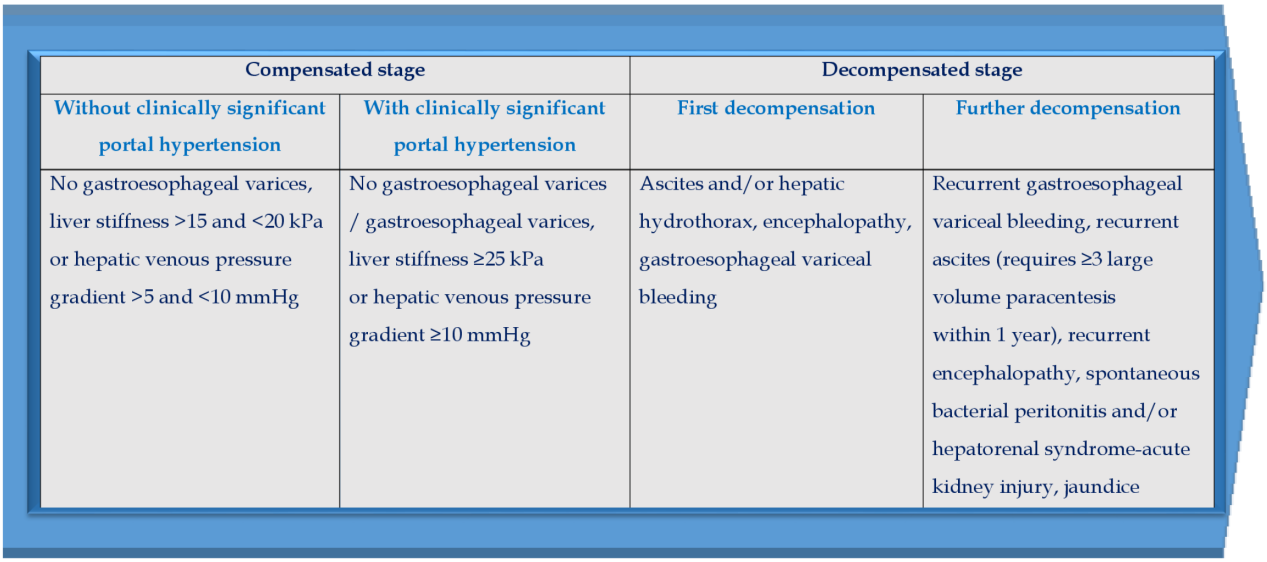
The purpose of the review is to provide up–to-date information on the role of etiological therapy in achieving recompensation of decompensated LC according to Baveno VII criteria. The PubMed and EMBASE databases, the Web of Science platform, the Google Scholar retrieval system, the Cochrane Database of Systematic Reviews, Reference Citation Analysis (https://www.referencecitationanalysis.com/), and the reference lists from related articles were used to search for relevant publications. Articles corresponding to the aim of the review were selected for 1996-2024. Articles were selected that evaluated the efficacy of etiological therapy in achieving recompensation of decompensated LC.

MODERN CONCEPT OF RECOMPENSATION OF DECOMPENSATED LC

According to the decisions of the Baveno VII consensus workshop, clinical confirmation of recompensation of decompensated LC requires compliance with the following criteria: (1) Removal/suppression/cure of the primary etiological factor of LC, namely long-term and complete alcohol abstinence for alcohol-related liver disease, suppression of HBV replication and elimination of HCV; (2) Resolution of ascites (off diuretics), encephalopathy (off lactulose/rifaximin) and absence of recurrent GEVB (for at least 12 months); and (3) Stable improvement of liver function tests [albumin, international normalized ratio (INR), bilirubin].

It should be noted that the resolution of ascites while on diuretics or after transjugular intrahepatic portosystemic shunt (TIPS) and/or the absence of recurrent GEVB while on traditional non-selective β-blockers (NSBBs) + endoscopic band ligation (EBL) or carvedilol + EBL or after TIPS without removal/suppression/cure of the primary etiological factor of LC and without improvement in liver synthetic function, is not evidence of recompensation[5].

In addition, normalization of the clinical status after removal/suppression/cure of the etiological factor is often not enough to achieve stable recompensation of decompensated LC. In order to prevent further decompensation, it is also necessary to influence the pathophysiological mechanisms of its development, which include: (1) Liver fibrosis and associated increase in intrahepatic vascular resistance; (2) Hyperdynamic circulatory state characteristic of clinically significant PH; and (3) Disturbance of the gut-liver axis contributing to systemic inflammation and immune dysfunction [10].



Compensated stage		Decompensated stage	
Without clinically significant portal hypertension	With clinically significant portal hypertension	First decompensation	Further decompensation
No gastroesophageal varices, liver stiffness >15 and <20 kPa or hepatic venous pressure gradient >5 and <10 mmHg	No gastroesophageal varices / gastroesophageal varices, liver stiffness ≥25 kPa or hepatic venous pressure gradient ≥10 mmHg	Ascites and/or hepatic hydrothorax, encephalopathy, gastroesophageal variceal bleeding	Recurrent gastroesophageal variceal bleeding, recurrent ascites (requires ≥3 large volume paracentesis within 1 year), recurrent encephalopathy, spontaneous bacterial peritonitis and/or hepatorenal syndrome-acute kidney injury, jaundice

Figure 1 Natural history and stages of liver cirrhosis.

Currently, it has been established that etiological therapy of diseases, the natural history of which is accompanied by liver fibrosis, is an effective method not only for their prevention, but also for the reversal of histological disorders with the restoration of liver structure and function to a normal state, contributing to a reduction in intrahepatic vascular resistance underlying the development of PH[11]. However, in some cases, clinically significant PH may persist despite achieving sustained virological response (SVR) and a decrease in liver stiffness values by transient elastography[12]. That is because the PH in LC is a consequence not only of morphofunctional rearrangement of the hepatic vasculature[13], but also of the subsequent formation of portosystemic shunts and the development of hyperdynamic circulatory state as a result of complex processes of angiogenesis, vascular remodeling and endothelial dysfunction[14]. Considering that maintaining elevated portal pressure contributes to further LC decompensation, NSBBs intake should not be discontinued until signs of clinically significant PH are eliminated, even in LC patients who have achieved recompensation[15].

Systemic inflammation and immune dysfunction are also important triggers for further LC decompensation. In this regard, it is obvious that in order to prevent further LC decompensation, it is necessary to affect key links in the pathogenesis, for example, a disturbed gut-liver axis, where specific alterations in the composition and function of gut microbiota play a crucial role[16].

ALCOHOL-RELATED DECOMPENSATED LC

Worldwide, approximately 2.4 billion people consume alcohol, while alcohol-related liver disease are one of the 30 main causes of death. In 2010, the rate of alcohol-related LC death worldwide was 7.2 per 100000 people[17]. According to the aggregate data of the Global Burden of Diseases, Injuries, and Risk Factors Study 2017 Cirrhosis Collaborators, the proportion of alcohol-related decompensated LC among other causes was 23%[18]. It is predicted that by 2040, the incidence of alcohol-related decompensated LC will increase from 9.9 to 17.5 per 100000 patient-years[19].

Alcohol abstinence is a top priority and one of the main therapeutic approaches for all forms of alcohol related liver diseases, including LC[20]. Long-term alcohol abstinence has a positive effect on the natural history of alcohol-related LC and significantly reduces the risk of decompensation[21]. Back in the mid-90s of the last century, Vorobioff *et al*[22] revealed a relationship between alcohol abstinence in patients with alcohol-related decompensated LC and improved liver function assessed by the Child-Turcotte-Pugh (CTP) score, reduction of HVPG and regression of gastroesophageal varices. Recent studies have shown that a significant proportion of liver transplant candidates with alcohol-related decompensated LC can be removal from the waiting list due to abstinence-associated recompensation[23,24]. Independent predictors of abstinence-associated recompensation in patients with alcohol-related decompensated LC may be values of the model for end stage liver disease (MELD) score < 20 points and serum albumin levels ≥ 32 g/L at the time of listing[25].

In a retrospective, single-center, observational study involving 204 patients with alcohol-related decompensated LC, long-term alcohol abstinence contributed to recompensation in 18.1% of cases at 2 years, which resulted in a > 90% risk reduction in liver-related mortality[26]. In a retrospective, single-center, observational study involving 320 patients with alcohol-related decompensated LC and a median HVPG of 20 mmHg (interquartile range: 17-23 mmHg), alcohol abstinence was linked to a significantly reduced risk of further decompensation [adjusted hazard ratio (aHR): 0.391, P < 0.001] as in groups with HVPG 10-19 mmHg (P < 0.001) and HVPG ≥ 20 mmHg (P = 0.002), as well as liver-related (aHR: 0.428, P < 0.001) and all-cause (aHR: 0.453, P < 0.001) mortality, after adjusting for baseline HVPG, MELD, and previous decompensation. The 3-year decompensation probability was 32.4% *vs* 60.0% in HVPG 10-19 mmHg and 57.5% *vs* 82.6% in HVPG ≥ 20 mmHg for abstinent patients *vs* active drinkers, respectively[27].

HBV AND C VIRUS-RELATED DECOMPENSATED LC

Chronic HBV and HCV infections damage liver cells, leading to liver fibrosis and the progression to LC. Statistically, the risk of developing LC is as high as 40% for HBV infection and 10%–20% for HCV infection if not treated with antiviral therapy[28]. At the same time, recent studies have shown that achieving SVR in patients with HBV-related and HCV-related decompensated LC can stop the progression of the disease, contributes to a significant improvement of histological necroinflammation and fibrosis, lead to regression of LC, decrease the risk of life-threatening complications associated with decompensation and reduce the need for liver transplantation[29,30]. However, their important limitations are the small number of randomized controlled trials (RCTs) and the retrospective design of many studies.

HBV-related decompensated LC

Chronic HBV infection is an important cause of morbidity and mortality worldwide. A systematic analysis for the Global Burden of Disease Study 2019 showed the presence of chronic HBV infection in 316 million people. HBV-related diseases were the cause of 555000 (487000-630000) deaths, and HBV-related LC was responsible for 331000 (279000-392000) deaths [31].

Currently, pegylated interferon and 6 nucleos(t)ide analogues (NAs), including lamivudine, adefovir dipivoxil, entecavir, telbivudine, tenofovir disoproxil fumarate and tenofovir alafenamide fumarate, have been approved for the treatment of chronic HBV infection. In patients with HBV-related LC, indefinite treatment with NAs is recommended, regardless of the serum level of HBV DNA. Patients with HBV-related decompensated LC should be treated with NAs with high barrier to HBV resistance (entecavir, tenofovir disoproxil fumarate, tenofovir alafenamide), irrespective of the level of HBV replication[32]. Many studies have shown the efficacy of NAs in achieving recompensation of HBV-related decompensated LC (Table 1)[33-42]. For example, in a systematic review and meta-analysis of 39 RCTs and observational studies involving a total of 14212 patients with HBV-related decompensated LC and clinically significant PH, therapy with NAs prevented further decompensation [relative risk (RR): 0.51, 95%CI: 0.37-0.71], decreased the risk of GEVB (RR: 0.44, 95%CI: 0.26-0.74), ascites (RR: 0.10, 95%CI: 0.01-1.59) and hepatocellular carcinoma (RR: 0.48, 95%CI: 0.30-0.75), and also reduced the likelihood of liver transplantation or death (RR: 0.36, 95%CI: 0.25-0.53). The first-line NAs (entecavir or tenofovir) were superior to non-first-line NAs in improving these outcomes (RR: 0.85, 95%CI: 0.75-0.97 and RR: 0.85, 95%CI: 0.73-0.99, respectively)[43].

A number of studies have focused on determining predictors of recompensation in NAs-treated patients with HBV-related decompensated LC. In a study by Deng *et al*[44], a serum albumin levels ≥ 34 g/L at treatment with entecavir week 24 predicted recompensation of HBV-related decompensated LC by week 120. The accuracy of the BC2AID score, developed to predicted recompensation of HBV-related decompensated LC and based on six independent clinical parameters, such as serum total bilirubin levels ≤ 5 mg/dL, absence of severe complications, α-fetoprotein ≥ 50 ng/mL, alanine aminotransferase (ALT) ≥ 200 IU/L, INR ≤ 1.5, and ≤ 6 months from initial decompensation until initiation of NAs therapy, was significantly higher than that of the CTP, MELD, MELDNa and BE3A scores (0.813 *vs* 0.691, 0.638, 0.645 and 0.624, respectively; all $P < 0.05$)[45]. The nomogram created by Wen *et al*[46] of the six independent factors, including age, ALT, serum albumin levels, serum sodium, α-fetoprotein, and SVR, predicted recompensation in NAs-treated patients with HBV-related decompensated LC significantly better than CTP, MELD, MELDNa, MELD 3.0, and albumin–bilirubin scores.

The criteria for recompensation in NAs-treated patients with HBV-related decompensated LC can be values of the MELD score < 10 points and/or liver function tests within CTP class A (serum albumin levels > 35 g/L, INR < 1.5 and serum total bilirubin levels < 34 μmol/L)[39].

HCV-related decompensated LC

HCV infection is one of the main causes of chronic liver disease worldwide[47], which remains the leading cause of global deaths related to LC[48]. Currently, direct acting antivirals (DAAs) have been approved for the treatment of chronic HCV infection within interferon-free combination regimens. Their action is aimed at simultaneous inhibition of several targets in the HCV life cycle, namely non-structural proteins NS3, NS4A, NS5A. The choice of DAAs treatment regimen depends on the HCV genotype, the presence or absence of LC, clinical experience and financial capabilities. In patients with HCV-related decompensated LC, only NS5A inhibitors can be used[49]. Many studies have shown the efficacy of DAAs in achieving recompensation of HCV-related decompensated LC (Table 2)[50-66]. For example, in a systematic review and meta-analysis of 49 studies involving a total of 7,886 patients with HCV-related decompensated LC of genotypes 1, 3, and 4, after DAAs treatment for 12-96 weeks, the SVR rate was 86% (95%CI: 0.83-0.88). Improvement in liver function was observed in 51% (95%CI: 0.44-0.58) of patients, and 16% (95%CI: 0.05-0.40) were delisting from liver transplantation[67].

El-Sherif *et al*[68] performed a retrospective analysis of data from four RCTs (SOLAR-1, SOLAR-2, ASTRAL-4, and GS-US-334-0125) examining the efficacy of sofosbuvir-based therapy in patients with HCV-related decompensated LC (502-CTP class B and 120-CTP class C) to determine factors associated with recompensation, namely, a reduction of CTP score to class A. In these trials, patients were given 12 or 24 weeks of treatment with ledipasvir, sofosbuvir, and ribavirin or velpatasvir, sofosbuvir, and/or ribavirin, or 48 weeks of treatment with sofosbuvir and ribavirin. It turned out that the presence of ascites or encephalopathy, serum albumin levels < 3.5 g/dL or ALT < 60 U/L, and body mass index (BMI) > 25 kg/m^2 were associated with an increased risk of not achieving a reduction in CTP to class A, independent of SVR to therapy. The serum albumin levels < 2.8 g/dL and abnormal serum total bilirubin levels were associated with an increased likelihood of liver transplantation or death. The authors developed a BE3A score based on five baseline factors (BMI, encephalopathy, ascites, and serum levels of ALT and albumin) associated significantly with patient outcomes. For patients with scores of 4-5, the HR for reduction of CTP score to class A was 52.3 (95%CI: 15.2-179.7).

Table 1 Efficacy of antiviral therapy in achieving recompensation of hepatitis B virus-related decompensated liver cirrhosis

Ref.	Research design, number of patients	CTP score, points	MELD score points	Antiviral therapy	Treatment period, SVR	Main results[1]
Nikolaidis *et al* [33]	Prospective, single-center, *N* = 20	B/C, 9.3 ± 2.0	16.2 ± 3.1	LAM	12-24-36 months, 55.0%	In 55% of patients, the CTP score decreased by more than 2 points, and in 45% of patients they reached CTP class A
Manolakopoulos *et al*[34]	Prospective, single-center, *N* = 19	A/B/C, 6 (5–12)	12 (7–26)	LAM	12 months, 78.9%	In 10 out of 13 patients with clinically significant portal hypertension and those who achieved SVR, there was a reduction in hepatic venous pressure gradient to less than 12 mmHg or 20% of the baseline
Shim *et al*[35]	Prospective, single-center, *N* = 55	B/C, 8.1 ± 1.7	11.5 ± 3.9	ETV	12 months, 89.1%	The CTP and MELD scores have improved. In 49% of patients, the CTP score values decreased by more than 2 points, and in 65.5% of patients they reached CTP class A
Liaw *et al*[36]	Randomized, open-label, comparative, *N* = 100, *N* = 91	B/C, ≥ 7, B/C, ≥ 7	17.1 (SE = 0.50), 15.3 (SE = 0.48)	ETV, ADV	12 months, 57.0%, 12 months, 20.0%	In 2/3 of the patients in both groups, the CTP score improved. The MELD score decreased by 2.6 points when treated with entecavir, and by 1.7 points when treated with adefovir
Jang *et al*[37]	Prospective, multicenter, *N* = 423	A/B/C, 8.7 ± 2.0	13.9 ± 6.4	LAM/ETV/ADV/clevudine/LdT	12 months, 57.9%	In 2/3 of the patients in both groups, the CTP score improved. The MELD score decreased by 2.6 points when treated with entecavir, and by 1.7 points when treated with adefovir
Lee *et al*[38]	Retrospective, multicenter, *N* = 57	B/C, 8.0 ± 1.5	13.4 ± 4.7	TDF	12 months, 70.2%	In 14.7% of patients, the initial values of the CTP score ≥ 7 decreased by more than 2 points, and in 12% of patients they reached CTP class A. Within 12 months of starting treatment, 33.9% of liver transplant candidates were excluded from the waiting list
Wang *et al*[39]	Prospective, multicenter, *N* = 283	B/C, 8.3 ± 1.9	13.4 ± 4.4	ETV	120 weeks, 92.2%	The CTP and MELD scores have improved. In 49.1% of patients, the initial values of the CTP score ≥ 7 decreased by more than 2 points, and in 68.4% of patients they reached CTP class A
Hui *et al*[40]	Retrospective, cohort, *N* = 1109	B, 6, 7	7.3 ± 4.5	ETV/TAF	12 months, N/A	For at least 12 months, 60.4% of patients had no ascites (off diuretics), encephalopathy (off lactulose/rifaximin) and recurrent gastroesophageal variceal bleeding. The serum albumin levels increased from 31.7 ± 6.4 to 42.4 ± 6.2, international normalized ratio values, serum levels of total

						bilirubin, ALT and aspartate aminotransferase decreased. The CTP and MELD scores improved: 5.77 ± 1.37 *vs* 8.33 ± 1.90 and 10.45 ± 4.58 *vs* 13.37 ± 4.44, respectively
Zhang *et al*[41]	Retrospective, two-center, cohort, *N* = 71	B/C, 8.5 ± 1.6	13.3 ± 4.3	ETV/TDF/TAF	12 months, N/A	Patients with decompensated LC who achieved recompensation showed a similar 5-year survival rate with those with compensated LC (76% and 89.3%, respectively)
Li *et al*[42]	Retrospective, cohort, *N* = 196	A/B/C	11.0 (8.0-15.0)	ETV/LAM + ADV/LdT + ADV/TDF/TAF	12 months, 78.1%	The cumulative incidence of hepatocellular carcinoma after 2 years, 4 years, and 6 years in patients with decompensated LC who achieved recompensation was the same as in those with compensated LC, which was 1.2%, 5.2%, 24.5%, and 1.3%, 5.4%, 20.0%, respectively. The rate of ascites regression was higher in SVR cohort when compared with that in non-SVR cohort. The serum ALT levels and load of serum hepatitis B virus DNA at baseline were predictors of ascites regression

[1]The research results are statistically significant.

ADV: Adefovir dipivoxil; ALT: Alanine aminotransferase; CTP: Child-Turcotte-Pugh; ETV: Entecavir; LAM: Lamivudine; LC: Liver cirrhosis; LdT: Tebivudine; MELD: Model for end stage liver disease; N/A: Not available; SE: Standard error; SVR: Sustained virological response; TAF: Tenofovir alafenamide fumarate; TDF: Tenofovir disoproxil fumarate.

Table 2 Efficacy of antiviral therapy in achieving recompensation of hepatitis C virus-related decompensated liver cirrhosis

Ref.	Research design, number of patients	CTP score, points	MELD score points	Hepatitis C virus genotype	Antiviral therapy	Treatment period, SVR	Main results[1]
Belli *et al* [50]	Retrospective, multicenter, *N* = 103	B/C	16 (6-31)	1a, 1b, 2, 3, 4	SOF, SOF/LED, SOF/DAC, SOF/SIM ± RBV	12 weeks, 98%	The median CTP score decreased from 10.0 to 8.0, and the median MELD score decreased from 15.5 to 14.0. The median serum albumin levels increased by 0.5 g/dL, the median serum total bilirubin levels decreased by 0.9 mg/dL, and the median international normalized ratio values reduced by 0.13 points. Of the entire cohort of 103 patients, 33% of liver transplant candidates were excluded from the waiting list due to clinical improvement
Foster *et al* [51]	Prospective, multicenter, *N* = 409	B/C, ≥ 7	12 (7-32)	1, 3	SOF/LED, SOF/DAC ± RBV	12 weeks, 81.6%	The MELD score decreased by an average of 0.85 points. Patients with baseline serum albumin levels < 35 g/L, sodium < 135 mmol/L, and over 65 years of age were least

							likely to benefit from therapy
Mandorfer *et al*[52]	Retrospective, single-center, *N* = 41	A/B	8 (7-9)	1, 2, 3, 4	SOF/DAC, SOF/LED, SOF/SIM ± RBV	12/24 weeks, 63%	The HVPG reduced by more than 10% of the baseline. The probability of HVPG reduction in CTP class B patients was lower compared with CTP class A patients
Perricone *et al*[53]	Prospective, cohort, *N* = 142	A/B/C	16 (13-18)	1a, 1b, 2, 3, 4	SOF, SOF/LED, SOF/DAC, SOF/SIM ± RBV	12 weeks, N/A	The CTP and MELD scores have improved. In 79.5% of patients, ascites was completely gone, and in 20.5% of patients it required low doses of diuretics. The hepatic encephalopathy disappeared. Within 12 weeks of starting treatment, 30.9% of liver transplant candidates were excluded from the waiting list
Macken *et al*[54]	Prospective, cohort, *N* = 39	N/A	6 (6-7)	1, 3	OMB/PAR/DAS, SOF/LED, SOF/DAC ± RBV, SOF/pegylated interferon alpha-2a/RBV	12 weeks, 77%	The recompensation was recorded in 51% of patients. The associated criterion was a lower baseline serum creatinine levels
Hanafy *et al* [55]	Interventional, *N* = 160	B/C, 11.2 ± 1.2	20.6 ± 2.04	4	SOF/DAC/RBV	12/24 weeks, 90%	There were improvements in platelet count, serum albumin levels, CTP and MELD scores, a significant reduction in the frequency of hepatic encephalopathy. Hepatocellular carcinoma developed in 10% of patients within 6.8 months ± 2.5 months after DAAs, survival was higher in the treated *vs* the control group
Moon *et al* [56]	Prospective, cohort, *N* = 9399	N/A	> 9 (70%)	1 (approximately 60%)	PAR/RIT/OMB/DAS, SOF ± DAC, SOF + SIM	12 weeks, 84.3%	On average, 5.1% of patients (1.66 cases per 100 patient-years) developed GEVB over a follow-up of 3.1 years. This complication was less common in patients who achieved SVR (1.55 cases per 100 patient-years) than without it (2.96 cases per 100 patient-years)
Puigvehí *et al*[57]	Prospective, multicenter, *N* = 247	A	6 (6–14)	N/A	SOF/SIM, SOF/DAC, SOF/LED ± RBV, PAR + RIT/OMB/OMB	12 weeks, 93.1%	Over a follow-up of 3 years, GEV developed in 12.5% of patients who had not had it before and increased in 33.1% of patients with low-risk GEV (< 5 mm)
Liu *et al*[58]	Prospective, multicenter, *N* = 107	B/C	10 (7–13)	1, 1a, 1b, 2, 3, 6	SOF/VEL + RBV	12 weeks, 89.7%	The CTP and MELD scores have improved in 84.4% and 64.6% of patients, respectively. The initial values of the MELD score ≥ 15 points decreased by more than 3 points
Tada *et al* [59]	Retrospective, multicenter, *N* = 65	B/C, ≥ 7	N/A	1, 2	SOF/VEL	12 weeks, 92.3%	The albumin–bilirubin score have improved during and after treatment
Tahata *et al* [60]	Prospective, multicenter, *N* = 82	A/B/C	N/A	1, 2, 3, 4	SOF/VEL	12 weeks, 90.2%	In 50% of CTP class B patients, the CTP score decreased to class A, in

							27% of CTP class C patients, the CTP score decreased to class B, and in 9% of CTP class C patients, the CTP score decreased to class A. The serum albumin level increased when its initial value exceeded 28 g/L
Takaoka *et al*[61]	Prospective, multicenter, *N* = 72	B/C	9 (7-11)	1, 2	SOF/VEL	12 weeks, 95.8%	In 75% of patients who achieved SVR, there was a decrease in CTP score, and in 5.9% of patients they increased. The serum albumin levels and prothrombin time values increased, ascites decreased, while serum total bilirubin levels and the severity of hepatic encephalopathy did not change significantly
Meunier *et al*[62]	Retrospective, multicenter, *N* = 75	A/B/C	14 (11-18)	1	SOF/DAC	24 weeks, 92%	Five years after treatment, 25.3% of liver transplant candidates were excluded from the waiting list due to clinical improvement. The predictors of this were the absence of ascites, the MELD score ≤ 15 points and the CTP score ≤ 7 points
Su *et al*[63]	Retrospective, single-center, *N* = 50	B/C	12 (6–21)	1, 2, 6	SOF/DAC, SOF/LED, SOF/VEL ± RBV	12 weeks, 96%	The values of the following scores decreased: Fibrosis-4 (8.1 ± 4.0 *vs* 11.2 ± 6.9), CTP (6.8 ± 1.4 *vs* 8.0 ± 1.2), and MELD (11.6 ± 3.0 *vs* 12.7 ± 3.6)
Kotani *et al* [64]	Observational, *N* = 50	B/C, 8 (7–9)	10 (9–13)	1b, 2a, 2b	SOF/VEL	24 weeks, 89%	In 42% of patients who achieved SVR, the HVPG reduced by more than 20% of the baseline, and the percentage of patients with HVPG > 12 mmHg decreased from 92% to 58%. At the same time, clinically significant PH persisted in 75% of patients
Premkumar *et al*[65]	Prospective, cohort, *N* = 1152	A/B/C, 12.7 ± 1.6	16.6 (16.5 ± 4.6)	1, 2, 3 (87.1%), 4, 5, 6	SOF/DAC, SOF/VEL	12 weeks, 81.8%	The SVR resulted in recompensation in 24.7% of patients over a follow-up of 4 years. The ascites resolved in 86% of patients (diuretic withdrawal achieved in 24% of patients). Despite SVR, new hepatic decompensation evolved in 19% of patients. PH progressed in 13.7% of patients, with the development of recurrence GEVB in 4%. The hepato-cellular carcinoma developed in 2.9% of patients
Yuri *et al* [66]	Retrospective, single-center, *N* = 109	A/B/C	N/A	N/A	N/A (DAAs)	24 weeks, 34,9%	At 7 years, the cumulative GEV progression rate in the DAA-SVR group was significantly lower than that in the non-SVR group. GEVB occurred in 11.3% of patients in the non-SVR group, while no GEVB events were observed in the DAA-SVR group

	during the observational period

[1]The research results are statistically significant.

CTP: Child-Turcotte-Pugh; DAAs: Direct acting antivirals; DAC: Daclatasvir; DAS: Dasabuvir; GEV: Gastroesophageal varices; GEVB: Gastroesophageal variceal bleeding; HVPG: Hepatic venous pressure gradient; LED: Ledipasvir; N/A: Not available; MELD: Model for end stage liver disease; OMB: Ombitasvir; PAR: Paritaprevir; PH: Portal hypertension; RBV: Ribavirin; RIT: Ritonavir; SIM: Simeprevir; SOF: Sofosbuvir; SVR: Sustained virological response; VEL: Velpatasvir.

CONCLUSION

Numerous publications in recent years have shown the efficacy of etiological therapy in achieving recompensation of alcohol-related, as well as HBV-related and HCV-related decompensated LC according to Baveno VII criteria. So far, only the first steps have been taken in studying this problem. To further understand it, research is needed to identify pathophysiological mechanisms, modifying factors, predictors, and potential noninvasive biomarkers of recompensation of decompensated LC.

FOOTNOTES

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