


Hepatitis C Treatment Outcomes in Persons With HIV and Decompensated Cirrhosis Using a Collaborative Multidisciplinary HIV-Centered Approach

Journal of the International Association of Providers of AIDS Care
Volume 20: 1-6
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DOI: 10.1177/23259582211024771
journals.sagepub.com/home/jia


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Abstract

Fifty-four consecutive persons with HIV co-infected with hepatitis C virus (HCV) and liver decompensation were treated with direct-acting antivirals (DAA). The HCV treatment was delivered using a multidisciplinary HIV-coinfection model of care integrating sub-specialty services in 3 countries. Of those treated, 91% (95% confidence interval, 80.1 to 95.9) achieved sustained viral response, and only one person died during treatment. Our study provides evidence that HIV providers achieve excellent outcomes when treating patients with histories of decompensated liver disease, with characteristics similar to those studied using a multidisciplinary HIV-centered approach.

Keywords

HIV, HCV, liver decompensation, DAA, multidisciplinary

Date received: 9 February 2021; revised: 18 May 2021; accepted: 24 May 2021.

Background

With the advent of Direct-acting antivirals (DAA), HIV medical providers routinely treat hepatitis C (HCV) in persons with HIV (PWH) who do not have cirrhosis or have cirrhosis without decompensation.¹

Worldwide, liver transplantation is not an option for most PWH in need. Yet, many PWH co-infected with HCV and decompensated cirrhosis remain untreated due to health-system and personal barriers to access sub-specialty services.² The delay in HCV treatment often leads to frequent hospitalizations, increases resource health care utilization, and death.³

HIV care is increasingly delivered within integrated HIV-primary care multidisciplinary care models, including but not limited to HIV medical providers, clinical pharmacists, social workers, substance counselors, and psychiatrists.⁴ These models allow the real-time interaction of HIV physicians with sub-specialty services and a tandem approach to managing complex PWH, such as those with liver decompensation. HIV physicians implement the

medical hepatology recommendations while managing multiple concurrent medical comorbidities. The HIV team addresses potential medical interactions while also supporting services to overcome PWH competing barriers to care,

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What Do We Already Know About This Topic?

Treatment of hepatitis C using direct-acting antivirals (DAA) in persons with HIV (PWH) is well-tolerated and highly effective. Yet access to hepatitis C treatment sub-specialty services, especially among PWH with liver decompensation, remains suboptimal in many parts of the world, and this is detrimental to their quality of life and survival.

How Does Your Research Contribute to the Field?

This work provides evidence that the specialty care that PWH with decompensated cirrhosis needed during DAA treatment can be accomplished in a multidisciplinary HIV-centered model of care lead by trained HIV providers.

What Are Your Research's Implications Toward Theory, Practice, or Policy?

To accomplish the goal of HCV eradication, the pool of providers treating HCV needs to be expanded, and systems that facilitate HCV treatment of PWH with advanced liver disease need to be augmented.

such as drug use, unstable housing, and mental illness.⁵ Herein, we report the sustained viral response (SVR) and safety outcomes when HIV teams treated HCV in PWH with liver decompensation, integrating sub-specialty services in 3 different countries.

Methods

We conducted a retrospective cohort analysis of consecutive PWH co-infected with HCV who had decompensated cirrhosis and were treated with DAA in the HCV-HIV Transatlantic Research Network (HCV-TREN). The cohort comprises 5 academically affiliated HIV clinics in 3 different countries. Using de-identified electronic medical records (EMR) information, we collect data on demographics; HIV regimen, CD4 counts, and viral load; HCV-genotype, prior HCV treatment history, DAA regimen, Child-Turcotte-Pugh (CPT) class, Model for End-Stage Liver Disease (MELD) scores; and Charlson comorbidity index. We also collected patient-reported outcomes relative to current alcohol, illicit drug use, unstable housing, and active psychiatric illness as previously described.⁶ Each participating site obtained approval from their designated research Ethics committees: The University of California at San Diego Human Research Protection Program (approval no. 171749X), the Research Ethics Committee of the University of Sassari, Italy (approval no. 2424/CE), the Ethics Committee of Health Department and Social Services and Equity of Spain (approval no. CAC-HCV-2016-01). For this observational retrospective

study, all ethics committees waived the requirement for written informed consent as it was a retrospective review of existing medical records.

We included PWH who were 18 years of age or older with a detectable HCV viral load and a Child-Turcotte-Pugh (CPT) score B or higher treated for HCV between January 2014 and July 2018. In each clinic, there was an HIV clinician experienced in HCV treatment who led team efforts. We followed 4 principles in the management of HCV among PWH: (1) evaluation of HIV control and medical interactions; (2) liver fibrosis staging and prevention of cirrhosis-related complications; (3) addressing concurrent medical comorbidities; and (4) management of ongoing barriers to care.⁶

HIV providers used electronic messaging to coordinate all aspects of cirrhosis staging before DAA initiation. The hepatology team completed the required esophagogastroduodenoscopy (EGD) for esophageal varices screening. PWH also completed abdominal ultrasound examination to rule out hepatocellular carcinoma (HCC). In coordination with hepatology teams, HIV teams followed standard of care protocols to manage portal hypertension, hepatic encephalopathy, and spontaneous bacterial peritonitis prophylaxis. HIV pharmacists reviewed the safety of combining different DAA with antiretrovirals and collaborated with HIV providers on any needed ART changes to allow HCV treatment. Depending on each site's resources, HIV clinical pharmacists, social workers, substance counselors, and psychiatrists provided counseling on avoiding liver-related hepatotoxic medications and alcohol while offering harm reduction counseling to prevent HCV reinfection.

The primary study endpoint was the proportion of patients who achieved SVR defined as having an undetectable serum HCV RNA 12 weeks after the estimated DAA treatment completion date. The secondary endpoint was the proportion of patients who died after receiving at least one dose of DAA at the estimated date of SVR.

Descriptive statistics were presented using medians with ranges and frequencies with percentages. Bivariate analyses with pairwise comparisons were conducted to investigate factors associated with lack of HCV SVR. We used χ^2 test for comparison of categorical variables and the Wilcoxon rank-sum test for numerical variables. Analyses were performed using Stata Statistical Software (Release 14.2. College Station, TX).

Results

During the study period, 54 PWH with decompensated cirrhosis were treated for HCV using different DAA regimens. Of them, 18 (33%) had failed prior HCV treatments, and 41 (76%) had HCV genotype 1. Treated patients had a median age of 52 years and a CD4 cell count of 312/mm³, with most (90%) having an undetectable HIV viral load. Forty-six patients (85%) were males, and 35 of them had had as their main HIV

Table 1. Characteristics of PWH and liver decompensation co-infected with hepatitis C treated with DAA.

	TOTAL N = 54	SVR: YES N = 49	SVR: NO N = 5
Age: median (range), y	52.5 (41–65)	53 (41–65)	53 (47–56)
Male gender: No. (%)	46 (85.2)	41 (83.4)	5 (100.0)
HCV genotype, No. (%)			
1/1a/1b	41 (75.9)	37 (75.4)	4 (80.0)
2	1 (1.9)	1 (2.0)	0 (0.0)
3	6 (11.1)	6 (12.5)	0 (0.0)
4	6 (11.1)	5 (10.1)	1 (20.0)
Prior HCV treatment failure: No. (%)	18 (33.3)	16 (32.7)	2 (40)
HCV viral load: median (range), Log ₁₀ IU/L	5.95 (4.20–7.22)	5.97 (4.28–7.22)	5.84 (4.20–6.63)
Child-Turcotte-Pugh score, No. (%)			
B7	34 (63.0)	32 (65.3)	2 (40.0)
B8	8 (14.8)	7 (14.3)	1 (20.0)
B9	6 (11.1)	5 (10.2)	1 (20.0)
C10/11	6 (11.1)	5 (10.2)	1 (20.0)
MELD score: median (range)	11 (8–20)	11 (8–19)	14 (10–20)
Decompensation events per patient, No. (%)		**	**
Ascites ± leg edema	26 (48.2)	25 (51.0)	1 (20.0)
Only bleeding esophageal varices (EV)	2 (3.7)	2 (4.1)	0 (0.0)
Only hepatic encephalopathy (HE)	4 (7.4)	4 (8.2)	0 (0.0)
Ascites and spontaneous bacterial peritonitis (SBP)	5 (9.3)	5 (10.2)	0 (0.0)
Ascites and bleeding EV	3 (5.6)	3 (6.1)	0 (0.0)
Ascites and HE	5 (9.3)	4 (8.2)	1 (20.0)
Ascites, HE, and bleeding EV	7 (12.9)	6 (12.2)	1 (20.0)
Ascites, SBP, and HE	1 (1.8)	0 (0.0)	1 (20.0)
Ascites, SBP, HE, and bleeding EV	1 (1.8)	0 (0.0)	1 (20.0)
HIV risk factor, No. (%)			
Men who have sex with men (MSM)	2 (3.7)	1 (2.0)	1 (20.0)
Heterosexual	12 (22.2)	12 (24.5)	0 (0.0)
Hemophilia	1 (1.9)	1 (2.0)	0 (0.0)
MSM and intravenous drug use	2 (3.7)	2 (4.1)	0 (0.0)
Heterosexual and intravenous drug use	35 (64.8)	32 (65.4)	3 (60.0)
other	2 (3.7)	1 (2.0)	1 (20.0)
CD4+ count: median (range), cells/mm ³	312.5 (75–1279)	319 (75–1279)	417 (256–682)
Detectable HIV viral load (≥20 copies/ml): No. (%)	5 (9.3)	5 (10.2)	0 (0.0)
HIV viral load: median (range)	0 (0–19,000)	9 (0–19,000)	0 (0)
Charlson comorbidity score, median (range)	7 (2–12)	6 (2–12)*	10 (7–12)*
Current hazardous alcohol use: No. (%)	14 (25.9)	14 (28.6)	0 (0.0)
Current illegal drugs: No. (%)	11 (20.4)	8 (16.3)*	3 (60)*
Current unstable housing: No. (%)	4 (7.4)	4 (8.2)	0 (0.0)
Active psychiatric illness: No. (%)	15 (27.8)	14 (28.6)	1 (5)
Direct Acting Antiviral regimen, No. (%)		**	**
Sofosbuvir plus simeprevir– 12weeks	7 (12.9)	4 (8.2)	3 (60.0)
Sofosbuvir plus simeprevir and ribavirin– 12weeks	7 (12.9)	7 (14.2)	0 (0.0)
Sofosbuvir plus ledipasvir– 24weeks	13 (24.2)	12 (24.5)	1 (20.0)
Sofosbuvir plus ledipasvir and ribavirin – 12weeks	10 (18.5)	10 (20.4)	0 (0.0)
Sofosbuvir plus ledipasvir and ribavirin – 24weeks	1 (1.8)	0 (0.0)	1 (20.0)
Sofosbuvir plus daclatasvir – 24weeks	5 (9.3)	5 (10.2)	0 (0.0)
Sofosbuvir plus daclatasvir and ribavirin – 24weeks	4 (7.4)	4 (8.2)	0 (0.0)
Sofosbuvir plus velpatasvir and ribavirin – 12weeks	5 (9.3)	5 (10.2)	0 (0.0)
Sofosbuvir plus velpatasvir – 24weeks	2 (3.7)	2 (4.1)	0 (0.0)
Antiretroviral therapy: No. (%)			
abacavir/lamivudine & dolutegravir	11 (20.4)	9 (18.5)	2 (40.0)
abacavir/lamivudine & raltegravir	3 (5.5)	3 (6.1)	0 (0.0)
abacavir/lamivudine, dolutegravir & rilpivirine	3 (5.5)	2 (4.1)	1 (20.0)
abacavir/lamivudine & ritonavir-boosted darunavir	3 (5.5)	3 (6.1)	0 (0.0)
tenofovir/emtricitabine & raltegravir	10 (18.5)	9 (18.5)	1 (20.0)
tenofovir/emtricitabine & dolutegravir	8 (14.8)	8 (16.3)	0 (0.0)

(continued)

Table 1. (continued)

	TOTAL N = 54	SVR: YES N = 49	SVR: NO N = 5
tenofovir/emtricitabine & rilpivirine	2 (3.7)	2 (4.1)	0 (0.0)
tenofovir/emtricitabine & ritonavir-boosted darunavir	6 (11.0)	6 (12.2)	0 (0.0)
tenofovir/emtricitabine, ritonavir-boosted darunavir & etravirine	2 (3.7)	2 (4.1)	0 (0.0)
tenofovir/emtricitabine, rilpivirine & maraviroc	1 (1.9)	1 (2.0)	0 (0.0)
tenofovir/emtricitabine, rilpivirine & dolutegravir	1 (1.9)	1 (2.0)	0 (0.0)
rilpivirine, ritonavir-boosted darunavir & raltegravir	1 (1.9)	1 (2.0)	1 (20.0)
rilpivirine & ritonavir-boosted darunavir	1 (1.9)	1 (2.0)	0 (0.0)
rilpivirine, maraviroc, & raltegravir	1 (1.9)	1 (2.0)	0 (0.0)
tenofovir/emtricitabine, ritonavir-boosted darunavir, dolutegravir & maraviroc	1 (1.9)	0 (2.0)	1 (20.0)

SVR: sustained viral response, MELD: Model for End-Stage Liver Disease.

Bivariate comparison between the groups based on SVR status: * $p < 0.05$, ** $p < 0.01$.

Table 2. Clinical characteristics of the 5 patients who failed hepatitis C treatment.

	Relapse #1	Relapse #2	Relapse #3	Died	Lost to follow-up
Age in years	54	56	50	47	51
Gender	male	male	male	male	male
HCV genotype	1a	1a	4	1a	1a
Prior HCV treatment failure	No	Yes	Yes	No	No
HCV viral load: Log IU/L	4296194	3088341	697000	50600	15888
Child-Turcotte-Pugh score	C10	B7	B7	B8	B9
MELD score	20	14	15	10	13
Decompensation events	Ascites, HE, bleeding EV	Ascites	Ascites, HE	Ascites, SBP, HE	Ascites, SBP, HE, bleeding EV
HIV risk factor	Hemophilia	Hetero & IDU	Hetero & IDU	Hetero & IDU	Hetero
CD4+ count, cells/mm ³	251	230	499	387	256
HIV viral load	UD	UD	UD	UD	UD
Charlson comorbidity score	12	12	7	8	10
Current hazardous alcohol use	No	No	No	No	No
Current illegal drugs	Yes	Yes	No	Yes	No
Current unstable housing	No	No	No	No	No
Active psychiatric illness	No	No	Yes	No	No
Direct Acting Antiviral regimen	sof/smv for 12 weeks	sof/smv for 12 weeks	sof/l dv + rbv for 24 weeks	sof/l dv for 24 weeks	Sof/smv for 12 weeks
Antiretroviral therapy	abc/3tc+dtg	abc/3tc+rpv+dtg	tdf/ftc+etv+dtg+ mvc+ drv/r	abc/3tc+dtg	tdf/ftc+ral

HE: hepatic encephalopathy, EV: esophageal varices, IDU: intravenous drug use, hetero: heterosexual, UD: undetectable

sof: sofosbuvir, smv: simeprevir, ldv: ledipasvir, rbv: ribavirin.

abc: abacavir, 3tc: lamivudine, tdf: tenofovir disoproxil, ftc: emtricitabine, rpv: rilpivirine, etv: etravirine, mvc: maraviroc, dtg: dolutegravir, ral: raltegravir, drv/r: ritonavir-boosted darunavir, sbp: spontaneous bacterial peritonitis.

risk factor being heterosexual with intravenous drug use history. We were required to change ART in 22 PWH (41%) to allow DAA initiation. All of them had complex HIV resistance history. The prevalence of current alcohol, drug use, and psychiatric illness was 26%, 20%, and 28%, respectively. At the time of HCV treatment initiation, and according to CPT score distribution, 63% of patients had class B7, 15% B8, 11% B9, 5% C10, and 4% C11, with the median baseline MELD score was 11 (range, 8 to 20). Twenty-two patients (41%) had at least 2 decompensated cirrhosis manifestations. Seven (13%) had ascites plus esophageal varices, and hepatic encephalopathy (Table 1).

A total of 49 patients (91%, 95% confidence interval [CI], 80.1 to 95.9) achieved SVR: 94% (95% CI: 80.1 to 98.4) among those with CPT B7, 88% (95% CI: 52.9 to 97.8) among those with CPT B8, 83% (95% CI: 43.7 to 96.9) among those with CPT B9, and 83% (36.5 to 99.1) among those with CPT C10/11. The mortality proportion during HCV treatment was 1.9% (95% CI: 0.3 to 9.7). Of the 5 patients who failed HCV treatment, 3 relapsed, 1 was lost to follow up, and one died (Table 2). The death was attributed to the patient's noncompliance with his secondary antibiotic prophylaxis to prevent recurrent spontaneous bacterial peritonitis. Unlike those who achieved SVR, PWH who failed DAA treatment had more

frequent current drug use, multiple comorbidities, and signs of liver decompensation (Table 1). After a median of 2.1 years of follow-up, all PWH who achieved initial SVR remain alive.

Discussion

In this international cohort of PWH with decompensated cirrhosis treated with DAAs, we observed high HCV cure rates (91% overall) when following treatment models led by HIV physicians using a multidisciplinary care model adjusted to regional resource availability. HCV treatment success was possible despite the high prevalence of ongoing barriers to care in our cohort and the presence of complex advanced liver disease manifestations.

The role of hepatology is vital for the management of persons with decompensated cirrhosis. For example, hepatologists decide the timing of DAA treatment relative to the patient's candidacy for liver transplantation. Unfortunately, a liver transplant is not a viable option for most PWH in need, and many with advanced liver disease fail to link to HCV sub-specialty care.⁷ Challenges to this conventional approach include the need for a sub-specialty referral and insurance approval in non-universal healthcare systems and long waiting times for a hepatology intake appointment.⁸ The combination of PWH with ongoing barriers to care, internalized stigma, and low HCV knowledge also accentuate the lack of HCV linkage to care of some PWH with advanced liver disease to hepatology services.⁹

Our success in developing effective cures for HCV requires us to find ways to treat PWH who have traditionally been challenging to treat. To accomplish the goal of HCV eradication, the pool of providers treating HCV needs to be expanded, and systems that facilitate HCV treatment of patients with advanced liver disease need to be created.⁵ Our results suggest that the special care that PWH with decompensated cirrhosis needed during DAA treatment can be accomplished in a collaborative, multidisciplinary HIV-centered model of care. Collaboration between hepatology and HIV teams is essential to create concurrent strategies to address the medical, personal, and social needs of PWH to facilitate their access to HCV treatment. Indeed, our study SVR rates and safety outcomes were comparable to those of registrational clinical trials of PWH and decompensated cirrhosis and other hepatology-based studies.¹⁰ Noteworthy, successful HCV treatment in PWH with liver decompensation does not eliminate the risk of HCC or liver disease progression.¹¹ Thus, PWH with liver decompensation require ongoing follow-up with hepatology services after SVR. Finally, HIV physicians who treat HCV need to be aware that HCV protease inhibitors should not be given to patients with decompensated cirrhosis because of the potential for worsening hepatic decompensation.¹²

Our study has limitations. Our sample size was small (N = 54) and thus may not be generalizable. Notwithstanding, our goal is to report a successful approach to treat HCV in PWH with liver decompensation who are also burdened by complex social and medical needs commonly encountered in clinical practice. Our models rely on collaboration with

sub-specialty services while maintaining HIV primary care coordination. We recognize that there are multiple strategies to treat HCV in PWH with advanced liver disease. When providers participate in a weekly telehealth-based didactic and clinical case discussion using the ECHO model, successful HCV therapy can be accomplished in patients with advanced liver disease.¹³ Regardless of the implemented model tailored to geographic and structural health system needs, we believe that to close the gaps on HCV elimination in our communities, we need to foster inclusion and collaboration among multiple providers caring for PWH with decompensated cirrhosis.

In conclusion, HCV cure was achieved in 91% of PWH with liver decompensation in 3 different countries using a multidisciplinary HIV-centered treatment approach.

Acknowledgments

We want to thank our patients, whose resilience and perseverance continue to inspire us to improve our clinical services.


Declaration of Conflicting Interests


The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.


Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

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