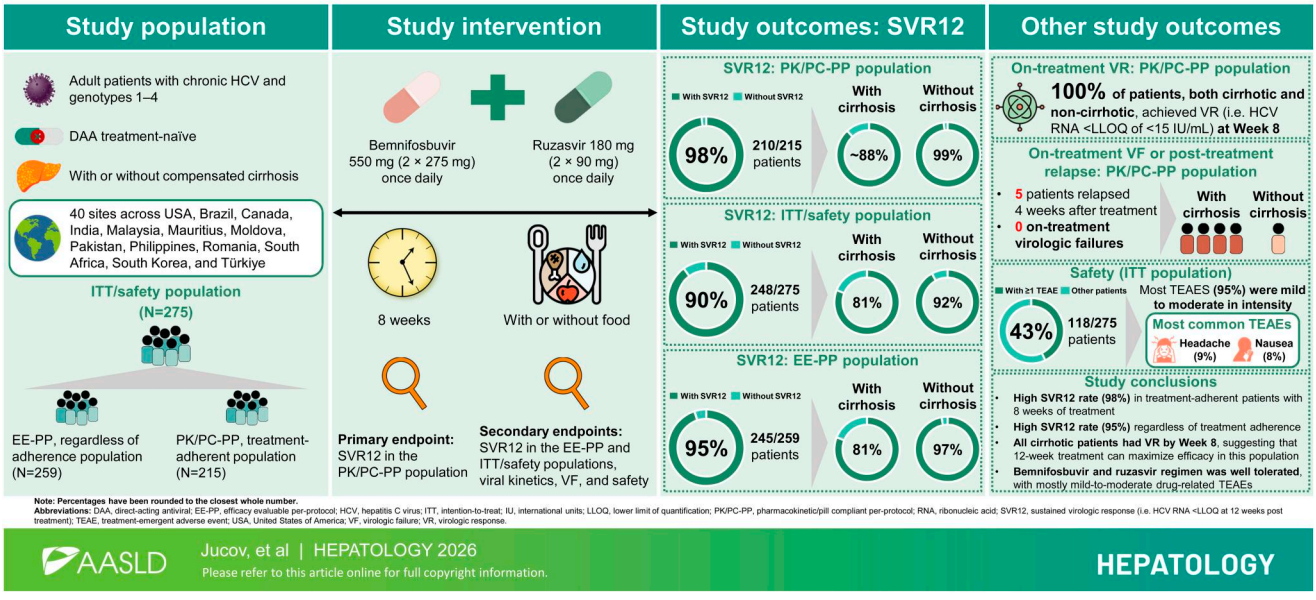
Efficacy and safety of an 8-week bemnifosbuvir and ruzasvir regimen in chronic hepatitis C: Results from a Phase 2 study

VISUAL ABSTRACT



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Efficacy and safety of an 8-week bemnifosbuvir and ruzasvir regimen in chronic hepatitis C: Results from a Phase 2 study

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Abstract

Background and Aims: Bemnifosbuvir and ruzasvir are potent pan-genotypic inhibitors of the hepatitis C virus (HCV) NS5B polymerase and NS5A protein, respectively. This single-arm, Phase 2 study evaluated the efficacy and safety of bemnifosbuvir and ruzasvir in treatment-naïve patients with chronic HCV infection of any genotype, with or without compensated cirrhosis.

Approach and Results: Patients received bemnifosbuvir 550 mg and ruzasvir 180 mg once daily for 8 weeks (intention-to-treat [ITT] population. The primary efficacy endpoint was the proportion of patients achieving a sustained virologic response at 12 weeks post-treatment (SVR12) in the Pharmacokinetic and Pill-Compliant Per-Protocol (PK/PC-PP) population. Secondary efficacy endpoints included SVR12 in the Efficacy Evaluable Per-Protocol (EE-PP; those with outcomes regardless of study drug adherence) and safety (all dosed) populations, virologic failure, and SVR24. Of the 275 patients enrolled and who received treatment with bemnifosbuvir and ruzasvir, 13% had compensated cirrhosis and 27% had NS5A resistance-associated substitutions (RASs) at baseline. SVR12 was 90% (248/275; 95% CI: 86–93%) in the ITT population, 98% (210/215; 95% CI: 95–99%) in the PK/PC-PP population, and 95% (245/259; 95% CI: 91–97%) in the EE-PP population. Five patients in the PK/PC-PP population experienced virologic failure (post-treatment relapse). No drug-related serious

Abbreviations: AE, adverse event; BCRP, breast cancer resistance protein; CI, confidence interval; CYP3A4, cytochrome P450 3A4; DAA, direct-acting antiviral; eGFR, estimated glomerular filtration rate; EE-PP, efficacy evaluable per-protocol; GT, genotype; HCV, hepatitis C virus; ITT, intention-to-treat; LiPa, line probe assay; LLOQ, lower limit of quantitation; NGS, next-generation sequencing; NS5A, nonstructural protein 5A; NS5B, nonstructural protein 5B; OATP1B1, organic anion transporting polypeptide 1B1; P-gp, P-glycoprotein; PK/PC-PP, pharmacokinetic and pill-compliant per-protocol; RAS, resistance-associated substitution; SAE, serious adverse event; SVR4, sustained virologic response at 4 weeks post-treatment; SVR12, sustained virologic response at 12 weeks post-treatment; TEAE, treatment-emergent adverse event.

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adverse events or premature treatment discontinuations due to drug-related adverse events were reported.

Conclusions: An 8-week treatment with bemnifosbuvir and ruzasvir resulted in high rates of SVR12 across genotypes, regardless of the presence or absence of baseline RASs, and was well tolerated among treatment-naïve HCV patients.

Keywords: bemnifosbuvir, direct-acting antiviral, hepatitis C virus (HCV), ruzasvir

INTRODUCTION

Despite historical success in the treatment of people with chronic hepatitis C virus (HCV) infection, it remains one of the most prevalent blood-borne infectious diseases, with around 1 million new infections and 242,000 associated deaths each year.[1] The World Health Organization supports Global Health Sector Strategies on viral hepatitis, with the goal of eliminating HCV by 2030; however, meeting this target requires diagnosis and successful treatment of the 50 million people still living with HCV infection worldwide.[1] Persistent barriers to achieving elimination goals include limited screening coverage, weak care linkage, and regional inequities, particularly among the key populations: HIV-infected individuals, people who inject drugs, men who have sex with men, and incarcerated individuals.[2] Furthermore, the demographics of the contemporary HCV-infected population have shifted globally, with the appearance of a second peak driven by an increase in cases associated with intravenous drug use and the opioid epidemic.[3–9] Younger individuals (20–40 years of age) now represent the largest population of individuals infected with HCV in the United States of America.[10–13] Globally, the burden of chronic HCV is largely concentrated in countries and regions with low sociodemographic indices and aging populations.[14] Migration can influence the epidemiology of HCV in destination countries by contributing to the introduction and circulation of viral genotypes that may differ from those predominant in the local population, while migrants from high-prevalence regions may have higher HCV prevalence than the host population.[15,16]

The availability and use of combinations of oral direct-acting antiviral (DAA) therapies, with favorable safety and tolerability profiles compared with previous interferon-containing regimens, have dramatically improved outcomes for HCV-infected people able to undergo treatment. Twelve weeks of nucleoside inhibitor-based treatment regimens lead to sustained virologic response (SVR) rates >95% in the majority of patient subgroups in pivotal studies.[17,18] Shorter treatment durations and reduced disruption to daily life are associated with improved patient adherence, which may be crucial for the successful treatment of the remaining population infected with HCV.[19]

Of the currently approved DAA regimens for HCV-infected individuals, glecaprevir/pibrentasvir is the only approved pan-genotypic treatment with an 8-week option.[20–23] However, since glecaprevir is a protease inhibitor, there are drug–drug interaction considerations that affect its applicability in people undergoing complex medication regimens.[23] In addition, it must be taken with food, which may further complicate adherence. While the alternative approved treatment, sofosbuvir/velpatasvir, is a widely used protease-inhibitor-free treatment, it is a 12-week regimen for patients with and without cirrhosis.[24–26] Moreover, the use of sofosbuvir/daclatasvir in the context of large-scale elimination campaigns features a 24-week regimen for patients with cirrhosis or difficult-to-treat genotypes and is associated with difficulties maintaining treatment adherence.[27–29] Thus, a protease-inhibitor-free, pan-genotypic regimen with a convenient 8-week treatment course, allowing for rapid completion of therapy with lower resource utilization, represents a meaningful improvement upon the limitations of currently available DAA treatments for patients with chronic HCV infection.

Bemnifosbuvir and ruzasvir are potent, pan-genotypic inhibitors of the HCV non-structural protein (NS) 5B polymerase and NS5A protein, respectively.[30,31] Bemnifosbuvir is a phosphoramidate protide that is converted after multi-step activation to a pharmacologically active intracellular metabolite that inhibits NS5B RNA-dependent RNA polymerase, causing termination of RNA chain elongation.[32] It has demonstrated ~10-fold greater activity compared with sofosbuvir against laboratory strains and clinical isolates of HCV, including the S282T resistance-associated variants.[32] Ruzasvir inhibits NS5A to block multiple steps in the virus life cycle, at the RNA replication and virus assembly stages, and was developed with the specific aim of achieving a higher barrier to resistance compared with existing NS5A inhibitors.[31]

Guidelines from the European Association for the Study of the Liver and the American Association for the Study of Liver Diseases recommend the assessment of drug–drug interactions before DAA initiation.[33,34] Bemnifosbuvir has been demonstrated as an inhibitor of P-gp and BCRP and a weak inhibitor of CYP3A4, and dose adjustments are unlikely to be required for drugs that are substrates of P-gp, BCRP, OATP1B1, or CYP3A4 when coadministered with bemnifosbuvir.[35,36] Furthermore, previous clinical studies of both bemnifosbuvir and ruzasvir in different combination regimens (bemnifosbuvir with daclatasvir, and ruzasvir with uprifosbuvir), have each demonstrated high sustained virologic response rates in patients with chronic HCV attesting to their efficacy against this virus.[37,38] The combination of bemnifosbuvir and ruzasvir has already demonstrated favorable tolerability, low drug–drug interaction potential, and minimal food effect when administered in healthy volunteers.[39] Therefore, the combination of bemnifosbuvir and ruzasvir has the potential to provide a convenient, protease-sparing, pan-genotypic regimen, with a high barrier to resistance, for HCV-infected patients with or without cirrhosis. With this in mind, we conducted a Phase 2, open-label, single-arm study to evaluate the efficacy and safety of bemnifosbuvir and ruzasvir, administered for 8 weeks in treatment-naïve HCV-infected patients of any genotype, with or without compensated cirrhosis (ClinicalTrials.gov: NCT05904470).

METHODS

Study design and patient population

This Phase 2, open-label, single-arm study was conducted between May 30, 2023, and January 28, 2025, and planned to enroll ~280 patients. Patients were enrolled from 40 sites across 13 countries: the USA, Brazil, Canada, India, Malaysia, Mauritius, Moldova, Pakistan, Philippines, Romania, South Africa, South Korea, and Turkey. DAA treatment-naïve adults aged 18–85 years with chronic HCV infection (HCV RNA levels of at least 1000 IU/mL at screening), either without cirrhosis or with compensated cirrhosis, were eligible. Patients with a Child–Pugh score >6 (class B or C), signs of hepatocellular carcinoma, hepatitis B, or human immunodeficiency virus infection were ineligible. Detailed eligibility criteria are provided in the Supplemental Appendix, https://links.lww.com/HEP/K559. A lead-in cohort of 60 patients without cirrhosis was initially enrolled and evaluated for sustained virologic response at 4 weeks post-treatment (SVR4); full enrollment was then opened after confirming that 6 or fewer compliant patients experienced virologic failure and that no study stopping rules were met.

All patients provided written informed consent before enrollment and before any study procedures were performed. The study protocol was approved by the Institutional Review Board or independent ethics committees at all participating sites before study initiation, and was conducted in accordance with the Declaration of Helsinki, International Conference on Harmonization Guideline E6 (R2) for Good Clinical Practices, and local regulations.

All patients received 550 mg bemnifosbuvir and 180 mg ruzasvir for 8 weeks. Bemnifosbuvir was administered orally once daily as two 275 mg tablets along with ruzasvir, administered orally once daily as two 90 mg capsules, and both drugs were taken with or without food. All patients who received at least one dose of the study drug (bemnifosbuvir or ruzasvir) were included in the ITT population, which is equivalent to the safety population. Patients who met all eligibility criteria, completed treatment (defined as $\geq$90% compliance with the study drug regimen), had outcomes at post-treatment week 12, and had adequate study drug exposure corroborated by pill counts and plasma drug levels adjudicated by an independent clinical pharmacologist, were included in the Pharmacokinetic and Pill-Compliant Per-Protocol (PK/PC-PP) population. A secondary, Efficacy Evaluable Per-Protocol (EE-PP) population included patients who met all eligibility criteria, received at least one dose of study drug, and had outcomes at post-treatment week 12, regardless of drug adherence. Definitions for each analysis population are provided in the Supplemental Appendix, https://links.lww.com/HEP/K559. For those patients who generally complied with the study protocol and experienced a lack of efficacy with the investigational regimen, the Sponsor offered to reimburse the investigator for the cost of a course of post-study medication, depending on local drug access and reimbursement policies.

Study assessments

Plasma HCV RNA was evaluated using the cobas 6800 HCV Quantitative Nucleic Acid Test (Roche, Indianapolis, IN, USA), with a lower limit of quantitation (LLOQ) of 15 IU/mL. Initial HCV genotype was determined using VERSANT HCV Genotyping 2.0 Assay (LiPA) (Siemens, Erlangen, Germany), and nucleotide sequencing of the phylogenetically informative region of NS5B, which is the gold standard for HCV genotyping, was also performed for accurate genotyping and subgenotyping. Next-generation sequencing (NGS) was performed to identify NS5A and NS5B resistance-associated substitutions (RASs) at baseline and at the time of virologic failure using DDL Athena NGS analysis pipeline (DDL Diagnostic Laboratory, Rijswijk, The Netherlands), with a detection limit of 1%. The resulting sequences were compared with genotype-specific

reference sequences to determine the prevalence of RASs and their association with virologic outcomes. For NS5A, RASs were monitored at amino acid positions 24, 28, 30, 31, 32, 38, 58, 92, and 93 (identified in the sofosbuvir/velpatasvir prescribing information).[40] For NS5B, RASs were monitored at amino acid positions 15, 206, 223 (identified in an in vitro bemnifosbuvir HCV resistance study [data on file; Atea Pharmaceuticals, Inc.]) and 142, 159, 237, 282, 289, 314, 321 (identified in the sofosbuvir/velpatasvir prescribing information).[40] RASs which occurred at a frequency of at least 15% were reported.

A single blood sample for pharmacokinetic analysis was collected pre-dose at baseline, and on day 8 (week 1), day 15 (week 2), day 29 (week 4), day 43 (week 6), and day 57 (week 8) of the treatment period. Patients also had the option to participate in an 8-hour intensive pharmacokinetic evaluation on any one of these days after week 1. Samples from all patients who received at least one dose of both study drugs were analyzed, and plasma concentrations of the study drugs and their metabolites (as appropriate) were measured. Safety assessments were performed during treatment and 4 weeks after the end of treatment. All adverse events (AEs) were coded according to the Medical Dictionary for Regulatory Activities.

Study endpoints

The primary efficacy endpoint was the proportion of patients in the PK/PC-PP population achieving sustained virologic response at 12 weeks post-treatment (SVR12), defined as HCV RNA < LLOQ at 12 weeks after treatment. Secondary efficacy endpoints included SVR12 in the EE-PP and safety populations, virologic failure (on-treatment virologic failure or post-treatment relapse), and sustained virologic response at 24 weeks after treatment (SVR24). On-treatment virologic failure was defined as any of the following: not achieving HCV RNA < LLOQ, confirmed 1 $\log_{10}$ increase in HCV RNA from post-baseline nadir, or confirmed increase in HCV RNA ≥ LLOQ in any patient who achieved HCV RNA < LLOQ. Post-treatment relapse was defined as HCV RNA ≥ LLOQ after treatment discontinuation in patients who completed the treatment regimen (at least 51 days) and had HCV RNA < LLOQ at the time of treatment discontinuation. Safety endpoints included the incidence of treatment-emergent adverse events (TEAEs) or serious adverse events (SAEs), AEs resulting in treatment discontinuation, clinical laboratory abnormalities, vital-sign measurements, and electrocardiographic parameters. Treatment-emergent AEs were those with a start date/time within the treatment period, defined as the time from first study drug administration to 4 weeks after treatment. Events that occurred after 4 weeks with a relationship to the study drug recorded as "related" were also considered treatment emergent.

Statistical analysis

It was determined that enrollment of 266 patients (including 71 patients with HCV GT3 infection) into the PK/PC-PP population would provide at least 90% confidence that the true overall SVR rate is at least 93% if the observed overall SVR rate exceeds 95%. With this sample size, the expected width of the two-sided 95% confidence interval (CI) for SVR is ≤ 5.3% if the true SVR rate is ≥ 95%.

In the ITT population, missing 12-week post-treatment SVR data were forward imputed as failure for any patient with HCV RNA > LLOQ at the last observation before the time point and backward imputed as success for any patient who had HCV RNA < LLOQ at 24 weeks after treatment. Patients who discontinued the study early for treatment-related AEs were imputed as failures if they did not have an HCV RNA result at 12 weeks after treatment. The per-protocol populations included patients who were evaluable for SVR, that is, patients for whom SVR was observed or could be imputed. For each population analyzed, the proportion of patients achieving SVR was estimated with two-sided 95% CIs computed using the Wilson Score method. All statistical analyses were conducted using Statistical Analysis System Version 9.4 or higher (SAS Institute, Cary, NC, USA).

RESULTS

Patients and analysis groups

Of the 376 patients screened, 275 were enrolled, received at least one dose of study drug, and were included in the ITT population. Of these, 259 patients had outcomes at post-treatment week 12 regardless of adherence and were included in the EE-PP population, while 215 patients were treatment-adherent, as corroborated by pill count and plasma drug levels, and were included in the PK/PC-PP population (Figure 1). The most common reasons for screen failure (n = 101) were patient withdrawal (eg, unable to comply with study requirements; n = 21), laboratory parameters outside the eligible limits (n = 18), and HCV RNA level ≤ 1000 IU/mL at screening (n = 15). A full list of reasons for screen failure is provided in Supplemental Table S1, https://links.lww.com/HEP/K559. The baseline characteristics of patients in the PK/PC-PP population were generally consistent with the ITT population (Table 1). The mean age of patients was 50.5 years, 47% of the patients were male, and 73% were White. The majority of patients (84%) did not have cirrhosis, and HCV

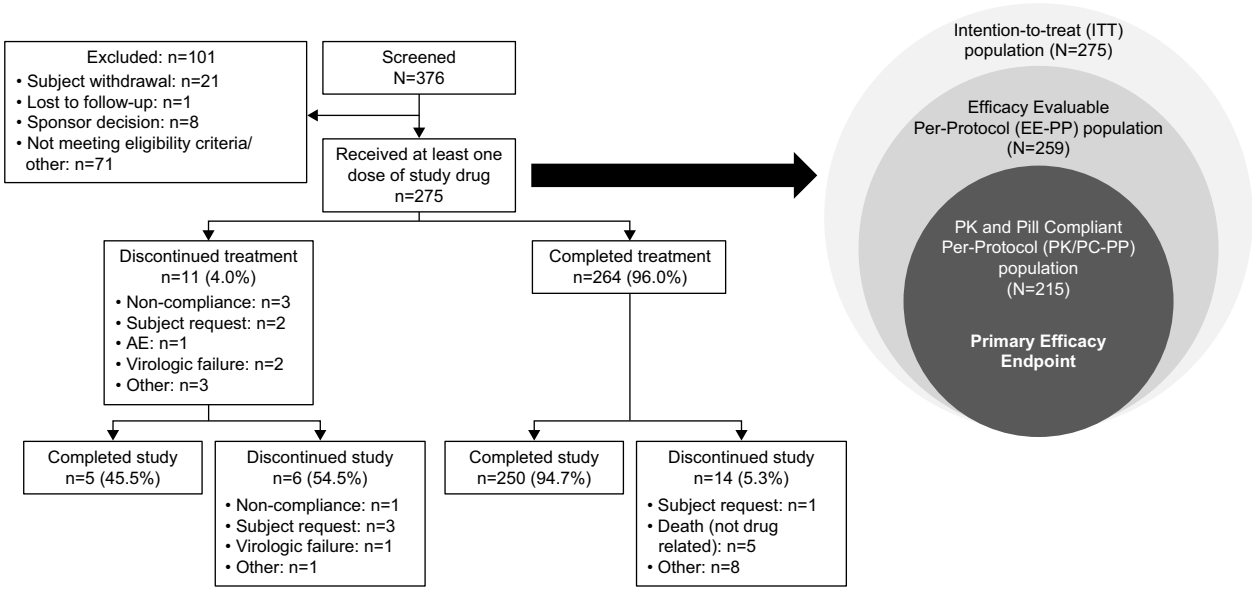


FIGURE 1 Patient disposition and analysis sets. Abbreviations: AE, adverse event; EE-PP, efficacy evaluable per-protocol; ITT, intention-to-treat; PK/PC-PP, pharmacokinetic and pill-compliant per-protocol.

genotypes 1, 2, 3, and 4 represented 70%, 2%, 27%, and 1% of infected patients, respectively. Baseline NS5A RASs were detected in 57 of the 215 patients (27%) in the PK/PC-PP population. Baseline NS5B RASs were detected in 70 of 208 evaluable patients (34%). The baseline characteristics of all analysis populations are listed in Supplemental Table S2, https://links.lww.com/HEP/K559.

Efficacy analysis

Of the 275 patients in the ITT population, 248 (90%) achieved SVR12 (95% CI: 86–93%)—92% in those without cirrhosis and 81% in those with compensated cirrhosis (Figure 2A). Thirteen patients (5%) in this population did not have measurements to determine SVR12. Missing data were treated as failures in these analyses; however, 11 of the 13 had HCV RNA < LLOQ at the last available post-baseline time point, 1 did not have data available beyond week 1, and 1 did not have post-baseline data. Further details on these patients are included in Supplemental Table S3, https://links.lww.com/HEP/K559.

Overall, 98% of patients in the primary PK/PC-PP population achieved SVR12 (210/215; 95% CI: 95–99%). SVR12 was 88.2% in patients with cirrhosis and 99.4% in those without cirrhosis, with overall rates of 80%–90% and 99%–100%, across genotypes, in those with and without cirrhosis, respectively (Table 2 and Figure 2B).

In the secondary EE-PP population, which included patients regardless of study drug adherence, SVR12 rates were similar. Overall, 95% of patients (245/259; 95% CI: 91%–97%) achieved SVR12 in this population —97% in those without cirrhosis and 81% in those with compensated cirrhosis (Figure 2C). Furthermore, in the subset of 44 patients who were included in this EE-PP population but excluded from the primary PK/PC-PP population (ie, non-adherent patients), 41 were non-cirrhotic, and 3 were cirrhotic. Of the non-cirrhotic patients in this group, 90% (35/39) with study drug compliance > 60% (based on pill count) achieved SVR12, while neither of the 2 patients with study drug compliance ≤ 60% achieved SVR12. None of the 3 cirrhotic patients in this group achieved SVR12, and each had study drug compliance > 60%.

Viral kinetics

Patients without cirrhosis achieved on-treatment HCV RNA < LLOQ earlier than those with compensated cirrhosis. This delay in reaching undetectable HCV RNA levels among cirrhotic patients may have contributed to their higher relapse rate and consequently lower SVR12 rates (Table 2). Nonetheless, all patients in the PK/PC-PP population, including those with cirrhosis, achieved HCV RNA < LLOQ by week 8 (end of treatment), indicating treatment responsiveness but suggesting that a longer treatment duration may be warranted for patients with cirrhosis.

Virologic failure, impact of baseline RASs, and treatment-emergent RASs

In the primary PK/PC-PP population, 5 patients (2%) who did not achieve SVR12 experienced post-

TABLE 1 Baseline characteristics of the PK/PC-PP population and ITT population

Characteristic	PK/PC-PP population (N = 215)	ITT population (N = 275)
Mean age, years (range)	51 (20–85)	50 (20–85)
Male sex, n (%)	100 (47)	144 (52)
Mean body mass index (range)	26 (16–44)	26 (16–47)
Race, n (%)		
White	156 (73)	192 (70)
Black/African American	6 (3)	11 (4)
Asian	44 (20)	55 (20)
Other	9 (4)	16 (6)
HCV genotype, n (%)		
1	151 (70)	189 (69)
1a	31 (14)	49 (18)
1b	119 (55)	139 (51)
1c	1 (< 1)	1 (< 1)
2	4 (2)	7 (3)
2a	2 (< 1)	2 (< 1)
2b	2 (< 1)	5 (2)
3	58 (27)	77 (28)
3a	53 (25)	72 (26)
3b	5 (2)	5 (2)
4	2 (< 1)	2 (< 1)
4a	2 (< 1)	2 (< 1)
Compensated cirrhosis, n (%)	34 (16)	37 (13)
Mean HCV RNA level, log_{10} IU/mL (SD)	6.3 (0.8)	6.2 (1.0)
Baseline RAS, n (%)		
NS5A baseline RAS	57 (27)	73 (27)
NS5B baseline RAS	70 (34[a])	89 (34[a])

[a]208 patients in the PK/PC-PP population and 261 patients in the ITT population had baseline samples sequenced for NS5B RASs.
Abbreviations: HCV, hepatitis C virus; ITT, intention-to-treat; NS5A, non-structural protein 5A; NS5B, non-structural protein 5B; PK/PC-PP, pharmacokinetic and pill-compliant per-protocol; RAS, resistance-associated substitution.

treatment relapse within 4 weeks of treatment discontinuation. One non-cirrhotic patient with post-treatment relapse had GT1a infection and did not have baseline RASs. Of the 4 cirrhotic patients with post-treatment relapse (2 GT1a, 1 GT1b, 1 GT3a), 2 had baseline NS5A RAS (Y93H/N in 1 GT1a patient; L31M in 1 GT1b patient), and 2 did not have baseline RAS (with GT1a and GT3a infection). None of these 5 patients had NS5B RASs at baseline.

No treatment-emergent RASs were observed at the time of relapse in the non-cirrhotic patient with GT1a infection with virologic failure. The following treatment-emergent RASs were observed in 3 of the 4 cirrhotic patients at the time of relapse: M28T, Q30H, and Y93H (enriched) NS5A RASs in the patient with GT1a infection and Y93H/N at baseline; N206K NS5B RAS and L31M (enriched) NS5A RAS in the patient with GT1b infection and L31M at baseline; Y93H NS5A RAS in the patient with GT3a infection and no RASs at baseline. Overall, high rates of SVR12 were achieved regardless of the presence or absence of baseline NS5A and NS5B RASs across genotypes in the PK/PC-PP population (Supplemental Table S4, https://links.lww.com/HEP/K559). In the ITT population (which included non-compliant and lost to follow-up patients), 14 patients (5%) experienced virologic failure—7/238 (3%) were non-cirrhotic, and 7/37 (19%) had cirrhosis. Five of the 14 were on-treatment failures, and 9 of the 14 were post-treatment relapses. Viral kinetics and pharmacokinetic analyses showed that most virologic failures were due to treatment non-adherence. None of the compliant patients experienced on-treatment virologic failure.

With regard to long-term durability of response, no patients experienced virologic failure between 12 and 24 weeks of post-treatment follow-up. Therefore, SVR24 rates were the same as the SVR12 rates. In the PK/PC-PP population, 98% (206 of 211 patients) achieved SVR24.

Safety and tolerability

Overall, 43% of patients (118 of the 275 patients comprising the ITT population) experienced at least 1 TEAE, with the most commonly reported events summarized in Table 3. These were mild to moderate in 112 of 118 patients (95%) reporting TEAEs. Treatment-emergent SAEs were reported in 9 patients (3%); however, there were no clear patterns with respect to SAE preferred term or accumulation of events in any particular System Organ Class category (Supplemental Table S5, https://links.lww.com/HEP/K559). One treatment-emergent SAE (esophageal squamous cell carcinoma) resulted in death. An additional 10 patients (4%) reported SAEs that occurred at least 4 weeks after discontinuation of study treatment (ie, after the treatment-emergent analysis period), of which 4 resulted in death. Two patients died from an overdose (unrelated to bemnifosbuvir and ruzasvir), 1 patient died from a gunshot wound, and 1 patient died from an unknown cause. None of the SAEs were assessed as related to study treatment. There was one premature treatment discontinuation due to SAE of complete atrioventricular block, deemed unrelated to the study drug. Grade 3/4 laboratory abnormalities were infrequent. The most common were decreased glomerular filtration rate (GFR) (5%), elevated glucose (1%), and elevated lipase (1%) (Supplemental Table S6, https://links.lww.com/HEP/K559). It should be noted that the study allowed enrollment of

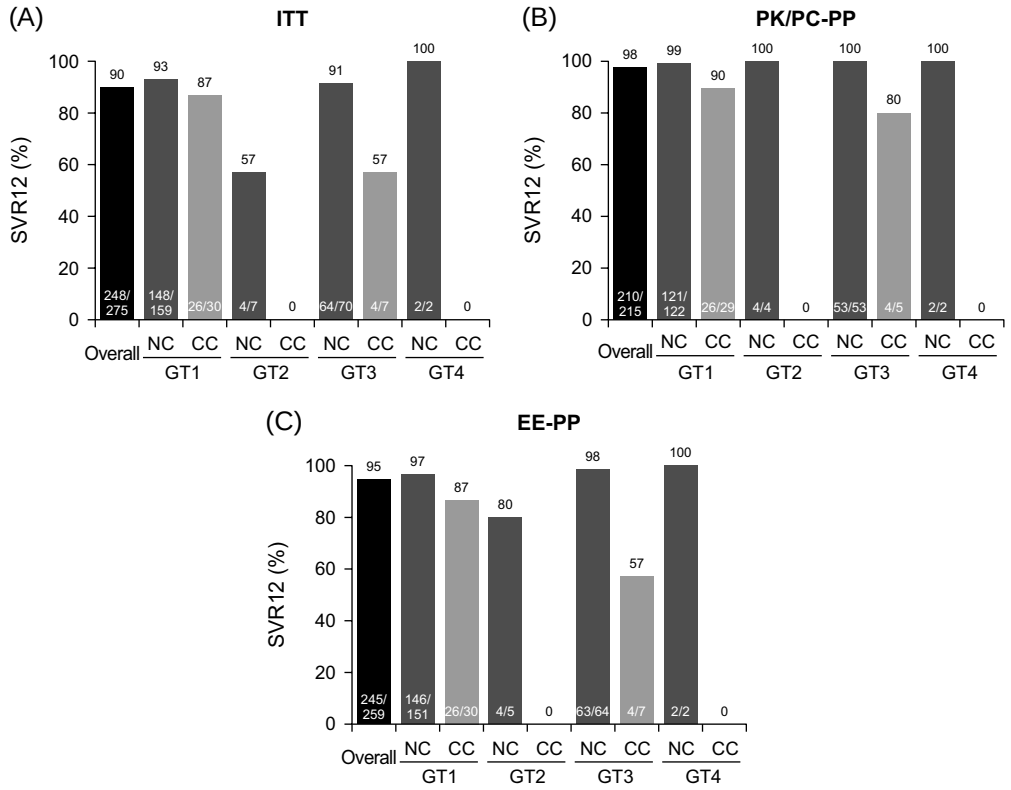


FIGURE 2 Proportion of patients achieving SVR12 according to HCV genotype and cirrhosis status in (A) the ITT population, (B) the PK/PC-PP population, and (C) the EE-PP population. Abbreviations: CC, compensated cirrhosis; EE-PP, efficacy evaluable per-protocol; GT, genotype; ITT, intention-to-treat; NC, no cirrhosis; PK/PC-PP, pharmacokinetic and pill-compliant per-protocol; SVR12, sustained virologic response at 12 weeks post-treatment.

TABLE 2 Response during and after treatment in the PK/PC-PP and ITT population by cirrhosis status

	PK/PC-PP population (N = 215)		ITT population (N = 275)	
Response	**No cirrhosis (N = 181)**	**Compensated cirrhosis (N = 34)**	**No cirrhosis (N = 238)**	**Compensated cirrhosis (N = 37)**
HCV RNA < 15 IU/mL—no./total no. (%)				
During treatment				
Week 2	107/179 (60)	16/34 (47)	137/238 (58)	16/37 (43)
Week 4	168/178 (94)	29/34 (85)	214/238 (90)	31/37 (84)
Week 8	181/181 (100)	34/34 (100)	229/238 (96)	35/37 (95)
After the end of treatment				
SVR4	180/181 (99)	30/34 (88)	225/238 (95)	31/37 (84)
SVR12	180/181 (99)	30/34 (88)	218/238 (92)	30/37 (81)
GT1a	26/27 (96)	2/4 (50)	39/45 (87)	2/4 (50)
GT1b	94/94 (100)	24/25 (96)	108/113 (96)	24/26 (92)
GT1c	1/1 (100)	0	1/1 (100)	0
GT2	4/4 (100)	0	4/7 (57)	0
GT3	53/53 (100)	4/5 (80)	64/70 (91)	4/7 (57)
GT4	2/2 (100)	0	2/2 (100)	0
Virologic failure—no. (%)				
During treatment	0	0	3/238 (1)	2/37 (5)
After treatment	1 (< 1)	4 (12)	4/238 (2)	5/37 (14)

Abbreviations: GT, genotype; HCV, hepatitis C virus; ITT, intention-to-treat; PK/PC-PP, pharmacokinetic and pill-compliant per-protocol; SVR4, sustained virologic response at 4 weeks post-treatment; SVR12, sustained virologic response at 12 weeks post-treatment.

TABLE 3 Treatment-emergent adverse events (≥2% of patients)

	ITT population (N = 275)
Any treatment-emergent adverse event, n (%)	118 (43)
Headache	24 (9)
Nausea	22 (8)
Dyspepsia	12 (4)
Vomiting	12 (4)
Pyrexia	11 (4)
Diarrhea	10 (4)
Fatigue	7 (3)
Abdominal pain upper	6 (2)

Abbreviation: ITT, intention-to-treat.

patients with moderate-to-severe chronic kidney disease (patients with eGFR ≥30 mL/min/1.73 m^2 were allowed to enroll). As such, 11 of the 13 patients with Grade 3/4 GFR had Grade 2 or higher abnormalities pre-dose (screening or day 1), and post-baseline reports of decreased GFR were generally single-time point, isolated decreases that returned to baseline with continued dosing or repeat confirmatory testing. One of the 13 patients who did not have low GFR at baseline had a Grade 4 result at week 4, which appeared to be a lab artifact, as testing was repeated 4 days later and was normal. Similarly, high-grade glucose and lipase elevations were infrequent, asymptomatic, isolated single-time-point elevations that generally returned to pre-dose levels with continued dosing or repeat confirmatory testing, with no clinical evidence of pancreatitis. No clinically relevant trends were observed in laboratory or electrocardiogram parameters collected during the study.

DISCUSSION

In this Phase 2 study, 8 weeks of treatment with bemnifosbuvir and ruzasvir resulted in high SVR12 rates of 98% in the primary per-protocol population and 90% in the ITT population of treatment-adherent, DAA-naïve HCV-infected patients. Furthermore, in patients without cirrhosis, SVR12 rates were 99%–100% across genotypes with only 8 weeks of treatment. In this non-cirrhotic group, only a single patient (with GT1a infection) did not achieve SVR12. In contrast to the ASTRAL-1 study (Phase 3 registrational study evaluating sofosbuvir/velpatasvir for 12 weeks),[24] the present study's ITT population is notably more heterogeneous and reflects a broader, real-world HCV population, aligning more closely with the realities of contemporary HCV care. In ASTRAL-1, 2 patients relapsed, 2 patients were lost to follow-up, 1 patient withdrew consent, and 1 patient died during follow-up, leaving just 4 participants without virologic outcome data and yielding an overall SVR12 rate of ~99% after 12 weeks of sofosbuvir/velpatasvir. The HCV-infected population today is characterized by more complex social and medical circumstances, in which adherence challenges are more frequent and are captured within the ITT analyses of this study. Accordingly, evaluating the primary endpoint in this setting offers a more rigorous assessment of the intrinsic antiviral efficacy of the bemnifosbuvir plus ruzasvir regimen, largely independent of nonvirologic failures such as early discontinuation or loss to follow-up.

As the HCV-infected population has evolved since ASTRAL-1, its findings may not fully reflect the more complex patient profile encountered in contemporary real-world practice, making direct comparisons with the outcomes of the current study challenging.[3,4] Topline results from C-BEYOND (NCT06868264), a pivotal Phase 3 study conducted in North America, comparing the efficacy of the fixed-dose combination of bemnifosbuvir and ruzasvir with the fixed-dose combination of sofosbuvir and velpatasvir for the treatment of chronic HCV, have been reported, with complete results to be published in the future.[41] Within the evolving HCV treatment landscape, an 8-week nucleoside polymerase inhibitor/NS5A-inhibitor regimen has the potential to improve treatment completion rates, as well as the proportion of patients who ultimately achieve cure, by reducing the overall treatment duration. The Phase 3 study results, pending publication of complete data, may support the potential of the bemnifosbuvir and ruzasvir combination to become the only protease inhibitor-free 8-week treatment option for patients without cirrhosis.

In patients with cirrhosis, viral clearance appeared to occur more slowly than in those without cirrhosis, based on time to achieve HCV RNA <LLOQ. These kinetics likely explain the lower SVR12 rates observed in cirrhotic patients after 8 weeks of treatment (88% SVR12 rate in cirrhotic patients in the PK/PC-PP population). Considering that all patients in this population achieved HCV RNA <LLOQ by 8 weeks and that failures were post-treatment relapses (as opposed to on-treatment breakthroughs), this would suggest that longer treatment duration could further optimize efficacy. The quantifiable virus observed at week 4 in some cirrhotic patients may be characterized as attenuated viral decay. Virologic failure, being relapse rather than breakthrough, suggests that treatment duration was the critical factor underlying virologic failure in cirrhotic patients, and therefore extension of treatment duration may enhance efficacy.

As the PK/PC-PP population was the primary analysis population, it allowed for a more accurate efficacy assessment of the study drugs by restricting the analysis to instances of virologic failure in patients who were adherent, verified by adequate drug exposure and pill count. The secondary analyses in the EE-

PP population, however, included an additional 44 patients (which comprised 17% of the EE-PP population), where the observed SVR12 rate of 95% was similar to the rate observed in the primary analysis. SVR12 rates of 90% were observed in the safety population. The lower rates in the ITT population can be explained by the 5% patients who did not have a measurement at SVR12 (with missing data considered SVR12 failures), but these patients without SVR12 data did not meet the criteria for virologic failure, as they responded to treatment per their last available assessment. Although the number of non-adherent patients was relatively small compared with the broader population, the secondary EE-PP population, which included patients regardless of study drug adherence, showed a similar SVR12 rate (95%) to those observed in the ITT and primary PK/PC-PP populations. However, given the limited number of non-adherent patients and the availability of SVR12 data for only a subset of the ITT population, these findings should be interpreted with caution and warrant further evaluation in larger studies. The number of virologic failures was small, with no clear patterns associated with the impact of baseline RASs on SVR12 rates. These results are consistent with earlier data demonstrating the high resistance barrier and potency of bemnifosbuvir and ruzasvir, reducing the risk that baseline resistance will affect treatment outcomes.[30,31] Treatment with bemnifosbuvir and ruzasvir was well tolerated, with no reported SAEs related to the study treatment. One patient experienced an SAE of complete atrioventricular block leading to premature treatment discontinuation, which was considered unrelated to the study drug. Nonserious AEs included mild-to-moderate headache and nausea, none of which resulted in treatment discontinuations. Notably, ~17% of patients had a history of psychiatric disorders, including 9% who had a history of drug abuse, which is representative of the current HCV-infected patient population.[42]

There are some limitations to this present study, one of which is that this was a single-arm study. Considering the known historical high rates of SVR12 that are observed with current standard-of-care HCV regimens, this was acceptable considering the objectives of this Phase 2 study; however, these results are being further evaluated in the C-BEYOND Phase 3 study. Ensuring adherence to the study drug and continued study participation are especially challenging in the current population of HCV-infected patients, particularly in patients with comorbidities such as alcohol or drug addiction and mental illness.[43] The primary analysis population was selected to ensure the efficacy of the drug combination could be effectively characterized and compared with prior clinical studies. Additional study limitations include the lack of systematic screening for drug use at breakthrough or relapse, the inability to evaluate SVR12 outcomes by drug-use history because of the small sample size, and the modest sample size, resulting in a relatively small proportion of genotypes other than GT1 and GT3, which may also limit the generalizability of the data to other genotypes.

In conclusion, 8-week treatment with the combination of bemnifosbuvir and ruzasvir was well tolerated and achieved high rates of SVR12 across genotypes, regardless of the presence or absence of baseline RASs. These data support the ongoing clinical development of bemnifosbuvir and ruzasvir as a DAA treatment with high rates of efficacy with a short treatment duration, low risk of drug–drug interactions, and may be taken with or without food. A fixed-dose combination regimen is currently being evaluated in 2 ongoing Phase 3 studies: as an 8-week treatment for non-cirrhotic patients and as a 12-week treatment for cirrhotic patients with chronic HCV infection (ClinicalTrials.gov: NCT06868264 and NCT07037277).

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

AUTHOR CONTRIBUTIONS

Bruce Belanger, Shannan Lynch, Qi Huang, Xiao-Jian Zhou, and Arantxa Horga contributed to the conception and design of the study. Alina Jucov, Brian Conway, Jordan J. Feld, Saeed Hamid, Elena Ermacicova, Laura Iliescu, and Eric Lawitz contributed to data acquisition. Bruce Belanger performed the formal analysis. Bruce Belanger, Shannan Lynch, Qi Huang, Xiao-Jian Zhou, and Arantxa Horga contributed to the investigation, data interpretation, and drafting of the manuscript. All authors have critically reviewed, edited, and approved the final version of the manuscript for submission.

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CONFLICTS OF INTEREST

Alina Jucov reports receiving advisor or consultancy fees from Bluejay Therapeutics, Gossamer Bio, Precision Bio, and Vir Biotechnology. Brian Conway reports receiving speaker and/or consultancy fees from AbbVie, Gilead, Merck & Co., and Pfizer. Jordan J. Feld reports receiving grants and consultancy fees from AbbVie, Atea Pharmaceuticals, Inc., and Gilead. Bruce Belanger, Shannan Lynch, Qi Huang, Xiao-Jian Zhou, and Arantxa Horga are employees of Atea Pharmaceuticals, Inc and may own stock in Atea Pharmaceuticals, Inc. Eric Lawitz reports receiving research, speaker, or consultancy fees

from 89Bio Inc., AbbVie, Akero Therapeutics, Alnylam Pharmaceuticals, Inc., Amgen, Atea Pharmaceuticals, Inc., AstraZeneca, Boehringer Ingelheim, Bristol Myers Squibb, Corcept Therapeutics, Cour Pharmaceuticals, Eli Lilly and Company, Enanta Pharmaceuticals, Enyo Pharma, Exalenz Bioscience, Galectin Therapeutics, Galmed Pharmaceuticals, Genfit, Gilead Sciences, GlaxoSmithKline, Hanmi Pharmaceuticals, Hightide Biopharma, Intercept Pharmaceuticals, Inventiva, Ipsen, Janssen Pharmaceuticals, Madrigal Pharmaceuticals, Inc., Merck & Co., NGM Biopharmaceuticals Inc., Northsea Therapeutics, Novartis, Novo Nordisk Inc., Organovo, Poxel Co., Regeneron, Sagimet Biosciences, Takeda, Terns Pharmaceuticals, Viking Therapeutics, and Zydus Pharmaceuticals. The remaining authors have no conflicts to report.

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