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SOF/VEL Post Transplant

**Sofosbuvir/Velpatasvir for 12 Weeks in Genotype 1-4 HCV-Infected Liver Transplant Recipients**

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Kosh Agarwal receives grant support from Gilead, AbbVie, BMS, and MSD.

Lluis Castells has nothing to disclose.

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William M.C. Rosenberg receives grant support from Gilead, is on the advisory board for Gilead, BMS and AbbVie, and has received institutional research support from Gilead, BMS, AbbVie, and MSD.

Brian McNabb, Sarah Arterburn, Gregory Camus, John McNally, Luisa M. Stamm, Diana M. Brainard, and G. Mani Subramanian are employees of Gilead.

Zoe Mariño has acted as advisor for Gilead and BMS.
Jean-François Dufour is on the advisory board for AbbVie, Bayer, BMS, Falk, Genfit, Gilead, Intercept, Lilly, Merck, and Novartis, has received speaking grants from AbbVie, Bayer, BMS, Genfit, Gilead, and Novartis, and has received an unrestricted research grant from Bayer.

Xavier Forns received unrestricted grants from AbbVie and acted as advisor for Gilead and AbbVie.

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**Author contributions:**

Kosh Agarwal, Lluis Castells, Brian McNabb, Diana M. Brainard, Jean-François Dufour, and Xavier Forns participated in the concept and design, data analysis.

Beat Müllhaupt, William M.C. Rosenberg, Sarah Arterburn, Gregory Camus, Luisa M. Stamm, and Zoe Mariño participated in the data analysis.

G. Mani Subramanian participated in concept and design.

All authors participated in the critical revision and approval of the submitted manuscript.

**Clinical trial number:** NCT02781571
ABSTRACT [275 words]

**Background & Aims:** Sofosbuvir, an NS5B inhibitor, combined with velpatasvir, an NS5A inhibitor (SOF/VEL), produces high sustained virologic response rates 12 weeks after treatment (SVR12) in patients with genotype 1-6 HCV infection, and has no anticipated clinically relevant drug-drug interactions with immunosuppressants. This study evaluated the safety and efficacy of SOF/VEL in adults with recurrent chronic genotype 1-4 HCV infection after liver transplant.

**Methods:** Patients received SOF/VEL 400/100 mg daily for 12 weeks. Patients could be treatment experienced or treatment naïve with no cirrhosis or with compensated cirrhosis. The primary endpoints were SVR12 and discontinuations due to adverse events.

**Results:** The study enrolled and treated 79 patients (37 [47%] had genotype 1, 3 [4%] genotype 2, 35 [44%] genotype 3, and 4 [5%] genotype 4 HCV). Of these, 81% were male, 82% were white, 18% had compensated cirrhosis, and 59% were treatment experienced. The most commonly used immunosuppressants were tacrolimus (71%), mycophenolic acid (24%), cyclosporine (14%), and azathioprine (11%). Median (range) time from liver transplantation was 7.5 (0.3, 24) years. The SVR12 rate was 96%. By genotype, SVR12 rates were 95% (genotype 1), 100% (genotype 2), 97% (genotype 3), and 100% (genotype 4). Two patients experienced virologic relapse: one with genotype 1a infection was non-cirrhotic and treatment naïve, and one with genotype 3 infection was non-cirrhotic and treatment experienced. One patient discontinued SOF/VEL due to hyperglycemia. No serious or severe adverse events were deemed SOF/VEL-related by
the investigator, and no liver transplant rejection episodes or deaths occurred during the study period.

**Conclusions:** Treatment with SOF/VEL for 12 weeks was highly effective and well tolerated in genotype 1-4 HCV-infected liver transplant recipients with and without cirrhosis.
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LAY SUMMARY

Sofosbuvir/velpatasvir is a combination of two drugs in one tablet that is approved for the treatment of patients with chronic hepatitis C virus (HCV) infection. When patients with chronic HCV infection receive a liver transplant, the HCV infection usually recurs, and damages the liver transplant. This study tested the effects of 12 weeks of sofosbuvir/velpatasvir treatment in patients who had HCV recurrence after a liver transplant. Three months following the end of treatment, 96% of patients were cured of HCV infection.
INTRODUCTION

Among HCV-infected liver transplant recipients, HCV recurrence emerges in nearly all patients [1]. Within 5 years post-transplant, cirrhosis related to HCV ensues in approximately 30% of patients with recurrent, chronic HCV infection and is associated with increased graft loss rates and death [2-5]. In the setting of post-transplant immunosuppression, the rate of hepatic fibrosis is accelerated in HCV-infected patients compared to the pre-transplant period [6].

Historically, the therapeutic regimen to treat HCV recurrence in liver transplant recipients was the combination of interferon and ribavirin. This regimen was marked by sustained virologic response (SVR) rates of less than 30% and poor tolerability [7-9]. More recently, the regimen of sofosbuvir (a nucleotide analog HCV NS5B polymerase inhibitor) with ribavirin has led to improvements in tolerability and SVR rates coupled with shorter treatment duration [10-11]. Further improvement in SVR rates (88% to 97%) has been observed in liver transplant recipients treated with sofosbuvir in combination with a second direct-acting antiviral (DAA; simeprevir, ledipasvir, or daclatasvir) and ribavirin, or the regimen of ombitasvir, paritaprevir, ritonavir, dasabuvir, and ribavirin [12-16]. Despite these important advances in therapy, some regimens have suboptimal efficacy (<90% SVR rates), none of these regimens is pangenotypic, and all contain ribavirin, exposing patients to the known hematologic, constitutional, and neuropsychiatric effects [17,18]. Glecaprevir/pibrentasvir is a recently approved ribavirin-free regimen that led to high SVR rates in liver and kidney transplant recipients in clinical trials, but was not evaluated in cirrhotic or treatment-experienced genotype 3 HCV-infected transplant recipients [19]. Thus, an unmet need remains among liver
transplant recipients for interferon- and ribavirin-free regimens that are effective for all genotypes regardless of prior treatment experience or cirrhosis status, are well tolerated, have high barriers to resistance, and low propensity for drug interactions.

The single-tablet regimen of sofosbuvir and velpatasvir (SOF/VEL) combines sofosbuvir with the second-generation, pangenotypic HCV NS5A inhibitor velpatasvir; both drugs demonstrate potent antiviral activity against HCV replicons in genotypes 1 through 6. In phase 3 trials in non-transplant patients without cirrhosis or with compensated cirrhosis, a 12-week regimen of SOF/VEL led to SVR rates at 12 weeks after the end of treatment (SVR12) of 99% in patients with HCV genotypes 1, 2, 4, 5, and 6, and 95% in patients with HCV genotype 3, regardless of prior non-NS5A treatment experience [20,21]. These results have been confirmed in real-world trials [22-25]. Importantly, no drug interactions between SOF/VEL and immunosuppressant medications are anticipated [26,27]. These features make SOF/VEL well suited to address the unmet medical needs of liver transplant recipients.

To evaluate SOF/VEL in the setting of HCV recurrence following liver transplantation, this multicenter, open-label study evaluated the safety, tolerability, and efficacy of SOF/VEL administered for 12 weeks in liver transplant recipients with chronic HCV infection.

### PATIENTS AND METHODS

#### Patients
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Patients were enrolled between 10 August 2016 and 14 February 2017 at 15 clinical sites in the United Kingdom, Spain, and Switzerland (ClinicalTrials.gov number, NCT02781571). Eligible patients were men and women at least 18 years of age with chronic genotype 1-6 HCV infection who received a liver transplant alone or combined liver-kidney transplant at least 3 months prior to study entry with no clinical signs of rejection in the past 3 months. Patients without cirrhosis or with compensated cirrhosis who were treatment experienced or treatment naïve were eligible. Patients with prior NS5A inhibitor treatment or coinfection with hepatitis B virus or human immunodeficiency virus infection were not eligible. Long-term corticosteroids at a dose of >5 mg of prednisone per day for maintenance of immunosuppression were prohibited. A full list of eligibility criteria is provided in the Supplement. All patients provided written informed consent.

Study Design

In this phase 2 open-label study, patients self-administered one SOF/VEL 400 mg/100 mg tablet (EPCLUSA®, Gilead Sciences, Inc., Foster City, CA) [26,27] once daily with or without food for 12 weeks. Patients were treated with immunosuppressive agents for post-transplant care or to manage any episodes of rejection per the treating physician’s discretion in accordance with local institutional guidelines. Immunosuppressant drug levels, dose adjustments, and medication changes were recorded. Study visits with assessments were performed at screening, baseline/day 1, weeks 2, 4, 8, and 12 of treatment, and posttreatment weeks 4 and 12.

For further details regarding the materials used, please refer to the CTAT table. The study design, which was in compliance with the Declaration of Helsinki, Good Clinical
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Practice Guidelines, and local regulatory requirements, was approved by the institutional review board or independent ethics committee at each participating site (Supplement). The study was conducted according to the protocol by the sponsor (Gilead Sciences) in collaboration with the academic investigators. All patients provided informed consent.

Assessments

Serum HCV RNA was measured using the COBAS AmpliPrep/COBAS TaqMan HCV Quantitative Test, v2.0 (Roche Molecular Diagnostics, Pleasanton, CA, United States) with a LLOQ of 15 IU/mL. HCV genotype and subtype were determined using the VERSANT HCV genotype INNO-LiPA 2.0 Assay (Siemens Healthcare GmbH, Erlangen, Germany). For patients for whom genotyping data could not be provided with the VERSANT assay, either the TRUGENE HCV 5’NC Genotyping Kit (Siemens Healthcare GmbH) or NS5B sequencing was used to determine genotype/subtype.

Deep sequencing of the HCV NS5A and NS5B coding regions was performed on all patients from the Resistance Analysis Population (patients who achieved SVR12 or had virologic failure) to detect resistance associated substitutions (RASs) present at baseline and again for patients with virologic failure to detect treatment-emergent RASs. RASs present in more than 15% of read sequences were reported [28,29].

Safety assessments included monitoring of adverse events (AEs), clinical laboratory tests, and vital signs assessment at all on-treatment and post-treatment visits and physical examinations at baseline, week 12, and post-treatment weeks 4 and 12.

Statistical Analyses
The primary efficacy endpoint was the proportion of patients achieving SVR12, defined as HCV RNA <LLOQ 12 weeks after treatment cessation, among all enrolled patients who received at least one dose of study drug. The proportion of patients with SVR12 was calculated along with a 2-sided 95% confidence interval (using the Clopper-Pearson method) by genotype and for the overall study group. The effects of age (<65 versus ≥65 years old), gender, cirrhosis status, and prior treatment experience were assessed through subgroup analyses using identical methods. The primary safety endpoint was any AE leading to discontinuation of study drug. SAS Software version 9.4 was used for statistical analysis (SAS Institute, NC, United States).

Because this is an exploratory study, no formal power calculations were used to determine sample size. A sample size of 80 patients was selected for practical reasons.

**RESULTS**

Of the 85 patients screened, 79 patients were enrolled, treated, and completed the study (Supplemental Figure 1). Baseline characteristics are shown in Table 1. The majority of patients were male (81%) and white (82%). The study enrolled 37 patients with genotype 1, 3 with genotype 2, 35 with genotype 3, and 4 with genotype 4 HCV infection; no patients with genotypes 5 or 6 HCV infection were enrolled. Two patients had received combined liver-kidney transplants. Cirrhosis was present in 14 (18%) patients and 47 (59%) patients were treatment-experienced (4 [9%] DAA + ribavirin +/- pegylated interferon, 26 [55%] pegylated interferon +/- ribavirin, and 17 [36%] interferon +/- ribavirin).
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Efficacy

Overall, SVR4 was achieved by 77/79 (97%) patients, and SVR12 was achieved by 76/79 (96%) patients (95% for genotype 1, 100% for genotype 2, 97% for genotype 3, and 100% for genotype 4) (Figure 1). Three patients failed to achieve SVR12: two patients with virologic relapse and one patient with a non-virologic failure. One relapse occurred at post-treatment week 4 in a noncirrhotic, treatment-naïve patient with genotype 1a HCV infection whose HCV RNA level had become <LLOQ at treatment weeks 4, 8, and 12. According to pill counts, this patient had a 98% adherence rate. The second relapse occurred at post-treatment week 12 in a noncirrhotic, treatment-experienced (non-DAA) patient with genotype 3b HCV infection whose HCV RNA level was <LLOQ from treatment week 4 through post-treatment week 4. The patient did not return pill bottles for adherence calculations. Pharmacokinetic assessments could not confirm non-adherence for either patient. The non-virologic failure occurred in a patient with genotype 1b HCV infection who discontinued therapy on day 7 due to an AE of hyperglycemia (further described below).

Subgroups defined by age, gender, cirrhosis status, and prior treatment experience had no clinically relevant effect on SVR12 (Supplemental Table 1). Plasma levels of HCV RNA declined rapidly with treatment, with 100% of patients having HCV RNA less than the LLOQ after 8 weeks of treatment. No patients experienced on-treatment virologic failure.

Viral Resistance
There were 78 (99%) patients with virologic outcomes who were included in the Resistance Analysis Population. At baseline, 31% of patients had HCV NS5A RASs present, with substantial variability by genotype (13% of genotype 1a, 43% of genotype 1b, 100% of genotype 2, 17% of genotype 3, and 100% of genotype 4). Overall, 92% of patients with baseline NS5A RASs achieved SVR12 including 100% (3/3) of patients with genotype 3a and Y93H. Six patients had NS5B RASs at baseline, all of whom achieved SVR12.

The genotype 1a HCV-infected patient who relapsed had the NS5A RAS K24R at baseline and at relapse and the treatment-emergent NS5A RAS L31V; the genotype 3b HCV-infected patient who relapsed had NS5A RASs A30K and L31M at baseline and relapse and the treatment-emergent NS5B RAS S282T.

Safety

One patient with a history of diabetes discontinued SOF/VEL on day 7 due to an AE of hyperglycemia that started on day 4. All other patients completed treatment. A summary of the AEs and laboratory abnormalities is provided in Table 2. The most common AEs were headache (24%), fatigue (20%), and cough (10%). Serious AEs occurred in 3 (4%) patients (hepatocellular carcinoma, joint swelling, and Klebsiella pneumonia) and severe (grade 3 or grade 4) AEs occurred in 3 (4%) patients (toothache, joint swelling, and Klebsiella pneumonia), none of which were deemed related to study treatment by the investigators. No adverse events associated with renal dysfunction were reported. The only renal adverse events in the study included one patient each reporting Grade 1 dysuria, hematuria, and pollakiuria. No clinically meaningful changes in estimated glomerular filtration rate occurred during study treatment. Grade 3 and grade 4
laboratory abnormalities were uncommon: three patients with histories of diabetes experienced grade 3 hyperglycemia, 4 patients experienced asymptomatic grade 3 hyperuricemia, 1 patient with baseline proteinuria experienced a transient grade 3 proteinuria, and 1 patient experienced transient grade 4 lymphocytopenia. No patient experienced an episode of acute rejection, and no deaths occurred.

Eighteen (23%) patients had changes to their immunosuppression regimens (either dose change or medication change). Reasons for the changes of immunosuppression included following institutional guidelines (14 patients), improvement in organ function (1 patient), and other reasons (3 patients). For tacrolimus, the initial change was an increase for 11 patients and a decrease for one patient (due to improved organ function). No patients required changes to immunosuppression regimens for rejection or suspected drug-drug interactions.

DISCUSSION

Once-daily treatment with SOF/VEL for 12 weeks led to an SVR12 rate of 96% in liver transplant recipients with recurrent genotype 1-4 HCV infection. Treatment was well tolerated with one treatment discontinuation due to an AE, no episodes of rejection, and no adjustments to immunosuppression to manage suspected drug-drug interactions. These results occurred in the setting of no restrictions on concomitant immunosuppression therapy allowed in this study.

These results are especially notable because they were achieved in a broad HCV genotype 1-4 infected liver transplant population. Notably, 59% of the patient population
was treatment experienced, 44% had HCV genotype 3, and 18% had compensated cirrhosis. Characteristics associated with harder-to-cure patient populations, such as HCV genotype, the presence of cirrhosis, prior treatment experience, and pretreatment RASs had no clinically meaningful impact on SVR12 rates.

The SVR12 rates in the current trial were equal to or exceeded those of previously studied ribavirin-containing DAA regimens. Across genotypes, SVR12 rates in this study were 95% to 100%, while sofosbuvir combined with ribavirin and a second DAA (simeprevir, ledipasvir, or daclatasvir), or the regimen of ombitasvir, paritaprevir, ritonavir, dasabuvir, and ribavirin led to SVR12 rates of 88% to 97% [12-16]. Ribavirin-free regimens such as SOF/VEL constitute a great advantage, especially in liver transplant recipients who are more vulnerable to the hematologic and constitutional adverse effects of ribavirin [17,18]. Additionally, SOF/VEL is a one-pill-per-day regimen, and as such may be particularly important to liver transplant recipients who have high underlying pharmacologic burden.

The results of this study also compare favorably with the results from the MAGELLAN-2 trial, in which SVR12 was achieved in 99% of patients treated with glecaprevir/pibrentasvir after liver (80%) or kidney (20%) transplantation [19]. In contrast to our trial, MAGELLAN-2 did not evaluate patients with cirrhosis and fewer patients had HCV genotype 3 (24% in MAGELLAN-2 versus 44% in our trial) and prior treatment experience (34% in MAGELLAN-2 versus 47% in our trial).

One limitation of this phase 2 study is the relatively small sample size. Additionally, although patients with genotypes 5 or 6 were eligible to enroll in this study, none were enrolled. However, data from this trial are consistent with efficacy and safety data from
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the broader phase 3 ASTRAL clinical development program in non-liver transplant recipients, where SVR12 rates were 95% in genotype 3 and 99% in genotypes 1, 2, 4, 5, and 6 following 12 weeks of SOF/VEL treatment [20,21]. Similar results have also been observed in real-world studies of non-liver transplant recipients [22-25]. The consistency among studies provides confidence that the current results are representative of the safety and efficacy of SOF/VEL in liver transplant recipients.

Conclusions

The single-tablet regimen of SOF/VEL was well tolerated and highly effective in genotype 1-4 HCV-infected liver transplant recipients. SOF/VEL is a pangenotypic, ribavirin-free, simple, and well-tolerated treatment option for HCV-infected liver transplant recipients.
ACKNOWLEDGEMENTS

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REFERENCES


TABLES

Table 1. Baseline demographics and disease characteristics.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>N=79</th>
</tr>
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<tbody>
<tr>
<td>Mean age (range), y</td>
<td>62 (45, 81)</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>64 (81)</td>
</tr>
<tr>
<td>Mean body mass index (range), kg/m²</td>
<td>28 (18, 39)</td>
</tr>
<tr>
<td>Race, n (%)</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>65 (82)</td>
</tr>
<tr>
<td>Asian</td>
<td>12 (15)</td>
</tr>
<tr>
<td>Black or African American</td>
<td>2 (3)</td>
</tr>
<tr>
<td>HCV genotype, n (%)</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>37 (47)</td>
</tr>
<tr>
<td>a</td>
<td>15 (19)</td>
</tr>
<tr>
<td>b</td>
<td>22 (28)</td>
</tr>
<tr>
<td>2</td>
<td>3 (4)</td>
</tr>
<tr>
<td>3</td>
<td>35 (44)</td>
</tr>
<tr>
<td>4</td>
<td>4 (5)</td>
</tr>
<tr>
<td>HCV-RNA level, mean (SD), log₁₀ IU/mL</td>
<td>6.4 (0.55)</td>
</tr>
<tr>
<td>HCV-RNA ≥800,000 IU/mL, n (%)</td>
<td>61 (77)</td>
</tr>
<tr>
<td>IL28B genotype, n (%)</td>
<td></td>
</tr>
<tr>
<td>CC</td>
<td>39 (49)</td>
</tr>
<tr>
<td>CT</td>
<td>34 (43)</td>
</tr>
</tbody>
</table>
### SOF/VEL Post Transplant

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td>TT</td>
<td>6 (8)</td>
</tr>
<tr>
<td>Cirrhosis, n (%)</td>
<td>14 (18)</td>
</tr>
<tr>
<td>Prior HCV treatment, n (%)</td>
<td>47 (59)</td>
</tr>
<tr>
<td>Last HCV regimen received (n=47)</td>
<td></td>
</tr>
<tr>
<td>Pegylated interferon +/- ribavirin</td>
<td>26 (55)</td>
</tr>
<tr>
<td>Interferon +/- ribavirin</td>
<td>17 (36)</td>
</tr>
<tr>
<td>Direct-acting antiviral$^a$ + ribavirin +/- pegylated interferon</td>
<td>4 (9)</td>
</tr>
<tr>
<td>Median time from transplant (range), y</td>
<td>7.5 (0.3, 24)</td>
</tr>
<tr>
<td>Immunosuppressive therapy, n (%)</td>
<td></td>
</tr>
<tr>
<td>Tacrolimus</td>
<td>56 (71)</td>
</tr>
<tr>
<td>Mycophenolic acid</td>
<td>19 (24)</td>
</tr>
<tr>
<td>Cyclosporine</td>
<td>11 (14)</td>
</tr>
<tr>
<td>Azathioprine</td>
<td>9 (11)</td>
</tr>
<tr>
<td>Sirolimus</td>
<td>8 (10)</td>
</tr>
<tr>
<td>Everolimus</td>
<td>5 (6)</td>
</tr>
<tr>
<td>Prednisolone</td>
<td>1 (1)</td>
</tr>
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</table>

HCV, hepatitis C virus

$^a$Sofosbuvir, telaprevir (n=2), boceprevir
Table 2. Adverse events.

<table>
<thead>
<tr>
<th>Adverse Events, n (%)</th>
<th>N=79</th>
</tr>
</thead>
<tbody>
<tr>
<td>Any adverse event</td>
<td>62 (78)</td>
</tr>
<tr>
<td>Adverse events leading to sofosbuvir/velpatasvir discontinuation(^a)</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Serious adverse events(^b)</td>
<td>3 (4)</td>
</tr>
<tr>
<td>Severe adverse events(^c)</td>
<td>3 (4)</td>
</tr>
<tr>
<td>Adverse events occurring in (\geq 10%) of patients</td>
<td></td>
</tr>
<tr>
<td>Headache</td>
<td>19 (24)</td>
</tr>
<tr>
<td>Fatigue</td>
<td>16 (20)</td>
</tr>
<tr>
<td>Cough</td>
<td>8 (10)</td>
</tr>
<tr>
<td>Grade 3 and 4 laboratory abnormalities</td>
<td></td>
</tr>
<tr>
<td>Hyperuricemia(^d)</td>
<td>4 (5)</td>
</tr>
<tr>
<td>Hyperglycemia(^e)</td>
<td>3 (4)</td>
</tr>
<tr>
<td>Lymphocytopenia</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Proteinuria</td>
<td>1 (1)</td>
</tr>
</tbody>
</table>

\(^a\)Hyperglycemia on day 4 in a patient with diabetes.

\(^b\)Hepatocellular carcinoma, joint swelling, and *Klebsiella* pneumonia; all unrelated to study treatment.

\(^c\)Toothache, joint swelling, and *Klebsiella* pneumonia; all unrelated to study treatment.

\(^d\)All asymptomatic.

\(^e\)All in patients with histories of diabetes.
Figure Legend

Figure 1. Sustained virologic response rate 12 weeks after a 12-week course of sofosbuvir/velpatasvir by genotype. GT, genotype; SVR12, sustained virologic response rate 12 weeks after treatment cessation.
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SVR12, % (95% CI)

- Total: 96% (79/79)
- GT1a: 93% (14/15)
- GT1b: 95% (21/22)
- GT1: 95% (35/37)
- GT2: 100% (3/3)
- GT3: 97% (34/35)
- GT4: 100% (4/4)
Sofosbuvir/velpatasvir 400 mg/100 mg daily x 12 weeks

Rates of Sustained Virologic Response 12 Weeks After Treatment Cessation

HCV infected liver transplant recipient

Percent of Patients

<table>
<thead>
<tr>
<th>Genotype</th>
<th>All (n=79)</th>
<th>1 (n=37)</th>
<th>2 (n=3)</th>
<th>3 (n=35)</th>
<th>4 (n=4)</th>
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<tbody>
<tr>
<td></td>
<td>96</td>
<td>95</td>
<td>100</td>
<td>97</td>
<td>100</td>
</tr>
</tbody>
</table>
HIGHLIGHTS

- Study patients had recurrent hepatitis C infection following liver transplantation.
- The study included patients with cirrhosis (18%) and genotype 3 HCV (44%).
- Twelve weeks of sofosbuvir/velpatasvir resulted in an SVR12 of 96%.
- No liver transplant rejection episodes or deaths occurred.
- Treatment did not impact immunosuppressive therapy.