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Original Article

CLINICAL PROFILE AND TREATMENT RESPONSE TO SOFOSBUVIR-VELPATASVIR REGIMEN AND ITS IMPACT ON THE QUALITY OF LIFE IN CHRONIC HEPATITIS C PATIENTS

AJIT PRATAP SINGH, PANKAJ ASATI*, KANISHK YADAV

Department of Pharmacology, Netaji Subhash Chandra Medical College and Hospital, Jabalpur-482003, Madhya Pradesh, India
***Corresponding author: Pankaj Asati; *Email: drpankajasati88@gmail.com**

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ABSTRACT

Objective: Chronic Hepatitis C infection is one of the major global contributors to liver-related morbidity and mortality. Successful antiviral therapy with the direct-acting antiviral combination sofosbuvir-velpatasvir has shown to improve survival, liver-related outcomes, all-cause mortality, and even reverse fibrosis. We evaluated the treatment response of this regimen in terms of its impact on the Quality of life of patients during and after the therapy.

Methods: This is an Observational, prospective, descriptive study of serologic HCV RNA-positive cases. Data on change in clinical, biochemical profile, and quality of life using 2 standardized questionnaires SF-36 and CLDQ was analyzed at baseline, 12 w from baseline, and 24 w after therapy cessation.

Results: 62 out of the 87 patients registered during the study completed the desired study duration. 95% achieved sustained virological response (SVR) at 12 w and 100% at 24 w from baseline. Improved clinical profile with symptom resolution and change in the biochemical parameters and quality-of-life scores was statistically significant (P value-<0.001 and 0.005) at 12 w from baseline and 24 w after therapy cessation, respectively.

Conclusion: Patients achieving SVR with sofosbuvir-velpatasvir have been shown to improve clinical profile with normalization in biochemical markers of liver disease, which is reflected in all the quality-of-life domains of SF-36 and CLDQ-HCV. However, long-term follow-up with larger sample size is required for improved study validity and to improve long-term liver-related outcomes.

Keywords: Chronic hepatitis C, Direct-acting antiviral, Sofosbuvir, Velpatasvir, Quality of life

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INTRODUCTION

Infection with one of the five hepatotropic viral agents-hepatitis A virus (HAV), hepatitis B virus (HBV), hepatitis C virus (HCV), the HBV-associated delta agent or hepatitis D virus (HDV), and hepatitis E virus (HEV)—can cause viral hepatitis, which can result in an acute episode of liver inflammation that can either lead to the infectious agent's spontaneous clearance or can persist and, in turn, evolve into chronic infection for a subset of these viruses (>90% in Hepatitis B and 50-80% in Hepatitis C). 12-32 percent of HCC and 12-20 percent of cirrhosis in India are caused by chronic HCV infection. The level of HCV replication does not directly correspond with the severity of chronic liver disease or with the chance of developing cirrhosis or hepatocellular carcinoma; rather, the degree of intrahepatic inflammation brought on by HCV is the primary factor determining development. Most liver injuries and fibrosis stages are modest, making early identification and treatment difficult because so many individuals aren't aware they have the condition [1–4]. Histologic damage can worsen, with 20–25 percent of cases leading to cirrhosis, the most common reason for liver transplantation [5].

In the 25 y since IFN-was launched for this reason in 1991, the management of chronic hepatitis C infection has undergone significant development [6]. Candidates for antiviral therapy are those with detectable HCV RNA in the serum, regardless of aminotransferase levels and grade or stage of cirrhosis [7]. Sustained virological response (SVR) is defined as having no measurable HCV RNA in the blood following cessation of treatment. Earlier clinical trials with various IFN-based therapies in combination with ribavirin demonstrated significant, sustained virological response (SVR) rates [8]. The most recent generation of direct-acting antivirals (DAAs), a fixed-dose combination of NS5A inhibitor Veplatasvir (100 mg) (VEL) and NS5B RNA-dependent RNA polymerase inhibitor Sofosbuvir (400 mg) (SOF), approved by the FDA in June 2016 against HCV genotypes 1-6, have replaced IFN-based treatments at this time. Highly successful (>95% SVR) in noncirrhotic, cirrhotic, and treatment-experienced individuals with significant barriers to resistance and is well-tolerated [9].

Successful chronic hepatitis C antiviral therapy with DAAs has been shown to increase survival, decrease the evolution of chronic hepatitis C, lower the risk of liver failure, liver-related death, all-cause mortality, and even reverse fibrosis and even cirrhosis [1, 5, 10]. While earlier research has shown that even before virologic cure, the clinical profile (signs and symptoms); biochemical profile (LFT, PT/INR, inflammatory markers) demonstrate a rapid recovery/faster remission in comparison to earlier regimens [11]. The health-related quality of life (HRQoL) is not typically evaluated in routine clinical practice, but there is a dearth of information regarding how the patient's quality of life varies during treatment and after a successful virologic cure. The present study is being conducted to a) Determine the clinical profile, therapeutic response to sofosbuvir-velpatasvir therapy, and its impact on quality of life in chronic hepatitis C patients in real-world settings. b) Identify and document treatment-related adverse events that occurred during the therapy.

MATERIALS AND METHODS

Study procedure

After receiving approval from the institutional ethical committee, the study was carried out at the Liver Clinic in the Department of Medicine and the Department of Pharmacology at the Netaji Subhash Chandra Bose Medical College in Jabalpur, Madhya Pradesh. After receiving approval from the institutional ethical committee (No. *IEC/2022/8629-145*), the study was carried out at the Liver Clinic in the Department of Medicine and the Department of Pharmacology at the Netaji Subhash Chandra Bose Medical College in Jabalpur, Madhya Pradesh

Study design

Patients were invited to take part in this prospective, observational, descriptive, and longitudinal study if they began treatment with the SOF/VEL regimen (manufactured by Hetero Labs Limited, Unit-II, Kalyanpur, Himachal Pradesh, India) between January 2021 and March 2022 and had detectable HCV RNA on a quantitative

immunoassay. Patients who did not give informed consent had a history of liver transplantation, or had been identified as co-infected with HIV and hepatitis B were not included in the study. During patient enrolment and registration, the clinical and sociodemographic information (age, gender, personal history, including any addictions and tattoos, medical history, including co-morbidities, history of prior surgeries or blood transfusions, and family history) was gathered. Data on serum and biochemical parameters to assess clinical profile and therapeutic response, comprising HCV RNA quantitative assay, complete blood count, liver function tests, renal function tests, and coagulation profile, were gathered at three predetermined regular intervals: at the start of treatment (baseline), at the conclusion of the therapy regimen either at 12 w after therapy initiation or when SVR with undetectable HCV RNA was achieved (EOT), and 24 w after virological response SVR was achieved (SVR24). The data was simultaneously recorded in the patient's liver clinic diary.

Study tools

The collected serum and biochemical marker readings during routine follow-ups in patients were used to calculate changes in the non-invasive indicators of liver fibrosis (FIB4 and AST to Platelet Ratio Index (APRI)) and to categorise (presence/absence of cirrhosis) them. FIB-4 has been used to compare how liver fibrosis develops in HCV patients and to track changes in the stage of liver fibrosis over time. In order to prevent LB testing, patients with moderate or considerable fibrosis can be correctly identified with scores of 1.45 and>3.25, respectively. APRI can accurately predict severe fibrosis and cirrhosis in CHC patients who have not yet received treatment. The proposed APRI cut-off values for bridging fibrosis and cirrhosis are>1.5 and>2, respectively. Two validated and Hindi language translated versions of the Quality of Life (QoL) questionnaires were utilised: a) a 36-point Short Form Health Survey (SF-36) score relating to health that represents the average of physical and mental domains; and b) a disease-specific Chronic Liver Disease Questionnaire-Hepatitis C Version (CLDQ-HCV). Average scores from both questionnaires assessed the effect of virologic cure on the quality of life in chronic hepatitis C patients in the presence or absence of concomitant cirrhosis or comorbidities prior to therapy. Following appropriate patient counselling, self-reported data from both questionnaires were collected during routine follow-ups simultaneously after serum and biochemical tests.

Data management and statistical analysis

With the aid of the computer programme MS Excel, information on clinical and sociodemographic characteristics was stored, separated, and processed into the following categories: a) Age group (50,>50 y); b) Gender; c) Co-morbidities; and d) presence/absence of cirrhosis, defined from non-invasive liver indices scores at baseline. At EOT and SVR24 following baseline, data on mean serum and biochemical parameters of CBC (haemoglobin, platelet counts), Liver and renal function profiles (AST/ALT, Total albumin, Total bilirubin, PT/INR, and Serum creatinine), Non-invasive liver indices (APRI and FIB4), and mean values of quality-of-life scores SF-36 and CLDQ-HCV were compared. HCV RNA levels were quantified by using COBAS TaqMan HCV Test, version 2.0 (Roche Molecular Systems, Inc., Branchburg, NJ), which has a lower limit of quantification of 15 IU/ml). A frequency analysis of adverse events linked to treatment was conducted. The statistical evaluations were performed using the IBM SPSS Statistics programme. A paired t-test was used to determine whether a continuous variable was significant. Continuous variables are described as the mean plus standard deviation (SD). A p-value of 0.05 was regarded as significant. The mean change in SF-36 domains (PCS/MCS) and CLDQ-HCV scores from baseline to SVR24 were independently analysed in relation to clinical (co-morbidities, cirrhosis) and demographic (age and gender) variables.

RESULTS

Only 71 individuals were started on the SOF/VEL regimen following the identification of HCV RNA using a quantitative immunoassay test after 87 HCV rapid-positive patients had successfully registered. Nine patients were further eliminated from the study during the follow-up period, leaving 62 subjects who completed the duration of the study after giving written informed permission (fig. 1).

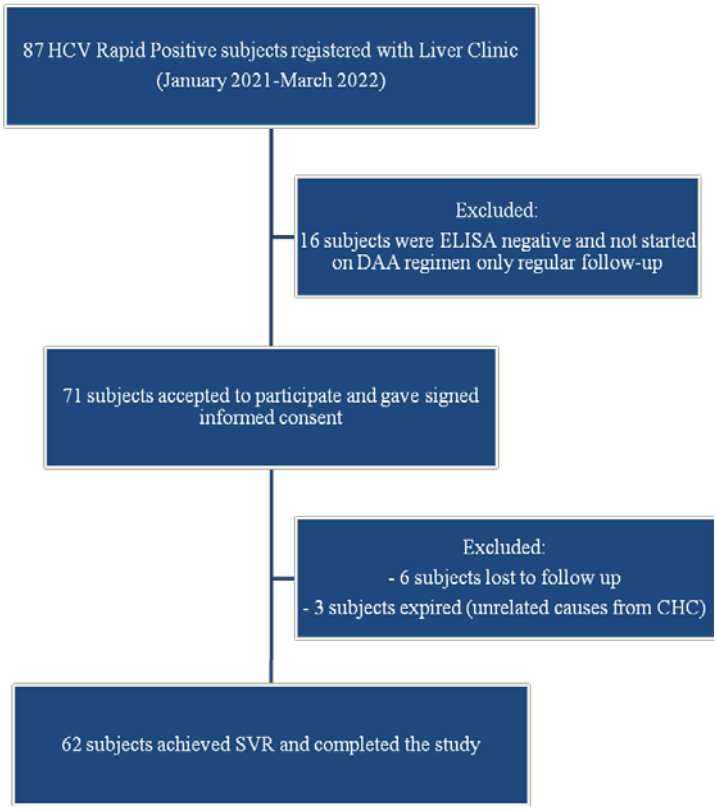

Fig. 1: Overview of study design (CHC: Chronic hepatitis C; DAA: Direct-acting antivirals; SVR: Sustained virological response)

The sociodemographic features of the patients who completed the routine follow-up revealed a mean age of 44.6±15.7 y, a gender ratio of 2:1 (71% men, 29% women), 43 percent in the over-50s, and 56 percent in the under-50s. At baseline, approximately 39% of the patients were found to have a certain degree of liver cirrhosis, and 60% had concurrent comorbidities (table 1). At 12 w (EOT) and 24 w (SVR24) following EOT, 59 (95%) and 62 (100%) of the trial participants, respectively, achieved successful virological cure with no detectable HCV RNA in the quantitative immunoassay test.

Table 1: Sociodemographic and clinical characteristics of study subjects (N=62)

Sociodemographic characteristics		Clinical characteristics	
Age in years, mean (SD)	44.6 (15.7)	Cirrhosis n (%)	
		Present	24 (38.7)
		Absent	38 (61.3)
Age group n (%)		Comorbidity n (%)	
<50 y	35 (56.5)	Present	37 (59.7)
>50 y	27(43.5)	Absent	25 (40.3)
Gender n (%)			
Male		44 (71)	
Female		18 (29)	

The table 1 describes the sociodemographic and clinical characteristics of the study cohort. (SD: Standard Deviation)

Considering the cohort's clinical and demographic sub-analysis, In the group of patients older than 50, there were 18 (48.5%) comorbidities (hypertension, diabetes mellitus type 2, chronic kidney disease, thalassemia, and sickle cell disease), of which 15 (55.5%) had cirrhosis, determined based on the baseline APRI and FIB-4 scores. When it came to the group of people under 50, 19 (51.4%) of them had comorbidities at the time of registration, and of those, 9 (25.7%) had been identified as having the presence of liver cirrhosis using non-invasive liver indicators (table 2, fig. 2).

Table 2: Distribution of clinical characteristics of the study's subjects according to their age group (N=62)

Age group	Comorbidities	Cirrhosis
<50 y	19 (51.4%)	9 25.7%)
>50 y	18 (48.6%)	15 (55.5%)
Total (n-62)	37 (59.6%)	24 (38.7%)

Table 2 illustrates the distribution frequency of clinical characteristics of the study group according to their age group of<50 y and>50 y. Out of the 37 participants, 19 participants aged<50 and 18 participants aged>50 had comorbidities. Out of the 24 individuals, 15 (>50 y old) and 9 (<50 y old) had cirrhosis that had already developed clinically.

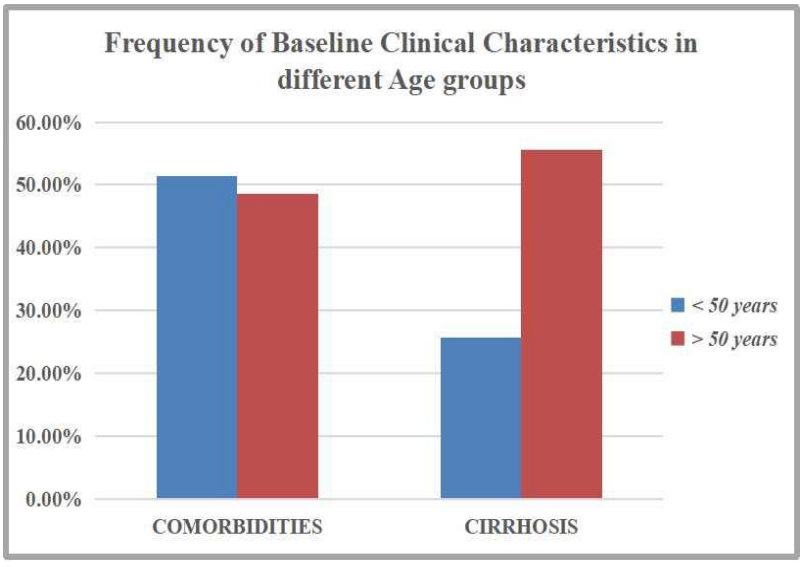

Fig. 2: Distribution of clinical characteristics according to age groups

From baseline to SVR24, there was a substantial mean change in serum and biochemical parameters, particularly hemoglobin (*p-value = 0.002), total serum albumin, and bilirubin (*p-value = 0.005), as well as serum aminotransferases ALT and AST levels (p-value = 0.005). Changes in serum creatinine (p-value 0.020), prothrombin time (p-value 0.062), and total platelet count (p-value 0.017) were not statistically significant (table 3). This was closely correlated with a significant difference between baseline and SVR24 in the mean liver indices APRI and FIB-4 scores (*p-value = 0.005) (table 4, fig. 3).

Table 3: Change in the biochemical parameters of study subjects from baseline to SVR24

Serum and biochemical parameters	Baseline	EOT (12 w)	SVR24 (24 w post EOT)	Mean change (mean±SD) baseline-SVR24	P-value
Haemoglobin (g/dl)	11.27±2.69	11.29±2.41	12.80±2.18	1.53±0.45	0.002**
Platelet count (×10^5/cumm)	2.05±1.15	2.28±0.81	2.40±0.79	0.35±0.36	0.017
Total Bilirubin (mg/dl)	1.15±0.93	0.87±0.49	0.75±0.31	0.44±0.62	<0.005*
Serum Albumin (g/l)	3.62±0.74	4.13±0.74	4.24±0.71	0.62±0.03	<0.005*
Pro-Thrombin Time (sec)	1.34±0.39	1.32±0.27	1.25±0.23	0.09±0.16	0.062
Alanine aminotransferase (U/l)	65.22±53.1	44±20.4	31.68±11.9	33.54±41.2	0.001**
Aspartate aminotransferase (U/l)	66.90±53.4	45.62±18.7	35.32±11.7	31.58±41.70	0.001**
Serum Creatinine (mg/dl)	2.91±3.58	2.52±3.08	2.53±3.26	0.38±0.32	0.020

Table 3 illustrates the mean values of biochemical parameters observed at Baseline, EOT, and SVR24. Mean change with SD and statistical significance from Baseline to SVR24.

Table 4: Change in the liver indices of study subjects from baseline to SVR24

Liver indices	Baseline	EOT	SVR24	Mean change mean±SD	P value
APRI Score	1.17±1.59	0.52±0.28	0.39±0.22	0.78±1.37	<0.005*
FIB-4 Score	2.69±2.77	1.50±0.87	1.18±0.89	1.51±1.88	<0.005*

Table 4 illustrates the mean values of two non-invasive liver indices AST to Platelet Ratio Index (APRI) and FIB-4 at Baseline, EOT, and SVR24. Mean change and statistical difference from Baseline to SVR24.

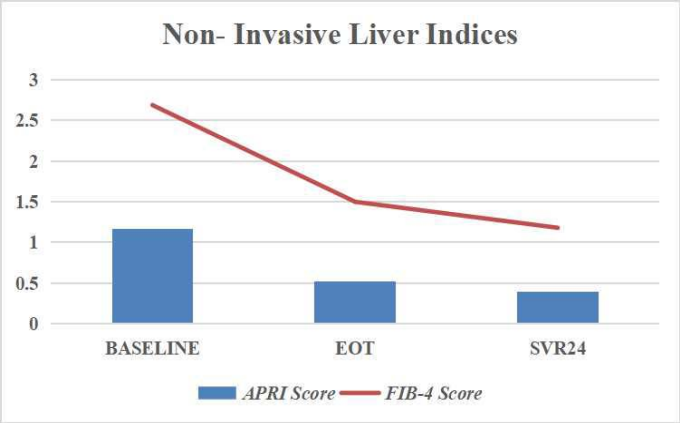

Fig. 3: Change in the liver indices of study subjects from baseline to SVR24

Since the start of therapy until SVR24, there has been a significant improvement in the quality-of-life parameters, with mean changes in both physical and mental domains of the SF-36 questionnaire (table 5, fig. 4) and vitality and emotional components of the CLD-HCV questionnaire (table 6, fig. 5) being observed at EOT before gradually plateauing at SVR24. There was also a highly significant (**p-value 0.001) statistical change when the mean SF-36 domain scores and CLDQ-HCV values were independently analysed for gender (male/female), age group (50 y and>50 y), co-morbidities, and cirrhosis (present/absent). Additionally, the overall SF-36 score continued to rise at SVR24 (table 7).

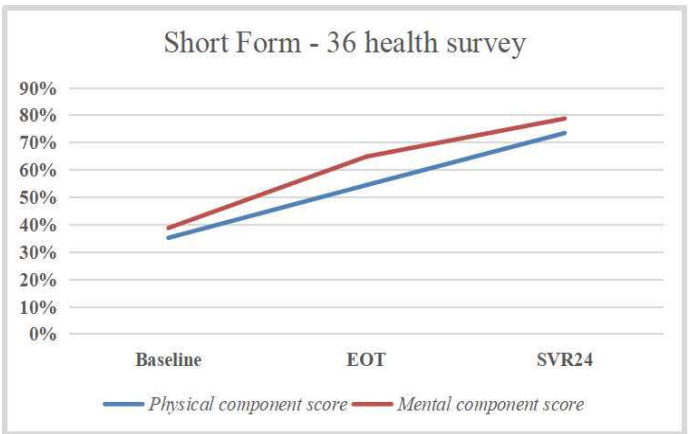

Fig. 4: Change in short-form health survey (SF-36) domains of study subjects from baseline to SVR24

Table 5: Change in short-form health survey (SF-36) domains of study subject from baseline to SVR24

SF-36 health survey	Baseline	EOT	SVR24	Mean change (mean±SD)	P-value
Physical component score	35.2%±13.9	54.5±16.6	73.5±14.8	38.3±0.9	<0.001**
Mental component score	38.8%±13.5	64.9±16.2	78.8±14	40±0.5	<0.001**

Table 5 shows the mean values of the two SF-36 domains that were assessed at the baseline visit, during the EOT, and 24 w after the EOT (SVR24); as well as the mean change and statistical significance from Baseline to SVR24.

Table 6: Change in the chronic liver disease questionnaire–hepatitis c version (CLDQ-HCV) scores of study subjects from baseline to SVR24

CLDQ-HCV				
Baseline	**EOT**	**SVR24**	**Mean change (mean±SD)**	**P-value**
4.39±0.26	6.00±0.16	6.61±0.11	2.22±0.15	<0.001**

Table 6 illustrates the mean values of the CLDQ-HCV scores that were observed during baseline, EOT, and 24 w post-EOT. The mean change and statistical significance from Baseline to SVR24.

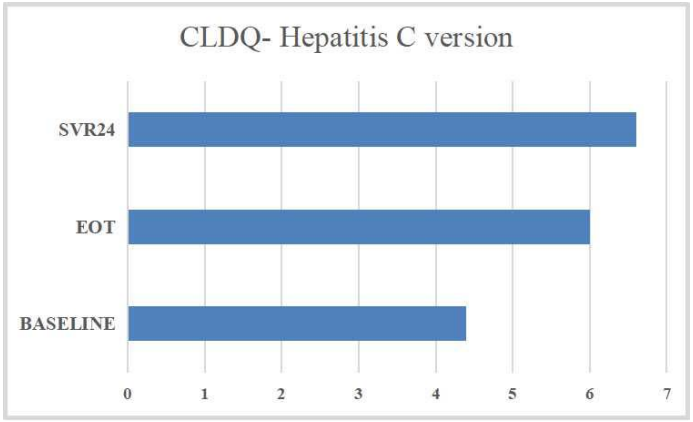

Fig. 5: Change in the chronic liver disease questionnaire–hepatitis C version (CLDQ-HCV) scores of study subjects from baseline to SVR24

Table 7: Sub-analysis of change in SF-36 and CLDQ-HCV values from baseline to SVR24

Quality of life domains *Vs.* Clinical and demographic characteristics	SF-36							CLDQ–HCV			
	Baseline		SVR24		Mean change		P-value	baseline	SVR24	Mean change	P-value
	PCS	*MCS*	*PCS*	*MCS*	*PCS*	*MCS*					
Gender											
Male	36.2	39.6	75	79.3	38.7	39.7	<0.001	4.3	6.6	2.2	<0.001
Female	32.7	36.8	70.3	77.4	37.2	40.6	<0.001	4.4	6.5	2.1	<0.001
Age											
<50 Y	38.6	40.3	76.8	81.1	38.1	40.7	<0.001	4.4	6.6	2.2	<0.001
>50 Y	30.8	36.8	69.3	75.8	38.5	39	<0.001	4.3	6.5	2.2	<0.001
Cirrhosis											
Present	35.7	43.4	76.1	82.5	40.3	39.1	<0.001	4.3	6.6	2.3	<0.001
Absent	35.2	37	72.2	77.2	36.9	40.1	<0.001	4.4	6.6	2.2	<0.001
Comorbidities											
Present	33.4	38.6	73.8	77.2	40.3	38.5	<0.001	4.4	6.6	2.2	<0.001
Absent	37.8	39.1	73.2	81.2	35.3	42	<0.001	4.3	6.5	2.2	<0.001

Table 7 Independent analysis of the change in the mean QoL scores for the PCS and MCS domains of the SF-36 and the CLDQ-HCV from baseline to SVR24, broken down according to gender, age group, cirrhosis, and comorbidities. From Baseline to SVR24, the mean change and statistical significance.

Headache and Fatigue (25%), Nausea (33%), and Anorexia (16%) were identified and recorded in 12 study subjects and appropriately managed after consulting with a physician in medicine OPD (table 8, fig. 6).

Table 8: Type and frequency of adverse drug reactions observed in the study group

Adverse drug reaction	Frequency
Nausea	4 (33%)
Fatigue	3 (25%)
Anorexia	2 (16%)
Headache	3 (25%)
Total (N=62)	12 (19%)

Table 8 illustrates the different types of drug-related adverse events and the frequency in which they were reported during the duration of the study by the study subjects.

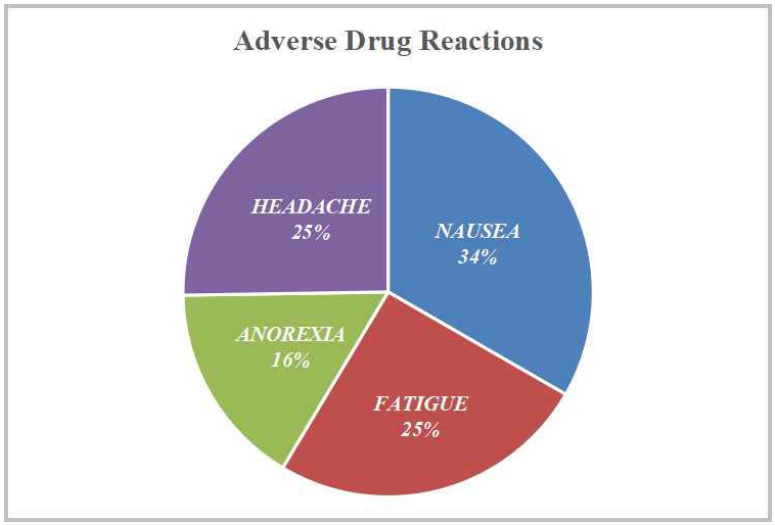

Fig. 6: Frequency of adverse drug reactions observed in the study group

DISCUSSION

We evaluated the improvements in clinical, virologic, and biochemical markers during the course of a 62-patient chronic hepatitis C follow-up study that was conducted up to six months after SVR after a 12-week SOF/VEL regimen. The main objective of the current study was to identify the advantages of reaching SVR and to compile the data to examine how it impacts the clinical and biochemical markers in these patients and translates to enhanced long-term quality of life and clinical outcomes. It is safe and effective to treat all phases of liver disease, including decompensated cirrhosis, with the SOF/VEL regimen in naive non-cirrhotic patients, with SVR rates up to 99 percent in all genotypes. Following 24-48 w of therapy, patients with SVR are typically regarded as permanently cured [12, 13]. According to our study, patients who received the SOF/VEL regimen for at least 12 w (EOT) had a 95% SVR rate (59/62; 95% CI 87%-100%) and 100% after 24 w. Three participants who had HCV RNA detectable (<34 IU/ml) at 12 w underwent an additional 4 w of therapy before the target HCV was undetectable. This variation can be attributable to the degree of liver disease, the presence or absence of concomitant conditions, and genotypic differences that are significant to the clinical presentation and therapeutic response [14].

It is well documented that, barring the presence of another liver illness, the majority of patients with an SVR quickly return to normal levels of serum ALT, AST, platelet count, and total bilirubin after stopping medication [15, 16]. In our study, AST, ALT, and PLT levels all showed overall lasting improvements after successful chronic HCV treatment, with the exception of one patient (70 y old) who continued to have persistently elevated ALT levels. After undergoing a liver biopsy, the patient was found to have grade II/IIIA HCC, confirming the significant risk of primary liver cancer in chronic cirrhosis patients, which rises with older age and the presence of diabetes mellitus [17]. Cirrhotic patients did not exhibit a statistically significant change in bilirubin, prothrombin, or platelet values, in addition to an increase in white blood cell count and a rise in serum albumin. The current study's findings suggest that the decrease in serum biomarker values may not always indicate a reversal of histologic fibrosis [18].

From baseline to SVR24, the mean change in the APRI and FIB-4 values of our study group was significant (*p-value 0.005) and was linked with the effective suppression of viral replication at EOT. Our results demonstrated that patients who responded well to treatment displayed a considerable reduction in FIB4 and APRI trajectories, suggesting stability in markers of liver disease or perhaps fibrotic retreat. Although the exact mechanism is unknown, viral replication inhibition may lessen hepatic inflammation and the ensuing fibrosis, enabling the regeneration of healthy tissue. These results offer additional support for the usefulness of repeated assessments of non-invasive liver indices in HCV-infected individuals and show a relationship between improving serum indicators and a declining fibrosis stage [19–21].

The SF-36 is an instrument that is most frequently used in HRQoL studies, and studies that used it almost universally demonstrated that patients with SVR had higher scores than non-responder/relapser/untreated populations, both in terms of sub-domains and physical and mental component summary scores, with a significant portion of between-group differences [22–24]. Between baseline and SVR24, there was a significant shift in the mean values of our study group for both the SF-36 PCS and MCS domains (*p-value 0.005). The mental component of the domain was found to be progressively plateauing after the end of therapy until SVR24 and could be determined to be influenced by the treatment outcome in the form of successful viral replication suppression. More specifically, its maintenance after the end of the treatment proves to be a significant indicator of the observed changes. The Chronic Liver Disease Questionnaire (CLDQ), a well-known disease-specific tool, contains 29 items with seven-point scale response options to assess HRQoL in patients with CLD regardless of the a etiology and severity. Higher scores indicate better HRQoL. The CLDQ appears to be more responsive than the standard SF-36, as evidenced by the strong correlation between CLDQ scores and clinical indications of deterioration [25, 26]. Our findings of a significant (**p-value 0.001) change in the mean CLDQ score from baseline to SVR24 describe a positive correlation of this change with improved mental and emotional function with fewer abdominal symptoms after therapy with increased vitality and less worry about chronic diseases at SVR24.

The validity of our data is considerably increased when these measures are used in combination since they cover the effects of therapy on the patient's physical and mental health as well as their unique ailment. In our trial, QoL significantly improved in patients who achieved SVR12 as early as 2 w into treatment, and this improvement persisted during lengthier follow-ups of up to 24 w following treatment termination. A significant increase in the quality of life and well-being of patients is confirmation of the high efficacy and perhaps better adherence to treatment regimens.

We conducted a separate analysis of mean changes in the SF-36 and CLDQ domain scores from baseline to SVR24 in relation to the clinical (comorbidities and cirrhosis) and demographic (age and gender) features of the study population. In the demographic characteristics, female participants consistently had higher mean mental domain scores than male respondents, who had higher mean physical domain scores. This merits additional research and may be explained by the different psychophysiological components in each gender. The CLDQ scores for each gender are roughly equal. The mean change in the SF-36's mental component was greater in the

age group under 50 than in the age group over 50. The CLDQ scores between the two groups don't differ all that much. The rise in CLDQ domains was no longer substantially different across the cirrhosis groups after examining the clinical characteristics of the cohort. It was shown that there was a strong correlation between the physical domain score of the SF-36 and cirrhotic patients. However, it's also probable that part of these improvements can be due to the early improvement in liver function that largely affects decompensated cirrhotics. In comparison to subjects without pre-existing comorbidities, those with pre-existing comorbidities had a larger mean change in the physical domain of the SF-36. Following the end of therapy, improvements in every domain gradually got better throughout the course of the 24-week follow-up. The results from both QoL questionnaires further improve our understanding of the long-term changes in quality of life based on previous studies with increasing validity [27].

This early reduction in fibrosis parameters should be taken with caution as it is primarily a reflection of the decline in necro-inflammation rather than the actual decline in fibrosis, signifying the need for careful long-term follow-up in these patients. In people who have had effective treatment, sustained virological suppression appears to cause long-term fibrosis regression [28, 29]. Our results strongly imply that patients achieving SVR with the SOF/VEL combination are likely to display a persistent regression of hepatic fibrosis based on the improvement of serum biomarkers and examination of FIB-4 and APRI trajectories spanning 1.5 y [30]. We now build on earlier studies and demonstrate that this marker may be utilised well for longitudinal analysis by combining two biomarkers for liver fibrosis [31, 32].

LIMITATIONS

The fact that all patients were participants in a clinical trial with stringent eligibility requirements, including the lack of serious hepatic comorbidities, is one of the study's drawbacks. The generalizability of the study results to the total population of patients with HCV infection in a real-world environment is also uncertain due to the limited sample size. Furthermore, we did not examine the relationship between parameter correlation and HCV genotypes, which reduces the validity of our investigation. Self-reported data on medication compliance and responses to quality-of-life questionnaires could have introduced subjectivity bias.

CONCLUSION

According to our real-world experience study, patients who clear their HCV using the SOF/VEL combo regimen exhibit a progressive improvement in their HRQoL, as measured by the SF-36 and CLDQ-HCV. Improvements in quality of life that were made at 12 w persisted or got better after 6 mo of follow-up. Following the successful eradication of HCV, improvements in the clinical profile of the patients with a decrease in symptoms were in line with a decline in the biochemical indicators of chronic liver disease. Regression of liver fibrosis and cirrhosis is indicated by a decline in liver indices, which results in favourable clinical outcomes and successful treatment response, including a decrease in mortality from chronic liver disease, primarily from end-stage liver disease and hepatocellular carcinoma.

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ABBREVIATION

CHC-Chronic Hepatitis C, GT-Genotype, EIA-Enzyme Immuno-Assay, SOF/VEL-Sofosbuvir-Velpatasvir, LFT-Liver Function Test, RFT-Renal Function Test, CBC-Complete Blood Count, PT/INR-Prothrombin Time/International Normalized Ratio, HCV RNA-Hepatitis C Virus Ribonucleic Acid, IFN-α-Interferon-Alpha, LB-Liver Biopsy, DAA-Direct-Acting Antiviral, SVR-Sustained Virological Response, HCC-Hepato-Cellular Carcinoma, APRI-AST to Platelet Ratio Index, ULN-Upper Limit of Normal, EOT-End of Treatment, SVR24-Sustained Virological Response at 24 w post-EOT, HRQoL-Health Related Quality of Life.

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AUTHORS CONTRIBUTIONS

All the authors have contributed equally.

CONFLICT OF INTERESTS

Declared none

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