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A Prospective Observational Study on Outcomes of Hepatitis B, Hepatitis C Patients in Gastroenterology Department of Tertiary Care Hospital

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ABSTRACT

Oral direct-acting antiviral (DAA) therapies have significantly improved the treatment outcomes for chronic hepatitis C virus (HCV) infection, achieving sustained virological response (SVR) rates exceeding 95%. However, co-infection with hepatitis B virus (HBV) and HCV presents greater clinical complexity than mono-infection with either virus. In this study, we evaluated the incidence of HBV reactivation during and three months post-DAA therapy, as well as the SVR rate at 12 weeks in patients with HBV/HCV co-infection. Among the study cohort, 361 patients (95.5%) achieved SVR. Factors associated with failure to achieve SVR included older age, lower platelet count, elevated blood creatinine levels, and increased liver stiffness measured by FibroScan. HBV reactivation occurred in 13 patients (4%), who had significantly higher baseline levels of ALT, serum creatinine, and HCV RNA viral load compared to those without reactivation. While DAA therapy is highly effective in achieving SVR in HBV/HCV co-infected patients, careful monitoring is essential due to the risk of HBV reactivation during and after treatment.

Keywords: *HCV, Treatment, Direct-acting antivirals (DAAs), HBV, Viral load*

1. INTRODUCTION

Viral infections like hepatitis B and C are one of the main causes of chronic liver disease worldwide. Over 250 million individuals are currently infected with the hepatitis B virus (HBV), and over 70 million are infected with the hepatitis C virus (HCV), according to the World Health Organization [1]. HBV and HCV have fundamentally different life cycles, despite the fact that they both like to replicate in hepatocytes. Unlike HCV, which only replicates in the cytoplasm of hepatocytes, HBV is a DNA virus that does so in the nucleus. Though theoretically able to interact in co-infected cells, both viruses contain RNA replicative intermediates, which can change viral expression and serologic patterns [2]. The definition of coinfection is the coexistence of two or more reproducing organisms in a single host. There are two ways that HBV and HCV can co-infect people. Viral co-transmission is possible since HBV and HCV share several common

routes of transmission, including intravenous drug use, blood transfusion, and vertical transfer [3]. However, superinfection, where one virus is acquired in a patient who already has a chronic infection with the other virus, can also result in HCV/HBV coinfection. The most frequent cause of coinfection is superinfection, and HCV superinfection is more frequent than HBV superinfection [4]. With sustained virological response (SVR) rates of > 95% for patients with HCV monoinfection, recent developments in all-oral direct-acting antiviral (DAA) regimens for HCV have significantly enhanced therapy efficacy. Unfortunately, it happens frequently and can result in death when HBV reactivation or HBV-related clinical reactivation occurs during or after DAA therapy. Uncertainty persists regarding the longer-term clinical outcome of HBV among patients with HBV/HCV coinfection following DAAs [5, 6]. The purpose of this study was to assess the rates of SVR in patients with HCV/HBV coinfection 12 weeks after therapy with DAAs and the risk of HBV reactivation both during and 6 months after treatment.

2. METHODS

Relevant information was gathered from patient prescriptions and case sheets in the hospital's gastroenterology departments in IP and OP. Medication records for patients from the Amaravathi Institute of Medical Sciences in Guntur, Andhra Pradesh, were evaluated. The investigation was conducted over the course of three months. (August 2021–October 2021) is the study's time frame. The following criteria will be taken into account when conducting the study: those who had HBV or HCV were included in the trial while those who did not have HBV or HCV were not. The medical records of the patients were examined, and information was gathered regarding their medical histories, physical examination findings, and the outcomes of the following laboratory tests: total bilirubin, albumin (Alb), alanine aminotransferase (ALT), and aspartate aminotransferase (AST). The hemoglobin level (Hb), hematocrit (HCT), white blood cell (WBCs), red blood cell (RBCs), and platelet count (PLT) were all measured as part of the complete blood count.

In order to detect the measured levels of HBV-DNA and HCV-RNA, respectively, quantitative polymerase chain reactions (PCR) were carried out. According to technical guidelines, fibroscan measured liver stiffness values in kilopascals (kPa) and expressed them in accordance with the liver's stiffness. DAAs were treated for 12 weeks with sofosbuvir and daclatasvir, either with or without ribavirin. HBV infection monitoring. After the end of the treatment, all patients were routinely followed up for 24 weeks [4].

Statistical analysis

SPSS version 22 was used for data entry, coding, and analysis. All obtained variables had proper descriptive statistics conducted on them. Analyses of variance and paired t tests were used to compare the quantitative variables. Chi-square and Fisher's exact tests were used to compare qualitative variables.

3. RESULTS

Among the 361 patients with HCV/HBV coinfection who received DAAs for 12 weeks, 361 (95.5%) achieved SVR following treatment, among them 242 males and 119 females.

Table 1: Laboratory investigation in the studied patient in relation to SVR to DAAs

Laboratory Investigations	Mean ±SD
FBS (Mg/dl)	94.32±1.02
HB (g/dl)	12.3±1.11
WBC ($\times 10^3$)	7.42±1.04
PLT ($\times 10^3$)	196.58±56.84
ALT (U/L)	34.3±11.8
AST (U/L)	38.6±17.92
Albumin (g/dl)	3.42±0.22
Creatinine (Mg/dl)	0.67±0.09
INR	1.02±0.1
PC (%)	89.19±7.77
Total bilirubin (Mg/dl)	0.62 ±0.25
AFP (ng/ml)	4.25 ± 1.71

Liver stiffness (Kpa)	8.11 ±2.54

In addition, while there was no significant difference between groups in the levels of Hb, WBC, ALT, AST, international normalized ratio (INR), prothrombin concentration (PC), total or direct bilirubin, or AFP, 31.9% of patients who achieved SVR had splenomegaly, compared to a rate of 88.9% in patients who did not achieve SVR (p 0.001).

Table 2: Laboratory investigations to PCR for HBV DNA after end of HCV treatment

Laboratory Investigations	Patients without HBV reactivation after HCV treatment (N=361)	P value
FBS (Mg/dl)	96.74±26	0.46
HB (g/dl)	12.98±1.1	0.33
WBC ($\times 10^3$)	7.24±1.04	0.06
PLT (× 10)	168.2±59.3	0.53
ALT (U/L)	34.43±10.81	**0.049**
AST (U/L)	39.16±18.18	0.73
Albumin (g/dl)	3.19±0.22	0.16
Creatinine (Mg/dl)	0.74±0.09	**0.042**
INR	1.11±0.1	0.21
PC (%)	92.09±7.62	0.41
Total bilirubin (Mg/dl)	0.55±0.25	0.98
AFP (ng/ml)	4.64±1.63	0.86
Liver stiffness (Kpa)	8.87±3.46	0.07
HCV RNA (IU/Ml)	$1.15\times10^5\pm2.34\times10^5$	**0.047**

After HCV treatment, 361 patients did not experience HBV reactivation. There was a significant increase in the proportion of males from the group without HBV reactivation to the group with it (p = 0.045). Additionally, HCV RNA levels were greater at baseline in individuals with HBV reactivation compared to patients without HBV reactivation (p = 0.047). Hb, WBC, PLT, AST, serum albumin, INR, PC, bilirubin total and direct, AFP, and splenomegaly were not significantly different between the two groups.

Table 3: Univariate regression analysis of laboratory investigations

Parameters	Odds Ratio	SD	P value
Age /year	1.232	1.855	**0.034**
Gender	1.542	1.998	0.132
FBS (mg/dl)	2.132	2.870	0.212
HB (gm/dl)	0.988	1.211	0.453
WBCs (10^3 U/L)	1.211	1.956	0.766
PLT (10^3 /L)	3.211	4.776	0.343
ALT(U/L)	2.377	3.376	**0.045**
AST(U/L)	1.966	2.656	0.119

PC (%)	0.989	1.233	0.234
INR	1.355	1.788	0.256
Albumin(G/L)	2.133	2.768	0.311
Creatinine(mg/dl)	1.989	2.450	0.066
Fibroscan (Kpa)	3.420	5.144	0.112
Splenomegaly	2.188	2.988	0.781
HCV treatment	4,343	5.321	<**0.001**

In addition, univariate regression analysis was carried out for the purpose of predicting HBV reactivation. According to the results of this analysis, age, ALT, the level of HCV RNA present at baseline, and the length of time spent on HCV therapy were all related with an increased risk of HBV reactivation.

4. DISCUSSION

In the present study, we assessed the rate of HBV reactivation in patients with HCV/HBV coinfection after HCV treatment; only 13 patients (4%), after treatment for chronic HCV infection, suffered HBV reactivation. There were no reported deaths or flare-ups. Omar et al. [7] listed a number of variables that can affect genotype 4 patients' SVR12. Gender, bilirubin, albumin, INR, and PLT were some of them. Patients who attained SVR12 were discovered to be younger, mostly female, less likely to have diabetes, had lower baseline levels of ALT, AST, and bilirubin, and have higher baseline levels of albumin, Hb, WBC, and PLT. In China, HBV endemic regions, Wang et al.'s [8] investigation of 327 patients taking oral DAA medicines for HCV infections. 124 patients had occult HBV infection, and 10 patients tested positive for HBsAg. Serial serum samples were taken every two weeks while receiving DAA medication and subsequently every four weeks until week 12 after treatment to assess HBV reactivation. 10 individuals (3.1% of the overall study group) had hepatitis, and 3 of those instances had HBV reactivation. Soliman et al. [9] observed SVR rates of 96.29% and 84.61%, respectively, in cirrhotic and noncirrhotic patients ($p = 0.002$). Patients with and without HBV reactivation had significantly different baseline levels of ALT, serum creatinine, and HCV RNA in the current study. However, there was no discernible difference between the two groups in terms of the Hb level, WBC, PLT, AST, serum albumin, INR, PC, bilirubin total and direct, AFP, or presence of splenomegaly. Our findings were in line with those of Kawagishi et al. [10], who compared the risk of HBV reactivation following treatment for HCV with interferon (IFN)-based therapy (72 patients) versus DAA (85 patients). They found that six patients had HBV reactivation ($n = 2$) or reappearance ($n = 4$) after IFN-free DAA therapies, but none had it after IFN-based therapies. Patients with and without HBV reactivation or reappearance did not significantly differ in terms of age or sex ($p = 0.55$ and 0.13, respectively).

Additionally, at 12 weeks after treatment, however, in a previous study by El Kassas et al. [11] on chronic HCV patients with positive HBsAg who had DAAs in Egypt had a risk of reactivation in the absence of HBV treatment that ranged from 15.6 to 46.4% (95% confidence interval [CI]), and the risk of hepatitis in patients who experienced reactivation ranged from 10.0% to 57.8% (95% CI). On the other hand, it is believed that HBV reactivation can be triggered when inhibition of HBV replication is lost. These medications directly decrease the immune response to HBV replication, which results in HBV reactivation in cases where an immunosuppressive agent is provided. Patients who are infected with both HBV and HCV typically have HCV as the dominant virus, and it is believed that the presence of HCV suppresses the replication of HBV. Therefore, the elimination of HCV may render the control of HBV replication useless, leading to the revival of HBV. Doi et al. [12] investigated the prevalence of and risk factors for HBV reactivation in HCV patients receiving all-oral DAA therapy. They found that 3.4% (5/147) of patients experienced HBV reactivation. In the current study, there was a significant difference in FBS, PLT, serum albumin, serum creatinine, and fibroscan between patients who did and did not reach SVR. On the other hand, there was no significant difference between the groups in terms of WBC, ALT, AST, INR, PC, bilirubin total and direct, or AFP. Patients who were able to reach SVR had lower blood glucose levels, lower serum creatinine levels, and lower values for the fibroscan evaluation of liver stiffness. Patients who were able to achieve SVR also had greater albumin levels and PLT counts. In addition, splenomegaly was present in 31.9% of patients who went on to achieve SVR [13]. The findings that Shousha and colleagues reported were consistent with our own.

Age, gender, PLT count, serum creatinine, fibroscan, and the presence of splenomegaly were all found to be linked with SVR in the present study by univariate regression analysis. Univariate analysis found no associations between any of these factors and virological reactivation of HBV. To treat HCV/HBV coinfection, DAAs have shown a high SVR and are finally becoming broadly available. The risk of HBV reactivation during and after DAA treatment should, however, be kept in mind.

5. CONCLUSION

Our results showed that DAA-treated patients with HCV/HBV coinfection had a very high SVR (95.5%); however, factors such as advanced age, low PLT count, high serum creatinine, and increased liver stiffness were regarded to be predictors for failure to attain SVR. Only the HCV RNA level at baseline and the length of HCV treatment were substantially linked with the probability of HBV reactivation; 13 patients (4%) suffered HBV reactivation, and these patients had significantly higher ALT and serum creatinine levels.

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ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Written informed consent was signed by study participant. The study approval was taken from the Institutional Ethical Committee (IEC), Amaravathi Institute of Medical Sciences, Amaravathi Hospital, Guntur, with Ethical Committee number —IEC/2023/07/AIMS/D

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Conflict of Interest – Nil

Authors contribution

RK & AK contributed to the idea of the study and work proposal and supervision; ST collected the patient data, consent for the study, documentation and writing the manuscript. “All authors read and approved the manuscript.”

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