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STUDY THE EFFECTIVENESS OF ANTIVIRAL TREATMENTS IN PREVENTING EPISODES OF HEPATIC DECOMPENSATION IN CHRONIC HEPATITIS PATIENTS WITH DECOMPENSATED CHRONIC LIVER DISEASE

Dr Amina Saliha[1*], Dr Shanza Zafar[2], Dr Aatka Rauf[3], Dr Aiza Rao[4], Dr Aasma Ismail[5], Dr Palwasha Shabbir[6]

[1*,2,3,5]House Officer, CMH, Multan, Pakistan
[4]House Officer, Shalamar Hospital, Lahore, Pakistan
[6]House Officer, CMH, Karachi, Pakistan

[*]**Corresponding author:** Dr Amina Saliha
*Email: salihaamina467@gmail.com

ABSTRACT

Background: Chronic hepatitis B and decompensated chronic liver disease (DCLD) are significant medical conditions worldwide, with a tendency to cause hepatic decompensation in most cases. Antiviral therapy is a crucial intervention in hepatic decompensation and enhancement in prognosis in patients with CHB and DCLD.

Objective: To determine the effectiveness of antiviral therapy in preventing hepatic decompensation in patients with chronic hepatitis B and decompensated cirrhosis.

Study Design: Quasi-experimental study.

Duration and Place of Study: The study was conducted between August 2023 and June 2024 at the Department of Gastroenterology.

Methodology: A total of 191 patients aged ≥18 years with chronic hepatitis B and decompensated cirrhosis were enrolled. Patients received either Entecavir (ETV) or Tenofovir (TDF) as antiviral therapy. Efficacy was defined by the prevention of hepatic decompensation episodes, including ascites, variceal bleeding, hepatic encephalopathy, and spontaneous bacterial peritonitis (SBP). Data on demographic, clinical, and laboratory parameters were recorded.

Results: The study found that antiviral treatment was effective in preventing hepatic decompensation in 60.2% of patients. Tenofovir demonstrated superior efficacy (76.4%) compared to Entecavir (53.7%) (p=0.004). Significant predictors of efficacy included INR levels (Exp(B)=0.208, p=0.014) and the type of treatment (Tenofovir vs Entecavir) (Exp(B)=2.432, p=0.022).

Conclusion: Antiviral therapy, and Tenofovir in particular, can effectively reduce hepatic decompensation in cirrhotic and HBV-related chronic hepatitis B infection patients. INR is a key predictive indicator for successful therapy.

Keywords: Chronic hepatitis B, Decompensated cirrhosis, Hepatic decompensation, Antiviral therapy, Tenofovir, Entecavir.

INTRODUCTION

Chronic hepatitis with decompensated chronic liver disease (DCLD) carries significant medical complications with a disease whose progression can compromise its function to a significant level, most commonly a consequence of long-standing infection and cirrhosis secondary to hepatitis C and B viruses.[1] Patients with DCLD present with symptoms such as hepatic failure, variceal hemorrhage, hepatic encephalopathy, and jaundice, and diminished quality of life.[2] Advancement in hepatic injury in such subjects predisposes them towards severe complications such as hepatic decompensation, in which the liver fails to maintain homeostasis. Therapy in such a group of subjects involves a multidimensional therapeutic approach, not only for the causative virus but its complications of cirrhotic disease as well.[3]

Management of DCLD in chronic hepatitis has evolved over a period of time, with a direction towards arresting disease progression and controlling complications.[4] Traditional therapies include lifestyle modifications, nutritional care, and drugs for controlling individual symptoms such as diuretics for ascites and lactulose for hepatic encephalopathy.[5] In extreme cases, a definitive remedy in terms of a liver transplant can be planned. All such therapies, nevertheless, control symptoms and not the etiological cause of disease, i.e., a persistent infection with a virus and a continuous hepatic injury. With ongoing development in studies, growing emphasis is placed on therapies with direct inhibition of the virus and, possibly, reversal of a proportion of hepatic injury, with hope for an enhanced prognosis in such a high-risk subgroup of cases.[6]

Antiviral treatments have become a cornerstone in controlling chronic hepatitis, most specifically for DCLD patients. Antiviral drugs inhibit and target virus replication, and consequently, reduce the inflammatory reaction that is responsible for cirrhosis and fibrosis in the liver.[7] For instance, HBV is effectively inhibited with tenofovir and entecavir, a family of nucleoside analogs, and direct-acting antiviral (DAA) drugs have become a breakthrough in HCV therapy with sustained virologic response in over 90%.[8] By acting at specific stages of a virus life cycle, not only is viral burden reduced, but immune-related damage to hepatocytes is reduced as well. Antiviral drugs, therefore, become a highly effective tool in stabilizing hepatic function and arresting deterioration in cases of chronic hepatitis and DCLD.

In the prevention of hepatic decompensation, antiviral therapy is an important intervention in improving prognosis for a patient with DCLD and chronic hepatitis.[9] By suppressing viral activity, such therapies allow a break in the continuous attack of the virus on the liver, allowing it an opportunity to regain and possibly reacquire functional capabilities.[10] Successful suppression of the virus in trials has been observed to coincide with fewer complications such as variceal hemorrhage, hepatic encephalopathy, and ascites.[11] Moreover, initiation of early antiviral therapy has been observed to coincide with preservation of liver function and delayed progression towards decompensation.[12] Restoration of function in an advanced stage of liver damage may not always be possible, but antiviral therapies form an important intervention in stabilizing a patient, enhancing survival, and overall quality of life, and therefore, their utility in overall care for a patient with DCLD and chronic hepatitis.[13]

Chronic hepatitis B remains a cause of hepatic morbidity and mortality, particularly in cases with decompensated chronic liver disease, in whom hepatic failure and decompensation have a high risk. With consideration of effective antiviral therapy in terms of tenofovir and entecavir, a real-life determination of efficacy in terms of hepatic decompositions and improvement in such a high-risk group's prognosis is warranted. Virological suppression, improvement in function of the liver, and survival information will enable maximization of management protocols, a reduced burden of transplantation of the liver, and an improvement in prognosis in such cases.

METHODOLOGY

This quasi-experimental study was conducted between August 2023 and June 2024 at the Department of Gastroenterology. A total of 191 patients with chronic hepatitis B (CHB) and decompensated chronic liver disease (DCLD) were enrolled using a non-probability consecutive

sampling technique, based on a 95% confidence level, 7% margin of error, and 58% expected efficacy[14] of antiviral treatments in preventing episodes of hepatic decompensation.

Patients of both gender aged ≥18 years with a confirmed diagnosis of chronic Hepatitis B and decompensated cirrhosis, defined as a Child-Pugh-Turcotte (CPT) score ≥7 or a history of portal hypertension complications such as variceal bleeding, hepatic encephalopathy, or ascites, were included. Patients with hepatitis C, hepatitis D, HIV co-infection, or prior solid organ transplantation were excluded. Baseline demographic data, including age, sex, liver function tests, HBV-DNA levels, CPT and MELD scores, were recorded.

All patients received Entecavir (ETV) 0.5 mg/day or tenofovir (TDF) 300 mg/day as per clinical guidelines, with adjustments for renal function if required. Efficacy was defined in terms of preventing further episodes of hepatic decompensation and overall clinical improvement in patients over a 6-month period. Preventing further episodes of hepatic decompensation was measured by the absence of new episodes of hepatic decompensation, including ascites requiring paracentesis or escalation of diuretics, variceal bleeding, hepatic encephalopathy, or spontaneous bacterial peritonitis (SBP), during follow-up. The final outcome was assessed based on clinical stability or improvement, defined by virological response (HBV-DNA undetectability), biochemical response (ALT normalization <1x upper limit of normal), liver function improvement (reduction in MELD score by ≥2 points and CPT class stabilization or improvement), and survival without liver transplantation at 6 months. Patients who developed worsening hepatic decompensation, required liver transplantation, or died due to liver-related complications within the 6-month period were categorized as treatment failures.

Data were analyzed using IBM SPSS version 27. Categorical variables were expressed as frequency and percentage, and continuous variables as mean ± standard deviation. Kaplan-Meier survival analysis was used to estimate cumulative survival rates, with a log-rank test comparing survival between different subgroups. A p-value <0.05 was considered statistically significant.

RESULTS

Patient demographics and baseline characteristics (Table-I) showed a mean age of 53.335±11.19 years, with a strong male predominance (163 males [85.3%] vs 28 females [14.7%]). Laboratory parameters showed mean HBV DNA levels of 5.229±1.71 log10 IU/mL, elevated ALT at 311.56±99.11 U/L, total bilirubin of 1.401±1.04 mg/dL, albumin of 4.086±0.54 g/dL, INR of 1.162±0.28, platelet count of 148.193±53.36 ×10^9^/L, and creatinine of 1.02±0.29 mg/dL. Disease severity was assessed using CPT Score (mean 4.79±1.69) and MELD Score (mean 10.979±4.26). The mean duration of disease was 7.34±4.61 years. Treatment history revealed that 100 patients (52.4%) had previous nucleos(t)ide analogues treatment, while 91 (47.6%) did not. Current treatment distribution showed 136 patients (71.2%) receiving Entecavir and 55 (28.8%) receiving Tenofovir.

Table- I: Patient Demographics

Demographics		**Mean ± SD / n (%)**
Age (years)		53.335±11.19
HBV DNA (log10 IU/mL)		5.229±1.71
ALT (U/L)		311.56±99.11
Total Bilirubin (mg/dL)		1.401±1.04
Albumin (g/dL)		4.086±0.54
INR		1.162±0.28
Platelet Count (×10^9/L)		148.193±53.36
Creatinine (mg/dL)		1.02±0.29
CPT Score		4.79±1.69
MELD Score		10.979±4.26
Duration of Disease (years)		7.34±4.61
Gender	Male	163 (85.3%)

	Female	28 (14.7%)
Previous Nucleos(t)ide Analogues Treatment	Yes	100 (52.4%)
	No	91 (47.6%)
Type of Treatment	Entecavir	136 (71.2%)
	Tenofovir	55 (28.8%)

Efficacy analysis (Table-II) demonstrated that antiviral treatments were effective in preventing episodes of hepatic decompensation in 115 patients (60.2%), while 76 patients (39.8%) did not achieve the desired efficacy.

Table- II: Efficacy of antiviral treatments in preventing episodes of hepatic decompensation

Efficacy	**Frequency**	**%age**
Yes	115	60.2%
No	76	39.8%
Total	191	100%

Association analysis (Table-III) revealed several important findings: Gender showed no significant association with efficacy (males: 62% effective vs females: 50% effective, p=0.232). Previous nucleos(t)ide analogues treatment also showed no significant difference (64% vs 56% efficacy, p=0.262). However, significant associations were found with type of treatment (Tenofovir: 76.4% efficacy vs Entecavir: 53.7% efficacy, p=0.004).

Table-III: Association of efficacy with demographic variables

Demographic variables		**Efficacy**		**p-value**
		Yes n(%)	**No n(%)**	
Gender	Male	101 (62%)	62 (38%)	0.232
	Female	14 (50%)	14 (50%)	
Previous Nucleos(t)ide Analogues Treatment	Yes	64 (64%)	36 (36%)	0.262
	No	51 (56%)	40 (44%)	
Type of Treatment	Entecavir	73 (53.7%)	63 (46.3%)	0.004
	Tenofovir	42 (76.4%)	13 (23.6%)	

Correlation analysis (Table-IV) revealed multiple significant relationships: Albumin showed positive correlations with age (r=0.165, p<0.05) and ALT (r=0.148, p<0.05). ALT demonstrated negative correlation with platelet count (r=-0.195, p<0.01) and positive correlation with creatinine (r=0.282, p<0.01). MELD Score showed significant negative correlations with total bilirubin (r=-0.245, p<0.01) and CPT Score (r=-0.403, p<0.01), but positive correlations with albumin (r=0.234, p<0.01) and platelet count (r=0.412, p<0.01).

Table-IV: Pearson Correlation Matrix of Clinical and Laboratory Parameters in Chronic Hepatitis B Patients

		Age	**ALT**	**Total Bilirubin**	**Albumin**	**INR**	**Platelet Count**	**Creatinine**	**CPT Score**	**MELD Score**
Age	Pearson Correlation	1	-0.020	-0.028	.165*	-0.059	0.068	-0.013	-0.015	0.102
	Sig. (2-tailed)		0.780	0.705	0.023	0.419	0.353	0.862	0.837	0.158

ALT	Pearson Correlation	-0.020	1	0.038	.148*	-0.111	-.195**	.282**	.213**	-0.043
	Sig. (2-tailed)	0.780		0.606	0.041	0.128	0.007	0.000	0.003	0.558
Total Bilirubin	Pearson Correlation	-0.028	0.038	1	-0.095	0.065	-0.067	-0.103	0.050	-.245**
	Sig. (2-tailed)	0.705	0.606		0.193	0.373	0.354	0.156	0.492	0.001
Albumin	Pearson Correlation	.165*	.148*	-0.095	1	.261**	-.185*	.191**	-0.114	.234**
	Sig. (2-tailed)	0.023	0.041	0.193		0.000	0.011	0.008	0.115	0.001
INR	Pearson Correlation	-0.059	-0.111	0.065	.261**	1	0.059	-0.045	-0.034	-0.035
	Sig. (2-tailed)	0.419	0.128	0.373	0.000		0.418	0.539	0.638	0.634
Platelet Count	Pearson Correlation	0.068	-.195**	-0.067	-.185*	0.059	1	-.255**	-0.133	.412**
	Sig. (2-tailed)	0.353	0.007	0.354	0.011	0.418		0.000	0.067	0.000
Creatinine	Pearson Correlation	-0.013	.282**	-0.103	.191**	-0.045	-.255**	1	0.137	-0.119
	Sig. (2-tailed)	0.862	0.000	0.156	0.008	0.539	0.000		0.060	0.100
CPT Score	Pearson Correlation	-0.015	.213**	0.050	-0.114	-0.034	-0.133	0.137	1	-.403**
	Sig. (2-tailed)	0.837	0.003	0.492	0.115	0.638	0.067	0.060		0.000
MELD Score	Pearson Correlation	0.102	-0.043	-.245**	.234**	-0.035	.412**	-0.119	-.403**	1
	Sig. (2-tailed)	0.158	0.558	0.001	0.001	0.634	0.000	0.100	0.000	

*. Correlation is significant at the 0.05 level (2-tailed). **. Correlation is significant at the 0.01 level (2-tailed).

The most significant predictor of treatment efficacy was INR, which showed a strong negative association with the likelihood of treatment failure (Exp(B) = 0.208, p = 0.014). This indicates that higher INR values are associated with better treatment efficacy and a reduced risk of hepatic decompensation. The Type of Treatment was also a significant factor, with antiviral treatments other than Tenofovir being associated with a substantially higher risk of treatment failure (Exp(B) = 2.432, p = 0.022). This suggests that Tenofovir may be more effective in preventing hepatic decompensation compared to other treatments.

Other variables did not show significant associations with treatment efficacy. Age (Exp(B) = 1.005, p = 0.753) did not significantly influence treatment outcomes, indicating that age does not substantially affect the efficacy of antiviral treatment. Sex (Exp(B) = 0.768, p = 0.595) also did not show a significant effect, with no meaningful difference in efficacy between male and female patients. HBV DNA levels (Exp(B) = 0.935, p = 0.511) and Previous CHB NAs Treatment (Exp(B) = 1.476, p = 0.260) were not significantly associated with efficacy, indicating that viral load and prior treatments do not appear to strongly influence the success of antiviral therapies in this population. Similarly, ALT (Exp(B) = 1.031, p = 0.497) and Total Bilirubin (Exp(B) = 0.875, p = 0.404) were not significant predictors of efficacy. Albumin (Exp(B) = 1.139, p = 0.706), Platelet Count (Exp(B) = 0.995, p = 0.265), and Creatinine (Exp(B) = 0.687, p = 0.543) showed no meaningful effect on the efficacy of treatment. While the CPT Score (Exp(B) = 3.197, p = 0.283) showed a positive but non-significant association, it did not prove to be a strong predictor. The MELD Score (Exp(B) = 0.965, p = 0.496) did not significantly impact the efficacy of antiviral treatments as shown in Table-V.

Table-V: Multivariate Logistic Regression Analysis of Factors Associated with Treatment Efficacy in Chronic Hepatitis B Patients

Variable	B	S.E.	Wald	Sig.	Exp(B)	95% C.I. for Exp(B)
Age	0.005	0.015	0.099	0.753	1.005	0.975 - 1.035
Gender	-0.263	0.495	0.283	0.595	0.768	0.291 - 2.028
HBV DNA	-0.066	0.1	0.431	0.511	0.935	0.781 - 1.119
Previous CHB NAs Treatment	0.39	0.346	1.269	0.26	1.476	0.750 - 2.908
ALT	0.031	0.002	0.461	0.497	1.031	0.999 - 1.008
Total Bilirubin	-0.134	0.161	0.697	0.404	0.875	0.635 - 1.198
Albumin	0.13	0.345	1.042	0.706	1.139	0.579 - 2.239
INR	-1.569	0.64	6.01	0.014	0.208	0.075 - 0.577
Platelet Count	-0.005	0.004	1.245	0.265	0.995	0.987 - 1.002
Creatinine	-0.375	0.616	0.37	0.543	0.687	0.285 - 1.575
CPT Score	1.161	1.212	1.152	0.283	3.197	0.718 - 14.494
MELD Score	-0.036	0.053	0.462	0.496	0.965	0.874 - 1.074
Type of Treatment (Tenofovir)	0.889	0.563	2.558	0.022	2.432	1.138 - 5.500
Duration of Disease	-0.074	0.037	3.95	0.047	0.929	0.863 - 0.997

B is the regression coefficient (log-odds), **S.E.** is the standard error of B, **Wald** is the statistic used to test significance, **Sig.** is the p-value indicating statistical significance, and **Exp(B)** is the odds ratio

DISCUSSION

This study examined the effectiveness of antiviral therapy in preventing hepatic decompensation events in cirrhotic and decompensated liver disease and in cases of chronic hepatitis B infection. In our analysis, an overall efficacy of 60.2% in hepatic decompensation prevention was observed, underlining the therapeutic significance of antiviral therapy in such a high-risk group of subjects. High efficacy of Tenofovir (76.4%) compared with Entecavir (53.7%) (p=0.004) in preventing decompensation can be understood through Tenofovir's effective suppression of viral activity and its potential for rapid HBV DNA suppression, a critical factor in preventing further deterioration in cirrhotic subjects.

Logistic regression analysis revealed that long-standing disease duration was associated with marginally lowered odds of efficacy (OR=0.929, p=0.047), possibly secondary to mounting liver impairment and portal hypertension in long-standing disease, with such cases becoming increasingly at risk for decompensation in the face of viral suppression.

The significant association between INR and therapeutic effectiveness (OR=0.208, p=0.014) holds most in the scenario of decompensated liver disease. Higher INR values, indicative of increased impairment in synthetic function, predicted less effective therapy in avoiding new episodes of decompensation. This suggests that impairment level at therapy initiation significantly influences successful prevention of future episodes of decompensation, in favor of early initiation of antiviral therapy when impairment of the liver is not yet considerable.

The complex interrelationship between tests of liver function is corroborated by correlations between MELD score and a variety of clinical tests. Negative correlation between CPT score and MELD score ($r=-0.403$, $p<0.01$) and positive correlation between platelet count and MELD score ($r=0.412$, $p<0.01$) confirm not only that severity of impairment of the liver is responsible for an increased risk of its decompensation, but for an increased probability of success in its therapy for such an episode, too.

Our study revealed that antiviral therapy successfully prevented hepatic decompensation in 60.2% of cases, with Tenofovir having a remarkably high efficacy (76.4%) over that of Entecavir (53.7%). Consistent with Miquel et al. [14] our observation conforms with that of these workers, in that, in cirrhotic, both compensated and decompensated, subjects, antiviral drugs, including Tenofovir and Entecavir, effectively restored function and survival. Nevertheless, in contrast to a head-to-head comparison between these two drugs in their work, our observation attests to the comparative superiority of Tenofovir in preventing hepatic decompensation. Consistent with Jang et al. [15] and Peng et al. [16] supporting evidence comes from these workers, who stressed early and effective antiviral therapy, including Tenofovir, in preventing complications and improving long-term prognosis in HBV-related cirrhosis.

A notable contrast in our study was that no significant relation between a background of nucleos(t)ide analogue therapy and efficacy of current therapy (p=0.262) was observed, suggesting that such a background in antiviral therapies did not have an impact on current therapy efficacy. That observation is in contrast with that of Jang et al. [15] who emphasized that a virologic response (MVR) following first antiviral therapy must be sustained in order for such therapy to have a beneficial effect. In their study, survival and reduced transplantation requirements in subjects with an MVR were observed, but a principal cause for such a contrast could have been that a larger proportion of our cohort included subjects with no background of antiviral therapy (47.6%), and hence, any role of such therapy in overall efficacy of current antiviral therapies could have been minimized to a minimum.

In terms of predictive factors at a clinical level, in our work, increased INR values correlated with increased efficacy of therapy and less hepatic decompensation, suggesting that, in general, preserved liver function (reflected in INR values) patients respond well to antiviral therapy, with increased INR values tending to represent less severe hepatic impairment. This observation is in agreement with general practice in assuming that liver function tests, including INR, represent important markers of prognosis in cirrhosis of long duration. On the other hand, studies such as performed by Jang et al. [17] and Peng et al. [16] revealed baseline MELD score and virologic response to have a strong predictive value for survival and for liver transplant-free survival, and therefore, it can be postulated that MELD

score could represent a more sensitive marker for disease severity in HBV-related cirrhotic decompensated cirrhosis.[18] Inability to detect a significant correlation between MELD score and efficacy of therapy in our work could have been a function of specific antiviral therapy utilized, or of patient population in our work.

Interestingly, our analysis did not reveal any significant age and effectiveness of treatment correlation (p=0.753), in contrast with, for example, Jang et al. [17] in whom older age was correlated with less successful survival. That no age impact in our analysis may have been a result of a relatively narrow age range in our cohort (mean age of 53.3 years), possibly not having a high enough proportion of older age groups, in whom, in general, poor outcomes in decompensated cirrhosis occur. In older age groups, comorbidities, or even general medical complications not even of a hepatic origin, could have played a larger role in affecting outcomes, in agreement with Jang et al. [17]

The findings of our study in relation to tests of liver function, such as albumin and ALT, agree with current studies, in that such tests have been found to have a relation with disease severity. In our study, albumin showed positive relations with age and ALT, and ALT showed a relation with platelet count, in a negative direction. All these observations agree with those of Miquel et al. [14] in that such markers of liver function, such as albumin, have a significant role in predicting cirrhotic patients' prognosis. Nevertheless, no predictive value in terms of efficacy in therapy in our study for ALT and for bilirubin level in total could be determined, a contrast with studies such as Jang et al. [17] in which serum ALT level showed a strong relation with survival and with transplant-free survival. Inability to detect such a relation in our study could possibly represent an expression of ALT's high sensitivity to acute, but not necessarily to disease progression in, liver impairment in cirrhotic patients with a state of decompensated cirrhosis.

Our results contribute to the growing body of evidence supporting first-line use of Tenofovir and Entecavir in antiviral therapy for HBV-related cirrhosis with decompensation.[19] As Peng et al. [16] and Jang et al. [15] have stressed, both drugs have been effective in survival and in avoiding complications, most notably when initiated early in disease progression. Similarly, Miquel et al. [14] emphasized both efficacy and safety of Tenofovir and Entecavir in real-life practice, most notably in preventing additional hepatic decompensation. What distinguishes our work, then, is its head-to-head comparison between these two drugs, and additional evidence for a relative benefit of Tenofovir in preventing hepatic decompensation events.

In summary, even though our study provides useful information about the effectiveness of antiviral therapy in preventing hepatic decompensation in cirrhotic hepatitis, it mirrors the diversity and complexity between and even within groups of patients and between regimens. Comparison with studies in current times re-emphasizes early and effective antiviral therapy, with Tenofovir, in improving prognosis in such patients. However, variation in predicting factors for effectiveness between studies suggests that baseline hepatic function and a patient's background therapy could have an impact on success with antiviral therapy in such a scenario.

Our study carries several limitations. First, it is a single institution study, and its generalizability to larger, more diverse populations is in doubt. In addition, its relatively small size can impact its statistical power and its ability to detect minor associations. What is more, its observational nature prohibits causality inference for detected relations. To confirm our observations and evaluate additional factors responsible for success with therapy, including genetic factors and long-term follow-up data, future multi-center, larger studies are indicated.

CONCLUSION

Our study concluded that antiviral therapy can effectively avert hepatic decompositions in cirrhotic and decompensated cirrhosis cases of hepatitis. Among these, Tenofovir exhibited a high level of efficacy when compared with Entecavir, and therefore, can be considered a preferred drug for such cases. Our work signifies markers of liver function, such as INR, in predicting successful therapy. All such observations contribute to an ever-growing pool of evidence in favor of powerful antiviral therapy in improving prognosis in cirrhotic, decompensated, and chronic disease.

Conflict of interest: None

Disclaimer: None

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