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Therapeutic Efficacy of Sofosbuvir Plus Velpatasvir Antiviral Therapy in Patient with Hepatitis C Virus Related Compensated Cirrhosis of Liver

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Abstract

Background: Liver cirrhosis is a critical stage of chronic liver dis¬ease. Chronic hepatitis C virus (HCV) infection is one of the most common causes of chronic liver disease worldwide. The long term impact of HCV infection may range from minimal histological changes to extensive fibrosis and cirrhosis with or without hepatocellular carcinoma (HCC).The primary goal of HCV treatment is to cure the infection. The aim of this study was to observe efficacy of sofosbuvir plus velpatasvir therapy in patient with HCV related compensated cirrhosis of liver. **Material & Methods:** The study was conducted from January, 2020 to September, 2020 in the Hepatology Department of BSMMU. Thirty seven patients (37) were included in the study. Anti HCV positive patient were primarily evaluated by history, clinical examination and investigation. Patients who met the inclusion criteria were informed in details about the study. After proper evaluation those who were labeled as CHC infection, having features of decompensation (ascites, encephalopathy, variceal bleeding, jaundice), and features of HCC were excluded from the study. **Results:** In this present study thirty seven (37) patients had detectable HCV RNA in pretreatment and at 12th weeks 34(91.9%) patients had undetectable HCV RNA. End of treatment response (ETR) was achieved 91.9% and at 24th weeks 33 (89.2%) patients were HCV RNA undetectable so sustained virological response (SVR12) was achieved 89.2%. Liver functions were significantly improve ($p<0.05$) from baseline to subsequent 4th, 12th, 24th weeks follow-up. The common adverse effects were nausea, fatigue, headache, sleep disturbance and some haematological abnormalities were observed 1(2.7%) patient haemoglobin <10 gm/dl and 2(5.4%) patients had thrombocytopenia < 50000/cumm. None of them experienced any serious adverse event and discontinued treatment. **Conclusions:** From this study it can be concluded that treatment with the single-tablet regimen of sofosbuvir and velpatasvir for 12 weeks was highly effective and safe pan- genotypic treatment for patients with compensated HCV cirrhosis of liver and also improvement of liver function.

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INTRODUCTION

Hepatic cirrhosis: A final pathway for a wide variety of chronic liver diseases is a pathologic entity defined as diffuse hepatic fibrosis with the replacement of the normal liver architecture by regenerative nodules.[1] Liver cirrhosis is one of the major causes of morbidity and morta¬lity throughout the world.[2] It is clinically described as either 'compensated' or 'decompensated'. Compensated cirrhosis may be diagnosed at routine check-up, biochemical tests may be normal or slightly deranged, and patients may remain compensated until death from other cause. Compensated cirrhosis may be asymptomatic or presented with vascular spider, palmar erythema, unexplained epistaxis, clubbing, pigmentation, Dupuytren's contracture, collateral vessels in abdomen, gynaecomastia, parotid enlargement, hair loss in axilla or loss of libido etc. splenomegaly, testicular atrophy are helpful diagnostic signs. Cirrhosis may be confirmed by biochemical test, imaging or histopathology. Liver cirrhosis is a critical stage of chronic liver dis¬ease, including that caused by hepatitis C virus (HCV). In the absence of antiviral therapy 67%-91% of pa¬tients with HCV-related LC patients die of liver-related causes.[3] Hepatitis C virus (HCV) infection is one of the most common causes of chronic liver disease worldwide.[4] The long term impact of HCV infection is highly variable, ranging from minimal histological changes to extensive fibrosis and cirrhosis with or without hepatocellular carcinoma (HCC) . The number of chronically infected persons worldwide is around 160 million.[5] The chronic disease is generally slowly progressive and cirrhosis develops within 20 years in about 10-20% of patients.[6] although fibrosis progression can vary due to several factors such as age, alcohol consumption or hepatitis B or human immunodeficiency virus (HIV) co-infection.[7] Once cirrhosis is established, a yearly incidence of hepatocellular carcinoma of 1.4-3.4%, and a yearly incidence of hepatic decompensation (including episodes of ascites, jaundice,hepatic encephalopathy or variceal bleeding) of 3.9-5.7%.[8] It was estimated that in 2005 more than 185 million people had hepatitis C antibody. Although HCV related chronic disease with cirrhosis is common in our clinical practice In Bangladesh the prevalence of HCV infection is 0.88%.[9] DAA-based regimens are the best options in HCV- infected patients with compensated cirrhosis including newly diagnosed patients. Indications depend on the HCV genotype, the severity of liver disease and/or prior therapy. Treatment-naïve HCV related compensated (child- pugh A) cirrhosis, should be treated with the fixed-dose combination of sofosbuvir and velpatasvir for

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12 weeks.[10] However, Bangladesh is a developing country in South Asia. It is among those countries where HCV infection is moderately prevalent (1.5 to 3.5 percent). Globally HCV genotype 1 is the most common genotype (46.2%). HCV genotype 3 is the next commonest genotype (30.1%). Around three-quarter of world's total HCV genotype 3 patients are distributed among the countries of south Asia. Other genotypes are found sporadically in south Asia.[11] Sofosbuvir plus Velpatasvir is a highly effective and safe pangenotypic drug regimen in patient with HCV infected compensated cirrhosis which was 98% achieved sustained virological response 12 weeks after the end of treatment with improved liver function.[12]

MATERIAL AND METHODS

This is an quasi experimental study. The study was conducted in the department of Hepatology, Bangabandhu Sheikh Mujib Medical University, Dhaka.This study was carried out from January, 2020 to September 2020. All subjects who met the eligibility criteria, willing to take part in the study were enrolled in study. After taking informed written consent 37 patients out of 40 selected through purposive sampling due to COVID-1 pandemic .Anti HCV positive patient were primarily evaluated. They were evaluated by proper history, clinical examination and investigation patients who meet the criteria of Compensated cirrhosis. Clinical (any one of five stigmata of CLD – vascular spider, gynaecomastia, palmar erythema, leuconychia, testicular atrophy) and laboratory features (prolonged prothrombin time, reduced serum albumin) suggestive of cirrhosis of liver. Other supportive evidence of cirrhosis of liver like presence of oesophageal varices on endoscopy and coarse echotexture of liver on ultrasonography and fibroscan of liver (McCormick P et al. 2018) was informed in details about the study. The potential benefits and risks of the use of the regimens containing combination of Sofosbuvir plus Velpatasvir were explained to them. Tablet Sovosbuvir (400mg) plus Velpatasvir (100mg) were given orally daily for 12 weeks. Patients were followed up at 4, 12, 24 weeks. All data were collected in a preformed data collection sheet. All data were analyzed by SPSS version 20. Quantitative data were presented as mean ±SD and qualitative data were presented as percentage. Qualitative data were analyzed by Chi-square test and quantitative data were analyzed by paired t-test and ANOVA test. Statistical significance was considered when P value less than 0.05

RESULTS

Total 37 (thirty seven) treatment naïve HCV related compensated cirrhosis patients were enrolled in this study. All patients received standard treatment with sofosbuvir (400mg) plus velpatasvir (100mg) for 12 weeks.

It was observed that almost half of the (48.6%) patients were ≤50 years. The mean age was found 50.7±8.9 years with rage from 30-70 years. More than half (54.1%) patients was male and 17(45.9%) was female. More than half (51.4%) of the patient's monthly income was 5001-10000 taka [Table 1].

It was observed that mean age was found 50.7±8.9 years with range from 30-70 years. The mean prothrombin time was found 14.1±2.1 second with range from 10.9-20 second. The mean serum albumin was found 35.0±6.6 g/l

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with range from 20-48 g/l. The mean HCV RNA was found 6.7±1.5 IU/ml with range from 2.4-9.3 IU/ml. The mean Hb% was found 11.8±1.8 gm/dl with range from 5.5-15.5 gm/dl. The mean WBC was found 5860.5±2105.5 cumm with range from 3500-11000 cumm. The mean platelet count was found 160.0±72.8 (109/l) with range from 41-450 (109/l). The mean ALT was found 82.7±41.1 IU/L with range from 28-177 IU/L. The mean AST was found 99.0±53.3 IU/L with range from 34-275 IU/L. The mean AST/ALT ratio was 1.7±0.68 with rang from 0.6-2.4. The mean serum creatinine was found 0.96±0.17 mg/dl with range from 0.60-1.30 mg/dl. The mean serum bilirubin was found 1.77±0.30 mg/dl with range from 0.60-6.9 mg/dl. The mean INR was found 1.30±0.20 with range from 0.91-1.94. The mean fibroScan test was found 43.5±17.9 kpa with range from 12.5-75 kpa. [Table 2].

In pretreatment the mean prothrombin time was found 14.1±2.1 seconds, at 4th weeks mean was found 13.2±2.1 second, at 12th weeks mean was 13.6±2.8 second, 24th weeks at mean was 14.3±1.4 second. In pretreatment the mean serum albumin was found 35.0±6.6 g/l, at 4th weeks mean was found 34.1±5.2 g/l, at 12th weeks mean was 36.2±9.5 g/l, 24th weeks at mean was 38.2±6.1g/l. In pretreatment the mean ALT was found 82.7±41.1 IU/L, at 4th weeks mean was found 87.9±103.3 IU/L, at 12th weeks mean was 56.5±21.5 IU/L, 24th weeks at mean was 43.2±28.4 U/L. In pretreatment the mean AST was found 99.0±23.3 IU/L, at 4th weeks mean was found 76.3±27.4 IU/L, at 12th weeks mean was 79.8±21.4 IU/L, 24th weeks at mean was 87.6±23.5 IU/L. In pretreatment the mean AST /ALT ratio was found 1.70±0.64, at 4th weeks mean was found 1.56±0.76, at 12th weeks mean was 1.34±0.68, 24th weeks at mean was 1.30±0.64. In pretreatment the mean serum bilirubin was found 1.77±0.30 mg/dl. 1.45±1.2 mg/dl, at 4th weeks mean was found 1.29±0.93 mg/dl, at 12th weeks mean was 0.83±0.46 mg/dl, 24th weeks at mean 1.45±1.2 mg/dl. In pretreatment the mean INR was found 1.30±0.20, at 4th weeks mean was found 1.20±0.17, at 12th weeks mean was 1.19±0.24, 24th weeks at mean was 1.22±0.19. Serum albumin and INR at baseline vs 4th weeks, 12th weeks and 24th weeks statistically significant ($p<0.05$). Other parameters were at baseline vs 12th weeks, 24th weeks were statistically significant ($p<0.05$) [Table 3].

The viral kinetics at the end of treatment (ETR) and after 12 weeks of completion of treatment (SVR12) was assessed in all enrolled patients. In pretreatment all (100%) patients had HCV RNA detectable. After end of treatment 34(91.9%) patients had undetectable HCV RNA and 3(8.1%) patients had detectable HCV RNA. End of treatment response (ETR) was achieved 91.9%. After 12 weeks of completion of treatment 33(89.2%) patients had undetectable HCV RNA and 4(10.8%) patients had detectable HCV RNA. Sustained virological response (SVR12) was achieved 89.2%. [Table 4].

Common adverse events during and after treatment were 2(5.4%) patient had fatigue and headache, 1(2.7%) patients had sleep disturbance and 4 (10.8%) patient had Nausea. Some haematological abnormalities were observed 1(2.7%) patients had haemoglobin <10 gm/dl and 2(5.4%) patients had thrombocytopenia < 50000/cumm. [Table 5].

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Table 1: Distribution of the study patients by demographic variable (n=37)

Demographic variable	Number of patients	Percentage
Age (in years)		
≤50	18	48.6
>50	19	51.4
Mean±SD	50.7±8.9	
Range (min-max)	30-70	
Sex		
Male	20	54.1
Female	17	45.9
Monthly income (in Taka)		
≤5000	2	5.4
5001-10000	19	51.4
10001-20000	9	24.3
>20000	7	18.9

Table 2: Distribution of the study patients by baseline characteristics (n=37)

Parameters	Mean±SD	Range (min-max)
Age (in years)	50.7±8.9	30-70
Prothrombin Time (sec)	14.1±2.1	10.9-20
Serum Albumin(g/L)	35.0±6.6	20-48
HCV RNA (IU/ml) (LOG)	6.7±1.5	2.4-9.3
Hb% (gm/dl)	11.8±1.8	8.5-15.5
WBC (cumm)	5860.5±2105.5	3500-11000
Platelet count (109/l)	160.0±72.8	41-450
ALT (IU/L)	82.7±41.1	28-177
AST (IU/L)	99.0±53.3	34-275
AST/ALT ratio	1.70±0.68	0.6-2.4
Serum creatinine (mg/dl)	0.96±0.17	0.60-1.30
Serum bilirubin(mg/dl)	1.77±0.30	0.60-6.9
INR(%)	1.30±0.20	0.91-1.94
FibroScan test of liver (kpa)	43.5±17.9	12.5-7.5

Table 3: Distribution of the study patients by liver function tests in different follow-up (n=37)

Investigations report	Pretreatment	After 4th weeks	After 12th (ETR) weeks	After 24th (SVR 12) weeks	P value
	Mean±SD	Mean±SD	Mean±SD	Mean±SD	
Prothombin Time (sec)	14.1±2.1	13.2±2.1	13.6±2.8	14.3±1.4	$^{a}0.012^{s}$
P value		$^{b}0.146^{ns}$	$^{b}0.045^{s}$	$^{b}0.013^{s}$	
Serum Albumin g/l	35.0±6.6	35.9±5.2	36.2±9.5	38.7±6.1	$^{a}0.033^{s}$

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P value		[b]0.044[s]	[b]0.047[s]	[b]0.008[s]	
ALT (U/L)	82.7±41.1	87.9±13.3	56.5±21.5	43.2±28.4	[a]0.001[s]
P value		[b]0.094[ns]	[b]0.014[s]	[b]0.001[s]	
AST (U/L)	99.0±23.3	76.3±27.4	79.8±21.4	87.6±23.5	[a]0.001[s]
P value		[b]0.098[ns]	[b]0.026[s]	[b]0.007[s]	
AST/ALT ratio	1.70±0.64	1.56±0.76	1.34±0.68	1.30±0.74	[a]0.028[s]
P value		[b]0.068[ns]	[b]0.045[s]	[b]0.027[s]	
Serum bilirubin gm/dl	1.77±0.30	1.29±0.93	0.83±0.46	1.45±0.86	[a]0.022[s]
P value		[b]0.098[ns]	[b]0.046[s]	[b]0.009[s]	
INR	1.30±0.20	1.20±0.17	1.19±0.24	1.22±0.19	[a]0.005[s]
P value		[b]0.038[s]	[b]0.029[s]	[b]0.001[s]	

S= significant, ns= not significant, ap value reached from ANOVA test, bp value reached from paired t-test, p value analyzed baseline vs 4th weeks, baseline vs 12th weeks, baseline vs 24th weeks,

Table 4: Distribution of the study patients by HCV RNA (PCR) (n=37)

Time of duration	HCV RNA (PCR)	Number of patients	Percentage
Pretreatment	Detectable	37	100.0
	undetectable	0	0.0
End of treatment response (ETR)	Detectable	3	8.1
	Undetectable	34	91.9
Sustained virological response (SVR12)	Detectable	4	10.8
	Undetectable	33	89.2

Table 5: Adverse event & haematological abnormalities (n=37)

Adverse events AE)	Number of patients	Percentage
Common AEs (%)		
Fatigue	2	5.4
Pruritus	0	0.0
Flu like illness	0	0.0
Headache	2	5.4
Depression	0	0.0
Sleep disturbance	1	2.7
Skin reactions	0	0.0
Photo sensitivity	0	0.0
Nausea	4	10.8
Diarrhoea	0	0.0
Irritibilty	1	2.7
Haematological abnormalities Haemoglobin		
<10gm/dl	1	2.7
<8.5gm/dl	0	0.0
Neutropenia		

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<750/mm3	0	0.0
Thrombocytopenia		
<50000/mm3	2	5.4

DISCUSSION

This study included treatment naïve HCV related compensated cirrhosis patients. They received sofosbuvir400 mg plus velpatasvir100 mg combination once daily for 12 weeks. This study was conducted over a period from January 2020 to September 2020 in, department of hepatology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh.

This study was observed therapeutic efficacy of sofosbuvir plus velpatasvir therapy in HCV related compensated cirrhosis of liver. Patients were selected after inclusion and exclusion criteria. Finally it was planned to carry out a study with 37 patient after taken informed written consent.

It was observed that more than half of the patients (51.4%) were >50 years. The mean age was found 50.7±8.9 year minimum age was 30 year and maximum age was 70 years. Almost similar age distribution was seen in clinical trials involving compensated cirrhosis patients by13. Mean age was observed 52.60 ±12.29 years in the study conducted.[13] Our finding was more or less similar with their findings.

In this present study it was observed that more than half of the patients (54.1%) were male. Similar male predominance has also been observed in studies by,[12] which was consistent with our study.

In this current study, it was observed that in baseline all (100.0%) patients had HCV-RNA (RT-PCR) detected and mean was found 6.7±1.5 Log 10 (IU/ml). After end of treatment 34 (91.9%) out of 37 patients had undetectable HCV RNA. End of treatment response (ETR) was achieved in 91.9%. After 12 weeks of completion of treatment 33(89.2%) patients had undetectable HCV RNA. Sustained virological response (SVR12) was achieved in 89.2%. No patient had lost from follow up. This result was similar with13. In their study 35 (92.1%) patients with compensated cirrhosis achieved ETR at 12 weeks from the start of treatment. A total of 90.5% (95% CI: 84.2-95.8) of patients without cirrhosis and 92.1% (95% CI: 84.2-100) of patients with compensated cirrhosis achieved SVR 12 weeks after the end of therapy.

Study shows that12. SVR12 was achieved by 98% of patients (490/501; 95% CI, 96%-99%). Among patients with cirrhosis 96% (212/220) achieved SVR12, versus 99% (278/281) for those with advanced fibrosis. SVR12 was 98% (306/311) for treatment-naïve patients and 97% (184/190) for treatment-experienced patients.

In this present study baseline mean prothrombin time was found 14.1±2.1, After 4th weeks of treatment prothrombin time was improve but not statistically significant ($p>0.05$), After 12th weeks and After 24th weeks clinically significant reduction of prothrombin time from baseline was found ($p<0.05$). Serum albumin level was improved from baseline after 4th weeks, 12th weeks and 24th weeks of treatment which was statistically significant ($p<0.05$). Baseline platelet count mean was 160.0±22.4, at 4th weeks of treatment no

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significant improvement was found (p>0.05). At 12th and 24th weeks significant increase of platelet count was found (p<0.05).

In baseline ALT, AST and ALT/AST ratio were 82.7±41.1 IU/L, 99.0±23.3 IU/L, and 1.70±0.64, statistically significant reduction of ALT,AST,AST/ALT ratio was occurred in every subsequent follow –up from baseline to at 12th ,24th weeks (p<0.05). In baseline serum bilirubin was found 1.45±1.2 mg/dl, at 12th weeks and 24th weeks clinically significant reduction of bilirubin was found (p<0.05). Baseline INR was 1.30±0.20, statistically significant improvement (p<0.05) was occurred at 4th, 12th and 24th weeks. The result of this study was similar.[12,14,15] In their study median values of platelet, albumin, total bilirubin and serum creatinine was improved after 4 weeks.

In this current study some adverse events were occurred that 2 (5.4%) patients had fatigue and headache,1 (2.7%) patients had sleep disturbance and 4 (10.8%) had Nausea. Hematological abnormalities was observed 1(2.7%) patients hemoglobin <10 gm/dl and 2(5.4%) patients had thrombocytopenia < 50000/cumm. None of the patients experienced any serious adverse event or discontinued treatment. This finding consistent.[12,13] Their study observed that most common adverse events were occurred headache (31%).fatigue (21%), nausea (13%),12 which nearly in our study. In haematological abnormalities was found haemoglobin <10 g/dl had (28.9%),[13,16,17] and platelet count <90000/cmm had (18.42%). Which were more or less similar with our study.

Limitations of the study

The limitations of studies evaluating the therapeutic efficacy of Sofosbuvir plus Velpatasvir in HCV-related compensated cirrhosis include potential biases in trial participant selection, limited long-term follow-up data, and variations in patient demographics. Additionally, the generalizability of findings may be affected by the exclusion of certain patient populations or comorbidities.

CONCLUSIONS

Study shows that treatment with the single-tablet regimen of sofosbuvir–velpatasvir for 12 weeks was highly effective for patients with compensated HCV cirrhosis of liver and also improvement of liver function. However management of Hepatitis C is very rapidly changing. Newer drugs are coming with better efficacy.

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