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Efficacy And Tolerance of Hepatitis C Treatment with Direct-Acting Antivirals at the CNHU-HKM In Cotonou (Benin)

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1. Abstract

1.1. Background

The treatment of hepatitis C has undergone a revolution due to the advent of direct-acting antivirals (DAAs).

1.2. Objective

This study evaluated the effectiveness and safety of direct-acting antivirals for the treatment of hepatitis C in Cotonou (Benin).

1.3. Methods

This was a descriptive and analytical cross-sectional study, with both retrospective and prospective data collection over a 5-year period from December 1, 2015, to September 1, 2020. All patients with hepatitis C were treated with DAAs (SOF/VEL, SOF/LED, SOF/RBV, SOF/DCV, SOF/VEL/RBV, SOF/LED/RBV) at the University Clinic of Hepato-gastroenterology at the National Hospital and University Center Hubert Koutoukou Maga (CNHU-HKM) in Cotonou. Efficacy was assessed by sustained virological response (SVR) at 12 weeks after the end of treatment.

1.4. Results

During the study period, 206 patients were enrolled, including 137 women (66.5%) and 69 men (33.5%). The median patient age was 65 years (range 18-87 years). The mean viral load was 3,507,337 IU/ml ± 1,156,444 (range 3422-75,674,348 IU/ml). The overall SVR rate was 96% (197/206). Genotyping was performed carried out for 151 participants and revealed an SVR of 93% (64/69) in patients with genotype 1, 97.5% (78/80) in genotype 2, and 100% (2/2) in genotype 4. The SVR was 86% (24/28) in cirrhotic people and 96% (171/178) in non-cirrhotic people. Adverse effects developed by the patients included asthenia (20/206; 10%), anemia (14/206; 7%), weight loss and digestive disorders (6/206; 3%).

1.5. Conclusion

Direct-acting antivirals are a very effective and well-tolerated treatment in Benin with a Sustained Virological Response evaluated at 95% at the CNHU-HKM during the study period.

2. Introduction

Since its discovery in 1989, the hepatitis C virus (HCV) has been the subject of intensive research, due to the growing awareness of the potential severity of the infection and the significance of the epidemic of HCV infection [1]. HCV constitutes a major public health problem and is one of the main causes of chronic liver diseases around the world [2]. In 2015, according to the World Health Organization (WHO), 1% of the world population, or approximately 71 million individuals, were chronic carriers of HCV, responsible for 399,000 deaths per year [3]. Africa has an estimated regional prevalence of HCV of 15% representing 11 million people according to the WHO [3]. In Benin, the seroprevalence of HCV is 1% in the general population according to a study carried out in 2019 [4]. Hepatitis C is generally asymptomatic during early

infection and most often goes unnoticed until the advent of cirrhosis. In 50 to 90% of cases, the presence of HCV leads to chronic infection. This is defined as carriage of the virus for a period of more than 6 months. Chronic forms may eventually progress to cirrhosis and primary liver cancer [5]. The main goal of hepatitis C treatment is to eradicate the virus. Currently, treatment is based on direct-acting antivirals (DAAs) [6]. DAAs are very effective, well tolerated and consist of the use of oral medications in short-term therapeutic combinations [7,8]. The evaluation of the effectiveness of treatment of viral hepatitis C is based on sustained virological response (SVR). A preliminary study carried out in Benin from 2015 to 2017 on the first patients with hepatitis C treated with DAAs (n = 44) revealed an SVR rate of 91.7% with very few side effects [9]. However, the latter was limited by small sample size. The present study aimed to evaluate the effectiveness and tolerance of treatment of hepatitis C with DAAs in a large cohort at the CNHU-HKM in Cotonou.

3. Methods

3.1. Study Design and Setting

This was a cross-sectional study carried out between December 1, 2015 and September 1, 2020. Data collection was both retrospective (from December 2015 to December 2019) and prospective (from January 2020 to September 2020). The study was carried out in the university clinic of Hepato-Gastroenterology (CU-HGE) at the CNHU-HKM, the largest hospital in Benin. All subjects placed on DAAs treatment and who had given their informed consent for the study were included. Subjects who had an incomplete file and who could not be seen to complete the collection of information were not included.

3.2. Definitions

Cirrhosis was determined based on cluster of clinical and biological characteristics mainly morphological changes in the liver (painless hepatomegaly with a sharp lower edge), hepatocellular insufficiency (lower prothrombin levels, hypoalbuminemia) and portal hypertension (collateral abdominal venous circulation, splenomegaly, thrombocytopenia) or defined by a Fibroscan® value greater than 13 kPa. The treatment was considered effective when there was virological cure, meaning when SVR was achieved. The latter was defined by undetectable HCV RNA 12 weeks after the end of treatment (SVR12). Biochemical response was defined by normalization of aminotransferases (ALAT and ASAT) after treatment. Treatment failure corresponded to HCV RNA remaining detectable 12 weeks after the end of treatment.

3.3. Data collection and statistical analysis

Data collection was done following direct interview using a standardized questionnaire. Data entry, processing and analysis were carried out using EPI info software version 7.2.1.0. For the comparison of frequencies, the Chi2 (x^2) test was used. A $p \leq 0.05$ was considered statistically significant. A multivariate analysis by logistic regression was carried out to study the factors associated with the effectiveness of DAAs treatment.

3.4. Ethics

Ethical approval for this study was obtained from the local ethics committee for biomedical research of the University of Parakou (CLERB-UP); (REF: 0341/CLERB-UP/P/SP/R/SA). Verbal consent was obtained from participants, and confidentiality observed during data collection and processing.

5. Results

5.1. Characteristics of the Study Population (Table 1)

During the study period, 206 patients were enrolled, 137 women (66.5%) and 69 men (33.5%). The median age was 65 years (range 18-87 years). There was a predominance of cases in the age group over 55 (159/206, 77%). In the 151 patients that had genotyping carried out, genotype 2 was the most common (80/151, 53%). Genotype 1 was found in 69/151 patients (46%) and genotype 4 in 2 patients (1%). 28/206 patients (14%) were cirrhotic. 18 patients had received treatment and failed therapy. 11 patients had received previous treatment with 1st generation DAAs (SOF-RBV and SOF-LED) 7 patients had received previous treatment with IFN and RBV. These were patients who had failed treatment with DAAs (the first patients treated with DAAs in Benin) or with IFN – RBV. Among the patients previously treated with DAAs, 6 patients had genotype 2 and had been treated with the combination of SOF and RBV. The other 5 patients were genotype 1 and had been treated with SOF and LED. The average viral load in IU/ml was 3,507,337 ± 1,156,444 (range 3422-75,674,348 IU/ml). There was 1 case of co-infection with HIV and 11 cases of HBV co-infection.

Table 1: Characteristics of the study population.

	Number	Percentage (%)
Sex (n=206)		
Male	69	33.5
Female	137	66.5
Age in years Less than 25	02	01.0
[25- 34[	07	03.4
[35- 44[	11	05.3
[45- 54[	27	13.1
55 and older	159	77.2
Genotype (n=151)		
1	69	45.7
2	80	53.0
4	02	01.3
Cirrhosis		
Yes	28	14.0
No	178	86.0

5.2. Efficacy and tolerance of DAA treatment

Seven DAAs regimens were used: SOF-VEL (75/206, 36% of patients), SOF- RBV (58/206, 29%), SOF-LED (36/206, 17%), SOF-DCV (3/206, 1%), SOF-VEL-RBV - (16/206, 8%), SOF - LED - RBV (12/206, 6%), SOF - DCV - RBV (6/206, 3%) (Table 2). Treatment lasted 12 weeks for 200 patients (200/206, 97%) and was given for 24 weeks for 6 cirrhotic patients (6/206, 3%). RBV was combined with an NS5A inhibitor in patients who were cirrhotic or had failed a first treatment with DAAs. The overall SVR was 95.6% (197/206). The biochemical response was 95% (115/121). By genotype, the SVR was 93% (64/69) for patients with genotype 1 and 97.5% (78/80) for those with genotype 2. We recorded 2 patients with genotype 4, both with SVR. The SVR was 96% (171/178) in non-cirrhotic patients and 86% (24/28) in cirrhotic patients (p=0.20) (Figure 1).

The SVR was 97% (73/75) in patients treated with SOF-VEL, 96.5% (56/58) for those treated with SOF–RBV, 97% (35/ 36) for those treated with SOF–LED, 66.7% (2/3) for those treated with SOF–DCV, 94% (15/16) for those treated with SOF–VEL–RBV, 92% (11/12) for those treated with SOF–LED–RBV and 83.3% (5/6) for those treated with SOF–DCV–RBV (Figure 2). Nine patients (9/206; 4.5%), experienced virological failure (Table 3).

Factors associated with SVR were studied through a univariable analysis. We observed that the treatment was statistically less effective in obese patients (SVR of 90.9% in obese subjects and 98.5% in non-obese subjects; p=0.03). No statistically significant association was found between SVR and digestive or extra-digestive clinical manifestations, nor with biological factors including genotype, viral load or aminotransferases. Likewise, the existence of cirrhosis, previous treatment against hepatitis C, co-infections with HBV or HIV were not statistically associated with SVR (Table 4 and 5). The most common adverse events were asthenia, in 20/206 patients (10%); anaemia in 14/206 patients (7%); weight loss and digestive disorders in 6/206 patients (3%). Of note, no serious adverse events leading to discontinuation of treatment were observed.

Table 2: Distribution according to specific anti-HCV treatment of patients treated for hepatitis C at the CUHGE of the CNHU-HKM from 2015 to 2020.

	Liver status	Number	Percentage (%)	Genotype (Number)	SVR %
SOF + VEL		73	36.4	1(13) 2(57) 4(3)	96
	Cirrhotic	5	6.8	1(2) 2(3)	60
	Non cirrhotic	68	93.2	1(11) 2(54) 4(3)	100
SOF + RBV		58	28.1	1(8) 2(50)	96.5
	Cirrhotic	4	6.9	1(2) 2(2)	50
	Non cirrhotic	54	93.1	1(6) 2(48)	100
SOF + LED		34	17.5	1(30) 2(2) 4(2)	97.2
	Cirrhotic	1	2.9	1(1)	0
	Non cirrhotic	33	97	1(29) 2(2) 4(2)	100
SOF + VEL + RBV		16	7.8	1(10) 2(6)	93.7
	Cirrhotic	5	31.3	1(2) 2(3)	20
	Non cirrhotic	11	68.8	1(8) 2(1)	100

SOF + LED + RBV		12	5.8	1(10) 2(2)	91.7
	Cirrhotic	1	8.3	2(1)	0
	Non cirrhotic	11	91.7	1(10) 2(1)	100
SOF + DCV + RBV		06	2.9	1(3) 2(3)	83.3
	Cirrhotic	3	50	1(1) 2(2)	33.3
	Non cirrhotic	3	50	1(2) 2(1)	100
SOF + DCV		03	1.5	1(1) 2(2)	66.7
	Cirrhotic	0	0	0	0
	Non cirrhotic	3	10	1(1) 2(2)	66.6

Table 3: Characteristics of patients who had virological failure.

Age (years)	**Sex**	**Genotype**	**Presence of cirrhosis**	**Status**	**Treatment received**	**Comorbidites**
59	F*	1	No	naive	SOF+ RBV	-
46	M**	2	No	naive	SOF+ RBV	-
69	F	1	Yes	naive	SOF+LED+RBV	-
72	M	Not typed	No	naive	SOF+ VEL	-
68	F	1	No	naive	SOF+ LED	Obesity
68	F	1	No	pretreated***	SOF+ VEL+ RBV	-
66	F	Not typed	No	naive	SOF+VEL	-
65	M	Not typed	No	naive	SOF+DCV	Obesity
52	M	2	Yes	naive	SOF+DCV+RBV	Obesity

F: Female*

M: Male**

.This patient was treated with SOF + LED***

Table 4: Association between SVR and biological factors.

	SVR		p-value	OR	IC95% [OR]
	Yes	No			
ALAT					
< 45	85	0		1	
45 - 100	81	6	0.2	0.9	0.84 - 0.97
> 100	33	1		0.97	0.91 - 1.02
ASAT					
< 45	67	1		1	
45 - 100	85	6	0.2	0.2	0.02 - 1.79
> 100	27	2		0.2	0.01 - 2.3
Viral load (UI/ml)					

≤ 1000000	94	2	0.1	1	
> 1000000	101	7		3.25	0.66 - 16.0
Genotype					
1	64	4		1	
2	78	2	0.55	2.43	0.43 - 13.7
4	2	0		1.06	1.0 - 1.12
Fibrosis status					
F4	27	1	0.64	1	
Status other than F4 (F0. F1. F2. F3)	104	25		1.29	0.14 –11.5

Table 5: Association between SVR and medical history or comorbidities.

	SVR		p-value	OR	IC95% [OR]
	Yes	No			
Blood transfusion					
No	142	8		1	
Yes	54	1	0.27	3.04	0.37 - 24.9
History of dental care					
No	150	6	0.44	1	
Yes	46	3		0.61	0.14 - 2.54
Scarification					
No	50	1	0.45	1	
Yes	146	8		0.36	0.04 - 2.99
Hepatitis B					
No	182	7	0.08	1	
Yes	9	2		0.17	0.6 - 1.12
Cirrhosis				1	
No	174	7	0.20	0.34	0.06 - 1.7
Yes	17	2			
Diabetes					
No	156	8	0.21	1	
Yes	39	0		1.17	0.47 - 2.88
Obesity					
No	132	2	**0.03**	1	
Yes	40	4		0.15	0.02 - 0.85
HTA					
No	82	6	0.18	1	
Yes	111	3		2.7	0.65 - 11.14
Phytotherapy					
No	124	5	0.71	1	
Yes	58	3		0.77	0.18 - 3.37
Chronic alcohol intake					

No	183	9	0.54	1	
Yes	13	0		1.12	0.36 - 2.5
Smoking/tobacco					
No	191	9	0.88	1	
Yes	4	0		1.04	1.01 - 1.07
Previous treatment with DAAs					
No	185	8	0.39	1	
Yes	10	1		0.43	0.04 - 3.8
Previous treatment with RBV					
No	189	9	0.72	1	
Yes	7	0		1.04	0.10 - 34.16

Figure 1: Association between SVR and medical history or comorbidities.

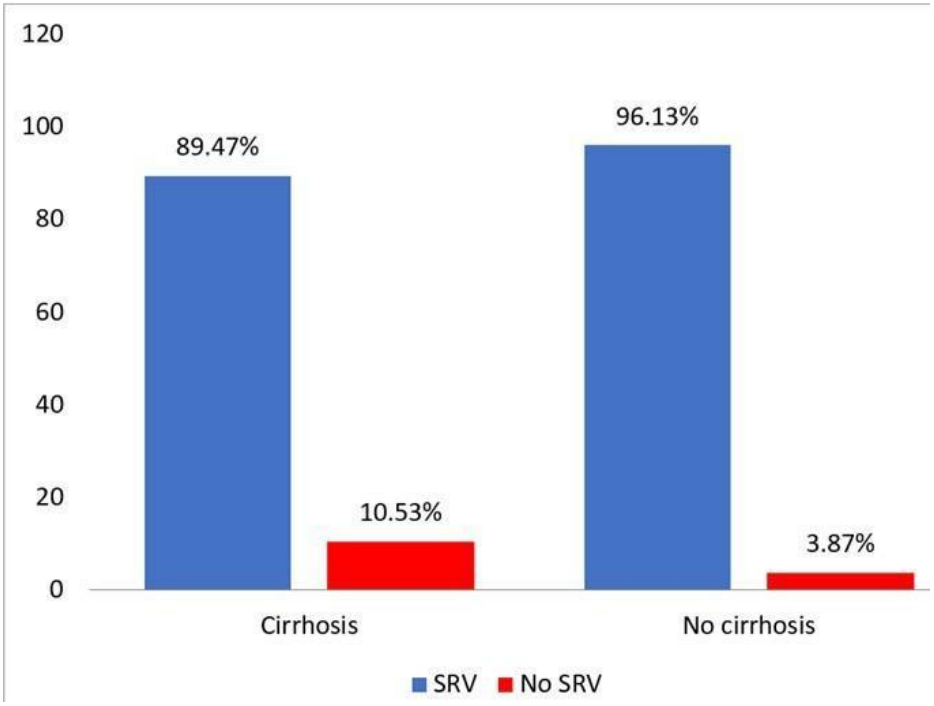
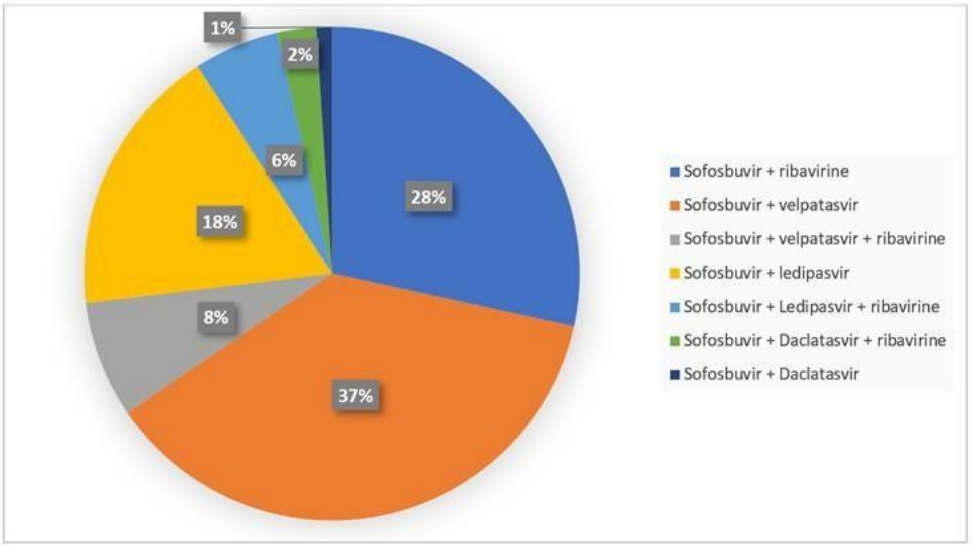


Figure 2: Sustained virological response according to the type of treatment in patients treated for hepatitis C at the CUHGE of the CNHU-HKM from 2015 to 2020.



6. Discussion

For the study population, the overall SVR was 95.6% (197/206). This result corroborates that of Kodjoh et al. [10] and Ahovegbe et al [11] in Benin, who observed respectively an SVR of 91.7% in 44 patients and 94% in 52 patients treated with DAAs. In Togo in 2018, Lawson-Ananissoh et al. [12] found SVR rates greater than 90% in 84 treated patients. Our results are also similar to those reported in high-income countries where many studies reported an SVR greater than 90% [6]. As for the biochemical response, it was 95% (115/121). This result is similar to that of Kodjoh et al. [10] who found a biochemical response of 93.3% in all the patients studied. The different therapeutic protocols used in patients with genotypes 1 and 4 were largely in accordance with international recommendations for the treatment of hepatitis C with the exceptions that (a) some patients who received retreatment were given additional RBV [6,13] and (b) some patients received the SOF-RBV combination no longer recommended by WHO. Triple therapies combining RBV with DAA treatment were also used in some patients with advanced cirrhosis. The use of SOF-RBV in some patients reflects the pragmatic considerations and resource constraints that countries such as Benin face, including delays in availability of generic pan-genotypic DAAs. This cheaper combination was therefore used in the absence of contraindications for some people who may otherwise have progressed to cirrhosis. Among patients treated with the SOF-VEL regimen, 95% were naïve and 91% non-cirrhotic, and 96% (73/75) obtained an SVR12. These results are in keeping with similar studies in Europe and North America. In a real-world study by Mangia et al. [14], 12 cohorts from 7 countries had an overall SVR of nearly 99%. Patients treated with the SOF-VEL-RBV combination, including 44% (7/16) of previously treated patients and 31% cirrhotic patients (5/16), obtained an SVR in 94% of cases (15 out of 16 patients). Among 58 patients, all naïve, and (93%) non-cirrhotic treated with SOF-RBV, 56/58 (96.5%) achieved an SVR. This result is similar to that of Lawson-Ananisoh et al. [12] in Togo who described an SVR rate of 92% in genotype 2 patients treated with SOF-RBV. Of 36 patients, 94% naive and 92% non-cirrhotic, who were treated with the SOF-LED regimen, 35 (97%) achieved SVR. This rate is close to that found by Eloumou et al. [15] in 2017 in Cameroon where the SVR rate was 94% in 111 patients treated with the combination SOF-LED. Of 12 patients, including 8% (1/12) treatment-experienced and cirrhotic patients, treated with the SOF-LED-RBV combination, 11/12 (92%), obtained an SVR. These results are consistent with those of Mizokami et al. [16] in 2015 who found an SVR of 98% in genotype 1 patients treated with the same combination. During the study, three patients were treated with SOF–DCV. An SVR was observed in 2/3 patients (66.7%). Failure was observed in one genotype 1 cirrhotic patient. This result is lower than that found by Sulkowski et al. [17] in 2014 in the USA who described an SVR of 91% in a study involving 211 patients with genotypes 1, 2 and 3 treated with the SOF- DCV regimen. In our study, 83% (5/6) patients treated with the SOF-DCV-RBV combination achieved SVR; among these 6, 3 were cirrhotic. A similar result was observed in the ALLY-3C study [18] where a rate of 88% was found in a cohort of patients with cirrhosis. The low SVR rate observed in our study may be linked to the very small number of patients treated with this combination but requires further evaluation. SOF-DCV has a lower barrier to resistance than 2nd generation DAAs and while still a feature of the WHO guidelines, may require revision if larger studies support our finding. One of the main factors for SVR found in the existing literature is the absence of cirrhosis [19,20]. In the present study, we also had a higher sustained virological response rate in non-cirrhotic patients (96%) than in cirrhotic patients (89.5%) (Figure 1). However, this difference was not found to be statistically significant (p=0.20). This could be explained by the fact that in our study, treatment in cirrhotic patients was further strengthened by the addition of RBV and an extension of the duration of treatment to 24 weeks to improve the SVR rate. Non-obese subjects had a higher SVR (98.5%) compared to obese subjects (90.9%) (p=0.03). The lower virological response observed in obese patients could be explained by hepatic steatosis aggravated by obesity. The latter accelerates the progression of liver fibrosis, possibly making patients less responsive to treatment. This situation could also be explained by pharmacokinetics properties of drugs, notably an increase of the volume of distribution and thus an alteration in the bioavailability of drugs in these subjects. It would therefore be preferable to obtain weight loss in these obese subjects in parallel with the initiation of antiviral treatment for a better therapeutic response [21]. The factors for virological failure found in the literature were essentially cirrhosis, failure of previous antiviral treatment against hepatitis C, genotype 3, and the presence of resistant mutations within the virus [20,21]. In a recent study, we found that only 8/10 (80%) patients with genotype 2d infection in Benin had an SVR, suggesting that this genotype may be less amenable to treatment [11]. In this study, 9 patients (4.5%) of the study population experienced virological failure (Table III). Among the latter, 2 patients were cirrhotic, and 1 patient was previously treated with the SOF-LED regimen. Obesity was found in 3/9 patients who failed treatment. Adverse effects developed by the patients during the study were infrequent and mild. These included asthenias 9.7% (20 patients), anemia 6.8% (14 patients), weight loss and digestive disorders (nausea, constipation) in 6 patients. In Cameroon, Eloumou et al. [15] in a study on the effectiveness and safety of the SOF-LED combination with or without RBV, also identified asthenia as the most frequent side effect (10.6%). In the phase III therapeutic trial, ASTRAL-1 [22], involving patients treated with the SOF-VEL combination, the most common adverse effects were headache, asthenia and nausea. Of note, the adverse effects observed were mild and did not require interruption of treatment. There were several limitations to our real-world study. Firstly, genotyping was not available for all patients. The presence of resistance mutations could not be assessed due to the unavailability of resistance tests in our country. Treatment could have been linked to poor adherence with treatment or co-administration of other medications, but not disclosed during the interview. These results demonstrate the effectiveness and good tolerance of direct-acting antivirals in the treatment of hepatitis C in a real-world setting in West Africa.

This therapeutic revolution must be accompanied by broad screening of the population, better monitoring and greater accessibility to treatment so that the hepatitis C epidemic can be eradicated across the Beninese population in the near future, by 2030 as recommended by the WHO.

7. Conclusion

At the end of this study, the overall SVR response was high. Side effects were mild. DAAs constitute a very effective and well-tolerated treatment from which all subjects suffering from hepatitis C in Benin should benefit with a view to eradicating this threat.

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