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ORIGINAL ARTICLE

Assessment of Liver Stiffness among Chronic Hepatitis C Patients after Oral Antiviral Drugs in Zagazig University Hospitals

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ABSTRACT

Background: The hepatitis C virus (HCV) may be an important explanation for hepatic pathology, cirrhosis, and hepatocellular carcinoma. The aim of the study was to assess the liver stiffness before and after oral antiviral medication in chronic viral hepatitis c patients attending Zagazig University Hospitals**. Methods:** This study included forty three Egyptian patients with HCV infection diagnosed by HCV RNA real time quantitative PCR whose ages were ranged from 29-71 , All patients took anti-viral treatment according to the rules of the committee responsible for hepatitis c virus control in Egypt. Treatment regimens included: Sofosbuvir, daclatasvir and ribavirin (RBV). The patients were collected and followed up in the period between April 2017 and August 2018. , radiological tool liver transient elastography (TE) and laboratory tests APRI, FIB4 test were done for all patients. **Results:** There were improvements in liver stiffness measurements,APRI,FIB4 test hemoglobin, fasting blood sugar , platelets count, and ALT, AST levels in patients who achieved a 48-week sustained infectious agent response. The failure to achieve improvement within the level of liver stiffness was related to treatment failure. **Conclusions:** Treatment with sofosbuvir drug regimen makes significant decrease in liver stiffness measurements and fibrosis indices.

Key words: Liver stiffness; Fibroscan; Direct antivirals

INTRODUCTION

Hepatitis C Virus (HCV) may be a major reason for hepatic disease, failure and hepatocellular carcinoma, 14.7% is the highest percentage in the world acquiring the virus infection in Egypt [1].

The treatment of HCV aimed at achieving a sustained virological response (SVR), to reduce the liver harm, good cure decrease pathology and death rates. Studies before found that HCV cure diminishes hepatocellular carcinoma risk [2]. Staging of hepatic pathology determines liver illness progression throughout chronic infection.

In 2014, the NS5B RNA-polymerase inhibitor sofosbuvir was first drug used for the HCV treatment in conjunction with pegylated Interferon- (Peg- IFN) with or without ribavirin (RBV), yielding high SVR rates [3].

The corner stone of liver fibrosis assessment is a liver histopathologic diagnostic test exploitation; using Ishak or Metavir scores, Non-invasive methods assessing liver pathology can replace liver diagnostic test utterly [4] , results were obtained with liver stiffness (LS) measurements obtained from transient elastography (TE) (Fibroscan) ,which is used in routine clinical follow up were suggested to be another technique to evaluate the fibrosis of liver [5]. Serial liver fibrosis measurements are taken for analysis of liver harm caused by HCV [5]. Transient elastography is a suitable tool for getting continual measurements as a result of it

eliminates all the hazards of invasive liver diagnostic test technique .In great study, they found that fibroscan has a 91% specificity and an 87% sensitivity in comparison to biopsy regarding fibrosis in the liver [6].

Researchers found a decline in hepatic fibrosis after treatment in relation to before the treatment [7].

Noninvasive ways evaluating hepatic pathology induced by HCV were planned over few years recently like the AST to platelet magnitude relation index (APRI) and the fibrosis index based on 4 factors (FIB4) [8].

This research goal is to assess the liver stiffness before and after oral antiviral medication in chronic viral hepatitis c patients attending Zagazig University Hospitals using non-invasive methods like fibroscan and APRI and FIB4 test. .

METHODS

Site of study

This study was carried out in Gastroenterology and hepatology unit and Advanced center for liver diseases, Internal Medicine Department, Faculty of medicine, Zagazig University Hospitals between April 2017 and August 2018.

Written informed consent was obtained from all participants and the study was approved by the research ethical committee of Faculty of Medicine, Zagazig University. The work has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Type of the study: prospective cohort study.

Sample size

Out of forty five Egyptian patients with chronic HCV infection diagnosed by HCV PCR (polymerase chain reaction) and fulfilled the inclusion and exclusion criteria of the study, 43 patients (aged 29-71 years old) continued the follow up period and were enrolled in the study.

Inclusion criteria:

Treatment naïve or treatment experienced patients with chronic HCV infection proven by HCV PCR. Age 18-75 years, any Body Mass Index (BMI) (weight in kilograms/squared height in meters) was included. Patients with either liver cirrhosis or not, with or without decompensation was included.

Exclusion criteria:

Patients with other cause of chronic disease like hepatitis B or HIV, bilharzasias or hemochromatosis or chronic alcoholism or wilson illness. Patients with HCC or any hepatic focal lesion, intravenous drug abusers were excluded.

LS measurements and fibrosis indices (APRI) and (FIB4) were performed to all the study patients at zero week (baseline) and 48 weeks after end of treatment using fibroscan device (transient elastography) and laboratory analysis like complete blood picture (CBC) and liver function test (LFT) alpha fetoprotein (AFP) lipid profile and fasting blood sugar (FBS) and international normalization ratio(INR) were done. HCV PCR was done before treatment, at end of treatment and 48 weeks after treatment.

Statistical analysis

All data were analyzed using SPSS 20.0 for windows, MedCalc Statistical Software version 15.8. Continuous variables were expressed as the mean ± SD, median and range while the categorical variables were expressed as a number (percentage). Continuous variables were checked for normality by using Shapiro Wilk test. One-Way ANOVA, Kruskal-Wallis H (KW), Post-hoc Fisher's, Least Significant Difference test (LSD) tests were used, Pearson product-moment correlation coefficient, Receiver operating characteristic (ROC) curve analysis were used. $p< 0.05$ was considered statistically significant (S).

RESULTS

There was a marked improvement in liver stiffness measurements; fibrosis indices blood hemoglobin levels, blood sugar levels for fasting, platelets, and liver enzymes ALT, AST in patients who achieved a 48-week sustained virological response more than non-responders (table 1). There is a significant difference between the baseline and 48 weeks groups as regard FBS, Hb, AST and positive PCR (table 2) in naieve patients , There is a significant

difference between the cirrhotic and non-cirrhotic patients regarding FIB scan, APPRI, FIB4, Hb, PLT, AST, Albumin, Total bilirubin, alpha FB and TG (table3).

There is a significant difference regarding FIB scan, APPRI, FIB4, AST, Total bilirubin and alpha FB more than experienced group (table 4). The failure to realize improvement within the level of liver stiffness was related to treatment failure, sustained viral response. APRI, FIB4 measurements, alpha FB, FIB Scan measurements were done (table 5).

Table 1. Comparison between responders and non-responders.

Variable		*Responders (n=39)*	*Non-Responders (n=4)*	t / χ^2	*P*
***Age** (years) Mean ± SD*		49.13 ± 10.21	42.5 ± 9.98	1.238	.223
Sex	*Male*	19 (48.7%)	1 (25%)	.863	.353
	Female	20 (51.3%)	3 (75%)		
FIB scan		8.01 ± 2.45	15.19 ± 3.71	**5.344**	**.000**
APRI		.832 ± .422	2.07 ± 1.08	**4.705**	**.000**
FIB4		2.365 ± 1.04	4.778 ± 3.26	**3.446**	**.001**
FBS(mg/dl)		112.08 ± 35.25	129.75 ± 48.47	.925	.360
Hemoglobin(g/dl)		13.64 ± .869	11.18 ± 1.03	**5.320**	**.000**
TLC		7.64 ± 1.34	7.45 ± 1.53	.266	.792
PLT		151.21 ± 36.45	88.5 ± 3.69	**3.402**	**.002**
ALT(u/dl)		46.85 ± 18.11	60.75 ± 11.09	1.497	.142
AST(uldl)		46.89 ± 19.24	84.75 ± 22.16	**3.704**	**.001**
Albumin(gldl)		3.87 ± .502	3.0 ± .183	**3.393**	**.002**
INR		1.02 ± .067	1.03 ± .050	.056	.956
Total bil.(mg/dl)		1.029 ± .228	2.188 ± .312	**9.362**	**.000**
Alpha FB(ng/ml)		8.9 ± 4.102	23.5 ± 2.082	**6.971**	**.000**
TC(mg/dl)		182.46 ± 16.37	203.5 ± 3.87	**2.537**	**.015**
TG(mg/dl)		148.87 ± 14.09	144.0 ± 19.95	.635	.529
LDL(mg/dl)		92.72 ± 16.69	95.75 ± 7.72	.356	.723
DM		15 (38.5%)	2 (50%)	.198	.656
Child-Pugh	*A*	37 (94.9%)	4 (100%)	.400	.527
	B	2 (5.1%)	0 (0%)		

FIBscan = fibroscan,APRI=alt/platelets ratio index, FIB4=fibrosis index based on 4 parameters,TLC=total leucocytic count,PLT= platelets count,INR=international normalization ratio ,alpha FP= alpha fetoprotein,TC=total cholesterol,TG=triglycerides,LDL=low density lipoprotein,DM= diabetes mellitus.ALT=alanine aminotransferase,AST=asprtate aminotransferase.Total bil=total bilirubin

Table 2. Comparison between pre and post treatment.

Variable	*Pre (n=43)*	*Post (n=43)*	t / χ^2	*P*
***Age** (years) Mean ± SD*	48.51 ± 10.26	50.14 ± 11.54	.691	.491
FIB scan(kpa)	8.673 ± 3.293	7.598 ± 3.026	1.576	.199
APPRI	.948 ± .614	.763 ± .694	1.303	.196
FIB4	2.589 ± 1.496	2.229 ± 1.694	1.045	.299
FBS(mg/dl)	113.72 ± 36.32	97.67 ± 21.40	**2.496**	**.015**
Hemoglobi(g/dl)	13.41 ± 1.13	10.99 ± 1.19	**9.655**	**.000**
TLC	7.62 ± 1.34	7.47 ± 1.35	.505	.615
PLT	145.37 ± 39.28	161.84 ± 38.89	1.953	.054
ALT(u/dl)	48.14 ± 17.95	41.33 ± 13.91	1.968	.052
AST(u/dl)	50.42 ± 22.22	41.23 ± 19.88	**2.020**	**.047**
Albumin(g/dl)	3.79 ± .544	3.99 ± .531	1.830	.071
INR	1.023 ± .065	1.032 ± .102	.506	.614
Total bil.(mg/dl)	1.14 ± .412	1.05 ± .318	1.069	.288
Alpha FB(ng/ml)	10.26 ± 5.83	8.44 ± 5.65	1.469	.145
TC(mg/dl)	184.42 ± 16.79	187.77 ± 17.32	.911	.365
TG(mg/dl)	148.42 ± 14.51	147.53 ± 16.03	.268	.789
LDL(mg/dl)	93.0 ± 16.03	92.14 ± 15.0	.257	.798
Positive PCR	43 (100%)	4 (9.3%)	**91.861**	**.000**
Cirrhosis	10 (23.3%)	5 (11.6%)	2.051	.152

FIBscan= fibroscan,APRI=alt/platelets ratio index, FIB4=fibrosis index based on 4 parameters,TLC=total leucocytic count,PLT= platelets count,INR=international normalization ratio ,alpha FP= alpha fetoprotein,TC=total cholesterol,TG=triglycerides,LDL=low density lipoprotein,DM= diabetes mellitus.,PCR=polymerase chain reaction.FBS=fasting blood sugar. ALT=alanine aminotransferase,AST=asprtate aminotransferase.total bil=total bilirubin

Table 3. Comparison between patients regarding cirrhosis.

Variable		*Cirrhotic (n=10)*	*Non- Cirrhotic (n=33)*	*t / χ^2*	*P*
***Age** (years) Mean ± SD*		46.9 ± 9.04	49.0 ± 10.68	.562	.577
Sex	*Male*	4 (40%)	16 (48.5%)	.224	.636
	Female	6 (60%)	17 (51.5%)		
FIB scan(kpa)		13.71 ± 2.49	7.15 ± 1.46	**10.447**	**.000**
APPRI		1.64 ± .885	.737 ± .280	**5.192**	**.000**
FIB4		4.169 ± 2.24	2.11 ± .71	**4.655**	**.000**
FBS(mg/dl)		117.9 ± 38.21	112.45 ± 36.24	.411	.683
Hemoglobin(gm/dl)		12.48 ± 1.46	13.69 ± .857	**3.289**	**.002**
TLC		8.02 ± 1.64	7.5 ± 1.23	1.081	.286
PLT		107.2 ± 33.99	156.94 ± 33.27	**4.122**	**.000**
ALT(u/l)		55.2 ± 16.89	46.0 ± 17.95	1.438	.158
AST(u/l)		69.9 ± 24.66	44.52 ± 17.95	**3.584**	**.001**
Albumin(g/dl)		3.26 ± .268	3.94 ± .505	**4.090**	**.000**
INR		1.03 ± .067	1.02 ± .065	.371	.712
Total bil.(mgldl)		1.75 ± .449	.951 ± .109	**9.597**	**.000**
Alpha FB(ng/ml)		19.75 ± 4.29	7.38 ± 1.64	**13.825**	**.000**
TC(mg/dl)		184.3 ± 20.0	184.45 ± 16.04	.025	.980
TG(mg/dl)		140.1 ± 14.22	150.94 ± 13.82	**2.159**	**.037**
LDL(mg/dl)		96.1 ± 17.02	92.06 ± 15.87	.694	.492
DM		4 (40%)	13 (39.4%)	.001	.973

FIBscan = fibroscan,APRI=alt/platelets ratio index, FIB4=fibrosis index based on 4 parameters,TLC=total leucocytic count,PLT= platelets count,INR=international normalization ratio ,alpha FP= alpha fetoprotein,TC=total cholesterol,TG=triglycerides,LDL=low density lipoprotein,DM= diabetes mellitus, bil= bilirubin,FBS=fasting blood sugar, ALT=alanine aminotransferase,AST=asprtate aminotransferase

Table 4. Comparison between patients regarding treatment history.

Variable		Experienced (n=7)	Naïve (n=36)	t / χ^2	P
Age *(years) Mean ± SD*		50.0 ± 10.28	48.22 ± 10.38	.415	.680
Sex	*Male*	4 (57.1%)	16 (44.4%)	.379	.538
	Female	3 (42.9%)	20 (55.6%)		
FIB scan(kpa)		12.9 ± 3.73	7.85 ± 2.52	**4.480**	**.000**
APPRI		1.55 ± .94	.829 ± .462	**3.139**	**.003**
FIB4		4.42 ± 2.39	2.23 ± .952	**4.176**	**.000**
FBS(mg/dl)		127.43 ± 43.28	111.06 ± 34.88	1.094	.280
Hemoglobin(gm/dl)		12.67 ± 1.25	13.55 ± 1.07	1.944	.059
TLC		8.16 ± 1.22	7.52 ± 1.35	1.166	.250
PLT		118.86 ± 35.09	150.53 ± 38.37	2.022	.051
ALT(u/l)		47.86 ± 18.13	48.19 ± 18.17	.045	.964
AST(u/l)		66.86 ± 27.43	47.22 ± 19.97	**2.239**	**.031**
Albumin(g/dl)		3.66 ± .739	3.81 ± .507	.678	.502
INR		1.029 ± .049	1.022 ± .068	.234	.816
Total bil.(mgldl)		1.56 ± .585	1.06 ± .32	**3.294**	**.002**
Alpha FB(ng/ml)		16.67 ± 6.62	9.01 ± 4.84	**3.610**	**.001**
TC(mg/dl)		188.86 ± 20.69	183.56 ± 16.13	.761	.451
TG(mg/dl)		143.29 ± 15.96	149.42 ± 14.23	1.024	.312
LDL(mg/dl)		88.71 ± 16.18	93.83 ± 16.09	.769	.446
DM		4 (57.1%)	13 (36.1%)	1.060	.303

FIBscan= fibroscan,APRI=alt/platelets ratio index, FIB4=fibrosis index based on 4 parameters,TLC=total leucocytic count,PLT= platelets count,INR=international normalization ratio ,alpha FP= alpha fetoprotein,TC=total cholesterol,TG=triglycerides,LDL=low density lipoprotein,DM= diabetes mellitus,FBS=fasting blood sugar.total bil=total bilirubin , ALT=alanine aminotransferase,AST=asprtate aminotransferase

Table 5. Multivariate regression analysis to identify factors associated with failure of liver stiffness improving.

	β	*S.E.*	*Sig.*	*OR*	*95% CI for OR*	
					Lower	*Upper*
Age	.130	.177	.463	1.138	.805	1.609
Sex (F vs M)	1.403	.932	.132	0.246	.040	1.529
DM	1.406	1.691	.406	4.080	.148	2.187
T. Bil(mg/dl)	.939	1.171	.423	2.557	.258	5.362
INR	.797	.720	.104	6.403	.563	7.912
APRI	.294	.461	**.001**	12.461	.641	6.348
FIB4	.649	.261	**.000**	9.164	.265	8.649
Alpha FB(ng/ml)	.164	.346	**.007**	4.316	.894	2.491
FIB SCAN	.023	.330	**.038**	1.024	.536	1.954
SVR	4.294	1.664	**.000**	14..26	2.35	32.164

FIBscan = fibroscan,APRI=alt/platelets ratio index, FIB4=fibrosis index based on 4 parameters,TLC=total leucocytic count,PLT= platelets count,INR=international normalization ratio ,alpha FP= alpha fetoprotein, ,DM= diabetes mellitus.,SVR=sustained virological response.F=female,M=male,T.BIL=total bilirubin,

DISCUSSION

The Egyptian health authorities provided direct antiviral agents(DAAs) regimens as an a treatment for patients infected with HCV at 2014 [9].

This treatment can achieve SVR, DAAs do not have an anti-fibrotic impact however primarily involved with viral eradication [10].

This results in resolution of the hepatic damage, and decline hepatic cell failure complications like variceal hemorrhage and liver cancer [11].

HCV can be a great factor of liver fibrosis and an important agent of liver injury and inflammation [12].

In spite of the gradual decrease in liver fibrosis once SVR, hepatic pathology is still its accurate and repeated estimation is required [13].

The aim of the present study was to assess the changes in liver stiffness measurements by transient elastography and also the variations in indices of fibrosis as determined by APRI, FIB-4 following HCV treatment with sofosbuvir based regimens. Estimating its effect without IFN on the changes of liver fibrosis measuring by fibroscan device and hepatic fibrosis indices was done.

Similar to Bachofner, et al. [13], and Elsharkawy, et al. [14] who found that LS measurements considerably declined twelve weeks following the end of antiviral therapy,

our study revealed that LS considerably declined 48weeks following the end of antiviral drug treatment in patients who achieve SVR. In addition, there was a significant improvement in APRI and FIB4 scores. These scores are affected by the declining of liver enzymes and platelets count denoting vital improvement of liver pathology and necro inflammation following Sofosbuvir based treatment. Also, treatment responders showed vital decrease in these indices whereas decline of these indices in relapsers wasn't obvious [14].

Our results showed significant improvement in hepatic fibrosis measuring in responders patients (90.6%) while in non-responders patients (9.4%) no significant decrease in LS measurements. In cirrhotic patients (23.2%) receive the treatment there was significant improvement of LS measuring, 50% of these patients became non cirrhotic and also the alternative 50% persisted but with degrees of liver fibrosis less than degrees before treatment may be due to persistent vireamia and treatment faiure and these results are in agreement with Elsharkawy et al. showed significant improvement in hepatic fibrosis measuring in 81.1% of cirrhotic patients, but they stay to be cirrhotic with liver fibrosis degrees less than what before treatment [13]. D'ambrosio et al, found there is cirrhosis even in patients who achieve sustained virological response (SVR) [15]. Chekuri et al reported important decline in hepatic fibrosis degree in patients who are cirrhotic, but sixty percent who had cirrhotic before treatment were still cirrhotic at SVR24 [12].

CONCLUSION

Drug regimens containing sofosbuvir make a major decline in liver pathology degree proved by utilizing the fibroscan and fibrosis indices like APRI and FIB4 test even in cirrhotic patients, but starting treatment early is imperative except liver injury will be constant . These non-invasive fibrosis scores might be useful in the developing regions of restricted resources for patients follow up. Long term keeping eye on these patients is recommended to completely perceive the effect of SVR on liver pathology dynamics.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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