Research Article

Fibrosis evaluation of hepatitis C patients

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Evaluation of fibrosis in chronic hepatitis C patients treated with direct-acting antivirals

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Abstract

Background and Aim: This study is an evaluation of liver fibrosis measurements determined using transient elastography and aspartate aminotransferase-to-platelet ratio index (APRI) scores of patients diagnosed with chronic hepatitis C (CHC) who were treated with direct-acting antivirals (DAAs).

Materials and Methods: The liver fibrosis measurements recorded using transient elastography, APRI scores, and the biochemical data from before and after treatment of 40 patients with CHC who were treated with DAA were reviewed. Patients who received paritaprevir+ritonavir/ombitasvir+-dasabuvir were included in Group 1 (n=20), and patients who received sofosbuvir+ledipasvir±ribavirin in Group 2 (n=20).

Results: The mean liver fibrosis measurement of the patients was 15.73±10.63 kPa (min–max: 5.20–45.00 kPa) before treatment and 2.56±8.84 kPa (min–max: 4.30–42.00 kPa) after treatment. There was a significant improvement in liver fibrosis with a regression of 20.16% at the end of treatment compared with the start (p=0.001) with no significant difference between treatment groups (p=0.542). The highest regression rate of 75% was seen in patients with F2 fibrosis at the end of treatment. Significant regression was also found in patients with F3 fibrosis, with a rate of 57.2%, and in those with F4 fibrosis, with a rate of 17.6% (p=0.035). Significant reduction was also observed in the APRI scores of patients at the end of treatment compared with the start of treatment (p<0.001), with no significant difference between treatment groups (p=0.328).

Conclusion: Noninvasive assessments of CHC patients treated with DAA revealed regression in liver fibrosis measurements and APRI scores and significant improvements were seen in the stage of fibrosis in the early phases following treatment.

Keywords: Direct acting antivirals; fibroscan; hepatitis C.

Introduction

Hepatitis C virus (HCV) is an important cause of chronic liver disease that infects 130–150 million people worldwide with a prevalence of 2–3%.[1] It has been established that worldwide, some 350,000 patients per year lose their lives due to HIV infection.[2] Spontaneous clearance of HCV within six weeks following acute hepatitis C infection occurs in only 15–25% of the patients, and chronic hepatitis and fibrosis develop in the majority of patients.[3] Structural changes of the parenchyma resulting from progression of the fibrosis are accompanied by clinical manifestations of chronic liver disease and hepatic dysfunction.[4,5] The onset of liver fibrosis is usually insidious, and mortality and morbidity are associated with cirrhosis usually developing 20–30 years later.[6] The stage of liver fibrosis is associated with prognosis in chronic hepatitis C (CHC) infection; it has an important effect on treatment strategy and follow-up.[7] A liver biopsy is currently regarded as the gold standard procedure for the assessment of fibrosis; however, it is an invasive procedure with serious complications in rare conditions, there may be discrepancies between observers, and sampling errors may occur.[8] It is difficult to monitor the dynamic process of progressive and regressive liver fibrosis with repeated liver biopsies, as it is not easily tolerated by the patients. These limitations to the use of a liver biopsy led to the need for a reliable, repeatable, noninvasive method for evaluating liver fibrosis.[9]

Transient elastography (TE) uses a device that quantitatively measures liver fibrosis and is regarded as an important tool in the evaluation, follow-up, and treatment of fibrosis in chronic liver disorders.[10] The combined use of serum markers and TE is known to increase the accuracy in the diagnosis of fibrosis.[11] The most effective approach recommended in the current guidelines for the evaluation of severity and fibrosis of liver disease is the combination of direct biological markers and TE. A biopsy is recommended for any patient with discrepancies between the first two methods when those results affecting clinical decision-making, and it has been reported that the need for a liver biopsy can be markedly decreased with this approach.[12,13]

The aim of this study was to evaluate the changes in fibrosis occurring after treatment with noninvasive methods by analyzing liver fibrosis measurements obtained with TE and aspartate aminotransferase (AST)-to-platelet ratio index (APRI) scores in CHC patients treated with direct-acting antivirals (DAA).

Materials and Methods

Patients

Forty CHC patients followed at Tepecik Training and Research Hospital Department of Gastroenterology who had completed a treatment regimen with DAA and who had undergone liver fibrosis measurements with a Fibroscan 502 Touch (Echosens, Paris, France) before and after treatment and at 12 and 24 weeks were included in the study. The patients were evaluated in 2 groups: those receiving paritaprevir+ritonavir/ombitasvir+dasabuvir treatment were assigned to Group 1 (n=20), and those receiving sofosbuvir+ledipasvir±ribavirin to Group 2 (n=20). Patients included in the study provided written, informed consent.

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Table 1. Demographic data of the patients

Characteristics	Total patients (n=40)		Group 1 (n=20)		Group 2 (n=20)		p
	n	%	n	%	n	%	
Age (years) (mean)	62.53±12.82		61.75±10.49		63.30±15.04		0.708
Male	14	35	9	45	5	25	0.185
Female	26	65	11	55	15	75	
Treatment naive	24	60	14	70	10	50	0.197
Non-treatment naive	16	40	6	30	10	50	
Mean BMI (kg/m^2)	27.18±4.41		27.03±4.95		27.33±3.92		0.836
BMI (normal) (kg/m^2)	11	27.5	6	30	5	25	
BMI (overweight) (kg/m^2)	21	52.5	10	50	11	55	0.933
BMI (obese) (kg/m^2)	8	20	4	20	4	20	

BMI: Body mass index.

HCV-RNA was undetectable in all of the patients included in the study (n=40) at the end of treatment. The sustained virological response (SVR) 12 weeks after treatment (SVR12) and/or 24 weeks (SVR24) after the end of treatment are conventionally used as CHC therapy endpoints.

Approval for this study was obtained from the Ethics Committee of Clinical and Laboratory Research of Tepecik Training and Research Hospital (no: 2017/13-1).

Data Collection

Details of age, gender, HCV genotype, cirrhosis status, and previous treatment for HCV of all of the patients included in the study were recorded. AST, alanine aminotransferase (ALT), gamma-glutamyl transferase (GGT), total bilirubin, international normalized ratio (INR), serum creatinine, white blood cell (WBC), hemoglobin, and platelet data before and after treatment with DAA were retrospectively reviewed and noted. Liver fibrosis measurements obtained with the Fibroscan device and APRI scores from before and after treatment were also retrospectively reviewed and recorded.

Non-invasive Tests

APRI scores were calculated using the Wai formula:[14] (AST/upper limit of normal)/platelet count (expressed as platelets x 10^9/L) x 100

The fibrosis stage cut-off values for APRI scores were:[14] mild/no fibrosis (F0-1), APRI<0.5; significant fibrosis (F2-3), APRI=0.5–1.9; cirrhosis (F4), APRI≥2.

The liver fibrosis measurements were performed by a single experienced operator using the Fibroscan device and an M-probe. The measurements were expressed as kPa and the mean value of 10 measurements taken at a depth of 25–65 mm was recorded. Measurements with a success rate (successful measurements ×100/all measurements) of at least 60% and an interquartile range/M ratio of less than 30% were considered valid and used for statistical analysis. The cut-off values for the liver fibrosis measurements according to the Metavir fibrosis stages were as follows:[15] F0-1: 2.5–7 kPA, F2: 7.1–9.5 kPA, F3: 9.5–12.5 kPA, F4: >12.5 kPA.

Statistical Analyses

All of the study data were analyzed using IBM SPSS Statistics for Windows, Version 22.0 (IBM Corp., Armonk, NY, USA) Numerical variables were first tested for normality with the Shapiro-Wilk test. Normality was achieved for data that did not conform to normal distribution by applying log transformation. Variables that did not conform to normality with log transformation were analyzed using non-parametric tests.

Analysis of variance with repeated measures was carried out in patients before and after treatment. Pre- and post-treatment analyses of all of the patients were conducted using a paired groups t-test without taking the group into consideration. Cross tabulation was performed on categorical data, and chi-square and McNemar tests were used for analysis. Analysis of triple fibrosis scoring for APRI values was performed with Wilcoxon paired groups testing in each patient group.

For numerical variables, Pearson's correlation coefficient was calculated for those conforming to normal distribution, and Spearman's rho correlation coefficient for those not conforming to normal distribution. The level of statistical significance was a p value <0.05.

Results

Demographic and Clinical Characteristics

The mean age of the 40 patients included in the study was 62.5 years (min–max: 32–83 years). In all, 35% were male and 65% were female (Table 1). The mean body mass index (BMI) was 27.18±4.41 kg/m^2. There was no significant difference between the treatment groups when the patients were evaluated according to the BMI classes of normal (18.5–24.9 kg/m^2), overweight (25–29.9 kg/m^2), and obese (≥30 kg/m^2) in total and by each group (p=0.933). Twenty-four patients (60%) were treatment-naive and 16 (40%) were not.

Twenty patients were non-cirrhotic (50%), 18 (45%) had Child-A cirrhosis, and 2 (5%) had Child-B cirrhosis. Ten patients in Group 1 were non-cirrhotic (50%), 10 had Child-A cirrhosis (50%); in Group 2, 10 patients were non-cirrhotic (50%), 8 had Child-A cirrhosis (40%), and 2 had Child-B cirrhosis (10%). There was no significant difference between the groups in terms of cirrhosis (p=0.329).

HCV Genotypes

When HCV genotypes were analyzed, 37 of the 40 patients had genotype 1b (92.5%), 1 had genotype 2 (2.5%), and 2 had genotype 3 (5%). All of the patients in Group 1 had genotype 1b (100%), while 37 pa-

Table 2. Mean laboratory test values before and after treatment

Laboratory results	Group 1 (n=20)	Group 2 (n=20)	Total patients (n=40)
AST (UI/L)			
BT	49.10±23.63	58.25±38.85	53.67±32.07
AT	21.45±5.38	27.05±10.57	24.25±8.75
ALT (UI/L)			
BT	50.65±36.72	63.90±95.00	57.28±71.41
AT	17.30±11.13	18.90±9.76	18.10±10.36
GGT (U/L)			
BT	63.50±57.09	72.80±74.50	68.15±65.68
AT	30.65±34.17	36.10±25.92	33.38±30.06
Total bilirubin (mg/dL)			
BT	0.83±0.27	0.99±0.65	0.91±0.50
AT	0.81±0.48	0.83±0.50	0.82±0.48
INR			
BT	1.13±0.17	1.13±0.21	1.13±0.19
AT	1.10±0.16	1.18±0.19	1.14±0.18
Serum creatinine (mg/dL)			
BT	1.54±1.60	0.89±0.24	1.22±1.18
AT	1.42±1.39	0.91±0.29	1.17±1.02
WBC (10^3/uL)			
BT	7.30±2.56	6.45±2.55	6.87±2.56
AT	7.90±2.43	6.32±2.55	7.11±2.58
Hemoglobin (g/dL)			
BT	13.42±1.50	13.10±2.29	13.26±1.92
AT	13.01±1.78	12.13±2.33	12.57±2.09
Platelet (x10^3/µL)			
BT	215.25±88.37	175.05±79.60	195.15±85.48
AT	222.80±88.63	181.95±66.00	202.38±79.86

ALT: Alanine aminotransferase; AST: Aspartate aminotransferase; AT: after treatment (within 1 month after treatment); BT: Before treatment; GGT: Gamma-glutamyl transferase; INR: International normalized ratio; WBC: White blood cell.

tients in Group 2 had genotype 1b (92.5%), 1 had genotype 2 (2.5%), and 2 had genotype 3 (5%). There was no statistically significant difference between the groups regarding genotype distribution (p=0.198).

Biochemical Findings

The mean AST, ALT, GGT, bilirubin, INR, creatinine, WBC, hemoglobin, and platelet values of the patients before and after treatment are provided in Table 2.

There was a significant reduction in AST (p<0.001), ALT (p=0.001), and GGT (p<0.001) values following treatment with no significant difference between the groups. Significant changes were not seen in serum bilirubin, creatinine, or INR values following treatment, again without significant difference between the treatment groups.

The mean pre- and post-treatment WBC value of the patients was 6.87±2.56 10^3/uL and 7.11±2.58 10^3/uL, respectively. A mild elevation was noted following treatment; however, this change was not statistically significant (p=0.383).

Table 3. Distribution of patients by fibrosis group according to APRI score before and after treatment

	APRI (After treatment)				
Total patients	F0-1	F2-3	F4	Total	
(n=40)	(n)	(n)	(n)	n	%
F0-1 (n)	15	0	0	15	37.5
F2-3 (n)	14	6	0	20	50
F4 (n)	0	5	0	5	12.5
Total					
n	29	11	0	40	
%	72.5	27.5	0	100	

APRI: Aspartate aminotransferase-to-platelet ratio index.

The mean pre- and post-treatment hemoglobin level was 13.26±1.92 g/dL and 12.57±2.09 g/dL, respectively, and a statistically significant decrease was seen in the hemoglobin level at the end of treatment (p=0.004). There was no significant difference between the groups (p=0.236).

The mean pre- and post-treatment platelet count was 195.15±85.48 10^3/µL and 202.38±79.86 10^3/µL, respectively. An elevation was noted in platelet counts following treatment but it was not statistically significant (p=0.219). There were mild increases in the platelet count in both groups following treatment, but without a significant difference between the groups (p=0.956).

APRI Scores

The mean pre- and post-treatment APRI score was 0.90±0.79 and 0.39±0.29, respectively, and a significant decrease was noted in the APRI score following treatment (p<0.001). The pre- and post-treatment APRI scores were statistically comparable between the groups (p=0.960) and there was no significant difference between the groups regarding the decrease in APRI score following treatment (p=0.328).

The APRI scores before treatment indicated that 15 of the 40 patients (37.5%) had F0-1 fibrosis, 20 (50%) had F2-3, and 5 (12.5%) had F4 fibrosis. When the patients were distributed in terms of the APRI score following treatment, all 5 patients who had F4 fibrosis before treatment (100%) had regressed to F2-3 fibrosis, 14 of 20 patients who had F2-3 fibrosis (70%) had regressed to F0-1, and 6 patients (30%) remained at F2-3 stage. The regression in fibrosis at the end of treatment according to the APRI score was statistically significant (p<0.01) (Table 3)

The APRI score showed a positive correlation with AST, ALT, total bilirubin, INR, and a negative correlation with WBC and platelet counts (Table 4).

Liver Fibrosis Measurements

The mean liver fibrosis measurement was 15.73±10.63 kPa (min–max: 5.20–45.00 kPa) before treatment and 12.56±8.84 kPa (min–max: 4.30–42.00 kPa) at the end of treatment; a regression of 20.16% (mean difference: -3.17±5.83 kPa) was noted and the reduction following treatment was found to be significant (p=0.001). The liver fibrosis measurement values of the two groups were statistically similar before and after treatment (p=0.833) and there was no significant difference between the groups following treatment (p=0.542).

Table 4. Comparison of APRI scores with other parameters

	r	p
APRI versus AST	0.766	**<0.001**
APRI versus ALT	0.514	**0.001**
APRI versus total bilirubin	0.369	**0.019**
APRI versus INR	0.678	**<0.001**
APRI versus WBC	-485	**0.002**
APRI versus platelet count	-727	**<0.001**

APRI scores showed a positive correlation with AST, ALT, total bilirubin, and INR, and a negative correlation with WBC and platelet counts. ALT: Alanine aminotransferase; APRI: Aspartate aminotransferase-to-platelet ratio index; AST: Aspartate aminotransferase; INR: International normalized ratio; WBC: White blood cell.

Table 5. Fibrosis stage according to liver fibrosis measurements before and after treatment

	Liver fibrosis after treatment					
All patients	**F0-1**	**F2**	**F3**	**F4**	**Total**	
(n=40)	**n**	**n**	**n**	**n**	**n**	**%**
F0-1 (n)	8	0	0	0	8	20
F2 (n)	6	1	0	1	8	20
F3 (n)	3	1	2	1	7	17.5
F4 (n)	0	0	3	14	17	42.5
Total						
n	17	2	5	16	40	
%	42.5	5	12.5	40	100	

When changes in fibrosis stage were analyzed according to the pre- and post-treatment measurements of liver fibrosis, it was noted that 20% of the patients (n=8) had F0-1 fibrosis, 20% (n=8) had F2 fibrosis, 17.5% (n=7) had F3 fibrosis, and 42.5% (n=17) had F4 fibrosis before treatment. Fourteen of 17 patients who had F4 fibrosis before DAA treatment (82.4%) remained at F4, while 3 patients (17.6%) regressed to F3 fibrosis. One of 7 patients who had F3 fibrosis before treatment (14.3%) progressed to F4 fibrosis, 1 patient (14.3%) regressed to F2 fibrosis and 3 patients (42.9%) regressed to F0-1 fibrosis. One of 8 patients who had F2 fibrosis before treatment (12.5%) progressed to F4 fibrosis, 1 patient (12.5%) maintained an F2 stage, and 6 patients (75%) regressed to F0-1 fibrosis. When the changes in fibrosis stage following antiviral treatment were evaluated in all of the patients according to the liver fibrosis measurements, a statistically significant regression was noted compared with the pre-treatment values (p=0.035) (Table 5). When the two groups were compared regarding changes in the degree of fibrosis, there was no significant difference between the groups (p=0.917).

The liver fibrosis measurements obtained with TE showed a powerful correlation with APRI and AST, a positive correlation with INR, and a negative correlation with hemoglobin and platelet counts (Table 6).

Table 6. Comparison of liver fibrosis measurements with other parameters

	r	p
Liver stiffness versus AST	0.512	**0.001**
Liver stiffness versus ALT	0.160	0.324
Liver stiffness versus GGT	0.079	0.626
Liver stiffness versus total bilirubin	0.140	0.931
Liver stiffness versus INR	0.318	**0.045**
Liver stiffness versus serum creatinine	-0.189	0.244
Liver stiffness versus WBC	0.790	0.670
Liver stiffness versus hemoglobin	-0.329	**0.038**
Liver stiffness versus platelet count	-0.341	**0.031**
Liver stiffness versus APRI	0.483	**0.002**

Liver fibrosis measurements obtained using transient elastography demonstrated a powerful correlation with APRI and AST, a positive correlation with INR, and a negative correlation with hemoglobin and platelet counts. ALT: Alanine aminotransferase; APRI: Aspartate aminotransferase-to-platelet ratio index; AST: Aspartate aminotransferase; GGT: Gamma-glutamyl transferase; INR: International normalized ratio; WBC: White blood cell.

Discussion

End-stage liver disease due to CHC is currently a major cause of liver-related deaths worldwide.[16] Most of the patients infected with HCV develop a chronic infection and if left untreated, approximately 30% of the patients will progress to cirrhosis in 20–30 years. On the other hand, successful antiviral treatment and achieving SVR reduces liver-related morbidity and mortality, HCC incidence, and the need for liver transplantation.[17,18] The reported SVR rate is 50–55% in all genotypes with regimens containing pegylated interferon and ribavirin; however, the SVR rate has notably reached 99% with currently available regimens containing DAA.[19]

Numerous serological, biochemical indicator, and imaging methods have been developed to evaluate liver fibrosis noninvasively.[20] Among the frequently administered noninvasive methods, APRI, an indirect biochemical indicator, has been validated in studies performed on patients with CHC, and is one of the simple panels that can diagnose marked fibrosis and cirrhosis with acceptable accuracy.[6] Another method to evaluate liver fibrosis noninvasively is TE. TE with FibroScan is a reliable, noninvasive, and repeatable assessment technique that can measure liver fibrosis and evaluate large amounts of liver tissue.[21] Current guidelines for the management of HCV infection recommend that the most effective approach to the evaluation of the severity of liver disease and fibrosis is combining direct biochemical indicators with the use of TE.[12,13] A biopsy is recommended for any patient when there are discrepancies between these two methods with results that could affect clinical decision-making. It is thought that need to perform a liver biopsy can be markedly reduced with this approach.

While liver fibrosis was once regarded as an irreversible progression, it is now known to be a dynamic process as a result of recent evidence demonstrating regression and variability.[22] In many studies with long-term follow-up of fibrosis in patients with CHC, a marked decrease has been noted in liver fibrosis values in patients achieving SVR with antivirals compared with those unresponsive to treatment.[23,24]

In 2017, Gheorghe et al.[25] reported on 681 compensated cirrhotic patients with CHC and genotype 1b who had received a 12-week DAA (ombitasvir/paritaprevir/ritonavir+dasabuvir) treatment. Liver fibrosis was measured before treatment, at the end of treatment, and in the third month after treatment (SVR12). A significant improvement was observed in the third month after treatment compared with the baseline

(p<0.0001), and similarly there were significant regressions in APRI and Fibrosis 4 scores (p<0.0001). In our study, the mean liver fibrosis measurement was 15.73±10.63 kPa (min–max: 5.20–45.00 kPa) before treatment and 12.56±8.84 kPa (min–max: 4.30–42.00 kPa) at the end of treatment. Significant improvement was seen in patients following treatment, with a 20.16% regression in liver fibrosis measurements compared with the baseline (mean difference: -3.17±5.83 kPa) (p=0.001). The study groups were statistically similar regarding liver fibrosis measurements before and after treatment (p=0.833) and there was no significant difference between the groups in terms of regression in liver fibrosis at the end of treatment (p=0.542). Similarly, the APRI values at the end of treatment were significantly lower compared with those recorded at the start of treatment (p<0.001) and there was no difference between treatment groups (p=0.328).

Tag-Adeen et al.[26] found that 80 of 84 CHC patients who received combination therapies containing sofosbuvir demonstrated a significant regression in liver fibrosis measurements six months after treatment (SVR 24) compared with the baseline (p<0.001) (mean difference: -3.5 kPa). When the patients were evaluated according to the degree of fibrosis, the greatest change compared with baseline measurements occurred in cirrhotic patients (F4). However, only 39% of these patients regressed to lower fibrosis groups and 61% were still cirrhotic and at risk for liver function disorders and HCC despite achieving SVR 24. In our study, the greatest regression in terms of liver fibrosis measurements to a lower fibrosis group following treatment was seen in patients with F2 fibrosis, with a rate of 75%, and regression was noted at a rate of 57.2% in patients with F3 fibrosis, and 17.6% in those with F4 fibrosis (p=0.035). There was less improvement in liver fibrosis in the cirrhotic patient group compared with the other fibrosis groups and this was similar in both treatment groups (p=0.199). This was considered to be related to the use of post-treatment measurements at an early stage.

In 2017, Pons et al.[27] examined early-stage and long-term liver and spleen fibrosis in 41 CHC patients following treatment with DAA and it was noted that there was rapid improvement in liver fibrosis during the first four weeks of treatment (p=0.002). Improvement was sustained during treatment and significant improvement was also seen in post-treatment measurements (p=0.014) and persisted until the 48th week after treatment (p=0.003). When the patients were categorized into two groups of those demonstrating ≥10% improvement and those with <10% improvement in liver fibrosis measurements at the end of treatment, there was no significant difference in the final improvement in liver fibrosis at the sixth month (p=0.487). This study has shown that there can be significant, persistent improvement in liver fibrosis until the 48th week, and that those showing a slow improvement in fibrosis at the end of treatment may improve like those showing rapid initial improvement when examining long-term, persistent regression in liver fibrosis. In our study, patients with F3-F4 fibrosis had lower rates of post-treatment fibrosis compared with the F2 group, but considering that the available information indicates that the improvement process may continue, long-term follow-up and repeated measurements are needed for a better evaluation.

Tag-Adeen et al.[26] also noted significant decreases in APRI, FIB4, AST, ALT, bilirubin, INR and hemoglobin levels at six months after treatment (p<0.001) and a positive correlation was shown between improvement in liver fibrosis and APRI (p=0.002), AST (p=0.04), ALT (p=0.04), bilirubin (p=0.03). In our study, as with post-treatment APRI, there were also significant decreases in AST (p<0.001), ALT (p=0.001), and hemoglobin (p=0.004) but the decreases in bilirubin (p=0.199) and INR (p=0.491) were not significant. A positive correlation was found between improvement in liver fibrosis and APRI (p=0.002), AST (p=0.001) and INR (p=0.045). In addition, a negative correlation was found with hemoglobin (p=0.038) and platelet count (p=0.031). It was thought that the positive correlation between the improvement in liver fibrosis, APRI, and AST was due to the regression in inflammation with antiviral treatment, and the positive correlation with INR and the negative correlation with platelet count were related to the improvement in the synthesis function of the liver due to the improvement in fibrosis.

In a study by Bernuth et al.[28] that included 32 CHC patients, liver fibrosis measurements and biochemical scores following combination treatments with sofosbuvir were analyzed and liver fibrosis measurements in the third month after treatment (SVR12) revealed marked regression compared with the onset of treatment values (p=0.016), and reduction in fibrosis severity was noted in 24% of cirrhotic patients (F4). There was also a significant reduction in APRI scores (p<0.001), which was thought to be largely due to the association between the reduction in AST and hepatic inflammation. When APRI was assessed in cut-off values, all of the patients moved to the non-cirrhotic group. Similarly in our study, the patients were evaluated according to APRI cut-off values in the fibrosis groups of mild/no fibrosis (F0-1: APRI<0.5), significant fibrosis (F2-3: APRI=0.5–1.9), and cirrhosis (F4: APRI≥2), and all of the patients were found to be in the non-cirrhotic group according to post-treatment APRI values. This decrease in fibrosis severity was statistically significant (p<0.01).

Conclusion

In our study, a significant regression was found in liver fibrosis measurements and APRI values in patients with CHC following treatment with current antiviral treatments and significant improvement was seen in fibrosis with antiviral treatment with DAAs even at the early stage of following treatment. However, it should be kept in mind that patients who remain in the cirrhotic group after treatment, which was 40% of our study group, are still at risk for cirrhosis complications. A longer period of follow-up and measurements are needed particularly regarding fibrosis changes in patients with advanced fibrosis and cirrhosis. As an easy-to-use and a repeatable non-invasive method, TE and APRI can be useful tools in the evaluation of the dynamic process of fibrosis in CHC and in the prediction of complications due to chronic liver disease.

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