

Dynamic of Transient Elastography after Direct-acting Antiviral Based-therapy in Chronic Hepatitis C Infection

Preeyamas Koonsiripaiboon, MD¹, Supatsri Sethasine, MD¹

¹ Division of Gastroenterology and Hepatology, Department of Medicine, Faculty of Medicine, Vajira Hospital, Navamindradhiraj University, Bangkok, Thailand

Background: Chronic hepatitis C infection (CHC) is a leading cause of liver cancer. CHC treatment goal is sustained virological response (SVR) achievement. Direct-acting antiviral (DAA) therapy has been introduced as a standard treatment for CHC.

Objective: The present study was to assess the impact of CHC treatment using DAA-based therapy on non-invasive assessments of liver fibrosis, namely by transient elastography (TE) fibrosis-4 (FIB-4) and aspartate aminotransferase-to-platelet ratio index (APRI) scores. The secondary objective was to find factors that affected changes in dynamic liver stiffness.

Materials and Methods: A retrospective analysis of data collected from individuals aged 18 to 70 years who were diagnosed with CHC and treated with DAA-based regimens at the Gastroenterology and Liver unit of Vajira Hospital between 2020 and 2021. Patients' attributes were documented in electronic medical E-phis. TE measurements, FIB-4 and APRI were conducted at baseline, 3 and 12 months after the end of (EOT) treatment.

Results: Cirrhosis was diagnosed in 55.3% of the individuals. Among individuals treated with CHC, eighty out of 85 (94.1%) achieved SVR. The majority of participants (95%) who attained SVR showed a notable reduction in liver stiffness. Median TE value decreased significantly from baseline [16.8 (9.2, 24.2)] kPa, 3 months after EOT [12.2 (7.0, 21.3)], and 12 months after EOT [10 (6.0, 18.0) kPa], respectively. A significant reduction in TE was achieved by more than half (56.47%) of the participants. Furthermore, a decrease of >30% in TE value was observed in one-third of individuals diagnosed with cirrhosis. Nevertheless, the presence of baseline cirrhosis or an APRI score >1.5 decreases the probability of experiencing a substantial reduction in TE. The odds ratios for cirrhosis and APRI were 0.13 (95% CI: 0.04 to 0.40, p<0.001) and 0.29 (95% CI: 0.10 to 0.84, p=0.023), respectively.

Conclusion: The one-year dynamic of liver stiffness reduction by transient elastography was demonstrated in the majority of individuals with chronic hepatitis C who achieved a sustained viral response, regardless of the presence of cirrhosis.

Keywords: Aspartate aminotransferase-to-platelet ratio index; Chronic hepatitis C infection; Direct-acting antiviral-based therapy; Fibrosis-4 score; Liver stiffness; Transient elastography

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Chronic hepatitis C infection (CHC) is a leading cause of chronic hepatitis, cirrhosis, and hepatocellular carcinoma (HCC). The global and Thailand prevalence of CHC are 2.8% and 0.94%, respectively. Most patients develop chronic infections that can progress to cirrhosis in the following decades. In the cirrhotic stage, HCC can develop at 1 to 3% per year⁽¹⁻⁴⁾. CHC treatment goal is to

achieve sustained virological response (SVR). A previous study observed improved liver fibrosis and reduced liver-related complications, including HCC, in patients with SVR⁽⁵⁾. Liver biopsy is the gold standard for grading stage of liver fibrosis; however, it is invasive, has a post-biopsy bleeding risk, and may have a sampling error. Moreover, a dynamic fibrosis assessment cannot be done by a repeat biopsy. Recently, liver stiffness measurement using transient elastography (TE) was developed as an alternative tool. It can accurately differentiate each liver fibrotic stage in clinical practice, especially in advanced stage detection, and can be safely repeated at intervals. Furthermore, fibrosis-4 (FIB-4) and aspartate aminotransferase-to-platelet ratio index (APRI) are other non-invasive techniques used to assess fibrosis. These computations are efficient and applicable in any hospital setting^(6,7).

The previous era of pegylated interferon plus ribavirin regimens produced non-sustained efficacy due to various HCV genotypes and severities of liver diseases. Regarding

Correspondence to:

Sethasine S.

Division of Gastroenterology and Hepatology, Department of Medicine, Faculty of Medicine, Vajira Hospital, Navamindradhiraj University, Bangkok 10300, Thailand.

Phone: +66-2-2443480

Email: supatsri@nmu.ac.th

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the side-effects and severity of cirrhosis, the pegylated-interferon-based regimen was unsuitable for advanced cirrhotic patients. Consequently, these patients were unable to undergo the dynamic fibrotic assessment. Recently, direct-acting antiviral (DAA)-based regimens were effective with a high SVR achievement, good compliance, and can treat all liver disease stages. From 2023, Thailand's government policy now allows reimbursement for all CHC patients with active viral load to receive the new DAA-based medication. We were interested in dynamic TE, FIB-4, and APRI changes at baseline and the end of treatment (EOT) at 3 and 12 months.

The present study aimed to (i) evaluate the dynamic TE changes measured using transient elastography and changes in FIB-4 and APRI after successful CHC treatment and (ii) find factors involved in dynamic changes in transient elastography.

Materials and Methods

Study design and populations

Vajira Institutional Review Board approved this study (approval number: 162/2565). Patients diagnosed with CHC, at least within minimum significant fibrosis, who fulfilled the government reimbursement policy criteria were eligible for the DAA-based therapy in the Liver Unit, Faculty of Medicine, Vajira hospital, between October 2019 and October 2021. Participants who met the exclusion criteria were excluded. Therefore, 85 participants met the following eligibility criteria, and their data were retrospectively collected. Either an ultrasound or CT scan demonstrating liver nodularity with left lobe enlargement, or the TE value of >13 kPa, was used to diagnose cirrhosis⁽⁸⁾.

The eligibility following criteria were patients who: (i) received DAA-based therapy, (ii) underwent liver stiffness measurement using TE (FibroScan®) at baseline and 3 and 12 months after EOT, (iii) had laboratory panel including HCV-viral load and genotype and parameters for APRI, and FIB-4 scores and (iv) provided informed consent. The exclusion criteria were: (i) allergy to DAA-based therapy, (ii) pregnancy, (iii) active alcohol drinking, (iv) substance abuse, (v) organ transplant history, and (vi) active cancer, including HCC.

Liver fibrosis assessment using transient elastography (FibroScan®)

Prior to the assessment, the patient had to be fasting. The FibroScan® was done with an ultrasound probe, 50 Hz waves, using the elastic modulus equation ($E=3pv^2$) to calculate the liver tissue elasticity, represented in kilopascal (kPa), and high fibrosis degree resulted in high velocity of shear waves and elastic modulus. TE for each patient was calculated as a median value of 10 consecutive kPa

values, with an acceptable interquartile range/median value of <30%. This measurement cannot be done with ascites, morbidly obesity (body mass index [BMI] above 30 kg/m²), and pregnancy. A value of 7 kPa was a significant fibrosis threshold, while >13 kPa indicated cirrhosis^(4-6,8,9).

Liver fibrosis assessment using APRI and FIB-4 were calculated as follows link: <https://www.hepatitisc.uw.edu/page/clinical-calculators/apri> <https://www.mdcalc.com/calc/2200/fibrosis-4-fib-4-index-liver-fibrosis>.

FIB-4 score of <1.45 has a negative predictive value of 95% for advanced fibrosis (fibrosis stage 3 to 4), while >3.25 give 98% specificity for advanced fibrosis or cirrhosis. Values within 1.45 to 3.25 are between F1 and F3 with modest calculation accuracy⁽¹⁰⁾. In contrast, APRI is generally an adjunct test with less accuracy, the value >0.7 has sensitivity and specificity (77%; 72%) for stage 2 fibrosis or more, and >1 has sensitivity and specificity (61 to 76%: 64 to 72%) for stage 3 and 4^(7,11-13).

Statistical analysis

The data were presented using either the mean ± standard deviation or the median and interquartile range to characterize the characteristics of the patients. The Chi-squared test or Fisher's exact test was used to compare categorical variables. Continuous variables were compared using either a t-test or a Mann-Whitney U-test. The study employed linear mixed model regression analysis to compare the changes in TE following DAA-based therapy in individuals with chronic hepatitis C. Factors associated with TE reduction were determined using logistic regression analysis. The results were expressed as odds ratios (OR), 95% confidence intervals (CI), and p-values. All analyses were performed using STATA version 15 (Stata Corp, College Station, TX, USA). A p-value of less than 0.05 was considered significant.

Results

Baseline characteristics

The total number of eligible patients with CHC treated using a DAA-based regimen was 85, and the mean age was 54±9.9 years, with 37 (43.5%) being males. The mean BMI was 24.3±4 kg/m². Prevalence of HCV genotype 1, 3, 6, and 2 were 47%, 36.5%, 15.3%, and 1.2% respectively. Approximately half (55.3%; n=47) of the patients had baseline cirrhosis, 11 (12.9%) had type 2 diabetes, and 3 (3.5%) were HIV positive. Mean baseline alanine transaminase (ALT) indicated active inflammation (108.6±70 IU/L). Our patients had a median TE of 16.8 (9.2, 24.2) kPa, which was within the cirrhotic range with moderate steatosis (controlled attenuation parameter (CAP) 228.6±50 dB/m). Meanwhile, median FIB-4 and APRI were within moderate fibrosis degree 1.7 (0.9,4) and 1.2 (0.6,

2.5), respectively.

All the patients received one of the four available DAA-based therapy; 29 (34.1%) were treated with sofosbuvir/PEG-IFN/ribavirin for genotype 3 with/without cirrhosis, 28 (33%) with sofosbuvir/ledipasvir/ribavirin for genotype 1 with cirrhosis, and 24 (28.2%) with sofosbuvir/ledipasvir for genotype 1 without cirrhosis. Only four (4.7%) patients were treated with sofosbuvir/velpatasvir (Table 1).

Most patients (94%; n=80) achieved SVR after treatment, and they had lower baseline TE, FIB-4, and APRI than the those who did not; (8.7 kPa vs. 16.9 kPa, p=0.07); (1.6 vs. 2; p=0.5) and (1.1 vs. 1.7; p=0.3), respectively (Table 2).

Regarding the SVR, there were no differences observed in body mass index, degree of hepatic steatosis, or HCV genotypes. Five patients who did not achieve SVR had baseline TE and APRI values that indicated cirrhosis stage. These participants also did not experience any reduction in TE.

Table 1. Baseline characteristics of patients (n=85)

	All (n=85)
Age (years)	54±9.9
Sex (%)	
Male	37 (43.5)
Genotype (%)	
1	40 (47)
2	1 (1.2)
3	31 (36.5)
6	13 (15.3)
Underlying disease	
Human immunodeficiency virus infection	3 (3.5)
Diabetes mellitus	11 (12.9)
Cirrhosis	47 (55.3)
BMI (kg/m ²)	24.3±4
DAA regimen (%)	
Sofosbuvir/pegylated interferon /ribavirin	29 (34.1)
Sofosbuvir/ledipasvir/ribavirin	28 (33)
Sofosbuvir/ledipasvir	24 (28.2)
Sofosbuvir/velpatasvir	4 (4.7)
ALT (IU/L)	108.6±70
AFP (IU/ml)	12.7±22
CAP (dB/m)	228.6±50
APRI*	1.2 (0.6, 2.5)
FIB-4*	1.7 (0.9, 4)
Transient elastography (kPa)*	16.8 (9.2, 24.2)

* Data are presented as number (%) or mean ± standard deviation and median (Interquartile range).

AFP=alpha fetoproteins; ALT=alanine transaminase; APRI=aspartate aminotransferase-to-platelet ratio index; BMI=body mass index; CAP=controlled attenuation parameter; DAA=directed-acting antiviral; FIB-4=fibrosis-4

Approximately half (53.2%; 25 patients) of patients with cirrhosis were infected with HCV genotype 1. Patients diagnosed with cirrhosis were more likely to have a comorbidity of diabetes compared to those without cirrhosis (21.3% vs. 2.6%, p=0.02). All individuals with cirrhosis had TE measurements above 13 kPa, ranging from 16.8 to 32 kPa. However, when using a FIB-4 cut point of > 3.25, only 47% (n=22) of cases were identified with cirrhosis (Table 3).

The dynamic of transient elastography, FIB-4 and APRI reduction

There was a notable and consistent reduction in TE over the follow-up period. The baseline measurement of TE was 16.8 kPa, which decreased to 12.2 kPa (7.0, 21.3) at 3 months after the EOT, and further decreased to 10.0 kPa (6.0, 18.0) at 12 months after EOT. A significant decrease in median APRI was seen from baseline to 3 months after EOT, with values changing from 1.2 (0.6 to 2.5) to 0.5 (0.3, 0.9). A decrease in FIB-4 was seen only at 3 months after the EOT [1.7 (0.9, 4) vs. 1.0 (0.5, 2.8); p<0.001] (Table 4).

Within a period of 3 months following the EOT, the mean ALT levels of most patients decreased significantly from being higher than twice the upper normal limit (UNL) to reaching values close to the normal range. Nevertheless, there was no reduction in FIB-4 from 3 to 12 months after the EOT, which could be due to other factors influencing transaminase levels, such as medication or body weight.

Regarding virologic outcomes, most patients who achieved SVR presented significant improvements in TE over time (16.9 kPa at baseline, 13.1 kPa and 10.1 kPa at 3 and 12 months respectively. Nevertheless, four patients in the SVR group did not have any reduction in TE. Conversely, TE reduction was not observed in all non-SVR patients (Figure 1).

Sixty percent (n=48) of patients who attained SVR had a substantial reduction in TE of 30% or more. However, there were no appreciable differences in BMI, degree of liver steatosis, or HCV viral load among those in the substantial TE reduction group compared with those who did not (Table 5).

According to our multivariate Cox proportional regression analysis, the presence of baseline cirrhosis or an APRI score >1.5 decreases the probability of experiencing a substantial reduction in TE. The odds ratios for cirrhosis and APRI were 0.13 (95% CI: 0.04 to 0.40, p<0.001) and 0.29 (95% CI: 0.10 to 0.84, p=0.023), respectively (Table 6).

Discussion

At the time of our study, along with the National primary drug account, the Government policy provides CHC treatment for all patients with at least one stage of significant liver fibrosis with active HCV viral load. The initiation of

Table 2. Baseline characteristics based on virologic response achievement

	SVR (n=80)	Non-SVR (n=5)	p-value
Age (years)	54.7±9	43.8±12	0.02
Male (%)	45 (56.3)	3 (60)	1.0
Genotype (%)			0.9
1	37 (46.2)	3 (60)	
2	1 (1.3)	0	
3	29 (36.3)	2 (40)	
6	13 (16.2)	0	
BMI (kg/m ²)	24.3±4	22.8±2	0.4
DAA regimen (%)			0.03
Sofosbuvir/pegylated interferon/ribavirin	28 (35)	1 (20)	
Sofosbuvir/ledipasvir/ribavirin	27 (33.7)	1 (20)	
Sofosbuvir/ledipasvir	23 (28.8)	1 (20)	
Sofosbuvir/velpatasvir	2 (2.5)	2 (40)	
AFP (ng/mL)	12.3±22	27.7±30	0.3
CAP (dB/m)	229.1±51	220.4±19	0.7
APRI*	1.1 (0.5, 2.6)	1.7 (1.2, 1.8)	0.3
FIB-4*	1.6 (0.9, 4)	2 (1.7, 4)	0.5
Transient elastography* (kPa)	8.7 (7.6, 10.6)	16.9 (9.9, 24.4)	0.07

* Data are presented as number (%), mean ± standard deviation, or median (Interquartile range).

AFP=alpha fetoproteins; APRI=aspartate aminotransferase-to-platelet ratio index; BMI=body mass index; CAP=controlled attenuation parameter; DAA=directed-acting antiviral; FIB-4=fibrosis-4; SVR=sustained virologic response

Table 3. Baseline characteristics based on cirrhosis status

	Cirrhosis (n=47)	Non-cirrhosis (n=38)	p-value
Age (years)	54.7±10	53.3±10	0.5
Male (%)	26 (55.3)	22 (57.9)	0.8
Genotype (%)			0.5
1	25 (53.2)	15 (39.5)	
2	0	1 (2.6)	
3	16 (34)	15 (39.5)	
6	6 (12.8)	7 (18.4)	
BMI (kg/m ²)	24.3±4	24.2±5	0.9
Diabetes mellitus (%)	10 (21.3)	1 (2.6)	0.02
DAA regimen (%)			
Sofosbuvir/ledipasvir	3 (6.4)	21 (55.3)	
Sofosbuvir/ledipasvir/ribavirin	28 (59.6)	0	
Sofosbuvir/pegylated interferon/ribavirin	15 (31.9)	14 (36.8)	<0.001
Sofosbuvir/velpatasvir	1 (2.1)	3 (7.9)	
AFP (ng/mL)	12.4±14	13.1±31	0.9
CAP (dB/m)	230.8±60	226.7±39	0.7
APRI*	1.7 (0.9, 3.8)	0.7 (0.5, 1.2)	<0.001
FIB-4*	3.5 (1.4, 5.3)	1.1 (0.7, 1.8)	<0.001
Transient elastography (kPa) *	22.5 (16.8, 32)	9.6 (8.1, 14.3)	<0.001

* Data are presented as number (%), mean ± standard deviation, or median (Interquartile range).

AFP=alpha fetoproteins; APRI=aspartate aminotransferase-to-platelet ratio index; BMI=body mass index; CAP=controlled attenuation parameter; DAA=directed-acting antiviral; FIB-4=Fibrosis-4

treatment based on DAA coincided with the study period, utilizing broad criteria for the diagnosis of cirrhosis.

For this reason, at baseline, the majority of eligible

patients had advanced fibrosis, and about half already had cirrhosis. TE was the best in diagnosing cirrhosis, especially in patients for whom imaging ultrasound or estimated APRI

Table 4. Dynamic changes in hepatic steatosis and non-invasive test for liver fibrosis after DAA-based therapy in chronic hepatitis C

	Pretreatment (0)	EOT3mo (1)	EOT12mo (2)	p	p (0 vs. 1)	p (0 vs. 2)	p (1 vs. 2)
CAP (dB/m)	228.6±50	228.1±47	219.1±52	0.3	0.9	0.1	0.2
APRI*	1.2 (0.6, 2.5)	0.5 (0.3, 0.9)	0.5 (0.3, 0.8)	<0.001	<0.001	<0.001	0.04
FIB-4*	1.7 (0.9, 4)	1.0 (0.5, 2.8)	2.0 (1.1, 3.3)	<0.001	<0.001	0.08	<0.001
Transient elastography (kPa)	16.8 (9.2, 24.2)	12.2 (7.0, 21.3)	10 (6.0, 18.0)	<0.001	<0.001	<0.001	<0.001

Data are presented as mean ± standard deviation or median (interquartile range).

APRI=Aspartate aminotransferase-to-platelet ratio index; CAP=Controlled attenuation parameter; EOT 3 mo=3 months after the end of treatment; EOT 12 mo=12 months after the end of treatment; FIB-4=Fibrosis-4

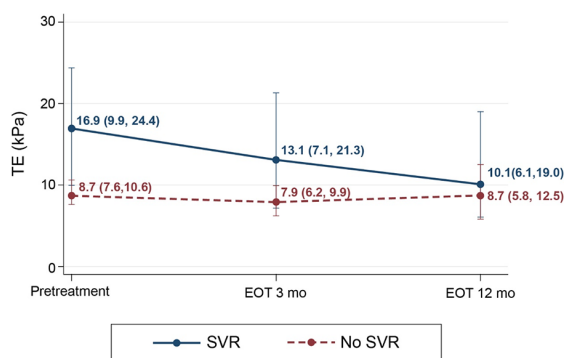


Figure 1. Dynamic changes in transient elastography in the SVR and non-SVR groups at baseline, 3 months, and 12 months after the end of treatment. EOT=end of treatment; SVR=sustained virologic response; TE=transient elastography

and FIB-4 were inconclusive.

The present study reported the high efficacy of the DAA-based regimen, with 94.1% of patients who achieved SVR. All non-SVR patients had cirrhosis at baseline with no TE reduction 12 months after EOT. Regardless of fibrosis severity, we demonstrated a dynamic reduction of TE from baseline to one year after EOT in most patients who achieved SVR. Previous studies reported the proportion of treated CHC patients who experienced significant TE reduction, a 30% reduction in TE value at 12 months⁽¹³⁾ and further significant TE value reduction at ≥24 months⁽¹⁴⁻¹⁹⁾.

Our study demonstrated a higher rate of significant TE reduction with DAA-based regimens at one-year follow-up (60% vs. 48.6%)⁽¹³⁾. The WHO aims that all CHC infections should be eligible for DAA-based regimens by 2030. Interestingly, therapy benefit was observed in over half of our patients who achieve a substantial TE reduction after HCV viral load eradication. Most patients who achieved a step of TE reduction had significant fibrosis or advanced non-cirrhotic stage at baseline. The success rate in liver restoration was preserved, supporting the earlier stage of treatment initiation.

Through CHC treatment, cirrhosis and hepatocellular carcinoma are averted. In the group of patients with cirrhosis, DAA therapy resulted in TE reduction in most patients and TE decline to the advanced F3 stage in

approximately one-third of the patients. Nevertheless, we discovered that the regression model indicated that the likelihood of a reduction in TE decreased with increasing liver disease severity. However, most patients with cirrhosis achieved TE decline. Moreover, the number of patients whose TE readings were in the baseline cirrhosis but fell below 13 KPa at the 12-month follow-up. This information is pivotal as it demonstrates not just reduction but a shift out of the cirrhotic range. Due to the heterogeneity of the residual HCC risk after SVR, the cost-effectiveness of HCC screening programs in clinical practice recommendations for patients in the advanced stage of liver disease was the subject of discussion^(4,20-23). Fortunately, the majority of our patients with an advanced F3 stage at baseline experienced TE reduction. Regarding liver stiffness measurement, TE reduction to minimal disease was achieved in 46% of the patients with advanced stage F3 at baseline.

The TE reduction after treatment should be affected by multiple factors. Aside from diminishing liver fibrosis, the mitigation of liver inflammation following treatment is important factor for reducing TE. Therefore, the regression of fibrosis may need to be confirmed with liver biopsy, although it is not necessary in practical terms. It is important to be cautious when interpreting the decrease in TE after SVR, particularly in individuals who had advanced fibrosis or cirrhosis at baseline.

For alternative methods of assessing liver fibrosis without invasive procedures, FIB-4 and APRI demonstrate moderate accuracy in detecting fibrosis at an intermediate stage. Therefore, it may be convenient to use pretreatment FIB-4 and APRI in a community hospital setting. However, the majority of the decrease in FIB-4 and APRI following SVR is attributed to decreased hepatic inflammation rather than fibrosis improvement. However, the simultaneous use of various non-invasive diagnostics in clinical settings has proven to be beneficial in evaluating the extent of liver disease.

The study's limitation was the absence of sequential liver biopsies to correlate with TE values, therefore we cannot demonstrate the dynamic improvement of both necroinflammation and fibrosis following DAA treatment.

Our study demonstrated the advantages of DAA-based

Table 5. The characteristics of chronic hepatitis C patients who attained SVR according to the degree of transient elastography reduction

	More than 30% of TE reduction (n=48)	Less than 30% of TE reduction (n=32)	p-value
Pretreatment HCV Viral load (IU/mL)	1,347,888	1,519,511	0.852
Diabetes mellitus	5 (10.4)	6 (18.75)	0.543
Cirrhosis	16 (33.3)	26 (81.3)	<0.001
Pretreatment CAP (dB/m)	228±40	229.4±60.5	0.892
Genotype			
1	20 (41.7)	16 (50)	0.544
2	1 (2.1)	0	
3	18 (37.5)	12 (37.5)	
6	9 (18.7)	4 (12.5)	
BMI [kg/m ²]	23.76±3.60	24.93±4.35	0.179
APRI	0.7 (0.5, 1.5)	1.8 (1, 4.2)	<0.001
>1.5	12 (25)	24 (75)	<0.001
>2	7 (14.6)	18 (56.3)	0.001

Data are presented as number (%), mean ± standard deviation, or median (Interquartile range).

APRI=Aspartate aminotransferase-to-platelet ratio index; CAP=Controlled attenuation parameter; SVR=sustained virologic response; TE=transient elastography

Table 6. Univariate and multivariate analysis of factors associated with substantial transient elastography reduction

	Univariate analysis			Multivariate analysis*		
	OR	95% CI	p-value	OR	95% CI	p-value
Cirrhosis	0.09	0.03, 0.27	<0.001	0.13	0.04, 0.40	<0.001
APRI >1.5	0.18	0.07, 0.46	<0.001	0.29	0.10, 0.84	0.023
APRI >2	0.18	0.06, 0.50	0.001	0.27	0.08, 0.86	0.027

* Variable was included in the multivariate model due to a p<0.050 in the univariate analysis.

APRI=Aspartate aminotransferase-to-platelet ratio index; CI=confident interval; OR=odds ratio

therapy, which encompassed the complete elimination of HCV viral infection and a decrease in TE. Patients in the advanced fibrotic stage may undergo successful decrease of TE leading to exemption from HCC surveillance.

Conclusion

The one-year dynamic of liver stiffness reduction by transient elastography was demonstrated in the majority of individuals with chronic hepatitis C who achieved a sustained viral response, regardless of the presence of cirrhosis.

What is already known on this topic?

Following treatment with direct-acting antiviral-based regimens, most patients with chronic hepatitis C showed a reduction in transient elastography.

What this study adds?

The efficacy of direct-acting antiviral therapy in transient elastography reduction was shown in the majority of patients with chronic hepatitis C who achieved sustained virologic response, regardless of the presence of liver

cirrhosis.

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Conflicts of interest

The authors declare no conflict of interest.

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