Influence of Meal Intake on Liver Stiffness in Patients with Chronic Hepatitis B and C

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Background: *Transient elastography (TE) is a non-invasive test for evaluation of fibrosis in chronic hepatitis B (CHB) and chronic hepatitis C (CHC). Meal intake has been found to affect liver stiffness (LS) values in CHC patients, but there is still lack of data in CHB patients.*
Objective: *Evaluate the influence of meal intake on LS values by TE in non-cirrhotic CHB patients and compare its effect with non-cirrhotic CHC patients.*
Material and Method: *Forty-five CHB patients and 37 CHC patients were included. LS measurements by TE were done at three different times including 4-hour fasting, immediately, and 60 minutes after finishing 500 kcal meal.*
Results: *Mean fasting LS values in CHB patients were 5.40±1.7 kPa. LS values in CHB patients significantly increased at both immediately and 60 minutes after finishing meal by 0.31±0.1 kPa (p = 0.035) and 0.33±0.1 kPa (p = 0.018), respectively. Difference in the peak changes of LS values after meal were not significant between CHB and CHC patients (CHB 0.72±0.1 vs. CHC 1.16±0.1, p = 0.076). No other variables associated with the changes of LS values after meal in either CHB patients or CHC patients.*
Conclusion: *Meal intake significantly increases LS values in CHB and CHC patients. It was considered to be a confounding factor in LS measurements. An appropriate time of fasting should be done before LS measurement in both CHB and CHC patients.*

Keywords: *Meal, Liver stiffness, Transient elastography, Chronic hepatitis B, Chronic hepatitis C*

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Liver fibrosis assessment is a part in management of chronic liver diseases (CLD), especially chronic hepatitis B (CHB) and chronic hepatitis C (CHC). Gold standard for evaluation in staging of liver fibrosis is the liver biopsy[1-4]. However, it is an invasive procedure with minimal but significant complications such as bleeding in 0.04% and mortality in 0.001%. Furthermore, it may have sampling errors as well as intra- and inter-observer variability[5-7].

Transient elastography (TE) (Fibroscan®, Echosens, Paris, France) is a non-invasive test for liver stiffness (LS) measurement, which is performed by the transmission of a mechanical wave, generated by vibration[8]. Nowadays, strong correlation between LS measurements and liver fibrosis stages has been demonstrated in many chronic liver diseases including CHB and CHC[9,10].

Confounding factors have been found in LS values. They affect the diagnostic accuracy. These include hepatic inflammation[11,12], cholestasis[13] and liver congestion[14]. Another factor, meal, may also influence LS. The study by Mederacke et al presented that LS values in CHC patients significantly increased immediately, 15 minutes, and 60 minutes after finishing meal, and normalized 180 minutes after finishing meal[15]. The physiological changes have been suggested to result by the increase in portal pressure after meal, known as postprandial hyperemia[16,17]. Nevertheless, there is no study that evaluates the effect of the meal in CHB patients, which is also a major group of chronic hepatitis patients and compare this effect between CHB and CHC patients.

The aim of the present study was to evaluate the influence of meal intake on the LS values in non-cirrhotic CHB patients and compare this effect with CHC patients.

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Material and Method

Patients

All patients with CHB and CHC who visited the outpatient liver clinic of the NKC institute of Gastroenterology and Hepatology, Songkhla, Thailand between October 2011 and December 2011 were included in the study.

Inclusion criteria were age 18 to 75 years and diagnosis of CHB or CHC for more than six months before enrollment. CHB was diagnosed by positive HBsAg. CHC was diagnosed by positive anti-hepatitis C virus (HCV) and detectable HCV-RNA. Exclusion criteria were cirrhosis as documented by either physical examination, laboratory, radiology, or pathology, vascular disease of the liver, the presence of any focal hepatic lesions, the presence of ascites by ultrasonography, body mass index (BMI) more than 40 kg/m^2, co-infection with HIV, combination of other chronic liver diseases, pregnancy, congestive heart failure, alanine aminotransferase (ALT) above five times upper limit of normal (ULN) within four weeks before enrollment, and refuse to participate in the study. Written informed consent was obtained from all patients before enrollment. The study was approved by the Ethical Committee of Prince of Songkla University and in accordance with Helsinki Declaration of 1975.

Flow chart of the study and cause of exclusion were summarized in Fig. 1.

End points

The primary end point was changes in LS values between fasting and immediately and 1-hour after finishing meal in CHB patients. The secondary end points were the differences in the change of LS values after meal between CHB and CHC patients and association between variables.

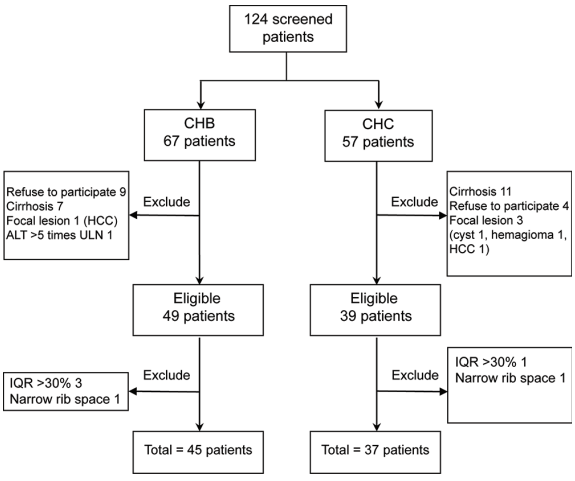

Fig. 1 Flow chart of the study.

Study design

All of the eligible patients were collected clinical and laboratory data and underwent LS measurement by TE on the same day. The following data were determined, age, gender, BMI, mean arterial pressure (MAP), liver function tests, and fasting plasma glucose (FPG).

All patients underwent LS measurements at three different times. First measurement was performed after a 4-hour fasting ($LS_{fasting}$). After that, each patient had intake a standardized Thai meal (approximately 500 kcal, 55% carbohydrate, 15% protein, 30% fat) over a maximum period of 30 minutes. Second and third LS measurements were performed immediately (LS_{0min}) and 60 minutes after finishing meal (LS_{60min}), respectively.

LS measurements by TE

LS was determined using Fibroscan® (Echosens, Paris, France) as described by Sandrin[8]. TE operators were two physicians who had previously performed LS by fibroscan at least 30 patients (Chamroonkul N and Witeerungrot T). These two investigators were validated and had low interpersonal variability. All measurements in the same patients were performed at the same position and location. The operators performed at least 10 valid measurements at each time. The results of a LS determination were expressed in median (kilopascal; kPa). Patients with interquartile range (IQR) over median ratio more than 30% and success rate less than 80% were excluded from the study.

Statistical analysis

Sample size was calculated based on CHC patients data from Mederacke et al study[15] and two related group method. Sample size needed to be at least 36 patients in each groups to detect an increase 1.4 kPa in LS values after finishing meal (peak values after meal and fasting stiffness was 6.3±2.1 kPa and 4.9±0.8 kPa, respectively) with assuming a standard deviation of 2.1 kPa as significant at an alpha of 0.05 and power of 80%.

The baseline characteristics were presented as frequencies and percentages or means and SDs. Comparison of baseline characteristics between CHB

and CHC were using Chi-square test for percentage data and independent-sample t test for mean data. Differences in mean LS values at different time were analyzed using paired-samples t test. Independent-samples t test was used to evaluate the differences in the changes of LS values after meal between CHB and CHC patients. The baseline characteristics were analyzed using independent-sample t test or Pearson correlation to find association with the changes of LS values. Statistical significance was set at $p<0.05$.

Results

Characteristics of patients

As shown in Fig. 1, 124 patients were screened and included 67 CHB and 57 CHC patients (Fig. 1). The two most common causes of exclusion were cirrhosis (18 patients) and refused to participate in the study (13 patients). Forty-nine CHB patients and 39 CHC patients were eligible patients and underwent LS measurement. However, IQR over median ratio more than 30% and failure of LS measurement due to narrow rib space were found in four and two patients, respectively. These patients were also excluded from the study. Finally, 45 CHB and 37 CHC patients were included in the present study.

Male and female genders were nearly equal in CHB patients (male 23 patients; female 22 patients). The patients with CHB patients were significant older than CHC patients (CHB 47.7±10.1 years; CHC 39.7±9.4 years; $p<0.001$). Total protein was only another factors that was different between both groups (CHB 7.5±0.6 g/dL, CHC 7.8±0.5 g/dL, $p = 0.022$). Baseline characteristics of all patients were shown in Table 1.

Influence of meal intake on LS values

LS values significantly increased at both post-prandial times in both CHB and CHC patients (Table 2). Mean increased LS values between fasting ($LS_{fasting}$) and immediately after finishing meal (LS_{0min}) and 60 minutes after finishing meal (LS_{60min}) in CHB patients were 0.31±0.1 kPa ($p = 0.035$) and 0.33±0.1 kPa ($p = 0.018$), respectively and in CHC patients were 0.8±0.2 kPa ($p = 0.001$) and 0.57±0.2 kPa ($p = 0.006$) respectively. The maximal increment of LS values after meal (LS_{max}-$LS_{fasting}$) in CHB and CHC patients were 0.72±0.1 kPa ($p<0.001$) and 1.16±0.2 kPa ($p<0.001$), respectively.

The proportion of patients that had increased LS values after meal was more than decreased LS values in both CHB and CHC patients (CHB 73% vs. 20%; CHC 70% vs. 22%). Moreover, the proportion of patients that had at least 1 kPa increment of LS values were highly found in both groups that were 44% of CHB patients and 59% of CHC patients (Table 3).

Table 1. Baseline characteristics of the 82 patients included in the study

Characteristics	CHB (n = 45)		CHC (n = 37)		*p*-value
	Mean ± SD	Min, max	Mean ± SD	Min, max	
Male - patients (%)	23 (51.1%)	-	25 (67.6%)	-	0.132
Age (years)	47.7±10.1	24-72	39.7±9.4	19-64	<0.001
BMI (kg/m^2)	24.2±3.5	13.3-31.6	23.0±3.3	17.5-30.8	0.145
MAP (mmHg)	94.5±11.4	73-114	91.7±13.1	67-115	0.311
Direct bilirubin (mg/dL)	0.16±0.1	0.02-0.53	0.18±0.1	0.05-0.50	0.428
Total bilirubin (mg/dL)	0.6±0.3	0.2-1.8	0.5±0.3	0.10-1.16	0.094
AST (mg/dL)	29.3±15.1	15-104	33.1±20.0	13-91	0.345
ALT (mg/dL)	31.7±23.2	7-148	40.9±36.3	6-146	0.185
ALP (mg/dL)	66.6±21.1	29-135	70.9±16.0	47-108	0.309
Total protein (g/dL)	7.5±0.6	5.4-8.8	7.8±0.5	6.7-8.8	0.022
Albumin (g/dL)	4.5±0.5	1.6-5.3	4.6±0.2	4.0-5.1	0.159
FPG (mg/dL)	99.4±29.1	62-233	90.6±14.1	69-149	0.101

AST = aspartate aminotransferace; ALT = alanine aminotransferase; BMI = body mass index; CHB = chronic hepatitis B; CHC = chronic hepatitis C; FPG = fasting plasma glucose; MAP = mean arterial pressure
Statistical significance were calculated by independent-sample t test except gender data which using Chi-square test

Table 2. Mean liver stiffness values at fasting, immediately after meal and 1h after meal

	Liver stiffness (kPa): mean±SD	
	CHB (n = 45)	CHC (n = 37)
Fasting	5.40±1.7	6.09±2.8
0 min after meal	5.71±1.8	6.88±3.0
60 min after meal	5.73±1.7	6.66±2.6
Max	6.12±1.8	7.25±2.9
0 min-fasting	0.31±0.1	0.80±0.2
p-value	0.035	0.001
60 min-fasting	0.33±0.1	0.57±0.2
p-value	0.018	0.006
Max-fasting	0.72±0.1	1.16±0.2
p-value	<0.001	<0.001

CHB = chronic hepatitis B; CHC = chronic hepatitis C
Statistical significance were calculated by paired-samples t test

Table 3. Proportions of the patients according to the changes in liver stiffness values after meal

	Change in LS	Number (%)	
		CHB (n = 45)	CHC (n = 37)
0 min-fasting	Increase	26 (58)	23 (62)
	Increase >1 kPa	12 (27)	14 (38)
	Decrease	16 (36)	10 (27)
	Decrease >1 kPa	6 (13)	3 (8)
	Not change	3 (6)	4 (11)
60 min-fasting	Increase	28 (62)	25 (68)
	Increase >1 kPa	12 (27)	15 (41)
	Decrease	15 (33)	12 (32)
	Decrease >1 kPa	4 (9)	4 (11)
	Not change	2 (5)	0
Max-fasting	Increase	33 (73)	26 (70)
	Increase >1 kPa	20 (44)	22 (59)
	Decrease	9 (20)	8 (22)
	Decrease >1 kPa	2 (4)	1 (3)
	Not change	3 (7)	3 (8)

CHB = chronic hepatitis B; CHC = chronic hepatitis C; LS = liver stiffness

Influence of underlying CHB and CHC on LS values after meal

The peak changes in LS values was non-statistical significant higher in CHC patients than in CHB populations by 0.44±0.2 kPa ($p = 0.076$).

Influence of other variables on LS values after meal

Among the baseline characteristic data, there is no variables that associated with the changes of LS values after meal in either CHB patients or CHC patients.

Discussion

In management of CHB and CHC patients, liver fibrosis evaluation is an important part for severity assessment and treatment decision. Although liver biopsy is the gold standard to determine the stage of fibrosis, it is an invasive procedure with minimal but significant risk of morbidity and mortality. LS measurements from TE have a satisfied correlation with the stage of fibrosis from liver pathology, thus becoming an optional tool for liver fibrosis assessment. Unfortunately, confounding factors including hepatic inflammation, cholestasis, and liver congestion affect the diagnostic accuracy[11-14]. Another factor, meal was also considered as confounding factors in CHC patients[15,18].

Since CHB and CHC have different associated factors that are steatosis and insulin resistance, these factors may influence different LS values after meal between CHB and CHC patients. The present study was the first study that evaluated meal effect to LS values in non-cirrhotic CHB patients. LS values in CHB patients were significantly increased after meal by 0.72 kPa or 15.16% while LS values in CHC patients were significantly increased by 1.16 kPa or 22.67%. This finding correlates with Mederacke et al and Arena et al studies. Mederacke et al study reported increased LS values after meal of 1.4 kPa in the group that had fasting LS values less than 6 kPa[15]. Arena et al study reported increased LS values after meal of 1.9 kPa or 33.6% in the group that had Metavir fibrosis stage 0-1 from liver histology[18]. These groups of patients were similar to CHC patients from the present study. The peak changes of LS values after meal in CHC patients trended to be higher than in CHB patients. Nevertheless, these findings were a secondary outcome and the sample size in the present study was not calculated to answer this question. A study that evaluates this difference as a primary outcome is required.

Importantly, about half of patients, which were 44% in CHB and 59% in CHC, had increased LS values after a meal, of more than 1 kPa. The LS values can change from insignificant to significant fibrosis in many patients and mislead the physician management.

The time of increased LS values after meal is still controversial. Mederacke et al study allowed eight pilot patients to ingest 600 kcal continental breakfast within 30 minutes and found the peak LS values

immediately after finishing meal (30 minutes after the onset of the meal) and still significantly elevated until 60 minutes after finishing meal (90 minutes after the onset of the meal). LS values returned to baseline at 180 minutes after finishing meal (210 minutes after the onset of the meal)[15]. Arena et al allowed the patients to ingest 600 kcal liquid meal within only five minutes. That study found the peak at 30 minutes after the onset of the meal and still significantly elevated until 60 minutes after the onset of the meal. LS values returned to baseline at 120 minutes after the onset of the meal[18]. Otherwise, studies using Doppler sonography or direct vascular measurements also showed marked increase in portal blood flow 30 minutes after the onset of the meal[19-23].

In the present study, the authors allowed the patients to ingest 500 kcal meal within 30 minutes. CHB patients was found to have higher LS values at 60 minutes after finishing meal, but CHC patients was found to have higher LS values immediately after finishing meal. However, the present study was not designed to evaluate this question because LS measurements were done at only two time points after finishing meal.

Therefore, time to peak of LS values after meal is likely 30 minutes after the onset of the meal, and time of increased LS values after meal may range from 120 to 210 minutes after the onset of the meal[15,18]. However, variation in meal types, calories intake, ingested times, and time parameters (time after finishing meal vs. time after the onset of the meal) were found in different studies. Further evaluations are need to verify these data. In the view of the staging of fibrosis, the LS values at fasting were not less accurate in predicting stage of fibrosis than any of the LS values at any after meal times[18].

Different LS values after meal have been proposed as the result of postprandial hyperemia. Increased portal blood flow and consequent hepatic microcirculation adaptation after meal are the main physiological changes that lead to increased rigidity of liver tissue[16,17]. However, this theory is still not definitely conclusive. Other factors that may be associated with LS values after meal have been recently published. Study in cirrhosis patients from Berzigotti et al showed the lack of correlation between the changes of LS values after meal with portal blood flow and hepatic venous pressure gradient, but unexpectedly found significant negative correlation with the hepatic artery blood flow (HABF). Less change in HABF refer to increased hepatic blood supply on its arterial site due to severe portal hypertension thus, impair hepatic artery buffer response. Finally, elevated liver perfusion occurs and leads to increase LS values after meal[24].

Conclusion

Meal intake significantly increases LS values in CHB patients as they do in CHC patients. To avoid this effect, LS measurements should be done at fasting or at least 3.5 hours (210 minutes) after the onset of the meal.

What is already known on this topic?

Few studies showed the influence of food with liver stiffness values in CHC patients[15,18]. However, data lacks in CHB patients, which is also a major group of chronic hepatitis patients in Thailand.

What this study adds?

Meal intake significantly increases LS values in CHB patients as same as CHC patients, and should be considered as one of the confounding factors in LS measurements. An appropriate time of fasting is necessary before LS measurements in both CHB and CHC patients.

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Potential conflicts of interest

Teerha Piratvisuth has received advisory board from BMS, MSD, NOVARTIS, and ROCHE, research grants from BAYER, BMS, MSD, NOVARTIS, and ROCHE, and speaker honorarium from BMS, GSK, MSD, NOVARTIS, and ROCHE. Warisara Tangpradabkiet, Surat Praneenararat, Naichaya Chamroonkul, and Teepawit Witeerungrot declare that they have no conflict of interest.

References

1. Lok AS, McMahon BJ. Chronic hepatitis B: update 2009. Hepatology 2009; 50: 661-2.

2. European Association for the Study of the Liver. EASL clinical practice guidelines: Management of chronic hepatitis B virus infection. J Hepatol 2012; 57: 167-85.
3. Ghany MG, Strader DB, Thomas DL, Seeff LB. Diagnosis, management, and treatment of hepatitis C: an update. Hepatology 2009; 49: 1335-74.
4. European Association for the Study of the Liver. EASL Clinical Practice Guidelines: management of hepatitis C virus infection. J Hepatol 2011; 55: 245-64.
5. Regev A, Berho M, Jeffers LJ, Milikowski C, Molina EG, Pyrsopoulos NT, et al. Sampling error and intraobserver variation in liver biopsy in patients with chronic HCV infection. Am J Gastroenterol 2002; 97: 2614-8.
6. Bedossa P, Dargere D, Paradis V. Sampling variability of liver fibrosis in chronic hepatitis C. Hepatology 2003; 38: 1449-57.
7. Rockey DC, Caldwell SH, Goodman ZD, Nelson RC, Smith AD. Liver biopsy. Hepatology 2009; 49: 1017-44.
8. Sandrin L, Fourquet B, Hasquenoph JM, Yon S, Fournier C, Mal F, et al. Transient elastography: a new noninvasive method for assessment of hepatic fibrosis. Ultrasound Med Biol 2003; 29: 1705-13.
9. Friedrich-Rust M, Ong MF, Martens S, Sarrazin C, Bojunga J, Zeuzem S, et al. Performance of transient elastography for the staging of liver fibrosis: a meta-analysis. Gastroenterology 2008; 134: 960-74.
10. Tsochatzis EA, Gurusamy KS, Ntaoula S, Cholongitas E, Davidson BR, Burroughs AK. Elastography for the diagnosis of severity of fibrosis in chronic liver disease: a meta-analysis of diagnostic accuracy. J Hepatol 2011; 54: 650-9.
11. Arena U, Vizzutti F, Corti G, Ambu S, Stasi C, Bresci S, et al. Acute viral hepatitis increases liver stiffness values measured by transient elastography. Hepatology 2008; 47: 380-4.
12. Sagir A, Erhardt A, Schmitt M, Haussinger D. Transient elastography is unreliable for detection of cirrhosis in patients with acute liver damage. Hepatology 2008; 47: 592-5.
13. Millonig G, Reimann FM, Friedrich S, Fonouni H, Mehrabi A, Buchler MW, et al. Extrahepatic cholestasis increases liver stiffness (FibroScan) irrespective of fibrosis. Hepatology 2008; 48: 1718-23.
14. Millonig G, Friedrich S, Adolf S, Fonouni H, Golriz M, Mehrabi A, et al. Liver stiffness is directly influenced by central venous pressure. J Hepatol 2010; 52: 206-10.
15. Mederacke I, Wursthorn K, Kirschner J, Rifai K, Manns MP, Wedemeyer H, et al. Food intake increases liver stiffness in patients with chronic or resolved hepatitis C virus infection. Liver Int 2009; 29: 1500-6.
16. Moneta GL, Taylor DC, Helton WS, Mulholland MW, Strandness DE Jr. Duplex ultrasound measurement of postprandial intestinal blood flow: effect of meal composition. Gastroenterology 1988; 95: 1294-301.
17. Sabba C, Ferraioli G, Genecin P, Colombato L, Buonamico P, Lerner E, et al. Evaluation of postprandial hyperemia in superior mesenteric artery and portal vein in healthy and cirrhotic humans: an operator-blind echo-Doppler study. Hepatology 1991; 13: 714-8.
18. Arena U, Lupsor PM, Stasi C, Moscarella S, Assarat A, Bedogni G, et al. Liver stiffness is influenced by a standardized meal in patients with chronic hepatitis C virus at different stages of fibrotic evolution. Hepatology 2013; 58: 65-72.
19. Gaiani S, Bolondi L, Li BS, Santi V, Zironi G, Barbara L. Effect of meal on portal hemodynamics in healthy humans and in patients with chronic liver disease. Hepatology 1989; 9: 815-9.
20. Dauzat M, Lafortune M, Patriquin H, Pomier-Layrargues G. Meal induced changes in hepatic and splanchnic circulation: a noninvasive Doppler study in normal humans. Eur J Appl Physiol Occup Physiol 1994; 68: 373-80.
21. Ludwig D, Schwarting K, Korbel CM, Bruning A, Schiefer B, Stange EF. The postprandial portal flow is related to the severity of portal hypertension and liver cirrhosis. J Hepatol 1998; 28: 631-8.
22. Bellis L, Berzigotti A, Abraldes JG, Moitinho E, Garcia-Pagan JC, Bosch J, et al. Low doses of isosorbide mononitrate attenuate the postprandial increase in portal pressure in patients with cirrhosis. Hepatology 2003; 37: 378-84.
23. Zardi EM, Dobrina A, Uwechie V, Cacciapaglia F, Rollo M, Laghi V, et al. Postmeal portal flow variations in HCV-related chronic hepatitis and liver cirrhosis with and without hyperdynamic syndrome. In Vivo 2008; 22: 509-12.
24. Berzigotti A, De Gottardi A, Vukotic R, Siramolpiwat S, Abraldes JG, Garcia-Pagan JC, et al. Effect of meal ingestion on liver stiffness in patients with cirrhosis and portal hypertension. PLoS One 2013; 8: e58742.

อิทธิพลของการรับประทานอาหารต่อความยืดหยุ่นของตับในผู้ป่วยไวรัสตับอักเสบบีและซีเรื้อรัง

วริศรา ตั้งประดับเกียรติ, สุรัตน์ ปราณีนรารัตน์, ณัยชญา จำรูญกุล, ทีปวิทย์ วิถีรุ่งโรจน์, ธีระ พิรัชวิสุทธิ์

ภูมิหลัง: การตรวจความยืดหยุ่นของตับ (transient elastography) เป็นวิธีที่ไม่อันตรายและเริ่มเป็นที่นิยมในการประเมินระดับพังผืดในตับในผู้ป่วยไวรัสตับอักเสบบีและซีเรื้อรัง การรับประทานอาหารพบว่ามีผลต่อระดับพังผืดที่วัดได้จากการตรวจความยืดหยุ่นของตับในผู้ป่วยไวรัสตับอักเสบซีเรื้อรัง อย่างไรก็ตามยังไม่มีข้อมูลดังกล่าวนี้ในผู้ป่วยตับอักเสบบีเรื้อรัง

วัตถุประสงค์: เพื่อประเมินอิทธิพลของการรับประทานอาหารต่อระดับพังผืดที่วัดได้จากการตรวจความยืดหยุ่นของตับในผู้ป่วยไวรัสตับอักเสบบีเรื้อรังที่ยังไม่มีภาวะตับแข็ง และเปรียบเทียบผลดังกล่าวกับผู้ป่วยไวรัสตับอักเสบซีเรื้อรัง

วัสดุและวิธีการ: ผู้ป่วยไวรัสตับอักเสบบีเรื้อรัง 45 ราย และไวรัสตับอักเสบซีเรื้อรัง 37 ราย ถูกรวมเข้ามาในการศึกษา การตรวจความยืดหยุ่นของตับจะทำที่ 3 เวลา คือ หลังจากอดอาหาร 4 ชั่วโมง ทันที และ 60 นาทีหลังจากรับประทานอาหารซึ่งเป็นอาหารไทยที่มีพลังงาน 500 กิโลแคลอรี เสร็จ

ผลการศึกษา: ค่าเฉลี่ยของระดับพังผืดหลังจากอดอาหาร 4 ชั่วโมง ในผู้ป่วยไวรัสตับอักเสบบีเรื้อรังคือ 5.40±1.7 กิโลปาสคาล ระดับพังผืดของตับในผู้ป่วยกลุ่มนี้มีระดับที่สูงขึ้นอย่างมีนัยสำคัญทางสถิติทั้งทันที และ 60 นาทีหลังอาหาร คือ เพิ่มขึ้น 0.31±0.1 กิโลปาสคาล ($p = 0.035$) และ 0.33±0.1 กิโลปาสคาล ($p = 0.018$) ตามลำดับ อย่างไรก็ตามการเพิ่มขึ้นของระดับพังผืดของตับไม่พบว่าแตกต่างอย่างมีนัยสำคัญทางสถิติระหว่างผู้ป่วยไวรัสตับอักเสบบีและซีเรื้อรัง คือ 0.72±0.1 และ 1.16±0.1 ตามลำดับ ($p = 0.076$) จากการศึกษานี้ไม่พบปัจจัยอื่นที่มีผลต่อการเปลี่ยนแปลงของระดับพังผืดของตับทั้งในผู้ป่วยไวรัสตับอักเสบบีและซีเรื้อรัง

สรุป: การรับประทานอาหารมีผลทำให้ระดับพังผืดของตับในผู้ป่วยไวรัสตับอักเสบบีเรื้อรังมีการเพิ่มขึ้นอย่างมีนัยสำคัญเช่นเดียวกับผู้ป่วยไวรัสตับอักเสบซีเรื้อรัง และควรพิจารณาเป็นหนึ่งในปัจจัยรบกวนของการตรวจความยืดหยุ่นของตับ ผู้ป่วยควรมีการเตรียมตัวก่อนทำการตรวจความยืดหยุ่นของตับด้วยการงดน้ำและอาหารด้วยเวลาที่เหมาะสม