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How splenic stiffness is affected by changes in the liver stiffness measurements and commonly seen comorbidities

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Abstract

Chronic liver disease is broadly categorized into two stages- the compensated and decompensated liver disease. The transition into decompensated liver disease is marked be a significant increase in morbidity and mortality. Hence, there is a continuous effort to develop criteria's to predict development of decompensation and clinically significant portal hypertension(CSPH). Baveno's criteria is used to predict development of CSPH on the basis on the liver stiffness(LSM) and splenic stiffness measurements (SSM) values. The current study was done to study the correlation between LSM and SSM values in the different categories of fibrosis. With changing LSM values, SSM changes and this correlation is seen to increase with increasing fibrosis. Hence confirming that SSM has a higher importance in patients with moderate to severe fibrosis. While in cases with mild fibrosis with low LSM values, the SSM values are corroborative but to a lesser extent. Also, the impact of different aetiologies on LSM and SSM values was evaluated.

Keywords: Spleen stiffness measurement, Liver stiffness measurement, Clinically significant portal hypertension (CSPH) Hepatic decompensation

Introduction

The most important complication of cirrhosis is the development of portal hypertension. This marks the transition from compensated liver disease to decompensated liver disease [1]. Onset of decompensation heralds a wide set of complications such as ascites, varices and encephalopathy, which leads to a significant rise in morbidity and mortality. [2] The gold standard for the diagnosis of elevated portal venous pressure is either the measurement of hepatic venous pressure gradient (HVPG) through hepatic vein catheterisation or esophagogastroduodenoscopy (EGD) for presence of varices. [3] However, the major limitation with measurement of HVPG is the invasive nature of the investigation, it high cost and it limited access. [4]. While EGD an expensive procedure, it is also operator dependent. [5].

Hence, there is a constant endeavour to find ways non-invasive, reliable, reproducible and low-cost tools for patients of chronic liver disease for diagnostic as well as for prognostic significance. According to the Baveno's VII consensus, a number non-invasive investigations such as liver stiffness measurement (LSM) and splenic stiffness measurement (SSM) may be used for diagnosis of clinically significant portal hypertension(CSPH).[6] SSM has even more importance in the patients who are suffering from viral infections, inflammatory conditions, diseases causing biliary congestion; in which LSM

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is unreliable.[7] Thus while LSM was considered a marker of static assessment of the liver parenchyma; SSM is more dynamic assessment of the status of the HVPG.[8] Practically SSM measurements can prove to be difficult in presence of normal sized spleen ; while in cases with splenomegaly and portal hypertension, this evaluation is easier.

The principle behind the current technique used to measure liver stiffness (LSM) and splenic stiffness measurements is vibration controlled transient elastography [9] LSM has been quite extensively studied, and it forms an important tool for non-invasive diagnosis. It serves as an important prognostic marker for patients with chronic liver disease. [10] SSM evaluation using transient elastography was initially introduced in 2011, which was commercially available for application in 2020. Thereafter, there have been a number studies evaluating SSM. The pathological basis of rising splenic stiffness with portal hypertension was the increased splenic vein pressure, leading to congestion, leading to splenomegaly and eventually increasing splenic stiffness. [11]

Now there is increasing interest in evaluating SSM, whether for its role in the diagnosis of CSPH [12,13] or for a more prognostic evaluation for staging liver cirrhosis and possible response to treatment. [14,15] A large number of studies have been done comparing the efficacy of the LSM and SSM findings in detection of CSPH. The SSM limit is variable in all these cases. Few studies have predicted higher efficacy of SSM over LSM [16,17]. In a prospective cohort study done on 107 patients comparing both the LSM and SSM showed similar findings for diagnosing patients with portal hypertension. [18] Another single centre prospective study conducted over 185 patients detected no statistically significant difference in the LSM and SSM findings over a] Anothertrum of liver diseases. [19] Similarly there are recent studies for the evaluation of clinically significant portal hypertension (CSPH) with the use of spleen stiffness measurements (SSM) [20,21] or the importance of SSM for liver disease staging and treatment response [22,23] In a recently publishes meta-analysis of ten studies evaluating the diagnostic accuracy of spleen stiffness for predicting development of portal hypertension in chronic liver disease. The above said meta- analysis predicted a positive correlation between the LSM and SSM values. [24] In another study in patients with chronic liver disease no significant difference is noted in the prediction using LSM and SSM measurements in 107 patients referred for HVPG measurement for detecting portal hypertension. [25] Thus it is expected for the LSM and SSM to be equally affected in patients with chronic liver disease; unless there is factor affecting the LSM measurement congestion such as hepatic inflammation, fibrosis, or biliary congestion. [26]

Similarly, a number of studies have been conducted for the significance of correlating the SSM and LSM values in cases of hepatic steatosis or steato-fibrosis. With the impending availability of approved treatments for MASLD and metabolic dysfunction-associated steatohepatitis (MASH), it would be interesting to determine the impact of treatment on SSM values as a surro-gate for improvement in portal hypertension. [27]

Materials and Methods

This is a cross-sectional study in which we evaluated 604 patients who had presented to our department for fibroscan.

The data set was evaluated and patients in whom either LSM or SSM was not done, due to varying reasons which could have been obesity, not fasting status or extremely small size of the liver ; data of those patients was excluded.

The procedure was carried out on Fibroscan 630 EXPERT version to measure both LSM and SSM.

The data was divided into three categories according to the LSM measurement according to the Bavenos VII criteria- as less than 10 Kpa, 10-15 Kpa and more than 20 Kpa. Then the splenic EKPA measurements were assessed in these groups. Pearson's correlation was assessed in all these three groups. Descriptive analysis was also done in these groups.

Next the data was assessed based on the CAP into three categories – less than 230, 230-270 and more than 270. Then the splenic EKPA values were correlated in these groups. Correlation coefficients were calculated, and linear relationship curves were drawn.

Discussion

LSM measurements have been used to assess the status of fibrosis in liver and to predict the development of clinically significant portal hypertension (CSPH). However, the LSM measurements could be indeterminate if patient is very obese, has ascites, or in medical situations such as acute hepatitis, transaminitis, in the presence of cholestasis (due to any cause) and hepatic congestion due to underlying heart disease.

Table 1: Table illustrating the variation of splenic EKPA, age statistics and Liver EKPA values across the three groups categorized according to liver EKPA

	SPLEEN EKPA						AGE		LIVER EKPA	
	count	mean	median	std	min	max	mean	std	mean	std
LIVER_EKPA_GROUP										
<10 kPa	388	11.18	10.1	16.7	0.0	92.6	46.6	14.27	6.1	1.71
10-15 kPa	81	15.11	6.5	18.78	0.0	82.6	50.75	15.11	12.4	1.42
>15 kPa	135	31.47	29.0	30.23	0.0	100.0	54.61	10.83	37.53	19.12

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In such cases SSM can be used to correlate the findings and serve complementary to the LSM or be an independent factor in classification of severity of fibrosis and predictor of development of CSPH. Although the SSM measurements are a little difficult to evaluate as compared to the LSM in cases where the spleen is small or the patient is obese. However, these difficulties can be overcome with localizing the spleen first with a convex ultrasound probe (3-5 Mhz). Also, in all the medical conditions where LSM is unreliable such as hepatitis, cholestatis and biliary congestion, SSM could prove reliable.

We had a total of 604 patients with valid fibroscan data done over a period of 8 months from august, 2024 to March 2025. The total data of these patients was evaluated, and they were divided into 3 groups (according the the Baveno's VII criteria) with EKPA values less than 10 Kpa, 10-15 Kpa and more than 20 Kpa- correlating with mild, moderate and severe fibrosis category (Table 1).

1. Group 1 (<10 kPa): 388 patients (64.2%)
   • Mean SPLEEN EKPA: 11.18 kPa.
2. Group 2 (10-15 kPa): 81 patients (13.4%)
   • Mean SPLEEN EKPA: 15.11 kPa
3. Group 3 (>15 kPa): 135 patients (22.4%)
   • Mean SPLEEN EKPA: 31.47 kPa

Pearson's correlation coefficient was calculated for all these 3 groups between the liver and splenic EKPA. (Figure 1) The Pearson's coefficient for these three groups was:

1. Group 1 (<10 kPa): r = 0.156
2. Group 2 (10-15 kPa): r = 0.053
3. Group 3 (>15 kPa): r = 0.450

Table 2: Percentage of co- morbidities present in the three groups

Variable	<10 kPa	10-15 kPa	>15 kPa
Sample Size (n)	388	81	135
Age (years)	46.6 ± 14.3	50.8 ± 15.1	54.6 ± 10.8
BMI (kg/m²)	28.8 ± 18.6	29.7 ± 4.9	27.8 ± 4.4
Alcohol/Drug Use (%)	19.1%	23.5%	27.4%
Diabetes Mellitus (%)	18.0%	40.7%	42.2%
HbA1c Abnormal (%)	14.2%	22.2%	18.5%
Hypertension (%)	22.4%	28.4%	25.9%
Thyroid Disease (%)	2.6%	1.2%	4.4%

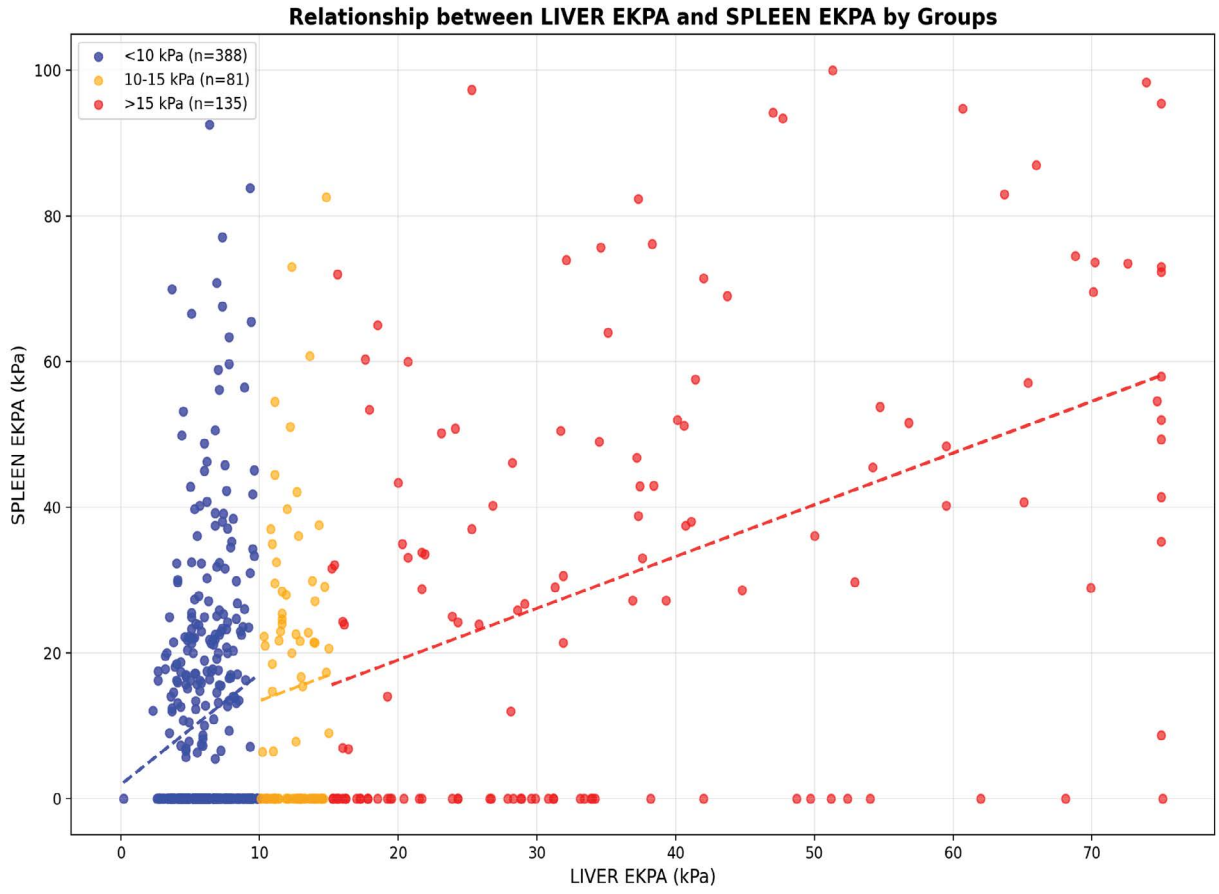

Figure 1: Linear graph representation of the Pearson's correlation between spleen EKPA and liver EKPA in the three groups. Less than 10 EKPA- in blue colour; 10-15 EKPA- In yellow colour and > 15 EKPA in red colour.

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So analysing these values suggests there is a strong positive correlation between the splenic and liver stiffness measurements; with the highest correlation seen in the group with EKPA values more than 15 Kpa.

Furthermore, application of the ANOVA test of the significance of spleen EKPA values suggests a p value of less than 0.001 which is highly significant. As with LSM, SSM is seen to show progressive increase in the values showing a positive correlation. This corroborates with the underlying pathological process of fibrosis. As the LSM values increase in the severe fibrosis category with more than 15 EKPA, the SSM values also rise. These findings reflect rising portal venous pressure, leading to vascular congestion and splenomegaly with worsening hepatic fibrosis. The mean SSM in this group was 31.47Kpa. Hence in cases with severe fibrosis, SSM can be equally reliable as LSM.

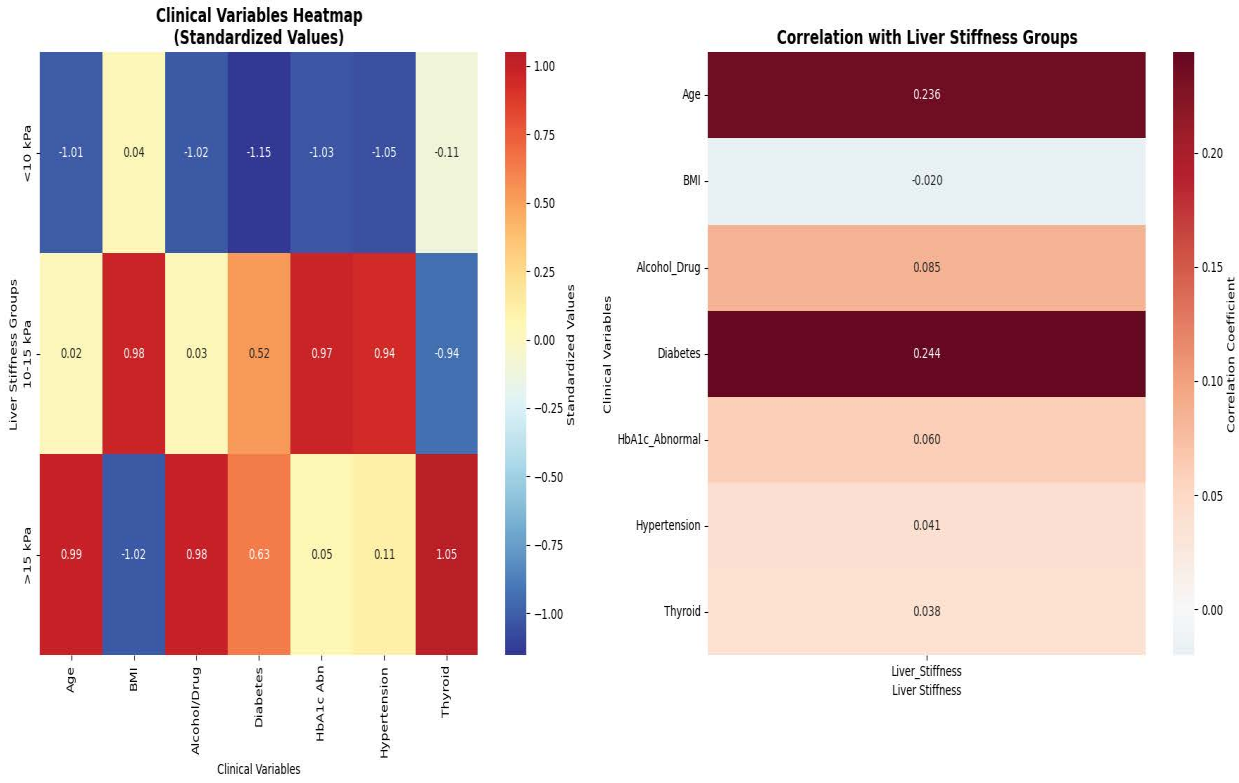

Figure 2: Heat map distribution of the different co- morbidities according to LSM values.

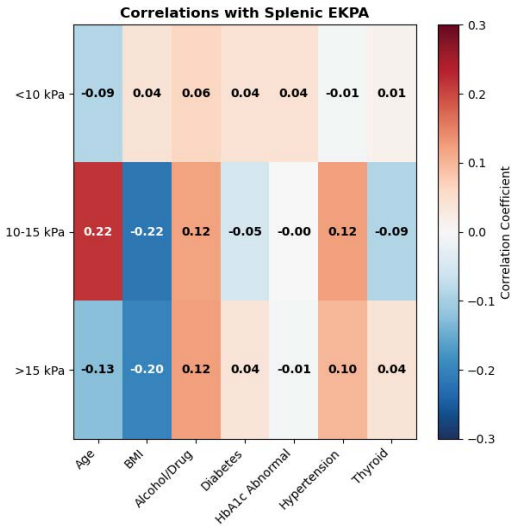

Figure 3: Heat map reflecting different co – morbidities according to SSM (Splenic EKPA) values.

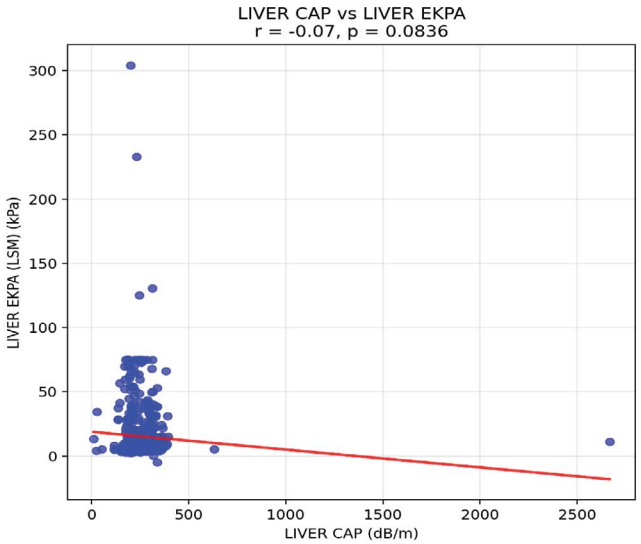

Figure 4: Linear correlation between Liver EKPA and CAP values showing a mild negative correlation.

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Table 3: Classification of the data into groups according to CAP values and accordingly LSM and SSM values in these groups.

CAP_Group	N_patients	CAP_mean	CAP_std	LSM_mean	LSM_std	LSM_median	SSM_mean	SSM_std	SSM_median
Less than 238	178	196.6	36.2	20.3	33.4	7.1	21.1	24.4	15.1
238-260	88	248.6	6.5	15.4	21.1	7.3	15.3	21.8	0.0
260-290	119	276.5	9.3	11.4	12.7	7.0	13.9	21.1	0.0
More than 290	221	336.0	160.5	12.9	14.0	8.3	13.7	20.1	0.0

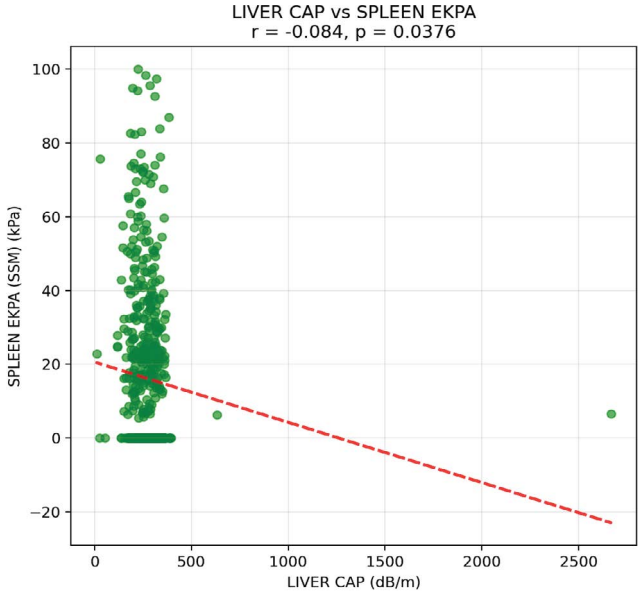

Figure 5: Negative linear correlation between spleen EKPA levels and CAP values.

The co- morbidities of patients in these three groups were evaluated for any possible impact they could have on the SSM and LSM measurement. The HbA1c measurement of 6.5 was used as a cutoff for presence or absence of disease. Amongst the thyroid diseases, presence of hypothyroidism was assumed to be significant.The basal metabolic rate (BMI), history of alcohol/ drug use, presence of diabetes, hypertension, hypothyroidism and elevated HbA1C levels were assessed. (Table 2).

The liver stiffness measurement showed a strong positive correlation with higher age groups, hypothyroidism, and alcohol intake. There was a moderate positive correlation of LSM with hypertension, high HbA1c levels and BMI. However, on correlation these comorbidities with SSM; there was no significant correlation with any of these factors. This further strengthens the fact that the SSM only varies positively with LSM; with no significant impact of the comorbidities. Thus, SSM can serve as a more reliable marker for both diagnosis and prognosis in patients with co- morbidities. (Figure 2 and 3).

The data separated into the three groups according to the CAP levels is evaluated for corresponding LSM and SSM measurements (Table 3). The mean LSM levels and SSM levels is seen to decrease successively in the three categories: suggesting a negative correlation. This negative correlation is maximum in the categories with highest CAP values (>290) (Figure 4,5,6). This implies that fatty liver does not correlate with hepatic fibrosis.

Conclusion

The present study reliably concludes that in cases of severe fibrosis, SSM can be as reliable as LSM for follow-up studies, and also for detecting response to treatment. Although SSM correlation with LSM is maximum in cases of severe fibrosis, there is a positive correlation in cases of moderate and mild fibrosis. This confirms its importance as an auxiliary marker for early fibrosis and categorisation of fibrosis into mild, moderate and severe categories.

In cases with co-morbidities such as alcohol intake, hypertension, elevated HbA1C levels, hypothyroidism and higher age groups, which tend to influence the LSM findings, it was noted that the SSM values were not affected to a significant degree. Hence, SSM would be reliable in such cases.

The drawback of the current study is that laboratory parameters, such as low platelet count, transaminase levels, or even clinical criteria, such as MELD score, could have also been correlated. Also, for the cormorbidities, each factor requires an exclusive study with a larger number of positive cases, respectively, to prove reliably that in such cases SSM is superior to LSM.

References

1. Engelmann C, Claria J, Szabo G, Bosch J, Bernardi M. Pathophysiology of decompensated cirrhosis: portal hypertension, circulatory dysfunction, inflammation, metabolism and mitochondrial dysfunction. J Hepatol 75 (2021): S49–S66.

2. Axley P, Ahmed Z, Arora S, Haas A, Kuo YF, et al. NASH is the most rapidly growing etiology for acute-on-chronic liver failure-related hospitalization and disease burden in the United States: a population-based study. Liver Transpl 25 (2019): 695–705.

3. Tripathi D, Stanley AJ, Hayes PC, Patch D, Millson C, et al. UK guidelines on the management of variceal haemorrhage in cirrhotic patients. Gut 64 (2015): 1680–1704.

4. Targownik LE, Spiegel BM, Dulai GS, Karsan HA,

Gralnek IM. The cost-effectiveness of hepatic venous pressure gradient monitoring in the prevention of recurrent variceal hemorrhage. Am J Gastroenterol 99 (2004): 1306–1315.

5. Hu X, Huang X, Hou J, Ding L, Su C, et al. Diagnostic accuracy of spleen stiffness to evaluate portal hypertension and esophageal varices in chronic liver disease: a systematic review and meta-analysis. Eur Radiol 31 (2021): 2392–2404.
6. De Franchis R, Abraldes JG, et al. Baveno VII – Renewing consensus in portal hypertension. J Hepatol 76 (2022): 959–974.
7. Reiberger T. The value of liver and spleen stiffness for evaluation of portal hypertension in compensated cirrhosis. Hepatol Commun 6 (2022): 950–964.
8. Nedredal GI, Yin M, McKenzie T, Joseph L, Jennifer LW, et al. Portal hypertension correlates with splenic stiffness as measured with MR elastography. J Magn Reson Imaging 34 (2011): 79–87.
9. Vergniol J, Foucher J, Terrebonne E, Bernard PH, Le Bail B, et al. Noninvasive tests for fibrosis and liver stiffness predict 5-year outcomes of patients with chronic hepatitis C. Gastroenterology 140 (2011): 1970–1979.
10. De Franchis R, Abraldes JG, et al. Baveno VII – Renewing consensus in portal hypertension. J Hepatol 76 (2022): 959–974.
11. Brol MJ, Godiker J, Uschner FE, Micheal P, Jonel T. Non-invasive assessment of clinically significant portal hypertension. Curr Hepatol Rep 22 (2023): 206–215.
12. Colecchia A, Ravaioli F, Marasco G, Colli A, Dajti E, et al. A combined model based on spleen stiffness measurement and Baveno VI criteria to rule out high-risk varices in advanced chronic liver disease. J Hepatol 69 (2018): 308–317.
13. Dajti E, Ravaioli F, Colecchia A, Marasco G, Reggiani MLB, et al. Spleen stiffness measurements predict the risk of hepatic decompensation after direct-acting antivirals in HCV cirrhotic patients. Ultraschall Med 43 (2022): 280–288.
14. Marasco G, Dajti E, Ravaioli F, Alemanni LV, Capuano F, et al. Spleen stiffness measurement for assessing the response to β-blockers therapy for high-risk esophageal varices patients. Hepatol Int 14 (2020): 850–857.
15. Wong GLH, Kwok R, Hui AJ, Tse YK, Ho KT, et al. A new screening strategy for varices by liver and spleen stiffness measurement (LSSM) in cirrhotic patients: a randomized trial. Liver Int 38 (2018): 636–644.
16. Colecchia A, Montrone L, Scaioli E, Reggiani MLB, Colli A, et al. Measurement of spleen stiffness to evaluate portal hypertension and the presence of esophageal varices in patients with HCV-related cirrhosis. Gastroenterology 143 (2012): 646–654.
17. Stefanescu H, Grigorescu M, Lupsor M, Procopet B, Maniu A, Badea R. Spleen stiffness measurement using Fibroscan for the noninvasive assessment of esophageal varices in liver cirrhosis patients. J Gastroenterol Hepatol 26 (2011): 164–170.
18. Zykus R, Jonaitis L, Petrenkiene V, Pranculis A, Kupcinskas L. Liver and spleen transient elastography predicts portal hypertension in patients with chronic liver disease: a prospective cohort study. BMC Gastroenterol 15 (2015): 183.
19. Lantinga MA, Kleef LA, Hoed CM, Knegt RJ. Spleen stiffness measurement across the spectrum of liver disease patients in real-world practice. J Clin Exp Hepatol 13 (2023): 414–427.
20. Colecchia A, Ravaioli F, Marasco G, Colli A, Dajti E, et al. A combined model based on spleen stiffness measurement and Baveno VI criteria to rule out high-risk varices in advanced chronic liver disease. J Hepatol 69 (2018): 308–317.
21. Dajti E, Ravaioli F, Colecchia A, Marasco G, Reggiani MLB, et al. Spleen stiffness measurements predict the risk of hepatic decompensation after direct-acting antivirals in HCV cirrhotic patients. Ultraschall Med 43 (2022): 280–288.
22. Marasco G, Dajti E, Ravaioli F, Alemanni LV, Capuano F, et al. Spleen stiffness measurement for assessing the response to β-blockers therapy for high-risk esophageal varices patients. Hepatol Int 14 (2020): 850–857.
23. Wong GLH, Kwok R, Hui AJ, Tse YK, Ho KT, et al. A new screening strategy for varices by liver and spleen stiffness measurement (LSSM) in cirrhotic patients: a randomized trial. Liver Int 38 (2018): 636–644.
24. Hu X, Huang X, Hou J, Ding L, Su C, Meng F. Diagnostic accuracy of spleen stiffness to evaluate portal hypertension and esophageal varices in chronic liver disease: a systematic review and meta-analysis. Eur Radiol 31 (2021): 2392–2404.
25. Zykus R, Jonaitis L, Petrenkiene V, Pranculis A, Kupcinskas L. Liver and spleen transient elastography predicts portal hypertension in patients with chronic liver disease: a prospective cohort study. BMC Gastroenterol 15 (2015): 183.
26. Reiberger T. The value of liver and spleen stiffness for evaluation of portal hypertension in compensated cirrhosis. Hepatol Commun 6 (2022): 950–964.
27. Behari J. Spleen stiffness measurement in metabolic dysfunction-associated steatotic liver disease—value added or work in progress? Dig Dis Sci 69 (2024): 1093–1095.