Assessment of splenic elasticity in patients with advanced chronic liver disease: a retrospective monocentric study

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Introduction

Spleen stiffness measurement (SSM) is a non-invasive screening tool proposed to assess significant portal hypertension. SSM cut-offs have been proposed by Baveno VII for patients with compensated advanced chronic liver disease (cACLD): group 1 (rule-out zone) with a SSM < 21 kPa, group 2 (grey-zone) with a SSM between 21 and 50 kPa and group 3 (rule-in zone) with a SSM > 50 kPa.

Aim

Given the relative novelty of this technique, our aim is to assess the number of patients in each of these groups and their clinical characteristics, particularly regarding the complications of cirrhosis, and to correlate these findings to the hepatic venous pressure gradient (HVPG) measurement.

Patients and methods

Adult patients with available SSM were retrospectively included at our single centre between August 2023 and June 2025. Patients without cACLD (liver stiffness measurement (LSM) < 10 kPa), with cardiac hepatopathy or history of liver transplantation were excluded. SSM and LSM were measured with the 110 and 50 Hz modules respectively on the same day by vibration-controlled transient elastography using the FibroScan 630 Expert®.

Baseline clinical and biological data of patients were collected at the time of the SSM. Baveno VII cut-offs were used to divide patients into three groups based on the values of the SSM.

Results

161 patients were included, with a median age of 60 years (interquartile, IQR 48 - 67), mainly men for 66% and with a median body mass index of 27.2 kg/m² (IQR 24.5 - 31.2). The causes of cACLD and cirrhosis were as follows: alcohol-related liver disease (39%), metabolic dysfunction-associated steatotic liver disease (32.5%), hepatitis C virus (10.0%), auto-immune hepatitis (3.7%), primary biliary cholangitis (3.1%), hepatitis B virus (3.1%) and others (8.6%). SSM was successfully measured, with an average of 12 valid measurements and an average IQR range of 11.7 kPa.

Based on SSM cut-offs proposed by Baveno VII, only 3 patients were classified in group 1 (mean SSM 12.9 kPa, IQR 9.1-14.3), 75 in group 2 (mean SSM 37.3 kPa, IQR 32-42.4) and 83 in group 3 (mean SSM 71.9 kPa, IQR 55.9-83.5).

Liver elasticity significantly differed between group 3 (median 29.5kPa) and groups 1 and 2 (14.7 and 21.7 kPa respectively, $p < 0.001$). LSM and SSM values were correlated ($r = 0.43$, $p < 0.001$).

Groups 2 and 3 were also different in terms of platelet count (151 vs 115 $*10^3$/mL, $p < 0.001$), total bilirubin levels (0.7 vs 0.9 mg/dL, $p = 0.03$), albumin levels (44 vs 41 g/L, $p = 0.004$), white blood cell count (5.3 vs 6.9 $*10^3$/µL, $p = 0.003$), C-reactive protein (1.6 vs 3.0 mg/L, $p = 0.005$) and Model for End Stage Liver Disease (MELD) score (9 vs 10, $p = 0.02$). Those two groups also differed in terms of HVPG measurement (9.5 -n = 22- vs 12.0 mmHg -n = 29-, $p = 0.01$), spleen size (11.3 vs 13.0 cm, $p < 0.001$),

history of ascites (15 vs 30%, p = 0.039), presence of esophageal varices (32 vs 53%, p = 0.006) but not in terms of history of variceal bleeding (4 vs 11%, p = 0.32). No patients in group 2 were taking beta-blockers, whereas 15 patients (18 %) in group 3 were (p < 0.001).

In the subgroup of patients with HVPG measurement (n = 51), there was a good correlation between LSM and HVPG values (r = 0.49, p < 0.001) but not between SSM and HVPG values (r = 0.26, p = 0.064).

Conclusion

Less than 2% of patients with cACLD or cirrhosis fall within the range that allows portal hypertension to be ruled out (group 1) based on the cut-offs proposed by Baveno VII. Patients in the grey zone (group 2) and the rule-in zone (group 3) differ according to biological markers of liver function. Patients in the rule-in zone also have a higher prevalence of ascites but not of variceal bleeding. Surprisingly, HVPG correlates more strongly with hepatic elasticity than splenic elasticity. The Baveno VII algorithm and cut-offs can therefore be considered as indicators but should be re-evaluated before their clinical utility can be confirmed in patients with cACLD and cirrhosis. The longitudinal evolution of splenic elasticity and long-term follow-up will also be important to assess.