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Sequential Shear-wave elastography of liver and spleen rules out clinically significant portal hypertension in compensated advanced chronic liver disease.

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Keywords: shear-wave elastography, clinical significant portal hypertension, spleen stiffness, liver stiffness

Abbreviations:

OV: oesophageal varices, HVPG: hepatic venous pressure gradient, CSPH: clinically significant portal hypertension, TE: transient elastography, SWE: Shear-wave elastography, L-SWE: liver SWE, S-SWE: spleen SWE, MELD: model for end-stage liver disease

Word count: 589

References: 4

Table: 2

With interest we read the letter entitled “Ruling in and ruling out with elastography in compensated advanced chronic liver disease” by Agustin et al.¹ to our study “Algorithm to rule-out clinically significant portal hypertension combining Shear-wave elastography of liver and spleen: a prospective multi-centre study”². Indeed, we would like to thank the colleagues for their interest in our study and deciphering the strengths and weaknesses of this study. Besides acknowledging the merits of the study, the colleagues comment on the selection of patients regarding severity and aetiology of advanced chronic liver disease (ACLD), as well as the clinical importance to rule-out or rule-in of clinical significant portal hypertension (CSPH).

The criticism about the selection of patients stimulated us to perform new calculations. Even though the algorithm was calculated including decompensated ACLD (dACLD) patients, it performed much better in patients with compensated ACLD (Table 1). We agree that the selection of patients is crucial for the determination of cut-off points. However, our algorithm improved sensitivity and specificity of SWE compared to preceding studies analysing patients with CSPH without dACLD. Therefore, including dACLD could be also a benefit to identify a specific cut-off, which is valid in a wider range of portal hypertension. This collective allowed us to build up an algorithm using spleen stiffness in order to rule out CSPH, especially effective in Child A patients with a sensitivity of 97.4% and a specificity of 81.5% and negative predictive value of 95.7% (Table 1).

We clearly agree with the comment that aetiology of cACLD influences liver stiffness, especially to discriminate fibrosis stages. However, CSPH might be present across different etiologies, and liver stiffness alone is insufficient to rule out CSPH, even with different cut-offs. Indeed, also in Baveno VI consensus conference liver stiffness was combined with platelet count to rule out CSPH and decide the timing of screening endoscopy³. To address this point, we recalculated our data and observed that despite low numbers, CSPH due to alcoholic and non-alcoholic steatohepatitis (NASH) could be ruled out with a negative predictive of higher than 90% and diagnostic accuracy of more than 80% (Table 2). CSPH due to viral hepatitis seems not to be reflected adequately by this algorithm, which might be a limitation to be addressed in the future studies. Importantly, taking into account the changes in the management of successfully treated HCV-patients and increasing prevalence for

1 NASH and alcoholic liver disease, this algorithm might be useful for the majority of
2 cACLD patients at the moment and especially in the future.

3 The management of patients with cACLD includes many different aspects, the
4 surveillance for hepatocellular carcinoma and varices, but also prevention of
5 decompensation, especially with regard to treatment options of comorbidities (any
6 surgery, any medication, etc.). CSPH is the driving force for these events and
7 therefore ruling out CSPH in these patients might render the general management of
8 these patients easier and save economic and logistic resources. Our algorithm ²
9 might be helpful to improve the management and to avoid over-diagnostics. In the
10 case when CSPH cannot be ruled out, further diagnostics are required.

11 While liver stiffness might be influenced also by other factors, especially aetiology,
12 spleen stiffness remains usually not influenced by these factors, but only by portal
13 pressure. The usefulness of spleen SWE alone in the prediction of CSPH could not
14 be demonstrated in previous publications and will be focus of future research.

15 In summary, the sequential use of liver and spleen SWE rules out efficiently CSPH
16 especially in cACLD due to alcoholic and NASH. These observations need
17 independent confirmation and further studies but are a step in the right direction.

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References

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Competing interests: none declared

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Table 1. Diagnostic performance of the algorithm for the diagnosis of CSPH.

	Sensitivity [%]	Specificity [%]	Positive predictive value [%]	Negative predictive value [%]	Diagnostic accuracy [%]	Correctly calssified [%]
All patients (n=109)	98.6	70.3	86.6	96.3	89.0	84.5
Child A (n=66)	97.4	81.5	88.4	95.7	90.9	91
Child B/C (n=43)	100	40	84.6	100	86	83

CSPH, clinically significant portal hypertension.

Table 2. Diagnostic performance of the algorithm for the diagnosis of CSPH.

	Sensitivity [%]	Specificity [%]	Positive predictive value [%]	Negative predictive value [%]	Diagnostic accuracy [%]	Correctly classified [%]
Alcoholic cirrhosis (n= 61)	100	40	89.5	100	90	90
NASH-cirrhosis (n=19)	75	86.7	60	92.9	84	84
Viral and other (n=29)	92	82	86	90	75	75

NASH, non-alcoholic steatohepatitis.