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Non-invasive assessment of esophageal varices: Status of today

Tarana Gupta

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Abstract

With increasing burden of compensated cirrhosis, we desperately need non-invasive methods for assessment of clinically significant portal hypertension. The use of liver and spleen stiffness measurement helps in deferring unnecessary endoscopies for low risk esophageal varices. This would reduce cost and patient discomfort. However, these special techniques may not be feasible at remote areas where still we need only biochemical parameters. More prospective studies validating the non-invasive risk prediction models are definitely needed.

Key Words: Compensated cirrhosis; Spleen stiffness measurement; Liver stiffness measurement; High-risk esophageal varices; Clinically significant portal hypertension

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Core Tip: The liver stiffness measurement is time tested tool for assessing liver fibrosis. The new application of spleen stiffness has again supplemented for assessment of portal hypertension and has alleviated the need for unnecessary endoscopies. The novel spleen dedicated stiffness measurement @100 Hz has further improved the screening of high-risk esophageal varices.

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INTRODUCTION

We read with great interest “Non-invasive model for predicting high-risk esophageal varices (HEVs) based on liver and spleen stiffness” by Yang *et al* [1]. In view of risk,

discomfort and cost associated with invasive procedures like liver biopsy, endoscopy *etc.* it has become essential to validate the non-invasive model for predicting clinically significant portal hypertension (CSPH). Yang *et al*[1] created a model using spleen and liver stiffness measurement (SSM and LSM). The initial models LSPS (LS-spleen diameter to platelet ratio score), Baveno VI, improved Baveno VII have already given better understanding of worsening portal hypertension to CSPH[2]. Baveno VII criteria proposed additional use of SSM cut-off 40 kPa in patients where Baveno VI criteria were not met to rule out HEVs (high risk esophageal varices). This prevented more unnecessary endoscopies in clinical practice. The proposed RESIST-HCV criteria in the SIMPLE study included platelet count and serum albumin for predicting the development of HEVs in future in patients with HCV-related compensated cirrhosis with LEV[3].

The increasing burden of alcohol and metabolic dysfunction-associated steatotic liver disease (MASLD), viral cirrhosis with their long follow-up warrants the use of non-invasive strategies to risk stratify these patients. Follow-up of the compensated advanced chronic liver disease requires close monitoring to limit future complications as well as health care burden. The LSM is affected by hepatic inflammation, cholestasis, congestion due to right heart failure, infiltrative diseases *etc.* and may be spuriously high (false positive). In these settings, it may not be truly reflective of portal hemodynamics *i.e.* portal hypertension. On the other hand, spleen stiffness is affected by splenomegaly, increased blood pooling in spleen, associated splenic fibrosis and all these factors are reflective of portal hypertension[4]. However, patients with small spleen having < 4 cm thickness may not be suitable candidates for elastography measurement. Studies have shown magnetic resonance (MR) based spleen stiffness of 7.23 kPa indicative of presence of esophageal varices[5]. The assessment of SSM has been done by both 50 Hz and 100 Hz probes. One recent study demonstrated endoscopy spare rate by SSM at 100 Hz to be significantly better than SSM at 50 Hz (38.9% *vs* 26.5%; $P < 0.001$) respectively[6].

Yang *et al*[1] derived model based upon LSM and SSM; however, it is essential to note the shortcomings of this particular model. First, it requires specialized equipment to measure elastography and may not be feasible in remote areas. Second, as already mentioned there is a subset of patient population with right heart failure, chronic kidney disease with fluid overload, infiltrative and cholestatic liver diseases where elastography is not accurate and may fallaciously give high values. Though here we are focusing on cirrhosis related portal hypertension, but in clinical practice, this may not be reproducible in non-cirrhotic portal hypertension. Third, we need to address the issues related to heterogeneity in etiology of liver disease such as MASLD where the corresponding LSM values for F3-F4 fibrosis are higher than in viral and alcohol-related cirrhosis. The authors have taken predominant virus related cirrhosis and patients of alcohol and MASLD have been excluded. Fourth, they have used Baveno VI for comparing their model whereas Baveno VII has already included SSM in assessing portal hypertension. Finally, they have not mentioned as to whether endoscopist doing the evaluation of EVs as HEV/LEV was aware of LSM and SSM values. As this may lead to additional bias in reporting.

CONCLUSION

Therefore, the current model may not be generalizable to all etiologies. But nonetheless, these models are the need of hour for addressing long-term complications in these patients. In future, we shall need more studies including adequate number of patients from every etiology to validate the current model.

FOOTNOTES

Author contributions: Gupta T did collection of data, review of literature, wrote the manuscript, critically analysed and did final drafting.

Conflict-of-interest statement: Tarana Gupta have no conflict of interest.

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