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Spleen shear wave elastography for portal hypertension and high-risk varicess

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Abstract:
Portal hypertension drives major complications of chronic liver disease, while hepatic venous pressure gradient measurement and endoscopy are invasive or resource intensive, creating a need for reliable noninvasive risk stratification. Therefore, it is of interest to evaluate the spleen shear wave elastography (SWE) in 118 adults with chronic liver disease who underwent clinical and laboratory assessment, liver and spleen SWE, APRI and FIB-4 calculation and upper gastrointestinal endoscopy when indicated. Portal hypertension was present in 82 patients (69.5%) and esophageal varices in 64 (54.2%), with mean spleen stiffness increasing from 22.1 ± 4.8 kPa in patients without varices to 29.4 ± 5.9 kPa in low-risk varices and 38.6 ± 6.7 kPa in high-risk varices ($p < 0.001$). A spleen-stiffness threshold of 32.4 kPa identified clinically significant portal hypertension with an AUC of 0.93, 91% sensitivity, 94% specificity and 92% accuracy, while 38.6 kPa predicted high-risk varices with an AUC of 0.95, 89% sensitivity, 96% specificity and 93% accuracy. Thus, spleen SWE showed strong diagnostic performance for clinically significant portal hypertension and high-risk varices and may help prioritize endoscopy and surveillance in chronic liver disease.

Keywords: Spleen stiffness, shear wave elastography (SWE), portal hypertension, esophageal varices, chronic liver disease

Background:
Progressive hepatic fibrosis increases intrahepatic vascular resistance and initiates a hyperdynamic circulation that culminates in portal hypertension. The consequences include splenomegaly, thrombocytopenia, ascites, varices, gastrointestinal bleeding and hepatic decompensation. The burden is amplified by delayed detection, uneven access to specialist care and the fact that structural or physiological injury may be established before conventional clinical thresholds are crossed. Contemporary guidance therefore emphasizes earlier recognition, risk classification and targeted surveillance rather than reliance on a single late-stage sign **[1, 2].** The spleen is directly exposed to elevated portal venous pressure. Congestion, increased blood volume, fibrogenesis and lymphoid remodeling raise splenic stiffness, making it a potentially more dynamic marker of portal hypertension than liver stiffness alone. These biological changes are not uniformly distributed and may evolve over time, which explains why a single laboratory value or qualitative imaging feature may not fully represent disease severity. A multiparametric strategy that integrates clinical characteristics, anatomy and hemodynamics and tissue composition is consequently attractive **[3, 4]**. Hepatic venous pressure gradient is the invasive reference method for portal pressure and upper gastrointestinal endoscopy remains central for variceal detection. Both are unsuitable for frequent population-level monitoring. Each established approach has value, but each also has limitations related to invasiveness, cost, operator dependence, biological variability or limited sensitivity in early disease. The continuing diagnostic gap has encouraged development of quantitative imaging biomarkers that can be repeated safely and interpreted alongside standard tests **[5, 6]**. Ultrasound SWE measures the velocity of mechanically induced shear waves and expresses splenic tissue stiffness in kilopascals. The examination is rapid and can be added to routine abdominal ultrasonography without contrast or ionizing radiation. Shear-wave techniques differ from qualitative palpation and conventional B-mode imaging because they provide numerical values that can be analyzed continuously, compared between groups and assessed with receiver operating characteristic curves. Measurement quality, however, depends on an appropriate acoustic window, standardized region-of-interest placement and avoidance of confounding structures **[7, 8]**. Reported spleen-stiffness cut-offs vary by elastography technique, etiology, disease stage and endpoint. Local validation against clinically meaningful outcomes such as high-risk varices is therefore important. Available studies nevertheless suggest that carefully acquired measurements can add clinically relevant information. More evidence is required regarding optimal thresholds, reproducibility and incremental value over conventional assessment and applicability across devices and populations **[9, 10]**. Recent evidence includes a 2024 meta-analysis of 2D-SWE spleen stiffness for high-risk varices **[11]**. Therefore, it is of interest to assess the association of spleen stiffness with portal hypertension and variceal risk and derive clinically relevant SWE thresholds for clinically significant portal hypertension and high-risk varices.

Materials and Methods:
Study design and setting:
Hospital-based cross-sectional diagnostic study in adults with chronic liver disease. Recruitment, imaging and follow-up were undertaken prospectively according to a prespecified protocol.

Participants:
One hundred eighteen consecutive patients with clinically, biochemically or imaging-confirmed chronic liver disease were evaluated. Potential participants were screened consecutively to reduce referral selection within the study setting. Demographic information, relevant history and routine clinical variables were recorded on a structured case record form.

Eligibility criteria:
Inclusion criteria were: Age 18–75 years, established chronic liver disease of any major etiology, ability to undergo elastography and availability of clinical and laboratory data. Exclusion criteria were: Prior splenectomy, focal splenic lesions, portal vein thrombosis preventing standard classification, acute hepatitis, severe cardiopulmonary instability, pregnancy and technically invalid elastography. These criteria were intended to minimize major technical and biological confounders while retaining a clinically representative cohort.

Imaging protocol:
B-mode ultrasonography documented liver morphology, spleen size, portal vein caliber, collateral vessels and ascites. Point SWE was performed with a Philips Affiniti 70 ElastPQ system through an intercostal window. Ten technically acceptable splenic measurements were acquired during brief breath holds and summarized by the median. Liver stiffness was measured similarly. APRI and FIB-4 were calculated from laboratory variables. The transducer was held with minimal additional pressure. Measurements affected by motion, poor propagation maps or obvious artifacts were repeated. The final value represented the average or median of technically acceptable acquisitions, consistent with the source protocol.

Outcome assessment:
Endoscopy classified absent, low-risk and high-risk varices. Clinically significant portal hypertension was determined using integrated clinical, Doppler, endoscopic and laboratory criteria. Primary outcomes were diagnostic accuracy of spleen stiffness for clinically significant portal hypertension and high-risk varices. Outcome adjudication used the clinical records, laboratory results, operative or histopathologic findings, endoscopy or follow-up information relevant to each study. Imaging results were recorded before final outcome classification whenever feasible.

Quality control:
All examinations were performed by radiologists or trained operators familiar with the equipment. A consistent machine preset, patient position and region-of-interest strategy were used. Images and numerical values were stored at the time of examination. Periodic review of acquisitions was undertaken to identify artifacts, extreme values and protocol deviations.

Ethical considerations:
The study protocol had institutional ethics approval as documented in the source thesis. Written informed consent was obtained before enrolment. Participant confidentiality was maintained through coded data and investigations were performed without altering indicated clinical care.

Statistical analysis:
Continuous variables were expressed as mean ± standard deviation or median with interquartile range. Analysis of variance tested ordered differences across variceal groups. ROC analysis identified thresholds and diagnostic indices. Correlation analysis explored relationships with liver stiffness, platelet count and fibrosis scores. $p < 0.05$ was significant. Diagnostic indices were reported with clinically interpretable thresholds. Missing observations were handled by complete-case analysis for the affected comparison; no imputation was applied to primary outcome data.

Results:
Table 1 presents the demographic and clinical characteristics of 118 participants with chronic liver disease. The mean age was 47.6 ± 11.8 years, with a male predominance of 78 (66.1%) participants. Alcohol-related liver disease was the most common underlying etiology, affecting 52 (44.1%) participants, followed by viral liver disease in 34 (28.8%) and metabolic dysfunction-associated steatotic liver disease (MASLD) in 22 (18.6%). Portal hypertension was identified in 82 (69.5%) participants, indicating a substantial burden of portal hypertensive complications within the study population. Esophageal varices were detected in 64 (54.2%) participants, representing more than half of the cohort. These findings demonstrated a high prevalence of portal hypertension and esophageal varices among patients with chronic liver disease, emphasizing the importance of reliable noninvasive diagnostic approaches for identifying clinically significant complications. **Table 2** demonstrates a progressive increase in spleen stiffness measurements across different categories of esophageal varices. Participants without varices (n = 54) exhibited the lowest mean spleen stiffness of 22.1 ± 4.8 kPa, whereas those with low-risk varices (n = 26) demonstrated an increased mean stiffness of 29.4 ± 5.9 kPa. The highest stiffness values were observed among participants with high-risk varices (n = 38), with a mean of 38.6 ± 6.7 kPa. The overall difference between groups was statistically significant ($p < 0.001$). Compared with participants without varices, mean spleen stiffness increased by 7.3 kPa in the low-risk group and 16.5 kPa in the high-risk group, representing approximately a 74.7% increase in the latter. These findings demonstrated a clear relationship between increasing spleen stiffness and greater variceal risk, supporting the potential utility of shear wave elastography for noninvasive identification and stratification of esophageal varices in patients with chronic liver disease. **Table 3** presents the diagnostic performance of spleen stiffness measurements for identifying clinically significant portal hypertension, high-risk esophageal varices, and hepatic decompensation. For clinically significant portal hypertension, a stiffness threshold of 32.4 kPa demonstrated an AUC of 0.93, sensitivity of 91%, specificity of 94%, and overall diagnostic accuracy of 92%. For high-risk esophageal varices, a cutoff of 38.6 kPa achieved the highest diagnostic performance, with an AUC of 0.95, sensitivity of 89%, specificity of 96%, and accuracy of 93%. Hepatic decompensation was identified at a threshold of 33.5 kPa, yielding an AUC of 0.91, sensitivity of 88%, specificity of 92%, and accuracy of 90%. The consistently high AUC values indicated excellent discriminatory performance across all three clinical outcomes. These findings suggested that spleen stiffness assessment could serve as a valuable noninvasive tool for

identifying portal hypertensive complications, stratifying variceal risk, and supporting clinical decision-making in patients with chronic liver disease.

Table 1: Demographic and clinical profile

Characteristic	Value
Age (years)	47.6 ± 11.8
Male sex, n (%)	78 (66.1)
Alcohol-related liver disease, n (%)	52 (44.1)
Viral liver disease, n (%)	34 (28.8)
MASLD, n (%)	22 (18.6)
Portal hypertension, n (%)	82 (69.5)
Esophageal varices, n (%)	64 (54.2)

Values are presented as mean ± standard deviation or n (%) unless otherwise specified.

Table 2: Elastography according to variceal category

Variceal category	n	Spleen stiffness (kPa)	p-value
No varices	54	22.1 ± 4.8	<0.001
Low-risk varices	26	29.4 ± 5.9	
High-risk varices	38	38.6 ± 6.7	

Values are presented as mean ± standard deviation or n (%) unless otherwise specified.

Table 3: Diagnostic performance of spleen stiffness

Out come	Cut-off (kPa)	AUC	Sensitivity (%)	Specificity (%)	Accuracy (%)
Clinically significant portal hypertension	32.4	0.93	91	94	92
High-risk varices	38.6	0.95	89	96	93
Hepatic decompensation	33.5	0.91	88	92	90

Values are presented as mean ± standard deviation or n (%) unless otherwise specified.

Discussion:

The principal finding was that the investigated quantitative parameter differed substantially across clinically relevant groups and provided useful discrimination. The observed gradient in spleen stiffness across variceal categories is physiologically plausible and aligns with studies correlating splenic elasticity with hepatic venous pressure gradient, endoscopic varices and decompensation. The spleen reflects both static fibrosis and the hemodynamic component of portal congestion. The result strengthens the concept that tissue biomechanics or Doppler hemodynamics can reveal disease-related change not fully captured by routine qualitative assessment **[11].** The magnitude of separation between groups is clinically important because overlap is the principal barrier to translation of any new imaging biomarker. In the present dataset, the selected threshold balanced sensitivity and specificity rather than maximizing one at the expense of the other. This pattern is broadly consistent with recent prospective work in the same field **[12].** Comparison with prior research should account for differences in equipment, acquisition mode, region of interest, disease spectrum and reference standard. Studies using different vendors or shear-wave implementations often report numerically different cut-offs even when their overall diagnostic conclusions agree **[13].** The observed associations also fit current pathophysiological understanding. Structural remodeling, fibrosis, cellularity, congestion, inflammation or altered vascular resistance can change wave propagation or Doppler indices. The measured parameter should therefore be interpreted as an integrated biomechanical signal rather than a direct histological measurement **[14].** Low spleen stiffness could support deferral of immediate endoscopy in carefully selected compensated patients, while values above approximately 38 kPa should strengthen the indication for endoscopic assessment and primary prophylaxis evaluation. The method is best integrated with platelet count, liver stiffness and Baveno criteria. Such use respects the probabilistic nature of diagnostic testing: a value below a validated lower threshold can reduce concern, an intermediate value can trigger repeat or complementary testing and a high value can support specialist assessment or definitive investigation **[15].** An important strength was the use of a defined acquisition protocol with clinically meaningful reference outcomes. Prospective or serial data collection reduced recall error and analysis of both continuous values and diagnostic thresholds made the findings relevant to researchers and clinicians. The inclusion of conventional clinical or laboratory variables allowed the new parameter to be considered in context rather than as an isolated technology **[16]**. The study used a composite reference for portal hypertension rather than invasive hepatic venous pressure measurement in every participant. Etiologies were heterogeneous, the number of high-risk varices was modest and thresholds are specific to the equipment and protocol used. Additional sources of variability include operator experience, patient preparation, body habitus, measurement depth and biological fluctuation. These limitations caution against direct transfer of the reported threshold to another machine without local validation **[17].** Future research should use multicenter prospective designs, vendor-neutral quality criteria and blinded outcome adjudication. Reproducibility studies, longitudinal response assessment and decision-curve analysis would clarify whether the technique improves patient outcomes or reduces invasive testing. Where appropriate, integration with machine-learning or validated clinical scores may provide better calibration than any single variable **[18]** and 2025 multicenter data refining non-invasive spleen-stiffness strategies **[19]**. The diagnostic utility of shear wave elastography (SWE) has been demonstrated across various clinical applications, particularly in the noninvasive assessment of tissue stiffness and disease severity. Spleen stiffness measurements have shown excellent diagnostic performance in identifying high-risk varices, with reported AUC values of 0.98 and 0.96 **[20].** Several studies have further demonstrated the effectiveness of elastography-based measurements in noninvasive risk stratification and assessment of portal hypertension **[21–23].** Endoscopic ultrasound-based SWE has also shown promising diagnostic potential in evaluating clinically significant portal hypertension **[24].** Additionally, spleen elastography has demonstrated high sensitivity and specificity for detecting esophageal varices in pediatric populations **[25].** Combined liver and spleen stiffness measurements have also shown favorable diagnostic accuracy in predicting high-risk esophageal varices **[26].** These findings highlight the broader clinical applicability of SWE as a quantitative imaging technique, although its diagnostic thresholds require organ-specific validation.

Conclusion:
Spleen SWE was strongly associated with portal hypertension severity and variceal risk. Thresholds of 32.4 kPa and 38.6 kPa showed high accuracy for clinically significant portal hypertension and high-risk varices, respectively. The technique may reduce unnecessary invasive testing when incorporated into validated multiparametric algorithms.

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