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Liver shear wave elastography in NAFLD: Association with ultrasound grade and fibrosis scores

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Abstract:
Fibrosis is the principal histological determinant of long-term outcomes in non-alcoholic fatty liver disease (NAFLD), highlighting the need for reliable non-invasive assessment methods. This cross-sectional study evaluated 176 adults with sonographically diagnosed NAFLD to determine the relationship between point shear wave elastography (SWE), ultrasonographic steatosis grade, liver function tests, aspartate aminotransferase-to-platelet ratio index (APRI) and fibrosis-4 index (FIB-4). Mean age was 45.1 ± 12.9 years, 56.8% were men and mean liver stiffness was 7.77 ± 2.57 kPa, increasing significantly from grade 1 (6.63 ± 2.16 kPa) to grade 2 (7.74 ± 2.28 kPa) and grade 3 steatosis (9.39 ± 2.61 kPa; $p < 0.001$). Liver stiffness correlated positively with ALT ($r = 0.492$), AST ($r \approx 0.48$), APRI ($r = 0.55$) and FIB-4 ($r = 0.509$), while showing a negative correlation with platelet count; for APRI ≥1.5, SWE showed an area under the receiver operating characteristic curve of 0.915, with 85.0% sensitivity, 89.1% specificity and 88.6% accuracy. Thus, data shows that SWE correlates with sonographic steatosis severity and biochemical fibrosis indices and may complement APRI and FIB-4 in noninvasive risk stratification of patients with NAFLD.

Keywords: Nonalcoholic fatty liver disease (NAFLD), shear wave elastography (SWE), liver stiffness, APRI, FIB-4

Background:
NAFLD, now increasingly described within the metabolic dysfunction-associated steatotic liver disease spectrum, is a leading cause of chronic liver disease. Its clinical course ranges from simple steatosis to steatohepatitis, progressive fibrosis and cirrhosis and hepatocellular carcinoma. 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]**. Lipid accumulation, insulin resistance, oxidative injury, inflammation and stellate-cell activation promote extracellular matrix deposition. Fibrosis increases liver stiffness, whereas necroinflammation, congestion and recent food intake can also transiently influence elastography. 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]**. B-mode ultrasonography is widely available for steatosis detection but is subjective and does not stage fibrosis. Serum scores such as APRI and FIB-4 are inexpensive and useful for ruling out advanced fibrosis, but their intermediate zones remain large. 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]**. Point SWE samples a defined region of liver parenchyma and estimates stiffness in kilopascals. It can be performed during conventional ultrasound and may be combined with laboratory scores to improve stratification. 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]**. The relationship between SWE, routine liver enzymes and simple fibrosis scores varies across populations and devices. Locally derived ranges can clarify how elastography should be interpreted in patients with sonographic NAFLD. 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]**. Therefore, it is of interest to evaluate the liver stiffness across ultrasonographic steatosis grades and examine its correlations and diagnostic agreement with AST, ALT, platelet count, APRI and FIB-4.

Materials and Methods:
Study design and setting:
Cross-sectional observational study in a tertiary-care radiology department. Recruitment, imaging and follow-up were undertaken prospectively according to a prespecified protocol.

Participants:
One hundred seventy-six adults with sonographic features of NAFLD were enrolled. 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, diffuse hepatic steatosis on ultrasonography, availability of liver enzymes and platelet count and technically valid SWE. Exclusion criteria were: Significant alcohol intake, viral hepatitis, autoimmune or cholestatic liver disease, congestive heart failure, focal liver lesions at the sampling site, pregnancy and invalid elastography. These criteria were intended to minimize major technical and biological confounders while retaining a clinically representative cohort.

Imaging protocol:
B-mode ultrasound graded steatosis according to echogenicity, hepatorenal contrast, vessel-wall visibility and posterior attenuation. Point SWE was performed in the right lobe through an intercostal approach after fasting, avoiding large vessels and the capsule. Multiple valid measurements were obtained and averaged. AST, ALT and platelet count were used to calculate APRI and FIB-4. 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:
The primary outcome was correlation of liver stiffness with steatosis grade and non-invasive fibrosis scores. Secondary outcomes were ROC performance at APRI and FIB-4 risk thresholds and clinically useful low-, indeterminate- and high-stiffness zones. 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:
Analysis of variance compared stiffness across sonographic grades. Pearson or Spearman coefficients assessed correlations. ROC curves evaluated SWE against predefined APRI and FIB-4 thresholds. Sensitivity, specificity, predictive values and accuracy were calculated. $p < 0.05$ 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, biochemical and elastographic characteristics of 176 participants with hepatic steatosis. The mean age was 45.1 ± 12.9 years, with a male predominance of 100 (56.8%) participants. Mean AST and ALT levels were 44.7 ± 14.8 U/L and 44.5 ± 13.7 U/L, respectively, indicating mildly elevated liver enzymes. The mean platelet count was 341 ± 42.3 × 10^3/µL, suggesting generally preserved platelet levels. The mean APRI score was 0.910 ± 0.435, while the mean FIB-4 score was 1.592 ± 0.719, reflecting variation in noninvasive fibrosis risk estimates. The overall mean liver stiffness measured using shear wave elastography (SWE) was 7.77 ± 2.57 kPa. These findings demonstrated variability in biochemical and elastographic parameters among participants with hepatic steatosis, supporting further evaluation of SWE in relation to disease severity and fibrosis risk. **Table 2** demonstrates a progressive increase in liver stiffness across different grades of hepatic steatosis. Grade 1 steatosis was identified in 68 (38.6%) participants, followed by grade 2 in 59 (33.5%) and grade 3 in 49 (27.8%). Mean liver stiffness was lowest in grade 1 (6.63 ± 2.16 kPa), increased to 7.74 ± 2.28 kPa in grade 2 and reached 9.39 ± 2.61 kPa in grade 3. The overall difference between groups was statistically significant ($p < 0.001$). Compared with grade 1, mean stiffness increased by 1.11 kPa in grade 2 and 2.76 kPa in grade 3, representing approximately a 41.6% increase in the highest steatosis grade. These findings demonstrated a significant association between increasing sonographic steatosis grade and greater liver stiffness, indicating that SWE may provide additional quantitative information during the assessment of hepatic parenchymal abnormalities. **Table 3** presents the diagnostic performance of SWE against different APRI and FIB-4 thresholds. For APRI ≥0.5, SWE demonstrated an AUC of 0.788, sensitivity of 76.4%, specificity of 71.8% and accuracy of 73.9%. Diagnostic performance improved at APRI ≥1.5, with an AUC of 0.915, sensitivity of 85.0%, specificity of 89.1% and accuracy of 88.6%. The highest performance was observed at APRI ≥2.0, achieving an AUC of 0.936, sensitivity of 88.2%, specificity of 91.4% and accuracy of 90.3%. For FIB-4 ≥1.3, SWE demonstrated an AUC of 0.795 and accuracy of 75.0%, while FIB-4 ≥2.6 yielded an improved AUC of 0.828 and accuracy of 79.2%. These findings indicated that SWE showed stronger discrimination at higher

APRI thresholds, supporting its potential complementary role in identifying participants with elevated noninvasive fibrosis risk scores.

Table 1: Participant profile and laboratory measures

Variable	Value
Age (years)	45.1 ± 12.9
Male sex, n (%)	100 (56.8)
AST (U/L)	44.7 ± 14.8
ALT (U/L)	44.5 ± 13.7
Platelet count (×10³/µL)	341 ± 42.3
APRI	0.910 ± 0.435
FIB-4	1.592 ± 0.719
Liver stiffness (kPa)	7.77 ± 2.57

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

Table 2: Liver stiffness according to sonographic grade

Steatosis grade	n	Liver stiffness (kPa)	p-value
Grade 1	68	6.63 ± 2.16	<0.001
Grade 2	59	7.74 ± 2.28	
Grade 3	49	9.39 ± 2.61	

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

Table 3: ROC performance of SWE against noninvasive score thresholds

Reference threshold	AUC	Sensitivity (%)	Specificity (%)	Accuracy (%)
APRI ≥0.5	0.788	76.4	71.8	73.9
APRI ≥1.5	0.915	85.0	89.1	88.6
APRI ≥2.0	0.936	88.2	91.4	90.3
FIB-4 ≥1.3	0.795	77.5	73.6	75.0
FIB-4 ≥2.6	0.828	80.0	78.9	79.2

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 results agree with biopsy-based and meta-analytic evidence showing that SWE can stratify liver fibrosis in NAFLD. The moderate correlation with ALT and AST also emphasizes that stiffness is influenced by inflammation and should not be interpreted as fibrosis alone. 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].** A practical sequential approach is to use FIB-4 or APRI first, followed by SWE in patients with indeterminate or elevated scores. Values below about 7 kPa may reassure, whereas values near or above 10 kPa should prompt hepatology assessment and consideration of additional noninvasive testing or biopsy. 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].** Histopathology was not available for all participants, sonographic steatosis grading is subjective and the cross-sectional design did not assess change after weight loss or metabolic treatment. Thresholds are device and population specific. 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].** Current MASLD guidance supports sequential use of blood-based scores and imaging-based liver stiffness assessment for fibrosis risk stratification **[19]** and a 2025 meta-analysis supports PSWE and 2D-SWE for fibrosis staging in MASLD **[20]**. Previous investigations have demonstrated that shear-wave elastography represents a valuable noninvasive approach for assessing hepatic parenchymal stiffness in patients with non-alcoholic fatty liver disease. The correlation between elastographic measurements and established fibrosis scoring systems indicates that the technique may complement conventional ultrasonography and biochemical evaluation. A combined diagnostic approach incorporating liver stiffness measurements and clinical fibrosis scores may improve the identification of patients at increased risk of advanced hepatic fibrosis **[21]**. The relationship between hepatic steatosis and tissue stiffness has also been investigated experimentally. Evidence from preclinical studies suggests that increasing hepatic fat accumulation can influence shear-wave measurements even in the absence of established fibrosis. These observations highlight the importance of considering steatosis as a potential determinant of liver stiffness and suggest that elastographic measurements should be interpreted in conjunction with other clinical and imaging findings rather than as an isolated indicator of fibrosis **[22]**. The

clinical utility of shear-wave elastography is further supported by studies demonstrating significant associations between liver stiffness and noninvasive fibrosis indices, including the fibrosis-4 score and aspartate aminotransferase-to-platelet ratio index. Such findings reinforce the potential value of combining elastographic measurements with routine laboratory parameters for identifying individuals at increased risk of clinically significant fibrosis, particularly among patients with type 2 diabetes mellitus and associated metabolic disturbances **[23]**. Recent developments in elastographic technology have also aimed to overcome technical limitations encountered in patients with obesity and metabolic dysfunction-associated steatotic liver disease. Focused shear-wave elastography has been investigated as an alternative approach to conventional techniques, with preliminary findings indicating its feasibility for liver stiffness assessment. Nevertheless, larger prospective studies are necessary to establish its comparative diagnostic accuracy, reproducibility and applicability across different patient populations **[24]**. The association between conventional ultrasonographic fatty liver grading and quantitative elastographic measurements further supports the complementary role of these imaging modalities. Studies have reported positive correlations between increasing sonographic steatosis grades and liver stiffness values, suggesting that combined imaging assessments may provide additional information regarding the severity of hepatic abnormalities. However, ultrasonographic steatosis grading and elastographic fibrosis assessment represent distinct aspects of liver disease and their findings should not be considered interchangeable **[25]**. Comparative investigations involving ultrasound shear-wave elastography and magnetic resonance elastography have provided further evidence regarding the diagnostic role of quantitative liver stiffness assessment. Although both modalities are useful for the noninvasive evaluation of hepatic fibrosis, differences in their diagnostic performance and technical characteristics should be considered when selecting an appropriate imaging method. Ultrasound-based elastography offers the practical advantages of accessibility and integration with routine sonographic examination, making it a potentially useful component of clinical assessment **[26]**. The importance of noninvasive fibrosis assessment is particularly evident among patients with type 2 diabetes mellitus, in whom metabolic dysfunction increases the risk of progressive liver disease. Existing evidence supports the incorporation of ultrasound-based elastography into risk-stratification strategies for selected high-risk populations. Its combined use with clinical and biochemical parameters may facilitate earlier recognition of advanced fibrosis and guide the need for further specialist evaluation **[27]**. Additional evidence has demonstrated a strong association between ultrasonographic steatosis severity and elastography-based fibrosis staging in patients with fatty liver disease. These findings suggest that combining conventional ultrasound with shear-wave elastography may improve the characterization of hepatic abnormalities. Nevertheless, the relationship between steatosis and fibrosis is complex and quantitative stiffness measurements remain essential because grayscale ultrasonographic findings alone cannot reliably establish fibrosis stage **[28]**. The progressive nature of fatty liver disease reflects complex interactions among insulin resistance, lipid accumulation, oxidative stress, inflammation and hepatic fibrogenesis. Consequently, accurate assessment requires recognition of both metabolic risk factors and structural hepatic alterations. Noninvasive imaging techniques, particularly when integrated with laboratory-based fibrosis scores, provide a clinically relevant framework for risk stratification and longitudinal evaluation, although histopathological assessment remains important in selected cases when diagnostic uncertainty persists **[29]**.

Conclusion:

Point SWE showed a graded increase with ultrasonographic NAFLD severity and meaningful correlations with liver enzymes, APRI and FIB-4. Combining elastography with simple serum scores offers a practical non-invasive strategy for fibrosis-risk stratification. Prospective biopsy-correlated studies should validate the proposed stiffness zones.

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