Assessment of Liver and Splenic Stiffness Using Shear Wave Elastography in Pediatric Patients with Hepatic Fibrosis

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Abstract

Background: Chronic liver diseases in children, often progress to cirrhosis and fibrosis. Accurate staging of liver fibrosis is crucial for treatment and prognosis. Noninvasive methods like shear wave elastography (SWE) have emerged as alternatives to liver biopsy. Both liver and spleen SWE show diagnostic potential in pediatric chronic liver disease.

Objectives: This study evaluated SWE's accuracy versus liver biopsy in staging hepatic fibrosis and assessed the reliability of spleen SWE in pediatric patients.

Patients and methods: A cross-sectional analysis of 115 children (70 with liver disease, 45 healthy controls) was conducted at Abu El Reesh Pediatric Hospital. Liver and spleen stiffness were measured via SWE and compared with biopsy findings.

Results: There was no age or gender differences between patients and controls ($p>0.05$). Patients with chronic liver disease had significantly higher liver SWE (6.9±3.68kpa vs. 4.23±1.22kpa, $p<0.001$) and (3.19±1.77 m/s vs. 1.86±0.5 m/s, $p<0.001$). In particular, patients with biliary atresia exhibited the highest stiffness values (19.7±15.2 kPa). Patients had higher spleen SWE (5.16±3.2kpa vs. 3.5±1.25kpa, $p=0.003$) and (2.78±0.7 m/s vs. 2.49±1.18, $p=0.081$) than the control group, yet it was statistically insignificant. ROC analysis demonstrated liver elastography (AUC=0.744) as a robust predictor of liver disease, while spleen elastography was a moderate predictor (AUC=0.666). Liver SWE (Kpa) showed strong correlation with METAVIR stages ($r=0.699$, *p*<0.001) while Spleen SWE (Kpa) showed moderate correlation ($r=0.565$, *p*<0.001).

Conclusion: Liver and spleen SWE reliably detect hepatic fibrosis stages in children, offering a noninvasive alternative to biopsy. These findings underscore SWE's clinical utility in pediatric liver disease management.

Keywords: Liver; Spleen; SWE; Pediatric; Chronic liver diseases.

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Introduction

Chronic liver disease (CLD) in children, driven by etiologies such as biliary atresia (20%), autoimmune hepatitis (14.3%), and portal hypertension (14.3%), frequently progresses to hepatic fibrosis and cirrhosis. Pediatric liver and immune system functions differ significantly from adults, and most childhood liver disorders currently lack definitive treatments. Therefore, a deeper understanding of disease-specific fibrogenesis is crucial **(Ozdogan and Arikan, 2022; Galina et al., 2021)**.

Splenomegaly and hypersplenism are common but relatively non-fatal complications of liver cirrhosis, especially in the absence of bleeding varices. Splenic enlargement often coexists with hypersplenism and is associated with thrombocytopenia and cytopenia in cirrhotic patients. Although these conditions are significant clinical indicators of cirrhosis, they do not necessarily reflect the disease stage. The underlying mechanisms of splenic changes in cirrhosis remain unclear, though altered hemodynamics, tissue damage, and inflammatory signaling are considered major contributors **(Li et al., 2017).**

Fibrosis staging plays a vital role in determining treatment and prognosis. Although liver biopsy has been the gold standard for fibrosis assessment, its limitations have driven interest in noninvasive diagnostic alternatives **(Kennedy et al., 2018).**

Non-invasive methods, including shear wave elastography (SWE), have emerged to quantify tissue stiffness as a surrogate for fibrosis severity. SWE measures liver and spleen stiffness, which correlate with portal hypertension and clinical outcomes such as variceal bleeding and decompensation. **(Giunta et al, 2016).**

While SWE is well-established in adult hepatology, pediatric data remain limited due to anatomical and physiological differences, including smaller organ size and variable cooperation during imaging. This study aims to evaluate the correlation between SWE-measured liver/spleen stiffness and histological fibrosis stages in children. Additionally to assess the diagnostic accuracy of SWE in differentiating pediatric CLD patients from healthy controls. Also to compare liver and spleen stiffness measurements with established biomarkers (APRI, FIB-4).

Patients and methods

Study Design and Population

This cross-sectional study was conducted in the Radiology and Hepatology departments of Abu El Reesh Pediatric Hospital from October 2024 till March 2025 that was approved by Cairo university faculty of medicine research ethics committee (ethical approval no.: MS-130-2024). The study enrolled 115 children (70 CLD cases, 45 healthy controls) aged 6 months–18 years. Patients were enrolled after obtaining a written, informed consent from their parents or guardians. The study was conducted in accordance with ethical principles for medical research and the declaration of Helsinki.

Inclusion Criteria

- Confirmed hepatic fibrosis via liver biopsy (METAVIR staging).
- Clinical and laboratory evidence of CLD (e.g., elevated ALT, AST, thrombocytopenia).

Exclusion Criteria

- Severe obesity (BMI >95th percentile).
- Ascites or recent abdominal surgery.
- Acute infections or hemodynamic instability.

Shear Wave Elastography (SWE)

Was done for all patients and control using Canon APLIO 400 ultrasound system with a 5–7 MHz curvilinear transducer.

Patients fasted for ≥2 hours. Measurements were taken in the supine position with the right arm elevated to optimize intercostal access. Three valid measurements per organ were averaged.

The transducer was kept in a stable position for few seconds perpendicularly to reduce the compression artifact.

During a dedicated ultrasound examination, the spleen and liver dimensions were calculated.

The parenchyma was assessed based on echogenicity, the extent of heterogeneity, and then 2D-SWE software was activated in split-screen mode.

Elasticity is then qualitatively assessed using color-coded image; in which red refers to stiffer tissues and blue refers to softer tissues.

The investigation involved 3 measurements with a 3 mm in diameter round-shaped ROI avoiding large blood vessels.

All measurements were recorded as m/sec and Kilopascal. The mean stiffness value was calculated by averaging the 3 measurements.

Laboratory and Histopathological Assessments

-Blood Tests: Alanine aminotransferase (ALT), aspartate aminotransferase (AST), g-glutamyltransferase (GGT), alkaline phosphatase (ALP), bilirubin, albumin, platelet count, INR. - Biomarkers: AST to Platelet Ratio Index (APRI) and FIB-4 scores calculated.

- Liver Biopsy: METAVIR staging (F0–F4) performed by blinded pathologists.

- Endoscopy: Variceal grading (Grade I–IV) for portal hypertension assessment.

Statistical analysis:

- Continuous variables reported as mean±SD or median.

- Group comparisons used Mann-Whitney U test.

- Correlations analyzed via Spearman's rank test.

- ROC curves evaluated diagnostic accuracy (AUC, sensitivity, specificity).

- SPSS v26 used for analysis; significance threshold: *p*<0.05.

Results

Demographics and Clinical Characteristics

- Age and Gender: The case group's mean age was 6.04±4.17 years and included 36 (51.4%) males and 34 (48.6%) females while the control group's mean age was 5.67 ± 3.5 and included 24 (53.3%) males and 21 (46.7%) females. There was no age or gender differences between the two groups (p>0.05).

-Laboratory findings: Laboratory results of cases revealed elevated levels of ALT, AST, ALP and GGT (median, IQR) as follows; 63.0 (34.75-119.75), 86.5 (51.75-129.25), 258.5 (171.5-328.75), and 86.5 (41.75-144.75) respectively

-Etiologies: Biliary atresia (20%), autoimmune hepatitis (14.3%), and portal hypertension (14.3%) were most prevalent. Variceal staging and fibrosis distribution as showed in **(Table.1)**

Table 1. Variceal grades and Liver Biopsy findings among cases (n=70).

Variables	**N (%)**
Variceal grading	
No varices	31 (44.3)
Grade I	8 (11.4)
Grade II	14 (20.0)
Grade III	10 (14.3)
Grade IV	3 (4.3)
Endoscopy not done *	4 (5.7)

Liver Biopsy findings	
0 (No Fibrosis)	13 (18.6)
I (Mild Fibrosis)	17 (24.3)
II (Moderate Fibrosis)	30 (42.9)
III (Severe Fibrosis)	6 (8.6)
IV (Bridging Fibrosis)	1 (1.4)
Cirrhosis	0 (0)
Not done *	3 (4.3)

SWE Measurements

Patients with chronic liver disease had significantly higher liver SWE (6.9±3.68kpa vs. 4.23±1.22kpa, p <0.001) and (3.19±1.77 m/s vs. 1.86±0.5 m/s, p<0.001). In particular, patients with biliary atresia exhibited the highest stiffness values (19.7±15.2 kPa). Patients had higher spleen SWE (5.16±3.2kpa vs. 3.5±1.25 kPa, p=0.003) and (2.78±0.7 m/s vs. 2.49±1.18, p=0.081) than the control group, yet it was statistically insignificant as illustrated in (**Table.2).**

There was no correlation between APRI or FIB4 scores and liver or spleen SWE; as illustrated in **(Table 3).**

Table 2. Liver and spleen SWE measurements in cases versus control

Variables	**Cases (n=70)**	**Control (n=45)**	**P ***
Liver SWE (kpa)			**<0.001**
(mean± SD)	6.9±3.68	4.23±1.22	
Liver SWE (m/s)			**<0.001**
(mean± SD)	3.19±1.77	1.86±0.5	
Spleen SWE (kPa)			**0.003**
(mean± SD)	5.16±3.2	3.5±1.25	
Spleen SWE (m/s)			0.081
(mean± SD)	2.49±1.18	2.78±0.7	

Table 3.Correlation between liver and spleen SWE and lab findings.

Variables	**Liver SWE**		**Spleen SWE**	
	r	**P value**	**r**	**P value**
APRI score	-0.094	0.437	-0.113	0.354
FIB4 score	-0.046	0.702	-0.100	0.408

Grade III and IV liver fibrosis showed higher APRI and FIB4 scores than lower grades **(Fig.1)**, however, without significant difference between groups; as illustrated in (**Table .4)**.

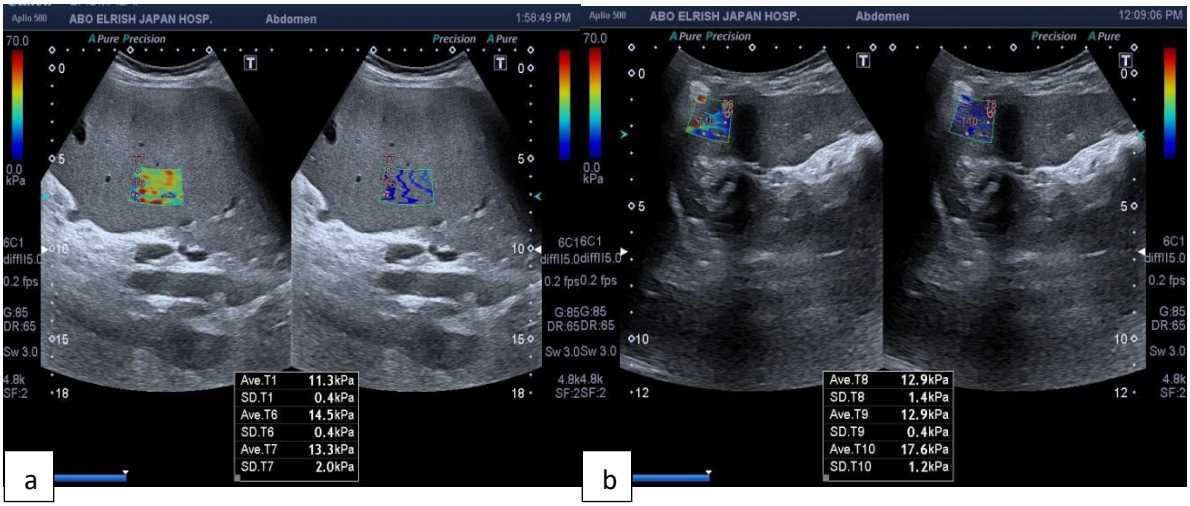

Fig.1. A 10-year-old girl with portal hypertension diagnosed 2 years ago, currently under routine follow-up. Recent laboratory findings revealed: Normal (ALT 47, AST 75 and ALP 229); hyperbilirubinemia (Bil T/D 2.7/1.35) with normal coagulation profile(PT/INR 14.9/1.1) and serum albumin level was within normal range (3.6).The endoscopic examination showed grade I esophageal varices and the liver biopsy showed mild degree of fibrosis. The portal vein diameter was within normal limit (8mm). The APRI score (8.1) and FIB4 score (4.3) were markedly elevated.(a) liver average SWE :13 Kpa (b) Spleen average SWE:14.47 Kpa

Table 4.Relation between liver fibrosis and lab findings

Variables	0 (No Fibrosis)	I (Mild Fibrosis)	II (Moderate Fibrosis)	III (Severe Fibrosis)	IV (Bridging Fibrosis)	P value *
APRI score	1.07 (0.88-4.3)	1.3 (0.7-1.9)	1.3 (0.6-2.1)	2.03 (0.4-9.4)	2.2 (2.2-2.2)	0.778
FIB4 score	0.6 (0.06-1.2)	0.34 (0.08-0.7)	0.24 (0.1-0.48)	0.7 (0.05-0.9)	0.48 (0.48-0.48)	0.854

Diagnostic Accuracy

ROC curve analysis revealed that SWE (kPa) was a good predictor of chronic liver disease, with AUC=0.744, Sensitivity, Specificity, PPV, NPV, and Accuracy of 52.9%, 95.6%, 94.9%, 56.6% and 69.6% respectively at a cut-off ≥6.35 kPa. **(Fig.2).**

ROC curve analysis revealed that spleen SWE (KPa) was a fair predictor of liver disease, with AUC=0.666, Sensitivity, Specificity, PPV, NPV, and Accuracy of 27.1%, 97.8%, 95%, 46.3% and 54.8% respectively at a cut-off >6kpa; (**Fig.3)**

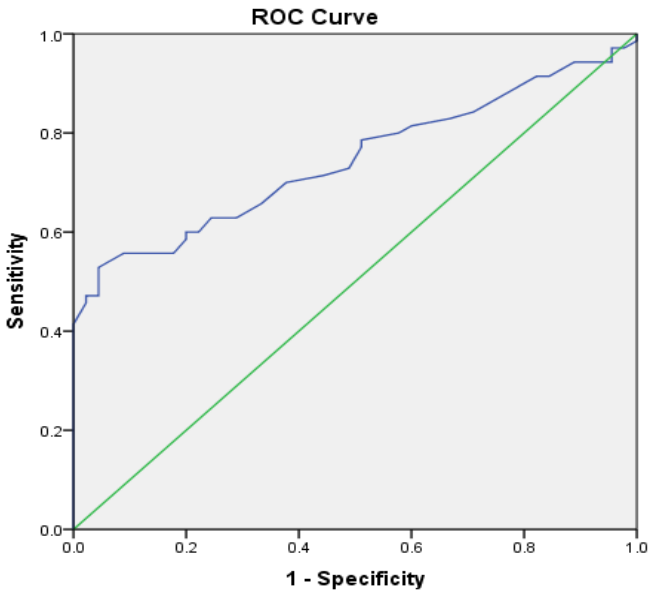

Fig .2. AUC describing the discriminatory ability of liver SWE (kPa) in liver disease

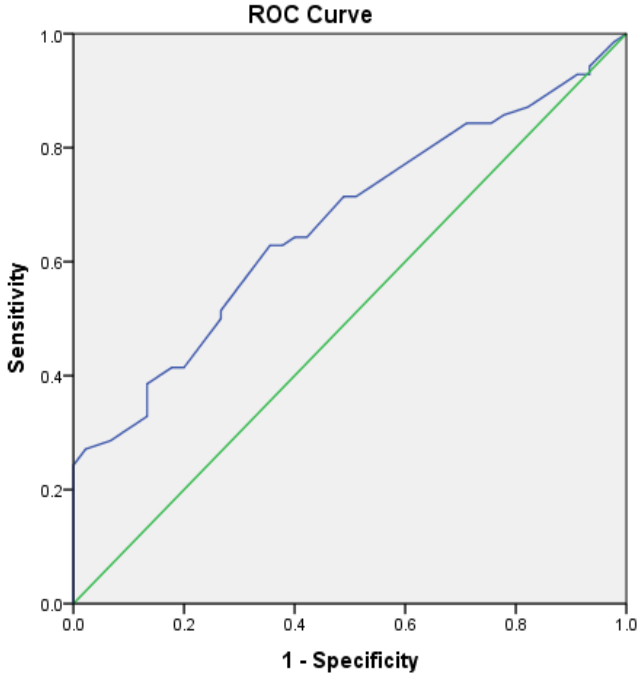

Fig.3. AUC describing the discriminatory ability of spleen elastography (kPa) in liver disease.

Correlation with fibrosis Stages

Liver SWE (kPa) showed Strong correlation with METAVIR stages (r=0.699, *p*<0.001) while Spleen SWE (kPa) showed moderate correlation (r=0.565, *p*<0.001).

Discussion

The rising prevalence of chronic liver disease in children underscores the

need for non-invasive diagnostic tools to assess fibrosis and portal hypertension. While liver biopsy remains the gold standard, its invasiveness and sampling limitations have driven interest in shear wave elastography (SWE), which evaluates larger hepatic areas and correlates with fibrosis severity **(Ozdogan and Arikan, 2022; Jusufi et al., 2022)**. Spleen stiffness measurement via SWE has emerged as a dynamic marker of PH, offering superior diagnostic accuracy for complications like esophageal varices compared to liver stiffness **(Giunta et al., 2016; Reiberger, 2022)**. This study evaluated SWE's accuracy in staging liver fibrosis and explored spleen stiffness as a correlate of disease severity in pediatric CLD.

Our cohort included 115 children (70 CLD, 45 controls) with a mean age of 5.85 ± 3.84 years, younger than populations in prior studies. For instance, Hebelka et al. studied 86 children (mean age 10.2 years), while Cetinic et al. and Upadhyay et al. included older cohorts (median ages 12.1 and 10.0 years, respectively **(Hebelka et al, 2022; Cetinic et al., 2022; Upadhyay et al., 2025)**. Despite age differences, our findings align with these studies in demonstrating SWE's utility across pediatric age groups.

Our findings demonstrate that liver stiffness measurements (LSM) via SWE strongly correlate with histological fibrosis stages ($r=0.699$, $p<0.001$), reinforcing its validity as a surrogate marker for fibrotic burden. These results align with prior pediatric studies **(Galina et al., 2021; Cetinic et al., 2022)**

Liver SWE and fibrosis scores were significantly higher in CLD patients than controls (SWE: 3.19 ± 1.77 vs. 1.86 ± 0.5 m/s, $p <0.000$; fibrosis: 6.9 ± 3.68 vs. 4.23 ± 1.22 kPa, $p< 0.000$). ROC analysis showed that liver SWE cut-off value ≥6.35 kPa had moderate diagnostic accuracy AUC=0.744, Sensitivity, Specificity, PPV, NPV, and Accuracy of 52.9%, 95.6%, 94.9%, 56.6% and 69.6%, consistent with Hebelka et al. who identified SWE cut-offs (≤4.5 kPa for excluding significant fibrosis, AUC = 0.77), reinforcing SWE's role in early fibrosis detection **(Hebelka et al., 2022).** Notably, Galina et al. highlighted disease-specific cut-offs (e.g., 16 kPa for biliary atresia), underscoring SWE's adaptability to diverse CLD etiologies **(Galina et al., 2021)**.

The progressive increase in liver stiffness values across METAVIR stages (F0-F4) mirrors the pathophysiological continuum of fibrogenesis. Notably, patients with biliary atresia exhibited the highest stiffness values (19.7±15.2 kPa), consistent with the rapid fibrotic progression characteristic of this disease. The diagnostic accuracy of liver SWE in our study (AUC=0.744) was robust, though slightly lower than adult studies (AUC=0.85–0.92) **(Kumar et al., 2001)**. This discrepancy may reflect:

Technical challenges: Smaller liver size and variable cooperation in children can reduce measurement precision.

Disease heterogeneity: Pediatric CLD etiologies (e.g., metabolic disorders, congenital fibrosis) may influence stiffness differently than adult causes (e.g., viral hepatitis).

Dynamic liver remodeling: Pediatric livers have greater regenerative capacity, potentially altering stiffness- fibrosis relationships.

Spleen stiffness correlated moderately with liver fibrosis ($r = 0.565$). While spleen SWE values were higher in CLD patients (2.78 ± 0.7 vs. 2.49 ± 1.18 m/s), this difference was not significant ($p = 0.081$), contrasting with studies emphasizing splenic SWE diagnostic value. For example, Cetin et al. found significantly elevated spleen stiffness in portal hypertension (PH) cases ($*p* < 0.05$), and Upadhyay et al. reported that the splenic stiffness/liver

stiffness measurement (LSM) ratio excelled in diagnosing non-cirrhotic portal fibrosis (AUC = 0.992) **(Upadhyay et al., 2025; Cetin et al., 2024)**.

Our ROC analysis for spleen elastography (cut-off >6 kPa) showed high specificity (97.8%) but low sensitivity (27.1%), limits standalone use, likely due to:

Anatomic variability: Pediatric spleens are smaller and more mobile, complicating ROI placement.

Delayed splenic response: Early fibrosis may not immediately increase splenic stiffness, unlike in advanced cirrhosis.

Splenic SWE may complement liver SWE where elevated splenic stiffness could signal impending portal hypertensive complications (e.g., variceal bleeding).

APRI and FIB-4 scores correlated poorly with SWE in our cohort, consistent with other pediatric studies highlighting their limitations in non-alcoholic fatty liver disease (NAFLD) and biliary disorders **(Mann et al., 2016)**.

SWE's advantage lies in: Direct tissue assessment: Unlike serum biomarkers, SWE is unaffected by extrahepatic inflammation or malnutrition; Real-time imaging: Combines structural (B-mode) and functional (stiffness) data in a single exam.

Limitations and Future Directions: Larger cohorts are needed to validate cutoffs across etiologies. Prospective studies should assess SWE's prognostic value for decompensation events. Technological refinement: Pediatric-specific transducers and motion-artifact algorithms would improve reliability.

Conclusion

SWE is a promising non-invasive tool for pediatric CLD, with liver stiffness providing reliable fibrosis staging and spleen stiffness offering insights into PH severity hence combined LSM/SSM ratios can eventually optimize diagnostic accuracy in pediatric hepatology.

Declaration of conflicting interests : The author(s) have no conflicts of interest to declare with respect to the content of this manuscript.

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