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ORIGINAL ARTICLE

Integration of 2D Shear Wave Elastography, Doppler Hemodynamics, and Clinical Scores for Noninvasive Assessment of Liver Fibrosis in Chronic Liver Disease

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Abstract

Purpose: Chronic liver disease (CLD) is a major global health burden with increasing prevalence in India due to rising alcohol use and non-alcoholic fatty liver disease (NAFLD). Non-invasive tools such as shear wave elastography (SWE) and Doppler ultrasound are gaining importance in evaluating liver fibrosis and associated hemodynamic changes. **Methods:** This cross-sectional observational study included 60 patients with CLD and 20 healthy controls who underwent 2D-SWE and Doppler ultrasound. Liver stiffness (LS) was graded according to SRU 2020 criteria. Hemodynamic parameters—spleen index (SI), portal vein velocity (PVV), portal vein width (PVW), splenoportal index (SPI), and hepatic vein waveform—were analyzed in relation to LS. Additionally, correlations were assessed with clinical fibrosis scores including AST/ALT ratio, APRI, and FIB-4. **Results:** The majority of CLD patients (51.66%) had advanced fibrosis (F4). LS showed strong positive correlations with SI, PVW, and SPI, and a significant negative correlation with PVV ($p<0.001$). Altered hepatic vein waveforms were also associated with higher LS. Non-invasive fibrosis scores were significantly elevated in CLD patients and showed strong correlation with LS and inverse correlation with PVV. **Conclusion:** SWE and Doppler ultrasound offer complementary, non-invasive evaluation of liver fibrosis and portal hemodynamics. Their integration with clinical scores enhances diagnostic accuracy and may reduce dependence on liver biopsy in CLD assessment.

Key Words

Liver Fibrosis; Chronic Liver Disease; Shear Wave Elastography; Doppler Ultrasound; Portal Hemodynamics; Non-invasive Assessment.

Introduction

Chronic liver disease (CLD), including cirrhosis, is a major global health concern and a leading cause of mortality and morbidity. In 2016, cirrhosis accounted for approximately 2.2% of global deaths[1]. The principal etiologies include viral hepatitis, alcohol-associated liver disease, and non-alcoholic fatty liver disease (NAFLD), with regional variation in prevalence. In India, the burden of CLD is increasingly driven by alcohol use and NAFLD, although viral hepatitis remains significant[2].

The etiology of liver disease in India is evolving, with a shift from hepatitis B virus (HBV) to alcohol-related and NAFLD as leading causes of cirrhosis, though viral hepatitis remains prominent[2]. Traditionally, liver biopsy has been the gold standard for diagnosing and staging

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liver fibrosis, but its invasive nature, risk of complications, and limitations in routine use have driven the need for non-invasive diagnostic tools[3,4]. Among these, elastography techniques, particularly shear wave elastography (SWE), have become increasingly important. These methods offer a reliable way to assess liver stiffness, which correlates with fibrosis severity[5].

In addition to elastography, Doppler ultrasound has emerged as a useful, non-invasive tool for assessing hepatic vascular hemodynamics, particularly portal blood flow. Changes in portal venous flow, which occur as a result of liver disease progression, are valuable indicators of liver damage[6,7].

Primary Objective: To assess the correlation between Doppler-derived hemodynamic parameters and liver stiffness measured by shear wave elastography in patients with chronic liver disease.

Secondary Objective: To evaluate the association of these imaging findings with non-invasive fibrosis scores, including AST/ALT ratio, APRI, and FIB-4.

Significance: Integrating elastography, Doppler ultrasound, and clinical fibrosis scores may improve non-invasive assessment of liver fibrosis, enhance diagnostic accuracy, and reduce reliance on liver biopsy in chronic liver disease.

Material and Methods

After Institutional Ethics Committee approval (IEC No: IEC/GMCJ/2023/1521; dated 21/09/2023), this cross-sectional observational study was conducted from October 2023 to October 2024. A total of 60 patients with suspected or diagnosed chronic liver disease were included using convenience sampling.

Sample size estimation was based on detecting a moderate correlation (r = 0.35–0.40) between non-invasive parameters, with 80% power and $\alpha = 0.05$, yielding a minimum requirement of 55 participants.

All ultrasound, Doppler, and shear wave elastography examinations were performed by a single radiologist to ensure technical consistency. The radiologist was blinded to laboratory parameters and clinical fibrosis scores at the time of imaging to minimize observer bias.

Inclusion Criteria: Adult patients with clinical, laboratory, and imaging evidence of CLD and LS >5 kPa.

Exclusion Criteria: Patients with hepatocellular carcinoma (HCC), portal/hepatic vein or IVC thrombosis, or advanced renal/cardiac failure were excluded.

Demographic and Clinical Data: Collected from medical records; informed consent obtained as applicable. All participants fasted for 4–6 hours before examination.

Ultrasound and Doppler Evaluation: B-mode USG and duplex Doppler were performed using a convex probe (1–8 MHz). Recorded parameters included liver size, parenchymal echotexture, liver margins, ascites, splenic index (SI), portal vein width, portal vein phasicity and velocity (PVV), hepatic vein waveform, and splenoportal index (SPI = SI/PVV).

Elastography: 2D-SWE was conducted via intercostal approach with the patient supine and right arm maximally abducted. Ten valid measurements were taken from the right hepatic lobe; median LS values (kPa) were used. Exams with >20% SD or IQR >30% were excluded. Fibrosis staging was done according to Update to the Society of Radiologists in Ultrasound Liver Elastography Consensus Statement published in 2020 : F0 ($\leq$ 5 kPa), F1 (>5-<9 kPa), F2 (9–13 kPa), F3 (>13 <17kPa), F4 (>17 kPa).

Laboratory Investigations: Included AST, ALT, platelet count, AST/ALT ratio, APRI, and FIB-4 index. FIB-4 was calculated using standard formula:

FIB-4 = Age × AST / [Platelets × $\sqrt{}$ALT]

Statistical Analysis: Conducted using SPSS v26. Data were expressed as mean ± SD or percentages. Groups were compared using independent t-tests, Spearman's and Pearson's correlation, and Kruskal–Wallis test. Significance was set at $p<0.05$. Patients were sub-grouped into mild (F1–F2) and significant fibrosis (F3–F4) categories for comparative analysis.

Results

The present study included a total of 60 patients with chronic liver disease (CLD) and 20 healthy controls. Among the cases, 46 were male (76.67%) and 14 female (23.33%), while controls consisted of 12 males (60%) and 8 females (40%). The mean age of CLD cases was 52.15 years, ranging from 19 to 85 years, with the majority falling in the 50–59 year age group, while controls had a mean age of 41.2 years and a median of 40 years. The most frequently identified etiology of CLD was alcoholic liver disease (68.33%), followed by non-alcoholic fatty liver disease (23.33%) and viral hepatitis (5%). Two cases were suspected to be autoimmune-related but remained under investigation at the time of the study.

Liver stiffness was assessed using shear wave elastography (SWE) and classified into four categories based on the 2020 Society of Radiologists in Ultrasound (SRU) guidelines. More than half of the cases (51.66%) demonstrated liver stiffness values in the F4 range ($\geq$ 17 kPa), consistent with advanced fibrosis or cirrhosis. Moderate fibrosis (F2: 9–13 kPa) was present in 20% of

Table 1: Ultrasound Doppler parameters and clinical fibrosis scores in controls and cases (mild vs significant fibrosis).

Parameter	Controls (n=20)	Mild fibrosis (F1,F2) (n=20)	Significant fibrosis (≥F3) (n=40)
SI (Mean +/- S.D)(cm^2)	42.9 +/- 12.65	57.09+/-25.27	95.51+/- 36.05
PVW (Mean +/- S.D)(mm)	8.91 +/- 2.05	11.29 +/-2.39	13.69+/- 1.69
PVV (Mean +/- S.D) (cm/sec)	23.60 +/- 2.65	19.45 +/- 4.08	13.86 +/- 2.82
SPI (Mean +/- S.D)	1.83 +/- 0.60	3.21 +/- 2.32	7.27+/- 3.47
AST/ALT Ratio (Mean +/- S.D)	0.90 +/- 0.17	1.15+/-0.48	1.28 +/- 0.57
APRI (Mean +/- S.D)	0.31 +/- 0.06	0.50+/-0.29	1.29+/- 0.70
Fib-4 Index (Mean +/- S.D)	0.99 +/- 0.53	2.24 +/-0.71	3.55+/- 1.52

Table 2: t-test for equality of means.

		t-test for Equality of Means						
		t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
							Lower	Upper
SGOT(U/L)	Equal variances assumed	-4.791	78	.000	-30.6225	6.3914	-43.3467	-17.8983
	Equal variances not assumed	-4.791	56.042	.000	-30.6225	6.3914	-43.4257	-17.8193
SGPT(U/L)	Equal variances assumed	-3.513	78	.001	-23.4375	6.6713	-36.7191	-10.1559
	Equal variances not assumed	-3.513	51.045	.001	-23.4375	6.6713	-36.8305	-10.0445
Platelets (×109/l)	Equal variances assumed	6.022	78	.000	54.575	9.062	36.533	72.617
	Equal variances not assumed	6.022	73.854	.000	54.575	9.062	36.517	72.633
FIB-4	Equal variances assumed	-6.944	78	.000	-1.93650	.27887	-2.49168	-1.38132
	Equal variances not assumed	-6.944	62.678	.000	-1.93650	.27887	-2.49383	-1.37917
Liver size(cm)	Equal variances assumed	.534	78	.595	.2050	.3841	-.5597	.9697
	Equal variances not assumed	.534	77.956	.595	.2050	.3841	-.5597	.9697
SI(cm2)	Equal variances assumed	-6.892	78	.000	-45.46600	6.59651	-58.59864	-32.33336
	Equal variances not assumed	-6.892	62.694	.000	-45.46600	6.59651	-58.64933	-32.28267
PV width(mm)	Equal variances assumed	-7.513	78	.000	-3.5950	.4785	-4.5476	-2.6424
	Equal variances not assumed	-7.513	68.357	.000	-3.5950	.4785	-4.5498	-2.6402
PVV(cm/s)	Equal variances assumed	9.916	78	.000	7.6700	.7735	6.1301	9.2099
	Equal variances not assumed	9.916	70.133	.000	7.6700	.7735	6.1274	9.2126
SPI	Equal variances assumed	-7.674	78	.000	-4.756023359672406	-.619788652628754	-5.989927782040859	-3.522118937303954
	Equal variances not assumed	-7.674	58.793	.000	-4.756023359672406	-.619788652628754	-5.996308802930734	-3.515737916414078
OTPT	Equal variances assumed	-2.382	78	.020	-.26041	.10931	-.47802	-.04280
	Equal variances not assumed	-2.382	67.916	.020	-.26041	.10931	-.47853	-.04229
APRI	Equal variances assumed	-6.506	78	.000	-.78737	.12102	-1.02830	-.54645
	Equal variances not assumed	-6.506	52.267	.000	-.78737	.12102	-1.03018	-.54456

cases, severe fibrosis (F3: 13–17 kPa) in 15%, and early-stage fibrosis (F1: <9 kPa) in 13.33% of the cohort. The predominance of advanced fibrosis in the study group underscores a concerning trend of late-stage diagnosis in CLD and highlights the utility of non-invasive imaging for earlier detection. When regrouped into mild (<13 kPa) and significant fibrosis (≥ 13 kPa), 60% of cases exhibited significant fibrosis, further emphasizing the burden of advanced disease.

Grey-scale ultrasound showed no significant correlation between absolute liver size and liver stiffness values. However, liver echotexture was significantly associated with increased stiffness, particularly in cases displaying coarse or heterogeneous echogenicity, indicating underlying fibrotic changes. Steatosis was observed in a subset of patients, but its effect on stiffness values was

inconsistent.

On Doppler evaluation, several hemodynamic alterations were observed and correlated significantly with liver stiffness measurements. Spleen index (SI), portal vein width (PVW), and splenoportal index (SPI) all demonstrated a significant positive correlation with liver stiffness values, with p-values <0.001 in each case. These findings reflect the pathophysiological development of portal hypertension in advanced CLD. Conversely, portal vein velocity (PVV) showed a significant negative correlation with liver stiffness values ($p < 0.001$), indicating that lower PVV is associated with greater fibrotic burden. The mean PVV in patients with significant fibrosis (13.86 cm/sec) was markedly reduced compared to those with mild fibrosis (19.45 cm/sec), consistent with a progressive reduction in forward flow due to increased intrahepatic resistance. Patients with significant fibrosis also had significantly elevated SI, PVW, and SPI values compared to those with mild fibrosis, further reinforcing the clinical utility of these Doppler parameters in assessing disease severity. (Table 1)

Hepatic vein waveform analysis provided additional insights into the progression of liver fibrosis. Triphasic waveforms, typically seen in healthy individuals, were associated with lower liver stiffness values (mean: 16.53 kPa). In contrast, biphasic and monophasic waveforms were associated with significantly higher stiffness values, measuring 21.77 kPa and 36.46 kPa, respectively. The correlation was statistically significant ($p < 0.001$) and suggests that hepatic vein waveform alteration may serve as an adjunct marker of fibrosis staging.

In addition to imaging parameters, the study also compared liver stiffness values with established non-invasive clinical scores: AST/ALT ratio, APRI, and FIB-4 index. All three indices were significantly higher in CLD patients than in healthy controls ($p < 0.001$ for each), and each demonstrated significantly greater values in patients with significant fibrosis compared to those with mild disease. Specifically, the mean AST/ALT ratio in CLD cases was 1.24 versus 0.90 in controls ($p = 0.020$), and 1.28 versus 1.15 when comparing significant versus mild fibrosis ($p = 0.015$). APRI values showed a strong association with fibrosis severity, averaging 1.10 in cases and 0.31 in controls ($p < 0.001$), and 1.29 in significant fibrosis versus 0.50 in mild fibrosis ($p = 0.001$). FIB-4 scores were also significantly elevated in the CLD group, with a mean of 3.11 compared to 0.99 in controls, and 3.55 versus 2.24 in significant versus mild fibrosis groups,

Table 3 : Pearson Correlation between various variables.

		APRI	PVV(cm/s)	FIB-4	OTPT	SI(cm2)	SPI
APRI	Pearson Correlation	1	-.537**	.846**	.470**	.368**	.398**
	Sig. (1-tailed)		.000	.000	.000	.000	.000
	N	80	80	80	80	80	80
PVV(cm/s)	Pearson Correlation	-.537**	1	-.546**	-.308**	-.612**	-.749**
	Sig. (1-tailed)	.000		.000	.003	.000	.000
	N	80	80	80	80	80	80
FIB-4	Pearson Correlation	.846**	-.546**	1	.620**	.360**	.382**
	Sig. (1-tailed)	.000	.000		.000	.001	.000
	N	80	80	81	80	80	80
OTPT	Pearson Correlation	.470**	-.308**	.620**	1	.165	.179
	Sig. (1-tailed)	.000	.003	.000		.072	.056
	N	80	80	80	80	80	80
SI(cm2)	Pearson Correlation	.368**	-.612**	.360**	.165	1	.956**
	Sig. (1-tailed)	.000	.000	.001	.072		.000
	N	80	80	80	80	80	80
SPI	Pearson Correlation	.398**	-.749**	.382**	.179	.956**	1
	Sig. (1-tailed)	.000	.000	.000	.056	.000	
	N	80	80	80	80	80	80

**. Correlation is significant at the 0.01 level (1-tailed).

respectively (all $p < 0.001$).

Further correlation analysis revealed a strong inverse relationship between PVV and all three non-invasive fibrosis scores: AST/ALT ratio ($p = 0.006$), APRI ($p <$

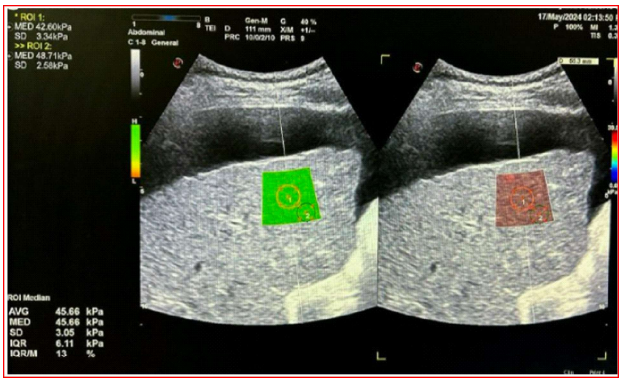

Fig 1: 2D-SWE images of a cirrhotic patient secondary to ALD ; ROI placed on right lobe of liver approx. 2-3 cm beneath the liver capsule. Median LS value obtained was 45.66 kPa(F4). Nodular liver margins, coarse echo pattern and ascites is noted in both cases.

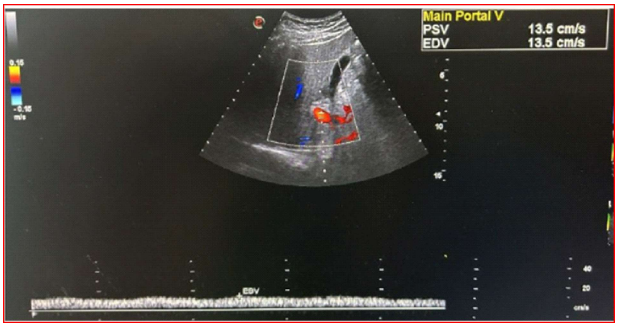

Fig 2: Color doppler ultrasound image showing reduced peak portal venous velocity (13.5 cm/sec) with reduced phasicity in a case of NAFLD.

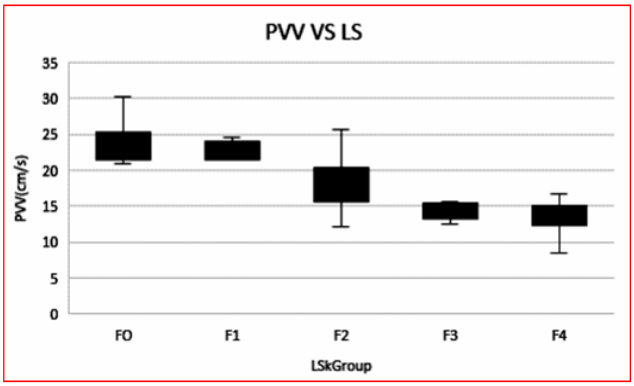

Fig 3: Box and whisker diagram showing relation between liver stiffness grades and PVV value.

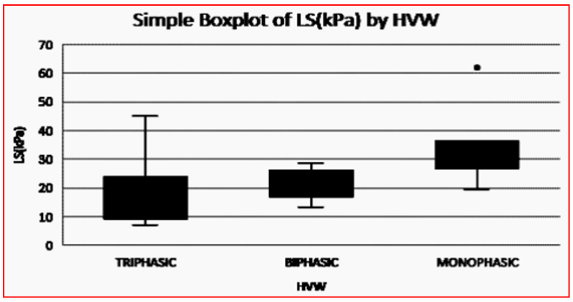

Fig 4: Box and whisker diagram showing relation between type of hepatic waveform and LS value.

0.001), and FIB-4 ($p < 0.001$). This supports the hypothesis that hemodynamic alterations measured by Doppler ultrasound may serve as practical and reproducible indicators of disease severity, potentially comparable in accuracy to established laboratory-based fibrosis indices.

Discussion

Chronic liver disease continues to impose a substantial clinical burden, with alcohol-associated liver disease and NAFLD emerging as dominant etiologies in India. In the present study, alcoholic liver disease was the most common cause of CLD, with a demographic profile consistent with prior Indian studies. A large proportion of patients demonstrated advanced fibrosis, underscoring delayed presentation and the need for reliable non-invasive assessment.

Liver stiffness measured by SWE showed strong correlation with Doppler-derived parameters reflecting portal hypertension, including spleen index, portal vein width, splenoportal index, and portal vein velocity. The inverse relationship between portal vein velocity and fibrosis severity highlights its potential utility as a simple hemodynamic marker. Alterations in hepatic vein waveform patterns were also associated with increasing stiffness, reflecting reduced hepatic compliance in advanced disease.

Non-invasive fibrosis scores (AST/ALT ratio, APRI, and FIB-4) were significantly elevated in CLD patients and correlated well with liver stiffness and Doppler parameters. These findings support the complementary role of imaging-based and laboratory-based indices in fibrosis assessment, particularly in settings where liver biopsy is not feasible.

Overall, the integration of SWE and Doppler ultrasound offers a comprehensive, reproducible, and non-invasive approach for evaluating liver fibrosis and portal hemodynamics. This combined strategy may aid in disease

stratification, monitoring, and clinical decision-making in patients with chronic liver disease.

In our study of 60 CLD patients and 20 healthy controls, alcoholic liver disease was the most common cause (68.3%), followed by NAFLD (23.3%) and viral hepatitis (5%). These findings echo recent Indian epidemiological trends showing a rise in alcohol-related liver disease, which may soon surpass viral hepatitis as the predominant cause of CLD[9,2]. The demographic profile—male predominance and mean age in the 50s—is also in concordance with prior studies[7].

Liver stiffness measurements (LSM) using SWE were categorized per the Society of Radiologists in Ultrasound (SRU) 2020 consensus. A majority of patients (51.66%) demonstrated advanced fibrosis (F4, ≥ 17 kPa), highlighting the burden of late-stage liver disease and the need for earlier detection. We also compared LSMs across a binary classification of mild (<13 kPa) and significant fibrosis (≥ 13 kPa), similar to other recent studies[10].

Interestingly, liver size showed no significant correlation with LSM, whereas altered liver echotexture demonstrated a strong association. The impact of hepatic steatosis on LS has been debated; while some authors reported no correlation[11,12], others noted potential overestimation due to steatosis[13], underscoring the complexity of accurate fibrosis quantification.

Significant positive correlations were observed between LS and spleen index (SI), portal vein width (PVW), and splenoportal index (SPI), with higher values in the significant fibrosis group. These findings are consistent with previous studies reporting that these parameters reflect portal hypertension and fibrosis severity[14,15]. Conversely, portal vein velocity (PVV) demonstrated a highly significant negative correlation with LS values ($p<0.001$), as previously reported[7,16,17]. Notably, PVV <17 cm/s has been proposed as a sensitive threshold for fibrosis prediction[7].

Alterations in hepatic vein waveforms further supported SWE findings. Patients with biphasic and monophasic waveforms had significantly higher LS values than those with triphasic patterns, reflecting increasing hepatic parenchymal stiffness. These results support earlier conclusions by Zytoon *et al*. [6] that waveform simplification correlates with fibrosis severity and impaired hepatic compliance.

In addition to imaging-based metrics, we assessed correlations between LSM and established non-invasive fibrosis scores: AST/ALT ratio, APRI, and FIB-4 index. All three indices were significantly elevated in CLD cases compared to controls and were notably higher in patients with significant fibrosis. These results validate prior findings that these indices are reliable tools for fibrosis risk stratification, particularly in resource-limited settings[8,18,19]. Furthermore, a significant inverse correlation was found between PVV and all three fibrosis scores, suggesting that Doppler ultrasound may complement biochemical scoring systems in the non-invasive assessment of fibrosis[7].

Our findings collectively support the clinical utility of combining SWE and Doppler ultrasound parameters for evaluating liver fibrosis and related hemodynamic changes. The integration of these modalities offers a comprehensive, non-invasive approach to assess disease severity, monitor progression, and potentially predict complications such as portal hypertension and variceal formation.

Limitations

This study has certain limitations. The relatively small sample size may limit generalizability. All imaging and elastography examinations were performed by a single radiologist, precluding assessment of interobserver variability. Histopathological correlation with liver biopsy was not available due to ethical and practical constraints. Additionally, Doppler measurements may have been influenced by respiratory variations and cardiac pulsations.

Despite these limitations, the study demonstrates the clinical utility of SWE and Doppler ultrasound as non-invasive tools for evaluating liver fibrosis and portal hemodynamics. Larger, multicentric studies with histological correlation and longitudinal follow-up are warranted to further validate these findings.

Conclusion

The study demonstrates a significant correlation between liver stiffness measurements obtained via SWE and both Doppler-derived hemodynamic parameters and clinical fibrosis scores. SWE values increase in parallel with portal hypertension features such as spleen enlargement, reduced PVV, and waveform abnormalities in hepatic veins. The non-invasive nature of these tools makes them particularly advantageous for routine assessment, monitoring progression, and potentially guiding management in patients with CLD. These comprehensive correlations suggest that combining elastography with Doppler ultrasound and clinical indices provides a holistic and sensitive approach to evaluating

liver fibrosis, particularly where liver biopsy is not feasible.

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Conflict of Interest: Nil

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