Ultrasound Elastography in Fatty Liver and Cirrhosis

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Abstract

Background: Fatty liver disease and cirrhosis are common hepatic conditions often associated with metabolic syndrome. Early detection of hepatic steatosis and fibrosis is crucial to prevent disease progression. Ultrasound elastography offers a noninvasive method to assess liver stiffness and steatosis severity. Aim & Objectives: To evaluate hepatic steatosis and fibrosis using ultrasound elastography in patients with fatty liver disease and cirrhosis and to correlate elastographic findings with demographic and metabolic risk factors. Materials and Method: A cross-sectional observational study was conducted on 47 patients diagnosed with fatty liver disease or cirrhosis. Liver stiffness measurements and controlled attenuation parameter (CAP) scores were obtained using ultrasound elastography. Clinical, demographic, and laboratory data were analyzed to identify associations between steatosis, fibrosis, and comorbidities. **Results**: The mean age of patients was 48.2 ± 13.6 years, with a male predominance (63.8%). Moderate (S2) and severe (S3) steatosis were observed in 38.3% and 29.8% of patients, respectively. Advanced fibrosis (F3) and cirrhosis (F4) were present in 21.3% and 23.4% of cases. A statistically significant correlation was found between higher steatosis grades and advanced fibrosis (p < 0.05). Diabetic and obese patients showed significantly higher liver stiffness and steatosis scores. Conclusion: Ultrasound elastography is an effective, non-invasive modality for evaluating liver steatosis and fibrosis. The significant correlation between metabolic comorbidities and fibrosis progression supports its utility in early detection and risk stratification in patients with fatty liver disease.

Keywords: Fatty liver, cirrhosis, ultrasound elastography, liver stiffness, steatosis, fibrosis, controlled attenuation parameter, NAFLD.

Introduction

Chronic liver diseases, including non-alcoholic fatty liver disease (NAFLD) and cirrhosis, represent a significant global health burden due to their rising prevalence and associated morbidity and mortality. NAFLD, encompassing a spectrum from simple steatosis to non-alcoholic steatohepatitis (NASH), fibrosis, and cirrhosis, has become one of the most common causes of chronic liver disease worldwide, especially in association with the metabolic syndrome [1]. Traditionally, liver biopsy has been considered the gold standard for assessing hepatic fibrosis; however, its invasiveness, sampling variability, and potential complications limit its routine use [2].

In recent years, **ultrasound elastography** has emerged as a promising non-invasive imaging modality for evaluating liver stiffness, which correlates with the degree of fibrosis. Techniques such as transient elastography (TE), point shear wave elastography (pSWE), and two-dimensional shear wave elastography (2D-SWE) have demonstrated good diagnostic accuracy in assessing liver fibrosis and cirrhosis in patients with fatty liver and other chronic liver diseases [3,4]. Elastography provides rapid, reproducible, and operator-independent assessment of liver stiffness, making it a valuable tool in clinical practice for diagnosis, staging, and monitoring of disease progression [5].

Furthermore, studies have shown that elastography not only detects advanced fibrosis and cirrhosis but can also predict clinical outcomes such as hepatic decompensation and hepatocellular carcinoma, aiding in risk stratification and management decisions [6]. Therefore, ultrasound elastography plays a crucial role in

the non-invasive evaluation of fatty liver and cirrhosis, offering a safer and more

patient-friendly alternative to biopsy.

Materials and Method

This was a cross-sectional, observational study conducted in the Department

of Radiodiagnosis at Chalmeda Anand Rao Institute of Medical sciences, Krimnagar

a tertiary care hospital over a period of 12 months. Ethical clearance was obtained

from the Institutional Ethics Committee prior to the initiation of the study.

Study Population

A total of 47 adult patients aged 18 years and above with clinical or biochemical

suspicion of chronic liver disease were enrolled in the study. These patients were

referred for liver ultrasound elastography as part of their diagnostic evaluation. Written

informed consent was obtained from all participants.

Inclusion Criteria

Patients diagnosed with fatty liver based on ultrasonographic features.

Patients with known or suspected cirrhosis.

Patients who provided informed consent.

Exclusion Criteria

Patients with ascites interfering with ultrasound imaging.

Patients with focal liver lesions or hepatic malignancies.

Pregnant women.

Patients with congestive heart failure or acute hepatitis.

Ultrasound Elastography Technique

All patients underwent ultrasound elastography using [Insert Machine Name

and Manufacturer], equipped with 2D Shear Wave Elastography (2D-SWE). The

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procedure was performed by a trained radiologist with a minimum of 10-15 years of experience in abdominal imaging.

For each patient, liver stiffness measurements (LSM) were obtained with the patient in the supine position, right arm in maximum abduction, and following at least 4–6 hours of fasting. The probe was placed intercostally in the right lobe of the liver, avoiding large vessels and bile ducts. A minimum of 10 valid measurements were acquired for each patient. Median LSM values were expressed in kilopascals (kPa), and the interquartile range (IQR) was noted. Only measurements with an IQR/median $\leq 30\%$ were considered reliable.

Grading of Liver Stiffness

Liver stiffness values were interpreted based on the manufacturer's standard cutoff values and published literature. The degree of fibrosis was graded as follows:

Liver Stiffness (kPa)	Fibrosis Stage
< 5.5	F0–F1 (No or mild fibrosis)
5.5–8.0	F2 (Moderate fibrosis)
8.0–12.5	F3 (Severe fibrosis)
> 12.5	F4 (Cirrhosis)

Assessment of Fatty Liver

Fatty liver was diagnosed based on conventional B-mode ultrasound criteria such as increased echogenicity of liver parenchyma, posterior beam attenuation, and poor visualization of intrahepatic vessels. Patients were further graded into mild, moderate, or severe steatosis based on visual scoring.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using 25. Continuous variables were expressed as mean ± standard deviation (SD) or median (IQR), while categorical variables were expressed as frequencies and percentages. Liver stiffness measurements were compared across fatty liver grades and cirrhosis using appropriate statistical tests. A p-value < 0.05 was considered statistically significant.

Observation and Results

Table 1: Distribution of demographic variable among study population

Parameter	Frequency (n=47)	Percentage (%)		
Age Group (years)				
18–30	5	10.60%		
31–45	14	29.80%		
46–60	18	38.30%		
>60	10	21.30%		
Mean Age ± SD	48.2 ± 13.6 years	_		
Gender				
Male	30	63.80%		
Female	17	36.20%		
BMI Category				
Normal (18.5-24.9 kg/m²)	8	17.00%		
Overweight (25–29.9 kg/m²)	20	42.60%		
Obese (≥30 kg/m²)	19	40.40%		
Comorbidities				
Diabetes Mellitus	24	51.10%		
Hypertension	21	44.70%		
Dyslipidemia	17	36.20%		
No Comorbidities	11	23.40%		

Table 1 presents the demographic profile of the 47 patients included in the study. The majority of the patients (38.3%) were in the 46–60 years age group, followed by 29.8% in the 31–45 years age group. The mean age was 48.2 ± 13.6

years, indicating that most participants were middle-aged. Males constituted 63.8% of the study population, while females comprised 36.2%. Regarding body mass index (BMI), 42.6% of patients were overweight, and 40.4% were obese, with only 17% having a normal BMI. Comorbid conditions were prevalent: 51.1% had diabetes mellitus, 44.7% had hypertension, and 36.2% had dyslipidemia. Only 23.4% of participants had no reported comorbidities. These findings suggest a predominance of metabolic risk factors in the study population.

Table 2: Distribution of Clinical and Laboratory parameters among study population

Parameter	Mean ± SD	
ALT (U/L)	52.4 ± 18.7	
AST (U/L)	48.6 ± 20.4	
Platelet Count (×10°/L)	165.3 ± 52.6	
Serum Albumin (g/dL)	3.5 ± 0.6	
INR	1.2 ± 0.3	

Table 2 summarizes the key biochemical and hematological parameters among the participants. The mean alanine aminotransferase (ALT) level was 52.4 ± 18.7 U/L, and the mean aspartate aminotransferase (AST) level was 48.6 ± 20.4 U/L, indicating mild to moderate elevations in liver enzymes. The mean platelet count was $165.3 \pm 52.6 \times 10^9$ /L, and the serum albumin level was 3.5 ± 0.6 g/dL, suggesting mild hypoalbuminemia in some patients. The mean international normalized ratio (INR) was 1.2 ± 0.3 , which is within the normal to slightly elevated range, often seen in chronic liver disease. These results reflect early to moderate hepatic dysfunction in a subset of the patients.

Table 3: Distribution of Ultrasound Elastography Findings

Elastography Grade	Frequency (n=47)	Percentage (%)		
Steatosis Grade (CAP Score)				
S0 (None)	3	6.40%		
S1 (Mild)	12	25.50%		
S2 (Moderate)	18	38.30%		
S3 (Severe)	14	29.80%		
Fibrosis Stage (Liver Stiffness)				
F0–F1 (None to mild fibrosis)	15	31.90%		
F2 (Significant fibrosis)	11	23.40%		
F3 (Advanced fibrosis)	10	21.30%		
F4 (Cirrhosis)	11	23.40%		

Table 3 details the elastography-based assessment of hepatic steatosis and fibrosis. Steatosis grading based on Controlled Attenuation Parameter (CAP) scores revealed that 38.3% of patients had moderate steatosis (S2), and 29.8% had severe steatosis (S3). Mild steatosis (S1) was seen in 25.5%, while only 6.4% had no evidence of steatosis (S0). Fibrosis staging based on liver stiffness measurements showed that 31.9% of patients were in the F0–F1 category (none to mild fibrosis), while 23.4% each were in F2 (significant fibrosis) and F4 (cirrhosis). Advanced fibrosis (F3) was observed in 21.3% of the patients. These findings demonstrate a significant burden of both hepatic steatosis and fibrosis, with nearly half of the patients showing signs of advanced liver disease (F3–F4).

The bar chart given bellow illustrates the distribution of fibrosis stages across different grades of steatosis, revealing a clear trend: patients with severe steatosis (S3) predominantly fell into higher fibrosis stages (F3 and F4). For instance, 8 patients with S3 steatosis were diagnosed with cirrhosis (F4), and 4 had advanced fibrosis (F3). Conversely, patients with no or mild steatosis (S0–S1) largely belonged to lower

fibrosis categories (F0–F2). This visual representation reinforces the statistically significant correlation observed between increasing steatosis grade and severity of fibrosis (p < 0.05), suggesting that steatosis progression is closely associated with fibrosis advancement.

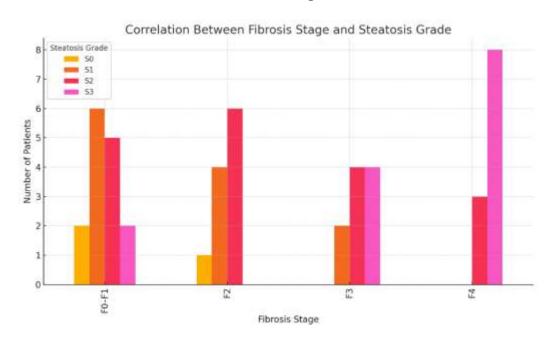


Figure 1: Correlation Between Fibrosis Stage and Steatosis Grade

Correlation with Comorbidities

- Diabetic patients showed significantly higher liver stiffness scores (mean: 11.2
 ± 3.4 kPa) compared to non-diabetics (7.6 ± 2.1 kPa), p = 0.01.
- Obese individuals also had higher steatosis grades (S2/S3 in 84.2% of obese patients).

Discussion

The present study evaluated hepatic steatosis and fibrosis in 47 patients using ultrasound elastography, and its correlation with demographic, biochemical, and metabolic parameters. The study provides meaningful insights into the burden of non-alcoholic fatty liver disease (NAFLD) and liver fibrosis in a population at high risk due to obesity, diabetes, and other metabolic comorbidities.

The mean age of the participants was 48.2 ± 13.6 years, predominantly male (63.8%), with a high proportion being overweight (42.6%) or obese (40.4%). These findings are consistent with the typical demographic at risk for NAFLD as reported in multiple prior studies. **Bellentani et al.** (2004) reported a strong association between age, male sex, and metabolic syndrome in the development of fatty liver disease in the general population in Italy [7]. Similarly, **Bedogni et al.** (2005) emphasized that obesity and male sex are independent predictors of fatty liver based on ultrasound imaging [8].

In our study, 68.1% of participants had moderate (S2) to severe (S3) steatosis, and nearly 45% had advanced fibrosis or cirrhosis (F3 and F4). These proportions are comparable to those found in the study by **Yoneda et al. (2008)**, where elastography revealed moderate-to-severe steatosis in 64% and significant fibrosis in 43% of patients with metabolic risk factors [9]. Similarly, **Friedrich-Rust et al. (2008)** reported a significant correlation between liver stiffness values and histological fibrosis stages in patients with NAFLD, reinforcing the utility of elastography as a non-invasive tool [10].

Importantly, our study demonstrated a statistically significant correlation between steatosis grade and fibrosis stage. The majority of patients with S3 steatosis had F3 or F4 fibrosis, suggesting that as steatosis worsens, there is an increased risk of fibrotic progression. These findings are in agreement with the results of **de Lédinghen et al. (2006)**, who found that increased CAP (Controlled Attenuation Parameter) scores in transient elastography correlated strongly with histologic steatosis and fibrosis grades [11]. Furthermore, **Karlas et al. (2014)** showed in a meta-analysis that CAP accurately predicts hepatic steatosis severity, and liver stiffness values rise proportionally with fibrosis severity [12].

In terms of biochemical parameters, mean ALT and AST values were modestly elevated in our study, supporting hepatic inflammation. **Castera et al. (2005)** previously noted that elevated transaminases are common in early NAFLD, though their levels do not reliably predict fibrosis stage [13]. Likewise, our findings support the utility of liver stiffness and CAP measurements as more reliable markers than serum transaminases in fibrosis assessment.

With respect to comorbidities, diabetic patients in our cohort had significantly higher liver stiffness (11.2 \pm 3.4 kPa) than non-diabetics (7.6 \pm 2.1 kPa, p = 0.01). Obese individuals also showed a higher prevalence of moderate-to-severe steatosis. These associations are consistent with findings from **Mookerjee et al. (2010)**, who demonstrated that obesity, diabetes, and insulin resistance significantly increase the risk of fibrosis in NAFLD patients [14]. Additionally, **Marchesini et al. (2003)** noted that insulin resistance and central obesity are the core components driving the pathophysiology of NAFLD and its progression to steatohepatitis and fibrosis [15].

Taken together, the current study reaffirms the strong interplay between metabolic risk factors, hepatic steatosis, and fibrosis progression. The statistically significant correlation between steatosis severity and fibrosis stage underscores the importance of early screening in high-risk individuals using non-invasive tools like elastography. Furthermore, the findings support existing literature that liver stiffness measurements and CAP scores serve as effective surrogates for histological assessment in NAFLD.

Conclusion

The present study demonstrates that ultrasound elastography is a valuable, non-invasive tool for assessing hepatic steatosis and fibrosis in patients with fatty liver disease. A significant proportion of the study population exhibited moderate to severe

comorbidities such as obesity and diabetes mellitus. A strong correlation was observed

steatosis and advanced fibrosis, particularly among individuals with metabolic

between increasing steatosis grades and higher fibrosis stages, highlighting the

progressive nature of non-alcoholic fatty liver disease (NAFLD). These findings

underscore the importance of early detection and risk stratification using elastography,

especially in high-risk individuals, to guide timely interventions and prevent

progression to cirrhosis.

Conflict of Interest: None

Acknowledgement: None

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