

Correlation of Serum Alanine Aminotransferase Levels with Hepatic Fibrosis and Steatosis Score Assessed by FibroScan and Controlled Attenuation Parameter (CAP) in Patients with Nonalcoholic Fatty Liver Disease

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ABSTRACT

Background: Nonalcoholic fatty liver disease (NAFLD) is an increasingly prevalent cause of chronic liver disease worldwide, affecting approximately one-third of adults in Bangladesh. Serum alanine aminotransferase (ALT) is routinely used as a biochemical marker of hepatocellular injury; however, its ability to accurately reflect the severity of hepatic steatosis and fibrosis remains uncertain. FibroScan-derived liver stiffness measurement (LSM) and controlled attenuation parameter (CAP) offer noninvasive approaches for assessing hepatic fibrosis and steatosis, respectively, and may provide more direct measures of disease severity.

Objective: This study aimed to determine the correlation between serum ALT levels and hepatic fibrosis and steatosis severity measured by FibroScan and CAP in patients with NAFLD.

Methods: This observational study included 97 patients with ultrasonographic evidence of fatty liver disease who attended the Hepatology outpatient department of MH Samorita Hospital and Medical College, Dhaka, Bangladesh between April 2026 to June 2026. The mean age was 40.6 ± 11.3 years, and 52.6% were female. Liver FibroScan with CAP was performed after at least four hours of fasting. Fibrosis was staged according to METAVIR criteria, while steatosis was assessed using CAP. Serum ALT was categorized as normal (≤ 40 U/L) or elevated (>40 U/L). Spearman rank correlation and Mann-Whitney U tests were used for statistical analysis.

Results: Using an upper normal limit of 40 U/L for ALT, 39 patients (40.2%) had normal ALT levels, while 58 (59.8%) had elevated levels. Common comorbidities included diabetes mellitus (54.7%), hypertriglyceridemia (60.4%), and hypertension (38.0%). The mean liver stiffness measurement was 6.48 ± 3.16 kPa, with the majority of patients (86.6%) classified as having F0-F1 fibrosis. Fibrosis stages F2, F3, and F4 were observed in 2.1%, 5.2%, and 6.2% of patients, respectively. The mean controlled attenuation parameter (CAP) was 302.2 ± 39.5 dB/m. Severe steatosis (S3) was present in 49.5% of patients, while significant steatosis (S2-S3) was identified in 83.5%. The correlation between ALT levels and liver stiffness was weak and did not reach statistical significance (Spearman's $r = 0.191$, $p = 0.061$).

Conclusion: In this cohort of patients with NAFLD, serum ALT levels showed only a weak correlation with the severity of hepatic fibrosis, suggesting that ALT alone may not accurately reflect the extent of fibrotic changes. In contrast, FibroScan and CAP may provide more objective and reliable noninvasive methods for evaluating hepatic fibrosis and steatosis in patients with NAFLD.

Keywords- Nonalcoholic fatty liver disease; Fibrosis; Steatosis; FibroScan; Controlled attenuation parameter; Alanine aminotransferase.

I. INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is closely associated with metabolic syndrome and affects approximately 25-30% of adults globally, with substantially higher prevalence among individuals with obesity and type 2 diabetes. In Asia, prevalence is estimated at 15-30% in the general population and exceeds 50% among patients with diabetes or metabolic syndrome. In Bangladesh, a substantial proportion of patients with NAFLD may have non-alcoholic steatohepatitis (NASH), suggesting important population-specific characteristics^{1,2,3,4}.

Disease prognosis is primarily determined by the severity of hepatic fibrosis. NASH is the progressive form of NAFLD and is associated with greater morbidity, mortality, and fibrosis progression than simple steatosis. Although serum alanine aminotransferase (ALT) is commonly used to assess hepatocellular injury, normal ALT levels do not exclude significant NAFLD, NASH, or advanced fibrosis. Therefore, reliance on ALT alone may underestimate disease severity^{5,6}.

Liver biopsy remains the reference standard for evaluating steatohepatitis and hepatic fibrosis; however, its invasive nature, sampling variability, and potential procedural risks limit its clinical utility. FibroScan offers a non-invasive alternative by assessing liver stiffness as a surrogate marker of fibrosis and measuring the controlled attenuation parameter (CAP) to quantify hepatic steatosis⁷.

Although previous studies have demonstrated associations between ALT levels and advanced fibrosis, evidence from Bangladesh is limited⁴. Findings from other Asian populations may not be directly applicable because of differences in genetic, dietary, and metabolic characteristics. Therefore, the present study aims to evaluate the relationship between serum ALT levels and FibroScan-derived liver stiffness and CAP among Bangladeshi patients with NAFLD, and to determine whether ALT can adequately reflect the severity of hepatic fibrosis and steatosis in this population.

II. MATERIALS AND METHODS

Study design and setting

This was a cross-sectional observational study conducted at the Department of Hepatology, MH Samorita Hospital and Medical College (MHSHMC), Tejgaon, Dhaka, Bangladesh. Data collection was performed from April 2026 to June 2026.

Study population

Consecutive patients attending the Hepatology outpatient department (OPD) of MHSHMC with

ultrasonographic evidence of fatty liver disease were screened for enrollment.

➤ Inclusion criteria

- Age 18 to 65 years.
- Ultrasonographic evidence of fatty liver of any grade.
- Willing to provide informed written consent.

➤ Exclusion criteria

- Significant alcohol consumption (>30 g/day in males, >20 g/day in females).
- Hepatotoxic drugs use (tamoxifen, valproic acid, amiodarone, methotrexate, or corticosteroids etc.)
- Chronic liver disease from other etiology; Chronic HBV infection, chronic HCV infection, Wilson's disease, hemochromatosis or drug induced liver injury etc.
- Pregnancy & Liver failure.

Sample size

The sample size was calculated based on power analysis for a single proportion, hypothesizing 85% sensitivity of clinical scores to assess NAFLD activity, with a power of 80% and a two-sided significance level of 5% (based on Pulzi et al., 2011)⁸. This yielded a minimum of 56 patients, rounded up to 100 to account for exclusions. A total of 97 patients fulfilled inclusion criteria and were enrolled.

Clinical Evaluation

All participants underwent a standardized clinical history and physical examination. Blood pressure was measured using a mercury sphygmomanometer after at least 5 minutes of rest, and both systolic and diastolic blood pressure were recorded in mmHg. Body weight (kg) and height (cm) were measured, and body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m²).

Obesity status was categorized according to the Asian-specific criteria of the Western Pacific Regional Office of the World Health Organization (WPRO 2000), with BMI classifications of normal weight (18.5-22.9 kg/m²), overweight (23.0-24.9 kg/m²), and obese (≥25.0 kg/m²)^{9,10}. Waist circumference was measured in centimeters at the midpoint between the lower costal margin and the iliac crest.

Metabolic syndrome was defined using the modified Asian criteria as the presence of at least three of the following five components: (1) waist circumference ≥90 cm in men or ≥80 cm in women; (2) serum triglyceride concentration ≥150 mg/dL; (3) HDL-cholesterol concentration <40 mg/dL in men or <50 mg/dL in women; (4) systolic blood pressure ≥130 mmHg and/or diastolic blood pressure ≥85 mmHg, or current use

of antihypertensive medication; and (5) fasting plasma glucose ≥ 100 mg/dL (5.6 mmol/L) and/or current use of antidiabetic medication.¹⁰

Laboratory investigations

Following an overnight fast of at least 8 hours, venous blood samples were collected to assess fasting plasma glucose, 2-hour postprandial glucose, total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, triglycerides, alanine aminotransferase (ALT), platelet count, hepatitis B surface antigen, anti-hepatitis C virus antibodies, and thyroid-stimulating hormone (TSH). All biochemical analyses were performed using an automated platform in the hospital biochemistry laboratory. An ALT level of ≤ 40 U/L was considered within the normal range, whereas a level >40 U/L was classified as elevated, based on the conventional upper reference limit used locally¹¹. Due to financial constraints, not all investigations could be performed in every patient.

Abdominal ultrasonography

Abdominal ultrasonography was performed in the Department of Radiology and Imaging by a radiologist who was blinded to the laboratory findings. Hepatic steatosis was diagnosed when at least two of the following three ultrasonographic features were present: 1. increased hepatic echogenicity relative to the kidney or spleen; 2. blurring of the portal vein walls; and 3. attenuation of the ultrasound beam in the deeper hepatic tissues. Ultrasonography can detect hepatic steatosis involving approximately 20-30% or more of the liver, with reported sensitivity and specificity of approximately 84% and 94%, respectively¹².

FibroScan Examination

FibroScan examination (Echosens FibroScan 502 Touch, Paris, France) was performed by a trained and certified operator after a minimum fasting period of 4 hours. Patients were positioned supine with the right arm fully abducted. A minimum of 10 valid liver stiffness measurements (LSMs) were obtained by scanning the right hepatic lobe through an intercostal space. A median interquartile range-to-median ratio (IQR/M) of $<30\%$ was required for a reliable measurement. The M probe was used as the default probe, while the XL probe was used in patients with a body mass index (BMI) >28 kg/m² or a skin-to-liver capsule distance >25 mm, in accordance with the manufacturer's recommendations¹³.

Hepatic fibrosis was staged according to liver stiffness measurements (kPa) using the Metavir classification: F0-F1 (<7.8 kPa), indicating no or mild fibrosis; F2 (7.8-8.8 kPa), indicating moderate fibrosis with few septa; F3 (8.8-11.6 kPa), indicating severe fibrosis; and F4 (>11.6 kPa), indicating cirrhosis¹³.

Controlled Attenuation Parameter (CAP) is a non-invasive, ultrasound-based technique used to quantify hepatic fat content independently of the presence or severity of liver fibrosis. CAP measures the attenuation of ultrasound waves, defined as the progressive reduction in wave amplitude and intensity as the waves propagate

through tissue, and compares this attenuation with that observed in normal liver tissue¹⁴. CAP can be routinely measured using FibroScan probes and has demonstrated good diagnostic accuracy for detecting and grading hepatic steatosis when compared with liver biopsy findings¹⁴.

CAP values typically range from approximately 100 to 400 dB/m. Hepatic steatosis is generally defined as a median CAP value of ≥ 235 dB/m. Similar to histological grading by liver biopsy, CAP measurements can be used to categorize steatosis into four grades: absent (S0), mild (S1), moderate (S2), and severe (S3). Commonly used thresholds are <235 dB/m for S0, 235-267 dB/m for S1, 268-301 dB/m for S2, and ≥ 302 dB/m for S3¹⁵.

Ethical Considerations

Written informed consent was obtained from all participants prior to enrollment. Participant anonymity and confidentiality were strictly maintained throughout the study. The study was approved by the Institutional Review Board of M H Samorita Hospital and Medical College, Dhaka, Bangladesh. The study was conducted in accordance with the Declaration of Helsinki.

Statistical analysis

Statistical analyses were performed in Python 3.12 using the pandas, NumPy and SciPy libraries. Data distribution was assessed using the Shapiro-Wilk test. Non-normally distributed variables were compared using the Mann-Whitney U test, and categorical variables using the chi-square test, or Fisher's exact test where expected cell counts were below five. Correlations were assessed using Spearman's rank correlation. A two-sided $p < 0.05$ was considered statistically significant.

III. RESULTS

A total of 97 patients with ultrasonographically confirmed NAFLD were enrolled. The mean age was 40.6 ± 11.3 years (range 19-64 years). The cohort was predominantly female ($n = 51$, 52.6%) with males accounting for 47.4% ($n = 46$). The largest occupational group was housewives ($n = 43$, 44.3%), followed by service employees ($n = 17$, 17.5%), and business persons ($n = 14$, 14.4%). Baseline demographic and clinical characteristics are presented in Table 1.

BMI was available for 91 patients (mean 28.9 ± 4.0 kg/m²). Applying the WHO Western Pacific Regional Office (WPRO) criteria for Asian populations, 81 patients (89.0%) were classified as obese (BMI ≥ 25 kg/m²), 6 (6.6%) as overweight (BMI 23.0-24.9 kg/m²), and 4 (4.4%) as having a normal BMI (18.5-22.9 kg/m²)^{16,17}. Diabetes mellitus was present in 47 of 86 patients with the field completed (54.7%), hypertension in 30 of 79 (38.0%), and hypothyroidism in 16 of 77 (20.8%), reflecting a high metabolic comorbidity burden characteristic of this NAFLD population.

Table 1. Baseline demographic and clinical characteristics (n = 97)

Characteristic	Value	Minimum	Maximum
Age (years), mean $\pm$ SD*	40.6 $\pm$ 11.3	19	64
Sex: Female, n (%)	51 (52.6%)	-	-
Sex: Male, n (%)	46 (47.4%)	-	-
BMI (kg/m ²), mean $\pm$ SD †	28.9 $\pm$ 4.0	20.5	44.0
Obese ($\geq$ 25.0 kg/m ²)	81/91 (89.0%)	-	-
Overweight (23.0-24.9 kg/m ²)	6/91 (6.6%)	-	-
Normal (18.5-22.9 kg/m ²)	4/91 (4.4%)	-	-
Comorbidities			
Diabetes mellitus, n (%)	47/86 (54.7%)	-	-
Hypertension, n (%)	30/79 (38.0%)	-	-
Hypothyroidism, n (%)	16/77 (20.8%)	-	-
Occupation			
Housewife	43 (44.3%)	-	-
Service / employed	17 (17.5%)	-	-
Business	14 (14.4%)	-	-
Student	4 (4.1%)	-	-
Other / not recorded	19 (19.6%)	-	-
Blood pressure			
Systolic BP (mmHg)	93	119.9 $\pm$ 13.7	120
Diastolic BP (mmHg)	93	77.5 $\pm$ 10.0	80
			90-150
			60-110

* Mean age computed from 95 patients due to lack of data. † BMI computed from height and weight entries (n = 91 with complete data); Asian WPRO 2000 cut-offs applied. Comorbidity fields were incomplete on some proformas, so percentages use the number of patients with the field completed as the denominator. BMI = body mass index; SD = standard deviation.

Biochemical parameters

Serum ALT was measured in all 97 patients (100.0%). The mean ALT was 61.1 $\pm$ 56.0 U/L, with a median of 46.0 U/L (range 13-422 U/L), reflecting the characteristically right-skewed distribution of ALT in NAFLD populations. Applying the upper normal limit of 40 U/L, 39 patients (40.2%) had normal ALT and 58

(59.8%) had elevated ALT. Dyslipidemia was highly prevalent. Mean total cholesterol was 204.8 $\pm$ 46.4 mg/dL, mean LDL was 123.0 $\pm$ 40.8 mg/dL, and mean HDL was 43.5 $\pm$ 8.2 mg/dL. Hypertriglyceridemia ($\geq$ 150 mg/dL) was present in 58 of 96 tested patients (60.4%), with a mean TG of 235.8 $\pm$ 167.1 mg/dL. Detailed biochemical parameters are presented in Table 2.

Table 2. Biochemical parameters

Parameter	N	Mean $\pm$ SD	Median	Range
Glycaemic parameters				
FBS (mmol/L)	93	7.1 $\pm$ 2.9 *	5.8	3.5-18.1
2h post-breakfast plasma glucose (mmol/L)	83	9.3 $\pm$ 4.7	7.0	1.0-27.6
Liver enzymes				
ALT / SGPT (U/L)	97	61.1 $\pm$ 56.0	46.0	13-422
Normal ALT $\leq$ 40 U/L, n (%)	39 (40.2%)	-	-	-
Elevated ALT $>$ 40 U/L, n (%)	58 (59.8%)	-	-	-
Lipid profile				
Total cholesterol (mg/dL)	94	204.8 $\pm$ 46.4	200.5	117-363
LDL-cholesterol (mg/dL)	90	123.0 $\pm$ 40.8	118.5	52-278
HDL-cholesterol (mg/dL)	95	43.5 $\pm$ 8.2	44.0	24-69
Triglycerides (mg/dL)	96	235.8 $\pm$ 167.1	183.5	66-938
Hypertriglyceridemia $\geq$ 150 mg/dL, n (%)	58/96 (60.4%)	-	-	-

* One implausible fasting glucose value (639 mmol/L) and one implausible total cholesterol value (21 mg/dL) were treated as missing. FBS = fasting blood sugar; ALT = alanine aminotransferase; LDL = low-density lipoprotein; HDL = high-density lipoprotein.

FibroScan findings: fibrosis stage and steatosis grade

FibroScan was successfully performed in all 97 patients, yielding a technical success rate of 100.0%. The mean liver stiffness measurement (LSM) was 6.48 ± 3.16 kPa, with a median of 5.80 kPa and a range of 2.5-24.0 kPa. The majority of patients had minimal or no fibrosis, with 84 (86.6%) classified as F0-F1. Advanced fibrosis (F3-F4) was identified in 11 patients (11.3%), including 5 (5.2%) with F3 fibrosis and 6 (6.2%) with F4 fibrosis (cirrhosis).

Controlled attenuation parameter (CAP) measurements were available for all 97 patients (100.0%). The mean CAP was 302.2 ± 39.5 dB/m, with a median of 301.0 dB/m and a range of 189-400 dB/m. Severe steatosis (S3; ≥ 302 dB/m) was observed in 48 patients (49.5%), while 33 (34.0%) had moderate steatosis (S2), 10 (10.3%) had mild steatosis (S1), and 6 (6.2%) had no steatosis (S0). Overall, 81 patients (83.5%) had significant steatosis (S2-S3). These findings are summarized in Table 3.

Table 3. FibroScan liver stiffness and CAP steatosis results (n = 97)

Category	N	%	Reference (kPa or dB/m)
Liver stiffness measurement (kPa) — Mean 6.48 ± 3.16	97		
F0-F1 - No/mild fibrosis	84	86.6%	≤ 7.8 kPa
F2 - Moderate fibrosis	2	2.1%	7.8-8.8 kPa
F3 - Severe fibrosis	5	5.2%	8.8-11.6 kPa
F4 - Cirrhosis	6	6.2%	> 11.6 kPa
Advanced fibrosis (F3-F4)	11	11.3%	
CAP (dB/m) - Mean 302.2 ± 39.5	97		
S0 - No steatosis ($< 5\%$)	6	6.2%	< 235 dB/m
S1 - Mild steatosis (5-33%)	10	10.3%	235-267 dB/m
S2 - Moderate steatosis (34-66%)	33	34.0%	268-301 dB/m
S3 - Severe steatosis ($> 67\%$)	48	49.5%	≥ 302 dB/m
Significant steatosis (S2-S3)	81	83.5%	

kPa = kilopascals; dB/m = decibels per meter; CAP = controlled attenuation parameter; METAVIR staging system used for fibrosis.

Comparison of Fibrosis and Steatosis by ALT Group

Patients were stratified into two groups according to ALT levels: normal ALT (≤ 40 U/L; n = 39) and elevated ALT (> 40 U/L; n = 58). Mean liver stiffness measurement (LSM) was 5.97 ± 3.27 kPa in the normal-ALT group and 6.82 ± 3.07 kPa in the elevated-ALT group. Although LSM was numerically higher in patients with elevated ALT, the difference was not statistically significant (Mann-Whitney U = 890.0, p = 0.077). However, advanced fibrosis (F3-F4) was more frequently observed in the elevated-ALT group, affecting 10 patients (17.2%) compared with 1 patient (2.6%) in the normal-ALT group. This represented an approximately six-fold higher prevalence of advanced fibrosis in the elevated-

ALT group, and the difference reached statistical significance on Fisher's exact test (p = 0.046).

Mean controlled attenuation parameter (CAP) values were similar between the two groups, at 303.3 ± 43.4 dB/m in the normal-ALT group and 301.5 ± 36.9 dB/m in the elevated-ALT group. There was no statistically significant difference in CAP values (Mann-Whitney U = 1162.5, p = 0.820) or in the distribution of steatosis grades (p = 0.922). Severe steatosis (S3) was present in 51.3% of patients with normal ALT and 48.3% of those with elevated ALT, indicating a broadly similar distribution of steatosis severity across ALT groups. The findings are summarized in Table 4.

Table 4. Comparison of fibrosis and steatosis by ALT group

Parameter	Normal ALT ≤ 40 U/L (n = 39)	Elevated ALT > 40 U/L (n = 58)	p-value
Liver stiffness (kPa)			
Mean $\pm$ SD	5.97 ± 3.27	6.82 ± 3.07	0.077 §
Median	5.30	6.05	
F0-F1, n (%)	37 (94.9%)	47 (81.0%)	
F2, n (%)	1 (2.6%)	1 (1.7%)	
F3, n (%)	0 (0.0%)	5 (8.6%)	
F4, n (%)	1 (2.6%)	5 (8.6%)	
Advanced fibrosis F3-F4, n (%)	1 (2.6%)	10 (17.2%)	0.046 ¶

Parameter	Normal ALT ≤ 40 U/L (n = 39)	Elevated ALT > 40 U/L (n = 58)	p-value
CAP steatosis (dB/m)			
Mean $\pm$ SD	303.3 $\pm$ 43.4	301.5 $\pm$ 36.9	0.820 §
S0, n (%)	3 (7.7%)	3 (5.2%)	
S1, n (%)	4 (10.3%)	6 (10.3%)	
S2, n (%)	12 (30.8%)	21 (36.2%)	
S3, n (%)	20 (51.3%)	28 (48.3%)	
Significant steatosis S2-S3, n (%)	32 (82.1%)	49 (84.5%)	0.785 ¶

§ Mann-Whitney U-test. ¶ Fisher's exact test, used in place of the chi-square test because expected cell counts were below five. Values expressed as mean $\pm$ SD or n (%). ALT = alanine aminotransferase; kPa = kilopascals; dB/m = decibels per metre; CAP = controlled attenuation parameter.

Correlation analysis

Spearman rank correlation analysis between serum ALT and liver stiffness yielded a weak positive but statistically non-significant coefficient ($r_s = 0.191$, $p =$

0.061, $n = 97$). Virtually no correlation existed between ALT and CAP ($r_s = 0.003$, $p = 0.974$, $n = 97$). Results are shown in Table 5.

Table 5. Spearman rank correlation between ALT, liver stiffness, and CAP

Correlation pair	N	Spearman r_s	p-value	Significance
ALT vs liver stiffness (kPa)	97	0.191	0.061	ns
ALT vs CAP (dB/m)	97	0.003	0.974	ns

ns = not statistically significant ($p > 0.05$). ALT = alanine aminotransferase; kPa = kilopascals; dB/m = decibels per meter; CAP = controlled attenuation parameter.

IV. DISCUSSION

This observational study evaluated the relationship between serum alanine aminotransferase (ALT) levels and FibroScan-derived liver stiffness and controlled attenuation parameter (CAP) among 97 Bangladeshi patients with non-alcoholic fatty liver disease (NAFLD). The principal finding was that serum ALT showed no significant association with hepatic steatosis and only a weak, non-significant association with liver stiffness. Notably, 40.2% had normal ALT, and 82.1% of those still had significant steatosis, indicating that ALT alone is an unreliable marker of NAFLD severity. These findings support the clinical utility of FibroScan as a non-invasive tool for assessing hepatic steatosis and fibrosis.¹⁸

There was virtually no correlation between ALT and CAP ($r_s = 0.003$, $p = 0.974$). Mean CAP values were almost identical among patients with normal and elevated ALT levels (303.3 vs. 301.5 dB/m, $p = 0.820$), and the prevalence of severe steatosis (S3) was also comparable between the two groups. These findings suggest that serum ALT does not accurately reflect the extent of hepatic fat accumulation. This observation is consistent with the biological understanding that hepatic steatosis does not necessarily result in hepatocellular injury or increased ALT release. Rather, ALT elevation is more closely associated with hepatocellular inflammation and injury.^{19,20}

The proportion of patients with normal ALT levels in the present cohort was higher than that reported by Ma et al. (2020),²¹ who found normal ALT levels in

approximately 25% of patients with NAFLD and 19% of those with NASH. The relatively high proportion of patients with normal ALT in the present study may partly be explained by the predominantly early stage of fibrosis, as 86.6% of participants were classified as F0-F1. Nevertheless, this finding has important clinical implications, as patients with substantial hepatic steatosis may remain undetected when ALT is used as the sole screening marker.

ALT showed a weak positive association with liver stiffness ($r_s = 0.191$, $p = 0.061$), although this relationship did not reach statistical significance. Despite the absence of a significant linear correlation, advanced fibrosis was significantly more common among patients with elevated ALT than among those with normal ALT levels (17.2% vs. 2.6%; Fisher's exact $p = 0.046$). This suggests that while ALT is a poor continuous predictor of fibrosis severity, an elevated value does flag a subgroup at higher risk of advanced disease. The small number of patients with advanced fibrosis ($n = 11$) limits the precision of this estimate, and larger prospective studies are required to confirm the association and define clinically useful ALT thresholds.²²

The study population also demonstrated a substantial burden of metabolic risk factors. Diabetes mellitus was present in 54.7% of patients, hypertriglyceridemia in 60.4%, and obesity, according to Asian BMI criteria, in 89.0%. Severe hepatic steatosis (S3) was identified in 49.5% of patients, while 83.5% had moderate-to-severe steatosis (S2-S3). These findings underscore the close relationship between NAFLD and metabolic dysfunction and are consistent with established

evidence linking NAFLD with obesity, diabetes, dyslipidemia, and metabolic syndrome.^{6,7,23} They also support the role of non-invasive liver assessment in individuals with metabolic risk factors, particularly those with diabetes or obesity, as recommended by contemporary clinical practice guidelines.^{6,7} Importantly, normal aminotransferase levels do not exclude clinically significant NAFLD or advanced liver disease, emphasizing the limitations of relying on ALT alone for disease detection and risk assessment.^{6,7}

Female participants constituted 52.6% of the study population, which is broadly consistent with findings from some previous studies conducted in Bangladesh⁴. The relatively high proportion of housewives may be associated with differences in physical activity patterns, while age-related hormonal and dietary factors may also contribute to the observed sex distribution.^{3,4} However, these potential explanations should be interpreted cautiously because physical activity, hormonal status, and dietary factors were not directly assessed in the present study.

CAP provided additional information regarding the severity of hepatic steatosis beyond that obtained through conventional ultrasonography. Although ultrasonography is useful for detecting moderate hepatic steatosis, its ability to accurately differentiate between varying degrees of steatosis is limited. CAP may therefore provide a useful non-invasive approach for quantifying and stratifying hepatic fat accumulation.²⁴

Several limitations of this study should be acknowledged. First, its cross-sectional design prevents assessment of temporal relationships, disease progression, or causal associations between ALT levels and hepatic steatosis or fibrosis. Second, liver biopsy was not performed; therefore, histological confirmation and direct grading of steatosis, steatohepatitis, and fibrosis were not available. Liver biopsy remains the reference method for histological characterization of metabolic dysfunction-associated steatotic liver disease (MASLD).²⁵

In conclusion, the findings of this study indicate that serum ALT does not reliably reflect the severity of hepatic steatosis and has only a weak, statistically non-significant linear association with liver stiffness in this Bangladeshi cohort with NAFLD. A substantial proportion of patients with significant hepatic steatosis had normal ALT levels, demonstrating that normal ALT should not be interpreted as evidence of the absence of clinically relevant NAFLD. An elevated ALT nevertheless identified a subgroup with a significantly higher prevalence of advanced fibrosis. FibroScan, including controlled attenuation parameter (CAP), provides valuable non-invasive information regarding hepatic steatosis and liver stiffness and may be particularly useful for risk assessment in patients with metabolic risk factors, including those with normal ALT levels.^{7,25,26}

LIST OF ABBREVIATIONS

ALT: alanine aminotransferase; BMI: body mass index; CAP: controlled attenuation parameter; dB/m: decibels per meter; HDL: high-density lipoprotein; IQR: interquartile range; kPa: kilopascals; LDL: low-density lipoprotein; MSHMC: M H Samorita Hospital and Medical College; NAFLD: nonalcoholic fatty liver disease; NASH: nonalcoholic steatohepatitis; SD: standard deviation.

Conflict of interests

The authors declare no conflict of interests.

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