BIOCHEMICAL DIFFERENTIATION OF ALCOHOLIC AND NON-ALCOHOLIC FATTY LIVER DISEASE: A COMPARATIVE STUDY OF LIPID PROFILE AND DE RITIS RATIO IN ULTRASONOGRAPHY-DIAGNOSED PATIENTS

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ABSTRACT

Fatty liver disease encompasses two clinically overlapping but pathophysiologically distinct entities: alcoholic fatty liver disease (AFLD) and non-alcoholic fatty liver disease (NAFLD). While abdominal ultrasonography remains the standard first-line imaging modality, it cannot differentiate between these two conditions, making non-invasive biochemical markers essential - particularly in resource-limited settings where liver biopsy and advanced elastography are often inaccessible. This comparative cross-sectional study enrolled 146 adult patients (≥18 years) with ultrasonography-confirmed fatty liver disease at Nepal Medical College and Teaching Hospital between July and December 2025; 59 (40.4%) were classified as AFLD and 87 (59.6%) as NAFLD. Serum AST, ALT, total cholesterol (TC), triglycerides (TG), HDL, and LDL were measured, and the De Ritis ratio (AST/ALT) was calculated. The Shapiro-Wilk test confirmed non-normal distribution for most variables; the Kruskal-Wallis and Mann-Whitney U tests were used for group comparisons, with multivariable logistic regression to identify independent predictors. The De Ritis ratio showed a significant bivariate association with AFLD (χ^2 =5.78, p =0.016; Cramér's V =0.199) but did not retain independent significance on multivariable analysis (adjusted OR=1.30, 95% CI: 0.55-3.05, p =0.545). Total cholesterol (adjusted OR =1.05, 95% CI: 1.03-1.06, p <0.001) and triglycerides (adjusted OR =0.96, 95% CI: 0.92-0.99, p =0.012) were the key independent discriminators. ROC analysis confirmed excellent diagnostic accuracy for TC (AUC =1.000) and LDL (AUC =0.808) in identifying AFLD. Lipid profiling, combined with clinical history and ultrasonography, offers a practical and cost-effective non-invasive approach to differentiating AFLD from NAFLD in resource-constrained clinical settings.

KEYWORDS

NAFLD, AFLD, De Ritis ratio, lipid profile, ultrasonography, fatty liver disease, AST/ALT ratio

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INTRODUCTION

Fatty liver disease has emerged as one of the foremost contributors to chronic liver disease globally, encompassing two principal forms: non-alcoholic fatty liver disease (NAFLD) and alcoholic fatty liver disease (AFLD). NAFLD, now increasingly reclassified as metabolic dysfunction-associated fatty liver disease (MAFLD), affects approximately 25-30% of the world's adult population and is strongly linked to the twin epidemics of obesity and type 2 diabetes.[1,2] AFLD, driven by sustained alcohol misuse, continues to impose a disproportionate burden in high-consumption regions and accounts for a significant proportion of cirrhosis and hepatocellular carcinoma diagnoses.[3]

Despite their distinct aetiologies, AFLD and NAFLD share remarkably similar histological appearances, clinical presentations, and disease trajectories progressing from simple steatosis through steatohepatitis, fibrosis, cirrhosis, and potentially hepatocellular carcinoma.[4] Abdominal ultrasonography remains the most widely used first-line imaging modality for fatty liver detection, offering 85.0% sensitivity and 94.0% specificity for moderate-to-severe steatosis.[5] However, ultrasonography cannot reliably distinguish alcoholic from non-alcoholic aetiology, nor can it accurately stage fibrosis without supplemental techniques.[6] Liver biopsy, the gold standard for definitive diagnosis and staging, is invasive and costly making it impractical for routine clinical use - particularly in resource-limited healthcare settings.[7]

Two readily available biochemical parameters - the De Ritis ratio (AST/ALT) and serum lipid profile have long been investigated as non-invasive adjuncts for differentiating liver pathologies. The De Ritis ratio, first described in 1957, has been classically associated with alcoholic liver disease when exceeding 2.0, attributed to mitochondrial injury from acetaldehyde preferentially elevating AST.[8,9] Conversely, NAFLD patients typically exhibit ratios below 1.0, reflecting disproportionate ALT elevation driven by insulin resistance and hepatic steatosis.[10] With regard to lipid profiles, NAFLD is characterised by elevated triglycerides, reduced HDL cholesterol, and elevated LDL - the hallmarks of metabolic dyslipidaemia while AFLD demonstrates more variable and complex lipid patterns reflecting alcohol's dual effects on hepatic lipid synthesis and degradation.[11,12]

Despite the clinical utility of these markers, relatively few studies have systematically compared lipid profiles and the De Ritis ratio. Accurate and non-invasive differentiation of AFLD from NAFLD has direct implications for treatment strategy, counselling, and prognosis.[13] The present study, therefore, aims to compare lipid profile parameters and the De Ritis ratio between patients with ultrasonography diagnosed fatty liver and to evaluate their utility as non-invasive discriminatory markers.

MATERIALS AND METHODS

A comparative cross-sectional study was conducted at Nepal Medical College and Teaching Hospital (NMCTH), Kathmandu, Nepal, from July to December 2025. Ethical approval was obtained from the Nepal Medical College Institutional Review Committee (NMC-IRC). Adult patients (≥18 years) with ultrasonography-confirmed fatty liver disease presenting to the outpatient and inpatient departments of NMCTH were eligible for inclusion. Fatty liver was graded on ultrasonography as Grade 1 (mild), Grade 2 (moderate), or Grade 3 (severe) based on standard echogenicity criteria.[14] Venous blood samples were collected after an overnight fast of at least 8 hours. The following biochemical parameters were measured using standard laboratory methods: serum alanine aminotransferase (ALT), aspartate aminotransferase (AST), total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL) cholesterol, and low-density lipoprotein (LDL) cholesterol. The De Ritis ratio was calculated as the AST/ALT ratioand was subsequently dichotomized using a clinical threshold of ≥2 versus <2 for bivariate analysis. The TG/HDL ratio was also derived as an additional atherogenic index.

Patients were classified into two groups:

AFLD group: History of significant alcohol consumption as defined by WHO criteria (>14 standard drinks per week for women and >21 standard drinks per week for men), assessed using the WHO STEPwise Approach to Surveillance (STEPS) questionnaire. Alcohol content in traditional Nepali beverages was estimated using published local data.[15]

NAFLD group: No significant alcohol use history.

Exclusion criteria included patients with viral hepatitis (B or C), drug-induced liver injury, autoimmune or hereditary liver diseases, those on lipid-lowering medications, pregnant women, and patients with incomplete biochemical data.

All statistical analyses were performed using IBM SPSS-20.

RESULTS

Demographic and clinical characteristics: A total of 146 patients with ultrasonography-confirmed fatty liver disease were enrolled. Of these, 59 (40.4%) were classified as AFLD and 87 (59.6%) as NAFLD. The majority of participants were aged 46-55 years (n≈64, 43.8%), followed by those aged 36-45 years (n≈49, 33.6%), over 55 years (n≈27, 18.5%), and 25-35 years (n≈10, 6.8%). Among all participants, (81.5%) did not have diabetes, while 31 (21.2%) had a diagnosis of type 2 diabetes mellitus. The study cohort comprised 98 (67.1%) male and 48 (39.2%) female participants.

The Shapiro-Wilk test revealed that age (W=0.970, p=0.278 for AFLD; W=0.984, p=0.535 for NAFLD) and TC (W=0.964, p=0.160 for AFLD; W=0.974, p=0.179 for NAFLD) were normally distributed in both groups and were therefore analysed using the independent samples *t*-test. All other variables including TG, HDL, LDL, ALT, AST, De Ritis ratio, and TG/HDL ratio were non-normally distributed in one or both groups (p <0.05) and were analysed using non-parametric methods.

Table 2 presents the comparison of biochemical parameters between the AFLD and NAFLD groups. TC was significantly higher in the AFLD group (mean 270.96 ± 12.89 mg/dL) compared to the NAFLD group (mean 180.87 ± 6.54 mg/dL; p < 0.001). TG were higher in the AFLD group (median 192.0 mg/dL) compared to NAFLD (median 184.0 mg/dL), though this difference was also statistically significant on Mann-Whitney U testing (p <0.001). HDL was comparable between groups (AFLD median 38.0 vs NAFLD median 36.0 mg/dL). LDL was significantly higher in the AFLD group (median 148.0 mg/dL vs 110.5 mg/dL; p <0.001). AST (median 130.0 vs 75.0 U/L; p <0.001) and the De Ritis ratio (median 2.06 vs 1.47; p <0.001) were both significantly higher in the AFLD group.

De Ritis ratio and AFLD: bivariate analysis: The De Ritis ratio was dichotomised with threshold of ≥2 versus <2 and its association with AFLD was assessed by Chi-square analysis. Of 59

Table 1: Association between De Ritis ratio (AST/ALT) and type of fatty liver disease

		Fatty liver disease		Total	P value from Chi-square test	Pearson X^2 (df)
		Alcoholic	Non -alcoholic			
De Ritis ratio (AST/ALT)	No (<2)	24 (31.2%)	53 (63.8%)	77 (100%)	0.016 (<0.05)	5.779
	Yes ≥ 2	35 (50.7%)	34 (49.3%)	69 (100%)		
Total		**59 (40.4%)**	**87 (59.6%)**	**146 (100%)**		

Table 2: Comparison of parameters between AFLD and NAFLD groups

Variable		AFLD (n=59)	NAFLD (n=87)	Test statistic	P value
Age (years)		46.15 ± 7.94	47.76 ± 8.75	t = -0.997	0.3210
Sex	Male	46 (78.0%)	52 (59.8%)	X^2 = 5.11	0.0240
	Female	13 (22.0%)	35 (40.2%)		
Total cholesterol (mg/dL)		270.96 ± 12.89	180.87 ± 6.54	t = 48.829	<0.0001 *
Triglycerides (mg/dL)		189.00 (187.0-196.5)	171.00 (165.0-198.0)	U = 2221.5	<0.0001 *
HDL cholesterol (mg/dL)		36.00 (34.0-41.8)	35.00 (34.0-40.5)	U = 1690.5	0.3808
LDL cholesterol (mg/dL)		149.00 (125.8-155.8)	110.00 (103.5-116.0)	U = 2491.0	<0.0001 *
ALT (U/L)		59.50 (44.5-102.8)	56.00 (48.0-64.5)	U = 1789.5	0.1471
AST (U/L)		121.50 (96.0-221.0)	72.00 (58.5-99.0)	U = 2444.5	<0.0001 *
De Ritis ratio (AST/ALT)		2.22 (1.7-2.5)	1.31 (1.0-2.0)	U = 2444.0	<0.0001 *
TG/HDL ratio		5.28 (4.6-5.7)	4.89 (4.4-5.3)	U = 1888.0	0.0429 *

*Normally distributed variables expressed as mean ± SD (independent t-test); non-normally distributed variables expressed as median (IQR) (Mann-Whitney U test). * p <0.05.*

Table 3: Multivariable logistic regression: independent predictors of AFLD

Variable	Crude OR (95% CI)	Adjusted OR	95% CI	P value
De Ritis ratio	2.27 (1.15-4.46)	1.30	0.55–3.05	0.545
Age	0.98 (0.94-1.02)	0.99	0.93–1.05	0.792
Male sex	**2.38 (1.11-5.11)**	**3.05**	1.00–9.31	**0.050 ***
Diabetes mellitus	0.85 (0.37-1.95)	0.91	0.27–3.16	0.888
Triglycerides (TG)	**1.02 (1.0-1.03)**	**0.96**	0.92–0.99	**0.012 ***
Total cholesterol (TC)	**4.35 (2.95-6.42)**	**1.05**	1.03–1.06	**<0.001 ***
HDL cholesterol	1.02 (0.97-1.07)	1.06	0.99–1.14	0.099

*OR = Odds Ratio; CI = Confidence Interval. * p < 0.05. Reference category: NAFLD.*

AFLD patients, 35 (59.3%) had an elevated De Ritis ratio, compared to 34 of 87 (39.1%) NAFLD patients. Chi-square analysis (Table 1) demonstrates a statistically significant associationbetween the dichotomized ratio and fatty liver etiology, yielding a *p*-value of 0.016 derived from Pearson Chi-square test (Pearson χ^2 =5.78, df = 1, p 0.016), with a weak but significant effect size (Cramér's V =0.199). Fisher's exact test confirmed this association (p =0.019). All chi-square assumptions were met (minimum expected count =27.88).

Multivariable logistic regression: Multivariable logistic regression was performed to identify independent predictors of AFLD. The model demonstrated good fit (omnibus χ^2 =82.97, df =5, p <0.001; Nagelkerke R^2 =0.585). On univariate screening, TC was the strongest predictor (Score statistic =59.61, p <0.001), followed by the De Ritis ratio (Score =5.78, p =0.016). Male sex showed a clear link with AFLD, coming right to the edge of being statistically significant (adjusted OR =3.05, 95% CI: 1.00-9.31, p =0.050). After multivariable

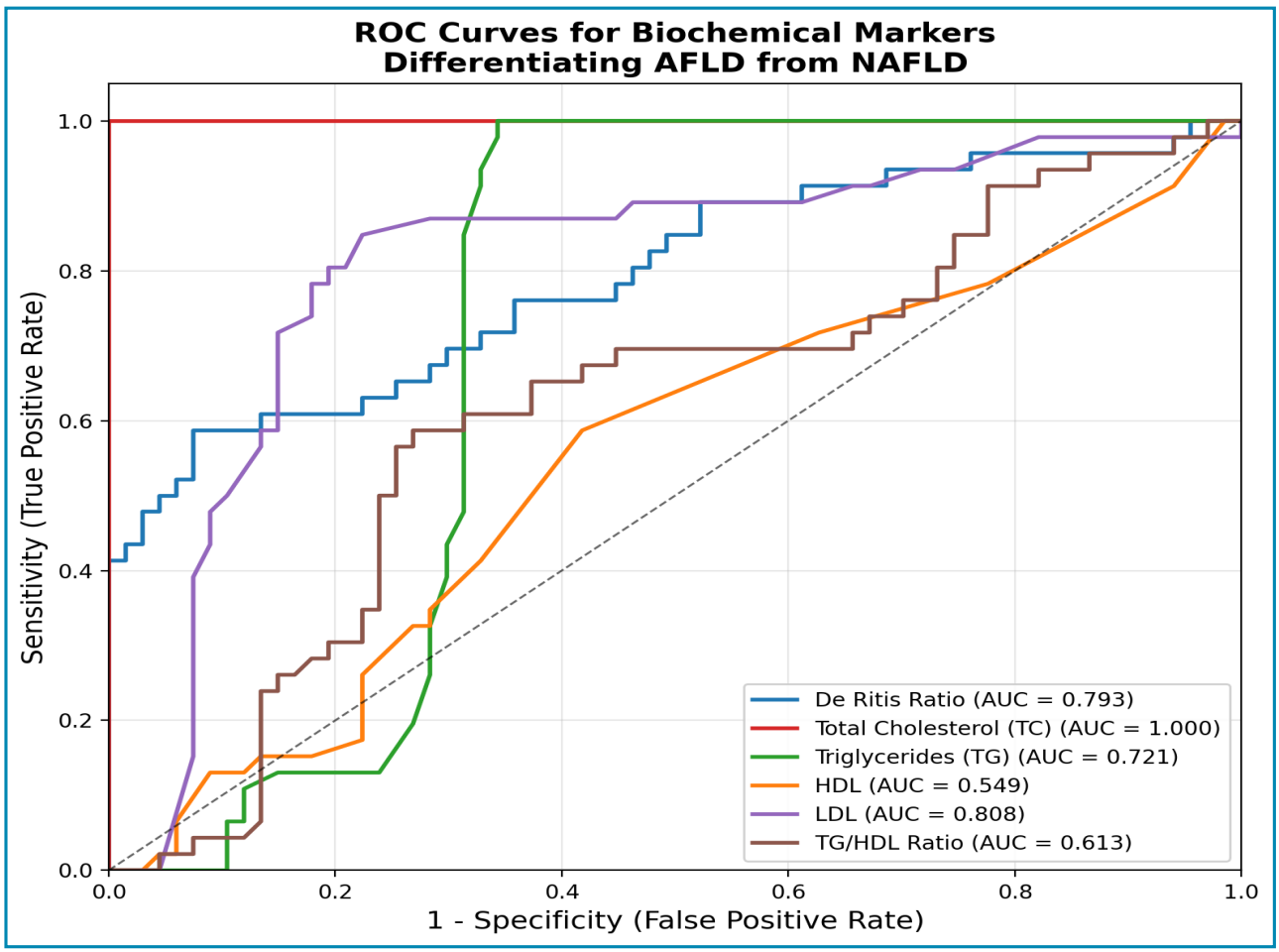

Fig. 1: ROC curves for biochemical markers discriminating AFLD from NAFLD. TC = Total cholesterol; TG = Triglycerides; HDL = High-density lipoprotein; LDL = Low-density lipoprotein; AUC = Area under the curve

adjustment, TC remained a highly significant independent predictor (adjusted OR =1.05, 95% CI: 1.03-1.06, p <0.001). TG was independently and inversely associated with AFLD (adjusted OR =0.96, 95% CI: 0.92–0.99, p =0.012). Male sex showed marginal independent significance (adjusted OR =3.05, 95% CI: 1.00–9.31, p =0.050). Importantly, the De Ritis ratio did not retain independent significance after adjustment (adjusted OR =1.30, 95% CI: 0.55–3.05, p =0.545). Notably, diabetes mellitus status was analysed into the multivariable logistic regression model to evaluate its role as a confounding metabolic factor; however, it did not emerge as a statistically significant independent predictor of fatty liver etiology (adjusted OR =0.91, 95% CI: 0.27-3.16, p =0.888). Age and HDL were non-significant in the multivariable model (Table 4).

Odds ratios shows that for every 1 mg/dL increase in TC, the odds of a patient having AFLD increase by 5% (Adjusted OR =1.05). Conversely, TG showed an inverse pattern, with each 1 mg/dL increase reducing the odds of AFLD classification by 4% (Adjusted OR =0.96). Male sex showed a strong trend, with men having roughly 3 times higher odds of being classified with AFLD than women (Adjusted OR =3.05), though this result sits exactly on the significance boundary (p =0.050). Other factors, including the De Ritis ratio, age, diabetes, and HDL, did not show independent predictive value after adjustment."

ROC curve analysis: ROC curve analysis was performed for all biochemical markers (Fig. 1). TC demonstrated perfect discriminatory performance (AUC =1.000) for AFLD, consistent with the complete non-overlap in TC values between groups (AFLD: 248-295 mg/dL; NAFLD: 165-193 mg/dL). LDL showed excellent discriminatory ability (AUC =0.808), followed by the De Ritis ratio (AUC =0.793) and TG (AUC =0.721). The TG/HDL ratio (AUC =0.613) and HDL alone (AUC =0.549) demonstrated limited discriminatory value, approaching chance level.

DISCUSSION

This comparative cross-sectional study evaluated the utility of the De Ritis ratio and lipid profile in differentiating ultrasonographically-diagnosed AFLD from NAFLD. The principal findings were: (i) total cholesterol and triglycerides are independent biochemical discriminators between AFLD and NAFLD; (ii) the De Ritis ratio, while significantly associated with AFLD on bivariate analysis, does not retain independent predictive value after controlling for metabolic variables; and (iii) ROC analysis confirms excellent discriminatory performance for TC (AUC =1.000) and LDL (AUC =0.808) for the identification of AFLD. These findings have important implications for non-invasive clinical differentiation of fatty liver subtypes, particularly in resource-limited settings.

The demographic profile of our cohort - predominantly middle-aged adults with a higher prevalence of NAFLD mirrors global epidemiological data. NAFLD affects approximately one in four adults worldwide, with incidence rising in parallel with metabolic syndrome.[1] The predominance of patients aged 46-55 years reflects the convergence of cumulative metabolic risk factor burden and alcohol exposure at this life stage.

The De Ritis ratio has historically been considered a reliable marker for alcoholic liver disease, with values ≥2.0 strongly suggesting alcoholic aetiology.[8,9] In our cohort, a higher proportion of AFLD patients had an elevated ratio compared to NAFLD patients (59.3% vs 39.1%), yielding a significant bivariate association (p =0.016). However, the effect size was modest (Cramér's V =0.199) and on multivariable regression, the ratio failed to retain significance. This finding is consistent with emerging literature suggesting that the De Ritis ratio is susceptible to metabolic confounding.[10,17] In patients with NAFLD characterized by insulin resistance and hepatocyte damage - ALT may be substantially elevated, attenuating the AST/ALT ratio even when alcohol-related mitochondrial injury is present. Furthermore, the ratio's discriminatory power is diminished in mild or early disease, where aminotransferase elevations are modest in both conditions.

The most clinically significant finding of this study is the independent discriminatory role of TC and TG. Higher TC was independently associated with AFLD (adjusted OR =1.05 per unit increase, p <0.001), while higher TG was inversely associated (adjusted OR =0.96, p =0.012), indicating that NAFLD patients exhibited more pronounced hypertriglyceridaemia. These findings align with established pathophysiological differences: NAFLD is driven by insulin resistance and hepatic de novo lipogenesis, generating high VLDL output and consequent hypertriglyceridaemia with reduced HDL.[11,12] In contrast, chronic AFLD especially in advanced stages is complicated by hepatic synthetic dysfunction, malnutrition, and disrupted apolipoprotein production, leading to variable and often lower TG levels.[18] The higher TC

in AFLD likely reflects alcohol-mediated impairment of hepatic cholesterol esterification and altered lipoprotein synthesis pathways.[19] These lipid differences are consistent with the findings of Shah and Ali[13] and Ramesh *et al*,[16] who similarly documented distinct lipid profiles between AFLD and NAFLD cohorts.

ROC analysis revealed that TC is an exceptional discriminator between AFLD and NAFLD in our cohort, with a perfect AUC of 1.000. This reflects the complete absence of overlap in TC values between the two groups - a striking finding that, while biologically plausible, should be interpreted with caution given the relatively modest sample size and the possibility of centre-specific referral patterns. LDL also performed well (AUC = 0.808), consistent with its role in reflecting alcohol's differential effects on hepatic cholesterol metabolism. The De Ritis ratio achieved an AUC of 0.793, confirming its utility as a screening tool despite its limitations as an independent multivariable predictor.

Male sex was marginally independently associated with AFLD (adjusted OR=3.05, p=0.050), consistent with the globally documented higher prevalence of hazardous alcohol consumption among men.[3] In Nepal, cultural and social factors further amplify male alcohol use, making sex a relevant clinical consideration in the differential diagnosis of fatty liver disease.

The findings of this study have direct clinical relevance for healthcare providers in resource-limited settings. Liver biopsy - the definitive diagnostic standard remains inaccessible for the majority of patients in Nepal and across South Asia. The identification of TC and TG as independent non-invasive discriminators, combined with a structured alcohol history using validated instruments such as the WHO STEPS questionnaire, offers a practical, low-cost diagnostic algorithm applicable at the primary and secondary care levels. While the De Ritis ratio should not be abandoned, its bivariate significance and ROC performance remain clinically useful - clinicians should exercise caution in relying upon it as a standalone discriminator, particularly in patients with concurrent metabolic risk factors.

Limitations: Several limitations of this study warrant consideration. First, the cross-sectional design precludes causal inference or assessment of disease progression over time. Second, classification into AFLD relied upon self-reported alcohol history, which is susceptible to underreporting and social desirability bias, potentially leading to group misclassification. Third, the absence of histopathological confirmation via liver biopsy or non-invasive fibrosis assessment means that the degree of steatohepatitis, fibrosis, and disease staging could not be verified in either group. Fourth, despite controlling for several confounders, residual confounding from factors such as insulin resistance, dietary patterns, and physical activity levels cannot be excluded. Fifth, as a single tertiary care centre study, the findings may not be fully generalisable to community or primary care populations, where milder disease predominates. Finally, the striking AUC of 1.000 for TC, while a genuine finding in this dataset, should be interpreted cautiously and requires external validation in larger, multi-centre cohorts.

In conclusion, TC and TG are independent non-invasive biochemical markers capable of differentiating AFLD from NAFLD in patients with ultrasonography-confirmed fatty liver disease. The De Ritis ratio, while a valuable initial screening marker, does not independently discriminate between the two conditions after accounting for metabolic confounders. Lipid profiling, integrated with thorough clinical history and abdominal ultrasonography, represents a practical, accessible, and cost-effective diagnostic strategy - particularly suited to resource-limited clinical settings such as Nepal. These findings support the routine incorporation of a full fasting lipid panel in the diagnostic workup of fatty liver disease. Multi-centre, longitudinal studies with histopathological validation are warranted to confirm and extend these findings across diverse populations.

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