

Original Article

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Association of Serum Lipids with Ultrasound-Based MASLD Severity

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Abstract:

Background and Objectives: Metabolic dysfunction-Associated Steatotic Liver Disease (MASLD) is a common global condition associated with metabolic dysfunction. This study investigated the relationship between ultrasound-graded MASLD severity and serum lipid levels in adults.

Methods: A total of 101 participants (32 males, 69 females) with ultrasound-diagnosed MASLD were included. Participants were categorized as Grade I (n=79), Grade II (n=21), and Grade III (n=1). Serum lipids, including total cholesterol, triglycerides, HDL, and LDL, were measured. Data were analyzed using one-way ANOVA and Pearson correlation.

Results: The mean age was 50.7±13.1 years, with a mean BMI (Body Mass Index) of 27.2±4.43 kg/m². No statistically significant differences in lipid levels were observed across MASLD grades (p>0.05). Correlation analysis showed weak, non-significant associations between MASLD grade and lipid levels, as well as between the lipid parameters.

Conclusion: Lack of significant relationship between ultrasound-graded MASLD severity and serum lipid levels, suggested that factors beyond lipid levels may contribute to disease progression.

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Introduction:

Metabolic dysfunction-Associated Steatotic Liver Disease (MASLD) has emerged as the most prevalent chronic liver disease worldwide, affecting approximately 25%–30% of the global population and showing a greater prevalence among males.¹

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The disease encompasses a spectrum ranging from simple steatosis to non-alcoholic steatohepatitis, potentially progressing to cirrhosis and hepatocellular carcinoma. MASLD is strongly associated with metabolic syndrome components, including obesity, insulin resistance, dyslipidemia, and type 2 diabetes mellitus.² The pathophysiology of MASLD involves complex metabolic derangements, with lipid metabolism disturbances playing a central role. Dysregulated lipid homeostasis contributes to hepatic triglyceride accumulation, yet the precise relationship between circulating lipid profiles and disease severity remains incompletely understood.³ While numerous studies have documented lipid abnormalities in

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MASLD patients, the correlation between disease grade and specific lipid parameters shows considerable variability across populations.^{4,5} Ultrasound remains the primary non-invasive imaging modality for MASLD diagnosis and grading due to its widespread availability, cost-effectiveness, and safety profile.⁶ However, questions persist regarding whether ultrasound-determined disease severity correlates with metabolic parameters such as serum lipid concentrations.⁷ This study aimed to evaluate the association between ultrasound-graded MASLD severity and lipid profile abnormalities, providing insights into disease mechanisms.

Materials and Method

Study Design

This was a cross-sectional observational study conducted at a tertiary care teaching hospital. Ethical approval was obtained from the institutional review board.

Participant Selection

Adult patients aged 18-80 years with ultrasound-confirmed MASLD were enrolled in the study by convenience sampling. Individuals were excluded if they had a history of diabetes, alcohol consumption, viral hepatitis, autoimmune liver disease, drug-induced hepatotoxicity, or other secondary causes of hepatic steatosis.

Hepatic Ultrasound

Hepatic ultrasonography was performed using standard protocols by experienced radiologists. The severity of MASLD (hepatic steatosis) was graded according to predefined sonological criteria (Table I).

Table I: Working definition of MASLD Grades⁸

Grade of MASLD	Criteria
Grade I	Mild steatosis with diffusely increased hepatic echogenicity with normal visualization of the diaphragm and intrahepatic vessel borders.
Grade II	Moderate steatosis with moderate increase in echogenicity with slightly impaired visualization of intrahepatic vessels and the diaphragm.
Grade III	Severe steatosis with marked increase in echogenicity with poor or no visualization of intrahepatic vessels, diaphragm, and posterior portion of the liver.

Laboratory Assessment

Venous blood samples were collected after a 12-hour overnight fast. Serum lipid parameters-including total cholesterol, triglycerides, high-density lipoprotein and, low-density lipoprotein were measured using standardized photometric colorimetric methods on automated analyzers calibrated in accordance with the manufacturer's specifications.

Statistical Analysis

Data were analyzed using the statistical software R. Continuous variables were expressed as mean $\pm$ standard deviation (SD). Differences in lipid parameters across MASLD grades were assessed using one-way analysis of variance (ANOVA). Pearson's correlation coefficients were used to evaluate relationships between variables. A p-value <0.05 was considered statistically significant, and 95% confidence intervals were reported where applicable.

Result

The final study sample comprised 101 participants, with a male-to-female ratio of 1:2.2 (32 males and 69 females). This distribution pattern remained consistent across disease grades: Grade I patients included 25 males and 54 females, while Grade II comprised 7 males and 14 females. Only one participant had Grade III disease.

Analysis of age distribution showed that the majority of MASLD patients were in the 50–59-year age group, indicating peak disease prevalence among middle-aged adults. The overall mean age was 50.7 ± 13.1 years, with minimal variation between males (50.4 ± 13.6 years) and females (50.8 ± 12.9 years), suggesting an age-independent gender distribution of disease occurrence.

Figure 1: Male to Female ratio (A) and distribution of grades of MASLD (B) among participants

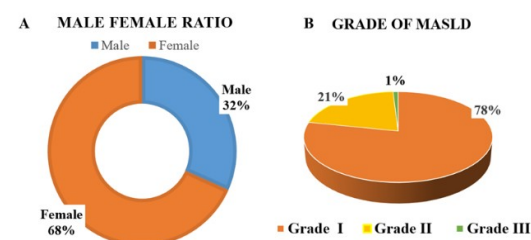
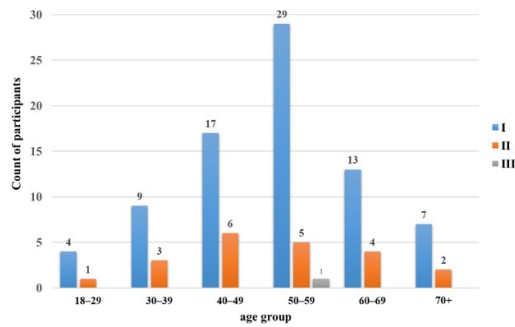


Figure 2: Grades of MASLD by age group distribution



Anthropometric measurements and baseline lipid parameters

Anthropometric measurements demonstrated expected sex-based differences in physical characteristics. Male participants exhibited a significantly greater mean height (164.5 ± 6.83 cm) than females (153.1 ± 5.53 cm), consistent with population norms. Body weight followed similar patterns, with males averaging 76.97 ± 14.70 kg versus 62.49 ± 10.77 kg in females. Consequently, body mass index showed modest elevation in males (28.4 ± 5.14 kg/m²) compared to females (26.6 ± 3.96 kg/m²), with an overall cohort mean of 27.2 ± 4.43 kg/m², placing the population in the overweight-to-obese category. Baseline lipid profile analysis revealed notable patterns. Male participants demonstrated higher mean concentrations of total cholesterol (199.0 ± 96.5 mg/dL), triglycerides (238.0 ± 152.0 mg/dL), and LDL cholesterol (109.0 ± 36.6 mg/dL) compared to females. Conversely, HDL cholesterol levels were lower in males (32.8 ± 13.7 mg/dL) than in females (36.0 ± 11.5 mg/dL).

Table II: Summary Statistics of the study participants*

Parameter	Overall mean $\pm$ SD	Male (n=32) mean $\pm$ SD	Female (n=69) mean $\pm$ SD
Age (year)	50.7 $\pm$ 13.1	50.4 $\pm$ 13.6	50.8 $\pm$ 12.9
Height (cm)	156.7 $\pm$ 7.96	164.5 $\pm$ 6.83	153.1 $\pm$ 5.53
Weight (kg)	67.08 $\pm$ 13.85	76.97 $\pm$ 14.70	62.49 $\pm$ 10.77
BMI (Kg/m ²)	27.2 $\pm$ 4.43	28.4 $\pm$ 5.14	26.6 $\pm$ 3.96
TC (mg/dL)	191.0 $\pm$ 69.7	199.0 $\pm$ 96.5	187.0 $\pm$ 53.4
TG (mg/dL)	229.0 $\pm$ 146.0	238.0 $\pm$ 152.0	224.0 $\pm$ 143.0
HDL (mg/dL)	35.0 $\pm$ 12.3	32.8 $\pm$ 13.7	36.0 $\pm$ 11.5
LDL (mg/dL)	104.0 $\pm$ 37.6	109.0 $\pm$ 36.6	101.0 $\pm$ 38.0
*Average BMI above 20 kg/m ²			

Lipid profile across MASLD grades

One-way analysis of variance was conducted to evaluate potential differences in serum lipid concentrations across the three MASLD severity grades. The analysis included 79 Grade I patients (78% of the cohort), 21 Grade II patients (21%), and one Grade III patient (1%). Despite theoretical expectations of progressive lipid derangement with increasing disease severity, the statistical analysis yielded unexpected findings. Mean total cholesterol levels remained remarkably consistent across grades: 191.0 ± 74.3 mg/dL in Grade I, 194.0 ± 51.9 mg/dL in Grade II, and 167 mg/dL in the single Grade III patient, producing an F-statistic of 0.929 ($p=0.929$). Triglyceride concentrations showed greater numerical variation, with Grade II patients exhibiting the highest mean (282.0 ± 183.0 mg/dL) compared to Grade I (216.0 ± 132.0 mg/dL) and Grade III (131 mg/dL), yet this difference failed to achieve statistical significance ($F=0.141$, $p=0.141$).

Table III: Comparison of serum lipid profile among different grades of MASLD

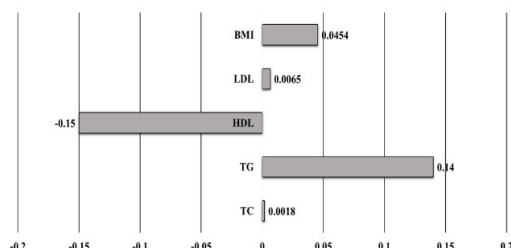
Lipids	Overall Mean $\pm$ SD	Grade I Mean $\pm$ SD (78%)	Grade II Mean $\pm$ sd (21%)	Grade III (1%)	ANOVA
TC	191.0 $\pm$ 69.7	191.0 $\pm$ 74.3	194.0 $\pm$ 51.9	167	0.929
TG	229.0 $\pm$ 146.0	216.0 $\pm$ 132.0	282.0 $\pm$ 183.0	131	0.141
HDL	35.0 $\pm$ 12.3	36.2 $\pm$ 13.2	29.8 $\pm$ 5.67	46	0.067
LDL	104.0 $\pm$ 37.6	103.0 $\pm$ 37.7	105.0 $\pm$ 38.9	95	0.961

HDL cholesterol demonstrated the most intriguing pattern, with Grade II patients showing the lowest mean concentration (29.8 ± 5.67 mg/dL) while the single Grade III patient presented an unexpectedly elevated value (46 mg/dL). Despite approaching significance, the overall comparison yielded $p=0.067$. LDL cholesterol levels remained virtually identical across all grades, with an F-statistic of 0.961 ($p=0.961$), indicating minimal between-group variance relative to within-group variation.

Correlation between MASLD grade and lipid parameters

Pearson correlation analysis was performed to assess linear relationships between ultrasound-determined MASLD grade and individual lipid parameters. This approach provides continuous assessment of association strength beyond categorical group comparisons offered by ANOVA. The correlation between MASLD grade and total cholesterol was essentially negligible ($r=0.002$, 95% CI: -0.194 to 0.197, $p=0.985$), indicating complete absence of linear association. Triglyceride levels showed the strongest positive correlation coefficient ($r=0.140$, 95% CI: -0.053 to 0.330), suggesting a weak trend toward elevated triglycerides with increasing disease severity, though this relationship remained statistically non-significant ($p=0.152$). HDL cholesterol exhibited a weak negative correlation with MASLD grade ($r=0.150$, 95% CI: -0.340 to 0.037, $p=0.112$), approaching but not reaching statistical significance. This inverse relationship aligns with pathophysiological expectations, as progressive hepatic steatosis might theoretically impair HDL metabolism. However, the wide confidence interval crossing zero indicates substantial uncertainty. LDL cholesterol correlation with disease grade was negligible ($r=0.007$, $p=0.948$). Body mass index similarly demonstrated no meaningful association with MASLD severity ($r=0.045$, $p=0.652$).

Figure 3: Pearson correlation for MASLD and dyslipidemia



Discussion

The present study evaluated the relationship between serum lipid profile and ultrasonographic severity of metabolic dysfunction-associated steatotic liver disease (MASLD) among 101 participants. The findings demonstrated that although dyslipidemia was highly prevalent in the study population, serum lipid parameters did not show statistically significant associations with

increasing MASLD grade. These observations provide important insights into the metabolic characteristics of MASLD and highlight the complex pathophysiology underlying disease progression.⁹

A notable demographic finding was the predominance of female participants, with a male-to-female ratio of 1:2.2. Similar female predominance has been reported in several South Asian hospital-based studies, particularly among middle-aged populations where obesity and metabolic syndrome are common among women. However, global epidemiological data often report higher MASLD prevalence in males, especially in younger age groups.¹⁰ The higher proportion of females in this cohort may therefore reflect local referral patterns, healthcare-seeking behavior, or sociocultural and metabolic factors specific to the study population. Importantly, the mean age was nearly identical between males and females, suggesting that age-related disease occurrence was independent of gender in this cohort.

The majority of patients belonged to the 50–59-year age group, with an overall mean age of approximately 51 years. This finding is consistent with the recognized natural history of MASLD, which commonly manifests during middle age after prolonged exposure to metabolic risk factors such as obesity, insulin resistance, dyslipidemia, and sedentary lifestyle.¹¹ Similar age distributions have been documented in previous regional and international studies, supporting the concept that MASLD is predominantly a disease of middle-aged adults.

Anthropometric assessment showed that the overall cohort was overweight to obese, with a mean BMI of 27.2 ± 4.43 kg/m². Obesity is a well-established risk factor for hepatic steatosis and plays a central role in insulin resistance and lipid accumulation within hepatocytes.¹² Male participants demonstrated higher body weight and BMI than females, reflecting sex-related differences in body composition and visceral adiposity.^{13,14} Despite the recognized relationship between obesity and MASLD development, BMI in this study showed no significant correlation with disease severity. This finding suggests that while obesity contributes to MASLD occurrence,

progression of steatosis may depend more on metabolic and inflammatory factors than on body weight alone. Similar observations have been reported in previous studies where BMI failed to predict histological severity or fibrosis stage.³

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The baseline lipid profile findings revealed a pattern characteristic of metabolic dyslipidemia. Male participants exhibited higher levels of total cholesterol, triglycerides, and LDL cholesterol, along with lower HDL cholesterol levels compared to females. These findings are physiologically plausible, as males tend to have greater visceral adiposity and insulin resistance, both of which contribute to adverse lipid metabolism. Low HDL cholesterol and elevated triglyceride levels are particularly characteristic of insulin-resistant states and are commonly associated with MASLD.¹³ Triglyceride levels showed a tendency toward higher values in Grade II disease, while HDL cholesterol demonstrated lower mean levels in Grade II patients; however, these differences did not achieve statistical significance. These findings may indicate that lipid abnormalities contribute more strongly to the initiation of hepatic steatosis rather than to progression in ultrasonographic severity.^{3,4}

Several factors may explain the absence of statistically significant associations. First, the

study population was heavily skewed toward Grade I disease, which accounted for 78% of participants, while only one patient had Grade III disease. Such unequal distribution substantially reduces statistical power to detect intergroup differences. Second, ultrasonography, although widely used and practical, has limited sensitivity in differentiating mild from moderate steatosis and cannot accurately assess inflammation or fibrosis. Therefore, ultrasonographic grading may not precisely reflect the metabolic severity of MASLD. Third, lipid metabolism in MASLD is influenced by multiple confounding factors including diabetes mellitus, dietary habits, physical activity, genetic predisposition, and use of lipid-lowering medications, which may obscure direct associations. The Pearson correlation analysis further supported the absence of strong linear relationships between lipid parameters and disease severity. Total cholesterol and LDL cholesterol demonstrated essentially negligible correlations with MASLD grade. Triglycerides showed the strongest positive correlation, although the relationship remained weak and statistically non-significant. This trend is nevertheless biologically meaningful because hypertriglyceridemia plays an important role in hepatic fat accumulation through increased free fatty acid delivery and impaired lipid export from hepatocytes. Previous studies have similarly reported triglycerides as one of the lipid parameters most closely linked with hepatic steatosis.¹⁶

HDL cholesterol demonstrated a weak inverse relationship with MASLD grade, consistent with current understanding of MASLD pathophysiology. Progressive hepatic steatosis is associated with impaired HDL synthesis and altered reverse cholesterol transport. Although the association did not reach statistical significance, the observed trend toward lower HDL levels in more advanced disease aligns with findings from earlier investigations. The lack of significance may again be attributable to limited sample size and underrepresentation of severe disease.¹⁷

An important consideration is the unexpectedly elevated HDL value observed in the single Grade III patient. Because only one participant belonged to this category, the value

likely represents an outlier and should not be interpreted as evidence against the expected inverse association between HDL and disease severity. The extremely small Grade III sample limits meaningful interpretation of advanced disease characteristics and represents a major limitation of the study.

Overall, the findings suggest that while dyslipidemia is common among patients with MASLD, serum lipid parameters alone may not reliably predict ultrasonographic disease severity. MASLD progression is increasingly recognized as a multifactorial process involving insulin resistance, oxidative stress, inflammatory cytokines, mitochondrial dysfunction, and genetic susceptibility in addition to lipid abnormalities. Therefore, reliance solely on conventional lipid markers may underestimate the complexity of disease progression.

Limitation

The cross-sectional design of the study precludes assessment of temporal or causal relationships. The sample size, particularly for advanced MASLD grades, was limited. Ultrasonography was used instead of histopathology or advanced imaging modalities such as transient elastography or MRI-based techniques. Additionally, important confounding variables such as medication use, dietary patterns, and physical activity were not analyzed in detail.

Conclusion

This study found no statistically significant associations between ultrasound-determined MASLD severity grades and serum lipid profile parameters, including total cholesterol, triglycerides, HDL, and LDL cholesterol. The absence of correlation suggests that circulating serum lipid concentrations may not reliably reflect the degree of hepatic steatosis, indicating that MASLD progression involves complex mechanisms beyond simple lipid accumulation detectable in the blood. Further investigation using histological confirmation, advanced imaging techniques, longitudinal designs, and assessment of additional metabolic and genetic factors is necessary to fully reveal the relationship between hepatic fat deposition and systemic lipid metabolism in MASLD patients.

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