SSR Institute of International Journal of Life Sciences
ISSN (O): 2581-8740 | ISSN (P): 2581-8732
Mohanty *et al*., 2026
doi: 10.21276/SSR-IIJLS.2026.12.5.18

Original Article

open access

Serum Interleukin-6 and Serum Fibrinogen as Non-Invasive Biomarkers of Hepatic Inflammation and Fibrosis in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD): A Cross-Sectional Study

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Received: 20 Jun 2026/ Revised: 30 Jul 2026/ Accepted: 21 Aug 2026

ABSTRACT

Background: Metabolic dysfunction-associated steatotic liver disease (MASLD), formerly non-alcoholic fatty liver disease, is the common chronic liver disease worldwide and is associated with chronic hepatic inflammation. This study evaluated serum interleukin-6 (IL-6) and fibrinogen as non-invasive biomarkers of hepatic steatosis and fibrosis severity and assessed their correlation with liver stiffness measurement (LSM).

Methods: This cross-sectional study included 100 subjects fulfilling diagnostic criteria for MASLD recruited from General Medicine, Hepatology, and Gastroenterology departments at a tertiary centre in Odisha, India. Serum fibrinogen and IL-6 were estimated, and subjects underwent abdominal ultrasonography and transient elastography (FibroScan) for LSM. FIB-4, BARD, and APRI scores were calculated. Associations were assessed using Chi-square test, ANOVA, and independent t-test; Pearson correlation assessed continuous variables. A p-value ≤0.05 was considered statistically significant.

Results: Among 100 subjects, 55 were female and 45 male, with mean age 49 years. Obesity (BMI >25 kg/m²), diabetes, dyslipidaemia, and hypertension were present in 83%, 97%, 88%, and 73%, respectively. Male sex, older age, higher BMI, and poor glycaemic control (HbA1c ≥7%) were associated with higher LSM ($p<0.05$). Raised fibrinogen (>400 mg/dL) and IL-6 (>7 pg/mL) occurred in 58% and 75%, respectively. Both markers positively correlated with LSM ($r=0.92$ and $r=0.84$, respectively; $p<0.05$) and were associated with FIB-4 score. They also correlated with each other ($r=0.85$, $p<0.05$).

Conclusion: Serum IL-6 and fibrinogen correlated strongly with LSM and fibrosis risk scores in MASLD, supporting their potential utility as non-invasive adjunct biomarkers for assessing hepatic inflammation and fibrosis severity. Larger multicentre longitudinal studies are warranted.

Key-words: MASLD; Interleukin-6; Fibrinogen; Liver Stiffness Measurement; FIB-4; Hepatic Fibrosis

How to cite this article

Mohanty B, Sethy S, Sethi SS, Nanda G, Rout SK. Serum Interleukin-6 and Serum Fibrinogen as Non-Invasive Biomarkers of Hepatic Inflammation and Fibrosis in Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD): A Cross-Sectional Study. SSR Inst Int J Life Sci., 2026; 12(5): 10805-10812.

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INTRODUCTION

Metabolic dysfunction-associated liver disease (MASLD) is a recently redefined term that replaces non-alcoholic fatty liver disease (NAFLD) to better reflect its underlying metabolic origins [1]. MASLD is a chronic liver disorder characterized by excessive hepatic fat accumulation in individuals with cardiometabolic risk factors such as obesity, type 2 diabetes mellitus, dyslipidemia, and insulin resistance. It is the most common chronic liver disease globally, affecting approximately 25-30% of the

doi: 10.21276/SSR-IIJLS.2026.12.5.18

population, with rising prevalence driven by increasing rates of metabolic syndrome and sedentary lifestyles [1-3]. MASLD encompasses a spectrum ranging from simple hepatic steatosis to metabolic dysfunction-associated steatohepatitis (MASH), characterized by hepatocyte injury, inflammation, and progressive fibrosis, which can culminate in cirrhosis and hepatocellular carcinoma. The disease also carries significant systemic implications, with well-documented associations with cardiovascular, muscular, and renal complications [2,4-9].

Chronic low-grade inflammation is central to the pathogenesis and progression of MASLD. Insulin resistance promotes hepatic lipid accumulation, which triggers oxidative stress and the release of pro-inflammatory cytokines, driving hepatocellular injury and fibrogenesis. Among these mediators, interleukin-6 (IL-6) and fibrinogen, an acute-phase reactant induced by IL-6, are of particular interest as potential circulating markers of ongoing hepatic inflammation and fibrosis.

Liver biopsy remains the gold standard for diagnosing and staging hepatic steatosis and fibrosis [10], but its invasiveness, cost, and sampling variability limit its use for large-scale screening. This has driven interest in non-invasive alternatives, including serum-based composite scores such as the FIB-4 index, BARD score, and APRI score, as well as imaging-based techniques such as ultrasonography and transient elastography (FibroScan), which provides liver stiffness measurement (LSM) as a surrogate for fibrosis severity [11,12].

While established non-invasive tools such as FIB-4, BARD, APRI, ultrasonography, and transient elastography are widely used for assessing hepatic fibrosis, the utility of simple circulating inflammatory markers such as serum IL-6 and fibrinogen remains incompletely characterized in MASLD. Therefore, this study was undertaken to determine the possible association of serum IL-6 and fibrinogen with ongoing hepatic inflammation, fibrosis, and disease severity in patients with MASLD, and to correlate these serum marker levels with the degree of hepatic fibrosis assessed by ultrasonography and transient elastography.

MATERIALS AND METHODS

Study Design- This was a cross-sectional, observational study conducted at a tertiary care teaching hospital. All subjects fulfilling the inclusion criteria underwent simultaneous estimation of serum fibrinogen and serum interleukin-6 (IL-6), together with ultrasonography of the abdomen and pelvis and transient elastography (FibroScan) for correlation of biomarker levels with imaging-based disease severity.

Study Population- The study population comprised patients with MASLD attending the Departments of General Medicine, Hepatology, and Gastroenterology at a tertiary care medical college and hospital in Odisha, India.

Inclusion Criteria

- Age 30-75 years, both male and female subjects.
- Subjects fulfilling the cardiometabolic diagnostic criteria for MASLD, defined by hepatic steatosis plus at least one of the following five cardiometabolic criteria: (i) BMI >25 kg/m² (>23 kg/m² in Asian populations) or increased waist circumference; (ii) fasting glucose >100 mg/dL, 2-hour post-load glucose >140 mg/dL, HbA1c >5.7%, or type 2 diabetes/ treatment for it; (iii) blood pressure >130/85 mmHg or antihypertensive treatment; (iv) plasma triglycerides >150 mg/dL or lipid-lowering treatment; (v) plasma HDL-cholesterol <40 mg/dL in males or <50 mg/dL in females, or lipid-lowering treatment.

Exclusion Criteria

- Subjects unwilling to provide informed consent.
- Chronic alcoholics or diagnosed cases of alcohol-associated fatty liver disease.
- Subjects with clinical or laboratory evidence of iron deficiency anemia.
- Diagnosed cases of rheumatoid arthritis or other rheumatologic disease.
- Subjects on anti-IL-6 therapy (e.g., sarilumab).
- Subjects testing positive for hepatitis B, hepatitis C, or HIV.
- Subjects with prior liver transplantation.
- Subjects on regular blood transfusion for any haematological condition.
- Diagnosed cases of storage disorders.
- Subjects with technically inadequate imaging due to poor visualization or anatomical constraints.

Study Procedure- After providing informed consent, subjects underwent venous blood sampling following an overnight fast of 8 hours for estimation of serum fibrinogen and serum interleukin-6 (IL-6). At the same

doi: 10.21276/SSR-IIJLS.2026.12.5.18

visit, subjects underwent ultrasonography of the abdomen and pelvis to assess hepatic steatosis, and FibroScan (transient elastography) to quantify liver stiffness in kilopascals (kPa) as a surrogate for hepatic fibrosis. Liver stiffness measurements were subsequently correlated with serum fibrinogen and IL-6 levels, as well as with FIB-4, BARD, and APRI fibrosis risk scores.

Statistical Analysis- The study was conducted on 100 subjects. Data were analysed using IBM SPSS Software, version 29.0. Qualitative data were presented as frequency and percentage; quantitative data were presented as mean ± standard deviation. The Chi-square test was used to assess associations between categorical variables. ANOVA and independent t-tests were used to compare means across groups, and Pearson correlation coefficients were calculated for continuous variables. A p-value ≤0.05 was considered statistically significant.

RESULTS

A total of 100 subjects with MASLD were enrolled, comprising 55 women and 45 men, with a mean age of 49 years. Most subjects were aged 41–60 years. Obesity (BMI ≥25 kg/m²) was present in 83%, while diabetes, dyslipidemia, and hypertension were present in 97%, 88%, and 73%, respectively. AST elevation was more frequent than ALT elevation (88% vs. 28%). Transient elastography showed LSM <7 kPa in 52% and ≥7 kPa in 48%, with 21% having LSM >10.3 kPa. LSM ranged from 4.10–14.10 kPa. Ultrasonography showed grade II steatosis in 48% and grade III in 39%, with no sonographic evidence of overt cirrhosis (Fig. 1).

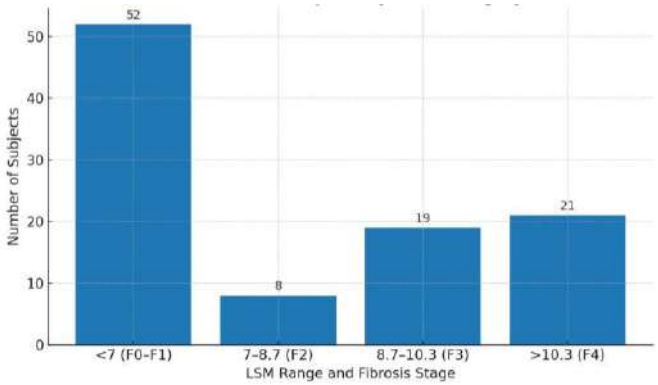

Fig. 1: Distribution of study subjects by liver stiffness measurement (LSM) category and corresponding fibrosis stage (F0-F4).

FIB-4 index values were in the intermediate-risk range (1.3–2.67) for the majority of subjects (64%), while 32% scored >2.67, indicating a high probability of advanced fibrosis, and only 4% fell in the low-risk range (<1.3) (Fig. 2). All subjects had a BARD score ≥2, with 53% scoring exactly 3 and 39% scoring 4. All subjects had APRI values within the 0.5–1.5 range.

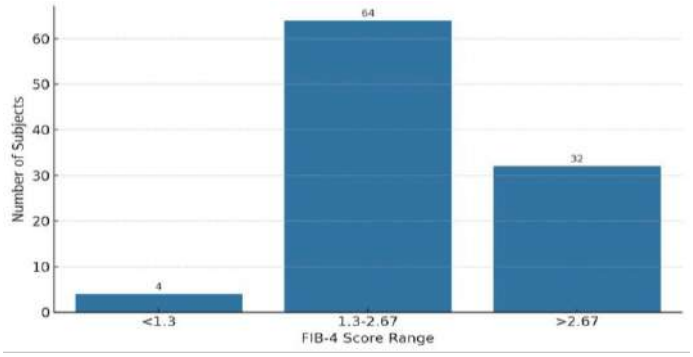

Fig. 2: Distribution of study subjects by FIB-4 index score category

Table 1 shows the association between demographic and metabolic risk factors and liver stiffness measurement (LSM). Male sex, age >50 years, BMI ≥25 kg/m², and poor glycemic control (HbA1c ≥7%) were significantly associated with LSM ≥7 kPa. In contrast, the presence of diabetes, dyslipidemia, and hypertension alone did not show a statistically significant association with LSM.

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Table 1: Association of demographic and metabolic risk factors with Liver Stiffness Measurement (LSM)

Variable	Category	LSM ≥7 kPa (%)	p-value
Gender	Male vs. Female	16/45 (36%) vs. 32/55 (58%)	0.017
Age group	≤50 vs. >50 yrs	23/55 (42%) vs. 25/45 (56%)	0.027
BMI	≥25 vs. <25 kg/m²	48/83 (58%) vs. 0/17 (0%)	0.00004
Glycemic control	HbA1c ≥7% vs. <7%	48/88 (55%) vs. 0/12 (0%)	0.001
Diabetes (presence)	Present vs. Absent	48/97 (49%) vs. 0/3 (0%)	0.27 (ns)
Dyslipidemia	Present vs. Absent	43/88 (49%) vs. 5/12 (42%)	0.44 (ns)
Hypertension	Present vs. Absent	33/73 (45%) vs. 16/27 (59%)	0.81 (ns)

Interpretation: *Male sex, older age, higher BMI, and poor glycemic control (HbA1c ≥7%) were each significantly associated with higher LSM (p<0.05), whereas the mere presence of diabetes, dyslipidemia, or hypertension — independent of glycemic or metabolic control — showed no significant association (p>0.05, ns = not significant), indicating that disease control, not diagnosis alone, drives fibrosis risk.*

Distribution of serum fibrinogen and serum interleukin-6 (IL-6) levels among the study subjects. Fig. 3A demonstrates the distribution of serum fibrinogen levels, showing the proportion of subjects with elevated fibrinogen concentrations, while Fig. 3B illustrates the distribution of serum IL-6 levels and the proportion of subjects with elevated IL-6 concentrations.

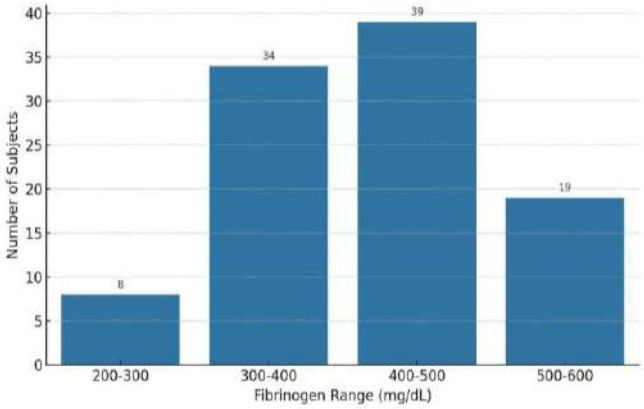
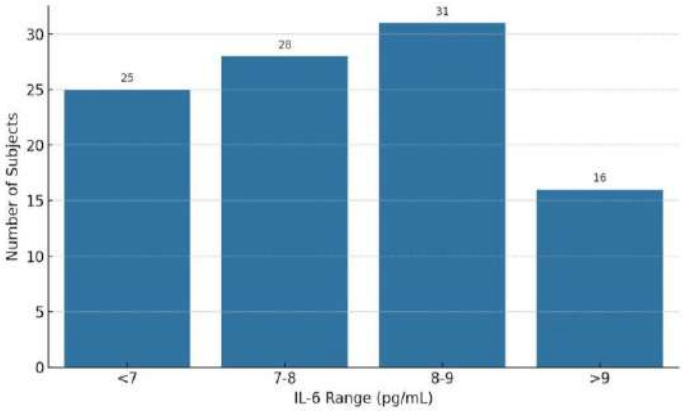


Fig. 3 (A): Distribution of serum fibrinogen levels among study subjects;



(B): Distribution of serum interleukin-6 (IL-6) levels among study subjects

Table 2 demonstrates the relationship of serum fibrinogen and serum IL-6 with LSM and FIB-4 score. Both serum fibrinogen and IL-6 showed strong positive correlations with LSM (r=0.92 and r=0.84, respectively) and were significantly associated with higher FIB-4 scores. Serum fibrinogen and IL-6 also showed a strong positive correlation with each other (r=0.85), indicating concordant elevation of both inflammatory markers.

Table 2: Serum fibrinogen and serum IL-6 vs. Liver Stiffness Measurement (LSM) and FIB-4 score

Comparison	Finding	Pearson r	p-value
Serum fibrinogen vs. LSM	58% elevated; concentrated in LSM ≥7 kPa group	0.92	<0.00001
Serum IL-6 vs. LSM	75% elevated; concentrated in LSM ≥7 kPa group	0.84	<0.0001
Serum fibrinogen vs. FIB-4	Elevated fibrinogen tracks high-risk FIB-4 (>2.67)	-	0.0001
Serum IL-6 vs. FIB-4	Elevated IL-6 tracks high-risk FIB-4 (>2.67)	-	<0.00002
Serum fibrinogen vs. serum IL-6	Concordant elevation in the same subjects	0.85	<0.0001

Interpretation: *Both serum fibrinogen and serum IL-6 showed strong, statistically significant positive correlations with liver stiffness and with FIB-4 score, and were themselves strongly correlated with one another — subjects with fibrinogen ≤400 mg/dL almost universally had IL-6 ≤7 pg/mL, and vice versa — pointing to a shared, concurrently activated inflammatory pathway underlying fibrosis severity in MASLD.*

doi: 10.21276/SSR-IIJLS.2026.12.5.18

Fig. 4A and 4B: Correlation between serum inflammatory biomarkers and liver stiffness measurement (LSM) in subjects with MASLD. (A) Correlation between serum fibrinogen levels and LSM, demonstrating a strong positive correlation (Pearson r = 0.92). (B) Correlation between serum interleukin-6 (IL-6) levels and LSM, demonstrating a strong positive correlation (Pearson r = 0.84).

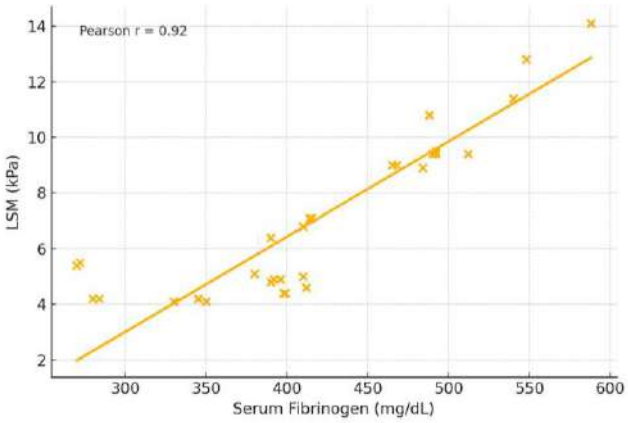
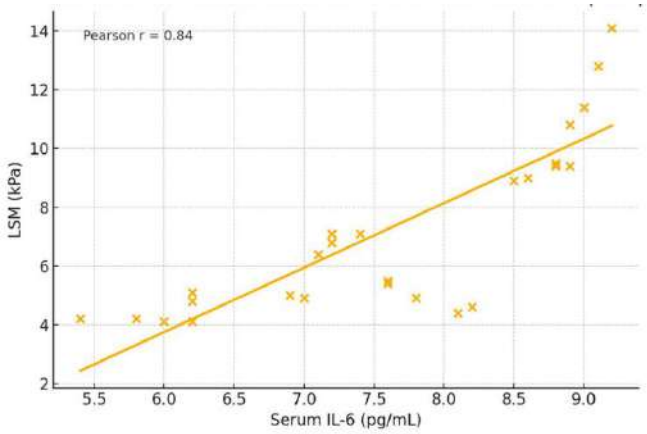


Fig. 4 (A): Correlation between serum fibrinogen and liver stiffness measurement (Pearson r = 0.92);



(B): Correlation between serum IL-6 and liver stiffness measurement (Pearson r = 0.84)

Correlation between serum interleukin-6 (IL-6) and serum fibrinogen levels among the study subjects. A strong positive correlation was observed between serum IL-6 and fibrinogen levels (Pearson r = 0.85), indicating concordant elevation of these two inflammatory markers (Fig. 5).

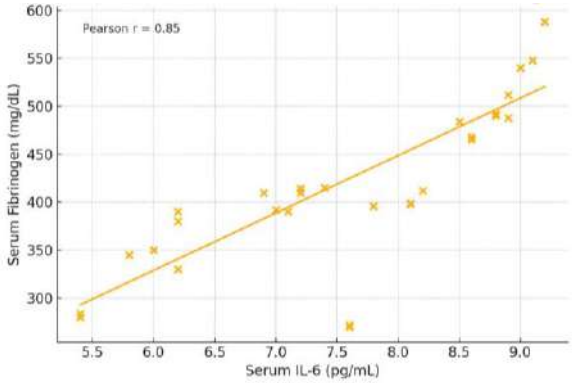


Fig. 5: Correlation between serum interleukin-6 and serum fibrinogen (Pearson r = 0.85).

DISCUSSION

MASLD has emerged as a major public health concern in India and worldwide, driven by an interplay of genetic, environmental, and lifestyle factors, with rapid urbanization, dietary transition, and sedentary behaviour accelerating its rise. Chronic low-grade hepatic inflammation, mediated in part by IL-6 and its downstream acute-phase product fibrinogen, is now recognized as central to the transition from simple steatosis to fibrosis. This study examined how these two circulating markers relate to liver stiffness and to establish non-invasive fibrosis scores (FIB-4, BARD, and APRI) in 100 subjects with MASLD.

Our cohort was almost evenly split by sex (55% women, 45% men), yet men showed significantly greater liver stiffness than women (p = 0.0176), consistent with the sex disparity in MASLD severity reported by Lonardo *et al*., who described more frequent and more severe disease recognition in men [13]. The predominance of subjects in the 41-60-year range, with a wider age dispersion among men, broadly mirrors the demographic pattern described by Hong et al. in their large MASLD cohort, although that study's subjects were on average a decade younger, possibly reflecting differences in screening thresholds and population risk-factor onset between settings [14].

Obesity was near-universal in our subjects (83% with BMI ≥25 kg/m²), and higher BMI correlated significantly with greater liver stiffness (p = 0.00004; Pearson r = 0.33). This aligns with prior work identifying elevated BMI as the most common cardiometabolic risk factor accompanying MASLD and linking obesity to both metabolic syndrome and progressive steatotic liver disease [13]. Similarly, while diabetes, dyslipidemia, and

doi: 10.21276/SSR-IIJLS.2026.12.5.18

hypertension were each present in the large majority of our subjects, only poor glycemic control (HbA1c ≥7%) — not the diagnosis of diabetes per se — showed a significant association with liver stiffness (p = 0.0012; Pearson r = 0.36). This distinction is clinically relevant: current guidance recognizes type 2 diabetes as an independent risk factor for progressive fibrosis and recommends simple serum-based scores such as FIB-4 as a first-line screen, reserving elastography for risk stratification in those who screen positive, a pathway our findings support.

Consistent with reports from Asian MASLD cohorts describing AST as a marker of fibrosis severity when substantially elevated, our subjects showed an AST-predominant transaminase pattern (88% with raised AST vs. 28% with raised ALT) [15]. Among the three fibrosis risk scores assessed, FIB-4 showed the strongest correlation with liver stiffness (Pearson r = 0.92), followed by APRI (r = 0.84) and BARD (significant at p = 0.013). This ordering mirrors the broader literature: FIB-4, originally developed and validated in HCV/HIV co-infected cohorts before being extended to MASLD, has repeatedly demonstrated strong negative predictive value for advanced fibrosis and been adopted by international liver societies as a first-line non-invasive screening tool [16-19]. Our APRI correlation with LSM was numerically stronger than that reported by Jin *et al.* in a paediatric MASLD cohort (r = 0.354) [20], and Our BARD findings are consistent with the findings reported by Cichoż-Lach *et al.* regarding the utility of the BARD score in identifying subjects unlikely to have advanced fibrosis [11].

The central findings of this study concern serum fibrinogen and IL-6. Both markers were frequently elevated (58% and 75% of subjects, respectively) and each correlated strongly with liver stiffness (r = 0.92 and r = 0.84) and with FIB-4 score, while also correlating strongly with one another (r = 0.85). This concordance is biologically coherent, since fibrinogen is an acute-phase reactant synthesized in response to IL-6 signalling, and supports earlier observations linking IL-6 elevation to hepatic inflammatory activity in MASLD. Jarrar *et al.* reported elevated IL-6 and related cytokines in patients with non-alcoholic fatty liver disease, supporting the role of inflammatory mediators in disease progression [21]. Taken together, our findings suggest that serum fibrinogen and IL-6 are closely associated with fibrosis severity and may serve as accessible adjunctive biomarkers alongside established non-invasive tools such as FIB-4, particularly in settings where transient elastography is not readily available.

LIMITATIONS

This study has limitations that temper these conclusions. It was conducted at a single centre with a modest sample size of 100 subjects, which may limit generalizability, and its cross-sectional design precludes any inference about whether fibrinogen and IL-6 elevation precedes or follows fibrosis progression. Fibrinogen and IL-6 are also non-specific acute-phase reactants that can be influenced by intercurrent infection or inflammatory conditions unrelated to the liver, which were not exhaustively excluded beyond the study's defined exclusion criteria. Larger, multicentre, longitudinal studies are needed to validate these correlations, determine cut-off values with adequate sensitivity and specificity, and clarify the incremental value these markers would add to existing non-invasive fibrosis scores in routine clinical practice.

CONCLUSIONS

Metabolic dysfunction-associated steatotic liver disease (MASLD), formerly known as NAFLD, is characterized by hepatic steatosis associated with metabolic risk factors, with chronic inflammation contributing to disease progression. This study evaluated the correlation between serum IL-6 and fibrinogen levels in MASLD patients and their association with liver stiffness measurement (LSM), an indicator of fibrosis severity. A strong, statistically significant positive correlation was observed between IL-6 and fibrinogen, suggesting a shared inflammatory pathway. Both markers were significantly associated with LSM, while higher FIB-4 and APRI scores showed parallel elevations in IL-6 and fibrinogen, supporting their relationship with hepatic inflammation and fibrosis. Diabetes, hypertension, and dyslipidemia alone were not significantly associated with LSM; however, poor glycemic control was significantly associated with increased liver stiffness. Overall, IL-6 and fibrinogen may serve as accessible, non-invasive biomarkers of fibrosis in MASLD. Their association warrants further validation through larger, multicentre, longitudinal studies to establish their clinical utility in risk stratification.

doi: 10.21276/SSR-IIJLS.2026.12.5.18

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