

Association of Systemic Immune-Inflammation Index with Metabolic Dysfunction Associated Steatotic Liver Disease (MASLD)

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Abstract

Background: Metabolic dysfunction-associated steatotic liver disease (MASLD), previously termed non-alcoholic fatty liver disease (NAFLD), represents a growing burden of chronic liver disease worldwide. Chronic low-grade inflammation is central to disease pathogenesis, but the value of systemic inflammatory biomarkers in diagnosis and risk stratification remains unclear. The systemic immune-inflammation index (SII), calculated from peripheral blood counts, has been proposed as a composite marker reflecting the balance between pro- and anti-inflammatory components. This study evaluates the diagnostic and predictive utility of SII in MASLD.

Aim of Study: To investigate the association between the systemic immune-inflammation index (SII) and non-alcoholic fatty liver disease (NAFLD) and to explore potential factors associated with elevated SII levels in individuals with NAFLD.

Patients and Methods: A matched case-control study was conducted, enrolling 174 adult Egyptian patients: 87 with ultrasound-confirmed MASLD and 87 age- and sex-matched non-MASLD controls. Participants underwent clinical assessment, laboratory testing, and abdominal ultrasound with shear-wave elastography to exclude other liver diseases and stage fibrosis non-invasively. SII was calculated as platelet count $\times$ neutrophil count / lymphocyte count. The NAFLD fibrosis score (NFS) and body mass index (BMI) were also computed. Diagnostic performance was assessed with receiver-operating characteristic (ROC) curves, and multivariate logistic regression was used to evaluate independent predictors of MASLD.

Results: Median SII values did not differ significantly between MASLD and control groups ($p = 0.997$). In ROC analysis, SII alone showed poor discriminatory ability for MASLD (area under the curve (AUC) = 0.50). In multivariate regres-

sion, however, lower SII emerged as an independent protective factor against MASLD (odds ratio = 0.99; $p = 0.037$) after adjusting for BMI and fasting blood glucose (FBG). Higher BMI and FBG were significant risk factors. A composite model incorporating SII, BMI and FBG improved diagnostic accuracy (AUC = 0.75), outperforming SII alone or traditional fibrosis scores.

Conclusion: Although SII lacks stand-alone diagnostic value for MASLD, it contributes to risk prediction when combined with metabolic variables. These findings emphasize the predominance of metabolic risk factors in early disease and highlight the potential of composite scores for non-invasive screening. Further longitudinal studies using histological or magnetic resonance imaging confirmation and broader biomarker panels are warranted to validate the role of SII in MASLD.

Key Words: Non-alcoholic fatty liver disease NAFLD – MASLD.

Introduction

METABOLIC dysfunction-associated steatotic liver disease (MASLD), formerly known as non-alcoholic fatty liver disease (NAFLD), is characterized by hepatic fat accumulation associated with metabolic risk factors, including obesity, insulin resistance, type 2 diabetes mellitus, dyslipidaemia and hypertension [1,2]. Recent consensus statements advocate a shift to MASLD terminology to emphasize the positive diagnostic criteria and metabolic underpinnings of the disease rather than the exclusion of alcohol consumption [1,3]. MASLD affects approximately a quarter of the global population and is anticipated to become the leading indication for liver transplantation [2,4]. In the updated nomenclature, MASLD requires hepatic steatosis plus at least one cardiometabolic risk factor, whereas individuals with steatosis and higher alcohol intake are classified as metabolic dysfunction-associ-

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ated alcohol-related liver disease (MetALD), and steatohepatitis is now termed metabolic dysfunction-associated steatohepatitis (MASH) [1,3].

The pathogenesis of MASLD is multifactorial. The “multiple-hit hypothesis” integrates insulin resistance, adipokine dysregulation, oxidative stress, gut microbiota alterations, and chronic low-grade inflammation [5,6]. Pro-inflammatory cytokines such as tumour necrosis factor- α (TNF- α), interleukin-6 (IL-6) and interleukin-1 β (IL-1 β) drive hepatic injury and fibrosis [5,7]. Traditional inflammatory markers, including C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), provide limited insight into the dynamic balance between immune cell subsets. The systemic immune-inflammation index (SII) has emerged as a composite biomarker derived from routine blood counts. Calculated as platelet count multiplied by neutrophil count divided by lymphocyte count, SII reflects the interplay between innate and adaptive immune responses and has prognostic value in cardiovascular disease and malignancies [8,10].

Few studies have evaluated SII in MASLD. Reports from Asian cohorts suggested elevated SII correlates with advanced fibrosis and liver stiffness, but data remain inconsistent [11,12]. Moreover, whether SII can aid early diagnosis or risk stratification in MASLD populations outside East Asia is unclear. This study aimed to assess the diagnostic and predictive performance of SII in Egyptian adults with MASLD and to compare it with established clinical and biochemical indices.

Patients and Methods

Study design and participants:

This matched case-control study was conducted at Misr University for Science and Technology (MUST) Hospital. Adult participants (>18 years) presenting to the internal medicine clinic between 2024 and 2025 were screened. Cases were patients with MASLD diagnosed by abdominal ultrasound and shear-wave elastography demonstrating steatosis with or without fibrosis. Controls were age- (± 3 years) and sex-matched individuals without evidence of hepatic steatosis on ultrasound. Exclusion criteria included significant alcohol consumption (>20g/day for men, >10g/day for women), viral hepatitis, autoimmune liver disease, Wilson disease, haemochromatosis, hepatotoxic medication use, malignancy, pregnancy and severe systemic illness.

Data collection:

All participants underwent physical examination, anthropometric measurements (weight,

height, BMI), and laboratory testing. Fasting blood samples measured complete blood count, fasting blood glucose (FBG), liver function tests (ALT, AST, ALP, bilirubin), lipid profile (triglycerides, LDL-C, HDL-C), and serologic markers for viral hepatitis and autoimmune liver disease. The SII was calculated as platelet count $\times$ neutrophil count / lymphocyte count. The NAFLD fibrosis score (NFS) was computed using age, BMI, impaired fasting glucose or diabetes status, AST/ALT ratio, platelet count and albumin levels. Ultrasonography assessed steatosis severity, while shear-wave elastography quantified liver stiffness; values >8 kPa suggested significant fibrosis.

Statistical analysis:

Descriptive statistics summarized continuous variables using mean $\pm$ standard deviation or median (interquartile range) depending on distribution. Categorical variables were expressed as counts and percentages. Independent *t*-tests or Mann–Whitney U tests compared continuous data between MASLD cases and controls; chi-square or Fisher’s exact tests compared categorical variables. ROC curves evaluated the discriminatory ability of SII and other indices for MASLD; the Youden index identified optimal cut-off values. Multivariate logistic regression assessed independent predictors of MASLD, including SII, BMI and FBG. Statistical significance was set at $p < 0.05$. Analyses were performed using SPSS version 26.

Results

Baseline characteristics:

The study included 87 patients with MASLD and 87 matched controls. The groups were similar in age and sex distribution (mean age $\approx$ 50 years; $\sim$ 60% female). MASLD cases had significantly higher BMI (mean $\approx$ 32kg/m² vs. 27kg/m² in controls), higher FBG and triglyceride levels, and lower HDL-C. Most MASLD patients ($\approx$ 96%) had mild fibrosis (F0–F1) by shear-wave elastography; only a small proportion exhibited significant fibrosis (F2 or higher).

This case-control study was conducted on 174 subjects (87 patients with NAFLD and 87 non-NAFLD control). The demographic information of studied groups is presented in (Table 1). There was no significant difference between the two groups regarding age and sex ($p=0.12$ and $p=1.0$, respectively).

Table (1) summarizes the demographic data for the MASLD and control groups. For completeness, the original table from the thesis is reproduced below.

Table (1): Demographic data among NAFLD patients.

	NAFLD (N=87)	Non- FAFLD (N=87)	Test of Significance	p- value
<i>Age (years):</i>				
Mean $\pm$ SD	43.93 $\pm$ 8.21	41.93 $\pm$ 9.0	$t=1.53$	$p=0.12$
<i>Sex, n (%):</i>				
Males	86 (98.85%)	87 (100%)	FET	$p=1.0$
Females	1 (1.15%)	0 (1.15%)		

Data is expressed as Mean $\pm$ SD and frequency (percentage).

NAFLD: Non-alcoholic fatty liver disease.

FET: Fisher-exact test. t : Student's t -test.

Table (1) Demographic data among MASLD and non-MASLD participants.

Imaging findings:

Abdominal ultrasound demonstrated that all MASLD patients had bright or fatty liver parenchyma, whereas none of the controls did; moreover, 17.2% of MASLD patients had hepatomegaly compared with 0 % of controls [4]. These findings are summarised in Table (2). Shear wave elastography (SWE) provided quantitative assessment of liver stiffness and fibrosis stage. The mean liver stiffness among MASLD patients was 6.51 ± 0.63 kPa and the mean shear-wave velocity was 1.44 ± 0.10 m/s; 96.55 % of patients were categorised as having mild fibrosis (F0–F1), 2.3% had significant fibrosis (F2) and 1.15% had severe fibrosis (F3) as shown in the thesis (1955†screenshot). These data are presented in Table (3). According to recent AASLD practice guidance, conventional B-mode ultrasound has low sensitivity for detecting mild steatosis and provides only a subjective semi-quantitative assessment, while controlled attenuation parameter measured with transient elastography offers only a semiquantitative point-of-care assessment and the cut-points for SWE are not well validated [3].

Table (2): Abdominal ultrasound findings among MASLD and control groups.

Finding	MASLD (N = 87)	Control (N = 87)	p- value
Bright/fatty liver, n (%)	87 (100 %)	0 (0%)	$<0.001^{**}$
Hepatomegaly, n (%)	15 (17.2 %)	0 (0%)	0.001^{**}

Table (3): Shear wave elastography findings among MASLD patients.

Parameter	Value
kPa (mean $\pm$ SD)	6.51 ± 0.63 kPa
Velocity (mean $\pm$ SD)	1.44 ± 0.10 m/s
Mild fibrosis, n (%)	84 (96.55%)
Significant fibrosis, n (%)	2 (2.3%)
Severe fibrosis, n (%)	1 (1.15%)

Shear Wave Elastography (SWE) is used to evaluate the stiffness changes in the liver associated with fibrosis, where US waves are pulsed into the liver to generate a shear wave that is measured by assessing tissue displacement. SWE is used to discriminate between different stages of fibrosis as: (absent/mild fibrosis “FO-F1”); (significant fibrosis “F2”); (severe fibrosis “F3”) and (cirrhosis “F4”) among patients with fatty liver based on the velocity and Kpa values, (Fig. 1A,B).

The mean kPa of all the patients was 6.51 ± 0.63 and the mean velocity was 1.44 ± 0.10 . From all the patients with fatty liver, (96.55%) were found to have mild fibrosis, (2.3%) were found to have significant fibrosis and (1.15%) of them had severe fibrosis, as presented in (Table 4, Fig. 2).

SII and other inflammatory markers:

Median SII values did not differ significantly between MASLD and control groups ($p \approx 0.997$). Likewise, neutrophil counts and platelet counts showed no meaningful differences. CRP and ESR levels were not measured. The lack of difference suggests systemic inflammation may not be prominent in early MASLD within this cohort. Fig. (3) illustrates the distribution of SII values between the two groups using a box-plot. It demonstrates considerable overlap and the absence of a significant difference, supporting the text description.

The comparison of SII between the NAFLD and non-NAFLD groups showed no significant difference ($p=0.997$, $z=-0.002$), as shown in (Fig. 3).

ROC analysis:

In ROC analysis, SII alone yielded an AUC of ≈ 0.50 , indicating no discriminatory power for identifying MASLD. This is visualised in Fig. (4), which shows the ROC curve for SII alone; the curve lies close to the 45° reference line, reflecting poor discrimination. The NAFLD fibrosis score had modest performance (AUC ≈ 0.66). The optimal SII cut-off derived from the Youden index (≈ 270) did not meaningfully differentiate cases from controls. Combining SII with BMI and FBG improved discrimination substantially, yielding an AUC of ≈ 0.75 ($p<0.001$). Fig. (5) displays the ROC curve for this composite model, highlighting improved sensitivity and specificity. This composite model outperformed SII alone and the NFS.

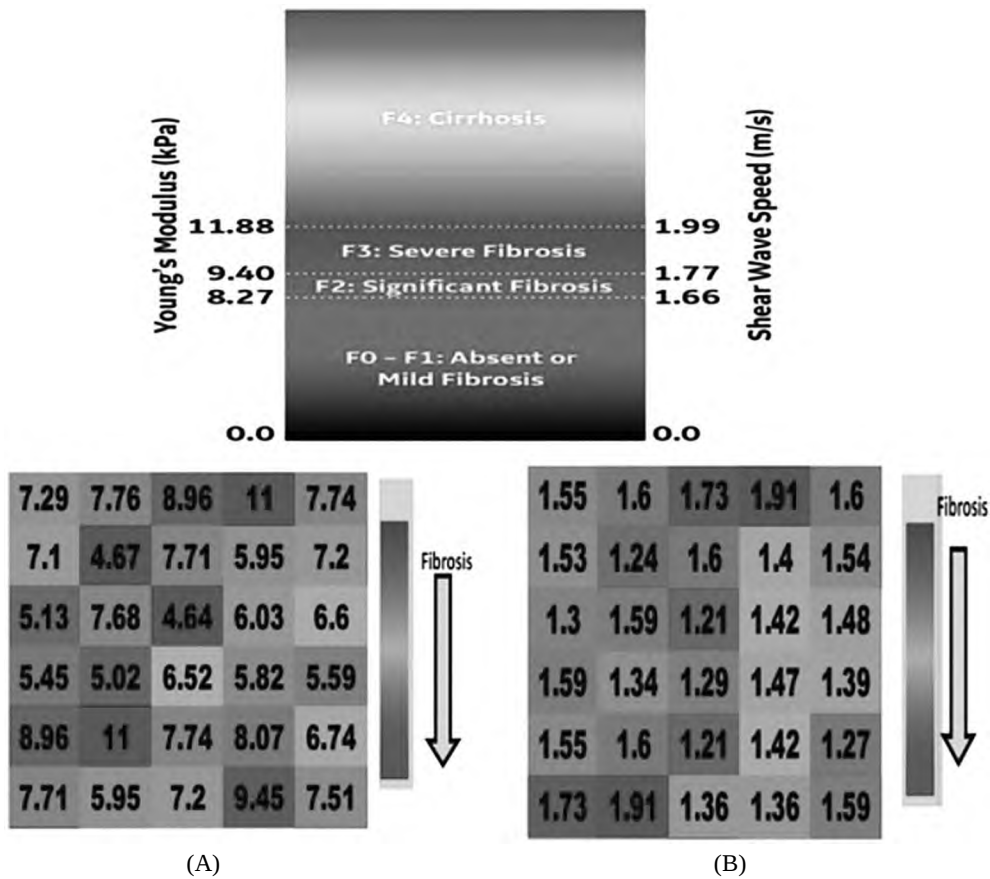


Fig. (1): (A) Young's Modulus values among the studied patients; (B) Shear wave speed values among the studied patients.

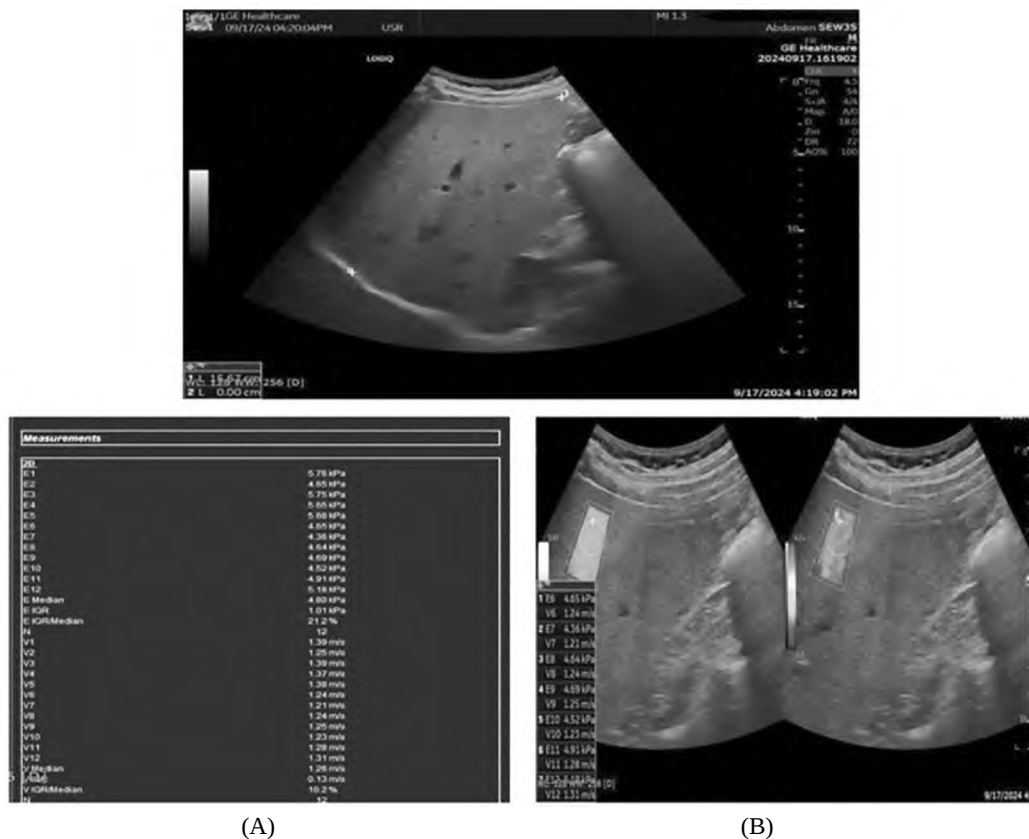
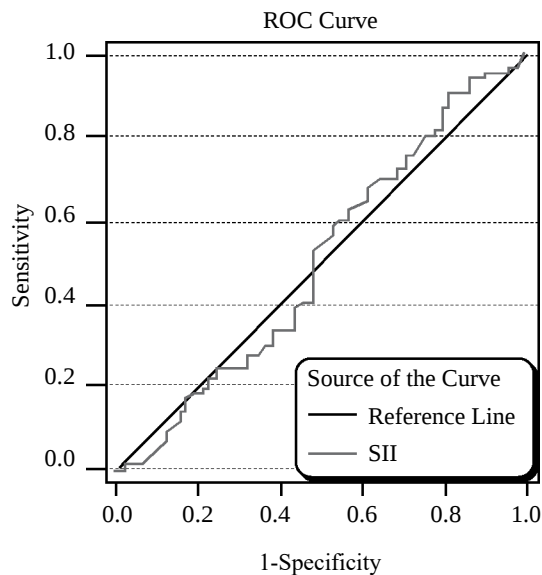


Fig. (2): The figure shows the (A) U/S and (B) SWE results of a case with mild fibrosis.

Table (4): Shear wave elastography finding of the studied patients.

All cohort (N=87)		
<i>kPa:</i>		
Mean ± SD	6.51±0.63	
<i>Velocity:</i>		
Mean ± SD	1.44±0.10	
<i>SWE score, n (%):</i>	n	%
Mild fibrosis	84.0	96.55
Significant fibrosis	2.0	2.3
Severe fibrosis	1.0	1.15

Data is expressed as Mean $\pm$ SD, or frequency (percentage).
SWE: Shear wave elastography.



Figs. (4): ROC curve of SII to discriminate between NAFLD and non-NAFLD groups.

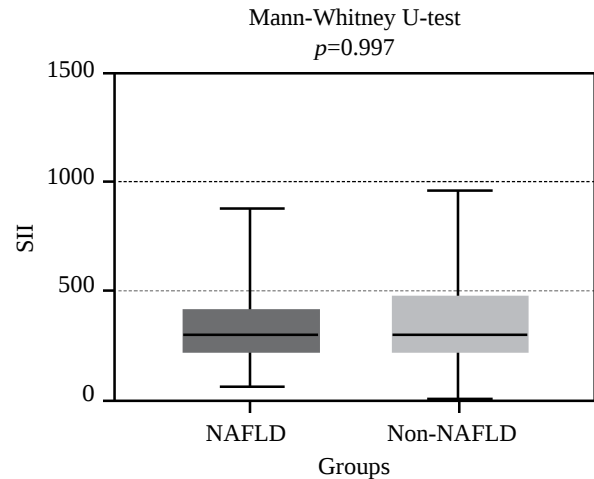
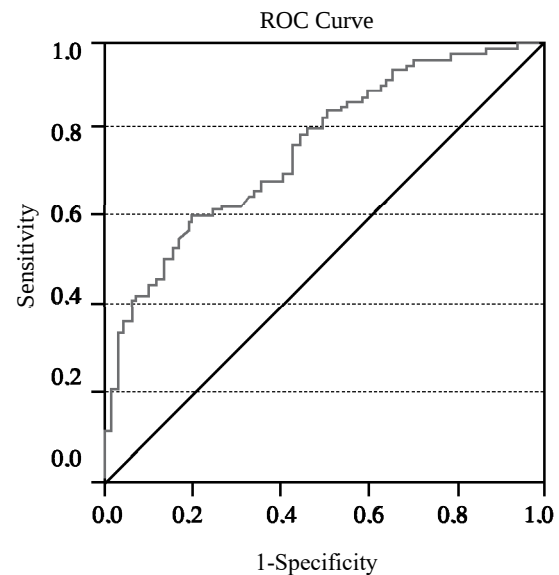


Fig. (3): Comparison of SII between the NAFLD and non-NAFLD groups.



Figs. (5): ROC curve of the combination (SII+BMI+FBG) to discriminate between NAFLD and non-NAFLD groups.

Regression analysis:

In multivariate logistic regression adjusting for BMI and FBG, lower SII values were independently associated with a reduced likelihood of MASLD (OR = 0.99; $p = 0.037$). BMI and FBG were significant positive predictors of MASLD (OR = 1.17 and OR = 1.01, respectively). These findings suggest that metabolic factors are dominant determinants of MASLD risk, whereas SII exerts only a modest, perhaps protective influence when considered alongside metabolic variables. Table (5) presents the regression coefficients, standard errors, Wald

statistics and odds ratios with 95% confidence intervals. The reproduced table from the thesis is included below.

Correlation analysis:

In addition to regression modelling, the relationships between BMI, FBG and SII were explored using correlation matrices. Fig. (6) depicts the Pearson correlation coefficients among these variables in patients with MASLD. Correlations were generally weak ($r = 0.10$ – 0.14), highlighting that BMI and FBG are largely independent of SII in this cohort.

Table (5): Multi-variate logistic regression analysis for prediction of NAFLD.

Variable	B	Std. error	Wald	df	p-value	Exp(B)	95% Confidence interval for Exp(B)	
							Lower bound	Upper bound
SII	-.002	.001	4.343	1	0.037*	.998	.996	1.000
BMI	.160	.043	14.130	1	<.000*	1.174	1.080	1.276
FBG	.044	.013	11.218	1	0.001*	1.045	1.019	1.073

B: Regression coefficient,

*: Significant ($p < 0.05$).

FBG: Fasting blood glucose.

BMI: Body mass index.

SII : Systemic immune inflammatory index.

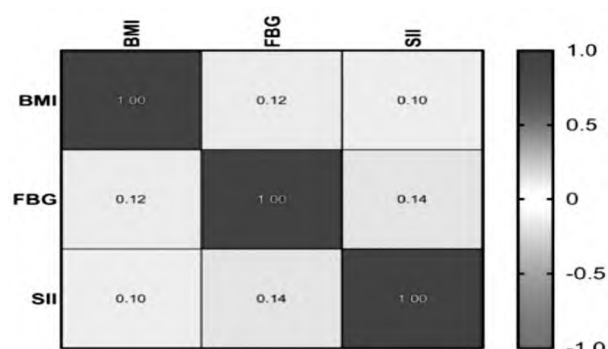


Fig. (6): Correlation matrix of SII, BMI and FBG among NAFLD patients.

Discussion

This matched case-control study examined the utility of the systemic immune-inflammation index as a non-invasive marker for MASLD in Egyptian adults. Contrary to some prior reports from Asian cohorts, [11,12] SII did not differ between MASLD patients and controls and had poor stand-alone diagnostic performance. Nevertheless, SII contributed to risk prediction when combined with BMI and fasting glucose, implying that inflammatory dysregulation may modulate, but not dominate, early MASLD pathogenesis [5,6,8].

Several explanations may account for the weak association between SII and MASLD observed here. First, most cases had mild steatosis and minimal fibrosis; systemic inflammatory markers may rise only in advanced stages [13,14]. Second, ethnic differences in immune response and genetic polymorphisms (e.g., PNPLA3) could influence inflammatory profiles [15,16]. Third, SII is influenced by a variety of conditions, including infections and metabolic syndrome, which may confound its relationship with MASLD [8,10,17].

The study confirms the central role of metabolic risk factors (BMI, hyperglycaemia) in MASLD [1,2,5] and supports the rationale for the recent ter-

minological shift from NAFLD to MASLD [1,3]. The composite model incorporating SII highlights the potential value of multi-parameter algorithms using readily available clinical and laboratory data to identify individuals at risk for MASLD in resource-limited settings [18,19].

Strengths and limitations:

Strengths of this study include its matched case-control design, use of non-invasive imaging (ultrasound and shear-wave elastography) alongside biochemical scoring (NFS), and integration of the MASLD nomenclature. Limitations include the absence of histological or magnetic resonance imaging confirmation, the cross-sectional assessment preventing causal inference, and the single-centre setting limiting generalizability. Additionally, dietary factors, physical activity and genetic predisposition were not assessed.

Conclusions:

In this Egyptian cohort, the systemic immune-inflammation index failed to differentiate MASLD patients from healthy controls and demonstrated poor diagnostic accuracy. However, when combined with BMI and fasting glucose, SII contributed modestly to risk prediction. These findings underscore the importance of metabolic dysfunction in early MASLD and suggest that SII may be most valuable as part of a multi-marker algorithm rather than as a stand-alone biomarker. Larger, longitudinal studies with histological endpoints and broader biomarker panels are needed to validate these observations.

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Funding and Conflict of Interest:

No funding was received for this study. The author declares no conflicts of interest.

References

- 1- ESLAM M., NEWSOME P.N., SARIN S.K., et al.: A new definition for metabolic dysfunction-associated fatty liver disease: An international expert consensus statement. *Lancet Gastroenterol Hepatol.*, 5 (5): 484-498, 2020. doi: 10.1016/S2468-1253(19)30313-4.
- 2- YOUNOSSI Z.M., KOENIG A.B., ABDELATIF D., FAZEL Y., HENRY L. and WYMER M.: Global epidemiology of non-alcoholic fatty liver disease—Meta-analytic assessment of prevalence, incidence, and outcomes. *Hepatology*, 64 (1): 73-84, 2016. doi: 10.1002/hep.28431.
- 3- RINELLA M.E., LAZARUS J.V., RATZIU V., et al.: A multisociety Delphi consensus statement on new fatty liver disease nomenclature. *J. Hepatol.*, 79 (4): 1099-1111, 2023. doi:10.1016/j.jhep.2023.05.010.
- 4- ESTES C., ANSTEE Q.M., ARIAS-LOSTE M.T., et al.: Modeling NAFLD disease burden in China, France, Germany, Italy, Japan, Spain, United Kingdom, and United States for the period 2016-2030. *J. Hepatol.*, 69 (4): 896-904, 2018. doi:10.1016/j.jhep.2018.05.036.
- 5- BUZZETTI E., PINZANI M. and TSOCHATZIS E.A.: The multiple-hit pathogenesis of non-alcoholic fatty liver disease (NAFLD). *Metabolism.*, 65 (8): 1038-1048, 2016. doi:10.1016/j.metabol.2015.12.012.
- 6- TILG H. and MOSCHEN A.R.: Evolution of inflammation in nonalcoholic fatty liver disease: The multiple parallel hits hypothesis. *Hepatology*, 52 (5): 1836-1846, 2010. doi:10.1002/hep.24001.
- 7- PAROLA M. and PINZANI M.: Liver fibrosis: Pathophysiology, pathogenetic targets and clinical issues. *Mol. Aspects Med.*, 65: 37-55, 2019. doi:10.1016/j.mam.2018.09.002.
- 8- HU B., YANG X.R., XU Y., et al.: Systemic immune-inflammation index predicts prognosis of patients after curative resection for hepatocellular carcinoma. *Clin. Cancer Res.*, 20 (23): 6212-6222, 2014. doi:10.1158/1078-0432.CCR-14-0442.
- 9- FEST J., RUITER R., MULDER M., et al.: The systemic immune-inflammation index is associated with an increased risk of cancer mortality in the general population. *Eur. J. Cancer*, 124: 91-99, 2019. doi:10.1016/j.ejca.2019.10.014.
- 10- YANG R., CHANG Q., MENG X., GAO N. and WANG W.: Prognostic value of systemic immune-inflammation index in cancer: A meta-analysis. *J. Cancer*, 9 (18): 3295-3302, 2018. doi:10.7150/jca.25691.
- 11- ZHOU Y.J., ZHANG Z., ZHENG K.I., et al.: Association between systemic immune-inflammation index and the severity of NAFLD: A large population-based study. *Liver Int.*, 40 (12): 2868-2878, 2020. doi:10.1111/liv.14612.
- 12- FIDAN E., ISIK O., YILDIZ B., et al.: Systemic immune-inflammation index is associated with liver fibrosis in patients with non-alcoholic fatty liver disease. *Clin. Res. Hepatol. Gastroenterol.*, 45 (5): 101603, 2021. doi:10.1016/j.clinre.2020.101603.
- 13- YONEDA M., IMAJO K., EGUCHI Y., et al.: Noninvasive scoring systems in predicting histological severity and future risk of nonalcoholic fatty liver disease. *Hepatol. Res.*, 48 (10): 822-830, 2018. doi:10.1111/hepr.13195.
- 14- PETTA S., DI MARCO V., CAMMÀ C., et al.: Systemic inflammation and liver disease progression in patients with nonalcoholic fatty liver disease. *PLoS One*, 8 (10): e73054, 2013. doi:10.1371/journal.pone.0073054.
- 15- SOOKOIAN S. and PIROLA C.J.: Meta-analysis of the influence of I148M variant of patatin-like phospholipase domain-containing 3 gene (PNPLA3) on the susceptibility and histological severity of nonalcoholic fatty liver disease. *Hepatology*, 53 (6): 1883-1894, 2011. doi:10.1002/hep.24283.
- 16- ESLAM M., VALENTI L. and ROMEO S.: Genetics and epigenetics of NAFLD and NASH: Clinical impact. *J. Hepatol.*, 68 (2): 268-279, 2018. doi:10.1016/j.jhep.2017.09.003.
- 17- FARIA S.S., FERNANDES P.C. Jr., SILVA M.J., et al.: The neutrophil-to-lymphocyte ratio: A narrative review. *E cancer medical science*, 10: 702, 2016. doi:10.3332/ecancer.2016.702.
- 18- COREY K.E., VUPPALANCHI R., CUMMINGS O.W., et al.: Development and validation of a scoring system to predict histologic fibrosis in NAFLD: The NAFLD fibrosis score. *Hepatology*. 2007;45(4):846-854. doi:10.1002/hep.21599.
- 19- ANGULO P., HUI J.M., MARCHESINI G., et al.: The NAFLD fibrosis score: A noninvasive system that identifies liver fibrosis in patients with NAFLD. *Hepatology*, 45 (4): 846-854, 2007. doi:10.1002/hep.21496.

ارتباط مؤشر الالتهاب المناعي الجهازى بمرض الكبد الدهنى المرتبط باضطراب التمثيل الغذائى (MASLD)

إجريت هذه الدراسة لتقييم دور مؤشر الالتهاب المناعى الجهازى فى تشخيص مرض الكبد الدهنى المرتبط باضطراب التمثيل الغذائى، وشملت ١٧٤ مريضاً مصرياً قُسموا بالتساوى إلى مجموعة MASLD ومجموعة ضابطة. خضع جميع المشاركين لتقييمات سريرية ومعملية شاملة، واستخدم الباحثون الموجات فوق الصوتية وإيلاستوجرافى لتقدير درجة التليف. حُسب المؤشر من حاصل ضرب الصفائح الدموية والعدلات مقسوماً على اللمفاويات، ولم يظهر اختلافاً جوهرياً بين المرضى والأصحاء؛ لكن تحليل الانحدار أوضح أن انخفاض المؤشر يرتبط بحماية خفيفة، فى حين أن زيادة مؤشر كتلة الجسم وسكر الدم الصائم كانت عوامل خطيرة واضحة. عند دمج المؤشر مع هذين العاملين زادت دقة النموذج التشخيصى، حيث بلغ منحنى ROC للنموذج المركب حوالى ٠,٧٥٢. وتشير النتائج إلى أن الالتهاب يلعب دوراً ثانوياً فى المراحل المبكرة للمرض، وأن الخلل الأيضى يظل المحرك الرئيس لتطور MASLD، مما يجعل الاعتماد على المؤشر كأداة تشخيصية مستقلة غير موصى به، وإن كان يمكن استخدامه ضمن مجموعة مؤشرات لتحديد المخاطر. توصى الدراسة بإجراء بحوث مستقبلية للتأكد من هذه النتائج باستخدام عينات أكبر وتقنيات أكثر دقة.