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The prevalence of positive C-reactive protein and elevated erythrocyte sedimentation rate among individuals with type 2 diabetes mellitus and non-alcoholic fatty liver disease

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6. BSTRACT

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Chronic inflammation plays a central role in both type 2 diabetes mellitus (T2DM) and non-alcoholic fatty liver disease (NAFLD). This study evaluated the association of C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) among patients with T2DM, NAFLD, and concurrent disease. In this cross-sectional study, 150 participants were equally divided into three groups: T2DM or NAFLD, and concurrent T2DM and NAFLD. Demographic and clinical data were recorded, and CRP and ESR levels were measured. Statistical analyses included ANOVA, Kruskal–Wallis, Chi-square tests, and logistic regression models adjusted for age and gender by reporting odds ratios (ORs) and 95% confidence intervals (CIs). A p-value <0.05 was considered statistically significant. ESR levels differed significantly among groups (p <0.001), with the highest values observed in the T2DM+NAFLD group. Adjusted logistic regression showed that patients with T2DM+NAFLD had a 3.8-fold increased odds of elevated ESR compared to NAFLD alone (95% CI: 1.345–10.759, p=0.012). CRP positivity did not significantly differ between groups; however, categorical CRP analysis revealed significant distribution differences (p <0.001). Coexistence of T2DM and NAFLD is associated with greater systemic inflammatory burden, particularly reflected by elevated ESR. ESR may serve as a more sensitive inflammatory marker than CRP in patients with combined metabolic disorders.

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1. Introduction

Type 2 diabetes mellitus (T2DM) represents a major global health challenge, characterized by chronic hyperglycemia resulting from insulin resistance and progressive pancreatic β-cell dysfunction [1,2]. Its prevalence has risen dramatically over recent decades, affecting an estimated 10.5% of adults worldwide, with projections indicating further increases due to lifestyle transitions, urbanization, and aging populations, which resulted in further complications [3–5]. Beyond its metabolic derangements, T2DM is increasingly recognized as a pro-inflammatory state, with low-grade systemic inflammation contributing to the onset and progression of both microvascular and macrovascular complications [6–8]. Key inflammatory biomarkers, such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), have been shown to correlate with insulin resistance, endothelial dysfunction, and cardiovascular risk, providing clinically relevant insight into the inflammatory milieu of T2DM patients [9–11].

Non-alcoholic fatty liver disease (NAFLD) is the hepatic manifestation of metabolic dysfunction, affecting approximately 25–30% of the global adult population and up to 70% of individuals with T2DM [12,13]. NAFLD encompasses a spectrum from simple steatosis to non-alcoholic steatohepatitis (NASH), fibrosis, and cirrhosis. Its pathogenesis is multifactorial, driven primarily by insulin resistance, lipotoxicity, oxidative stress, dysregulated adipokine secretion, and chronic systemic inflammation [12,14,15]. The interplay between metabolic dysregulation and inflammatory processes not only contributes to hepatocellular injury but also amplifies cardiovascular risk, particularly in patients with coexisting T2DM [16,17].

Although inflammation is a key feature of both T2DM and NAFLD, data on the prevalence of elevated inflammatory markers, particularly CRP and ESR, in patients with both conditions remain limited [18]. CRP indicates systemic inflammation and is associated with insulin resistance, atherogenesis, and liver injury, while ESR provides a complementary, nonspecific measure of chronic inflammatory activity [19,20]. Assessing the prevalence and magnitude of these markers in patients with T2DM and NAFLD provides valuable insights into the inflammatory burden and may inform risk stratification, early intervention strategies, and therapeutic monitoring. In this context, the present study aims to evaluate the prevalence of positive CRP and elevated ESR among individuals with T2DM and NAFLD.

2. Materials and Methods

2.1 *Study Design and Population*

This cross-sectional study was conducted on patients diagnosed with T2DM or NAFLD, and concurrent T2DM and NAFLD. A total of 150 participants were enrolled and equally allocated into three groups: T2DM alone (n = 50), NAFLD alone (n = 50), and combined T2DM+NAFLD (n = 50). The sample consisted of 150 patients selected through a convenience sampling method from individuals referred to Amir al-Momenin Hospital, Zabol, Iran. The study protocol was approved by the Ethics Committee of Zabol University of Medical Sciences, Zabol, Iran (IR.ZBMU.REC.1402.103) and all individuals consented to participate in the study.

For the T2DM group, patients were required to have a confirmed diagnosis of diabetes defined as fasting blood sugar (FBS) ≥126 mg/dL on two separate occasions or glycated hemoglobin (HbA1c) ≥6.5% [21]. For the NAFLD group, diagnosis was established based on ultrasonographic or other imaging findings consistent with fatty liver disease, after exclusion of other causes such as significant alcohol consumption or viral hepatitis. Participants in the combined T2DM+NAFLD group fulfilled the diagnostic criteria for both conditions. All diagnoses were confirmed by specialist physicians based on established clinical and laboratory criteria. Patients were excluded if they had active infections, systemic inflammatory diseases (e.g., rheumatologic disorders), active malignancy, or had used corticosteroids or potent anti-inflammatory medications within the two weeks prior to enrollment.

2.2 *Data Collection*

Demographic and clinical data including age, gender, and body mass index (BMI), were recorded. Blood samples were obtained under standardized conditions and analyzed using validated laboratory kits in the central laboratory of Amir al-Momenin Hospital, Zabol, Iran. Inflammatory markers assessed included CRP and ESR. CRP levels were evaluated both as binary (positive/negative) and categorical variables (+1, +2, +3). ESR values were recorded as continuous variables (mm/hr) and categorized as normal or elevated according to laboratory reference ranges.

2.3 *Statistical Analysis*

Data were analyzed using SPSS Statistics, version 21.0 (IBM Corp., Armonk, NY, USA). Continuous variables were presented as mean ± standard deviation (SD), while categorical variables were expressed as frequency and percentage. Normality of continuous variables was assessed using the Shapiro–Wilk test. For normally distributed variables, one-way analysis of variance (ANOVA) was applied to compare means across the three groups. For non-normally distributed variables, the Kruskal-Wallis test was used, followed by pairwise comparisons with the Mann-Whitney U test when appropriatex. Post-hoc comparisons for normally distributed variables were performed using Tukey's test. Categorical variables were compared using the Chi-square test. Pearson correlation analysis was conducted

to assess the association between ESR and CRP levels. Additionally, unadjusted and age- and gender-adjusted logistic regression models were performed to evaluate the association between disease groups and inflammatory marker abnormalities, with results reported as odds ratios (ORs) and 95% confidence intervals (CIs). A p-value <0.05 was considered statistically significant in all analyses.

3. Results

The mean age of T2DM group, NAFLD, and T2DM+NAFLD, were 37.06±8.33 years, 47.92±9.54 years, and 53.06±9.05 years, respectively (p<0.001). Gender distribution was relatively balanced across all groups, with no statistically significant differences observed (p=0.57). Gender distribution was comparable among groups (p = 0.57), with a slightly higher proportion of males in the DM+NAFLD group (60%). Body mass index (BMI) did not significantly differ between groups (p = 0.78), and all groups had a mean BMI around 25 kg/m².

CRP positivity did not significantly differ across groups (p = 0.31), with prevalence rates of 64% in DM, 62% in NAFLD, and 50% in DM+NAFLD. In contrast, ESR levels showed a highly significant overall difference (p <0.001), with mean ESR progressively increasing from DM (18.22 ± 7.77 mm/hr) to NAFLD (21.12 ± 8.51 mm/hr) and DM+NAFLD (28.18 ± 13.32 mm/hr). Although ESR abnormality was more frequent in the DM+NAFLD group (36%) compared with DM (30%) and NAFLD (20%), this difference did not reach statistical significance (p = 0.2) (Table 1).

While binary CRP positivity was not statistically different, detailed categorical analysis demonstrated marked heterogeneity ($\chi^2 = 43.613$, $p < 0.001$). The DM group was characterized predominantly by +1 CRP levels (64%), whereas the NAFLD group showed a broader distribution including +2 levels (14%). Notably, the DM+NAFLD group demonstrated a substantial shift toward higher CRP categories (+2 in 38% and +3 in 2%), despite a lower overall binary positivity rate (Table 2).

According to the level of ESR, no significant difference was observed between DM and NAFLD groups (p = 0.12). However, ESR was significantly higher in the DM+NAFLD group compared to both DM (p <0.001) and NAFLD (p = 0.01). Median ESR values followed a progressive pattern (DM < NAFLD < DM+NAFLD) (Table 3, Figure 1). CRP positivity prevalence did not significantly differ across groups ($\chi^2 = 2.364$, p = 0.31). Similarly, ESR abnormality prevalence was not statistically different ($\chi^2 = 3.195$, p = 0.2), although numerically higher in the DM+NAFLD group (36%) (Table 4).

In unadjusted models, no significant associations were observed between disease groups and CRP positivity. Compared with NAFLD, DM showed an OR of 1.126 (95% CI: 0.499–2.543, p = 0.78), while DM+NAFLD showed an OR of 0.633 (95% CI: 0.285–1.407, p = 0.26). These findings remained non-significant after adjustment for age and gender. Similarly, no significant association was found when comparing DM+NAFLD with DM (adjusted OR = 0.695, p = 0.51). Overall, CRP positivity did not independently discriminate between disease groups (Table 5). For ESR abnormality, the unadjusted model demonstrated a borderline association between DM+NAFLD and NAFLD (OR = 2.500, 95% CI: 0.991–6.307, p = 0.052). After adjustment for age and gender, this association became statistically significant, with DM+NAFLD showing a 3.804-fold increased odds of abnormal ESR compared to NAFLD alone (95% CI: 1.345–10.759, p = 0.01). When comparing DM+NAFLD with DM, the adjusted OR was 2.926 (95% CI: 0.879–9.737), approaching statistical significance (p = 0.08). In contrast, DM alone was not significantly associated with ESR abnormality compared to NAFLD in either unadjusted or adjusted models (Table 5).

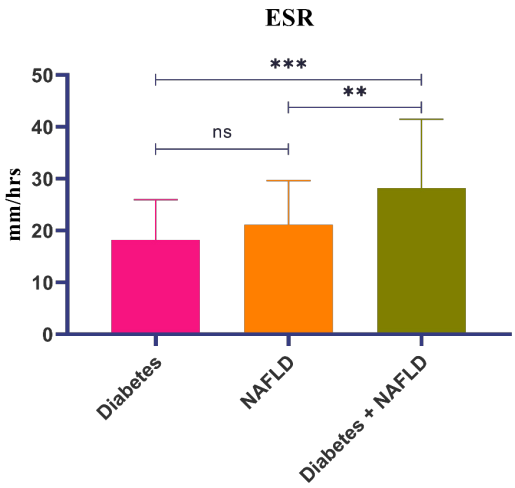

Figure 1. Prevalence of Elevated ESR level Across Study Groups. Erythrocyte Sedimentation Rate (ESR); Type 2 Diabetes Mellitus (T2DM), Non-Alcoholic Fatty Liver Disease (NAFLD).

Table 1. Baseline Demographic, Clinical Characteristics, and Inflammatory Markers Across Study Groups

Variable	DM (n=50)	NAFLD (n=50)	DM+NAFLD (n=50)	p-value
Age (years)	37.06 ± 8.33	47.92 ± 9.54	53.06 ± 9.05	<0.001*
Male, n (%)	25 (50.0%)	26 (52.0%)	30 (60.0%)	0.57***
BMI (kg/m²)	25.66 ± 3.80	25.11 ± 3.63	25.31 ± 4.45	0.78*
CRP Positive, n (%)	32 (64.0%)	31 (62.0%)	25 (50.0%)	0.31***
ESR (mm/hr)	18.22 ± 7.77	21.12 ± 8.51	28.18 ± 13.32	<0.001**
Elevated ESR, n (%)	15 (30.0%)	10 (20.0%)	18 (36.0%)	0.20***

Data presented as mean ± SD for continuous variables and n (%) for categorical variables. Statistically significant ($p < 0.05$). ANOVA. **Kruskal-Wallis. ***Chi-square. C-Reactive Protein (CRP); Erythrocyte Sedimentation Rate (ESR); Type 2 Diabetes Mellitus (T2DM), Non-Alcoholic Fatty Liver Disease (NAFLD).

Table 2. Distribution of CRP Categories Among Patients with T2DM, NAFLD, and Combined Disease

CRP Level	DM (n=50)	NAFLD (n=50)	DM+NAFLD (n=50)	*χ^2	p-value
Negative	18 (36%)	19 (38%)	25 (50%)	43.613	< 0.001
+1	32 (64%)	24 (48%)	5 (10%)		
+2	0 (0%)	7 (14%)	19 (38%)		
+3	0 (0%)	0 (0%)	1 (2%)		

*Chi-square test. Statistically significant ($p < 0.05$). C-Reactive Protein (CRP); Erythrocyte Sedimentation Rate (ESR); Type 2 Diabetes Mellitus (T2DM), Non-Alcoholic Fatty Liver Disease (NAFLD).

Table 3. Post-hoc Pairwise Comparisons of Erythrocyte Sedimentation Rate (ESR) Between Disease Groups

Comparison	Group 1 Median (IQR)	Group 2 Median (IQR)	Statistics*	p-value
DM *vs.* NAFLD	18.0 (12.0-24.8)	19.5 (15.0-26.8)	1025	0.12
DM *vs.* DM+NAFLD	18.0 (12.0-24.8)	24.5 (16.2-38.0)	708	<0.001
NAFLD *vs* DM+NAFLD	19.5 (15.0-26.8)	24.5 (16.2-38.0)	881	0.01

* Mann-Whitney U. Statistically significant ($p < 0.05$). C-Reactive Protein (CRP); Erythrocyte Sedimentation Rate (ESR); Type 2 Diabetes Mellitus (T2DM), Non-Alcoholic Fatty Liver Disease (NAFLD).

Table 4. Prevalence of CRP Positivity and ESR Abnormality Across Study Groups

Group		Number	Prevalence (%)	*χ^2	p-value
CRP Positive	T2DM	32/50	64.0%	2.364	0.31
	NAFLD	31/50	62.0%		
	NAFLD+T2DM	25/50	50.0%		
Elevated ESR	T2DM	15/50	30.0%	3.195	0.20
	NAFLD	10/50	20.0%		
	NAFLD+T2DM	18/50	36.0%		

*Chi-square test. Statistically significant ($p < 0.05$). C-Reactive Protein (CRP); Erythrocyte Sedimentation Rate (ESR); Type 2 Diabetes Mellitus (T2DM), Non-Alcoholic Fatty Liver Disease (NAFLD).

Table 5. Unadjusted and Age- and Gender-Adjusted Logistic Regression Analysis of CRP Positivity and ESR Abnormality

Outcome	Comparison	Unadjusted OR (95% CI)	p-value	Age and gender adjusted OR (95% CI)	p-value
CRP Positive	DM *vs* NAFLD	1.126 (0.499–2.543)	0.775	0.852 (0.324–2.242)	0.75
	DM+NAFLD *vs.* NAFLD	0.633 (0.285–1.407)	0.262	0.683 (0.298–1.568)	0.37
	DM+NAFLD *vs.* DM	0.562 (0.253–1.252)	0.159	0.695 (0.233–2.077)	0.52
Elevated ESR	DM *vs.* NAFLD	1.905 (0.742–4.890)	0.180	1.087 (0.359–3.292)	0.88
	DM+NAFLD *vs.* NAFLD	2.500 (0.991–6.307)	0.052	3.804 (1.345–10.759)	0.01
	DM+NAFLD *vs.* DM	1.312 (0.569–3.029)	0.524	2.926 (0.879–9.737)	0.08

Statistically significant ($p < 0.05$). C-Reactive Protein (CRP); Erythrocyte Sedimentation Rate (ESR); Type 2 Diabetes Mellitus (T2DM), Non-Alcoholic Fatty Liver Disease (NAFLD).

4. Discussion

The present study demonstrates a graded inflammatory profile across T2DM, NAFLD, and their coexistence, with the most pronounced inflammatory burden observed in patients with concurrent T2DM and NAFLD. Although gender distribution and BMI in patients with combined disease were significantly older, suggesting a cumulative metabolic and inflammatory exposure over time [22,23].

While the T2DM group predominantly exhibited low-grade CRP elevation in the current study, the NAFLD group demonstrated a broader distribution, and the T2DM+NAFLD group showed a clear shift toward higher CRP categories. This discrepancy between binary and graded analyses highlights the limitation of dichotomizing inflammatory biomarkers. CRP is an acute-phase reactant synthesized by hepatocytes under interleukin-6 (IL-6) stimulation, reflecting systemic inflammatory activation [24]. In NAFLD, hepatic steatosis and lipotoxicity promote Kupffer cell activation, inflammasome signaling, and cytokine release, directly enhancing hepatic CRP production. In T2DM, insulin resistance promotes adipose tissue macrophage infiltration and increased pro-inflammatory adipokine secretion.

When both conditions coexist, synergistic activation of hepatic and adipose inflammatory pathways likely

explains the upward shift in CRP severity [25–28].

In contrast to CRP, ESR showed a clear and statistically significant progressive increase (DM < NAFLD < DM+NAFLD), with the combined group demonstrating significantly higher levels than either condition alone. ESR reflects alterations in plasma protein composition, particularly fibrinogen and immunoglobulins, which increase during chronic inflammatory states and enhance erythrocyte aggregation [29].

The stronger association observed for ESR in the current study, particularly after age and gender adjustment, suggests that chronic, sustained inflammatory activity may be more prominent in patients with dual pathology [28,30].

Hyperglycemia promotes advanced glycation end-product formation and oxidative stress, activating NF-κB–mediated inflammatory pathways. Concurrently, hepatic steatosis induces mitochondrial dysfunction, endoplasmic reticulum stress, and further stimulate signaling pathways in hepatocytes and macrophages. These converging pathways amplify cytokine production, which not only worsens insulin resistance but also stimulates hepatic acute-phase protein synthesis, thereby elevating ESR-related plasma proteins [31,32]. The result is a chronic, systemic pro-inflammatory milieu more pronounced than either disease alone.

Several studies have shown that both T2DM and NAFLD are independently associated with elevated inflammatory markers, including CRP and ESR [33,34]. Large cohort analyses have reported that CRP correlates more strongly with obesity and visceral adiposity than with glycemic status alone [35,36].

Conversely, studies examining NAFLD severity have demonstrated stronger relationships between advanced steatosis or fibrosis and markers of chronic inflammation, including elevated ESR and fibrinogen levels [37,38]. Furthermore, recent meta-analyses indicate that patients with coexisting T2DM and NAFLD exhibit higher levels of inflammatory mediators [18,39].

Clinically, these findings underscore the importance of recognizing T2DM+NAFLD as a metabolically and immunologically distinct entity with heightened inflammatory burden. Given that chronic inflammation contributes to cardiovascular disease, hepatic fibrosis progression, and microvascular complications [40,41], the significantly increased odds of abnormal ESR in the combined group may have prognostic implications. However, this study has some limitations. Its cross-sectional design prevents causal inference, and the relatively small sample size may have limited statistical power.

Additionally, potential confounders such as disease duration, glycemic control, medication use, and NAFLD severity were not fully adjusted, and measurements were obtained at a single time point. Future studies should use larger, longitudinal, and multicenter designs to clarify causal relationships between inflammation and the coexistence of T2DM and NAFLD. Incorporating comprehensive inflammatory biomarkers, detailed assessment of glycemic control and NAFLD severity, and evaluation of long-term hepatic and cardiovascular outcomes would provide deeper mechanistic and prognostic insight.

Systemic inflammation showed a progressive pattern from T2DM to NAFLD and was most pronounced in patients with combined T2DM and NAFLD. Although CRP positivity did not independently distinguish groups, ESR levels were significantly higher in the combined group, even after adjustment for age and gender.

These findings suggest a synergistic inflammatory interaction in coexisting T2DM and NAFLD, which may contribute to increased metabolic and hepatic risk.

Declaration of artificial intelligence (AI) in the writing process

The authors declare whether AI or AI-assisted technologies were used during the preparation of this manuscript. If used, AI tools were employed solely to improve language quality, grammar, readability, and organizational structure. The authors carefully reviewed and edited all AI-generated content and take full responsibility for the accuracy, integrity, and originality of the final manuscript. No AI tool was used to generate, analyze, or interpret scientific data or images, or to draw scientific conclusions. The use of AI-assisted technologies complies with current publication ethics recommendations and journal policies.

Authors' contributions

AA: Conceptualization, investigation, writing first draft, and reviewing and editing. HA: Conceptualization, investigation, data analysis, supervision. JP: Conceptualization, methodology, data curation, investigation. All authors read and approved the final version of the manuscript.

Conflict of interest

No potential conflict of interest was reported by the authors.

Ethical declarations

The study protocol was approved by the Ethics Committee of Zabol University of Medical Sciences, Zabol, Iran (IR.ZBMU.REC.1402.103), and all individuals singed an informed consent to participate in the study.

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