Derna Academy Journal for Applied Sciences

E-ISSN: 3006-3159

DAJAS

Evaluation of Inflammatory Markers in Patients with Type 2 Diabetes Mellitus in Derna City, Libya: A Case–Control Study

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Volume: 5 **Issue: 2** **Page Number: 161 - 166**

Keywords:
Diabetes Type 2, Inflammatory Mmarker, NLR, CRP

Received: 01\01\2026
Accepted: 02\02\2026
Published: 03\02\2026

https://doi.org/10.71147/427prn62

ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is increasingly recognised as a metabolic disorder with an inflammatory component. Low-grade chronic inflammation contributes to insulin resistance and the development of diabetic complications. This study aimed to evaluate inflammatory markers in patients with T2DM compared with healthy controls and to assess their relationship with glycemic control. Methods: A case–control study was conducted in Derna City, Libya, including 45 patients with T2DM and 30 healthy controls. Fasting blood sugar (FBS), glycated haemoglobin (HbA1c), white blood cell count (WBC), neutrophil count, lymphocyte count, C-reactive protein (CRP), and neutrophil-to-lymphocyte ratio (NLR) were measured. Statistical analysis was performed using SPSS version 26. Independent-sample t-test or Mann–Whitney U test was applied as appropriate. Spearman's correlation was used to assess associations between inflammatory markers and glycemic parameters. Results: Patients with T2DM showed significantly higher CRP, NLR, and WBC values compared with controls. FBS and HbA1c were markedly elevated in the diabetic group. Spearman analysis revealed a strong positive correlation between FBS and HbA1c ($r = 0.889$, $p < 0.001$). CRP and NLR were moderately correlated with WBC, indicating an inflammatory linkage. However, no significant correlation was observed between glycemic parameters and inflammatory markers. Conclusion: Inflammatory markers such as CRP and NLR are elevated in patients with T2DM, supporting the role of low-grade inflammation in diabetes. Although direct correlations with glycemic control were not significant, these markers may serve as useful indicators of systemic inflammation in T2DM patients.

1. INTRODUCTION

Type 2 diabetes is a global, multifactorial chronic disease. In addition to being a metabolic disease marked by insulin resistance and hyperglycemia, type 2 diabetes mellitus (T2DM) is also linked to persistent low-grade inflammation (Elbaruni 2023). Because diabetes and its consequences, such as cardiovascular disease, nephropathy, and neuropathy, are both influenced by this inflammation (Wang 2013).

Furthermore, the majority of diabetic patients with poor glycemic control and elevated glucose levels have impaired immune function. Patients with type 2 diabetes (T2DM) are not insulin dependent. Nonetheless, inflammatory indicators can be helpful in risk stratification, assisting in the identification of T2DM patients who are more likely to have high-risk problems (Habibi 2025). Moreover, Treatment efficacy may be determined by longitudinal monitoring of these indicators, particularly when anti-inflammatory tactics are used. In addition, the neutrophil-to-lymphocyte ratio (NLR) is used as an inflammatory measure to track glycated hemoglobin (HbA1c) in patients with type 2 diabetes who have poor glycemic control (Adane 2023). However, the relationship between inflammation and glycemic control remains controversial. Some studies suggest that low-grade inflammation is not consistently present in all patients with T2DM, and inflammatory markers may remain within normal ranges, particularly in early or well-controlled disease (Pickup 1998). Although there was no significant connection between NLR and HbA1c in early or well-managed T2DM (Lou 2015). Similarly, no significant correlation between CRP and HbA1c after controlling for obesity and metabolic factors (Festa 2000). Although WBC was linked to complications, baseline WBC and neutrophil counts did not differ significantly from controls in uncomplicated T2DM (Tong 2004). This study aims to determine and evaluate the inflammatory markers (WBC, Neutrophils, CRP, NLR) between diabetic patients and the control group. And it is correlated with glycemic control (FBS, HbA1c).

2. METHOD

1. Study Design:

This study is a case–control study including 75 participants: 45 patients with type 2 diabetes mellitus (DM) and 30 healthy controls.

The data collection: and duration were conducted at the Laboratory Medicine and Medical Centre, private clinics at Derna city, between the period of May and November 2025. While the participants with Type 2 DM are regular patients for routine analysis monthly, fasting blood sugar & HbA1c and complete blood count.

Participants were excluded from the study if they met any of the following criteria

- presence of acute or chronic inflammatory diseases.
- history of autoimmune disorders, malignancy, or active infection.
- Chronic kidney disease, liver disease, or cardiovascular disease.
- Use of anti-inflammatory drugs, corticosteroids, or immunosuppressive therapy.
- Pregnancy or lactation.
- Recent surgery or trauma within the last three months.
- Type 1 diabetes mellitus or gestational diabetes.

2. Laboratory analysis

- **Demographic data:** Age group between (18-70)، gender, Data collected in a questionnaire form.
- **Laboratory test:** FBS (fasting blood sugar)، HbA1c (glycated Hemoglobin)، WBC (white blood cells)، Neutrophil count, Lymphocyte count, CRP (C-reactive protein)، and Neutrophil to lymphocyte ratio (NLR). Venous blood samples were collected in the morning after a fasting period for chemistry analysis. Using the Coulter counter method, we obtained the neutrophil count (NC) and lymphocyte count (LC) from the whole blood (complete Blood Count) by Mindray BC-5000. The ratio of NC to LC was then used to calculate the NLR. although, the HS-CRP estimation was by Mindray BS-240Pro with normal range>0.***3Mg/l***

Statistical Methods

The statistical analyses were performed using software (SPSS 26.0). Continuous variables were presented as mean ± SD values, and categorical variables were expressed as percentages. Normality was assessed using the Shapiro–Wilk test. Fasting blood glucose showed a non-normal distribution in the diabetic group ($p = 0.001$) but a normal distribution in the healthy group ($p = 0.294$). Glycated hemoglobin (HbA1c) was non-normally distributed in both diabetic ($p = 0.009$) and healthy subjects ($p = 0.002$). Therefore, non-parametric tests (Mann–Whitney U) were used for group comparison Student t test (independent sample t test) was used for continuous variables, those with normal distribution and the Mann–Whitney U test was used for continuous variables that do not have a normal distribution. The chi-square test was used for categorical variables. Spearman correlation analysis was used to assess the relationship between DM and inflammatory markers. Those factors showed significance in univariate logistic regression analysis and were pooled in multivariate analysis to identify independent risk factors of DM.

3. ETHIC APPROVAL

The ethical approval was waived and conducted accordance with the ethical principles of the Declaration of Helsinki. the sample used in this study were utilized archived residual samples collected during routine diagnostic procedures. Therefore, informed consent was not required, and the ethical approval was waived.

4. RESULT

A total of 45 DM patients, 25 male (55,6%) and 20 female (44.4%), and 30 healthy subjects were included. The data are presented as frequency and Percentage. the differences between groups were assessed by using non-parametric using Mann-Whitney U test. Although, a highly significant difference in age groups between the study groups ($U = 84$, $p < 0.001$). Additionally, a statistically significant difference in gender distribution was observed ($U = 502$, $p = 0.031$). There are significant differences between the two groups regarding to gender and age,

as shown in (Table 1).

Table 1: **Demographic Characteristics of the Type 2 diabetes and Healthy groups**

Variable	Diabetic Group (n=45)	Control Group (n=30)	Statistical Test	P-value
Gender	Male:25 (%55.6) Female: 20 (44.4%)	Male: 9 (%30.0) Female: 21 (70.0%)	U = 502	0.031*
Age (Mean)	45.7	18.2	U = 84.5	< 0.001*

A p-value <0.05 was considered statistically significant

Descriptive analysis was assessed according to Fasting blood sugar (FBS) of DM patients(n=45), the mean value is 217±81.7mg\dl with a minimum concentration 106mg\dl and a maximum concentration of glucose 420 mg/dl. while among healthy group (n=30) the mean value is 90±13 mg\dl with rang between (68 mg\dl-120 mg\dl). According to Glycated hemoglobin (HbA1c), the descriptive analysis for Diabetic patients and Healthy participants was shown in Table 2.

Table 2: ***Clinical characteristics between of study participants for FBS and HBA1c***

Variables DM (45)	Minimum	Maximum	Mean	SD±	P-Vlue
FBS	106 mg\dl	420 mg\dl	217.167	81.72	0.001
HbA1c	4.99	13.00	9.6881	2.458	0.001
Healthy control (30) FBS	68.0	120.0	90.533	13.07	0.001
HbA1c	4.00	7.22	4.8037	0.72279	0.001

Also, this table (2) show Mann–Whitney U test demonstrated a highly significant difference in glycemic parameters between the diabetic and healthy groups. Fasting blood sugar ($U = 11$, $p < 0.001$) and HbA1c levels ($U = 19.50$, $p < 0.001$) were significantly higher in patients with diabetes compared to healthy controls."

Mann–Whitney U test showed a significant difference in C-reactive protein levels between diabetic and healthy subjects ($U = 317.0$, $p = 0.032$). However, no significant differences were observed for white blood cell count ($U = 573.0$, $p = 0.270$) or NLR ($U = 646.0$, $p = 0.875$).: Comparison of inflammatory markers (WBS, C-reactive proteins, NLR) within DM and healthy controls (Mean±SD).

Table 3: Comparison of Inflammatory marker between T2DM patients and controls

Variables	DM (n=45) Mean±SD	Control (n=30) Mean±SD	P-value
WBC	8.64±4.79	7.20±2.19	0.270
C-Reactive proteins	10.45±21.59	2.65±2.88	0.032
NLR	3.91±3.65	2.60±1.54	0.875

A p-value <0.05 was considered statistically significant.

Table 4: Spearman's Correlation Between Glycemic Parameters and Inflammatory Markers

Variables Compared	P- value	Strength & Direction	Interpretation
Study group – FBS	<0.001	Strong negative	Higher FBS in T2DM
Study group – HbA1c	<0.001	Strong negative	Poor glycemic control in T2DM
Study group – CRP	0.031	Weak negative	Not statistically significant
Study group – NLR	0.430	Very weak	No association
Study group – WBC	0.271	Weak	No association
FBS – HbA1c	<0.001	Strong positive	Consistent glycemic markers
CRP – WBC	0.007	Moderate positive	Inflammatory linkage
NLR – WBC	0.009	Moderate positive	Hematological inflammation

5. DISCUSSION

The present study demonstrated a strong and statistically significant association between diabetes status and glycemic parameters, including fasting blood sugar (FBS) and glycated hemoglobin (HbA1c). Patients with type 2 diabetes mellitus (T2DM) showed significantly higher levels of FBS and HbA1c compared with healthy controls, reflecting poor short-term and long-term glycemic control. These findings are consistent with the well-established pathophysiology of T2DM, where insulin resistance and β-cell dysfunction led to persistent hyperglycemia. (Chukwuka,2025)

A strong positive correlation was observed between FBS and HbA1c, indicating consistency between short-term : glycemic status and long-term glucose exposure. This relationship supports the validity of the biochemical measurements used in this study and aligns with previous research demonstrating that elevated fasting glucose levels are predictive of increased HbA1c values. (Nathan 2008)

Despite extensive evidence linking type 2 diabetes mellitus to chronic low-grade inflammation, the present study did not demonstrate significant correlations between diabetes status and inflammatory markers such as white blood cell count (WBC) and neutrophil-to-lymphocyte ratio (NLR). These findings suggest that inflammatory activation in T2DM may not always be detectable using routine hematological indices, particularly in clinically stable patients.

(Pickup 2004). However, the absence of significant inflammatory associations in this study may be attributed to the low-grade nature of inflammation commonly observed in T2DM. Such inflammatory activity is often subtle and may not be sufficiently reflected by non-specific markers like WBC and NLR. More sensitive biomarkers, including high-sensitivity C-reactive protein (hs-CRP) with pro-inflammatory cytokines (IL-6, TNF-α), have been shown to better capture inflammatory changes in diabetes. (Pradhan 2001). In the current study, C-reactive protein (CRP) showed a weak inverse correlation with diabetes status, which, although statistically significant, was of limited clinical relevance. This finding may reflect inter-individual variability, differences in metabolic control, or the use of conventional CRP rather than hs-CRP. Previous studies have emphasized that only high-sensitivity CRP reliably reflects low-grade systemic inflammation in metabolic disorders. (Ridker 2003).

The observed positive correlations between WBC and both CRP and NLR are biologically plausible and reflect the coordinated response of innate immune components during inflammatory activation. These associations indicate that while inflammatory markers may correlate with each other, they do not necessarily correlate with glycemic control or diabetes status in all populations (Gabay 1999) In contrast, several previous studies have reported elevated inflammatory markers in patients with T2DM; however, many of these investigations included individuals with poor glycemic control, obesity, or advanced diabetic complications. In contrast, the present study population may represent a relatively stable diabetic cohort, which could explain the absence of strong inflammatory associations. (Dandona 2004).

Obviously, the findings of this study suggest that glycemic dysregulation is a defining feature of T2DM, whereas inflammatory activation may vary depending on disease stage, metabolic status, and individual patient characteristics. Therefore, inflammation should not be assumed to be uniformly elevated in all patients with T2DM (Donath 2011).

The study has certain limitations, including a moderate sample size and the use of non-specific inflammatory markers. Additionally, potential confounding factors such as body mass index, duration of diabetes, and medication use were not adjusted for, which may have influenced inflammatory profiles (Pearson 2003).

6. CONCLUSION

While inflammation plays an important role in the pathogenesis of type 2 diabetes mellitus, the present study indicates that inflammatory markers may not be consistently elevated in all diabetic populations, particularly in stable cases without advanced complications.

7. REFERENCES

Adane, T., Melku, M., Worku, Y. B., Fasil, A., Aynalem, M., Kelem, A., & Getawa, S. (2023). The Association between Neutrophil-to-Lymphocyte Ratio and Glycemic Control in Type 2 Diabetes Mellitus: A Systematic Review and Meta-Analysis. Journal of diabetes research, 2023, 3117396.

Dandona P, et al. Inflammation: the link between insulin resistance, obesity, and diabetes. Trends Immunol. 2004;25(1):4–7.

Donath MY, Shoelson SE. Type 2 diabetes as an inflammatory disease. Nat Rev Immunol. 2011;11(2):98–Limitations.

Elbaruni K, Abdulwahed E, Khalfalla W, Alsudany R, Jerbi R, Alwaseea N, et al. Association Between Some Inflammatory Markers and HbA1c in Patients with Type 2 Diabetes Mellitus. Alq J Med App Sci. 2023;6(1):137-141 .

Festa, A., D'Agostino Jr, R., Howard, G., Mykkänen, L., Tracy, R. P., & Haffner, S. M. (2000). Chronic subclinical inflammation as part of the insulin resistance syndrome: the Insulin Resistance Atherosclerosis Study (IRAS). Circulation, 102(1), 42-47.

Gabay C, Kushner I. Acute-phase proteins and other systemic responses to inflammation. N Engl J Med. 1999;340(6):448–454.

Habibi, A., Letafatkar, N., Sattari, N., Nobakht, S., Rafat, Z., Soltani Moghadam, S., Mirdamadi, A., Javid, M., Jamilian, P., Hassanipour, S., Keivanlou, M. H., & Amini-Salehi, E. (2025). Modulation of inflammatory markers in type 2 diabetes mellitus through gut microbiome-targeted interventions: An umbrella review on meta-analyses. Clinical nutrition ESPEN, 65, 93–104.

Lou, M., Luo, P., Tang, R., Peng, Y., Yu, S., Huang, W., & He, L. (2015). Relationship between neutrophil-lymphocyte ratio and insulin resistance in newly diagnosed type 2 diabetes mellitus patients. BMC endocrine disorders, 15(1), 9.

Nathan DM, et al. Translating the A1C assay into estimated average glucose values. Diabetes Care. 2008;31(8):1473–147.

Pearson TA, et al. Markers of inflammation and cardiovascular disease. Circulation. 2003;107(3):499–511.

Pickup JC. Inflammation and activated innate immunity in the pathogenesis of type 2 diabetes. Diabetes Care. 2004;27(3):813–823.

Pickup, J. C., & Crook, M. A. (1998). Is type II diabetes mellitus a disease of the innate immune system?. Diabetologia, 41(10), 1241-1248.

Pradhan AD, et al. Inflammatory biomarkers, incident type 2 diabetes, and risk prediction. Lancet. 2001;358(9286):1109–1113.

Ridker PM. Clinical application of C-reactive protein for cardiovascular disease detection and prevention. Circulation. 2003;107(3):363–369.

Tong, P. C., Lee, K. F., So, W. Y., Ng, M. H., Chan, W. B., Lo, M. K., ... & Chan, J. C. (2004). White blood cell count is associated with macro-and microvascular complications in Chinese patients with type 2 diabetes. Diabetes care, 27(1), 216-222.

Wang, X., Bao, W., Liu, J., Ouyang, Y. Y., Wang, D., Rong, S., Xiao, X., Shan, Z. L., Zhang, Y., Yao, P., & Liu, L. G. (2013). Inflammatory markers and risk of type 2 diabetes: a systematic review and meta-analysis. Diabetes care, 36(1), 166–175.