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# NEUTROPHIL–LYMPHOCYTE RATIO AS A MARKER OF GLYCEMIC STATUS IN TYPE 2 DIABETES

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**ABSTRACT**

**Background**:
Type 2 Diabetes Mellitus (T2DM) is associated with chronic low-grade inflammation, and the Neutrophil–Lymphocyte Ratio (NLR) has emerged as a simple and accessible marker of systemic inflammatory status. This study aimed to evaluate the correlation between NLR and glycaemic indicators, particularly HbA1c, in patients with T2DM.

**Methods**:
This prospective cross-sectional study was conducted on 80 participants, including 40 patients with T2DM and 40 age- and sex-matched healthy controls. Demographic details, clinical parameters, anthropometric measurements, and biochemical indices including fasting blood sugar (FBS), postprandial blood sugar (PPBS), HbA1c, lipid profile, and complete blood count were recorded. NLR was calculated from absolute neutrophil and lymphocyte counts. Group comparisons were performed using the unpaired t-test and Chi-square test. Correlations were assessed using Pearson's correlation coefficient, with $p < 0.05$ considered statistically significant.

**Results**:
Diabetic patients had significantly higher FBS (211.37 ± 146.40 mg/dL vs. 94.94 ± 8.94 mg/dL), PPBS (326.63 ± 201.67 mg/dL vs. 130.53 ± 11.57 mg/dL), and HbA1c (8.27 ± 1.70% vs. 5.29 ± 0.31%) compared with controls (all $p < 0.001$). NLR was markedly elevated in diabetics (3.55 ± 1.19) compared with non-diabetics (1.39 ± 0.41) ($p < 0.001$), with 97.5% of diabetics showing NLR > 2 versus 5% of controls. Significant correlations were observed between NLR and FBS ($r = 0.559$, $p < 0.001$), PPBS ($r = 0.546$, $p < 0.001$), HbA1c ($r = 0.341$, $p = 0.031$), duration of diabetes ($r = 0.316$, $p = 0.047$), and abdominal diameter ($r = 0.392$, $p = 0.012$).

**Conclusion**:
NLR is significantly elevated in T2DM and correlates with key glycaemic parameters and anthropometric indices. As an inexpensive and routinely available inflammatory marker, NLR may serve as a useful adjunct in assessing metabolic status in T2DM. Further longitudinal studies are needed to explore its prognostic value.

**KEYWORDS**: Type 2 Diabetes Mellitus, Neutrophil–Lymphocyte Ratio, HbA1c, Inflammation, Glycaemic control.

## INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by persistent hyperglycaemia resulting from a combination of insulin resistance and impaired insulin secretion. The global burden of diabetes has risen dramatically over recent decades. The prevalence among adults increased from 4.7% in 1980 to 8.5% in 2014, with the number of people living with diabetes escalating from 108 million to 422 million over the same period. [1]According to the World Health Organization, diabetes is projected to become the seventh leading cause of mortality by 2030.[2] Diabetes is broadly classified into four categories—Type 1 diabetes mellitus (T1DM), Type 2 diabetes mellitus (T2DM), gestational diabetes mellitus (GDM), and other specific types associated with defined pathologies.[3-6]

T2DM accounts for approximately 85–96% of all diabetes cases and is strongly linked to modifiable and non-modifiable risk factors.[7,8] Advancing age, central obesity, physical inactivity, dietary factors, hypertension, dyslipidaemia, and genetic predisposition significantly contribute to disease development.[9,10] The pathophysiological hallmark of T2DM involves progressive β-cell dysfunction along with insulin resistance, influenced by glucotoxicity, lipotoxicity, inflammatory mediators, and genetic susceptibility.[11-13] These processes collectively lead to chronic hyperglycaemia, which predisposes individuals to both microvascular and macrovascular complications, including retinopathy, nephropathy, neuropathy, cardiovascular disease, and peripheral vascular disease.[14,15]

Glycated haemoglobin (HbA1c) is widely used to assess long-term glycaemic control, reflecting average blood glucose

levels over 2–3 months.[16] It is formed by a non-enzymatic reaction between glucose and the N-terminal valine of the β-chain of haemoglobin, producing stable ketoamine structures.[17] HbA1c was first detected in the 1950s and later recognized as a reliable indicator for monitoring diabetes.[18] Consequently, the American Diabetes Association (ADA) recommends HbA1c ≥ 6.5% as a diagnostic criterion for diabetes. However, despite its widespread utility, HbA1c does not fully reflect the inflammatory burden associated with T2DM and may be influenced by hematological, renal, and genetic factors.

Chronic low-grade inflammation plays a central role in the pathogenesis and progression of T2DM. Elevated leukocyte counts, including neutrophils, are associated with systemic inflammation, endothelial dysfunction, atherosclerosis, and subsequent vascular complications.[19] The neutrophil–lymphocyte ratio (NLR), derived from routine complete blood counts, has emerged as a simple, cost-effective, and widely accessible biomarker reflecting systemic inflammation.[20] Compared with isolated leukocyte parameters, NLR is more stable and less affected by physiological variability, making it a potentially superior inflammatory marker.

Several studies have demonstrated that increased NLR is associated with poor glycaemic control and a higher risk of diabetes-related complications. Elevated NLR correlates with higher fasting blood glucose, HbA1c levels, and insulin resistance.[21,22] Studies also show that NLR is significantly higher in diabetic patients with microvascular complications such as retinopathy, nephropathy, and neuropathy.[23,24] Further investigations have reported that NLR progressively increases with worsening glycaemic control and may serve as a surrogate marker for early detection of inflammation-associated vascular damage in diabetes. [25,26] Additionally, research indicates that higher NLR may identify diabetic individuals at increased risk for cardiovascular and renal complications.[26-28]

Despite substantial evidence supporting the association between NLR and diabetes severity, limited data are available from the Indian population, particularly from the northern Himalayan region. Considering the high and rising prevalence of T2DM in India, understanding simple inflammatory markers that correlate with glycaemic status may have significant clinical utility.

Therefore, the present study was undertaken to evaluate the correlation between neutrophil–lymphocyte ratio and HbA1c levels in patients with type 2 diabetes mellitus. This may help determine whether NLR can serve as an accessible adjunct marker for assessing glycaemic control and identifying patients at risk of inflammation-mediated complications.

## MATERIALS AND METHODS

### Study Design and Setting

This prospective observational cross-sectional study was conducted in the Department of General Medicine, Maharishi Markandeshwar Medical College and Hospital, Kumarhatti, Solan, over an 18-month period. Institutional Ethics Committee approval was obtained before the start of the study (IEC No. MMMCH/IEC/23/640).

### Study Population

The study included 80 subjects aged 30–70 years, comprising 40 patients with Type 2 Diabetes Mellitus (T2DM) and 40 healthy controls. T2DM was diagnosed using ADA criteria (HbA1c ≥ 6.5%), while controls had HbA1c < 5.7%. Inclusion criteria were adults aged 30–70 years diagnosed with T2DM, both newly diagnosed and those on oral hypoglycaemic agents or insulin. Exclusion criteria included current use of statins, aspirin, or thiazolidinediones; acute infections; chronic inflammatory conditions (inflammatory bowel disease, rheumatoid arthritis, gout, asthma, chronic hepatitis); history of myocardial infarction or stroke; and chronic kidney disease. Sample size was calculated using standard deviation (SD = 2.98) from previous data, with 5% significance and 80% power, requiring 37 participants in each group; therefore, 40 subjects were included per group.

### Data Collection and Laboratory Analysis

Detailed demographic information, duration of diabetes, dietary habits, physical activity, and comorbidities were recorded for all participants, and anthropometric measurements such as Body Mass Index (BMI) and abdominal diameter were measured. After overnight fasting, venous blood samples were collected and analysed for Fasting Blood Sugar (FBS), Postprandial Blood Sugar (PPBS), HbA1c (ROCHE C311), lipid profile (triglycerides and HDL), liver and renal function tests, and viral markers. Complete blood count (CBC) was performed using an automated 5-part hematology analyser (XN-550), and Neutrophil–Lymphocyte Ratio (NLR) was calculated as absolute neutrophil count divided by absolute lymphocyte count. The primary study outcome was the correlation between NLR and HbA1c among T2DM patients, while secondary analyses included comparison of NLR between diabetic and non-diabetic groups and correlations of NLR with FBS, PPBS, duration of diabetes, and abdominal diameter.

### Statistical Analysis

Statistical analysis was performed using SPSS version 26.0 (IBM Corp., USA). Continuous variables were expressed as mean ± standard deviation and compared using the unpaired Student's t-test, whereas categorical variables were compared using the Chi-square test or Fisher's Exact test. Pearson's correlation coefficient (r) was used to evaluate correlations between NLR and glycaemic parameters (FBS, PPBS, HbA1c), duration of diabetes, and abdominal diameter. A p-value $<0.05$ was considered statistically significant.

### Ethical Considerations

Written informed consent was obtained from all participants prior to enrolment, participation was voluntary with the option to withdraw at any time, and no additional financial burden or risk was imposed during the study.

**RESULTS**

A total of 80 subjects were included in the study, comprising 40 patients with Type 2 Diabetes Mellitus (T2DM) and 40 healthy controls. The age distribution across the study population was similar between the two groups. The mean age of non-diabetic participants was 54.45 ± 9.76 years, while that of diabetic participants was 55.00 ± 8.05 years, with no statistically significant difference ($p = 0.78$). Age-wise distribution also showed comparable proportions across the categories ≤40 years, 41–50 years, 51–60 years, and >60 years ($p = 0.956$). Baseline demographic and clinical characteristics of the study participants are shown in Table 1.

| Characteristic | Non-Diabetic (N = 40) | Diabetic (N = 40) | Total (N = 80) |
|---|---|---|---|
| Age — yr | 54.45 ± 9.76 | 55.00 ± 8.05 | 54.73 ± 8.90 |
| ≤40 yr | 2 (5.0) | 2 (5.0) | 4 (5.0) |
| 41–50 yr | 13 (32.5) | 13 (32.5) | 26 (32.5) |
| 51–60 yr | 13 (32.5) | 11 (27.5) | 24 (30.0) |
| >60 yr | 12 (30.0) | 14 (35.0) | 26 (32.5) |
| Sex — no. (%) | | | |
| Male | 18 (45.0) | 22 (55.0) | 40 (50.0) |
| Female | 22 (55.0) | 18 (45.0) | 40 (50.0) |
| Hypertension — no. (%) | 11 (27.5%) | 14 (35.0%) | 25 (31.3) |
| Body-mass index — kg/m² | 25.81 ± 3.63 | 27.72 ± 4.22 | 26.76 ± 4.00 |
| Abdominal circumference — cm | 91.79 ± 8.05 | 112.63 ± 19.94 | 102.21 ± 17.96 |
| Fasting blood sugar — mg/dL | 94.94 ± 8.94 | 211.37 ± 146.40 | — |
| Postprandial blood sugar — mg/dL | 130.53 ± 11.57 | 326.63 ± 201.67 | — |
| HbA1c — % | 5.29 ± 0.31 | 8.27 ± 1.70 | — |
| Triglycerides — mg/dL | 148.88 ± 51.61 | 191.28 ± 75.13 | — |
| HDL — mg/dL | 48.63 ± 7.05 | 38.27 ± 9.32 | — |
| NLR (continuous) | 1.39 ± 0.41 | 3.55 ± 1.19 | — |
| NLR ≥ 2 — no. (%) | 2 (5.0) | 39 (97.5) | 41 (51.3) |

**Table 1. Demographic and Clinical Characteristics of the Study Participants at Baseline. (**Plus–minus values are means ± SD. Percentages may not total 100 because of rounding. NLR denotes Neutrophil-Lymphocyte Ratio, and BMI denotes Body Mass Index. A dash (—) indicates data not calculated for the total population.)

Sex distribution showed 40 males and 40 females in total, with equal representation across groups ($p = 0.371$). Hypertension prevalence was similar in the two groups, with no significant association between hypertensive status and diabetes ($p = 0.469$). Exercise habits did not differ significantly between diabetics and non-diabetics ($p = 0.18$). Dietary patterns (vegetarian vs. mixed diet) were also comparable between the groups ($p = 0.361$). Body Mass Index (BMI) was significantly higher in the diabetic group (27.72 ± 4.22 kg/m²) compared with the non-diabetic group (25.81 ± 3.63 kg/m²) ($p = 0.03$). Abdominal diameter also showed a significant difference, with diabetics having a larger mean abdominal diameter (112.63 ± 19.94 cm) than non-diabetics (91.79 ± 8.05 cm) ($p < 0.001$).

Glycaemic parameters differed significantly between the two groups. Fasting blood sugar (FBS) was substantially higher in T2DM patients (211.37 ± 146.40 mg/dL) than in controls (94.94 ± 8.94 mg/dL) ($p < 0.001$). Postprandial blood sugar (PPBS) values showed a similar pattern, with diabetics having a mean PPBS of 326.63 ± 201.67 mg/dL compared with 130.53 ± 11.57 mg/dL in controls ($p < 0.001$). HbA1c values were significantly elevated in diabetic subjects (8.27 ± 1.70%) compared to non-diabetic individuals (5.29 ± 0.31%) ($p < 0.001$). Lipid profile assessment revealed significantly higher triglyceride (TGL) levels among diabetics (191.28 ± 75.13 mg/dL) compared with controls (148.88 ± 51.61 mg/dL) ($p < 0.001$). HDL cholesterol levels were lower in the diabetic group (38.27 ± 9.32 mg/dL) compared with the control group (48.63 ± 7.05 mg/dL) ($p < 0.001$). Comparisons of continuous variables between diabetic and non-diabetic participants are presented in Table 2.

| Variable | Non-Diabetic (N = 40) Mean ± SD | Diabetic (N = 40) Mean ± SD | t-value | p-value |
|---|---|---|---|---|
| Age (years) | 54.45 ± 9.76 | 55.00 ± 8.05 | –0.28 | 0.78 |

| | | | | |
|---|---|---|---|---|
| Body-mass index — kg/m² | 25.81 ± 3.63 | 27.72 ± 4.22 | –2.17 | 0.03 |
| Abdominal circumference — cm | 91.79 ± 8.05 | 112.63 ± 19.94 | –6.13 | <0.001 |
| Fasting blood sugar — mg/dL | 94.94 ± 8.94 | 211.37 ± 146.40 | –5.02 | <0.001 |
| Postprandial blood sugar — mg/dL | 130.53 ± 11.57 | 326.63 ± 201.67 | –6.14 | <0.001 |
| HbA1c — % | 5.29 ± 0.31 | 8.27 ± 1.70 | –10.89 | <0.001 |
| Triglycerides — mg/dL | 148.88 ± 51.61 | 191.28 ± 75.13 | –2.94 | 0.004 |
| HDL — mg/dL | 48.63 ± 7.05 | 38.27 ± 9.32 | 5.61 | <0.001 |
| NLR (continuous) | 1.39 ± 0.41 | 3.55 ± 1.19 | –10.88 | <0.001 |

**Table 2. Comparison of Continuous Variables Between Non-Diabetic and Diabetic Participants** (Plus–minus values are means ± SD. P values were calculated with the independent samples t-test. NLR denotes Neutrophil–Lymphocyte Ratio; HDL denotes High-Density Lipoprotein.)

Neutrophil–Lymphocyte Ratio (NLR) showed a marked difference between the groups. Mean NLR in diabetics was 3.55 ± 1.19, whereas in controls it was 1.39 ± 0.41 ($p < 0.001$). Distribution analysis demonstrated that 97.5% of diabetic subjects had an NLR > 2, while only 5% of controls showed NLR > 2, a difference that was statistically significant ($\chi^2 = 68.49$, $p < 0.001$).

Correlation analysis performed among diabetic patients showed significant associations between NLR and multiple metabolic parameters and is summarised in table 3. A strong positive correlation was observed between NLR and fasting blood sugar ($r = 0.559$, $p < 0.001$). Similarly, NLR correlated strongly with postprandial blood sugar ($r = 0.546$, $p < 0.001$). A mild but statistically significant correlation was noted between NLR and HbA1c ($r = 0.341$, $p = 0.031$). NLR also showed a mild correlation with duration of diabetes in months ($r = 0.316$, $p = 0.047$). Additionally, NLR was positively correlated with abdominal diameter ($r = 0.392$, $p = 0.012$). A positive association was observed between the neutrophil–lymphocyte ratio (NLR) and multiple metabolic parameters. As shown in Figure 1, NLR increased with rising fasting blood glucose (Panel A) and postprandial blood glucose (Panel B). A mild upward trend was also noted between NLR and glycated hemoglobin (HbA1c) (Panel C). In addition, NLR showed a positive relationship with abdominal diameter (Panel D).

| Variable | Pearson Correlation (r) | p-value |
|---|---|---|
| Fasting blood sugar (FBS) | 0.559 | <0.001 |
| Postprandial blood sugar (PPBS) | 0.546 | <0.001 |
| HbA1c (%) | 0.341 | 0.031 |
| Duration of diabetes (months) | 0.316 | 0.047 |
| Abdominal circumference (cm) | 0.392 | 0.012 |

**Table 3: Correlation analysis between NLR and key glycemic and metabolic parameters in the diabetic group.** (Correlation analysis was performed using Pearson's correlation coefficient. NLR denotes Neutrophil–Lymphocyte Ratio.)

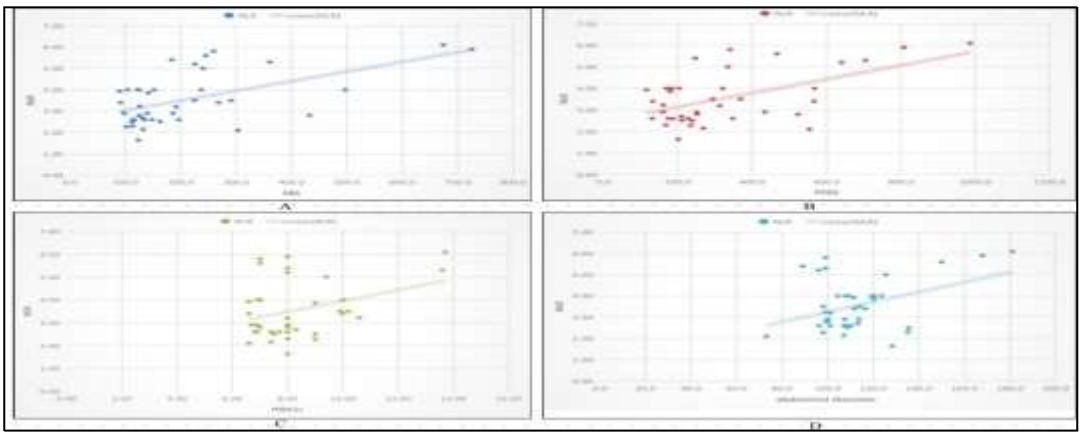

**Figure 1. Correlation of the Neutrophil–Lymphocyte Ratio with Glycemic and Anthropometric Variables.**

(Panel A shows the relationship between the neutrophil–lymphocyte ratio (NLR) and fasting blood glucose; Panel B, the relationship between the NLR and postprandial blood glucose; Panel C, the relationship between the NLR and glycated hemoglobin (HbA1c); and Panel D, the relationship between the NLR and abdominal diameter. A linear regression line is shown for each association.)

In summary, significant differences were present between diabetic and non-diabetic groups across BMI, FBS, PPBS, HbA1c, lipid profile parameters, abdominal diameter, and NLR, while age, sex, diet, exercise, and hypertension did not differ significantly. Correlation analysis within the diabetic group demonstrated significant associations between NLR and glycaemic as well as anthropometric parameters.

## DISCUSSION

Type 2 Diabetes Mellitus (T2DM) represents a chronic metabolic and inflammatory disorder in which insulin resistance, hyperglycaemia, oxidative stress, and immune dysregulation play interconnected roles. The Neutrophil–Lymphocyte Ratio (NLR), derived from a complete blood count, has recently gained recognition as an accessible marker of systemic inflammation in metabolic diseases. Several studies in the literature have demonstrated a strong relationship between NLR and glycaemic control, insulin resistance, and diabetes-related complications. The present study adds to this evidence by evaluating NLR among T2DM patients and its correlation with glycaemic markers, particularly HbA1c.

In the present study, NLR levels were significantly higher in patients with diabetes than in controls, with 97.5% of diabetics showing NLR > 2, whereas only 5% of non-diabetics exhibited such values. This observation aligns with earlier findings summarised in the thesis, including those by Sefil et al. (2014) [21], Shiny et al. (2014) [22], and Varma et al. (2021) [29], all of which reported elevated NLR among uncontrolled diabetics. Dayama et al. (2024)[30] and Yadav et al. (2022)[27] also found significantly higher NLR in Indian diabetic populations.

In addition to the between-group differences, a statistically significant positive correlation was observed between NLR and fasting blood sugar ($r = 0.559$) as well as postprandial blood sugar ($r = 0.546$). Similar associations were documented by Hussain et al. (2017) [19], Gowda et al. [31], and Moursy et al. (2015) [23], who noted that higher NLR levels accompany worsening glycaemic parameters. A mild but significant correlation ($r = 0.341$) was also observed between NLR and HbA1c, consistent with studies such as Khandare et al. (2017) [32] and Akin et al. (2019) [33], all of which reported a positive relationship between long-term glycaemic control and NLR.

The positive association between abdominal diameter and NLR observed in this study aligns well with the established role of central obesity in generating chronic inflammation. Visceral adipocytes release numerous cytokines, including TNF-α and IL-6, which promote neutrophil activation and reduce lymphocyte counts. These mechanisms have been well described in studies by de Luca and Olefsky (2008) [34], Ceriello and Motz (2004) [35], and Guo et al. (2015) [36], The correlation between NLR and diabetes duration found in this study is supported by earlier work by Devamsh et al. (2019) [26], suggesting that prolonged metabolic stress contributes to persistent elevation of inflammatory markers.

Although our study did not evaluate microvascular or macrovascular complications separately, the thesis literature includes multiple studies—such as Moursy et al. [23], and Khandare et al. [32]—demonstrating significantly elevated NLR in patients with diabetic retinopathy, nephropathy, neuropathy, and cardiovascular involvement. These findings suggest that NLR may reflect both metabolic dysregulation and complication risk.

The underlying pathophysiological basis for elevated NLR in T2DM is supported by several mechanistic studies referenced in the thesis. Hyperglycaemia promotes neutrophil activation, oxidative stress, and lymphocyte apoptosis, while chronic inflammation from visceral obesity disrupts adaptive immune function. These processes are well documented in mechanistic studies by de Luca & Olefsky (2008) [34] and Chiasson et al. [13], reinforcing the biological plausibility of the observed associations.

The findings of the present study are in agreement with numerous Indian and international studies, including works by Varma et al. [29], Yadav et al. [27], Sefil et al. [21], and Shiny et al. [22], all of which have shown that elevated NLR correlates with markers of glycaemic dysregulation.

## STRENGTHS AND LIMITATIONS

The present study has several strengths, including the use of age- and sex-matched controls, which minimized demographic confounding as recommended by Duman et al. [25], and strict exclusion criteria that reduced the influence of acute infections, chronic inflammatory diseases, and medications known to alter leukocyte counts, consistent with the methodological approach of Sefil et al. [21]. Standardized biochemical and hematological procedures were used for HbA1c and CBC estimation, in line with established laboratory practices described by Koenig et al. [18], and the inclusion of multiple clinical, biochemical, and anthropometric variables allowed a comprehensive assessment similar to Shiny et al. and Varma et al. [22, 29]. The study also contributes valuable regional Indian data, supporting findings by Yadav et al. and Dayama et al. [27,30]. However, the study is limited by its modest sample size from a single tertiary-care centre, which restricts generalizability, a limitation also noted by Devamsh et al. [26]. The cross-sectional design prevents assessment of causality, similar to limitations in studies by Sefil et al. [21]. Although exclusion criteria were carefully applied, the possibility of subclinical inflammation influencing NLR cannot be completely ruled out, as emphasized by Moursy et al. [23]. Additionally, the study did not evaluate microvascular or macrovascular complications, and it did not include other inflammatory biomarkers such as CRP, IL-6, ESR, or TNF-α, which limits comparison with mechanistic work by de Luca and Olefsky [34].

## CONCLUSION

The present study demonstrates that the Neutrophil–Lymphocyte Ratio (NLR) is significantly higher in patients with Type 2 Diabetes Mellitus compared with healthy individuals and shows meaningful positive correlations with fasting blood sugar, postprandial blood sugar, HbA1c, abdominal diameter, and duration of diabetes. These findings, consistent with previously published work cited in the thesis, indicate that NLR reflects the underlying inflammatory burden associated with hyperglycaemia and metabolic dysregulation. As NLR is inexpensive, easily obtainable from routine complete blood counts, and correlates with key glycaemic parameters, it may serve as a useful adjunct inflammatory marker for evaluating metabolic status in Type 2 diabetes. Further multicentric and longitudinal studies are warranted to determine its prognostic value and its potential role in predicting diabetes-related complications.

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