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Evaluation of Neutrophil-to-Lymphocyte Ratio as a Predictive Marker of Glycemic Control in Patients with Type 2 Diabetes Mellitus

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ABSTRACT

Introduction: Type 2 diabetes mellitus (T2DM) is a chronic metabolic disease marked by sustained hyperglycaemia and a persistent, low-grade inflammatory state. The neutrophil-to-lymphocyte ratio (NLR), which can be readily derived from a routine complete blood count, has gained attention as a pragmatic marker of systemic inflammation and metabolic disturbance. This study evaluates the relationship between NLR and glycaemic control in individuals with T2DM.

Materials and Methods: This cross-sectional observational study was conducted in the Departments of General Medicine and Endocrinology at Satguru Partap Singh Hospital, Ludhiana, between May 2023 and July 2024. We enrolled 100 adults (≥18 years) with T2DM diagnosed per ADA criteria. For each participant, clinical and anthropometric data were obtained, and laboratory measurements included fasting and post-prandial plasma glucose, glycated haemoglobin (HbA1c), and a complete blood count. Participants were stratified as having good glycaemic control (HbA1c ≤7%) or poor control (HbA1c >7%). The neutrophil-to-lymphocyte ratio (NLR) was computed as absolute neutrophils divided by absolute lymphocytes. Statistical analyses included the independent t-test, Pearson correlation, and receiver operating characteristic (ROC) analysis.

Results: The cohort had a mean age of 52.1 ± 8.8 years, and 56% were female. Average HbA1c was 9.09 ± 2.55%, and the mean NLR was 2.19 ± 0.94. Overall, 81% of participants had poor glycaemic control. NLR was significantly higher in the poor-control group than in the good-control group ($p < 0.05$). NLR correlated positively with HbA1c ($r = 0.34$, $p < 0.01$). Receiver-operating characteristic analysis indicated moderate discriminative ability of NLR for poor control (AUC = 0.71).

Conclusion: An increased neutrophil-to-lymphocyte ratio is independently linked with higher HbA1c, indicating poorer glycaemic control in T2DM. As a simple, inexpensive, and non-invasive marker of systemic inflammation, NLR can complement HbA1c in routine diabetes monitoring, especially in resource-constrained settings.

Keywords*: Type 2 diabetes mellitus, Neutrophil-to-lymphocyte ratio, Glycemic control, HbA1c, Inflammation, Biomarker*

Introduction

Type 2 diabetes mellitus (T2DM) is a multisystem metabolic disease defined by chronic hyperglycaemia and disordered metabolism. Globally, it affects an estimated 6.28% of the population, over 462 million people (1), with India carrying a particularly heavy burden (prevalence 9.3%) (2). T2DM arises from complex gene–environment interactions (3) and is

marked by impaired insulin secretion, insulin resistance, or both (4,5). It is the predominant diabetes phenotype worldwide and continues to rise in both developed and developing settings; the International Diabetes Federation estimated ~425 million adults (8.8% aged 20–79 years) had diabetes in 2017, ~75% in low- and middle-income countries, with projections reaching 642 million (10%) by 2040, driven by urbanisation, sedentary behaviour, and nutrition transitions (6).

T2DM substantially increases the risk of microvascular (retinopathy, nephropathy, neuropathy) and macrovascular (coronary disease, stroke, peripheral arterial disease) complications, worsening quality of life and elevating morbidity and mortality (7). Sustained glycaemic control is therefore essential. Glycated haemoglobin (HbA1c) remains the standard readout of chronic glycaemia, reflecting the preceding 2–3 months and predicting vascular risk (8), but it may be unreliable in contexts such as haemoglobinopathies, severe anaemia, or altered red-cell turnover and does not capture the inflammatory milieu underpinning T2DM.

Accordingly, interest has turned to complementary biomarkers—glycated albumin, fructosamine, 1,5-anhydroglucitol, and the neutrophil-to-lymphocyte ratio (NLR)—to refine assessment and monitoring (9). NLR, obtainable from a routine complete blood count, offers a low-cost index of systemic inflammation accessible across care settings. Inflammation has long been linked to diabetes, from historical observations of salicylate effects on glycosuria to contemporary evidence that chronic, low-grade inflammation is a hallmark of T2DM. Key pathways include c-Jun N-terminal kinase and NF-κB signalling (10), with a cytokine/adipokine milieu (e.g., TNF-α, IL-1, IL-6, IL-10, leptin, adiponectin, resistin) that promotes insulin resistance and endothelial dysfunction (10,11). Hyperglycemia stimulates neutrophil activation, leading to the release of cytokines, chemokines, and reactive oxygen species, while neutrophil extracellular traps can directly injure pancreatic β-cells. Simultaneously, chronic inflammation promotes lymphocyte apoptosis and redistribution, diminishing adaptive immune regulation. This imbalance, reflected by an elevated neutrophil-to-lymphocyte ratio (NLR), signifies a pro-inflammatory milieu intricately linked to glycaemic dysregulation (12).

Emerging studies link higher NLR with poorer glycaemic control and vascular complications (nephropathy, retinopathy, endothelial dysfunction, arterial stiffness, atherosclerosis), with performance comparable to established inflammatory markers such as CRP, TNF-α, and IL-6 for detecting subclinical inflammation and vascular risk (13,14). Given its non-invasive, inexpensive, and widely available nature, NLR is feasible for routine and serial monitoring. However, interpretation of NLR values requires careful clinical context, as factors such as intercurrent infection, physiological stress, medication use, age, sex, and comorbidities may influence its levels. Moreover, universally accepted cut-off values for defining risk categories have yet to be established. Future work should harmonise thresholds and evaluate NLR across diverse populations, ideally alongside HbA1c, fasting plasma glucose, and post-prandial glucose for integrated assessment. Against this backdrop, the present study examines the association between NLR and HbA1c in T2DM to determine whether NLR can function as a practical, predictive marker of poor glycaemic control.

Materials and Methods

Study Design and Setting

This cross-sectional observational study was carried out in the Departments of General Medicine and Endocrinology at Satguru Partap Singh (SPS) Hospital, Ludhiana, Punjab, which is a tertiary care centre in North India. The study included both inpatient and outpatient participants recruited between May 2023 and July 2024.

Sample Size Determination

According to Shambel et al. (2021) (15), the prevalence of poor glycaemic control among patients with type 2 diabetes mellitus (T2DM) was 45.2% (95% CI: 40.6–50.0%). Using this prevalence, the minimum required sample size was calculated as **77**, assuming a 10% margin of error and a 95% confidence level. To improve statistical reliability and reduce sampling variability, the study ultimately enrolled 100 patients with T2DM.

Inclusion Criteria

1. Patients aged ≥18 years of either sex.
2. Diagnosed with type 2 diabetes mellitus as per the American Diabetes Association (ADA) guidelines.

Exclusion Criteria

1. Patients unwilling to provide informed consent.
2. Presence of acute infections or chronic inflammatory diseases (e.g., inflammatory bowel disease, osteoarthritis, rheumatoid arthritis, gout, bronchial asthma, chronic hepatitis).
3. History of malignancy, hematologic disorders, or blood transfusion within the previous three months.
4. Chronic liver disease, chronic heart disease, or acute/chronic renal failure.
5. Recent myocardial infarction or cerebrovascular accident.
6. Use of anti-inflammatory or immunosuppressive medications.

Ethical Considerations and Informed Consent

Ethical approval was obtained from the Institutional Ethics Committee of SPS Hospital, Ludhiana. Eligible patients were briefed about the study objectives in their local vernacular language, and written informed consent was obtained using an

IEC-approved proforma. Participants were assured of confidentiality and informed of their right to withdraw from the study at any time without affecting their medical care.

Study Procedure
All enrolled subjects underwent detailed clinical evaluation. A structured case record form was used to collect demographic information, disease duration, treatment history, and relevant comorbidities. Physical examination included measurement of height, weight, body mass index (BMI), blood pressure, and systemic examination findings.

Sample Collection and Laboratory Investigations
Venous blood samples were collected under aseptic precautions using sterile disposable syringes:

- **Fasting Blood Sugar (FBS):** 1 mL of venous blood was collected in a grey-top (sodium fluoride) Vacutainer after an overnight fast of at least 8 hours. Samples were analyzed using the glucose oxidase-peroxidase (GOD-POD) method.
- **Post-prandial Blood Sugar (PPBS):** 1 mL of venous blood was drawn 2 hours after breakfast and analyzed by the same GOD-POD method.
- **Glycated Hemoglobin (HbA1c):** 2 mL of whole blood was collected in an EDTA Vacutainer and analyzed using the turbidimetric inhibition immunoassay method on the Dimension ExL 200 Auto Analyzer.
- **Complete Blood Count (CBC):** 2 mL of blood was collected in a separate EDTA Vacutainer and analyzed using an automated hematology analyzer based on the electrical impedance principle. Then, the Neutrophil-to-Lymphocyte Ratio (NLR) was calculated as the ratio of absolute neutrophil count to absolute lymphocyte count.

Grouping of Study Participants
Based on HbA1c levels, participants were categorized as:

- **Good Glycemic Control:** HbA1c ≤ 7%
- **Poor Glycemic Control:** HbA1c > 7%

The mean NLR values were compared between the two groups to determine the relationship between systemic inflammation and glycemic status.

Data Management and Statistical Analysis
The dataset was checked for completeness and accuracy, entered into Microsoft Excel, and analyzed in SPSS v24.0 (IBM, Chicago, IL, USA). Baseline characteristics were summarized with descriptive statistics (means ± SD, frequencies, percentages). Group differences were tested using the chi-square test for categorical variables and the unpaired t-test for continuous variables. Associations between NLR and HbA1c were assessed with Pearson's correlation (r). Discriminative performance for poor glycaemic control was evaluated using receiver operating characteristic (ROC) analysis. Statistical significance was set at $p < 0.05$.

Results
Participant characteristics (Table 1)
A total of 100 adults with type 2 diabetes mellitus (T2DM) were enrolled over 15 months (May 2023–July 2024). The mean (SD) age was 52.11 (8.80) years (range 28–71), with most participants aged 41–60 years (72.0%); 12.0% were ≤40 years and 16.0% were >60 years. Females comprised 56.0% of the cohort. The mean weight, height, and BMI were 76.81 (9.98) kg, 167.22 (6.90) cm, and 27.54 (3.87) kg/m², respectively. The mean diabetes duration was 5.77 (3.94) years (range 1–15). Co-existent hypertension was present in 44.0% of participants.

Table 1. Baseline characteristics (N=100)

Variable	Mean (SD) or n (%)	Range
Age, years	52.11 (8.80)	28–71
Age group ≤40 / 41–60 / >60	12 (12.0) / 72 (72.0) / 16 (16.0)	—
Female sex	56 (56.0)	—
Weight, kg	76.81 (9.98)	50.0–101.0
Height, cm	167.22 (6.90)	150.0–178.0
BMI, kg/m²	27.54 (3.87)	18.4–38.7
Diabetes duration, years	5.77 (3.94)	1–15
Hypertension	44 (44.0)	—

Glycaemic status and laboratory profile
Mean fasting blood sugar (FBS) and post-prandial blood sugar (PPBS) were 190.51 (87.77) mg/dL and 274.46 (99.45) mg/dL, respectively. Mean HbA1c was 9.09% (2.55), with values ranging from 5.68% to 17.99%. Overall, 81.0% had poor glycaemic control (HbA1c >7.0%) and 19.0% had good control (HbA1c ≤7.0%).

Haematology showed mean haemoglobin 12.71 (1.83) g/dL, total leukocyte counts 8.07 (2.11) ×10⁹/L, neutrophils 60.31% (8.68), lymphocytes 30.77% (8.10), and a mean neutrophil-to-lymphocyte ratio (NLR) of 2.19 (0.94) (range 0.82–4.81).

Table 2. Glycaemic and haematological profile (overall)

Variable	Mean (SD)	Median	Range
FBS, mg/dL	190.51 (87.77)	165.50	75.0–456.0
PPBS, mg/dL	274.46 (99.45)	258.00	129.0–568.0
HbA1c, %	9.09 (2.55)	8.31	5.68–17.99
Haemoglobin, g/dL	12.71 (1.83)	12.60	9.3–16.2
TLC, $\times 10^9$/L	8.07 (2.11)	7.69	3.90–13.28
Neutrophils, %	60.31 (8.68)	60.70	39.0–78.0
Lymphocytes, %	30.77 (8.10)	30.80	15.4–47.6
NLR	2.19 (0.94)	1.97	0.82–4.81

Group comparisons (good vs poor glycaemic control)

Anthropometrics (weight, height, BMI) and diabetes duration did not differ between groups (all $p>0.05$). As expected, glycaemic indices were higher in the poor-control group (FBS 208.40 vs 114.26 mg/dL; PPBS 294.14 vs 190.58 mg/dL; both $p=0.001$). Inflammatory indices were also less favourable: NLR was higher (2.29 vs 1.73; $p=0.017$), neutrophils were higher (61.47% vs 55.39%; $p=0.005$), and lymphocytes were lower (29.67% vs 35.46%; $p=0.004$). Haemoglobin and total leukocyte count were similar ($p=0.787$ and $p=0.876$, respectively). Age-group and sex distributions did not differ significantly between control strata ($p=0.530$ and $p=0.175$, respectively).

Table 3. Comparison by glycaemic control (HbA1c ≤7.0% vs >7.0%)

Variable	Good control (n=19) Mean (SD)	Poor control (n=81) Mean (SD)	p-value
Weight, kg	77.05 (8.92)	76.75 (10.27)	0.907
Height, cm	167.26 (7.16)	167.21 (6.89)	0.976
BMI, kg/m²	27.57 (3.04)	27.53 (4.05)	0.962
Diabetes duration, y	5.68 (3.15)	5.79 (4.12)	0.917
FBS, mg/dL	114.26 (29.90)	208.40 (87.33)	0.001
PPBS, mg/dL	190.58 (36.72)	294.14 (99.36)	0.001
NLR	1.73 (0.79)	2.29 (0.94)	0.017
Neutrophils, %	55.39 (9.06)	61.47 (8.23)	0.005
Lymphocytes, %	35.46 (8.42)	29.67 (7.67)	0.004
Haemoglobin, g/dL	12.61 (2.41)	12.74 (1.68)	0.787
TLC, $\times 10^9$/L	8.15 (2.62)	8.06 (1.99)	0.876

Age-group distribution ($p=0.530$) and sex distribution ($p=0.175$) did not differ significantly between groups.

Correlation analyses

HbA1c correlated strongly with FBS ($r=0.839$, $p<0.001$) and PPBS ($r=0.888$, $p<0.001$). HbA1c also showed a moderate positive correlation with neutrophil percentage ($r=0.568$, $p<0.001$) and a moderate negative correlation with lymphocyte percentage ($r=-0.593$, $p<0.001$); there was no meaningful correlation with haemoglobin or total leukocyte count ($p=0.230$ and $p=0.228$).

Critically, NLR correlated positively with HbA1c ($r=0.572$, $p<0.001$). NLR also correlated with FBS ($r=0.489$, $p<0.001$) and PPBS ($r=0.462$, $p<0.001$), but not with diabetes duration ($r=-0.196$, $p=0.058$).

Table 4. Correlations

Pair	r	p-value
HbA1c – FBS	0.839	<0.001
HbA1c – PPBS	0.888	<0.001
HbA1c – Neutrophils %	0.568	<0.001
HbA1c – Lymphocytes %	−0.593	<0.001
HbA1c – Haemoglobin	0.059	0.230
HbA1c – TLC	0.060	0.228
HbA1c – NLR	0.572	<0.001
NLR – FBS	0.489	<0.001
NLR – PPBS	0.462	<0.001
NLR – Duration (years)	−0.196	0.058

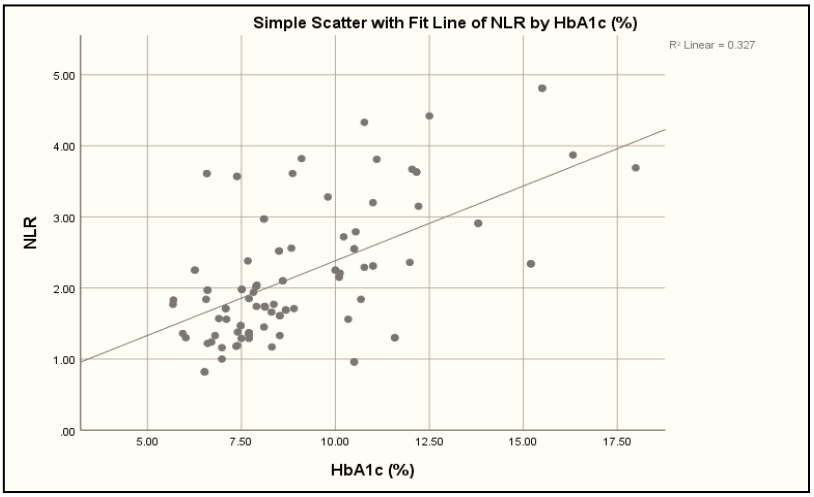

Figure 5: Scatter Plot Showing the Correlation between NLR and HbA1c (%)

ROC analysis for NLR

Receiver-operating characteristic (ROC) analysis for NLR predicting poor glycaemic control (HbA1c>7.0%) yielded an AUC of 0.699 (SE 0.068; 95% CI 0.565–0.832; p=0.007), indicating fair discriminative ability. At the data-derived cut-off NLR ≥1.98, sensitivity was 55.56%, specificity 78.95%, positive predictive value 91.84%, negative predictive value 29.41%, and overall accuracy 60%.

Table 5. ROC metrics for NLR predicting poor control (HbA1c >7.0%)

Metric	Value
AUC (95% CI); p	0.699 (0.565–0.832); 0.007
Optimal cut-off (Youden)	NLR ≥1.98 (Youden index 0.35)
Sensitivity / Specificity	55.56% / 78.95%
PPV / NPV	91.84% / 29.41%
Accuracy	60%

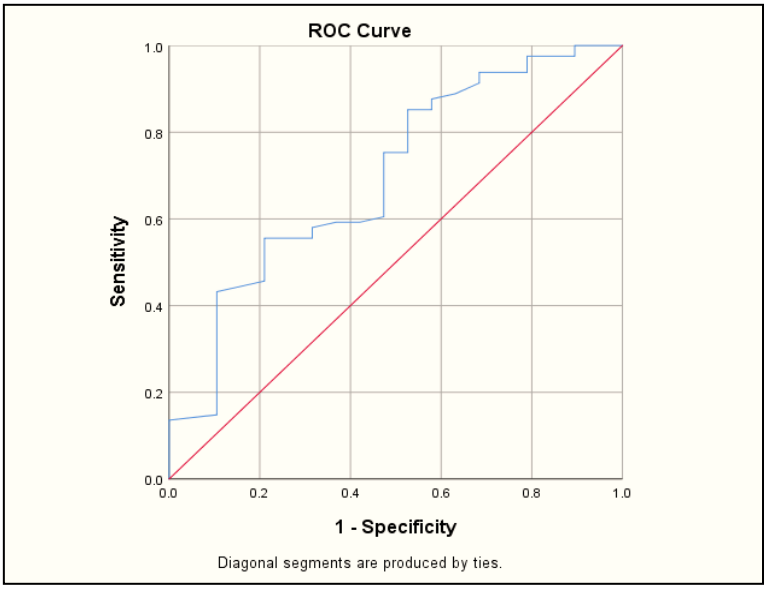

Figure 6: ROC Curve for NLR in Predicting Poor Glycemic Control

Subgroup analyses

Mean NLR did not differ significantly by age group (≤40: 2.23; 41–60: 2.27; >60: 1.83; p=0.241), sex (female: 2.12 vs male: 2.29; p=0.362), or hypertension status (HTN: 2.20 vs no HTN: 2.18; p=0.905).

Discussion

Type 2 diabetes mellitus (T2DM) is driven by chronic hyperglycaemia with attendant micro- and macrovascular complications; achieving and monitoring glycaemic control remains central to mitigating this burden (7). While HbA1c is the accepted standard for long-term control, there is growing interest in adjunct, inexpensive biomarkers that mirror the inflammatory milieu of T2DM. The neutrophil-to-lymphocyte ratio (NLR), derivable from a routine complete blood count,

has emerged as one such candidate. In this cross-sectional study, we assessed the association between NLR and glycaemic control in T2DM and evaluated NLR's discriminatory performance for poor control.

Age, sex, and clinical profile

Our cohort reflected the typical age distribution of T2DM, with most participants being middle-aged to older adults (mean 52.1 years). The slight female predominance (56%) likely reflects local healthcare-seeking patterns rather than true prevalence differences; comparable studies report mixed sex distributions, ranging from balanced samples to female-predominant cohorts (16,17). Although uncontrolled diabetes was numerically more frequent among younger participants and women, neither age-stratified nor sex-stratified analyses reached statistical significance in our data—consistent with prior reports suggesting that age and sex effects on glycaemic control are modest or context-dependent (18–20). BMI values were in the overweight range across groups and, consistent with Devamash GN et al. (21), anthropometry and diabetes duration were not discriminative for glycaemic control in this sample.

NLR and glycaemic control: alignment with the literature

The principal finding is a higher NLR among those with poor glycaemic control (2.29 vs 1.73; $p=0.017$), accompanied by neutrophilia and relative lymphopenia. This agrees with multiple studies demonstrating higher NLR with worsening HbA1c and poorer control (21–27). Our correlation between NLR and HbA1c ($r=0.572$, $p<0.001$) is concordant with effect sizes reported elsewhere (e.g., $r\approx0.49$–0.58) (17,23,27), supporting the premise that NLR captures clinically relevant inflammatory signalling linked to hyperglycaemia and insulin resistance. That HbA1c correlated positively with neutrophils and negatively with lymphocytes in our study further reinforces the mechanistic plausibility that NLR integrates complementary information from both innate and adaptive arms of immunity.

Importantly, NLR's association appeared robust to common clinical covariates in our data: no significant differences by age group, sex, or hypertension status were detected, echoing reports that NLR shows limited interaction with these baseline factors in many cohorts (17,21). The absence of association with diabetes duration in our sample and in the meta-analytic synthesis by Adane T et al. (28) suggests NLR reflects current inflammatory activity more than disease chronicity.

Discriminative performance and clinical utility

ROC analysis yielded an AUC of 0.699 (95% CI 0.565–0.832; $p=0.007$), indicating fair discrimination of poor control by NLR. Using a data-derived cut-off ≥1.98, specificity was 78.95% and PPV was high (91.84%), whereas sensitivity (55.56%) and NPV (29.41%) were modest. Two points follow for practice:

1. **Triage value for "ruling in" poor control:** In clinics where the prevalence of poor control is high (81% in our cohort), a raised NLR efficiently enriches for patients very likely to have HbA1c >7%, aligning with prior work advocating NLR as an adjunct "alert" marker to prioritise tighter follow-up or intensification (16,17,21,25–27).
2. **Limited "rule-out" capacity:** Given the comparatively low sensitivity and negative predictive value, a low NLR cannot exclude poor glycaemic control. HbA1c and, where appropriate, capillary or venous glucose remain indispensable for definitive assessment. Therefore, NLR should serve as a complementary rather than a substitute marker for standard glycaemic indices.

Because test performance is prevalence-dependent, the high PPV here partly reflects the large proportion of poorly controlled patients; cut-offs may need calibration across settings with different case-mix and background inflammation.

Pathophysiological context

Chronic, low-grade inflammation is integral to T2DM pathogenesis—via stress-kinase (JNK) and NF-κB activation and a cytokine/adipokine milieu that promotes insulin resistance (10,11). Hyperglycaemia-driven neutrophil activation (including cytokine/ROS release and NET formation) alongside relative lymphocyte suppression provides a biologically coherent explanation for the observed elevation in NLR with worse control (12). Consistent elevations of NLR in T2DM versus non-diabetic controls in prior cohorts further support NLR as an inflammation-linked diabetes biomarker independent of overt leukocytosis (17,24–27,29,30).

Comparison with prior studies

Our results parallel those of Varma S et al. (positive NLR–HbA1c association) (23), Sefil F et al. (higher NLR with HbA1c>7%) (27), and Devamash GN et al. (stepwise NLR increase across HbA1c strata) (21). Studies by Akin S et al. and Mazhar H et al. similarly demonstrate higher NLR in poorly controlled subgroups (24,25). Overall, these findings support NLR as a simple and readily available inflammatory indicator of glycaemic status in type 2 diabetes mellitus.

Strengths and limitations

Strengths include an adequately powered sample for primary contrasts, standardized laboratory methods, and comprehensive profiling (glycaemic indices, full differential counts). Limitations are inherent to the cross-sectional, single-centre design; causal inference is not possible, and generalisability beyond similar settings may be limited. We excluded overt confounders (e.g., acute infection), yet residual confounding from subclinical inflammation, unrecorded medications, or intercurrent stressors may persist. Absence of non-diabetic controls limited between-population contextualisation, and we did not concurrently assay canonical inflammatory markers (e.g., CRP, IL-6, TNF-α) to triangulate pathways.

Implications and future directions

Given its low cost and widespread availability, NLR can serve as a complementary marker to help identify high-risk patients who require confirmatory testing and treatment intensification, particularly in resource-limited or high-volume clinical settings. (16,17,21). Prospective, longitudinal studies should test whether improving glycaemic control lowers NLR (and vice-versa), whether anti-inflammatory strategies favourably modulate both, and define clinically actionable NLR thresholds for risk stratification across diverse populations. Integrative models that pair NLR with HbA1c, fasting/post-prandial glucose, and (where feasible) CRP/IL-6/TNF-α may yield stronger predictive performance than any single marker alone. Finally, larger studies linking NLR to specific complications (retinopathy, nephropathy, cardiovascular events) are warranted to clarify its role in complication surveillance and targeted preventive care (16,21).

Conclusion

This study reveals a significant correlation between the Neutrophil-Lymphocyte Ratio (NLR) and glycemic control in patients with Type 2 Diabetes Mellitus (T2DM). Our findings reveal that NLR was significantly higher in participants with uncontrolled diabetes (HbA1c > 7.0%) compared to those with controlled diabetes, with corresponding higher neutrophil counts and lower lymphocyte counts. NLR is a moderate predictor of poor glycemic control in T2DM. Furthermore, NLR demonstrated a moderate positive correlation with HbA1c, fasting blood sugar, and postprandial blood sugar. The relationship between NLR and glycemic parameters, independent of age, gender, and hypertension status, highlights the potential utility of NLR as a simple, cost-effective marker that could complement traditional glycemic parameters in the assessment and monitoring of T2DM. As a readily available component of routine complete blood counts, NLR may serve as a valuable tool for evaluating inflammatory status and potentially predicting glycemic control, particularly in resource-limited settings where frequent HbA1c monitoring may be challenging.

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REFERENCES

1. Adane T, Melku M, Worku YB, Fasil A, Aynalem M, Kelem A, Getawa S. The association between neutrophil-to-lymphocyte ratio and glycemic control in type 2 diabetes mellitus: A systematic review and meta-analysis. J Diabetes Res. 2023 Jun 3;2023:3117396. Doi: 10.1155/2023/3117396. (n.d.).
2. Adnyani PY, Mahartini NN, Herawati S, Mulyantari NK, Lestari AAW. Comparison of neutrophil to lymphocyte Ratio (NLR) and lymphocyte to monocyte ratio (LMR) values in controlled and uncontrolled type 2 diabetes mellitus (T2DM) patient. Bali Med J. 2021;10(2):798-801. Doi: 10.15562/bmj.v10i2.2501. (n.d.).
3. Akin S, Aydin Z, Yilmaz G, Aliustaoglu M, Keskin O. Evaluation of the relationship between glycaemic regulation parameters and neutrophil-to- lymphocyte ratio in type 2 diabetic patients. Eur Med J Diabetes. 2019 Nov;7[1]:91-6. (n.d.).
4. Alvin CP, Kevin DN, Carmella E-M. Diabetes mellitus: Diagnosis, classification, and pathophysiology. In: Loscalzo J, Kasper DL, Longo DL, Fauci AS, Hauser SL, Jameson JL, editors. (n.d.).
5. American Diabetes Association Professional Practice Committee. 6. Glycemic Targets: Standards of Medical Care in Diabetes—2022. Diabetes Care. 2022 Jan 1;45(Suppl 1):S83-S96. (n.d.).
6. Barbu E, Mihaila A, Filippi A, Stoenescu A, Ciortan L, Butoi E, Beiu C, Popescu MN, Balanescu S. Stress, hyperglycemia, and insulin resistance correlate with neutrophil activity and impact acute myocardial infarction outcomes. Cureus. 2024 Jul 3;16(7):e63731. Doi: 10.7759/cureus.63731. PMID: 39100008; PMCID: PMC11295428. (n.d.).
7. Centers for Disease Control and Prevention (CDC). National Diabetes Fact Sheet: General Information and National Estimates on Diabetes in the United States. Atlanta (GA): US Department of Health and Human Services; 2007. (n.d.).
8. Devamsh GN, Parvathi M, Madhumathi R, Raghavan L. Study of neutrophil lymphocyte ratio in patient with type 2 diabetes mellitus and its correlation with glycemic control. Int J Adv Med. 2019 Oct; 6(5): 1637-41. (n.d.).
9. Duman TT, Aktas G, Atak BM, Kocak MZ, Erkus E, Savli H. Neutrophil to lymphocyte ratio as an indicative of diabetic control level in type 2 diabetes mellitus. Afr Health Sci. 2019 March;19(1):1602-06. (n.d.).
10. Elsayed AM, Araby E. Neutrophil-lymphocyte and platelet-lymphocyte ratios as a marker for diabetes control and complications. Benha Med J. 2021;38(3):984-95. Doi: 10.21608/bmfj.2021.89950.1453. (n.d.).
11. Emerging Risk Factors Collaboration, Sarwar N, Gao P, Seshasai SR, Gobin R, Kaptoge S, Di Angelantonio E, Ingelsson E, Lawlor DA, Selvin E, et al. Diabetes mellitus, fasting blood glucose concentration, and risk of vascular disease: A collaborative meta-analysis of 102 prospective studies. (n.d.).
12. Gebeyaw ED, Lema GD. Gender based difference in glycemic control and diabetes related chronic complications among type 2 diabetic patients in Debre Berhan city public hospitals. Metabol Open. 2025 Jan 23;25:100349. Doi: 10.1016/j.metop.2025.100349. PMID: 39944659; PMCID: PMC11816214. (n.d.).
13. Guo X, Zhang S, Zhang Q, Liu L, Wu H, Du H, Shi H, Wang C, Xia Y, Liu X, Li C, Sun S, Wang X, Zhou M, Huang G, Jia Q, Zhao H, Song K, Niu K. Neutrophil: Lymphocyte ratio is positively related to type 2 diabetes in a large-scale adult population: A Tianjin chronic low-grade systemic inflammation and health cohort study. Eur J Endocrinol. 2015 Aug;173(2):217-25. (n.d.).

14. Hirosumi J, Tuncman G, Chang L, Gorgun CZ, Uysal KT, Maeda K, Karin M, Hotamisligil GS. A central role for JNK in obesity and insulin resistance. Nature. 2002 Nov; 420:333-6. (n.d.).
15. Hu FB. Globalization of diabetes: The role of diet, lifestyle, and genes. Diabetes Care.2011 Jun;34(6):1249-57. (n.d.).
16. Hussain M, Babar MZM, Akhtar L, Hussain MS. Neutrophil lymphocyte ratio (NLR): A well assessment tool of glycemic control in type- 2 diabetic patients. Pak J Med Sci. 2017 Nov;33(6):1366-70. (n.d.).
17. Kankonkar NP, Dsouza BD, Kankonker NM, Vadivel D. Determinants of good glycemic control among individuals of type II diabetes mellitus seeking treatment at a rural health-care facility in Goa – a cross-sectional study. Indian J Public Health. 2023;67(4):550-53. Doi: 10.4103/ijph.ijph_1654_22. (n.d.).
18. Khan MAB, Hashim MJ, King JK, Govender RD, Mustafa H, Al Kaabi J. Epidemiology of type 2 diabetes – global burden of disease and forecasted trends. J Epidemiol Glob Health. 2020;10(1):107-11. (n.d.).
19. Mathur P, Leburu S, Kulothungan V. Prevalence, awareness, treatment and control of diabetes in India from the countrywide national NCD monitoring survey. Front Public Health. 2022 Mar 14;10:748157. Doi: 10.3389/fpubh.2022.748157. PMID: 35359772; PMCID: PMC8964146. (n.d.).
20. Nagabhushan BK, Geetha JP. Neutrophil lymphocyte ratio and platelet lymphocyte ratio: Novel markers in diabetes mellitus. J Med Sci 2019;5(2):31–3. (n.d.).
21. Nigussie S, Birhan N, Amare F, Mengistu G, Adem F, Abegaz TM. Rate of glycemic control and associated factors among type two diabetes mellitus patients in Ethiopia: A cross sectional study. PloS One. 2021;16(5):e0251506. (n.d.).
22. O'Connor PJ, Desai JR, Solberg LI, et al. Variation in diabetes care by age: Opportunities for customization of care. BMC Fam Pract. 2003;4:16. Doi: 10.1186/1471-2296-4-16. (n.d.).
23. Olefsky JM, Glass CK. Macrophages, inflammation, and insulin resistance. Annu Rev Physiol. 2010 March;72: 219-46. (n.d.).
24. Ozturk C, Balta S, Balta I, Demirkol S, Celik T, Turker T, et al. Neutrophil lymphocyte ratio and carotid intima media thickness in patients with Behcet disease without cardiovascular involvement. Angiology. 2015 Mar;66(3):291-96. Doi: 10.1177/0003319714527638. (n.d.).
25. Selfil F, Ulutas KT, Dokuyucu R, Sumbul AT, Yengil E, Yagiz AE, et al. Investigation of neutrophil lymphocyte ratio and blood glucose regulation in patients with type 2 diabetes mellitus. J Int Med Res. 2014 Apr;42(2):581-8. Doi:10.1177/0300060513516944. (n.d.).
26. Selvaraju V, Dharshini RP, Jayaganesh, Kaif M. Neutrophil to lymphocyte ratio as a ddenotative of type II diabetes mellitus. Saudi J Pathol Microbiol. 2023 Mar;8(3): 55-9. (n.d.).
27. Shiny A, Bibin YS, Shanthirani CS, Regin BS, Anjana RM, Balasubramanyam M, Jebarani S, Mohan V. Association of neutrophil-lymphocyte ratio with glucose intolerance: An indicator of systemic inflammation in patients with type 2 diabetes. Diabetes Technol Ther. 2014 Aug;16(8):524-30. Doi: 10.1089/dia.2013.0264. Epub 2014 Jan 23. PMID: 24455985. (n.d.).
28. The Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. Report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care. 2003;26(Suppl 1):S5-20. (n.d.).
29. Turkmen K, Guney I, Yerlikaya EH, Tonbul HZ. The relationship between neutrophil to lymphocyte ratio and inflammation in end stage renal disease patients. RenFail.2012;32(2):155-159. Doi: 10.3109/0886022X.2011.641514. (n.d.).
30. Varma S, Khande M, Gupta P, Archana, Khare RL, Malhotra Y, et al. Correlation of neutrophil lymphocyte ratio with HbA1c in patients of type 2 diabetes mellitus. Int J Adv Med. 2021 Jul;8(7):952-56. (n.d.).