

Correlation of an Emerging Inflammatory Marker Neutrophil Lymphocyte Ratio with Obesity Markers among Medical Students: A Cross-sectional Study

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ABSTRACT

Introduction: Obesity contributes to chronic low-grade systemic inflammation and increases the risk of non-communicable diseases such as cardiovascular disease, diabetes, and hypertension. The Neutrophil-to-Lymphocyte Ratio (NLR) is a simple and accessible marker that reflects the balance between innate and adaptive immunity, thereby serving as an indicator of early systemic inflammation. Although elevated NLR has been associated with metabolic and cardiovascular disorders, limited evidence is available on its significance in healthy young adults, particularly among medical students. This highlights the need to explore its relationship with obesity markers in this population.

Aim: To assess the relationship between obesity markers and NLR among first-year medical students.

Materials and Methods: The present cross-sectional study was conducted at a rural medical college in southern India and included 202 first-year undergraduate medical students who provided written informed consent. Demographic details and medical history were recorded. Anthropometric measurements, including height, weight, and waist circumference, were taken using standardised methods. Body Mass Index (BMI)

was calculated and classified according to World Health Organisation (WHO) Asian criteria. Venous blood samples were collected for complete blood count, and NLR was calculated by dividing the absolute neutrophil count by the lymphocyte count. Associations were analysed using Pearson correlation in GraphPad Prism version 10.60.

Results: Mean NLR values increased across BMI categories: 1.98 in normal weight, 2.2 in overweight, and 2.5 in obese participants ($p < 0.0001$). BMI showed a strong positive correlation with NLR ($r = 0.604$, $p < 0.0001$), while waist circumference also demonstrated a significant correlation ($r = 0.542$, $p < 0.0001$). Total leukocyte count did not differ significantly between BMI groups.

Conclusion: NLR increases with rising BMI and waist circumference, indicating subclinical inflammatory changes in healthy young adults. Monitoring NLR may serve as a practical and early marker of systemic inflammation, helping to guide timely preventive interventions for at-risk young individuals. Screening for NLR in obese individuals can aid in the early identification of inflammatory risk.

Keywords: Body mass index, Obesity, Systemic inflammation, Waist circumference

INTRODUCTION

Neutrophils constitute the majority of circulating leukocytes and act as the first line of defense against infection, playing a central role in acute and chronic inflammatory responses, including tissue repair and modulation of adaptive immunity [1]. Lymphocytes mediate adaptive immunity through targeted cellular and humoral responses. The Neutrophil-to-Lymphocyte Ratio (NLR), calculated by dividing the absolute neutrophil count by the absolute lymphocyte count, is a simple, inexpensive, and sensitive marker of systemic inflammation, reflecting the balance between innate and adaptive immune pathways [2]. NLR is a practical biomarker for detecting low-grade inflammation in both clinical and community settings. Reference ranges for NLR in healthy adults are reported to vary between 1.0 and 3.0, with higher values associated with increased cardiometabolic risk [2].

Obesity has reached pandemic proportions worldwide, driven by the availability of inexpensive calorie-dense foods, reduced physical activity, and sedentary lifestyles [3]. According to the WHO, the prevalence of obesity has nearly tripled since 1975, with one in three adults overweight and one in 10 obese. In India, the National Family Health Survey-5 indicates that approximately one in four adults meets the criteria for obesity [4]. Obesity is strongly associated with insulin resistance, type 2 diabetes, dyslipidemia, hypertension, and cardiovascular disease, all of which contribute to increased

morbidity and mortality [5,6]. Chronic low-grade inflammation resulting from excess adipose tissue plays a significant role in these metabolic complications, making inflammatory biomarkers valuable for early risk assessment [1,2,5]. Although extensive data exist in older adults, information on early inflammatory changes in young adults with excess adiposity remains limited [3-6].

Medical students represent a unique high-risk group for weight gain and metabolic disturbances. Academic stress, irregular sleep, long study hours, and easy access to fast foods and sugary drinks contribute to reduced physical activity and positive energy balance [7]. Several surveys in India and abroad have documented rising rates of overweight and obesity in this population, emphasising the importance of preventive strategies during the formative years of medical training [3,4,7]. Obesity in young adults not only predicts future cardiovascular and metabolic diseases but also has immediate effects on quality of life and academic performance. Understanding inflammatory changes in this subgroup could help guide early lifestyle interventions and health-promotion programs [7].

Inflammatory markers, including NLR, have been investigated in diverse clinical contexts. Elevated NLR has been associated with cardiovascular disease and adverse cardiac events, supporting its role as a predictor of atherosclerotic burden and prognosis [8]. Similarly, higher NLR correlates with poor glycaemic control in diabetes and with adverse outcomes in malignancies, underscoring

its broad clinical utility [9,10]. These findings suggest that NLR integrates multiple inflammatory pathways involved in metabolic dysregulation, providing insight into the interplay between immune activation and chronic disease.

However, data on the relationship between obesity and NLR in apparently healthy young adults, particularly Indian medical students, remain scarce. Regional dietary habits, lifestyle patterns, and genetic predispositions can influence both adiposity and systemic inflammation, highlighting the need for population-specific research. Studies assessing whether common obesity indicators, such as BMI and waist circumference, predict changes in NLR in this population are limited. This study evaluated the association between NLR and obesity markers in first-year medical students in southern India to determine whether higher BMI and waist circumference correspond to elevated NLR, thereby providing an accessible marker of subclinical inflammation and future cardiometabolic risk.

MATERIALS AND METHODS

The present cross-sectional study was conducted in the department of Physiology at a rural medical college southern India over a period of six months from July 2023 to January 2024 after approval from the Institutional Ethics Committee (IEC No. AIMS/IEC/019/2023; dated 08 July 2023).

Sample size calculation: A priori sample size was calculated using G*Power software (version 3.1) based on an expected moderate correlation (r=0.25) between BMI and NLR, with 80% power and α=0.05, yielding a minimum requirement of 180 participants. To account for potential attrition, 202 students were recruited.

Inclusion and Exclusion criteria: Apparently healthy volunteers aged 18-21 years were eligible for inclusion. Individuals with chronic illnesses, recent infections within the preceding three months, or use of medications that could influence complete blood count values were excluded.

Study Procedure

Investigators explained the objectives and procedures to all potential participants, and written informed consent was obtained before enrolment. A total of 202 first-year medical students, both male and female, were randomly selected according to the inclusion criteria. Participant privacy and confidentiality were maintained throughout the study. A structured questionnaire was used to capture lifestyle information, including physical activity typical dietary pattern (24-hour recall), average nightly sleep duration, and perceived stress.

Anthropometric measurements: Demographic details and medical history were recorded. Height was measured with participants standing barefoot against a wall, and weight was obtained using a digital scale. BMI was calculated using the Quetelet index. Waist circumference was measured at the midpoint between the lower rib margin and the iliac crest with a non-stretchable tape. BMI was categorised for Asian populations as normal (18.5-22.9 kg/m²), overweight (23-24.9 kg/m²), and obese (≥25 kg/m²) [11].

Blood pressure and laboratory analysis: Blood pressure was measured on the right arm in a sitting position after 5-10 minutes of rest using a sphygmomanometer and stethoscope. Two readings were taken five minutes apart and averaged [12]. Venous blood samples (2 mL) were collected under aseptic conditions, and complete blood counts, including neutrophil and lymphocyte counts, were analysed using a fully automated haematology analyser (Horiba Yumizen H550). NLR was calculated from these values. Laboratory quality control included daily calibration of the analyser using manufacturer-provided standards, duplicate sampling for 10% of specimens, and blinded re-analysis of discrepant results to ensure accuracy.

STATISTICAL ANALYSIS

Data were entered into Microsoft Excel and analysed using GraphPad Prism. Numerical variables were expressed as mean±standard deviation. One-way ANOVA was used to compare Total Leukocyte Count (TLC) and NLR across BMI groups. Pearson's correlation assessed relationships between continuous variables. Lifestyle variables such as physical activity, diet quality score, sleep duration, and stress score were evaluated as potential confounders using partial correlation and multivariable linear regression. A p-value <0.05 was considered statistically significant.

RESULTS

Total 202 medical students were included with a mean age of 19.15±0.83 years. The average BMI was 23.26±3.00 kg/m², indicating a mixture of normal, overweight, and obese individuals, while the mean waist circumference was 84.35±7.18 cm. The mean TLC was 7,553±1,767/mm³, and the mean NLR was 2.29±0.46, within the normal reference range. Blood pressure measurements showed a mean Systolic BP (SBP) of 114.28±9.72 mmHg and a mean Diastolic BP (DBP) of 72.5±7.54 mmHg. These baseline values indicate that the cohort comprised generally healthy young adults with varying obesity markers [Table/Fig-1].

Basic parameters	Mean	Standard deviation
Age (years)	19.15	0.83
BMI (kg/m²)	23.26	3.001
WC (cm)	84.35	7.18
TLC (count/mm³)	7553	1767
NLR	2.29	0.46
SBP (mmHg)	114.28	9.72
DBP (mmHg)	72.5	7.54

[Table/Fig-1]: Basic parameters of subjects.

[Table/Fig-2] shows that the mean TLC did not differ significantly among normal-weight (7,397 cells/cumm), overweight (7,751 cells/cumm), and obese (7,690 cells/cumm) groups (F=0.82, p=0.441). In contrast, NLR increased progressively with BMI: 1.98 in normal-weight, 2.20 in overweight, and 2.50 in obese participants, showing a highly significant difference (F=36.28, p<0.0001**). This indicates that NLR rises with increasing adiposity, while overall leukocyte count remains relatively stable.

Parameters	Normal weight (n=99)	Overweight (n=46)	Obese (n=57)	F value	p-value
TLC (Cells/cumm)	7397	7751	7690	0.82	0.441
NLR	1.98	2.2	2.5	36.28	<0.0001**

[Table/Fig-2]: Comparison of NLR, TLC between BMI categories.

*Data presented as Means, F value of ANOVA test, p-value, *shows statistically significant

[Table/Fig-3] shows the correlation of obesity markers (BMI and WC) with NLR, TLC, and blood pressure (SBP and DBP). TLC demonstrated a weak, non-significant correlation with BMI (r=0.08, p=0.205) and WC (r=0.092, p=0.192). NLR showed a strong positive correlation with both BMI (r=0.604, p<0.0001*) and WC (r=0.542, p<0.0001). Systolic BP was strongly correlated with BMI (r=0.54, p<0.0001) and WC (r=0.467, p<0.0001), while diastolic BP

Study parameters	Obesity Markers			
	BMI		WC	
	r value	p-value	r value	p-value
TLC	0.08	0.205	0.092	0.192
NLR	0.604	<0.0001**	0.542	<0.0001**
SBP	0.54	<0.0001**	0.467	<0.0001**
DBP	0.245	0.0004**	0.267	0.0001**

[Table/Fig-3]: Correlation of obesity markers with NLR, TLC, SBP, DBP.

Data presented as Pearson's correlation test r value and p-value, * shows statistically significant

showed moderate positive correlations (BMI: $r=0.245$, $p=0.0004$; WC: $r=0.267$, $p=0.0001^*$). These findings indicate that both NLR and blood pressure increase with higher adiposity.

DISCUSSION

This study showed that participants had an average BMI of 23.26 ± 3.001 kg/m², representing a spectrum from normal weight to obese. Analysis of TLC and NLR across BMI categories revealed that TLC remained relatively unchanged, whereas NLR increased progressively from normal weight to overweight and obese groups (1.98, 2.2, and 2.5, respectively; $p<0.0001$). These observations indicate that NLR is a sensitive marker for detecting subclinical inflammation associated with obesity. However, some community-based cohorts have reported weaker or non-significant associations between NLR and BMI after adjusting for lifestyle factors, suggesting that the strength of this relationship may vary across populations [12].

NLR and Obesity Markers

The study observed a progressive increase in NLR among overweight and obese participants, indicating higher systemic inflammation with increasing adiposity. Previous research supports this pattern. NLR has been shown to correlate positively with metabolic syndrome severity in obese adults, although such associations are less consistent in children and adolescents [12]. Studies in adolescents demonstrated significantly higher NLR in obese individuals compared to controls, suggesting that low-grade inflammation develops early with excess weight [13]. Similarly, research in women found elevated NLR in obese subjects compared with non-obese counterparts, confirming that adiposity drives inflammatory activity [14]. A multicenter European analysis reported only a borderline elevation of NLR in young adults after controlling for smoking and physical activity, highlighting the potential confounding effect of lifestyle behaviours [13,14].

In this study, NLR demonstrated strong positive correlations with BMI ($r=0.604$, $p<0.0001$) and WC ($r=0.542$, $p<0.0001$), indicating that both overall and abdominal obesity contribute to systemic inflammation. Central fat accumulation appeared particularly influential, as visceral adiposity has been associated with higher NLR in adults when measured by waist-to-height ratio [15]. These findings suggest that NLR reflects inflammatory changes linked to both generalised and abdominal obesity, even in young, apparently healthy populations. Monitoring NLR can therefore, provide an accessible measure of early obesity-related inflammation and help identify individuals at risk of metabolic complications.

Mechanisms of Obesity-related Inflammation

Obesity-associated increases in NLR result from adipose tissue remodelling and immune cell activation. Hypertrophied adipocytes increase the diffusion distance from capillaries, leading to local hypoxia. Hypoxia, in turn, triggers fibrosis, necrotic adipocyte accumulation, and macrophage infiltration, elevating the production of pro-inflammatory cytokines such as TNF- α and IL-6 [16,17]. This chronic low-grade inflammation is a hallmark of obesity. In the present study, higher BMI and WC were associated with elevated NLR, indicating early inflammatory changes in young adults. The range of NLR observed (mean values approaching 2.5 in obese participants) lies near the upper limit of the reported normal range (1.0-3.0) and overlaps thresholds associated with heightened cardiovascular risk in longitudinal cohorts, suggesting possible clinical relevance for early intervention [16,17].

Blood pressure correlations further reinforced the systemic impact of obesity-related inflammation. Both SBP and DBP correlated positively with BMI and WC, demonstrating that adiposity contributes to cardiovascular stress. Elevated NLR reflects systemic low-grade inflammation associated with increased metabolic burden and early vascular changes. This inflammatory state contributes to endothelial

dysfunction, insulin resistance, and heightened cardiometabolic risk, even in asymptomatic young adults.

The findings therefore establish a clear relationship between NLR, obesity markers, and blood pressure, confirming that subclinical inflammation accompanies early indicators of cardiometabolic stress [18-20]. Monitoring NLR can help identify young adults at risk and guide early preventive strategies to mitigate future cardiovascular and metabolic complications.

Evidence from Healthy Populations

Recent studies support the use of NLR as a marker of systemic inflammation in healthy adults with obesity. Elevated NLR shows significant correlations with anthropometric measures, indicating that both generalised and central adiposity contribute to low-grade inflammation [21]. Higher NLR also predicts type 2 diabetes in morbidly obese patients, suggesting its value as an early metabolic risk indicator [22]. In obese children, NLR and other inflammatory markers correlate with non-alcoholic fatty liver disease, showing that subclinical inflammation precedes metabolic disorders [23]. Elevated NLR and delta neutrophil index in obese women with polycystic ovary syndrome further demonstrate that shifts in neutrophil-lymphocyte balance sensitively reflect metabolic stress [24].

These findings indicate that obesity, even in young and otherwise healthy individuals, is associated with measurable systemic inflammation. Observations across adolescents, adults, and women with endocrine or metabolic disorders confirm that NLR is a practical, accessible biomarker for the early detection of obesity-related inflammatory changes. The present study adds regional context by evaluating first-year medical students in southern India, a population at elevated risk of obesity due to sedentary lifestyles and dietary habits [7]. The influence of daily physical activity, diet quality, sleep duration, and psychosocial stress factors known to modulate systemic inflammation—could not be fully disentangled and may partly explain interstudy variability [21-24]. These findings strengthen the role of NLR in recognising at-risk young adults and guiding early preventive measures to reduce long-term cardiometabolic complications.

The study shows that NLR can detect early inflammatory changes before clinical disease appears. Total leukocyte count did not vary significantly across BMI categories, indicating that NLR reflects shifts in immune cell balance rather than overall leukocytosis. Intervention trials in adults have shown that lifestyle modification can reduce NLR, suggesting that student health programs could use routine NLR monitoring as a practical trigger for counselling when values exceed approximately 2.5, particularly in individuals with additional metabolic risk factors. Elevated NLR in young adults may therefore indicate future metabolic and cardiovascular risk. Including NLR in student health screenings offers a low-cost, routine method for early detection.

Limitation(s)

However, limitations of the present study include its single-centre design, exclusive focus on first-year medical students, and modest sample size, which reduce generalisability. Residual confounding from unmeasured or self-reported lifestyle factors (physical activity, diet composition, sleep quality, stress) remains possible despite adjustment attempts, warranting cautious interpretation of the observed associations. Multicentre, longitudinal studies across different age groups, incorporating additional inflammatory markers such as hs-CRP and IL-6, along with body composition analysis using DEXA or bioimpedance, would strengthen the evidence for NLR as a predictive biomarker.

CONCLUSION(S)

The study demonstrates that NLR identifies early inflammatory changes before clinical disease develops. Total leukocyte count

remained similar across BMI categories confirming that NLR reflects shifts in immune cell balance rather than overall leukocytosis. Elevated NLR in young adults may signal future metabolic and cardiovascular risk. Incorporating NLR into student health screenings provides a low-cost, practical approach for early detection. Thus, these findings highlight NLR as a valuable biomarker for the proactive identification of at-risk young adults, enabling timely preventive strategies to mitigate long-term cardiometabolic complications. Despite limitations such as a single-centre design, focus on first-year medical students, and modest sample size, the results are promising. Future multicentre, longitudinal studies across diverse age groups, incorporating additional inflammatory markers (e.g., hs-CRP and IL-6) and body composition assessments (e.g., DEXA or bioimpedance), are needed to reinforce the evidence for NLR as a predictive biomarker.

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