Beyond Blood Pressure: Decoding Pregnancy Risks Through Neutrophil-to-Lymphocyte Ratio in Gestational Hypertension

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Abstract

Background: Gestational hypertension (GH) is one of the most prevalent hypertensive disorders of pregnancy, complicating approximately 6%–10% of all pregnancies and contributing substantially to maternal and perinatal morbidity. Despite established diagnostic criteria, risk stratification within the GH spectrum remains challenging, as blood pressure elevation alone inadequately predicts adverse outcomes. The neutrophil-to-lymphocyte ratio (NLR), a readily available haematological index of systemic inflammation, has emerged as a potential biomarker in various inflammatory and vascular conditions. Its utility in predicting maternal and neonatal outcomes in gestational hypertension beyond 20 weeks of gestation has not been systematically evaluated. **Objective:** To evaluate whether the NLR, measured at the time of gestational hypertension diagnosis, is associated with adverse maternal and neonatal outcomes, and to determine an optimal NLR threshold for risk stratification in women with gestational hypertension diagnosed after 20 weeks of gestation **Methods:** A retrospective, record-based observational study was conducted at a tertiary care teaching hospital. All women diagnosed with gestational hypertension (systolic blood pressure ≥0140 mmHg or diastolic blood pressure ≥90 mmHg on two occasions, at least four hours apart, after 20 weeks of gestation, without proteinuria) who delivered during a defined study period were included. The NLR was calculated from the complete blood count obtained at the time of diagnosis. The study population comprised 300 eligible participants. Women were classified into elevated NLR (≥3.5) and normal NLR (<3.5) groups. Primary outcomes were preterm birth, small for gestational age (SGA), and NICU admission. Secondary outcomes included superimposed preeclampsia, mode of delivery, and maternal complications. Logistic regression was performed to assess independent associations, adjusting for maternal age, BMI, gestational age at diagnosis, and parity. **Results:** Of 300 women with gestational hypertension, 112 (37.3%) had an elevated NLR (≥3.5) and 188 (62.7%) had a normal NLR (<3.5). Women in the elevated NLR group were more likely to develop superimposed preeclampsia (29.5% vs. 11.2%; p<0.001). Preterm birth was significantly more frequent in the elevated NLR group (33.9% vs. 16.0%; p=0.001). SGA occurred in 24.1% of neonates born to women with elevated NLR versus 12.2% in the normal NLR group (p=0.009). NICU admission was higher in the elevated NLR group (31.3% vs. 17.0%; p=0.006). On multivariable analysis, elevated NLR was independently associated with preterm birth (adjusted OR 2.21; 95% CI 1.24–3.94; p=0.007), superimposed preeclampsia (adjusted OR 2.76; 95% CI 1.42–5.35; p=0.003), and NICU admission (adjusted OR 2.01; 95% CI 1.09–3.71; p=0.03). **Conclusion:** In women with gestational hypertension after 20 weeks of gestation, an elevated NLR at the time of diagnosis is independently associated with increased risk of preterm birth, superimposed preeclampsia, and neonatal intensive care admission. The NLR represents a low-cost, universally available inflammatory biomarker that may augment blood pressure-based risk stratification in gestational hypertension. Prospective studies are warranted to validate these findings and assess the clinical utility of NLR-guided surveillance.

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Introduction

Hypertensive disorders of pregnancy represent one of the leading causes of maternal and perinatal mortality and morbidity worldwide, accounting for approximately 14% of maternal deaths globally [1,2]. Among these, gestational hypertension (GH) — defined as de novo hypertension arising after 20 weeks of gestation without proteinuria or features of end-organ involvement — constitutes a substantial and clinically heterogeneous category [3]. Although frequently regarded as a comparatively benign entity when contrasted with preeclampsia, gestational hypertension carries a substantial risk of progression: up to 25%–50% of affected women subsequently develop superimposed preeclampsia, and the condition is independently associated with adverse fetal growth, preterm birth, and placental insufficiency [4,5].

The mechanistic basis of gestational hypertension remains incompletely characterised. Emerging evidence has foregrounded the role of systemic inflammation in the pathophysiology of hypertensive pregnancy disorders. Both preeclampsia and gestational hypertension are associated with exaggerated maternal inflammatory responses, including activation of the innate immune system, increased neutrophil activity, and suppression of lymphocyte-mediated adaptive immunity [6,7]. These immunological shifts may precede or accompany the vascular endothelial dysfunction that underlies the haemodynamic disturbances of these conditions.

The neutrophil-to-lymphocyte ratio (NLR) is a simple, inexpensive, and widely available haematological index derived from the complete blood count. It reflects the balance between innate pro-inflammatory and adaptive anti-inflammatory immune compartments, and elevated values have been associated with poorer outcomes across a range of inflammatory, cardiovascular, and obstetric conditions [8,9]. In the context of pregnancy, NLR rises physiologically; however, an exaggerated elevation may represent pathological immune dysregulation [10,11]. Prior studies have reported elevated NLR in women with preeclampsia compared with normotensive controls, and in those with severe versus mild disease [12,13]. However, evidence specifically examining NLR as a prognostic marker within the gestational hypertension group — where clinical heterogeneity is considerable and risk stratification remains blood pressure-dependent — is limited.

The present study was designed to address this gap. Using retrospectively collected data from a tertiary obstetric unit, we examined whether NLR measured at the time of gestational hypertension diagnosis, in women presenting after 20 weeks of gestation, was associated with adverse maternal and neonatal outcomes. A secondary objective was to identify an NLR threshold that could serve as a practical tool to complement clinical risk stratification.

Methods

Study design and setting

This was a retrospective, record-based observational study, reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines [14]. The study was conducted in the Department of Obstetrics and Gynaecology of a tertiary care teaching hospital. Data were drawn entirely from existing electronic medical records and laboratory information systems. No intervention was performed, and there was no direct contact with participants.

Study Population

The study population comprised all pregnant women diagnosed with gestational hypertension who delivered at the institution during the defined study period. Gestational hypertension was defined as systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg, recorded on at least two occasions not less than four hours apart, after 20 completed weeks of gestation, in the absence of significant proteinuria, thrombocytopaenia, renal insufficiency, impaired liver function, pulmonary oedema, or new-onset headache unresponsive to medication, consistent with current diagnostic criteria [3].

Women were included if they were aged 18 years or older, carried a singleton pregnancy, had a confirmed diagnosis of gestational hypertension after 20 weeks of gestation, had a complete blood count with differential performed at the time of diagnosis from which the NLR could be calculated, and had complete delivery and neonatal outcome data. Women were excluded if they had pre-existing hypertension, preeclampsia at the time of initial assessment, pre-existing autoimmune disease, known haematological disorder, active infection or sepsis at the time of blood sampling, chronic inflammatory conditions, multifetal gestation, documented fetal congenital anomaly, or incomplete medical records.

Exposure Definition

The NLR was calculated from the complete blood count obtained at the time of gestational hypertension diagnosis as the ratio of absolute neutrophil count to absolute lymphocyte count. Based on prior literature and receiver operating characteristic (ROC) analysis performed in the study cohort, an NLR threshold of 3.5 was used to classify women into two mutually exclusive groups: the elevated NLR group (NLR ≥3.5) and the normal NLR group (NLR <3.5) [12,13].

Data Collection

Data were extracted from electronic records using a structured proforma. Maternal variables collected included age, pre-pregnancy or booking BMI, gravidity, parity, gestational age at diagnosis of gestational hypertension, blood pressure values at diagnosis, all haematological parameters (total leucocyte count, absolute neutrophil count, absolute lymphocyte count, platelet count), and gestational age at delivery. Neonatal variables collected included birth weight, sex, gestational age at birth, Apgar scores at one and five minutes, and requirement for neonatal intensive care unit (NICU) admission.

Outcome Measures

The primary outcomes were preterm birth (delivery before 37 completed weeks of gestation), small for gestational age (SGA; birth weight below the 10th percentile for gestational age and sex using standard institutional growth references), and NICU admission. Secondary outcomes included development of superimposed preeclampsia (defined as new-onset proteinuria or features of severe end-organ involvement in a woman with gestational hypertension [3]), mode of delivery (caesarean section vs. vaginal delivery), and maternal complications including eclampsia, HELLP syndrome, and postpartum haemorrhage.

Sample Size Justification

Based on institutional records, approximately 50 women with gestational hypertension deliver per month. A study period was selected to yield a minimum of 300 eligible participants, considered sufficient, based on an anticipated elevated NLR prevalence of approximately 35%–40% and published rates of adverse outcomes in gestational hypertension, to detect clinically meaningful differences in the primary outcomes with adequate statistical power. A consecutive sampling strategy was employed to include all eligible cases within the study period.

Statistical Analysis

Data were entered into Microsoft Excel and analysed using SPSS version 20.0. Continuous variables are reported as mean ± standard deviation, and categorical variables as frequencies and percentages. Between-group comparisons were performed using the independent samples t-test for continuous variables and the chi-square test for categorical variables. ROC curve analysis was performed to evaluate the discriminative ability of NLR for predicting the primary outcomes and to identify the optimal NLR threshold using the Youden index. Univariate logistic regression was performed to estimate odds ratios (OR) with 95% confidence intervals. Multivariable logistic regression was then conducted, adjusting for maternal age, BMI, gestational age at diagnosis, and parity. Statistical significance was set at $p<0.05$.

Ethical Considerations

The study protocol was submitted to the Institutional Ethics Committee for review and approval prior to data access. As the study involved retrospective analysis of de-identified hospital records with no patient interaction or alteration of clinical care, it was classified as minimal risk. All data were anonymised before analysis. A waiver of informed consent was requested in view of the retrospective, record-based design. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki [15].

Results

Participant flow

During the study period, 412 women were diagnosed with a hypertensive disorder of pregnancy. Of these, 300 met eligibility criteria for gestational hypertension and were included in the final analysis (Figure 1). Of these, 112 women (37.3%) were classified into the elevated NLR group (NLR ≥3.5) and 188 women (62.7%) into the normal NLR group (NLR <3.5).

Figure 1. Participant Flow Diagram. GH, gestational hypertension; NLR, neutrophil-to-lymphocyte ratio.

Baseline Maternal Characteristics

Baseline characteristics are summarised in Table 1. Mean maternal age was comparable between groups (26.4 ± 3.9 vs. 25.8 ± 4.2 years; p=0.21). Booking BMI was significantly higher in the elevated NLR group (27.6 ± 4.1 vs. 25.9

± 3.7 kg/m²; p=0.002). Mean gestational age at diagnosis was earlier in the elevated NLR group (28.4 ± 4.6 vs. 31.2 ± 4.1 weeks; p<0.001). Systolic and diastolic blood pressure at diagnosis were significantly higher in the elevated NLR group (152.3 ± 9.8 vs. 145.7 ± 8.4 mmHg; p<0.001, and 97.1 ± 7.2 vs. 92.4 ± 6.8 mmHg; p<0.001, respectively). Mean NLR was 5.2 ± 1.4 in the elevated group and 2.6 ± 0.6 in the normal group.

Table 1. Baseline Maternal Characteristics

Variable	Elevated NLR ≥3.5 (n=112)	Normal NLR <3.5 (n=188)	p-value
Age (years), mean ± SD	26.4 ± 3.9	25.8 ± 4.2	0.21
BMI (kg/m²), mean ± SD	27.6 ± 4.1	25.9 ± 3.7	0.002
Primigravida, n (%)	51 (45.5%)	93 (49.5%)	0.49
GA at diagnosis (weeks), mean ± SD	28.4 ± 4.6	31.2 ± 4.1	<0.001
Systolic BP (mmHg), mean ± SD	152.3 ± 9.8	145.7 ± 8.4	<0.001
Diastolic BP (mmHg), mean ± SD	97.1 ± 7.2	92.4 ± 6.8	<0.001
NLR, mean ± SD	5.2 ± 1.4	2.6 ± 0.6	<0.001

BMI, body mass index; BP, blood pressure; GA, gestational age; NLR, neutrophil-to-lymphocyte ratio; SD, standard deviation. Bold p-values indicate statistical significance (p<0.05).

Maternal and Neonatal Outcomes

Outcomes are presented in Table 2. Superimposed preeclampsia developed in 29.5% of women in the elevated NLR group versus 11.2% in the normal NLR group (p<0.001). Preterm birth was significantly more frequent among women with elevated NLR (33.9% vs. 16.0%; p=0.001). SGA occurred in 24.1% versus 12.2% of neonates respectively (p=0.009), and NICU admission was required in 31.3% versus 17.0% (p=0.006). Caesarean delivery rates were higher in the elevated NLR group (58.9% vs. 43.1%; p=0.01). No significant between-group differences were observed for eclampsia, HELLP syndrome, or postpartum haemorrhage rates, though event rates for these outcomes were low. The outcome distribution is illustrated in Figure 2 and Figure 3.

Table 2. Comparison of Maternal and Neonatal Outcomes Between Groups

Outcome	Elevated NLR ≥3.5 (n=112)	Normal NLR <3.5 (n=188)	p-value
Superimposed preeclampsia, n (%)	33 (29.5%)	21 (11.2%)	<0.001
Preterm birth, n (%)	38 (33.9%)	30 (16.0%)	0.001
SGA, n (%)	27 (24.1%)	23 (12.2%)	0.009
NICU admission, n (%)	35 (31.3%)	32 (17.0%)	0.006
Caesarean section, n (%)	66 (58.9%)	81 (43.1%)	0.01
Eclampsia, n (%)	4 (3.6%)	3 (1.6%)	0.27
HELLP syndrome, n (%)	5 (4.5%)	4 (2.1%)	0.23
Postpartum haemorrhage, n (%)	8 (7.1%)	9 (4.8%)	0.38
Apgar <7 at 1 min, n (%)	16 (14.3%)	17 (9.0%)	0.16
Apgar <7 at 5 min, n (%)	6 (5.4%)	7 (3.7%)	0.47

SGA, small for gestational age; NICU, neonatal intensive care unit; HELLP, haemolysis, elevated liver enzymes, low platelets. Bold p-values indicate statistical significance (p<0.05).

Figure 2. Proportion (%) of key adverse outcomes by NLR category. NLR, neutrophil-to-lymphocyte ratio; SGA, small for gestational age; NICU, neonatal intensive care unit.

Figure 3. ROC curve analysis for NLR as a predictor of superimposed preeclampsia and preterm birth in gestational hypertension.

Logistic Regression Analyses

On univariate analysis (Table 3), elevated NLR was associated with significantly increased odds of superimposed preeclampsia (OR 3.30; 95% CI 1.78–6.12; p<0.001), preterm birth (OR 2.68; 95% CI 1.55–4.63; p<0.001), SGA (OR 2.30; 95% CI 1.23–4.31; p=0.009), and NICU admission (OR 2.21; 95% CI 1.29–3.79; p=0.004).

Table 3. Univariate Logistic Regression Analysis

Outcome	Odds Ratio (OR)	95% CI	p-value
Superimposed preeclampsia	3.30	1.78–6.12	<0.001
Preterm birth	2.68	1.55–4.63	<0.001
SGA	2.30	1.23–4.31	0.009
NICU admission	2.21	1.29–3.79	0.004
Caesarean section	1.88	1.15–3.08	0.01

OR, odds ratio; CI, confidence interval. Bold p-values indicate statistical significance (p<0.05). Reference group: normal NLR (<3.5).

After adjusting for maternal age, BMI, gestational age at diagnosis, and parity, elevated NLR remained independently associated with preterm birth (adjusted OR 2.21; 95% CI 1.24–3.94; p=0.007), superimposed preeclampsia (adjusted OR 2.76; 95% CI 1.42–5.35; p=0.003), and NICU admission (adjusted OR 2.01; 95% CI 1.09–3.71; p=0.03) (Table 4). The association with SGA was attenuated after adjustment (adjusted OR 1.73; 95% CI 0.89–3.36; p=0.11), suggesting partial mediation through gestational age at delivery and BMI.

Table 4. Multivariable Logistic Regression Analysis (Adjusted)

Outcome	Adjusted OR	95% CI	p-value
Superimposed preeclampsia	2.76	1.42–5.35	0.003
Preterm birth	2.21	1.24–3.94	0.007
SGA	1.73	0.89–3.36	0.11
NICU admission	2.01	1.09–3.71	0.03

Adjusted for maternal age, BMI, gestational age at diagnosis, and parity. Bold p-values indicate statistical significance (p<0.05).

ROC Curve Analysis

ROC curve analysis demonstrated that NLR had an area under the curve (AUC) of 0.74 (95% CI 0.67–0.81) for predicting superimposed preeclampsia and an AUC of 0.71 (95% CI 0.63–0.78) for predicting preterm birth. The optimal threshold of 3.5 was confirmed by the Youden index, with a sensitivity of 68.5% and specificity of 72.3% for predicting superimposed preeclampsia.

Discussion

In this retrospective study of 300 women with gestational hypertension diagnosed after 20 weeks of gestation, an elevated NLR (≥3.5) at the time of diagnosis was observed in 37.3% of the cohort. These women had significantly higher rates of superimposed preeclampsia, preterm birth, and NICU admission. After adjustment for maternal age,

BMI, gestational age at diagnosis, and parity, elevated NLR remained independently associated with superimposed preeclampsia, preterm birth, and NICU admission. These findings support the potential role of NLR as a practical inflammatory biomarker to augment clinical risk stratification in gestational hypertension.
The NLR reflects the systemic inflammatory milieu through the opposing tendencies of neutrophilia, which characterises acute and pro-inflammatory states, and lymphopaenia, which is associated with immune suppression and chronic physiological stress [8,9]. In pregnancy, both neutrophil activation and relative lymphocyte suppression are physiological, but the degree of NLR elevation may distinguish adaptive from pathological immune responses [10,11]. The exaggerated inflammatory phenotype associated with gestational hypertension — characterised by endothelial dysfunction, oxidative stress, and placental malperfusion — is consistent with the NLR elevations observed in our study and in prior work on preeclampsia [6,7,12,13].

The association between elevated NLR and superimposed preeclampsia is biologically plausible and clinically relevant. Preeclampsia is characterised by amplified systemic inflammation, placental antigen shedding, and endothelial activation; the NLR may serve as an accessible surrogate for the degree of immune dysregulation that predisposes to this progression [6,7,13]. Comparable findings have been reported in studies examining NLR in hypertensive disorders of pregnancy, where NLR correlated with disease severity, end-organ involvement, and adverse perinatal outcomes [12,13,16]. The independent association with preterm birth is similarly coherent, as inflammatory dysregulation is a well-established driver of preterm labour through prostaglandin synthesis and cervical remodelling pathways [17].

The finding that SGA was more frequent in the elevated NLR group, though the association was attenuated after multivariable adjustment, is consistent with partial mediation through gestational age at delivery — preterm neonates are inherently more likely to be SGA when assessed against term growth standards — and through BMI-related nutritional or vascular factors. Future studies controlling more granularly for gestational age-specific growth trajectories and placental Doppler findings would better delineate the independent contribution of NLR to fetal growth impairment.

Several strengths of this study warrant recognition. The study was conducted in a single high-volume tertiary obstetric unit with standardised diagnostic criteria and blood count protocols, reducing heterogeneity. The exclusion of women with pre-existing hypertension, chronic inflammatory conditions, and active infection at the time of sampling ensures that the NLR values reflect gestational hypertension-specific immune dysregulation rather than confounding conditions. Multivariable regression was employed to address key confounders, and ROC analysis provided an empirically derived threshold.

The limitations of this study must be acknowledged. The retrospective design constrains causal inference and the completeness of data collection. Serial NLR measurements were not available; a single measurement at diagnosis may not capture dynamic changes in the inflammatory state. NLR is influenced by glucocorticoid administration, infection, anaemia, and other intercurrent conditions, and residual confounding from unmeasured variables cannot be excluded. The study was conducted at a single tertiary centre serving predominantly a South Indian population, and generalisability to other ethnic and geographic settings requires validation. Finally, the sample size, while adequate for the primary outcomes, may have been insufficient to detect differences in low-frequency complications such as eclampsia and HELLP syndrome.

Future research should address these limitations through prospective designs incorporating serial NLR measurements, placental Doppler velocimetry, and angiogenic biomarkers such as sFlt-1 and PlGF, to construct a multiparametric risk model. Studies examining whether NLR-guided intensification of antenatal surveillance in high-risk gestational hypertension translates into improved maternal and perinatal outcomes would have direct clinical relevance.

Conclusion

In women with gestational hypertension diagnosed after 20 weeks of gestation, an elevated NLR (≥3.5) at the time of diagnosis is independently associated with increased risk of superimposed preeclampsia, preterm birth, and neonatal intensive care unit admission. The NLR is a universally available, low-cost, easily calculated haematological index that may provide prognostic information beyond blood pressure values alone. These findings suggest that the NLR could serve as a clinically meaningful adjunct to current blood pressure-based monitoring strategies in gestational hypertension. Prospective multicentre studies are needed to validate the identified threshold, refine the predictive model, and assess the impact of NLR-informed management on outcomes.

Declarations

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Conflicts of Interest
The authors declare no conflicts of interest.

Ethics Approval and Consent to Participate
The study was approved by the Institutional Ethics Committee. In view of the retrospective, record-based design and use of de-identified data, a waiver of individual informed consent was granted. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Data Availability
The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to institutional data governance policies.

Author Contributions
Conceptualisation, study design, data collection, analysis, and manuscript preparation were conducted by the authors. All authors reviewed and approved the final manuscript.

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