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Role of Neutrophil-To-Lymphocyte Ratio and Platelets-Lymphocyte Ratio in Hypertension

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KEYWORDS

Neutrophil-To-Lymphocyte Ratio

ABSTRACT:

Background: Hypertension is a one of the most common chronic diseases and is a risk factor for cardiovascular disease worldwide. Neutrophils and platelets play a crucial role in the progression of inflammation. Studies have reported, that the Neutrophil-to-lymphocyte ratio (NLR) and Platelet-Lymphocyte ratio (PLR) derived from a complete blood count, are elevated in inflammatory condition but the results were debatable.

Objective- Thus, present study was aimed to assess the association between the NLR and PLR with Blood pressure in hypertensive patients.

Methods: Based on specific inclusion and exclusion criteria, 50 diagnosed Hypertensive subjects and 50 healthy controls were enrolled with their written informed consent. Subject's anthropomorphic data, clinical history were noted down. In aseptic collection blood samples were collected, and Complete Blood Count (CBC) was carried out. The statistical analysis of data was done by using SPSS software.

Results- Statistically significant elevated level of SBP, DBP, Neutrophil, Lymphocyte, Platelet, NLR and PLR (140.0±6.60; 92.1±5.49; 86.3±4.86; 71.4±6.3; 423849.1(7419); 2.48±0.51; 5263.7(720.5)) were observed in Hypertensive Cases as compared to Controls. Serum SBP and DBP showed significant positive correlation with NLR and PLR (r- .665, .595; .538, .403).

Conclusion- In our study blood pressure showed positive significant correlation with NLR and PLR. In Hypertensive subjects with increase in blood pressure it leads to increase in NLR and PLR. Thus overall, the outcome of the study provides clear insight into the role of these ratios in inflammatory condition such as Hypertension which may acts as a novel, easily accessible and cost-effective inflammatory marker specially in elderly population and suggest the use of these markers as prognostic tools to reduce the severity of the disease.

Introduction

Hypertension is a common chronic medical condition that affects millions of individuals worldwide. It is a major risk factor for cardiovascular diseases, including heart attacks and strokes. Recent research has shown that inflammation plays a crucial role in the pathogenesis of hypertension and its associated complications. Neutrophils, lymphocytes, platelets, and lipid profile are important components of the immune system and have been implicated in the development and progression of hypertension[1,2].

Hypertension, or high blood pressure, is a prevalent cardiovascular condition affecting millions worldwide and is a significant risk factor for heart disease, stroke, and other cardiovascular complications. The pathophysiology of hypertension is multifactorial, involving genetic, environmental, and lifestyle factors. Recent advances in cardiovascular research have

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highlighted the role of inflammation in the development and progression of hypertension[3].

Among the various biomarkers that reflect inflammatory status, the neutrophil to lymphocyte ratio (NLR) and the platelet to lymphocyte ratio (PLR) have garnered attention due to their simplicity and cost-effectiveness in clinical practice. The NLR serves as an indicator of systemic inflammation, with higher values suggesting an increased inflammatory response, while the PLR reflects the interplay between thrombocyte activity and immune response. Both ratios have been linked to adverse cardiovascular outcomes, making them potentially valuable in predicting the risk of hypertension-related complications[4,5].

The NLR and PLR have been proposed as markers of systemic inflammation and cardiovascular risk. Neutrophils are the most abundant type of white blood cells and play a key role in the innate immune response. Lymphocytes are a type of white blood cell that are involved in adaptive immunity. Platelets are small cell fragments that play a crucial role in blood clotting. Dysregulation of these cell types has been implicated in the pathogenesis of hypertension and cardiovascular disease.

Methodology

A case-control study was conducted with 60 diagnosed Hypertensive Cases and 60 healthy controls within the age group of 30-60 years. The Study was conducted in Department of Medicine and Department of Physiology in Index Medical College and Hospital, Indore, Madhya Pradesh. The enrollment of Hypertensive cases was based on JNC - 8 guidelines for hypertension. For Control group apparently healthy controls were enrolled. Subjects with History of Secondary hypertension causes (e.g., renal artery stenosis), Acute infections, inflammation, or malignancy within 3 months, hematological diseases, Chronic diseases (diabetes, CKD) other inflammatoryr condition and pregnant women were excluded from the study. Written informed consent was obtained from all the participants. A data collection proforma was used to collect subject's demographic, anthropometric and clinical history.

Sample Collection -

Around (2 ml) of blood sample was drawn from the subject in an aseptic condition. Blood sample was transferred into EDTA vials and CBC was carried out using Hematology analyzer.

Statistical analysis

IBM SPSS Statistics software, version 25 was used for statistical analysis. All the data were presented as mean ± Standard deviation. An unpaired t-test was used to compare the study parameters between the Cases and controls. Pearson's correlation coefficient was employed to determine the relationship between the variables in Cases.

Result & observation

The results of the statistical analysis for a total of 120 subjects enrolled in this case-control study are summarized in (Table 1).

Table no- 1 - Comparison of parameters in Hypertensive Cases and Healthy Controls.

Test parameters	Hypertensive Cases	Normotensive Controls	P- value
SBP (mmHg)	148.9±6.6	120.3±4.17	<.0001
DBP	92.24±7.369	92.24±7.369	<.0001
Neutrophil (%)	86.38±4.860	50.66±3.159	<.0001
Lymphocyte (%)	71.94±6.3324	37.58±3.446	<.0001
Platelets (X 10^3 / µl)	4238±7419	176315.8(3917)	<.0001
N/L Ratio	2.4844±0.511	1.343±0.146	<.0001
P/L Ratio	5263±720.5	4283.27(733)	<.0001

Hypertensive subjects demonstrated significantly elevated systolic blood pressure (SBP) levels (148.9 ± 6.6 mmHg) compared to normotensive subjects (120.3 ±

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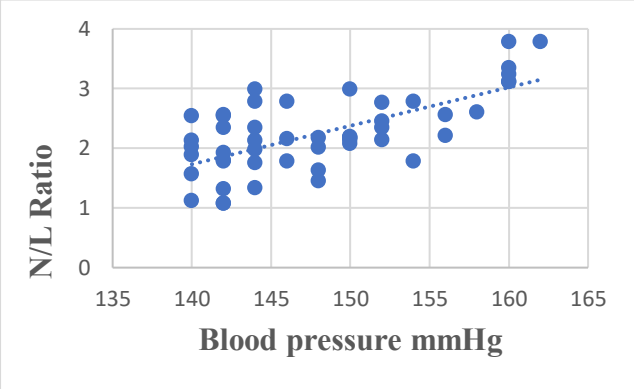
4.17 mmHg), with a highly significant statistical difference ($p < 0.0001$). Although diastolic blood pressure (DBP) values were reported to be identical in both groups (92.24 ± 7.369 mmHg), which may indicate a possible duplication or data recording error, the comparison still yielded a statistically significant difference ($p < 0.0001$). Haematological parameters showed marked alterations in hypertensive individuals, with neutrophil percentage significantly higher (86.38 ± 4.86%) compared to normotensive individuals (50.66 ± 3.16%) ($p < 0.0001$). Similarly, lymphocyte percentage was reported to be elevated in hypertensive subjects (71.94 ± 6.33%) relative to normotensives (37.58 ± 3.45%) ($p < 0.0001$). Platelet count appeared substantially higher in hypertensives (4238 ± 7419 $\times 10^3/\mu L$) compared to normotensives (176,315.8), although unit inconsistency or formatting variation may be present and requires verification. Furthermore, inflammatory indices were significantly elevated in hypertensive individuals, with neutrophil-to-lymphocyte ratio (NLR) recorded at 2.48 ± 0.51 compared to 1.34 ± 0.15 in normotensives ($p < 0.0001$). Likewise, platelet-to-lymphocyte ratio (PLR) was higher among hypertensives (5263 ± 720.5) than normotensives (4283.27), also demonstrating strong statistical significance ($p < 0.0001$). These findings suggest a significant association between hypertension and altered inflammatory as well as haematological parameters.

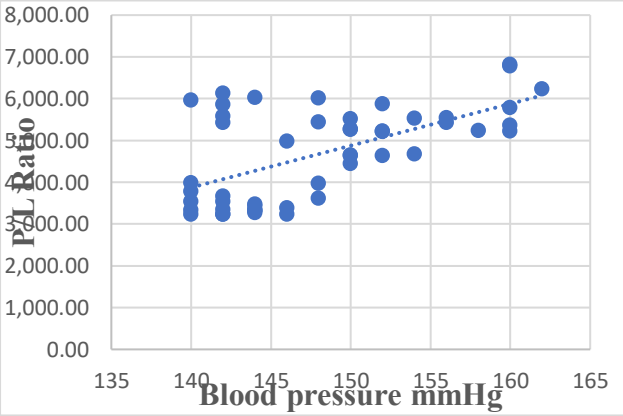
The findings suggest that hematological parameters showed statistically significant differences ($p < 0.0001$) between hypertensive and controls, suggesting strong associations between hypertension and elevated inflammatory markers. Further we also carried out person-correlation to understand the association between Blood pressure and these inflammatory ratio (NLR and PLR)

Table no.1 Pearson correlation of SBP and DBP with N/L and P/L Ratio

	Test parameter	r- value
SBP	N/L ratio	.665**
	P/L ratio	.595**
DBP	N/L ratio	.538**
	P/L ratio	.403*



Graph-1 Correlation between SBP and N/L Ratio



Graph – 2 Correlation between SBP and P/L Ratio

Correlation with SBP (Systolic Blood Pressure):

- **N/L Ratio:** Shows a strong positive correlation (**r = 0.665**, **p < 0.01**), indicating that as SBP increases, the N/L ratio also tends to increase.
- **P/L Ratio:** Also positively correlated (**r = 0.595**, **p < 0.01**), suggesting a moderate to strong association between higher SBP and elevated P/L ratio.

Correlation with DBP (Diastolic Blood Pressure):

- **N/L Ratio:** Displays a moderate positive correlation (**r = 0.538**, **p < 0.01**), indicating DBP is also associated with increased N/L ratio.
- **P/L Ratio:** Shows a weaker but still significant positive correlation (**r = 0.403**, **p < 0.05**),

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suggesting a modest association between DBP and P/L ratio.

Both SBP and DBP are significantly positive correlated with the N/L and P/L ratios, with stronger correlations observed with SBP. This implies a possible link between elevated blood pressure and underlying systemic inflammation.

DISCUSSION

The study demonstrated that hypertensive individuals exhibited significantly elevated systolic blood pressure (SBP) and altered haematological inflammatory markers in comparison to normotensive controls. Moreover, correlation analysis demonstrated a robust positive correlation between systolic blood pressure (SBP) and inflammatory markers, specifically the neutrophil-to-lymphocyte ratio (NLR) ($r = 0.665$, $p < 0.01$) and the platelet-to-lymphocyte ratio (PLR) ($r = 0.595$, $p < 0.01$). Diastolic blood pressure (DBP) exhibited a moderate positive correlation with neutrophil-to-lymphocyte ratio (NLR) ($r = 0.538$, $p < 0.01$) and platelet-to-lymphocyte ratio (PLR) ($r = 0.403$, $p < 0.05$). These findings corroborate the increasing evidence that systemic inflammation is crucial in the pathophysiology and advancement of hypertension.

The markedly elevated SBP noted in hypertensive individuals aligns with existing literature indicating that heightened arterial pressure is closely linked to endothelial dysfunction, vascular inflammation, and oxidative stress (Harrison et al., 2011)[6]. Persistent elevation of systolic blood pressure leads to arterial stiffness and vascular remodelling, mediated by the activation of inflammatory cells and the release of cytokines. The robust positive correlation between SBP and NLR identified in this study corroborates earlier findings indicating that neutrophil-mediated inflammatory responses play a role in vascular damage and heightened peripheral resistance (Imtiaz et al., 2012)[7].

The increased percentage of neutrophils in hypertensive individuals corresponds with prior research indicating that neutrophils play a role in the development of hypertension by generating reactive oxygen species (ROS), proteolytic enzymes, and pro-inflammatory mediators (Schiffrin, 2014)[8]. Elevated neutrophil activity has been linked to endothelial injury and reduced nitric oxide bioavailability, leading to vasoconstriction and heightened blood pressure. Numerous clinical studies have indicated increased NLR values in hypertensive individuals and have suggested NLR as a straightforward, economical indicator of systemic inflammation and cardiovascular risk (Uthamalingam et al., 2011; Tamhane et al., 2008)[9,10].

While the current dataset indicates a higher percentage of lymphocytes in hypertensive individuals, most prior studies document relative lymphocytopenia in chronic inflammatory conditions, which leads to increased NLR values (Azab et al., 2012)[11]. This discrepancy may arise from population variation, acute inflammatory status, laboratory variability, or data recording inconsistencies, necessitating further validation.

In this study, platelet count and PLR were significantly elevated in hypertensive individuals. Platelets are recognized for their significant involvement in vascular inflammation, thrombosis, and atherosclerosis. In hypertensive individuals, increased platelet activation has been thoroughly documented and is associated with endothelial dysfunction and an elevated cardiovascular risk (Vizioli et al., 2009)[12]. This study's robust positive correlation between SBP and PLR aligns with prior research indicating a close association between platelet-mediated inflammatory responses and the severity of hypertension (Yilmaz et al., 2015)[13].

The moderate positive correlation between DBP and inflammatory markers (NLR and PLR) reinforces the inflammatory hypothesis of hypertension. Nonetheless, the correlation strength was inferior relative to SBP. Prior research indicates that systolic blood pressure (SBP) exhibits a stronger correlation with arterial stiffness and vascular inflammation, while diastolic blood pressure (DBP) is more closely linked to peripheral vascular resistance, potentially elucidating the comparatively weaker associations noted (Franklin et al., 2001)[14].

The markedly elevated NLR and PLR values found in hypertensive individuals in this study align with prior clinical research indicating that these ratios serve as dependable indicators of systemic inflammation and cardiovascular risk. These parameters possess clinical utility, as they can be readily acquired from routine complete blood count (CBC) testing without incurring additional costs or necessitating specialized testing (Balta et al., 2016)[15].

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Nonetheless, specific limitations must be acknowledged when analysing these findings. The identical DBP values observed in both groups indicate a potential data duplication or entry error that necessitates verification. The exceedingly elevated platelet and PLR values indicate potential inconsistencies in the units, necessitating standardization prior to conclusive clinical interpretation.

The findings of this study substantiate the hypothesis that inflammatory markers, including NLR and PLR, are significantly correlated with hypertension and may function as valuable adjunct biomarkers for evaluating disease severity and cardiovascular risk. Additional extensive, prospective studies are advised to corroborate these findings and determine standardized cutoff values for clinical use.

CONCLUSION

In conclusion, the neutrophil to lymphocyte ratio and platelet to lymphocyte ratio present promising avenues for understanding the complex interplay between inflammation, Further research is warranted to establish these ratios as standard markers in clinical practice for the management and prediction of cardiovascular events in hypertensive patients.

Limitations -

1. Non-specific markers influenced by infections, autoimmune diseases, malignancies
2. Affected by medications, lifestyle factors (e.g., smoking, diet)
3. Lack of universal cutoff values.
4. Need for standardization in clinical use.

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Conflict of interest

The authors declare that they have no conflicts of interest.

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