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The Correlation of Nlr and Plr with Lipid Profile in Hypertension.

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KEYWORDS

In-situ gel; Gelling system; Approaches, Mechanism; Polymers; Routes of drug delivery

ABSTRACT:

Hypertension, a significant risk factor for cardiovascular disease, is characterized by inflammation in which neutrophils, platelets, and lipid profiles are integral; however, the relationships among neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and lipid profiles remain ambiguous. This study sought to assess NLR and PLR levels in hypertensive patients compared to normotensive controls and their associations with lipid

Fifty diagnosed hypertensive individuals and 50 healthy controls were recruited following informed consent. Blood samples were analysed for complete blood count (CBC) and lipid profiles. Data was statistical evaluated by using SPSS. Hypertensive individuals exhibited significantly elevated systolic blood pressure (148.8 ± 6.60 mmHg), diastolic blood pressure (92.3 ± 7.34 mmHg), neutrophil-to-lymphocyte ratio (NLR) (2.48 ± 0.51), platelet-to-lymphocyte ratio (PLR) (5268 ± 720), cholesterol (262.9 ± 5.18 mg/dL), low-density lipoprotein (LDL) (143.8 ± 2.81 mg/dL), very low-density lipoprotein (VLDL) (45.9 ± 3.03 mg/dL), and triglycerides (208.6 ± 5.9 mg/dL), along with a reduced high-density lipoprotein (HDL) level (33.1 ± 4.7 mg/dL), all with $p < 0.0001$ compared to controls. Pearson correlations indicated positive associations (NLR-VLDL r=0.30; PLR-cholesterol r=0.55) and a negative correlation between NLR and HDL (r=-0.29

Elevated NLR and PLR signify increased inflammation in hypertension, associated with Dyslipidemia and highlighting their potential as economic indicators for risk stratification and early intervention in conjunction with standard lipid profiling.

INTRODUCTION

Hypertension is 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. Lipid profile plays an important role as an indicator for identifying high-risk groups of subjects. Studies have reported that the role of the neutrophil-lymphocyte ratio (NLR) and platelet-lymphocyte ratio (PLR) in inflammatory conditions such as hypertension and its association with lipid profile may play an important role, but the findings are not conclusive. Neutrophils and lymphocytes are essential cellular elements of the immune system, representing the equilibrium between innate and adaptive immune responses. The neutrophil-to-lymphocyte ratio (NLR), obtained from standard complete blood count parameters, has emerged as a new indicator of systemic inflammation. An elevated NLR is linked to endothelial dysfunction, heightened arterial stiffness, and negative cardiovascular outcomes. Platelets are crucial not only for haemostasis but also for inflammation and atherothrombosis. The platelet-to-lymphocyte ratio (PLR) is proposed as an additional inflammatory marker that integrates platelet activation and immune response[1].

Numerous studies have shown that both NLR and PLR are elevated in patients with hypertension and correlate with disease severity, target organ damage, and cardiovascular risk. These ratios are cost-effective, readily available, and replicable, rendering them appealing instruments for regular clinical application. In contrast to traditional inflammatory markers like C-reactive protein, NLR and PLR can be acquired without

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supplementary expenses or specialized assessments. Comprehending the significance of NLR and PLR in hypertension may elucidate the inflammatory underpinnings of the condition and assist in identifying patients at elevated risk for complications[2].

Dyslipidemia is another well-established risk factor for cardiovascular disease and commonly coexists with hypertension. Alterations in lipid profile parameters, including elevated total cholesterol, triglycerides, low-density lipoprotein (LDL), very-low-density lipoprotein (VLDL), and reduced high-density lipoprotein (HDL), contribute to endothelial dysfunction and atherosclerosis[3]. Several studies have explored the relationship between inflammatory markers and lipid parameters in hypertension; however, the findings remain inconsistent and inconclusive.

METHODOLOGY

Based on specific inclusion and exclusion criteria, 50 diagnosed Hypertensive subjects and 50 healthy controls were enrolled with their written informed consent. 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 - 7 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 inflammatory condition and pregnant women were excluded from the study. A data collection proforma was used to collect subject's demographic, anthropometric and clinical history.

Sample Collection -

Around (5 ml) of blood sample was drawn from the subject in an aseptic condition. Blood sample was transferred into EDTA vial for CBC analysis and half of the sample was transferred into plain gel vial for further centrifugation, and thus serum is used for lipid profile. CBC was carried out on Hematology analyzer and Lipid profile was estimated by enzymatic kit method.

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 AND OBSERVATION

The table compares various cardiovascular and biochemical parameters between hypertensive and normotensive subjects, with all parameters showing statistically significant differences ($p < 0.0001$).

Table – 1 Comparison of variables between Hypertensive and Normotensive subjects

Parameter	Hypertensive Subjects (Mean ± SD)	Normotensive Subjects (Mean ± SD)	P-value
DBP	92.34 ± 7.34	75.34 ± 3.15	<0.0001
SBP	148.8 ± 6.60	120.3 ± 4.17	
N/L	2.48 ± 0.51	1.34 ± 0.14	
P/L	5268 (720)	4283 (733)	
Cholesterol	262.9 ± 5.18	139.6 ± 5.31	
LDL	143.8 ± 2.81	104.8 ± 4.5	
HDL	33.1 ± 4.7	54.8 ± 2.9	
VLDL	45.9 ± 3.03	15.2 ± 4.84	
TG	208.6 ± 5.9	119.9 ± 5.47	

Hypertensive subjects have a much higher systolic blood pressure (148.8 ± 6.60 mmHg) and diastolic blood pressure (92.34 ± 7.34 mmHg) than normotensive subjects, whose values are 120.3 ± 4.17 mmHg and 75.34 ± 3.15 mmHg respectively.

The inflammatory markers are also elevated in hypertensive individuals. The neutrophil-to-lymphocyte ratio (2.48 ± 0.51) and platelet-to-lymphocyte ratio (5268 ± 720) are both higher than those in normotensive individuals (1.34 ± 0.14 and 4283 ± 733, respectively), suggesting increased systemic inflammation among

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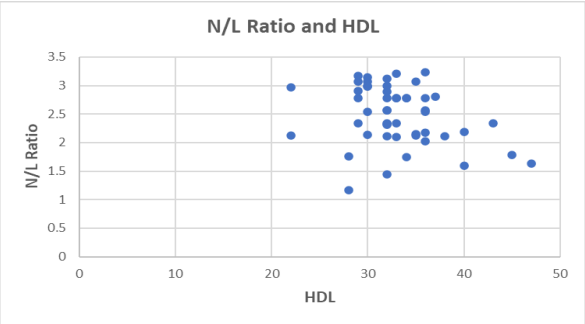
hypertensive subjects. Regarding lipid parameters, hypertensive individuals show a more unfavourable lipid profile. The total cholesterol level is higher (262.9 ± 5.18 mg/dL) compared to normotensive subjects (139.6 ± 5.31 mg/dL). Similarly, LDL (143.8 ± 2.81 mg/dL) and VLDL (45.9 ± 3.03 mg/dL) levels are elevated, while HDL is lower (33.1 ± 4.7 mg/dL) compared to normotensive subjects (54.8 ± 2.9 mg/dL). The triglyceride levels are also significantly higher in hypertensive individuals (208.6 ± 5.9 mg/dL) than in normotensive ones (119.9 ± 5.47 mg/dL).(Table no-1)

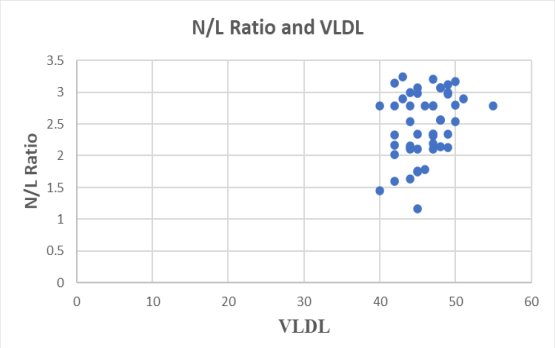
Further to understand the correlation between these inflammatory ratios (NLR and PLR) and lipid profile in Hypertensive cases, we carried out Pearson correlation.

Overall, hypertensive subjects exhibit higher blood pressure, greater systemic inflammation, and a markedly adverse lipid profile, all of which contribute to an increased cardiovascular risk compared to normotensive subjects.

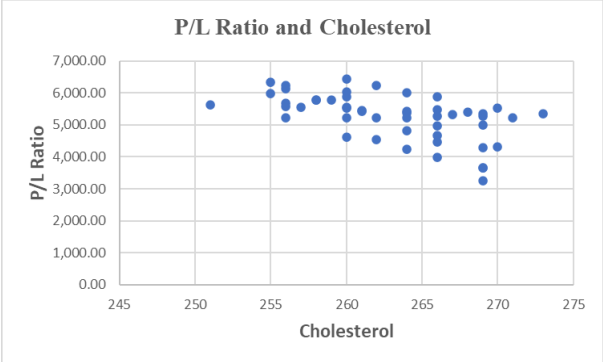
Graph -1 Correlation between N/L Ratio and HDL



Graph-2 Correlation between N/L Ratio and VLDL



Graph -3 Correlation between P/L Ratio and Cholesterol



DISCUSSION

This study evaluated the relationship between Neutrophil–Lymphocyte Ratio (NLR) and Platelet–Lymphocyte Ratio (PLR) with lipid profile parameters in hypertensive patients, comparing the results with previously published research. The findings of this study revealed markedly increased NLR and PLR values in hypertensive individuals relative to normotensive controls, indicating systemic inflammation in hypertension. Moreover, lipid profile parameters were heightened in hypertensive individuals, and significant correlations were noted between inflammatory ratios and lipid parameters. The findings indicated that inflammation and Dyslipidemia coexisted and may have contributed to the advancement of hypertension and cardiovascular complications.

The elevated NLR identified in this study aligns with the findings of Uthamalingam et al. (2011)[4], which indicated that an increased NLR correlates with negative cardiovascular outcomes and systemic inflammation. Imtiaz et al. (2012)[5] similarly reported a significant association between elevated NLR and hypertension as well as metabolic abnormalities. The studies indicated that neutrophilia signified an active inflammatory response, whereas relative lymphocytopenia indicated physiological stress, leading to increased NLR values. The current findings corroborated these observations and reinforced the role of NLR as a biomarker of vascular inflammation and endothelial dysfunction.

Bhat et al. (2013)[6] reported that hypertensive patients exhibited significantly elevated NLR compared to healthy individuals, thereby corroborating the

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inflammatory hypothesis of hypertension. The current study results corroborated these observations, suggesting that chronic low-grade inflammation significantly contributes to the pathogenesis of hypertension. The current study also revealed increased PLR levels in hypertensive individuals. The results were consistent with the research by Azab et al. (2012)[7], which identified PLR as an independent inflammatory marker linked to cardiovascular diseases. Likewise, Gary et al. (2014)[8] established that PLR correlated with vascular inflammation and could forecast unfavourable cardiovascular outcomes. These studies indicated that platelet activation is pivotal in atherosclerosis and vascular injury, frequently observed in hypertensive individuals. The current findings supported these prior observations.

The current study found elevated lipid profile parameters, including total cholesterol, VLDL, and altered HDL levels, in hypertensive individuals. The results aligned with the research by Gupta et al. (2012)[9], which indicated a significant prevalence of dyslipidemia in hypertensive individuals within the Indian population. Likewise, Joshi et al. (2007)[10] indicated that lipid abnormalities markedly elevated cardiovascular risk in hypertensive individuals. The findings indicated that dyslipidemia contributed to endothelial dysfunction, oxidative stress, and vascular stiffness, thereby exacerbating hypertension. The correlation analysis in this study revealed a significant relationship between NLR and HDL, as well as VLDL, and between PLR and total cholesterol. The findings aligned with Horne et al. (2005)[11], who indicated a strong correlation between inflammatory markers, lipid abnormalities, and cardiovascular risk. Previous studies demonstrated that inflammatory cytokines influence lipid metabolism by altering hepatic lipid synthesis and lipoprotein clearance. This study corroborated the pathophysiological connection between inflammation and lipid metabolism.

This study proposed that the simultaneous assessment of inflammatory ratios and lipid profiles could enhance cardiovascular risk stratification in hypertensive individuals. Pearson et al. (2003)[12] underscored the significance of inflammatory markers in forecasting cardiovascular risk. Given that CBC and lipid profiles are standard investigations, NLR and PLR may function as economical and readily available markers in routine clinical practice.

Nonetheless, the current study possessed specific limitations. The sample size was modest, and the study was performed at a single centre. Extensive multicentric studies are necessary to determine the predictive significance of inflammatory ratios in hypertension. Comparable constraints were likewise documented in prior small-scale observational studies.

The findings of this investigation align with existing literature and substantiate the notion that hypertension is linked to chronic low-grade inflammation and Dyslipidemia. The study highlighted that NLR and PLR could be useful, easy, cheap, and accessible markers for spotting high-risk patients with hypertension early on.

CONCLUSION

Our findings indicated an elevated level of NLR and PLR, indicating increased inflammation in hypertension. Simultaneously, we analyzed lipid profiles, which showed an dearranged lipid profile in hypertensive subjects in comparison to normotensive. Further, Pearson correlation was carried out between the variables, which showed the significant association between these ratios with lipid profile in hypertension. Thus overall, the outcome of the study provides a clear insight into the role of these ratios in hypertension, which may act as novel, easily accessible and cost-effective inflammatory markers specially in high-risk population.. It also highlights the importance of routine investigation of lipid profile in hypertension as it elevated level indicates the risk of cardiovascular disease associated with hypertension. The use of these prognostic tools would help to reduce the severity of disease and may help in early and better management of the disease.

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Conflict of interest

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