

## SJMLS - 10(1) - 020

**Haematological and Coagulation Parameters in Newly Diagnosed Systemic Hypertensive Patients: A Comparative Study of elevated vs. normal CRP level**

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<https://dx.doi.org/10.4314/sokjmls.v10i1.20>

**Abstract**

Systemic hypertension is progressively acknowledged as not just a cardiovascular condition but also one associated with systemic inflammation. In recent years, growing evidence has suggested an intricate relationship between inflammation and haematological and coagulatory parameters in systemic hypertension. Among the biomarkers of inflammation, C-reactive protein (CRP) has gathered significant attention due to its predictive value in cardiovascular diseases. However, it remains enigmatic whether elevated CRP is responsible for the alteration in haematological and coagulatory parameters in hypertension or not. Understanding these differences is critical for elucidating the role of inflammation in hypertension-associated haematological and coagulatory abnormalities. A total of 160 NDHTN were serially recruited from the Cardiac clinic of LAUTECH Teaching Hospital, Ogbomosho between May to December 2024. Medical and socio-demographic data of the participants were gathered via questionnaire, interview and retrieval from their folders. About 7 mL of blood was collected for the evaluation of haematology, coagulatory, and inflammatory biomarkers using standard procedure. The participants were categorized into two; elevated CRP; NDHTN+eCRP (n = 148) and normal CRP, NDHTN+nCRP (n = 12). Continuous and categorical data were expressed in mean standard deviation and percent respectively. Data was analyzed using SPSS version 26.1. Significant elevation in lymphocytes and triglyceride and total cholesterol ( $p < 0.005$ ) in NDHTN+eCRP. On the other hand, neutrophil and neutrophil to

lymphocytes ratio was significantly elevated ( $p < 0.05$ ) in NDHTN+nCRP. The findings emphasize the role of inflammation and lipid abnormalities in NDHTN, particularly in individuals with elevated CRP. CRP is not associated with significant haematology and coagulatory changes.

**Keywords:** C-reactive protein, Hypertension, Inflammation

**Introduction**

Hypertension, a primary risk factor for cardiovascular morbidity and mortality, affects a significant portion of the global population, contributing to the growing burden of non-communicable diseases. Systemic hypertension, defined as persistently elevated arterial blood pressure, often remains asymptomatic in its early stages, earning it the moniker "silent killer" (Whelton et al., 2018). According to the World Health Organization (WHO), hypertension affects nearly 1.28 billion adults globally, contributing to the development of cardiovascular diseases, strokes, and renal impairments (WHO, 2021). In recent years, growing evidence has suggested an intricate relationship between inflammation and hematological and coagulatory parameters in systemic hypertension (Saeed *et al.*, 2020). Among the many physiological disturbances associated with hypertension, inflammatory markers such as C-reactive protein (CRP) have garnered increasing attention for their potential role in the pathogenesis and progression of the disease (Boncler *et al.*, 2019).

CRP, an acute-phase reactant synthesized predominantly by the liver in response to systemic inflammation, is a well-established marker of low-grade inflammation (Akgul *et al.*, 2008). It is regulated by cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF- $\alpha$ ), which are themselves associated with cardiovascular disease and hypertension (Ridker *et al.*, 2000). Elevated CRP levels have been linked to endothelial dysfunction, vascular remodeling, and arterial stiffness, all of which are hallmark features of hypertension (Zacho *et al.*, 2008). The link between CRP and hypertension highlights its potential as a biomarker for early detection and risk stratification in newly diagnosed hypertensive (NDHTN) patients. Identifying CRP as an early indicator could provide valuable insights into the inflammatory and coagulative cascades involved in hypertension, enabling more targeted therapeutic interventions.

Hematological and coagulatory parameters also exhibit significant alterations in hypertensive patients. Changes in parameters such as platelet count, platelet distribution width (PDW), and mean platelet volume (MPV) reflect underlying vascular inflammation and endothelial activation. Similarly, coagulation markers such as fibrinogen and D-dimer indicate hypercoagulability, which may predispose patients to thrombotic events (Azab *et al.*, 2011). The interplay between these parameters and systemic inflammation suggests that CRP may have a dual role, contributing to both biochemical derangements and hemostatic imbalances in hypertension (Bautista *et al.*, 2005). However, it remains unclear whether elevated CRP levels are causative in the development of hypertension, or a consequence of the underlying pathophysiological changes associated with the condition. Moreover, whether CRP responsible for the derangement in hematological and coagulatory parameters remains critically evaluated. In this context, the comparative evaluation of hematological and coagulatory parameters in hypertensive patients with elevated versus normal CRP levels becomes critical for understanding its clinical relevance.

Furthermore, despite this growing body of evidence, there is paucity of indigenous

comparative studies on hematological and coagulatory parameters in newly diagnosed hypertensive patients with elevated CRP levels versus those with normal CRP levels. In view of the foregoing, this study aims to bridge this gap by investigating hematological and coagulatory parameters in newly diagnosed systemic hypertensive patients, with a comparative analysis based on their CRP levels. By evaluating and contrasting the profiles of patients with elevated versus normal CRP levels, this research seeks to highlight potential biomarkers and mechanisms underlying the interplay between inflammation, hematology, and coagulation in systemic hypertension. Such insights could pave the way for more targeted therapeutic approaches and improved clinical outcomes.

## **Methodology**

### **Study design**

This was a hospital based cross-sectional study carried out at cardiovascular clinic of LAUTECH Teaching Hospital Ogbomoso, between May and December 2024

### **Inclusion and exclusion criteria**

All patients attending the clinic who were willing to participate and within the age range of 18 to 65 years were enrolled in the study. Those excluded from this study includes pregnant women, those taking anticoagulants and antiplatelet agent, those below 18 or above 65 years of age, those unwilling to provide informed consent for study participation, those with history of bleeding and clotting disorders, those with tuberculosis, cerebrovascular, liver, kidney, diabetes mellitus and autoimmune diseases were also excluded.

### **Samples and data collection**

About 7 mL of whole blood was collected from the eligible participants into plain, citrated and EDTA bottles. The samples in plain bottle were allowed to clot, retracted and centrifuged at 3000 rpm (Uniscope Inc, USA) and serum was used for the assessment of biochemical parameters. The EDTA was make use of for hematological and citrated blood was used for coagulatory assessment. Standard questionnaire was used to gather relevant socio-demography and clinical data. All the participant's medical records were retrospectively reviewed for relevant data.

### Inflammatory biomarkers

CRP, TNF and IL-6 level were analyzed using commercial ELISA kit (Bio-Inteco, UK and Monobond Inc. USA). All the participants were eligible and ultimately undergone inflammatory testing.

### Hematology assessment

The EDTA anticoagulated specimen was used for full blood count. Sysmex hematology auto-analyzer was made use of.

### Lipid profile

Lipid profile parameters were determined using a commercial kit (Agape Diagnostic Switzerland GMBH). All participants were eligible, with 160 ultimately undergoing lipid profile testing.

### Ethical clearance

Ethical approval was obtained from the ethical review committee of Oyo state ministry of health with reference number LTH/OGB/EC/2024/513.

The research was carried out in line with the ethics governing the use of human samples and in accordance with Helsinki declaration. Ethical practices such as participant consent, confidentiality and safety laboratory practice were observed during the study. A questionnaire was used to gather socio-demographic data such as age, gender, marital status, family type, history of blood transfusion, presence of tattoos, number of partners and maternal HBsAg status.

### Statistical analysis

Our data were analyzed using the IBM SPSS version 26 for window software (SPSS Inc. Chicago, IL USA). Quantitative and qualitative variables were presented as mean  $\pm$  SD and as percent respectively. Descriptive analysis and Independent t-test were used for the comparisons of data, p-value  $<$  0.05 were considered significant. Pearson correlation was adopted to evaluate the relationship between different variables.

**Table 1: Demographics and clinical data of the participants**

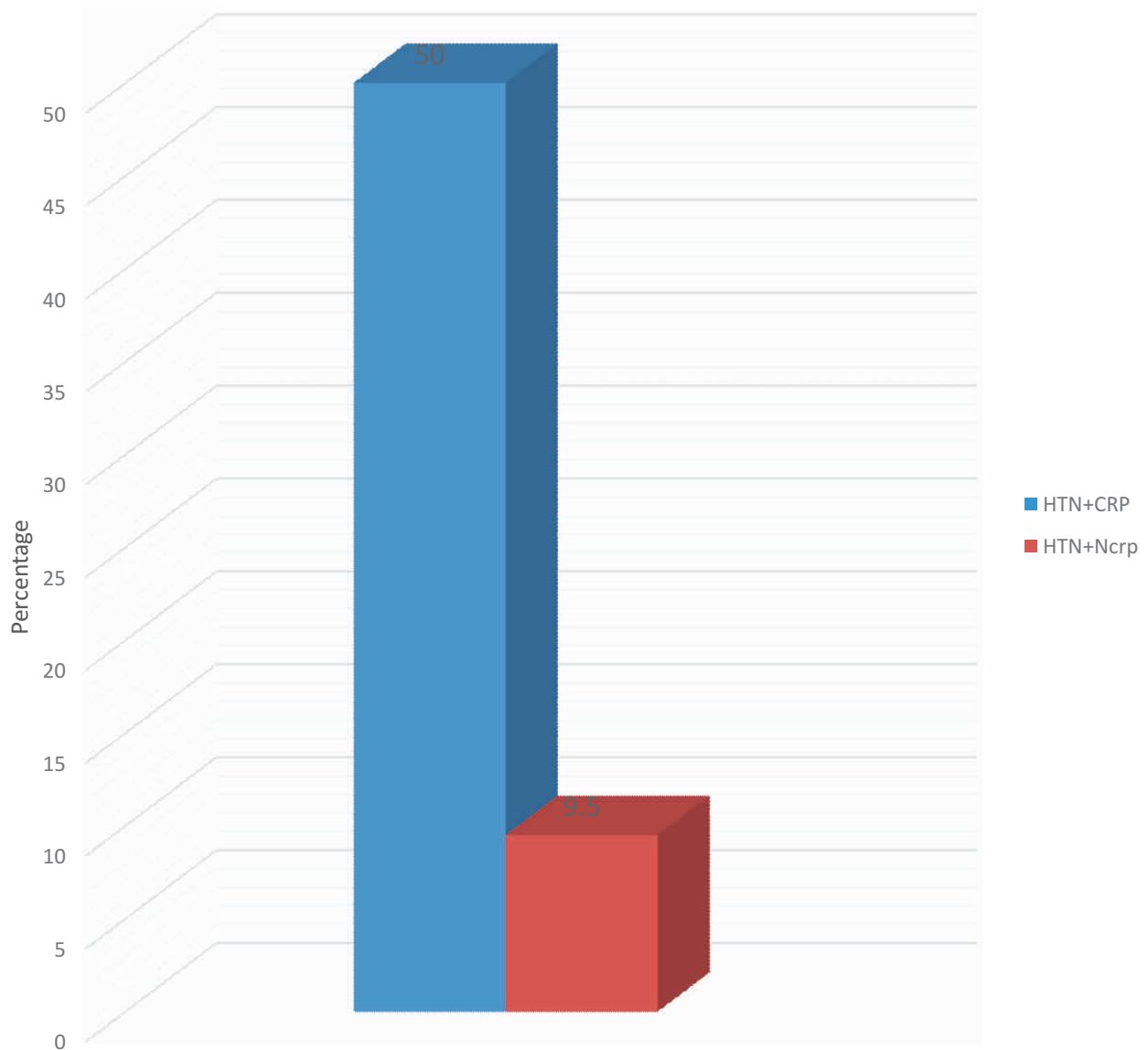
Characteristics		NDHTN+eCRP	NDHTN+ nCRP
Frequency (%)		148 (92.5)	12 (7.5)
Age (years)	18 – 39	59 (39.9)	10 (83.3)
	40 – 64	87 (58.8)	2 (16.7)
	65	2 (1.3)	Nil
Gender	Male	59 (39.9)	2 (16.7)
	Female	89 (60.1)	10 (83.3)
BMI (Kgm <sup>2</sup> )	< 18.5	11 (7.4)	Nil
	18.5 – 24.9	65 (43.9)	8 (66.7)
	25 – 29.9	19 (12.8)	4 (33.3)
Category of obesity		53 (35.8)	Nil
	1 (30 – 34.9)	24 (16.2)	Nil
	2 (35 – 39.9)	27 (18.2)	Nil
Marital status	3 (	2 (1.3)	Nil
	Married	126 (85.1)	12 (100)
	Single	22 (14.8)	0
Stage of HTN	140-159/90-99	133 (89.9)	11 (91.7)
	160-179/100-109	15 (10.1)	1 (8.3)
		Nil	Nil
Symptomatic	Headache	100 (67.6)	7 (58.3)
	Dizziness	32 (21.6)	1 (8.3)
	No sign	16 (10.8)	4 (33.3)

The values are occurrences (%), HTN = hypertension, NDHTN+ CRP = newly diagnosed hypertension with elevated CRP, NDHTN+nCRP = newly diagnosed hypertension with normal CRP,

**Table 2: Comparison of hematological, coagulatory and inflammatory parameters between HTN with elevated and normal CRP**

Parameters	NDHTN+eCRP	NDHTN+Ncrp	P-value
White blood cells count (x 10 <sup>9</sup> /L)	5.69 ± 1.64	4.53 ± 1.71	0.161
Lymphocytes (%)	44.76 ± 8.77	57.15 ± 12.36	0.016*
Neutrophil (%)	48.18 ± 8.29	33.80 ± 11.41	0.007*
Monocytes (%)	7.38 ± 3.59	8.90 ± 4.37	0.358
Red blood cells count (x 10 <sup>12</sup> /L)	4.49 ± 0.62	4.23 ± 0.55	0.310
Hemoglobin (g/dL)	12.31 ± 1.75	12.27 ± 1.36	0.944
Hematocrit (%)	39.15 ± 5.49	36.61 ± 4.31	0.215
MCV (fL)	87.13 ± 8.79	86.87 ± 7.01	0.931
MCH (Pg)	28.85 ± 7.71	29.13 ± 1.67	0.775
MHCH (g/dL)	31.91 ± 3.97	34.44 ± 0.97	0.011*
Platelet count (x 10 <sup>9</sup> /L)	227.32 ± 60.18	246.00 ± 91.75	0.691
aPTT (sec)	26.14 ± 7.96	31.83 ± 9.62	0.210
Prothrombin time	1.31 ± 0.35	1.31 ± 0.21	0.197
Fibrinogen (g/L)	2.93 ± 0.58	2.91 ± 0.55	0.943
C-reactive protein (mg/L)	58.20 ± 11.08	32.26 ± 7.37	0.001*
IL-6 (pg/mL)	0.87 ± 0.84	0.41 ± 0.22	0.007*
Neutrophil to lymphocytes ratio	0.66 ± 0.36	1.14 ± 0.43	0.042*
Platelet to lymphocytes ratio	4.11 ± 2.07	4.75 ± 2.68	0.588
Tissue necrosis factor (pg/mL)	0.77 ± 0.89	0.68 ± 0.69	0.780
Total cholesterol (mmol/L)	6.97 ± 0.91	3.87 ± 1.29	0.034*
HDL-cholesterol (mmol/L)	1.08 ± 0.33	1.19 ± 0.32	0.098
LDL- cholesterol (mmol/L)	3.95 ± 0.18	1.98 ± 0.69	0.036*
Triglyceride (mmol/L)	1.49 ± 0.74	1.28 ± 0.39	0.238
TC/TG	2.89 ± 0.73	1.67 ± 0.59	0.027*
HDL/TG	0.71 ± 0.30	0.97 ± 0.20	0.066

The values are mean ± standard deviation, Student t-test was used to compare the means and p = 0.005, MCH = mean corpuscular hemoglobin, MCHC = mean corpuscular hemoglobin concentration, MCV = mean corpuscular volume, aPTT = activated partial thromboplastin time.



**Figure 1: bar chart showing the percentage of newly diagnosed HTN with elevated NLR between those with elevated and normal CRP.**

**Table 3: Correlation of parameters in NDHTN+eCRP**

		Age	BMI	WBC	LYM	NEU	INR	aPTT	PLT	IL-6	TNR	CRP	TC	TG	
Age	r	1	-.086	-.071	-.180	.119	-.050	.008	.072	.055	.097	.057	.039	.189	
	p		.381	.469	.063	.222	.608	.933	.461	.575	.320	.557	.691	.052	
BMI	r	-.086	1	.133	-.101	.048	.193*	.208*	-.009	.141	.062	-.052	.043	.005	
	p	.381		.171	.301	.620	.046	.032	.925	.146	.527	.598	.659	.956	
WBC	r	-.071	.133	1	-	.418**	.128	-.106	.299**	.025	-.095	.051	.231*	.047	
	p	.469	.171			.427**	.000	.000	.188	.279	.002	.798	.330	.601	.017
LYM	r	-.180	-.101	-	1	-.917**	-.007	.025	-.440**	-.051	-.067	.132	.060	-.095	
	p	.063	.301			.427**	.000	.945	.801	.000	.603	.493	.177	.538	.330
NEU	r	.119	.048	.418**	-	1	.069	.003	.381**	.098	.068	-.171	-.087	.064	
	p	.222	.620	.000		.917**	.483	.979	.000	.313	.486	.078	.373	.512	
PT	r	-.050	.193*	.128	-.007	.069	1	.128	-.171	-.084	-.269**	.022	-.046	.045	
	p	.608	.046	.188	.945	.483		.190	.079	.391	.005	.820	.640	.643	
aPTT	p	.008	.208*	-.106	.025	.003	.128	1	-.057	.131	.040	.067	-.181	.134	
	r	.933	.032	.279	.801	.979	.190		.561	.180	.686	.492	.062	.168	
PLT	p	.072	-.009	.299**	-	.381**	-.171	-.057	1	.080	.051	-.035	-.049	.118	
	r	.461	.925	.002		.000	.000	.079	.561		.413	.600	.720	.616	.228
IL-6	p	.055	.141	.025	-.051	.098	-.084	.131	.080	1	.696**	.094	.029	.058	
	r	.575	.146	.798	.603	.313	.391	.180	.413		.000	.335	.764	.552	
TNR	p	.097	.062	-.095	-.067	.068	-	.040	.051	.696**	1	-.065	.066	.014	
	r	.320	.527	.330	.493	.486	.005	.686	.600	.000		.508	.498	.887	
CRP	p	.057	-.052	.051	.132	-.171	.022	.067	-.035	.094	-.065	1	.116	-.088	
	r	.557	.598	.601	.177	.078	.820	.492	.720	.335	.508		.235	.370	
TC	p	.039	.043	.231*	.060	-.087	-.046	-.181	-.049	.029	.066	.116	1	-.019	
	r	.691	.659	.017	.538	.373	.640	.062	.616	.764	.498	.235		.845	
TG	p	.189	.005	.047	-.095	.064	.045	.134	.118	.058	.014	-.088	-.019	1	
	r	.052	.956	.629	.330	.512	.643	.168	.228	.552	.887	.370	.845		

\* Correlation is significant at the 0.05 level (2-tailed). \*\*. Correlation is significant at the 0.01 level (2-tailed). WBC = white blood cells, LYM = lymphocytes, PLT = platelet, NEU = neutrophil, PT = prothrombin time, TNR = Tissue necrosis factors, TC = total cholesterol, TG = triglyceride.

**Discussion**

**Comparison of clinical data**

A total of one hundred and sixty newly diagnosed systemic hypertensive patients were serially recruited for the study. Table 1 depicted the socio-demographic and clinical data of the participants. The majority of participants in the study (92.5%) had elevated CRP, while only 7.5% had normal CRP. This imbalance in distribution suggests that elevated CRP is more common among those with newly diagnosed

hypertension, potentially implicating CRP in the pathophysiology of hypertension. CRP, as a marker of systemic inflammation, is known to be associated with the development and progression of hypertension (Pepys & Hirschfield, 2003; Boncler *et al.*, 2019). However, it remains enigmatic whether elevated CRP is a cause or consequences of hypertension and other associated biochemical abnormalities. In respect to the age and gender of participants, majority of those with elevated CRP were in age

range of 40–64 years (58.8%), followed by those aged 18–39 years (39.9%), with very few aged 65 years (1.3%). In contrast, those with normal CRP were predominantly younger, with 10 participants (83.3%) aged 18–39 years and only 2 participants (16.7%) aged 40–64 years. No participants aged 65 years were observed in this group. These findings suggest that elevated CRP may be linked to hypertension across a wider age range, particularly in middle-aged individuals, aligning with studies highlighting the interplay between inflammation and cardiovascular risks in middle-aged populations (Ridker *et al.*, 2000). Females predominated in both groups, with 60.1% in the NDHTN+ CRP group and 83.3% in the NDHTN+nCRP group, compared to males at 39.9% and 16.7%, respectively. This sex-based distribution raises the question of whether biological differences, including hormonal influences, play a role in the relationship between CRP levels and hypertension. Research has shown that estrogen may modulate CRP levels and inflammatory responses differently in males and females (Lakoski *et al.*, 2006).

BMI and obesity were notably higher in the NDHTN+ CRP group. While 43.9% of participants in this group had a BMI within the normal range (18.5–24.9 kg/m<sup>2</sup>), a significant proportion were overweight (12.8%) or obese (35.8%). Interestingly, none of the participants in the NDHTN+nCRP group had a BMI  $\geq$  30 kg/m<sup>2</sup>, suggesting a strong association between elevated CRP, obesity, and hypertension. Obesity-induced inflammation is a well-established driver of elevated CRP and hypertension due to the secretion of pro-inflammatory cytokines from adipose tissue (Hotamisligil, 2006).

With respect to the stages of hypertension according AHA classification, majority of the participants in both groups had stage 1 hypertension (140–159/90–99 mmHg), with 89.9% in the NDHTN+ CRP group and 91.7% in the NDHTN+nCRP group. Stage 2 hypertension (160–179/100–109 mmHg) was more common in the NDHTN+ CRP group (10.1%) than in the NDHTN+nCRP group (8.3%), with no participants in either group having severe

hypertension ( $\geq$  180/ 110 mmHg). This finding suggests that CRP elevation is associated with earlier stages of hypertension, potentially serving as an early marker for cardiovascular risk (Bautista *et al.*, 2005).

### Comparison of different parameters between the groups

Table 2 displays the comparison of hematological parameters between the two groups. Notably, variations were observed in lymphocyte and neutrophil percentages. Lymphocyte percentage was significantly higher in the NDHTN+ CRP group compared to the NDHTN+nCRP group ( $p = 0.016$ ), suggesting a potential adaptive immune response linked to elevated CRP levels. Conversely, neutrophil percentage was significantly lower in the NDHTN+ CRP group compared to the NDHTN+nCRP group ( $p = 0.007$ ). This shift in the neutrophil-to-lymphocyte ratio may reflect a chronic inflammatory state in individuals with elevated CRP. MCHC was significantly lower in the NDHTN+ CRP group than in the NDHTN+nCRP group ( $p = 0.011$ ), possibly indicating altered erythropoiesis or inflammation-related changes in red blood cell properties. However, other haematological indices, such as red blood cell count, haemoglobin, and platelet count, were comparable between the groups ( $p > 0.05$ ), suggesting no overt haematological dysfunction.

Furthermore, the data in Table 2 revealed that no significant differences were observed in coagulation parameters, such as aPTT, PT, and fibrinogen levels ( $p > 0.05$ ) between the two groups. Both groups had similar fibrinogen levels ( $p = 0.943$ ), indicating that systemic inflammation in NDHTN+ CRP individuals might not have translated into marked hypercoagulability. However, previous research suggests that prolonged inflammation, often associated with elevated CRP, could increase the risk of thrombotic events in the long term due to endothelial dysfunction and elevated pro-coagulatory factors (Ridker *et al.*, 2004).

Inflammatory biochemical were markedly elevated in the NDHTN+ CRP group, as

evidenced by significantly higher CRP levels ( $p = 0.001$ ) and IL-6 concentrations ( $p = 0.007$ ). Thus, suggesting that IL-6 may have a more pronounced role in CRP-associated hypertension. This underscores the central role of systemic inflammation in individuals with elevated CRP and newly diagnosed hypertension. IL-6 is known to mediate CRP production in the liver and has been implicated in endothelial dysfunction, vascular remodeling, and hypertension development (Brasier *et al.*, 2002). More so, IL-6 is a later responder and typically produced later in the inflammatory response, thus it is more involved in chronic inflammation (Ridker *et al.*, 2018). While TNF- $\alpha$  levels were higher in the NDHTN+ CRP group compared to the NDHTN+nCRP group, this difference was not statistically significant ( $p = 0.780$ ). TNF is typically produced earlier in inflammatory response and a potent inducer of apoptosis (Markandey, 2015). It can thus be inferred that despite the elevation in CRP in some NDHTN, the inflammation was not at its peak, consequentially there may not be clinical consequences.

Dyslipidaemia was prominent in the NDHTN+ CRP group, with significantly higher total cholesterol ( $p = 0.034$ ) and LDL cholesterol levels ( $p = 0.036$ ). Elevated LDL levels, often associated with inflammation, contribute to endothelial dysfunction and atherogenesis, which are key contributors to hypertension (Chrysant and Chrysant, 2003). The triglyceride levels were higher in the NDHTN+ CRP group than in the NDHTN+nCRP group, although this difference was not statistically significant ( $p = 0.312$ ). Additionally, lipid ratios, such as the total cholesterol/triglyceride ratio (TC/TG) and HDL-cholesterol/triglyceride ratio (HDL/TG), were significantly different between the groups ( $p = 0.045$  and  $p = 0.038$ , respectively), reflecting worse lipid metabolism in the NDHTN+ CRP group. Dyslipidemia associated with inflammation and elevated CRP is well-documented and contributes to cardiovascular disease risk (Libby *et al.*, 2002).

#### **Discordance between inflammatory markers**

The major finding of our study is the discordance

between inflammatory biochemical markers, CRP, and haematological inflammatory markers, such as NLR. It was expected that those HTN participants with elevated CRP supposed to have higher NLR, but contrarily, the reverse was our finding (Table 1). Though the observation can be accounted for by several conditions and factors. Biochemical markers and haematological indices respond to inflammation through different mechanisms and dynamics. CRP, a liver-derived acute-phase reactant, rises rapidly during systemic inflammation and reflects the presence of acute or chronic inflammatory states (Rodríguez-Iturbe *et al.*, 2017). In contrast, NLR, which is derived from the relative counts of neutrophils and lymphocytes, reflects the balance between innate immune activation (neutrophilia) and adaptive immune suppression (lymphopenia). The observed discrepancy, where NLR was higher in hypertension patients with normal CRP compared to those with elevated CRP, could suggest that NLR may be more sensitive to subclinical or localized inflammation, such as that driven by vascular or endothelial dysfunction, which is common in HTN (Ma *et al.*, 2020). This phenomenon highlights the role of HTN as a low-grade inflammatory condition, where alterations in immune cell dynamics may occur independently of systemic acute-phase responses (Guzik *et al.*, 2017). Furthermore, the timing of measurement may play a role, as haematological markers may reflect chronic or subacute inflammation, whereas CRP levels tend to respond rapidly to systemic inflammatory triggers (Abu *et al.*, 2017). Also, non-inflammatory influences on haematological markers, such as stress, cortisol levels, or coexisting conditions like metabolic syndrome or obesity, may disproportionately elevate NLR in hypertensive patients (Zahorec, 2001). More so, underlying vascular inflammation in HTN, driven by oxidative stress, endothelial dysfunction, or low-grade immune activation, may preferentially affect immune cell dynamics (e.g., neutrophil recruitment and lymphocyte suppression) rather than triggering a significant CRP response (Rodríguez-Iturbe *et al.*, 2017). Additionally, CRP levels are influenced by genetic factors and liver function, which may vary between individuals and may not fully reflect the inflammatory state in certain contexts. NLR, on the other hand, may provide a broader reflection of immune activation, capturing subtle changes not detected by CRP (Sproston and Ashworth, 2018). Moreover, HTN-associated conditions such as arterial stiffness or microvascular injury could further exacerbate immune cell

imbalances, contributing to higher NLR in these patients (Zhou *et al.*, 2022). It is also possible that methodological differences, including variability in laboratory measurements or sample handling, might influence the interpretation of CRP and NLR levels (Rodríguez-Iturbe and Johnson, 2017). Finally, individual variability, including demographic factors such as age, gender, or genetic predisposition, may differentially affect inflammatory marker expression (Boncler *et al.*, 2019).

### **Lack of significant relationship between CRP and coagulatory parameters**

As evident on Table 3, the outcome of the correlation analysis between different parameters in NDHTN+eCRP revealed that there were no significant association between the three inflammatory markers evaluated and the majority of hematological parameters and the entire coagulatory biomarkers evaluated in this study. These findings further supported the notion that CRP is not likely responsible for the hematological imbalance in early diagnosis of hypertension.

### **Conclusion**

The clinical data and parameters analyzed reveal a significant interplay between systemic inflammation, dyslipidaemia, and newly diagnosed hypertension with elevated CRP levels. Individuals in this group demonstrated notable inflammatory markers, including significantly higher CRP and IL-6 levels, indicative of a heightened inflammatory state that may exacerbate endothelial dysfunction and promote hypertension development. Dyslipidemia further increases the risk of cardiovascular complications.

Although haematological and coagulatory parameters did not show overt dysfunction, shifts in lymphocyte and neutrophil percentages and lower MCHC levels suggest underlying subclinical haematological changes linked to inflammation. The lack of significant differences in coagulation markers such as fibrinogen, PT, and aPTT indicates that hypercoagulability may not be immediately evident but warrants long-term monitoring.

The finding of higher NLR in HTN patients with normal CRP highlights the complex relationship between different components of the immune

system in hypertension. This observation underscores the importance of using complementary markers to assess inflammation comprehensively, as reliance on a single marker like CRP may underestimate subclinical or localized inflammation. Future studies should explore the mechanistic underpinnings of this discordance and its implications for cardiovascular risk assessment and management in HTN patients.

The findings underscore the role of inflammation and lipid abnormalities in newly diagnosed hypertension, particularly in individuals with elevated CRP. These parameters are not only markers of disease progression but also potential therapeutic targets to mitigate cardiovascular risks and improve outcomes. Generally, the inflammatory state, particularly elevated CRP level at early stages of systemic hypertension has little impact on haematology and coagulatory parameters.

### **Recommendations**

Patients with newly diagnosed hypertension, particularly those with elevated CRP levels, should undergo routine evaluation of inflammatory markers (CRP and IL-6) and lipid profiles. This assessment will help identify individuals at higher cardiovascular risk and guide early intervention strategies. Lifestyle changes, including weight reduction, dietary improvements (e.g., DASH or Mediterranean diets), regular physical activity, and smoking cessation,

### **Author's contribution**

Oyeleke, O.K.: Conceptualization, editing and validation

Oni, Janet: Haematological and coagulation analysis

Suleiman, I.E.: Data curation, biochemical and statistical analysis.

Yusuf, O.A.: Patient recruitment, data generation and Provision of reagents

Oladoja, O.O: Patient recruitment, writing of original manuscript

Onadipe, A.O: Provision of reagents, Proofreading, and final editing

Nassar, S: Editing and validation

### **Conflict of interest**

There is no potential conflict of interest relevant to this article.

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**Citation:** Oni Janet Oluwaseun, Oyeleke Kikelomo Olayemi, Suleiman Ibrahim Eleha, Nassar Sulaiman A., Oladoja Olubayo Olumide, Onadipe Abdulateef Oluwasina. Haematological and Coagulation Parameters in Newly Diagnosed Systemic Hypertensive Patients: A Comparative Study of elevated vs. normal CRP level. *Sokoto Journal of Medical Laboratory Science*; **10(1)**: 181 – 191. <https://dx.doi.org/10.4314/sokjmls.v10i1.20>

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