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Neutrophil Lymphocyte Ratio as an Inflammatory Marker in Chronic Kidney Disease: Determinants and Correlates

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Abstract

Introduction: Inflammation has been implicated as a major reason for the higher morbidity and mortality in chronic kidney disease (CKD) compared to the diseases that commonly precedes it. The neutrophil lymphocyte ratio (NLR) has increasingly been reported to be a marker of systemic inflammation. We studied the neutrophil lymphocyte ratio and its relationship with kidney function and other markers of inflammation in health and in CKD. **Methods:** Two hundred and forty four participants in three cohorts: healthy, CKD stage 1 - 2 and, stage 3 - 4, were studied. Data of clinical, NLR, uric acid, urine albumin creatinine ratio (UACR), electrolytes were documented and independent associates of NLR were determined. **Results:** The NLR was higher in the CKD cohorts, $P < 0.001$ and females, $P = 0.01$. The mean NLR of all participants, the healthy and, CKD cohorts were 2.8 ± 0.7, 1.5 ± 0.6 and 3.9 ± 1.4, $P < 0.001$. The mean NLR of all participants ≥ 65 years, all males ≥ 65 years and, all females ≥ 65 years were 4.0 ± 1.6, 3.7 ± 1.0 and 4.2 ± 1.2, $P = 0.01$. The NLR was positively related to the age ($P < 0.001$), systolic blood pressure ($P = 0.012$), uric acid ($P = 0.018$), UACR ($P = 0.006$) and platelet lymphocyte ratio, $P = 0.04$. The NLR was negatively related to the hematocrit ($P < 0.001$), albumin ($P < 0.001$) and glomerular filtration rate ($P < 0.001$). Multivariate analysis after ruling out cofounders, showed age (aOR5.8, CI-4.26 - 10.22), systolic hypertension (aOR1.5, CI-1.21 - 2.07), hyperuricemia (aOR-1.5, CI-0.94 - 2.09), elevated urine ACR (aOR-1.7, CI-1.25 - 2.47) and

CKD (aOR-7.2, CI-1.45 - 8.94) as independent predictors of NLR. **Conclusion:** The NLR as an inflammatory marker is elevated in chronic kidney disease, and increases with disease severity hence it can be a useful tool in determining the presence and severity of inflammation in CKD.

Keywords

Neutrophil Lymphocyte Ratio, Inflammation, Chronic Kidney Disease, Platelet Lymphocyte Ratio, Hyperuricemia, Albuminuria

1. Introduction

Chronic kidney disease (CKD) has continued to pose great health challenges to its victims worldwide despite vast literature on its pathophysiologic mechanisms [1]. Mortality from the disease has persistently gone beyond the sequel of traditional cardiovascular risk factors which could also be classified as pre CKD conditions particularly, diabetes, hypertension, dyslipidemia and obstructive uropathy among others [2]. Though the pre CKD conditions are mostly inflammatory, the increased mortality in CKD compared to any of these conditions has been attributed to more tissue destruction secondary to the persistent inflammatory cascade seen in CKD.

The neutrophil lymphocyte ratio (NLR) has long been identified as an inflammatory marker whose level can predict the level of severity of inflammatory conditions such as CKD [3]. The high prevalence of cardiovascular disease in CKD has been reported to be associated with higher levels of inflammatory makers and this has subsequently been implicated as a contributor to the poor adverse outcome of CKD compared to pre CKD conditions [2]. Markers of inflammation could appear in blood, urine or feces or other body fluids before, during or after the onset of symptoms of disease. While markers of systemic inflammation are mostly elevated in diseases, others could be depressed. Commonly used inflammatory markers in supporting diagnosis, monitoring progression/remission or prognostication include: IL-1, IL-6, fibrinogen, plasma viscosity, ferritin, tissue necrosis factor (TNF), erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) [2] [3].

The commonly seen cardiovascular and central nervous (CNS) disorders in CKD have been attributed to the pan systemic inflammation in CKD [4]. The inflammation-induced tissue remodeling precedes, and continues with CKD and could progress to tissue fibrosis and loss of renal functional capacity leading to proteinuria, atherosclerosis, left ventricular hypertrophy (LVH), cerebral atrophy and textural changes in the brain parenchyma [5] [6]. Proteinuria and LVH have been associated with increased cardiovascular events and mortality in CKD [5]. The hyperuricemia commonly seen in CKD is also reported to augment the inflammatory cascade in CKD due to the endothelial tissue injury, a pivotal oc-

currence in the filtration apparatus of the kidneys [7].

Higher neutrophil counts are commonly seen in inflammatory, infective and stressful states but lower platelets and lymphocytes are often associated with general poor health particularly chronic conditions [8]. Elevated NLR has been reported to predict mortality and heart failure in patients with coronary artery disease, as it predicts CKD occurrence in hypertension and mortality in some cancers [9], in addition to predicting poor prognostic outcome in acute coronary syndromes, particularly, with ST elevation [10].

Despite the volume of literature on NLR as an inflammatory marker, its associations with other known markers of inflammation in CKD such as uric acid and albuminuria have not been completely elucidated coupled with an almost absent literature on NLR and its correlates in sub Sahara Africa. We studied NLR as an inflammatory marker in CKD and determined its correlation with serum uric acid and albuminuria in a low income setting.

2. Materials and Methods

This was a hospital based descriptive study in which patients 16 years and older, with CKD according to the 2012 KDOQI criteria [11] and receiving treatment at the nephrology and hypertension clinic of Babcock University Teaching Hospital, Ilishan-Remo between January and July 2021 were consecutively recruited after giving informed consent. The healthy volunteers were recruited from the staff and patients who had previously been treated for acute disease conditions at the Family Medicine clinics of Federal Medical Centre, Abeokuta. The participants were grouped into three: healthy controls, CKD stages 1 - 2 and, stage 3.4. All patients had renal ultrasound scan (RUS) where obstructive lesions and secondary causes of hypertension and kidney disease were rule out by determining the flow pattern (using Doppler) and the resistivity indexes.

Exclusion criteria included patients with any acute disease, infections, cancers, connective tissue diseases, diabetes, heart failure, chronic liver disease (CLD), blood dyscrasias and other hematologic disorders, frequent users of non-steroidal anti-inflammatory drugs (NSAIDs) and those who were using or who at the time of recruitment, had used within the preceding 6 months, steroids and/or heavy metal containing soaps, creams, ointments or "eye paints".

All participants gave detailed history covering socio-demographics, family and personal illnesses, drug use (prescribed and non-prescribed) and their hospital case files were retrieved to rule out any of the exclusion criteria. The height and weight were measured using standardized protocols and the BMI was calculated. The blood pressure was taken in the sitting position after five minutes rest, with the back and arm resting on a support.

All participants were tested for microalbuminuria using the Micra Albustix Test strip to determine the urine albumin creatinine ratio (UACR). The premenopausal women were asked to abstain from urine collection a day to, and up to a day after, their menstrual flow. All females were asked to clean the vulva and

part the labia to collect urine. The micra albustix bottle was closed immediately after taking a strip out. The end of the strip with pad was completely immersed into the urine to cover the full length of the pads for 50 seconds and then remove by rolling it against the edge of the universal bottle to remove excess urine. The strip pad color was matched with the "strip pad color" inscribed on the strip container and the results were documented.

Venous blood sample was collected from a peripheral vein in the sitting position, in room air for analysis of the full blood count (FBC), erythrocyte sedimentation rate (ESR) and, the serum electrolytes, urea, creatinine and uric acid. The creatinine-based glomerular filtration rate (GFR) was calculated using the CKD epidemiological collaboration (CKD-EPI) formula [12].

3. Definitions

Diabetes: diagnosis or drug treatment of diabetes or a fasting blood glucose (FBG) of ≥7.0 mmol [13].

Hypertension: elevated blood pressure (BP) ≥ 140/90 mmHg [14].

Microalbuminuria: ACR > 3.4 mg/mmol [15].

Hyperuricemia: uric acid (UA) > 0.42 mmol/l (males); 0.36 mmol/l (females) [16].

Anemia: hematocrit < 39% (males) and <36% (females) [17].

Elevated NLR: >3 [18].

Elevated PLR: >160 [18].

Metabolic acid: bicarbonate < 22 mmol/l [19].

CKD: stage 1 and 2 (GFR ≥ 60); stages 3 - 4 (GFR 15.0 - 59.9) [12].

Statistical analysis

Continuous variables were presented as mean with standard deviation and compared using student's t-test while categorical variables were presented as proportions with frequencies and compared using Chi-square. Correlation was done by linear regression analysis using Spearman's correlation. Variables with a p value of <0.25 on univariate analysis were entered into the multivariate model, with backward elimination to adjust for confounders, to determine independent predictors of elevated NLR [20]. The p-value < 0.05 was considered statistically significant.

The research followed the tents of the Declaration of Helsinki. The study was approved by the Babcock University Human Research Ethics Committee (BUHREC) and the Human Ethics Committee of the Federal Medical Centre.

4. Results

A total of two hundred and forty four (127 men and 117 women) participants were studied. The participants were grouped into 3: those in health, CKD stages 1 and 2 and, CKD stages 3 and 4 (**Table 1**). The mean age of all participants, males and females were 49.9 ± 7.3 yrs, 49.2 ± 6.8 yrs and 50.7 ± 8.2 yrs, P = 0.07. A greater proportion of participants 65 yrs and older had CKD stage 3 or 4. The

Table 1. Sociodemographic and clinical characteristics of participants.

Variables	Total	Healthy	CKD 1 - 2	CKD 3 - 4	ANOVA
	N = 244 (%)	N = 100 (%)	N = 76 (%)	N-68 (%)	
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	
Sex,					
Males	127 (52.0)	45 (45.0)	44 (57.9)	38 (55.9)	0.04
Females	117 (48.0)	55 (55.0)	32 (42.1)	30 (44.1)	
Age, (*mean*) *years*	49.9 ± 7.3	46.0 ± 4.2	51.5 ± 8.6	53.9 ± 10.4	0.001
16 - 39	78 (32.0)	37 (37.0)	29 (38.2)	12 (17.7)	0.001
40 - 64	131 (53.7)	49 (49.0)	39 (51.3)	43 (63.2)	
≥65	35 (14.3)	14 (14.0)	8 (10.5)	13 (19.1)	
Smoking					
Yes	13 (5.3)	11 (11.0)	2 (2.6)	0 (0.0)	<0.001*
No	231 (94.7)	89 (89.0)	74 (97.4)	68 (100.0)	
Mean BMI, kg/m^2	26.1 ± 5.9	25.6 ± 6.1	25.3 ± 4.5	27.9 ± 5.7	0.07
Mean SBP, mmHg	125.7 ± 7.0	117.6 ± 6.2	122.5 ± 8.8	141.3 ± 8.9	<0.001
Mean DBP, mmHg	77.3 ± 6.3	72.4 ± 4.7	75.9 ±6.3	86.1 ± 7.7	0.001

CKD-chronic kidney disease, *-Fisher's exact test, BMI-body mass index, SBP-systolic blood pressure, DBP-diastolic blood pressure.

BMI and the blood pressures of the CKD cohorts were higher than the healthy participants.

The serum bicarbonate concentration (SBC), hematocrit, albumin and lymphocytes were lower in the CKD 3 - 4 cohorts compared to the CKD 1 - 2 and, the healthy cohorts, P = 0.03, 0.001, P = 0.002 and P = 0.002 (Table 2). The mean NLR of all participants, the healthy and, CKD cohorts were 2.8 ± 0.7, 1.5 ± 0.6 and 3.9 ± 1.4, P < 0.001. The mean NLR of all participants ≥ 65 years, all males ≥ 65 years and, all females ≥ 65 years were 4.0 ± 1.6, 3.7 ± 1.0 and 4.2 ± 1.2, P = 0.01. The serum creatinine, uric acid, urine ACR, neutrophil, NLR and PLR were higher in the CKD 3-4 cohorts compared to the CKD 1 - 2 and, the healthy cohorts, P < 0.001, P < 0.001, P < 0.001, P = 0.001, P < 0.001 and P = 0.04 respectively.

Elevated NLR was associated with the female gender (P0.04), aging (P < 0.001) and systolic hypertension, P = 0.012 (Table 3), as it was associated with anemia (P < 0.001), hypoalbuminemia (P < 0.001), hyperuricemia (P = 0.018), microalbuminuria (P = 0.006), CKD (P < 0.001) and elevated PLR (P = 0.04).

Pearson correlation coefficient (Table 4) showed an insignificant positive association between NLR and the urine ACR. There was a weak but significant negative correlation between the NLR and GFR (Figure 1) and there was a very weak (insignificant) positive correlation between NLR and the uric acid (Figure 2).

Table 2. Laboratory characteristics of participants.

Variables	Total	Healthy	CKD 1 - 2	CKD 3 - 4	ANOVA
	N = 244 (%)	N = 100 (%)	N = 76 (%)	N-68 (%)	
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD	
	Median (R)	Median (R)	Median (R)	Median (R)	
Bicarbonate, mmol/l	21.7 ± 5.3	23.6 ± 7.6	21.2 ± 4.4	19.5 ± 4.6	0.03
Urea, mmol/l	10.1 ± 3.7	7.1 ± 5.5	9.9 ± 6.3	14.6 ± 6.9	0.001
Creatinine, umol/l	154.5 ± 7.3	83.8 ± 6.6	154.9 ± 8.2	259.5 ± 9.5	<0.001
Uric acid, mmol/l	459.7 ± 12.3	354.2 ± 11.3	497.4 ± 11.8	572.6 ± 14.8	<0.001
ACR, mg/g	29.1 ± 6.1	19.6 ± 3.8	31.2 ± 5.4	40.7 ± 8.8	<0.001
Hematocrit, %	36.5 ± 6.8	41.5 ± 7.3	35.8 ± 5.1	29.9 ± 3.2	0.001
Albumin, mg/dL	38.8 ± 8.2	42.3 ± 8.8	37.8 ± 7.4	34.7 ± 7.1	0.002
Low HDL (n, %)	89 (36.5)	19 (19.0)	29 (38.2)	41 (60.3)	0.002
Elevated LDL, (n, %)	86 (35.2)	21 (21.0)	34 (44.7)	31 (45.6)	0.06
Triglyceride, (n, %)	99 (40.6)	34 (34.0)	30 (39.5)	35 (51.5)	0.05
Leucocytes 10^3//ul					
median (range)	5.3 (3.1 - 7.8)	5.4 (3.8 - 6.2)	5.8 (3.8 - 6.9)	6.1 (4.9 - 7.8)	0.05
Neutrophil 10^3//ul					
median (range)	5.1 (2.9 - 7.7)	3.8 (3.3 - 4.7)	4.8 (4.1 - 5.8)	6.8 (4.1 - 7.7)	0.001
Lymphocytes 10^3//ul					
median (range)	2.1 (1.0 - 4.1)	2.2 (1.9 - 4.1.)	1.8 (1.3 - 2.6)	1.3 (1.0 - 1.9)	0.002
Platelets 10^3//ul	205.3	228.4	195.2	183.2	0.04
(range)	(155 - 289)	(190 - 289)	(168 - 226)	(155 - 223)	
NLR, median (range)	2.8 (0.9 - 7.2)	1.5 (0.9 - 2.1)	2.3 (1.7 - 3.4)	5.3 (2.6 - 7.2)	<0.001
PLR, median	104.1	108.4	110.3	124.4	0.04
(range)	(74.2 - 205.3)	(74.2 - 197.3)	(92.7 - 220.5)	(104.3 - 205.3)	

CKD-chronic kidney disease, R-range, ACR-albumin creatinine ratio, NLR-neutrophil lymphocyte ratio, PLR-platelet lymphocyte ratio.

Table 3. Correlates of neutrophil lymphocyte ratio in participants.

Variables	NLR <3.0	NLR ≥3.0	OR	95% CI	P-value
	N = 196 (%)	N = 48 (%)			
Sex					
Males	105 (82.7)	22 (17.3)	1.44	1.03 - 1.96	0.04
Females	91 (77.8)	26 (22.2)			
Age, years					
<65	163 (85.8)	27 (14.2)	5.1	1.38 - 6.46	<0.001
≥65	33 (61.1)	21 (38.9)			

Continued

BMI, kg/m^2					
<25.0	71 (85.5)	12 (14.5)	1.87	0.93 - 2.02	0.030
≥25.0	125 (77.6)	36 (22.4)			
SBP, mmHg					
<140	148 (85.1)	26 (14.9)	2.77	1.89 - 3.97	0.012
≥140	48 (68.6)	22 (31.4)			
Hematocrit, %					
<39 (M); <36 (F)	44 (52.4)	40 (47.6)	7.43	5.03 - 11.48	<0.001
≥39 (M); ≥36 (F)	152 (95.0)	8 (5.0)			
Albumin, mg/dL					
<35	54 (60.0)	36 (40.0)	5.92	5.24 - 10.16	<0.001
>35	142 (92.2)	12 (7.8)			
Uric acid, mmol/l					
<0.42 (M); <0.36 (F)	92 (91.1)	9 (8.9)	2.83	2.05 - 4.18	0.018
≥0.42 (M); ≥0.36 (F)	106 (73.1)	39 (26.9)			
ACR, mg/mmol					
<3.4	128 (88.9)	16 (11.1)	3.0	1.66 - 4.13	0.006
≥3.4	68 (68.0)	32 (32.0)			
eGFR, ml/min					
<60	30 (44.1)	38 (55.9)	7.9	5.29 - 12.59	<0.001
≥60	166 (94.3)	10 (5.7)			
PLR					
<160	148 (82.2)	32 (17.8)	1.60	0.82 - 1.97	0.04
≥160	48 (75.0)	16 (25.0)			

OR-odds ratio, CI-95% confidence interval, BMI-body mass index, SBP-systolic blood pressure, ACR-albumin creatinine ratio, eGFR-estimated glomerular filtration rate, PLR-platelet lymphocyte ratio.

Table 4. Pearson's linear correlation coefficient between NLR and eGFR, Uric acid and, ACR.

Variables	r	95% CI	P	Correlation
NLR and GFR of all participants	−0.144	(0.082 - 0.173)	0.049	weak negative significance
NLR and UA of all participants	0.022	(0.020 - 0.031)	0.764	very weakly positive
NLR and ACR of all participants	0.095	(0.093 - 0.101)	0.194	weakly positive

NLR-neutrophil lymphocyte ratio, eGFR-estimated glomerular filtration rate, ACR-albumin creatinine ratio, UA-uric acid.

Multivariate regression analysis (Table 5) showed that age (aOR5.8, CI-4.26 - 10.22), systolic hypertension (aOR1.5, CI-1.21 - 2.07), anemia (aOR-5.5, CI-3.59), hypoalbuminemia (aOR-4.9, CI-2.73 - 7.11), hyperuricemia (aOR-1.5, CI-0.94 - 2.09), elevated urine ACR (aOR-1.7, CI-1.25 - 2.47) and CKD (aOR-7.2, CI-1.45 - 8.94) as independent associates of high NLR.

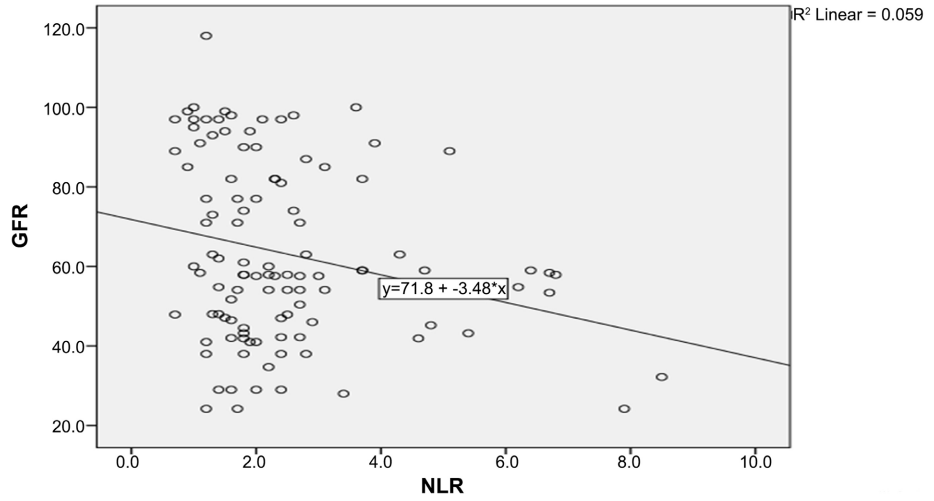

Figure 1. Pearson Correlation between NLR and eGFR. Very weakly significant negative correlation, r = −0.144, P-0.049.

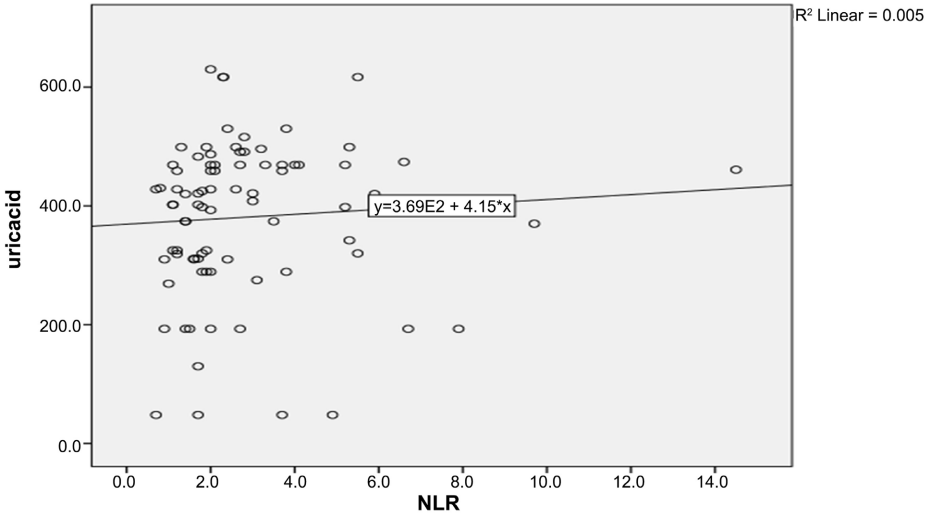

Figure 2. Pearson Correlation between NLR and Uric acid. Insignificant positive correlation: r = −0.022, P-0.764.

Table 5. Multivariate analysis showing independent associates of neutrophil lymphocyte ratio.

Variables	aOR	95% CI	P-value
Age	5.8	4.26 - 10.22	<0.001
Systolic blood pressure	1.5	1.21 - 2.07	0.04
Hematocrit	5.5	3.59 - 9.13	<0.001
Albumin	4.9	2.73 - 7.11	<0.001
Uric acid	1.5	0.94 - 2.09	0.03
Urine albumin creatinine ratio	1.7	1.25 - 2.47	0.03
Glomerular filtration rate	7.2	1.45 - 8.94	<0.001

aOR-adjusted odds ratio, CI-95% confidence interval.

5. Discussion

We assessed in our series the neutrophil lymphocyte ratio (NLR) as an inflammatory marker in CKD and its associations with other known inflammatory markers in CKD. To the best of our knowledge, our study is the first in sub Sahara Africa (SSA) to assess this association. We found a significant increase in NLR from health through CKD stage 1 and 2 to stage 3 and 4. The NLR was positively correlated with markers of inflammation: uric acid, urine albumin creatinine ratio (UACR) and the platelet lymphocyte ratio (PLR), as it was negatively related to the glomerular filtration rate, hematocrit and albumin. Hypertension (systolic) and obesity, both associated with increased inflammatory risk, were positively correlated with the NLR. De Cuiceis *et al.* [21] had reported an association between NLR and the inflammatory changes in the vasculature that could progress to atherosclerosis, hypertension and cardiovascular disease and events. Obesity is a known associate of inflammatory changes associated with lipid peroxidation, our finding of a positive relation between obesity and NLR further strengthen NLR as an inflammatory marker in CKD [22].

In inflammatory states of whatever source including stress, the involvement of the immune system mediates the recruitment of leucocytes, particularly neutrophils, mostly with a concurrent decrease in the lymphocytes during chronic illness associated with poor QOL and increased morbidity [23]. The positive association between NLR and hyperuricemia in this study also strengthen NLR as an inflammatory marker. Hyperuricemia induces an anti-oxidative state in the extracellular space, however, in the intracellular and intravascular compartments, along with other mediators of chronic inflammatory states, it induces renal microvascular/endothelial injury associated with release of vasogenic cytokines, decrease release of endothelium-derived nitric oxide (eNO) and prostaglandins [24]. The chronic vasoconstriction in the vascular bed favors the laying down of fibro-fatty deposits that progresses to atherosclerosis, associated with left ventricular hypertrophy with increased tendency for non-dipping blood pressure, a known risk factor for cardiovascular events and death [25]. The increased activity of sodium transporters in the renal proximal tubules secondary to hyperuricemia only worsens the hypertension and the cardiovascular diseases associated with hyperuricemia [26]. The increased production of the more atherogenic units of the low density lipoproteins (LDL) secondary to excessive renal protein (albumin) loss further aggravate the dyslipidemia that is common in CKD [27]. Though we didn't assess the relationship between NLR and cardiovascular disease and events, its positive association with systolic hypertension in the CKD cohorts, suggest an increased likelihood of cardiovascular disease and events in the future as the inflammatory cascade often runs a chronic course [28].

Chronic immunologic injury involving the renal bed leads to alteration of the lining of protein-like ligands on the endothelial surfaces as they undergo surface-capping thereby shedding immune complexes into the subepithelial spaces [29]. The foot processes of the glomerular visceral membrane are progressively

replaced by continuous cytoplasmic bands along the glomerular basement membrane losing their charge and size selectivity. The resulting podocyte "fusion" or "effacement" causes the loss of albumin and other large and/or negatively charged substances into the urine. The protein load on the renal filtration barrier induces an inflammatory injurious state causing a back flow of glomerular ultrafiltrate worsening the proteinuria. Microalbuminuria is a known inducer and facilitator of progressive kidney damage, and an initiator and facilitator of cardiovascular disease and events [30].

The higher NLR in the elderly in our study mirrors previous findings [21] [22]. The aging process is associated with several comorbidities associated with debilitating diseases, that cause suppressed lymphocyte release and activation. The higher NLR in women in our study is not in agreement with findings by Zang *et al.* [31]. We infer that the higher proportion of females among the elderly coupled with the loss of the hitherto protective estrogen in the post-menopausal year induces the reported higher inflammatory and cardiovascular risk in them compared to men of similar age. We found a declining pattern of platelets and lymphocytes in the CKD cohorts compared to the healthy cohorts. A significantly rising ratio as we found could be attributed to a greater decrease in the lymphocyte count as CKD progresses.

The negative relationship between NLR and the serum bicarbonate, hematocrit and albumin is well reported in previous studies, this also strengthens the NLR as an inflammatory marker. Albumin is known to be inversely related to markers of chronic inflammatory states in patients with CKD and in maintenance hemodialysis (MHD) [32]. The stimulatory role of hypoalbuminemia on antidiuretic hormone release can lead to hyponatremia, which if over or aggressively corrected can lead to a non-inflammatory, osmotic, demyelinating injury in the basal pons of the brain stem [32]. The resulting hemodilution can play an initiating role in the occurrence of anemia-induced dilated cardiomyopathy (DCM) [33].

Limitations encountered included our inability to relate NLR with inflammatory mediators like tissue necrosis (TNF) and 1L-6.

6. Conclusion

The neutrophil lymphocyte ratio as a marker of pan systemic inflammation, is a useful tool for determining the presence and severity of inflammation in CKD. It was positively correlated with other inflammatory markers like uric acid, urine ACR and platelet lymphocyte ratio. The NLR was higher in the CKD cohorts and in females and, was positively related to the age, systolic blood pressure, uric acid, UACR and platelet lymphocyte ratio, as it was negatively related to the hematocrit, albumin and glomerular filtration rate. Aging, systolic hypertension, hyperuricemia, elevated urine ACR and declining kidney function independently predicted the NLR. The NLR could be a cheap, readily available tool for monitoring the progression of CKD in hospitals and community based settings.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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