Al-Azhar International Medical Journal

Volume 4 | Issue 7 Article 13

2023
Section: Industrial medicine

Relationship of Neutrophil to Lymphocyte Ratio (NLR) With Bone Markers and Vascular Changes in Chronic Kidney Disease Patients

Ahmed Alaa Eldin Ahmed M. Saad
Department of Internal Medicine and Nephrology, Faculty of Medicine for boys, Al-Azhar University, Cairo, Egypt

Hani Ismail Hamed
Department of Internal Medicine and Nephrology, Faculty of Medicine for boys, Al-Azhar University, Cairo, Egypt

Mohamed Farok Ibrahim Mosa
Department of Clinical Pathology, Faculty of Medicine for boys, Al-Azhar University, Cairo, Egypt

Ahmed Khaled Mohamed Khaled
M.B.B.CH. – Al-Azhar University, Resident of Internal Medicine, ahmedkhaledmohamed614@gmail.com

How to Cite This Article

Saad, Ahmed Alaa Eldin Ahmed M.; Hamed, Hani Ismail; Mosa, Mohamed Farok Ibrahim; and Khaled, Ahmed Khaled Mohamed (2023) "Relationship of Neutrophil to Lymphocyte Ratio (NLR) With Bone Markers and Vascular Changes in Chronic Kidney Disease Patients," *Al-Azhar International Medical Journal*: Vol. 4: Iss. 7, Article 13.
DOI: https://doi.org/10.58675/2682-339X.1901

ORIGINAL ARTICLE

Relationship of Neutrophil to Lymphocyte Ratio (NLR) with Bone Markers and Vascular Changes in Chronic Kidney Disease Patients

Ahmed Ahmed Elsawy Ahmed Mohamed Saad [a], Hani Ibrahim Hamed [a], Mohamed Fawzy Ibrahim Mosa [b], Ahmed Khalud Mohamed Khaled [a,*]

[a] Department of Internal Medicine and Nephrology, Faculty of Medicine for Boys, Al-Azhar University, Cairo, Egypt
[b] Department of Clinical Pathology, Faculty of Medicine for Boys, Al-Azhar University, Cairo, Egypt

Abstract

Background: **The neutrophil/lymphocyte ratio (NLR), which increases during inflammation and may be linked to a worse prognosis in chronic kidney disease (CKD), has been the subject of several recent research.**

Aim and objectives: **The objective of the current study was to investigate how individuals with chronic renal disease's NLR correlated with bone markers and vascular alterations.**

Patients and methods: **This was a prospective case control study which done at Nephrology Department, Ahmed Maher Teaching Hospital, Cairo, Egypt. The study was conducted over a 1-year duration, from October 2021 to October 2022.**

Results: **Serum vitamin D showed a significant decline in patients with CKD compared with controls, as it had mean values of 30.34, 20.43, and 20.56 ng/ml in the study groups, respectively ($P < 0.001$). On the other hand, serum para thyroid hormone (PTH) had increased levels in CKD groups compared with controls, as it had median values of 40, 101.8, and 391.5 pg/ml in the three study groups, respectively.**

Conclusion: **End stage renal disease (ESRD) is associated with a significant increased carotid internal medical therapy (IMT), reflecting atherosclerotic burden in such patients. CKD is not associated with significant changes in NLR. NLR is not significantly correlated with either bone or vascular changes in CKD patients. Therefore, it should not be used to monitor these changes in the CKD setting.**

Keywords: Bone markers, Chronic kidney disease, Neutrophil to lymphocyte ratio (NLR), Vascular

1. Introduction

According to estimates, more than 9.1% of people worldwide suffer from chronic renal disease (697.5 million in 2017). Chronic kidney disease (CKD).[1]

Only 2 million people globally, or 10% of those with CKD Stage 5 who require it to survive, are believed to get life-saving dialysis.[2] The risk of cardiovascular mortality, undernutrition, and the complete spectrum of morbidities associated with maintenance hemodialysis are all primarily caused by chronic inflammation (MHD).[3]

The neutrophil to lymphocyte ratio determines the proportion of neutrophils to lymphocytes in peripheral blood (NLR). The outcome of a complete blood count led to it (CBC), a straightforward test that is frequently used to assess a variety of diseases, including anemia, inflammation, and infections. NLR is a commonly available and fairly priced indication for inflammation despite not being especially unique.[4] NLR is less expensive than other indicators of inflammation like CRP, IL6, or ferritin since it has been used for prognostication in cancer more than other professions. It has lately gained attention once again as an intriguing marker connected to a variety of outcomes, including

Accepted 9 January 2023.
Available online 30 November 2023

* Corresponding author at: Department of Internal Medicine and Nephrology, Faculty of Medicine for boys, Al-Azhar University, Naser City, Cairo, 111567, Egypt. Fax: 01202328815.
E-mail address: ahmedkhaledmohamed614@gmail.com (A.K.M. Khaled).

https://doi.org/10.58675/2682-339X.1901

cardiovascular mortality and death from all causes. It has been shown that in these people, it predicts all-cause death more precisely than Total Leukocyte WBC Count (TLC).[5]

However, other investigations have shown that NLR is substantially correlated with the development of CKD and is independently (of hsCRP) linked with endothelial dysfunction. In several studies, NLR has been analyzed as a substitute for High Sensitive CRP (hsCRP) in the population with CKD.[6]

One of a number of mineral and bone diseases (CKD-MBD) that develop from CKD, including secondary hyperparathyroidism (SHPT), which has a high bone turnover rate, is renal osteodystrophy.[7]

Given that the majority of hemodialysis patients have high levels of para thyroid hormone (PTH) and a high incidence of inflammation, it has been hypothesized that PTH and inflammation in this population are positively correlated. However, very few studies have examined the connection between PTH and inflammation in the dialysis population, and those that did change how they selected PTH-based subsets because they found a very weak correlation.[8] The objective of the current study was to investigate how individuals with chronic renal disease's NLR correlated with bone markers and vascular alterations.

2. Patients and methods

This was a prospective case control study which done at Nephrology Department, Ahmed Maher Teaching Hospital, Cairo, Egypt. The study was conducted over one-year duration, from October 2021 to October 2022.

The study included 90 participants who were divided into three equal groups; Group 1 included 30 healthy controls. Group 2 included 30 patients with conservative treatment (stage 3 and 4). Group 3 included 30 patients with end stage renal disease (ESRD) and on regular haemodialysis. The stage of CKD was determined based on the KDIGO classification.[9]

Inclusion criteria: Patient's age greater than 18 and less than 65 years old, patients in conservative treatment, patients on regular haemodialysis sessions greater than 6 months duration and healthy controls with serum creatinine less than 1.2 mg/dl.

Exclusion criteria: The exclusion criteria for healthy controls included limited mobility, extensive entrail medical procedure, short gut condition, malabsorption, glucocorticoid treatment, provocative problems (counting dynamic rheumatoid joint pain and fiery gut sickness requiring oral glucocorticoids), constant liver illness, flow therapy with teriparatide or strontium ranelate, and cooperation in a preliminary.

Ethical consideration: The Al-Azhar University faculty of medicine's regional ethical committee gave the study their blessing. All participants—patients and controls—felt free to voluntarily discontinue the research at any time. Personal privacy was maintained, and the data were only utilized for research purposes.

Patient consent: An informed written consent was obtained from all participants after complete explanation of the benefits of the study, and the possible complications of each intervention.

3. Patient evaluation

3.1. History taking

3.1.1. Clinical examination

Laboratory investigations: Complete blood count with its main parameters (hemoglobin, leucocytic count, and platelet). The differential leucocytic count was calculated as well (including neutrophils and lymphocytes). It was performed by Sysmex XS-500i, SYSMEX EUROPE GMBH, Germany.[10] Serum urea and creatinine were assessed via Cobas C 111, Roche Diagnostics, Germany.[11] We used automated methods to measure the levels of serum calcium and phosphorus (Roche Modular P800, Roche Diagnostics, Manheim, Germany) serum vitamin D level of,[12] Blood samples were taken and allowed to clot naturally for 30 min Following a 5 min, 3000 rpm centrifugation, samples were kept at 20 °C. With the help of the Diasorin RIA assay method, 25-OH levels were determined. In order to measure serum parathyroid hormone, an electrochemiluminescence immunoassay was used (Cobas, Roche Diagnostics, Mannheim, Germany).[13]

Measurement of carotid internal medical therapy (IMT): An accomplished radiologist estimated the thickness of the carotid intima-media utilizing the carotid ultrasound contraption (Mindray Constant ultrasound scanner DC-6 with Doppler offices: transducer test recurrence of 7.5 MHz).

And controls, comparing carotid IMT between CKD patients and controls.

3.2. Statistical analysis

The factual program for sociologies (SPSS 22.0, IBM/SPSS Inc., Chicago, IL) and Pearson's relationship coefficient were utilized to survey the outcomes genuinely. χ^2 test (2) Contrasting one subjective variable between at least two groups was applied. At the point when the assumption that somewhere

Table 1. Comparison of the demographic data between the study groups.

Variables	Control group ($n = 30$)	CKD stage 3–4 ($n = 30$)	ESRD ($n = 30$)	
Age (years) [Mean ± SD]	41.30 ± 7.23	54.03 ± 8.01	59.07 ± 9.42	$P_1 < 0.001$* **$P_2 < 0.001$*** $P_3 = 0.053$
Sex [*n* (%)]				
Male	17 (56.7%)	11 (36.7%)	23 (76.7%)	$P_1 = 0.019$* **$P_2 = 0.003$*** $P_3 = 0.186$
Female	13 (43.3%)	19 (63.3%)	7 (23.3%)	

F: ANOVA test - χ^2: Pearson Chi-square test; ESRD, end stage renal disease.
P: General intergroup significance.
P_1: Comparison between control group and CKD 3–4 group.
*P*2: Comparison between control group and ESRD group.
*P*3: Comparison between CKD 3–4 group and ESRD group.
*: Statistically significant ($P \leq 0.05$).

around 80% of the anticipated frequencies are more prominent than five was broken, the Fisher's Careful Test was applied instead of the Chi-Square (2) test. Test Kruskal–Wallis: It is a non-parametric option in contrast to ANOVA and is utilized to look at multiple gatherings of slanted information when ANOVA suppositions have been broken.

4. Results

Table 1.

The mean age of the included cases was 41.3, 54.03, and 59.07 years in Groups 1, 2, and 3, respectively, with a significant increase in the latter group ($P < 0.001$). Regarding gender distribution, men represented 56.7%, 36.7%, and 26.7% of patients in the three groups, respectively, while the remaining cases were women. The prevalence of male gender showed a significant increase in Group 3 ($P = 0.008$) Table 2.

The prevalence of diabetes was statistically comparable between the three study groups ($P = 0.093$), as it was present in 36.7%, 53.3%, 63.3% of cases in the three study groups, respectively. Nonetheless, the duration of hypertension showed a significant decline in Group 1 compared with the other two diseased groups ($P < 0.001$). It had mean values of 4.45, 11.56, and 11.47 years in the three groups, respectively Table 3.

Total leucocytic count had mean values of 6.14, 7.7, and 6.73 × 103/ml in the three study groups, with a significant difference on the statistical analysis ($P = 0.009$). However, both neutrophil and lymphocytic counts showed no significant difference between the three study groups ($P = 0.344$ and 0.829, respectively. The former had median values of 4.5, 4.3, and 4.34 × 103/ml, while the latter had median values of 1.63, 1.57, and 1.55 × 103/ml in the three study groups, respectively. NLR had median values of 2.55, 2.63, and 2.55 in the same three

Table 2. Analysis of chronic diseases and risk factors the cases of the study groups.

Variables	Control group ($n = 30$)	CKD stage 3–4 ($n = 30$)	ESRD ($n = 30$)	Significance test	*s*-value
Hypertension	10 (33.3%)	24 (80%)	18 (60%)	$\chi^2 = 13.482$ **$P = 0.001$***	$P_1 < 0.001$* **$P_2 = 0.010$*** $P_3 = 0.038$*
Duration of HTN (years) [Mean ± SD]	4 ± 1.25	10.13 ± 3	14 ± 3.05	F = 29.187 **$P < 0.001$***	$P_1 < 0.001$* **$P_2 < 0.001$*** $P_3 = 0.001$*
Diabetes mellitus	11 (36.7%)	16 (53.3%)	19 (63.3%)	$\chi^2 = 0.093$	$P_1 = 0.076$ **$P_2 = 0.018$*** $P_3 = 0.264$
Duration of DM (years) [Mean ± SD]	4.45 ± 1.86	11.56 ± 2.16	11.47 ± 5.07	KW = 15.809 **$P < 0.001$***	$P_1 < 0.001$* **$P_2 < 0.001$*** $P_3 = 0.997$
Stage of CKD					
Stage 3 A		2 (6.7%)			
Stage 3 B		12 (40%)			
Stage 4		16 (53.3%)			
Stage 5			30 (100%)		

F: ANOVA test - χ^2: Pearson Chi-square test; ESRD, end stage renal disease.

Table 3. Comparison of the complete blood count (CBC) in the study groups.

Parameter*	Control group ($n = 30$)	CKD stage 3–4 ($n = 30$)	ESRD ($n = 30$)	significance test	Intergroup Significance
Hemoglobin (gm/dl) [Mean ± SD]	12.98 ± 1.21	10.76 ± 1.48	10.06 ± 1.12	F = 42.332 **$P < 0.001$***	**$P_1 < 0.001$*** **$P_2 < 0.001$*** $P_3 = 0.092$
PLTs (10^3/ml) [Mean ± SD]	199.93 ± 52.06	222.43 ± 58.58	203.80 ± 53.32	F = 1.450 $P = 0.240$	**$P_1 = 0.254$** **$P_2 = 0.960$** **$P_3 = 0.389$**
WBCs (10^3/ml) [Mean ± SD]	6.14 ± 1.64	7.70 ± 2.10	6.73 ± 2.02	F = 4.987 **$P = 0.009$***	**$P_1 = 0.007$*** **$P_2 = 0.469$** **$P_3 = 0.132$**
Neutrophil (10^3/ml) [Median (range)]	4.5 (2.33–66)	4.3 (2.03–66)	4.34 (2.43–11.1)	KW = 0.340 **$P = 0.344$**	**$P_1 = 0.998$** **$P_2 = 0.707$** **$P_3 = 0.746$**
Lymphocytes (10^3/ml) [Median (range)]	1.63 (0.33–3.61)	1.57 (0.44–3.61)	1.55 (0.30–3.24)	KW = 0.375 **$P = 0.829$**	**$P_1 = 0.947$** **$P_2 = 0.753$** **$P_3 = 0.914$**
NLR [Median (range)]	2.55 (1.10–40.47)	2.63 (1.10–40.47)	2.55 (1.38–23)	KW = 0.456 **$P = 0.796$**	**$P_1 = 0.909$** **$P_2 = 0.919$** **$P_3 = 0.999$**

F, One-way ANOVA; KW, Kruskal–Wallis test; ESRD, end stage renal disease.

groups, respectively, which was nonsignificant on statistical analysis ($P = 0.796$) Table 4.

Serum vitamin D showed a significant decline in patients with CKD compared with controls, as it had mean values of 30.34, 20.43, and 20.56 ng/ml in the study groups respectively ($P < 0.001$). On the other hand, serum PTH had increased levels in CKD groups compared with controls, as it had median values of 40, 101.8, and 391.5 pg/ml in the three study groups, respectively Table 5.

Carotid IMT had mean values of 0.65, 0.63, and 0.69 mm in the three study groups, respectively, with a significant difference between the three groups ($P = 0.044$). Although group 3 had a comparable IMT with group 1 (controls), it had a significantly higher value compared with group 2 ($P = 0.036$) Fig. 1.

NLR had sensitivity and specificity of 43.3% and 63.3%, respectively, with a diagnostic accuracy of 55.3% to differentiate group 2 from controls, when a cut-off value of 2.92 was used Fig. 2.

Table 4. Comparison of the kidney functions and serum electrolytes in the study groups.

Parameter*	Control group ($n = 30$)	CKD stage 3–4 ($n = 30$)	ESRD ($n = 30$)	significance test	Intergroup Significance
eGFR (ml/min/1.73m^2) [Median (range)]	132 (74–190)	27 (15.5–56.6)	13 (5–15)	KW = 79.204 **$P < 0.001$***	**$P_1 < 0.001$*** **$P_2 < 0.001$*** **$P_3 = 0.003$***
Serum Urea (mg/dl) [Median (range)]	30 (20–50)	70 (39–192)	131 (109–167)	KW = 71.878 **$P < 0.001$***	**$P_1 < 0.001$*** **$P_2 < 0.001$*** **$P_3 < 0.001$***
Serum Creatinine (mg/dl) [Mean ± SD]	0.93 ± 0.16	2.27 ± 0.54	9.33 ± 2.96	F = 201.655 **$P < 0.001$***	**$P_1 = 0.010$*** **$P_2 < 0.001$*** **$P_3 < 0.001$***
Total Serum calcium (mg/dl) [Mean ± SD]	9.54 ± 0.73	8.76 ± 0.97	8.33 ± 0.51	F = 19.729 **$P < 0.001$ ***	**$P_1 < 0.001$*** **$P_2 < 0.001$*** **$P_3 = 0.073$**
Serum phosphorous (mg/dl) [Mean ± SD]	3.92 ± 0.52	4.31 ± 0.71	4.75 ± 0.99	F = 8.808 **$P < 0.001$***	**$P_1 = 0.126$** **$P_2 < 0.001$*** $P_3 = 0.073$
25-(OH-Vit D) (ng/ml) [Mean ± SD]	30.34 ± 5.92	20.43 ± 5.15	20.56 ± 3.42	F = 39.709 **$P < 0.001$***	**$P_1 < 0.001$*** **$P_2 < 0.001$*** $P_3 = 0.994$
PTH (pg/ml) [Median (range)]	40 (23–63)	101.8 (28.7–436.7)	391.5 (5.5–2000)	KW = 46.411 **$P < 0.001$***	**$P_1 = 0.025$*** **$P_2 < 0.001$*** **$P_3 < 0.001$***

KW, Kruskal–Wallis test; *F*, One-way ANOVA; PTH, para thyroid hormone; ESRD, end stage renal disease.

Table 5. Comparison of the intimal thickness in the study groups.

Parameter*	Control group ($n = 30$)	CKD stage 3–4 ($n = 30$)	ESRD ($n = 30$)	significance test	Intergroup Significance
Intimal thickness (mm) [Mean ± SD]	0.65 ± 0.09	0.63 ± 0.10	0.69 ± 0.11	F = 3.235 P= **0.044***	$P_1 = 0.627$ P_2= **0.255** P_3= **0.036***

KW, Kruskal–Wallis test; ESRD, end stage renal disease.

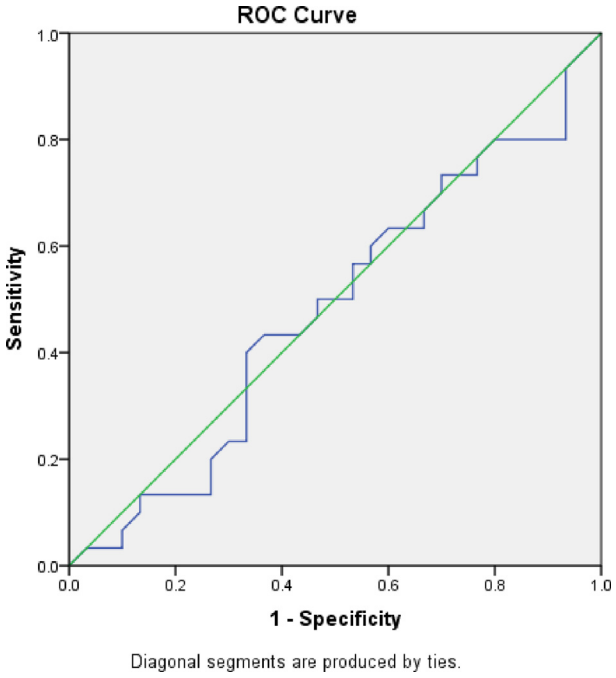

Fig. 1. ROC curve of NLR in differentiating control from CKD 3–4 cases.

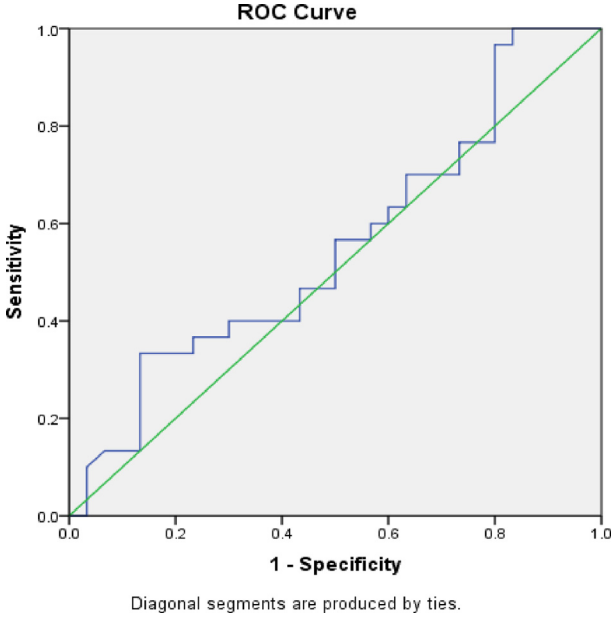

Fig. 2. ROC curve of NLR in differentiating CKD 3–4 cases from ESRD. ESRD, end stage renal disease.

Using a cut-off value of 2.46, NLR has sensitivity and specificity of 56.7% and 47.7%, respectively, with an accuracy of 53.3% to differentiate between Group 2 and 3.

5. Discussion

NLR is elevated during inflammation and may be linked to a worse prognosis in CKD, according to a number of recent studies Kocyigit and colleagues.[14] Recent studies have stressed that NLR could be used as a marker for inflammation because inflammation causes an increase in neutrophil numbers while a decrease in lymphocyte counts Yilmaz and colleagues.[15]

NLR are particularly useful as indicators of inflammation since they can be assessed by a straightforward blood count. Therefore, we conducted the current study at Ahmed Maher Teaching Hospital with the goal of examining the link between bone markers and vascular alterations in CKD patients and NLR. The mean age of the cases included in the current investigation was 41.3 years for Groups 1, 2, and 3, and 59.07 years for group 3, with the latter group showing a significant increase (p 0.001). According to Tonyali and colleagues,[16] CKD patients were significantly older than the control group (64.1 vs. 56.1, respectively—P 0.001), which is consistent with our findings. However, Behairy and colleagues 20 found that the ages of CKD patients and controls were comparable (64.23 and 61.53 years, respectively—$P = 0.123$).

Men made up 56.7, 36.7, and 26.7% of the patients in the current study's three patient groups, respectively, with women accounting for the remaining instances.

In group 3, there was a discernible rise in the proportion of men ($P = 0.008$).

Another recent study reported no significant difference between CKD patients and controls regarding gender ($P = 0.084$). Men represented 71.7 and 53.3% of patients in the CKD and control groups, respectively, while the remaining portion was occupied by women Behairy and colleagues.[17] The previous study agrees with our findings regarding the increased prevalence of male sex in association with CKD.

One more review revealed tantamount orientation dissemination between CKD patients and controls, as men addressed 43.3, 53.3, 70, and 51.4% of patients in charge, predialysis, hemodialysis, and peritoneal dialysis bunches separately ($P = 0.17$) Okyay and colleagues.[18] Our discoveries showed the critical expansion in the commonness of hypertension in Gatherings 2 and 3 contrasted with Gathering 1 ($P = 0.001$). That comorbidity was available in 33.3, 80, and 60% of patients in the three review bunches separately. A past report led by Shakeri and colleagues[19] likewise detailed a critical expansion in the commonness of hypertension in CKD patients contrasted with controls (29.2% vs. 0% separately − $P < 0.001$), which concurs with our discoveries.

Then again, Lahoti and colleagues 23 revealed that the commonness of hypertension was genuinely equivalent between the two gatherings (62% and 55.33% in CKD and control bunches separately—$P = 0.29$).

In the ongoing review, the commonness of diabetes was genuinely equivalent between the three review gatherings ($P = 0.093$), as it was available in 36.7%, 53.3%, 63.3% of cases in the three review bunches separately.

In accordance with our discoveries, another review detailed that the predominance of diabetes didn't essentially vary among CKD and control gatherings (36.67% vs. 33.33% separately—$P = 0.62$) Lahoti and colleagues.[20]

In our review, Hemoglobin showed a critical drop in Gathering 3 contrasted with the other two gatherings ($P < 0.001$). It had mean upsides of 12.98, 10.76, and 10.06 gm/dl in the three review bunches separately.

The diminished erythropoietin creation is the principal factor for hemoglobin drop in such cases Agarwal and colleagues.[21] Different reasons for draining incorporate breaking down platelets, iron deficiency, folate and vitamin B12 lack, uremia (which causes RBC disfigurement that causes hemolysis), and every so often, blood misfortune after hemodialysis Agarwal and colleagues.[21]

In our review, complete leucocytic count had mean upsides of 6.14, 7.7, and 6.73 × 103/ml in the three review gatherings, with a tremendous contrast on the measurable examination ($P = 0.009$). It showed a critical expansion in CKD patients.

In a similar setting, Arai and colleagues[22] detailed that raised WBC count is a notable indicator of CKD progression. They credited that ascent to the constant incendiary state related with CKD.

Oppositely, another new review noticed no huge distinction between CKD patients and controls in regards to WBCs ($P = 0.276$). It had mean upsides of 7.18 and 7.72 × 103/mm in CKD and control bunches, respectively Behairy and colleagues.[17]

Our discoveries showed that neutrophil count had middle upsides of 4.5, 4.3, and 4.34 × 103/ml in the three review bunches separately, with no huge contrast on measurable examination.

Solak and colleagues[23] likewise noticed equivalent neutrophil counts between various CKD stages ($P = 0.84$), which had mean upsides of 4.95, 5.12, and 5 × 103/mm^3 in patients with stages 3, 4, and 5 separately. This concurs with our discoveries.

In the ongoing review, lymphocytic count had middle upsides of 1.63, 1.57, and 1.55 × 103/ml in the three review bunches separately, which was tantamount on factual examination.

Similar discoveries were additionally revealed by Solak and colleagues,[23] who announced that a similar boundary had mean upsides of 1.72, 1.73, and 1.53 × 103/mm^3 in patients with CKD stages 3, 4, and 5 separately with no huge contrast on factual examination ($P = 0.07$).

In the ongoing review, NLR had middle upsides of 2.55, 2.63, and 2.55 in similar three gatherings separately, which was nonsignificant on factual examination ($P = 0.796$).

One more review concurred with our discoveries, as higher NLR was not related with CKD in ordinary weight people in a grown-up wellbeing assessment dataset. In any case, that relationship was available in overweight/stout population Lin and colleagues.[24]

Okyay and colleagues,[18] likewise concurred with the past discoveries, as NLR had middle upsides of 1.75, 2.54, 2.42, and 3.15 in the control, predialysis, hemodialysis, and peritoneal dialysis bunches separately, with a huge contrast between the review gatherings ($P < 0.001$).

In the ongoing review, NLR had responsiveness and explicitness of 43.3% and 63.3% separately, with a demonstrative exactness of 55.3% to separate Gathering 2 from controls, when a cut-off worth of 2.92 was utilized.

In the past review directed by Tonyali and colleagues,[16] the end an incentive for NLR related with CKD (Glomerular Filtrations Rate, GFR<60 ml/min/1.73 m^2) was 3.18, with 39% awareness and 81% explicitness ($P = 0.11$). One could see the low awareness of NLR in the discovery of such cases.

In the ongoing review, eGFR had middle upsides of 132, 27, and 13 ml/min/m^2 in the three review bunches separately, with a critical decrease in Gatherings 2 and 3 contrasted with controls ($P < 0.001$). Likewise, Gathering 3 showed a huge decay of a similar boundary contrasted with Gathering 2.

Another review concurs with our discoveries, as GFR showed a critical decrease in relationship with

CKD (34.7 vs. 108.3 ml/min/1.73 m^2 separately—$P < 0.001$) Yilmaz and colleagues.[15]

Furthermore, Solak and colleagues,[23] noticed an ever-evolving decline of GFR with the expanded CKD stage ($P < 0.001$). eGFR had middle upsides of 44, 28, and 11 ml/min/1.73 m^2 in patients with stage 3, 4, and 5 separately. The past investigations agree with our outcomes.

In our review, serum creatinine had middle upsides of 0.93, 2.27, and 9.33 mg/dl in the three review bunches separately, with a huge expansion in CKD patients. A byproduct of solid movement found in the blood is creatinine. The kidneys usually dispense with it from the blood, yet when kidney capability declines, the creatinine level increases Wong Vega and colleagues.[25] this makes sense of our discoveries.

Our findings showed that serum phosphorus had median values of 3.92, 4.31, and 4.75 mg/dl in the three study groups, respectively, with a significant rise in CKD patients ($P < 0.001$).

Behairy and his colleagues reported that serum phosphorus showed a significant rise in patients with CKD compared with controls (4.44 vs. 3.64 mg/dl, respectively − $P < 0.001$) Behairy and colleagues.[17]

In our study, blood serum PTH had magnified levels in CKD teams compared with controls, because it had median values of 40, 101.8, and 391.5 pg/ml within the 3 study teams severally. A previous Egyptian study conjointly rumored a big increase in blood serum PTH in CKD cases compared with controls (169.5 vs. 27.83 pg/ml severally − $P < 0.001$) 20, that coincides with our findings. Our discoveries showed that blood serum cholecarciferol showed a vital decrease in patients with CKD contrasted with controls, because it had mean upsides of 30.34, 20.43, and 20.56 ng/ml within the review bunches one by one. As indicated by data-based exploration, cholecarciferol inadequacy demolishes over the long haul, advancing from stage three to rearrange five-dimensional. 28 what is more, Guesseous and colleagues uncovered in an exceedingly cross-sectional examination that cholecarciferol inadequacy is seen in each CKD patients and everybody. 29 The previous examinations support our discoveries.

In our review, arteria IMT had mean upsides of 0.65, 0.63, and 0.69 metric linear.

6. Conclusion

An important increase in carotid IMT is linked to end-stage renal failure, reflecting atherosclerotic burden in such patients. CKD is not associated with significant changes in NLR. NLR is not significantly correlated with either bone or vascular changes in CKD patients. Therefore, it should not be used to monitor these changes in the CKD setting.

Disclosure

The authors have no financial interest to declare in relation to the content of this article.

Authorship

All authors have a substantial contribution to the article.

Sources of funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflict of interest

The authors declared that there were no conflicts of Interest.

References

1. Zafirah Y, Lin YK, Andhikaputra G, et al. Mortality and morbidity of chronic kidney disease associated with ambient environment in metropolitans in Taiwan. *Atmos Environ*. 2022; 289:119317.
2. Couser WG, Remuzzi G, Mendis S, Tonelli M. The contribution of chronic kidney disease to the global burden of major noncommunicable diseases. *Kidney Int*. 2011;80:1258–1270.
3. Achinger SG, Ayus JC. Inflammation from dialysis, can it be removed? *Nephrol Dial Transplant*. 2013;28:770–773.
4. Li H, Lu X, Xiong R, Wang S. High neutrophil-to-lymphocyte ratio predicts cardiovascular mortality in chronic hemodialysis patients. *Mediat Inflamm*. 2017;2017:9327136. https://doi.org/10.1155/2017/9327136.
5. Woziwodzka K, Dziewierz A, Pawica M, Panek A, Krzanowski M. Neutrophil-to-lymphocyte ratio predicts long-term all-cause mortality in patients with chronic kidney disease stage 5. *Folia Med Cracov*. 2019;59:4.
6. Chen IC, Yu CC, Wu YH, Chao TH. Elevated neutrophil-to-lymphocyte ratio predicts intermediate-term outcomes in patients who have advanced chronic kidney disease with peripheral artery disease receiving percutaneous transluminal angioplasty. *Acta Cardiol Sin*. 2016;32:532.
7. Cannata-Andía JB, Martín-Carro B, Martín-Vírgala J, et al. Chronic kidney disease—mineral and bone disorders: pathogenesis and management. *Calcif Tissue Int*. 2021;108:410–422.
8. Hou YC, Lu CL, Lu KC. Mineral bone disorders in chronic kidney disease. *Nephrology*. 2018;23:88–94.
9. Wagner M, Wanner C, Schich M, Kotseva K, Wood D. Patient's and physician's awareness of kidney disease in coronary heart disease patients–a cross-sectional analysis of the German subset of the EUROASPIRE IV survey. *BMC Nephrol*. 2017;18:1–12.
10. May JE, Marques MB, Reddy VVB, Gangaraju R. Three neglected numbers in the CBC: the RDW, MPV, and NRBC count. *Cleve Clin J Med*. 2019;86:167–172.
11. Zhou H, Yang M, He X, Xu N. eGFR, cystatin C and creatinine in shrunken pore syndrome. *Clin Chim Acta*. 2019;498:1–5.
12. Suki WN, Moore LW. Phosphorus regulation in chronic kidney disease. *Methodist DeBakey Cardiovasc J*. 2016;12(4 Suppl):6.

13. Garcia-Montemayor V, Sánchez-Agesta M, Agüera ML, et al. *Influence of pre-renal transplant secondary hyperparathyroidism on later evolution after transplantation*. 2019.
14. Kocyigit I, Eroglu E, Unal A, Sipahioglu MH, Tokgoz B. Role of neutrophil/lymphocyte ratio in prediction of disease progression in patients with stage-4 chronic kidney disease. *J Nephrol*. 2012;26:358–365.
15. Yilmaz G, Sevinc C, Ustundag S, Yavuz YC, Hacõbekiroglu T. The relationship between mean platelet volume and neutrophil/lymphocyte ratio with inflammation and proteinuria in chronic kidney disease. *Saudi J Kidney Dis Transpl*. 2017;28:90.
16. Tonyali S, Ceylan C, Yahsi S, Karakan MS. Does neutrophil to lymphocyte ratio demonstrate deterioration in renal function? *Ren Fail*. 2018;40:209–212.
17. Behairy MAEM, Shawky SM, Bawady SAEH, El Kezza GAEH, Ahmed FA. Neutrophil-to-Lymphocyte ratio and platelet-to-lymphocyte ratio in evaluation of inflammation and nutritional status in chronic kidney disease patients. *Egypt J Hosp Med*. 2022;89:5814–5823.
18. Gu Okyay, İnal S, Öneç K, Er RE, Ö Paşaoğlu. Neutrophil to lymphocyte ratio in evaluation of inflammation in patients with chronic kidney disease. *Ren Fail*. 2013;35:29–36.
19. Shakeri A, Abdi M, Khosroshahi HT, Fouladi RF. Common carotid artery intima-media thickness and atherosclerotic plaques in carotid bulb in patients with chronic kidney disease on hemodialysis: a case-control study. *Pakistan J Biol Sci*. 2011;14:844–848.
20. Lahoti S, Kumar S, Agrawal S. Study of carotid intimal medial thickness in chronic kidney disease at rural teaching hospital. *Ann Med Health Sci Res*. 2017;5:12–17.
21. Agarwal AK. Practical approach to the diagnosis and treatment of anemia associated with CKD in elderly. *J Am Med Dir Assoc*. 2006;7. S7–S12.
22. Arai Y, Kanda E, Iimori S, Naito S, Noda Y. Low white blood cell count is independently associated with chronic kidney disease progression in the elderly: the CKD-ROUTE study. *Clin Exp Nephrol*. 2018;22:291–298.
23. Solak Y, Yilmaz MI, Sonmez A, Saglam M, Cakir E. Neutrophil to lymphocyte ratio independently predicts cardiovascular events in patients with chronic kidney disease. *Clin Exp Nephrol*. 2013;17:532–540.
24. Lin CH, Li YH, Wang YY, Chang WD. Higher neutrophil-to-lymphocyte ratio was associated with increased risk of chronic kidney disease in overweight/obese but not normal-weight individuals. *Int J Environ Res Publ Health*. 2022;19:8077.
25. Wong Vega M, Swartz SJ, Devaraj S, Poyyapakkam S. Elevated serum creatinine: but is it renal failure? *Pediatrics*. 2020;146:1.