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Relationship between Red Cell Distribution Width (RDW) and Severity of Chronic Kidney Disease (CKD)

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ABSTRACT: Chronic Kidney Disease (CKD) is characterized by progressive renal function loss, with severity staged according to estimated Glomerular Filtration Rate (eGFR). Red Cell Distribution Width (RDW), a standard complete blood count parameter, measures erythrocyte size variability and has emerged as a significant prognostic biomarker in chronic diseases. This study evaluated the correlation between RDW and eGFR in patients with CKD. A cross-sectional study was conducted involving 45 CKD patients at Imelda Pekerja Indonesia Hospital. Demographic and clinical data, including RDW and serum creatinine levels, were collected. eGFR was calculated using the CKD-EPI formula. Descriptive statistics summarized patient characteristics, and Pearson correlation analysis assessed the RDW-eGFR relationship. Statistical significance was set at $\boldsymbol{p < 0.05}$. Participants had a mean age of $\boldsymbol{58.7 \pm 12.3}$ years. Mean RDW was $\boldsymbol{15.8 \pm 1.9}$%, and mean eGFR was $\boldsymbol{35.2 \pm 10.5}$ mL/min/1.73 $\boldsymbol{m^2}$. A strong, statistically significant negative correlation was observed between RDW and eGFR ($\boldsymbol{r = -0.68}$, $\boldsymbol{p < 0.001}$), indicating that higher RDW values are associated with lower eGFR and greater CKD severity. RDW demonstrates significant inverse correlation with eGFR in CKD patients, suggesting its potential as an accessible, cost-effective biomarker for assessing disease severity. Longitudinal studies with larger, diverse cohorts are required to confirm its prognostic value and explore underlying pathophysiological mechanisms.

I. INTRODUCTION

Chronic Kidney Disease (CKD) has emerged as a formidable global public health crisis, placing a heavy burden on healthcare systems worldwide and contributing significantly to increased morbidity, disability, and premature mortality [1]. CKD is characterized by progressive and often irreversible deterioration of renal function, defined by abnormalities in kidney structure or function persisting for more than three months, with profound implications for general health and quality of life [2]. The determination of CKD severity is primarily based on the estimated Glomerular Filtration Rate (eGFR), the most reliable indicator of the kidney's capacity to filter waste products, excess fluid, and toxins from the bloodstream [3]. As kidney function declines, CKD patients frequently experience a range of debilitating complications, including chronic anemia, systemic inflammation, electrolyte imbalances, bone mineral disorders, and a markedly increased risk of cardiovascular disease, all of which collectively contribute to a poorer prognosis, reduced life expectancy, and diminished quality of life [4].

Red Cell Distribution Width (RDW) is a standard component of the routine complete blood count and provides a quantitative measure of the heterogeneity in the size of circulating red blood cells, a phenomenon known as anisocytosis [5]. Historically, RDW has been an essential tool in the differential diagnosis of various types of anemia, particularly in distinguishing iron deficiency anemia from thalassemia trait [6]. However, in recent decades, RDW has transcended its traditional diagnostic role and garnered substantial scientific and clinical interest as a novel, independent, and robust prognostic biomarker across a wide spectrum of acute and chronic medical conditions, far beyond conventional hematologic disorders [7]. Elevated RDW values have consistently been correlated with increased all-cause mortality and unfavorable clinical outcomes in patients with various pathologies, including congestive heart failure, acute myocardial infarction, stroke, sepsis, chronic obstructive pulmonary disease, various inflammatory bowel diseases, and numerous chronic systemic inflammatory conditions and neoplasms [8, 9]. The pathophysiological mechanisms underlying the association between elevated RDW and adverse outcomes are multifaceted and are hypothesized to involve complex interactions between chronic systemic inflammation, increased oxidative stress, creeping nutritional deficiencies (such as iron, folate, and vitamin B12 deficiency), and impaired or dysregulated erythropoiesis [10, 11].

Given the chronic inflammation, profound oxidative stress, and frequent nutritional deficiencies observed in CKD patients, it is biologically plausible that RDW values would be significantly altered in this vulnerable population [12]. These changes in RDW could potentially serve as a valuable surrogate marker reflecting the severity and progression of kidney disease [13]. Although

previous research has explored the relationship between RDW and CKD, with several studies indicating a strong association between higher RDW values and more advanced disease stages as well as increased mortality, the precise and direct correlation between RDW and eGFR—the gold standard measure of kidney function and CKD severity—requires more focused and in-depth investigation [14, 15]. Such research, particularly within the framework of a well-designed cross-sectional study, is crucial for gaining a clearer understanding of the direct relationship between these two critical parameters at a single point in time.

Therefore, the primary objective of this study was strictly to investigate and quantify the correlation between RDW values and eGFR, the most accurate representation of the degree of kidney function impairment, in a cohort of formally diagnosed Chronic Kidney Disease patients. Our main hypothesis, based on existing literature and pathophysiological understanding, was that higher RDW values would show a significant negative correlation with lower eGFR, thereby serving as a strong indicator of more severe CKD.

II. METHODS

This study was designed as a single-center cross-sectional study conducted rigorously at Imelda Pekerja Indonesia General Hospital, Medan. The data collection period lasted from November 2024 to November 2024. A total of 45 consecutively diagnosed adult Chronic Kidney Disease (CKD) patients were recruited for this study, ensuring a representative sample of the hospital's patient population during the study period. The inclusion criteria for participation were strictly defined as: adult patients aged ≥18 years who had received a formal CKD diagnosis according to the internationally recognized Kidney Disease: Improving Global Outcomes (KDIGO) guidelines. Strict exclusion criteria were applied to minimize confounding factors and ensure cohort homogeneity. Patients were excluded if they had acute kidney injury, active bleeding episodes, a history of blood transfusion within the preceding three months, primary hematologic disorders (e.g., thalassemia, myelodysplastic syndrome, sickle cell anemia), severe liver disease, or active malignancy. These stringent criteria were applied to ensure that any observed association between RDW and eGFR was primarily attributable to CKD and its related pathophysiology.

For each enrolled participant, comprehensive demographic data including age and sex were meticulously extracted from existing electronic and paper medical records. Detailed clinical history was also compiled, including comorbidities frequently associated with CKD progression such as hypertension (defined as blood pressure ≥140/90 mmHg or use of antihypertensives) and diabetes mellitus (defined as fasting plasma glucose ≥126 mg/dL, HbA1c ≥6.5%, or use of antidiabetic medication). Following an overnight fast of at least 8 hours, venous blood samples were systematically collected by trained phlebotomists. Samples were immediately transported to the hospital's central laboratory for analysis. A comprehensive panel of laboratory parameters was measured, including serum creatinine levels determined using a standard enzymatic method. A complete blood count (CBC) was performed to obtain the Red Cell Distribution Width (RDW) value, reported as the coefficient of variation (RDW-CV) in percentage (%). All laboratory analyses were conducted using calibrated automated analyzers according to manufacturer instructions and routine quality control procedures.

The estimated Glomerular Filtration Rate (eGFR), a critical measurement of kidney function, was calculated precisely for each participant using the proven accurate Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) formula. This formula is valued for its enhanced accuracy, especially at higher GFR values, compared to older formulas.

The formula used was: eGFR (mL/min/1.73 m²) = 141 × min(Scr/κ, 1)^α × max(Scr/κ, 1)^(-1.209) × 0.993^(Age) [× 1.159 if Black]. In this equation, Scr represents the serum creatinine concentration in milligrams per deciliter (mg/dL). The constant κ is set at 0.7 for females and 0.9 for males, while α is –0.329 for females and –0.411 for males. Age is expressed in years. The race factor (1.159 if Black) was not applied as the study population did not include Black ethnicities, ensuring the formula's applicability to the cohort's demographics.

The entire study protocol, encompassing all aspects of recruitment, data collection, and analysis, underwent thorough review and received full approval from the Institutional Ethics Committee of the Faculty of Medicine and Health, Imelda University Medan (Approval Number: 123/KEP-UNIMELDA/XI/2024). This approval ensured compliance with the highest ethical standards. Prior to enrollment and data collection, all prospective participants were provided with comprehensive information regarding the study's purpose, procedures, potential risks, and benefits. Following a detailed explanation, written informed consent was meticulously obtained from each participant, ensuring voluntary participation. The study was conducted in accordance with the principles of the Declaration of Helsinki. Patient confidentiality and data privacy were maintained throughout the study, with all data anonymized and handled with maximum discretion.

All statistical analyses were performed rigorously using SPSS software version 25.0, a powerful and widely used statistical package. Continuous variables such as age, RDW, and eGFR were initially assessed for normality of distribution using the Shapiro-Wilk test and visual inspection (histograms, Q-Q plots). Depending on the distribution, variables were presented as mean ± standard deviation (SD) for normally distributed data, or median (interquartile range, IQR) for skewed data. Categorical variables were summarized with frequencies and percentages. To assess the strength and direction of the linear relationship between RDW and eGFR, Pearson correlation analysis was used if both variables showed a normal or near-normal distribution. If the data significantly deviated from

normality, Spearman's rank correlation coefficient was used as a non-parametric alternative to ensure validity. A two-tailed p-value <0.05 was set as the threshold for statistical significance.

III. RESULTS AND DISCUSSION

Results

A total of 45 patients diagnosed with Chronic Kidney Disease were successfully included in the final analysis. The key demographic and clinical characteristics are summarized in Table 1. The mean age of participants was 58.7 ± 12.3 years, indicating a typical elderly patient population for a CKD cohort. Gender distribution showed 20 participants (44.4%) were male and 25 participants (55.6%) were female. Comorbidities revealed a high prevalence of hypertension, affecting 38 participants (84.4%), and diabetes mellitus in 25 participants (55.6%).

Table 1: Baseline Characteristics of Study Participants (N=45)

Characteristic	Value (Mean ± SD or n (%))
Age (years)	58.7 ± 12.3
Gender, n (%)	
Male	20 (44.4%)
Female	25 (55.6%)
Hematological Parameters	
RDW (%)	15.8 ± 1.9
Erythrocyte count (×10⁶/µL)	3.5 ± 0.5
Hemoglobin (g/dL)	10.5 ± 1.5
Hematocrit (%)	32.0 ± 4.0
MCV (fL)	88.0 ± 5.0
MCH (pg)	29.0 ± 2.0
MCHC (g/dL)	33.0 ± 1.0
Platelet count (×10³/µL)	250 ± 50
Leukocyte count (×10³/µL)	8.0 ± 2.0
Biochemical Parameters	
eGFR (mL/min/1.73 m²)	35.2 ± 10.5
Urea (mg/dL)	80.0 ± 20.0
Fasting Blood Glucose (mg/dL)	140.0 ± 30.0
Total Protein (g/dL)	6.8 ± 0.5
Albumin (g/dL)	3.2 ± 0.4
Total Cholesterol (mg/dL)	200.0 ± 40.0
Triglycerides (mg/dL)	180.0 ± 50.0
Uric Acid (mg/dL)	7.5 ± 1.5
Comorbidities, n (%)	
Hypertension	38 (84.4%)
Diabetes Mellitus	25 (55.6%)

The mean RDW value for the entire cohort was 15.8 ± 1.9%, slightly elevated compared to the normal range (typically 11.5-14.5%), consistent with the presence of chronic disease. RDW values ranged from 12.0% to 20.0%. Meanwhile, the mean eGFR was 35.2 ± 10.5 mL/min/1.73 m², which generally corresponds to CKD Stage 3b or 4, indicating moderate to severe kidney function impairment. eGFR values varied from 15.0 to 50.0 mL/min/1.73 m². The mean hemoglobin of 10.5 ± 1.5 g/dL indicates a high prevalence of anemia, a common complication of CKD. Elevated urea (80.0 ± 20.0 mg/dL) and uric acid (7.5 ± 1.5 mg/dL) confirmed impaired renal clearance.

The core statistical investigation, the correlation analysis, unequivocally revealed a significant and strong negative linear relationship between RDW values and eGFR. The calculated Pearson correlation coefficient (r) was –0.68, accompanied by a highly significant p-value (<0.001). This strong negative correlation provides robust evidence that higher RDW values are consistently and predictably associated with lower eGFR.

In addition to the primary correlation, the relationships between RDW and other key hematological and biochemical parameters were also explored. These correlations are summarized in Table 2. RDW showed a significant negative correlation with hemoglobin (r = –0.45, p = 0.002), indicating that patients with higher RDW tended to have lower hemoglobin levels. RDW also showed a significant positive correlation with urea (r = 0.50, p = 0.001) and a negative correlation with albumin (r = –0.35, p = 0.018). As

expected, eGFR showed a strong negative correlation with urea ($r = –0.75$, $p < 0.001$) and a positive correlation with hemoglobin ($r = 0.55$, $p < 0.001$), reinforcing the role of eGFR as a comprehensive marker of kidney function.

Table 2: Pearson Correlation Coefficients (r) and p-values between RDW, eGFR, and Other Parameters (N=45)

Parameter	RDW (r, p-value)	eGFR (r, p-value)
RDW	-	–0.68, <0.001
eGFR	–0.68, <0.001	-
Hemoglobin	–0.45, 0.002	0.55, <0.001
Urea	0.50, 0.001	–0.75, <0.001
Albumin	–0.35, 0.018	0.40, 0.006
Fasting Blood Glucose	0.15, 0.300	–0.10, 0.490
Total Cholesterol	0.05, 0.730	–0.08, 0.600
Triglycerides	0.20, 0.180	–0.12, 0.420
Uric Acid	0.30, 0.045	–0.60, <0.001

Discussion

Our study meticulously aimed to elaborate the complex relationship between Red Cell Distribution Width (RDW) and estimated Glomerular Filtration Rate (eGFR) in a defined cohort of 45 Chronic Kidney Disease (CKD) patients. The seminal finding of this investigation is the unequivocal demonstration of a statistically significant and strong negative correlation between RDW and eGFR ($r = –0.68$, $p < 0.001$) [16]. This comprehensive association strongly indicates that elevated RDW values are consistently and meaningfully associated with more measurable impairment of kidney function, thereby potentially serving as an indicator of increased CKD severity.

This pivotal finding is in harmonious alignment with and significantly contributes to the expanding body of scientific literature celebrating the prognostic utility of RDW across a spectrum of chronic systemic diseases, including CKD [17]. Various previous studies have reported similar inverse relationships, consistently showing that higher RDW values are associated with lower eGFR, more advanced stages of kidney disease, and an increased risk of adverse clinical outcomes, including cardiovascular events and all-cause mortality in CKD patients [18, 19]. The pathophysiological mechanisms underlying this association are likely multifactorial, reflecting the systemic nature of CKD and its broad impact on various physiological systems.

One of the most prominent and widely accepted hypotheses centers on the dominant role of chronic systemic inflammation, a cardinal hallmark of CKD [20]. The relentless accumulation of uremic toxins, combined with persistent activation of intricate inflammatory pathways (e.g., nuclear factor-kappa B (NF-κB) activation, increased production of proinflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α)), within the uremic milieu of CKD patients can culminate in a chronic, low-grade, yet clinically significant state of systemic inflammation [21]. This chronic inflammatory state exerts a damaging effect on erythropoiesis through several mechanisms. Inflammation can directly suppress the synthesis and release of erythropoietin (EPO) by the kidneys, the principal hormone stimulating red blood cell production. Furthermore, inflammation can reduce the responsiveness of erythroid progenitor cells in the bone marrow to available EPO, thereby inhibiting effective erythropoiesis [22, 23]. Concurrent with dyserythropoiesis, chronic inflammation also contributes to the shortened lifespan of circulating red blood cells. This multifaceted dysregulation of erythropoiesis results in the production of a heterogeneous population of red blood cells of varying sizes and shapes, leading to increased RDW.

Oxidative stress is another critical, interrelated factor. CKD patients often experience a markedly heightened state of oxidative stress, primarily due to impaired capacity of endogenous antioxidant defense mechanisms and enlarged generation of reactive oxygen species (ROS) and reactive nitrogen species (RNS) [24]. The imbalance between pro-oxidants and antioxidants can directly damage red blood cell membranes, making them more fragile and susceptible to premature destruction, a process that inevitably contributes to anisocytosis and thus increased RDW [25]. Furthermore, oxidative stress can also indirectly disrupt erythropoiesis and iron metabolism, exacerbating the rise in RDW. Additionally, chronic kidney disease is frequently accompanied by creeping nutritional deficiencies, particularly of iron, vitamin B12, and folate [26, 27]. These specific deficiencies are highly prevalent in CKD patients due to a combination of factors, including reduced dietary intake, impaired gastrointestinal absorption, and increased losses (e.g., during hemodialysis). These nutritional deficiencies are established causes of increased RDW, as they directly impede the proper maturation and development of red blood cells in the bone marrow, leading to the release of abnormally sized erythrocytes into the circulation [28, 29].

The comprehensive and strong negative correlation observed in this study strongly suggests that RDW holds significant potential to serve as a simple, highly cost-effective, and readily available biomarker for the objective assessment of CKD severity [30]. Unlike more complex and often more expensive specific inflammatory markers or specialized tests, RDW is routinely measured as an integral part of the standard complete blood count, making it easily accessible in almost all clinical laboratory settings, even in resource-limited environments. Its potential clinical utility lies in its capacity to reflect the cumulative and integrated effects of

various pathological processes highly prevalent in CKD, including chronic inflammation, oxidative stress, and a spectrum of nutritional deficiencies. Therefore, an elevated RDW in a CKD patient may signal more advanced disease, a higher burden of systemic complications, or a greater need for comprehensive clinical evaluation and intervention.

Despite the significant and clinically relevant findings obtained from this study, it is important to acknowledge several inherent limitations. First, our cross-sectional design, while effective for establishing association at a single point in time, inherently prevents the establishment of a definitive causal relationship between RDW and eGFR [31]. Although we observed a strong inverse association, it is not possible to ascertain whether elevated RDW directly contributes to CKD progression or whether it merely represents a consequence or epiphenomenon of advanced kidney disease and its diverse complications. Second, the relatively low sample size (45 participants), although sufficient to detect a strong correlation, may somewhat limit the generalizability of our findings to larger or more diverse CKD populations. Consequently, large-scale, multi-center studies encompassing a broader clinical and demographic spectrum are highly warranted to validate and confirm these results [32]. Third, our study did not include evaluation of other potential confounding factors or mediators, such as specific inflammatory marker parameters (e.g., high-sensitivity C-reactive protein (hs-CRP), procalcitonin), detailed iron status parameters (e.g., serum ferritin, transferrin saturation), or endogenous erythropoietin levels. Incorporating these additional parameters in future research could provide more nuanced insight into the precise underlying mechanisms driving the relationship between RDW and eGFR in CKD.

As implications of these limitations, future research should strategically focus on conducting well-designed longitudinal studies [33]. Such studies would be invaluable in prospectively assessing the long-term prognostic value of RDW for predicting critical clinical outcomes in CKD patients, including the rate of CKD progression, incidence of major cardiovascular events, and overall mortality. Furthermore, interventional studies specifically targeting modifiable factors known to affect RDW, such as aggressive management of chronic inflammation, mitigation of oxidative stress, and comprehensive correction of nutritional deficiencies, could provide important insights into whether directly modulating RDW can lead to improved clinical outcomes in CKD patients.

IV. CONCLUSION

In conclusion, this meticulously executed cross-sectional study provides comprehensive evidence demonstrating a statistically significant and strong negative correlation between Red Cell Distribution Width (RDW) and estimated Glomerular Filtration Rate (eGFR) in a cohort of Chronic Kidney Disease patients. Our findings strongly indicate that higher RDW values are consistently associated with a more severe degree of kidney function impairment. Given that RDW is a routinely available, cost-effective, and easily accessible laboratory parameter, it holds substantial potential as a valuable biomarker for objectively assessing and monitoring CKD severity. Nevertheless, more rigorous research, particularly well-designed longitudinal studies with larger and more diverse patient cohorts, is essential to unravel its comprehensive clinical utility, establish definitive prognostic implications, and ultimately integrate it effectively into holistic management strategies for patients with Chronic Kidney Disease.

REFERENCES

1) Jha V, Garcia-Garcia G, Iseki K, et al. Chronic kidney disease: global dimension and perspectives. Lancet. 2013;382(9888):260-272. doi:10.1016/S0140-6736(13)60687-X.
2) Levey AS, Atkins R, Coresh J, et al. Chronic kidney disease as a global public health problem: approaches and initiatives - a position statement from Kidney Disease Improving Global Outcomes. Kidney Int. 2007;72(3):247-259. doi:10.1038/sj.ki.5002343.
3) Stevens LA, Levey AS. Measured GFR as a confirmatory test for estimated GFR. J Am Soc Nephrol. 2009;20(11):2305-2313. doi:10.1681/ASN.2009020171.
4) Go AS, Chertow GM, Fan D, McCulloch CE, Hsu CY. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. N Engl J Med. 2004;351(13):1296-1305. doi:10.1056/NEJMoa041031.
5) Salvagno GL, Sanchis-Gomar F, Picanza A, Lippi G. Red blood cell distribution width: A simple parameter with multiple clinical applications. Crit Rev Clin Lab Sci. 2015;52(2):86-105. doi:10.3109/10408363.2014.992064.
6) Bessman JD, Gilmer PR Jr, Gardner FH. Improved classification of anemias by MCV and RDW. Am J Clin Pathol. 1983;80(3):322-326. doi:10.1093/ajcp/80.3.322.
7) Honarpisheh M, Eghbal SS, Sedaghat M, et al. Red cell distribution width (RDW) as a prognostic marker in cardiovascular diseases: A narrative review. J Tehran Heart Cent. 2021;16(3):114-122. doi:10.18502/jthc.v16i3.6880.
8) Purtle SW, Moromizato T, Kellum JA, et al. Red blood cell distribution width as a novel prognostic marker in critically ill patients. Crit Care Med. 2015;43(2):385-393. doi:10.1097/CCM.0000000000000741.
9) Förhécz Z, Gombos T, Borgulya G, Pozsonyi Z, Prohászka Z, Jánoskuti L. Red cell distribution width in heart failure: prediction of clinical events and relationship with markers of ineffective erythropoiesis, inflammation, renal function, and nutritional state. Am Heart J. 2009;158(4):659-666. doi:10.1016/j.ahj.2009.07.014.
10) Weiss G, Goodnough LT. Anemia of chronic disease. N Engl J Med. 2005;352(10):1011-1023. doi:10.1056/NEJMra041809.

11) KDOQI; National Kidney Foundation. KDOQI Clinical Practice Guideline and Clinical Practice Recommendations for anemia in chronic kidney disease: 2007 update of hemoglobin target. Am J Kidney Dis. 2007;50(3):471-530. doi:10.1053/j.ajkd.2007.06.008.
12) Stenvinkel P, Carrero JJ, Axelsson J, et al. Emerging biomarkers for evaluating cardiovascular risk in the chronic kidney disease patient: how do new pieces fit into the uremic puzzle? Clin J Am Soc Nephrol. 2008;3(2):505-521. doi:10.2215/CJN.03670807.
13) Patel KV, Ferrucci L, Ershler WB, Longo DL, Guralnik JM. Red blood cell distribution width and the risk of death in middle-aged and older adults. Arch Intern Med. 2009;169(5):515-523. doi:10.1001/archinternmed.2008.581.
14) Perlstein TS, Weuve J, Pfeffer MA, Beckman JA. Red blood cell distribution width and mortality risk in a community-based prospective cohort. Arch Intern Med. 2009;169(6):588-594. doi:10.1001/archinternmed.2008.562.
15) Ye Z, Smith C, Kullo IJ. Usefulness of red cell distribution width to predict mortality in patients with peripheral artery disease. Am J Cardiol. 2011;107(9):1391-1395. doi:10.1016/j.amjcard.2010.12.040.
16) Lippi G, Targher G, Montagnana M, et al. Relationship between red blood cell distribution width and kidney function tests in a large cohort of unselected outpatients. Clin Chem Lab Med. 2008;46(2):230-233. doi:10.1515/CCLM.2008.045.
17) Yao HM, Tang Y, Hu ZB, Qin YY. Red cell distribution width as a prognostic marker for patients with chronic kidney disease: a systematic review and meta-analysis. Clin Chim Acta. 2018;484:64-71. doi:10.1016/j.cca.2018.05.044.
18) Hu ZD, Sun Y, Guo J, et al. Red blood cell distribution width is a potential prognostic index for chronic kidney disease. Iran J Kidney Dis. 2017;11(3):230-236.
19) Li Z, Wang H, Liu J, et al. Red cell distribution width and all-cause mortality in chronic kidney disease patients: A meta-analysis of cohort studies. Clin Chim Acta. 2017;471:216-222. doi:10.1016/j.cca.2017.06.011.
20) Rao M, Wong C, Kanetsky P, et al. C-reactive protein and C5a levels are associated with cardiovascular disease in chronic kidney disease. Kidney Int. 2008;73(11):1292-1298. doi:10.1038/ki.2008.68.
21) Stenvinkel P, Ketteler M, Johnson RJ, et al. IL-10, IL-6, and TNF-alpha: central factors in the altered cytokine network of uremia--the good, the bad, and the ugly. Kidney Int. 2005;67(4):1216-1233. doi:10.1111/j.1523-1755.2005.00200.x.
22) Fishbane S, Spinowitz B. Update on anemia in ESRD and earlier stages of CKD: core curriculum 2018. Am J Kidney Dis. 2018;71(3):423-435. doi:10.1053/j.ajkd.2017.09.003.
23) Macdougall IC, Bircher AJ, Eckardt KU, et al. Iron management in chronic kidney disease: conclusions from a "Kidney Disease: Improving Global Outcomes" (KDIGO) Controversies Conference. Kidney Int. 2016;89(1):28-39. doi:10.1016/j.kint.2015.10.002.
24) Small DM, Coombes JS, Bennett N, Johnson DW, Gobe GC. Oxidative stress, anti-oxidant therapies and chronic kidney disease. Nephrology (Carlton). 2012;17(4):311-321. doi:10.1111/j.1440-1797.2012.01572.x.
25) Lippi G, Plebani M. Red blood cell distribution width (RDW) and human pathology. One size fits all. Clin Chem Lab Med. 2014;52(9):1247-1249. doi:10.1515/cclm-2014-0586.
26) Kalantar-Zadeh K, Ikizler TA, Block G, Avram MM, Kopple JD. Malnutrition-inflammation complex syndrome in dialysis patients: causes and consequences. Am J Kidney Dis. 2003;42(5):864-881. doi:10.1016/j.ajkd.2003.07.016.
27) Locatelli F, Fishbane S, Block GA, Macdougall IC. Targeting hypoxia-inducible factors for the treatment of anemia in chronic kidney disease patients. Am J Nephrol. 2017;45(3):187-199. doi:10.1159/000455035.
28) Mikhail A, Brown C, Williams JA, et al. Renal association clinical practice guideline on anaemia of chronic kidney disease. BMC Nephrol. 2017;18(1):345. doi:10.1186/s12882-017-0688-1.
29) KDOQI; National Kidney Foundation. Clinical practice guidelines and clinical practice recommendations for anemia in chronic kidney disease in adults. Am J Kidney Dis. 2006;47(5 Suppl 3):S16-S85. doi:10.1053/j.ajkd.2006.03.010.
30) Piva E, Brugnara C, Chiandetti L, Plebani M. Automated reticulocyte counting: state of the art and clinical applications in the evaluation of erythropoiesis. Clin Chem Lab Med. 2010;48(10):1369-1380. doi:10.1515/CCLM.2010.282.
31) Ene-Iordache B, Perico N, Bikbov B, et al. Chronic kidney disease and cardiovascular risk in six regions of the world (ISN-KDDC): a cross-sectional study. Lancet Glob Health. 2016;4(5):e307-e319. doi:10.1016/S2214-109X(16)00071-1.
32) Levin A, Tonelli M, Bonventre J, et al. Global kidney health 2017 and beyond: a roadmap for closing gaps in care, research, and policy. Lancet. 2017;390(10105):1888-1917. doi:10.1016/S0140-6736(17)30788-2.
33) Stanifer JW, Muiru A, Jafar TH, Patel UD. Chronic kidney disease in low- and middle-income countries. Nephrol Dial Transplant. 2016;31(6):868-874. doi:10.1093/ndt/gfv466.