

Impact of Chronic Kidney Disease Stages on Hematological Parameters: Insights from a Tertiary Care Hospital

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Abstract

Background: Chronic Kidney Disease (CKD) is a global health concern characterized by progressive kidney dysfunction, often leading to various hematological abnormalities. This study was designed to assess the impact of CKD stages on hematological parameters in patients attending a tertiary care hospital.

Materials & Methods: This cross-sectional study was conducted at Sylhet MAG Osmani Medical College Hospital from April 2019 to March 2020. A total of 60 predialytic CKD patients (Stages 3 to 5) were included. Patients on hemodialysis, with hematological disorders, recent hemorrhagic events, blood transfusions, or evidence of recent infection were excluded. Sociodemographic data, hematological parameters and clinical information were collected and analyzed. Statistical analyses, including descriptive statistics, Spearman correlation, ANOVA and regression analyses were performed to evaluate the relationships between CKD stages and hematological parameters.

Results: A total of 60 CKD patients were enrolled in the study, with a mean age of 53.7 years. Significant negative correlations were observed between CKD stages and hemoglobin levels ($r = -0.515$, $p < 0.001$), RBC count ($r = -0.481$, $p < 0.001$) and total WBC count ($r = -0.092$, $p = 0.481$). Regression analysis revealed that for each increase in CKD stage, hemoglobin levels decreased by an average of 1.717 g/dL ($p < 0.001$), while RBC counts decreased by 0.577 million cells/iL ($p = 0.006$) and WBC counts decreased by 1.702 thousand cells/iL ($p = 0.011$). Platelet count showed no significant changes ($p = 0.834$).

Conclusion: The findings of this study highlight the significant impact of CKD stages on hematological parameters, particularly hemoglobin and RBC counts.

keywords: Chronic Kidney Disease (CKD), Hematological Parameters, Hemoglobin, RBC Count, WBC Count, Platelet Count.

(J Bangladesh Coll Phys Surg 2025; 43: 145-153)

DOI: <https://doi.org/10.3329/jbcps.v43i2.80748>

Introduction:

Chronic kidney disease (CKD) is a progressive and irreversible deterioration of renal function, usually

occurring over months or years. It manifests as a gradual loss of kidney functions. It is considered a worldwide public health problem, since millions of people are

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Received: 01 November, 2024

Accepted: 11 January, 2025

affected globally, accompanied by very high morbidity and mortality rates due to its complications. According to a recent systematic analysis from the Global Burden of Disease Study 2017 [1], 9.1% of people worldwide are expected to have CKD, indicating a significant prevalence of the disease worldwide. CKD and its cardiovascular complications caused 2.6 million deaths and 35.8 million DALYs, two-thirds of which were in countries with large populations and middle and low-middle socio-demographic index (SDI). The burden of CKD has increased over the past 27 years due to aging populations, increased prevalence of risk factors such as diabetes and hypertension—which accounted for more than half of CKD-related deaths in 2017^{1,2}.

CKD is classified into five stages based on the estimated glomerular filtration rate (eGFR), with each stage reflecting the degree of renal impairment and correlating with distinct clinical manifestations. CKD frequently affects the hematological system due to several key factors related to the kidney's role in maintaining blood and bone marrow function. As the disease progresses, patients often experience various hematological abnormalities, including anemia, alterations in white blood cell (WBC) counts and disturbances in platelet function.

Anaemia is the most common, consistent and severe of the various haematological abnormalities. Anemia in CKD is primarily due to erythropoietin deficiency, although shortened red blood cell half-life and functional iron deficiency, increased hemolysis, suppression of bone marrow erythropoiesis, hematuria and gastro intestinal blood loss also contribute to the anemia of CKD [3]. Anemia becomes more severe with the progression of CKD, especially in patients with end-stage renal disease. If left untreated, it significantly increases the risk of all-cause and cardiovascular mortality, reduces quality of life and raises morbidity in CKD patients^{4,5}. Additionally, untreated anemia can hasten the decline of kidney function by causing tissue hypoxia and changes in renal blood flow⁶.

CKD significantly impacts leukocyte count and function. Many patients experience leukopenia or reduced white blood cell counts, particularly as CKD progresses, which weakens the immune system and raises infection risk. Additionally, leukocyte function is impaired in CKD; neutrophils, crucial for fighting bacterial infections,

exhibit reduced chemotaxis and phagocytosis. This dysfunction exacerbates the increased susceptibility to infections commonly observed in CKD patients^{7,8}.

In CKD, multiple factors contribute to thrombosis, including platelet activation, enhanced inflammation, uremic toxins and accelerated atherosclerosis. Conversely, the propensity for bleeding is influenced by platelet dysfunction, diminished platelet adhesion to vascular walls and the impact of uremic toxins. Numerous studies have demonstrated that individuals with CKD typically exhibit lower platelet counts compared to their healthy counterparts^{9,10}.

Despite the well-documented effects of CKD on hematological parameters, there remains a paucity of comprehensive studies that explore the relationship between different stages of CKD and specific hematological profiles. Understanding the hematological profile of CKD patients is crucial for early identification and management of complications, as well as for improving patient outcomes.

The primary objectives of this study were to assess the prevalence of anemia among predialytic CKD patients and analyze key hematological parameters—hemoglobin levels, red blood cell counts, white blood cell counts and platelet counts—across CKD stages 3, 4 and 5. Additionally, the study aimed to investigate the relationship between these hematological changes with CKD progression, identifying significant trends and correlations as the disease advances.

Materials & Methods:

This cross-sectional study was conducted at Sylhet MAG Osmani Medical College Hospital over 12 months (April 2019 to March 2020). A total of 60 patients with CKD stages 3 to 5 (eGFR < 60 mL/min/1.73 m² BSA) were recruited using purposive sampling. Patients on hemodialysis, with hematological disorders, recent hemorrhagic events or blood transfusions and having evidence of recent infection were excluded.

Data collection included the use of structured questionnaires to gather sociodemographic information (e.g., age, gender, education level, occupation) and clinical history from participants. CKD staging was performed using the CKD-EPI equation, which calculates the eGFR based on serum creatinine levels, age, sex, and race to classify patients into stages 3, 4 or

5. Blood samples were collected using standard venipuncture techniques by trained phlebotomists and hematological parameters—such as hemoglobin levels, RBC counts, WBC counts and platelet counts—were analyzed using automated hematology analyzers. Ethical approval was obtained from the institutional review board and written informed consent was secured from all participants before data collection.

Statistical Analysis

The statistical analysis for this study was performed using the Statistical Package for the Social Sciences (SPSS) software, version 26. Descriptive statistics were calculated for sociodemographic and clinical variables, including means, standard deviations (SD), medians and interquartile ranges (IQR) for continuous variables, while frequencies and percentages were determined for categorical variables. The normality of the distribution of continuous variables was assessed using the Shapiro-Wilk test.

To compare hematological parameters across different stages of CKD, one-way ANOVA was utilized for normally distributed data, while the Kruskal-Wallis test was applied for non-normally distributed data. Post-hoc analyses, such as Tukey's test for ANOVA and Dunn's test for the Kruskal-Wallis test, were performed

to identify specific group differences when significant results were found.

Spearman's rank correlation coefficient was calculated to evaluate the relationship between hematological parameters and CKD stages. Multiple linear regression analysis was employed to assess the impact of CKD stages on various hematological parameters while adjusting for potential confounding factors such as age, gender and comorbidities. A significance level of $p < 0.05$ was considered statistically significant for all tests. The R-squared value was reported for regression models to indicate the proportion of variance explained by the model. Data were presented in tables and figures to facilitate the interpretation of results, ensuring clarity in the presentation of findings.

Results:

Sociodemographic characteristics

Total patients were 60 with mean age of 53.7 years. Table I summarizes the base-line characteristics of the patients. There was 1 patient in Stage 3, 8 in Stage 4, and 51 in Stage 5.

Descriptive Statistics of Hematological Parameters

Table II presents the descriptive statistics of hematological parameters across different stages of

Table-I

Sociodemographic Table of CKD Patients (n= 60)

| Variable | Category | N | Percentage (%) |
|-----------------|-----------------------|----|----------------|
| Age (years) | Mean age : 53.7 years | | |
| | <30 | 2 | 3.3 |
| | 30-39 | 4 | 6.7 |
| | 40-49 | 12 | 20.0 |
| | 50-59 | 22 | 36.7 |
| | ≥60 | 20 | 33.3 |
| Gender | Male | 35 | 58.3 |
| | Female | 25 | 41.7 |
| Education Level | No formal education | 5 | 8.3 |
| | Primary | 15 | 25.0 |
| | Secondary | 20 | 33.3 |
| | Tertiary | 20 | 33.3 |
| Occupation | Unemployed | 10 | 16.7 |
| | Farmer | 15 | 25.0 |
| | Laborer | 20 | 33.3 |
| | Service Sector | 10 | 16.7 |
| | Business | 5 | 8.3 |
| Marital Status | Single | 2 | 3.3 |
| | Married | 53 | 88.3 |
| | Widowed | 5 | 8.3 |

Table-II

| <i>Descriptive Statistics of Hematological Parameters by Chronic Kidney Disease Stage</i> | | | |
|---|--------------|-----------------------|------------------------|
| Hematological Parameter (Mean \pm SD) | Stage 3(n=1) | Stage 4(n=8) | Stage 5(n=51) |
| Hemoglobin (g/dL) | 11.2 | 9.86 (\pm 0.39) | 7.81 (\pm 0.18) |
| RBC Count (millions/ μ L) | 4.05 | 3.93 (\pm 0.25) | 3.18 (\pm 0.64) |
| WBC Count (thousands/ μ L) | 14 | 7.96 (\pm 1.35) | 7.72 (\pm 2.01) |
| Platelet Count (thousands/ μ L) | 300 | 364.38 (\pm 95.37) | 400.05 (\pm 103.94) |

CKD. Hemoglobin levels decreased significantly ($p = 0.00$) with advancing CKD stages. Similarly, the RBC counts showed a decline, WBC counts were highest in Stage 3 at 14 thousand/ μ L, decreasing to 7.96 thousand/ μ L (\pm 1.35) in Stage 4 and 7.72 thousand/ μ L (\pm 2.01) in Stage 5. The platelet count increased across the stages, with averages of 300 thousand/ μ L in Stage 3, 364.38 thousand/ μ L (\pm 95.37) in Stage 4, and 400.05 thousand/ μ L (\pm 103.94) in Stage 5.

Correlation of Blood Parameters with CKD Stages

Table III presents the Spearman correlation coefficients between various blood parameters and the stages of CKD. Significant negative correlations were observed for hemoglobin ($r = -0.515$, $p < 0.001$) and RBC count ($r = -0.481$, $p < 0.001$) with CKD stages. Basophil count also showed a significant negative correlation ($r = -0.302$, $p = 0.019$). Other blood parameters, including WBC count, neutrophil count, lymphocyte count, monocyte count, eosinophil count, and platelet count, exhibited either weak or non-significant correlations with CKD stages.

ANOVA Results for Hematological Parameters across Different Stages of CKD

Hemoglobin level showed a significant difference between groups ($F = 12.357$, $p < 0.001$), indicating that

CKD stages affect hemoglobin levels (Table IV). Similarly, RBC absolute count ($F = 5.899$, $p = 0.005$) and absolute total WBC count ($F = 5.154$, $p = 0.009$) also demonstrated significant differences among CKD stages. Other parameters, including absolute neutrophil count, absolute lymphocyte count, absolute monocyte count, absolute eosinophil count, absolute basophil count, and absolute platelet count, did not show statistically significant differences across the CKD stages.

Regression Analysis Results

Regression analysis revealed significant relationships between CKD stages and hematological parameters. For hemoglobin levels, each increase in CKD stage led to a decrease of 1.717 units, with 33.3% of the variation explained by CKD stage ($p < 0.001$) (Table V). The RBC count decreased by 0.577 million cells/ μ L per CKD stage, with 17.4% of the variation explained ($p = 0.006$). The WBC count decreased by 1.702 thousand cells/ μ L per CKD stage, with 13.9% of the variation explained ($p = 0.011$). In contrast, platelet count increased by 29.720 thousand cells/ μ L per CKD stage, but this relationship was not statistically significant ($p = 0.834$), with only 4.5% of the variation explained by CKD stage.

Table-III

| <i>Correlation of Blood Parameters with CKD Stages</i> | | | |
|--|--------------------------|----------|---|
| Blood Parameter | Spearman Correlation (r) | p-value | Interpretation |
| Hemoglobin | -.515 | 0.000026 | Significant negative correlation |
| RBC Count | -.481 | 0.000101 | Significant negative correlation |
| WBC Count | -0.092678 | .481 | negative correlation, but Not Significant |
| Neutrophil count | -0.143953 | .272 | negative correlation, but Not Significant |
| Lymphocyte count | .204 | .118 | Positive correlation, but Not Significant |
| Monocyte Count | -0.107906 | .412 | negative correlation, but Not Significant |
| Basophil Count | -0.302222 | .019 | Significant negative correlation |
| Eosinophil Count | -0.218813 | .093 | negative correlation, but Not Significant |
| Platelet Count | .169 | .198 | Positive correlation, but Not Significant |

Table-IV*ANOVA Results for Hematological Parameters across Different Stages of Chronic Kidney Disease (CKD)*

| Hematological Parameter | Sum of Squares (Between Groups) | df (Between Groups) | Mean Square (Between Groups) | F | Sig. (p-value) |
|---------------------------|------------------------------------|------------------------|---------------------------------|--------|-------------------|
| Hemoglobin Level | 38.792 | 2 | 19.396 | 12.357 | 0.000 |
| RBC Absolute Count | 4.435 | 2 | 2.217 | 5.899 | 0.005 |
| Absolute Total WBC Count | 38.664 | 2 | 19.332 | 5.154 | 0.009 |
| Absolute Neutrophil Count | 18.577 | 2 | 9.289 | 2.896 | 0.063 |
| Absolute Lymphocyte Count | 0.992 | 2 | 0.496 | 1.024 | 0.366 |
| Absolute Monocyte Count | 0.024 | 2 | 0.012 | 0.156 | 0.856 |
| Absolute Eosinophil Count | 0.156 | 2 | 0.078 | 2.602 | 0.083 |
| Absolute Basophil Count | 0.003 | 2 | 0.002 | 1.578 | 0.215 |
| Absolute Platelet Count | 17721.235 | 2 | 8860.617 | 0.836 | 0.439 |

Table-V*Regression Summary for the Effect of Chronic Kidney Disease Stage on Hematological Parameters (adjusted for Age and Sex)*

| Dependent Variable | Coefficient (CKD Stage) | Standard Error | p-value | R-squared |
|--------------------|-------------------------|----------------|---------|-----------|
| Hemoglobin Level | -1.717 | .406 | <0.001 | 0.333 |
| RBC Absolute Count | -.577 | .203 | 0.006 | .174 |
| Total WBC Count | -1.702 | .648 | 0.011 | .139 |
| Platelet Count | 29.720 | .834 | .383 | .045 |

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Discussion:

This study aimed to evaluate the hematological profile of patients with predialytic CKD stages 3 to 5, revealing significant alterations in hematological parameters correlating with the progression of CKD. Our findings indicate a notable decrease in hemoglobin levels, RBC counts, and WBC counts as CKD stages advance, supporting existing literature that highlights the prevalence of anemia and leukopenia in CKD patients.

Anemia, a hallmark complication of CKD, was observed in all CKD stages, with the severity escalating in more advanced stages. The reported prevalence of anemia among patients with CKD ranges from 23.3% to 57.8% across various studies. This discrepancy can likely be attributed to differences in patient characteristics, the stage of CKD, ongoing treatment regimens, and dialysis status¹³⁻¹⁵. In this study, we observed a 100% prevalence of anemia among the participants. This finding can be

attributed to the distribution of patients across different stages of CKD, with only one patient in stage 3, eight in stage 4, and the remaining fifty-one in stage 5. The overwhelming majority of patients being in advanced stages of CKD likely accounts for the high prevalence of anemia observed in our cohort.

Our study observed a progressive decline in hemoglobin (Hb) levels across different stages of CKD, with mean Hb levels of 11.2 g/dL in stage 3, 9.86 ± 0.39 g/dL in stage 4, and 7.81 ± 0.18 g/dL in stage 5. These findings are consistent with other studies demonstrating similar trends of declining Hb levels as CKD progresses. For example, one study reported that the percentage of patients with Hb levels greater than 12 g/dL was significantly higher in stage 3 CKD compared to stages 4 and 5 (55.1% vs. 26.2% and 16.1%, respectively; $p < 0.001$). Conversely, the percentage of patients with Hb levels below 10 g/dL was substantially lower in stage

3 CKD compared to stages 4 and 5 (6.9% vs. 26.5% and 39.8%, respectively; $p < 0.001$), highlighting the progressive nature of anemia in advanced CKD stages [16]. Another study reported Hb levels ranging from 3.6 to 14.2 g/dL, with a mean of 9.31 ± 0.52 g/dL. In this cohort, 33% of patients had normal to mildly reduced Hb levels (>10 g/dL), while 67% had Hb levels below 10 g/dL. Notably, 60% and 74% of patients in stages 4 and 5 CKD, respectively, exhibited Hb levels below 10 g/dL¹⁷. These findings further corroborate our results, which also showed a significant decline in Hb levels in advanced CKD stages, underscoring the burden of anemia as kidney function deteriorates.

The similarities between our findings and those of previous studies reinforce the association between CKD progression and worsening anemia.

This situation highlights a significant gap in healthcare, indicating that the majority of our patients are either not seeking treatment early enough or are inadequately managed for their condition. This is alarming and calls for urgent attention in policy-making and subsequent implementation of treatment protocols. Additionally, untreated anemia not only exacerbates the burden of CKD but also directly increases cardiovascular risk and significantly impairs patients' quality of life. Therefore, addressing this issue through targeted interventions and improved access to care is essential to enhance outcomes for CKD patients and reduce the associated morbidity and mortality rates.

CKD stages significantly affect total leukocyte counts, with marked differences in absolute WBC counts across stages. Previous observations [18, 19] indicate that leukopenia is common in CKD, potentially compromising immune function. While total WBC counts show significant variations, specific leukocyte subsets, such as neutrophils and lymphocytes, do not exhibit statistically significant changes. CKD leads to immune activation and deficiency, resulting in systemic inflammation that contributes to complications like cardiovascular disease and anemia while impairing responses to infections. This state involves an expansion of monocytes with reduced phagocytic capacity, depletion of regulatory T cells, and decreased populations of T and B cells, compromising the immune response overall²⁰.

Our study observed a progressive decline in WBC count with decreasing GFR, highlighting an inverse relationship between CKD stage and WBC count. However, these findings contrast with results from some previously published studies. For instance, one study reported a mean WBC count of $6.4 \pm 3.9 \times 10^3/\text{iL}$ in stage 3 CKD and $6.6 \pm 3.3 \times 10^3/\text{iL}$ in stages 4 and 5 CKD, showing no significant change in WBC count across CKD stages²¹. Another study found a progressive increase in WBC count as GFR declined, contrary to the trend observed in our study²².

The discrepancy between our findings and those reported in other studies could be attributed to several factors. First, differences in study populations, including demographic variations and comorbid conditions, may have influenced WBC counts. For example, higher prevalence of inflammation or infections in the populations studied could explain the increased WBC counts observed in other studies. In contrast, the exclusion of patients with recent infections in our study might have contributed to the lower WBC counts observed in our cohort. Additionally, differences in the inclusion criteria, particularly regarding the severity of CKD and associated complications, may have led to varying WBC trends. Finally, the possibility of subclinical inflammation or immune dysregulation in advanced CKD stages, which may suppress WBC production or promote sequestration, could partially explain the declining WBC counts in our study.

This study revealed that CKD significantly impacts platelet count. While the data show a range of platelet counts across different stages of CKD, the overall trend indicates that platelet counts in patients with CKD are elevated compared to healthy individuals. This phenomenon may be attributed to factors such as increased platelet activation, a response to chronic inflammation, and the effects of uremic toxins. However, despite the elevated counts, CKD patients may still experience platelet dysfunction, leading to impaired hemostatic function and an increased risk of bleeding complications²³.

Our study observed a progressive increase in platelet count with advancing stages of CKD, which contrasts with findings from previous studies. For instance, one study reported median platelet counts of 199.0 (157.0–246.0), 191.0 (157.0–241.0), and 167.0 (131.0–210.0) $\times 10^3/$

iL in CKD stages 3, 4, and 5, respectively, indicating a progressive decline in platelet count as CKD advanced [24]. Similarly, another study demonstrated mean platelet counts of $2.09 \pm 0.85 \times 10^9 / \text{iL}$ in stage 3 CKD, $2.02 \pm 1.3 \times 10^9 / \text{iL}$ in stage 4, and $1.77 \pm 1.1 \times 10^9 / \text{iL}$ in stage 5, which also reflected a declining trend [25]. The divergence between our findings and those of earlier studies may be explained by several factors. First, variations in patient characteristics, such as the presence of comorbidities, nutritional status, or differences in platelet production and turnover, could have contributed to the observed discrepancies. For instance, thrombocytosis in some of our patients could be attributed to secondary causes such as chronic inflammation, iron deficiency, or subclinical infections that were undiagnosed at the time of data collection. Methodological differences, such as variations in sample size, study design, or platelet measurement techniques, may have influenced the findings. Population-level differences, including ethnicity or genetic factors affecting platelet physiology, could also explain the contrasting results. Additionally, compensatory mechanisms in CKD, such as increased thrombopoietin levels in response to impaired platelet function, might account for the rise in platelet count observed in our study. These responses may vary across populations, contributing to the differences in trends.

The discrepancy between platelet count and function highlights the complex interplay of CKD on hematological parameters and underscores the need for careful monitoring and management of these patients to mitigate potential cardiovascular and thrombotic risks. These patients have an attenuated response to dual antiplatelet therapy compared with patients without renal insufficiency further complicating the scenario [26].

Strengths of the Study

This study presents several strengths, including a comprehensive assessment of hematological parameters across different stages of CKD, which offers valuable insights into the impact of CKD on blood health. The use of appropriate statistical tools, such as regression analysis and ANOVA, increases the validity of results by skillfully handling confounding variables like age and sex. The alarming detection of 100% prevalence of anemia among advanced chronic kidney disease patients indicates a big clinical problem and requires immediate emphasis and consideration in treatment

modalities.

Limitations of the Study

Despite its strengths, there are notable limitations to the study. The cross-sectional design limits the establishment of causal relationships between the CKD stages and changes in hematological parameters; thus, any assessment of temporal changes calls for a longitudinal study design. Conducting the research at a single tertiary care hospital may limit the findings' generalizability to broader populations, and the small sample size in Stage 3 (only one patient) could affect the statistical power and reliability of results for this group. Additionally, while age and sex were controlled for, other potential confounding factors, such as comorbid conditions and medication use, were not accounted for, which could influence the result. The uneven distribution of patients across CKD stages, particularly the very small number in Stage 3 ($n=1$), can be considered a limitation of the study. This imbalance may limit the generalizability of findings across all CKD stages and affect statistical comparisons.

Conclusion

This study highlights a significant and progressive alteration in hematological parameters with advancing stages of CKD in predialytic patients. Hemoglobin levels, RBC counts, and WBC counts showed decline as CKD progressed, reflecting the increasing severity of anemia and immune dysregulation in advanced stages. In contrast, a rise in platelet count was observed, possibly indicating compensatory responses to platelet dysfunction in CKD. These findings underscore the importance of early detection and management of hematological abnormalities in CKD to improve patient outcomes. Future studies are recommended to explore the underlying mechanisms and validate these findings in larger, diverse populations.

Conflict of Interest

The authors declare that there are no conflicts of interest regarding this study.

Author Contributions:

Md. Asaduzzaman conceptualized the study, designed the methodology, collected data, and drafted the manuscript, including the final version. M M Jahangir Alam, Md. Jakaria Mahmud, Md. Rezaul Karim, Soumitra Roy, and Ranjon Kumer Roy contributed to study design

and critically reviewed the final draft. Mohammad Nazrul Islam, Khandaker Mohammad Rezwanul Islam, Sushanta Das, and Mohammad Alamgir Chowdhury contributed to the literature review and interpretation of results.

Funding

This research received no external funding.

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