



Dyslipidemia and Hematological Abnormalities in Hemodialysis Patients: A Study on the Progression of Chronic Kidney Disease in Karbala Governorate

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ABSTRACT

Keywords: Dyslipidemia, Hemodialysis, Chronic Kidney Disease, Hematological Parameters, Lipid Profile.



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Chronic Kidney Disease (CKD) is a significant global health concern impacting various physiological systems. This study investigates the effects of CKD on lipid profiles and complete blood count (CBC) in hemodialysis patients. The aim was to evaluate the alterations in these parameters in CKD patients undergoing dialysis and to understand their correlation with disease severity. A case-control study was conducted at Imam Hussein Teaching Hospital in Karbala Governorate, Iraq. The study included 80 patients (51 males and 29 females) aged between 21 and 62 years, who suffered from CKD and were undergoing renal dialysis for periods ranging from three months to 15 years. Patients were divided into two groups: those undergoing dialysis for more than a year (40 samples) and those undergoing dialysis for less than a year (40 samples). Blood samples were collected and analyzed for RBC, WBC, Hb, PLT, Cholesterol, HDL, and Triglyceride levels. The findings indicated a clear inverse relationship between CKD severity and mean levels of RBC, Hb, and PLT, with these levels decreasing significantly as the disease severity increased. While total cholesterol, triglyceride levels, and white blood cell count were relatively elevated with increasing disease severity, high-density lipoprotein (HDL) cholesterol levels remained stable in this study and were not affected by disease severity. The results of this study indicate that chronic kidney disease affects blood parameters, lipid metabolism, and lipid dysregulation. Therefore, it is essential to monitor lipid levels and blood parameters periodically in dialysis patients due to the increased risk of cardiovascular disease and other associated systemic risks.

1. INTRODUCTION

Kidney failure is a major global public health problem that affects millions of people worldwide and is associated with high morbidity and mortality. It is defined as the stage where the kidneys lose many of their vital functions, including their ability to filter and purify the blood, remove waste products from the body's, and maintain the body's water and fluid balance [1]. This condition can develop rapidly, within days or even hours. Acute kidney injury may develop rapidly within hours or days and is often characterized by severe oliguria or anuria. This condition is referred to as acute kidney failure. The other type of kidney failure is known as chronic kidney failure, which begins

with a gradual decline in kidney function over months or years and in several stages, which is the last stage of kidney failure, which leads to kidney damage and its inability to perform its vital functions [1–4]. Chronic kidney disease (CKD) is a progressive disorder characterized by irreversible loss of kidney function and is strongly associated with diabetes mellitus, hypertension, and cardiovascular disease [5]. Chronic kidney failure is defined as the stage in which the glomerular filtration units (nephrons) begin to lose their normal functions gradually and irreversibly [6], resulting in damage and destruction of more than 95% of kidney tissue, causing the accumulation of nitrogenous (metabolic) wastes and an increase in urea, fluids, and salts within the body. The kidneys also lose part

of their physiological activity and this leads to the accumulation of fluids and toxins in the body [7] A medical procedure that aims to remove waste, toxic substances and excess fluids from the body when the kidneys stop working properly due, Dialysis is a medical procedure and surgical intervention that helps the kidney to perform its vital functions [8]. The patient resorts to it in the last stage of kidney failure when the glomerular filtration rate reaches less than 15 ml/minute and the kidney loses 90% of its vital functions [9-10]. Patients with chronic kidney failure suffer from qualitative and quantitative abnormalities in their lipid profile [11]. The loss of vital kidney functions is a major cause of elevated triglyceride levels and decreased levels of HDL cholesterol, which plays an important role in fighting inflammation and as an antioxidant [12-13]. The decrease in HDL cholesterol activity may be due to decreased paraoxon and glutamine peroxidase activity [14], or it may be due to the negative effect of oxidative stress on cholesterol function [15], leading to an increase in lipoprotein concentration, which in turn causes a decrease in the activity of lipoprotein lipase enzyme [16-17], resulting in impaired triglyceride clearance [18]. Studies conducted on many kidney failure patients have shown that low HDL cholesterol levels are closely associated with an increased risk of cardiovascular disease [19-20] due to functional and structural abnormalities in cholesterol levels, making patients more susceptible to atherosclerosis [21-22]. In summary, patients with renal failure develop certain functional and structural abnormalities in HDL cholesterol which makes them prone to develop atherosclerosis and thus contributing to their CVD burden [23] chronic kidney disease (CKD) is a global public health concern that significantly impacts various physiological systems, including the hematological system [24] The kidneys play a crucial role in maintaining fluid balance, hormone production, and waste elimination, As CKD progresses, the kidneys lose their ability to perform these functions efficiently, leading to the accumulation of toxins and disturbances in bone marrow activity, immune function, and nutritional balance, these changes are directly reflected in alterations in the Complete Blood Count (CBC) [25] One of the most prominent hematological manifestations of CKD is anemia, primarily caused by reduced production of erythropoietin, a hormone essential for stimulating red blood cell production in the bone marrow [26] Additionally, CKD patients often experience platelet dysfunction and abnormalities in white blood cell counts due to uremic toxins and a compromised immune system [27] Research highlights the importance of CBC as a diagnostic tool in evaluating the severity and complications of CKD, Understanding the relationship between CKD and hematological parameters provides valuable insights for developing effective management strategies to improve patient outcomes [28]

Study Objectives

- ❖ To investigate the effects of chronic kidney disease (CKD) on lipid profiles and complete blood count (CBC) in hemodialysis patients.
- ❖ To identify significant alterations in lipid metabolism, characterized by dyslipidemia, which may contribute to an increased risk of cardiovascular complications in these patients.

- ❖ To observe notable abnormalities in hematological parameters, including anemia and variations in white blood cell and platelet counts.

2. MATERIALS AND METHODS

This study was conducted in Karbala Governorate, at Imam Hussein Teaching Hospital (Al-Husseini), Dialysis Department (Artificial Kidney). The study included 80 patients whose ages ranged between (21-62) years, the majority of whom were males (51 samples) and (29) females. All patients were undergoing dialysis.. The majority of patients were suffering from kidney stones, diabetes and irregular blood pressure. The study was divided into two phases... All dialysis patients underwent a physical examination and a brief questionnaire, including the patient s age, sex, disease severity, weight, height, number of dialysis times per week, and other diseases. (Chronic or hereditary), all patient information was stored in computerized systems in the ANOVA test. In two study stages, this was confirmed: Group first; 40 samples of patients with chronic renal failure undergoing dialysis for more than a year, Group second; 40 samples of patients with chronic renal failure undergoing dialysis for less than a year. Blood samples were taken from dialysis patients for the purpose of conducting the required medical examinations for patients. Five ml of blood was taken using a 5ml medical syringe and the blood was placed in gelatin tubes (gel tube) free of anti-clotting material, as it contains a gelatinous substance that helps to increase the separation of serum formed with the centrifugation process. At room temperature, the blood samples were left to coagulate for 15 minutes. They were then centrifuged at 2500 rpm for 5-10 minutes to separate the serum, which was kept at -20°C until testing Exclusion Criteria Patients (blood-liver-heart). Data Analysis The data were entered and statistically processed using SPSS version 27. Qualitative variables were expressed as frequencies and percentages, while quantitative variables were expressed as mean and standard deviation. The chi-square test and Fisher s exact test were used to examine the correlation between qualitative variables as needed, while Pearson s correlation coefficient was used to assess the relationship between quantitative variables. A statistical significance level of 0.05 was adopted. Results The demographic information presented in the Table (1), The mean age of the participants was 45.65 years and the mean of BMI was 25.55 Kg/m2. The samples consisted of 29 females and 51 males. majority (70) of the participants had a history of hypertension. About 45 participants had a history of DM, and 8 participants reported a history of other diseases. Regarding the disease severity, 20 participants had a disease severity of less than 1 year, 39 participants had a disease severity between 1 and 4 years and 21 participants had a disease severity of more than 5 years. The total number of dialysis sessions ranged from 16 to 2340 times.

Table 1: Distribution of the Samples According to demographic information

Age (Mean± SD)	45.65± 8.13
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Gender (No./ %)	Female	29
	Male	51
History of Hypertension (No./ %)	YES	70
	No	10
History of DM (No./ %)	YES	45
	No	35
History of Other disease (No./ %)	YES	8
	No	72
BMI Kg/m2(Mean± SD)		25.55± 4.45
Severity of disease (No./ %)	<1	20
	1-4Y	39
	>5	21
TOTAL No. of dialysis (min-max)		(16-2340)

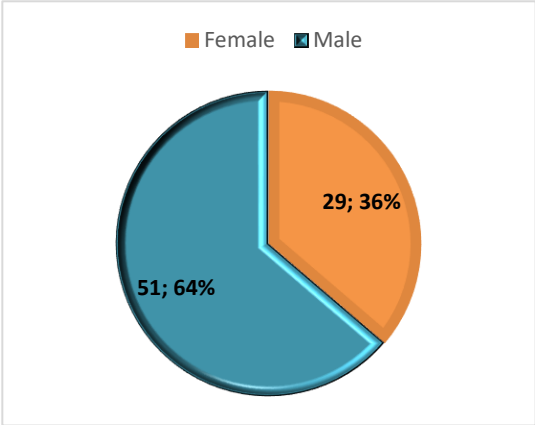


Figure 1: Gender distribution in the patients' groups

The mean level of red blood cell (RBC) in patients with chronic kidney disease (CKD) varied significantly across different disease severity, as presented in Table (2) & Figure (2). Patients with disease severity less than one year exhibited a mean RBC level of $3.3 \pm 0.68 \times 10^6$ cells/ μ L, with a median of 3.3×10^6 cells/ μ L (range: $2.0 - 4.8 \times 10^6$ cells/ μ L). Those with a disease severity of 1-4 years showed a mean RBC level of $3.1 \pm 0.85 \times 10^6$ cells/ μ L, with a median of 3.1×10^6 cells/ μ L (range: $1.5 - 5.0 \times 10^6$ cells/ μ L). Patients with a disease severity exceeding 5 years had the lowest mean RBC level, at $2.7 \pm 0.61 \times 10^6$ cells/ μ L, with a median of 2.8×10^6 cells/ μ L (range: $1.6 - 3.4 \times 10^6$ cells/ μ L). This data suggests a trend of decreasing RBC levels with increasing severity of CKD.

Table 2: Mean levels of RBC among patients with chronic kidney disease based on different disease severity.

RBC CKD Patients Group			
Severity of Diseases	Mean ± SD	Median(Min-Max)	P value
Acute	3.3 ± 0.68	3.3 (2.0 - 4.8)	0.04
chronic	3.1 ± 0.85	3.1 (1.5- 5.0)	
End stage	2.7 ± 0.61	2.8 (1.6- 3.4)	

*P value ≤ 0.05 was significant.

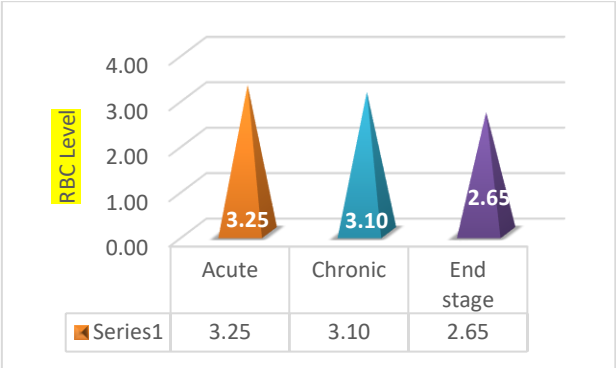


Figure 2: The distribution of serum RBC among patients with chronic kidney disease based on different disease severity.

Results were also shown that white blood cell (WBC) levels in patients with chronic kidney disease (CKD) demonstrated an increasing trend with longer disease severity, as presented in Table (3) & Figure (3). Patients with a disease severity of less than one year had a mean WBC count of $5.2 \pm 1.9 \times 10^3$ cells/ μ L, with a median of 5.3×10^3 cells/ μ L (range: $2.7 - 8.0 \times 10^3$ cells/ μ L). In patients with a disease severity of 1-4 years, the mean WBC level was $6.2 \pm 2.8 \times 10^3$ cells/ μ L, with a median of 5.4×10^3 cells/ μ L (range: $2.7 - 14 \times 10^3$ cells/ μ L). The group with a disease severity exceeding 5 years exhibited the highest mean WBC count, at $7.8 \pm 2.9 \times 10^3$ cells/ μ L, with a median of 6.4×10^3 cells/ μ L (range: $4.4 - 13 \times 10^3$ cells/ μ L). These findings indicate a potential association between increased CKD severity and elevated WBC counts.

Table 3: Mean levels of WBC among patients with chronic kidney disease based on different disease severity.

WBC CKD Patients Group			
Severity of Diseases	Mean ± SD	Median(Min-Max)	P value
Acute	5.2 ± 1.9	5.3 (2.7-8.0)	0.001
Chronic	6.2 ± 2.8	5.4 (2.7-14)	
End stage	7.8 ± 2.9	6.4 (4.4- 13)	

*P value ≤ 0.05 was significant.

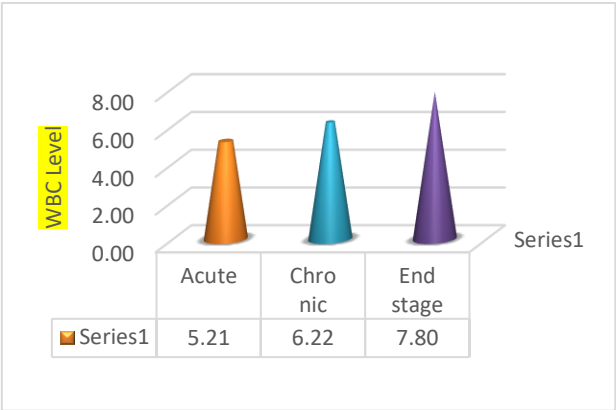


Figure 3: The distribution of WBC among patients with chronic kidney disease based on different disease severity.

On the other hand, the mean hemoglobin (Hb) levels in patients with chronic kidney disease (CKD) showed a trend of decreasing values with increasing disease severity, as

presented in Table (4) & Figure (4). Patients with a disease severity of less than one year had a mean Hb level of 9.7 ± 2.1 g/dL, with a median of 9.7 g/dL (range: 6.9 – 17 g/dL). In patients with a disease severity of 1-4 years, the mean Hb level was 9.2 ± 2.2 g/dL, with a median of 9.0 g/dL (range: 6.3 - 19.0 g/dL). The group with a disease severity exceeding 5 years exhibited the lowest mean Hb level, at 8.6 ± 0.95 g/dL, with a median of 8.8 g/dL (range: 6.1 – 9.8 g/dL). These findings suggest a correlation between longer CKD severity and lower hemoglobin concentrations.

Table 4: Mean levels of Hb among patients with chronic kidney disease based on different disease severity.

Hb	CKD Patients Group		
Severity of Diseases	Mean $\pm$ SD	Median(Min- Max)	P value
Acute	9.7 ± 2.1	9.7 (6.9- 17)	0.2
chronic	9.2 ± 2.2	9.0 (6.3- 19.0)	
End stage	8.6 ± 0.95	8.8 (6.1- 9.8)	

*P value ≥ 0.05 was no significant.

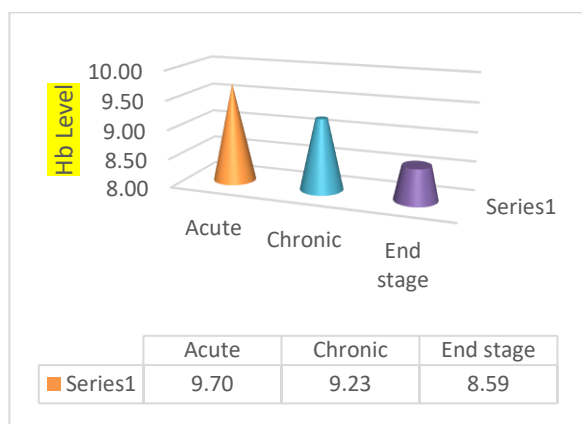


Figure 4: The distribution of Hb among patients with chronic kidney disease based on different disease severity.

Furthermore, the mean platelet (PLT) levels in patients with chronic kidney disease (CKD) demonstrated a decreasing trend with increasing disease severity, as presented in Table (5) & Figure (5). Patients with a disease severity of less than one year had a mean PLT count of $220 \pm 80 \times 10^3/\mu\text{L}$, with a median of $216 \times 10^3/\mu\text{L}$ (range: 107 – $372 \times 10^3/\mu\text{L}$). In patients with a disease severity of 1-4 years, the mean PLT level was $188 \pm 81 \times 10^3/\mu\text{L}$, with a median of $170 \times 10^3/\mu\text{L}$ (range: 66 – $472 \times 10^3/\mu\text{L}$). The group with a disease severity exceeding 5 years exhibited the lowest mean PLT count, at $153 \pm 46 \times 10^3/\mu\text{L}$, with a median of $167 \times 10^3/\mu\text{L}$ (range: 78 – $233 \times 10^3/\mu\text{L}$). These findings suggest an inverse relationship between CKD severity and platelet count, indicating a potential decrease in platelet levels with prolonged disease.

Table 5: Mean levels of serum PLT among patients with chronic kidney disease based on different disease severity.

PLT	CKD Patients Group		
Severity of Diseases	Mean $\pm$ SD	Median(Min- Max)	P value
Acute	220 ± 80	216 (107- 372)	0.01
chronic	188 ± 81	170 (66- 472)	
End stage	153 ± 46	167 (78- 233)	

*P value ≤ 0.05 was significant.

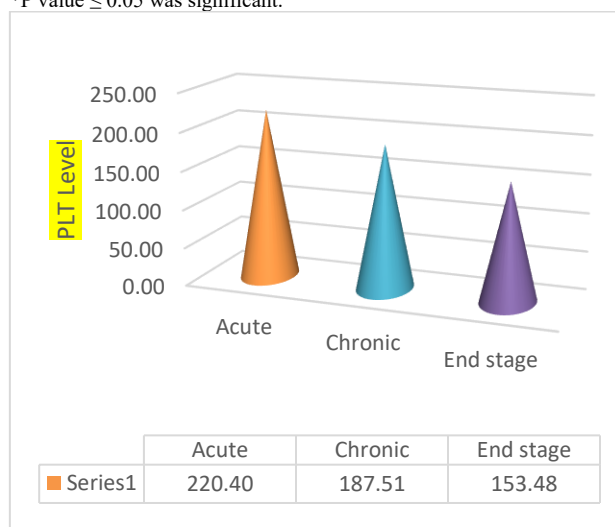


Figure 5: The distribution of PLT among patients with chronic kidney disease based on different disease severity.

In this study, the mean serum cholesterol levels in patients with chronic kidney disease (CKD) showed a clear increasing trend with longer disease severity, as presented in Table (6) & Figure (6). Patients with a disease severity of less than one year had a mean cholesterol level of 202 ± 72 mg/dL, with a median of 165 mg/dL (range: 135 – 361 mg/dL). In patients with a disease severity of 1-4 years, the mean cholesterol level was 275 ± 121 mg/dL, with a median of 252 mg/dL (range: 159 – 178 mg/dL). The group with a disease severity exceeding 5 years exhibited the highest mean cholesterol level, at 338 ± 139 mg/dL, with a median of 331 mg/dL (range: 660 – 628 mg/dL). Notably, the range values for the > 5 year group appear to have reversed minimum and maximum values. Overall, these findings indicate a positive correlation between CKD severity and serum cholesterol levels, suggesting that cholesterol levels tend to increase with prolonged disease.

Table 6: Mean levels of serum Cholesterol among patients with chronic kidney disease based on different disease severity.

cholesterol	CKD Patients Group		
Severity of Diseases	Mean $\pm$ SD	Median(Min- Max)	P value
Acute	202 ± 72	165 (135-361)	0.001
Chronic	275 ± 121	252 (159-178)	
End stage	338 ± 139	331 (660 -628)	

*P value ≤ 0.05 was significant.

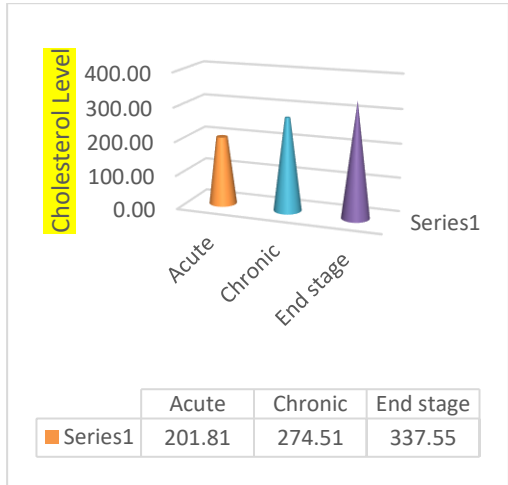


Figure 6: The distribution of serum Cholesterol among patients with chronic kidney disease based on different disease severity.

While, the mean serum high-density lipoprotein (HDL) levels in patients with chronic kidney disease (CKD) showed relatively consistent values across different disease severity, as presented in Table (7) & Figure (7). Patients with a disease severity of less than one year had a mean HDL level of 34 ± 7.3 mg/dL, with a median of 31 mg/dL (range: 23 – 54 mg/dL). In patients with a disease severity of 1-4 years, the mean HDL level was 34 ± 6.6 mg/dL, with a median of 34 mg/dL (range: 21 – 55 mg/dL). The group with a disease severity exceeding 5 years exhibited a mean HDL level of 35 ± 8.4 mg/dL, with a median of 34 mg/dL (range: 22 – 54 mg/dL). The results of this study indicated that high-density lipoprotein levels were relatively stable and were not affected by the severity of the disease.

Table 7: Mean levels of serum HDL among patients with chronic kidney disease based on different disease severity.

HDL CKD Patients Group			
Severity of Diseases	Mean ± SD	Median (Min- Max)	P value
Acute	34±7.3	31 (23 - 54)	0.3
Chronic	34 ± 6.6	34 (21 - 55)	
End stage	35 ± 8.4	34 (22- 54)	

*P value ≥ 0.05 was no significant.

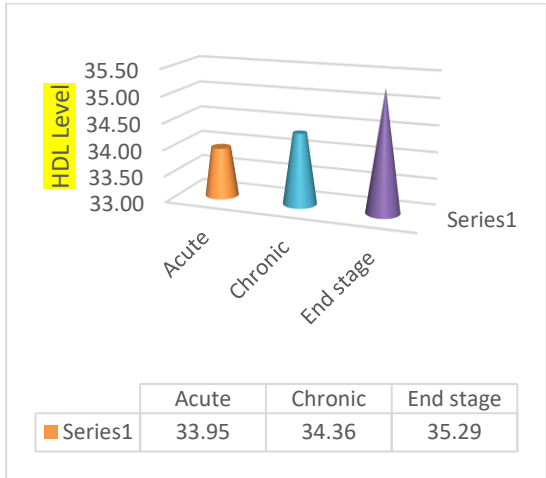


Figure 7: The distribution of serum HDL among patients with chronic kidney disease based on different disease severity.

It is worth noting that triglyceride levels in this study were associated with increased disease duration, with triglyceride levels in patients with less than a year of illness ranging between 215 ± 64 mg/dL, with a median of 191 mg/dL (range: 121 – 381 mg/dL). In those with the disease who had it for 1-4 years, triglyceride levels ranged between 267 ± 115 mg/dL, with a median of 261 mg/dL (range: 116 – 613 mg/dL). However, for patients with kidney failure for more than 5 years, triglyceride levels were high, sometimes reaching, at 324 ± 145 mg/dL, with a median of 273 mg/dL (range: 140 – 685 mg/dL). The results of this study show that with an increase in the duration of the disease, the rates of elevated triglycerides increase, as shown in Table (8) and Figure (8).

Table 8: Mean levels of serum Triglyceride among patients with chronic kidney disease based on different disease severity.

Tri.g CKD Patients Group			
Severity of Diseases	Mean ± SD	Median (Min- Max)	P value
Acute	215 ± 64	191 (121 - 381)	0.001
Chronic	267 ± 115	261 (116- 613)	
End stage	324 ±145	273 (140 - 685)	

*P value ≤ 0.05 was significant.

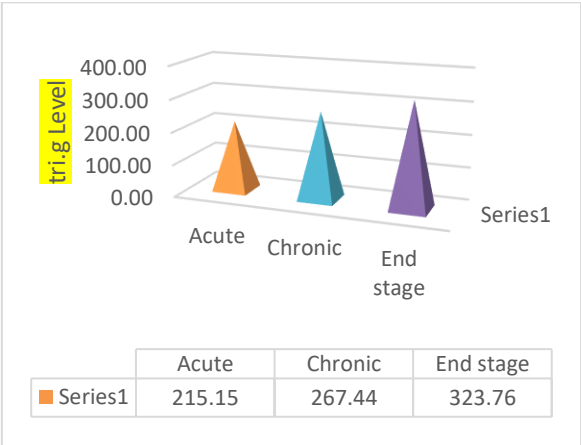


Figure 8: The distribution of serum triglyceride among patients with chronic kidney disease based on different disease severity.

3. DISCUSSION

There's a clear inverse relationship between the severity of CKD and mean RBC levels. As the severity of CKD increases, the mean RBC count decreases. The decrease in RBC levels is notable, especially when comparing the acute group (<1 year) to the end stage (>5 years) group. CKD often leads to reduced production of EPO, a hormone produced by the kidneys that stimulates red blood cell production. As kidney function declines with increasing disease severity, EPO deficiency becomes more pronounced, resulting in lower RBC levels (27, 29). CKD patients may also experience iron deficiency, which further contributes to anemia, this could be due to reduced iron absorption, blood loss, or inflammation. Chronic inflammation, common in CKD, can suppress erythropoiesis (RBC production) (25). The longer the disease severity, the greater the potential for chronic

inflammation to impact RBC levels. Uremic toxins, which accumulate in the blood as kidney function deteriorates, can suppress bone marrow function, further hindering RBC production. The observed decrease in RBC levels indicates the progressive development of anemia in CKD patients [30]. Anemia can cause several complications in dialysis patients, including fatigue and cardiovascular complications. The results of this study are consistent with previous findings indicating that anemia is a common complication in patients with renal failure, characterized by low hemoglobin levels, low transferrin saturation, and high or normal ferritin levels. Secondary hyperparathyroidism with increased erythropoietin resistance is another cause of anemia in these patients [31]. Finally, hepcidin, which is a key regulator of circulating iron absorption, has been found to be involved in the etiology of anemia in CKD [32]. Its concentrations were demonstrated to be influenced by inflammation [33]. The fact that anemia develops in spite of elevated EPO levels in CKD suggests that peripheral resistance or hypo responsiveness to EPO may be the factual reason for its occurrence [34]. Results were also shown a clear positive correlation between the severity of CKD and mean WBC levels. As the severity of CKD increases, the mean WBC count also increases. The increase is substantial, notably from the <1 year group to the >5 years group. CKD is associated with a state of chronic inflammation. This inflammation can stimulate the production and release of WBCs, leading to elevated counts. The longer the disease severity, the greater the potential for sustained and heightened inflammation. The accumulation of uremic toxins in CKD can also contribute to systemic inflammation and immune system activation, resulting in increased WBC count [35]. CKD patients are at increased risk of infections due to impaired immune function. The elevated WBC counts may, in part, reflect the body's response to underlying or subclinical infections, other comorbidities, such as diabetes or cardiovascular disease, which are common in CKD patients, can also contribute to chronic inflammation and elevated WBC counts. Elevated WBC counts in CKD patients may indicate an increased risk of cardiovascular events, infections, and overall morbidity [36]. It is important to rule out infection as a cause of the elevated counts. Some conflicting medical evidence suggests a link between deteriorating kidney function and the level of white blood cells in the body [37]. Some studies have also indicated that an elevated white blood cell count may be an indicator of future kidney function decline [38]. Hemoglobin (Hb) levels in CKD patients based on disease severity demonstrated a clear inverse correlation. As the severity of CKD increases, mean Hb levels decrease, the decline in Hb is progressive, with the lowest levels observed in patients with the end stage (>5 years), the kidneys produce EPO, which stimulates red blood cell production [39]. In CKD, kidney function declines, leading to reduced EPO production and consequently, lower Hb levels. The longer the disease severity, the more pronounced the kidney function decline and EPO deficiency [40], CKD patients often experience iron deficiency due to reduced iron absorption, blood loss (e.g., from dialysis), and inflammation. Iron deficiency further contributes to anemia and lower Hb levels [41]. Chronic inflammation, a

hallmark of CKD, can suppress erythropoiesis (red blood cell production) and contribute to anemia, low Hb levels indicate anemia, a common and significant complication of CKD [35]. Anemia can lead to fatigue, weakness, cardiovascular complications, and reduced quality of life. Early detection of hemoglobin levels in kidney failure patients plays an important role in helping to detect and treat anemia in these patients [42].

This study found an inverse relationship between low platelet levels and the severity of kidney failure. As the severity of CKD increases, the mean PLT count decreases, the decrease in PLT count is progressive, with the lowest levels observed in patients with the longest disease severity (>5 years). Uremic toxins, which accumulate in CKD, can suppress bone marrow function, including the production of platelets. This suppression may become more pronounced with longer disease duration. CKD can lead to increased platelet destruction due to factors such as increased oxidative stress, inflammation, and altered platelet activation [43]. Although platelet counts are decreasing, it is also important to know that CKD can result in dysfunctional platelets. Meaning that even at higher counts, they may not be functioning correctly. Chronic inflammation, common in CKD, can impact platelet production and turnover [44]. On the other hand, positive correlation between CKD severity and mean serum cholesterol levels was found. The increase is progressive and substantial, particularly between the acute (<1 year group) and end stage (>5 years group). Chronic kidney failure can cause problems with lipid levels, and these problems worsen with the duration of the disease, leading to elevated levels of both total cholesterol and triglycerides. Increased total cholesterol levels in the blood can be a major risk factor for cardiovascular disease in patients, and consequently, an increased risk of death [15]. The results of this study showed that levels of good cholesterol were not significantly affected by the increased duration of the disease, unlike the levels of both triglycerides and total cholesterol, which increased with the duration of the disease. This is attributed to the protective role of good cholesterol against cardiovascular diseases [45].

The results of this study showed that high triglyceride levels are closely linked to increased disease duration, with the highest triglyceride levels recorded in patients with kidney failure lasting more than five years. This led to impaired lipid clearance in the blood, causing reduced lipoprotein (VLDL) activity [46].

4. CONCLUSIONS

1. This study demonstrated that chronic kidney disease is one of the world's leading causes of blood dyscrasias and lipid metabolism abnormalities, leading to an increased risk of cardiovascular disease due to elevated triglyceride and total cholesterol levels and dyslipidemia in patients. It also increases the risk of systemic inflammation and impaired bone marrow function due to significant changes in white blood cell and platelet counts, in addition to the risk of anemia.
2. The results of this study confirm the need for regular monitoring of lipid levels and blood count

parameters in dialysis patients to reduce associated risks, in addition to emphasizing the importance of research to develop therapeutic interventions to control lipid levels and blood counts in these patients.

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AUTHORS' DECLARATION

We confirm that all the Figures and Tables in the manuscript belong to the current study.

CONFLICT OF INTEREST

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

REFERENCES

- [1] Adams GR, Vaziri ND. Skeletal muscle dysfunction in chronic renal failure: effects of exercise. *American Journal of Physiology-Renal Physiology* 2006.
- [2] Zuzda K, Walczak-Wieteska P, Andruszkiewicz P, Małyszko J. Acute Kidney Injury Biomarkers in Perioperative Care: A Scoping Review of Clinical Implementation. *Diagnostics* 2025; 16:94.
- [3] Webster AC, Nagler E V, Morton RL, Masson P. Chronic kidney disease. *The Lancet* 2017; 389:1238–52.
- [4] Makris K. The role of the clinical laboratory in the detection and monitoring of acute kidney injury. *J Lab Precis Med* 2018;3.
- [5] Khudhair DA, Kareem HL, Yaseen MA, Mohammed HA. Assessment of Arginase II with Biochemical Changes in Patients with Chronic Kidney Disease. *Medical Journal of Babylon* 2024; 21:748–52.
- [6] Raoof AA, Al-Saedi AJH, Nabil D. Correlation of Fibroblast Growth Factor-23 with Thyroid Disorder in Patients with Chronic Kidney Disease. *Medical Journal of Babylon* 2025; 22:28–32.
- [7] Habib A, Ahmad R, Rehman S. Hematological changes in patients of chronic renal failure and the effect of hemodialysis on these parameters. *Int J Res Med Sci* 2017; 5:4998–5003.
- [8] Nissenson AR, Fine RN, Mehrotra R, Zaritsky J. Handbook of dialysis therapy, E-Book. Elsevier Health Sciences; 2022.
- [9] Himmelfarb J, Vanholder R, Mehrotra R, Tonelli M. The current and future landscape of dialysis. *Nat Rev Nephrol* 2020; 16:573–85.
- [10] Ghodsian S, Ghafourifard M, Ghahramanian A. Comparison of shared decision making in patients undergoing hemodialysis and peritoneal dialysis for choosing a dialysis modality. *BMC Nephrol* 2021; 22:67.
- [11] Lacquaniti A, Bolignano D, Donato V, Bono C, Fazio MR, Buemi M. Alterations of lipid metabolism in chronic nephropathies: mechanisms, diagnosis and treatment. *Kidney Blood Press Res* 2010; 33:100–10.
- [12] Hall YN, Larive B, Painter P, Kaysen GA, Lindsay RM, Nissenson AR, et al. Effects of six versus three times per week hemodialysis on physical performance, health, and functioning: Frequent Hemodialysis Network (FHN) randomized trials. *Clinical Journal of the American Society of Nephrology* 2012; 7:782–94.
- [13] Lacount S, Tannock LR. Dyslipidemia in Chronic Kidney Disease. In: Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; Updated 2025 Apr 30.
- [14] Moradi H, Vaziri ND, Kashyap ML, Said HM, Kalantar-Zadeh K. Role of HDL dysfunction in end-stage renal disease: a double-edged sword. *Journal of Renal Nutrition* 2013; 23:203–6.
- [15] Barbagallo CM, Cefalù AB, Giammanco A, Noto D, Caldarella R, Ciaccio M, et al. Lipoprotein abnormalities in chronic kidney disease and renal transplantation 2021;11:315.
- [16] Gao F, Feng G, Li H, Qin W, Xiao C. Scavenger receptor BI induced by HDL from coronary heart disease may be related to atherosclerosis. *Clinical and Applied Thrombosis/Hemostasis* 2021; 27:10760296211029710.
- [17] Lee W-C, Chen J-B, Moi S-H, Yang C-H. Association of proportion of the HDL-cholesterol subclasses HDL-2b and HDL-3 and macrovascular events among patients undergoing hemodialysis. *Sci Rep* 2021; 11:1871.
- [18] Tsinari A, Roumeliotis S, et al. The Clinical Utility and Plausibility of Oxidative and Antioxidant Variables in Chronic and End-Stage Kidney Disease: A Review of the Literature. *Int J Mol Sci*. 2025;26(7):3376.
- [19] Honda H, Ueda M, Kojima S, Mashiba S, Michihata T, Takahashi K, et al. Oxidized high-density lipoprotein as a risk factor for cardiovascular events in prevalent hemodialysis patients. *Atherosclerosis* 2012; 220:493–501.
- [20] Deebeel NA, Matthew AN, Loloi J, Bernstein AP, Thirumavalavan N, Ramasamy R. Testosterone deficiency in men with end stage renal disease and kidney transplantation: a narrative review. *Int J Impot Res* 2025; 37:271–7.
- [21] Kazmi S, Zarovniaeva V, Cortez Perez K, et al. Iron-Deficiency Anemia in Chronic Kidney Disease: A Literature Review of Its Pathophysiology, Diagnosis, and Management. *Cureus*. 2025
- [22] Kenkre JS, Mazaheri T, Neely RDG, Soran H, Datta D, Penson P, et al. Standardising lipid testing and reporting in the United Kingdom; a joint statement by HEART UK and The Association for Laboratory Medicine. *Ann Clin Biochem* 2025; 62:257–86.
- [23] Sevinc C, Yilmaz G, Ustundag S. The relationship between calcification inhibitor levels in chronic kidney disease and the development of atherosclerosis. *Ren Fail* 2021; 43:1349–58.

- [24] Gwozdziński K, Pieniazek A, Gwozdziński L. Reactive oxygen species and their involvement in red blood cell damage in chronic kidney disease. *Oxid Med Cell Longev* 2021; 2021:6639199.
- [25] Hanna RM, Streja E, Kalantar-Zadeh K. Burden of anemia in chronic kidney disease: beyond erythropoietin. *Adv Ther* 2021; 38:52–75.
- [26] Wu HHL, Chinnadurai R. Erythropoietin-stimulating agent hyporesponsiveness in patients living with chronic kidney disease. *Kidney Diseases* 2022; 8:103–14.
- [27] Jain N, Corken AL, Kumar A, Davis CL, Ware J, Arthur JM. Role of platelets in chronic kidney disease. *Journal of the American Society of Nephrology* 2021; 32:1551–8.
- [28] El Kodmiri Z, Ghazi B, Benaddi EH, Ouamani A, Ahnach M. Evaluating Hematological Changes in Hemodialysis Patients: A Retrospective Study in a Moroccan Hospital. *Journal of Nursing, Education Sciences, and Medical Practice* 2024;1:21–31.
- [29] Alzubaidy SA, Mohammed MM, Malik AS. Evaluation of Erythropoietin Stimulating Agent's Responsiveness and Associated Factors in Hemodialysis Patients with Anemia. *Medical Journal of Babylon* 2024; 21:551–5.
- [30] Zhang H, Fan L, Liao H, Tu L, Zhang J, Xu D, et al. Correlations of cardiac function with inflammation, oxidative stress and anemia in patients with uremia. *Exp Ther Med* 2021; 21:250.
- [31] Gupta J, Mitra N, Kanetsky PA, Devaney J, Wing MR, Reilly M, et al. Association between albuminuria, kidney function, and inflammatory biomarker profile in CKD in CRIC. *Clinical Journal of the American Society of Nephrology* 2012; 7:1938–46.
- [32] Yilmaz MI, Solak Y, Covic A, Goldsmith D, Kanbay M. Renal anemia of inflammation: the name is self-explanatory. *Blood Purif* 2011; 32:220–5.
- [33] Tsai Y-C, Hung C-C, Kuo M-C, Tsai J-C, Yeh S-M, Hwang S-J, et al. Association of hsCRP, white blood cell count and ferritin with renal outcome in chronic kidney disease patients. *PLoS One* 2012;7: e52775.
- [34] Fan F, Jia J, Li J, Huo Y, Zhang Y. White blood cell count predicts the odds of kidney function decline in a Chinese community-based population. *BMC Nephrol* 2017; 18:190.
- [35] Kadatane SP, Satariano M, Massey M, Mongan K, Raina R. The role of inflammation in CKD. *Cells* 2023; 12:1581.
- [36] Sun Y, Jiang L, Shao X. Predictive value of procalcitonin for diagnosis of infections in patients with chronic kidney disease: a comparison with traditional inflammatory markers C-reactive protein, white blood cell count, and neutrophil percentage. *Int Urol Nephrol* 2017; 49:2205–16.
- [37] Hamza E, Metzinger L, Metzinger-Le Meuth V. Uremic toxins affect erythropoiesis during the course of chronic kidney disease: a review. *Cells* 2020; 9:2039.
- [38] Baaten CC, Sternkopf M, Henning T, Marx N, Jankowski J, Noels H. Platelet function in CKD: a systematic review and meta-analysis. *Journal of the American Society of Nephrology* 2021;32:1583–98.
- [39] Fujita Y, Doi Y, Hamano T, Hatazaki M, Umayahara Y, Isaka Y, et al. Low erythropoietin levels predict faster renal function decline in diabetic patients with anemia: a prospective cohort study. *Sci Rep* 2019; 9:14871.
- [40] Panjeta M, Tahirović I, Sofić E, Ćorić J, Dervišević A. Interpretation of erythropoietin and haemoglobin levels in patients with various stages of chronic kidney disease. *J Med Biochem* 2017; 36:145.
- [41] Smith Jr RE. The clinical and economic burden of anemia. *Am J Manag Care* 2010;16: S59–S66.
- [42] Ratcliffe LEK, Thomas W, Glen J, Padhi S, Pordes BAJ, Wonderling D, et al. Diagnosis and management of iron deficiency in CKD: a summary of the NICE guideline recommendations and their rationale. *American Journal of Kidney Diseases* 2016; 67:548–58.
- [43] Lacount S, Tannock LR. Dyslipidemia in chronic kidney disease. *Endotext* [Internet] 2025.
- [44] Nam KH, Chang TI, Joo YS, Kim J, Lee S, Lee C, et al. Association between serum high-density lipoprotein cholesterol levels and progression of chronic kidney disease: results from the KNOW-CKD. *J Am Heart Assoc* 2019;8: e011162.
- [45] Obermayr RP, Temml C, Knechtelsdorfer M, Gutjahr G, Kletzmayer J, Heiss S, et al. Predictors of new-onset decline in kidney function in a general middle-european population. *Nephrology Dialysis Transplantation* 2008; 23:1265–73.
- [46] Bah MS, Togtoga L, Ndong A, Ndoye PD, Niang K, Ndiaye P. Overweight/Obesity: Prevalence and Epidemiological Profile among High School Students in the District of Bamako. *Rwanda Public Health Bulletin* 2023; 4:12–21.