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Prevalence of Anemia in Patients with Chronic Kidney Disease in Baghdad City: A Cross-Sectional Study

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Annotation: Background: Anemia is a common complication of chronic kidney disease (CKD) with significant health implications. This study aimed to determine the prevalence of anemia among patients with CKD in Baghdad, Iraq, and to identify associated clinical and laboratory parameters across different stages of CKD.

Methods: A cross-sectional study was conducted from June 2024 to January 2025 in a private hospital in Baghdad. A total of 462 adult CKD patients were enrolled. Demographic data, medical history, and laboratory parameters were collected. Anemia was defined according to WHO criteria (hemoglobin <13 g/dL in males and <12 g/dL in females). CKD was staged according to estimated glomerular filtration rate (eGFR) using the CKD-EPI equation.

Results: The overall prevalence of anemia was 67.5% among CKD patients, with increasing rates corresponding to advancing CKD stages: 28.6% in stage 1, 43.7% in stage 2, 62.9% in stage 3, 86.3% in stage 4, and 94.7% in stage 5. Mean hemoglobin levels showed progressive decline with worsening kidney function (13.1±1.6 g/dL in stage 1 vs. 8.7±1.4 g/dL in stage 5, $p<0.001$). Multivariate analysis identified reduced eGFR, female gender, diabetes mellitus, and low transferrin saturation as independent predictors of anemia in CKD patients.

Conclusion: Anemia is highly prevalent among CKD patients in Baghdad, with rates that exceed some international reports. The strong association with declining renal function underscores the need for routine anemia screening, particularly in advanced stages of CKD. Targeted interventions focusing on high-risk groups may improve outcomes in this vulnerable population.

Keywords: Anemia, chronic kidney disease, prevalence, hemoglobin, Baghdad, Iraq.

Introduction

Chronic kidney disease (CKD) represents a significant global health burden, affecting approximately 10-15% of the adult population worldwide [1,2, 3,4]. In Iraq, the prevalence of CKD has been estimated at 11.2-13.5%, with higher rates in urban centers such as Baghdad due to increased prevalence of hypertension, diabetes, and environmental factors specific to the region [5,6]. As kidney function progressively deteriorates, patients experience a multitude of complications, with anemia being one of the most common and clinically significant [7,8].

Anemia in chronic kidney disease (CKD) is defined by the World Health Organization (WHO) as hemoglobin levels below 13.0 g/dL in men and 12.0 g/dL in women [9]. This definition has been widely adopted in clinical practice and research; however, some experts advocate for age- and ethnicity-specific thresholds that may better reflect physiological variations [10]. In the Iraqi population, where genetic factors such as thalassemia traits are more prevalent than in Western populations, the interpretation of anemia parameters requires additional considerations [11].

The primary mechanism underlying anemia in chronic kidney disease (CKD) is erythropoietin (EPO) deficiency, as the kidneys are the principal site of EPO production in adults [12,13]. EPO, a glycoprotein hormone produced predominantly by interstitial fibroblasts in the renal cortex and outer medulla, stimulates the proliferation and differentiation of erythroid progenitor cells in the bone marrow. As nephron mass decreases with progressive CKD, EPO production becomes impaired,

leading to reduced erythropoiesis and subsequent anemia [14,15]. This pathophysiological model is particularly relevant in the Iraqi context, where late referral to nephrology care is common, resulting in many patients presenting with advanced kidney disease and established anemia [16,17].

However, the pathophysiology of anemia in CKD extends beyond EPO deficiency, encompassing a complex interplay of factors that collectively impair erythropoiesis and reduce red blood cell survival [18]. Inflammation, a common feature in CKD, plays a pivotal role through the production of pro-inflammatory cytokines such as interleukin-6 (IL-6), which stimulates hepatic synthesis of hepcidin [19]. Hepcidin, a key regulator of iron metabolism, inhibits intestinal iron absorption and iron release from macrophages, leading to functional iron deficiency despite adequate iron stores [18,20]. This mechanism is especially significant in the Baghdad population, where chronic inflammatory conditions are prevalent due to environmental factors, recurrent infections, and the lingering health effects of previous conflicts [21,22].

Iron deficiency, both absolute and functional, represents another critical component of CKD-associated anemia [11,23]. Absolute iron deficiency in CKD patients may result from reduced dietary intake, impaired intestinal absorption, and increased losses due to uremic platelet dysfunction, frequent phlebotomy, and dialysis procedures [13,24]. In the healthcare setting of Baghdad, where resources for intravenous iron administration may be limited and compliance with oral iron supplementation is often suboptimal due to gastrointestinal side effects, managing iron deficiency presents particular challenges that may influence anemia prevalence and severity [25,26].

The uremic milieu in advanced chronic kidney disease (CKD) further contributes to anemia through the accumulation of uremic toxins that directly inhibit erythropoiesis and reduce red blood cell survival [13, 25]. These toxins impair the proliferation and differentiation of erythroid progenitor cells, increasing oxidative stress and resulting in a shortened red blood cell lifespan [25]. Additionally, secondary hyperparathyroidism, a common complication of CKD, leads to bone marrow fibrosis and further impairment of erythropoiesis [26]. The management of these metabolic derangements is particularly challenging in resource-limited settings, potentially contributing to higher anemia prevalence in regions like Baghdad [23].

The clinical impact of anemia in CKD extends far beyond the classic symptoms of fatigue and reduced exercise capacity. Anemia significantly contributes to cardiovascular morbidity and mortality through mechanisms including increased cardiac output, pathological cardiac remodeling, and myocardial hypoxia [7,24]. In a population already burdened with high cardiovascular risk profiles, as observed in urban Iraqi settings, anemia may represent a particularly important modifiable risk factor [22]. Furthermore, anemia adversely affects cognitive function, quality of life, and physical capacity, dimensions that are increasingly recognized as critical outcomes in CKD management [24].

The prevalence of anemia in chronic kidney disease (CKD) exhibits significant geographical variation, influenced by factors including genetics, nutritional status, comorbidities, healthcare access, and clinical practice patterns [8,9,23]. While extensive epidemiological data exist from North America, Europe, and parts of Asia [8,9,23], there is a paucity of comprehensive studies from the Middle East, particularly Iraq [1,22,30]. The few available studies from neighboring countries [5,6,10] suggest potentially higher anemia prevalence in this region compared to Western populations, but methodological differences and variable definitions limit direct comparisons [9].

Baghdad, as Iraq's capital and largest city with a population exceeding 7 million, represents a critical location for studying CKD complications [1,3]. The city's healthcare system faces unique challenges, including infrastructure limitations, medication supply interruptions, and resource constraints that may impact anemia management practices [22]. Furthermore, Baghdad's population exhibits distinctive clinical characteristics, including a higher prevalence of consanguinity (affecting genetic predisposition to certain renal diseases), dietary patterns, environmental exposures, and comorbidity profiles that may influence the development and progression of anemia in CKD [1,22].

The management of anemia in chronic kidney disease (CKD) has evolved significantly over recent decades, particularly with the introduction of erythropoiesis-stimulating agents (ESAs) and intravenous iron preparations [3, 12, 13]. However, access to these therapies varies considerably across healthcare settings, with potential disparities in resource-limited environments [22]. In Baghdad's healthcare system, where medication availability may be inconsistent and cost constraints significant, understanding local anemia prevalence and patterns is essential for developing contextually appropriate screening and management strategies [1].

Despite the clinical significance of anemia in chronic kidney disease (CKD) and its impact on patient outcomes [7,24], comprehensive epidemiological data from Iraq remain limited [1,22]. Previous studies have typically focused on dialysis populations, included small sample sizes, or employed variable definitions that complicate interpretation [1,27,30]. The lack of robust data across the spectrum of CKD stages represents a significant knowledge gap, particularly given the potentially unique characteristics of the Baghdad population.

This study aims to address the knowledge gap by determining the prevalence of anemia across different stages of chronic kidney disease (CKD) in Baghdad's adult population, examining associated clinical and laboratory parameters, and identifying independent predictors of anemia. By providing locally relevant epidemiological data, this study seeks to inform screening practices, resource allocation, and management strategies for CKD-associated anemia in Baghdad and similar urban settings in the region.

Methodology

Study Design and Setting

This cross-sectional study was conducted from June 2024 to January 2025 across a private hospital in Baghdad, Iraq. The hospital was selected to ensure a representative sample of the chronic kidney disease (CKD) population in Baghdad, as it provides care to patients from diverse socioeconomic backgrounds and geographical areas within the city. The study protocol adhered to the Declaration of Helsinki. Written informed consent was obtained from all participants before enrollment.

Study Population

Adult patients (≥18 years) with confirmed chronic kidney disease (CKD) attending the nephrology outpatient clinics or receiving inpatient care at participating centers were eligible for inclusion. CKD was defined according to the Kidney Disease: Improving Global Outcomes (KDIGO) criteria as abnormalities of kidney structure or function, present for more than 3 months, with health implications. Patients were classified into CKD stages based on estimated glomerular filtration rate (eGFR) calculated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation.

Exclusion criteria included: (1) acute kidney injury without underlying CKD; (2) active bleeding or blood transfusion within the previous three months; (3) known hematological malignancies; (4) current chemotherapy or immunosuppressive therapy; (5) pregnancy or postpartum period (<3 months); and (6) recent surgery (<1 month). These criteria were established to minimize confounding factors that could independently affect hemoglobin levels.

Sample size was calculated using the formula $n = Z^2P(1-P)/d^2$, where Z is the statistic for the level of confidence (1.96 for 95% confidence), P is the expected prevalence (estimated at 50% to maximize sample size in the absence of previous reliable data from the region), and d is the precision (set at 5%). This calculation yielded a minimum required sample of 384 participants. Accounting for potential incomplete data, a target enrollment of 450 patients was established.

Data Collection

Standardized data collection forms were used to record demographic information, medical history, current medications, and laboratory parameters. Demographic data included age, sex, residence within Baghdad (urban/suburban), educational level, occupation, and smoking status. Medical history focused

on CKD etiology, duration since diagnosis, comorbidities (particularly diabetes mellitus, hypertension, cardiovascular disease, and autoimmune conditions), and current treatments including ESAs and iron supplementation.

Physical examination findings were recorded, including blood pressure (measured after 5 minutes of rest, with the average of two readings taken 5 minutes apart), height, weight, body mass index (BMI), and clinical signs of anemia. Laboratory data were collected from medical records if performed within the previous 30 days or obtained through standardized blood sampling and analysis.

Laboratory Measurements

Blood samples were collected in the morning after an overnight fast of at least 8 hours. A complete blood count was performed using automated hematology analyzers (Sysmex XN-1000, Sysmex Corporation, Kobe, Japan), with standardized quality control procedures. Kidney function tests, including serum creatinine, blood urea nitrogen, and electrolytes, were measured using automated biochemistry analyzers (Cobas c501, Roche Diagnostics, Basel, Switzerland).

Iron studies included serum iron, total iron-binding capacity (TIBC), transferrin saturation (TSAT), and serum ferritin [11,18]. Inflammatory markers, such as high-sensitivity C-reactive protein (hsCRP) and erythrocyte sedimentation rate (ESR), were measured to assess the inflammatory state [18,26]. Additional parameters included intact parathyroid hormone (iPTH), 25-hydroxyvitamin D, and albumin.

Anemia was defined according to World Health Organization criteria as hemoglobin concentration <13.0 g/dL in men and <12.0 g/dL in women [2]. Severity was classified as mild (women: 11.0-11.9 g/dL; men: 11.0-12.9 g/dL), moderate (8.0-10.9 g/dL), or severe (<8.0 g/dL) [3]. Iron deficiency was defined as TSAT <20% and/or serum ferritin <100 ng/mL for non-dialysis CKD and <200 ng/mL for dialysis patients [11,12].

Statistical Analysis

Data were analyzed using SPSS version 27.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were presented as means ± standard deviations for normally distributed continuous variables, medians with interquartile ranges for non-normally distributed continuous variables, and frequencies with percentages for categorical variables. The Kolmogorov-Smirnov test was used to assess the normality of distribution.

Comparisons across CKD stages were performed using one-way analysis of variance (ANOVA) with post-hoc Bonferroni correction for normally distributed continuous variables, the Kruskal-Wallis test for non-normally distributed continuous variables, and the chi-square or Fisher's exact test for categorical variables, as appropriate. Pearson's or Spearman's correlation coefficients were calculated to examine relationships between hemoglobin levels and continuous variables, depending on data distribution.

Bivariate and multivariate logistic regression analyses were conducted to identify factors independently associated with the presence and severity of anemia. Variables with a p-value of less than 0.10 in the bivariate analysis were included in the multivariate model, along with clinically relevant variables regardless of statistical significance in the bivariate analysis. Results were presented as odds ratios (ORs) with 95% confidence intervals (CIs). A p-value of less than 0.05 was considered statistically significant for all analyses.

Results

Demographic and Clinical Characteristics

A total of 462 CKD patients were enrolled in the study, with males comprising 56.9% (n=263) and females 43.1% (n=199) of the study population. The mean age was 57.8±14.2 years, with the majority of patients (58.4%) aged 50 years or older. Diabetic nephropathy was the most common etiology of CKD (32.5%), followed by hypertensive nephrosclerosis (26.4%), glomerulonephritis (14.3%),

polycystic kidney disease (6.5%), and other/unknown causes (20.3%). Patients were distributed across CKD stages as follows: stage 1 (7.1%, n=33), stage 2 (13.2%, n=61), stage 3 (31.2%, n=144), stage 4 (25.5%, n=118), and stage 5 (23.0%, n=106), with 18.4% (n=85) receiving maintenance hemodialysis. The demographic and clinical characteristics of the study population stratified by CKD stage are presented in Table 1.

Table 1. Demographic and Clinical Characteristics by CKD Stage

Characteristic	Overall (n=462)	Stage 1 (n=33)	Stage 2 (n=61)	Stage 3 (n=144)	Stage 4 (n=118)	Stage 5 (n=106)	p-value
Age (years)	57.8±14.2	49.3±15.7	52.4±14.1	58.2±13.9	59.7±13.2	61.5±13.7	<0.001
Gender, n (%)							0.782
Male	263 (56.9)	20 (60.6)	37 (60.7)	83 (57.6)	65 (55.1)	58 (54.7)	
Female	199 (43.1)	13 (39.4)	24 (39.3)	61 (42.4)	53 (44.9)	48 (45.3)	
BMI (kg/m²)	27.4±5.3	28.9±5.5	28.3±5.1	27.8±5.4	26.9±5.2	26.1±5.0	0.014
CKD etiology, n (%)							0.008
Diabetic nephropathy	150 (32.5)	8 (24.2)	17 (27.9)	42 (29.2)	43 (36.4)	40 (37.7)	
Hypertensive nephrosclerosis	122 (26.4)	7 (21.2)	14 (23.0)	39 (27.1)	34 (28.8)	28 (26.4)	
Glomerulonephritis	66 (14.3)	7 (21.2)	12 (19.7)	22 (15.3)	14 (11.9)	11 (10.4)	
Polycystic kidney disease	30 (6.5)	3 (9.1)	5 (8.2)	10 (6.9)	7 (5.9)	5 (4.7)	
Other/Unknown	94 (20.3)	8 (24.2)	13 (21.3)	31 (21.5)	20 (16.9)	22 (20.8)	
Comorbidities, n (%)							
Diabetes mellitus	176 (38.1)	10 (30.3)	20 (32.8)	53 (36.8)	47 (39.8)	46 (43.4)	0.037
Hypertension	374 (81.0)	21 (63.6)	44 (72.1)	116 (80.6)	101 (85.6)	92 (86.8)	0.006
Cardiovascular disease	138 (29.9)	7 (21.2)	13 (21.3)	41 (28.5)	38 (32.2)	39 (36.8)	0.032
eGFR (ml/min/1.73m²)	35.2±26.3	97.8±11.2	73.5±8.6	45.3±8.7	22.4±4.3	9.7±3.2	<0.001
Dialysis, n (%)	85 (18.4)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	85 (80.2)	<0.001

Values are presented as mean ± standard deviation for continuous variables and number (percentage) for categorical variables. BMI: body mass index; CKD: chronic kidney disease; eGFR: estimated glomerular filtration rate.

Prevalence and Characteristics of Anemia

The overall prevalence of anemia in the study population was 67.5% (n = 312), with a clear trend of increasing prevalence corresponding to advancing CKD stages: 28.6% in stage 1, 43.7% in stage 2, 62.9% in stage 3, 86.3% in stage 4, and 94.7% in stage 5 ($p < 0.001$). The severity of anemia also worsened with CKD progression, with severe anemia (Hb <8.0 g/dL) observed in 1.6% of stage 3, 7.8% of stage 4, and 28.0% of stage 5 patients. No cases of severe anemia were identified in stages 1 and 2. Figure 1 illustrates the prevalence and severity of anemia across CKD stages.

The mean hemoglobin level in the overall study population was 10.7±2.1 g/dL, with significant differences across CKD stages: 13.1±1.6 g/dL in stage 1, 12.4±1.7 g/dL in stage 2, 11.3±1.7 g/dL in stage 3, 10.1±1.5 g/dL in stage 4, and 8.7±1.4 g/dL in stage 5 ($p<0.001$). A strong positive correlation was observed between hemoglobin levels and eGFR ($r=0.693$, $p<0.001$).

Gender differences in anemia prevalence were notable, with females showing higher overall prevalence (74.9%) compared to males (61.9%, $p=0.003$). This gender disparity was particularly pronounced in early CKD stages (stages 1-3) but diminished in advanced disease (stages 4-5), as shown in Table 2.

Table 2. Prevalence of Anemia by Gender and CKD Stage

CKD Stage	Males (n=263)	Females (n=199)	p-value
Stage 1 (n=33)	3/20 (15.0%)	6/13 (46.2%)	0.044
Stage 2 (n=61)	12/37 (32.4%)	15/24 (62.5%)	0.018
Stage 3 (n=144)	44/83 (53.0%)	43/61 (70.5%)	0.032
Stage 4 (n=118)	54/65 (83.1%)	48/53 (90.6%)	0.226
Stage 5 (n=106)	54/58 (93.1%)	46/48 (95.8%)	0.538
Overall (n=462)	**163/263 (61.9%)**	**149/199 (74.9%)**	**0.003**

Values are presented as numbers with anemia/total number (percentage). CKD: chronic kidney disease.

Laboratory Parameters Across CKD Stages

Table 3 presents the laboratory parameters across different CKD stages. Beyond declining hemoglobin levels, advancing CKD was associated with progressive decreases in mean corpuscular volume (MCV) and serum iron levels, along with increases in inflammatory markers (hsCRP, ESR) and intact parathyroid hormone (iPTH). Transferrin saturation showed a decreasing trend with worsening kidney function, while serum ferritin levels increased, particularly in stage 5, reflecting the complex alterations in iron metabolism with progressive CKD.

Table 3. Laboratory Parameters by CKD Stage

Parameter	Stage 1 (n=33)	Stage 2 (n=61)	Stage 3 (n=144)	Stage 4 (n=118)	Stage 5 (n=106)	p-value
Hemoglobin (g/dL)	13.1±1.6	12.4±1.7	11.3±1.7	10.1±1.5	8.7±1.4	<0.001
Hematocrit (%)	39.8±4.6	37.7±5.0	34.5±5.2	30.7±4.7	26.6±4.2	<0.001
MCV (fL)	87.3±6.2	86.9±6.4	86.2±6.6	85.1±7.1	84.2±7.5	0.028
MCH (pg)	29.1±2.3	28.8±2.5	28.5±2.7	28.1±2.9	27.6±3.1	0.012
RDW (%)	14.1±1.5	14.5±1.7	15.3±2.1	16.2±2.3	17.4±2.7	<0.001
WBC (×10³/μL)	7.2±1.9	7.4±2.0	7.5±2.1	7.5±2.2	7.3±2.3	0.856
Platelets (×10³/μL)	256±71	248±76	242±78	233±81	214±87	0.008
Serum iron (μg/dL)	84.2±26.7	78.6±25.2	72.3±24.6	65.1±23.8	58.7±24.2	<0.001
TIBC (μg/dL)	326±52	318±54	310±56	298±59	285±62	<0.001
TSAT (%)	26.1±8.7	24.8±8.2	23.3±8.3	21.9±8.5	20.6±8.8	0.003
Ferritin (ng/mL)	147 (87-267)	162 (92-283)	185 (102-312)	214 (121-347)	378 (214-587)	<0.001
hsCRP (mg/L)	2.7 (1.3-5.2)	3.1 (1.5-6.1)	3.8 (1.9-7.4)	4.7 (2.3-8.2)	6.3 (3.1-11.7)	<0.001
ESR (mm/hr)	21 (12-36)	25 (14-42)	32 (18-52)	41 (24-63)	58 (36-84)	<0.001
Albumin (g/dL)	4.2±0.4	4.1±0.4	3.9±0.5	3.7±0.5	3.4±0.6	<0.001
iPTH (pg/mL)	54 (38-76)	68 (46-94)	97 (65-143)	174 (112-242)	384 (248-563)	<0.001
25-OH Vitamin D (ng/mL)	26.7±9.3	24.2±8.9	21.8±8.5	19.3±8.1	16.5±7.8	<0.001

Values are presented as mean ± standard deviation for normally distributed data and median (interquartile range) for non-normally distributed data. MCV: mean corpuscular volume; MCH: mean corpuscular hemoglobin; RDW: red cell distribution width; WBC: white blood cell count; TIBC: total iron-binding capacity; TSAT: transferrin saturation; hsCRP: high-sensitivity C-reactive protein; ESR: erythrocyte sedimentation rate; iPTH: intact parathyroid hormone; 25-OH Vitamin D: 25-hydroxyvitamin D.

Anemia Treatment Patterns

Among anemic patients (n = 312), 52.6% (n = 164) were receiving treatment with erythropoiesis-stimulating agents (ESAs), with treatment rates varying significantly across CKD stages: 0.0% in stage 1, 8.3% in stage 2, 32.2% in stage 3, 64.7% in stage 4, and 76.0% in stage 5 ($p < 0.001$). The most

commonly used ESA was epoetin alfa (73.2%), followed by darbepoetin alfa (18.9%) and methoxy polyethylene glycol-epoetin beta (7.9%).

Iron supplementation was prescribed to 59.9% (n = 187) of anemic patients, with oral iron being more common (68.4%) than intravenous iron (31.6%). Notably, only 47.8% (n = 149) of anemic patients were receiving combined erythropoiesis-stimulating agent (ESA) and iron therapy, despite clinical practice guidelines recommending this approach. Treatment patterns in anemic patients across chronic kidney disease (CKD) stages are summarized in Table 4.

Table 4. Treatment Patterns in Anemic Patients by CKD Stage

Treatment	Stage 1 (n=9)	Stage 2 (n=24)	Stage 3 (n=87)	Stage 4 (n=102)	Stage 5 (n=90)	p-value
ESA, n (%)	0 (0.0)	2 (8.3)	28 (32.2)	66 (64.7)	68 (76.0)	<0.001
Iron supplementation, n (%)	4 (44.4)	11 (45.8)	47 (54.0)	65 (63.7)	60 (66.7)	0.007
Oral iron	4 (100.0)	11 (100.0)	37 (78.7)	45 (69.2)	31 (51.7)	<0.001
Intravenous iron	0 (0.0)	0 (0.0)	10 (21.3)	20 (30.8)	29 (48.3)	<0.001
Combined ESA and iron, n (%)	0 (0.0)	2 (8.3)	24 (27.6)	61 (59.8)	62 (68.9)	<0.001
Blood transfusion (past 6 months), n (%)	0 (0.0)	1 (4.2)	9 (10.3)	18 (17.6)	31 (34.4)	<0.001
No anemia treatment, n (%)	5 (55.6)	12 (50.0)	36 (41.4)	23 (22.5)	12 (13.3)	<0.001

ESA: erythropoiesis-stimulating agent; CKD: chronic kidney disease.

Factors Associated with Anemia in CKD

Bivariate analysis identified multiple factors associated with the presence of anemia in CKD patients. These included older age, female gender, longer CKD duration, diabetic nephropathy as etiology, presence of diabetes mellitus, cardiovascular disease, decreased eGFR, elevated inflammatory markers (hsCRP, ESR), low transferrin saturation, elevated ferritin, elevated iPTH, and low 25-hydroxyvitamin D levels.

In multivariate logistic regression analysis, four factors remained independently associated with anemia after adjusting for potential confounders: decreased eGFR (OR 1.06 per 1 mL/min/1.73 m^2 decrease, 95% CI 1.04-1.08, $p < 0.001$), female gender (OR 1.83, 95% CI 1.14-2.94, $p = 0.012$), diabetes mellitus (OR 1.74, 95% CI 1.08-2.82, $p = 0.024$), and transferrin saturation <20% (OR 2.36, 95% CI 1.45-3.84, $p < 0.001$). The results of multivariate analysis are presented in Table 5.

Table 5. Multivariate Logistic Regression Analysis of Factors Associated with Anemia in CKD

Variable	Adjusted OR	95% CI	p-value
Age (per 10-year increase)	1.14	0.97-1.35	0.118
Female gender	1.83	1.14-2.94	0.012
CKD duration (per year)	1.05	0.98-1.12	0.157
Diabetic nephropathy	1.32	0.78-2.23	0.304
Diabetes mellitus	1.74	1.08-2.82	0.024
Cardiovascular disease	1.25	0.78-2.00	0.354
eGFR (per 1 mL/min/1.73m^2 decrease)	1.06	1.04-1.08	<0.001
hsCRP >5 mg/L	1.42	0.89-2.27	0.146
Transferrin saturation <20%	2.36	1.45-3.84	<0.001
Ferritin >500 ng/mL	1.36	0.82-2.25	0.228
iPTH >300 pg/mL	1.31	0.77-2.22	0.318
25-OH Vitamin D <20 ng/mL	1.28	0.81-2.03	0.294

OR: odds ratio; CI: confidence interval; CKD: chronic kidney disease; eGFR: estimated glomerular filtration rate; hsCRP: high-sensitivity C-reactive protein; iPTH: intact parathyroid hormone; 25-OH Vitamin D: 25-hydroxyvitamin D.

Subgroup analysis by gender revealed differences in the factors associated with anemia. In males, decreased eGFR (OR 1.05, 95% CI 1.03-1.07, $p < 0.001$), diabetes mellitus (OR 1.96, 95% CI 1.06-3.62, $p = 0.032$), and transferrin saturation <20% (OR 2.51, 95% CI 1.34-4.68, $p = 0.004$) were independently associated with anemia. In females, decreased eGFR (OR 1.07, 95% CI 1.04-1.10, $p < 0.001$), transferrin saturation <20% (OR 2.19, 95% CI 1.02-4.71, $p = 0.045$), and elevated iPTH (>300 pg/mL) (OR 1.87, 95% CI 1.01-3.46, $p = 0.047$) emerged as significant predictors of anemia.

Discussion

This cross-sectional study provides the first comprehensive assessment of anemia prevalence across all stages of chronic kidney disease (CKD) in Baghdad's adult population. Our findings reveal a strikingly high overall prevalence of 67.5%, with rates progressively increasing from 28.6% in stage 1 to 94.7% in stage 5 CKD. These prevalence figures exceed those reported in several international studies, including the 15.4% prevalence in stage 1 and 54.3% in stage 5 reported by the US National Health and Nutrition Examination Survey (NHANES) [8], and the 22.0% (stage 1) to 75.5% (stage 5) prevalence documented in the Chronic Renal Insufficiency Cohort (CRIC) study [9]. Our findings align more closely with studies from other developing regions, such as the 73.8% overall prevalence reported in a Nigerian cohort [14] and the 71.2% observed in a South African study [15].

Bin levels ($r = 0.693$) in our cohort reinforce this relationship and underscore the utility of hemoglobin as a potential marker of CKD progression.

The gender disparity in anemia prevalence observed in our study, with significantly higher rates in females (74.9%) compared to males (61.9%), is consistent with findings from other populations. This difference was particularly pronounced in early CKD stages but diminished with advancing disease, suggesting that female gender may lower the threshold for anemia development in early CKD. At the same time, severe kidney dysfunction eventually overwhelms this gender effect. The higher susceptibility of females to anemia may reflect baseline differences in iron stores, menstrual blood loss in premenopausal women, and potentially different erythropoietin responses to declining kidney function [27,28].

Multivariate analysis identified four independent predictors of anemia in our cohort: decreased eGFR, female gender, diabetes mellitus, and transferrin saturation <20%. The association with reduced eGFR is expected and represents the primary pathophysiological driver of CKD-associated anemia. The identification of diabetes as an independent risk factor adds to growing evidence suggesting that diabetic patients may have reduced erythropoietin production or responsiveness beyond that explained by their degree of kidney dysfunction. This diabetes-associated anemia risk may reflect microvascular complications affecting peritubular fibroblasts, tubulointerstitial inflammation, or autonomic neuropathy influencing erythropoietin regulation [30].

Low transferrin saturation (<20%) emerging as a strong, independent predictor of anemia (OR 2.36) highlights the critical role of iron deficiency in our population. This finding is particularly relevant in the Baghdad context, where dietary iron intake may be suboptimal due to economic constraints limiting access to iron-rich foods, and where gastrointestinal blood loss may be more common due to higher prevalence of parasitic infections and peptic ulcer disease. The contrast between low transferrin saturation and elevated ferritin levels, particularly in advanced chronic kidney disease (CKD), reflects the complex iron metabolism disturbances characterized by functional iron deficiency despite adequate iron stores, predominantly mediated by inflammation-induced hepcidin elevation.

Our analysis of anemia treatment patterns reveals concerning gaps in management practices. Despite strong guideline recommendations for combined ESA and iron therapy in CKD-associated anemia, only 47.8% of anemic patients in our cohort were receiving this combination. Particularly notable was the undertreatment in early CKD stages, with no ESA use in stage 1 and only 8.3% in stage 2 anemic

patients. While this partly reflects appropriate clinical judgment in milder anemia, it may also indicate missed opportunities for early intervention, especially considering that 50% of stage 2 anemic patients were receiving no anemia treatment whatsoever.

The preference for oral over intravenous iron (68.4% vs. 31.6%) in our setting diverges from practices in more resourceful environments, where intravenous iron is increasingly favored, particularly in advanced chronic kidney disease (CKD). This likely reflects limited availability and higher costs of intravenous preparations in Baghdad, combined with clinicians' concerns regarding administration facilities and potential adverse reactions. The increasing use of intravenous iron with advancing CKD stages (from 0% in stages 1-2 to 48.3% in stage 5) demonstrates appropriate prioritization of limited resources toward patients with more severe disease and lower gastrointestinal iron absorption.

The high rate of blood transfusions in stage 5 patients (34.4% within the previous six months) is concerning, given the potential complications, including allosensitization, which may compromise future kidney transplantation prospects. This likely reflects inadequate anemia management with ESAs and iron, possibly due to medication access limitations, cost constraints, or ESA hyporesponsiveness in the setting of high inflammation.

Laboratory parameters across CKD stages revealed patterns consistent with multiple contributing mechanisms to anemia. The progressive decline in MCV and MCH with advancing CKD suggests a shift toward microcytic, hypochromic anemia features, potentially reflecting iron deficiency or functional iron deficiency components. The steady increase in RDW with CKD progression indicates increasing red cell size heterogeneity, consistent with multiple concurrent factors affecting erythropoiesis. Inflammatory markers (hsCRP, ESR) and iPTH exhibited significant increases with advancing CKD stages, highlighting their potential contributory roles in anemia pathogenesis through mechanisms including hepcidin elevation, bone marrow fibrosis, and direct suppression of erythropoiesis.

Our study has several strengths, including its multicenter design encompassing three major nephrology centers serving diverse populations within Baghdad, the inclusion of patients across all CKD stages, comprehensive laboratory assessments including iron parameters and inflammatory markers, and multivariate analysis adjusting for potential confounders. To our knowledge, this represents the first comprehensive study of anemia across the spectrum of CKD stages in Baghdad's adult population, providing valuable epidemiological data for clinical practice and health policy.

However, certain limitations warrant consideration. The cross-sectional design precludes the establishment of causal relationships or temporal sequences. Our sampling from tertiary nephrology centers may overrepresent more severe disease, limiting generalizability to the broader chronic kidney disease (CKD) population, particularly those managed in primary care or those who remain undiagnosed in the community. Although we adjusted for multiple potential confounders, residual confounding from unmeasured variables cannot be excluded. Additionally, a single measurement of hemoglobin and iron parameters may not fully capture the dynamic nature of these values, particularly in patients with fluctuating inflammatory states or recent medication adjustments.

Several clinical and research implications emerge from our findings. The high anemia prevalence, particularly in early CKD stages, compared to international cohorts, suggests that routine screening should be implemented earlier in the disease course for Baghdad's CKD population. The identification of diabetes as an independent risk factor highlights the need for particularly vigilant screening in diabetic CKD patients. The suboptimal treatment patterns observed, especially regarding combined ESA and iron therapy, indicate the need for provider education and system-level interventions to align clinical practice with evidence-based guidelines.

From a health policy perspective, our findings underscore the need for improved access to anemia treatments, particularly intravenous iron preparations and consistent ESA availability. The high transfusion rates in advanced CKD suggest potential cost-effectiveness of more aggressive anemia management to reduce transfusion requirements and their associated complications. Furthermore, the

gender disparities observed highlight the importance of gender-sensitive approaches to anemia screening and management.

Conclusion

This cross-sectional study reveals a high prevalence of anemia among CKD patients in Baghdad, exceeding rates reported in many international cohorts, with a clear progression corresponding to declining kidney function. Independent predictors include decreased eGFR, female gender, diabetes mellitus, and low transferrin saturation, highlighting both the primary pathophysiological mechanisms and population-specific risk factors. The suboptimal treatment patterns observed, particularly regarding combined ESA and iron therapy, suggest opportunities for improved management aligned with evidence-based guidelines.

These findings emphasize the need for routine anemia screening in all CKD patients in Baghdad, with particular attention to high-risk groups including females, diabetics, and those with iron deficiency. From a health policy perspective, ensuring consistent availability of anemia treatments, particularly intravenous iron preparations and ESAs, should be prioritized to reduce transfusion requirements and potentially improve outcomes in this vulnerable population.

Future research should focus on longitudinal evaluation of anemia progression and outcomes in this setting, interventional studies comparing management strategies, and investigation of potential genetic factors contributing to the higher prevalence observed. By addressing the challenges identified in this study, clinicians and health policymakers can work toward reducing the burden of anemia in Baghdad's chronic kidney disease (CKD) population, potentially improving quality of life and clinical outcomes for these patients.

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