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Frequency of Absolute and Functional Iron Deficiency Anemia in Non-Dialysis Dependent Chronic Kidney Disease Patients

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ABSTRACT

Background and Aim: Iron deficiency is a common and clinically significant complication in non-dialysis dependent chronic kidney disease (CKD) patients, manifesting as either absolute or functional iron deficiency. This study aimed to assess the frequency and distribution of these subtypes of iron deficiency anemia (IDA) across different CKD stages and to identify their independent predictors. **Materials and Methods:** A cross-sectional study was conducted at the Nephrology Outpatient Department of Nishtar Hospital, Multan over a period of 6 months from July 2024 to December 2024, enrolling 451 adult patients with CKD stages 3a to 5 (non-dialysis dependent). Hemoglobin, serum ferritin, transferrin saturation, and serum iron were evaluated to classify iron deficiency. **Results:** Anemia was observed in 346 patients (76.7%). Of these, 133 (29.5%) had absolute iron deficiency (AID), and 213 (47.2%) had functional iron deficiency (FID). Anemia prevalence increased significantly with advancing CKD stage ($p = 0.002$). AID was highest in stage 5 (49.1%), while FID peaked in stage 4 (58.2%) ($p < 0.05$ for both). Hemoglobin was significantly lower in AID (9.2 ± 1.1 g/dL) and FID (9.7 ± 1.3 g/dL) groups versus non-anemic patients (12.9 ± 0.8 g/dL; $p < 0.001$). Serum ferritin was lowest in AID (51.2 ± 15.7 ng/mL) and highest in FID (173.6 ± 42.5 ng/mL). Transferrin saturation and serum iron levels were significantly reduced in both deficiency groups ($p < 0.001$). Multivariate analysis revealed age <60 years (aOR: 1.79; $p = 0.027$) and CKD stage 5 (aOR: 2.23; $p = 0.024$) as independent predictors of AID, while CKD stage 4 was significantly associated with FID (aOR: 2.93; $p < 0.001$). **Conclusion:** Anemia, particularly functional iron deficiency, is prevalent in non-dialysis dependent CKD patients. Disease stage and age significantly influence the type of iron deficiency, highlighting the need for stage-specific evaluation and management.

INTRODUCTION

Chronic Kidney Disease (CKD) represents a significant public health burden, with current estimates suggesting that it affects roughly 10–15% of the adult population globally. Among its numerous systemic complications, anemia remains one of the most frequently encountered and clinically impactful, particularly as renal function progressively deteriorates. The underlying pathophysiology contributing to anemia in CKD are multifactorial; however, iron deficiency stands out as a predominant and modifiable factor. Iron deficiency in this population exists in two principal forms: absolute iron deficiency, characterized by depleted iron stores, and functional iron deficiency, in which iron stores are preserved or elevated but are inadequately mobilized for erythropoiesis due to impaired iron utilization or inflammation [1,2]. Absolute iron deficiency is characterized by depleted iron stores, typically assessed by low serum ferritin levels. Functional iron deficiency, on the other hand, occurs when iron stores are adequate but there is an impaired release of iron from storage sites, often indicated by low transferrin saturation (TSAT) despite normal or elevated ferritin levels [3,4]. Accurate differentiation between absolute iron deficiency (AID) and functional iron deficiency (FID) is essential for guiding appropriate therapy, as the underlying mechanisms and responses to treatment may vary. Patients with AID typically benefit from oral or intravenous iron supplementation alone, whereas those with FID may require additional therapeutic strategies, such as erythropoiesis-stimulating agents or measures to control systemic inflammation [5–7].

The prevalence of absolute and functional iron deficiency among non-dialysis CKD patients has shown considerable variation across published literature. For example, Awan et al. (2021) reported functional iron deficiency in approximately 19% of anemic non-dialysis CKD patients, based on a TSAT ≤20% and serum ferritin

between 100–500 ng/mL [6]. Similarly, Gonget al. (2023), analyzing data from over 4,000 individuals, found FID in about 20% of cases. In contrast, our study observed a notably higher frequency of FID at 47.2%, suggesting possible regional differences or varying exposure to iron therapies and inflammatory burdens [8].

Although recognition of AID and FID in non-dialysis CKD is important for anemia management, there remains a lack of large-scale data examining their distribution across different populations. Clarifying the prevalence of these subtypes of iron deficiency is important to enhance early detection and inform treatment protocols. The present study was undertaken to systematically evaluate the frequency of both AID and FID in patients with non-dialysis CKD and to explore clinical and demographic factors associated with each form of iron deficiency.

MATERIAL AND METHOD

This was a cross-sectional study. It was conducted at the Outpatients department of Nephrology, Nishtar Hospital Multan over a period of 6 months (July 2024 to December 2024). After obtaining ethical approval from the institutional review board of Nishtar Hospital Multan (18965/NMU), 451 non-dialysis dependent chronic kidney disease patients were recruited using non-probability consecutive sampling technique. The sample size was calculated assuming a prevalence of 25% for functional iron deficiency, with a confidence level of 95% and a margin of error of 4% [9]. The study included adults aged 18 years and above who were diagnosed with CKD stages 3-5 and were not currently receiving dialysis treatment. Patients were excluded if they were on dialysis, had active bleeding or blood transfusion within the past 3 months, had active malignancy, were pregnant, or had received erythropoiesis-stimulating agents or intravenous iron therapy in the past 3 months.

Following informed consent, baseline demographic and clinical data were collected from each participant, including age, gender, medical history, duration of CKD, and current medications. Each participant underwent a thorough clinical examination to assess their general health status and identify any signs of anemia or CKD complications. Information about comorbid conditions such as hypertension, diabetes, and cardiovascular disease was gathered through patient interviews and review of medical records. Laboratory assessments were performed to determine hemoglobin levels, serum ferritin, and transferrin saturation using standardized methods. Blood samples were collected and analyzed to measure these parameters for proper classification of iron deficiency status.

Chronic kidney disease in this study was characterized by persistent structural or functional kidney abnormalities lasting longer than three months. Staging was determined using the CKD-EPI formula for estimated glomerular filtration rate, categorizing patients into stage 3a (45–59 ml/min/1.73m^2), stage 3b (30–44 ml/min/1.73m^2), stage 4 (15–29 ml/min/1.73m^2), and stage 5 (less than 15 ml/min/1.73m^2), excluding those on dialysis. Anemia was diagnosed according to World Health Organization standards, defined as hemoglobin below 13 g/dL in males and below 12 g/dL in females. Absolute iron deficiency was identified when serum ferritin was under 100 ng/mL, whereas functional iron deficiency was defined by a transferrin saturation below 20% despite ferritin levels above 100 ng/mL. Based on these definitions, participants were stratified into three categories: those with absolute iron deficiency, those with functional iron deficiency, and those without iron deficiency anemia.

All study data were documented using structured forms and analyzed with SPSS version 26. Continuous variables were summarized using means and standard deviations, while categorical variables were reported as frequencies and percentages. Comparative analysis was conducted using the Chi-square test for categorical variables and one-way ANOVA for continuous variables. Multivariate logistic regression was used to identify independent predictors of absolute and functional iron deficiency, with results presented as adjusted odds ratios and 95% confidence intervals. A p-value less than 0.05 was considered statistically significant for all analyses.

RESULTS

A total of 451 patients with chronic kidney disease (CKD) stages 3a to 5 (non-dialysis dependent) were included. The mean age was 56.2 ± 13.1 years. There were 245 males (54.3%) and 206 females (45.7%). The median duration of CKD was 39 months (interquartile range: 21–66). Stage 4 was the most common CKD stage, observed in 189 patients (41.9%), followed by stage 3b in 123 (27.3%), stage 3a in 82 (18.2%), and stage 5 in 57 (12.6%). Hypertension was present in 323 patients (71.6%) and diabetes mellitus in 287 (63.6%). The distribution of CKD stages and other baseline characteristics is summarized in Table 1.

Table 1

Baseline Characteristics of the Study Population (n = 451)

Variable	n (%) or Mean ± SD
Age (years)	56.2 ± 13.1
Gender	
Male	245 (54.3%)
Female	206 (45.7%)
CKD Stage	
Stage 3a	82 (18.2%)
Stage 3b	123 (27.3%)
Stage 4	189 (41.9%)
Stage 5 (non-dialysis)	57 (12.6%)
Duration of CKD (months)	Median: 39 (IQR: 21–66)
Diabetes Mellitus	287 (63.6%)
Hypertension	323 (71.6%)

Among the 451 patients with non-dialysis dependent chronic kidney disease, anemia was observed in 346 (76.7%). Based on operational definitions, all anemic individuals were classified as having either absolute iron deficiency (AID) or functional iron deficiency (FID). AID was present in 133 (29.5%), while FID was identified in 213 (47.2%). The distribution of anemia and its subtypes across CKD stages is shown in Table 2. Anemia prevalence increased with disease severity, from 51 (62.2%) in stage 3a to 50 (87.7%) in stage 5 (p = 0.002). AID was most frequent in stage 5, seen in 28 (49.1%) patients, followed by 36 (29.3%) in stage 3b, 21 (25.6%) in stage 3a, and 48 (25.4%) in stage 4 (p = 0.014). FID was most common in stage 4, affecting 110 (58.2%) patients, followed by 60 (48.8%) in stage 3b, 30 (36.6%) in stage 3a, and 22 (38.6%) in stage 5 (p = 0.003).

Table 2

Distribution of Anemia, Absolute Iron Deficiency, and Functional Iron Deficiency by CKD Stage (n = 451)

Type of Anemia	Stage 3a (n=82)	Stage 3b (n=123)	Stage 4 (n=189)	Stage 5 (n=57)	p-value
Anemia (n=346)	51 (62.2%)	96 (78.0%)	158 (83.6%)	50 (87.7%)	0.002
AID (n=133)	21 (25.6%)	36 (29.3%)	48 (25.4%)	28 (49.1%)	0.014
FID (n=213)	30 (36.6%)	60 (48.8%)	110 (58.2%)	22 (38.6%)	0.003

The mean hemoglobin levels were significantly lower in patients with absolute iron deficiency (9.2 ± 1.1 g/dL) and functional iron deficiency (9.7 ± 1.3 g/dL) compared to non-anemic individuals (12.9 ± 0.8 g/dL; $p < 0.001$). Serum ferritin was lowest in the AID group (51.2 ± 15.7 ng/mL), markedly elevated in FID (173.6 ± 42.5 ng/mL), and intermediate in non-anemic patients (136.3 ± 34.8 ng/mL; $p < 0.001$). Transferrin saturation was reduced in both AID (17.1 ± 2.3%) and FID (14.8 ± 3.0%) compared to non-anemic patients (26.8 ± 4.6%; $p < 0.001$). Similarly, serum iron levels were lower in AID (34.7 ± 8.2 µg/dL) and FID (39.4 ± 10.4 µg/dL) than in non-anemic individuals (67.3 ± 12.8 µg/dL; $p < 0.001$). Total iron-binding capacity was highest in the AID group (403.2 ± 36.1 µg/dL), followed by FID (348.7 ± 28.9 µg/dL), and lowest in the non-anemic group (304.1 ± 26.2 µg/dL; $p < 0.001$) (Table 3).

Table 3

Comparison of Laboratory Parameters by Iron Status Group

Parameter	AID (n = 133)	FID (n = 213)	Non-Anemic (n = 105)	p-value
Hemoglobin (g/dL)	9.2 ± 1.1	9.7 ± 1.3	12.9 ± 0.8	< 0.001
Serum Ferritin (ng/mL)	51.2 ± 15.7	173.6 ± 42.5	136.3 ± 34.8	< 0.001
Transferrin Saturation (%)	17.1 ± 2.3	14.8 ± 3.0	26.8 ± 4.6	< 0.001
Serum Iron (µg/dL)	34.7 ± 8.2	39.4 ± 10.4	67.3 ± 12.8	< 0.001
Total Iron-Binding Capacity (µg/dL)	403.2 ± 36.1	348.7 ± 28.9	304.1 ± 26.2	< 0.001

Multivariate logistic regression analysis identified that age <60 years was independently associated with absolute iron deficiency anemia (aOR: 1.79; 95% CI: 1.07–2.98; $p = 0.027$), while CKD stage 5 also showed a significant association (aOR: 2.23; 95% CI: 1.11–4.49; $p = 0.024$). In contrast, CKD stage 4 was strongly associated with functional iron deficiency anemia (aOR: 2.93; 95% CI: 1.59–5.39; $p < 0.001$). Female gender, diabetes mellitus, and hypertension did not show statistically significant associations with either AID or FID after adjustment.

Table 4

Multivariate Logistic Regression Analysis for Predictors of AID and FID Among Anemic CKD Patients (n = 346).

Variable	AID (aOR, 95% CI)	p-value	FID (aOR, 95% CI)	p-value
Age < 60 years	1.79 (1.07–2.98)	0.027	1.16 (0.73–1.83)	0.528
Female gender	1.14 (0.70–1.86)	0.592	1.03 (0.66–1.60)	0.898
CKD Stage 4	1.31 (0.76–2.28)	0.328	2.93 (1.59–5.39)	<0.001
CKD Stage 5	2.23 (1.11–4.49)	0.024	1.52 (0.75–3.07)	0.245
Diabetes mellitus	1.27 (0.76–2.14)	0.357	1.08 (0.68–1.72)	0.743
Hypertension	1.10 (0.65–1.88)	0.723	1.39 (0.84–2.30)	0.206

DISCUSSION

In the present study, anemia was identified in 346 out of 451 non-dialysis dependent chronic kidney disease (CKD) patients, yielding a prevalence of 76.7%. This figure is substantially higher than rates reported in population-based studies from high-income regions, where anemia affects approximately 15–40% of stage 3–5 CKD patients depending on the cohort and definition used. For instance, large-scale U.S. and European studies have documented anemia prevalence ranging from 23% to 40%, with stage-specific rates rising from around 18% in stage 3a to 73% in stage 5, and only 2% meeting criteria for severe anemia requiring erythropoiesis-stimulating agents [10–12]. The NHANES data reported overall anemia prevalence in CKD as 15.4%, increasing steadily with advancing stage [7]. In contrast, our findings are closely aligned with data from regional South Asian studies. A recent multicenter investigation in Pakistan documented an anemia prevalence of 78.7% among non-dialysis CKD patients, while another study reported iron deficiency anemia in 38.8% of 188 pre-dialysis CKD patients [5,9]. These elevated rates in low-resource settings likely reflect limited access to timely anemia screening, nutritional deficiencies, and underutilization of iron and erythropoietin therapies. Indeed, oral iron therapy was administered to only 6.2% of stage 3 and 25% of stage 4 CKD patients in local audits, with fewer than half achieving hemoglobin targets over six months [9]. In India, stage-wise trends mirror our results, with anemia present in 39% at stage 1 and virtually all patients by stages 4–5 [13]. These findings underscore the critical need for early detection and structured anemia management, particularly in resource-constrained populations where advanced CKD stages dominate clinical presentation.

In the present study, absolute iron deficiency anemia (AIDA), defined by serum ferritin <100 ng/mL with low hemoglobin, was identified in 133 patients, representing 29.5% of the total CKD population and 38.4% among those with anemia. This frequency is consistent with several previous studies. A U.S. Veterans Affairs-based analysis reported absolute iron deficiency in 30% of anemic non-dialysis CKD patients [7]. Similarly, a hospital-based study from Pakistan reported iron deficiency anemia in 38.8% of pre-dialysis CKD patients, closely aligning with the current findings [5]. A European outpatient cohort of over 3,000 CKD patients showed a 41.7% prevalence of absolute iron deficiency, indicating a comparable burden in developed healthcare systems [14]. Notably, higher rates were observed in geriatric CKD populations (56.2%) and in children with CKD (38%), suggesting that age-related factors may influence iron depletion [15]. In contrast, studies from other regions have reported lower prevalence, such as 12% in a Middle Eastern cohort [16]. These variations likely reflect differences in dietary iron intake, CKD stage distribution, comorbid burden, and healthcare access. Collectively, the evidence reinforces that

absolute iron deficiency is a prevalent and under-recognized contributor to anemia in CKD [17].

In the present study, functional iron deficiency anemia (FID), defined as transferrin saturation <20% with serum ferritin >100 ng/mL, was observed in 47.2% of the total CKD population and accounted for 61.6% of all anemic patients. This prevalence is significantly higher than rates reported in most international studies. For instance, a large U.S. cohort identified FID in only 19% of non-dialysis CKD patients with anemia using similar criteria [6]. Comparable findings were noted in NHANES III data, which reported FID in 19.8% of anemic CKD patients [9]. Pediatric and geriatric studies have shown variable rates, with FID observed in 25% of children and 43.8% of elderly CKD patients with iron deficiency [18,19]. The disproportionately high FID frequency in this study may reflect chronic inflammation, poor access to iron therapy, and delayed or incomplete correction of absolute deficiency. Exclusion of recent iron or erythropoiesis-stimulating agent use likely amplified unaddressed functional deficits. Additionally, adherence to KDIGO-based thresholds (ferritin >100 ng/mL) may have classified borderline ferritin levels as FID, contributing to elevated prevalence estimates compared to studies using higher ferritin cutoffs. This highlights the impact of both population characteristics and diagnostic definitions on FID rates [20].

The present study highlights the stage-dependent variation in both prevalence and type of iron deficiency among non-dialysis chronic kidney disease (CKD) patients. Anemia was significantly more common in advanced CKD, increasing from 62.2% in stage 3a to 87.7% in stage 5 ($p = 0.002$), in agreement with previous findings that anemia severity escalates with declining kidney function. Notably, the type of iron deficiency shifted with disease progression. Absolute iron deficiency anemia (AID) was most frequent in stage 5 (49.1%), compared to 25–29% in earlier stages ($p = 0.014$), whereas functional iron deficiency (FID) peaked in stage 4 (58.2%) and then declined in stage 5 (38.6%) ($p = 0.003$). These findings suggest that iron sequestration dominates in mid-stage CKD due to inflammation and hepcidin elevation, but with prolonged disease, iron stores become depleted, leading to a rise in AID. Similar stage-wise trends were noted by Aoun et al., who observed that male CKD patients had declining ferritin with advancing stage, while in females, inflammation masked store depletion [21]. A pediatric study also demonstrated functional iron restriction despite elevated ferritin, which may evolve into absolute deficiency without timely intervention [13]. Laboratory values further validated these trends, with mean ferritin significantly lower in AID (51.2 ± 15.7 ng/mL) and TSAT lower in FID (14.8 ± 3.0%), consistent with functional iron blockade. These patterns reinforce that iron deficiency in CKD is dynamic and stage-specific, warranting individualized monitoring and timely therapy to address both functional and absolute deficits.

Multivariate analysis identified younger age (<60 years) as an independent predictor of absolute iron deficiency anemia (AID) (aOR 1.79, $p = 0.027$), consistent with previous findings that younger individuals are more prone to AID due to factors like menstrual loss and lower dietary iron intake [18]. Although females had higher unadjusted rates of AID, gender was not a significant predictor after adjustment, likely reflecting post-menopausal status in many participants. CKD stage showed a strong association: stage 5 was linked with increased odds of AID (aOR 2.23, $p = 0.024$), while stage 4 had the highest odds of functional iron deficiency (FID) (aOR 2.93, $p < 0.001$), supporting previous studies showing stage-wise shifts in iron deficiency types [22,23]. Comorbidities like diabetes and hypertension were not significant predictors after adjustment ($p > 0.20$), suggesting that CKD severity primarily drives iron deficiency, aligning with earlier research [6]. This emphasizes that reduced glomerular filtration rate and inflammation-related iron sequestration dominate the pathogenesis of anemia in CKD regardless of underlying comorbidities. These findings reinforce the need for stage- and age-specific assessment strategies to optimize anemia management.

This study underscores the importance of distinguishing between absolute and functional iron deficiency in non-dialysis dependent CKD patients, as their management differs significantly. Timely identification can aid in guiding appropriate interventions to prevent worsening anemia and its complications. The findings support routine assessment of iron indices in CKD care protocols. However, the study's cross-sectional nature limits causal inference, and being conducted at a single tertiary center may affect generalizability. Additionally, exclusion of patients on recent iron or ESA therapy may have led to underrepresentation of managed cases. Despite these constraints, the results provide a valuable contribution to regional clinical practice.

CONCLUSION

The study demonstrates that iron deficiency anemia is highly prevalent among non-dialysis chronic kidney disease patients, with a distinct pattern based on disease stage. Functional iron deficiency was more frequent in stage 4, while absolute iron deficiency predominated in stage 5. Younger age and advanced CKD stage were key predictors of absolute iron deficiency. These findings highlight the need for stage-specific assessment and timely classification of iron deficiency type to guide appropriate and individualized treatment in CKD patients.

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