



ORIGINAL ARTICLE

Effect of a 4-month dietary intervention on anemia in patients with chronic kidney disease stages I-III: a retrospective study in 29 Algerian patients

Ghalia KHELLAF, Ahmed KOCEIR EL HADJ, Mohamed BENABADJI

ABSTRACT

Background: Anemia is a common complication of chronic kidney disease (CKD), even in its early stages. Appropriate nutritional management may improve hemoglobin levels and reduce the need for erythropoiesis-stimulating agents (ESAs). This study assessed the impact of a 4-month standardized dietary intervention on anemia-related parameters in Algerian patients with CKD stages I-III. **Methods:** This retrospective study included 29 patients (72% women; mean age, 63 ± 15 years) with KDIGO stage I-III CKD and anemia (hemoglobin <13 g/dL in men and <12 g/dL in women). Biological parameters, including hemoglobin, mean corpuscular volume, serum ferritin, transferrin saturation, creatinine, urea, uric acid, electrolytes, vitamin B12, and folate, were assessed before and after a 4-month dietary intervention. The intervention consisted of individualized nutritional counseling supported by a personalized educational brochure recommending a protein intake of 0.6–0.8 g/kg/day, an energy intake of 30–35 kcal/kg/day, controlled potassium, phosphorus, and sodium intake, and increased consumption of heme iron and B vitamins. Paired t-tests and linear regression analyses were performed. **Results:** At baseline, 55% of patients had normocytic, normochromic anemia. Iron deficiency (serum ferritin <100 ng/mL) was present in 48% of patients, while 24% had low transferrin saturation (<20%). Vitamin B12 deficiency was identified in 34.5% of patients. Following the 4-month intervention, mean hemoglobin increased significantly from 10.9 ± 1.2 to 12.1 ± 0.9 g/dL ($p < 0.001$). Serum ferritin increased from 48.2 ± 32.1 to 72.5 ± 28.4 ng/mL ($p = 0.03$), and transferrin saturation improved from 18.3 ± 5.2% to 23.6 ± 4.8% ($p = 0.02$). The prevalence of microcytic anemia decreased from 24% to 10%. No significant correlation was observed between creatinine clearance and hemoglobin concentration ($r = 0.053$, $p = 0.79$). **Conclusion:** A 4-month targeted nutritional intervention significantly improved anemia-related parameters in patients with early-stage CKD, primarily through correction of iron and vitamin B12 deficiencies. Larger prospective studies are warranted to confirm the long-term benefits of this approach.

Keywords: chronic kidney disease, anemia, nutritional intervention, iron deficiency, Algeria.

1. University Health of Sciences El Moudjahid Youcef El Khatib, Faculty of Medicine, University of Algiers, Algiers, Algeria, Nephrology Department, Bab El Oued University Hospital Center, Algiers, Algeria, g.khellaf@yahoo.fr, Algérie. 2. Faculty of Biological Sciences, University of Science and Technology Houari Boumediene, Algiers, Algeria, e.koceir@gmail.com, Algérie. 3. University Health of Sciences El Moudjahid Youcef El Khatib, Faculty of Medicine, Algiers, Algeria, Nephrology Department, Beni Messous University Hospital Center, Algiers, Algeria, benabadji_cdr@yahoo.fr, Algérie.

Received: 12 Apr 2026

Accepted: 25 Jun 2026

Correspondance to: Ghalia KHELLAF
E-mail: g.khellaf@yahoo.fr

1. INTRODUCTION

Chronic kidney disease (CKD) has become a major public health challenge worldwide. According to the Global Burden of Disease Study 2023, CKD now affects one in seven adults globally, making it the ninth leading cause of death with nearly 1.5 million deaths annually (1). The Middle East and North Africa region, including Algeria, is among the most affected areas, with nearly one in five adults affected (The National, 2025). In Algeria, the crude incidence of stage 5 CKD treated in 2017 was 75 per million inhabitants, with diabetes (26.7%) and hypertensive nephropathy (22.6%) representing the two main causes (2).

Anemia is one of the most frequent complications of CKD, appearing early, often from stage 2, and worsening as glomerular filtration rate declines (3, 4) (Macdougall, 2024; Franczyk et al., 2024). The pathophysiology of CKD-related anemia is multifactorial, including relative erythropoietin deficiency, iron dysregulation, chronic inflammation, bone marrow dysfunction, and nutritional deficiencies (3, 5) (Franczyk et al., 2024; Matsuoka et al., 2024). Iron deficiency is particularly prevalent; disturbances in iron homeostasis frequently lead to iron-deficiency anemia in CKD, and dietitians/nutritionists can play a significant role in enhancing management through multidisciplinary care (6, 7) (Kazmi et al., 2025; Babitt & Lin, 2014).

In early CKD stages (I-III), nutritional management is a cornerstone of conservative treatment. The KDOQI 2006 guidelines emphasized the importance of monitoring iron status and maintaining hemoglobin levels within a defined target range (8) (KDOQI, 2006). Subsequent European Renal Best Practice (ERBP) position statements refined these principles for European practice (9). Current best practices for managing anemia in early-stage CKD, as outlined by (10), recommend initiating a diagnostic work-up when hemoglobin levels fall below 11 g/dL, a threshold that aligns with the clinical reality of our patient cohort. These guidelines also stress the need to systematically evaluate iron stores (ferritin and transferrin saturation) and to correct nutritional deficiencies before considering erythropoiesis-stimulating agents.

However, the specific effect of such dietary advice on anemia parameters has not been extensively studied in North African populations. Many patients, fearing disease progression, adopt overly restrictive diets that inadvertently reduce intake of heme iron and B vitamins, potentially worsening anemia (11, 12). We therefore conducted a retrospective study to evaluate the impact of a 4-month standardized dietary intervention on hemoglobin, iron status, vitamin B12, and folate levels in 29 Algerian patients with CKD stages I-III and established anemia.

2. PATIENTS AND METHODS

Study design and population

This retrospective observational study was conducted at the nephrology unit of Ouled Fayet polyclinic (Algiers, Algeria) between February and May 2018. Medical records of patients with CKD were reviewed. Inclusion criteria: age ≥ 18 years, CKD stages I-III according to KDIGO 2012 (creatinine clearance ≥ 30 mL/min), anemia (hemoglobin < 13 g/dL in men, < 12 g/dL in women), and availability of complete biological data before and after a 4-month dietary intervention. Exclusion criteria: acute kidney injury, CKD stages IV-V, active malignancy, ongoing treatment with erythropoiesis-stimulating agents, or iron/vitamin supplementation during the study period (except for vitamin D). A total of 360 patients were screened; 311 had available medical records. After applying inclusion and exclusion criteria, 29 patients with complete pre- and post-intervention data were included.

Dietary intervention

A personalized dietary brochure was provided to each patient, based on KDOQI guidelines (KDOQI, 2006) (8). The key advice: protein intake of 0.8 g/kg/day for stage II and 0.6 g/kg/day for stage III; energy intake of 30–35 kcal/kg/day; sodium < 5 –6 g/day; potassium and phosphorus restriction when blood levels were elevated; increased consumption of heme iron (lean red meat, poultry, fish) and non-heme iron (lentils, spinach) combined with vitamin C to enhance absorption; and increased intake of vitamin B12 (eggs, dairy, fish, meat) and folate (green leafy vegetables, legumes). Patients were instructed to follow the advice for 4 months. No additional iron or vitamin supplements were prescribed. Adherence was assessed through dietary recall at monthly visits.

Biological measurements

Blood samples were collected at baseline (before the dietary intervention) and after 4 months. Measurements included hemoglobin (Hb), mean corpuscular volume (MCV), mean corpuscular hemoglobin concentration (MCHC), serum ferritin, serum iron, transferrin saturation (TSAT), creatinine, urea, uric acid, sodium, potassium, calcium, phosphorus, vitamin B12, and folate. All analyses were performed using standard automated methods in the same laboratory. Creatinine clearance was estimated using the Cockcroft-Gault formula.

Statistical analysis

Data are expressed as mean $\pm$ standard deviation (SD) or percentages. Paired t-tests were used to compare pre- and post-intervention values. Pearson's correlation coefficient assessed the relationship between creatinine clearance and hemoglobin. A p-value < 0.05 was considered statistically significant. All analyses were performed using Excel 2016 (Microsoft Corp., Redmond, WA, USA).

Ethical considerations

The nutritional intervention was offered as first-line management for mild-to-moderate anemia (hemoglobin 8–12/13 g/dL), in keeping with KDIGO and ERBP guidelines that recommend correcting nutritional deficiencies before initiating erythropoiesis-stimulating agents or intravenous iron. The three patients with severe anemia (Hb <8 g/dL) at baseline were not denied pharmacological treatment; they received close monthly monitoring, and none experienced clinical deterioration or required rescue therapy during the 4-month period. Had hemoglobin fallen further or had symptoms worsened, iron or ESAs would have been initiated. Thus, no patient was harmed by the nutritional approach.

3. RESULTS

Baseline characteristics of the study population

A total of 29 patients (72% women, 28% men) with a mean age of 63±15 years (range 19–78) were included. Most patients were retired or without professional activity (89.7%). Overweight or obesity (BMI ≥25 kg/m²) was present in 81% of patients (55.2% overweight, 25.9% obese). The main etiologies of CKD were diabetic and hypertensive nephropathy (51.7%), isolated hypertensive nephropathy (24.1%), diabetic nephropathy (10.3%), and undetermined cause (13.8%). Baseline distribution of CKD stages: stage I (10.3%), stage II (20.7%), stage III (69.0%) (Table 1).

Table 1. Baseline characteristics of the 29 patients.

Characteristic	Value
Age (years), mean ± SD (range)	63.1±14.8 (19–78)
Female sex, n (%)	21 (72.4)
BMI (kg/m ²), mean±SD	28.6±4.9
Overweight (BMI 25–29.9), n (%)	16 (55.2)
Obesity (BMI ≥30), n (%)	8 (27.6)
CKD stage I, n (%)	3 (10.3)
CKD stage II, n (%)	6 (20.7)
CKD stage III, n (%)	20 (69.0)
Diabetic + hypertensive nephropathy, n (%)	15 (51.7)
Hypertensive nephropathy alone, n (%)	7 (24.1)
Diabetic nephropathy alone, n (%)	3 (10.3)
Undetermined etiology, n (%)	4 (13.8)

Baseline anemia and iron status

At baseline, mean hemoglobin was 10.9±1.2 g/dL. According to WHO severity criteria, 6.9% had severe anemia (Hb <8 g/dL), 24.1% moderate anemia (Hb 8–10 g/dL), and 69.0% mild anemia (Hb 10–12/13 g/dL). The morphological type of anemia (based on MCV and MCHC) was normocytic normochromic in 55.2%, microcytic normochromic in 13.8%, normocytic hypochromic in 17.2%, microcytic hypochromic in 10.3%, and macrocytic normochromic in 3.4%.

Iron deficiency (ferritin <100 ng/mL) was present in 48.3% of patients, and low transferrin saturation (<20%) in 24.1%. Mean ferritin was 48.2 ± 32.1 ng/mL, and mean TSAT was 18.3 ± 5.2%. Vitamin B12 deficiency (<200 pg/mL) was found in 34.5% of patients, and folate deficiency (<4 ng/mL) in 10.3%.

Effect of the 4-month dietary intervention

After 4 months of dietary advice, mean hemoglobin increased significantly to 12.1±0.9 g/dL ($p<0.001$). The proportion of patients with moderate or severe anemia decreased from 31.0% to 10.3%. Changes in hematological, iron, and vitamin parameters are summarized in Table 2. The prevalence of microcytic anemia fell from 24.1% to 10.3%, and normocytic normochromic anemia increased from 55.2% to 65.5%, indicating improved iron availability for erythropoiesis (Table 3).

Renal function and correlation with hemoglobin

Mean creatinine clearance (Cockcroft-Gault) was 41.3±11.7 mL/min at baseline and did not change significantly after 4 months (42.1±10.9 mL/min, $p=0.64$). There was no significant correlation between creatinine clearance and hemoglobin at baseline ($r=0.053$, $p=0.79$, Figure 1). However, given the small sample size ($n=29$), this lack of correlation should not be interpreted as proof that anemia

is independent of GFR; rather, it suggests that in this particular cohort, nutritional factors (iron and vitamin B12 deficiencies) were more prominent determinants of hemoglobin level than the degree of renal impairment.

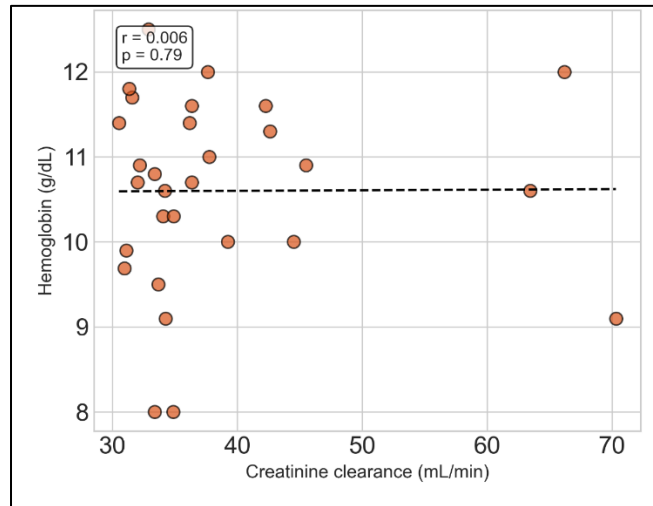


Figure 1. Correlation between creatinine clearance and hemoglobin at baseline (scatter plot with regression line; $r=0.053$, $p=0.79$). Correlation between creatinine clearance (mL/min) and hemoglobin (g/dL) at baseline. Scatter plot with a linear regression line. No significant correlation was found ($r = 0.053$, $p = 0.79$, Pearson's correlation).

Table 2. Biological parameters before and after 4-month dietary intervention (n=29).

Parameter	Before (mean $\pm$ SD)	After 4 months (mean $\pm$ SD)	p-value*
Hemoglobin (g/dL)	10.9 $\pm$ 1.2	12.1 $\pm$ 0.9	<0.001
MCV (fL)	82.7 $\pm$ 8.5	88.3 $\pm$ 6.2	0.02
Ferritin (ng/mL)	48.2 $\pm$ 32.1	72.5 $\pm$ 28.4	0.03
Serum iron (μ g/dL)	68.5 $\pm$ 22.3	85.2 $\pm$ 18.6	0.01
Transferrin saturation (%)	18.3 $\pm$ 5.2	23.6 $\pm$ 4.8	0.02
Vitamin B12 (pg/mL)	245 $\pm$ 89	312 $\pm$ 76	0.04
Folate (ng/mL)	6.2 $\pm$ 2.8	7.5 $\pm$ 2.1	0.08 (ns)
Creatinine (mg/L)	15.6 $\pm$ 1.9	14.5 $\pm$ 2.7	0.03
Urea (g/L)	0.64 $\pm$ 0.35	0.60 $\pm$ 0.31	0.03
Uric acid (mg/L)	83.1 $\pm$ 17.1	76.3 $\pm$ 14.1	0.03
Glycemia (g/L)	1.42 $\pm$ 0.66	1.31 $\pm$ 0.46	0.10 (ns)
HDL (g/L)	0.57 $\pm$ 0.23	0.48 $\pm$ 0.14	0.02
LDL (g/L)	1.10 $\pm$ 0.43	1.07 $\pm$ 0.40	0.40 (ns)
Triglycerides (g/L)	1.51 $\pm$ 0.46	1.39 $\pm$ 0.59	0.04

*Paired t-test.

Table 3. Distribution of anemia types before and after intervention.

Anemia type	Before (n, %)	After 4 months (n, %)
Normocytic normochromic	16 (55.2)	19 (65.5)
Microcytic hypochromic	3 (10.3)	1 (3.4)
Microcytic normochromic	4 (13.8)	2 (6.9)
Normocytic hypochromic	5 (17.2)	6 (20.7)
Macrocytic normochromic	1 (3.4)	1 (3.4)

Dietary changes reported by patients

Dietary recall indicated that before the intervention, most patients had spontaneously reduced meat and egg consumption for fear of protein overload. After receiving the brochure, heme iron intake increased in 72% of patients, and consumption of green leafy vegetables increased in 65%. Sodium and potassium restriction was well followed in 80% of patients.

4. DISCUSSION

This retrospective study demonstrates that a 4-month standardized dietary intervention significantly improves hemoglobin levels, iron status, and vitamin B12 levels in Algerian patients with CKD stages I-III and anemia. The improvement was achieved without the use of erythropoiesis-stimulating agents or iron supplements, highlighting the potential of nutritional management alone in early CKD.

The baseline characteristics of our cohort (predominance of women, mean age 63 years, high prevalence of overweight/obesity, and diabetic/hypertensive nephropathy as main etiologies) are consistent with previous Algerian and international CKD registries (1, 2). The high frequency of iron deficiency (48%) and vitamin B12 deficiency (34.5%) at baseline is striking and likely reflects both the uremic milieu (reduced intestinal absorption, inflammation) and, importantly, iatrogenic malnutrition - patients had excessively restricted protein-rich foods without medical advice, fearing progression to dialysis (11, 12).

After only 4 months of structured dietary advice (emphasizing adequate but not excessive protein, heme iron, and B vitamins), we observed a mean hemoglobin rise of 1.2 g/dL, a 50% relative increase in ferritin, and a 30% increase in TSAT. The morphological shift from microcytic/hypochromic to normocytic/normochromic anemia confirms the correction of iron-deficient erythropoiesis. These results align with the 2006 KDOQI guidelines, which emphasized iron repletion for low TSAT and/or ferritin thresholds and recognized the importance of nutritional assessment (8). More recent European recommendations also support systematic evaluation of iron stores before considering erythropoiesis-stimulating agents-(9) (13).

The lack of correlation between creatinine clearance and hemoglobin ($r=0.053$) is an important finding. It supports the view that in early CKD (stages I-III), anemia is not simply a consequence of reduced erythropoietin production but is largely driven by reversible nutritional deficiencies. This offers a window of opportunity for dietary intervention before pharmacologic therapy (iron or erythropoiesis-stimulating agents) becomes necessary (3, 6).

Our results are in line with a 2024 narrative review that identified nutritional deficiencies as a key component of anemia pathophysiology in CKD (3), and with the 2025 literature review on iron-deficiency anemia in CKD (6), which highlighted that dietitians/nutritionists can play a significant role in enhancing management through multidisciplinary care. Similarly, Di Iorio et al. (14) showed that a very low protein diet supplemented with iron and vitamins improved anemia in advanced CKD.

Limitations

This study has several limitations. First, its retrospective design and the inclusion of only 29 of the 360 initially screened patients introduce a substantial selection bias; patients with complete pre- and post-intervention data may differ systematically from those excluded. Therefore, our findings may not be representative of the broader early CKD population. Second, the small sample size ($n=29$) and the absence of a control group preclude causal inference.

Third, we did not measure inflammatory markers (e.g., CRP) or hepcidin, which would have allowed differentiation between absolute and functional iron deficiency. Thus, the observed improvement in iron parameters may partly reflect correction of functional iron deficiency, although the concomitant rise in ferritin and TSAT favors replenishment of iron stores. Fourth, the 4-month follow-up is relatively short. We cannot assess the long-term sustainability of the dietary changes, nor can we draw any conclusions about the impact on CKD progression or the eventual need for erythropoiesis-stimulating agents. Longer prospective studies are needed. Fifth, although we specifically asked patients about over-the-counter multivitamin, iron, or vitamin B12 supplements at each monthly visit, we cannot completely exclude unreported self-medication or recall bias. This remains a potential confounding factor.

Finally, the absence of a significant correlation between creatinine clearance and hemoglobin (Figure 1) should not be interpreted as proof of independence given the small sample size. In this cohort, nutritional factors appear to have been major contributors to anemia, but this observation requires confirmation in larger studies.

5. CONCLUSION

A 4-month targeted dietary intervention, based on moderate protein restriction and increased intake of heme iron and B vitamins, significantly improved anemia, iron stores, and vitamin B12 levels in patients with CKD stages I-III. These findings suggest that nutritional counseling should be an integral part of early CKD care, especially in populations where spontaneous dietary restrictions are common. Larger prospective controlled trials are warranted to confirm these results and to assess long-term outcomes, including the need for erythropoiesis-stimulating agents and progression of CKD.

Competing interests: The authors declare that they have no competing interest.

Funding: This research received no external funding.

REFERENCES

1. GBD 2023 Kidney Failure with Replacement Therapy Collaborators. Global, regional, and national prevalence of kidney failure with replacement therapy and associated aetiologies, 1990-2023: a systematic analysis for the Global Burden of Disease Study 2023. *Lancet Glob Health*. 2025;13(8):e1378-e1395. [https://doi.org/10.1016/S2214-109X\(25\)00198-6](https://doi.org/10.1016/S2214-109X(25)00198-6)
2. Meguellati N, Rayane T, Boulahia Y, Saidani M, Atik A, Saidi A, et al. [Epidemiology of chronic kidney disease stage V treated in southeastern Algeria: prospective study from 2016 to 2018]. *Nephrol Ther*. 2023;19(1):45-57. <https://doi.org/10.1684/ndt.2023.4>
3. Badura K, Janc J, Wąsik J, Gnitecki S, Skwira S, Młynarska E, et al. Anemia of Chronic Kidney Disease - A Narrative Review of Its Pathophysiology, Diagnosis, and Management. *Biomedicines*. 2024;12(6):1191. <https://doi.org/10.3390/biomedicines12061191>
4. Macdougall IC. Anaemia in CKD - treatment standard. *Nephrol Dial Transplant*. 2024;39(5):770-777. <https://doi.org/10.1093/ndt/gfad250>
5. Matsuoka T, Abe M, Kobayashi H. Iron Metabolism and Inflammatory Mediators in Patients with Renal Dysfunction. *Int J Mol Sci*. 2024;25(7):3745. <https://doi.org/10.3390/ijms25073745>
6. Kazmi S, Zarovniaeva V, Cortez Perez K, Sandhu S, Anwar S, Mohammed L. Iron-Deficiency Anemia in Chronic Kidney Disease: A Literature Review of Its Pathophysiology, Diagnosis, and Management. *Cureus*. 2025;17(1):e77598. <https://doi.org/10.7759/cureus.77598>
7. Zumbrennen-Bullough K, Babitt JL. The iron cycle in chronic kidney disease (CKD): from genetics and experimental models to CKD patients. *Nephrol Dial Transplant*. 2014;29(2):263-273. <https://doi.org/10.1093/ndt/gft443>
8. KDOQI; National Kidney Foundation. KDOQI Clinical Practice Guidelines and Clinical Practice Recommendations for Anemia in Chronic Kidney Disease. *Am J Kidney Dis*. 2006;47(5 Suppl 3):S11-145.
9. Locatelli F, Bárány P, Covic A, De Francisco A, Del Vecchio L, Goldsmith D, et al. Kidney Disease: Improving Global Outcomes guidelines on anaemia management in chronic kidney disease: a European Renal Best Practice position statement. *Nephrol Dial Transplant*. 2013;28(6):1346-1359. <https://doi.org/10.1093/ndt/gft033>
10. Bhandari S, Spencer S, Oliveira B, Mikhail A, Brooks O, Bryant G, et al. UK kidney association clinical practice guideline: update of anaemia of chronic kidney disease. *BMC Nephrol*. 2025;26(1):193. <https://doi.org/10.1186/s12882-025-04115-1>
11. Wu HHL, McDonnell T, Chinnadurai R. Physiological Associations between Vitamin B Deficiency and Diabetic Kidney Disease. *Biom edicines*. 2023;11(4):1153. <https://doi.org/10.3390/biomedicines11041153>
12. Azar R, Al-Moubarak I, Barsumau J, Smessaert C, Vairon MX. [Assessment and follow-up of nutritional status in hemodialysis patients]. *Nephrol Ther*. 2009;5 Suppl 5:S317-322.
13. Padhi S, Glen J, Pordes BA, Thomas ME; Guideline Development Group. Management of anaemia in chronic kidney disease: summary of updated NICE guidance. *BMJ*. 2015;350:h2258. <https://doi.org/10.1136/bmj.h2258>
14. Di Iorio BR, Di Micco L, Marzocco S, De Simone E, De Blasio A, Sirico ML, et al. Very Low-Protein Diet (VLPD) Reduces Metabolic Acidosis in Subjects with Chronic Kidney Disease: The "Nutritional Light Signal" of the Renal Acid Load. *Nutrients*. 2017;9(1):69. <https://doi.org/10.3390/nu9010069>