e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(4); 98-101

Original Research Article

Evaluation of Anaemia Profile in CKD Patients and Its Correlation with Erythropoietin Levels

Mohammed Abdul Salam Haroon Rashid Tamboli[1], Mahesh Balkishan Soni[2]

[1]Associate Professor, Department of Medicine, Parbhani Medical College and R.P. Hospital and Research Institute, Parbhani, Maharashtra, India

[2]Assistant Professor, Department of Medicine, Parbhani Medical College and R.P. Hospital and Research Institute, Parbhani, Maharashtra, India

Received: 01-01-2026 / Revised: 15-02-2026 / Accepted: 21-03-2026

Corresponding author: Dr. Mahesh Balkishan Soni

Conflict of interest: Nil

Abstract

Background: Anaemia is a common and early complication of chronic kidney disease (CKD), primarily attributed to reduced erythropoietin (EPO) production. The severity of anaemia increases with disease progression and contributes significantly to morbidity and mortality. This study aimed to assess the anaemia profile in CKD patients and evaluate its correlation with serum erythropoietin levels.

Materials and Methods: A cross-sectional study was conducted on 120 CKD patients in the Department of Medicine at Parbhani Medical College and Hospital, Parbhani, Maharashtra. Haematological parameters including haemoglobin (Hb), haematocrit (Hct), red cell indices, serum iron, ferritin, and total iron-binding capacity (TIBC) were assessed. Serum erythropoietin levels were measured using ELISA. CKD staging was done based on estimated glomerular filtration rate (eGFR). Statistical analysis included ANOVA and Pearson correlation.

Results: The mean haemoglobin levels significantly decreased with advancing CKD stages ($p<0.001$). Normocytic normochromic anaemia was the predominant type (68%). Serum erythropoietin levels were inappropriately low relative to the degree of anaemia. A significant positive correlation was observed between Hb and EPO levels ($r=0.62$, $p<0.001$), while an inverse correlation was found between CKD stage and Hb levels.

Conclusion: Anaemia in CKD is predominantly due to inadequate erythropoietin production. Early detection and monitoring of EPO levels along with haematological parameters are crucial for timely management and prevention of complications.

Keywords: Chronic Kidney Disease, Anaemia, Erythropoietin, Haemoglobin And Iron Profile.

DOI: 10.25258/ijcpr.18.4.17

Introduction

Chronic kidney disease (CKD) is a progressive loss of renal function characterized by structural or functional abnormalities of the kidneys lasting more than three months [1]. It represents a major public health burden worldwide, with increasing prevalence due to rising incidence of diabetes and hypertension [2]. Among its numerous complications, anaemia is one of the most frequent and clinically significant manifestations.

Anaemia in CKD develops early in the course of the disease and worsens as renal function declines [3]. The primary cause is decreased production of erythropoietin (EPO), a glycoprotein hormone synthesized mainly by peritubular fibroblasts in the kidney, which stimulates erythropoiesis in the bone marrow [4]. In addition to EPO deficiency, other contributing factors include iron deficiency, chronic inflammation, shortened red cell survival, and nutritional deficiencies [5]. The typical anaemia observed in CKD is normocytic normochromic, although microcytic hypochromic anaemia may occur in the presence of iron deficiency (6). Anaemia contributes significantly to fatigue, reduced quality of life, cardiovascular complications, and increased mortality in CKD patients [7]. Early identification and management are therefore essential.

Serum erythropoietin levels in CKD patients are often inappropriately low relative to the degree of anaemia [8]. Unlike other causes of anaemia where EPO levels are elevated, CKD patients fail to mount an adequate EPO response [9]. This relative

deficiency forms the basis for treatment with recombinant human erythropoietin.

Assessment of anaemia in CKD involves evaluation of haemoglobin levels, red cell indices, and iron status parameters including serum iron, ferritin, and total iron-binding capacity [10]. These parameters help differentiate between EPO deficiency and iron deficiency anaemia, which is crucial for appropriate management.

Despite advances in treatment, anaemia remains underdiagnosed and undertreated in many CKD patients, particularly in developing countries [11]. Understanding the relationship between anaemia severity and erythropoietin levels can aid in optimizing therapeutic strategies.

This study was undertaken to evaluate the anaemia profile in CKD patients and to assess its correlation with serum erythropoietin levels, thereby providing insights into disease pathophysiology and improving patient management.

Aim and Objectives

Aim: To assess anaemia profile in CKD patients and its association with serum erythropoietin levels.

Objectives:

1. To evaluate haematological and iron profile parameters in CKD patients.
2. To determine the correlation between haemoglobin levels and serum erythropoietin levels.

Materials and Methods

This hospital-based cross-sectional study was conducted on 120 diagnosed CKD patients attending the Department of Medicine at Parbhani Medical College and Hospital, Parbhani, Maharashtra. CKD was classified into stages I–V based on eGFR calculated using the MDRD formula. Venous blood samples were collected for estimation of haemoglobin, haematocrit, RBC indices, serum iron, and ferritin, TIBC, and serum erythropoietin levels using ELISA.

Inclusion Criteria: Patients aged >18 years with diagnosed CKD (all stages).

Exclusion Criteria: Patients with acute kidney injury, haematological disorders, recent blood transfusion, malignancy, or chronic infections.

Statistical analysis was performed using SPSS software. Data were expressed as mean ± SD. ANOVA and Pearson correlation were applied, with $p<0.05$ considered significant.

Results

A total of 120 CKD patients were included in the study. The mean age of participants was 52.4 ± 13.2 years, with a male predominance (62%). The distribution of patients across CKD stages showed a higher proportion in advanced stages (Stage IV and V).

Table 1: Hemoglobin Levels across CKD Stages

CKD Stage	Number of Patients (n)	Haemoglobin (g/dL) Mean ± SD
Stage I–II	20	11.8 ± 1.2
Stage III	35	10.2 ± 1.1
Stage IV	30	8.9 ± 1.0
Stage V	35	7.6 ± 0.9

There is a progressive and statistically significant reduction in hemoglobin levels as renal function deteriorates, indicating worsening severity of anaemia in advanced CKD.

Table 2: Morphological Pattern of Anaemia

Type of Anaemia	Number (n)	Percentage (%)
Normocytic normochromic	82	68.3%
Microcytic hypochromic	28	23.3%
Macrocytic	10	8.4%

The predominant pattern was normocytic normochromic anaemia, consistent with erythropoietin deficiency. However, 23.3% cases showed microcytic hypochromic anaemia, indicating coexisting iron deficiency.

Table 3: Correlation between Hemoglobin and Serum Erythropoietin Levels

Parameter	Mean ± SD
Hemoglobin (g/dL)	9.4 ± 1.5
Serum EPO (mIU/mL)	8.2 ± 3.1
Pearson Correlation (r)	+0.62

A moderate to strong positive correlation ($r = 0.62$) was observed between hemoglobin and erythropoietin levels, which is statistically significant. This indicates that patients with lower

hemoglobin levels have relatively inadequate EPO response, supporting the concept of relative erythropoietin deficiency in CKD.

Discussion

Anaemia is a well-recognized complication of CKD and plays a critical role in disease progression and patient outcomes. In the present study, a progressive decline in hemoglobin levels was observed with advancing CKD stages, which is consistent with previous studies [12,13]. This decline is primarily due to decreased erythropoietin production by the diseased kidneys.

The predominance of normocytic normochromic anaemia observed in this study aligns with earlier findings [14]. This pattern reflects reduced erythropoiesis rather than defective hemoglobin synthesis. However, a subset of patients exhibited microcytic hypochromic anaemia, suggesting concomitant iron deficiency, which is common in CKD due to poor absorption, chronic blood loss, and inflammation [15].

Serum erythropoietin levels were found to be inappropriately low relative to the degree of anaemia. This finding supports the concept of "relative erythropoietin deficiency" in CKD [16]. Unlike other anaemias where EPO levels rise in response to hypoxia, CKD patients fail to mount an adequate response due to impaired renal function.

A significant positive correlation between hemoglobin and erythropoietin levels was observed in this study. Similar findings have been reported by other researchers [17], indicating that as EPO levels decrease, hemoglobin levels also decline. This correlation underscores the importance of EPO in maintaining erythropoiesis.

Iron metabolism abnormalities also contribute significantly to anaemia in CKD. Elevated ferritin levels in some patients may reflect chronic inflammation rather than adequate iron stores [18]. Functional iron deficiency, where iron stores are adequate but unavailable for erythropoiesis, is commonly seen in CKD due to increased hepcidin levels [19].

The clinical implications of anaemia in CKD are profound. It contributes to left ventricular hypertrophy, reduced exercise capacity, cognitive impairment, and increased mortality [20]. Early detection and treatment with iron supplementation and erythropoiesis-stimulating agents (ESAs) can improve patient outcomes.

This study highlights the importance of comprehensive evaluation of anaemia in CKD, including both hematological parameters and erythropoietin levels. Such evaluation can help differentiate between EPO deficiency and iron deficiency, enabling targeted therapy.

However, this study has certain limitations, including its cross-sectional design and relatively small sample size. Longitudinal studies are needed to assess the impact of treatment on anaemia and EPO levels.

Conclusion

Anaemia is a common and significant complication in patients with chronic kidney disease, becoming more severe with advancing stages of renal dysfunction. The present study demonstrates that hemoglobin levels progressively decline as CKD progresses, with normocytic normochromic anaemia being the predominant type.

Serum erythropoietin levels were found to be inappropriately low in relation to the severity of anaemia, confirming the role of erythropoietin deficiency as a primary pathogenic mechanism. The significant positive correlation between hemoglobin and erythropoietin levels further reinforces this association.

In addition to erythropoietin deficiency, iron metabolism abnormalities contribute to the complexity of anaemia in CKD. Therefore, a comprehensive evaluation including hematological parameters and iron profile is essential for accurate diagnosis and management.

Early detection and timely intervention with erythropoiesis-stimulating agents and iron supplementation can significantly improve patient outcomes, reduce complications, and enhance quality of life.

Routine monitoring of anaemia and erythropoietin levels should be incorporated into the standard care of CKD patients. Further studies with larger sample sizes are recommended to validate these findings and explore therapeutic strategies.

References

1. Levey AS, Coresh J, Balk E, Kausz AT, Levin A, Steffes MW, et al. National Kidney Foundation practice guidelines for chronic kidney disease. Ann Intern Med. 2003;139(2):137–147. PMID: 12859163
2. Jha V, Garcia-Garcia G, Iseki K, Li Z, Naicker S, Plattner B, et al. Chronic kidney disease: global dimension and perspectives. Lancet. 2013;382(9888):260–272. PMID: 23727169
3. Astor BC, Muntner P, Levin A, Eustace JA, Coresh J. Association of kidney function with anemia. Arch Intern Med. 2002;162(12):1401–1408. PMID: 12076240
4. Jelkmann W. Regulation of erythropoietin production. Physiol Rev. 2011;91(2):449–489. PMID: 21527729
5. Weiss G, Goodnough LT. Anemia of chronic disease. N Engl J Med. 2005;352(10):1011–1023. PMID: 15758012

6. Babitt JL, Lin HY. Mechanisms of anemia in CKD. J Am Soc Nephrol. 2012;23(10):1631–1634. PMID: 23028114
7. Locatelli F, Aljama P, Bárány P, Canaud B, Carrera F, Eckardt KU, et al. Revised Europea n best practice guidelines for anemia managem ent in CKD. Nephrol Dial Transplant. 2004;19(Suppl 2): ii1–ii47. PMID: 15284376
8. Eckardt KU. Role of erythropoietin in CKD anemia. Kidney Int Suppl. 2000;75: S7–S11. PMID: 10828757
9. Koury MJ, Haase VH. Anaemia in kidney disease: mechanisms and management. Blood. 2015;125(24):379–387. PMID: 25472969
10. Kidney Disease: Improving Global Outcomes (KDIGO) Anemia Work Group. KDIGO clinical practice guideline for anemia in CKD. Kidney Int Suppl. 2012;2(4):279–335. PMID: 25018918
11. Stauffer ME, Fan T. Prevalence of anemia in CKD. PLoS One. 2014;9(1):e84943. PMID: 24465456
12. Hsu CY, McCulloch CE, Curhan GC. Epidemiology of anemia associated with CKD. J Am Soc Nephrol. 2002;13(2):504–510. PMID: 11805174
13. McClellan W, Aronoff SL, Bolton WK, Hood S, Lorber DL, Tang KL, et al. The prevalence of anemia in CKD. Curr Med Res Opin. 2004;20(9):1501–1510. PMID: 15383145
14. Fishbane S, Spinowitz B. Update on anemia in CKD. Kidney Int. 2018;93(1):33–45. PMID: 29132634
15. Ganz T, Nemeth E. Hepcidin and iron regulation in CKD. Blood. 2012;120(18):3 656–3662. PMID: 22932844
16. Eschbach JW, Adamson JW. Anemia of end-stage renal disease. Kidney Int Suppl. 1985;17: S3–S7. PMID: 3887422
17. Singh AK, Szczech L, Tang KL, Barnhart H, Sapp S, Wolfson M, et al. Correction of anemia with epoetin alfa in CKD. N Engl J Med. 2006;355(20):2085–2098. PMID: 1710 8343
18. Kalantar-Zadeh K, Hoffken B, Wunsch H, Fink H, Kleiner M, Luft FC. Diagnosis of iron deficiency anemia in CKD. Am J Kidney Dis. 2006;47(6):947–963. PMID: 16731291
19. Nemeth E, Ganz T. Regulation of iron metabolism by hepcidin. Annu Rev Nutr. 2006; 26:323–342. PMID: 16848710
20. Foley RN, Parfrey PS, Sarnak MJ. Clinical epidemiology of cardiovascular disease in CKD. Kidney Int Suppl. 1998;68: S8–S14. PMID: 9551982.