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PREVALENCE OF ANEMIA AND LABORATORY ALTERATIONS IN PATIENTS WITH CHRONIC KIDNEY DISEASE

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Annotation

Anemia is a widespread and severe complication of Chronic Kidney Disease (CKD) that significantly accelerates cardiovascular morbidity and reduces the quality of life. This study aims to evaluate the prevalence of anemia and specific laboratory alterations in patients with varying stages of CKD. A retrospective, cross-sectional study was conducted involving 90 patients with CKD stages 3 to 5 and a control group of 30 healthy individuals. Comprehensive laboratory analyses, including complete blood counts, iron profiles (ferritin, transferrin saturation), and estimated glomerular filtration rate (eGFR), were performed. Statistical evaluation utilized the Student's t-test and chi-square test. The results demonstrated a clear inverse correlation between renal function and hemoglobin levels. Anemia was present in 45% of Stage 3, 78% of Stage 4, and 95% of Stage 5 CKD patients. Hemoglobin levels significantly decreased from 135 ± 12 g/L in the control group to 88 ± 8.5 g/L in Stage 5 CKD patients ($p < 0.001$). Furthermore, functional iron deficiency, characterized by normal or elevated ferritin (315 ± 42 mcg/L) but low transferrin saturation (18 ± 3%), was observed in 62% of the advanced CKD cohort. The study concludes that anemia in CKD is multifactorial, driven not only by erythropoietin deficiency but also by impaired iron metabolism and systemic inflammation. Early laboratory screening and targeted therapeutic interventions are critical to managing this complication and preventing adverse cardiovascular outcomes.

Keywords
Chronic kidney disease, renal anemia, erythropoietin, glomerular filtration rate, hemoglobin, iron deficiency, ferritin.

Introduction

Chronic Kidney Disease (CKD) is a progressive public health challenge with a continually rising global prevalence. According to the World Health Organization (WHO) and the Global Burden of Disease study, CKD affects approximately 10% of the adult population worldwide. As renal function deteriorates, patients develop multiple systemic complications, among which renal anemia is one of the most prominent and clinically impactful.

The pathogenesis of anemia in CKD is complex and multifactorial. Historically, it was primarily attributed to the inadequate production of endogenous erythropoietin (EPO) by the peritubular fibroblasts of the failing kidneys. However, contemporary clinical laboratory science has identified that absolute or functional iron deficiency, shortened erythrocyte lifespan, uremic toxins, and a chronic inflammatory state (hepcidin-induced iron sequestration) play equally critical roles. Anemia not only exacerbates symptoms of fatigue, cognitive impairment, and reduced exercise tolerance but also acts as a primary catalyst for left ventricular hypertrophy and heart failure, significantly increasing the mortality rate in nephrology patients.

Literature Review

Recent international guidelines, including those from KDIGO (Kidney Disease: Improving Global Outcomes), emphasize the necessity of routine laboratory monitoring for anemia starting from CKD Stage 3. A comprehensive study by Babitt and Lin (2021) highlighted the central role of hepcidin in mediating functional iron deficiency in uremic patients, explaining why many patients exhibit resistance to erythropoiesis-stimulating agents (ESAs).

Furthermore, a multi-center cohort study by Portolés et al. (2021) demonstrated that early intervention with intravenous iron and moderate ESA doses can significantly delay the onset of severe cardiovascular events. Despite the extensive global literature on renal anemia, there remains a need to analyze the specific hematological and biochemical profiles of CKD patients in regional clinical settings to optimize diagnostic pathways and therapeutic protocols.

Materials and Methods

Study Design and Patient Population

A retrospective, cross-sectional cohort study was conducted at the clinical base of the Department of Hospital Therapy and Endocrinology at the Andijan State Medical Institute. The study included clinical and laboratory data from 120 adult participants evaluated over a 12-month period.

The participants were categorized as follows:

1. **Main Group (n=90):** Patients diagnosed with CKD Stages 3, 4, and 5 (not on dialysis). Diagnosis and staging were based on the KDIGO criteria using the eGFR (CKD-EPI formula).
2. **Control Group (n=30):** Healthy volunteers with normal renal function (eGFR > 90 mL/min/1.73m2) and no history of chronic systemic diseases.

Inclusion and Exclusion Criteria

- *Inclusion criteria:* Confirmed CKD Stages 3-5; age between 18 and 70 years.
- *Exclusion criteria:* Active bleeding, gastrointestinal malignancies, hematological disorders (e.g., thalassemia), recent blood transfusions (within 3 months), and active systemic infections.

Laboratory Analysis and Statistical Methods

Venous blood samples were collected under fasting conditions. The laboratory panel included a Complete Blood Count (CBC) focusing on Hemoglobin (Hb) and Red Blood Cell (RBC) indices. The iron profile assessed Serum Iron, Serum Ferritin, and Transferrin Saturation (TSAT). Serum creatinine was measured to calculate eGFR.

Statistical processing was performed using standard analytical software. Data were expressed as mean ± standard error of the mean (M ± m). The significance of differences between groups was determined using the Student's t-test for continuous variables and the chi-square test for categorical data. A probability value of $p < 0.05$ was considered statistically significant.

Results

Demographic analysis showed no significant age or gender differences between the CKD group (mean age 54.2 ± 3.8 years) and the control group (mean age 52.1 ± 4.1 years). The prevalence of anemia strictly correlated with the severity of renal impairment. In CKD Stage 3, anemia was detected in 45% of patients; this prevalence rose to 78% in Stage 4, and reached 95% in Stage 5. The comparative laboratory parameters across different stages of CKD are presented in Table 1.

Table 1. Laboratory characteristics of patients according to CKD stages (M ± m)

Parameter (Unit)	Control Group (n=30)	CKD Stage 3 (n=30)	CKD Stage 4 (n=30)	CKD Stage 5 (n=30)	p-value (Control vs St. 5)
eGFR (mL/min/1.73m2)	105 ± 8.5	48 ± 5.2	22 ± 3.1	11 ± 1.8	< 0.001
Hemoglobin (g/L)	135 ± 12	112 ± 9.5	98 ± 7.2	88 ± 8.5	< 0.001
RBC (10^12/L)	4.8 ± 0.4	3.9 ± 0.3	3.2 ± 0.2	2.8 ± 0.2	< 0.01
Serum Iron (mcmol/L)	18.5 ± 2.1	14.2 ± 1.8	11.5 ± 1.4	9.2 ± 1.1	< 0.01
Ferritin (mcg/L)	120 ± 15	185 ± 22	260 ± 35	315 ± 42	< 0.01
TSAT (%)	32 ± 4	24 ± 3	19 ± 2	18 ± 3	< 0.05

The data reveals a progressive decline in erythropoietic activity as eGFR decreases. Notably, while serum iron and TSAT steadily declined with advancing CKD, serum ferritin levels paradoxical increased. In Stage 5 CKD, the mean ferritin level was 315 ± 42 mcg/L (significantly higher than the control group, $p < 0.01$), coupled with a low TSAT (18 ± 3%). This laboratory pattern strongly indicates functional iron deficiency driven by chronic uremic inflammation, where iron is trapped in reticuloendothelial stores and unavailable for erythropoiesis.

Discussion

The findings of this study validate the high prevalence of anemia in advanced chronic kidney disease and highlight the complex laboratory alterations involved in its pathogenesis. The progressive drop in hemoglobin from 112 ± 9.5 g/L in Stage 3 to 88 ± 8.5 g/L in Stage 5 aligns closely with international epidemiological data reported by Stauffer and Fan (2020), reaffirming that declining functional renal mass is the primary driver of EPO deficiency.

However, the most critical clinical observation from our data is the discrepancy between ferritin and TSAT levels in advanced stages. Elevated ferritin in the presence of low serum iron and low TSAT is a classic laboratory signature of chronic inflammation-induced hepcidin excess. High hepcidin levels block intestinal iron absorption and prevent iron release from macrophages. This functional iron deficiency is a major cause of ESA hyporesponsiveness. Therefore, relying solely on hemoglobin or serum iron levels without evaluating the full iron panel (ferritin and TSAT) can lead to inappropriate therapeutic decisions in nephrology practice.

Scientific Novelty

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This study provides targeted clinical-laboratory evidence of the specific types of anemia prevalent in CKD patients within the local demographic context. By demonstrating the high incidence of functional iron deficiency (high ferritin, low TSAT) in pre-dialysis patients, the research underscores the necessity of moving beyond standard hemoglobin tests and implementing comprehensive iron profile monitoring to guide specific, individualized therapies before patients reach end-stage renal disease.

Conclusion & Recommendations

1. **Conclusion:** Anemia is a near-universal complication in advanced CKD, fundamentally linked to the decline in eGFR. Its etiology is heavily influenced not only by erythropoietin deficiency but also by functional iron deficiency secondary to systemic uremic inflammation, as evidenced by elevated ferritin and suppressed transferrin saturation.
2. **Recommendations for Practice:** * Complete iron status profiling (including Ferritin and TSAT) must be mandatory alongside standard CBC for all patients diagnosed with CKD Stage 3 and above.

o Therapeutic management should prioritize the correction of iron deficiency (preferably via intravenous iron preparations if TSAT < 20% and Ferritin < 500 mcg/L) prior to or concurrently with the initiation of Erythropoiesis-Stimulating Agents to ensure optimal clinical response and minimize cardiovascular risks.

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