RESEARCH PAPER

Association Of Hepcidin With Anemia And Iron Profile In Early Chronic Kidney Disease: A Cross-Sectional Study From A Tertiary Care Center In India

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ABSTRACT

Background

Anemia is a common and clinically important complication of chronic kidney disease (CKD), contributing significantly to morbidity and reduced quality of life. Hepcidin, a liver-derived peptide hormone, is the principal regulator of systemic iron metabolism and has emerged as an important mediator of anemia of chronic disease. In CKD, elevated hepcidin levels may impair iron availability and contribute to functional iron deficiency.

Objective To evaluate the association of serum hepcidin with hematological parameters and markers of iron metabolism in patients with chronic kidney disease.

Methods A hospital-based cross-sectional study was conducted in the Departments of Biochemistry and Nephrology at SMS Medical College and Attached Hospitals, Jaipur. A total of 347 adult patients (>18 years) with stable clinical condition across CKD stages I–III were enrolled after obtaining informed consent. Estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI creatinine equation. Patients with active infection, malignancy, recent blood transfusion, hematopoietic or immunosuppressive therapy, thrombosis, or acute cardiovascular events were excluded. Fasting venous blood samples were collected and analyzed for routine hematological and biochemical parameters using standard automated methods. Serum hepcidin-25 was measured by sandwich enzyme-linked immunosorbent assay (ELISA). Pearson correlation analysis was used to assess the relationship between serum hepcidin and hematological and iron profile markers.

Results Serum hepcidin showed a strong positive correlation with serum ferritin ($r = 0.885$, $p < 0.0001$). Significant negative correlations were observed with hemoglobin ($r = -0.577$, $p < 0.0001$), hematocrit ($r = -0.372$, $p < 0.0001$), serum iron ($r = -0.240$, $p < 0.0001$), and total red blood cell count (TRBC) ($r = -0.349$, $p < 0.0001$). No statistically significant correlation was found between serum hepcidin and mean corpuscular volume (MCV), transferrin saturation (TSAT), or total iron-binding capacity (TIBC).

Conclusion

Serum hepcidin is significantly associated with anemia and iron storage parameters in patients with CKD. These findings support the important role of hepcidin in inflammation-mediated disturbances of iron metabolism in chronic kidney disease and suggest its potential utility as a biomarker in the evaluation of CKD-related anemia.

Keywords: Hepcidin; Chronic kidney disease; Anemia; Ferritin; Iron metabolism; Inflammation
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INTRODUCTION

Chronic kidney disease (CKD) is a major global public health problem affecting millions of individuals worldwide and is associated with substantial morbidity, mortality, and healthcare burden **(1)**. It is characterized by a progressive decline in renal function and is frequently accompanied by multiple metabolic and hematological complications, among which anemia is one of the most common and clinically significant. Anemia in CKD adversely affects exercise tolerance, cognitive function, cardiovascular outcomes, and overall quality of life **(2)**.

The pathogenesis of anemia in CKD is multifactorial and includes reduced erythropoietin production, iron deficiency, chronic inflammation, nutritional deficiencies, and shortened red blood cell survival **(2)**. In recent years, the peptide hormone hepcidin has gained considerable attention as a key regulator of iron metabolism and an important mediator of anemia of chronic disease **(3)**.

Hepcidin is primarily synthesized by hepatocytes and regulates systemic iron homeostasis by binding to ferroportin, the only known cellular iron exporter, resulting in its internalization and degradation. This process inhibits intestinal iron absorption and prevents the release of iron from macrophages and hepatocytes into the circulation **(4)**. Consequently, elevated hepcidin levels reduce iron availability for erythropoiesis and contribute to functional iron deficiency.

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In patients with CKD, serum hepcidin levels are often elevated because of persistent low-grade inflammation and reduced renal clearance **(5)**. This elevation contributes to impaired iron utilization despite adequate or even increased body iron stores. Therefore, serum hepcidin has been proposed as a promising biomarker for evaluating iron metabolism disturbances and anemia in CKD patients **(6)**.

Several studies have investigated the relationship between hepcidin and iron profile parameters such as ferritin, serum iron, transferrin saturation, and total iron-binding capacity, but the results remain variable **(7,8)**. Moreover, limited data are available from the Indian population. Therefore, the present study was undertaken to evaluate the association of serum hepcidin with hematological parameters and iron profile markers in patients with chronic kidney disease attending a tertiary care center.

MATERIALS AND METHODS

A hospital based cross-sectional research was conducted inside the Department of Biochemistry in association with the Department of Nephrology, S.M.S. Medical College and Attached group of hospitals, Jaipur, Rajasthan. 347 Adult patients (>18 years) in stable clinical state in the 1 st, 2 nd , and 3rd stage of CKD, with estimated glomerular filtration rate (eGFR) >90 mL/min, 60–89, and 30–59 mL/min, respectively, calculated from CKD-EPI equation, were enrolled after obtaining consent. Patients having active infection, active cancer, on any hemopoetic drug, thrombosis, blood transfusions within three months preceding the study, immuno- suppressive therapy and acute cardiovascular complications (uncontrolled hypertension, acute coronary syndrome, and acute heart failure), were excluded from the study. All venous blood samples were taken in the morning after an overnight rest. Hematological measurements were made in fresh EDTA venous blood and clotted blood. The plasma and serum obtained after centrifugation was then frozen at –80°C until further laboratory analysis. Hemoglobin (Hb) concentration, red blood count (RBC), hematocrit (HCT), platelet count, iron profile, creatinine, urea, and uric acid were measured using standard laboratory methods (automated system) in a central laboratory. Commercially available kit was used to measure the following: hepcidin-25 by sandwich enzyme-linked immunosorbent assay (ELISA) method kit of elabscience, USA. The study was approved by the Institutional Ethical Committee.

Statistical Analysis:

All data was entered in Microsoft Excel sheet to prepare master chart and was transported to the statistical software for analysis. Linear variables were summarised as mean and standard deviation whereas nominal/categorical variables were presented as proportions (percentage).In univariate analysis, one way ANOVA test with post-hoc tukey HSD and unpaired t-test was used for comparison of linear variables. Pearson's correlation coefficient both crude and log adjusted was calculated to study correlation between two linear variables. For nominal/categorical variables chi-square test was used to determine associations. 'p' value <0.05 was taken as significant. SPSS 26.0 version and Medcalc 14.0.0 version softwares were used for all statistical calculations.

RESULTS (Table 1)

Correlation of Serum Hepcidin with Hematological Parameters

Serum hepcidin demonstrated a **moderate negative correlation with hemoglobin** (**$r = -0.577$, $p < 0.0001$**), indicating that higher hepcidin levels were associated with lower hemoglobin concentrations (figure 1). A **significant negative correlation with hematocrit** was also observed (**$r = -0.372$, $p < 0.0001$**), suggesting a close relationship between elevated hepcidin levels and worsening anemia (figure 2).

In contrast, **no statistically significant correlation** was found between serum hepcidin and **mean corpuscular volume (MCV)** (**$r = 0.031$, $p = 0.5622$**), indicating that hepcidin may not have a direct influence on red blood cell size in CKD patients (figure 3).

Correlation of Serum Hepcidin with Iron Profile Markers

A **strong positive correlation** was observed between serum hepcidin and **serum ferritin** (**$r = 0.885$, $p < 0.0001$**), suggesting that hepcidin is closely associated with iron storage status and inflammatory activity in CKD (figure 4).

Serum hepcidin also demonstrated a **significant negative correlation** with:

- **Serum iron** (**$r = -0.240$, $p < 0.0001$**) (figure 6)
- **Total red blood cell count (TRBC)** (**$r = -0.349$, $p < 0.0001$**) (figure 8)

However, **no significant correlation** was observed between serum hepcidin and:

- **Transferrin saturation (TSAT)** (**$r = 0.021$, $p = 0.6915$**) (figure 5)
- **Total iron-binding capacity (TIBC)** (**$r = 0.034$, $p = 0.5234$**) (figure 7)

These findings indicate that serum hepcidin is more strongly associated with **iron storage and inflammation-related markers** than with **iron transport indices**.

Table 1: Correlation of hepcidin with various markers of iron profile of study participants.

HB	Correlation Coefficient	-0.577
	Significance Level P	<0.0001

	n	347
HCT	Correlation Coefficient	-0.372
	Significance Level P	<0.0001
	n	347
MCV	Correlation Coefficient	0.031
	Significance Level P	0.5622
	n	347
S. FERRITIN	Correlation Coefficient	0.885
	Significance Level P	<0.0001
	n	347
TSAT	Correlation Coefficient	0.021
	Significance Level P	0.6915
	n	347
S. IRON	Correlation Coefficient	-0.24
	Significance Level P	<0.0001
	n	347
S. TIBC	Correlation Coefficient	0.034
	Significance Level P	0.5234
	n	347
TRBC	Correlation Coefficient	-0.349
	Significance Level P	<0.0001
	n	347

Figure 1: Correlation between Hb and S. Hepcidin

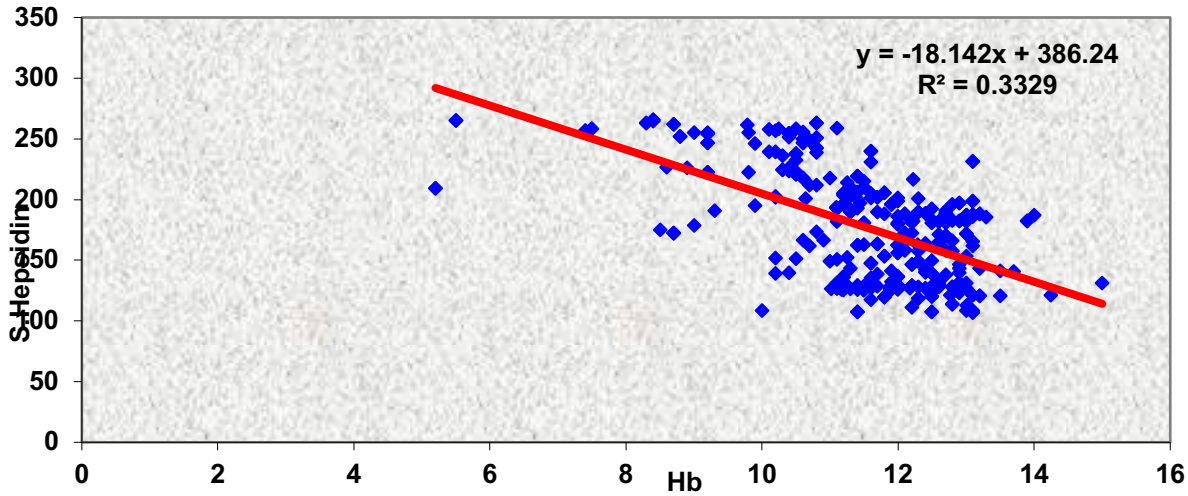


Figure 2: Correlation between HCT and S. Hepcidin

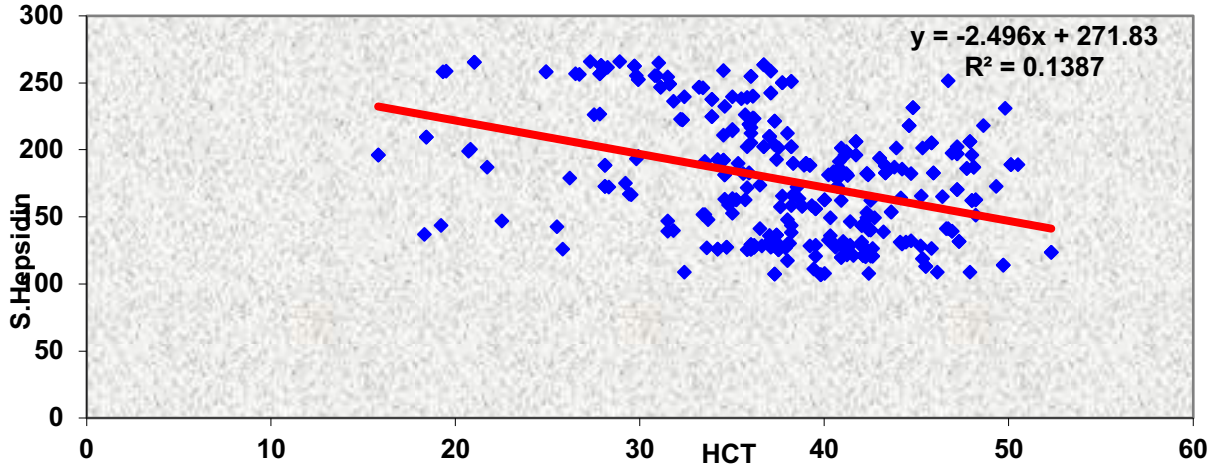


Figure 3: Correlation between MCV and S. Hepcidin

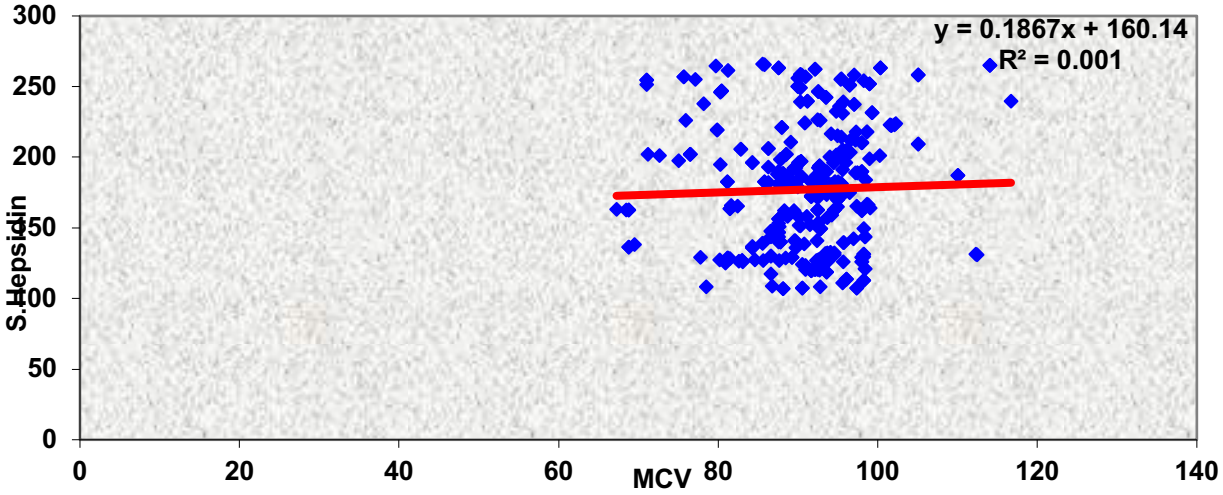


Figure 4 : Correlation between S. Ferritin and S. Hepcidin

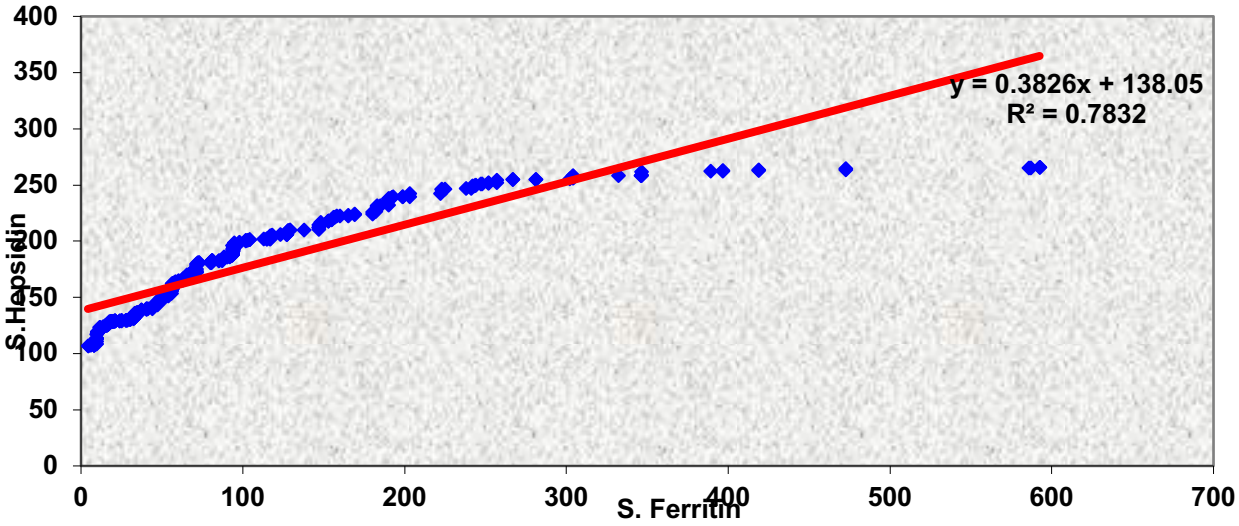


Figure 5: Correlation between TSAT and S. Hepcidin

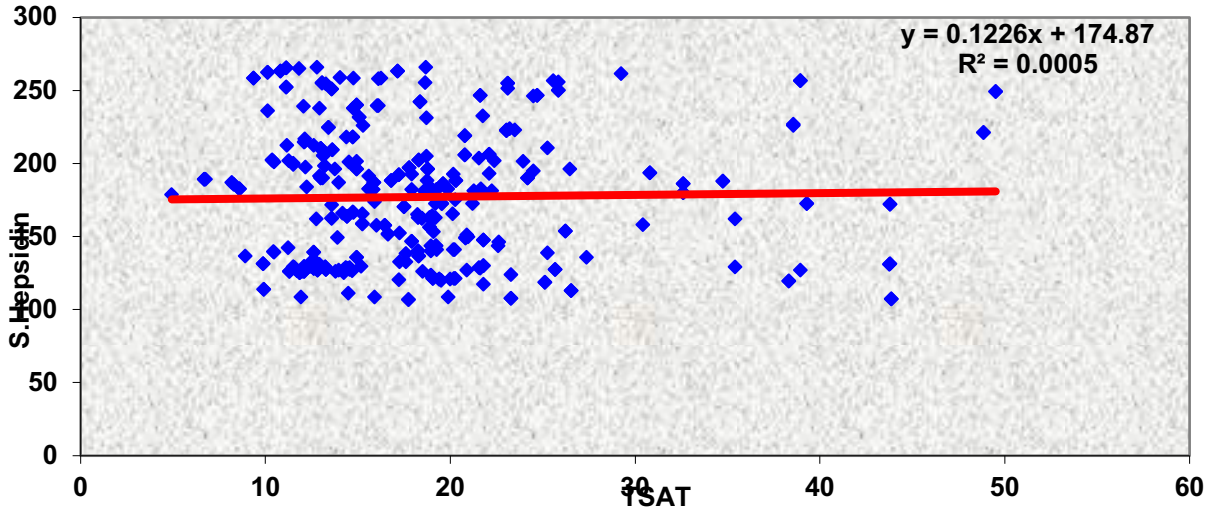


Figure 6: Correlation between S. Iron and S. Hepcidin

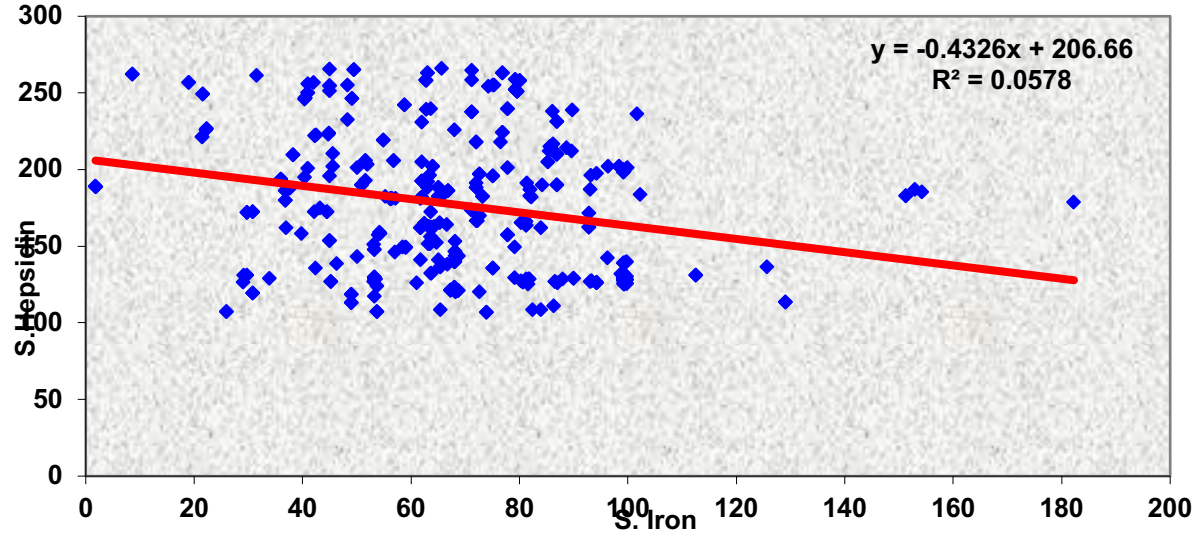


Figure 7: Correlation between TIBC and S. Hepcidin

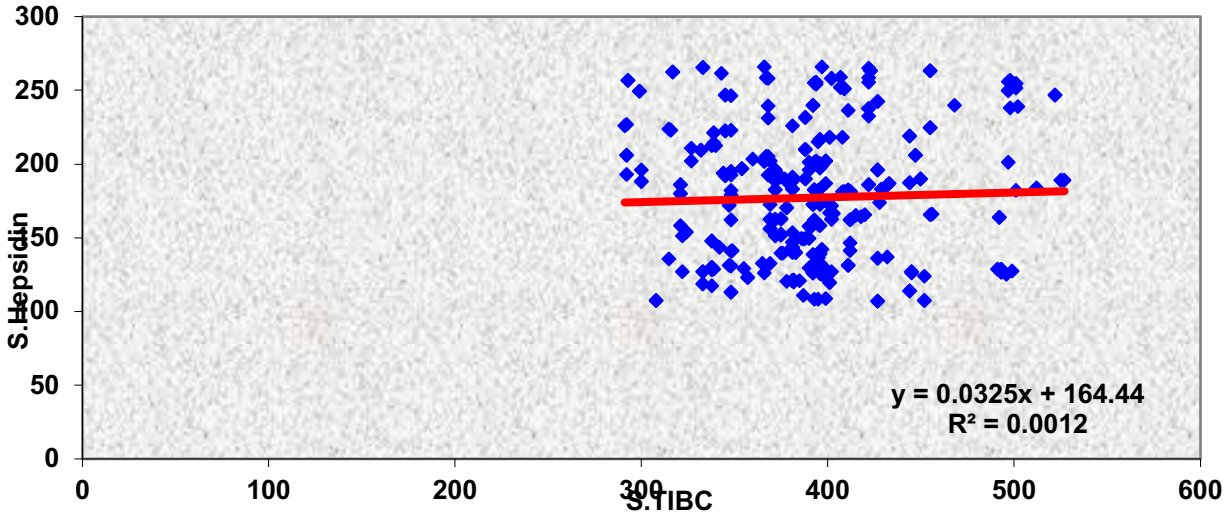
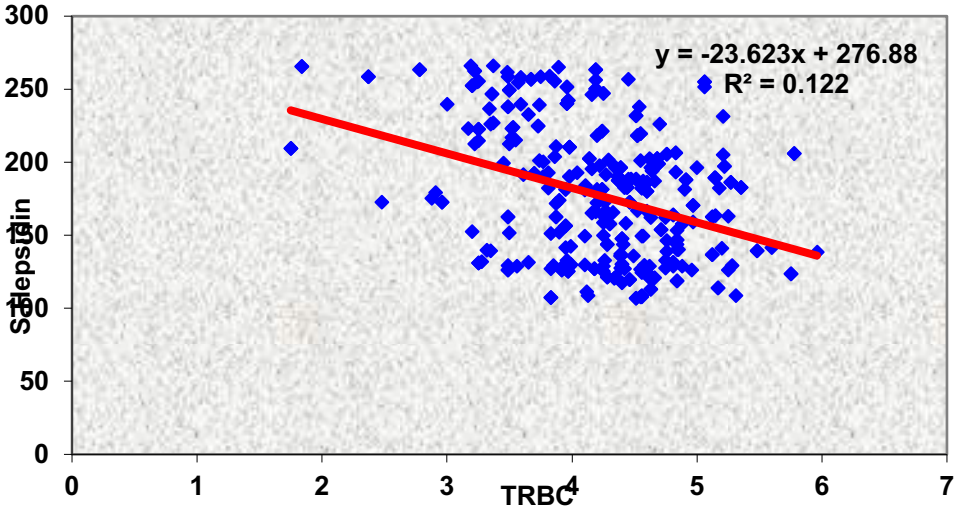


Figure 8: Correlation between TRBC and S. Hepcidin



DISCUSSION

Anemia is a frequent and clinically significant complication of chronic kidney disease (CKD), affecting a large proportion of patients as renal function declines. The pathogenesis of anemia in CKD is multifactorial and includes reduced erythropoietin production, iron deficiency, inflammation, and shortened red blood cell survival. In recent years, the peptide hormone hepcidin has emerged as a key regulator of systemic iron homeostasis and an important mediator of anemia associated with chronic inflammatory conditions such as CKD **(2,9)**.

In the present study, serum hepcidin demonstrated a strong positive correlation with serum ferritin. Ferritin is the principal intracellular iron storage protein and also behaves as an acute-phase reactant. Elevated ferritin levels in CKD patients often reflect both increased iron stores and ongoing inflammatory activity. The strong association observed between hepcidin and ferritin in the present study supports the concept that inflammation stimulates hepcidin synthesis, which in turn contributes to iron sequestration within macrophages and hepatocytes **(10,11)**.

Kalantar-Zadeh et al. **(12)** similarly reported that ferritin is one of the strongest predictors of circulating hepcidin levels, particularly in inflammatory states. Comparable findings were reported by Roy et al. **(13)**, who demonstrated significantly elevated ferritin and hepcidin levels in CKD patients compared to healthy controls. Malyszko et al. **(14)** also found significantly higher ferritin and hepcidin levels in patients with chronic renal failure and those undergoing hemodialysis. Mercadel et al. **(15)** reported a strong positive correlation between ferritin and hepcidin in a cohort of non-dialyzed, non-transplanted CKD patients. Similar observations have also been described by Farag et al. **(16)** and Ganz and Nemeth **(9)**, reinforcing the close relationship between iron storage and hepcidin regulation.

The present findings are also in agreement with Peters et al. **(7)**, who identified serum ferritin as a significant predictor of hepcidin-25 levels in CKD patients through multiple regression analysis. Dallalio et al. **(17)** similarly reported a positive association between ferritin and hepcidin in anemic individuals. However, not all studies have shown consistent results. Malyszko and Mysliwiec **(18)** did not observe a significant correlation between hepcidin and ferritin in patients with chronic renal failure, and Yeşilbaş et al. **(19)** also failed to demonstrate such an association. These discrepancies may be due to differences in study design, patient populations, inflammatory burden, dialysis status, iron therapy, and assay methodology.

Another important finding of the present study was the significant **negative correlation between serum hepcidin and hemoglobin**, indicating that higher hepcidin levels were associated with more severe anemia. This relationship is biologically plausible because hepcidin regulates iron availability for erythropoiesis by binding to ferroportin on enterocytes and macrophages, causing its internalization and degradation. This suppresses intestinal iron absorption and inhibits the release of stored iron into the circulation **(4)**. As a consequence, iron becomes unavailable for hemoglobin synthesis despite adequate or increased body iron stores, resulting in functional iron deficiency and worsening anemia **(12)**.

Our findings are consistent with those of Gao et al. **(20)** and Niikura et al. **(21)**, who also reported associations between elevated hepcidin levels and anemia-related parameters in CKD patients. Sonkar et al. **(22)** observed a progressive decline in hemoglobin levels with advancing stages of CKD despite relatively preserved iron stores, suggesting a role for reticuloendothelial iron blockade. Although their correlation between hepcidin and hemoglobin was not

statistically significant, the biological trend was similar. Farag et al. **(16)** likewise reported a negative correlation between hepcidin and hemoglobin, though it did not reach statistical significance.

The relationship between elevated hepcidin and iron-restricted erythropoiesis has been documented in multiple studies. Ashby et al. **(6)** and Weiss et al. **(23)** demonstrated that increased hepcidin concentrations are associated with lower hemoglobin levels and impaired erythropoietic response. Studies by Tessitore et al. **(24)**, van der Weerd et al. **(25)**, and Kuragano et al. **(26)** in dialysis populations, as well as those by Ganz and Nemeth **(3)** and Zaritsky et al. **(5)** in non-dialysis CKD, further support the inverse association between hepcidin and anemia severity. In contrast, Das et al. **(28)** reported a positive correlation between hepcidin and hemoglobin, and similar findings were observed by Valenti et al. **(29)**. Mercadel et al. **(15)** suggested that iron status may, in some settings, exert a stronger influence on hepcidin regulation than hemoglobin alone.

A similar negative relationship was observed between serum hepcidin and **hematocrit** in the present study. Since hematocrit reflects the proportion of circulating blood volume occupied by red blood cells, it closely parallels hemoglobin status. Elevated hepcidin levels may impair erythropoiesis through iron restriction and inflammation-mediated pathways, thereby contributing to lower hematocrit values in CKD patients **(30)**. These findings are also consistent with previous observations reported in CKD populations **(7)**.

The present study also demonstrated a **negative correlation between serum hepcidin and serum iron**, which is consistent with the central physiological role of hepcidin in controlling plasma iron availability. By inhibiting ferroportin-mediated iron export from macrophages and hepatocytes, hepcidin reduces the release of iron into the circulation, resulting in lower serum iron levels. This mechanism is central to the development of anemia of chronic disease, in which iron becomes sequestered within the reticuloendothelial system and is unavailable for effective erythropoiesis **(3)**. Similar results were reported by Zaritsky et al. **(5)**. However, Farag et al. **(16)** found a positive correlation between hepcidin and serum iron, while Yeşilbaş et al. **(19)** reported no significant association, reflecting the heterogeneity of findings across studies.

The observed **negative correlation between hepcidin and total red blood cell count (TRBC)** further supports the suppressive effect of elevated hepcidin on erythropoiesis. Chronic inflammation in CKD is associated with increased production of cytokines, particularly interleukin-6 (IL-6), which stimulates hepatic hepcidin synthesis. Elevated hepcidin then restricts iron availability to erythroid precursors in the bone marrow, ultimately impairing red blood cell production **(31,34)**.

Interestingly, the present study did not demonstrate a significant correlation between hepcidin and **mean corpuscular volume (MCV)**. MCV reflects the average size of red blood cells and is influenced by a variety of factors including iron deficiency, vitamin B12 deficiency, and folate deficiency. Anemia in CKD is classically **normocytic and normochromic**, which may explain the lack of a strong relationship between hepcidin and MCV in this cohort **(32)**.

Similarly, **no statistically significant correlation** was observed between serum hepcidin and **transferrin saturation (TSAT)** or **total iron-binding capacity (TIBC)**. TSAT is commonly used as an indicator of iron availability for erythropoiesis, while TIBC reflects transferrin concentration. In inflammatory states such as CKD, transferrin behaves as a negative acute-phase reactant, which may reduce the reliability of TSAT and TIBC as indicators of true iron availability **(33)**. Therefore, these markers may not directly reflect hepcidin-mediated alterations in iron distribution and utilization.

Another factor contributing to elevated hepcidin levels in CKD is **reduced renal clearance**. Since the kidneys are involved in the elimination and metabolism of hepcidin, declining renal function may lead to its accumulation in the circulation **(5)**. This accumulation may further aggravate iron restriction and anemia. In addition, CKD is characterized by persistent low-grade inflammation, which stimulates hepatic hepcidin production through cytokine-mediated signaling pathways. Interleukin-6, in particular, upregulates hepcidin expression through activation of the **JAK-STAT pathway (34)**. This inflammatory cascade contributes to functional iron deficiency, where iron stores are adequate but unavailable for erythropoiesis.

Overall, the findings of the present study are in agreement with several previous investigations that have highlighted the important role of hepcidin in the pathophysiology of CKD-related anemia **(6,35)**. The strong positive association of hepcidin with ferritin and its inverse relationship with hemoglobin, hematocrit, serum iron, and TRBC suggest that hepcidin may serve as a useful biomarker for evaluating iron metabolism disturbances in CKD patients.

This study has certain limitations. First, its cross-sectional design limits the ability to establish causality between serum hepcidin and anemia-related parameters. Second, the study was conducted at a single tertiary care center, which may limit generalizability of the findings. Third, inflammatory markers such as C-reactive protein (CRP) and interleukin-6 (IL-6) were not evaluated, which could have provided additional insight into the inflammatory regulation of hepcidin. Finally, patients in advanced CKD stages, including dialysis-dependent patients, were not included in the present analysis.

From a clinical perspective, the assessment of serum hepcidin may help identify patients with **functional iron deficiency** and may potentially guide

individualized treatment strategies involving iron supplementation or erythropoiesis-stimulating agents **(36)**. However, larger multicentric studies and longitudinal analyses are needed before serum hepcidin can be routinely incorporated into clinical practice.

CONCLUSION

The present study highlights **serum hepcidin as a key biomarker linking anemia, inflammation, and iron dysregulation in chronic kidney disease**. The strong positive association with ferritin and significant inverse relationships with hemoglobin, hematocrit, serum iron, and total red blood cell count emphasize its important role in the development of **functional iron deficiency and impaired erythropoiesis in CKD**.

These observations support the growing evidence that **hepcidin plays a central role in the pathogenesis of CKD-related anemia** and may provide clinically meaningful information beyond conventional iron parameters alone. Incorporation of hepcidin into the diagnostic evaluation of CKD patients may improve the identification of underlying iron-restricted erythropoiesis and help refine anemia management strategies.

Further **prospective, multicentric, and interventional studies** are warranted to validate its clinical utility and to explore its role as a potential **diagnostic, prognostic, and therapeutic biomarker** in chronic kidney disease.

CONFLICT OF INTEREST

The authors declare that there is **no conflict of interest**.

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