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CLINICAL RELEVANCE OF HEPCIDIN IN IRON DEFICIENCY ANEMIA PATIENTS: A CASE CONTROL STUDY

Meetu Maradi[1], Nilima Dongre[2], Kadappa Jaligidad[3]

[1.] PhD scholar, Department of Biochemistry, Shri B M Patil Medical College, BLDE (DU), Vijayapura, Karnataka, India.
[2.] Professor Department of Biochemistry, Shri B M Patil Medical College, BLDE (DU), Vijayapura, Karnataka, India.
[3.] Professor, Department of Medicine, SNMC, Bagalkot, Karnataka, India.
Corresponding Author: Meetu Maradi, Email ID: meetumaradi@gmail.com, Orcid: 00090004299086X

Abstract
Background: Iron deficiency anemia (IDA) is the most common cause of anemia worldwide. It is defined as a decreased iron stores in the body. A liver derived peptide hepcidin is been identified as the key systemic regulator of iron homeostasis. The diagnosis and treatment of iron deficiency anaemia requires an understanding of its relationship with demographic, socioeconomic, nutritional and haematological factors in IDA.
Objective: Evaluate the ways a liver-derived peptide hepcidin regulates the body's iron balance.
Methodology: A case-control study was conducted comprising 100 participants: 50 patients with clinically confirmed newly diagnosed IDA and 50 healthy controls aged 16–60 years. Demographic data, dietary habits, income levels, complete blood counts, peripheral blood smears and comprehensive iron profiles were evaluated. Receiver Operating Characteristics (ROC) curve analysis was utilized to determine the optimal diagnostic cut-off value for serum hepcidin.
Results: Serum hepcidin levels were significantly higher in IDA patients than in healthy controls. Hepcidin and iron levels were positively correlated. Serum hepcidin diagnostic cut-off value was 280.94 ng/ml & was determined by ROC curve analysis, resulting in a sensitivity of 92% and a specificity of 98%.
Conclusion: Elevated serum hepcidin is a highly sensitive and specific marker for diagnosing Iron Deficiency Anaemia (IDA), which helps in differentiating true iron deficiency from inflammatory conditions.

Keywords: Hepcidin**,** Iron Deficiency Anemia **(**IDA), Total Iron Binding Capacity (TIBC), Red Cell distribution Width (RDW), Iron refractory Iron Deficiency Anemia (IRIDA), Receiver Operating Characteristic (ROC), Transferrin saturation (TSAT).

INTRODUCTION

Anaemia is a serious public health challenge, primarily because of essential nutrients required for hemoglobin synthesis and RBC production. Iron transport shows that variety of proteins and enzymes, but it was discovered that hepcidin, a hormone generated in the liver, strictly regulates iron homeostasis by influencing erythrocyte synthesis. Among these nutritional deficiencies is the predominant cause. For diagnosis of severity from mild to severe is based on WHO criteria, [1]

Iron is a vital mineral that our bodies need to balance. IDA is the global nutritional disorder causing >40% of anemic cases in India according to Anemia Mukt Bharath Abhiyan programme by ICMR in India. IDA often have symtoms like pallor, weakness, fatigue, shortness of breath and dizziness with the lab findings like low Hemoglobin and low serum iron, but traditional marker alone is not sufficient to rely on. So important diagnostic tool is required for further management of IDA which is hepcidin. [2,3]

Transferrin saturation (TSAT) is a vital clinical calculation that measures the percentage of the body's iron-transport protein, transferrin currently bound to iron. In a healthy individual, normal TSAT ranges from 16% to 45%. In iron deficiency anemia (IDA), this balance is disturbed. As systemic iron stores diminish, the percentage of transferrin carrying iron reduces, making it a secondary biomarker when ferritin levels are undetermined.[4]

Hepcidin is a 25-amino acid peptide hormone that is produced and secreted predominantly by hepatocytes, circulates in the bloodstream, and is excreted by the kidneys. Serum hepcidin being a sensitive indicator of IDA is a superior diagnostic marker than ferritin because it can directly reflect the functional iron & resist confounding factors like inflammation & Anemia of chronic inflammation. This indicating a high accuracy can be helpful for predicting better marker for further iron therapy. [5]

C-reactive protein (CRP) is a acute-phase protein synthesized by the liver, primarily in response to macrophage-derived interleukin-6 (IL-6) secretion. While CRP is independent of biological iron status, its measurement is clinically useful during the evaluation of iron deficiency anemia (IDA). Because systemic inflammation alters standard iron profiles, CRP serves as a vital diagnostic filter. It tells whether concurrent inflammatory processes are distorting the patient's iron markers. [6]

The present study showed that hepcidin can be more accurate diagnostic marker than ferritin by high diagnostic precision with the more specificity and sensitivity by remaining suppressed in true IDA cases, which can be helpful

for replacing bone marrow iron staining as a diagnostic tool. It directly measures iron availability, facilitating the differentiation between absolute IDA and anemia of chronic disease & this may help in identifying a genetic disorder IRIDA. [7,8]

Methodology

Patient selection

The study was conducted at Medicine and Biochemistry department of S N. Medical College and H S K Hospital and Research Centre, Bagalkot & BLDE (Deemed to be University), Vijayapura, Karnataka, India. Clearance from the Institutional Ethics Committee (IEC) was obtained from both the institution within the period of November 2024 to October 2025. Written informed consent and questionnaire was also taken from the patients prior to the collection of blood samples.

Inclusion Criteria

Iron deficiency anemia patients characterized by nutritional deficiency within the age group of 16-60 years, attending medicine OPD, were included in the study. Subjects in the same age group without anemia were referred as healthy controls.

Exclusion Criteria

Patients having anemia due to chronic inflammation like trauma, PIH, excessive menorrhagia, anemia due to bleeding, iron overload, hemolytic anemia, on iron therapy and systemic illness and did not provide consent for the study will be excluded.

Sample Collection

In the present study sample size was calculated to 100 samples & divided as 50 clinically confirmed newly diagnosed IDA Cases and 50 Controls. Patients involved were from Medicine OPD under the inclusion criteria selected. 5ml of blood sample was collected, 3ml of blood sample was taken in plain tube, 2ml in EDTA tube under aseptic precautions which were collected based on the WHO criteria[1].

Population Group	**Haemoglobin (Hb) Cut-off**
Children 6–59 months	< 11.0 g/dL (110 g/L)
Children 5–11 years	< 11.5 g/dL (115 g/L)
Children 12–14 years	< 12.0 g/dL (120 g/L)
Non-pregnant Women (≥15 years)	< 12.0 g/dL (120 g/L)
Pregnant Women	< 11.0 g/dL (110 g/L)
Men (≥15 years)	< 13.0 g/dL (130 g/L)

Complete Blood count was done by combination of Electrical Impedance (the Coulter Principle) and Flow Cytometry using Horriba 7 parts haematology analyser (Cell Counter). Serum Iron by Ferrozine method by Semi automated analyser (Erba chem), Serum Ferritin was estimated by CLIA using Autolumo 1000 machine & TIBC was calculated using the transferrin multiplication method. Serum Hepcidin was analysed using ELISA method. [9,10,11,12]

STATISTICAL ANALYSIS:

Statistical analysis was performed using a statistical package for the social sciences (SPSS) (Version 20). Results presented as Mean± SD, counts and percentages and diagrams. Normally distributed continuous variables between two groups were compared using an independent t-test. Categorical variables between the two groups were compared using the Chi-square test. For Normally distributed continuous variables Pearson Correlation coefficient value, the Spearman's rho Correlation coefficient value were used. $p<0.05$ were considered statistically significant. All statistical tests were performed by two-tailed. ROC curve was used to determine best cut off value for the hepcidin and ferritin.

Results

Present study group comprised of 50 confirmed IDA cases and 50 healthy controls in the age group of 16-60 years.

Table 1: Demographic data of study groups.

	Controls (n=50) Mean ±SD	Cases (n=50) Mean ±SD	p value
Age (years)	38.86 ±14.32	32.9±12.63	0.07

The above table shows the average age of the Cases group (32.9±12.63) is lower than that of the Controls group (38.86 ±14.32).

Table 2: Gender distribution among study groups

Gender	Controls	Cases	Total
Male	22 (44%)	15 (30%)	37%
Female	28 (56%)	35 (70%)	63%

The distribution based on gender showed that females 35 (70%) were affected more than males 15 (30%).

Table 3: Distribution based on demographic data in the study group.

Demographic data		Frequency (n=50)	100 %
Nutritional status	Vegetarian	43	86 %
	Non-Veg	7	14 %
Socioeconomic status	High income	3	6%
	Middle income	27	54%
	Low income	20	40%

The evaluation of nutritional status with 80% (43) were vegetarian, whereas 14 % (7) were non vegetarian. Socioeconomic status indicated that majority of the cases belonged to middle income with 54%, low income 40% and high income 6%, respectively.

Table 4: Hematological parameters in controls & IDA cases.

Parameters	Controls (50)	Cases (50)	P value
Hb (g/Dl)	14.25±1.40	8.08±1.37	<0.0001*
PCV/HCT (%)	40.69 ±4.61	22.39±7.87	<0.0001*
MCV (Fl)	94.57 ±6.50	67.09±14.48	<0.0001*
MCH (pg)	30.83 ±2.39	22.01±5.59	<0.0001*
MCHC (%)	33.03 ±1.14	30.18±2.54	<0.0001*
RDW (%)	45.86±5.60	62.88±9.02	<0.0001*

p value <0.005 is significant. * significant, ** highly significant

All measured blood parameters showed highly significant differences between the two groups ($p < 0.0001$). Compared to the Control group, the Cases group had significantly lower levels of Hemoglobin (8.08 g/Dl vs. 14.25 g/Dl), hematocrit (22.39% vs. 40.69%), and red blood cell indices (MCV, MCH, and MCHC). Additionally, the Cases group showed a significantly higher Red Cell Distribution Width (RDW) (62.88% vs. 45.86%), reflecting high variation in red blood cell size.

Figure 1: Microscopic view of blood smear of controls and cases.

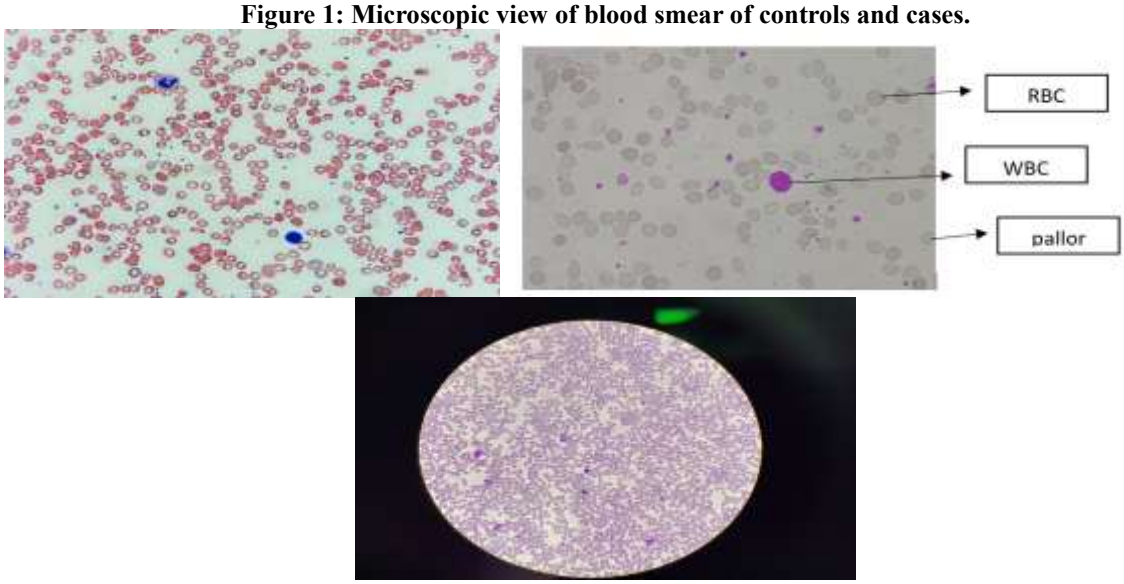


Figure 1 shows the peripheral smear of the controls & IDA case, the red blood cells shows zone of central pallor is increased and overall sizes and shapes of the RBC's are less uniform (increased anisocytosis and poikilocytosis) showing hypochromic microcytic anemia.

Table 5: Biochemical parameters in controls and cases.

Parameters	Control (50) Mean ±SD	Case (50) Mean ±SD	P value
Sr Iron (µg/dL)	114.4± 28.02	66.99±11.79	<0.0001*
Sr. Ferritin (ng/mL)	93.96±42.05	25.39±7.02	<0.0001*
TIBC (µg/dL)	327.0±52.79	344.09±67.79	<0.0001*
TSAT	36.25± 12.46	19.84± 5.47	0.0001**
Hepcidin (ng/ml)	198.26± 55.70	375.07± 66.99	0.0001**
CRP (mg/L)	3.53 ±1.68	4.50±1.68	0.005*

Serum iron (66.99±11.79), serum ferritin (25.39±7.02), TSAT (19.84± 5.47) levels were found to be decreased in cases in comparison with controls showing statistical significance with the p value <0.001 & TIBC (344.09±67.79), Hepcidin (375.07± 66.99), CRP (4.50±1.68) were increased in cases in comparison with the controls with statistically significance p value <0.0001.

Table 6: Correlation of hepcidin with iron in IDA cases.

Parameters	r value	p value
IDA Cases (n=50)		
Sr Hepcidin with Sr Iron	0.038	0.017

p value <0.005 is significant. * significant, ** highly significant

Correlation analysis between serum hepcidin and serum iron showed positive correlation (0.038) with no statistical significant difference.

Figure 2: Correlation between Hepcidin and Serum iron levels in cases

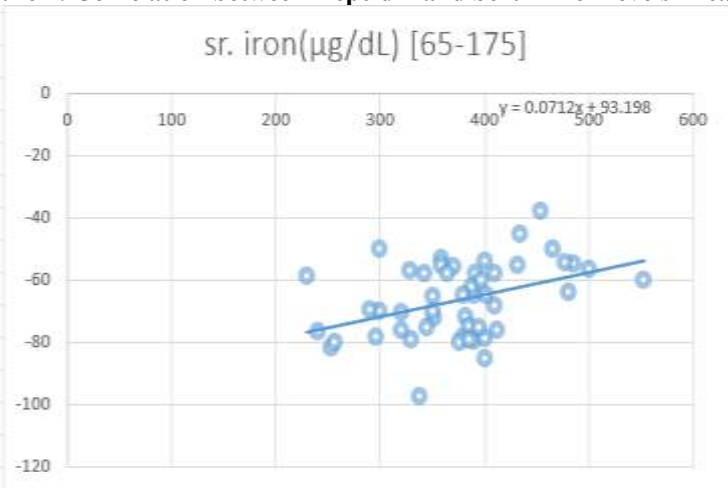
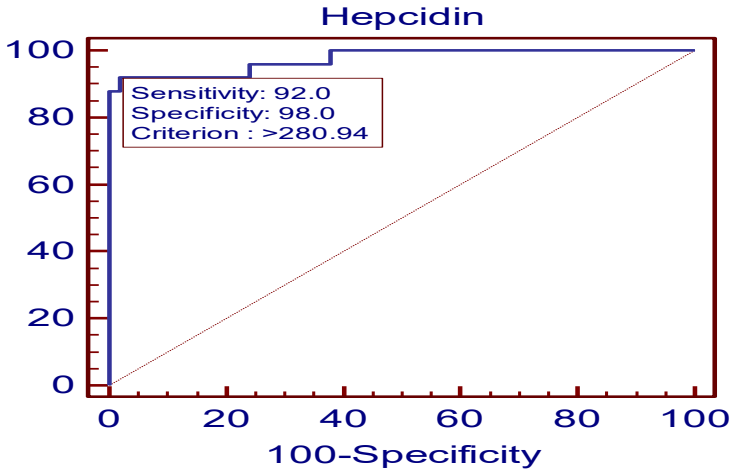
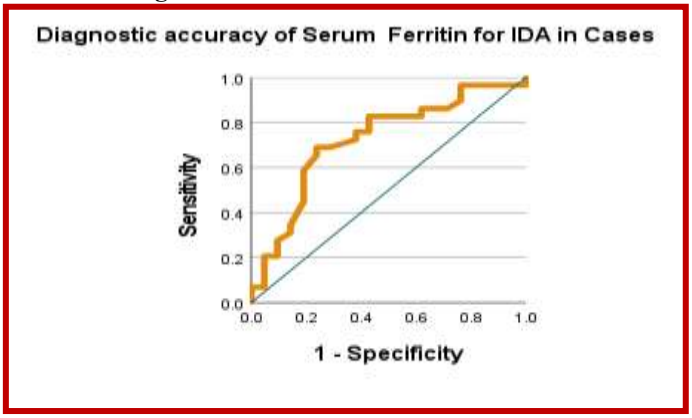


Figure 3: The ROC curve for hepcidin.



Receiver operating characteristics (ROC) curve showed the best cut off value for Hepcidin level is ≥ 280.94 ng/ml with sensitivity of 92%, specificity of 98% & AUC is 0.974.

Figure 4: The ROC curve for ferritin.



Receiver operating characteristics (ROC) curve showed the best cut off value for Ferritin level is <24 ng/mL with sensitivity of 62%, specificity of 72% & AUC is 0.72.

Discussion

In India iron deficiency anemia is not just a disease but a manifestation of an underlying disease, hence searching for the cause is more important than replacing the iron stores, as it has been associated with significant morbidity and mortality. Blood loss is the major cause of IDA, but commonest cause of IDA is still nutritional deficiency in many developing countries.

Hepcidin, a master peptide hormone produced by the liver, regulates the body's systemic iron homeostasis. By controlling the amount of iron absorbed & released from internal storage, hepcidin directly regulates the amount of iron available for erythropoiesis. In Iron Deficiency Anemia, the hepcidin-ferroportin axis functions in a state of maximal suppression to prioritize plasma iron replenishment Targeting the Hepcidin-Ferroportin Axis. Hepcidin levels that are abnormally high or low are therefore necessary for the development of various forms of anaemia.

In the present study demographic profile of the current study revealed a higher prevalence of iron deficiency anemia (IDA) among female participants, which aligns with the findings of Sharma et al. 2024[13] Biological causes including increased nutritional demands during reproductive years are frequently blamed for this gender difference. Sohaib Akbar et al. (2023) [14] showed that although a large proportion of their sample was female, gender did not show up as a statistically significant risk factor that distinguished cases from controls. Although there were no significant differences in our data according to age or socioeconomic status, there was a significant correlation with vegetarian diets, which is consistent with findings by Siddiqui et al. (2024)[15] that anaemia affects people of all income levels because of poor dietary diversity or awareness in middle-class populations and lack of nutrition in low-income groups.

When compared to controls, our study revealed that anaemia patients had significantly lower haemoglobin levels and fewer red blood cell indices. Microcytic hypochromic anaemia was confirmed by the small, pale cells with irregular sizes and shapes. According to a 2024 study by Zahid et al., a microscopic blood smear examined using red blood cell indices provides a clear picture of how blood evolves. [16] Study by Vyas S et.al (2017) showed statistically lower RBC indices but decreased hepcidin levels were seen in contrast to our study which may be because of occult conditions.[17]

The present study showed serum ferritin levels were statistically low in IDA group (25.39± 7.02 ng/ml) than the control group (65.67±16.76 ng/ml). in accordance with the Pfeiffer et al. (2023) examined the entire iron profile rather than just blood iron, providing a clear picture of how anaemia may result from early low iron levels.[18]

Our analysis showed transferrin saturation (TSAT) levels were statistically lower in the IDA group (19.84±5.47) compared to the healthy control (36.25±12.46) group. This reduction shows that serum ferritin levels also reduce in IDA in comparison with controls, indicating depletion of both intracellular iron stores and circulating transport iron. In accordance with study done by Pfeiffer et al.(2023) [19] Evaluating a patient's complete iron panel is much more accurate than depending on only complete blood count (CBC) or biochemical parameters alone.

Clinical analysis on anemic patients showed Hepcidin levels were higher in cases (375.07±66.99 ng/ml) than in controls (207.06±60.24). Hepcidin levels were significantly elevated in individuals with IDA, according to a study by Elizabeta Nemeth et al. (2014).[20] Even in cases of iron deficiencies, hepcidin production is increased in IRIDA. According to research by Svenson N et al., elevated hepcidin during an iron deficit is a characteristic linked to iron disorders brought on by inflammation or genetic factors.[21]

Present study shows that checking C-reactive protein (CRP) with regular iron tests is essential. It proves that the anemia is caused by a lack of iron and not by inflammation trapping iron inside cells. While our iron deficiency anemia (IDA) group had extremely low serum CRP levels (4.50 ± 1.68 mg/L) in comparison with controls (3.53 ± 1.68 mg/L). According to studies done by McCullough JH (2015) [22]. Inflammation releases proteins that artificially raise ferritin levels, hiding a true iron deficiency. Checking CRP confirms there is no inflammation. This proves that low ferritin levels in the body's iron stores are completely not seen. This complete analysis helps true iron deficiency early and avoid misdiagnosing it as chronic disease anemia.

In our analysis we saw a positive correlation between hepcidin and iron levels (0.038) but no statistical significance difference. According to a study by Svenson N et al., the lack of a clear association between hepcidin and circulating iron levels means that more research is needed to understand how the normal regulatory system is altered in these patients. [21]
Our study used a Receiver Operating Characteristics (ROC) curve to define the best diagnostic cut-off value for serum hepcidin levels. A cut-off value of 280.94 ng/ml or higher showed a high sensitivity of 92% and a strong specificity of 98% in identifying true anemia cases from healthy controls. In accordance to studies done by Olinder J et.al, using an ROC curve is an excellent tool to evaluate the diagnostic accuracy of a clinical biomarker. The exceptionally high sensitivity and specificity found at this cut-off level mean that hepcidin is highly reliable at both correctly identifying patients with anemia and ruling out healthy individuals.[23]
The body's primary regulator for controlling iron is hepcidin. Hepcidin normally decreases in IDA to allow the intestines to absorb iron from food or medication. Hepcidin levels remain elevated in IDA even with a significant iron deficiency may be because of IRIDA? Because of this large amount, iron cannot enter the bloodstream, making commonly used oral iron supplements completely ineffective.

Conclusion
In conclusion, our study showed that increased Hepcidin can be important marker for IDA, with high sensitivity and specificity which can help in looking for iron panels to differentiate inflammatory interference. This approach may help in ensuring diagnosis to be accurate. Furthermore, our findings support the measurement of Hepcidin to be a accurate marker for diagnosis, severity assessment & management of iron therapies showing that elevated Hepcidin levels in anemic cases compared to healthy control may actually have an underlying, undetectable inflammation or a genetic condition such as IRIDA.

Limitations
Smaller sample size. Molecular basis for increased hepcidin can be understood with the larger sample size.

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