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Original Research Article

ASSOCIATION OF SERUM HEPCIDIN WITH OBESITY, IRON STATUS, AND ANAEMIA IN INDIAN ADULTS: A CROSS-SECTIONAL STUDY

Saba Amin Khan[1], Deepali Tonde[2], Komal Meshram[3], Karuna Kachhawa[4]

[1]*Assistant Professor, Department of Biochemistry, Dr. Rajendra Gode Medical College and Hospital, Amravati, Maharashtra, India.*
[2]*Assistant Professor, Department of Biochemistry, Government Medical College and Hospital, Nagpur, Maharashtra, India.*
[3]*Assistant Professor, Department of Biochemistry, Government Medical College and Hospital, Nagpur, Maharashtra, India.*
[4]*Associate Professor, Department of Biochemistry, Dr. Rajendra Gode Medical College and Hospital, Amravati, Maharashtra, India.*

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Corresponding Author:
Dr. Saba Amin Khan,
Assistant Professor, Department of Biochemistry, Dr. Rajendra Gode Medical College and Hospital, Amravati, Maharashtra, India
Email: drsaba2112@gmail.com

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ABSTRACT

Background: Hepcidin integrates iron sensing, erythropoietic demand, and inflammatory signalling. Obesity may elevate hepcidin levels through low-grade inflammation; however, the relationship between hepcidin, adiposity, and circulating iron indices remains inadequately characterized within the Indian population.

Materials and Methods: We conducted a cross-sectional analysis involving 100 participants, with comprehensive measurements including anthropometric data, glycaemic status, haematological parameters, and iron-related indices. The associations between hepcidin and both body mass index (BMI) and iron parameters were assessed using correlation analysis and multivariable linear regression models, adjusted for age, BMI, sex, diabetes status, ferritin, serum iron, and total iron-binding capacity (TIBC.

Results: The study group included 73 females and 27 males; mean age was 41.41 ± 10.01 years, mean BMI was 45.07 ± 6.53 kg/m², and mean hepcidin was 22.15 ± 4.01 ng/mL. Anaemia was present in 26 participants (26.0%). Hepcidin was not correlated with BMI ($r=0.019$, $p=0.848$), but correlated positively with serum iron ($r=0.593$, $p<0.001$), ferritin ($r=0.230$, $p=0.022$), and inversely with TIBC ($r=-0.299$, $p=0.002$). Serum iron was the only independent predictor ($B=0.099$ ng/mL per µg/dL; 95% CI 0.060–0.138; $p<0.001$).

Conclusions: In our study, serum hepcidin demonstrated a correlation with circulating iron indices rather than with BMI. The association with BMI should be interpreted in light of the limited range of adiposity and the absence of inflammatory markers or a normal-weight comparison group.

Keywords: hepcidin; serum iron; obesity; body mass index.

INTRODUCTION

Iron deficiency is the most prevalent micronutrient deficiency globally, particularly affecting developing countries like India.[1] Iron deficiency anaemia (IDA) remains a significant public health issue in India, notwithstanding the continuous implementation of nutritional intervention programmes. According to the National Family Health Survey (NFHS-5), anaemia impacts approximately 57% of women aged 15–49 years, 25% of men, and nearly 67% of children aged 6–59 months, thus emphasising the considerable prevalence of compromised iron status across all demographic groups.[2]

India is experiencing a rapid epidemiological transition characterised by an increase in overweight and obesity, affecting approximately 40% of young adults, particularly in urban areas.[3] Urbanisation, sedentary lifestyles, and dietary modifications have consistently contributed to the rising prevalence of obesity among Indian adults.[4] Obesity, defined by abnormal energy metabolism leading to excess fat accumulation, presents significant physical and psychological challenges. It is recognised as a chronic, low-grade inflammatory condition and is a

well-established risk factor for insulin resistance, metabolic syndrome, cardiovascular disease, and type 2 diabetes mellitus.[5,6] Although it involves an energy surplus, numerous studies have identified that obesity is frequently associated with micronutrient deficiencies.[7,8] The coexistence of iron deficiency anaemia and excess adiposity represents a dual burden of malnutrition within the Indian population. The simultaneous occurrence of obesity and iron deficiency has become a significant yet not fully understood clinical issue in developing countries undergoing nutritional transition. These overlapping conditions complicate the interpretation of conventional iron biomarkers, as inflammation related to adiposity can affect iron distribution even when total body iron stores are not depleted.

Hepcidin is the principal systemic regulator of extracellular iron homeostasis.[9] Produced mainly by the liver, it binds the iron exporter ferroportin and promotes its internalization and degradation, thereby reducing intestinal iron absorption and iron release from macrophages and hepatocytes.[10] Hepcidin production increases in response to iron overloading and inflammatory signalling, whereas iron deficiency and erythropoietic demand suppress its expression.[11,12] Consequently, hepcidin sits at the intersection of iron availability, inflammation, and erythropoiesis. The expansion of adipose tissue may disrupt this regulatory system. Severe obesity has been correlated with increased hepcidin expression in adipose tissue and enhanced interleukin-6 signalling.[13,14] Furthermore, several clinical investigations have documented elevated circulating hepcidin levels in individuals with obesity or central adiposity.[15,16,17] Chronic low-grade inflammation associated with obesity may therefore increase circulating hepcidin concentrations, reduce intestinal iron absorption, and promote functional iron deficiency despite adequate or even increased iron stores. Several studies have demonstrated altered iron metabolism in obese individuals; however, the independent contribution of body mass index to circulating hepcidin remains controversial after adjustment for other determinants of iron homeostasis.[18,19]

Among Indian women, obesity has also been associated with diminished iron absorption and an increased risk of iron deficiency.[20] Nevertheless, the findings reported in the literature are heterogeneous, partly due to differences among studies in variables such as age, sex, fat distribution, inflammatory burden, iron status, and calibration of hepcidin assays.[21,22] A cross-sectional positive relationship between hepcidin and circulating iron may additionally reflect physiological feedback mechanisms, whereas elevated inflammation-driven hepcidin is anticipated to inhibit iron egress over time.

The present study therefore evaluated the association of serum hepcidin with body mass index (BMI), haemoglobin, ferritin, TIBC, and serum iron in an Indian cohort with obesity. The primary objective was to determine whether BMI was independently associated with hepcidin. Secondary objectives were to quantify anaemia using sex-specific haemoglobin cut-offs, compare iron-related biomarkers by anaemia status, and identify independent predictors of circulating hepcidin.

MATERIALS AND METHODS

Study Design and Setting

This hospital-based cross-sectional observational study was conducted within the Department of Biochemistry, in collaboration with the Department of Internal Medicine, at a tertiary care teaching hospital in India, over a period of twelve months from January to December 2024. The research was carried out subsequent to obtaining approval from the Institutional Ethics Committee, and all procedures adhered to the ethical standards outlined in the Declaration of Helsinki. Written informed consent was obtained from all participants prior to their enrollment in the study.

Study Subject

The study included 100 adult participants who attended the outpatient or inpatient departments during the study period. Inclusion criteria for the study comprised patients with a body mass index (BMI) greater than 25, aged between 18 and 65 years, of either sex, and willing to provide consent. Exclusion criteria included patients with renal or hepatic impairment, congestive heart failure, hypothyroidism, or those taking medications that could affect iron levels.

The outcome variables included serum ferritin, serum iron, Total Iron-binding Capacity (TIBC), and serum hepcidin.

Data Collection and Clinical Assessment

A predetermined proforma was used to assess socio-demographic factors and clinical information, including age, gender, dietary habits, physical activity, comorbidities (such as diabetes, hypertension), and chronic illnesses (such as chronic liver disease, chronic kidney disease, or hypothyroidism).

Anthropometric measurements, including height, weight, and waist circumference, were recorded using standard methods. Body mass index (BMI) was calculated as weight (kg) divided by the square of height (m2). WHO BMI bands were used for descriptive classification: overweight, 25.0–29.9 kg/m²; obesity class I, 30.0–34.9 kg/m²; obesity class II, 35.0–39.9 kg/m²; and obesity class III, ≥40.0 kg/m².[3]

Laboratory Analysis

After an overnight fasting period of 8–12 hours, five mL venous blood sample was collected in a plain vial under aseptic precautions from all participants. The serum samples were separated by centrifugation at 1500 rpm for 15 minutes. All samples were stored at –20 °C for further analysis. Samples were analysed by a certified clinical laboratory for blood

chemistry, including haemoglobin, iron status markers (serum iron, serum ferritin, serum hepcidin, and total iron-binding capacity), and glycemic markers (fasting glucose, serum insulin, and glycated haemoglobin [HbA1c]).

Haemoglobin and serum Iron were estimated using the colourimetric method, while serum Ferritin levels were measured by a latex-based immunoturbidimetric method. Serum HbA1c was determined using high-performance liquid chromatography (HPLC). Fasting Plasma Glucose was measured by the glucose oxidase peroxidase method (Randox Laboratories, Crumlin, UK). Serum insulin was estimated by an immunoassay-based electrochemiluminescent technique (Cobas-Roche Diagnostics). Serum hepcidin levels were determined using a commercially available enzyme-linked immunosorbent assay (ELISA) kit.

Statistical Analysis

The data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) software version 25.0 (IBM Corp., Armonk, NY, USA). Normality was tested using the Shapiro-Wilk/Kolmogorov-Smirnov test. Continuous parametric data were expressed as mean ± standard deviation (SD), and qualitative (categorical) variables as percentages. A Student's t-test was used to compare normally distributed variables between groups. The Chi-square test was used to compare qualitative data. Bivariate associations with hepcidin were estimated using Pearson correlation coefficients. Regression analysis was used to evaluate the unadjusted association between BMI and hepcidin. The primary multivariable ordinary least-squares model included age, BMI, sex, diabetes, ferritin, serum iron, TIBC, and haemoglobin. Coefficients are reported with standard errors, 95% CIs, standardised β coefficients, and p values. A p-value of less than 0.05 was considered statistically significant. Reporting follows STROBE recommendations.[23]

RESULTS

General characteristics of the study population

Baseline anthropometric characteristics and serum biomarkers of the 100 participants who fulfilled the inclusion criteria are presented in Table 1. The study consisted of 73 females (73.0%) and 27 males (27.0%), with 48 participants (48.0%) diagnosed with diabetes. The average age was 41.41 ± 10.01 years. The mean Body Mass Index (BMI) was 45.07 ± 6.53 kg/m², ranging from 26.93 to 61.27. All participants had a BMI of at least 25 kg/m², and 78.0% presented with class III obesity. The mean hepcidin level was 22.15 ± 4.01 ng/ml.

Table 1: Baseline anthropometric characteristics and serum biomarkers (overall and by anaemia status)

Characteristic	Overall (n=100)	No anaemia (n=74)	Anaemia (n=26)	p value
Age, years	41.41 ± 10.01	41.96 ± 10.38	39.85 ± 8.85	0.322
Female sex	73 (73.0%)	51 (68.9%)	22 (84.6%)	0.121
Diabetes	48 (48.0%)	36 (48.6%)	12 (46.2%)	0.827
Weight, kg	114.19 ± 19.91	115.93 ± 19.90	109.23 ± 19.48	0.141
BMI, kg/m²	45.07 ± 6.53	45.59 ± 6.72	43.58 ± 5.81	0.150
Fasting glucose, mg/dL	111.22 ± 20.76	111.40 ± 22.31	110.69 ± 15.89	0.601
Insulin, µU/L	19.84 ± 9.14	19.49 ± 9.42	20.83 ± 8.36	0.383
HbA1c, %	6.43 ± 1.35	6.38 ± 1.35	6.57 ± 1.35	0.472
Haemoglobin, g/dL	12.83 ± 1.31	13.36 ± 1.00	11.31 ± 0.80	<0.001
Serum ferritin, ng/mL	52.98 ± 39.95	60.01 ± 38.69	32.97 ± 37.23	<0.001
TIBC, µg/dL	344.30 ± 70.01	329.06 ± 62.69	387.68 ± 72.74	<0.001
Hepcidin, ng/mL	22.15 ± 4.01	22.81 ± 4.04	20.26 ± 3.31	0.002
Serum iron, µg/dL	65.21 ± 24.32	72.43 ± 23.22	44.65 ± 13.22	<0.001

Iron deficiency status among study participants

In this study, we used one essential criterion for defining iron deficiency anaemia: haemoglobin less than 12 g/dL in females and less than 13 g/dL in males.[1] Because ferritin is also an acute-phase reactant and, in the absence of C-reactive protein or other inflammatory markers, iron deficiency was not diagnosed from ferritin alone.[24]

Anaemia was observed in 26 participants, accounting for 26.0%. The prevalence was 30.1% among females and 14.8% among males; however, the difference between sexes was not statistically significant (p=0.198; Table 2). Compared to participants without anaemia, those with the condition exhibited significantly lower levels of serum hepcidin (20.26 ± 3.31 ng/ml versus 22.81 ± 4.04 ng/mL; p=0.002), serum ferritin (32.97 ± 37.23 ng/mL versus 60.01 ± 38.69 ng/mL; p<0.001), and serum iron (44.65 ± 13.22 µg/dl versus 72.43 ± 23.22 µg/dL; p<0.001). Conversely, they demonstrated higher total iron-binding capacity (TIBC) (387.68 ± 72.74 µg/dL compared to 329.06 ± 62.69 µg/dL; p<0.001).

Table 2: Prevalence of anaemia using sex-specific haemoglobin cut-offs

Group	Anaemia, n	Total, n	Prevalence, %	95% CI, %	Sex comparison p
Overall	26	100	26.0	18.4–35.4	0.198
Female	22	73	30.1	20.8–41.4	
Male	4	27	14.8	5.9–32.5	

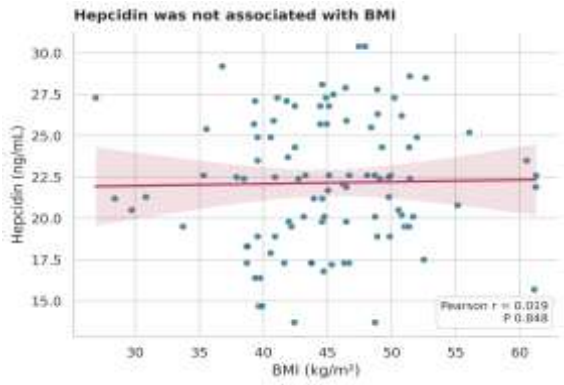

Figure 1. Association between body mass index and serum hepcidin. Points represent individual participants; the solid line is the ordinary least-squares fit, and shading is the 95% confidence interval.

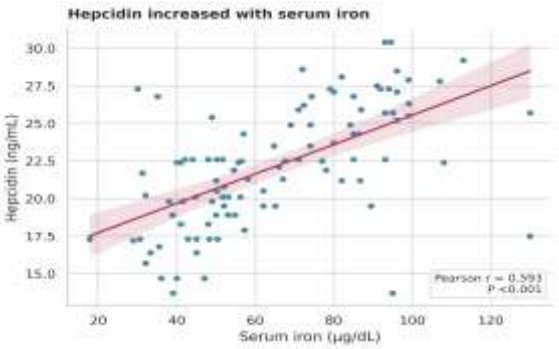

Figure 2: Association between serum iron and serum hepcidin. The line and shaded band show the linear fit and 95% confidence interval.

Relationship of serum hepcidin with BMI and Iron indices

Hepcidin was not associated with BMI by correlation (r=0.019, p=0.848; Figure 1). The simple BMI model explained <0.1% of the variability in hepcidin (B=0.012 ng/mL per kg/m²; R^2<0.001; p=0.848). Hepcidin correlated most strongly with serum iron (r=0.593, p<0.001; Figure 2). Positive correlations were also observed with haemoglobin and ferritin, while TIBC correlated inversely. [Table 3]

Table 3: Bivariate correlations of serum hepcidin with clinical and laboratory variables

Variable	r	p
Age	-0.059	0.562
BMI	0.019	0.848
Fasting glucose	-0.058	0.563
Insulin	0.064	0.524
HbA1c	-0.052	0.609
Haemoglobin	0.401	<0.001
Ferritin	0.230	0.022
TIBC	-0.299	0.002
Serum iron	0.593	<0.001

Serum iron was the only independent predictor: each 1 µg/dL higher serum iron was associated with 0.099 ng/mL higher hepcidin (95% CI 0.060–0.138; standardized β=0.602; p<0.001). BMI was not independently associated with hepcidin (B=−0.017; 95% CI −0.123 to 0.089; standardized β=−0.028; p=0.745). Age, sex, diabetes, ferritin, TIBC, and haemoglobin were also not independently associated with hepcidin. [Table 4 and Figure 3]

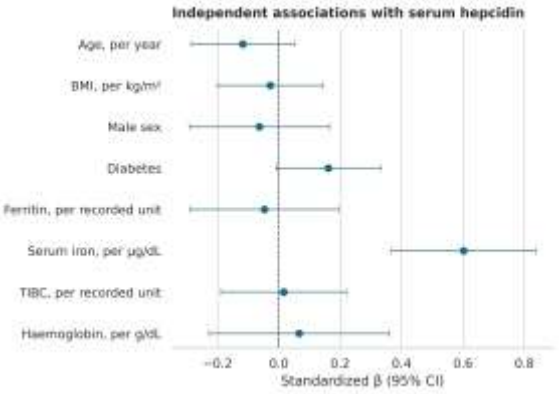

Figure 3: Standardized coefficients from the multivariable model for serum hepcidin. Points indicate standardized β coefficients and horizontal bars indicate 95% confidence intervals.

Table 4: Multivariable linear regression with serum hepcidin (ng/mL) as the outcome

Predictor	B	95% CI	Standardized β	p value
Age, per year	-0.047	-0.115 to 0.020	-0.118	0.167
BMI, per kg/m²	-0.017	-0.123 to 0.089	-0.028	0.745
Male sex	-0.570	-2.625 to 1.484	-0.063	0.583
Diabetes	1.286	-0.075 to 2.648	0.161	0.064
Ferritin, ng/mL	-0.005	-0.029 to 0.020	-0.048	0.697

Serum iron, per µg/dL	0.099	0.060 to 0.138	0.602	<0.001
TIBC, per µg/dL	0.001	-0.011 to 0.013	0.015	0.882
Haemoglobin, per g/dL	0.201	-0.698 to 1.101	0.066	0.658

DISCUSSION

In the present study, we conducted an analysis of haematological parameters (haemoglobin, serum iron, serum ferritin, serum hepcidin) and glycemic index (fasting glucose, serum insulin, and HbA1c) in 100 participants with obesity. All participants in the current study exhibited overweight or obesity, with 78% classified as having class III obesity, and BMI values ranged from 26.9 to 61.3 kg/m². Anaemia affected approximately one quarter of participants and was accompanied by a coherent iron-restricted phenotype—lower ferritin and serum iron, higher TIBC, and lower hepcidin.

Serum hepcidin was marginally elevated in individuals with class III obesity compared to other classes; however, the difference was minimal and did not show statistical significance. Furthermore, no correlation was observed between increasing body mass index (BMI) and serum hepcidin levels. Serum hepcidin demonstrated a strong association with circulating iron levels. Additionally, hepcidin showed a positive correlation with haemoglobin and ferritin levels and an inverse correlation with transferrin saturation (TIBC). The absence of an association between BMI and hepcidin contrasts with findings from studies reporting increased adipose tissue or circulating hepcidin in cases of severe obesity,[15,25,26] as well as with research linking central adiposity to elevated hepcidin levels and impaired iron homeostasis.[16] However, some investigations have documented no significant correlation between BMI and serum hepcidin concentrations, thereby supporting our findings.[27,28] This discrepancy does not inherently invalidate the potential existence of an adiposity–hepcidin pathway. Moreover, BMI alone does not account for visceral adiposity, fat distribution, or inflammatory activity within adipose tissue, which may serve as more direct determinants of hepcidin levels.

The positive cross-sectional association between serum iron and hepcidin is biologically plausible as a snapshot of iron-responsive feedback: circulating and stored iron stimulate hepatic hepcidin, which then limits further iron entry into plasma.[29] In contrast, persistently elevated inflammation-driven hepcidin can lower circulating iron over time. Population work has often identified ferritin as a major biochemical correlate of hepcidin.[21] In our adjusted model, ferritin did not remain significant after serum iron, TIBC, and haemoglobin were entered simultaneously, suggesting that these correlated markers captured overlapping components of iron physiology.

The findings are relevant to the Indian double burden of anaemia and adiposity. Obese Indian women have shown reduced iron absorption despite similar micronutrient intake,[20] and multicountry analyses demonstrate that the co-occurrence of anaemia with overweight or obesity is increasing.[30] Although the anaemia prevalence in this clinical dataset should not be generalised to the national population, the combination of severe obesity and biochemical iron restriction illustrates why ferritin, serum iron, haemoglobin, and hepcidin must be interpreted together rather than in isolation.

CONCLUSION

Serum hepcidin was strongly associated with serum iron and other iron-related indices but was not associated with BMI in this cohort with overweight or obesity. Anaemia was common and accompanied by lower hepcidin, ferritin, and serum iron and higher TIBC, although the independent association with anaemia disappeared after accounting for iron indices. These findings support integrated interpretation of hepcidin and conventional iron biomarkers and highlight the need for prospective, inflammation-aware studies spanning a wider range of adiposity.

Limitations

First, the cross-sectional design prevents causal or temporal inference.

Second, all participants had overweight or obesity and most had class III obesity, limiting the ability to detect an adiposity gradient.

Third, the sample was modest and female-predominant.

Fourth, inflammatory markers, renal and hepatic functions, and fat distribution were not assessed.

Conflict of Interest: The authors declare no conflict of interest.

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