

Original Article

Role of Serum Ferritin as an Inflammatory Marker in Non-Infectious

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ABSTRACT

Background: Serum ferritin is an indicator of iron status, but it can also be elevated in conditions of chronic inflammation, which may contribute to its association with cardiometabolic disorders. **Objective:** This study aimed to compare ferritin, inflammatory, and iron status indicators in adults with type 2 diabetes mellitus (T2DM), hypertension (HTN), both T2DM and HTN, and healthy controls. **Methods:** An analytical cross-sectional study was conducted in Karachi, Pakistan, in 2025–2026, comprising 85 adults divided into four groups: controls (n=20), T2DM only (n=22), HTN only (n=21), and T2DM with HTN (n=22). Serum ferritin, has-CRP, serum iron, total iron binding capacity (TIBC), transferrin saturation percentage (TSAT), HbA1c, blood pressure, and body mass index (BMI) were measured. One-way analysis of variance (ANOVA) with Tukey–Kramer multiple comparisons test was used to compare variables between groups and Pearson's correlation analysis was used to assess the association of serum ferritin with other variables. **Results:** Serum ferritin and has-CRP varied significantly in the four groups ($F=74.635$ and $F=62.213$; $p<0.001$), with the highest concentrations observed in the dual disease group (242.8 ± 41.5 ng/mL; 4.8 ± 1.2 mg/L). Serum iron, TIBC, and TSAT were comparable between the groups (for TSAT: $F=0.592$; $p=0.622$). Serum ferritin correlated with has-CRP ($r=0.68$; $n=220$), HbA1c ($n=221$; $n=222$), systolic blood pressure ($n=223$; $n=224$), and BMI ($n=225$; $n=226$), but not with TSAT ($n=227$; $n=228$). **Conclusion:** Serum ferritin appeared to be influenced by inflammatory and cardiometabolic conditions to a greater extent than TSAT and should therefore be interpreted in conjunction with these variables. **Keywords:** Ferritin; C-Reactive Protein; Type 2 Diabetes Mellitus; Hypertension; Inflammation; Iron Status.

INTRODUCTION

Ferritin is the main protein that stores iron inside cells and protects them by keeping the iron in a soluble and bioavailable form that is less toxic. Normally, the serum ferritin levels reflect the amount of iron in the body, so the indicator can be used to assess iron deficiency and overload. However, it is also an acute-phase reactant that

can be elevated due to inflammation, cell damage, metabolic disorders, and liver disease independent of iron levels (1-3). Therefore, high ferritin concentrations should not be considered in isolation but in the context of other markers of iron status and inflammation such as serum iron, total iron-binding capacity, and transferrin saturation (2-4).

Type 2 diabetes mellitus (T2DM) and essential hypertension are two cardiometabolic diseases associated with chronic low-grade inflammation and derangements in metabolic and vascular homeostasis. Dysregulated inflammatory pathways and iron metabolism can contribute to the development of insulin resistance, endothelial dysfunction, oxidative stress, and cardiovascular disorders, whereas these conditions can reciprocally affect serum ferritin levels (5). Cross-sectional studies in patients with T2DM have revealed increased serum ferritin and high sensitivity C-reactive protein (hs-CRP) concentrations and a positive association of these inflammatory markers with glycated hemoglobin and dyslipidemia (6,7). Similarly, in hypertensive populations, serum ferritin has been linked to inflammatory status, glucose tolerance and, arterial stiffness, reflecting the complex interplay between cardiometabolic and vascular dysfunction (8).

The interpretation of ferritin measurements becomes further complicated by the strong interconnections between obesity, metabolic syndrome, hepatic steatosis, inflammation, and abnormal iron metabolism. Studies of metabolic syndrome and non-alcoholic fatty liver disease have demonstrated that increased ferritin concentrations can occur in the presence of largely normal or only mildly deranged transferrin saturation, but some individuals may also have hepatic iron overload (9-11). Thus, even in the setting of apparently normal transferrin saturation, normal ferritin levels may reduce the likelihood of classical systemic iron overload, but they cannot independently confirm that an elevated serum ferritin is exclusively inflammatory. The clinical interpretation of ferritin measurements requires careful consideration of the inflammatory status, iron indices, metabolic and liver factors, and other potential causes of increased serum ferritin concentrations (3,4,10,11).

This overlap presents an important clinical dilemma in patients with diabetes and hypertension. Interpreting an isolated ferritin elevation as iron overload could lead to additional testing or food recommendations that are unnecessary while attributing it purely to inflammation may miss important abnormalities of hepatic and iron metabolism. Much of the evidence cited relates to these different diseases separately, so comparison of these patient populations with healthy controls, patients with only T2DM and patients with only essential hypertension while using concurrent measures of inflammation and iron may help make this more meaningful clinically. This cross-sectional study aimed to determine if serum ferritin was more closely associated with indices of inflammation and cardiometabolic health or with transferrin saturation across these four cohorts and to explore the cross-sectional relationships of serum ferritin with indices of inflammation, glyCAked hemoglobin, Systolic BP, BMI and transferrin saturation.

MATERIALS AND METHODS

The cross-sectional analysis was carried out from August 2025 through to January 2026 in Karachi, Pakistan. The study was designed as a cross-sectional comparison of serum ferritin and a set of inflammatory markers and iron status indicators. The primary outcome measure was serum ferritin concentrations across the sample study groups. The secondary outcome measure was markers of inflammation, iron deficiency, and iron status indices in relation to serum ferritin concentrations. The study sample comprises a convenience sample population of 85 individuals with reference to comparable observational biomarker studies as well as feasibility of recruitment of study sample within the study period. The sample consists of four mutually exclusive groups, controls, individuals with T2DM, individuals with essential hypertension, and individuals with T2DM and hypertension. The study population was adults from Karachi aged 30-65 years.

Inclusion and exclusion criteria include the following: eligible participants in the disease groups (T2DM and/or essential hypertension) must have had a clinical diagnosis of T2DM or essential hypertension, and/or both conditions for six or more months; control participants must not have any of these conditions; and all participants should have had a confirmed clinical diagnosis that can be established through review of the clinical records provided by each participant. Exclusion criteria are those who had an active acute or chronic infection; a diagnosis of liver cirrhosis; stage 3 or more advanced stage of chronic kidney disease; evidence of presence of any other diagnosed disease; any known malignancy or hematological disorder; any autoimmune disorders such as

rheumatoid arthritis; any other diagnosed disease, known stage 3 or above chronic kidney disease; autoimmune disease such as rheumatoid arthritis; previous blood transfusion within the preceding 6 months; use of any oral or intravenous iron supplements; and any inflammatory disease.

Data collection included a clinical interview and physical examination, review of clinical record of any established diagnoses and collection of fasting venous blood samples. Variables collected included demographic data, age, sex, body mass index, fasting blood glucose and glycated hemoglobin levels, and systolic and diastolic blood pressure. Blood pressure was recorded using calibrated automated blood pressure monitors, according to best clinical practice and guidelines.

After 8 hours of fasting, venous blood samples for the measurement of specific markers of inflammation and iron status were obtained to control for time-dependent variables.

Measurement of biomarkers Serum ferritin was the primary outcome measure and was determined using a chemiluminescent immunoassay.

TSAT was derived by dividing serum iron by TIBC then multiplying the resultant ratio by 100.

HbA1c was assessed using high-performance liquid chromatography and fasting blood glucose was measured on the fasting blood sample. Clinical group was the primary exposure variable and serum ferritin, hs-CRP, serum iron, TIBC and TSAT were the main laboratory outcomes. HbA1c, systolic blood pressure, body mass index and TSAT were also assessed in relation to serum ferritin. The use of a consistent fasting duration, standardised sample collection, calibrated blood-pressure instruments, simultaneous measurement of inflammation and iron-related biomarkers, and the exclusion of major alternate causes of ferritin elevation sought to reduce measurement variability and allow comparison of the four groups.

The potential for metabolic and iron-related confounding was adjusted for using multivariable analysis involving hs-CRP, HbA1c, systolic blood pressure, body mass index and TSAT.

The analysis was carried out on the 85 study participants with complete clinical and laboratory data and no imputation of missing outcome data was required.

Data analysis was performed using the IBM SPSS Statistics version 26.0 statistical software. Continuous variables were assessed for normality using the Shapiro-Wilk test, normally distributed variables were summarized as mean ($\pm$ standard deviation), and log transformed skewed variables would have been summarized as median (interquartile range). Categorical variables are presented as number (percentage). The baseline characteristics of serum ferritin, hs-CRP, serum iron, TIBC and TSAT and the baseline continuous variables were compared by one-way analysis of variance for the 4 groups; if an overall test was significant, pairwise comparisons were made using Tukey's post-hoc test. The sex distribution between the 4 groups was compared using the Pearson chi-square test.

Pearson correlations were used to investigate the relationships between serum ferritin, hs-CRP, HbA1c, systolic blood pressure, body mass index and TSAT and reports with 95% confidence intervals. Multivariable linear regression models investigated the independently associated baseline relationships between serum ferritin, sex, hs-CRP, HbA1c, systolic blood pressure, body mass index and TSAT; the dependent variable was serum ferritin. The model was estimated by calculating unstandardized coefficients, standard errors, standardized beta coefficients, t-statistics, and the corresponding p values for each independent variable. Any relationships were statistically significant at $p < 0.05$.

Prior to participant recruitment, institutional ethics committee approval was obtained. Participants were fully informed regarding study procedures and consented in writing before assessment and blood withdrawal.

RESULTS

Among 92 subjects screened, 85 of them completed all the clinical and laboratory tests and were enrolled into this study: 20 normal control, 22 T2DM, 21 hypertension, and 22 T2DM combined with hypertension (92.4% of the screened samples). 20 normal control, 22 T2DM, 21 hypertension, and 22 T2DM combined with hypertension, respectively. In our samples, 45 of participants were male (52.9%) and 40 were female (47.1%). The age and sex were

not significantly different in the four groups, but body mass index, blood pressure, fasting blood glucose and HbA1c were significantly different among the four groups (Table 1).

Table 1. Demographic and Clinical Characteristics by Study Group (N=85)

Characteristic	Health controls (n=20)	T2DM alone (n=22)	Hypertension alone (n=21)	T2DM + hypertension (n=22)	Test statistic	p-value	Effect size
Age, years	47.1 ± 7.5	50.2 ± 8.1	50.8 ± 8.9	50.1 ± 8.4	F=0.826	0.483	$\eta^2=0.030$
Male, n (%)	11 (55.0)	12 (54.5)	11 (52.4)	11 (50.0)	$\chi^2=0.136$	0.987	V=0.040
Female, n (%)	9 (45.0)	10 (45.5)	10 (47.6)	11 (50.0)	—	—	—
BMI, kg/m ²	23.8 ± 2.1	28.5 ± 3.1	28.1 ± 3.0	29.9 ± 3.4	F=16.313	<0.001	$\eta^2=0.377$
Systolic BP, mmHg	116.5 ± 7.2	124.8 ± 8.1	145.2 ± 11.3	148.6 ± 12.0	F=52.060	<0.001	$\eta^2=0.658$
Diastolic BP, mmHg	74.2 ± 5.1	79.5 ± 6.2	91.4 ± 7.5	92.1 ± 8.3	F=34.452	<0.001	$\eta^2=0.561$
Fasting blood glucose, mg/dL	88.5 ± 8.1	152.4 ± 24.6	94.2 ± 9.8	161.8 ± 28.3	F=76.688	<0.001	$\eta^2=0.740$
HbA1c, %	5.2 ± 0.3	8.1 ± 1.1	5.4 ± 0.4	8.5 ± 1.2	F=85.989	<0.001	$\eta^2=0.761$

Data are expressed as mean SD unless otherwise specified. Comparison among groups for continuous variables was performed with the one-factor analysis of variance, sex with the Pearson chi-square test. η^2 =eta-squared, V=Cramer's V, BMI=body mass index, BP=blood pressure, HbA1c=glycated hemoglobin, T2DM=type 2 diabetes mellitus. Serum ferritin was higher in the 4 groups with omnibus effect sizes of $\eta^2=0.734$ for ferritin and 0.697 for hs-CRP.

This mean ferritin has been got 82.1 22.4 ng/mL in controls compared with 242.8 41.5 ng/mL in the combined T2DM and hypertension group.

Mean hs-CRP grew from 1.1 0.4 mg/L in controls to 4.8 1.2 mg/L in the combined disease group. Quite not difference was There in serum iron, TIBC, or TSAT across the 4 groups (Table 2).

Table 2. Serum Ferritin, hs-CRP, and Iron-Status Measures by Study Group

Laboratory measure	Healthy controls (n=20)	T2DM alone (n=22)	Hypertension alone (n=21)	T2DM + hypertension (n=22)	F-statistic	p-value	η^2
Serum ferritin, ng/mL	82.1 ± 22.4	198.6 ± 38.9	185.3 ± 36.2	242.8 ± 41.5	74.635	<0.001	0.734
hs-CRP, mg/L	1.1 ± 0.4	3.4 ± 0.9	3.1 ± 0.8	4.8 ± 1.2	62.213	<0.001	0.697
Serum iron, µg/dL	88.5 ± 18.2	92.1 ± 20.4	86.9 ± 19.1	90.4 ± 21.0	0.281	0.839	0.010
TIBC, µg/dL	320.5 ± 35.1	315.2 ± 38.4	324.1 ± 32.8	318.9 ± 36.5	0.228	0.876	0.008
TSAT, %	27.6 ± 5.2	29.1 ± 6.1	26.8 ± 5.8	28.3 ± 6.4	0.592	0.622	0.021

Values are expressed as the mean SD. Statistics were calculated by one-way analysis of variance, based on the number of individuals in each group, means and SDs. Hs-CRP, high-sensitivity C-reactive protein; TIBC, total iron-binding capacity; TSAT, transferrin saturation; T2DM, type 2 diabetes mellitus; η^2 , eta-squared.

Tukey-Kramer assessments said that ferritin was above average in for each disease group than in healthy controls; mean differences were 116.5 ng/mL for T2DM, 103.2 ng/mL for hypertension alone, and 160.7 ng/mL for T2DM combined with hypertension.

Ferritin levels were alike in the T2DM-alone and hypertension-alone sets but differed from each of the other groups. A similar pattern of pairwise assessments was reflected for hs-CRP (Table 3).

Table 3. Tukey-Kramer Pairwise Comparisons for Serum Ferritin and hs-CRP

Outcome	Comparison	Mean difference	Simultaneous 95% CI	Adjusted p-value	Hedges' g	95% CI for g
Ferritin, ng/mL	T2DM – controls	116.5	87.5 to 145.5	<0.001	3.56	2.58 to 4.54
Ferritin, ng/mL	Hypertension – controls	103.2	73.9 to 132.5	<0.001	3.34	2.39 to 4.30
Ferritin, ng/mL	T2DM + hypertension – controls	160.7	131.7 to 189.7	<0.001	4.66	3.48 to 5.85
Ferritin, ng/mL	Hypertension – T2DM	–13.3	–41.9 to 15.3	0.617	–0.35	–0.94 to 0.24
Ferritin, ng/mL	T2DM + hypertension – T2DM	44.2	15.9 to 72.5	<0.001	1.08	0.45 to 1.70
Ferritin, ng/mL	T2DM + hypertension – hypertension	57.5	28.9 to 86.1	<0.001	1.45	0.78 to 2.11
hs-CRP, mg/L	T2DM – controls	2.30	1.58 to 3.02	<0.001	3.19	2.27 to 4.10
hs-CRP, mg/L	Hypertension – controls	2.00	1.28 to 2.72	<0.001	3.08	2.17 to 3.99
hs-CRP, mg/L	T2DM + hypertension – controls	3.70	2.98 to 4.42	<0.001	3.98	2.92 to 5.04
hs-CRP, mg/L	Hypertension – T2DM	–0.30	–1.01 to 0.41	0.682	–0.35	–0.94 to 0.25

Outcome	Comparison	Mean difference	Simultaneous 95% CI	Adjusted p-value	Hedges' g	95% CI for g
hs-CRP, mg/L	T2DM + hypertension – T2DM	1.40	0.70 to 2.10	<0.001	1.30	0.65 to 1.94
hs-CRP, mg/L	T2DM + hypertension – hypertension	1.70	0.99 to 2.41	<0.001	1.63	0.94 to 2.31

Mean differences and simultaneous 95% confidence intervals were calculated using the Tukey–Kramer procedure. Positive values favour the first group named in each comparison. Hedges' g represents the standardized mean difference. hs-CRP, high-sensitivity C-reactive protein; T2DM, type 2 diabetes mellitus; CI, confidence interval.

Serum ferritin was really related with hs-CRP ($r=0.68$, 95% CI 0.55 to 0.78; $p<0.001$). Good co-relations were also known amongst ferritin and HbA1c, systolic blood pressure, and bmi. The co-rrrelation between ferritin and TSAT was small and did not get statistical significance ($r=0.09$, 95% CI -0.13 to 0.30 ; $p=0.415$) (Table 4).

Table 4. Crude Correlations of Serum Ferritin With Inflammatory and Cardiometabolic Measures (N=85)

Correlate	Pearson's r	95% CI	p-value
hs-CRP, mg/L	0.68	0.55 to 0.78	<0.001
HbA1c, %	0.45	0.26 to 0.60	<0.001
Systolic BP, mmHg	0.41	0.22 to 0.57	<0.001
BMI, kg/m ²	0.38	0.18 to 0.55	<0.001
TSAT, %	0.09	-0.13 to 0.30	0.415

BMI, body mass index; BP, blood pressure; CI, confidence interval; HbA1c, glycated hemoglobin; hs-CRP, high-sensitivity C-reactive protein; TSAT, transferrin saturation.

DISCUSSION

The principal finding of this study was that levels of serum ferritin and serum hs-CRP varied by study group, with levels being the greatest in the combined T2DM/hypertension group. Levels of serum iron, TIBC and TSAT did not vary by study group. Serum ferritin was positively correlated with serum hs-CRP, HbA1c, BMI and systolic blood pressure, and was not significantly correlated with serum TSAT.

The higher ferritin and hs-CRP concentrations in the two disease groups are consistent with previous reports of positive relationships among ferritin, hs-CRP, HbA1c, and other cardiometabolic abnormalities in people with T2DM (6,7,12). This extends this body of work by illustrating this association between levels of ferritin using a four-way comparison between people without any chronic disease, T2DM alone, hypertension alone, and T2DM/hypertension combined. The T2DM-only pattern suggests that one or both diseases in isolation did not drive the pattern of greater ferritin concentrations, but it may be in the T2DM/hypertension group that greater concentrations may be due to the additive impact of the two disease states on cardiovascular and metabolic health. Similar results were observed among hypertensive cohorts with ferritin concentrations being related to inflammation, vascular health, and glucose status (8).

It is, however, not possible to comment on the independent contribution of combined disease due to a lack of control for confounders such as disease duration, treatment, adiposity, and glycemia.

Several biological mechanisms could potentially account for the observed positive correlation between ferritin and hs-CRP: ferritin is both an intracellular iron-storage protein and a positive acute-phase reactant, and activation of inflammatory cytokines could upregulate hepatic ferritin synthesis and alter iron homeostasis via the acute-phase response, macrophage iron retention, and regulation of the hormone hepcidin (1,3,5,13). Chronic adiposity, vascular dysfunction, and insulin resistance may therefore similarly upregulate serum ferritin, without elevating TSAT. The significant group difference in BMI may be particularly relevant in this context, as adiposity itself could contribute to both hs-CRP and serum ferritin concentrations (14).

The null finding of no group differences in the circulating iron indices of serum iron, TIBC, and TSAT was somewhat reassuring that the group differences in ferritin were not accompanied by corresponding increases or decreases in the traditional circulating indices of iron status. Nevertheless, it cannot rule out excess tissue iron, which is characterized by evidence of human metabolic syndrome and fatty liver disease that may be as common as 30% in healthy populations, 20,33 despite normal or minimally altered TSAT (9–11,14). The study did not measure any of the inflammatory hepatic indices, the concentration of hepatic iron, hepcidin, soluble transferrin receptor, liver enzymes, or causes of iron overload of any kind. Therefore, the report can demonstrate only a more robust crude

association of serum ferritin with the inflammation marker, hs-CRP than with TSAT, but cannot determine whether it indicated inflammation alone or a confluence of inflammatory, hepatic, metabolic, and iron overload processes.

This is an important addition to the literature that underpins the rationale for any investigation of ferritin in patients with diabetes and hypertension. Ferritin should be considered in the context of TSAT, serum iron, TIBC, relevant markers of inflammation, tests of hematological health, clues from the liver, potential etiology, and, when available, sex-specific reference intervals (2-4). Normal TSAT may lessen the suspicion of classical systemic iron overload, but this is by no means synonymous with the absence of any all too common disorder of iron accumulation. Similarly, an elevated ferritin should not be taken as a clear indication of iron overload when an inflammatory or other metabolic abnormality is present. The evaluation of suspected hereditary hemochromatosis also employs a comprehensive review of biochemistry, clinical hints and symptoms, and, when flashed, solutions for liver iron and gene mutations rather than depend on ferritin alone (15). As the study did not tell analytical thresholds or clinical conclusions, it should not be used to state whether a analytical cut point should be used or whether an probe for iron overload should go on.

Some strengths of this work were the simultaneous assessment of four groups with distinct clinical diagnoses, and the measure of ferritin, hs-CRP, and traditional iron status biomarkers in parallel. Collecting blood in fasting state, and excluding many infectious, autoimmune, renal, hepatic, haematological, neoplastic, transfusion-related, and supplement-related causes of ferritin increase, increased comparability. Inclusion of both diabetes and hypertension groups enabled recognition of the biomarker pattern in combined disease separate from that observed in either disease.

Several limitations are notable.

Cross-sectional studies do not allow for evaluation of temporality or causality. The small sample size of recruits from one city, with 20-22 subjects per group, reduced precision and generalizability. The context of recruitment and details of sampling procedures were insufficient for assessment of selection bias.

Menopausal status and sex separately affect ferritin and may alter pattern of biomarker correlations but were neither stratified nor controlled for in the adjusted analysis.

Potential confounders for ferritin analysis were not examined, including alcohol consumption, tobacco and drug use, anemia, nonmetabolic liver damage, and abnormal lipids.

There was large variation among the groups in mean values for body mass index, blood glucose, and blood pressure; crude pooled correlation coefficients among these variables and serum ferritin may partly reflect the separation of diseases into diagnostic groups. The reported multivariable model could not be validated because the outcome has a very similar construction to the descriptive tables and the regression coefficients bear little relation to the estimates presented; in addition, absent the assay-specific reference ranges, the proportion of participants with elevated serum ferritin, and lacking direct measures of hepatic or molecular iron, definitively can be attributed to inflammation and distinguish from iron overload, remain unknown. Larger multicentre longitudinal studies adjusting for prespecified confounders and including accurate measures of liver and iron status are needed to determine the nature and direction of independent and temporally relative associations among ferritin, inflammation, and cardiometabolic disease.

CONCLUSION

Serum ferritin and hs-CRP levels were higher in this cross-sectional sample of patients with T2DM, hypertension or both conditions compared to healthy controls, with highest levels observed in the combined condition group, whereas serum iron, TIBC and TSAT levels did not differ significantly. Ferritin was more closely associated with hs-CRP and the cardiometabolic parameters than with TSAT, which lends support to the notion of using ferritin as an indicator of inflammatory and metabolic excess in these patients. These associations neither imply a causal relationship nor exclude the presence of tissue iron excess, and hence support the use of ferritin in conjunction with markers of inflammation, blood cell production, the liver and iron levels.

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