RESEARCH PAPER

Association Between Serum Ferritin And Glycated Hemoglobin (Hba1c) In Type 2 Diabetes Mellitus: A Cross-Sectional Study

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Abstract

Background: Type 2 diabetes mellitus (t2dm) is associated with dysregulated iron metabolism. Elevated serum ferritin is a marker of body iron stores and inflammation and has been implicated in the pathogenesis of insulin resistance and glycemic dysfunction. Decoding the relationship between serum ferritin and glycated hemoglobin (hba1c) is expected to provide insights into metabolic disturbances in t2dm and identify potential therapeutic targets.

Objectives: To evaluate the association between serum ferritin levels and hba1c in patients with t2dm and to determine whether serum ferritin can help as an adjunct marker of glycemic control.

Materials And Methods: This cross-sectional study was conducted among 108 t2dm patients attending the department of medicine, sharda hospital, greater noida over 18 months. Patients with acute infections, chronic inflammatory conditions, liver or kidney disease and iron deficiency anemia were excluded. Serum ferritin was estimated using chemiluminescent immunoassay and hba1c by high-performance liquid chromatography (hplc). Statistical analysis included independent t-test and pearson correlation using spss version 22.0.

Results: Among 108 participants (52.78% males, mean age 51.99±11.56 years), mean serum ferritin was 538.16±79.15 ng/ml and mean hba1c was 9.47±2.19%. Elevated ferritin (>300 ng/ml for males, >200 ng/ml for females) was present in 57.41% of patients. Mean hba1c was significantly higher in patients with elevated ferritin (9.92±2.05%) compared to those with normal ferritin (8.30±1.57%; $p<0.01$). Pearson correlation analysis demonstrated a significant positive correlation between serum ferritin and hba1c ($r=0.52$, $p<0.01$).

Conclusion: A significant positive correlation exists between serum ferritin and hba1c in t2dm patients, suggesting that elevated body iron stores are associated with poorer glycemic control. Serum ferritin may serve as an accessible adjunct biomarker for assessing metabolic status in diabetes management.

Keywords: Type 2 Diabetes Mellitus, Serum Ferritin, Glycated Hemoglobin, Hba1c, Iron Metabolism, Glycemic Control, Oxidative Stress.

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INTRODUCTION

Diabetes mellitus (DM) is a major global health challenge. The International Diabetes Federation reported 415 million affected individuals worldwide which is projected to rise to 642 million by 2040.[1] India accounts for the second largest diabetic population globally with 69.1 million affected individuals and this number continues to rise due to urbanization, sedentary lifestyles and dietary changes.[2] Type 2 diabetes mellitus (T2DM) accounts for approximately 90-95% of all diabetes cases and is characterized by insulin resistance and progressive β-cell dysfunction.[3]

The pathophysiology of T2DM involves complex interactions between genetic predisposition, environmental factors and metabolic dysregulation. Emerging evidence suggests that iron metabolism plays a significant role in glucose homeostasis and diabetes pathogenesis.[4] Iron, while essential for numerous biological processes, can be detrimental

when present in excess due to its ability to catalyze the formation of reactive oxygen species (ROS) through Fenton chemistry.[5] This oxidative stress contributes to pancreatic β-cell damage, hepatic insulin resistance and the development of diabetic complications.[6]

Serum ferritin serves as the primary clinical marker of body iron stores and shows intracellular iron content.[7] Beyond its role in iron storage, ferritin is also an acute-phase reactant that increases in inflammatory states. Multiple epidemiological studies have demonstrated elevated serum ferritin levels in patients with T2DM compared to non-diabetic controls and prospective studies have identified elevated ferritin as a predictor of incident diabetes.[8] A meta-analysis by Kunutsor et al. reported that individuals in the highest quintile of ferritin levels had a 73% increased risk of developing T2DM compared to those in the lowest quintile.[9]

Glycated hemoglobin (HbA1c) shows average plasma glucose concentration over the preceding 8-12 weeks and serves as the cornerstone of diabetes monitoring.[10] The relationship between serum ferritin and HbA1c is of particular clinical interest because both markers provide integrated measures of metabolic status - ferritin showing iron homeostasis and chronic inflammation and HbA1c showing glycemic control. Several mechanisms may link these parameters, including glycation-induced prolongation of ferritin half-life, hyperglycemia-mediated oxidative stress promoting iron release and inflammation-driven alterations in iron trafficking.[11]

While studies from various geographical settings have examined the ferritin-HbA1c relationship, data from the Indian subcontinent remain limited despite the high diabetes burden. Understanding this association in Indian populations is important given potential ethnic differences in iron metabolism, dietary patterns and diabetes phenotypes. Hence the present study was undertaken to evaluate the association between serum ferritin levels and HbA1c in patients with T2DM attending a tertiary care centre in North India.

MATERIALS AND METHODS

Study Design and Setting

This cross-sectional observational study was conducted in the Department of Medicine at School of Medical Science and Research, Sharda University, Greater Noida, Uttar Pradesh, India. The study was carried out over a period of 18 months after obtaining approval from the Institutional Ethics Committee. Written informed consent was obtained from all participants prior to enlistment. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Sample Size Calculation

The sample size was calculated using Cochran's formula: $n = z^2pq/d^2$, where $z = 1.96$ (standard normal variate at 95% confidence interval), $p = 11.4\%$ (expected prevalence of elevated ferritin in T2DM based on prior literature), $q = 1-p$ and $d = 6\%$ (margin of error). The calculated minimum sample size was 108 patients. For detection of a correlation coefficient of $r=0.30$ with 80% power and $\alpha=0.05$, a minimum of 85 subjects was required; our sample exceeded this requirement.

Inclusion Criteria

Patients aged more than 35 years, of either gender, with diagnosed T2DM according to American Diabetes Association (ADA) 2023 criteria (HbA1c ≥6.5% or fasting plasma glucose ≥126 mg/dL or 2-hour plasma glucose ≥200 mg/dL during oral glucose tolerance test or random plasma glucose ≥200 mg/dL with classic symptoms of hyperglycemia) who provided informed consent were included in the study.[12]

Exclusion Criteria

Confounding by non-diabetic causes of elevated ferritin was minimized by excluding the following conditions: Type 1 diabetes mellitus, hypertension, acute or chronic infections, chronic inflammatory conditions (systemic lupus erythematosus, rheumatoid arthritis, inflammatory bowel disease), chronic liver disease, chronic or acute kidney disease, iron deficiency anemia, hemochromatosis or known iron overload disorders, recent blood transfusion (within 3 months), thyroid disorders, chronic alcoholism, malignancy, pregnancy and those unwilling to participate. All participants had undergone detailed clinical history and physical examination to screen for occult inflammatory or infectious conditions prior to enlistment.

Data Collection and Clinical Assessment

A detailed history including demographic details, duration of diabetes, symptoms and past medical history was obtained from each participant. Complete physical examination including general and systemic examination was performed. Vital parameters were recorded and fundoscopy was performed to assess for diabetic retinopathy.

Laboratory Investigations

Blood samples were collected under aseptic precautions after overnight fasting (8-10 hours). All samples were processed within 2 hours of collection to minimize pre-analytical variability. The following investigations were performed:

Glycated Hemoglobin (HbA1c): Estimated by high-performance liquid chromatography (HPLC) using the BIORAD D10 system, standardized against the International Federation of Clinical Chemistry (IFCC) reference method and traceable to the Diabetes Control and Complications Trial (DCCT)/National Glycohemoglobin Standardization Program (NGSP).

Serum Ferritin: Measured using chemiluminescent immunoassay (CLIA) on the Roche COBAS 6000 analyzer. The assay has an analytical sensitivity of 0.5 ng/ml with intra-assay coefficient of variation (CV) <5% and inter-assay CV <7%. Elevated ferritin was defined as >300 ng/ml for males and >200 ng/ml for females – this is consistent with WHO guidelines for iron excess.

Other Investigations: Fasting blood sugar (FBS) and postprandial blood sugar (PPBS) were analyzed using electrochemiluminescence immunoassay (ECLIA) on the Roche COBAS 6000 analyzer. Complete blood count including hemoglobin, RBC count, MCV, MCH and MCHC was performed to exclude iron deficiency anemia. Serum iron, total iron binding capacity (TIBC) and transferrin saturation were also measured.

Statistical Analysis

Data was collected and then input into Microsoft Excel and analysis was done using SPSS version 22.0. Normality of distribution was assessed using the Shapiro-Wilk test. Independent samples t-test was used to compare means between groups. Chi-square test was applied for categorical variables. Pearson correlation coefficient was calculated to assess the linear relationship between serum ferritin and HbA1c. A p-value <.05 was considered statistically significant.

RESULTS

Demographic and Clinical Characteristics

A total of 108 patients with T2DM were enrolled in the study. Of these, 57 (52.78%) were males and 51 (47.22%) were females. Most subjects belonged to the age group 51-60 years (35.19%), followed by 61-70 years (25.93%). The mean age of the study population was 51.99±11.56 years. The mean HbA1c was 9.47±2.19%. This indicated overall poor glycemic control in the study population. The baseline demographic, clinical and biochemical characteristics are presented in Table 1.

Table 1: Baseline Demographic, Clinical and Biochemical Characteristics of Study Subjects (n=108)

Parameter	N / Mean	% / SD
Gender		
Male	57	52.78
Female	51	47.22
Age Groups (years)		
35-40	23	21.30
41-50	27	25.00
51-60	38	35.19
61-70	20	18.52
Mean Age (years)	51.99	±11.56
Glycemic Parameters		
HbA1c (%)	9.47	±2.19
FBS (mg/dL)	235.06	±94.07
PPBS (mg/dL)	282.02	±118.13
Iron Parameters		
Serum Ferritin (ng/ml)	538.16	±79.15
TIBC (µg/dL)	260.02	±101.05
Transferrin Saturation (%)	27.56	±10.45
Hematological Parameters		
Hemoglobin (g/dL)	12.0	±2.1
RBC (million/mm³)	4.4	±0.6

FBS: Fasting Blood Sugar; PPBS: Postprandial Blood Sugar; TIBC: Total Iron Binding Capacity; RBC: Red Blood Cells

Distribution of Serum Ferritin Levels

Serum ferritin ranged from 350.2 to 780.5 ng/ml with a mean value of 538.16±79.15 ng/ml. Using gender-specific cutoffs (>300 ng/ml for males, >200 ng/ml for females), elevated ferritin was present in 62 (57.41%) patients, while 46 (42.59%) had normal ferritin levels. The distribution of serum ferritin among the study population is presented in Table 2.

Table 2: Distribution of Serum Ferritin Levels among Study Subjects (n=108)

Serum Ferritin Status	N	%
Elevated (High)	62	57.41
Normal	46	42.59
Total	**108**	**100**

Elevated ferritin defined as >300 ng/ml for males and >200 ng/ml for females

Comparison of HbA1c Levels According to Serum Ferritin Status

Mean HbA1c was significantly higher in patients with elevated serum ferritin (9.92±2.05%) compared to those with normal ferritin levels (8.30±1.57%). This difference was statistically significant (p<0.01). The comparison is presented in Table 3 and illustrated in Figure 1.

Table 3: Comparison of HbA1c Levels According to Serum Ferritin Status

Serum Ferritin	Mean HbA1c (%)	SD	t-value	p-value
Elevated (n=62)	9.92	2.05	4.52	<0.01*
Normal (n=46)	8.30	1.57		

**Statistically significant (p<0.05); Independent samples t-test*

Correlation between Serum Ferritin and HbA1c

Pearson correlation analysis showed a significant positive correlation between serum ferritin and HbA1c (r=0.52, p<0.01). This results as a moderate positive linear relationship, suggesting that as serum ferritin levels increase, HbA1c levels also tend to increase. The correlation analysis is presented in Table 4 and illustrated in Figure 2. Additionally, serum ferritin showed significant positive correlations with fasting blood sugar (r=0.44, p<0.01) and postprandial blood sugar (r=0.49, p<0.01).

Table 4: Correlation of Serum Ferritin with Glycemic Parameters

Parameter	r-value	p-value
Serum Ferritin vs HbA1c	+0.52	<0.01*
Serum Ferritin vs FBS	+0.44	<0.01*
Serum Ferritin vs PPBS	+0.49	<0.01*

**Statistically significant (p<0.05); Pearson correlation coefficient; FBS: Fasting Blood Sugar; PPBS: Postprandial Blood Sugar*

DISCUSSION

Our present study demonstrates a significant positive correlation between serum ferritin levels and HbA1c in patients with type 2 diabetes mellitus (r=0.52, p<0.01). Mean HbA1c was significantly higher in patients with elevated ferritin (9.92±2.05%) compared to those with normal ferritin levels (8.30±1.57%, p<0.01). These findings suggest that elevated body iron stores, as showed by serum ferritin are associated with poorer glycemic control in T2DM.

Our finding of a moderate positive correlation (r=0.52) between ferritin and HbA1c is consistent with several studies from diverse geographical settings. Bayih et al. (2024) in an Ethiopian cohort reported a similar correlation (r=0.457, p<0.001) and demonstrated significantly elevated ferritin in uncontrolled T2DM patients (243.05±91.77 ng/ml) compared to controlled patients (169.30±89.98 ng/ml).[13] Rawat et al. (2016) from India reported a comparable correlation coefficient (r=0.582, p<0.01) with mean ferritin of 271.40±47.76 ng/ml.[14] Son NE (2019) in a Turkish population similarly observed that serum ferritin levels significantly increased with increasing HbA1c levels (p<0.01), with a strong positive correlation between ferritin and both HbA1c and fasting blood glucose.[15]

The consistency of our correlation coefficient with these studies, despite differences in absolute ferritin values, suggests that the ferritin-HbA1c relationship is robust across populations. Gandhi et al. (2018) from India also reported a significant positive correlation between HbA1c and serum ferritin which led them to conclude that higher HbA1c is associated with elevated ferritin and vice versa.[16] In their 2021 case-control study: Al-Miraj and Khan found that serum ferritin levels were significantly higher in patients with type 2 diabetes mellitus and were positively correlated with HbA1c; which they interpreted as a relationship between increased iron stores and poorer glycemic control.[17]

However, not all studies have demonstrated this association. A recent study from Al-Azhar University, Egypt (2025) found no significant correlation between ferritin and HbA1c in diabetic patients, despite ferritin being higher in diabetics than controls.[18] Similarly, a Delhi-based study (2025) involving 351 T2DM patients reported no significant association between ferritin and HbA1c (p=0.26), although ferritin was associated with microvascular complications.[19] These discrepancies can be attributed to differences in glycemic control status of study populations, exclusion criteria or the presence of confounding inflammatory conditions. Notably, studies with predominantly well-controlled diabetics (HbA1c <7.5%) may not demonstrate the ferritin-HbA1c correlation that becomes apparent in poorly controlled populations such as ours (mean HbA1c 9.47%).

The mean serum ferritin in our study (538.16±79.15 ng/ml) was higher than values reported in several comparable studies, which ranged from 170 to 380 ng/ml.[13-17] Several factors may explain this observation. (1) our study population had notably poor glycemic control (mean HbA1c 9.47±2.19%), higher

than many comparison studies. Since ferritin increases with worsening glycemic status - the central finding of our study - higher ferritin values are expected in poorly controlled populations. (2) our stringent exclusion of hypertension, chronic kidney disease and liver disease may have selected for patients with metabolically distinct diabetes phenotypes. (3) regional variations in dietary iron intake and genetic factors influencing iron metabolism in the North Indian population may contribute.[20] Importantly, despite the higher absolute values, the correlation coefficient (r=0.52) was within the range reported globally (r=0.25-0.58) and this suggests that the underlying ferritin-HbA1c relationship is consistent.

The biological plausibility of the ferritin-HbA1c association is well-established. Chronic hyperglycemia promotes non-enzymatic glycation of hemoglobin and other proteins, including ferritin itself. Carenini et al. (1985) demonstrated that glycosylated ferritin has a prolonged serum half-life which leads to elevated circulating levels in poorly controlled diabetes.[21] Additionally, hyperglycemia induces oxidative stress through multiple pathways including advanced glycation end-product (AGE) formation and protein kinase C activation. Iron, through Fenton chemistry, catalyzes the generation of reactive oxygen species, further amplifying oxidative damage to pancreatic β-cells and perpetuating insulin resistance.[22]

Conversely, elevated iron stores may contribute to diabetes pathogenesis through a bidirectional relationship. Iron accumulation in hepatocytes impairs insulin signaling and promotes hepatic gluconeogenesis, while iron deposition in pancreatic β-cells induces apoptosis through oxidative mechanisms.[23] This creates a vicious cycle: hyperglycemia elevates ferritin and elevated iron worsens glycemic control. The significant correlations we observed between ferritin and both fasting and postprandial glucose (r=0.44 and r=0.49, respectively) support this bidirectional relationship.

These findings have practical clinical implications. Serum ferritin, a widely available and inexpensive test, may serve as an adjunct marker for assessing glycemic control and identifying patients at risk for complications. Patients with elevated ferritin despite adequate iron intake should be evaluated for suboptimal glycemic control. Furthermore, therapeutic strategies targeting iron reduction - such as dietary modification, phlebotomy in selected cases or iron chelation - have shown promise in improving insulin sensitivity and glycemic control in small trials.[24] However, larger randomized controlled trials are needed before such interventions can be recommended routinely.

Our study has several strengths. We employed standardized laboratory methods using automated analyzers with documented quality control measures. Stringent exclusion criteria were applied to minimize confounding by inflammatory conditions, liver disease, kidney disease and iron deficiency anemia - all of which can independently affect ferritin levels. The sample size was adequate for correlation analysis and the consistency of our correlation coefficient with global literature supports external validity.

We point out some of the limitations to be considered when interpreting these findings. (A) the cross-sectional design precludes causal inference; whether elevated ferritin precedes or follows glycemic deterioration cannot be determined from this study. (B) we did not measure inflammatory markers such as C-reactive protein or interleukin-6, which may influence both ferritin and glycemic status. (C), duration of diabetes was not systematically recorded, limiting our ability to assess its confounding effect. (D) the single-centre design in a tertiary referral setting may limit generalizability to primary care populations where patients may have better glycemic control. (E) we did not assess dietary iron intake - which could have provided additional context for the elevated ferritin values observed.

Future research should focus on prospective cohort studies to establish temporal relationships between ferritin changes and glycemic trajectories. Intervention studies evaluating the effect of iron reduction strategies on glycemic control in diabetic patients with elevated ferritin would be valuable. Additionally, mechanism / pathway based studies which explore the linkages between ferritin, inflammation and insulin could help in the identification of new therapeutic targets.

CONCLUSION

The present study demonstrates a significant positive correlation between serum ferritin levels and HbA1c in patients with type 2 diabetes mellitus. Patients with elevated ferritin exhibited significantly higher HbA1c levels compared to those with normal ferritin. This indicated that elevated body iron stores are associated with poorer glycemic control. These findings support the role of iron metabolism in diabetes pathophysiology and suggest that serum ferritin may serve as an accessible adjunct biomarker for assessing metabolic status in diabetes management. The consistency of our correlation coefficient with global

literature, despite higher absolute ferritin values in our population, underscores the robustness of this relationship. Longitudinal studies are warranted to establish causal relationships and evaluate the potential of iron-targeted interventions in improving glycemic outcomes.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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FIGURE LEGENDS

Figure 1: Comparison of mean HbA1c levels between patients with elevated and normal serum ferritin levels. Bar chart showing significantly higher mean HbA1c levels in patients with elevated ferritin (9.92±2.05%) compared to those with normal ferritin (8.30±1.57%). Error bars represent standard deviation. *p<0.01 indicates statistically significant difference (independent samples t-test).

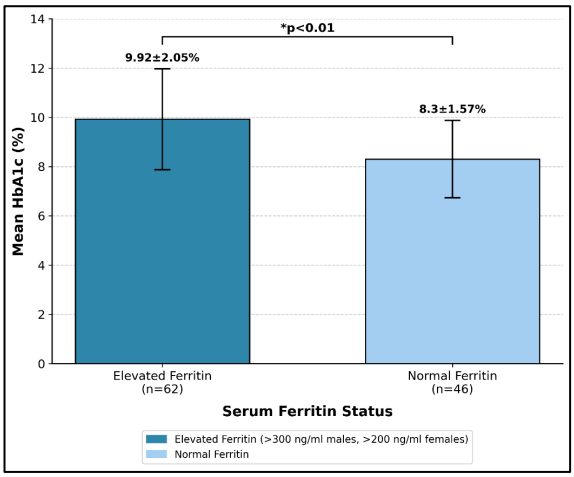

Figure 2: Scatter plot for the correlation between serum ferritin levels and HbA1c in type 2 diabetes mellitus patients. Significant positive correlation was observed (Pearson correlation coefficient r=0.52, p<0.01). The dashed line is to show the linear regression fit. Each point pertains to an individual patient.