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Serum ferritin and glycemic control (HbA1c) in type 2 diabetes mellitus: A cross-sectional study

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Abstract
Background: Insulin resistance and increasing β-cell dysfunction are hallmarks of type 2 diabetes mellitus, a chronic metabolic disease. There is growing evidence that the onset and course of type 2 diabetes may be influenced by iron metabolism, specifically serum ferritin levels. Measuring serum ferritin and iron levels in T2DM patients compared to non-diabetic controls and examining the relationship between these levels and glycemic control (HbA1c) were the goals of this study.
Methods: At Al-Sader City Hospital in Iraq, 100 participants 50 T2DM patients and 50 controls took part in a cross-sectional study from March to July 2025. Samples of venous blood were drawn during a fast. Serum ferritin and iron levels were assessed using standard laboratory methods, and HbA1c was determined using EDTA samples. The data was examined using Pearson's correlation and Student's t-test; $p<0.05$ was considered significant.
Results: Patients with diabetes had significantly higher serum ferritin levels than controls (65.67±10.40 ng/mL vs. 34.11±6.31 ng/mL, $p<0.001$). Similarly, HbA1c levels were considerably higher in the diabetes group (7.87±1.74%) compared to the control group (4.56±1.49%, $p<0.001$). Correlation analysis revealed no significant relationship between serum ferritin and HbA1c in either the control group ($r = 0.22$, $p = 0.38$) or diabetes patients ($r = -0.17$, $p = 0.49$). In conclusion, blood ferritin and HbA1c levels were significantly greater in diabetic patients than in non-diabetic individuals. Serum ferritin does not, however, significantly correlate with HbA1c, indicating that higher ferritin levels in diabetes may be more indicative of metabolic or inflammatory changes than of direct glycemic management.

Keywords: Ferritin, iron, HbA1c, diabetes mellitus, Iraq

Introduction
Diabetes mellitus (DM) represents a persistent metabolic disorder characterized by chronic hyperglycemia arising from impairments in insulin secretion, insulin action, or both [1] . Among its various types, type 2 diabetes mellitus (T2DM) is the most prevalent form, primarily resulting from insulin resistance coupled with progressive pancreatic β-cell dysfunction [2] Unlike type 1 diabetes, which is largely autoimmune in nature, T2DM arises from a complex interplay between genetic predisposition and environmental factors, including obesity, sedentary lifestyle, and dietary habits [3]. This combination causes persistent hyperglycemia and a slow deterioration in β-cell function, which can lead to long-term issues with the neurological, renal, and cardiovascular systems. [4]. T2DM development is influenced by multiple genetic loci, including TCF7L2 and KCNJ11, which modulate glucose metabolism and insulin secretion [5]. However, lifestyle-related risk factors such as excessive caloric intake and physical inactivity play a central role in its increasing prevalence worldwide [6]. Compensatory hyperinsulinemia is first brought on by insulin resistance, especially in skeletal muscle, adipose tissue, and the liver. Persistent metabolic stress eventually damages β-cell function, resulting in chronic hyperglycemia and inadequate insulin production. [7]. Ferritin, an iron-binding protein, plays a crucial role in maintaining systemic iron homeostasis by storing and releasing iron in a controlled manner [8, 9]. Ferritin and iron metabolism dysregulation has been linked to oxidative stress and inflammation, which are known to worsen insulin resistance and β-cell dysfunction. People who are more likely to acquire type 2 diabetes have higher blood ferritin levels, which may

indicate that iron excess plays a part in the pathogenesis of the disease. [10]. Given the increasing prevalence of type 2 diabetes and its aftereffects worldwide, it is nevertheless essential to comprehend both modifiable and non-modifiable risk factors. Analyzing the relationship between ferritin levels and type 2 diabetes may reveal information on early diagnosis methods and preventative strategies. Clarifying the link between iron metabolism, insulin resistance, and pancreatic β-cell activity may produce a helpful biomarker for identifying high-risk individuals and devising specific treatments to slow the course and effects of the disease [3, 7]. In conclusion, type 2 diabetes is a complicated illness that is impacted by genetic, environmental, and metabolic factors. The pathophysiology of type 2 diabetes may involve ferritin and iron homeostasis, which highlights the need for comprehensive study to clarify their molecular relationships, improve risk assessment, and direct preventive and therapeutic approaches for this increasingly prevalent metabolic disease [11, 9].

Methods

Study Design and Subjects

This study was conducted at Al-Sader City Hospital in Iraq between March and July of 2025. The 100 participants were divided into 50 patients with type 2 diabetes mellitus (T2DM) and 50 non-diabetic controls. All subjects were matched for age and gender and had HbA1c values more than 6.2% in order to reduce confounding variables. Each participant provided informed consent prior to sample collection.

Blood Sample Collection

Venous blood samples (5 mL) were drawn from each participant between 8:00 and 10:00 a.m. after an overnight fast. The blood was divided as follows: Use 2 mL in EDTA tubes to estimate HbA1c. After serum separation, 3 mL are placed in gel tubes for ferritin testing. Gel tubes were allowed to coagulate for half an hour at room temperature before being centrifuged for five minutes at 3000 rpm. The separated serum was placed in 1.5 mL Eppendorf tubes and stored at -20 °C before analysis. EDTA tubes were processed immediately for HbA1c testing.

Serum Ferritin Measurement

Ferritin levels were measured using enzyme-linked immunosorbent assay (ELISA) kits according to the manufacturer's protocol. All samples were analyzed in duplicate to ensure accuracy.

Statistical Analysis

Data were expressed as mean±standard deviation (SD). Comparisons between diabetic and control groups were performed using Student's t-test. Correlations between ferritin levels and HbA1c were assessed using Pearson's correlation coefficient. A p-value ≤ 0.05 was considered statistically significant. Statistical analyses were performed using SPSS version 21.0 [12].

Results

Table 1: Serum ferritin (ng/ml) distribution between studied groups.

Serum Ferritin (ng/ml)	Diabetic (n=50)	Control (n=50)	p-value
Mean ± SD (ng/mL)	65.67±10.40	34.11±6.305	<0.001
Standard Error of Mean	3.94	1.85	
Median (ng/mL)	66.50	33.00	
Minimum (ng/mL)	54	32	
Maximum (ng/mL)	80	43	

*T-test for two independent means is significant at the 0.05

Table 2: Serum HbA1c (%) distribution between studied groups.

Serum HbA1c (%))	Diabetic (n=50)	Control (n=50)	p-value
Mean±SD (ng/mL)	7.87± 1.74	4.56± 1.49	<0.001
Standard Error of Mean	1.23	1.05	
Median (ng/mL)	7.80	4.50	
Minimum (ng/mL)	7	3	
Maximum (ng/mL)	9	7	

*T-test for two independent means is significant at the 0.05

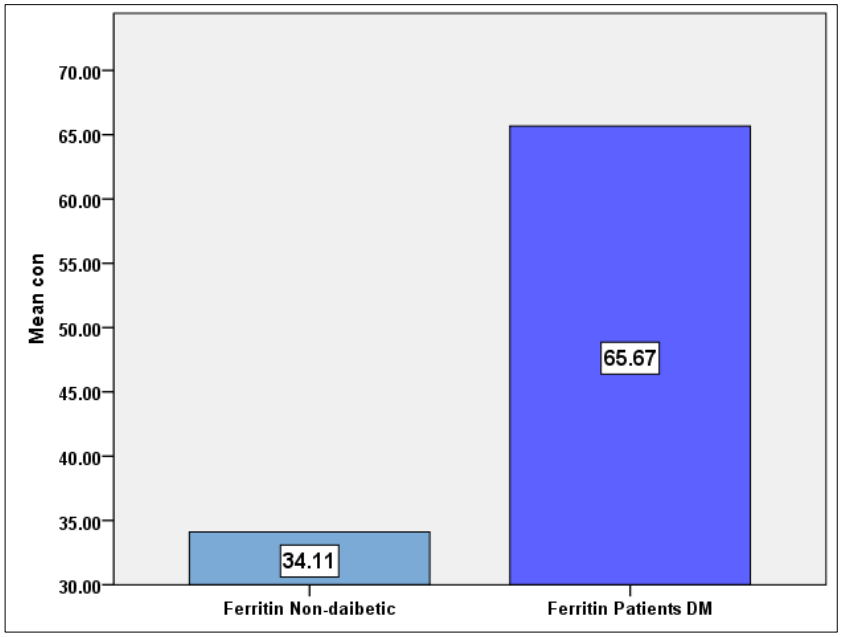

Fig 1: Mean serum ferritin level (ng/ml) in the two different studied groups

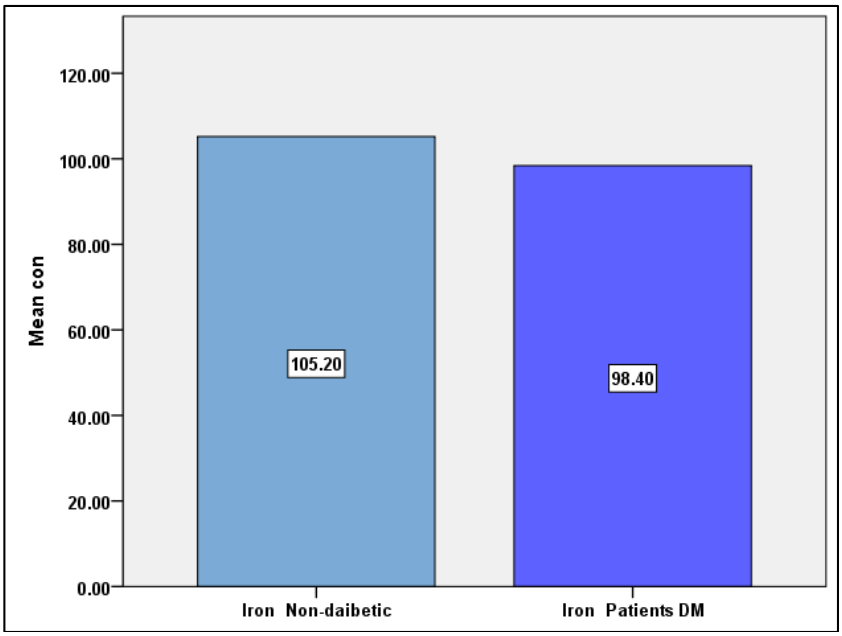

Fig 2: Mean serum Iron level (µg /dl)) in the two different studied groups

Table 3: Correlation between Ferritin and HbA1c in Patients DM with Non-daibetic group.

Group	r (Pearson)	P-value	Interpretation
Diabetic	−0.17	0.49	Weak, not significant
Control	+0.22	0.38	Weak, not significant

Discussion

This study examined serum ferritin and HbA1c levels in diabetic patients in comparison with non-diabetic individuals, with particular attention to the possible relationship between iron status and glycemic control. The results demonstrated clear differences between the two groups, whereas no statistically significant association was observed between ferritin and HbA1c within each group.

Serum ferritin concentrations were significantly higher in diabetic patients than in the control group. In addition to serving as an acute-phase reactant that rises in response to inflammatory circumstances, ferritin is commonly considered an indication of body iron reserves [13] Diabetes mellitus frequently causes chronic low-grade inflammation and metabolic stress, which may raise ferritin levels in those who have the condition [14]. Furthermore, excess iron has been reported to promote insulin resistance through oxidative stress and impaired insulin signaling pathways [15]. Concurrently, the HbA1c values of the diabetic group were significantly higher, suggesting that these patients had inadequate long-term glycemic management. HbA1c is still crucial for identifying and monitoring diabetes mellitus because it is a trustworthy measure of average blood glucose levels during the preceding two to three months [16]. Although both values were significantly higher in diabetic patients, correlation analysis did not reveal a meaningful relationship between ferritin and HbA1c. The diabetic group displayed a weak negative correlation, whereas the control group displayed a faint positive correlation. Similar findings from earlier studies indicate that ferritin rise in diabetes does not necessarily correlate with glycemic control as determined by HbA1c. [17, 18]. The lack of a significant correlation may be explained by the multifactorial regulation of ferritin levels. Factors such as inflammatory status, liver function, obesity, and subclinical infections may influence ferritin concentrations independently of glucose metabolism [19]. Therefore, ferritin may be more suggestive of inflammatory or metabolic stress than it is of long-term glycaemic control. Overall, the findings demonstrate that diabetic individuals have significantly higher serum ferritin and HbA1c levels than non-diabetic controls. However, the lack of a significant correlation between these two markers suggests that iron metabolism alterations in diabetes may occur independently of glycaemic control. Further studies utilizing inflammatory markers and larger sample sizes are required to fully comprehend this connection.

Conclusion

The current investigation revealed significantly higher serum ferritin and HbA1c levels in diabetic patients compared to non-diabetic controls, indicating altered iron metabolism and poor glycaemic control in individuals with diabetes mellitus. Despite these considerable differences between the two groups, there was no statistically significant correlation between serum ferritin and HbA1c in either the diabetes or non-diabetic patients. These findings suggest that elevated ferritin levels in diabetics may be a sign of underlying inflammatory or metabolic conditions rather of being directly related to long-term glycaemic control. Consequently, serum ferritin cannot be regarded as a trustworthy measure for glycaemic management as determined by HbA1c, but it may be a sign of metabolic stress or inflammation in diabetes mellitus. Further studies with larger sample sizes and inclusion of inflammatory markers are recommended to better clarify the complex relationship between iron metabolism and diabetes mellitus.

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