İstanbul Med J 2026; 27(2): 136-42
DOI: 10.4274/imj.galenos.2026.59265

The Relationship Between Iron Stores, Type 2 Diabetes, and Diabetic Complications

Muhammet Ateş, Tayyibe Saler

University of Health Sciences Türkiye, Adana City Training and Research Hospital, Clinic of Internal Medicine, Adana, Türkiye

ABSTRACT

Introduction: The present investigation sought to examine the relationship of circulating ferritin concentrations with type 2 diabetes mellitus (T2DM) and with diabetes-related complications. Additionally, we aimed to determine whether serum ferritin could serve as a biomarker of metabolic and inflammatory status in individuals with diabetes.

Methods: This retrospective observational study included 422 subjects: 195 with T2DM, 82 with insulin resistance (IR), and 145 metabolically healthy controls. Demographic characteristics and biochemical variables were evaluated. Intergroup differences were analyzed using analysis of variance. Correlation analyses and receiver operating characteristic (ROC) curve assessments were performed to determine diagnostic accuracy. Furthermore, multivariable logistic regression was conducted to identify independent factors associated with diabetic complications.

Results: Participants with T2DM had significantly greater mean age and body mass index than control subjects ($p<0.001$). Serum ferritin concentrations were significantly increased in both the T2DM and IR groups relative to controls ($p<0.001$). Ferritin levels were positively associated with HbA1c (r: 0.179, $p=0.012$) and age (r: 0.133, $p=0.006$). In contrast, inverse relationships were observed between ferritin and both diabetes duration (r: −0.192, $p=0.007$) and initiation of insulin treatment (r: −0.243, $p=0.007$). The ROC curve analysis indicated that ferritin demonstrated fair discriminatory performance in identifying T2DM (area under the curve: 0.725, $p<0.001$). Elevated ferritin concentrations were observed in patients with diabetic neuropathy, retinopathy, and nephropathy compared with controls ($p<0.001$); however, no statistically significant differences were detected among the specific complication subtypes ($p=0.111$). In multivariate analysis, diabetes duration emerged as the sole independent determinant of diabetic complications ($p=0.046$).

Conclusion: Serum ferritin is significantly elevated in T2DM and correlates with poor glycemic control. Although ferritin reflects metabolic and inflammatory activity, diabetes duration remains the strongest predictor of diabetic complications.

Keywords: Type 2 diabetes mellitus, ferritin, insulin resistance, diabetic complications

Introduction

Type 2 diabetes mellitus (T2DM) represents a multifactorial metabolic disease defined by impaired insulin sensitivity, gradual deterioration of pancreatic ß-cell function, and persistent hyperglycemia (1). The global prevalence of T2DM has risen dramatically in recent decades, driven by changes in lifestyle, diet, and an aging population (2,3). Persistent hyperglycemia in diabetic individuals contributes to a wide range of complications, including cardiovascular disease, nephropathy, neuropathy, and retinopathy (4-6). Understanding the metabolic and biochemical pathways that contribute to the onset and progression of T2DM is therefore of major clinical and public health importance.

Iron metabolism has been increasingly recognized as a potential factor influencing glucose homeostasis and insulin sensitivity (7). Iron, while essential for oxygen transport and cellular energy metabolism, can exert toxic effects in excess through the generation of reactive oxygen species (8). Elevated body iron stores have been linked to oxidative stress, lipid peroxidation, and tissue damage—all of which may impair insulin action and pancreatic ß-cell function (9). Conversely, low iron levels may disrupt mitochondrial energy balance and metabolic regulation, suggesting that both iron overload and deficiency contribute to metabolic disorders.

Multiple investigations have reported a significant relationship between circulating ferritin levels—commonly regarded as an indirect indicator of total body iron stores—and the likelihood of developing T2DM (10-12). Elevated ferritin concentrations have consistently been documented in individuals with diabetes and are linked to insulin resistance (IR), systemic inflammatory activity, and features of the metabolic syndrome (MetS) (13). Moreover, abnormalities in iron

Address for Correspondence: Muhammet Ateş, MD, University of Health Sciences Türkiye, Adana City Training and Research Hospital, Clinic of Internal Medicine, Adana, Türkiye
E-mail: m_ates0@hotmail.com **ORCID ID:** orcid.org/0009-0009-8807-6010

Cite this article as: Ateş M, Saler T. The relationship between iron stores, type 2 diabetes, and diabetic complications. İstanbul Med J. 2026; 27(2): 136-42

Received: 19.02.2026
Accepted: 01.04.2026
Publication Date: 12.05.2026

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homeostasis are thought to contribute to the development of diabetes-related complications, primarily via mechanisms involving endothelial dysfunction, increased oxidative stress, and microvascular impairment.

Beyond its function as an iron-storage protein, ferritin serves as an acute-phase reactant and may reflect the underlying low-grade inflammation frequently observed in metabolic disorders. Chronic subclinical inflammation is a recognized component of IR and type 2 diabetes, contributing to endothelial dysfunction and progressive microvascular injury. In this context, elevated ferritin concentrations may represent not only altered iron balance but also an integrated signal of inflammatory and metabolic stress. Distinguishing whether ferritin acts as a causal mediator or merely as a surrogate biomarker in the diabetic milieu remains an area of ongoing investigation, underscoring the need for further clinical and mechanistic studies.

The present study aims to investigate the relationship between iron stores and the presence of type 2 diabetes and its associated complications. By evaluating serum ferritin and related iron parameters in diabetic and non-diabetic individuals, this study seeks to clarify the potential role of iron metabolism in the pathophysiology of diabetes and its complications. A better understanding of these interactions may contribute to identifying novel biomarkers and therapeutic targets for preventing or mitigating diabetic complications.

Methods

Study Design and Population

This retrospective, observational study was conducted at the internal medicine outpatient clinics and inpatient wards of the Adana City Training And Research Hospital. Ethical approval was obtained from the Non-Interventional Clinical Research Ethics Committee of Çukurova University (meeting number: 66, date: July 7, 2017). Due to the retrospective nature of the study, the requirement for informed consent was waived by the ethics committee.

Medical records of adult patients (≥18 years) diagnosed with T2DM and/or IR between January 1, 2015 and July 1, 2017 were reviewed through Adana City Training and Research Hospital's electronic medical database. The study population included 422 participants: 195 patients with T2DM (123 receiving insulin and 72 receiving oral antidiabetic drugs), 82 individuals with IR, and 145 healthy controls who presented to the internal medicine clinics for routine check-ups.

Data Collection

Demographic characteristics and laboratory parameters were extracted from institutional medical records for all participants. The recorded variables included body mass index (BMI), fasting plasma glucose, glycated hemoglobin A1c (HbA1c), serum ferritin, low-density lipoprotein cholesterol, triglycerides, and serum creatinine levels. In individuals with diabetes, the presence of chronic microvascular complications—specifically retinopathy, nephropathy, and neuropathy—was documented, excluding diabetic foot involvement. BMI was computed as body weight in kilograms divided by the square of height measured in meters (kg/m^2). IR was quantified using the Homeostasis Model Assessment for IR (HOMA-IR), calculated using the following equation: HOMA-IR = (fasting insulin×fasting plasma glucose)/405. A threshold value of ≥2.5 was defined as indicative of IR. Serum ferritin concentrations were measured using an electrochemiluminescence immunoassay method with the Elecsys Ferritin assay kit (Roche Diagnostics, Mannheim, Germany) on the Elecsys 2010 analyzer platform. The established laboratory reference intervals were 30–400 ng/mL for males and 13–150 ng/mL for females.

Inclusion and Exclusion Criteria

This study included adult patients aged 18 years or older who had been diagnosed with T2DM, IR, or both and whose medical records were fully accessible in Adana City Training and Research Hospital's electronic database. Healthy individuals presenting to the internal medicine outpatient clinics for routine health examinations during the same period and with no known systemic or metabolic disorders were included as the control group. Patients were excluded from the study if they were younger than 18 years of age, were pregnant, or had incomplete or missing clinical data. Individuals with anemia (defined as hemoglobin <13 g/dL in men and <12 g/dL in women), with genetic disorders of iron metabolism (such as hemochromatosis or hemosiderosis), or with a history of iron supplementation or blood transfusion were also excluded. In addition, patients with chronic systemic diseases known to affect ferritin levels—such as chronic kidney disease, rheumatologic disorders, or malignancy—were not included. Those with diabetic foot or other active infectious diseases were likewise excluded from the analysis.

Statistical Analysis

Data were analyzed using SPSS software (version 27.0; IBM Corp., Chicago, IL, USA). Continuous variables are presented as mean ± standard deviation, whereas categorical data are summarized as counts and percentages. Normality of the distribution was assessed using the Kolmogorov–Smirnov test. For comparisons between two independent groups, the Student's t-test was applied when parametric assumptions were satisfied, and the Mann–Whitney U test was used for non-normally distributed variables. Differences among multiple groups were examined using one-way analysis of variance, followed by appropriate post hoc analyses when overall significance was observed. Equality of variances was evaluated with Levene's test, and corresponding F values were reported. Associations between continuous parameters, including serum ferritin, HbA1c, and diabetes duration, were examined using Pearson correlation analysis. Correlation strength was classified as weak (0.1–0.3), moderate (0.3–0.5), or strong (>0.5). All analyses were conducted using two-sided tests, and p values below 0.05 were considered statistically significant.

In addition, binary logistic regression analysis was performed to identify independent factors associated with diabetic complications and to estimate effect size using odds ratios (ORs) with 95% confidence intervals (CIs). Receiver operating characteristic (ROC) curve analysis was also conducted to evaluate the discriminatory performance of serum ferritin for predicting the presence of T2DM, and the area under the curve (AUC), sensitivity, specificity, and optimal cut-off value were calculated.

Results

The diabetes cohort was significantly older than the control group (55.8±11.6 vs. 39.2±15.5 years, p<0.001). BMI was likewise greater among patients with diabetes (30.5±5.3 kg/m^2) compared with controls (28.5±4.3 kg/m^2) (p<0.001). Mean serum ferritin concentrations were markedly higher in the diabetic group (96.7±82.5 ng/mL) than in the control group (45.5±33.7 ng/mL) (p<0.001). In contrast, the sex distribution did not differ significantly between groups (p=0.587) (Table 1).

Patients with diabetes were older than both the control and IR groups (55.8±11.6 vs. 39.2±15.5 and 39.9±12.3 years, respectively; p<0.001). The highest BMI values were observed in the IR group (33.4±6.2 kg/m^2), whereas the lowest values were recorded in controls (28.5±4.3 kg/m^2) (p<0.001). Ferritin concentrations were elevated in both the diabetes (96.7±82.5 ng/mL) and IR groups (87.6±66.4 ng/mL) compared with the control group (45.5±33.7 ng/mL), with statistical significance (p<0.001). The sex distribution did not differ among the three groups (p=0.622) (Table 2).

Serum ferritin demonstrated a weak but statistically significant positive association with age (r: 0.133, p=0.006). In contrast, no meaningful relationship was detected between ferritin and BMI (r: 0.010, p=0.842). An inverse correlation was identified between ferritin levels and diabetes duration (r: −0.192, p=0.007), and between ferritin levels and transition to insulin treatment (r: −0.243, p=0.007). Additionally, ferritin showed a modest positive correlation with HbA1c concentrations (r: 0.179, p=0.012) (Table 3).

Although ferritin levels were lower in patients with complications (88.1±66.8 ng/mL) compared to those without complications (100.5±88.5 ng/mL), the difference was not statistically significant (p=0.612). Similarly, the presence of nephropathy (p=0.309), retinopathy (p=0.937), or neuropathy (p=0.571) did not result in significant changes in ferritin levels. No significant differences in serum ferritin levels were observed either when all complications were evaluated together (p=0.583) or among patients with two or more complications (p=0.602) (Table 4).

The AUC for serum ferritin was 0.725, indicating that ferritin has moderate discriminative ability for predicting type 2 diabetes (p<0.001). Using a cut-off value of 48.5 ng/mL, the sensitivity and specificity were 72% and 70%, respectively. The 95% CI for the analysis was 0.672–0.778 (Table 5, Figure 1).

Among the variables included in the analysis, only the duration of type 2 diabetes showed a statistically significant association with the presence of complications (B: 0.121, p=0.046, OR: 1.129, 95% CI: 1.002–1.272). No significant associations were observed between complications and sex, age, BMI, transition to insulin therapy, serum ferritin levels or HbA1c levels (p>0.05) (Table 6).

Serum ferritin levels were significantly higher in patients with neuropathy (87.1±68.6 ng/mL), retinopathy (76.6±83.8 ng/mL), and nephropathy (110.2±75.1 ng/mL) compared to the control group (45.4±33.6 ng/mL) (p<0.001). However, when all complication types were evaluated together, no statistically significant differences were observed among them (p=0.111) (Table 7).

Discussion

In this study, serum ferritin levels were significantly higher in individuals with T2DM than in both the control and IR groups. The higher mean age and BMI observed in the diabetic group indicate a greater metabolic burden and a more pronounced systemic inflammatory state within this population. The positive association between ferritin and HbA1c, together with its negative relationships with diabetes duration and transition to insulin therapy, suggests that ferritin may act as a dynamic biomarker, reflecting acute metabolic activity and glycemic control, rather than as a static indicator of iron stores. Although ferritin levels tended to be higher in patients with diabetic complications, no

Table 1. Comparison of demographic and laboratory characteristics between control and diabetes groups

		Control (n=145)	Diabetes (n=195)	
Sex	Female	79 (41.4%)	112 (58.6%)	0.587
	Male	66 (44.3%)	83 (55.7%)	
Age (years)		39.2±15.5	55.8±11.6	**<0.001**
BMI (kg/m²)		28.5±4.3	30.5±5.3	**<0.001**
Serum ferritin (ng/mL)		45.5±33.7	96.7±82.5	**<0.001**

Values are expressed as mean ± standard deviation. BMI: Body mass index

Table 2. Comparison of demographic and laboratory characteristics among control, insulin resistance, and diabetes groups

		Control (n=145)	Insuline resistance (n=82)	Diabetes (n=195)	p value
Sex	Female	79 (33.9%)	42 (18.0%)	112 (48.1%)	0.622
	Male	66 (34.9%)	40 (21.2%)	83 (43.9%)	
Age (years)		39.2±15.5	39.9±12.3	55.8±11.6	**<0.001**
BMI (kg/m²)		28.5±4.3	33.4±6.2	30.5±5.3	**<0.001**
Serum Ferritin (ng/mL)		45.5±33.7	87.6±66.4	96.7±82.5	**<0.001**

Values are expressed as mean±standard deviation. Statistical significance post-hoc analysis. Age: control vs. diabetes: p<0.001, insulin resistance vs. diabetes: p<0.001. BMI: Control vs. insulin resistance: p<0.001, control vs. diabetes: p<0.01, insulin resistance vs. diabetes: p<0.001. Serum ferritin: Control vs. insulin resistance: p<0.001; control vs. diabetes: p<0.001. BMI: Body mass index

significant differences were observed across complication types. This pattern implies that ferritin may represent a marker of metabolic and inflammatory stress rather than directly mirroring the extent of diabetes-related organ damage. Collectively, these findings highlight the complex interplay between iron metabolism and glucose regulation and suggest that ferritin could serve as an indirect indicator of both metabolic control and systemic inflammation in patients with T2DM.

In the present analysis, serum ferritin concentrations were significantly higher in individuals with type 2 diabetes and IR compared with healthy controls. ROC curve analysis demonstrated that ferritin has moderate diagnostic performance in identifying T2DM (AUC: 0.725). At a threshold of 48.5 ng/mL, sensitivity was 72% and specificity was 70%.

Table 3. Correlations between serum ferritin levels and clinical parameters

		Serum ferritin (ng/mL)
Age (years)	r	0.133
	p	**0.006**
BMI (kg/m²)	r	0.010
	p	0.842
Duration of type 2 diabetes (years)	r	-0.192
	p	**0.007**
Transition to insulin therapy	r	-0.243
	p	**0.007**
Hemoglobin A1c (%)	Correlation coefficient (r)	0.179
	p value	**0.012**

r: Pearson correlation coefficient, BMI: Body mass index

Table 4. Comparison of serum ferritin levels according to the presence and type of complications in the diabetic population

		Mean ± SD	p value
Presence of complication	No	100.5±88.5	0.612
	Yes	88.1±66.8	
Diabetic nephropathy	No	95.5±83.4	0.309
	Yes	106.0±76.0	
Diabetic retinopathy	No	97.3±84.6	0.937
	Yes	93.9±72.5	
Diabetic neuropathy	No	99.1±85.6	0.571
	Yes	87.1±68.6	
All complication	No	97.7±83.5	0.583
	Yes	71.9±47.2	
2 or more complications	No	97.6±83.4	0.602
	Yes	85.1±71.2	

Values are expressed as mean ± standard deviation (ng/mL). SD: Standard deviation

These findings align with prior cross-sectional evidence indicating elevated ferritin levels in poorly controlled T2D and a parallel increase in ferritin with worsening glycemic status. Consistent with this, Bayih et al. (12) reported significantly greater ferritin levels in uncontrolled diabetic patients relative to both controlled diabetics and non-diabetic subjects, along with a positive association between ferritin and HbA1c. Genetic evidence from Mendelian randomization analyses further supports a causal contribution of iron status to T2DM risk. Wang et al. (14) demonstrated that genetically elevated systemic iron indices—including serum iron, ferritin, and transferrin saturation—were positively associated with T2DM risk. Similarly, a recent meta-analysis linking iron overload with MetS and metabolic dysfunction–associated steatotic liver disease established a dose–response relationship, highlighting ferritin as a validated biomarker of metabolic derangement, with a stronger effect observed among women (15). In line with these findings, observational and mechanistic studies have shown that ferritin rises in parallel with IR markers and inversely with HDL cholesterol, supporting the directionality of our results in both T2DM and IR populations (13).

In our dataset, ferritin showed a significant positive correlation with HbA1c and negative associations with diabetes duration and transition to insulin therapy. The positive relationship with HbA1c reinforces the concept that ferritin may reflect long-term glycemic burden, a finding consistent with the study by Bayih et al. (12), who reported a moderate-to-strong ferritin–HbA1c correlation among individuals with T2DM. Moreover, the link between ferritin and the IR phenotype has been confirmed on a population scale using the MetS-IR index; a large NHANES-based analysis identified an independent and linear association between ferritin and MetS-IR, particularly among women (16). The absence of a significant relationship between ferritin

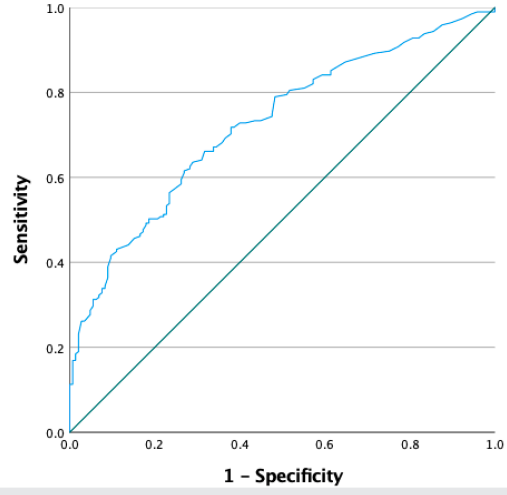

Figure 1. Receiver operating characteristic curve of serum ferritin levels for predicting type 2 diabetes

Table 5. ROC analysis for serum ferritin in predicting the presence of type 2 diabetes

	Area	Cut-off	Sensitivity	Specificity	p value	Asymptotic 95% CI	
						Lower bound	Upper bound
Ferritin	0.725	48.5	72	70	**<0.001**	0.672	0.778

ROC: Receiver operating characteristic, CI: Confidence interval

Table 6. Binary logistic regression analysis for predictors of diabetic complications

	B	S.E.	Sig.	Exp (B)	95% CI for exp. (B)
Sex (female)	0.169	0.457	0.712	1.184	0.483–2.899
Age (years)	0.013	0.022	0.553	1.013	0.971–1.056
BMI (kg/m²)	0.017	0.043	0.694	1.017	0.935–1.105
Duration of type 2 DM (years)	**0.121**	**0.061**	**0.046**	**1.129**	**1.002–1.272**
Transition to insulin therapy	0.042	0.070	0.545	1.043	0.910–1.197
Serum ferritin (ng/mL)	–0.002	0.003	0.562	0.998	0.993–1.004
Hemoglobin A1c (%)	0.119	0.116	0.307	1.126	0.896–1.415
Constant	–4.226	2.507	0.092	0.015	–

B: Regression coefficient, S.E.: Standard error, CI: Confidence interval, DM: Diabetes mellitus, BMI: Body mass index, exp.: Exponential, Sig: Significance

Table 7. Comparison of serum ferritin levels among diabetic complication types

Complication type	Group	Ferritin (ng/mL)	p value
Neuropathy	Neuropathy	87.1±68.6	**<0.001**
	Uncomplicated DM	96.9±85.8	
	Control	45.4±33.6	
Retinopathy	Retinopathy	76.6±83.8	**<0.001**
	Uncomplicated DM	96.9±85.8	
	Control	45.4±33.6	
Nephropathy	Nephropathy	110.2±75.1	**<0.001**
	Uncomplicated DM	96.9±85.8	
	Control	45.4±33.6	
All complications	Neuropathy	89.7+70.5	**0.111**
	Nephropathy	110.2+75.1	
	Retinopathy	60.2+56.9	

Values are expressed as mean ± standard deviation (ng/mL). DM: Diabetes mellitus

and BMI in our study suggests that confounding factors such as visceral adiposity and low-grade inflammation may influence this association. This interpretation aligns with a recent meta-analysis indicating that the ferritin– MetS relationship may be confounded by both BMI and C-reactive protein (15). In addition, contemporary reviews summarizing the iron–insulin axis and the bidirectional interaction between hepcidin-mediated inflammation and IR further clarify the biological basis of the observed ferritin–glycemic linkage (17).

In our study, serum ferritin levels were significantly higher in diabetic subgroups with neuropathy, retinopathy, and nephropathy than in the control group, suggesting a potential association between iron overload and diabetes-related organ damage. However, no significant difference in ferritin concentrations was observed among the different complication types, indicating that ferritin may reflect systemic metabolic stress rather than the specific pattern of end-organ involvement. Experimental data have shown that retinal ferritin expression increases in response to systemic iron accumulation, yet the penetration of circulating iron into retinal tissues appears limited, implying that serum ferritin may serve only as an indirect or confounded marker for diabetic retinopathy (18). Clinically, studies have also reported that **serum iron**, rather than ferritin, may be inversely related to the presence of retinopathy, emphasizing the heterogeneity of the iron–retinopathy relationship (19). Regarding renal involvement, recent reviews have highlighted the critical role of ferroptosis and oxidative stress pathways in the pathogenesis of diabetic kidney disease (DKD), suggesting that excess iron may aggravate tubulointerstitial injury, thereby explaining the relatively higher ferritin levels observed in our nephropathy subgroup (20). Emerging evidence on urinary ferritin as a potential early biomarker for DKD further supports the notion that circulating ferritin alone may not adequately capture the severity or progression of diabetic complications (21).

In multivariate logistic regression analysis, only the duration of type 2 diabetes was independently associated with diabetic complications, whereas serum ferritin did not emerge as a significant predictor. The predominance of disease duration and chronic glycemic exposure as determinants of microvascular damage has been consistently demonstrated in large-scale population studies; for instance, studies conducted in Chinese adults identified diabetes duration and glycemic control as the principal risk factors for diabetic retinopathy (22). Ferritin's limited predictive performance may be partly attributed to its nature as an acute-phase reactant and to its modulation by hepcidin-dependent pathways, which can attenuate its independent association in adjusted models. Indeed, in patients with T2DM, hepcidin levels, rather than ferritin, have been shown to correlate with long-term fatal and non-fatal cardiovascular outcomes, highlighting the stronger prognostic role of regulatory hormones within iron metabolism (23). Collectively, these findings suggest that while ferritin reflects aspects of metabolic and inflammatory burden, disease duration and glycemic indicators remain the primary determinants in predicting diabetic complications and should be prioritized in clinical risk assessment.

The effects of iron on ß-cell function, hepatic gluconeogenesis, adipocyte signaling, and oxidative stress underscore its central role in the pathophysiology of T2DM. A recent review highlighted that iron overload accelerates ß-cell dysfunction through ferroptosis, enhances lipid peroxidation, and contributes to worsening glycemic control (24). On the other hand, hepcidin, the key hormonal regulator of iron homeostasis, is also upregulated by inflammation and obesity via the JAK–STAT3 and BMP–SMAD signaling pathways. Therefore, failure to interpret hepcidin levels in conjunction with ferritin may lead to misclassification of metabolic iron status. In this context, comprehensive analyses of systemic iron signaling and insulin sensitivity have emphasized the need for caution when interpreting ferritin-based risk assessments (17). Finally, genetic evidence from

Mendelian randomization studies supports a causal contribution of systemic iron status to T2DM risk, reinforcing the potential benefit of therapeutic strategies that reduce iron load, such as targeted phlebotomy, iron-restricted diets, or modulation of the hepcidin–ferroportin axis (14). This causal inference aligns with our finding of elevated ferritin levels in both the T2DM and IR groups, further supporting the biological plausibility of our results.

Study Limitations

This study has several limitations. First, it was designed as a retrospective, single-center study, which may limit the generalizability of the findings due to potential selection bias and the characteristics of the local patient population. Factors that could influence serum ferritin levels—such as acute-phase reactions, subclinical inflammation, or mild hepatic dysfunction—were not assessed; therefore, ferritin may reflect not only iron storage status but also an underlying inflammatory response. Additionally, other markers of iron metabolism (e.g., serum iron, transferrin saturation, hepcidin, or soluble transferrin receptor) were not evaluated, limiting the ability to comprehensively define ferritin's pathophysiological role within iron homeostasis. The presence of diabetic complications was ascertained from medical records, which may introduce diagnostic misclassification bias. Furthermore, the relatively modest sample size and the cross-sectional nature of the data prevented the evaluation of temporal changes in ferritin levels and their potential causal relationship with the development of diabetic complications. Despite these limitations, this study provides valuable insights by simultaneously assessing diabetes, IR, and ferritin levels within the same population and exploring the relationship between ferritin and different diabetic complications, thereby contributing meaningful data to the existing literature.

Conclusion

Serum ferritin concentrations were significantly higher in individuals with T2DM and IR than in non-diabetic controls. Ferritin showed moderate accuracy in distinguishing T2DM and demonstrated a positive relationship with HbA1c, indicating its association with metabolic and inflammatory load. Although ferritin levels were elevated in patients with microvascular complications compared with controls, they did not independently predict microvascular complications after multivariable adjustment. Disease duration was the only independent factor linked to diabetic complications. Overall, ferritin appears to reflect metabolic dysregulation rather than serving as a reliable standalone predictor of diabetes-related organ damage. Large-scale prospective studies incorporating broader iron metabolism parameters are needed to better define its clinical relevance in risk assessment and management of type 2 diabetes.

Ethics

Ethics Committee Approval: Ethical approval was obtained from the Non-Interventional Clinical Research Ethics Committee of Çukurova University (meeting number: 66, date: July 7, 2017).

Informed Consent: Due to the retrospective nature of the study, the requirement for informed consent was waived by the ethics committee.

Footnotes

Authorship Contributions: Surgical and Medical Practices - M.A.; Concept - T.S.; Design - T.S.; Data Collection or Processing - M.A.; Analysis or Interpretation - T.S.; Literature Search - M.A.; Writing - M.A.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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