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Original Research Article

SERUM FERRITIN AND ALBUMIN LEVELS IN DIABETIC PATIENTS: A CASE–CONTROL STUDY

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ABSTRACT

Background: Ferritin and albumin are major circulating proteins with opposing behavior during inflammation: ferritin tends to rise as an acute-phase reactant, while albumin declines. Type 2 diabetes mellitus (T2DM) is associated with chronic low-grade inflammation, which can alter these biomarkers. We were comparing serum ferritin and albumin levels in T2DM patients versus non-diabetic controls and assessing the relationship between ferritin and albumin after adjusting for age, sex, and glycemic control (HbA1c).

Materials and Methods: In this case-control study, we analyzed 100 patients with T2DM (cases) and 100 age- and sex-matched non-diabetic controls. Serum ferritin and albumin were measured in the VITROS 5600 Autoanalyser (Ortho Clinical Diagnostics, Raritan, NJ, USA), and HbA1c was measured in Bio-Rad D10 analyser (Bio-Rad Laboratories, Hercules, CA, USA) by the HPLC method. Descriptive statistics were computed for each group. Group comparisons were performed using independent t-tests or chi-square tests. Pearson's and Spearman's correlations between ferritin, albumin, and other variables were calculated. A multiple linear regression model was constructed with ferritin as the dependent variable, albumin as the main independent variable, and age, sex, and HbA1c as covariates. Beta coefficients with 95% confidence intervals (CI) and p-values were reported.

Results: Diabetic patients had markedly higher mean ferritin (312.6 ng/mL) compared to controls (162.3 ng/mL) and lower mean albumin (3.96 g/dL vs 4.31 g/dL) (both $p < 0.001$). HbA1c was significantly elevated in diabetics (7.42% vs 4.83%, $p < 0.001$), while age and sex distribution were similar between groups so insignificant ($p>0.7$). Ferritin was inversely correlated with albumin (Pearson $r = –0.19$, $p = 0.007$; Spearman $\rho = –0.21$, $p = 0.002$) and positively correlated with HbA1c ($r = 0.42$, $p < 0.001$). In multivariate regression, albumin was not an independent predictor of ferritin after adjustment ($\beta = –27.7$ ng/mL per g/dL [95% CI –83.3 to 27.9], $p = 0.33$), whereas HbA1c remained strongly associated ($\beta = +33.8$ ng/mL per 1% HbA1c [22.3 to 45.3], $p < 0.001$).

Conclusion: Ferritin and albumin showed a significant inverse correlation overall, this relationship was largely explained by glycemic status; albumin did not independently predict ferritin when adjusting for HbA1c and other factors. High ferritin in T2DM appears to indicate poor glycemic control and inflammation, whereas serum albumin changes were modest. Clinically, monitoring ferritin can help identify patients with subclinical Inflammation or inadequate diabetes control, along with the maintenance of normal albumin

levels, normal albumin levels may signify better metabolic health. Further research should explore whether improving glycemic control and reducing inflammation can concurrently lower ferritin and raise albumin levels in diabetic patients.
Keywords: Albumin, Diabetes, Ferritin, Inflammation, Regression analysis.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a metabolic disorder marked by persistent chronic hyperglycemia and insulin resistance, often associated with of low-grade systemic inflammation.[1] Inflammatory mediators have been associated with the pathogenesis of T2DM and its complications.[2] Serum ferritin, an iron-storage protein, is a recognised known positive acute-phase reactant that can be elevated in inflammatory conditions.[3] High ferritin levels may indicate inflammation or iron overload, both of which can negatively impact metabolism in diabetes.[4] Too much iron and ferritin have been linked to insulin resistance and β-cell dysfunction.[5] Epidemiological research have shown that patients with T2DM tend to have higher serum ferritin levels compared to healthy individuals.[6] High ferritin levels correlate positively with measures of poor glycemic control such as fasting glucose and glycated hemoglobin (HbA1c), and elevated ferritin has been identified as a risk factor for the development of T2DM.[7]

Albumin, on the other hand, is the most common serum protein and a negative acute-phase reactant.[8] During systemic inflammation, albumin synthesis in the liver decreases, leading to lower circulating albumin levels.[9] Low serum albumin has historically been associated with malnutrition, but it is now also act as a marker of inflammation and metabolic risk. In relation of diabetes, mild hypoalbuminemia may occur in patients with inflammation, nephropathy, or other comorbidities, and reduced albumin has been associated with a poorer metabolic profile and increased risk of developing T2DM.[10,11] However, findings on albumin's role in diabetes have been somewhat inconsistent; some longitudinal studies reported that lower baseline albumin predicts higher diabetes risk, whereas others showed no association or even a paradoxical inverse relationship.[12] A developing perspective suggests that chronic inflammation in obesity and T2DM can lower albumin and raise ferritin as part of the acute-phase response.[13]

The relationship between serum ferritin and albumin in diabetes is of interest for this research. In inflammatory conditions, we can expect ferritin to be elevated and albumin to be depressed, potentially leading to an inverse correlation. This inverse relationship could have clinical implications, a combination of high ferritin and low albumin might signal heightened inflammatory burden or risk of complications in diabetic patients. However, the direct association between serum ferritin and albumin, independent of other factors, has not been well studied in diabetic populations.

The objective of this study was to compare serum ferritin and albumin levels between individuals with Type 2 Diabetes Mellitus (T2DM) and non-diabetic controls, as well as to assess the correlation between these two biomarkers.We used a multivariate approach to determine whether albumin is an independent predictor of ferritin after adjusting for key covariates such as age, sex, and HbA1c. Understanding this relationship could provide insights into the inflammatory and nutritional status of diabetic patients and the potential utility of these laboratory parameters in clinical risk assessment.

MATERIALS AND METHODS

Study design: We conducted a cross-sectional case-control analysis comparing individuals with type 2 diabetes mellitus to non-diabetic control subjects. A total of 200 participants were included, comprising 100 T2DM patients and 100 healthy controls.

Study Duration: This study was conducted from July 2024 to June 2025 in the department of Biochemistry, Tezpur Medical College and Hospital with collaboration with Department of Medicine, Tezpur Medical College and Hospital.

Inclusion criteria

Cases (T2DM group): Adults aged 21–79 years, clinically diagnosed with Type 2 Diabetes Mellitus, defined as prior physician diagnosis, current use of anti-diabetic medication, HbA1c ≥ 6.5%, or fasting plasma glucose ≥ 126 mg/dL. Controls (non-diabetic group): Adults aged 21–79 years, with no history of diabetes and HbA1c < 5.7% (or fasting glucose < 100 mg/dL), matched to cases by age and sex. Every participants were required to be clinically and mentally stable, free from acute illness, and willing to undergo overnight fasting prior to venous blood sampling.

Exclusion criteria

Participants were excluded if they had: Type 1 Diabetes Mellitus (T1DM) or gestational diabetes, Acute infection, febrile illness, recent major surgery or trauma,Blood transfusion within the last 3 months, Chronic inflammatory diseases (e.g., rheumatoid arthritis, inflammatory bowel disease, systemic lupus erythematosus), Active malignancy or cancer therapy within the last 12 months, Chronic liver disease (cirrhosis, chronic viral hepatitis, alcoholic liver disease), Iron overload disorders (e.g., hereditary haemochromatosis) or currently treated iron-deficiency anemia, Pregnancy or lactation, Any condition likely to affect ferritin or albumin interpretation, such as severe malnutrition

or protein-losing enteropathy and Inability to provide informed consent or comply with study procedures.

Data collection and laboratory measurements: For each participant, demographic and clinical data were recorded, including age, sex, and diabetes status (case or control). Venous blood samples were collected after an overnight fast. Serum ferritin was measured (in ng/mL) using a standard immunoassay, and serum albumin (g/dL) was measured by the bromocresol green colorimetric method. Hemoglobin A1c (HbA1c, %) was determined via high-performance liquid chromatography for all participants as an index of glycemic status. All assays were performed after appropriate quality controls and calibration check.

Statistical analysis: In this study descriptive statistics were used to summarize the characteristics of cases and controls. Continuous variables (age, HbA1c, ferritin, albumin) were assessed for normality. Means and standard deviations (SD) are done for approximately normally distributed variables, Categorical variables (sex) are summarized as counts and percentages.

Group comparisons between the T2DM and control groups were performed using independent two-sample t-tests for continuous variables and chi-square tests for categorical variables. Ferritin values were right-skewed, the t-test is significant, but we also verified key results with non-parametric Mann-Whitney U tests for ferritin which showed consistent significance. A p value < 0.05 was considered statistically significant.

Correlation analyses were conducted to evaluate the bivariate relationships of ferritin with albumin, age, and HbA1c. Both Pearson's correlation coefficient (r) and Spearman's rank correlation (ρ) were calculated, given that ferritin's distribution is skewed. Correlations were interpreted along with their p-values (two-tailed).

To assess the independent association between albumin and ferritin, we performed a multivariable linear regression. We included age (years), sex (coded as 0 = female, 1 = male), and HbA1c (%) as covariates in the model, based on clinical relevance. These covariates adjust for potential confounding: age and sex can influence ferritin levels for example higher ferritin in males and older individuals, and HbA1c reflects glycemic control which is linked to ferritin in diabetes. The regression results are presented as β coefficients with 95% confidence intervals and corresponding p-values. All statistical analyses were performed using Microsoft Excel and Jamovi software.

Ethical considerations: In this study informed consent was obtained from participants and the study had ethics committee approval from Institutional Ethical Committee, Tezpur Medical College & Hospital. The research was conducted in accordance with the principles of the Declaration of Helsinki.

Conflict of Interest: The authors declare that there is no conflict of interest regarding the publication of this paper.

RESULTS

A total of 200 individuals were analyzed, with 100 in the T2DM case group and 100 in the non-diabetic control group. The mean age of participants was approximately 49.5 years in both groups (Table 1), and age did not differ significantly between cases and controls ($p = 0.91$). The sex distribution was also similar (58% male in cases vs 61% male in controls; $p = 0.77$), reflecting successful matching. As expected, glycemic status differed markedly: the diabetic group had a mean HbA1c of 7.42 ± 1.46%, compared to 4.83 ± 0.45% in controls, indicating normoglycemia in controls and poor glycemic control in the T2DM group, this difference was highly significant, $p < 0.001$.

Serum ferritin levels were substantially higher in T2DM patients than in controls. The mean ferritin in cases was 312.6 ± 150.2 ng/mL, versus 162.3 ± 86.4 ng/mL in controls ($p < 0.001$). Conversely, serum albumin was modestly but significantly lower in the T2DM group (mean 3.96 ± 0.32 g/dL) compared to controls (4.31 ± 0.29 g/dL, $p < 0.001$).

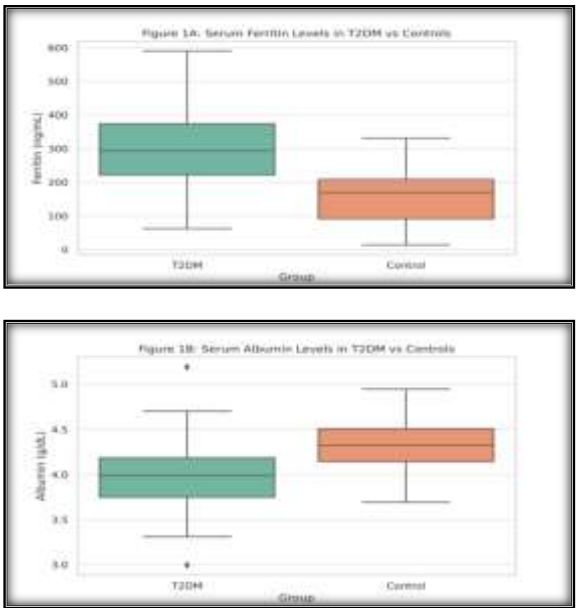

Figure 1: Boxplots of serum ferritin (1A) and albumin (1B) levels in type 2 diabetes cases versus healthy controls.

The diabetic group shows a markedly higher median ferritin and broader distribution, whereas the control group has lower ferritin levels. For albumin, diabetics have a slightly lower median serum albumin compared to controls. Differences in ferritin and albumin between groups were statistically significant (both $p < 0.001$).

Table 1: Baseline characteristics of group (cases vs controls). Values are mean ± SD or n (%).

Variable	T2DM Cases (n = 100)	Controls (n = 100)	P-value
Age (years)	49.3 ± 10.6	49.6 ± 18.0	0.91
Male sex, n (%)	58 (58%)	61 (61%)	0.77
HbA1c (%)	7.42 ± 1.46	4.83 ± 0.45	< 0.001
Ferritin (ng/mL)	312.6 ± 150.2	162.3 ± 86.4	< 0.001
Albumin (g/dL)	3.96 ± 0.32	4.31 ± 0.29	< 0.001

Correlations between ferritin, albumin, and other variables: In this study we examined the associations between serum ferritin and key variables (albumin, HbA1c, age) using correlation analysis. In all 200 samples, ferritin was negatively correlated with albumin (Pearson r = –0.189, p = 0.007; Spearman ρ = –0.214, p = 0.002) [Figure 2]. This indicates that individuals with lower albumin tended to have higher ferritin levels, although the correlation was modest in strength.

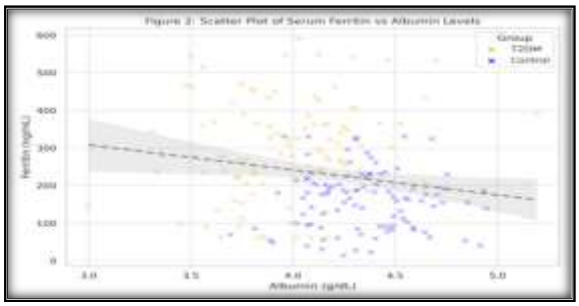

Figure 2: Scatter plot of serum ferritin vs albumin levels in all participants (N = 200).

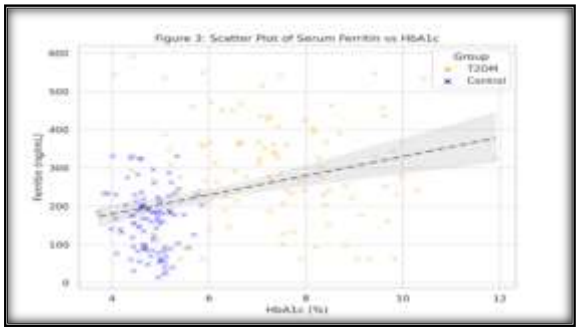

Figure 3: Serum ferritin showed a significant positive correlation with HbA1c (Pearson r = +0.421, p < 0.001; Spearman ρ = +0.441, p < 0.001).

Multivariate regression analysis: To determine whether serum albumin independently predicts ferritin levels after accounting for potential confounders, we fitted a multiple linear regression model with ferritin as the outcome. The predictors were albumin (primary variable of interest), age, sex, and HbA1c. The regression results (Table 2) showed that, after adjustment, albumin's association with ferritin was substantially attenuated and no longer statistically significant. The adjusted β coefficient for albumin was –27.7 (95% CI –83.3 to +27.9) ng/mL per 1 g/dL increase in albumin (p = 0.327). In other words, after controlling for age, sex, and HbA1c, a one unit higher albumin was associated with an estimated 27 ng/mL lower ferritin on average, but this estimate had a wide confidence interval crossing zero (indicating no clear effect).

By contrast, HbA1c emerged as a strong independent predictor of ferritin. Each 1% increase in HbA1c was associated with a +33.8 ng/mL higher ferritin (95% CI +22.3 to +45.3), holding other factors constant (p < 0.001). This suggests that poor glycemic control has a significant positive relationship with ferritin levels, consistent with our correlation findings and indicating an inflammatory/iron storage response to hyperglycemia. Age showed a positive but non-significant association (β ≈ +0.99 ng/mL per year, 95% CI –0.26 to +2.24, p = 0.120), hinting that older age might be associated with slightly higher ferritin, but our sample did not have enough evidence to confirm this (particularly since groups were age-matched and the age range in controls included some younger individuals, increasing variability). Sex (male vs female) was not a significant determinant of ferritin in the adjusted model (β for male = –17.5 ng/mL compared to female, 95% CI –55.3 to +20.2, p = 0.361). This result indicates that, in general population, men often have higher ferritin; however, in our dataset the diabetic group had a high ferritin regardless of sex (indeed, diabetic women had ferritin levels comparable to or higher than diabetic men, perhaps due to postmenopausal status and inflammation), which may have negated the usual sex difference.

Table 2: Multivariate linear regression predicting serum ferritin (ng/mL) as the outcome. Beta coefficients (β) with 95% confidence intervals and p-values are shown for each predictor (N = 200).

Predictor	β coefficient (95% CI)	p-value
Albumin (per 1 g/dL)	–27.7 (–83.3 to +27.9)	0.327
Age (per 1 year)	+0.99 (–0.26 to +2.24)	0.120
Male sex (vs female)	–17.5 (–55.3 to +20.2)	0.361
HbA1c (per 1%)	+33.8 (+22.3 to +45.3)	< 0.001

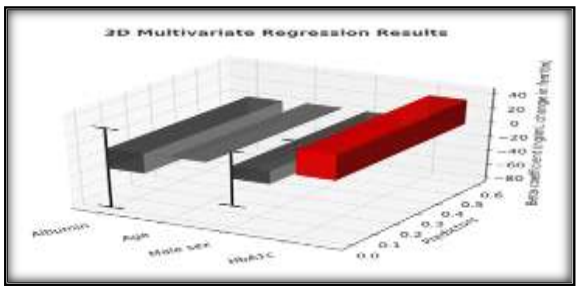

Figure 4: 3D bar chart version of our multivariate regression results. Bar height = β coefficient (effect size on ferritin),Black vertical lines = 95% confidence intervals,Red bars = statistically significant (HbA1c), Gray bars = not significant (Albumin, Age, Sex).

In summary, our adjusted analysis indicates that serum albumin is not significantly associated with serum ferritin once we account for glycemic control and other factors. The initial crude inverse association between albumin and ferritin appears to have been confounded, mainly by the markedly higher HbA1c in the diabetic group. HbA1c itself showed a robust relationship with ferritin, reinforcing the link between hyperglycemia and elevated ferritin levels in T2DM.The strong correlation between ferritin and HbA1c further supported the idea that hyperglycemia and high ferritin levels are related in type 2 diabetes.

DISCUSSION

In this study, we investigated the serum ferritin and albumin levels in type 2 diabetic patients compared to healthy controls, and explored the relationship between these two biomarkers in the context of diabetes. Our findings highlight a clear divergence between the groups: T2DM patients had significantly higher ferritin and slightly lower albumin than non-diabetics. We also observed an inverse correlation between ferritin and albumin in the combined population, but this correlation disappeared after adjusting for covariates, particularly HbA1c. These results provide insight into the inflammatory and metabolic changes in diabetes and their impact on common laboratory markers.

The elevated ferritin in diabetic individuals observed in our study is consistent with a wealth of evidence linking T2DM to higher iron stores and inflammatory activation. Numerous cross-sectional studies have reported higher serum ferritin in patients with T2DM compared to controls.[6,18] For example, Sunar et al. (2021) found that recently diagnosed T2DM patients had significantly greater ferritin levels than healthy controls (mean 153 vs 63 ng/mL). Our cases, who had established diabetes with suboptimal glycemic control, had an even greater ferritin elevation (mean 313 vs 162 ng/mL in controls). The presence of high ferritin in diabetes is widely interpreted as a marker of chronic inflammation and altered iron metabolism associated with insulin resistance.[3] Kacimi et al. (2024) demonstrated that type 2 diabetics exhibit higher ferritin and C-reactive protein (CRP) levels than controls, indicative of a low-grade inflammatory state.[1]

The positive correlation we found between ferritin and HbA1c further supports the connection between iron-related inflammation and glycemic status. Prior research has shown that serum ferritin correlates with indices of poor glycemic control and insulin resistance.[6,19] Improved glycemic control can lead to reductions in ferritin levels. Momeni et al. (2015) reported that when hyperglycemia was brought under control in T2DM patients, serum ferritin concentrations significantly decreased.[20] They suggested that ferritin could potentially serve as an index of diabetes control. This aligns with our regression results, wherein HbA1c was the only significant predictor of ferritin. It implies that chronic hyperglycemia and its downstream effects (possibly oxidative stress and inflammation) are driving ferritin elevations in diabetics,[5] Mechanistically, hyperinsulinemia and insulin resistance may increase ferritin by causing iron accumulation in tissues and upregulating ferritin synthesis.[3] High ferritin can contribute to a vicious cycle of metabolic dysfunction: it may promote insulin resistance and β-cell stress by catalyzing oxidative reactions. it also signals higher risk of complications e.g. it has been linked to cardiovascular disease and fatty liver in diabetes.[21]

Regarding serum albumin, we found a modest yet significant reduction in the diabetic groups. Albumin is well known to decrease during inflammation because hepatic protein synthesis shifts towards acute-phase proteins (like CRP, ferritin, fibrinogen) and away from albumin.[9] Chang et al. (2019) noted that low albumin is associated with markers of inflammation (e.g. higher adipose tissue macrophage activation) and a worse metabolic profile. They concluded that "reduced albumin is associated with an unfavorable metabolic profile. and with an increased risk for T2DM. In established diabetes, lower albumin has been linked to greater risk of complications such as diabetic nephropathy and retinopathy in some studies.[22] It is important to clarify that the albumin changes in T2DM are usually subtle unless there is significant comorbidity (e.g. nephrotic syndrome or severe inflammation). Our finding of ~0.3 g/dL lower albumin in diabetics likely reflects a mild chronic inflammation and perhaps effects of hyperglycemia (e.g. non-enzymatic glycation of albumin could slightly reduce measured concentrations). Somewhat paradoxically, higher serum albumin has been associated with lower incidence of microvascular complications in diabetes,[23]reinforcing that maintaining a normal albumin is a sign of better health.

We initially hypothesized that ferritin and albumin would be inversely related due to their opposing acute-phase responses. The unadjusted data did show an inverse correlation overall. However, our regression analysis indicates that this relationship is not independent of glycemic control. Essentially, it

appears that T2DM status raises ferritin and lowers albumin simultaneously, creating an overall inverse correlation, but within a given glycemic stratum, albumin may not have a direct impact on ferritin. In our diabetic patients, albumin and ferritin were not significantly correlated when isolated, suggesting that factors like glucose control, insulin levels, and possibly other inflammatory mediators are the common drivers affecting both. This finding is important for interpretation: a low-normal albumin in a diabetic patient does not necessarily cause high ferritin, nor vice versa; rather, both could be consequences of the underlying inflammatory metabolic state.

From a clinical perspective, our results underscore ferritin's potential utility as a marker in T2DM management. Since ferritin reflects inflammation and correlates with HbA1c, it might be used to identify patients with poorly controlled diabetes who also have an inflammatory profile. This is relevant because inflammation in diabetes is linked to complications. For instance, elevated ferritin and CRP in T2DM have been associated with a higher risk of atherosclerosis and cardiovascular events.[2]

Our study results align with recent literature emphasizing the dual role of ferritin as both an iron storage indicator and an inflammatory marker in diabetes.[5] For example, a recent study by Huang et al. (2023) described the "dual nature" of ferritin in older diabetic patients: low ferritin indicating iron deficiency anemia and high ferritin associated with obesity, hepatic enzyme elevations, and poor glycemic control.[24] A unique aspect noted by Chang et al. was that even in non-diabetic individuals, lower albumin (within normal range) predicted future diabetes onset,[10] possibly as a marker of chronic inflammation and oxidative stress. Therefore, in a preventive context, maintaining higher normal albumin could be favorable.

Strengths and limitations: Key strengths of our study were include the well-defined case-control design with matching for age and sex, allowing a clear comparison of ferritin and albumin between diabetics and healthy controls. We also included multivariate analysis to adjust for confounders and provided detailed statistical outputs (including both Pearson and Spearman correlations, to account for non-normal distributions). The sample size (n=200 total) provided sufficient power to detect the observed differences. However, several limitations should be noted. First, the study is cross-sectional, so causal inferences cannot be made. We cannot definitively say that diabetes causes high ferritin or low albumin, only that they are associated. It is possible that high ferritin could precede and contribute to diabetes.[7]Longitudinal studies are needed to untangle this bidirectional relationship. Second, we did not measure other inflammatory markers (like CRP, IL-6) or body iron indices (like transferrin saturation). Such data would help confirm that the ferritin elevation was due to inflammation rather than primary iron overload. Third, our regression model included only age, sex, and HbA1c as covariates. There are other potential confounders we did not account for: body mass index (BMI) and visceral adiposity (obesity can influence both ferritin and albumin), duration of diabetes, medications e.g. metformin has been reported to lower some inflammatory markers, liver function, and renal function where albumin can drop in nephropathy. Particularly, low albumin in diabetes could result from nephrotic-range proteinuria in advanced diabetic nephropathy, which was beyond our scope to evaluate. Our study assumed that none of the participants had advanced liver or kidney disease, but we did not have direct measures of those organ functions.

Another limitation is we do not include Type1DM cases but we exclude with the help of clinical senerio and symtops and another limitation is that the control group's HbA1c was measured and found to be ~4.8%, which is slightly lower than population averages (around 5.1–5.4% for normoglycemic adults). This suggests our controls might have been particularly healthy or possibly younger on average. However, their age range did include older individuals (up to 79). It's possible some controls had lifestyle factors that kept their HbA1c low. Regardless, the gap between 7.4% and 4.8% in HbA1c is reflective of true diabetic vs non-diabetic glycemic statuses. Finally, our findings are based on a single-center dataset (geographically from one region); thus, generalizability may be limited. Different ethnic populations may have different baseline ferritin levels due to genetic variations (e.g. HFE gene mutations) and dietary iron intake, and albumin can be influenced by nutritional status differences. Further studies in diverse populations would be beneficial.

Implications: The clinical relevance of this study lies for a patient with T2DM, a high ferritin level should alert the clinician to consider concomitant inflammation or liver iron overload. It might prompt further evaluation (such as checking CRP, liver enzymes, or iron studies) and more aggressive management of risk factors. On the other hand, serum albumin is often part of routine metabolic panels; a low-normal albumin in a diabetic patient might be overlooked, but it could be an early sign of systemic inflammation or malnutrition that merits attention. Our results also suggest that interventions which improve glycemic control could beneficially impact ferritin levels. There is evidence that treating underlying conditions (better glucose control, weight loss, or using anti-inflammatory strategies) can normalize these markers.[14]

Future studies could explore if a composite index (like ferritin-to-albumin ratio) has any prognostic value in diabetes, analogous to how ratios of inflammatory markers are sometimes used.

CONCLUSION

In summary, our study demonstrates that patients with type 2 diabetes have significantly higher serum ferritin and slightly lower serum albumin levels compared to non-diabetic controls, consistent with an underlying state of chronic inflammation and metabolic dysregulation. Although ferritin and albumin show an inverse correlation overall, this association is not independent of glycemic control; rather, it appears that poor glycemic control (reflected by high HbA1c) drives ferritin upward and may modestly suppress albumin. After adjusting for HbA1c (and age, sex), albumin is not a significant predictor of ferritin levels. These findings highlight serum ferritin as a potential indicator of inflammatory burden and glycemic derangement in T2DM, whereas albumin changes are less pronounced. Clinicians should be aware that an elevated ferritin in a diabetic patient could signal poor diabetes control or associated inflammation, prompting closer monitoring or therapeutic interventions.

Maintaining good glycemic control and reducing inflammation may help normalize ferritin (and preserve albumin) levels, potentially improving patient outcomes. Further research is warranted to investigate whether interventions targeting inflammation and iron metabolism in diabetes can positively influence these biomarkers and reduce complication risks.

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