

Association of Iron Status, Serum Creatinine, and Liver Enzymes with Glycemic Control in Patients with Type 2 Diabetes Mellitus: A Retrospective Observational Study

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is a multifaceted metabolic illness represented by long term hyperglycemia and coincided by alterations in iron metabolism, renal-related biochemical markers, and hepatic enzymes.

Objective: This study aimed to evaluate the relationship among serum iron, ferritin, creatinine, alanine aminotransferase (ALT), aspartate aminotransferase (AST) and gamma-glutamyl transferase (GGT), with glycemic control in (T2DM) patients.

Methodology: This retrospective observational study used previously recorded clinical and laboratory data from 60 adults with T2DM and 60 matched apparently healthy controls. Patients with T2DM were further classified according to blood glucose control as well controlled (HbA1c <7%) or poorly controlled (HbA1c ≥7%). Serum iron, ferritin, creatinine, ALT, AST, GGT, and HbA1c values were retrieved from existing records. Associations between biochemical parameters and HbA1c were assessed within the T2DM group.

Results: Serum iron and ferritin values were considerably reduced in patients with T2DM than in controls (both $p < 0.001$), and serum creatinine also varied significantly between groups ($p = 0.001$). No significant variations were noticed in ALT, AST, or GGT between T2DM patients and controls. Within the T2DM group, HbA1c was positively linked with GGT ($\rho = 0.348$, $p = 0.006$), creatinine ($\rho = 0.338$, $p = 0.008$), and age ($\rho = 0.405$, $p = 0.001$), and negatively linked with serum iron ($\rho = -0.260$, $p = 0.045$). Ferritin presented a non-significant inverse association with HbA1c ($\rho = -0.234$, $p = 0.072$). GGT and creatinine were significantly elevated in patients with poor glycemic control than in those with HbA1c <7% ($p = 0.037$ and $p = 0.024$, respectively).

Conclusion: Here in retrospective observational study of patients with T2DM, lower serum iron and ferritin concentrations were observed compared with matched controls, while GGT and creatinine were positively associated with HbA1c. GGT was also higher among patients with poor glycemic control. These findings indicate associations between glycemic control and selected biochemical markers but do not establish causality or independent predictive value.

Keywords: Type 2 Diabetes Mellitus, Glycemic Control, HbA1c, Serum Iron, Ferritin, Gamma-glutamyl Transferase, Liver Enzymes, Creatinine.

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INTRODUCTION

Diabetes mellitus type 2 (T2DM) is a principal persistent metabolic illness caused by long term hyperglycemia, which is due to insulin resistance and gradual loss of pancreatic β -cells function (1). Prevention or delay of diabetes complications is related to glycemic control, and the chief laboratory indices to monitor for chronic glycemic exposure is currently HbA1C, representing

glycemic exposure over the past 2–3 months (2). It is now recommended by the American Diabetes Association that an HbA1C concentration of less than 7% is considered appropriate for many adults who are NOT pregnant, but is individualized based on age, comorbidities, hypoglycemic risk, and overall health (3, 4).

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Metabolic abnormalities associated with T2DM go beyond glucose (5). Dysfunctions in iron metabolism have been of great interest, given that iron plays a main duty in mitochondrial function, oxidative reactions, insulin signaling, and pancreatic β -cell function (6). Inflammatory, infectious, liver, and metabolic disorders can also affect ferritin (7), the predominant iron-storage protein (8). Low ferritin levels indicate insufficient iron stores, while higher ferritin levels could indicate iron overload. Suggesting that ferritin as a good marker of iron stores (9). Thus, an elevated or decreased serum ferritin level should not be used as a definitive indicator of total-body iron stores without taking the clinical context into account (7). Insulin resistance and T2DM are associated with higher ferritin levels or greater body iron stores, as previously reported (10-12). A recent study on HIC concluded that HIC is inconsistently identified in people with prediabetic levels or T2DM, and that liver iron influences health outcomes, indicating there may be variations in iron distribution/metabolism between prediabetic and T2DM (13). Therefore, a more complex relationship exists between iron status and glycemic control than between higher stores and hyperglycemia (14-16). Another factor is iron deficiency and its effect on the interpretation of HbA1c. Changes in erythrocyte lifespan and iron deficiency may affect HbA1c without glucose exposure (17). A study published in 2023 revealed a general decrease in HbA1c with iron replacement therapy, but substantial variation was observed among the included studies (18). The 2026 American Diabetes Association (ADA) Standards of Care also warn that other factors that influence red blood cell lifespan can disrupt the correlation between HbA1c and glycemic status (19).

The metabolic pattern of T2DM, which may be vital, is liver enzymes (20, 21). ALT, AST, and GGT are traditionally used as markers of hepatocyte and hepatobiliary abnormalities and may also be a reflection of abnormalities of metabolism (22). GGT has been related to insulin resistance, metabolic syndrome, oxidative stress, and future diabetes risk (23-25). In a recent prospective long-term study, elevated GGT levels were correlated with the risk of developing incident metabolic syndrome and T2DM (26, 27). In human studies specifically performed on Saudi T2DM patients, it has also been shown that increasing GGT levels are related to poor glycaemic control, dyslipidaemias, hypertension, and abdominal obesity (28, 29).

Accordingly, this retrospective observational study was designed to compare serum iron, ferritin, creatinine, ALT, AST, and GGT between T2 diabetics and apparently healthy controls, and to assess their associations with HbA1c. The study also examined differences in these biochemical parameters according to glycemic-control status.

MATERIAL AND METHODS

Study design: This retrospective observational study was achieved using previously recorded clinical and laboratory data from the outpatient clinic of King Abdulaziz Specialized Hospital in Aljouf Province, Saudi Arabia. Records of 60 adults (age ≥ 18 years) diagnosed as T2DM, and 60 apparently healthy controls were reviewed. Patients with T2DM were classified according to HbA1c as having well-controlled glycemia (HbA1c $< 7\%$) or poor controlled glycemia (HbA1c $\geq 7\%$). Eligibility and exclusion criteria were applied on the basis of information documented in the available medical records. The study used existing records and involved no direct recruitment, examination, or prospective collection of clinical specimens by the investigators.

Data collection: Demographic and biochemical variables, including age, sex, serum iron, ferritin, creatinine, ALT, AST, GGT, and HbA1c, were obtained from existing hospital medical records and laboratory reports. No new patient samples were collected specifically for this study. The extracted data were entered into the study dataset for statistical analysis. Information on exclusion criteria, including recent iron supplementation or anemia treatment, recent blood donation or transfusion, pregnancy, malabsorptive disorders, bleeding disorders, acute medical conditions, ferritin-influencing medications, type 1 diabetes, and chronic or end-stage kidney disease requiring erythropoietin, was obtained from the available medical records.

Ethical approval: This study involved retrospective analysis of previously recorded clinical and laboratory data from hospital medical registry. The ethics committee determined that formal approval or individual consent was not required for this de-identified record review

Statistical analysis: The data have been analysed using SPSS version 27.0. The Continuous variables were summarized using medians and interquartile ranges, if any, and the categorical variables were summarized using frequency and percentage. Biochemical parameters were compared among T2 diabetic patients and healthy controls and between well-controlled and poorly controlled T2DM groups. Spearman rank association was conducted to evaluate associations between biochemical markers and HbA1c within the T2DM group. A two-sided p-value < 0.05 was thought-out statistically significant. Because multiple biochemical parameters were examined, borderline p-values were interpreted cautiously, and the analyses were considered associative rather than causal.

RESULTS

Sixty patients with T2DM were compared with 60 matched apparently healthy controls. There were no

considerable variations in age ($p = 0.793$) or sex distribution ($p = 0.465$) among the groups (Table 1).

Table 1 Demographic Variables of T2DM Patients and Healthy Controls

Variable	T2DM Patients (n = 60)	Healthy Controls (n = 60)	p-value
Age (years), median (IQR)	39.5 (30–57)	41.0 (30–54)	0.793
Sex, Male, n (%)	33 (55.0%)	28 (46.7%)	0.465
Sex, Female, n (%)	27 (45.0%)	32 (53.3%)	—

Serum iron, ferritin, and creatinine concentrations were considerably diminished in patients with T2DM than in healthy controls ($p < 0.001$, $p < 0.001$, and $p = 0.001$, respectively). No significant variations were noticed in ALT, AST, or GGT among the two groups (Table 2).

These findings indicate that the biochemical differences observed in this cohort were not accompanied by significant between-group variations in the measured liver enzymes.

Table 2 Comparison of Biochemical Parameters Between T2DM Patients and Healthy Controls

Parameter	T2DM Median (IQR)	Control Median (IQR)	p-value
HbA1c (%)	6.00 (5.7–7.0)	4.90 (4.7–5.1)	<0.001***
Iron (µg/dL)	57.0 (36.0–86.5)	95.0 (76.8–122.5)	<0.001***
Ferritin (ng/mL)	25.0 (10.0–68.5)	82.8 (49.1–111.0)	<0.001***
Creatinine (mg/dL)	0.62 (0.54–0.79)	0.77 (0.68–0.83)	0.001***
AST (U/L)	17.0 (15.0–22.0)	21.5 (16.0–26.0)	0.110
GGT (U/L)	22.0 (12.8–41.0)	21.0 (13.0–28.3)	0.331
ALT (U/L)	17.5 (12.8–27.0)	19.5 (13.0–26.0)	0.709

*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$

Within the T2DM group, Spearman association analysis showed significant positive correlations of HbA1c with GGT ($\rho = 0.348$, $p = 0.006$), creatinine ($\rho = 0.338$, $p = 0.008$), and age ($\rho = 0.405$, $p = 0.001$). Serum iron was inversely associated with HbA1c ($\rho = -0.260$, $p = 0.045$), whereas ferritin displayed a non-significant inverse

association ($\rho = -0.234$, $p = 0.072$). ALT and AST were not significantly associated with HbA1c (Table 3). These analyses indicate associations rather than independent effects because no multivariable adjustment was performed.

Table 3 Correlation Between Biochemical Parameters and Glycemic Control (HbA1c) in T2DM Patients (n = 60)

Parameter	Spearman's ρ	p-value
Age	+0.405	0.001*
GGT	+0.348	0.006*
Creatinine	+0.338	0.008*
Iron	-0.260	0.045*
Ferritin	-0.234	0.072

ALT	+0.174	0.183
AST	-0.025	0.851

*** p<0.001, ** p<0.01, * p<0.05

When patients were stratified by glycemic-control status, GGT was significantly increased in the poorly controlled group (HbA1c $\geq 7\%$) than in the well-controlled group (median 38.0 vs. 16.0 U/L, p = 0.037). Creatinine was also significantly elevated in the poorly controlled group

(median 0.79 vs. 0.61 mg/dL, p = 0.024). Iron and ferritin were lower in the poorly controlled group, nevertheless, these variations did not attain statistical significance (p = 0.052 and p = 0.093, respectively) (Table 4).

Table 4 Comparison of Biochemical Parameters Between T2DM Patients with Well vs. Poor Glycemic Control

Parameter	Well Control (HbA1c < 7%, n=43) Median	poorly Control (HbA1c $\geq$ 7%, n=17) Median	p-value
GGT (U/L)	16.0	38.0	0.037*
Creatinine (mg/dL)	0.61	0.79	0.024*
Iron (μ g/dL)	72.0	55.0	0.052
Ferritin (ng/mL)	43.0	15.0	0.093
AST (U/L)	18.0	16.0	0.098
ALT (U/L)	18.0	17.0	0.915

*** p<0.001, ** p<0.01, * p<0.05

Among the biochemical markers examined, GGT displayed a statistically significant positive association with HbA1c and was significantly elevated in the poorly-glycemic-control group (Figure 1 and Table 4). This finding is coherent with prior reports linking GGT with metabolic dysfunction and insulin resistance; however,

the present retrospective study did not directly evaluate insulin resistance, oxidative stress, hepatic steatosis, or other potential mechanisms. Therefore, GGT should be described as an associated biochemical marker rather than an independent marker of glycemic deterioration.

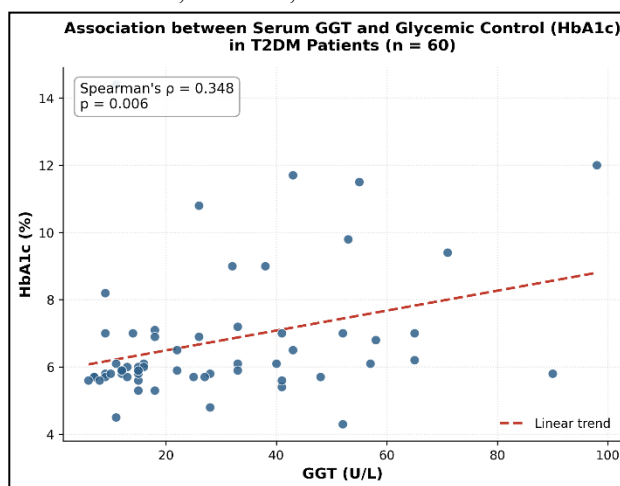


Figure 1 Scatter Plot of the Most Appropriate Association: Serum GGT vs. HbA1c

Figure 1: GGT was selected for the scatter plot because it showed a statistically significant positive correlation with HbA1c among the biochemical markers examined (Spearman's $\rho = 0.348$, p = 0.006). The figure illustrates an association and should not be elucidated as evidence of causality or independent prediction.

DISCUSSION

This retrospective observational study looked-into the associations of serum iron, ferritin, creatinine, and liver enzymes with glycemic control in type 2 diabetics. Serum

iron and ferritin were significantly reduced in type 2 diabetics than in matched apparently healthy controls. In contrast, ALT, AST, and GGT did not vary significantly

among the two groups. Within the T2DM group, GGT and creatinine were positively correlated with HbA1c, while serum iron showed a modest inverse correlation. GGT and creatinine were also significantly higher among patients with poor glycemic control.

As seen in the present work, the strongest link with HbA1c was with age ($p = 0.405$, $p = 0.001$). This aligns with the known variability of T2DM in older individuals, who may have varying levels of complexity, duration of diabetes, concurrent medical conditions, functional status, and medication-related disease risks, all of which could affect glycemic goals and treatment outcomes (30, 31).

Based on our results, mean values of ferritin and iron were significantly reduced in T2DM patients than in healthy controls. This result is coherent with a research done by *Alqahtani et al* which indicates no correlation between ferritin and T2DM (32), as well as a study done by *Wolide et al* find out there was a significant decline in both iron and ferritin in T2DM (33), moreover, study done by *Al Argan et al* confirmed that there was no correlation between ferritin and T2DM (34). Conversely, our study was inconsistent with a study done by *Bayih et al* and other study by *Zhang et al.*, which reported significantly increased serum ferritin in T2DM patients more than non-diabetic control group (35, 36), Reduced ferritin and iron levels may actually be due to factors other than hyperglycemia, such as dietary intake variation, asymptomatic illness, intestinal abnormality and red blood cells disorders.

This study exhibited a positive relationship of serum GGT with HbA1c, and it was significantly elevated in patients with HbA1c levels $\geq 7\%$. The current results are in alignment with *Bahijri et al.*, who stated that GGT elevation was correlated with bad glycemic control and some of the metabolic syndrome markers (27) *Bi et al* also reported that elevation of GGT is strongly related to increased T2DM risk (37). Moreover, no significant difference reported in ALT and AST concentrations among the total diabetic group and the control group. This outcome was inconsistent with a study by *Bi et al.*, which reported an association between ALT and AST elevations and increased T2DM risk (37). An overwhelming number of hepatic metabolic abnormalities cannot be ruled out in the diabetic group. Conventional reference ranges for liver enzyme levels may still be within normal limits in the presence of liver steatosis or early metabolic liver disease. Several studies have reported that liver enzyme concentrations within the normal value may be linked with the development of metabolic syndrome and fatty liver (38).

Creatinine was positively correlated with HbA1c and was elevated in diabetics with poor glycemic control. This observation is compatible with previous reports of an

association between creatinine and glycemic status (39). Nevertheless, serum creatinine alone is an imperfect criterion of kidney function because it is affected by age, sex, muscle mass, and other factors (40). Accordingly, the present study supports an association between serum creatinine and HbA1c but does not establish impaired renal function. If eGFR data can be derived from the available records, reporting eGFR would strengthen the renal assessment.

CONCLUSION

In this retrospective observational study of type 2 diabetics, serum iron and ferritin concentrations were significantly lower than in matched apparently healthy controls, whereas ALT, AST, and GGT did not vary significantly among the groups. Within the T2DM group, GGT and creatinine were positively associated with HbA1c, and GGT was significantly elevated among patients with poor glycemic control. These outcomes support associations between glycemic status and selected biochemical markers but do not establish causality or independent predictive value. Larger, prospective and appropriately adjusted studies are essential to clarify the clinical significance of these associations.

LIMITATIONS

This study has numerous limitations. First, its retrospective observational design bouncer's causal inference. Second, the sample size was relatively tiny and was drawn from a single hospital, which may bouncer's generalizability. Third, the analysis was based on variables available in existing records; therefore, potentially important confounders such as diabetes duration, medication use, body mass index, inflammatory markers, anaemia status, and other metabolic characteristics could not be comprehensively evaluated unless documented in the records. Fourth, serum creatinine was used as the renal-related biochemical parameter, and eGFR was not reported. Finally, multiple biochemical associations were examined without multivariable adjustment; therefore, the findings should be considered exploratory and hypothesis-generating.

Author's Contribution: Solo Authorship.

Conflict of Interest: The study has no conflict of interest to declare by the author.

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