https://doi.org/10.54361/ajmas.269938

Original article

Association Between HbA1c Levels and Iron Deficiency Anaemia Among Diabetic and Non-Diabetic Libyan Patients

Mahmoud Ashawesh*, **Sarah Khalafullah**, **Fatim Albeshri**, **Naema Swisi**, **Nourya Salem**

Department of Medical Laboratories Sciences, Faculty of Medical Technology, the University of Tripoli, Libya
Email. *M.ashawesh@uot.edu.ly*

Abstract
A haemoglobin A1c (HbA1c) is an essential marker for assessing long-term glycemic control and diagnosing diabetes mellitus. However, iron deficiency anaemia (IDA), one of the most common nutritional disorders worldwide, may alter HbA1c levels even in non-diabetic individuals, leading to misinterpretation of glycemic status. This study aims to examine the effect of IDA on HbA1c levels in Libyan patients with and without diabetes, while also exploring the correlation between HbA1c levels and the iron profile. A cross-sectional study was conducted in a period from March to June 2026. The study included 90 participants: 30 diabetic individuals with IDA, 30 non-diabetic individuals with IDA, and 30 healthy individuals. Venous blood samples were collected under sterile conditions, including CBC, blood glucose measurements (Fasting blood glucose (FBS) and HbA1c), and an iron profile assessment. Data were analysed using SPSS v27 and significance was set at $p < 0.05$. The results showed that HbA1c levels differed significantly among the three groups ($p < 0.001$), increasing from the control (5.16%) to non-diabetic IDA participants (5.55%) and reaching the highest values in diabetic IDA patients (8.31%). Notably, HbA1c was significantly higher in non-diabetic IDA subjects than in healthy controls despite normal FBS levels. HbA1c correlated negatively with serum iron and ferritin and positively with total iron-binding capacity (TIBC) ($p < 0.001$). After adjustment for fasting glucose, serum iron remained the strongest independent correlate of HbA1c ($r = −0.30$, $p = 0.004$). IDA was associated with significantly higher HbA1c levels, even in non-diabetic individuals with normal fasting glucose, indicating that iron status can influence HbA1c independently of glycemia. These findings suggest that HbA1c results should be interpreted carefully in patients with IDA to avoid potential misclassification of glycemic status. Assessment of iron parameters alongside HbA1c may improve diagnostic accuracy, and further large-scale studies are recommended to clarify the underlying mechanisms and clinical impact of this independent relationship.
Keywords. Iron Deficiency Anaemia, Anaemia, Diabetes Mellitus, HbA1c, Iron Profile

Introduction

Diabetes mellitus is a chronic disease linked to unusually high levels of the sugar glucose in the bloodstream, characterised by chronic hyperglycemia with disruption of carbohydrate metabolism leading to deficiency in insulin secretion, insulin action or both [1]. The international diabetes federation (IDF) has announced that diabetes mellitus impacts over 634.8 million individuals in 2024, and this number shows a rapid increase over time, with a prevalence rate of 15.8% of adults (ages 20–79), and about 90% of total diabetes cases are type 2 diabetes [2]. Furthermore, IDF has reported that the incidence of diabetes is expected to reach one million cases by the year 2050 [2]. Likewise, the World Health Organisation (WHO) reported that the prevalence and incidence of diabetes in Libya have increased significantly over the past two decades [3]. Unfortunately, a previous publication conducted by A. Eltobgi (2009) has indeed reported that Libya has the highest prevalence of type 2 diabetes in North Africa and in the Arab world as well. This study in particular examined around 4000 cases and found that around 73% of these individuals were encountering type II diabetes [4]. Mainly, there are three types of diabetes mellitus: type I diabetes mellitus (T1DM), type II diabetes mellitus (T2DM) and gestational diabetes mellitus (GDM). T1DM is a condition in which the immune system destroys insulin-making cells in the pancreas [5], while T2DM starts when the cells become unable to use the amount of insulin produced by the pancreas, leading to hyperglycemia. Finally, GDM occurs during pregnancy in women and can end after birth or develop into type II [6].

There is no argument that early diagnosis is crucial for managing diabetes effectively. The condition can be identified through measurements of HbA1c or plasma glucose levels [4,5]. Actually, HbA1c serves as a key biomarker in this context, as it is widely recognised for diagnosing, monitoring, and controlling diabetes [6]. This glycated haemoglobin reflects blood glucose levels over a period of 2-3 months, formed when glucose bonds irreversibly to haemoglobin, making it an essential tool for evaluating long-term glycemic control [7,8]. HbA1c reflects glucose attached to haemoglobin over the lifespan of the erythrocyte (~ 120 days). Thus, significant changes in HbA1c can be clinically identified over a period exceeding 120 days [9]. The American Diabetes Association (ADA) categorises individuals with HbA1c levels ranging from 5.7% to 6.4% as prediabetic, while a value of ≥ 6.5% is considered the diagnostic cutoff for diabetes mellitus [10]. Monitoring these levels plays an important role in managing the disease and preventing its progression. It is an essential method for assessing long-term glycemic control, which informs treatment approaches and patient education [11].

The lifespan of erythrocytes significantly influences HbA1c levels. Conditions that reduce erythrocyte lifespan or increase red cell turnover, such as acute or chronic blood loss and hemolytic anaemia, lead to lower HbA1c levels due to decreased exposure to glucose [12]. Conversely, conditions that extend erythrocyte lifespan or decrease red cell turnover allow for prolonged glucose exposure, resulting in elevated HbA1c levels. Iron anaemia (IDA) has been observed to cause falsely elevated levels of HbA1c in both diabetic and non-diabetic patients [11]. Research indicates that addressing IDA can lead to a reduction in HbA1c levels [12].

Received: *14-07-2026 - **Accepted:** 13-09-2026 - **Published:** 20-09-2026*

Anaemia is a major global health concern, affecting approximately 1.6 billion people worldwide, with a significant proportion suffering from iron deficiency [13]. Hence, there is a potential risk of misclassification or misdiagnosis of diabetes in many populations [14]. Anaemia, particularly IDA, accounts for 50% of global cases [15]. Research examining the impact of IDA on HbA1c levels yields inconsistent findings; some studies indicate that IDA is associated with elevated HbA1c levels, whereas others report a significant reduction in HbA1c among individuals suffering from iron deficiency anaemia when compared to non-anaemic patients. [16,17,18]. IDA can be determined by examining the Complete Blood Count (CBC) to identify anaemia and a serum ferritin test as the most accurate indicator of iron stores. To confirm iron deficiency, it is possible to further assess the serum iron levels, total iron-binding capacity (TIBC), and transferrin saturation [19].

IDA is known to artificially raise HbA1c levels, potentially resulting in the misdiagnosis of diabetes or prediabetes and creating a misleading impression of glycemic control in individuals with an established diabetes diagnosis [20]. Consistent with previous findings, IDA has been shown to influence the levels of HbA1c, highlighting a potential correlation between this nutritional deficiency and glycemic control markers [20, 21, 22]. Accordingly, it is necessary to investigate the impact of IDA on HbA1c levels, as it may lead to potential errors in patient diagnosis or disease monitoring. In Libya, the current situation regarding the effect of IDA on HbA1c levels among Libyan diabetic patients is scarce. This study aims to examine the impact of IDA on HbA1c levels in Libyan patients, both with and without diabetes, while also correlating HbA1c levels of HbA1c with the iron profile.

Material and Methods

Study Design

A cross-sectional study was conducted in several comprehensive primary healthcare settings and clinics in Tripoli between March and July 2026. The research included a total of 90 participants of both genders, aged from 18 to 75, evenly divided into three groups: 30 individuals with diabetes and IDA, 30 non-diabetic individuals with IDA, and a control group of 30 healthy individuals.

Clinical and Laboratory Parameters

According to the ADA, diabetes is diagnosed through fasting blood glucose (FBS) levels, with a normal range of 70-99 mg/dL, pre-diabetes at 100-125 mg/dL, and diabetes at levels above 126 mg/dL. The HbA1c test classifies results as normal (less than 5.7%), pre-diabetes (5.7% to 6.4%), and diabetes (more than 6.5%) [5]. On the other hand, IDA is characterised by reduced haemoglobin concentration accompanied by evidence of depleted iron stores. In adults, anaemia is generally defined as a haemoglobin concentration below 13 g/dL in men and below 12 g/dL in non-pregnant women. Complete blood count (CBC) parameters can provide supportive evidence of IDA; a mean corpuscular volume (MCV) below 80 fL indicates microcytosis. However, red-cell indices are not specific for IDA, and assessment of iron status, particularly serum ferritin, is important for confirming iron deficiency. Normal iron-profile values are approximately 60-150 µg/dL for serum iron, 30-300 ng/mL for ferritin, and 250-450 µg/dL for total iron-binding capacity (TIBC), although reference ranges may vary between laboratories. In established IDA, the typical laboratory pattern includes low haemoglobin, low MCV, low serum ferritin and serum iron, with increased total iron-binding capacity (TIBC) [23].

Inclusion Criteria

Eligible participants were Libyan adults (aged from 18 to 75) attending participating healthcare facilities. Subjects with confirmed iron deficiency anaemia, with or without diabetes mellitus, were included. Diagnosis of IDA was based on haematological and biochemical parameters, including low haemoglobin concentration and reduced serum ferritin levels. All participants provided informed consent and had available HbA1c and relevant laboratory data.

Exclusion Criteria

Patients were excluded from the study if they had conditions such as thalassemia, coagulation disorders, or had received a blood transfusion within the last three months. Those taking iron supplements, vitamin B12, or folic acid, as well as individuals with chronic kidney disease, liver disease, uncontrolled thyroid disorders, or who were pregnant, were also excluded. Additionally, factors that might influence blood glucose levels, including corticosteroid use, severe psychological stress, and acute illnesses at the time of sample collection, were considered as criteria for exclusion.

Data Collection

After receiving informed consent from participants, venous blood samples were obtained from participants, and demographic and clinical data were documented, including sex, age, medical condition, participant identification number (Patient ID), along with the date and time of sampling, particularly for fasting blood sugar (FBS) analysis.

Blood Investigations

This study conducted various blood tests, including CBC, blood glucose measurements (FBS and HbA1c), and an iron profile assessment (serum ferritin, TIBC, and serum iron levels). Blood samples were collected after an overnight fast, specifically 8–12 hours, to accurately estimate fasting blood glucose levels. Different blood collection tubes were utilised: EDTA tubes for CBC and HbA1c analysis, fluoride tubes for fasting blood sugar, and plain tubes for serum iron, ferritin, and

Received: *14-07-2026 -* ***Accepted:*** *13-09-2026 -* ***Published:*** *20-09-2026*

TIBC measurements. CBC was performed using an automated haematology (Sysmex) analyser. FBS, serum iron, TIBC, and HbA1c were measured using the Mindray BS-430 Clinical Chemistry Analyser. Lastly, serum ferritin was measured using the Mindray CL-1200i Chemiluminescence Immunoassay (CLIA) Analyser.

Statistical Analysis

Statistical analysis was performed using appropriate parametric and non-parametric tests to assess differences and associations among the study variables. Data were analysed using SPSS v27. Continuous variables were presented as mean ± standard deviation (SD), and differences among the three study groups were assessed using one-way analysis of variance (ANOVA) and the Kruskal-Wallis test. For significant Kruskal-Wallis results, pairwise comparisons were performed using Dunn's test with Bonferroni adjustment. Comparisons between the control and non-diabetic IDA groups were performed using Welch's independent-samples t-test and the Mann–Whitney U test. Pearson and Spearman correlation coefficients were calculated to assess the relationships between HbA1c and iron-profile parameters, while partial correlation analysis was used to control for fasting blood glucose. Multiple linear regression analysis was performed to determine the independent associations of fasting blood glucose and iron-profile parameters with HbA1c. Significance was set at $p < 0.05$.

Results

Baseline Characteristics of the Study Groups

To examine how IDA affects HbA1c and how HbA1c relates to the body's iron status. Ninety participants were studied in three equal groups of thirty: a healthy control group, a group of non-diabetic patients with IDA, and a group of patients who had both type 2 diabetes and IDA. For each participant, the analysis drew on HbA1c, FBS and a full iron profile including serum iron, ferritin and TIBC, along with the haematological indices. First, the mean values of the main variables for each group were calculated and shown in Table 1. The two anaemic groups showed the laboratory picture expected of iron deficiency: their serum iron and ferritin were markedly lower than in the controls, their TIBC was raised, and their MCV was reduced, consistent with a microcytic anaemia. Fasting glucose confirmed the clinical grouping, being normal in the controls and the non-diabetic IDA group and clearly elevated in the diabetic IDA group. The contrast in the iron markers across the three groups is shown in (Figure 1).

Table 1. Mean (± SD) values of the study variables by group (n = 30 per group).

Variable	Control	IDA non-diabetic	IDA diabetic
HbA1c (%)	5.16 ± 0.29	5.55 ± 0.48	8.31 ± 2.11
Fasting glucose (mg/dL)	89.0 ± 6.5	81.8 ± 13.8	158.9 ± 51.4
Serum iron (µg/dL)	76.4 ± 21.1	40.5 ± 14.3	36.9 ± 19.3
Ferritin (ng/mL)	49.7 ± 20.4	10.4 ± 5.1	16.0 ± 11.3
TIBC (µg/dL)	327.8 ± 38.8	408.4 ± 52.8	412.3 ± 62.9
Haemoglobin (g/dL)	13.8 ± 0.7	8.8 ± 3.9	10.5 ± 2.2
MCV (fL)	87.4 ± 4.3	75.5 ± 6.6	77.6 ± 7.5

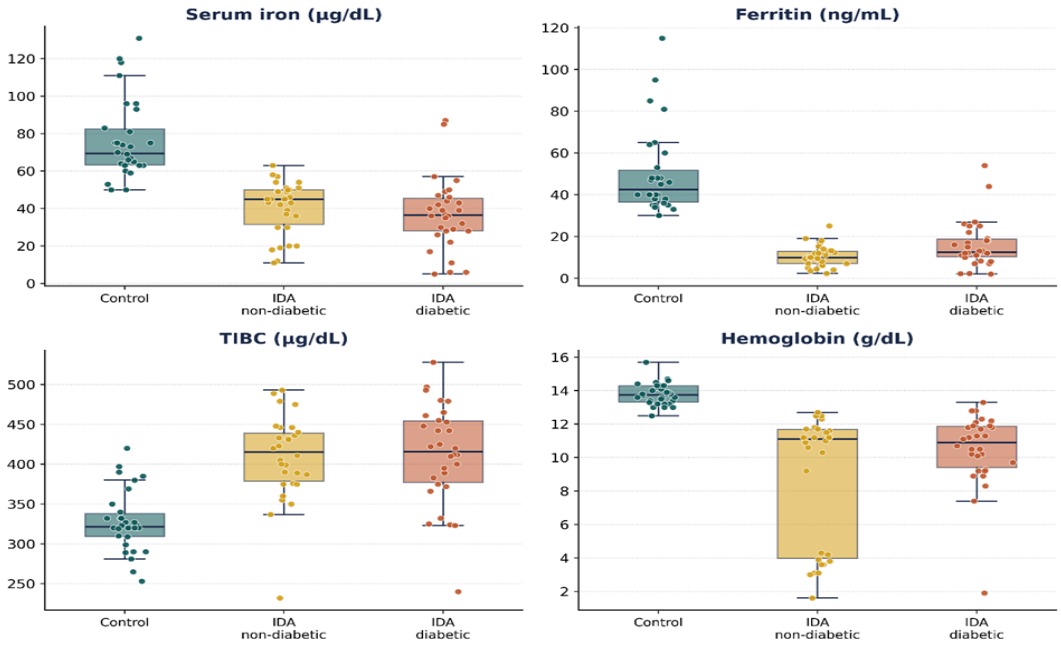

Figure 1. Serum iron, ferritin, TIBC and haemoglobin across the three groups. Both anaemic groups show the characteristic pattern of iron deficiency: low iron, low ferritin, raised TIBC and reduced haemoglobin.

Received: 14-07-2026 - **Accepted:** 13-09-2026 - **Published:** 20-09-2026

HbA1c Across the Three Groups

Next, the Kruskal-Wallis test was performed to evaluate HbA1c level differences among three groups, revealing significant variations ($H = 64.5$, $df = 2$, $p < 0.001$). This finding was further confirmed by a one-way ANOVA test, which showed that the differences in HbA1c levels were both statistically and substantially significant ($F = 55.6$, $p < 0.001$). One-way ANOVA also showed that the effect size was very large ($\eta^2 = 0.56$), indicating that group status explained approximately 56% of the variability in HbA1c values. This suggests that differences in HbA1c were strongly associated with participant group, with HbA1c increasing progressively from controls (5.16%), to non-diabetic IDA participants (5.5%), and reaching the highest levels in diabetic IDA participants (8.31%) (Table 1). Having established significant variations in HbA1c levels among the three groups using the Kruskal-Wallis test, it was necessary to verify which specific groups differed from each other. Thus, Pairwise comparisons (Dunn's test, Bonferroni-adjusted) were performed. As presented in (Figure 2) and (Table 2), the diabetic IDA group differed from both other groups at $p < 0.001$, as expected. More notably, the non-diabetic IDA group also differed significantly from the controls ($p = 0.026$), even though these patients had normal fasting glucose.

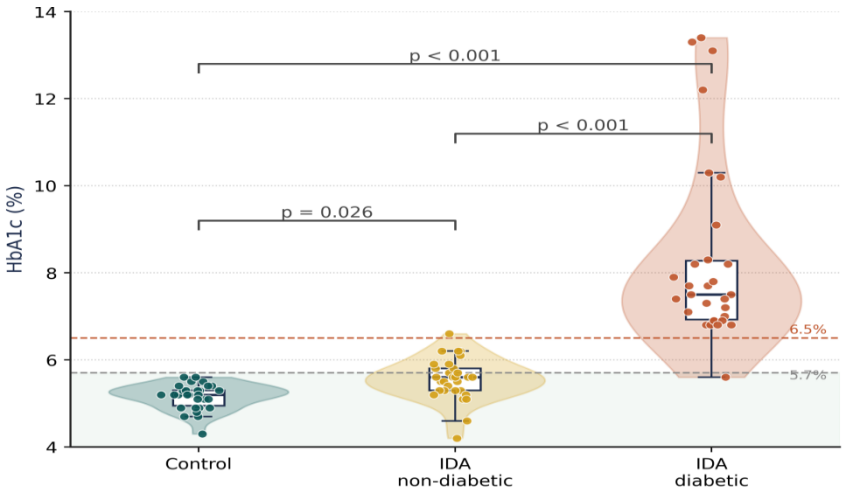

Figure 2. HbA1c by group. The shaded band marks the normal range; dashed lines indicate the 5.7% (pre-diabetes) and 6.5% (diabetes) thresholds. Brackets show the adjusted significance of each pairwise comparison.

Table 2. Pairwise comparison of HbA1c between groups (Dunn's test, Bonferroni-adjusted).

Comparison	Adjusted p	Significance
Control vs IDA non-diabetic	0.026	Significant
Control vs IDA diabetic	< 0.001	Significant
IDA non-diabetic vs IDA diabetic	< 0.001	Significant

The Independent Effect of Iron Deficiency on HbA1c

Because the control and non-diabetic IDA groups both had normal fasting glucose, comparing them isolates the effect of iron deficiency on HbA1c from any effect of hyperglycaemia. Despite their similar and normal glucose levels (89.0 vs 81.8 mg/dL), the non-diabetic IDA patients had a significantly higher HbA1c than the controls (5.55% vs 5.16%). The difference was confirmed by both the Mann–Whitney test ($p < 0.001$) and the Welch t-test ($t = -3.82$, $p < 0.001$), and the effect was large (Cohen's $d \approx 0.99$). In a regression of HbA1c on fasting glucose and ferritin, glucose was a strong positive predictor while ferritin showed an inverse association that approached significance ($p = 0.066$), meaning that lower iron stores tended to accompany higher HbA1c even after glucose was taken into account. Taken together, these results indicate that iron deficiency raises HbA1c in its own right, independently of blood glucose.

Association Between HbA1c and the Iron Profile

To look at the association between HbA1c and the iron profile, across the whole sample, HbA1c was significantly associated with all three iron markers (Figure 3 and Table 3). It correlated negatively with serum iron and ferritin and positively with TIBC, so that lower iron stores and a higher TIBC went hand in hand with higher HbA1c. Serum iron showed the strongest relationship (Pearson $r = -0.45$; Spearman $\rho = -0.54$), followed by TIBC and then ferritin.

Received: *14-07-2026 - **Accepted:** 13-09-2026 - **Published:** 20-09-2026*

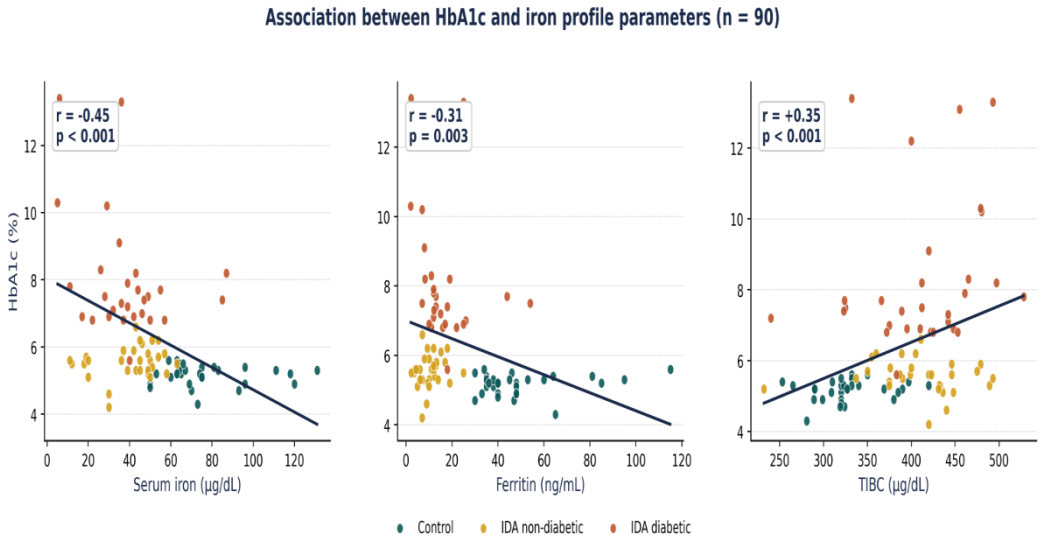

Figure 3. Scatter plots of HbA1c against serum iron, ferritin and TIBC, with the fitted regression line and Pearson correlation for each.

Table 3. Correlation of HbA1c with the iron-profile parameters (n = 90).

Parameter	Pearson r	p	Spearman ρ	Partial r (adj. FBS)
Serum iron	−0.45	< 0.001	−0.54	−0.30 (p = 0.004)
TIBC	+0.35	< 0.001	+0.44	+0.26 (p = 0.012)
Ferritin	−0.31	0.003	−0.40	−0.20 (p = 0.064)

In order to separate the iron effect from the influence of glucose, the correlations were repeated as partial correlations controlling for fasting glucose (last column of Table 3). Serum iron remained a robust correlate of HbA1c (partial r = −0.30, p = 0.004), with TIBC also remaining significant and ferritin falling just short. The relative strength of the three markers is shown in Figure 4. In a multiple regression including glucose and all three iron markers ($R^2 = 0.59$), glucose was the dominant predictor and, among the iron markers, serum iron came closest to independent significance (p = 0.067).

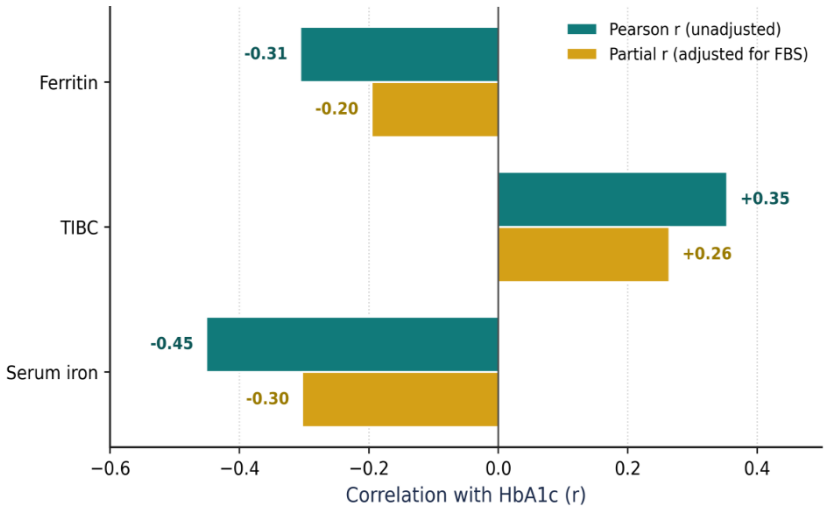

Figure 4. Strength of the association between HbA1c and each iron marker, shown before and after adjustment for fasting glucose. Serum iron is the strongest correlate in both analyses.

HbA1c and the Severity of Iron Deficiency

Finally, the relationship between HbA1c and the degree of iron deficiency was assessed in the non-diabetic IDA group, where the impact of glycaemia is absent. HbA1c was not significantly correlated with ferritin (r = +0.17, p = 0.36) or serum iron (r = +0.21, p = 0.26), and its correlation with haemoglobin was weak (r = +0.38, p = 0.038), suggesting that no clear relationship was found between HbA1c and the severity of the deficiency.

Discussion

This study aimed to evaluate the effect of IDA on HbA1c levels among Libyan diabetic and non-diabetic patients and to investigate the relationship between HbA1c and various iron parameters. The findings demonstrated significant differences among the three study groups, with the highest HbA1c levels observed in diabetic patients with iron deficiency, while

Received: *14-07-2026 -* ***Accepted:*** *13-09-2026 -* ***Published:*** *20-09-2026*

elevated HbA1c levels were also detected in non-diabetic patients with iron deficiency despite normal fasting blood glucose levels, suggesting that iron deficiency may influence HbA1c independently of blood glucose concentration.

The results of this study revealed differences between the three groups in HbA1c levels and iron status indicators. The diabetic group with IDA recorded the highest mean HbA1c level (8.31 ± 2.11) compared to the non-diabetic IDA group (5.55 ± 0.48) and the healthy group (5.16 ± 0.29) (Table 1). Both IDA groups showed lower haemoglobin, serum iron, and ferritin levels, along with elevated TIBC and decreased MCV compared to the control group. The mean haemoglobin levels were 8.8 ± 3.9 g/dL and 10.5 ± 2.2 g/dL in the non-diabetic and diabetic anaemic groups, respectively, compared to 13.8 ± 0.7 g/dL in healthy individuals (Table 1). This confirms the presence of microcytic anaemia due to iron deficiency, which explains the characteristic laboratory findings. These results are consistent with the Coban and Madhu studies, which found that patients with IDA had higher HbA1c levels compared to healthy individuals, and that HbA1c decreased significantly after iron deficiency treatment. The researchers interpreted these results as suggesting that iron deficiency may increase haemoglobin's susceptibility to non-enzymatic glucose binding, in addition to prolonging red blood cell life, leading to an elevated HbA1c that does not reflect the true state of blood glucose control in the patient [24, 25]. In contrast, the results of Sinha and co-workers in 2012 differed from those of the current study. They found that the mean HbA1c level in patients with IDA was lower than in the control group (4.6% vs. 5.5%), and that HbA1c levels increased after iron deficiency treatment. The researchers suggested that other factors, such as dietary differences, anaemia severity, and red blood cell production rate, might influence HbA1c measurement [26].

Furthermore, the study results showed highly significant differences in HbA1c levels among the three groups. The Kruskal-Wallis test demonstrated a strong statistical significance difference between the groups ($H = 64.5$, $df = 2$, $p < 0.001$), confirming a clear difference in HbA1c distribution among the three groups. The results of the one-way ANOVA further supported this conclusion, showing highly significant differences between the groups ($F = 55.6$, $p < 0.001$) with a very large effect size ($\eta^2 = 0.56$). This suggests that group status explains approximately 56% of the variance in HbA1c values, while the remaining 44% is attributed to other potential factors such as age, nutrition, genetics, and normal individual variation. Obviously, the clear upward trend in mean HbA1c levels starts with the lowest values in the control group (5.16%), followed by IDA non- diabetic (5.55%), then IDA diabetic (8.31%, reflecting a strong correlation between iron status and HbA1c levels, in addition to the influence of diabetes status. In fact, some studies support the current findings and confirm that the difference in HbA1c between the groups is not only a statistical difference but also a clinically meaningful difference. For example, Kim *et al*. (2010) reported that iron deficiency was associated with higher HbA1c levels, even among non-diabetic individuals, with iron-deficient women showing an increased likelihood of HbA1c values above 5.5% [27]. Conversely, a systematic review by English and colleagues concluded that the association between IDA and HbA1c remains blurred. The review reported inconsistent findings across studies, suggesting that the influence of iron deficiency on HbA1c may vary according to anaemia severity and other clinical factors [28].

Pairwise comparisons showed that HbA1c levels were significantly higher in diabetic patients with IDA than in both healthy controls and non-diabetic IDA patients ($p < 0.001$) (Table 2). Interestingly, non-diabetic individuals with IDA also had significantly higher HbA1c levels than controls despite having normal glucose levels, supporting an independent effect of iron deficiency on HbA1c. These findings are consistent with previous studies reporting an inverse relationship between iron status and HbA1c, possibly due to prolonged red blood cell lifespan and increased haemoglobin glycation in iron deficiency [29, 30]. Regression analysis further confirmed that while fasting blood glucose was the strongest predictor of HbA1c, reduced iron stores remained associated with higher HbA1c values after adjustment for glucose, indicating that iron deficiency may contribute independently to elevated HbA1c levels. These results agree with those of Soliman *et al*., who reported that IDA is associated with elevated HbA1c levels in both diabetic and non-diabetic individuals. The authors suggested that altered RBC turnover in iron deficiency may increase haemoglobin glycation, leading to higher HbA1c values independent of actual blood glucose levels [31].

To determine whether these associations were solely due to differences in glucose levels, partial correlation analysis was performed after adjusting for FBS. The negative association between HbA1c and serum iron remained significant (partial $r = -0.30$, $p = 0.004$), and the positive association with TIBC also persisted (partial $r = +0.26$, $p = 0.012$) (Figure 4). Although the association with ferritin did not reach statistical significance ($p = 0.064$) (Table 3), it approached significance. These findings suggest that the effect of iron status on HbA1c is not entirely dependent on glucose levels and that iron deficiency may have a partially independent effect on HbA1c values. Moreover, Multiple regression analysis ($R^2 = 0.59$) showed that the studied variables explained 59% of the variation in HbA1c levels, whereas the remaining 41% may be attributed to other unmeasured factors. FBG remained the strongest predictor of HbA1c, while serum iron was closest to independent significance ($p = 0.067$), indicating a possible relationship that did not reach full statistical significance. Collectively, these findings were contradicted by Çetinkaya *et al*. (2021) and consistent with El-Agouza *et al*. (2002) [32, 33].

In regard to the relationship between severity of IDA and HbA1c, no statistically significant associations were observed between HbA1c and iron parameters among non-diabetic individuals. Neither ferritin ($r = +0.17$, $p = 0.36$) nor serum iron ($r = +0.21$, $p = 0.26$) showed significant correlations with HbA1c, whereas haemoglobin demonstrated a weak but statistically significant association ($r = +0.38$, $p = 0.038$). These findings suggest that, in the absence of hyperglycemia, the relationship between iron deficiency and HbA1c may be less pronounced and may be influenced by other factors in non-diabetic individuals with iron deficiency.

In conclusion, this study demonstrated that IDA is associated with higher HbA1c levels in both diabetic and non-diabetic individuals, with the highest values observed in diabetic patients with IDA. Importantly, elevated HbA1c was also detected

Received: *14-07-2026 -* ***Accepted:*** *13-09-2026 -* ***Published:*** *20-09-2026*

in non-diabetic IDA patients despite normal fasting glucose, and statistical analyses confirmed a significant overall difference between groups, with iron status explaining a meaningful proportion of HbA1c variability. These findings indicate that iron deficiency may independently influence HbA1c, potentially leading to misinterpretation of glycemic control if iron status is not considered. Routine assessment of iron parameters alongside HbA1c is advised, particularly in anaemic or borderline glycemic patients. Larger, well-controlled studies are needed to confirm causality and further clarify the underlying mechanisms linking iron metabolism to HbA1c formation.

References

1. Mehta SR, Kashyap AS, Das S. Diabetes mellitus in India: the modern scourge. Med J Armed Forces India. 2009 Jan;65(1):50-4.
2. International Diabetes Federation. IDF diabetes atlas. 11th ed. Brussels: International Diabetes Federation; 2025.
3. World Health Organization. Noncommunicable diseases – Libya. Cairo: WHO Regional Office for the Eastern Mediterranean; [cited 2026 Sep 16]. Available from: https://www.emro.who.int/lby/programmes/noncommunicable-diseases.html
4. Eltobgi A. Libya has the highest prevalence of diabetes mellitus type 2 in North Africa and in the Arab world. Endocr Abstr. 2009;19:138.
5. American Diabetes Association. Classification and diagnosis of diabetes mellitus: standards of medical care in diabetes—2022. Diabetes Care. 2022;45(Suppl 1):S17-S38.
6. Ghanem SS, Abdulkreem EM, Alfurayh MS, Ahmed AA, Rikabi HA. The Role of HbA1c in the Follow-Up and Control of Diabetes Mellitus. Cureus. 2025 Feb 1;17(2):e78357.
7. Kurniawan LB. HbA1c as diabetes mellitus biomarker and its methods evolution. Indones J Clin Pathol Med Lab. 2024;30(2):191-196.
8. Jordaan B, Outhoff K. Haemoglobin A1c (HbA1c): clinical relevance, history, and role in diabetes mellitus management - a South African perspective. S Afr Pharm J. 2025;92(3):46-49.
9. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2026 Jan;49(Suppl 1):S27-S49.
10. American Diabetes Association Professional Practice Committee. 2. Diagnosis and classification of diabetes: standards of care in diabetes—2026. Diabetes Care. 2026 Jan 1;49(Suppl 1):S27-S49.
11. Vasava SN, Sadaria R, Shah T. Effect of iron deficiency anemia on HbA1c levels in patients of diabetes mellitus with controlled plasma glucose levels. Indian J Forensic Med Toxicol. 2021;15(2):3493-8.
12. Mahabeer S. HbA1c measurement and assay interference. Pathologist LANCET Laboratories; 2018 Sep [cited 2026 Sep 16]. Available from: https://www.lancet.co.za/wp-content/uploads/2019/03/Hba1C-measurement-and-assay-interference.pdf
13. Solomon A, Hussein M, Negash M, Ahmed A, Bekele F, Kahase D. Effect of iron deficiency anemia on HbA1c in diabetic patients at Tikur Anbessa specialized teaching hospital, Addis Ababa Ethiopia. BMC Hematol. 2019 Jan 9;19:2.
14. Frezza SJ, Pimentel AL, Lins CJ. Effect of iron deficiency anaemia on HbA1c levels is dependent on the degree of anaemia. Clin Biochem. 2016;49(1-2):117-20.
15. Madhu BG, Shashidhara KC, Akash N, Siddappa AL, Ramaswamy PKH, Srinath KM. Effect of iron deficiency anemia on HbA1c levels among diabetic and nondiabetic patients. DY Patil J Health Sci. 2024;12(2):51-7.
16. Alzahrani BA, Salamatullah HK, Alsharm FS, Baljoon JM, Abukhodair AO, Ahmed ME, et al. The effect of different types of anemia on HbA1c levels in non-diabetics. BMC Endocr Disord. 2023 Jan 28;23(1):24.
17. Mahmood I, Hamza A, Fazry S, Law D, Dutta PD, Najm A. Critical relationship between iron deficiency anaemia (IDA) and glycated haemoglobin (HbA1c): an updated review. Mini Rev Med Chem. 2026;26(6):389-398.
18. Srinath KM, Akash N, Siddappa AL, Madhu BG, Shashidhara KC, Ramaswamy PKH. Effect of iron deficiency anemia on HbA1c levels among diabetic and nondiabetic patients. DY Patil J Health Sci. 2024;12(2):51-7.
19. Bouri S, Martin J. Investigation of iron deficiency anaemia. Clin Med (Lond). 2018 Jun;18(3):242-4.
20. Janice D, Prathima MB, Sushith S, Narayanan R, Reshma S, Nair S, et al. Effect of iron deficiency anaemia over glycated hemoglobin in non-diabetic women. Int J Biochem Mol Biol. 2022 Jun 15;13(3):23-7.
21. Basheer AA. Effect of iron deficiency anemia on HbA1c levels in non-diabetic females in Brack Alshatti. Alq J Med App Sci. 2023 Aug:476-81.
22. Guo W, Zhou Q, Jia Y, Xu J. Increased levels of glycated hemoglobin A1c and iron deficiency anemia: a review. Med Sci Monit. 2019 Nov 7;25:8371-8.
23. Snook J, Bhala N, Beales ILP, Cannings D, Kightley C, Logan RPF, et al. British Society of Gastroenterology guidelines for the management of iron deficiency anaemia in adults. Gut. 2021;70(11):2030-51.
24. Coban E, Ozdogan M, Timuragaoglu A. Effect of iron deficiency anemia on the levels of hemoglobin A1c in nondiabetic patients. Acta Haematol. 2004;112(3):126-8.
25. Madhu BG, Shashidhara KC, Akash N, Siddappa AL, Ramaswamy PKH, Srinath KM. Effect of iron deficiency anemia on HbA1c levels among diabetic and nondiabetic patients. DY Patil J Health Sci. 2024;12(2):51-7.
26. Sinha N, Mishra TK, Singh T, Gupta N. Effect of iron deficiency anemia on hemoglobin A1c levels. Ann Lab Med. 2012 Jan;32(1):17-22.
27. Kim C, Bullard KM, Herman WH, Beckles GL. Association between iron deficiency and A1C levels among adults without diabetes in the National Health and Nutrition Examination Survey, 1999-2006. Diabetes Care. 2010 Apr;33(4):780-5.
28. English E, Idris I, Smith G, Dhatariya K, Kilpatrick ES, John WG. The effect of anaemia and abnormalities of erythrocyte indices on HbA1c analysis: a systematic review. Diabetologia. 2015 Jul;58(7):1409-21.
29. Kumar D, Rasheed T, Zuberi BF, Sadaf R, Ali FS. Correlation of HbA1c with serum iron & transferrin saturation in non-diabetic patients with iron deficiency. Pak J Med Sci. 2023 Jul-Aug;39(4):956-60.
30. Kumar K, Singh A, Ranga G, Kotru M, Maroof KA, Bordoloi T. Impact of iron deficiency anemia on the measurement of HbA1c in non-diabetic population. Asian Hematol Res J. 2024 May 2 [cited 2026 Sep 7];7(2):75-82.
31. Soliman AT, De Sanctis V, Yassin M, Soliman N. Iron deficiency anemia and glucose metabolism. Acta Biomed. 2017 Apr 28;88(1):112-8.

Received: *14-07-2026 -* ***Accepted:*** *13-09-2026 -* ***Published:*** *20-09-2026*

32. Çetinkaya Altuntaş S, Evran M, Gürkan E, Sert M, Tetiker T. HbA1c level decreases in iron deficiency anemia. Wien Klin Wochenschr. 2021 Feb;133(3-4):102-6.
33. El-Agouza I, Abu Shahla A, Sirdah M. The effect of iron deficiency anaemia on the levels of haemoglobin subtypes: possible consequences for clinical diagnosis. Clin Lab Haematol. 2002 Oct;24(5):285-9.

Received*: 14-07-2026 -* ***Accepted****: 13-09-2026 -* ***Published****: 20-09-2026*