JHWCR JOURNAL OF HEALTH, WELLNESS AND COMMUNITY RESEARCH 3007-0570

DOI: https://doi.org/10.61919/rr43nj89

Original Article

Effect of Iron Deficiency Anemia on HbA1c Interpretation in Non-Diabetic Adults

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"Cite this Article" Received: 08 January 2026; **Accepted**: 18 March 2026; **Published**: 26 March 2026

Author Contributions: Concept: WU, NS; Design: WU, NS, AK; Data Collection: WU, AHB, SJS, MR; Analysis: NS, AK, MH; Drafting: WU, NS, AHB, SJS, MR, MH. **Ethical Approval:** Hayat Medical Complex, Dera Ismail Khan, Pakistan. **Informed Consent**: Written informed consent was obtained from all participants; **Conflict of Interest**: The authors declare no conflict of interest. **Funding**: No external funding; **Data Availability**: Available from the corresponding author on reasonable request; **Acknowledgments**: NA

ABSTRACT

Background: Iron-deficiency anemia may alter HbA1c interpretation by affecting erythrocyte turnover and hemoglobin glycation independently of plasma glucose. Objective: To compare HbA1c concentrations between non-diabetic adults with iron-deficiency anemia and non-anemic controls and assess their relationship with fasting plasma glucose and iron-status parameters. Methods: This hospital-based case-control study included 200 adults in Dera Ismail Khan, Pakistan, comprising 100 participants with iron-deficiency anemia and 100 non-anemic controls. Hemoglobin, HbA1c, fasting plasma glucose, serum ferritin, serum iron, total iron-binding capacity, and transferrin saturation were assessed. Groups were compared using independent-samples tests, with $p < 0.05$ considered statistically significant. Results: Mean HbA1c was higher in the iron-deficiency anemia group than in controls (6.18 ± 0.52% vs 5.49 ± 0.41%; mean difference, 0.69%; 95% CI, 0.56–0.82; $p < 0.001$), whereas fasting plasma glucose was comparable (91.4 ± 9.8 vs 89.7 ± 8.9 mg/dL; $p = 0.201$). The iron-deficiency anemia group had lower hemoglobin, serum ferritin, serum iron, and transferrin saturation and higher total iron-binding capacity (all $p < 0.001$). Conclusion: Iron-deficiency anemia was associated with disproportionately higher HbA1c despite similar fasting plasma glucose. Iron status should therefore be considered when HbA1c and plasma-glucose findings are discordant. Keywords: Iron-deficiency anemia; HbA1c; glycated hemoglobin; fasting plasma glucose; ferritin; non-diabetic adults.

INTRODUCTION

Glycated hemoglobin A1c (HbA1c) is widely used for screening, diagnosing, and monitoring diabetes mellitus because it reflects average glycemic exposure during the preceding two to three months. Compared with plasma glucose measurements, HbA1c does not require fasting and is less affected by acute stress, recent food intake, and short-term glycemic fluctuations. International recommendations recognize HbA1c as a diagnostic marker when measurements are performed using standardized assays and when clinical conditions that may interfere with its interpretation are absent (1,2).

Although HbA1c is primarily regarded as an indicator of glycemia, its concentration is also influenced by erythrocyte lifespan, red-cell turnover, hemoglobin structure, and conditions affecting erythropoiesis. Disorders that alter the age distribution or survival of circulating erythrocytes may therefore produce HbA1c values that are disproportionate to actual glucose exposure. Anemia and abnormalities of erythrocyte indices are among the most clinically relevant factors affecting HbA1c interpretation (3).

Iron-deficiency anemia may influence HbA1c through altered erythropoiesis, changes in the proportion of older circulating erythrocytes, modifications in hemoglobin glycation kinetics, and increased oxidative stress, although the relative contribution of these mechanisms remains uncertain (4).

Previous studies have reported higher HbA1c concentrations among non-diabetic individuals with iron-deficiency anemia. Coban et al. observed elevated HbA1c values in non-diabetic patients with iron-deficiency anemia, followed by a reduction after iron replacement therapy (5). Sinha et al. also examined changes in HbA1c associated with iron-deficiency anemia, although the direction and magnitude of the effect have not been consistent across all study populations (6). Analysis of data from the National Health and Nutrition Examination Survey demonstrated an association between iron deficiency and higher HbA1c concentrations among adults without diabetes (7). Other studies have shown that the influence of anemia on HbA1c may vary according to the underlying type of anemia, severity of iron depletion, demographic characteristics, assay method, and accompanying nutritional or inflammatory conditions (8,9).

The potential effect of iron deficiency on HbA1c has important diagnostic implications. Individuals with normal plasma glucose may be classified as having prediabetes or diabetes when HbA1c is interpreted without consideration of hematological status. Studies from South Asian populations have suggested that iron deficiency may contribute to an apparently increased prevalence of prediabetes when HbA1c is used as the principal diagnostic measure (10). More recent investigations have similarly reported variation in HbA1c among non-diabetic individuals with different forms of anemia and among patients with iron-deficiency anemia attending tertiary-care facilities (11–14). Pakistani studies have also demonstrated associations between anemia, iron indices, and HbA1c concentrations in individuals without established diabetes (15,16).

This issue is particularly relevant in Pakistan, where iron deficiency and anemia remain prevalent among children, adolescents, women of reproductive age, and other nutritionally vulnerable populations (17–19). Evidence from Dera Ismail Khan has also documented a substantial local burden of iron-deficiency anemia, although much of the available regional evidence has focused on children rather than the broader adult population (20). Despite the routine use of HbA1c for health screening, preoperative evaluation, and assessment of individuals at risk of diabetes, limited local evidence is available regarding the extent to which iron-deficiency anemia may influence HbA1c interpretation among non-diabetic adults.

The present study therefore aimed to compare HbA1c concentrations between non-diabetic adults with iron-deficiency anemia and non-anemic controls and to examine the relationships of HbA1c with fasting plasma glucose and iron-status parameters. It was hypothesized that participants with iron-deficiency anemia would have higher HbA1c concentrations than controls despite comparable fasting plasma glucose levels.

MATERIALS AND METHODS

A hospital-based case-control study was conducted over a six-month period in the Pathology Department of a tertiary-care teaching hospital in Dera Ismail Khan, Khyber Pakhtunkhwa, Pakistan. The study compared non-diabetic adults with iron-deficiency anemia with non-anemic, non-diabetic controls recruited from the same hospital setting during the same study period. Participants were adults aged 18–60 years who attended the hospital for routine laboratory testing or general health assessment.

Individuals were eligible for inclusion when they had no previous diagnosis of diabetes mellitus and their fasting plasma glucose was outside the diabetic range at the time of assessment. Participants with known diabetes mellitus, impaired renal function, chronic liver disease, hemoglobinopathies, recent blood transfusion, pregnancy, acute infection, or other conditions known to independently alter HbA1c

concentrations or erythrocyte turnover were excluded. Written informed consent was obtained from all participants before enrollment, and personal and laboratory information was handled confidentially.

Cases were non-diabetic adults with iron-deficiency anemia. Anemia was defined as a hemoglobin concentration below 13 g/dL in men and below 12 g/dL in women. Iron deficiency was established using a combination of reduced serum ferritin, reduced serum iron, increased total iron-binding capacity, and decreased transferrin saturation. Low mean corpuscular volume and mean corpuscular hemoglobin were used as supportive hematological findings. Controls were non-diabetic adults with hemoglobin concentrations within the normal range, normal red-cell indices, and no biochemical evidence of iron depletion. Controls were selected from the same source population to reduce differences attributable to setting and recruitment period.

A total of 200 participants were enrolled, comprising 100 individuals with iron-deficiency anemia and 100 non-anemic controls. Participants were recruited through non-probability consecutive sampling until the required sample size was achieved. The sample size was based on the expected between-group difference in HbA1c reported in previous studies of non-diabetic individuals with and without iron-deficiency anemia (5–8).

Demographic and clinical information, including age, sex, dietary history, symptoms, relevant medical history, previous diagnosis of diabetes, medication use, recent transfusion, pregnancy status, and known hematological or systemic disease, was recorded using a structured data-collection form. Participants underwent assessment of fasting plasma glucose, HbA1c, complete blood count, serum ferritin, serum iron, total iron-binding capacity, and transferrin saturation.

Venous blood was collected under aseptic conditions after an overnight fast of 8–10 hours. Samples intended for complete blood count and HbA1c analysis were collected in appropriate anticoagulated tubes, while serum samples were used for biochemical and iron-profile measurements. Fasting plasma glucose was measured using an enzymatic glucose oxidase method. HbA1c was measured using high-performance liquid chromatography with calibration traceable to the National Glycohemoglobin Standardization Program. Serum ferritin, serum iron, and total iron-binding capacity were measured using automated laboratory methods. Transferrin saturation was calculated as serum iron divided by total iron-binding capacity and multiplied by 100.

The primary outcome was HbA1c concentration. The principal exposure was iron-deficiency anemia status. Secondary laboratory variables included fasting plasma glucose, hemoglobin, mean corpuscular volume, mean corpuscular hemoglobin, serum ferritin, serum iron, total iron-binding capacity, and transferrin saturation. Age and sex were considered potential confounding variables. Fasting plasma glucose was used as a concurrent indicator of glycemic status, whereas hematological and iron-profile measurements were used to characterize the presence and severity of iron deficiency.

Laboratory instruments were calibrated according to routine laboratory procedures, and internal quality-control materials were analyzed with each testing batch. The laboratory also participated in applicable external quality-assurance procedures. Samples producing technically invalid or inconsistent results were reassessed before inclusion in the final analysis, and selected specimens were retested to evaluate analytical reproducibility.

Data were analyzed using IBM SPSS Statistics version 25.0. Continuous variables were summarized as mean and standard deviation, while categorical variables were presented as frequency and percentage. The distribution of continuous variables was assessed before inferential analysis. Between-group comparisons of normally distributed continuous variables, including HbA1c and fasting plasma glucose, were performed using the independent-samples t-test. Categorical variables were compared using an appropriate chi-square test or Fisher's exact test. Pearson correlation analysis was used to examine linear

relationships between HbA1c and iron-status parameters when the assumptions for parametric correlation were satisfied.

Multivariable linear regression analysis was used to assess the association between iron-deficiency anemia and HbA1c after adjustment for age and sex. HbA1c was entered as the dependent variable, and iron-deficiency anemia status was entered as the principal independent variable. Statistical significance was defined as a two-sided p-value of less than 0.05. Effect estimates, confidence intervals, correlation coefficients, and regression coefficients were reported where available to support interpretation of the magnitude and precision of the observed associations.

RESULTS

A total of 200 non-diabetic adults were included in the analysis, comprising 100 participants with iron-deficiency anemia and 100 non-anemic controls. Complete group-level data were available for the demographic, hematological, iron-status, and glycemic variables presented below.

Table 1. Demographic and Hematological Characteristics of Participants

Variable	Iron-Deficiency Anemia Group (n = 100)	Non-Anemic Control Group (n = 100)	Mean Difference or Group Comparison	95% CI	p-value
Age, years	37.8 ± 11.2	36.9 ± 10.5	0.90	−2.13 to 3.93	0.558
Male sex, n (%)	42 (42.0)	45 (45.0)			0.669
Female sex, n (%)	58 (58.0)	55 (55.0)			—
Hemoglobin, g/dL	9.6 ± 1.3	13.8 ± 1.1	−4.20	−4.54 to −3.86	<0.001
Serum ferritin, ng/mL	11.2 ± 4.8	64.5 ± 18.2	−53.30	−57.03 to −49.57	<0.001

Values are presented as mean ± standard deviation or n (%). Mean differences were calculated as the iron-deficiency anemia group minus the non-anemic control group. Continuous variables were compared using independent-samples t-tests based on the reported summary statistics. Sex distribution was compared using the Pearson chi-square test. CI, confidence interval.

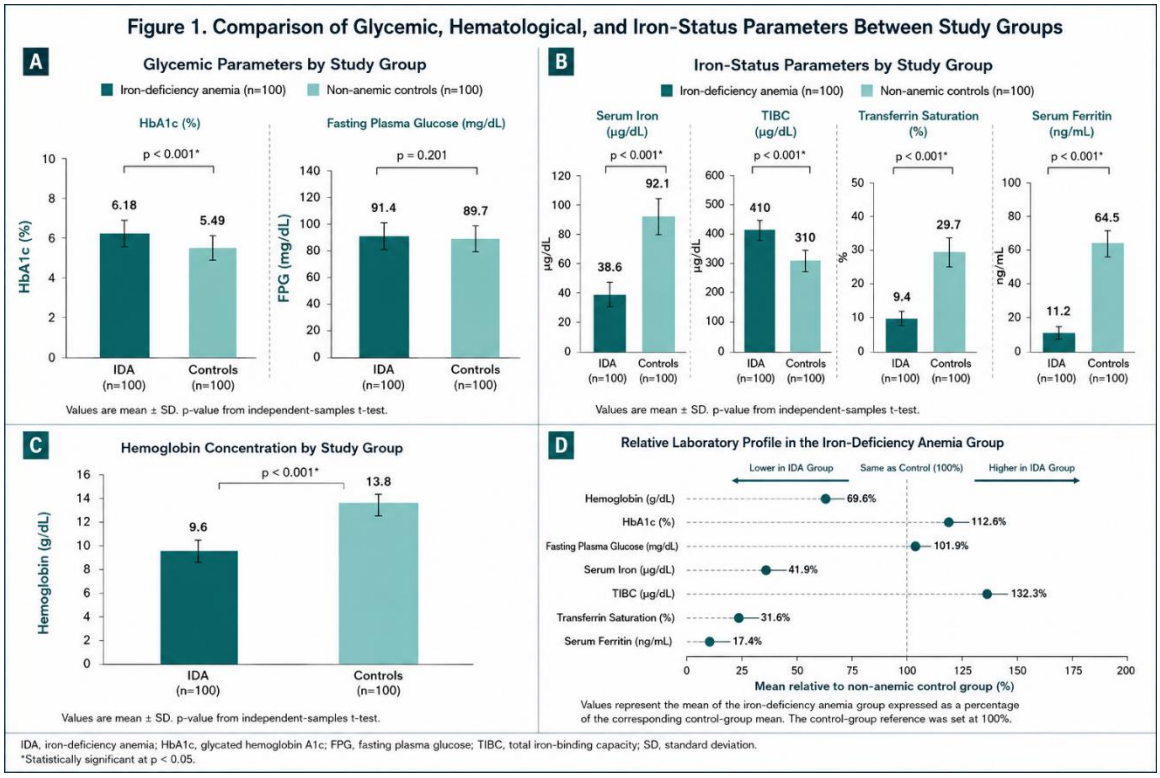

Figure 1 demonstrates that participants with iron-deficiency anemia had significantly higher HbA1c levels despite comparable fasting plasma glucose, alongside markedly lower hemoglobin, serum iron, transferrin saturation, and ferritin and higher total iron-binding capacity than non-anemic controls (all p < 0.001 except fasting plasma glucose, p = 0.201). The relative profile further shows that HbA1c and TIBC were elevated to 112.6% and 132.3% of control values, whereas hemoglobin and iron-status markers were substantially reduced.

The mean age was comparable between the iron-deficiency anemia and control groups, with a between-group difference of 0.90 years (95% CI, −2.13 to 3.93; p = 0.558). Sex distribution was also similar between the groups (p = 0.669). In contrast, participants with iron-deficiency anemia had substantially lower mean hemoglobin concentrations than controls, with a mean difference of −4.20 g/dL (95% CI, −4.54 to −3.86; p < 0.001). Mean serum ferritin was also markedly lower in the iron-deficiency anemia group, with a between-group difference of −53.30 ng/mL (95% CI, −57.03 to −49.57; p < 0.001).

Table 2. Glycemic and Iron-Status Parameters According to Study Group

Variable	Iron-Deficiency Anemia Group (n = 100)	Non-Anemic Control Group (n = 100)	Mean Difference	95% CI	p-value	Hedges' g
Fasting plasma glucose, mg/dL	91.4 ± 9.8	89.7 ± 8.9	1.70	−0.91 to 4.31	0.201	0.18
HbA1c, %	6.18 ± 0.52	5.49 ± 0.41	0.69	0.56 to 0.82	<0.001	1.47
Serum iron, µg/dL	38.6 ± 12.4	92.1 ± 21.3	−53.50	−58.37 to −48.63	<0.001	−3.06
Total iron-binding capacity, µg/dL	410 ± 52	310 ± 44	100.00	86.56 to 113.44	<0.001	2.07
Transferrin saturation, %	9.4 ± 3.1	29.7 ± 6.8	−20.30	−21.78 to −18.82	<0.001	−3.83

Values are presented as mean ± standard deviation. Mean differences were calculated as the iron-deficiency anemia group minus the non-anemic control group. Confidence intervals, p-values, and Hedges' g values were derived from the reported means, standard deviations, and group sizes using independent-samples comparisons. Positive Hedges' g values indicate higher values in the iron-deficiency anemia group, whereas negative values indicate lower values. HbA1c, glycated hemoglobin A1c; CI, confidence interval.

Mean fasting plasma glucose did not differ significantly between the iron-deficiency anemia and control groups. The mean difference was 1.70 mg/dL (95% CI, −0.91 to 4.31; p = 0.201), with a small standardized effect size (Hedges' g = 0.18). In contrast, mean HbA1c was 0.69 percentage points higher in participants with iron-deficiency anemia than in controls (95% CI, 0.56 to 0.82; p < 0.001). The standardized between-group difference in HbA1c was large (Hedges' g = 1.47).

Iron-status parameters differed markedly between the groups. Participants with iron-deficiency anemia had lower mean serum iron by 53.50 µg/dL (95% CI, −58.37 to −48.63; p < 0.001) and lower transferrin saturation by 20.30 percentage points (95% CI, −21.78 to −18.82; p < 0.001). Conversely, mean total iron-binding capacity was 100.00 µg/dL higher in the iron-deficiency anemia group (95% CI, 86.56 to 113.44; p < 0.001). The standardized differences in serum iron, total iron-binding capacity, and transferrin saturation were large, supporting clear biochemical separation between the iron-deficiency anemia and control groups.

Overall, HbA1c was substantially higher among participants with iron-deficiency anemia, whereas fasting plasma glucose was similar between the groups. This discordant pattern indicates that the observed difference in HbA1c was not accompanied by a corresponding difference in fasting plasma glucose. The findings should therefore be interpreted as an association between iron-deficiency anemia and higher HbA1c rather than definitive evidence of altered long-term glycemia or a causal effect of iron deficiency.

DISCUSSION

The present study found that non-diabetic adults with iron-deficiency anemia had substantially higher mean HbA1c concentrations than non-anemic controls despite having similar mean fasting plasma glucose levels. Participants with iron-deficiency anemia also demonstrated the expected hematological and biochemical profile, including lower hemoglobin, serum ferritin, serum iron, and transferrin saturation and higher total iron-binding capacity. The difference in HbA1c was clinically and statistically notable, with a mean between-group difference of 0.69 percentage points and a large standardized effect size. These findings suggest that iron-deficiency status may influence HbA1c interpretation independently of a corresponding difference in fasting plasma glucose, although the observational design does not establish causality or confirm that the elevation was entirely non-glycemic.

The observed HbA1c difference is consistent with previous studies involving non-diabetic individuals with iron-deficiency anemia. Coban et al. reported higher HbA1c concentrations among iron-deficient patients and observed a decline after iron replacement, supporting the possibility that correction of iron deficiency may alter HbA1c without a parallel change in glycemic status (5). Sinha et al. also examined the relationship between iron-deficiency anemia and HbA1c, although the reported direction and magnitude of change differed from some other investigations (6). Population-based analysis by Kim et al. demonstrated an association between iron deficiency and higher HbA1c among adults without diabetes, while Ford et al. showed that the effect of anemia on HbA1c may vary according to anemia type (7,8). These differences indicate that the relationship is influenced by the severity and cause of anemia, erythrocyte kinetics, population characteristics, and analytical methodology.

The present findings also agree with evidence that iron deficiency can affect diabetes classification when HbA1c is used without complementary glucose measurements. Attard et al. found that iron deficiency and anemia could alter the concordance between HbA1c-based and glucose-based classifications of glycemic status (9). Hardikar et al. similarly reported that hematological factors and iron deficiency contributed to an apparently increased prevalence of prediabetes among young Indian adults when HbA1c criteria were applied (10). Studies involving nutritional anemia and iron-deficient women have also demonstrated higher or altered HbA1c concentrations despite the absence of diagnosed diabetes (11,12). More recent evidence has confirmed that different anemia types do not exert uniform effects on HbA1c and that iron-deficiency anemia remains an important source of interpretive uncertainty in clinical practice (13,14).

Regional studies provide additional support for the relevance of this association. Saeed et al. reported that anemia affected HbA1c among individuals with normal glucose tolerance, while Kumar et al. identified relationships between HbA1c, serum iron, and transferrin saturation in non-diabetic patients with iron deficiency (15,16). The present study extends these observations to adults recruited in Dera Ismail Khan and demonstrates a clear discordance between HbA1c and fasting plasma glucose at the group level. This discordance is clinically important because an HbA1c value near or above a diagnostic threshold may be interpreted differently when anemia and depleted iron stores are present.

Several biological mechanisms may explain the higher HbA1c observed in iron-deficient individuals. HbA1c reflects cumulative non-enzymatic glycation of hemoglobin over the circulating lifespan of erythrocytes. Iron deficiency may alter erythropoiesis, shift the distribution of circulating erythrocytes toward older cells, modify hemoglobin structure, and influence oxidative stress and glycation kinetics (3,4). Older erythrocytes have longer exposure to circulating glucose and may therefore contain a greater proportion of glycated hemoglobin. Nevertheless, these mechanisms remain incompletely resolved, and iron-deficiency anemia may not affect HbA1c uniformly across all patients or degrees of anemia. The pathophysiology of iron deficiency is also influenced by nutritional intake, blood loss, inflammation, and comorbid disease (25).

The findings are particularly relevant in Pakistan, where anemia and iron deficiency remain common in several population groups. National and regional studies have documented a substantial burden among women of reproductive age, children, adolescents, university students, and other nutritionally vulnerable populations (17–24). In settings where both iron deficiency and diabetes screening are common, reliance on HbA1c alone may increase the possibility of discordant or misleading classification. Clinicians should therefore interpret HbA1c alongside the complete blood count, relevant iron indices, and plasma glucose measurements when anemia is present or when HbA1c appears inconsistent with the clinical picture.

The study has several strengths. It included equal-sized comparison groups, assessed fasting plasma glucose concurrently with HbA1c, and used multiple hematological and biochemical indicators to characterize iron-deficiency anemia. The marked differences in hemoglobin, ferritin, serum iron, total iron-binding capacity, and transferrin saturation support clear biochemical separation between the

groups. The analysis also demonstrated that the HbA1c difference was accompanied by only a small and statistically non-significant difference in fasting plasma glucose.

The findings should nevertheless be interpreted in light of important limitations. The hospital-based, non-probability sampling method may have introduced selection bias and limits generalizability beyond similar clinical populations. A single fasting plasma glucose measurement does not fully represent average glycemic exposure over the preceding two to three months. Oral glucose tolerance testing, fructosamine, glycated albumin, continuous glucose monitoring, and repeated plasma glucose measurements were not reported. The study also did not reassess HbA1c after iron replacement; therefore, the reversibility of the observed difference could not be evaluated. Residual confounding from inflammation, vitamin B12 or folate deficiency, body mass index, dietary factors, medication use, menstrual blood loss, and other conditions affecting erythrocyte turnover may also have influenced the findings. Furthermore, participant-level correlation and adjusted regression estimates were not available for inclusion in the final analysis, preventing a more detailed assessment of dose-response and independent associations.

Overall, the results indicate that iron-deficiency anemia is associated with higher HbA1c concentrations in non-diabetic adults even when mean fasting plasma glucose is similar. HbA1c should therefore be interpreted as a context-dependent biomarker rather than an isolated measure of glycemia, particularly in patients with anemia or depleted iron stores. Prospective studies incorporating repeated glycemic assessment and HbA1c measurement before and after iron replacement are needed to determine the magnitude, clinical relevance, and reversibility of this association.

CONCLUSION

Non-diabetic adults with iron-deficiency anemia had significantly higher mean HbA1c concentrations than non-anemic controls despite comparable mean fasting plasma glucose, together with marked reductions in hemoglobin, ferritin, serum iron, and transferrin saturation and an increase in total iron-binding capacity. These findings indicate that iron-deficiency status may influence HbA1c interpretation and should be considered when HbA1c results are discordant with plasma glucose or the clinical presentation.

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