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Hematological Manifestations of Iron Deficiency Anemia in Patients with Type 2 Diabetes Mellitus: A Cross-Sectional Analytical Study

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ABSTRACT

Background: Iron deficiency anemia (IDA) represents a prevalent but frequently overlooked comorbidity in type 2 diabetes mellitus (T2DM), potentially distorting glycemic monitoring and accelerating complications. **Methods:** A cross-sectional study was conducted among 200 participants (72 T2DM with IDA, 78 T2DM without IDA, 50 healthy controls) at Fergana Medical Institute of Public Health. Hematological indices, serum iron parameters, HbA1c, fasting glucose, and inflammatory markers were assessed. **Results:** T2DM patients with IDA demonstrated significantly elevated HbA1c (9.6 ± 1.7% vs. 8.2 ± 1.3%, $p < 0.001$), reduced hemoglobin, serum ferritin, and transferrin saturation, alongside markedly impaired renal function and greater diabetes duration. Diabetic nephropathy and peripheral neuropathy were significantly more frequent in the IDA group. **Conclusion:** IDA independently amplifies glycemic dysregulation in T2DM and correlates with advanced microvascular complications, highlighting the necessity of routine iron screening in diabetic care.

Keywords: *iron deficiency anemia; type 2 diabetes mellitus; HbA1c; glycemic control; hematological indices; transferrin saturation; diabetic complications*

INTRODUCTION

Type 2 diabetes mellitus (T2DM) has emerged as one of the most significant global health challenges of the twenty-first century. According to the International Diabetes Federation (IDF), approximately 537 million adults were living with diabetes globally in 2021, with projections estimating this number will exceed 780 million by 2045 [1, 2]. In Central Asian countries, including Uzbekistan, the prevalence of T2DM has shown a sharply rising trajectory over the past decade, attributable to urbanization, dietary transitions, and sedentary lifestyles. The disease imposes an immense burden through its microvascular and macrovascular complications, including diabetic nephropathy, retinopathy, peripheral neuropathy, and cardiovascular disease.

Iron deficiency anemia (IDA) is the most widespread nutritional deficiency disorder worldwide, affecting nearly two billion individuals, predominantly women of reproductive age, children, and populations in low- and middle-income countries [3].

IDA is characterized by inadequate iron stores necessary to sustain normal erythropoiesis, resulting in microcytic, hypochromic red blood cells with diminished oxygen-carrying capacity. While the relationship between T2DM and anemia has long been recognized, the specific intersection of IDA within the diabetic population has received comparatively less systematic attention.

Recent evidence underscores the bidirectional and complex relationship between iron metabolism and glucose regulation. Iron is an essential cofactor in multiple enzymatic processes governing mitochondrial function, oxidative phosphorylation, and insulin biosynthesis. Disruptions in iron homeostasis thus potentially impair insulin secretion and peripheral glucose utilization [4, 5]. Conversely, the chronic inflammatory milieu and associated hepcidin dysregulation in T2DM contribute to functional iron deficiency, even in the presence of adequate dietary iron intake [6, 7]. Furthermore, long-term metformin therapy—the first-line pharmacological agent for T2DM—has been associated with reduced vitamin B12 absorption, and emerging data suggests it may also influence iron absorption through gastrointestinal mechanisms [5].

A particularly critical clinical concern relates to the effect of IDA on glycated hemoglobin (HbA1c), the gold standard for long-term glycemic assessment. IDA accelerates HbA1c glycation by increasing erythrocyte lifespan and reducing erythrocyte turnover, resulting in falsely elevated HbA1c readings that may lead to therapeutic over-intensification and consequent hypoglycemia [8, 9]. Conversely, hemolytic states may produce falsely low HbA1c values, obscuring inadequate glycemic control. These analytical interferences have substantial implications for clinical decision-making in a population where accurate glycemic monitoring is paramount [10].

Despite growing international evidence on this intersection, data from the Fergana region of Uzbekistan remain absent from the scientific literature. T2DM prevalence in Fergana Oblast is rising steeply, while nutritional iron deficiency, especially among women, remains prevalent due to dietary patterns heavily reliant on plant-based iron sources with limited bioavailability [11, 12]. The co-existence of these two conditions in a population with limited specialist healthcare access warrants urgent investigation. This study, therefore, was designed to characterize the hematological manifestations of IDA in T2DM patients attending a tertiary care institution in Fergana, and to evaluate the implications of IDA for glycemic indices and diabetic microvascular complications.

METHODS

Study Design and Setting. A cross-sectional analytical study was conducted between January 2024 and December 2024 at the Department of Endokrinologiya, Ftizatriya va Gematologiya, Fergana Medical Institute of Public Health, Fergana, Uzbekistan.

Ethical approval was obtained from the institutional review board, and all participants provided written informed consent in accordance with the Declaration of Helsinki.

Participants. A total of 200 participants were enrolled, comprising three groups: 72 patients with T2DM and IDA; 78 patients with T2DM without IDA; and 50 healthy age- and sex-matched controls. Patients with T2DM were diagnosed according to the American Diabetes Association 2024 criteria. IDA was defined as hemoglobin < 12 g/dL in women and < 13 g/dL in men, serum ferritin < 12 ng/mL, and transferrin saturation < 16%. Exclusion criteria included known hemoglobinopathies, chronic inflammatory or autoimmune conditions, malignancy, pregnancy, recent blood transfusion within three months, and severe hepatic disease.

Laboratory Assessments. Fasting venous blood samples were drawn under standardized conditions. Complete blood count (CBC) was performed using automated hematology analyzer (Sysmex XN-1000). Serum ferritin, serum iron, and total iron-binding capacity (TIBC) were measured by enzyme-linked immunosorbent assay (ELISA) and colorimetric methods, respectively. HbA1c was determined by high-performance liquid chromatography (HPLC). Fasting plasma glucose was measured by enzymatic hexokinase method. C-reactive protein (CRP) and estimated glomerular filtration rate (eGFR) were assessed by nephelometry and the CKD-EPI equation, respectively.

Statistical Analysis. Data were analyzed using SPSS version 26.0 (IBM Corp., Armonk, NY). Continuous variables are expressed as mean ± standard deviation. Intergroup comparisons were performed using one-way ANOVA with Bonferroni post-hoc correction. Categorical variables were compared using the Chi-square test. Pearson correlation analysis was used to explore associations between iron indices and HbA1c. A p-value < 0.05 was considered statistically significant.

Table 1. Comparison of hematological and metabolic parameters across study groups

Parameter	T2DM with IDA (n = 72)	T2DM without IDA (n = 78)	Healthy Controls (n = 50)	p-value
Hemoglobin (g/dL)	9.8 ± 1.2	12.7 ± 1.1	13.9 ± 0.9	< 0.001
Serum Ferritin (ng/mL)	8.3 ± 3.1	74.2 ± 28.5	98.4 ± 22.1	< 0.001
Serum Iron (µg/dL)	52.1 ± 14.2	87.6 ± 18.4	96.3 ± 15.7	< 0.001
TIBC (µg/dL)	421.3 ± 48.6	364.8 ± 39.2	342.5 ± 31.4	< 0.001

Parameter	T2DM with IDA (n = 72)	T2DM without IDA (n = 78)	Healthy Controls (n = 50)	p-value
Transferrin Saturation (%)	11.2 ± 3.4	22.6 ± 5.1	25.8 ± 4.3	< 0.001
HbA1c (%)	9.6 ± 1.7	8.2 ± 1.3	5.2 ± 0.4	< 0.001
Fasting Glucose (mg/dL)	186.4 ± 42.1	162.3 ± 38.7	89.1 ± 7.4	< 0.001
MCV (fL)	72.4 ± 6.8	86.3 ± 5.2	88.6 ± 4.1	< 0.001
RDW (%)	16.8 ± 2.1	13.2 ± 1.4	12.6 ± 1.1	< 0.001
CRP (mg/L)	7.4 ± 2.8	5.1 ± 1.9	1.8 ± 0.7	< 0.001
eGFR (mL/min/1.73 m²)	54.2 ± 18.3	68.7 ± 15.6	92.4 ± 11.2	< 0.001

T2DM: type 2 diabetes mellitus; IDA: iron deficiency anemia; TIBC: total iron-binding capacity; MCV: mean corpuscular volume; RDW: red cell distribution width; CRP: C-reactive protein; eGFR: estimated glomerular filtration rate. Values are mean ± SD.

RESULTS

Demographic and Clinical Characteristics. The study enrolled 200 participants, including 72 patients with T2DM and IDA (mean age 51.4 ± 9.2 years; 65.3% female), 78 patients with T2DM without IDA (mean age 49.8 ± 8.7 years; 48.7% female), and 50 healthy controls (mean age 48.6 ± 7.9 years). No statistically significant intergroup difference was observed in mean age or BMI. Female sex was significantly more prevalent among T2DM-IDA patients compared to T2DM-non-IDA patients (65.3% vs. 48.7%; $p = 0.041$). Disease duration was significantly longer in the IDA group (9.3 ± 4.6 years vs. 6.8 ± 3.9 years; $p < 0.001$). The demographic and clinical features of all groups are summarized in Table 2.

Table 2. Demographic and clinical characteristics of the study participants

Characteristic	T2DM with IDA (n=72)	T2DM without IDA (n=78)	p-value
Age (years)	51.4 ± 9.2	49.8 ± 8.7	0.271
Female sex (%)	65.3%	48.7%	0.041
BMI (kg/m²)	29.6 ± 4.1	28.8 ± 3.9	0.198

Characteristic	T2DM with IDA (n=72)	T2DM without IDA (n=78)	p-value
Duration of T2DM (years)	9.3 ± 4.6	6.8 ± 3.9	< 0.001
Metformin use (%)	88.9%	79.5%	0.117
Diabetic nephropathy (%)	43.1%	21.8%	0.006
Peripheral neuropathy (%)	51.4%	30.8%	0.011
Vegetarian diet (%)	38.9%	17.9%	0.004

T2DM: type 2 diabetes mellitus; IDA: iron deficiency anemia; BMI: body mass index. Values are mean ± SD or percentage (%).

Hematological Parameters. Patients with T2DM and IDA demonstrated markedly reduced hemoglobin (9.8 ± 1.2 g/dL), serum ferritin (8.3 ± 3.1 ng/mL), serum iron (52.1 ± 14.2 µg/dL), and transferrin saturation (11.2 ± 3.4%) compared to both T2DM-non-IDA patients and healthy controls (all $p < 0.001$). Mean corpuscular volume (MCV) was significantly lower in the IDA group (72.4 ± 6.8 fL vs. 86.3 ± 5.2 fL; $p < 0.001$), confirming a microcytic pattern consistent with iron-deficient erythropoiesis. Red cell distribution width (RDW) was elevated in T2DM-IDA patients (16.8 ± 2.1% vs. 13.2 ± 1.4%; $p < 0.001$), reflecting anisocytosis secondary to heterogeneous iron availability for erythropoiesis. Total iron-binding capacity (TIBC) was significantly higher in the IDA group (421.3 ± 48.6 µg/dL), consistent with compensatory upregulation of transferrin synthesis in iron-depleted states.

Glycemic Parameters. HbA1c values were significantly higher in T2DM-IDA patients (9.6 ± 1.7%) compared to T2DM-non-IDA patients (8.2 ± 1.3%; $p < 0.001$) and healthy controls (5.2 ± 0.4%). Fasting plasma glucose was also elevated in the IDA group (186.4 ± 42.1 mg/dL vs. 162.3 ± 38.7 mg/dL; $p < 0.001$). Pearson correlation analysis revealed a significant negative correlation between serum ferritin and HbA1c ($r = -0.48$; $p < 0.001$) and between transferrin saturation and HbA1c ($r = -0.43$; $p < 0.001$), suggesting that progressive iron depletion is associated with rising glycated hemoglobin values independent of actual glucose control.

Inflammatory Markers and Renal Function. CRP levels were significantly elevated in T2DM-IDA patients (7.4 ± 2.8 mg/L) compared to T2DM-non-IDA patients (5.1 ± 1.9 mg/L; $p < 0.001$) and healthy controls (1.8 ± 0.7 mg/L), suggesting a heightened inflammatory state contributing to functional iron sequestration via hepcidin activation. eGFR was markedly reduced in T2DM-IDA patients (54.2 ± 18.3 mL/min/1.73 m²) relative to T2DM-non-IDA patients (68.7 ± 15.6 mL/min/1.73 m²; $p < 0.001$), consistent with a higher burden of diabetic nephropathy (43.1% vs. 21.8%; p

= 0.006). Peripheral neuropathy prevalence was also significantly greater in the IDA group (51.4% vs. 30.8%; p = 0.011).

DISCUSSION

The findings of this cross-sectional study illuminate the clinically significant and multifaceted intersection between IDA and T2DM in an underrepresented Central Asian patient population. The high burden of IDA among diabetic patients observed in our cohort aligns with previously reported global estimates, which indicate IDA prevalence ranging from 34% to 64% among patients with T2DM [12, 13]. In our study, 48% of enrolled T2DM patients met diagnostic criteria for IDA, a figure that underscores the need for routine iron screening in endocrine clinical practice within Uzbekistan.

The significantly elevated HbA1c values in T2DM-IDA patients merit particular attention. Several well-established mechanisms account for this phenomenon. Iron deficiency prolongs erythrocyte lifespan, increasing the duration each hemoglobin molecule is exposed to circulating glucose and thereby augmenting HbA1c glycation independently of prevailing plasma glucose concentrations [4, 8, 40-42]. Additionally, reduced erythropoietic output in IDA leads to a population of older, more glycated red cells [43, 44]. Our Pearson correlation data (r = -0.48 between ferritin and HbA1c) corroborate these proposed mechanisms, extending prior findings from South Asian and East African cohorts [4, 5, 10] to a previously unstudied Uzbek population. The clinical implication is profound: HbA1c-based intensification of antidiabetic therapy in IDA patients may be inappropriate and potentially harmful, as it may reflect a measurement artifact rather than true glycemic deterioration.

The significantly elevated CRP levels in T2DM-IDA patients support the hypothesis that systemic inflammation, a defining feature of T2DM pathophysiology [54, 55], contributes to iron sequestration via hepcidin upregulation. Hepcidin, the master regulator of systemic iron homeostasis, binds ferroportin on enterocytes, hepatocytes, and macrophages, limiting dietary iron absorption and iron recycling from reticuloendothelial stores [51]. In the inflammatory context of T2DM, chronically elevated hepcidin levels may thus induce or perpetuate functional iron deficiency, creating a pathological feedback loop that simultaneously worsens anemia and augments inflammatory signaling [15, 60].

The association between IDA and microvascular complications observed in our study—specifically the higher rates of diabetic nephropathy (43.1%) and peripheral neuropathy (51.4%) in IDA patients—is consistent with emerging literature linking anemia to accelerated endothelial dysfunction, reduced tissue oxygenation, and impaired nerve conduction [46, 47, 50]. Reduced eGFR in the IDA group likely further contributes to anemia through impaired renal erythropoietin synthesis, a well-

recognized consequence of diabetic nephropathy [49]. These overlapping mechanisms create a vicious cycle wherein renal insufficiency exacerbates IDA, which in turn promotes further glycemic dysregulation and oxidative stress, hastening nephropathic progression [34].

A notable finding was the significantly greater proportion of vegetarian dietary patterns (38.9%) among IDA patients. In Central Asian dietary traditions, while meat consumption is customary, economic constraints among low-income groups, combined with the physiological demands of pregnancy and menstrual losses among female patients, may substantially reduce bioavailable iron intake [23]. The higher prevalence of female sex in the T2DM-IDA group (65.3%) reflects these well-established epidemiological patterns [3, 12]. The longer disease duration in IDA patients (9.3 vs. 6.8 years) may further reflect cumulative iron losses attributable to gastrointestinal effects of chronic metformin use, diminished appetite in elderly diabetic patients, and progressive renal involvement [5, 56, 57].

This study is limited by its cross-sectional design, which precludes causal inference. Dietary iron intake was assessed by self-reported patient history rather than formal dietary recall, and the sample size, while adequate for the primary comparisons, limits subgroup analysis. Future prospective studies should evaluate the impact of iron supplementation on HbA1c accuracy, glycemic outcomes, and complication progression in T2DM patients, using concurrent fructosamine or continuous glucose monitoring as reference glycemic measures [38, 39].

CONCLUSION

Iron deficiency anemia is a highly prevalent and clinically consequential comorbidity in patients with type 2 diabetes mellitus, with a prevalence approaching half of the diabetic patients evaluated at our institution. Beyond causing microcytic anemia and impaired oxygen delivery, IDA profoundly distorts HbA1c—the primary tool of glycemic monitoring—through prolongation of erythrocyte lifespan and accelerated hemoglobin glycation, producing artificially elevated readings that may mislead therapeutic decision-making. The co-existence of IDA with diabetic nephropathy, peripheral neuropathy, and heightened systemic inflammation signals a more advanced and metabolically complex disease phenotype demanding integrated endocrine-hematological management. The strong negative correlations between ferritin, transferrin saturation, and HbA1c observed in this Uzbek cohort provide compelling evidence that IDA correction should precede or accompany glycemic intensification decisions. Routine iron profile screening—including serum ferritin and transferrin saturation—must be incorporated into the standard diabetic care pathway at primary and secondary care levels across Uzbekistan. Early identification and treatment of IDA offers not only the potential to resolve anemia but also to restore the accuracy of glycemic monitoring, reduce the inflammatory burden, and potentially slow the

progression of life-altering diabetic complications. This study lays the foundation for future interventional research and calls for national clinical guideline revisions to address this underrecognized but modifiable dimension of diabetes management.

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