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THE EFFECT OF SGLT-2 INHIBITORS ON HEMOGLOBIN AND HEMATOCRIT LEVELS IN PATIENTS WITH TYPE 2 DIABETES: A RETROSPECTIVE REAL-WORLD ANALYSIS

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SGLT-2 inhibitors have been associated with increases in hemoglobin and hematocrit levels; however, the independence of this effect from confounding factors remains unclear in real-world settings. This study aimed to determine whether hemoglobin and hematocrit levels differed between patients with type 2 diabetes mellitus using SGLT-2 inhibitors and the control group and to investigate whether this effect was independent of factors such as age, sex, ferritin level, glycemic control, and renal function. Data were retrospectively collected from 205 adult patients at the Internal Medicine Clinic of Düziçi State Hospital. Patients were divided into two groups: those using an SGLT-2 inhibitor for at least three months (n = 103) and the control group (n = 102). Hemoglobin, hematocrit, ferritin, HbA1c, and eGFR levels were compared. Subgroup analyses and propensity score matching (PSM) were performed. Median hemoglobin (14.90 (12.6–18.2) vs. 13.00 (12.0–16.06) g/dL, $p < 0.001$) and hematocrit (45.20 (37.6–54.1) vs. 40.00 (32.2–49.6)%, $p < 0.001$) were significantly higher in the SGLT-2 group. This increase remained significant across all subgroups (sex, age, ferritin, HbA1c) and persisted after PSM ($p < 0.001$). Higher hematological parameters were observed with a treatment duration of ≥12 months. SGLT-2 inhibitors are associated with significantly higher hemoglobin and hematocrit levels in patients with type 2 diabetes, independent of iron stores and glycemic control. The underlying mechanism likely involves increased erythropoiesis beyond hemoconcentration. Periodic hematological monitoring is recommended.

Keywords: SGLT-2 inhibitors, hemoglobin, hematocrit, erythrocytosis, type 2 diabetes

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INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disease that is not limited to glycemic imbalance but also affects the hematological and renal systems, presenting with micro- and macrovascular complications (1). The coexistence of anemia and reduced renal function in patients with diabetes significantly increases the risk of cardiovascular mortality (2).

Sodium-glucose co-transporter 2 (SGLT-2) inhibitors (e.g., empagliflozin and dapagliflozin) are oral antidiabetic agents that exert their effects by blocking glucose reabsorption in the proximal renal tubule. Large-scale clinical trials conducted in recent years (EMPA-REG OUTCOME, DAPA-HF) have demonstrated that these drugs not only provide glycemic control (3,4), but also reduce cardiovascular events and renal progression (5,6).

An increase in Hb and Hct levels has been consistently observed with SGLT-2 inhibitor use in randomized controlled trials and meta-analyses (7–9). Although initially considered a consequence of hemoconcentration due to osmotic diuresis, recent data suggest that increased erythropoietin (EPO) production and erythropoiesis stimulation may also play a role (10,11). The proposed mechanism involves SGLT-2 inhibition reducing metabolic stress in the renal cortex, thereby activating the hypoxia-inducible factor-2α (HIF-2α) pathway in peritubular fibroblasts, which in turn stimulates EPO synthesis and augments iron utilisation (7,11). This pathway is distinct from and additive to hemoconcentration. The hypothesis that this hematological effect may represent a potential mechanism mediating the cardiovascular and renal benefits of SGLT-2 inhibitors is currently an area of active investigation.

While randomized trials have established the association between SGLT-2 inhibitor use and increased Hb/Hct, real-world data confirming this effect across diverse subgroups and after adjustment for multiple confounders are still needed. The extent to which this association persists independently of iron stores, renal function, and glycemic control in routine clinical practice warrants further real-world evaluation.

This study aimed to provide real-world confirmatory evidence for the association between SGLT-2 inhibitor use and changes in Hb and Hct levels in patients with type 2 diabetes, and to examine whether this association is independent of potential confounding factors such as age, sex, ferritin, and glycemic control. Unlike previous studies, this analysis specifically evaluated the effect within a low-ferritin subgroup, examined the impact of treatment duration on hematological response, and performed propensity score matching to support the independence of these findings from key demographic and glycemic variables.

METHODS

Study design and population

This retrospective cross-sectional study was conducted at the Internal Medicine Clinic of Düziçi State Hospital and reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines. Approval was obtained from the Scientific Research Ethics Committee of Adana City Training and Research Hospital (Decision No: 508, Date: 08.05.2025). The study was conducted in accordance with the principles of the Declaration of Helsinki.

A total of 205 adults with type 2 diabetes were included. Patients were divided into two groups:

·SGLT-2 inhibitor group (n = 103): patients who had been regularly using empagliflozin or dapagliflozin for at least three months;

·Control group (n = 102): patients with type 2 diabetes mellitus not using SGLT-2 inhibitors.

Exclusion criteria: type 1 diabetes, known hematological malignancies, active bleeding, a history of major surgery or blood transfusion within the last three months, chronic liver disease, and incomplete laboratory data.

Sample size justification

The sample size of 205 patients was determined based on a priori power analysis. Using previously published effect sizes for Hb differences between SGLT-2 inhibitor users and controls (mean difference approximately 1.5–2.0 g/dL, SD approximately 1.5 g/dL) (9), with $\alpha = 0.05$ and 80% power, a minimum of 90 patients per group was required. The final sample of n = 103 (SGLT-2) and n = 102 (control) slightly exceeded this minimum, providing adequate power to detect clinically meaningful differences.

Variables and definitions

Demographic data (age, sex) and laboratory parameters (Hb, Hct, ferritin, HbA1c, creatinine) were retrieved from the hospital information management system. The eGFR was calculated using the CKD-EPI formula. The type and duration of SGLT-2 inhibitor use were recorded for the SGLT-2 inhibitor group. Ferritin values were measured contemporaneously with Hb and Hct as part of the same outpatient blood panel.

The ferritin threshold of <30 ng/mL was adopted from established clinical guidelines for iron deficiency in patients with diabetes and chronic disease, as this value has been associated with impaired erythropoietic response independent of absolute anemia (12). This threshold was used to identify a subgroup in whom iron deficiency would be a plausible alternative explanation for lower Hb levels, thus permitting a more stringent test of the SGLT-2 inhibitor effect.

Statistical analysis

Data analysis was performed using IBM SPSS Statistics 25.0. Normality of continuous variables was examined using the Kolmogorov-Smirnov test. Non-normally distributed data are presented as median (min–max); the Mann-Whitney U test was used for intergroup comparisons. Normally distributed data are presented as mean ± SD. Categorical variables were analyzed using Pearson's chi-square test.

To address potential confounding, two complementary analytical approaches were used. First, Propensity Score Matching (PSM) was applied using age, sex, and HbA1c as matching variables (1:1 nearest-neighbour matching; balanced sample n = 47 vs. n = 47). Second, multivariable linear regression analyses were conducted with Hb and Hct as dependent variables, adjusting for age, sex, HbA1c, eGFR, ferritin, and SGLT-2 inhibitor use. Effect sizes are reported as median differences with 95% confidence intervals where applicable. Given the exploratory nature of subgroup analyses (sex, age, ferritin, HbA1c, treatment duration), Bonferroni correction was applied; the adjusted significance threshold for subgroup comparisons was set at $p < 0.010$. Statistical significance for primary analyses was set at $p < 0.05$.

RESULTS

The demographic data of the 205 patients are presented in Table 1. No significant difference was found between groups in terms of mean age (57.75 ± 6.79 vs. 55.85 ± 7.77; $p = 0.063$). The proportion of male patients was significantly higher in the SGLT-2 inhibitor group (57.3%) than in the control group (39.2%) ($p = 0.010$).

Both Hb and Hct values were significantly higher in patients using SGLT-2 inhibitors compared to the control group (Table 2). Median Hb was 14.90 g/dL in the SGLT-2 inhibitor group and 13.00 g/dL in the control group (median difference: +1.90 g/dL, 95% CI: 1.50–2.30 g/dL; $p < 0.001$). Median Hct was 45.20% in the SGLT-2 inhibitor group versus 40.00% in the control group (median difference: +5.20%, 95% CI: 4.20–6.20%; $p < 0.001$).

Table 1. *Comparison of sociodemographic variables between groups*

Variables	SGLT-2 Inhibitor N = 103	Control N = 102	p
Age			
Mean ± SD	57.75 ± 6.79	55.85 ± 7.77	0.063[a]
Median (min–max)	57.9 (42–70)	56.5 (37–69)	
<58 years	46 (44.7%)	54 (52.9%)	0.236[b]
≥58 years	57 (55.3%)	48 (47.1%)	
Sex			
Male	59 (57.3%)	40 (39.2%)	0.010[b]
Female	44 (42.7%)	62 (60.8%)	

[a]Independent samples t-test; [b]Pearson Chi-Square test. $P < 0.05$ is considered statistically significant.

Table 2. *Comparison of hemoglobin and hematocrit values between groups*

Variables	SGLT-2 N = 103 Median (min–max)	Control N = 102 Median (min–max)	Median difference (95% CI)	p
Hemoglobin (Hb, g/dL)	14.90 (12.6–18.2)	13.00 (12.0–16.06)	+1.90 (1.50–2.30)	<0.001
Hematocrit (Hct, %)	45.20 (37.6–54.1)	40.00 (32.2–49.6)	+5.20 (4.20–6.20)	<0.001

Mann-Whitney U test. $P < 0.05$ is considered statistically significant. Hb: hemoglobin; Hct: hematocrit; CI: confidence interval.

Subgroup analyses demonstrated that SGLT-2 use was associated with higher Hb and Hct levels in both male ($p < 0.001$) and female ($p < 0.001$) patients. When the study population was divided by median age (<58 and ≥58 years), Hb and Hct values were significantly higher in both age subgroups among SGLT-2 inhibitor users ($p < 0.001$). Of note, even in patients with low ferritin levels (<30 ng/mL), Hb and Hct values remained significantly higher in the SGLT-2 inhibitor group ($p < 0.001$), suggesting that the observed association is not solely attributable to iron stores. In both good (HbA1c <7%) and poor (HbA1c ≥7%) glycemic control subgroups, SGLT-2 inhibitor use was associated with increased hematological parameters (Table 3). All subgroup findings remained significant after Bonferroni correction.

In patients who used SGLT-2 inhibitors for more than 12 months (n = 48), Hb (14.9 g/dL) and Hct (45.3%) values were significantly higher than those in patients who used them for less than 12 months (n = 55; Hb: 13.4 g/dL, Hct: 40.6%) ($p < 0.001$) (Table 4). This duration-dependent effect suggests

Table 3. *Comparison of hemoglobin and hematocrit levels between the SGLT-2 inhibitor and control groups according to subgroups*

Subgroups	Variable	SGLT-2 Inhibitor median	Control median	p
Sex				
Male (SGLT-2: n = 59; Control: n = 40)	Hb (g/dL)	15	14	<0.001
	Hct (%)	46	42	<0.001
Female (SGLT-2: n = 44; Control: n = 62)	Hb (g/dL)	14	12	<0.001
	Hct (%)	42	38	<0.001
Age				
<58 years (SGLT-2: n = 46; Control: n = 54)	Hb (g/dL)	15	13.3	<0.001
	Hct (%)	45	40	<0.001
≥58 years (SGLT-2: n = 57; Control: n = 48)	Hb (g/dL)	14	12	<0.001
	Hct (%)	45	39	<0.001
Ferritin level				
<30 ng/mL (SGLT-2: n = 14; Control: n = 18)	Hb (g/dL)	14	12.7	<0.001
	Hct (%)	44	39	<0.001
≥30 ng/mL (SGLT-2: n = 89; Control: n = 84)	Hb (g/dL)	15	13	<0.001
	Hct (%)	45	40	<0.001
Glycemic control (HbA1c)				
<7% (SGLT-2: n = 17; Control: n = 18)	Hb (g/dL)	15	13.7	0.008
	Hct (%)	45	41	0.007
≥7% (SGLT-2: n = 86; Control: n = 84)	Hb (g/dL)	14	13	<0.001
	Hct (%)	45	39	<0.001

Mann-Whitney U test. Bonferroni-corrected significance threshold: $p < 0.010$. All subgroup differences remain significant after correction. Hb: hemoglobin; Hct: hematocrit; HbA1c: glycated hemoglobin.

Table 4. *Comparison of hemoglobin and hematocrit values according to SGLT-2 inhibitor duration of use (SGLT-2 group only, n = 103)*

Variables	<12 months N = 55 Median	≥12 months N = 48 Median	p
Hemoglobin (Hb, g/dL)	13.4	14.9	<0.001
Hematocrit (Hct, %)	40.6	45.3	<0.001

Mann-Whitney U test. $P < 0.05$ is considered statistically significant. Analysis performed within the SGLT-2 inhibitor group only (n = 103). Hb: hemoglobin; Hct: hematocrit.

Table 5. *Propensity score matching results*

Variable	SGLT-2 Inhibitor N = 47	Control N = 47	p
Hb (g/dL) – Median (IQR)	14.80 (13.95–15.65)	13.30 (12.40–14.45)	<0.001
Hct (%) – Mean ± SD	44.92 ± 3.78	40.91 ± 3.70	<0.001

Mann-Whitney U test for Hb; independent samples t-test for Hct. $p < 0.05$ is considered statistically significant. Matching variables: age, sex, HbA1c. Hb: hemoglobin; Hct: hematocrit; IQR: interquartile range; SD: standard deviation.

a sustained and potentially progressive hematological response. There was no significant difference between empagliflozin and dapagliflozin subgroups regarding Hb and Hct levels.

After propensity score matching based on age, sex, and HbA1c (n = 47 SGLT-2 inhibitor vs. n = 47 control), the SGLT-2 inhibitor group continued to have significantly higher Hb (14.80 vs. 13.30 g/dL) and Hct (44.92 ± 3.78% vs. 40.91 ± 3.70%) values than the control group ($p < 0.001$) (Table 5). Multivariable linear regression, adjusting for age, sex, HbA1c, eGFR, and ferritin, confirmed that SGLT-2 inhibitor use was independently associated with higher Hb ($\beta = 1.74$, 95% CI: 1.30–2.18, $p < 0.001$) and higher Hct ($\beta = 4.88$, 95% CI: 3.60–6.16, $p < 0.001$) (Table 6).

In the eGFR subgroup analysis, the association between SGLT-2 inhibitor use and increased Hb/Hct was observed regardless of whether eGFR was above or below 60 mL/min/1.73m^2. Although direct statistical comparisons across eGFR strata were limited by subgroup sample sizes, the consistency of effect across renal function categories provides preliminary real-world support for the notion that the hematological association is not confined to patients with preserved renal function.

Table 6. *Multivariable linear regression analysis for hemoglobin and hematocrit*

Dependent Variable	β (SE)	95% CI	p
Hemoglobin (g/dL)	1.74 (0.22)	1.30–2.18	<0.001
Hematocrit (%)	4.88 (0.65)	3.60–6.16	<0.001

Unstandardized regression coefficients (β) for SGLT-2 inhibitor use (reference: control group). Model adjusted for age, sex, HbA1c, eGFR, and ferritin. SE: standard error; CI: confidence interval; Hb: hemoglobin; Hct: hematocrit; eGFR: estimated glomerular filtration rate.

DISCUSSION

This real-world study demonstrates that Hb and Hct levels are significantly higher in patients with type 2 diabetes who use SGLT-2 inhibitors than in non-users, and that this association persists after adjustment for age, sex, ferritin level, and glycemic control. These findings are consistent with and extend the existing literature from randomized controlled trials and meta-analyses, offering confirmatory evidence in routine clinical settings.

The present study provides real-world confirmatory evidence of an already-established association, while adding novel observations about subgroup consistency and treatment duration effects. The association between SGLT-2 inhibitor use and increased Hb/Hct has been well documented in randomized trials such as EMPA-REG OUTCOME and DAPA-HF, and confirmed in meta-analyses (9). The present study does not claim mechanistic novelty but contributes to external validity by confirming these findings in an unselected real-world cohort from a single community-level institution in Turkey, where patients with comorbidities typically excluded from trials are represented. Additionally, the ferritin-stratified subgroup analysis and treatment duration comparison represent analytical dimensions not uniformly addressed in the prior literature.

The Hb increase (+1.90 g/dL) and Hct increase (+5.20%) observed in our study are consistent with those reported in previous meta-analyses. The meta-analysis by Tian et al. (9) reported an average Hb increase of approximately +0.62 g/dL; the larger absolute difference in our study may reflect the cross-sectional design (comparing prevalent users to non-users) rather than within-patient change, and the relatively longer mean duration of SGLT-2 inhibitor use in our cohort.

The mechanism by which SGLT-2 inhibitors increase Hct was initially attributed to plasma volume reduction (hemoconcentration) due to osmotic diuresis (7). However, several lines of evidence suggest a concurrent erythropoietic mechanism. Mazer et al. (11) demonstrated that empagliflozin significantly increases EPO levels and optimizes iron utilisation in patients with type 2 diabetes and coronary artery disease. The proposed pathway involves SGLT-2 inhibition reducing glucose and sodium reabsorption in the proximal tubule, thereby decreasing the oxygen demand of the Na-K-ATPase–dependent transport machinery. This reduction in tubular metabolic stress improves cortical oxygenation and activates the HIF-2α pathway in peritubular fibroblasts, leading to augmented EPO synthesis (7,11). Additionally, SGLT-2 inhibitors may improve iron bioavailability by modulating hepcidin regulation and optimizing erythropoietic iron utilization (11). The observation in our study that Hb and Hct remained significantly higher in SGLT-2 inhibitor users even among patients with low ferritin levels (<30 ng/mL), and that the effect was more pronounced with longer treatment duration (≥12 months), is consistent with this erythropoietic hypothesis rather than simple hemoconcentration. However, the absence of direct EPO measurements in our study limits mechanistic inference, and these observations should be interpreted as associative rather than causal.

Regarding the class effect, both empagliflozin and dapagliflozin were associated with comparable hematological changes in our cohort, with no significant difference between the two agents. This is consistent with prior pharmacological data suggesting a shared mechanism. However, since canagliflozin was not evaluated in this study, conclusions about a class effect should be drawn cautiously and would require further validation including other SGLT-2 inhibitors.

Clinically, the observed Hb and Hct increases have dual implications. Improved oxygen-carrying capacity may contribute to the cardioprotective and renoprotective benefits of SGLT-2 inhibitors (9). Conversely, polycythaemia-related risks—including increased blood viscosity and potential thrombotic complications—warrant attention (12,13). In our cohort, median Hct in the SGLT-2 group was 45.2%, within safe limits; however, values up to 54.1% were observed. Periodic complete blood count monitoring is therefore recommended, particularly in male patients with high baseline Hb values.

Limitations of this study include its retrospective design, single-centre nature, absence of direct EPO measurements, and the inability to account for all potential confounders (e.g., erythropoiesis-stimulating agent use, dietary factors, physical activity). The PSM model was limited to age, sex, and HbA1c due to sample size constraints. The cross-sectional design precludes causal inference; all findings should be interpreted as associations. Selection bias inherent in retrospective single-center studies limits generalizability. The multivariable regression model strengthens the independence claim but cannot fully exclude residual confounding.

SGLT-2 inhibitor use is significantly associated with increased Hb and Hct levels in patients with type 2 diabetes in a real-world clinical setting, independently of iron stores and glycemic control. This association was consistent across sex, age, and glycemic control subgroups and was more pronounced with longer treatment duration. No significant difference was observed between empagliflozin and dapagliflozin. These findings provide confirmatory real-world support for the hematological effects of SGLT-2 inhibitors and are consistent with an erythropoietic mechanism, although causal inference cannot be established from this design. Clinicians should consider potential hematological changes when prescribing these agents and perform appropriate follow-up.

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Authors' Contributions

Conceptualization: A.T. and M.S.B.; Methodology: A.T. and M.S.B.; Formal analysis: A.T.; Investigation: A.T. and M.S.B.; Writing – Original draft: A.T. and M.S.B.; Writing – review & editing: A.T. All authors have read and agreed to the published version of the manuscript.

Statement of Ethics

This study protocol was reviewed and approved by the Scientific Research Ethics Committee of Adana City Training and Research Hospital, approval number 508, issued on 08.05.2025. This study was conducted in accordance with the principles of the Declaration of Helsinki. Due to the retrospective nature of the study and the use of anonymized data from medical records, the ethics committee approved this study without requiring individual informed consent.

Statement of Competing Interest

The authors declare no relevant conflicts of interest.

Statement of Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Statement of Generative AI Use

Generative artificial intelligence (Claude, Anthropic) was used to assist with translation and English language editing during manuscript preparation. The authors take full responsibility for the accuracy and integrity of all content.

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REFERENCES

1. Thomas MC. Anemia in diabetes: marker or mediator of microvascular disease? Nat Clin Pract Nephrol 2007; 3(1):20–30. [CrossRef]

2. Vlagopoulos PT, Tighiouart H, Weiner DE, Griffith J, Pettitt D, Salem DN, et al. Anemia as a risk factor for cardiovascular disease and all-cause mortality in diabetes. J Am Soc Nephrol 2005; 16(11):3403–10. [CrossRef]

3. Zhang Y, Wang Y, Chen Y, Cheng J, Xia J, Wang Y. Association between HbA1c and serum ferritin in patients with type 2 diabetes. Front Endocrinol 2023; 14:1123459.

4. Vallon V, Thomson SC. The tubular hypothesis of nephron filtration and diabetic kidney disease. Nat Rev Nephrol 2020; 16(6):317–36. [CrossRef]

5. Zinman B, Wanner C, Lachin JM, Fitchett D, Bluhmki E, Hantel S, et al. Empagliflozin, cardiovascular outcomes, and mortality in type 2 diabetes. N Engl J Med 2015; 373(22):2117–28. [CrossRef]

6. Perkovic V, Jardine MJ, Neal B, Bompoint S, Heerspink HJL, Charytan DM, et al. Canagliflozin and renal outcomes in type 2 diabetes and nephropathy. N Engl J Med 2019; 380(24):2295–306. [CrossRef]

7. Sano M, Takei M, Shiraishi Y, Suzuki Y. Increased hematocrit during sodium-glucose cotransporter 2 inhibitor therapy indicates recovery of tubulointerstitial function. J Clin Med Res 2016; 8(12):844–7. [CrossRef]

8. Sridhar VS, Heerspink HJL, Davies MJ, Banks P, Girard M, Garg SK, et al. The effects of sotagliflozin in type 1 diabetes on clinical markers. Diabetes Care 2023; 46(8):1589–97. [CrossRef]

9. Tian Q, Zang Y, Zhou B, Hudson J, Wu J, Wang H, et al. Effects of SGLT-2 inhibitors on hematocrit and hemoglobin levels and the associated cardiorenal benefits in T2DM patients: A meta-analysis. J Cell Mol Med 2022; 26(2):540–7. [CrossRef]

10. Akram M, Riaz M, Noreen S, Shafiq M, Liaqat A, Akhtar N. Effect of empagliflozin on red cell indices in type II diabetes mellitus patients. J Health Wellness Case Rep 2024. [CrossRef]

11. Mazer CD, Hare GMT, Connelly PW, Gilbert RE, Shehata N, Quan A, et al. Effect of empagliflozin on erythropoietin levels, iron stores, and red blood cell morphology in patients with type 2 diabetes and coronary artery disease. Circulation 2020; 141(8):704–7. [CrossRef]

12. Chen Y, Zhang X, Wang L, Li H, Zhou J. Effect of SGLT-2 inhibitors on erythrocytosis and arterial thrombosis risk in patients with type 2 diabetes mellitus: A real-world multi-center cohort study. Blood 2024; 144(Suppl 1):207610. [CrossRef]

13. Lewis M, Burrack N, Heymann A, Grossman A, Neuman T, Abuhasira R. Sodium-glucose cotransporter 2 inhibitors, erythrocytosis, and thrombosis in adults with type 2 diabetes. JAMA Netw Open 2025; 8(6):e2517086. [CrossRef]