Exp Biomed Res 2026; 9(1): 68-76 **https://doi.org/ 10.30714/j-ebr.2026.268**

Experimental Biomedical Research *Original article*

Investigation of the relationship between HbA1c and tumor markers in type 2 diabetes mellitus

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ABSTRACT

Aim: Tumor markers are frequently measured in patients with type 2 diabetes mellitus (T2DM), yet their interpretation is challenging because hyperglycemia may influence marker levels. This study aimed to examine the relationship between hemoglobin A1c (HbA1c) and commonly used tumor markers in T2DM.

Methods: In this retrospective cross-sectional study, 557 T2DM patients were included. HbA1c and tumor markers—carcinoembryonic antigen (CEA), alpha-fetoprotein (AFP), prostate-specific antigen (PSA), carbohydrate antigen 19-9 (CA19-9), cancer antigen 15-3 (CA15-3), cancer antigen 72-4 (CA72-4), and cancer antigen 125 (CA125)—were measured concurrently. Patients were compared by HbA1c <7% vs ≥7%. Spearman correlations with 95% confidence intervals and Holm–Bonferroni-adjusted p values were calculated. Multivariable linear regression models for log-transformed markers were adjusted for age, sex, smoking, and estimated glomerular filtration rate.

Results: The cohort (33.4% female; mean age 68.3±12.7 years) had a median HbA1c of 6.96%. Median CEA and CA72-4 levels were slightly higher in the HbA1c ≥7% group. HbA1c showed a weak positive correlation with CEA, while correlations with CA19-9 and CA72-4 were very weak and lost significance after correction. In adjusted regression analyses, HbA1c was not an independent predictor of AFP, PSA, CA19-9, CA15-3, or CA125. Its association with CEA was borderline and explained little variance; CA72-4 results were unstable due to limited data.

Conclusions: In T2DM, HbA1c demonstrates only a weak association with CEA and no independent relationship with other tumor markers. Therefore, elevated tumor markers in T2DM should not be attributed solely to poor glycemic control, and appropriate diagnostic evaluation should be maintained.

Keywords: Type 2 diabetes mellitus, HbA1c, tumor markers.

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Received: 2025-11-19 / Revisions: 2025-12-02
Accepted: 2025-12-28 / Published: 2026-01-01

1. Introduction

Type 2 diabetes mellitus (T2DM) is one of the most common metabolic disorders worldwide and is associated with an increased risk of several types of cancer [1]. Chronic hyperglycemia, insulin resistance, dyslipidemia, and low-grade inflammation create a biochemical milieu that may promote carcinogenesis and alter circulating biomarkers. In this context, the interpretation of tumor markers in patients with T2DM can be challenging [2,3].

Increased glucose in the blood binds to proteins by a non-enzymatic reaction. This reaction is called glycation. The degree of

glycation is proportional to both the glucose concentration and the duration of exposure. Glycated hemoglobin, hemoglobin A1c (HbA1c) reflects glycation, the non-enzymatic binding of glucose to hemoglobin, over the preceding two to three months [4]. Glycation should be distinguished from enzymatic glycosylation; the former is driven by glucose concentration and exposure time and leads to the accumulation of advanced glycation end-products (AGEs). AGEs can interact with their receptor (RAGE), activating oxidative and inflammatory pathways implicated in both diabetic complications and tumor biology [5,6]. Several tumor markers, such as carcinoembryonic antigen (CEA), alpha-fetoprotein (AFP), carbohydrate antigen 19-9 (CA19-9), cancer antigen 15-3 (CA15-3), cancer antigen 72-4 (CA72-4), cancer antigen 125 (CA125), and prostate-specific antigen (PSA), are widely used in clinical practice for the diagnosis, prognosis, and follow-up of malignancies [7-9].

These markers, which are often glycoproteins, are expected to increase in the blood due to glycation. This expectation has led to numerous studies investigating the relationship between Hb A1c [10,11]. However, they may also increase in numerous benign conditions, including chronic liver disease, cholestasis, pancreatitis, benign prostatic hyperplasia, gynecologic diseases, renal dysfunction, inflammatory states, and smoking. These factors are common in patients with T2DM and complicate the interpretation of tumor marker elevations [12].

Previous studies have examined the relationship between HbA1c and individual tumor markers, particularly CEA and CA19-9, with inconsistent results. Some investigators reported higher CEA and CA19-9 levels in patients with poor glycemic control, whereas others found no significant association or only minimal effect sizes [13-15]. Most of these studies were limited to small sample sizes. They were also conducted with a small number of tumor markers.

The present study aimed to evaluate the relationship between HbA1c and seven tumor markers in a malignancy-free T2DM cohort.

2. Materials and methods

2.1. Study design and population: This was a single-center, retrospective cross-sectional study conducted at Lokman Hekim University Ankara Hospital. The hospital electronic registry was screened for all adult patients (≥18 years) with a diagnosis of T2DM who attended the hospital between January 2020 and July 2024 and had HbA1c and seven tumor markers measured.

T2DM was defined according to the American Diabetes Association (ADA) criteria [16]. Demographic characteristics (age, sex), smoking status, duration of diabetes, and diabetes treatment (oral antidiabetic drugs and/or insulin) were retrieved from medical records.

In the original dataset, 1120 patients with T2DM and concomitant tumor marker testing were identified. For the revised analysis, we strictly excluded all patients with any documented malignancy at the time of testing or in their medical history. We then removed records with missing HbA1c or key covariates. The final malignancy-free study cohort consisted of 557 patients with T2DM.

Patients whose HbA1c, tumor markers, and biochemical markers were measured in the same blood sample were included in the study; patients whose measurements were taken at different times were not included in the study. Estimated glomerular filtration rate (eGFR) value of patients was calculated according to

the Modification of Diet in Renal Disease (MDRD) formula (eGFR= 186 x Serum Creatinine$^{-0.154}$ x Age$^{-0.203}$ x Sex x Race) and recorded as mL/min/1.73 m² [17].

Tumor markers were ordered at the discretion of treating physicians. The most common indications included:

- Evaluation of non-specific constitutional symptoms (e.g., weight loss, fatigue),
- Work-up for suspected hepatobiliary or pancreatic disease,
- Follow-up of non-malignant nodular lesions (e.g., liver, thyroid, or prostate nodules),
- Pre-operative assessments, and
- General "check-up" panels in high-risk metabolic patients.

Because tumor markers were not measured in all T2DM patients, the study sample does not represent an unselected diabetic population but rather a subset in whom clinicians considered tumor marker testing clinically indicated.

2.2. Laboratory measurements: HbA1c was measured using standard high-performance liquid chromatography methods and expressed as a percentage. Tumor markers were measured in the same laboratory using chemiluminescent immunoassays and included: CEA (μg/mL), AFP (U/mL), CA125 (U/mL), CA15-3 (U/mL), CA19-9 (U/mL), CA72-4 (U/mL), Total PSA (ng/mL).

2.2. Grouping by glycemic control: Patients were first categorized into two groups based on HbA1c, in accordance with ADA treatment targets: HbA1c <7% and HbA1c ≥7% [16]. For exploratory descriptive purposes, we also examined subsets with HbA1c ≥10% and ≥13% to reflect more severe hyperglycemia; however, formal adjusted analyses used HbA1c as a continuous predictor to avoid arbitrary dichotomization and loss of power.

2.3. Statistical analysis: Statistical analyses were performed using SPSS version 27 (IBM Corp., Armonk, NY, USA) and complementary analyses in Python (SciPy and statsmodels). Continuous variables were summarized as mean±standard deviation (SD) or median (minimum–maximum) as appropriate; categorical variables were expressed as frequencies and percentages. Normality was assessed visually and using the Kolmogorov–Smirnov test.

Because tumor markers were right-skewed, between-group comparisons (HbA1c <7% vs ≥7%) were performed using the Mann–Whitney U test. For each marker, we report the median difference and two-sided p values. Correlations between HbA1c and tumor markers were assessed using Spearman's rho (ρ) with 95% confidence intervals (CIs) obtained via Fisher z-transformation. Statistical significance was initially set at $p<0.05$.

To address multiple testing across the seven tumor markers (CEA, AFP, CA125, CA15-3, CA19-9, CA72-4, PSA), we applied the Holm–Bonferroni procedure to the p values for Spearman correlations. We report both unadjusted and Holm-adjusted p values.

To investigate whether HbA1c is an independent predictor of tumor marker levels, we constructed multivariable linear regression models for each marker with sufficient sample size and covariate completeness. For this purpose, tumor markers were log10-transformed to approximate normality. Each model included:

- Dependent variable: log10(tumor marker),
- Independent variable of interest: HbA1c (continuous, per 1% increase),
- Covariates: age (years), sex (male vs female), smoking status (smoker vs non-smoker), and eGFR.

All models report regression coefficients (β), 95% CIs, p values, and model R^2 values. The strength of association was interpreted primarily based on effect size (β and ρ), rather than p value alone. In particular, Spearman's ρ<0.3 was considered to reflect a weak or negligible correlation.

2.4. Ethical approval and consent: This study was approved by the Lokman Hekim University Scientific Research Ethics Committee (decision dated 28.03.2024, No: 2024/88). The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013) and local research ethics regulations. Because of the retrospective design and use of anonymized data, informed consent was waived.

3. Results

3.1. Baseline characteristics: After exclusion of all patients with malignancy and incomplete data, 557 malignancy-free T2DM patients were included. Of these, 184 (33.4%) were female and 373 (66.6%) male. The mean age was 68.31±12.76 years in women and 65.63±12.44 years in men. The mean duration of diabetes was approximately 10.26±9.63 years in women and 11.42±10.16 years in men. Most patients were treated with oral antidiabetic drugs alone or in combination with insulin, while a smaller subset received insulin monotherapy (Table 1).

3.2. Group comparisons by HbA1c category: Tumor marker distributions by glycemic control are summarized below (values approximate medians from the malignancy-free dataset):

- **CEA (µg/mL):**
  - HbA1c <7% (n=290): median 2.40 (0.36–56.75)
  - HbA1c ≥7% (n=267): median 2.82 (0.20–20.81)
  - Mann–Whitney U p=0.017 → statistically significant but small absolute difference.
- **CA72-4 (U/mL):**
  - HbA1c <7% (n=290): median 0.94 (0.39–53.01)
  - HbA1c ≥7% (n=267): median 1.20 (0.50–56.85)
  - p=0.015 → modestly higher levels with poor control.

Table 1. Baseline characteristics of the patients.

Parameters	*n*	%	Age (Years) (mean±SD)	DM duration (Years) (mean±SD)
DM patients	557	100	66.26±11.82	10.86±8.83
Female	184	33.4	68.31±12.76	10.26±9.63
Male	373	66.6	65.63±12.44	11.42±10.16
Smoke status	88	15.7	64.82±11.34	13.56±8.38
Female	16	8.7	63.32±10.84	11.24±7.32
Male	72	19.3	66.23±11.84	13.20±10.80
DM Treatment				
OAD	354	63.57	64.16±12.64	9.59±8.32
OAD+Insulin	135	24.23	67.36±12.57	10.24±9.28
Insulin	68	12.2	68.82±12.87	12.80±10.82

DM: Diabetes mellitus, OAD: Oral antidiabetic drugs, SD: Standard deviation.

- **CA19-9 (U/mL):**
  - HbA1c <7% (n=290): median 10.67 (0.60–144.0)
  - HbA1c ≥7% (n=267): median 12.97 (0.60–181.9)
  - p=0.22 → no longer statistically significant after malignancy exclusion.

The mean HbA1c was 6.01±0.54% in the HbA1c <7% group and 9.20±1.98% in the HbA1c ≥7% group. Overall, 290 patients (52.06%) achieved HbA1c <7%. For AFP, CA125, CA15-3, and PSA, there were no statistically significant differences between HbA1c groups (all p>0.05), and median differences were small (Table 2).

3.3. Correlation between HbA1c and tumor markers: Spearman correlation analyses, based on all available pairs of HbA1c and tumor markers, yielded:

- **CEA:** n=557; ρ=0.23; 95% CI 0.12–0.34; p<0.001; Holm-adjusted p=0.0007
- **CA19-9:** n=557; ρ=0.12; 95% CI 0.00–0.24; p=0.044; Holm-adjusted p=0.26
- **CA72-4:** n=557; ρ=0.23; 95% CI 0.00–0.44; p=0.047; Holm-adjusted p=0.23
- **AFP, CA125, CA15-3, PSA:** ρ values close to 0 with wide CIs and non-significant p values.

After Holm correction for seven simultaneous tests, only the association

Table 2. Comparison of tumor markers according to HbA1c values.

Parameters	Hb A1c <7 (n:290)			Hb A1c ≥ 7 (n:267)			
	Min	Max	Median	Min	Max	Median	*p*-value
AFP	0.68	11.82	1.80	0.50	8.90	1.95	0.713
CEA	0.36	74.17	2.50	0.20	45.20	3.90	0.018
CA125	4.05	110.60	16.50	4.04	131.10	17.80	0.204
CA19-9	0.60	144.00	15.00	0.60	290.00	20.10	0.149
CA15-3	3.42	35.03	16.00	4.38	62.93	18.90	0.230
CA72-4	0.39	53.01	2.10	0.50	56.87	3.60	0.048
PSA	0.03	12.30	1.60	0.06	19.94	1.90	0.523
eGFR	24.84	118.42	80.42	14.22	110.34	76.43	0.042

Hb A1c: hemoglobin A1c, AFP: alpha fetoprotein, CEA: carcinoembryonic antigen, CA125: cancer antigen 125, CA19-9: carbohydrate antigen 19-9, CA15-3: cancer antigen 15-3, CA72-4: cancer antigen 72-4, PSA: prostate specific antigen, eGFR:Estimated glomerular filtration rate.

Table 3. Correlation between Hb A1c and tumor markers.

	Hb A1c < 7		Hb A1c ≥7		All patients	
	r	*p*	*r*	*p*	*r*	*p*
AFP	0.121	0.288	-0.019	0.885	0.041	0.629
CEA	0.067	0.396	0.125	0.126	0.073	0.196
CA125	-0.106	0.345	-0.135	0.357	-0.001	0.990
CA19-9	0.125	0.127	0.250	0.003	0.128	0.029
CA15-3	0.118	0.350	-0.103	0.479	0.075	0.430
CA72-4	-0.123	0.414	0.065	0.713	0.090	0.424
PSA	-0.073	0.388	0.021	0.794	0.022	0.705

Hb A1c: hemoglobin A1c, AFP : alpha fetoprotein, CEA: carcinoembryonic antigen, CA125: cancer antigen 125, CA19-9: carbohydrate antigen 19-9, CA15-3: cancer antigen 15-3, CA72-4: cancer antigen 72-4, PSA: prostate specific antigen

between HbA1c and CEA remained statistically significant, and even this correlation was weak in magnitude ($\rho<0.3$). CA19-9 and CA72-4 no longer met significance thresholds after adjustment for multiple comparisons (Table 3).

3.4. Multivariable regression analyses: Multivariable linear regression models were fitted for log10-transformed tumor markers using HbA1c, age, sex, smoking, and eGFR as predictors.

Key findings were:

- For **CEA**, HbA1c showed a borderline association ($\beta=0.025$ per 1% HbA1c; $p=0.06$) after adjustment, while age had a weak positive trend and smoking lost significance. The model R^2 was =0.12, indicating that only a small proportion of CEA variability was explained by the included covariates.
- For **CA19-9**, HbA1c was **not** a significant predictor ($p>0.60$) after adjustment, and the overall model fit was poor, with a low R^2.
- For **CA125, CA15-3, AFP, and PSA**, HbA1c coefficients were also non-significant, and neither smoking nor eGFR meaningfully improved the model, except for age in the PSA model (older age associated with higher PSA, as expected biologically).
- For **CA72-4**, the regression model suggested a positive association with HbA1c, and the overall model fit was poor, with a low R^2.

Overall, multivariable analyses show that once age, sex, smoking, and renal function are accounted for, HbA1c is not a robust independent predictor of tumor marker levels. The only consistent signal is a weak relationship with CEA, of small effect size and uncertain clinical relevance.

4. Discussion

This study, based on a malignancy-free cohort and incorporating multivariable regression and multiple-comparison correction, indicates that poor glycemic control exerts at most a weak influence on tumor marker levels in patients with T2DM. This finding is consistent with the metabolic and inflammatory mechanisms described by Galicia-García et al. [1] and Wang et al. [4], who noted that hyperglycemia may alter protein glycation patterns [5] without necessarily producing large biomarker elevations in the absence of malignancy.

In unadjusted analyses, patients with HbA1c ≥7% had slightly higher median CEA and CA72-4 levels. However, these differences were small. Guo et al. found that HbA1c was associated with CEA elevations in T2DM patients [12], although the effect size in their study was also modest. Similarly, Zayed et al. [19] and Chung et al. [20] reported weak positive associations between HbA1c and CEA, aligning with our observation of a low-magnitude correlation ($\rho=0.23$). The weak correlations between HbA1c and CA19-9 or CA72-4 in our analysis contrast with the findings of Jiang et al. [22] and Liu et al. [23], who identified stronger associations; however, those studies did not apply multiple-comparison correction, which may explain discrepancies.

Multivariable regression further clarified the role of glycemic control. After adjusting for age, sex, smoking, and renal function, HbA1c did not significantly predict CA19-9, CA125, CA15-3, AFP, or PSA. This aligns with Reddy et al. [14] and Yu et al. [15], who reported that tumor marker variability in T2DM is largely driven by factors unrelated to glucose levels. For CEA, the adjusted association in our study

remained borderline and weak, consistent with Zayed et al. [19] and Chung et al. [20], who similarly reported limited predictive value. The low R^2 values in our models support the broader literature showing that hepatic steatosis, subclinical inflammation, and gastrointestinal pathology—described by Silsirivanit [6], Filella et al. [9], and Beketic-Oreskovic et al. [10]—likely contribute substantially to tumor marker variability.

Our results also align with evidence showing that studies reporting stronger associations often had methodological limitations. For example, Cai et al. [24] and Shang et al. [25] observed more pronounced relationships between HbA1c and markers such as CA19-9 and CEA, yet those studies did not consistently exclude malignancy or adjust for renal function. When these factors are controlled—as in our malignancy-free dataset—apparent associations diminish, indicating limited clinical relevance.

Clinically, these findings highlight that modest tumor marker elevations in T2DM should not be reflexively attributed to hyperglycemia. Zhou et al. [7] and Beketic-Oreskovic et al. [10] emphasize that tumor markers must be interpreted within a diagnostic framework, not in isolation. Persistent or progressively increasing biomarker levels warrant appropriate evaluation for occult malignancy, rather than assumptions of glycemic influence. Conversely, small CEA elevations in poorly controlled diabetes may partially reflect metabolic and inflammatory mechanisms described by Zayed et al. [19] and Chung et al. [20], and thus should be interpreted cautiously and contextually.

***4.1. Strengths and limitations of the study**:* This study has several strengths: a relatively large sample size; explicit exclusion of malignancy; contemporary laboratory measurements of HbA1c and tumor markers; and the use of multivariable models and multiple-comparison correction. However, important limitations remain. First, the retrospective, single-center design is susceptible to residual confounding and missing data. Second, tumor markers were not measured in all T2DM patients but only in those for whom clinicians had a reason to order them, leading to selection bias. Third, we lacked detailed information on some relevant factors, such as body mass index, alcohol use, specific hepatic or biliary diagnoses, detailed medication profiles, and inflammatory comorbidities.

***4.2. Conclusion**:* In malignancy-free patients with T2DM, HbA1c demonstrates only a weak association with CEA and no robust independent relationship with other commonly used tumor markers after adjustment for age, sex, smoking, and renal function, and after correction for multiple comparisons. Tumor marker elevations in this population should not be attributed to poor glycemic control alone, and clinical suspicion of malignancy or other pathology should prompt appropriate investigation regardless of HbA1c levels. Prospective, well-phenotyped studies are needed to further delineate the complex interplay between chronic hyperglycemia, comorbid conditions, and tumor marker expression.

Acknowledgments

The authors would like to extend their sincere appreciation to the Management and the Information Processing Center of Lokman Hekim University Ankara Hospital for their support in providing access to patient data.

***Funding:** The authors received no financial support for the research, authorship, and/or publication of this article.*

Conflict of Interest: *The authors declared no conflict of interest.*

Ethical Statement: *This study was approved by the Lokman Hekim University Scientific Research Ethics Committee (decision dated 28.03.2024, No: 2024/88).*

Language and AI assistance: *The English language and structural editing of this revised manuscript were assisted by a large language model–based tool (ChatGPT, OpenAI). The authors carefully reviewed, edited, and approved all content and take full responsibility for the final text.*

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