

Original Article

Limited predictive value of HbA1c, fructosamine, hemoglobin glycation index and glycation gap for early kidney injury in newly diagnosed type 2 diabetes mellitus

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Abstract

This study aims to evaluate the predictive value of protein glycation markers, including glycated hemoglobin (HbA1c), fructosamine, glycation gap (GG), and hemoglobin glycation index (HGI), for early kidney injury in patients with newly diagnosed type 2 diabetes mellitus (T2DM). A cross-sectional analytical study was conducted in 103 patients with newly diagnosed T2DM. Early kidney injury was assessed using the urinary albumin-to-creatinine ratio (uACR). The predictive performance of HbA1c, fructosamine, GG, and HGI for albuminuria was evaluated using univariate and multivariable logistic regression analyses adjusted for age, sex, and body mass index (BMI), along with receiver operating characteristic (ROC) curve analysis. The study showed that in logistic regression analyses, none of the glycation markers independently predicted albuminuria after adjustment for age, sex, and BMI (all $p > 0.05$). ROC analysis demonstrated poor discriminatory performance for HbA1c (AUC=0.597), fructosamine (AUC=0.602), and the combined model (AUC=0.599), and none of the AUC values reached statistical significance (all $p > 0.05$). In patients with newly diagnosed T2DM, HbA1c, fructosamine, HGI, and GG did not independently predict albuminuria. These findings suggest that protein glycation markers have limited value as biomarkers for identifying early kidney injury at the time of T2DM diagnosis.

Keywords: type 2 diabetes mellitus, hemoglobin glycation index, glycation gap, fructosamine, albuminuria, diabetic kidney disease

Introduction

Diabetes mellitus (DM) represents one of the greatest challenges to global health systems. According to the International Diabetes Federation (IDF), DM currently affects hundreds of millions of adults worldwide, and its prevalence continues to increase [1]. Because the disease often progresses silently, more than 50% of type 2 diabetes patients already have microvascular or macrovascular complications at the time of diagnosis [2]. Among these, diabetic kidney disease (DKD) is one of the most dangerous complications, not only impairing quality of life but also being a leading cause

of end-stage renal disease (ESRD) and cardiovascular death [3]. From a pathophysiological standpoint, chronic hyperglycemia promotes the glycation of proteins in the body, producing advanced glycation end products (AGEs). The accumulation of AGEs damages the glomerular filtration membrane structure through oxidative stress and inflammatory responses [4].

In current clinical practice, HbA1c (the glycation product of hemoglobin) is a common standard for monitoring glycemic control and predicting complications [5]. However, because HbA1c is formed within erythrocytes, its level is influenced by factors affecting red blood cell lifespan, and therefore may not accurately



reflect the actual plasma glucose concentration of each individual [6, 7].

To overcome these limitations, new glycation indices have been proposed to provide a more comprehensive picture of glucose toxicity. The glycation gap (GG), defined as the difference between the measured HbA1c and HbA1c predicted from fructosamine (an extra-cellular glycated protein), has attracted increasing attention [6]. Likewise, the hemoglobin glycation index (HGI), calculated as the difference between measured HbA1c and HbA1c predicted from fasting plasma glucose (FPG), has also emerged as a promising marker of interindividual variation in glycation [8]. Numerous studies worldwide have demonstrated that high HGI and GG are strongly correlated and serve as independent predictors of renal dysfunction in patients with long-standing type 2 diabetes [6, 9–11]. However, to our knowledge, no studies have yet evaluated the independent predictive value of HGI and GG for early kidney damage in diabetic patients, especially in newly diagnosed type 2 diabetes patients. During the early stage of T2DM, the pathogenesis of albuminuria (early kidney damage) is thought to be primarily due to hemodynamic disturbances in the glomeruli (increased perfusion, increased filtration) [12]. The accumulation of persistent AGEs (reflected in HGI and GG) requires long exposure to actually cause fibrosis of the filter membrane structure [13].

In our previous study involving patients with newly diagnosed T2DM, we found a marked increase in both HGI and GG, with 90.3% of patients having an HGI > 0.15%. In the same cohort, 34.95% of patients had elevated urinary albumin excretion, defined as a urinary albumin-to-creatinine ratio (uACR) ≥ 3 mg/mmol [14]. However, the question arises whether the early increase in HGI and GG is strong enough to be an independent risk factor predicting early-stage renal damage. Accordingly, the present study aimed to evaluate the independent predictive ability of glycation indices for hyperalbuminuria and reduced glomerular filtration rate (eGFR) in patients with newly diagnosed type 2 diabetes. We hypothesized that HGI and GG would independently predict early kidney injury damage.

Material and methods

Study design and participants

The cross-sectional descriptive study was conducted at Military Hospital 103 (Hanoi, Vietnam) from

September 2024 to June 2025. The study population included 103 adult patients (≥ 18 years old) with newly diagnosed T2DM. None of the participants had received glucose-lowering medications before enrollment. The T2DM was defined according to the 2024 American Diabetes Association (ADA) criteria, specifically: fasting plasma glucose (FPG) ≥ 7.0 mmol/L (126 mg/dL) and/or HbA1c $\geq 6.5\%$ (48 mmol/dL) [15]. Patients were excluded from the study if they had: (1) A history of or current hypertension; (2) Known renal or urinary tract diseases (such as nephrolithiasis, glomerulonephritis, nephrotic syndrome, history of kidney transplantation); (3) Hemoglobin disorders or anemia (Hemoglobin < 118 g/L); (4) Hypoproteinemia/hypoalbuminemia; and (5) pregnancy or malignancy.

Sample size calculation

The required sample size was calculated using the following formula:

$$n = \frac{(Z_{1-\alpha/2} + Z_{1-\beta})^2}{\frac{1}{4} \left[\log_e \left(\frac{1+r}{1-r} \right) \right]^2} + 3$$

where: n is the minimum required sample size; $Z_{1-\alpha/2} = 1.96$, corresponding to a 95% confidence level; $Z_{1-\beta} = 0.84$, corresponding to a statistical power of 80%; r is the expected correlation coefficient, assumed to be 0.30.

The calculated minimum sample size was 92 participants. Assuming a 10% dropout rate, the final minimum sample size was determined to be 103 participants for each study group (diabetes group and control group).

Clinical and biochemical measurements

After collection of demographic and anthropometric data, including height, weight, body mass index (BMI), and blood pressure, fasting venous blood samples and random urine samples were collected from all participants after an overnight fast of at least 8 hours.

HbA1c was measured using boronate affinity high-performance liquid chromatography (HPLC) on the PREMIER 9210 analyzer (Trinity Biotech, USA) which minimizes interference from hemoglobin variants. Serum fructosamine was determined by a colorimetric assay using the Alinity analyzer (Abbott, USA). Other biochemical parameters, including fasting

plasma glucose (FPG), serum creatinine, serum cystatin C, urinary microalbumin (MAU), and urinary creatinine, were measured using the AU5800 automated chemistry analyzer (Beckman Coulter, USA).

Calculations of novel glycation indices and renal function

Renal function: The degree of renal impairment is assessed through albuminuria and glomerular filtration rate (GFR). The urinary albumin – to – creatinine ratio (uACR) is calculated as the ratio of MAU (mg/L) to urinary creatinine (mmol/L). Hyperalbuminuria is defined as uACR ≥ 3 mg/mmol according to the ADA 2026 criteria. Estimated glomerular filtration rate (eGFR) is calculated using the CKD-EPI 2021 formula, which combines both plasma creatinine and cystatin C. Renal dysfunction is defined as eGFR < 60 mL/min/1.73 m² [16].

Glycation gap (GG): The GG is calculated based on the standard deviation of normal (SND) of fructosamine using the method of Macdonald D.R. (2008). The formula is the difference between the actual measured HbA1c and the HbA1c predicted from fructosamine concentration (FHbA1c).

The GG is a quantitative variable, calculated as the difference between the actual measured HbA1c and the HbA1c predicted from fructosamine (FHbA1c) using the formula [7]:

$$\begin{aligned} \text{SND}_{\text{FA}} &= (\text{FA} - \text{mean FA}) / \text{SD}_{\text{FA}} \\ \text{FHbA1c} &= (\text{SND}_{\text{FA}} \times \text{SD}_{\text{HbA1c}}) + \text{mean HbA1c} \\ \text{GG} &= \text{HbA1c} - \text{FHbA1c} \end{aligned}$$

The HGI is a quantitative variable, calculated as the difference between the actual measured HbA1c and the predicted HbA1c from fasting blood glucose (GHbA1c) using the formula [8]:

$$\begin{aligned} \text{GHbA1c} &= 0,024 \text{ FPG} + 3,1 \\ \text{HGI} &= \text{Measured HbA1c} - \text{GHbA1c} \end{aligned}$$

Statistical analysis

Statistical analyses were performed using SPSS version 20.0 (IBM Corp., Armonk, NY, USA).

Data normality was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests. Continuous variables were expressed as mean $\pm$ standard deviation (SD) or median (interquartile range [IQR]), as appropriate according to data distribution. Comparisons between

groups were performed using the independent-samples t-test, one-way analysis of variance (ANOVA), Mann–Whitney U test, or Kruskal–Wallis test, as appropriate. Correlations between variables were evaluated using Pearson's or Spearman's correlation coefficients.

To determine the independent predictive value of protein glycation markers for early kidney injury, univariate and multivariable logistic regression analyses were performed. Multivariable models were adjusted for age, sex, and BMI, which were selected a priori based on their established pathophysiological relevance. The results are presented as odds ratios (ORs) with 95% confidence intervals (95% CIs).

Receiver operating characteristic (ROC) curve analysis was further performed to evaluate the diagnostic performance of HbA1c, fructosamine, HGI, and GG for identifying albuminuria and reduced eGFR. The area under the ROC curve (AUC), sensitivity, and specificity were calculated. A two-sided p-value < 0.05 was considered statistically significant.

Results

The clinical characteristics of protein glycation markers and renal function parameters in the study population are summarized in Table 1.

The mean HbA1c level was significantly higher in patients with newly diagnosed T2DM than in the control group ($9.53 \pm 2.38\%$ vs. 5.71 ± 0.25 ; $p < 0.001$). Similarly, the mean fructosamine concentration was significantly higher in the patient group than in controls (468.31 ± 137.16 $\mu\text{mol/L}$ vs. 252.6 ± 22.9 $\mu\text{mol/L}$; $p < 0.001$).

The median GG was -0.02% in patients with newly diagnosed T2DM and tended to be lower than that in the control group; however the difference was not statistically significant ($p > 0.05$). In contrast, the median HGI was significantly higher in the patient group than in the control group ($p < 0.001$).

Urinary albumin concentration was significantly higher in patients than in controls, where as no significant differences were observed in serum creatinine, serum cystatin C, and urinary creatinine concentrations (all, $p > 0.05$).

The median uACR was 1.98 mg/mmol in the patient group, which was significantly higher than that in the control group ($p < 0.001$).

The median eGFR was 104.27 (mL/min/1.73 m²) in the patient group. Although, eGFR was higher than that in the control group the difference did not reach statistical significance ($p > 0.05$).

Table 1: Characteristics of protein glycation markers and renal function parameters in the study population.

Characteristic	Patient group (n=103)	Control group (n=103)	p
HbA1c (%) Mean±SD	9.53±2.38	5.71±0.25	<0.001 *
Fructosamine (μmol/L) Mean±SD	468.31±137.16	252.6±22.9	0.000 *
GG (%) Median (Q1-Q3)	-0.02 (-0.75–0.98)	0.05 (-0.2–0.3)	0.823 **
HGI (%) Median (Q1-Q3)	1.34 (0.65–2.34)	0.34 (0.19–0.54)	0.000 **
MAU (mg/L)	17.39 (7.65–41.42)	6.0 (3.7–9.1)	0.000 **
Urinary creatinine (mmol/L)	9.13 (4.85–13.99)	8.1 (4.2–12.5)	>0.05 **
uACR (mg/mmol) Median (Q1-Q3)	1.98 (0.87–5.37)	0.69 (0.48–1.28)	0.000 **
eGFR (mL/min/1.73 m²) Median (Q1-Q3)	104.27 (87.94–116.39)	1001.67 (93.8–108.63)	>0.05 **

Note: Data are presented as quartiles (quaternary 1 – quaternary 3). * – Perform independent T-test; ** – Mann-Whitney test.

Among the 103 patients included in the study, 34.95% showed evidence of early kidney damage as indicated by an increased uACR. Specifically, 33 patients (32.04%) had moderately increased albuminuria (uACR from 3–30 mg/mmol), whereas 3 patients (2.91%) had severely increased albuminuria (uACR≥30 mg/mmol).

Regarding renal filtration function, 5 patients (4.9%) had a reduced estimated glomerular filtration rate (eGFR<60 mL/min/1.73 m²) at the time of T2DM diagnosis. Overall 36.9% of patients had either a reduced eGFR (<60 mL/min/1.73 m²) and/or increased albuminuria (uACR>3 mg/mmol) (Figure 1).

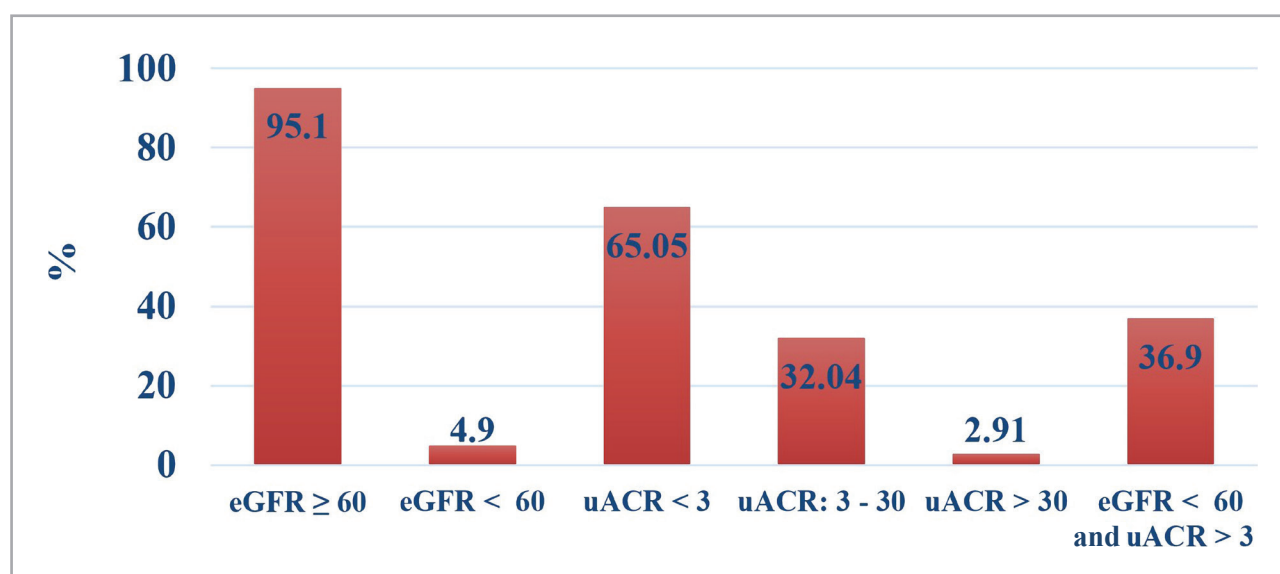


Figure 1: Illustrates the prevalence of abnormal uACR and eGFR in patients with newly diagnosed T2DM.

Table 2: Characteristics of stratified glycation indices according to the degree of renal impairment in the study subjects.

Index	uACR (mg/mmol)			P-value
	A1 (<3) (n=67)	A2 (3–30) (n=33)	A3 (≥30) (n=3)	
HbA1c (%) Mean±SD	9.28±2.34	9.67±2.22	13.8±0.9	0.004 *
Fructosamine (μmol/L) Mean±SD	453.6±133.9	479±133.8	677±83	0.018 *
GG (%) Median	-0.02	- 0.07	0.59	>0.05 **
HGI (%) Median	1.26	1.43	2.64	>0.05 **

Note: * – Perform Oneway Anova; ** – Kruskal-Wallis Test.

The distribution of protein glycation markers according to albuminuria categories is presented in Table 2.

When patients stratified according to albuminuria category, conventional glycation markers showed a progressive increase with increasing albuminuria severity. Mean HbA1c levels increased progressively from 9.28±2.34% in the A1 group to 9.67±2.22% in the A2 group and 13.8±0.9% in the A3 group (p=0.004). Similarly, mean serum fructosamin levels increased progressively across the A1, A2 and A3 groups (453.6±133.9, 479±133.8, and 677±83 μmol/L, respectively; p=0.018). In contrast, the new glycation indices did not differ significantly among albuminuria categories. The median GG were -0.02%, -0.07%, and 0.59% in the A1, A2, and

A3 groups, respectively (p>0.05). Likewise, the median HGI showed an increasing trend, from 1.26% in the A1 group to 2.64% in the A3, although the difference was not statistically significant (p>0.05).

To evaluate the independent predictive value of glycation indices for early kidney damage defined as albuminuria (uACR≥3 mg/mmol), univariate and multivariate logistic regression analyses were performed.

In the univariate regression analysis, although 90.3% of patients had an elevated HGI (>0.15%) (Figure 1), neither HGI nor GG was significantly associated with albuminuria. Specifically, the odds ratio (OR) for HGI was 0.996 (95% CI: 0.76–1.307; p=0.980), where as that for GG was 0.975 (95% CI: 0.749–1.268; p=0.848) (Table 3). Similarly, traditional glycation markers

Table 3: Logistic regression to assess the predictive value of glycation indices for hyperalbuminuria (uACR ≥3 mg/mmol).

	Odds (OR)	95%CI	P-value
Univariate logistic regression model			
Model 1: HbA1c (%)	1.138	0.959–1.349	0.138
Model 2: Fructosamine (μmol/L)	1.002	0.999–1.005	0.145
Model 3: GG (%)	0.975	0.749–1.268	0.848
Model 4: HGI (%)	0.996	0.76–1.307	0.98
Logistic multivariate regression model			
Model 1: HbA1c (%)	1.098	0.919–1.312	0.304
Model 2: Fructosamine (μmol/L)	1.001	0.998–1.005	0.395
Model 3: GG (%)	1.005	0.762–1.324	0.974
Model 4: HGI (%)	0.992	0.751–1.31	0.955

Note: Adjustments were made for age, gender, and BMI. Each logistic regression model was constructed separately for each glycation index to avoid multicollinearity, as HGI and GG were calculated from HbA1c and fructosamine.

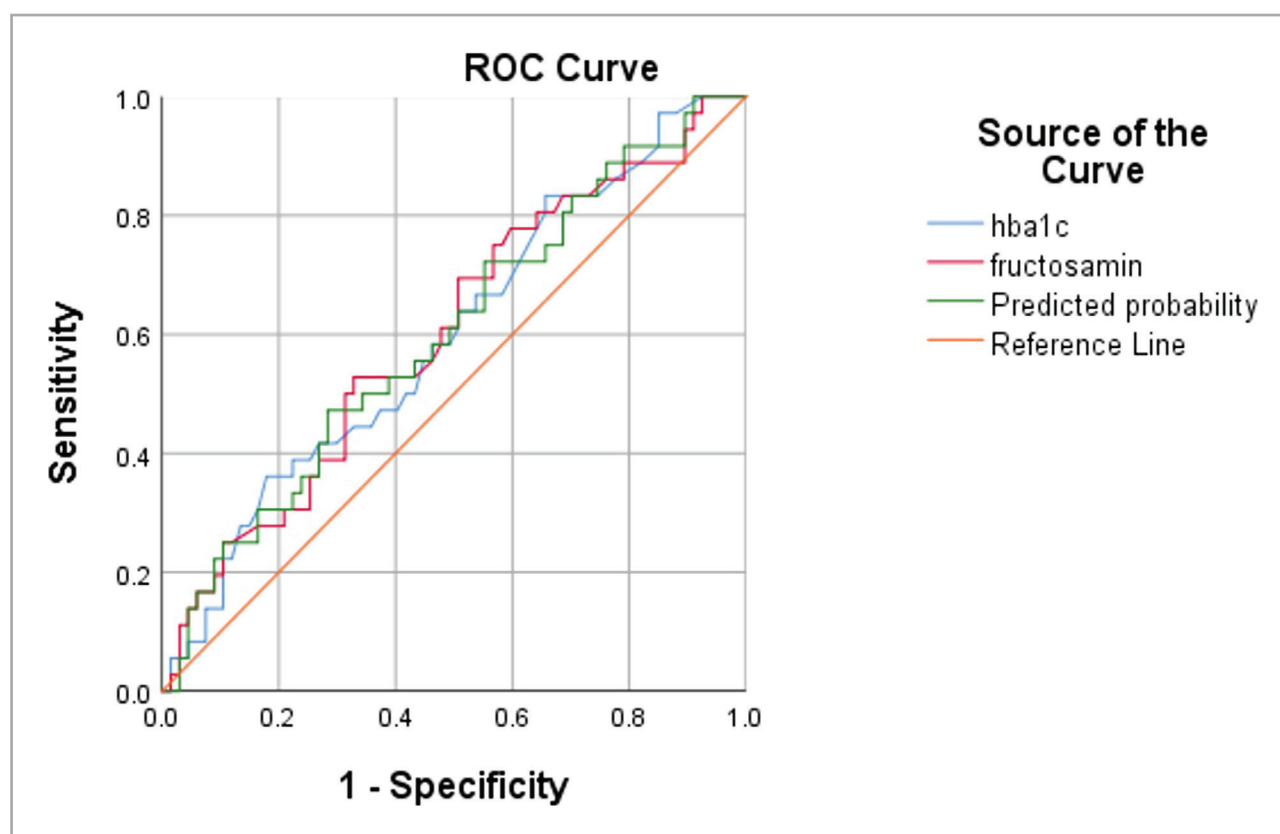


Figure 2: The ROC curve compares the diagnostic value of HbA1c, fructosamine, and the combined model (predicted probability) in differentiating patients with and without albuminuria.

including HbA1c and fructose were not significantly associated with albuminuria in the univariate model.

In the multivariate logistic regression analyses, with each glycation marker entered into a separate model and adjusted for age, gender, and BMI, none of the glycation markers independently predicted albuminuria. The adjusted OR was 0.992 (95% CI: 0.751–1.31; $p=0.955$) for HGI and 1.005 (95% CI: 0.762–1.324; $p=0.974$) for GG.

Receiver operating characteristic (ROC) analysis demonstrated poor discriminatory performance of HbA1c, serum fructosamine, and the combined model for identifying patients with albuminuria. The area under the curve (AUC) values were 0.597, 0.602, and

0.599, respectively. Although, serum fructosamine achieved the highest AUC (0.602), the difference compared with HbA1c and the combined model was minimal. The 95% confidence intervals for all three AUCs included 0.5, and none reached statistical significance ($p=0.106$, 0.090, and 0.100, respectively).

At the optimal cutoff values, HbA1c showed the highest specificity (82.1%) but relatively low sensitivity (36.1%). In contrast, serum fructosamine demonstrated higher sensitivity (52.8%) but lower specificity at 67.2%. The combined model yielded a sensitivity of 47.2% and a specificity of 71.6% without improving the differentiation compared to the individual marker alone (Figure 2 and Table 4).

Table 4: Area under the ROC curve (AUC) of HbA1c, fructosamine, and the combined model in differentiating patients with and without albuminuria.

Index	AUC	Cut-off	95%CI	Sensitivity (%)	Specificity (%)	P-value
HbA1c (%)	0.597	11.65	0.483–0.711	36.1	82.1	0.106
Fructosamine ($\mu\text{mol/L}$)	0.602	467.5	0.487–0.716	52.8	67.2	0.09
Combined model	0.599	0.37	0.484–0.713	47.2	71.6	0.1

Discussion

In our study, despite a significant increase in glycation indices (with 90.3% of patients having HGI>0.15%) and a relatively high rate of early kidney damage (34.95% had elevated uACR), multivariate regression analysis models showed that neither HbA1c, fructosamine, HGI, nor GG were independent predictors of hyperalbuminuria after adjusting for age, gender, and BMI. This suggests that the increase in glycation indices in newly diagnosed type 2 diabetes patients does not directly reflect the risk of early kidney damage. Although HbA1c and fructosamine tend to increase with the level of albuminuria, this association did not persist after controlling for confounding factors. These results indicate that in the early stages of the disease, glycation indices primarily reflect the degree of hyperglycemia rather than the process of glomerular damage. The ROC analysis further reinforces this observation. The area under the curve for HbA1c, fructosamine, and the combined model ranged only from 0.597 to 0.602, significantly lower than the 0.70 threshold typically considered acceptable for differentiation in clinical practice. Furthermore, the 95% confidence intervals of the AUCs all included values of 0.5 and were not statistically significant, demonstrating that the ability of these indices to differentiate patients with and without albuminuria was only slightly better than random. Combining HbA1c with fructosamine also did not improve diagnostic value, suggesting that indices reflecting glycation status alone are insufficient to predict renal damage in the very early stages of the disease.

Many previous studies worldwide have demonstrated the predictive power of HGI and GG for renal complications. Tatli E.'s study (2025) showed that HGI is an independent predictor of diabetic nephropathy with an odds ratio OR=2.579 [17]. Similarly, Le Quoc Tuan's study (2022) also noted a clear positive correlation between GG gap and uACR and a negative correlation with eGFR [18]. However, the core difference is that these studies were all conducted on patients who had had diabetes for a long time (average duration of the disease was over 9 years). Our study is one of the rare studies that focused on in-depth analysis of the group of patients at the time of their recent diabetes diagnosis.

The lack of independent association of HGI and GG with early-stage renal damage is explained primarily by differences in pathogenesis over time. In the first years of diabetes onset, the appearance of albuminuria is mainly a consequence of hemodynamic changes

in the glomeruli, characterized by increased perfusion and increased glomerular capillary pressure (hyperfiltration). Damage at this stage tends towards filtration membrane dysfunction and, in modern clinical practice, this albuminuria can sometimes resolve spontaneously or be reversed if the interfering factors are well controlled [12, 19]. HGI and GG gap are indicators representing the increased accumulation of advanced glycation end products (AGEs). Although AGEs production begins silently before diagnosis, the accumulation of AGEs in renal tissue requires a long period of time to cause irreversible structural damage, such as glomerular basement membrane thickening, mesangial matrix dilation, and glomerular sclerosis [20, 21]. Therefore, at the "window period" when the disease is first diagnosed, the structural accumulation of AGEs (represented by increased HGI and GG) is not yet severe enough to exceed the critical threshold, and thus does not act as an independent driver and predictor of renal damage compared to hemodynamic disturbances.

Although the study did not prove the independent predictive role of HGI and GG, the results are still of significant scientific importance. Most previous studies were conducted on diabetic patients who had been ill for many years, when glomerular damage had progressed and the accumulation of glycation end products (AGEs) had occurred over a long period. In contrast, the current study was conducted on patients at the time of diagnosis, reflecting a very early stage of the disease. Therefore, the results showing that HGI and GG do not have independent predictive value at this stage contribute to clarifying that the prognostic ability of glycation indices depends on the stage of disease progression, rather than having consistent value at all times. This is a significant finding for the selection of appropriate biomarkers in screening for diabetic kidney disease.

Our study still has some limitations. Firstly, the cross-sectional descriptive study design does not allow for the definitive confirmation of a causal relationship between glycation indices and renal function decline over time. Secondly, the study sample size (n=103 in each group) is relatively modest for a multivariate predictive analysis.

Conclusion

HbA1c, fructosamine, HGI, and GG were not independent predictors of hyperalbuminuria after adjusting for age, gender, and BMI. These results suggest that

glycation indices are not sufficiently valuable as biomarkers for screening for early renal damage in newly diagnosed type 2 diabetes patients.

Acknowledgments

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Conflict of interest

The authors declare no conflict of interest.

Ethics approval

The study protocol was approved by the Institutional Review Board of Military Hospital 103 (Approval No. 86/CNChT-HĐĐĐ) and the Ethics Committee of the National Institute of Hygiene and Epidemiology (Approval No. HĐĐĐ-32/2025).

Consent to participate

Informed consent was obtained from all individual participants included in the study. Prior to participation, all subjects were informed about the study objectives, potential risks and benefits, and their right to withdraw at any time without consequence

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