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Triglyceride-glucose Index and Triglyceride/HDL-cholesterol Ratio as Predictors of Poor Glycemic Control in Elderly Patients with Type 2 Diabetes

Trigliserid-glikoz İndeksi ve Trigliserid/HDL-kolesterol Oranının Tip 2 Diyabetli Yaşlı Hastalarda Kötü Glisemik Kontrolün Öngörücüsü Olarak Kullanılması

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Abstract

Objective: Glycated haemoglobin (HbA1c) is the standard measure of glycemic control, but its reliability is limited in older adults. This study compared the triglyceride-glucose (TyG) index and the triglyceride/HDL-cholesterol (TG/HDL-C) ratio for identifying poor glycemic control in elderly patients with type 2 diabetes.

Method: This cross-sectional study included 137 consecutive patients aged ≥65 years with type 2 diabetes from an internal medicine outpatient clinic. Patients on lipid-lowering therapy or with conditions interfering with HbA1c interpretation were excluded. The TyG index was calculated as Ln [triglycerides (mg/dL)×fasting plasma glucose (mg/dL)/2]. Patients were classified as having good (HbA1c<7%) or poor (HbA1c≥7%) control. Discrimination was assessed by receiver operating characteristic analysis, areas under the curve (AUC) compared using the DeLong test, and independent associations by multivariable logistic regression.

Results: Fifty-nine patients (43.1%) had good and 78 (56.9%) poor control. The TyG index was higher in the poor control group (9.47±0.62 vs. 9.05±0.47; p<0.001), whereas the TG/HDL-C ratio did not differ between groups (3.98±3.46 vs. 3.24±2.30; p=0.125). The TyG index identified poor control with an AUC of 0.711 [95% confidenve interval (CI) 0.624-0.798; p<0.001] at a cut-off of 9.35 (sensitivity 57.7%, specificity 79.7%), whereas the TG/HDL-C ratio performed no better than chance (AUC 0.577, 95% CI 0.480-0.674; p=0.120); the difference was significant (DeLong p<0.001). After adjustment

Öz

Amaç: Glikozillenmiş hemoglobin (HbA1c) glisemik kontrolün standart ölçütüdür; ancak yaşlı bireylerde güvenilirliği sınırlıdır. Bu çalışmada, tip 2 diyabetli yaşlı hastalarda kötü glisemik kontrolün belirlenmesinde trigliserid-glukoz (TyG) indeksi ile trigliserid/HDL-kolesterol (TG/HDL-C) oranı karşılaştırıldı.

Yöntem: Bu kesitsel çalışmaya bir iç hastalıkları polikliniğine ardışık olarak başvuran, tip 2 diyabetli 65 yaş ve üzeri 137 hasta alındı. Lipid düşürücü tedavi alan ya da HbA1c yorumunu etkileyen durumları bulunan hastalar dışlandı. TyG indeksi Ln [trigliserid (mg/dL)×açlık plazma glukozu (mg/dL)/2] formülüyle hesaplandı. Hastalar iyi (HbA1c<%7) ve kötü (HbA1c≥%7) kontrol olarak sınıflandırıldı. Ayırt edicilik alıcı çalışma karakteristiği analiziyle değerlendirildi, eğri altı alanlar (EAA) DeLong testiyle karşılaştırıldı, bağımsız ilişkiler çok değişkenli lojistik regresyonla incelendi.

Bulgular: Hastaların 59'u (%43,1) iyi, 78'i (%56,9) kötü kontrole sahipti. TyG indeksi kötü kontrol grubunda daha yüksekti (9,47±0,62 ve 9,05±0,47; p<0,001); TG/HDL-C oranı ise gruplar arasında farklılık göstermedi (3,98±3,46 ve 3,24±2,30; p=0,125). TyG indeksi kötü kontrolü 0,711 EAA ile belirledi [%95 güven aralığı (GA) 0,624-0,798; p<0,001]; 9,35 kesim değerinde duyarlılık %57,7, özgüllük %79,7 idi. TG/HDL-C oranı ise şanstan daha iyi bir ayırt edicilik göstermedi (EAA 0,577; %95 GA 0,480-0,674; p=0,120); iki alan arasındaki fark anlamlıydı (DeLong p<0,001). Yaş, cinsiyet, diyabet süresi, beta-bloker kullanımı, DPP-4 inhibitörü kullanımı, diğer antidiyabetik

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Abstract

for age, sex, diabetes duration, beta-blocker use, DPP-4 inhibitor use, other antidiabetic treatment and body mass index, the TyG index remained independently associated [adjusted odds ratio (OR) 3.65; 95% CI 1.66-8.05; p=0.001], whereas the TG/HDL-C ratio did not (OR 1.08; 95% CI 0.94-1.23; p=0.276). Findings were reproduced in all sensitivity analyses.

Conclusion: In elderly patients with type 2 diabetes, the TyG index was independently associated with poor glycemic control and outperformed the TG/HDL-C ratio, which showed no discriminative value. Of these two indices, only the TyG index appears informative in geriatric diabetes care.

Keywords: Glycemic control, HbA1c, older adults, triglyceride/HDL-cholesterol ratio, triglyceride-glucose index, type 2 diabetes mellitus

Öz

tedavi ve vücut kitle indeksi için düzeltme yapıldıktan sonra TyG indeksi bağımsız olarak ilişkili kalırken [düzeltilmiş olasılık oranı (OO) 3,65; %95 GA 1,66-8,05; p=0,001], TG/HDL-C oranı için böyle bir ilişki saptanmadı (OO 1,08; %95 GA 0,94-1,23; p=0,276). Bulgular tüm duyarlılık analizlerinde doğrulandı.

Sonuç: Tip 2 diyabetli yaşlı hastalarda TyG indeksi kötü glisemik kontrolle bağımsız olarak ilişkili bulunmuş ve hiçbir ayırt edicilik göstermeyen TG/HDL-C oranından üstün çıkmıştır. Bu iki indeksten yalnızca TyG indeksi geriatrik diyabet bakımında bilgi verici görünmektedir.

Anahtar kelimeler: Glisemik kontrol, HbA1c, trigliserit/HDL-kolesterol oranı, trigliserit-glukoz indeksi, tip 2 diabetes mellitus, yaşlı

Introduction

Diabetes mellitus (DM) is a widespread chronic disorder and represents a significant challenge for public health systems, particularly in ageing populations. Its global incidence continues to rise, especially among older adults, as a consequence of demographic change and increased life expectancy (1,2). Elderly patients with DM frequently present with complex metabolic disturbances, multiple comorbidities and an increased risk of microvascular and macrovascular complications. Achieving adequate glycemic control is therefore essential, since poor control is strongly associated with nephropathy, cardiovascular disease, cognitive decline and mortality (3,4).

Glycated haemoglobin (HbA1c) is the measure most commonly used to evaluate long-term glycemic regulation. In older adults, however, its reliability may be limited by anaemia, chronic kidney disease, altered erythrocyte turnover and polypharmacy (5). This has generated interest in alternative, simple biomarkers that reflect the metabolic disturbances central to the pathophysiology of type 2 DM.

In this context, the triglyceride-glucose (TyG) index and the triglyceride-to-HDL cholesterol (TG/HDL-C) ratio have emerged as surrogate markers of insulin resistance in population studies. The TyG index, derived from fasting triglyceride and glucose concentrations, correlates with insulin resistance and predicts incident DM, cardiovascular disease and metabolic disorders (6-8). The TG/HDL-C ratio reflects atherogenic dyslipidaemia and has been associated with impaired glucose metabolism and cardiometabolic risk (9,10). Both are widely used because of their simplicity, low cost and routine availability.

Several studies have demonstrated that an increased TyG index is linked to a greater likelihood of developing type 2 DM and cardiovascular events (7,11), and to metabolic dysfunction and worse outcomes in older adults (12). A higher TG/HDL-C ratio has similarly been reported to be related to inadequate glycemic control, insulin resistance and cardiovascular risk (9,13), and Liu et al. (14) reported that the TG/HDL-C ratio is an effective marker for new-onset DM in individuals over 75 years of age.

Despite this growing body of evidence, studies focusing specifically on glycemic control in patients with established diabetes remain limited. Most data derive from mixed-age populations, and geriatric-specific evidence is scarce (15,16). More importantly, although both indices are frequently described in parallel, direct comparisons of their discriminative performance in elderly diabetic patients are lacking, and previous reports that claimed superiority of one index over the other did so without formal statistical comparison of the corresponding receiver operating characteristic (ROC) curves.

This distinction is clinically relevant, because metabolic regulation in older adults is influenced by ageing-related physiological changes, comorbidity and treatment factors, and because the two indices differ in their susceptibility to pharmacological and lipid-related confounding. The present study was therefore designed to assess the relationship of the TG/HDL-C ratio and the TyG index with glycemic control in elderly patients with type 2 DM, and to compare their ability to identify poor glycemic control.

Materials and Methods

Study Design and Participants

This cross-sectional study enrolled 137 patients aged 65 years and older with type 2 DM who were consecutively recruited from the internal medicine outpatient clinic of

our hospital. Informed written consent was obtained from all participants before enrolment.

Patients with type 1 DM, malignancy, dementia, chronic liver disease including cirrhosis, congestive heart failure, stage 4-5 chronic kidney disease, current use of any lipid-lowering therapy, or refusal to participate were excluded. Because HbA1c served as the reference standard for glycemic control, patients with conditions known to alter erythrocyte turnover and thereby confound HbA1c interpretation were also considered ineligible; accordingly, none of the included participants had a known haemoglobinopathy or thalassaemia major, haemolytic anaemia, previous splenectomy, human immunodeficiency virus infection, active bleeding, or a history of blood transfusion within the three months preceding blood sampling.

The study protocol was approved by the Institutional Ethics Committee of University of Health Sciences Turkey, Prof. Dr. Cemil Taşcıoğlu City Hospital (approval number: E-48670771-514.99-299780546, date: 30.12.2025) and the study was conducted in accordance with the Declaration of Helsinki.

The diagnosis of type 2 DM was established according to American Diabetes Association (ADA) criteria, and lipid reference ranges were defined in line with the National Cholesterol Education Program Adult Treatment Panel III guidelines. Data on demographic characteristics (age, sex, education level, marital status), smoking status, comorbid conditions, diabetic complications and medication use were systematically recorded. Medication data were retrieved from prescription records and confirmed by patient interview at enrolment. Antidiabetic treatment was classified by pharmacological group [metformin, sulfonylureas, dipeptidyl peptidase-4 (DPP-4) inhibitors, sodium-glucose cotransporter-2 (SGLT-2) inhibitors, thiazolidinediones, alpha-glucosidase inhibitors and insulin], and cardiovascular medications were recorded by class. Only patients whose antidiabetic and cardiovascular treatment had remained unchanged for at least three months prior to blood sampling were included.

Laboratory Measurements

Blood samples were obtained after a 12-hour fasting period, with participants instructed to avoid high-fat meals and strenuous physical activity for three days before sampling. Fasting plasma glucose (FPG) was measured by the glucose oxidase method. HbA1c was determined by high-performance liquid chromatography (HPLC) using an NGSP-certified, IFCC-standardized system; this method permits chromatographic identification of the most frequent haemoglobin variants and is less susceptible to analytical interference than immunoassay-based techniques.

Serum total cholesterol (TC) and HDL-C were analysed by enzymatic methods and triglycerides (TG) by enzymatic hydrolysis. LDL-C was estimated using the Friedewald formula; in accordance with the validity limits of this formula, LDL-C was not calculated in participants whose triglyceride concentration exceeded 400 mg/dL (n=6). Aspartate aminotransferase, alanine aminotransferase, albumin, uric acid, vitamin B12, urea and creatinine were measured by standard automated methods, and estimated glomerular filtration rate (eGFR) was calculated using the 2021 race-free CKD-EPI creatinine equation (17). Complete blood count parameters were obtained on an automated haematology analyser, and anaemia was defined according to World Health Organization criteria (haemoglobin <12 g/dL in women and <13 g/dL in men). No participant had a known haemoglobin variant expected to interfere with HbA1c quantification. Serum bilirubin was not measured as part of the study protocol; patients with chronic liver disease and those with clinically evident jaundice were excluded on the basis of medical records and clinical examination, but hyperbilirubinaemia was not excluded biochemically. HPLC-based HbA1c assays are, however, considerably less susceptible to bilirubin interference than immunoassay-based or enzymatic methods.

The TG/HDL-C ratio was calculated by dividing the triglyceride concentration by the HDL-C concentration (both in mg/dL). The TyG index was calculated as Ln{[TG (mg/dL)×FPG (mg/dL)]/2}.

Group Classification

Participants were divided into two groups according to glycemic control status: good control (HbA1c < 7%) and poor control (HbA1c ≥7%). Although current guidelines recommend individualized glycemic targets in older adults, the ADA Standards of Care recommend an HbA1c goal of <7.0-7.5% for healthy older adults with few coexisting chronic illnesses and intact cognitive and functional status, and less stringent goals (<8.0-8.5%) for those with complex or poor health status (3). Since patients with dementia, malignancy, congestive heart failure, chronic liver disease and advanced chronic kidney disease were excluded and all participants were ambulatory outpatients, our cohort corresponds to the healthy older adult category, for which the 7% threshold is guideline-concordant. This cut-off also

permits comparability with previous studies evaluating the same indices (14,18). To ensure that the findings were not dependent on this choice, sensitivity analyses were performed using thresholds of HbA1c ≥7.5% and ≥8.0%.

Statistical Analysis

Statistical analyses were performed using SPSS version 27 (IBM Corp., Armonk, NY, USA). Continuous variables are presented as mean ± standard deviation and as median (minimum-maximum), and categorical variables as frequencies and percentages. The normality of distributions was assessed using the Shapiro-Wilk test together with graphical evaluation. Group comparisons of continuous variables were performed with Student's t-test or the Mann-Whitney U test as appropriate, and categorical variables were compared using the Pearson chi-square test or Fisher's exact test. Correlations with HbA1c were examined using the Spearman rank correlation coefficient.

The ability of the TyG index and the TG/HDL-C ratio to identify poor glycemic control was assessed by ROC curve analysis. Optimal cut-off values were determined using the Youden index, and sensitivity, specificity, positive and negative predictive values and accuracy were calculated. Areas under the curve (AUC) were compared using the non-parametric DeLong test for correlated ROC curves (19). Multivariable binary logistic regression analysis was performed with poor glycemic control as the dependent variable; models were adjusted for age, sex, diabetes duration longer than ten years, beta-blocker use, DPP-4, inhibitor use, number of oral antidiabetic drugs, any insulin therapy and body mass index; beta-blocker use and DPP-4 inhibitor use were included as covariates because their distribution differed significantly between glycemic control groups. Models are reported as adjusted odds ratios with 95% confidence intervals, together with Nagelkerke R^2 and the likelihood ratio test. Sensitivity analyses were conducted using alternative HbA1c thresholds and after exclusion of anaemic patients, patients with TG above 400 mg/dL, insulin-treated patients, and patients with eGFR below 60 mL/min/1.73 m^2. A two-sided p-value below 0.05 was considered statistically significant.

Results

A total of 137 patients were included, of whom 50 (36.5%) were male and 87 (63.5%) female. The mean age was 68.34±4.06 years and the mean body mass index 29.53±4.34 kg/m^2. HbA1c values ranged from 5.5% to 13.8%, with a mean of 7.49±1.52%. According to glycemic control status, 59 patients (43.1%) were classified as having good control (HbA1c <7%) and 78 (56.9%) as having poor control (HbA1c ≥7%).

Demographic and clinical characteristics are presented in Table 1. The two groups were comparable with respect to sex, age, education level, marital status, smoking status, body mass index, duration of diabetes, hypertension, coronary artery disease, cerebrovascular disease, depression and the prevalence of microvascular complications (all p>0.05). Systolic and diastolic blood pressure were significantly higher in the poor glycemic control group (138.08±16.58 vs. 128.98±14.79 mmHg, p<0.001, and 81.21±10.12 vs. 77.80±9.79 mmHg, p=0.024, respectively).

Treatment characteristics are shown in Table 2. Metformin was the most frequently prescribed antidiabetic agent (73.7% of the cohort), followed by DPP-4 inhibitors (55.5%), SGLT-2 inhibitors (22.6%) and sulfonylureas (20.4%); 28 patients (20.4%) were receiving insulin, and the mean number of oral antidiabetic drugs was 1.85±1.09. No participant was receiving lipid-lowering therapy, as this constituted an exclusion criterion. Most treatment categories were comparably distributed between groups, including metformin, SGLT-2 inhibitor, sulfonylurea, pioglitazone and acarbose use, the number of oral antidiabetic drugs (p=0.069) and any insulin therapy (p=0.128). DPP-4 inhibitor use was significantly more frequent among patients with poor glycemic control (65.4% vs. 42.4%, p=0.012), as was beta-blocker use (25.6% vs. 8.5%, p=0.019). Angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, diuretics, acetylsalicylic acid and proton pump inhibitors did not differ significantly between groups (all p>0.05).

Biochemical and haematological parameters are presented in Table 3. As expected, FPG (171.26±60.48 vs. 120.05±23.79 mg/dL) and HbA1c (8.39±1.43% vs. 6.30±0.40%) were significantly higher in the poor control group (both p<0.001). In contrast, none of the individual lipid parameters differed significantly between groups: TC (p=0.964), LDL-C (p=0.860), HDL-C (p=0.145) and TG (p=0.292). Renal function indicators, including urea, creatinine and eGFR, were comparable between groups (all p>0.05), as were aspartate aminotransferase, alanine aminotransferase, uric acid and vitamin B12. Haemoglobin (13.63±1.34 vs. 13.23±1.06 g/dL, p=0.135) and the prevalence of anaemia defined by World Health Organization criteria (15.4% vs. 11.9%, p=0.733) did not differ between groups, indicating that altered erythrocyte indices are unlikely to have confounded HbA1c interpretation in this cohort.

Table 1. Demographic and clinical characteristics according to glycemic control

	Good glycemic control (HbA1c <7%) (n=59)	Poor glycemic control (HbA1c ≥7%) (n=78)	Test	p
Sex			[a]	0.277
Male	18 (30.5)	32 (41.0)		
Female	41 (69.5)	46 (59.0)		
Age (years)	68.08±3.68	68.53±4.34	[b]	0.485
Median (min-max)	67.00 (65.00-83.00)	67.00 (65.00-86.00)		
Education level			[a]	0.745
Illiterate/no formal education	12 (20.3)	12 (15.4)		
Primary school	41 (69.5)	57 (73.1)		
High school or above	6 (10.2)	9 (11.5)		
Marital status			[a]	0.783
Married	43 (72.9)	54 (69.2)		
Single or widowed	16 (27.1)	24 (30.8)		
Smoking status			[a]	0.269
Never smoker	42 (71.2)	46 (59.0)		
Current smoker	7 (11.9)	10 (12.8)		
Ex-smoker	10 (16.9)	22 (28.2)		
Body mass index (kg/m^2)	29.77±4.84	29.35±3.94	[b]	0.950
Median (min-max)	29.22 (22.68-43.71)	29.07 (21.87-40.27)		
Duration of diabetes			[a]	0.119
<5 years	12 (20.3)	7 (9.0)		
5-10 years	28 (47.5)	37 (47.4)		
>10 years	19 (32.2)	34 (43.6)		
Hypertension			[a]	0.309
No	12 (20.3)	23 (29.5)		
Yes	47 (79.7)	55 (70.5)		
Antihypertensive use			[a]	0.136
No	12 (20.3)	26 (33.3)		
Yes	47 (79.7)	52 (66.7)		
Systolic blood pressure (mmHg)	128.98±14.79	138.08±16.58	[b]	**<0.001**
Median (min-max)	130.00 (100.00-200.00)	135.00 (110.00-180.00)		
Diastolic blood pressure (mmHg)	77.80±9.79	81.21±10.12	[b]	**0.024**
Median (min-max)	80.00 (60.00-110.00)	80.00 (60.00-100.00)		
Coronary artery disease			[a]	0.279
No	55 (93.2)	67 (85.9)		
Yes	4 (6.8)	11 (14.1)		
Cerebrovascular disease			[d]	1.000
No	58 (98.3)	77 (98.7)		
Yes	1 (1.7)	1 (1.3)		
Depression			[a]	0.311
No	45 (76.3)	66 (84.6)		
Yes	14 (23.7)	12 (15.4)		
Any microvascular complication			[a]	0.195
No	42 (71.2)	46 (59.0)		
Yes	17 (28.8)	32 (41.0)		

Table 1. Continued

	Good glycemic control (HbA1c <7%) (n=59)	Poor glycemic control (HbA1c ≥7%) (n=78)	Test	p
Nephropathy			[a]	0.209
No	48 (81.4)	55 (70.5)		
Yes	11 (18.6)	23 (29.5)		
Retinopathy			[a]	0.301
No	45 (76.3)	52 (66.7)		
Yes	14 (23.7)	26 (33.3)		
Neuropathy			[a]	0.134
No	49 (83.1)	55 (70.5)		
Yes	10 (16.9)	23 (29.5)		

Data are presented as mean ± standard deviation and median (minimum-maximum) for continuous variables and as n (%) for categorical variables. [a]: Pearson chi-square test, [b]: Mann-Whitney U test

Serum albumin was slightly lower (4.18±0.37 vs. 4.31±0.30 g/dL, p=0.036) and the white blood cell count slightly higher (8.01±1.79 vs. 7.27±1.71×10^3/μL, p=0.013) in the poor control group.

When the two metabolic indices were compared (Table 4), the TyG index was significantly higher in patients with poor glycemic control (9.47±0.62 vs. 9.05±0.47; p<0.001), whereas the TG/HDL-C ratio did not differ significantly between groups (3.98±3.46 vs. 3.24±2.30; p=0.125

Comparative ROC analysis is summarized in Table 5 and illustrated in Figure 1. The TyG index identified poor glycemic control with an AUC of 0.711 (95% CI 0.624-0.798; p<0.001); the optimal cut-off determined by the Youden index was 9.35, yielding a sensitivity of 57.7%, a specificity of 79.7%, a positive predictive value of 78.9%, a negative predictive value of 58.8% and an overall accuracy of 67.2%. The TG/HDL-C ratio yielded an AUC of 0.577 (95% CI 0.480-0.674; p=0.120), which did not differ significantly from chance. The difference between the two areas was 0.134 (z=4.19) and was highly significant on the DeLong test (p<0.001). A TyG index of 9.35 or above was associated with a 5.34-fold increased likelihood of poor glycemic control (OR 5.34, 95% CI 2.46-11.62; p<0.001), whereas the corresponding unadjusted odds ratio for a TG/HDL-C ratio of 3.69 or above was substantially lower (OR 2.27, 95% CI 1.08-4.74; p=0.030).

Because FPG is a component of the TyG index and is itself closely related to HbA1c, the discriminative performance of FPG alone was also examined. FPG yielded an AUC of 0.824 (95% CI 0.753-0.895; p<0.001), which was significantly higher than that of the TyG index (DeLong p=0.020).

Multivariable logistic regression analyses are presented in Table 6. After adjustment for age, sex, diabetes duration longer than ten years, beta-blocker use, DPP-4 inhibitor use, number of oral antidiabetic drugs, any insulin therapy and body mass index, the TyG index remained independently associated with poor glycemic control both as a continuous variable (adjusted OR 3.65 per unit, 95% CI 1.66-8.05; p=0.001; Nagelkerke R^2=0.275) and when dichotomised at the ROC-derived cut-off (adjusted OR 4.94, 95% CI 2.15-11.33; p<0.001; Nagelkerke R^2=0.298). The TG/HDL-C ratio was not independently associated with poor glycemic control (adjusted OR 1.08 per unit, 95% CI 0.94-1.23; p=0.276). Beta-blocker use was an independent predictor in the model containing the TG/HDL-C ratio (adjusted OR 3.66, 95% CI 1.23-10.88; p=0.020) but not in the models containing the TyG index (adjusted OR 2.98, 95% CI 0.96-9.20; p=0.058). DPP-4 inhibitor use showed the same pattern: It was independently associated with poor glycemic control in the model containing the TG/HDL-C ratio (adjusted OR 2.54, 95% CI 1.04-6.20; p=0.042) but not in those containing the TyG index (adjusted OR 2.09, 95% CI 0.82-5.37; p=0.124 for the continuous index and adjusted OR 2.28, 95% CI 0.88-5.89; p=0.090 for the dichotomised index).

The results were consistent across all sensitivity analyses (Table 7). The TyG index remained significantly associated with poor glycemic control when the threshold defining poor control was raised to HbA1c ≥7.5% (AUC 0.754, 95% CI 0.670-0.839) and ≥8.0% (AUC 0.723, 95% CI 0.625-0.821), after exclusion of anaemic patients (AUC 0.680, 95% CI 0.583-0.777), of patients with TG above 400 mg/dL (AUC 0.703, 95% CI 0.614-0.792), of insulin-treated patients (AUC 0.738, 95% CI 0.643-0.833), and when the analysis was restricted to patients with eGFR ≥60 mL/min/1.73 m^2 (AUC

Table 2. Antidiabetic and cardiovascular medication use according to glycemic control

	Good glycemic control (HbA1c <7%) (n=59)	Poor glycemic control (HbA1c ≥7%) (n=78)	Test	p
Diet only			[d]	1.000
No	58 (98.3)	76 (97.4)		
Yes	1 (1.7)	2 (2.6)		
Oral antidiabetic drugs only			[a]	0.112
No	9 (15.3)	22 (28.2)		
Yes	50 (84.7)	56 (71.8)		
Number of oral antidiabetic drugs	1.71±1.13	1.95±1.06	[b]	0.069
Median (min-max)	1.00 (0.00-5.00)	2.00 (0.00-4.00)		
Insulin monotherapy			[d]	0.390
No	58 (98.3)	74 (94.9)		
Yes	1 (1.7)	4 (5.1)		
Oral antidiabetic drugs + insulin			[a]	0.267
No	52 (88.1)	62 (79.5)		
Yes	7 (11.9)	16 (20.5)		
Any insulin therapy			[a]	0.128
No	51 (86.4)	58 (74.4)		
Yes	8 (13.6)	20 (25.6)		
Metformin			[a]	0.117
No	11 (18.6)	25 (32.1)		
Yes	48 (81.4)	53 (67.9)		
DPP-4 inhibitor			[a]	**0.012**
No	34 (57.6)	27 (34.6)		
Yes	25 (42.4)	51 (65.4)		
SGLT-2 inhibitor			[a]	1.000
No	46 (78.0)	60 (76.9)		
Yes	13 (22.0)	18 (23.1)		
Sulfonylurea			[a]	0.274
No	50 (84.7)	59 (75.6)		
Yes	9 (15.3)	19 (24.4)		
Pioglitazone			[d]	0.698
No	57 (96.6)	73 (93.6)		
Yes	2 (3.4)	5 (6.4)		
Acarbose			[d]	1.000
No	56 (94.9)	75 (96.2)		
Yes	3 (5.1)	3 (3.8)		
ACE inhibitor			[a]	0.439
No	46 (78.0)	66 (84.6)		
Yes	13 (22.0)	12 (15.4)		
Angiotensin receptor blocker			[a]	0.178
No	35 (59.3)	56 (71.8)		
Yes	24 (40.7)	22 (28.2)		
Beta-blocker			[a]	**0.019**
No	54 (91.5)	58 (74.4)		
Yes	5 (8.5)	20 (25.6)		
Diuretic (any)			[a]	0.696
No	42 (71.2)	59 (75.6)		
Yes	17 (28.8)	19 (24.4)		
Thiazide diuretic			[a]	0.635
No	44 (74.6)	62 (79.5)		
Yes	15 (25.4)	16 (20.5)		
Loop diuretic (furosemide)			[d]	1.000
No	57 (96.6)	76 (97.4)		
Yes	2 (3.4)	2 (2.6)		
Spironolactone			[d]	0.431
No	58 (98.3)	78 (100.0)		
Yes	1 (1.7)	0 (0.0)		
Acetylsalicylic acid			[a]	0.956
No	48 (81.4)	62 (79.5)		
Yes	11 (18.6)	16 (20.5)		
Proton pump inhibitor			[a]	0.636
No	30 (50.8)	44 (56.4)		
Yes	29 (49.2)	34 (43.6)		

Data are presented as mean ± standard deviation and median (minimum-maximum) for continuous variables and as n (%) for categorical variables.
[a]: Pearson chi-square test, [b]: Mann-Whitney U test, [d]: Fisher's exact test

0.736, 95% CI 0.649-0.823); in every analysis p was below 0.001 and the TyG index remained significantly superior to the TG/HDL-C ratio on the DeLong test (all p<0.001).

Correlations of the two indices and their components with HbA1c are presented in Table 8. The TyG index correlated moderately with HbA1c (Spearman ρ=0.491, p<0.001), whereas the correlation of the TG/HDL-C ratio did not reach statistical significance (ρ=0.164, p=0.056).

Fasting plasma glucose showed the strongest correlation with HbA1c (ρ=0.715, p<0.001), while neither triglycerides (ρ=0.132, p=0.124) nor HDL-cholesterol (ρ=-0.131, p=0.128) was significantly correlated with HbA1c.

Table 3. Biochemical and haematological parameters according to glycemic control

	Good glycemic control (HbA1c <7%) (n=59)	Poor glycemic control (HbA1c ≥7%) (n=78)	Test	p
Fasting plasma glucose (mg/dL)	120.05±23.79	171.26±60.48	[b]	**<0.001**
Median (min-max)	115.00 (90.00-188.00)	155.00 (80.00-389.00)		
HbA1c (%)	6.30±0.40	8.39±1.43	[b]	**<0.001**
Median (min-max)	6.20 (5.50-6.90)	8.00 (7.00-13.80)		
Total cholesterol (mg/dL)	211.19±37.42	210.67±41.65	[b]	0.964
Median (min-max)	200.00 (153.00-300.00)	207.00 (136.00-342.00)		
LDL-cholesterol (mg/dL)[a]	128.14±34.14	126.03±32.53	[b]	0.860
Median (min-max)	127.00 (72.00-205.00)	125.00 (62.00-198.00)		
HDL-cholesterol (mg/dL)	54.10±11.45	51.15±11.91	[c]	0.145
Median (min-max)	53.00 (32.00-80.00)	50.00 (22.00-83.00)		
Triglycerides (mg/dL)	160.85±81.56	184.08±122.44	[b]	0.292
Median (min-max)	144.00 (63.00-523.00)	158.00 (63.00-760.00)		
Urea (mg/dL)	33.86±10.53	33.19±10.60	[b]	0.813
Median (min-max)	30.00 (17.00-71.00)	32.00 (9.00-62.00)		
Creatinine (mg/dL)	0.83±0.20	0.80±0.17	[c]	0.292
Median (min-max)	0.81 (0.45-1.50)	0.74 (0.46-1.25)		
eGFR (mL/min/1.73 m^2)[b]	83.13±16.38	88.43±12.99	[b]	0.131
Median (min-max)	84.24 (37.26-106.70)	93.03 (45.83-104.53)		
AST (U/L)	23.31±4.88	21.86±5.15	[b]	0.081
Median (min-max)	24.00 (13.00-34.00)	21.00 (13.00-34.00)		
ALT (U/L)	27.15±5.71	25.71±5.66	[c]	0.143
Median (min-max)	28.00 (14.00-37.00)	26.00 (14.00-39.00)		
Albumin (g/dL)	4.31±0.30	4.18±0.37	[b]	**0.036**
Median (min-max)	4.30 (3.50-4.80)	4.20 (3.00-5.00)		
Uric acid (mg/dL)	5.54±1.45	5.00±1.56	[c]	**0.081**
Median (min-max)	5.25 (2.50-9.60)	4.95 (2.10-9.92)		
Vitamin B12 (pg/mL)	323.91±241.81	428.12±383.14	[b]	0.221
Median (min-max)	265.00 (50.00-1484.00)	291.00 (78.00-2000.00)		
Haemoglobin (g/dL)	13.23±1.06	13.63±1.34	[b]	0.135
Median (min-max)	12.90 (11.40-16.80)	13.35 (11.70-17.50)		
White blood cell count (×10^3/μL)	7.27±1.71	8.01±1.79	[b]	**0.013**
Median (min-max)	7.03 (3.39-11.40)	8.03 (4.23-14.90)		
Platelet count (×10^3/μL)	273.88±76.49	279.47±74.36	[c]	0.669
Median (min-max)	265.00 (112.00-471.00)	269.00 (138.00-470.00)		
Anaemia (WHO criteria)			[a]	0.733
No	52 (88.1)	66 (84.6)		
Yes	7 (11.9)	12 (15.4)		

Data are presented as mean ± standard deviation and median (minimum-maximum) for continuous variables and as n (%) for categorical variables.
[a]: Pearson chi-square test, [b]: Mann-Whitney U test, [c]: Student's t-test
LDL-cholesterol was calculated using the Friedewald formula and was not computed in patients with triglycerides >400 mg/dL (n=6). eGFR was calculated using the 2021 CKD-EPI creatinine equation. Anaemia was defined according to World Health Organization criteria (haemoglobin <12 g/dL in women and <13 g/dL in men).
LDL: Lower low-density lipoprotein, HDL: High-density lipoprotein, AST: Aspartate aminotransferase, ALT: Alanine transaminase, eGFR: Epidermal growth factor receptors, WHO: World Health Organization

Table 4. Triglyceride/HDL-cholesterol ratio and triglyceride-glucose index according to glycemic control

	Good glycemic control (HbA1c <7%) (n=59)	Poor glycemic control (HbA1c ≥7%) (n=78)	Test	p
TG/HDL-C ratio	3.24±2.30	3.98±3.46	[b]	0.125
Median (min-max)	2.75 (0.87-15.38)	3.22 (0.86-23.05)		
TyG index	9.05±0.47	9.47±0.62	[b]	**<0.001**
Median (min-max)	9.09 (8.23-10.52)	9.43 (8.31-11.37)		

Data are presented as mean ± standard deviation and median (minimum-maximum) for continuous variables and as n (%) for categorical variables. TG/HDL: Triglyceride/High-density lipoprotein, TyG: Triglyceride-glucose
[a]: Pearson chi-square test, [b]: Mann-Whitney U test

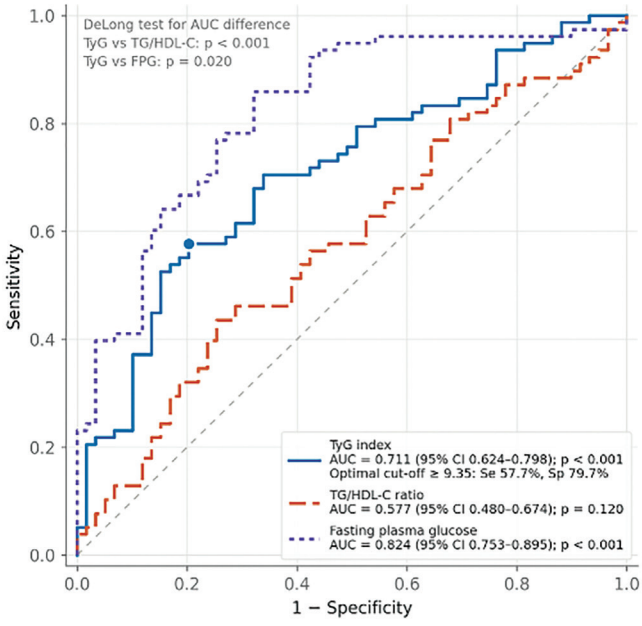

Figure 1. Comparative receiver operating characteristic curves of the TyG index, the triglyceride/HDL-cholesterol (TG/HDL-C) ratio and fasting plasma glucose for the identification of poor glycemic control (HbA1c ≥7%) in elderly patients with type 2 diabetes mellitus. Areas under the curve with 95% confidence intervals are shown in the legend; the optimal cut-off of the TyG index determined by the Youden index is marked. Areas under the curve were compared using the DeLong test

TyG: Triglyceride-glucose, AUC: Area under the curve, CI: Confidence interval

Discussion

In this single-centre cross-sectional study of a geriatric diabetic population, the TyG index was significantly higher in patients with poor glycemic control and remained independently associated with it after multivariable adjustment, whereas the TG/HDL-C ratio showed neither a significant group difference nor discriminative value. Formal comparison of the two ROC curves confirmed that the TyG index performed significantly better than the TG/HDL-C ratio, and this finding was reproduced in every sensitivity analysis performed. These results indicate that, of the two simple lipid-derived indices most often proposed for this purpose, only the TyG index carries information about glycemic control in elderly patients with type 2 diabetes.

The relationship between lipid-derived indices and glycemic control is generally interpreted as a reflection of underlying insulin resistance and metabolic dysregulation. The TyG index, derived from fasting triglyceride and glucose concentrations, has been proposed as a reliable surrogate indicator of insulin resistance in population-based studies, in which it has frequently shown associations with metabolic outcomes at least comparable to those of traditional indices such as HOMA-IR (20,21). In the present study the index was evaluated not as a substitute measure of insulin resistance but as a readily available marker of the combined lipid and glucose dysregulation that characterizes inadequately controlled diabetes in older adults. Recent studies have consistently reported that higher TyG index values are associated with poor glycemic control, an increased risk of type 2 DM and adverse cardiometabolic outcomes (22-24). Notably, Shang et al. (25) recently demonstrated a significant inverse relationship between the TyG index and time in range assessed by continuous glucose monitoring in patients with type 2 diabetes, which supports our findings using a reference standard independent of HbA1c and is therefore not subject to the limitations of HbA1c in older adults. Our observations extend this evidence specifically to a geriatric diabetic population, which remains relatively underrepresented in the literature.

The corrected cut-off value identified in our cohort, 9.35, lies within the range of thresholds reported in previous studies of the TyG index, which have generally fallen between approximately 8.5 and 9.5 for the identification of insulin resistance and dysglycemia (26). This external consistency supports the plausibility of our finding, and the associated adjusted odds ratio indicates that patients above this threshold have a substantially higher likelihood of inadequate glycemic control.

Table 5. Comparative receiver operating characteristic analysis for the prediction of poor glycemic control

Index	Cut-off	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Accuracy (%)	AUC	95% CI	p
TyG index	≥9.35	57.7	79.7	78.9	58.8	67.2	0.711	0.624-0.798	**<0.001**
TG/HDL-C ratio	≥3.69	43.6	74.6	69.4	50.0	56.9	0.577	0.480-0.674	0.120
Fasting plasma glucose	≥126.00	85.9	66.1	77.0	78.0	77.4	0.824	0.753-0.895	**<0.001**

DeLong test for pairwise comparison of areas under the curve: TyG index vs. TG/HDL-C ratio, difference 0.134 (z=4.19), p<0.001; TyG index vs. fasting plasma glucose, difference -0.113 (z=-2.33), p=0.020. PPV: Positive predictive value, NPV: Negative predictive value, CI: Confidence interval, TyG: Triglyceride-glucose, TG/HDL: Triglyceride/High-density lipoprotein, AUC: Area under the curve. Cut-off values were determined by the Youden index

Table 6. Multivariable logistic regression analysis for poor glycemic control (HbA1c ≥7%)

Model 1 — TyG index (continuous) (n=137)			
Variable	**Adjusted OR**	**95% CI**	**p**
TyG index (per 1 unit)	3.65	1.66-8.05	**0.001**
Age (per year)	1.04	0.95-1.15	0.385
Female sex	0.65	0.27-1.56	0.332
Diabetes duration >10 years	1.11	0.47-2.62	0.808
Beta-blocker use	2.98	0.96-9.20	0.058
DPP-4 inhibitor use	2.09	0.82-5.37	0.124
Number of oral antidiabetic drugs	1.00	0.65-1.55	0.997
Any insulin therapy	1.78	0.63-5.06	0.276
Body mass index (per kg/m^2)	0.97	0.88-1.07	0.530
Model 2 — TyG index ≥9.35 (dichotomised) (n=137)			
TyG index ≥9.35	4.94	2.15-11.33	**<0.001**
Age (per year)	1.04	0.95-1.15	0.394
Female sex	0.74	0.30-1.78	0.497
Diabetes duration >10 years	1.09	0.46-2.60	0.845
Beta-blocker use	2.86	0.90-9.12	0.076
DPP-4 inhibitor use	2.28	0.88-5.89	0.090
Number of oral antidiabetic drugs	1.00	0.64-1.56	0.999
Any insulin therapy	2.17	0.76-6.22	0.150
Body mass index (per kg/m^2)	0.95	0.86-1.05	0.356
Model 3 — TG/HDL-C ratio (continuous) (n=137)			
TG/HDL-C ratio (per 1 unit)	1.08	0.94-1.23	0.276
Age (per year)	1.04	0.95-1.14	0.447
Female sex	0.67	0.29-1.55	0.349
Diabetes duration >10 years	1.07	0.47-2.45	0.872
Beta-blocker use	3.66	1.23-10.88	0.020
DPP-4 inhibitor use	2.54	1.04-6.20	0.042
Number of oral antidiabetic drugs	0.96	0.63-1.46	0.853
Any insulin therapy	1.97	0.71-5.53	0.195
Body mass index (per kg/m^2)	0.97	0.89-1.07	0.572

Model 1- Nagelkerke R^2=0.275; likelihood ratio test p<0.001; Model 2- Nagelkerke R^2=0.298; likelihood ratio test p<0.001; Model 3- Nagelkerke R^2=0.182; likelihood ratio test p=0.018
All models were adjusted for age, sex, diabetes duration >10 years, beta-blocker use, DPP-4 inhibitor use, number of oral antidiabetic drugs, any insulin therapy and body mass index. OR: Odds ratio, CI: Confidence interval, DPP-4: Dipeptidyl peptidase-4, TyG: Triglyceride–glucose, TG/HDL: Triglyceride/HDL-cholesterol

Table 7. Sensitivity analyses

Analysis	n (good/poor)	TyG p	TyG AUC (95% CI)	TG/HDL-C p	TG/HDL-C AUC (95% CI)	DeLong p
Primary analysis (HbA1c ≥7.0%)	137 (59/78)	**<0.001**	0.711 (0.624-0.798)	0.125	0.577 (0.480-0.674)	**<0.001**
HbA1c ≥7.5%	137 (82/55)	**<0.001**	0.754 (0.670-0.839)	0.019	0.619 (0.520-0.717)	**<0.001**
HbA1c ≥8.0%	137 (97/40)	**<0.001**	0.723 (0.625-0.821)	0.189	0.572 (0.459-0.684)	**<0.001**
Excluding anaemic patients	118 (52/66)	**<0.001**	0.680 (0.583-0.777)	0.237	0.564 (0.459-0.668)	**<0.001**
Excluding triglycerides >400 mg/dL	131 (58/73)	**<0.001**	0.703 (0.614-0.792)	0.256	0.558 (0.458-0.658)	**<0.001**
Excluding insulin-treated patients	109 (51/58)	**<0.001**	0.738 (0.643-0.833)	0.171	0.576 (0.468-0.684)	**<0.001**
eGFR ≥60 mL/min/1.73 m² only	123 (50/73)	**<0.001**	0.736 (0.649-0.823)	**0.049**	0.605 (0.505-0.705)	**<0.001**

TyG: Triglyceride-glucose, TG/HDL: Triglyceride/High-density lipoprotein, AUC: Area under the curve, eGFR: Epidermal growth factor receptors

Table 8. Spearman correlation with HbA1c

Variable	Spearman ρ	p
TyG index	+0.491	**<0.001**
TG/HDL-C ratio	+0.164	0.056
Fasting plasma glucose	+0.715	**<0.001**
Triglycerides	+0.132	0.124
HDL-cholesterol	-0.131	0.128

TyG: Triglyceride-glucose, TG/HDL: Triglyceride/High-density lipoprotein

Our findings regarding the TG/HDL-C ratio differ from several earlier reports. Babic et al. (18), in a cross-sectional study of 113 diabetic patients of mixed age, found that both the TG/HDL-C ratio and the TyG index were elevated in patients with HbA1c ≥7% and elevated TG/HDL-C ratios have elsewhere been linked to atherogenic dyslipidemia, impaired glucose metabolism and cardiometabolic risk (10,27). Several considerations may explain the discrepancy. First, our population consisted exclusively of patients aged 65 years and older, in whom lipid metabolism is influenced by ageing-related changes and in whom HDL-C concentrations are less closely coupled to glycemic status. Second, and importantly, patients receiving lipid-lowering therapy were excluded from our study by design; in cohorts in which such treatment is permitted, differential statin use between glycemic control groups may itself generate apparent associations for a lipid-based ratio. Third, in our cohort none of the individual lipid parameters, including TG and HDL-C, differed significantly between glycemic control groups, so that a ratio composed of these two variables could not be expected to discriminate. This last observation also clarifies the origin of the difference in the TyG index between groups, which was driven predominantly by its glycemic rather than its lipid component.

This latter point deserves explicit consideration. Because FPG is a component of the TyG index and is itself strongly correlated with HbA1c, the discriminative performance of the index for an HbA1c-defined outcome is necessarily influenced by this shared variance. In our analysis, FPG alone discriminated poor glycemic control better than the TyG index (AUC 0.824 vs. 0.711; DeLong p=0.020). We report this openly, as it delimits the appropriate clinical interpretation of our findings: The TyG index should not be regarded as a superior substitute for fasting glucose when the objective is simply to identify an elevated HbA1c, since fasting glucose is both cheaper and more accurate for that purpose. The potential value of the TyG index lies elsewhere—in combining glycemic and lipid information into a single measure that reflects the broader metabolic disturbance of type 2 diabetes, and that may retain interpretability in precisely those situations, common in geriatric practice, in which HbA1c itself is unreliable because of anaemia, altered erythrocyte turnover or renal dysfunction (28). Prospective studies using glycemic reference standards independent of HbA1c, such as continuous glucose monitoring, will be required to establish whether this theoretical advantage translates into clinical utility.

From a pathophysiological perspective, the associations observed can be explained by the interplay between insulin resistance, lipid metabolism and glucose homeostasis. Insulin resistance promotes lipolysis, leading to elevated circulating free fatty acids and TG, while simultaneously impairing glucose uptake in peripheral tissues. This metabolic environment contributes to both hyperglycaemia and dyslipidaemia and is reflected in an elevated TyG index (29,30). In geriatric populations these mechanisms may be amplified by age-related decline in β-cell function, increased visceral adiposity, chronic low-grade inflammation and reduced physical activity (31).

The potential contribution of pharmacological treatment to the observed associations also warrants consideration. Several antidiabetic agents modify the components of the indices examined here: Metformin, SGLT-2 inhibitors

and GLP-1 receptor agonists lower fasting glucose and may reduce triglyceride concentrations, whereas thiazolidinediones influence both triglyceride and HDL-C levels. In our cohort most antidiabetic classes were comparably distributed between glycemic control groups, and the difference observed for DPP-4 inhibitors most plausibly reflects treatment intensification in response to inadequate control rather than a causal effect on the indices. DPP-4 inhibitor use was nevertheless entered as a covariate in all multivariable models, and its inclusion did not alter the independent association of the TyG index with glycemic control. Of particular note, beta-blocker use was significantly more frequent among patients with poor glycemic control. Non-vasodilating beta-blockers raise triglyceride concentrations, reduce HDL-C and impair insulin sensitivity, and in our multivariable analysis beta-blocker use was an independent predictor of poor glycemic control in the model containing the TG/HDL-C ratio but not in the models containing the TyG index. This pattern is consistent with the greater susceptibility of a purely lipid-based ratio to pharmacological confounding, and offers a further explanation for its poor performance in this setting. An identical pattern was observed for DPP-4 inhibitor use, which was independently associated with poor glycemic control in the model containing the TG/HDL-C ratio but not in those containing the TyG index. Importantly, patients receiving lipid-lowering therapy were excluded by design, thereby eliminating the single most powerful pharmacological confounder of lipid-derived indices—a methodological strength relative to much of the existing literature.

It should also be acknowledged that the overall strength of evidence supporting the TyG index requires a measured appraisal. In a recent umbrella review of 29 meta-analyses encompassing 95 associations, only 6.3% were supported by evidence of moderate quality, the majority resting on very low-quality evidence (32). Our findings should therefore be regarded as adding a geriatric-specific and methodologically controlled contribution to this literature rather than as establishing the index as a validated clinical tool.

From a clinical perspective, our findings carry two practical implications. First, they argue against the routine use of the TG/HDL-C ratio as a marker of glycemic control in elderly patients with established diabetes, since it provided no discriminative information in this population. Second, they identify the TyG index as the more informative of the two indices, with a moderate but statistically robust association with glycemic control that persisted after adjustment for demographic, treatment-related and anthropometric factors and across a wide range of sensitivity analyses. In elderly patients, in whom glycemic control is influenced by comorbidity, polypharmacy, nutritional status and functional capacity, and in whom reliance solely on HbA1c may not fully capture metabolic risk, an index that integrates glycemic and lipid information may serve as a useful adjunct to conventional assessment.

Study Limitations

The strengths of our study include its focus on a geriatric diabetic population, the exclusion of patients receiving lipid-lowering therapy and of conditions known to interfere with HbA1c interpretation, the comprehensive evaluation of both lipid- and glucose-based indices, and the use of formal comparative ROC analysis with multivariable adjustment and extensive sensitivity testing. Several limitations should nevertheless be recognized. First, the cross-sectional design prevents the establishment of causal relationships and restricts the assessment of temporal associations. Second, the single-centre design and the modest sample size may restrict the generalizability of the findings, and the study was not powered to detect small differences. Third, a uniform HbA1c threshold of 7% was applied to define poor glycemic control, whereas contemporary guidelines advocate targets individualized to comorbidity burden, cognitive and functional status and life expectancy; as comprehensive geriatric assessment data were not collected, patient-specific targets could not be applied, although sensitivity analyses at higher thresholds yielded consistent results. Fourth, although patients with overt conditions affecting erythrocyte turnover were excluded and haematological parameters did not differ between groups, serum bilirubin and iron status indices such as ferritin and transferrin saturation were not measured, so that subclinical iron deficiency and hyperbilirubinaemia of any degree could not be formally excluded as sources of analytical or biological interference with HbA1c; patients with chronic liver disease and those with clinically evident jaundice were, however, excluded on clinical grounds, and the HPLC method used here is comparatively resistant to bilirubin interference. Fifth, although antidiabetic treatment classes were largely comparable between groups, the cross-sectional design does not permit adjustment for cumulative treatment exposure, medication adherence or dose intensity. Finally, potential confounding factors including dietary habits, physical activity levels and inflammatory markers were not

assessed. Future prospective studies with larger cohorts, longitudinal follow-up and glycemic reference standards independent of HbA1c are needed to confirm these associations and to evaluate the predictive value of these indices for long-term outcomes such as cardiovascular events and mortality.

Conclusion

In elderly patients with type 2 DM, the TyG index was significantly associated with poor glycemic control and remained an independent predictor after multivariable adjustment, whereas the TG/HDL-C ratio showed no significant association and no discriminative value. Formal comparison of the two ROC curves demonstrated the superiority of the TyG index, and this result was robust across all sensitivity analyses. Of the two widely used lipid-derived indices, only the TyG index appears to carry information relevant to glycemic status in this age group. Given that its discriminative performance did not exceed that of FPG alone, the TyG index should be regarded as a complementary rather than a substitute measure, whose principal potential lies in settings where HbA1c interpretation is itself limited by comorbidity or altered erythrocyte turnover.

Ethics

Ethics Committee Approval: The study protocol was approved by the Institutional Ethics Committee of University of Health Sciences Turkey, Prof. Dr. Cemil Taşcıoğlu City Hospital (approval number: E-48670771-514.99-299780546, date: 30.12.2025) and the study was conducted in accordance with the Declaration of Helsinki.

Informed Consent: Informed written consent was obtained from all participants before enrolment.

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For transparency, the authors note that an artificial intelligence-assisted language model (ChatGPT, OpenAI) was utilized to support language correction. This assistance was limited to linguistic refinement; all scientific content, critical analysis, and final editorial decisions were made exclusively by the authors.

Footnotes

Authorship Contributions

Surgical and Medical Practices: M.G.A., A.Y., Concept: M.G.A., A.Y., Design: M.G.A., A.Y., Data Collection or Processing: M.G.A., A.Y., Analysis or Interpretation: M.G.A., A.Y., Literature Search: M.G.A., A.Y., Writing: M.G.A., A.Y.

Conflict of Interest: No conflict of interest was declared by the authors.

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