Turkish Journal of Clinics and Laboratory

To cite this article: Yılmaz A, Çon E. Association of Tp-e Interval, Tp-e/QT and Tp-e/QTc ratios with the triglyceride–glucose index: a retrospective cross-sectional study. Turk J Clin Lab 2025; 4: 582-590.

Research Article

Association of Tp-e Interval, Tp-e/QT and Tp-e/QTc ratios with the triglyceride–glucose index: a retrospective cross-sectional study

Trigliserit-glikoz indeksi ile tp-e aralığı, tp-e/qt ve tp-e/qtc oranları arasındaki ilişki: retrospektif kesitsel bir çalışma

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Abstract

Objective: The aim of this study was to evaluate the relationship between the triglyceride glucose (TyG) index, a practical biomarker of insulin resistance, and the electrocardiographic indicators of ventricular repolarisation, namely the Tp–e interval, Tp–e/QT, and Tp–e/QTc ratios.

Materials and Methods: This retrospective cross-sectional study was conducted at the Department of Cardiology, Faculty of Medicine, Karamanoğlu Mehmetbey University, between 1 January 2024 and 31 September 2025. The demographic, laboratory, and electrocardiographic data of 200 adult individuals who met the inclusion criteria were retrieved from the electronic record system. The TyG index was calculated from fasting glucose and triglyceride levels, and patients were divided into four quartiles (Q1–Q4) according to their TyG values. Tp–e, Tp–e/QT, and Tp–e/QTc measurements were performed by two independent observers from digital ECG recordings. Intergroup differences were evaluated using the ANOVA test, and relationships were assessed using correlation and multivariate regression analyses.

Results: The mean age of participants was 51.5 ± 18.8 years, and 51% were male. With an increase in the TyG index, the Tp–e duration ($r = 0.558$, $p < 0.001$), Tp–e/QT ratio ($r = 0.374$, $p < 0.001$), and Tp–e/QTc ratio ($r = 0.422$, $p < 0.001$) significantly increased. In multivariate regression analysis, the TyG index remained an independent predictor of the Tp–e interval ($\beta = 0.38$, $p < 0.001$). According to ROC analysis, a TyG cutoff value > 8.70 predicted the presence of prolonged Tp–e with 77% sensitivity and 72% specificity (AUC = 0.78, $p < 0.001$).

Conclusion: This study is the first to demonstrate an independent association between the TyG index and the Tp–e interval, Tp–e/QT, and Tp–e/QTc ratios. These findings suggest that metabolic stress may contribute to cardiac electrical instability by increasing the heterogeneity of ventricular repolarisation. The TyG index, as an easily calculable parameter, may be a potential biomarker for early detection of subclinical arrhythmic risk.

Keywords: triglyceride-glucose index, Tp–e interval, Tp–e/QT ratio, Tp–e/QTc ratio, ventricular repolarisation, arrhythmogenic risk

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Doi: 10.18663/tjcl.1820757
Received: 10.11.2025 accepted: 08.12.2025

Öz

Amaç: Bu çalışmanın amacı, insülin direncinin pratik bir biyobelirteci olan trigliserit glikoz (TyG) indeksi ile ventriküler repolarizasyonun elektrokardiyografik göstergeleri olan Tp-e aralığı, Tp-e/QT ve Tp-e/QTc oranları arasındaki ilişkiyi değerlendirmektir.

Gereç ve Yöntemler: Bu retrospektif kesitsel çalışma, 1 Ocak 2024 ile 30 Eylül 2025 tarihleri arasında Karamanoğlu Mehmetbey Üniversitesi Tıp Fakültesi Kardiyoloji Anabilim Dalı'nda yürütüldü. Dahil edilme kriterlerini karşılayan 200 yetişkin bireyin demografik, laboratuvar ve elektrokardiyografik verileri elektronik kayıt sisteminden alındı. TyG indeksi, açlık glikozu ve trigliserit seviyelerinden hesaplandı ve hastalar TyG değerlerine göre dört çeyreğe (Q1–Q4) ayrıldı. Tp-e, Tp-e/QT ve Tp-e/QTc ölçümleri, dijital EKG kayıtlarından iki bağımsız gözlemci tarafından yapıldı. Gruplar arası farklılıklar ANOVA testi ile, ilişkiler ise korelasyon ve çok değişkenli regresyon analizleri ile değerlendirildi.

Bulgular: Katılımcıların ortalama yaşı 51,5 ± 18,8 yıl olup, %51'i erkekti. TyG indeksindeki artışla birlikte Tp-e süresi ($r = 0,558$, $p < 0,001$), Tp-e/QT oranı ($r = 0,374$, $p < 0,001$) ve Tp-e/QTc oranı ($r = 0,422$, $p < 0,001$) anlamlı şekilde arttı. Çok değişkenli regresyon analizinde TyG indeksi, Tp-e aralığının bağımsız bir öngördürücüsü olarak kaldı ($\beta = 0,38$, $p < 0,001$). ROC analizine göre, 8,70'in üzerindeki bir TyG eşik değeri, uzamış Tp-e varlığını %77 duyarlılık ve %72 özgüllük ile öngördü (AUC = 0,78, $p < 0,001$).

Sonuçlar: Bu çalışma, TyG indeksi ile Tp-e aralığı, Tp-e/QT ve Tp-e/QTc oranları arasındaki bağımsız ilişkiyi gösteren ilk çalışmadır. Bu bulgular, metabolik stresin ventriküler repolarizasyon heterojenliğini artırarak kardiyak elektriksel instabiliteye katkıda bulunabileceğini düşündürmektedir. Kolayca hesaplanabilen bir parametre olan TyG indeksi, subklinik aritmik riskin erken tespiti için potansiyel bir biyobelirteç olabilir.

Anahtar Kelimeler: trigliserit-glikoz indeksi, tp-e aralığı, tp-e/qt oranı, tp-e/qtc oranı, ventriküler repolarizasyon, aritmik risk

Introduction

Ventricular repolarisation heterogeneity is one of the fundamental electrophysiological basis for malignant arrhythmias and sudden cardiac death (SCD). The Tp–e interval (Tp–e), measured from the peak (Tpeak) to the end of the T wave (Tend) on the surface electrocardiogram (ECG), is a reliable indicator of the transmural repolarisation distribution of the ventricular myocardium and is considered an important parameter for determining arrhythmogenic potential [1,2]. The Tp–e/QT and Tp–e/QTc ratios, on the other hand, are derivative indicators that allow for the assessment of repolarisation dispersion independently of heart rate and are increasingly used in clinical research [3,4]. The reported upper limit values for Tp–e and corrected Tp–e (Tp–e-c) in healthy adults, approximately 90 ms and 100 ms respectively, provide a practical reference range for evaluating subclinical repolarisation prolongation [5]. As the spectrum of coronary artery disease (CAD) progresses, a marked prolongation in Tp–e-based parameters is observed, with Tp–e, Tp–e/QT, and Tp–e/ QTc (corrected QT interval) ratios reported to increase significantly, particularly in acute ischaemic syndromes [4,6,7].

The triglyceride-glucose (TyG) index, calculated from fasting triglyceride and glucose levels, has been defined as an easily applicable biomarker of insulin resistance [8]. In recent years, data suggesting that the TyG index may be associated not only with metabolic status but also with cardiovascular remodelling and electrical instability have been increasing. Large cohort studies have demonstrated that as TyG levels increase, the likelihood of QTc prolongation is independently elevated [6]. Furthermore, recent studies have shown a significant, linear, and independent association between increased TyG index and the risk of developing atrial fibrillation (AF). These studies have revealed that the TyG index is not only an indicator of metabolic dysfunction but also a potential electrophysiological risk marker reflecting cardiac electrical remodelling processes. The findings suggest that glucose-lipid interaction associated with insulin resistance and atherogenic dyslipidaemia may affect ion channel expression and repolarisation continuity in myocytes, thereby suggesting that the TyG index may be associated with subclinical atrial arrhythmogenicity [9-11]. These findings suggest that metabolic dysfunction may increase arrhythmogenic risk through ventricular repolarisation dynamics.

Although Tp–e–based markers have been shown to be elevated in various endocrine, inflammatory, and structural heart diseases, reflecting increased arrhythmic risk [12-14]. Few studies have investigated the direct relationship between these repolarisation markers and the TyG index, a marker

of metabolic stress. Elucidating the relationship between metabolic dysfunction and ventricular electrical heterogeneity may provide valuable insights into the identification of arrhythmogenic susceptibility in the early stages.

This retrospective cross-sectional study aimed to evaluate the relationship between the Tp–e interval, Tp–e/QT, and Tp–e/QTc ratios and the TyG index. The aim of our study is to reveal the effect of the TyG index, an easily obtainable metabolic marker, on ventricular repolarisation and to provide a new perspective on the possible link between metabolic risk and electrical instability.

Material and Methods

This retrospective, single-centre, observational cross-sectional study was conducted between 1 January 2024 and 31 September 2025 at the Department of Cardiology, Faculty of Medicine, Karamanoğlu Mehmetbey University, and the Cardiology Clinic, Karaman Training and Research Hospital. The aim of the study was to evaluate the relationship between the triglyceride glucose (TyG) index and the electrocardiographic indicators of ventricular repolarisation, namely the Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio. Patient data were obtained from the electronic hospital record system of individuals who attended the cardiology outpatient clinic.

The study protocol was conducted in accordance with the principles of the Helsinki Declaration and was approved by the Clinical Research Ethics Committee of Karamanoğlu Mehmetbey University Faculty of Medicine (date: 16.10.2025, decision no: 24-2025/20). As the study had a retrospective design, the requirement for written informed consent was deemed exempt by the ethics committee. All patient data were anonymised, and full compliance with confidentiality and data security principles was ensured.

Patients aged 18 years or older with documented sinus rhythm were eligible for inclusion, provided they had concurrent measurements of electrolyte levels, urea, creatinine, fasting blood glucose, and lipid profiles on the same day as a standard 12-lead electrocardiogram (ECG) recorded at 25 mm/s and 10 mm/mV. Conversely, patients were excluded if they exhibited rhythm disturbances such as atrial fibrillation or flutter, a QRS duration exceeding 120 ms, or a history of acute coronary syndrome, coronary artery disease, myocardial infarction, congenital heart disease, advanced heart failure, cardiomyopathy, or known channelopathies. Additionally, individuals with electrolyte imbalances, those using medications known to affect the QT interval, and those with incomplete or unanalysable ECG or laboratory data were excluded. To ensure the accuracy of baseline triglyceride–glucose (TyG) index values, patients with a prior diagnosis of diabetes or hypertriglyceridaemia, as well as those receiving lipid-lowering or glucose-altering therapies, were also excluded from the study. According to these criteria, 200 adult patients were included in the study.

Electrocardiographic Measurements

ECG recordings were obtained in the supine position after 5 minutes of rest, at a speed of 25 mm/s and a calibration of 10 mm/mV. The Tp-e interval was measured as the time from the peak of the T wave (Tpeak) to its end (Tend); the V5 lead was preferred, with V4 or V6 leads used when inappropriate. The QT interval was measured from the onset of the QRS complex to the end of the T wave, and QTc was calculated using the Bazett formula ($QTc = QT/\sqrt{RR}$). All measurements were performed digitally by two independent cardiologists, blinded to clinical data. Each parameter was measured in three consecutive cycles, and the mean value was taken. The Tp-e/QT and Tp-e/QTc ratios were calculated by dividing Tp-e by QT and QTc, respectively. Inter-observer measurement reliability was assessed using the intraclass correlation coefficient (ICC) and found to be > 0.90.

Biochemical Measurements and TyG Index Calculation

Venous blood samples taken after an 8–12-hour fast from participants were used. Laboratory levels such as serum glucose, triglycerides, total cholesterol, LDL, HDL, HbA1c, and creatinine were obtained from the hospital information processing electronic record system The TyG index was calculated using the following formula $TyG = \ln((\text{Triglycerides (mg/dL)} \times \text{Glucose (mg/dL)}) / 2)$ Participants were divided into four quartiles (Q1-Q4) from low to high based on the median TyG value.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics v.26.0 (IBM Corp., USA) software. Data distribution was assessed using the Kolmogorov–Smirnov test. Variables showing a normal distribution were expressed as mean ± standard deviation (SD), and those not normally distributed were expressed as median interquartile range (IQR). For intergroup comparisons, one-way ANOVA was used for variables showing a normal distribution,

and the Kruskal–Wallis test was used for those not normally distributed. Categorical variables were evaluated using the chi-square test. The relationship between the TyG index and Tp-e, Tp-e/QT, and Tp-e/QTc was assessed using Pearson or Spearman correlation analysis. Multivariate linear regression models were created to determine the independent predictors affecting Tp-e parameters. The predictive performance of the TyG index for prolonged repolarisation markers (Tp-e ≥ 75.5 ms or Tp-e/QTc ≥ 0.25) for the electrocardiogram [5, 15] was examined using receiver operating characteristic (ROC) analysis; area under the curve (AUC), sensitivity, specificity, and Youden's index were calculated. Statistical significance was set at $p < 0.05$.

Results

A total of 200 cases were included in the study. The mean age of participants was 51.5 ± 18.8 years, with ages ranging from 18 to 84 years. The male-to-female ratio was 51%. The mean body mass index (BMI) was 26.7 ± 4.1 kg/m^2 (Table 1). The mean Tp-e interval was 76.0 ± 5.8 ms, the Tp-e/QT ratio was 0.196 ± 0.018, and the Tp-e/QTc ratio was 0.177 ± 0.015 (Table 2). According to the Kolmogorov–Smirnov test, the variables of age, glucose, triglycerides, HDL, and creatinine deviated from normal distribution ($p<0.05$). Therefore, these variables are presented as median (IQR), while the others are presented as mean ± SD.

The cases included in the study were divided into four quartiles (Q1–Q4) from low to high according to the distribution of the TyG index. Demographic variables (age, gender, body mass index) were found to be similar between groups (all $p > 0.05$). When metabolic parameters were examined, a significant increase in glucose ($p=0.007$) and triglycerides ($p<0.001$) levels was observed with increasing TyG quartiles, while a significant decrease in LDL cholesterol levels ($p = 0.036$) was noted. This decrease is thought to be due to age and gender. No differences were found between groups in terms of HDL cholesterol, creatinine, and eGFR ($p > 0.05$). A gradual increase in the prevalence of hypertension was observed according to TyG quartiles (24% in Q1, 42% in Q4), but this increase did not reach statistical significance ($p = 0.093$); only a trend-level increase was observed. The mean diastolic BP increased from 76.4 ± 9.8 mmHg in the Q1 group to 84.7 ± 8.9 mmHg in the Q3 group. This trend, along with the increase in the TyG index, suggests that there may be an increase in peripheral vascular resistance and arterial stiffness (Table 1). These results support the notion that TyG increases as a reflection of metabolic impairment and cardiometabolic burden.

According to Pearson correlation analysis results, a significant and positive relationship was found between the TyG index and ventricular repolarisation parameters. A strong correlation was found between the TyG index and Tp-e duration ($r = 0.558$, $p < 0.001$), Tp-e/QT ratio ($r = 0.374$, $p < 0.001$), and Tp-e/QTc ratio ($r = 0.422$, $p < 0.001$). These findings indicate that an increase in the TyG index is associated with prolongation of ventricular repolarisation parameters. No significant correlation was found between the TyG index and QTc (Bazett) ($p > 0.05$).

When ventricular repolarisation parameters were compared according to TyG quartiles, significant differences were found between groups in terms of Tp-e, Tp-e/QT ratio, and Tp-e/QTc ratio ($p < 0.001$, one-way ANOVA). Homogeneity of variance was confirmed by the Levene test ($p > 0.05$). According to the Tukey HSD post-hoc analysis, Tp-e, Tp-e/QT, and Tp-e/QTc values in the highest TyG quartile (Q4) were significantly higher than in Q1, Q2, and Q3 (all $p < 0.001$), whereas no significant differences were observed among the lower quartiles themselves (all $p > 0.05$). This pattern suggests a clear dose–response relationship, with a marked step-up in repolarisation indices at the highest TyG levels.

In the multivariate linear regression analysis, the Tp–e interval was included as the dependent variable; age, gender, BMI, LDL, HDL, and TyG index were included as independent variables in the model. The TyG index remained an independent predictor of the Tp–e interval (standardized $\beta = 0.38$, 95% CI: 0.21–0.56; $p < 0.001$; $R^2 \approx 0.29$–0.31). Each one-unit increase in the TyG index corresponded approximately to a 5.8 ms increase in absolute Tp–e duration, indicating a clinically relevant effect size, whereas none of the other covariates showed a significant association with Tp–e (all $p > 0.05$).

The power of the TyG index in predicting prolonged Tp–e was evaluated in ROC curve analysis. The AUC value for TyG was calculated as 0.78 (95% CI: 0.71–0.85; $p < 0.001$). When the cut-off value was TyG > 8.70, sensitivity was 77%, specificity was 72%, and the Youden index was 0.49. The TyG index also demonstrated significant discriminatory performance in predicting Tp–e/QTc > 0.25 (AUC = 0.74, $p < 0.001$) (Figure 1).

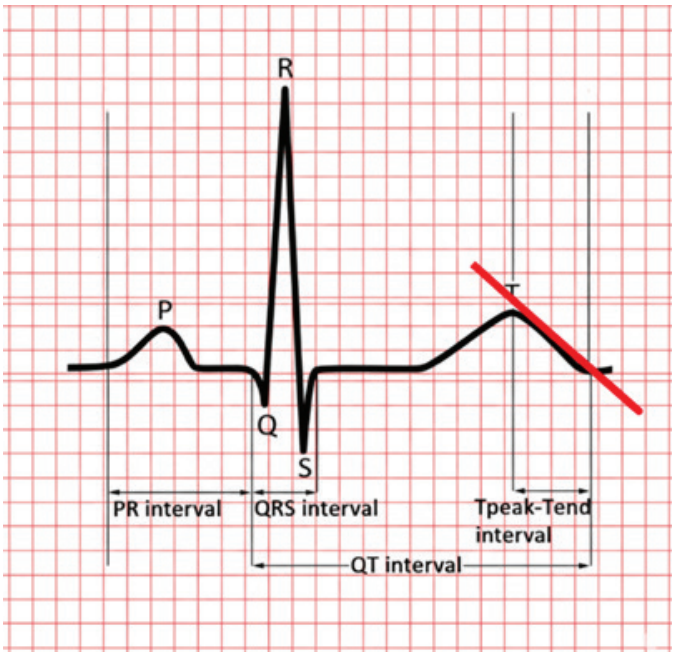

Figure 1. Tp-e interval and QT interval measurement.

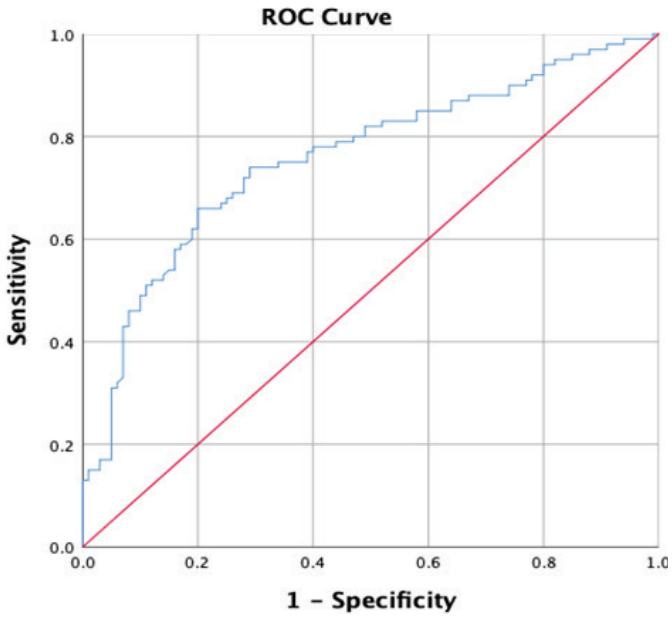

Figure 2. ROC curve analysis shows the discriminatory performance of the Triglyceride-Glucose (TyG) index in predicting the presence of prolonged Tp–e. (AUC) 0.78 (95% CI: 0.71–0.85; $p < 0.001$).

Boxplot analyses revealed a gradual increase in Tp-e, Tp-e/QT, and Tp-e/QTc values across TyG quartiles. Both the median and spread were markedly increased in the Q4 group, supporting that increased metabolic stress parallels ventricular electrical heterogeneity (Figure 2).

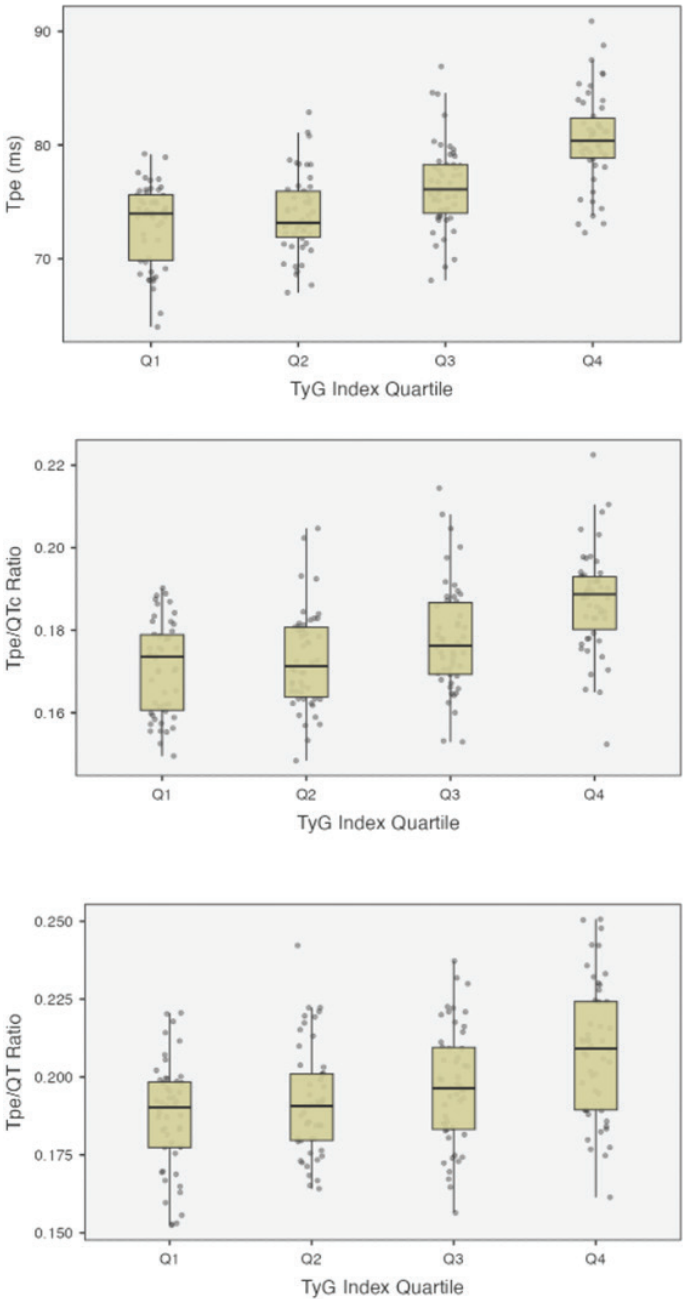

Figure 3. Distribution of ventricular repolarisation parameters according to TyG index quartiles (Q1–Q4). (The upper panel shows the Tp-e duration, the middle panel shows the Tp-e/QTc ratio, and the lower panel shows the Tp-e/QT ratio.)

These findings indicate a marked tendency for prolongation in transmural heterogeneity of ventricular repolarisation with increasing metabolic risk, suggesting a potential increase in arrhythmogenic risk in individuals with high TyG levels.

Table 1. Baseline demographic and clinical characteristics according to TyG quartiles (Continuous variables are presented as mean±SD or median (IQR), as appropriate. p-values were obtained by one-way ANOVA or Kruskal–Wallis test, and χ^2 test for categorical variables, $p < 0.05$).

Variable	Q1 (Lowest) (n=50)	Q2 (n=50)	Q3 (n=50)	Q4 (Highest) (n=50)	Total (n=200)	p-value
Age (years)	47.5 (34.5–64.0)	56.0 (38.0–67.0)	46.5 (36.0–64.8)	53.0 (37.5–69.8)	51.5 (36.0–66.2)	0.395
Gender (Male, %)	50	48	64	42	51	0.158
Height (cm)	169.7 ± 9.1	169.7 ± 10.1	170.1 ± 8.1	165.6 ± 9.4	168.8 ± 9.3	0.050
Weight (kg)	75.8 ± 12.3	76.0 ± 13.1	78.7 ± 11.5	73.9 ± 12.4	76.1 ± 12.4	0.268
BMI (kg/m^2)	27.1 ± 4.4	26.4 ± 4.5	27.2 ± 3.5	26.9 ± 4.2	26.7 ± 4.1	0.703
Systolic BP (mmHg)	130.7 ± 16.9	129.5 ± 13.8	128.2 ± 14.0	129.0 ± 14.9	129.4 ± 14.9	0.858
Diastolic BP (mmHg)	76.4 ± 9.8	80.8 ± 8.1	84.7 ± 8.9	77.4 ± 9.6	79.8 ± 9.6	<0.001
Hypertension (%)	24	22	42	42	34	0.093
Heart rate (bpm)	72.8 ± 10.1	73.4 ± 9.4	74.1 ± 9.3	74.6 ± 11.5	73.7 ± 10.1	0.829
Glucose (mg/dl)	98.5 (91.0-111.0)	100.5 (96.2–108.8)	102.0 (94.2–112.8)	108.5 (103.0–117.0)	104.0 (97.0–112.0)	0.007
Triglycerides (mg/dl)	95.5 (81.2–108.5)	130.0 (118.2–139.0)	160.0 (153.0–180.8)	210.5 (185.0–238.8)	145.0 (115.0–183.0)	<0.001
LDL (mg/dl)	119.7 ± 23.9	125.4 ± 28.2	120.1 ± 27.3	110.0 ± 26.7	118.8 ± 26.9	0.036
HDL (mg/dl)	48.0 (43.2–53.0)	48.0 (41.2–53.0)	47.0 (39.0–53.8)	45.5 (38.0–52.8)	47.0 (40.0–53.0)	0.449
Creatinine (mg/dl)	1.0 (0.8–1.1)	0.9 (0.8–1.1)	0.9 (0.7–1.1)	0.9 (0.8–1.1)	0.9 (0.8–1.2)	0.577

Table 2. Electrocardiographic parameters according to TyG quartiles (Continuous variables are presented as mean ± SD, $p < 0.05$.

Variable	Q1 (n=50)	Q2 (n=50)	Q3 (n=50)	Q4 (n=50)	Total (n=200)	p-value
TyG index	8.53 ± 0.12	8.83 ± 0.09	9.03 ± 0.10	9.35 ± 0.18	8.93 ± 0.36	< 0.001
Tp-e (ms)	73.1 ± 4.2	73.8 ± 4.7	76.4 ± 5.1	80.5 ± 6.2	76.0 ± 5.8	< 0.001
Tp-e/QT ratio	0.187 ± 0.014	0.192 ± 0.016	0.197 ± 0.017	0.208 ± 0.019	0.196 ± 0.018	< 0.001
Tp-e/QTc ratio	0.171 ± 0.012	0.173 ± 0.013	0.178 ± 0.015	0.187 ± 0.016	0.177 ± 0.015	< 0.001
QTc (Bazett, ms)	428.1 ± 15.2	428.2 ± 17.1	430.3 ± 18.7	430.8 ± 19.3	429.3 ± 17.5	0.839

Discussion

In this study, the effect of the triglyceride-glucose (TyG) index on ventricular repolarisation parameters was evaluated. According to our findings, significant differences were detected in Tp-e duration, Tp-e/QT ratio, and Tp-e/QTc ratio among the four quartile groups formed based on the TyG index ($p<0.001$). In particular, these parameters were significantly prolonged in patients in the highest TyG quartile (Q4) ($p<0.001$). The findings support the adverse effects of metabolic stress and insulin resistance on cardiac electrical stability and suggest that the TyG index may be a potential biomarker for identifying subclinical arrhythmogenic risk.

The TyG index is accepted as an easy and reliable indicator of insulin resistance and has been associated with various metabolic and cardiovascular risk factors [16]. Previous studies have reported that high TyG levels are associated with subclinical atherosclerosis, endothelial dysfunction, and cardiac remodelling [17] . In recent years, studies investigating the effect of the TyG index on cardiac electrical activity have also increased. Lee et al. reported that QTc prolongation increased independently as the TyG index rose in Chinese male workers [6]. Similarly, Tian et al. noted that insulin resistance and glycaemic markers showed a significant correlation with major ECG abnormalities in an elderly population [9]. Studies by Vandael et al. and Zhao et al. also reported that high TyG levels are associated with QT dispersion and repolarisation heterogeneity [17,18]. Furthermore, the UK Biobank analysis demonstrated a linear relationship between the TyG index and atrial fibrillation risk [11] .

Pathophysiologically, chronic exposure to hyperglycaemia and elevated triglyceride levels creates a metabolic milieu that is unfavourable for stable myocardial electrophysiology. Experimental and clinical data suggest that oxidative stress, lipotoxicity, and ion-channel remodelling—particularly

involving repolarizing potassium currents (I_Kr and I_Ks)—may collectively prolong action potential duration and increase transmural dispersion of repolarisation. Hyperglycaemia-related oxidative stress further modifies ion-channel kinetics, while the accompanying autonomic imbalance characterised by enhanced sympathetic activity and reduced vagal tone augments dispersion of repolarisation. Clinically, these mechanisms manifest as prolongation of Tp-e and its derivatives, which are strong predictors of malignant ventricular arrhythmias and sudden cardiac death [19]. Indeed, Porthan et al. demonstrated that Tp-e parameters outperform QTc in predicting sudden cardiac death in the general population [7]. Consistent with this literature, our study demonstrates a significant association between the TyG index and ventricular repolarisation dispersion.

Tp-e and Tp-e–derived ratios offer a more specific assessment of arrhythmogenic risk than QTc because they directly reflect transmural dispersion of repolarisation, the key substrate for malignant ventricular arrhythmias. QTc represents global repolarisation duration, whereas Tp-e captures regional heterogeneity across the myocardial wall. Supporting this, Porthan et al. showed that Tp-e parameters outperform QTc in predicting sudden cardiac death [7]. In this context, the independent association between TyG and Tp-e-based markers in our study reinforces the electrophysiological relevance of TyG as a potential indicator of ventricular vulnerability.

In our correlation analysis, significant positive relationships were found between the TyG index and Tp-e duration ($p<0.001$), Tp-e/QT ratio ($p<0.001$), and Tp-e/QTc ratio ($p<0.001$). This result supports the notion that ventricular repolarisation is prolonged with increasing metabolic impairment. Regression analysis also demonstrated that the TyG index is an independent predictor of Tp-e and related ratios, with each unit increase in TyG corresponding to an approximate 5.8 ms prolongation in Tp-e duration. This finding suggests that the TyG index may be an indicator not only of metabolic but also of electrophysiological risk.

In addition, recent studies have demonstrated a significant association between the TyG index and cardiac autonomic nervous system function. For example, Balcıoğlu et al. examined the TyG index in 400 adults without metabolic syndrome using 24-hour Holter-ECG-based HRV (Heart Rate Variability) and HRT (Heart Rate Turbulence) parameters; they reported that TyG was an independent predictor of HRV/HRT [20]. In this context, it is thought that the Tp–e and Tp–e/QTc prolongation observed with high TyG levels in our study may also be related to impaired autonomic cardiac regulation mechanisms. This theory supports the idea that insulin resistance and high triglycerides may increase transmural repolarisation heterogeneity by decreasing vagal tone and increasing sympathetic tone.

The literature has shown that in cases with obstructive coronary artery disease, the Tp-e interval is independently associated with cardiac death, while QTc may remain normal [1,21]. In our study, the parallel increase in the TyG index with the prolongation of Tp-e, Tp-e/QT, and Tp-e/QTc ratios suggests that metabolic dysfunction may converge with atherosclerotic processes on a common pathophysiological axis. The effect of TyG on repolarisation, when combined with ischaemic mechanisms, suggests that both metabolic stress and coronary flow impairment may increase cardiac electrical heterogeneity. Therefore, the significant prolongation of Tp-e, Tp-e/QT, and Tp-e/QTc ratios ($p<0.001$) parallel to the increase in TyG in our study may be an indicator of subclinical ischaemia and early electrical reflections of atherosclerotic processes.

Supporting our findings, Eyiol et al. demonstrated that higher TyG levels independently predict greater disease severity and adverse outcomes in myocarditis, underscoring TyG as a marker of metabolic burden and cardiac vulnerability [22]. Additionally, Tezcan et al. showed that metabolic disturbances such as vitamin D deficiency significantly influence repolarization markers, while their complementary work in fibromyalgia highlighted the contribution of autonomic imbalance to repolarization behaviour. Together, these studies emphasize that both metabolic stress and autonomic dysregulation play key roles in shaping ventricular electrical heterogeneity [23,24].

A strength of this study is that the relationship between the TyG index and repolarisation parameters was evaluated in the same cohort using multivariate statistical analyses (ANOVA, post-hoc, correlation, regression). However, the cross-sectional design of the study limits causal interpretations. Nevertheless, the homogeneous patient distribution and systematic measurement method mitigate the impact of these limitations.

One of the key findings of our study is that when the cut-off value for TyG is >8.70, the TyG index can predict the presence of prolonged Tp–e with 77% sensitivity and 72% specificity (AUC = 0.78) ($p<0.001$). This cut-off value represents the optimal threshold determined in our study for ventricular repolarisation prolongation. However, the mean values reported in the literature for the TyG index vary according to the metabolic risk

profile of the population. For example, TyG is generally reported to be in the range of 8.6–9.0 in individuals with metabolic syndrome or prediabetes, and in the range of 7.8–8.4 in the healthy population [6]. Therefore, external validation in diverse and prospective cohorts is required before this cut-off value can be generalized to broader clinical practice. The cut-off point of 8.70 in our study is close to the upper limit of these ranges, indicating the threshold for early metabolic dysfunction. Therefore, in individuals with TyG > 8.70, not only metabolic impairment but also the onset of subclinical cardiac electrical instability should be considered. This cut-off value can be used as an "early warning" indicator in clinical practice.

Limitations of the study

This study has several important limitations that should be acknowledged. First, the retrospective and single-centre design restricts the establishment of causal relationships and may limit the generalisability of the findings to broader populations. Second, because the study lacked longitudinal follow-up, we were unable to evaluate the occurrence of clinical arrhythmic events or determine whether the observed repolarisation abnormalities translated into adverse outcomes. Third, several potential confounders that may influence ventricular repolarisation could not be fully controlled. These include variability in electrolyte levels (particularly potassium and calcium), the use of medications known to affect QT and Tp-e intervals, and the absence of objective assessments of autonomic function—such as heart rate variability (HRV) or heart rate turbulence (HRT). Despite excluding patients with overt rhythm disturbances or metabolic derangements, residual confounding remains possible. Finally, prospective, multicentre studies with detailed electrophysiological evaluation and arrhythmic follow-up are needed to validate our findings and clarify the clinical implications of the TyG–repolarisation relationship.

In conclusion, to our knowledge, this is the first study in the literature to statistically demonstrate the independent relationship between the TyG index and the Tp–e interval, Tp–e/QT, and Tp–e/QTc ratios within the same model. Previous studies have generally associated the TyG index with QTc prolongation or atrial arrhythmias; however, our study, which examines the direct relationship with Tp–e-based parameters reflecting the transmural heterogeneity of ventricular repolarisation, is valuable as it is the first study in the literature in this regard. In this respect, our study makes a unique contribution to the literature by demonstrating the bridge mechanism between metabolic stress and cardiac electrical instability at the clinical level. The findings support that insulin resistance may be a determinant not only of metabolic dysfunction but also of subclinical ventricular arrhythmogenicity risk. These results suggest that the TyG index is practical, easy to calculate, and could potentially be used as an early arrhythmic risk indicator. Future prospective studies will make an important contribution to confirming the predictive value of TyG for arrhythmic events.

Declaration of conflicting interests

The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding

The authors received no financial support for the research and/or authorship of this article.

Ethics approval

This study was approved by Karamanoğlu Mehmetbey University Faculty of Medicine Clinical Research Ethics Committee with protocol number 24-2025/20.

Authors' contribution

AY: Concept, Design, Materials, Data Collection and/or Processing, Analysis and/or Interpretation, Literature Review, Writing of the Article. EÇ: Design, Supervision, Sources, Materials, Data Collection and/or Processing, Critical Review.

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