Vildan Ozer[1], Ozlem Bulbul[2], Savas Ozer[3], Ercan Aydın[3],
Ozgen Gonenc Cekic[4], Hulya Karakus[5], Arif Mansur Cosar[6]

[1] Karadeniz Technical University, Faculty of Medicine, Department of Emergency Medicine, Trabzon, Turkey

[2] Ministry of Health Giresun Prof Dr A Ilhan Ozdemir State Hospital, Emergency Department, Giresun, Turkey

[3] Trabzon Kanuni Training and Research Hospital, Cardiology Clinic, Trabzon, Turkey

[4] Trabzon Kanuni Training and Research Hospital, Department of Emergency Medicine, Trabzon, Turkey

[5] Ministry of Health Merzifon Kara Mustafa Pasa State Hospital, Emergency Department, Amasya, Turkey

[6] Karadeniz Technical University, Faculty of Medicine, Department of Internal Medicine, Division of Gastroenterology, Trabzon, Turkey

Association Between Tp-e Interval Prolongation and Myocardial Injury in Gastrointestinal Bleeding

Aim	This study aimed to evaluate ventricular arrhythmia risk among patients who developed myocardial injury due to gastrointestinal bleeding by using the Tp-e interval, a predictive marker of ventricular arrhythmia. It is known that myocardial injury may develop secondary to gastrointestinal bleeding. This condition has been linked to an elevated risk of cardiac arrhythmia, a condition for which the Tp-e interval on electrocardiography is a marker.
Material and methods	The study included patients who presented to the emergency department with subsequently confirmed gastrointestinal bleeding. Based on high-sensitivity troponin T (hs-TnT) concentrations at presentation, two groups were formed: Patients with myocardial injury (hs-TnT >14 ng/l) and those without injury (hs-TnT ≤14 ng/l). Ventricular repolarization markers were measured to explore the association between these parameters and myocardial injury.
Results	Among cases of gastrointestinal bleeding, those presenting with myocardial injury exhibited significantly elevated Tp-e interval (73.5±9.1 vs. 62.1±6.8 msec, p<0.001) and Tp-e/QTc (0.16±0.02 vs. 0.13±0.07, p<0.001) values compared to those without myocardial injury. Myocardial injury correlated positively with the Tp-e interval and Tp-e/QTc (r=0.577 and r=0.528, respectively; p<0.001 for each).
Conclusion	In cases of gastrointestinal bleeding, myocardial injury was positively correlated with prolonged Tp-e interval and increased Tp-e/QTc, parameters associated with elevated risk of ventricular arrhythmia. The clinical significance of this association remains uncertain and requires confirmation and further assessment in future studies.

Keywords	Gastrointestinal bleeding; myocardial injury; Tp-e interval; Tp-e/QTc
For citations	Vildan Ozer, Ozlem Bulbul, Savas Ozer, Ercan Aydın, Ozgen Gonenc Cekic, Hulya Karakus, Arif Mansur Cosar. Association Between Tp-e Interval Prolongation and Myocardial Injury in Gastrointestinal Bleeding. Kardiologiia. 2026;66(3):87–92. [Russian: Вильдан Озер, Озлем Бульбул, Савас Озер, Эрджан Айдын, Озген Гоненч Чекич, Хулья Каракус, Ариф Мансур Косар. Взаимосвязь между удлинением интервала Tp-e и повреждением миокарда у пациентов с желудочно-кишечным кровотечением. Кардиология. 2026;66(3):87–92].

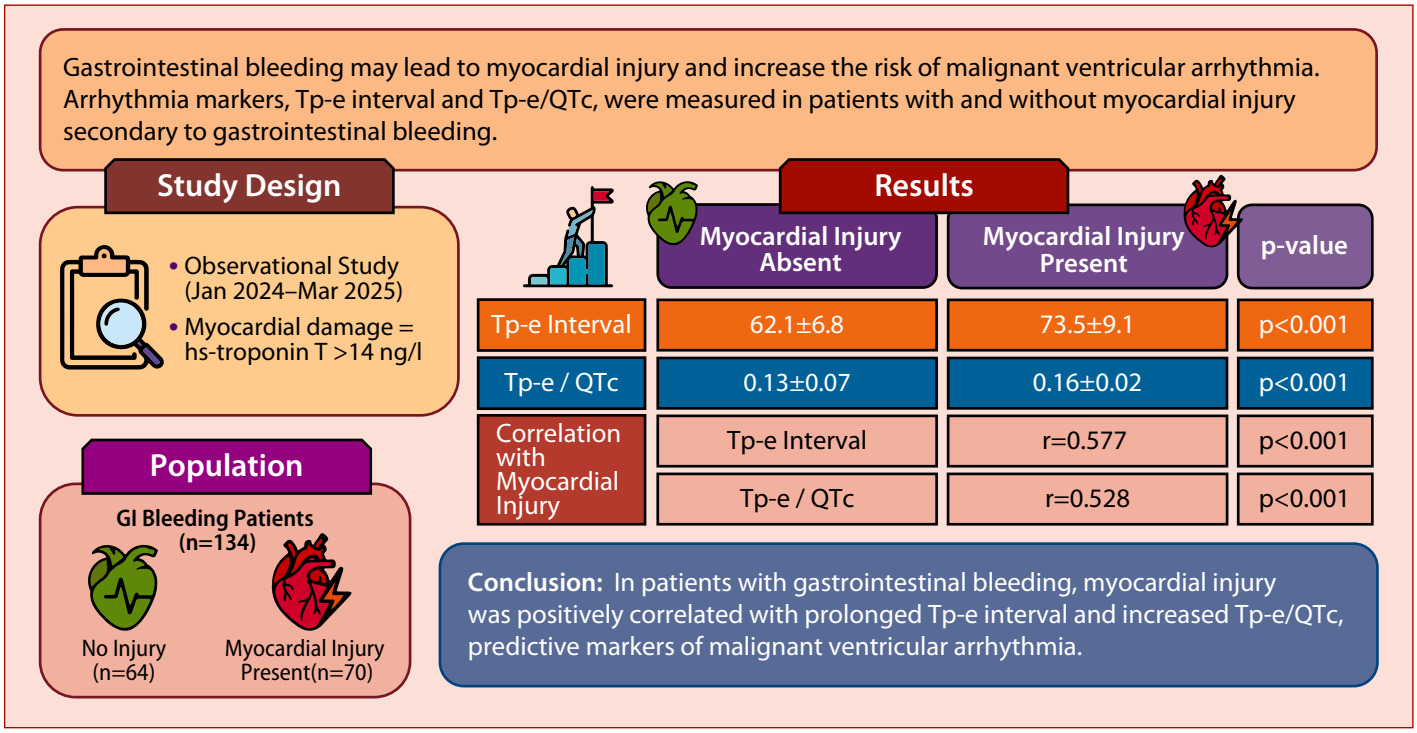
Corresponding author	Vildan Ozer. Email:dr.vilzan@hotmail.com

Introduction

Gastrointestinal bleeding is a commonly encountered and life-threatening clinical emergency [1, 2]. Gastrointestinal bleeding occurs due to a range of factors including the growing use of antiplatelet and anticoagulant agents, malignancies, smoking, stress, and the widespread use of non-steroidal anti-inflammatory agents. Despite increased clinical focus on the prevention and management of gastrointestinal bleeding, this patient population continues to experience elevated rates of morbidity and mortality [3, 4]. It has been suggested that cardiovascular events occurring in patients with gastrointestinal bleeding are associated with increased mortality. This association may be linked to myocardial injury, which may develop secondary to gastrointestinal bleeding [5, 6]. Various hypotheses have been proposed to explain the connection between gastrointestinal bleeding-induced myocardial injury. In cases of massive bleeding, hypovolemia, hemodynamic instability, and myocardial hypoperfusion may lead to myocardial injury [7]. In addition, reduced renal perfusion may result in increased myocardial oxidative stress and inflammation [8].

The presence of myocardial injury has been linked to cardiac arrhythmia [9]. The Tp-e interval is electrocardiographically obtained by measuring the T wave from the peak to the end [10]. It has been suggested that the Tp-e interval can be used as an index of total (transmural, apico-basal, and

Central Illustration. Association Between Tp-e Interval Prolongation and Myocardial Injury in Gastrointestinal Bleeding



global) repolarization dispersion, and that prolongation of the Tp-e interval is a non-invasive marker for predicting malignant ventricular arrhythmia [10]. Prolongation of the Tp-e interval has also been associated with an increased risk of sudden cardiac death (SCD) in patients with idiopathic ventricular premature complexes [11]. Prolongations in other parameters that are related to ventricular repolarization (e.g., QT and QTc intervals), are also regarded as non-invasive predictive markers of malignant ventricular arrhythmogenesis [12].

While gastrointestinal bleeding has been linked to substantial mortality and morbidity, cardiac events are generally assessed in the context of myocardial perfusion disturbances, type 2 myocardial infarction, and myocardial injury. While the literature contains reports on myocardial injury and arrhythmia following gastrointestinal bleeding, there is a limited number of studies specifically evaluating ventricular repolarization markers such as the Tp-e interval and Tp-e/QTc ratio in this clinical context. Thus, this study comparatively evaluated malignant ventricular arrhythmia markers, namely including the Tp-e interval, and Tp-e/QTc ratio, between patients who developed myocardial injury secondary to gastrointestinal bleeding and those who did not. Additionally, the association between myocardial injury and ventricular repolarization parameters in this patient population was addressed.

Material and methods

Study population

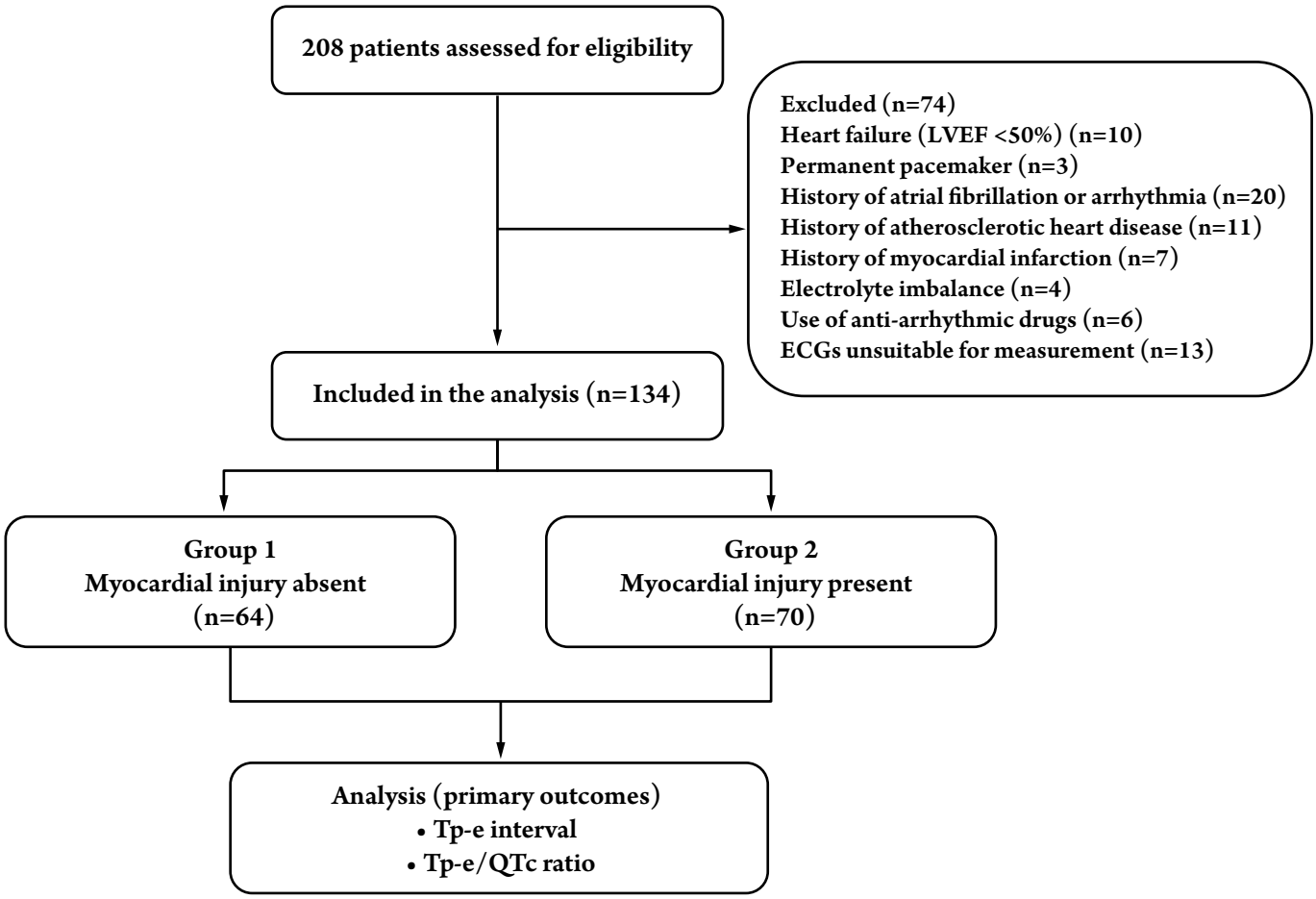
Patients who visited the emergency department between January 2024 and March 2025, due to signs of gastrointestinal bleeding, such as hematemesis, melena, or hematochezia, and whose diagnosis was confirmed via endoscopy and/or colonoscopy were assessed for eligibility. Patients with conditions that might have affected electrocardiographic measurements of the investigated parameters were excluded from the study. These conditions were atherosclerotic heart disease, myocardial infarction, heart failure (based on a left ventricular ejection fraction value of <50%), permanent pacemakers, atrial fibrillation, arrhythmia history, electrolyte imbalance, use of antiarrhythmic drugs. Also excluded were patients whose electrocardiograms (ECGs) did not allow measurements. After excluding patients based on these criteria, the sample prospectively consisted of the remaining 134 cases.

In our hospital laboratory, the upper limit of the troponin test kit (Elecsys Troponin T-hs, Roche Diagnostics, Mannheim, Germany) was 14 ng/l. Based on high-sensitivity troponin T (hs-TnT) concentrations obtained at the time of presentation, two groups were formed: Group 1, consisting of those without myocardial injury (hs-TnT ≤14 ng/l), and Group 2, comprising those with myocardial injury (hs-TnT >14 ng/l). The groups were compared in terms of demographics, biochemical values, and ventricular arrhythmia marker concentrations (Figure 1).

All patients provided verbal and written informed consent before participating in the study. The study was conducted in accordance with the principles stated in the Declaration of Helsinki and received approval from the Scientific Research Ethics Committee of Karadeniz Technical University Faculty of Medicine (approval number: 2024–50).

ISSN 0022-9040. Кардиология. 2026;66(3). DOI: 10.18087/cardio.2026.3.n3024

Figure 1. Flowchart of the patient selection and analysis



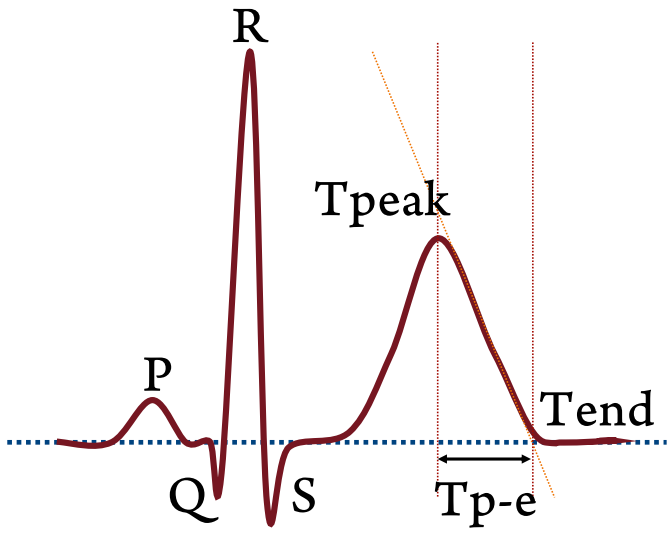
Electrocardiographic measurements

Standard 12-lead ECG measurements were acquired from patients in the supine position with electrodes placed at standard anatomical locations. The following parameters were used: filter range, 0.15–100 Hz; AC filter, 50 Hz; recorder speed, 25 mm/s; amplification, 10 mm/V. A Cardiofax GEM 9022 K model ECG instrument was used (Nihon Kohden, Tokyo, Japan). Manual ECG measurements using calipers and magnifying glasses were made by two cardiologists who were unaware of the patient's group. Basic ECG parameters including heart rate, PR interval, QRS duration, QT interval, and corrected QT (QTc) interval were recorded manually. Bazett's formula, defined as

$$QTc = QT/\sqrt{(R\text{–}R\ interval)}$$

was used for the calculation of the QTc interval [13]. The Tp-e interval was measured from the T wave as the distance from the peak to the point where the tangent of the terminal T wave intersects the isoelectric baseline (Figure 2). From these measurements, the ratios Tp-e/QT and Tp-e/QTc were calculated. Each ECG was evaluated independently, and the average of minimum two measurements performed consecutively was used to improve accuracy. ECGs were excluded if fewer than nine leads were measurable. The following ventricular repolarization values were obtained: QT interval, QTc interval, QT dispersion, QTc dispersion, Tp-e interval, Tp-e/QT ratio, and Tp-e/QTc ratio.

Figure 2. Tpeak to Tend (Tp-e) interval measurement



T wave end was designated as the point where the tangent on the terminal T wave intersected with the Tp-e baseline [14].

Table 1. Demographic and laboratory values of Groups 1 and 2

Parameters	Group 1 (n=64) (47.8%)	Group 2 (n=70) (52.2%)	p-value
Age (yrs)	55±18	73±15	<0.001
Male (%)	41 (64.1)	42 (60)	0.629
Diabetes mellitus (%)	15 (23.4)	33 (49.1)	0.004
Hypertension (%)	24 (37.5)	44 (62.9)	0.003
CVD (%)	5 (7.8)	6 (8.6)	0.873
Smoker (%)	2 (3.1)	3 (54.3)	0.723
COPD (%)	4 (6.3)	2 (2.9)	0.343
Systolic BP (mmHg)	130±5	125±6	0.652
Diastolic BP (mmHg)	70±2	65±2	0.563
Hemoglobin (g/dl)	9.9±2.2	8.8±2	0.007
Creatinine (mg/dl)	0.8±0.2	1.1±0.4	<0.001
BMI (kg/m^2)	28±3	30±4	0.115
ALT (U/l)	22±7	21±9	0.733
AST (U/l)	29±8	37±6	0.395
LDH (U/l)	246±100	263+90	0.470
Estimated GFR (ml/min/1.73 m^2)	92±20	65±14	<0.001
Sodium (mmol/l)	138±2	138±4	0.243
Potassium (mmol/l)	4.3±0.4	4.5±0.6	0.256

Data are mean±SD or number (percentage). Group 1, myocardial injury absent; Group 2, myocardial injury present; CVD, cerebrovascular disease; COPD, chronic obstructive pulmonary disease; LDH, lactate dehydrogenase; GFR, glomerular filtration rate; BP, blood pressure; BMI, body mass index; ALT, alanine aminotransferase; AST, aspartate aminotransferase.

Statistical analysis

The Kolmogorov-Smirnov test was utilized to evaluate whether the data had a normal distribution. Normally distributed numerical variables are presented as means and standard deviations (SD). The chi-square test was used to analyze differences in values of categorical variables, and the independent-samples t-test was used intergroup comparison of continuous variables. Spearman correlation analysis was performed to evaluate between group correlations. The significance level was taken as $p < 0.05$.

Results

The sample consisted of 134 patients who presented to the emergency department with gastrointestinal bleeding. 47.8% of these patients did not develop myocardial injury secondary to gastrointestinal bleeding (Group 1), whereas 52.2% of the patients developed secondary myocardial injury (Group 2). The demographic and laboratory values of the patients are presented in Table 1. The means ages of Groups 1 and 2 were 55±18 yrs and 73±15 yrs, respectively (p<0.001). The prevalence of diabetes mellitus and hypertension was significantly greater in Group 2. Also, the mean estimated glomerular filtration rate was significantly lower in Group 2. Other demographic data, laboratory test results, and patient clinical values did not differ significantly between the groups.

Table 2. The Tp-e interval, QT interval, and related values of Groups 1 and 2

Parameters	Group 1 (n=64) (47.8%)	Group 2 (n=70) (52.2%)	p-value
Tp-e interval, (msec)	62.1±6.8	73.5±9.1	<0.001
QT interval, (msec)	385±47	388±42	0.741
QT interval, corrected, (msec)	452±33	453±38	0.762
QT/QTc	0.85±0.7	0.85±0.6	0.870
Tp-e/QTc	0.13±0.07	0.16±0.02	<0.001

Group 1, myocardial injury absent; Group 2, myocardial injury present Tp-e, T wave peak to T wave end interval; QTc, QT was corrected using Bazett's formula.

Table 3. Correlations between myocardial injury and the Tp-e interval and the Tp-e/QTc values

Parameters	R	p-value
Tp-e interval	0.577	<0.001
Tp-e/QTc ratio	0.528	<0.001

Table 2 presents the comparison of the Tp-e and QT/QTc values between Groups 1 and 2. The Tp-e interval and the Tp-e/QTc ratio were significantly elevated in Group 2.

Table 3 presents correlations between myocardial injury and the Tp-e interval and the Tp-e/QTc ratio values. These correlations were moderate but significant.

Discussion

In this study, we observed that patients who developed myocardial injury secondary to gastrointestinal bleeding exhibited significantly elevated Tp-e intervals and Tp-e/QTc ratios compared to values measured patients who did not develop myocardial injury. These elevated values are considered predictive markers of malignant ventricular arrhythmia. Furthermore, both the Tp-e interval and the Tp-e/QTc ratio were positively correlated with myocardial injury.

Myocardial injury frequently develops secondary to gastrointestinal bleeding and has been associated with both short- and long-term increases in mortality [15, 16]. The presence of myocardial injury has also been shown to correlate with the incidence of cardiac arrhythmias [9]. Sourour et al. investigated the association between myocardial injury and ventricular arrhythmia in cases of heart failure [9]. They found that myocardial injury elevated the risk of ventricular arrhythmia, and they noted that myocardial injury independently increased the possibility of ventricular arrhythmia development, regardless of patient age, left ventricular systolic function, previous myocardial infarction, and renal function.

An increase in the Tp-e interval has been found to be associated with cardiac adverse events, malignant ventricular arrhythmia, and mortality [12, 17, 18]. Increases in

ISSN 0022-9040. Кардиология. 2026;66(3). DOI: 10.18087/cardio.2026.3.n3024

the Tp-e/QT ratio have similarly been correlated with cardiovascular mortality and malignant ventricular arrhythmia [17, 18]. Zhao et al. observed that in patients presenting with idiopathic ventricular premature complexes, a prolonged Tp-e interval significantly elevated the risk of SCD [11]. Shimizu et al. compared patients with hypertrophic cardiomyopathy who did and did not develop SCD and found a significantly increased Tp-e/QT ratio in the SCD group [19]. Shu et al. followed 120 patients with ST-segment elevation for an average of 17 months after percutaneous coronary intervention and determined that Tp-e/QT was significantly higher among patients who developed malignant ventricular arrhythmia compared to those without malignant ventricular arrhythmia (0.32±0.07 vs. 0.26±0.05, p<0.000) [17]. The current study found significantly higher values for Tp-e interval and Tp-e/QTc in the myocardial injury group with gastrointestinal bleeding. These findings suggest that myocardial injury associated with gastrointestinal bleeding may elevate the risk of ventricular arrhythmia and potentially contribute to adverse clinical outcomes.

SCD, which most often develops secondary to ventricular arrhythmia, is responsible for nearly half of mortality cases related to cardiovascular events [20]. Despite advances in preventive measures and treatments resulting in a significant overall reduction in cardiovascular mortality, this benefit has been less pronounced for SCD [21, 22]. Therefore, there is a pressing need for updated risk stratification models to better assess the risk of SCD in patients with gastrointestinal bleeding. Preventing complications after myocardial injury has critical value. Thus, we suggest that closer monitoring of patients with myocardial injury who exhibit a prolonged Tp-e interval and a higher Tp-e/QTc ratio may help to reduce both morbidity and mortality. Early detection of prolonged Tp-e interval and higher Tp-e/QTc values in patients with gastrointestinal bleeding may assist in risk stratification and clinical management of these patients.

In summary, in patients with gastrointestinal bleeding, this study compared ventricular repolarization parameters between patients with and without myocardial injury to assess the risk of malignant ventricular arrhythmia. The results show a significant association and positive correlation between myocardial injury secondary to gastrointestinal bleeding and increases in the Tp-e interval and the Tp-e/QTc values. The tendency toward development of malignant ventricular arrhythmia in patients with gastrointestinal bleeding and increased Tp-e interval and the Tp-e/QTc values requires confirmation through prospective studies with clinical endpoints.

Limitations

There are several limitations to note:

1. The limited sample size.
2. Due to the cross-sectional design, the findings are unable to establish causal relationships.
3. The patients were evaluated solely on clinical and electrocardiographic data obtained emergency department presentation, and no long-term follow-up was conducted.

Conclusion

Patients who developed myocardial injury secondary to gastrointestinal bleeding had a significant increase in the Tp-e interval and in the Tp-e/QTc ratio, parameters associated with elevated risk of malignant cardiac arrhythmia. Long-term, arrhythmic follow-up studies are required to establish the clinical relevance of these findings.

No conflict of interest is reported.

The article was received on 20/08/2025

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ISSN 0022-9040. Кардиология. 2026;66(3). DOI: 10.18087/cardio.2026.3.n3024