Submitted: 17 March, 2025 Accepted: 27 May, 2025 Published: 08 December, 2025 DOI:10.22514/sv.2025.194

Open Access
Signa Vitae

ORIGINAL RESEARCH

Correlation of frontal QRS-T angle with mortality in ischemic stroke patients: a multicenter study

Murat Genç[1,*], Didem Çankaya Gökdere[2], Adil Emre Gezer[3], Melih Çamcı[4], Bensu Bulut[5], Medine Akkan Öz[6], Ramiz Yazıcı[7], Huseyin Mutlu[8], Zekeriya Uykan[9]

[1]Department of Emergency Medicine, Ankara Training and Research Hospital, 06230 Ankara, Turkey
[2]Department of Emergency Medicine, Bodrum State Hospital, 48480 Muğla, Turkey
[3]Department of Emergency Medicine, Ankara Etlik City Hospital, 06170 Ankara, Turkey
[4]Department of Emergency Medicine, Ankara Bilkent City Hospital, Yıldırım Beyazıt University, 06800 Ankara, Turkey
[5]Department of Emergency Medicine, Ankara Gulhane Training and Research Hospital, Health Science University, 06010 Ankara, Turkey
[6]Department of Emergency Medicine, Ankara Gulhane Training and Research Hospital, 06010 Ankara, Turkey
[7]Department of Emergency Medicine, Istanbul Kanuni Sultan Suleyman Training and Research Hospital, Health Science University, 34303 Istanbul, Turkey
[8]Department of Emergency Medicine, Aksaray Training and Research Hospital, Aksaray University, 68200 Aksaray, Turkey
[9]College of Engineering and Technology, American University of the Middle East, 54200 Egaila, Kuwait

***Correspondence**
murat.genc4@saglik.gov.tr
(Murat Genç)

Abstract

Background: Acute ischemic stroke (AIS) is a leading cause of mortality and morbidity worldwide. Cardiac electrical abnormalities, such as changes in the frontal QRS-T angle (fQRS-TA), have been linked to adverse outcomes in various cardiovascular disorders. However, the prognostic significance of fQRS-TA in patients with AIS has not been clearly established. This study aimed to evaluate the association between fQRS-TA and 30-day mortality in patients with AIS. **Methods**: This prospective, two-center observational study was conducted at Bilkent City Hospital and Etlik City Hospital between 01 June and 31 July, 2023. Patients aged $\geq$18 years diagnosed with AIS were enrolled, whereas individuals with hemorrhagic stroke, congenital heart disease, a history of cerebrovascular events or significant electrocardiographic abnormalities were excluded. Baseline demographic data, clinical parameters, and electrocardiogram (ECG) findings were collected. fQRS-TA was defined as the absolute difference between the frontal plane QRS and T-wave axes. Patients were classified into survivor and deceased groups based on 30-day outcomes. Statistical analysis was performed using SPSS version 25. **Results**: The data of 85 AIS patients were assessed (mean age: 67.29 $\pm$ 12.88 years; 51.8% female), and the results demonstrated that fQRS-TA was significantly elevated in patients with hypertension and congestive heart failure ($p < 0.05$). A weak positive correlation was observed between age and fQRS-TA ($p = 0.017$), with the deceased group demonstrating significantly higher fQRS-TA values than the survivors ($p = 0.045$). The NIHSS (National Institutes of Health Stroke Scale) scores were also found to be significantly higher in the deceased group ($p = 0.014$). **Conclusions**: fQRS-TA is an easily obtainable ECG parameter that may serve as a predictor of short-term mortality in AIS patients. When combined with NIHSS scores, fQRS-TA may improve risk stratification and inform early clinical decision-making. Further large-scale, randomized studies are needed to confirm these findings.

Keywords

Ischemic stroke; Frontal QRS-T angle; Electrocardiography; Mortality prediction; NIHSS

1. Introduction

Ischemic stroke, the second leading cause of death and disability worldwide, is a major complication of cardiovascular disease and presents with clinical presentations ranging from transient ischemic attacks to extensive cerebral infarction [1–3]. In these patients, increased atherosclerotic burden and age-related myocardial fibrosis may influence cardiac conduction, contributing to increased mortality risks [4]. In addition, recent evidence suggests that acute ischemic stroke (AIS) can induce immediate adverse effects on cardiac function, including electrical disturbances [5–7], and these acute neurocardiogenic alterations have been linked to long-term outcomes in AIS patients [5, 6]. Given the five-year cumulative mortality rate, which ranges from 1% to 7%, early risk stratification and the development of appropriate therapeutic strategies remain essential [8].

During AIS, a cascade of neurochemical processes, referred to as the ischemic cascade, is initiated following the reduction in cerebral blood flow. This cascade involves excitotoxicity, oxidative stress, peri-infarct depolarizations, blood-brain barrier disruption, microvascular injury, altered hemostasis, inflammation and apoptosis. Although these processes evolve over several hours, they may continue for days even after achieving reperfusion. In line with previous findings, our study identified elevated serum troponin and urea levels, which may

reflect systemic effects of the inflammatory response triggered by cerebral ischemia [7–9].

The ischemic core, the region of the brain directly surrounding the obstructed vessel, is usually the most severely damaged. Cellular necrosis in this area occurs rapidly through mechanisms such as lipolysis and proteolysis, making the tissue unsalvageable even despite prompt reperfusion [7–10].

Electrocardiography (ECG) is a widely used, non-invasive diagnostic method for detecting cardiac electrical alterations in various clinical settings, including emergency and acute care units [9]. A standard 12-lead ECG provides multiple electrical parameters, including QT interval and ST segment changes, which have demonstrated prognostic relevance in cardiovascular morbidity and mortality [10, 11]. Among these parameters, the frontal QRS-T angle (fQRS-TA), defined as the absolute difference between the frontal plane QRS and T-wave axes, has emerged as a potential marker for mortality risk [9, 12]. Elevated fQRS-TA has been associated with increased mortality in conditions such as acute myocardial infarction, hypertension, coronary artery disease and malignant arrhythmias [12–14].

Given that AIS can lead to cardiac electrical abnormalities, it is plausible that fQRS-TA may also be affected. Therefore, this study aimed to assess changes in fQRS-TA among patients with AIS and to investigate its association with 30-day mortality.

2. Materials and methods

2.1 Study design and participants

This two-center, prospective observational study was conducted between 01 June 2023, and 31 July 2023, at Bilkent City Hospital and Etlik City Hospital, both located in Ankara, Türkiye. Bilkent City Hospital serves an average of 100,000 patients per month, while Etlik City Hospital serves an average of 120,000 patients monthly. Both institutions are designated comprehensive stroke centers. Ethical approval for this study was obtained from the Bilkent City Hospital Clinical Research Ethics Committee No. 2 (Approval Number: E2-23-4296), and the study was conducted following the principles outlined in the Declaration of Helsinki. This prospective study was conducted with the necessary institutional approvals, and informed consent was obtained from all participants.

Patients aged 18 years or older who were diagnosed with AIS in the emergency department were eligible for study inclusion. Exclusion criteria comprised a history of previous cerebrovascular incident (CVI), acute hemorrhagic stroke (AHI), congenital heart disease, acute or chronic infectious or inflammatory disease, pregnancy or lactation and chronic liver disease. Additionally, patients for whom NIHSS scores were not calculated, or ECGs were not recorded were excluded. Patients with complete or incomplete right or left bundle branch block, early repolarization, or ventricular hypertrophy on ECG were also excluded due to the potential confounding effects of these conditions on the fQRS-T angle.

2.2 Study protocol and data collection

Patients presenting to the emergency department with a diagnosis of AIS were initially assessed by an Emergency Medicine specialist or resident in either the red or yellow emergency triage areas. Simultaneously, physical examination, fingerstick blood glucose testing, ECG acquisition, and routine blood sampling were performed. The evaluating physician calculated and recorded the NIHSS score at the time of initial assessment. Once the patient was stabilized, non-contrast cranial computed tomography (CT) imaging was conducted. Patients with suspected AIS were quickly referred to neurology for consideration of thrombolysis or embolectomy to minimize treatment delays. AIS was defined in accordance with the American Heart Association/American Stroke Association (AHA/ASA) guidelines as follows [15]:

(a) acute symptom onset within 0–4.5 hours;

(b) presence of neurological deficits on physical examination;

(c) confirmation of ischemic stroke by cranial CT or magnetic resonance imaging (MRI); and

(d) exclusion of cerebral hemorrhage and non-vascular etiologies.

The AIS patients' demographic, clinical, and laboratory data were retrieved from the hospital's electronic medical records. The collected information included age, sex, history of chronic diseases, ECG parameters (such as QT, QRS, fQRS-TA), NIHSS score at admission, and the duration of hospitalization in both the general ward and intensive care unit (ICU). Based on 30-day follow-up outcomes, patients were classified into two groups: those who died from any cause within 30 days (deceased group) and those who survived (survivor group).

2.3 Electrocardiogram (ECG)

A standard 12-lead ECG was obtained and analyzed for each patient using a device with an acquisition rate of 25 mm/s and an amplitude scale of 10 mm/mV (Cardioline, Trento, Italy). The QRS and T axes were automatically calculated and reported by the ECG system. As described in previous studies, fQRS-TA was determined by calculating the absolute difference between the frontal plane QRS axis and the T-wave axis. For cases where the calculated value exceeded 180°, the fQRS-TA was determined using the formula: 360° − angle [16]. An example illustrating the use of the ECG device to determine fQRS-TA is presented in Fig. 1, where the section labeled "axes" displays the P, QRS and T axes sequentially.

2.4 Statistical analysis

Statistical analysis was performed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics for categorical variables were presented as frequencies and percentages, while continuous variables were expressed as mean ± standard deviation (SD) or median with interquartile range (Q1–Q3), as appropriate. The distribution of continuous variables was assessed for normality. For comparisons between two independent groups, the Mann-Whitney U test was employed due to the non-normal distribution of dependent vari-

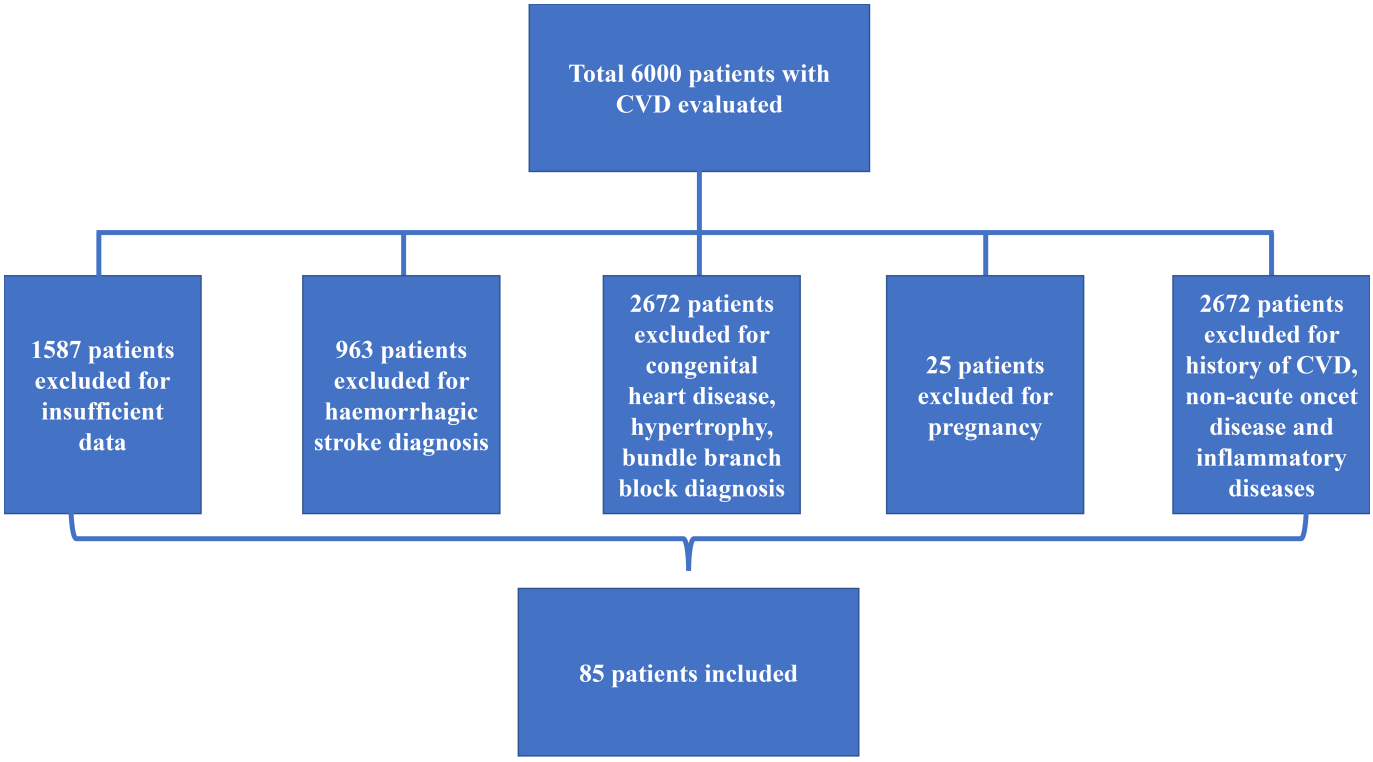

FIGURE 1. Inclusion and exclusion criteria. CVD: cardiovascular disease.

ables. Correlation analyses were conducted using Spearman's rank correlation coefficient, as the variables did not meet the assumptions of normality.

3. Results

A total of 85 patients meeting the inclusion criteria from both centers were enrolled in the study. A flowchart of the study design and patient selection is shown in Fig. 2.

The mean age of patients with AIS was 67.29 ± 12.88 years, and 51.8% (n = 44) were female. A weak positive correlation was observed between age and fQRS-TA ($r = 0.259$, $p = 0.017$). No significant correlation was identified between age and NIHSS score. Patients admitted to Bilkent City Hospital had significantly higher NIHSS scores compared to those admitted to Etlik City Hospital ($p = 0.001$). Additionally, fQRS-TA values were significantly higher in patients with a history of hypertension (HTN) and congestive heart failure (CHF) than in those without these conditions ($p < 0.05$). The comparison of fQRS-TA and NIHSS scores based on the sociodemographic and clinical characteristics of AIS patients is summarized in Table 1.

A weak positive correlation was identified between serum urea and troponin levels and fQRS-TA, while a moderate positive correlation was found between white blood cell (WBC)/neutrophil counts and NIHSS scores. These relationships between laboratory parameters and both fQRS-TA and NIHSS scores are presented in Table 2.

A positive correlation was also noted between fQRS-TA and NIHSS scores in patients with AIS, as shown in Table 3.

The median fQRS-TA in deceased patients was 92.5 (Interquartile Range (IQR): 36.5–154.75), significantly higher than the median value of 47 (IQR: 19–99) observed in survivors ($p = 0.045$). Similarly, the median NIHSS score was significantly higher in deceased patients ($p = 0.014$). The associations of fQRS-TA and NIHSS scores with 30-day mortality are detailed in Table 4.

4. Discussion

In this study, fQRS-TA values were higher in patients with HTN, CHF and older age compared to those without these characteristics. Both fQRS-TA and NIHSS scores were significantly elevated in patients who died within 30 days, indicating that these parameters may be associated with short-term mortality in AIS. Moreover, we observed a positive correlation between fQRS-TA and NIHSS scores in patients with AIS ($p = 0.023$).

O'Donnell MJ *et al.* [17], in a large multicenter study involving cardiovascular disease (CVD) patients from 22 countries, reported that AIS frequently occurs at older ages and is commonly associated with comorbidities such as HTN, diabetes mellitus (DM) and atrial fibrillation (AF). Studies investigating the relationship between fQRS-TA and conditions such as coronary artery disease, CHF, and AF have found that elevated fQRS-TA is associated with older age [18–20]. Similarly, Tassone *et al.* [19] demonstrated a significant increase in fQRS-TA in patients with HTN, while Böyük *et al.* [20] reported a similar association in a case-control study examining elevated blood pressure. Consistent with these findings, our results indicated that fQRS-TA increased with age and was higher in AIS patients with HTN and CHF.

Becker *et al.* [21] showed that elevated high-sensitivity cardiac troponin (Hs-cTn) levels in AIS patients were often due

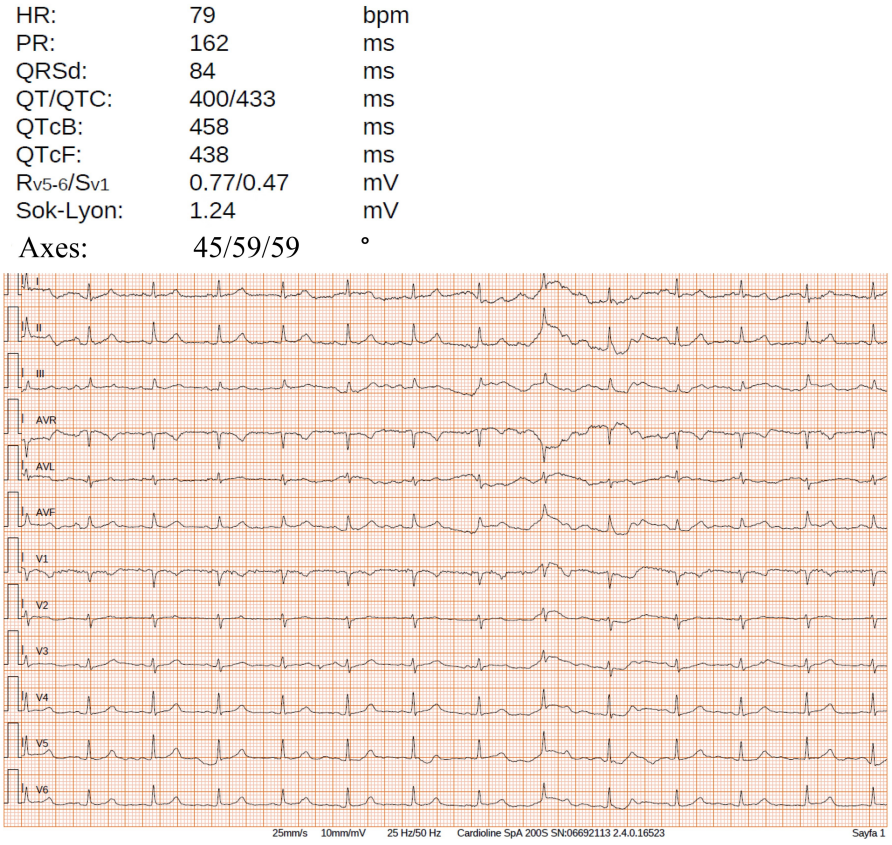

FIGURE 2. Using ECG device's report to measure fQRS TA. HR: Heart Rate; PR: PR Interval; QRSd: QRS Duration; QT: QT Interval; QTC: Corrected QT Interval; QTcB: Interval Corrected by Bazett's Formula; QTcF: Interval Corrected by Fridericia's Formula; AVR: Augmented Vector Right; AVL: Augmented Vector Left; AVF: Augmented Vector Foot.

TABLE 1. QRS-T angle and NIHSS scores according to sociodemographic characteristics of the participants.

Feature	N (%)	fQRS-TA (Q1–Q3)	*p* value	NIHSS (Q1–Q3)	*p*
Gender					
Male	41 (48.2)	47.0 (17–120.5)	0.651*	4 (3–9.5)	0.839*
Female	44 (51.8)	53.5 (29–128.75)		4.5 (2–10.75)	
Hospital					
Etlik City	61 (71.8)	48.0 (18–125)	0.369*	3 (2–7)	0.001*
Bilkent City	24 (28.2)	68.0 (36–126)		7.5 (4.25–11.75)	
HTN					
Yes	54 (63.5)	66.5 (39.75–139.5)	0.010*	4 (2–8.5)	0.967*
No	31 (36.5)	29.0 (18–72)		4 (2–11)	
DM					
Yes	34 (40.0)	55.5 (29–131.25)	0.650*	4 (2–8.5)	0.623*
No	51 (60.0)	53.0 (19–120)		4 (2–10)	
CAD					
Yes	21 (24.7)	64.0 (18.5–1224)	0.684*	6 (3–10.5)	0.191*
No	64 (75.3)	50.5 (24–127)		4 (2–9.75)	
CHF					
Yes	9 (10.6)	138.0 (83–167.5)	0.009*	4 (1.5–6.5)	0.377*
No	76 (89.4)	47.5 (20.25–99.75)		4 (2–10)	

*HTN: High Blood Pressure; DM: Diabetes mellitus; CAD: Coronary artery disease; CHF: Congestive heart failure; fQRS-TA: Frontal QRS-T angle; NIHSS: National Institute of Health Stroke Scale; *: Mann Whitney-U test.*

TABLE 2. QRS-T angle and NIHSS scores of participants according to laboratory findings.

Feature	Mean ± SD	fQRS-TA	*p* value	NIHSS	*p* value
Urea (mg/dL)	47.19 ± 33.36	0.228*	**0.036**	−0.017*	0.880
Creatine(mg/dL)	1.08 ± 0.63	0.088*	0.424	−0.021*	0.846
Sodium (mmol/L)	139.11 ± 4.12	0.036*	0.746	0.194*	0.075
Potassium (mmol/L)	4.33 ± 0.49	0.064*	0.560	0.068*	0.536
ALT (U/L)	19.94 ± 10.72	0.031*	0.776	0.195*	0.074
AST (U/L)	23.87 ± 16.68	0.072*	0.511	0.139*	0.205
Hb (g/dL)	13.11 ± 2.15	−0.122*	0.265	0.082*	0.458
Plt	260.88 ± 97.86	−0.140*	0.203	0.078*	0.475
WBC	9.11 ± 2.74	−0.138*	0.208	0.325*	**0.002**
Neutrophile	6.51 ± 2.78	−0.058*	0.597	0.409*	**<0.001**
Lymphocite	1.74 ± 0.87	−0.121*	0.271	−0.204*	0.061
Troponine (ng/mL)	33.33 ± 90.77	0.271*	**0.012**	0.070*	0.526
CRP (mg/L)	23.13 ± 45.51	0.186*	0.107	0.026*	0.827

*ALT: Alanin aminotransferase; AST: Aspartate transferase; Hb: Hemoglobin; Plt: Platelet; WBC: White blood count; CRP: C-reactive protein; SD: standard deviation; fQRS-TA: Frontal QRS-T angle; NIHSS: National Institute of Health Stroke Scale. *: Spearman correlation coefficient. The results in bold indicate statistically significant findings.*

TABLE 3. Correlation analysis of QRS-T angle and NIHSS correlation coefficient.

	NIHSS score	*p* value
QRS-T angle	0.246*	**0.023**

*NIHSS: National Institute of Health Stroke Scale; *: Spearman correlation coefficient. The results in bold indicate statistically significant findings.*

TABLE 4. Evaluation of fQRS-T angle and NIHSS scores according to mortality.

Mortality	N (%)	fQRS-TA (Q1–Q3)	*p* value	NIHSS (Q1–Q3)	*p* value
(+)	18 (21.2)	92.50 (36.50–154.75)	**0.045***	9 (3.75–14)	**0.014***
(–)	67 (78.8)	47 (19–99)		4 (2–7)	

*fQRS-TA: Frontal QRS-T angle; NIHSS: National Institute of Health Stroke Scale; *: Mann Whitney-U test. The results in bold indicate statistically significant findings.*

to non-ischemic myocardial injury linked to the neurological event itself. In a separate study, Peng *et al.* [22] found that elevated blood urea nitrogen (BUN) levels, even within normal clinical limits, were associated with increased stroke risk and mortality. The onset of AIS initiates a cascade of neurochemical events, including excitotoxicity, oxidative stress, peri-infarct depolarizations, disruption of the blood-brain barrier, microvascular injury, hemostatic alterations, inflammation and apoptosis [23, 24]. These mechanisms may explain the elevations in biomarkers such as troponin and urea observed in our study. Additionally, AIS can affect cardiac metabolism and electrical activity by activating the central and autonomic nervous systems and alterations in neurohormonal pathways [25, 26]. As previously reported, AIS may induce various pathological ECG changes such as QT prolongation, U waves, biphasic T waves and ST segment deviations [8, 26]. Mboi *et al.* [27] emphasized that AIS is not merely a cerebral ischemic event but may also lead to systemic inflammatory responses and cardiac involvement, resulting in electrocardiographic alterations. Autonomic dysfunction following AIS may disrupt the cardiac conduction system and contribute to increases in fQRS-TA. Similar to these findings, our study demonstrated that fQRS-TA, as well as troponin and urea levels, were elevated in AIS patients and that fQRS-TA was positively correlated with these markers.

Fonarow *et al.* [28] identified a nearly linear association between increasing NIHSS scores and 30-day mortality in AIS patients. Consistent with this, our study found that NIHSS scores were significantly higher in the deceased group.

Gunduz *et al.* [29] reported that elevated fQRS-TA predicted in-hospital mortality and the need for mechanical ventilation in COVID-19 patients. Walsh *et al.* [30] showed that increased fQRS-TA was associated with adverse outcomes in coronary artery disease and could be used to predict mortality risk. In a large cohort study, Kors *et al.* [31] demonstrated that higher fQRS-TA was linked to increased 14-year mortality even in patients without established CVD. Similarly, Whang *et al.* [32] reported that increased fQRS-TA could predict adverse cardiovascular events and all-cause mortality. These findings support the notion that elevated fQRS-TA reflects a greater

atherosclerotic burden. Given that AIS is a consequence of cerebral atherosclerosis, it could be reasonable to assume that increased fQRS-TA in AIS patients may reflect underlying vascular pathology. Usalp *et al*. [33] further reported that age-related atherosclerosis and fibrosis impair the cardiac conduction system, leading to increased fQRS-TA, arrhythmias and mortality [33]. In agreement with these studies, our findings suggest that elevated fQRS-TA in AIS patients is associated with higher NIHSS scores and increased short-term mortality.

The strengths of our study include its prospective design, the inclusion of two high-volume stroke centers, and the novel demonstration that fQRS-TA correlates with both NIHSS scores and 30-day mortality in AIS patients. However, several limitations must be acknowledged. First, the sample size was relatively small. Second, patients were not stratified according to treatment modality (*e.g.*, thrombolysis, embolectomy), which may have influenced outcomes. Third, repeated fQRS-TA measurements were not performed. Lastly, due to limited classification of comorbidities, regression analysis for mortality could not be conducted, thereby limiting the ability to establish independent predictive value.

5. Conclusions

This study demonstrated that the fQRS-TA is an easily obtainable and potentially reliable electrocardiographic parameter for predicting short-term mortality in patients presenting to the emergency department with AIS. The integration of fQRS-TA with NIHSS scores may enhance early risk stratification and inform clinical decision-making in AIS management. Nevertheless, further large-scale randomized controlled trials are warranted to validate these findings and to establish the clinical utility of fQRS-TA in routine practice.

AVAILABILITY OF DATA AND MATERIALS

Data used in the study will be made available by the author upon reasonable request.

AUTHOR CONTRIBUTIONS

MG, DÇG, MÇ—conceptualization, methodology; supervision. AEG, MÇ, ZU, MAÖ—data curation; formal analysis and drafting of the manuscript. MG, RY, MÇ—investigation; resources and critical revision of the manuscript. BB, MAÖ, ZU, AEG—data visualization and statistical analysis. MG, BB, RY, HM—literature review; writing—original draft preparation and editing. MG, MAÖ, RY, HM—validation; methodology and review of the final draft. All authors have read and agreed to the published version of the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Prior to the study, approval was obtained from Bilkent City Hospital Clinical Research Ethics Committee No. 2 (approval number: E2-23-4296) and the study was conducted in accordance with the tenets of the Declaration of Helsinki. Informed consent was obtained from all patients who participated in the study.

ACKNOWLEDGMENT

Not applicable.

FUNDING

This research received no external funding.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

REFERENCES

[1] Feske SK. Ischemic stroke. The American Journal of Medicine. 2021; 134: 1457–1464.

[2] Norrving B, Barrick J, Davalos A, Dichgans M, Cordonnier C, Guekht A, *et al*. Action plan for stroke in Europe 2018–2030. European Stroke Journal. 2018; 3: 309–336.

[3] Owolabi MO, Thrift AG, Mahal A, Ishida M, Martins S, Johnson WD, *et al*.; Stroke Experts Collaboration Group. Primary stroke prevention worldwide: translating evidence into action. The Lancet Public Health. 2022; 7: e74–e85.

[4] Wang MF, Hou C, Jia F, Zhong CH, Xue C, Li JJ. Aging-associated atrial fibrillation: a comprehensive review focusing on the potential mechanisms. Aging Cell. 2024; 23: e14309.

[5] Ma S, Li J, Kong Q, Xu Z, Wu H, Jin Y, *et al*. Impact of acute myocardial injury on short- and long-term outcomes in patients with primary intracerebral hemorrhage. Journal of the American Heart Association. 2025; 14: e037053.

[6] Nistor IR, Gherasim L. From neurocardiology to stroke-heart syndrome. Romanian Journal of Internal Medicine. 2023; 61: 177–185.

[7] Scheitz JF, Sposato LA, Schulz-Menger J, Nolte CH, Backs J, Endres M. Stroke-heart syndrome: recent advances and challenges. Journal of the American Heart Association. 2022; 11: e026528.

[8] Powers WJ, Rabinstein AA, Ackerson T, Adeoye OM, Bambakidis NC, Becker K, *et al*. Guidelines for the early management of patients with acute ischemic stroke: 2019 update to the 2018 guidelines for the early management of acute ischemic stroke: a guideline for healthcare professionals from the American heart association/American stroke association. Stroke. 2019; 50: e344–e418.

[9] Oehler A, Feldman T, Henrikson CA, Tereshchenko LG. QRS-T angle: a review. Annals of Noninvasive Electrocardiology. 2014; 19: 534–542.

[10] Adewole KS, Mojeed HA, Ogunmodede JA, Gabralla LA, Faruk N, Abdulkarim A, *et al*. Expert system and decision support system for electrocardiogram interpretation and diagnosis: review, challenges and research directions. Applied Sciences. 2022; 12: 12342.

[11] Singh PK, Akhtar S, Gupta A, Singh S. The clinical relevance of ECG parameters in the prediction of cardiac mortality: a comprehensive review. The Open Bioinformatics Journal. 2024; 17: e18750362295563.

[12] Zhang X, Zhu Q, Zhu L, Jiang H, Xie J, Huang W, *et al*. Spatial/frontal QRS-T angle predicts all-cause mortality and cardiac mortality: a meta-analysis. PLOS ONE. 2015; 10: e0136174.

[13] Raposeiras-Roubín S, Virgós-Lamela A, Bouzas-Cruz N, López-López A, Castiñeira-Busto M, Fernández-Garda R, *et al*. Usefulness of the QRS-T angle to improve long-term risk stratification of patients with acute myocardial infarction and depressed left ventricular ejection fraction. The American Journal of Cardiology. 2014; 113: 1312–1319.

[14] Turen S, Yilmaz E. Predictive value of the frontal QRS-T angle for a permanent pacemaker requirement in patients undergoing transcatheter aortic valve implantation. European Review for Medical and Pharmacological Sciences. 2023; 27: 6238–6246.

[15] Zhang Y, Zhu Y, Jiang T, Liu J, Tang X, Yi W. An in-hospital stroke system to optimize emergency management of acute ischemic stroke by reducing door-to-needle time. American Journal of Emergency Medicine. 2023; 69: 147–153.
[16] Algül E, Özbeyaz NB, Şahan HF, Aydınyılmaz F, Gezer E, Sunman H, *et al*. Frontal QRS-T angle is associated with severity and prognosis of acute pulmonary embolism. Journal of Electrocardiology. 2023; 79: 8–12.
[17] O'Donnell MJ, Xavier D, Liu L, Zhang H, Chin SL, Rao-Melacini P, *et al*; INTERSTROKE investigators. Risk factors for ischaemic and intracerebral haemorrhagic stroke in 22 countries (the INTERSTROKE study): a case-control study. The Lancet. 2010; 376: 112–123.
[18] Selvaraj S, Ilkhanoff L, Burke MA, Freed BH, Lang RM, Martinez EE, *et al*. Association of the frontal QRS-T angle with adverse cardiac remodeling, impaired left and right ventricular function, and worse outcomes in heart failure with preserved ejection fraction. Journal of the American Society of Echocardiography. 2014; 27: 74–82.e2.
[19] Tassone B, Ricchio M, Serapide F, Lionello R, Trecarichi EM, Torti C. Predictivity of frontal QRS-T angle for death in COVID-19 patients may differ by age. American Journal of Emergency Medicine. 2022; 57: 230–233.
[20] Böyük F, Çalışkan S, Demirkıran A. The relationship between the frontal QRS-T angle and high blood pressure response to exercise. Istanbul Medical Journal. 2023; 24: 94–99.
[21] Becker L, Alonso A, Kruska M, Baumann S, Grassl N, Lesch H, *et al*. Acute ischemic stroke and troponin elevation: update of the Mannheim clinical algorithm. Innere Medizin. 2024; 65: 830–839. (In German)
[22] Peng R, Liu K, Li W, Yuan Y, Niu R, Zhou L, *et al*. Blood urea nitrogen, blood urea nitrogen to creatinine ratio and incident stroke: the Dongfeng-Tongji cohort. Atherosclerosis. 2021; 333: 1–8.
[23] Minnerup J, Schmidt A, Albert-Weissenbreger C, Kleinschnitz C. Stroke: pathophysiology and therapy. 1st edn. Morgan & Claypool Life Sciences: USA. 2013.
[24] Brouns R, De Deyn PP. The complexity of neurobiological processes in acute ischemic stroke. Clinical Neurology and Neurosurgery. 2009; 111: 483–495.
[25] Silvani A, Calandra-Buonaura G, Dampney RA, Cortelli P. Brain-heart interactions: physiology and clinical implications. Philosophical Transactions of the Royal Society A. 2016; 374: 20150181.
[26] Kulahcioglu S, Baysal PK, Kup A, Imanov E, Uslu A, Demir S, *et al*. Bidirectional ventricular tachycardia induced by respiratory alkalosis mediated hypokalemia in a patient with acute ischemic heart failure. Pacing and Clinical Electrophysiology. 2021; 44: 2115–2118.
[27] Mboi N, Murty Surbakti I, Trihandini I, Elyazar I, Houston Smith K, Bahjuri Ali P, *et al*. On the road to universal health care in Indonesia, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. The Lancet. 2018; 392: 581–591.
[28] Fonarow GC, Saver JL, Smith EE, Broderick JP, Kleindorfer DO, Sacco RL, *et al*. Relationship of national institutes of health stroke scale to 30-day mortality in medicare beneficiaries with acute ischemic stroke. Journal of the American Heart Association. 2012; 1: 42–50.
[29] Gunduz R, Yildiz BS, Ozgur S, Ozen MB, Bakir EO, Ozdemir IH, *et al*. Frontal QRS/T angle can predict mortality in COVID-19 patients. American Journal of Emergency Medicine. 2022; 58: 66–72.
[30] Walsh JA III, Soliman EZ, Ilkhanoff L, Ning H, Liu K, Nazarian S, *et al*. Prognostic value of frontal QRS-T angle in patients without clinical evidence of cardiovascular disease (from the Multi-Ethnic Study of Atherosclerosis). The American Journal of Cardiology. 2013; 112: 1880–1884.
[31] Kors JA, Kardys I, van der Meer IM, van Herpen G, Hofman A, van der Kuip DA, *et al*. Spatial QRS-T angle as a risk indicator of cardiac death in an elderly population. Journal of Electrocardiology. 2003; 36: 113–114.
[32] Whang W, Shimbo D, Levitan EB, Newman JD, Rautaharju PM, Davidson KW, *et al*. Relations between QRS|T angle, cardiac risk factors, and mortality in the third National Health and Nutrition Examination Survey (NHANES III). The American Journal of Cardiology. 2012; 109: 981–987.
[33] Usalp S, Bağırtan B. A novel electrocardiographic marker for predicting total mortality in ischemic stroke: frontal QRS-T angle. Archives of the Turkish Society of Cardiology. 2025; 53: 29–34.

How to cite this article: Murat Genç, Didem Çankaya Gökdere, Adil Emre Gezer, Melih Çamcı, Bensu Bulut, Medine Akkan Öz, *et al*. Correlation of frontal QRS-T angle with mortality in ischemic stroke patients: a multicenter study. Signa Vitae. 2025; 21(12): 107-113. doi: 10.22514/sv.2025.194.