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Evaluation of Fragmented QRS in General Surgical ICU Patients: No Association with Short-and Long-Term Mortality

Genel Cerrahi Yoğun Bakım Hastalarında Fragmented QRS Değerlendirmesi: Kısa ve Uzun Dönem Mortalite ile İlişkisi Yoktur

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Evaluation of Fragmented QRS in General Surgical ICU Patients: No Association with Short-and Long-Term Mortality

ABSTRACT

Background: Fragmented QRS (fQRS) on electrocardiography has been proposed as a marker of myocardial conduction heterogeneity and an independent predictor of mortality in various cardiac and medical intensive care populations. However, its prognostic value in general surgical intensive care unit (ICU) patients remains uncertain.

Methods: This retrospective, single-center observational study included 184 patients admitted to the general surgery ICU between January 2021 and December 2024. Demographic, clinical, and laboratory data were collected at ICU admission. fQRS was defined as the presence of additional R′ waves, notching, or fragmentation in at least two contiguous leads. The primary outcome was 28-day mortality; in-hospital mortality served as a secondary endpoint. Associations were analyzed using t-tests, chi-square tests, and Pearson correlation coefficients.

Results: The mean age of the cohort was 67 ± 15 years, and 55.4% were male. The 28-day and in-hospital mortality rates were 32.1% and 11.4%, respectively. Non-survivors had significantly higher MFI-5 frailty scores (4.5 ± 0.7 vs. 1.4 ± 1.0; *p<0.001*), longer ICU stays (6.56 ± 6.06 vs. 4.25 ± 4.20 days; *p=0.003*), and higher APACHE II scores (51.5 ± 23.5 vs. 38.1 ± 19.3; *p<0.001*). Elevated BUN, creatinine, and lactate, and lower albumin and pH were all associated with mortality (*p<0.001* for each). In contrast, fQRS showed no significant association with either 28-day (*p=0.929*) or in-hospital mortality (*p=0.900*).

Conclusions: In surgical ICU patients, fQRS lacked prognostic significance. Mortality was instead driven by systemic metabolic and physiologic derangements. These findings highlight the context-dependent value of fQRS and underscore the need for integrative, multivariable risk models rather than reliance on isolated ECG markers.

Keywords: Critical care, Electrocardiography, Fragmented QRS, Mortality, Surgical intensive care unit.

Genel Cerrahi Yoğun Bakım Hastalarında Fragmented QRS Değerlendirmesi: Kısa ve Uzun Dönem Mortalite ile İlişki Saptanma

ÖZ

Arka plan: Elektrokardiyografide fragmented QRS (fQRS), miyokardiyal ileti heterojenliğinin bir göstergesi olarak önerilmiş olup çeşitli kardiyak ve tıbbi yoğun bakım popülasyonlarında mortalitenin bağımsız bir belirteci olarak bildirilmiştir. Ancak, genel cerrahi yoğun bakım ünitesi (YBÜ) hastalarında prognostik değeri belirsizliğini korumaktadır.

Yöntemler: Bu retrospektif, tek merkezli gözlemsel çalışmaya Ocak 2021–Aralık 2024 tarihleri arasında genel cerrahi YBÜ'ye kabul edilen 184 hasta dahil edildi. Yoğun bakım kabulündeki demografik, klinik ve laboratuvar verileri kaydedildi. fQRS, en az iki komşu derivasyonda ek R′ dalgası, çentiklenme veya fragmentasyon varlığı olarak tanımlandı. Birincil sonlanım noktası 28 günlük mortalite, ikincil sonlanım noktası ise hastane içi mortaliteydi. İlişkiler t-testi, ki-kare testi ve Pearson korelasyon analizi ile değerlendirildi.

Bulgular: Kohortun ortalama yaşı 67 ± 15 yıl olup, %55,4'ü erkekti. Yirmi sekiz günlük mortalite oranı %32,1, hastane içi mortalite oranı ise %11,4 olarak bulundu. Kaybedilen hastalarda MFI-5 kırılganlık skorları anlamlı derecede yüksek (4,5 ± 0,7 vs. 1,4 ± 1,0; *p<0.001*), YBÜ kalış süreleri daha uzun (6,56 ± 6,06 vs. 4,25 ± 4,20 gün; *p=0.003*) ve APACHE II skorları daha yüksek bulundu (51,5 ± 23,5 vs. 38,1 ± 19,3; *p<0.001*). Artmış BUN, kreatinin ve laktat; düşmüş albümin ve pH değerleri mortalite ile anlamlı ilişki gösterdi (her biri için *p<0.001*). Buna karşılık fQRS ne 28 günlük mortalite (*p=0.929*) ne de hastane içi mortalite (*p=0.900*) ile ilişkiliydi.

Sonuçlar: Cerrahi YBÜ hastalarında fQRS'nin prognostik bir değeri bulunmamıştır. Mortalite daha çok sistemik metabolik ve fizyolojik bozulmalar tarafından belirlenmiştir. Bulgular, fQRS'nin bağlam-bağımlı değerini vurgulamakta ve izole EKG belirteçlerine dayanmak yerine çok değişkenli, bütüncül risk modellerine ihtiyaç olduğunu göstermektedir.

Anahtar Kelimeler: Cerrahi yoğun bakım ünitesi, Elektrokardiyografi, Fragmented QRS, Mortalite, Yoğun bakım.

Introduction

Fragmented QRS (fQRS) is an electrocardiographic marker of ventricular conduction heterogeneity, typically reflecting underlying myocardial scarring, fibrosis, or patchy ischemia (1). In cardiology and medical ICU populations, fQRS has repeatedly been linked to increased mortality, arrhythmic events, and adverse cardiovascular outcomes across conditions such as acute coronary syndrome, heart failure, and sepsis (2,3). Several meta-analyses have shown a two- to threefold increase in short-term mortality in patients with fQRS on admission ECG (2).

However, the prognostic value of fQRS in surgical intensive care units remains largely unexplored. Surgical ICU patients represent a unique population: postoperative physiology is characterized by major hemodynamic shifts, systemic inflammation, ischemia-reperfusion injury, electrolyte and acid-base disturbances, and catabolic stress responses that can profoundly affect both myocardial conduction and survival trajectories (4,5) Unlike cardiac ICU cohorts, in which death is often related to primary myocardial pathology, mortality in surgical ICUs is frequently driven by global physiologic failure, metabolic derangement, and frailty rather than localized myocardial injury (6).

Importantly, fQRS in non-cardiac critically ill patients may be transient, reflecting reversible perioperative factors such as hypoxia, electrolyte imbalance, or anesthetic effects rather than fixed myocardial scar (7,8). Prior fQRS literature has largely focused on stable cardiac populations and has not adequately controlled for confounding factors such as acidosis, hypoalbuminemia, or lactate elevation, all of which are powerful prognosticators in surgical critical illness (9,10).

These pathophysiologic and methodological gaps raise a crucial question: Does fQRS retain its prognostic value in a high-stress, metabolically unstable surgical ICU environment?

To address this gap, we evaluated the association between admission fQRS and 28-day and in-hospital mortality in a heterogeneous cohort of general surgical ICU patients. We specifically hypothesized that the predictive value of fQRS would be attenuated in this context, as systemic metabolic and physiologic determinants of outcome are expected to outweigh localized conduction abnormalities. Furthermore, we sought to contextualize fQRS against established ICU prognostic markers such as frailty, renal indices, acid-base balance, and lactate, thereby bridging cardiologic and perioperative critical care frameworks.

Material and Method

Study Design and Ethical Approval

This was a retrospective, single-center observational cohort study conducted in the 10-bed General Surgery Intensive Care Unit (ICU) of Mehmet Akif Ersoy State Hospital, a tertiary referral center providing comprehensive perioperative and postoperative care for critically ill surgical patients. The study was carried out between January 2021 and December 2024.

The study protocol was reviewed and approved by the Çanakkale Onsekiz Mart University Non-Interventional Clinical Research Ethics Committee (approval number: 2025-126, meeting date: April 17, 2025). The requirement for informed consent was waived due to the retrospective design and use of anonymized patient data. The study was conducted in accordance with the Declaration of Helsinki (2013 revision) and the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines.

Study Population

All adult patients (≥18 years of age) admitted to the general surgery ICU during the study period were screened for eligibility.

Inclusion criteria were as follows:

1. Availability of a standard 12-lead electrocardiogram (ECG) recorded within the first 6 hours after ICU admission,

2. Complete physiologic and biochemical data enabling calculation of the APACHE II score, and

3. Documentation of both short-term (28-day) and in-hospital outcomes.

Exclusion criteria included:

(a) ICU stay shorter than 24 hours,

(b) Missing or poor-quality ECG recordings precluding fQRS evaluation,

(c) Documented pacemaker rhythm or bundle branch block,

(d) Known pre-existing congenital conduction abnormalities, and

(e) Incomplete clinical or laboratory data.

After exclusions, 184 patients met the study criteria and were included in the final analysis (Figure I).

Data Collection and Definitions

Clinical data were retrieved from the hospital's electronic medical records (ENLİS® system). Demographic variables (age, sex, height, weight, BMI), comorbidities, frailty score, mechanical ventilation status, vasopressor use, hypotension, and diagnoses (myocardial infarction, pneumonia, acute kidney injury) were recorded. Laboratory parameters included blood urea nitrogen (BUN), creatinine, albumin, calcium, magnesium, pH, lactate, complete blood count, and coagulation indices (PT, APTT, INR).

Frailty was assessed using the Modified Frailty Index-5 (MFI-5), which includes five pre-existing domains: functional dependence, diabetes mellitus, chronic obstructive pulmonary disease, congestive heart failure, and hypertension. Each component was coded as present (1) or absent (0), yielding a total score ranging from 0 to 5. Frailty was analyzed as a continuous variable without applying a predefined cut-off. All MFI-5 variables were derived from baseline comorbidities and functional status documented at ICU admission and reflected chronic health status rather than acute postoperative or illness-related impairment. Data extraction was performed retrospectively by two independent investigators using standardized electronic medical records, with discrepancies resolved by consensus. Illness severity was quantified by the APACHE II score calculated on the first ICU day.

Outcomes:

The primary outcome was 28-day mortality after ICU admission. The secondary outcome was total in-hospital mortality during the same hospitalization episode.

Electrocardiographic Assessment

All patients underwent a standard 12-lead ECG at a paper speed of 25 mm/s and amplitude of 10 mm/mV upon ICU admission.

The presence of fragmented QRS (fQRS) was defined according to the criteria of Das et al. (11) as the presence of additional R′ waves, notching in the nadir of the S wave, or multiple R′ components in at least two contiguous leads corresponding to a major coronary artery territory.

The ECGs were independently reviewed by two experienced intensivists blinded to patient outcomes. In case of disagreement, a third senior intensivist adjudicated the final classification. The intra-observer and inter-observer agreement were tested in a subset of 30 random ECGs, yielding kappa values of 0.91 and 0.88, respectively, indicating excellent reliability.

Fragmented QRS was analyzed as a dichotomous variable (present/absent) rather than being stratified by lead distribution, number of affected leads, QRS width, or associated Q waves. This approach was chosen because the primary aim was to evaluate the clinical screening value of fQRS in a heterogeneous surgical ICU population, where ECG patterns are frequently influenced by transient metabolic and perioperative factors. In addition, the relatively small number of fQRS-positive patients limited meaningful subgroup analyses based on morphological or regional characteristics.

Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics for Windows, version 29.0 (IBM Corp., Armonk, NY, USA). Continuous variables were tested for normality using the Kolmogorov–Smirnov test and presented as mean ± standard deviation (SD) or median (interquartile range, IQR) where appropriate. Categorical variables were expressed as frequencies and percentages.

Comparisons between survivors and non-survivors were made using the independent-sample t-test or Mann–Whitney U test for

continuous variables, and the chi-square test or Fisher's exact test for categorical variables.

Correlation analyses between mortality and continuous variables were performed using Pearson correlation coefficients (r). A two-tailed *p-value < 0.05* was considered statistically significant.

To explore potential multicollinearity among mortality predictors, inter-variable relationships between APACHE II, lactate, albumin, and frailty were also examined. Missing data were minimal (<5%) and handled via pairwise deletion.

Although multivariable regression would be the preferred approach to evaluate independent predictors of mortality, formal multivariate modeling was limited in this study by the relatively small number of outcome events—particularly for in-hospital mortality—and by substantial multicollinearity among major prognostic variables (MFI-5, APACHE II, lactate, albumin, pH, BUN, and creatinine). Exploratory models demonstrated unstable coefficient estimates and variance inflation beyond acceptable thresholds. Therefore, analyses were restricted to univariate and correlation-based methods to avoid overfitting and misleading inferences.

Sample Size and Statistical Power

Based on previous literature indicating an expected 30% mortality rate among surgical ICU patients and assuming an effect size of 0.4 for the potential association between fQRS and mortality (α = 0.05, power = 0.80), the minimum required sample size was estimated as n = 156 using G*Power version 3.1.9.7. This effect size (0.4) was chosen based on previously reported mortality associations for fragmented QRS in critically ill populations, where moderate-to-large effect sizes have been observed (9,10). The final sample of 184 patients provided adequate statistical power for univariate and correlation analyses.

Data Presentation

All analyses were summarized in three tables and one figure:

- Table I: Baseline demographic, clinical, and laboratory characteristics according to 28-day mortality.
- Table II: Baseline characteristics according to total in-hospital mortality.
- Table III: Correlation coefficients (r) of clinical and laboratory parameters with mortality endpoints.
- Figure I: Forest plot displaying Pearson correlations between predictors and mortality outcomes.

Table I. Baseline Demographic, Clinical, and Laboratory Characteristics According to 28-day Mortality

Variable	Survivors (n=125)	Non-survivors (n=59)	*p value*
Age (years)	65.9 ± 15.3	70.5 ± 14.5	*0.060*
Male sex, n (%)	72 (57.6)	30 (50.8)	*0.401*
BMI (kg/m^2)	28.7 ± 8.5	26.7 ± 5.8	*0.097*
ICU stay (days)	4.25 ± 4.20	6.56 ± 6.06	*0.003*
MFI-5 score	1.4 ± 1.0	4.5 ± 0.7	*<0.001*
Mechanical ventilation, n (%)	104 (63.8)	59 (36.2)	*0.001*
Vasopressor use, n (%)	14 (19.7)	57 (80.3)	*<0.001*
Hypotension, n (%)	24 (39.3)	37 (60.7)	*<0.001*
Myocardial infarction, n (%)	9 (18.4)	40 (81.6)	*<0.001*
Acute kidney injury, n (%)	20 (43.5)	26 (56.5)	*<0.001*
Pneumonia, n (%)	16 (51.6)	15 (48.4)	*0.033*
Glucose (mg/dL)	154.1 ± 68.6	160.2 ± 92.5	*0.621*
BUN (mg/dL)	52.6 ± 37.5	79.9 ± 47.0	*<0.001*
Creatinine (mg/dL)	1.03 ± 0.51	1.50 ± 0.86	*<0.001*
Albumin (g/dL)	2.94 ± 0.64	2.50 ± 0.55	*<0.001*
Calcium (mg/dL)	8.23 ± 1.09	7.78 ± 1.05	*0.010*
Magnesium (mg/dL)	1.88 ± 0.59	1.83 ± 0.60	*0.646*
CK (U/L)	366.9 ± 671.1	609.9 ± 1139.4	*0.072*
WBC (10^3/µL)	13.4 ± 13.3	13.9 ± 11.7	*0.816*
Hemoglobin (g/dL)	11.2 ± 2.1	10.9 ± 2.1	*0.351*
Platelet (10^3/µL)	257.2 ± 123.8	233.4 ± 142.9	*0.248*
INR	1.32 ± 0.66	4.09 ± 19.9	*0.122*
APTT (s)	30.9 ± 8.8	37.0 ± 15.2	*0.001*
PT (s)	14.5 ± 6.5	16.2 ± 6.6	*0.108*
pH	7.38 ± 0.09	7.30 ± 0.16	*<0.001*
Lactate (mmol/L)	2.0 ± 1.9	3.7 ± 3.3	*<0.001*
APACHE II	38.1 ± 19.3	51.5 ± 23.5	*<0.001*
QT prolongation, n (%)	13 (86.7)	2 (13.3)	*0.105*
QTc prolongation, n (%)	36 (73.5)	13 (26.5)	*0.333*
Fragmented QRS, n (%)	24 (68.6)	11 (31.4)	*0.929*
HR (bpm)	95.4 ± 22.5	98.5 ± 26.4	*0.408*
PR (ms)	140.4 ± 42.7	135.0 ± 32.1	*0.392*
QRS (ms)	98.2 ± 48.9	90.5 ± 16.2	*0.241*
QT (ms)	364.5 ± 59.3	357.9 ± 51.3	*0.465*
QTc (ms)	422.7 ± 48.2	421.7 ± 43.6	*0.892*

Data are presented as mean ± standard deviation or n (%). p values calculated by independent t-test for continuous variables and χ^2/Fisher's exact test for categorical variables.

BMI = Body Mass Index; ICU = Intensive Care Unit; MFI-5 = Modified Frailty Index-5; BUN = Blood Urea Nitrogen; CK = Creatine Kinase; WBC = White Blood Cell; INR = International Normalized Ratio; APTT = Activated Partial Thromboplastin Time; PT = Prothrombin Time; QTc = Corrected QT Interval; HR = Heart Rate.

Table II. Baseline Demographic, Clinical, and Laboratory Characteristics According to Total In-hospital Mortality

Variable	Survivors (n=163)	Non-survivors (n=21)	p value
Age (years)	66.7 ± 15.1	72.9 ± 14.4	*0.050*
Male sex, n (%)	95 (58.3)	12 (57.1)	*0.905*
Height (cm)	169.2 ± 11.0	168.5 ± 11.1	*0.801*
Weight (kg)	79.4 ± 21.1	76.2 ± 18.0	*0.422*
BMI (kg/m^2)	28.1 ± 7.7	27.1 ± 7.0	*0.591*
ICU stay (days)	4.8 ± 5.1	7.9 ± 6.5	*0.010*
MFI-5 score	2.2 ± 1.8	4.6 ± 0.7	*<0.001*
Mechanical ventilation, n (%)	112 (68.7)	51 (31.3)	*0.003*
Vasopressor use, n (%)	23 (32.4)	48 (67.6)	*<0.001*
Hypotension, n (%)	23 (37.7)	38 (62.3)	*<0.001*
Myocardial infarction, n (%)	7 (14.3)	42 (85.7)	*<0.001*
Acute kidney injury, n (%)	20 (43.5)	26 (56.5)	*<0.001*
Pneumonia, n (%)	18 (58.1)	13 (41.9)	*0.052*
Glucose (mg/dL)	155.8 ± 77.0	160.3 ± 85.0	*0.731*
BUN (mg/dL)	56.3 ± 41.5	90.2 ± 52.0	*<0.001*
Creatinine (mg/dL)	1.12 ± 0.60	1.72 ± 0.90	*<0.001*
Albumin (g/dL)	2.84 ± 0.64	2.30 ± 0.50	*<0.001*
Calcium (mg/dL)	8.12 ± 1.09	7.61 ± 0.99	*0.010*
Magnesium (mg/dL)	1.86 ± 0.59	1.82 ± 0.62	*0.741*
CK (U/L)	432.0 ± 854.1	693.5 ± 1099.2	*0.157*
WBC (10^3/μL)	13.5 ± 12.9	14.0 ± 11.9	*0.834*
Hemoglobin (g/dL)	11.1 ± 2.1	10.8 ± 2.1	*0.529*
Platelet (10^3/μL)	252.7 ± 131.8	229.3 ± 134.1	*0.357*
INR	1.58 ± 6.8	2.47 ± 5.7	*0.351*
APTT (s)	32.2 ± 11.6	40.5 ± 21.8	*0.008*
PT (s)	14.9 ± 6.5	18.6 ± 7.2	*0.003*
pH	7.36 ± 0.12	7.27 ± 0.16	*<0.001*
Lactate (mmol/L)	2.4 ± 2.5	4.5 ± 3.8	*<0.001*
APACHE II	41.5 ± 21.4	56.0 ± 25.2	*<0.001*
QT prolongation, n (%)	15 (100)	0 (0.0)	*0.012*
QTc prolongation, n (%)	42 (85.7)	7 (14.3)	*0.014*
Fragmented QRS, n (%)	25 (71.4)	10 (28.6)	*0.900*
HR (bpm)	96.4 ± 23.8	95.0 ± 24.2	*0.833*
PR (ms)	138.7 ± 41.3	136.5 ± 30.2	*0.783*
QRS (ms)	96.0 ± 43.2	91.3 ± 16.5	*0.583*
QT (ms)	361.8 ± 56.6	357.4 ± 53.4	*0.711*
QTc (ms)	422.6 ± 46.7	420.8 ± 41.2	*0.867*

Data are presented as mean ± standard deviation or n (%). p values calculated by independent t-test for continuous variables and χ^2/Fisher's exact test for categorical variables.

BMI = Body Mass Index; ICU = Intensive Care Unit; MFI = Modified Frailty Index; BUN = Blood Urea Nitrogen; CK = Creatine Kinase; WBC = White Blood Cell; INR = International Normalized Ratio; APTT = Activated Partial Thromboplastin Time; PT = Prothrombin Time; QTc = Corrected QT Interval; HR = Heart Rate.

Table III. Significant Correlations of Clinical and Laboratory Variables with Mortality

Variable	In-hospital mortality r (p)	28-day mortality r (p)
Age	*0.353 (<0.001)*	*0.139 (0.060)*
Weight (kg)	*-0.126 (0.087)*	*-0.165 (0.025)*
ICU stay (days)	*0.204 (0.005)*	*0.217 (0.003)*
Total MFI-5 score	*0.816 (<0.001)*	*0.847 (<0.001)*
BUN (admission)	*0.257 (<0.001)*	*0.301 (<0.001)*
Creatinine (admission)	*0.290 (<0.001)*	*0.329 (<0.001)*
Albumin	*-0.309 (<0.001)*	*-0.322 (<0.001)*
Calcium	*-0.104 (0.159)*	*-0.190 (0.010)*
Creatine kinase (CK)	*0.173 (0.019)*	*0.133 (0.072)*
Hemoglobin (g/dL)	*-0.173 (0.019)*	*-0.069 (0.351)*
APTT (s)	*0.183 (0.013)*	*0.246 (0.001)*
pH	*-0.199 (0.007)*	*-0.295 (<0.001)*
Lactate (mmol/L)	*0.212 (0.004)*	*0.321 (<0.001)*
APACHE II	*0.245 (0.001)*	*0.291 (<0.001)*

Legend: Only statistically significant correlations (*p < 0.05*) are shown. Pearson correlation coefficients (r) with two-tailed significance (p-value). Positive r indicates direct correlation; negative r indicates inverse correlation.

MFI-5 = Modified Frailty Index-5; BUN = Blood Urea Nitrogen; CK = Creatine Kinase; APTT = Activated Partial Thromboplastin Time; APACHE II = Acute Physiology and Chronic Health Evaluation II.

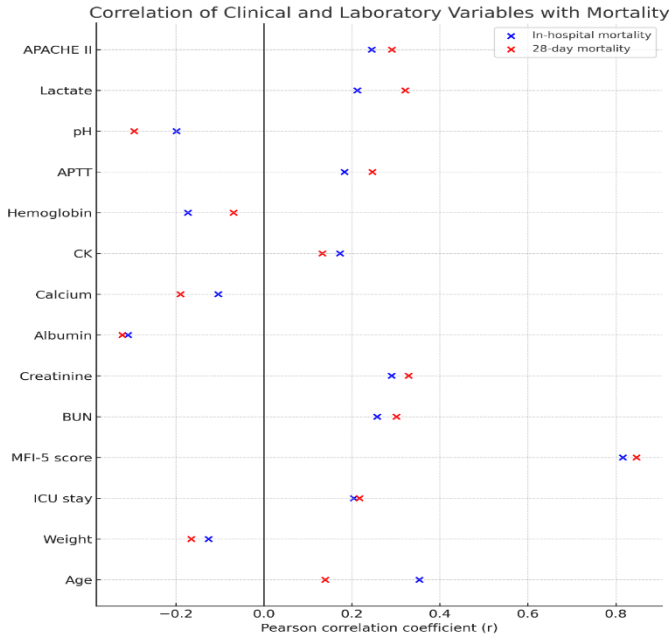

Figure I. Forest plot showing Pearson correlation coefficients (r) between clinical and laboratory variables and mortality outcomes. Blue markers indicate correlations with in-hospital mortality, and red markers indicate correlations with 28-day mortality. The vertical line at r=0 represents no correlation.

MFI = Modified Frailty Index; BUN = Blood urea nitrogen; CK = Creatine kinase; APTT = Activated partial thromboplastin time; APACHE II = Acute Physiology and Chronic Health Evaluation II.

Results

Baseline Characteristics

A total of 184 patients admitted to the general surgery intensive care unit between January 2021 and December 2024 were included in the final analysis. The mean age was 67 ± 15 years, and 55.4% of patients were male. Overall, 59 patients (32.1%) died within 28 days of ICU admission, and 21 patients (11.4%) died during their overall hospital stay.

When patients were stratified according to 28-day mortality, non-survivors were slightly older than survivors (70.5 ± 14.5 vs. 65.9 ± 15.3 years; *p=0.060*) and had significantly longer ICU stays (6.56 ± 6.06 days vs. 4.25 ± 4.20 days; *p=0.003*). Frailty burden was markedly higher among non-survivors, with mean MFI-5 scores of 4.5 ± 0.7 compared to 1.4 ± 1.0 in survivors (*p<0.001*). The need for mechanical ventilation (*p=0.001*), vasopressor use (*p<0.001*), and the presence of hypotension, myocardial infarction, and acute kidney injury (all *p<0.001*) were substantially more common in the non-survivor group.

In contrast, body mass index, sex distribution, and most hematologic variables did not differ significantly between groups. The incidence of pneumonia was higher among non-survivors (p = 0.033), but white blood cell count and C-reactive indices showed no significant difference.

Laboratory comparisons revealed marked metabolic and biochemical disparities between survivors and non-survivors. Patients who died had significantly higher BUN (79.9 ± 47.0 vs. 52.6 ± 37.5 mg/dL, *p<0.001*), creatinine (1.50 ± 0.86 vs. 1.03 ± 0.51 mg/dL, *p<0.001*), and lactate levels (3.7 ± 3.3 vs. 2.0 ± 1.9 mmol/L, *p<0.001*). Conversely, non-survivors had lower albumin (2.50 ± 0.55 vs. 2.94 ± 0.64 g/dL, *p<0.001*), calcium (7.78 ± 1.05 vs. 8.23 ± 1.09 mg/dL, *p=0.010*), and arterial pH (7.30 ± 0.16 vs. 7.38 ± 0.09, *p<0.001*). Coagulation markers demonstrated longer APTT values among non-survivors (37.0 ± 15.2 vs. 30.9 ± 8.8 s, *p=0.001*), while PT and INR differences were not statistically significant.

The APACHE II score was markedly higher in patients who died within 28 days (51.5 ± 23.5 vs. 38.1 ± 19.3, *p<0.001*). Similar patterns were observed when mortality was analyzed by overall hospital outcome (Table II).

Electrocardiographic Findings

Admission ECG parameters—including heart rate, PR interval, QRS duration, QT, and corrected QT interval—did not differ significantly between survivors and non-survivors.

The prevalence of fragmented QRS was comparable across both mortality strata (28-day mortality: 31.4 % vs 68.6 %, *p=0.929*; in-hospital mortality: 28.6 % vs 71.4 %, *p=0.900*). Likewise, QT and QTc prolongations failed to demonstrate prognostic significance in 28-day mortality analysis, though mild differences emerged in the smaller in-hospital mortality subset (QT prolongation *p=0.012*; QTc prolongation *p=0.014*), likely due to sample size imbalance.

In-Hospital Mortality Comparisons

When total in-hospital deaths were evaluated separately (n = 21), non-survivors exhibited similar trends of older age, longer ICU stay (7.9 ± 6.5 vs. 4.8 ± 5.1 days, *p=0.010*), and higher frailty (MFI-5 = 4.6 ± 0.7 vs 2.2 ± 1.8, *p<0.001*). Consistent with the 28-day results, non-survivors displayed greater renal dysfunction (BUN = 90.2 ± 52.0 mg/dL; Creatinine = 1.72 ± 0.90 mg/dL; both *p<0.001*), elevated lactate (4.5 ± 3.8 mmol/L; *p<0.001*), lower albumin (2.30 ± 0.50 g/dL; *p<0.001*), and higher APACHE II scores (56.0 ± 25.2 vs 41.5 ± 21.4; *p<0.001*). Markers of systemic hypoperfusion—acidosis (pH = 7.27 ± 0.16 vs 7.36 ± 0.12; *p<0.001*) and prolonged coagulation times (PT *p=0.003*; aPTT *p=0.008*)—were significantly associated with death.

Correlation Analysis

Pearson correlation coefficients (Table III, Figure I) highlighted strong positive associations between both mortality endpoints and the MFI-5 frailty score (*r=0.847* for 28-day, *r=0.816* for in-hospital; *p<0.001*), renal indices [BUN (*r=0.30–0.26, p<0.001*), creatinine (*r=0.33–0.29, p<0.001*), and APACHE II (*r≈0.29, p<0.001*)]. Inverse

relationships were observed for albumin (*r≈–0.32, p<0.001*) and pH (*r≈–0.30, p<0.001*). Of note, none of the ECG variables—including QRS duration, QTc interval, or fQRS presence—showed statistically significant correlation with mortality (*all p>0.05*).

Summary of Key Findings

1. Mortality (28-day = 32.1 %, in-hospital = 11.4 %) was mainly driven by physiologic frailty and metabolic-renal dysfunction.

2. fQRS showed no prognostic value, contrasting with prior reports from medical or cardiac ICUs.

3. Strongest predictors of death were higher MFI-5, APACHE II, BUN, creatinine, and lactate, together with lower albumin and pH.

4. No sex-related or BMI-related survival differences were observed.

Collectively, these results suggest that short- and long-term mortality in surgical ICU patients are dominated by systemic factors—frailty, multiorgan failure, and metabolic acidosis—rather than by electrophysiologic abnormalities detected on standard ECG.

Discussion

In contrast to prior reports in cardiac and medical ICU settings, we found no prognostic association between fragmented QRS and mortality in surgical ICU patients (11–13). This supports the concept that fQRS is not a universal mortality marker but a context-dependent phenomenon whose predictive value may be attenuated when non-cardiac mechanisms dominate patient outcomes. In our cohort, mortality was instead driven by frailty (MFI-5), renal dysfunction (BUN, creatinine), systemic metabolic derangement (pH, lactate), nutritional status (albumin), and overall illness severity (APACHE II), indicating that global physiologic reserve and multiorgan failure outweigh localized myocardial conduction abnormalities in surgical ICU patients (4,14).

Although fQRS has been linked to adverse outcomes in cardiomyopathy, ischemic heart disease, and sepsis (9,11,15), its lack of significance in our surgical ICU cohort is consistent with evidence that its prognostic value diminishes when systemic rather than cardiac mechanisms determine outcome (16,17). Indeed, while studies in COVID-19, sepsis, and acute myocardial infarction populations have reported associations between fQRS and mortality (9,15,18–21), these conditions are characterized by primary myocardial or microvascular injury, which differs fundamentally from the heterogeneous, metabolically driven physiology of postoperative and surgical critical illness.

Consistent with this interpretation, prior investigations have shown that the prognostic effect of fQRS often weakens after adjustment for cardiac functional parameters. Allescher et al. (16) observed no mortality difference after myocardial infarction, while Umapathy et al. (22) and Ma et al. (23) demonstrated that fQRS primarily reflects impaired ventricular function rather than acting as an independent determinant of survival. Our findings align with these studies, suggesting that in surgical ICU patients, fQRS may represent a surrogate of transient myocardial stress rather than a stable marker of fatal risk.

From a mechanistic perspective, fQRS reflects inhomogeneous ventricular depolarization related to myocardial scar, ischemia, or fibrosis (24,25), and in medical ICU settings such patterns may arise from inflammatory or septic myocardial injury (26). In contrast, surgical ICU mortality is more closely related to systemic hypoxia, metabolic acidosis, renal failure, and frailty (14,17). The strong associations observed in our cohort between mortality and albumin, lactate, and pH reinforce the dominant role of metabolic and nutritional failure over electrophysiologic abnormalities. Moreover, conduction disturbances in this setting are often transient and influenced by perioperative stress and electrolyte imbalance, making a single ECG less reflective of underlying myocardial vulnerability (27).

Frailty emerged as the strongest predictor of mortality in our study, supporting the concept that reduced physiologic reserve may be more critical than cardiac electrical markers in determining outcome. This observation is in line

with evidence that frailty outperforms cardiac biomarkers for mortality prediction in older ICU populations (28).

By focusing on a heterogeneous surgical ICU cohort rather than a disease-specific population, our study extends the fQRS literature into an underexplored clinical domain. The consistency of null findings across both 28-day and in-hospital mortality, together with a comprehensive assessment of metabolic and physiologic determinants, indicates that fQRS lacks prognostic relevance in this context. These results also provide an important counterbalance to predominantly positive reports, helping to mitigate publication bias and to refine the clinical boundaries within which fQRS may—or may not—be useful as a prognostic marker.

Importantly, this study represents a rigorously conducted negative investigation. In critical care research, selective publication of positive biomarker studies contributes to substantial publication bias, often leading to premature clinical adoption of markers that fail to generalize across populations. By demonstrating that fragmented QRS lacks prognostic value in a heterogeneous surgical ICU cohort, our findings provide an important corrective to the predominantly cardiology- and infection-focused fQRS literature and underscore the necessity of context-specific validation before clinical implementation.

Limitations

This study has several limitations. First, its retrospective and single-center design limits generalizability and precludes causal inference. The absence of a fully adjusted multivariable model represents a limitation of this study. However, this was primarily driven by limited event numbers and strong collinearity among key physiologic and metabolic predictors, which rendered regression models unstable and highly sensitive to variable selection. Under these conditions, univariate and correlation-based analyses provide more robust and transparent inference. Second, fQRS was assessed from a single baseline ECG, without evaluating its lead distribution, dynamic resolution, or temporal persistence—factors known to influence prognostic power. Third, echocardiographic and troponin data were unavailable in all patients, preventing integration of electrophysiologic and myocardial structural assessments. This limitation is particularly important because the prognostic interpretation of fragmented QRS depends heavily on the presence of underlying structural heart disease or myocardial injury, which could not be systematically assessed in this cohort. Fourth, sample size restricted multivariate adjustment, particularly for rare ECG findings such as prolonged QT. Finally, postoperative variables—surgical type, anesthesia duration, and fluid balance—were not systematically analyzed and could affect both conduction patterns and outcomes.

Despite these constraints, the uniformity of results across endpoints and the comprehensive biochemical dataset lend robustness to our findings.

Clinical and research implications

Our findings indicate that fragmented QRS should not be used in isolation for mortality risk stratification in surgical ICU patients. Instead, clinical decision-making should primarily rely on frailty, metabolic status, and established severity scores. Future studies should evaluate fQRS within integrated, multimodal prognostic models using prospective and multicenter designs.

Conclusion

In this cohort of general surgical ICU patients, fragmented QRS did not predict 28-day or in-hospital mortality, whereas frailty, renal dysfunction, metabolic acidosis, hypoalbuminemia, and elevated lactate were the dominant prognostic determinants. These findings indicate that, in non-cardiac critical illness, systemic physiologic and metabolic failure outweighs isolated ECG abnormalities. Our results support the need for context-specific risk assessment strategies rather than reliance on single electrophysiologic markers.

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