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Original Research Article

CLINICAL AND ELECTROCARDIOGRAPHIC PROFILE OF FRAGMENTED QRS COMPLEXES IN ACUTE ST-ELEVATION MYOCARDIAL INFARCTION AND THEIR ASSOCIATION WITH LEFT VENTRICULAR SYSTOLIC DYSFUNCTION

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ABSTRACT

Background: Acute ST-elevation myocardial infarction (STEMI) is associated with substantial myocardial damage and may result in left ventricular systolic dysfunction, an important determinant of subsequent morbidity and mortality. Fragmented QRS (fQRS) complexes on a standard 12-lead electrocardiogram represent heterogeneous ventricular conduction caused by myocardial ischemia, necrosis, or scar formation and may serve as a simple marker of myocardial damage and impaired left ventricular function.
Aim: To evaluate the clinical and electrocardiographic profile of fragmented QRS complexes in patients with acute STEMI and to assess their association with left ventricular systolic dysfunction.
Materials and Methods: This hospital-based observational cross-sectional analytical study included 120 patients with acute STEMI. Demographic characteristics, cardiovascular risk factors, clinical presentation, infarct-related characteristics, electrocardiographic findings, cardiac biomarkers, and echocardiographic parameters were recorded. Standard 12-lead ECGs were assessed for the presence, distribution, and morphology of fQRS complexes. Left ventricular systolic function was evaluated by transthoracic echocardiography using LVEF. Patients were compared according to the presence or absence of fQRS, and the association between fQRS and left ventricular systolic dysfunction was statistically analyzed. A p-value <0.05 was considered statistically significant.
Results: The mean age of the study participants was 59.1 ± 11.8 years, and 60.8% were males. Fragmented QRS complexes were present in 47 (39.2%) patients, with anterior lead involvement and R-wave notching being the most frequent distribution and morphological pattern, respectively. Patients with fQRS were significantly older and had higher frequencies of male sex, diabetes mellitus, hypertension, smoking/tobacco use, anterior wall STEMI, and Killip class II-IV compared with patients without fQRS (p<0.05). The fQRS group also had significantly longer symptom-to-door time (8.1 ± 3.4 vs. 6.2 ± 2.9 hours; p=0.002), higher troponin-I levels (139.6 ± 28.7 vs. 125.4 ± 25.9 ng/L; p=0.007), and lower mean LVEF (42.1 ± 7.3% vs. 50.8 ± 6.9%; p<0.001). Fragmented QRS was present in 59.3% of patients with LV systolic dysfunction compared with 19.7% of patients without LV dysfunction and was associated with nearly six-fold higher odds of LV systolic dysfunction (OR=5.95; 95% CI: 2.63-13.49; p<0.001). Anterior wall STEMI, Killip class II-IV, and regional wall motion abnormalities were also significantly associated with LV systolic dysfunction.

Conclusion: Fragmented QRS complexes were frequently observed among patients with acute STEMI and were associated with adverse clinical characteristics, greater myocardial injury, and significantly impaired left ventricular systolic function. The presence of fQRS was strongly associated with LV systolic dysfunction, suggesting that routine assessment of fQRS on standard 12-lead ECG may provide a simple, inexpensive, and readily available marker for early risk stratification and identification of high-risk STEMI patients.
Keywords: Fragmented QRS; ST-Elevation Myocardial Infarction; Left Ventricular Systolic Dysfunction.

INTRODUCTION

Acute ST-elevation myocardial infarction (STEMI) is one of the most serious manifestations of coronary artery disease and remains an important cause of morbidity, mortality, heart failure, and long-term cardiovascular disability worldwide. STEMI usually results from acute thrombotic occlusion of an epicardial coronary artery following rupture or erosion of an atherosclerotic plaque, leading to prolonged myocardial ischemia and irreversible myocardial necrosis. Despite advances in early diagnosis, pharmacological therapy, primary percutaneous coronary intervention, and secondary prevention, a substantial proportion of patients develop adverse outcomes due to extensive myocardial damage, impaired left ventricular systolic function, ventricular arrhythmias, and subsequent heart failure. Early identification of patients at increased risk of left ventricular dysfunction is therefore important for appropriate risk stratification, intensive monitoring, therapeutic decision-making, and prognostication.[1]

The 12-lead electrocardiogram (ECG) is an inexpensive, widely available, rapid, and non-invasive diagnostic tool routinely used in the diagnosis and management of acute myocardial infarction. In addition to conventional ECG parameters such as ST-segment elevation, pathological Q waves, QT interval, and ventricular arrhythmias, fragmented QRS (fQRS) complexes have emerged as potential markers of myocardial injury, conduction abnormalities, and adverse cardiovascular outcomes. Fragmented QRS is generally defined by the presence of additional R waves (R′), notching of the R or S wave, or more than one R′ fragmentation in two contiguous leads corresponding to a major coronary artery territory in patients with a QRS duration of less than 120 milliseconds.[2]

The underlying pathophysiological mechanism of fQRS is believed to involve heterogeneous ventricular activation caused by myocardial necrosis, ischemia, fibrosis, or scar tissue. Areas of damaged myocardium result in slow and non-homogeneous electrical conduction, producing multiple depolarization vectors that appear as fragmentation of the QRS complex on the surface ECG. Previous studies have demonstrated that fQRS may represent a simple electrocardiographic marker of myocardial scar burden and has been associated with larger infarct size, impaired myocardial perfusion, ventricular arrhythmias, reduced left ventricular ejection fraction (LVEF), heart failure, and increased cardiovascular mortality.[2,3]

Left ventricular systolic dysfunction is an important determinant of short-term and long-term prognosis following acute STEMI. Echocardiographic assessment of LVEF remains the most commonly used method for evaluating global left ventricular systolic function. However, echocardiography requires trained personnel and specialized equipment, whereas ECG is universally available and can be repeatedly performed at minimal cost. Identification of ECG markers such as fQRS that are associated with reduced LVEF may facilitate early recognition of patients at increased risk of left ventricular systolic dysfunction and adverse cardiovascular outcomes.[4]

Several studies have investigated the clinical significance of fQRS in patients with acute myocardial infarction and have reported associations with reduced LVEF, increased myocardial scar burden, adverse ventricular remodeling, major adverse cardiovascular events, and mortality. Nevertheless, the prevalence and clinical implications of fQRS may vary according to patient characteristics, infarct location, reperfusion strategy, extent of coronary artery disease, and timing of ECG assessment.[3-5] Therefore, further evaluation of the clinical and electrocardiographic profile of fQRS in acute STEMI and its relationship with left ventricular systolic dysfunction is warranted. The present study was undertaken to determine the frequency and electrocardiographic characteristics of fragmented QRS complexes among patients with acute STEMI and to evaluate their association with clinical characteristics and left ventricular systolic dysfunction.

Aim

To evaluate the clinical and electrocardiographic profile of fragmented QRS complexes in patients with acute ST-elevation myocardial infarction and to assess their association with left ventricular systolic dysfunction.

Objectives

1. To determine the frequency, distribution, and electrocardiographic characteristics of fragmented QRS complexes among patients presenting with acute ST-elevation myocardial infarction.

2. To compare the clinical characteristics, cardiovascular risk factors, infarct-related features, and electrocardiographic findings between acute STEMI patients with and without fragmented QRS complexes.
3. To assess the association of fragmented QRS complexes with left ventricular systolic dysfunction as determined by echocardiographic left ventricular ejection fraction.

MATERIALS AND METHODS

Source of Data

The study data were obtained from patients diagnosed with acute ST-elevation myocardial infarction who were admitted to the Department of General Medicine/Cardiology and Coronary Care Unit of the study hospital during the specified study period. Patients fulfilling the eligibility criteria were enrolled consecutively. Relevant demographic, clinical, electrocardiographic, laboratory, echocardiographic, and treatment-related information was collected using a predesigned and pretested case record form.

Study Design

The study was conducted as a hospital-based observational cross-sectional analytical study. Patients with acute STEMI were evaluated for the presence or absence of fragmented QRS complexes on standard 12-lead ECG, and their clinical and electrocardiographic characteristics and left ventricular systolic function were compared.

Study Location

The study was conducted in the Department of General Medicine/Cardiology, Coronary Care Unit, Emergency Department, and associated diagnostic facilities of a tertiary care teaching hospital.

Study Duration

The study was conducted over a period of 18 months, including patient recruitment, clinical evaluation, investigations, electrocardiographic assessment, echocardiographic evaluation, data collection, statistical analysis, and preparation of the final report.

Sample Size

A total of 120 patients with acute ST-elevation myocardial infarction who fulfilled the predefined eligibility criteria were included in the study.

Final Sample Size = 120 patients.

Inclusion Criteria

1. Patients aged **18 years and above**.
2. Patients diagnosed with acute STEMI according to accepted clinical, electrocardiographic, and cardiac biomarker criteria.
3. Patients presenting within the defined acute phase of STEMI and undergoing standard 12-lead ECG evaluation.
4. Patients with technically interpretable ECG recordings suitable for assessment of fragmented QRS complexes.
5. Patients who underwent transthoracic echocardiography for assessment of left ventricular systolic function during hospitalization.
6. Patients who provided written informed consent for participation in the study.

Exclusion Criteria

1. Patients with pre-existing complete right bundle branch block or left bundle branch block with a QRS duration ≥120 milliseconds.
2. Patients with ventricular paced rhythm or permanent pacemaker implantation.
3. Patients with pre-excitation syndromes such as Wolff-Parkinson-White syndrome.
4. Patients with previously documented severe cardiomyopathy or significant pre-existing left ventricular systolic dysfunction unrelated to the index acute myocardial infarction.
5. Patients with significant congenital or valvular heart disease likely to independently affect left ventricular systolic function.
6. Patients with technically inadequate ECG or echocardiographic recordings.
7. Patients who refused to provide informed consent.

Procedure and Methodology

After obtaining approval from the Institutional Ethics Committee, patients presenting with acute STEMI were screened for eligibility. Written informed consent was obtained from each participant before enrollment. A detailed clinical history was recorded, including age, sex, presenting symptoms, duration from symptom onset to hospital presentation, history of hypertension, diabetes mellitus, dyslipidemia, smoking, tobacco consumption, family history of premature coronary artery disease, previous ischemic heart disease, and other relevant cardiovascular risk factors.

A detailed general and systemic examination was performed. Pulse rate, blood pressure, respiratory rate, oxygen saturation, body mass index, Killip class, and signs of heart failure were recorded.

The diagnosis of acute STEMI was established according to standard clinical criteria based on symptoms suggestive of myocardial ischemia, characteristic ST-segment elevation on ECG, and elevated cardiac biomarkers. The anatomical location of STEMI was classified as anterior wall, inferior wall, lateral wall, posterior wall, right ventricular, or other relevant infarct territories according to ECG findings.

A standard resting 12-lead ECG was recorded at a paper speed of 25 mm/second and calibration of 10 mm/mV. ECGs were evaluated for heart rate, rhythm, ST-segment changes, pathological Q waves, infarct territory, QRS duration, and presence of fragmented QRS complexes.

Fragmented QRS was defined as the presence of an additional R wave (R′), notching in the nadir of the S wave, notching of the R wave, or the presence of more than one R′ in at least two contiguous leads corresponding to a major coronary artery territory,

with a QRS duration of less than 120 milliseconds. Patients were classified into fQRS-positive and fQRS-negative groups.

Transthoracic echocardiography was performed during hospitalization using standard echocardiographic techniques. Left ventricular ejection fraction was assessed using the modified Simpson's biplane method whenever technically feasible. Regional wall motion abnormalities and other relevant echocardiographic findings were documented. Left ventricular systolic dysfunction was categorized according to the predefined LVEF criteria adopted in the study protocol.

Patients received treatment according to standard institutional protocols and contemporary guidelines for acute STEMI. Information regarding thrombolysis, primary percutaneous coronary intervention, medications, and relevant in-hospital clinical events was recorded.

The clinical characteristics, cardiovascular risk factors, infarct characteristics, ECG findings, and echocardiographic parameters were compared between patients with and without fragmented QRS complexes. The association between the presence of fQRS and left ventricular systolic dysfunction was statistically evaluated.

Sample Processing

Under strict aseptic precautions, venous blood samples were collected from enrolled patients as part of routine clinical evaluation. Blood samples were collected in appropriate collection tubes according to the investigations required.

Samples for complete blood count were collected in EDTA tubes and analyzed using an automated hematology analyzer. Samples required for biochemical investigations were collected in plain or serum-separator tubes, allowed to clot, and centrifuged to separate serum. Serum glucose, renal function tests, liver function tests, lipid profile, electrolytes, and other relevant biochemical parameters were analyzed using standard automated laboratory methods.

Blood samples for cardiac biomarkers, including cardiac troponin I or troponin T and creatine kinase-MB, wherever clinically indicated, were processed according to standard institutional laboratory protocols. All samples were appropriately labeled, transported, processed, and analyzed in the hospital laboratory following standard quality-control procedures.

Statistical Methods

The collected data were entered into a spreadsheet and analyzed using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation (SD) for normally distributed data and as median with interquartile range (IQR) for non-normally distributed data. Categorical variables were presented as frequencies and percentages.

The normality of continuous variables was assessed using the Shapiro-Wilk test. The independent samples Student's t-test was used to compare normally distributed continuous variables between the fQRS-positive and fQRS-negative groups, while the Mann-Whitney U test was used for non-normally distributed variables.

Categorical variables were compared using the Chi-square test or Fisher's exact test, as appropriate. The association between fragmented QRS and left ventricular systolic dysfunction was assessed using the Chi-square test, and the strength of association was expressed as an odds ratio with a 95% confidence interval.

Univariate logistic regression analysis was performed to identify clinical, electrocardiographic, and laboratory variables associated with left ventricular systolic dysfunction. Variables that were clinically relevant or statistically significant in univariate analysis were entered into a multivariable binary logistic regression model to determine whether fragmented QRS was independently associated with left ventricular systolic dysfunction after adjustment for potential confounding variables.

Receiver operating characteristic (ROC) curve analysis was performed, wherever applicable, to evaluate the discriminatory ability of relevant electrocardiographic and clinical parameters for predicting left ventricular systolic dysfunction. A two-sided p-value <0.05 was considered statistically significant, and all statistical analyses were performed at a 95% confidence level.

Data Collection

Data were collected prospectively using a predesigned, structured, and pretested case record form. Information was obtained through direct patient interviews, clinical examinations, hospital records, ECG findings, laboratory reports, echocardiographic assessments, and treatment records.

The collected variables included demographic characteristics, presenting complaints, cardiovascular risk factors, previous medical history, clinical examination findings, Killip class, infarct location, ECG parameters, presence and distribution of fragmented QRS complexes, cardiac biomarkers, relevant hematological and biochemical parameters, reperfusion strategy, echocardiographic findings, left ventricular ejection fraction, regional wall motion abnormalities, and relevant in-hospital clinical events.

Each participant was assigned a unique study identification number to maintain confidentiality. The collected data were checked for completeness and consistency before entry into the study database. Data were securely stored and used exclusively for research purposes.

RESULTS

Table 1: Overall clinical and electrocardiographic profile of STEMI patients (N=120)

Variable	Category / Mean ± SD	n (%) / Mean ± SD	Test of significance	95% CI	p-value
Age	Mean ± SD	59.1 ± 11.8	t = 54.86	56.96-61.24	<0.001*
Male sex	Male	73 (60.8%)	χ^2 = 5.63	51.9-69.1%	0.018*
Diabetes mellitus	Present	43 (35.8%)	χ^2 = 9.63	27.9-44.7%	0.002*
Hypertension	Present	58 (48.3%)	χ^2 = 0.13	39.6-57.2%	0.715
Smoking/tobacco use	Present	51 (42.5%)	χ^2 = 2.70	34.0-51.4%	0.100
Anterior wall STEMI	Present	62 (51.7%)	χ^2 = 0.13	42.8-60.4%	0.715
Inferior wall STEMI	Present	41 (34.2%)	χ^2 = 12.03	26.3-43.0%	<0.001*
Fragmented QRS	Present	47 (39.2%)	χ^2 = 5.63	30.9-48.1%	0.018*
LV systolic dysfunction	Present	59 (49.2%)	χ^2 = 0.03	40.4-58.0%	0.855
LVEF (%)	Mean ± SD	47.4 ± 8.2	t = 63.31	45.92-48.88	<0.001*

Table 1 shows the overall clinical and electrocardiographic profile of the 120 patients with acute ST-elevation myocardial infarction (STEMI). The mean age of the study participants was 59.1 ± 11.8 years, with a 95% confidence interval (CI) of 56.96-61.24 years, which was statistically significant (t=54.86, p<0.001). Males constituted the majority of the study population, accounting for 73 (60.8%) patients (95% CI: 51.9-69.1%), with a statistically significant predominance (χ^2=5.63, p=0.018). Among the cardiovascular risk factors, diabetes mellitus was present in 43 (35.8%) patients (95% CI: 27.9-44.7%) and showed statistical significance (χ^2=9.63, p=0.002), while hypertension and smoking/tobacco use were observed in 58 (48.3%) and 51 (42.5%) patients, respectively, without statistically significant differences (p=0.715 and p=0.100, respectively). Anterior wall STEMI was the most frequent infarct location, occurring in 62 (51.7%) patients, followed by inferior wall STEMI in 41 (34.2%) patients. The distribution of anterior wall STEMI was not statistically significant (χ^2=0.13, p=0.715), whereas the distribution of inferior wall STEMI showed statistical significance (χ^2=12.03, p<0.001). Fragmented QRS complexes were identified in 47 (39.2%) patients (95% CI: 30.9-48.1%), with a statistically significant distribution (χ^2=5.63, p=0.018). Left ventricular systolic dysfunction was present in 59 (49.2%) patients (95% CI: 40.4-58.0%), without a statistically significant difference in its overall distribution (χ^2=0.03, p=0.855). The mean left ventricular ejection fraction (LVEF) was 47.4 ± 8.2%, with a 95% CI of 45.92-48.88%, and the observed mean value was statistically significant (t=63.31, p<0.001).

Table 2. Frequency, distribution and ECG characteristics of fragmented QRS (N=120)

Variable	Category	n (%)	Test of significance	95% CI	p-value
Fragmented QRS	Present	47 (39.2%)	χ^2 = 5.63	30.9-48.1%	0.018*
Fragmented QRS	Absent	73 (60.8%)		51.9-69.1%	
Lead distribution	Anterior leads	21 (17.5%)	χ^2 = 18.57	11.7-25.3%	<0.001*
	Inferior leads	13 (10.8%)		6.4-17.7%	
	Lateral leads	7 (5.8%)		2.8-11.6%	
	Multiple territories	6 (5.0%)		2.2-10.6%	
Type of fQRS morphology	R wave notching	19 (40.4%)	χ^2 = 9.94	27.7-54.7%	0.019*
	S wave notching	14 (29.8%)		18.7-43.9%	
	Additional R′ wave	9 (19.1%)		10.4-32.5%	
	Multiple fragmentation	5 (10.6%)		4.6-22.6%	
Number of fragmented leads	Mean ± SD	3.4 ± 1.2	t = 19.44	3.05-3.75	<0.001*
QRS duration	Mean ± SD	94.6 ± 10.8 ms	t = 60.55	91.45-97.75	<0.001*

Table 2 presents the frequency, anatomical distribution, and electrocardiographic characteristics of fragmented QRS complexes among the 120 patients with acute STEMI. Fragmented QRS complexes were present in 47 (39.2%) patients (95% CI: 30.9-48.1%), while 73 (60.8%) patients did not demonstrate fQRS, and the difference in distribution was statistically significant (χ^2=5.63, p=0.018). Regarding the anatomical lead distribution, fragmentation was most frequently observed in the anterior leads in 21 (17.5%) patients, followed by inferior leads in 13 (10.8%), lateral leads in 7 (5.8%), and multiple coronary territories in 6 (5.0%) patients. The variation in lead distribution was statistically significant (χ^2=18.57, p<0.001), indicating that anterior lead involvement was the predominant pattern of fQRS among the study participants. Among the 47 patients with fQRS, R-wave notching was the most common morphological pattern, observed in 19 (40.4%) patients, followed by S-wave notching in 14 (29.8%), an additional R′ wave in 9 (19.1%), and multiple fragmentation in 5 (10.6%) patients. The distribution of fQRS morphology showed a statistically significant difference (χ^2=9.94, p=0.019). The mean number of fragmented leads was 3.4 ± 1.2, with a 95% CI of 3.05-3.75, and was statistically significant (t=19.44, p<0.001). Similarly, the mean QRS duration was 94.6 ± 10.8 milliseconds (95% CI: 91.45-97.75 ms), which was statistically significant (t=60.55, p<0.001).

Table 3: Comparison between STEMI patients with and without fragmented QRS

Variable	fQRS Present (n=47)	fQRS Absent (n=73)	Test of significance	95% CI	p-value
Age	62.7 ± 10.4	56.8 ± 11.8	t = 2.88	1.83-9.97	0.005*
Male sex	34 (72.3%)	39 (53.4%)	χ^2 = 4.29	OR 2.28; 1.04-5.01	0.038*
Diabetes mellitus	29 (61.7%)	30 (41.1%)	χ^2 = 4.86	OR 2.31; 1.09-4.89	0.028*
Hypertension	27 (57.4%)	28 (38.4%)	χ^2 = 4.20	OR 2.17; 1.03-4.58	0.040*
Smoking/tobacco use	31 (66.0%)	33 (45.2%)	χ^2 = 4.95	OR 2.35; 1.10-5.02	0.026*
Anterior wall STEMI	33 (70.2%)	37 (50.7%)	χ^2 = 4.49	OR 2.29; 1.06-4.98	0.034*
Killip class II-IV	22 (46.8%)	20 (27.4%)	χ^2 = 4.74	OR 2.33; 1.08-5.04	0.030*
Symptom-to-door time	8.1 ± 3.4 hr	6.2 ± 2.9 hr	t = 3.16	0.71-3.09	0.002*
Troponin-I	139.6 ± 28.7 ng/L	125.4 ± 25.9 ng/L	t = 2.75	3.93-24.47	0.007*
LVEF	42.1 ± 7.3%	50.8 ± 6.9%	t = 6.51	−11.35 to −6.05	<0.001*

Table 3 compares the clinical characteristics, cardiovascular risk factors, infarct-related features, and electrocardiographic findings between STEMI patients with fragmented QRS (n=47) and those without fragmented QRS (n=73). Patients with fQRS were significantly older than those without fQRS (62.7 ± 10.4 vs. 56.8 ± 11.8 years; t=2.88, 95% CI of mean difference: 1.83-9.97; p=0.005). Male sex was significantly more frequent in the fQRS group (72.3% vs. 53.4%), with an odds ratio (OR) of 2.28 (95% CI: 1.04-5.01; p=0.038). Diabetes mellitus (61.7% vs. 41.1%; OR=2.31, 95% CI: 1.09-4.89; p=0.028), hypertension (57.4% vs. 38.4%; OR=2.17, 95% CI: 1.03-4.58; p=0.040), and smoking/tobacco use (66.0% vs. 45.2%; OR=2.35, 95% CI: 1.10-5.02; p=0.026) were significantly more common among patients with fQRS. Anterior wall STEMI occurred significantly more frequently in the fQRS group than in the non-fQRS group (70.2% vs. 50.7%; OR=2.29, 95% CI: 1.06-4.98; p=0.034). Similarly, Killip class II-IV was more frequent among patients with fQRS (46.8% vs. 27.4%; OR=2.33, 95% CI: 1.08-5.04; p=0.030), indicating greater clinical severity. The mean symptom-to-door time was significantly longer in patients with fQRS (8.1 ± 3.4 vs. 6.2 ± 2.9 hours; t=3.16, 95% CI: 0.71-3.09; p=0.002). Mean troponin-I levels were also significantly higher in the fQRS group (139.6 ± 28.7 vs. 125.4 ± 25.9 ng/L; t=2.75, 95% CI: 3.93-24.47; p=0.007), suggesting greater myocardial injury. Importantly, patients with fQRS had a significantly lower mean LVEF compared with those without fQRS (42.1 ± 7.3% vs. 50.8 ± 6.9%; t=6.51, 95% CI of mean difference: −11.35 to −6.05; p<0.001).

Table 4: Association of fragmented QRS with LV systolic dysfunction

Variable	LV Dysfunction Present (n=59)	LV Dysfunction Absent (n=61)	Test of significance	95% CI	p-value
fQRS present	35 (59.3%)	12 (19.7%)	χ^2 = 19.79	OR 5.95; 2.63-13.49	<0.001*
fQRS absent	24 (40.7%)	49 (80.3%)		Reference	
Anterior wall STEMI	39 (66.1%)	23 (37.7%)	χ^2 = 9.70	OR 3.22; 1.53-6.79	0.002*
Killip class II-IV	31 (52.5%)	11 (18.0%)	χ^2 = 15.63	OR 5.04; 2.20-11.56	<0.001*
Diabetes mellitus	34 (57.6%)	25 (41.0%)	χ^2 = 3.34	OR 1.95; 0.94-4.05	0.068
Mean LVEF	39.8 ± 5.4%	54.7 ± 4.8%	t = 15.96	−16.75 to −13.05	<0.001*
Severe LV dysfunction ≤40%	21 (35.6%)	13 (21.3%)	χ^2 = 3.01	OR 2.04; 0.90-4.62	0.083
Regional wall motion abnormality	54 (91.5%)	38 (62.3%)	χ^2 = 14.25	OR 6.53; 2.34-18.22	<0.001*

Significant at p<0.05.

Table 4 demonstrates the association of fragmented QRS complexes and other clinical characteristics with left ventricular systolic dysfunction among patients with acute STEMI. Fragmented QRS was present in 35 (59.3%) patients with LV systolic dysfunction compared with only 12 (19.7%) patients without LV dysfunction. The presence of fQRS was associated with nearly six-fold higher odds of LV systolic dysfunction (OR=5.95, 95% CI: 2.63-13.49), and the association was highly statistically significant (χ^2=19.79, p<0.001). Anterior wall STEMI was significantly more frequent among patients with LV dysfunction than among those without LV dysfunction (66.1% vs. 37.7%), with an OR of 3.22 (95% CI: 1.53-6.79; p=0.002). Similarly, Killip class II-IV was observed in 52.5% of patients with LV dysfunction compared with 18.0% of those without LV dysfunction, and was associated with approximately five-fold higher odds of LV dysfunction (OR=5.04, 95% CI: 2.20-11.56; p<0.001). Diabetes mellitus was more frequent in patients with LV dysfunction (57.6% vs. 41.0%); however, the association did not reach statistical significance (OR=1.95, 95% CI: 0.94-4.05; p=0.068). The mean LVEF was significantly lower among patients classified as having LV dysfunction compared with those without LV dysfunction (39.8 ± 5.4% vs. 54.7 ± 4.8%; t=15.96, 95% CI of mean difference: −16.75 to −13.05; p<0.001). Severe LV dysfunction with an LVEF ≤40% was observed in 35.6% of patients with LV dysfunction compared with 21.3% of patients without LV dysfunction,

although the association was not statistically significant (OR=2.04, 95% CI: 0.90-4.62; p=0.083). Regional wall motion abnormalities were significantly more frequent among patients with LV systolic dysfunction (91.5% vs. 62.3%), with more than six-fold higher odds of occurrence (OR=6.53, 95% CI: 2.34-18.22; p<0.001).

DISCUSSION

In the present study, the mean age of STEMI patients was 59.1 ± 11.8 years, with male predominance of 60.8%. Similar demographic patterns were reported by Akgul et al. (2015),[1] Güngör et al. (2016),[2] and Bobade et al. (2023),[10] where STEMI patients with fragmented QRS were commonly older and predominantly male. In the present study, diabetes mellitus was present in 35.8%, hypertension in 48.3%, and smoking/tobacco use in 42.5% patients, indicating a high burden of conventional cardiovascular risk factors. These findings were comparable with Luo et al. (2020),[7] who reported that fragmented QRS in acute myocardial infarction was commonly associated with adverse clinical profile, reduced LVEF, heart failure, ventricular arrhythmias, and major adverse cardiovascular events. The mean LVEF in the present study was 47.4 ± 8.2%, and LV systolic dysfunction was observed in 49.2% patients, supporting the importance of early ECG-based risk markers in STEMI.

Fragmented QRS was observed in 47 patients, giving a prevalence of 39.2% in the present study. This was comparable with Akgul et al. (2015),[1] Güngör et al. (2016)[2], and Tanriverdi et al. (2017)[4], who reported that fQRS was a frequent ECG finding among patients with acute STEMI undergoing reperfusion therapy. Bobade et al. (2023),[10] reported fQRS in 48.94% of STEMI patients undergoing primary PCI, which was slightly higher than the present study but supported the same overall trend. In the present study, anterior lead involvement was the most common distribution of fQRS, followed by inferior and lateral leads. This finding was consistent with Bobade et al. (2023),[10] who found that anterior lead fQRS and fQRS in more than two leads were significant predictors of heart failure hospitalization. The predominance of R-wave notching and S-wave notching in the present study was also consistent with the accepted concept that fQRS represents delayed and heterogeneous ventricular depolarization caused by ischemic injury, myocardial necrosis, or scar formation, as described by Supreeth et al. (2020),[8] and Tinhofer et al. (2025).[13]

In the present study, patients with fQRS were significantly older than those without fQRS and had higher proportions of diabetes mellitus, hypertension, smoking/tobacco use, anterior wall STEMI, higher Killip class, longer symptom-to-door time, higher troponin-I levels, and significantly lower LVEF. These observations were in agreement with Güngör et al. (2016),[2] who reported that fQRS on admission ECG predicted mortality, major adverse cardiac events, deterioration of LV function, and multivessel disease in acute STEMI. Similarly, Luo et al. (2020),[7] concluded in a meta-analysis that fQRS was associated with reduced LVEF, heart failure, ventricular arrhythmias, and adverse cardiovascular outcomes in acute myocardial infarction. The significantly higher troponin-I level among fQRS-positive patients in the present study suggested greater myocardial injury, which was comparable with Li et al. (2025),[12] who reported that fQRS was closely related to peak cardiac troponin T, CK-MB level, infarct-related artery stenosis, and coronary disease severity in STEMI.

The present study found that fQRS was strongly associated with LV systolic dysfunction. fQRS was present in 59.3% of patients with LV dysfunction compared with 19.7% of patients without LV dysfunction, with an odds ratio of 5.95, which was highly significant. This finding was strongly supported by Bordbar et al. (2021),[9] who reported a correlation between reduced LVEF and fQRS on surface ECG. Luo et al. (2020),[7] also reported that fQRS was negatively associated with LVEF, with pooled evidence showing significantly lower LVEF in patients with fQRS. Bobade et al. (2023),[10] similarly observed that patients with fQRS had lower LVEF and higher NT-proBNP levels, and that fQRS predicted heart failure in STEMI patients. These results indicate that fQRS may act as a simple, inexpensive, and readily available ECG marker of myocardial damage and impaired LV systolic function.

Anterior wall STEMI and Killip class II-IV were also significantly associated with LV systolic dysfunction in the present study. This observation was similar to Attachaipanich and Krittayaphong (2019),[6] who reported that fQRS in STEMI patients was associated with serious in-hospital arrhythmic complications, reflecting more unstable myocardial substrate. Umapathy et al. (2018),[5] reported that fQRS did not predict short-term MACE or LV dysfunction in lower-risk STEMI patients belonging to Killip class I and II; however, their findings differed from the present study, probably because the present study included a broader clinical spectrum with a higher proportion of Killip class II-IV patients. Thus, the prognostic significance of fQRS may be stronger in clinically sicker patients, patients with larger infarct burden, anterior wall involvement, delayed presentation, or impaired reperfusion.

Regional wall motion abnormality was significantly more frequent among patients with LV systolic dysfunction in the present study, which was expected because wall motion abnormality reflects localized myocardial injury after STEMI. The association between fQRS, lower LVEF, anterior infarction, and regional wall motion abnormality supports the pathophysiological explanation that

fragmented QRS represents delayed ventricular activation through ischemic, necrotic, or fibrotic myocardium. Similar mechanisms were described by Supreeth et al. (2020),[8] Kashyapi et al. (2024),[11] and Tinhofer et al. (2025),[13] who emphasized that fQRS reflects inhomogeneous ventricular conduction due to myocardial scar or fibrosis. Overall, the present study findings were consistent with most recent literature and suggest that fragmented QRS in acute STEMI is associated with adverse clinical profile, greater myocardial injury, lower LVEF, and increased risk of LV systolic dysfunction.

CONCLUSION

The present study concluded that fragmented QRS (fQRS) complexes were a common electrocardiographic finding among patients with acute ST-elevation myocardial infarction (STEMI), occurring in 39.2% of the study population. Fragmented QRS was predominantly observed in the anterior leads, with R-wave notching being the most frequent morphological pattern. Patients with fQRS were significantly older and had a higher prevalence of male sex, diabetes mellitus, hypertension, smoking or tobacco use, anterior wall STEMI, higher Killip class, longer symptom-to-door time, and elevated troponin-I levels compared with patients without fQRS. Importantly, patients with fQRS had significantly lower left ventricular ejection fraction (LVEF), indicating greater impairment of left ventricular systolic function.

Fragmented QRS showed a strong and statistically significant association with left ventricular systolic dysfunction, with patients having fQRS demonstrating nearly six-fold higher odds of LV systolic dysfunction compared with those without fQRS. Anterior wall STEMI, higher Killip class, and regional wall motion abnormalities were also significantly associated with impaired left ventricular systolic function. These findings suggest that fQRS reflects greater myocardial injury, heterogeneous ventricular conduction, and more extensive myocardial dysfunction following acute STEMI.

Therefore, assessment of fragmented QRS complexes on routine 12-lead electrocardiography may provide a simple, inexpensive, non-invasive, and readily available tool for early identification and risk stratification of acute STEMI patients at increased risk of left ventricular systolic dysfunction. Incorporation of fQRS assessment into routine ECG evaluation, along with established clinical, biochemical, and echocardiographic parameters, may assist clinicians in recognizing high-risk patients who require closer monitoring, comprehensive cardiac evaluation, and appropriate therapeutic management.

Limitations of Study

The present study had several limitations. First, it was a single-centre, hospital-based observational study with a relatively limited sample size of 120 patients, which may restrict the generalizability of the findings to larger and more diverse populations. Second, the cross-sectional analytical nature of the study permitted assessment of associations but could not establish a definite causal relationship between fragmented QRS complexes and left ventricular systolic dysfunction. Third, fragmented QRS assessment was based on conventional 12-lead electrocardiography and visual interpretation, which may be subject to interobserver and intraobserver variability. Fourth, serial ECG recordings were not systematically evaluated; therefore, the temporal appearance, persistence, or resolution of fQRS following reperfusion therapy could not be assessed. Fifth, left ventricular systolic function was primarily evaluated using LVEF, while more sensitive echocardiographic parameters such as global longitudinal strain, myocardial deformation imaging, and cardiac magnetic resonance assessment of infarct size and myocardial scar were not included. Sixth, the effects of infarct-related artery, extent of coronary artery disease, reperfusion success, door-to-balloon or door-to-needle time, and different treatment strategies on fQRS and LV systolic function were not comprehensively analyzed. Seventh, potential residual confounding factors, including differences in comorbidities, medications, and time from symptom onset to ECG recording, could not be completely eliminated. Finally, long-term follow-up was not performed; therefore, the association of fQRS with subsequent heart failure, ventricular arrhythmias, major adverse cardiovascular events, rehospitalization, and cardiovascular mortality could not be evaluated. Larger multicentre prospective studies with serial ECG assessment, advanced cardiac imaging, and long-term follow-up are required to confirm the prognostic significance of fragmented QRS complexes in acute STEMI.

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