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Original Article

Prognostic significance of fragmented QRS in patients with ST-elevation myocardial infarction undergoing revascularization

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ABSTRACT

Background & Objectives: This longitudinal study was carried out to evaluate the prognostic significance of fragmented QRS (fQRS) in patients with acute ST elevation myocardial infarction (STEMI) undergoing revascularization.

Methods: This study included 103 STEMI patients belonging to Killip class I and II who underwent primary revascularization. All patients underwent twelve lead ECG at admission before PCI. Serial ECG were done after PCI at 3 hours, 6 hours, 24 hours, 48 hours and at discharge for detection of fQRS and echocardiography on day 3 post revascularization. Patients developing fQRS within 48 hours and with persistence of fQRS till discharge were included in "persistent fQRS" group. They were followed up after 30 days for major adverse cardiac events (MACE) and assessment of LV function by echocardiography.

Results: fQRS was present in 64 patients (61.5%) of study population with 37 patients (57.8%) having persistent fQRS. MACE rates were low (4.8%) and did not differ with respect to fQRS. fQRS significantly correlated with LV dysfunction at 30 days on univariate analysis (p-0.003) but not on multivariate analysis (p -0.10). fQRS was significantly related to impaired myocardial reperfusion as assessed by ΣSTR (percent of total ST segment resolution) (adjusted odds ratio, 95% CI [4.265 (1.034 − 17.58)], p = 0.04).

Conclusion: In our study, fQRS did not predict MACE and LV dysfunction in acute STEMI patients belonging to Killip class I and II on short term follow-up of 30 days. But, fQRS independently predicted impaired microvascular myocardial reperfusion as assessed by ΣSTR.

1. Introduction

Fragmented QRS (fQRS) is defined as the presence of an additional R wave (R′) or notching in the nadir of the S wave or the presence of >1 R' (fragmentation) in two contiguous leads, corresponding to a major coronary artery territory.[1] fQRS is postulated to be due to altered myocardial activation caused by the presence of myocardial scar or myocardial ischemia.[2,3] It occurs in different patient populations such as coronary artery disease, cardiomyopathies, arrhythmogenic right ventricular cardiomyopathy, Brugada syndrome, congenital heart disease, and long QT syndrome.[4] fQRS occurrence varies from 34% to 60% in patients with acute coronary syndrome (ACS) and usually appears by 48 hours.[5] It is not related to the type of myocardial infarction (MI) [ST-elevation MI (STEMI) or non-ST elevation MI (NSTEMI)].

Recent studies have shown that fQRS may be of significant prognostic value in patients with STEMI undergoing reperfusion therapy.[6–10] STEMI patients with fQRS in electrocardiogram (ECG) tend to have higher cardiac biomarker levels, higher inflammatory markers, higher angina to balloon time, lower left ventricular function, extensive coronary artery involvement, and poorer reperfusion parameters.[11–13] fQRS predicts short-term and long-term mortality and major cardiac events and thus is helpful in risk stratification in patients with STEMI. This study was planned to assess the short-term prognostic significance of fQRS in Indian patients undergoing revascularization for STEMI.

2. Patients and methods

The study was approved by the institute's ethics committee. Each subject gave written informed consent before being included in the study. The guidelines laid down by the Indian Council of Medical Research (1994) and Helsinki declaration (modified in 1989) were adhered to in all patients in the study.

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2.1. Patient population and study protocol

This was a prospective observational study that enrolled 103 patients admitted with the diagnosis of acute STEMI undergoing revascularization [either primary percutaneous coronary intervention (PCI)/thrombolysis] from May 2016 to December 2016. The exclusion criteria were final diagnosis other than STEMI, late presentation after symptom onset (>24 h), cardiogenic shock at admission, mechanically ventilated patients, history of prior MI (<6 months), significant primary valvular disease, presence of bundle branch block (QRS >120 ms), patients with permanent pacemaker implantation, chronic active medical conditions such as chronic kidney disease (glomerular filtration rate <60 ml/min), chronic liver disease, malignancy, recent surgery, or trauma (<1 month), and patient refusal to participate in the study.

Patients with a final diagnosis of STEMI based on the following diagnostic criteria were included: ST-segment elevation of at least 0.1 mV in at least two contiguous leads (in V2-3: >0.2 mV in men >40 years, > 0.25 mV in men <40 years or > 0.15 mV in women) combined with troponin I values > 0.025 ng/ml.[14] Baseline clinical characteristics including patient history, presence of traditional risk factors such as diabetes mellitus, hypertension, smoking, and dyslipidemia were collected. Laboratory parameters including hematology, biochemistry, and lipid profile were measured. Killip score at admission and angina to balloon time were noted as well. Quantitative troponin I was measured using immunoassay-based "point of care" testing (Radiometer, AQT90 FLEX).

2.1.1. Electrocardiogram

Twelve-lead standard ECG was recorded for all patients at admission using (Philips Medical Systems Andover, MA, 01810, USA (PageWiter TC30); filter range 0.5 Hz−150 Hz, alternating current filter 60 Hz) and usual standardization (25 mm/s, 10 mm/mV) and analyzed for the presence of fQRS (as defined earlier), localization of infarct territory, presence of Q waves, and QRS duration.

All patients underwent serial ECG at 3 h, 6 h, 24 h, and 48 h and at the time of discharge for detecting the presence of fQRS and its changes after revascularization. Patients showing fQRS involving infarct territory within 48 h of admission were included in the "fQRS group". Patients who did not develop fQRS even after 48 h of admission were included in the "no fQRS" group. Patients with persistence of fQRS at discharge were considered as having "persistent fQRS" and those who demonstrate resolution of fQRS before discharge were considered as having "transient fQRS".

Localization of infarct territory by fQRS was carried out by using the following criteria: anterior, presence of fQRS in two contiguous anterior leads (V1 to V5); lateral, presence of fQRS in two contiguous lateral leads (I, aVL, V6); and inferior, presence of fQRS in two contiguous inferior leads (II, III, aVF).[15] QRS duration before and after revascularization was recorded in milliseconds, and delta QRS time was calculated by the following formula: (QRS duration pre-PCI) − (QRS duration post-PCI) in milliseconds. Jeopardized myocardium was determined by the sum of ST elevations (in mm) on each ST-elevated derivation on prerevascularization and post-revascularization ECG (total ST elevation score). Percent of total ST-segment resolution (ΣSTR) was calculated by the following formula: (Sum of ST elevations on pre-PCI ECG) − (Sum of ST elevations on post-PCI ECG)/(Sum of ST elevations on pre-PCI ECG) × 100, and 3rd hour ECG recorded after PCI was used.[12] ΣSTR less than 50% is considered as imperfect ST-segment resolution and a marker of impaired myocardial reperfusion.

2.1.2. Coronary angiography and primary PCI

After primary PCI, all patients were monitored in the coronary care unit until stabilization and were treated based on the recommendations of the American College of Cardiology/American Heart Association guidelines for the management of patients with STEMI.[16]

Angiographic data including the number of diseased vessels (≥50% obstruction), infarct-related artery, number of coronary stents placed, and thrombolysis in MI score (TIMI) flow (before and after PCI) in the infarct-related artery were recorded in all patients.[17]

2.1.3. Echocardiography

All patients underwent 2D echocardiography on the third day after revascularization to determine left ventricular (LV) function (Simpson's method), LV volumes, and regional wall motion abnormalities. In-hospital major adverse cardiac events (MACEs) such as death, reinfarction, cardiac arrhythmias, and heart failure were recorded.

2.1.4. Follow-up

Patients were followed up at 1 month after index hospitalization for clinical assessment, 12-lead ECG, and 2D echocardiography. The primary end point was MACE (composite of death from any cause, readmission with acute coronary syndrome (ACS) or congestive heart failure, or ventricular arrhythmia) within 1 month. The secondary end point was association of fQRS with LV function at 1 month and change in LV function compared with baseline (▲LVEF). Exploratory end point was association of fQRS with respect to ΣSTR, a reperfusion parameter.

2.2. Statistical analysis

Descriptive statistics such as mean, median, and proportion were calculated. Continuous variables were given as mean ± standard deviation. Categorical variables were defined as percentages. Continuous variables were compared using Student *t*-test, and the chi-square test was used for the categorical variables between the two groups. All tests with regard to significance were two tailed. Multivariate regression analysis was performed between the independent and dependent variables. Data were entered and analyzed using SPSS 20 (SPSS Inc., Chicago, IL). In this study, *p*-value less than 0.05 was considered as statistically significant.

3. Results

One hundred three patients with acute STEMI who fulfilled the eligibility criteria were enrolled in the study. The mean age of study population was 53 years, of which 87.4% were males and 12.6% were females.

Fig. 1 shows flow of patients in the study. fQRS was present in 64 patients (61.5%), of which 27 patients (42.2%) had fQRS at admission and 37 patients (57.8%) developed fQRS within 48 hours of STEMI (Fig. 2). All patients except one underwent primary PCI (PCI, 102 patients and thrombolysis, 1 patient). Median duration of hospital stay was 4 days (range: 2−6 days).

3.1. Patients with absence or resolution of fQRS (Group 1) versus patients with persistent fQRS (Group 2)

Patients having "transient fQRS" were similar to the "no fQRS" group, hence were analyzed as a single group (absence or resolution of fQRS) and compared with the "persistent fQRS" group. Patients with persistent fQRS had fQRS at admission, prolonged QRS duration, and lesser ST-segment resolution compared with group 1 (Fig. 3). Both groups were comparable in other study parameters including LV function (Table 1).

Event rates were low in this study and occurred during index hospitalization in 5 patients (7.6%) in group 1 (1 patient died, 2

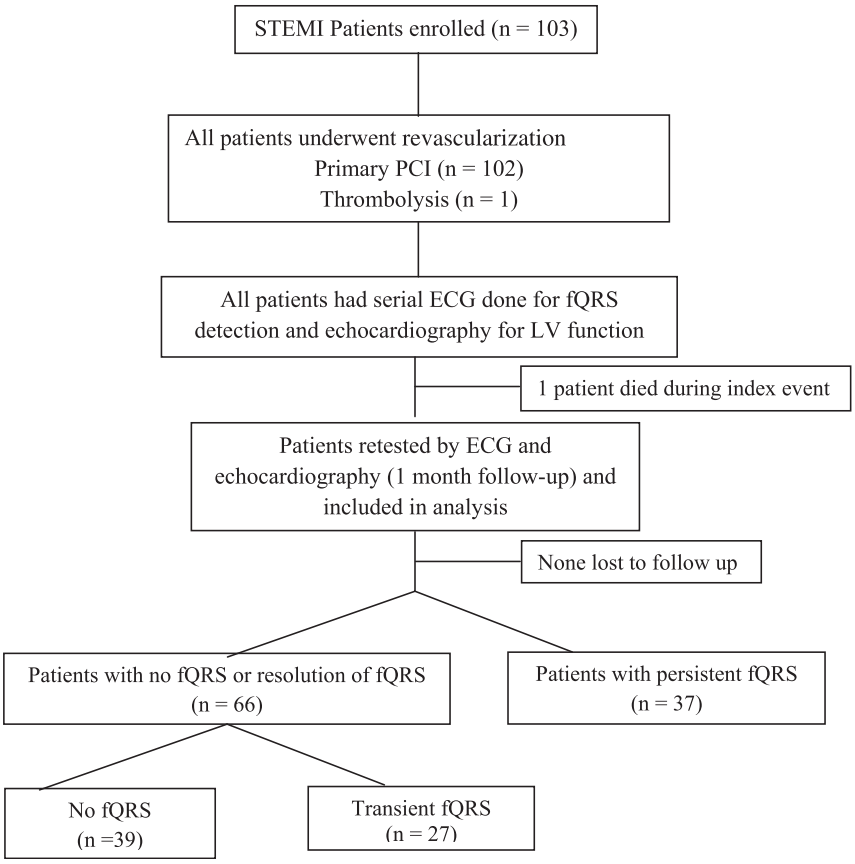

Fig. 1. Flow chart of patients in the study.

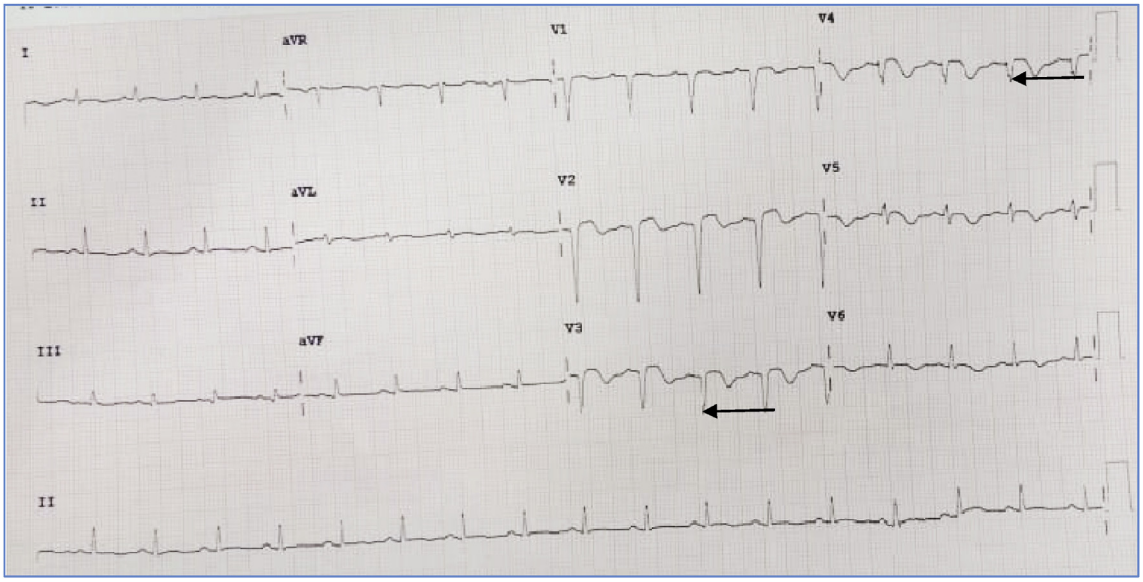

Fig. 2. Illustrative ECG showing fragmented QRS in anterior leads (arrows) in a patient with anterior wall STEMI after PCI.

patients had ventricular tachycardia, and 2 patients had congestive heart failure). No MACE occurred at 30 days of follow-up.

3.1.1. Follow-up (Group 1 versus Group 2)

The persistent fQRS group had lower LV ejection fraction (LVEF) ($p = 0.01$) with increased end-systolic volume ($p = 0.003$) with no difference in end-diastolic volume. They had lesser improvement in LVEF (▲LVEF, $p = 0.003$) (Fig. 4), end-systolic volume (▲ESV, $p = 0.03$), and end-diastolic volume (▲EDV, $p = 0.05$) compared to baseline with respect to group 1 (Table 2). Median duration of follow-up was 30 days (ranging from 25 to 38 days).

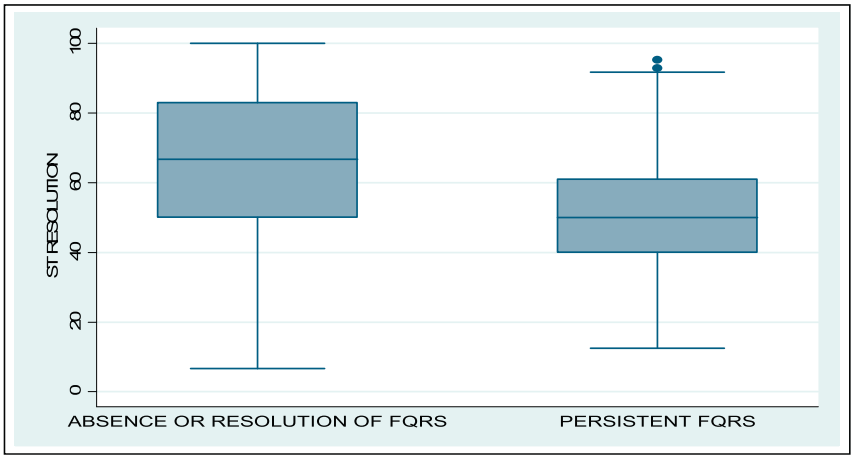

Fig. 3. Relationship of percent of ST resolution with respect to fragmented QRS.

Table 1
Analysis of different parameters in study population.

Parameter	Absence or resolution of fQRS (group 1) ($n = 66$)	Persistent fQRS (group 2) ($n = 37$)	p value
Age (years)	53 ± 13	52 ± 12	0.84
BMI (kg/m^2)	25.6 ± 2.4	26.1 ± 2.4	0.35
Gender (male)	56 (84.8%)	34 (91.8%)	0.30
Hypertension	23 (34.9%)	18 (48.6%)	0.17
Diabetes mellitus	18 (27.2%)	8 (21.6%)	0.52
Smoking	40 (60.6%)	25 (67.5%)	0.48
Dyslipidemia	17 (25.7%)	11 (29.7%)	0.48
History of prior MI	4 (6%)	2 (5.4%)	0.99
Total leukocyte count (10^3/mm^3)	11.6 ± 3.1	11.3 ± 2.5	0.61
Baseline troponin I (ng/ml)	1.12 ± 1.64	0.64 ± 0.84	0.02
Angina to balloon time (hours)	6.1 ± 3.5	6 ± 3	0.95
Median	5 (1–22)	6 (1–14)	
Killip class			
I	59 (89.3%)	32 (86.4%)	0.75
II	7 (10.6%)	5 (13.5%)	
GRACE score			
I (low risk)	32 (48.5%)	19 (51.3%)	0.69
II (intermediate risk)	21 (31.8%)	9 (24.3%)	
III (high risk)	13 (19.7%)	9 (24.3%)	
Q waves on ECG	51 (77.3%)	28 (75.7%)	0.53
ST resolution (%)	66.7 ± 19.8	52.1 ± 20	**<0.001**
QRS duration (ms) pre-–PCI	75.6 ± 11.2	78 ± 15.8	0.35
QRS duration (ms) post-PCI	68.2 ± 11.5	78.6 ± 18	**< 0.001**
Delta QRS time (ms)	−7.4 ± 9.2	0.54 ± 16.6	**0.01**
Presence of fQRS at admission	11 (16.6%)	16 (43.2%)	**0.003**
Noninfarct fQRS	14 (21%)	14 (37.9%)	0.10
STEMI territory			
Anterior	30 (45.5%)	22 (59.5%)	0.28
Inferior	35 (53%)	14 (37.8%)	
Lateral	1 (1.5%)	1 (2.7%)	
Coronary artery disease			
SVD	39 (59.1%)	22 (59.5%)	0.87
DVD	20 (30.3%)	10 (27%)	
TVD	7 (10.6%)	5 (13.5%)	
Post-PCI TIMI score			
0, 1	4 (6%)	3 (8.1%)	0.70
2, 3	62 (94%)	34 (91.9%)	
LV ejection fraction (%) (post-PCI)	45.3 ± 7.5	43 ± 8	0.31
End-systolic volume (ml)	42.6 ± 12	43 ± 12	0.74
End-diastolic volume (ml)	77.5 ± 18	76 ± 17.6	0.68
In-hospital MACE	5 (7.6%)	0 (0%)	0.15

Data are expressed as mean ± standard deviation or n (%) or median (range). Bold values highlight significant p values.
BMI, body mass index; DVD, double-vessel disease; ECG, electrocardiogram; fQRS, fragmented QRS; GRACE, Global Registry of Acute Coronary Events; LV, left ventricle; MI, myocardial infarction; MACE, major adverse cardiovascular events; PCI, percutaneous intervention; STEMI, ST-elevation myocardial infarction; SVD, single-vessel disease; TVD, triple-vessel disease; TIMI, thrombolysis in myocardial infarction score.

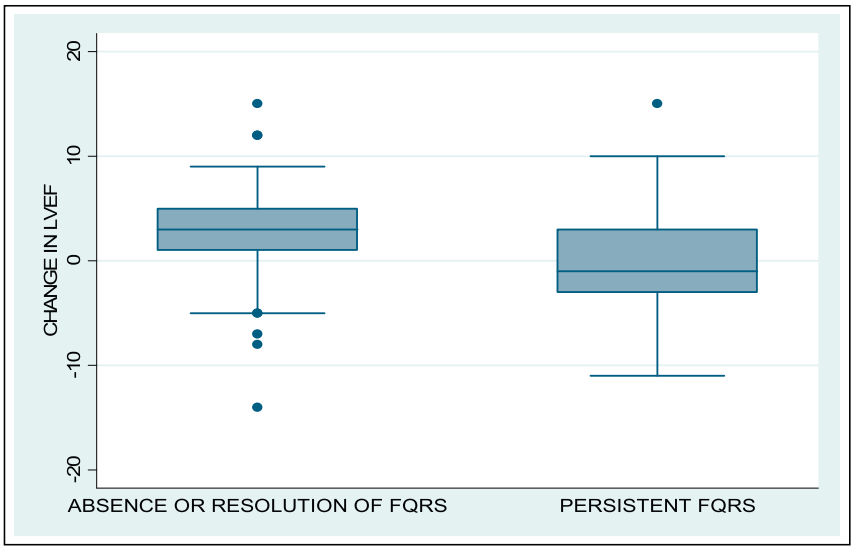

Fig. 4. Relationship of change in LVEF (▲ LVEF) with respect to fragmented QRS.

Table 2
Analysis of parameters at 1-month follow-up in the study population with relation to fragmented QRS.

Parameter	Absence or resolution of fQRS (group 1) ($n = 66$)	Persistent fQRS (group 2) ($n = 37$)	p value
Presence of fQRS at 1 month	2 (3%)	31 (83.8%)	**<0.001**
LV ejection fraction (%) at 1 month	48.2 ± 7.7	43.8 ± 9.9	**0.01**
End-systolic volume (ml) at 1 month	38.5 ± 14.4	45 ± 16	**0.003**
End-diastolic volume (ml) at 1 month	72 ± 20.6	77.8 ± 19.6	0.19
Change in LV ejection fraction (▲LVEF = LVEF1− LVEF) median	2.64 ± 4.8 3 (−14 to 15)	0.03 ± 5 −1 (−11 to 15)	**0.003**
Change in end-systolic volume (▲ESV = ESV−ESV1) median	3.76 ± 11.6 3 (−23 to 34)	−1.86 ± 10.9 −1 (−28 to 14)	**0.03**
Change in end-diastolic volume (▲EDV = EDV−EDV1) median	4.5 ± 16.7 3 (−47 to 50)	−1.8 ± 15.8 −1 (−44 to 39)	**0.05**

Data are expressed as mean ± standard deviation or n (%) or median (range). Bold values highlight significant p values.
fQRS, fragmented QRS; LV, left ventricle.

3.2. Reperfusion parameters

Patients were stratified into two groups based on the degree of ΣSTR (a marker of reperfusion) obtained after primary PCI. Patients with ΣSTR less than 50% had a new onset or persistence of fQRS ($p = 0.004$), greater number of leads with fQRS ($p = 0.001$), more anterior localization of fQRS ($p = 0.01$), and more LV dysfunction at index hospitalization ($p = 0.02$) and at 30 days ($p = 0.004$) (Table 3).

3.3. Multivariate analysis

On univariate analysis, fQRS significantly predicted LV dysfunction and lower LVEF at 30 days. However, after adjusting for other confounding factors, fQRS did not significantly predict the change in LV function (▲LVEF) at short-term follow-up [adjusted coefficient, 95% confidence interval (CI): −2.45 (−5.44 to −0.53p = 0.10] (Table 4). In relation to myocardial reperfusion as assessed by ΣSTR,

Table 3
Analysis of myocardial reperfusion by ΣSTR after PCI with relation to study parameters.

Post-PCI	ΣSTR		
Parameter	<50% ($n = 26$)	≥50% ($n = 77$)	p value
Presence of fQRS at admission	7 (27%)	20 (26%)	0.92
Presence of fQRS (post-PCI)	21 (80.7%)	37 (48.1%)	**0.004**
Number of fQRS (pre-PCI)	0.65 ± 1.2	0.62 ± 1.1	0.94
Number of fQRS (post-PCI)	2.5 ± 1.8	1.2 ± 1.4	**0.001**
Delta QRS time (ms)	−3.5 ± 15.5	−4.9 ± 11.9	0.74
Total ST elevation on pre-PCI ECG (mm)	10.9 ± 5.9	11.8 ± 9.9	0.64
Total ST elevation on post-PCI ECG (mm)	6.9 ± 3.7	3.7 ± 3.6	**<0.001**
Localization fQRS (%)			
Anterior	18 (78.3%)	17 (41.5%)	**0.01**
Lateral	0 (0%)	1 (2.4%)	
Inferior	5 (21.8%)	23 (56.1%)	
GPI therapy	13 (50%)	34 (44.2%)	0.60
LV ejection fraction (%) (post-PCI)	41.8 ± 6.96	45.7 ± 7.7	**0.02**
LV ejection fraction (%) at 1 month	42.4 ± 8.2	48 ± 8.6	**0.004**

Data are expressed as mean ± standard deviation or n (%) or median (range). Bold values highlight significant p values.
ECG, electrocardiogram; fQRS, fragmented QRS;GPI, glycoprotein inhibitor; LV, left ventricle; ΣSTR, percent of total ST-segment resolution; PCI, percutaneous intervention.

Table 4
Regression analysis for change in LV function (▲LVEF = LVEF1−LVEF).

Variable	Adjusted coefficient (95% CI)	*p* value
Persistent fQRS	−2.45 (−5.44 to 0.53)	0.10
Presence of Q waves	−1.64 (−4.82 to 1.53)	0.30
QRS duration post-PCI	−0.03 (−0.13 to 0.06)	0.48
Delta QRS time	−0.008 (−0.12 to 0.10)	0.87
Presence of fQRS ≥ 3 leads (post-PCI)	−0.03 (−2.78 to 2.73)	0.98
ST resolution (%)	−0.0003 (−0.07 to 0.06)	0.99
Beta-blocker therapy	1.98 (−8.13 to 12.1)	0.69

CI, confidence interval; fQRS, fragmented QRS; PCI, percutaneous intervention.

Table 5
Adjusted odd ratios of study parameters for prediction of myocardial reperfusion (ST resolution <50%).

Variable	Adjusted odds ratio (95% CI)	*p* value
QRS duration post-PCI	0.974 (0.929−1.021)	0.27
ST elevation (pre-PCI)	0.954 (0.889−1.023)	0.18
Delta QRS	1.013 (0.962−1.066)	0.60
Persistent fQRS	4.265 (1.034−17.58)	**0.04**
Presence of fQRS ≥ 3 leads (post-PCI)	1.920 (0.505−7.302)	0.33

CI, confidence interval; fQRS, fragmented QRS; PCI, percutaneous intervention. Bold value highlights significant *p* values.

new onset or persistent fQRS is significantly associated with imperfect ST-segment resolution (<50%) on multivariate analysis [adjusted odds ratio, 95% CI: 4.265 (1.034−17.58), p = 0.04] (Table 5).

4. Discussion

Our study demonstrated that persistence of fQRS in acute STEMI patients belonging to Killip class I and II who underwent revascularization did not significantly predict the occurrence of MACE and LV dysfunction on short-term follow-up of 30 days.

fQRS originates from abnormal ventricular depolarization due to nonhomogeneous electrical activation of ischemic and/or injured ventricular myocardium.[1,5] fQRS occurred in 61.5% of the study population, of which 42.2% had fQRS at admission and 57.8% developed after primary PCI. Almost all of them developed fQRS within 48 h after primary PCI.

It has been reported that fQRS was significantly associated with in-hospital adverse cardiovascular events and long-term mortality in patients with STEMI.[6–10] Our study differed from other studies as it included STEMI patients belonging only to Killip class I and II.[18] In this low-risk cohort with acute short-term mortality of 1%, fQRS did not predict MACE and LV dysfunction in patients with STEMI. This lower cardiac event rate can be attributed to relatively younger STEMI population (mean age: 53 years) with 94.2% being first STEMI, early primary revascularization with shorter median angina to balloon duration of 5.5 h (range: 1−22 h) and 93.2% patients attaining TIMI ≥ 2 flow in culprit vessel, lower incidence of triple-vessel disease (11.7%), and fQRS resolving in 26% patients indicating better myocardial revascularization. In contrast, previous outcome studies involving fQRS had sicker patients belonging to Killip class III and IV, patients with severe coronary artery disease, much larger infarct size, and longer duration of follow-up.[6–10]

In patients with STEMI, prolonged QRS time was associated with increased long-term mortality due to increased incidence of heart failure, arrhythmia, and ischemia.[7–19] In our study, fQRS was related to prolonged QRS duration after PCI (p < 0.001) with lesser delta QRS time ($p = 0.01$). This observed relationship indicates myocardial conduction delay caused by acute ischemia leading to fragmentation of QRS and prolongation of QRS complex.

Early reperfusion therapy, which could prevent necrosis of the ischemic myocardium and improve prognosis, is the preferred treatment option for STEMI. We used ΣSTR to evaluate the effect of reperfusion therapy. Despite TIMI 3 flow after reperfusion therapy, there were 19.8% of patients with ΣSTR <50% (imperfect ST-segment resolution) in this cohort. This can be explained by "no-reflow phenomenon" due to microcirculation embolism, microvascular spasm, microcirculation reperfusion injury, and microvascular stunning.[20] ST-segment resolution which is dependent on microcirculation reperfusion can be regarded as an indicator of myocardial reperfusion. Imperfect ST-segment resolution after PCI was independently associated with cardiac dysfunction, cardiac death, and short- and long-term clinical prognosis in patients with STEMI.[21] Recent studies have negatively correlated fQRS with reperfusion parameters such as ΣSTR[12,13] and myocardial blush grade[11] in patients with STEMI. Similarly, we have found that new onset or persistent fQRS was significantly associated with impaired myocardial reperfusion as estimated by ΣSTR after PCI in our cohort. More number of leads with fQRS and anterior localization of fQRS were associated with inadequate microvascular myocardial reperfusion as well. This may be attributed to the association of fQRS with C-reactive protein levels (systemic inflammation) in patients with ACS and the inflammatory response mediated by oxygen-free radicals causing microvascular injury and subsequently no-reflow phenomenon.[13] Even in our relatively lower risk cohort, patients with fQRS were at higher risk of inadequate myocardial reperfusion and subsequent LV dysfunction which persisted on follow-up.

Our study had few limitations. This is a single-center study involving relatively smaller sample size and shorter duration of follow-up. Almost all patients underwent primary PCI in our study, which may not reflect true Indian scenario. In this study, we did not investigate myocardial blush grade during coronary angiography for assessment of myocardial reperfusion.

5. Conclusion

This study demonstrated that fQRS did not predict MACE and LV dysfunction in acute STEMI patients of Killip class I and II during short-term follow-up of 30 days. However, fQRS independently predicted impaired microvascular myocardial reperfusion as assessed by ΣSTR.

Conflict of interests

All authors have none to declare.

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What is already known?
- Recent studies have shown that fQRS (ECG parameter) may be of significant prognostic value in STEMI patients undergoing reperfusion therapy.

What we add?
- In our study, fQRS did not predict MACE and LV dysfunction in acute STEMI patients belonging to Killip class I and II on short-term follow-up.
- However, fQRS independently predicted impaired myocardial reperfusion as assessed by ΣSTR

Appendix A. Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.ihj.2018.07.014.

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