Original Article

Acute stemi presenting with fragmented QRS complex: A small cohort study from North India

Kunal Mahajan[1*], Gunjan Gupta[2], Virender Katyal[3], Vivek Bansal[4], Archana Meena[5], Hibu Habung[6]

{[1]Senior Resident, Department of Cardiology} {[2]Junior Resident, Department of ENT} Indira Gandhi Medical College, Shimla, Himachal Pradesh, INDIA.
{[3]Professor, Department of Medicine} {[4,5,6]Senior Resident, Department of Medicine} PGIMS Rohtak, INDIA.
Email: kunalmahajan442@gmail.com

Abstract

Background: Q waves on a 12-lead ECG are markers of a prior myocardial infarction (MI). However, they may regress or even disappear over time, and there is no specific ECG sign of a non–Q-wave MI. Fragmented QRS complexes (fQRSs), which include various RSR' patterns, without a typical bundle-branch block are markers of altered ventricular depolarization owing to a prior myocardial scar. We postulated that the presence of fQRS might be associated with a poorer outcome in patients of acute acute ST Elevation Myocardial Infarction (STEMI). **Methods and Results:** The study included 255 consecutive patients of acute ST Elevation Myocardial Infarction. After excluding 31 patients, the remaining 214 patients were divided into two groups, one consisting of patients who developed fragmented QRS during hospital stay (GROUP A with 139 patients) and the other group consisting of those who did not(GROUP B with 75 patients). In comparison Q wave appeared in 137 patients (64.01%). In 77 patients who did not develop a Q wave on ECG post MI, fragmented QRS developed in 57 of them (74 %), thus making it a valuable tool in identifying a non-Q wave MI. Significantly increased levels of cardiac biomarkers on presentation and increased rates of post MI angina, congestive heart failure, and major tachyarrhythmias were observed up to 30 days of follow up in patients of group A as compared to group B patients. **Conclusion:** This study of 255 patients highlighted the usefulness of fragmented QRS in identifying patients at higher cardiac risk with larger areas of ischemic jeopardized or necrotic myocardium, and it can provide very useful information in the risk stratification of acute STEMI patients. It also helps to identify the evidence of infarction in patients without a Q wave on surface ECG. These all features make fragmented QRS an area to explore in the field of electrocardiography in the near future in large randomized clinical trials.
Keywords: ECG, Fragmented QRS, Q wave, STEMI.

[*]**Address for Correspondence:**
Dr. Kunal Mahajan, Department of Cardiology, Indira Gandhi Medical College, Shimla-171001, Himachal Pradesh, INDIA.
Email: kunalmahajan442@gmail.com
Received Date: 18/11/2015 Revised Date: 18/12/2015 Accepted Date: 18/01/2016

Access this article online

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Volume 6
Issue 1

INTRODUCTION

ST Elevation myocardial infarction represents the most lethal form of acute coronary syndrome, one in which a completely occlusive thrombus results in total cessation of coronary blood flow in the territory of the occluded artery and the resultant ST-segment elevation on the ECG.[1-2] This clinical syndrome should be distinguished from non–ST segment elevation MI, in which the blockage of coronary flow is incomplete and for which different acute therapies are appropriate. There are several stigmas on the resting surface electrocardiogram that are indicators of past myocardial injury. Broad QRS pattern with bundle branch block, Q waves, persistent ST elevation are some of those. The presence of pathological Q waves on the 12-lead ECG signifies a prior transmural myocardial infarction (MI).[3-5] However, the Q wave may regress or even disappear over time in as many as 25% to 63% of patients with a history of a Q-wave MI by ECG.[6-7] Furthermore, there is no established ECG sign for a remote non–Q-wave MI. This limits our ability to detect the presence of a myocardial scar by ECG in as many as two thirds of patients with a documented Q-wave or non–Q-wave MI.[8] Therefore, clinicians have to depend on various noninvasive and invasive studies, such as echocardiography, nuclear imaging, or cardiac

catheterization, to confirm the presence of obstructive coronary artery disease (CAD). Fragmented QRS complex is a lesser known entity. This marker of myocardial injury may often be the only electrocardiographic marker in patients with non-Q myocardial infarction and in patients with resolved Q wave. Fragmented QRS complexes (fQRS) are defined as various RSR' patterns with or without Q waves on a 12-lead resting ECG with duration less than 120 msec. Various RSR' patterns include an additional R wave (R') or notching in the nadir of the S wave, or the presence of >1 R' (fragmentation) in 2 anatomically contiguous leads, corresponding to a major coronary artery territory. Complete and incomplete bundle branch block are excluded from the definition of fragmented QRS.[3] Anterior fQRS is defined by the presence of fQRS in ≥2 contiguous anterior leads (V1 to V5) and is assigned to represent the left anterior descending coronary artery territory or the anterior left ventricular wall. Lateral fQRS is defined by the presence of fQRS in ≥2 contiguous lateral leads (I, aVL, and V6) and is assigned to represent the left circumflex artery territory or the posterolateral left ventricular wall. Inferior fQRS is defined by the presence of fQRS in ≥2 contiguous inferior leads (II, III, and aVF) and is assigned to represent the right coronary artery territory or the inferior left ventricular wall. Following diagram demonstrates various morphologies of fragmented QRS:

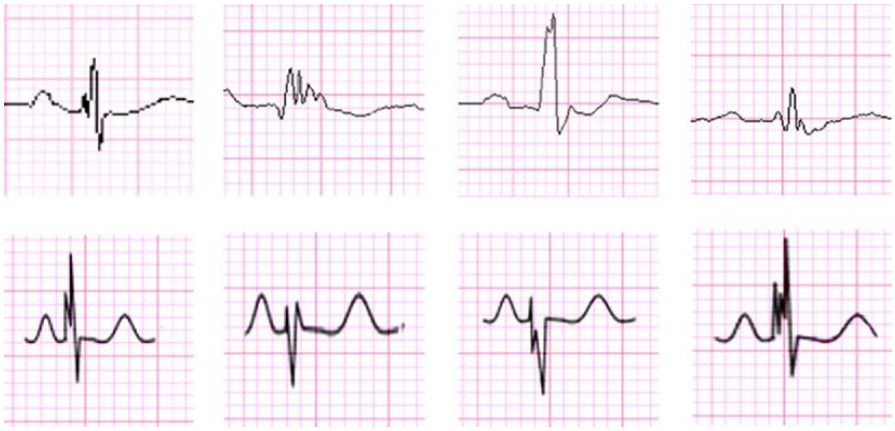

Figure 1: Various fragmented qrs morphologies

It has been observed in various studies[9] that fragmented QRS on the resting electrocardiogram has a moderate sensitivity (62.2%) and high specificity (up to 94%) in detecting ischemic heart disease. It is especially relevant in cases where the baseline electrocardiogram does not have a Q wave. The presence of a Q wave in addition to QRS fragmentation further augments the sensitivity of detecting ischemic heart disease up to 92.4%. Many studies have reiterated the significance of the fragmented QRS on patients with resolved Q waves and non-Q myocardial infarction. Das MK *et al*[10] using myocardial perfusion imaging have shown that fragmented QRS has a superior sensitivity and negative predictive value compared to Q waves in detecting myocardial scar; though there was a small compromise in specificity, especially in inferior wall myocardial necrosis. Fragmented QRS electrocardiogram is an independent predictor of left ventricular function. It is a marker of higher stress myocardial perfusion abnormalities and functional deterioration.[11] Recently studies have demonstrated that QRS fragmentation with or without Q waves is found to predict a higher mortality and recurrent cardiac events in patients with coronary artery disease, than either Q wave alone or resolved Q wave without QRS fragmentation.[12-13] It is postulated that fQRS represents myocardial scars, which may create a milieu for re-entry and malignant ventricular arrhythmias. Myocardial scar or ischemia is associated with a poor prognosis because of the risk for ventricular arrhythmias and heart failure. Hence fragmented QRS, though not extensively studied yet, is probably a reliable indicator of past myocardial ischemia and also suggests increased scar burden and poorer prognosis. The aims and objectives of the present study were to study the incidence of development of fragmented QRS complexes in ECGs of patients with acute STEMI and to assess its prognostic significance.

MATERIAL AND METHODS

All patients of STEMI who were admitted in ICCU during a period of six months w.e.f. 23 november, 2014 to 11 may, 2016 were considered for inclusion in the study. All patients were managed conservatively with or without thrombolysis, as facility of primary PCI was not available in our institute. Patients were treated according to the current STEMI guidelines.[14] Serial ECG recordings were done at regular intervals during first 5 days of

hospital stay, to look for the appearance of fragmented QRS. Based on the appearance of fragmented QRS in serial ECGs, incidence of development of fragmented QRS was calculated as per criteria mentioned below. Patients with fragmented QRS and those without fragmented QRS were then followed up to compare the cardiovascular prognosis and thereby assess the importance of fragmented QRS in influencing cardiovascular morbidity. Fragmented QRS complex on ECG was defined by the presence of various RSR' patterns with or without a Q wave, and included

- An additional R wave (R') or
- Notching of the R wave, or
- Notching of the down stroke or upstroke of the S wave, or
- The presence of >1 R',

In 2 contiguous leads, corresponding to a major coronary artery territory, with duration of QRS $\leq$ 120 msec.

Inclusion Criteria

1. Patient must have presented with typical ischemic symptoms (note that the main symptom instead of pain is sometimes dyspnea and acute left ventricular failure).
2. Following diagnostic criteria of acute STEMI must be fulfilled on ECG
    a. $\geq$2mm ST segment elevation at the J point in at least two chest leads or
    b. $\geq$1mm ST segment elevation in at least two limb leads or
    c. Reciprocal ST segment depression (V_2-V_3) due to posterior wall damage.
3. Elevated cardiac biomarkers.

Exclusion Criteria

1. Patients not giving informed consent.
2. Patients having bundle branch block on ECG (LBBB or RBBB).
3. Patients with fQRS in old ECGs.
4. Patients with pericardial and myocardial diseases.
5. Patients on digoxin, amiodarone which make ST-T changes difficult to interpret.
6. Patients with valvular heart disorders, infiltrative disorders and thyroid disorders.
7. Death of the patient within 48 hours of hospital admission.
8. Patients who left against medical advice / or their attendants wanted to take them to some other hospital.

Study methods

All the patients of acute STEMI coming to ICCU, who satisfied the already described inclusion and exclusion criteria, were provisionally included in the study. At the time of ICCU admission, 12 lead ECG was done and blood samples were tested for blood urea, blood sugar, serum sodium, serum potassium, and CPK-MB. Detailed clinical examination of the patients were done and entered in patient's proforma. Patient were treated as per the standard treatment protocol of acute STEMI Serial ECGs of the patients were done 8 hourly for first two days, and then once daily till discharge, and were evaluated for the appearance of fragmented QRS complex. Haemodynamic monitoring was done and KILLIP class was determined at the time of admission as follows:

KILLIP CLASS I = No clinical signs of heart failure
KILLIP CLASS II = crackles, S3 gallop and elevated jugular venous pressure
KILLIP CLASS III = frank pulmonary oedema
KILLIP CLASS IV= cardiogenic shock

Following investigations were sent on day 1 after admission.

- CPK-MB: 12 hrs and 24 hrs.
- Troponin I – 12-24 hours (By chemiluminescence assay)
- Blood sugar (fasting/post prandial)
- Complete lipid profile
- SGOT/PT
- ECG
- Chest X-ray PA view

Clinical profile of the patient during hospital stay was studied and observed for any complications of MI and thrombolysis:

a. Cardiogenic shock
b. Arrythmias
c. AV block
d. Pulmonary edema
e. Heart failure
f. Cardiac arrest
g. Recurrent myocardial ischemia
h. Myocardial reinfarction
i. Myocardial rupture
j. Cardiac tamponade
k. Pericarditis
l. Pericardial effusion
m. Mitral regurgitation
n. Thrombosis
o. Embolism
p. Electromechanical dissociation etc.

Wherever indicated echocardiography was done. The patients in whom fragmented qrs appeared on serial ecgs, were then taken into 'fragmented qrs study group', labelled as group a. Rest of the patients who do not develop fragmented qrs morphology on serial ecgs were taken into 'non fragmented qrs study group', labelled as group b. It was planned that inclusion of new cases in the study would continue untill

A. 100 cases with development of fragmented qrs were identified, or
B. A total of 200 provisional cases of acute stemi were taken up for study, whichever comes earlier.

Incidence of fragmented QRS was then calculated during first 5 days of hospital stay by the following formula : Incidence = Number of cases who develop fragmented QRS within first 5 days Total number of provisional cases included in the study

Follow up protocol

Patients were re-evaluated after one month and following evaluation was done:

1. Clinical assessment
2. ECG to evaluate QRS morphology and ST- T changes. Post MI ECG changes and any post MI complications were documented.
3. TREADMILL TEST (TMT) to assess residual ischemia and ventricular ectopy. TMT was fixed and conducted in 1-2 weeks time.
4. ECHOCARDIOGRAPHY was done wherever indicated.

The data of hospital stay, post MI ECG changes, and complications were analysed with an appropriate statistical method to reach a conclusion.

Statistical analysis

Continuous variables were given as mean ± standard deviation; categorical variables were defined as percentages. Continuous variables were compared by Student t-test and the chi-square test was used for the categorical variables between two groups. All tests with regards to significance were two tailed. Statistical significance was defined as $p<0.05$.

OBSERVATIONS

A Total of 255 patients of acute STEMI were enrolled in study. 41 patients were excluded as they did not meet the inclusion criteria (figure 2). Out of remaining 214 STEMI patients, fragmented QRS appeared in 139 patients during the hospital stay that varied from 3-5 days. These 139 patients were included in group A, while the remaining 75 were included in group B. Incidence of fragmented QRS in STEMI patients was calculated by dividing the total number of patients developing fragmented QRS during the hospital stay by the total number of patients in the study population. Thus in our study incidence of fragmented QRS in STEMI patients was calculated to be 64.9 %. Similarly the incidence of new Q wave was found to be 64.01%. Number of patients in whom fragmented QRS appeared on 1st day were 115 (82.73%). In 20 patients (14.39%) fragmented QRS appeared on the 2nd day while in 4 patients (2.88%), it appeared after 48 hours of symptom onset. Our study revealed that 97 % of patients with fragmented QRS developed it within 48 hours.

Fragmented QRS appeared in one of three myocardial territories of infarction on ECG (either anterior, inferior or lateral) in 82 patients (58.99%). While it appeared in two of three territories on ECG in 52 patients (37.41%), there were 5 patients in whom it appeared in all the three territories. When the data was analyzed for the most commonly involved ECG leads to develop fragmentation of QRS, it was seen that inferior leads (II, III, aVf) were the most commonly involved (76.2%). While 53.9% patients had involvement of anterior leads (V1-V6), lateral leads (I, aVl) were involved in 10.8% patients. There were 70 patients (50.3%) in whom fragmented QRS appeared in 2 leads while in 69 patients (49.7%) it was present in 3 or more leads. Mean age of the study population was 53.54 ± 13.74 years. When the mean ages of two study groups was compared, it was observed that the mean age was marginally higher in group A as compared to group B, although the difference did not reach statistical significance (p-value NS). The total number of male patients in study were 177 while females were 37, thus the male: female ratio was 4.8: 1. Thus males out-numbered the females in our study, which is justified as males are more prone to AMI as compared to females. The total number of males in group A was 117 (84.2%) and in group B was 60 (80%). In terms of sex ratio, group A had more males (sex ratio of 5.3:1) in comparison to group B (sex ratio of 4:1). Both the groups were equally matched in terms of risk factors of diabetes, hypertension, smoking and family history. They were also equally matched in terms of presenting complaints, and hemodynamic parameters at time of presentation and location of infarct based on ECG changes. (table 1)

Cardiac biomarkers in group A were higher than that of group B ($p <0.01$). This suggests greater myocardial necrosis in patients with fragmented QRS. Serum SGOT of group A was 128.62±57.27 U/mL which was significantly higher ($p <0.01$) than that of group B (108.68±31.11 U/mL). Total Leucocyte count was also significantly higher in group A. Total number of major adverse cardiovascular cardiovascular events (MACE) which included death, cardiogenic shock, post MI angina, congestive heart failure and significant arrythmias, upto a period of one month was 178 (with one patient may be

having more than one event), and the number was much higher in group A (figure 3). The occurrence of MACE was higher in group both in first 5 days post MI as well as 5-30 days after MI (table 1)

Table 1: Composite table of the study results

	GROUP A (n=139)	GROUP B (n=75)	p-value
PRESENTATION			
a) Age(years)	*54.43±14.15*	51.86±12.84	NS
b) Male Sex (%)	*84.2 %*	80 %	NS
c) Chest pain (%)	*71.2 %*	70.7 %	NS
d) KILLIP class II-IV	*31.7 %*	22.7%	NS
RISK FACTORS			
a) Smoking	*104*	52	NS
b) Diabetes	*44*	24	NS
c) Hypertension	*31*	20	NS
d) Family history	*23*	15	NS
INFARCT LOCATION AS PER ST ELEVATION			
a) Anterior	61.2 %	60 %	NS
b) Inferior	30.2 %	36 %	NS
c) Lateral	8.6 %	4 %	NS
LOCATION OF FRAGMENTED QRS			
a) Anterior	53.9 %		
b) Inferior	76.2 %	NA	NA
c) Lateral	10.8 %		
NUMBER OF TERRITORIES ON ECG INVOLVED BY FRAGMENTED QRS			
a) One	58.9 %		
b) Two	37.4 %	NA	NA
c) Three	3.5 %		
Q WAVE ON ECG			
a) Present	82	55	<0.05
b) Absent	57	20	<0.05
CARDIAC BIOMARKERS			
a) CPK-MB (U/L) at 24 hours	244.7±188.9	171.7±169.9	<0.01
b) TROP-I (ng/ml) at 24 hours	31.64±19.04	23.81±18.4	<0.01
TOTAL LEUCOCYTE COUNT (10^3/mm^3)	10502.1±2195.4	9522.7±2262.7	<0.01
SGOT (U/L)	*128.6±57.3*	108.7±31.11	<0.01
TOTAL NUMBER OF PATIENTS WITH ≥1 MACE			
a) 0-30 days	75	26	<0.01
b) 0-5 days	58	19	<0.05
c) 5-30 days	44	8	<0.001
POST MI ANGINA			
a) 0-30 days	52	10	<0.001
b)0-5 days	40	8	<0.01
c) 5-30 days	39	6	<0.001
CONGESTIVE HEART FAILURE			
a) 0-30 days	37	9	<0.05
b) 0-5 days	36	9	<0.05
c) 5-30 days	1	0	NS

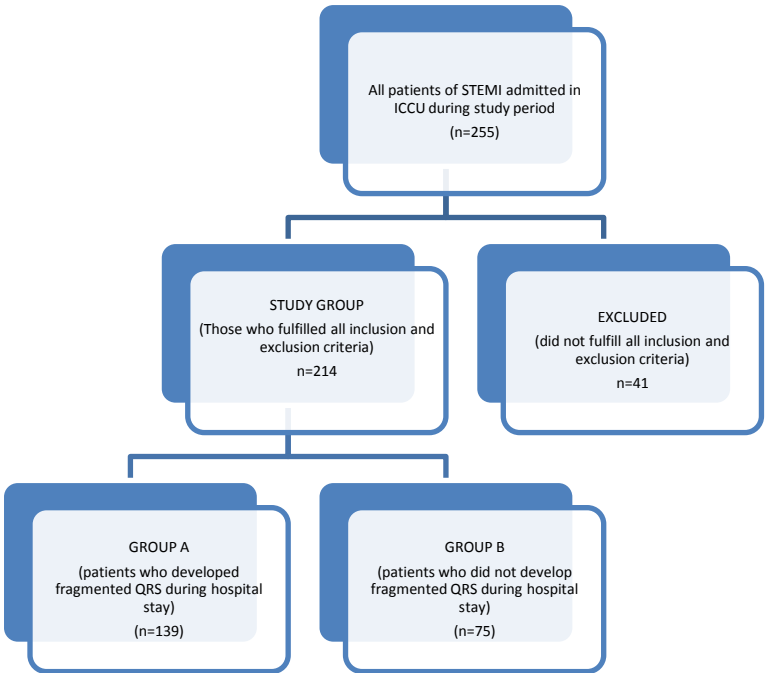

Figure 2: Study Design

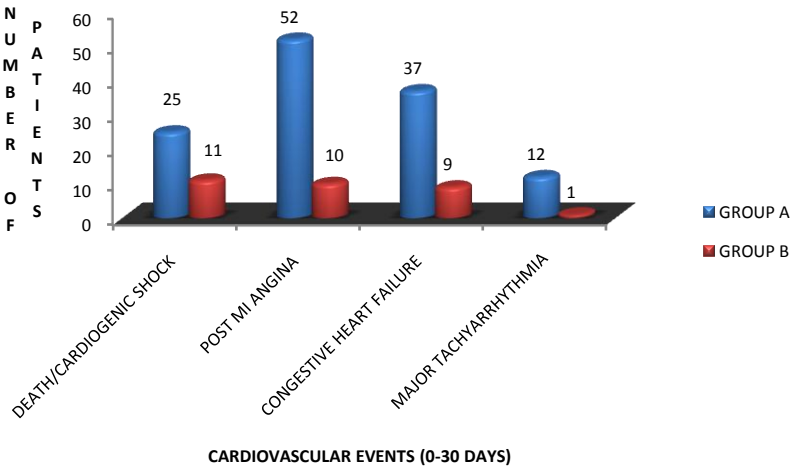

Figure 3: Comparison of major cardiovascular events (0-30 days)

DISCUSSION

Fragmentation of QRS (fQRS) complex is an easily evaluated non-invasive electrocardiographic parameter. Presence of fQRS has been associated with alternation of myocardial activation due to myocardial scar and myocardial fibrosis. The importance of fQRS complexes was first suggested by Das in 2006[3]. The exact mechanism of fragmentation of the QRS complex in the 12-lead surface ECG is still unclear. Previous studies have shown[15-16] that the post-infarction scar tissue morphology correlated well with the patterns of the fQRS complex. The presence of myocardial scar and/or ischemia results in the inhomogeneous activation of the ventricles which alters ventricular depolarization, which probably represents fragmentation in the QRS complex on the surface 12-lead ECG.[(17-18)] These studies suggest that different fragmented QRS patterns result from shifting of the QRS vector during depolarization of areas around the myocardial scar or ischemic myocardium, depending on the extent of injury and its location in the ventricles[15]. The present study was done to evaluate the number of patients who develop fragmented QRS complexes on surface ECGs in the immediate period following ST elevation myocardial infarction and also to

find out its significance in predicting the outcome of STEMI patients. Patients were divided into two groups based on the development of fragmented QRS and were then compared during hospital stay after admission to ICCU and up to 30 days thereafter. All patients were managed on standard protocol of STEMI. In the past there have been very few studies which evaluated the significance of development of fragmented QRS in STEMI patients especially in the immediate period following the myocardial infarction. Studies done on Indian population are largely missing. This study revealed very important observations regarding the development of this new electrocardiographic marker of myocardial necrosis which should be taken into consideration while identifying or risk stratifying any patient of STEMI. Mean CPK-MB and TROP-I of group A were significantly higher ($p < 0.01$) than group B (Table XIV). In the previous study by Kocaman *et al*,[19] patients with fragmented QRS had higher CPK-MB and TROP-I values. Similarly in the study by Cetin *et al* [(20)], fragmented QRS was associated with higher cardiac biomarker values. It has been considered that fragmented QRS results from the presence of significant myocardial necrosis, with islands of viable myocardial tissue interspersed in abundant fibrous tissue in coronary artery disease. It would be expected that a larger number of fragmented QRS must be associated with a larger size of myocardial scars.[3] Also it is an established fact that the rise in cardiac enzymes during myocardial infarction correlates with the extent of myocardial necrosis and infarct size [(21-22)]. These findings suggested that more cardiac necrosis occurred in those who developed fragmented QRS as compared to those who did not. Similarly mean SGOT level was higher in fragmented QRS group. Similar results were seen in the studies done by both Cetin *et al*[20] and Kocaman *et al*[19]. This is probably related to increased myocardial necrosis which contributes to increased SGOT. However mean SGPT of group A was lower than that of group B. Previous studies have never compared SGPT among patients with fragmented QRS and non-fragmented QRS complexes. Till date no study has evaluated the immediate outcome of fragmented QRS in STEMI patients. So no data is available to compare our results. Studies on long term outcome are also very few and that too also for myocardial infarction as a whole including NSTEMI as well. In a study by Ari *et al*[23] a cohort of 85 patients with no prior history of CAD underwent primary percutaneous coronary intervention (PCI) for acute MI, 48 hour post-procedure ECG were analyzed for the presence of fragmented QRS and predictive value for major adverse cardiac events (MACE) was assessed. There was no clear definition of MACE in that study. During follow-up of 6.6 ± 2.3 years, there was significantly higher frequency of MACE among the patients with fragmented QRS as compared to those who did not have fragmented QRS (29.4% vs 5.9%; $p < 0.01$). It was concluded that since fragmented QRS represents myocardial scars, its presence post STEMI was associated with higher event rate in STEMI patients. In our study, It was observed that patients with fragmented QRS had increased number of major adverse cardiovascular events (MACE) than patients without fragmented QRS during hospital stay (0-5 days) as well as up to 1 month of follow up (Table 1). The cardiovascular events that were more common were congestive heart failure, post MI angina, and major tachyarrhythmias. For these events difference among the two groups was significant. Death and cardiogenic shock were also higher in group A (p-value NS). Probable explanation is that fragmented QRS signifies underlying myocardial scar and/or ischemia in patients with coronary artery disease, which will cause inhomogenous activation of the ventricles and also creates a milieu for re-entry and will thus increase the risk for ventricular arrhythmias and heart failure.[3,10-11] At one month post discharge patients were made to perform treadmill test which could be used as a marker of residual ischemia. Group A patients had higher percentage of positive results (42.9%) compared to group B patients (32%) (p-value NS).

CONCLUSION

Fragmented QRS, which may be derived from the effects of the individual risk factors, MI, and perfusion related factors on myocardial electrical conduction at cellular level, can represent increased cardiac risk by different causative mechanisms in patients with STEMI. Twelve-lead surface ECG, which is an inexpensive, non-invasive, and easily apprehensible method, is presently the gold standard in differential diagnosis, determining treatment methods, and performing risk stratification of STEMI. This study of 255 patients highlighted the usefulness of fragmented QRS in identifying patients at higher cardiac risk with larger areas of ischemic jeopardized or necrotic myocardium, and it can provide very useful information in the risk stratification of acute STEMI patients. It also helps to identify the evidence of infarction in patients without a Q wave on surface ECG. These all features make fragmented QRS an area to explore in the field of electrocardiography in the near future in large randomized clinical trials.

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Source of Support: None Declared
Conflict of Interest: None Declared