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Original Article

Prognostic significance of the distortion of terminal portion of QRS complex on admission electrocardiogram in ST segment elevation myocardial infarction



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ABSTRACT

Background: ECG on admission has been used in predicting prognosis and risk stratification in ST segment elevation acute myocardial infarction (STEMI).

Objective: To analyze the admission ECG in STEMI based on abnormality observed in terminal portion of QRS and its correlation to hospital mortality.

Method: 160 consecutive patients of STEMI were classified into subjects without (Group I) and with distortion of terminal QRS (Group II), Pattern A – Emergence of J point at $\geq 50\%$ of the R wave amplitude in leads with qR configuration or Pattern B – Absence of the S waves, in leads with Rs configuration in two consecutive leads.

Results: Out of 160 patients of STEMI, 69 (43.1%) had distortion of QRS. There were 13 deaths (8.1%). Hospital mortality was found to be significantly more in subjects with distortion than those without (15.9% V/S 2.1%, $p < 0.001$). Patients with QRS distortion tended to have larger infarction as assessed by Killip class on admission ($p < 0.05$), anterior location of MI ($p < 0.01$) and presence of significant Q waves in leads with ST segment elevation ($p < 0.0001$).

With multiple logistic regression analysis using hospital mortality as a dependent variable and all studied risk factors as independent variables, QRS distortion on admission ECG was the only variable found to be statistically significant (Adjusted OR = 7.161, $p < 0.05$).

Conclusion: ECG on admission is a simple, cheap, universally available investigation that can predict the short term prognosis in STEMI and would help in deciding which patients should go for other myocardial revascularization procedures.

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1. Introduction

Early risk stratification of patients presenting with STEMI is commonly done using various risk scores based primarily on

the clinical presentation and evidence of left ventricular failure. Risk assessment needs to be done shortly after admission, when only the history, physical examination and the ECG are available.¹ The role of ECG in diagnosis and

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prognostication of AMI is well established.² In the early 1970s, it was found by epicardial and precordial mapping that the magnitude of ST segment elevation is a reflection of the extent of myocardial injury.^{3–6} As a result, ST segment elevation was used to define subsets of patients who can benefit most from early thrombolysis^{2,7,8} and even monitor the effects of reperfusion therapy.^{9–11} However, the magnitude of ST elevation is influenced not only by the extent and severity of the ischemia, but also by variation of the shape and size of the chest and by localization of infarction.¹² Birnbaum et al.^{13,14} observed that, on admission ECG distortion of the terminal portion of the QRS complex was associated with larger infarct size and increased mortality.

The purpose of this study was to analyze admission ECG in patients of STEMI based on terminal portion of QRS and its correlation to hospital mortality.

2. Methods

In this prospective study, we studied 160 consecutive patients of STEMI eligible for thrombolysis, admitted in intensive coronary care unit within 12 h of onset of chest pain which lasted for at least 20 min. STEMI was diagnosed when ECG showed ST elevation of 0.1 mv in two or more limb leads or 0.2 mv in two or more consecutive chest leads with positive T waves in leads with ST segment elevation with increase in cardiac enzymes. Patients with left bundle branch block (LBBB), ventricular rhythm, ventricular pacing or negative T waves in leads demonstrating ST segment elevation were excluded.¹⁴ Clinical assessment was done and various risk factors were noted. All patients received streptokinase 1.5 million units infusion over a period of 1 h and standard medications like beta blockers, ACEI/ARBS, statins and antiplatelets as per the discretion of the treating physician. Time from onset of chest pain and administration of thrombolytic therapy was recorded. The use of medications was not analyzed.

2.1. Electrocardiographic evaluation

All admission ECGs were analyzed by three experienced physicians. In case of uncertain ECG classification, two other physicians were consulted. The investigators analyzing ECGs were blinded to clinical data of the patient. The number of leads in the admission ECG with ST segment elevation and the presence of abnormal Q wave in leads showing ST elevation were recorded. The site of infarction was determined. Abnormal Q waves were defined as follows^{8,14}: 1) a Q wave >25% of the R wave or with a width 0.04 s in limb leads, 2) a QR complex in lead V₁ (an isolated QS complex in lead V₁ was considered), 3) any Q wave in lead V₂ or V₃, 4) a Q wave in lead V₄ with a duration 0.02 s and 0.1 mv or deeper than the Q wave in lead V₅, 5) a Q wave in lead V₅ or V₆ 25% of the R wave with a duration of 0.04 s. The patients were classified into two electrocardiographic patterns based on terminal portion of the QRS–ST pattern.¹⁴ Group I – Those with tall symmetric T waves and ST elevation in two or more adjacent leads without major changes in the configuration of the terminal portion of the QRS complex. Group II – Those with tall symmetric T waves and ST elevation and distortion of the

terminal portion of the QRS complex in two or more consecutive leads. Pattern A – Emergence of the J point at a level above the lower half of the R wave (at $\geq 50\%$ of the R wave amplitude as measured from isoelectric line) in leads with qR configuration. Pattern B – Absence of S waves in leads with Rs configuration (leads without Q waves) (Fig. 1).

2.2. Endpoint

Hospital mortality was recorded in the two groups.

2.3. Statistical analysis

The data was recorded and analyzed using statistical package for social sciences (SPSS) version 12. Mean values $\pm$ SE were calculated for continuous variables and absolute and relative frequencies were measured for discrete variables. For continuous variables difference between groups was analyzed for statistical significance by two-tailed student's 't' test. The chi-square test was used to compare difference between discrete variables. Logistic regression analysis included hospital mortality as the dependent variable and age, gender, history of diabetes mellitus, hypertension, prior coronary artery disease, smoking status, time from onset of symptoms to thrombolytic therapy, heart rate, Killip class on admission, location of AMI, number of leads with ST segment elevation, presence of Q waves on admission and initial QRS pattern on ECG as dependent variables. Odds ratio (OR) and 95% confidence intervals (CI) were calculated. A *p* value <0.05 was considered statistically significant.

3. Results

Out of 160 patients of STEMI studied 123 (76.7%) were males and 37 (23.3%) were females with an M:F ratio of 3.3:1. Age of

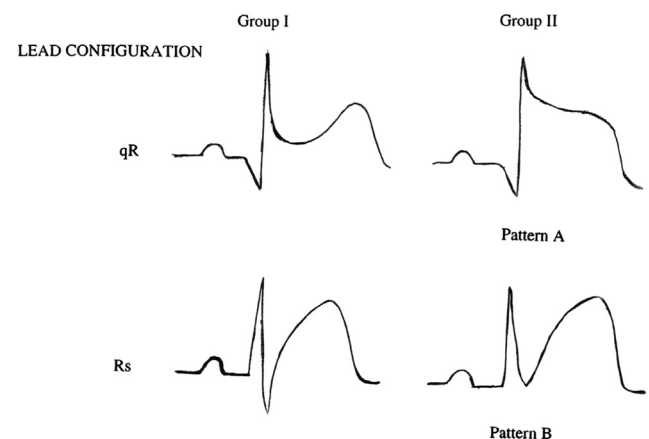


Fig. 1 – Group I: No QRS distortion. J point emerges below 50% of the R wave amplitude. Group II: Terminal QRS distortion. Pattern A – emergence of J point $\geq 50\%$ of the R wave amplitude in lead with qR configuration or Pattern B – absence of the S wave in lead with Rs configuration.

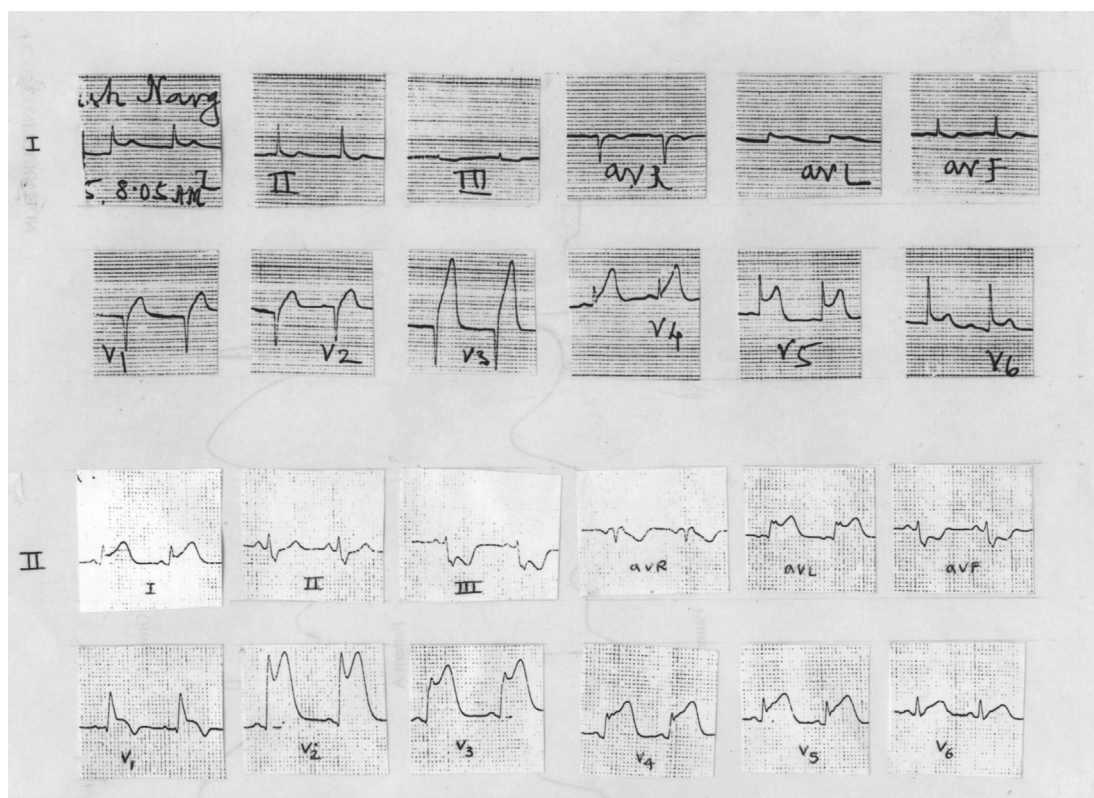


Fig. 2 – Admission electrocardiograms of 2 patients with anterior wall AMI. (I) is an example of Group I – i.e. without QRS distortion. Despite having a high degree of ST elevation, the S waves in leads V₁, V₂, V₃ are preserved and the J points emerge at <50% of the R wave amplitude in leads V₄, V₅, and V₆ (II) is an example of Group II – i.e. with QRS distortion. Despite having a high degree of ST elevation, the S waves in leads V₁, V₂, V₃, V₄ and V₅ are absent and the J points in leads V₂, V₃, and V₄ emerge at ≥50% of the R wave amplitude.

the patients ranged from 25 years to 110 years with a mean of 53.4 ± 12.3 years. Out of 160 patients 91 (56.9%) had no QRS distortion (Group I), while 69 (43.1%) showed evidence of QRS distortion (Group II) (Figs. 2 and 3).

There were 13 deaths (8.1%). The cause of death was cardiogenic shock in 5, left ventricular failure (LVF) in 4 and arrhythmias in 4 cases. Out of 10 patients with systolic BP <90 mmHg, 5 (50%) died. Out of 150 patients with systolic BP ≥90 mmHg, 8 (5.3%) died ($p < 0.001$).

Out of 132 patients with Killip class <II on admission, 3 (2.3%) died while out of 28 with Killip class ≥II on admission, 10 (35.7%) died ($p < 0.05$).

Out of 117 patients who were thrombolysed within 6 h of onset of chest pain, 6 (5.1%) died. Of the 43 patients who received thrombolysis ≥6 h after onset of chest pain, 7 (16.2%) died ($p < 0.05$).

Risk factors and time interval from onset of chest pain to thrombolysis were comparable in both the groups. Patients with QRS distortion (Group II) tended to have larger infarction as assessed by Killip class ≥II on admission ($p < 0.05$), anterior wall location of MI ($p < 0.01$) and presence of significant Q waves in leads with ST segment elevation ($p < 0.0001$) which is reflected in significant increase in hospital mortality (15.9% V/S 2.1%, $p < 0.05$) than those who had no QRS distortion (Table 1).

3.1. Multivariate analysis

Multiple logistic regression analysis using hospital mortality as a dependent variable and all studied risk factors as independent variables, QRS distortion on admission ECG was the only variable found to be statistically significant (OR = 7.161, $p < 0.05$) (Table 2).

4. Discussion

Clinical outcome after AMI largely depends on the final size of infarct.^{15–17} The latter depends on at least two factors: (i) the presence of residual blood supply to the infarct zone, either by antegrade flow in the infarct related artery or by collateral circulation^{18–20} and (ii) metabolic factors such as oxygen requirement of the myocardium and the presence of protective mechanisms.^{21–23} An ability to estimate the ischemic area at risk and the severity of ischemia immediately on admission may enable physicians to identify patients in whom reperfusion could not be expected to salvage myocardium and conversely, those who might benefit from reperfusion even if much time has elapsed from onset of symptoms.

The extent of myocardial infarct can be estimated by searching for symptoms and signs of heart failure and using

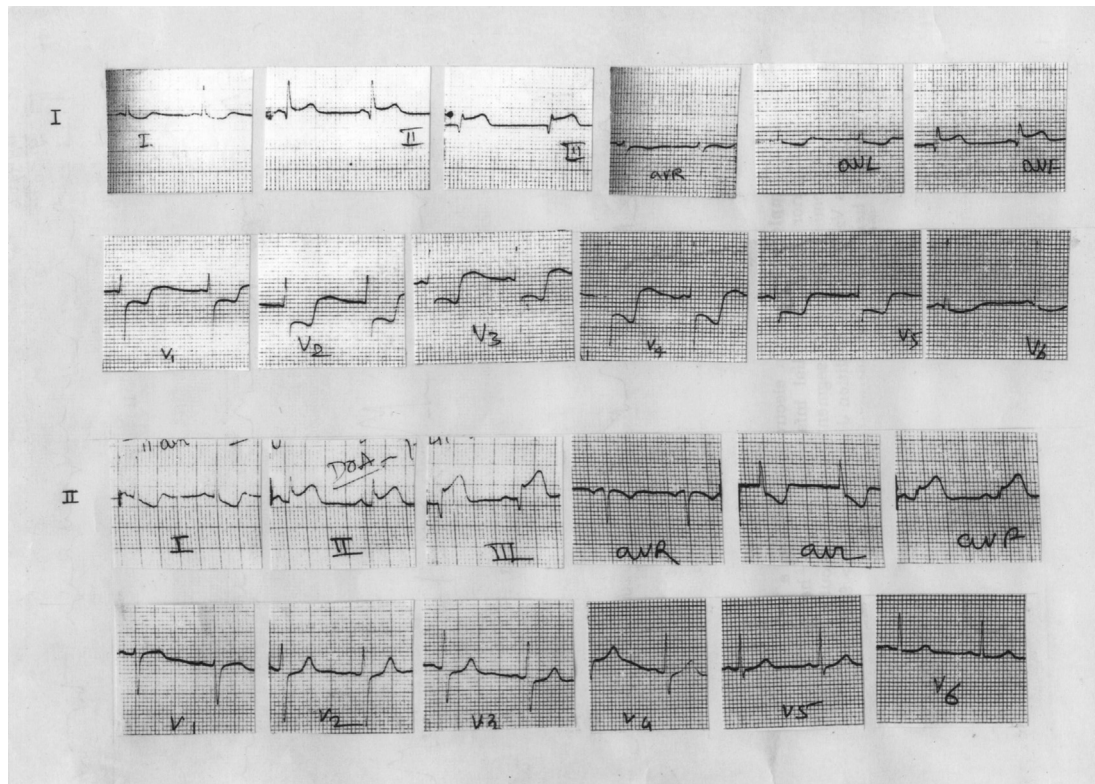


Fig. 3 – Admission electrocardiograms of 2 patients with inferior wall AMI (1) is an example of Group I – i.e. without QRS distortion. J points emerge at <50% of the R wave amplitude in leads II and avF (II) is an example of Group II – i.e. with QRS distortion. J points emerge at ≥50% of the R wave amplitude in leads III and avF.

echocardiography or vectorcardiography. Imaging methods increase delay in treatment and can neither measure the 'severity' of ischemia nor differentiate between viable myocardium and myocardium that already has infarcted. Although single photon emission computed tomography (SPECT) with 99mTc sestamibi can quantify residual flow to infarct area and the size of the area at risk, this method likewise is not only time consuming but also expensive and technically demanding.^{24–26}

Studies of the ability to estimate the area at risk or final infarct size by the admission ECG have conflicted results.^{23–25} All previous studies assessed the correlation between either the number of leads with or magnitude of ST segment elevation/deviation and infarct size. All these studies were based on the assumption that each lead represents the same amount of myocardium and that a similar size of ischemic area in different segment of the left ventricle will result in similar magnitude of ST deviation in the same number of

Table 1 – : Showing characteristics of 160 patients of AMI without QRS distortion (Group I) and with QRS distortion (Group II) on admission ECG.

Characteristics	Group I n = 91(56.87%)	Group II n = 69(43.12%)	p value
Age (mean)	53.4 ± 1.1	53.04 ± 1.6	0.09
Sex (males)	72 (79.1%)	51 (73.9%)	0.43
Diabetes mellitus	10 (10.9%)	9 (13%)	0.69
Hypertension	23 (25.2%)	11 (15.9%)	0.15
Prior CAD	12 (13.1%)	6 (8.6%)	0.37
Smokers	44 (48.3%)	33 (47.8%)	0.94
Killip class ≥II	11 (12%)	17 (24.6%)	0.03
Anterior location of AMI	41 (45%)	45 (65.2%)	<0.01
≥5 leads showing ST elevation	36 (39.5%)	33 (47.8%)	0.29
Heart rate on admission ECG ≥100/min	17 (18.6%)	12 (17.3%)	0.83
Mean heart rate/min	83.23 ± 1.8	84.86 ± 2.4	0.20
Time to thrombolysis ≥6 h	26 (28.5%)	27 (39.1%)	0.15
Q waves in leads with ST elevation	6 (6.5%)	30 (43.4%)	<0.0001
Hospital mortality	2 (2.1%)	11 (15.9%)	<0.001

Table 2 – : Showing multiple logistic regression analysis using hospital mortality as dependent variable.

Variable	Ex.(B) Odds ratio	95% CI for Exp.(B)		p value
		Lower	Upper	
Age ≥ 60 v/s <60	0.36	0.05	2.3	>0.05
Sex: Male v/s Female	0.23	0.03	1.3	>0.05
Hypertension: Yes v/s No	0.48	0.05	3.9	>0.05
Diabetes mellitus: Yes v/s No	3.14	0.58	17	>0.05
Prior coronary artery disease: Yes v/s No	0.72	0.04	11	>0.05
Smoking: Yes v/s No	0.84	0.10	6.4	>0.05
≥ 6 h for thrombolysis: Yes v/s No	1.85	0.37	9.1	>0.05
Heart rate: ≥ 100 v/s <100 /min	2.77	0.46	16.7	>0.05
Killip class: $\geq$ II v/s I	1.58	0.29	8.3	>0.05
Location of AMI: Ant. v/s Inf.	0.77	0.10	5.5	>0.05
≥ 5 leads with ST elevation: Yes v/s No	1.76	0.30	10.2	>0.05
Q wave on admission: Yes v/s No	1.31	0.26	6.5	>0.05
QRS distortion: Yes v/s No	7.16	1.11	45.8	<0.05

leads. However, not all myocardial regions are equally represented by 12 lead ECG. Furthermore, involvement of opposed regions may cancel or augment ST deviation.²⁷

Time interval between onset of chest pain and thrombolysis is an important factor.²⁸ Patients who received thrombolysis within 6 h of onset of chest pain had less mortality (5.1%) than those who were thrombolized after ≥ 6 h (16.2%). The difference observed was found to be significant ($p < 0.05$).

After AMI, changes in the terminal portion of the QRS complex may be detected in addition to changes in the ST segment and T waves. Birnbaum et al.^{27,29} found that patients with terminal QRS distortion on admission ECG in STEMI have worse prognosis, larger MIs and less benefit from thrombolysis.

Significant association between Killip class $\geq$ II on admission and in hospital mortality has been observed in various studies.^{30,31} In the present study, in hospital mortality was found to increase with increase in LV dysfunction represented by increase in Killip class on admission. The mortality was 2.3% with Killip class I, 15.8% with class II, 60% with class III and 100% with class IV ($p < 0.05$).

In the present study of 160 patients with STEMI, 91 (56.9%) had no QRS distortion (Group I) while 69 (43.1%) showed presence of QRS distortion (Group II). Gracia-Rubira et al.³⁰ observed QRS distortion in 20.48% patients. Mager et al.³² observed QRS distortion in 50% patients. Birnbaum et al.³¹ observed QRS distortion in 31% patients.

In the present study, there was no statistically significant difference between the two groups in the basic characteristics (Table 1). Patients with QRS distortion tended to have larger infarction as assessed by Killip class $\geq$ II on admission ($p < 0.05$), anterior location of MI ($p < 0.01$) and presence of significant Q waves in leads with ST segment elevation ($p < 0.0001$) which is reflected in increased hospital mortality (15.9% V/S 2.1%, $p < 0.05$) than those who had no QRS distortion.

On multivariate analysis using hospital mortality as a dependent variable and all studied risk factors as independent variables, QRS distortion on admission ECG was the only variable found to be statistically significant (OR = 7.161, $p < 0.05$) (Table 2).

Birnbaum et al.²⁹ observed that infarct size as assessed by creatine kinase release over 24 h and predischARGE sestamibi defect size was significantly higher in patients with QRS distortion than those without ($p < 0.003$ and <0.001 respectively). Birnbaum et al.²⁷ assessed final infarct size using predischARGE Selvester score in pattern of AMI with [QRS (+)] and without QRS distortion [QRS (-)]. The average Selvester score was 19.7 ± 9.9 for QRS (-) and 26.1 ± 10.4 for QRS (+) patients ($p < 0.001$).

During regional myocardial ischemia the duration of the conduction of the activation wave in Purkinje fiber is prolonged. Delayed conduction decreases the degree of cancellation increasing the R wave and decreasing the S wave amplitude in surface ECG.³³ The Purkinje system is less sensitive than the contracting myocytes to ischemia.³¹ Hence only a severe and prolonged ischemia that affects the Purkinje fibers would alter the terminal portion of the QRS complex.^{33,34}

Birnbaum et al.³⁵ have hypothesized that ischemia is 'more severe' and necrosis of the myocardial area at risk progresses more rapidly in patients with QRS distortion than those without. Whether the absence of 'terminal QRS distortion' is a sign of residual perfusion of the infarcted area by antegrade flow or collateral circulation is a question that is yet to be answered. Indeed in the study by Spekhorst et al.³⁶ no such changes in QRS complex were detected in patients with collateral circulation during angiography.

Lower rates of successful reperfusion in patients with 'QRS distortion' might explain the difference in infarct size between the groups. However, in the Thrombolysis In Myocardial Infarction (TIMI-4) trial⁴³ and the Global Utilization of Streptokinase and TPA (Alteplase) for Occluded coronary arteries (GUSTO-I) angiographic substudy,³¹ the proportion of patients who had TIMI grade 2 or 3 flow 90 min after thrombolysis did not differ as far as QRS distortion was concerned on admission ECG.³⁵

4.1. Limitations

Though Killip class may correlate with LV function in general, it cannot replace echocardiographic LVEF. A patient with

STEMI with large infarct may present with low systolic BP due to forward failure and still no pulmonary congestion and a low Killip class. Due to non-availability of round the clock expertise services for echocardiographic evaluation, LV function assessment was based on clinical assessment.

5. Conclusion

ECG on admission in STEMI is a simple, cheap, universally available investigation which can predict short term prognosis and would help in deciding which patients should go for myocardial revascularization procedures to higher centers after initial stabilization. Further study with large number of patients is advocated to ascertain this assumption.

Conflicts of interest

All authors have none to declare.

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Obituary

The Cardiological Society of India expresses its deep shock and grief at the sudden demise of Dr. C.A. Abdul Latheef,

Aluva, Kerala. Dr. Abdul Latheef was a valuable and esteemed member of our society.

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