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Original Article

Association of Cardiac Axis, Q Waves, Low Voltage QRS-R Waves, R/S Ratio, ST-T Segment Abnormalities with Infarct Electrocardiogram

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ABSTRACT

Introduction: Electrocardiogram is a useful tool to predict the likely anatomic location of myocardial infarctions (MI). The present study was carried out to assess correlation of cardiac axis, Q waves, QRS-R voltage, R:S, ST-T abnormalities with pertinent infarction site in electrocardiogram.
Methods: If Q wave or QS complex are present in any lead, or lead VI-V2 show R:S≥1 or lead VI-V2 have a positive T, the electrocardiogram is called infarct electrocardiogram.
Results: ST-T abnormalities and Q waves are important predictors of acute and prior MI with pertinent infarction sites in electrocardiogram. Strong association between pathological Q, loss of R voltage and low QRS voltage is present in prior anterior MI. Acute inferior ischemia is manifested by strong association between ST segment elevation (STE), ST segment depression (STD), T inversion (TI), R/S>1 and hyperacute T while in inferior infarction Q is correlated with low QRS voltage. Acute lateral and anterolateral ischemia is manifested by strong association between STE, STD, TI whereas the infarction is characterized by Q, low QRS in both along with loss of R voltage in the former.
Conclusion: ST elevation is the most important abnormality helpful in predicting acute myocardial ischemia along with the presence of hyperacute T waves. The R ≥0.04 s, R/S ratio in leads V1-V2 with or without positive T waves was of less use in prediction of prior MI. Deviation of cardiac axis was not at all related to any of the electrocardiographic lead abnormality in MI.
Key words: Myocardial infarction, ECG, ST-T abnormalities, Q wave, R:S ratio

1. INTRODUCTION

Myocardial infarction is one of the major health problems in the world caused by interrupted blood supply to a part of the heart leading to death of the local myocardial cells and scarring of the local myocardial tissue.[1] It is detected by clinical characteristics, comprising results of electrocardiographic examination, raised levels of

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biochemical indicators of myocardial cell injury, and by diagnostic imaging, or by pathology.[2] Statistical data related to myocardial infarction rates provide valuable information concerning the problem of cardiac disease within and through populations, particularly if consistent figures are obtained in a way that differentiates between occurrence and persistence of events.[3] There have been several reports on the relation between the electrocardiogram and ventricular depolarization and repolarization abnormalities.[4,5,6] All these studies evaluated the significance of Q waves. There are only few investigations on the studies related to the importance of ST segment elevation (STE).[5,7,8] Moreover, there is scarcely any reports on the association of cardiac axis, Q waves, QRS-R voltage, R:S, ST-T abnormalities with pertinent infarction site in electrocardiogram, especially from our region of the globe. The present study is thus an attempt to examine in myocardial infarction, the possible predictive value of the electrocardiographic abnormalities such as ST segment elevation, ST segment depression (STD) at the J point, inverted T waves, frontal plane cardiac axis, low ventricular depolarization voltage and notching. The aim of the current study is to determine the pattern of distribution of ECG parameters in different MI types patients prevailing in the Institutional hospital settings, to evaluate the correlation between different ECG features in MI, to develop multiple regression model to detect MI types using relevant characteristic ECG parameters.

2. METHOD

Forty eight patients were recruited from MGM Medical College & LSK Hospital, Kishanganj, Bihar, between Aug-Oct 2015, with a diagnosis of first MI, defined as chest pain lasting for >30 min with criteria defined. Patients with left bundle branch block and left ventricular hypertrophy, with a history of previous cardiac surgery were excluded from the study. The study was carried out with clearance from the Institutional Ethical Committee. According to Third Global MI Task Force along with European Society of Cardiology, the American College of Cardiology Foundation, the American Heart Association, and the World Heart Federation, the electrocardiographic changes associated with acute myocardial ischemia is characterized by the presence of new STE≥0.1 mV at the J point in any two adjacent leads except leads V2–V3 where STE≥0.2 mV in men ≥40 years; STE ≥0.25 mV in men <40 years, or STE ≥0.15 mV in women, are applicable.[3] The electrocardiographic manifestation of prior myocardial ischemia is characterized by the presence of STD ≥0.05 mV in two adjacent leads and/or T inversion ≥0.1 mV in two adjacent leads with prominent R wave or R:S>1.[3] An electrocardiogram was referred to be infarcted when a Q wave or QS complex existed in any lead excluding leads III and V1, or by the presence of R:S≥1 in lead VI-V2, or by the presence of a positive T wave in lead VI-V2.[3]

3. RESULTS

There were 16 (33.3%), 15 (31.25%), 12 (25%) and 5 (10.42%) cases respectively suffering from anterior, inferior, anterolateral and lateral myocardial infarction (Figure 1). The electrocardiographic findings in 11 leads in 48 patients is depicted in Table 1. The patients showed abnormalities in unipolar, bipolar and augmented limb leads with ischemia characterized by ST elevation, depression, T wave inversion, R wave greater than S wave, hyperacute T waves and prior MI characterized by presence of pathological Q waves, QS complex, R ≥0.04 s and R/S≥1 in V1-V2, Positive T, Loss of R voltage. The maximum percentage of patients exhibited STE in leads I (47.9%), aVL (45.8%), and V3 (41.7%); STD in III (41.6%) and aVF (43.7%); TI in III (54.2%); R/S>1 in I (22.9%); Hyperacute T waves in V3 (45.8%) and V4 (41.6%);

similarly the prevailing lead abnormalities in prior MI is depicted in Table 1.

Out of 16 patients with anterior ischemia, STE was prevalent in respectively 10, 9, 11, 13, 12 number of patients in leads I, aVL, V2, V3, V4 while STD were mostly (n=8) in III and aVF; maximum 8 number of cases with TI was found in lead III, and hyperacute T was prevalent in leads V2-V4 (Table 2). Prior anterior MI was characterized by the presence of Q waves in I and V4 in maximum 4 and 5 patients; QS complexes in V2 and V3; positive T waves in V1 and V2; loss of R voltage in V2 (Table 2).

Inferior ischemia was present in 15 patients with prevailing STE in II, III, aVF with n=12, 13, 10 respectively; STD (n=9 and 12) and R/S > 1 (n= 8 and 7) in I and aVL respectively; TI in aVL (n=11); hyperacute T waves (n=7 and 8 respectively) in II and III (Table 2). Prior inferior MI is characterized by the presence of Q waves predominantly (n=8, 8, and 9 respectively) in leads II, III, and aVF; R≥0.04 s (n=12 and 14 respectively) and R/S≥1 (n= 4 and 6 respectively) in V1-V2; positive T waves and loss of R voltage (LR) mainly in lead V2 with n=11 and 2 respectively (Table 2).

Anterolateral ischemia and prior MI respectively was evident with STE (n=7 each) and pathological Q waves (n=5, 5, and 4) in corresponding leads namely I, aVL, and V6 while STD was prevalent in reciprocal leads III and aVF (n=7 and 8 respectively); besides the most abnormal leads were with R≥0.04 s (n=9 and 8 respectively) and R/S≥1 (n= 4 and 6 respectively) in V1-V2 (Table 3).

Lateral ischemia and prior MI respectively with STE and pathological Q was prevalent in I and aVL with n=5 in each. An R wave ≥0.04 s in V1, V2 (RG) and a positive T wave were mostly seen in V1, V2 with n=2, 3 and n= 4, 4 respectively (Table 3).

Table 4 depicts the correlation coefficient ($p < 0.05$ as significant difference) between the ECG parameters in different types of myocardial ischemia and infarction along with the regression equation expressing the association between the variables. The ST elevation was highly associated with positive correlation coefficient (r= 0.78, p>0.05; insignificant difference between the variables) with hyperacute T (HT) waves in anterior myocardial ischemia and the regression equation was expressed as STE=3.61+1.112 HT. A high correlation coefficient (r=0.8, p=0.77) was obtained between STE and HT in inferior myocardial ischemia with STE = 0.50 HT + 1.35. There was an insignificant difference between all the variables as shown in Table 4 except that between STE and STD in anterior myocardial ischemia (r=-0.60, p=0.01, STE=5.72-0.422 STD). A positive correlation was found between the presence of pathological Q waves and the loss of R voltage in prior anterior MI; similarly the QS complex was positively associated with the loss of R voltage and the presence of positive T (PT) wave; for Q and PT r was -0.22 in prior anterior MI whereas for STE and STD r was -0.56 in inferior myocardial ischemia. However, the other remaining variables as indicated in Table 4 were negatively correlated in anterolateral and lateral abnormalities; similarly the variables in prior inferior MI are also negatively associated between Q, RG, and PT.

Multiple regression equations were established to express the association between ST segment elevation, the presence of pathological Q waves, QS complex as the dependent variables and the independent variables such as STD, HT, LR, PT, TI, RG (Table 5). The STE was related with STD and TI while Q was associated with RG and PT in inferior, anterolateral, and lateral myocardial ischemia and infarction respectively (Table 5).

The ventricular depolarization voltages represented by QRS complex in limb leads and chest leads characterized by QRS ≤0.5 mV and QRS ≤0.7 mV respectively as well as the presence of QRS notch in 11 electrocardiographic leads in different MI types are represented in Figure 2. In anterior MI, the characteristic QRS height of ≤0.5 mV in limb leads was prevalent in I, II, and aVF with n= 6, 5, and 7 respectively while QRS height of ≤0.7 mV in chest leads was prevalent in V1, V5, and V6 with n= 6, 4, and 5 respectively (Figure 2). In anterolateral MI, the QRS height of ≤0.5 mV in limb leads was prevalent in I, III, and aVL with n= 9, 7, and 9 respectively while QRS height of ≤0.7 mV in chest leads was prevalent in V1 and V6 with n= 8 and 5 respectively (Figure 2). In inferior MI, the QRS height of ≤0.5 mV in limb leads was prevalent in II and aVL with n= 5 and 4 respectively while QRS height of ≤0.7 mV in chest leads was prevalent in V1 and V6 with n= 4 and 3 respectively (Figure 2).

The cardiac axis categorized as normal, LAD, RAD, and EAD in MI types is shown in Figure 3. In anterior MI, there were 13 patients with normal cardiac axis, 2 patients with LAD, 1 patient with RAD, 1 patient with EAD. Similarly, normal axis was dominant in the other MI types that is, anterolateral, inferior, and lateral MI with n=9, 13, and 3 out of 12, 15, and 5 cases respectively (Figure 3).

4. DISCUSSION

The ECG constitutes a vital diagnostic measure for the monitoring of patients with suspected MI that should be accomplished and construed promptly upon clinical presentation.[9]The diagnosis of myocardial ischemia and infarction is made based on electrocardiographic criteria. ECG manifestation of myocardial ischemia or infarction occurs in the PR segment, the QRS complex, the ST segment or the T wave.[3]The initialpresentations of myocardial ischemia are usually T wave and ST-segment alterations. The presence of an enlarged, prominent, and symmetrical hyperacute T wave in at least two adjacent leads, is an initial indication that generally pave the way for the elevation of the ST segment.[10]The lead with ST segment elevation highlights the infarct. In the present study (Table 2 and 3), ST elevation is prevalent in all types of myocardial ischemia found, that is, out of 16 patients with anterior ischemia, STE was prevalent in respectively 10, 9, 11, 13, 12 number of patients in leads I, aVL, V2, V3, V4, and hyperacute T was prevalent in leads V2-V4; inferior ischemia was present in 15 patients with prevailing STE in II, III, aVF with n=12, 13, 10 respectively, and hyperacute T waves (n=7 and 8 respectively) in II and III; anterolateral ischemia was evident with STE (n=7) while lateral ischemia with STE was prevalent in I and aVL with n=5. Moreover, the ST elevation was highly associated with positive correlation coefficient (r= 0.78, p>0.05; insignificant difference between the variables) with hyperacute T waves in anterior myocardial ischemia, and a high correlation coefficient (r=0.8, p=0.77) was obtained between STE and HT in inferior myocardial ischemia. Thus, ST elevation is the most important abnormality helpful in predicting acute myocardial ischemia along with the presence of hyperacute T waves. A brief Q waves may be found in the course of an acute ischemia.[3]

An anterior wall infarct results in STE in the precordial leads. In anterior myocardial infarction, the STE occurs in the anterior leads at the J point when anterior myocardial tissue commonly delivered by the left anterior descending coronary artery undergoes necrosis due to lack of blood supply.[12] The acute anterior MI is defined electrocardiographically by the presence ofconcave shaped downward STE in the anterior leads at the J point commonly engulfing the T wave, along with reciprocal STD in the inferior leads.[6]A lateral wall

infarct results in ST segment elevation in leads I and aVL.[12]Acute lateral and anterolateral ischemia is manifested by strong association between STE, STD, TI (in lateral STE and STD, r = -0.54, p = 0.57, STE = -0.4 STD + 2.6; in lateral STE and TI, r = -0.15, p = 0.16, STE = -0.0958 TI + 1.3; in anterolateral STE and STD, r = -0.77, p = 0.47, STE = -0.8018 STD + 5.3153; in anterolateral STE and TI, r = -0.24, p = 0.14, STE = -0.0991 TI + 2.3423); whereas the infarction is characterized by Q, low QRS in both along with loss of R voltage in the former (in lateral Q and RG, r = -0.16, p = 0.58, in lateral Q and PT, r = -0.10, p = 0.60; in anterolateral Q and RG, r = -0.34, p = 0.63, in anterolateral Q and PT, r = -0.30, p = 0.46), depicted in Table 4.The sequential evolution of an evolving STEMI comprises five phases, hyperacute T waves, ST-segment elevation, pathological Q waves, T-wave inversion, normalization of the ST-segment.[13]An acute inferior MIis found with STE in leads II, III and aVF.[14]Acute inferior ischemia is manifested by strong association between STE, STD, TI, R/S>1 and HT (for STE and HT, r = 0.80, p = 0.77, STE = 0.50 HT + 1.35; for STE and STD, r = -0.56, p = 1.0, STE = -0.44 STD + 5.5; STE = 1.71- 1.09 HT + 0.38 STD) while in inferior infarction Q is correlated with low QRS voltage (for Q and RG, r = -0.38, p = 0.69, Q = -0.69 RG + 5.0301; for Q and PT, r = -0.22, p = 0.36, Q = -0.50 PT + 3.6425; Q = 3.08 + 0.18 RG - 0.05 PT) as indicated in Table 4 and 5.According to Minnesota code 9-2, for the categorization of an aberrant ST-segment elevation, criteria of 1 mm STE in at least 1 peripheral lead, or 2 mm STE in at least 1 precordial lead is considered.[15] As per the American Heart Association Electrocardiography and Arrhythmias, the American College of Cardiology recommendation the current threshold for STE vary according to age, gender, and ECG lead.[16] Of the two convex and concave patterns of ST slope, the former is associated with STEMI and the latter is usually associated with conditions other than STEMI, such as non-malignantinitial repolarization, acute pericarditis.[17] In patients with congestive heart failure specific ECG alterations, such as pathological Qwaves or persistent STE in MI-related leads, may be seen after trans mural myocardial infarction.[18] Prior MI in the current study was characterized by the presence of pathological Q waves in I and V4 in maximum 4 and 5 patients in anterior infarction; predominantly (n=8, 8, and 9 respectively) in leads II, III, and aVF in inferior infarction; with n=5, 5, and 4 in I, aVL, and V6 leads in anterolateral infarction; in I and aVL leads with n=5 in each in lateral infarction. A positive correlation was found between the presence of pathological Q waves and the loss of R voltage in prior anterior MI; similarly the QS complex was positively associated with the loss of R voltage and the presence of positive T wave.The presence of pathological Q wave is confirmed when the duration of Q wave is 0.04 second or more or when the voltage of Q wave was more than 25% of the R wave or a QS complex.[19]The term notching is applied to a small extra deflection of approximately 1–2 mm height bearingopposite polarity, contained in the Q,R,or S wave of the QRS wave.[20]Fragmented QRS complexes are indicated by QRS waves havingeither an extra R wave, notching in the base of the S wave, broken RS complex, or QS complexes in two adjacent leads, and are observed in non-STEMI, which are accounted asindependent predictor of most important severe cardiac events.[21]In anterior MI, the QRS notch is predominantly in lead aVF (n=4), in anterolateral MI in leads II, III, aVF, in lateral QRS notch is present in lead V3, in inferior MI QRS notch is prevalent in III and aVF (Figure 2). Notching in the limb leads may also be seen in old myocardial infarction, with or without pathologic Q waves. In cases of notching in three or more precordial leads, an intraventricular

conduction disturbance is probable, or due to an infarction scar.[20]In anterior MI, there are 13 patients with normal cardiac axis, 2 patients with LAD, 1 patient with RAD, 1 patient with EAD. Similarly, normal axis is dominant in the other MI types that is, anterolateral, inferior, and lateral MI with n=9, 13, and 3 out of 12, 15, and 5 cases respectively (Figure 3).Deviation of cardiac axis was not at all related to any of the electrocardiographic lead abnormality in MI. A completely positive T wave in lead V1 is ascertained in presence of R:S ratio in lead V1 or V2, or both, ≥1; a decrease in R wave voltage from lead V1-V4 is taken as positive in presence of R:Sratio < 1 in leads V1-V4.[19]However, the R ≥0.04 s, R/S ratio in leads V1-V2 with or without positive T waves was of less use in prediction of prior MI.

Table 1: Electrocardiographic findings in 11 leads in 48 patients

	I	II	III	aVL	aVF	V1	V2	V3	V4	V5	V6
Ischemia	% age of patients										
STE	47.9	29.2	33.3	45.8	25	10.4	37.5	41.7	37.5	35.4	35.4
STD	18.7	25	41.6	27	43.7	8.3	25	25	14.5	8.3	4.2
TI	20.8	14.5	54.2	35.4	29.2	8.3	16.7	10.4	18.7	16.7	14.5
R/S>1	22.9	8.3	12.5	6.25	14.5	14.5	10.4	16.6	8.3	8.3	6.2
Hyperacute T	2	14.5	18.7	2	14.5	2	50	45.8	41.6	20.8	4.1
Prior MI	% age of patients										
Q	33.3	20.8	16.6	31.2	18.7	4.1	10.4	10.4	22.9	14.5	14.5
QS	6.2	0	6.2	8.3	2	0	4.1	6.2	4.1	2	2
R ≥0.04 s in V1-V2	0	0	0	0	0	54.1	47.9	0	0	0	0
R/S≥1 in V1-V2	0	0	0	0	0	10.4	14.5	0	0	0	0
Positive T	0	0	0	0	0	66.7	85.4	0	0	0	0
Loss of R voltage	0	0	0	0	0	0	37.5	12.5	12.5	10.4	18.7

STE: ST elevation; STD: ST depression; TI: T inversion

Table 2: Electrocardiographic findings in 11 leads in 16 patients with anterior and inferior ischemia and infarction

Anterior (n=16)	**I**	**II**	**III**	**aVL**	**aVF**	**V1**	**V2**	**V3**	**V4**	**V5**	**V6**
Ischemia											
STE	10	2	3	9	2	3	11	13	12	8	5
STD	0	5	8	0	8	1	2	2	2	1	1
TI	0	3	8	2	5	0	1	2	4	3	3
R/S>1	1	1	3	0	3	0	0	1	1	0	0
Hyperacute T	0	0	0	0	0	0	13	14	12	7	1
Prior MI											
Q	4	2	0	3	0	0	2	2	5	2	1
QS	2	0	2	2	0	1	3	3	2	1	1
R ≥0.04 s in V1-V2	0	0	0	0	0	3	1	0	0	0	0
R/S≥1 in V1-V2	0	0	0	0	0	0	0	0	0	0	0
Positive T	0	0	0	0	0	14	15	0	0	0	0
Loss of R voltage	0	0	0	0	0	0	13	5	4	0	0
QRS height											
≤ 0.5mV in limb	6	5	2	3	7	0	0	0	0	0	0
≤ 0.7mV in chest	0	0	0	0	0	6	0	0	0	4	5
QRS notch	1	0	2	1	4	0	0	0	0	0	0
Inferior (n=15)											
Ischemia											
STE	1	12	13	1	10	1	1	0	1	1	1
STD	9	0	0	12	1	3	7	5	3	2	0
TI	7	2	9	11	5	3	4	2	3	2	2
R/S>1	8	2	2	7	1	5	2	4	1	1	1
Hyperacute T	0	7	8	1	7	0	6	4	2	1	0
Prior MI											
Q	2	8	8	2	9	1	1	1	3	2	1
QS	0	0	1	0	1	0	0	0	0	0	0
R ≥0.04 s in V1-V2	0	0	0	0	0	12	14	0	0	0	0
R/S≥1 in V1-V2	0	0	0	0	0	4	6	0	0	0	0
Positive T	0	0	0	0	0	8	11	0	0	0	0
Loss of R voltage	0	0	0	0	0	0	2	0	0	0	0
QRS height											
≤ 0.5mV	3	5	3	4	1	0	0	0	0	0	0
≤ 0.7mV	0	0	0	0	0	4	0	0	1	2	3
QRS notch	0	0	1	0	1	0	0	0	0	0	0

STE: ST elevation; STD: ST depression; TI: T inversion

Table 3: Electrocardiographic findings in 11 leads in 12 patients with anterolateral and lateral ischemia and infarction

Anterolateral (n=12)	**I**	**II**	**III**	**aVL**	**aVF**	**V1**	**V2**	**V3**	**V4**	**V5**	**V6**
Ischemia											
STE	7	0	0	7	0	1	4	4	4	4	7
STD	0	5	7	0	8	0	1	3	3	1	0
TI	1	1	5	2	2	1	2	1	2	3	2
R/S>1	1	1	1	2	2	1	2	1	2	3	2
Hyperacute T	0	0	1	0	0	1	2	2	3	0	0
Prior MI											
Q	5	0	0	5	0	1	2	2	2	2	4
QS	1	0	0	1	0	0	0	0	0	0	0
R ≥0.04 s in V1-V2	0	0	0	0	0	9	8	0	0	0	0
R/S≥1 in V1-V2	0	0	0	0	0	4	6	0	0	0	0
Positive T	0	0	0	0	0	6	11	0	0	0	0
Loss of R voltage	0	0	0	0	0	0	2	0	0	2	9
QRS height											
≤ 0.5mV	9	4	7	9	7	0	0	0	0	0	0
≤ 0.7mV	0	0	0	0	0	8	2	0	0	2	5
QRS notch	0	2	2	1	2	0	1	1	1	0	0
Lateral (n=5)											
Ischemia											
STE	5	0	0	5	0	0	2	3	1	4	4
STD	0	2	5	1	4	0	2	2	1	1	1
TI	2	1	4	2	2	0	1	0	0	0	0
R/S>1	1	0	0	0	1	1	1	1	0	0	0
Hyperacute T	1	0	0	0	0	0	3	2	3	2	1
Prior MI											
Q	5	0	0	5	0	0	0	0	1	1	1
QS	0	0	0	1	0	0	0	0	0	0	0
R ≥0.04 s in V1-V2	0	0	0	0	0	2	3	0	0	0	0
R/S≥1 in V1-V2	0	0	0	0	0	0	0	0	0	0	0
Positive T	0	0	0	0	0	4	4	0	0	0	0
Loss of R voltage	0	0	0	0	0	0	1	1	1	0	0
QRS height											
≤ 0.5mV	1	2	0	1	2	0	0	0	0	0	0
≤ 0.7mV	0	0	0	0	0	1	1	0	0	0	0
QRS notch	0	0	0	0	0	0	0	1	0	0	0

STE: ST elevation; STD: ST depression; TI: T inversion

Table 4: Association between ECG parameters in different myocardial ischemia and infarction

	r	p value	Regression equation
Anteriormyocardial ischemia			
ST elevation (STE) and Hyperacute T (HT)	0.78	0.21	STE=3.61+1.112 HT
STE and ST depression (STD)	-0.60	0.01	STE=5.72-0.422 STD
Prior anterior myocardial infarction			
Q and loss of R voltage (LR)	0.21	0.95	Q=1+0.52 LR
Q and positive T wave (PT)	-0.22	0.79	Q=3.76-0.73 PT
QS and loss of R voltage (LR)	0.66	0.73	QS = 2.61 LR - 2.03
QS and PT	0.29	0.63	QS = 1.48 PT + 0.08
Inferior myocardial ischemia			
STE and HT	0.80	0.77	STE = 0.50 HT + 1.35
STE and STD	-0.56	1.0	STE = -0.44 STD + 5.5
Prior inferior myocardial infarction			
Q and R>0.04 s in V1-V2 (RG)	-0.38	0.69	Q = -0.69 RG + 5.0301
Q and PT	-0.37	0.36	Q = -0.50 PT + 3.6425
Anterolateral myocardial ischemia			
STE and STD	-0.77	0.47	STE = -0.8018 STD + 5.3153
STE and T inversion (TI)	-0.24	0.14	STE = -0.0991 TI + 2.3423
Prior anterolateral myocardial infarction			
Q and RG	-0.16	0.58	Q = -0.2708 RG + 2.0208
Q and PT	-0.10	0.60	Q = -0.1849 PT + 1.8411
Lateral myocardial ischemia			
STE and STD	-0.54	0.57	STE = -0.4 STD + 2.6
STE and TI	-0.15	0.16	STE = -0.0958 TI + 1.3
Lateral myocardial infarction			
Q and RG	-0.34	0.63	Q = -0.2877RG + 1.2466
Q and PT	-0.30	0.46	Q = -0.3288PT + 1.4247

Table 5: Multiple regression equation between ECG parameters invarious myocardial ischemia and infarction

	Regression equation
Anterior myocardial ischemia (STE, STD, HT)	STE = 6.81+0.47 HT-0.64 STD
Anterior prior myocardial infarction (Q, LR, PT)	Q = 1.86 + 0.25 LR -0.19 PT
Anterior prior myocardial infarction (QS, LR, PT)	QS = 1.23 + 0.21 LR -0.05 PT
Inferior myocardial ischemia (STE, STD, TI)	STE = 1.71- 1.09 HT + 0.38 STD
Inferior prior myocardial infarction (Q, RG, PT)	Q = 3.08 + 0.18 RG -0.05 PT
Anterolateral myocardial ischemia (STE, STD, TI)	STE = 4.33 - 0.44 STD + 0.12 TI
Anterolateral myocardial infarction (Q, RG, PT)	Q = 2.21- 0.25 RG + 0.17 PT
Lateral myocardial ischemia (STE, STD, TI)	STE = 3.34 – 1.04 STD + 0.6 TI
Lateral myocardial infarction (Q, RG, PT)	Q = 1.56 – 2.48 RG + 2.5 PT

STE: ST elevation; STD: ST depression; HT: Hyperacute T; LR: Loss of R voltage; PT: Positive T wave; TI: T inversion; RG: R>0.04 s in V1-V2

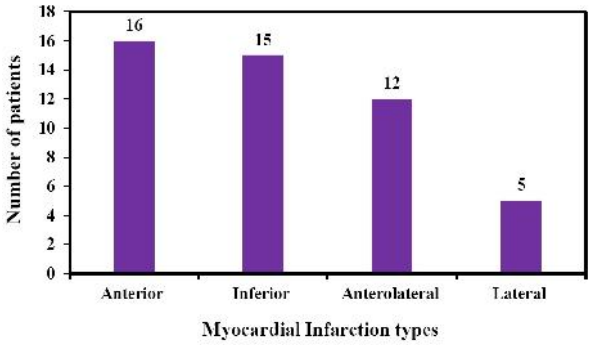

Fig 1: Distribution of Myocardial Infarction types

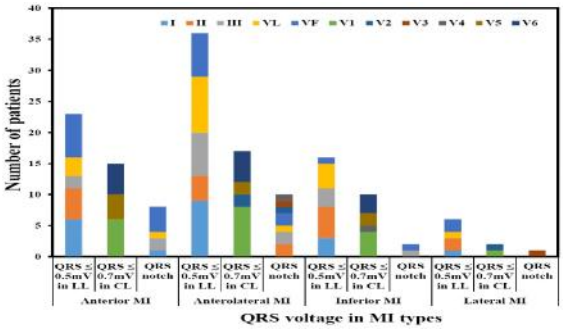

Fig 2: QRS voltage in MI types [MI: Myocardial infarction; LL: Limb leads: CL: Chest leads]

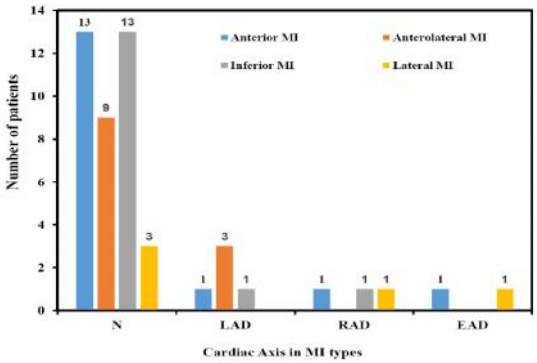

Fig 3: Cardiac Axis in MI types [MI: Myocardial infarction; N: Normal; LAD: Left axis deviation; RAD: Right axis deviation; EAD: Extreme axis deviation]

5. CONCLUSION

ST elevation is the most important abnormality helpful in predicting acute myocardial ischemia along with the presence of hyperacute T waves. Strong association between pathological Q, loss of R voltage and low QRS voltage is present in prior anterior myocardial infarction. Acute inferior ischemia is manifested by strong association between STE, STD, TI, R/S>1 and HT while in inferior infarction Q is correlated with low QRS voltage. Acute lateral and anterolateral ischemia

is manifested by strong association between STE, STD, TI whereas the infarction is characterized by Q, low QRS in both along with loss of R voltage in the former. The R ≥0.04 s, R/S ratio in leads V1-V2 with or without positive T waves was of less use in prediction of prior MI. Deviation of cardiac axis was not at all related to any of the electrocardiographic lead abnormality in MI.

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