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Electrocardiographic prediction of lateral involvement in acute non-anterior wall myocardial infarction ☆

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Abstract

Purpose: Recent research has established that a tall R-wave in V_1 indicates lateral wall involvement in non-anterior wall myocardial infarction (MI). The objective of this study was to assess the value of the admission electrocardiogram (ECG) to predict R-waves and consequently lateral wall damage in the late phase of non-anterior MI.

Methods: ECGs of 69 patients were analyzed. ST-segment changes in representative leads for lateral wall infarction such as V_1, V_2, V_6 and I were correlated with the extent of QRS-wave changes in V_1 and V_6.

Results: ST-segment elevation in V_6 showed correlations with R/S ratio in V_1 ($r = 0.802$, $B = 0.440$, $P = <0.001$) and with the depth of Q-waves in V_6 ($r = 0.671$, $B = 0.441$, $P = 0.007$). This correlation was higher in a small subgroup where the left circumflex branch (Cx) was the culprit vessel ($r = 0.888$, $B = 1.469$ and $P = 0.018$). ST-segment depression in lead I correlated with the height of R and the surface of R in V_1 (height times width of R) ($r = 0.542$, $B = -0.150$, $P = 0.005$ and $r = 0.538$, $B = -0.153$, $P = 0.005$ respectively), especially in the subgroup without proximal occlusions of RCA ($r = 0.711$ and $r = 0.699$). ST-segment depression in lead I also predicted Q-waves in V_6 ($r = 0.538$, $B = 0.114$, $P = 0.006$). ST-segment changes in V_2 showed no significant correlation with either R- or Q-wave measurements.

Conclusions: ST-segment elevation in V_6 in the acute phase of non-anterior MI predicts lateral involvement as expressed by the R/S ratio in V_1 in the post reperfusion phase. A subgroup with Cx occlusion showed especially strong correlations, although the size of the group was small. In lead I ST-segment depression is correlated to height and surface of R in V_1 and Q-waves in V_6.

Keywords: Electrocardiography; ST elevation myocardial infarction; Infarct location; CMR

Introduction

Acute transmural myocardial ischemia is represented as ST-segment elevation on a standard 12-lead surface electrocardiogram (ECG) in leads with the positive pole facing the ST-vector [1–3]. In the acute phase of inferior wall ischemia ST-elevation is present in leads II, III and aVF. If the lateral wall is involved there may be ST-elevation in V_5-V_6[2]. In addition, ST-depression, which can be ST-segment elevation observed reversely, often occurs in leads V_1-V_3 and aVR [3]. In the chronic phase, the infarcted area presents as a loss of R-waves and/or formation of Q-waves at the positive poles facing the affected tissue. Non-anterior wall myocardial infarction (MI) typically shows those changes in leads II, III, aVF, V_5 and V_6[4–6]. In addition, a gain of height and width of R-waves in V_1 facing the lateral wall from the opposite side are observed. Recently the latter has been stressed to indicate lateral, instead of long-believed 'true posterior', wall involvement [7–10].

Recent research from our group (11) has quantified the relationship between R-waves in V_1 and Q-waves in V_6 with the extent of lateral wall involvement in the chronic stage of non-anterior wall myocardial infarction by correlating cardiac magnetic resonance (CMR) imaging with R-wave measurements [11]. In the same study, height, surface (measured as height times width) and R/S ratio of R-waves in lead V_1, and the height of R in V_6 were found to correlate with the regional infarct size in the lateral wall (all results

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approximately r = 0.65). For inferolateral wall infarction correlations were found for the depth and width of Q in V_6 (r = 0.38 and r = 0.40 respectively).

Because lateral involvement is a clinically relevant extension in non-anterior wall MI it is important to recognize lateral involvement in the acute phase. Subsequent to these earlier findings the objective of this study was to assess the ST-segment changes in lead I, V_1, V_2 and V_6 in the acute phase, being most closely correlated to the R- and S-wave changes in lead V_1 and the Q-wave changes in V_6 in the late phase. Quantification of the relationship between acute ST-segment deviation and R-wave and Q-wave development in the post reperfusion phase could provide an estimate of the area at risk to the (infero)lateral wall. The authors hope to identify the most predictive early ST-segment changes for (infero)lateral involvement as reflected by QRS wave measurements from earlier research for early recognition and prompt cardiac intervention.

Methods

Patient population

The same study group as in reference 11 was analyzed, presenting with a first acute non-anterior wall ST-elevation MI (STEMI) at Maastricht University Center (UMC+) from August 2006 to March 2008 in the Maastricht ST elevation (MAST) study. Inclusion criteria were (1) symptoms consistent with an acute STEMI lasting for more than 30 minutes but less than 6 h, (2) ST-segment elevation of more than 1 mm in anatomically adjacent leads in the initial ECG, (3) occlusion of either the right coronary artery (RCA) or left circumflex branch (Cx), and (4) availability of ECG images. Exclusion criteria were (1) age below 18 years, (2) cardiogenic shock, (3) pregnancy, (4) inability to obtain informed consent, (5) occlusion of the left anterior descending artery, and (6) the presence of a bundle branch block. From the original 106 patients of the MAST database 30 had LAD occlusion and 7 patients were excluded because of artifacts eliminating the ECG analysis.

ST-elevation MI was defined by ECG evaluation and enzymatic changes according to the clinical standard. Non-anterior infarction was present when the culprit artery was the Cx, RCA or one of their branches.

All patients underwent the standard treatment for acute MI and primary percutaneous coronary intervention (PCI) of the culprit artery. The culprit artery was defined by the interventional cardiologist based on standard operating procedures using the European Society of Cardiology (ESC) guidelines: irregular borders, eccentricity, ulcerations, and filling defect suggestive of intraluminal thrombi.

The review board of our institution approved our study protocol, and informed consent was obtained in all patients.

Electrocardiographic evaluation

Standard 12-lead ECGs were recorded using a General Electric (GE) MAC 5500 12-lead recorder, produced in Freiburg, Germany. A single investigator blinded to clinical data analyzed the electrocardiograms. ECGs taken at the emergency room before PCI were selected showing the most prominent ST-segment deviation. The study ECG was recorded on average 2:10 h (SD 1:07 h) after the onset of coronary complaints.

All ECGs were scanned into a software environment and magnified 8 times. After magnification the degree of ST-deviation was measured between the J-point and the baseline in leads V_1, V_2, V_6 and lead I (as shown in Fig. 1). The TP-TP segment was considered as baseline and the J-point was defined as first point of inflection of the upstroke of the S-wave. Measurements were displayed in mm, with 1 mm corresponding to 0.1 mV for height. The amount of ST-segment deviation was related to the extent of R-wave formation as reported in reference 11. All QRS-waves from the database were measured again using the reported methods to calculate interobserver variability. Pearson correlations varying from r = 0.960 to r = 0.969 for all respective variables were found, suggesting high accuracy and good reproducibility of the measured data. The ECGs, displaying post reperfusion QRS-wave changes, were recorded within a mean of 2 days (SD 2 days) after admission and intervention of the patients.

Statistics and data analysis

ST-segment deviation was correlated with R- and Q-wave measurements. A subgroup analysis was applied for RCA and Cx MI separately. The group of patients with RCA as the culprit vessel was also assessed by excluding proximal occlusion to investigate the effect of right ventricular involvement. Simple linear regression was used to assess the relation between the degree of acute ST-segment deviation and the occurrence of R- and Q-waves in the late phase. In addition these correlations were assessed by multiple regression correcting for the most significant patient characteristics such as sex, right ventricle infarction, and age. The correlation coefficient was calculated by the Pearson test and is displayed as *R*. B values reflect the unstandardized (regression) coefficient. Fisher's exact test was performed to

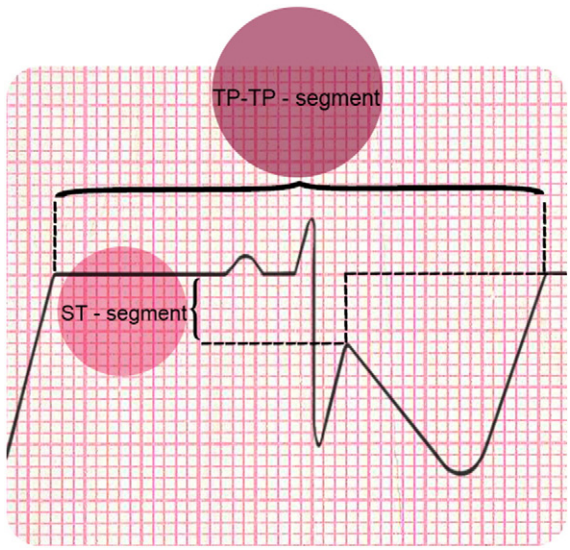

Fig. 1. Electrocardiographic measurement. Image showing ST-segment depression measured from the baseline at the J-point in V_1. ST-segment elevation in V_6 was measured using the same method.

Table 1
Patient characteristics.

Variable	No. of patients	%
Total	69	100
Male	47	68
RCA as culprit vessel	59	86
Proximal RCA occlusion[a]	32	46
Cx as culprit vessel	10	15
Hypertension	23	33
Dyslipidemia	20	29
Smokers or a history of smoking	63	91
Positive family history	33	48
Diabetes mellitus	5	7
Right ventricular involvement[b]	41	59
Peripheral artery disease	7	10
AP in preceding 24 h	31	45
Baseline TIMI 0–1 flow[c]	48	70
Final TIMI 3 flow[c]	59	86
Multiple vessel disease	36	52
Variable	**Mean**	**SD**
Age (years)	60	11
Total minutes of ischemia[d]	210	71
Troponin-T t = 0 h	0.05	0.1
Troponin-T t = 6 h	4.8	4.7
CRP t = 0 h (mg/L)	5.3	9.6
Pro-BNP t = 48 h (pmol/L)	114	98
ECG taken after onset symptoms[e]	130	67

Normal values: CK-MB male, <0.01 μg/L; CRP, <10 mg/L, Pro-BNP, <35 pmol/L. AP indicates angina pectoris; RCA, right coronary artery; Cx, left circumflex branch; SD, standard deviation.

[a] Proximal RCA is defined as the portion prior to the origin of the acute marginal branch.

[b] Right ventricular involvement was defined as 1 mm ST-segment elevation in right precordial V_4.

[c] Data from angiography.

[d] Calculated as time from onset of symptoms until intervention.

[e] Calculated as time from onset of symptoms until ECG evaluation.

investigate discrete data in groups with 10 individuals or less. An increase of QRS-waves in 1 mm equals an increase of the correlated variable by 1.00. Correlations were regarded significant if $P < 0.05$. Data was analyzed using SPSS version 21.0 (IBM, New York). Only relevant correlations are displayed, focusing on leads V_1, V_6 and I.

Results

The baseline characteristics are shown in Table 1. Of a total of 106 patients with STEMI, 69 patients (47 male, 22 female) with a mean age of 59.5 years (SD 10.5) diagnosed with non-anterior wall MI were studied. Of those, 59 (85.5%) presented with RCA occlusion and 10 (14.5%) with Cx as the culprit vessel; 63 (91.3%) had a smoking history, 5 patients had diabetes mellitus and 23 (33.3%) had hypertension, 48 patients (69.5%) had thrombolysis in myocardial infarction (TIMI) flow grade 0 to 1 before PCI, and TIMI 3 was established in 59 (85.5%) patients. Fig. 2 displays typical acute ST-segment changes and late R-wave development in Cx occlusion.

ST-segment deviation in relation to R-wave measurements

Simple linear regression showed moderate positive correlations between ST-segment depression in V_1 and R height, R surface and R/S ratio in V_1 and a negative correlation with the R-waves in V_6 (varying from $P = 0.001$ to $P = 0.091$) (Table 2). However, ST-segment elevation in

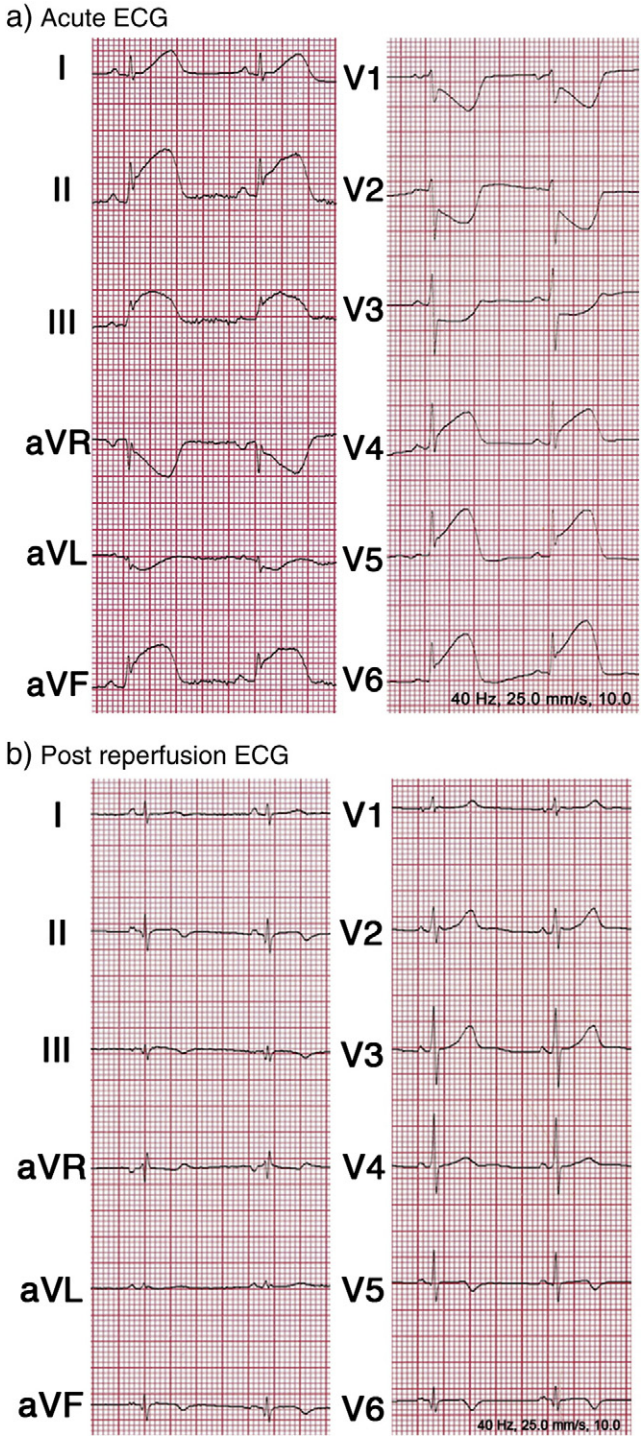

Fig. 2. (a) Electrocardiogram of a patient with Cx occlusion. In the acute ECG isoelectricity can be noticed in I. The same amount of ST-segment elevation in II and III and the same amount of ST-segment depression in aVR and aVL is indicating a downwards pointing ischemic vector with elevation in aVF, as is typically seen in Cx occlusions. V_1, V_2 and V_3 show major ST-segment depression, while elevation is seen in V_4, V_5 and V_6.(b) Electrocardiogram of the same patient in the post reperfusion phase of lateral myocardial infarction. There are Q-waves in II, III and aVF and R-waves in aVR and a loss of R in aVL. In V_1 the S-wave is less deep than would be expected and a gain of height of R-wave has occurred. In V_6 a Q-wave has developed and also R shows a loss in height.

Table 2
Simple and multiple linear regression.

	R	B	95% CI	*P*
Simple linear regression for analysis of acute ST-segment deviation and R or Q measurements in the chronic phase				
ST-segment depression V_1 in mm*				
Height of R in V_1 in mm	0.271	0.414	0.094 to 0.883	0.059
R/S in V_1 in mm	0.447	0.225	0.041 to 0.522	0.001
Surface of R in V_1 in mm^2	0.244	0.351	0.050 to 0.879	0.091
Height of R in V_6 in mm	0.328	−1.916	−3.105 to −0.389	0.024
Depth of Q in V_6 in mm	0.401	0.384	0.121 to 0.648	0.005
ST-segment deviation V_2 in mm*				
Height of R in V_1 in mm	0.042	0.028	−0.152 to 0.208	0.759
R/S in V_1 in mm	0.155	−0.034	−0.093 to 0.025	0.251
Surface of R in V_1 in mm^2	0.053	0.033	−0.136 to 0.203	0.694
Height of R in V_6 in mm	0.239	0.614	−0.080 to 1.308	0.082
Depth of Q in V_6 in mm	0.119	−0.053	−0.175 to 0.069	0.386
ST-segment elevation V_6 in mm				
Height of R in V_1 in mm	0.220	0.275	−0.293 to 0.830	0.261
R/S in V_1 in mm	0.780	0.402	0.030 to 0.532	<0.0001
Surface of R in V_1 in mm^2	0.201	0.251	−0.252 to 0.905	0.304
Height of R in V_6 in mm	0.448	−2.273	−4.911 to −0.759	0.022
Depth of Q in V_6 in mm	0.464	0.360	0.071 to 0.649	0.017
ST-segment depression lead I in mm*				
Height of R in V_1 in mm	0.304	−0.268	−0.503 to −0.032	0.027
Surface of R in V_1 in mm^2	0.275	−0.262	−0.520 to −0.005	0.046
Multiple linear regression for analysis of acute ST-segment deviation and R measurements in the chronic phase				
ST-segment depression V_1 in mm*				
Height of R in V_1 in mm	0.385	0.345	−0.079 to 0.770	0.037
R/S in V_1 mm	0.496	0.185	0.034 to 0.337	0.024
Surface of R in V_1 in mm^2	0.386	0.279	−0.111 to −0.669	0.036
Height of R in V_6 in mm	0.319	−1.883	−3.671 to −0.096	0.039
Depth of Q in V_6 in mm	0.401	0.343	0.068 to 0.617	0.033
ST-segment elevation V_6 in mm				
Height of R in V_1 in mm	0.449	0.333	−0.131 to 0.797	0.075
R/S in V_1 in mm	0.802	0.440	0.290 to 0.590	<0.0001
Surface of R in V_1 in mm^2	0.443	0.223	−0.236 to 0.683	0.178
Height of R in V_6 in mm	0.407	−2.021	−4.024 to −0.018	0.048
Depth of Q in V_6 in mm	0.671	0.441	0.159 to 0.722	0.007
ST-segment depression lead I in mm*				
Height of R in V_1 in mm	0.542	−0.150	−0.434 to 0.133	0.005
Surface of R in V_1 in mm^2	0.538	−0.153	−0.476 to 0.169	0.005

Confounders in multiple regression are sex, right ventricle infarction, and age. *P* values in the table correspond to the B value. R indicates correlation value; (unstandardized) regression coefficient B, the increase of QRS-wave variable in mm when the variable increases by 1.00; CI, confidence interval.

* An increase in 1 mm of Q-, R- or S-wave equals an increase in 1 mm ST-segment depression and vice versa.

V_6 showed a strong positive correlation with the R/S ratio in V_1 (r = 0.780, B = 0.402, P = <0.001) and also correlated to some extent negatively with the height of R in V_6 (r = 0.448, B = −2.273, P = 0.022). ST-segment depression in lead I predicted the height and surface of R moderately (r = 0.304, B = −0.268, P = 0.027 and r = 0.275, B = −0.262, P = 0.046). The confounders sex, acute right ventricle infarction, and age showed to be significant in predicting R-waves. The most significant relationship including these variables in multiple regression was found for ST-segment elevation in V_6 and R/S ratio in V_1 (r = 0.802, B = 0.440 and P = <0.001) and ST-segment depression in lead I and the height and surface of R (r = 0.542, B = −0.150, P = 0.005 and r = 0.538, B = −0.153, P = 0.005 respectively). As seen in Table 3, evaluating subgroups of patients with RCA and Cx occlusions improved the correlation of ST-segment elevation in V_6 with the R/S ratio in V_1, especially for the small group of Cx occlusion (n = 10) (r = 0.888, B = 1.469 and P = 0.018). To test the correlation and account for the small sample size (n = 10) the Fisher's exact test was performed. The two variables were discretized into two groups with the cut-off point at 2 mm (0.2 mV). This showed a significance of P = 0.008. Excluding proximal RCA occlusion, which is prone to causing right ventricular infarction, improved the correlation of the height and surface of R in V_1 with lead I, but had only a small impact on variables predicted by acute ST-segment changes in V_1 and V_6. ST-deviation in V_2 showed little value in predicting R-waves. Pearson correlations for all variables predicted by acute lead V_2 ST-segment deviation varied between r =0.239 and r = 0.042 and showed no significant regression coefficients (Table 2).

ST-segment deviation in relation to Q-waves

As seen in Table 2, when correcting for the above mentioned confounders, the depth of Q-waves in V_6 was

predicted best by acute ST-segment elevation in this same lead (r = 0.671, B = 0.441, $P = 0.007$).

Discussion

The most prominent findings of this study are (1) ST-segment elevation in V_6 predicts the R/S ratio in V_1 and the depth of Q-waves in V_6 in the post reperfusion phase, especially in the subgroup of Cx occlusions that consisted of 10 patients; (2) the ST-segment depression in lead I respectively correlated moderately with the height and surface of R-waves in V_1, especially in a group of mid and distally occluded RCA and Cx; (3) ST-segment deviation in V_2 showed no significant correlation to any R- or Q-wave measurement.

R-wave changes in V_1 are sign of lateral wall infarction

R-waves in V_1 were considered to show infarction of the posterior wall before CMR studies proved them to be a sign of lateral wall MI [12]. Only 5% of patients have a 'true posterior wall': where the basal part of the inferior wall bends far upwards or if the heart is in an upright position with the base and the apex aligned vertically, as can be seen in very lean individuals. In the majority of patients however, the heart is situated in a postero-anterior and right-to-left angled position with V_1 facing the lateral wall instead of the posterior wall [13]. These findings have lead to a replacement of the expression 'posterior' with 'inferolateral' over the past years [14]. Consequently lateral MI loss of electrical forces in the lateral wall are depicted in the opposite located lead V_1 as an increase in R- and loss of S-wave. With its positive pole facing the lateral wall, V_6 registers a gain of Q and a loss of R in lateral wall MI (Fig. 2) [15].

Acute ST-segment changes and R-waves

CMR studies of our group correlating R-waves with CMR imaging found the best relation to be between height of R and surface of R with lateral wall involvement (r = 0.68 for both variables) [11]. These variables are however only partly predicted by acute ST-segment depression in lead I. The R/S ratio in V_1 and the depth of Q in V_6 showed higher correlations with our measurements, while being slightly less correlated (r = 0.65) to the extent of infarction as evaluated by CMR in our earlier research (11). This implies that in the acute phase, lateral wall involvement can be best assessed by ST-segment elevation in V_6, whereas in the chronic phase R/S ratio in V_1 is the best indicator.

The high correlation between ST-segment elevation in V_6 and the extent of R/S ratio supposedly results from the positive pole of V_6 facing directly onto the lateral wall and alongside the right ventricle, giving the most precise information about the lateral wall. Also, combining a gain of R- and loss of S-wave in V_1 in one variable seems to be a suitable measurement of the extent of R-waves, since acute ST-segment changes affect the height of R as well as the depth of S. In the clinical setting, lateral wall infarction is specifically due to Cx occlusion. The strong correlation of ST-segment elevation in V_6 in the small group of Cx occlusion and R/S ratio in V_1 was expected considering its laterally located supply area, and is in accordance with earlier studies investigating V_6 ST-segment elevation in single vessel Cx occlusion [16]. However, the correlation between ST-segment elevation in V_6 and R/S ratio in V_1 needs further testing because of underrepresentation of Cx occlusions in STEMI populations.

Right ventricular involvement in (infero)lateral infarction

Right ventricular infarction, commonly found in proximal RCA occlusion, was thought to possibly counterbalance lateral involvement, thus concealing lateral wall ischemia. We hypothesized the sensitivity of lead I and V_6 for lateral involvement to be affected by right ventricle infarction. Since the right ventricle is opposing the lateral wall, an infarction of the area attenuates the ischemic vector in lateral involvement in the aforementioned leads. To assess the effect of right ventricular infarction on the prediction of lateral involvement, a subgroup was analyzed, in which proximal RCA occlusion was excluded. ST-segment changes in lead I proved to be affected by right ventricular infarction in the prediction of R surface and R height, since their correlations improved in the subgroup without proximal RCA occlusion (Table 3).

When RCA is proximally occluded, larger parts of the inferior wall and the right ventricle are involved, shifting the ischemic vector away from the positive pole of lead I and causing the ST-segment to become more negative. In contrast, a mid- or distally-occluded RCA or involvement of the left marginal branch of Cx can cause the ischemic vector to point more perpendicularly toward lead I or even toward its positive pole, presenting as ST-segment elevation on the ECG. This results in a negative correlation between acute ST-segment depression in lead I and the extent of R-waves in V_1. By excluding proximal RCA occlusion, lateral involvement can attenuate the reciprocal ST-segment depression in lead I more easily. Lead I then becomes more sensitive to lateral involvement. Our findings were supported by analyzing both ST-segment depression and ST-segment elevation in lead I as one continuous variable, further improving the correlation between acute ST-segment changes in lead I and the height of R in V_1 (r = 0.785, B = 0.268, $P = <0.0001$).

It is well accepted that ST-segment elevation in V_6 predicts Q-waves in the same lead. Earlier studies found Q-waves in V_6 to be quantitatively correlated to the inferolateral segment instead of the lateral segment, which is supported by studies that found poor correlations between Q-waves and lateral wall MI [5,11,12]. Excluding proximal RCA occlusion did not improve the prediction of Q-waves as depicted by lead V_6 ST-segment changes, assuming that the diagnosis of inferolateral wall MI, as predicted by acute ST-segment changes in V_6, is not affected by right ventricular involvement.

Limitations

The group of patients with Cx occlusions was relatively small; further confirmation of the findings in a different patient population is needed.

Table 3
Subgroups.

Simple linear regression for RCA, Cx and RCA and Cx (without proximal RCA occlusions)				
	R	B	95% CI	*P*
ST-segment elevation V_6 in mm				
R/S in V_1 mm in RCA occlusion	0.625	0.188	−0.031 to 0.282	0.002
R/S in V_1 mm in Cx occlusion	0.888	1.469	0.414 to 2.524	0.018
R/S in V_1 mm in RCA and Cx occlusion (excluding proximal occlusions of RCA)	0.782	0.444	0.232 to 0.656	0.001
ST-segment depression lead I in mm*				
Height of R in V_1 in mm in RCA and Cx occlusion (excluding proximal occlusions of RCA)	0.357	−0.203	−0.408 to 0.003	0.053
Surface of R in V_1 in mm in RCA and Cx occlusion (excluding proximal occlusions of RCA)	0.339	−0.195	−0.405 to 0.014	0.067
Multiple linear regression for RCA, Cx and RCA and Cx (without proximal RCA occlusions)				
ST-segment elevation V_6 in mm				
R/S in V_1 mm in RCA occlusion	0.750	0.172	0.063 to 0.281	0.004
R/S in V_1 mm in Cx occlusion	*Group too small for multiple regression*			
R/S in V_1 mm in RCA and Cx occlusion (excluding proximal occlusions of RCA)	0.809	0.508	0.255 to 0.760	0.003
ST-segment depression lead I in mm*				
Height of R in V_1 in mm in RCA and Cx occlusion (excluding proximal occlusions of RCA)	0.711	−0.206	−0.431 to 0.019	0.004
Surface of R in V_1 in mm in RCA and Cx occlusion (excluding proximal occlusions of RCA)	0.699	−0.189	−0.424 to 0.047	0.005

* An increase in 1 mm of Q-, R- or S-wave equals an increase in 1 mm ST-segment depression and vice versa.

Clinical implications

The results of this study suggest that lateral involvement reflected as QRS changes in non-anterior wall infarction can be identified by ST-segment elevation in V_6 in the acute phase. ST-segment elevation in V_6 is correlated not only with R/S in V_1 and R loss in V_6, but also with late Q-wave formation in the same lead, the latter suggesting additional inferolateral involvement. This is important as it indicates the area at risk in the lateral wall and the need for prompt intervention.

Summary and conclusion

Our findings show that acute ST-segment elevation in V_6 is correlated with the extent of R- and S-waves in V_1 and Q-waves in V_6 in non-anterior infarction reflecting lateral wall involvement.

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