Original Article

The Pattern of Reciprocal Electrocardiography Changes in ST-Segment Elevation Myocardial Infarction Patients Presenting with Single-Vessel Disease versus Multi-Vessel Disease

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Abstract

Introduction: The reciprocal ST-segment depression in the electrocardiography (ECG) leads overlying noninfarcting areas was studied previously in acute myocardial ischemia. Multi-vessel disease (MVD) subset of patients have more vague and confusing presentations on ECG; they usually show less ST-segment elevation and profound and diffuse ST-segment depression compared to ST-segment elevation myocardial infarction (STEMI) patients with single-vessel disease (SVD) involving occlusion of one coronary artery only, namely the infarct-related artery (IRA). **Aim of the Work:** The aim was to study and compare the pattern of reciprocal ECG changes in STEMI patients presenting with SVD versus MVD. **Methods and Results:** A total of 125 consecutive patients admitted from April 2014 to August 2015 from the emergency room with the diagnosis of acute STEMI and treated by primary percutaneous coronary intervention (PPCI) at our cath lab at Ainshams University Hospitals (a 24/7 tertiary referral center for PPCI) were included. ST-segment deviations were measured at the J-point. Reciprocal ST-segment changes were identified as per guidelines published by the European Society of Cardiology and the American College of Cardiology, i.e., ST-segment depression ≥0.1 mV in any ECG lead other than aVR, while the cutoff value is different for leads V2 and V3 being only 0.05 mV. Coronary angiographies were evaluated by two independent operators blinded to the clinical and electrocardiographic data. Regarding the left anterior descending (LAD) occlusion, the reciprocal ST-segment depression magnitudes in lead III and in lead arteriovenous fistula (aVF) were significantly less in the MVD group compared to the SVD group, i.e., lead III (−0.08 ± 0.10 mV vs. −0.19 ± 0.15, $P = 0.015$) and lead aVF (−0.07 ± 0.06 mV vs. −0.15 ± 0.11, $P = 0.02$); while regarding the left circumflex coronary artery (LCX) occlusion, the reciprocal ST-segment depression extended significantly in V4 chest lead in the MVD group compared to the SVD group (−0.16 ± 0.08 mV vs. −0.1 ± 0.04, $P = 0.025$); and finally regarding the right coronary artery (RCA) occlusion, the reciprocal ST-segment depression extended significantly in V3 chest lead in the MVD group compared to the SVD group (−0.18 ± 0.07 mV vs. −0.1 ± 0.06, $P = 0.02$). **Conclusion:** The pattern of reciprocal ST-segment depression was more profound when the LAD was the culprit artery causing the anterior STEMI compared to the same case if the LAD was a part of MVD; this does not apply to the LCX and RCA when they were the culprit in cases of inferior STEMI where the MVD group showed more reciprocal ST-segment depression.

Keywords: Reciprocal, ST segment, ST-segment elevation myocardial infarction

Introduction

Electrocardiography (ECG) is the main tool for the diagnosis of ST-segment elevation myocardial infarction (STEMI) and should be performed quickly within 10 min from the first medical contact with a patient presenting with symptoms including typical chest pain suggestive of acute myocardial ischemia. The ECG criteria for the diagnosis of STEMI in the Fourth Universal Definition of Myocardial Infarction include new ST-elevation measured from the J-point in two contiguous leads. The cut-points are ≥1 mm in all leads other than leads

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V2–V3. In V2 and V3, the cut-points are ≥2 mm in males aged 40 years and older; ≥2.5 mm in males younger than 40 years; or ≥1.5 mm in females regardless of age.[1]

The pathophysiology of the elevation of the ST segment in STEMI is due to the current of injury flowing between ischemic and normally perfused myocardial regions.[2] These currents flow during the resting phase and also flow during the phase II of the action potential of the myocardial cells, and they give rise to TQ-segment depression and the true ST-segment elevation in direct-current electrical recordings.[3] The conventional 12-lead ECG machines use alternating current instead of direct current; the TQ- and ST-segment shifts are summed up to be recorded as an overall ST-segment elevation in the 12-lead surface ECG.[4]

The mechanism of reciprocal depression in the ST segment is influenced by multiple factors. First of all, an electrical mirror projection of the ST-segment elevation at a distance from the infarcted area,[5] and secondly, genuine ST-segment depression due to additional subendocardial ischemia at a distance from the infarction due to reduction of the coronary blood flow.[6]

Multi-vessel disease (MVD) subset of patients have more vague presentation on ECG, and sometimes, their ECG findings are confusing; they usually show less ST-segment elevation and profound and diffuse ST-segment depression compared to STEMI patients with single-vessel disease (SVD), involving occlusion of one coronary artery only, namely the infarct-related artery (IRA). Patients with STEMI and MVD may develop superadded subendocardial ischemia at a distance from the infarction. This could magnify the reciprocal ST-segment changes and also counteract the ST-segment elevation in the opposite infarcted region.

Methods

We performed a prospective, single-center, observational study at Ainshams University Hospitals, a tertiary referral hospital with a 24/7 primary percutaneous coronary intervention (PCI) service offered to all incoming STEMI patients. A total of 125 consecutive patients admitted from April 2014 to August 2015 from the emergency room (ER) with the diagnosis of acute STEMI were included. The ECG diagnosis of STEMI was based on the Fourth Universal Definition of Myocardial Infarction and the European Society of Cardiology (ESC) guidelines for STEMI diagnosis,[7] including clinical symptoms suggestive of myocardial ischemia, and the ECG criteria for diagnosis of STEMI in the Fourth Universal Definition of Myocardial Infarction including new ST-elevation measured from the J-point in two contiguous leads. The cut-points are ≥1 mm in all leads other than leads V2–V3. In V2 and V3, the cut-points are ≥2 mm in males aged 40 years and older; ≥2.5 mm in males younger than 40 years; or ≥1.5 mm in females regardless of age. Patients with ECGs that showed changes in the ST-segment secondary to bundle branch block or artificial ventricular paced rhythm were excluded, and patients with normal coronaries or those with nonsignificant lesions upon angiography were also excluded from the study.

The first 12-lead ECG was recorded within 10 min of the first medical contact and recorded as the admission ECG as per guideline recommendations.[7]

ST-segment deviations were measured at the J-point. Reciprocal ST-segment changes were identified as per guidelines published by the ESC and the American College of Cardiology, i.e., ST-segment depression ≥0.1 mV in any ECG lead other than aVR, while the cutoff value is different for leads V2 and V3 being only 0.05 mV. Two cardiologists unaware of the angiographic data revised the electrocardiographic parameters.

Primary PCI (PPCI) was performed in all patients within the first 60 min of presentation to our ER as per the guidelines of revascularization of STEMI patients presenting to PPCI-capable center.[7] The coronary angiograms were revised by two independent operators blinded to the ECG data. The IRA was agreed upon to be identified on the coronary angiogram as the major epicardial coronary artery showing signs of fresh total occlusion or by angiographic evidence of an intraluminal thrombus as seen in the diagnostic coronary injections prior to PPCI. Flow through the culprit artery was graded by using the thrombolysis in myocardial infarction (TIMI) criteria for grading coronary blood flow:[8] TIMI I flow: the epicardial coronary artery shows dye penetration without perfusion. The dye passes beyond the area of obstruction, but fails to opacify the entire coronary bed distal to the obstruction for the duration of the cine-angiographic filming sequence; TIMI II flow: partial epicardial coronary perfusion. The epicardial coronary artery shows dye passage across the obstruction and opacifies the coronary artery beyond the obstruction, provided that the rate of entry of dye into the coronaries distal to the obstruction and/or its rate of clearance is slower than its flow into or clearance from other areas that are not perfused by the occluded vessel; and TIMI III flow: the epicardial coronary artery shows complete perfusion. The antegrade flow into the coronary bed beyond the obstruction happens at the same pace as the antegrade flow into the bed proximal to the obstruction, and clearance of contrast material from the affected coronary territory is within the same pace as clearance from an unaffected or normal territory.

Any additional coronary stenosis of >70% as visually assessed by two experienced operators blinded to the clinical and ECG data was considered significant.[7] Patients were divided according to the major epicardial coronary IRA into left anterior descending (LAD) group, right coronary artery (RCA) group, and left circumflex coronary artery (LCX) group. In each vessel, we divided the proximal and mid-distal locations of the occlusion taking the first diagonal branch as a reference in the LAD group, the first obtuse marginal branch in the LCX group, and the halfway distance to the acute margin of the heart in cases of the RCA.

The study protocol was approved by the local hospital's medical ethics committee. The Ethical Committee of Ainshams University Hospitals approved the protocol of the study without

further modifications as the study does not involve administration of any new medication under trial or performing an additional procedure rather than the standard PPCI, which is the regular service offered as per the guidelines of clinical practice.

Patients undergoing PPCI procedure which is offered free of charge for all STEMI comers at our center were demanded to sign a consent of approval to the procedure itself including usage of their clinical data in the studies ongoing within our center so long as secrecy and discretion are guaranteed. All data were summarized and displayed as mean ± standard deviation for continuous variables and as number (percentage) of patients in each group for categorical variables. The *P* values for the categorical variables were calculated with the Chi-square test. Continuous variables were compared using the independent sample *t*-test. A two-tailed $P < 0.05$ was considered statistically significant for all analyses. All analyses were performed with the SPSS software (SPSS Inc., Chicago, Illinois, USA).

Results

The demographic and clinical characteristics of the 125 patients presenting clinically and by ECG diagnosis as acute STEMI are listed in Table 1.

Coronary angiography evidenced SVD in 61 patients (48%), two-vessel disease in 32 patients (26%), and three-vessel disease in 32 patients (26%). The percentage of patients with multi-vessel coronary artery disease (CAD) was homogeneously distributed among the three study groups [Table 2].

Regarding the anterior STEMI patients due to acute LAD occlusion, the magnitude of ST-segment elevation was expressed in millivolts, and the distribution of the ST-segment elevation within the ECG leads caused by acute occlusion of the proximal or mid-distal LAD segments was comparable in patients with SVD or MVD, showing a pattern of ST-segment elevation in leads I, aVL, V1 → V6 in the proximal LAD subgroup; ST-segment elevation in V1 → V4 in the mid-distal LAD subgroup; and reciprocal ST depression in leads II, III, and arteriovenous fistula (aVF); however the reciprocal ST-segment depression in leads III and aVF was significantly less in the MVD group compared to the SVD group, i.e., lead III (−0.08 ± 0.10 mV, interquartile range [IQR] [−0.04 to −0.12] vs. −0.19 ± 0.15, IQR [−0.08 to −0.26], $P = 0.015$) and aVF (−0.07 ± 0.06 mV, IQR [−0.01 to −0.13], vs. −0.15 ± 0.11, IQR [−0.1 to −0.21], $P = 0.02$) [Figure 1].

Regarding the inferior/inferolateral STEMI patients due to acute LCX occlusion, there were no isolated posterior MI patients in this study; the magnitude of ST-segment elevation was expressed in millivolts, and the distribution of the ST-segment elevation within the ECG leads caused by acute occlusion of the proximal or mid-distal LCX segments was comparable in patients with SVD or MVD, showing a pattern of ST-segment elevation in leads II, III, and aVF and a comparable pattern of reciprocal ST depression in V2 and V3, but the reciprocal ST-segment depression extended

Table 1: Demographics and clinical characteristics of the study groups

Variable	Major epicardial coronary artery affected, *n* (%)					
	LAD (65 patients)		LCX (14 patients)		RCA (46 patients)	
	Proximal (*n*=41)	Mid and distal (*n*=24)	Proximal (*n*=9)	Mid and distal (*n*=5)	Proximal (*n*=31)	Mid and distal (*n*=15)
Age	59±15	62±13	61±15	58±15	61±14	59±13
Gender (male)	29 (71)	15 (63)	7 (78)	2 (40)	19 (61)	8 (53)
Smoking	21 (51)	11 (46)	6 (67)	1 (20)	17 (55)	6 (40)
Hypertension	27 (66)	18 (75)	7 (78)	2 (40)	22 (71)	7 (47)
Diabetes mellitus	23 (56)	15 (63)	5 (56)	2 (40)	19 (61)	5 (33)
Dyslipidemia	24 (59)	13 (54)	4 (44)	3 (60)	16 (52)	8 (53)
Positive family history	11 (27)	6 (25)	3 (33)	2 (40)	8 (26)	4 (27)

Data are presented as mean±SD or *n* (%). SD: Standard deviation, LAD: Left anterior descending, LCX: Left circumflex coronary artery, RCA: Right coronary artery

Table 2: Number and distribution of major epicardial coronary arteries affected

Number of major epicardial coronary arteries affected	Major epicardial coronary artery affected					
	LAD (65 patients)		LCX (21 patients)		RCA (39 patients)	
	Proximal (*n*=41)	Mid and distal (*n*=24)	Proximal (*n*=11)	Mid and distal (*n*=10)	Proximal (*n*=25)	Mid and distal (*n*=14)
Single-vessel disease (*n*=61)	25 (61)	11 (46)	5 (45.5)	3 (30)	11 (44)	6 (43)
Two-vessel disease (*n*=32)	9 (22)	8 (33)	1 (9)	3 (30)	7 (28)	4 (28.5)
Three-vessel disease (*n*=32)	7 (17)	5 (21)	5 (45.5)	4 (40)	7 (28)	4 (28.5)

Data are presented as *n* (%). LAD: Left anterior descending, LCX: Left circumflex coronary artery, RCA: Right coronary artery

significantly in V4 chest lead in the MVD group compared to the SVD group (−0.16 ± 0.08 mV, IQR [−0.12 to −0.19], vs. −0.1 ± 0.04, IQR [−0.08 to −0.12], $P = 0.025$) [Figure 2].

Regarding the inferior/inferolateral STEMI patients due to acute RCA occlusion, the magnitude of ST-segment elevation was expressed in millivolts, and the distribution of the ST-segment elevation within the ECG leads caused by acute occlusion of the proximal or mid-distal RCA segments was comparable in patients with SVD or MVD, showing a pattern of ST-segment elevation in leads II, III, and aVF and a comparable pattern of reciprocal ST depression in leads I, aVL, and V2, but the reciprocal ST-segment depression extended significantly in V3 chest lead in the MVD group compared to the SVD group (−0.18 ± 0.07 mV, IQR [−0.15 to −0.21], vs. −0.1 ± 0.06, IQR [−0.08 to −0.12], $P = 0.02$) [Figure 3].

Discussion

The reciprocal ST-segment depression seen in the ECG leads recording electrical activity from noninfarcting regions has been reported in previous studies on acute myocardial ischemia.[9,10] The mechanism is complex with multiple interplaying factors.[11,12]

MVD subset of patients have more vague presentation on ECG, and sometimes, their ECG findings are confusing; they usually show less ST-segment elevation and profound and diffuse ST-segment depression compared to STEMI patients with SVD involving occlusion of one coronary artery only,

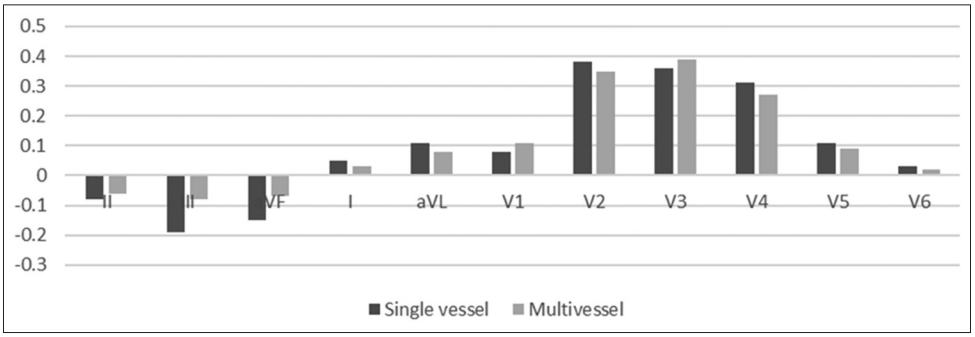

Figure 1: Pattern of ST-segment deviation in the electrocardiography leads in patients presenting with acute left anterior descending occlusion. Values represent the mean ST-segment deviations in millivolts

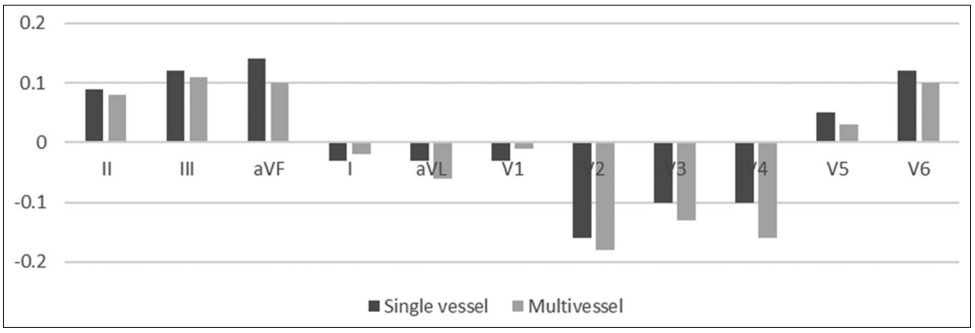

Figure 2: Pattern of ST-segment deviation in the electrocardiography leads in patients presenting with acute left circumflex coronary artery occlusion. Values represent the mean ST-segment deviations in millivolts

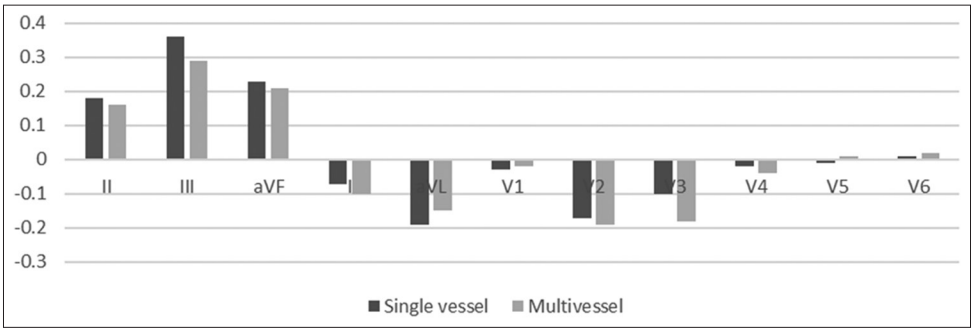

Figure 3: Pattern of ST-segment deviation in the electrocardiography leads in patients presenting with acute right coronary artery occlusion. Values represent the mean ST-segment deviations in millivolts

namely the IRA. Patients with STEMI and multi-vessel CAD involving coronary stenosis approaching the critical 90% level of luminal stenosis may develop superadded subendocardial ischemia at a distance from the infarction, and this could magnify the reciprocal ST-segment changes and counteract the ST-segment elevation in the opposite infarcted region.[13]

The pathological mechanisms include an electrical mirror projection of the ST-segment elevation observed in the reciprocal (mirroring) leads,[5] and/or in combination with a true ischemic ST-segment depression caused by additional subendocardial ischemia remote from the infarction due to reduction of the coronary blood flow.[6]

Our study showed that, regarding the LAD occlusion, the reciprocal ST-segment depression in leads III and aVF was significantly less in the MVD group compared to the SVD group, while regarding the LCX occlusion, the reciprocal ST-segment depression extended significantly in V4 chest lead in the MVD group compared to the SVD group, and finally regarding the RCA occlusion, the reciprocal ST-segment depression extended significantly in V3 chest lead in the MVD group compared to the SVD group.

The mechanism of electrical mirroring[5] may explain our results regarding LAD being the IRA in the subset of patients presented with anterior STEMI; the SVD group in our study showed more profound reciprocal ST-segment depression compared to the MVD group, this could be explained by the physical fact of mirroring the electrical changes, where the ST-elevation magnitude is directly mirrored as a negative deflection in the reciprocal leads in the SVD group without being affected by another ischemic electrical deflection of the ST segment from another territory. While in the MVD group, transmural ischemia in opposite territories may attenuate the reciprocal ST-segment changes in the anterolateral chest leads,resulting in a lesser amplitude of negative deflections in the inferior leads.[14,15]

The ischemia at a distance explanation[6] can verify the pattern of observed reciprocal ST-segment changes in the LCX and RCA groups of patients in our study; this is explained by the volume and distribution of coronary collateral vessels that reduce the extent of myocardial ischemic burden caused by acute blockage of a major epicardial vessel. In the case of extensive MVD, when those collaterals are fed by the IRA and feeding another critically stenosed major epicardial vessel, the distribution of reciprocal ST depression will be wider and involving more leads with more negative amplitude due to more profound ischemia at a distance once the IRA is totally occluded.

Conclusion and Study Limitations

Data from this study show two major important results; first of all, the pattern of reciprocal ST-segment depression was more profound when the LAD was the IRA causing the anterior STEMI compared to the same case if the LAD was a part of MVD, and the second result is that this does not apply to the LCX and RCA when they were the IRA in cases of inferior STEMI; still the MVD group showed more reciprocal ST-segment depression.

There are several important limitations in this study. This was a single-center, nonrandomized observational study although we included consecutive patients. Future larger studies on bigger samples are required to confirm these preliminary results.

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Conflicts of interest

There are no conflicts of interest.

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