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Acute Anterior Wall Myocardial Infarction Entailing ST-Segment Elevation in Lead V_1: Electrocardiographic and Angiographic Correlations

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Summary

Background: The correlation between ST elevation in lead V_1 during anterior wall acute myocardial infaction (AMI) and the culprit lesion site in the left anterior descending (LAD) coronary artery is poor.

Hypothesis: The study was undertaken to assess the electrocardiographic (ECG) characteristics and angiographic significance of ST-segment elevation in lead V_1 during anterior wall acute myocardial infarction (AMI).

Methods: Data from 115 patients with anterior wall AMI, who underwent coronary angiography within 14 days of hospitalization, were studied. The admission 12-lead ECG was examined and the coronary angiogram was evaluated for the nature of the conal branch of the right coronary artery (RCA) and for the culprit lesion site in the left anterior descending (LAD) coronary artery.

Results: Mean ST-segment deviation and the frequency of patients with ST-segment elevation >0.1 mV were significantly lower in lead V_1 than in lead V_2 (0.136 ± 0.111 mV vs. 0.421 ± 0.260 mV, and 37 vs. 96%, for leads V_1 and V_2, respectively). A small conal branch not reaching the interventricular septum (IVS) was more prevalent among patients with ST-segment elevation >0.1 mV in lead V_1 (67%), whereas a large conal branch was more prevalent in patients with ST-segment deviation (1 mV in that lead (83%, $p<0.001$). No relation was found between ST-segment deviation in lead V_1 during anterior wall AMI and the culprit lesion site in the LAD.

Conclusion: ST-segment elevation in lead V_1 during first anterior wall AMI was found in one third of the patients, and its magnitude was lower than that in the other precordial leads. ST-segment elevation in lead V_1 favors the presence of a small conal branch of the RCA that does not reach the IVS.

Key words: electrocardiogram, anterior wall acute myocardial infarction, V_1 ST segment, coronary angiography, conal branch

Introduction

During anterior wall acute myocardial infarction (AMI), the electrocardiogram (ECG) shows ST-segment elevation in leads facing the anterior wall and sometimes also in leads facing the lateral wall, the inferior wall,[1] or even the right ventricle.[2,3] The significance of ST-segment elevation in lead V_1 during anterior wall AMI is still unclear. Traditionally, ST-segment elevation in lead V_1 during anterior wall AMI has been regarded as indicative of anteroseptal involvement, although this has been questioned lately.[4] Most patients with anterior wall AMI have ST-segment elevation in leads V_2–V_4, however, fewer than 50% of the patients present ST-segment elevation in lead V_1.[5] Recently, in a small group of patients with anterior wall AMI,[6] we found an association between ST-segment elevation in leads V_1 and V_{3R}, and the absence of a large conal branch of the right coronary artery (RCA). The purpose of the present study was to investigate retrospectively the ECG characteristics and angiographic significance of ST-segment deviation in lead V_1 during anterior wall AMI in a larger group of patients.

Methods

Study Patients

All patients who were admitted from July 1993 to March 1996 to the coronary care unit at our institution with first anterior wall AMI, and who underwent coronary angiography within 14 days of hospitalization, were evaluated retrospec-

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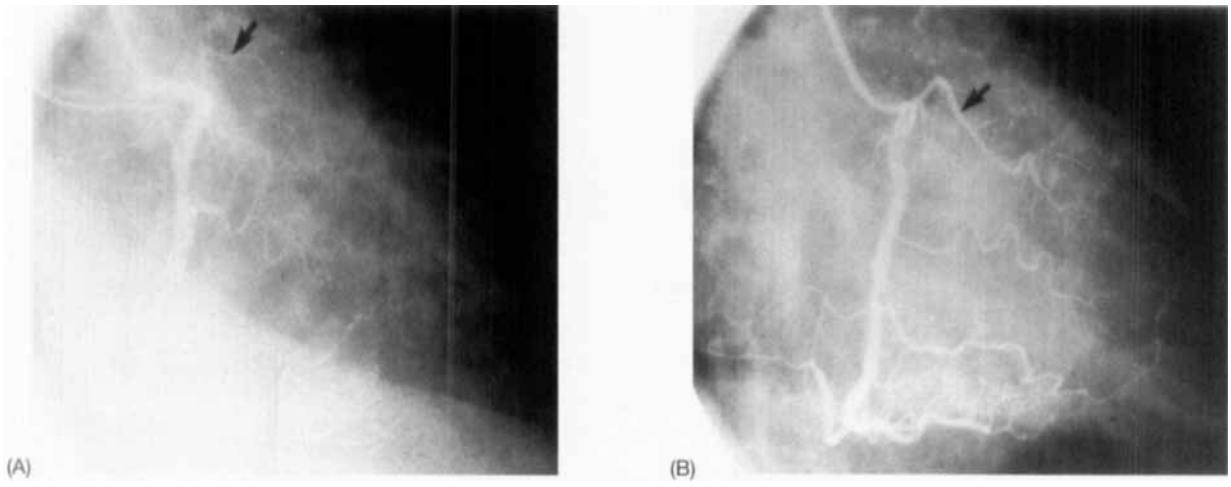

FIG. 1 Coronary angiogram: Injection to the right coronary artery in the right anterior oblique projection. (A) A small conal (black arrow) that does not reach the interventricular septum (IVS). (B) A large conal branch that reaches the IVS.

tively. The diagnosis of anterior wall AMI was established by the presence of typical precordial pain lasting more than 30 min, evolving characteristic ECG abnormalities that included ST-segment elevation >0.1 mV in at least two consecutive precordial leads (V_1–V_4) and an increase in serum creatine kinase levels to more than twice the upper limit of normal. Patients with electrocardiographic evidence or history of previous myocardial infarction, acute or chronic bundle-branch block, ECG evidence of left ventricular hypertrophy, or advanced ECG stages of infarction (Q waves or inverted T waves) were excluded from the study. No patient received digitalis before admission.

Electrocardiographic Evaluation

Standard 12-lead ECGs were recorded at admission using a paper speed of 25 mm/s and a calibration of 1 mV = 10 mm. All ST-segment deviations from the isoelectric line, as determined by a line drawn between subsequent TP segments, were measured manually to the nearest 1.0 mm at 0.08 s after the J point in every lead. All ECGs were evaluated separately by two investigators (T.B. and S.S.) blinded to the angiographic findings.

Coronary Angiographic Evaluation

Selective coronary cineangiography was performed within 14 days of admission by the Judkins technique. Cine recordings of the left coronary system were made in at least four projections—right anterior oblique (RAO), left anterior oblique (LAO), anteroposterior cranial, and caudal. The right coronary system was screened in the RAO and LAO projections at least. When more than one lesion was present in the left anterior descending coronary artery (LAD), the culprit lesion site was determined by the angiographic appearance of either a residual thrombus or an ulcerated plaque. The location of the culprit lesion in the LAD was determined as proximal or distal to the origin of the first major septal branch. Analysis of the right coronary system included the type of the conal branch (small or large), best seen in the RAO projection, according to the following definitions: small branch: <0.5 mm in diameter, not reaching the interventricular septum (IVS); and large branch: >0.5 mm in diameter, reaching the IVS (Fig. 1). The films were interpreted by two experienced angiographers blinded to the ECG findings (I.H. and A.S.). In case of a discrepancy, another reading was done by a third investigator.

Statistical Analysis

All analyses were done using the Statistical Analysis System (SAS) software. A probability value <0.05 was considered statistically significant. To compare the mean of continuous variables between the groups, the Student's *t*-test was performed. The chi-square test was used to compare between the distribution of patients in the various groups by the amount of ST-segment deviation in lead V_1, for the presence of a large conal branch, and for the lesion site in the LAD (proximal or distal to the branching of the first septal artery).

Results

Patient Characteristics

In all, 115 patients (17 women, 98 men, mean age 59 ± 11 years) were evaluated. The conal branch was not identified in 19 patients; these patients were excluded from the study. Of the remaining 96 patients, 7 (7%) and 11 (11%) had primary and rescue angioplasty, respectively. The rest was referred for coronary angiography because of postinfarction angina, residual ischemia (demonstrated by positive submaximal ergometry or thallium 201 radionuclide scan), or non-Q-wave AMI.

FIG. 2 V_1 and V_2 ST-segment changes during anterior wall acute myocardial infarction: (A) Electrocardiogram (ECG) of a patient with ST-segment elevation in lead V_2 without concomitant changes in lead V_1. (B) ECG of a patient showing concomitant ST-segment elevation in leads V_2 and V_1.

Electrocardiographic Findings

ST-segment elevation ≤ 0.1 mV in lead V_1 was observed in 60 patients [<0.1 mV in 19 (20%), equal to 0.1 mV in 41 (43%)] and >0.1 mV in 36 (37%) patients [>0.2 mV in 11 (11%) patients], (Fig. 2). ST-segment elevation >0.1 mV in leads V_2, V_3, and V_4 was observed in 92 (96%), 86 (90%), and 71 (74%) patients, respectively (Fig. 3). Mean ST-segment elevation was significantly lower in lead V_1 (0.136 ± 0.111 mV) compared with leads V_2 (0.421 ± 0.260 mV), V_3 (0.453 ± 0.349 mV), and V_4 (0.305 ± 0.291 mV), $p<0.001$ (Fig. 4). When only the patients with ST-segment elevation >0.1 mV in lead V_1 (n = 36) were considered, mean ST-segment elevation was also significantly lower in lead V_1 (0.253 ± 0.090 mV) than in lead V_2 (0.542 ± 0.300 mV), ($p<0.001$). There was a good correlation between the magnitude of ST-segment elevation in lead V_3 and the magnitude of ST-segment elevation in lead V_2 (Fig. 5A). In contrast, the correlation between ST-segment amplitudes in lead V_1 and V_2 was poor (Fig. 5B).

Coronary Angiographic Findings in Relation to ST-Segment Elevation in Lead V_1

The conal branch was defined as large in 62 patients and small in 34 patients. The prevalence of a large conal branch was significantly higher in the group of patients with ST-segment deviation ≤ 0.1 mV in lead V_1 than in patients with ST-segment >0.1 mV in lead V_1 (Table I). The sensitivity of ST-segment elevation of >1 mm and >2 mm for detecting a small conal branch was 80.6 and 97%, respectively; specificities were 70.6 and 26%, respectively. The positive predictive value of ST-segment elevation ≤ 0.1 mV in lead V_1 for the presence of a large conal branch was 83.3%. Mean ST-segment elevation in lead V_1 was significantly lower in the presence of a large conal branch than a small one (0.099 ± 0.085 mV vs. 0.225 ± 0.115 mV, respectively, $p<0.01$). When the magnitude of ST-segment elevation in lead V_1 was plotted against the ST-segment elevation in lead V_2, it was found that irrespective of the ST segment amplitude in lead V_2, patients with a larger conal branch had less ST-segment elevation in lead V_1 than patients with a small conal branch. The culprit lesion in the LAD was proximal to the bifurcation of the first septal branch in 36 patients and distal to it in 60 patients. The relationship between mean ST-segment elevation in lead V_1 and the LAD culprit lesion site with regard to the bifurcation of the first septal branch was not statistically significant (0.162 ± 0.111 mV vs. 0.124 ± 0.109 mV for proximal and distal obstruction, respectively).

Discussion

The correlation between ST-segment elevation in the precordial leads V_1–V_4 and anterior wall AMI caused by LAD artery obstruction is good.[7] However, the angiographic significance of ST-segment elevation in lead V_1 during evolving an-

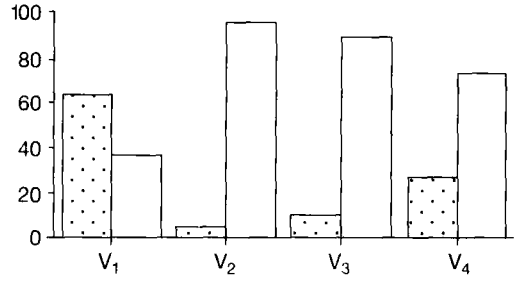

FIG. 3 ST-segment deviations in the precordial leads V_{1-4} during anterior wall acute myocardial infarction (% of patients). ☐ = ST <1 mm, ☐ = ST >1 mm.

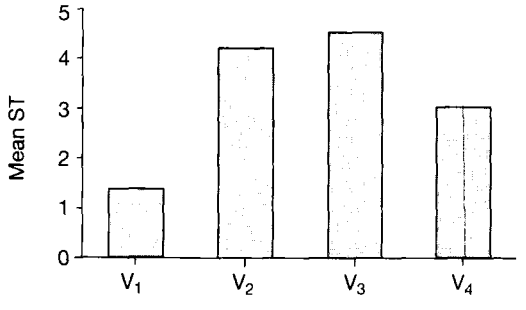

FIG. 4 Mean ST-segment elevation in the precordial leads V_1–V_4 during anterior wall AMI.

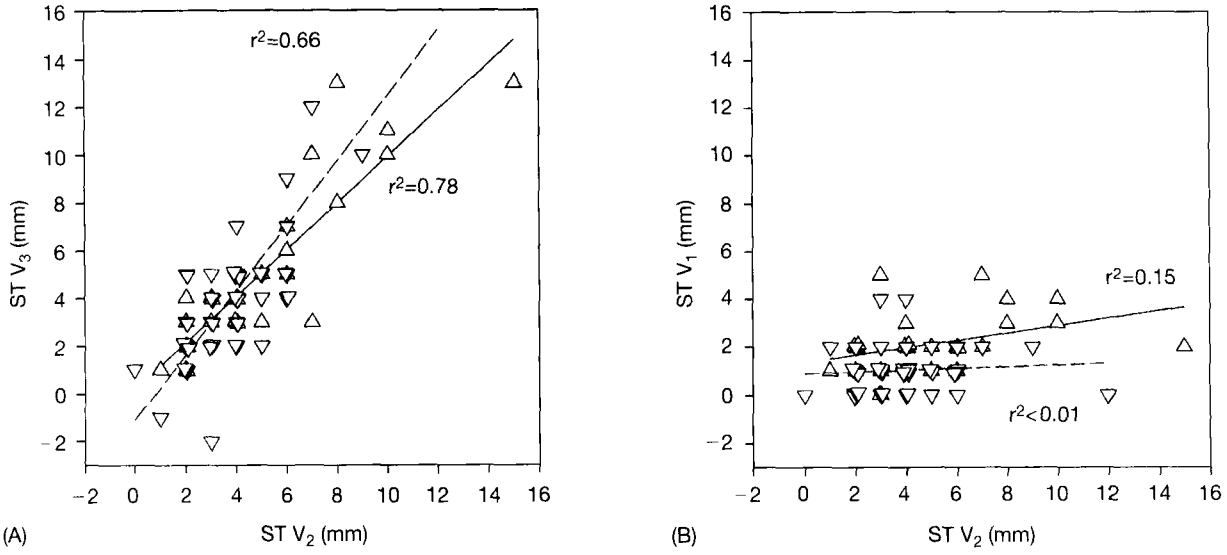

FIG. 5 (A) ST-segment amplitude in lead V_3 plotted against ST-segment amplitude in lead V_2 in patients with a large and a small conal branch. (B) ST-segment amplitude in lead V_1 plotted against ST-segment amplitude in lead V_2 in patients with a large and a small conal branch. △ = Small conal branch, —— = regression line, ▽ = large conal branch, – – – = regression line.

terior wall AMI is as yet unclear. Recently, in a study performed on a small group of patients (n = 28) with anterior wall AMI,[6] ST-segment elevation in lead V_1 was found to be associated with the presence of a small conal branch not reaching the interventricular septum. We sought to determine the characteristics and significance of ST-segment elevation in lead V_1 during evolving anterior wall AMI using a larger group of patients (n = 96). The mean ST-segment deviation was found to be significantly lower in lead V_1 than in the other precordial leads (V_2–V_4). Moreover, the correlation between the magnitude of ST-segment elevation in leads V_1 and V_2 was poor, in contrast to the good correlation between ST-segment amplitude in V_3 and V_2 (Fig. 4). ST-segment elevation > 0.1 mV in lead V_1, found in only 37% of the patients, was clearly associated with the presence of a small conal branch and unrelated to the site of the culprit lesion in the LAD and to the magnitude of ST-segment elevation in lead V_2.

Characteristics of ST-Segment Elevation in Lead V_1 during Evolving Anterior Wall AMI

In patients with inferior wall AMI, ST-segment elevation > 0.1 mV in lead V_1, associated with ST-segment elevation in the right precordial leads (V_{3R}–V_{4R}), suggests right ventricular involvement.[8] The amplitude of the ST-segment elevation in the right precordial leads during right ventricular involvement is usually small (< 2.5 mm) and short lived,[8] probably reflecting the lower muscle mass of the right ventricle. Aldrich *et al.*,[5] studying the ECG of patients with anterior wall AMI, found mean ST-segment deviation in lead V_1 to be significantly lower than in the other precordial leads. Birnbaum *et al.*,[9] postulated that ST-segment elevation in lead V_1 does not correlate with the site of the culprit lesion in the LAD (with regard to the bifurcation of the first septal branch). In the present study, in accordance with the above mentioned studies, mean ST-segment deviation as well as the frequency of ST-segment elevation > 1 mV in lead V_1 were found to be significantly lower than those in lead V_2. No such difference was observed among the other precordial leads: V_2, V_3, and V_4.

The magnitude of the electric deflection in the precordial leads depends on the proximity of each lead to the surface of

TABLE I Comparison of angiographic data based on ST-segment elevation in lead V_1

	Angiographic data					
V_1 ST-deviation	BC	SC	p Value	PS	DS	p Value
≤ 1 mm	50	10		18	42	
(n = 60)	(81%)	(29%)		(50%)	(70%)	
> 1 mm	12	24		18	18	
(n = 36)	(19%)	(71%)	< 0.001	(50%)	(30%)	p = 0.09
Total	62	34		36	60	
≤ 2 mm	60	25		32	53	
(n = 85)	(97%)	(74%)		(89%)	(88%)	
> 2 mm	2	9		4	7	
(n = 11)	(3%)	(2%)	< 0.001	(11%)	(12%)	p = 0.80
Total	62	34		36	60	

Data are expressed as number (%) of patients.

Abbreviations: PS = lesion proximal to the origin of the first septal branch, DS = lesion distal to the origin of the first septal branch, BC = big conal branch reaching the interventricular septum (IVS), SC = small conal branch not reaching the IVS.

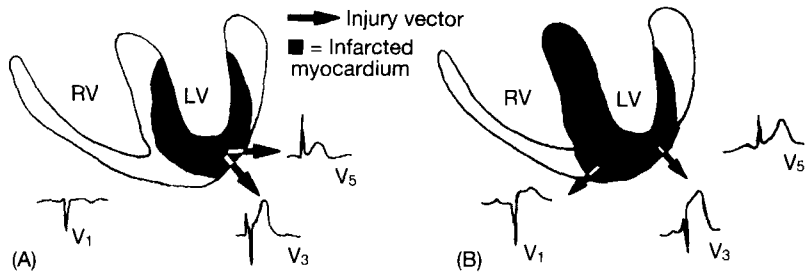

FIG. 6 Illustration showing the proposed mechanism for the ST-segment elevation in lead V_1 during acute anterior wall acute myocardial infarction: (A) with no ST elevation in lead V_1 (no transseptal involvement), (B) with ST elevation in lead V_1 (true transseptal involvement). RV = right ventricle, LV = left ventricle.

the heart and on the heart's muscle mass that the electrode overlies. According to the "solid angle" theory,[10] two adjacent precordial leads can successfully distinguish between two different myocardial areas. Therefore, the significantly lower mean ST-segment elevation in lead V_1 may suggest that this lead represents a distinct area probably related to the right rather than to the left ventricle. The recently described relationship between ST-segment elevation in leads V_1 and V_{3R} during anterior wall AMI,[6] further supports this assumption.

ST-segment elevation in the right precordial leads and in V_1 during inferior wall AMI implies that the obstruction in the RCA is proximal to the branching of the right ventricular acute marginal branches.[11] The angiographic significance of ST-segment elevation in lead V_1 during anterior AMI is as yet unclear. In our study, in accordance with previous works,[4,6,9] no relationship was observed between ST-segment elevation in lead V_1 and the presence of a culprit lesion in the LAD proximal to the origin of the first septal branch (Table I). We assumed that the absence of ST-segment elevation in lead V_1 during anterior wall AMI probably implies that this lead faces the myocardial area which is not always ischemic during LAD occlusion (Fig. 6A).

Interventricular Septum Blood Supply

The blood supply to the IVS is complex and at times may involve all three major coronary vessels. In 85% of patients, the posterior IVS is supplied by the posterior descending coronary artery (PDA) branching from the RCA ("right dominance"), in 10% the PDA branches from the circumflex coronary artery ("left dominance"), and in the remainder the posterior IVS is supplied by arteries branching from both the RCA, and the circumflex ("co-dominance" or "balanced circulation").[12,13] James[14] found that the basal portion of the IVS is supplied by septal branches of the LAD, alone or together with the conal branch of the RCA. The conal branch, the first artery branching from the RCA pierces the anterior wall of the right ventricle and crosses subendocardially on the supraventricular crest toward the anterior IVS.[15] This artery rarely develops atherosclerosis[16] and has been found to be a source of excellent collaterals to the LAD.[17] The presence of the conal branch may have some ethnic predilection, for it was seen more frequently (85%) in patients from northwest India[15] than in patients from Western countries.[13] In 30 to 50% of patients, the conal branch is replaced by the conal artery which originates from a distinct ostium in the right coronary sinus.[12,18] In the present study, the conal branch was not identified in 19 patients (17%) who were excluded from the study. A small conal branch was found in 67% of the patients with ST-segment >1 mV in lead V_1 and a large conal branch in 83% of the patients without ST-segment elevation in that lead. We hypothesized that ST-segment elevation in lead V_1 during anterior wall AMI represents ischemic injury to the right side of the basal portion of the IVS (Fig. 6B). The presence of a large conal branch probably protects the right side of the IVS; this is manifested by the absence of ST-segment elevation in lead V_1. In the case of a small conal branch, both sides of the IVS are probably supplied by branches of the LAD, and therefore the septum is left unprotected during LAD occlusion. This is reflected electrocardiographically by ST-segment elevation in lead V_1.

Conclusions and Clinical Implications

ST-segment elevation in lead V_1 was found in 37% of the patients with anterior wall AMI, and its magnitude was lower than in leads V_2–V_4. The absence of ST-segment elevation in lead V_1 during anterior wall AMI suggests the presence of a large conal branch probably protecting the septum from a transseptal infarction. ST-segment elevation in lead V_1 during anterior wall AMI may suggest ischemic involvement of the right paraseptal area because of the absence of collateral circulation from the right conal branch; it has to be determined whether ST-segment elevation in lead V_1 during anterior wall AMI has adverse prognostic implications.

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