

Does the Electrocardiographic Pattern of "Anteroseptal" Myocardial Infarction Correlate With the Anatomic Location of Myocardial Injury?

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The current electrocardiographic (ECG) definition of anteroseptal acute myocardial infarction (AMI) is a Q wave or QS wave >0.03 second in leads V_1 to V_3 , with or without involvement of lead V_4 . To verify whether there is a correlation between the ECG pattern of anteroseptal AMI and the location of an AMI, we compared ECG, echocardiographic, and cardiac catheterization findings of 80 patients who fit the traditional definition of anteroseptal AMI. We found that 48 of 52 patients (92%) who presented with ST elevation in leads

V_1 to V_3 had an anteroapical infarct and a normal septum. The culprit narrowing was more frequently found (in 85% of patients) in the mid to distal left anterior descending artery. We conclude that there is no correlation and that the ECG pattern traditionally termed anteroseptal AMI should be called an anteroapical AMI; the term anteroseptal AMI should be defined as extensive anterior wall AMI associated with diffuse ST changes involving the anterior, lateral, and occasionally, inferior leads.
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Cardiologists have found standard 12-lead electrocardiograms useful for diagnosing acute myocardial infarction (AMI) and healed infarcts,¹⁻³ and for localizing an infarct within subdivisions of the diaphragmatic, posterior, and anterior walls.⁴⁻⁶ The current electrocardiographic (ECG) definition of anteroseptal AMI is a Q wave or QS wave >0.03 second in leads V_1 to V_3 , with or without involvement of lead V_4 .⁷⁻⁹ If one assumes that this definition is valid, then most patients with anteroseptal AMI should have cardiac catheterization findings of acutely abnormal anteroseptal wall motion and a greater incidence of either high-grade stenosis or occlusion of the proximal left anterior descending artery (LAD). We wanted to verify whether there is such a correlation between the ECG pattern of anteroseptal AMI, the location of an AMI, and the site of the LAD narrowing.

METHODS

Between August 1990 and November 1992, of all patients consecutively admitted to the coronary care unit at our institution with a diagnosis of anteroseptal AMI, 80 (18 women and 62 men, 46 to 82 years) were enrolled in a study comparing electrocardiographic, echocardiographic, and cardiac catheterization findings. All patients had the typical increase in cardiac enzymes associated with an infarct.

The following criteria were established for inclusion: (1) first myocardial infarction; (2) not a candidate for either thrombolysis or emergency coronary angioplasty on presentation (however, these patients received intravenous heparin and other supportive medications); and

(3) echocardiographic images that adequately demonstrated the entire myocardium. Patients were excluded for the following criteria: (1) previous myocardial infarction; (2) acute or chronic complete left or right bundle branch block; (3) an extended original infarct or an additional infarct in a different location; (4) mortality within 24 hours; and (5) clinical evidence of early, spontaneous reperfusion (within the first 2 hours of admission).

Definitions: Anteroseptal AMI was defined as an acute ST-segment elevation >0.1 mV in leads V_1 to V_3 . Q-wave AMI was defined as development of a Q wave or QS wave >0.03 second. Non-Q-wave infarction was defined as evolution of inverted T waves in leads V_1 to V_3 . The typical increase in cardiac enzymes was present in all patients. Patients with acute ST changes in leads V_1 to V_3 and nonspecific ST-segment and T-wave changes in lead V_4 were analyzed as a separate group.

Study design: According to the protocol, within 24 hours of admission, patients with anteroseptal AMI received a standard 2-dimensional echocardiogram in 3 views: parasternal long- and short-axis views and an apical 4-chamber view. Only those whose entire myocardium could be visualized were included in our study. The subgroup of patients who had acute ST changes in leads V_1 to V_3 and underwent left ventricular angiography within 7 days of their infarct were analyzed for wall motion abnormality using both echocardiography and angiography.

Echocardiographic parameters: We evaluated the contractility of the apex, anterior wall, lateral wall, posterior wall, and septum using the following categories: normal, hypokinetic, akinetic, or dyskinetic. We divided the septum into 3 segments, with the apical aspect of the septum considered as part of the apex. Only abnormal wall motion of the remaining two thirds of the septum was considered evidence of true septal injury. Two independent echocardiographers, blinded to all information about the patient's clinical data, interpreted the echocardiographic studies.

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TABLE I Echocardiographic Findings of Segmental Wall Motion During AMI

Leads	Location of ST Elevation											
	Apex Wall Motion				Anterior Wall Motion				Septal Wall Motion			
	A (no.)	D (no.)	H (no.)	N (no.)	A (no.)	D (no.)	H (no.)	N (no.)	A (no.)	D (no.)	H (no.)	N (no.)
V ₁ -V ₃ (n = 52)	11 (21)	1 (2)	40 (77)	0	4 (8)	0	44 (85)	4 (8)	1 (2)	0	3 (6)	48 (92)
V ₁ -V ₄ (n = 28)	12 (43)	4 (14)	12 (43)	0	8 (29)	0	19 (68)	1 (4)	4 (14)	0	7 (25)	17 (61)
Total (n = 80)	23 (29)	5 (6)	52 (65)	0	12 (15)	0	63 (79)	5 (6)	5 (6)	0	10 (13)	65 (81)
p Value	<0.05	NS	<0.05	NS	<0.05	NS	<0.05	NS	NS	NS	<0.05	<0.002

A = akinetic; AMI = acute myocardial infarction; D = dyskinetic; H = hypokinetic; N = normal; p Value = comparing wall motion between the 2 groups of patients.

TABLE II Left Ventricular Angiographic Findings of Segmental Wall Motion Approximately Seven Days After AMI

Leads	Location of ST Elevation											
	Apex Wall Motion				Anterior Wall Motion				Septal Wall Motion			
	A (no.)	D (no.)	H (no.)	N (no.)	A (no.)	D (no.)	H (no.)	N (no.)	A (no.)	D (no.)	H (no.)	N (no.)
V ₁ -V ₃ (n = 35)	4 (11)	3 (9)	23 (66)	5 (14)	3 (9)	0	26 (74)	6 (17)	1 (3)	0	1 (3)	33 (94)
V ₁ -V ₄ (n = 9)	1 (11)	2 (22)	6 (67)	0	2 (22)	0	7 (78)	0	1 (11)	1 (11)	1 (11)	6 (67)
Total (n = 44)	5 (11)	5 (11)	29 (66)	5 (11)	5 (11)	0	33 (75)	6 (14)	2 (5)	1 (2)	2 (5)	39 (89)
p Value	NS	NS	NS	<0.05	NS	NS	NS	<0.05	NS	NS	NS	<0.05

Abbreviations as in Table I.

Cardiac catheterization: Patients were referred for cardiac catheterization with the following conditions: (1) ECG findings of non-Q-wave AMI; (2) presentation of postinfarct angina; or (3) ischemia induced at the pre-discharge, submaximal exercise test. All cardiac catheterizations consisted of left heart catheterization, coronary angiography, and left ventriculography in both the 30° right and 60° left anterior oblique projections. We evaluated wall motion of the apex, anterior wall, inferior wall, posterior wall, anterobasal aspect, and the septum using the same categories as for the echocardiographic study: normal, hypokinetic, akinetic, or dyskinetic. Two independent angiographers interpreted the catheterization studies for wall motion abnormality and for description of coronary artery stenosis.

Statistics: The 2-tailed Student's *t* test was used to analyze paired and unpaired data and the chi-square test was used for multivariate analysis. A probability value ≤ 0.05 was considered statistically significant. The interobserver average variability for echocardiographic evaluation was 0.87 and for cardiac catheterization evaluation, it was 0.90.

RESULTS

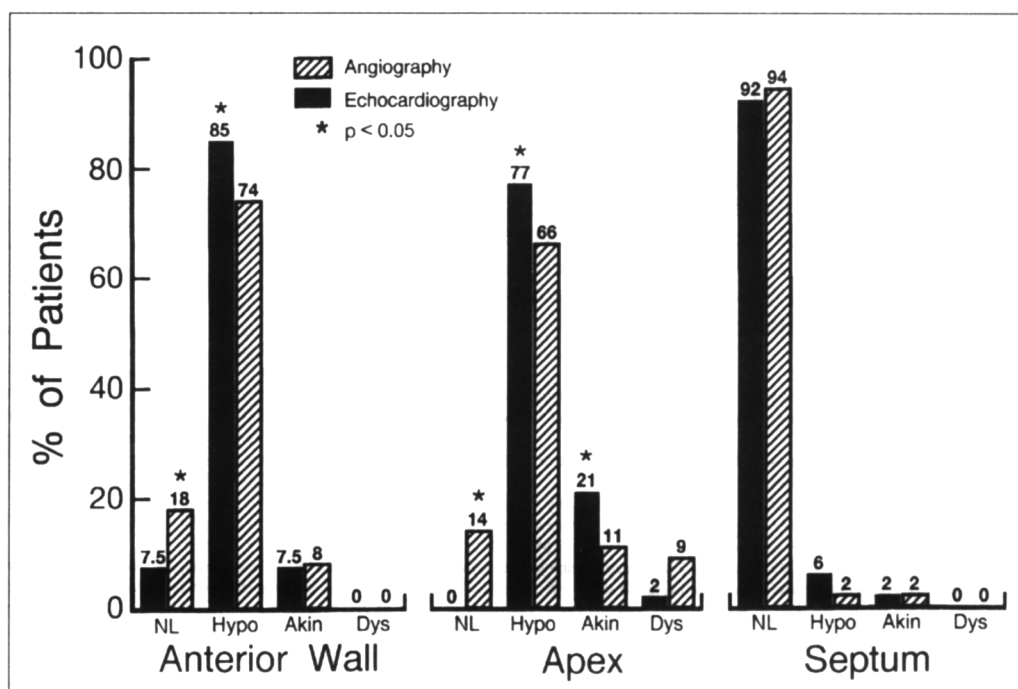
Electrocardiographic analysis: Eighty patients were included in the study; of these, 69 developed Q-wave anteroapical AMI and 11 were categorized as patients with non-Q-wave AMI. Of these 80 patients, 52 presented with ECG findings of acute ST-segment elevation (>1 mm) in leads V₁ to V₃ alone; the remaining 28

also presented with minimal ST-segment elevation (<1 mm) in lead V₄. Only 44 patients underwent cardiac catheterization within the first 7 days of the AMI; therefore, we were able to analyze the wall motion abnormalities related to the ECG presentation and type of AMI by using echocardiography in all patients and by using both echocardiography and angiography in 44 patients.

Echocardiography: When acute ST changes in all patients were compared with wall motion abnormality, the septum was normal in 65 patients (81%) (Table I). The anterior wall was hypokinetic in 63 patients (79%), with akinesia in 12 (15%). The apex was abnormal in all patients, with hypokinesia in 52 patients (65%), akinesia in 23 (29%), and dyskinesia in 5 (6%). Table I also shows the subgroup analysis: patients with ST elevations in leads V₁ to V₄ had more extensive injury to the anterior wall ($p < 0.05$) and apex ($p < 0.05$), and more septal injury ($p < 0.002$) than did those with ST elevations in leads V₁ to V₃ alone. In the latter group, 48 of 52 (92%) had normal septal wall motion, but had myocardial injury in the anteroapical wall. All had abnormal wall motion in the apex.

Angiography: Table II shows that of the 44 patients who underwent left ventricular angiography within 7 days of their AMI, 35 had acute ST changes in leads V₁ to V₃ and 9 in leads V₁ to V₄. Because these 9 patients were considered too small a group for statistical analysis, only the 35 in the other group were analyzed for wall motion abnormality using both echocardiography and angiography. Left ventriculographic findings of these 35

FIGURE 1. Comparison of echocardiographic and angiographic wall motion abnormality in 35 patients with acute ST elevation in leads V_1 to V_3 . Akin = akinesia; Dys = dyskinesia; Hypo = hypokinesia; NL = normal.



patients showed normal motion of the septum in 33 patients (94%), abnormal wall motion in the apex in 30 patients (86%), and in the anterior wall in 29 patients (83%). Wall motion of the apex and anterior wall improved to normal after the AMI event in 14% and 17%, respectively (Table II).

The dual comparison method used for patients with ST elevation in leads V_1 to V_3 showed similar findings of normal septal wall motion (92% for echocardiography vs 94% for angiography), although there was a significantly higher incidence of hypokinesia and akinesia of the apex, and of hypokinesia of the anterior wall during echocardiography ($p < 0.05$); during angiography, wall motion in the apex and anterior walls improved and became normal in a significant number of patients (14% and 18%, respectively) ($p < 0.05$) (Figure 1). In considering the entire group of 44 patients who underwent coronary angiography, 17 (39%) had isolated severe stenosis or occlusion of the LAD, 12 (27%) had 2-vessel, and 15 (34%) had 3-vessel disease. The LAD was occluded in 13 patients (30%) and showed high-grade stenosis ($>75\%$) in 31 (70%). Location of the culprit lesion was proximal in 10 patients (23%), midartery in 30 (68%), and distal in 4 (9%). Acute ST elevation in leads V_1 to V_3 was associated with mid to distal LAD disease (5% distally, 80% at mid LAD) in 85% of patients, and with proximal LAD in only 15% of patients.

DISCUSSION

Our findings challenge the traditional definition of anteroseptal AMI, since 92% of patients had normal septal wall motion during AMI and 94% 7 days after infarction, as determined by echocardiography and by cardiac catheterization, respectively. Thus, rather than defining acute ST changes in leads V_1 to V_3 as a pattern of anteroseptal AMI, we believe that our findings justify defining it as anteroapical AMI. Further, despite the use

of the electrocardiogram to locate most infarcts,¹⁻⁴ several investigators have challenged its accuracy for precisely locating the site of anatomic injury within subdivisions of the myocardium.^{10,11} A study comparing the pathologic findings in patients who die from a heart attack with ECG findings of AMI concluded that "the ECG provides no specific pattern to indicate myocardial infarction of the septum."¹⁰ One year later, a similar pathologic study found that a pattern of diffuse ST elevation in the anterior and lateral leads correlates with extensive damage from an infarct involving the septum and anterior wall.¹²

Our study differs from those previously published because we evaluated abnormal wall motion during the acute phase of injury and during recovery, demonstrating real-time abnormality. We could not analyze or compare data because they gave no clear description of septal injury as part of the anteroseptal or anteroapical AMI, and there was an overlap between the various anatomic sections of the subsegmental infarcts.^{5,10,12}

Our data disagree with the traditional definition of anteroseptal AMI. Using cardiac catheterization and echocardiography, we found a statistically significant correlation between ST-segment elevation in leads V_1 to V_3 and the anatomic findings of an anteroapical infarction. In addition, the culprit lesion was in the mid LAD in 80%, the distal LAD in 5%, and the proximal LAD in only 15% of patients. During the recovery phase, angiography showed total occlusion of the LAD in only 30% of patients and a patent vessel with high-grade stenosis in 70%. This might explain why wall motion abnormality improved during the infarction healing process. After spontaneous reperfusion, myocardium may gradually recover, resulting in improved wall motion in the region of the infarcted artery. Of the 9 patients who underwent angiography and had ST elevation in leads V_1 to V_4 at presentation of the AMI, 7

formed a subgroup with lesions in the proximal LAD and more abnormal, extensive wall motion. It is well known that proximal LAD occlusion is associated with increased mortality and morbidity and diffuse ST changes.¹³⁻¹⁵

We conclude that an infarct associated with ECG changes in leads V₁ to V₃ is due mostly to occlusion in the mid to distal LAD, and it should be defined as anteroapical AMI. Conversely, anteroseptal AMI is an extensive infarct associated with diffuse ECG changes and is due to occlusion in the proximal LAD.

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