Acute Myocardial Infarction
With Isolated ST-Segment Elevation in Posterior Chest Leads V7–9

“Hidden” ST-Segment Elevations Revealing Acute Posterior Infarction

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OBJECTIVES
This study was done to determine whether electrocardiographic (ECG) isolated ST-segment elevation (ST ↑) in posterior chest leads can establish the diagnosis of acute posterior infarction in patients with ischemic chest pain and to describe the clinical and echocardiographic characteristics of these patients.

BACKGROUND
The absence of ST ↑ on the standard 12-lead ECG in many patients with acute posterior infarction hampers the early diagnosis of these infarcts and thus may result in inadequate triage and treatment. Although 4% of all acute myocardial infarction (AMI) patients reveal the presence of isolated ST ↑ in posterior chest leads, the significance of this finding has not yet been determined.

METHODS
We studied 33 consecutive patients with ischemic chest pain suggestive of AMI without ST ↑ in the standard ECG who had isolated ST ↑ in posterior chest leads V7 through V9. All patients had echocardiographic imaging within 48 h of admission, and 20 patients underwent coronary angiography.

RESULTS
Acute myocardial infarction was confirmed enzymatically in all patients and on discharge ECG pathologic Q-waves appeared in leads V7 through V9 in 75% of the patients. On echocardiography, posterior wall-motion abnormality was visible in 97% of the patients, and 69% had evidence of mitral regurgitation (MR), which was moderate or severe in one-third of the patients. Four patients (12%), all with significant MR, had heart failure, and one died from free-wall rupture. The circumflex coronary artery was the infarct related artery in all catheterized patients.

CONCLUSIONS
Isolated ST ↑ in leads V7 through V9 identify patients with acute posterior wall myocardial infarction. Early identification of those patients is important for adequate triage and treatment of patients with ischemic chest pain without ST ↑ on standard 12-lead ECG.

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The standard 12-lead electrocardiogram (ECG) is a relatively insensitive tool for detecting posterior infarction (1–4). This is particularly true in the acute stage when prompt and accurate diagnosis of acute myocardial infarction (AMI) is critical in determining the initiation of reperfusion therapy (5,6). The major obstacle in the ECG diagnosis of posterior infarction lies in the absence of standard leads facing the posterior left ventricular wall, which results in failure to reveal ST-segment elevation (ST ↑) in a high proportion of patients with acute posterior infarction (6,7). Recently, we and others have demonstrated that during acute inferior infarction, ST ↑ in the posterior chest leads V7 through V9 identifies those patients with concomitant posterior wall involvement (8,9). Previous studies (10–12) have shown that ST ↑ is present solely in posterior chest leads in 3% to 4% of all patients with AMI, and in as many as 20% of AMI patients without ST ↑ on the standard 12-lead ECG. However, the combined data from these reports (10–12) included only 14 patients with isolated ST ↑ in leads V7 through V9. Moreover, the posterior location of the infarction was not confirmed in these patients, and the extent and clinical outcome of patients with such infarctions was not investigated.

In the present study we report the clinical, echocardiographic and angiographic findings of 33 consecutive AMI
patients with isolated ST ↑ in posterior chest leads V_7 through V_9.

**METHODS**

Routine admission ECG of each patient referred to the Sheba Medical Center intensive cardiac care unit (ICCU) includes three posterior chest leads (V_7 through V_9) and three right chest leads (RV_3 through RV_5), in addition to the 12 standard leads. Leads V_7 through V_9 are recorded on the same horizontal plane as lead V_6 and in the posterior axillary line (lead V_7) beneath the scapular angle (V_8) and on the paravertebral (V_9) line, respectively. From January 1994 to December 1997, a total of 33 of the patients who were admitted to our ICCU with a provisional diagnosis of AMI or unstable angina pectoris had isolated ST ↑ in posterior chest leads V_7 through V_9 on admission ECG, and they are the subject of our present report.

**ECG.** In accordance with previous studies (8,9,13), we defined significant ST ↑ in leads V_7 through V_9 as an elevation of at least 0.5 mm in two or more of the leads, based on the increased distance between the posterior chest wall and the heart. Q-waves wider than 0.04 s or deeper than one-quarter of the amplitude of the succeeding R-wave were considered pathologic in leads V_7 through V_9.

**Echocardiographic evaluation.** A commercially available echocardiographic system (Hewlett-Packard Sonos 1500; Hewlett-Packard, Andover, Massachusetts) employing a 2.5-MHz transducer was used in this study. Conventional two-dimensional (2DE) and color flow Doppler imaging were carried out to detect valvular regurgitation in multiple views (parasternal, apical and subcostal). Echocardiographic imaging was performed in all patients within 48 h of admission. Images were divided and evaluated by the standard 16-segment model and scoring system (1 = normal or hyperdynamic; 2 = hypokinetic; 3 = akinetic; 4 = dyskinetic) as recommended by the American Society of Echocardiography (14).

For the purpose of wall-motion analysis, the severity of regional wall-motion abnormality in each of the following territories—inferior, posterior and lateral—was determined by the segment with the most severe regional wall-motion abnormality. The presence and severity of mitral regurgitation (MR) was estimated by color flow Doppler imaging using the semiquantitative method based on maximal area of regurgitation jet/left atrial area in the view with maximal regurgitation flow area as previously described (15).

**Coronary angiography.** Twenty of the patients (61%) underwent coronary angiography according to the decision of the attending physician. Indications for catheterization were postinfarct angina in 7 patients, heart failure in 2 and young age in 11. In patients with one-vessel disease, the diseased artery was considered the infarct-related artery (IRA). In those with multivessel disease, the IRA was determined by the presence of angiographic markers of intracoronary thrombus or by the characteristics of the lesions as proposed by Ambrose et al. (16).

**Statistical analysis.** In the comparison of patients with and without significant MR, continuous variables were compared by t test and categorial variables by the two-sided Fisher exact test.

**RESULTS**

The baseline characteristics of the 33 patients representing the study population are presented in Table 1. The mean age of these patients was 57 ± 12.8 years (28 to 74 years). Acute myocardial infarction was confirmed in each of the 33 patients by creatine kinase (CK) elevation of at least twice the upper normal limit and CK-MB of 6% or more of the highest CK value. The mean peak CK value was 663 ± 314 IU/liter (the upper normal range in our laboratory is 80 IU/liter for women and 90 IU/liter for men).

**Admission ECG.** ST-segment elevation was present in leads V_7 and V_9 in 30 patients (91%) and in all 33 patients in lead V_6. ST-segment depression (ST ↓) was noted in leads V_1 through V_3 in 20 patients (61%), and in 22 patients (67%) in at least two consecutive leads of the anterior chest leads V_1 through V_6 (Figs. 1A and 2). Prominent R-waves appeared in lead V_1 in 3 patients (9%) and in lead V_2 in 14
One patient who had complete right bundle branch block (CRBBB) on admission ECG was excluded from the analysis of prominent R waves in leads V1 and V2. In eight patients, no pathologic ST-segment changes were noted on the admission standard 12-lead ECG and the ST \( \uparrow \) in leads V7 through V9 was the only pathologic ST-segment change.

**Discharge ECG.** Discharge ECG was performed on average on the sixth day. Pathologic Q-waves appeared in leads V7, V8, V9 in 20 (61%), 27 (82%) and 28 (85%) patients, respectively (Figs. 2 and 3), and prominent R-waves appeared in 23 (72%) and 28 patients (88%) in leads V1 and V2, respectively. Q-waves in two or more of the leads V7 through V9 appeared in 26 patients (79%) (posterior Q-wave myocardial infarction) and 7 (21%) had posterior non-QMI. Prominent R-waves in leads V1 and/or V2 appeared in 24 (92%) of the posterior QMI patients, but also in 4 of the 7 patients without Q-waves in the posterior leads. Pathologic Q-wave or T-wave inversion occurred in at least two of the leads II, III and aVF in 11 patients, and in at least two of the leads I, aVL, V5 and V6 in 14 patients.

**Echocardiography.** The echocardiographic examination of 32 out of 33 patients (97%) was technically suitable for

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**Figure 1.** Admission ECG showing isolated ST \( \uparrow \) in leads V7 through V9 (A) and the discharge ECG of the same patient showing the appearance of pathologic Q-waves in leads V7 through V9 (B).
interpreting and segmental motion scoring (Fig. 4). Left ventricular posterior wall-motion abnormality was the most common regional dysfunction, which was evidenced in 31 (97%) patients. In the remaining patient, no regional wall-motion abnormality could be detected. Inferior and lateral wall-motion abnormalities were found in 15 patients (47%) and 13 patients (41%), respectively. The posterior wall was also the most severely abnormal, being akinetic or dyskinetic in 19 (61%) of the 31 patients with posterior wall-motion abnormality. Akinesis of the inferior or lateral walls was demonstrated in only approximately 40% of the patients with wall-motion disturbances in these regions. The mean left ventricular ejection fraction (LVEF) percent in the entire study population was 48 ± 6%.

Moderate or severe MR was detected in 7 patients (22%), and mild MR in an additional 15 patients (47%). Patients with moderate or severe MR, as compared with those without, had evidence of a larger infarction, indicated by a tendency toward higher peak CK (822 ± 419 IU/liter vs. 615 ± 261 IU/liter, p = 0.12), lower LVEF (44 ± 5% vs. 49.5 ± 7%, p = 0.06, Table 2), more severe posterior asynergy and more frequent lateral extension (6/7 = 86% vs. 7/25 = 28%, p = 0.005). There was no significant difference in the prevalence of inferior regional wall abnormalities between patients with and without moderate or severe MR (48% vs. 43%).

**In-hospital clinical course.** In-hospital complications (Table 3) included at least one episode of heart failure in four patients (12%), all of whom demonstrated moderate or severe MR. One patient had complete atrioventricular block (CAVB) on admission, which was associated with hemodynamic decompensation and necessitated implantation of a temporary pacemaker. The CAVB was resolved by the fourth day. One patient died suddenly on the eighth day; on autopsy, a free-wall rupture was discovered within the area of a posterior infarction.

**Coronary angiography.** Thirteen patients had one-vessel, while seven had multivessel coronary artery disease. The
culprit lesion was located in the mid-portion of the circumflex coronary artery in 16 patients (80%), and in the first marginal branch of the circumflex coronary artery in 4 patients (20%). Coronary angioplasty was performed in 15 patients, and one patient with multivessel coronary artery disease underwent coronary artery bypass grafting.

**DISCUSSION**

In the present study we demonstrated that in patients with a clinical picture suggestive of AMI, isolated ST ↑ in posterior chest leads (V7 through V9) established the diagnosis of acute posterior infarction. These infarcts were circumflex-artery-related and associated with a relatively high prevalence of moderate or severe MR.

**ECG detection of posterior infarction.** The ECG diagnosis of acute posterior infarction has traditionally been based on the presence of ST ↓ on the precordial chest leads (17–23). However, such ST ↓ are neither specific nor sensitive for the diagnosis of a posterior infarction (1–4,22). These ECG changes might be caused by anterior ischemia (22–27) and therefore do not constitute an indication for thrombolytic therapy (28). Prominent R-waves on leads V1 and V2, which might be the only manifestation of posterior myocardial infarction, do not enable the determination of the age of the infarction.

A few studies (1,13,29) suggested that posterior chest leads improved the diagnostic accuracy of the ECG for the detection of old posterior myocardial infarction (MI). Other studies (10–12) have shown that in 2% to 12% of patients with enzymatically confirmed AMI, ST ↑ occurred only on posterior chest leads. Our results complement these studies by demonstrating that isolated ST ↑ in leads V7 through V9 is indeed associated with acute posterior MI. In addition, we have further expanded these previous studies by investigating the clinical course, and the echocardiographic and angiographic findings in patients with such infarcts.

**Prevalence and clinical significance of MR.** In the present study, moderate or severe MR was detected in 22% of the patients with isolated ST ↑ in leads V7 through V9, and was evident in all the patients who had at least one episode of heart failure. This finding is in accordance with a previous study from our group (30), which showed that posterior involvement was associated with the development of significant MR in patients with acute inferior infarction. Moreover, even mild MR, which was echocardiographically estimated in 47% of our patients, was recently shown to be an independent predictor of long-term heart failure and mortality (31). Thus, ST ↑ in leads V7 through V9 identify infarctions that are potentially harmful, out of proportion to their size owing to their special location in the left ventricle and thereby their potential to cause MR.

**Angiographic characteristics of AMI patients with isolated ST ↑ in leads V7 through V9.** In all catheterized patients the culprit lesion was either in the mid-circumflex coronary artery or in its first marginal branch. This, too, is in accordance with previous studies showing that posterior infarction was associated with circumflex coronary artery occlusion (5,32,33) and that circumflex artery-related infarctions often did not cause ST ↑ on the standard ECG (5–7).

**Clinical implications.** The presence of ST ↑ in leads V7 through V9 may contribute to the triage of patients with chest pain and help in the early differentiation between patients with acute posterior infarction and those with anterior wall ischemia. This distinction may influence the decision-making process regarding the treatment of patients with acute coronary syndrome. Currently, the indication for thrombolytic therapy requires the presence of ST ↑ in the standard 12-lead ECG. However, because ST ↑ is not seen on the standard 12-lead ECG in up to 50% of patients with posterior or circumflex-related infarction (5–7), the pres-

### Table 2. Comparison of Patients With and Without Moderate to Severe Mitral Regurgitation

<table>
<thead>
<tr>
<th>Moderate to Severe MR</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>No. of patients</td>
<td></td>
</tr>
<tr>
<td>7 (22%)</td>
<td>25 (78%)</td>
</tr>
<tr>
<td>ST ↓ V1 through V3</td>
<td>4 (57%)</td>
</tr>
<tr>
<td>Previous MI</td>
<td>1 (14%)</td>
</tr>
<tr>
<td>Infarct Size</td>
<td></td>
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<tr>
<td>Peak CK</td>
<td>822 ± 419</td>
</tr>
<tr>
<td>LVEF</td>
<td>44 ± 5</td>
</tr>
<tr>
<td>Lateral involvement</td>
<td>6 (86%)</td>
</tr>
<tr>
<td>Posterior akinesis</td>
<td>6 (86%)</td>
</tr>
<tr>
<td>Clinical outcome</td>
<td></td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>4 (57%)</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>2 (29%)</td>
</tr>
<tr>
<td>Angiographic results</td>
<td></td>
</tr>
<tr>
<td>Catheterized patient</td>
<td>4 (57%)</td>
</tr>
<tr>
<td>IRA: Mid-CX</td>
<td>3 (75%)</td>
</tr>
<tr>
<td>Multivessel disease</td>
<td>2 (50%)</td>
</tr>
</tbody>
</table>

MR = mitral regurgitation.
ence of ST ↑ should be sought in leads V7 through V9. The identification of the latter ECG pattern will enable this subgroup of AMI patients to benefit from thrombolysis or direct percutaneous transluminal coronary angioplasty. In contrast, patients without ST ↑ in the posterior chest leads (V7 through V9) who present with anterior ischemia manifested by ST-segment depression on precordial anterior chest leads might be candidates for other antithrombotic forms of therapy—for example, Ib/IIa receptor inhibitors or low molecular weight heparin.

Conclusions. The identification of isolated ST-segment elevation on posterior chest leads V7 through V9 in patients with chest pain without ST ↑ on standard 12-lead ECG could be helpful in the early diagnosis of patients with acute posterior infarction, thereby facilitating prompt and accurate treatment. Further investigation is needed to ascertain the accuracy of isolated ST-segment ↑ in V7 through V9 in a large cohort of consecutive patients with chest pain, and to determine the beneficial effect of reperfusion therapy in those patients, especially regarding the prevalence and severity of the resulting MR.

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