An Automated Algorithm to Improve ECG Detection of Posterior STEMI Associated with Left Circumflex Coronary Artery Occlusion

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Abstract

The under-recognition, underestimate of area at risk, and under-treatment of ST elevation posterior myocardial infarct in patients with acute coronary syndrome are often due to the insensitive nature of the 12-lead electrocardiographic diagnosis and bias toward "anterior subendocardial ischemia". We designed a new computer algorithm to improve ECG detection of isolated and combined posterior myocardial infarct using extended posterior leads V7, V8 and V9. ECG criteria for PMI include "age" (acute, recent, age indeterminate or old) and "probability" (consider, probable or definite) infarct. Combining the subjects with posterior myocardial infarct (n=182), we obtained an increase of 26 percentage points in sensitivity of posterior myocardial infarction detection from 14% using standard 12-leads to 40% including posterior leads with a small drop in specificity from 98% to 96.4%. The highest increase was seen in a subset of subjects having undergone for PTCA procedure from 21% to 86% (65 percentage points). We conclude the automated posterior myocardial infarct detection algorithm provides another valuable tool for diagnosis of ST elevation posterior myocardial infarct.

1. Introduction

The under-recognition, underestimate of area at risk, and under-treatment of posterior ST elevation myocardial infarct (STEMI) in patients with acute coronary syndrome (ACS) are often due to the insensitive nature of the 12-lead electrocardiographic (ECG) test. The 12-lead ECG is known to be inadequate in representation of the posterior, lateral, and apical wall of the left ventricle [1]. Posterior STEMI associated with left circumflex (LCx) coronary artery occlusion often missed in admission ECG testing and myocardium at risk in the posterior area is often not identified early. In practice, ST depression in leads V1 to V4 is often used as alternative ECG criteria in PMI detection. However, the diagnostic ECG criterion is not well established in current clinical practice guidelines and no ECG criteria with specific limits and description of ST depression in anterior leads V1-V4 can be followed [2,3]. In addition, bias toward "anterior subendocardial ischemia" due to similar presence of minor ST depression in anterior leads V1-V4 contributes to more problems rather than solutions in posterior STEMI diagnosis (Figure 1). Published clinical studies strongly support the value of posterior leads in posterior STEMI [4-14]. The hypothesis tested in the current study is that the addition of posterior leads V7, V8 and V9 to the standard 12 leads will allow an automated algorithm to better detect ST elevation PMI associated with high-grade LCx coronary artery occlusion regardless of the presence or absence of inferior myocardial infarct, to improve the sensitivity of PMI interpretation, and to better separate posterior STEMI from “anterior subendocardial ischemia”.

2. Study population

Consecutive patients (n = 1,970) admitted for chest pain, chest discomfort or other ACS over a period of 12 months were collected in Rui-Jin Hospital (n = 1,342), Shanghai, China and Lund University Hospital, Lund (n = 565), Sweden. Group I consisting of 154 subjects with single-vessel or multi-vessel disease and LCx greater or equal to 80% occlusion was selected from the 1970 patients. Group II contains 28 subjects undergoing PTCA (percutaneous transluminal coronary angioplasty).
procedures with 100% occlusion induced in LCx at Dalhousie University. Group III is a subset of study group one with ECG presence of PMI identified by a cardiologist manually. Group IV is an ACS-matched control group with negative angiograms. Group V consists of 368 subjects without ACS or prior MI collected at Dalhousie University. It is also important to point out that some of patients’ chest pain was chronic, and that the ECGs were not recorded at patient admission or at the peak of cardiac enzymes for all study subjects in Group I. In Ruijin Hospital, most subjects included in the study were transferred for PCI (percutaneous coronary intervention). In Lund University Hospital, most ECGs were recorded hours after patients received intervention.

3. Data analysis

In our study, AMI is defined as one or more of elevated cardiac enzymes or wall motion abnormality by echocardiogram if available. All subjects with paced ECGs or other ECG confounders such as left and right bundle branch blocks (LBBB, RBBB) or left ventricular hypertrophy (LVH), were excluded in this study. Philips ECG analysis program PH09 was used to detect ECG-LVH, LBBB and RBBB for exclusion. In Group I, standard 12-leads plus V8 and V9 were used for data analysis. In Groups II and V, all subjects had 120 lead body surface mapping ECGs. For data analysis, we used standard 12-leads plus posterior leads V7, V8 and V9. A subset of Group I was used for algorithm training and the rest of the data sets were used for testing.

Automated measurements of selected waveform amplitudes and durations were used for data analysis and criteria development. Measurements were analyzed using the commercial statistical analysis program S-plus. Measurements studied focused on ST amplitude at j point (STj), ST amplitude at j point plus 80 ms (STj+80), and T amplitude. Q wave amplitude and duration from the study and control groups were also compared.

ECG waveform characteristics with three levels of threshold values defined the ECG criteria. ECG criteria for posterior MI include “age” of the infarct and “probability” of the ECG pattern being considered as posterior MI. Though the focus of the study is in acute PMI, we addressed all ages of infarct including "acute", "recent", "age indeterminate" and "old." The probability is divided into three levels: low (consider), medium (probable), and high (definite). The ECG criteria are based on the combination of ST elevation at different levels (greater than 50 \( \mu V \), between 30 to 50 \( \mu V \), or lower than 30 \( \mu V \)), upward or inverted T amplitudes, and sizes of Q wave in posterior leads V7, V8 and V9.

Figure 1. A 79 years old female patient presented with severe chest pain at admission. Based on the ST depression in precordial leads V1-V4, the patient was diagnosed as “anterior subendocardial ischemia”. Cardiac enzyme tests showed elevated TnT (3.2) and CK-MB (135). Isolated ST elevation presented on leads V8 & V9 suggests acute posterior infarct.
4. Results

We tested for the algorithm sensitivity in study groups I, II, III and tested for specificity in groups IV and V. We then compared the algorithm sensitivity based on standard 12 plus V7-V9 with sensitivity based on standard 12-lead only. Sensitivity 2 is from standard 12-leads plus leads V8 and V9. As shown in Table 1, the sensitivity based on standard 12-leads plus V7-V9 increased significantly. The highest increase of 65% (from 21% to 86%) occurred in the single-vessel disease group. In Group II, we also studied the distribution of isolated PMI from PMI combined with other MI regions and found that 42% were isolated PMI and 58% were IPMI (inferoposterior MI) or LPMI (lateroposterior MI).

Combining the Group II single-vessel disease cases (n=28) and Group I multi-vessel disease cases (n=154) together, we obtained a 26% increase in sensitivity in PMI detection from 14% using standard 12-leads to 40% including posterior leads. The specificity dropped slightly, by 2%, from 98% to 96%.

Table 1. Sensitivity of PMI detection algorithm using two lead groups: Sensitivity 1 is from 12 standard leads only; Sensitivity 2 is from standard 12-leads plus leads V8 and V9.

<table>
<thead>
<tr>
<th>Group</th>
<th>Sensitivity 1 (12 leads)</th>
<th>Sensitivity 2 (12 leads + V7-V9)</th>
<th>Δ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I</td>
<td>13%</td>
<td>32%</td>
<td>+19%</td>
</tr>
<tr>
<td>Group II</td>
<td>21%</td>
<td>86%</td>
<td>+65%</td>
</tr>
<tr>
<td>Group III</td>
<td>47%</td>
<td>86%</td>
<td>+39%</td>
</tr>
</tbody>
</table>

Table 2. Specificity of PMI detection algorithm obtained using standard 12-leads only (Specificity 1) and standard 12-lead plus V8 & V9 (specificity 2).

<table>
<thead>
<tr>
<th>Group IV+V</th>
<th>Specificity 1 (12 leads)</th>
<th>Specificity 2 (12 leads + V8, V9)</th>
<th>Δ</th>
</tr>
</thead>
<tbody>
<tr>
<td>752</td>
<td>98%</td>
<td>96%</td>
<td>-2%</td>
</tr>
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5. Discussion and conclusions

A major limitation in the study design is that we used “high-grade occlusion” for selection of the true positive cases and “coronary angiography negative” for selection of the true negative cases. Those criteria are not the best standards for study population selection. Some of the high-grade occlusion may be chronic and may not induce ST elevation and should not be considered as acute PMI. An angiography test if not done properly could potentially miss culprit arteries. Since it is difficult for a single research group to collect the best possible database, a commonly available standard posterior STEMI ECG database would greatly aid ECG criteria development for PMI. However, no such common database is available at present. Another assumption we made in the study is the uniform anatomic distribution of LCx in all subjects. In reality, the posterior region of the myocardium may be supplied by the left circumflex artery or by a dominant right coronary with prominent posterolateral or posterior descending branches.

Despite the limitation in study design, we have obtained some meaningful findings. We confirmed that ST elevation in the posterior leads is the most reliable measure in ECG diagnosis of acute PMI, and that isolated PMI is not rare, especially in patients with LCx occlusion. We found the level of ST elevation in Leads V7-V9 is relatively low compared to ST elevation in other lead groups in association with STEMI in other regions. This observation confirms published data [15,16]. We also found that the Q wave in lead V7-V9 is very helpful in classifying “age indeterminate” or “old” PMI. Our observation matches Dr. Selvester’s study in patients with LCx occlusion showing localized maximum ST elevation on the posterior leads with significant ST elevation in V6 and V7 but without ST elevation in inferior leads [17]. Published studies concluded that 2-12% of patients with enzymatically confirmed AMI, ST elevation occurred only on posterior leads. Matetzky et al. have reported their observation that the acute MI with isolated ST elevation in posterior leads are most likely due to mid left circumflex occlusion or of its first marginal branch [13]. As pointed out by Somers, the use of the additional leads might not only confirm the presence of AMI, but also provide a more accurate reflection of the true extent of myocardial damage [14].

Commonly used PMI ECG criteria include ST elevation in II, III, aVF and/or ST depression, prominent R and upright T in V1-V3. In a study conducted in 2003, we found that based on standard 12-leads alone, it is difficult to establish the limits for ST depression in V1-V4 and high false positive rate was a serious issue [18]. Our results in the current study also show that Leads V7-V9 are the most reliable, and sensitivity is greatly increased when posterior leads are available. Our results support previously published clinical studies that isolated
PMI is not rare. ST depression in V1-V3 is neither sensitive nor specific for diagnosis of ST elevation PMI associated with LCx occlusion. Prominent R in V1 and V2 is not useful to determine the acuteness of PMI, and may be due to poor lead placement. The most reliable ECG findings should be ST elevation in posterior leads with or without associated ST elevation in inferior leads.

Considering the additional time required and the discomfort caused to place additional leads on the back of a patient who is suffering severe chest pain, another possible solution would be to reconstruct Leads V7-V9 from the standard 12-leads as supplemental leads in PMI detection. Given the lead reconstruction techniques developed in recent years, it is possible to reconstruct Leads V7-V9 for use in PMI detection. The reconstructed leads may not be presentable, but certainly are usable in a computer algorithm. We plan to test this approach and to report the results in the future.

In conclusion, the addition of posterior leads allows capture of ECG changes due to isolated PMI or posterior wall involvement associated with MI in other regions with significantly improved sensitivity and only a slight sacrifice of specificity. The automated PMI detection algorithm will provide clinicians with another valuable tool for diagnosis of PMI. PMI detection in the presence of paced ECG and ECG confounders such as LVH, LBBB and RBBB should be further investigated [19,20].

References


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