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Estimated Body Surface Potential Maps in Emergency Department Patients with Unrecognized Transient Myocardial Ischemia

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Abstract

We report on 5 patients who presented to the Emergency Department (ED) with chest pain, were Troponin negative, and were discharged with a presumed noncardiac diagnosis. Thereafter, retrospective analysis of Holter monitoring data recorded for a clinical trial revealed ST events indicative of transient myocardial ischemia that was unrecognized clinically.

Study Aim— The purpose of this analysis was to determine whether initial body surface potential maps estimated from optimal ischemia electrode sites (EBSPM) showed signs of ischemia in the missed ischemia group that could have prevented misdiagnosis.

Methods— Secondary analysis of data from the IMMEDIATE AIM Study, a prospective clinical trial in which patients were attached to 2 Holter monitor devices for simultaneous recordings. One Holter device recorded a standard Mason-Likar 12-lead ECG and the other recorded a 10-electrode lead set considered optimal for ischemia detection. A body surface potential map was then estimated from the optimal lead set.

Results— At 1 year, 2 of the 5 patients with missed ischemia died and a 3rd had an acute myocardial infarction (40% mortality; 60% death/non-fatal MI). In comparison, 1-year mortality was 5.7% in 159 similar patients treated for unstable angina at the same institution over the same time period (p=. 037). The initial standard ECG was normal in 3 and showed LVH in 1. The 5th patient with history of recent MI had slight ST elevation in leads III and aVF and Q waves that were considered indicative of recent (not acute) MI. EBSPM data recorded at the time of ED presentation matched the standard ECG (normal in 3; LVH or inconclusive in 2). During transient ischemia all 5 EBSPMs showed areas of ischemia overlapping with standard electrode sites.

Conclusion— Patients evaluated in the ED for chest pain are at high risk for death or non-fatal MI if they have ischemic events with continuous ST-segment monitoring that are unrecognized clinically. Initial body surface potential maps estimated from optimal ischemia electrode sites do not improve on 12-lead ST segment monitoring in identifying this high risk group.

Introduction

In patients who present to the ED with chest pain, it may be difficult to identify those who have unstable angina given the many causes of chest pain, often inconclusive electrocardiographic findings, and the potential for serum biomarkers to remain negative unless unstable angina progresses to infarction. We report here on 5 patients who presented to the ED with chest pain,

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were Troponin negative, and were discharged with a presumed noncardiac diagnosis. Thereafter, retrospective analysis of Holter monitoring data recorded for a clinical trial revealed ST events indicative of transient myocardial ischemia that were unrecognized clinically. The presumed reasons that transient ischemia was missed in this group were that clinicians were monitoring a single lead II which, by itself, is often insensitive for detecting ischemia. In addition, automated ST-segment monitoring was not in use with the bedside monitors in the ED or hospital units.

The purpose of the present analysis was twofold: 1.) To compare outcomes at one year between this "missed ischemia" cohort with similar Troponin-negative patients treated for unstable angina at the same institution over the same time period and, 2.) To determine whether initial body surface potential maps estimated from electrode sites optimized for ischemia detection showed signs of ischemia that may have prevented misdiagnosis.

Methods

Study Design

This report is a secondary analysis of data from a prospective clinical trial, the IMMEDIATE AIM study (Ischemia Monitoring & Mapping in the Emergency Department In Appropriate Triage & Evaluation of Acute Ischemic Myocardium). Patients were enrolled in the study between 2002–04 and one-year follow-up was completed in December, 2005. The overall goal of the IMMEDIATE AIM study was to improve the noninvasive ECG diagnosis of patients who present to the ED with acute coronary syndrome. Specific aims were to: (1) acquire continuous, 24-hour, standard 12-lead ECG Holter recordings in cohorts of ED patients undergoing evaluation for possible acute coronary syndrome, (2) simultaneously acquire continuous, 24-hour Holter recordings from electrode sites considered optimal for ischemia detection (Figure 1) and then estimate body surface potential maps,[1] and (3) compare sensitivity and specificity of standard electrocardiography with the EBSPM method for identifying acute myocardial ischemia and infarction. All Holter recordings were stored for later "off-line" analysis and neither method was used for "real-time" clinical decision-making.

Sample

All patients presenting to the ED with chest pain or anginal equivalent at the University of California, San Francisco Medical Center between the hours of 7 am and 7 pm, Monday through Friday, were invited to participate in the IMMEDIATE AIM Study. Patients with deviated ST segments due to left bundle branch block or ventricular pacemaker rhythm were excluded. In order to start Holter monitoring immediately upon the patient's presentation to the ED, institutional review board approval was granted for application of Holter monitors with an initial verbal assent followed by informed written consent when the patient was stable. Median time from ED "door to Holter" in the parent IMMEDIATE AIM Study was 45 minutes (n=1308).

The comparison groups for this secondary analysis included 159 patients in the IMMEDIATE AIM Study who had a final diagnosis of unstable angina and the 5 patients with a (presumed) final diagnosis of noncardiac chest pain who had unrecognized transient myocardial ischemia. Both groups were Troponin negative for acute infarction. Unstable angina was defined as a clinical history consistent with a diagnosis of unstable angina, in whom ischemia has been confirmed by presence of ST changes on the initial ECG or in association with recurrent rest pain, or presence of small elevations of troponin that do not meet infarction criteria.[2]

Procedures

Specially trained research nurses applied standard and optimized ECG leads, supervised the Holter recordings over the 24 hour period, and down-loaded stored ECG data for subsequent analysis. Radio-lucent electrodes and lead wires were used to insure uninterrupted monitoring during portable chest X-rays and cardiac catheterization procedures.

Follow-up Data

Research nurses collected one-year mortality data via telephone calls to subjects, hospital and clinic electronic records, and a public internet-based mortality database. One-year follow-up was successfully obtained from all 5 of the missed ischemia cohort and from 87.4% of the unstable angina patients.

Analysis of ECG Data

Standard 12-lead ECG—All standard Holter monitor recordings were analyzed by one cardiologist (KEF) using the H-Scribe System (Mortara Instruments, Milwaukee, WI). Transient myocardial ischemia was defined as a change in ST amplitude at J+60 milliseconds of ≥100 μV in ≥2 contiguous ECG leads or ≥200 μV in one lead lasting ≥1 minute(s). At the start of Holter monitoring, ECGs were recorded in supine, left and right side-lying positions to prevent body position ST-T wave changes being misdiagnosed as transient ischemia. ST events occurring in the cardiac catheterization laboratory were not counted as ischemic events because of the likelihood they were induced by catheter manipulation or balloon inflation.

Estimated Body Surface Potential Maps—ECG data recorded from optimal leads were transmitted to the co-investigator's research laboratory (RLL) at the University of Utah where body surface maps and ST amplitudes at J+60 ms were estimated for 192 torso sites. Investigators generating EBSPM data were blinded from standard ECG and clinical data. For the present analysis, EBSPMs were analyzed at the time of the initial standard 12-lead ECG and at the time of ST events diagnosed by the cardiologist's review of standard Holter recordings. The EBSPM displays were developed using Map3d.[3] The torso displays were color-coded with areas of isoelectric ST segments shown in green, ST elevation shown in yellow, orange and red in order of increasing amplitude, and increasing ST depression shown in light to dark blue.

Statistical Analysis

Chi square statistical analysis was used to determine whether one-year mortality was different between the missed ischemia cohort and similar patients treated for unstable angina. Because some of the 2 X 2 cells had a count less than 5, the Fisher's Exact (2-sided) method was used to determine statistical significance.

Results

Sample

Sample characteristics are summarized in Table 1. The missed ischemia cohort included 3 males and 2 females with a mean age of 67 years and 60% were an ethnic minority.

The clinically unrecognized ST events detected with retrospective Holter analysis included 1 event in 3 patients and 2 events in 2 patients. The median time from ED presentation to the first ST event was 5.5 hours (25^{th} percentile, 3.6 hours; 75^{th} percentile, 14.4 hours). Two patients had ST elevation events and 3 patients had ST depression events. In the ECG lead with maximal ST deviation, the mean change in ST amplitude from pre-event to event was 170 ± 45.7 μV.

Initial Standard ECG in the Missed Ischemia Cohort

The standard 12-lead ECG at initial presentation to the ED was normal in 3 patients and abnormal in 2. Patient #4 had LVH with the expected secondary ST elevation in leads V_2-V_3 and ST depression in V_5-V_6. Patient #5 with a history of recent MI had slight ST elevation (≤100 μV) in leads III and aVF and Q waves in the same leads that were thought to be due to recent (rather than acute) MI.

Initial and ST Event EBSPMs in the Missed Ischemia Cohort

Initial and ST event EBSPMs for the 5 patients with missed ischemia are shown in Figures 2 and 3. Figure 2 shows the EBSPMs for the 3 patients with a normal initial standard 12-lead ECG. The initial EBSPMs in these 3 individuals were also normal showing isoelectric ST segments across the torso. During transient ischemia in these 3 patients, the EBSPMs showed ST segment elevation (patient #1) or depression (patients #2 and #3) overlapping with standard precordial lead sites.

Figure 3 shows the EBSPMs for the 2 patients with initial abnormal standard ECGs due to LVH or recent MI. During transient ischemia in the 4th patient with LVH, ST depression became more extreme in the region overlapping standard leads V_5-V_6. During transient ischemia in the 5th patient with recent MI, the EBSPM showed ST deviation in line with the axis of standard leads III (ST elevation) and aVL (ST depression). Figure 4 shows this patient's initial and ST event ECGs and confirms that standard leads III and aVL were as sensitive as the EBSPM in depicting transient ischemia.

One-Year Patient Outcome

Two of the 5 patients in the missed ischemia cohort died and a 3rd had an acute MI (40% mortality; 60% death/non-fatal MI). Both deaths occurred in the patients with abnormal initial standard ECGs due to LVH or recent MI. The patient with recent MI died 15 days following hospital discharge. In comparison to the missed ischemia group, one-year mortality was 5.7% in IMMEDIATE AIM Study patients presenting with similar symptoms and negative biomarkers who were treated for unstable angina at the same institution during the same time period (p=.037).

Discussion

This is the first study to analyze body surface potential maps in patients with unstable angina and spontaneous transient myocardial ischemia. All prior mapping studies have focused on patients with acute MI or the angioplasty balloon occlusion model of acute MI.[4–8] Continuous recording of body surface maps in clinical practice is difficult, if not impossible to maintain, because the multiple electrodes and lead wires tether the patient and interfere with patient care, including emergency interventions such as defibrillation and cardiopulmonary resuscitation. We developed a body surface map estimated from a reduced number of electrodes/leads that would be feasible for continuous monitoring. However, these preliminary findings in a small cohort of patients with missed ischemia indicate that EBSPMs would not have prevented misdiagnosis at the time of ED presentation. Moreover, during transient ischemia, ST deviation in the EBSPMs overlapped with standard lead locations.

In prior studies, investigators have reported that body surface maps, when compared to standard 12-lead electrocardiography, result in higher sensitivity and specificity for acute MI.[4–8] However, there are issues to consider in these studies. For example, all but one was from the same institution and all studies acknowledged funding from the manufacturer of the commercial body surface map system. Moreover, when standard ECGs were illustrated alongside the body surface maps in publications, ST-T wave changes of ischemia were apparent

in standard ECGs as well as the body surface maps.[6, 8] Thus, it was unclear whether the body surface maps truly provided independent information above and beyond the standard ECG. In the small study using the angioplasty balloon occlusion model of acute MI, investigators reported that the maximal ST segment deviation occurred in the body surface map in 21/23 (91.3%) of patients.[7] However, it was not stated whether ST deviation less than maximal was observed in the standard ECG that would lead to the same diagnosis of ischemia.

In ED settings, the ECG diagnosis of acute ischemia that is likely to develop into infarction is difficult. Patients often have pre-existing ECG confounders such as bundle branch block, LVH with strain, digitalis therapy, etc. that produce ST-T wave abnormalities mimicking acute ischemia/infarction. Ischemic ST-T wave changes are more complex than simple J+60 ms ST amplitudes, whether generated from body surface mapping or standard electrocardiography. For example, clinicians are more suspicious of ischemia/infarction if there is a coved shape to the ST segment with ST elevation, a horizontal or down-sloping segment with ST depression, or a sharp angle to the initial limb of the T wave or symmetry with T wave inversion. These qualitative features are difficult to translate into computer algorithms. Despite these subtle features that can be appreciated by the human eye, cardiologists have not been found to be much better at the diagnosis of acute MI (sensitivity 45%, specificity, 94%) than a computer algorithm (sensitivity 32%, specificity, 98%).[6]

In the present study, continuous ST-segment monitoring for 24 hours was the only method useful in identifying the high risk cohort of patients with unrecognized ischemia. Continuous ST-segment monitoring was also beneficial for the patients with ECG confounders of LVH or recent MI because it detected a change using the patient's own abnormal baseline as the reference point. Our findings showed that the median time to 1st ST event in the missed ischemia group was just 5.5 hours. This time period is similar to the time patients spend in the ED for evaluation of chest pain. For example, the median ED length of stay for patients in the IMMEDIATE AIM Study was 6.75 hours with 75% of patients staying nearly 10 hours. Thus, it is likely that if multi-lead ST-segment monitoring had been used in the ED, clinicians would have been alerted to these ST events in the missed ischemia cohort before they left the ED.

Study Limitation

Because analysis of the full 24 hours of EBSPM data has not yet been completed, it is unknown whether EBSPMs showed ST events at times the standard ECG did not. It is unlikely; however, that the EBSPM method would have identified the 5 patients with missed ischemia any earlier than the standard ECG because both were initially normal or inconclusive due to confounding conditions. Whether there is clinical benefit from continuous monitoring of EBSPMs in other cohorts of patients with ACS remains the topic of ongoing analysis of the IMMEDIATE AIM Study.

Conclusion

Patients who present to the ED with chest pain and who have unrecognized transient myocardial ischemia have a high one-year mortality compared to similar patients who are treated for unstable angina. Findings from this preliminary analysis suggest that it may be more important to monitor ST segments over time than over a larger region of the body torso in the identification of patients with acute coronary syndrome.

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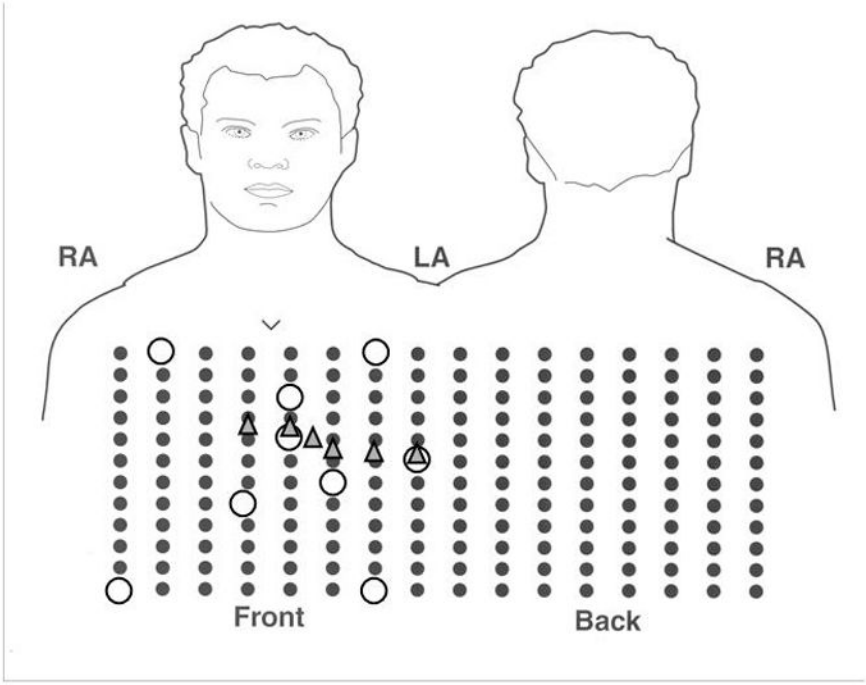

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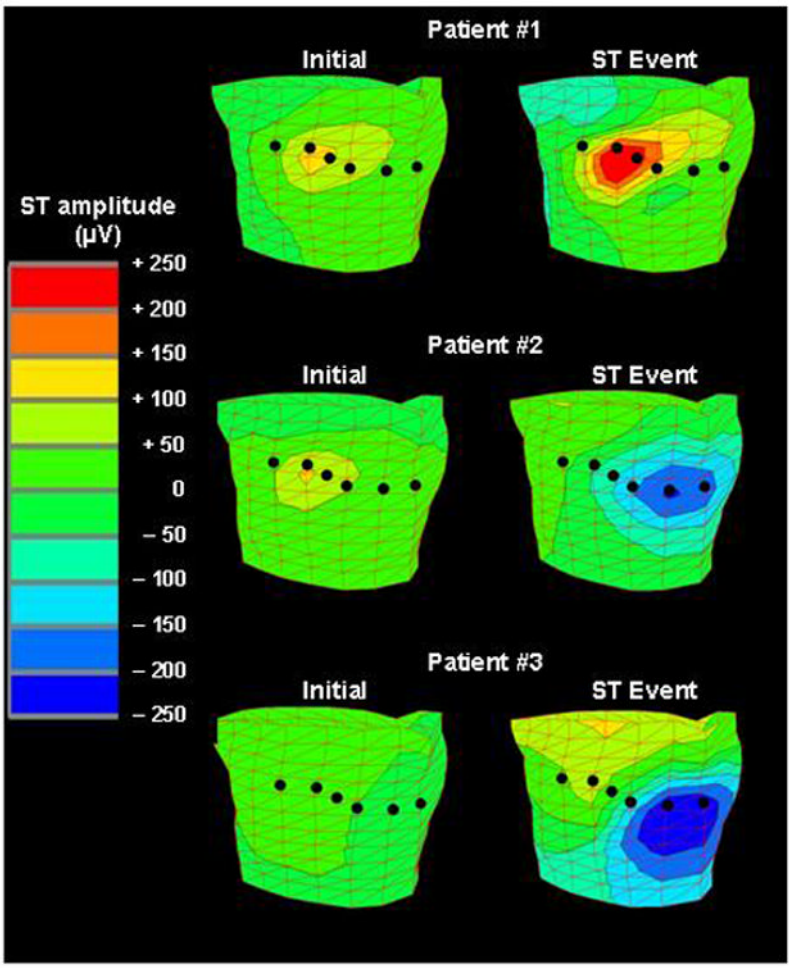

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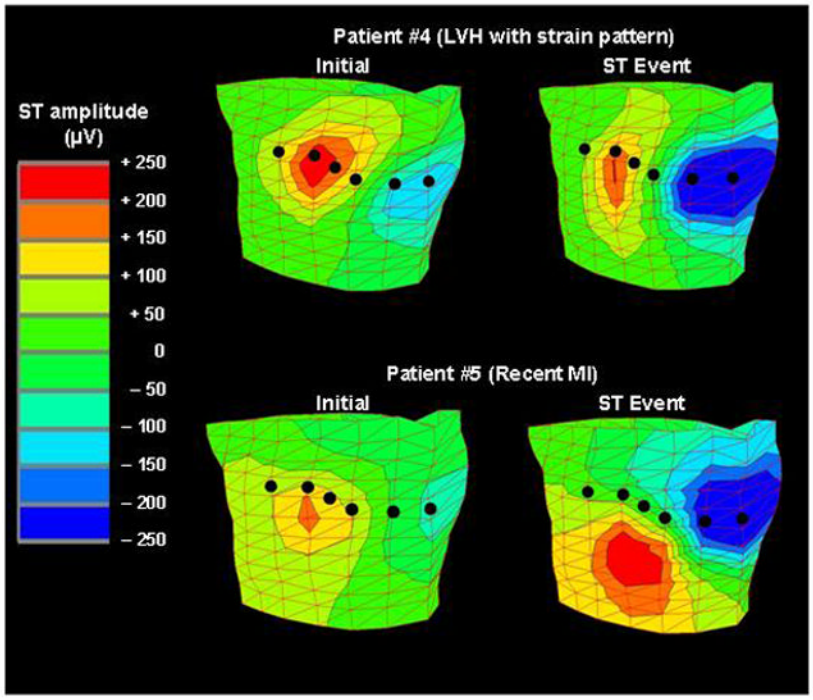

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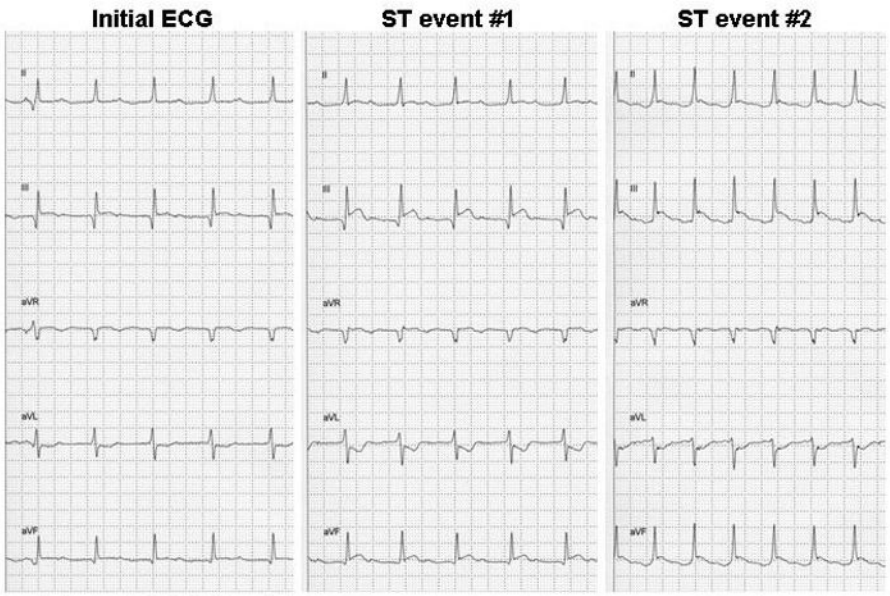

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Table 1

	Transient Myocardial Ischemia Detected with Holter Monitoring				1-Year Outcome (death/MI?)
	# events over 24 hr	Type of Event (ST elevation or depression)	Maximal ΔST (pre-event to peak ST amplitude with event)	Max ST Lead	
	1	ST↑	244 μV	V_3	No
	1	ST↓	158 μV	V_4	No
	2	ST↓	148 μV	III	Non-fatal MI
	1	ST↓	123 μV	V_5	Died
lished Q waves & history of recent MI	2	ST↑	177 μV	III	Died