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ScienceDirect
Journal of Electrocardiology 40 (2007) S15 – S20
JOURNAL OF Electrocardiology
www.jecgonline.com

Estimated body surface potential maps in emergency department patients with unrecognized transient myocardial ischemia☆

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Received 7 April 2007; accepted 30 May 2007

Abstract

Background: We report on 5 patients who presented to the emergency department (ED) with chest pain, had negative serum troponin levels, 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 estimated body surface potential map (EBSPM) showed signs of ischemia in the missed ischemia group that could have prevented misdiagnosis.

Methods: This is a secondary analysis of data from 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 electrocardiogram (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 third had an acute myocardial infarction (MI) (40% mortality, 60% death/nonfatal MI). In comparison, 1-year mortality was 5.7% in 159 similar patients treated for unstable angina at the same institution over the same period (P = .037). The initial standard ECG showed no abnormalities in 3 patients and showed left ventricular hypertrophy in 1. The fifth patient with a 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, left ventricular hypertrophy 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 nonfatal MI if they have ischemic events with continuous ST-segment monitoring that are unrecognized clinically. In this small cohort with unrecognized ischemia, the initial body surface potential maps estimated from optimal ischemia electrode sites did not improve on 12-lead ST-segment monitoring to identify this high-risk group.

Keywords: Electrocardiogram; Body surface map; Myocardial ischemia; Acute coronary syndrome; Emergency department; Monitoring, physiologic

Introduction

In patients who present to the emergency department (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 biomarker results to remain negative unless unstable angina progresses to infarction. We report

☆ This study was supported by a grant from the National Heart, Lung, and Blood Institute (RO1HL69753), by the General Clinical Research Center, University of California, San Francisco, and the Cardiovascular Research and Teaching Institute, University of Utah.

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0022-0736/$ – see front matter © 2007 Elsevier Inc. All rights reserved.
doi:10.1016/j.jelectrocard.2007.05.032

here on 5 patients who presented to the ED with chest pain, had negative serum troponin levels, 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) that, 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 2-fold: (1) to compare outcomes at 1 year between this "missed ischemia" cohort with similar troponin-negative patients treated for unstable angina at the same institution over the same 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 Ischemia Monitoring and Mapping in the Emergency Department in Appropriate Triage and Evaluation of Acute Ischemic Myocardium (IMMEDIATE AIM) study. Patients were enrolled in the study between 2002 and 2004 and 1-year follow-up was completed in December 2005. The overall goal of the IMMEDIATE AIM study was to improve the noninvasive electrocardiogram (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 (Fig. 1) and then estimate body surface potential maps (EBSPM),[1] and (3) compare the sensitivity and specificity of standard electrocardiography with the EBSPM method for identifying acute myocardial ischemia and infarction. All Holter recordings were stored for later "offline" analysis and neither method was used for "real-time" clinical decision making.

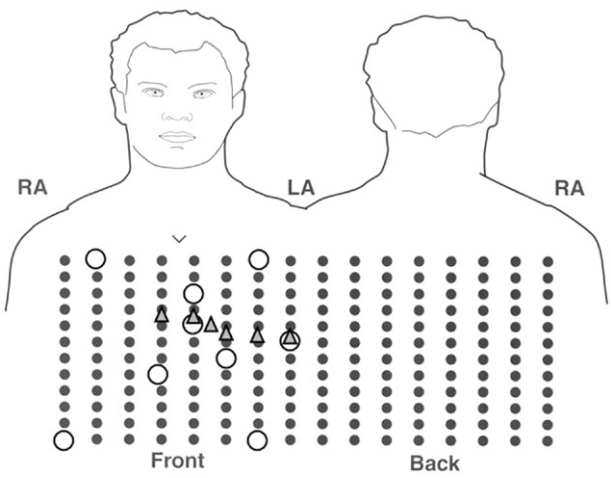

Fig. 1. The 9 white circles plus a 10th ground electrode (not shown) comprise the electrode sites considered optimal for ischemia monitoring that were used in the IMMEDIATE AIM Study. The 192 body surface leads were estimated from these optimal leads. The gray triangles represent electrode sites for the standard precordial leads.

Sample

All patients presenting to the ED with chest pain or anginal equivalent at the University of California, San Francisco Medical Center, between 7:00 AM and 7:00 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 downloaded stored ECG data for subsequent analysis. Radiolucent electrodes and lead wires were used to insure uninterrupted monitoring during portable chest x-ray and cardiac catheterization procedures.

Follow-up data

Research nurses collected 1-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 patients with unstable angina.

Analysis of ECG data

Standard 12-lead ECG

All standard Holter monitor recordings were analyzed by 1 cardiologist (K.E.F.) 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 or greater in 2 or more contiguous